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Case Series: Neurological Manifestations Following Snake Bite in Rural India

Case Report

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Abstract

Snakebite remains a significant health hazard in rural India, with neurotoxic envenomation posing a life-threatening challenge. This case series describes four patients presenting with varied neurological manifestations following snakebites, managed at a secondary care center in northern India. The report highlights the need for early recognition and timely administration of antivenom to prevent complications and mortality.

Case 1: Early Morning Krait Bite with Delayed Respiratory Paralysis

A 22-year-old male farmer was brought to the emergency department at 7:30 AM with complaints of blurred vision, ptosis, and difficulty breathing. The patient had slept on the floor of his thatched house and did not notice any bite. Neurological examination revealed bilateral ptosis, ophthalmoplegia, and early signs of respiratory muscle weakness. Pupils were dilated but reactive. No local signs of envenomation were noted.

A presumptive diagnosis of krait bite was made based on clinical features and setting. ASV was administered within 1 hour of presentation. The patient was intubated and ventilated for 48 hours and gradually regained muscle power. He was discharged on day 5 with full recovery.

Case 2: Cobra Bite with Rapid-Onset Bulbar Palsy

A 34-year-old woman presented to the emergency room 2 hours after being bitten on the right hand while working in a paddy field. She developed drooling, dysphagia, and slurred speech within an hour. Examination revealed right upper limb swelling and fang marks, along with cranial nerve involvement (VII, IX, X, XII).

ASV therapy (10 vials) was initiated immediately along with supportive care. Although her bulbar symptoms worsened initially, she did not require intubation. Neurological improvement began by day 3, and she was discharged by day 6 with residual mild dysarthria.

No neuroimaging was performed during the hospital stay, as the clinical presentation and bite history strongly suggested cobra envenomation and the patient showed clinical improvement with antivenom therapy.

Follow-Up: The patient was advised regular follow-up in the neurology outpatient department. Speech therapy was initiated to aid recovery.

At her 3-week **follow-up visit**, she reported improvement in her speech

Case 3: Snakebite Mimicking Brainstem Stroke

A 40-year-old male presented with acute-onset ptosis, external

ophthalmoplegia, and areflexic quadriparesis. Initial differential included brainstem stroke, Guillain–Barré syndrome, and botulism. However, the patient reported a snake bite to his left foot while walking through farmland the previous night.

MRI brain was normal. Given the setting, nocturnal bite, absence of local reaction, and pattern of descending paralysis, a **krait (Bungarus spp.) bite** was strongly suspected. ASV was administered with good response.This case emphasizes the potential for neurotoxic envenomation to masquerade as central nervous system pathology.

Case 4: Pediatric Case with Seizure and Neuroparalysis

An 8-year-old girl was admitted with generalized tonic-clonic seizures followed by flaccid paralysis and respiratory distress. The parents recalled a snake sighting near her sleeping mat the previous night. She exhibited bilateral ptosis, dilated pupils, and shallow respiration. No bite marks were visible.

She was intubated and given ASV empirically. EEG showed generalized slowing, likely due to hypoxia or venom effect. **MRI brain and nerve conduction studies were not performed**, as the clinical context and rapid response to ASV supported a diagnosis of neurotoxic envenomation, most likely due to **krait .**After 72 hours of ventilation and supportive care, she showed gradual recovery of muscle strength. She was extubated on day 4 and discharged neurologically intact on day 8.Given the nocturnal presentation, absence of local signs, and severe neuroparalysis with seizures, a **krait (*Bungarus* spp.) bite** is the most probable etiology.

Discussion

Neurotoxic snakebites in India, primarily caused by kraits (Bungarus spp.) and cobras (Naja spp.), frequently result in a wide spectrum of neurological manifestations. These range from isolated cranial nerve palsies to generalized flaccid paralysis and respiratory failure. The neurotoxins involved—primarily alpha- and beta-neurotoxins—interfere with neuromuscular transmission either pre- or postsynaptically, contributing to the classic descending paralysis observed in envenomed patients [1].

A notable challenge, especially in rural settings, is the lack of visible bite marks or a clear history of envenomation, as seen in krait bites, which often occur at night while the victim is asleep on the floor [2]. This can lead to misdiagnosis as neurological disorders such as brainstem stroke, Guillain–Barré syndrome (GBS), or botulism, potentially delaying appropriate treatment [3]. In such scenarios, a high index of clinical suspicion and knowledge of endemic species are critical for early diagnosis.

This case series demonstrates several classical and atypical presentations:

- **Case 1 and Case 3** are characteristic of krait envenomation with minimal or absent local signs, nocturnal bite, and rapid progression to neuromuscular paralysis. Case 3 initially mimicked a central neurological event, underlining the importance of considering snakebite in the differential diagnosis for acute flaccid paralysis in endemic areas.
- **Case 2**, most likely due to a cobra bite, presented with rapid-onset bulbar palsy and local signs. Bulbar weakness is a typical manifestation of postsynaptic neurotoxins found in cobra venom. Interestingly, despite significant cranial nerve involvement, the patient did not require mechanical ventilation and showed partial recovery with ASV and supportive care.
- **Case 4** uniquely involved a paediatric patient with seizures followed by neuroparalysis. Seizures are uncommon in snakebite victims and may be related to hypoxic encephalopathy, **direct neurotoxicity**, or less commonly, metabolic derangements. Although MRI and nerve conduction studies were not performed, clinical presentation and rapid improvement post-ASV pointed to a likely krait bite.

Across all cases, anti-snake venom (ASV) therapy formed the cornerstone of management. Timely administration—ideally within 4 hours—is associated with significantly better outcomes[4]. However, supportive care, especially respiratory support, remains critical and often determines survival, particularly in settings where ASV response may be delayed or incomplete.

The absence of advanced diagnostics in rural settings poses a further challenge. While imaging and nerve conduction studies may help differentiate neuroparalytic syndromes, clinical context and timely therapeutic trials of ASV remain pivotal where resources are limited. Notably, early neurorehabilitation, including speech and physical therapy, plays a key role in managing persistent deficits, as illustrated in Case 2.

Conclusion

Snakebite envenomation with neurological manifestations poses diagnostic and management challenges in rural India. This case series underscores the variability of presentation and the importance of early clinical recognition and intervention. Strengthening rural healthcare systems with better awareness, timely ASV availability, and ventilatory support can reduce morbidity and mortality.

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