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CASE REPORT

Baclofen toxicity in a patient with baclofen pump

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SUMMARY

Baclofen toxicity is a severe condition that can suppress brainstem reflexes, leading to coma and death. Therefore, emergency evaluation, correct diagnosis, and treatment are crucial to prevent any possible neurological sequelae. Here, we present a case of a patient with a previous baclofen pump, who was brought to the Emergency Department after being found unresponsive in bed. Brainstem reflexes were absent. The patient was taken to the emergency surgery room, and with the diagnosis of baclofen toxicity, the reservoir was removed from the pump, and cerebrospinal fluid drainage was initiated. The patient was discharged with a baseline neurological examination the following day without any complications.

Keywords: Baclofen pump; baclofen toxicity; coma.

Introduction

Intrathecal baclofen (Lioresal; Saol Therapeutics, Roswell, GA) is a commonly used and well-tolerated medication for the treatment of spasticity. It has an inhibitory effect on GABA-B receptors in presynaptic motor neurons in the spinal cord.[1–3] At therapeutic doses, baclofen acts at the spinal level. At higher dosages, such as in the case of baclofen toxicity, its action penetrates the blood-brain barrier and causes central nervous system depression. Baclofen toxicity may lead to central and respiratory depression, autonomic dysfunction, resulting in deep coma, loss of brainstem reflexes, hypotonia, areflexia, flaccid paralysis, seizures, cardiac arrhythmias, and arrest.[1,2,4]

Baclofen toxicity related to the baclofen pump is rarely reported in the literature, and it is usually iatrogenic.[1,5]

Case Report

A 59-year-old man was brought to the Emergency Department of Kyrenia University Hospital with depressed consciousness and breathing. His wife reported that she was not able to wake him up in the morning. The patient had a history of hypertension and coronary artery disease, and he had been on a baclofen pump for 3 years for the treatment of spastic paraparesis resulting from thoracic arteriovenous fistula. His wife also reported that the baclofen pump had been refilled and the dosage had been increased to improve his gait 5 days previously.

Initial vital signs included a blood pressure of 135/85 mmHg, a pulse of 44 beats per minute, a temperature of 37 °C, and a room air pulse oximetry of 80%. At the initial examination, he had a Glasgow Coma Scale score of 8 points, and his pupils were midsized

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and unresponsive to light. Corneal, oculocephalic, and gag reflexes, as well as deep tendon reflexes, were absent. There were no signs of trauma.

Complete blood count, electrolytes, renal and hepatic panels, and procalcitonin were unremarkable. An ECG showed sinus bradycardia. Cranial magnetic resonance (MR) imaging, including diffusion-weighted (DWI) and three-dimensional time-of-flight MR angiography, did not provide a clear cause for the coma etiology.

He was mechanically ventilated and taken to the surgery room, where 20 cc of baclofen in the pump was removed. Also, cerebrospinal fluid drainage was performed via lumbar catheter. The clinical condition regressed soon after the procedure. He returned to his initial neurological examination the following day.

Discussion

Rapidly progressing coma may present a diagnostic challenge in emergency services. The differential diagnosis is very broad, including toxic, metabolic, traumatic, vascular, neoplastic, and infectious etiologies. We present a unique case of baclofen toxicity presenting with coma. The pre-diagnosis was confirmed through anamnesis in this case. Information regarding the history of the baclofen pump and the increasing dosage was very important. These pump systems are generally considered safe, with reported complication rates between 0 and 5%. These intoxications are usually due to human errors during pump programming, refilling, or both.[3,6,7]

No correlation has been reported between baclofen serum levels and central nervous system depression in the case of baclofen toxicity.[1,8–10] We were not able to determine serum baclofen concentration in our case.

It is known that baclofen produces a global encephalopathy and coma similar to sedative hypnotics.[4] It may also mimic post-hypoxic encephalopathy.[1] A burst suppression pattern in EEG has been reported in patients with baclofen toxicity.[4,11]

In a recent review, baclofen was identified as the second most frequent cause, after neurotoxic snake envenomation, mimicking brain death.[12]

Conclusion

As there is no specific antidote available, management is primarily supportive. The half-life of baclofen in the CSF is 2–5 h.[5]

Another major problem with intrathecal baclofen intoxication is the timing to restart baclofen therapy, as baclofen withdrawal is another life-threatening condition.[13,14]

The presented case was re-transferred to the outpatient hospital, where he is followed with the baclofen pump after successful detoxification. He received a careful slow dosage titration again without further complications. Informed consent was obtained from the patient.

Ethics Committee Approval: This is a single case report, and therefore ethics committee approval was not required in accordance with institutional policies.

Informed Consent: Informed consent was obtained from the patient.

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