Case Report / Olgu Sunumu

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Recognizing and Managing Life-threatening Toxicity in Pediatric Baclofen Poisoning: A Case Report

Pediyatrik Baklofen Zehirlenmesinde Hayati Tehlike Arz Eden Toksisitenin Tanınması ve Yönetimi: Bir Olgu Sunumu

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Abstract

Baclofen is a GABA-β receptor agonist used to treat spasticity, and its rising recreational use has led to increased reports of intoxication. Overdose may cause central nervous system depression, coma, seizures, and respiratory depression. This case report describes a case of accidental baclofen intoxication in a 2-year-old boy with a history of neonatal encephalopathy. A 2-year-old boy presented to the emergency department with sudden-onset drowsiness one hour after his sister suspected accidental ingestion of baclofen syrup (10 mg/mL) prescribed to his bedridden mother. The child was found holding the bottle, which was missing approximately 25% of its contents, suggesting an estimated ingestion of 300 mg, nearly 66 times the maximal daily pediatric dose. Upon arrival at the hospital, three hours post-ingestion, the patient was unresponsive with a Glasgow Coma score of 6, requiring emergency intubation. He was also noted to be bradypneic, bradycardic, hypotensive, and hypothermic with constricted and unreactive pupils, generalized hypotonia, and areflexia. Laboratory tests were largely unremarkable except for hypokalemia. An electrocardiogram showed bradycardia with a prolonged QTc interval. The patient's treatment was primarily supportive. After 19 hours of ventilation, he showed significant improvement, regained consciousness, and was successfully weaned off ventilation. He was later discharged without neurological sequelae. This case highlights the importance of considering baclofen overdose in pediatric patients presenting with coma, hypotonia, bradypnea, bradycardia, and hypotension, even with normal kidney function. Diagnosing baclofen toxicity can be challenging as it is not routinely detected in urine toxicology screenings. The case emphasizes the need for caregiver education regarding medication safety and proper storage to prevent unintentional poisoning. Although treatment is

Öz

Baklofen, spastisite tedavisinde kullanılan bir GABA-β reseptör agonistidir ve eğlence amaçlı kullanımının artması, zehirlenme olgularının da artmasına neden olmuştur. Aşırı doz, merkezi sinir sistemi depresyonu, koma, nöbetler ve solunum depresyonuna neden olabilir. Bu olgu raporu, yenidoğan ensefalopatisi öyküsü olan 2 yaşındaki bir erkek çocukta kazara baklofen zehirlenmesi olgusunu anlatmaktadır. İki yaşındaki bir erkek çocuk, yatağa bağımlı annesine reçete edilen baklofen şurubu (10 mg/mL) kazara içtiğinden şüphelenildikten bir saat sonra ani uyuşukluk şikayetiyle acil servise getirildi. Çocuk, içeriğinin yaklaşık %25'i eksik olan şişeyi elinde tutarken bulundu. Bu da tahmini 300 mg'lik bir alım olduğunu ve bu miktarın pediyatrik maksimum günlük dozun yaklaşık 66 katı olduğunu gösteriyordu. Hastaneye vardığında, ilacı aldıktan üç saat sonra, hasta Glasgow Koma skoru 6 ile tepkisizdi ve acil entübasyon gerektiriyordu. Ayrıca bradipneik, bradikardik, hipotansif ve hipotermi ile birlikte daralmış ve tepkisiz göz bebekleri, genel hipotoni ve arefleksi olduğu da kaydedildi. Laboratuvar testleri, hipokalemi dışında büyük ölçüde normaldi. Elektrokardiyogramda QTc aralığı uzamış bradikardi saptandı. Hastanın tedavisi öncelikle destekleyiciydi. On dokuz saatlik ventilasyonun ardından, hasta önemli ölçüde iyileşme gösterdi, bilincini geri kazandı ve ventilatörden başarıyla çıkarıldı. Daha sonra nörolojik sekeli olmaksızın taburcu edildi. Bu olgu, böbrek fonksiyonu normal olsa bile koma, hipotoni, bradipne, bradikardi ve hipotansiyon ile başvuran pediyatrik hastalarda baklofen doz aşımının göz önünde bulundurulmasının önemini vurgulamaktadır. Baklofen toksisitesinin teşhisi, idrar toksikoloji taramalarında rutin olarak tespit edilmediği için zor olabilir. Bu olgu, kaza sonucu zehirlenmeleri önlemek için ilaç güvenliği ve uygun saklama koşulları konusunda bakım verenlerin eğitilmesinin

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mainly supportive, this case adds to the growing body of literature on pediatric baclofen toxicity and the potential for good outcomes with timely intervention.

