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*Case Report*

# Apparent brain death in snakebite: A reversible neurotoxic crisis

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## ABSTRACT

In the field of toxicology, snake bites are recognized as a significant cause capable of mimicking brain death. A neurotoxic snakebite can manifest with a wide range of symptoms, causing asymptomatic cases to more severe presentations. Common symptoms include ptosis and complete external and internal ophthalmoplegia, which indicate paralysis of the eye muscles. In severe cases, the bite can lead to complete respiratory failure, necessitating urgent medical intervention. Here, we present a patient with a neuro paralytic snake bite with conditions mimicking brain death. The patient achieved full neurological recovery following treatment with polyvalent snake antivenom and supportive care. This case underscores the importance of considering the neuroparalytic effects of snake bites when assessing patients, as it may lead to a false diagnosis of brain death. Intensivists should carefully evaluate these potential effects before deciding to withdraw ventilatory supp]ort or consider organ donation.

**Keywords:** Brain death, Neurotoxicity, Snake bite

## INTRODUCTION

Snake bites in India are a significant public health issue, especially in rural areas where people often live close to snake habitats. There are more than 2,000 snake species globally. India hosts 52 venomous snakes, mainly belonging to three families: Elapidae (cobras and kraits), Viperidae (vipers), and Hydrophiinae (sea snakes).[1] Each year, India reports over 500,000 snakebites, though only around 30% are from venomous snakes. Published mortality figures indicate approximately 35,000 to 50,000 deaths annually, likely an undercount. The clinical presentation can vary widely, from asymptomatic cases to severe local tissue damage, and includes potentially life-threatening symptoms such as neuroparalysis, vasculotoxicity, and myotoxicity, depending on the snake involved.[2] A rare neurotoxic effect can mimic brain death, causing complete ophthalmoplegia from severe envenomation.

Brain death is diagnosed when a patient demonstrates irreversible loss of all functions of the brain, including the brainstem. The three essential findings in brain death are coma, absence of brainstem reflexes, and apnea. We discuss a case of a patient who initially seemed to be brain dead due to a neurotoxic snakebite but ultimately recovered fully with supportive care and anti-snake venom (ASV). Additionally, we intend to enhance awareness among healthcare professionals about the possibility of snakebite-induced effects that can resemble brain death. We suggest that this should be considered as a differential diagnosis before ending supportive care, especially in areas where neurotoxic snakes are found.

## CASE REPORT

### Case: 1

A 14-year-old male presented to the medicine emergency of our hospital with an alleged snake bite at around 01:00 while he was asleep at home. The patient's attendants stated that a black-colored, striped snake bit him on the right side of his neck. It had to be pulled away with considerable effort, but they had managed to bring him to the hospital within 40 minutes of the bite. The patient complained of pain in the abdomen, blurring of vision, and shortness of breath. On initial examination, the patient appeared drowsy and unable to speak in full sentences; his pulse rate was 102 per min, blood pressure was 132/92 mm Hg, and respiratory rate (RR) was 12 per min, and his saturation was 96% on room air. His single breath count was 12. Fang marks were visible on the right side of the neck, with surrounding redness and edema.

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On central nervous system examination, he was drowsy. Ptosis was apparent; pupils were of normal size bilaterally and reacting equally to light. Ten vials of polyvalent ASV were administered immediately, considering the given symptoms. On re-examination after 15 minutes, the patient's condition had significantly deteriorated, with Glasgow coma scale (GCS) E2V2M5, RR 8 breaths per minute (BPM), and single breath count had dropped to below 6. The ptosis increased, and pupils were mid-dilated but reactive to light. He was administered an additional 10 vials of polyvalent ASV along with neostigmine repeated at 30-minute intervals, while electively intubating him in view of respiratory failure. Routine blood investigations were unremarkable with hemoglobin of 12.23 g/dL, total leukocyte count of 7,700/μL, platelet count of 1,93,000/μL, urea of 20 mg/dL, and creatinine 0.5 mg/dL. He was then shifted to the ICU from the emergency room. On examination, 4 hours later, the patient was comatose with a GCS of E1VTM1. CNS examination revealed bilateral plantar reflexes were mute, bilateral pupils were fixed and dilated, and corneal reflexes were absent. Doll's eye reflex was also absent. The patient did not have any spontaneous respiration and was put on pressure support ventilation (assisted ventilation).

