

A Case Report and Literature Review of Fluoxetine-Induced Akathisia in a Patient with Depression

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ABSTRACT

Akathisia is a movement disorder characterized by an overwhelming and uncontrollable need to move. It is generally connected with the use of antipsychotic medications, with just a few cases found to be produced by selective serotonin reuptake inhibitors. Although reported previously, fluoxetine-induced akathisia remains uncommon and often underrecognized. We report a case of a 24-year-old male with major depressive disorder, who presented with fluoxetine-induced akathisia three weeks after increasing his fluoxetine dose. The Barnes Akathisia Scale confirmed moderate akathisia (score of 4), and his symptoms were severe enough to keep him awake at night. Treatment consisted of discontinuing the offending medication, and symptoms subsided after two weeks. This report highlights that early detection and treatment are crucial, since untreated akathisia can negatively impact a patient's quality of life and medication adherence or, as reported in many studies, increase the risk of aggressive or suicidal behaviour.

Keywords: Selective serotonin reuptake inhibitor, movement disorder, fluoxetine, akathisia, depression.

INTRODUCTION

Selective serotonin reuptake inhibitors (SSRIs) are the first-line pharmacotherapy for managing some of the most common psychiatric conditions, including depression and anxiety [1]. Common adverse effects of SSRIs include impaired sexual functioning, drowsiness, weight gain, dry mouth, insomnia, fatigue, nausea, and dizziness [2]. One less acknowledged adverse effect is akathisia [3].

The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) defines akathisia as "subjective complaints of restlessness, often accompanied by objective excessive movements (such as continuous leg movements, rocking from foot to foot, pacing, or an inability to sit down and remain still)" [4]. In many cases, the symptoms of akathisia are more disturbing than the underlying mental health issue. Akathisia is frequently misdiagnosed, and symptoms are often attributed to underlying anxiety or mental health problems. As a result, physicians continue to prescribe psychiatric medicines, which can worsen akathisia symptoms and create a vicious cycle [5].

Herein, we report a case of a 24-year-old male with major depressive disorder, presenting with akathisia after adjusting his fluoxetine dose, to reinforce clinical awareness of this uncommon yet significant adverse effect despite previous reports.

CASE PRESENTATION

A 24-year-old non-smoker, single male patient had a known case of major depressive disorder for 6 months,

for which he was taking 20 mg/day of fluoxetine and attending psychotherapy sessions. His fluoxetine dose was increased from 20 to 40 mg/day due to fatigue and concentration problems. During his follow-up appointment, the patient complained of restlessness and repetitive shifting from one foot to another. These symptoms, which he noticed three weeks after increasing his fluoxetine dose, were severe enough to keep him awake at night. His past medical history was negative, except for depression. He was not taking any other medication and had no history of drug abuse.

On observation, he appeared to be slightly anxious and in mild distress. He was able to sit but shifted his foot frequently. On physical examination, no other extrapyramidal symptoms were detected. The Barnes Akathisia Scale confirmed moderate akathisia (score of 4). His baseline investigations, including complete blood cell count, thyroid function test, liver function test, and renal function test, were normal.

Based on his clinical examination, the patient was diagnosed with fluoxetine-induced akathisia. Treatment included discontinuing the fluoxetine and scheduling a follow-up consultation. His akathisia symptoms progressively improved and disappeared completely after two weeks. However, he complained of depression symptoms, such as low mood, fatigue, and difficulty concentrating. The patient was started on 10 mg/day of escitalopram. Three weeks later, his depressed symptoms had gradually improved, and his akathisia symptoms had not reappeared.

DISCUSSION

Extrapyramidal symptoms (EPS), usually referred to as drug-induced movement disorders, include akathisia, tardive dyskinesia, dystonia, and Parkinsonism [6]. EPS are typical side effects of neuroleptics; however, they are

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less often associated with antidepressant medications such as selective serotonin reuptake inhibitors (SSRIs) [6, 7]. Akathisia is the most prevalent EPS linked with SSRIs. Moreover, SSRI-induced akathisia often seems milder but is clinically indistinguishable from neuroleptic-induced akathisia [7].

