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CASE REPORTS

[Abstract:0003][Psychopharmacology]

Quetiapine associated with angioedema

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Quetiapine is an atypical antipsychotic indicated for the treatment of schizophrenia and bipolar disorder, both manic and depressive episodes. Quetiapine is also widely off label used for treatment of various psychiatric disorders. Angioedema is characterized with swelling of deep dermis and subcutaneous tissues without itching, often seen around the eyes, lips and genitals where subcutaneous tissue is loose. Histamine and bradykinin release by stimulation of mast cells via foods, drugs, infections, and physical stimuli lead vascular permeability and dilatation. Drug-related angioedema is most commonly seen with ACE inhibitors and NSAIDs. Here, we report a male patient of angioedema associated with quetiapine use.

Case: A 36 year-old male patient with a 12 years follow-up diagnosis of bipolar disorder presented to our outpatient clinic with symptoms of reduced sleep, hyperactivity, irritability, restlessness, excessive talkativeness, distractibility, flight of ideas, grandiose, and mystic delusions. His physical and laboratory examinations (complete blood count, renal and hepatic function tests, urinalysis, electrolytes, thyroid function tests, and sedimentation rate), electrocardiography, and magnetic resonance imaging were normal. The patient was diagnosed with bipolar disorder, manic episode with psychotic features according to the DSM-5. Quetiapine 100 mg/day was started in the first day of the hospitalization. The dose was titrated to 200 mg/day in the 2nd day and 400 mg/day in the 3rd day. On the 4th day after introduction of quetiapine, facial swelling and periorbital edema was observed in his physical examination. He had no symptoms of pruritus or any infections. The patient was consulted with dermatology and was diagnosed with angioedema. Repeated laboratory tests were unremarkable. He had no medical and family history of angioedema and allergies. There were no other medical or herbal drug use by the patient. Quetiapine was stopped. Facial and periorbital edema was subsided within 2 days. Risperidone was started and titrated to 6 mg/day. No cutaneous drug reactions were observed. Naranjo Probability Scale revealed a score of 6, probable adverse effect caused by quetiapine. Drug re-challenge was not considered because of angioedema can be fatal when involving the larynx. The diagnosis was compatible with "quetiapine associated angioedema". Facial and periorbital edema was subsided again within 3 days. The patient was discharged from the hospital in complete remission with risperidone 6 mg/ day. On his follow-up visits, no skin reactions were observed. To our knowladge, this is the first case report of angioedema associated with quetiapine. In literature, several antipsychotics (e.g., haloperidol, droperidol, chlorpromazine, clozapine, risperidone, olanzapine, ziprasidone, and paliperidone) were found to be associated with angioedema. Angioedema associated with drug use, is usually IgE mediated and usually occurs in the first week of drug therapy. As in our case, angioedema usually resolves within 24- 48 hours after cessation of the drug and recurs in case the same drug is given again.

Keywords: quetiapine, angioedema, antipsychotics

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[Abstract:0007][Schizophrenia and other psychotic disorders]

Sulfasalazine-induced major depressive disorder with psychotic features

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Ankylosing spondylitis (AS) is a chronic inflammatory disease of unknown cause and affects mainly the spine, but can also affect other joints. Ankylosing spondylitis is the prototype of spondyloarthropathies that affects approximately 0.49% of the Turkish population and 0.9% of the world population. Non-steroid anti-inflammatory drugs are the primary treatment of choice. Sulfasalazine is a disease-modifying antirheumatic drug used in the treatment of AS. Sulfasalazine may cause central nervous system adverse effects such as serious psychiatric problems including mania, depression, and psychosis, and these symptoms have been reported to occur only infrequently. This present case reports a male patient who presented due to major depressive disorder with psychotic features. He had been receiving 1000 mg/day sulfasalazine for AS while he developed serious psychiatric symptoms.

Case: The patient was a 46 year-old male, married with two children, works as a security guard, and high school graduate. The patient presented to our psychiatry outpatient clinic due to psychiatric symptoms like depressed mood, disturbed sleep and appetite, severe anxiety, and parapid thoughts like he was being followed. His psychiatric examination revealed that he was wide-awake and that his orientation and cooperation were good, though he spoke much slower than he was used to. His mood and affect were depressed. He had delusions of persecution and reference. The patient's medical history showed that he had been diagnosed as having AS. He had been prescribed 500 mg/day sulfasalazine and treatment was increased to 1000 mg/day. After he was prescribed sulfasalazine, his depressive symptoms increased in 2 months. He had visited our outpatient clinic 3 months after the onset of sulfasalazine treatment with his severe psychiatric symptoms such as depressive mood, delusions of persecution and reference. The rheumatologist had discontinued his sulfasalazine treatment after rheumatology consultation. We started an outpatient treatment by prescribing sertraline 50 mg/day and olanzapine 10 mg/day, and arranged a follow-up appointment in 10 days. On follow-up examination, we realized that he had started using the medications as instructed. His psychotic symptoms disappeared and depressive symptoms diminished significantly in 1 month. It was concluded that sulfasalazine could be responsible for the psychiatric symptoms due to fact that they disappeared upon the discontinuation of sulfasalazine. He was followed up with the diagnosis of 'substance -induced depressive disorder' according to DSM-5. He was examined monthly during the follow-up period. It has been a year since he was diagnosed by our outpatient clinic and he has exhibited no psychiatric symptoms so far.

After the patient started taking antidepressant and antipsychotic drugs, a rapid disappearance of severe psychiatric symptoms led us to conclude that this improvement might not be simply related to use of drugs but probably due to discontinuation of sulfasalazine. Also, neither the patient nor his family members had a history of psychiatric disorders. For these reasons, we considered a correlation between sulfasalazine use and appearance of psychiatric symptoms. With this in mind, we reviewed the current literature and found three psychiatric cases in association with sulfasalazine.

Keywords: depression, psychosis, sulfasalazine

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[Abstract:0019][Psychopharmacology]

Lithium intoxication and neuroleptic malignant syndrome induced by aripiprazole: a case report

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Neuroleptic malignant syndrome (NMS) is a life-threatening neurologic complication associated with the use of neuroleptic agents and characterized by a distinctive clinical syndrome of fever, rigidity, autonomic nervous system dysfunction, and mental status change. Indeed, second-generation antipsychotics (SGAs) were originally assumed to be free from the risk of causing NMS, however several cases of NMS induced by SGAs (SGA-NMS) have been reported. We herein presented a patient suffering from bipolar disorder who had developed NMS with using aripiprazole and lithium.

Case: A 45 year-old female presented to our clinic with the diagnosis of bipolar disorder; the complaints of increased energy, decreased need for sleep, unusual talkativeness, and racing thoughts. She had a history of subsequent hospitalizations for four times after an initial diagnosis of bipolar disorder was made 18 years ago. She was hospitalized for seven days and maintained with lithium 1200 mg/day, aripiprazole 30 mg/day, biperiden 2 mg/day for this manic episode. One week after her discharge, she presented to the psychiatric emergency unit with insomnia, fatigue, rigidity, confusion, fever, and tremors. Investigations revealed the leukocytosis (WBC: 14.4 µ/l) concomitant with an elevated creatinine phosphokinase (CK) level of 535 IU/L (normal:<200 IU/L) and a high lithium level of 1.75 mEq/L (normal: 0.6-1.2 mEq/L). In light of her fever, encephalopathy, elevated creatine kinase levels, and muscle rigidity, a diagnosis of NMS and lithium intoxication was made. She was referred to neurology intensive care unit. The clinical condition subsided under supportive therapy for two weeks and she was discharged under treatment with lithium 600 mg/day and quetiapine 50 mg/day.
NMS is an idiosyncratic, life threatening adverse effect to antipsychotic drugs. With the wide spread use of SGAs, the incidence of NMS has been decreasing progressively and it has been estimated that 0.01% to 0.02% of patients develop this fatal adverse reaction with antipsychotics. Although lithium-induced NMS was reported as rare case reports, it is mostly speculated that combination therapy with other antipsychotics may more likely lead to NMS. Lithium is hypothesized to contribute to the development of NMS by strengthening the effect of dopamine blockade of antipsychotics or presynaptic inhibition of dopamine synthesis. In the literature, description of NMS in a patient with aripiprazole combined with lithium therapy is a distinct clinical entity and is worthy of particular attention due to rarity of this condition. Clinicians should be aware that NMS is virtually associated with all antipsychotics. Although apparently less severe than NMS induced by typical antipsychotics, SGA-NMS still represent a relevant clinical issue and a possible risk of toxic interactions between lithium and SGAs may prelude NMS in clinical grounds.
Keywords: neuroleptic malignant syndrome, aripiprazole, lithium

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[Abstract:0020][Psychopharmacology]

Parkinsonism that develops with quetiapine: a case report

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Movement disorders such as dystonia, akathisia, late parkinsonism, tics, myoclonus, tremors, chorea, and ballismus could develop due to drug use. Parkinsonism could appear as an adverse effect of several drugs that inhibit dopamine transmission in nigrostriatal system, primarily typical antipsychotics. However, parkinsonism risks of drugs that bond with both D2 and serotonin (5-HT2A) receptors such as atypical antipsychotics were reported to be lower. Quetiapine is a di-benzodiazepine derivative antipsychotic drug with high serotonin/dopamine bonding rate. Despite this fact, literature review would reveal that there were cases of EPS due to quetiapine use, albeit rare. Here, a late period parkinsonism picture developed in a patient using quetiapine for bipolar disorder diagnosis will be discussed.
Case: The subject was a 32 year-old, single, elementary school graduate, female patient. She presented to the outpatient clinic with complaints of insomnia, chatterbox syndrome, nervousness, and distress and was admitted as an inpatient with a tentative diagnosis of bipolar disorder, hypomanic episode. The patient was admitted for treatment with a diagnosis of bipolar disorder manic and depressive episodes previously as well. Both physical examination and serum laboratory analyses were within normal range. The patient was prescribed Valproate 1000 mg/day and Quetiapine 600 mg/day treatment. The patient was discharged since her complaints decreased significantly. The patient was compliant with her follow-up appointments and on the fifth month follow-up examination, she presented with slowing in walking pace and in her mimics. Neurological examination revealed bilateral gear, rigidity, bradymimia, bradykinesia, and intentional tremor symptoms. Her current condition was evaluated as parkinsonism due to quetiapine use. Quetiapine dose was gradually lowered and discontinued. Biperiden 2 mg/day treatment was added to the treatment. In her follow-up examination a month later, it was observed that the patient's complaints continued, although slightly reduced.
The present article is a case report where parkinsonism developed due to quetiapine drug use. Late period parkinsonism could be observed due to quetiapine, albeit quite rare. Reporting such adverse effects would contribute to the current literature.
Keywords: parkinsonism, quetiapine, dopamine

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[Abstract:0023][Psychopharmacology]

Dystonia induced by sertraline use: a case report

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Selective serotonin reuptake inhibitors (SSRI) are effective antidepressant group drugs with lower adverse effect profiles than other antidepressant drugs. The adverse effects of SSRIs are primarily nausea and gastrointestinal problems, tremor, insomnia, dizziness, and sexual dysfunction. Rarely, extrapyramidal symptoms such as akathisia and dystonia were also observed with this drug group. In the present case report, a case of dystonia that developed in the jaw region of a patient, who was diagnosed with obsessive-compulsive disorder, due to sertraline use will be discussed.

Case: The subject was a 43 year-old male patient, father of two and employed as a civil servant. The patient's background and family history was unremarkable and presented to psychiatry outpatient clinic due to intense obsessional thoughts. His complaints were evaluated and he was started on sertraline 50 mg and the dose was increased to 100 mg after a week of treatment. The patient, who was not on any medications other than sertraline, developed complaints of contraction in his lower face, especially the jaw region, which caused difficulties to open his mouth, in the fourth week of the treatment. Neurological examination revealed normal muscle tonus, isochoric pupillary, normoactive deep tendon reflexes in all extremities; the patient lacked pathological reflexes, and no abnormal findings were found during sensual and cerebellar system examination. Due to the sudden onset of the patient's complaints and the lack of previous similar complaints, it was considered that the dystonia developed in his jaw region was as a result of sertraline use and the administration of sertraline was discontinued. Pursuant to the discontinuation, contraction complaint of the patient gradually receded and he completely recovered after five days. Due to his existing psychological complaints, the patient was started on fluoxetine and a follow-up appointment was arranged for 20 days later.

It was estimated that extrapyramidal symptoms induced by SSRI use were due to the suppressive effects of serotonin on dopaminergic pathways in basal ganglia. In a previous case report, it was reported that a patient, who was on sertraline 50 mg/day, developed dystonic movement disorder after 3 months. However, this adverse effect due to sertraline use is limited to a few case studies in the literature. Patients on sertraline rarely develop dystonia. It needs to be kept in mind that patients that we advise SSRI use could develop such extrapyramidal adverse effects.

Keywords: sertraline, dystonia, extrapyramidal symptom

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[Abstract:0025][Mood disorders]

A family with bipolar affective disorder: a case report

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Bipolar disorder (BD) is a mood disorder with relapses and remissions and could significantly result in morbidity and mortality. It was reported that bipolar disorder runs in the family. Studies demonstrated that the prevalence of Mood disorders and other mental disorders among the immediate relatives of patients with BD were higher compared to other individuals. Here, we report BD diagnoses in a family of two adult parents and their daughter, who concurrently presented to the neurology clinic with complaints of headache and insomnia.

Case 1: The subject was a 54 year-old, male patient, married with 3 children. Inpatient psychiatric consultation was requested from the neurology unit with the complaints of insomnia, nervousness, and headaches, and he was evaluated in the psychiatry clinic. Patient's history reflected that he was married to his aunt's daughter and the aunt was a BD patient. The patient presented to the psychiatry outpatient clinic several times. The patient lacked insight and his mood was expansive. Also examination symptoms such as logorrhea, irritability, insomnia, distractibility, aggression, excessive spending, increase in religious activities, and libido were identified in the patient. The patient was admitted in our unit with BD (manic episode) diagnosis based on DSM-IV TR diagnostic criteria.

Case 2: The subject was 49 year-old, housewife, female patient, married with 3 children. Family history revealed that her mother was a bipolar disorder patient. Psychiatric consultation was requested for the patient, who was receiving inpatient treatment in neurology unit due to occasional headaches, since she had chatterbox syndrome and nervousness. Patient's history taken from her relatives revealed that she received psychiatric treatment previously due to nervousness, distress, talking a lot, and insomnia. She had a clear consciousness, she was oriented and cooperative in his psychiatric examination. The patient lacked insight for the disorder and demonstrated logorrhea, aggression, irritability, and insomnia symptoms. The patient was tentatively diagnosed as BD, hypomanic episode and medication treatment was prescribed as outpatient therapy.
Case 3: The patient was 23 year-old, single, female patient. Family history showed that her maternal grandmother, father and mother were followed up with BD diagnosis. Patient was hospitalized in the neurology unit for her headache and nervousness complaints, psychiatric consultation was requested for the patient due to her absurd speech and behavior, and she was evaluated by our consult service. It was learned that the patient inconsistently used medications for her BD diagnosis. Psychiatric examination demonstrated symptoms such as expansive mood, logorrhea, aggression, insomnia, disorganized speech, and behavior, auditory and visual hallucinations and persecutory delusions. The patient was diagnosed with BD with manic episode due to the existing symptoms and admitted to our inpatient service.
The present case report discussed the bipolar disorder developed in first degree relatives, namely a mother, a father, and their daughter. Genetic predisposition plays a significant role in the etiology of BD. Therefore, it is important to examine the existence of psychiatric disorders in the immediate relatives of patients diagnosed with bipolar disorder.
Keywords: bipolar disorder, mania, genetic predisposition

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[Abstract:0027][ADHD]

Treatment of attention deficit and hyperactivity disorder in visually impaired children: a case report

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Attention deficit and hyperactivity disorder (ADHD) is an early occurring neurodevelopmental disorder that has been reported to persist into adolescence. Problems of focusing and maintaining attention can also be seen in the visually impaired children. There is no information concerning the association of visual impairment and treatment of attention deficit and hyperactivity disorder in the literature. Methylphenidate (MPH) is the most common drug treatment of ADHD in childhood. In treating nocturnal enuresis with ADHD in children, positive effects of methylphenidate were reported.
Cases: An eight year-and-nine-month-old-boy and an eight year-and-nine-month-old-girl, which are visually impaired (congenital) presented to the Marmara School of Medicine's Child and Adolescent Psychiatry Outpatient Clinic. The complaints of boy were school failure, inability to concentrate, rocking movements, and bed-wetting. His parents expressed that he is very clever but he does not like studying and when he concentrates he can be successful at all things. And they added that he wets the bedding most days of the week. He was diagnosed as ADHD and enuresis nocturna based on DSM-IV criteria. After one month of treatment with methylphenidate, his parents expressed that a dramatic improvement in the symptoms of school failure, difficulty in concentrating, and rocking movement. And his bed-wetting frequency per week also decreased. In the second case; her parents reported that she was hyperactive and she has behavioral problems that leads impairments in her life. She was also diagnosed as ADHD per DSM-IV criteria. After the treatment with methylphenidate, her hyperactivity decreased and he began to adapt to the new environment easily.
In this case report, informing the clinicians on not to overlook of attention problems in visually impaired cases and proper treatment of these cases are aimed. And methylphenidate may be tried treatment of enuresis nocturna in children with no response to traditional medications.
Keywords: ADHD, enuresis nocturna, methylphenidate, visual impairment

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[Abstract:0030][Psychopharmacology]

Two cases of priapism associated with quetiapine

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Priapism is a painful, prolonged, and sustained erection without any sexual stimulation. It is an emergency condition that may lead to impotence, urinary retention, and gangrene as long-term consequences. Atypical antipsychotics owing to favorable side effects are increasingly being prescribed and not frequently considered to cause priapism. Quetiapine has been implicated in causing priapism in a limited number of reports. Here, we report two cases of quetiapine-induced priapism.

Case 1: A 49 year-old male patient presented to psychiatry clinic due to complaints of grandiose delusions, insomnia, rapid speech, agitation, and visual and auditory hallucinations for a week. The patient met DSM-5 criteria for a manic episode and hospitalized and initiated a treatment with haloperidol 20 mg/day, biperiden 4 mg/day, and quetiapine 100 mg/day. Within 10 days of quetiapine use, priapism was reported. According to urology consultation the dosage of quetiapine was decreased to 50 mg/day. His priapism still continued in the one week after follow-up examination. Therefore 100 mg/day chlorpromazine prescribed instead of quetiapine. Patient responded well clinically to interruption of quetiapine treatment and his priapism resolved completely within a week..

Case 2: A 22 year old male-patient with the history of unipolar depression for 1.5 years was hospitalized for his suicidal ideations and attempts. In personal medical history, there was a spongio-cavernosal shunt surgery as a treatment of recurrent priapism. The patient met DSM-IV criteria for major depressive disorder with psychotic features; was started the treatment of quetiapine 50 mg/day and electroconvulsive therapy (ECT) was planned. On the sixth day of quetiapine treatment, priapism occurred. Following the urology consultation, 25 mg/day chlorpromazine was started instead of quetiapine and increased to 200 mg/day. He responded well to the discontinuation of quetiapine and his priapism resolved completely within a week.

Quetiapine has been suggested in causing priapism in a limited number of case reports. In one case, a 25 year-old African-American male, who was taking 300 mg/day, with no other factors or drugs known to predispose this side effect was reported to have priapism. Another case occurred in a patient who had taken 675 mg quetiapine as a suicide attempt. In both of our cases; quetiapine which is known to have antagonistic activity both alpha-1 and alpha-2 receptors caused priapism. Priapism may be considered as an idiosyncratic reaction which is correlated with neither the dosage nor the duration of antipsychotics. In one report, a 44 year-old man with a 23 year of schizophrenia developed priapism previously with combination of risperidone 8 mg/day and trazodone. After switching to quetiapine 600 mg/day priapism occurred again within 24 days. Finally with olanzapine 20 mg/day within 53 days priapism developed. In our first case, priapism occurred within 10 days with 100 mg/day quetiapine; and it continued although the decreased dosage of quetiapine as a consequence of idiosyncratic reaction. We recommend clinicians that it is important to inform the patient about the occurrence of this side effect and monitor patients taking antipsychotics. Monitoring should include asking sexual side effects of antipsychotics. The clinician should be aware of the history of prolonged and painless erections as a predictor of priapism.

Keywords: priapism, quetiapine, antipsychotics

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[Abstract:0033][Psychopharmacology]

Paliperidone palmitate -induced tardive dystonia: a case report

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Paliperidone palmitate is an atypical long-acting injectable antipsychotic designed for once-monthly intramuscular injection for the treatment of schizophrenia and schizoaffective disorder. Tardive dystonia (TD) is delayed side effect, reported with the use of

antipsychotics. The specific pathophysiologic processes underlying movement disorders still remain poorly understood. Dystonic reactions are variable in location and severity and are occasionally painful. We report a case of TD following the administration of antipsychotic, which was improved by paliperidone palmitate.

Case: A 24 year-old male patient was hospitalized to the Department of Psychiatry, Konya Training and Research Hospital for increasingly aggressive and disruptive behavior, irritable mood, and persecutory delusions. He has been diagnosed as having schizoaffective disorder since 2013. Lithium (600 mg/day) and paliperidone palmitate (150 mg eq) was started. The patient improved clinically and was discharged on day 20. He had developed persistent right-side torticollis, muscular pain within 5 months of treatment. There was no history of head trauma, seizure, medical disease, and family history of neuropsychiatric disorders. Biperiden (4 mg/day) and diazepam (10 mg/day) were prescribed to treat his dystonia, but these were not effective. The Abnormal Involuntary Movement Scale (AIMS) score was 14. We administered clozapine (12.5 mg/day) for the treatment of tardive dystonia and gradually increased to 300 mg/day. Lithium and paliperidone palmitate was stopped. After 4 months, clozapine was started and dystonic symptom improved. His AIMS score decreased to 6.

TD occurs with longer exposure to antipsychotics, with the Diagnostic and Statistical Manual specifying a minimum duration of 3 months. Drug-induced tardive dystonia is difficult to treat and persist in many patients. Stopping the offending agent alone is shown to be insufficient to resolve TD. If continued antipsychotic therapy is necessary, clinicians should consider switching the antipsychotic medication to clozapine. Clinicians should consider the possibility of the development of tardive dystonia when used antipsychotics.

Keywords: paliperidone palmitate, tardive dystonia, schizoaffective disorder

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[Abstract:0034][Schizophrenia and other psychotic disorders]

Interferon-β-1a-induced psychosis in a patient with multiple sclerosis

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Multiple sclerosis (MS) is the most common demyelinating disease. Psychiatric symptoms are common in MS. Psychosis is often seen in the late stages of the disease. Interferons are a class of peptide that regulates functions of immune system and has antiviral effects. Rebif is a form of interferon-β-1a which is administered as an injection under skin three times a week. FDA reports the number of patients with any side effect of Rebif was 34,420. The number of patients with acute psychosis with Rebif was 11 in FDA reports. Average percentage for all medicated patients where acute psychosis is reported as a complication was 0.0052%. Average percentage of acute psychosis as a complication in patients with using Rebif was 0.032%. Here, we report a patient with psychotic symptoms associated with interferon-β-1a therapy for multiple sclerosis. 47 year-old male patient presented to our psychiatry outpatient unit with symptoms of paranoid, persecutory, reference, and somatic passivity delusions. He thought that his car and home are monitored by cameras and his neighbours planted a voice recorder to his house. He had no psychiatric history. His laboratory studies were in normal limits. It was learned that psychotic symptoms of the patient occurred after he started using Rebif® (interferon-β-1a) for multiple sclerosis. His psychotic symptoms resolved with discontinuation of Rebif and initiation of risperidone. Depression is highly seen in patients using Rebif. Otherwise, somnolence and suicidal thoughts are frequently seen with using Rebif. Development of acute psychosis with using Rebif is a serious side effect and this may worsen treatment adherence. Rebif should also be avoided in patients with psychosis because of this drug could worsen or exacerbate psychosis. Rebif can also stimulate psychosis in patients without a history of psychotic disorders. So it is important that patients who are under interferon beta-1a for multiple sclerosis should be screened for psychotic symptoms.

Keywords: interferon-β-1a, multiple sclerosis, psychosis

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[Abstract:0036][Psychopharmacology]

Venlafaxine -induced bruxism: a case report

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Bruxism is an unconsciously or involuntary grinding of the teeth which may lead to jaw pain, muscle contraction headaches, periodontal diseases, weaking of teeth, and temporo-mandibulary articular dysfunction. It may be associated with drugs. SSRIs and venlafaxine have been reported to cause bruxism. Here, we present a bruxism case that was reported after venlafaxine treatment. The patient was grinding his teeth during sleep at night and he had discomfort due to jaw pain. Clinicians might consider that venlafaxine might induce bruxism in some patients. Bruxism is a disorder described as excessive activity that results from clenching and grinding of the teeth and certain strong jaw movements. 85-90% of the population experience bruxism at anytime during their lives, but it is clinically significant in only 5% of them. It can result in temporomandibulary articular pain and periodontal serious damage. Bruxism can occur in association with anxiety, benzodiazepines, use of alcohol, selective serotonin reuptake inhibitors (SSRIs), and venlafaxine. Although pathophysiology of bruxism is unknown, it has been suggested that serotonergic action on mesocortical neurons arising from the ventral tegmental area may lead to dopaminergic deficit. We hereby report a case of bruxism induced by venlafaxine.

Case: Mr. X., a 33 year-old male patient, working in a supermarket. The presenting complaints were feeling bad, poor sleep, anxiety, poor concentratione, loss of interest in work, and unhappiness. He also reported irritability and aggression. He was admitted to a psychiatry clinic and was diagnosed with major depressive disorder. The patient was started 37.5 mg/day venlafaxine. Dosage was increased gradually to 150 mg/day at the 4th day of medication. 5 days after the onset of medication, the patient reported that he was grinding his teeth during sleep at night and he had discomfort because of jaw pain. Therefore, treatment was discontinued on the suspicion that the bruxism might be related to venlafaxine. Paroxetine was started at the dose of 20 mg/day. Following discontinuation of venlafaxine, bruxism was reduced gradually and after 15 days it completely resolved. He was observed by a home health nurse for 3 days with no relapse of bruxism.

Bruxism can lead to periodontal diseases, weaking of teeth, temporomandibulary articular dysfunction, and headache. Drug related dopaminergic and serotonergic systems can induce bruxism. Venlafaxine is a serotonin reuptake inhibitor at lower doses but has increasing effect of dopamine reuptake inhibition especially with higher dosages. It is not certain the time range of the onset of medication-induced bruxism. In conclusion, while starting venlafaxine, it should be remembered that it may cause bruxism, and the signs of bruxism should be asked during psychiatric treatment.

Keywords: Venlafaxine, bruxism, SSRI

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[Abstract:0038][Dependencies]

Oxybutynin abuse in an adolescent leading to psychotic symptoms: a case report

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Drug abuse is defined as either recurrent use of illicit drugs or the misuse of prescription or over-the-counter drugs leading to negative consequences in multiple domains of functioning. Among the prescription drugs; anticholinergic abuse have been reported since 1980s; primarily among adult patients with serious, chronic mental disorders. Oxybutynin is an anti-cholinergic agent used in treatment of enuresis, frequent urination and urge incontinence by decreasing muscle contraction in bladder. Post-marketing experience revealed psychotic symptoms and hallucinations as adverse effects of oxybutynin use. However, to the best of our knowledge there are no reports of chronic adolescent abuse of oxybutynin leading to psychotic symptoms. Here, we report a male adolescent who chronically abused oxybutynin and developed psychotic symptoms; presumably due to abuse.

Case: A 17 year-old male patient presented with the anxiety symptoms. He was referred to the Department of Child and Adolescent

Psychiatry by pediatrics services because of the history of drug abuse. According to the history taken from the patient and his mother, he started to use Uropan® tablets by the advice of his friend to get high a year ago. He used to take Uropan® tablets two or three times a week but he needed dose escalation to catch the euphoria that he acquired from the previous usages.
In his mental status examination; the patient appeared at his stated age and was well-groomed and well-dressed. He did not have difficulty in making eye-contact, looked eager to communicate, was articulate, and answered questions spontaneously. His thought processes were coherent and goal directed. His thoughts included themes about fear of looking at the mirrors and fear of being left alone at home. His affect and mood were anxious. He was alert and oriented to time, person, and place. He appeared to maintain attention and concentration on the interviewer's tasks and questions. His past and recent memory was intact. He reported some visual and auditory hallucinations after taking Uropan®, but his intellectual functioning appeared to be normal. According to the criteria of the DSM-5, other (or unknown) substance use disorder was diagnosed. Risperidone 1 mg/day was started for hallucinatory symptoms.
Anticholinergic drugs are muscarinic receptor antagonists that suppress the activity of the cholinergic system in the brain. It is considered M5 mAChR subtype plays the major role in the field of drug dependence and addiction. M5 mAChRs display a very discrete CNS localization in the ventral tegmental area (VTA), a brain region known to be involved in drug reward and addiction. Anticholinergic drugs have a number of neuropsychiatric effects, such as decreasing depressive-anxiety symptoms, causing euphoria, improving relaxation, ameliorating parkinsonian symptoms and reducing the side-effects of antipsychotic treatments. The drugs, which have abuse potential are well-known by adolescents. Although most of anticholinergic medication is controlled, Oxybutynin could be obtained from pharmacies without prescription in Turkey. The medicolegal arrangements about the drugs including anticholinergic agents should be made promptly to prevent the drug abuse.
Keywords: abuse, oxybutynin, adolescent, psychotic symptoms

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[Abstract:0041][Psychopharmacology]

Methylphenidate -induced hallucinations in a 9 years-old female patient

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Methylphenidate is a psychostimulant, which is used for attention deficit and hyperactivity disorder (ADHD) in children and adolescents as first-line pharmacological treatment. The most common side effects of psychostimulants are the loss of appetite, insomnia, weight loss, abdominal pain, and headache. There are reports that hallucinations may appear when methylphenidate is used alone or in conjunction with other drugs. Here, we report a 9 year-old girl with attention deficit hyperactivity disorder who had visual hallucinations during methylphenidate treatment.
Case: A 9 year-old girl who was an elementary school 4th grade student had been introduced to our clinic by her family because of her teacher's advice. She had symptoms like hyperactivity, restlessness, and irritability since her younger ages. Also, her family was warned by her teacher because of her not listening lessons, not doing her homeworks, and unable to get along with her friends. There were no problems related to her mental status, affect, and perception except hyperactivity and attention problems. The diagnosis of hyperactivity and attention disorder - combined type according to DSM-5 criteria. The Parents and Teacher Conners scale scores also supported this diagnosis. The patient's sleep and appetite were normal. There was no violence and abuse history in her family. Her psychomotor development was normal too. 30 mg long-acting of methylphenidate was started. After the treatment, the patient showed no improvement in symptoms of ADHD. On the second week, the patient started to feel sensations especially at night such as discomfort, night terrors, seeing bugs on the walls and in the bed and feeling them in her hair. She was trying to extract herself from insects continuously. She began to sleep with her mother because of extreme fear. Her family had ceased the drug due to these side effects at the end of the 2nd week. The patient's visual hallucinations disappeared with the discontinuation of the treatment. The result of the re-examination and routine laboratory tests showed no abnormal findings.
There were no psychotic or affective disorder patients in patient's family history. Also, her routine laboratory tests that might have caused hallucinations were in normal ranges. Besides all these, during the patient's hallucinations, the absence of another drug intake, toxicity and infection findings suggested that hallucinations were thought to be associated with the use of methylphenidate. The literature reveals several case reports regarding the development of hallucinations with methylphenidate treatment. The mechanism underlying this side effect is not clear yet. Young has proposed two hypotheses regarding hallucinatory effect of methylphenidate. First one proposes

a simplification of noradrenergic transmission in optical pathways and the second one suggests interactions with monoamine systems. It was also reported that it might be related to synaptic dopamine elevations. In conclusion, clinicians must be aware of hallucinations that may appear during methylphenidate treatment of which the cause is unknown.
Keywords: methylphenidate, attention deficit and hyperactivity disorder, hallucinations

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[Abstract:0043][Psychopharmacology]

Pramipexole-induced hypersexuality: a case report

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Pramipexole is a synthetic aminothiazole derivative dopamine agonist, which is commonly used in the treatment of various diseases such as Parkinson's disease and restless leg syndrome. Abnormal behavior such as excessive eating, compulsive shopping, hypersexuality, and pathological gambling could be observed after Pramipexole use in Parkinson's patients, albeit quite rare. The present article addresses a hypersexuality case developed in a Parkinson's patient due to Pramipexole use.
Case: A 65 year-old, married with 7 children, retired male patient. He presented to our psychiatry outpatient clinic with complaints of malaise, sadness, anhedonia, increase in libido, nervousness, and distress. Personal history revealed Parkinson's disease, hypertension, arrhythmia, and benign prostatic hypertrophy. The patient, who had presented to the neurological outpatient clinic three months ago with complaints of tremors in hands, lips, and the tongue was diagnosed with Parkinson's disease and was started on Pramipexole (0.25 mg initial dose and the dose was gradually increased to 1.5 mg, however the patient did not follow the advice of the physician and took 0.25 mg one day and 1.5 mg in the next day). The patient, who previously had sexual intercourse once a week, three weeks after he started using 1.5 mg Pramipexole developed an intense sexual desire to have an intercourse everyday, and started to watch erotic movies, for which he did not show interest before. In addition to these urges, he demonstrated autonomous symptoms such as perspiration, palpitation, and breathing heavily. In his psychiatric examination, depressive mood, anxiety, increase in libido and irritability findings were identified. In the neurological examination, bradymimia, resting tremor in the right hand, tremors of the jaw and the tongue, cog-wheel rigidity on the right side, bradykinesia, and reduction in associated activities on the right arm during walking were observed. There was no postural instability. To exclude organic factors that could lead to hypersexuality, endocrinology and urology consultations were requested. Examinations conducted by the related departments did not reveal any factors that could explain hypersexuality, apart from the drug he was on. The patient was diagnosed with impulse control disorder due to Pramipexole. In consultation with the neurology specialist, Pramipexole was replaced by Ropirinol. Hypersexuality complaints started to decrease by the 4th day of the termination of Pramipexole and disappeared completely on the 7th day.
It was speculated that dopaminergic drugs used in Parkinson's patients activate ventral striatum, which contains extensive number of D3 receptors, and cause hypersexuality through mesocortical and mesolimbic pathways. Since impulsive behavior such as hypersexuality, compulsive shopping, hyperphagia could develop due to dopaminergic drugs intake, albeit quite rare, patients and relatives should be informed about such adverse effects, and patients should be questioned for these symptoms during follow-ups. If this condition is not diagnosed, it could result in detrimental situations for the patients and their partners. This condition, which could be diagnosed only by questioning and could be treated by simply taking the patient off the drug, should not be neglected in clinical care.
Keywords: pramipexole, hypersexuality, impulse control disorder

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[Abstract:0047][Schizophrenia and other psychotic disorders]

Hyponatremia cases induced due to psychogenic polydipsia in a patient with schizophrenia

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Polydipsia is consumption of high amount of water, more than 3 liters per day and hyponatremia due to polydipsia can be seen. Rapid decrease in sodium levels can cause to neurological symptoms, coma, and death in severe cases, this is called as water intoxication. Polydipsia can be seen in 20% of schizophrenia patients with chronic illness, its pathology is not known with certainty despite it is common. Polydipsia is a dangerous, deadly condition that is often overlocked by physicians since the actual consumption of water is not understood due to communication problems with patients. The treatment of hyponatremia due to polydipsia is difficult, the most effective method is to restrict fluid intake but the patient and its relative may need to be consistent and psychiatric unit treatment may be required. Demeclocycline, propranolol, captopril, and antipsychotics may be used pharmacologically, but the role of antipsychotics in the treatment of polydipsia is controversial. In this case report, a hyponatremia case in a 25 year-old male patient currently in remission using olanzapine 10 mg PO BID, quetiapine 300 mg PO daily and with diagnosis of schizophrenia for 6 years is presented. Polydipsia was treated with fluid restriction successfully in compliance with the treatment, with effective communication between the patients and relatives. Diagnoses such as tumor and diabetes were excluded in differential diagnosis and hyponatremia was developed due to 9-10 liters water consumption per day.
As a result, since hyponatremia with polydipsia is common in patients with schizophrenia and as it can be fetal, every patients should be questioned and biochemistry laboratory test should be performed in these patients at regular intervals.
Keywords: schizophrenia, polydipsia, hyponatremia

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[Abstract:0048][Psychopharmacology]

Akathisia growing due to escitalopram usage

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Akathisia is characterized by internal unrest and the desire to act, and it is often a serious side effect associated with the use of typical antipsychotics. However it can be also observed due to the use of atypical antipsychotics and antidepressants. Diagnosis of akathisia symptoms by the physicians can be skipped because they are subjective. Akathisia is a side effect that must be treated immediately due to disruption of treatment compliance and due to the increased risk of aggression and suicide. It can be seen as acute, tardive and cutting akathisia. Since akathisia is the side effect of extrapyramidal system, its mechanism is not known. Akathisia is limited only by the case reports due to the use of Selective Serotonin Reuptake Inhibitor (SSRI) in the literature. It has been proposed that SSRI' s can cause akathisia by reducing the basal firing rate of dopaminergic neurons in the ventral tegmental area. Risk factors for developing akathisia with antidepressant use can be counted as sudden increase in SSRI dose, history of akathisia, and previous head injury. Akathisia can be treated by adding beta blocker or benzodiazepine to the treatment or by switching to another antidepressant.
Case: A 41 year-old male patient, who is presenting with depressive symptoms such as anhedonia and reluctance, diagnosed with mild depression, started with escitalopram 5 mg/day and increased to 10 mg/day at the 5th day and developed a drug-induced akathisia in the 3rd week of treatment with escitalopram. Akathisia was treated successfully with venlafaxine treatment by stopping the escitalopram. Akathisia due to antidepressant use are rarely seen and it is believed that physicians should bear in mind akathisia

in all patients taking psychotropic and they should treat them immediately since it increases the risk of suicide in depressed patients and it can disrupt the treatment compliance.
Keywords: akathisia, escitalopram, depression

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[Abstract:0050][Mood disorders]

Major depressive disorder with psychotic features associated with menstrual cycle

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It is known that sex hormone rises during the menstrual cycle are associated with the increase in exacerbation and complaints in many psychiatric diseases. But the data on these issues is associated more with premenstrual dysphoric disorder and there is not sufficient evidence to show that it takes part in the pathogenesis of diseases such as major depressive disorder with psychotic features and menstruation psychosis. There are no data in the literature except for a few case reports of menstrual cycle-related psychosis. Symptoms in the psychotic associated with menstruation are typically starts a few days before menstruation and ends with the termination of bleeding. Next month the same clinical manifestations repeated, patient was asymptomatic between the menstruation period and it was not easy to predict how long this situation would continue.
Case: A 35 year-old female patient, presented with demoralization, reluctance, fatigue, suicidal thoughts, fear of damage, auditory hallucination, loss of appetite, restlessness, and agitation complaints associated with menstruation for 7-8 years. It repeats a similar clinical period which takes about 5-6 days each month, and complaints were disappearing after the menstruation. She has been treated in the past with no resolution. Patient reported that she was comfortable during the pregnancy since the complaints were not repeated. Consultation for gynecology and obstetrics were requested and no pathological findings were found in her physical examination and in sex hormone levels. Bipolar disorder, premenstrual dysphoric disorder, and schizophrenia diagnosis were excluded. The patient was diagnosed with major depressive disorder with psychotic features associated with menses since clinical condition was associated with the menstrual cycles. She was treated with paroxetine 20 mg daily and olanzapine 5 mg daily and major depressive disorder with psychotic features clinic was not seen in the follow-up exams. In sum, there is a need for extensive search in such patients in order to evaluate the role of sex hormones in the development of psychiatric disorders.
Keywords: psychosis, depression, menstrual cycle

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[Abstract:0051][Others]

Enuresis and encopresis due the use of aripiprazole

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Aripiprazole is an antipsychotic with partial dopamine agonist, it is used in the treatment of psychotic disorders, Mood disorders, and anxiety disorders. Side effects such as tremor, insomnia, akathisia, and nausea were reported, but drug-induced enuresis are very rare and there is insufficient information in the literature on this subject. Although it is not known clearly how this rare side effect ensues, it may be suggested the role of decreased internal bladder sphincter tone due to alpha-1 adrenergic blockade induced by antipsychotics,

decreased dopamine transmission in the basal ganglia, blockage of the pudendal reflex, sedation caused by the activation of detrusor muscle, but it is not known how encopresis occurs and the mechanisms of encopresis can be explained by the blockage of pudendal reflex similarly. Although rare enuresis cases due to arpiprazole have been reported, cases that are seen together enuresis and encopresis due to aripiprazole have not been reported before. Here, we present a 28 year-old female patient who is using quetiapine XR-300 daily for mild mental retardation and enuresis and encopresis occurred on the 3rd day after addition of 5 mg daily aripiprazole, continued for 5 days, and rapidly improved after 1-2 days following the discontinuation of aripiprazole. There were no neurological and urological pathology in the patient's medical history, EEG, USG, and urinalysis were all normal, when it is questioned it is further strengthened our diagnosis that the side effect seen before while they are using the same drug. As a result, it disrupted the compliance of the treatment because enuresis and encopresis due to the usage of antipsychotics are seen rarely and it is a serious adverse effect, due to facct that fecal and urinary incontinence especially during the day can impair the person's social relationship. Further research is needed for this rare adverse effect.
Keywords: enuresis, encopresis, aripiprazole

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[Abstract:0052][Sleep disorders]

The diagnostic process of a patient with sleep paralysis and hypnogogic/ hypnopompic hallucinations misdiagnosed as epilepsy

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Hypnagogic and hypnopompic hallucinations are prolonged episodes of dream-like imagery occur at the transition from wakefulness to sleep (hypnagogic) or from sleep to wakefulness (hypnopompic). Hypnagogic/ hypnopompic hallucinations are hypothesized to be related with rapid eye movement (REM) sleep intrusion into the wakeful state. They can be experienced by individual in various sensory modalities like visual, tactile, and auditary or a combination of all. Sleep paralysis is defined as a transient conscious inability to perform voluntary movements, typically arising during the transition between wakefulness and rapid eye movement (REM) sleep. Misdiagnosis of epilepsy is common. The dream-like behavior and cataplexy in narcolepsy is often misdiagnosed as epileptic seizure. Here, it has been aimed to report a patient with sleep paralysis and hypnagogic/ hypnopompic hallucinations clinically resembling psychomotor epilepsy.
Case: A 27 year-old female patient presented the tactile, visual and auditory halucinations. The hallucinations recur while she was falling asleep, she reported her hallucinations; frightening perceptions of alien figures, felt arms and legs holding her and felt like she was raped by these aliens. Limb-muscle atonia accompanied by hypnogogic and hypnopompic hallucinations. Also she described excessive daytime sleepiness and she often had irresistible naps for about 3 to 4 times per day. Each nap lasted for 30 minutes to 45 minutes. She presented a neurologist with these complaints two years ago and antiepileptic treatment was started by the neurologist. Despite the dose increase of antiepileptics, her complaints were non-responsive to the antiepileptic treatment. So she quit the treatment and presented to our clinic. Neurologic examination and brain magnetic resonance imaging were unremarkable. The patient was not taking any medications at that time. The limb-muscle atonia was assessed as an sleep paralysis and the hallucinations assessed as an hypnogogic/ hypnopompic hallucinations. She was administered further investigation tests including polysomnogram (PSG) followed by a multiple sleep latency test (MSLT) for the initial diagnosis of narcolepsy. We are waiting the results of tests for the diagnose.
Narcolepsy is characterized by excessive daytime sleepiness, cataplexy, sleep paralysis, hypnagogic, and hypnopompic hallucinations. Narcolepsy is considered to be caused by a complex interaction of genetic and environmental factors. The clinical symptoms of narcolepsy may overlap with other neuropsychiatric symptoms. Incorrect diagnosis of epilepsy may lead to inappropriate investigation and treatment. The differential diagnosis between epilepsy and narcolepsy has attracted the attention of both psychiatrists and neurologists. History taking, included sleep-related symptoms can be useful.
Keywords: epilepsy, hypnogogic/hypnopompic hallucination, narcolepsy, sleep paralysis

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[Abstract:0053][Mood disorders]

Mania following temporal epileptic seizure: a case report

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Psychiatric symptoms are observed in patients with temporal lobe epilepsy frequently. Depression is seen in temporal lobe epileptic seizures more than other psychiatric disorders. However, literature is limited regarding postictal mania in these patients. This case report describes a 36 year-old male patient with temporal lobe epilepsy, who developed manic symptoms during postictal period. The patient, with no history of any psychiatric disorders, had suffered from epilepsy since age 1. There were no abnormalities in the neurologic examination. MRI and routine laboratory tests were unremarkable. Postictal EEG pattern typically showed epileptiform activity and sporadic spikes on left temporal lobe. Clinicians might consider that manic symptoms may occur in postictal period. Epilepsy is one of the most common of chronic neurological disease. Patients with epilepsy may experience some of psychiatric and cognitive symptoms or behavioral manifestations after the seizures. Postictal psychiatric symptoms are commonly observed and include confusion, aggression, psychosis, and mood changes like depression or hypomania/mania. Psychiatric symptoms were seen in temporal lobe epileptic seizures more than other types of seizures. Here, we report a case with temporal lobe epilepsy associated with manic symptoms.

Case: Mr. X., a 36 year-old, married, male patient, with no history of any psychiatric disorders, had suffered from epilepsy since age 1 and carbamazepine 800 mg/day was prescribed. There were no convulsive and psychiatric disorders in his family history. Approximately 3 days after epileptic seizure had occurred, his wife took him to the emergency department with complaints of insomnia, impaired awareness, and disorganized speech. Neurological examination revealed no abnormalities. The brain MRI and routine laboratory tests (serum electrolytes, including total blood count, kidney and liver function tests) were unremarkable. He was admitted to the neurology service with diagnosis of complex partial seizure and the dose of carbamazepine was gradually increased to 1200 mg/day. Post-ictal EEG pattern typically showed epileptiform activity and sporadic spikes on the left temporal lobe. Psychiatry consultation was requested. Irritability, psychomotor agitation, logorrhea, decreased need to sleep, persecutory delusions, flights of ideas, lack of appetite, a feeling of grandiosity, and distractibility were noted in the psychiatric examination. Following psychiatric consultation, the patient was diagnosed with manic episode based on the diagnostic criteria of DSM-IV-TR and he was admitted to the psychiatry inpatient service. His YMRS (Young Mania Rating Scale) score was 26 that supported the diagnosis. Clonazepam 1 mg/day was added to the carbamazepine 1200 mg/day treatment. He had significant recovery after 1 week of treatment when he was discharged from the hospital.

Patients with epilepsy may experience some of psychiatric and cognitive symptoms or behavioral changes after the seizures. Recognition of psychiatric symptoms in patients with epilepsy might contribute to treatment and quality of life in these patients positively.

Key words: epilepsy, mania, temporal lobe

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[Abstract:0055][Psychopharmacology]

Type 2 second-degree atrioventricular block due to carbamazepine: a case report

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Carbamazepine blocks voltage-dependent sodium channels, reduces neural firing, glutamate release, and decreases turnover of dopamine and noradrenaline. It has a molecular structure similar to tricyclic antidepressants. Carbamazepine is widely used for the treatment of grand mal and focal seizures, bipolar disorders, and trigeminal neuralgia. Besides this highly effective in aggressive patterns that can be seen in personality disorders, affective disorders and schizophrenia. Neurologic, cardiovascular, hepatic,

hematological, and dermatological side effects have been reported in the therapeutic use of carbamazepine. The cardiovascular well-known side effects are hypotension, bradycardia and atrioventricular (AV) block. AV conduction delays have rarely been reported predominantly in elderly women, during the course of routine treatment. Here, we present a case with an AV block during carbamazepine treatment.

Case: 25 year old female inpatient was evaluated during inpatient psychiatry consultation in the cardiology clinic. She presented with recurrent syncopes. In her electrocardiogram there was a type 2 second-degree AV block with no other cardiologic abnormalities. In clinical examination any other side effect of carbamazepine have not been revealed. She was using olanzapine 2.5 mg/day, venlafaxine 75 mg/day and carbamazepine 1200 mg/day. In her past story she had major depressive disorder episodes with multiple suicidal behaviors. In psychiatric examination her mood was euthymic and she was in remission state. She had disinhibition problems with aggressive and suicidal behaviors. Diagnosis was recurrent unipolar depression according to the DSM-IV. Due to AV block effect of carbamazepine, it was excluded from treatment gradually and no other medication added to her treatment. Naranjo Adverse Drug Reaction Scale score was 4 which means adverse effect was probably related to carbamazepine. After 17 days of hospitalization in cardiology clinic the patient was discharged with normal sinus rhythm and with euthymic mood and did not have any suicidal thoughts. Unfortunately, after that, she had no psychiatric admission.

Carbamazepine decrease atrial ventricular conduction and is relatively contraindicated in patients with heart block. There are several reports of undesirable effects of carbamazepine on the cardiac conduction system. These effects have been reported especially in older patients and cardiac conduction evaluation has been suggested before the initiation of carbamazepine treatment. In a literature review, almost exclusively of elderly women who developed potentially life-threatening bradyarrhythmias or atrioventricular conduction delay, associated with either therapeutic or modestly elevated carbamazepine serum levels, was seen. In this case report, we can not say anything about the relationship between plasma level of carbamazepine and AV block due to absence of carbamazepine level measurement. Adverse effects of psychotropics are usually considered when prescribing in elderly patients and in the follow-up of these patients. But in this case we report a young patient without any cardiac risk factors.

Like in this case, cardiac adverse effects of carbamazepine should be kept in mind in young patients and routine cardiologic examination should be considered to check to see if there is any risk factor or any other cardiologic symptoms.

Keywords: carbamazepine, adverse effect, atrioventricular block

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[Abstract:0056][Psychopharmacology]

Quetiapine-induced bradycardia without QT

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Hypotension is usually seen with quetiapine and bradycardia may also be a severe adverse effect of it. Since bradyarrhythmogenic mechanism of quetiapine has not been fully understood, QTc interval prolongation is usually related to bradycardia. Cardiac actions of quetiapine are suggested to be within the central nervous system and the bradycardia may be mediated by vagal stimulation at the periphery. It is known that afferent cardiac branches of the vagus nerve may cause a tonic inhibition. It reduces sympathetic output and peripheral vascular resistance leading to bradycardia and continued hypotension. It is important to note that hypotension with concomitant bradycardia may be a true medical emergency and hospitalization is usually needed for detailed investigation of the underlying causes. Here, we report a case who suffered from hypotension and bradycardia unrelated to QT interval prolongation with small dosages of quetiapine.

Case: A 31 year-old male patient was hospitalized with the complaints of nervousness, insomnia, and anger management problems. He had been using 1 mg of risperidone for one month and 150 mg dosage of quetiapine was added to the treatment regimen. There was no prior history of any other medical disease. At physical examination a blood pressure of 110/80 mm Hg without orthostasis and a regular pulse rate (80 bpm) were found. After two hours of quetiapine 150 mg was given, he complained of vertigo and dizziness. His blood pressure was 70/40 mm Hg. The electrocardiogram showed sinus bradycardia at 40 beats per minute. QT and QTc intervals were 434 ms and 426 ms respectively. His routine hematological and biochemical parameters were within normal limits. He was followed at the emergency service. After six hours his pulse rate was 60 bpm. His blood pressure was 110/70 mm Hg. QTc prolongation has never occurred. Quetiapine was stopped and the same reaction was not observed again. The Naranjo Probability Scale score was 4 and there was a probable association between quetiapine and bradycardia and hypotension without QTc prolongation. In the literature there is

another paper reporting a relationship between high dosage of quetiapine and bradycardia unrelated to QT interval. But as far as we know, our case is the first showing the possible adverse effect with only one and low dosage of quetiapine in a young patient. Although quetiapine is known as a safe drug, the clinicians should be aware of possible cardiac side effects and routine ECG monitorization should be applied.

Keywords: bradycardia, hypotension, quetiapine, without QTc prolongation

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[Abstract:0059][Psychopharmacology]

Maculopapular rash and pruritus induced by mirtazapine: a case report

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Mirtazapine is an antidepressant with serotonergic and noradrenergic activities. In addition to Mirtazapine's powerful presynaptic alpha-2 adrenergic antagonistic activity, there are weak 5-HT1 and powerful 5-HT2, and 5-HT3 antagonistic actions. Due to its effects on histamine receptors, mirtazapine was tried on cancer patients with pruritus complaint and was reported as effective. In the present article, a case with rash complaints followed by pruritus induced by mirtazapine, which were ameliorated after discontinuation of the medication is discussed.

Case: The patient was a 29 year-old, female, married with one child. With antiphospholipid antibody syndrome and systemic lupus erythematosus diagnoses in her history, she was an inpatient in rheumatology clinic due to development of avascular necrosis. Due to the onset of headaches, asthenia, insomnia, and nausea, which do not respond to medicines and could not be explained by organic reasons, psychiatry consultation was requested. The patient was on Lansoprozole 30 mg daily, Methylprednisolone 8 mg daily, Vancomycin 2 gr I.V. daily, Hydroxychloroquinine 400 mg daily, Warfarin 2.5 mg daily, Dexketoprofentrometamol I.V. daily, and Indomethacin 50 mg supp daily. The examination of the patient who did not receive any psychiatric treatment before demonstrated depressed mood, anhedonia, insomnia, anxiety, anergia, and somatic complaints of anxiety. The patient was pre-diagnosed with depression due to organic condition and mirtazapine 15 mg/day was started. Generalized pruritus on right arm on the first day of mirtazapine use, erythematous maculopapular lesions with generalized pruritus on the whole body on the second day were identified and dermatology consultation was requested to exclude a possible organic factor. Tests and physical examination showed that pruritus complaints and skin lesions could be maculopapular medicine eruption due to mirtazapine. Mirtazapine was considered as responsible agent and discontinued, and symptomatic drug treatment was started. The patient was examined again and it was observed that on the second day following the discontinuation of the drug the rash resolved and disappeared completely after five days.

Antidepressant medications can cause dermatologic adverse effects, albeit quite rare. These adverse effects should be taken into consideration since they would deteriorate the patient's quality of life and could cause cosmetic problems for the individuals.

Keywords: mirtazapine, maculopapular lesions, pruritus

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[Abstract:0061][Psychopharmacology]

Citalopram-induced galactorrhea in an adolescent

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Citalopram, a specific serotonin reuptake inhibitor (SSRI), is considered as the first line agent in the treatment of depressive disorders, anxiety disorders and, obsessive compulsive and related disorders and has common side-effects such as nausea, vomiting, dyspepsia, insomnia, anxiety, and diarrhea. However, in the last few decades, few case reports of galactorrhea with SSRIs have been reported with

fluoxetine, paroxetine, fluvoxamine, and sertraline. Here, we present an adolescent girl who developed galactorrhea after treatment with citalopram for her depressive symptoms.

Case: A 14 year-old female patient was referred to our clinic by a pediatrician with a complaint of sleep problems. In her psychiatric assessment, the girl reported sadness, hopeless, helpless, worthless, guilt feelings, irritability, loss of interest in activities, and concentration problems. Her Children's Depression Inventory score was 35. The patient was diagnosed as having major depressive disorder according to the DSM-5 criteria and was prescribed citalopram 20 mg/day and showed significant improvement in her depressive symptoms at a follow-up exam 4 weeks later. After 5 weeks of continuing with citalopram treatment, she started experiencing discomfort, pain, and engorgement in her breasts. She noticed whitish milky discharge from bilateral nipples. Patient was extensively evaluated for the galactorrhea, and her serum prolactin (PRL) levels were found to be raised (71 ng/ml). She consulted her endocrinology and gynecologist and minimal ductal dilatation was revealed in mammography testing and breast ultrasonography. MRI of the pituitary, laboratory tests, including hemogram, liver, thyroid, and kidney function tests, and a beta-HCG, FSH, LH, dehydroepiandrosterone-sulfate were found to be within normal limits. In this case, citalopram-induced galactorrhea was a clear association with patient's presentation and temporal correlation with pharmacotherapy initiation. Subsequently, citalopram treatment was stopped and and venlafaxine was started at the dose of 75 mg per day and the dose was increased to 150 mg per day within 4 weeks. After discontinuation of the citalopram, the discharge reduced and breast pain was relieved and were absent after 15 days of stopping citalopram. At the same time, serum prolactin levels was 12.50 ng/ml. After 8 weeks, the patient's depressive symptoms had completely resolved.

In recent years, as the use of SSRIs increases, clinicians can anticipate to observe uncommon side effects such as extrapyramidal symptoms, hyperprolactinemia, and galactorrhea. One possible mechanism is the through serotonergic activation of PRL-releasing factors such as thyrotropine-releasing hormone and gamma-aminobutyric acid via GABA interneurons. As second possible mechanism, serotonin may mediated by serotonergic inhibition of PRL inhibitor factors such as dopamine or indirectly through vasoactive intestinal peptide and oxytocin release. Serotonin may stimulate prolactin elevation directly via postsynaptic 5-HT receptors in the tuberoinfundibular dopaminergic neurons.

To our knowledge, this is the first report that describes an adolescent girl with galactorrhea as a result of citalopram use. Although this is quite rare, clinicians should be aware that adolescents taking SSRIs could experience hyperprolactinemia and galactorrhea. Further studies are needed to explain the pathophysiological mechanisms by which serotonergic medications affects neurohormones.

Keywords: citalopram, galactorrhea, adolescent

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[Abstract:0062][Others]

Diurnal enuresis secondary to aripiprazole

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Aripiprazole is a second generation antipsychotic drug that is partial agonist of dopamine D2 receptor and serotonin 5-HT1A receptor besides being 5-HT2A receptor antagonist. Aripiprazole is used in the treatment of some psychiatric disorders such as schizophrenia, bipolar disorder, and major depressive disorder. This drug is preferred among the other antipsychotics due to its fewer side effects with both short and long term use and limited effect on weight change. In this case presentation diurnal enuresis secondary to aripiprazole treatment will be discussed.

Case: A 27 year-old female patient presented to our clinic with the complaints of tendency to sleep, unwillingness to speak, loss of appetite, reluctance, and malaise. The patient was speaking with a low voice and answering questions after waiting for a while during the interview. Delusion of worthlessness was dominant on her thought content. Her mood was depressive and her affect was correlated with her mood. Her laboratory results, neurological examination and imaging studies were within normal range. In her psychiatric history she was diagnosed with bipolar disorder 5 years ago. Our patient was diagnosed as bipolar disorder with depressive episode in regard to her medical history and psychiatric examination. She was on 900 mg lithium and 400 mg quetiapine but she was complaining of gaining weight. It was planned to switch from quetiapine to aripiprazole. 5 mg of aripiprazole was added to treatment and raised to 10 mg one week later. Two weeks later, she had complaints of bed-wetting and enuresis in daytime. Laboratory tests were all within normal range. No organic disorders was found beneath this symptom. Aripiprazole treatment was discontinued gradually as it may be the cause for enuresis. Following discontinuation of aripiprazole, enuresis was disappeared.

It has rarely been reported that aripiprazole has side effects of urinary retention and enuresis in children. The development of enuresis in our patient after aripiprazole treatment and its disappearance after discontinuation of the medication besides the lack of any other causes are the reasons that made us link this symptom with aripiprazole treatment. 5-HT2A antagonism of 5-HT2A receptor on detrusor muscle and antagonism of alpha-1 receptor on internal sphincter may be the mechanisms for enuresis effect of aripiprazole treatment in our case. Besides, serotonin reuptake effect of aripiprazole treatment may also have role on enuresis mechanism when cholinergic neuromuscular impact of serotonin on isolated detrussor muscle and the enuresis cases after SSRI usage are considered. Antagonism of 5-HT1A inhibits bladder contractions and partial agonism of aripiprazole on 5-HT1A receptor eliminates bladder dysfunction. Unexpected effects should always be kept in mind besides probable side effects in case of psychotropic drug use. More observations and studies are needed in order to understand these adverse effects. Our case is important in the aspect of being the first report of aripiprazole treatment with diurnal enuresis side effect in the literature.

Keywords: aripiprazole, enuresis, adverse effect

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[Abstract:0065][Psychopharmacology]

REM sleep behavior disorder caused by mirtazapine therapy: a case report

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REM sleep behavior disorder (RBD) is a sleep disorder characterized by loss of muscular atonia and prominent motor behaviors during REM sleep. RBD may lead to sleep disruption and severe injuries for the patient or bed partner. In this study, we present a patient with depressive disorder who developed acute RBD following mirtazapine treatment.

Case: A 33 year-old unmarried, unemployed male patient presented to our outpatient clinic with a 5-month history of insomnia, loss of appetite, and general malaise. The patient also had a history of psychiatric treatment due to social anxiety disorder but had no apparent family history. On examination, the patient looked older than his stated age and his mood was depressive and an hedonic. His thought content was preoccupied with themes of personal inadequacy and the patient had sporadic impulsive behavior. Sleep, appetite, and sexual desire were decreased. Depending on these signs and symptoms, the patient was diagnosed as depressive disorder. The citalopram therapy (20 mg/day), which had been initiated at another health center 15 days ago was continued and combined with mirtazapine 30 mg/day. Three days later, the patient presented to our outpatient clinic and reported that he had difficulty in recalling what he saw in his dreams and that he had fallen off bed and injured his upper lip after a nightmare. The patient also stated that he had never had such complaints before. These signs were considered to indicate RBD. Therefore, the mirtazapine therapy was terminated and clonazepam was initiated at a dose of 1 mg/day. On subsequent follow-ups, the symptoms related to RBD were completely resolved and the depressive symptoms were significantly relieved. After the symptoms related to sleep disorder were relieved, the clonazepam therapy was terminated and an isolated therapy of citalopram 20 mg/day was initiated.

RBD is one of the most common sleep disorders. The clinical form of RBD is usually seen in males aged over 50 years and it has a prevalence of 0.5% in general population. The acute and temporary form of RBD often results from drug treatments (SSRI, SNRI, MAO inhibitors, and antidepressants such as mirtazapine) or various withdrawal symptoms (alcohol, barbiturates or meprobamate). Polysomnography is usually not needed in the diagnosis of RBD and the diagnosis is usually established after a careful history.

The case presented in this study represents a good example for sudden-onset, acute, and temporary RBD.

Keywords: mirtazapine, REM sleep behavior disorder, side effect

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[Abstract:0067][Psychopharmacology]

Elevated blood lithium concentration following concomitant lithium and propranolol therapy: case report

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Lithium is used in the acute and maintenance treatment of bipolar disorder. Combining lithium with other medications with the aim of reducing side effects or supporting the therapy may result in substantial elevations in lithium concentrations and may lead to signs of intoxication.. In this study, we present a patient who had been on lithium therapy for the last one year due to the diagnosis of bipolar disorder and presented with increased blood lithium concentration after the initiation of the concomitant therapy of lithium and propranolol, which is a beta-blocker drug, due to the complaint of tremor causing functional impairment.
Case: A 32 year-old male patient had been followed up due to the diagnosis of bipolar disorder for the last 9 years. The patient had been treated by various physicians with numerous pharmacotherapeutic agents and had been receiving a monotherapy with lithium 1,200 mg/day for the last one year. The patient presented with tremor that caused functional impairment in both hands, which was also detected during the physical examination. Due to the presentation of tremor, propranolol was added to the lithium therapy in a divided dosage of 40 mg/day. Two weeks later, the patient presented to the outpatient clinic with the complaints of dizziness, nausea, exhaustion, and weakness. The blood lithium level was elevated to 1.6 mEq/Lit. No pathology was detected in electrocardiography (ECG). Laboratory tests revealed that hemogram, blood biochemical parameters (glucose, urea, creatinine, electrolytes, AST, and ALT), thyroid hormone levels, and parathyroid hormone levels were in normal range. Considering the risk of drug interaction, the propranolol therapy was terminated and the blood lithium levels were followed up every other day. All the symptoms were completely resolved within the first week and the blood lithium level progressively decreased to 0.78 mEq/Lit on day 10.
Tremor is the most common neurological side effect caused by the use of lithium. In patients with side effects such as tremor, if reduction or cessation of the use of lithium is not considered, a beta-blocker drug such as propranolol should be added to the lithium therapy. In our patient, since the patient had a history of several manic episodes, we did not reduce the dosage of lithium due to the risk of repeated manic episodes and added propranolol to the lithium therapy. Propranolol is a sympatholytic agent with a hepatic elimination of 90% and is not likely to interact with lithium. In our patient, the blood lithium level was elevated, likely because of the concomitant lithium and propranolol therapy. Hypothetically, the elevation in lithium concentration may be associated with the lowering effect of propranolol on glomerular filtration and the 19% reduction in lithium clearance, the interaction of lithium and propranolol with the phosphatidyl inositol turnover and the adenylate cyclase activities, or an unknown drug interaction. The case presented in this study indicates that concomitant lithium and propranolol therapy may lead to toxic levels of lithium concentration in blood.
Keywords: lithium, beta-blocker, propranolol, drug interactions

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[Abstract:0068][Schizophrenia and other psychotic disorders]

Add-on aripiprazole mitigated olanzapine- induced metabolic syndrome in a patient with first episode psychosis patient: a case report

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32 year-old, married, father of a child, non-smoking, employed (on sick leave) office worker was admitted to our Day Care Unit of the Department of Psychiatry, University Medical Center Sarajevo. He has been diagnosed with first episode schizophrenia spectrum psychosis and has been treated for 3 months in the community mental health center- on the outpatient basis because he preserved some insight, has not been aggressive nor his behaviors was seriously disorganized. Also he was medication -compliant. He was prescribed olanzapine

10 mg a day and had initial remission of symptoms. The reason behind referral to our day care ward was psychotherapy and socio-occupational treatment and rehabilitation. Immediately upon admission basic metabolic panel blood tests (minerals, creatinine, glucose, tryglycerides and cholesterol) as well as complete blood count were done. His Body mass index (BMI) was 30 - above normal value and reported gaining weight of more then 5 kilograms since the initiation of the olanzapine treatment. Results of the performed metabolic tests in addition to abnormal BMI and slightly higher blood pressure have indicated presence of metabolic syndrome (Glucose 3.9-5.9 mmol/L 6.0 mmol/L Total triglycerides up to 2.2 mmol/L 4.3 mmol/L Total cholesterol up to 5.2 mmol/L 6.6 mmol/L Blood pressure 80/ 120 mmHg 145/90 BMI up to 25-30). In order to properly assess the metabolic syndrome we have provided results of patient's metabolic tests and blood pressure check ups that had been performed prior to the commencement of the treatment with olanzapine. Although slightly increased, these values were significantly lower than those presented after the 3-month olanzapine therapy. Only the values of blood pressure were higher months before patient had undergone treatment for his first psychotic episode. BMI was 29 and above normal. Patient adhered to olanzapine therapy and shown improvement in positive symptoms of psychosis. Also his initial insomnia was alleviated and pronounced anxiety reduced. Besides infrequent sleepiness and metabolic syndrome with weight gain, patient did not experience any other serious adverse events. Therefore dilemma whether to switch olanzapine to another second generation antipsychotic (SGA) was imminent. Study of Zhao et al. recently shown that adding aripiprazole to risperidone is safe and could reduce risperidone-induced weight gain (and hyperprolactinemia). Hence we have administered aripiprazole adjunctive to olanzapine. During the first week the dosage of aripiprazole was 2.5 mg/day and then increased to 5 mg a day.

After 3 weeks of adjunctive treatment with aripiprazole, which was well tolerated, some symptoms of the metabolic syndrome were to some extent mitigated. (Glucose 5.2 mmol/L, total triglycerides 2.8 mmol/L, total cholesterol 5.7 mmol/L, blood pressure 140/80 mm Hg, and BMI 29).

Keywords: aripiprazole, olanzapine, metabolic syndrome

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[Abstract:0069][Schizophrenia and other psychotic disorders]

Acute psychosis in an adolescent with sickle cell disease: a case report

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Sickle cell disease (SCD) is a hereditary blood disorder characterized by abnormal hemoglobin synthesis (Hb S), which is more prevalent in Mediterranean countries. Production of HbS causes increase in blood viscosity, decrease in perfusion and oxygen transportation which are thought to be responsible from the clinical features. Patients usually suffer from recurrent occlusive and pain crisis, hand foot syndrome, sequestration crisis and anemia. In addition to their medical conditions, living with a chronic illness, frequent hospitalization, being absent from school and social life contribute psychosocial and emotional difficulties that they face. In literature, studies with SCD show higher rates of internalizing disorders. Psychotic attack is reported rarely in SCD within case reports. Our aim is to present and discuss the diagnosis and treatment process of a case with SCD and psychosis.

Case: A 13 year-old typically developed girl with SCD was consulted to our clinic from child hematology inpatient service. Her complaints were exhibiting inappropriate laughs and crying, not responding to questions, mentioning about sexual and physical abuse history about her teacher starting from 10 days ago. She was claiming her teacher was swearing, beating her, crushing out a cigarette on her body, left her naked and abused her with the shape of a dog head. There was no family history about any psychiatric disorder. She was evaluated as 'Acute Psychotic Attack'. Her cerebral MRI and EEG were normal. Alprazolam 0.5 mg was used for a short while for the agitation. Pain crisis had started in following days and erythrocyte exchange was administered which lessened her complains substantially. 4 months later, she was referred to our clinic with 4 days of insomnia, motor agitation and submitting that she has the ability to predict the happenings in the future. She was referred to child hematology service for another exchange and Risperidone 0.5 mg was started. Her hallucinations appeared in crisis and vanished in inter periods, and her functionality is much better with Risperidone treatment over the 18 month follow up.

In patients with SCD, complications such as pain crisis, hospitalizations, anemia, delayed growth may play a role in forming psychological problems. It has been reported that individuals with SCD present more emotional and behavioral problems and show higher rates of adjustment disorder, anxiety and depression where psychosis seems to be an uncommon diagnosis. The link between SCD and psychosis is not clearly revealed. There are two possible explanations: (1) Psychiatric symptoms are associated with SCD exacerbation or complication due to sickling and silent infarcts in related regions in brain and considered as 'Organic Mental Disorders'; or (2) these

conditions are independent. Absence of family history in any psychiatric disorder, concurrent occurrence of psychiatric symptoms and exacerbation in SCD and relieve in psychiatric symptoms after exchange procedure suggest the former explanation as the pathogenesis in our case. In her follow up, the need to start antipsychotic in addition to exchange also raise the question that these conditions occur separately. Further studies are required to find out the underlying pathogenesis in this field.
Keywords: psychosis, sickle cell, adolescent

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[Abstract:0072][Psychopharmacology]

Acute urinary retention requiring urinary catheterization related to paroxetine use

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Selective serotonin reuptake inhibitors (SSRIs) are the first-line treatment for depressive disorders, having replaced tricyclic antidepressants (TCAs). One concern about TCAs is the prevalence of side effects, in particular, anticholinergic symptoms such as dry mouth, sedation and constipation. Urinary retention is another potential adverse effect of TCAs but is also rarely seen with SSRIs. The following is a case of an otherwise healthy female patient with major depression who developed acute urinary retention after taking paroxetine in treatment.
Case: The patient was a 27 year-old female patient, with average weight, and no previous medical or substance use history, who presented to the outpatient psychiatry clinic for treatment of depression. He had no prior history of psychiatric medication treatment. A comprehensive physical examination and blood tests (chemistry, comprehensive blood count, hepatic function panel and thyroid function), all done 1 week prior, were normal. she was evaluated and diagnosed with major depressive disorder of mild severity, according to DSM-IV diagnostic criteria. She was given a prescription for paroxetine and told to start taking 10 mg for 3 days and then 20 mg daily thereafter. She returned a week later stating that she was still depressed. On psychiatric evaluation, she reported no acute changes in mood or any anxiety symptoms. She denied any physical complaints. Her only complaint was that, for the past day, she had great difficulty passing urine and felt that his "bladder was full" and painful. The patient was sent for a consultation to urology and internal medicine. Her physical examination revealed mild suprapubic distention, with mild tenderness, but was otherwise normal. Her blood pressure, pulse and temperature were normal. An abdominal ultrasound was negative for renal stones and positive for a distended bladder. Laboratory tests were normal and a urinalysis revealed normal range, as was her urine drug screen. The patient was catheterized. Paroxetine was immediately discontinued, and her urinary retention subsided within 24 hours. The same pattern reemerged upon a second trial of paroxetine a week later. She was started on sertraline and titrated to 50 mg/day without any urinary side effects.
This is the only known published report of acute urinary retention in a patient taking only paroxetine. Urinary retention can be either acute or chronic and has multiple etiologies. It is possible to suggest that the sudden surge in central serotonin contributed to this patient's urinary retention, especially given the absence of concomitant medications or medical comorbidities. However, an other SSRI, sertraline, did not cause to any urinary symptom in this patient. Therefore, the anticholinergic properties of paroxetine seem to be more reasonable etiological factor related to urinary retention in the present case. Otherwise, it is important to note that paroxetine has the strongest serotonin reuptake activity compared to other SSRIs. In conclusion, clinicians should be aware of paroxetine's potential to induce acute urinary retention in patients with no urological illness, consider the anticholinergic properties of this drug when prescribing it to their patients.
Keywords: urinary retention, paroxetine, SSRI, sertraline

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[Abstract:0076][Psychopharmacology]

Polypharmacy as the cause of epilepsy and postictal psychosis: a case presentation with an emphasis to rational drug use

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Chronic course of the illness, inability to achieve the optimal response and drug side effects are among the main reasons for multiple drug use in psychiatric disorders. Although there is not a solid agreement on the definition of "polypharmacy", the term for multiple drug use, it is usually used to mean taking several drugs at the same time for more than one indication. Polypharmacy involves a number of risk factors including a multiplicity of side effects, drug interaction, and decreased patient compliance with the treatment. One of the side effects of multiple drug use is that it may lead to epilepsy by lowering the seizure threshold. The patient described in this case report is a 51 year-old male patient who was on formoterol fumarate 800 mg/day for his shortness of breath; fenofibrate 267 mg/day for hyperlipidemia, rosuvastatin calcium 20 mg/day, and acetylsalicylate 300 mg/day for his dyslipidemia; insulin detemir 15 units/day and insulin aspart 45 units/day for his diabetes mellitus; levatiracetam 2000 mg/day and carbamazepine 1200 mg/day for his epilepsy; and clomipramine150 mg/day, duloxetine 60 mg/day, and olanzapine 10 mg/day for his psychiatric problems at the admission. Since the patient's EEG tracing was indicating a non-convulsive status we decided to discontinue medications in order to make a differential diagnosis. After his antipsychotic and antidepressant therapies were stopped, no change was observed in the patient's symptoms. As his anti-epileptic therapy was re-instituted and his EEG tracing recovered, there was a small improvement in the patient's current symptoms. When his levatiracetam therapy was also stopped, all the antidepressant, antipsychotic and anti-epileptic therapies included in his current treatment were stopped completely. After that, his self-care and content of speech improved. The level of his attention gathering and maintaining became adequate. The deficit in his remote memory decreased, his moods and emotions became euthymic. His speed of association increased, his malaise, loss of motivation and unhappiness decreased, his sleep became orderly, his psychomotor retardation, anhedonia, anergy, avolition, social isolation, and social and occupational functioning deficit decreased. The patient was discharged from the clinic in good condition with a therapy including formoterol fumarate 800 mg/day, fenofibrate 267 mg/day, insulin detemir 15 units/day, insulin aspart 45 units/day, rosuvastatin calcium 20 mg/day and acetylsalicylate 300 mg/day. During his first 3-month follow-up period, no psychiatric symptoms were seen in the patient, nor was there any abnormality in his neurologic examinations for epilepsy. In his outpatient follow-up, it was observed that his therapies for diabetes mellitus and dyslipidemia were also stopped by the endocrinologists. Three months after his discharge, the patient was started an essitalopram 10 mg therapy because he had religious obsessions and anxiety. When an increase was seen in his obsessions a month later, his EEG was taken again. As epileptiform discharges were found in his EEG, the patient's escitalopram therapy was discontinued and he was hospitalized once more to receive antiepileptic drug infusion. Following the improvement in his EEG tracing, the patient's obsessions decreased and he was discharged. When his anxiety symptoms reemerged during his follow-up examination, he was started sertraline therapy. The patient had no complaints during his regular follow-up examinations performed for 18 months. The patient's social and occupational functioning improved notably. Care should be taken when deciding on what would be the "best first-line medication" and "how long to use it" for a specific disease and if the desired outcome is not achieved from the first-line medication, deciding on what would be the "best second-line medication". This is called evidence-based "rational psychopharmacology". In this case after all of the patient's antidepressant, antipsychotic and antiepileptic therapies were discontinued completely, his self-care improved, his content of speech improved, his concentration and maintaining it became better, his memory functions recovered, his associations became faster, his psychomotor retardation, anhedonia, anergy, avolition, social isolation, and poor social and occupational functioning improved. We think that this case report is valuable in that it shows the importance of differential diagnosis and rational treatment in psychiatry. When treating psychiatric disorders, instead of adding a new medication for each symptom, a holistic approach should be employed for the patient and the therapy should be arranged accordingly. Otherwise, it should be noted that adding a new drug may lead to new adverse effects; multiple antipsychotic drug use, in particular, may result in epilepsy; epileptic discharges may cause psychiatric disorders; and all these may end up in misdiagnoses and treatment dilemmas.

Keywords: polypharmacy, psychosis, epilepsy

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[Abstract:0077][Schizophrenia and other psychotic disorders]

A rare case: Clozapine related neuroleptic malignant syndrome

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NMS may also result from the use of dopamine antagonists, withdrawal of anti-Parkinson agents and in rare cases, from abrupt discontinuation of antipsychotics. Reported incidences range between 0.5 and 3% in patients receiving first-generation antipsychotic, which regresses to as low as 0.01 to 0.02% with the use of second-generation antipsychotics. Typical symptoms are muscle rigidity and increased body temperature. Although the exact incidence in second-generation antipsychotic users is not known, there are reports of NMS cases with almost all types of second-generation antipsychotics. According to the literature, NMS associated with clozapine can potentially appear in different clinical manifestations than those that are due to second-generation antipsychotics, and vigilance is therefore warranted for diagnosis.

Case: A 22 year-old male patient has been under treatment and follow-up for schizoaffective disorder by our clinic for 3 years and receiving treatment with clozapine 400 mg/day, valproic acid 1500 mg/day, lamotrigine 200 mg/day and escitalopram 20 mg/day for about 3 months. While on treatment, the patient presented to our emergency unit with sweating, tremor, fever and visual hallucinations. Following the initial assessment in the emergency unit, the patient was admitted to the intensive care unit with pneumonia diagnosis. The patient was started on meronem 6 g/day. On the second day in the intensive care unit, creatine kinase (CK) and white blood cell (WBC) elevations developed in addition to the existing sweating and spasms and therefore opinion of the neurology department was requested, and encephalitis or NMS was considered for differential diagnosis. Cranial MR was taken for differential diagnosis showing no evidence of encephalitis. Psychiatric examination could not rule out NMS diagnosis either and patient's neuroleptic drugs were withdrawn, following which a decrease in WBC and CK levels was observed. On day 5 of hospitalization, the patient developed delirium characterized by blurred vision, inability focus and maintain attention, psychomotor agitation and visual hallucinations. Haloperidol 5 mg ½ ampoule was administered to the patient under close supervision but the patient's spasms and tremors persisted and increased in severity with simultaneous increases in CK and WBC. On day 9 of hospitalization, patient's agitation worsened and was treated with intravenous haloperidol 5 mg ampoule but spasms, tremor, fever worsened and WBC and CK elevated on the day after, supporting NMS diagnosis. Based on these findings, bromocriptine treatment was started and titrated up to 20 mg/day. On day 7 of treatment initiation, there was a marked improvement in patient's spasms, tremor, sweating and pyrexia. CK and WBC values returned to normal. The patient became hemodynamically stable. NMS is a potentially life-threatening condition and it should be borne in mind that it can occur with all types of antipsychotics at any time and that early diagnosis is the most efficient step in preventing progression of NMS to a serious and fatal condition. Given that NMS may present with atypical manifestations, even a single NMS symptom in any patient receiving an antipsychotic should alert the physician and prompt consideration of NMS in differential diagnosis.

Keywords: clozapine, neuroleptic malignant syndrome, antipsychotic

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[Abstract:0084][PTSD]

Effects of risperidone augmentation treatment in an adolescent diagnosed with post traumatic stress disorder: a case report

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Risperidone is an atypical antipsychotic which blokes dopamine D2, serotonin 5HT2a, alpha-adrenergic and histaminergic receptors. Risperidone is using in treatment of schizophrenia and acute manic or mixed attacks of Mood disorders. Autism, mental retardation, tourette syndrome and aggression are the other usage areas.

Case: A 17 year-old female patient with post-traumatic stress disorder and major depressive disorder was reported. One and a half years ago patient's father was killed by a villager unexpectedly. After this event patient had begun to afraid of killing by murderer's family. There were serious and continuous post-traumatic stress disorder and major depressive disorder symptoms. After patient's evaluation, hospitalization suggested but patient did not accept. In treatment we began sertraline 25 mg/day and one week later increased dose to 50 mg/day. One month later her complaints were continues in the same way. In that interview her mother especially complained about aggression, sleep disturbance and uneasiness so risperidone 1 mg/day added to treatment. One month later her depressive and traumatic symptoms decreased markedly, almost completely improved.
Antidepressants are priority medicines in post-traumatic stress disorder. But antidepressants found less effective in acute than chronic post-traumatic stress disorder. To find more effective treatment for post-traumatic stress disorder is an important and serious issue because of low response rate and frequent relapses. In the literature it's shown that atypical antipsychotics like risperidone, olanzapine, quetiapine, clozapine and aripiprazole are effective in post-traumatic stress disorder alone or additional treatment. A study showed that 90% of post traumatic stress disorder diagnosed patients have benefited from risperidone augmentation treatment. In literature some studies pointed out that risperidone augmentation treatment is effective in post traumatic stress disorder's symptoms like hyperarousal symptoms, anxiety, irritation, intrusions, nightmares, flashbacks, re-experiencing and avoidance. In some studies it's determined that taking additional risperidone treatment is more effective and effects in shorter time.
Additional atypical antipsychotic treatment using frequently. But In foreign literature and our country there are a few studies about this subject, especially on children and adolescents. With this case report it's aimed to attract the attention to the role of antipsychotics in post traumatic stress disorder's treatment and deficiency of investigations on this subject. So with new studies about the role of antipsychotics in treatment, post traumatic stress disorder would be treated earlier and better.
Keywords: antipsychotic agents, risperidone, stress disorders, post-traumatic

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[Abstract:0085][Mood disorders]

Post-traumatic Stress Disorder and Bipolar Disorder after sexual abuse: a case report

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Childhood traumas include events which occur unexpectedly and threaten a person's life. There are studies which show that stress and emotional trauma in early childhood are associated with increased psychopathology in the etiology of BD. In PTSD, conditions such as observable irritability, sleep irregularity can be similar to symptoms in BD. This situation can both create confusion in diagnosis and can also make the treatment of an existing comorbidity patient more difficult. In this case report, we wanted to discuss a case who developed PTSD and BD after a sexual abuse she experienced.
Case: An 11 year-old girl, was brought to our outpatient clinic by Samsun Chief Public Prosecutor's Office to find out whether she had a mental disorder. "My parents were chopping wood. When I went to the woodshed, my uncle came near me, gave me money, touched my foot with his, tried to take off my pants, I pushed him away." In the psychiatric examination, it was found that complaints such as startling when touched by someone, not being able to stay alone at home, not being able to sleep alone, being scared to go out, having nightmares, getting angry quickly, feeling like re-experiencing of what happened, and not being able to concentrate on lessons started gradually. The patient was started sertraline with a diagnosis of PTSD and the dose was increased to 200 mg/day when the complaints did not improve, later the patient's medication was changed with 20 mg/day fluoxetine when her complaints did not alleviate. During this process, the patient's PTSD symptoms decreased by more than 50% when compared with the baseline. In her first follow-up in 2015, the patient who had symptoms of an increase in irritability, restlessness, increase in the amount of talking, becoming more outgoing, laughing for no reason, grandiosity and also thoughts of excessive guilt and regret, anhedonia and psychomotor agitation symptoms was diagnosed with BD mix episode and Fluoxetine was stopped and Valproic acid 1000 mg/day was started and Risperidone was increased to 3 mg/day. Quetiapine 300 mg/day was added to the treatment of the patient whose manic symptoms had not decreased at the follow up. The patient whose complaints had decreased later had similar attacks during the year. The patient was diagnosed with rapid cycling bipolar disorder and antipsychotics were added to her treatment during the attacks and the attacks were taken under control.
It has been assumed that early life traumas can play a role as predisposing factor for psychiatric diseases. Studies have shown traumatic experiences to have significant and long term influences on bipolar disorder symptoms. Thus, in the presence of PTSD in patients with BD

due to traumatic experiences, worse prognosis is considered. It has been found that in patients who experienced abuse, the onset age of the disease was younger and manic symptoms were more severe when compared with those who did not experience abuse. In our case, the early onset age, resistance to treatment and rapid cycling episodes support the information in literature.
In patients who have childhood trauma, BD starts at an earlier age and these patients have more mood attacks.
Keywords: bipolar disorder, child, PTSD, sexual abuse

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[Abstract:0086][Mood disorders]

Delirious mania: a case report

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Delirious mania (DM) is a syndrome of excitement, delirium and psychosis of acute onset, not associated with prior systemic or mental disorder. Manic signs include insomnia, acute excitement, grandiosity, emotional lability, delusions, altered consciousness, disorientation characterized by delirium often accompanied by posturing, stereotypy, mutism, negativism and echo-phenomena suggesting catatonia. Here we report a case of DM.
Case: 18 year-old female patient with a former psychiatric history (diagnosed as major depressive disorder, 4 months ago, after escitalopram and mirtazapine treatment symptoms disappeared and patient stopped to take medications) suddenly had started to write on walls and in the following days she began to have pressured speech, outbursts of anger and destructive behaviors as well. Transferred to our hospital after recent admission only at one other hospital. Olanzapine 20 mg/day, sodium valproate 1000 mg/day, biperiden 4 mg/day treatment started with a bipolar affective disorder (BAB) mixed episode. After the increment in sodium valproate dosage eruptions had appeared in patient's skin and sodium valproate was stopped. After 20 days of hospitalization in the other clinic we evaluated and patient had disorganized behaviors, insomnia, logorrhea, sexually disinhibited and hallucinatory behaviors, irritable mood, loosening of associations and delirium signs. She was exhibiting negativism by refusing to follow commands and was highly energetic and required sedation. Young Mania Rating Scale (YMRS) score was 57. Lithium carbonate 1200 mg/day, quetiapine 900 mg/day, clonazepam 6 mg/day treatment was started. She demonstrated severe psychomotor agitation and was being placed in 4 point restraints mostly during her hospitalization. Echolalia, echopraxia, verbigeration, hallucinatory behaviors were rarely seen in the follow-up period, besides this rigidity and autonomic disturbances were not seen. Head MRI and EEG revealed no pathology that explains this clinical situation. In other investigations only subclinical hyperthyroidism was detected and in consequence of consultation only diet therapy was suggested. Patient diagnosed as DM. At the sixth day of hospitalization clonazepam treatment was stopped and lorazepam treatment 10 mg/day, on the 7th day of treatment 12.5 mg/day lorazepam was administered. After lorazepam treatment patient's sleep problem has disappeared and patient started to sleep almost 7 hours at nights. Besides we completed our electroconvulsive therapy (ECT) preperations because of deficiency of pharmacotherapy but patient's family had wanted to take their patient to a different hospital and patient discharged from our clinic upon to family's demand at the tenth day of hospitalization. YMRS score was 54 at the time of discharge.
The patient meets DSM-5 criteria both for hyperactive type delirium and bipolar I disorder manic episode. Besides this catatonic symptoms were rarely seen during follow-up. In DM catatonic signs are usually seen. In our case, delirium and manic symptoms were predominant and there was no sign supporting neuroleptic malign syndrome (NMS) or malignant catatonia (MC). In differential diagnosis of DM MC and NMS should be kept in mind and in treatment of DM high dose benzodiazepines and ECT should be considered and rapid diagnosis and treatment is critical for the reason that when this syndrome reaches a malignant state mortality rate increases easily.
Keywords: delirious mania, electroconvulsive therapy, catatonia

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[Abstract:0094][Psychopharmacology]

Oral megadose oros methylphenidate (Concerta) ingestion for suicide attempt

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ADHD is one of the most common childhood onset psychiatric disorders, prevalence of which is approximately 4-9% in children. More than 2.7 million children are estimated to use ADHD medications in the USA. As a result, an increased risk of therapeutic errors and intentional overdoses due to abuse, misuse or suicide attempts can be mentioned. A few cases of overdose with oros methylphenidate was reported. In this report, a suicide attempt with high-dose oros MPH and alprazolam in a 14 year-old boy with ADHD is described.
Case: A 14 year-old-boy presented to emergency service of our hospital because of suicide attempt with 1080 mg oros MPH and 1.5 mg alprazolam. He had been diagnosed as having ADHD 3 years ago and had been on medication Concerta for a year, but discontinued because of decrease in his symptoms. 8 months ago he lost his mother and he began to live problems again. He had also sleep disturbance and anxiety. With these complaints, a psychiatrist prescribed him 36 mg/day Concerta and 0.5 mg Alprazolam. He had taken these drugs for 20 days, until his impulsive suicide attempt. Two hours after ingestion, he felt weak and sleepy so he told his father and was brought immediately to emergency service. The child's mental status was clear on examination, only sedation was observed; his blood pressure, heart and respiration rates and all laboratory analyses were between normal levels. No additional therapy was needed and he was discharged after 8 hours' observation.
In 2010, 17,000 intoxications with ADHD medications were reported by US poison centers, with 80% of them occurring in children under 19 years old. In younger children, these overdoses may be accidental due to exploratory behavior of childhood; but in adolescents, it is commonly related with abuse and misuse. The primary clinical syndrome of overdose is presented with a sympathomimetic overdrive with cardiovascular and neurological effects. Agitation, mydriasis, hyperreflexia, tremor, movement disorders, anxiety, confusion, paranoia, hallucinations, delirium, seizures and in severe cases, multi-organ failure can be seen. However, most of methylphenidate overdoses have presented with mild or moderate severity. In symptomatic cases, overdose management is largely supportive, targeting to control the sympathomimetic syndrome with benzodiazepines. Central alpha-adrenoreceptor agonists and antipsychotics can also be used. Until now, a few cases of overdose with oros methylphenidate was reported and all reported none or mild intoxication symptoms as our case. In our case, Alprazolam intake with MPH may also have reduced the sympathomimetic overdrive.
In conclusion, this case supports the general opinion that oros MPH's safety margin is high. Even so, the clinicians should be aware of disease related impulsiveness which may result in suicide attempts especially in adolescents with ADHD even in the absence of depression. Another point worth mentioning is the necessity of being careful about using benzodiazepines, especially in impulsive or suicidal adolescents, as they can use them for suicide attempt. Education of children, parents and teachers about and storage and usage of drugs is also important.
Keywords: oros methylphenidate, overdose, adolescent

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[Abstract:0095][Schizophrenia and other psychotic disorders]

Confusion in diagnosis: a cylic psychotic process triggered by massive internet use or just internet addiction disorder?

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Internet use is a rapidly growing technological and social phenomenon, especially in adolescents and young adults. This is particularly concerning when considering mental health, where there is evidence to suggest that problematic internet use is linked with psychiatric symptoms and syndromes.
Case: An 11 year-old boy, without any other medical disease, presented to our clinic with social withdrawal, school refusal, sleep disturbance, deterioration in self care, school success, hand writing and spending long hours on internet games. He sometimes acted and talked as if

there was someone else with him, although he was alone. During the examination, he refused to talk and did not ever make eye contact. His physical examination routine laboratory analyses and magnetic resonance imaging of the brain were normal. There was genetic predisposition of psychotic disorders in the family history of both father and mother. Risperidone 1 mg was started with a tentative diagnosis of psychosis and the dosage was increased to 1.5 mg after one week. Termination of internet use was also recommended. In the third week, a significant improvement in his symptoms was observed. As he began to school again and his peer relations became better, family stopped to apply him. A year after his first application, he presented again, with the same clinic presentation. It was learned that he had not taken his drugs regularly and as he did not have any complaints during this time, his grandparents gave him an I-pad as a gift, which triggered the process again. His medication was re-started and a quick improvement was observed again. Because of the genetic predisposition history, severe clinic presentation, presence of auditory and/or visual hallucinations and cyclic nature of the disorder, we treated this case as a psychotic disorder, but we have doubts as it improved quickly after termination of internet use and presented with the same trigger again.
Although internet addiction has been a popular topic for researchers, we still know very few about it. It is found to be associated with elevated symptoms of depression and anxiety 1 but there are few investigations about its probable relation with psychotic disorders. In a recent study, it is reported that non-clinical psychotic-like experiences (PLEs) (e.g. fleeting auditory hallucinations) are commonly experienced by internet addicted ones and are considered to be a risk factor for formal psychosis. Other characteristics, such as interpersonal deficits, social anhedonia, proneness to magical thinking and impulsivity may increase susceptibility to problematic internet use are also found to be common among individuals experiencing PLEs. Given a diathesis-stress conceptualization of psychosis, where a constitutional vulnerability (confluence of genetic and early environmental factors) later interacts with social stressors, it is possible that the internet serves as an environmental factor that may unmask the subtle vulnerability. Alternatively, it is possible that individuals becoming increasingly prone to psychosis choose to spend more time using the internet in a problematic way; or basically, the clinical presentation of internet addiction disorder mimics psychosis. Further studies are needed in this area.
Keywords: internet addiction, psychosis

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[Abstract:0099][Anxiety disorders]

Panic attack case after a box of energy drink consumption

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Energy drinks are used for the first time in the 1960s in Asia and Europe. The use of energy drinks has become widespread after the 1990's. Energy drinks is used with increasing frequency for various purposes especially among young people in recent years. Of these energy drink users, 22% reported headaches and 19% reported heart palpitations from drinking energy drinks. Psychiatric studies on side effects of the energy drinks are in the form of case reports. Psychotic symptoms and manic symptoms have been reported in the cases reported so far.
Case: 21 year-old patient with no previous psychiatric applications presented with panic attack symptoms after a box energy drink consumption. Panic attack symptoms like palpitation, fear of death, sweating, chills, shortness of breath, anxiety, numbness, anticipatory anxiety in the hands are developed 2 hours after drinking a box energy drink. Then similar complaints have repeated two more times in a month. The patient presented to the emergency service twice for these attacks. The patient was admitted for treatment after a month later symptoms begin. Patient is treated with pharmacotherapy (citalopram 20 mg/day and alprazolam 0.25 mg/day) and cognitive behavioral therapy. Anticipatory anxiety was later treated symptom. There was no pathology in cardiological and endocrinological examinations done for organic pathology.
Side effects about energy drinks increases in the publications. There substances such as vitamin B types (B2, B3, B5, B6, B12), caffeine, glucose and glucuronolactone in energy drinks. Death has been reported after three boxes energy drink consumption in Turkey. In another case a person who consuming 1-2 boxes energy drink every day for a month was found to develop psychotic disorders. Caffeine is often held responsible for resulting side effects. Caffeine blocks adenosine receptors in the central nervous system. It has been showed that adenosine A2A receptor mutations can cause both panic disorder and caffeine induced panic attack. There is not published cases of panic attacks associated with energy drinks previously. Therefore we report a case about caffeine induced panic attack here.
There are a lot of side effects of energy drink especially in recent years. Energy drinks contain many chemical substances. These chemicals can cause various side effects in susceptible people. Therefore they should be used with caution.
Keywords: energy drink, panic attack, caffeine

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[Abstract:0104][Motor disorders]

Fluoxetine-induced tic disorder with depressive disorder: a case report

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Major depressive disorder (MDD) causes significant disturbance in emotional, social and academic functioning in adolescence. The rates of MDD particularly in adolescent boys may have found higher rates than girls. Tic disorders are defined in the Diagnostic and Statistical Manual of Mental Disorders (DSM) based on type (motor or phonic and simple or complex), duration of tics (sudden, rapid). Secondary causes of tics are include drug-induced tics, head trauma, encephalitis, stroke, and carbon monoxide poisoning. Tic disorders are commonly considered to be childhood syndromes, and also tics occasionally develop during adulthood; adult-onset tics often have a secondary cause. Induction or exacerbation of tics have been reported following exposure to agents which increasing the central dopaminergic activity as well as stress.
Case: An anxious mother presented with her 15 year-old high school-boy. He suffered from depressive symptoms (such as sadness, irritability, eating problems, fatigue, withdrawal, abnormal reactions to strangers) for last three years. His symptoms, such as guilt, low self worth, anhedonia, hopelessness, concentration problems were increased, particularly within last four months. Beck depression inventory is used to evaluate the depression of adulthood included in our study. He was diagnosed with major depressive disorder according to Diagnostic and Statistical Manual of Mental Disorders-Five Edition (DSM-5) criteria through psychiatric examination and tests. Whereupon, fluoxetine 10 mg/day was initiated and behavioral interventions were made for his depressive thoughts. The patient had not been exposed to any other pharmacologic agents prior to this treatment. After the first visit to our clinic, he was invited to a follow-up meeting within one month. At the follow-up meeting, intensity of depressive symptoms were decreased. But, at the tenth day of treatment, he had tics in the form of winking, and arm movements. These tics were more frequently at the beginning but they decreased in time. Patient was consulted by experienced neurologist. Neurologic examination with laboratory findings (complete blood count, biochemical analysis, thyroid function tests) revealed no abnormality. After then, parents were informed by researchers that tics may be related to the medical treatment and may be transient. During three months of the follow-up, his medical treatment, accompanied by tics of winking and arm movements at non-disturbing level, was continued. These rare and mild tics disappeared completely, at the second weeks after cessation of fluoxetine. The patient has been followed for six months after the cessation of the drug without any medication, and he no longer had any ticks during follow-up period.
Our patient's chronology of events, symptoms consistent with previously documented cases of fluoxetine -induced tic disorder with MDD. As in our case, tic disorders are more common among males than females. Tics should be distinguished from possible secondary causes. Antidepressants, including the SSRIs are known to occasionally cause various movement disorders. In our case, although, the treatment period was very short but extrapyramidal side effects were marked. Although, we could not find any organic or pathological sign to explain the EPS. One plausible explanation may have been effects of serotonin modulates to dopaminergic neurons.
Keywords: tic disorder, depressive disorder, SSRIs, adolescence

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[Abstract:0105][Mood disorders]

Urinary retention in a male patient associated with aripiprazole

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Urinary retention and voiding dysfunction is a rare side effect during the use of antipsychotic, adrenergic or anticholinergic medications. It is known that urinary retention may develop during the use of low-potency first generation antipsychotics; but it is rare with the use of second generation antipsychotics (SGA). Aripiprazole has a lower incidence of adverse effects than other second generation antipsychotics and aripiprazole can be used as an alternative treatment on patients who have urinary retention depending on antipsychotic use. Although there are limited data in literature about the effect of aripiprazole on urinary retention, a case was encountered urinary retention with the use of antidepressants and aripiprazole together. Also there is only one case in the literature about urinary retention occurred with aripiprazole. According to the Naranjo criteria which is developed to assess adverse drug reactions, urinary retention of our

case can be considered as a "probable" negative side effect of aripiprazole

Case: M.A., 23 year-old male patient, presented to the psychiatric clinic with depressive symptoms. Last one month, depressive symptoms exacerbated, so we added 5 mg/day aripiprazole to paroxetine 10 mg/day treatment. The paroxetine did not increased according to the history of drug (paroxetine 20 mg/day) related hypomania. In his outpatient clinic follow-ups he had a complaint of decrease in urine volume and urinary frequency one day after aripiprazole treatment and developped urinary retention second day of aripiprazole treatment. There was not any etiological cause of that can cause to acute urinary retention according to physical examinational and ultrasonogrophic findings in urology consultation. Aripiprazole treatment was stopped because of patient's complaints started immediately after initiation of aripiprazole. After the withdrawal of drug, patient's symptoms gradually decreased and disappeared completely within 1 week. Several neurotransmitters including dopamine, serotonin, noradrenaline, GABA, excitatory and inhibitory amino acids, opioids, acetylcholine and neuropeptides are involved in the control of micturition. α 1 adrenergic receptors increase the contraction of bladder neck, urethra and prostate for enhance the bladder outflow resistance. 5 HT receptors have critical function in voiding. Several studies revealed that activation of D1 and D5 receptors represses micturition while D2, D3, D4 activates micturition 15 According to this knowledge, aripiprazole can be considered to have facilitate effect on voiding via partial agonism on D2 and 5-HT1A receptors. Our case emerged urinary retention with the addition of aripiprazole while under treatment of paroxetine. Patient's complaints have stopped with the discontinuation of aripiprazole and never emerged under treatment of paroxetine and risperidone. We aimed to contribute this case because there is limited data in literature about this subject.