Keywords: Drug overdose, central nervous system, GABA agonists, baclofen

önemini vurgulamaktadır. Tedavi esas olarak destekleyici olsa da, bu olgu pediyatrik baklofen toksisitesi ve zamanında müdahale ile iyi sonuçlar elde edilme olasılığı konusunda giderek artan literatüre katkıda bulunmaktadır.

Anahtar Kelimeler: İlaç aşırı dozu, merkezi sinir sistemi, GABA agonistleri, baklofen

Introduction

Baclofen is a GABA-β receptor agonist that reduces excitatory neurotransmitter release, causing muscle relaxation and central nervous system (CNS) depression. It is used to treat spasticity in multiple sclerosis, spinal cord injuries, cerebral palsy, and stroke; with additional off-label uses, for alcohol withdrawal, chronic hiccups, and cocaine dependence.[1,2] Rising recreational use has led to increased reports of intoxication.[1] Diagnosing baclofen toxicity in children is challenging due to its rarity, non-specific symptoms, absence from routine toxicology screens, and often unreliable history. These factors leading to potential delays or misdiagnosis. An baclofen overdose may cause coma, seizures, respiratory depression, bradycardia, hypotension, and rhabdomyolysis with severe cases leading to cerebral edema.[1] Treatment is mainly supportive, with hemodialysis, effective, in severe toxicity or renal impairment.[3] We report a case of baclofen toxicity due to accidental ingestion in a 2-year-old boy with normal kidney function presenting with reduced consciousness, respiratory depression, and autonomic dysfunction.

Case Report

A 2-year-old boy with a history of neonatal encephalopathy presented to the emergency department with sudden-onset drowsiness an hour prior to presentation. His eldest sister suspected accidental baclofen ingestion after discovering the medication, which had been prescribed to their bed-bound mother, in his possession. His mother had a postpartum amniotic embolism after delivering him, and had been bedridden ever since. The baclofen syrup (10 mg/mL) was in a 100 mL bottle, which had initially been about half full but was later found to contain only 25% of the syrup. The child was playing alone in the living room when his sister found him sitting on the floor, holding a bottle of baclofen syrup. She observed yellowish residue, consistent with the medication's color, around his mouth. She immediately washed his mouth and gave him water. Two hours post-ingestion, he became drowsy and less active, prompting urgent hospital transport.

During transport, he had two episodes of vomiting. Otherwise, he had no seizures, abnormal movements, cyanosis, breathing difficulty, diarrhea, abdominal pain, haematemesis, melaena, sweating, rashes, and bowel or urinary incontinence. Prior to the incident, there was no recent or preceding head trauma or clinical signs and symptoms suggestive of meningitis.

The patient was born at 38 weeks via a perimortem cesarean section following a maternal cardiac arrest due to an amniotic embolism. He had a poor Apgar score and required resuscitation at birth. Consequently, he was diagnosed with neonatal encephalopathy and underwent therapeutic hypothermia. He was discharged on day 8 of life with normal neurology. Serial magnetic resonance imaging (MRI) of the brain showed a small subdural collection with no evidence of hypoxic-ischaemic encephalopathy. His mother had been bedridden since the postpartum period. The patient has had no further admissions to the hospital after that incident. This was his first accidental medication ingestion. His immunisations were up to date for his age.

At his latest follow-up, the main developmental concern was delayed expressive speech, as he could only say one meaningful word, "*baba*." However, his receptive language was appropriate; he could follow commands, identify six body parts, and use gestures to communicate needs. A hearing test two months ago showed normal bilateral hearing, and the audiologist referred him to speech therapy. His gross and fine motor skills were age-appropriate; he could walk, run, climb stairs with support, kick and catch a ball, scribble in circles, and turn book pages. Socially, he could put on slippers, eat with his hands, drink from a cup, pull off his pants, but not put them back on, stay dry during the day, and play with his cousin. No other developmental concerns were noted, and he was otherwise thriving well.

There was no significant family medical or psychiatric history, and no history of self-harm or prior accidental ingestion. Medications were usually stored safely, but the baclofen syrup was unintentionally left in the living room. There was no risk of exposure to illicit drugs, alcohol, or household poisons, and no recent family stressors. His eldest sister, aged 18 years, was his primary caretaker. The father was the sole breadwinner for the family.

Upon arrival at the hospital, three hours post-ingestion, the patient was unresponsive with a Glasgow Coma score of 6 (E1V1M4) and required emergency intubation. He was noted to be bradypnoeic, bradycardic, hypotensive and hypothermic. There were no peculiar breath odor, perioral burns, or drooling. His pupils were constricted and unreactive to light.

