A Case of Tardive Dyskinesia Due to Olanzapine Treatment

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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

ÖZET: Olanzapin kullanımına bağlı gelişen bir geç diskinezi olgusu


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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OLGU SUNUMLAŞI:

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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, hyperactivity, 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 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 symptoms 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 of psychotic 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: