

CASE REPORT

Olanzapine Overdose in a Patient with Pinpoint Pupil with Altered Sensorium

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Abstract

Background: Olanzapine is a highly tolerable and easily affordable atypical antipsychotic drug which has been commonly prescribed in both inpatient and outpatient settings for several mental disorders. Olanzapine overdose is commonly seen in psychiatric patients, who attempt suicide by intoxicating themselves with their own prescribed medications. Increased olanzapine use is associated with increased incidence of overdosing.

Case Presentation: We are reporting a case of olanzapine overdosage as a cause of pinpoint pupils and altered sensorium with exclusion of other differentials. The mainstay of management of olanzapine overdose is general supportive and symptomatic measures. *Discussion:* Pinpoint pupils with altered sensorium and agitation are always an alarming situation for a clinician, because of differentials like organophosphorus poisoning, pontine hemorrhage and opium overdosing. Due to olanzapine overdosage, similar clinical picture can be confusing in the emergency department and early identification of such cases is helpful to decrease the risk of fatality.

Conclusion: This case highlights the significance of olanzapine overdosing as a differential diagnosis for patients presented with altered sensorium and pinpoint pupils in the emergency department. Olanzapine overdosage is associated with high morbidity and mortality. Although there is no specific antidote for olanzapine overdose, appropriate history, assessment and early diagnosis are very useful for the better outcome.

Keywords: Agitation; Antipsychotics; Pontine Hemorrhage

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INTRODUCTION

Olanzapine is an atypical antipsychotic widely prescribed for several mental disorders like schizophrenia and bipolar disorder (1, 2). Incidences of antipsychotic drug overdosing are very common because psychotic patients often have a high suicidal tendency (3).

Olanzapine poisoning or overdosing is associated with clinical features like agitation, tachycardia, delirium, drowsiness and pinpoint pupils. It is characterized by alterations in laboratory parameters such as hyperglycemia, dyselectrolytemia, leukocytosis and conduction abnormalities on ECG (4).

CASE PRESENTATION

An 18-year-old unmarried female was brought to our outpatient department in altered sensorium with a Glasgow Coma Scale of 6 (E1, V1, M4). As per history provided by the mother, she was initially irritable and then developed rapidly progressive drowsiness within the last 12 hours. There was no history of any ongoing treatment, any physical

or mental trauma and underlying disease like bipolar or mood disorder. There was no history of any substance abuse like opium. During the examination, she was afebrile with altered sensorium, responding only to painful stimuli. She had no neck stiffness. Her pupils were pinpoint but reactive to light. Bilateral extensor plantar response, increased tone in all four limbs and brisk reflexes were observed. There was no lacrimation, salivation or any peculiar odour to suggest organophosphorus poisoning. She also developed hypotension (90/50 mm Hg) and tachycardia (110/min). Complete hemogram, ABG analysis, electrolytes, liver function and renal function tests were normal. To rule out pontine hemorrhage, urgent non-contrast computed tomography (NCCT) head and CSF examination were performed which also came out to be normal. After gastric lavage, she was treated with intravenous fluids and supportive therapy. In the initial six hours, she started showing signs of gradual improvement. She completely recovered after 24 hours and became fully conscious and oriented. On further evaluation, the patient gave a history of some unknown tablets ingestion which actually had been prescribed for her sister.

*Correspondence to: Gopal Krishna Bohra; MD. Assistant Professor, Department of General Medicine, All India Institute of Medical Sciences Jodhpur, Jodhpur, India Phone: +91 958 77 77 231, E-mail: gopalbohra17@gmail.com Received 9 July 2017, Accepted 27 August 2017 The history was confirmed by family members and one empty blister packet of Olanzapine 10mg was obtained from her room.

After 4 days of treatment, she was discharged from the hospital. The patient is under regular follow-up and is doing reasonably well in her social life.

DISCUSSION

Olanzapine overdose is commonly seen in psychiatric patients, who attempt suicide by intoxicating themselves with their own prescribed medications. The clinical presentation of Olanzapine overdose is found variably dependent upon the ingested dose and other factors. Most common neurological symptoms of olanzapine overdosing are somnolence (77%) and agitation (42%) (5). Miosis is seen in about 30-35% of cases of all olanzapine overdoses and 82% of moderately severe overdoses (5).

Olanzapine associated dopamine-resistant hypotension is also a documented feature which responds well to norepinephrine (6, 7). QT prolongation and consequent fatal arrhythmias are the important consideration especially in patients with underlying cardiovascular disease.

Severe overdosing of olanzapine is often associated with rapid deterioration in consciousness level, miosis, upgoing plantars, increased tone and lacrimation mimicking organophosphate poisoning, opiate intoxication and pontine hemorrhage. There is no specific antidote to olanzapine and treatment is largely supportive. Early gastric lavage and administration of activated charcoal may decrease its complications (8).

CONCLUSION

This case highlights the significance of olanzapine overdosing as a differential diagnosis for patients presented with altered sensorium and pinpoint pupils in the emergency department. In most of the developing countries, toxicological screening and drug level estimation facilities are not readily available. Hence, gathering appropriate history and finding evidence of drug overdosing are very useful for better management.

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