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CASE REPORT

Neurotoxic snake bite mimicking brain death

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Abstract

Neurotoxic snake bites are common in tropical countries, leading to hospital admissions due to symptoms ranging from mild ptosis to severe neuromuscular paralysis with respiratory failure. Among these, krait bites stand out for their potent neurotoxins, which can lead to a deceptive clinical picture mimicking brain death.

We present a case of krait envenomation where the patient presented to the emergency care unit, in a state of pseudo-coma, with fixed dilated pupils and absent brain stem reflexes, resembling brain death. Diagnosing such cases in emergency settings is challenging without proper history and diagnostic tests, often necessitating a high level of suspicion. By recognizing this mimicry, timely administration of antivenom and supportive measures can improve patient outcomes.

KEYWORDS

Brain death mimicry, krait bite, neurotoxin envenomation

INTRODUCTION

Kraits belong to the family Elapidae and the genus Bungarus consists of 14 species distributed throughout Asia.[1] Out of these 14 species, *Bungarus caeruleus* (Common krait) and *Bungarus ceylonicus* (Ceylon krait) are found in Sri Lanka and are reputed to be two of the deadliest snakes in the country. Krait venom contains β-bungarotoxin, a presynaptic blocker that causes muscle paralysis. Krait bites can manifest with varying clinical presentations ranging from mild ptosis and ophthalmoplegia to severe neuromuscular paralysis.[2] While external ophthalmoplegia has an established association with neurotoxin envenomation, in some cases, the occurrence of both internal and external ophthalmoplegia can mimic brain death and can pose a diagnostic challenge, potentially leading to inappropriate withdrawal of supportive care.[3]

CASE PRESENTATION

A 39-year-old male was transferred to the emergency unit from a local hospital after being found unresponsive in his paddy field. Upon admission to the local hospital, his Glasgow Coma Scale (GCS) was recorded as 3, with sluggish bilateral pupillary reactions. His oxygen saturation was normal, his pulse rate was 110/min, and his blood pressure was 140/90 mmHg. His Whole Blood Clotting Time (WBCT) was less than 20 minutes. However, he later developed low oxygen saturation and signs of impending respiratory arrest, which necessitated intubation. He was intubated and transferred to the General Hospital Polonnaruwa with a provisional diagnosis of stroke or seizure. Although, there was no clear history available upon admission, later his family members revealed that he had been sleeping on the bare ground of the paddy field the night before under the influence of alcohol with friends.

At the General Hospital Polonnaruwa, the patient exhibited a persistently low GCS score of E1VTM1, fixed dilated pupils, absent doll's eye reflex, and flaccid limbs with absent reflexes. His plantar response was bilaterally equivocal. Optic fundi were normal.

His initial investigations, including a full blood count, serum electrolytes, liver function tests, and a non-contrast computed

tomography (CT) scan of the brain, yielded normal results. Toxicology screening was negative. Arterial blood gas analysis was not performed at the local hospital due to unavailability. However, the one done in the intensive care unit (ICU) after intubation was normal.

On the seventh day of ICU care, in the absence of brainstem reflexes-including pupillary, gag, cough, corneal, and oculocephalic reflexes – an electroencephalogram (EEG) and CT cerebral angiogram were performed. The EEG was normal, excluding alpha coma and status epilepticus, and the cerebral angiogram revealed normal cerebral blood flow. Cerebrospinal fluid analysis and microbiology culture results were unremarkable.

His GCS remained 3 with fixed dilated pupils over next two weeks. However, the ventilatory support continued, given the suspicion of a krait bite, supported by a prick mark observed over the ankle resembling a fang mark. Antivenom was not given due to the delay in presentation to the general hospital. Over his ICU stay, the patient received supportive care including physiotherapy. The patient gradually improved and flickers of eye movements were noted on day 10. He was able to wean off ventilator support on the 16th day after admission and stepped down from the ICU on the 18th day. He gradually regained his motor functions over time and was able to walk with support by the end of fourth week. Retrospective history upon recovery excluded the possibility of poisoning.

DISCUSSION

Snake bites are a significant medical concern, with krait bites being particularly dangerous due to their neurotoxic effects. Krait venom contains β-bungarotoxin, a presynaptic blocker that leads to muscle paralysis by hindering acetylcholine release at motor nerve endings. This can mimic brain death, complicating diagnosis in emergency settings.

People who sleep on the ground in incompletely built houses and huts are prone to be bitten by these snakes if they are disturbed.[1] Most bites are inflicted on sleeping victims. As a result, the bite site could be anywhere, including such areas as the trunk, scalp, face, genitalia and buttocks.[1] Bites are often painless and without skin manifestations like swelling or necrosis. Early symptoms include nausea, vomiting, fatigue, and abdominal pain, progressing to severe paralysis. The paralysis starts with ptosis and external ophthalmoplegia and affects the distal muscles later.[1] Autonomic disturbance included transient hypertension, tachycardia, lacrimation, sweating, and salivation.[4] Dilatation of pupils is due to the paralytic action of krait venom on cholinergic receptors. The sphincter pupillae is rich in acetylcholine, and its dense microvascular circulation makes it highly susceptible to venom. Krait venom irreversibly blocks acetylcholine receptors in this muscle, leading to prolonged, non-reactive pupil dilation.[4] In patients with neurotoxin envenomation, death is usually caused by secondary respiratory failure. The presynaptic action of krait venom requires new neuromuscular junction formation for recovery, leading to a slow clinical improvement.[5]

The course of neuromuscular paralysis from krait envenomation includes a rapid-onset phase resulting in profound paralysis within 30-60 minutes, a stable phase with deep paralysis persisting for 2-3 days and a recovery phase during which gradual return of neuromuscular function occurs over 2-3 weeks.[5]

Prompt medical attention and the early administration of an appropriate dose of anti-venom serum can neutralise circulating venom and potentially improve outcomes. Complete recovery from krait envenomation is thought to involve the regeneration of damaged neuromuscular junctions. Currently, no effective treatment measures exist once significant neuromuscular junction damage or receptor degeneration has occurred.[2]

Dilated, non-reactive pupils in krait envenomation should not be misconstrued as irreversible brain damage. EEG, transcranial Doppler ultrasonography, and cerebral angiography provide valuable diagnostic tools to distinguish between brain death and envenomation.

Our patient did not receive antivenom due to the delayed presentation. It took almost eight weeks for his complete recovery. Supportive management remained the cornerstone of treatment, although the recovery took longer than expected.

Learning points

Early and accurate diagnosis is critical for patients with krait envenomation, especially those presenting with absent brainstem reflexes, including dilated and non-reactive pupils. While this symptom can be mistaken for brain death, it does not necessarily indicate irreversible brain damage. Misdiagnosis can lead to the withdrawal of vital support like ventilation, putting the patient at greater risk. Utilising diagnostic tools like CT angiogram, EEG and providing continued supportive care are crucial for a good prognosis in cases of krait envenomation.

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