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Extrapyramidal Symptoms After Fluoxetine

Dr Amitabh Saha
Dept of Psychiatry,Command Hospital,Pune,India

CASE REPORT

ABSTRACT

Selective Serotonin Reuptake Inhibitors (SSRIs) are commonly prescribed medicines for depression. It has certain side effects but having extra pyramidal symptoms due to use of Cap Fluoxetine is not commonly seen in many patients. This study introspects and highlights one such case and reminds us to know and understand the various side effects that use of psychotropics have in regular practice .

Keywords: Extra pyramidal symptoms, Selective serotonin Reuptake inhibitor,side effects, Fluoxetine-induced EPS,antidepressant, akathisia, dystonia

INTRODUCTION

Extra pyramidal symptoms (EPS) are an uncommon side effect of Selective Serotonin Reuptake Inhibitors (SSRIs)[1,2]. They are commonly prescribed antidepressants. Among the SSRIs, Fluoxetine has been associated with most case reports of EPS[3], though other SSRI, particularly, Paroxetine[4] and Sertraline[5,6] has also been reported in certain cases. Most of these reports are from other countries, and relevant searches have not yet reported such occurrences in India, though, it is certain that there would be examples of such adverse effects in clinical practice. We present such a case.

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MATERIALS AND METHODS

A 32 yr old serving person, native of Indian village,educated up to 10thstd, married, who presented with features of low mood, heaviness of head, reduced sleep, anxiety, paresthesia of extremities and poor concentration of few months duration. He was diagnosed as Depressive Episode (moderate) and treated with Nortryptiline up to 100 mg/d with adequate response. While he was on sick leave for convalescence, he discontinued the drug due to dryness of mouth, dysphoria, constipation and premature ejaculation. When he returned back from leave, he was having residual depressive symptoms, and started on Cap Fluoxetine 20 mg/d wef 02 May'12. He showed satisfactory remission of symptoms and later, discharged on 22 May'12. He reported to hospital on 29 May'12,

with difficulty in speaking fluently, difficulty in opening mouth and swallowing food, especially solids, stiffness of extremities and unable to walk properly since 3 days duration. His colleagues would enquire whether he was drunkduring day-time. Symptoms were of insidious onset and progressive. He felt anxious, restless and decided to seek medical help.

General exam and vitals were stable. Neurological exam showed bradykinesia, cog-wheel rigidity of upper limbs, absent arm swing while walking, hyper salivation and slurring of speech, tremors and hypographia. He had no cranial nerve deficits, cerebellar signs or cognitive deficits. Hematological parameters were normal, Blood sugar random 104 mg/dl, ECG was normal and MRI Scan of Brain showed normal study. He was managed by Physician and Psychiatrist. Cap Fluoxetine was stopped considering it as a case of Fluoxetine-induced EPS, and he was started on anticholinergics – Trihexyphenidyl 10 mg/d and Benzodiazepines – Clonazepam 2 mg/d. He started showing improvement in symptoms over the next 3-4 days. Emotional distress was better, he was able to open mouth and swallow solids, stiffness of limbs reduced. He was later, placed on Mirtazepine 30 mg/d and discharged after 10 days.

RESULTS AND DISCUSSION

EPS are most commonly seen as an adverse effect of antipsychotics, usually the first generation ones, although several other classes of drugs viz. anticonvulsants, antidepressants, Metoclopramide, etc are also associated with the same[7]. Among the antidepressants, both the tricyclic[8] and SSRI group has reports of causing EPS in anecdotal case reports, though more reports are available with SSRIs, and the signs does not show much changes over time[9]. A recent review of literature using FDA AERS (Adverse Event Rating Scale) showed preponderance of EPS with SNRIs (Duloxetine 66%), followed by SSRI and Bupropion[10]. In a review of 71 cases of SSRI-induced EPS, it was found that the most common was akathisia, followed by dystonia, Parkinsonism and tardive dyskinesia-like states [3,7,10,16]. Although no consistent risk factor has been associated with the emergence of EPS, it was found that older age group, females, use of concomitant drugs such as anti-psychotics, neurological illness such as Parkinsonian Disease (PD)[11] and autistic children [12] were implicated.

As already stated, among the SSRIs, Fluoxetine was the most common to cause EPS. Majority of the side effects occurred within the first month of therapy [13]. Research has now established a distinct form of melancholic or endogenous depression with biological underpinnings similar to those of basal ganglia disorders such as PD, which are more often implicated in such cases[14]. The neuropathophysiological mechanism for SSRI-induced EPS is still not properly understood, though the stimulation of $5HT_{2A}$ receptors in basal ganglia may lead to motor movement disorders. Increased serotonin activism has been shown to inhibit both the nigro-striatal and tubero-infundibular dopaminergic neuronal pathways, causing EPS[2,15]. This has been further confirmed by levels of low Homovanillic acid (HVA) in CSF and increased Serum Prolactin in patients after Fluoxetine-induced EPS[16]. The factors which single out Fluoxetine as common among the SSRIs could be due to its affinity for $5HT_{2C}$ receptors and risk of accumulation due to long half-lives [17]. It has been postulated that Fluoxetine-induced EPS, particularly akathisia, may be a mediator of de-novo suicidal ideation [18,19].

In our case, the emergence of EPS could have been an incidental finding or due to some organic neurological disorder. However, the latter was ruled out by relevant investigations. His signs started showing improvement after stopping Fluoxetine. Hence, a diagnosis of Fluoxetine-induced EPS seemed appropriate in this case.

CONCLUSION

EPS due to Fluoxetine can be understood as an undesirable action of serotonin in undesirable pathways and receptor subtypes. Fortunately, such adverse effects are more of a nuisance than a danger [20]. Recent studies have shown only a moderate association [21,22], and for most patients, the benefit of Fluoxetine far outweighs the potential problem. The rare occurrence of EPS as established in recent studies does not preclude SSRIs even in patients with PD. However, one should always keep close observation for emergence of EPS in patients on SSRIs, and immediate cessation of the offending drug, neuroimaging and treatment must be instituted for the benefit of such patients. Treatment options included addition of centrally-acting beta-blocker, a benzodiazepine or an anti-cholinergic agent [23].

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