Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Psychiatry

Psychosis and Catatonia in a Patient after Taking Baclofen: A Case Report

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DOI: https://doi.org/10.36347/sjmcr.2025.v13i09.034 | **Received:** 02.07.2025 | **Accepted:** 07.09.2025 | **Published:** 18.09.2025

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Abstract **Case Report**

Baclofen is a centrally acting GABA-B receptor agonist commonly used to manage spasticity from conditions such as cerebral palsy, spinal cord injury, and multiple sclerosis. It is also employed off-label for alcohol dependence and other conditions like persistent hiccups. Baclofen's mechanism of action involves reducing neuronal excitability by stimulating inhibitory signals and reducing excitatory neurotransmitter release. Although rare, overdose or dose escalation can lead to severe neurological effects, including delirium, seizures, and psychosis. We report a case of a 45-year-old man who developed catatonia and psychosis after high-dose baclofen use for dorsal pain.

Keywords: Psychosis, Catatonia, Baclofen, Induced psychosis, Dorsal pain.

INTRODUCTION

Baclofen (4-amino-3-[4-chlorophenyl]-butanoic acid), a centrally acting agonist of gamma-aminobutyric acid (GABA) B receptors, has numerous therapeutic properties.

GABA B has many therapeutic applications in the field of neuropsychiatry. The therapeutic indications of baclofen include spasticity resulting from cerebral palsy, traumatic spinal cord injury, strokes, and multiple sclerosis [1,2]. It is also used as an anti-craving agent in alcohol dependence syndrome [3]. Additionally, this muscle relaxant has several off-label uses, including persistent and chronic hiccups. The available dosage forms of baclofen include oral, transdermal, and intrathecal administration via an infusion pump. Serious adverse effects associated with this drug are rare at commonly prescribed doses. However, baclofen overdose has been associated with severe neurological effects such as delirium, seizures, and coma [4].

In this article, we report a case of baclofen-induced catatonia and psychosis in a young adult suffering from dorsal pain, who received baclofen orally.

CLINICAL CASE REPORT

The patient is a 45-year-old man, married and father of three children, working as a clothing merchant, with a history of dorsal pain for which baclofen was prescribed and taken for one week. He was referred for psychiatric consultation after a 27-day hospitalization in the neurology department. The initial presentation was marked by mutism, visual blurriness, abnormal posture, and atypical facial expressions. He was conscious with a GCS of 15 but non-verbal, unresponsive to stimuli, showing resistance and opposition. Vital signs and neurological examination were unremarkable, except for catatonia (mutism, negativism, mannerisms, and posturing).

Complete radiological assessments (CT scan, EEG, and MRI) as well as a full biological workup were performed, all of which returned normal results.

After 27 days of monitoring and with no improvement in the clinical picture, a psychiatric origin was suspected, and the patient was referred to the psychiatric emergency services.

The initial psychiatric interview with the patient, accompanied by his wife, was marked by mutism, a fixed gaze, negativism with facial grimaces. On physical examination, the patient also exhibited mild resistance during induced positioning. The patient also displayed soliloquy with inappropriate laughter. An evaluation using the Bush-Francis Catatonia Rating Scale revealed a score of 28.

The wife reports that the patient has no personal or family psychiatric history and no known use of psychoactive substances. Aside from the dorsal pain for which he started taking baclofen 3 times a day one week prior to the onset of the current symptoms, she also noted that she did not find any remaining medication at home.

The patient was started on lorazepam 3 mg per day and amisulpride 200 mg per day. After the first week, there was a complete disappearance of catatonic symptoms. However, the patient began to verbalize a persecution delusion centered on his surroundings, stating that everyone meant him harm and that he was hearing evil voices threatening him. The patient was maintained on amisulpride with a progressive reduction in the lorazepam dosage, and by the third week, the patient reported a complete disappearance of all psychotic symptoms with 200 mg/day of amisulpride.

After his clinical stabilization, the patient reported that one week after starting baclofen treatment and following intense pain from his back, he should have taken double or triple the usual dose prescribed by his doctor.

