NÖRO PSİKİYATRİ ARŞİVİ

Case Report

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Withdrawal-Emergent Dyskinesia and Supersensitivity Psychosis Due to Olanzapine Use

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ABSTRACT

Tardive dyskinesia (TD) usually appears after years of antipsychotic drug use and appears to be related to the total lifetime medication dose. In withdrawal-emergent dyskinesia (WE-D), which is considered to be a subtype of TD, dyskinetic symptoms often appear shortly after a rapid reduction in antipsychotic drug dose or sudden discontinuation of the drug. Supersensitivity psychosis, which is frequently observed along with TD and is considered to have a similar etiology as TD, is a psychotic relapse phenomenon that occurs after the withdrawal of an antipsychotic drug or a rapid reduction in the drug dosage. In general, atypical antipsychotics tend to be associated with less propensity to cause TD when compared with typical antipsychotics. Furthermore, olanzapine and clozapine may have a therapeutic potential in improving or totally curing TD. In this study, a case of WE-D because of discontinuing olanzapine use and supersensitivity psychosis is discussed.

Keywords: Olanzapine, tardive dyskinesia, withdrawal-emergent dyskinesia, supersensitivity psychosis

INTRODUCTION

Tardive dyskinesia (TD) is a neuromuscular disorder that is characterized with involuntary, repetitive, and unintentional movements occurring during treatment or a short time after discontinuing treatment, which develops in response to long-term antipsychotic use. Although it may occur in any muscle in the body, it is most commonly observed in the mouth, arm, leg, and trunk muscles. TD is observed in 15%–30% of patients who have used antipsychotic drugs for more than 3 months (1).

Although the pathophysiology of TD has not been completely elucidated, the most commonly accepted hypothesis is dopamine receptor hypersensitivity. According to this hypothesis, hypersensitivity develops in dopamine receptors that are found in the nigrostriatal dopamine pathway because of long-term antipsychotic medication use. Exacerbation of the picture with dopamine agonists supports this assumption, which is also called "denervation hypersensitivity" (2).

In withdrawal-emergent dyskinesia (WE-D), which is considered a TD subtype, dyskinetic symptoms often appear shortly after rapidly reducing the antipsychotic drug dose or suddenly discontinuing the drug in a portion of patients who did not display abnormal involuntary movements during antipsychotic treatment. Spontaneous improvement has been reported in 90% of cases with WE-D, which usually require no treatment (3).

Supersensitivity psychosis, which is frequently observed together with TD and is considered to have a similar etiology as TD, is a psychotic relapse phenomenon that occurs after the withdrawal of an antipsychotic drug or rapid reduction of the drug dosage (4). In addition, supersensitivity psychosis has been reported in patients who are known to comply with the antipsychotic medication. Supersensitivity psychosis has been found to be related with an increase antipsychotic drug dose with the aim of preventing progression of symptoms and sensitivity to life events along with the presence of abnormal involuntary movements and TD (5).

In general, TD is almost never observed with atypical antipsychotics (6). Furthermore, marked improvement in TD findings arising from other antipsychotic drugs is observed when olanzapine and clozapine are used (7). In this study, a patient who developed withdrawal dyskinesia and supersensitivity psychosis because of discontinuing olanzapine is presented.

CASE

Mr. C.K. was a 59-year-old married male patient. No prior psychiatric disease was described in his medical history. The first symptoms occurred 2 years prior to his hospital admission because of fears about being hurt, cheated, or killed by someone and suspicions about his wife's

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unfaithfulness. Complaints of insomnia and loss of appetite were also present in the patient who could not leave his house because of his fears. Olanzapine (10 mg/day), sertraline (50 mg/day), and chlorpromazine (100 mg/day) were prescribed by the psychiatrist, whom he was referred to by his relatives 3 months after his complaints started. After a while, a marked regression occurred in his complaints with his regular use of medications. Then he was recommended to use only olanzapine and to discontinue other drugs. He continued to use olanzapine (10 mg/day) for 2 years without being controlled by a physician, and he had no psychiatric complaints during this period. He was brought to the emergency department of the Bakırköy Mental Health and Neurology Research Hospital with complaints of suspicions that he was being followed and fears about being hurt and killed, remaining at home all the time, and insomnia, which started a few days after he discontinued his medication. On psychiatric examination, his self-care appeared normal and appropriate for his age. The patient was observed to be anxious and looked around with frightened eyes and had psychomotor restlessness. He maintained eye contact but did not spontaneously speak and gave short answers to questions. He was conscious, cooperative, and completely oriented. His affect was anxious. Impairment of perception was not observed. Thought content was notable for persecutory delusions.

