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A Rare Case of Intensive Care Admission with Baclofen Toxicity

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Abstract

This case describes a 58-year-old female who presented to the emergency department with profound altered mental status following the ingestion of approximately 400 mg of baclofen. Because baclofen is not detected on standard urine drug screens and serum levels are not routinely available, the diagnosis remained elusive until the patient recovered enough to report the ingestion. The patient required intensive care unit admission for supportive care, as she remained somnolent for 48 hours. This report emphasizes that baclofen toxicity should be considered in cases of unexplained altered mental status and warns that severe toxicity can mimic brain death, necessitating caution before making irreversible clinical prognostications.

Keywords

Baclofen, GABA-B, Overdose

1. Introduction

Baclofen is a skeletal muscle relaxant and structural analog of gamma-aminobutyric acid (GABA) that acts as a GABA-B receptor agonist, resulting in inhibition of excitatory neurotransmission at the spinal level [1]. It is indicated for muscle spasticity caused by conditions such as spinal cord lesions and multiple sclerosis. There are several off-label indications as well, including trigeminal neuralgia, gastroesophageal reflux disease, alcohol use disorder, and intractable hiccups [1] [2].

Due to its limited therapeutic window and rising use in outpatient settings, careful monitoring is necessary because even a small dosing error could result in toxicity or subtherapeutic levels. Baclofen has high bioavailability and is rapidly absorbed by the gastrointestinal tract, with peak plasma concentrations achieved within approximately 1 - 2 hours of oral administration [1]. Although it crosses the blood-brain barrier to exert its central effects, its penetration is limited, result-

ing in CSF concentrations approximately 8.5 times lower than plasma concentrations [3]. It needs to be taken frequently to maintain a significant effect because of its short half-life, which is approximately 3.5 - 5.6 hours in patients with normal renal function [1]. Approximately 10% - 15% of the drug undergoes hepatic metabolism, while the remaining 85% - 90% is excreted unchanged by the kidneys [1]. Baclofen has a low molecular weight (213 Da), approximately 30% - 35% protein binding, and an apparent volume of distribution of approximately 0.7 - 0.8 L/kg in adults [1] [3].

Several organ systems may experience adverse effects, including the cardiovascular, gastrointestinal, genitourinary, neuromuscular, cutaneous, and central nervous systems. Simultaneous opioid use is linked to increased toxicity, particularly over an extended period of time [4]. This medication carries an FDA boxed warning specifically for intrathecal administration regarding the risk of abrupt discontinuation, which can cause severe rebound spasticity, rhabdomyolysis, multi-organ failure, hypermetabolism, and impaired mental status [1]. Baclofen should be used with caution in patients who have had a stroke or Parkinson's disease [1]. In oral overdose, toxicity can cause symptoms ranging from drowsiness and vomiting to respiratory failure, seizures, and profound coma that may mimic brain death [1] [3] [5].

2. Case Presentation

A 58-year-old female was brought to the emergency department by emergency medical services with altered mental status. The patient was found at home by her husband in a somnolent state, difficult to arouse, but maintaining her airway. Her past medical history was notable for chronic lower back pain, for which she had recently been prescribed baclofen 10 mg three times daily by her primary care physician. She was not taking any other scheduled medications. The time of ingestion was unknown at presentation.

On initial examination, the patient was profoundly somnolent but responsive to sternal rub with nonpurposeful withdrawal of extremities. Vital signs were notable for blood pressure 104/62 mmHg, heart rate 72 beats per minute, respiratory rate 14 breaths per minute, temperature 36.8˚C (98.2˚F), and oxygen saturation 98% on room air. Pupils were 3 mm, equal, and bilaterally reactive to light. Corneal reflexes were intact bilaterally. The patient had an intact gag reflex and cough with suctioning, confirming preserved airway-protective reflexes. Deep tendon reflexes were diffusely diminished (1 + throughout). There was no clonus. Motor examination demonstrated withdrawal to painful stimuli in all four extremities without localizing responses. Plantar responses were flexor bilaterally. No spontaneous myoclonus or seizure activity was observed.

Given the unclear altered mental status, a comprehensive workup was initiated to evaluate for metabolic, toxic, infectious, and structural etiologies. Venous blood gas showed a pH of 7.38 with a pCO_2 of 44 mmHg. Blood counts, electrolytes, and renal function were within normal limits. Ammonia, thyroid-stimulating hormone, serum acetaminophen, and salicylate levels were normal. Urine drug screen

was negative. Non-contrast computed tomography (CT) of the head showed no acute intracranial abnormality, no hemorrhage, mass effect, or midline shift. Naloxone 0.4 mg was administered intravenously with no improvement in mental status, and a repeat dose of 2 mg IV was given without response, making opioid toxicity unlikely.