As a result, adrenergic, cholinergic, serotonergic, dopaminergic and histaminergic pathways have role on control of micturition but it is not clear that on which pathway aripiprazole acts on urination. However there is no symptom of urinary retention after stopping aripiprazole and adding risperidone, shows that there is a different mechanism associated with voiding in this case. Additional studies need to be done to clarify the mechanism.

Keywords: urinary retention, aripiprazole, side effect

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[Abstract:0111][Eating disorders]

Methylphenidate treatment for bulimia nervosa with adult ADHD

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There is an increasing literature suggesting a link between attention deficit and hyperactivity disorder (ADHD) and eating disorders (ED), especially bulimia nervosa (BN). BN is defined by repeated episodes of binge eating followed by inappropriate compensatory behaviors. The treatment of BN is a complex process including the use of psychotropic drugs, psychotherapy, nutritional counseling and the treatment of the medical complications. Psychotherapy and antidepressant medication are helpful to many patients with BN. However, a substantial number of bulimics respond poorly to such treatments. Recent studies suggest that many of the poor responders have comorbid disorders as ADHD. There are few reports of Methylphenidate (MPH), a psychostimulant drug used to treat ADHD, administered to patients with BN and ADHD which describe a significant decrease in binge eating. We report two cases suffering from BN and previously unsuspected ADHD in adulthood treated with extended-release methylphenidate.

Cases: First case; A 33 year-old male patient had a 7 year history of BN, purging subtype. For the past 7 years, he binged and purged at least two or three times per day. Adequate trials of fluoxetine, paroxetine and venlafaxine, combined with psychotherapy, had not been helpful enough in the past. We administered MPH due to his ADHD symptoms. During the 24 weeks of treatment with extended-release MPH, he reported absence of desire to binge or induce vomiting. He has less impulsivity, an increase in attention, and a decrease in distractibility, decrease in alcohol use. His work performance, as well as his interpersonal relationships have improved significantly.

Second case; A 36 year-old female patient had suffered from BN, non-purging subtype, for at least 15 years. On admission, she had a comorbid diagnosis of depression and was experiencing suicidal thoughts. She relates a lifelong history of difficulty concentrating, abnormal body image, labile mood, and obsessionality. She was treated with fluoxetine 60 mg/day that reduced anxiety, obsessionality and depression but had little effect on her binge frequency or ADHD like symptoms. After adding MPH treatment to her fluoxetine regime, she reported decreased binging (from 15-18 times per week to 1-2 times per week), as well as improved concentration. She has continued on extended-release MPH 36 mg/day and fluoxetine 60 mg/day for 3 months.

We performed Eating Attitude Test, Adult ADHD Self-Report Scale, Beck Anxiety Scale, Beck Depression Inventory for each patient either on admission or on follow-up controls. Both patients had decreased binging after the first days of extended-release methylphenidate

up to 36 mg a day. They continued well on this treatment, reporting decreased ADHD symptoms and binging, with no adverse effects. Identifying comorbid ADHD in adults is crucial for the patients with BN. Comorbidities and overlap of symptomatology need to be taken into account for treatment of bulimics. We administered MPH to 2 patients with BN and ADHD and found beneficial effects. These patients had not responded to adequate trials of psychotherapy and antidepressant medications. MPH may be useful for bulimics with ADHD who respond poorly to conventional treatments. Further studies of MPH administration in BN may be worthwhile.

Keywords: attention deficit and hyperactivity disorder, bulimia nervosa, methylphenidate

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[Abstract:0116][Others]

Transcranial magnetic stimulation in a depressive patient with cardiac pacemaker

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Transcranial magnetic stimulation (TMS) modulates cortical excitability via the application of magnetic field pulses. The magnetic field generated by TMS may shift or damage any implanted medical device sensitive to these magnetic fields. Implanted devices, including aneurysm clips, implanted electrodes, and cochlear implants, could potentially suffer movement or demagnetization during stimulation. TMS can induce an unintended generation of voltage in nearby devices. TMS pulses can also damage the internal circuitry of electronic implants near the coil, causing them to malfunction or permanently damage. The aim of this poster is to add to the current limited knowledge about the safety issues related to rTMS in patients with cardiac pacemakers. To our knowledge, this is the first report of rTMS use in a patient with cardiac pacemaker

Case: A 72 year-old male patient with a four year history of dual-chamber pacemaker implantation (Symphony DR 2550, Ela Medical INC, Sorin Group, France) for sick sinus syndrome presented to psychiatry clinics. He suffered from a 16 year history of treatment-resistant recurrent major depressive disorder and referred for a course of rTMS. Written informed consent containing the risks of this procedure including pacemaker dysfunction or damage, fatal arrhythmia, and death was obtained from the patient and his family. His current medications lamotrigine 200 mg/day, escitalopram 20 mg/day were kept stable for 3 weeks and remained unchanged during rTMS treatment. r TMS was delivered using a Magstim Super Rapid Stimulator set with a 70 mm, figure-eight coil (Magstim Company Ltd., UK) to the left dorsolateral prefrontal cortex with the parameters of 10 Hz, 110% of motor threshold intensity, 1000 stimuli/day (40 trains of 2.5 seconds, each with an interstimulus interval of 20 seconds), 6 sessions/week. His baseline blood pressure and heart rate were 125/70 mmHg and 64 beats per minute. During the rTMS sessions, he was continuously monitored by electrocardiography and pulse oximeter. The patient did not report any distress during the rTMS sessions, and no ECG parameter changed significantly after rTMS sessions. Depressive symptoms started to decrease after 1 week of rTMS and almost disappeared toward the end of rTMS treatment. The major contraindications to rTMS treatment are arranged in two categories: conditions that increase the risk of seizure induction and the presence of a device that could interact with the induced magnetic field. Cardiac pacemakers may be adversely affected by magnetic fields. Most concerns related to magnetic field affected pacemakers are switch closure, loss of battery life, heating, alteration of programming and potential damage to the pacemaker circuitry, rapid atrial pacing, racing at multiples of the radio frequency pulse, asynchronous pacing, inhibition of pacing output and induction of ventricular fibrillation. However in rTMS, there is a substantial distance between the implanted wires/pulse generators and where the TMS coil is discharged.

The present case suggests that rTMS could be safely administered to a patient with a cardiac pacemaker. However, the absolute safety of rTMS in patients with a cardiac pacemaker cannot be guaranteed, and each case requires a deliberate harm–benefit ratio evaluation.

Keywords: transcranial magnetic stimulation, cardiac pacemaker, safety

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[Abstract:0117][Psychopharmacology]

Paliperidone and paliperidone palmitat -induced leukocytoclastic vasculitis

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Paliperidone and paliperidone palmitat (PP) are novel antipsychotic drugs that may cause adverse reactions in several organs and systems. Here we present a case with skin lesions and arthiritis after initiation of both paliperidone and PP.

Case: A 50 year-old female patient with a history of schizophrenia had been on treatment with haloperidol and chlorpromazine for about 21 days. Haloperidole was discontinued and paliperidone 6 mg/day oral and paliperidone palmitat 100 mg/month long-acting injectable form (i.m.) were initiated because of treatment-resistant psychotic symptoms. On the second day of initiation, multiple erythematous, partly bullous and palpable purpura appeared on her ankles, legs, thighs, wrists, forearms and abdomen. Arthirithis was observed in wrist, knee and proximal interphalangeal joints. Laboratory tests including serum electrolyte levels, renal function tests, hepatic enzyme levels, prothrombin time and activated partial thromboplastin time were within normal ranges. In complete blood count; hemoglobin, hematocrit, leukocyte and platelet counts were normal but neutrophilia and elevated monocyte levels were observed. Erythrocyte sedimentation rate, C-reactive protein and rheumatoid factor levels were elevated. Urinalysis showed 2-3 leukocytes per field and negative for protein and red blood cells. Anti-neutrophil cytoplasmic antibodies (c-ANCA), perinuclear anti-neutrophil cytoplasmic antibodies (p-ANCA), anti-double stranded DNA, anti-nuclear (ANA), proteinase 3, anti-Cyclic Citrullinated Peptide (anti-CCP) antibodies were negative. Chest X-ray showed no characteristics. All drugs were discontinued after appearance of the skin lesions and corticosteroid therapy was initiated by the rheumatology policlinic. Punch biopsy of skin lesion from the right lower extremity revealed leukocytoclastic vasculitis (LCV) with perivascular neutrophilic, eosinophilic and lymphocytic infiltrate and hystiocytes with nuclear dust in the papillary dermis.

LCV is characterized by the inflammation of the small sized blood vessels, most commonly developed in the skin. Clinical features generally manifest as palpable purpura (may vary from maculopapuler rash to ulcers) often localized to the lower extremities. Drugs, infections, autoimmune diseases and malignancies are the etiologic factors that may trigger LCV. Biopsy of skin lesion is performed for histological examination to confirm the diagnosis. Treatment of skin-limited LCV is based on removal of the suspicious etiological factor and supportive interventions such as rest, elevation of legs and anti-inflammatory drugs but generally requires no treatment. Drugs are most common cause of LCV. Antipsychotic drugs such as haloperidol, thioridazine, clozapine and olanzapine were reported to be related with LCV. In this case skin lesions and arthritis healed without any sequela after 3 weeks tapering of antipsychotic therapy. Clinicians should be aware of this side effect in order to discontinue the drug and avoid progression.

Keywords: leukocytoclastic vasculitis, paliperidone palmitate, side effect

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[Abstract:0119][OCD]

Aripiprazole in the treatment of OCD and aggressive behaviors in Prader-Willi syndrome: a case report

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Prader-Willi syndrome (PWS) is a complex neurodevelopmental disorder caused by an abnormality on chromosome 15 (q11–q13) that results in behavioral characteristics including aggression and obsessive compulsive disorder (OCD). In addition to the typical obsessions and compulsions seen in OCD, some atypical OCD symptoms may also be seen with PWS (i.e., skin picking, excessive interest in food, and difficulty in changing routines). Treatments for OCD typically involve a combination of modalities including cognitive behavioral therapy, selective serotonin reuptake inhibitors (SSRI), and interventions targeting mood, sleep, and skin picking. Fluoxetine, fluvoxamine, risperidone, and N-acetyl cysteine have been reported as medications used in PWS for OCD, however, there have been no reports on the

effectiveness of aripiprazole with these conditions. Here, we present a case with PWS whose OCD and aggressive symptoms disappeared with aripiprazole treatment.

Case: The patient was an 11 year-old girl diagnosed with PWS in the genetics clinic and referred to psychiatry for her aggressive and obsessive-compulsive behaviors. Her mother reported that she was refusing to obey her diet, was irritable, and aggressive almost all day every day. She had obsessions that included sticking houseflies into her eyes and being touched by others; thus, she was frequently asking questions concerning these fears. She tampered with her eyes and frequently wanted her mother to blow on her eyes. These behaviors and questions would occur 6–7 times an hour. She was diagnosed as OCD with a moderate intellectual disability according to DSM-5. The Clinical Global Impression-Severity Scale score on her admission was 6 for OCD, and 6 for aggression. Fluoxetine (30 mg/day) and sertraline (75 mg/day) treatments were administered to the patient consecutively. Despite each drug being administered for the recommended treatment duration, neither OCD nor the aggressive symptoms improved. Because the aggressive behaviors were not diminished and she began damaging property during the treatment process, aripiprazole (5 mg/day) was administered. Following 8 weeks of treatment, the aggressive behaviors and OCD symptoms decreased in both frequency and severity, thus, the dose was increased to 2x5 mg/day. At the 12th week of aripiprazole treatment, all of the OCD symptoms disappeared and the aggressive symptoms had decreased. Her parents reported that she had shown aggressive behaviors only once or twice in the previous month (Clinical Global Impression-Improvement Scale score for OCD was 1, and for aggression was 1). Her OCD symptoms did not recur and the aggressive behaviors were not disturbing according to her parents during the 10-month follow-up period.

Like other atypical antipsychotic drugs, the mechanism of aripiprazole on OCD symptoms in PWS is not well known. Also, it is not known whether aripiprazole is more effective than other antipsychotic drugs. The present report suggests that it may be an effective treatment option. Dopaminergic and serotoninrgic activity of aripiprazole might contribute to its effectiveness on OCD symptoms in PWS. Further studies on this topic will improve our knowledge.

Keywords: aripiprazole, prader willi syndrome, obsessive compulsive disorder, aggression

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[Abstract:0121][Others]

The rare mutation under unexplained neuropsychiatric symptoms: Niemann-Pick Type C (NP-C) disease

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Obsessive-compulsive disorders (OCD) may be encountered after basal ganglia lesions of various etiologies. Most obsessive compulsive disorders cases cannot be related to any brain disease or focal lesion. However, many neurological diseases that affect basal ganglia, such as Parkinson's disease, Huntington's disease, Wilson or Tourette syndrome may be associated with OCD. Furthermore, lesions of basal ganglia or frontal cortex can induce secondary OCD and/or auto-activation deficit. We present a case with a 13 year history of unexplained psychiatric symptoms beginning with compulsions, diagnosed many different psychiatric disorders and demonstrate metabolic disease.

Case: A 26 year-old female patient, with a 13 year psychiatric history beginning with compulsions, was diagnosed many different psychiatric disorders including Tourette's Syndrome, having a history of family psychiatric that 2 year younger brother is suffering from a psychotic disorder. She was referred to our psychiatric clinic to evaluate for diagnose with the symptoms of dysarthria, motor compulsive behavior, gait disturbance, postural instability, aggressive behaviors. In her hospital stays she was tearing the sheets unstoppable in the clinic and playing with her saliva by not swallowing. In her physical examination hepatosplenomegaly was determined with elevated liver function tests. In our physical exam, her hepatomegaly was diminished despite of her splenomegaly. In her laboratory tests, autoimmune antibodies were negative but anti-DNAseB levels were high and also ASO titers were elevated, Creatinine Kinase levels were above 13000. She sometimes had suicidal thoughts because of her unvolunteered motor movements and motor compulsions. In contrast of her symptoms her cognitive functions seemed not to be effected. In her brain MRI, caudate nuclei and lentiform nuclei were atrophic. Screening test for Wilson, Huntington, Neuroacathocytosis, Muscle eye brain syndrome, Limbic Encephalitis were negative. Metabolic and genetic panels had also been worked. Dysphagia, hepatosplenomegaly, elevated creatinine kinase levels and neuropsychiatric symptoms was leading a metabolic disorder, primarily Niemann-Pick Type C (NP-C) disease. Plasma cholestane-3β,5α,6β-triol level was high but 7-ketocholesterol level was normal. The genetic analysis showed c.1947+11_1947+12insA intronic changes in NPC1 gene. So we planned

to launch the drug Miglustat.
NPC is an autosomal recessive lipid storage disorder characterized by hepatosplenomegaly and severe progressive neurological dysfunction. There are two genes involved with NP-C, but most patients (95%) have a mutation in the NP-C1 gene. Although it is often perceived as a pediatric disorder, significant numbers of patients with NPC present for the first time in adult life or survive into adult life. Adult patients are often referred to clinicians with psychosis or other major psychiatric problems. Miglustat treatment delayed onset of symptoms and increased lifespan in NP-C mice. Moreover, detailed analyses of such unusual cases can help improve our understanding of the pathogenesis of unexplained psychiatric symptoms. Therefore, we recommend that patients with demonstrate with unexplained psychiatric symptoms who have also other multiple physical symptoms undergo a thorough multidisciplinary evaluation for specific diagnoses; that they receive individualized treatment plans; and that they remain in active follow-up in order to refine treatment and monitor for the emergence of new symptoms.
Keywords: miglustat, mutation, NP-C, unexplained psychiatric symptoms

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[Abstract:0126][Psychopharmacology]

Olanzapine-induced parkinsonism: a case report

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Dopamine receptor antagonism is associated not only with antipsychotic action, but also with the generation of extrapyramidal side-effects of antipsychotic medications. Positron emission tomography studies reveal that an approximate 60-70% blockade of the D2 receptors is required for typical antipsychotics to be efficacious, but a blockade of > or =75-80% results in acute extrapyramidal side-effects. The newer atypical antipsychotics have a lower propensity to produce extrapyramidal side-effects. A new class of antipsychotics with a novel pharmacological profile and improved tolerability have emerged. Olanzapine is an atypical antipsychotic. It is associated with a good safety and tolerability profile including extrapyramidal side-effects. Parkinsonism in drug-induced parkinsonism (DIP) patients is sufficiently severe to affect daily activities and may persist for long periods of time even after cessation of the offending drug. The exact prevalence and incidence of DIP are unclear because it is frequently unrecognized or misdiagnosed as PD. Age is the most obvious risk factor for DIP, since dopamine concentrations decrease and nigral cells degenerate with age. Another risk factor is female gender, suggesting that estrogen suppresses the expression of dopamine receptors.
Case: We report on a 52 year-old female patient with paranoid schizophrenia who developed parkinsonian symptoms after three weeks of olanzapine 10 mg per day. She had been admitted to our unit for a psychotic episode with delusions of persecution and grandiosity. It was her second hospitalization. During the first hospitalization, seven years earlier, she had been treated with haloperidol. We do not have any information about the tolerability of that treatment. At the start, she received olanzapine with good tolerability but after three weeks, the patient developed severe Parkinsonian symptoms. After three weeks, the patient developed severe parkinsonian symptoms including hypertonia and akinesia. We reduced the dose to 5 mg per day without any effect on the extrapyramidal symptoms. We decided to discontinue olanzapine. After discontinue olanzapine, all the Parkinsonian symptoms had disappeared within 5 days without any other medication. Overall, it was noted that her parkinsonian symptoms were gradually improving; two days before hospital discharge, she was noted to have no definite remaining evidence of parkinsonism.
As an atypical antipsychotic, olanzapine is thought to have a favorable adverse-effect profile compared with first-generation antipsychotics, as it is less likely to cause extrapyramidal symptoms and tardive dyskinesia. Few reports of parkinsonian symptoms with olanzapine have been published in the adult population. However, clinicians need to be aware of olanzapine-induced parkinsonism.
Keywords: olanzapine, parkinsonism, extrapyramidal side-effects

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[Abstract:0131][Mood disorders]

Late-onset mania secondary to stroke: a case report

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A number of emotional and behavioral disorders, particularly Mood disorders can be observed secondary to a stroke. Depression is the most common mental disorder after a stroke, although mania can also occur more rarely. There are only a few systematic studies of mania in acute stroke. According to previous case reports, post-stroke mania has been related to: predisposing genetic factor, subcortical brain atrophy, and damage to the right corticolimbic pathways. Mania seems to be more frequent after right-sided lesions, but there are also reports of mania following left lesions. More frequently, these patients were male, without psychiatric antecedents or subcortical atrophy, with vascular risk factors and right infarct. In this report we present a case who presented with manic symptoms and evaluated as late onset mania secondary to stroke of left hemisphere.

Case: A 46 year-old male patient that presented to our outpatient clinic due to elevated mood, reduced need of sleep, pressured speech, thoughts race, elevated energy, and disinhibition with a recent onset of 3 months. Although he was hospitalized in an inpatient psychiatric unit, after a brief silent period the worsening of the symptoms led his family to contact with our outpatient psychiatry services. Three years ago, he had suffered acute right-sided limb weakness and he was admitted at the neurology inpatient service. Computed tomography scan and brain magnetic resonance imaging revealed the left parietal acute infarct. After 3 years he began to suffer from of manic mood and behavior. Neither the patient, nor his family had a history of psychiatric disorder. Physical and neurological examination were normal except for a minor weakness in right extremities. Psychiatric interview led to the diagnosis of late onset mania secondary to stroke. The patient started pharmacological therapy with quetiapine 400 mg/day and lithium 900 mg/day. After a month, the psychiatric symptoms were remarkably improved with therapeutic plasmatic levels of lithium 0.78 mEq/Lt.

The most relevant brain imaging findings in late onset mania are silent cerebral infarctions and also subcortical lesions. These findings, however, need to be considered in the evidence of higher cerebrovascular risk factors in patients with late onset bipolar disorder that may predict a poor prognosis compared to early onset bipolar disorder patients. Post-stroke mania should be considered in any manic patient who presents concomitant neurological focal deficits and is older than expected for the onset of primary mania.

Keywords: late onset, stroke, mania, left hemisphere

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[Abstract:0133][Psychopharmacology]

Olanzapine -induced tardive oculogyric crisis: case reports

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Tardive oculogyric crisis one of the extrapyramidal syndromes who chronically antipsychotic-treated patients and usually reported as a subtype of tardive dystonia. Tardive dystonia occurs in about 3% of patients during long-term antipsychotic treatment and may be associated with dopamine receptor antagonism. Initial symptoms may include restlessness, agitation, malaise or a fixed stare followed by the more characteristically described maximal upward deviation of the eyes in a sustained fashion. The eyes may also deviate upward, laterally or downward. Backward and lateral flexion of the neck, wide open mouth, tongue protrusion and ocular pain are the most frequently reported associated findings. Additionally, they would define auditory hallucinations and exacerbation of psychiatric symptoms.

Case 1: A 45 year-old female patient, living with her family. She met the DSM-IV criteria for bipolar disorder, since she was 18 years old. Since 2005, she had been receiving regular treatment including lithium 900 mg/day, carbamazepine 800 mg/day and olanzapine 10 mg/day. The patient complained to reversible and repeater oculogyric crisis, has reported feelings of anxiete and restlessness during crisis nearly 6 years. There was no familial history of movement disorder. After thorough neurological examination and brain imaging revealed

no other abnormalities, tardive oculogyric crisis was diagnosed, olanzapine dosage had been reduced was switched to clozapine 150 mg/day. The frequency and duration of oculogyric crisis markedly decreased after one month.

Case 2: A 19 year-old male patient, living with his family. He met the DSM-IV criteria for schizophrenia, since 2013. He had been receiving olanzapine 15 mg/day for two years. The patient complained to reversible and repeater oculogyric crisis, has reported auditory hallucinations, restlessness and agitation during crisis for one year. After thorough neurological examination and brain imaging revealed no other abnormalities, tardive oculogyric crisis was diagnosed, olanzapine dosage had been reduced was switched to clozapine 200 mg/day. There is no described oculogyric crisis after two month.

Tardive oculogyric crisis is rare side-effect of long-term antipsychotic treatment and can easily be overlooked. Recurrent exacerbation of psychotic symptoms associated with episodes of oculogyric crisis may prompt the clinician to enhance the dose of antipsychotic medication. Discontinuing or tapering the dose of antipsychotic drug or switching to a drug with lesser risk of extrapyramidal side effect is recommended especially clozapine treatment. In both cases, olanzapine-induced tardive oculogyric crisis has occurred and this side-effect decreased with switched to clozapine treatment.

Keywords: tardive dystonia, olanzapine, clozapine

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[Abstract:0137][Psychosomatic medicine-Liaison psychiatry]

Case report: artifacts dermatitis

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Artifacts dermatitis is primarily a psychiatric illness but known as among psychodermatologic diseases due to created lesions in the skin. Serious difficulties in the diagnostic process is experienced because it has a wide variety of morphological characteristics of the lesions, imitating many skin diseases.

Case: A 36 year-old female patient, married, works as house cleaner. The patient who presented to dermatology clinic by reason of the wounds on the right arm was evaluated by a psychiatric consultation. Her complaints were demoralization and insomnia. She had been evaluated by us and a diagnosis of depression was made one and a half years ago. During the examination of the patient she often declared that she was not at a good financial situation and often she was ruminating this subject on his mind. She said she had presented to health board 4 times for maintenance salary in the last 2 years, but it was unable to, she said she would apply again. In her mental status examination; she was in depressed mood, her affect was superficial, she had a facial expression ignores lesions. In her thought content there was financial problems. In dermatologic examination nonspecific excoriation, erosion and necrotic lesions were present in the easy reachable regions of arms and legs of the patient (Figure 1 and 2). Other physical examination findings and laboratory values were normal. Psychotherapeutic interviews underwent regularly with patient. Sertraline 50 mg/day was started. While she expressed the lesions arouse spontaneously during the first interview she sad lesions might have been the result of being in contact in with the washing water in the next meeting. Artifacts dermatitis is a psychodermatologic disease that the diagnosis process is very difficult for physicians. Various methods such as pulling, etching, chemical exposure is can be used to formation of lesions. Significantly seen more in women, age range is variable. Mainly, this illness is located in the obsessive-compulsive disease group but the underlying psychopathology may range from depression to psychosis, and personality disorders. Extreme mental activities related to the financial situation, applying history to health board and going on to apply again and again suggested us that it may be malingering. However indifferent attitude towards existing lesions rather than the extreme physical or psychological symptoms is thought-provoking for malingering. While declaration of the welcome the interest of the family after the brain surgery and wandering around the hospital with open lesions in patient's head suggest that diagnosis may be factitious disorder, the lack of input and redundant multi-hospital medical treatment and/ or absence of surgical history was away from factitious disorder.

Gender of the case, atypical structure of lesion and blurred story suggests that it may be artifacts dermatitis. The presence of various psychosocial stressors, the history of dealing with corrosive substances, the history of following up with depression and secondary gains in the questioning suggested our diagnosis. Physicians the should be more careful in the patients with underlying psychiatric problems cause various clinical manifestations, especially in atypical cases who has suspectful medical history and physical examination.

Keywords: artifacts dermatitis, psychodermatologic diseases, skin lesions

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[Abstract:0141][Schizophrenia and other psychotic disorders]

Treatment of clozapine-resistant schizophrenia with paliperidone: a case report

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One third to one fifth of the schizophrenia patients are thought to be the treatment resistant. Clozapine is the only antipsychotic whose efficacy has been shown by a clear evidence in treatment-resistant schizophrenia. In addition, the other significant proportion of the patients (40% to 70%) are inadequate or partial responders to a clozapine treatment. In the other words, the therapeutic efficacy of clozapine is reported to be 30-50% in treatment-resistant patients. 1/3-2/3 of the patients are resistant to clozapine monotherapy. In this study, we presented 51 years-old female patient diagnosed as paranoid schizophrenia for 35 years who followed by clinicians at our in-patient clinic many times and thought to be treatment-resistant disease and clozapine initiated. When the treatment of clozapine was regulated as 600 mg/day, some side effects (excessive sedation, severe skin rashes including whole body, sialorrhea) and inadequate treatment response were seen so switched to treatment with paliperidone. The symptoms of patient were improved with adding depot palliperidone palmitate to oral paliperidone administration by the time.

Case: A 54 year-old female patient, literate, housewife, was married; but her husband had died 15 years ago, lives in Kalandere/Rize with her family. She was brought to the outpatient clinic by her mother with complaints of aggression, restlessness, susceptibility and suspicion about other relative members, self-talk, auditory and visual hallucinations, smacking of the mouth, rolling of tongue and licking herself for a month. She had accepted to psychiatry service for two times at past and multiple pharmacological agents had been administered (risperidone, olanzapine, quetiapine, amisulpride) to control her complaints in adequate dose and duration. Although she have used multiple pharmacological agents, her complaints (delusions, visual and auditory hallucinations, psychomotor excitement, aggression, restlessness, susceptibility and suspicion) were going on. In addition of this situation, tardive dyskinesia had been appeared due to prolonged and multiple antipsychotic treatment. With considering all data (treatment resistant, existence of tardive dyskinesia) clozapine was thought to start as a monotherapy. When the treatment of clozapine was regulated as 600 mg/day, some side effects (excessive sedation, severe skin rashes including whole body, sialorrhea) and inadequate treatment response were seen so switched to treatment with paliperidone.

Developmental History: The patient reported that she was born following full-term pregnancy with normal delivery, normal weight and without any complications. There were normal growth and development process and not had a big problem. Parents are alive and healthy. There was no significant medical history, history of head trauma and substance abuse, psychiatric and neurological disorders; but grandmother had a psychotic disorder. The results showed normal cranial MRI, sleep EEG, normal blood tests.

Mental Status Examination: The female patient, who looked an older than chronological age, looked poor self care and behaved with respect. She had no eye contact. Her speech was slow, tangential and had a poor content. The answers of the questions were short and time of the reaction was long. Her affect was blunt and she had a depressive mood. She was conscious and her orientation was complete. She had visual and auditory hallucinations. Her ability of intangible thought, assessment of reality and judgment were impaired. Her thoughts include delusions of persecution and reference.

Partial psychomotor retardation and loosening of associations were exist. Her self-respect and social participation were poorly.

Follow-up: After physical examination was done at outpatient clinic, due to DSM-5 schizophrenia was thought then the patient was accepted to the psychiatry clinic. When we review the literature; due to treatment resistant and tardive dyskinesia, starting of clozapine was thought to be the most appropriate decision. After the treatment of clozapine was regulated as 600 mg/day in two months, some side effects (excessive sedation, severe skin rashes including whole body, intolerable salivation) and inadequate treatment response were seen. If we examine the medical history, irregular use of the oral antipsychotics can be seen for this patient; so that new generation depot antipsychotics like paliperidone should be considered for use once in a month intramuscular. At the same time, the patient have never tried paliperidone up to day. Thus clozapine treatment was switched with paliperidone. The dose of paliperidone was started as 3 mg/day and increased up to 9 mg/day. 75 mg/month depot paliperidone also was added to oral paliperidone treatment as an intramuscular once a month. Day by day the complaints of patient decreased. When we administered The Scale for the Assessment of Positive Symptoms (SAPS), the Scale for the Assessment of Negative Symptoms (SANS), Extrapyramidal Symptom Rating Scale (ESRS); Clinical Global Impressions Scale (CGI); satisfactory improvement was observed. The patient was discharged with partial recovery.

Insufficient response or intolerable side effect of clozapine directs clinicians to the other choices or augmentation of clozapine with other drugs. Unfortunately, this is not clear that how to approach to the patients who can't use clozapine or unable to do the titration of adequate dose because of the side effects (agranulocytosis, leukopenia, myocarditis, cardiomyopathy, paralytic ileus etc.) and do not respond to the combination of the other treatments to clozapine. There are studies like adding typical or atypical antipsychotics to

clozapine that have conflicting results. There is not enough information about transition to different antipsychotic drugs when the patient failed to respond to clozapine.
Keywords: schizophrenia, clozapine, treatment resistance, paliperidone

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[Abstract:0143][Psychotherapy]

Eye movement desensitization and reprocessing (EMDR) treatment In social anxiety disorder

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Social anxiety disorder (SAD) also known as social phobia is an anxiety disorder characterized by an intense fear in one or more social situations causing considerable distress and impaired functioning in at least some parts of daily life as defined in DSM-5. This situation cause personal distress and impairment of functioning in one or more domains, such as interpersonal or occupational functioning. The most common psychotherapeutic treatment approach in SAD is cognitive behavioral therapy. Much as EMDR is a proven psychotherapeutic approach in post traumatic stress disorder, there are studies about efficiency in some other psychiatric disorders.

Case: A 29 year-old single male patient with social anxiety disorder, graduated from the university and unemployed. He presented to Bitlis State Hospital with complaints such as tremor in hands, fear of being in community and overexcitement in some conditions. He told that, he cannot talk easily with strangers and also with his friends and family members. He had fear of being laughed at in community therefore he always had behavioral avoidance from the community. He never had a girlfriend before. His symptoms have been present for 8 years. He has been using psychiatric medication since the beginning of the symptoms. He told that he used sertraline, fluoxetine, paroxetine, mirtazapine, olanzapine, clonazepam and alprazolam before. He indicated that only alprazolam and clonazepam medications worked. In the last 2 years, he had cognitive behavioral therapy and still using bupropion 300 mg/day, escitalopram 10 mg/day, propranolol 40 mg/day and indicated he still has symptoms significantly when he first referred to us. He told that there was no psychiatric disorder in his family. Before his psychiatric treatment he had completed his blood tests, cranial magnetic resonance imaging and no organic pathology had been determined. After a detailed psychiatric examination he was diagnosed with social phobia according to DSM-5. No additional psychiatric disorder was observed. EMDR therapy was decided to be administered and the patient was informed about the EMDR. To evaluate the efficiency of therapy Liebowitz social anxiety scale (LSAS), Beck depression scale (BDS) and Beck anxiety scale (BAS) were administered at the beginning of the therapy, just after 4 sessions of EMDR therapy and after 6 months beginning from the therapy.

Although only proven treatment field of EMDR is post traumatic stress disorder, there are articles and case reports that EMDR is working on other psychiatric disorders such as specific phobias, pain disorders and vaginismus. Single or combined usage of medical agents and cognitive behavioral therapies is the most common and effective method in treatment of social phobia. In this case, 4 sessions of EMDR therapy were administered to the patient, LSAS, BDS and BAS scores significantly decreased. EDMR therapy can be an alternative treatment method in social phobia.
Keywords: social anxiety disorder, EMDR, psychotherapy

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[Abstract:0144][Mood disorders]

Three pregnant bipolar patients treated with quetiapine with manic episodes

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Bipolar disorders (BD) are reported to have a high incidence during childbearing years and the need for pharmacological treatment may arise to start or continue during pregnancy and the postpartum period. Quetiapine is an atypical antipsychotic which is effective in treatment of BD during pregnancy and shows the lowest value of placenta transition with a rate of 23.8%. Even though studies with humans are very limited with respect to embryonic/fetal risk assessment, the incidence does not seem to be higher than in the general population nor are the malformation specific. We report three cases treated with relatively higher doses of quetiapine throughout pregnancy without any pre/peri/post natal complications but with healthy deliveries.

Case: The first case: a 30 year old female patient, with a history of BD since age 18. When she had learned that she was pregnant during routine monitoring, she was taking quetiapine 1600 mg/day and lithium 600 mg/day, but the patient had discontinued the use of the drugs and referred to our psychiatry clinic at the 8th week of her pregnancy. She was hospitalized with diagnosis of bipolar disorder manic episode with psychotic features. Quetiapine was administered and dosage was increased to 1000 mg/day in 4 weeks and at the end of 4 weeks, six session of electroconvulsive therapy had been administered. Two weeks after discharging she had discontinued the use of drugs and her symptoms were reappeared. Eight sessions of electroconvulsive therapy performed because of ongoing psychomotor agitation, quetiapine dosage increased up to 1200 mg/day within five weeks time. She had no side effects and no symptoms during treatment and gave birth to a female infant at 39th weeks.

The second case: a 35 year old patient was taking 2000 mg/day of valproate, 1200 mg/day of lithium were gradually decreased because of planning pregnancy. She was hospitalized with psychotic mania on 13th week of pregnancy. Quetiapine was increased to 800 mg/day, because of her ongoing agitation, 12 sessions of electroconvulsive therapy was administered with haloperidol 10 mg/day treatment. Her symptoms showed considerable improvement at week 19, without any complication, the baby was delivered healthy.

The third case: a 33 year-old female patient, was taking 1000 mg/day valproate but discontinued because of 6th week of pregnancy. After ten days, she was hospitalized with manic episode. Because of her aggression and disinhibition, quetiapine dosage was increased up to 900 mg/day, within three weeks. Clinical improvement was monitored at the 5 week. Her maintenance dosage of 600 mg/day reduced to 300 mg/day at week 24, finally stopped just before the healthy delivery. In literature cases usually received 300–400 mg/day or 600 mg/day dosage of quetiapine. But in one case, a higher dosage of quetiapine (1200 mg/day) at 21st week of pregnancy with healthy outcomes in babies was reported. In our cases quetiapine was administered higher doses than others, but no teratogenic or neuropsychiatric anomaly was noted.

In this case series report, no negative results have been found in terms of teratogenicity or any complications in the cases of three pregnant women, being treated with respectively higher doses of quetiapine (1200, 800, 900 mg/day) in manic episodes. So we can say that quetiapine even though relatively higher dosages can safely be used during pregnancy.

Keywords: bipolar disorder, mania, pregnancy, quetiapine

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[Abstract:0147][ADHD]

Worsening of ADHD symptoms with sugar consumption in a 7 year-old boy: a case report

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Attention deficit and hyperactivity disorder (ADHD) is one of the most common childhood onset psychiatric disorders, prevalence of which is approximately 4–9% in children. According to a theory, sugar consumption may have a role in ADHD, and parents are often of the opinion that acute ingestion of sugar may cause bouts of hyperactivity in their children, followed by sedation and inactivity. However, performed studies had contradictory results.

Case: A 7 year-old boy, without any other medical disease, presented to our clinic with impulsiveness, attention deficit, excessive talking,

hyperactivity and school and peer relationship problems. Parents also spontaneously gave a story of worsening in symptoms with sugar intake. He had an ADHD diagnosis before, but did not take any medication. 10 mg long-acting methylphenidate (Medikinet Retard) was prescribed to him and one month later, a partial improvement was observed but parents' complaint of agitation after sugar intake continued. During clinic examination, one cube sugar was given to him and in 30 minutes, obvious hyperactivity was observed. An endocrinology consultation was requested but his all laboratory analyses were between normal levels. For the second month, termination of sugar was recommended and 20 mg long-acting methylphenidate was given and his symptoms totally improved. With 20 mg MPH, a only partial hyperactivity after sugar intake was seen. It is still controversial whether or not there is an association between ADHD and sugar consumption. Some early studies supported the concept that increased intake of added sugars may have a role in ADHD, as studies performed in the 1980s appeared to rule out sugar as a likely cause of ADHD4. Recently, chronic effects of sugar has been studied. According to them, in some subjects, the initiating process that leads to the development of ADHD is excessive sugar (or sweetener) intake, resulting in enhanced dopamine release. Over weeks to months, this leads to a reduction in D2 receptors and D2 receptor-mediated signaling. In response, sugar intake increases. However, over time, the dopamine response to sugar slowly decreases, and the intervening periods are associated with a reduction in striatal dopamine levels.
In conclusion, frontal lobe sensitivity to natural rewards is reduced, resulting in the development of behaviors such as overeating and ADHD. In conclusion, a reduction in sugary food intake is offerable as it has also general health benefits and carries no risk as our case.
Keywords: attention deficit and hyperactivity disorder, sugar consumption

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[Abstract:0153][Others]

Gardner diamond syndrome in a 15 year-old girl

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Psychocutaneous disorders are characterized by psychiatric and skin manifestations. Gardner-Diamond Syndrome is one of psychocutaneous disorders. GDS, or autoerytrhocyte sensitization syndrome is characterized by recurrent, painful, transient bruises, which appear without any specific causes, but sometimes can occur after emotional stress and trauma. We report an adolescent girl with Gardner Diamond Syndrome who has also having psychiatric co-morbidities.
Case: A 15 year-old girl presented with a history of recurrent bruise like lesions on the face. The lesions used to appear abruptly. Lesions occurred since five years and generally at intervals of 3-4 months. The lesions improved spontaneously within a week. The exhaustive laboratory investigation were normal. After these examinations, the patient was referred for psychiatric evaluation. On the examination, the patient was having depressive symptoms since a year. The detailed psychiatric evaluation, which was made via Kiddie-Sads, revealed the patient had major depressive disorder, PTSD, social phobia. In the first session, the patient had lesions. The patient was exposed to physical and sexual abuse by her father for about 4 years. After first session and evaluation, the diagnosis was Gardner-Diamond Syndrome, and the patient was started to treat with escitalopram 10 mg/day. Gardner Diamond syndrome is a rare and poorly understood clinical presentation of unexplained painful ecchymotic lesions. Skin lesions are at first preceded by burning and itching and a stinging sensation or pain, painful edematous pink or red plaques of variable sizes become visible. Lesions may become less tender and eventually disappear completely within a week. The most reliable diagnostic test is, in fact, intradermal injection of the patient's own washed erythrocytes which is called as autoerytrocyte sensitization test. In our patient, all hematologic tests were normal, and sensitization test result was positive. Diagnosis is mainly clinic, where previous episodes of physical or emotional stress, combine with typical skin lesions, usually in women with regular coagulation parameters. In our patient, hematologic and dermatologic evaluations were normal, the patient was referred to the child and adolescent liaison psychiatry. After assessment and rule out other bleeding causes and revealed that physical abuse by the father was not the cause of lesions, diagnosis was made as Gardner-Diamond Syndrome.
Various associated psychopathologies include depression, anxiety, emotional instability, masochism, as well as conversion. In our patient, Kiddie-Sads revealed the patient had major depressive disorder, PTSD, social phobia, and her past diagnosis was separation anxiety disorder and primer enuresis nocturna.
Treatment of GDS is still a particular problem. Psychiatric treatment in young age groups such as children and adolescents. In our patient, we started escitalopram treatment and supportive therapy, during treatment lesions were not occurred again.
Keywords: gardner diamond syndrome, psychogenic purpura

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[Abstract:0157][ADHD]

Development of tics and recovery of enuresis nocturna with atomoxetine in a 9 year-old boy with ADHD

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Attention deficit hyperactivity disorder (ADHD) is a common problem which presents in 5-10%of children and 3% of adults on 14% of cases accompanies with tic disorders. Also over half of Tourette Syndrome patients suffer from ADHD. Enuresis is also a common comorbid disorder in children with ADHD, with a rate of about %30. Although stimulants are associated with occurrence of new tics and exacerbations of the existing tics and treatment of ADHD with atomoxetine is recommended in case of comorbid tic disorder, there are several case reports contradictory to that notion. In addition, enuretic symptoms have decreased dramatically in some children with atomoxetine in clinical research studies. Here we present a case of development of new motor tics and significant increase in the number of dry nights in an ADHD patient with nocturnal enuresis (NE) with atomoxetine use.
Case: A 9 year-old boy presented to our outpatient clinic with hyperactivity, frequently losing his properties, walking around in the classroom during lesson, bothering his friends, difficulty in completing activities and maintaining attention, and enuresis nocturna. He had also continued to bed–wetting six-seven times in a week, despite behavioral treatment. He was consulted with nephrology but no organic etiologic reason was found. After his psychiatric examination, psychometric evaluations and teacher's observation form for ADHD, we diagnosed him as ADHD-combined type and comorbid NE. Atomoxetine 10 mg/day was prescribed for first week and the medication was increased to 35 mg by the fourth week. Two months after atomoxetine treatment, his NE symptoms dramatically recovered and an obvious improvement in his ADHD symptoms was observed. However, he developed tics as twinkling and mouth movements which were affecting his daily functions at home and at school. We did not stop atomoxetine because of its beneficial effects on ADHD and EN. We added risperidone 0.5 mg/day. One month after combination therapy, his tics were totally resolved and there was no other side effects due to medications.
Atomoxetine is the first FDA approved non-stimulant drug for ADHD. Treatment of ADHD with atomoxetine has been shown to result in decrease in occurrence of new tics and exacerbation of existing tics; however there are several case reports contradictory to that notion. Atomoxetine increases norepinephrine levels in the presynaptic cleft by blocking the action of the norepinephrine transporter. However as a result of increased levels of norepinephrine, it might affect dopaminergic system and may result in occurrence of new tics or exacerbation of existing motor tics. Enuretic symptoms also have decreased dramatically in some children with atomoxetine in clinical research studies. Noradrenergic effect of atomoxetine on bladder contractility, bladder capacity and detrusor relaxation may be effective in treating NE.
In this case after initiating a treatment of atomoxetine, a cessation of enuresis and new motor tics occurred in a patient with ADHD and comorbid NE. Further studies are necessary to support these findings and elaborate on appropriate usage.
Keywords: ADHD, atomoxetine, tic, enuresis nocturna

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[Abstract:0162][Psychopharmacology]

Methylphenidate -induced increase in intraocular pressure in a patient

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Attention Deficit and Hyperactivity Disorder (ADHD) is a neurobiological disorder presenting with impairment in attention, hyperactivity and impulsivity. Metylphenidate, which is a well known and commonly used central nervous system stimulant is one of the agents used in the treatment of ADHD. Most frequently seen side effects are difficulty in falling asleep, decreased appetite, stomach ache and headache. Also some ocular side effects may be presented. Here we aimed to discuss a case with methylphenidate -induced increase in intraocular pressure.
Case: A 23 year-old female patient, single, university student living with her family. 2 months ago she had presented to psychiatry clinic because of difficulty in sustaining attention especially during listening and reading. She had also complained of easily getting bored by

activities and decrease in school performance. She was given the treatment of methylphenidate 18 mg/day. She had used this medicaiton for 2 months regularly. When she presented to our center, she claimed that some of her complaints were gone away and some were better. In her psychiatric history, it was understood that she had had complaints since early childhood, but she did not seek help. In her psychiatric examination there were signs of distractibility, difficulty in concentration, anxiety, irritability and impulsivity. In addition to these signs the patient had complained of blurred vision increasing gradually for 2 weeks. She was referred to ophthalmology clinic, and it was reported that her intraocular pressure (IOP) was high, 32 mmHg; and there were hemorrhagic lesions on optical disc. As she had never had pre-existing problems related to eye and vision; these signs are thought to be adverse effects of methylphenidate. In result methylphenidate was stopped. In following dates on 2nd week and 4th week intraocular pressure of the patients have decreased to values 25 mmHg and 18 mmHg respectively. In conclusion, regarding our case and other literature results we concluded that methylphenidate treatment in short duration and even in low doses may effect intraocular pressure. Therefore intraocular pressure and visual problems should be taken account before and after prescribing stimulants as methylphenidate. Also clinicians should inform ophthalmologist about increased intraocular pressure and other potential intraocular changes due to methylphenidate treatment by time of referral.

Keywords: ADHD, methylphenidate, intraocular pressure

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[Abstract:0168][Autism]

A case report: does risperidone improve hyperacusis in children with autism?

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Hyperacusis has been defined as 'unusual tolerance to ordinary environmental sounds' or 'consistently exaggerated or inappropriate responses to sounds' that are neither threatening nor uncomfortably loud to a typical person. In the great majority of cases, no underlying medical condition can be found, but it can be related with some diseases (e.g., Lyme disease), syndromes (e.g., Williams Syndrome), mental disorders (e.g., depression, PTSD) and drugs.

Case: An 11 year-old boy with autistic spectrum disorder presented to our clinic with a complaint of "auditory hypersensitivity" by his parents. He has been attending special education since he was diagnosed as autism and now he is attending elementary school, in a class for children with special needs. He learned reading and writing last year. His teachers are pleased with him and they do not state behavioral problems with risperidone treatment. Hyperacusia has always been present but the severity of it increased 3 years ago when Risperidone was prescribed 1 mg/day to the patient because of hyperactivity symptoms. Because of hyperacusis Risperidone was discontinued and hyperacusia decreased. Atomoxetine and then aripiprazole were prescribed but they did not reduce hyperactivity, so risperidone was prescribed again.

He is especially disturbed by the voice of call to prayer, children and electric appliances. He covers his ears not to hear the voices. Nowadays this situation started to affect his functionality. According to his parents he is so disturbed in the classroom that he can't ever hold the pencil to write something because of covering his ears. During clinical assessment he made little eye contact either when listening or speaking and his non-verbal interaction was limited. His answers to questions were minimal. He was given bender gestalt visual motor perception test and he completed successfully. The patient referred to ENT and pediatric neurology to exclude organic etiology. Physical examination and screening was in normal range. Risperidone treatment discontinued and hyperacusia decreased again. Autistic spectrum disorder is a neurodevelopmental disorder characterized by qualitative impairments in social interactions and communication skills, along with a restricted repetitive and stereotyped pattern of behavior. In addition to these core features, the coincidence of sensory processing abnormalities in autistic disorder has been reported at a frequency of 42–88%. Auditory hypersensitivity is the most common sensory impairment, interrupting behavioral adaptation. In the literature there is only one case about risperidone-related hyperacusis in a 5–year-old girl with autism; hyperacusis of whom was improved after taking risperidone, disappeared after discontinuation of the medication and re-occurred in the re-challenge test.

To the best of our knowledge, our case report is the second one about risperidone-related hyperacusis in autism. Clinicians should be aware of such rare side affects of antipsychotic use, and monitor patients closely. Parent education will also help to increase the awareness, especially in children with autistic spectrum disorders, such as in this case.

Keywords: autism, hyperacusis, risperidone

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[Abstract:0173][Psychopharmacology]

Aripiprazole augmentation in treating kleptomania: a case report

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Kleptomania is a distressing and disabling disorder by ever-increasing tension leading up to the theft, and an intense feeling of gratification or relief after. While kleptomania does cause strong impulses to steal, these stealing events are not triggered or accompanied by hallucinations or feelings of anger or revenge. Although the disorder has been known to psychiatrists for a long time, the cause of kleptomania is still ambiguous. Therefore, a diverse range of therapeutic approaches have been introduced for its treatment. These treatments include: pharmacotherapy, psychoanalytic oriented psychotherapy and behavioral therapy. In the treatment of kleptomania, various antidepressants, lithium, other mood stabilizers and even naltrexone is used. Aripiprazole is an atypical antipsychotic drug that is reported to be a high-affinity D_2-dopamine receptor partial agonist. Aripiprazole has a wide range of use in psychiatric disorders as monotherapies and augmentation therapies. In literature, there is not enough data about augmentation therapies for Kleptomania. In this case report, we report the results of aripiprazole augmentation therapy in a patient with kleptomania.
Case: Our patient was a 42 year-old female patient, housewife, who is fulfilling DSM-5 criteria for kleptomania. She was given various antidepressant monotherapies such as fluoxetine, paroxetine, escitalopram, sertraline at the appropriate dose and duration for two years in different hospitals. Despite receiving antidepressant treatment, she couldn't resist to the urge to steal small objects in markets and news stands. She was stealing especially magazine ads and small promotion products. None of the stolen goods were to meet her any need. She was also in the process of a criminal case. She was receiving 200 mg/day of sertraline for two months. 5 mg of Aripiprazole was added to her treatment. In the sixth week of aripiprazole augmentation, she was stating that her urge to steal was completely reduced and she was not stealing anymore. The remission of her kleptomania remained in the six months follow up.
Kleptomania is a rare impulse control disorder with poor prognosis. Kleptomania, can lead to legal and social problems if not treated. In certain cases, augmentation therapies in order to improve the efficacy of SSRIs can be useful. An SSRI augmentation by additionally administering Aripiprazole may be useful for the treatment. Aripiprazole remains a viable option in augmentation therapy with low side effect profile.
Keywords: kleptomania, aripiprazole, augmentation, impulse control disorders

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[Abstract:0174][Psychopharmacology]

Reversible cycloplegia caused by duloxetine: a case report

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Duloxetine is a balanced and potent dual reuptake inhibitor of serotonin and norepinephrine (SNRI) that has previously been shown to be effective in the treatment of major depressive disorder (MDD), generalized anxiety disorder and diabetic peripheral neuropathic pain (DPNP). Comorbid somatic symptoms increase the use of duloxetine for depressive symptoms. Absorption of duloxetine begins two hours after oral administration, reaching a maximum plasma concentration in six hours. Half-life and volume of distribution are 12 hours and 1640 liters, respectively. Similar remission rates with selective serotonin-reuptake inhibitors (SSRIs) increase the use of SNRIs in MDD. The adverse effects of duloxetine are similar to traditional SSRIs. Most common adverse effect of duloxetine is nausea that is generally associated with discontinuation. Other common adverse effects are constipation, insomnia, hypersomnia, dizziness, weakness, drowsiness, sedation, fatigue, diarrhea, headache, and xerostomia. With the increased use of duloxetine, clinicians have begun to encounter different adverse effects. Cycloplegia is paralysis of the ciliary muscle of the eye, resulting in a loss of accommodation.We present a reversible cycloplegia caused by duloxetine use with this case report.
Case: A 24 year-old female patient who has been diagnosed with MDD. She has had somatic symptoms like fatigue, myalgia and headache, besides her depressive symptoms like anhedonia, feeling of unworthiness, pessimistic thoughts, irritability, reduced self

confidence, aversion, lack of appetite and insomnia for last two months. She was examined in internal medicine and neurology outpatient clinics in last month and her blood tests and cranial imaging tests revealed no problem. She did not have suicidal thought or any psychotic symptom. She mentioned that she used escitalopram and sertraline for MDD four years ago, when she was at college education. She stated that she had to quit escitalopram and sertraline owing to side effects such as nausea and drowsiness. Duloxetine 30 mg/day treatment was begun in our outpatient clinic. In her first routine control at the end of first week, she suffered from light sensitivity and an increasing visual impairment. The visual impairment led dizziness and an increase in headache. She was consulted to ophthalmology unit of our hospital and cycloplegia detected in her eye examination. Duloxetine was stopped in the 9th day of treatment but cycloplegia negatively affected the patient's daily life for almost 4 weeks and impaired her functionality. Cycloplegia is paralysis of the ciliary muscle of the eye, resulting in a loss of accommodation. Because of the paralysis of the ciliary muscle, the curvature of the lens can no longer be adjusted to focus on nearby objects. Antidepressants have various ocular and visual adverse effects. For instance, the tricyclic antidepressants produce many anticholinergic side effects. Symptoms of blurred vision, cycloplegia and dry eye are transient and reversible. Reducing or changing the medication may improve the symptoms. In some cases, near vision lenses may be helpful. SSRIs are known not to have any significant ocular effects. Eye pain, changes in vision and swelling or redness in or around the eye are mentioned as possible visual side effects in the medication of duloxetine. The ocular and visual side effects from a patient's systemic medication can range from mild to severe. These side effects may or may not be serious enough to warrant discontinuing treatment. Recognition of ocular and visual side effects is important to prevent and minimize serious complications.

Cycloplegia seems as a rare adverse effect in antidepressant treatment and may take a long time to wash out. In case of visual disturbances, eye examination of the patient should be performed and the responsible drug should be discontinued as soon as the cycloplegia detected.

Keywords: duloxetine, cycloplegia, accommodation disorder, adverse effect

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[Abstract:0175][Psychopharmacology]

Quetiapine-induced peripheral edema in a patient with opiate dependence

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Peripheral edema could be caused by various medical conditions, such as obstruction of venous or lymphatic drainage, congestive heart failure, nephrotic syndrome, cirrhosis and medication. Common pharmacologic agents known to cause edema are antihypertensives, nonsteroidal anti-inflammatory drugs, endocrine agents and immunotherapies. Peripheral edema has been infrequently described with several atypical antipsychotics, including case reports with olanzapine and risperidone. Only a handful of cases of peripheral edema associated with quetiapine use have been reported. Most of these reports have occurred while patients have been on treatment with a combination of medications. Here, we present a case of quetiapine-induced peripheral edema in a patient with opiate dependence.

Case: A 39 year-old male patient presented to our hospital with heroin use for six years. He was diagnosed as opiate dependence and hospitalized to inpatient unit. Before psychiatric admission, he was not taking any medications. The initial examinations on admission revealed no remarkable medical conditions. He was started on sublingual buprenorphine–naloxone 8 mg/day, quetiapine 100 mg/day, and diclofenac potassium 100 mg/day. Two day after initiation of these treatments, the patient noticed bilateral swelling in his ankles. Edema gradually increased with treatment. During the first week of treatment, edema was prominent. There was no evidence of any rash, skin thickening, ulceration or pigmentation. Physical examination and investigation revealed no other abnormalities: pulse, blood pressure and temperature were normal. There were no signs of cardiac failure, pulmonary edema, varicose or spider veins on the legs. He had no known drug allergies and there was no other systematic illness. Laboratory results including complete blood cell counts, albumin, serum electrolytes, liver–renal–thyroid function were within normal limits. There was no eosinophilia. Hepatitis B and C surface antigens were negative. Due to suspicion of drug-related effects, quetiapine was discontinued immediately. The peripheral edema decreased in the first day and was completely resolved within 3 days.

The case mentioned above was evaluated as quetiapine -induced peripheral edema because that edema gradually dissolved when the drug was discontinued. Also the absence of any systemic disease, clinical and laboratory findings explaining edema have supported our opinion. The mechanism of peripheral edema caused by quetiapine remains unclear. A number of plausible mechanisms have been proposed for the peripheral edema associated with atypical antipsychotic agents. These mechanisms include vasodilatation and edema

due to alpha 1 receptor antagonism, allergic reactions mediated by immunoglobulin E and also at least two mechanisms that cause relaxation of smooth muscle. Since the precise explanation of quetiapine-induced peripheral edema remains unclear, further research is warranted to understand this condition. The case report discussed here is expected to help the clinicians to be vigilant to identify this uncommon side effect that requires no intervention, except monitoring of edema, once the causative agent has been removed.
Keywords: quetiapine, peripheral edema, opiate dependence

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[Abstract:0176][Psychopharmacology]

Duloxetine-induced hypertension: a case report

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Serotonin-noradrenaline reuptake inhibitors (SNRIs), are the second generation drugs used in the treatment of depression. Since venlafaxine hit the market in 1993, all SNRIs has become common and reliable choice for psychiatrist in treatment of depression. They are the second most commonly prescribed antidepressant drugs after the selective serotonin reuptake inhibitors in European countries. Duloxetine, one of SNRIs, are used in treatment of anxiety disorder, fibromyalgia, diabetic neuropathic pain as well as depression treatment. The most common side effects of duloxetine are nausea, dry mouth, dizziness, decreased appetite, constipation and insomnia. SNRIs may lead to them via the noradrenergic increased blood pressure in addition to their effects on cardiovascular side effects. The placebo-controlled trials of venlafaxine more than 200 mg/day doses cause a 5% clinically significant increase in blood pressure (especially diastolic blood pressure) has been reported. In the literature, hypertension cases have been reported associated with the use of milnacipran and venlafaxine in SNRIs. However, duloxetine has been reported to increase the blood pressure of 1-2 mmHg, one case was observed that cause hypertension.

Case: A 53 year-old female patient presented that hypertension symptoms appeared after duloxetine treatment. The patient has had a history of depression for ten years, treated with fluoxetine 20 mg/day regularly and trazodone 50 mg/day when needed for insomnia. Her condition had been stable until three months ago when she felt insomnia, depression, and irritability without significant causes. Thinking her depression might have relapsed, she presented to the our psychiatry clinic who recommended she take duloxetine 30 mg/day, instead of fluoxetine. The dosage of duloxetine was gradually increased to 60 mg/day over two weeks. The depressive symptoms were well controlled within three weeks of initiation of duloxetine. The patient is a housewife, has been postmenopausal for 4 years, always adheres to physical exercise, and is usually physical healthy. She has never taken estrogen and has no history of other substance abuse, surgery, trauma or chronic systemic medical history. There was no family history of hypertension. ECG, echocardiogram, and endocrine evaluation were all normal. Her BMI is 25.0 kg/m^2 and the vital signs recorded were blood pressure 115/70 mm Hg, heart rate 80 beats/min, respiratory rate 24 breaths/min, and body temperature 36.5°C. The cardiovascular examination disclosed a regular. During the treatment with duloxetine, the patient did experience significant blood pressure 150/90 mmHg. The patient was followed by a period and duloxetine treatment was stopped.

Duloxetine, not as much as venlafaxine, increases blood pressure and heart rate a bit but significantly (1-2 mm Hg). However, clinicians need to be aware of duloxetine-induced hypertension.
Keywords: SNRIs, duloxetine, hypertension

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[Abstract:0179][Psychopharmacology]

Skin picking disorder with DSM-5 criteria: a case report

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Skin picking disorder (SPD) is known by different names such as pathological skin picking, neurotic excoriation, dermatillomania, or psychogenic excoriation. SPD is characterized by the repetitive and compulsive picking of skin, leading to tissue damage. Compulsive picking makes SPD a public health problem. DSM-5 Skin Picking Disorder's diagnostic criteria are that recurrent skin picking resulting in skin lesions, repeated attempts to decrease or stop skin picking, the skin picking causes clinically significant distress or impairment in social, occupational, or other important areas of functioning, the skin picking is not attributable to the direct physiological effects of a substance (e.g., cocaine) or another medical condition (e.g., scabies); and the skin picking is not better accounted for by symptoms of another DSM-5 disorder (e.g., skin picking due to delusions or tactile hallucinations in a psychotic disorder, preoccupation with appearance in body dysmorphic disorder). Since DSM-5, skin picking has not diagnostic criteria. Selective serotonin reuptake inhibitors (SSRIs) are one of the most frequently prescribed drugs for the psychiatric disorder's treatment in children and adolescents. Most studies suggest that selective serotonin reuptake inhibitors (SSRIs) are useful in treating SPD. The long-term follow-up data also suggest SSRIs effectiveness. Responsiveness to the SSRIs was over 60% reduction as measured by the observer and self-rated scales was 3 points reduction by 6 weeks.
Case: A 16 year-old otherwise healthy boy presented to our clinic with entirely wound lips because of peeling lips' skin. He reported that the peeling would occur in lips after the spontaneous picking desire. He did not have the desire of peeling elsewhere on the lips. He had no other physical and psychiatric complaints. His complaints of lips' skin peeling started when he was 5 years old. He has never presented to the doctor before he came to us. He had gone psychologist before presenting us, but he had no benefits. He took drug for treatment first time, 50 mg/day of sertraline treatment was started. Since the one month sertraline treatment, there were no complaints about lips peeling and wound. In the past, he used 50 mg/day for 4 months and there has been no lips' peeling. To evaluate patient's complaints we use clinic global impression scales (CGI-S and CGI-I) for clinic evaluation. When he came to the clinic, CGI-S score was 6. After one month sertraline treatment, CGI-I score was 1.
Here, we present a case of a patient with lifelong skin picking behavior, which started when the patient was 5 years old and continued to the present day and at the end of one month sertraline treatment, there is no symptoms about SPD.
Keywords: skin picking disorder, DSM-5, sertraline

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[Abstract:0181][Mood disorders]

Late onset single manic episode following a stroke related essential thrombocytosis

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Essential thrombocytosis (ET) is clinically rare pluripotent stem cell disorder. It often seen at the age of between 50-60's. ET is generally asymptomatic and diagnosed during routine hemogram workup but it can cause ischemic and hemorrhagic complications. The clinical spectrum of essential thrombocytosis is quite broad. Vasomotor symptoms such as erythromelalgia, headache, confusion, visual disturbances are commonly seen in ET. Transient ischemic attacks, stroke, seizures, severe arterial thrombotic complications such as angina and myocardial infarction may also occur. Infarctions based on thrombotic occlusions in central nervous system, cause sensorimotor deficits and also personality and behavioral changes with cognitive dysfunction related to affected area. On the other hand, late-onset mania following cerebral infarctions is reported several times in literature. Among reported cases, mania may occur concomitantly with

stroke or even up to 3-5 years or more.
Case: A 46 year-old male patient who had an ischemic stroke 6 years ago due to essential thrombocytosis, presented with first episode manic excitation. He had no past history of psychiatric disorder. In his physical examination, any focal neurological symptom or finding detected. His current cranial diffusion MRI scan showed left lateral ventricle dilatation and chronic infarct zone in the left parietal lobe. With 15 mg/day haloperidol injection for ten days, his manic symptoms had regressed. Close follow ups were suggested after discharge in order to avoid to overlook clinical progress.
In this case report we aimed to draw attention association between late-onset mania and thrombotic ischemic cerebrovascular event that is related with an uncommon hematologic disease, i.e., essential thrombocytosis.
Keywords: bipolar disorder, stroke, thrombocytosis

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[Abstract:0186][Autism]

Autism spectrum disorder in a child with oculocutaneous albinism

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Oculocutaneous albinism (OCA) is an autosomal recessive disorder resulting in deterioration in melanin biosynthesis and characterized by hipopigmentations located in eyes, skin and hear. Its prevalence is 1/17000. In the literature there are fewer case related with autism spectrum disorder (ASD) and OCA seen together. In this case, a boy with OCA and diagnosed later with ASD was reported and discussed in terms of possible common points playing role in their etiologies.
Case: A 6 year-old boy presented to our clinic because of not to be able to talk, bizarre and obsessive behaviors, temper tantrums, stubbornness, self-harming behavior to himself and to the others. His phenotypic features was very distinctive displaying with white hair and eyelashes, very pale skin with light blue eyes. His neurological examination revealed normal findings except bilaterally horizontal nystagmus. He had earlier OCA diagnosis. His ocular examination also revealed although his weak cooperation of the vision with alternating esotropia and bilateral nystagmus, hypopigmentated fundus and hypoplasic optic disc, he could be able to count fingers from at least 2-3 meters.
Psychiatric examination of the patient revealed his incapability to be built social relation and interaction with his peer, delay in speech, restricted eye contact, stereotypic movements like hand clapping. He could not be able to look his name when calling to him consistently, but he could understand simple orders and could be done it, and he could not be able to start talking deliberately. He was also displaying symptoms of restlessness, short attention span, not to be able to sustain the structured games to play, intense interest to key and lock devises. He simply had limited vocabulary with not be able to build a complete sentence. Ankara Developmental Screen Inventory (ADSI) was administered to him and "moderate level of developmental retardation" was detected. Autism interview form based on DSM-5 criteria was carried out. Autism Behavior Control Checklist (ABC) and Child Autism Rating Scale (CARS) were administered to his parents. All mentioned scales revealed the higher scores. Based of all together with clinical evaluation, the patient was diagnosed by "ASD and Moderate Mental Retardation". An individual education programme was reported and a preschool education was recommended. He was followed up by our clinic over a year. He could become to be capable of doing greeting and farewell, repeating spoken words, learning to perform his self-care with help.
Autistic children would have more hypomelanotic skin lesions and abnormal dermatoglyphics than that of not having ASD. Mismaturation and misdifferentiation of ectodermal precursory cells during embryogenesis might play role in etiologies of ASD and OCD and this might stem from a neuroectodermal developmental anomaly. It also could be possible that this two disorder might be seen together because of their close chromosomal locations, which might play a role in both hypomelanosis and autism. This speculations need to be evaluated with further genetic and clinical studies and it would provide new insights in terms of understanding their etiologies.
Keywords: autism spectrum disorders, oculocutaneous albinism, genetic etiology

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[Abstract:0209][Dependencies]

Buprenorphine maintenance treatment on a patient with meperidine use disorder: case report

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Used to palliate pain before and after medical operations, Meperidine hydrochloride (MH) is a short-term and relatively weak agonist of the μ receptor. Opiate use disorder is a clinically determined psychological disorder that causes an apparent distress or functionality fall over 12 months or more, along with the manifestation of a problematic opiate use pattern. MH use disorder (MHUD) is frequently seen among health officers; it is the second most widely abused material by medical practitioners after alcohol. Buprenorphine (BN) is a partial agonist of the μ receptor used on maintenance treatments for opiate use disorders. In this study, a case with an opiate (MH) use disorder and its maintenance treatment with BN are discussed.

Case: N.C. was a 50 year-old, married father of two, a college-graduated male living in a city center and employed as a religious official. MH was first prescribed to the patient 15 years ago after an acute pancreatic attack, since when use has continued due to patient reports of ongoing intermittent pain. Ten years ago, the dosage was increased after the patient suffered trigeminal neuralgia, and for the past year it has been 500-900 mg/day. During the examination, the mood of the patient was depressively anxious. Opiate and benzodiazepine were established positive on the toxicological analysis of the urine, while, apart from iron and folic acid deficiency, nothing suspicious has been found pathologically in the urine and blood parameters. After the patient had been hospitalized, buprenorphine induction was administered and pregabalin 900 mg/day with carbamazepine 800 mg/day were given for trigeminal neuralgia treatment. Because of the patient's depression, anxiety and somatic grievances, amisulpride, duloxetine, mirtazapine and quetiapine were started. After discharge of the patient, during his one-and-half-month follow-up, except for BN, no other material was found on the urine toxicological analysis. The patient was in an early remission stage. In view of his history, clinical records and toxicological analysis, the patient had a severe opiate use disorder as diagnosed according to the DSM-5 criteria. In the literature, a case of MHUD and chronic pains was cured with venlafaxine treatment. In another case of MHUD and chronic pains, treated with benzodiazepine and mirtazapine, the patient died after an overdose of MH. In another case, the patient was discharged from hospital with continuous BN treatment at a five-month early remission stage.

On this case, in view of the severe MHUD diagnosis, duration of disease, unsuccessful treat attempts and issues arising from trigeminal neuralgia, BN was the favored treatment.

This case presents a successful example for the literature of BN maintenance treatment for chronic pain and MH use disorder. BN maintenance treatment is a good and beneficial alternative for patients suffering from opiate analgesic use disorder. In order to achieve a better treatment algorithm, much more research is needed.

Keywords: buprenorphine maintenance treatment, meperidine use disorder, opiate

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[Abstract:0211][Sleep disorders]

The effect of clonazepam on sleep terrors and co-occurring nocturnal enuresis: a case report

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Sleep terrors are parasomnias defined as a sudden arousal accompanying with a scream or cry which arising out of slow wave sleep. The person returns to sleep and cannot remember what they experienced during the night. The cause of the disorder is not clear, and behavioral treatments are suggested first line treatment. Also there are several case reports about the efficacy of imipramine and clonazepam on sleep terrors. Enuresis nocturna is classified as a parasomnia according to the International Classification of Sleep Disorders. Children with enuresis have longer time in bad and increased number of sleep cycles, are heavy sleepers and enuresis occurs in NREM stage of sleep and some reports are available about the possible links among enuresis nocturna to other parasomnias. Here in, we present an adolescent with sleep terror and co-occurring nocturnal enuresis who displayed complete remission after clonazepam therapy.

Case: A 15 year-old boy presented to the outpatient clinic with complaints of awakening in middle of the night, screaming, and having difficulty about calming down. Patient also had nocturnal enuretic events from his childhood three to four nights per week. The patient was diagnosed as Sleep Terrors and Primary Enuresis Nocturna according to DSM-5 diagnostic criteria. Because of the symptom severity and chronicity, clonazepam treatment was started at 1 mg/day. These treatment resulted in complete resolution of both sleep terrors and enuresis after the first day of the treatment. This patient has no signs of sleep terror or enuresis over the follow-up period of five weeks. Enuresis and sleep terrors occurs in the NREM phase of sleep and it is known that benzodiazepines like clonazepam inhibit stages N3 and N4, which are known as slow-wave sleep of the NREM sleep. Benzodiazepines also stabilize sleep by decreasing transitions between sleep stages, decrease stage shifts. Furthermore, imipramine, that has an inhibiting effect on NREM sleep phase, is also used to treat both disorders. Therefore, clonazepam might treat these disorders with affecting NREM phase of sleep in our patient.
This is the first report suggesting clonazepam may be effective for both sleep terrors and nocturnal enuresis. Further research needed for explain these effects of clonazepam.
Keywords: clonazepam, enuresis, nocturna, sleep, terror

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[Abstract:0212][Mood disorders]

Cerebral palsy and bipolar disorder: a case report

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Cerebral palsy (CP) is a disorder of motor impairment resulting from brain injury prior to the completion of cerebral development. In a study about preschool children with CP, a three to four fold increased risk found for emotional and behavioral problems compared to controls. Another study indicate that a significant proportion of children with cerebral palsy are at high risk of poor mental health. However, limited data is available about the relationship between bipolar disorder and CP in youths and only two CP patients with juvenile-onset bipolar disorder were reported in existing literature. In this report, we want to represent a BAD case in an adolescent who has cerebral palsy.
Case: A 16 year-old male adolescent presented to the child and adolescent psychiatry outpatient clinic. Patient's symptoms were insomnia, loss of appetite, decreased speech, anhedonia, tremor, increased spasticity, decreased social activity. His symptoms started 2 weeks ago and increased by the time. The patient was diagnosed as major depressive disorder according to DSM-5 diagnostic criteria. During this follow-up period his symptoms decreased spontaneously, we did not give any medical treatment and follow patient with as a diagnosis remitted major depression. After 3 months from his first admission, patient presented our clinic second time. At this time his symptoms were decreased duration of sleep, increased speech speed, extreme cheerfulness, and strange talkings. After mental status examination patient diagnosed as bipolar disorder manic episode. After evaluation, valproate 1000 mg/day and risperidone 1 mg/day were started. At next follow-up examination two weeks later, symptoms was decreased dramatically. Several literatures suggest that depressive disorders are more prevalent in children with CP. However, few literatures emphasize bipolar disorder and CP relationship. A recent study indicates a high proportion of psychiatric disorders and behavioral problems in children with CP. Brain damage in CP can be a vulnerability factor in the occurrence of psychiatric disorders. Because of both brain damage and psychical disabilities, symptom of psychiatric disorders, especially bipolar disorder, in CP may be varied and masked comparatively people with do not have CP or other disorder which damage brain morphology and function.
The relationship between CP and BAD is unclear. It is known that premature birth is a risk factor for psychiatric disorder including bipolar affective disorder. Similar genetic properties, shared similar obstetric environment and developmental trajectories may cause this relationship. In addition to all these, during daily outpatient and consultation practice, we come across patient with CP. We know that this patient has higher rates psychopathology including internalizing problems, such as depression. We think, it is important not to forget that this depression might has a bipolar depression, and any manic attacks or symptoms must have investigated. Detailed examination should be made for bipolar disorder in CP patients.
Keywords: bipolar disorder, cerebral palsy, valproate

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[Abstract:0215][Psychopharmacology]

Symptomatic hyperammoniemia associated with valproic acid use in bipolar disorder: a case report

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Valproic acid (VPA), is commonly used drug in treatment of several psychiatric and neurological disorders. Hyperammoniemia is a well-known adverse effect of treatment with valproic acid, with an incidence of 16.7 – 100 %. Ammonia levels greater than 60 mmol/L are commonly associated with clinical symptoms including anorexia, irritability, lethargy, vomiting, somnolence, asterixis, coma and death. Elevation of ammonia is usually less in patients with VPA-induced hyperammoniemia and asymptomatic hyperammoniemia usually goes unnoticed in clinical practice. The mechanism remains incompletely understood, but is likely to relate to a defect in the urea cycle. Combination treatment with liver enzyme-inducing antiepileptic drugs and antipsychotic drugs increases the risk of VPA-induced hyperammonemia.
Case: A 50 year-old female patient with bipolar disorder for fifteen years had a history of hospitalization in psychiatry clinics because of manic episodes for three times. She has been in remission for the last 8 years with lithium treatment, but she was consulted owing to impairment in kidney function tests (GFR:39.37 and serum creatinine:1.49). She did not have any psychiatric complaint and her psychiatric examination was normal in our consultation liaison psychiatry outpatient clinic. We planned a gradual change her lithium treatment to valproic acid treatment. After cessation of lithium, VPA 500 mg/day treatment was started and its dosage was increased to 1 g/day on the third day. She presented to the medical assessment unit with acute symptoms such as nausea, vomiting and fatigue on the first day of the VPA 1 g/day treatment. Blood tests and electroencephalography (EEG) were done immediately. Routine blood tests, liver function tests, serum sodium valproate level (70.9 µg/mL) were all normal. Only serum ammonia was elevated (203 µg/dL), consistent with a diagnosis of VPA-induced hyperammonemia. EEG resulted normal. Withdrawal of VPA led to clinical recovery in 24 hours. Serum ammonia was 43 µg/dL on the second day of VPA cessation. Immediate recovery after cessation of VPA and absence of clinical symptoms such as fever, neck pain/stiffness, seizures, confusion and leukocytosis, excluded encephalitis diagnosis. Within therapeutic dose and serum levels, VPA can lead to symptomatic hyperammonemia. Most of the patients with VPA-induced hyperammonemia are asymptomatic and liver function tests and EEG are normal. However, if patients taking VPA present with symptoms such as nausea, fatigue, somnolence, ataxia, or behavioral changes with decreased level of consciousness; serum ammonia level should be measured besides other routine blood tests. VPA-associated hyperammonemic encephalopathy is a serious complication of VPA, rarely observed and can result in death unless diagnosed early. Although encephalopathy could appear even with normal serum ammonia levels, serum ammonia level is an important parameter in the early diagnosis of this rare adverse event. As a result, nausea and vomiting in a patient with VPA treatment could be a warning sign before appearance of neurological symptoms of hyperammoniemia.
In conclusion, any clinical sign and symptom in patients under VPA treatment should be carefully evaluated with blood tests including serum ammonia and EEG, in order to prevent serious results such as VPA-associated hyperammonemic encephalopathy.
Keywords: valproic acid, adverse effects, hiperammonemia

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[Abstract:0216][Psychopharmacology]

Rheumatologic adverse effects associated with psychotropic drugs

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There were several case reports about rheumatologic adverse effects of drugs in literature. Rheumatologic adverse effects have been reported to be induced by multiple classes of drugs, but there are only a few case report about psychotrop drugs induced rheumatologic adverse effects in literature. Here; we report a case who presented with rheumatologic adverse effects due to psychotrop drugs.
Case: A 34 year-old female patient. Her main symptoms included depressive mood, irritability, insomnia, loss of interest and anxiety which these symptoms have begun about 9 months ago. She had not any diagnosed major systemic diseases. She was diagnosed major depression and generalized anxiety disorder according to DSM-IV, and the treatment has arranged as sertaline 100 mg/day, hiyosin-N-butilbromid and medazepam. After a while rheumatologic symptoms appeared as pain, erythema, limitations on the movement of the

lower extremity joints. Then she stopped taking drugs by herself after using for 2 months. Also her depressive and anxious symptoms were regressed. After a while, anxiety and depressive symptoms occurred again, the treatment arranged as sertraline, alprazolam, agomelatine, and then rheumatologic symptoms appeared again. She did not use her drugs regularly and then stopped taking sertraline and agomelatine. We arranged the treatment as escitalopram 10 mg/day, mirtazapine 15 mg/day. Her depressive symptoms regressed, anxiety symptoms partially persisted. She hospitalized in rheumatology service due to the continuation of rheumatologic symptoms. She had also fever, erythema especially in the evenings, spasms of the lower extremities and swollen left cervical lymph node. Crp and sedimentation also increased. Psychotropic drugs were gradually stopped. Furthermore; she had treated with hidroxicloroquina 200 mg/day, prednisolone 60 mg/day, pregabalin 150 mg/day, colchicine 1 mg/day and vitamin d. Finally, her rheumatologic and anxiety symptoms also regressed.
Hence, the temporal relation between the appearance of the rheumatologic symptoms and psychotropic drugs use supported the diagnosis of psychotropic drugs - induced rheumatologic adverse effects. Developing rheumatologic symptoms while using psychotrop drugs and regression of symptoms after stop the psychotrop drugs have found remarkable. The mechanism of psychotrop drugs action is not known exactly. It has been thought that increased serotonin transmission can play an important role in the pathophysiology of rheumatologic symptoms by consisting inflammatory effects of increased serotonin level in synovial fluid. Otherwise, hypersensitivity reaction is suggested to be a possible mechanism of development of arthritis. An association between serotonergic antidepressants and worsening of arthritic complaints has been reported. After using of antidepressants, it has appeared arthritis/ arthralgia in patients, especially serum sickness-like reactions have been reported. Increased serotonin transmission caused by antidepressants may particularly explain the rheumatologic symptoms in this patient. In conclusion, physicians should be attentive about the possible appearance of rheumatologic symptoms when using psychotropic drugs, like the other drugs.
Keywords: rheumatologic reaction, psychotropic drugs, adverse effects

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[Abstract:0219][Psychopharmacology]

Nickname is 'Baby Ecstasy': a case with misusing of venlafaxine

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Venlafaxine is a serotonin norepinephrine reuptake inhibitor which is used in varies neuropsychiatric conditions from major depression and anxiety disorders to fibromyalgia and headache. Although maximum recommended dosage is 375 mg, here we described a case who had 'self-medication' with venlafaxine 900 mg/day for her depressive symptoms.
Case: A 41 year-old female patient presented to psychiatry outpatient clinics with complaining of depressed, anhedonia and anxiety. In mental examination: self-care is reduced; the speech rate and contents are decreased. Affect is sad and mood was depressive and anxious. She had no suicidal ideation.
In history, she had postpartum depression diagnosis 11 and 4 years ago. She was taking venlafaxine 900 mg/day for 4 months while she presented our clinic. Treatment was offered as venlafaxine 600 mg and lithium 300 mg/day. One month later her depressive complaints are increased, also she described withdrawal symptoms like nausea, dizziness, anxiety and restless. Fluoxetine 20 mg and alprazolam 1 mg/day was added. After one month her withdrawal symptoms were better however she had depressive complaints again, so treatment was ordered as lithium 600 mg/day, venlafaxine 450 mg/day and also bupropion was added 150 mg and up to 300 mg/day. She did not persuade to get venlafaxine blood level although asked repeatedly. At the end of the one year follow up, she confessed that she used venlafaxine 600-900 per/day dosages whole the following period!
In this case, we have diagnosed 'venlafaxine misuse' according to DSM-5 having following these criteria: withdrawal, using larger amounts and repeated attempts to control using. Venlafaxine has also dopamine (DA) reuptake inhibitor act at high doses (>300 mg/day) with serotonin (SA) and noradrenalin (NA) reuptake inhibition. DA is the main neurotransmitter for rewarding system; having SA/DA booster effect at high dosage is the reason that venlafaxine could be associated with both addiction and amphetamine like effects. In a current review, there were some cases that used venlafaxine with dosage from 1500 mg to 4050 mg/day. A misuse called venlafaxine as 'Baby Ecstasy' comparing with the ecstasy effects. So we must be aware of venlafaxine misuse when we see withdrawal, tolerance, and some physical/ psychological problems related to use.
Keywords: venlafaxine, misuse, withdrawal

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[Abstract:0223][ADHD]

Methylphenidate-related severe and diffuse hair loss: a case report

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Drug induced alopecia is a reversible side effect that commonly occurs as general alopecia, usually developing in the first three months of treatment and resolving after the cessation of the responsible drug. Drugs that induce hair loss may be classified as agents that affect either phase of the hair follicles' cycle. Attention deficit and hyperactivity disorder (ADHD) is one of the most commonly diagnosed and treated childhood psychiatric disorders. Methylphenidate is the most commonly used ADHD medication. In this report, we present a severe and diffuse alopecia case, which developed after the onset of methylphenidate treatment and discontinued after stopped the drug.

Case: A 7 year-old girl was referred to our outpatient clinic with complains of hyperactivity, irritability, distractibility attention, often lose her belongings, forced to make her homework for about two years. She was assessed by clinical interview and structured teacher and family scale. Attention deficit hyperactivity disorder (ADHD) was diagnosed to DSM-5 criteria. She did not have any other known medical illness. Methylphenidate 20 mg /day controlled delivery form was started. About a week after the start of methylphenidate, she and her mother noticed that a mild degree hair loss. Symptoms of attention deficit and hyperactivity were dramatically reduced. However, while continuing treatment they noticed increased hair loss and appeared more hair on her comb and bed daily. Because hair loss is too much, her mother said that she was afraid to comb her hair. While having previously very bushy hair, her hair was diminished and her scalp was visible. There were no losses in the eyebrows, eyelashes or elsewhere. She was not taking any other medications. There was no personal or family history of hair loss. To investigate the causes of hair loss, she was referred to the Dermatology Clinic. Complete blood counts, biochemical analysis, folic acid, vitamin B12, thyroid function tests, serum iron and total iron-binding capacity, ferritin values were normal. There were no any infectious or other reasons that cause hair loss. In this case, methylphenidate-related hair loss was a clear association with patient's presentation and temporal correlation with pharmacotherapy initiation. Subsequently, methylphenidate treatment was stopped after discontinuation of the methylphenidate; she and her mother noticed that hair loss completely stopped.

Hair loss is an undesired side effect, usually resulting treatment non-adherence. Drug-induced hair loss is difficult to diagnose because there is no special measurement method. Other conditions that can cause should be excluded. In our case, no dermatological disease or other medical conditions that may cause hair loss has been detected. Case reports and studies of methylphenidate relationship alopecia is limited. Our case report aimed to contribute to the literature and to the physicians in clinical practice. Large-scale studies are to explain the mechanisms of hair loss required.

Keywords: hair loss, methylphenidate, side effect

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[Abstract:0224][Impulse control disorders]

A patient with restless leg syndrome presenting with pramipexole -induced pyromania

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Restless Legs Syndrome (RLS) is a common sensory-motor disturbance, characterized by distressing deep sensations in limbs, in particular legs associated with an urge to move often at rest time. Iron deficiency, disturbances in the central dopaminergic system, and genetic factors are thought to be the main etiological factors. Pramipexole, a non-ergot D3 dopamine agonist, could be used as the first step of RLS treatment. Impulse control disorder due to dopaminergic drug usage in Parkinson patients was demonstrated. Pathological gambling, hypersexuality, compulsive shopping, compulsive pounding, and compulsive medication use were some of the entities as impulse control behaviors. Recently impulse control disorder was demonstrated in patients with RLS using dopamine agonists. Pyromania

was not observed in patients on Dopamine agonist treatment. Here we report a case report of RLS patient on pramipexole therapy demonstrated pyromania.

Case: A 54 year-old female patient presented with distressing deep sensations legs, associated with an urge to move often at rest time. She was diagnosed with RLS. Pramipexole treatment at 0.25 mg/day dose was started four years before. Her symptoms were released. One year before her admission she experienced argument with her husband. Her RLS symptoms were exacerbated and dose of pramipexole was elevated 1.0 mg/day, by her physician's advice. She has increased Pramipexole dose over 3.0 mg/day, without her physician's advice. After a while she experienced amnesia, irritability against her husband, hearing buzzing noise, such as sound of a door closing. Within the same time in a house opposite of her home a fire setting incident was occurred. It was confirmed at the fourth time that she was the person who set fire, by video recording of the owner of the house. In the expression at the court she insisted on setting fire in that house again. After these repeated fire settings family members advise her to hospitalize. At her admission her psychiatric examination her cognition, and orientation was completely normal. She demonstrated anxiety. Her content of thought was composed of elevated thoughts about fire setting. There was no sign of hallucination and delusion. Her impulse control was decreased and intelligence level was normal. On her follow up, she said that she had a tendency to set fire. She declared that she enjoyed watching the fire, and the firefighting. She also told that she suffered anxiety and dysphoria before setting fire, and relaxation after setting fire. Organic disorders were excluded by routine biochemistry tests, cranial MRI, and EEG. She diagnosed with pramipexole -induced pyromania. She was treated with gabapentin 600 mg/day which made her RLS symptoms released.

The powerful D3 dopaminergic affinity on limbic system of pramipexole is probably the rationale under pramipexole associated impulse control disorder. In patients under the treatment of dopamine agonists with RLS demonstrated higher impulse control disorder than patients without using dopamine agonists. Recently pathological gambling plus hypersexuality, compulsive habits, and psychosis was reported on patients under dopaminergic agent treatment. This is the first case report of a RLS patient demonstrated pyromania under the treatment of pramipexole.

Keywords: restless legs syndrome, pramipexole, pyromania

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[Abstract:0231][Psychopharmacology]

Methylphenidate -induced acute dystonia in a child: a case report

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ADHD is one of the most prevalent childhood psychiatric disorders. Central nervous system stimulant medications are the most commonly used psychotropic drugs to treat the symptoms of individuals with ADHD. Stimulant medications are known to alter the transmission of dopaminergic neurons in the prefrontal cortex, striatum, basal ganglia, and cerebellum. Stimulant medications are generally well-tolerated. However case presentations indicated that movement disorders such as dyskinesia and dystonia may rarely occur. In most case reports antipsychotic-induced dystonia started after stimulant discontinuation in patients receiving concomitant stimulant and antipsychotic therapy.

In this case, the treatment of dystonia with biperiden after administration of immediate release methylphenidate (IR-MPH) will be discussed.

Case: A 6 year-old male patient presented to our clinic with complaints of hyperactivity, lack of attention, and learning difficulties. No problems were described in the perinatal period and in the stages of development. Conners parent and teacher rating scale and WISC-R were administered. According to information from parents and teachers and psychometric tests (WISC-R verbal IQ: 85, performance IQ: 68, total IQ: 74); ADHD and specific learning disorder were diagnosed. IR-MPH 3x5 mg treatment was started. The ADHD improved significantly and the treatment was arranged as 18 mg OROS-MPH. This treatment was performed for approximately 1.5 years. The OROS-MPH dose was increased to 27 mg at the age of 7.5 but motor tics appeared after dose increased. Haloperidol 2x0.5 mg was added to the treatment. The tics disappeared with haloperidol. Haloperidol and OROS-MPH was used for 4 months and drug holiday were made in the summer. After summer holiday IR-MPH 3x5 mg was started again. On the 10th day after the IR-MPH treatment, the patient visited the emergency service two hours after the morning dose with complaints of lack facial expression and inability to swallow salivary. No other medications were used besides IR-MPH. Except for stiffness in the elbow joint and inability to swallow, neurological assessment and blood tests was normal. The present symptoms of the patient were evaluated as acute dystonia. Intramuscular 2.5 mg biperiden was

administered. The patient's symptoms completely disappeared 30 minutes after administration of biperiden.
Acute dystonic reactions usually occur after the use of dopamine receptor blocking agents. The mechanism underlying acute dystonia is unknown: both increase and decrease of the striatal dopamine transmission have been put forward as possible causes. Although psychostimulants may initially enhance release of dopamine in the striatum, their long-term use is associated with striatal dopaminergic down-regulation. Also methylphenidate increases the extracellular dopamine level in the striatum that has been proposed to induce movement disorders. In addition children with preexisting basal ganglia pathology may be vulnerable to methylphenidate. The prior occurrence of tics in our patient after methylphenidate treatment might suggest the presence of a basal ganglion sensitivity. It can be considered treatment with methylphenidate has caused an acute dystonic reaction due to striatal down-regulation and basal ganglion sensitivity. It has to be taken into consideration that the solitary use of methylphenidate might cause acute dystonic reactions especially in individuals with striatum sensitivity.
Keywords: attention deficit hyperactivity disorder, dystonia, methylphenidate

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[Abstract:0233][Psychosomatic medicine-Liaison psychiatry]

Conversion disorder with persistent movement disorder and ataxia: a case report

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Psychogenic speech and voice disorders (PSVDs) are characterized by a variety of abnormal speech and voice patterns that are incongruous with organic neurologic disorders. Although PSVDs can occur alone, they usually coexist with other psychogenic disorders, including psychogenic movement disorders. Most patients, by DSM-5 terminology, are mainly categorized as Conversion Disorder, or Functional Neurological Symptom Disorder under the general category of Somatic Symptom and Related Disorders.
Here we present a case diagnosed with conversion disorder with persistent movement disorder and ataxia.
Case: A 36 year-old man with a high school education was referred to psychiatry clinic for evaluation of speech and gait disorder. The manifestations started after a trigger stressor, marital conflict. Although during the first month symptoms were aggravated by stressor factors, within next two years no remission had occurred. There was paraesthesia and tingling at the left upper extremity for last two months. He had been struggling to work, and had been socially isolated. After several neurological examinations, no neurological disorder had been diagnosed. Cell blood counting (CBC), serum electrolyte, thyroid function tests, tumor markers were all negative. MRI of the brain and lumbar punction evaluation were normal. VDRL test was positive and confirmed with FTA-ABS Ig G; patient was treated with penicillin for 6 days. Although treatment was completed, patient did not have any significant improvement. Psychiatric medication history included escitalopram (up to 20 mg per day), bupropion (150 mg per day) and duloxetine (30 mg per day). Patient was alexithymic, the finding of "la belle indifference" was present. Speech disorder had a complex presentation with combination of stuttering and speech arrests. Patient was wobbling around, which was incongruent with known gait disorders. His balance was much better than the claim. These phenomena were easily distractible during examination. Patient started to take 60 mg/day duloxetine, 10 mg/day escitalopram treatment at inpatient clinic. Supportive psychotherapy and insight oriented therapy was added to pharmacotherapy. Recognition of emotions was the central element of therapy techniques; behavioral suggestions were combined with cognitive formulations to motivate patient work through balance and gait exercises. After two months his symptoms remitted, patient could easily express himself and gait disability disappeared.
Psychogenic movement disorders are those thought to be due to a psychological cause, although in fact full pathophysiology is not really known. In our case, a life stressor was the main trigger of symptoms. Having an exact moment when the disorder began, incongruent course with neurological disorders, change in the nature of the movement over time and history of stress were consequent. Early life trauma and stressors are shown to be related with psychogenic movement disorders, although our patient did not have. Data indicate that conversion disorder is most common among persons with little education and the ratio of women to men among adult patients is at least 2 to 1 and as much as 10 to 1. In our case, patient was male and high-educated. Despite having no remission within two years, our patient's symptoms remitted after two months of treatment.
Keywords: ataxia, conversion disorder, movement

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[Abstract:0237][OCD]

Hoarding disorder in men: a case report

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Hoarding disorder (HD) is a persistent difficulty discarding or parting with possessions due to a perceived need to save them. The patient experiences distress at the thoughts of getting rid of these items. The fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) included a new chapter on obsessive-compulsive and related disorders to reflect the increasing evidence of these disorders' relatedness to one another and distinction from other anxiety disorders. HD has an early age onset, chronic course and significant levels of comorbidity and morbidity. Information about gender differences in hoarding has not been consistent. Some studies have found hoarding to be more common in men than in women while some studies have found the opposite. At this time there appear to be no differences between the genders on measures of clutter or difficulty with getting rid of clutter, but some research has shown that women are more likely to go out and acquire more possessions more often than men.

Case: A21 year-old male patient, single and a student, lives with his family. He came to Psychiatry Outpatient clinic with his mother. His mother said that he hoarded balloons, gums, balls, books, money and ropes, and he added he did not want to throw them away. He collected balls at the streets and brought them home when he was 12-13 years old. Then, he began to hoard gums, money, balloons, chips, pencils, erasers and ropes. He blew the balloons and left them in the house but did not play with them. It was reported that there were nearly 300 balls in the house. He hoarded the balls and the balloons behind the night stand, under his bed and on the boards in the bathroom. He hoarded gums on the same plate in the kitchen. His mother found pencils, erasers and ropes in his schoolbag. It was also reported if he did not take a shower for a long time every day, he could not sleep. Also the next day he when he woke up, he took a shower. When his father asked him to wash his shop, he took it as his duty and washed the shop every day. Also, he got angry with his mother when his mother imposed him another duty. He hanged 4 towels on the hanger behind the bathroom door whenever he came out of the bathroom. While he was painting when the paint went out of the figure, he immediately cleaned the paint and repainted the picture. He did not like going out of lines. He has had a sleep problem recently. He started to get up at 3 or 4 a.m. and make his mother wake up too. He made his mother wait until the morning and began to sleep in the morning again. Also he had money on him every time even though his family did not give him any money. Therefore, they began to suspect him, but they could not find the source of the money. They presented to Psychiatry Outpatient clinic a year ago, but the patient did not have a regular treatment. They came to our department this year again. Mr. R.E was born by spontaneous vaginal delivery. He is the second child of a four-children family. He said only one word when he was 2 years old. Later, he began to speak. He was sounding meaninglessly and trying to make himself understood with hand gestures. He was taken to a hospital in Istanbul when he was 5 because he could not speak. He had some tests there and he was diagnosed with mental retardation. Then, he attended a state school for 4 years in Rize because there was not an institute giving a special education there. However, he studies at a high school giving a special education. He has 4 siblings. The first child in the family is 22, female and studies at Anadolu University Distance Education Program. The second child is our patient. The third one is 17, male and he dropped out after elementary school. Now he works with his father. He was also diagnosed with attention deficit and hyperactivity and began to use medicine. His pursuit was done in Trabzon and he discontinued medication when he was 12 under the control of a doctor. He does not have a current health problem. The fourth child is 15, male and has mental deficiency. He attends a special training program. The parents are not consanguineously married. The mother is 48 years old, alive- healthy and a housewife. She studied until the third grade at elementary school. The father is 50 years old, alive-healthy, elementary school graduate and a butcher. The patient lives with his family. His grandmother also lives with them. He shares the same room with his 3 brothers. He does not smoke and have alcohol. Physical examination and Laboratory results: normal, EEg: normal, and the Brain MRI: normal. In the first psychiatric review of the case; the male patient who looked well cared, respectful and older than his age was looking around, was not speaking spontaneously and was replying hesitant only when asked questions. He was agitated during the interview, her attention and concentration was poor, and he lacked insight. Intelligence quotient (50-60) is considered as a mild mental retardation. The Yale–Brown Obsessive Compulsive Scale (Y-BOCS) point was determined as 30. The patient was diagnosed with HD. Having increases in psychiatric symptoms, the patient's treatment was started to treated with fluoxetine 20 mg/day and risperidone 2 mg/day; and doses of drugs gradually increased (fluoxetine 40 mg/day, risperidone 4 mg/day). Currently, the patient's psychiatric treatment and controls are ongoing and her complaints partially decreased. mild mental retardation.

HD, a severe form of collecting or hoarding, is currently described in various psychiatric disorders in men. This case report describes an excessive HD confirming the complexity of this trouble and the possibility to consider it as an independent diagnosis.

Keywords: Hoarding disorder, gender, obsessive-compulsive disorder

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[Abstract:0240][ADHD]

A case of attention deficit hyperactivity disorder developed after being exposed to general anesthesia

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The findings of a great number of studies on whether pediatric anesthesia causes neural damage in humans are inconsistent. However some researchers have shown that being exposed to multiple anesthesia and long term behavioral disorders are associated. Genetic factors and environmental factors play a role in the etiology of ADHD. As well as anesthetic substances, being exposed to neurotoxic agents can be considered as one of the environmental risk factors. In this case report, developed attention deficit hyperactivity disorder symptoms after being exposed to general anesthesia will be discussed.
Case: An 8 year-old male patient studying in 3rd grade was brought to our polyclinics with the complaints of hyperactivity, talking too much, a decrease in achievement and short temper.
It was learned that the patient's complaints had started nine months ago after he had undergone tonsillectomy operation under general anesthesia and propofol and sevoflurane were used during the anesthesia. It was learned that the patient who had normal developmental stages did not have any psychological problems or complaints previously. After the underwent tonsillectomy operation, he did not listen to lessons, he talked during class, he was easily distracted, his writing became more inelaborate, he forgot his things at school, he started to fight with his friends, he had difficulty while waiting for his turn, he began interrupting people, he was quickly distracted while doing his homework, he got interested in other things or left his desk with various excuses and seemed not to be listening most of the time. During the interview, he had difficulty in sitting down, he was easily distracted and he talked a lot. With the existing findings, the patient was diagnosed with ADHD according to DSM-5 diagnosis criteria. The patient was started long acting methylphenidate treatment and he was called for outpatient clinic follow-up. In his follow-ups two weeks later, a decrease was found in the complaints.
In immature animals, being exposed to GABA receptor agonists or NDMA receptor antagonists cause apoptotic degeneration in neurons in various areas of the brain. Being exposed to anesthetic agents which influence through these receptors may affect normal brain development. Behavioral changes after surgery have been reported in children who have used sevoflurane as anesthetic agent. Although short term neurocognitive changes have been shown in case reports of children who were given propofol, there are no studies which assess long term impairment. In our patient, these two agents he was exposed to during general anesthesia may have caused attention and behavior problems. In conclusion, the clinical associations of these findings in children who receive anesthesia are not clear but being exposed to anesthetic agents can be an environmental risk factor in ADHD in the etiology of which environmental and genetic factors play a role.
Keywords: ADHD, general anesthesia, sevoflurane

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[Abstract:0241][Schizophrenia and other psychotic disorders]

A case of a blind epileptic patient experienced a psychotic episode and had visual hallucinations

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The comorbidity of psychotic disorders and epilepsy is well-known and the prevalence rate of psychosis in patients with epilepsy is estimated to be 6-12 times higher than the general population. In addition, visual hallucinations have been reported in blind psychotic patients. In this report, the association between psychotic disorders and epilepsy is discussed through a case who became blind due to bilateral uveitis and had visual hallucinations.
Case: 23 year-old male patient has been followed up with the diagnosis of epilepsy since he was two years old and was treated with antiepileptic medications. His first psychiatric admission was at the age of 17, because of his aggressive behaviors. He was treated with low doses of antipsychotic medications but because of extrapyramidal symptoms and frequent seizures these medications were discontinued.