On physical examination, involuntary oral movements, including chewing, lip smacking, and licking lips, were observed. It was learned that oral dyskinesia was not present before and had started a few days after he discontinued his medication. Other examination findings were found to be normal. No other pathology was considered on neurological consultation, and no additional treatment was recommended. His EEG and hepatic, renal, thyroid functions as well as complete blood count were found to be normal.

The present condition of the patient was evaluated to be WE-D and supersensitivity psychosis. Treatment with olanzapine 10 mg/day and outpatient follow-up were initiated. On follow-up examination that was performed one week later, a marked reduction in the psychotic symptoms was observed, and complete improvement in his involuntary movements occurred after the fifth day.

DISCUSSION

Although definite criteria have not been determined for TD, the first criteria used in diagnosis were developed by Schooler and Kane (8) in 1982 for the first time. According to these criteria, TD diagnosis should be based on the history of at least 3 months of total cumulative neuroleptic exposure, presence of at least "moderate" abnormal, involuntary movements in one or more body areas or "mild" movements in two or more body areas, and the absence of any other condition that might explain these abnormal involuntary movements.

Various hypotheses related with the pathophysiology have been proposed. Mostly, the underlying cause has been assumed to be antipsychotic drugs and other dopamine agonists, and the phenomenon of dopamine receptor hypersensitivity in the nigrostriatal dopamine pathway has been emphasized (2). There are also opinions proposing that TD develops because of GABA insufficiency and cellular neurotoxicity and degeneration (3).

It is known that the frequency of TD can be reduced with gradually increasing the usage of atypical antipsychotic drugs. High efficiency in the treatment of psychosis together with fewer side effects of atypical antipsychotic drugs are generally related with dopaminergic blockage in the mesolimbic pathway rather than the nigrostriatal pathway and increased dopamine release due to serotoninergic blockage in the nigrostriatal pathway (6).

It has been reported that TD is almost never observed with olanzapine and clozapine use; clozapine has a place in the treatment of TD and sometimes an improvement is observed with olanzapine use (7). However, there are studies reporting that olanzapine use may rarely cause TD because of its higher affinity to D2 receptors compared to clozapine (9).

In WE-D, which is considered as a subtype of TD, abnormal movements in the neck, face, mouth, arms, and legs appear shortly after a rapid reduction of the antipsychotic drug dose or sudden discontinuation of the drug in a portion of patients who do not display abnormal involuntary movements during antipsychotic treatment. WE-D is a reversible dyskinesia and generally improves spontaneously in 1–2 months. It is thought that a sudden change in the dopamin-acetylcholine balance in the striatum causes WE-D, which is similar to TD arising from long-term antipsychotic use. Although it has been reported that it needs no treatment and spontaneous improvement is observed with a rate reaching up to 90%, it is recommended that the antipsychotic be restarted and tapered gradually over 1–3 months (3).

In our case, no organic or metabolic disease that might have caused TD was found. No factor that might have played a role was found other than the use of medication. The fact that the patient used olanzapine (10 mg/day) for approximately 2 years, described no abnormal, involuntary movement during this time period, and that dyskinetic movements in the mouth region started in the weeks following the discontinuation of medication supports WE-D.

The fact that no psychotic symptom was described during the period when the patient used medication, but psychotic symptoms recurred in the weeks following the sudden discontinuation of olanzapine suggests supersensitivity psychosis. The appearance of supersensitivity psychotic symptoms in the same time period with WE-D increases the possibility of supersensitivity psychosis because of similar underlying mechanisms.

Further studies are needed to elucidate the underlying mechanisms of TD and supersensitivity psychosis. Knowing and recognizing these two pictures is important in the adjustment of antipsychotic treatment, dose reduction, drug discontinuation, and medication switching.

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