

A Case Report on Escitalopram Induced Parkinsonism in a Young Girl

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Abstract: *SSRIs especially Escitalopram are widely used by general physicians, neurologists, and psychiatrists for a myriad of different symptomatology. Common side effects like nausea, vomiting, and sleep disturbance are well documented but rare side effects like extrapyramidal syndrome or movement disturbances tend to go unreported because of many reasons, from the use of combination psychotropics or to the presence of neurological diseases in the patients. In this case report, we present a case of EPS induced by Escitalopram which was promptly diagnosed and managed thoroughly. Early diagnosis and treatment are necessary to improve the functioning which is already compromised due to the psychiatric disease. Furthermore, this report tries to bring much-needed attention to non-psychiatrists to this phenomenon.*

Keywords: SSRI, Escitalopram, EPS

1. Introduction

SSRIs are widely used by general practitioners, physicians, and psychiatrists to treat various facets of psychological disturbances. They have a relatively safer side effect profile than conventional antidepressants. Although side effects like nausea and sexual dysfunction are widely recognized but extrapyramidal symptoms still don't hover around a clinician's mind. This may be due to the lack of its representation in mainstream textbooks or the rarity of presentation in the clinics. Recent literature suggests a paradigm shift in understanding these adverse effects of SSRIs. Incidences of EPS with the use of various SSRIs are as follows: Escitalopram (12%), Sertraline (11%), Paroxetine (10%), and fluoxetine (8%)^[1]. Pathophysiological changes in the central dopamine receptors in the basal ganglia (nigrostriatal pathway) have been postulated as a cause of SSRI-induced EPS^[2]. In patients with ongoing akathisia, the role of dopamine receptors in the mesocortical pathway has also been implicated^[3].

We bring forward this case report prompting the dire need for awareness.

2. Case Report

Miss A, a 15-year-old female had consulted Psychiatry OPD 6 months back with symptoms of depressed mood, anhedonia, decreased energy, difficulty concentrating in studies, decreased sleep, decreased appetite. Her MSE revealed decreased psychomotor activity, depressed affect, thought content revealed depressed ruminations. Her HAM-D^[4] was 14. General physical examination and systemic examination were normal. Routine blood investigations were done which were normal. After talking about the importance of pharmacological intervention at the earliest with her parents, she was started on Tablet Escitalopram 10 mg once daily. She has also been given Tablet Zolpidem 10 mg for

sleep disturbance. Follow-up after 3 weeks revealed improvement in symptoms. At 2 months of follow-up, she was free of all symptoms and had returned to her previous level of academic performance. It was planned to have follow-up every month till 6 months, then taper the dose of Escitalopram gradually and stop treatment by 9-12 months. During the 6 month follow-up after starting Escitalopram, she presented with decreased body movement rigidity of the upper limbs, pooling of saliva in the oral cavity, postural tremors, and slurring of speech. Routine biochemical tests, EEG, MRI Brain were all normal in the patient. Modified Simpson Angus Scale (MSAS)⁽⁵⁾ was 8 which is a clinically significant degree of movement disorder. Relevant history of head injury, movement disorder, and family history of parkinsonism was negative. No such reactions were observed in this patient in the past. So a diagnosis of Drug-Induced Movement disorder was made and worked upon. Escitalopram was discontinued immediately, and Tablet Trihexyphenidyl 2 mg twice daily was started. The patient was reassured regarding the reversible nature of the symptoms. In the ensuing 2 weeks, all the above symptoms decreased considerably. Tablet Trihexyphenidyl was stopped after a week and the patient was started on Tablet Sertraline 50 mg once daily. It was increased to 100 mg in the following week for maintenance of depressive symptoms. It was continued for the next 6 months and eventually tapered and discontinued. Naranjo probability scale applied retrospectively revealed a score of 5 pointing towards probable drug reaction.

3. Discussion

Recent data indicate SSRIs to be more common than earlier thought to cause EPS. 1 in 1000 who use SSRIs present with EPS to outpatient and emergency clinics⁽⁶⁾. In a country like India, the statistics can be more alarming as most patients with mood disorders still visit physicians who relatively are not exposed to the side effects of psychotropics as compared to psychiatrists in their routine practice. Various risk factors

are attributed to SSRI-induced parkinsonism like advanced age, female gender, concurrent use of other neuroleptics, head trauma, etc. ⁽¹⁾. Our patient was young with no history of neuroleptic intake other than the perceived offending agent, had no history of head trauma, no family history of movement disorder. A similar patient profile was brought forward in a recent case report in India ⁽⁷⁾.

Most reports give evidence of extrapyramidal symptoms with the use of SSRIs occurring in a month ⁽¹⁾. Our case was presented after 6 months of drug initiation. EPS due to Escitalopram has been implicated even as late as 18 months ⁽⁷⁾. So clinicians should be vigilant throughout treatment as complications of drug use can present at any time.

Extrapyramidal symptoms in a young patient treated for depression should be promptly looked upon for differentials like Young-onset Parkinsonism, Wilson's disease, and very rarely psychological disorders like Conversion disorder. Absence of family history, prompt relief of symptoms after discontinuation of the offending drug, absence of KF ring, normal urine copper, and presence of symptoms when distracted and continuous symptoms rule out the above diagnostic-induced inhibition of tyrosine hydroxylase has been postulated to be a contributory cause in the evolution of EPS ⁽⁸⁾. The finding is yet to resonate in human studies. Case reports like ours should promote research in understanding the idiosyncrasy associated with SSRI-induced EPS.

4. Conclusion

In conclusion, our report aims at drawing valuable notice of clinicians regarding the rare adverse event associated with Escitalopram. Distinctive findings of the report include early age of onset, absence of risk factors like head trauma, preexisting movement disorders, comorbid agitation, and lack of family history. EPS symptoms can have a deteriorating impact on drug adherence and the overall health of the patient. So early recognition and prompt treatment should be the utmost responsibility of a clinician.

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