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Case Report Open Access

Escitalopram Intoxication Induced Acute Psychosis

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Abstract

Objective: Side effects of escitalopram overdose including serotonin syndrome were previously found. We report a case with escitalopram-related psychosis.

Case report: A 33 year-old woman had no previous history of psychotic symptoms experienced a psychotic episode with with quik recovery following taking 400 mg of escitalopram.

Conclusion: This case report raises the issue that the escitalopram intoxicated effect and pharmacological sensitivity of serotonin syndrome exist individual variations. The systematic analysis and follow-up regarding escitalopram-associated serotonin syndrome and the incidence rate of severe psychotic symptoms will be further investigated.

Keywords: Escitalopram overdose; Psychosis; Serotonin syndrome

Introduction

Selective serotonin reuptake inhibitors (SSRIs) were first introduced in the late 1980s for the treatment of major depressive disorder. Escitalopram is a pharmacologically active S-enantiomer of citalopram, and it has been reported to be a more potent inhibitor of serotonin reuptake than citalopram, which is a racemic mixture of R- and S-citalopram [1,2]. Escitalopram has gained an increasing share of the antidepressant market. The major side effects of escitalopram overdose including serotonin toxicity, serotonin syndrome, seizure, and QTc prolongation were previously found [2]. However, reports regarding escitalopram-related psychosis are lacking. Herein, we report a 33-year-old woman with a prominent psychotic episode and serotonin toxicity after an large dose of escitalopram overdose.

Case Report

This 33-year-old woman was diagnosed with major depressive disorder and was regularly followed up at our hospital. She was referred to our department by her family who reported the sudden onset of vertigo, uncontrollable muscle shaking, agitation, perceptual misinterpretation and visual hallucination for 10 hours. She claimed to have attempted suicide by swallowing 40 pills (400 mg) escitalopram about 2-3 hours before the symptoms developed. She was then immediately sent to the emergency department.

On admission to the emergency department her body temperature was 37.2°C, blood pressure 172/99 mmHg, pulse rate 102 beats/minute, respiratory rate 20 breaths/minute with oxygen saturation of 99%. She had a Glasgow Coma Scale score of 15. During the evaluation and physical examination, she was noted to be diaphoretic and tremulous, however normal deep tendon reflexes and muscle power were found. A mental status examination revealed that she was disoriented, confused, anxious an suspicious, irritable and restless. She also had incoherent and irrelevant speech, poor attention, and visual hallucinations of seeing many people walking. A complete blood count, other metabolic panels including glucose level, liver and renal function, electrolytes and creatinine phosphokinase test were done and revealed an elevated white blood cell count of 12330/mm^3, and mildly elevated alaninaminotransferase level of 58 IU/L. Electrocardiography, chest X-ray and computerized tomography of her brain showed no particular findings. A match of pill count by the empty packets provided and the clinical history of dosage taken was also found. By intravenous hydration with normal saline was maintained for 2 hours, and intramuscular haloperidol 5 mg was also given for her acute psychotic symptoms and agitation. Then her physical condition began to normalize and the hallucinatory symptoms were subsided. She was then discharged under a stable condition after 12 hours since ER visit.

When followed up at the outpatient department 6 days later, she was afebrile with normal vital signs and no myoclonus or muscle rigidity. She was well oriented and alert, although perplexed and amnesic about the previous event. She also denied any illicit substance use. A mental status examination showed a normal appearance and a cooperative attitude with eye contact, and coherent and relevant speech. She was mildly anxious and depressed and she still had negative thoughts, but she denied any suicidal or homicidal ideation. She did not experience any delusions or hallucinations during the interview and counldnt recall any psychotic symptoms before this incidence, and her insight and judgment were acceptable.

Discussion

Serotonin syndrome often manifests as a clinical triad of mental status changes, autonomic hyperactivity, and neuromuscular abnormalities [3-5]. Mental status and behavioral changes include confusion, hypomania, anxiety, delirium, dysphoria, irritability, agitation, and restlessness [6]. Neither the serotonergic hyperactivity nor the psychotic symptoms had occurred in our patient before and were temporally related with the drug overdose. A dramatic improvement after removing the blood serotonin accumulation via

intravenous hydration was also noted, thereby suggesting that escitalopram was the most likely cause of the patient's symptoms.

Previous studies have reported that 10% to 14% of patients who overdose with SSRIs experience serotonin toxicity many of whom with only mild presentations [7,8]. In a study of 469 isolated cases of SSRI ingestion, sertraline, paroxetine, and fluvoxamine were the most likely causes [8]. Although escitalopram has been reported to show less serotonin toxicity compared with other SSRIs, its effect was best described by one previous study in which 15% of 46 patients exposed to escitalopram alone exhibited significant serotonin toxicity, while isolated serotonergic neuromuscular findings developed in as many as 46% of the patients at a mean ingested dose of 140 mg [9]. Another study reported that the most common clinical effects after an overdose with escitalopram with a mean dose of 130 mg were tachycardia (19.5%), drowsiness (15.0%) and hypertension (9.0%) [10]. Seizure was noted in one patient who took 300 mg of escitalopram [10]. All of these effects were related to serotonin toxicity, and the overall severity of the adverse effects was reported to be dose-related, although no specific outcomes or prognostic data were revealed [10]. Other case reports have also described severe serotonin syndrome after taking escitalopram at doses ranging from 10 mg to 100-200 mg a day [1,11-14], sometimes with concomitant drug-drug interactions [15].

Our case first presented with symptoms mainly of central serotonin activity and particularly psychosis, which has been commonly reported with other serotonergic drugs and SSRIs, but not escitalopram [16-18]. In addition, the patient had ingested a relatively large dose compared with other case reports. The symptoms of intoxication were recovered quickly without notable sequelae in the following period. Based on our expereince, education on the neuropsychiatric manifestations in toxic dose to patients and the family would be a necessary issue after prescribing the mediation.

It will be an interesting question to clarify the dose effect on the development of serotonin syndrome in the therapeutic course or accidental hazardous overdose.

Conclusion

This case report raises the issue that the escitalopram intoxicating effect and the pharmacological sensitivity of serotonin syndrome existed individual variations. The systemic analysis and follow-up regarding escitalopram-associated serotonin syndrome and the incidence rate of severe psychotic symptoms will be further investigated.

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