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Fluoxetine-Induced Dyskinesia in a Pediatric Patient: A Case Report and Literature Review on Implications of Polypharmacy and Serotonergic Regulation

Pediatrik Bir Hastada Fluoksetin Kaynaklı Diskinezi: Polifarmasi ve Serotonerjik Düzenlemenin Etkileri Üzerine Bir Vaka Raporu ve Literatür İncelemesi

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ABSTRACT

Movement disorders associated with psychiatric medications are primarily related to the side effects of dopamine receptor-blocking drugs, which are mostly antipsychotic medications. These side effects are acute dystonic reactions, akathisia, dyskinesia, neuroleptic malignant syndrome, parkinsonism, and various tardive syndromes. Dyskinetic movements are characterized by hyperkinetic, repetitive movements of the extremities, facial, and perioral muscles. In rare cases, dyskinetic movements may occur with the use of selective serotonin reuptake inhibitors (SSRIs) through an indirect D2 blocking mechanism. In this case report, dyskinesia observed after increasing the dose of fluoxetine in a patient using methylphenidate and risperidone was presented. It was thought that the SSRI inhibited dopamine activity through dopamine receptor antagonism and that SSRIs may increase risperidone concentrations by inhibiting CYP450 metabolism. There are few cases reported in the literature regarding SSRI-induced dyskinesia, information is limited on this subject. This case report highlights the potential dyskinesia side effect of SSRIs.
Keywords: Dyskinesia; extrapyramidal side effects; dopamine receptor blockers; fluoxetine.

ÖZ

Psikiyatrik ilaçlara bağlı hareket bozuklukları, çoğunlukla antipsikotik ilaçlar olan dopamin reseptör bloke edici ilaçların yan etkileriyle ilgilidir. Bu yan etkiler, akut distonik reaksiyonlar, akatizi, diskinezi, nöroleptik malign sendrom, parkinsonizm ve çeşitli tardif sendromlara neden olabilir. Diskinetik hareketler, ekstremitelerin, yüz ve perioral kaslarının hiperkinetik, tekrarlayan hareketleri ile karakterize edilir. Nadir durumlarda dolaylı bir D2 blokaj mekanizması yoluyla selektif serotonin gerialım inhibitörleri (selective serotonin reuptake inhibitors, SSRIs) kullanımı sonucu diskinetik hareketler ortaya çıkabilir. Bu olgu sunumunda, metilfenidat ve risperidon kullanan bir hastada fluoksetin doz artışı sonrasında görülen diskinezi sunulmuştur. SSRI'nın, dopamin reseptör antagonizması ile dopamin aktivitesini inhibe ettiği ve SSRI'ların CYP450 metabolizmasını inhibe ederek risperidon konsantrasyonunda bir artışa neden olabileceği düşünülmüştür. Literatürde SSRI kaynaklı diskinezi vakaları az sayıdadır, bu alanda kısıtlı bilgi mevcuttur. Bu olgu sunumu, SSRI'ların potansiyel diskinezi yan etkisine dikkat çekmektedir.
Anahtar kelimeler: Diskinezi; ekstrapiramidal yan etkiler; dopamin reseptör blokerleri; fluoksetin.

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INTRODUCTION

Movement disorders caused by psychiatric drugs are predominantly linked to the adverse effects of medications that inhibit dopamine receptors, mostly antipsychotics. Acute dystonic reactions, akathisia, dyskinesia, neuroleptic malignant syndrome, parkinsonism, and tardive syndromes are some of the extrapyramidal movement disorders that can result from these drugs' blocking of dopamine D2 receptors (1,2). Hyperkinetic, repetitive movements of the limbs, facial, and perioral muscles characterize

dyskinetic movements (2). Drug-induced dyskinesias are classified based on the timing of onset, duration of the abnormal movements, and their phenomenological characteristics. In this classification, they are primarily divided into acute and tardive types. Acute movement disorders and dyskinesias emerge within days to weeks after the initiation of the medication or an increase in its dosage. This type of disorder is generally common and tends to resolve upon discontinuation of the drug or dose reduction (3,4). Tardive dyskinesias, on the other hand, are chronic movement disorders that can develop following a long period of exposure to medication, and in the vast majority of cases, persist after discontinuation of the drug and do not regress (5,6). According to the DSM-5, a diagnosis of tardive dyskinesia requires involuntary movements that persist for at least one month after discontinuation or reduction of the treatment.

