

Snakebite Masquerading as Brain Death: A Case Report from Surkhet, Nepal

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ABSTRACT

Neurotoxic envenomation from snake bites, particularly krait bites, can mimic brain death. A 17-year-old male was referred to our hospital intubated, presenting with clinical features consistent with brain death. However, the absence of a clear preceding event and his residence in a snakebite-endemic region raised suspicion of snakebite. Prompt administration of anti-snake venom led to significant recovery, and he was discharged without neurological deficits. This case underscores the need for clinicians to recognize neurotoxic snake bites as a potential differential diagnosis of brain death, particularly in resource-limited endemic areas where diagnostic capabilities may be restricted.

Keywords

Anti-snake venom; brain death; snakebite

INTRODUCTION

Snakebites are a serious health issue in Nepal, especially in the Terai region, with 20,000–37,661 cases and up to 3,225 deaths annually.^{1,2} Symptoms vary widely and can mimic conditions like brain death. This report from Surkhet, Nepal, presents a case of a young male initially managed as brain death of unknown cause but was later successfully treated as a case of snakebite envenomation based on epidemiological and clinical suspicion. The case highlights diagnostic challenges and emphasizes the need for increased awareness and training among healthcare professionals about conditions that can mimic critical neurological states but remain reversible.

CASE PRESENTATION

A 17-year-old male was intubated and referred to our hospital after a seizure at a nearby facility. The patient had presented to the facility after he developed difficulty swallowing, neck weakness, nausea, abdominal pain, and vomiting while asleep. At the initial hospital, he experienced

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difficulty speaking and breathing, followed by hypoxia (oxygen saturation of 35%) and seizure, leading to intubation. There was no definite history of snakebite, visible bite marks, illicit drug use, trauma, poisoning, or chronic illness.

On arrival, the patient had a GCS of E₁V₁M₁, BP 140/90 mm Hg, pulse 134 bpm, and SpO₂ 99% on AMBU bag ventilation (15 L/min O₂). He was afebrile, with bilaterally dilated (6 mm), non-reactive pupils. Oculocephalic and corneal reflexes were absent, with no spontaneous breathing or response to tracheal suctioning. Limb reflexes were absent. Laboratory investigations including coagulation studies, and head CT were normal. These findings were consistent with the presentation of brain death.

The patient was admitted to the ICU and placed on volume-assist control mechanical ventilation with a fraction of inspired oxygen at 40%. The rapid onset of symptoms resembling brain death in an otherwise healthy young adult from a snakebite-endemic region raised concerns about an unnoticed snakebite. After discussing this possibility with the family, they consented to administer anti-snake venom (ASV) on the second day of admission. A total of 150 ml of polyvalent ASV was given. Shortly after, flickers of movement appeared in the lower limbs, and his pupils began to constrict and respond to light. Over the next four days, he gradually regained limb strength and started breathing spontaneously. He was extubated on the sixth day of mechanical ventilation, with muscle strength improving to 4/5 in both upper and lower limbs. Following extubation, the patient provided the history of feeling something cold on his upper limb prior to onset of the symptoms. His strength continued to improve over the following week, and he was discharged with full muscle strength. A follow-up visit a month later showed no residual neurological deficits.

DISCUSSION

In Nepal, venomous snakes belong to two families: Elapidae and Viperidae. Cobras and kraits, which are common neurotoxic snakes, belong to the Elapidae family, while pit vipers are part of the Viperidae family. Krait bites are typically painless and may leave no visible marks. Most occur indoors, primarily at night.³ In addition to neurotoxicity, abdominal pain is a common symptom of a krait bite.⁴

Neurotoxicity from snake bites occur either due to blocking of post-synaptic receptors or inhibition of the release of acetylcholine at the neuromuscular junction. Common neurotoxic symptoms include ptosis, ophthalmoplegia, non-reactive pupillary dilation, difficulty opening the mouth, inability to protrude the tongue past the incisors, difficulty

swallowing, inability to lift the neck while lying down, limb weakness, loss of the gag reflex, and respiratory failure.⁵ Recovery from krait envenomation is slow with the duration of recovery ranging from 30 hours to 6 days.⁶

Since a Krait bite may not leave a visible bite mark, it is often difficult to suspect snake envenomation when the patient's history cannot be obtained. The above case was initially misdiagnosed as a seizure disorder with coma. A strong clinical suspicion by the ICU medical team led to the prompt administration of anti-snake venom, and the patient began to show clinical improvement. The history of a possible snake crawling over the patient's upper limb resulting in cold sensation, followed by difficulty swallowing and lifting the neck, was only obtained after the patient was extubated.

Brain death is defined as the irreversible cessation of all cerebral and brainstem activity. The diagnosis of brain death requires persistent coma, absence of brainstem reflexes, and the inability to breathe independently.⁷ Neuromuscular paralysis caused by a neurotoxic snake bite can lead to all the clinical features necessary for diagnosing brain death.^{6, 8-10} Misdiagnosis of brain death in snake bite can result in the fatal withdrawal of ventilatory support. In such cases, additional tests to confirm brain death—such as electroencephalography, transcranial Doppler ultrasonography, cerebral angiography, or radionuclide imaging—may be considered.^{6,7} However, these tests are not always readily available, and in resource-limited areas where snake bites are common, the vigilance of treating physicians is crucial for the prompt diagnosis and treatment of these patients.

Similar cases have been documented by Agarwal et al.,⁶ Dayal et al.,⁸ John et al.¹⁰ These reports focus on instances of painless Krait bites in India, where, in many cases, the snake bite was not directly observed. To our knowledge, this is the first report of such cases from Nepal.

CONCLUSION

The case presented above most likely involved a painless krait bite that caused neurotoxicity, mimicking the clinical presentation of brain death. This case emphasizes the importance of considering snake bites in the differential diagnosis of brain death, particularly in otherwise healthy young patients. Prematurely diagnosing brain death, failing to administer anti-snake venom, and withdrawing ventilatory support too early in such cases can have catastrophic consequences.

CONSENT

Written informed consent was taken from the patient for the case report publication.

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CONFLICT OF INTEREST

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