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Baclofen Toxicity: A Mimic of Brain Stem Death

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Abstract

Presentation
A 46-year old female presented to the emergency department when her partner awoke to find her seizing. Initially she was spontaneously ventilating with reactive pupils and a Glasgow Coma Scale (GCS) of 3 but five hours after presentation her pupils became unreactive and spontaneous respiratory effort ceased.

Diagnosis
A presumptive diagnosis of baclofen toxicity was made when her partner found empty baclofen packets at home. This was further supported when baclofen levels confirmed a toxic level. All other investigations were normal.

Treatment
No antidote is available so supportive management was continued with intubation for airway protection and management of hypertension

Discussion
The number of presentations of baclofen exposures are increasing worldwide. It should be considered in any suspected overdose who develops signs consistent with brainstem death.

Introduction

We describe a case of a 46-year-old female who, following a baclofen overdose, had signs consistent with brain stem death. Baclofen is a synthetic derivative of gamma-aminobutyric acid which at toxic levels can mimic brainstem death. Patients with baclofen toxicity have been incorrectly diagnosed with brainstem death in the past but have made a full neurological recovery after allowing an extended time for clearance of baclofen[1].

Case Report

A 46-year-old female presented to the emergency department after her partner awoke to find her seizing in bed. She presented with a GCS of three, reactive pupils, hypertensive and spontaneously breathing; tolerating an oropharyngeal airway and a C circuit. Her partner had last seen her awake and intoxicated eight hours prior to the seizure. Her past medical history included depression, alcohol dependency but no history of seizures. Clinically there was a strong suspicion of an overdose.

The decision was made to intubate due to her low GCS. Five hours after her initial presentation her pupils became fixed. On examination, she was making no respiratory effort, had an absent cough and gag reflex and was unreactive to painful stimulus despite sedation hold. She remained hypertensive with systolic blood pressure >180mmHg on propofol infusion of 200mg/hour.

Serial radiological brain imaging over the first six hours of her admission revealed no acute abnormality. Toxicology screen was positive for opioids, but this was ascribed to opioids administered at the time of intubation. Baseline haematological and biochemical investigations were within normal limits.

Her husband returned home and found an empty packet of baclofen which had been prescribed previously for alcohol withdrawal. A baclofen level, taken 16 hours after she was last seen awake (the time of her presumptive overdose), was 1.78mg/L (therapeutic range 0.08-0.6mg/L). Supportive management was continued with the presumptive diagnosis of baclofen toxicity. Dialysis was considered but, given her normal eGFR and urine output, it was felt this would not significantly increase the clearance of baclofen. Her pupils became reactive seven hours later. She was successfully extubated on day 4 of admission. On discharge from hospital she required no medical follow up.

Discussion

Baclofen at therapeutic levels acts on spinal GABAb receptors. In overdose selectivity is lost and GABA receptors in the brain are targeted causing seizures, coma and mimicking brainstem death. Indications include muscle spasticity from MS or spinal cord lesions. Off label uses include alcohol abstinence [3], hiccups and trigeminal neuralgia. Baclofen is 15% metabolised by the liver but otherwise excreted unchanged by the kidneys. Peak serum effect is 2 hours post ingestion. It has a half-life of 3-4 hours [4] but this can be prolonged in toxic doses [7]. The baclofen level taken in this case was 16 hours post ingestion likely significantly underestimating the highest level.

Toxicity can present with CNS and respiratory depression, cardiac arrhythmias and haemodynamic instability. Initial treatment is supportive with early intubation for CNS or respiratory depression and benzodiazepines for seizure activity. Dialysis should be considered in cases of renal impairment.

Risk assessment is important as doses >200mg can cause severe toxicity within two hours of ingestion [2]. Investigations for polypharmacy overdose should be performed. Decontamination with activated charcoal is reasonable if the patient presents within 2 hours of ingestion. There are no antidotes available.

Baclofen toxicity is increasing worldwide. Exposures reported to poison centres in Australia and the USA are increasing [5,6]. A high index of suspicion is required for diagnosis but with supportive treatment outcomes are good [7].

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No author listed has any conflicts of interest.

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