Selective serotonin reuptake inhibitors (SSRIs) are frequently prescribed medications in outpatient settings for depression, anxiety, and other related disorders. Although they are generally considered to be well tolerated, recent published data suggest that these drugs may cause movement disorders, including extrapyramidal system side effects such as dystonia, akathisia, parkinsonism, and tardive dyskinesia (7,8). The possible causes and mechanisms of SSRI-induced extrapyramidal symptoms (EPS) continue to be investigated; however, the interaction between serotonin and dopamine in the basal ganglia is considered significant. SSRIs increase serotonin levels in synapses, which in turn affects the dopaminergic system. Specifically, serotonin can inhibit nigrostriatal dopamine neurons by influencing 5HT2C receptors, leading to a decrease in dopamine activity (2,8). This is thought to mimic dopamine receptor antagonism and can cause movement disorders similar to those induced by antipsychotic medications. Tardive dyskinesia is typically associated with long-term dopamine receptor antagonism; however, it has been proposed that SSRIs may also trigger dyskinesia through several different mechanisms, including dopamine receptor hypersensitivity and oxidative stress in striatal neurons (6,8-10).

Fluoxetine, a widely used SSRI, differs from others of its class in terms of its pharmacokinetic properties. Fluoxetine has a marked inhibitory effect on cytochrome P450 2D6 (CYP2D6), an important enzyme in the metabolism of many psychiatric drugs, such as risperidone and other antipsychotics (11,12). By inhibiting CYP2D6, fluoxetine can increase plasma concentrations of concurrently used antipsychotics, thereby increasing the risk of EPS (12,13). Additionally, fluoxetine has been shown to enhance stimulant-induced gene expression in the striatum, which can explain its contribution to movement disorders when used in combination with medications such as methylphenidate (14,15).

Fluoxetine-induced dyskinesia is not commonly observed and has been reported in only a few cases in the literature. Several case reports have described movement disorders associated with fluoxetine, ranging from orofacial dyskinesia to more complex motor disturbances (9,16). A review of the literature reveals that the majority of reported cases belong to adult patients, and there is a significant lack of research regarding fluoxetine-induced dyskinesia in the pediatric population.

This case report aimed to highlight a dyskinesia that developed after an increase in fluoxetine dosage in a pediatric patient who was concurrently using risperidone and methylphenidate. This case underlines the necessity of careful monitoring and meticulous attention to EPS in young patients undergoing polypharmacy. The mechanisms of SSRI-induced dyskinesia, the role of CYP2D6 inhibition, and the increased risks associated with polypharmacy were also discussed.

CASE REPORT

A 9-year-old male patient was referred to our clinic due to irritability, behavioral problems, low self-esteem, and family issues. He had been diagnosed with attention deficit hyperactivity disorder (ADHD) at an external center and was taking methylphenidate 27 mg/day, atomoxetine 36 mg/day, and risperidone 1 mg/day at the time of examination. Although the patient had been using the current treatment for approximately one year, issues such as self-confidence problems, anger outbursts, and behavioral problems persisted. The first Conners scale, filled out by the teacher, indicated inattention, hyperactivity, and oppositionality. Considering that the patient did not benefit from the current treatment, it was planned to gradually taper and discontinue atomoxetine over 8 weeks, and the patient was closely monitored. No withdrawal symptoms such as restlessness, sleep disturbances, dizziness, or feelings of lightheadedness were observed. Since ADHD symptoms persisted, the dose of methylphenidate was increased to 36 mg/day at the 8th week. At the follow-up visit two months later, according to information obtained from the teachers and the mother, it was observed that the patient's attention at school had improved and academic performance had increased; however, problems with teachers, behavioral issues, and aggressive behaviors towards peers continued. The Conners teacher subscale scores were calculated as 1 point in inattention, 2 points in hyperactivity, and 9 points in oppositionality. Due to these reasons, the risperidone dose was increased to 1.5 mg/day. During the patient’s regular follow-up visits, it was learned that the mother and stepfather had recently divorced. Additionally, it was noted that the patient changed schools after the divorce and experienced adjustment problems at the new school. During the examination, depressive symptoms were also observed. On the revised child anxiety and depression scale (RCADS), the depression subscale was notably elevated, and social phobia was found to be above the threshold. Therefore, it was decided to add fluoxetine 10 mg/day to the treatment. After the treatment, the patient's mother and teachers reported a significant benefit; however, depressive findings were still observed in the RCADS. Therefore, it was decided to increase the fluoxetine dose to 10 mg on one day and 20 mg on the following day, alternating. During a follow-up visit after increasing the dose, his mother noted the presence of oral movements, including lip licking and repetitive opening and closing of the mouth, following the administration of 20 mg of fluoxetine. In the abnormal involuntary movement scale (AIMS) test evaluation of facial and oral movements, the patient exhibited moderate involuntary movements in the lips and perioral area (score=3) and jaw (score=3) while the tongue showed mild involuntary movements (score=2). The AIMS result supported the diagnosis of a movement