Assessment after 6 hours revealed similar findings, suggesting the possibility of brain death. However, supportive care was continued. Another five vials of polyvalent ASV were administered, amounting to a total of 25 vials over 10 hours. Almost 30 hours later, he started showing signs of recovery, with flickering movements of his distal extremities and spontaneous blinking. He showed gradual recovery and became fully conscious and started to follow commands at around 48 hours after the incident; however, he did not regain full muscle power then. He was kept on a mechanical ventilator for 5 days, during which he recovered his muscle strength. The patient was then shifted to synchronized intermittent mandatory ventilation on day 5 and was weaned off the ventilator on day 7 of admission.

**Case: 2**

A 15-year-old male from Meerut, Uttar Pradesh, presented to the hospital with complaints of abdominal pain, dysphagia, and rapidly progressive shortness of breath. The symptoms began suddenly with generalized abdominal pain, which was severe, dull aching, and non-radiating. Over the next few hours, the patient developed dysphagia for both solids and liquids, associated with throat pain, followed by an insidious onset of shortness of breath, which progressed rapidly from Modified Medical Research Council (MMRC) grade 1 to grade 4. He was intubated at a private hospital in view of impending respiratory failure and referred to a tertiary care center.

At admission, on general examination, the patient was unconscious. The patient did not have spontaneous respiratory efforts and was put on mechanical ventilation in the volume control mode. His vital signs were stable, with a supine blood pressure of 124/80 mmHg, a pulse rate of 92 beats per minute, a respiratory rate of 18 breaths per minute, and oxygen saturation of 99% on mechanical ventilation.

On neurological examination, the patient was unconscious with a GCS score of E1VTM1. Pupils were bilaterally mid-dilated and non-reactive, with absent doll's eye reflexes. The patient had decreased tone in all four limbs and no voluntary movement even on painful stimulus. Superficial and deep tendon reflexes were absent in both upper and lower limbs, and bilateral plantar reflexes were mute. Higher mental functions, cranial nerve, and sensory examination, muscle power grading could not be assessed due to the patient's unconscious state. Other systemic examinations were within normal limits. Complete assessment suggested the possibility of brain death.

On investigations, hematological findings revealed a hemoglobin of 11.3 g/dL, a total leukocyte count of 4800/mm$^3$, and a platelet count of 2.9 lakhs/mm$^3$, all within normal ranges. Renal function tests, liver function tests, and serum electrolytes were within normal limits. Chest X-ray and ECG showed no abnormalities. Fundoscopic examination showed no signs of papilledema. Urine toxicology screening was negative for common toxins. Cerebrospinal fluid analysis showed normal glucose (59 mg/dL) and protein levels (22 mg/dL), with no cells or growth on gram staining and culture. A computed tomography scan of the head showed no abnormalities.

These findings suggested severe neurotoxic effects, consistent with suspected snakebite envenomation, despite the absence of visible bite marks. A clinical suspicion of neurotoxic snakebite envenomation was made, and he was treated empirically with 20 vials of ASV on day 1, followed by 10 vials each on days 2 and 3. Initial improvement was limited, with persistent low GCS and non-reactive pupils until day 5, when signs of recovery, including head nodding and slight improvement in sensorium emerged. Neurological recovery was gradual, with distal muscle power improving first, followed by proximal and truncal strength. A nerve conduction study was planned later in the disease, which indicated axonal motor polyneuropathy involving bilateral peroneal nerves, consistent with neurotoxic envenomation The clinical course was complicated by ventilator-associated pneumonia, necessitating prolonged ICU care. He was extubated on day 9. Over the next 20 days, the patient demonstrated gradual and complete neurological recovery.