He was diagnosed as uveitis when he was 18 and he lost his vision gradually within two years. He presented to our outpatient clinic with symptoms of irritability, sleeplessness, self-talk, suspiciousness and agitation. He had persecution and reference delusions and defined visual hallucinations. He refused to take his antiepileptic drugs because of the thoughts of being poisoned. He was diagnosed as "Psychotic Episode" and because of treatment non-adherence Zuclopenthixol acetate (50 mg/ml) and Biperiden HCL (5 mg/ml) was administered, family was informed about extrapyramidal symptoms and the patient was consulted to neurology. The routine blood tests were in normal range and epileptic activity was established in electroencephalogram. After the administration of Zuclopenthixol acetate the agitations of patient were decreased but there was no change in other symptoms. Also he developed extrapyramidal symptoms and because of the discontinuation of antiepileptic drugs he had seizures frequently. Olanzapine 10 mg/day and Diazepam 15 mg/day was started and the symptoms like agitation, irritability and sleeplessness were resolved. The delusions and hallucinations were decreased but continued in the sixth week of treatment. He started to take his antiepileptic drugs too and have no more seizures. Gradually discontinuation of diazepam was scheduled.

Severity of epilepsy, parental history of psychosis, history of mental retardation and status epilepticus, the occurrence of minor seizures with impairment of consciousness, antiepileptic medications may play a role in development of psychosis among epileptic patients. Antiepileptic drugs like vigabatrin and neurologic disorders may also cause psychosis in these patients and the treatment approach changes due to underlying reason and whether the patient diagnosed as interictal, postictal and ictal psychosis. All antipsychotic medications decrease the seizure threshold in varying degrees and therefore the choice of antipsychotic medication has a critical significance. As in Charles Bonnet Syndrome visually impaired patients may experience complex visual hallucinations.

An epileptic patient who develops psychosis should be investigated to determine the cause, as treatment approach may vary due to underlying pathology. If needed, the antipsychotic medication should be started carefully and monitored closely to prevent antipsychotic -induced epileptic seizures.

Keywords: epilepsy, psychosis, visual hallucinations

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[Abstract:0243][Psychosomatic medicine-Liaison psychiatry]

Severe major depression: a case of neurobrucellosis

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Brucellosis is a zoonotic disease that can affect various systems. Generally, the symptoms are anorexia, weight loss, nausea, vomiting, muscle and joint pain, fever, head, and abdominal pain. Neurobrucellosis is a rare complication that can be found 2-3% of brucellosis cases. Our case presented to the hospital with major depression and radiculopathy table and recovered only with antibiotherapy.

Case: A 59 year-old male, living in the village. The patient presented to the neurology clinic with problems of back pain, started 8 months ago, which cause restriction in mobility. In this period significant weight loss, urinary incontinence, severe depressive symptoms and speech disorder developed. During the neurologic evaluation, no significant findings were noticed except proximal right lower extremity was 4/5 muscle power. Examination results show that CRP 1.78 mg/dl, sedimentation 52 mm, LDH 539 U/l. His cranial MRI shows the presence of cystic area as a result of an old bleeding. Spinal MRI indicated the presence of a T1A hypo intense and T2A hyper intense at the level of L2-3, T9-10, T11-12. In blood, Brucella tube agglutination was 1/160. CSF analysis could not be performed because the patient did not give consent. The patient was diagnosed with neurobrucellosis due to brucella tube agglutination test results and the clinical findings, which cannot be explained otherwise. Doxycycline and rifampicin treatment was begun at the infectious diseases clinic. The patient was referred to the Psychiatric Department due to the depressive symptoms in the same day. Psychiatric examination of the patient revealed that he has depressive mood, decrease of self-care and reduction in speech tone. His thoughts were dominated by guilt, worthlessness and hopelessness. Decreased pleasure, sleeping disorder, forgetfulness, speech disorder, loss of appetite and weight loss were noticed. Hamilton Depression Rating Scale scored 45 and Beck Depression Scale scored 42. The patient was diagnosed with severe depression but these symptoms thought to be possibly associated with brucellosis therefore antidepressant treatment was not started. He received treatment at infection service and evaluated daily by the consultation liaison psychiatry unit. On the 7th day of the treatment it was observed that symptoms of depression began to decrease. On the 14th day, Hamilton depression scale score was 6, Beck depression score was 8. The assessment made in 6th month of the treatment, clinical, radiological and laboratory findings of the patient showed significant improvement.

Neurobrucellosis may appear in all stages of the disease with a wide variety of neuropsychiatric symptoms. Our case presents an

example of the both central and peripheral involvement with the findings of depression and radiculopathy symptoms. In literature, there is psychiatric cases reported depression, amnesia, psychosis, agitation, personality changes due to the brucellosis. The purpose of presenting this case is to show that the all the psychiatric and physical symptoms due to brucellosis can be rapidly reduced and disappeared with antibiotic therapy without additional psychiatric intervention. Neurobrucellosis should be considered for the patients with neuro-psychiatric symptoms as a differential diagnosis, especially endemic areas like our country.

Keywords: consultation liaison psychiatry, depression, neurobrucellosis

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[Abstract:0246][Psychopharmacology]

Terazosin in treatment of clozapine-induced sialorrhea: A case report

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Clozapine is an atypical antipsychotic which has a high efficacy in treatment resistant schizophrenia. However, use of clozapine is limited because of serious side effects and associated mortality. Sialorrhea is a common side-effect, observed in approximately one-third of patients under clozapine medication. Clozapine -induced sialorrhea may lead to drug discontinuation by influencing quality of life. The theory behind the pathophysiology of hypersalivation is blockade of adrenergic α1, α2, muscarinic M3 receptors and M4 agonism. Terazosin is an adrenoceptor antagonist used in the treatment of hypertension. Adrenergic α1 blockade effect of terazosin makes it a choice for treatment of clozapine -induced hypersalivation. Here we present a case of clozapine associated sialorrhea treated with terazosin.

Case: A 42 year-old female patient with 10 years history of schizophrenia presented to forensic psychiatry unit with persecutory delusions and auditory hallucinations. The patient was hospitalized as a judicial case, therefore her situation had been required a long-term internalization. Before hospitalization she used haloperidol, aripiprazole and risperidone irregularly. She was started on haloperidol 20 mg/day biperiden and zuclopenthixol decanoate depot i.m. 200 mg/two weeks. Her treatment was alternately changed to aripiprazole, risperidone and haloperidol decanoate in order to reduce her resistant psychotic symptoms along 13 months. She developed extrapyramidal side effects, hyperprolactinemia and her psychotic symptoms were poorly controlled. The patient's symptoms were intractable therefore clozapine was initiated. Along eight weeks the dose was gradually increased to 300 mg/day. Sialorrhea became distinguishable and obviously bothering. Amitriptyline was added to minimize sialorrhea. Her hypersalivation got worsened when the clozapine dose increased to 400 mg/day. The patient drooled abundantly all day. Her saliva was dripping all her clothes and seat. The front of her shirt was getting wet all the time. She was carrying a napkin to wipe drooling. The patient also complained about her pillow was getting wet when she woke up. Amitriptyline was discontinued and patient was started on terazosin-hydrochlorid 2.5 mg/day after a week. Her medication was continued with the clozapine dose of 400 mg/day. Within two weeks of increasing the terazosin-hydrochlorid dose to 5 mg/day the patient's hypersalivation related complaints decreased distinctively. She did not need to carry napkin and dripping was not noticeable objectively anymore.

In the treatment of clozapine -induced sialorrhea; lowering clozapine dose and activating swallowing reflex are non-pharmacologic strategies. The main targets for pharmacological prevention of hypersalivation are adrenergic and muscarinic receptors. Besides of pharmacologic drugs including adrenoceptor agonists and antimuscarinic agents, terazosin; an adrenoceptor antagonist is an efficient choice in treatment of clozapine-induced sialorrhea.

Keywords: terazosin, clozapine, sialorrhea

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[Abstract:0247][Psychopharmacology]

Hepatic enzyme elevation with quetiapine treatment: a case report

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Quetiapine is an atypical antipsychotic agent common used for the treatment of depression, bipolar disorders and schizophrenia. Pharmacologically, quetiapine has antagonistic effects on serotonin 5-HT1A, 5-HT2A, dopamine D1, D2, histamine H1, and adrenergic α1, α2 receptors and it is mostly metabolized in the liver via the cytochrome p450 isoenzyme CYP3A4. Primary effects associated with overdose are delirium, rarely seen hypotension, respiratory depression, confusion and high of creatine kinase (CK). Massive elevations of serum creatine kinase (CK) can occur in a significant number of patients treated with antipsychotic in the absence of neuroleptic malignant syndrome (NMS). In the literature there are several case reports associated with elevation of creatine kinase with the use of atypical antipsychotics. However, not only elevated liver enzymes have been reported during the use of atypical antipsychotics. Liver enzyme elevation was associated with more use of valproic acid. We report a case of a 52 year-old female patient with bipolar disorder (manic episode) who has alanine aminotransferase (ALT), aspartate aminotransferase (AST), and lactate dehydrogenase (LDH) levels increased significantly with quetiapine use. We report a case of hepatic enzyme -elevations associated with quetiapine treatment, which disappeared after drug discontinuation. To our knowledge, case number one is the first case of quetiapine-induced hepatic enzyme elevation. In this case, possible mechanisms underlying the increase in liver enzymes and symptoms clinicians should pay attention will be discussed. We recommend clinicians to monitor liver enzymes during treatment with quetiapine use.
Keywords: hepatic enzyme, quetiapine, serum creatine kinase

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[Abstract:0252][Psychopharmacology]

Buspirone in the treatment of fluoxetine-induced sleep bruxism

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Fluoxetine is a selective serotonin reuptake inhibitor (SSRI) which is commonly used in several psychiatric disorders like major depressive disorder (MD), anxiety disorders, obsessive-compulsive disorder (OCD) in children and adolescents. It is a highly tolerable drug with its low anticholinergic and cardiotoxic side effects. Common side effects of fluoxetine are headache, irritability, sleep abnormalities, nausea, diarrhea, and weight loss. Sleep bruxism is characterized by non-functional locking and grinding of the teeth in sleep, which leads to temporo-mandibulary articular dysfunction, weakening of teeth, periodontal diseases, hypertrophy of masseter, and headache. Despite the lack of controlled study on the relationship of fluoxetine and sleep bruxism, several cases were reported that fluoxetine can induce sleep bruxism. In this report, we present a school-age children with separation anxiety disorder (SAD) displayed sleep bruxism during fluoxetine therapy which responded to buspirone treatment successfully. A 7 year-old boy was referred to the outpatient clinic with complaints of inability in adapting school, fear that something would happen to his mother and that she would not be able to return to pick him up. Also, he has been complaining from headaches and stomach-aches in all mornings before going to school. His mother has been waiting at the school for two months since the beginning of school because he could not stay at school alone. He was dropped off at kindergarten for this reason. However, because he could not comply with the kindergarten with same problems, he was taken from the school by his family two weeks after admission. He was diagnosed with SAD based on clinical evaluation and the information received from teachers and family, and fluoxetine 15 mg/day was started. At the 4th week of the medical treatment, the patient's complaints improved dramatically and he was able to begin to go to school alone together with minor anxiety symptoms. However, his mother reported that he started to grind his teeth 1-2 hours every night. No nocturnal abnormal movements or daytime dyskinesia involving any other part of the body were noted by his mother. Because his SAD symptoms were improved, fluoxetine treatment was continued and buspirone 5 mg three time per day was added to the treatment based on previous reports on its effectiveness on sleep bruxism. After 5 days of the buspirone treatment, a significant reduction in the sleep bruxism was observed and the family did not note any adverse

reactions. At the end of the first month of buspirone treatment, his family reported no bruxism symptoms. In conclusion, this report suggest clinician should be aware of the possibility fluoxetine induces bruxism in childhood as in adulthood. Further systematic studies are required in order to establish the relationship between fluoxetine and bruxism and to clarify the effectiveness of buspirone in the treatment of these cases.
Keywords: buspirone, bruxism, fluoxetine

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[Abstract:0253][Psychopharmacology]

Atomoxetine treatment for depression and attention deficit hyperactivity disorder (ADHD) comorbidity: a case report

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In this poster, it is intended to present a case with the symptoms of difficulty in maintaining attention, amnesia, making simple mistakes in exams, plucking and eating hair. Also definitive diagnosis and treatment of this case will be explained in this poster. Reviewing the knowledge in literature and comparing it with the case is the method being followed.
Case: A 14 year-old female patient came to our clinic first time in December 2011. Her mother mentioned that she has been plucking and eating her hair since early years and her friends have been making fun of her behaviors so that the relationship with her friends is affected and she has been lonely most of the time. Her mother said that she is having difficulty in maintaining attention, amnesia and making simple mistakes in exams so her success rate in the school is decreased. Besides, her mother told that her teacher is also complaining about the same symptoms. In psychiatric examinations and psychometric tests, it is determined that the patient has diagnoses of depression and depression related ADHD symptoms. After, Selective Serotonin Reuptake Inhibitor (SSRI) treatment planned for the patient. Fluoxetine, escitalopram and fluvoxamine treatments are started respectively. Yet, the treatment of the patient who evolved enuresis symptoms with all the three agents' dose independent was continued with atomoxetine. Atomoxetine is a potent inhibitor of the presynaptic norepinephrine transporter, with minimal affinity for serotonin and dopamine transporters in frontal cortex. Norepinephrine transporters in prefrontal cortex have a role in norepinephrine and dopamine reuptake. Atomoxetine is an inhibitor of presynaptic norepinephrine transporters in the prefrontal cortex, as a result dopamine and norepinephrine increase. Atomoxetine is used alone in depression and ADHD comorbidity or used with stimulant, SSRI and bupropion.
Keywords: atomoxetine, depression, enuresis

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[Abstract:0254][Schizophrenia and other psychotic disorders]

Delusion of pregnancy in a 50 year-old female patient

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Delusion of pregnancy is relatively infrequent syndrome that might be discrete or a part of other psychiatric disorders. The literature about delusion of pregnancy is based on case reports and less than 90 cases have been reported since 2000. Although delusion of pregnancy can be seen in different ages, one fourth of the reported cases are developed in women after the age of 50 years. Here, we aimed to present a female patient with delusion of pregnancy as a somatic delusion.
Case: A 50 year-old female patient was referred to outpatient clinics of psychiatry with thought of "being pregnant" and "suspicion of missed abortus" from an obstetrician. We obtained the history of amenorrhea after irregular menorrhea complaints, negative finding in urinary pregnancy test, and several applications to obstetrics clinics with negative findings after few abdominal ultrasonography

administrations and B-HCG level within normal range. At her psychiatric examination: delusion of being pregnant, paranoid delusions of "not detecting or concealing the pregnancy by obstetricians is due to threats of her husband", and tactile hallucinations of "fetal movements and birth squeeze" were present. Behavior of food craving was detected. No insight was observed and judgment was impaired. Conversion disorder was detected in her previous history which has been no longer present for 12 years. Suicide history in her aunt was detected. Chronic marital conflicts were present which led her to make a decision about divorce that was closer to initiation of her distorted thoughts about pregnancy. Laboratory findings including the brain MRI were within normal range. Risperidone 1 mg/day was initiated and the dose was planned to titrate taking account the response rate. However, patient did not adhere to treatment and follow up.

One fourth of patients develop delusion of pregnancy after the age of 50 years. Psychodynamically, pregnancy is a special relationship between mother and fetus that may put loneliness and helplessness away. Furthermore, delusion of pregnancy might be caused by acute loneliness, real/imagined loss of a relationship. In cognitive view, perceptions as somatic sensations from abdomen such intestinal dilation, and endocrinological changes such as the consequences of antipsychotic -induced hyperprolactinemia or peri-menopausal symptoms might be distorted as signs of pregnancy. Our case was in line with these above mentioned suggestions in literature. The duration of delusion of pregnancy is variable up to 20 years and in our case it has been present for one year and the patient misinterpreted this over prolongation as missed abortus. Although our case had a delusion of pregnancy for one fetus, there are some cases with delusion of pregnancy for multiple fetuses. Nosologically, the difference between delusion of pregnancy and pseudocyesis might be important and pseudocyesis is primarily based on the presence of physical signs and symptoms of pregnancy. It may be delusional or not. If pseudocyesis is without delusion, it should be included in the other specified somatic symptom and related disorder, in DSM-5. In conclusion, delusion of pregnancy is a relatively infrequent entity that can be seen in various neuropsychiatric disorders with variable clinical courses.

Keywords: delusion, delusional pregnancy, delusion of pregnancy, pseudocyesis, psychosis

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[Abstract:0257][Psychopharmacology]

Facial and bilateral leg edema in a patient using quetiapine

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Quetiapine's mechanism of action is not fully elucidated though it involves antagonism at serotonin type 1 (5-hydroxytryptamine [5-HT1A]) and type 2 (5-HT2A, 5-HT2C) receptors with relatively weak antagonism at dopamine (D1, D2) receptors. In addition, quetiapine exhibits some α1-adrenergic antagonism that may explain its cardiovascular side effects, like orthostatic hypotension. Bilateral leg edema has been infrequently described with several atypical antipsychotics, including case reports with olanzapine, risperidone, and ziprasidone. Currently, there are some published cases of peripheral edema related to quetiapine in literature and one case with facial, eyelid and added bilateral lower extremity edema. Edema is not currently listed as a potential complication in its prescribing information. Here, we report a case presented simultaneously with both face and leg edema associated with quetiapine use.

Case: A 54 year-old female patient with bipolar disorder presented to our outpatient psychiatry clinic for facial and bilateral leg edema. She had been hospitalised in a mental health hospital for a manic psychosis episode, treated with haloperidol and quetiapine for about one month. She was discharged on haloperidol decanoate 50 mg/ml and quetiapine 600 mg/day. She developed 2+ bilateral leg and face edema after 8 days from hospital discharge. Cardiac and pulmonary exam and blood pressure measurements were within normal limits. Laboratory work-up was unremarkable (thyroid stimulating hormone (TSH), albumin, electrolytes, blood urea nitrogen (BUN), creatinine, erythrocyte sedimentation rate, N-terminal fragment brain-type natriuretic peptide, complete blood count with differential, and urinalysis). In accordance with cardiology consultation, treatment with furosemide started which decreased edema 1+ bilaterally. We did not suspect haloperidol decanoate to be the cause of edema in highlight of the literature but there were some cases of quetiapine associated peripheral edema. One week after decreasing the dose of quetiapine from 600 to 300 mg/day, edema had improved. No other furosemide treatment were implemented. Despite other reports, our case had edema after 38 days of quetiapine use and she is the first case having both face and leg edema simultaneously.

With only one case report, a dose-dependent relationship between quetiapine and leg edema can not be clearly established. In the case described by Rozzini et al., a dose increase from 100 mg to 150 mg quetiapine daily was followed the next day by bilateral leg edema correspondingly our report describes symptomatic improvement when the quetiapine dose decreased but not discontinued, perhaps

suggesting a dose-response relationship. Time to appearance of leg edema after initiation of quetiapine was 38 days. Time to resolution after drug discontinuation was one week. The mechanism of quetiapine-induced edema remains uncertain though likely parallels that of other second-generation antipsychotics. Further clinical observation and research is needed to clarify the characteristics, risk factors, dose-dependence, and potential mechanisms of quetiapine-associated edema. We hope our report will alert physicians to this potential vascular complication to promote prompt recognition and intervention.
Keywords: antipsychotics, edema, quetiapine

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[Abstract:0261][Mood disorders]

Affective disorder following cerebrovascular disease: a case report

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Cognitive impairment, motor dysfunction and emotional changes after cerebrovascular attack are the major cause of disability at the middle age and older populations. From the neurological point of view, emotion is a multidimensional process in response to external and internal stimuli, which implicates perceptive (fear, joy, disgust), motivational (arousal), autonomic (sympathetic reaction), motor response, and cognitive evaluation. After stroke, emotional disturbances include Mood disorders, emotional dyscontrol, and alteration of emotional reactions. These symptoms are unrecognized by clinicians, not only in the acute phase, but also in the chronic stage, when behavioral changes are noted. Affective disorders are important to consider in stroke patients, since they may influence neurological recovery and treatment response.
Case: A 60 year-old female prisoner was hospitalized in order to receive a forensic medicine expertise. Approximately five years ago, she had a treatment due to a cerebrovascular disorder. After this period, her psychiatric complaints started which included disorganized and increased amount of speech, psychomotor activity, insomnia, aggressive behaviors and irritability. She was hospitalized at the psychiatry department of the Manisa State Hospital for psychiatric diseases. According to the information gathered from the family of this patient, she has not been taking her medications for the last six months, which as a result caused the above mentioned complaints to be observed again. After the first psychiatric evaluation of the patient, olanzapine 5 mg/day was advised because of the diagnoses of an affective disorder following a cerebrovascular disease. During this period of the time, she was consulted to a neurologist with a CT, EEG, MRI of the brain, and Bilateral Carotid and Vertebral Artery Doppler USG. In addition, acetylsalicylic acid 300 mg/day was started due to the diagnoses of atherosclerotic variations. At the end of all these examinations and treatments, a forensic report was completed with the diagnoses of the organic mental disorder with affective features. After following a partly cessation of her complaints, she was discharged with a medication of olanzapine 5 mg/day and acetylsalicylic acid 300 mg/day.
The relationship between cerebrovascular disorder and neuropsychiatric disorder has been known for more than hundred years. The most possible emotional disorder in relation with cerebrovascular disease is most likely the depression. But, mood swings and emotional hyperactivity are well known after bilateral cerebrovascular lesion and in vascular dementia. However, it has been known that post stroke mania is strongly associated with both a right hemisphere lesion in a limbic-connected area and a second predisposing factor, such as genetic loading for affective disorder, pre-existing sub cortical atrophy or seizure disorder. In this case, we have diagnosed an affective disorder following a cerebrovascular disease. The MRI reports of her were compatible with risk factors as mentioned above. In conclusion, the patients who have psychiatric symptoms after cerebrovascular disease have to be evaluated comprehensively with risk factors, laboratory results, and a family history to ensure an appropriate treatment for this special group of cases.
Keywords: affective disorder, cerebrovascular disease, forensic case

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[Abstract:0264][Forensic psychiatry]

Evaluation of criminal lability in a mother and daughter diagnosed as schizotypal personality disorder

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A 57 year-old mother and a 32 year-old daughter accused of calumniation were referred for evaluation of criminal liability. Patients were accused of systematic rape by soldiers but, there was no evidence of soldiers DNA although DNA of an old man was found everywhere. When asked about that, patients refused to talk, they were very defensive, gave short answers, their affect was anxious, mother's mood was depressive, they had no hallucinations but had overvalued persecutory and referential ideas that were not of delusional strength. They were hospitalized, put in different services and started on low dose of antipsychotic. After treatment, their defensive attitude softened. It was learned that they had been living in an isolated way for a long time. They had always been suspicious, did not trust people and thought that they would be harmed by people in a magical way. Their suspiciousness and distrust increased after their father's death 3 years ago. The mother thought that people had done some magic to destroy her relationship with her husband, daughter also thought that a magic was done to their family and believed that her father's personality changed after that magic and he had become irritable, then couldn't move, became bedridden and died. The daughter thought that this magic affected both her and her mother and they felt ill sometimes because of that magic. The mother's illness started after her first labor and she became withdrawn eating and talking nothing and was taken to that hodja and became well after that visit but her symptoms recurred from time to time. After the father's disease, they decided to invite that hodja believing that he had some supernatural powers and would help them to overcome that magic and heal them. During his stay, hodja told them that there was magic on them and that it couldn't be destroyed and they would continue to be harmed by people who made that magic if they do not obey him and do whatever he says. After these intense suggestions, they started to have sexual intercourse with hodja, daughter believed that her mother's illness was getting better when she had sexual intercourse with hodja and believed that her mother would die if she did not do that. They started to get more isolated from the environment and got more suspicious of people and totally stopped their relationships with even their closest relatives. After soldier's visit to their home to investigate their fathers suspicious death, hodja who was also being searched by soldiers because of fraudelence helped patients to write an accusation letter about soldiers. SCID I-II evaluation revealed that both had schizotypal personality disorder and mother had recurrent depression. MRI of the brain was normal and lab evaluation showed that mother had subclinical hypothyroidism. Although they were separated, their belief in magical power of hodja continued in both of them, so shared psychotic disorder was ruled out. It was decided that, on a schizotypal personality background, their reality testing was distorted from time to time upon stress and intense suggestions of hodja, so their criminal lability was partially affected.

Keywords: schizotypal personality disorder, suggestion, criminal liability

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[Abstract:0267][Others]

Frontal Lobe Syndrome

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Frontal cortex damage is a neuropsychiatric disorder rarely assessed in psychiatry clinics. Neurological and/or psychiatric symptoms might be detected due to damage of frontal lobes as detected in damages of many brain regions. Symptoms due to prefrontal region damage, varies according to the size and location of the lesion. In this case; we have a frontal cortex damaged patient whose symptoms

were related with behavioral changes such as impaired social functioning, attention deficit, difficulty in communication, disinhibition, aggression and combativeness.

Case: A 38 year-old single male patient consulted the physician for his attention deficits, memory and impulse control problems. He had never used alcohol or psychotropic agents and never consulted a psychiatrist before. It has been specified that patient's academic success has been normal and showed normal development until he had a car accident. He had injured in a car accident 2 years ago, frontal damage occurred and was retained in the intensive care unit for twenty days. At his brain MRI reports Frontal encephalomalacia was identified especially at the dorsolateral prefrontal cortex, ventromedial prefrontal cortex and orbitofrontal cortex region. After this accident he had 1 year convalescence period. In the physical examination, general condition of the individual was good, his conscience was clear; he was oriented and cooperative. An operation scar at the left and right frontal area and surface level differences compatible with the bone defect at the same area were observed with palpation. At the neurologic examination, no pathology was determined. At the psychiatric evaluation, conscience was clear, oriented, cooperative, self-care was compatible with his socio-economic status, he was eager to interview and his eye contact was normal during the interview. Cognitive tests evaluating the overall intelligence and frontal lobe functions were administered. As a result of Porteus Labyrinths and Kent E-G-Y tests, general ability and intelligence score was observed as 105 (at the normal limits). The results of the Stroop and N-Back Test measuring the ability to prevent the inconvenient reaction and selective attention functions were determined within the abnormal range considering the age and educational status of the individual. Wisconsin Card Sorting Test (WCST) which evaluates the executive functions, attention and abstract assessment resulted lower than the expected average values. Total blood count, routine biochemistry and thyroid function test results were normal. Electroencephalography and electrocardiography scans were also normal. In the Magnetic Resonance imaging (MRI) of the brain; bone defect in the right frontal area, encephalomalacia and parenchymal volume loss compatible with gliosis, increase in intensity at the area adjacent to the bone defect and focal sulcus enlargement secondary to parenchymal volume loss were observed (Figure 1, 2, 3, 4). Following the evaluations, he was diagnosed as having 'behavioral disorders due to brain disease damage and dysfunction (frontal lobe syndrome).

In this case; symptoms related with behavioral changes such as impaired social functioning, attention deficit, difficulty in communication, disinhibition, aggression and combativeness were observed. The imaging and clinical findings in the case were evaluated in line with prefrontal cortex damage. In the mental examination, no finding arose from any other psychiatric disorder was determined. Pre-traumatic psychosocial development was normal and differential diagnosis has been made by determining organic reason. Frontal damaged peoples' symptoms can be confused with non-organic based schizophrenia, attention deficit and hyperactivity disorder and impulse control disorders. Executive dysfunctions and impaired judgement occurring by the influence of the disease can affect the criminal responsibility in a decreasing or completely removing way in case of a criminal act. We have to be careful about the frontal damage history for the patients with these symptoms and neuroimaging should be evaluated.

Keywords: behavioral symptoms, frontal cortex damage, personality disorders

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[Abstract:0270][Mood disorders]

Venlafaxine use in treatment resistant major depression and association with hypertension

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Major depressive disorder is a disease that markedly reduces the quality of life and causes disability. Currently, about one-third of patients with major depression was reported to be unresponsive to antidepressant therapy. Venlafaxine is anti-depressant serotonin/noradrenalin reuptake inhibitor drug which alternative forms of treatment-resistant major depression (TRD). In our case, The use of venlafaxine will be described with hypertension in clinical comorbidity treatment resistant major depression patients.

Case: A 54 year-old female patient, widow, has two children, recurrent thoughts of suicide, suicide attempts is still continuing, irritability, decreased self-esteem, unhappiness, inability to enjoy life was hospitalized due to complaints. 15 years history of depression, learned to 5 years history of hypertension. The diagnosis of major depression was made Structured Clinical Interview for DSM-IV (SCID 1). In the first interview the patient Hamilton Depression Rating Scale (HDRS) score 25, suicidal behavior scale (SBS) 13, Clinical Global Impression (CGI) disease severity score of 6 were evaluated. In the last four years the application of different treatment the control of the psychiatrist regularly but learned there is insufficient improvement in treatment results. Venlafaxine treatment was started TRD patients by considering. 150 mg dose increase was made up. HDRS 16, SBS 4, CGI severity of illness was evaluated as 4. The mean arterial blood measured pressure was 155/95 mmHg. It was consulted with cardiology. Continuation of existing triple antihypertensive therapy was

recommended. The elevation in blood pressure was reported to be concluded for that psychogenic. Venlafaxine dose was increased up to 225 mg. HDRS score 8, SBS score 6 evaluated. The mean arterial blood measured 135/80 mmHg. Currently, our patient treatment continues for 3 months with the current antihypertensive therapy and venlafaxine 225 mg. The mean day morning and evening looking including blood pressure values measured in the clinical follow-up was 130/80 mmHg. HAM-D scores were 4, SBS scale score of 0, the CGI severity of illness as 1 was evaluated.

Venlafaxine, which is nausea, blurred vision, asthenia, akathisia, sexual and cardiac side effects a drugs. The most serious side effects of the cardiac of hypertension. Especially 300 mg and higher doses to causing hypertension has been reported. However, most cases are mild increase in blood pressure and self-limiting. Therefore, absolute contraindication it has not been reported patients with hypertension venlafaxine regarding. In our case, The patient's medical comorbidities as hypertension, when the upgrade is started at 225 mg venlafaxine treatment mean arterial blood pressure is 130/85 mmHg together with a significant decrease in HDRS scores and SBS shows blood pressure regulation can be facilitated depending on the reduction of emotional stress. However, it is important for psychiatric assessment also in patients with treatment-resistant hypertension should be remembered, comorbid psychiatric disorders can be found next to the medical disease should be considered. Based on these results, the use of venlafaxine should be avoided in TRD patients comorbid with hypertension. In these patients, blood pressure monitoring and cardiac check ups should be carefully conducted.

Keywords: hypertension, treatment resistant depression, venlafaxine

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[Abstract:0271][Autism]

The effect of sertraline on self-injurious behavior in autistic spectrum disorders

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Autism spectrum disorders (ASD), a lifelong neurodevelopmental disorder spectrum characterized by problems in communications, disturbances in social interaction, restricted interests, and repetitive movements. In treatment of ASD, priority areas of pharmacotherapy are irritability symptoms(severe tantrums, aggression, self-injurious behaviors, restricted stereotypic movements, hyperactivity). Antipsychotics, alpha-2 agonists, mood stabilizers, stimulants, atomoxetine, and naltrexone can be used. Self-injurious behavior is a class of behavior, causes more specifically tissue damage and physical harm to oneself. According to the researchers ASD is with approximately 50% self-injurious behavior throughout life. In this presentation the case whom followed-up with ASD and resistant self-injurious behavior, will be discussed about reduction of aggression with sertraline.

Case: A 7 year-old male patient, has been followed-up for ASD since 3.5 years old, referred to us because of self-injurious behavior and hyperactivity. When he presented to our department, he was receiving valproic acid (VPA) 1250 mg/day and olanzapine 25 mg/day. Detrimentally dabbing motion in particular to right cheek region was a treatment-resistant complaint so far. The average number of dabbing was 70-80 times per hour. His baseline Aberrant Behavior Checklist (ABC) total score was 108, irritability subscale was 41, stereotypy subscale was 18. In treatment history there were intermittently usage of risperidone, aripiprazole, and diazepam in variable doses. Zuclopenthixol was added stepwise up to 18 mg/day. After pediatric neurology consultation, VPA level was reduced to 800 mg/day, lorazepam 1.5 mg/day and biperiden 2 mg/day were added. Despite these his complaints were continuing, sertraline 25 mg/day was added. Two days after addition of sertraline, his dabbing motion decreased markedly such as 5-10 times per day. Because of no benefit from zuclopenthixol, discontinued stepwise and haloperidol was added. After one month of hospitalization, the treatment was adjusted to VPA 800 mg/day, olanzapine 20 mg/day, biperiden 3 mg/day, lorazepam 1.5 mg/day, sertraline 37.5 mg/day and haloperidol 15 mg/day. His dabbing, hyperactivity and irritability were decreased and there was a significant progress in general accordance compared to the hospitalization. After a month his ABC irritability subscale score was decreased 36.5%, stereotypy subscale score decreased 61%, and total score decreased 65%.

There is evidence of deterioration of the regulation of serotonin in pathogenesis of ASD and findings about that serotonin affects the phenotype of behavior in autism. The similarities between Obsessive Compulsive Disorder and ASD about restricted interests and repetitive behaviors are considered to constitute evidence of serotonin metabolism dysfunctions. Antidepressants have become a new point of interest for the treatment of ASD. Most studies have focused primarily on the effects of antidepressants on repetitive behavior, more emphasis is on SSRIs because of fewer side effects profile. In studies, antidepressants found predominantly reducing behavioral symptoms (compulsions, repetitive behaviors, poor social communication) in autism. Sertraline is an SSRI and also partially has dopamine reuptake inhibitor effect. According to a study with adult ASD individuals, after 12 weeks sertraline application, restricted repetitive

behaviors and aggression were reduced. Still in a study with 42 adult diagnosed ASD (mean age 26), aggression and repetitive behaviors were reduced with sertraline in 57% of patients as our case. Although it is less known in this area involving children several studies have been conducted in recent years. According to a study with 9 autistic children (6-12 years) the behavior of aggression was reduced 33% after a few months of sertraline application, likewise our case. In that study the range of dose is 25-50 mg/day like our case, doses above 75 mg/day symptoms were reported to worsen.

Keywords: autism, self-injury, sertraline

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[Abstract:0272][Psychopharmacology]

Aripiprazole in treatment of fluoxetine -induced bruxism: a case report

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Bruxism is a repetitive jaw-muscle activity characterized by clenching or grinding of the et or by bracing or thrusting of the mandible. Bruxism is a common problem and may cause several symptoms which include jaw-muscle hypertrophy; tooth wear; fracture or failure of teeth restorations or implants; sensitivity or pain of teeth, muscles or joints; and temporomandibular joint displacements. Bruxism is termed "secondary" when occurs in the presence of a neurological or psychiatric disorder or following in the use of medication. L-dopa, neuroleptics, amphetamine and selective serotonin re-uptake inhibitors cause bruxism.

Case: An 11 year-old male patient with severe obsessive compulsive disorder (OCD) and fluoxetine -induced bruxism successfully treated with low-dose aripiprazole. Patient who had been followed-up with OCD diagnosis for 4 years was treated with fluoxetine and cognitive behavioral therapy. Fluoxetine 20 mg was started and then increased to 30 mg, due to having sexual obsessions at 9 years old after 13 months asymptomatic period. Patient's OCD symptoms has improved bruxism that milder for years has significantly increased. After it has said that dental surface has sustained, fluoxetine has decreased to 20 mg and then bruxism has slightly relieved. Aripiprazole was started 0.25 mg because both augmentation of OCD treatment and effects on relieving bruxism. Bruxism was sligtly increased after administering 0.25 mg dose for a week. It was reported that symptom has slightly relieved, when dose has increased to 0.50 mg. It was observed that bruxism has significantly decreased, when dose has increased to 0.75 mg after a week. Aripipirazole treatment has ended over time after six months. Bruxism has significantly improved according to beginning.

The pathophysiology of SSRI associated nocturnal bruxism is unclear. It has been proposed that dopamine and serotonin involved in regulating motor pathways is the origin of SSRI-induced sleep bruxism. The mechanism hypothesized is that an excess of serotonin in the synapses leads to an inhibitory effect on the release of dopamine from the mesocortical tract that evolves itself in specific forms of oromandibular akathisia, thereby leading bruxism. Several case reports and studies showed that dopamine agonists may be successful in treating bruxism. The adminisitration of the dopamine precursor levodopa, D2 receptor agonist bromocriptine and pergolide caused decrease in the number of bruxism episodes. Aripiprazole is a partial dopamine agonist at the D2, D3 and 5HT1A receptors and antagonist at the 5-HT2A receptors. It is hypothesized that its beneficial effect on bruxism is attributable to its partial agonist activity on dopamine receptors and 5-HT1A receptors, jointly overwhelming its antagonism at 5-HT2A receptors. It was impossible that ending the medication because patient has benefits of fluoxetine treatment and patient can not use any other tablet SSRI medication. Aripipirazole has begun for preventing the damage that bruxism could cause dental and temporomandibular joint. After symptoms have decreased, aripipirazole has ended over time. Relief on bruxism has continued at second month after medication has stopped. Our case is the first report of SSRI-induced bruxism succesfully treated with low-dose aripipirazole in children.

Keywords: aripiprazole, bruxism, children, fluoxetine

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[Abstract:0274][Psychotherapies]

A supplemental approach to dissociative disorder: a case report

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Common feature of Dissociative phenomenon is deterioration or change in memory, identity or consciousness which is normally integrative. Modern dissociation theories agree on childhood traumas play a central part in progression of dissociative disorders. It's thought while dissociation were used as overcoming of traumatic living at the beginning, it is turned into pathological progress over time.
Case: Our patient is a 30 year-old female and married with 2 children. She had some troubles with her spouse's family at their engagement period. Her marriage generally troubled. After an argue with her husband, she consulted emergency department complaining about headache, crying jag, nervous temperament and agitation. After taking 1 mg lorazepam she was calmed down and slept trough the night. When she woke up in the morning she had amnesia about her problems and arguments with her husband and his family and process of her divorce though she remembered her children's names, their birthdays, present and other family members. There are no diseases detected from organicity tests that is requested from the patient (blood tests, brain MRI, EEG) and neurologic consultation. The validity scales configuration in MMPI profile and from clinical scales the increase near the limit values in the Hy low test showed that hysterical personal features, intensive emotional supression defense mechanism and inadequate insight. Dissociative Experience Scale (DES) score was 45%, and Dissociation Scale (DIS –Q) score was 2.9; values were above the cut-off scores and also were evidencing positive for dissociative living. In the Rorschach test, the structure of tongue is labile; in the fluent expression of patient, labile identification, alongside with the seeking of an affective support and hysterical dramatization depressive effect is primarily noticed. It is found that the patient, whose comprehension of reality is solid and strong, is using the suppression defence intensively, the problematique around castration anxiety is formed in oedipal period and created a hysterical – neurological function. The onset of illness shortly after going to the court for getting divorce brings that there are differentiation between wishes and desires at the apparently conscious and unconscious level to mind. As Nasio said; hysterical person's unconcious pathological fantastic logic enforces to agree on to other despite of herself or himself (conscious wishes). In that fantasy, hysterical person plays unhappy and unsatisfied victim. Case wonders what happened to her as well as why she cannot remember somethings and because she suffers physically, she denies any anger and forgets immediately when she was reminded of things that she forgot. As Nacio quoted from Freud, person's attention, who works on hysteria, goes towards to fantasies, which are source of these symptoms, with getting out of symptom analysis. In this case also practice of psychoanalytical psychotherapy has started with face to face in once a week. Although it has not been more than 2 months (start-up phase), a decrease had seen on anxiety which is caused by disremembering and forgetting. Patient cooperates with therapist on researching of reasons of these symptoms.
Keywords: dissociative disorder, Rorschach's test, psychoanalytical psychotherapy

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[Abstract:0278][ADHD]

Comorbidity of congenital adrenal hyperplasia and ADHD, ASD

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Congenital adrenal hyperplasia (CAH) is a disease group which develops as a result of the deficiency of one of the enzymes which are necessary for cortisol synthesis. There is androgen excess. Androgen levels one is exposed to during the prenatal development affect brain permanently. The fact that brain development is exposed to androgens within this sensitive period of time causes behavioral changes. Some researchers have thought androgens to be responsible for some of the psychiatric disorders which are common in males. This case report will discuss the comorbidity of ASD and ADHD in a CAH case.
Case: A 10 year-old 5th grader female patient presented to our outpatient clinic for the first time when she was five year-old with the complaints of hyperactivity, restlessness, problems in relationships with peers and preferring the games and toys of boys. It was learned

from the mother she was diagnosed with CAH which coursed with 21-hydroxylase deficiency and delay in the development of spoken language. It was learned that her complaints about attention deficiency were realized after she started school, she had limited eye contact, she had difficulty in communicating with her peers, she was extremely interested in especially planets and dinosaurs, knew the names and orbits of all planets, watched videos about them again and again and she could not communicate properly with her peers. As a result of the psychiatric interviews, the patient who was diagnosed with ADHD and ASD is still being followed by our polyclinic.
In CAH cases, the decrease in cortisol synthesis increases the release of hypophysial ACTH and hypothalamic CRH. The increase in these hormones causes the release of more cortisol and aldosterone processors. Since these processors slide to the production pathway of sex steroids, androgen excess and the clinical manifestation of this arises. In girls with a diagnosis of CAH, there is an increase in playing boys' games and preferring to play with boys and there is a decrease in preferring girl games. ASD is more popular in boys when compared with girls. While the male/female rate is 4/1 for autism. This gender difference is thought to result from the levels of androgen in prenatal period. In a study conducted with a diagnosis of CAH, autistic properties were seen more in the CAH group when compared with the healthy relatives. In this case report, the comorbidity of ASD and CAH which are rare disorders in girls brought to our minds that levels of exposed androgen may be effective in the etiology of ASD. The male/female rate is 3/1 for ADHD. A deficiency in the dopamine system plays a primary role in the neurobiology of ADHD disorder. Androgens are known to modulate the dopamine activity in the mesolimbic system. The fact that our patient was exposed to increased androgen can be a risk factor for the formation of ADHD. In conclusion, hyperandrogenism exposed in prenatal period may be a risk factor for ADHD and ASD.
Keywords: ADHD, ASD, congenital adrenal hyperplasia, androgen

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[Abstract:0283][Psychopharmacology]

Temporomandibular joint dislocation as a result of neck and face dystonia based on use of risperidone: a case report

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Acute dystonia is characterized by involuntary, continuous or spasmodic muscle contractions in head and neck region. This phenomenon, in 90% of the patients, occurs in the first four days of antipsychotic treatment or as a result of overdose. Antipsychotic medications causes dystonia through D2- Dopamine receptors. Risperidone effects this receptor and gives result to acute dystonia. Acute dystonias generally give a good response to treatment. And if response to treatment is delayed, temporomandibular joint dislocation may occur. In this case report, we aimed to emphasize the diagnosis of temporomandibular joint dislocation in patients who have not ameliorated the acute dystonia in orofacial region.
Case: A 40 year-old male patient, followed with the diagnosis of obsessive compulsive disorder and schizophrenia for 15 years. He used different antipsychotics in this period. He started his clomipramine 225 mg/day and aripiprazole 20 mg/day treatment ten years ago and he did not take those medications regularly in the last 4 years. He used clomipramine 75 mg/day in the last one month. Because of his aggression, agitation, insomnia and with the suspicion of self- injury, his family added risperidone 4 mg to his meals two times a day. Two days later, the patient showed swallowing difficulties, difficulty in speaking and spasm of the neck region. The patient presented to our policlinic with those complaints and physical examination revealed that he was not able to swallow his saliva because of swallowing difficulties, his speech was disturbed, he was not able to close his jaw and his neck movements were restricted. He was internalized with the diagnosis of acute dystonia related to risperidone use. Biperiden 6 mg/day was started orally for the treatment of orofacial region dystonia. Antipsychotic treatment was changed to clozapine and increased to 100 mg/day. One week later his dystonia regressed partially but he was not able to close his jaw and had difficulty in speech. He was consulted in otorhinolaryngology clinic because of clicking noise and pain of his jaw. As a result of consultation, bilateral temporomandibular joint dislocation was identified, and reduction was administered, elastic bandage was put. His clic noise on the jaw was lost and his pain regressed in the follow up, he was discharged from the hospital. The rate of acute dystonic reaction as a result of risperidone use is not known completely. In a study which investigates the side effects of high dose risperidone use, 5 of 38 patients (11%) with side effects of risperidone showed dystonic reaction. It was reported in literature that acute dystonic reaction as a result of antipsychotic use has proceeded to temporomandibular joint dislocation just in very few cases.
As shown in this case, diagnosis of temporomandibular joint dislocation can only be made by taking this possibility into consideration and by making physical examination oriented for this. Even it is rarely seen in the patients with insufficient responses, with acute dystonia

in orofacial region, temporomandibular joint dislocation diagnosis should be considered.
Keywords: risperidone, temporomandibular joint dislocation, acute dystonia

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[Abstract:0289][Mood disorders]

Varenicline induces manic shift in a depression patient: a case report

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Varenicline (known as Champixin in Turkey) is partial agonist at the α4β2 nicotinic acetylcholine receptor. Varenicline is currently the most effective smoking cessation medication. This drug was blamed for manic shifts in bipolar patients. In addition, manic symptoms were observed in two patients who did not have prior diagnose as bipolar disorder after discontinuing varenicline therapy. We report a female patient who had a manic shift after receiving Varenicline while she was under sertraline treatment.
Case: A 31 year-old single female patient presented to psychiatric emergency department with complaints of increased energy, decreased need for sleep, ideas of new projects like writing a book, overvalued idea of being accurate in fortune telling, and irritability which were present for one week. In her history, varenicline had been recommended 20 days ago while she was under the sertraline therapy aiming to treat nicotine addiction although she continued to smoke. At mental examination: speech amount was excessive with loosening of association, mood was irritable, and affect was labile. Young Mania Rating Scale score was 41 points. The family of her did not consent for hospitalization but accepted to cease sertraline and varenicline treatment. In previous history, she had been operated for aortic valve replacement two years ago. Then, the patient had been consulted to a psychiatrist for her low-mood and self-confidence problems. Escitalopram 10 mg/day had been given for her depressive mood. After the first month of escitalopram use, her psychiatrist had observed decreased need for sleep, excessive speed and amount of speech. The escitalopram therapy had been switched to sertraline 50 mg/day because of ongoing depressive symptoms. She was under sertraline treatment for two years and was adherent for appointments of psychiatric follow up. Manic symptoms in our patients appeared after adding varenicline on sertraline therapy. We considered that varenicline would induce manic shift. Probably varenicline is responsible of dopamine secretion from neurons in ventral tegmental area via nicotinic receptors in ventral tegmental area. Or considering hypomanic symptoms under escitalopram therapy in her previous history as a sign of vulnerability, we proposed that concomitant use of varenicline and sertraline might reveal manic symptoms. Interestingly, manic symptoms have been associated with discontinuation of varenicline in several cases while manic shift in our case seems to be caused by the start of varenicline therapy.
The patients who are under concomitant antidepressant and varenicline therapies have to be informed about the early manifestations of manic symptoms for prevention or early intervention.
Keywords: varenicline, manic shift, side effect

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[Abstract:0290][Mood disorders]

Can olfactory hallucination be an early sign of manic episodes?: a case report

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The bipolar prodrome constitutes a period of disturbance characterized by some signs and symptoms that lead up to the onset of the full-blown disorder. Early recognition of their can be used to control of an impending manic episode. Here, we aimed to present an infrequent

symptom as olfactory hallucination of pleasant smelling during the manic episode in a patient with bipolar disorder.
Case: A forty eight year-old male patient presented to our outpatient clinics because of elevated mood, expanded self-esteem, increased psychomotor activity and pleasant smells. His first manic episode had occurred about 20 years ago. He was being followed by our outpatient clinics for three years and was on lithium 900 mg/day (lithium levels were between 0.6 and 1.2 mEq/liter). The lithium dose was reduced to 300 mg/day from 900 mg/day within five one month because of his mood has remained stable for the last two years. After the lithium dosage was reduced, his symptoms began about a month later. The blood lithium level was 0.31 mEq/L. Laboratory examinations including biochemistry, complete blood count, thyroid function tests, urine screen, electrocardiography, electroencephalography and cranial magnetic resonance imaging were within normal ranges. Olfactory hallucinations were not detected in that previous episodes. Lithium was rapidly titrated up to 900 mg/day. After one month lithium level was 1.1 mEq/L. Young Mania Rating Scale decreased from 14 to 8 and olfactory hallucination was disappeared.
Olfactory hallucinations are known as prodromal symptoms of many neurological diseases (epilepsy, migraine, etc.), but they are reported as more common among patients with bipolar disorder than expected. The mechanism of olfactory hallucinations is not yet fully understood. Pleasant smells may be an early sign of manic episode in bipolar disorder as reported in a previous case. In the current literature there are some cases with different diagnosis in whom olfactory hallucinations have been successfully treated with mood stabilizers. In our case, besides the other manic symptoms, lithium was successful in treating olfactory hallucinations. During the patient and/or residency education for early signs of mania such as decrease need for sleep, it may be important to draw attention on pleasant smells that might be helpful for early diagnosis and intervention.
Keywords: olfactory hallucination, early sign, manic episodes

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[Abstract:0291][Others]

A case of stuttering due to olanzapine treatment

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Stuttering is defined as the disturbance in timing and fluency of speech inappropriate with the age. There are two types of stuttering classified as acquired and developmental. Developmental stuttering is more often, begins at childhood or puberty and may evolve slowly. Developmental stuttering generally occurs between ages 2-7. Acquired stuttering may begin at any age due to stroke, head injury, brain neoplasm and other situations that affects brain. Pathophysiology of stuttering is not certain. Some published studies show that, stuttering is related with increased number of D2 receptors at basal ganglia. Also 50-200% rate of increased dopaminergic activity found at stuttering cases in comparison to the control group.
Case: A 21 year-old male patient presented to our psychiatry outpatient unit with increased psychomotor activity, aggressive behavior, increased rate of speech. His parents gave the information that he had taken synthetic cannabinoid a day ago. During psychiatric examination a partial cooperation could be established with him, he was talking meaningless and abusive. The results of laboratory tests (CBC, AST, ALT, urea, creatine, electrolyte panel, infection panel, endocrine tests, EEG, Brain MRI) were normal. In the emergency service 10 mg haloperidol, and 5 mg biperiden intramuscular were administered to the patient. Then he has been hospitalized at psychiatry inpatient unit, and 10 mg/day oral olanzapine treatment was initiated. At the second day of olanzapine treatment, fluency and timing disturbances in his speech began and at the fifth day this symptoms increased. He did not have any previous history of stuttering as confirmed by his parents. As a result of continuing stutter, olanzapine treatment was stopped after fifth day and 100 mg/day quetiapine was given. After 3 days of quetiapine administration his speech gradually became normal.
In the current literature only a few number of stuttering cases due to affects of antipsychotics like chlorpromazine, clozapine, olanzapine, risperidone, trifluoperazine are reported. A case series of stuttering due to Olanzapine usage consisted of six subjects. Olanzapine is also used to treat the symptoms of stuttering by D2 receptor antagonism and in literature there are cases which were treated by administration of olanzapine. It is important that this case shows us despite olanzapine may be used in treatment of stuttering, it may also cause stuttering as an adverse affect.
Keywords: adverse effects, olanzapine, stuttering

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[Abstract:0295][Psychotherapies]

Treatment of a PTSD case with EMDR technique

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Post-traumatic stress disorder (PTSD) is a disorder in which autonomic and cognitive symptoms such as hyperalertness, avoidance and flashback are observed in the patient in response to a traumatic event. Eye Movement Desensitization and Reprocessing (EMDR) is a therapy technique which facilitates the process of data processing and which provides the reprocessing of the traumatic memories that has been stored in fragments. The memories become compatible with life due to this technique. In the neuroimaging studies that takes part in PTSD cases, atrophy at hippocampus, volume and activity increase in amygdala has been detected. Therefore it 's believed that symptoms of intrusive thoughts, flashback, avoidance and hyperalertness occur. However its neurobiological bases have not been disclosed yet, in PTSD cases who was treated with EMDR technique, amygdala blood stream and activity was determined to be lower in imaging studies performed with SPECT.
Case: Our case is 41 year old female patient, single and unemployed. She presented to our clinic with the symptoms of dysfunctionality, avoidance, occurrence of nightmares, flashback, irritability, hypervigillance, and depression. She claimed that she was raped by her boss at her workplace. After the incident, he was scared of all men and worried that any of them could damage her. There fore she stated she could not get outside of her house alone. Duloxetine, mirtazapine, trazodone, quetiapine was used in her treatment with maximum dosage and duration. We decided to use EMDR due to the lack of considerable improvement in her symptoms. We worked through eight stages of EMDR. We tried to find out and work on the resources of the patient that the patient to gain skill in coping with her negative emotions and negative beliefs. The patient's negative and positive cognitions were determined related to the traumatic incident. A significant improvement was observed in her symptoms. The case was updated to "able to go outside on her own" In the studies of treatment with PTSD cases, EMDR proved to be faster and more efficient compared to other psychotherapy techniques. Especially in the cases of avoidance symptoms, as in our case it was observed that adherence to therapy is better and faster.
The more common usage of EMDR in PTSD cases, as a treatment technique which is effective in shorter notice and not hard to implement, will allow patients the chance of faster recovery.
Keywords: avoidance, EMDR, PTSD, therapy

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[Abstract:0296][PTSD]

Difficulties in medical management of anxiety disorder child with Brugada syndrome

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Brugada syndrome has been described in 1992 characterized by coved-type ST segment elevation in leads V1-V3 with successive negative T wave in the right precordial leads with no structural heart disease. Clinic presentations of syndrome are syncope, ventricular tachycardia, ventricular fibrillation, and sudden cardiac death while some patients remain asymptomatic. The major cause of BS is SCN5A gene mutation which encodes for the alfa- subunit of the sodium channel. BS is diagnosed with both clinical findings and characteristic EKG pattern. Implantable cardiac defibrillator is the only treatment for BS, treats ventricular tachycardia and fibrillation and prevents sudden cardiac arrest.
Case: A 9 year-old boy who was diagnosed with BS and presented with stress and anxiety symptoms after a sudden cardiac arrest. We aimed to underline how difficult to treat psychiatric symptoms with BS patients. He aspirated his food while having lunch in class, he collapsed and lost hic consciousness 6 months before apply us. After the examination, Brugada syndrome was diagnosed despite no history of structural heart disease.. He underwent subcutaneous ICD placement and stayed in an intensive care unit about 3 months. Then, he became more angry, refused to sleep, had fear about death and during 3 months he thought that his heart would stop if he falls asleep.

He left school because of anxiety and right hemiparesis. His relationship with friends and parents got worse because of temper tantrums. Patient has followed with psychotherapeutic interview weekly. His wellbeing was provided after 1 year follow up.
Psychiatrists have limited to medical management of psychiatric symptoms in patients with Brugada syndrome. Psychiatrists must be careful that psychotropic drugs blocking sodium-channels that not to increase the risk of syncope and sudden cardiac death. Carbamazepine/ oxcarbazepine, lamotrigine, valproic acid, tricyclic antidepressants, phenothiazines, clozapine and SSRI's- all of which may influence sodium-channel activity. BS has an autosomal dominant inheritance, usually diagnosed at avarage ages of 40 years, prevalence in the pediatric population is very low (0.0098%) compared with adult population (0.14% - 0.7%) and common in man with a ratio of 9:1 in a study. There are a few case reports, series and studies about treatment of psychiatric disorders in BS that rarity of it. Furthermore it is difficult to understand which symptoms are based on BS or anxiety. So more studies must be carried out in this area.
Keywords: anxiety disorder, brugada syndrome, treatment

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[Abstract:0303][Others]

Pediatric misophonia with comorbid obsessive compulsive disorder

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The term misophonia was used first by audiologists Jastreboff to describe a strong dislike of certain sounds and an abnormally strong reaction to them, characteristically anger and even rage. Misophonia or selective sound sensitivity syndrome is defined as responses to specific sounds which cause some emotional and behavioral reactions (i.e., extreme dislike or disgust, anxiety, discomfort, anger and avoidance, aggressive behavior). People suffering from misophonia usually complain about auditory attacks (e.g., chewing,loud breathing, foot tapping), but mostly feel anxiety to repeated visual movements (i.e., misokinesia), such as leg rocking. The sounds which most frequently provoke misophonia are bodily sounds, especially those associated with eating (81%) or breathing (64%). Misophonia that commonly begins in childhood is a chronic condition. Misophonia and obsessive–compulsive disorder (OCD) are frequently reported together. Here, the case suffering from misophonia and OCD which are not improved by serotonergic and antidopaminergic agents will be presented.
Case: An 11 year-old girl presented to child psychiatry department, complain about misophonia symptoms included irritability, distress and avoidance in response to finger tapping, whistle, tempo typically produced by family members especially by her father. Her complaints have begun 2 years ago. Besides misophonia symptoms she was also suffering from OCD symptoms. She was straightening paper and pens on a desktop or books in a bookcase and arranging objects many times until they are symmetrically aligned or matched. According to her parents, the child's development was normal and no history of significant medical illnesses. She was successful at school. There was no familial history of psychiatric disorder except her father had discomfort about eating sounds. The patient was referred to otorhinolaryngology and neurology consultation. Audiometry with tympanometry and neurological examination were normal. She had been prescribed fluoxetine 10 mg/day since 6 month at first admission. However, her complaints got worse and the treatment was ended by doctor. Thus, we prescribed aripiprazole 2.5 mg/day for OCD and misophonia symptoms. She had a slight improvement on the frequency of misophonia symptoms at 5 mg/day aripiprazole. After 4 months because of the continuous complaints regarding misophonia; although sertraline 25 mg/day was administered and the dose was increased to 50 mg/day, her symptoms were not recovered. Patient began psychotherapy one day per week with her parents' request at another center. At last interview her symptoms did not change.
Despite the slight knowledge on etiology and mechanisms in misophonia, many reports describe considerable comorbidity and phenomenological correspondence especially with OCD. Patients suffering from misophonia may also have many obsessive compulsive symptoms, could benefit from serotonergic or antidopaminergic agents which are currently theoretical suggestions. Because of that we prescribed serotonergic and antidopaminergic agents in this case in order to improve misophonia and OCD symptoms.
In this case report, our objective was to emphasize misophonia symptoms which impair people and often missed. Management strategies for misophonia are based on empirical case reports. Further research is essential to investigate the nature of this comorbidity and to determine effective psychotherapeutic and pharmacological treatments for misophonia.
Keywords: misophonia, obsessive compulsive disorder, psychotherapeutic and pharmacological treatment

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[Abstract:0304][ADHD]

Skin rash as a side effect in an ADHD patient treated with OROS-Methylphenidate: a case report

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Attention deficit hyperactivity disorder (ADHD) is a neurodevelopmental disorder characterized by three group of symptoms: inattention, hyperactivity and/or impulsivity. ADHD is one of the most common neurobehavioral disorders of childhood. ADHD has a negative impact on several aspects of life. The etiology of ADHD is multifactorial. Its heritability has been estimated as 76%. Treatment of ADHD includes educational approaches, behavioral therapy and medication. Medication is reported to be useful in most of the patients and accepted as a first line treatment choice. Pharmacologic treatments of ADHD are stimulants (Methylphenidate and Amphetamines) and non-stimulant drugs. These agents are usually well tolerated with mild side effects. The most common side effects of methylphenidate are insomnia, headache, irritability, agitation, nervousness, tremor, loss of appetite, nausea and weight loss. Skin rash has rarely been reported in patients with ADHD treated with Methylphenidate. Here, we present an adolescent patient with ADHD who developed skin rash during OROS-Methylphenidate treatment.

Case: A 14 year-old and 84 kg male patient with ADHD was referred to our outpatient clinic for typical symptoms of ADHD. His developmental milestones were normal. There was no familial history of any psychiatric disorder. According to DSM-IV criteria, he was diagnosed as ADHD Predominantly Inattentive Type with normal intellectual capacity. OROS-Methylphenidate 54 mg/day was started. Three days after, he presented to the emergency department due to pruritic and maculopapular skin rash on his both hands, palms and feet. He was consulted by dermatologist and pediatrician, and these lesions are reported as drug eruptions associated with OROS methylphenidate and IV Methylprednisolone and Feniramin was started. At the same time, he was recommended to stop the medication of OROS Methylphenidate and to use the antihistamine drug for one week. After one week, his skin eruptions fully recovered.

Stimulants including methylphenidate are the most effective medications for the treatment of ADHD. Dermatologic adverse reactions of Methylphenidate are cold extremities, increased sweating, skin eruptions and acne. A few cases of methylphenidate-related skin rash have been reported in the literature. In cases who were treated with Immediate Release Methylphenidate and OROS Methylphenidate, allergic reaction was developed only when OROS Methylphenidate was used. It is not known as to whether this reaction was due to Methylphenidate itself or substance of the OROS Methylphenidate capsules. OROS Methylphenidate capsules contain several substances that is not available in Immediate Release Methylphenidate tablets.

In conclusion, we recommend that users should be monitored closely for possible side effects when using OROS Methylphenidate. If side effects occur such as skin rash, they should be consulted to relevant departments. It should be kept in mind that standard treatment methods such as Antihistamines and steroids are safe and effective in drug eruptions.

Keywords: ADHD, oros-methylphenidate, skin rash

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[Abstract:0305][Schizophrenia and other psychotic disorders]

Acute psychosis during the postictal period in a patient with no previous diagnosis of epilepsy

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Relationship between epilepsy and psychosis is known for long years. It is known that psychotic disorders with epilepsy patient is seemed 7% frequently and risk is remained much more in 6 to 12. Psychotic indications are divided according to season of detection relevant to crisis. These indications could follow crisis like postictal or preictal (related with just crisis). Postictal psychosis (PIP), which has stretched secondarily to complex partial seizures, is a manifestation that compose after 24-72 hours generalized tonic-clonic seizures. In this case, we will discuss conduct of postictal psychosis case followed with short psychotic seizure and occurred after a generalized complex epileptic attack.

Case: A female patient, married with 3 children, elementary school graduate and housewife. Patient was brought to the emergency service with complaints such as waking up screaming at night, losing her consciousness, accuring tonic-clonic myospasms, urinary incontinence, and advent suds in her mouth. Patient was consulted to us because of being postictal confusion and almost 24 hours later inducing persecution and referencial delusion with secondary audio-visual hallucination during observing of patient at the emergency who hadn't epilepsy diagnosis follow up previosuly. According to psychiatric examination of patient: conscious, orientation and co-operation was straight. Amount of speech has decreased, she had poverty of content of speech. Sensation was limited, mood was coherent with content of thought. There were persecutory and reference delusions. There were audio-visual hallucinations. Patient was taken to our service with psychotic attack diagnosis. Patient was consulted to neurology department. Brain MRG and EEG was postulated. Parenteral antipsychotic 10 mg/dayn was got because patient had an agitation and did not adopt oral treatment. Patient was observed unmedicated and parenteral antipsychotic was cut out because of being remission on positive psychological symptoms 72 hours later. Lamotrigine treatment was initiated because of brain MRG and EEG of patient and complying between seizure and epileptic attack. When the previous patient-file was examined; in both of hospitalizations; patient has been followed-up like psychotic attack because of complaints with scepticism, insomnia, irritability, remission has been observed rapidly with antipsychotic treatment on 4 days in first hospitalization and 5 days in second one. In this hospitalization, patient, whose psychotic symptoms got remission in almost 72 hours with antipsychotic treatment after crisis was kept under, has not had any negative and positive psychotic symptoms.
Postictal psychosis, whose prevalence has been determined as 6.4%-10, constitutes 25% in all epileptic psychosis. It is a manifestation proceed between 24 hours and 3 months, in which observing of mood indications or psychotic indications like delusion and hallucination after transitional period in which it consorts with mental fog but not being psychotic indications. Postictal psychosis recruits either spontaneously or regulation of antiepileptics dosages and treatment of lower dose antipsychotic. 10% of cases may turn into chronic psychosis. Postictal psychosis may be seen with patients having epilepsy history proceeding frequently more than 10 years, however, in our case, it is remarkable that there is no previous epilepsy history of the patient.
Keywords: acute psychosis, epilepsy, postictal psychosis

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[Abstract:0309][Autism]

The use of complementary therapies and the role of electroconvulsive therapy in the treatment of adult autism

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Autism spectrum disorder (ASD) is a chronic developmental disorder with symptoms like disturbance in verbal and non-verbal communication and limited, stereotypical patterns of behavior in social interactions. Catatonia can also be observed in autistic patients. Catatonia shown in autistic patients frequently as increased aggression, repeated self-destructive behaviors, echolalia, psychomotor retardation, increased aimless behavior, posturing, mannerism, odd behaviors like stereotyped behavior, refusal to eat, decreased speech or mutism. Since the catatonic symptoms resemble with ASD symptoms, the diagnosis of catatonia, which co-occur with autism in clinical practice is frequently missed. There are instances where electroconvulsive therapy (ECT) is successful in treating catatonia of autistic patients and ECT is suggested for patients responding insufficiently or not at all to medical therapy. There are cases reported where maintenance ECT are useful in avoiding relapses. Even there is no definitive therapy of this disorder; it is known that large part of families with autistic children is tending towards non-medical therapy because of underlying reasons. Besides of high financial loss of families due to non-medical therapy, it can also have negative outcomes for the patients.
Case: An 18 year-old male patient. While our patient was in the past a well-adjusted autistic person he had to be hospitalized because of catatonic symptoms like ritual behavior, negativism, grimacing, stupor, echolalia, repeated inappropriate behavior and depression and compulsion complicating the harmonization by showing up in the last 4 months. His treatment was rearranged as risperidone 2 mg BID, valproic acid 500 mg BID, and quetiapine 100 mg/daily. During his stay at the department the symptoms did not weaken and the patient had to be restraint for once and it was decided that the patient get a bilateral ECT. During the ECT the dose of the valproic acid treatment was reduced and then terminated, whereas the risperidone 2 mg BID and quetiapine 100 mg/daily treatment were carried on. In our case, the severity of the symptoms got weaker a short time after the implementation of ECT.

It is known that complementary and alternative medicine (CAM) strategies do not have scientific proof but they are prevalent and are a high financial burden for families. In our case the use of complementary therapies began also with the directing of the aunt especially and went on via internet with the advices of other family members for different CAM therapies. The patient in this case had gone through a long period with at least four different CAM therapies including diet without gluten and casein, chelation, hyperbaric oxygen therapy and cortexin injections. The family has spent around 20-25 TL during this period for the CAM therapies. In this case the autistic adult's family's use of CAM until today, the emerging catatonic symptoms within the process and the efficiency of ECT and the following maintenance ECT therapy will be discussed.

Keywords: Autism spectrum disorder, Catatonia, Complementary therapies, Electroconvulsive therapy

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[Abstract:0311][Psychopharmacology]

Possible olanzapine -induced hepatotoxicity: case reports

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A variety of medications are associated with mild elevations of hepatic enzymes. The psychotropic agents, especially the first generation antipsychotics, are classified in the high-risk group for the hepatic enzyme elevation. Atypical antipsychotics commonly cause isolated asymptomatic increases in the aminotransferase levels. Here, we represent two cases of acute and severe liver enzyme elevation after starting olanzapine with no previous history of liver disease.

Case 1: A 31 year-old man with known type I Diabetes Mellitus, had normal baseline biochemistry tests including liver function tests. One day after starting olanzapine his ALT and AST levels were 289,6 IU/L and 514 IU/L, LDH was 296 IU/l and INR increased from 1 to 2.5. Olanzapine was discontinued immediately and the patient was consulted with gastroenterology department. Other causes of acute toxic hepatitis were evaluated, but not found. His INR and liver function tests were followed up daily. Over the next twelve days, INR and the liver function tests were gradually decreased and finally returned to normal limits.

Case 2: A 31 year-old male patient, was brought to the emergency department by his parents because he was refusing food intake. His baseline biochemistry tests including liver function tests were within the normal range. Intramuscular olanzapine (1 doses of 10 mg) was administered on the first day. The next day, his ALT and AST were 83IU/L and 202 IU/L and LDH was 549 IU/L. Olanzapine was stopped. INR and liver function tests were followed up daily. Over the next nine days, his liver function tests were gradually decreased and returned to the normal limits. Then, olanzapine was restarted at 10 mg IM, this time liver function tests were mildly increased (AST:33, ALT:61) at second day, after one dose.

Those two cases were diagnosed with probable olanzapine-induced hepatotoxicity. Elevation of the hepatic enzymes more than ten fold in a shorter period than 48 hours may be incompatible with drug induced hepatotoxicity, but the exclusion of other causes of acute hepatitis, the onset of liver injury after starting olanzapine and a rapid decline in liver enzyme levels once olanzapine is stopped suggested possible hepatotoxic drug reaction due to olanzapine. The malnutritious state for both of the patient and the intensive care unit period for the first patient may facilitate the hepatotoxic reaction. In current literature hepatotoxicity induced by atypical antipsychotics has been reported more infrequently than asymptomatic enzyme elevations. Comparing to risperidone and quetiapine, clozapine and olanzapine are less frequently associated with elevation of hepatic enzymes.

Although the slight elevation in hepatic enzymes is expected after olanzapine treatment, the clinicians must be careful about the probable toxic drug reactions. Before atypical antipsychotic treatment, evaluating baseline levels of patients' biochemistry including liver function tests, and monitoring regularly, especially in the high-risk patients, such as malnourished patients, may decrease the risk of unrealized hepatotoxicity.

Keywords: olanzapine, hepatotoxicity, atypical antipsychotic

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[Abstract:0314][Psychopharmacology]

Steroid -induced isolated visual hallucinosis: a case report

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It has been stated in literature for a long time that, psychiatric disorders is one of the possible adverse effects of corticosteroids. Here, a case of isolated visual hallucinosis, which is probably related to the use of corticosteroid is presented.
Case: A 46 year-old male patient was referred for a psychiatric evaluation by rheumatology department. Patient had a diagnosis of rheumatoid arthritis for 6 years. During a follow-up of 6 years, no further psychiatric symptoms were observed, although he is still being treated with methylprednisolone, 2 mg/day PO. For the last one month, he has been treated with methylprednisolone 8 mg/day PO who was admitted because of active rheumatoid arthritis. After one week at this dosage, the patient suffered from visual hallucinations with feelings of discomfort, but he continued his treatment because of he was benefiting from the treatment. After 1 month, he was referred to psychiatry outpatient clinic. The patient reported that he never had a complaint of visual hallucination before. There was no psychiatric illness in his medical history and family history. In psychiatric examination; his self-care was moderate, he was fully conscious, oriented and cooperative, his affect was anxious and sometimes dysphoric. He showed decreased appetite and sleep. Physical and neurological examination was normal and there was no personal history of alcohol-substance abuse or use of drugs for any illness. Routine hematological and biochemical test results were normal. His cranial MR and EEG revealed no pathology. The patient was evaluated by an ophthalmologist. The examination did not reveal any signs that explain the visual hallucinations. Methylprednisolone was gradually discontinued, and methotrexate (10 mg/week) was introduced. The visual hallucinosis gradually disappeared within two weeks upon discontinuation of without adjusting the psychopharmacotherapy. There has been no relapse of visual hallucinations since methylprednisolone was discontinued, despite being on methotrexate for 6 months and the dose of methotrexate was continued at the same dose (10 mg/week). Clinicians should keep in mind that visual hallucinosis may occur with steroid treatment. It is also important that patients should be informed about this.
Keywords: isolated visual hallucinosis, rheumatoid arthritis, steroid

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[Abstract:0316][Schizophrenia and other psychotic disorders]

Late-onset mycophenolate mofetil-related delusional parasitosis: a case report

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Delusional parasitosis is a rare psychiatric disorder which is characterized by a person's persistent misconception of being infested by bugs or parasites despite the absence of any medical evidence. It is classified as primary or secondary delusional parasitosis according to precipitating causes. Mycophenolate mofetil (MMF) is an oral immunosuppressive agent commonly to prevent allograft rejection and to manage select autoimmune diseases. Common adverse events included diarrhea and nausea.
Case: A 64 year-old male patient was brought by the wife to psychiatric outpatient clinic. He had been diagnosed with nephrotic syndrome diagnosis and had received oral prednisone 4 mg/day for 2 years. Two weeks ago, he was diagnosed with active nephrotic syndrome and MMF 1200 mg/day treatment was added on. In the 2nd week of the MMF treatment, he complained worms crawling through his skin, mainly on his face and scalp. MRI and EEG were performed in order to exclude organic pathologies. Electroencephalography did not reveal any other pathologies and MRI was normal. Physical examination and laboratory assessment were in normal range. No pathological finding was detected in the dermatological examination and consultation. In the psychiatric examination; he was conscious, cooperated, oriented to time, person and place. His speech was understandable and fluent. Affect and mood were anxious. He had infestational delusions. He had no hallucinations. There were no psychiatric illness in patient or family history. MMF was discontinued and risperidone

2 mg/day treatment was given. He showed remarkable improvement. His symptoms resolved completely within a week. In his outpatient follow-up visits no psychiatric symptoms were observed during 12 months. That patient had received combination therapy of MMF and prednisone. Thus, it is unclear whether both agents were responsible for the delusional parasitosis or MMF alone. The situation in our case is clearer than in the previous one, because the discontinuation of MMF improved the psychiatric symptoms, in spite of persisting prednisone treatment. Thus, it is possible that MMF alone is responsible for delusional parasitosis. To the best of our knowledge, this is the first report of MMF-related delusional parasitosis successfully treated with risperidone in the existing literature. In conclusion, this report suggests that clinicians should be aware of the possibility that MMF may induce delusional parasitosis in the short-term period. The exact mechanisms by which MMF causes psychiatric side-effects are not known. Further research is required to determine the frequency and clarify the mechanism of this adverse effect.

Keywords: delusional parasitosis, mycophenolate mofetil, risperidone

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[Abstract:0319][Neuroscience: Neuroimaging-Genetic Biomarkers]

Non convulsive status epilepticus with psychiatric symptoms: a case report

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Comorbidity of epilepsy and psychosis are common. Epileptic psychosis is categorized as peri-ictal and interictal psychosis, and peri-ictal psychosis have close temporal relationship with seizures. Peri-ictal psychosis includes the following categories: pre-ictal psychosis, ictal psychosis and postictal psychosis. Ictal psychosis is a rare phenomenon. Epileptic psychosis may exist with the symptoms of audial, visual and somatic hallucinations, affective symptoms, and delirium.

Case: A 41 year-old male patient, was brought in 4 days ago with the symptoms of behavioral changes and perseveration in speech. The patient without previous history of epilepsy, had visited to emergency department after falling home eight days ago. Four days later, he visited to our psychiatry clinic for continued complaints. He suffered from a psychotic episode with delusional thoughts and hallucinations of voices talking about his behavior. He had disorientation and perseverated speech. Self care of the patient was reduced, and he was in a confused state. Although the place and person orientation of the patient was intact, his time orientation was impaired. Mood was depressive, affect was limited. His speech was reduced and did not talk spontaneously. The patient claimed to have been told by Great Creator, that he only had 40 year left to live. He had auditory hallucinations and had no insight. In the neurological examination conducted the following was determined; dysarthric confused speech, cranial nerves, muscle strength and motor system natural. Clonus found in both lower extremities. Incapable of tip toe tests, dysmetria and dysdiadochokinesia on the left hand. Babinski response on the right leg was extensor. Brain MRI revealed that, secondary changes took place after he had fallen at the age of 2, and an encephalomalacic area was formed as a result. His waking EEG showed temporal hyperexcitability with bioelectrical deceleration baseline. Other medical tests were within normal limits. The patient was consulted to neurology department and assessed for the probable diagnosis of ensefalitis. Carbamezapine 400 mg was started by neurology department, and his symptoms disappeared.

Ictal psychosis is usually short term and lasts for few days or hours. Ictal psychosis is a manifestation of simple and complex partial or absence epileptic seizures in the non convulsive status. Most common association is with complex partial status. Absence and complex partial status may demonstrate confusion. Psychiatric findings are similar between seizures for the same patient. One of the most common symptoms is auditory and visual hallucinations. Epileptic psychosis may appear with the symptoms of audial and visual hallucinations, affective, cognitive and behavioral symptoms. These symptoms usually appear with the activation of limbic system. Ictal psychosis is one of the rare reasons of organic etiology. Therefore the aim of this case report is discussing ictal psychosis and non-convulsive status epilepticus based on the literature, and to emphasize the importance of investigating organic etiology in the psychiatric patients.

Keywords: ictal psychosis,status, epilepsy

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[Abstract:0325][Sleep disorders]

Kleine–Levine syndrome: a case report

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Kleine–Levine Syndrome (KLS) is a rather rare syndrome characterized by hyperphagia, hypersexuality, abnormal behaviors, and confusion accompanying periodic hypersomnia episodes. Episodes are recurrent in KLS and patients are normal between episodes. This paper presents the case of a young male patient experienced episodes of excessive sleeping, emotional impairment, confusion, and excessive eating.

Case: A 23 year-old male university student, 164 cm and 105 kg, who lives with his family, complained of excessive sleeping, excessive eating during attacks, irritability, and difficulty concentrating. His first attack was in 2006 and lasted for almost a week. During this attack, he only woke up to fulfill his daily needs. Although he weighed 74 kg in 2006, he reached a weight of 125 kg by 2010. He expressed that he had some improper behaviors during this period, such as excessive eating, and sleeping attacks that lasted for 1–2 weeks. In 2013, he experienced a serious 10-day attack during which he woke up only to use the toilet and could only stay awake for 2 or 3 hours each day. He said that, except for this attack, his attacks generally last around a week and cause him to sleep for 14 to 16 hours each day. In between attacks, he experienced normal alertness and behavior. He said that he has hypersomnia attacks at least 6 or 8 times a year. During these periods, he went to neurology, internal medicine, and psychiatry clinics, but being diagnosed and receiving treatments did not alleviate his symptoms. Physical, neurological, and mental examinations determined that he was normal. Laboratory tests were within normal limits. Routine electroencephalography examination was normal. In addition, a cranial magnetic resonance imaging examination did not show any pathology. Lithium (600 mg/day) and venlafaxine (75 mg/day) were prescribed for the patient as it was thought that he had KLS. It was observed in follow-ups that the patient benefited from the treatment and experienced fewer attacks. His well-being was further improved by increasing the lithium dose to 900 mg/day. KLS is a very rare sleep disorder characterized by relapsing-remitting episodes of hypersomnia, cognitive and behavioral disturbances. KLS most frequently occurs in adolescent men, and the female to male ratio of patients suffering from this syndrome varies from 1/3 to 1/4. KLS's etiopathogenesis has still not been fully elucidated. The syndrome causes patients to experience attacks that generally repeat every three months and last for 10 days. They start suddenly and end spontaneously. In between attacks, normal wakefulness, cognition, and behavior and complete remission are observed. It resembles particularly bipolar disorder and many other neuropsychiatric diseases. Patients are at risk of improper treatment because they are often diagnosed with other diseases or syndromes before it is determined that they suffer from KLS. However, patients' response to proper treatment is usually good. As a result, KLS must be considered when attempting to diagnose hypersomnia attacks accompanied by behavioral disorders in adolescent men.

Keywords: hypersomnia, hyperphagia, kleine–Levine syndrome

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[Abstract:0326][Others]

The Use of EMDR in complicated grief: a case report

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Eye Movement Desensitization and Reprocessing (EMDR) is a psychotherapy method which emphasizes emotional problems that are caused by disturbed memories, such as war stress, harassment, natural disasters or traumatic event in childhood and combines different aspects such as psychodynamic, cognitive, behavioral and client-centered that are used for treatment in clinical situations; to illustrate; phobia, performance anxiety, panic disorder, body dysmorphic disorder, trauma in childhood, grief, chronic pain, rape, harassment. Complicated grief (CG) is defined with functional impairment when patient has severe grief symptoms; and also the duration of grief lasts longer than expected according to social values. We want to get attention in CG and EMDR, an approach to treat CG. Here, patient with

complicated grief who lost his son unexpectedly two years ago and her treatment with EMDR are discussed.
Case: A 42 year-old female patient works as an employee in a private company. Patient presented to our policlinic with the symptoms of unhappiness, depression and qualm that are developed after the death of her 26 year-old son whose death by wounding by firearms. She lost his son 2 years ago. After that day; everything in her life became almost meaningless. Patient stated that, she is thought about her son, cries for hours and disaffirms death by every day. She felt drowsiness just because she had insufficient night sleep. Patient had overreacted petty troubles usually and she had regret for that kind of behaviors. Psychic examination of admission stated that patient had standing and gave away her age, decreased self-care, dolorous mood and gestures, anxious sociability, depressive mood, insufficient of depth of thinking, decreased psychomotor activities and social functionality. Patient was re-experiencing the moments of when she had learnt her son's death, the cause of death and funeral. Declaration of her son's death was chosen as the best representative of traumatic experience. Patient's validity of positive cognition is defined as 3 and subjective units of distress scale of patient were 9. Chest pain was assigned to sense of body. Bilateral stimulating sets were administered on patient. Total of eight EMDR sessions were administered to the patient. After the eighth session, patient's SUD and VOC values were 1 and 7; respectively. No signs of chest pain were observed in the patient's sense of body. Patient has attended her monthly routine controls. Patient had started working a while ago.
While EMDR can be seen as a good option to treat patients with complicated grief; however, limited number of studies about EMDR were published in our Turkey. Complicated grief symptoms were relieved within eight-session EMDR in a patient whom had both functional impairment and resistance to medical treatment, according this case report. Taking into consideration of this method into the clinical practices of clinicians should both increase the ability of the treatment of psychiatric disorders and give the chance to the patient about the relieving concerns of grief and grief-like disorders more rapidly.
Keywords: EMDR, depression, grief

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[Abstract:0327][Mood disorders]

Importance of medical evaluation of psychiatric episodes

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Bipolar Disorder episodes may present with a large variety of clinical manifestations. The current psychiatric episode may mask the underlying medical conditions, even situations which need immediate treatments. In this case report, we aimed to discuss the dramatic clinical improvement of a bipolar depressive episode, after treating the underlying electrolyte imbalance and acute renal failure.
Case: A 71 year-old, male patient, married with 3 children, retired with the diagnosis of bipolar disorder for 40 years. He has been followed by a psychiatrist on private practice for the last ten years. He had symptoms like depressed mood, reduced interest, loss of energy, slowed behavior, excessive sleep and refusal to eat at his first psychiatric examination. He was sluggish and he had abdominal swelling due to constipation he had for several days. His medication was lithium 600 mg, citalopram 20 mg, mianserin 10 mg, memantine 10 mg and levodopa benserazide combination 250 mg daily. Despite the insistence of his relatives for immediate psychiatric admission, his blood tests for blood count, biochemical and endocrinological parameters were performed in the outpatient service. Due to signs of electrolyte imbalance and acute renal failure, he was admitted to the internal medicine inpatient clinic of the hospital promptly. He began to be treated for the dehydration and electrolyte imbalance intravenously. He was consulted to neurology service for the comorbid Alzheimer's Disease and Parkinson's Disease diagnosis reported by his relatives. The relatives stated that these diagnoses were given and the drugs were prescribed by his private practice psychiatrist over the phone call they complaint about his slowed behavior and forgetfulness. Due to lack of clinical signs of these two diseases, his anti-dementia and antiparkinson drugs were discontinued. Lithium bicarbonate and mianserin was discontinued and citalopram deducted to 10 mg daily after the psychiatric assessment he had at the internal medicine inpatient clinic. His general medical condition has improved rapidly and sodium valproate - valproic acid 1000 mg plus olanzapine 2.5 mg daily added to his treatment before the end of the first week of his admission. He began to cooperate and communicate verbally after a few days of psychiatric assessment. His mood was depressive but there was no sign of psychotic symptoms. He was capable to report his vital needs such as hunger and thirst. He was discharged to be followed at the psychiatric outpatient service 10 days after his admission. Psychiatric patients frequently fail to maintain their basic self care skills, particularly when their symptoms are predominant, so that they may not sustain their healthy state. Many of the psychotropic drugs, such as lithium, have serious side effects. Some of those side effects lead to psychiatric symptoms, which may imitate the symptoms of the ongoing psychiatric disorder. Therefore, the side effects should

be monitored closely. Many psychiatric symptoms are common in different psychiatric diagnoses. The distinction should be made by attentive examination to prescribe the appropriate treatment. Psychiatric patients should be examined carefully to uncover the possible side effects and medical comorbidities in order to treat the patient properly.
Keywords: bipolar disorder, psychiatric episode, medical comorbidity, evaluation, side effect

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[Abstract:0328][Others]

Antidepressant use in pregnancy: a case who is taking escitalopram

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Pregnancy is an unsteady period in human life with both hormonal and psychosocial life style changes. When considered that the most of the pregnancies are unplanned, these problems may difficult to overcome by both patient and her health care squad. According to the 122,400 women from two broad studies; 14-20% of that population had major depressive disorders in their pregnancy periods. Termination of the medication in pregnant women is also seem as a problem, relapse risk of patients whom had terminated medication in pregnancy period is 2.6 times higher. Case who had gave up her psychiatric follow-up two years ago, but continued escitalopram at her sole decision in both pregnancy and breast feeding period is presented.
Case: A 32 year-old, housewife and secondary school graduated female patient presented to our emergency service at the evening hours with the symptoms of heart throb, feeling of discomfort and fear of death. Patient was started escitalopram 10 mg in epicenter where she presented with the symptoms of family, social matters and concurrent fear, anxiety and feeling of discomfort. According to her medical history, patient had a 9-month old baby. She was using escitalopram while she learned about her pregnancy. She had not presented to her psychiatric follow up, during her pregnancy period; however, she continued to use drug until the day of admission of emergency polyclinic. Patient had given up breastfeeding a while ago and she decided to stop taking antidepressant gradually. Baby's development was similar to peers. Last examination of the baby is done in well baby clinic in two weeks before her admission to our clinic. No signs of any pathologic findings were observed in that examination. Patient had informed about usage of antidepressant drugs in pregnancy period, therefore, it is also explained that, if antidepressant drugs had to administered to the pregnant women, dose should be at a minimum, corresponding a beneficial effect. Taking antidepressant drugs in pregnancy period should also monitored under physician supervision. Decision of starting, increasing or decreasing doses of the drugs should be decided both patient and clinician together. Patient had been kept under observation for a while and discharged after therapeutic session.
It is clear that none of psychotropic drugs which have to be used in pregnancy period are safe. Therefore, pregnant women whom have severe mental diseases are faced with several severe problems, such as suicide; if they are not treated well. For that reason, clinicians have to choice safe drugs and follow strategies which are included personally identifiable treatment plans and follow ups. Just like the reportation of anomaly cases which arise from the usage of psychotropic drugs, reportation of cases without anomaly are also important for the clinicians in the situation of cases that they have to use antidepressant drugs.
Keywords: pregnancy, escitalopram, anxiety

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[Abstract:0329][Others]

The Use of EMDR in traumatic stress disorders: a case report

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Eye Movement Desensitization and Reprocessing (EMDR) is a psychotherapy method which emphasizes emotional problems that are caused by disturbed memories, such as war stress, harassment, natural disasters or traumatic event in childhood and combines different aspects such as psychodynamic, cognitive, behavioral and client-centered that are used for treatment in clinical situations; to illustrate; phobia, performance anxiety, panic disorder, body dysmorphic disorder, trauma in childhood, grief, chronic pain, rape, harassment Post Traumatic Stress Disorder (PTSD) is a, mental disorder that defined by the combination of autonomic, dysphoric and cognitive findings that causes in people who are facing a severe traumatic stressor, reliving this traumatic event; and also have an avoidant pattern. We want people to pay attention of the efficiency of EMDR in post traumatic stress disorder. Here, an EMDR-treated case who has shown the symptoms of post traumatic stress disorder because of facing a shotgun in 3 months ago is presented.

Case: A 28 year-old male patient is still working in private company. He presented to our outpatient clinician 3 months ago, with the symptoms of fear, anxiety, sleeping problems and nervousness which had started after facing a shotgun. Patient had overreacted petty troubles usually and he decided to keep himself away from the people. Patient had startled every time when he faced with any kind of voice or rumble. 20 mg escitalopram had started to the patient for these complaints in epicenter. Psychic examination of admission stated that patient had standing and gave away her age, decreased self-care and intimate sociability, anxious mood, focused on the event which he had suffered in his thought content, excitation-mobility, decreased psychomotor activities and insufficient social functioning. Hamilton depression and anxiety scores were 18 and 35, respectively. Evaluation of the sources and information of the patient had done in the first session. Best representative image of traumatic experience is selected. VOC and SUD values were 3 and 9, respectively. Heart-throb is assigned to sense of body. Total of three EMDR sessions were administered to the patient. After the third session, patient's SUD and VOC values were 1 and 7; respectively. No signs of heart-throb were observed in the patient's sense of body. Patient attends his follow-up examinations. General well-being of patient continues.

It is proved that EMDR is an effective treatment method for PTSD. EMDR is seen as a good method, because it is easily to known about its procedure and to get results in a short span of time. Taking into consideration of this method into the clinical practices of clinicians should both increase the ability of the treatment of psychiatric disorders and give the chance to the patient about the relieving concerns of post traumatic stress disorders. It is also reported that EMDR has the ability of more rapid improvement than other treatment methods and also it requires less treatment sessions and it has equally efficient with trauma-focused cognitive behavioral treatment.

Keywords: EMDR, stress, trauma

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[Abstract:0330][Forensic psychiatry]

Judicial report in a sexual abuse case live covered on periscope

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Sexual abuse of children -a child under the age of consent- is defined as to be a part in an action would lead to sexual satisfaction of a sexually mature adults or as the condoning of this situation.

Case: A 14 years 2 months-old male adolescent, first of two brothers. He was brought to Sakarya University Department of Child Psychiatry in 2015 by police officers to check the legal meaning and consequences of their behavior and determine whether the development of routing capabilities are existing; and reporting of these results. Case was attending 9th class of the school and school success was normal. He reported that he has sexually intercoursed with his 9 year-old little brother for three times and there was two cousins of him in the last time they had this intercourse while live streaming on Periscope. Case is very sorry for what they did, no longer he can sleep regular after the emergence of situation, he is very angry at home and he said he constantly shouted at home. The mature

reflection in the judicial process rooted from a porn website published the periscope broadcast and continued by Interpol in the United States reported Turkish Officials. Child crime prevention and intervention is not a situation which can only be solved by the security forces. More recently, although especially the increase in crimes against children is clear; also there is an increase in crimes committed via social media. Photographs of naked adolescents, their intercourse and sharing of these photographs on social media is getting frequent, also heavy accusations towards each other by opening fake accounts is wide spread. The majority of children drifting into crime has been determined that male children and adolescents. It is stated in our report that male children and adolescents show more aggressive behavior and driven to crime frequent.

Keywords: social media, social networks, child abuse, adolescent

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[Abstract:0331][Others]

Neurodegeneration after chronic exposure of toluene: a casewith psychiatric signs

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It is known that both toluene (metil benzene) and n-hexane which have replaced benzene in the content of volatile organic compounds because of benzene's toxic effects on both liver and bone marrow have neurotoxic effects. As a result of the recent advancements of petrochemistry and related-industries, abuse of volatile organic compounds is an increasing public health problem all around the world, especially in young adults. Volatile organic compounds which are used in adhesives, thinners, diluents, aerosols, grease and stain extractors, air and hair sprays, deodorants and cosmetic industry have broad- spectrum chemical. In this study, neurological changes and psychiatric findings of a patient who is exposed of toluene chronically.

Case: A 21 year-old male patient, secondary school graduate, started to smoking at the age of ten, also estrayed frequently. In that time, he started to use "bally- a kind of glue" within the influence of his friends. He used that substance hardly a day goes. He preferred bonding material because of easily accessible. Psychiatric findings such as indefiniteness of his sociability, disorganized and agitated behaviors, unfavorableness of affect was were aroused 2-3 years later. Soliloquy and physical aggression were seen in periods which patient did not use the substance; however, at the initial term, these findings were occurred in the time when patient did use the substance. Patient is hospitalized in intensive care unit for 28 days, two months ago before the admission of our clinic, with the reason of the deterioration of his general medical condition, which is due to use the substance-bally intensively. He referred in our clinic with the symptoms of unfavorableness of affect, alogia, decreased functionability, amnesia and undifferantiation of his mood. He continued to use substance before the day for the admission of our clinic. Mental examination showed that mediocre self-care, unlimited mood, unfavorableness of affect, and also alogia, decreased functionability, amnesia and undifferentiation of his mood were also observed. Patients psychomotor activities and sociabilities were decreased. Cranial magnetic resonance imaging showed that chronic encephalomalasic areas in both cerebellar hemisphere, cerebral hemispheres, significantly in parieto-occipital lobe area, basal ganglia on the right side, cortex and basal matter on thalamus; and also glial changes in circumference parenchyma. These findings were made think us vasculopathy due to chronic intoxication in patients with substance use disorders. Patient had diagnosed cognitive disorder+ psychotic disorder due to chronic toluene use. He received 4 mg risperidone and 20 mg paroxetine. Decrease of disorganized behaviors is observed on the second week of his hospitalization. Cognitive disorder is continued. Patient had discharge at the fourth week of his hospitalization with 3 mg risperidone and 20 mg paroxetine treatment.

In the literature, both human case studies and detailed animal studies which are evaluated in metabolic and neurotoxic effects on both acute and chronic toluene use are found. Cerebral and cerebellar atrophy may observed in patients with chronic substance use. Further studies are needed to find any affinity to limbic system in abuse.

Keywords: toluene, neurodegeneration, psychosis

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[Abstract:0332][Psychopharmacology]

Paliperidone -induced edema on feet

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Paliperidone, an active metabolite of risperidone is an atypical antipsychotic that is commonly used for the treatment of schizophrenia and schizoaffective disorder. Here we report a case of a 31 year-old female patient, who developed edema on both of the feet following two weeks of paliperidone administration.

Case: A 31 year-old female patient presented to our outpatient clinic with symptoms of auditory hallucinations, delusions of persecution, insomnia, and angry outbursts. She had received risperidone 1 mg/day, and valproic acid and sodium valproate 1000 mg/day for several years. She had discontinued her medication 1.5 years ago, because of the edema on her both feet. In her examination, self-care was impaired, the amount of speech was mildly increased, her mood was dysphoric. She had some auditory hallucinations such as hearing voices talking about her. She reported persecutory delusions about being watched or being recorded with camera by others to be transmitted through internet while she is naked. She was initiated 3 mg/day of paliperidone and the dosage was gradually increased to 6 mg/day over 2 weeks. At the end of 2-weeks of treatment, while her psychotic symptoms decreased, a 2+ pitting edema developed dorsally in both of her feet. No laboratory abnormality was detected to explain her later occurred edema. Also, a doppler ultrasound of both lower limbs were considered as normal. There were no local skin changes or itching. We suggested that her edema might be due to paliperidone, as she previously reported a similar side effect possibly due to risperidone. Paliperidone dose was gradually discontinued while aripiprazole 5 mg/day were instituted. The feet edema was resolved following the changes in her treatment.

There are reports of peripheral edema associated with antipsychotic use and the mechanism underlying the development of peripheral edema due to antipsychotic treatment is not known exactly. Chen et al., reported a case of peripheral edema in a patient being treated with paliperidone. They have proposed the following hypotheses for edema; a peripheral vasodilation and decreased venous return possibly mediated by antipsychotic antagonism to alpha and 5-HT2 receptors, an indirect increase in water retention via activating the hormone system such as renin and aldosterone, and allergic response to antipsychotic treatment. Antipsychotic-related vasodilation seemed to be more plausible. In a recent cross-sectional study has shown a significant change in vascular compliance in patients treated with risperidone or quetiapine. In this case report we thought that feet edema might be due to the use of paliperidone treatment. We recommend that physicians should be aware of this adverse effect of paliperidone.

Keywords: edema, feet, paliperidone

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[Abstract:0334][Forensic psychiatry]

Incestous relationship between sisters and brothers: a case report

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Incest is estimated to comprise 20-25% of sexual abuse cases and is defined as abuse of children for sexual gratification within the family. In previous studies of incest, most incidents have been reported between father and daughter (39%), followed by brother and sister (15%). Most cases of incest remain hidden when there is no tangible evidence such as a witness or a subsequent pregnancy or become more chronic until the child reaches a mental level where they can understand the wrongness of the event.

Case: A 16 year-old female adolescent was referred by the Public Prosecutor to Erciyes University School of Medicine Health Board related to the crime of sexual abuse and assault. She was student in high school and living with her four brothers and parents. During the examination, she stated that she had interfemoral intercourse with two of her brothers by force and the other two of her brothers attempted to rape. The abuse to the little sister started after her older sister, who had been sexually abused by her brothers, married and left home. The girls told their mother about what happened but the mother did not believe and ignored. When the girl tried to withstand to her brothers, she was exposed to violence and threatened. The abuse had been going on until she escaped from home, a month ago,

after another brother attempted to rape. She came back home, because her older sister called her to threaten and to prevent her from reporting to the police. Though, after coming back, she told her teachers about the incident. After all, she was removed from the family and given into the care of the Social Services and Child Protection Institute. The psychological examination determined her to have impaired perception, feeling of guilt and ''Major Depression'' following trauma.
Here, we present a case of incest, which both of sisters were abused and continued to the recently with the contribution of concealing the incident. Our aim was to draw attention to destructive psychological aspect of incest, which is encountered more in children can be seen in families of social level in all societies and to increase the awareness and sensitivity of family members, community workers and health care personal to be able to make a diagnosis.
Keywords: incest, sisters and brothers, abuse

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[Abstract:0336][Stress and related conditions]

Misophonia: a case report

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Misophonia, hatred of sounds, firstly coined by the US scientist Pawel Jastreboff and Margaret Jastreboff. It's a chronic disorder causes intense emotional reaction and autonomic stimulation of person which does not fall into DSM-5 and ICD-10. A misophonia case, consistent with the characteristic specifications defined in literature, will be discussed here.
Case: A female case, 16 year-old, sophomore student in high school, lives with her family. She presented to our clinic with the complaining of discomfort feeling from certain sounds which has started when she was 12 years old. She tells; when she hears the swallow and smack sound, clock ticking, yawning, eating chips and chewing gum sounds etc., she feels deep distress and anger and warn the people. She leaves there or wears earphone if they don't stop making the sound. She stated that she intentionally hears those sounds and she has been sleeping with wearing earphones since her complains started. She feels more distress of the sounds made by her contacts that's why she can't eat with her family for a long time. She hasn't had a psychiatric visit and she doesn't have any known medical illness. Obsessive compulsive characteristic has been detected at the mental evaluation. Her complaints severely affect her daily functionality. Evaluating the studies on misphonia, it seems that patient's symptoms, characteristics of patients the mechanism to cope with the illness is remarkably resemble with each other. With this regard, clinic is defined, symptom classification has been discussed and diagnose criteria has been proposed. This case`s clinic characteristics meet with all the diagnose criteria. Sufficient evidence level has not met in order misophonia take place into the diagnose systems; further studies are needed.
Keywords: adolescent, child, misophonia

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[Abstract:0337][PTSD]

An EMDR case report

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Eye movement desensitization and reprocessing (EMDR) is a form of exposure therapy that entails assessment and preparation, imaginal flooding and cognitive restructuring in the treatment of individuals with traumatic memories. The treatment involves the use of rapid, rhythmic eye movements and other bilateral stimulation to treat clients who have experienced traumatic stress. Developed by Francine Shapiro (2001), this therapeutic procedure draws from a wide range of behavioral interventions. Designed to assist clients in dealing

with posttraumatic stress disorders, EMDR has been administered to a variety of population including children, couples, sexual abuse victims and individuals dealing with anxiety, panic, depression, grief, addictions and phobias. The treatment consists of three basic phases involving assessment, preparation, imaginal flooding and cognitive restructuring. The procedure involves using back-and-forth eye movements combined with recall of an image, focus on arelated cognition, and awareness of physical sensations.

Case: A 20 year-old female patient, unmarried, nursing student, lives with her family in Istanbul. She was sexually abused by her uncle when she was six years old. Not only she did not blame her uncle, but also she felt sorry for him because her father had sexual intercourse with this brother's wife; besides, uncle's son was died. She blamed her father of all the bad things that happened. The patient has been on a regimen of mirtazapine 30 mg/day and sertraline 50 mg/day for posttraumatic stress disorder since one year. She used amitriptyline 2 years ago. However, she suffers from headache, aggression, insomnia, hypervigilance, problems with concentration, exaggerated startle response and pseudohallucinations. In psychiatric examination, depressed mood, insomnia, dissociative symptoms such as depersonalization and derealization were recorded. This patient had received 3 months therapy without making a dent in her trauma response. After 30 minutes of EMDR, this patient could thought about the events calmly. She showed new insight about her own behavior and no longer was upset with herself. We continued with processing all targets associated with current symptoms until all the necessary targets have been reprocessed. EMDR facilitated the processing of memories and promoted more adaptive cognitions regarding the trauma. She still dislike the father but she was not overwhelmed with rage when talking about him. Yet after 20 minutes of brief conversation and 10 minutes of eye movement totally, her response to this trauma was, to all appearances, resolved. Intrusive aspects of PTSD were potentially quickly and effectively resoluble. EMDR uses a structured eight-phase approach and addresses the past, present and future aspects of the dysfunctional stored memory. In a review of controlled studies of EMDR in the treatment of trauma, Shapiro reports that EMDR clearly outperforms no treatment and achieves similar or superior results as other methods of treating trauma. Increasing numbers of practitioners will receive training in EMDR; outcome research will shed light on EMDR's effectiveness compared to other current therapies for trauma; and further research and practice will provide a sense of its effectiveness with disorders besides posttraumatic stress disorders.