disorder. The Naranjo adverse drug reaction probability scale was applied to the patient; a significant score of 6 was obtained, indicating a strong possible relationship between the fluoxetine dose increase and the development of dyskinesia. His mother reduced the dose to 10 mg, after which these movements were no longer observed. Discontinuation of fluoxetine treatment and a switch to another SSRI were planned; however, the mother stated that they had benefited greatly from the current treatment and dosage and did not want any change. Supportive interviews were added, and the current treatment was continued. Follow-ups are ongoing in our clinic.

DISCUSSION

In this case report, dyskinesia in the oral region was observed in a pediatric patient following an increase in the fluoxetine dose. This scenario indicates two primary mechanisms: the direct influence of serotonin on dopaminergic pathways (5,8) or heightened exposure to concurrently administered antipsychotics resulting from enzyme inhibition (8,12,13). While it is essential to primarily examine these mechanisms, we will also explore additional putative mechanisms in depth.

Approximately 11.3% of SSRI-related EPS adverse effect cases contain dyskinesia-like symptoms (2). In 1979, Jeste et al. (17) published one of the initial articles on persistent and transient dyskinesia, wherein 286 individuals over the age of 50, who were administered antipsychotics and tricyclic antidepressants, were evaluated for dyskinesia during a one-year period. Tardive dyskinesia was identified in 23 patients, while dyskinesia in 2 patients was attributed to tricyclic antidepressants, and both instances were linked to temporary dyskinesia. A literature analysis from 1987 assessed 50 adult patients diagnosed with depression, all undergoing different antidepressant therapies (doxepin, clomipramine, thioridazine, amoxapine, etc.). Dyskinesia in the buccolingual region occurred in two of the 50 patients, whereas one patient displayed dyskinesia affecting the limbs and trunk. Dyskinetic movements diminished following discontinuation or reduction of antidepressant medication in these three patients (4). A 1991 letter to the editor detailed a case of an adult patient with schizoaffective disorder, who had a history of antipsychotic medication but no previous movement disorder, and who developed buccolingual dyskinesia following 8 days of fluoxetine administration. In this case, the buccolingual movements commenced a decline within 10 days following discontinuation of fluoxetine (16). A case documented by Mander et al. (9) in 1994 was an older woman who experienced oral dyskinesia immediately after starting fluoxetine. The patient was receiving a low dose of haloperidol, and the dyskinesia manifested soon after the beginning of fluoxetine, resolving promptly upon discontinuation of the medication. In 1996, Dubovsky and Thomas (18) reported three cases of adult patients treated with fluoxetine for depression who showed aberrant movement patterns suggestive of dyskinesia. In the first case, atypical facial movements occurred four weeks following the incorporation of fluoxetine into a treatment regimen of doxepin and lithium, and decreased following the discontinuation of fluoxetine. For the rest cases, movement disorders affecting the mouth, face, and hands that occurred with fluoxetine endured for months after discontinuation. A 1995 study showed that EPS can be associated with fluoxetine in 15 patients with depression who were simultaneously administered antipsychotics and tricyclic antidepressants; one patient had tardive dyskinesia, and movement disorders improved following the discontinuation of fluoxetine (19).