Poison Control was contacted at the time of ED evaluation. Given the negative standard toxicology workup and unknown ingestion history, Poison Control advised ICU admission for supportive care with close neurologic and respiratory monitoring.

Given the patient's ability to maintain her airway, preserved airway-protective reflexes, and hemodynamic stability, she was admitted to the intensive care unit for close monitoring and supportive care. No specific antidote or intervention was administered. The patient remained profoundly somnolent for the first 48 hours, arousable only to deep painful stimuli but protecting her airway. Serial neurologic assessments demonstrated persistent diffuse hyporeflexia and non-purposeful motor responses during this period, with intact pupillary and corneal reflexes throughout. Vital signs remained stable throughout her ICU stay without the need for mechanical ventilation or vasopressor support.

On hospital day 3 (approximately 60 - 72 hours after estimated ingestion), the patient began to show gradual improvement in her level of consciousness. By the afternoon of day 3, she was able to follow simple commands and answer yes/no questions appropriately. Deep tendon reflexes normalized. Her mental status continued to improve over the subsequent 24 hours. Once able to provide a history, she reported that she had been experiencing severe lower back pain for several weeks and had been prescribed baclofen 10 mg three times daily by her primary care physician. On the day before admission, frustrated by inadequate pain relief, she ingested approximately 20 tablets of baclofen 20 mg (estimated total ingestion of 400 mg). She reported that she had obtained the 20 mg tablets from a family member who had a separate prescription for baclofen 20 mg for a different indication; these were not her own prescribed tablets. She denied suicidal intent and stated she was unaware of the potential toxicity of this dose. Psychiatry was consulted for risk assessment prior to discharge.

A brief clinical timeline is summarized below:

1) Day 0 (estimated evening): Ingestion of approximately 400 mg baclofen (20 tablets of 20 mg).

2) Day 1 (morning): Found unresponsive by husband; EMS transport to ED; comprehensive workup negative; naloxone administered without response; Poison Control contacted; admitted to ICU.

3) Days 1 - 2: Profound somnolence with preserved airway-protective reflexes; supportive care in ICU.

4) Day 3: Gradual improvement in consciousness; able to follow commands by afternoon; patient reports ingestion history.

5) Day 4: Continued neurologic improvement; psychiatry consultation completed.

6) Day 5: Discharged home with outpatient follow-up.

3. Discussion

This case illustrates the diagnostic challenge and clinical course of oral baclofen overdose presenting as profound altered mental status requiring intensive care admission. Baclofen has a narrow therapeutic index, with the recommended maximum oral dose of 80 mg per day [1]. This patient ingested an estimated 400 mg, approximately five times the maximum therapeutic dose, resulting in severe central nervous system depression. A recent systematic review of oral baclofen toxicity found that CNS depression occurred in 68% of cases, seizures in 36%, and respiratory depression in 21%, with severe toxicity particularly common at doses ≥300 mg [6].

The clinical presentation of baclofen toxicity is dose-dependent and can range from mild drowsiness and confusion at lower doses to profound coma, respiratory failure, seizures, and cardiovascular instability at higher doses [3] [6]. In severe cases, baclofen overdose can produce a clinical picture that mimics brain death, including absent brainstem reflexes, fixed dilated pupils, and loss of all motor responses [3] [5] [7]. This phenomenon has been well documented in both adult and pediatric patients and is an important consideration in the evaluation of comatose patients with unknown ingestion history, as premature determination of brain death must be avoided until drug clearance is confirmed [5] [7] [8]. The most common EEG findings in severe baclofen toxicity are burst suppression and nonspecific diffuse slowing, with generalized triphasic waves reported in up to 71% of cases in one series [3]. Our patient demonstrated profound somnolence with diffuse hyporeflexia but preserved pupillary and corneal reflexes and intact airway-protective reflexes, consistent with a moderate-to-severe overdose that did not reach the threshold for the most catastrophic manifestations.

A critical teaching point from this case is that baclofen is not detected on standard urine drug screens, which typically test for amphetamines, barbiturates, benzodiazepines, cannabinoids, cocaine, opioids, and phencyclidine. Serum baclofen levels can be obtained but are not widely available in most hospital laboratories and are not routinely measured at the time of presentation [3] [6]. Furthermore, the apparent rate of baclofen elimination from nervous tissue is slower than from serum, which may explain prolonged neurologic manifestations despite normal or negligible serum levels [3]. This diagnostic gap means that baclofen toxicity must be considered clinically in any patient presenting with unexplained altered mental status, particularly when the standard toxicology workup is negative. The diagnosis is often made retrospectively once the patient recovers and is able to provide a history, as occurred in this case.

