Case Report

Baclofen Overdose Presenting as Psychosis with Catatonia

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ABSTRACT

Baclofen is a centrally acting gamma-aminobutyric acid receptor agonist with many therapeutic uses in neurology and psychiatry. An overdose of baclofen is known to cause serious side effects such as encephalopathy, seizures, respiratory depression, and delirium. Association of baclofen with psychosis has also been reported. In this case report, we are highlighting the manifestation of catatonic features in addition to psychosis following baclofen overdose.

Key words: *Baclofen, catatonia, gamma-aminobutyric acid-B receptor, psychosis*

INTRODUCTION

Baclofen (4-amino-3-[-4-chlorophenyl]-butanoic acid), a centrally acting gamma-aminobutyric acid (GABA) B receptor agonist, has many therapeutic uses in the field of neuropsychiatry. Therapeutic indications include spasticity resulting from conditions such as cerebral palsy, traumatic spinal injury, stroke, and multiple sclerosis.[1,2] It is also used as an anticraving agent in alcohol-dependence syndrome.[3] Overdose of baclofen is associated with serious adverse effects such as encephalopathy, respiratory depression, muscular hypotonia, and generalized hyporeflexia. Studies have reported that overdose >200 mg of baclofen has resulted in coma, delirium, and seizures.[4]

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The psychiatric side effects of baclofen drug have not been commonly reported as compared to neurological side effects. This article describes the development of acute-onset psychosis with catatonic symptoms following an overdose of baclofen.

CASE REPORT

A 30-year-old married female with no significant family and past history of psychiatric illness presented to us with an acute-onset behavioral change. On clarification, it was found that, following an altercation with her family members, she had attempted self-harm by consuming ~300 mg of baclofen. The next day morning, she was found unconscious with frothing near

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mouth, following which the patient was rushed to the emergency department of a medical college, where she was given stomach wash and shifted to the Intensive Care Unit. She was on nasogastric tube feeding for the next 3 days during which the patient was conscious but not responding to any questions. From the 4th day, she was also found to be talking to self, irritable, and hostile toward family members and was found to have auditory hallucinations in the form of commenting and commanding type of voices and decreased sleep. She started having grandiose ideas that she is going to become a Goddess along with a delusion of persecution against her family members along with other patients in the ward. She was also found to be excessively cheerful and disinhibited at times. She was treated with antipsychotic and parenteral sedation (details of medication not available) for the above symptoms. Subsequently, she was referred to our hospital and on evaluation was found to be irritable and agitated with similar findings as described above. A day later, the patient was observed to have catatonic symptoms in the form of posturing, staring, negativism, reduced food intake, ambitendency, and mutism. Central nervous system examination revealed no focal neurological deficits. The catatonia severity was assessed using the Bush–Francis Catatonia Rating Scale score[5] and the score was 20. Low-dose lorazepam was started (3 mg/day in divided doses per orally) following which the patient showed a dramatic improvement and had no catatonic symptoms thereafter (Bush–Francis Catatonia Rating Scale score = 0). Tablet olanzapine 5–10 mg/day was initiated for psychotic and affective symptoms. She was discharged on tablet olanzapine 10 mg/day and tapering doses of lorazepam. Routine blood investigations and thyroid profile, erythrocyte sedimentation rate, serum Vitamin B12, and folate were within the normal limits. Magnetic resonance imaging of the brain (plain with contrast) was done, and no significant abnormality was detected. There was no history of consumption of any psychoactive substances, any herbal medications, and other over-the-counter medications. There was no history of any medical illness. The patient was evaluated during the follow-up after a month and was found to be maintaining well on 10 mg of tablet olanzapine in the absence of any psychotic or catatonic symptoms.

DISCUSSION

The abrupt onset of psychotic and catatonic symptoms on baclofen overdose and their rapid resolution within a week on treatment in the absence of any other contributing factor suggested a diagnosis of baclofen-induced psychosis. The scoring on the Naranjo probability scale[6] suggested a probable association of baclofen with acute psychiatric symptoms in the patient. A review describing psychiatric presentation following abrupt withdrawal of baclofen highlights the symptoms mostly characterized by delirium and associated perceptual disturbances.[7] Our patient too was in an altered sensorium for the initial 4 days following an overdose but was in clear consciousness following the onset of psychotic and affective disturbance. An earlier case report has discussed about the emergence of psychosis following the therapeutic dose of baclofen and was characterized predominantly by auditory hallucinations and persecutory and referential delusions.[8] In an another case report, a patient with traumatic brain injury developed psychosis following intrathecal administration of baclofen.[9] In both the above-discussed cases, psychosis subsided once baclofen was stopped.

Case reports of mania induced by therapeutic doses of baclofen have been described in the literature, and these symptoms can also arise in patients without any history of bipolar disorder.[10]

The mechanism by which baclofen can induce psychotic symptoms is not clear. Modulation of firing of dopamine neurons in the ventral tegmental area by GABA-B receptor stimulation could be contribute to the development of psychotic symptoms. In addition, its role in inducing mania-like symptoms could be due to increased noradrenergic turnover by altering the firing rate of GABAergic neurons and indirect disinhibition of 5-hydroxytryptamine (5-HT) neuron activity by presynaptic GABA-B receptors on non-5-HT neurons in the dorsal raphe nucleus.[10] The relationship between GABA-B agonist baclofen and catatonia has been described previously in a patient who was prescribed the medication for persistent lower-extremity muscle spasm following surgery for spinal cord ependymoma. The patient developed catatonic features after increase in the dose of baclofen, and there was remission of symptoms after the drug was tapered and stopped. Moreover, the symptoms re-emerged after the patient was restarted on baclofen.[11] Increased GABA-B receptor activity and reduced activity at GABA-A receptors have been proposed to cause catatonia. The balance between the GABA-A and GABA-B receptors can be disturbed by baclofen, leading to catatonic signs and symptoms.[12]

CONCLUSION

Baclofen is a commonly used drug in various neurological illnesses to reduce spasticity, and also as an anticraving agent in substance-use patients. Baclofen even though relatively safe at therapeutic doses can sometimes lead to life-threatening complications in overdose. This case demonstrates the possibility of baclofen overdose with psychosis with catatonic symptoms.

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Conflicts of interest
There are no conflicts of interest.

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