Escitalopram-induced severe akathisia leading to suicide attempt

ABSTRACT

Akathisia, a distressing adverse reaction, is usually underdiagnosed or misdiagnosed in patients, who are treated with selective serotonin reuptake inhibitors (SSRIs). Escitalopram-induced akathisia is rarely reported in the literature. We report a case of severe akathisia leading to a suicide attempt in a 25-year-old male induced by 5 mg of escitalopram, that remitted completely after discontinuation of escitalopram and did not reappear later. Patient and their caretakers should be warned of symptoms of akathisia even when a very low dose of SSRI is prescribed.

Keywords: Akathisia, escitalopram, selective serotonin reuptake inhibitors, suicide-attempt

Escitalopram is a SSRI and acts by inhibiting the reuptake of serotonin in the nerve terminals. A search of the literature for escitalopram-induced akathisia was conducted using the PubMed, Google Scholar, Web of Science, Ovid, Science Direct, Wiley online library, Cambridge, Sage, MD Consult, and Springer-link. The term akathisia is of Greek derivation and translated literally into English, means “not to sit.” Akathisia is a subjective feeling of restlessness, objective signs of restlessness, or both.[1] Akathisia is a distressing adverse reaction, is usually underdiagnosed or misdiagnosed in patients who are treated with selective serotonin reuptake inhibitors (SSRIs), including fluoxetine, paroxetine, sertraline, fluvoxamine, and citalopram.[2]
electronic database. In the literature, there are only three case reports of escitalopram-induced akathisia. Here, we report a case of escitalopram-induced severe akathisia leading to a suicide attempt that remitted completely after discontinuation of escitalopram.

CASE REPORT

Mr. VKR, 25-year-old male consulted psychiatry OPD with complaints of cannabis intake for 3 years, elevated mood, physical restlessness, increased talkativeness, tall claims, overfamiliarity, and decreased need of sleep for 2 months. The patient was diagnosed as per the ICD-10-DCR with mental and behavioral disorders due to the use of cannabinoids, psychotic disorder predominantly manic symptoms. The patient was admitted and he was started on carbamazepine 600 mg, haloperidol 10 mg and trihexyphenidyl 2 mg, with which his symptom improved and on the above medication, patient was discharged. Subsequently, after 2 months’ follow-up, he presented with symptoms of low mood, lethargy, decreased interest in work, reduced self-esteem, and reduced attention and concentration. The patient was started on escitalopram 5 mg and his dose of haloperidol was reduced to 7.5 mg and he continued carbamazepine and trihexyphenidyl as mentioned above. Within 2 days of starting of escitalopram, the patient became restless and could not remain seated or standing and kept on walking or pacing. He was thrashing his hands all around and hitting his hands on the wall and several occasions, sustained injury. He was constantly swinging his legs, rocking from foot to foot while standing and was crossing and uncrossing the legs when sitting. The patient had severe distress due to these symptoms and was not able to sleep. He ran out of home and jumped into a pond near his house, but he was saved. He was kept tied with a rope by his family members as he was running around and was constantly expressing suicidal ideas. The next day patient was brought to psychiatry OPD. During the interview, he confessed that he had severe restlessness, which was intolerable, so he jumped into the pond to commit suicide. His score on the Barnes Akathisia Rating Scale (BARS) scale was 9 (severe akathisia). As per the Naranjo Adverse Drug Reaction Scale, the probability of association of this adverse reaction with escitalopram was 7 (i.e., probable). Results of routine blood investigation and computed tomography scan of the brain revealed no abnormality. He had no remarkable neurological findings. After other possible disorders were excluded, the diagnosis of severe akathisia, possibly induced by escitalopram, was made. Subsequently, escitalopram was stopped, and other medications were continued. His symptoms continued despite prompt discontinuation of the drug. Akathisia was treated with propranolol 20 mg once daily and clonazepam 0.5 mg thrice daily and was tapered over 10 days. Akathisia quickly resolved and didnot reappear later.

DISCUSSION

In this case, the temporal relationship suggests escitalopram as the causative molecule, but it is difficult to pinpoint the offending medication. As the patient was maintaining well on haloperidol, carbamazepine and trihexyphenidyl for 2 months, and only within 2 days of initiation of escitalopram the Akathisia appeared. The interaction and combinational effect may be attributed, as haloperidol and carbamazepine are well-known molecules responsible for inducing akathisia. In this case, Akathisia was very severe (score 9 on BARS), which led to the suicide attempt, but we did not find corresponding tremors, muscular rigidity or any other signs of extrapyramidal side effects, which may be expected with neuroleptic-induced Akathisia. Similarly, we did not find sedation, blurring or double vision or ataxia to suggest carbamazepine overdose. However, the symptoms resolved quickly on stopping escitalopram and rechallenge with escitalopram was not done for ethical reasons. There was no need to stop haloperidol and carbamazepine, and they were continued uneventfully, but in a future course, any need of SSRIs or antipsychotics will need precautions.

Akathisia does not appear as a dose-dependent side effect, as the earlier report of escitalopram-induced Akathisia was with 10 mg of escitalopram. However, in our case low dose (5 mg) of escitalopram caused severe Akathisia, implicating individual susceptibility plays an important role.

Our patient had severe Akathisia leading to suicidal behavior, which indicates its seriousness. Literature
suggests that Akathisia should be considered as an independent risk factor for self-harm and suicidality. This case report highlights the risk of escitalopram-induced severe akathisia, even at a low dose, but along with other neuroleptics or antiepileptics, with a risk of a suicide attempt. The recognition of such adverse effect requires a high index of suspicion, early recognition, and prompt management. Patient and their caretakers should be warned of symptoms of Akathisia even when a very low dose of SSRI is prescribed.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understand that name will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Conflicts of interest
There are no conflicts of interest.

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