In a case reported by Chen and Swope (10) in 2005, clinicians identified an unusual complex movement disorder characterized by fluoxetine-induced orofacial dyskinesia, jaw-closing dystonia, bruxism, and parkinsonism. This case underscored that SSRIs may induce complex movement disorder syndromes impacting the oral and facial regions.

The study from 2015 revealed that among 91 individuals using SSRIs or SNRIs, 17 exhibited akathisia, 18 displayed dyskinesia, 27 experienced dystonic reactions, 19 showed parkinsonism, and 10 reported various extrapyramidal adverse effects. These side effects typically manifested soon after initiating or escalating the dosage of antidepressants, with 22% of these individuals simultaneously utilizing antipsychotic drugs. Given that antidepressants are rarely linked to EPS, any EPS manifestations associated with these medications may be misinterpreted and improperly addressed (13).

Similar to other SSRIs, fluoxetine may disturb the balance between dopamine and serotonin, perhaps resulting in dyskinesia. SSRIs elevate serotonin levels in the raphe nuclei, resulting in enhanced GABA release through 5HT2C receptor activation, which subsequently inhibits nigrostriatal dopamine neurons; this serotonin-induced dopamine suppression in the basal ganglia can replicate the effects of dopamine blockade (2,13,20). In summary, the serotonergic inhibition caused by fluoxetine leads to a functional shortage of dopamine, perhaps resulting in dopamine receptor supersensitivity over time. Exaggerated responses at postsynaptic receptors can result in dyskinesia as a result of chronic dopamine blockade, which can contribute to upregulation of dopamine receptors (6,13). Another explanation proposes that elevated serotonin levels resulting from fluoxetine administration specifically bind to 5HT2A receptors, hence augmenting dopamine release, which may lead to movement problems (21). The escalation of the SSRI dosage may have exacerbated the serotonergic-dopaminergic imbalance, perhaps triggering dyskinetic movements. An additional hypothesis emphasizes the existence of oxidative stress as a consequence of antidepressant-induced dopamine receptor blockade, which increases dopamine metabolism. This results in the generation of free radicals in the basal ganglia, potentially causing movement problems. This oxidative stress may exert a direct neurotoxic effect (6). Another potential mechanism is the suppression of cytochrome P450 (CYP450) metabolism by SSRIs, which may elevate risperidone levels and hence increase the risk of movement disorders (8). Fluoxetine and its active metabolite norfluoxetine are strong inhibitors of CYP2D6 and, to a lesser degree, inhibit CYP2C9, CYP2C19, and CYP3A4. This inhibitory action may lead to elevated plasma concentrations and adverse effects of antidepressants and antipsychotics. Risperidone undergoes metabolism via CYP2D6 and CYP3A4 enzymes. Fluoxetine's inhibition of some CYP enzymes may elevate blood concentrations of risperidone (8,12,22). Administration of risperidone or any medication metabolized by CYP2D6

when combined with an elevated SSRI dosage may result in unexpectedly heightened antipsychotic levels, thereby amplifying the risk of dyskinesia. The complex interaction between serotonin modulation and CYP enzyme inhibition must be acknowledged in fluoxetine-induced movement disorders (11,12).

Distinguishing these mechanisms is difficult. The rapid onset of dyskinesia after increasing the fluoxetine dosage and its subsequent resolution upon discontinuation imply that the underlying cause is likely serotonergic dysregulation rather than enzyme inhibition. This may correspond to earlier data indicating that fluoxetine induced dyskinesia rapidly resolves upon discontinuation of the medication and does not remain as an extended effect associated with antipsychotic activity (9,10).

Dyskinesia can also occur in different situations; it is known that involuntary movement problems can occur in individuals who do not receive medical treatment, called spontaneous dyskinesia (23). In our case, the temporal link between the beginning of aberrant orofacial movements after increasing the drug dose and recovery following dose decrease does not support spontaneous dyskinesia. Dyskinesia can also occur when patients' previous medication doses are lowered or withdrawn completely. These dyskinesias are classified as withdrawal-emergent dyskinesias. They are generally observed to disappear spontaneously within weeks or months (24). This is believed to result from the overexpression of dopamine D2 receptors and postsynaptic receptor supersensitivity (5). In this case, because six months had passed since the discontinuation of the medication, it was tapered slowly, and the movement disorders appeared after the increase in fluoxetine dosage, the resulting dyskinesia was not considered to be withdrawal-related.