Although the exact mechanism of the link between akathisia and SSRIs is unknown, it has been proposed that increased serotonin availability may indirectly decrease dopamine release in the striatum by increasing 5-HT₂ receptor activation [8]. Furthermore, it is controversial whether some SSRIs are more likely than others to trigger akathisia, although fluoxetine receives the most attention. It is unclear if this is because fluoxetine is the most prescribed medication or if the variations in affinity for 5-HT₂ receptors amongst SSRIs may explain the disparities in the frequency and intensity of EPS observed in patients [8, 9].

Numerous factors may contribute to SSRI-induced akathisia, including recent dosage increases, previous history of akathisia, taking multiple akathisia-causing drugs, and taking SSRIs with longer half-lives [3, 10]. The Barnes Akathisia Rating Scale (BARS) can be used to evaluate a patient with suspected akathisia [11]. Diagnosis usually depends on both objective and subjective components [3]. Patients commonly describe akathisia as a sensation of inner restlessness, and symptoms typically include frequent leg crossing, weight shifting, or walking in place. These symptoms can appear days or weeks after starting or adjusting a medication [3, 10]. While akathisia is an uncommon extrapyramidal manifestation of SSRIs, it is usually underdiagnosed or misdiagnosed among patients who use these medications, as symptoms of akathisia are often confused with agitation, anxiety, or even restless leg syndrome [3, 5]. Moreover, patients may be unable to define their feelings of inner restlessness until specifically asked [3].

A review of the literature revealed that a few occurrences of akathisia related to fluoxetine, with varying symptom intensity [9, 12]. In our case, the patient developed akathisia three weeks after increasing his fluoxetine dosage from 20 to 40 mg/day. He described it as leg restlessness and a continual craving to move, which caused sleeplessness. Similarly, Ajufo *et al.* reported the case of an elderly female who experienced fluoxetine-induced akathisia that kept her up at night [12]. On the other hand, Hansen and Rothschild documented cases of severe akathisia and suicidal thoughts with fluoxetine treatment [9, 13]. Although the pathophysiology of akathisia and suicidal ideation is unknown, it may appear in response to the akathisia-induced restlessness. Hamilton reported on one patient, "Despite her mood being good, she was afraid that she would kill herself because of these restless and out-of-control feelings" [14].

In most cases, the initial step to treat SSRI-induced akathisia is to discontinue the offending drug, which will resolve symptoms within 2 to 6 days [3]. This method effectively resolved akathisia symptoms in our patient. However, the literature presents alternatives, including reducing the dose, switching to another antidepressant, or utilizing additional medications such as beta-blockers, anticholinergic medicines, or benzodiazepines [3, 5]. Notably, the fact that propranolol has proven successful in the treatment of akathisia but not EPS provides additional evidence that separate mechanisms are involved in SSRI-induced akathisia etiology [3]. The inadequate understanding of SSRI side effects and SSRI-specific mechanisms raises clinically significant concerns [2].

This report emphasizes the significance of identifying SSRI-induced akathisia early, since akathisia has a significant impact on a patient's psychological health and quality of life. The consequences can be severe, and some studies have connected akathisia to patient noncompliance and suicidality. Moreover, healthcare providers should keep this side effect in mind while using SSRIs, because akathisia will intensify if antipsychotic drugs augment the regimen. More research is required to understand the prevalence and mechanism of fluoxetine-induced akathisia, as well as to confirm its association with suicidal behaviour.

CONCLUSION

Fluoxetine-induced akathisia is similar to neuroleptic-induced akathisia, although it is uncommon and often milder. To avoid negative impacts on patient compliance and subjective well-being, akathisia must be identified early and managed appropriately. This case underscores the importance of maintaining clinical caution for SSRI-induced akathisia, even though it has been previously reported, to enhance early recognition and patient safety.

CONSENT FOR PUBLICATION

Written informed consent was obtained from the patient to publish this case report.

CONFLICT OF INTEREST

The author declares no conflict of interest.

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