DISCUSSION

Catatonia is a severe neuropsychiatric syndrome involving emotions, speech, movement, and complex behavior. Its incidence is about 10 per 100,000 person-years [5]. Although it was considered a form of schizophrenia for much of the 20th century, the major diagnostic manuals, the International Classification of Diseases (ICD-11) and the Diagnostic and Statistical Manual of Mental Disorders (DSM-5-TR), now recognize that catatonia occurs in a wide range of psychiatric, neurological, and medical disorders.

The physical and mental evaluation of our patient revealed signs such as mutism, negativism, waxy flexibility, and grimacing facial expressions, allowing us to diagnose catatonia. Catatonia was assessed using the Bush-Francis Catatonia Rating Scale (CRS), with a score of 28. The significant improvement in motor symptoms after the administration of a benzodiazepine further supports the diagnosis of catatonia [6].

Baclofen is a lipophilic analogue of gamma-aminobutyric acid (GABA). Its mechanism of action is not yet fully understood. It is assumed that this drug acts as an agonist of the beta subunit of GABA on monosynaptic and polysynaptic neurons in the spinal cord and brain. In this way, baclofen likely reduces spasticity by stimulating inhibitory neuronal signals at the postsynaptic neurons and also by reducing the release of excitatory neurotransmitters at the presynaptic neurons [7].

The sudden onset of psychotic and catatonic symptoms in our patient following a probable baclofen overdose, as well as their rapid resolution within a few weeks after treatment, without any other contributing factors, suggested a diagnosis of baclofen-induced psychosis.

Baclofen-induced catatonia is rarely reported in the literature. Some studies [8,4-9] have investigated the relationship between catatonia and baclofen. Most of these studies were conducted in patients with a history of psychiatric disorders.

A recent study [10] reported the case of a young girl without psychiatric history who developed catatonia after ingesting 200 mg of baclofen in a suicide attempt. She showed significant improvement after the administration of low-dose lorazepam (3 mg/day in divided oral doses). This is similar to our case, in which the patient had no prior psychiatric or catatonia history but developed catatonia with major clinical symptoms after increasing baclofen doses.

The study by Nahar et al. [8] was conducted in adult patients who attempted suicide by ingesting baclofen in doses ranging from 200 to 300 mg. All these patients developed catatonic symptoms, including postural disturbances, mutism, negativism, and stereotyped movements. They were all treated with lorazepam 3 mg/day, leading to symptom resolution.

Patients with baclofen-induced catatonia generally present major clinical features of catatonia, such as mutism, stupor, and rigidity. Pauker and Brown highlighted the role of multiple factors influencing catatonic symptoms [11]. They suggested that a possible interaction between various medications could contribute to the development of catatonia. They previously described a patient who was prescribed baclofen for persistent lower limb muscle spasms following spinal cord ependymoma surgery. The patient developed catatonic features after an increase in baclofen dosage, with symptom remission occurring after dose reduction and eventual discontinuation of the drug. Furthermore, symptoms reappeared when the patient resumed baclofen [11].

Case reports of mania induced by therapeutic doses of baclofen have been described in the literature, and these symptoms can also occur in patients without a history of bipolar disorder [12].

Psychosis with catatonic features has also been reported with other muscle relaxants. In 1983, Beeber and Manring likely described the first case of catatonia secondary to a muscle relaxant [13]. They reported the case of a 38-year-old woman who had received cyclobenzaprine for back pain. She developed manic psychosis along with some catatonic symptoms (posturing and echolalia). It is worth noting that the patient had a significant psychiatric history, including manic episodes.

A more recent hypothesis explaining catatonia involves an imbalance between GABA receptors. In rat models, it has been observed that GABA-B receptor hyperactivity and GABA-A receptor hypoactivity can induce catatonic symptoms. In this context, baclofen may lead to an increase in selective GABA-B receptor activity in predisposed individuals [7]. This imbalance between GABA-A and GABA-B receptors could be disrupted by baclofen, resulting in catatonic signs and symptoms [14].

CONCLUSION

Baclofen is a commonly used drug in neurology to reduce spasticity and also as an anti-craving agent in patients with substance use disorders. Baclofen-induced catatonia has been documented in the literature. Most affected patients had a history of psychotic symptoms and catatonia, but patients without a history of catatonia or psychiatric disorders can also develop baclofen-induced catatonia or psychosis. It is essential for clinicians, especially neurologists, to be aware of this potential side effect in order to quickly establish a diagnosis and ensure prompt and appropriate management.

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