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A Case of Tardive Dyskinesia Due to Olanzapine Treatment

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ÖZET:

Olanzapin kullanımına bağlı gelişen bir geç diskinezi olgusu

Geç diskinezi (GD) , antipsikotik kullanımı sonrasında dilde, çenede, gövdede, kol ve bacaklarda ortaya çıkan anormal istemsiz hareketler kompleksidir. Bu hareketler çoğunlukla koreiform, atetoid veya ritmik hareketlerdir. Antipsikotik kullanımının neden olduğu GD tanısı için, DSM-IV ölçütlerine göre, bu istemsiz hareketlerin en az 4 hafta süreyle var olması, antipsikotik ilacın en az 3 ay, eğer 60 yaş ve üstünde ise en az 1 ay süre ile kullanılması gerekmektedir.

Nigro-striatal bölgedeki dopamin reseptörlerini bloke eden yüksek potensli klasik antipsikotiklerin kullanımı ile GD gelişimi daha fazladır ve doz arttıkça risk artmaktadır. Risperidon, olanzapin, ketiapin, ziprasidon gibi atipik antipsikotikler daha düşük GD gelişimi riskine sahiptir. Olanzapin, diğer psikotrop ilaçlara bağlı gelişen geç diskinezi olgularının tedavisinde kullanılmaktadır ve geç diskineziye yol açtığı ile ilgili olgu sunumları nadirdir.

Bu yazıda, mevcut literatür ışığında 24 yaşında erkek bir hastada olanzapin kullanımına bağlı gelişen geç diskinezi tartışılmıştır.

Anahtar sözcükler: Olanzapin, geç diskinezi, antipsikotik

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ABSTRACT:

A case of tardive dyskinesia due to olanzapine treatment

Tardive dyskinesia (TD) is a complex of abnormal reflex movements involving the tongue, body, arms, and legs after the use of antipsychotics. These movements are generally choreiform, athetoid, or rhythmic. According to the DSM-IV, these reflex movements must have been present for at least four weeks and the antipsychotic medication should have been used for at least 3 months (at least 1 month if the patient is 60 years or older) for the TD to have been caused by the use of antipsychotic drugs.

TD development is much more common with the use of high potency classic antipsychotics that block dopamine receptors in the nigrostriatal area and the risk increases as the dosage increases. Atypical antipsychotics such as risperidone, olanzapine, quetiapine, and ziprasidone have lower risks for TD development. Olanzapine has been used in the treatment of cases where tardive dyskinesia occurred due to other psychotropic medications and there are rare case reports about olanzapine caused tardive dyskinesia.

In this report, a case of tardive dyskinesia due to olanzapine use in a 24 year-old male patient is discussed in the light of existing literature.

Key words: Olanzapine, tardive dyskinesia, antipsychotic

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INTRODUCTION

Tardive dyskinesia (TD) is a complex of abnormal reflex movements involving the tongue, body, arms, and legs after the use of antipsychotics. These movements are generally choreiform, athetoid, or rhythmic.

Tardive dyskinesia may occur in 20-30% of the general population that use antipsychotic drugs. This may increase up to 40-47% in the elderly. If a psychiatric illness begins at an older age, this situation makes the emergence of late dyskinesia more likely. The risk is found to be 3-5 times higher in elderly patients compared to young patients. In addition to age, the risk is directly proportional to: female gender, daily and total dose of the antipsychotic drug, presence of mood disorder,, the use of anticholinergics with neuroleptics, antipsychotic drop out, previous physical therapies (ECT), the presence of other physical illness such as diabetes or an organic disorder, younger age

of exposure, and the presence of extrapyramidal symptoms early in treatment (1).

The risk for the development of tardive dyskinesia increases with high potency classical antipsychotics that blocks dopamine receptors in the nigrostriatal region and with higher dosages (2). Atypical antipsychotics such as risperidone, olanzapine, quetiapine and ziprasidone have a lower risk for the development of TD (3). Olanzapine has been used to treat tardive dyskinesia induced by other psychotropic drugs (4, 5, 6) and it has been reported to be less likely to induce extrapyramidal side effects (7).

In this report, a late dyskinesia (LD) case due to olanzapine use is presented.

CASE

A 24 year old, single, male patient with a history of excessive speech, abnormal behavior, grandious ability delusions, hyperactivitiy, and insomnia starting nearly five years ago was prescribed valproic acid and olanzapine for treatment of bipolar disorder (manic episode with psychotic features) and he benefited from the treatment. Recently, he had a recurrent episode with a clinical picture of reference delusions, social withdrawal, insomnia, and abnormal behavior due to a medication discrepancy and he was hospitalized with a diagnosis of psychotic disorder, not otherwise specified. Olanzapine 15 mg/day was initiated. Three weeks later, the dose of olanzapine was increased to 20 mg/day, as no improvement had been seen in the clinical picture. Because of lack of clinical improvement with the pharmacological treatment, 14 sessions of ECT were given. The patient responded well to the treatment and was discharged with monthly follow up visits. The patient came for follow up 3 months after discharge. In the clinical examination, it was observed that the disease had progressed with residual symptoms such as restricted affect, social withdrawal, and anhedonia. In the physical examination involuntary protrusion of the tongue,, which became evident during speech,, was observed. The patient was not aware of this movement which was first noticed by a relative and it was not causing a loss in functionality. According to the information taken from the patient's family, the symptoms started 15 days after discharge and only occurred during speech. Initially, there also had been involuntary eye movements but these gradually decreased. This movement occurred at the beginning of every period of speech and was frequently repeated during conversations but disappeared during sleep.