Keywords: EMDR, PTSD, eye movement desensitization and reprocessing, sexual trauma

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[Abstract:0340][PTSD]

Eye movement desensitization and reprocessing: a case report

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Eye movement desensitization and reprocessing (EMDR) is an evidence-based psychotherapy for posttraumatic stress disorder (PTSD). This technique developed by Shapiro (1996, 2001). This structured treatment approach was designed to alleviate distress related to traumatic memories and problematic cognitions related to trauma. In addition, successful outcomes are well-documented in literature for EMDR treatment of chronic pain, somatoform disorders, conversion, dissociation, OCD, panic disorder, phobia, oncology, migraine, addiction, sexual dysfunction disorders (vaginismus) dermatological diseases, personality disorders, pathological gambling and eating disorders. The model on which EMDR is based, Adaptive Information Processing (AIP), posits that much of Psychopathology is due to the maladaptive encoding of and/or incomplete processing of traumatic or disturbing adverse life experiences. This impaires client's ability to integrates those experiences in an adaptive manner.

Case: A 42 year-old female, patient divorced, model, lives with her partner in Istanbul. She was sexually abused by her ex-husband. She informed that her ex-husband used to enforce her to have sexual intercourse with him in front of his friends while they were watching them. After this trauma, she suffered from angry outbursts (with little or no provocation), irritability, dissociative reactions (flashbacks), problems with concentration, sleep disturbance and depressive symptoms such as feeling guilty. She expressed that she had problems in trusting men. She blamed herself. She felt envy to happy women who have healthy relationships. In psychiatric examination, irritable mood, insomnia, low self esteem, pessimistic thoughts were recorded. She declared recurrent, involuntary and intrusive distressing memories of the traumatic events. Also, we identified efforts to avoid distressing memories, thoughts, or feelings closely associated with the traumatic events. Posttraumatic stress disorder was diagnosed based on signs, symptoms and a thorough psychological evaluation. The patient did not want to get or to lose weight because of her job so we followed the patient without medication. We presented the therapeutic rationale for EMDR, explained the mechanics of EMDR and explored various forms of bilateral stimulation. We measured Validity of Cognition (VoC) 7, Subjective Units of Disturbance (SUD) 10, identified the location of body sensations. We continued with

EMDR treatment to complete any unresolved targets.
Results: After 5 sessions of EMDR, desensitization occurred as a byproduct of reprocessing. The distress associated with the memory decreased and new adaptive cognitions are installed regarding the experience. EMDR treatment produced a significant decline in her symptoms and this decline appeared to continue after a four month follow-up.
EMDR has received increasing attention from both clinicians and researchers. As an intervention, EMDR has demonstrated effectiveness in treating PTSD in numerous well-controlled trials and in comparison with other types of psychotherapy, including trauma-focused CBT. The eight-phase, three-pronged process of EMDR facilitates the resumption of normal information processing and integration. This treatment approach, which targets past experience, current triggers and future potential challenges, results in the alleviation of presenting symptoms, a decrease or elimination of distress from the disturbing memory, improved view of the self, relief from bodily disturbance, and resolution of present and future anticipated triggers.
Keywords: eye movement desensitization and reprocessing, PTSD, sexual trauma

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[Abstract:0344][Psychopharmacology]

Successful treatment of neuropathic pain with pregabaline plus exercise In an elderly patient

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Neuropathic pain occurs due to nervous system lesions, diseases, injury or such drugs. It could be seen as hyperesthesia, paresthesia, dysesthesia, hyperalgesia, and allodynia (1). Especially in elderly people, it affects commonly functional status and is accepted as a precursor factor for geriatric pain syndrome. Pregabaline, a gamma aminobutyric acid (GABA) analog, is an anticonvulsant drug and indicated for epilepsy, neuropathic pain, anxiety, and sleep disorders. Here, we present a case of neuropathic pain treated successfully with pregabaline plus exercise.
Case: A 65 year-old male patient presented to our geriatrics outpatient clinic with complaints of a one month history of hand and feet numbness, weakness of both upper and lower extremities, and generalized pain. His complaints affecting severely activities of daily living had been developed after radiotherapy and chemotherapy for tongue cancer being completed. He gave no history of a comorbid disease. His psychical and neurological examinations were normal. Laboratory data out of creatinine (serum creatinine: 2.32 mg/dL, glomerular filtration rate: 27.4 ml/min) did not show abnormalities. He was taking B1 vitamin 250 mg/day, B6 vitamin 250 mg/day, B12 vitamin 1 mg/day, magnesium 365 mg/day, and gabapentine 300 mg/day adjusted for renal failure. After this treatment regime, his symptoms continued exactly. He was consulted with a neurologist for the revision of drug treatment. His electroneuromyography revealed sensoriomotor axonal polyneuropathy (sensorial dominant). Gabapentine was stopped and pregabaline was started with renal dosage adjustment (150 mg/day). In addition to the medical therapy, an exercise program 3 day per week and 45 minute per day was performed (10 minutes warming, 25 minute flexibility and strengthening, and 10 minute cooling down). The response to treatment was assessed after 12 weeks. Following to the treatment, parameters indicating pain, muscle strength, walking and balance, cognition, and basic activities of daily living were improved. In addition to drug treatment, neuropathic pain could be managed with several physical techniques and rehabilitation modalities. These are low frequency TENS (Transcutaneous Electrical Nerve Stimulation), hot and cold administration, whirlpool, medical massages, and exercises. Kuphal et al. showed that exercises programs reduced neuropathic pain in animal models. Although data from human studies indicating the effectiveness of exercise programs on neuropathic pain are conflicting, it has demonstrated that addition of exercises to drug treatment for neuropathic pain may be beneficial.
To the best of our knowledge, this is the first report of the combination use of pregabaline plus exercise for the treatment of neuropathic pain caused by anticancer therapy in an elderly patient. We think that such a therapeutic modality may be a good option especially for elderly patient with poor functional status developed after neuropathic pain. Additionally, pregabaline could be an attractive agent to use in elderly patient with neuropathic pain because of their specific strong binding effect on calcium channels.
Keywords: neuropathic pain, pregabaline, exercise, elderly

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[Abstract:0346][Psychosomatic medicine-Liaison psychiatry]

Malingering or munchausen syndrome?

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Malingering and Munchausen syndrome are a subset of factitious disorders, which are hard to diagnose. Munchausen by Proxy is defined as "deliberate production or feigning of physical or psychological signs or symptoms in another person who is under the individual's care". The motivation for the perpetrator's behavior is considered to be the psychological need to assume the sick role by Proxy. Malingering, on the other hand, involves the intentional production or exaggeration of symptoms (illness) for obtaining compensation or avoidance of duty/punishment and specifically requires the presence of potential for secondary gain. This report is a about a mother who alters his daughter's lab results to benefit from a salary due to chronic kidney failure.
Case: A 16 year-old girl was applied to ER by her mother with a story of kidney failure. Laboratory results confirmed the deterioration in kidney functions, so the patient was referred to pediatric nef nephrology for dialysis. But after admission to pediatry clinic, an ECG (K: 13 but no pathology) and a blood gas analysis was performed and both were seen to be in normal ranges (K:4.1 BUN:14 Kre:0.66). Dialysis was delayed but the patient remained under supervision. High levels especially on morning blood tests and her mother's persistence of staying with her while giving blood was found to be suspicious. Thus lab tests were repeated. This time every value was in normal range. First we thought Munchausen by Proxy and made an interview with patient. It was revealed that she is from a tribe of Van, left high school from first grade, second out of five siblings and had health problems for last 3-4 years. Her mental and physical development was concordant with her age. She was also coping too well with her supposed chronic kidney failure. Further investigation revealed that mother had a salary before for many years due to her diseases and also family had very low income. In addition, daughter and mother were visiting hospitals city by city, leaving other siblings in Van. At last mother confessed that she switched the blood samples so the final diagnosis became malingering. Detection of malingering is not always this easy. Malingering has always a purpose, and when that goal is reached symptoms usually fades out. In this case there were many clues that ease detection: almost suspicious daughter-mother cooperation, repeated blood tests, extreme well coping of the teen patient and salary history of mother. There are many case reports of malingering like this among kids and teens. Most of these patients have borderline personality disorders. Being skeptical is the first and foremost step of detection of malingering.
Keywords: child, malingering, munchausen

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[Abstract:0347][Psychopharmacology]

Increased frequency of encopresis in a child diagnosed with attention deficit hyperactivity disorder (adhd) and encopresis after atomoxetine use: a case report

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Attention deficit hyperactivity disorder (ADHD) is among the most frequently reported coexisting psychiatric conditions in children with encopresis. Some case reports state that both OROS methylphenidate and atomoxetine, approved for treatment of ADHD are also effective in the treatment of co-existing encopresis. Contrasting those reports, here, we report a complex case of an 11 year old boy with ADHD and primary encopresis (without constipation and overflow) whose encopretic symptoms increased after initiation of stimulant treatment along with risperidone. Increased dose of stimulants and cessation of risperidone did not help in reducing encopretic symptoms and the patient also developed de novo enuresis. Initiation of atomoxetine along with aripiprazole helped to control ADHD and enuresis while encopresis increased in a dose dependent fashion. Tapering the dose of atomoxetine and continuing treatment with aripiprazole helped control all of the patients symptoms. This observation was unexpected due to frequent reports of beneficial effects of agents used to control ADHD (both stimulants and atomoxetine) on encopresis concomitant with ADHD. Authors suggested that the anti encopretic effects of atomoxetine or methlyphenidate may be related to direct impact on executive functioning, self-organizing skills and impulse control which

enabled children to recognize and respond to internal cues to defecate and also posited that the effects of ADHD drugs on encopresis may be secondary to the resolution of child-parent relationship conflicts and poor social and academic functioning. But in some other reports, it was suggested that the anti-encopretic effects of stimulants may be related to their direct peripheral effects on GIS motility rather than effects on executive functioning, self-organizing skills or impulse control and also atomoxetine may help encopresis via increased noradrenaline levels and therefore sympathetic activity in gastrointestinal system. The regulation of bowel movements involves complex physiological processes, including long- and short-reflexes, peptides and neurotransmitters. The complex and variable presentation of encopresis symptoms in our patient along with dissociation of treatment response of ADHD symptoms and those of encopresis argue against a relationship between bowel control and executive functions. Risperidone which along with OROS methylphenidate increased encopresis in our patient binds to D2, 5HT2A, alpha 1 and 2 and H1 receptors. D2 is also known to be involved in gastric and intestinal motility and MPH increases dopamine (DA) levels in gastro intestinal system (GIS). Therefore, we posit that D2 blockage superimposed on a chronically low tonic and high phasic tonus of the autonomic nervous system (ANS) may have increased encopresis in our patient. The cessation of risperidone as well as OROS methylphenidate may have changed the D2 occupancy, however, addition of aripiprazole may have stabilized DA functioning in the GIS at a low level, while elevation of norepinephrine (NE) via atomoxetine may have affected tonic and phasic activity of the ANS; increasing encopresis further. Reduction of atomoxetine dosage while continuing aripiprazole may have normalized the ANS activity without changing D2 occupancy, which may have helped reduce encopresis.

Keywords: atomoxetine, encopresis, ADHD

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[Abstract:0348][Psychopharmacology]

Alprazolam -induced disinhibition in a 15 year-old girl: a case report

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Anxiety is a state of being bored, worried or distressed everyone might experience during their lives. It can happen with or without a reason. It might have a positive influence on performance up to a certain level by increasing motivation. However once it passes a certain threshold and starts affecting the patient in a negative way reducing the quality of life and even harming the patient, it becomes a pathological problem.

Case: A 15 year-old female adolescent, attending to the 10th grade. She presented to the Emergency service after a suicide attempt. According to her family, she had anxiety for about two weeks, especially about her academic achievement, reduced appetite, difficulty in sleeping and social withdrawal. She was a successful student and even her worst grades were above average. With these complaints, she presented to a psychiatrist and prescribed a treatment of daily 20 mg of fluoxetine and 0.5 mg of risperidone. As no improvement in her symptoms was seen and she started not to be able to write even a word in her exams, three daily doses of 0.5 mg alprazolam was added to her treatment. 5 days after the addition of alprazolam, the patient started to feel so angry, unrestful, worried and stressful that she attempted to suicide by jumping off the balcony. During examination, the patient seemed to be dysphoric, agitated and irritable. She could not stay still and was pacing in the room. She was also complaining about not being able to breath and hold her attention. She was constantly repeating her feelings of despair, but did not have suicidal ideas or psychotic symptoms. She was suffering from excessive sweating and minor tremors in her hands. The first diagnosis for the patient was disinhibition and alprazolam was stopped. She continued taking fluoxetine and risperidone. The patient's symptoms decreased after the removal of alprazolam.

Alprazolam is a triazolo benzodiazepine derivative and is primarily used as an anxiolytic. It is also known as effective in the treatment of Agoraphobia, Panic Attack and Panic related disorders. It is absorbed rapidly in the gastrointestinal tract and reaches its maximum blood concentration in 1.5 hours (90 minutes). Its elimination half-life is about 11 hours which is shorter in younger patients. The effects of all benzodiazepines are similar and they all bind to gamma-aminobutyric acid (GABA)/ benzodiazepine (BZ) complex receptors and create an effect of sedation. They primarily reduce anxiety and remove physical symptoms like muscle tension. Alprazolam is a mild BZ derivative. Its adverse effects include dizziness, confusion, sedation, psychomotor impairement, hypertension, tachycardia, experiencing nightmares and rarely, paradoxical aggression. The mechanism that causes the paradoxical anxiety of benzodiazepines which are used for the treatment of anxiety is not clear. This disinhibition is thought to be related with the fact that GABA receptors also have excitatory effects in children and teenagers. Due to this disinhibition effect, starting a treatment that includes GABA receptors in that age group should be well considered.

Keywords: adolescent, alprazolam, disinhibition

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[Abstract:0350][Psychopharmacology]

Reduction in the prolactin level of a patient with the addition of aripiprazole to her treatment: a case report

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Prolactin is poly-peptide hormone that is secreted by the lacto-trophic cells of the Anterior Pituitary Gland with a circadian rhythm. Dopamine reduces the prolactin secretion by binding to the D2 receptors present on the lacto-trophic cells. The inhibition of the dopaminergic messages by the anti-psychotics increases the prolactin level. All the typical anti-psychotics and some atypical agents effectively cause DA2 inhibition and increase in the prolactin levels. Here, we describe a schizophrenic female patient suffering from amenorrhea due to hyperprolactinemia caused by risperidone, the prolactin levels of whom reduced to normal levels with the addition of aripiprazole, a partial dopamine antagonist.

Case: A 20 year-old female patient, presented to our clinic with paranoid delusions, deterioration in self-care and auditory and visual hallucinations. At the age of 15, she was diagnosed as schizophrenia and have used olanzapine, quetiapine, amisulpride and haloperidol until this time. The patient presented to our clinic and a daily 6 mg dose of risperidone was started. After her discharge, improvement in her psychotic symptoms was observed but she started to experience amenorrhea. Her prolactin level was 112.4 ng/ml so risperidone was reduced to 3 mg/day, 10 mg pimozide was added and her prolactin level fell to 72.15 ng/ml. However, extension on the QT diagnosed on ECG, so the pimozide treatment was stopped, risperidone was reduced to 1.5 mg/day and 30 mg aripiprazole was added. On the 15th day of the combination treatment, the patient was re-admitted to the clinic due to an increase in her psychotic symptoms, her prolactin level was 43.65 ng/ml then. In addition to 30 mg aripiprazole, risperidone was increased to 6mg/day. With new treatment, significant clinical improvement was seen with a prolactin level of 14.35 ng/ml one month later.

Risperidone, a commonly used drug in the treatment of schizophrenia, may cause hyperprolactinemia. Risperidone acts as a serotonin and dopamine antagonist based on the dosage. Compared to other atypical anti-psychotics, it binds to 5-HT2 and D2 receptors with a greater affinity, and this is thought to be the cause of increase in prolactin levels. Aripiprazole is the first potent D2 partial agonist among the new generation anti-psychotics. As it acts as an antagonist in a hyperdopaminergic state and acts as an agonist in a hypo-dopaminergic state, it is considered as a dopamine regulator. While it is a partial agonist for the 5-HT1 receptors, it is also an antagonist for the 5-HT2 receptors. In one study it was reported that aripiprazole causes hyperprolactinemia less than 5%, while in an other study it was said to have been tolerated well and reduced the prolactin levels much better compared to placebo. Similarly, in our case, we noticed a reduction in the prolactin level of the patient with the addition of aripiprazole to her treatment.

Aripiprazole can be used to reduce the effects of the symptomatic hyperprolactinemia while having an antipsychotic influence on the psychotic patients.

Keywords: aripiprazole, prolactin, risperidone

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[Abstract:0351][Schizophrenia and other psychotic disorders]

Psychotic disorder caused by hyperthyroidism: a case report

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Thyroid function disorder can be commonly observed in patients who complain from psychiatric symptoms. Thyroid function disorders can lead to mental symptoms and those symptoms can vary from delirium to manic hyperexcitability. In literature, psychosis associated with hyperthyroidism is typically presents with affective symptoms. Schizophrenia-like psychosis cases are seen rarely. In this case report, we will be presenting a case of hyperthyroidism phenomenon with schizophrenia-like psychotic symptoms.

Case: A 37 year-old female patient, brought by her family, family complained about her violent behavior, disorganizated speech, and

disorganizated behavior. The patient who has no prior psychiatric applications and treatment stories began her first complaints 5 weeks ago. Early complaints were avolution, loss of appetite, crying and misery where later on, 3 weeks before the application, constipation, palpitation, fever and headache were also added on those complaints. Those symptoms were soon followed by, fear, insomnia, unreasonable attacks to self and other people and meaningless mumbling. The patient presented to the outer center psychiatry and was prescribed 20 mg per day paroxetine treatment, only to have her complaints increase, after the treatment. Her movements and talking became faster. After the patient's reapplication to psychiatry, she was treated with 800 mg per day quetiapine and 15 mg per day olanzapine. After this medications, her violent behavior and disorganizated speech was decrease. But she complained about another symptoms and sedation therefore applied our psychiatric polyclinic, and then she was hospitalized for the purpose of diagnosis and treatment. In psychiatric examination; she was alert and orientated place, time and person. Self care was reduced, at the examination she was unclean groomed and dressed. Her movements was slowly. Her mood was irritable, affect was monotonous and her range of mood reduced. In thought stream, was observed loose associations and blocking at few times. In thought content was determined poorness, homocidal thinking, some delusional schema. She has not insight about her condition. She exhibits normal perception BPRS was scored 63. In thyroid tests; TSH (<0.006) was suppressed, free T3 (10) and free T4 (3.06) was increased. Nodules, which detected in throid USG and scintigraphy was consulted endocrinology. Methimazole and propranolol was prescribed with thyrotoxicosis diagnosis. During clinic follow-up, paliperidone was started, and was increased to 6 mg per day. During her hospitalization, in thyroid function tests; TSH continued suppressed value, free T4 was declined 3.06 to 2.01. She benefitted from treatment. BPRS was scored during follow-up and discharging, respectively 39 and 13. She was discharged when her symptom and complaint reduced. She was reconsulted endocrinology before discharge. In monthly follow, after discharge, psychotic symptoms was disappeared completely in few months. Thyroid values was in normal range.
In literature, psychosis cases related to hyperthyroidism is popular. Especially in schizophrenia-like psychosis cases, one should always remember to include a possible thyroid function disorder. Literature states that, patients in similar conditions, who became euthyroid, report declines in psychiatric symptoms. Similar process happened in our case. For early diagnosis and treatment in patients like this, thyroid function tests hold significant importance.
Keywords: hyperthyroidism, psychosis, treatment

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[Abstract:0352][ADHD]

OROS metylphenidate-induced visual and tactile hallucinations

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Metylphenidate is a stimulant which is mainly used in attention deficit hyperactivity disorder (ADHD) treatment. Psychiatric symptoms like euphoria, delirium, confusion, psychosis and hallucination are reported with stimulant usage. Tactile hallucination is a sensation of physical contact with something or somebody that does not exist in reality. In this case, we report a child with metylphenidate -induced tactile hallucination.
Case: A 8 year-old boy presented to our outpatient clinic with the initial complaints of poor attention, concentration problems, poor academic performance, hyperactivity at home, being exposed to accidents very often, forgetfulness and peer relation problems. Same problems occurred in many other situations and were reported by the school teacher as well. Parents also reported that they complaints first occurred while he was 3 years old. In our sessions he was fidgety and restless and he did not want to do the reading and writing tasks. The Turgay forms filled by parents and teacher confirmed that the problems were over threshold. There was no history of a medical illness or a family history of psychiatric disorders. He was diagnosed with ADHD as per DSM-IV. Metylphenidate immediate release form 5 mg twice daily was prescribed, the dose was titrated to 10 mg twice daily in the second week. No side effects were reported in this period. We prescribed 27 mg Osmotic Release Oral System (OROS) methylphenidate in the third week of treatment. On the Sixth day of MPH OROS treatment, the patient and his mother presented to our outpatient clinic. He was anxious. He told that their neighbours were touching his hair and patting his head. He was anxious about seeing them and beeing touched again. His mother told that although he did not meet with their neighbours these worries started suddenly he was anxious and avoiding to see the neighbours. He wanted to stay at home all day long. Hallucinations disapperad after Osmotic Release Oral System methylphenidate was ceased and switching back to Metylphenidate (MPH) immediate release form 10 mg twice daily. His ADHD symptoms improved and no significant side effect occurred in follow up sessions.
Metylphenidate may be related with hallucinatory symptoms. Individual variability in hallucinatory symptoms related with methylphenidate

attributed to the differences in dopamine metabolism. In our case it is unclear how hallucinations occur with OROS MPH but not with immediate release form but it may be related with exposure time rather than dopamine metabolism. Clinicians should be careful about hallucination in all forms of MPH.

Keywords: ADHD, methylphenidate, hallucination

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[Abstract:0353][Psychopharmacology]

Hair- pulling after use of fluoxetine: two cases

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Trichotillomania (TTM) is a mental disorder characterized by uncontrolled and impulsive hair-pulling leading to hair loss, distress and disordered functioning. Treatment choices include behavioral therapy and Selective Serotonin Reuptake Inhibitors (SSRI). Fluoxetine is among the most frequently used SSRIs for this indication. However, randomized controlled trials conducted with it have led to controversial results of effectiveness for TTM. Here, we report two female patients one of whom developed TTM immediately after fluoxetine use while the complaints increased after fluoxetine use in the other.

Case 1: A 5 year-old girl diagnosed with adjustment disorder (with depressive symptoms) and was started on fluoxetine 5 mg/day. On the third day of treatment, she displayed irritability, hair pulling and collecting and plucking her eyebrows. The behaviors were severe enough and led to her mother to stop treatment on the 3rd day. An evaluation with Naranjo Algorithm led to a score of 3 (possible) for determining the relationship of causality between fluoxetine and hair-pulling. The patient was re-evaluated and started supportive psychotherapy with frequent visits. The hair-pulling remitted quickly and recovered in follow-up.

Case 2: A 7 year-2 months old girl who was brought to our department with complaints of "plucking her eye-lashes and eye-brows as well as hair pulling". The parents reported that they presented to a different center a few months ago for "hyperactivity, inattention, irritability, disobedience and plucking her eyebrows and eyelashes" and that she was started on fluoxetine 5 mg/day. Within days of receiving this treatment, the parents reported an increase in irritability, disobedience, insomnia, plucking eyebrows and eyelashes as well as an onset of hair-pulling. History and mental status examination was concurrent with diagnoses of attention deficit hyperactivity disorder (ADHD) and TTM. An evaluation with Naranjo Algorithm led to a score of 3 (possible) for determining the relationship of causality between fluoxetine and hair-pulling and fluoxetine was stopped. Irritability, insomnia, disobedience, hair, eye-lash and eyebrow pulling all reduced immediately after cessation of fluoxetine. The scientific literature includes reports of alopecia with fluoxetine while there were no reports of onset/flaring of TTM. Contrarily, it was reported that SSRIs, including fluoxetine may be used for managing TTM. Onset/ flaring of TTM in both of our patients was observed after monotherapy with fluoxetine and remitted with its cessation, leading to a suspicion of causality. On the other hand, onset/flaring of TTM was immediately after receiving treatment in both of our patients and coincided with other signs of behavioral activation/ agitation, such as irritability.

All of those observations may point to an alternative hypothesis, that TTM may be a sign of behavioral activation in some of the predisposed children receiving fluoxetine. The exact mechanism by which fluoxetine may trigger TTM is not known, but we posit that it may indirectly increase dopamine and norepinephrine release in prefrontal cortex via 5HT1C antagonism.

Keywords: fluoxetine, hair-pulling, trichotillomania, attention deficit hyperactivity disorder

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[Abstract:0354][Others]

Different faces of dissociative disorder: a case report

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Dissociative disorder is a commonly misdiagnosed disorder. Different behaviors of the alter personalities and presence of hallucinations or complaints about mind control may result in misdiagnosis like psychotic disorders or affective disorders. Here we report a case with multiple misdiagnosis like MS and mental retardation.
Case: A 39 year-old female patient presented to our hospital with anxiety, anhedonia and blurred vision complaints. She had been on therapy for a while but as her complaints were increased and it was hard for her to keep up with the therapy sessions she was hospitalized. Previously, lamotrigine was prescribed for her depressive symptoms but it was later stopped because of rash side effect. Currently she was on venlafaxine 150 mg/day treatment. In psychiatric examination her affect was puerile and her mood was depressive. No psychotic symptoms were detected. In her medical history she was hospitalized in a university hospital 8 months ago. At this time she claimed to be blind but later her vision was improved and the diagnosis was suspected as multiple sclerosis. As the tests came negative for MS and other organic disorders she was referred to psychiatry. Because of her puerile affect and history of failing in elementary schoolmental retardation was considered. But during hospitalisation medical staff observed changes in her affect and behaviors. One day she was fixing the broken things in hospital, one day she was seductive and on another day she had puerile behaviors. She scored 35.3 on Dissociative Experiences Scale. She kept asking for discharge and eventually she was discharged from the hospital on the 6th day by her family members with a diagnosis of dissociative disorder. Haloperidol drops 3x10 and venlafaxine 150 mg/day was recommended. During outpatient follow-up different alters with different intellectual levels, and speaking in different accents were observed.
Different misdiagnoses are seen in literature in patients with dissociative disorders. Usually bipolar disorder or psychosis are considered in these patients. In this case it is interesting to see a patient who was mistakenly considered as mentally retarded.
Keywords: dissociative disorder, alter personalities, misdiagnosis

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[Abstract:0357][Others]

Childhood disintegrative disorder: a case report

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Childhood disintegrative disorder is a very rare pervasive condition distinct from childhood autism, characterized by normal development of verbal and nonverbal communication skills, social interaction, play, bladder and bowel control and motor behavior at least in the first two years, followed by regression between 2-10 years of age in two or more of the mentioned developmental areas and resulting from disintegration of mental functions and progressive neurological abnormality. Estimated prevalence of the childhood disintegrative disorder is around 1.1-6.4 per 100.000 children and the common age of onset is 3.36 years. Childhood disintegrative disorder is not determined in DSM-5, the present case is evaluated in the content of DSM-IV criteria system.
Case: A 4 years and 10 months-old girl who developed childhood disintegrative disorder after approximately of normal development. The symptoms appeared suddenly 2 weeks ago before the first outpatient clinical control. Till two weeks ago, she was totally ordinary, talkative and communicative with other people. Firstly, her speech quantity suddenly decreased and in a few days she stopped talking. Nonsense laughs supervened to the clinical scene. In addition, her sleep quality dramatically decreased, she was screaming while sleeping, and seeing nightmares. And she also has loss of appetite. During the first policlinic interview, she has not talked. She was not willing to set eye contact. All information were taken from the family. Traumatical life event was not determined except nightmares. At the end of first outpatient clinic follow-up, her treatment was planned as risperidone 1 mg/day, and next follow-up was determined as two weeks later. After two weeks, at the second inspection, it was learned that risperidone treatment terminated by patients family at the third

day because of risperidone's hypersomnia side effect. Family reported that the patient's anxiety dramatically decreased and she started talking again. Pediatric Neurology consultation was also planned, firstly they looked for the neurometabolic disorders, thyroid problems, by blood analysis, there was not any abnormal result. And there was not any pathological findings at both routine eeg and sleep egg monitoring and cranial magnetic resonance monitoring. Neurological examination findings reported as normal. With these consultation notes the patient has been referred to children psychiatry clinic.
Clinical features are discussed in the light of the current literature.
Keywords: disintegrative disorder, childhood, regression

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[Abstract:0358][ADHD]

Determining of the adult ADHD and major depressive disorder comorbidity is valuable for functioning: a case report

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Attention deficit and hyperactivity disorder (ADHD) is the most common neurodevelopment disorder of childhood that persists into adulthood in the majority of cases. In adults, the clinical picture of ADHD is complex and comorbidity with other psychiatric disorders is the rule. However, the diagnosis of adult ADHD is sometimes difficult among the comorbitidity of major depression and adult ADHD. In this case, ADHD, which can be co-morbid with major depressive disorder (MDD) diagnosis, has been presented. The recognition and treatment of this disease will be presented.
Case: A 21 year-old male patient, university second grade student completed the first grade in two years. He expressed his finals were about to begin. Sadness, hopelessness, intense common worries, unwillingness, emotional lability, decreased self esteem, self blame, psychomotor restlessness, attention/concentration disorder, sleep disorder and 10 kg weight gain have been determined. These symptoms have been present for a year. Currently, there has not been a responded in symptoms with Fluoxetione 40 mg. Basic laboratory tests were normal. With preliminary diagnosis of MDD with anxious type, Venlafaxine 75 mg/day and Trazodone 50 md/day have been started. Venlafaxine 225 mg/day has been gradually increased. Depressed mood, hopelessness, common worries, self blame, psychomotor restlessness and sleep disorder symptoms have responded to the treatment. Inability to plan studying, inability to focus, inability to sustain attention, the absence of the concept of time, inability to organize, lack of motivation and decrease in self esteem symptoms have decreased slightly but continued. It has been identified that these symptoms have been present even before depressive and anxious symptoms. It has been stated that these symptoms were accompanied lightly in elementary and high school and they started to become evident when he began university. With ADHD comorbidity methylphenidate extended-release 18 mg/day has been added to the treatment. Methylphenidate treatment was increased to the 27 mg/day. With this treatment, a significant well being was observed in current symptoms. Venlafaxine 225 mg/day and methylphenidate 27 mg/day pharmacotherapy has been functional with support-oriented psychotherapy and family support. He passed the class with a grade above grade point average. The symptoms he had experienced in the previous year were not observed in the first period of third grade. It has been identified that his self esteem, attention, focus, organization and planning are functional. He has still been using Venlafaxine 75 mg and Methylphenidate/day.
Adult ADHD is often comorbid with psychiatric disorders. Many adults with ADHD visit psychiatrists with psychiatric symptoms. Focusing on the growth history and difficulties in daily life persisting after the remission of major depression enables psychiatrists to diagnose adult ADHD. However, the diagnosis of adult ADHD is sometimes difficult, because ADHD symptoms can be regarded as symptoms of major depression. There exist, however, slight differences in symptoms of adult ADHD and major depression.
ADHD therapy may help to improve symptoms of certain psychiatric comorbidities, such as depression. Therefore, management of ADHD may help to stabilize daily functioning and facilitate a fuller recovery.
Keywords: adult ADHD, major depression, comorbidity, diagnosis

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[Abstract:0359][Motor disorders]

A case of bipolar disorder with tardive dyskinesia responding well to clozapine treatment

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Tardive dyskinesia (TD) is an iatrogenic, neurological, hyperkinetic movement disorder characterized by repetitive, involuntary, purposeless movements in the oral/ lingual/ buccal area, or choreoathetoid movements of the extremities. The subtypes of TD include movement disorders such as tardive dystonia, akathisia, blepharospasm, myoclonus, tics/tourettism, tremor, and gait. TD prevalence is estimated to be 20–50% of all patients treated with neuroleptics. TDs are caused exclusively by Dopamine Receptor Blockers (DRBs) such as antipsychotics, also by DRBs prescribed for nausea (metoclopramide) and dizziness (prochlorperazine).
Case: A 78 year-old, male patient. Married, has 8 children. He presented to our hospital with involuntary movements of tongue, mouth and bilateral eyes. In the psychiatric history, he had mood episodes for 1-2 weeks which includes talkativeness, sleeplessness and increased rate of spending money. He had been prescribed depot injection treatment (probably antipsychotic) several times, 20 years ago. After those injections, he has got involuntary movements, beginning at 1995 and being more prominent in the last 5 years. He used 100 mg of sertraline and 25 mg of imipramine irregularly for psychiatric problems without admission to psychiatrists, and hydrochlorothiazide/ losartan (12.5 mg/50 mg) daily for hypertension. In psychiatric evaluation, there were mild depressive symptoms, involuntary movements prominent in eyes and orobuccal area. AIMS score was 24. Neurology consultation was requested. They decided to start 1000 mg/day of valproic acid (VPA) and 30 mg/day of baclofen with the diagnosis of blepharospasm and orobuccal dystonia. Our diagnosis was TD. During the next 3 weeks he continued to take 1500 mg/day VPA and 30 mg/day baclofen. His involuntary movements began to decrease but cognitive impairments came out, which were thought to be due to side effects of VPA. So we decided to decrease the dosage of VPA to 1000 mg and begin clozapine 25 mg. During the follow up we increased clozapine dosage to 37.5 mg. After two weeks of clozapine treatment AIMS score was 6 and involuntary movements were totally under control.
Treating TD is often unsuccessful, making prevention particularly important. Prevention can be achieved by limiting DRB use to only when it is necessary and using atypical DRB where possible. For the second generation APs the risk of TD was found to be similar among all APs except for clozapine and risperidone which have lesser risk. Clozapine, has also been shown to reduce abnormal movements in several small studies. Although there are some reports in the literature mentioning about TD due to clozapine treatment, clozapine successfully treated the TD symptoms in our patient. Other treatment alternatives are, tetrabenazine, dopamine depleters, vitamin E, calcium channel blockers, noradrenergic antagonists,benzodiazepines, cholinergic agonists, dopamine agonists, buspirone, GABA agonists, opioid antagonists, ECT, Deep brain stimulation, botulinum toxin type A. TD treatment is compelling in clinical practice and evidence-based treatment algorithms are insufficient. According to studies; risperidone and clozapine have low risk of causing TD. Some studies show that clozapine reduce involuntary movements and in our case low dosage of clozapine reduced involuntary movements successfully.
Keywords: tardive dyskinesia, blepharospasm, clozapine, bipolar disorder

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[Abstract:0361][Eating disorders]

A case of anorexia nervosa responding well to paliperidone palmitate

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Anorexia Nervosa (AN) is defined as the maintenance of body weight significantly less than expected, characteristic psychopathology including fear of weight gain and disturbance in the experience of body shape and weight. It's a chronic disease with a lifetime prevalence of about 0.48–0.6% and long-term relapse rates up to 50%. It has the highest mortality rate of any psychiatric disorder. Current management of severe AN typically involves a multi-disciplinary approach combining refeeding, psychoeducation, cognitive behavioral therapy and family interventions. At present, pharmacotherapy with antipsychotics is not recommended according to treatment guidelines and psychotherapeutic treatment approaches appear to be more promising. However, the number of prescriptions of second-

generation antipsychotic drugs (SGAs) to anorectic patients has substantially increased over the last years. Unfortunately, treatment is often unsuccessful, with a 40% dropout rate from intensive programs, and only 23% of patients achieving a good outcome at one year.
Case: A 45 year-old, female patient, divorced, has one child, lives with her mother, father, brother and daughter. Nearly 1 year ago, she presented to our outpatient department with the complaints of aggression, food refusal and vomiting, she was diagnosed as Anorexia Nervosa with comorbid anxiety. Mirtazapine 15 mg/daily was prescribed and consulted to internal medicine department for medical complications In disease history; at the age of 21, she had feelings of discomfort due to 93 kg of weight (BMI: 43) and began heavy exercise programs, low calorie diets and self-induced vomiting. By the years; she lost weights up to 30 kg (BMI: 13). She had amenorrhea, dental carries, hair loss, constipation, repetitive headaches and dizziness, but she still had thoughts about her overweight. She persisted on frequent eating and spontaneous vomiting attacks. She missed next check-up and was supposed as drop-out, but after 6 months she presented again due to demands of her family. She complained about irritability, social withdrawal, sleeplessness, frequent eating and spontaneous vomiting. In psychiatric evaluation, it was noticed that she got predominant negative psychotic symptoms and reference ideas. She was diagnosed as Anorexia Nervosa; binge-eating type with extreme severity and comorbid atypical psychosis. She refused inpatient psychiatric treatment and we asked for general medical consultation. She was prescribed paliperidone palmitate 50 mg/ monthly/ IM injection, supported socially and motivationally. The response to treatment was satisfactory. In couple of weeks, vomiting stopped and she began to gain weight, psychotic symptoms decreased. After 6 months of follow up,her weight was 60 kg and she told that "I regained the hope of life". She is still in complete remission and continuing her treatment.
Atypical antipsychotics appear safe and there is some evidence of positive effects on depression, anxiety and core eating disordered psychopathology in patients with anorexia nervosa. The most studied drugs were olanzapine, quetiapine and risperidone. In literature, there is no studies about paliperidone, main opinion about SGAs in AN treatment is negative. So this case is point of rising expectancy about AN treatment.
Keywords: anorexia nervosa, psychosis, atypical antipsychotics, paliperidone

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[Abstract:0363][Psychopharmacology]

Encephalopathy associated with valproic acid used in the treatment of bipolar disorder: a case report

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Bipolar disorder (BD) is a chronic psychiatric disorder with recurrent depressive and manic attacks and euthymic mood in-between these attacks. Valproic acid is a FDA approved mood stabilizer used in the treatment of BD in children and adolescents. Encephalopathy is one of the rare side effects of valproic acid; however, it may cause serious consequences. Valproic acid-induced encephalopathy symptoms, sudden disoriented behavioral and cognitive changes, loss of memory,changes in personality, myoclonus, ataxia, throwing up, convulsion, coma, and death. In this report, we will discuss a case who developed valproic acid-induced encephalopathy.
Case: A 18 year-old female patient was brought to our clinic with complaints of talking too much, maladaptive behaviors, spending too much money improperly, little sleep, increase in restlessness, exaggerated make up, meeting people she did not know and having sexual conversation with them and causing damage which had started a week ago. The patient whose examination showed distractibility, flight of ideas, irritability, illusions of grandiosity, increased psychomotor activity, increased speed of speech was hospitalized with a diagnosis of "Manic Episode". The patient's complete blood count,routine biochemistry, renal, liver and thyroid function tests were made and organic etiology was excluded. Quetiapine 600 mg/day, valproic acid 1000 mg/day, lorazepam 7.5 mg/day treatment was started. On the ninth day of the treatment, there occurred a 30 second generalized spasm in the patient who had increased predisposition for sleep and developed mental fog. The patient's neurological examination showed that her conscious was confused and she was not following the orders given. No other pathological symptom was found in her physical examination, vital values and laboratory results. Serum valproic acid level which was checked for intoxication was found as 108 µg/mL. Brain CT was reported as "normal", while EEG was reported as "diffused slow waves,there may be drug-induced encephalopathy". Thinking that the patient's clinical picture may have developed due to valproic acid,all medical treatment including valproic acid was cut and the patient was given fluid support. After valproate treatment was stopped, clinical picture was seen to improve within 36 hours. The patient was discharged due to significant improvement in clinical picture with a therapy of lithium 900 mg/day.

Drug-induced encephalopathy is a serious clinical picture and a great number of drugs may cause this picture. Valproic acid is the most known of the drugs which cause encephalopathy. The pathophysiology of valproic acid induced encephalopathy has not been fully explained. Although there is an emphasis in literature that hyperammonemia due to liver or renal failure may be the main reason,the great number of cases in which there is an absence of elevated serum ammoniac levels is remarkable. This case also developed a picture of encephalopathy without hyperammonemia. The quick and obvious change in the clinical picture after the valproic acid therapy was stopped, supported our diagnosis. Early recognition of the encephalopathy picture in the present case and taking the necessary precautions prevented the picture from becoming more severe and from developing serious complications. With this case report, we wanted to emphasize encephalopathy which is a rare but serious side effect of valproic acid. Early recognition of the valproic acid -induced encephalopathy is very valuable in terms of the reversal of this severe picture which may result in death. Valproic acid-induced encephalopathy is a rare complication of valproic acid which may cause fatal consequences.

Keywords: bipolar disorder, encephalopathy, valproic acid

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[Abstract:0364][ADHD]

Is she an adult ADHD or borderline personality disorder patient?: a case report

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Adult Attention deficit and hyperactivity disorder (ADHD) can resemble, and often co-occurs with, borderline personality disorder (BPD). This can lead to mistaken diagnoses and ineffective treatment, resulting in potentially serious adverse consequences. Two conditions can substantially impair well-being and functioning, while it may be difficult to determine the ADHD.

Case: A 30 year-old female patient, single, resident physician. She feels unsuccessful in human relations. She thinks that she has had superficial relationships and got in touch with wrong people in her relationships. She states that her thoughts and behaviors are different from each other and thus she is unable to stand on truths. She can be angry, sadness, tense. She is unable to concentrate. She is unable to put in order, start or leave unfinished her dissertation and existing publications. She states she gets up tired and unwillingly. She states that her life is generally irregular. She expresses that she increased alcohol intake in the past years but she has not drunk alcohol lately. It was determined that she has material extravagances, difficulty in managing her economy, and a large amount of debt. She has a sense of failure and feels herself powerless. She stated that there was no change in her symptoms with antidepressant treatments in the past. Mood stabilizer valproate 1000 mg per day were started for emotional lability. There were not any changes in symptoms in two months period. With preliminary diagnosis of adult ADHD, methylphenidate extended release 18 mg/day was added. She has stated that she is able to get up willingly and lively in the mornings. She has also stated that she has started her dissertation and put her articles in order. The dosage of methylphenidate was adjusted to 27 mg/day. She has expressed that she has become to be more stabile in human relations. It has been determined that she has started to maintain control over her behaviors. Emotional lability has not been significantly observed. It has been reported that she has started to have economy control. It has been expressed that there has not been a sense of failure and she has not felt herself powerless anymore. She has still been in her first year of Methylphenidate 27 mg/day treatment.

There is substantial symptomatic overlap between adult ADHD and BPD in adults, but the nature of the relationship between these disorders needs further clarification. Attentional uncontrol might be a useful criterion for differential diagnosis. ADHD in adults and BPD share some similar clinical features (e. g., impulsivity, emotional dysregulation, and cognitive impairment). Some evidence for a potential common neurobiological dysfunction suggesting the hypothesis that ADHD and BPD may not be two distinct disorders, but represent at least in a subgroup of patients two dimensions of one disorder. It is important to accurately diagnose ADHD, and BPD to ensure correct targeting of treatments and improvements in patient outcomes. Differential diagnosis, comorbidity, and treatment of attention deficit hyperactivity disorder in relation to borderline personality disorder in adults.

Keywords: adult ADHD, borderline personality disorder, differential diagnosis, comorbidity, treatment

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[Abstract:0370][Mood disorders]

Manic episode after left hemisphere infarct in two cases

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Depression is the most common mood disorder after cerebrovascular incidents at a rate of 50%. However, post-stroke manic episodes are rarely seen. In this report, we presented two cases with post-stroke manic symptoms.
Case 1: A female case of a right-handed, 64 year-old presented to psychiatry department because of insomnia, increased in talkativeness, and irritability. She had severe paranoid delusions about being followed and captured her photos by strangers. She also reported some jealousy delusions for her husband, and visual hallucinations. The total score of Young Mania Rating Scale (YMRS) was 42. Her manic symptoms emerged sixteen months later from her cerebrovascular incident 1.5 years ago. Magnetic resonance imaging showed cystic encephalomalacia areas in left basal ganglia related to underlying infarcts (Figure 1). For her manic symptoms, 25 mg/day of quetiapine was initiated and increased to 100 mg/day. After 5 weeks, it was observed that her manic and psychotic symptoms disappeared. Her total score of YMRS was decreased to 2.
Case 2: A male case of right-handed, 69 year-old presented to psychiatry department because of talkativeness, and increased sexual desire, spending too much money, insomnia, and irritability. He was euphoric and had a marked grandiosity, and jealousy delusions for her wife. He had a total score of 31 on YMRS. These symptoms first occurred 1 years later from cerebrovascular incident 2 year ago. Magnetic resonance imaging showed chronic infarct regions in left parieto-occipital area (Figure 2). He was initiated a 5 mg/day of olanzapine and then gradually increased to 15 mg/day. 4 weeks later, there was a substantially reduction in manic symptoms with a score of 9 on YMRS.
Secondary mania may occur following several metabolic, neurological and/or toxical disorders. Previous observations demonstrated that manic symptoms secondary to cerebrovascular events occurred frequently within 2 years after the incident cerebrovascular event. In our first case, manic symptoms occurred within first year following the cerebrovascular incident. In the second case, we suggested that manic symptomatology emerged one year later following the diagnosis of left parieto-occipital infarcts, in accordance with previous findings. It is well known that there are significant relationships between the localization of the cerebrovascular incidents and neuropsychiatric disorders. Manic symptoms are more likely to be related to right hemisphere lesions. To date, a few cases of mania related to left hemisphere lesions were reported. Our cases is of interest since a rare casual relationship existed between left hemisphere lesions and manic symptomatology. First manic epizode in elderly may be related to disorders that cause seconder mania. For this reason, patient's medical history, examination and laboratory results should be assessed detailed. Also,we should take into account that manic cases can be related to left hemisphere lesions not only due to right hemisphere lesions as shown in the literature.
Keywords: Infarction, left cerebral hemisphere, mania, stroke

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[Abstract:0372][Others]

A case of catatonia with atypical clinical presentation

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18 year-old male presented to our emergency service with complaints of increase in sleep, increased food intake, irritability and mutism for one week period. After the tests and consultations had been carried out in the emergency service the patient was admitted to the psychiatry inpatient unit. In the neurologic examination: the consciousness; stupor, motor examination; not cooperative, sense and cerebellar tests; not cooperative. In the first week after his admission he slept for 18-21 hr/day, he was eating a lot and compulsively. When he awakened he was conscious, but apathetic and negativistic. When conversation ended he was falling asleep. He was bringing his hands above his penis often (hypersexuality?). He was talking about events that were not happened in reality (confabulation). Fever, pulse and blood pressure has been checked 8 times/day and were normal. The laboratory tests, electroencephalography and cranial MRI were normal. For differential diagnosis lomber punction has been maden, the microbiological and pathological results were normal. In the first

week the symptoms were hyperphagia, increase in sleep, apathy, cognitive problems, hypersexuality? Our pre-diagnose was Klein-Levine syndrome. At the 8th day the patient stopped eating and speaking. The mutism, cessation of oral intake, negativism, waxy flexibility and rigidity symptoms continued for 4 days. The diagnose was catatonia. The patient has been prepared for electroconvulsive therapy (ect) and lorazepam 2 mg/day oral treatment initialized. After four days he started eating and drinking; the mutism, waxy flexibility, rigidity symptoms ended without ECT. The food intake, hyperphagia and compulsive eating, negativism, apathy, hypersexuality symptoms has started again. Lorazepam 2 mg/d treatment has been continued. After 25th day symptoms started to reduce and at the 30th day he was sleeping 8 h/day, he was eating 3 times/day and he was not eating compulsively. Also his cognitive functions were almost normal. At 31st day he was discharged from hospital. Catatonia was originally described in 1874 by Kahlbaum as a unique clinical presentation of motor, vocal and behavioral abnormalities. It was incorporated as a type of dementia praecox by Kraepelin in 1896. In DSM-5 catatonia was conceptualized as an independent syndrome. The creation of a separate diagnostic class for catatonia in future classification systems seems the safest approach for this syndrome in patients of all ages and the best approach to promote research. In literature there are lots of case presentations and studies, show that catatonia may include a variable symptomatology. Especially in children and adolescents, catatonia may be hidden in plain sight among different disorders like autistic and other developmental disorders, autoimmune encephalitis, tic disorders, Kleine-Levine syndrome and various other disorders. Also in our case there are variable symptoms like change in food intake, increase in sleep and hypersexuality before and after the classic symptoms of catatonia, so this shows us catatonia may include a different variable symptomatology. The etiopathogenesis of catatonia remains unclear. Several models are available to guide future research but are limited because of our lack of understanding of brain-behavior relations. Future studies should test EEG, brain imaging techniques that can quantify neurotransmitter levels as in magnetic resonance spectroscopy, neuroendocrine tests and autoimmune parameters to assess possible biomarkers of catatonia and predictors of treatment response and outcome.

Keywords: catatonia, hyperphagia, Kleine-Levine syndrome

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[Abstract:0374][Anxiety disorders]

Montelukast-induced anxiety in a child with allergic asthma

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Montelukast, a leukotriene receptor antagonist, is approved by the United States Food and Drug Administration (FDA) for the prophylaxis and long-term treatment of asthma, prevention of exercise-induced bronchoconstriction, and symptomatic relief of allergic rhinitis.

Case: A 6 year-old girl with sudden onset anxiety, sleep problems and behavioral problems after montelukast therapy. She had allergic asthma and did not have any psychological complaint until montelukast therapy. The patient experienced of new onset various anxiety symptoms and sleep disturbances within 1 month of starting montelukast 5 mg at bedtime. She started to refuse sleep alone, had nightmares and fear from cartoon characters and darkness. She had difficulties to separate from her parents, could not speak or read anything in front of the classroom. She was nervous, anxious and crying a lot and had difficulty to falling asleep. She did not want to play and spend time with her friends. We got information from our patient, her parents and her teachers in school, about any bad life event, something which can traumatized her or any kind of psychological and biological stressors. In physical examination and laboratory investigation, there was no significant reason to explain these complaints. In psychiatric examination, mood was anxious, affect was appropriate, speech,thought content and thought process was normal. There was no perceptual disturbance, no suicidal or hostile thoughts. Appetite was not changed, sleep time and quality decreased compared to the past. These symptoms subsequently resolved 2 weeks after discontinuation by communicate with her pediatrician. There is no recurrence so far at her follow-up visits.

Anxiety disorders can negatively affect children's daily lives, can lead to school underachievement, social problems, sleep disorders and behavioral problems. Although we know the allergic diseases and asthma can be comorbid situations with anxiety disorders, or a cause of anxiety disorders, in this case, we think that there is a probable relationship between development of patient's anxiety symptoms and montelukast therapy. There are studies about explaining tendency relationship between allergic diseases especially asthma and psychiatric disorders. It is suggested that the role of immunoregulatory role of dopamine and serotonin, increased limbic system symptomatology and sensitizability, immune responsiveness and hypersensitivity and neuroimmunomodulation mechanisms. It may be that leukotriene receptor antagonist, montelukast, precipitates somehow all these symptoms. In literature, there are case reports about montelukast -induced suicidal behaviors, depressive and anxiety symptoms, auditory and visual hallucinations, mood swings, restlessness sleep disturbances like insomnia, somnolence, night terrors, sleep walking, bruxism, nightmares. In these articles, both adults and children

seem to develop psychiatric symptoms after the intake of the medication. In every patient, the symptomatology seems to becomeless with the reducing the dose or discontinuation of the therapy with montelukast within few hours or days. Although anti-leucotrienes are safe drugs, these symptoms have to be monitored especially in children. It is not very clear yet, with which mechanism and how often, montelukast makes these neuropsychiatric adverse effects. Health care professionals should be aware of these side effects and assess the process carefully.

Keywords: adverse reaction, allergic asthma, anxiety, montelukast

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[Abstract:0381][Mood disorders]

Mania occurring following epileptic attack: a case report

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Epilepsy is a condition known for centuries, but in spite of the developments in its diagnosis and treatment, it is comorbid with psychiatric disorders and affects the quality of life of the patients. Data on the relationship between epilepsy and manic episodes of bipolar disorder (BD) is limited. Here, it is aimed to evaluate the relationship between epilepsy and manic episodes of BD as well as their common features through a case presentation.

Case: A 62 year-old male patient with a 30 year history of epilepsy, who was a elementary school graduate farmer and who was married with three children, presented to the outpatient neurology clinic with epileptic attack, sleeplessness, hearing voices, suspiciousness and refusing treatment. The patient's history revealed that he had refused to take anti-epileptic medication for the last two months, and was deprived of sleep for a week, and had tonic-clonic epileptic attacks 20 times a day. Upon application to the emergency department, the magnetic resonance imaging scan and blood tests carried out did not indicate any abnormality. It was also learned that the symptoms other than epileptic attacks began on the third day of epileptic attacks. It was reported that the patient had suffered from similar symptoms following epileptic attacks twice a year for 30 years, and Carbamazepine and Levetiracetam treatment was not effective, yet there was a significant decrease in the number of epileptic attacks following the Valproate(VPA) addition to Levetiracetam, which also inhibited manic attacks. To decrease the frequency of attacks, doses of Levatiracetam (1500 mg/day) and VPA (500 mg/day) were increased to 3000 mg/day and 1000 mg/day, respectively. As the patient presented with mood disorder and accompanying psychotic symptoms, Quetiapine treatment was started and the dose was increased to 400 mg/day within a week. During the follow-up examination carried out in 3 weeks following his admittance, it was learned that his symptoms had disappeared and he did not suffer from any epileptic attacks.

BD and epilepsy have some common features, in that they are both of cyclic nature and may become chronic, and the etiology of both involve kindling phenomenon, neurotransmitter deficits, irregularities in voltage gated ion channels as well as secondary messenger systems. Changes in G proteins, phosphadityl-inositol, protein kinase C, alanine rich C kinase substrate, and calcium activity have been defined in particular, whereas the common mechanism in ion channels is that anti-epileptics have regulatory effects on potassium expression and antagonistic/anti-kindling effects on calcium. Antiepileptic medications may inhibit the manifestation of manic attacks in epileptic patients. In this case, manic attacks manifested themselves, following a severe epileptic attack, which indicates that they were triggered by the epileptic attack, rather than due to a comorbidity of BD. In epilepsy with BD comorbidity, carbamazepine, VPA, lamotrigine, and oxcarbamazepine can be used as a monotherapy. As psychiatric disorders' association with epilepsy is more common, psychiatric symptoms in epileptic patients need to be carefully examined.

Keywords: epilepsy, mania, bipolar disorder

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[Abstract:0383][PTSD]

Effects of EMDR on prolonged grief reaction process

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Prolonged grief reaction turns into depression and may lead to severe deterioration on functionality. This case is discussed, depressed and anxious feature of prolonged grief reaction symptoms change in the direction of significant and rapidly decreasing, during Eye Movement Desensitization and Reprocessing (EMDR) therapy.
Case: A 44 year-old female patient, married and she has four children. She is working in her own bakery. She lives with her husband and children. The complaints started ten months ago, before her 12 year-old son died of drowning in the pool. After the accident, patient has some complaints about anxious and somatic features including dejection, not able to do own duties and house works, tertian sleeping, head and facial numbness, shortness of breath and shaking hands and legs. After one session EMDR treatment, there were a significant decrease observed, about 50% percent of clinical scales as the known criterias of prolonged grief reaction process and depressive disorder acute and chronical complaints due to DSM-5. At the same time observed improvement to anxiety and depressive symptoms.
According to studies, EMDR is effective in many disorders. In this case discussed about extensive effects of EMDR therapy on prolonged grief reaction process and depressive symptoms. In addition determined effects of treatment on the sources of stress that makes the symptoms and observed effectiveness of EMDR treatment.
Keywords: grief reaction, eye movement desensitization and reprocessing, EMDR, depressive disorder

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[Abstract:0384][Anxiety disorders]

A conversion disorder case coming to emergency service due to prediagnosis of acute asthma attack

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Acute asthma attack encountered in emergency service can cause serious consequences if it is not interfered in the right intervention. In addition, conversion disorder is a situation causing deterioration in functionality. The cases which have high comorbidity in clinical features are not very common in the literature.
Case: A 20 year-old male patient, graduated from elementary school, single, living in a village with his family. His first complaints started two years ago in the form of breath shortness and the feeling of drownings when he learned his grandfather's death, but he did not get any treatment and his complaints decreased within a few hours and never occurred again. In the phenomena evaluated in the emergency service with current complaints, pulse oximeter and oxygen saturation decreased, coarsening of his lung volume and deterioration in the overall situation was identified in auscultation. It was treated with pre diagnosis of acute asthma attack, but within three or four hours after the response of the treatment through I.V. diazepam, the clinical situation rapidly resolved.
Psychological vocal cord dysfunction was firstly described in 1974, in the beginning it was thought that it imitates upper airway obstruction and then it presented that it can imitate asthma or it can worsen existing asthma symptoms. As it known asthma is a chronic disorder and it has an effect on patients' physical, emotional, and social life. Especially in the circumstances that symptoms cannot be controlled well, the importance of emotional factors comes forward more. Anxiety and conversion disorder are the most common psychiatric problems in the respiratory system diseases. Psychiatric problems, beside facilitating the emergence of respiratory symptoms of asthma, are a condition that complicates the asthma control. For this reason, especially if in the cases that necessary control is not provided psychic factors should come to mind.
Keywords: anxiety disorder, acute asthma attack, conversion disorder

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[Abstract:0385][Anxiety disorders]

A generalized anxiety disorder and panic disorder comorbidity due to unilateral hippocampal lesion: case report

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It is known that hippocampus (HCP) have an important role in learning and some pathways of fear. Isolated unilateral ischemic strokes of the hippocampus are rare. However, damage to this area may cause anterograde amnesia and prolonged cognitive deterioration (especially spatial learning). In contrast to transient global amnesia (TGA), amnesia is not transient. Although numerous causes of hippocampal infarction have been reported, an infarction which caused generalized anxiety disorder (GAD) and panic disorder (PD) has rarely been described. In this case, we aimed to demonstrate the importance of evaluating cognitive functions (e.g. memory) and conducting a radiological examination when evaluating older patients with anxiety disorders.
Case: A 68 year-old female patient, left handed, widow, living with her son presented to our outpatient clinic with a suicide attempt. Although the patient was taking her medications regularly, she was complaining from persistence of anxiety. She told that she decided to commit suicide rather than feeling anxiety. Patient was taking escitalopram 20 mg/day since ten years and was clinically stable for a long time; but she was complaining from severe anxiety since moving to new home 2 months ago. She was especially bad in "new home", was feeling about to be dying and was not remembering where rooms are or where things in the house are. Because her symptoms were mostly related to "new home", her drugs did not change in the first visit and she was hospitalized. Patient's neurological examination was in normal rate. She was diagnosed with GAD and panic disorder comorbidity. Two days later, 1 mg alprazolam per 12 hours for anxiety, on 10th day she wanted to be discharged. Treatment was not changed except adding alprazolam but patient told that "I feel very nice". At the night of the day she discharged, she presented again to clinic with a severe panic attack. Because of rapid progression of symptoms, the patient was reevaluated. In this evaluation, memory deficits was found. Although there was not a neurological finding, we decided to make radiological evaluation for demential findings and treatment resistance Brain MRI findings has shown total left hippocampal ischemia. Patient send to neurologist for further evaluation. In this patient, we described "treatment resistant" GAD and PD caused by HCP lesion. Because of this lesion, patient can't learn where are the "rooms of new home" and where are the "things", so that she felt an anxiety due to feeling of loss of control. Also HCP has an important role for regulation of fear, its' role in memory is also important. We thought that memory deficits played a more important role in this case. Organic etiology should always be kept in mind for older patients in not only new onset symptoms, treatment resistance or exacerbation of "older" symptoms may be signs of organic events. Patients with abnormally elevated or late onset GAD and PD need to be elevated for organic causes. Also existence of treatment resistance and cognitive findings should aware doctors for radiological examinations.
Keywords: hippocampus, anxiety, amnesia

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[Abstract:0388][Psychopharmacology]

Adding aripiprazole or reducing paliperidone dose for amenorrhea?: a case report

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Antipsychotics increase prolactin level by affecting dopamine D2 receptors in tuberoinfundibular tract and thus causes amenorrhea. In addition to classical antipsychotics, prolactin increase may occur as a result of the interaction with the same receptor during the use of amisulpride, risperidone and paliperidone, which is a metabolite of risperidone. Hyperprolactinemia may be asymptomatic, but sometimes it can lead to hirsutism, galactorrhea, gynecomastia, amenorrhea, infertility and a decrease in bone mineral density. Probably due to partial agonistic effect, use of Aripiprazole was reported to lower prolactin levels and improvement was experienced in the associated clinical symptoms in patients with this side effect.
Here, management and treatment regulation of amenorrhea was discussed in a patient with amenorrhea which was seen after the use of paliperidone.

Case: 41 year-old female patient, single. She has been followed for about 20 years with the diagnoses of schizophrenia. Because of a history of drug incompliance and frequent relapses due to incompliance, transition to long acting antipsychotic treatment was planned and paliperidone palmitate was started at a dose of 100 mg/month. Psychotic symptoms was improved with treatment but amenorrhea was developed in patient at the 3rd month of treatment. Prolactin levels were elevated in the patient and therefore she was consulted with a gynecologist and endocrinologist. Upon not finding an organic cause that will explain the clinical picture except antipsychotic use, increased prolactin levels was considered to be due to paliperidone treatment. 5 mg/day aripiprazole was added to treatment without changing the paliperidone dose. Although there was a decrease in prolactin levels at fourth month it was still higher than normal (the initial value: 45 mmol/L, second value: 32 mmol/L) and therefore the dose of paliperidone was reduced to 75 mg per month. Menstruation was seen in patient at the sixth month of treatment. Prolactin was decreased to normal levels. Treatment was continued with paliperidone 75 mg/day and aripiprazole 5 mg/day.
In this case report, prolactin increase and amenorrhea induced by long-acting paliperidone use has been shown to improve with dose reduction and adding aripiprazole to treatment. Although female patients in reproductive age may not feel prolactin increase as it happens, they are very sensitive to clinical outcomes of it and increased prolactin levels is an important factor influencing medication adherence in women. Last studies showed that due to its pharmacological profile as a dopamine D2-receptor antagonist, paliperidone ER was found to elevate mean serum prolactin levels in the 6-week studies. We observed same results, but decrease in prolactin level was lesser than expected. So that this case is different from most of paliperidone related amenorrhea cases; because of adding aripiprazole was not enough for reducing prolactin. According to information obtained from the literature, although there are reports on the management of paliperidone -induced amenorrhea with aripiprazole, by this case, we aim to contribute to the literature by showing that it could be also necessary to try dose reduction when adding aripiprazole is not enough.
Keywords: paliperidone, hyperprolactinemia, aripiprazole

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[Abstract:0394][Mood disorders]

Results of long term hospitalization in a geriatric patient with bipolar depression

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Bipolar depression period takes longer time to reach remission compared with manic episodes in bipolar disorder (BD) patients. In this paper, it has been aimed to report management of comorbid conditions during extended hospitalization period in a geriatric patient with bipolar depression.
Case: A 70 year-old male patient, followed-up for 22 years with a diagnosis of BD not compatible with the treatment of recommended, presented to hospital with sleepiness, loss of appetite, fatigue, immobility and suicide attempt. For two months he had limited oral fluid intake up to hospitalized. According to the history taken from his family, the lithium treatment was started him in psychiatric ward with the diagnosis of BD-manic episode in 1994 and he had hypomania episodes twice a year. The patient was hospitalized in our psychiatric ward with these complaints and quetiapine 300 mg/day was started. The patient had a deep venous thrombosis episode and CT angiogram showed pulmonary thromboembolism 10 days later from the start of quetiapine treatment. Quetiapine treatment was stopped and he was consulted to the department of chest disease, so coumarin 5 mg/day was started. After adjustment of anticoagulant therapy, sertraline 100 mg/day and quetiapine 100 mg/day were started. On the 58th day of his treatment, lithium 600 mg/day was added since his depressive symptoms did not regressed. At that period sertraline increased to 200 mg/day and quetiapine increased to 300 mg/day. He had also consulted to the department of internal medicine with constipation, dysphagia because of the decrease serum hemoglobin value and iron levels. Histological examination revealed tubular adenoma and colonoscopy was planned 1 year later. On the 80th day of his treatment, 0.9% NaCl infusion was started by the internal medicine department to the patient whose creatine level increased. On the 6th day of fluid infusion, since hypernatremia occurred, lithium and fluid infusion were stopped, dextrose infusion was started and lamotrigine 12.5 mg/day was added to the treatment. In the following days electrolyte levels was balanced, lamotrigine was increased to 200 mg/day. Sertraline was decreased to 100 mg/day and aripiprazole 10 mg/day was added to the treatment of the patient whose depressive symptoms did not regressed. Despite the sertraline treatment at the appropriate dose and duration. Mirtazapine 30 mg/day was started for depressive symptoms of patient who had also difficulties with maintenance the sleep and the dose was increased to 45 mg/day. His depressive symptoms regressed and he was discharged with fully remission.
As in this case, old age (>65), chronic respiratory disease, immobilization, active or recovered malignity history, having dehydration and

antipsychotic treatment increase the risk of venous thromboembolism. On the other hand lithium may cause polyuria. If fluid intake is restricted, there is the risk of hypernatremia. Extended hospitalization time brings along some difficulties related to the management of comorbid conditions especially in older patients.
Keywords: bipolar depression, geriatric psychiatry, comorbidities

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[Abstract:0395][Substance-related and addictive disorders]

Two cases of compulsive stock market investing

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Nowadays there is a debate about behavioral addictions. Behaviors such as gambling, shopping, sexuality,internet and video game playing have hedonic properties and have the potential of compulsivity among individuals that have tendency for addiction. So far, these behaviors are considered “impulse control disorders not elsewhere classified” in the DSM-IV-TR. However, gambling disorder has been reconceptualized as a "behavioral addiction" in the DSM-5, based on emerging parallels with substance use disorders. Other behavioral addictions have been the focus of much research in recent years. We present here two cases of compulsive stock market investing.
Case 1: A 33 year-old married male patient. He is a portfolio manager in a bank and has been investing money randomly on the stock market for 8 years. The patient had a history of online betting and gambling between ages 15-25. The patient had a great amount of credit debt and he even used the credit card of his wife. He spend very little time with his wife and children. He had no future plans in his life. The only activity that makes him excited is stock investing. We plan family counseling, cognitive therapy and drug therapy; however, he stopped continuing the therapy and follow-up after 3 sessions.
Case 2: A34 year-old married male shopkeeper. The patient had compulsive stock market investing problem for 10 years. He defines the excitement of following changes on the stock market as “heroine” for himself. He was also diagnosed as having major depressive disorder, obsessive compulsive disorder and schizoidal personality disorder. He was preoccupied with stocks and had been investigating the companies throughout the day and the night. However, he had also been investing according to his magical thoughts. He had sold 2 flats to pay his debts. He was divorced because of this problem. He had been initiated pharmacological treatment and supportive therapy. Compulsive stock market investing may be a new type of behavioral addictions. It has also been suggested as a subset of the gambling disorder. Diagnostic criteria of gambling disorder have been found to be applicable in excessive trading on the stock market. It is one of the most socially acceptable forms of risk taking and may cause severe economical and psychosocial problems. Moreover,it is easier nowadays to invest on the stock market via Internet. Compulsive stock market investors have been found to be similar with pathological gamblers in psychometrical measures. There is a need for neurobiological and psychometrical research, development and validation of specific assessment tools, prevention and treatment strategies.
Keywords: addiction, behavioral addiction, compulsivity, gambling

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[Abstract:0397][Schizophrenia and other psychotic disorders]

Paranoid schizophrenia induced by stressful life events

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People with psychotic disorder have genetical, psychological and physiological vulnerability and generally stressful life events trigger psychotic process. We aim to remark in this case, stressful life events contribute to psychotic disorder in vulnerable people.

Case: A 35 years-old female patient, elementary school graduate, unemployed, married for 12 years, has two daughters (4 and 10 year-old). Although formal marriage continues, because of her husband's refusal, she has been living with her parents for 6 months. Legal divorce process has also been started. Her parents live in village and ranching. Due to difficult life conditions, her parents could not give enough care and interest to the patient. Because of her parents' attitude, the patient had lived in the care of her aunt in another city. In this period, she had worked with her aunt on house cleaning. When she was 23 year-old, she had got arranged marriage and had given birth twice at the age of 25 and 31. After both delivery, she had complaints like joylessness, unwillingness, afraid of could not care of children and she had stayed in hospital for a few times with these complaints. Also in these hospitalizations she had minor psychotic symptoms like suffer evil and not being able to maintain social interactions. The puerperal period is a critical one in the life of a woman and may emerge during this period which represents a "risky" phase from a psycho-pathological point of view. Patient's current complaints; unhappiness, anhedonia, sleepiness, unwilling to talk anybody, thinking that people backbiting, doing something behind her and suffer evil got started before 6 months. And finally she shot herself from her abdomen by rifle. There is no remarkable feature in family psychiatric history.
In childhood having careless parent (emotional neglect), in adolescent living with her aunt and working in house cleaning (emotional and physical abuse), not getting enough interest by her husband (emotional neglect) can make our patient vulnerable. This case shows us that people who got affected from developmental life events and have vulnerability may manifest as psychotic disorder.
Keywords: psychotic disorder, stressful life event, vulnerability

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[Abstract:0399][ADHD]

Adult ADHD or bipolar disorder: a case report

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The attention deficit disorder and the bipolar disorder maintain a complex relationship. Indeed, these two syndromes share numerous symptoms that engender numerous diagnostic difficulties. The author report a case of an adult with attention deficit hyperactivity disorder erroneously diagnosed and treated for bipolar disorder for 15 years.
Case: A38 year-old male patient, married with 4 children, businessman. He describes his complaints with psychiatric terminology. He has expressed that he has had attacks continuously, he has been in depression for the last one last year and he has been in new quests when he is in hypomanic episodes. He states that he has spent time at home for months and has not gone to work. He expresses that he has tried six different jobs before his current job but none of them has turned out all right. Most of the time, he can not fulfill his work and family responsibilities sufficiently. It is determined that he has not taken any responsibilities. He has spent most of his time on the internet. His academic achievement was low in his previous history. Social adjustment difficulty has been reported. His spouse has received Major Depression treatment, and his children have been treated with diagnosis of ADHD. Lithium 1200 mg/day, lamotrigine 200 mg/day, quetiapine 200 mg/day in his treatment have been decreased gradually and discontinued. Methylphenidate was started as 18 days and has still continued gradually 54 mg/day. He has gone to work regularly for the first year of the treatment. He has expressed that he has not experienced depression and hypomania episodes which he described in the past periods. The challenge of accurate diagnosis remains at the heart of good psychiatric treatment. In the current state of psychiatry, a confluence of forces has increased this challenge for the clinician. These include practical pressures-such as limited time for diagnostic evaluation. The issue of directing patients to acute treatments and also trends in nosology, such as the descriptive focus on signs and symptoms in the current official diagnostic system.
Distinguishing between adult ADHD and bipolar disorder requires careful attention to phenomenology with a focus on childhood history, lifetime course of symptoms. It is important to accurately diagnose ADHD and BD to ensure correct targeting of treatments and improvements in patient outcomes. This can lead to mistaken diagnoses and ineffective treatment, resulting in potentially serious adverse consequences. Two conditions can substantially impair well-being and functioning. The current case shows the importance of careful differential diagnosis when evaluating for bipolar disorder.
Keywords: adult ADHD, bipolar disorder, diagnosis

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[Abstract:0400][ADHD]

Adult ADHD can be detrimental to educational functioning: a case report

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Adults with ADHD may underestimate the impact of ADHD symptoms. In many cases have learned to compensate for ADHD related impairments by choosing lifestyles, but some cases with ADHD can be detrimental to some areas of life including work, daily activities, social and family relationships and psychological and physical well-being. In this case, an adult with ADHD, who has been unable to complete his university education, will be presented.
Case: A24 year-old male patient, second grade university student, has been in the sixth year of his education. He came to interview with his parents. His family learnt that Mr. B was still a second grade student in the last six months. This year, he left the city where the university was located and has started to live in the city where his parents live. The anamnesis taken from his parents and approved by Mr. B includes the following statements. He completed his elementary education in intermediate level, high school education in low/intermediate level and he was entitled to study at a university in his second year. It was stated that his grades were better in the first year of the university but his motivation decreased excessively since he did not get along with his teachers. It was determined that he has always left things unfinished through her life, changed his decisions constantly, had new ideas all the time but he either never started them or left them immediately even if he started, he does not have self control, does not have much enthusiasm for anything, does not go outside the house very often, does not have a definite time to go to bed or get up, he is messy, he treats as if he never listened what was spoken. He is unable to sustain attention, unable to learn anything completely. He smokes too much, He does not comply with his appointments. His affect and mood are euthymic; there is no perception disorder; his thought content is normal. With preliminary diagnosis of ADHD. Methylphenidate extended release 18 mg/day was started. It was gradually increased to 36 mg/day. In their psycho-education, the patient and his parent were informed about current symptoms and disorders. In his treatment; it has been observed that he feels energetic, he becomes to get sleepy spontaneously at nights and gets up with everybody in the mornings although he has not been recommended any regulation in his sleep –wake times, he has a controlled motivation and has started design business, is able to start and finish a task, he does not start another task/subject/agenda before finishing the one and thus his self-confidence starts to flourish again. To raise awareness of Attention deficit and hyperactivity disorder (ADHD) as an underdiagnosed, undertreated, and debilitating condition in adults, such as educational, social and family relationships and psychological and physical well-being. Adult with ADHD often experience chaotic lifestyles, with impaired educational and vocational achievement. The recognition of adult ADHD and effective treatment of adults' ADHD improves symptoms, emotional lability, and patient functioning.
Keywords: adult ADHD, functioning, education

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[Abstract:0401][Substance-related and addictive disorders]

Treatment of obsessive-compulsive symptoms in patients with opioid use disorder: case reports

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The relationship between obsessive-compulsive disorder (OCD) and substance use disorders is complex. An overlapping mechanism in OCD and substance use disorders has been proposed. OCD incidence in patients with opioid use disorder (OUD) is reported to be 4 times higher than the general population. In this case series, we present four patients diagnosed with both OUD and OCD and were treated with either buprenorphine-naloxone or naltrexone. The effects of these medications on their OCD symptoms were observed and discussed. Yale-Brown Obsessive Compulsive Scale (Y-BOCS) was administered on the first day of the hospitalization and repeated after 30 days.
Case 1: A 24 year-old male patient was included in the treatment program and was started on buprenorphine-naloxone 4-8 mg/day and mirtazapine 30 mg/day and continued with buprenorphine-naloxone 8 mg/day. Contamination and symmetry obsessions were detected and he was diagnosed with OCD according to DSM-5 and was put on sertraline 50 mg/day. His Y-BOCS score was 14 in the beginning of

the treatment program and 12 after 30 days. Sertraline was increased to 100 mg/day because of his remaining symptoms.
Case 2: A 25 year-old male patient was included in the treatment program and was started on buprenorphine-naloxone 4-8 mg/day and quetiapine 300 mg/day. He has suffered from religious obsessions for the past 2 years and was diagnosed with OCD. He was started on escitalopram 10 mg/day. His Y-BOCS score was 23 in the beginning of the treatment program and 21 after 30 days. Escitalopram was increased to 20 mg/day because of his remaining symptoms.
Case 3: A 22 year-old male patient was included in the detoxification program and was started on quetiapine 300 mg/day. Naltrexone (25 mg/day) was started on the 7th day. He demonstrated contamination obsessions along with cleaning compulsions, and was diagnosed with OCD at the age of 14. He was already on escitalopram (20 mg/day). His Y-BOCS score was 29 in the beginning of the treatment program and 24 after 30 days.
Case 4: A 22 year-old male patient was included in the detoxification program and was started on quetiapine 300 mg/daily. Naltrexone (25 mg/daily) was started on the 8th day. Having contamination and doubt obsessions along with control compulsions, he was diagnosed with OCD according to DSM-5 and was started on sertraline 50 mg/day. His Y-BOS scored 19 in the beginning of the treatment program and 13 after 30 days.
Recent studies indicate that the opioid system may have a role in OCD pathophysiology. Studies conducted on animals have revealed stereotypical behaviors. However, there are also studies showing significant and rapid improvement in OCD with opioid agonists such as morphine and tramadol. Likewise, increasing OCD symptoms after intravenous application of naloxone have also been reported. Contradictory to these findings, two of our patients had improved after the treatment of naloxone, an opioid antagonist. These findings may be attributed to a reduction of anxiety after quitting heroin use. In conclusion, the link between the opioid system and OCD should be considered when treating patients with OCD and OUD.
Keywords: substance, OCD, treatment

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[Abstract:0403][Others]

Is the problem in the sacral or in the unconscious?

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Persistent Genital Arousal Disorder(PGAD) is a rare disease which is characterized by uninvited, intrusive, unwelcome and distressing genital sensations. The origin of PGAD is yet unknown. Review of literature on Tarlov cysts indicated that they form characteristically at the S2 and S3 dorsal root ganglia. These roots convey the sensory pudendal and pelvic nerves, which innervate the external and internal genitalia. We introduce a case of PGAD who has Tarlov cysts on S2 and S3 location which may be related to the patient`s sexual symptoms in this case-report.
Case: A 43 year-old female patient presented with uninvited, intrusive, unwanted persistent genital sensation and genital arousal, similar to sexual arousal. This disease affected her quality of life in a negative way, as the patient felt irritated and disgusted. When the PGAD symptoms first started one half a years ago, she was on fluoxetine 40 mg per day regimen for the last three months because of her depressive symptoms. Her psychiatric examination at the time had not revealed any findings except depressive mood. We switched from fluoxetine to agomelatine 25 mg per day. Agomelatine was chosen because of lack of sexual side effects. IPT (Interpersonal Psychotherapy) was also done simultaneously with pharmacotherapy. Although her depression improved, her genital sensations and arousal continued. After adding Risperidone 0.5 mg per day to her treatment, patient's symptoms were improved relatively although they did not totally resolve. She had a history of back pain for the past twenty years which she described as a stabbing pain. She indicated that her genital sensation and arousal was aggravated depending on her posture, such as when driving a car or bending forward. After evaluation of these symptoms, we concluded that there was an underlying organic pathology. She had been examined by a gynecologist and a gynecological examination including pelvic ultrasound revealed no pathology. Spinal MRI scan revealed two sacral perineural cysts (Tarlov cysts) in relation to S2 and S3 in the sacral spine. The opinion of two different consultant neurosurgeons were that the patient's symptoms were not related to the Tarlov cysts because of the size and location of the cysts. They suggested that even if there was a causal relationship, symptoms of the patient was not an indication for a surgical treatment.
We reported a case of female patient both suffering from persistent genital arousal disorder (PGAD) and depression who has two Tarlov cysts on S2 and S3. Although her depression resolved after agomelatine, risperidone and IPT,her genital arousal symptoms not totally relieved. There is an association between sacral nerve root compression by spinal meningeal cysts and persistent genital arousal disorder

and it is reasonable to conclude that it is causal. Komisaruk and Lee suggested to rule out Tarlov cyst for patients suffering from PGAD. We want to introduce the treatment choice for our patient whether surgical treatment or psychologic treatment to help to improve her quality of life as a challenging topic.

Keywords: persistent genital arousal, tarlov cyst, depression

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[Abstract:0406][Others]

Developing myasthenia gravis patients using lithium management

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Bipolar disorder (BD),which,is a disease is progressing with relapse and remission perid and lead to loss of competence. lithium is usually disposed of BAD is an agent. It demonstrates accumulation in nerve cells. According to this some neorutoxic adverse effect could be seen. In this phenomenon, oversigting of myasthenia gravis which is progressing with a patient taking up with a BD diagnosis.

Case: A 42 year-old male patient,, married, consulted neurology department with complaining of ptozis, loss of strength and fatigue,that demonstrating rise in late afternoon. Patient who was diagnosed with BD, has a complaining of insomnia, was running away from home at the age of 15-16, had demonstrated recruitment with a treatment of haloperiodal and valproic acid (VPA). Patient that has complaining of insomnia, irritability has treated with lithium and quetiapine in a diagnosis of manic episode. In a recent year, to patient was observed loss of strength on lower extremity proximal muscles as ascending stairs, also fatigue and clarifid ptosis in late afternoon. Patient has consulted to us because of observing de novo of treatment of lithium as he is undergoing in patient treatment with pre-diagnosis at neurology department. Examination of mood chart during the consultation; he looks his age, has enough self-care. He is conscious, his tendency of place, subject and time axis were complement. Memory and and attention examination were typical. Mood was euthymic, sensation was anxious. There were no hallucination and delusion. Proceeding. cognisance and discrete were typical. He had insight.His rest and appetite were typical. Patient's examination, that was made in neurology department, in the monofilament EMG examination, orbicularis occuli and extensor digitorum communis muscles, persisted in typical limited in individual jitter value while average jitter was found upper on typical limit. It was suspicious for neuromuscular conduction anomaly. Coherent lesion with thymoma weren't determined in thorax BT. Patient was medicated pridostigmin bromur 60 mg QID, azatioprin 50 mg daily with pre-diagnosis of mysthenia gravis (MG) by neurology. Lithium 300 mg TID, used by patient for BD, gradually cut out and VPA treatment was mediated. Two months later check-up of psychiatry, patient's blood VPA degree was observed 45 and mood was eutymic. Also, at the neurology outpatient clinic follow-up exam, patient was in clinical remission and taking pridostigmin and azatiopirin treatment presently decided to proceed. MG, is characterized with antibodies that consist against nicotinic agonist in postsynaptic membrane, is an otoimmune disease that takes place in neuromuscular junction. If MG history was being thought, this diagnosis shouldn't be given up.Current lithium compete with calcium for rolling in to nerve ending at neuromuscular junction and decrease acetycholine synthesis and oscillation. Generally, there is regression in indications after cutting out lithium or decreasing of dosage. If the myasthenia gravis diagnosis get a patient who use lithium, it might not be enough that anticholinesterase like pyridostigmin is undertaken in treatment and it is important to know that decreasing of dosage or completely cutting of lithium is necessary.

Keywords: bipolar affective disorder, lithium, myasthenia gravis

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[Abstract:0407][Mood disorders]

Varenicline -induced depressive episode in a patient with bipolar disorder

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The onset of major depressive disorder with psychotic features in this patient after initiation of varenicline treatment for smoking cessation certainly suggests that varenicline has the capacity to induce depression and psychosis at least in patients with a history of Mood disorders. This has been reported in another patient with a documented history of bipolar disorder [Kohen and Kremen, 2007; Pumariega et al. 2008]. Possible mechanisms include dopaminergic stimulation secondary to agonism of the α4β2 nicotinic receptor. Since the approval of varenicline in May 2006, post marketing surveillance of it suggests an association between varenicline and increased risk of erratic behavior, agitation, suicidal attempt, depression, psychosis, and severe injuries [Williams et al. 2007]. Some of the behavioral changes and mood changes seen in patients who use varenicline may be associated with nicotine withdrawal. However, some occurred in people who continued smoking while they were on varenicline medication [Xi, 2010].
Case: A 56 year-old female patient with bipolar disrder diagnosis had a depressive episode with psychotic features after varenicline use. Although clinician and patient reports of adverse events associated with varenicline suggest the possibility of serious side effects, controlled studies are required to quantify the degree of risk, distinguish the side effects of varenicline from the effects of smoking cessation [Gunnell et al. 2009]. The risk for psychiatric side effects from varenicline could be greatly diminished by screening for family history and past history of serious mood disturbance in individuals who are candidates for its use in smoking cessation [Pumariega et al. 2008]. Smoking rates are particularly pronounced among persons with a history of anxiety, depression, bipolar disorder, and psychotic disorder [Ziedonis et al. 2008]. Providing effective cessation treatment to these individuals is important, but there are limited data on the effectiveness of cessation treatments among persons with these conditions [Hall and Prochaska, 2009]. There may be some explanations for the exacerbation of major depressive disorder with psychotic features in our patient. First, it is well known that increased dopaminergic activity in the brain plays a crucial role in the etiology of psychotic episodes seen in bipolar disorder [Cousins et al. 2009]. Varenicline, with its partial agonistic effect on nicotinergic receptors, stimulates the release of multiple neurotransmitters including dopamine [Benowitz, 2007]. It also increases the release of dopamine from nucleus accumbens. Dopamine dysregulation is probably responsible for the development of neuropsychiatric adverse reactions due to varenicline. Second, our patient was also taking lower doses of amisulpride for the last 3 years. Amisulpride, at low doses, has the potential to block presynaptic dopamine autoreceptors which consequently lead to the frontotemporal dopamine release [Scatton et al. 1997]. When considered together, our patient was under low-dose amisulpride treatment when he started varenicline treatment. Thus, one may think that both agents would have contributed to an increase in dopaminergic activity in the brain of our patient which biologically underlined the development of a depressive episode with psychotic features in our patient. This case report provides valuable support of previously published cases that demonstrate the risk of exacerbation of psychotic symptoms and depression with varenicline use in patients with severe mental illness. With proper assessment and management of varenicline-induced neuropsychiatric effects, healthcare professionals can provide an important role in helping to prevent and manage worsening psychiatric symptoms. We suggest close observation of patients with bipolar disorder on varenicline treatment.
Keywords: bipolar disorder, depression, depressive episode, mania, nicotine, varenicline

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[Abstract:0409][Mood disorders]

Lithium intoxication in a patient taking lithium for 20 years while increasing dosage

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Lithium is the most widely used drug in bipolar disorder and its dosage range is very narrow. This case report aimed to be careful while increasing the dose of lithium.
Case: A 53 year-old male patient who is married, retired, has left high school (for political reasons) and has two children. He has a 25 year

history of bipolar disorder and had been taking lithium for 20 years. He also diagnosed hepatitis B carrier and hipertension. He had been taking lithium with olanzapine 10 mg BID, quetiapine 100 mg daily, enalapril+nitredipin 10/20 mg daily. He was admitted to hospital for increased depressive symptoms one month ago. His treatment rearranged to lithium 300 mg TID from BID and his blood level of lithium were checked one week later. His lithium level measured as 0.7 mEq/L thereupon lithium dose is increased as 300 mg QID. After one week of this treatment, he suffered from nausea, vomiting, frequent urination and tremors on his hands. In the process of time loss of balance, speech impairment and perception difficulty are added of his symptoms. He presented to emergency service for these symptoms, and his lithium blood level is checked as 3.5 mEq/L. The patient spent 7 days in the intensive care unit and hemodialised two sessions. He had hematemesis on 2nd day after admission of service, then he had an endoscopy and his oral intake stopped. His confusion and orientation was recovered in 3 days and he began to meet his own needs. In this case report, hypertensive medicines and quickly increasing the dose of lithium and not checking blood level of lithium in short period may lead to lithium intoxication.
In conclusion, physicians should pay attention to adjust dose of lithium especially in older patients who uses ACEI, thiazide diuretics, potassium-sparing diuretics, methyldopa, phenylbutazone, indomethacin, naproxen and haloperidol.
Keywords: lithium intoxication, bipolar disorder, increasing dosage

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[Abstract:0410][Psychopharmacology]

Antipsychotic related hyponatremia: a case report

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Hyponatremia is an electrolyte abnormality that occurs when serum sodium levels decrease below 135 mEQ/lt. This condition is more common in psychiatric population with schizophrenia, particularly related to results of polidipsia. Many commonly used drugs including the antipsychotics, carbamazepine, selective serotonin reuptake inhibitors can cause hyponatremia. The frequently used atypical antipsychotics which including the risperidone, olanzapine and clozapine are most commonly associated with hyponatremia. We are presented to here, a schizophrenic patient whom entered into drug -induced hyponatremia during the increasing to drug dosage.
Case: A 33 year-old male patient, single, unemployed admitted to the psychiatry inpatient clinic by the parents for exacerbation of schizophrenia. He had diagnosis of schizophrenia for about nine years and his complaints were progressively increased, in last two months. During the admission, he took antipsychotics including the clozapine 300 mg per day and amisulpiride 800 mg per day treatment for schizophrenia. After then, he was hospitalized to adjust his drug doses. Drug dosage was gradually increasing on amisulpiride 1200 mg/d and clozapine 400 mg per day. In the next six days of increasing drug dosage, he developed dry mouth, excessive water drinking, and polyuria (3–4 L per day). Upon this, patient was consulted by the department of internal medicine. Laboratory investigations revealed that except for hyponatremia of 125 mEq/L, there were no abnormal findings such as, serum levels of thyroid hormones, and cortisols. Physical and neurological examination revealed normal vital signs and euvolemic status. We strictly struggled to reduce his fluid intake, during the inpatient treatment. Clozapine was decreased at the level of 350 mg per day. He was treated with intravenous NaCl 0.9% for her sodium deficit and with water restriction, less than 1.0 L daily, during the 5 days. After the 6 days, the serum sodium level recovered to 136 mmol/L. His symptoms of polydipsia were improved, paralleling the resolution of his hyponatremia. Also, his psychotic symptoms with aggression were gradually normalized after correction of hyponatremia.
Hyponatremia occurs in about 4% of patients with schizophrenia However, most patients with hyponatremia are asymptomatic. It is should remembered that, the development of clinical signs and symptoms also depends on the rapidity with which the plasma sodium level decreases. Many commonly used drugs can cause hyponatremia, either by dilution or by salt wasting (such as enemas). It should be noted that, hyponatremia may also be an adverse drug reaction. Nevertheless, it is generally opinion that antipsychotics (both typical and atypical) may be stimulate ADH release in the brain, although it has also been suggested that both typical and atypical antipsychotics induce severe polydipsia by stimulating the thirst center or by causing a dry mouth. It is probably wise, therefore, to correct the hyponatremia at less than the maximum rate in asymptomatic patients. This can be accomplished by fluid restriction. Also, close monitoring for clinical and laboratory evidence of these is essential. Psychiatrists should be aware of the possibility of hyponatremia associated with the use of antipsychotics.
Keywords: antipsychotics, hyponatremia schizophrenia

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[Abstract:0412][Substance-related and addictive disorders]

Pheniramine dependence: a case report

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Pheniramine is a first generation antihistaminic agent. It is used to treat hypersensitivity reactions and pruritus related to various conditions. Sedation, appetite stimulation, cognitive and sensoriomotor abnormalities are included to a large side-effects list. Available data suggest that due to the possible stimulating effects of antihistamines, pheniramine has the potential for abuse. There are a few cases of pheniramine dependence reported from Turkey. Here, we present the case of a patient with dependence to pheniramine.
Case: A 33 years-old female patient, single. She was working in health sector. She presented to the psychiatry clinic right after the suicidal attempt with 20 vials of pheniramine used intra-venously. In her personal history, in the past, she has seen some psychiatrists and her treatment consisted of occasional psychotropic drugs for her depressive symptoms for the last 6-7 years. She had been given the first dose of pheniramine by her pulmonologist for her severe allergic asthma attacks 7 months ago. She had noticed its sedative effects. She has started using it on her own for relaxing. She has gradually increased the dose up to 10 vials-a-day and has shortened the dose interval. Recently, she quit her job and moved to another city without ending her relationship. She was struggling with feelings of guilt at that time and soon she attempted suicide first time in her life with 20 vials of iv pheniramine. She experienced visual hallucinations and she called her family immediately. Her parents brought her to our clinic and she was convinced to be hospitalized. Venlafaxine 150 mg/day, diazepam 15 mg twice a day and 200 mg of quetiapine was started initially, 400 mg/day carbamazepine was a few days later added to her treatment. On the fourth day of hospitalization, the patient had intense craving for pheniramine, she was unrest, dysphoric and depressive, her hands were tremulous. All of the withdrawal symptoms subsided over the next two weeks; the dose of diazepam was gradually reduced and stopped in two weeks. She was discharged in a symptom-free state and without craving. She was seen three times after discharge. She said that she did not use it again and have no craving for it three months after, she dropped out of treatment.
Generally, antihistamines have a low potential for addiction when used in the recommended doses for a short time, yet first generation sedating antihistamines such as pheniramine, promethazine are known to be more likely to cause addiction particulary when used in higher doses for a long time. We have limited data on the prevalence of misuse of antihistamines. Lack of awareness of this addiction and easy access to this drug in our country make the treatment much more harder. Prescription drugs are amongst the most commonly abused category of drugs. Our case shows that it's judicious to keep in mind that the drugs prescribed by physicians with appropriate indications can also be abused. Particular attention to the personality traits as predisposing factors to addiction and being aware of potential risk for antihistamines misuse may help prevent antihistamines dependence.
Keywords: antihistaminic, dependence, pheniramine

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[Abstract:0413][Autism]

A child with Angelman syndrome and late diagnosis of comorbid autism spectrum disorder: importance of early diagnosis

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Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by social interaction, communication, field of interests and behaviors skewed from normal development. Although its etiology has not been fully elucidated, several studies have been conducted on genetic factors, infections, gastrointestinal system, metabolic factors and environmental factors. Autistic symptoms can be seen in many genetic disorders such as Fragile X syndrome, Angelman syndrome or tuberous sclerosis. Angleman syndrome is a neurogenetic disorder characterized by micro-deletion on chromosome 15q11-13 and maternal inheritance, which exhibits many behavioral characteristics of autism. Major signs included mental and growth retardation, marked speech disorder, epileptic seizures and

happy demeanor. It is also termed as "happy puppet syndrome" due to happy demeanor. Here, we presented a 10 year-old girl diagnosed as autism, who had been diagnosed as Angelman syndrome at 5 years of age.

Case: A 10 year-old girl was referred to our outpatient clinic with self-harming behavior, acute laughing and crying episodes by pediatric neurology department. In psychiatric examination, it was found out that she was diagnosed as Angelman syndrome at 5 years of age by identification of micro-deletion on chromosome 15 in genetic analysis and that she was referred to a special rehabilitation facility with mental retardation. It was also found that she presented to child psychiatry department for the first time and that she did not establish eye contact, and speak; that she had stereotypical movements such as hand clapping; that she had limited social communication and interaction in addition to failure to establish a relationship with peers; and that she had symptoms such as sweeping lips by her tongue, excessive movement, inattention and inability to maintain play activities. In addition, parents reported that she had self-harming behaviors, sudden and senseless laughter, unable to perform self-care activities and retarded perception and motor development when compared to peers. No additional test was ordered since relevant genetic, metabolic, electrophysiological tests and MR imaging studies were performed in pediatric neurology department. In history, there was no abnormal finding other than consanguinity between parents. The patient was considered as severe mental retardation and ASD. Special rehabilitation program and follow-up were recommended to the patient.

Chromosome 15 anomaly is frequently seen in patients with ASD. Interstitial duplication and deletion on chromosome 15q11-13 are identified in most cases. In this case, it is striking that ASB diagnosis was delayed for 5 years after diagnosis of Angelman syndrome at 5 years of age. It is thought that delayed diagnosis of ASD could be resulted from due to failure of referring the patient to child psychiatric for potential psychiatric problems in Angelman syndrome and idea that current psychological problems could be due to mental retardation in this case. Our case presented to emphasize that there may be an association of Angelman syndrome and ASD and psychiatric examination shouldn't be disregarded in order to make early diagnosis and provide appropriate treatment.

Keywords: Angelman syndrome, autism spectrum disorder, mental retardation

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[Abstract:0414][Psychopharmacology]

Risperidone related raynaud's phenomenon

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Raynaud's phenomenon (RP) is a recurrent vasospastic condition, characterized with white, blue or red discoloration of effected body part, due to cold or emotional stress. RP is classified as primary and secondary. Primary RP, in other terms Raynaud's Disease or idiopathic RP, is the term to define the absence of any etiological cause. Secondary RP is known as Raynaud's Syndrome, and this term is used to define an underlying pathology (like drugs) that causes RP. Here, an adolescent case that has RP during risperidone therapy will be discussed.

Case: A 12 year-old male presented to our clinic with his parents for the complaints of irritability and aggression. He was exhibiting aggressive behaviors such as destruction to property, or shouting at his family members. There has been an increase in these complaints by adolescence. No problem with attention in classroom was evident however he was reported not to study enough, therefore his academic performance was not good. We initiated risperidone 1 mg/day therapy for behavioral problems and impulse control. One month after starting risperidone, the patient communicated with our clinic, he was describing cyanosis following pallor in his right and left fingers. During the clinical evaluation, we observed cyanosis in right and left hands which was limited to the fingers, see figure 1. His Fingers were cold with palpation, we did not observe necrosis, ulceration and the patient did not describe pain. The patient was consulted to the general pediatrics clinic for further evaluation. Neurological and rheumatological examinations were normal. To examine RP etiology, common blood and urine count, routine biochemical tests, thyroid function tests, coagulation factors, sedimentation, CRP, rheumatoid factor, and antinuclear anticores were tested, and all of them were normal. We stopped risperidone therapy and suggested a periodic follow-up. Three weeks after stopping risperidone, the patient did not experience any pallor or cyanosis in fingers. Six months later, the patient reported he was free of RP symptoms. Therefore, we accepted the patient as a RP secondary to risperidone.

Pathophysiological mechanisms of RP are not clearly understood. Vasospasm of digital arterioles is thought to be a potential mechanism of primary RP. Endothelial damage and following perivascular changes could be resulting with arteriolar spasm. Spasm of arterioles as a response to alpha-beta adrenergic and serotonergic receptors is the other potential mechanism. The most common cause of secondary RP is connective tissue diseases. In addition, trauma, hematological, neurological, or arterial diseases, toxins, and drugs may also cause seconder RP. There are several drugs like bromocriptine, fluoxetine, citalopram, methylphenidate and dextroamphetamine were reported

to cause secondary RP. In this case, symptoms of RP started after initiating risperidone, and RP attacks stopped after discontinuation. We excluded other seconder causes of RP with anamnesis, physical examinations, and laboratory tests. Therefore, risperidone was the only potential cause for RP.

Keywords: raynaud's phenomenon, risperidone, child

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[Abstract:0415][Autism]

Coexistence of 9p deletion syndrome and autism spectrum disorder

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Deletions of the terminal portion of 9p are associated with monosomy 9p syndrome, which is characterized by mental retardation, development delay and characteristic craniofacial abnormalities (trigonocephaly, midface hypoplasia, hypertelorism, epicanthus, small palpebral fissures, flat nasal bridge, anteverted nares, low-set malformed posteriorly angulated ears, a long upper lip, and micrognathia). In addition, cardiac abnormalities, epilepsy, inguinal hernia, omphalocele, cryptorchism, and scoliosis can be seen in patients with 9p deletion syndrome. Autism spectrum disorders, including autism, are neurodevelopmental disorders characterized by impairments of social interactions, deficits in communication, and repetitive or stereotyped behaviors and restricted interests. Hereby, we aimed to present a rare case report of five year-old boy with 9p deletion syndrome and autism.

Case: A-five year-old boy presented to child and adolescent psychiatry clinic for the complaints of delayed speech and general development. He was operated because of inguinal hernia two years ago. He had a ventricular septal defect in his heart. Denver-II test showed delays in all developmental areas and, BERA test was normal. Common blood and biochemical tests were in normal range. Magnetic resonance imaging of the brain detected cortical atrophy. No abnormalities were reported in prenatal, natal, and postnatal history. Chromosome karyotype analysis revealed 46,XY,del(9)(p22) and, he was diagnosed with 9p deletion syndrome by medical genetics clinic. He had marked delays in basic motor skills, never begun speaking and has been receiving special education for two years. He had no eye contact, impairments in social interaction, and verbal and non-verbal communication, along with restricted, stereotyped interests and behaviors. During the clinical evaluation, we observed trigonocephaly, hypertelorism, flat nasal bridge, micrognathia, a long upper lip, and low-set malformed posteriorly angulated ears. The patient was diagnosed with autism spectrum disorder and moderate mental retardation accompanied with 9p deletion syndrome.