The differential diagnosis for this presentation included other sedative-hypnotic drug overdoses (benzodiazepines, barbiturates, gamma-hydroxybutyrate), opioid overdose, other CNS depressants, metabolic encephalopathy (hepatic, uremic, thyroid), structural intracranial pathology (stroke, hemorrhage, mass lesion),

and central nervous system infection. The systematic exclusion of these etiologies through laboratory testing, toxicology screening, and neuroimaging was appropriate. Notably, naloxone 0.4 mg and subsequently 2 mg was administered intravenously without improvement in mental status, which, combined with the absence of miosis and respiratory depression, helped exclude opioid toxicity [3]. The normal metabolic panel and neuroimaging excluded metabolic and structural causes.

The management of oral baclofen overdose is primarily supportive, as there is no specific antidote [1] [3] [6]. The cornerstone of treatment includes airway protection, hemodynamic monitoring, and supportive care in an intensive care setting. Activated charcoal may be considered if the patient presents within one hour of ingestion and the airway is protected, though this was not applicable in this case given the delayed presentation [1]. Patients with baclofen neurotoxicity exhibit a marked vulnerability to the depressant effects of benzodiazepines, and these agents should be avoided unless clearly indicated for seizure management [3].

The role of hemodialysis in baclofen overdose has been clarified by the EXTRIP (Extracorporeal Treatments in Poisoning) workgroup. Baclofen's low molecular weight (213 Da), low protein binding (~30% - 35%), and moderate volume of distribution (0.7 - 0.8 L/kg) make it theoretically amenable to removal by hemodialysis [1] [3]. However, the EXTRIP workgroup recommends against performing extracorporeal treatment in addition to standard care for acute baclofen poisoning in patients with normal renal function, as baclofen's high endogenous clearance and short half-life limit the added benefit of dialysis in this setting. [3]. In contrast, the EXTRIP workgroup suggests performing extracorporeal treatment for baclofen toxicity from therapeutic doses in patients with impaired kidney function, especially in the presence of coma requiring mechanical ventilation, as renal impairment significantly prolongs the elimination half-life (from ~3.5 hours to 15 hours or longer) [1] [3] [9]. Our patient had normal renal function and maintained hemodynamic stability, making supportive care the appropriate management strategy.

The clinical course, with gradual improvement beginning at approximately 48–72 hours, is consistent with the expected pharmacokinetics of baclofen in overdose. Although the normal elimination half-life is approximately 3.5 - 5.6 hours, in overdose settings, the apparent half-life may be prolonged due to saturation of elimination pathways, and clinical effects can persist for 48 - 96 hours depending on the dose ingested [1] [3] [6]. Importantly, CNS clearance is significantly delayed compared to other body compartments, which may explain the prolonged neurologic manifestations [1] [3]. Despite the severity of presentation, the prognosis for baclofen overdose is generally favorable: the systematic review by Iqbal *et al.* (2026) reported full clinical recovery in 97.7% of cases, with mortality in retrospective cohorts generally low at 0 - 4% [6].

The psychiatric dimension of this case warrants discussion. Although the patient denied suicidal intent and attributed the overdose to frustration with inade-

quate pain control, any intentional ingestion of a potentially lethal dose of medication requires formal psychiatric evaluation before discharge. Baclofen is increasingly implicated in intentional self-poisoning, particularly in populations prescribed baclofen for substance use disorders, where the risk of self-poisoning is elevated due to the frequent comorbidity of psychiatric disorders [2] [10].

This case highlights several important clinical learning points. First, baclofen toxicity should be included in the differential diagnosis of unexplained altered mental status, particularly when standard toxicology screening is negative. Second, the clinical presentation of baclofen overdose can mimic brain death, and clinicians must exercise caution before making irreversible prognostic determinations in the setting of possible drug intoxication [5] [7]. Third, supportive care remains the mainstay of treatment for oral baclofen overdose in patients with preserved renal function, with hemodialysis reserved for cases complicated by renal impairment, per EXTRIP recommendations [3]. Fourth, patient education regarding the narrow therapeutic window of baclofen and the dangers of dose self-escalation is essential at the time of prescribing. Finally, psychiatric evaluation should be performed in all cases of intentional overdose regardless of stated intent.

Patient Consent and Ethics

Written informed consent was obtained from the patient for publication of this case report.

AI-Assisted Tool Disclosure

AI was used to assist with the language polishing of this manuscript, improving its fluency and clarity of expression.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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