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Case report / Olgu sunumu

Paroxetine induced reversible dyskinesia: a case report*

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Abstract

Selective serotonin reuptake inhibitors (SSRI) are the primary medications that have been used recently for the treatment of psychiatric disorders. Paroxetine is one of the medications in the category of SSRI. In the literature, dyskinesia cases related to paroxetine are quite rare in comparison to other movement disorders, and the ones that have been presented are facial dyskinesia. The purpose of this report is to present an involuntary choreiform dyskinesia case that appears in the extremities related to addition of paroxetine on chlorpromazine. To our knowledge, our case, extremity dyskinesia related to paroxetine is the first case that has been observed so far. (Anatolian Journal of Psychiatry 2017; 18(5):516-518)

Keywords: paroxetine, side effects, extrapyramidal disorders, dyskinesias

Introduction

Although most common side effects of Selective serotonin reuptake inhibitors (SSRI) are gastrointestinal side effects and sexual dysfunction, SSRI might also cause side effects related to the extrapyramidal system (EPS).¹,² These side effects are generally thought to emerge due to dopaminergic hyperfunction developed through D₂ receptors' suppression inside the nigrostriatal pathway or through the increase in D₂ postsynaptic receptors' sensitivity. Dopaminergic neurons inside the nigrostriatal pathway also have serotonergic inputs causing inhibition activity on the dopaminergic swing.¹,² Therefore, medications that increase serotonergic activity might also cause the inhibition of dopaminergic activity by hyperarousal of 5-HT₂A receptors in the basal ganglia.² While the EPS related side effects during the period of SSRI treatment have mostly been in the akathisia mode, other movement disorders might also have been observed.²-¹¹ It is known that the incidence of EPS in SSRI¹ use is 1/1000 or less.³,⁴ EPS related side effects can both occur with short-term and long-term use of SSRI, and it is not dose related.³,⁴ Paroxetine is one of the medications in the category of SSRI. Past research on dyskinesia cases related to paroxetine are quite rare, and

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the cases that have been presented up to date consist of tongue, mouth, and ear movements after the addition of paroxetine on antipsychotics.\textsuperscript{2,5-9} Therefore, the purpose of this study is to contribute to the literature on dyskinesia cases by presenting an involuntary choreiform dyskinesia case appearing in the extremities related to the addition of paroxetine on chlorpromazine. To our knowledge, this report is the first case report on the observation of extremity dyskinesia related to paroxetine use.\textsuperscript{2,5-9}

**CASE**

A female patient, who was a 32-year-old, came to our outpatient clinic with complaints of unhappiness, anhedonia and insomnia for three months. In her mental state examination, her affect and mood were depressed. She had thoughts about self-harm in addition to thoughts of worthlessness and inadequacy. She had insomnia, anhedonia, and loss of appetite. She had several treatments for depression and she did not have any physical illness in her medical history. Before applying to our clinic, she had been prescribed 30 mg/day of duloxetine at another clinic 2 months ago. With a pre-diagnosis of major depressive disorder based on DSM-V, the patient was admitted to the inpatient clinic.\textsuperscript{12}

Her treatment was organized as 60 mg/day of duloxetine and 5 mg/day of diazepam. At the end of her first week of treatment, 100 mg/day of chlorpromazine was added to her treatment because she was still complaining about insomnia. In the fourth week of her treatment her affect was still depressed. So her treatment with duloxetine was terminated, and she instead started to take 20 mg/day of paroxetine. In her fourth day of treatment with paroxetine, recurrent choreiform movements in her extremities were observed. In her physical examination, there was no pathological sign that could explain the patient’s complaints based on routine blood count and biochemical examinations. There was no pathological sign that could explain the current movement disorder in the cranial imaging. To deal with her current situation, all of the medications were terminated but to control her anxiety about dyskinesia, she was given 10 mg/day of diazepam again. As parallel with the termination of drugs, her dyskinesia started decreasing and completely ended the second day. A 30 mg/day dose of duloxetine was then added to her treatment because of the confident side effect profile for this patient before, and the dosage was increased to 60 mg/day at a follow-up. Because her recurrent insomnia had not benefited from other medications, 100 mg/day of chlorpromazine was added to her treatment again. With the treatment of 60 mg/day of duloxetine, 10 mg/day of diazepam, and 100 mg/day of chlorpromazine, her all complaints decreased significantly, and she was discharged.

**DISCUSSION**

Past research has reported several movement disorders ranging from dystonia to dyskinesia that are associated with different antidepressant medications.\textsuperscript{2-11} In a study by Hawthorne et al. on cases with EPS related side effects, 80.2\% of them were found to have SSRI use.\textsuperscript{9} In another study, SSRI, as independent from the use of additional medications, were found to cause 2.2 times as many EPS related side effects regarding the other antidepressants. Paroxetine was also found to be the most related medication to EPS in the category of SSRI (49\%) and among all antidepressants (38\%).\textsuperscript{10}

In the literature, there exists few studies on dyskinesia cases related to paroxetine use.\textsuperscript{5-7} Botsaris et al. reported first two cases on dyskinesia related to paroxetine use, and found that involuntary tongue and mouth movements appeared with the addition of paroxetine on haloperidol and prochlorperazine treatment in different cases.\textsuperscript{7}

Movement disorders stemming from medications are essentially related to the nigrostriatal pathway. Serotoninergic input that goes into the nigrostriatal pathway has an inhibitor impact on the dopaminergic system, and causing EPS.\textsuperscript{13,14} Moreover, the use of neuroleptic medications simultaneously or before the SSRI treatment was predicted to cause the decrease in the nigrostriatal dopamine reserves.\textsuperscript{2,5} Certain individual genetic factors might also play a role on the EPS development as independent of age and the simultaneous use of other medications. Hedenmalm et al. claim that the observation of EPS related side effects in the process of SSRI treatment might depend on the Taq1A polymorphism of the DRD2 gene associated with the existence of the A1 allel within D2 receptor, in addition to CYP enzyme changes in these patients.\textsuperscript{15} This situation might also explain why every patient who uses antipsychotic medication and paroxetine does not develop similar EPS related side effects. Paroxetine is an agent often associated with the CYP2D6 inhibition, and in
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our case, the dyskinesia appeared to slow down after the termination of the paroxetine treatment.1

In conclusion, although SSRI provide more satisfactory side effect profiles regarding most of the antidepressant medication groups, they also might cause EPS related side effects in some of the patient groups such as those who use simultaneous antipsychotics. Clinicians should be aware that EPS related side effects, despite being reversible, might influence patients’ quality of life and the compliance with the medication.

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REFERENCES