Chromosomal abnormalities are among the commonly recognized causes of autism and mental retardation. Microscopically detectable chromosomal abnormalities are found in 7.4% of autism spectrum disorders patients. Chromosomal abnormalities involving 9p have been shown in some children with autism. These chromosomal abnormalities include 9p copy number variations, an isolated 9p deletion, or inversion. The genes DOCK8, DMRT1, DMRT2 and DMRT3 from the area of 9p have been suggested to contribute to the autism spectrum disorder phenotype. In conclusion, this case presented to specify the coexistence of 9p deletion syndrome and autism spectrum disorders. Clinicians who take care of children with 9p deletion syndrome must be aware of autistic spectrum symptoms.

Keywords: 9p deletion syndrome, autism, child

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[Abstract:0417][Psychopharmacology]

Toxic hepatitis developed after oral and intravenous forms of haloperidol in a mentally retarded patient with diagnosis of autism and behavioral disorder

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Haloperidol is a typical type antipsychotic which is known and used for long years in recent years depot form is started to be used for various psychotic disorders. Extrapyramidal system side effects are most common side effects. However even as a rare adverse effect, we will present a case in which acute hepatitis developed after application of oral and intravenous forms of haloperidol in a mentally retarded patient with diagnosis of Autism and behavioral disorder.
Case: A21 year-old male patient presented to gastroenterology outpatient clinic with complaints of nausea, vomiting and loss of appetite. He declared that he had never experienced problems related to his liver or other organs before. His laboratory results was detected as AST: 2300, ALT: 6400, increased amylase enzyme. He was hospitalized with diagnosis of acute hepatitis. In the department of gastroenterolgy, psychiatry consultation was requested. According to the history taken from relatives, it is said that he has been on treatment and follow-up with diagnosis of autism and mental retardation since he was 5 years old. After adolescence period his behaviors had started to be worse as increased aggressiveness, improper behavior and decreased impulse control. He was on multiple medication. As his complaints had increased for 4 months and stayed responseless to medication; he was hospitalized to a psychiatry clinic. He was observed in service for 2 months. He was treated with haloperidol 10 mg/day, biperiden 2 mg/day and quetiapine 200 mg/day as oral form and also haloperidol depot as intramuscular form. During hospitalization period he had 9 doses of haloperidol depot. 15 days ago he was discharged. In psychiatric evaluation: He was conscious, partially oriented and partially cooperated. He had no eye contact. He was responding questions with short and meaningless answers. Psychomotor activity was increased. His mood and affect was anxious and irritable. He had disorganized and stereotypical movements. Also he had extrapyramidal system findings as dystonia and rigidity in extremities and neck; and also oculogyric crisis. According to history and psychiatric evaluation, haloperidol given in high doses and frequencies had thought to be responsible for acute increase in liver enzymes. That's why his psychiatric medication is changed as amisulpride 200 mg/day, lorazepam 2 mg/day and previous drugs are stopped. In follow up on 3rd and 7th days; EPS signs were observed to regress and also the level of the enzymes to decrease less than 1000 units.
Toxic hepatitis is a common pathological state in the range from mild biochemical abnormalities to acute hepatic failure. Major etiological factors are drugs, natural toxic agents and chemical agents. In this report we presented a case of toxic hepatitis after treatment of haloperidol. It should be kept in mind with treatment of haloperidol beside common EPS side effects, we might come across with conditions as toxic hepatitis.
Keywords: haloperidol depot, side effects, toxic hepatitis.

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[Abstract:0418][Psychopharmacology]

Emergence of manic episode as a result of the treatment of antiviral agent named forcarnet in a patient used after kidney transplantation

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Causes of Manic episode may be primary or secondary to medical illness or any medication etc. In this case we presented a patient with a first manic episode who had never experienced a manic episode before kidney transplantation and treatment of foscarnet-antiviral agent.
Case: A 37 year-old male patient, staying in nephrology ward had complaints of hiperactivity, increase in energy and libido, insomnia, irritability, decrease impulse control and frustration tolerance 3 days after the treatment of foscarnet. Psychiatry consultation was requested. According to the history taken from relatives and himself; 1 year ago he had kidney transplantation operation. He was hospitalized recently because of increase in levels of BUN and creatinine. His treatment was regulated and one of his drugs was changed with antiviral agent

celled forcarnet. They said that 3 days after this change he started to suffer from these symptoms. In psychiatric evaluation: He was conscious, oriented and partially cooperated. He was responding questions with quick, short and messy answers. His associations were loosened, speech rate was increased. Psychomotor activity, distractibility was increased. His mood was euphoric. He had grandious delusions. It is said that he had never experienced psychiatric complaints before. As psychological tests YMRS and MMSE was administered, found to be 30 and 25 respectively. His neurological examination was normal. In his physical examination apart from nephrological values; complete blood count, blood glucose level, liver function tests, tiroid hormone levels, levels of Vitamin B12 and folic acid were all in normal range. As psychiatric treatment medication of olanzapine 5 mg/day and lorazepam 2.5mg/day was regulated. He was planned to be observed daily.
Foscarnet is indicated for the treatment of acyclovir-resistant mucocutaneous HSV infections in immunocompromised patients. Common central nervous system (CNS) symptoms associated with foscarnet therapy are headache, tremor, irritability, seizures, and hallucinations. However in our case; different from these central nervous system side effects we observed mood changes pointing manic episode. We wanted to take attention to the point that; psychiatric disorders may be seen secondarily to treatment of medical disorders with steroids, immune suppressive agents and some antibiotics and also antiviral agents.
Keywords: foscarnet, antiviral, manic episode

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[Abstract:0420][Impulse control disorders]

Uromania: Is it type of a new impulse control disorder?

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Impulse control disorders characterized with the unable to resist an intense impulse, drive, or temptation to perform a particular act that is clearly harmful to self and/or others. Before the event, patient generally experiences increasing tension and arousal, sometimes—but not always—mingled with conscious anticipatory pleasure. Completing the event brings quickly gratification and relief. Within a variable time afterward, the patient feels a conflation of guilt, remorse, self-reproach and dread. These feelings may stem from obscure unconscious conflicts or awareness of the deed's impact on others. Shameful secretiveness about the repeated impulsive activity frequently expands to pervade the individual's entire life, mostly significantly delaying treatment. Impulse control disorders classified under "Disruptive, Impuls Control and Conduct Disorders". There are oppositional defiant disorder, conduct disorder, intermittent explosive disorder, kleptomania and pyromania. Impulse control disorder diagnosis is beyond being a special group, gives the impression of forcing a diagnosis has been included in the nosology. In this case we will present an impulse control disorder associated with drinking urine and rub the urine his own body, which is not previously described on the literature.
Case: A 20 year-old male patient, graduated from high school, single, unemployed. Patient brought by his friend because of drinking his own urine and rub the urine to his own body. Patient seemed in his own age, the dresser is good, self-care in place, temperament calm, sociability is sincere, speech and tone of voice normal, thought content focused on social issues without content pathology, flow rate of ideas was up to normal, and reaches the goal. There was not any hallucination or pseudo hallucination in perception. Psychomotor activity and social functioning was not impaired. Despite patient was known that urine is a dirty and useless thing, patient was feeling relieving when drinking and rubbing. Patient was feeling arousal in tention before doing these and was feeling tranquilized after. Despite of identify these behaviors "unusual, absurd and useless", patient was complaining not to stop himself. At first glance, the psychological examination of the patient suggested a psychotic disorder, but patient's affect was anxious and there was no evidence to suggest psychotic disorder. In addition intelligence was in normal range. Barratt Impulsiveness Scale (BIS-11) score of the patient was 73. Sertraline 50 mg/day and oxcarbazepine 600 mg/day were started. On the 8th week of treatment BIS-11 score was 57.
According to these findings, we may define a new impulse control disorder which may called "Uromania". The most important statement that should not be overlooked in similar cases is psychotic disorder. We believe that uromania could become clear with cases about this area. This case might be diagnosed as not other specified impulse control disorder according to DSM-IV. However, DSM-5 makes it impossible for this definition. Impulse control disorders were overshadowed disruptive and conduct disorders by DSM-5. It is no longer categorized under an disease group which further studies could be done. Although an attempt is being made to eliminate this problem by ICD-11, the size of debate is growing exponentially.
Keywords: impulse control disorders, uromania, new phenomenon

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[Abstract:0421][Psychopharmacology]

Paliperidone related acute hyperglycemia: a case report

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In most cases the mechanisms of hyperglycemia are not fully understood, although several possible theories have been proposed for each drug class. Some medications have risk of hyperglycemia, such as corticosteroids, beta-blockers, thiazide diuretics and some antipsychotic agents. Paliperidone is the 9-OH metabolite of risperidone and shows same antipsychotic activity in general. Metabolic side effects of atypical antipsychotic drugs is very high, and paliperidone was assessed at moderate risk for developing the metabolic syndrome. Here, the use of paliperidone after a patient developed hyperglycemia will be discussed.
Case: A 28 year-old male patient presented to our outpatient clinic with depression, loss of pleasure and fatigue. Psychiatric examination showed appearing older than age, poor self care and clothing, depressed facial expressions and gestures, slow speech, lower volume, less sleep, goal directed but slowed thoughts, worthlessness and inadequacy thoughts, retarded psychomotor activity and inadequate social functioning. According to these findings the patient was diagnosed with major depressive disorder. Sertraline 50 mg/day was started. Because of the presence of both nihilistic and guilt delusions, paliperidone 3 mg/day was added on the 1st month of treatment Because of patients mother ant father had DM, patient was in the risk group for DM. So that, we learned that almost before every breakfast whole family measures the fasting plasma glucose and weight. There was not any abnormality in fasting blood glucose levels of patient during treated with sertraline. Fasting blood glucose elevated 186 mg/dL on the second day of adding paliperidone 3 mg/day. Because of possible mistaken measures, test was repeated and the fasting glucose level was 183 mg/dl. Patient stopped taking paliperidone by himself because of belief that elevation of fasting blood glucose was a result of paliperidone. On the following measurements, fasting blood glucose level decreased pre-treatment level (80-90 mg/dL). Paliperidone 3 mg/day switched to aripiprazole 5 mg/day on the next visit of patient. There was any abnormality on fasting blood glucose level during the switched treatment.
Based on information we get from the literature, this is the first case of acute hyperglycemia status which caused by paliperidone. Metabolic side effects of atypical antipsychotics in general it is mentioned in long-term side effects. Hyperglycemia is the "component" of routine laboratory examinations for these side effects. As we showed in this case, hyperglycemia may also occurred solitary. Drug -induced hyperglycemia is a situation which can be seen in prediabetic patients. From the perspective of this case, it is ironically seen that the presence of DM in patient's parents were identified both risk and protective factor, the reason for former one is to be having a tendency of hyperglycemia, latter one is to lead opportunity to diagnose early. According to this case, usage of atypical antipsychotics may require strict follow ups about the presence of family history of diabetes. Based on this case, strict follow ups are needed to use paliperidone (or another possible atypical antipsychotic drugs) in patients who have family history of DM.
Keywords: paliperidone, side effect, hyperglycemia

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[Abstract:0422][ADHD]

The efficacy of atomoxetine on the symptoms of restless legs syndrome comorbid with ADHD: a case report

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Restless legs syndrome (RLS) is a circadian disorder characterized by focal akathisia accompanied by dysesthesia and motor unrest which is seen in extremities, especially in legs, while at rest. RLS may be idiopathic or symptomatic. An autosomal dominant genetic inheritance is mentioned in idiopathic form. In this group, the symptoms start at an earlier age and they are more resistant to treatment. The

pathophysiology of RLS is not known for sure. However, especially the dopaminergic system are thought to play a role in the development of the disease. Attention deficit hyperactivity disorder (ADHD) is frequently accompanied with sleep disorders such as obstructive sleep apnea, PLMS(Periodic limb movement disorder), RLS (Restless legs syndrome), and circadian rhythm disorder.
Case: A 9 year-old male patient was taken to our outpatient clinic by his mother. He had complaints of avoiding academic activities, not being able to continue the activities, untidiness, losing things, being very active, not obeying the rules of the game, talking too much and not being able to wait in the queue. Due to these symptoms, 7 year-old child was taken to the psychiatry polyclinic; however, the recommended medication was not used. In addition, the patient's anamnesis revealed that he had nocturnal dysesthesia and unrest symptoms in the legs; he felt a need to move his legs and thus had difficulty in falling asleep. Conners' parent rating scale-Short form (CPRS-48) and Conners' teacher rating scale-Short form (CTRS-48) were given. Laboratory analyses which included serum iron (112 mcg/dl), ferritin (75 ng/ml), and iron binding capacity (285 mcg/dl) were found to be normal. As a result of the assessment, the patient was diagnosed as ADHD (combined type) and RLS. Atomoxetine 0.8 mg/kg/day (28 mg/day) was given in ½ doses to the patient who was 35 kg. In the follow up, the dose was increased to 1 mg/kg/day (35 mg/day). In the second month of the treatment, interview for the diagnosis was renewed and it was seen that the symptoms had totally recovered.
The association between Atomoxetine and sleep has been shown in many studies. In a study conducted on animals in 2010, it was found that 5 day long atomoxetine use caused intermittent REM sleep and also a decrease was found in starts during the sleep of animals. In addition to these, a meta analysis conducted has reported lower rates of insomnia and somnolence. Our case is significant since it is the first in literature to present the positive effects of atomoxetine on RLS. The frequent comorbidity of ADHD and RLS, heritability and presence of common neurotransmitter in pathophysiology brings to mind the question whether these two illnesses are neurobiological subgroups. We have limited information on the effects of drugs used in the treatment of ADHD and neurotransmitter systems except dopamine on RLS. This subject can be a center of interest for future studies.
Keywords: atomoxetine, sleep disorders, restless legs syndrome, attention deficit hyperactivity disorder.

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[Abstract:0426][Psychopharmacology]

Methylphenidate and benign positional vertigo: a case report

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Methylphenidate (MPH), is a stimulant of Central Nervous System, is well known and widely used. MPH is used efficiently in cases like particularly Attention Deficit Hyperactivity Disorder (ADHD) and conduct disorder. The efficacy and safety of MPH are well known. And like all medicines, MPH can cause side effects, although not everybody gets them. Dizziness, that can appear in one out of every 10 patients who take MPH, said to be a common side effect. Brief episodes of mild to intense dizziness can be seen in benign positional vertigo. When you lie down, or when you turn over or sit up in bed trigger benign paroxysmal positional vertigo.
Case: A 17 year-old boy, who had dizziness after the start of treatment with methylphenidate and then who had diagnosed benign positional vertigo (BPV). He presented to the our clinic with complaints of failure in school, inability attention and often involved in fights. He was diagnosed ADHD with clinical interview on based DSM-5 and MPH 20 mg/day were prescribed. At the third day of methylphenidate treatment he presented to our clinic as an emergency. He had complaint of severe dizziness for two days after the beginning MPH treatment. Then MPH treatment stopped and he was monitorized. The day after there was no change about severity of dizziness. He said that when he stood up or was getting up out the bed, severity of dizziness was worse. He was consulted to neurologist and was diagnosed BPV. Side effects of drugs, which is even if common, can confuse picture of organic pathology. Clinicians should be aware of complaints of patients with drug therapy, which may be part of a disease. Making correctly differential diagnosis of the complaints will help patients to get the right therapy.
Keywords: benign positional vertigo, methylphenidate, dizziness

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[Abstract:0427][Non-biological treatment]

Oromandibular dystonia treated with Botilinum toxin: a rare case

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Dystonia is characterized by sustained muscle contractions, frequently causing repetitive twisting movements or abnormal postures, whereas the Oromandibular dystonia (OMD) is a disorder where repetitive or sustained spams of the masticatory muscles result in involuntary jaw opening, closing or a combination. OMD can be idiopathic (either focal or as part of generalized dystonia), or secondary to various medications, trauma, metabolic disorders or other neurologic movement disorders. Botulinum toxin (BT) is a neurotoxic protein produced by the bacterium Clostridium botulinum and related species. BT has been used in several medical problems with movement disorders, such as the focal dystonias, hemifacial spasm, tremors, tics, spasticity, strabismus, and also pain, tension headache, migraine, some autonomic disorders and sialorrhoea, since 1980. For OMD, BT injection has been shown to be superior to medical therapy.
Case: A 37 year-old male patient, married, unemployed with a past psychiatric history of schizophrenia, since 11 years. The patient had been maintained for 4 years on olanzapine 10 mg/day with in remission. Although, his complaints were progressively increased, in last 1 year. Therefore, olanzapine dose has been held on the level with 15 mg/day. Four days later, after the increasing doses of olanzapine 15 mg/day, the patient became difficulty with mastication and swallowing. He presented our outpatient department of psychiatry. Initial management has been to add the biperiden 4 mg/day; no change has been made to psychotropic medications. At the 15 days of this treatment, his paranoid symptoms was decreasing whereas the other symptoms such as, difficulty with mastication and swallowing were continued. Furthermore, the painful chewing with difficulty articulating was added. After then, biperiden doses was increased, at the 6 mg/day. One month later, his OMD symptoms were continued. Upon this, our patient was consulted in terms of the BT injection by the department of neurology. A diagnosis of peripherally induced OMD was made, which following increase of antipsychotic dose. They have treated our patient with BT injection into the pterygoid lateralis muscle. Each treatment session consisted of multiple injections into the pterygoid lateralis muscle. The injections in OMD patients were performed with at least a three-months interval, under the electromyographic (EMG) guidance. Consequently, he has responded excellently to BT injection, this treatment has been successfully repeated, about the 3 times in 9-month intervals.
Diagnosis of OMD is clinical but requires multiple investigations to rule out organic causes. Often idiopathic jaw opening dystonia may be misdiagnosed as dental problems, bruxism or temporo-mandibular joint disorders. Until the advent of BT therapy, systemic pharmacologic agents including the Anticholinergics (e.g. trihexyphenidyl, benztropine), benzodiazepines, baclofen, and tetrabenazine, have been the traditional mainstay of treatment for OMD. Recently, BT treatment continues to be the first choice treatment for most types of focal dystonia, including the OMD. As in our case report, patients suffering from focal dystonias require repeated injections to treat symptoms. As a result, clinicians should keep in mind that BT injection could be implemented for dystonia patients who had resistance of pharmacotherapy or can not get the drug due to the side effects.
Keywords: botulinum toxin, oromandibular dystonia, olanzapine

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[Abstract:0431][Schizophrenia and other psychotic disorders]

Lamotrigine augmentation in clozapine resistant schizophrenia: a case report

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However clozapine is effective in treatment resistant schizophrenia, it's known that between 40% to 70% of patients do not respond clozapine. Therefore, augmentation with other antipsychotics and anticonvulsants has been widely used. Lamotrigine is considered to be a potential treatment for schizophrenia because of its effects on glutamatergic neurotransmission. In several studies lamotrigine

and clozapine combination was observed to reduce symptoms in patients with treatment resistant schizophrenia. We report a case of reduction of persistent delusions after lamotrigine combination with clozapine treatment.
Case: A 24 year-old female patient was followed-up for 6 years with a diagnosis of treatment resistant schizophrenia. On admission, she had auditory and visual hallucinations, paranoid, reference, and persecutory delusions, thought withdrawal, broadcasting, thought insertion delusions, and affective dullness. Her treatment included aripirazole 30 mg/day and quetiapine 400 mg/day. We followed her with this treatment for 6 weeks but her symptoms did not reduce. Then we combined clozapine with aripiprazole and increased clozapine dose to 700 mg/day but she had partial response. After 12 week of clozapine therapy thought withdrawal/broadcasting/insertion delusions were persistant and BPRS score was 28, SANS score was 37, SAPS score was 36. So, we changed aripiprazole to lamotrigine. At the 4th week of lamotrigine 100 mg/day, clozapine 700 mg/day combination, delusions reduced, scores of BPRS, SANS and SAPS were reduced to 6, 30, and 7 respectively. The patient is being followed in a stable condition in the outpatient clinic follow-up exams for one year.
Although there are contrary reports in the literature, in the light of this case, we can speculate that lamotrigine augmentation in clozapine resistant schizophrenia is an effective treatment modality.
Keywords: clozapine, schizophrenia, lamotrigine

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[Abstract:0432][Eating disorders]

A treatment resistant binge eating disorder case report

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Binge Eating Disorder (BED), one of the three formal eating disorder diagnoses in the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), is defined by recurrent binge eating, marked distress about the binge eating and the absence of inappropriate weight compensatory behaviors that characterize bulimia nervosa. Recent community-based surveys suggest that BED occurs in 1-2% of children and adolescents between 10-19. In a study about impulse control disorders in obese patients, the participants with regular binge eating reported more depressive symptoms and significantly more often a history of any impulse control disorder.
Case: A 13 year-old girl who presented to Dokuz Eylul University Hospital Child and Adolescent Psychiatry Outpatient Unit with symptoms of binge eating, stealing money, skin picking and being depressed. Several investigation included MRI, hormone profile and further blood tests have been administered to the patient who has been hospitalized to make treatment arrangement. There haven't been abnormally results on tests. At hospitalization, her length has been sized 158 cm (50%), weight:109 kg (>97%), BMI:43,6 (>95%) and waist circumference: 110 cm (>97%). According to the history, binge eating episodes have been occurring especially in nights when nobody could notice, and therefore she has eluded choking hazard for three times. Therefore the mother has started to lock the kitchen door in nights. The client's behavior of stealing money has occurred at first grade when the mother and grandmother pushed her to go on a diet. Her complaint of skin picking and depressive mood continued for nearly two years have started after her father's suicide. The patient has presented a child and adolescent psychiatrist with these complaints and has taken fluoxetine, atomoxetine and oxcarbazepine for 6 months. Because she has not got benefit from fluoxetine 80 mg/day, she has been transferred to sertraline adding aripiprazole. She has been taking sertraline 125 mg/day and aripiprazole 2.5 mg/day at admission. The medication doses have been gradually increased to 200 mg/day for sertraline and 10 mg/day for aripiprazole. On progress, topiramate has been added and increased gradually to 300 mg/day. She has been consulted to the child endocrinology department and dietary modification has been recommended. She has been unable to adapt to the diet and continued to gain weight. She has been referred back to the child endocrinology department and topiramate has been stopped and metformin has been started and gradually increased to 2000 mg/day by their recommendation. The patient's weight gain has been continued during hospitalization for four months. At the discharge: her length has been measured as 158 cm (50%), weight: 117 kg (>97%), BMI:46.9 (>95%) and waist circumference: 112 cm (>97%).
We present a case with binge eating disorder who did not respond to two different SSRIs that both used at max dose and optimal period combined an effective and mild antipsychotic agent at optimal dose and period. It is needed more research about treatment of patients suffering from impulse control disorders and binge eating disorders.
Keywords: binge eating disorder, impulse control disorder, treatment resistance

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[Abstract:0433][Substance-related and addictive disorders]

Mescaline abuse via peyote cactus: the first case report in Turkey

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Mescaline, β-3,4,5-trimethoxy phenethylamine, is a psychoactive hallucinogenic alkaloid that resemble the natural neurotransmitters epinephrine and dopamine. It is extracted from peyote cactus (Lophophora williamsii), and a member of phenylalkylamines that include in DOM, 2C-B, 2C-E, 2C-T-2 and 2C-T-7. Peyote cactus is slow growing in the desert zone along Texas and Mexico zone and has been used for centuries by the natives of Mexico and Southwestern United States for ceremonial purposes, providing Mescaline in Turkey where is located far away this zone does not count much in common. Following the civil war in Syria 2011, large number of refugees from Syria where is the neighboring country immigrated to southern cities of Turkey. These people have intense interactions with young individuals living in Turkey, particularly for novelty seeking drug-addicted teens.

Case: An 18 year-old male patient. He was subjected to probation service due to use of cannabis for 3 months by the court decision, and being treated in our Forensic outpatient Clinic. He presented to the emergency room with symptoms such as intense anxiety, panic attacks and visual hallucinations. After the patient was getting stabilized, we performed a routine examination and asked for medical history. Patient told that he consumed peyote cactus instead using cannabis not to get caught in urine analyze. As he told, his friends were using this cactus with same motivation. He had started to use peyote cactus powder by bringing from Syria. He prior used to consume by brewing as tea or wrapping cigarette paper. Afterwards, he started to obtain via buying internet way. He then started to use peyote cactus by chewing. After the patient took treatment with intravenous fluids, we administered a single-dose coal and performed full bowel irrigation with PEG-ES (WBI). We used benzodiazepines to reduce anxiety. The patient was kept under close observation for 1 day, and then symptoms disappeared. Currently, his follow-up progress by our psychiatry clinic.

In this report, we presented the first case of Mescaline abuse via peyote cactus in Turkey. Currently, Mescaline has no psychological and physiological dependence. But, its euphoric effect and visual hallucinations attractes adolescent age group in Turkey. Numbers of mescaline user are getting increased because peyote cactus could not be detected via blood and urine samples in routine toxicology laboratories and it can be easily reachable over the Internet. This case presentation stated that Mescaline abuse via peyote cactus is a novel venture in Turkey, and Judicial authorities and physicians should give more attention to this subject.

Keywords: mescaline, peyote cactus, abuse, hallucinogen

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[Abstract:0437][Neuroscience: Neuroimaging-Genetic Biomarkers]

Is it schizoaffective disorder itself or cluster of symptoms caused by normal pressure hydrocephalus?

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Normal pressure hydrocephalus (NPH), also known as Hakim–Adams syndrome, was first described by Hakim and Adams in 1965 and is characterized by urinary incontinence, gait disturbance, potentially reversible dementia, associated with ventriculomegaly that usually presents with bradyphrenia and apathy. Apathy has been suggested to have associations with brain areas such as the anterior cingulate cortex (ACC), and thalamus, which constitute the frontal-subcortical circuits. Because hypoperfusion in the ACC and thalamus was found in iNPH (idiopathic NPH) patients, apathy in iNPH could arise from dysfunction in these regions. In the early stages, the cognitive profile is characterized mainly by impairments of attention, psychomotor speed and memory, suggesting frontal involvement; patients with more advanced iNPH show overall cognitive deterioration. The cognitive and behavioral disturbances accompanying iNPH have been commonly described as “fronto-subcortical dementia”, characterized by executive dysfunction,

psychomotor slowing and mood symptoms, especially apathy.
Case: A 64 year-old female patient, with 13 years of psychiatric history, diagnosed with bipolar and schizoaffective disorder, having a positive family psychiatric history, with her father and her son, both died of intracranial events.
It is already stated that patients with NPH may exhibit certain neuropsychiatric symptomatology, possibly related to alterations in central neurotransmitter activity. NPH patients may develop symptoms with frontal dominance, such as personality changes; anxiety; depression; psychotic syndromes, like delusional states, hallucinations and aggressive states; obsessive compulsive disorder; Othello syndrome; shoplifting and mania. Basal ganglia symptoms are exemplified by chorea. Thalamic symptoms may be exemplified by apathy and Othello syndrome, which is a content-specific delusion characterized by fixed false belief that one's partner has been or is being unfaithful. In some cases of NPH, dementia may be slow to develop and the psychiatric, particularly the affective symptoms may be the presenting picture. The development of psychiatric symptoms may be related to disturbed CSF circulation and ventricular enlargement. Moreover, the affective disorders associated with NPH may not be influenced by the shunting operations. Therefore, the affective symptoms in NPH cases need to be treated with medication and electroconvulsive therapy. This case demonstrates the need to consider NPH when older patients present with psychotic symptoms, particularly in the presence of cognitive impairment, gait disturbance, or incontinence. The dementia is characteristic of the subcortical type, with prominent frontal lobe features, including psychomotor retardation, amotivation, and apathy, resembling a picture of depression. Alongside the symptoms triad, we emphasize that apathy and bradyphrenia present a distinct quality of indifference and psychomotor slowing compared to actual depression. The correct differentiation of psychiatric and organic disorders is essential to avoid the development of severe neuropsychiatric conditions and proceed with the institution of appropriate treatment. Psychiatrists play a crucial role in recognizing this entity, initiating diagnostic investigations, managing symptoms, and referring patients to specialized neuropsychiatric assessment.
Keywords: apathy, Mood disorders, normal pressure hydrocephalus

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[Abstract:0440][Schizophrenia and other psychotic disorders]

Schizophrenia and EMDR, a case report

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Schizophrenia is a psychotic disorder that alters brain functions. Severe ruminations, delusions, hallucinations, and recognizable behavioral changes are main symptoms of schizophrenia. Some studies have found that psychosis, in particular schizophrenia are strongly related with childhood traumatic events. Eye Movement Desensitization Reprocessing (EMDR) is a therapy method, which was found by Shapiro in 1980s, specifically developed for the treatment of posttraumatic stress disorder (PTSD). In this case report, effectiveness of EMDR therapy was examined on a paranoid schizophrenia patient who has traumatic events, which are related to childhood sexual abuse and forced to hospitalization in a psychiatric clinic.
Case: A 43 year-old, female patient, architect having a daughter from divorced husband presented to our clinic with complaints of inability to sleep, suspicious, and thoughts of being followed to be murdered. Mental health examinations implied salient delusions and impaired insight. In addition, she had been treated as a patient with a diagnosis of schizophrenia since 2009. Furthermore, she had history of traumatic events such as childhood sexual abuse and being forced to hospitalization with police brutality for psychiatric residency. Therefore, two sessions of EMDR therapy is conducted on patient for both of the noted traumatic instances together with anti-psychotic medication treatment. Clinician Administered PTSD Scale (CAPS), Calgary Schizophrenic Depression Scale (CSDS), Brief Psychiatry Rating Scale (BPRS), Peri-traumatic Dissociation Scale, Beck Anxiety Inventory (BAI), Beck Depression Inventory (BDI), Impact of Events Scale-Revised (IES-R) which were administered before and after EMDR treatment indicated significantly decreased scores. On the other hand, EMDR subjective unit of disturbance (SUD) scores for the traumatic scenes were reduced.
EMDR therapy was administered for two sessions to the patient and a noticeable improvement was observed. Consideration of scale scores clarified that paranoid symptoms of schizophrenia substantially decreased, emotional vividness of the traumatic events (childhood abuse and being forced to hospitalization) lost its significant, and her sleep quality got better. Ruminations of the patient have remarkably reduced also; daily functionality of her has visibly got better.
In conclusion, EMDR may be a useful treatment option to help decrease severity of psychotic and schizophrenic symptoms. It can be

suggested that, psychotic and schizophrenic patients who have traumatic background might benefit from EMDR treatment.
Keywords: treatment outcome, schizophrenia/epidemiology, schizophrenia/rehabilitation, eye movement desensitization reprocessing/methods, schizophrenia/rehabilitation, stress disorders post-traumatic/epidemiology

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[Abstract:0441][Mood disorders]

Clozapine's effectiveness in treatment-resistant bipolar mania

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Literature data showing the effectiveness of clozapine for the treatment-resistant bipolar disorders has been growing recently. Niesen and colleagues reported that clozapine treatment decreases psychiatric admissions, additional psychotropic drug use, and suicidal attempts in bipolar disorder patients. These findings in the literature indicate the effectiveness of clozapine as a mood stabilizer.
Case: A 20 year-old female patient who had psychomotor agitation, intense irritability, grandiose delusions, and decreased need for sleep along with active suicidal ideas during her admission to emergency department. Moreover, she had experienced unhappiness, decreased mood, feeling of inadequacy, anhedonia, and psychomotor retardation for the last 2 weeks. Then, patient was admitted to the hospital with pre-diagnosis of mixed mania attack with psychotic features. This was second mania attack of the patient. She had her first mania attack at the age of 18 and was hospitalized. Thereafter, she has been followed with valproic acid 750 mg/day, quetiapine 900 mg/day, and olanzapine 10 mg/day as an outpatient for 2 years without any symptoms. Laboratory findings, cranial MRI, and EEG were normal and negative for any organic pathology. For the first week of her hospitalization, her acute treatment included haloperidol 10 mg/day IM, chlorpromazine 200 mg/day IM, and biperiden 10 mg/day IM. Following, the treatment was regulated as haloperidol 5 mg/day IM, chlorpromazine 100 mg/day IM, biperiden 5 mg/day IM, and quetiapine 300 mg/day. However, in the third day of this treatment, patient's reference and grandiose delusions and aggressive behaviors against her mother recurred. Hence, patient had the treatment of haloperidol 10 mg/day IM, chlorpromazine 200 mg/day IM, and biperiden 10 mg/day IM for one more week. Next, her treatment was changed gradually to risperidone 4 mg/day, quetiapine 900 mg/day, and biperiden 4 mg/day. However, aggressive and self-harming behaviors had continued despite the treatment. Following, the treatment gradually changed to aripiprazole 30 mg/day and quetiapine 100 mg/day. Because of unresponsiveness to treatment, electroconvulsive therapy was started on the 54th day of her hospitalization. She was given 11 sessions of ECT along with her medical treatment. Unfortunately, patient's depressive mood, aggressive behavior, psychomotor retardation, and social isolation behaviors continued despite of given treatment. On the hospitalization day of 97, clozapine 100 mg/day was added and quetiapine was removed from the treatment. Patient's psychotic symptoms, mood, and social interactions were markedly improved within first week of clozapine. Patient was discharged from the hospital with aripiprazole 30 mg/day and clozapine 100 mg/day after 2 weeks. No adverse effects was observed due to quick dose titration. Patient's functionality and social relationships improved significantly.
The knowledge about the effectiveness of clozapine in the treatment of psychotic mania and mixed mania attack has been enlarging in the current literature. Future studies investigating the effect of clozapine for the treatment-refractory bipolar disorder might be promising for the bipolar patient population.
Keywords: bipolar disorder, clozapine, treatment-resistant mania

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[Abstract:0442][Psychopharmacology]

A case of psychogenic polydipsia: treatment with aripiprazole

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Polydipsia was defined as the consumption of 3 or more liters of fluid per day. The prevalence of polydipsia among psychiatric patients is 6-17%, schizophrenia is 80% of these patients. Here in we present a schizophrenia case with polydipsia and psychotic relapse that was treated with aripiprazole.
This 48 year-old single female patient was diagnosed with schizophrenia who had received risperidone 2 mg/d for ten years. She presented auditory hallucinations, insomnia for two months. Polydipsia (approximately 5 L/d) was noted by relatives of patient. The patient did not give any reason for polydipsia but she described that request to drink water. She had no history of another medical illness, head trauma or surgery. A complete blood count, serum electrolyte, kidney, liver, thyroid function tests, physical examination and magnetic resonance imaging of brain were normal. We did not do a water deprivation test because of her water intake was not supervised by relatives of patient. Based on her history and laboratory tests; we diagnosed psychogenic polydipsia. Risperidone was switched to aripiprazole, and gradually titrated to 10 mg/d on the 20th day. After 20 days, she showed improvement in her symptoms and she had reduction in her water intake (2 L/d) in last 5 days. Laboratory tests were normal. The underlying pathophysiology of psychogenic polydipsia is unclear; studies revealed that the patients with secreted excess antidiuretic hormone (ADH). Inappropriate antidiuretic hormone secretion was followed by relapse with further psychosis. In addition, a hyperdopaminergic state is associated with polydipsia. Involuntary fluid restriction may be sufficient to treatment but some patients have difficulty in adapting to the fluid restrictions such as our patient. Various pharmacological agents,such as demeclocycline, propranolol, naloxone, clonidine and enalapril have been used for the treatment of psychogenic polydipsia. ECT has been suggested to be effective in cases with psychosis. However Antipsychotics role is not clear; clozapine appears to be effective for polydipsia. Efficacy of risperidone on polydipsia is controversial, positive and negative results have been reported. Here we present a schizophrenia case who had received risperidone developed psychogenic polydipsia and responded well aripiprazole. The improvement in polydipsia can be related to the aripiprazole's mechanism of action is different from other atypical antipsychotics;it acts as a D2 partial agonist. There are no case reported in the literature about the aripiprazole for use of treatment psychogenic polydipsia. Future studies need to aripiprazole which should be considered as an alternative in the treatment of psychogenic polydipsia.
Keywords: polydipsia,treatment,aripiprazole

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[Abstract:0444][Others]

Tuberculous meningoencephalitis -induced Kluver-Bucy: a case report

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The syndrom of Kluver-Bucy, includes a series of emotional and behavioral changes observed after removing bilateral temporal lobes in primates in 1930s. These changes are psychic blindness (visual agnosia), hypersexuality, hyperorality (eating large amounts or unusual objects), hypermetamorphosis (interest grown with each object within the field of view) and emotional changes. The diagnose is made with at least 3 of these symptoms. Aphasia, amnesia and dementia may be accompanied. In literature, several clinical case reports with different combinations occurring due to various causes such as herpes simplex encephalitis, head trauma, epilepsy surgery, cerebrovascular disease have been reported.
Case: A 64 year-old, married male patient. He is living with his wife in Konya. The patient, whose first complaints had began 15 years ago, presented to our clinic for the first time two months ago. By that time no systematic and psychiatric disorders were known. He had been presented to Ege University School of Medicine hospital with a febrile infection and sudden changes in consciousness. As a result of the investigation he had been diagnosed with tuberculosis meningoencephalitis and followed about five weeks in the department of infectious diseases. When he was in hospital he had losed orientation to place, time and persons, and also had had emotional instability

with inappropriate laughing and crying. After discharge from the hospital the caregivers observed him that speaking by the same words repeatedly, can not learn new information, can not stop eating and smoking. He had received some psychiatric treatment due to the agitated and hipersexuel behavior during this period. He had partial benefit from the use of sodium valproate and olanzapine. He had not used any medication during last year. Because of inappropriate sexual behavior, insomnia, shouting and swearing, he was brought to our clinic by his family. In the mental status examination, he was reported as limited in orientation, deficits in attention and memory, notable anterograde amnesia, perseverative at speech, poor thought content, disturbed sleep pattern and increased appetite. He had no insight into his behaviors and amnesia. MR imaging showed severe cystic encephalomalacic changes in bilateral temporal lobe stated cortical and subcortical and also changes compatible with gliosis around the area. Paroxysmal EEG abnormalities was found in the right hemisphere frontal lobe. The mini mental test scores were identified 18/30. His performance was impaired in the late part of Burdon attention test. He performed at the border level in Benton visual memory test. Clock Drawing Test showed no impairment in planning and configuration areas.
Perseverative speech, anterograde amnesia, hyperorality, hypersexuality, emotional changes are present in the clinic of this case. It is reported that the disinhibited behavior was decreased partially with antiepileptic and antipsychotic medication. It is also reported that the efficiency of treatment was increased after the addition of SSRI.
Keywords: kluver- bucy, temporal lobe, tuberculous meningoencephalitis

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[Abstract:0446][Mood disorders]

Hypomania induced by hyponatremia

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Hyponatremia is a common and important condition that may present with psychiatric and neurological symptoms. Here we report a case of hyponatremia presented with hypomanic symptoms.
Case: A 47 year-old female patient presented to endocrinology outpatient clinic with complaints of high blood glucose levels, polydypsia and polyuria. Her blood glucose: 199 mg/dL,serum sodium: 131 mmol/dL and HBA1C: 10.1. She was admitted to endocrinology inpatient clinic for regulation of antidiabetic treatment with a diagnosis of type 2 diabetes mellitus. She was taking insulin treatment for 11 years. During her hospitilisation insulin doses were regulated. On the eighth day of hospitalization serum sodium: 119 mmol/dL, BUN: 53.9 mg/dL, creatinine: 0.84 mg/dL and she started to sleep just a few hours and had no more need for sleeping. In the following days increased talkativeness, irrational thoughts such as she was a holy person or she had some important tasks, refusing the treatment and euphoria symptoms were added to her clinical presentation. At this time hyponatremia was persisted although iv saline infusion. On the forth day of sleeplessness, she was consulted to psychiatry. It was very difficult to cooperate with her, she had a nonstop speech, moving consistently, had some irrational thoughts about her present illness. Also she had a profound desire to be discharged from hospital despite all explanations. Her serum sodium: 123 mmol/dL, blood glucose: 157 mg/dL. She had no history of psychiatric disease and psychotropic drug use, also did not have any family history of psychiatric disease. We made a diagnoses of organic affective disorder with hypomanic episode secondary to hyponatremia. Zuclopenthixol 6 mg /day treatment was started. But she was discharged from hospital with her own decision and did not take zuclopenthixol treatment. Because her psychiatric symptoms persisted she presented to emergency room 2 days later. She was consulted to psychiatry. Her symptoms were more or less the same and also hyponatremia was persisted. Haloperidol 5 mg intramuscular injection was administered in the emergency room. After sedation of her, she was discharged from emergency room with suggestions of zuclopenthixol 6 mg/day treatment and psychiatry outpatient clinic follow-up 2 days later. In the control examination she had no more psychiatric symptoms, she was euthymic, her speech was normal and slept enough. Zuclopenthixol treatment was continued at the same dose for 2 weeks. At the end of 2 weeks she had no more affective symptoms. Just she complained about difficulty on falling asleep. Also she had minimal extrapyramidal system findings. Serum sodium was 136 mmol/dL, blood glucose was 186 mg/dL. Zuclopenthixol treatment was stopped and quetiapine 25 mg treatment was recommended even if she had a problem to fall asleep.
To our knowledge there is only one case report related to hyponatremia -induced hypomania in the literature and our report is the second one. It is important to be cautious about psychiatric symptoms of hyponatremia. When psychiatric symptoms are emerged, main treatment is to correct the hyponatremia itself, but also antipsychotics and benzodiazepines may be used to control the psychiatric symptoms.
Keywords: hypomania, hyponatremia, organic affective disorder

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[Abstract:0455][Anxiety disorders]

Visual hallucinations developed in the course of alprazolam administration: a case report

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Benzodiazepines have a more rapid onset of action than antidepressants, but their long term use is not recommended because of their potential for dependence and lack of antidepressant properties. Alprazolam is a drug used as a anxiolytic and hypnotic. Seizures, hallucinations, and depersonalization have been reported in less than 1% of patients. Amnesia, psychomotor impairment, anterograde memory loss and ataxia have also been reported. In this article, we present the association between alprazolam and developing of psychotic symptoms with a case after initiation of alprazolam as an add-on treatment.

Case: A 32 year-old female patient presented with an sudden onset illness, with a precipitating factor of her nephew's traffic accident, complaining of severe anxiety. She had multiple unpredictable, spontaneous episodes starting suddenly, severity reaching to its peak after about 10 min and characterized by fear of dying, pounding heart, apprehension, difficulty in breathing, abdominal distress, dizziness and sweating. Each episode would last up to two hours, followed by hospital emergency room visits and given medication included diazepam intramuscular or intravenous injections. Symptoms started one year ago in frequency of four or more episodes per week and had worsened in the past 3 months. There was no family history of significant mental illnesses. The patient had no history suggestive of illicit drug or alcohol abuse, head injury, significant medical illness except for regular monitoring of asthma or other psychiatric illness. She presented to a neurology clinic at first, her complete blood count, renal function, thyroid function and liver function tests, electrocardiogram (EKG), electroencephalography (EEG) was normal. Due to the patient's anxiety symptoms and agoraphobia, the brain magnetic resonance imaging (MRI) was not performed. For the treatment of symptoms, paroxetine 20 mg/day was started and the dosage of paroxetine was increased gradually to 30 mg/day in a month. At the same time, upon the continuation of anxiety complaints, alprazolam 0.5 mg/day per oral was added to the treatment. After 2 days, with alprazolam adding, she described psychotic symptoms included visual hallucinations in the form of light flashes, shapes of creatures and her symptoms worsened despite good compliance and no other psychosocial reason for worsening. She was referred to our psychiatry clinic for evaluation of changes that psychotic symptoms. Inourclinic, alprazolam was immediately stopped. She rapidly improved and over the following 2 days there was complete resolution of her psychotic symptoms.

Hallucinations rarely occur in individuals with anxiety disorders. This present case report underscores the fact that visual hallucinations are not always indicators of a psychotic disorder. Visual hallucinations have numerous etiologies and they may be related with the medication. The case reported here presented with severe symptoms of anxiety and concurrent visual hallucinations after an add-on treatment. Based on these findings, we assumed that the hallucinations were associated with the anxiety disorder treatment. Moreover, detailed analyses of such unusual cases can help improve our understanding of the pathogenesis of psychotic-like symptoms in anxiety disorders.

Keywords: alprazolam, psychotic-like symptoms, visual hallucinations

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[Abstract:0456][Impulse control disorders]

Trichotillomania in elderly: a case report

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Trichotillomania characterized by recurrent chronic hair pulling is an impulse control disorder which is associated with comorbid conditions. In most cases trichotillomania results in a total or partial scalp alopecia. Dermatology is of special importance in consultation-liaison practice. In fact, it has been already accepted that there is a need for association between psychiatry and dermatology in many cases. The psychiatric comorbidity is usually seen with trichotillomania in adults. The most common psychiatric comorbidities are affective disorders, anxiety disorders. The emergence of trichotillomania is generally in childhood and adolescence and it is generally diagnosed in females. Once presumed to be an obscure condition, the estimated source for lifetime prevalence is 1.5% for male and 3.4% for female

college students. Because of the secrecy and shame about their behavior, many remain silent sufferers, and treatment is often delayed. Trichotillomania is rare in the elderly.
Case: A 66 year-old female who had complaints of irritability, restlessness, anger, demoralization, thoughts of suicide, insomnia and a 3 month history of a large, irregular area of hair loss over the scalp is presented in this case report. The our patient was evaluated as very late onset trichotillomania. Physical examination revealed scattered short hairs of varying length, follicular hyperkeratosis and hyperpigmentation throughout the area of alopecia. Rarity of late-onset trichotillomania in old age is the significant aspect of the case.
Furthermore, this study discusses the benefits and limitations of such a short treatment for comorbid trichotillomania and major depressive disorder while positing the relationship between the two disorders. Taking notice of items about diagnosing and treating in this kind of patients are discussed in this case report.
Key words: Trichotillomania, very late onset, comorbidity

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[Abstract:0462][Schizophrenia and other psychotic disorders]

Suicide attempts of male case with early-onset schizophrenia and juvenile Huntigton disease

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Huntington disease (HD) is an autosomal dominant neurodegenerative disease developing with movement disorders such as chorea dystonia, and cognitive decline and psychiatric disorders. HD beginning before the age of 21 is defined as juvenile HD and is observed in 0.5-2% of all HD cases. Studies have reported that suicide attempts are 8 times more likely in HD patients compared to the general population, with up to 20% having suicidal thoughts and attempts. In the literature there are many case reports of HD accompanied by psychotic findings. However all of these cases are older. This report is about a HD patient with psychosis beginning in the childhood period and repeated suicide attempts. To the best of our knowledge, no case of pediatric HD and accompanying psychosis has been reported.
Case: A 16.5 year-old male patient. In his whole body, but especially extremities, rough, repetitive, irregular, uncontrolled involuntary choreoathetotic movements were present. These movements had occurred in the last year and severity had increased in recent months. Walking was disrupted and he experienced frequent falls. His school success had begun to decline. His writing was worsening. He was unable to attend school. He was quick to anger, displayed damaging behavior to himself and those around him and had attacks of aggressiveness. In recent months his speaking was disrupted and he was difficult to understand. Obsessive compulsive symptoms such as hand washing, cleaning, controlling and habits were present. He was unhappy, introverted and had reduced self-care. In the last 6 months he had attempted suicide by jumping from height and swallowing a spoon. He was operated twice for swallowing a spoon. In the previous month he had attempted to jump from a height and fell together with his father who was trying to hold him back. While he broke his arm, his father died. In the last six months audible hallucinations with ordering style demanding suicide and telling him to swallow spoons had occurred. Family history showed father, paternal aunt and uncle had HD. Cranial MR found atrophy in accordance with HD in bilateral caudate nucleus and putamens. Genetic testing found repeated CAG.
When clinical observation and examination results are evaluated with laboratory findings of the patient,
HD accompanied by psychiatric symptoms was identified. These psychiatric findings were in accordance with previous case reports and research results. However, there was no case reports of psychosis and suicide attempts in juvenile HD beginning at a young age encountered in the literature. There are case reports showing successful treatment of HD accompanied by psychosis with risperidone and quetiapine. Accordingly the patient was begun on 3 mg/day risperidone and 100 mg/day quetiapine. With mild depressive symptoms and repeated suicide attempts, 20 mg/day escitalopram was added. We believe this case is important in being the first in the literature of movement disorder accompanied by early onset schizophrenia in pediatric and adolescent psychiatry.
Keywords: suicide, schizophrenia, juvenile huntington disease

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[Abstract:0464][Autism]

Kabuki syndrome accompained by autistic findings: a case report

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Kabuki syndrome (KS) is a rarely-observed syndrome with multiple congenital abnormalities and mental retardation. Diagnosis is based on observation of five cardinal symptoms determined by clinical observations: (1) The strange facial appearance unique to the syndrome, (2) mild to moderate mental retardation, (3) skeletal anomalies, (4) dermatoglyphic anomalies, and (5) postnatal growth retardation. The cause in the majority of patients is identified as later mutations occurring in the KMT2D gene. KMT2D and KDM6A genes code for histone modifying proteins. This report presents a KS case aged 11 years 9 months with autistic symptoms.

Case: A 11 year and 9 month old male patient was brought to the clinic in a wheelchair. Dysmorphic facial characteristics included strabismus, hypertelorism, sparse, scattered and high eyebrows, long palpebral fissure, clear large and forward ears, clear flattening of the nasal bridge, wide forehead, micrognathia, open mouth and drooling, lower eyelid extroversion more prominent at younger ages. However, with support the patient could walk with tenuous small steps. There was clear asymmetric appearance of the back and costovertebral deformity. Thoracolumbar scoliosis was present. Slightly atrophic appearance of lower gastrocnemius muscles below the knees. Chest cage was slightly hollow toward the front. Fingers on both hands were short. There was clear shortness of the 5th fingers. The finger pads appeared swollen. Muscle tone was slightly weak. Pathological reflexes were not taken. There was no vision or hearing loss. The patient had difficulty swallowing solid food (dysphagia). Psychiatric examination found the patient did not speak. Communication could not be provided. Eye contact could not be made. He was in his own world. Moderate levels of metal limitation were present. Psychometric tests identified IQ points of about 40. On the childhood autism rating scale (CARS) advanced autistic findings were found with 38 points.

The patient's facial appearance, skeletal system abnormalities, mental retardation and psychomotor development retardation were in accordance with KS. When Niikawa and Kuroki first described this syndrome the characteristic facial appearance was similar to makeup used in traditional Japanese Kabuki theatre and so they gave the name kabuki makeup syndrome. This facial appearance includes long palpebral fissure, extraversion of the lateral one third of the lower eyelids, eyebrows arched, scattered and sparse, prominent ears and flattened nose. Skeletal anomalies include rib and vertebral anomalies, short (brachydactyly) or crooked (clinodactyly) fingers, observed in 80% of cases.

The diagnosis of the patient presented in this case was supported by scoliosis and brachydactyly. Nearly all KS are found to have swelling of the fingertip pads. In our patient similar appearance of the fingertips was present. It is reported that KS may be accompanied by findings similar to autistic disorders. This case never learned to talk, had limited communication, was introverted, did not make eye contact and had stereotypical movements such as swaying back and forth evaluated as autistic findings. To the best of our knowledge this case report is the fifth KS case in Turkish children and is the only case with accompanying autism reported.

Keywords: kabuki syndrome, autism, mental retardation

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[Abstract:0465][Others]

Mentally retarded adolescent with holmes-schimke syndrome: a case report

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Inherited MR can be classified as non-syndromal (nonspecific) type in patients where intellectual insufficiency is the only symptom of disease, or as a syndromal type occurring with phenotypic appearance of a more holistic syndrome. In 1989 Holmes-Schimke was described as an unknown syndrome involving mental retardation, microcephaly, congenital heart disease, skeletal abnormalities, micropenis and mild hypothyroidism in two brothers. In this report we aim to present a 16 year-old severe MR case fulfilling criteria for Holmes-Schimke syndrome to remind clinicians of this rarely observed syndrome and other similar syndromes.

Case: A 16 year old male patient with dysmorphic appearance, moderate-severe mental retardation (MR) and hypothyroidism. Physical and psychiatric examination of the patient found obese appearance, hyperactivity, limited vocabulary and speaking without full pronunciation

of sounds. Additionally physical appearance noted low hair line, irregular and gappy teeth, high palate, large-prominent ears, hypertelorism, hypoplasia of nostrils, retromicrognathia, fusiform and short fingers, polydactyly operation scars on both hands and feet and pes planus in feet. According to anamnesis obtained from family though he had no problems walking, seeing and hearing, he was in need of absolute care as he required constant guidance, occasionally became upset and shouted, even becoming aggressive at times. After feedback from the mother, examination confirmed micropenis and 2 nipples on the left side. The patient's appetite was large (hyperphagia) and no abnormalities were described in his sleeping habits. During the interview limited communication with the patient was possible, with another important point that he had epileptic seizures learned from the family. Previous history showed mother and father were cousins. The patient was the first pregnancy for the 31 year-old father and 29 year-old mother, born at term at 3700 g by C/S. No problem was encountered during birth. After birth he received two days phototherapy due to jaundice. The baby was identified as hypotonic. Psychomotor development was delayed. At the 4th month, 10% relative microcephaly was found. The patient began to experience epileptic seizures when he was 2 year-old and seizures were controlled by valproic acid treatment for 8 years. Additionally diagnosed as hypothyroidic at 6 years of age, the patient continued to receive thyroid hormone replacement treatment. At birth atrial-septal defect (ASD) was identified and the lesion spontaneously closed at 4 years of age. Tests found sharp waves on EEG, with current head circumference 3% below normal, in other words microcephalic. The case in this report meets the characteristics of Holmes and Schimke syndrome, together with similarities to clear ASD (autism). Additionally there are some slight differences. Facial dysmorphia and obesity were very clear. Perhaps in the brothers first reported with Holmes-Schimke may have had obesity and more definite hypothyroidism in advancing years; however no follow-up reports were found. Though rarely observed, if clinicians are aware of these syndromes, it may prevent these types of cases being overlooked.
Keywords: holmes-schimke syndrome, mental retardation, skeletal abnormalities, micropenis

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[Abstract:0468][Eating disorders]

Chocking phobia treatment with low dose haloperidol: a case report

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Chocking phobia is characterized by the extreme fear about swallowing and avoidance of eating, swallowing solids and/or liquids. During childhood, vomiting and painful traumatic experiences can cause maladaptive eating behavior. These patients reject eating solid foods and may lose weight as a result of this condition. Choking phobia was involved in the new diagnostic category of avoidant/restrictive food intake disorder (ARFID), in the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5). This disorder is different from other eating disorders, but occasionally chocking phobia patients have been misdiagnosed with anorexia nervosa. Here, we report a pediatric patient who began refusing food after the death of her grandfather, and was treated successfully with haloperidol and behavioral intervention.
Case: A 8 year-old female patient presented to Child and Adolescent Outpatient Clinic with the complaints of eating refusal and death fear. Her symptoms began to show after the death of her grandfather, in January 2015. While she was fed normally until 2 months ago, she abruptly began to eat only liquid foods and formulas since the death of her grandfather. According to her parents, she feared choking and she had heart palpitations while she was eating something. She thinks that "If I chock any food, I can aspirate and I can die". Her prenatal, perinatal and postnatal history was normal. She had surgery of adenotonsillectomy about one year ago. Her routine blood analysis and ECG were normal. When the patient was asked for making a daily list of the food she ate, and the list consisted of only milk and formula, she was diagnosed with choking phobia. Haloperidol was started 0.5 mg/day and intervention was administered against her fears. In this process, some behavioral intervention such as motivation and exposure homework was given at certain day of the weeks. Amount of the solid food was increased gradually, in progress. At the third week of haloperidol treatment, her symptoms were fully resolved. By the end of the two-month treatment of haloperidol and a three-month follow up, she could eat solid, liquid and any other forms of food without any anxiety reactions.
There is no standard treatment protocol for chocking phobia in literature. The Selective Serotonin Reuptake Inhibitors and behavioral interventions were mentioned as effective treatment strategies in some case reports. In this case, we firstly used haloperidol in order to reduce anxiety, and then, we practiced behavioral interventions with patient. After a while, significant decrease in anxiety about swallowing was seen, and patient began to be fed with solid food. We concluded that low-dose haloperidol as an anxiolytic agent may be an effective treatment in children with chocking phobia. Further research about the efficacy of haloperidol in this age group is needed.
Keywords: behavioral intervention, chocking phobia, haloperidol

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[Abstract:0470][Substance-related and addictive disorders]

Binge drinking of alcohol and psychogenic polydipsia co-occurency: a case report

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Psychogenic polydipsia (PPD) is a disorder that leads to substantial morbidity and mortality and an increase in health service utilization. It occurs in psychiatric patients and is more likely to be seen in schizophrenics. It is also common in middle-aged women who have anxiety disorders. PPD is characterized by low plasma sodium in 10 percent to 20 percent of those presenting with compulsive drinking. Symptoms are infrequent unless patients continue to drink excessively after they reach their limit of urine dilution Binge drinking is defined as a pattern of drinking that brings blood alcohol concentration levels to 80 mg/dl. This typically occurs after four drinks for women over the course of about two hours. The DSM-5 defines mild, moderate and severe alcohol use disorders according to severity. This case report is about a patient who has intermittent periods of binge alcohol consumption (BAC) and PPD.

Case: A 31 year-old married female patient who has two children presented to our addiction policlinic because she complained of an alcohol problem. According to information she provided, at the age of 20, her alcoholic drink consumption was five drinks at least three times per week, and that rate of consumption continued for approximately 10 years. It has been changed every 3-4 weeks 10 to 17 standard drink since two years. Her alcohol consumption continued at the same pattern throughout her two pregnancies. The patient has been drinking water more than 10 litre per day for the past 10 years, but did not have any complaints related to her water consumption. Her blood sodium and urine creatinine were determined to be low. This data shows that she did force diuresis. Further testing demonstrated that there was no endocrine disturbance in this case. The patient's history revealed that she previously had three short hospitalisations during the past two years, and she had received psychiatric treatment for depressive and social phobic complaints at irregular intervals for 10 years. In addition, she had attempted suicide many times. The treatment of the patient initially involved prescribing diazepam and vitamin replacement, and after that, venlafaxine and lamotrigine were prescribed. Eight months after discharge, the patient drank a large amount of alcohol only one time, but with the exception of the polydipsia, the other psychiatric complaints have continued.

In light of her history and clinical examination, the patient had a moderate alcohol use disorder diagnosed according to the DSM-5 diagnostic criteria. Concurrently, she had symptoms of PPD. There are a lot of separate case reports about PPD and BAC, but no reports of co-occurrence in people, as far as we know. This case suggests that PPD and BAC may have similar mechanisms of neurobiology and psychopathology. In this case, PPD was not discovered because appropriate laboratory testing was not conducted for a long period of time. It's possible that other BAC and PPD co-occurrence cases have not been discovered as well.

This case suggests that the co-occurrence of alcohol use disorders and PPD should be examined.

Keywords: alcohol, binge drink, psychogenic polydipsia

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[Abstract:0478][Sleep disorders]

REM sleep behavior disorder with uncal sclerosis

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Rapid eye movement sleep behavior disorder (RBD) occurs during the REM stage of sleep. Disorders causing RBD affect brainstem structures or supratentorial limbic regions. Dream enactment occurs in the presence of increased activity of locomotor centers and incomplete REM sleep associated muscle atonia. This results in a variety of behaviors such as kicking, punching, jumping from bed in response to the content of dreams, noises such as talking, laughing, and even violence. Violence with RBD results in injury to the patient or to the bed partner in 32–69% of cases. Here we represent a case with RBD.

Case: A 35 year-old male patient presented with the complaints of violent episodes of sleepwalking, hitting and hurting his bed partners,

damage surrounding objects while asleep and difficulty falling asleep. The onset of parasomniac episodes was at the age of 9. He presented to neurology and psychiatry outpatient clinics 2 years ago, but he did not use given medications regularly. He presented to our outpatient clinic after his complaints increased over the last few months. There was no family history of sleep walking or epilepsy. No stressors could be identified. His WAIS score was 72. He did not have a chronic illness. Brain MRI represent high intensity in the left hippocampus and uncus consistent with sclerosis. EEG result was normal profile. The patient was prescribed clonazepam 1 mg daily and a safe environment during sleep was recommended. In RBD the individual is oriented and able to recall the vivid dream imagery which have been shown to be predominantly unpleasant and aggressive if woken up during the episode.
Male gender, having a certain type of neurodegenerative disorder (Parkinson's disease, multiple system atrophy, dementia with Lewy bodies), having a chronic sleep disorder, taking antidepressants, use or withdrawal of drugs or alcohol are found to be associated with the development of RBD.
Keywords: uncal sclerosis, sleep behavior disorder, violence

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[Abstract:0481][Anxiety disorders]

Generalized anxiety disorder with cavum septum pellucidum

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Cavum septum pellucidum (CSP) is a putative marker of neurodevelopmental anomaly. It has been associated with an increased risk of several psychiatric disorders. We present a case of generalized anxiety disorder with CSP.
Case: A 59 year-old male patient, married, retired, presented to the outpatient clinic with the complaints of worrying about himself and his family most of the day. He was feeling that something terrible will happen. He had pathological doubt and checking compulsions. He was also complaining of persistent low mood and negative emotions for 2 years. According to the story that the patient and his close relatives told, he lay in the psychiatry clinic in 1982 with the same complaints, he received treatment by the cure of maprotiline and thioridazine, then he was discharged with the diagnosis of "depressive reaction". The patient who benefited from this treatment, released the treatment by himself. Until 1997, he hadn't been presented to the hospital because of his decreasing complaints. After his mother died, he lay in the psychiatry clinic in 1997 for the reason of his increasing complaints. He was discharged with the diagnosis of "mixed anxiety depressive disorder" with the treatment of paroxetine, trifluoperazine. Then his complaints increased two years ago, after he lend money to his son. The patient who is resistant to the venlafaxine treatment, was admitted to the service. Additional pathology was not detected according to the patient's detailed physical examination and routine observations. In his cranial MRI, cavum septum pellucidum variation was observed. The treatment of fluoxetine 60 mg/day was begun. In the patient's clinical observation, the patient's depressive complaints, and checking behaviors were decreased, but his catastrophic concerns has continued.
The septum pellucidum, a thin vertical partition of white matter separating the two lateral ventricles, is composed of two laminae. Cavum septum pellucidum is a space between the two laminae of the septum pellucidum. In normal development, the two laminae fuse by 3 to 6 months of birth. These fusion probably arises from rapid growth of midline structures such as the corpus callosum and hippocampus. Incomplete fusion results in the persistence of cavum, which may reflect neurodevelopmental abnormalities of these midline structures. CSP has been seen in different psychiatric disorders including schizophrenia, bipolar disorder, Tourette's syndrome and obsessive-compulsive disorder. In our patient, there had been treatment resistant anxiety symptoms. There was no other organic pathology except CSP. Based on the recent studies, which show the CSP is a putative marker of earlier neurodevelopment, it may be argued that neurodevelopmental origins also might play a role in treatment resistant anxiety symptoms.
We conclude that treatment resistant anxiety symptoms may be associated with neurodevelopmental anomalies, which may be represented by CSP.
Keywords: anxiety, cavum septum pellucidum, treatment resistance

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[Abstract:0483][Psychopharmacology]

Rabbit syndrome induced by aripiprazole

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Rabbit syndrome (RS) is an extrapyramidal syndrome (EPS) that is rarely seen, induced by neuroleptics and characterized by involuntary, rapid, rhythmic mouth movements seen at vertical axis. D2 partial agonistic effect of aripiprazole, that is one of new generation antipsychotics, makes safer this agent with respect to extrapyramidal side effects. In the literature, a few number of cases related to RS induced by aripiprazole were reported. For this reason, here we aim to present an older patient who have used aripiprazole for six years and developed RS by starting macrolide group-antibiotics three months ago.

Case: A 75 year patient referred to our clinic with complaint of involuntary movements around mouth presented approximately three months. The patient who has being continued 50 mg/day sertraline, 10 mg/day aripiprazole for six years and with no complaints until three months ago was prescribed with macrolide 400 mg daily, paracetamol 500 mg 2 tablets QID and acetylcysteine after upper respiratory infection occurred three months ago. In voluntary movements around mouth started after drugs. The patient referred to the Internal Medicine Outpatient Clinic and Neurology Clinic with these complaints. In performing examinations, any pathology was not detected. Quetiapine 25 mg BID was started to patient by the neurologist; because there was no regression in complaints, patient was directed to psychiatry. Thus, aripiprazole and sertraline treatments of patient referring our clinic were discontinued and quetiapine treatment was continued. Quetiapine of patient was increased to 100 mg and 0.5 mg alprazolam was added to treatment because of continuation of complaints in 1-week later visit. Alprazolam of patient with continuous complaints was discontinued and 2 mg biperiden was started and then complaints of patient completely regressed.

Aripiprazole is found safer than other atypical ones with respect to EPS thanks for having a special receptor interaction structure. However, in spite of all other characteristics, showing higher D2 receptor occupancy in striatum even if at lower doses and lower anticholinergic effect may provide contribution to development of RS. RS can occur after shortly from starting to treatment, any time during treatment particularly dose-change times, or following by discontinuation of the drug. Additionally, starting an agent particularly affecting cytochrome p450 system because of any indications can cause occurrence of clinical picture by changing speed of neuroleptic eliminations. Recognition of rarely seen extrapyramidal syndrome RS, differentiation from tardive dyskinesia, and knowing that even if aripiprazole that is thought to be safe with respect to EPS can lead to this clinical picture is important. Moreover, particularly in older patients when using an agent, drug interactions should be evaluated and it should be kept in mind that the risk can increase with respect to RS together with extrapyramidal symptoms.

Keywords: rabbit syndrome, aripiprazole, tardive dyskinesia

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[Abstract:0484][Psychopharmacology]

Fluoxetine and aripiprazole combination -induced extrapyramidal symptoms in an adolescent: a case report

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In the pharmacotherapy for Obsessive-Compulsive Disorder (OCD) the effectiveness of selective serotonin reuptake inhibitors (SSRIs) has been well established. However, in practice, only about 60% of patients respond to SSRIs administration. For such cases, additional pharmacological treatment strategies such as combination drug treatment strategies have been investigated. This case report discusses a case of OCD, who had extrapyramidal side effects (EPS) during a combination treatment with fluoxetine and aripiprazole.

Case: A 15 year-old female adolescent who suffered from OCD for one year. She was previously a very good student, behaviorally and academically. She presents to an outpatient clinic with symptoms of contamination obsession, washing compulsion, anxiety and school refusal. Her complaints began 1 year ago. However, she reported a persistent need to wash his hands, wasting up to 3–4

hours daily on ritual hand washing. She reported feeling anxious and restless if he tried to resist washing his hands. Her OCD had not responded to cognitive-behavioral strategies. She consistently scored around 25 on the Yale-Brown Obsessive-Compulsive Scale (Y-BOCS).The case is a chronic and resistant case who had not taken her medicine regularly. We planned to continue with fluoxetine and aripiprazole treatment. She was given fluoxetine 20 mg/day and aripiprazole 5 mg/day. Two weeks after the start of aripiprazole and fluoxetine therapy, rigidity appeared. The patient complained of the inability to remain still, tremor and pain of arms. There were no restlessness syndrome and akinesia. Biperiden 3 mg/day was ordered. Ten days after aripiprazole was discontinued, the side effects disappeared. Therefore the fluoxetine dosage was not lowered. Her total score on the Y-BOCS dropped to 20. She still comes to our outpatient clinic for her follow-up.

In this case, the cause of extrapyramidal side effects was unclear. It may be because of fluoxetine or aripiprazole or both of them. There are reports of EPS of fluoxetine and aripiprazole in child and adolescent. The combination of antidepressants with antipsychotics may also cause EPS. Drug interactions are very important and should be considered during combination treatments. Cytocrome P450 and other cytocrome enzyme systems have an important role in the therapeutics, effectiveness, interactions, side effects and these mechanisms fluoxetine and aripiprazole combination may potentialize each other or may cause elevation in blood levels which may increase the risk for EPS. In this case, although the treatment period was very short and the dosages were low, EPS were seen. We could not find any organic or pathological sign to explain the EPS. After aripiprazole was stopped, the side effects disappeared. Therefore EPS in this case we report may either be related to cytocrome P-450 enzyme system or may just be hypersensitivity reaction to aripiprazole. Clinicians should be aware of the combinations of SSRIs, especially of fluoxetine with antipsychotics as a potential causative factor for EPS.

Keywords: obsessive-compulsive disorder, extrapyramidal symptoms, aripiprazole, fluoxetine, drug interaction

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[Abstract:0486][Schizophrenia and other psychotic disorders]

A schizopheria patient with tongue abscess after self-medication

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Tongue abscess is considered to be a rare condition among otolaryngology patients. The rarity of lingual abscesses, despite frequent bite traumas to the tongue during mastication and seizure attacks. Despite self mutilation behaviors in schizophrenic patients, self- injection of self-produced medication is nearly not exist.

Case: A 39 year-old male patient presented to the otolaryngology outpatient clinic with loss of speech ability, swelling and pain at his tongue. He had this complains for three days. After physical examination, a tongue abscess was determined, so he hospitalizated for etiology, radiological scanning and treatment at otolaryngology yard. The first vital diagnosis of patient was normal. Abscess was drained. Because of his behaviors and abscess' character, psychiatry consultation was requested. During the examination, he hid himself other patients and nurses and spoke with low voice. His medical story based on medical records because he had no relatives. He previously told to the otolaryngologist that he made a liquid mixture that included olanzapine, goat's feces, liquorice plant, some herbal seeds, vinegar and injected the right side of his tongue. He was searching alternative solutions except prescribed medicines. According to him, he made better physicial condition owing to this mixture. For testing benefits of mixture, he had started first at his tongue. In the psychological examination of the patient he was not willing to collaborate. His conscious was open, he was orientative. He was seemed his chronocial age,he was tall, his weight was appropriate his length, his dressing was appropriate to season,self care was bad. He was suspicious and tense. Eye contact was indirect. His speech was clear. Speech rate was slow, he mainly whispered. Flow of speech was hesitant. Amount of speech was reduced. Thought process was relevant to topic. He answered only questions. He had invention, grandiose and persecution delusions. Mood was dysphoric, affect was appropriate with his delusions irritable, range of affect was restricted. His attention was easily distractible. Reality evaluation was distorted. He had complete denial of illness. He did not describe any visional, olfactory, tactile and auditory hallucinations. Person, time and place orientation was good. He hadn't got withdrawal of substance and alcohol. Short-term recall test and long term memory was good, there was no evidence of mental retardation. In order to evaluate the organic pathology; routine biochemical tests, EEG, and the brain MRI were performed. Results of biochemical tests were found to be within normal limits. Electroencephalography did not reveal any other pathologies and cranial MRI was normal. He had already schizophrenia diagnosis for 10 years but he had left his medications. His PANSS score was 73 at first psychiatric examination. After his iv antibiotic treatment he discharged. Patient was initiated on olanzapine treatment with a dose of

10 mg/day and the dose was gradually increased up to 20 mg/day. Significant improvements in symptoms were observed on the 45rd day of hospitalization (PANSS: 28).
To the best of our knowledge this is the first case report of a schizophrenic patient who self-injected self produced medication in his tongue and has created tongue abscess.
Keywords: schizophrenia, self medication, tongue abscess

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[Abstract:0488][Eating disorders]

Overvalued ideas and delusions in a male patient with anorexia nervosa

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Anorexia Nervosa (AN) is a chronic eating disorder that is recognised by irrational beliefs about body image and significant weight loss caused by excessive dieting and a fear of becoming fat or having some behaviors that make difficult to gain weight. Information of eating disorders in men is limited as they are much more common in female. Special features like having a specific goal or sexual orientation have been reported in male patients with eating disorders. The contents of body image beliefs in AN is very little understood. These beliefs have been suggested as overvalued ideas which has been compared to obsessions or delusions. Other symptoms of psychosis can also develop in AN independently. The comorbidity of diagnosable schizophrenia and eating disorders is calculated below 10%, with a higher risk for males. Male patients diagnosed for AN and then schizophrenia showed high comorbidity prevalence. AN-restricting subtype (AN-R) patients have a higher risk of developing symptoms of psychosis and have common neurocognitive deficits with schizophrenic patients. It is frequently observed that AN may precede schizophrenia or emerge after schizophrenia. And some symptoms can overlap in schizophrenia and AN; these disorders can be one within the other. It is proposed that AN may occur in a process from neurosis to psychosis.
Case: A 18 year-old male patient sought treatment because of his eating disorder which has emerged 2 months ago with restricting food and over exercising to lose weight, to have a well-shaped body and to improve athletic performance. However it was understood that he has been suffering from his paranoid ideas for almost 3 years. Having specific goals and describing himself as asexual support the data which points out the frequency of these contents in men with AN. In this case, beside his body image distortion there is a delusional symptomatology related to paranoid ideas that the others would follow and imitate him, that's why everyone changes behavior because of him. These delusional thoughts are different from the typical thoughts of an AN patient. Amisulpride was used for the pharmacologic treatment and the patient began to be cooperative and his social withdrawal and psychotic anxiety reduced, he began gaining weight and stopped over exercising. Activity based anorexia (ABA) refers to the weight loss, hypophagia and paradoxical hyperactivity. This case supports the literature which shows that D2/3 antagonist amisulpride reduced weight loss and hypophagia and increased survival during ABA; furthermore, amisulpride produced larger reductions in weight loss and hypophagia than olanzapine when it was studied in mice. The importance of this case to take attention to the comorbidity of AN and psychosis because of the frequency and negative impact on quality of life. Assessment of psychotic symptomatology provides a correct diagnose and an effective treatment. This clinical case suggests that the use of amisulpride could be useful in a severe case of AN with delusional symptoms and a multidisciplinary approach should be performed for the patients with AN and psychosis comorbidity.
Keywords: anorexia nervosa, delusions, psychotic disorders, schizophrenia

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[Abstract:0491][Psychopharmacology]

Depressive symptoms related to topiramate addition to fluoxetine treatment in a bulimia nervosa case

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Topiramate is an antiepileptic drug. Beside the use for psychiatric indications, topiramate has also some psychiatric side effects, especially when used in the treatment of neurological diseases. Most reported psychiatric side effect of topiramate is depressive symptoms. But there is not any report related to topiramate fluoxetine combination associated depressive symptoms. Here we report a case of depressive symptoms associated with topiramate addition to fluoxetine treatment.

Case: A 19 year-old female patient, single, presented to the outpatient clinic with complaints of binge eating episodes, obsessions related to her weight, aversion, adductive and social isolation because of the thoughts of being overweight one year ago. In her medical history these complaints existed for 5 years. With diagnosis of Major Depression and Bulimia Nervosa, fluoxetine 20 mg/day treatment was started and titrated to 40 mg/day. At the end of one year treatment depressive symptoms recovered totally and eating episodes decreased at the rate of 50%. Despite the decrease, eating episodes presented everyday. Topiramate 75 mg/day added to the fluoxetine treatment. With topiramate treatment eating episodes recovered totally. But depressive symptoms such as psychomotor retardation, anergy, anhedonia and depressive mood developed at the second week of topiramate addition. Depressive symptoms were thought to be associated with topiramate and topiramate treatment was stopped. After cessation of topiramate depressive symptoms improved gradually and there was no depressive symptom at the end of one week. On her last psychiatric evaluation her mood was euthymic and had no eating episode in the meantime. Bulimia Nervosa is characterized with binge eating episodes, compensatory behaviors as taking laxatives, diuretics, vomiting or excessive exercising and body image disturbance. Antidepressant treatment and cognitive behavioral therapy are first line treatments of bulimia nervosa. In the cases which eating episodes cannot be controlled, topiramate augmentation can be useful. Topiramate has also been linked to the development of depression in approximately 10% of patients. Topiramate related depressive symptoms appear to be dose-dependent and occur more often after rapid dose titration. Also patients who has a personal or family history of depression have increased risk for topiramate related depressive symptoms. In this case report, the patient was already under fluoxetine treatment and there was no depressive symptom when the topiramate treatment was added. Topiramate treatment was started in a low dose (25 mg/day) and titrated slowly to 75 mg/day. In the second week of topiramate addition depressive symptoms emerged and recovered totally after cessation of topiramate. Because of the temporal relationship between depressive symptoms and topiramate treatment, depressive symptoms were thought be developed as a side effect of topiramate. To our knowledge this is the first report related to depressive symptoms associated with topiramate addition to fluoxetine treatment. As a note, it should be considered topiramate may cause depressive symptoms even though used combination with fluoxetine. Slow dose-titration schedule and increased monitoring for depressive symptoms may be helpful to prevent depressive side effects related to topiramate addition.

Keywords: bulimia nervosa, depressive symptoms, topiramate

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[Abstract:0493][Psychopharmacology]

Anterograde amnesia after sildenafil use

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Sildenafil is a phosphodiesterase type-5 (PDE5) that is commonly used in the treatment of erectile dysfunction. The side effects due to sildenafil use are dizziness, headache, nausea, visual changes, tingling, flushing. Some rare neurological complications such as third cranial nerve palsy, tonic-clonic seizures, intracerebral hematoma and transient global amnesia are also reported. There are two cases developing the transient global amnesia due to sildenafil use. To best our knowledge, anterograde amnesia associated with sildenafil use has never been reported.

Case: A 62 year-old male patient, married and a farmer. He presented to the psychiatry clinic with the help of his relatives with complaints such as confusion and inability to perceive what was happening around. He had been already presented to neurology clinics on the first day that these complaints started. There was no abnormalities in the laboratuar tests. The brain magnetic resonance imaging (MRI), diffusion MRI and electroencephalography were normal range. He did not have a complaint regarding the mental and psychological aspects earlier and he did not use psychotropic agents in his life. Lately, he did not have a problem or traumatic situation as well as he did not complain about perception or comprehension problems till he used sildenafil. He had used Sildenafil 100 mg one day before the complaints started. According to the psychiatric examination, he showed the decreased self-care, scattered attention and he was responding to questions with short sentences consisting of a few words. Time orientation of the patients was damaged. He did not give correct answer recall and registration parts of the mini psychiatric exam test. As a result of his psychiatric examination, anterograde amnesia was diagnosed according to DSM-IV diagnosis criteria and it has been understood that it was developed secondary to sildenafil use.

Our case differs from others regarding the duration and type of the amnesia and accompanying comorbid factors when similar case reports are evaluated with respect to the amnesia due to sildenafil use. Patients with transient global amnesia generally recover after 24-48 hours of time, but our patient had the complaints, even on the 10th day. This is the main characteristic of our patient that differs from other cases.

Clinicians should consider that sildenafil can lead to alterations in the vascular structure, even there is no disease due to this deterioration. It should be kept in mind that advanced age can be the risk factor for sildenafil treatment. There should be more comprehensive studies that can examine the effects of sildenafil on cerebrovascular system.

Keywords: amnesia, sildenafil, adverse effect

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[Abstract:0494][Perinatal psychiatry]

Case report: depression during pregnancy

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Pregnant women must adapt to many physiological, psychological and social changes occur during gestational period. Antenatal mental illness are more prevalent in developing countries and woman with low socio-economical or low-educational status. Antenatal mental illness can complicate pregnancy and the postpartum period and affect maternal and fetal outcomes.

Case: A 43 year-old pregnant patient presented to our clinic in her 14th gestational week. According to her history she was a housewife and was living in Canakkale city center, and it was the first pregnancy from the second marriage. She has got two healthy children from the

previous marriage, and she delivered both of them vaginally. She had some some psychological complaints (sadness, unwillingness, feelings of worthlessness, insomnia, anorexia, death ideation, anhedonia), she was using sertraline 50 mg since 10th week. Four weeks later, she was brought to emergency department as a result of suicide attempt (used 700 mg sertraline). Obstetric conditions was stable. So, after the first intervention, the patient was hospitalized in psychiatric clinic. Electroconvulsive therapy was suggested by psychiatrist. But it was rejected by both the patient and her husband, so it was not performed. Her husband was constantly show the hostile behaviors during the hospitalization and outpatient follow-ups. During the hospitalization in the psychiatric clinic, her blood pressure was suddenly increased (about 150/100), but did not have any obstetric pathology. Although she had hypertension before pregnancy she did not use any antihypertensive drug. She also rejected to underwent analysis like 24 hour urine testing for proteinuria. Her blood pressure was regulated and she was discharged voluntarily from psychiatry clinic. She did not come to routine obstetric and psychiatric visits. In the 40th week of pregnancy, the patient presented to hospital with a fully dilated cervix, and she delivered a 2930 gr baby vaginally with 8/10 APGAR scores at the first and fifth minutes and after that, thoroughly increased of depressive mood in postpartum period. We wanted to consult the patient to psychiatrist for this reason, but she and her husband didn't allow us, and escaped from the hospital without permission. In postpartum period, she was continued the treatment by herself with the dosage suggested during pregnancy, and she was divorced from her husband. She did not come to visits anymore. But as far as we learnt from phone consultation she has not got a complaint about her emotional mood nowadays.

Depressive and anxiety disorders are the most common psychiatric conditions seen during pregnancy. While depression prevalence during pregnancy vary between 5%-25%, studies either on the prevalence of depression and anxiety or their effect on pregnancy outcomes have conflicting results. While some studies conclude that depression complicate perinatal outcomes by preterm birth, low birth weight, and low APGAR scores others report no significant impact of antenatal depression and anxiety on obstetric outcome. All pregnant women should be evaluated carefully for psychiatric disorders. Because, emotional stress is increasing with a changing hormonal pattern during this period and especially depression is very prevalent and decreases the quality of life significantly.

Keywords: anxiety, depression, pregnancy

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[Abstract:0496][Psychopharmacology]

Paliperidone-induced sialorrhea: a case report

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Excessive salivation is a frequent side effect associated with antipsychotic therapy. Clozapine is among the most widely reported antipsychotic to be associated with excessive salivation. Although sialorrhea, which mostly develops in the early period of antipsychotic treatment, has been regarded as a relatively benign adverse effect by the clinicians, it is reported as very disturbing by the patients. Drooling causes pooling in oral cavity and this leads to overflow of saliva from mouth which also may lead to shaming.

Paliperidone, an atypical antipsychotic, is a benzisoxazole derivative and the major and active metabolite of risperidone. Sialorrhea induced by paliperidone has not been covered in the literature. Herein, we wish to report a case of a patient who developed sialorrhea secondary to paliperidone coupled with extra pyramidal symptoms (EPS).

Case: A 62 year-old female patient diagnosed with schizophrenia for 25 years, presented with psychomotor agitation, confusion, disorganized behavior, and persecutory delusions. She had the problem of treatment noncompliance and had not been taking any type of antipsychotics for at least three months. We hospitalized her and started paliperidone (PO) treatment with the initial dose of 3 mg/day and increased to 6, and 9 mg/day. Although her symptoms improved four weeks after starting administration of paliperidone, she developed severe sialorrhea and EPS (e.g., slurring of speech, tremor, cog wheel rigidity). Thus, we gradually decreased the dose of paliperidone from 9 mg/day to 3 mg/day. Sialorrhea and EPS, however, did not subside. Subsequently, we stopped paliperidone and started olanzapine (PO) 10 mg/day. One week after stopping paliperidone, sialorrhea and EPS were totally resolved without exacerbation of psychotic symptoms. Paliperidone is a novel antipsychotic agent that demonstrates high affinity for central dopamine 2 and serotonin 2A receptors, a mode of action common to many atypical antipsychotics. Paliperidone is also active as an antagonist at α1- and α2-adrenergic receptors and H1 histaminergic receptors. Paliperidone has no affinity for cholinergic muscarinic or β1- and β2-adrenergic receptors. Excessive salivation is an unfavorable adverse effect of antipsychotic treatment and possible mechanisms include adrenergic muscarinic M4 agonism and alpha-2 antagonism. Thus, paliperidone may cause sialorrhea via α2-adrenergic antagonism. Several choices have been proposed in the treatment of clozapine-induced sialorrhea, including centrally acting alpha-2 adrenergic receptor agonists (i.e., clonidine, lofexidine) antimuscarinic agents (i.e., biperiden, atropine), beta-adrenoreceptor blockers, as well as diphenhydramine, and botulinum

toxin injection. So far, however, there are no data on the management of sialorrhea related with paliperidone treatment. In our patient, sialorrhea and EPS commenced after initiation of treatment with paliperidone and resolved after ceasing this agent. Thus, we may conclude that paliperidone can cause severe sialorrhea, even in low doses. Switching to another antipsychotic may be an appropriate treatment strategy in patients with paliperidone-induced sialorrhea.
Keywords: paliperidone, sialorrhea, treatment

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[Abstract:0498][OCD]

Obsessive compulsive disorder and Gardner syndrome comorbidity

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Obsessive compulsive disorder is a common disease, occurring in about two percent of the general population. Obsessions disturb the person and cause distress. Compulsions are motor or mental behavior to reduce the distress that occurs in response to obsessions. Obsessive- compulsive disorder etiopathogenesis is still unclear. It is believed that the disorder stems from multi factorial influences. Genetic, neuroimmune, infectious, social and cultural reasons can cause this disorder. Obsessive compulsive disorder occurs at a young age and the average age of onset is 18-25 in studies. The early onset occurs predominantly in male gender and the prognosis is worse and also comorbidity are more. In late onset; organic etiology should be excluded. In our case; we will discuss the obsessive compulsive disorder and Gardner syndrome.
Case: A 32 year-old male patient, single, student. The patient presented to the outpatient clinic with the symptoms of checking compulsions. He had OCD diagnosis since age of 13. When his mother died of cancer, he was 4 years-old and his brother died of cancer a year ago. In 2009, patients diagnosed with Gardner's syndrome and for the prevention colon was taken by surgeons. Fluoxetine 20 mg/day treatment was started to the patient. In follow-up exams, the symptoms were decreased.
Gardner syndrome is an autosomal dominant syndrome. It is one of the familial adenomatous polyposis syndrome and depends on mutations in APC (adenomatous polyposis coli) gene on the 5th chromosome long arm. Syndrome consists of intestinal polyposis with osteomas and multiple cutaneous and subcutaneous lesions. The colonic polyps usually undergo malignant change. In literature we have not seen Gardner syndrome and obsessive compulsive disorder comorbidity. But in literature, we know that APC gene is a risk factor for schizophrenia. Adenomatous polyposis coli (APC) gene is a tumor suppressor gene. APC is located at 5q21-22, which has been previously reported to be linked with schizophrenia. Studies show that APC may be a candidate gene conferring susceptibility to schizophrenia. Probably APC gene can cause several psychiatric disorders and one of them may be obsessive compulsive disorder. In future studies, investigating tumor suppressor genes and obsessive compulsive disorder may be a method to understand the etiology of some psychiatric disorders.
Keywords: adenomatous polyposis coli gene, gardner syndrome, obsessive compulsive disorder

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[Abstract:0499][Perinatal psychiatry]

Postpartum psychosis or autoimmune encephalitis?

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The incidence of postpartum psychosis is 0.1% and it's a serious condition. A complete physical and neurological examinations are necessary to rule out underlying diseases. The women who have psychiatric symptoms first time after delivery usually have significant

autoimmune pathologies. Recently, many studies have shown that some of the postpartum women with psychiatric symptoms have underlying autoimmune encephalitis. Before neurological manifestations, autoimmune encephalitis can make hallucinations, behavioral changes, delirium like psychiatric symptoms.

Case: A 26 year-old patient, had parturition 16 days ago. Gravida 1 parity 1, there is no significant health problem. She presented to emergency department because of abnormal behaviors, defecation on carpet, saying that somebody wants to catch her, sleeplessness and talking too much. She was hospitalized to psychiatry inpatient clinic. She was conscious but time orientation is false, not much cooperable, she had persecution delusions, auditory and visual hallucinations. Treatment started with haloperidol 20 mg\day, biperiden 10 mg\day intramuscular and on third day of treatment her consciousness get worse and she had minimal rigidity on her extremities. She was transferred to neurology intensive care unit. At ICU haloperidol was discontinued because of EPS. All causes of CNS disease were considered in differential diagnosis. Everything was normal except from minimal CSF leukocytosis. Autoimmune encephalitis was suspected. Some of autoantibodies and paraneoplastic markers in serum and CSF were negative. MRI and EEG were normal. She was given 5 days IVIg and 7 days pulse steroid treatment. After treatment she recovered and transferred inpatient service and discharged. One week later she presented to emergency room with similar complaints. After the all examinations continuous of encephalitis was diagnosed. She had taken the second cure of IVIg treatment, after some recovery she transferred to psychiatry inpatient clinic. At this stage; her MRI showed hypophyseal enlargement and EEG showed bioelectrical disorganization in both hemispheres. Her treatment was arranged as olanzapine 20 mg/day and valproate 500 mg/day. She was discharged from hospital with remission. Since then she was examining at outpatient clinic, there have been not any relapses and she is well.

The pathophysiology of postpartum psychotic disease is not well known. Activation of immune system is said in a group of patient. Autoimmune encephalitis mimic psychiatric diseases very much. For example anti-NMDA encephalitis is seen in women usually who have first psychiatric attack at postpartum period. Patients can come with a complete psychiatric picture. In our patient serum and CSF autoantibodies that were searched at acute period were negative but it does not rule out the diagnosis. Because the screening panel was neither include all antibodies nor the tests repeated. Also recovery with IVIg and steroid treatment support our diagnosis. Disease has not relapsed during 2 years follow up. Autoimmune encephalitis is a treatable condition and must be considered in newly diagnosed postpartum psychiatric patients. Patients with autoimmune encephalitis admit to psychiatrist with many psychiatric and underlying diseases may be misdiagnosed. Psychiatrist and neurologist cooperation, improvement of laboratory tests will help the early diagnosis and recovery rates will be better.

Keywords: postpartum psychosis, autoimmune, NMDA encephalitis

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[Abstract:0500][Psychopharmacology]

Mirtazapine in the treatment of emetophobia: a case report

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This article describes the treatment and follow-up of a 13 year-old female patient, who was diagnosed with emetophobia and showed significant improvement with mirtazapine.

Case: A 13 year-old female patient presented to our clinic with refusal to eat and avoiding any fluid intake other than water and milk. The patient had been avoiding going to school for approximately two months, The patient received fluoxetine, sertraline, clomipramine, and alprazolam for sufficient periods of time and with appropriate doses as well as 13 sessions of cognitive behavioral therapy with the diagnosis of generalized anxiety disorder, and did not respond to treatment. Detailed psychiatric evaluation showed that the patient's complaints are associated with fear of vomiting. She refused to eat with others and have any liquid intake other than water or milk with the fear of vomiting. Lately, her fear increased extensively, and she started to refuse going to school. She lost approximately 10 kg in the last two months (BMI at the time of interview: 16.24). In her medical history, we have found out that when she was in second grade, her friend sitting next to her suddenly started vomiting, the desk and books were covered with food remnants, her clothes were also soiled, her friends found this incident "very nauseating", and ever since she feared of vomiting and this fear increased extensively in the last year. We have consulted to pediatric gastroenterology for evaluating the patient for an organic etiology; her tests showed normal results. In the light of all findings, the patient was diagnosed with emetophobia. Considering the treatment history of the patient, it was decided that the patient would be started on mirtazapine. The patient was started on mirtazapine at 7.5 mg/day and checked at two-week intervals. In the first interview after two weeks, it was stated that the refusal to eat decreased to some extent. The dose of medication was increased to 15

mg/day. During the follow-up period, the patient changed the behavior of avoiding going to school notably, the behavior of refusal to eat completely and gained weight (BMI at the end of six months: 18.49). The patient well tolerated the medication during the treatment period. Emetophobia is a specific phobia characterized by excessive fear of vomiting. Mirtazapine is a noradrenergic and specific serotonergic antidepressant. It blocks the presynaptic alpha-2 adrenergic receptor and increases the release of noradrenaline and serotonin. Furthermore, there are investigators reporting an association between the blocking of postsynaptic 5HT2, 5HT3 receptors and earlier onset of anxiolytic efficacy. In reviewing the literature, we have encountered pediatric patients with a specific phobia, which have been successfully treated with mirtazapine. To our knowledge, this is the first report of successful treatment of emetophobia with mirtazapine in the period of childhood. For specific phobia affecting the daily functionality in child patients, mirtazapine may be a good therapeutic choice for obtaining an earlier positive response in. Our report is considered to be significant in this respect.
Keywords: child, emetophobia, mirtazapine

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[Abstract:0501][Psychopharmacology]

Visual hallucination associated with duloxetine: a male patient diagnosed with generalized anxiety disorder

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This article describes a male patient, who received duloxetine treatment for generalized anxiety disorder (GAD) and suffered from visual hallucinations with the use of duloxetine.
Case: A 16 year-old male patient had been monitored at an external child psychiatry clinic with the diagnosis of GAD for approximately three years. Over this period of time, the patient had received single or combined sertraline, fluoxetine, citalopram, venlafaxine, clomipramine, lorazepam, alprazolam, mirtazapine, hydroxyzine and propranolol for sufficient periods of time and with appropriate doses. Additionally, he had received 12 sessions of cognitive behavioral therapy (CBT). The patient presented to our clinic since he didn't respond to the treatment. At the time of application to our clinic, the patient presented with excessive worry about something bad happening to himself or his loved ones, or failing in his exams. The patient stated that he cannot control his thoughts and clear his mind from anxiety, causing him sleep onset insomnia and complaints of not having enough sleep. He stated that his anxiety negatively affects his school success, friends and family relationships. The patient was diagnosed with GAD. Considering the medical history of the patient, it was decided that the patient would be started on duloxetine. The patient's pretreatment blood tests (Hemogram, liver-kidney function tests, electrolytes, blood glucose and fat levels, and thyroid function tests), vital symptoms and cardiovascular system examination were normal. The patient had not been using any medication for approximately two months. The patient was started on duloxetine at 30 mg/day. On the fifth day of treatment, the patient presented to our outpatient clinic again. The patient stated that he had been seeing strange images since the previous evening, that he had occasionally seen a white cat or a black dog passing by him, and that these images had continued throughout the night and therefore he could not sleep well. The patient received no other medication during this period. No organic etiology was found in consultation with the pediatric neurology clinic. Two days after the discontinuation of duloxetine treatment, hallucinations disappeared completely.
Duloxetine is a serotonin-noradrenaline reuptake inhibitor which has been increasingly used in child and adolescent psychiatry practices. Its effects on child and adolescent patients are not currently known. However, some studies suggest that duloxetine can be used in pediatric population at doses similar to those of adults without any dose adjustment and is well tolerated. In reviewing the literature, we found that visual hallucinations considered to be associated with duloxetine were reported for two adult patients It is not clearly known how duloxetine molecule causes visual hallucinations. However, it is stated that dopamine increase, which is considered likely to be especially associated with inhibition of noradrenaline transporters in the prefrontal cortex, or which develops secondary to serotonin reuptake inhibition, may cause such side effects. As far as it can be seen, our patient is the first adolescent patient to describe visual hallucinations during duloxetine treatment. Our report is considered to be significant in this respect.
Keywords: adolescent, duloxetine, generalized anxiety disorder, visual hallucination

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[Abstract:0504][Schizophrenia and other psychotic disorders]

Cavum septum pellucidum and schizophrenia: a case report

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Septum pellucidum is a structure composed of two thin laminaes which forms the medial wall of the lateral ventricle. When this fusion is insufficient, the gap between septal laminaes is called the cavum septum pellucidum (CSP). CSP is a part of normal development. The fusion of the laminaes and closure of CSP ends during intrauterine period or in 3-6 months postnatally. This fusion is thought to be a result of simultaneous growth of corpus callosum and the hippocampus. Cavum less than 6 mm has been considered as normal variant, but enlarged cavum, greater than 6 mm, has been found to be associated with the development of psychiatric disorders or Mood disorders. Schizophrenia is the most common comorbid disorder with CSP.

Case: A 56 year-old female patient referred to our outpatient clinic with complaints of inability to control anger, forgetfulness, hearing voices, and seeing her death relatives. Her first psychiatric complaints; unhappiness, crying, spending too much time with water, suspicion and locking herself in her room, started 32 years ago after she divorced. Her first application to psychiatry was 10 years ago when aggression started. She hospitalized with the diagnosis of schizophrenia. After 3 years of using different antipsychotic treatments (risperidone, olanzapine, ziprasidone, aripiprazole) she did not have enough improvement and was given clozapine 900 mg daily. Clozapine treatment was found to be effective but stopped for some unknown reasons. After 2 years of treatment with amisulpride 600 mg daily, her complaints continued and she hospitalized in our clinic for the regulation of treatment. In mental status examination; she had low self-care. She was nervous, disoriented to time and had impaired memory. Her amount of speech was increased. Her attention was inadequate, associations were disorganized, thought content was impoverished and she had perseverations. She had visual and auditory hallucinations. Judgment and abstract thinking was inadequate. She had psychomotor restlessness. She had poor performance on neuropsychological tests. Routine blood tests and EEG results were normal. Cranial MRI showed variation of cavum septum pellucidum, cortical atrophy of frontal and temporal areas, scattered areas of gliosis in bifrontal subcortical white matter. In cranial MR angiography left transverse sinus and sigmoid sinus hypoplasia observed. Amisulpride treatment stopped and clozapine treatment started. When clozapine increased to 500 mg daily, EEG was repeated and epileptiform discharges over temporal lobe was found. Clozapine was reduced to 400 mg daily and valproic acid 1000 mg daily was added. She is still inpatient in our clinic.

The relationship between schizophrenia and developmental disorders such as CSP, corpus callosum agenesia is known. CSP was observed to be associated with severe thought disorders, intellectual functions, loss of cognitive abilities such as verbal learning and memory, retardation and seizures. Symptoms and lower scores on neuropsychological testing in this case is consistent with the literature. Resistance to treatment may be associated with neurodevelopmental abnormalities.

Keywords: cavum septum pellucidum, developmental disorders, schizophrenia

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[Abstract:0507][Substance-related and addictive disorders]

Pheniramine: a hidden danger!

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Pheniramine (Avil®) is reported mostly related to dependency which's mechanism of action is H1 receptor antagonism besides anticholinergic effects. There are multiple case reports based on antihistaminic dependency such as pheniramine like the other centrally acting antihistaminics but unfortunately they are acquirable without prescription and there is no limitation for accessibility. We aim to notice a rarely seen case and the dependency profile of this kind of drugs which is cheap and uncontrolled.

Case: A 44 year-old female patient, lives with her daughter and son, divorced twice, unemployed, grew up at the social service and

children protection institute, overweight, has major depressive episode in her past and immature personality traits. When she presented outpatient psychiatric clinic for the last time with low mood, unhappiness, restlessness, hopelessness, anxiety which have been alleviated dramatically by using pheniramine. She was using 20 IV injections of Avil® ampoules, which contains 22.7 mg pheniramine in each ampoule, per day. Therefore, the total consumption was 908 mg/day and it was 9.08 mg/kg/day (despite maximum dosage 3 mg/kg/day). This was the highest amount of pheniramine use reported in the literature. She started using pheniramine 7-8 years ago for her allergic symptoms and discovered sedative side effects. Afterwards she continued abusing pheniramine intravenously as chronic high dose self-administration but as long as tolerance grow up to its sedative side effects, she needs higher dose to get the same effect. She goes on to misuse pheniramine despite its harmful effects reach necessity for local surgical intervention for subcutan infection due to excessive IV injections. She ignored problematic pheniramine use, hates relatives when stopping her to use increased doses of pheniramine but she wanted to rescue from its withdrawal symptoms. At the same time she could not find any area in her body to apply injection; various sides of her wrist and knees. Despite she was not motive and willing to give up dependency, she was hospitalized by pheniramine dependence diagnose, she received aripiprazole 10 mg/day and fluoxetine 20 mg/day. She reported no withdrawal symptom or cravings for 2 days but after all she started developing physical and psychological deprivation symptoms like trembling at her hands, restlessness, worsened anxiety, dysphoria and craving. In these times, we interfere with short-acting benzodiazepines. There was no another drug or alcohol dependence in her history but although her refusal, her daughter said she was an internet use and virtual sex addict. Not just craving for pheniramine, also for the other dependencies and because of the lack of social support system; she quit, reject the treatment and discharged.

There are only a few publications about pheniramine dependency but it appears that not enough to provide more serious limitation or urgency system. This case is remindful for caution for all of the uncontrolled, easily accessible and potentially addictive drugs like antihistaminics. There is need a warning system for unsuitable use of these kind of drugs.