Another consideration in SSRI-induced movement disorders is polypharmacy, particularly when SSRIs are co-prescribed with psychostimulants like methylphenidate. Psychostimulants alone can induce tic-like dyskinetic movements, and when used in conjunction with SSRIs, they may further aggravate dopaminergic dysregulation, potentially resulting in EPS-like symptoms (14). Literature reviews indicate that the co-administration of SSRIs and methylphenidate may modify dopamine levels via neurotransmitter gene regulation, potentially leading to EPS-like adverse effects. Research in the field is continuing, however, evidence remains in an early stage of development (14,15). The suggested pathophysiology involves serotonergic overactivity, which removes inhibition in motor control circuits or causes oxidative stress in striatal neurons, resulting in involuntary mouth movements and stereotypies. Research with animals has demonstrated that fluoxetine can augment the gene expression induced by methylphenidate in the striatum, thereby elevating the expression of genes such as c-Fos and Zif268 (Egr-1), which are associated with the regulation of movement (14,15). This indicates that the interplay of SSRIs, methylphenidate, and antipsychotics may foster a pharmacodynamic environment that predisposes individuals to movement problems. Literature reviews on combination therapy reveal that polypharmacy elevates the risk of EPS, requiring caution from clinicians. Despite the limited clinical studies on this triple combination, diligent patient monitoring is essential due to the associated risks.

In contrast to classic tardive dyskinesia, SSRI-induced dyskinesias may manifest quickly and go away once the medication is stopped. The rapid emergence and reversibility are typically linked to non-tardive dyskinesias, so differentiating them from tardive dyskinesia, which endures for a minimum of one month following the cessation of the treatment. The prevalence of persistent dyskinesias linked with antidepressants is so low that it is difficult to establish a link between the two conditions. Consequently, antidepressant-induced dyskinesia may be seen as an acute, reversible condition rather than a chronic, persistent problem (25,26). In our case, the onset of movement disorder shortly after initiating fluoxetine and the resolution of these movements following dose reduction support this information.

This case has been evaluated as an instance of dyskinesia developing after an increase in the fluoxetine dose, supported by a high score on the Naranjo adverse drug reaction probability scale applied to the patient. Nowadays, it is accepted that involuntary movement problems can also arise in people without antipsychotic usage; these medical conditions are called spontaneous dyskinesia (23). The probability of spontaneous dyskinesia is low given the temporal link between the start of aberrant orofacial movements following the fluoxetine dose increase and the dyskinesia recovery following dose reduction. By affecting dopaminergic pathways in brain areas linked to movement through serotonergic modulation, fluoxetine may induce relative dopamine deficit and receptor supersensitivity, therefore triggering orofacial dyskinesia (5). Serotonergic dysregulation is considered the most likely cause, as the rapid remission of dyskinesia with fluoxetine dose reduction suggests an acute pathogenic process rather than enzyme inhibition or gene control alterations.

In most cases, dyskinesia caused by gene control modifications by methylphenidate and fluoxetine develops more slowly. This is related to the slow onset of changes in gene expression and neuroplastic processes throughout time (14,15). In this case, the little time from the start of symptoms and their remission upon drug cessation mostly rules out this hypothesis. Although risperidone accumulation via CYP2D6-mediated inhibition is a mechanism worth investigating in terms of gene regulation (22), the rapid onset of symptoms and their remission upon withdrawal suggest that serotonin-dopamine imbalance is more important. A survey of the literature reveals that many cases of fluoxetine-induced dyskinesia have been documented since the 1970s. There is little information on pediatric cases, which emphasizes the need for caution when prescribing SSRIs to younger patients. Moreover, polypharmacy, especially the mix of stimulants, antipsychotics, and SSRIs, has to be controlled carefully. Future studies should try to better comprehend SSRI-induced dyskinesia, particularly in groups including children, adolescents, and people using multiple psychiatric drugs.

CONCLUSION

Dyskinesia caused by SSRIs is rare in the literature. The patient's clinical presentation, combined with the onset of symptoms following an increase in the fluoxetine dosage and their improvement after reducing the dose, strongly suggests a diagnosis of SSRI-induced dyskinesia.

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