## DISCUSSION

Snake bites in India are a serious public health issue and a major cause of accidental and occupational mortality. Neuroparalytic snake bites primarily result from the action of neurotoxic venom, which disrupts normal neuromuscular function. Many neurotoxic venoms, such as those from cobras and kraits, contain toxins that bind to nicotinic acetylcholine receptors at the neuromuscular junction.[3] This binding inhibits the action of acetylcholine, a neurotransmitter essential for muscle contraction, leading to paralysis. Additionally, some venoms include enzymes, such as phospholipases, which can damage the presynaptic membranes of nerve terminals.[4] These neurotoxins can also affect the excitability of motor neurons, resulting in a failure to transmit nerve impulses, which leads to weakened or absent muscle contractions. Symptoms often progress in a characteristic pattern known as descending paralysis, where paralysis begins in the eyes and facial muscles and then descends to the neck, trunk, and limbs. In severe cases, paralysis of the respiratory muscles can occur, leading to respiratory failure, a common cause of death in severe envenomation.

Our patient presented with symptoms and signs of neuroparalysis and respiratory failure. His initial condition mimicked a situation of "brain death." However, he was continued on supportive care and recovered later. He showed initial improvement at around 30 hours post-admission and then went on to fully recover his consciousness and muscle strength by day 5. The snake, in our case, looked like a krait by the description given by the attendants of the patient. As krait venom binds irreversibly to the pre-synaptic receptors, the recovery is slow, and the duration varies from 30 hours to 6 days. During these lag hours, the patient's condition may mimic brain death. Supportive care might be withdrawn due to such a misdiagnosis, which should be avoided.

A similar case has been reported by Agarwal *et al.*[1] (2018) from Uttarakhand of an 18-year-old male who presented with a history of pain in the abdomen, vomiting, and frothing from the mouth in a comatose state, with atonic features, areflexia, fixed dilated pupils, absent respiratory efforts, and a GCS of E1VTM1. Initial investigations were largely normal. Despite no history of snakebite, polyvalent ASV was administered for suspected severe neurotoxic envenomation. He showed gradual improvement, with flickering movements after 8 hours and respiratory effort. A second ASV dose was given at 12 hours, and he was extubated on day 4, with improved muscle power but residual truncal weakness. This was similar to our case, as there was no history of snake bite, nor any fang marks, and the patient improved after being treated with the assumption of a snake bite.

Another case reported by John *et al.*[5] (2008) was of a 6-year-old female who developed pain and swelling in her left hand overnight, and later developed areflexia and brainstem dysfunction. Examination revealed puncture marks on the thumb, ptosis, limb weakness, and shallow breathing. Despite no history of snakebite, severe envenomation was suspected, and 100 ml of ASV was administered. After 36 hours, she showed signs of recovery, with paralysis improving distally first. She was extubated after five days but had residual truncal weakness. This case, similar to ours, highlights the importance of high clinical suspicion of snake bite and immediate management.

Similar cases have been reported by Dayal *et al.*,[6] (2014) and Prakash *et al.*,[7] (2008) where snake bite cases mimic brain death, with subsequent recovery. Hence, the clinicians must expect this "brain-dead" presentation of neurotoxic snake bites, especially after rains, floods, during harvest, and at night. Withdrawing the supportive care in these cases might be disastrous for the patient.

## CONCLUSION

Neurotoxic envenomation can simulate the clinical picture of brain death, necessitating cautious interpretation of neurological findings. Timely administration of polyvalent anti-snake venom and meticulous supportive care can result in complete neurological recovery, even in apparently brain-dead patients. Clinicians practicing in endemic regions should recognize this reversible neurotoxic crisis to avoid premature declaration of brain death. Enhanced awareness and adherence to structured protocols are essential to improve outcomes in such cases.

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