Keywords: addiction, antihistaminic, dependence, pheniramine, misuse

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[Abstract:0510][Others]

Dissociative identity disorder after a childhood trauma: a case report

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Dissociative identity disorder is still discussed by some experts as a diagnostic way. It is also called as "iatrogenic disorder" in some studies. In this study, DID which can be seen a rarely diagnose is presented with a case whom is followed up in our clinic.

Case: A 27 year-old, single male patient presented to our clinic with the symptoms of yakking and absurd movements when he did not remember after and hostile behaviors which due to that. Normal Neurologic examination, increased speech, normal voice tone, lacunar amnesia which due to dissociative intervals in his psychiatric examination were also observed in patient. He had anxious and passion (torture) affect. Forgetfulness that used to happen in his high school years was the first complaints in his medical history. Firstly, he felt temperature on his face, sense of water leakage into his nose; blackout and he had failure to perceive his surrounding areas. After than; he felt himself like an outsider and he did not remember these all events. He presented to the psychiatry policlinic for the complaints of amnesia, his clinic status is diagnosed as identity disorder and sertraline 50 mg/day and risperidone 2 mg/day were started. Patient used that treatment for 6 months, however patient was failed to respond to the therapy. He presented to another psychiatry clinic and paroxetine 40 mg/day was started for two months by the diagnose of dissociative fugue. He attempted suicide in 2013. He did not remember the event even the pictures of scene had showed to him. He had been raped by his uncle at the age of 7. He cannot love children, when he tries to love children, he thought himself as his uncle. He changed his name and surname because his family got mixed up in crime and drugs, his dad was also a drug user too.

Modern childhood dissociation theory admits that mental trauma plays a central role in the development of dissociative disorders. Last 20-30 years many publications defended that childhood trauma, especially which children experience sexual assault like incest species (rape) to be important factors of occurring the multiple person disorder. This split identity involves considerable deterioration in sense of self and continuity in manageability on the actions of perception and there are changes in emotion, behavior, consciousness, memory, perception, cognition or sensory functionality associated with it. The other identity which appears and completely dominant on that person, is called "divergent" or "the other" (alter identity). The number of alter identity of the individual is the most frequently 2-10. They usually show different personality traits that accustomed of the person's lifestyles. The lifetime prevalence rates such as 1-1.5% have been

reported in North America. So many different rates have been written such as 1-20% in hospitalized psychiatric patients. 5-9 times more common in women than men. In a study that performed in our country on psychiatric outpatients, the prevalence was found 3.9% and 0.4% in the general population.

Keywords: trauma, abuse, dissociative identity disorder

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[Abstract:0511][Psychopharmacology]

An unexpected side effect during escitalopram therapy: GIT bleeding

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Gastrointestinal tract bleeding (GIT) is not an expected Side effect of antidepressant (AD) pharmacological agents. Probability of this Side effect is further decreased in selective serotonin re uptake blockers (SSRI), especially. There is a complex relationship between GIS Bleeding and ulcers. In generally, use of AD has positive effects on GIS ulcers. However, there are some reports that AD causes GIS bleeding. Tendency of not prescribing of SSRI to the patients with GIS ulcers is common. There are not so many studies in literature which investigate the effects of escitalopram on GIS bleeding. Here, a GIS bleeding case who is taken escitalopram is presented just because to wanted to draw attention of clinicians in follow-ups of patients during treatment.

Case: A 37 year-old female patient presented to the outpatient clinic with the symptoms of enervation, crying, tediousness, anhedonia and sleep onset insomnia. Psychiatric examination showed conscious and orientation of the patient was exact. Depressive complaints in process of thought and difficulty to gather attention of patient was recorded. Distressed gesture and mimic, anxious mood and normal psychomotor activity of patient was also seen. There was no thought of suicide and homicide. There were no history of drug use and any physiological diseases. Major Depressive disorder is diagnosed in patient according to DSM-5 criteria. 10 mg/day Escitalopram was started. In his 3rd week follow up, he stated that he presented to the gastroenterology clinic with the symptom of change in his feces. Occult blood test was positive. Escitalopram, only drug he used, is stopped by gastroenterology clinic. After required examinations, reasonable medical explanation of bleeding was found. Occult blood test were negative in routine checkups. Patient had consulted to psychiatry clinic, another SSRI had started. Patient had not mentioned about any side effect during checkups. In the present case, patient whom treated with antidepressant had evaluated by the gastroenterology clinic just because the positive Occult blood test result in the 6. Day of the treatment was observed. Drug was stopped and gastrointestinal and Hematologic diseases which may result to bleeding were also excluded. Escitalopram related bleeding was the possible diagnose, because positive result for occult fecal blood test was disappeared by stopping drug. However, it is not investigated which mechanism causes bleeding by using SSRI's.

Clinical data show that any kind of GIS or hematologic disorders should be investigated before the SSRI treatment. Therefore, clinicians should be more careful if drugs which had bleeding side effects as the long term usage were applied; bleeding parameters should also follow up. Clinicans should be more careful in drug selection of both pre- and post-operative patients. Patients should be informed about psychiatric, metabolic, autonomic, neurological, sexual and gastrointestinal side effects on antidepressants. Drugs should be stopped when bleeding occurs during the SSRI treatment.

Keywords: escitalopram, SSRI, side effect

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[Abstract:0512][Psychopharmacology]

Tic disorder due to modafinil: a case report

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It is known that 15-33% of patients with depression did not respond to the major depression therapy, although combine treatment models were used, therefore, treatment failure in major depression leads both adverse outcomes and severe disabilities. Antidepressant drugs are still in the first choice in major depression cases, some addition therapies are also used in clinicians widely. Although modafinil is approved by FDA for the treatment of hypersomnia that relates some sleep disorders in adults, it is also used as enhancer of efficiency of the treatment. A meta analysis which investigates the modafinil efficiency of major depressive disorders, states that it has effects on fatigue in patients with both uni and bipolar depression. Mechanism of modafinil in major depression is not fully understood, however it is thought that modafinil affects both neurotransmitters, such as dopamine, noradrenalin, glutamate/GABA, histamine, orexin/hypocretin and hypothalamus which plays a role in sleeping pattern and circadian cycles. However, it is known that both physical side effects and exacerbation of psychosis, depression, anxiety, hallucination, mania, suicidal ideas and aggression are seen due to modafinil. Major depressive disorder diagnosed patient with tic symptoms under the modafinil treatment is aimed to present in this case.
Case: A 29 year-old female patient, married, housewife, secondary school graduate patient presented to our outpatient clinic with the symptoms of anhedonia, fatigue and unwillingness. Her symptoms started 3 years ago and she had received psychiatric treatment for 2 years. She had presented to our outpatient clinic 2 months ago, just because she had failed to her treatment. Psychiatric examination showed that, conscious and orientation of the patient was exact, she had distressed gesture and mimic, depressive complaints in content of thought. Her physical examination and routine examinations were normal. Family and her history were normal. Major Depressive disorder is diagnosed in patient according to DSM-5 criteria. Venlafaxine 75 mg/day had started to the patient whose Beck Depression Score was 36. Venlafaxine dose was increased gradually in 150 mg/day within two weeks. She used venlafaxine in two months; however fluoxetine 20 mg/day was started to the patient because of failure of the treatment. Patient had received fluoxetine 40 mg/day for two months; but she stated that she had no benefit on drugs; so duloxetine 30 mg/day had started. Modafinil 100 mg/day was also added on the therapy for the symptoms of profound unwillingness and fatigue, because patient stated a partial improvement on her status. After 3 weeks, patient informed an improvement on her symptoms, thereupon, dose of modafinil was increased to 200 mg/day. One week later, she presented to clinic with the observable tics in only her face. Haloperidol 20 drop/day also added her therapy regime, decreased numbers of tics were observed in follow-up examinations. Tics were totally disappeared after rearrangement her modafinil dose in 150 mg/day.
It is thought that modafinil blocked dopamine transporter (DAT) and this blockage could led into to increase of synaptic dopamine release and to occur of tics. Clinicians should be more careful to prescribe drugs to sensitive patients, especially.
Keywords: modafinil, tic disorder, side effect

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[Abstract:0514][Motor disorders]

A case with camptocormia treated with ECT

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Camptocormia is defined as an abnormal posture involving thoracolumbar flexion of trunk that increases during standing and disappear during recumbent position. Although the condition observed in young soldiers during World War I and II was initially considered as hysteria, it has been described in association with many diseases or conditions, particularly in Parkinson disease. We presented resolution of camptocormia with Electroconvulsive Therapy (ECT) in a patient with bipolar disorder.

Case: A 28 year-old female patient is working as a midwife in a healthcare facility. The patient, who had been following with a diagnosis of bipolar disorder over 8 years, presented to our outpatient clinic with insomnia, using abusive language to her mother, talking to herself and laughing by herself. It was found out that the patient was using lithium (1200 mg/day) and quetiapine (600 mg/day) over 2 years and biperiden (6 mg/day) was added to treatment due to contractions in her arms. In physical examination, a marked kyphosis (camptocormia) was present, which was more prominent during walking and standing. Neurological examination was normal. Biochemical parameters were found to be normal. In psychiatric examination, it was found that she had manic mood with dysphoria and lacking insight; thus, the patient presented to psychiatry ward with diagnosis of bipolar disorder with manic episode. Quetiapine dose was escalated to 800 mg/day. The patient was consulted to physical medicine and rehabilitation department. No apparent cause was found after evaluations. As it was failed to achieve complete response by medical therapy, ECT (7 sessions) was administered to the patient. A dramatic resolution was observed in symptoms of talking to herself and aggressiveness as well as posture. Although camptocormia was thought a psychogenic condition (hysteria) initially, it has been reported that it could be secondary to organic, psychiatric and neurological conditions, medications. There are cases reported in association with bipolar disorder and following antipsychotic use. It was suggested that camptocormia might develop not only in case of dopamine depletion but also in the presence of excess dopamine. There are studies suggesting that camptocormia may be an advanced form of rigidity and dystonia based on pathogenetic factors. In our case, both bipolar disorder and antipsychotic use could be the cause of camptocormia. Although quetiapine is an atypical antipsychotic agent, it is interesting to develop dystonia and subsequent camptocormia during maintenance therapy, which might be due to individual variation. It is important that camptocormia was resolved only after ECT in the patient using biperiden for dystonia. Recently, it was shown that ECT is beneficial in motor disorders such as oromandibular dystonia, blepharospasm and tic disorders.

It should be kept in mind these rare conditions, as in our case, could be due to an underlying psychiatric disorder and that ECT could be a treatment option in such cases.

Keywords: bipolar disorder, camptocormia, ECT

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[Abstract:0516][Impulse control disorders]

Rasagiline as adjunctive therapy induced impulse control disorders in parkinson's disease

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In recent years there are an increasing number of impulse control disorders (ICDs), such as pathologic gambling, hypersexuality, compulsive shopping, compulsive eating, punding and compulsive medication use related to aberrant or excessive dopamine agonist treatment of Parkinson's disease (PD). Here we report the case of ICDs become apparent from rasagiline treatment in PD patient.

Case: A 43 year-old man with a ten year history of idiopathic PD. He worked Computer Company as an operator and he was married. He was taking piribedil retard tablet 50 mg three times. With the initiation of a rasagiline (MAO-B inhibitor) some 4 years ago, impulsive behaviors began to arise of which hypersexuality has been the most problematic. He occurred an extremely desire for sex. His wife has become complained that he was obsessed with daily sexual coupling and she came to the divorce process. According to his wife's testimony that doing too much unnecessary shopping, excessive credit card use, the opportunity was spending a lot of time on the internet. It was difficult to adapt to patients in terms of treatment and follow-up it lasted irregularly. It is uncommonly reported hypersexuality that known is side effects of anti-Parkinson medications. Rasagiline blocking MAO-B enzyme allows the increase of dopamine in the synaptic gap. With the increased dopaminergic stimulation occurs dysfunction in neuroanatomical circuit containing on ventral tegmental area and related frontostriatal region. Including dopaminergic stimulation presumed abnormal or exaggerated behavior associated cluster, impulse control disorders is increasingly being defined by the day. Inability to resist the fascination which will be harmful to the basic features of an action or impulse him or others are described as a group of diseases in DSM-IV.

A resting tremor of present unlike sex, gambling and shopping is an activity common in everyday life. So problematic behavior is seen as a deviation from normal and can occur in the form of characters. ICDS is known that more young-onset patients seen. The prevalence of ICDs in PD has been reported to be approximately 6.6%.

The treatment of ICDs patients is difficult, and response to treatment is limited. During the examination of patients and their families

should be informed about these issues. Especially before the onset of the PD in patients with early-onset, family member with alcohol abuse disorders and impulse control is important in identifying patients at risk group of patients. This disease is a clear condition requiring cooperation between psychiatrists and neurologists for optimal care and research.
Keywords: dopamine, hypersexuality, impulse control disorders, parkinson disease, rasagiline

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[Abstract:0519][Psychopharmacology]

'I am a bad child!' is an obsession or a normal pubertal process?

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Puberty is an important, cognitive and social transformation process, involving physical, sexual and psychological development which extends from end of childhood to adult age. Provision of sexual information and sexual interest are among characteristics of normal developmental process at puberty. However, it's seen as a taboo to discuss sexual issues within family due to social and cultural reasons. It's known that parents often avoid to provide sexual education, and if do not, they usually avoid from issues such as masturbation and sexual pleasure which adolescents are interested and that peers' knowledge is insufficient in these issues. Here, we presented a patient with normal puberty who perceived himself as a bad child because of his sexual attitudes and behaviors.
Case: A 13 year-old male adolescent presented to our outpatient clinic by parents. Parents emphasized that their child experienced a challenging puberty; that he was fearing and crying due to changes in his body; that he had concerns about sexuality; that he was irritable and unhappy; that he perceived his experiences regarding puberty as negative and shared these experiences with them by crying; that they do not know how to behave;and that they did not give information about puberty to their child so far. In the interview with adolescent, he told that he masturbated after watching a sex movie with a boyfriend one year ago; that he always think about sexuality and occasionally masturbate since that day; that he's afraid of talking with girls; that he doesn't want to sleep due to sexual dreams; that he feels uneasy if he did not share sexual excitements and dreams with his mother; that he think that he remains as a bad child if he hides these experiences;and that he had inattention to the lessons, loss of appetite and self-expressions "I am a bad child" and that he had no other obsession. Based on psychiatric examination, psychological problems related to puberty and obsessive-compulsive disorder were considered as initial diagnoses. The patient was informed about physical and sexual changes experienced in puberty. Recommendations were given to parents,including need for provision of information to the child regarding puberty and sexuality in a manner appropriate to his age. In the follow-up visit after 2 weeks, it was reported that there was improvement in symptoms by 90% and after 3 months no symptoms of obsession or anxiety were observed.
Provision of accurate information about sexual issues to adolescents is important for development of sexual identity and not facing negative consequences of sexual attitudes and behaviors. Accurate information ensures recognition of his/her body and its features by child and improves self-confidence. Child informed about bodily changes experienced during puberty can yield this differentiation more quickly, resulting in less anxiety and fear about abnormality. In our case, it was thought that being not informed by his family about psychological, physical and sexual changes during puberty and being unprepared to pubertal process were involved in experiencing sexual obsession and anxiety. In parents, the reason underlying avoidance to provide sexual education to their child is being ashamed of talking about sexuality and not feeling well-informed. In order to help experiencing a healthy puberty, information about puberty and sexuality should be provided to children in a manner appropriate to their age. Being prepared to changes experienced will improve adjustment to this process.
Keywords: normal puberty, anxiety, obsession

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[Abstract:0521][Substance-related and addictive disorders]

Oxybutynin addiction: a case report

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Oxybutynin has antimuscarinic, direct muscle relaxant and local anaesthetic actions which is used to treat overactive bladder and nocturnal enuresis. Anticholinergic abuse in the population with chronic mental illness is a common problem, however the dependency of oxybutynin which has relatively low anticholinergic effect is an unknown matter. Anticholinergic drugs are abused for their euphoric and stimulant effects, at the same time usefulness of the mood elevating act. Oxybutynin also has a high inclination to cross the blood-brain barrier and has been associated with cognitive impairment.
Case: A 35 year-old male patient who was referred to our clinic for assessment in the judicial process has presented. The patient has an alcohol and substance abuse for almost twenty years and his preferred drug is oxybutynin for the last ten years over other anticholinergic agents which have been prescribed before within the neuroleptic medication. He has been diagnosed as substance -induced psychotic disorder and has cognitive impairment due to substance abuse. He had discovered oxybutynin's euphoriant effect after his friend in the prison suggested him for more intensely effect than biperiden. Oxybutynin can be obtained easily without prescription in Turkey and this case is presented to draw attention to the oxybutynin dependency which is a growing problem.
Keywords: addiction, anticholinergic, oxybutynin

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[Abstract:0524][Autism]

Childhood disintegrative disorder: case reports

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Childhood disintegrative disorder (CDD) is a rare and severe form of pervasive developmental disorders. A developmental regression between ages 2 and 10 is generally observed in patients after normal psychomotor development. We present here three male cases that are diagnosed as having childhood disintegrative disorder in accordance with DSM-IV criteria.
Case 1: A 5 year-old boy presented to our clinic. His psychomotor development was normal until 4 years. At age 4, deficits in social-emotional reciprocity, social withdrawal, stereotypic behaviors including echolalia, dysprosody, language impairments, loss of eye contact, loss of play skills, irritability began. He had no neurological, metabolical and auditory abnormalities. He was referred to behavioral treatment and risperidone was initiated.
Case 2: A 6 year-old boy presented to our clinic. His psychomotor development was normal until 6 years. He began to see objects on the walls and began to fear from pipes. He refused to go to school. He started to play with the wheels of the cars. Socio-emotional reciprocity was lost. He started to attack other children without any reason. Purposeless and stereotypical behaviors such as clapping hands began. He began to refuse playing games. He had no neurological, metabolical and auditory abnormalities. Loss of previously acquired language and play skills have been observed. He also had self mutilative behaviors such as hitting himself. He was referred to behavioral treatment and aripiprazole was initiated.
Case 3: A 4 year-old boy presented to our clinic. His psychomotor development was normal until 4 years. One months after a febrile infection, social withdrawal, loss of socio-emotional reciprocity, loss of play skills had begun. He started biting objects, clapping hands, sucking thumbs. Verbalisation had been completely lost. He began to extract meaningless letters. He had no neurological, metabolical and auditory abnormalities. This patient also lost his bladder control. Risperidone was initiated for hyperactivity and irritability.
Data are scarce about the etiology, prognosis, and treatment of CDD. There is also a debate about the validity of the diagnosis. The DSM-5 brought dramatical changes in the pervasive developmental disorders (PDD) section. CDD is deleted and merged into the diagnosis PDD-Not Otherwise Specified. Although scientific evidence was found poor for CDD, it has some different features including the acuity and severity of regression, as well as co-occurring physical symptoms, such as loss of bowel and bladder control. More clinical observation

and experience is needed for the differential diagnosis. CDD may also be considered as a different diagnosis in the light of the future observations and further clinical investigation is suggested.
Keywords: autism, childhood disintegrative disorder, pervasive developmental disorders

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[Abstract:0525][Psychopharmacology]

Metylphenidate and panic attacks: a case report

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Attention deficit and hyperactivity disorder (ADHD) is a common heritable disorder with persistent patterns of inattention, hyperactivity, and impulsivity. Methylphenidate (MPH), significantly increasing extracellular dopamine in the human brain, is the most commonly prescribed psychoactive drug for ADHD. In a review study 25% of participants reported minor adverse events that mostly related to sleep or appetite. Some minor adverse events can fright the patients, which can be a common events. After treatment with methylphenidate, although there are studies showing that higher levels of anxiety, but there aren't any studies with panic attacks. Panic attacks demonstrate itself palpitations, tremor, sweating, and an emotional sense of death. Inhere patient with panic attacks are presented after starting MPH for ADHD.
Case: A 18 year-old male patient presented our outpatient clinic, because of inability to study, inability to concentrate, lack of attention. After the examination he was diagnosed ADHD according to the DSM-5. He had received a diagnosis of ADHD for the first time and had no other psychiatric symptom. OROS methylphenidate was prescribed 20 mg once a day in mornings for ADHD treatment. In the evening of the first day of taking MPH, he presented an emergency room with palpitation and emotional death sense. The etiology of panic disorder is not currently known. In a subgroup of panic disorder patients, higher plasmatic concentration of homovanillic acid, which is a dopamine metabolite, was found. In the emergency room and cardiac examinations, results were normal, and complaints were decreased without any intervention when he was in hospital. Patient's ECG, levels of thyroid hormones and other biochemical measurement were normal. After the day he did not take MPH and there was no symptoms. In the second day of taking MPH, he experienced the same complaints and hospital process again. Then he presented to his psychiatrist because of panic attacks.
The etiology of panic disorder is not currently known. In a subgroup of panic disorder patients, higher plasmatic concentration of homovanillic acid, which is a dopamine metabolite, was found. He had panic attack symptoms such as fear of death, sweating, palpitation. So we think he had panic attacks after taking MPH. MPH increases DA in the brain, and in a study higher level DA metabolites were seen in panic disorder which has repetitive panic attacks. So it can explain patients' symptoms. According to our knowledge in literature, there was not any patients with panic attack after using methylphenidate. Inhere we aim to discuss to role of MPH on panic attack.
Keywords: ADHD, methylphenidate, panic attack

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[Abstract:0526][Psychopharmacology]

Digoxin intoxication in a geriatric patient with normal blood drug level

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Digoxin is a commonly used drug in the treatment of heart failure and rhythm control of atrial fibrillation. Therapeutic index is narrow and there are various factors that can trigger intoxication. Most common digoxin intoxication symptoms are nausea (54.8%), fatigue (42.9%), vomiting (33.3%), and loss of appetite (28.6%) but definitive diagnosis is based on the blood level measurement. We report a digoxin

intoxication case with normal blood digoxin level in a geriatric patient.
Case: A 74 year female patient was admitted to a tertiary care setting's Geriatrics clinic with complaints of nausea, loss of appetite, weakness, and chest pain. The patient had a past medical history of hypertension for 10 years, atrial fibrillation for 3 years, and diabetes mellitus for 6 years. Treatment was included Valsartan (160 mg/day), Hydrochlorothiazide (12.5 mg/day), Vildagliptin (50 mg/day), Metamorphine (850 mg/day), Glimepiride (1 mg/day), Digoxin (0.25 mg/day), Diltiazem (120 mg/day), and Warfarin (5 mg/day). The physical examination showed a temperature of 37.3ºC, blood pressure of 140/80 mmHg, and pulse rate of 58 beat/min (dysrhythmic). Inspiratory rales were auscultated in the right lung but pretibial edema was bilaterally negative. Neurological examination and cognitive assessment were correlated with the age (MMSE:25). In ECG, there were atrial fibrillation and AV block. Laboratory results were as follows;blood glucose: 155 mg/dL (65-107 mg/dL), creatinine: 1.19 mg/dL (0.81-1.40 mg/dl) Sodium: 137.6 mmol/L (135-145 mmol/l), Potassium: 4.1 mmol/L (3.5-5.5 mmol/L), calcium: 9.53 mg/dL (8.5-10.5 mg/dl),magnesium:1.54 mg/dL (1.9-2.5 mg/dl), INR: 2.1, sedimentation: 58 mm/h, TSH: 1.56 µIU/mL (0.5-4.8 µIU/mL) T4: 1.13ng/dL 0.7-2.0 ng/dL, T3: 2.44 pg/ml (2.60-4.80 pg/mL), Estimated Glomerular Filtration Rate: 59 ml/min (70-120 ml/min HgbA1c: 8.4% (4-6) in total urine examination, density: 1020, microscopy: 1-2 white blood cells and blood digoxin level: 1.9 ng/ml (0.8-2.0). 500 mg/day Levofloxacin and 365 mg/day magnesium were prescribed with the diagnosis of pneumonia and hypomagnesemia. Patient's complaints were not regressed after one week and it was considered that current clinical picture of case could be intoxication.
Hypokalemia and hypomagnesemia can cause an increase in digoxin sensitivity of myocardium. Lack of magnesium causes increase in digoxin uptake of myocardium, therefore, pump is inhibited because it is a cofactor of Na/K ATPase channel.
Therapeutical blood level of digoxin is 0.8-2 ng/ml. It is excreted by renal clearance, it is seen that there is an increase in intoxication risk during use of some drugs (such as amiodarone, verapamil) due to renal impairment and common drug interactions. Digoxin intoxication can occur in hypomagnesemia even with normal blood levels. Intoxication is a clinical condition that must be always considered in digoxin use. There were some causes that lead to increase in risk of intoxication including older age, female sex, diuretic use, and impairment in renal functions. It should be taken into consideration that digoxin intoxication signs can be overshadowed by other comorbid conditions of the patient and serum digoxin leves do not always correspond to this condition.
Keywords: digoxin intoxication, magnesium deficiency, elderly patients

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[Abstract:0527][Psychopharmacology]

ECT in adolescent pregnancy: a case report

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Electroconvulsive therapy (ECT) is a therapeutic modality that has been long used in the management of some psychiatric disorders in adult patients and pregnant women. As it is rarely used in children and occasionally in adolescents,there is limited data regarding its effectiveness and side effects in this age group. However, available data indicates that indication, safety and effectiveness in children and adolescents are similar to those in adults. There is a risk for worsening in affective disorders and increased relapse during pregnancy. Drugs that could be used in such case can cause teratogenicity, toxicity, perinatal syndromes in fetus through transport via placenta and withdrawal syndrome. ECT has been reported as rapid,safe and effective treatment option in the management of major depressive disorder,bipolar disorder and schizophrenia in adult pregnant women. Effects of ECT in first trimester of pregnancy aren't well understood. Since first 8 weeks of gestation is considered as embryogenesis period,it is not preferred to use anesthetic agents and ECT can be safely used in second and third trimester of gestation. To best of our knowledge,there is no case of adolescent pregnancy underwent ECT in the literature;thus,we presented our case.
Case: A 17 year-old female patient presented with meaningless speech, insomnia and irritability by her husband. He reported that they married 10 months without permission of their family; that she was pregnant (gestational age: 18 weeks); that she had no psychiatric complaints until 10 days ago but she had insomnia, anxiety, decelerated movements and perception, failure to communicate, timidity, meaningless speech and laugh by herself, walking in the night by awaking from sleep, hearing voices and seeing frightening visions. In the psychiatric assessment, it was found that there was marked deceleration in speech, talking about irrelevant details independent from subject during interview, inappropriate answers to questions, impaired time orientation, visual and auditory hallucinations, reference delusions and blunted affection. There was no abnormal finding in personal and family history. Based on these findings, the patient was diagnosed as psychotic disorder and informed about treatment options, medical therapy and risks. The patient and her relatives declined medical therapy due to pregnancy-related risks. The patient was referred to another facility as the patient preferred ECT. On the follow-up visit after one

month, it was found out that the patient was admitted to hospital for 3 weeks and received 7 session of ECT without complication.
In children and adolescents,it is generally recommended to use ECT after treatment attempts such as psychotherapy and/or medical therapy. In this case, the patient referred to another facility for ECT since relatives of the patient preferred ECT despite recommendation of medical therapy. In psychiatric examination,it was seen that there was significant improvement in complaints of the patients after 7 sessions of ECT and that there was no ECT-related complication. Currently, the patient at 7.5 months gestation has no complaints and attends to psychiatric follow-up. In small series on children and adolescents,it was reported that ECT is effective and safe by 61-100%; however, further studies are needed on this topic. In adolescent pregnancies, ECT can be considered as an alternative treatment modality in patients declining medical therapy or in case of failure to achieve effective response with medical therapy.
Keywords: adolescent, pregnancy, ECT

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[Abstract:0528][Mood disorders]

Varenicline -induced mania with spontaneous recovery

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Late-life onset manic attacks generally occur secondary to general medical conditions or drug use. Varenicline is an $\alpha_4\beta_2$ nicotinic acetylcholine receptor partial agonist, used for the cessation of smoking.
Case: A 39 year-old male patient with a new onset manic episode following varenicline treatment. The patient's manic symptoms (insomnia, excessive shopping, increased libido, nonprotected sexual relationship, increased psychomotor activation) elicited on the fifth day of varenicline treatment that was used for giving up smoking. He has a depression history and he was using venlafaxine 75 mg/day for nine months. He had no manic episode before varenicline treatment. After manic symptoms, he stopped varenicline treatment and continue to use venlafaxine. The patient presented our psychiatry outpatient clinic 20 day after stopping varenicline. The manic symptoms, except insomnia, regressed spontaneously without any medical treatment. Varenicline may cause manic episodes in patient with bipolar disorder and healthy individuals. In our case the patient's depression history may be a vulnerability factor for manic episode. Its important to evaluate psychiatric and mental disorder history before varenicline treatment.
Keywords: varenicline, manic episode, smoking cessation

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[Abstract:0532][Others]

Auditory hallucinations after acute stroke: a case report

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Auditory hallucinations are described as auditory experiences without the presence of external stimuli. They are most strongly linked to schizophrenia but are also characterized in patients with organic brain syndromes, Alzheimer disease and epilepsy, as well as in normal individuals. Acute stroke is a neurological emergency and can lead to ischaemia of the auditory pathways, resulting in bizarre, positive auditory hallucinations. On the other hand auditory hallucinations can occur mostly soon after lesions of the brain stem and remit soon after. In the literature there is very limited reported cases with prolonged auditory hallucinations. According to our knowledge this is the first case report with auditory hallucinations had been still continued after 10 months of post-cortical stroke.
Case: An 80 year-old right handed female patient, suffering from auditory hallucinations was admitted to our psychiatry clinic. The hallucinations were about an unfamiliar male voice that was speaking with other people. Voices were not very frightening and she did not define her condition as anxious, but as bizarre and unpleasant. She had poor insight to the hallucinations and was sometimes speaking

to herself, telling that she was responding to the voices. She was oriented and cooperative on mental status examination. Her emotional mood was euthymic and her affect was appropriate. She did not exhibit any formal thought disorders. She was able to answer questions spontaneously and directly but she could not hear clearly because of hearing problems. Her attention was normal. She did not have any memory deficits. In her anamnesis it was learnt that she had had a stroke 10 months ago before her admission. In emergency department, neurological examination had revealed moderate left hemiparesis with hemihypoesthesia. Acute ischemic infarct of the right insula and right temporal lobe had been shown by the brain Magnetic Resonance Imaging (MRI). Neurological symptoms had been disappeared in three months. This was the first stroke attack in her life. She had long-standing history of good health and no previous psychiatric disorder. Her routine hematological and biochemical parameters were within normal limits. EEG examination showed no epileptic discharges with temporal region on a normal background. Mini Mental State Examination (MMSE) was scored 28/30. There was no other psychiatric signs and symptoms so that all other psychiatric disorders were ruled out.

In the literature, there are some limited case reports about auditory hallucinations developed after acute stroke. In these reports, it was shown that auditory hallucinations were appeared in cortical strokes of both dominant and non-dominant hemispheres. It is suggested that an important aspect of auditory verbal hallucinations, namely the experience of the alien or non-self origin, results from an imbalance between the anterior cingulated gyrus and the anterior insula in the right hemisphere. In the presented case, the infarction was localized on the right insula and right temporal lobe which was non-dominant hemisphere.

In conclusion, this case shows that auditory hallucinations may prolong long after the stroke. Understanding the neurobiology of the non-self aspect would therefore be helpful in elucidating the pathophysiology of auditory verbal hallucinations.

Keywords: auditory hallucinations, acute stroke, post-cortical stroke

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[Abstract:0534][Others]

Neuroleptic malignant syndrome: a case report

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Neuroleptic Malignant Syndrome (NMS) occurs rarely due to antipsychotic medication and can be fatal in some circumstances. Muscle rigidity, hyperthermia, autonomic dysfunction, elevation in serum creatine phosphokinase (CPK) and transaminase levels, leukocytosis and changes in consciousness can be observed. It is usually described as an adverse event in high dosages and long-term usage especially with depo neuroleptics. Herein we present a NMS case developed after single intramuscular application of 10 mg haloperidol.

Case: A 21 year-old male patient with psychotic symptoms was hospitalized to the psychiatry clinic. At admission, the patient's vital signs were stable. Mutism, stereotyped movements, stupor, agitation and negativism were observed. He was obviously dehydrated. He was disorganized and excited, thus haloperidol of 10 mg was administered to control the symptoms. In his anamnesis, it was learnt that four months ago, first auditory hallucinations were begun and other symptoms were seen afterwards. Emergency blood biochemistry and hemogram results were in normal ranges. 14-16 hours after the injection, rigidity, confusion, fever (38.6^{o}C), excessive sweating occurred. There was not neck stiffness. Babinski reflex was negative, light reflex was normal and there was no evidence of lateralization. Laboratory results were as follows: White blood cell:13,500 K/mm^3 (4.5 -11,00 K/mm^3), CPK 4564 U/L (52-336 U/L), myoglobin 2546 ng/mL (0-85 ng/mL), bilirubin total 1.55 mg/dL (1.0-1.5 mg/dL), direct bilirubin 0.37 mg/dl (0-0.2 mg/dl), Protrombin time test (pTT): 20 seconds (10-14 second), International Normalized Ratio (INR): 1.37 seconds (0.9-1.2 second), aspartate amino transferase: 157 U/L (15-40 U/L),alanine amino transferase 41 U/L (10-40 U/L), pTT 15.9 second (25-36 second), C-reactive protein (CRP): 9 mg/L (0-4.9 mg/L), Anti-streptolysin O: 183 lU/ mL (0-200 lU/ mL), microbiological markers (Anti-HBsAg, HbsAg, Anti-HIV, Venereal Diseases Research Laboratory (VDRL), Rapid Plasma Reagin (RPR), HSV Type -1 IgG and IgM, rubella IgM,Anti- toxoplasma IgM) were negative and thyroid hormones, vitamin B12 and folate levels were in normal ranges. Diffusional Magnetic Resonance Imaging and Computerized Brain Tomography scan were reported as normal. Lumbar puncture and cerebrospinal fluid analysis could not be done due to increased pTT and INR. Within 2-3 days of follow-up and supportive treatment, his consciousness was improved. Laboratory findings were all returned to normal ranges. The patient underwent electroconvulsive therapy for eight sessions and psychotic symptoms were remitted.

Here, we highlight that NMS is a serious adverse effect that may occur even after single dose of haloperidol. In the literature it is reported that NMS occurs in 0.2% of patients treated with a neuroleptic and it develops in 30% of patients within 48 hours of the onset of treatment. Unless it is treated; acute renal failure, aspiration pneumonia, respiratory distress syndrome, disseminated intravascular coagulation and cerebral neuronal degeneration may occur. Early diagnosis, cessation of antipsychotics, effective supportive treatment are very important.

Hydration should be ensured due to the increase of muscle breakdown products and kidney function tests should be monitored closely for renal insufficiency.
Keywords: neuroleptic malignant syndrome, haloperidol, muscle rigidity

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[Abstract:0536][Psychopharmacology]

Resolution of diurnal enuresis with fluoxetine

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Enuresis has been defined as an intermittent wetting during sleep or daytime in children after their fifth birthday. Daytime wetting is also termed urinary incontinence. Both nocturnal enuresis and enuresis during daytime can be organic (structural, neurogenic or due to other physical causes) or functional.
Fluoxetine has been approved for the treatment of major depressive and obsessive compulsive disorders in children and adolescents. There is also substantial evidence about its efficacy on childhood anxiety disorders. Several previous case reports have shown improvement in nocturnal enuresis during fluoxetine treatment. Herein, we report resolution of enuresis during daytime with fluoxetine in an adolescent with social anxiety disorder.
Case: A 16 year-old female adolescent was referred to the child and adolescent psychiatry out-patient clinic for her social phobia. She had excessive anxiety in everyday social situations which was limiting her daily life. She also reported that she had enuresis which was occurring during daytime four or five times per week since 7 years of age. She was unable to hold her urine and leaked totally before going to the toilet. Her medical work-up, including urinary examination and radiological findings was normal. Because of her anxiety symptoms, we initiated fluoxetine 10 mg/day and increased the dose to 20 mg/day 3 weeks after. In her second visit 7 weeks later, she reported that her social anxiety diminished and she was feeling more comfortable during interaction with her peers. She also reported that the frequency of her enuresis decreased during the first 3 weeks and disappeared totally after the dose was titrated up to 20 mg/day. She did not experience enuresis during fluoxetine treatment for 4 months.
We presented a case with diurnal enuresis that had remitted after fluoxetine which was initiated for her social anxiety. Improvement on her enuresis was dose dependent and absolute resolution of enuresis was achieved during 20 mg/day dose of fluoxetine. In the literature there are reports on nocturnal enuresis improved with fluoxetine and sertraline-induced enuresis that was resolved after switching to fluoxetine. To our knowledge, this is the first reported case of diurnal enuresis remitted with fluoxetine. Although its mechanism of action is unclear, fluoxetine may have anti-enuretic effect due to its central pre-synaptic serotonin-1A (5-HT1A) antagonism and peripheral serotonin-3 (5-HT3) agonism. It was suggested that 5-HT1A antagonism inhibits bladder reactivity and 5-HT3 agonism inhibits urethral peristalsis and micturation via spinal reflexes. Enuresis may also be associated with central dopaminergic and noradrenergic dysfunction. Several drugs that increase noradrenergic (e.g. atomoxetine) and dopaminergic (e.g. stimulants) activity were reported as effective in enuresis. Fluoxetine may also improve enuresis by increasing central dopamine and noradrenalin via its serotonin-2C (5-HT2C) antagonism. In conclusion, this case suggests that fluoxetine may improve, in addition to nocturnal enuresis, diurnal enuresis.
Keywords: adolescent, enuresis, fluoxetine

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[Abstract:0538][Dementia syndromes]

History repeats itself: dementia due to neurosyphils

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Syphilis is one of the rare venereal disease treated with penicillin which is the only drug proven effective in the treatment. It is diagnosed many years after the first infection and it has a progressive course. It seems quite appropriate to define syphilis as "the great imitator" because it mimics different diseases. It can mimic psychiatric disorders such as dementia, depression, mania and psychosis. Despite penicillin treatment and reduction in the incidence of neurosyphilis; the incidence of the disease and related complications has been begun to rise in parallel with HIV positivity especially in European countries and United States. Tertiary or late syphilis develops decades after the initial infection and can affect any organ system. Neurological influence is seen in 10% of patients with untreated syphilis. Here, a case with dementia and personality changes due to neurosyphilis is reported. Differential diagnosis, monitoring and early interventions of dementia are also highlighted.
Case: A 71 year-old male patient was admitted to hospital in order to be examined. He had no complaints. He said he agreed to settle with his wife's insistence. According to his wife, he had been having less conversation, he was not interested in things that he had been interested before and he had become quite forgetful. He had been acting as if he did not care about anything. The duration of his sleep had decreased and it was usually interrupted. He has lost his appetite. According to his colleagues he had been preoccupied for many years. Ten days before the hospitalization he had fainted and vomited. He had no urinary incontinence or convulsions. He had not fainted before and it did not repeat after the first time. His premorbidity was very playful and talkative. His general appearance and self-care were found decent in the psychiatric examination. His hair was neatly combed. He had vacant eyes. His consciousness was clear and time orientation was impaired. His emotional expression was limited. He was facing psychomotor retardation. His recent memory was impaired, his judgment was weakened. We administered Mini Mental State Examination (MMSE) and it revealed significantly deterioration in cognitive functioning with 24 points as a result. EEG scale was in normal limits. Cortical atrophy was found in CT scan. Syphilis serologic testing (+) and TPHA: 1 /1280. Cranial MRI was reported as minmal cortical atrophy. Lumbar puncture was performed. Cerebrospinal fluid VDRL (+), TPHA: 1/2650, sugar 70% mg /dL, 60% protein, mg/dL, and the patient was diagnosed as neurosyphilis. He was consulted to infectious diseases and penicillin treatment was started. There was an increase in amount of communication during the patient's psychiatric examination at the end of the month. After six months MMSE scores were raised to 28 points. This case of neurosyphilis shows that neurosyphilis can mimic the symptoms of many diseases.
Keywords: neurosyphilis, dementia, psychiatric disorders

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[Abstract:0540][Psychopharmacology]

Tardive dyskinesia in an adolescent girl with autism spectrum disorder and Tourette's disorder

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Tardive Dyskinesia (TD) is a potentially irreversible, sudden, involuntary movements of lips, tongue, trunk and extremities seen as a long term side effect of neuroleptic medications. Rates of developing TD due to use of second generation antipsychotics are consistently low when compared to first generation antipsychotics. Unlike studies conducted on adults, there are only few data about its prevalence and presentation in children and adolescents. Here we present a 16 years-old girl diagnosed with Autism Spectrum Disorder (ASD) and Tourette's disorder on risperidone treatment for six years who developed tardive dyskinesia and remitted her symptoms after switching into clozapine.
Case: A 15 year-old female patient was brought to the Child and Adolescent Psychiatry outpatient clinic by her parents with symptoms of aggression, rocking, squeezing her hands, blinking, sniffing and obsessions such as closing doors, windows, switching off lambs.

She had been previously diagnosed with ASD and had special education since she was two and a half. At 10 years of age she was started risperidone for hyperactivity and aggression. Upon her initial assessment and psychiatric examination, she was diagnosed with ASD, Tourette's Disorder and Attention Deficit and Hyperactivity Disorder. She was treated with daily treatment of risperidone 2.5 mg, fluoxetine 20-40 mg, atomoxetine 18 mg and. bornaprin 8 mg After one year, small, involuntary dyskinetic movements around her mouth was observed in her neurological examination. The movements were entirely different from her pre-existing tics both anatomically and phenomenologically. The family reported that these movements started one month ago and did not show any remission. Upon deciding that she developed tardive dyskinesia associated with long time use of risperidone, it was decreased gradually and stopped. Treatment with clozapine was started and gradually increased up to 100 mg twice a day after examining complete blood count, lipid profile, liver and kidney function tests. After two weeks following this intervention, family reported improvement in her dyskinetic movements and complete resolution at third week. During her follow-up of eight months, dyskinetic movements did not appear again and improvement in her other symptoms were recorded.

TD is a socially disabling and sometimes incurable form of movement disorder related to long term use of dopamine blocking agents. While it is clearly established that the chance of developing TD is lower in second generation antipsychotics than the first generations, its association with younger population and particularly with children and adolescents diagnosed with autism spectrum disorder and tic disorders remains unclear. To our knowledge, this is the first report of TD that developed following long term risperidone use in a child with ASD and comorbid Tourette's disorder.

Keywords: tardive dyskinesia, young population, risperidone, autism spectrum disorder

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[Abstract:0543][Psychotherapies]

Confusion associated with valproate and quetiapine combination: a case report

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Combination therapy with valproate plus quetiapine is recommended as one of the first-line approaches to treatment of manic or mixed episodes in patients with bipolar disorder. This combination of drugs may have possible synergistic effects. However, using valproate together with quetiapine may increase side effects such as dizziness, drowsiness, confusion and difficulty concentrating. Here in, we report a case of confusion associated with valproate and quetiapine combination treatment for bipolar disorder.

Case: A 44 year-old female patient, graduated from elementary school, housewife, married, has two children. She presented to emergency service with complaints of getting angry rapidly, decrease in the need of sleep time, increase of self-esteem and speech, restlessness, scepticism. She had twenty-four year of bipolar disorder history, hospitalized five times before, not regularly followed up and used only lithium 300 mg/day irregularly. Alcohol or substance usage were not stated. Any pathology was not detected in personal or family history. In psychiatric examination, increment in psychomotor activity, defensive, reckless, elevated in mood, irritable in affect, increment in speech rate, quantity, acceleration of association, delusions of persecution, paranoid, jalousie, decrement in impulse control. She was admitted to our service with the diagnosis of "Bipolar disorder, manic episode with psychotic features". After intramuscular injection haloperidol 20 mg/day, biperiden 2x1, chlorpromazine 50 mg/day for 10 days, then their oral forms were continued. Due to ongoing excitation and treatment resistancy, nine sessions of electroconvulsive therapy was administered. After electroconvulsive therapy, haloperidol and biperiden was stopped gradually, valproate 1000 mg/day, quetiapine 300 mg/day were started and quetiapine increased up to 900 mg/day rapidly. On the third day of valproate and quetiapine combination, due to development of hypotension and sedation, chlorpromazine was stopped, quetiapine was decreased to 600 mg/day. Parenteral fluid was given. When the confusion became more evident, valproate and quetiapine were stopped. In computer tomography and magnetic resonance imaging of cranium and electroencephalogram, any pathology was not detected. Laboratory results were normal. The confusion was associated with rapid increase up to high dose of valproate and quetiapine combination. After discontinuation of drugs, confusion was regressed in three days, manic symptoms reappeared. New treatment was regulated as valproate 750 mg/day, risperidone 8 mg/day, biperiden 4 mg/day, olanzapine 20 mg/day. Manic symptoms were regressed and any problem was not observed. She was discharged with this treatment.

Several studies have addressed a possible interaction valproate and quetiapine, and it appears that valproate may somewhat increase quetiapine plasma concentrations and attribute this effect to valproate inhibition CYP3A4 and CYP2D6 activity. Clinical reports of interactions between valproate and quetiapine leading to an increased risk of adverse drug reactions have also emerged. Confusion and delirium in patients treated with quetiapine seems to be a rare phenomenon; however, Huang CC and Wei IH report two patients

with bipolar disorder who developed delirium when prescribed quetiapine as an adjunct to valproate for acute mania. As seen our case, valproate and quetiapine combination treatment should be used with caution due to possible side effects; and clinicians should avoid rapid dose escalation.

Keywords: confusion, quetiapine, side effects, valproate

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[Abstract:0544][Psychopharmacology]

Agomelatine in the treatment of post-stroke depression: a case report

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In recent years, post stroke depression has attracted worldwide interest. This review focuses on the major research themes that have emerged. Pooled data from studies conducted throughout the world have found prevalence rates for major depression of 19.3% among hospitalized patients and 23.3% among outpatient samples. The diagnosis of post stroke depression is most appropriately based on a structured psychiatric exam and DSM-IV criteria for depression due to stroke with major depressive–like episode or depressive features. The treatment of post stroke depression has been examined in severalplacebo-controlled randomized clinical trials with both nortriptyline and citalopram showing efficacy. The progression of recovery following stroke can be altered by treating depression, which has been shown to improve recovery in activities of daily living and cognitive impairment and to decrease mortality. In this case, we treatment post stroke depression with a novel mechanism of agomelatine.

Case: A 83 year-old female patient presented as an emergency because of sudden left-sided hemiplegia and confusion. Her heart rhythm was atrial fibrillation (155 beats/min), and he was hypertensive (180/110 mm Hg). A MR diffusion performed on admission showed ischaemic infarct in the right total middle cerebral arter territory. After second week of admission, the patient was given agomelatine 25 mg p.o with nasogastric canul for depression mood (due to criteria of DSM-4). After 6 weeks of treatment the regression of clinically significant fatigability, increase in the efficiency of work, normalization of the coefficient of mental health and improvement in sleep are noticed. After 8 weeks of treatment left sided of patient had neuphatic pain seen relatively to her infarct. Antidepressant treatment changed to duloxetine. After 2 week of duloxetine treatment her mental health was decreased and improve daytime sleepiness.

The unique mechanism of action of agomelatine accounts for its potential benefits in disorders other than major depressive disorder (MDD). In fact, agomelatine's sleep-promoting and chronobiotic actions mediated by MT1 and MT2 receptors in the suprachiasmatic nucleus as well as its effects on the blockade of 5-HT2c receptors, are beneficial in several disorders. Moreover, agomelatine may be potentially useful in the treatment of partial-responder schizophrenia, alcohol-dependent associated sleep disturbances, fibromyalgia and migraines.

Safety profile of agomelatine, including the incidence of its serious adverse events, was satisfactory. The most common observed adverse effects were headache, dizziness, somnolence, diarrhea, nausea, sedation, fatigue, and insomnia, but all were in the mild-to-moderate range. As the mechanism of action of agomelatine is not associated with increased serotonin levels, its adverse events profile is different from SSRIs and SNRIs, particularly regarding weight gain, headaches, sexual dysfunctions, psychomotor agitation, and serotonin syndrome.

Keywords: agomelatine, cerebrovascular disease, post-stroke depression

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[Abstract:0545][Dementia syndromes]

ECT treatment for frontotemporal dementia-related aggressive behavior

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Behavioral problems in the form of agitation and physical aggression are common sequelae of dementia. These symptoms are worrying to the patient and caregivers, disruptive to the home, and often lead to physical injury. The first intervention for dementia related behavioral agitation and aggression, after the failure of nonpharmacological measures, is judicious use of evidence-based medications including antidepressants, mood stabilizers, and atypical antipsychotics. On the other hand, treatment with atypical antipsychotics has been shown to increase overall risk of mortality. In addition, patients are often can not to tolerate the side effects of these medications. While no controlled trials have been published, case reports indicate that ECT can be successfully used for seriously adverse behaviors in the setting of dementia when other interventions have failed. Here we report a case of successful use of ECT after the failure of both nonpharmacological and pharmacological measures for patients diagnosed with frontotemporal dementia who developed the complications of severe agitation and aggression.
Case: A 65 year-old male patient with no known past psychiatric history who had a 4 year history of frontotemporal dementia. He was transferred to the medical psychiatry unit from neurology department. He exhibited a 1-month history of worsening aggression. His medications on admission included sertraline, pramipexole and acetaminophen. His neurocognitive examination demonstrated a global cognitive impairment. All lab work was within normal limits. Brain CT showed mild to moderate generalized cortical volume loss with secondary ventricular enlargement. Electroconvulsive therapy treatment for his unprovoked aggression was started. He received six treatments with bitemporal lead placement. Seizure durations lasted up to 22 seconds. His aggressive behaviors resolved after the sixth ECT treatment, and he was cooperative and friendly with other family members.
The case demonstrate the effectiveness of ECT in treating medication refractory dementia-related aggression. In a case, the ECT was well tolerated and led to marked improvement in social comportment and standard of living. Discerning the etiology of the behavioral changes in the setting of advanced dementia is not easy because agitation and aggression may be the result of impairment of cognitive functioning, psychosis, anxiety, mania, agitated depression, physical illness or discomfort, or a medication side effect. Although depressive disorders remain the most common indication for ECT in elderly persons, a growing literature identifies ECT as an effective intervention for severe refractory agitation and aggression for patients with dementia. Based on the report, we believe randomized, controlled trials with ECT are necessary to clearly delineate the effectiveness of ECT for these Patients
Keywords: electroconvulsive therapy, frontotemporal dementia, aggressive behavior

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[Abstract:0547][OCD]

Fluoxetine treatment in a prepubertal girl with hoarding disorder

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Hoarding is characterized by not only acquiring objects in great excess, but also being unable or unwilling to part with them, causing great personal and family distress. In an effort toward gaining a better understanding of hoarding, distinctions have recently been made between hoarding within the context of obsessive-compulsive disorder (OCD) and as a part of separate disorder, Hoarding Disorder (HD), which has been added to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5). The estimated prevalence of HD in adults is 2–5%. The estimated prevalence of hoarding in adolescents (2%) is similar to that observed in adults while the prevalence in children is still unknown. Previous research has shown that among the various subtypes of OCD, adults and older children and adolescents with problematic hoarding have distinct features and a poor treatment response. However, adults who are hoarding often report that onset of their symptoms were during childhood. Nevertheless there is a limited information on the phenomenology and prevalence of hoarding behaviors in young children. As the childhood hoarding literature is scant, the prevalence, trajectory and prognosis of hoarding disorder in children are unknown. In this case we aimed to discuss the clinical presentation and fluoxetine treatment of a prepubertal girl with hoarding disorder.

Case: A 7 year-old girl, living with parents, going to elementary school second grade attended to our outpatient clinic with the complaints of difficulty about discarding possession, not throwing garbage such as banana peels, not wanting to go to school and afraid of leaving his mother for 1 month. Her mother has OCD and her uncle was diagnosed with generalized anxiety disorder. In the assessment, the girl got 53 points from Screen for Child Anxiety and Related Disorders (SCARED), 17 points from Children's Depression Inventory (CDI), 28 points from Children's Yale-Brown Obsessive Compulsive Scale (CY-BOCS). We prescribed fluoxetine 5 mg/day and increased the dose to 10mg/day after two weeks. She responded to fluoxetine and she was able to throw garbage and discard possessions.
Although typical patients with OCD experience ego-dystonic symptoms, in hoarding disorder patients usually experience ego-syntonic symptoms and generally have poor insight. Previous research revealed that patients with hoarding disorders may be resistant to psychopharmacological interventions. Improvement in our case with fluoxetine could be associated with early detection and treatment. Perhaps early diagnosis and treatment of hoarding disorder in children may lead to better prognosis than adults with chronic hoarding disorder. Previous research has documented high rates of comorbidity in those with hoarding disorder and our case was also with findings of separation anxiety disorder.
Better understanding of the hoarding disorder in children would facilitate early identification and treatment and may also prevent the chronic burden of hoarding disorder in adulthood which is a generally chronic condition with poor treatment response.
Keywords: hoarding, children, fluoxetine treatment

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[Abstract:0548][Psychopharmacology]

The treatment of Capgras syndrome: a case report

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Capgras syndrome is the delusions of the existence of identical "doubles" of his/her close relatives, the objects and even of the patient himself or herself. It is commonly accompanied by schizophrenia or organic psychosis. At the beginning it was assumed to be a rare syndrome. But nowadays this syndrome is accepted to be seen fairly common. It could be seen in the wide range of ages and both sexes, but it was reported much more in women. In the etiology of this syndrome, psychodynamic and organic factors are considered. Here, a case of Capgras syndrome, treated with aripiprazole and paliperidone palmitate, is presented and discussed.
Case: A 28 year-old female patient was married with three children, elementary schoolgraduated and a housewife. She was taken to our clinic by her husband because of self-talk, the thought of being hurt by her husband, hostile behavior towards her husband and children, faith of her husband, the first child, mother and father may be replaced by similar, cheated on her husband with the other person who is similar and therefore guilt, hearing voices saying that her husband cheated, seeing unrecognized men's image. For the first time patient with no history of psychiatric symptoms thought that her husband was replaced with similar three years ago. It was learned that she had a partial recovery with antipsychotic medication after one and half year on the inclusion of feeling fear, insomnia, audio-visual hallucinations and thinking that her first child, her mother and father were also be replaced with similar and had been presented to various hospital. After approximately 6 months of treatment, complaints emerged again due to not taking drugs because of excessive weight gain. Due to aggravation of symptoms in the last month, she presented to our clinic. Aripiprazole is preferred as psychopharmacological treatment because of considering that dropped out of the treatment before, due to adverse metabolic effects. Paliperidone palmitate was added due to the lack of insight. During treatment of the patient, it was observed that the delusion associated with Capgras syndrome was decreased and partially insight was gained. She was discharged with partial recovery from the point of schizophrenia and Capgras syndrome. Aripiprazole dose was cut by decreasing on outpatient follow-up. She is still well-being with 100 mg/month paliperidone palmitate.
Our case is a female patient who has schizophrenia and Capgras syndrome, it is unlike the cases in the literature that Capgras syndrome occurred a long time before meeting DSM-5 criteria for schizophrenia. In the treatment of Capgras syndrome pharmacotherapy is generally used. In fact prognosis of the patient depends on the success of the treatment of psychosis. In the treatment of our patient, unlike the many of previously reported cases, an atypical antipsychotic agent aripiprazole and prolonged release paliperidone palmitate has been used and the response has been observed. This case suggests that a combined treatment of two antipsychotic agents can lead to a good clinical outcome with a remission of the psychotic symptoms.
Keywords: capgras syndrome, schizophrenia, aripiprazole, paliperidone palmitate

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[Abstract:0551][Autism]

Childhood disintegrative disorder

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Childhood Disintegrative Disorder -also known as Heller syndrome, a clinical syndrome distinct from childhood autism- is a rare pervasive developmental disorder. It is characterized by loss of previously acquired language and social skills, before age of ten, after a period of at least 2 years of normal development. Unlike progressive neuropathological processes, the child loses skills but stabilizes with no further deterioration. In The Diagnostic and Statistical Manual of Mental Disorders-V (DSM-5), the terms 'Autistic Disorder', 'Asperger Disorder', 'Childhood Disintegrative Disorder' and 'Pervasive Developmental Disorder Not Otherwise Specified' have been replaced by the collective term 'Autism Spectrum Disorder'.
Case: A 31 year-old male patient who was referred to our clinic with epileptic seizures and weight loss due to avoidance of solid food. In psychiatric examination, communication could not be established with patient. He had significant deficits in language and social developments as well as several stereotypic behaviors. He had few meaningful words and could not understand simple commands. He was reported to show normal development until 6 years of age followed by developmental regression of language, cognitive ability, interest in the social environment, and self-care ability with emergence of aberrant and stereotypic behaviors, restlessness, echolalia suggestive of childhood disintegrative disorder. His father refused treatment and special education options until he was 19 year-old. The family was referred to a psychiatrist fallowing the father's death when the patient was 19 and olanzapine, risperidone, diazepam treatment was started with the diagnosis of childhood-onset schizophrenia. Several antipsychotics had been tried and he was taking clozapine 450 mg at referral. The absence of present or previous positive symptoms of psychosis, normal developmental milestones until the age of 6, and the following progressive deterioration of verbal and social skills and settlement of autistic symptoms, stabilization with no further deterioration suggest that the clinical diagnosis of Autism Spectrum Disorder according to DSM-5 and Childhood Disintegrative Disorder according to DSM-IV. Neurological consultation was requested and cranial MRI, CT with contrast and EEG were taken to exclude organic pathologies that can cause seizures and to evaluate for epilepsy. MR, CT and EEG revealed no specific findings. EEG demonstrated widespread disorganization. The seizure was presumed to be due to the threshold lowering effect of clozapine. Clozapine was gradually decreased to be discontinued due to the change in diagnosis and presence of seizure and no aggressive behavior or agitation was observed following the discontinuation. Escitalopram was started and increased up to 10 mg targeting anxiety and rigid behavior patterns - refusing to eat solid food-. 15 days after admission to the hospital, it was determined in weight measurement that the patient was observed to gain 6 kgs- from 53 kgs to 59 kgs, with significant improvement in eating and appetite.
This case is reported because it is a very rare condition and it was previously misdiagnosed as childhood-onset schizophrenia.
Keywords: childhood disintegrative disorder, autism, childhood-onset schizophrenia

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[Abstract:0553][Psychopharmacology]

A Case of edema associated with risperidone

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Risperidone, a benzisoxazole derivative, is a second generation antipsychotic that binds to serotonin type 2 and α1-adrenergic receptors with high affinity, and causes antagonism at these receptor sites. Risperidone has been proven to be effective in psychotic disorders, bipolar disorder, aggression and several other psychiatric disorders. Some of the common side effects of risperidone are extrapyramidal disorders, akathisia, weight gain, anxiety and headache. Apart from these side effects, edema has been reported in several case reports.
Case: A 36 year-old female patient who had been treated for schizophrenia for three years was admitted to our inpatient clinic for the second time with complaints of suspiciousness, furious behavior and social avoidance. She was treated in our inpatient unit 2 years ago

with the diagnosis of schizophrenia. After her discharge, she had used paliperidone palmitate 100 mg i.m. inj./month for 1 year and then refused to use treatment. At her second admission, risperidone 4 mg/day treatment was initialized. At the second day of the risperidone treatment, she complained of swelling in her feet and hands. The swelling in her hands and feet increased day to day and also swelling in pretibial and periorbital zone occurred. She did not have a history of edema before. After the laboratory tests and consultations the other medical causes of edema were ruled out and then risperidone treatment was stopped. After the cessation of risperidone treatment, the edema decreased day by day and disappeared spontaneously after 4 days. Aripiprazole 10 mg/day treatment initialized for her psychotic symptoms. Edema related to risperidone has been recognised in the drug manufacturing company's monograph as an infrequent adverse event occurring in between 1/100 to 1/1000 patients. A medline search including all years up to 2001 revealed only six cases of edema associated with risperidone. In our clinical practice with hundreds of patients treated with risperidone, we only observed one case, anjioedema due to risperidone, which was reported in 2007. To explain the edema due to risperidone, various hypotheses have been put forth. Firstly, the edema can be attributed to vasodilatation and decrease in vascular resistance, which is secondary to blockade of α1 receptors by risperidone. What can also lead to vasodilatation is 5HT2 blockade and also dopaminergic blockade can alter the renal regulation of electrolytes, which may play a role in development of edema.
This case is also of importance as it demonstrates an adverse effect that had not occurred with the previous paliperidone treatment which is an active metabolite of risperidone with similar clinical efficiency.
Keywords: adverse effect, edema, risperidone

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[Abstract:0559][OCD]

How functional is functionality criteria: a very late diagnose of an obsessive compulsive case

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Obsessive-compulsive disorder (OCD) has been known as a serious, disabling disease and is one of the 10 leading causes of disability worldwide. Isolation, distress, persistent impairment, particularly in social and work functioning has been defined. It is characterized by early onset, chronic course, and significant comorbidity. Patients often delay or are unwilling to seek treatment. Here, an OCD patient who preserved both professional and family functionality for over 50 years without a diagnosis is presented and intended to be discussed in the context of the functionality.
Case: A 87 year-old male patient, widowed, retired was hospitalized in the orthopaedic service because of the infection in his legs. He was taking bath for up to 7 hours in hospital. His infection was thought to be due to excessive washing. He was refusing the staff recommendations and referred to psychiatry service. He had no complaints and the history was taken from family. His washing behaviors was present over 60 years. He was working as an officer. Since his marriage at 35 year-old he always had nervousness, fear, jealousy, thoughts of hurting those around him. At this time he got psychotherapy for two years but no improvement has been seen, after than the family has accepted these finding as a personality trait and he had no treatment. Because of his washing, cleaning and wearing rituals, he was waking up at 5:00 am to get on work. At work he was known as rigorous and compatible with superiors. He was paying great attention to the environment which his arms can extend from his chair and was ignoring the rest. This area was clean but he was throwing his water battle or napkin outside. After work he was going the same place, by using the same way to meet his friends. His son died at 1975 and his wife died at 2000, after these losses he his symptoms was increased, he refused cleaning of his house and did not allow his girls to get out from home for some years. On examination excessive dryness of the skin and irritation of the hands and feet was found. He was refusing to use eyeglasses and hearing aids. Overt obsessive thought and compulsive behaviors was observed in the follow up period. Yale Brown score was 34.
OCD was present for 50 years but the patient worked as an officer and retired normally, his family relationship was relatively normal despite some limitations. This are contrary findings with studies that reported significantly poor family functioning, social support and professional underachievement. When the social environment provides an acceptable or suitable atmosphere for the patient, some symptoms may be more tolerable and the diagnose and treatment may be delayed It has been stated that married patients had higher social functioning scores like in our patient but another factor in our patient was the patients' profession. In a profession that wants diligence, orderliness, obedience like being a soldier, OCD symptoms can paradoxically prevent the patient from the distorsion of professional compatibility.
Keywords: obsessive-compulsive disorder, functionality, diagnosis

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[Abstract:0560][Schizophrenia and other psychotic disorders]

Schizophrenia and psychogenic polydipsia

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At least 3 liters per day water consumption is considered as psychogenic polydipsia clinically. It is thought that psychogenic polydipsia has a prevalence of 6-17%. It is important to diagnose polydipsia because it causes rhabdomyolysis, coma and death. In the present case report, we introduced a schizophrenia patient with a daily 10-15 liters water consumption.
Case: A 50 year-old single male patient. He has never worked. In his last psychiatric examination, according to him he had no complaints except forgetfulness but his family complained about his excessive water consumption and fatigue. The patient had schizophrenia since he was 21 year-old. He was hospitalized several times. He was on treatment regularly for 2.5 years at our psychosis outpatient unit. The patient used various antipsychotics such as haloperidol, quetiapine, thioridazine during his previous treatment. In his past history, he presented to an Emergency Department with nausea and vomiting about three years ago. Hyponatremia was detected in his laboratory tests for the first time. He was hospitalized in Intensive Care Unit. He was discharged after his electrolyte balance had been achieved. Following his discharge, he stopped psychiatric treatment and then he was hospitalized in psychiatric inpatient unit. His hyponatremia was still persisted. The nephrology evaluation was normal and fluid restriction was suggested for hyponatremia. His psychiatric treatment was changed to clozapine, valproate and risperidone. During his follow-ups in psychosis outpatient unit, he presented to Emergency Department for 3 times with a diagnosis of delirium due to hyponatremia. He still continued to consumpt 10-15 liters/day water. During his follow-ups he had no positive psychotic symptoms, marked negative symptoms. He explained excessive water consumption because of his dry mouth and could not succeed to stop excessive water consumption. Clozapine was increased up to 350 mg/day. It helped to stop patient's excessive water consumption but, his sodium levels are monitored regularly in order to prevent him medical complications due to hyponatremia.
Psychogenic polydipsia occurrence is around 6-20% of patients with schizophrenia. Although polydipsia is a common symptom, its etiology is still unknown. The medial temporal lobe controls water intake and Anti-Diuretic Hormone (ADH) secretion. High levels of ADH is known to be the cause of psychotic relapses in schizophrenia. In the literature it is reported that increased dopaminergic activity due hypersensitivity of dopaminergic receptors causes polydipsia. It is also reported that typical antipsychotics cause polydipsia. The onset of polydipsia symptom is thought to be due to a mechanism as in in tardive dyskinesia. For that reason especially the atypical antipsychotics which have low D2 receptor antagonizm such as clozapine and quetiapine are used for the treatment of polydipsia. Effectiveness of risperidone and other atypical antipsychotic drugs is still controversial. In the present case report we want to notice the symptom of polydipsia which can lead to mortality in schizophrenia patients. Polydipsia is usually hard to treat and treatment options are still controversial. The clinicians should be aware of this symptom and regularly monitorize their patients' sodium levels in order to avoid mortal complications such as delirium and cardiac arrhythmia.
Keywords: schizophrenia, psychogenic polydipsia, treatment

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[Abstract:0568][OCD]

Self-amputation of tongue in a patient with obsessive compulsive disorder: a case report

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The tongue has important roles in speech, deglutition, taste, airway protection, and its amputation is very serious.
Case: A32 year-old female patient who presented with cutting her tongue and was brought to the emergency service by her relatives. Her tongue was replanted by otolaryngology and head and neck surgery. The result was that the state of the replanted her tongue was relatively good both functionally and aesthetically. After, the vital diagnosis of the patient was evaluated as normal. Physical examination,

laboratory tests and cranial MRI showed no pathology. There was no history of trauma (accidental), mental retardation, or neurological problems. Therefore, she was consulted to our psychiatry clinic after medical intervention. In the psychiatric examination; her affect was distressing, mood was anxious and reality testing was normal, there were no hallucinations or delusions. She had religious obsessions involving blessing rituals. Her complaints were having obsessive thoughts and compulsive behaviors for one year. She had no history of regular treatment use. She was hospitalized to our inpatient service for detailed examination. The patient demonstrated excessive worry about her condition and gave a detailed description of her religious obsessions. She also reported that she could not stop thinking about the possibility of a religious blasphemy and described that she was continuously worried and alert. She described these thoughts as being her own and recognised them to be irrational, but she could not resist them. The patient was diagnosed obsessive compulsive disorder (OCD) and Yale-Brown Obsessive Compulsive Scale (YBOCS) score was assessed as 40. In her personal history, there was no medical disease, no alcohol and drug use. She was diagnosed with OCD according to the framework of DSM-5, and fluvoxamine 50 mg/day, alprazolam 1 mg/day, risperidone 1 mg/day were started. At an initial dose of fluvoxamine 50 mg/day gradually and gradually increased to 200 mg/day over the next five weeks. Alprazolam was stopped gradually over three weeks. Her OCD symptoms were significantly decreased in terms of YBOCS at the end of the 7th month. YBOCS score was 14. She was externalized. Within 10 weeks of the start of treatment, her OCD had improved dramatically. She was followed up for six visits in the next one year without any reports of obsessions or compulsions. Our case is also unique because to the best of our knowledge, this is the first report who amputated tongue after having religious obsessions. This case report draws attention to the dramatic consequences of the OCD.

Keywords: obsessive-compulsive disorder, religious obsessions, tongue self-amputation

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[Abstract:0570][Substance-related and addictive disorders]

Pychotic disorder after using synthetic cannabinoid, persistent visual hallucination: case report

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In recent years abusing of synthetic cannabinoids have been increased in Turkey as in the world. Synthetic cannabinoids (SC) are discovered in 1960s. At first, SC's were advertised and sold as herbal products. In Turkey they are best known by the name of 'bonzai'. SC's are functionally similar to cannabis. Nevertheless, there are some similarities between these substances, but SC's bind to cannabinoid receptor-1 (CB-1) with full agonistic and high potency. There is no sufficient information about chronic abusing SC's. The aim of this paper is to share one male patient, who had persistent psychotic symptoms after using SC.

Case: A 24 year-old male patient. He presented to psychiatric outpatient clinic. He has used SC for 2 years. During this time, he tried to break his addiction for several times. He did not have any psychiatric complaint since the beginning of his usage. About one year ago, he saw his friend who was doing strange behaviors under the influence of SC. But, he thought that his friend was under the influence of gins. Three months later from this, after the usage of SC he had psychotic symptoms with visual and auditory hallucinations. He thought that gins wanted to disturb him. Than, he saw smoke, arising from his body. The smoke did not dissappeare and in addition to this, he started to hear unclear voices. These hallucinations increased when he used SC. He has seen the smoke more frequently in last 3 months. In this time, he has rarely heard auditory hallucinations, like rat-tat. Other psychotic symptoms ended when he presented to the clinic. He was not anxious or distress during the evaluation. He said that 'I am used to see the smoke, so I do not afraid of this'. His thought process was normal. He said that he would break his addiction. He has used SC for four times in last two months. He has canabis usage history for two years, before he started to use SC. He does not have any psychotic disorder history about his family.

There are some case reports about psychotic symptoms after using SC. Referred to a few of them, these psychotic symptoms are persistent. According to Spaderna et al. (2013) cannabis includes cannabinidole. Cannabinidole decreases psychotic disorders. There is no cannabinidole in SC, so psychotic disorders may increase with using SC. According to Peglow et al. 2012, there is a case about a patient who used cannabis for many years and has no psychotic symptoms. But, after he started to use SC, psychotic period was triggered. Here, we could not assessed the effect of medication because the patient did not want to use medication. SC's, which may cause various psychotic disorders, are becoming a serious problem nowadays. Clinicians must be careful about atypical and persistent psychotic symptoms that may associate with SC.

Keywords: psychosis, synthetic cannabinoid, bonzai, hallucination

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[Abstract:0574][Mood disorders]

Valproic acid-induced hair structure changes: a case report

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Valproic acid is a molecule for the treatment of both widely used in the treatment of bipolar disorder and epilepsy. Well-known side effects, especially nausea, vomiting, abdominal pain, gastrointestinal symptoms such as diarrhea, polycystic ovary syndrome, hirsutism, liver dysfunction, weight gain, alopecia counted. It has also been reported on hair growth, including hair thinning and hair colour changes that have various effects. We presented a 35 year-old female patient with a mood disorder whom changes in the structure of hair, depending on the use of valproic acid treatment.

Case: A 35 year-old female patient who has recurrent complaints such as reluctance, restlessness, loss of appetite, irritability for 10 years. Mood disorder was diagnosed and began 1000 mg/day dose valproic acid treatment. Within the first one month of starting treatment, she noticed that hair loss significantly. After 3 months later, her hair began to grow again, but she realized that significant changes in the structure of hair. While her hair was thin and shiny after treatment turned into thick and matt structures. While she had oily hair, after treatment has dry hair. Furthermore while she had straight hair after treatment has wavy hair. In addition, she said that she had no any perming or dyeing of the hair performed, before the treatment initiation. Last viewed valproic acid blood levels of 62.4 µg/ml. She did not receive any other treatment in this process. She has been using the treatment for 3 years, because it has been beneficial. She was pleased with the treatment and she did not think about termination.

Valproic acid associated with that curly hair has been little study because it is a rare side effect. According to the literature, the valproic acid to alter the mechanism by which hair texture is unclear, although some mechanisms are mentioned. One of these is related to the chelating property of valproic acid. Copper is necessary for hair growth and keratinization, metals such as zinc and magnesium, is lower in patients treated with valproate. But studies and case reports on this subject are inadequate. In publications mentioned that, if the termination of treatment, this side effect will be reversible. In our case, due to the continuation of valproate therapy could not be made an assessment as to whether this effect is reversible.

Clinicians should be aware of valproic acid's effect on changes to hair texture. They should inform their patients about the possible effects on their physical appearance.

Keywords: hair, mood disorder, valproic acid

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[Abstract:0577][Mood disorders]

TMS may be rapidly reduce suicidalthinking

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Transcranial magnetic stimulation (TMS) is one of novel techniques of the physical treatment in psychiatry. TMS for the treatment of depression was approved by the U.S. Food and Drug Administration (FDA) in 2008. Repeated daily left prefrontal TMS is a non-invasive approach to treating depression, differing from medicinal treatments and electroconvulsive therapy (ECT). Although ECT is more effective than repetitive TMS, patients may prefer repetitive TMS because it is better tolerated and unlike ECT, TMS does not need general anesthesia and induction of seizures. For acute treatment of unipolar major depression, repetitive TMS is commonly administered by stimulating the left dorsal lateral prefrontal cortex daily over four to six weeks. Recent studies suggest that stimulating the prefrontal cortex non-invasively with TMS might quickly reduce suicidal thinking. Here we report a case of use of TMS for patient who diagnosis of depression with a suicidal thinking.

Case: A 25 year-old female patient, presented to the outpatient psychiatry clinic for treatment of a 2-months history of depression. She presented with complaints of depressed mood, loss of energy, insomnia, poor concentration and suicidal thinking. She had been using 150 mg/day of venlafaxine for two month. Her routine hematological and biochemical parameters were within normal limits. Venlafaxine

dose of 225 mg/day was increased and treatment with lithium 600 mg/day, quetiapine 100 mg/day was added. One week later, her symptoms were not reduced. She was offered but refused ECT and hospitalization. Therefore TMS was planned for depression of this patient. It was administered by stimulating the left dorsal lateral prefrontal cortex (20 Hertz, 1000 stimuli/day; Monday through Friday). For five-days follow-up, reduced symptoms of depression, especially suicidal thinking. She answered two self-report questionnaires, the Beck Depression Inventory(BDI) and the Beck Anxiety Inventory (BAI). Before rTMS treatment, the BDI score was 45, and the BAI score was 43. five days after rTMS treatment, the BDI score was 13 and the BAI score was 10.
The case demonstrate the effectiveness of repetitive TMS depression. TMS may be a new method to rapidly reduce suicidal thinking and prevent suicides.
Keywords: transcranial magnetic stimulation, suicidal thinking, depression

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[Abstract:0581][Substance-related and addictive disorders]

Buprenorphine / naloxone therapy in the opioid-dependent elderly: case reports

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The buprenorphine/naloxone combination is used to treat the chronic relapsing disorder of opioid dependence. While numerous studies support the efficacy of buprenorphine /naloxone for the stabilization and maintenance of opioid dependence, there is no any publication regarding the usage in opioid-dependent elderly. In our cases, probable benefits and side-effects of buprenorfine/naloxone treatment in the elderly who are above age of 65 will be discussed.
Case 1: A 67 year-old male patient, graduated from elementary school, retired worker, never married. He presented to outpatient clinic of AMATEM (Alcohol and Substance Abuse Research and Treatment Center) in Erenkoy Research and Training Hospital for Psychiatry due to opioid dependence which is in a nasal manner, 1-2 gr/d for 18 months. In his medical history, after cannabinoid usage in the age of 27, he started to use opioid intravenously, he was hospitalized 12 years ago in a substance abuse treatment center, after the hospitalization he was in remission for opioid usage. 18 months ago, after a stressor in his life, the patient who is portor of chronic hepatitis C, has bening prostat hypertrophy, right bundle block started to reuse heroine. His buprenorphine/naloxone treatment which was started 1 year ago was stopped due to ongoing substance usage. 7 months ago, he presented to our outpatient clinic, he was hospitalized and he is on remission with the treatment of 12 mg/day buprenorphine / naloxone for 6 months.
Case 2: A 69 year-old male patient, graduated from elementary school, retired, married, has 5 children, lives with his family. Two years ago, he presented to our outpatient clinic of AMATEM due to opioid dependence which is in a nasal manner, 1-2 gr/d for 20 years. In his medical history, it was learnt that he had not any other application for treatment and remission period. He has diabetes mellitus, hypertension and history of by-pass intervention. He was hospitalized and he is on remission with the treatment of 8 mg/day buprenorphine/naloxone for 2 years.
Case 3: A 65 year-old male patient, retired, married and has 1 child. In his past medical history, he presented to different substance usage treatment center and had several hospitalization for opioid dependence which is in a nasal manner, 2.5 gr/d for 35 years. After his last hospitalization, he was on remission with the treatment of buprenorphine/naloxone for 4 years.
In all of the three cases, firstly purification via symptomatic treatment with analgesics, antiemetics, antidiarrheal and anxiolytics was tried. When they could not comply with these treatment, they acknowledged after detailed information and their treatment with buprenorphine/naloxone was started. In their outpatient clinic follow-up in the order of 6 months, 2 years and 4 years, it was seen that they were in remission. In every 3 month-interval routine medical scanning, any pathology was not seen.
To establish specific safety guidelines, the use of buprenorphine in the elderly with other drugs requires further investigation.
Keywords: opioid dependence, buprenorphine/naloxone combination, elderly

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[Abstract:0582][Sleep disorders]

Sleep disordered breathing and hypertension: a case report

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Obstructive sleep apnea (OSA) is a common form of sleep disordered breathing and has a relatively high prevalence in the general population. The frequency and severity of OSA is associated with age, male sex, and obesity, and OSA has been linked to cardiovascular complications and death. Importantly, OSA has a strong association with both prevalent and incidental hypertension and has a particularly high prevalence in patients with resistant hypertension. In these patients, CPAP and other OSA-directed treatments have been proposed as therapy to help control blood pressure (BP), especially in patients who have not attained optimal BP control despite maximum pharmacological therapy.

Case: A 30 year-old female patient, married, has a child, graduated from high school, housewife, 162 cm in length, and 80 kg in weight. One year ago, after abrupt severe noise bleeding, her blood pressure was measured as 220/180 mmHg and according to her blood pressure follow-up by internal medicine and cardiology clinics, she was diagnosed with hypertension. Any alcohol, substance or cigarette usage was not stated. Any family history was not expressed and all needed examination is done and any pathology was not detected. When her blood pressure was not be able to regulated with one antihypertensive drug treatment, two antihypertensive drug treatment was started. In her visit by her cardiologist in May, 2015, OSAS symptoms were detected as snoring, sleep apnea, feeling exhausted when waked-up which existed for a long time. He was consulted to Erenkoy Research and Training Hospital for Psychiatry, Sleep Service. In her polysomnography, sleep apnea index was recorded as 12 in supine position. He was diagnosed with OSAS with accompanying hypertension. His CPAP titration was 6 cm/H2O. Any pathology was not detected by recommended otorhinolaryngology policlinic outpatient follow-up.

OSAS is very frequently present during resistant hypertension. OSAS-related hypertension has several characteristics: it is highly prevalent, predominantly diastolic and nocturnal, and frequently affects non-dippers. There are many mechanisms linking OSAS to HT. The main stimulus is intermittent hypoxia and the most important pathophysiological consequence is high sympathetic activity. After doing the diagnosis of OSAS, its treatment principally associated lifestyle changes with continuous positive airway pressure (CPAP). CPAP can significantly decrease blood pressure, especially if compliance to treatment is good and hypertension is resistant.

Keywords: sleep disordered breathing, hypertension, treatment

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[Abstract:0584][Sleep disorders]

Association of psychiatric disorders and sleep apnea: a case report

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Obstructive sleep apnea syndrome (OSAS) is a common sleep disorder defined by frequent episodes of obstructed breathing during sleep, which is characterized by sleep-related decreases (hypopneas) or pauses (apneas) in respiration. The diagnosis of OSAS is confirmed when a polysomnography recording determines an Apnea-hypopnea-index (AHI) of >5 per hour of sleep. Continuous positive airway pressure (CPAP) which device maintains pharyngeal patency is the approved choice of treatment for OSAS.

The association between OSAS and medical disorders such as cardiovascular disease, genitourinary, metabolic disorders and psychiatric conditions, including depression and anxiety has been found in several studies. The mechanism underlying such a relationship are not revealed accurately. The affective disturbance may be a biologic and/or psychosocial consequence of sleep apnea. Inversely, psychiatric disorders may attribute to development of sleep disturbances. Hypoxemia and sleep fragmentation may provoke depressive symptoms, vice versa. Some researchers believe that the psychological impairments can be reversed after appropriate treatment, while some stated that they persist after treatment related to irreversible anoxic central nervous system damage probably.

Case: A 58 year-old male patient, married, has two children, retired. Complaint: Anhedonia, unhappiness, fragmented night sleep and

waking with the feeling of fear, somatic symptoms, irritability, anxiety, thought of an unexpected event will come true, restlessness, passive suicidal thoughts. The patient has a psychiatric history for three years and followed by various outpatient mental health for two years. He did not remitted with the various combination of escitalopram, sertraline, duloxetine, trazodone, quetiapine, mirtazapine and alprazolam despite appropriate dosage and timing of drug usage. He was hospitalized. His routine laboratory examination and cranial imaging were in normal range. His blood pressure and glucose level were in the normal range with the treatment of hypertension and diabetes mellitus. Any alcohol, substance or cigarette usage and any psychiatric family history are not stated. He was psychomotor agitated, depressed in mood, anxious in affect; light bilateral hearing loss was detected. He was diagnosed with both Major Depression and Generalized Anxiety Disorder according to DSM-5. His Hamilton depression/ anxiety scale score were 49/35. After admission to hospital, his treatment regulated, duloxetine was stopped and sertraline was increased upto 100 mg/day. Mirtazapine was changed with trazodone due to ongoing sleeplessness. He was observed as snoring and having sleep apnea by nurses and was directed to sleep laboratory. In his polysomnography, sleep apne index was detected 55 per hour. CPAP was determined as 9 cm/H_2O with constant pressure. He was discharged with treatment of sertraline 100 mg/day. During follow-up, his depressive symptoms regressed and sertraline dose declined to 50 mg/day.

Patients with OSAS have a high rate of psychiatric comorbidity, especially related to anxiety disorders and depression. In this case, we aim to present a case with treatment resistant depression-anxiety and comorbid OSAS and its treatment with CPAP. According to our observation; particularly in patients with treatment resistant psychiatric disorder clinicians should investigate sleep disorders.

Keywords: obstructive sleep apnea syndrome, psychiatric disorders, comorbidity

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[Abstract:0586][Sleep disorders]

Obstructive sleep apnea in children: a case report

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Obstructive sleep-disordered breathing is common in children. 1 percent to 10 percent of children is affected by obstructive sleep apnea syndrome. Most of these children have mild symptoms, therefore this disease may be ignored. The gold standard evaluation is overnight polysomnography (PSG). Treatment includes the continuous positive airway pressure (CPAP) usage which is tolerated poorly in children. Therefore, adenotonsillectomy is considered primary therapy and curative in most patients.

Case: A 10 year-old boy presents with snoring and excessive daytime somnolence. Upon further exploration significant history of obstructive seep apnea (OSA) symptoms such as witnessed pauses during sleep, poor academic performance, hyperactivity. He has appropriate weight/ height/ BMI. Otolaryngologist plans adenotonsillectomy for patient. But significant OSA symptoms continues after operation. His mother reports snoring, tired everyday after school, behavior issues reported in school like hyperactivity and poor academic performance. PSG evaluate the residual sleep disorder breathing. Patients' postoperative sleep study demonstrate severe OSA: REM 22.0%, AHI: 78.56/hr, REM AHI: 35.79/hr, NREM AHI: 90.59/hr. Then positive airway pressure is detected by second PSG as automatic n-CPAP at 6-11 cmH_2O. Symptoms of OSAS at the beginning such as poor academic performance, behavior disoders are disappeared during folow-up. Follow up sleep study is essentially normal: REM: 9.5%, AHI: 8.38/hr, REM AHI: 0, NREM AHI: 9.26/hr.

Positive airway pressure therapy (PAP) is used to treat children who have obstructive sleep apnea syndrome and do not respond to adenotonsillectomy. It is safe, effective and well tolerated by children and adolescents with OSA and positively impacts on a child's overall state of health, quality of life and wellbeing. Therefore, the widespread use of PAP should be discussed.

Keywords: obstructive sleep apnea syndrome, children, treatment

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[Abstract:0588][Psychopharmacology]

Orbital edema associated with quetiapine: a case report

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Quetiapine is a second-generation antipsychotic and is commonly prescribed for a broad range of psychiatric conditions with frequent off-label use. Quetiapine's mechanism of action is not fully elucidated though involves antagonism at serotonin type 1 (5- hydroxytryptamine [5-HT1A]) and type 2 (5-HT2A, 5-HT2C) receptors with relatively weak antagonism at dopamine (D1, D2) receptors. In addition, quetiapine exhibits some α1- adrenergic antagonism that may explain its cardiovascular side effects, like orthostatic hypotension. Bilateral orbital edema has been infrequently described with several atypical antipsychotics, including case reports with olanzapine, risperidone, and ziprasidone. Currently, edema is not currently listed as a potential complication in the its prescribing information. Here in, we report a case of bilateral orbital edema associated with quetiapine. The patient had resolution of symptoms with medication discontinuation with additional symptomatic treatment.

Case: A 50 year-old male patient, divorced, has 4 children, graduated from high school and retired. He was hospitalized to department of AMATEM (Alcohol and Substance Abuse Research and Treatment Center) in Erenkoy Research and Training Hospital for Psychiatry on 30th of May, 2015. His alcohol abuse was fifteen cans of beer daily for fifteen years. Any abnormality was not detected in his routine laboratory examination except slight elevation in ALT, AST and GGT level. His treatment started with 2000 ml of balanced solutions with b vitamin complex/day for three days, diazepam 60 mg/day, and mirtazapin 15 mg/day. His diazepam dosage decreased daily. Due to sleeplessness complaint on 5th of June, quetiapine 50 mg was given. Approximately 7 hour later after drug given, he had bilateral orbital redness and edema which prevented his opening of eyelids. He was consulted to eye clinic and diagnosed as bilateral orbital allergic edema related with quetiapine treatment. Oral antihistaminic treatment was started and his complaints regressed in three days. He was discharged with treatment of acamprosate 6 tablets /d, mirtazapine 30 mg/day.

Relationship between initiation of the medication and the appearance of edema supports the impression of an adverse reaction to quetiapine. The mechanism of quetiapine-induced edema remains uncertain though likely parallels that of other second-generation antipsychotics. An allergic reaction could pose an alternative explanation for drug-induced edema. Further clinical observation and research is needed to clarify the characteristics, risk factors, dose dependence, and potential mechanisms of quetiapine-associated edema. We hope our report will alert physicians to this potential vascular complication to promote prompt recognition and intervention.

Keywords: quetiapine, side effect, orbital edema

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[Abstract:0589][Autism]

A case report: autistic symptoms of a child with single-suture craniosynostosis

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There is growing evidence that single-suture craniosynostosis (SCC) is associated with neurobehavioral problems. We reported a 5 year-old boy with single-suture craniosynostosis impairment of social skills and persistent interests.

Case: A 5 year-old boy with single-suture craniosynostosis showing social interaction problems, persistent interests and ritualistic behaviors presented to our clinic. He gained developmental motor and cognitive milestones earlier than his peers, but his speech tended to focus around his interests and ritualistic in nature. He was interested in, and quite knowledgeable about, electronic goods. He had ritualistic behaviors such as interesting in electronic marks, watching repairing videos on the internet. He played mainly repairing old vacuum cleaners. He frequently displays inappropriate social responses, like over-familiar with strangers. He showed difficulties with temper control and frustration tolerance. In psychometric test, Stanford Binett. Total score is 116. During this assessment he displayed significant social impairments that fell within the autistic spectrum. However, attachment disorders are not excluded. In previous history, he was born at 34 weeks of gestation, during his first two week of life he stayed in the hospital. He was diagnosed as 'non-syndromic

single-suture craniosynostosis' after pediatric assessment and he was made surgical intervention to release suture fusion at the age of 5 months. His parents reported that their son had poor eye contact since this intervention. The pathogenesis of autism is unclear, although mutations in genes implicated in synaptogenesis have been identified and different neurochemical, neurophysiological, and neuropathological abnormalities have been demonstrated in these patients. Craniosynostosis refers to the premature fusion of one or more of the sutures that normally separate the bony plates of the infant's skull. SSC refers to an isolated premature fusion of one cranial suture. Physicians have anecdotally reported that the majority of children with simple craniosynostosis are within the normal range according to global developmental or IQ scores. However, the specific neuropsychological deficits identified in 35-40% of children with SSC including problems with learning disability, language impairment, or behavioral or cognitive abnormality may be related to the anatomic differences that persist after correction of suture fusion. There was a little evidence that surgery either prevented or reduced risk of neurobehavioral impairment. Some cases have been reported about craniosynostosis is associated autistic behavior; a child with Apert's syndrome, two siblings with craniosynostosis, a child with Saethre-Chotzen syndrome. On the other hand, parent's responsiveness and attachment to infant can potentially affected by some factors such as potentially life threatening surgeries and infant's unusual appearance.

The case described highlights the need to consider the presence of autism in sufferers of craniosynostosis. Most individuals with high functioning autism are not diagnosed while young children. The average age at diagnosis is 7.2 years. Although the causal relationship between SCC and autism uncertain, SCC is at very least a visible and easily diagnosed. This creates opportunities for early detection of neurobehavioral difficulties and preventative interventions.

Keywords: autism, single-suture craniosynostosis, neuropsychology

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[Abstract:0591][Others]

Psychogenic dysphonia: a case report

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Psychogenic dysphonia is a functional voice disorder, as there are no structural laryngeal lesions or neurological alterations directly related to the evident vocal symptoms. Psycho-emotional and psychosocial disorders are commonly identified, such as anxiety, distress, depression, conversion reaction, personality disorders, and interpersonal conflicts in the environment. The respiratory control, vocal intensity, vocal range, vocal resonance, fundamental frequency, articulation, and velocity of speech may be impaired. Usually, more than one vocal parameter is altered, either permanently or not. The onset of vocal symptoms related to psychogenic dysphonia is usually sudden and can be described by the patient. The intermittent nature of psychogenic dysphonia is the most prevalent form of evolution, in which periods of normal voice alternate with periods of aphonia or dysphonia. These fluctuations in vocal emissions are generally observed in the beginning of the medical consultation, leading the physician to the diagnosis.

Case: A 24 year-old female patient, married, has a children, housewife, graduated from elementary school, is living with her mother- in-law. His husband works in abroad. Ten months ago, she presented to emergency service with complaints of fever, fatigue, weakness, the patient was scared of intravenous injection administration and had a conversion reaction. After intravenous diazepam treatment, the patient was started to talk in a hoarse voice and her voice was hardly heard by people near to her. The patient and her family presented to internal medicine and otorhinolaryngology due to ongoing hoarseness although other flu-like symptoms resolved after one week. Any pathology was not detected, she was consulted to psychiatry. She presented with complaints of anhedonia, unhappiness,conversion reaction after stressor, feeling of fear, hoarseness for one month. Escitalopram 10 mg/day and diazepam 10 mg/day treatment were started for depression. Her complaints did not regress after 20 day of treatment and the patient stopped her treatment and presented to other psychiatry clinic, she was hospitalized and administered two sessions of Galvenic-Faradic current treatment. His hoarseness regressed. She was dicharged after one week with the treatment of clomipramine 75 mg/day and risperidone 1 mg/day for the diagnosis of dissociative disorder. After approximately forty days of discharge, patient's conversion reactions and hoarseness restarted with ongoing marital stressor. She presented to our hospital with these complaints, sertraline 50 mg/day and medazepam BID were started. After one month of regular drug usage, her complaint did not regress. It was learnt that patient had problems with her mother- in- law for long years and depressive symptoms for two years exacerbated occasionally and did not presented to psychiatry before. Due to her husband was working abroad, she could not live in a separate house.

The most frequent clinical presentation of psychogenic dysphonia is conversion aphonia, followed by musculoskeletal tension

and intermittent voicing. Considering the diversity in clinical and vocal presentation of patients with psychogenic dysphonia, a multidisciplinary approach (otorhinolaryngologic and psychologic, combined with speech therapy) is crucial to achieve a good outcome in these patients.

Keywords: conversion reaction, psychogenic dysphonia, treatment

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[Abstract:0592][Autism]

Autism spectrum disorder following herpes encephalitis: a case report

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Autism spectrum disorder (ASD) is described as a neurodevelopmental disorder which has some features like social deficits and communication difficulties, stereotyped or repetitive behaviors, narrow range of interests and limited functional skills. There is not any exclusive explanation of underlying mechanism of autism, etiology of ASD still remains unclear. Here, we present a case that shows autism symptoms, started after herpes encephalitis at nine-months-old. To best knowledge, it is firstly reported a post herpes encephalitis case with autistic symptoms in Turkey.

Case: A 3 year-old male patient, presented to our clinic with his mother for the unresponsiveness to his name when called and not speaking. In his personal history, he had had a period with fever, fatigue when nine-months-old. After 3 days with those symptoms, he had been brought to the hospital with a critical state showing disturbed consciousness. Because of suspected meningitis/encephalitis, he had been hospitalized. MRI had showed extensive cortical diffusion restriction and pathological signal changes in cortical-subcortical white matter of bilateral insular cortex, left antero-medial temporal lobe, left parahippocampal area and bilateral frontobasal area. With PCR-DNA method, HSV-1 had been confirmed. On day 16, an epileptic seizure resisted to treatment had led the patient to pediatric ICU with a diagnosis of status epilepticus. At the end of 2 months, patient had been discharged from hospital. Y had continued to treatment as outpatient. His mother said that until the infection, the development of patient was appropriate such as been looking when his name called, using two or three words, playing peekaboo game, socially smiling. He had loved to be cuddled. After the infection, he had changed in a bad way. He had stopped to play with his toys, to use meaningful words, to lose the interest towards his peers, to mimic the others. The mother told that all those changes have occurred during hospitalization. During examination, we observed that he had no eye contact, joint attention, social contact. He did not request objects and was not interested in showing objects to others. He was walking in the room, bringing toys to his mouth. He did not respond to his name. He showed some stereotypical activities like jumping or spinning in circles. He was crying out meaningless words. Taking no command, he did not react to separation from his mother. Autism Behavior Checklist's score was 92. In his family psychiatric history, the mother talked about his 6 years-old sister, diagnosed with ASD.

Linguistic deficits, verbal and non-verbal communication problems and stereotypic activities in this case led us to ASD diagnosis. Having no symptom of ASD and using 2-3 words before the herpes encephalitis made the diagnosis herpes encephalitis related autism. In regressive autism, 1/3 of all autism cases, environmental factors like infections can have an impact for disorder. The case we presented demonstrates the possibility of ASD's coming out because of herpes encephalitis in children who have genetic or any other predisposition.

Keywords: autism, autism behavior checklist, herper encephalitis

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[Abstract:0593][Mood disorders]

Lithium intoxication with early electrocardiogram changes: a case report

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Lithium salts are generally used to treat psychiatric conditions such as depressive and bipolar disorders. The action mechanism of lithium is not completely known. In general thought, it effects on biological membranes via reducing intracellular cAMP. The therapeutic range of lithium is very narrow and its toxicity or overdose can cause dysfunction of organs. Overdose of lithium may affect the heart and can lead to various ECG changes or cardiac arrhythmias. Cardiac effects of lithium is dependent on the displacement of intracellular potassium and lithium ion. The most common ECG changes that caused by the lithium are T wave changes (flattening or inversion) and sinus bradycardia. These changes are benign and disappear after lithium excreted from the body. We report a case with bipolar disorder who admitted to clinic with severe depressive symptoms and presented with ECG changes due to lithium overdose in early phase.

Case: A 65 year-old female patient, divorced, living with her family, had diagnosed bipolar disorder for 30 years. She has been consulted to the mental health clinic approximately seven times per a year for depressive symptoms. The last hospitalization of her was 5 months ago and she was discharged with the lithium treatment 900 mg/day, quetiapine XR 400 mg/day, quetiapine 200 mg/day, lamotrigine 175 mg/day. One month later she had become introverted, had not wanted to talk to anyone, had experienced a loss of appetite. In the last follow-up as outpatient, the dose of lithuril was increased to 1200 mg/day, the dose of lamotrigine was increased to 200 mg/day due to depresive symptoms and quetiapine was stopped. She has not eaten anything for the last two days. She was admitted our clinic with the complaints of autism, reduction of talking, the excessive increase in sleep and motor weakness particularly of lower extremities. In our clinical follow-ups, her pulse was low (about 55 beats/minute) and the patient's ECG was remarkable for short p-r interval (88ms), sinus bradycardia (vent. Rate 50bpm), T-wave inversion in leads V2, V3 and V4, prolonged QT (496ms). Due to the ECG results, she was consulted with cardiology. Acute cardiac pathology was not determined. Cardiologist suggested close monitoring with ECG in every 2 hours. After three hours, the level of lithium was 3.69 mEq/l that indicated lithium intoxication. Immediately, she was referred to the emergency department and admitted for hemodynamic support and urgent hemodialysis. After hemodialysis, the patient's lithium level decreased to 0.53 mEq/l and her general condition improved rapidly. Her follow-up ECG revealed any abnormalities except slight prolongation of the QT interval.

Lithium overdose causes toxicity through several mechanisms including competition with sodium, potassium, calcium and magnesium ions, each of which plays an important role in cellular membrane physiology and these mechanisms can lead to several ECG changes. The most frequent changes in the ECG during lithium overdose or intoxication were a biphasic or inverted T wave, sinus bradycardia and prolonged QT-interval. As in our case, early ECG changes may be a warning for the lithium intoxication ans clinicians should be very careful in this regard.

Keywords: bipolar disorder, ECG changes, lithium intoxication

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[Abstract:0594][Psychopharmacology]

Aripiprazole-induced slurred speech: case reports

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Aripiprazole is the first antipsychotic drug of a new group with a peculiar pharmacodynamic profile. The partial agonistic effect of aripiprazole on 5-HT1A receptors may be associated with improvements in anxiety, depression, negative psychotic symptoms and a reduction in extrapyramidal symptoms. Side effects like headache, insomnia, and extrapyramidal side effects, such as tremor and akathisia may be treatment limiting in some cases.

Case 1: A 43 year-old female patient was hospitalized with depressive symptoms unresponsive to fluoxetine 20 mg/day and alprazolam 1 mg/day treatment. Fluoxetine dose was gradually raised to 60 mg/day, alprazolam was continued. Aripiprazole 5 mg/day was added

to treatment and was gradually increased to 15 mg/day in the 4th week of hospitalization. In the following three weeks suicidal ideation and other depressive symptoms was regressed. Alprazolam treatment was discontinued and she was discharged. 10 days after discharge, she was referred to outpatient clinic with slurred speech, impaired pronunciation and paresthesia around mouth and lips. There was no pathological signs in neurologic examination and brain imaging. These symptoms was thought to be an akathisia-like extrapyramidal side-effect due to aripiprazole which had started after alprazolam discontinuation, so propranolol 60 mg/day was added to the treatment and aripiprazole dose was decreased to 5 mg/day. After 3 days slurred speech was significantly improved. In the following 4 weeks period, her slurred speech totally disappeared and propranolol and aripiprazole was discontinued.

Case 2: A 20 year-old female patient presented to our hospital with hypomanic symptoms while on sertraline 50 mg/day and aripiprazole 5 mg/day treatment. Sertraline was discontinued, aripiprazole was increased to 15 mg/day and sodium valproate 1000 mg/day was initiated. 4 days after the increase of aripiprazole dose, she started to experience difficulty in tongue movements while speaking, impaired pronunciation and paresthesia around mouth and lips. In physical examination another extrapyramidal symptom was not founded. Aripiprazole treatment was discontinued and quetiapine 300 mg/day was initiated. In the following 2 weeks of period, her complaints was gradually improved.

Case 3: A 29 year-old female patient presented to our psychiatry outpatient clinic one year ago with depressive symptoms. She was diagnosed as depressive disorder and venlafaxine 75 mg/day was initiated and gradually increased to 300 mg/day. There was no clinical improvement in her depressive symptoms like anhedonia and fatigue, so aripiprazole 5 mg/day was initiated and increased to 10 mg/day dose in two weeks. In the 3rd day of aripiprazole dose increase, she started to experience difficulty in pronunciation and involuntarily wiggling her tongue. In physical examination another extrapyramidal symptom was not founded. Aripiprazole was discontinued and slurred speech gradually improved in the following week.

