Olgu Sunumu / Case Report
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Tardive Dyskinesia Due to Short-Term and Low-Dose Use of Trifluoperazine: A Case Report

Kısa Süreli ve Düşük Dozda Trifluoperazin Kullanımına Bağlı Tardif Diskinezi: Bir Olgu Sunumu

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ÖZET

Tardif diskinezi (TD), vücudun herhangi bir bölümünü etkileyen amaçsız ve istemsiz hareketlerle karakterize bir hastalıktır. Bu hareketler tipik olarak ağız-yüz bölgesinde meydana gelir ve hasta genellikle bu hareketlerin farkında değildir. TD, kısa süreli ve düşük dozda antipsikotik kullanımına ikincil olarak daha nadir görülür. Burada kısa süreli ve düşük dozda trifluoperazin kullanımına bağlı bir TD olgusunu sunduk. Hasta 38 yaşında bir kadın hastaydı ve yaygın anksiyete bozukluğu tanısı ile 1 mg/gün trifluoperazin ile tedavi edilmişti. TD semptomları, trifluoperazin başlandıktan üç ay sonra ortaya çıkmıştı, ilaç kesilmesinden sonra yavaş yavaş azaldı ve altı ayın sonunda tamamen kesildi. TD tedavisine ilişkin veriler sınırlıdır ve en iyi yöntem ortaya çıkmasını önlemektir.

Anahtar Kelimeler: tardif diskinezi, trifluoperazin, antipsikotik, yan etki

ABSTRACT

Tardive dyskinesia (TD) is a disorder characterized by purposeless and involuntary movements affecting any part of the body. These movements typically occur in the oro-facial area and the patient is usually unaware of them. TD is rarely seen secondary to short-term and low-dose use of antipsychotics. Herein, we present a short-term and low-dose trifluoperazine-induced TD. The patient was a 38-years-old female patient who was being treated with trifluoperazine 1 mg/day with a diagnosis of generalized anxiety disorder. The symptoms of TD appeared three months after the introduction of trifluoperazine, decreased gradually and ceased at the six months of discontinuation. Data on TD treatment are limited, and the best management strategy remains prevention

Keywords: tardive dyskinesia, trifluoperazine, antipsychotic, side effect

INTRODUCTION

Tardive Dyskinesia (TD) is a hyperkinetic movement disorder that may occur during or shortly after discontinuation of antipsychotic (AP) medication. It is an iatrogenic condition that presents with involuntary movements of stereotypic, choreiform or athetoid, especially in the mouth, tongue and face. While any agent capable of dopamine receptor blockade can cause TD, it is more common in psychiatric disorders that require long-term use of antipsychotics such as schizophrenia and schizoaffective disorder. However, TD is rarely seen secondary to short-term and low-dose use of such APs (1). Herein, we present a short-term and low-dose trifluoperazine-induced TD.

CASE

A, 38-years-old married female patient had been followed at a psychiatry outpatient with a diagnosis of generalized anxiety disorder according to Diagnostic and Statistical Manuel of Mental Disorders, Fifth Edition (DSM-5) (2). She had been using 30 mg/day paroxetine and 5 mg/day diazepam for one year. Previously used drugs were escitalopram and sulpiride. On admission, there was no sleep problem, but her severe irritability symptom was not controlled with this treatment. We decided to manage the patient with adding trifluoperazine. Three months after starting 1 mg/day of trifluoperazine, the patient came to our clinic complaining of involuntary oro-facial and lingual movements. The patient's TD symptoms disappeared with sleep. No additional extrapyramidal findings were detected. The patient's thyroid, kidney and liver function tests were within normal limits. The fasting blood glucose, protein level and lipid profile were within normal limits. Chest X-ray, electrocardiogram, computed tomography, and magnetic resonance imaging gave normal results. Electroencephalography was unremarkable. The relatives of patient stated that there was no change in dietary and fluid intake in recent days. The patient had no drug use other than paroxetine, diazepam and trifluoperazine. She had no systemic disease such as hypertension or diabetes mellitus. No allergies

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to drugs, foods and pollens were reported. Past history of patient was unremarkable. There was no any antipsychotic sensitive neuropsychiatric disorder or movement disorder in the family history. TD was attributed to trifluoperazine. Trifluoperazine was stopped and other medications were continued. TD decreased gradually and ceased at the six months of discontinuation. The patient and his relatives were warned about TD due to trifluoperazine use and written informed consent was received from the patient to publish her data. Naranjo Adverse Dug Reaction Probability Scale (NADRPS) score of the patient was 5 (3).