Neurological assessment showed generalized hypotonia and areflexia, with no focal deficits or meningeal signs. Fundoscopy was normal with no papilledema or hemorrhages. The rest of the systemic examination was unremarkable.

Laboratory tests, including full blood count, renal profile, and liver function tests were largely unremarkable except for hypokalemia. A venous blood gas was performed on arrival to the emergency department to evaluate respiratory depression, metabolic acidosis, and electrolyte and lactate levels, with results within the normal range (pH: 7.34, HCO_3: 24.8 mmol/L, lactate: 1.8 mmol/L). This suggests a compensated or borderline acid-base status, likely normal or mildly compensated respiratory acidosis. Routine urine toxicology screening was negative, and serum baclofen levels were not available in our setting. A summary of his initial laboratory investigations is shown in Table 1. An electrocardiogram (ECG) showed bradycardia with a prolonged QTc interval.

The patient was transferred to the general ward and gradually recovered. After 19 hours of mechanical ventilation, the patient showed significant improvement and was hemodynamically stable. He regained consciousness rapidly and was successfully weaned off ventilation. While reflexes remained suppressed, muscle tone normalized, and power was normal in all limbs. He was later discharged without neurological sequelae.

Discussion

Baclofen is rapidly absorbed, reaching peak serum levels within 2 hours.[4,5] Approximately 69-85% is excreted unchanged in urine, while 15% is metabolized by the liver.[4] Its moderate lipophilicity allows it to cross the blood-brain barrier, leading to CNS depression at high doses.[5] The half-life ranges from 2-6 hours for oral baclofen and 5-6.8 hours for intrathecal administration.[1] It appears in urine within 2 hours and remains detectable for up to 48 hours in individuals with normal kidney function.[1]

Therapeutic serum levels range from 80 to 400 mg/mL, with severe toxicity in adults usually occurring above 200 mg/mL.[1] However, in children, there is no clear correlation between the dose and the severity of symptoms and the requirement for ventilation.[1] The typical adult dose starts at 5 mg three times daily, with a maximum of 80 mg/day, while pediatric dosing is weight-based (0.3 mg/kg/day for neurological disorders) with a maximum of 1.5-2 mg/kg per day or 80 mg.[6] In this case, the patient was estimated to have ingested a maximum of 300 mg of baclofen, assuming consumption of about 30 mL, or a quarter of a 100 mL bottle (10 mg/mL). The patient's weight was around 15 kg, which meant that the estimated ingested dose was 20 mg/kg, nearly 66 times the maximal daily dose. The upper limit of the dose was considered due to uncertainty about the initial volume, as reported by the caretaker.

This patient presented with coma, hypotonia, areflexia, bradypnea, bradycardia, and hypotension, consistent with baclofen toxicity, which typically manifests within 2-6 hours of ingestion.[1-3,7] Symptoms include respiratory depression, flaccidity, hyporeflexia, hypothermia, seizures, and autonomic disturbances. Autonomic dysfunction may manifest as bradycardia or tachycardia, hypo- or hypertension, and miosis or mydriasis.[7,8]

Cardiac abnormalities like arrhythmias, prolonged QTc, and conduction defects may occur, along with hallucinations (mostly in chronic users) and rhabdomyolysis.[2] Due to baclofen's proconvulsant nature, seizures often occur, but they

Table 1: Summary of initial blood results

Test	Parameter	Result	Normal range	Interpretation
Full blood count	Hb	12 g/dL	11.5-17.0 g/dL	Normal
	WBC	12.2 K/uL	6.0-17.5 K/uL	Normal
	Hct	35.4%	35-54%	Normal
	Plt	215 K/uL	150-400 K/uL	Normal
Renal profile	Na^+	142 mmol/L	135-148 mmol/L	Normal
	K^+	2.9 mmol/L	3.5-5.2 mmol/L	Hypokalemia
Liver function	Total serum bilirubin	0.9 µmol/L	<21 µmol/L	Normal
	AST	18 u/L	5-50 u/L	Normal
	ALT	16 u/L	10-49 u/L	Normal
	ALP	268 u/L	150-420 u/L	Normal
VBG	pH	7.34	7.35–7.45	Slightly acidic
	HCO_3-	24.8 mmol/L	22–26 mmol/L	Normal
	Lactate	1.8 mmol/L	0.5–2.2 mmol/L	Normal

VBG: Venous blood gas, Hb: Hemoglobin, WBC: White blood cell, Hct: Hematocrit, Plt: Platelet count, Na^+: Sodium, K^+: Potassium, AST: Aspartat aminotransferaz, ALT: Alanin aminotransferaz, ALP: Alkalen fosfataz, HCO_3-: Bicarbonate

are generally brief and responsive to treatment.[2] Severe cases can lead to prolonged coma and absent brainstem reflexes, mimicking brain death.[1] Thus, baclofen overdose should be considered in patients with acute unexplained CNS depression, reduced reflexes, seizures, and autonomic instability.