Various case reports of aripiprazole-induced acute dystonia report symptoms of neck extension, torticollis, rigidity, and tongue movements. In our cases, features like symptom initiation time, improvement after discontinuation, positive response to propranolol treatment, subjective restlessness sensation, difficulty in tongue movements show similarities between akathisia, acute dystonia and speech impairment related with aripiprazole. Clinicians must be watchful about aripiprazole-induced extrapyramidal phenomena like slurred speech.

Keywords: aripiprazole, slurred speech, extrapyramidal, antipsychotic

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[Abstract:0596][Motor disorders]

Two cases of neuroacanthocytosis manifesting with psychiatric symptoms

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Neuroacanthocytosis is a disease that may include findings of orofacial dyskinesia, choreiform movements of the extremities, dysarthria, dysphagia, areflexia, epilepsy, distal muscular atrophy and loss of strength, parkinsonism and dementia in later stages. On peripheral blood smear examination, acanthocytes over 3% accompanies the clinical findings above. It is a multisystemic degenerative disease that heterogeneous pathways found to be responsible in its etiology. In this report, we discussed two case of neuroacanthocytosis with psychiatric manifestation.

Case 1: A 38 year-old male patient presented to psychiatry outpatient clinic of Ankara University School of Medicine Department Of Psychiatry with self-talk, social withdrawal, impaired sleep patterns, difficulty while walking, eating and talking, involuntary contractions of face, neck and extremities. It is revealed when questioned during interview, after 2004 he had been experiencing social withdrawal, muttering by himself in front of TV, irritability, interpersonal problems, susceptibility until 2008 and consequently had been complaining of dysarthria, gait disturbance and involuntary contractions of face, neck and extremities. After the first admission to psychiatry outpatient clinic in 2009, he had been followed-up until 2011 with the diagnosis of behavioral disorder, dissociative disorder and psychotic disorder. During use of antidepressants and antipsychotics in this time interval, he had had one generalized tonic-clonic seizure. First line laboratory tests, electroencephalography, neuroimaging examinations that had performed in different medical centers had not given any evidence of disease. When he presented to our psychiatry outpatient clinic with increase in his involuntary contractions around the mouth in 2011, he was only able to feed by pipette with liquid food. He was diagnosed with neuroacanthocytosis after demonstrating over 20% acanthocytes on peripheral blood smear.

Case 2: A 34 year-old male patient were admitted to Etimeskut Military Hospital with involuntary contractions of mouth, arms and legs, insomnia and malaise. After a stressor event about 6 years ago, he had had social withdrawal, insomnia and fainting spells without tongue biting or urinary incontinence according to the information obtained from past medical history. For a long time he had followed with the diagnosis of conversion disorder and used antidepressants and antipsychotics. In this process, facial pain, feeling of instability, involuntary movements of arms and legs and the tongue had been added to initial symptoms. The patient were consulted a neurologist and internal medicine specialist. First line laboratory tests, electroencephalography, neuroimaging examinations were performed, but there was no pathological finding in the results. Nevertheless, over 20% acanthocytes on his peripheral blood smear indicated a diagnosis of neuroacanthocytosis.

Neuroacanthocytosis is a rare disease with uncertain etiology and characterized by korea, oro-facio-lingual dyskinesia, seizures and psychiatric disorders as well as acanthocytes on peripheral blood smear. It has a wide range of psychiatric symptoms. Cognitive impairment, personality and behavioral changes are common and also anxiety, paranoia, depression, obsessive behavior and emotional lability can be seen. In those cases we report, social-emotional withdrawal, suspiciousness and depressive symptoms were prominent. Neuroacanthocytosis, as a multisystemic degenerative disease that should be considered in patients with atypical appearance and with treatment unresponsiveness.

Keywords: neuroacanthocytosis, chore, dyskinesia

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[Abstract:0597][Mood disorders]

Valproic acid -induced acute and nearly complete hair loss: a case report

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Hair loss is a frequent side effect that can be seen with the use of psychotropic drugs, such as valproic acid, lithium, carbamazepine, SSRIs and antipsychotics. Valproic acid related hair loss is mostly described with the high dose serum valproic acid concentrations (70-124 µg/ml) and rarely with the low dose concentrations (25-50 µg/ml) and therapeutic window (40-110 µg/ml). In this report, we aimed to discuss possible induction of acute and near to complete hair loss by valproic acid, while serum level was in the therapeutic range, through a case report with bipolar affective disorder.

Case: A 45 year-old female patient, married, elementary school graduate woman with a history of bipolar disorder had been receiving olanzapine 7.5 mg/day and lithium 900 mg/day treatment since April 2013. The mood stabilizer medication was switched to valproic acid treatment at May 2015 due to patient's treatment non adherence because of the gastrointestinal and neurological side effects of lithium. Lithium treatment stopped within two weeks and valproic acid started at a dose of 500 mg/day and than increased. Her plasma concentration of valproic acid was 77 µg/ml on 21st day (dose: 1,250 mg/day). Hair loss which was acute and near to complete was detected on 28th day. Therefore, valproic acid treatment was discontinued. She consulted to dermatology clinic and she was diagnosed with hair loss of unknown reason. Blood samples including full blood chemistry, full blood count, thyroid function tests and prolactin, estrogen, dihydrotestosterone, vitamin B12, folic acid, copper and iron, which were assessed in order to exclude the probable organic causes, were in the normal ranges. Thyroid antibodies and antinuclear antibodies were not determined. No medical and mental alopecia history were detected in patient's family history. Other psychiatric diseases that may cause hair loss (e.g. trichotillomania) were excluded. There was no pre-treatment period that may cause hair loss. After discontinuation of the valproic acid treatment patient's hair loss diminished within three weeks and returned to its initial condition in about three months.

Hair loss with valproic acid having incidence of 3.5%, is diffused, non-scarring and mostly reversible with reduction or withdrawal. In most cases, hair loss is associated with long-term valproic acid pharmacotherapy and high serum levels, commonly beginning 2 to 6 months after initiating treatment and also complete hair loss is rare. In our case hair loss was acute and near to complete with therapeutic serum valproic acid levels. In conclusion hair loss has to be considered severely after valproic acid treatment was started on. In fact it may cause low compliance and than provoke relapses.

Keywords: bipolar disorder, hair loss, therapeutic serum level, valproic acid

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[Abstract:0599][Schizophrenia and other psychotic disorders]

Neuroleptic induced tardive myoclonus that misdiagnosed and treated as restless leg syndrome for years: a case report

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Tardive syndromes (TS) classified as tardive dyskinesia, tardive dystonia, tardive akathisia, tardive myoclonus and tardive parkinsonism. TS are found in about 20–25% of patients treated with dopamine receptor antagonists and 35% of those who stopped medication. Clozapine has been shown to improve TS in some cases and some reports. Restless legs syndrome (RLS) is characterized by the presence of abnormal feeling in the legs experienced when at rest or during periods of inactivity in the evening or at night and relieved by movement. Symptoms of RLS and tardive myoclonus seem like each other sometimes but treatments are not same. In this report we present a case of tardive myoclonus that was diagnosed as RLS than treated with pramipexole and pregabalin for five years but hadn't responded however improved with low dose clozapine treatment.

Case: A 60 year-old female patient, never married, living in care center, was diagnosed schizophrenia for 20 years. She had a hospitalization in 1998 with psychotic episode and haloperidol 10-20 mg/day treatment had started on. One month later she had discharged from hospital with the treatment haloperidol 15 mg/day and biperiden 4 mg/day. After 1 year, movement disorder had started on her two legs. She continued same treatment for 5 years and than gave up by herself but hadn't been on regular clinician visits. Her complaints about movement disorder continued and she had any drug treatment in this period. Four years ago pramipexole 0.250 mg/day and pregabalin 300 mg/day had started by a neurologist with diagnose of RLS. She had been receiving same treatment when consulted us for a forensic reason. At this time the abnormal movements that characterized by rapid, brief myoclonic jerks in the legs was determined by us. Considering the conventional antipsychotic use in past, not respond to RLS treatment and according to symptom features it is evaluated as tardive myoclonus (TM) and clozapine treatment initiated to her. TM relieved dramatically after ten days with 100 mg/day clozapine treatment.

RLS is widely underdiagnosed or misdiagnosed because the disorder is not well understood. Our patient was not clearly satisfied the criteria of RLS as per the International RLS Study Group (IRLSSG). Also her symptom of myoclonus on legs was not worsen at rest, inactivity, night time, and also that was not partially or completely relieved by movement. Myoclonus was continuously in all day and did not respond to pramipexole and pregabalin treatment that shown effective in RLS. The dose of clozapine used for management of TS varied from 100 mg to 900 mg/day but in most of the studies the dose was between 300 and 500 mg/day. The present studies suggest that improvement of TS starts within 1 to 12 week of after clozapine treatment. In our patient improvement on TD started within 1 week and myoclonus on legs regressed near complete in ten days. Tardive myoclonus must be considered in the assessment of patients with movement disorders. Evaluation of patients psychiatric history and physical examination carefully may prevent misdiagnose of RLS. The clinicians must pay attention more and get consultians in necessary.

Keywords: clozapine, restless leg syndrome, tardive myoclonus

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[Abstract:0600][Psychopharmacology]

Urticaria and angioedema associated with oros-methylphenidate

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Methylphenidate (MPH) is a first-line psychostimulant treatment for Attention deficit and hyperactivity disorder (ADHD). Several case reports have described allergic adverse events during MPH treatment including, skin eruptions, urticaria, angioedema, contact dermatitis, and exfoliative dermatitis. We present a 7 year-old-boy with ADHD who experienced urticaria and angioedema during OROS-MPH treatment.

Case: A 7 year-old boy was referred to the child and adolescent psychiatry out-patient clinic by his parents because of his hyperactivity

and poor attention. In his psychiatric assessment a diagnosis of ADHD (combined type) was given and he was started on OROS-MPH 18 mg once daily. His mother reported that on the 5th day of the OROS-MPH treatment, he developed swelling in his right infra-orbital area. He had rashes in his both hands and feet which spread to all over the body within the next day. He was not taking any medications other than OROS-MPH and no past history of any allergic reaction was reported. This condition was considered by his pediatric clinician to be a possible adverse effect of OROS-MPH as urticaria and angioedema. OROS-MPH administration was ceased, and he was treated with hydroxyzine (a first-generation antihistamine). Then his symptoms resolved completely in next few days. We decided to initiate immediate release (IR) methylphenidate 5 mg twice-daily two weeks after the allergic reaction. He had no adverse reaction and tolerated the medication well for 2 months.

We presented a pediatric case that developed urticaria and angioedema five days after the administration of OROS-MPH, showed complete resolution with subsequent removal of OROS-MPH, and did not experience any allergic reaction during IR-MPH treatment. In the literature, there are two case reports on the association between urticaria and angioedema and stimulant treatment. Angioedema is characterized by non-pitting edema of the dermis and subcutaneous layers. Urticaria is characterized by an eruption of wheals, with or without angioedema. It is suggested that acute urticaria and angioedema usually occur due to mast cell and basophile activation from multiple triggers such as infections, medications, and foods. The mechanism in our case most probably is that specific IgE was produced after first administration of OROS-MPH and five days later, on subsequent contact, the allergen bound to specific IgE, and then this complex cross-linked high-affinity IgE receptors on the surface of mast cells. This event triggered the mast cells to cause urticaria and angioedema through releasing histamine and vasoactive mediators. In our case, urticaria and angioedema occurred probably with a type I allergic reaction to the substances available in OROS-MPH capsules in which not available in IR-MPH tablets. Coskun et al. (2009) have reported a child who developed skin eruptions during OROS-MPH treatment but not with IR-MPH and suggested several substances in the OROS-MPH capsule as the probable cause of this allergic reaction. It may be also the same mechanism in our case. In conclusion, clinicians should be careful for this rare symptom when prescribing psychostimulants. Switching to another form of MPH may be an option for managing this rare adverse reaction.

Keywords: allergic reactions, angioedema, methylphenidate, urticaria

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[Abstract:0602][Psychopharmacology]

Aripiprazole-induced Raynaud's syndrome: a case report

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Aripiprazole is the first drug among second generation antipsychotic drugs that have the partial agonistic effect on dopamine D2. In addition, aripiprazole has a partial agonist effect on serotonin 5-HT1A while it has an antagonistic effect on serotonin 5-HT2A receptors. In current studies, it was demonstrated that aripiprazole is effective in the treatment of bipolar disorders, tic disorders, schizophrenia, developmental delays, aggressiveness and self-injury behavior seen in pervasive developmental disorders in children and adolescents. This case report, presents the Raynaud's syndrome which occur during the aripiprazole treatment and improve with stopping of the treatment.

Case: A 15 year-old female patient who dropped out at middle school. She was brought to our clinics by her family with the complaints run a way from home, frequently arguing with family members, lying, self-harm,increase in anger. In the clinical assessment it was revealed that she has had these complaints for the last one year, she has impulsive behavior, sexual behavior and suicidal attempts. It was found out that she had euthymic mood, regular sleep pattern, and her appetite was good. The patient did not have anhedonia. After psychiatric examination and psychometric tests, she was pre diagnosed with conduct disorder and borderline personality disorder. The patient was prescribed risperidone 0.5 mg/day. After two week follow-up, the risperidone was discontinued since the patient had excessively increased appetite and weight gain (5 kg). Thereupon, aripiprazole was started 2.5 mg and one week later aripiprazole was increased to 7.5 mg. During the next visits, it was revealed that the symptoms were gradually discontinued. In the fifth month of the treatment, the patient presented with the complaints of swelling and bruising of toes. The patient did not have these complaints before. The general pediatry, pediatric rheumatology, pediatric allergy, dermatology investigations were made. She was finally diagnosed with Raynaud's syndrome. In the ethiologic investigation any reason could not be found. It was thought that Aripiprazole -induced Raynaud's syndrome. Aripiprazole was stopped and follow up without medication was advised to the patient. One month later, it was seen that she had totally recovered from swollen and bruised toe syndromes, and they did not repeat again. The treatment and follow-up period of the patient is continued by our clinic.

In this case report, we presented a Raynaud's syndrome case, which occured during aripiprazole treatment and improved with stopping of the treatment. Since there is limited information in the literature,related to the side effects of aripiprazole in peripheral circulation, this study will make an important contribution to the clinicians who work in this field.
Keywords: adolescents, aripiprazole, Raynaud's syndrome

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[Abstract:0605][Psychopharmacology]

Case of gynecomastia and galactorrhea on a male patient due to the use of paliperidone palmitate

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Atypical antipsychotics are less potent D2 receptor antagonists than classic antipsychotics, therefore they cause a medium level or temporary increase in prolactin levels. Among second generation antipsychotics; risperidone and amisulpride increase prolactin levels similar to typical antipsychotics, whereas clozapine, olanzapine, quetiapine, aripiprazole and ziprasidone do not considerably increase prolactin levels. Paliperidone is an atypical antipsychotic, which is created by combining the active metabolite of risperidone called 9-hydroxy risperidone using osmotic controlled release oral delivery system (OROS) technology. Because of having a receptor profile similar to risperidone, retaining a longer half life and being less lipophilic, it's believed to play a significant role on elevated prolactin levels. In this case report; secondary hyperprolactinemia caused by long acting paliperidone palmitate use, resulting gynecomastia and galactorrhea on the bipolar disorder diagnosed patient, inclusion of partial dopamine antagonist aripiprazole and the following decrease in prolactin to normal level and disappearance of the symptoms of galactorrhea and gynecomastia are discussed.
Case: A 45 year-old male patient, dentist. Presented our outpatient service on January 2013 presenting complaints of excessive talkativeness, irritability, sleeplessness, depression, languidness, distractibility, skepticism and sensitivity. Diagnosed with mixed bipolar disorder, he was admitted to inpatient treatment. After being discharged the treatment was adjusted as valproic acid 1000 mg/day and aripiprazole 30 mg/day. On march 2015 after another mixed attack from the patient he was admitted once more. After discharge the treatment was adjusted as valproic acid 1750 mg/day, paliperidone prolonged release 100 mg once IM every 30 days, biperiden 2 mg/day. During outpatient service, symptoms of gynecomastia and galactorrhea appeared on patient. His prolactin levels were detected as 60 ng/ml at the time. As the patient visibly benefited from paliperidone palmitate prolonged release treatment, instead of decreasing the dosage aripiprazole 5 mg/day was added to the treatment. During the process aripiprazole dosage was increased up to 10 mg/day. Approximately 4 weeks later controlling prolactin levels showed a decline to 36.7 ng/ml. Symptoms of galactorrhea disappeared and gynecomastia visibly reduced on the patient and his latest medical treatment is valproic acid 2000 mg/day, paliperidone palmitate prolonged release 100 mg once every 30 days, aripiprazole 10 mg/day and biperiden 2 mg/day.
When hyperprolactinemia is detected, the decision should be made according to the patient's possible benefits from the treatment; lowering antipsychotic dosage, switching to another antipsychotic that does not affect prolactin levels or adding a partial dopamine agonist are some of the options. Aripiprazole is the first potent D2 partial agonist among new generation of antipsychotics. For its antagonist effects in hyperdopaminergic conditions and agonist effects in hypodopaminergic conditions, it is known as dopamine regulator. Aripiprazole bonds with D3 receptors with great affinity. It is a partial agonist to 5-HT1A receptors while an antagonist to 5-HT2A receptors. Aripiprazole can be used on patients to reduce the effects of symptomatic hyperprolactinemia and to create antipsychotic effect. The point we're drawing attention to in this case report is the need to pay attention towards male patients for hyperprolactinemia and related symptoms during antipsychotic treatment as much as female patients
Keywords: paliperidone palmitate, hyperprolactinemia, aripiprazole

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[Abstract:0607][Psychopharmacology]

Atomoxetine-induced qtc prolongation: a case report

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Attention deficit hyperactivity disorder (ADHD) is characterized by inattention, hyperactivity and impulsivity and is the most common psychiatric disorder in childhood with a prevalence of 8%-12%. Atomoxetine, the first non-stimulant drug approved by FDA for ADHD treatment, selectively inhibits norepinephrine reuptake. Several studies report that atomoxetine is well-tolerated with low side effect profile. Atomoxetine is associated with increase in heart rate, systolic and diastolic blood pressure, orthostatic hypotension, shortened PR-interval. Although studies conducted on cardiovascular side effects of atomoxetine reported no statistically significant change in QT-interval in general, life threatening prolongation of QT was reported as a very rare condition. In this report, we will present a case with atomoxetine-induced prolonged QT-syndrome, which recovered with drug withdrawal.
Case: A 16 year-old boy presented to our outpatient unit with complaints of distractibility, getting bored quickly, forgetfulness, frequently losing things, frequent quarrels with friends. His overactivity symptoms were present since preschool years and inattention, poor school performance, forgetfulness, not obeying class rules and fighting with friends since the first grade. The family stated that hyperactivity decreased in time; however, attention problems and impulsivity persisted. His medical history was otherwise unremarkable. his father and brother was said to have the same problems. His teacher's report about ADHD symptoms was parallel with the family. The Patient was diagnosed with ADHD according to DSM-5 criteria Hematologic and biochemistry tests were within normal limits. There were no problems Electrocardiography (QTc: 420 ms, sinus rhythm, heart rate: 80/min) and blood pressure. Atomoxetine 0.5 mg/kg/day was gradually increased to 1.2 mg/kg/day. He returned for follow-up visit after 2 months. Major improvements were observed in inattention and impulsivity complaints. However, he described post-stress dyspnea, quick exhaustion and palpitations. ECG showed a QTc prolongation (heart rate: 110/min and QTc: calculated as 550). Patient was consulted with Pediatric Cardiology. Blood parameters and echocardiography were within normal limits, prolonged QT-interval was attributed to atomoxetine treatment thus ATX was stopped. 1 week later a quick exhaustion and palpitations disappeared. ECG was in sinus rhythm (QTc: 396 ms, heart rate: 84).
Atomoxetine-induced prolonged QT-interval is a rare condition. Patients who develop palpitations, dyspnea and quick exhaustion after drug initiation should be monitored carefully and an ECG evaluation is curitial in this patients. The mechanism underlying QT prolongation related with ATX is unclear but considering that prolonged QTc has been related with high doses of drug in some case reports and that atomoxetine is metabolized in liver by CYP2D6, prolonged QT-interval may be related with slow metabolizing. Consequently, atomoxetine is generally considered a well-tolerated and safe drug; however, clinicians should keep cardiovascular side effects in mind.
Keywords: ADHD, atomoxetine, QTc

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[Abstract:0611][Schizophrenia and other psychotic disorders]

Risperidone induced drug eruption: a case report

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Risperidone is an atypical antipsychotic which has the potential of binding various receptors. It is used in schizophrenia and other psychotic disorders, bipolar disorder, autism, personality disorders and such others (e.g., impulse control disorders). Although lots of side effects associated with risperidone, there are a few reports about risperidone associated skin reactions in the literature. In this report, we aimed to discuss a case about risperidone associated drug eruption.
Case: A 37 year-old female patient, single, graduated from a university, but had not worked in last 1 year. She is living with her family. In last 2 years, she has had some complaints that auditory and visual hallucinations aggression, hostility, insomnia, suspiciousness, becoming introverted. It is learned form her history that her complaints have been increasing in some periods and she have never been

improved enough until now. She had a psychotic episode 8 years ago, than she had refused the treatment and never used medication. There was not a significant disease, allergy, skin hypersensitivity, substance abuse and alcohol use in her history and a psychiatric disorder in her family history. In her mental status examination irritability, paranoid thoughts, persecutory and referential delusions, sleeplessness, auditory and visual hallucinations were determined. In January 2015, she hospitalized for treatment and Risperidone 1 mg/day started orally. After a week, the dosage increased up to 3 mg/a day. After that some skin lesions developed at her skin particularly at her paunch and trunk within three days. She is consulted to a dermatologist for her lesions, dermatologist indicated that there were a lot of macular, erythematous, morbilliform eruptions in diameter 2-10 mm on her trunk. Discontinuing to current psychiatric medication and starting corticosteroid and antihystaminic treatment is recommended for her lesions. Her skin eruptions disappeared within one week. After that psychiatric medication was started as Olanzapine 5 mg/ a day orally and Zuclopenthixol depot 200 mg/ 15 days parenterally. Her drug eruptions did not repeat again.
In our case, we concluded that the skin eruptions were associated with risperidone due to drug eruptions was determined after using Risperidone than disappeared after discontinuing. Drug eruptions are associated with lots of medication, by immunologic and non-immunologic mechanism (like photodermatitis). Drug eruption induced by atypical antipsychotics are rare in the literature and the mechanism of that is not exactly known yet. In our case, a drug eruption developed in a patient who is not known any allergy and skin hypersensitivity in her history. In risperidone treatment, skin eruption like defined in this case must be considered and get consultations by clinicians in necessary.
Keywords: drug eruption, psychosis, risperidone

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[Abstract:0612][Psychopharmacology]

Aripiprazole-associated rhabdomyolysis in a child with mental retardation

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Aripiprazole is a partial dopamine agonist and its use has been spreading in child and adolescent clinical psychiatry practice for treatment of aggression and behavioral problems in psychotic disorder, bipolar disorder, autism and mental retardation. Although rare, antipsychotic treatment has the potential to cause side effects with severe consequences such as neuroleptic malignant syndrome and rhabdomyolysis. Early diagnosis and treatment is of utmost importance in rhabdomyolysis, which may lead to acute renal failure and compartment syndrome. There is very limited number of articles in literature regarding development of rhabdomyolysis in children secondary to antipsychotic agents. In this poster we report a child who developed rhabdomyolysis while receiving aripiprazole treatment and discuss related aspects of diagnosis, risk factors, and important points.
Case: A 6 year-old boy was brought to emergency service with complaints of fatigue, abdominal pain and dark urine. Patient was under follow-up for moderate mental retardation and conduct disorder. He received risperidone 2 mg/day for 1.5 years, but no benefit gained for his symptoms; therefore, aripiprazole 2.5 mg/day was started His family described significant improvement with aripiprazole treatment; however, he recently developed bradykinesia and contraction in extremities, and family decided to bring him to emergency after observing darkened urine. Patient's vital signs were stable. He was alert and cooperative. Orientation was limited due to mental retardation, affect and mood were irritable. Rigidity was evident in both arms. Laboratory test results; CBC values were within normal limits, AST: 1760 UI/L, ALT: 860 UI/L, Creatinine kinase: 8820mcg/L, myoglobin: 1820 ng/ml, LDH: 1854 IU/L, GGT:33 UI/L. Renal function tests and serum electrolyte levels were within normal limits. Arterial blood gases were normal and auto-antibodies were negative. Urinalysis; color: brown, pH: 7, Density: 1005, trace protein; ketone and glucose were negative, erythrocyte was negative, WBC: 2/hpf. Hepatitis A, B, C markers, EBV IgM, CMV IgM, Toxo IgM and anti-HIV were negative. Tandem-MS and urinary organic acid levels, determined to rule out metabolic diseases, were negative. Stable vital findings (lack of fever and autonomic instability) ruled neuroleptic malignant syndrome, serotonergic syndrome and lack of viral infection findings with negative viral parameters ruled out viral rhabdomyolysis. Rhabdomyolysis secondary to antipsychotic use was considered; aripiprazole was stopped and fluid replacement was performed. His findings and blood parameters normalized within a few weeks and he was discharged.
Rhabdomyolysis is leakage of skeletal muscle proteins to plasma as a result of muscular disintegration. Clinical presentation is generally characterized with muscle pain, fatigue and darkened urine. Serum creatine phosphokinase (CPK) level elevates along with myoglobinuria, which needs early intervention due to potential risk of acute renal failure and compartment syndrome. Common findings of rhabdomyolysis are muscle pain, abdominal pain, fatigue, cramps and dark urine. Intense physical exercise, intramuscular injection and

alcohol use increase risk of rhabdomyolysis; care should given at times of treatment initiation, dose increments and change in treatment regimen. Taking account of early findings and risky periods facilitates diagnosis of rhabdomyolysis secondary to antipsychotic agents and provide early intervention in order to prevent acute renal failure.
Keywords: antipsychotic, aripiprazole, rhabdomyolysis

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[Abstract:0614][Perinatal psychiatry]

Paternal postpartum depression: a case report

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Postpartum depression (PPD) affects 1 in 10 fathers worldwide. Paternal PPD tends to develop gradually during the first year. Maternal depression is one of the most important risk factors. Changes in hormones during the postpartum period in fathers may be biological risk factors in paternal PPD. Fathers who have ecological risk factors such as excessive stress from becoming a parent, lack of social supports for parenting and feeling excluded from mother-infant bonding may be more likely to develop paternal PPD. Paternal PPD has negative effects on the infant's development, independently of maternal PPD. It's essential to identify paternal PPD at early stage. We aimed to create awareness about this phenomenon by offering paternal PPD.

Case: A 28 year-old male patient, married since 1.5 years, has one child. The patient's symptoms have began five months before applying for examination with the birth of his first child. He said he was more comfortable when his sister was in home to help his wife and him for care of baby during first month but noted that his complaints increased after his sister go from house. He said he was worrying about the health of the baby when baby was crying, he felt anxiety and restlessness, he felt bad when he could not calm the baby. Then he said he felt angry to baby, and did not want baby, wanted to kill it and himself to get rid of this annoyance. In the biography of the patient we learned that his parents divorced when he was two year-old then he begun to live with his aunt but after 8 years when he was ten year-old because of the husband of aunt's conflict he have to live her and begun to live alone. In mental status examination the speech was as to answer questions, and arrived to goal. His mood was depressive and irritable. In his thought content there was no toleration to baby and worries about the unsettled theme about to return home. He had insomnia and decreased appetite.

New demands and responsibilities during the postpartum period often cause major changes in a father's life. In our case his own father was not a good role model for him. In addition because of being father for the first time, being new married, having new responsibilities related to marriage and baby, lack of social support predisposed the depression. Studies so far have only used diagnostic criteria for maternal PPD to investigate paternal PPD, so there's an urgent need to study the validity of these scales for men and develop accurate diagnostic tools for paternal PPD. For fathers, different types of support may ease the transition process to fatherhood during the postpartum period. The most effective supports likely come from their partners because paternal PPD is closely related to partners' mental health and their relationship with the fathers. Also, educational programs, policy for paid paternal leave, as well as consideration of psychiatric care may help fathers cope with stressful experiences during the postpartum period.
Keywords: paternal depression, postpartum depression, fathers

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[Abstract:0615][Psychopharmacology]

Levetiracetam -induced mania-like symptoms in an adolescent

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Levetiracetam, a novel second-generation antiepileptic agent with a good side-effect profile, is used for the treatment of partial and generalized epilepsy. Levetiracetam is widely used in children and adolescents owing to its ease of oral administration, excellent bioavailability, and low rate of drug interactions as it is not metabolized by the liver or bound significantly to serum proteins. Though generally well tolerated, it may cause some behavioral adverse effects including depression, hostility, agitation, emotional lability, anger, nervousness, and depersonalization. Hereby, we report a case of a 15 year-old boy who developed levetiracetam-induced mania-like symptoms.

Case: A 15 year-old boy presented to our clinic with complaints of decreased need for sleep, internal feeling of excitement, excessive desire to run and increased energy. His father reported that he was running between home and father's market (approximately 15 kilometers) six times a day. He had a history of generalized tonic clonic seizures for two years for which he was started on levetiracetam 20 mg/kg/day and dose had been increased to 40 mg/kg/day by a weekly dose titration. Three weeks after beginning levetiracetam treatment, seizures were controlled, but the patient developed the above-mentioned mania-like symptoms. There was no history of manic/hypomanic symptoms or psychiatric disorders before starting levetiracetam therapy. There was no known history of psychiatric disorders in both parents and extended family members. A levetiracetam-induced behavioral adverse effect was suspected and the patient was consulted to the pediatric neurology department for an evaluation of a change in antiepileptic medication treatment. Since seizures were controlled with levetiracetam, child neurology department had advised to continue ongoing treatment. To control the mania-like symptoms, risperidone in the dosage of 0.5 mg/day was added to the ongoing treatment with levetiracetam. One week later, risperidone dosage was increased to 1 mg/day and the mania-like symptoms gradually declined within a week.

Levetiracetam, a piracetam analog, has a novel chemical structure and a unique mechanism of action. It exerts its antiepileptic effects by specifically binding to synaptic vesicle protein 2A, inhibiting calcium release from intra-neuronal stores. Levetiracetam, leading to an indirect enhancement of benzodiazepine GABA receptor function by removing the negative modulation of this site by zinc and beta carbolines or other GABAergic mechanisms, is also used rarely as an adjunctive mood stabilization agent with a controversial effect. By contrast to its use on emotional symptoms as an adjunctive agent, levetiracetam has been shown to cause emotional and behavioral adverse effects in case reports. In the current literature, there are two adult female cases (37 and 58 year-old) which have been reported acute mania after levetiracetam treatment. Psychotic adverse events have been reported more than mania. To the best of our knowledge, this is the first case of levetiracetam -induced mania-like symptoms in an adolescent. Given the widely use of this antiepileptic agent, clinicians should be aware of this side effect. As in our case, if there is no chance of changing antiepileptic drug, risperidone may be considered as a good choice for reducing mania-like symptoms.

Keywords: adolescent, levetiracetam, mania

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[Abstract:0616][Psychopharmacology]

Exacerbation of urinary incontinence in an autistic adolescent with aripiprazole treatment

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Aripiprazole is a new generation atypical antipsychotic agent. Food and Drug Administration (FDA) indicated aripiprazole for the treatment of schizophrenia, bipolar disorder and behavioral symptoms associated with autistic disorder in children and adolescents. Hereby, we present a 13 year-old female adolescent with Down Syndrome who had an exacerbation of urinary incontinence with aripiprazole treatment.

Case: A 13 year-old female adolescent with Down Syndrome was referred to our clinic with the complaints of sadness, social withdrawal and repetitive behaviors which all increased through the past two years. She had been diagnosed with mild mental retardation and autism spectrum disorder in early childhood. Her medical history revealed mild urinary incontinence with a frequency of once a week. With the presenting depressive symptoms, she was diagnosed with depressive disorder not otherwise specified. In order to target both the repetitive behaviors and the depressive symptoms, aripiprazole was started in the dosage of 2 mg/day. Three weeks later, parents confessed that they have accidentally administered a dosage of 5 mg/day and the patient had a marked increase in her urinary incontinence. She was reported to have multiple incontinence episodes almost every day since the start of the aripiprazole treatment. With the suspect of a medication -induced exacerbation, aripiprazole was decreased to the former recommended dose of 2 mg/day. Upon decreasing the dose, the frequency of urinary incontinence was reported to reduce to the initial level. To confirm the adverse reaction, aripiprazole dosage was again increased to 4 mg/day in divided doses. Upon the increase, incontinence also exacerbated. After these trials, aripiprazole was discontinued and urinary incontinence was reported to resolve back.
We reported to increased in urinary incontinence in an autistic adolescence with usage of aripiprazole. Drug-induced urinary incontinence has been described with nearly all typical antipsychotics, but it has been very rarely reported with atypical antipsychotics, but these adverse effect probably has been seen more common than generally reported. Aripiprazole has a unique mechanism of action as a dopamine D2 partial agonist, serotonin 5-HT(1A) partial agonist and serotonin 5-HT(2A) antagonist. The antagonist activity of aripiprazole at 5HT2A and alfa 1 receptors on detrusor muscle and internal bladder sphincter might have contributed to urinary incontinence. There are only two case reports on urinary incontinence associated with aripiprazole, both are developed in autistic children as well as in our case. Therefore, we should be aware of urinary incontinence when prescribing aripiprazole especially in autistic children.
Keywords: aripiprazole, autism spectrum disorder, urinary incontinence

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[Abstract:0617][Mood disorders]

Can D-dimer serum be used in the diagnosis and the process of catatonia? A case report

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Catatonia symptoms include: Stupor, waxy flexibility, catalepsy, mutism, negativism, posturing, mannerism, stereotype, agitation, grimes, the echolalia and echopraxia. Clinicians generally identify this diagnoses at the hospital, 35% of schizophrenia patients have catatonia disorder but most of catatonia cases become depressive disorder and bipolar disorder. If we cannot diagnose,treat catatonia correctly, it will become the lethal catatonia, which is mortal, therefore early diagnoses and proper treatment is important in catatonia, and because of this, researchers try to find new biological markers to diagnose catatonia. Haozir et al. conducted a study on the etiology of catatonia and they hypothesized that coagulation system might be activated without immobility in non-lethal catatonic patients and this condition helps us to diagnose, treat and make follow-ups for reviews. For this hypothesis further researched that catatonic patients' serum D-dimer level and findings proved that there is a significant relationship between the D-dimer level and catatonia. This case report is based on the correlation between D-dimer level, catatonia diagnosis and management in a secondary catatonia and major depressive disorder with psychotic features patient and will be discussed in the scientific literature.
Case: A 20 year-old single male patient was brought to hospital emergency service because of mutism, refusal to eat and drink, auditory hallucination and decreased motor activity caused by lack of movement and lack of any social communication. According to his medical history, we admit him in the psychiatry clinic due to the catatonia associated with major depressive disorders with psychotic symptoms. His serum D-dimer level was measured the result was 5300 ng/ml. We could not diagnose the reason for the high D-dimer level. We had to start the medical treatment by giving him lorazepam 2.5 mg/day, sertraline 50 mg/day and olanzapine 5 mg/day. His D-dimer level on the first day of admission was 6700 ng/ml and his Hamilton depression rating scale (HAM-D) score was 39 and the Bush-Francis catatonia rating scale (BFCRS) score was 30. On the third day of admission, there was no clinical improvement and we start electroconvulsive therapy while continuing sertaline 50 and olanzapine 5mg/day treatment. After the twelfth ECT session the following results were observed HAM-D score of 6, BFCRS score of 1 and D-dimer level score of 400 ng/ml. After his recovery there was a clear psychiatric evidence with no evidence of catatonia. He was discharged with these treatments: sertraline 50 mg/day and olanzapine 5 mg/day.
In our case, based on our patient we were not able to find any organic reasons for the elevation of D-dimer level, however we suggest

that this elevation of D-dimer level is because of one of the pathophysiology of catatonia which depends on the increase in peripheral dopaminergic hypofunction and the activation of peripheral noradrenergic system, this has caused activation of coagulation. As one of the most important findings during our patient's treatment, we realized that there is a positive correlation between the serum level of D-dimer with HAM-D and BFCRS scores. After the ECT sessions while the HAM-D and BFCRS scores declined, the D-dimer level decreased too.

Keywords: catatonia, d-dimer, psychotic depression

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[Abstract:0621][Anxiety disorders]

A case report of a patient with bathmophobia treated with EMDR therapy

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Everyone has some fears; some of them are reasonable and some of them are not. Indeed, fears, and anxiety that comes up with fear can serve for taking precautions. However, if an anxiety is persistent and uncontrollable, it will decrease functionality in daily life. As an extreme form of fear, phobias are unreasonable, constant, and intense; moreover they become without any real danger. Phobias may be connected with different situations or objects such as height, chocking, water, flight, dental, and so on. On the other hand, some people are intensely afraid of slopes and stairs, which is called bathmophobia. Phobias can be treated with a variety of treatment options like Cognitive Behavioral Therapy (CBT), Prolonged Exposure Therapy (PE) and Eye Movement Desensitization and Reprocessing (EMDR). EMDR is a therapy method, which was found by Shapiro and used for treating Posttraumatic Stress Disorder (PTSD); nevertheless it had been used for treating other mental disorders including phobias. In the present case report, efficacy of two sessions of EMDR therapy on a patient with bathmophobia will be indicated.

Case: A 54 year-old female patient working as an engineer and engaging in hobbies such as trekking and reiki presented to our clinic with the complaints of feeling worried while going up with her car when she drives and feeling afraid of becoming lonely after an accident that took place during trekking. It was told that while going up in the trekking, her foot slipped and she fell down. After that, she felt anxious about going for trekking again. An EMDR protocol was administered to the patient in which she was asked to think of the oldest memories related to the beliefs about that event she feels anxiety about, therefore, she recalled an event from age 12 that she fell down while climbing a slope on road to home, leading her to feel alone and helpless. Hence, one session of EMDR was administered on patient focusing on that event. In the following interview arranged for 2 weeks later, she expressed that this event lost its impact and that she made new plans for trekking. As a result of two sessions of EMDR, the patient's Beck Anxiety Inventory (BAI), Beck Depression Inventory (BDI), Peritraumatic Dissociation Experiences Questionnaire (PDEQ), and Impacts of Events Scale-Revised (IES-R) scores significantly decreased. Her traumatic memory, which is about going up to the hill, lost its emotional vividness and the patient put away her self-belief of "helplessness". Additionally, functionality of the patient visibly increased; even she planned new trekking events. As an inference from the results of her treatment, it can be said that, EMDR may have an impressive effect on patients by decreasing the negative impacts of different types of phobias.

EMDR therapy can be a beneficial option for treating phobias besides of PTSD and other mental disorders. Even in a short span of time, EMDR may be helpful to decrease devastative impacts of phobias and good to increase operationalization of individuals.

Keywords: anxiety, bathmophobia, case, EMDR, phobia, treatment

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[Abstract:0623][Psychopharmacology]

Treatment of pathological internet use with aripiprazole, in an adolescent with ADHD comorbid OCD

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Aripiprazole is an atypical antipsychotic used for a variety of indications, including psychotic, autistic and bipolar disorders in children and adolescents. Pathological internet use (PIU) is being disable to control excess internet usage, spending more time with internet than permitted by parents, feeling more aggressive and irritable when internet is not accessible and as a result of these; weak relations with social surroundings and family. Here, we want to present a patient with PIU whose symptoms were improved with aripiprazole.
Case: A 12 year-old male patient with myelodysplastic syndrome, he presented to our department with the complaints of ruminative thoughts, checking mistakes, counting things and fear of losing someone. His mother and uncle were also being treated with OCD. Additionally he has symptoms such as hyperactivity, home-accidents, watching TV over 6 hours since early childhood. He was diagnosed as "ADHD comorbid OCD" based on DSM-IV. We started Oros Methylphenidate because of increasing accidental injury. One month later, we switched to Atomoxetine because of increased anxiety symptoms. After a treatment period of 6 months by using 40 mg Atomoxetine per a day, his obsessive-compulsive symptoms and fears have completely disappeared but the time he spends online and symptoms of hyperactivity and inability to concentrate have increased. Because of this, Atomoxetine treatment was exchanged by immediate release methylphenidate (IR-MPH). By an outpatient treatment during 2 years, obsessions and hyperactivity symptoms decreased partly. His mother mentioned that during last one year, patient had an increased wish of playing game online, sleeping difficulties and school discontinuity. Because of summer period and lack of appetite, IR-MPH was stopped due to parent's request. Aripiprazole 2.5 mg/day was started and titrated up to 5 mg/day gradually. Three weeks later; it was learned that he gave up spending time on pc, started reading books but anxiety and crying episodes began. His anxiety was assessed as akathisia and lorazepam 5 mg/day was given for three weeks. At the end of three weeks, firstly lorazepam treatment and then because of continuing anxiety symptoms, Aripiprazole also stopped. In the 15th of unmedicated day, excess usage of internet was observed again. The patient has still been treated by Aripiprazole and Lorazepam for three months. PIU and OCD have been totally treated but ADHD still remains.
Many studies show an association between PIU and ADHD in children and adolescents like this case. Self-control deficits and impulsivity may increase the predisposition to PIU in ADHD cases. Moreover, PIU can be classified as OCD and/or impulse control disorders. Aripiprazole has been reported as an effective agent in some cases with OCD and impulse control disorders. Therefore, the role of Aripiprazole in PIU treatment can be related to its effect on impulse control. Consequently the effectiveness of Aripiprazole seems likely to be related impulsive nature of OCD and ADHD, in this case. Further clinical studies are needed to clarify the therapeutic effect of aripiprazole in children and adolescents with PIU.
Keywords: aripiprazole, attention deficit hyperactivity disorder, obsessive-compulsive disorder, pathological internet use

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[Abstract:0625][Psychopharmacology]

Priapism associated with sertraline and risperidone: a case report

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Priapism is an urological emergency defined as painful and persistent penile erection that is unrelated to sexual stimulation. Priapism can occur as a rare side effect of antipsychotic medications and the mechanism of the antipsychotic-induced priapism is thought to be related to blockade of alpha-1 adrenergic receptors. We present a case, who suffered from priapism during treatment with risperidone and sertraline, and the treatment decisions that followed.
Case: A 40 year-old male patient presented to our emergency service suffering from a persistent and painful penile erection, with a

sudden onset while sleeping and continuing nearly 76-hours. There was no history of penile, genital, or pelvic trauma; and there was no evidence of any infection or malignancy. Routine laboratory tests were performed that included complete blood count, basic metabolic profile, and a coagulation study, and all the results were within normal limits. When investigated, it was understood that he has been referred to an psychiatrist with depressive symptoms 2 months ago. He has been diagnosed as depressive disorder and risperidone 1 mg/day at night and sertraline 50 mg/day has been started. There was no previous history of psychiatric admission, hospitalization or suicide attempt. No significant characteristic finding could be ascertained in his medical history. He was hospitalized to urology inpatient clinic with the diagnosis of priapism. In the preoperative period, the patient was consulted to our psychiatry clinic. In the psychiatric examination his depressive symptoms was in remission, so risperidone and sertraline treatment was stopped. In the context of priapism in duration exceeding 72 hours, where complete erectile dysfunction is likely to ensure, the patient was treated with penile prosthesis implantation.

Priapism is a sustained penile erection lasting more than 4 hours in the absence of sexual activity. Prompt treatment for priapism is usually needed to prevent tissue damage that could result in the inability to get or maintain an erection (erectile dysfunction). The treatment of priapism includes conservative management (observation, ice, and rest), corporal aspiration, intracavernous injection of sympathomimetic agents, and, if the above treatments fail, surgical intervention. In the literature, 15-26% of all reported cases of priapism were associated with the use of antipsychotic medications. Various psychoactive medications are also known to cause priapism, with trazodone the most commonly implicated member of this group. There are some reports of selective serotonin reuptake inhibitors (e.g. Fluoxetine and Citalopram) inducing priapism. Priapism may occur at any time during the treatment course of psychotropic medications. It could be considered an idiosyncratic reaction, because it is correlated neither with the dose nor duration of psychotropic drug use. Clinicians should monitor patients on these medications for this rare, yet significant side effect. Furthermore, caution must be used when adding new drugs to the regimen and patients should be closely monitored for this side effect. And also, patients should be educated on the possible risk of priapism, including future episodes, and to refer to the hospital emergency when symptoms develop.

Keywords: priapism, risperidone, sertraline

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[Abstract:0629][Motor disorders]

Oculogyric crisis with clozapine: a case report

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Oculogyric crisis (OGC) is a dystonic and distressing side effect, which occurs immediately after the administration of high-potency antipsychotic drugs, and is usually reported as a subtype of dystonia. Tardive type of OGC is a dystonic syndrome starting after long-term use of dopamine receptor antagonists. Atypical antipsychotics have reduced liability for inducing tardive dystonia and show anti-dystonic properties in patients with pre-existing tardive dystonia. Clozapine is an atypical antipsychotic drug, and there have been case reports that it may be an effective treatment for tardive dystonia. Surprisingly, we found that one of our patients appeared to develop tardive OGC while taking clozapine. The relationship between tardive OGC and clozapine is unknown, however, it is possible that the previous antipsychotic exposure could have created a sensitizing or priming effect on the striatum. Also, there are some suggestions of an underlying susceptibility and possibly a genetic predisposition, at least in some patients. Here, we report a case of a young man with schizophrenia who manifested tardive OGC related to clozapine.

Case: A 25 year-old male patient, single, with 11 years of education, presented to our clinic with 7 years history of continuous illness, characterized by academic decline, reduced social interaction and functioning, irritability, somatic delusions and delusions of reference, which were evolved over a 5 years period. Mental status examination confirmed these findings and schizophrenia diagnosis was made according to DSM-5. He had used different medications including risperidone, amisulpride, olanzapine, ziprasidone, haloperidole, with symptoms not changed but OCG had also been seen in the past with antipsychotic medication. We began clozapine treatment and after 100 mg/day OGC started to occur 2-3 times per week. As the psychotic symptoms continued, clozapine dosage was gradually increased. After a 1 year period OCG decreased to 1-3 times per month. Clozapine treatment continued due to resistant nature of schizophrenia symptoms, with added ECT treatment. Currently, the patient is on clozapine treatment of 900mg/day and OGC did not occur for the past 6 months.

We report a patient with schizophrenia who developed tardive OGC during ongoing clozapine treatment. The treatment of tardive dystonia is particularly difficult, with variable results from several pharmacologic and other somatic interventions described in the

literature. In this report, we describe a patient with schizophrenia who developed episodes of ocular dystonia as a delayed adverse effect of clozapine. This can be explained in two ways. The first explanation is that some patients are more susceptible to extrapyramidal signs. The second, on the other hand, is the sensitizing or priming effect created on these patients' striatum due to the use of typical antipsychotic (haloperidole) medication prior to clozapine. However, cases of tardive dyskinesia also exist, in which the patients had never been exposed to typical anti-psychotics and were treated exclusively with atypical antipsychotics.

Keywords: schizophrenia, oculogyric, clozapine

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[Abstract:0630][Schizophrenia and other psychotic disorders]

Post-ictal psychosis: a case report

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Epilepsy is one of the main causes of functional disability, and it is usually comorbid to psychiatric conditions such as psychosis of epilepsy (POE). POE requires more careful pharmacological treatment, considering the propensity of antipsychotics to provoke seizures and the risk of pharmacokinetic interaction with anti-epileptic drugs (AEDs). POEs are classified according to the temporal relationship of ictal events in ictal, post-ictal and inter-ictal psychoses. Post-ictal psychosis (PIP) comprise near 25% of all POEs. Generally, PIP appears after an increase in the frequency of epileptic seizures. Symptoms are variable, involving auditory, visual, or tactile hallucinations,sexual indiscretions,and persecutory, religious or grandiose delusions, also irritability, aggressiveness and depression.

Case: A 31 year-old single male patient with 11 years of standard state education, presented to our clinic with psychotic symptoms. The patient had been treated for epilepsy by neurology department since 1994. His psychotic symptoms had an onset 10 years after epileptic seizures, and antipsychotic treatment had been added over his already continuant-epileptic drugs. He was on multiple anti-epileptic drugs (AEDs) and antipsychotics with a drug regimen of sodium valproate, lamotrigine, oxcarbazepine, levetiracetam, risperidone, amisulpride, aripipirazole, haloperidol, and clozapine. He was consulted to psychiatry department from neurology department and antipsychotic drugs were reduced for resistant seizures. He presented to our clinic after his epilepsy was examined over EEG video monitorization in neurology department. His EEG showed multifocal spike and wave abnormalities and he was clinically classified to have epilepsy with non-convulsive status, 12-20 times per week and sometimes 4-5 times per day. His psychotic symptoms had (1) delusions of reference with occasional presentation of verbal and physical aggression, and even (2) tendencies of violence after non-convulsive seizures for 6-8 hours to 1 day. The patient had no usage of alcohol. There was no positive family history of epilepsy and psychosis. Spanning a 2 years period, his antipsychotic medication regime was properly reduced and changed to only haloperidol 10mg/d, with his antiepileptic usage regulated by neurology clinic. His seizures currently were reduced to 1-2 times per week (no seizures in some weeks), lasting a short period (minutes to 1-hour) and there was no aggression and violence for four mounts.

PIP is most commonly seen in patients with longstanding pharmaco-resistant epilepsy and typically occurs after a cluster of convulsive or complex partial seizures. After resolution of the immediate postictal state, there typically is a lucid interval of up to 72 hours followed by the onset of psychosis, which typically lasts less than a week and rarely longer than two. Paranoia and grandiose or religious delusions are common. EEG should strongly be considered to evaluate non-convulsive seizures. Haloperidol is one of the safest antipsychotics to treat psychosis of epilepsy, as it hardly decreases the epileptogenic threshold. With haloperidol, it appears to have a lower epileptogenic potential, and may be one of the antipsychotic drugs of choice in epilepsy.

Keywords: post-ictal, psychosis, epilepsy, haloperidol

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[Abstract:0631][Mood disorders]

Treatment resistant depression and total occlusion of internal carotid artery: a case report

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Treatment resistant depression is described as poor response to two trials with anti depressants from different pharmacologic classes which are adequate in dose, duration and compliance. Previous studies have shown a relationship between carotid artery stenosis and severe depressive symptoms. We present a case with treatment resistant depression with total internal carotid artery occlusion.
Case: A 60 year-old male patient, married, high school graduate, retired, male patient was diagnosed as major depressive disorder undergoing antidepressant treatment for the last three years. He has previously prescribed paroxetine, sertraline, venlafaxine, duloxetine, trazodone, quetiapine, aripiprazole and olanzapine in adequate doses. He has received electroconvulsive therapy two years ago. Poor response to these medications was reported in his medical records. He was admitted to our psychiatry inpatient clinic with complaints of anhedonia, loss of interest, depressive mood, insomnia, fatigue, loss of appetite, psychomotor retardation and suicidal ideas. In order to exclude organic causes, cranial MRI was performed and hyperintense gliotic lesions were detected in the left centrum semiovale area. Total occlusion of the left internal carotid artery was diagnosed by carotid doppler ultrasonography. He was referred to cardiovascular surgery clinic and left carotid-subclavian bypass was performed. At two month follow up, patient reported resolution of depressive symptoms.
It was seen in this case that occlusion of internal carotid artery may present as depression without any neurologic symptoms. Depressive disorders could be linked to degenerative processes because of cerebrovascular disease, described as vascular depression. It is known that depressive symptoms are more severe in elderly patients with carotid stenosis. We suggest that when a patient with severe depressive symptoms and poor response to treatment was evaluated, psychiatrists should be aware of the possibility of carotid artery occlusive diseases.
Keywords: carotid artery, depression, resistance

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[Abstract:0632][Psychopharmacology]

Like twenties at seventy year-old: a case with 'spontaneus' orgasm

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Olanzapine has been used to treat depressive disorders with psychotic symptoms (1). Olanzapine is known that decreases sexual drive, we reported a case with unexpected effect as spontaneous orgasm.
Case: A 70 year-old female patient was admitted to psychiatry inpatient clinic with complaining of anxiety, depressed, fear, screaming, thinking not to have any heart beating and hearing some voices like his dead husband said 'come to me' for eight months. In her mental examination: self-care is reduced; persecution, nihilistic and hypochondriac delusions are detected. Mood was anxious and depressed. Auditory hallucination is described. In history, after two years her husband died, she was diagnosed generalize anxiety disorder and citalopram were prescribed for eight years. One year ago, she stopped to take citalopram and her anxiety complaints increased and also had hypochondriac thoughts. Then she has had feeling guilty to make herself ill, felt depressed and anxious and had delusions. After hospitalized, treatment was ordered as olanzapine 10 mg and lorazepam 3 mg per/day. Her blood test was completely normal. Dementia diagnosis was excluded by neuropsychiatric evaluation. Hamilton depression inventory was 23 point. She was diagnosed as psychotic depression. After ten days, lorazepam was stopped that she had no anxiety. At the twenty second day in her olanzapine treatment, she said 'I have spasms in my genital area like when I was in my twenties'. She had never experienced in the past and had this complaint for twice or thrice in a day for five days. After olanzapine dose reduction to 5 mg/per day, spontaneous orgasms disappeared. One month later she discharged with olanzapine treatment 5 mg/day with nihilistic thoughts and partial depressed mood. She had not that complaint again.

Spontaneous orgasm without sexual arousal is a rare condition which has been described with zuclopentixol, trifluoperazine, thiothixene and ziprasidone (2). Sexual function is associated with serotonin and dopamine neurotransmitters (3). Antipsychotic drugs are commonly known that could made sexual dysfunction because of dopamine blockage effects. Also activation of serotonin receptors by SSRI could make sexual dysfunction that could reverse with serotonin receptor blockage e.g. mirtazapine. Olanzapine has serotonin blockage effect and this blockage could increase in dopamine activity in the mesocortical dopamine pathway which might potentially be associated with spontaneous orgasm in this case.

Keywords: olanzapine, spontaneus orgasm, elderly, depression

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[Abstract:0633][Psychopharmacology]

Dry eye related with escitalopram and treatment with agomelatine

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Dry eye syndrome (DES) is a common problem of the adult population worldwide. Patients typically suffer from complaints that include eye irritation, stinging, dryness, ocular fatigue, and fluctuating visual disturbances. This case report presents a patient who was being treated with escitalopram whose medication was changed to agomelatine because of DES.

Case: A 32 year-old female presented to my outpatient clinic with complaints of malaise, anorexia, insomnia, weakness, reluctance, loneliness, weight loss and anhedonia for two years. She had lost her job in the previoustwo months prior to being consulted by a psychiatrist. She was diagnosed with major depressive disorder (MDD) according to the DSM-5. Escitalopram 10 mg/day was initiated. In the second month of follow up, her psychiatric symptoms improved; however, she developed discomfort and irritability in both eyesthat gradually increased following the initiation of treatment. The patient, who hadno history of arthralgia or dry mouth, requested consultation withthedepartments of internal medicine and ophthalmology. The physical examination was normal. Her laboratory work for the following was within normal limits: C-reactive protein, creatinine kinase, vitamins, thyroid, renal and liver function indicators, blood count, ESR, antinuclear antibodies, Anti-Sjögren's-syndrome-related antigen A and rheumatoid factor. Sjogren's Syndrome was not suspected but an investigation was carried out; the Schirmer test determined moderate dry eye (7 mm wetting of the paper after 5 minutes). Considering the patient's condition, the physical and the laboratory examination, DES was thought to be caused by escitalopram. The escitalopram was stopped and agomelatine 25 mg/day was initiated. The discomfort in her tear film and ocular surface was ameliorated within one week and the Schirmer test evaluated 16 mm wetting of the paper after 5 minutes. In the follow up period, her depressive symptoms improved.

The prevalence of DES is estimated at 11–17% in the general population. Age, female gender, dehydration, conjunctivitis, vitamin A deficiency, systemic collagen diseases, meibomian gland dysfunction are considered etiologies for DES. Also, many medications that have anticholinergic affects, such as antipsychotics, antidepressants, antihistaminergics, and anti-Parkinsonian drugs, along with medications for acne and hypertension, may cause DES. Selective serotonin reuptake inhibitors (SSRIs) have anticholinergic side effects that are fewer than the tricyclic antidepressants. Additionally, it is suggested that escitalopram exhibits superior efficacy and tolerability than the other antidepressants. However, in a previous case, ophthalmological pathology was reported with escitalopram. Additionally, the Schirmer test of a patient being treated with an SSRI demonstrated lower results, and the investigators interpreted that DES caused by SSRIs may be associated with another mechanism other than the anticholinergic system. Agomelatine, an agonist of melatonin receptors and an antagonist of the 5-HT2C-receptor, is safe, well-tolerated and esteemed antidepressant. The amelioration of the symptoms of intraocular pressure and glaucoma with agomelatine was demonstrated via animal and human studies. In conclusion, physicians should be aware of DES related with SSRIs and agomelatine may be a good alternative for patients with depression in this condition. This observation needs to be supported with further studies, whereby the mechanism of this association could be explained.

Keywords: agomelatine, dry eye, escitalopram

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[Abstract:0634][Schizophrenia and other psychotic disorders]

Secondary vaginismus due to delusional disorder

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Sexual medicine healthcare professionals, who do not normally examine men and women with psychiatric disorders, need to be aware that those with psychiatric disorders can and do present with sexual medicine problems. Paranoid psychiatric individuals may present with vaginismus rarely. Delusional disorder with vaginismus symptoms may be encountered team effort with sexual medicine specialist and psychiatrist. We aim to report a case of a female patient with delusional disorder who had diagnosed with secondary vaginismus before.

Case: A 41 year-old female patient, teacher, married presented to our psychiatry outpatient clinic with sexual dysfunction. She reported of tightness of vagina and introital pain while attempting sex with her husband. These symptoms were present for 18 months. She had attended to gynecologist several times previously. But she had consulted to a psychiatrist because of nonresponsive to treatment. The patient had diagnosed with secondary vaginismus and antidepressant treatment was initiated by a psychiatrist. Her symptoms intensified after antidepressant treatment. After that, the patient attended to our clinic with sexual dysfunction. Her sexual dysfunction was started 18 months ago but before that she had been skeptical about her colleagues. These thoughts made difficulties in her job. Psychometric evaluation reported as psychosis. Her mental examination revealed that defensive behavior, dysphoric affect, paranoid thoughts, delusional thinking, difficulties in day-to-day performance on the job and in social situations. The patient considered as delusional disorder and her sexual dysfunction was considered as secondary to her psychosis. Her antidepressant treatment stopped and antipsychotic treatment started. The delusional symptoms of patient minimized.

In conclusion, sexual medicine healthcare providers should be prepared to manage sex health concerns of men and women with delusional disorder, in conjunction with a psychiatrist. The detailed history of patients with sexual dysfunction should be taken carefully.

Keywords: delusional disorder, sexual medicine, vaginismus

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[Abstract:0636][Psychopharmacology]

Acute dyskinetic reaction with methylphenidate overdose

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Acute dyskinetic reactions are a complication of medications that alter dopamine signaling. Most reactions occur following exposure to agents that block dopamine receptors (e.g., neuroleptics). However, agents that increase dopaminergic transmission (such as methylphenidate) can also trigger acute dyskinesias. In this case we report a 15 year-old-girl with mild mental retardation and ADHD presented with orofacial and upper extremity dyskinesia as a result of high dose intake.

Case: A 15 year-old girl who presented to our outpatient clinic one year ago with attention deficit, irritability and problems in intellectual functioning. After psychiatric examination and psychometric testing she was diagnosed with ADHD and mild mental retardation as per DSM-IV. We prescribed 36 mg osmotic release oral system (OROS) methylphenidate for ADHD.. During about a year of treatment, significant improvement was observed in the majority of ADHD complains. But on an exam day at school, she decided to take more pills in order to get a higher grades She had used a total of 108 mg drugs in 3 hours. Four hours later, she developed lip-licking, lip-smacking and tongue-rolling movements suddenly. Continuous dyskinetic tongue movements inside and outside the mouth and involuntary bilateral arm swinging were noted. She had opened and closed her fingers without complete extension. Her vital signs and general and neurological exams were normal with the exception of her just dyskinesias. After 24 hours observation and supportive treatment in Emergency Department abnormal movements ceased.

Dystonic reactions (i.e., dyskinesias) are characterized by intermittent spasmodic or sustained involuntary contractions of muscles in the face, neck, trunk, pelvis, extremities, and even the larynx. Symptoms may begin immediately or can be delayed hours to days. Although dystonic reactions are rarely life threatening, the adverse effects often cause distress for patients and families. Medical treatment is usually effective to abate acute symptoms. With treatment, motor disturbances resolve within minutes, but they can reoccur over subsequent days. Clinicians should consider this side effect particulary in children with comorbid mental retardation.

Keywords: methylphenidate, overdose, dyskinesia, mental retardation

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[Abstract:0637][Psychopharmacology]

Paliperidone palmitate and valproic acid combination in the treatment of bipolar affective disorder

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Bipolar disorder (BD) is a potentially disabling illness characterized by episodes of mania, depression, hypomania or mixed states. Pharmacologic treatment of BD is based on administration of mood stabilizers and second-generation antipsychotics. Patient adherence to a treatment is very important. Many patients experience recurrence or relapse of mood episodes due to nonadherence to the treatment. Several antipsychotic trials suggested that long action injection antipsychotics (LAI) are effective in reducing relapse in BD. Paliperidone palimitate (PP) is a second-generation LAI antipsychotic agent. Because of the treatment nonadherence and the lack of treatment response, we added PP to the current medication in these two cases.

Case 1: 45 year-old female patient had 5 manic and 2 depressive episodes until the last admission to our department. One of the manic episodes were induced by leaving the medication. When she presented to our hospital, she had discontinued medications unilaterally and hospitalized with 6. manic episode. Valproic acid (VPA) treatment was started and increased to 1500 mg/day (91.25 µg/L). Because of the nonadherence to the treatments for many times and inadequate treatment response, we added PP to the treatment on the 11th day of hospitalization. Before this alteration, the Young Mania (YM) score of the patient was 22 and VPA concentration was 91.25 µg/L. After 53 days of hospitalization, her YM score was decreased from 33 to 7. During the treatment, extrapyramidal system(EPS) side effects weren't observed and her plasma prolactin level was 97.75 µg/L at discharge. She was discharged with VPA 1000 mg/day (93.87 µg/L) and PP 100 mg monthly injection with remission.

Case 2: 53 year-old female patient who had one depressive and two manic episodes until admission to our inpatient unit and one of her manic episodes was induced with treatment discontinuation. When she presented to our clinic, she has again stopped the medication, and was hospitalized with 3. manic episode. Because of the prior benefit, VPA treatment was started and increased to 1500 mg/day. Because of the treatment nonadherence in the past and inadequate response in this follow-up PP was added to the treatment on the 28. of hospitalization. Before this alteration her YM score was 14 and VPA concentration was 64.16 µg/L. After 82 days of hospitalization, her YM score was decreased from 39 to 0. During the treatment, no EPS side effect was observed and her plasma prolactin level was 46.34 µg/L at discharge. She was discharged with VPA 2000 mg/day (76.09 µg/L) and PP 100 mg monthly injection with remission and full insight.

Until now data about the use of LAI atypical antipsychotic are very limited. Combinations of mood stabilizers with atypical antipsychotics can be taken into consideration in case of psychotic or severe forms of BD. However, combined treatments may be a problem in the case of poor-adherent patients, and LAI formulations of atypical antipsychotics can be a therapeutic option in these situations. PP therefore may be a therapeutic option in BD which has severe symptoms, psychotic features, and poor adherence to pharmacological treatment.

Keywords: bipolar disorder, long action injection antipsychotic, paliperidone palmitate

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[Abstract:0639][Psychopharmacology]

Lithium intoxication: a possible interaction with moxifloxacin

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Due to being used for more than a half-century, lithium is a well-known and established treatment for cases with Mood disorders such as bipolar disorders and recurrent unipolar depression. As lithium has a narrow therapeutic index, concentration must be kept within critical limits; especially in the elderly. Lithium intoxication may be lethal, and it should be observed carefully. We aimed to describe a possible interaction between moxifloxacin and lithium on an elderly patient.
Case: A-74 year-old female patient with a five year history of bipolar disorderpresented to the emergency service with complaints of severe tremor on both hands, myoclonic jerks on upper extremity, feeling exhausted and blurred speech. She was using lithium carbonate (600 mg/day), haloperidol (10 mg/day), risperidone (2 mg/day) and sertraline (50 mg/day). She did not have fever. Her blood pressure was 110/70 mmHg. Her complaints began up just the day after she had started using moxifloxacin 800 mg/day and serum lithium concentration had estimated as 0.8 mEq/L ten days before admission. She had neither vomiting or diarrhea nor nystagmus or dysarthria. On her psychiatric examination, she was slightly drowsy and oriented. Her speech was blurred. Her serum lithium concentration was estimated as 1.7 mEq/L (0.5-1mEq/L is normal range in laboratory). Other laboratory tests in the emergency unit showed no renal impairment, no sign of dehydration. Neither her cranial magnetic resonance imaging nor electrocardiography showed any abnormalities. She was diagnosed with the mild-moderate chronic toxicity of lithium. Lithium and moxifloxacin were stopped immediately; she was administrated fluid resuscitation with more than 2 liters of 0.9% saline solution for 6 hours. Serum lithium concentration decreased to 0,9 mEq/L 12 hours later and tremor, myoclonic jerks distinguished. On following two days, her drowsiness continued and she was disoriented. These remained neurological signs resolved one week later.
Lithium toxicity is life-threatening situation especially in elderly patients due to having altered pharmacokinetics, renal impairment, and multiple drug use. In elderly patients, the incidence of lithium toxicity is 1.5% per year. Lithium toxicity can be life threatening and the medications that may induce lithium toxicity are among the most common used medications in the elderly. Moxifloxacin is known to be safe and well-tolerated fluoroquinolone. Levofloxacin, another fluoroquinolone agent, was shown to increase the serum lithium levels in rabbits. To best of our knowledge, this is the first case report in the literature pointing out a possible interaction between moxifloxacin and lithium. Considering possible drug interactions on elderly patients receiving lithium is essential.
Keywords: moxifloxacin, lithium, interaction, elderly

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[Abstract:0640][Schizophrenia and other psychotic disorders]

Hypercalcemia related psychosis disorder: a case report

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Primary hyperparathyroidism is characterized by parathyroid gland pathology as decrease of the blood PTH levels, hypercalcaemia and hypophosphatemia. Cause of the hyperparathyroidism is mostly according to parathyroid gland adenoma that is usually asymptomatic but some symptoms such as nausea, stress, peripheral neuromuscular complains, nephrolitiasis and neuropsychiatric symptoms can be observed. Depression, anxiety, paranoid psychosis, delirium and stupor can also appear as neuropsychiatric disorders. In some reports, it has been noted that half of the patient have psychiatric symptoms. In our case, we will mention about the parathyroid adenoma related paranoid psychosis.
Case: A 45 year-old female patient, married. Patient did not have any psychiatric diagnosis or treatment. She had no family history of psychiatric disease. Her complaints first began 5 years ago. It was noted that she felt suspicious of her neighbors, that they were watching her and she was extremely jealous of her husband. Sometimes she presents aggressive, hostile behavior. She appeared healthy, looking sceptical, oriented. Her general condition was optimal, cooperation was limited due to her defensive attitude. Perceptual deviation was

not described. Her mood was irritable. Her affect was angry. Association of ideas was disregular. Her responses to the questions were inaccurate. It was also noted that her thought contents were composed of paranoid and persecutorydelusions. She had no insight on her symptoms. Her judgment and reality testing was impaired. Her first BPRS score was 48. Her treatment was ordered with risperidone 6 mg/per day. In laboratuary test detected high levels of calcium and parathyroid hormone (PTH), low levels D vitamins in her blood (Ca: 11.8 mg/dl (8.8-10.6 mg/dl), PTH: 945.9pg/ml (12-88 pg/ml), D vitamin=4.32 (ng/ml)). She was taken neck ultrasounds and parathyroid scintigraphy. Parathyroid adenoma was detected in the left, inferior lobe of the parathyroid. After 20 days of daily antipsychotic administration, her BPRS declined to 37. She was transferred to the otorhinolaringology for her parathyroidectomy. In her post-operation observations, her blood calcium and PTH level was normal. Her BPRS score declined from 37 to 20 within 18 days. The patient's paranoid and persecutorydelusions decreased significantly and her disorganizated speech got better. After 4 months in her psychiatric examination was not detected any pathology, and decided to discontinue for antipsychotic treatment.
In the current literature, psychotic disorders can be related to general medical condition. In ours, due to the detected of high level of calcium and parathyroid hormones. Psychotic symptoms was improve after her calcium level optimizations during her post-op period, it is concluded that the chart developed from hypercalcaemia related to hyperparathyroidism. In the literature, without the treating the essential medical condition, psychotic disorders will not improve. However, there is no information regarding the exact length of the antipsychotic treatments. The conclusion to be made from this case, it should be remembered that psychotic disorders can be related to general medical conditions. The information of blood calcium levels of the patient is crucial for diagnosis and determining the best course of treatment.
Keywords: hyperparathyrodism, psychosis, hypercalceamia

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[Abstract:0641][Mood disorders]

Delirious mania-induced with using of herbal weight loss supplement adulterated with sibutramine

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Worldwide herbal medicines are gaining popularity as a source of complementary and alternative remedies. However adulteration of herbal remedies with undeclared synthetic drugs is a common problem, which may potentially cause serious adverse effects.Sibutramine is one of the most occurring adulterants encountered in dietary supplements with slimming indication.Sibutramine is a synthetic noradrenaline, dopamine, and serotonin reuptake inhibitor used for the treatment of obesity. Although it was later inhibited from most markets following a series of reported adverse events including death, it is still being sold as “weight loss supplement” via websites and many of those products have been shown to contain far higher concentrations of sibutramine than that which was prescribed. Beside cardiovascular and fatal side effects, sibutramine has been associated with many neuropsychiatric side effects including hypomanic and manic episodes. We presented a case who had hypomanic episode and delirious mania induced with using recurrent weight loss supplement containing sibutramine.
Case: A 50 year-old female patient presented to the emergency service of our hospital with complaints of inappropriate laughing and talking, not recognizing of her relatives, inappropriate behaviors such as disrobing and getting out on the balcony naked for two days. In the emergency room, she was awake, but unwilling to speak. Either she did not answer the questions or answered with short and absurd words. Sometimes psychiatric interview was interrupting with inappropriate laughing and she was looking directly and fixedly to the ceiling. Vital signs, blood tests, computed tomography and diffusion magnetic resonance imaging of the brain was normal and she consulted to department of neurology. Because of disorientation and difficulty of cooperation, she was hospitalized to neurology service. EEG is done and no pathologic sign could be found. Neurological pathology was eliminated. During hospitalization in neurology service it was understood that Haloperidol, Biperiden, Chlorpromazine intramuscular injection was administered for once for her aggressive behaviors and two hours after this injection Quetiapine 100 mg tablet was given per orally. The patient was consulted daily and no another medication was advised. Within two days all of the pathological psychiatric symptoms and signs was improved. In the 4th day of her hospitalization she was transferred to psychiatry service for differential diognosis and observation. When her psychiatric and medical history was detailed, it was learned from herself that she purchased a weight loss supplement adulterated with sibutramine from website 3 weeks ago. After using this supplement, complaints of euphoria, excessive talkativeness, sleepnessless, increase in self-confidence, acceleration of thoughts and increased energy had started. After these complaints, she discontinued the drug 4 days ago prior to admission. It has been learned that nearly one year ago, similar symptoms with less severity had emerged 3 weeks after using the same

product, and after discontinuation of the product, symptoms had recovered within a fewdays.
Health care professionals must be more careful about the sale of such products including sibutramine via telephone and websites, in respect to their side effects.
Keywords: sibutramine, delirious mania, weight loss supplement

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[Abstract:0642][Psychopharmacology]

A rare side effect of paroxetine: hallucination

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Primary indications of SSRIs are major depression, depression, panic attack, post-traumatic stress disorder, and dementia. Dizziness, sleep disorders, headache, drowsiness, increased perspiration, decreased libido, mouth dryness, constipation, diarrhea, ejaculation disorders, tremor, and weakness are the most frequent side effects. Tachycardia, chest pain, itching, nausea, vomiting, blurred vision, and hallucination are relatively rare side effects. In this case report, we wanted to present a geriatric patient with hallucination as a rare side effect of paroxetine.
Case: A 73 year-old female patient was referred to a tertiary care setting's Geriatrics clinic with complaints of dysmnesia, fatigue, and visual hallucinations described as ''her dead spouse was coming to home and sitting without speaking". The patient has been diagnosed with essential tremor, COPD, depression, and vertigo. Hallucinations were alive and organized. Current medications were Paroxetine (20 mg/day), Propranolol (40 mg/day), Budesonide/ Formoterol Fumarate (400/24 mcg/day), Tiotropium Bromur (18 mcg/day), and Betahistine (32 mg/day). In physical examination, vital signs were stable and there was not any other abnormal findings that were detected apart from essential tremor. Mini Mental Test Score: 24/30, barthel index of activities of daily living: 90/100, The Lawton Instrumental Activities of Daily Living Scale (IADL): 14/17, and Yesavage Geriatric Depression Scale: 8/15 were determined in neuropsychiatric evaluation. In laboratory evaluation, it was measured that fasting blood glucose: 86 mg/dl (65-107 mg/dL), urea: 26 mg/dL (15-44 mg/dl), creatinine: 0.71 mg/dL (0.81-1.40 mg/dl), AST: 38 U/L (10-40 U/l), ALT 24 U/L (10-40 U/l), sodium: 142 mmol/L(135-145 mmol/l), potassium: 4.62 mmol/L(3.5-5.5 mmol/L), calcium: 9.43 mg/dL (8.5-10.5 mg/dl), vitamin B12: 755 pg/mL (220-940 pg/ml), vitamin D3: 21 ng/mL, and folic acid: 11.1. For depressive symptoms, paroxetine treatment was initialized as 10 mg/day and the dose was gradually increased up to 20 mg/day. Then hallucinations started again. Paroxetine treatment was stopped and hallucinations were disappeared. When drug was re-started, the case reported the visual hallucinations again and the hallucinations were diagnosed as a side effect of paroxetine. Causal relationship between paroxetine and hallucinations was assessed according to Naranjo Criteria and this side effect was reported as "possible".
Paroxetine is one of the most potent SSRIs and it partially blocks norepinephrine reuptake. It is indicated in the diagnoses of Major Depressive Disorder and Obsessive Compulsive Disorder (OCD), Panic Disorder with or without agoraphobia, Social Anxiety Disorder known as Social Phobia, and General Anxiety Disorder (GAD). Moreover, it has been known that in long-term use it can prevent relapses and recurrences of recurrent depression. Metabolites of SSRIs are inactive, and their inhibition power is weak. In older patients, paroxetine must be started with the dose of 10 mg/day and then gradually increased up to 40 mg/day through one week-intervals. Discontinuation must also be gradually done. Because of SSRIs' potent anticholinergic efficacy, geriatric patients with a history of falling and/or fracture must be evaluated in detail before prescription. Physicians should be aware of psychotic adverse effects of paroxetine, keep in mind these side effects while prescribing and patient evaluation especially patients with first-episode psychosis.
Keywords: paroxetine, hallucinations, elderly patient

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[Abstract:0644][Mood disorders]

Bipolar disorder and wilson's disease: a case report

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Wilson's Disease is a rare, autosomal recessive disorder of copper metabolism and is associated with abnormal liver and brain functions. The accumulation of copper in the brain, especially in the basal ganglia, can cause the degeneration of neurons and thus leads to neuropsychiatric symptoms such as Mood disorders, personality disorders, psychosis and cognitive disorders.
Case: A 46 year-old female patient presented with bipolar disorder and had tremors on examination which were present since 20 years. She was hospitalized for diagnosis of manic episode. Besides she had psychotic symptoms. We consulted to neurology for postural and intentional tremor. She also reviewed by ophthalmologist who pointed out the suspicious Kayser-Fleicher ring on the right eye, but not on the left. Investigations revealed increased urinary copper (116 microgram/24 hours), low serum ceruloplasmin, (18.9 mg/dl), normal levels of serum copper (103.4 microgram/dl). The abdominal ultrasound examination of her was normal. The clinical diagnosis of Wilson's Disease was made by co-decision of neurology, internal medicine and psychiatry departments.
There are few reports and limited reports in the literature of patients with co-existence of Wilson's disease and bipolar disorder. Especially, when clinical manifestation of the patients started with psychiatric symptoms, it can cause a delay in the diagnosis of Wilson's Disease. In our case, the diagnosis of Wilson's disease was established 25 years after first diagnosis of bipolar disorder.
Keywords: bipolar disorder, Wilson's disease, tremor

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[Abstract:0645][Anxiety disorders]

Late-onset separation anxiety: a case report

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Separation anxiety is defined as excessive anxiety and fear of child for at least 4 weeks, due to a separation of child from home or from individuals whom he/she is wedded on, according to his/her level of development. It is seen that children have a continuous and excessive anxiety, regarding to a possible lose or a possible accident of individuals whom they are wedded on. In addition, they do not want to join daily activities, such as going to school, due to a separation fear. Individuals in separation anxiety experience some difficulties in functional settings which are significant for themselves (relations of school, peer and family). Frequency of separation anxiety in children is 4%. The frequence of the disorder is equal between females and males in childhood.
Case: A 17 year-old female patient had complaints continuing for 6 months such as reluctance to separate from her mother, inability to stay alone, unwillingness to stay alone outside, excessive fear of something bad happen to herself or her family due to her seperation from her mother, not to take a shower, unwillingness to take a shower, unwillingness to eat, crying all day long following to a separation from her mother. The case is in undergoing treatment at the hospital due to these complaints, approximately for 3 months. The complaints of the patient, who has been followed by psychopharmacological treatment, as well as behavioral methods, have decreased for the last month. The treatment of the case, who has been followed at the clinics, is still ongoing.
Cases of separation anxiety are encountered rarely in late adolescence. It may be a prodrome of other psychopathologies. In particular, separation anxiety of which onset is in late ages may interfere with prodromal period symptoms of schizophrenia and disorders leading to psychosis, with depressions disorders, bipolar disorder and various psychopathologies. Consequently, detailed examination of these patients is significant, in terms of accurate diagnosis and treatment options.
Keywords: separation anxiety, adolescence, psychopathology

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[Abstract:0646][OCD]

Childhood skin picking disorder: a case report

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Skin picking disorders is characterized by the repeated urges to pick at the patient's own skin, often to the extent that skin damage is caused, although he/she does not have any dermatological problem. Age of onset of the disorder shows a wide range of variability and is more common in girls. The incidence is reported between 3-5%. Long term outcome of psychogenic skin picking disorder is not known exactly. A girl who is peeling her own skin with her nails since 6 months-of-age will be discussed.
Case: A 2 year-old female patient was brought to child psychiatry outpatient clinic with complaints of peeling her own skin on her neck and hip regions with hands and plucking oral mucosa with her teeth. Her mother declared that her complaints began when she was a 6 months old baby, picking own skin has increased over time, she did not get any benefit from treatments she received in-patient and out-patient dermatology clinics. The mother is a housewife, her father is a worker in constructions and she is the smallest of the four brothers and all stages of development had occurred normal except for speech development. She can say several words but cannot set up sentences yet, she understands what is told and could obey commands. Our patient had no family history of any psychiatric disorder. It was noticed that she was handcuffed with a rope by her family during psychiatric examination in our out-patient clinic. On physical examination, there were 1x2 cm lesion on her right hip, 2x3 cm crusted lesion above the neck and 1x1 cm hyperemic lesion on the oral mucosa was noted. The patient was normal otherwise in her physical examination and laboratory tests. She was introduced risperidone solution 0.25 mg/day. The skin picking behavior was significantly diminished, and her skin and mucosal wounds had greatly improved in the follow-up and she is still on treatment.
Skin picking disorder was classified in the "impulse control disorders not otherwise specified" in DSM-IV-TR; while it has been introduced into DSM-5 under "Obsessive Compulsive Disorder and Related Disorders" category. Picking behavior gives satisfaction and reduces tension therefore contributes to reinforcing. SSRIs have been shown to be effective in the treatment of skin picking disorder. In addition to antidepressant drugs, some of the antipsychotics (pimozide, olanzapine, aripiprazole) have been reported to be beneficial in these patients. There is little data about risperidone use in the treatment of skin picking disorder in the literature. It is known that dopaminergic agonists aggravate symptoms in skin picking behavior. Thus, we began treatment with risperidone in order to reduce dopaminergic pathway. Our patient was too young that prohibits starting most of the antipsychotic drugs. There are several case reports and survey studies about skin picking behavior. Our case contributes to the literature in terms of early age of onset and well-respond to risperidone treatment in skin picking disorder. We think that our case would give an idea in the treatment of early onset impulse control disorders.
Keywords: skin picking disorder, childhood, antipsychiotic

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[Abstract:0647][Impulse control disorders]

Treatment of nail biting with n- acetyl cysteine: a case report

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Nail biting behavior is defined in ''body-focused repetitive behavior disorders'' subgroup of ''other specified obsessive-compulsive and related disorder'' at DSM-5. Its prevalence in children is 22.3% and there is no difference between genders. Nail biting behavior often causes recurrent paronychia and chronic subungual infections. N-Acetyl cysteine (NAC) is an antioxidant and also regulates glutamate transmission. Recently, a few research suggest that NAC is useful for impulse control disorders such as obsessive compulsive disorder, trichotillomania and nail biting. There is only one case report and one placebo controlled clinical trial related to treatment of NAC and nail biting in literature. In this case report, we presented a patient who had nail biting improved with NAC treatment in 6 months period.
Case: A 9 year-old male patient presented to child psychiatry clinic for treatment of nail biting with her mother. He was a 4th grade student at elementary school. He has had nail biting since age 3 years and nail biting was happening several times a day. He had no

periods of prolonged abstinence. He has also skin picking of the nail edges. There was not any triggering factor. Family history revealed that his sister also had nail biting. His family attempted to stop nail biting by using some ways like warning, giving him chewing gum and applying bitter nail polish but all of them failed. He was third child of his family. Development milestones were normal. There was no physical disorder. His mother was a housewife. His father was a worker. Parents were not related to each other. None of first or second degree relatives had any psychiatric disorders. Psychiatric examination showed that physical appearance was age-appropriate and his mood and affect was normal. The process of thinking and thought content was age-appropriate. There was no other sign of psychiatric disorder except nail biting. He was monitored monthly for 6 months. At all interventions, his mother was wanted to grade severity of nail biting from 0 to10. ''0'' point was defined as there was no nail biting behavior and ''10'' point was defined as nail biting was the most severe situation. The child was also supported by motivational interviews and behavioral therapeutic interventions such as no warming, filling his time, distracting him when he bited his nail. In the first intervention, nail biting severity score was rated as 8 point by his mother. Treatment with NAC was started as 600 mg/day and increased to 600 mg BID after one week. Nail biting severity score was rated as 4 point at the first month of intervention, 3 point at the second, 2 point at the third and 1 point at the fourth month. Nail biting behavior was not observed at the fifth and sixth months.
Studies about treatment of nail biting with NAC are very limited. Treatment response of our case supports this limited literature. NAC is useful for impulse control disorders by antioxidant effects. More controlled trials about treatment of nail biting with NAC must be done.
Keywords: n- acetyl cystein treatment, nail biting, grooming disorders

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[Abstract:0650][Schizophrenia and other psychotic disorders]

It is not my kidney: a case report

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Prevalence of end-stage renal disease has been increasing in our country as well as all over the world. End-stage renal disease is a general health problem because of its high prevalence, changing pattern of etiology, complex and expensive treatment modalities required. The most common causes of end-stage renal disease are diabetic nephropathy, vascular nephropathy, glomerulonephritis, polycystic kidney disease and interstitial nephritis. Treatments in patients with end-stage renal disease are hemodialysis, peritoneal dialysis or renal transplantation. Kidney transplantation is the best treatment option for end-stage renal disease as it gives patients the chance to return to a satisfactory quality of life. Today, compared with dialysis, kidney transplantation in patients with end-stage renal disease is widely accepted as a treatment method due to its advanced short and long-term survival benefits. However, transplantation and life-long immunosuppressant treatment have their own problems. Many psychiatric disorders such as depression, mania, anxiety disorder, adjustment disorder and psychotic disorder with cognitive and memory impairment can be seen in any postoperative period in renal transplantation patients.
Case: A 26 year old male patient, single had kidney transplantation surgery after being treated with dialysis for four years. He was taken to the emergency department by his family because of vomiting and weakness symptoms. He was transferred to the nephrology service from there. While he was being treated, his doctors consulted the psychiatry department as he refused to take his drugs. As a result, the patient with no previous psychiatric history presented to our clinic with suspected acute psychotic episode four years after renal transplantation surgery while he was getting immunosuppressive agents. The patient's mental status examination revealed mutism and negativism. He did not give response to the questions and make eye contact. According to information received from family members the patient had strange behaviors, religious preoccupation that may be caused by his delusions of persecution and reference. His communication with people decreased and he was socially isolated. The most important thing was that the patient had not taken his immunosuppressive drugs for the last month. His hematologic and biochemical profiles including renal function tests and urine examination were all within the normal ranges. Serum electrolytes were within the normal limits as well. Cranial magnetic resonance imaging and electroencephalography showed no abnormalities. After the examinations, a regimen of parenteral haloperidol 20 mg/day and biperiden 10 mg/day was started in his inpatient following because the patient refused to take oral medication. Two weeks later, the patient agreed to take the oral medications. The patient was discharged after 4 weeks of hospitalization with improved functionally and the absence of previous delusions.
Immunosuppressive agents are commonly used to prevent graft rejection, graft survival and for the extension of the patient's life

in transplant patients. These agents, especially calcineurin inhibitors and high-dose steroids, have been associated with a range of neurological side effects, including overt psychotic symptoms. Most antipsychotic drugs are metabolized in the liver and their dosage is not affected by renal function such as haloperidol have no significant interactions with either corticosteroids or calcineurin inhibitors.

Keywords: renal transplantation, immunosuppressive agents, psychosis

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[Abstract:0651][Substance-related and addictive disorders]

Chronic venous insufficiency in a man with a history of heroin injection: a case report

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Chronic venous insufficiency (CVI) describes a condition that affects the venous system of the lower extremities. Risk factors found to be associated with CVI include sex, age, obesity, pregnancy, a family history of varicose veins, phlebitis, and previous leg injury. Persons with a history of injection drug use have many risk factors for the development of CVI because of damage to the deep and supercial venous systems from repeated venous trauma, thrombophlebitis, and deep vein thrombosis (DVT). A few studies appear in the literature about complications to the lower extremities from intravascular and perivascular injections of abused substances and their effects. We report a case CVI in a person with a history of heroin injection.

Case: A 24 year-old male patient presented to our hospital with heroin use. The patient had heroin inhilation for 9 years and had injected heroin into the femoral vein for 4 years. Before psychiatric admission, he was not taking any medication. The initial examinations on admission his left leg was oedematous. It was learned that this edema has been for one year. There was significant increase in diameter in the left lower extremity. There was no history of prolonged standing and obesity in patient. There was no leg infections/ cellulitis and deep vein thrombosis. There was no tension and Homans test was negative. Routine blood test results of liver and kidney electrolytes, hemoglobinopathies and coagulopathies were normal. But Hepatitis C was positive. Vascular color Doppler ultrasound revealed vascular hemodynamic impairment in the left sepheno femoral junction. It was thought that patient's chronic venous insufficiency is due to intravenous heroin use.

Intravenous drug use more as a serious problem for today. But little is known about CVI in injection drug users. CVI clinical manifestations are common in persons who have used injected drugs, especially in the veins of the groin, legs, and feet. People tend not to think about their legs until they are injured or in pain, Persons with a history of injection drug use have many risk factors for the development of CVI because of damage to the deep and supercial venous systems from repeated venous trauma, thrombophlebitis, and DVT. Leg changes may not be viewed as important or serious in light of other complications of injection drug use such as HIV/ AIDS or hepatitis C. Yet the leg changes associated with CVI may hamper a person's mobility, quality of life, and employment. In this case, we suggested that the long-term injection of heroin into the femoral vein of the patient leads to CVI.

Keywords: chronic venous insufficiency, heroin injection, opiate

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[Abstract:0653][Others]

Severe catatonia in an adolescent: a case report

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Catatonia is a syndrome of motor dysregulation characterized by mutism, immobility, negativism, posturing, staring, rigidity, stereotypy, mannerisms, echophenomena, perseveration, and automatic obedience, among others. Catatonia can occur in the context of several

disorders, including neurodevelopmental, psychotic, bipolar, depressive disorders, and other medical conditions. We present a case with major depressive disorder and associated severe catatonic symptoms.
Case: A 15 year-old girl she was referred to pediatric emergency service by his mother due to mutism, negativism and slowness in her movements. Before admission to the hospital, she had not eaten and not talked for 3 days and she had developed incontinence of urine and feces for 2 weeks. Her weight dropped from the initial of 40 to 34 kg. Her admission weight was 34 kg, with a height of 160 cm (BMI 13.5). Her self-care had deteriorated significantly over the past 2 weeks, so that she would only shower once a week at the insistence of her parents. In the patient's history, she had symptoms of depressed mood, poor energy and concentration for 2 months. There was no documented history of physical, emotional, or sexual abuse. Family history is negative for any other psychiatric problems. On psychiatric examination of the patient, blunt mood, mutism, negativism, rigidity, and fixed posturing were observed. An in-depth physical including laboratory tests and an electroencephalography all indicated that she had no significant medical problems. She was diagnosed with catatonia, according to Diagnostic and Statistical Manual of Mental Disorders (DSM-5) criteria. She was transferred to our child and adolescent inpatient psychiatry unit. Her total score on the Bush-Francis Catatonia Rating Scale (BFCRS) was 27. She was given Lorazepam 3 mg/day. The catatonia in this case seemed associated with depression therefore we added fluoxetine 10 mg/day to the treatment and it was increased to 20 mg/day gradually. She responds to lorazepam partially within a week. We added olanzapine 10 mg/day to the treatment and it was increased to 20 mg/day gradually. Within two weeks she began to eat. Her movements increased and she started talking to the people around her. In the fourth week of hospitalization her total score on the BFCRS dropped to 20. She is still hospitalized in our inpatient unit.
Although catatonia has been previously associated with schizophrenia, today it is more associated with Mood disorders and general medical conditions. In our patient's history, there was no psychotic symptom and there were depressed mood, poor energy and concentration. Therefore this article reports a case of major depression with catatonic symptoms which occurred in an adolescent. During severe stages of catatonia, the individual may need careful supervision to avoid self-harm or harming others. There are potential risks from malnutrition, exhaustion, hyperpyrexia and self-inflicted injury. In our case there are significantly risks from malnutrition and exhaustion. In the literature, reports about the catatonia in child and adolescent psychiatry are limited. In conclusion, our case report aims to increase clinicians' awareness regarding the diagnosis and treatment of catatonia in adolescents.
Keywords: catatonia, depression, adolescent psychiatry

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[Abstract:0657][OCD]

The differential diagnosis of early onset schizophrenia and obsessive-compulsive disorder: a case report

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Obsessive-Compulsive Disorder (OCD) is a common neuropsychiatric disorder characterized by the presence of obsessions and/or compulsions. Lifetime prevelance of OCD is 1 to 3%. OCD symptoms start before puberty in approximately one third to one–half of sufferers. Early onset cases display greater severity and persistance of symptoms and may be less responsive treatment. Children are less likely to recognize their symptoms as ego dystonic, making them less willing to resist the urge to perform a compulsive behavior. Symptoms may not be insight against the OCD patients. In this situation the differential diagnosis of delusions may develop to be distinguished from obsessions. Here, the difficulties in the differential diagnosis of OCD in children with early-onset schizophrenia over a case was discussed.
Case: An 11 year-old girl, normal growth and grades, no medical history. When she was seven, she started to having repeatedly touching places, hearing some voice and did not want to defecate. When she has made the first application, has started to Olanzapine 20 mg/day and Risperidone 3 mg/day for 8 months, but has not a benefit from this treatment. Her self-destructive behaviors were in this period. It is gradually started on 50 mg Sertraline for treatment but has not clinical benefit on combination therapy. And then doctors cut olanzapin treatment, increase apipirazol to 7.5 mg/day, sertraline to 200 mg/day, risperdion to 4 mg/day. Using these drugs regularly for 5 months but she has not benefit from this treatment; conversaly, there has been increase in her current complaints. She was hospitalized for the purpose of further evaluation and observation. Patient observed with her current medical treatment and in sessions she said the voices were coming inside her head and try to lead her behaviors if she did not do it she thinks bad things will happen to her. During the

observations in service, there was not any psychosis symptoms, the communication she had with her friends was good and she could go to the toilet regularly. Thereupon the dosage given to she has been reduced. It has been observed that the treatment of the patient become better. Patient discharged with risperdion 2.5 mg/day, sertraline 75 mg/day, aripipirazole 7.5 mg/day and follow up sessions dosage will be decrease.

It is difficult to separate obsessional thoughts, delusional thinking and hallucination from each other in some clinical cases. Delusional thoughts in schizophrenia are mostly absurd, stereotyped and egosintonik but the obsessive thoughts in OCD are egodystonic. When the distinction between hallucinations and obsessive thoughts is considered,the patients in OCD describe these thoughts as the voice coming from in their heads. But, especially for small children, it is difficult to make this distinction. The lack of insight in OCD is one of the reasons that makes our clinical diagnosis difficult. The negative and cognitive symptoms in schizophrenia are not found in OCD. In our case the patient had some obsessive thoughts (egodystonic) that bother her and had some compulsive behaviors because of the fear that something bad will happen to her. She had no negative or cognitive symptoms. The poor insight of our patient made clinical diagnosis difficult. Serious obsessions can be confused with psychotic disorders under extreme stress. Detailed medical examination(s) and if possible observing the patient for while may be required to get the correct clinical distinction.

Keywords: obsessive-compulsive disorder, early onset schizophrenia, differential diagnosis

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[Abstract:0659][Psychopharmacology]

Tenofovir -induced the comorbidity of fanconi syndrome and depression in an older adult

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Renal function abnormalities caused by tenofovir can be seen in patients with chronic Hepatitis B. Fanconi syndrome that is one of these side effects has been rarely reported. We presented here a case of fanconi syndrome and its related depression due to tenofovir in an older adult.

Case: A 73 year-old male patient was admittted to an geriatric outpatient clinic with complaint of difficulty in walking, weakness, and muscle cramps, fatigue, and drowsiness which were lasting for last three years. The patient has being treated for hepatitis B for approximately 10 years. Treatment of tenofovir had been started seven years ago because development of resistance against initially administered lamivudine. The case was not taking any medication other than tenofovir and herbal remedy. There was no history of alcohol or smoking. The family history was unremarkable. In physical examination, arterial blood pressure was 135/80 mmHg, pulse was 55 beat/min, body mass index was 25.3 kg/m^2 and weakness in in the left proximal muscles of leg was detected. it was found out that proteinuria was appeared for the first time at follow-up visits after four years from the start of tenofovir (123 mg and 1626 mg in 24-hour urine collections, respectively). His score on Yesavage Geriatric Depression Scale was 12 (range 0–15, with scores greater than 11 indicative of depression). Bone mineral density was consistent with osteoporosis in lumbar vertebrates and osteopenia in the femur. Fanconi syndrome and its related depression were thought caused by treatment of tenofovir. Tenofovir was discontinued and replacement of oral vitamin D with dosage of 10 drops/day for vitamin D deficiency was started. After three months; phosphorous and uric acid increased to 1.87 mg/dl and 2.06 mg/dl, respectively. In urinanalysis, proteinuria (+) and glucosuria (++) were persistent. Complaints of weakness, and difficulty in walking of the patient were partially decreased. After six months from the last visit, it was informed that tenofovir was started again to patient by gastroenterology. The patient showed again increases in complaints of difficulty in walking, weakness, muscle cramps, fatigue, and drowsiness.

Tenofovir is an analogous of adenosine nucleoside phosphonate used in treatment of HIV and chronic hepatitis B. Causal relationship between tenofovir and fanconi syndrome was assessed with Naranjo criteria and rated as probable (6 points). While mechanism of Fanconi syndrome is not very clear, it can occur due to renal proximal tubuler dysfunction which results in impairment of reabsorption of bicarbonate, phosphate, glucose, uric acid, and amino acids. In this case, renal proximal tubular dysfunction was tought to be developed after tenofovir use and resulted in hypophosphatemia, hypouricemia, glucosuria, proteinuria, and osteomalacia. To the best of our knowledge, this is the first case of fanconi syndrome and depression induced by tenofovir in an older adult with hepatitis B. Close medical follow-up of kidney function and psychological status are required during tenofovir treatment, especially in older adults at increased risk for metabolic abnormalities.

Keywords: tenofovir, fanconi syndrome, depression, elderly

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[Abstract:0662][Mood disorders]

Multiple sclerosis or bipolar disorder?

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Mental or psychological changes in multiple sclerosis(MS) include pathological laughing and crying episodes, euphoria, mania, depression, anxiety, psychosis and personality changes.The clinical presentation and progression of the disease showing differences according to affected regions of the brain. In this case report, difficulties about diagnosis and treatment of MS patients beacuse of their psychological presentations named a psychiatric disorder will be discussed.

Case: A 23 year-old female patient, a university student, has being followed for bipolar disorder (BPD) and MS for 4 years. In previous MS attacks, she had also psychotic symptoms. She presented to the emergency service because of drinking bleech, her thought was she might been poisoned from chicken. According to information, we learned that she had been fluctuating throughout the day, had meaningless speeches, laughing and crying episodes, the idea of poisoning and someone intended to harm her, insomnia, for a week. It was learned that her mother had treatment resistant BD. In the first interview, she had seductive behaviors which suddenly appeared and dropped, inappropriate laughing episodes followed by intense crying episodes, was talking pressured. There were short-term amnesia attacks. In her thought content persecutory thoughts and suspicion took attention. She asked to turn off all the electronic devices in the room at the beginning of interview. She told that she could get influenced by the radiation. Her psychomotor excitement emerged with trying to tear her clothes trying to hit the goods in the room suddenly. She was getting valproic acid 1000 mg/day, lamotrigine 100 mg/day, sertraline 25 mg/day for BD, glatirem acetate for MS. There were no significant findings in the neurological examination. Sertraline was stopped, olanzapine 10 mg/day was added. Demyelinating plaques were observed in the left parietotemporal region in MRI which performed in MS attack accompanied by psychotic symptoms. Common white matter demyelinating lesions were detected in the current brain MRI, the greatest lesion was measured 1.5cm in the left frontal region. The patient was consultated to neurology but they did not recommend anything. Her symptoms decreased within days but soon psychomotor excitement and persecutory thoughts raised again independently from stressors. Coexistence of MS and Mood disorders can be explained by the result of MS lesions, conditions could be simultaneous or may be a reflection of mental illness. MS symptoms can appear in a variety of common neuropsychiatric symptoms and their relationship with MRI abnormalities may vary. Although varied among publications 10-20% of MS patients pathological laughing and crying episodes were indicated, as our case.

In this case report, the rapid variability of affective and psychotic symptoms, the sitauation can not be fully explained according to the DSM-5 criteria of the BD, and despite using mood stabilizer, we have reason to believe it might be associated with MS. Because of psychiatrists are inclined to diagnose symptoms encountered in clinical practice only psychiatric disorders MS could have been missed. Although there is no active plaques seen in MR imaging in hospitalization placement of demyelinating lesions and because of neuropsychiatric symptoms accompanied the previous MS attacks,we thought that the psychiatric condition can not be just related with BD. Our aim in presenting this case, to take attention to the necessity of considering disorders affects central nervous system such as MS even though significant undetected neurological findings existence of atypical symptoms,resistance to treatment,neurlogical complaints in histroy in patients whom diagnosed psychosis or BD.