CLINICAL COURSE

There was no abnormality in the patient's complete blood count and comprehensive metabolic panel. During neurological examination, an essential tremor in the left hand which was evident while the hands were stretched forward, hitting the base of the foot to the ground slightly in the gait examination, protrusion of the tongue which was aggravated during speech and involuntary movements in the form of slight eye blinking were observed. The patient was evaluated as exhibiting antipsychotic induced tardive dyskinesia; the AIMS (Abnormal Involuntary Movement Scale) score was 11, the SANS (Scale for the Assessment of Negative Symptoms) score was 43, the SAPS (Scale for the Assessment of Positive Symptoms) score was 8, and the BPRS (Brief Psychiatric Rating Scale) score was 22. The dose of olanzapine was decreased from 20 mg/day to 15 mg/day. Synthetic vitamin E (300 mg/day) was added to the treatment. The AIMS was performed every 3 days. The dose of olanzapine was reduced down to 10 mg/day. When the patient was discharged after treatment, the last AIMS score was 6, the SANS was 40, the SAPS was 7, and the BPRS score was 19.

DISCUSSION

In this article, a case is discussed who was followed up with a diagnosis of psychotic disorder and who developed tardive dyskinesia after about 5 years of olanzapine treatment.

No strict criteria have been set for the diagnosis of tardive of dyskinesia (TD). The presence of three features, known as the Schooler and Kane criteria, is often deemed to be necessary for the diagnosis of tardive dyskinesia (8). These are: 1) the use of antipsychotic drugs for at least three months; 2) involuntary movements of moderate intensity observed at least in one region or of mild intensity in at least two regions, 3) exclusion of other conditions that cause movement disorders. In our case, these three criteria were met.

The beginning of the disorder with mood symptoms, ECT treatment and an antipsychotic drug break might be considered as risk factors for the occurrence of tardive dyskinesia.

The observation of TD that is usually observed after long-term use of typical antipsychotics such as haloperidol, is especially important in two ways in our case. First, olanzapine is known as reliable in terms of side effects related to the extrapyramidal system. The second is the importance of frequent follow up of patients and informing patients and families in terms of all kind of possible side effects of psychotropic drugs including tardive dyskinesia, which may be irreversible and clinically important.

Published cases of olanzapine induced tardive dyskinesia and tardive dystonia indicate chronic use of olanzapine at doses above 10 mg/day and this is compatible with our case (9-11). Improvement in the clinical picture as well as improved AIMS scores, after reducing the dose of olanzapine lend support to the conclusion that these sumptoms were due to olanzapine use.

In the case report of Çayköylü et al.,. a 39 year old male patient developed exacerbation of psychotic symptoms simultaneously with the initiation of symptoms of tardive dyskinesia in the last 15 days of a three-month olanzapine (7.5 mg/day) treatment (12). In our patient, the occurrence of dyskinesia signs without worsening of psychotic symptoms differs from this case.

Olanzapine is widely used in the treatment ofpsychotic disorders, as well as bipolar disorder, refractory depression, treatment resistant obsessive-compulsive disorder, obsessive-compulsive disorder with psychotic features, and conduct disorder. Given their ever-expanding usage, clinicians should keep in mind that not only typical antipsychotics, but rarely atypical antipsychotics such as olanzapine can cause dyskinesia. This finding is important in both improving treatment adherence of patients and providing data for more comprehensive models explaining the development of tardive dyskinesia.

References:

1. Woerner MG, Alvir JM, Saltz BL, Lieberman JA, Kane JM. Prospective study of tardive dyskinesia in the elderly: rates and risk factors. Am J Psychiatry 1998;155(11):1521-8.
2. Morgenstern H, Glazer WM. Identifying risk factors for tardive dyskinesia among long-term outpatients maintained with neuroleptic medications. Arch Gen Psychiatry 1993; 50(9):723-33.
3. Gupta S, Mosnik D, Black WD, Berry S, Masand PS. Tardive dyskinesia: review of treatments past, present and future. Ann Clin Psychiatry 1999;11(4):257-66.
4. Haberfellner EM. Remission of tardive dyskinesia after changing from flupenthixol to olanzapine. Eur Psychiatry 2000; 15(5):338-9.
5. Soutullo CA, Keck PE Jr, McElroy SL. Olanzapine in the treatment of tardive dyskinesia: a report of two cases. J Clin Psychopharmacol 1999;19(1):100-1.
6. Almeida OP. Olanzapine for the treatment of tardive dyskinesia. J Clin Psychiatry 1998;59(7):380-1.
7. Breier A, Hamilton SH. Comparative efficacy of olanzapine and haloperidol for patients with treatment-resistant schizophrenia. Biol Psychiatry 1999;45(4):403-11.
8. Owens DGC. Tardive dyskinesia. In: A Guide to the Extrapyramidal Side-effects of Antipsychotic Drugs. Cambridge, UK: Cambridge Üniversity Press 1999:166-226.
9. Lu LM, Lane HY. Olanzapine-induced tardive dyskinesia. BMJ 2003;182(1):81-2.
10. Conley RR, Meltzer HY. Adverse events related to olanzapine. J Clin Psychiatry 2000;61 (Suppl.8):S26-S9.
11. Dunayevich E, Strakowski SM. Olanzapine-induced tardive dystonia. Am J Psychiatry 1999; 156(10):1662.
12. Caykoylu A, Aydin N, Karalar F. Atipik antipsikotiklerle ortaya çıkan bir geç diskinezi ve süpersensitivite psikozu olgusu. Klinik Psikofarmakoloji Bülteni - Bulletin of Clinical Psychopharmacology 2001;11(1):37-40 (Turkish).