DISCUSSION

This case report was evaluated as a case of TD due to trifluoperazine. Because there was a temporal relationship between them, the side effect began with the addition of the drug and completely cured after discontinuation of the drug. The NADRPS score indicates a probable association between drug use and side effect (3).

The term tardive emphasizes the later development of abnormal movements following chronic neuroleptic treatment. Schooler-Kane research criteria are frequently used in the diagnosis of TD. According to these criteria, dopamine blocking agent should be used for at least three months in order to evaluate the present movement disorder as TD. For patients over 60 years, the duration of drug use is one month. Movement disorder should last for at least one month after oral treatment is discontinued and eight weeks for depot injection. However, clinical experience supports the rare but real potential of shorter exposures to cause TD, questioning the applicability of these criteria in everyday practice (1, 4).

Age is the most consistent risk factor for TD. When TD ratios were grouped by age, increased risk was found in women over 51 years of age. However, there are also documents in the literature showing that there is no relationship between gender and TD incidence (5). Extrapyramidal symptoms as a result of long-term use of antipsychotics, anticholinergic, use of antiparkinsonian agents, organic brain damage, mental retardation, negative symptoms of schizophrenia, mood disorders, smoking, alcohol and substance use, diabetes mellitus, malnutrition and vitamin deficiencies are other important risk factors (1, 6). As with other movement disorders, the involuntary movements in TD worsen with emotional stress, diminish with sedation, and disappear with sleep. TD was differentiated from perioral tremor (Rabbit syndrome) with the help of no persistence in sleep (7).

Typical antipsychotic drugs include chlorpromazine, flufenazine, trifluperazine, flupentixol, zuclopenthixol, haloperidol, pimozide, atypical antipsychotic drugs clozapine, quetiapine, sulpiride, amisulpride, ziprasidone, olanzapine, and risperidone (8). Cases of TD induced by low-dose trifluoperazine are not common in the literature. Chouinard et al. (9) reported a case of TD that occurred after 4 months of trifluoperazine at a dose of 6 mg/day. In our study, a case of TD using a lower dose of trifluoperazine for a shorter period is presented. Typical antipsychotics have been associated with higher risk of TD development than atypical agents. These rates are explained by the fact that atypical antipsychotics show low affinity for dopamine (D2) receptors in the dorsal striatum and have antagonistic effects on serotonin (5-HT2A/2C) receptors (10). Many theories have been proposed in TD mechanism. Different mechanisms other than dopamine blockade have been emphasized and these mechanisms have been thought to directly or indirectly affect the nigrostrial pathway. Hypersensitivity of the dopamine receptor system in the nigrositriatal pathway, disruption of dopaminergic and cholinergic systems, dysfunction of striatonigral GABA'ergic neurons and excitotoxicity are emphasized (11).

Since there is no gold standard in the treatment of TD, it is important to prevent the development of TD by taking preventive measures and identifying the initial symptoms. Patients and caregivers should be informed about abnormal movements. In patients who developed TD during the use of anticholinergic agents, the first change in treatment should be the discontinuation of the anticholinergic drug. Anticholinergics are known to cause deterioration of TD symptoms despite improvement in movement disorders such as acute dystonia. Anticholinergic agent should be discontinued if used primarily in the treatment of TD. Antipsychotic needs should be evaluated and medications that may exacerbate symptoms should be discontinued. Benzodiazepines have a positive effect on symptoms of tardive dyskinesia, tardive dystonia and tardive myoclonus by increasing GABA in the nigrostriatal region (11). In our patient, the patient had side effects while using a benzodiazepine drug and an anticholinergic drug. Discontinuation of the accused drug and continuation of benzodiazepine were thought to contribute to the clinical improvement. In addition, this study shows that TD can be reversible in the early period when the guilty agent is discontinued.

As a result, TD is an iatrogenic, hyperkinetic movement disorder especially caused by the use of antipsychotics. It is a difficult and lasting condition that may cause problems in treatment compliance, adversely affect the quality of life of the patient. Physicians prescribing quetiapine need to be aware of the risk of TD and should adequately counsel patients or consider alternative agents when supportive evidence exists, particularly with respect to its off-label use.

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