Keywords: bipolar disorder, multiple sclerosis, pseudobulbar affect

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[Abstract:0665][Neuroscience: Neuroimaging-Genetic Biomarkers]

Three brothers with neurofibromatosis type 1 and attention deficit hyperactivity disorder

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Neurofibromatosis type 1 (NF-1) is an autosomal dominant neurocutaneous disorder and affects approximately 1:3000 individuals. Tumor formation such as neurofibroma and optic glioma, hyperpigmented lesions and typical skeletal lesions can be seen in the clinical presentation. Neuropsychiatric disorders such as mental retardation, autism, developmental language disorders, learning disorders, attention deficit hyperactivity disorder (ADHD), and depression can accompany NF-1.
Case: Here, we will present the diagnosis and follow-up of three brothers with NF-1 and ADHD. Three brothers aged three, seven and ten years presented to the child neurology clinic with dark spots in the body and hyperactivity symptoms. In physical examination; the general appearance of children was well, but more than 10 cafe-au-lait spots greater than 0.5 cm size with small hyperpigmented lesions (freckles) in whole body were detected in each child's body. The medical history of children was unmarked, but it was learned that their parent are 1st degree relatives. Cranial magnetic resonance imaging (cMRI) revealed increased focal signal intensity consistent with NF-1 in globus pallidus, hippocampus and cerebellar peduncle. The cases were diagnosed with NF-1. All of the patients were consulted to the child psychiatry clinic because of excessive irritability, hyperactivity and poor school performance. After the structured interviews and psychometric tests, all three cases were diagnosed with ADHD and a regular follow-up was scheduled. NF-1 is an autosomal dominant hereditary syndrome and develops as a result of the defective gene on chromosome 17. cMRI shows focal increased signal intensity in NF-1 cases from an early period. This circumscribed hyperintense lesions are called as "unidentified bright objects (UBO)" in the literature. Most of these lesions accepted as hamartoma dissapear until puberty. Learning problems and attention deficit symptoms are commonly encountered in NF-1 patients. Cognitive impairment in NF-1 is alleged to be associated with the presence of NF-1 spesific hyperintense lesions on MRI.
The comorbidity of NF-1 and ADHD in 3 brothers patients may not be considered as surprising because NF-1 has an autosomal dominant inheritance, accompanied with high rates of psychiatric disorders; and ADHD is a familial-hereditary disease. The existence of the focal signal changes on cMRI of all three patients may be related to the development of psychiatric disorders as alleged in the literature. In addition, according to some researchers, these lesions are indicative of deterioration or delay in myelination. Future studies are needed to clarify the etiology of ADHD symptoms in NF-1.
Keywords: neurofibromatosis type 1, ADHD, child

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[Abstract:0670][Mood disorders]

Neuro-behcet disease and bipolar disorder: a case report

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In this report, we describe a case of Neuro-Behcet disease with bipolar mood disorder. Behcet's disease is a recurrent and chronic course which is characterized with oral inflammatory ulcers, genital ulcers, eye and skin lesions, arthritis and other neurovascular findings. Neurological involvement is seen in about 5% and in this case of the disease is defined as Neuro-Behcet's disease.
Case: A 29 year-old, high school graduate, married male patient presented to psychiatry service with insomnia, increased speech, and excessive spending money 3 months ago. Mental status examination showed elated mood, over abundant speech, racing thoughts, and irritablity. Although he was offered hospitalization he and his family refused. So sodium valproate 1000 mg/day and quetiapine 300 mg/day were prescribed. One week later he presented to our clinic with similar complaints. In his medical history it was revealed that he had diagnosed as Behcet's Disease 12 years ago and was using colchicine regulary. It was learned from the anamnesis that 3 months ago when his manic attack started he was using steroids for his Behcet's attack and facial paralysis was developed. The patient did not

have psychiatric complaints before and in his family there no history of psychiatric disorder. In his brain magnetic resolutioun image in pons, the left part of mesencephalon, the left thalamus and the left lateral ventricle posterior corpus there were hyperintense foci. He was hospitalized with prediagnosis of manic attack due to general medical condition and his medication is ordered as risperidone 4 mg/day and sodium valproate 1000 mg/day. Steroids that he was using was tapered and discontinued. Approximateley 30 days later he was discharged with a euthymic mood.

The Neuro-Behcet diagnosis of patient and having manic attack at the same time of lowering the steroid dose he was thought as manic attack due to general medical condition. Neurological involvement has been reported to be associated with mania, and in particular the limbic system involvement. In our case, the left part of mesencephalon, the left thalamus and the left lateral ventricle involvement may cause to manic symptoms. In the treatment of Behcet's Disease steroids (systemic or topical), colchicine, cytotoxic agents like azathioprine, chlorambucil, cyclosporin, cyclophosphamide, and interferon alpha are used. Agents used during the treatment of Behcet's Disease may have lead to manic attack.

Keywords: neuro-behcet's disease, bipolar disorder, mania

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[Abstract:0675][Psychopharmacology]

Sertraline-induced galactorrhea with hyperprolactinemia

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Hyperprolactinemia is an adverse effect of psychotropic drugs, especially antipsychotics and one of the clinical consequences of hyperprolactinoma is galactorrhea. Sertraline is a selective serotonin reuptake inhibitor (SSRI) effective for treatment anxiety disorders in children and adolescence. Although sertraline is well-tolerated, insomnia, headache, diarrhea, nausea, drowsiness, fatigue and decreased appetite are among the most reported side effects of sertraline. In this report, we will present a pediatric case who developed galactorrhea with hyperprolactinemia during sertraline treatment.

Case: A 16 year-old patient who was referred to our clinic from gastroenterology due to nausea and vomiting induced by stress. Although she used lansoprazole and domperidone treatment during four mounths regularly her complaints did not improve sufficently. She had excessive wories about safety of her family and her future. She found herself unable to sleep due to her worries and had difficult to shift her attention away from her worries. The patient was diagnosed with generalized anxiety disorder. Sertraline 25 mg/day was prescribed and dose was increased to 50 mg/day a week later. In the second visit which was four weeks later, she reported that in the third week of sertraline treatment she started complaining of flow of milky discharge from both breasts. She was consulted endocrinology. The pregnancy test was negative; thyroid function tests, serum estradiol, follicle-stimulating hormone and complete blood count levels were normal. The serum prolactine level was 97.81 ng/ml. The brain magnetic resonance (MR) imaging did not show any abnormalities. Galactorrhea was considered a possible adverse effect of sertraline and sertraline treatment was stopped. After the cessation of sertraline, the serum prolactine level decrease 34.69 ng/ml in three days and, galactorrhea diminished over time and disappeared totally in six weeks. Four weeks after cessation of sertraline, 10 mg/day citalopram treatment was initiated Because of not tolerating, citalopram dose was decreased to 5mg/day and during six weeks of citaloplam treatment she did not develop galactorrhea.

We presented a case who developed galactorrhea during sertraline treatment but did not during citalopram treatment. Galactorhea is reported side effect of escitalopram, paroxetine fluoxetine, fluvoksamine and sertraline. According to studies hyperprolactinemia is mediated by postsynaptic 5-HT receptors via serotonin activation. 5HT stimulates PRL release from pituitary gland directly and indirectly though stimulation of PRL-releasing factors such as vasointestinal peptid and oxytocin and inhibition of PRL inhibition factors such as dopamine. Although galactorrhea is a rare side effect of sertraline, clinicians should be aware that sertraline may cause galactorrhea and this does not eliminate other members of SSRIs from the future treatment options.

Keywords: galactorrhea, hyperprolactinemia, sertraline

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[Abstract:0680][Autism]

A case of alternating hemiplegia of childhood presenting with autism spectrum disorder and mental retardation

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Alternating hemiplegia of childhood (AHC) is a rare neurodevelopmental disorder characterized with abnormal eye movements, hemiplegic attacks, dystonic episodes, psychomotor regression, developmental delay and neurologic deficits. The incidence is approximately one in million. There is no sex difference between affected individuals. AHC is a type of pediatric migraine equivalent, like abdominal migraine, cyclic vomitting syndrome, benign paroxysmal torticollis, benign paroxysmal vertigo. They all share common clinical characteristics like episodic feature, positive family history of migraine, between episodes neurologic examination is normal, and proceeding to classic types of migraine. Early onset of hemiplegic attacks is usually characterized with poor development. The disease may be observed in three stages; abnormal eye movements, dystonic episodes and developmental delay. Mental retardation and autism spectrum disorders are childhood onset disorders that take place in topic of neurodevelopmental disorders. AHC and autism spectrum disorders are both neurodevelopmental disorders that may share similar origins and may affect individuals together. AHC patients may have psychiatric comorbidities like language delay, mental retardation, attention deficit and hyperactivity disorder. Our aim is to present a case of AHC with autism spectrum disorder and mental retardation.

Case: A 14 year-old boy who approved our child psychiatry clinic with aggression against his family. He was having hemiplegic attacks 2 times in a week, and when he was stresful, attacks are becoming more frequent. The attacks was beginning with sudden loss of tonicity and loss of conciousness was not accompanied. His treatment was flunarizine. His speech and walking are also deteriorated and he was taking physical therapy and speech therapy. There was also stereotypies like perseverative questioning, whirl round himself and restrictive areas of interest. He was left school from second year of elementary school. He also could not be able to go formal education, and his family is demanding health certificate for homeschooling. His intelligence quotient was 51 according to Stanford Binet test. We had started risperidone 1 mg/day for behavioral problems and irritability. His irritability is improved with risperidone and hemiplegic attacks did not get worsened.

AHC is a neurologic syndrome which also causes some life style challenges. Mental retardation and other motor development deteriorations may cause problem in childs' daily functioning. In addition to flunarizine treatment we may also organize educational and social achievement, and decrease severity of psychiatric comorbidity. We present here a paitent who become more stabile in terms of behavioral disturbances which improved after administration of risperidone. No side effect is recognized. To our knowledge, this is the first case of AHC with autism spectrum disorder. Both of them originat neurodevelopmentalliy and this may increase comorbidity. Comorbidities may complicate patients' life quality, learning difficulties and social adjustment. Therefore the clinicians should assess the behavioral and social problems originating from either from disease itself or another psychiatric comorbidity to get better improvement.

Keywords: alternating hemiplegia of childhood, autism spectrum disorder, risperidone

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[Abstract:0682][Schizophrenia and other psychotic disorders]

Menstrual psychosis in a 15 year-old girl: a case report

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Cyclic psychosis and menstruation during puberty have been subject of clinical interest since 1807. But, their underlying biological mechanisms are still unclear. Schizophrenia, bipolar disorder and depression can initially present with transient psychotic symptoms. Psychotic episodes with menstrual cycle represent a distinct and unfamiliar subgroup. Features of this subgrup is sudden onset, short duration and complete remission. Patients present to clinic with symptoms psychotic symptoms, and typical psychotic features comprise

perplexity, stupor, mutism, delusion and hallucinations. We report the case of an adolescent girl who was 15 year-old at initial presentation.
Case: A 15 year-old girl who came our emergency service in 2015 August with psychomotor retardation and agitation, refusing feeding and insomnia. It was learned that the symptoms had begun three days before. We could not cominicate in emergency servise and followed up patient in inpatient clinic. During the psychiatric interviews she talked about auditory hallucinations ("I heard a voice insulted me") and persecutory delusions ("I thought them mom put poison into my foods"). Her family noted a pattern that coincided with her menstrual cycle. She had menstrual cycles for six months and this was the first time with similar symptoms. An endocrine consult was obtained, and the diagnosis of catamenial psychosis was performed. After finishing the menstruation the patient's symptoms were resolved completely. We observed her for one more cyle and treated with antipsychotics.
Menstrual psychosis that we presented as a case has not still diagnosed as mental disorders in DSM-5 or ICD 10. But symptoms and the course of menstruel psychosis was defined by some authors. Brockington defined as; menstrual psychosis is a unique syndrome in that it presents with episodes of acute psychotic symptoms of sudden onset, which occur in premenstrual period, continues through menstrual period and spontaneously remits within 15 days since onset. The patient remained symptoms free until symptoms recur with the next menstrual cycle. In our case we presented a girl who had acute psychosis symtoms three days before catamenia and had spontaneous remission after catamenia. This psychosis symtoms repeated two more cycle as the same pattern. This case reports important to provide to occur menstrual psychosis as a diagnose in younger acute psychosis and draw attention for future studies about diagnosis criterias.
Keywords: menstrual psychosis, adolescents, cyclic psychosis

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[Abstract:0684][Perinatal psychiatry]

Successful buprenorphine-naloxone detoxification treatment of an adolescent opioid user with pregnancy in the outpatient setting

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A combination of buprenorphine and naloxone in sublingual tablet form, was proven as a safe and effective treatment for opioid dependence. Although it is approved for the treatment of individuals aged 16 years and older, no studies have been published which systematically assess the effectiveness of buprenorphine-naloxone in younger adolescents. Due to the dangers associated with untreated opioid addiction and favourable studies of buprenorphine in some studies, it may be a choice among opioid-dependent adolescents under 16 year-old. Unplanned pregnancy in opiod users is not a rare condition: a recent study from Australia found that opioid users have high pregnency rate (approximately 28.9%), poor uptake of contraception and high rates of adverse pregnancy outcomes including abortus, premature labour, low birth weight, foetal distress, microcephaly and neonatal opioid abstinence syndrome. For that reason, treatment is important and widely accepted in pregnancy, to achieve better neonatal and maternal outcomes. Although most guidelines recommend methadone and buprenorphine medication to manage opioid addiction in pregnancy, buprenorphine-naloxone combination is the only agent that is used for opioid maintenance treatment in Turkey. We report a case of opioid-dependent pregnant, who was 15 year-old and was followed with buprenorphine-naloxone detoxification treatment during the last stages of her pregnancy at the outpatient setting.
Case: A 15 year-old female patient who was brought to our department due to use of heroin for six months. Upon questioning it was learned that she was in the 31th weeks of pregnancy. According to the clinical evaluation of the patient based on DSM-5 diagnostic criteria, she was determined to fulfill the diagnostic criteria of opioid use disorder. She refused the treatment program as an inpatient. As severe-daily opioid use is more hazardous for developing fetus than opioid detoxification treatment, we prescribed buprenorphine-naloxone treatment for maintenance therapy during pregnancy. She used this drug combination at the dose 2/0.5 mg/day during one month. She had mild general withdrawal symptoms like muscle aches, nausea. She used heroin only once at the 35th weeks of pregnancy during this treatment. After the 36th weeks of gestation, buprenorhine-naloxone treatment was reduced and ceased gradually in two weeks. She gave birth at the 38th weeks by cesarean section. Her baby was healthy, does not have any assigned pathology. Her baby birth weight was 2400 gr, the birth height was 45 cm and the head circumference was 33 cm. There was no sign of opioid abstinence and withdrawal symptoms in the newborn period of the baby. After birth when she came for follow-up, she did not use any substance and did not experience any craving or withdrawal sign. The patient is still being followed in our outpatient clinic and at the clinical evaluation we did not determine any substance use in clinical assesment and urine drug analyses.
According to our case follow-up buprenorphine-naloxone combination may be safe and effective alternative in opioid-dependent

adolescent pregnants. Although naloxone has been suggested to be possible teratogenic, its oral bioavailability is negligible and we and the pediatricians did not observe any malformation or abnormality in the newborn.
Keywords: adolscent, buprenorphine-naloxone, opioid, pregnancy

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[Abstract:0688][Psychopharmacology]

A case report with lithium -induced myasthenia

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Bipolar illness is a major psychiatric disorder that affects 1–3% of the worldwide population. Lithium is the classic mood stabilizer and it was the first drug approved by the Food and Drug Administration (FDA) in 1974 for maintenance treatment of bipolar disorder. Even lithium is effective, it has several side effects such as ataxia, dysarthria, delirium, tremor, polyuria, polydypsia, cardiovascular changes. Little is known about the effects of lithium carbonate on peripheral neuromuscular function. The mechanism of the lithium-induced elicitation of myasthenia gravis symptoms is unclear and mainly reflects a drug's interaction with a concurrent or underlying neuromuscular disorder, rather than impending lithium toxicity. Lithium may produce myasthenic symptoms because of its competition for calcium inside the presynaptic motor nerve terminal, finally reducing acetylcholine (Ach)-synthesis and voltage-gated quanta release of acetylcholine. Lithium affects down-regulation of Nicotinic Acetylcholine-receptors (nAchRs) and this might be another reason of the lithium associated myasthenic syndrome. On literature there are five case report occurred myasthenic syndrome during lithium treatment.
Case: A 16 year-old girl who presented to Dokuz Eylul University Child and Adolescent Psychiatry Inpatient Unit with symptoms of elevation, irritability and aggressive behavior, grandiosity, decreased need for sleep, obsession of clean. The patient who presented to a child and adolescent psychiatrist for a month ago has been evaluated as bipolar disorder-manic episode. She has been taking 1000 mg/day valproic acid and olanzapine 10 mg/day when she has been referred to our clinic. Following hospitalization, because her serum valproic acid level has been elevated toxic doses, the dosage of drug has been reduced 500 mg/day. However valproic acid has not been effective at this dosage. Valproic acid has been replaced by the lithium. Her lithium dosing has been increased 900 mg/day in 3 days. Because of having difficulties to regulate serum level of lithium, the lithium dosage has been switched alternate-day dosing (1200-1200-900 mg). On twelfth day of treatment, symptoms such as strabismus, the left eyelid ptosis, slurred speech, nasal quality to her voice, difficulty swallowing, weakness in her arms and legs, intentional tremor has been appeared. She was consulted to the child neurology department and several investigations such as Antibodies against Acetylcholine-receptors (AchR), CK, EMG administered and they haven't been abnormal. According to test results, Myasthenia Gravis has been eliminated and symptoms have been diagnosed as lithium -induced myasthenic syndrome. At a common meeting with the child neurology department it is decided that lithium treatment must stop and dual antipsychotic agent must be given in order not to take the risk of bulbar involvement. On the seventh day of lithium discontinue myasthenic symptoms have been resolved. The client, whose manic symptoms were regressed with lithium, has been discharged after her medication's regulating as aripiprazole 30 mg/d quetiapine 700 mg/d and valproic acid 500 mg/d.
We aimed to present a case with bipolar disorder who has been occurred lithium-induced myasthenia. It is needed more research about the pathogenesis and treatment of the myasthenia both induced and unmasked by lithium.
Keywords: bipolar disorder, lithium-induced myasthenia, lithium side effect

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[Abstract:0690][Others]

Co-occurrence of hoarding and Sturge-Weber syndrome: a case report

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Sturge-Weber syndrome (SWS) is a neuro-cutaneous disorder characterized by cutaneous facial angioma, leptomeningeal angioma associated with seizures and other neurologic complications including mental retardation. Hoarding is a psychological disorder characterized by excessive collecting, storage and inability to discard large quantities of the objects, usually accompanying a severe level of distress or dysfunctionality. In DSM-IV-TR, hoarding is listed as one of the diagnostic criteria for obsessive–compulsive personality disorder (OCPD). However, hoarding disorder appears as a distinct psychiatric disorder in the last updated version of the DSM-5. Accordingly, in this case report we aimed to explain about hoarding phenomenon seen in a patient with Sturge-Weber syndrome. In the current literature, comorbidity of these two situations is extremely rare which is why we aimed to draw attention to this co-occurrence, possibly to raise a question regarding any form of association.

Case: A 44 year-old male patient, single, illiterate, presented to psychiatry department involuntarily with his brother's consent. He was complaining from irritability, ideas of reference, monologues speech and hoarding trash such as wood and bottles from the street for the past year. The patient was claimed to get angry irrationally, use foul language in improper setting and behave according to his ideas of reference for instance teasing dwellers in the neighborhood. Throughout the patient's symptomatology, he refused to apply to a medical facility. Neurodevelopmental stages were all retarded including walking, talking and reading. He never managed to continue school nor had an occupation. Having difficulties in maintaining self-care, he cannot cook but was able to eat on his own. The patient has been under the follow-up by neurology with the diagnosis of SWS and was under levatirecetam, carbamazepine and lamotrigine for the control of the epileptic seizures. Remarkable inspective sign was a port wine stain on the right side of his face. On mental examination he was reckless and puerile but still can cooperate with the interviewer. He had a dysarthria in his speech and looseness of associations. Psychomotor activity was in usual range, his affect was minimally blunt but reactive, with an irritable mood. He had reference thoughts but denied hallucinations. His insight was poor. He was started on aripiprazole and titrated up to 10 mg/day and responded well. In this case, onset of hoarding behavior was reported in the setting of intellectual disability which is a common manifestation of SWS. For the last 1 year, deterioration in frustration tolerance and poor impulse control might be in the underlying mechanism of development of hoarding behavior.

Up until recently, hoarding syndrome has been regarded as a subtype of OCD, but with a modification in DSM-5 gained importance as a distinct disorder. In the current literature, wide range of studies indicates that hoarding is related with impulse control disorders. In this case, in the context of intellectual disability and further deterioration in impulse control, onset of hoarding phenomenon might be considered as a sign of common etiological factor or outcome.

Keywords: Sturge-Weber syndrome, hoarding, aripiprazole

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[Abstract:0691][Psychopharmacology]

Difficulty with urination in an child patient caused by fluoxetine use

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The operating frequency of obsessive compulsive disorder (OCD) in children and adolescents are reported to occur with a rate of 1-3%. Obsessions are repetitive, annoying, failing to state that the removal effort is considered to be unreasonable thoughts, impulses and fantasies. Compulsions are in response to the obsessions, repetitive behaviors or mental acts appeared to reduce anxiety from the obsessive nature. Most studied on the most commonly used drugs in the treatment of OCD are clomipramine and SSRIs. Fluoxetine is a selective serotonin reuptake inhibitor (SSRI), wherein one of the drugs block the reuptake of serotonin by inhibiting the serotonin transporter, but also (5 HT 2C), serotonin also leads to an increase in dopamine and norepinephrine levels by an antagonistic action to

5HT2c receptor. The combination of fluoxetine with other drugs reported to cause urinating problems, however according to our research is quite limited that lead to urinary problems fluoxetine alone. Here, a 9 year-old girl who has difficultyin urinating due to fluoxetine use will be discussed with literature.

Case: A 9 year-old female patient presented to our outpatient clinic with complaints as for the last 15-20 days continuing, inability to make sure they do during the day, said before the act of receiving her mother for each action. Starter stress is not described and the mother in this case would result in the loss marked the day time. The patient need to receive consent was considered to be as a compulsion, no evidence accompanying obsessions and compulsions other. The depressive symptoms of the patients did not have psychotic symptoms. The patients resume was not described medical or psychiatric illness. The patient is describing the compulsions, affects the functionality of the patient and the patient's relatives. Fluoxetine 20 mg/day was started. Complaint of difficulty in urinating appeared despite the request of the patient. Physical examination and laboratory tests were normal. Urinary tract ultrasonography examination was normal. Research on patients while continuing to be the first time night bedwetting complaints was shown. After stopping fluoxetine treatment, the patient was followed, her complaints were regressed completely in one week.

It has multiple factors that cause difficulty urinating. SSRIs such as fluoxetine may cause this side effect very rarely. Serotonin increases the storage of urine by activating the sympathetic pathway and inhibiting the parasympathetic pathway. Although cases in the literature in combination of fluoxetine with haloperidol and risperidone resulting with difficulty in urination have been reported, no cases with fluoxetine alone have been reported. It is not known that by which mechanism SSRI drugs causing difficulty in urination. Therefore, further research is needed to examine this issue.

Keywords: difficulty urinating, fluoxetine, SSRI

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[Abstract:0692][Psychopharmacology]

Escitalopram-induced amenorrhoea: a case report

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A polypeptide hormone, prolactin, is produced in the anterior pituitary gland by lactotroph cells. Even though hyperprolactinaemia as an adverse effect of antipsychotic medications is well established, this phenomenon is less well identified among patients taking antidepressants. SSRI-induced (including sertraline, fluoxetine, paroxetine and fluvoxamine) hyperprolactinemia and galactorrhea cases have been reported. However, to the best of our knowledge there are no previous reports of escitalopram-induced amenorrhoea accompanied by hyperprolactinemia. Here, we present the case of a 36 year-old female patient who was treated with escitalopram for her panic disorder and developed amenorrhoea with hyperprolactinemia that resolved upon discontinuation of the drug.

Case: A 36 year-old female patient presented to the emergency department with complaints of palpitation, shortness of breath, sensations of tingling on both arms, fainting and fear of having a heart attack that started one month ago. After a thorough physical assessment, lab tests and cardiology consultation were completed, she has been referred to psychiatry. She presented to our outpatient clinic with the same symptoms, and she was diagnosed with Panic Disorder (without agoraphobia) according to DSM-IV-TR. Nothing significant was detected in her past medical history Laboratory tests including blood chemistry, complete blood count, thyroid function tests, and serum vitamin B12 level were within their normal ranges. Escitalopram 10 mg per day was prescribed with a brief psychoeducation about cognitive model of panic disorder. She stated at the tenth month that she had not had menstruation for the last 2 months. She was not pregnant nor was she nursing. She was not on birth control pills or any other medication except escitalopram at the time. She did not have any history of menstrual irregularity in the past. Beta-HCG was negative and her serum prolactin level was 27.77 ng/ml (reference interval: 4.79-23.3ng/ml). Escitalopram treatment was stopped and fluoxetine 20mg/day was administered. She reported that she had spotting after 2 weeks, and she had menstruation after the fourth week. Her serum prolactin level was found to be 10.13ng/ml at the second week of change in her regimen. She did not convey any irregularity in her cycles during her following appointments.

The return to normal menstrual cycle and serum prolactin levels dropping back to its normal range after changing escitalopram to fluoxetine lead us to thinking that the amenorrhoea was escitalopram-related. It still remains unclear how serotonergic agents cause hyperprolactinemia. Hyperprolactinemia caused by SSRIs might be the outcome of dopamine neuron inhibition at the hypothalamus which has an inhibitory control over prolactin release. It might also result from direct stimulation of prolactin release through post-synaptic serotonin receptors in the hypothalamus. Her (B-HCG) test was negative, we were able to eliminate underlying kidney disease, hypothyroidism or pregnancy as possible causes of amenorrhoea. Even though cases of hyperprolactinemia due to fluoxetine have been

reported, the symptoms of our patient diminished after the change in her treatment regimen from escitalopram to fluoxetine. As the use of SSRIs increases, clinicians can expect the emergence of relatively rare side effects.
Keywords: escitalopram, amenorrhoea, hyperprolactinaemia

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[Abstract:0694][Psychopharmacology]

Paliperidone -induced atypical neuroleptic malignant syndrome and delirium: a case report

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Neuroleptic malignant syndrome (NMS) is a life-threatening drug reaction which developed by neuroleptic drug use. Despite there is a high risk of developing NMS with typical antipsychotics, NMS can be seen with atypical antipsychotic use. But, paliperidone -induced NMS is rare. When NMS and delirium is together, treatment decisions for patients with agitation is difficult. In our case, we aimed to discuss a patient with paliperidone depot -induced atypical neuroleptic malignant syndrome and delirium together.
Case: A 63 year old male patient who was followed up with a diagnosis of schizophrenia was brought by relatives to the emergency room with muscle contraction, tremor, urinary and fecal incontinence, changes in consciousness, high fever symptoms. It was learned that these symptoms had began after 2 two days following paliperidone depot injection. In the psychiatric examination reduction in self-care, tremors and muscle rigidite were noted. He was confused. Orientation was impaired. The patient's vital signs were stable during follow-up in the emergency room, but it was learned that metamizole ampoule injection had been made because of 39.5ºC fever in the history. Creatine kinase level was 1895 U/ L. The patient was admitted to our psychiatry clinic. Patient were treated with bromocriptine 10 mg/day. In the seconde day of the treatment muscle rigidite and creatine kinase levels decreased and vital signs became stable. But the patient's delirium continued. In the laboratory examnations urinary tract infection were detected and antibiotic treatment was given to the patient. Neuroleptic malignant syndrome (NMS) is a highly mortal condition when it is late for diagnose. Clinical symptoms are fever, diaphoresis, muscle rigidite, changes in mental status and autonomic instability. Increase of creatine kinase (CK) in laboratory tests is significant in NMS, usually above 1,000 IU/ L. there is a high risk of developing NMS with typical antipsychotics associated with affinity for Dopamin 2 receptors. A decrease is observed in the prevalence of NMS in recent years. This situation is explained with the use of atypical antipsychotics, lower doses of antipsychotic use and less polypharmacy. NMS cases induced by atypical antipsychotics like clozapine, olanzapine, quetiapine, ziprasidone were reported in the literature. But paliperidone -induced NMS is rare. Duggal, reported a paliperidone -induced NMS in a 63 year-old, chronic schizophrenic patient. For clinicians; in NMS and delirium together, treatment decisions for patient's agitation is difficult. NMS is a rare but life-threatening condition and early recognition and intervention in such cases are essential to prevent the potential mortality. Clinicians have to be mindful for NMS in patients using atypical antipsychotic such as paliperidone
Keywords: paliperidone, neuroleptic malignant syndrome, delirium

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[Abstract:0695][OCD]

Improvement of OCD after fronto-parietal ischemia: a case report

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The etiology of obsessive compulsive disorder (OCD) is unclear and includes multiple genes and environmental factors. Disturbances in the frontal-striatal and frontoparietal brain circuits may contribute the pathogenesis. Generally, the structural and functional neuroimaging studies have consistently reported an abnormal over activity cortico– striato–pallido–thalamo–cortical circuits as well as fronto-striatal areas. We report a case of OCD who had an ischemic event damaged to fronto-parietal lobes and her symptoms of OCD

improved after injury without antiobsesional treatment.
Case: A 30 year-old female patient, presented to the outpatient with complaints of contamination obsessions and washing compulsions. She had no past history of psychiatric illness. Routine laboratory investigations were within normal limits. She was diagnosed with OCD according to DSM-5 criteria. The Yale Brown Obsessive Compulsive Scale (Y-BOCS) was scored as 30. Fluoxetine 20 mg/day was initiated. In the 2nd month of follow up, her total Y- BOCS score was 20, fluoxetine dose was increased to 40 mg/day dose. In the 4th month of follow up, she presented to the emergency 2 hours after the sudden onset of left weakness. On admission, the right-handed female patient had left hemiplegia and hypoesthesia. She was apyretic, and blood pressure was 140/80 mmHg with regular pulse of 70/min. Brain CT scan performed at 24 hours from admission showed a hypodense area on the right fronto-parietal lobes with an aspect of a subacute ischemic lesion; the ventricular system was not deviated from the midline. Brain MRI and diffusion MRI performed that demonstrated hyperintense areas at FLAIR AG, in right parieto-occipital lobes on the level of centrum semiovale. These findings were compatible with acute stage ischemia. MRI angiography was showed hyperintense in B1000, hypointense in ADC acute phase ischemic changes including occasionally hemoragic areas on right parieto-occipital lobes. Additionally, secondary to ischemia, compression findings observed on right lateral ventricule, leftward shift on midline and edema signs on right cerebral hemisphere. After immediate intervention she followed and treated in the department of neurology. In her first visit of psychiatry after discharge from the neurology, her total Y- BOCS score was 10 despite no treatment. Our follow-up continues without any antiobsessional treatment for 3 months since the stroke.
Many studies have reported the disturbances in the frontal-striatal and fronto-parietal neural circuits may conduce the pathogenesis although the etiology of OCD is still not fully understood. The longitudinal neuroimaging studies have been demonstrated that SSRIs reduce functional activity in the fronto-subcortical regions decrease thalamic volume and increase neuronal vitability in frontal regions. Approximately 10% of OCD patients maintain their unresponsive situation against all therapies and continue suffering from severe symptoms causing functional deterioration in refractory cases, the literature supports stereotactic lesional neurosurgical interventions which have provided expected effective response in OCD symptoms. In light of our case and literature related to the pathology of OCD and the mechanism of therapeutic interventions including SSRI and neurosurgery, we thought that the condition of our case is evidence related to over activity in fronto-parieto-occipital brain circuits blamed in the pathology of OCD.
Keywords: fronto-parietal ischemia, OCD, treatment

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[Abstract:0701][Psychosomatic medicine-Liaison psychiatry]

Differential diagnosis and treatment of tardive blepharospasm and psychogenic blepharospasm in a schizophrenic patient with long term antipsychotic drug use: Case report

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Blepharospasm is a movement disorder which that characterized by generally contraction of orbicularis oculi. The most common type is primary (idiopathic) subtypes though, common causes of secondary blepharospasm are structural brain lesions, neurodegenerative diseases such as parkinson's, tardive syndromes after use of drug, abnormal functioning of basal ganglia. Also it may be seen in the clinic with some psychiatric disorders, a side effect of antipsychotic drugs or as psychogenic blepharospasm. In thıs case we aimed to show that differential diagnosis of psychogenic blepharospasm in a patient using antipsychotic drug who diagnosed with schizophrenia.
Case: A 25 year-old male patient with paranoid schizophrenia who presented with difficulty opening his eyes for two years. For the previous 7 years he had been continuously treated with various antipsychotic drugs such as risperidone 4 mg/day, quetiapine 50 mg/day, chlorpromazine 50 mg/day, sulpiride 100 mg/day. During this period, he has been hospitalized in psychiatry clinic because of acute psychosis. However, the patient did not use his drugs regularly and paid no attention about his control examination. Afterwards he presented to our clinic due to his challenging symptoms such as sudden irritability, hostility, suspiciousness, overpowering intense feeling that people are talking about him, looking at him, thinking people are working together to intend to harm him, social withdrawal. He diagnosed with paranoid schizophrenia and treated with paliperidone palmitate 150 mg. Also he was consulted to neurology clinic for his complaint of difficulty opening eyes and diagnosed with blepharospasm. MRG ve EEG tests were normal. The patient was treated with injection of botulinum. After the botulinum treatment his complaints recovered completely but the day after his complaints repeated immediately. Furhermore during the clinical observation complaints of closing his eyes have seen specially when he was bored and the

doctors and nurses visit to him. In this case, early and acute onset, abnormal rapid response to treatment, especially emerging during the visit as factors that led us psychogenic blepharospasm.
Blepharospasm may be seen in the clinic with several psychiatric disorders, a side effect of antipsychotic drugs or as psychogenic blepharospasm. So that differential diagnosis psychogenic blepharospasm may be challenging in psychiatric patients. Clarification of etiology of blepharospasm can provide appropriate treatment and prevent misdiagnose and inessential treatment approach.
Keywords: antipsychotic drugs, psychogenic blepharospasm, schzophrenia

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[Abstract:0706][Substance-related and addictive disorders]

Alcohol-induced psychotic disorder: a case report

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Alcohol-Induced Psychotic Disorder (AIPD) also known as alcohol hallucinosis is a rare complication of chronic alcohol abuse. Usually it is characterized by acute onset of auditory hallucinations and persecutory delusions, in clear consciousness and the absence of thought process disorder in individuals with heavy alcohol consumption. The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) stipulates that the period of onset should be "during or soon" after intoxication or withdrawal of alcohol and that the disturbance should cause clinical significant distress or impairment. AIPD is associated with high co-morbidity with other psychiatric disorders, high re-hospitalization and mortality rates and suicidal behavior so it is important to be well diagnosed by clinician.
Case: A 48 year-old male patient was admitted to a psychiatric hospital with hallucinations of acute onset during alcohol abuse which persisted for eight days of abstinence. He had 15 year history of almost alcohol daily use. He was fully alert and oriented. His consciousness, attention and higher cognitive functions remained intact. Physical examination, laboratory findings and neuroimaging investigations showed no abnormalities. The patient recovered on treatment with risperidone.
AIPD is a rare complication of chronic alcohol abuse. The diagnosis must be distinguished from delirium tremens and schizophrenia spectrum disorder as treatment and prognosis are essentially different.
Keywords: alcohol hallusinosis, alcohol dependency, psychotic disorder

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[Abstract:0715][Psychopharmacology]

Dry eye syndrome associated with aripiprazole: a case report

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Atypical antipsychotics constitute the main treatment of schizophrenia during decades with significantly efficiency and tolerability profile. Aripiprazole has a different receptor binding profile and high affinity for dopamine D2 and D3. Many medications, including antipsychotic drugs, may cause dry eye problems. Dry eye syndrome(DES) is common ocular condition that is characterized by abnormal function of tear film and ocular surface inflammation. We report a case in which severe dry eye symptoms were induced by aripiprazole.
Case: A 42 year-old female patient presented to outpatient clinic with complaints of smile for no reason. In her mental examination, she was conscious, impaired concentration, bizarre thinking, trouble with emotional expression, speech with herself, lack of insight and insomnia. She was diagnosed with schizophrenia according to DSM-5. Physical and laboratory examinations were within normal limit. Aripiprazole 10 mg/day were administered to the patient for management of her psychiatric symptoms. In the 2nd month of follow up, the patient presented with a chief complaint of eye irritation, dryness, and fluctuating visual disturbances. She had no history of arthralgias or dry mouth previously, also with these eye discomfort symptoms, she had undergone internal medicine, rheumatology

and ophthalmology examination. Physical examination was normal except mild conjunctival congestion and redness. Laboratory results were determined normal results including thyroid, renal and liver function indicators, blood count, ESR, antinuclear antibodies, Ro, and rheumatoid factor concentration. According to findings, DES was thought. Aripiprazole was switched to paliperidone 9 mg/day. This is the beginning of 2nd month of paliperidone treatment, all symptoms associated with dry eye and her psychotic symptoms were improved.
Aripiprazole have shown effective response in both of the positive and negative symptoms accompanied with a rapid influence within one week. Studies demonstrated that aripiprazole is effective in various psychiatric disorders and have exhibited tolerable adverse event profile. In DES, the damage and discomfort of ocular surface may cause symptoms including blurred vision or visual disturbance, eye dryness, irritation, burning, itching, redness or grittiness of eye, sensitivity to light and foreign body sensation. In our patient we observed the symptoms of visual diturbance, eye dryness, irritation, redness. Various factors act the pathogenesis of this common condition however risk factors include female sex, older age, postmenopausal estrogen therapy, computer use, contact lens wear, alcohol, smoking, diet, malignancy therapies, viral infections such as HIV and Hepatitis C, diabetes mellitus, systemic and ocular medications, including antihistamines, isotretinoin, antidepressants, anxiolytics, antipsychotics, diuretics and beta-blockers. In our case, all risk factors, above-mentioned, were excluded except female sex and antipsychotic drug use. The function of aripiprazole is defined as a partial agonist at dopamine D2 and serotonin 5-HT1A receptors, and as an antagonist at the serotonin 5-HT2 receptor. It is known that the affinity for muscarinic receptors of aripiprazole is remissible therefore these findings, observed in our patient, might be helpful for further understanding of the mechanisms of aripiprazole-induced dry eye syndrome. Although most drug-induced DES have been established in previous studies, rare ones, such as the case described here, require considerable attention.
Keywords: aripiprazole, dry eye syndrome, paliperidone

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[Abstract:0719][Mood disorders]

Psoriasis activation with lithium: a case report

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The efficacy of lithium in the management of acute mania is well established. Lithium found efficacy and tolerability of this agent for the treatment of bipolar disorder. There are several side effects that result from lithium carbonate therapy. The common side effect reported are polydipsia, polyuria, nephrogenic diabetes insipidus, tremor, hypothyroidism, increase of PTH and calcium levels, and weight gain. There have also been reports of various skin lesions with lithium treatment too. An exacerbation or activation of psoriasis is also frequently associated with lithium treatment. Mechanisms involving both immunologic and non-immunologic factors have been examined in various studies. No consensus has been reached and further investigation is needed. However, findings such as improvement with inositol supplementation in cases of lithium-induced and -exacerbated psoriasis
Case: A 33 year-old, female patient, presented with psoriasis activation after started lithium during a new-onset manic episode treatment. Increased motor activity, energy and grandiosity, decreased sleep, talkativeness, irritability, aggression, euphoria, increase in religious interests, speaking spontaneity, mystic and grandiose delusions were started before two weeks ago. She had been hospitalized with the diagnosis of bipolar disorder, psychotic-manic episodes. Treatment of patient was started with lithium 600 mg/day and olanzapine 10 mg/day. Psoriatic skin lesions developed at her body after 3 weeks of medication intake. Psoriasis activation was considering related with lithuril. Lithuril was chanced sodium valproate. Her skin eruptions disappeared after was stopped lithium. She still using sodium valproate for two years without any problem.
Clinicians should know potential side effects (renal, endocrine and dermatological) associated with long-term treatment with lithium, for a correct management of the patient. Clinicians must know patient's dermatologic history before using any psychiatric treatment known as causing dermatologic adverse effects, and dermatology consultation should be established whenever necessary.
Keywords: bipolar disorder, lithium, psoriasis

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[Abstract:0724][Psychopharmacology]

Alopecia associated with agomelatine: a case report

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Alopecia areata is a common immune-mediated disorder characterized by non-scarring hair loss of scalp and body. Hair loss may seen in limited or diffuse forms. Limited form is defined oval, well-rounded, one or more patches on the scalp or body while diffuse form influence all the scalp named as alopecia totalis or all the body named alopecia universalis. Many psychotropic medications including mood stabilizers, antipsychotics and antidepressants such as TCA, SSRI, SNRI can cause hair loss with a heterogenous clinically presentation. We report a case of alopecia as an adverse effect of agomelatine.

Case: A 29 year-old female patient referred to psychiatry ward with depressive symptoms including unhappiness, unwillingness, malaise, anorexia and sleep problems. She had no history of psychiatric treatment. Beck depression scale score was 54 in her first visit. She was diagnosed with MDD according to DSM-5 and agomelatine was started 25 mg/day. During 2nd month of treatment, her depressive symptoms were improved and Beck depression scale score was 14. But she started to experience hairless on the scalp since 20th day of the treatment. Physical examination was unremarkable except for the hair loss on the scalp. Laboratory work for blood count, ESR, syphilis, HIV, rheumatoid factor, antinuclear antibody, anti-thyroglobulin antibody and anti-thyroid peroxidase antibody were unremarkable. She was consultated to a dermatologist who noticed alopecia areata. Discontinuation of agomelatine was suggested to the patient. Milnacipran 50 mg/d was initiated. In the 3th months of Milnacipran treatment, there was observed no hair loss again and the depressive symptoms were improved.

Here, we report a case of hair loss in a 29 year-old female patient suffering from major depressive disorder and treated with agomelatine. According to our knowledge, this is the first case related with agomelatine and hair loss. Alopecia areata is an autoimmune condition of hair loss and it's prevalence is about 2% in lifetime. The cases can be diagnosed by well-surrounded patches of hair loss without scarring. Scalp biopsy is typically not necessary for the diagnosis that is often characterized by a lymphocytic infiltrate around the bulb region of the hair follicle. Alopecia areata has a genetic predisposition further drug -induced alopecia suggested that it might be associated with chelation of zinc and selenium, factors affecting the T-cell-mediated inflammatory process and autoimmune process due to role of the psychopharmaceutical's on the monoaminergic pathways, hirsutism related to the elevation of prolactine caused by psychotropic drugs. According to our case, agomelatine related with hair loss should be taken to account while searching the cause of this condition. Further studies require exploring the mechanism of this relationship.

Keywords: agomelatine, alopecia, milnacipran

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[Abstract:0728][Mood disorders]

Kleine-Levine syndrome co-occurring with bipolar disorder: a case report

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Klein-Levine syndrome (KLS) is a rare (1-2 reported cases per 1 million) that affects mostly men in young adulthood. The etiology of KLS is unknown however the reported flu-like symptoms prior to the initial KLS attack suggest that autoimmune mechanisms and genetic factors might have a role in the ethiopathogenesis. The KLS attacks are characterized by hypersomnia (15-21 hours/day), cognitive impairment, derealization and more infrequently hypersexuality and depressive mood. Common mechanisms as circadian rhythm abnormalities and clinical similarities indicate a possible relationship between KLS and Mood disorders. A rare case of KLS and bipolar disorder (BD) co-occurrence is presented in this report.

Case: A 34 year-old male patient followed up for BD-I for 15 years was hospitalized with a preliminary diagnosis of mania after pathological behavious such as spending too much money, excessive talking and eating, hypersexuality, disorganized behaviors. The patient was not using his medications regularly and the previously prescribed treatment of valproic acid 1000 mg/day, Lithium 600 mg/day, lamotrigine

200 mg/day and risperidone 6 mg/day was initiated. On the sixth day of hospitalization the signs of hypersomnia were seen. The patient could only be woken up for his daily needs by external stimuli. The patient's past history revealed that most of the attacks were as such and that KLS was suspected 5 years before on his initial admission to our clinic but the diagnosis could not be supported for he failed to take a polisomnography test. PSG performed during hypersomnia period revealed increased sleep activity, shortening of sleep latency and reduction in phase III percentage consistent with KLS. The EEG was normal. Encephalomalasia in left parietal region of brain was detected on MRI. The treatment was continued with lithium 600 mg/day, lamotrigine 400 mg/day and aripiprazole 30 mg/day. After 13 days of excessive sleeping, all the symptoms improved and the patient was discharged.
In the current case, the overlapping symptoms of BD and KLS led to the complicated history and the delay in the treatment. The attacks were in two forms according to the history of the patient. In the first, insomnia, hypersexuality, disorganized behaviors were followed by hypersomnia periods. In the second form, symptoms of mania were evident without hypersomnia. The overlapping of KLS and mania symptoms and the occurrence of pure mania periods led to a delay in diagnosis for 15 years. There are 3 case reports on co-occurrence of BD and KLS in the literature. Due to the heterogenity of BD symptoms, it is challanging to differentiate KLS co-occurring with BD as in the current case. The timely diagnosis of KLS is important to initiate the appropriate treatment and have a beter prognosis. Immune-inflammatory etiology and genetic vulnerability is proposed for the two diseases. Moreover, detorioration of circadian rhythm is well known in BD and defined in KLS. Although BD and KLS have common clinical characteristcs the relationship of the two diseases is not clear. KLS is often misdiagnosed. In KLS and BD co-occurrence the diagnosis can be delayed and the prognosis can be negatively affected.
Keywords: bipolar disorder, hyperphagia, hypersexualit, hypersomnia, Kleine-Levine syndrome

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[Abstract:0729][Schizophrenia and other psychotic disorders]

Alternative psychosis occurrence after treatment with sodium valproate: a case report

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Following seizure control with antiepileptic drugs and normalization of electroencephalogram, behavioral problem may appear for the first time in an epileptic patient. This phenomenon has been termed 'alternative psychosis'. In 1953, Landolt described a group of patients with poorly controlled epilepsy who had psychotic episodes associated with remission of their seizures and disappearance of epileptiform activity on their EEGs. He called this phenomenon "forced normalization". Since then, neurologists and psychiatrists have been intrigued by this phenomenon, and although it has been also reported by others, its existence continues to be the source of much debate.
Case: A 42 year-old female patient, epileptic without psychiatrical background, who presented psychotic episodes after started sodium valproate. She has epilepsy characterized by focal generalize tonic-clonic seizures since one year-old. She was being treated with valproate (1500 mg/day) for the last ten years. After valproate was started, her epilepsy had been adequately controlled. She had an irritable mood, with thoughts preoccupied about his family's behavior, delusions, hallucinations, formal thought or perceptual disturbances since valproate was started. Psychotic seizure has occurred once in 10 to 25 days, taken for 5 to 30 min and her behavior remained normal outside of the psychotic seizure. She had consulted a neurologist, and this situation was considered to be valproate related. 24 hr Continuous Electroencephalogram (EEG) revealed slowing but no seizure activity. We prescribed risperidone 1 mg twice a day to the patient, while valproate was continued. She was asymptomatic after 1 month of starting the antipsychotic.
Alternative psychosis is an ill-defined entity. Although the neurophysiological basis for the phenomenon has not been fully elucidated, Wolf has proposed that it represents ongoing subcortical or mesial temporal epileptic activity with enhanced cortical inhibition. Role of kindling phenomenon and various neurotransmitters has also been implicated. Although non-specific aggressive behavior has not been listed as one of the criteria, this patient's clinical course does support the possibility of alternative psychosis, as the psychiatric syndrome clearly emerged after the introduction of an antiepileptic with good seizure control. Almost all anticonvulsants including valproate have been anecdotally reported in literature to induce the phenomenon. So, it probably has more to do with seizure control rather than the drug chosen to control seizures.
Keywords: alternative psychosis, sodium valproate, antipsychotic drugs

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[Abstract:0732][Others]

Synthetic cannabinoid use associated with acute kidney injury and rhabdomyolysis

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The use of synthetic cannabinoids is associated with many severe adverse effects. We report a case of a patient who developed acute kidney injury, fever and rhabdomyolysis after use of synthetic cannabinoid combined olanzapin and haloperidol. Acute kidney injury is the abrupt loss of kidney function, resulting retention of urea and the other nitrogenous waste products and in the dysreglation of volume and electrolytes. Rhabdomyolysis is characterized by muscle necrosis and increased muscle cell content in blood test.
Case: A 30 year-old male patient who had hallucinations excitation and aggresion was injected haloperidol 5 mg and biperiden 5 mg at the emergency service and had olanzapin 10 mg. After these medications we detected acute kidney injury and rhabdomyolysis indications and fever. We are reporting this case of patient for discussion to apply antipsychotic drugs for aggresion and excitation before other options.
Keywords: synthetic cannabinoids, kidney injury, rhabdomyolysis

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[Abstract:0733][Eating disorders]

Dissociation and disordered eating after sexual abuse: a case report

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Impulsivity, obsessive-compulsive characteristics, affect dysregulation, dissociation, self-criticizing cognitive style and need for control were identified as potential factors involved in the association between self-injurious behaviors (SIB) and eating disorders. However several studies have shown that physical and/or sexual abuse may lead to the development of disordered eating behavior later in life. Here, we present the association between dissociation and disordered eating with a case after sexual abuse.
Case: A 33 year-old female patient married, with two son patient presented with suicidal thoughts, of one year duration characterized by anhedonia, unhappiness, overeating, fainting, sleep disorders, ideas of reference, forgetfulness, nervousness, social withdrawal and sexual aversion. For the last week, images of stabbing her husband and children was coming into the eyes. At the end she tried to throw herself from the balcony the day before then she was admitted to hospital. There were no manic features or organic illness. In her family history no psychiatric disorder has been identified. Her complete blood count, renal function, thyroid function and liver function tests, electrocardiogram and cranial nuclear magnetic resonance imaging were made and all were normal. In her examination hypertension and hypothyroidism was determined. As the medical treatment, paroxetine for depressive mood, topiramate for binge eating episodes and quetiapine for anxiety during the day were started. During the clinical follow ups; the patient had attacks with shortness of breath and palpitations within the day. Where upon in interviews with patients, she said that bloody penis images came to in the front of her eyes. This pseudohallucinations occurred when she saw men. She was exposed to sexual abuse by the husband of her husband' sister for the last five years and in the last year she was forced to sexual intercourse with others for money. She pointed out that her complaints increased after this event. Regular supportive therapy sessions were made beside the medication. Patient's family were informed about being supportive. In the result, the patient's pseudohallucinations decreased, no depressive mood and episodes of binge eating were observed and there was no suicidal thoughts. She lost weight approximately ten kilograms during the seventy-seven days of hospitalization. The patient was discharged into clinical favor with the recommendation of supportive psychotherapy.

Dissociative symptoms were particularly frequent in patients who reported child and adult sexual abuse. A correlation between multiple forms of abuse and higher dissociation scores was only partially upheld in some reports. Although eating disorders are one of the most common psychiatric disorders among women, underlying causes are less known. On the other hand, adult sexual trauma has been suggested to be a risk factor for disordered eating. Our case highlights the relationship between dissociation and disordered eating following sexual abuse.
Keywords: disordered eating, dissociation, sexual abuse, sexual trauma

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[Abstract:0738][Mood disorders]

The mania case due to subarachnoid hemorrhage, with a history of antidepressant usage

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Subarachnoid hemorrhage usually occurs following the rupture of arterial aneurysm and its hemorrhage to subarachnoid space. It forms 7% of all stroke events. Post-stroke disorders are vascular dementia, depression, mania, anxiety disorders, psychotic disorders, apathy, pathological convulsions, crying episodes, and pseudobulbar affect. In this report, we will present a case of post-stroke mania which is rarely observed than depression.
Case: A 23 year-old male patient, single. One year ago, he was treated with venlafaxine 150 mg/day, because of the loss of concentration and symptoms of social anxiety disorder. He reported that he benefited from this treatment for the last 6 months. During his antidepressant treatment, he had a motorbike accident and Computerized Tomography (CT) revealed a subarachnoid hemorrhage of nearly 2 cm in right frontotemporal area. Within two days following the head injury several manic symptoms such as euphoria, spending too much money, insomnia, being more talkative, hypersexuality, and hyperactivity were observed. His baseline score of Young Mania Rating Scale (YMRS) was 43 during his admission at psychiatry department. His biochemical test results and electroencephalography report were normal. No aneurysm was detected on Digital Subtraction Angiography (DSA). For manic symptoms, he was administered haloperidol 2.5-10 mg/day, sodium valproate + valproic acid 500-1000 mg/day, clonazepam 1-3 mg/day. On the third week of treatment, the scores of YMRS were decreased to 8, and the size of hematoma in right frontotemporal area was reduced in Magnetic Resonance Imaging (MRI).
The manic episode may be associated with increased activity in a frontal cortical–subcortical neural system that comprise the anterior cingulate and caudate region. These episodes can also occur due to organic dysfunctions which are considered secondary mania in patients with no history of affective disorder. It has been suggested that most focal lesions associated with secondary mania involve the diencephalic region of brain and most of them on the right side. It could be associated with biochemical changes caused by right hemisphere lesion, which increases the level of brain serotonin. Mania can occur in the acute phase of stroke but it may also occur after until 2 years. In fact, a large part of the mania cases appears in the first days after stroke. In our case, the manic symptoms has emerged immediately after subarachnoid hemorrhage.As a resul t, causes of secondary mania should keep in mind and it should be known that signs and symptoms related Mood disorders should hide the underlying symptoms of a stroke.
Keywords: secondary mania, stroke, subarachnoid hemorrhage

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[Abstract:0740][Substance-related and addictive disorders]

Is an alcohol dependent patient just only an alcohol dependent patient?

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Alcohol use disorders apply to general or psychiatric emergency service with many different clinical states particularly intoxication and withdrawal syndrome. Many medical conditions include metabolic, traumatic, neurological or infectious associated with alcohol or not could be

overlooked in these patients. We aimed to present two cases who were chronic alcohol users and were evaluated in our psychiatric emergency service. Here they were assumed to have psychiatric disorders but they actually had cerebrovascular disease and subdural hematoma.
Case 1: A 32 year-old male patient who had a history of heavy alcohol and substance use presented to the general emergency service with complaints of weakness in left arm and leg started 18 hours ago. Cranial BT, ECG and blood tests were evaluated as normal. He has referred to our psychiatric emergency service with prediagnosis of alcohol withdrawal syndrome. When he was brought he had somnolence, his orientation and cooperation was impaired and he weren't able to speak. Vital signs were normal. Blood tests were normal. Thiamine and IV fluid replacement was started immediately. Hospitalization to Alcohol and Drug Addicts Treatment and Research Center (AMATEM) of our hospital were planned. Afterwards his eyes were spontaneously open, cooperation was sufficient, but still he was not able to speak. His clinic could not be explained with the classical alcohol withdrawal syndrome. He was crying and getting agitated, meanwhile it was noticed he could not move his left arm. It was thought to be pathologies related to the central nervous system. Detailed neurological examination was failed due to inability of cooperation but TCR was positive in left. He was consulted to neurology clinic, cranial CT was taken and he was admitted to neurological intensive care unit upon detection of ischemic stroke.
Case 2: A 41 year-old female patient who had a seizure at the hospital's garden. Then she was taken to the psychiatric emergency service. Her blood pressure: 180/90 mmhg, pulse: 90/min, blood glucose concentration: 130 mg/dL, temperature was normal. She was conscious, oriented. Blood tests and urine analysis was normal. There was no history of epilepsy or any other disease. She had a history of alcohol abuse for 3 years and she had used the last 12 hours ago. Thiamine and iv fluid replacement was started. Hospitalization to AMATEM was planned. There was no history of delirium tremens or seizure. It learned she had marital problems and suspicious domestic violence. She was consulted to neurology clinic to rule out other causes of seizures. Cranial CT and EEG were taken and the patient was referred to neurosurgery upon detection of subdural hematoma.
The reasons of the references to general and psychiatric emergency services of patients with alcohol use disorders may vary. If these patients assessed only diagnosis of alcohol use disorder and just focus on this diagnosis, many vital medical conditions may be overlooked. It would be wrong to relate alcohol user's every clinical problem only to their alcohol usage. Organicity should also be considered. Therefore, differential diagnosis and first medical intervention in these patients can be life-saving.
Keywords: alcohol dependency, differential diagnosis, psychiatric emergency

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[Abstract:0741][Anxiety disorders]

Nocturnal panic and treatment with escitalopram: a case report

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Panic disorder is characterized by recurrent catastrophic fear containing various somatic, physiological, and cognitive symptoms. The prevalence of panic disorder is estimated 4% and selective serotonin reuptake inhibitors use commonly for the treatment. Here, we report here a case of nocturnal panic attacks without daily panic attacks in a middle age female patient who treated well with escitalopram.
Case: A 42 year-old female presented to outpatient clinic of psychiatry. She reported that during sleep she suddenly gets up with a lot of fear with severe symptoms including palpitations, pounding heart and accelerated heart rate, sweating, trembling, choking, chest pain and fear of losing control. The episodes typically peaked within 5 minutes and lasted after 30 minutes in this order she went to outside for fresh air and quickly reached ward of emergency each time. She had been suffering two panic attacks peer week during sleep for six months. Her psychical examination and laboratory exams were all normal. The main presentation was intense fear about panic attack during the sleep over again. Furthermore, she arranged her daily life via avoidance from sleep. The patient was diagnosed with nocturnal panic and was given psychoeducation about the formation of this disorder. Treatment with escitalopram 10 mg/day was used, then the symptoms of patient and life quality showed improvement so the frequency of panic attacks was reduced once a month. At the dose of 20 mg/day, severity of the symptoms was decreased and she started to have short-term, mild and long-range panic attacks within a few months. The patient has been free of any panic attacks in six months follow-up.
Panic attacks, a hallmark of the diagnosis of PD, can occur in unexpected or expected situations and include an abrupt surge of intense fear and discomfort with the physiological symptoms such as palpitations, pounding heart, accelerated heart rate, sweating, trembling or shaking, sensations of shortness of breath or smothering, feeling of choking, chest pain or discomfort, nausea or abdominal distress, feeling dizzy, unsteady, lightheaded, or faint, chills or heat sensations, paresthesias, derealization or depersonalization. PA might be classified to respiratory, nocturnal, non-fearful, cognitive, or vestibular subtypes. However, nocturnal panic is a common condition in

patients with panic disorder that is defined by waking from sleep in a situation like panic attack. Also it is suggested that the NP might be associated with a history of traumatic events. NP appears in non-REM sleep and it exhibits dissimilarities from sleep terrors, sleep apnea, nightmares or dream-induced arousals. In this case, escitalopram was effective in containment the severity and frequency of NPA. It is demonstrated that the peripheral sympathetic arousal is increased, the function of α-adrenergic receptors is abnormal, the sensitivity of presynaptic and postsynaptic central α2-adrenergic receptors are decayed in panic disorder. Furthermore in NPA an autonomic nervous system dysfunction has been blamed conversely daily PA is associated with psychological and cognitive factors. But the etiology of the fear of dying or losing the control in panic disorder remains still unclear.

Keywords: escitalopram, nocturnal panic, panic disorder

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[Abstract:0742][ADHD]

Effects of methylphenidate and atomoxetine on encopresis; case reports

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Encopresis is a common comorbid psychiatric condition in children with attention deficit and hyperactivity disorder (ADHD). Some hypotheses have been proposed about the relationship between ADHD and encopresis. One of this is that inattentive/impulsive children are less able to recognize and respond to internal cues to defecate, which causes encopresis. Methylphenidate and atomoxetine were observed to be effective in the treatment of children with encopresis and ADHD. This paper presents children with ADHD and coexisting encopresis who displayed improvement in ADHD and encopresis symptoms with ADHD treatment.

Cases: The case reports consisted of 8 children and adolescents (6 boys and 2 girls) with encopresis and coexisting ADHD, 6-14 years of age. The demographic, clinical characteristics and treatment responses according to Clinical Global Impression-Improvement (CGI-I) were shown in Table 1.

Several mechanisms can be responsible for therapeutic effects of MPH or ATX in children with encopresis. Anti-encopretic effects of these drugs may be related to the direct impact on executive functioning which enable children to recognize and respond to internal cues to defecate or the positive effects of ADHD drugs may be secondary to the resolution of child-parent relationship conflicts. It has been suggested that coexisting conduct problems may be a vulnerability factor for the severity of encopresis, and they seem to be associated with the success of the LA-MPH treatment for encopresis in children and adolescents with ADHD. It was remarkable that the majority of our cases had ODD comorbidity. MPH may also have direct peripheral effects on gastrointestinal system (GIS) motility. There is no study investigating the peripheral effects of MPH/ATX on GIS. However, it is shown that amphetamine, which has similar effects as MPH was found to reduce the gastric emptying and intestinal motility via the D1 and D2 receptors in rats. Further studies are needed to clarify mechanisms of therapeutic effects of MPH and ATX in children and adolescents with encopresis.

Keywords: ADHD, encopresis, methylphenidate, atomoxetine

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[Abstract:0744][Others]

Unclassified persistant visual phenomen (visual snow) in an adolescent case

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Visual snow (VS) belongs to the group of persistent positive visual phenomena connected persistend migraine auras, but it can be seen in persons who do not have migraines. Patients with so-called 'visual snow' describe a persistent disturbance in the entire visual field looks like the 'static' or 'snow' of a badly-tuned analogue television. The normal findings on routine ophthalmological and neurological tests have led to

the condition being either dismissed as psychological or wrongly classified as migraine aura. Here, we present an adolescent patient with VS.
Case: The female adolescent patient was consulted to us by child neurologist at August 2015. Her visual symptoms started acutely at October 2014. The patient consistently described 'dotty' or 'pixelated' vision affecting the entire visual field of both eyes equally. She had continuous VS, 24/7 with eyes closed and open, independent of the outside light level, without periods of remission. The persistent visual phenomena developed without any associated migraine events. Basic blood tests were normal except for a low ferritin level. Patient had normal ophthalmic and neurologic examinations. Firstly, we thought that her symptoms may result in conversion disorder. Faradic current treatment (max.7 mA /10 min.) once a day was planned and administered five seans for the patient but this treatment had no effect on the persistent positive visual phenomena. Her visiual complaint persisted.
Patients with VS suffer significantly from being diagnosed as malingerers or as having a psychogenic disorder. This is mainly due to the inability to demonstrate pathologic findings in routine neurological and ophthalmological exams with also unremarkable brain imaging results like as our patient. Our patient have solely VS in contrast to typical migraine aura is very common in patients with VS. In one patient cohort study stated that VS and persistence of after-images are reported with neither ophthalmic nor neurological disease. All patients had normal ocular and neurological examinations in their case reports. Differential diagnosis must be done between visual hallucinations and VS. Our patient had not an established psychotic disorder, and usually in this setting visual hallucinations are more complex with occasional auditory component positive visual phenomen. VS also has been associated with the use of recreational drugs. However, our patient has not used any drug and she has not any psychotic symptoms. In one retrospective case series has demonstrated that neither migraine nor recreational drug use are necessary associations with symptoms of VS. Psychiatric evaluation and diferential diagnosis of pediatric VS cases are important to prevent incorrect medical processes and its complications. They clearly demonstrate the problem is unlikely to be merely "psychological" as has been suggested. The presence of this disturbance in children and its remarkably stereotypic phenotype, which seems clearly distinct from migraine aura, suggest the problem should be defined in terms of ethiopatogenesis. Understanding the VS syndrome will be crucial for advancing its management and will contribute to broader understanding of visual modulation. In our opinion, this rare visual phenomenon merits future study to diminish the burden of VS.
Keywords: adolescent, persistent visual phenomena, visual snow

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[Abstract:0745][Tic disorders]

Severe and treatment-resistant Tourette Syndrome that recovered with topiramate use: a case report

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Tourette Syndrome (TS), which is common in childhood, is a neurodevelopmental disorder characterized by long term motor and vocal tics and its etiology is not fully clarified yet. In the treatment of TS, antidopaminergic drugs alpha adrenergic agonists (clonidine), GABA modulators, very rarely antiepileptics (topiramate) and behaviorist approaches are recommended. Studies have found topiramate to be effective and safe in moderate TS cases and to cause obvious recovery in school performance. However, there are almost no studies on the use of topiramate in treatment resistant TS cases. We are presenting this case in order to increase awareness and to contribute to resistant TS treatment.
Case: A 10 year-old male patient was brought to our outpatient clinicdue to involuntary spitting behavior which started about three months ago and resulting anxiety, uneasiness, behavior problems, functionality disorder and social introversion. In addition, it was learned that vocal tics were added to motor tics which started on the shoulders and face 18 months ago and he was not using any medication. Considering TS, ADHD, behavioral disorder (BD) and social anxiety disorder (SAD), the patient was given fluoxetine 20 mg/day, risperidone 1 mg/day at first, then 3 mg/day. Since no clinical changes occurred, aripiprazole 2.5 mg at first, then 15 mg/day was added to the treatment and it was used for four weeks. Since there was no change in the follow-up, haloperidol drop was given with the other treatment for two weeks between 1 to 5 mg. In psychiatric interviews, no change was seen in the severity of the disease according to Clinical Global Impressions Scale (CGI) and according to Yale Global Tic Severity Scale (YGTSS) all tic and functionality disorder scores were at baseline values. Following a combined treatment of aripiprazole 15 mg/day, topiramate 150 mg/day and fluoxetine 20 mg/day, there was an obvious recovery in the patient's CGI and YGTTSS values. TS symptoms including spitting are not seen and there is an obvious increase in social functionality and school success.

Severe and treatment resistant TS in children and adults cause an obvious disruption in the functionality of the patient and also in parents' coping and family functionality levels. This situation makes urgent treatment an obligation. Although clonidine is known to be effective in TS cases with sleep and behavior problems, the fact that it is not sold in our country limits our treatment choices. Despite the combined use of effective and high dose risperidone, aripiprazole and haloperidol for a sufficient period of time, not getting any recovery in TS symptoms is bad. The presence of studies in literature which show the efficiency and reliability of topiramate in moderate TS encouraged use to use it in the treatment. Our case can present awareness for considering topiramate as an effective choice in resistant TS cases and it can create a stimulus for future studies on the subject.

Keywords: tourette syndrome, topiramate, treatment

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[Abstract:0748][ADHD]

Bruxism and halitosis comorbidity that developed due to atomoxetine use: case reports

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Bruxism can be associated with anxiety, benzodiazepines, alcohol consumption or selective serotonin reuptake inhibitors (SSRI). Dopamine agonist agents may cause bruxism or increase existing symptoms. It may develop as a result of halitosis, mouth, respiratory and digestive system diseases, infections, metabolic conditions and drugs such as amphetamine. Headache, abdominal pain, appetite loss, asthenia, nausea and throwing up are the most common effects of atomoxetine use in the treatment of ADHD and they are generally temporary. In this case report, our purpose was to present two cases that developed bruxism and halitosis synchronously and draw attention to a situation that was not reported in literature before.

Cases: Both cases were 9 and 10 year-old male patients, respectively, presented with similar complaints such as attention deficit and not being able to maintain attention, hyperactivity and decrease in school success which had been going on for more than a year but which had recently increased. According to DSM-IV diagnosis criteria, case 1 was diagnosed with predominantly hyperactive-impulsive type ADHD, and case 2 was diagnosed with combination type ADHD and they were not found to have comorbid psychopathology. 18 mg/day atomoxetine which was given to each case for 10 days was increased to 25 mg/day. New and suddenly appearing complaints of bruxism and halitosis started in case 1 after 2 weeks and after 10 days in case 2 following the new dose. Ibuprofen was given to case 1 for gnathalgia. Due to obvious halitosis, social functioning was disrupted in both patients. The possibility of atomoxetine developing adverse effects was assessed with Naranjo Scale for Adverse Drug Reactions and it was thought that bruxism and halitosis were associated with atomoxetine use. Atomoxetine treatment was stopped, the symptoms of bruxism and halitosis decreased and disappeared completely in a week. Both cases and their parents did not want to use atomoxetine again and methylphenidate treatment was started instead.

There is no reliable data about how long the onset of drug-induced bruxism is; however, it has been reported that it may develop 6 hours to 11 months after the treatment is started. Similarly, case 1 developed bruxism and halitosis symptoms 4 weeks later and case 2 developed the symptoms 3 weeks later. Atomoxetine can suppress the dopamine carrier proteins and cause bruxism as a result of dopaminergic activity. Further studies are required to research which system atomoxetine affects to cause halitosis. To our knowledge, there are no reports of bruxism and halitosis comorbidity due to atomoxetine use and no reports of atomoxetine causing halitosis. These two cases reported here may increase awareness that patients receiving atomoxetine treatment due to ADHD may develop a comorbidity of bruxism and halitosis.

Keywords: bruxism, atomoxetine, halitosis

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[Abstract:0750][Mood disorders]

Alternatives of treatment combinaitons in early onset bipolar disorder: 3 case reports

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Bipolar Disorder (BD) is a chronic, repetitive and serious psychopathology that emerges with at least one hipomanic, manic or mixed episode, which can cause short term and long term morbidities and mortality in all ages. Lifetime prevelance of BD in adolescents is shown as 1%. In 55% of adult BD, onset of symptoms occurs in childhood and adolescence. Discussions about early-onset BD (EOBD) focus on determining diagnostic criteria, principles of differential diagnosis, management of treatment. Clinic profile of EOBD is quite different then classical deppressive and manic manifestations of adult BD, which usually consists of separate and distinct episodes. EOBD, especially in prepubertal period, commonly emerges with symptoms of mixed episode and manifests with rapid cycling features. In this case report we aimed to present three adolescent patients with EOBD in terms of their symptom manifetations and treatment schemas of combination of mood stabilizers and alternative antipsychotic options.

Case 1: A 16 year-old girl with ADHD. She has been suffering from mixed episodes of BD for 1.5 years. She did not benefit from valproic acid(VA), risperidone, aripiprazole, haloperidol; had partial improvement in symptoms by adding lithium. She was charged to inpatient clinic. She did not remit with effective doses of valproic acid, lithium and olanzapine. She has remitted with 200 mg clozapine and effective doses of lithium and valproic acid. Grandiosity, excessive make up, exaggerated seductive dressing, frequent changes in partners, anhedonia, anergy, irritability, hostility have diminished subsequently. Soon after discharge, other antipsychotics were ceased, clozapine were continued.

Case 2: A 16 year-old boy who presented to psychiatry clinic with depressive symptoms when he was 8 year-old. He hospitalized in a private clinic with the diagnose of manic episode with psychotic features. Although he did not respond to risperidone and olanzapine treatment, his symptoms improved with 7 sessions of electroconvulsive treatment. Due to his low compliance of treatment in recent months he developped a new manic episode with psychotic symptoms. Then he presented to our clinic. The patient had benefit from the combination of risperidone and lithium. He had discharged in complete remission. Then he was followed with depot intramuscular injection form of risperidone on the purpose of treatment compliance.

Case 3: A 17 year-old girl diagnosed with BD for 3 years. Aripiprazole was discontinued because of intense tremor. Olanzapine was ceased because of excessive weight gain. With lithium treatment for the last 2 years, irritability, excessive self esteem, extravagancy have diminished significantly. In a depressive episode with anxiety she had benefit from escitalopram. The patient whose symptoms have exaggerated for the last 6 months had no improvement with valproic acid. With complaints of affective storms and frusturation intolerance she was hospitalized. She had remission with 550 mg of quetiapine and lithium and discharged in remission.

Although common guidelines state monotheraphy as first step in treatment of BD, it is known that most of the cases requires combinations of mood stabilizers and antipsychotics. In this presentation we discussed alternative treatment options.

Keywords: bipolar disorder, antipsychotic, combination treatments, mood stabiliziers

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[Abstract:0755][Anxiety disorders]

A case of emesis in a patient with generalized anxiety disorder who responded well to mirtazapine treatment

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Mirtazapine's antiemetic effect may be explained due to its antagonism of 5-HT3 receptors. There are reports showing the efficacy of mirtazapine as an effective antiemetic agent in adult patients related to chemotherapy-related nausea and vomiting, treatment-resistant hyperemesis gravidarum and postoperative nausea and vomiting. But, cases of primary psychiatric conditions for that matter are still lacking. We are reporting here a case of nausea and vomiting with Generalized Anxiety Disorder (GAD) who responded well to mirtazapine.

Case: A 23 year-old male patient, single, was referred to our psychiatry department with symptoms of nausea and vomiting over the past 3 months from Gastroenterology department. No underlying medical causes was found that might be attributable to his nausea and vomiting according to the report from the of gastroenterologist and neurologist. His previous medical examination was all within normal limits. He had been suffering from anxiety, restlessness, sleep disturbance, decreased attention with from time to time agitation, for about 12 months. When the patient was interviewed, the symptoms of anxiety were present, and according to DSM-IV-TR, the diagnosis of GAD was made. Profound history and interview did not find any evidence of eating disorders, or possible related severe psychiatric disorders as well as his symptoms were only relevant to GAD. The patient was consulted by the experienced internal specialists. Based on their comments, the patient was hospitalized due to the lack of food with poor oral feding and fluid replacement therapy was started. Firstly, we started on lorazepam 2.5 mg/day with mirtazapine 15 mg at bedtime and mirtazapine was gradually increased to 30 mg. After 1 week of mirtazapine, the nausea and vomiting had reduced, and over a period of 3 weeks, the vomiting had stopped. After 7 days of treatment with 30 mg/day of mirtazapine, the nausea and vomiting were dramatically reduced and on hospital day 10, he was completely free of nausea, and vomiting. Subsequently, lorazepam was gradually stopped in two weeks and he was discharged. Our patient was followed on a regular basis, in psychiatry outpatient department during 6 months. At the follow-up interview, he said that when he stopped to using mirtazapine, his complaints was immediately recurred. He was very pleased for his treatment.

Compared to the SSRIs, mirtazapine is not associated with nausea or sexual dysfunction. In a previous report, 3 patients with major depressive disorder who experienced nausea and vomitting showed complete remission by mirtazapine use. Mirtazapine was generally tolerated well in these subjects. As a result, the presence of comorbid psychiatric and medical conditions would guides drug therapy selection. As in our case, the conclusions would have been further confirmed, when using mirtazapine had been stopped a few days to see if nausea would have recurred. In our opinion, a timely psychiatric consultation may help to identify and treat possible psychiatric disorders. In line with, mirtazapine could be a treatment option in these subjects particularly in the presence of anxiety and depressive disorders.

Keywords: generalized anxiety disorder, mirtazapine, nausea, emesis

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[Abstract:0756][Schizophrenia and other psychotic disorders]

Indomethacin-induced psychotic disorder

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Several NSAIDs are known to have neuropsychiatric adverse drug reactions as confusion and seizures but psychosis is quite rare. Although there have been 3 case reports in the literature of indomethacin-induced psychosis, to our knowledge, this is the first report that describes "Substance or Medication Induced Psychotic Disorder" according to DSM-5 caused by indomethacin assosiation with Schizophrenia Spectrum and Other Psychotic Disorders.

Case: A 45 year-old male patient, referred to our Physical Medicine and Rehabilitation outpatient clinic from a family practice with neck, low back, and left ankle pain for one year. He also described a morning stiffness in his lower back and hips, especially in the early morning hours. The medical history and laboratory evaluations was consistent with AS. Magnetic resonance imaging (MRI), and X-ray studies of the spine, which showed characteristic spinal changes and inflammation of the sacroiliac joint. His medical examination revealed ankylosing spondylitis. Sulphasalazine 1 g/day and indomethacin 150 mg/day was started but he came three weeks later for first visit and he said, used only indomethacin as a causeless. He was markedly alert, irritable, isolative, suspicious, and found talking excessively and irrelevantly, talking to himself. He had visual and auditory hallucinations (hearing voices conversing with one another), religious delusion. Prior to this event, There was no personal or family history of psychiatric illness, neurologic disturbance, or substance use before the admission. Neurological examination and laboratory results was found to be normal. MRI brain was obtained to rule out tumor, infection or any vascular infarct. We considered a tentative diagnosis of drug-induced acute psychosis. He was referred to psychiatry clinic. We first stopped indomethacin and the patient was kept under observation. The patient was started on risperidone to address his psychotic symptoms, rapidly up-titrating from 1 to 2 mg. By treatment day 14, the patient began to refuse his previous delusional thoughts and demonstrated improved insight, understanding that his delusions were caused by indomethacin use. After 8 weeks, the patient's psychotic symptoms completely improved. Follow-up visits made periodically during 6 months revealed no recurrence of symptoms.

Many studies showed evidence for the impairment of AKT/GSK-3β signaling pathway in schizophrenia. Studies reported convergent

evidence for a decrease in AKT1 protein levels and levels of phosphorylation of GSK-3β in the peripheral lymphocytes and brains of individuals with schizophrenia. Activated Akt/GSK-3β is the important signaling molecule involved in regulating cell metabolism, proliferation, apoptosis, survival. Akt regulates the activities of transcription factors such as Forkhead box class O (FOXO), mTOR, NFkB, and MDM2. NSAIDs as Celecoxim and indomethacin, and glucocorticoids have been reported to inhibit PI3K/Akt signaling in several somatic cell lines. In this case, inhibition can be demonstrated on brain of patient with induce psychosis by Indomethacin. We suggest that indomethacin changes brain cell cycles, at least partially, through inactivating Akt. Further research is required to clarify the mechanism of induced psychosis.

Keywords: idomethacin, psychotic disorder, risperidone

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[Abstract:0757][Substance-related and addictive disorders]

Methanol poisoning and central scotoma: a case report

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Methyl alcohol is a toxic liquid. It is used for the manufacturing of many products in the industry such as dye, varnish and defrosting solutions. We intended to present a case related to central scotoma that develops after drinking cologne.

Case: A 33 year-old male patient, elementary school graduate, was working in bag manufacturing. He is married. The patient, who consumed five beers every day for the last 20 years, drank three bottles of cologne before five days and two days after his complaints such as seeing images, confusing time and place, clouding of consciousness and loss of vision began and he was referred to us. The patient, who had complaint of loss of vision, was consulted to ophthalmologist. The diagnosis of central scotoma was considered for the patient. Bilateral pupillary was mid-dilated and his bilateral direct and indirect reflex was observed to be weak. Optic disk boundaries were observed to be slightly obscure. The control of patient under outpatient clinic conditions was recommended. The patient was hospitalized to the alcohol department of our hospital due to the risk of delirium tremens. The patient, whose panic disorder started twenty years ago, started to drink alcohol for self-medication. He was followed up with the outpatient treatment of alprazolam 1 mg/day, paroxetine 10 mg/day treatment for fifteen years. Relapse occurred seventeen days after the patient, who was hospitalized in alcohol addiction treatment department of our hospital a month ago, was discharged. He was hospitalized again in our hospital a month later due to the risk of delirium tremens. The history of patient included intracranial haemorrhage and hip prosthesis history resulting from traffic accident under the influence of alcohol and glaucoma. The patient, who had abstinence symptoms of sweating, visual hallucination, time orientation disorder, was started to be treated with diazepam 60 mg/day and B1 vitamin replacement. Abstinence symptoms were followed up and diazepam treatment was reduced and stopped in 10 days. Acamprosate333 mg 3 tablets BID was added to his treatment. Paroxetine 20 mg/day was added to the treatment of patient, who had panic disorder and depressive complaints. After his inpatient treatment was completed, ophthalmic outpatient cliniccontrol was recommended and he was discharged.

Methyl alcohol is used for manufacturing formaldehyde and illegal alcohol. Poisoning may result in acidosis, blindness at various degrees, wide and non-reactive pupillary, retinal oedema, renal, liver and heart damage and brain oedema. Delirium and coma may emerge in case of high amounts of intake. Severe irrecoverable loss of vision and cerebral damage are permanent. It emerges with the deliberate or unintentional consumption of (cologne, home-made alcohol or illegal spirits) instead of genuine alcohol. Methyl alcohol is non-toxic until its toxic metabolites are converted into formaldehyde and formic acid. These metabolites and especially formic acid are responsible for clinical findings. Since it is easy and cheap to obtain, methanol is used for manufacturing illegal spirits in the world. Methanol poisoning by oral route is a serious problem in our country and causes high morbidity and mortality.

Keywords: central skotoma, delirium tremens, methanol

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[Abstract:0759][Autism]

Premenstrual exacerbation of schizoprenia symptoms in a child with autism spectrum disorder: a case report

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Autism spectrum disorders are often comorbid with other psychiatric disorders such as anxiety, Mood disorders, ADHD, schizophrenia. Schizophrenia and autism are chronic psychiatric disorders which share similar symptoms in social functioning and there is a clinical and genetic overlap between two disorder,but the underlying mechanism has not been fully determined. Current proposals for the diagnosis of schizophrenia allow for co-diagnosis of schizophrenia and of ASD. When presence of ASD, the diagnosis of schizophrenia requires prominent hallucinations or delusions. However determining whether the symptoms belong to autistic spectrum or result from comorbidity is mostly challenging because of common features such as social withdrawal, communication impairment, poor eye contact. Moreover any medical condition which affect the hormonal balance may confuse the diagnosis, for instance in female patients diagnosed with schizophrenia, psychotic symptoms may be aggrevated prior to menstrual periods. In this case presentation we will present a female patient who was diagnosed with autism spectrum disorder and schizoprenia and whose schizoprenia symptoms are exacerbated with menstruation.
Case: A 14 year-old adolescent female, who was diagnosed with autism spectrum disorder in early childhood. She was admitted to our clinic by her parents because of irritability, anxiety, hostility, hallucinatory behaviors, self harm behavior, thinking other people are working to harm her for a few months. Also her psychotic symptoms were exacerbating with menstrual periods. In the psychiatric examination, she was irritable, eye contact was poor, social interaction was restricted, echolalia was present in speech, hallucinatory behaviors were observed and she had delusions of persecution. Her physical and neurological examination was found as normal. The blood tests showed normal levels. Cranial magnetic resonance imaging was also reported as normal. She diagnosed with ASD and schizophrenia and treated with aripiprazole 15 mg/day. During the clinically follow-up her psychotic symptoms decrased with aripiprazole treatment but recurred each month by the onset of menses.
A careful medical history is crucial in psychiatry. Comorbidity should be evaluated comprehensively Clinicians should also be aware symptom exacerbations linked with menstrual periods in patients with schizoprenia as in this case.
Keywords: autism spectrum disorder, premenstrual dysphoric disorder, schizoprenia

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[Abstract:0760][Schizophrenia and other psychotic disorders]

Cycloid psychoses: a case report and literature review

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The emergence of the concept of cycloid pychoses goes back to the problem of "atypical psychoses" which arose from Kraepelin's dilemma of endogenous psychoses. Some psychoses which could be allocated neither to dementia praecox (schizophrenia) nor to manic-depressive disorder could be referred to as “cycloid psychosis”. Cycloid psychosis is a valid clinical concept that can be identified as boundary conditions in clinical practices. These clinical presentations are characterized by sudden onset and polymorphic symptomatology including delusions, hallucinations in every sensory modality, confusion, akinesia or hyperkinesia. They have a recurring course, rapid remission followed by their premorbid functionality . In this case report we aimed to present 17 year-old male adolescent who had episodic psychotic symptoms without residual finding between attacks and a literature review is shortly surveyed.
Case: A 17 year-old male patient presented to our child psychiatric outpatient clinic with recurrent dizziness, hallucinations of floaters and black spot, insomnia, psychomotor retardation and social withdrawal. He was spending all the time in his bed without sleeping because he felt severely dizzy and imbalanced. The first episode occurred 6 months ago with a similar pattern, accompanied with academic failure and was followed by recovery. There was a recrudescence of the symptomatology when his admission. He had a negative previous

psychiatric history and family history of mental illness. Extensive laboratory investigations, including complete blood count, serum chemistries, serology, cranial magnetic resonance imaging, and electroencephalography, revealed no significant abnormality. Substances abuse and medical conditions were excluded. He recovered on quetiapine treatment. The clinical picture had a rapid improvement in few days with remission. He showed no signs of residual psychosis or mood disorder and was able to return to school life. Currently, the patient is clinically stabilized.

When patients with acute and transient psychotic disorder with an episodic course and full recovery between recurrences presented to psychiatry clinics, the concept of cycloid psychoses should be considered as a differential diagnosis. This diagnosis seems to be ignored in modern psychiatry and in the present nosological constructions. Although the need of more studies to improve the understanding of its etiology and treatment, concept of cycloid psychosis may propose an alternative diagnosis for some psychotic disorders.

Keywords: Cycloid psychoses, adolescent, schizophrenia

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[Abstract:0761][ADHD]

The role of methylphenidate on school refusal: a case report

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School refusal behavior is defined as to be reluctant to go to school that may lead up to school absenteeism and drop out. It seriously impacts functioning of both parents and child in many areas (e.g. social, educational, family, emotional). Although school refusal behavior may occur at any age and socioeconomic status, it usually begins at elementary school. Various factors may precipitate and predispose to school refusal. It is mostly associated with anxiety disorders (especially separation anxiety disorder). However, depression, somatic complaints, disruptive behavior disorders, phobias, adjustment disorders are seen in also.

Case: A 8 year-old 3rd grader male patient referred to our clinic with the complaint of school refusal that started two months ago. In the case of preparing for school, he had suffered from abdominal pain. The patient had school refusal since the first grade but he had gone to school without any treatment. Previous school refusal period had lasted for less than ten days. He had facial paralysis and circumcision at the beginning of third grade. He had received cortisone for the treatment of facial paralysis. He was diagnosed as attention deficit/hyperactivity disorder (ADHD) and separation anxiety disorder based on the diagnostic findings in the KSADS-PL. We started to treat with OROS-Methylphenidate 27 mg/day (0.8 mg/kg/d). At the same time, he started to go to school accompanied by his mother. His mother stayed out of the classroom during one week. Next week, his mother did not wait at school. Two weeks after the methylphenidate treatment, both separation anxiety and ADHD symptoms were decreased.

Treatment of school refusal must include psychopharmacological and psychosocial interventions. Parents and teachers also play a necessary role as a part of treatment. Because of the frequent association between school refusal and anxiety disorders, SSRIs and benzodiazepines are most preferred drugs. But like this case, ADHD should consider at school refusal cases and ADHD symptoms should be examined. ADHD may cause to be bored in classroom, possibility of warning and punishment due to behavioral problems, low academic achievement. These problems may result in anxiety and adjustment difficulties that may induce school refusal. For an effective treatment of school refusal cases with ADHD comorbidity, psychostimulants should be a part of treatment. Further studies with large samples are needed.

Keywords: school refusal, ADHD, methylphenidate

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[Abstract:0762][Psychosomatic medicine-Liaison psychiatry]

Premature ejaculation associated with milnacipran: a case report

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Premature ejaculation (PE) is defined by ejaculation that seen repeatedly shortly before or after about 1 min of vaginal penetration or less or without sexual stimuli. Negative personal thoughts, depressive feelings and anxiety also accompanied by inability to delay ejaculation leading to avoidance to sexual intercourses. Generally, antidepressant and antipsychotics lead to decrease in libido, sexual willingness and delayed ejaculations. In our case, we present a patient who developed premature ejaculation related with milnacipran.
Case: A 35 year-old male patient presented to our outpatient clinic with depressive symptoms. Milnacipran 50 mg/day was initiated. Remission was observed at the 6th week of treatment according to Hamilton Depression Rating Scale (HAM-D) score of 7; however, he declared the PE since starting from the 2nd week of treatment. No any organic reason was detected related with PE according to physical, laboratory examination and urology consultation. Milnacipran treatment was changed with sertraline 50 mg/day. In the 2nd week of sertraline treatment, the PE improved and remission of the depression was going on.
PE is a common male sexual disorder associated with depression or antidepressant treatment. Antidepressant treatment may occasionally cause PE or sometimes an adverse effect of antidepressant drugs. According to our knowledge, this is the first case that milnacipran related PE in the literature. In this condition, SSRIs may be good alternative treatment options. Further studies require demonstrating the mechanism of PE related with milnacipran.
Keywords: milnacipran, premature ejaculation, sertraline

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[Abstract:0763][Schizophrenia and other psychotic disorders]

Menstrual psychosis in an adolescent girl

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Menstrual psychosis has the following characteristics: acute onset, against a background of normality; brief duration, with full recovery; psychotic features: confusion, stupor and mutism, delusions, hallucinations, or a manic syndrome; a circa-mensual periodicity, in rhythm with the menstrual cycle. We present an adolescent female patient who represents two brief psychotic episodes that are start before menstruation and end after menstrual bleeding with comparing our findings with literature.
Case: A 13 years-and-five-months-old female adolescent patient presented to our clinic with the complaint of extrapiramidal side effects following acute and long-acting antipsychotic drug enjections (Zuclopenthixol acetate 50 mg/ml and Zuclopenthixol decanoate 200 mg/ml) on October 19th 2015. She suffered from bradykinesia, bradymimia, hypersalivation, weakness, tremor, flushing, sweating, incontinence, mutism and decreased appetite. Her family reported that her past history involved two acute, relatively short-lasting psychotic episodes associated with menstrual cycle. The first episode, at 19th September, two days before menstruation bleeding, an acute and sudden change occurred in her mental state. A fearful and anxious mood, and a significant behavioral regression was described. She claimed to see the fire on lamppost and television and a a female patient who said to her "your house is on fire". The first episode ended suddenly with the 3rd day of menstrual bleeding on September 24th. In her first episode, she was hospitalized in another hospital, and 50 mg/day sertraline, 5 mg/day aripiprazole and 25 mg/day hydroxyzine HCL treatment were started. She was discharged with full recovery on September 24th. She had remained symptom free for 21 days. The second episode, on October 14th, one day before menstruation bleeding, an acute and sudden change occurred in her mental state again. Her family presented to the special doctor and her treatment was terminated, acute and long-acting antipsychotic drug enjections was started. Finally, the patient with her family presented to our clinic with the complaints, as defined above, on October 19th. The patient hospitalized with the diagnosis of menstrual psychosis on the sixth day of menstrual bleeding. Hormone analysis revealed that low estradiol, FSH and LH levels acording to follicular phase. Biperiden was started, and within 8 days at 6-8 mg/day, her extrapiramidal symptoms decreased markedly. Any psychotic episode was not seen on

consecutive three menstrual cycles without any treatment.
Although the etiology of menstrual psychosis is still unknown, it has been related to fluctuations in the concentration of the female reproductive hormones that are produced throughout the menstrual cycle. The exacerbation of cyclic menstrual psychosis may be synchronized with exceptionally high mistimed peaks of FSH, LH and estradiol that occur at the end of the menstrual cycle. Reproductive hormonal imbalances of our patient might be responsible for her episodes. Menstrual psychosis are not enough mentioned in child and adolescent psychiatry nomenclature, this case presentation could contribute to the increased awareness about menstrual psychosis which is not extremely rare.
Keywords: menstrual psychosis, adolescent, menarche

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[Abstract:0766][PTSD]

Paliperidone extended-release treatment effect on complex posttraumatic stress disorder in an adolescent case

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Childhood trauma almost always leads to many psychiatric disorders. Complex posttraumatic stress disorder (C-PTSD) usually results from prolonged exposure to traumatic event(s) and is characterized by long-lasting problems that affect many aspects of emotional and social functioning. C-PTSD involves affect dysregulation, relational dysfunction, self-mutilation or personality changes. The psychopharmacologic treatment of C-PTSD may consist of antidepressants, antipsychotics or mood stabilizers.
Case: A 16 year-old female patient was referred to our clinic from the local rehabilitation and care center. She and her sister were victims of repeated sexual abuse by their father. Also it was learned that her father murdered his sister. After this murder, she has declared sexual abuse to her mother. She has been living in the local rehabilitation and care center for four years. She suffered from unstable mood, flashbacks, emotional and behavioral avoidance, self-blame, self-mutilation and destructive behaviors. Her diagnosis was compatible with C-PTSD according to mental status examination. She has been using olanzapine 10 mg/day and valproic acid/sodium valproate 1000 mg/day treatment for one year. Her routine laboratory test results have revealed elevation in liver function tests. She was gained 16 kg at this year (69 kg to 85 kg). Because of her increased appetite and weight gain, olanzapine changed to aripiprazole. 15 days after this change, she started to injure herself and damage to center. Then, chlorpromazine is added to treatment (50 mg/day, then 100 mg/day). At second laboratory examination, liver enzymes were also increased. She was consulted to gastroenterology and we stopped her all drug. We started Paliperidone Extended-Release 3 mg/day, then 6 mg/day. Her affect dysregulation and self-mutilation behavior improved with treatment after one month. Also, liver enzymes returned to normal limits and her weight loss was 13 kg (85 kg to 72 kg).
In recent years, antipsychotic use progressively increases at child and adolescents. At this patient group, atypical antipsychotics more preferred because of the low side effect profile. But both atypical antipsychotics and mood stabilizers may lead to weight gain and abnormalities on metabolic parameters. So patients whom use this kind of drugs must be followed up for metabolic side effects. Paliperidone is the major metabolite of risperidone and has fewer side effects compared to risperidone. Moreover, ER formulation of Paliperidone provides stable blood concentration. This case suggests that Paliperidone ER may be an effective alternative in treatment of adolescents with complex PTSD and presents few side effects. Large sample studies are needed for further information.
Keywords: paliperidone, complex posttraumatic stress disorder, adolescent

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[Abstract:0769][ADHD]

Retrograde amnesia, attention deficit and learning disabilities after sudden hypoxic ischemic brain injury secondary to cardiac arrest in a 16 year-old boy: a case report

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Cardiac arrest (CA) is commonly accepted as cause of amnesia, pattern of impairment after CA is generally a combination of memory and motor deficits with variable executive impairment. Neurodevelopmental outcomes of acute hypoxia aren't well understood yet. We report a case of a 16 year-old boy, who had CA soon after consuming energy drink (ED) and exposed to 50 minutes of hypoxia.

Case: A 16 year-old boy had CA with unknown etiology 2 months ago and exposed to 50 minutes of hypoxia before responding to cardiopulmonary ressucitation. Pediatric cardiology associated CA with highly consumption of ED for the last two years and family history of sudden cardiac deaths in 3rd degree relatives in 16 and 32 years of age. Cardiologist suggested psychiatric evaluation for possible suicidal attempt, because of positive TCA level in subsequent two urine analyses soon after CA. He presented to our outpatient clinic for psychiatric evaluation. He had been diagnosed with Attention Deficit and Hyperactivity Disorder (ADHD) when he was 8 year-old but he did not use any psychostimulants. His school notes had graceful degradation along the years. Wechsler intelligence scale for children-revised (WISC-R), Auditory Verbal learning, Bender Gestalt -2, Verbal Fluency, GIST, Stroop tests were performed. Clinical opinion revealed his mental capasity was better than WISCR scores of verbal IQ 54, performance IQ:53, total IQ: 51. The neurocognitive test battery showed that short term memory was normal while he had long term memory and both visuel and auditory learning problems. His visual perception and motor coordination was normal, except a negligible intentional tremor. He has met Diagnostic and Statistical Manual-5 criteria of ADHD, SLD diagnosis was controversial. There was no evidence of suicidal attempt or substance abuse explaining urine TCA positivity. He was taken to an evidence based cognitive rehabilitation programme by Physiotherapy Department of our university, performing computer-based training batteries that target specific cognitive functions. We have planned to monitor the recovery of neurocognitive skills, attention problems, amnesia. Also neurocognitive tests will be repeated after 6 months to evaluate the benefit of neurocognitive rehabilitation.

Since there is no sufficient evidence about the effect of acute hypoxia and ischemia on exacerbation of previously known neurodevelopmental disorders such as ADHD, we can not make a certain evaluation in this case. There is inadequate evidence of the effectiveness of cognitive therapy in the early phase of rehabilitation, since the effects of cognition therapy cannot be distinguished from those of spontaneous remission. Highly consumption of ED which may have negative cardiac effects was estimated in our case as an attempt to self medicate untreated ADHD. Regular psychiatric and cardiac follow up in ADHD treatment are serious demands. Uncontrolled ED consumption may end up with more serious results than using psychostimulants with regular follow up

Keywords: ADHD, cognitive rehabilitation, cardiac arrest

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[Abstract:0772][Psychopharmacology]

Acute ataxia and hypotonia associated with methylphenidate

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Methylphenidate (MPH) exerts its pharmacological effects via increasing the levels of dopamine and norepinephrine in the synaptic cleft. A number of well recognized adverse effects such as nausea, loss of appetite and sleep problems are reported during MPH treatment in children. To the best of our knowledge there is no reported case of acute ataxia and hypotonia associated with immediate release methylphenidate (IR MPH). Here, we present a male child with ADHD who developed reversible acute ataxia and hypotonia after IR MPH receiving.

Case: A 7 year-old boy presented to the outpatient clinic of child and adolescent psychiatry due to complaints of irritability, impatience, restless and troubled behaviors, forgetfulness, learning disabilities, and nocturnal enuresis. His prenatal, natal, and postnatal histories were normal except for X-ray exposure in prenatal period. The patience has been using Levothyroxine sodium 100 mcg/g for hypothyroidism. He was diagnosed with ADHD and nocturnal enuresis. His laboratory test results were normal except for low ferritin level. In our clinic, we have tested 10 mg immediate-release methylphenidate (IR MPH) effect on cardiac parameters before the ADHD treatment especially in children less than 9 years of age. The test applied to patient at 13.30. Baseline cardiac parameters; his blood pressure (BP) was 102/62 mmHg and heart rate (HR) was 79 bpm. We reevaluated cardiac parameters at 40. minute after receiving IR MPH. His BP was 88/58 mmHg and HR was 67 bpm. At 60. minute after receiving IR MPH; drowsiness, loss of coordination and balance, ataxic walking, hypotonia and tending to sleep was observed in patient's clinic. His neurologic assessment revealed that ataxic walking and decrease in muscle strength (upper extremity: 3/5, lower extremity: 2/5). He was observed for 24 hours in emergency department. His symptoms disappeared and muscle strength returned to normal at 19:00 (five and a half hours after IR MPH).
In our case, ataxia and hypotonia were observed from one hour after oral IR MPH administration. These side effects have been observed correspond with time of the drug's maximum plasma concentration. This temporal relationship suggested that these side effects might be associated with IR MPH plasma concentration. It has been suggested the variation in the rate of hydrolysis of MPH would vary the bioavailability of MPH, leading to differential symptom changes and side effect profiles. Animal studies have shown that small differences in serum MPH could be associated with large differences in brain MPH concentrations. It has been postulated that even in small doses may result in different responses in some individuals. The ritalinic acid (RA) is the inactive metabolite of MPH, its peak plasma concentrations are higher than peak MPH plasma concentrations and it's half-life (t1/2) is approximately twice the t1/2 of MPH. We have speculated that the termination time of ataxia and hypotonia side effects might be associated with the t1/2 of RA. We suggested that variability in plasma MPH concentrations were not only important for therapeutic effect but also might be important for side effect profiles.
Keywords: ataxia, hypotonia, methylphenidate

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Psychogenic dysphonia: A case resistant to treatment

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Psychogenic dysphonia is a disorder characterized by loss of voice, in the absence of organic pathology. It is a type of conversion disorder, mostly seen in female gender and usually there is a link between dysphonia and psychological stressor factors such as chronic anxiety, depression, interpersonal problems, trauma. Symptoms are mostly short duration and problem can be resolved quickly but sometimes it may be very severe and resistant. A complete medical examination should be completed to rule out any possible organic cause for the disorder.
Case: A 55 year-old female patient who developed psychogenic dysphonia following her daughter divorcing. The patient had not any medical complaints before that condition. 5 year ago her daughter began to experience problems with her husband and they decided to divorce. Afterwards the patient developed dysphonia, her speech could be hearing hardly. He presented to various clinic for her dysphonia and many medical intervention administered. Her blood tests, physical examination, brain MRG and flexible endoscopic evaluation was normal. Acute onset, developing after stressfull life event, presence of la bella indifference was suggested as psychogenic to us. The patient improved partially with regular psychiatric interviews and voice therapy. But during the treatment after experiencing family conflict her complaints repeated and these symptoms were resistant to treatment. For treatment of dysphonia, clinician should be recognize association between psychological factor which that contribute to develop dysphonia. Obtaining detailed medical history and psychiatric assesment are essential in these case.
Keywords: conversion disorder, differential diagnosis, psychogenic dysphonia

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