de Marcellus et al.[1] compared 10 reported cases of pediatric baclofen overdose, involving patients aged 1 to 17 years who ingested doses ranging from 20 mg to 2250 mg. The most commonly reported symptoms included coma, hypotonia, hyporeflexia, bradycardia, seizures, hypotension, and hypothermia; however, baclofen blood concentrations varied widely, from undetectable levels to 2009 ng/mL.[1] Electroencephalogram (EEG) findings, when reported, frequently showed suppressed brain activity or seizure patterns, while computed tomography (CT) scans of the brain were mostly normal, though some revealed cerebral edema.[7]

Treatment predominantly involved ventilatory support, with some cases also receiving activated charcoal, gastric lavage, mannitol, or noradrenaline. Clinical outcomes were generally favorable, with most patients recovering and being discharged within 1 to 12 days, although several required prolonged ventilation support.

The patient in this case had risk factors, including a history of neonatal encephalopathy and speech developmental delay. Some literature suggests baclofen intoxication may lead to cerebral edema due to neurotoxicity, blood-brain barrier disruption, increased vascular permeability, respiratory depression, hypoxia-induced cerebral vasodilation, and autonomic dysregulation, all contributing to elevated intracranial pressure and potential ischemic injury.[2,3] However, cerebral edema remains a rare finding.[1]

Baclofen-induced encephalopathy is more common in patients with end-stage kidney disease or acute kidney failure due to impaired renal excretion, leading to drug accumulation and toxicity.[3,4] Therefore, assessing kidney and liver function is crucial in such cases. In this case, a CT scan or MRI may be considered in view of the severity of neurological complications, which could impact the patient's development. An EEG can aid in diagnosis by revealing reversible slow-wave activity and periodic epileptic discharges, while an ECG helps detect cardiac conduction abnormalities such as prolonged QTc, first-degree heart block, premature atrial contractions, and supraventricular tachycardia.[7]

The management of baclofen toxicity is primarily supportive, with gastric lavage and activated charcoal recommended for ingestions exceeding 5 mg/kg.[1,2] Stabilization focuses on airway, breathing, and circulation, with intubation and respiratory support as needed.[1,2] While no specific antidote exists, physostigmine may help with central side effects like somnolence and respiratory depression, though its effectiveness varies.[2] Intravenous atropine may improve cardiac output, bradycardia, ventilation, and temperature regulation.[2,7]

Patients with renal impairment may benefit from hemodialysis, a form of extracorporeal removal, but this effect was not previously documented in children with normal kidney function.[1-3] However, in a reported case of a 6-year-old boy suspected of ingesting 1300 mg of baclofen-one of the highest recorded pediatric doses-despite normal kidney function, hemodialysis was performed because of worsening clinical status and high serum baclofen levels (4 µg/mL, therapeutic range: 0.08-0.4 µg/mL).[3] The treatment proved effective, suggesting hemodialysis may be considered in severe cases, though further research is needed.[3] Overall, supportive care typically results in good outcomes if no hypoxic or ischemic injury occurs. Additionally, no pediatric deaths from baclofen overdose have been reported.[1,2]

This case emphasizes the need to consider baclofen overdose in patients with coma, hypotonia, bradypnea, bradycardia, and hypotension, especially when caregiver history is unavailable. Diagnosing baclofen toxicity is challenging due to its rarity and absence in routine toxicology tests. Clinicians should suspect drug overdose in pediatric cases, particularly in suspected child abuse or neglect. Recognizing its clinical presentation is essential for timely diagnosis, effective management, and better patient outcomes. A notable limitation in this case is the absence of neuroimaging and EEG, which may have provided further diagnostic clarity. Beyond clinical management, this case underscores the need for broader preventive strategies. These include establishing a centralized poison control center to support healthcare providers in toxicology-related cases and implementing structured caregiver education on medication safety-emphasizing the risks of accidental ingestion, adverse effects, and the importance of secure storage.

Ethics

Informed Consent: Informed consent for the publication of this case report was obtained from the patient's father.

Footnotes

Authorship Contributions

Surgical and Medical Practices: N.D.H.S., A.M.K., Concept: A.M.K., Design: N.D.H.S., A.M.K., Data Collection or Processing: N.D.H.S., A.M.K., Analysis or Interpretation: N.D.H.S., A.M.K., Literature Search: N.D.H.S., A.M.K., Writing: N.D.H.S., A.M.K.

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