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Indian Journal of Clinical Anaesthesia

Journal homepage: www.ijca.in

Case Report

Occult neuroparalytic snakebite: A diagnostic challenge

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Abstract

A 48-year-old woman with hypothyroidism presented with sudden hives and itching, later developing quadriparesis and reduced responsiveness. Initially treated for an allergic reaction, she was brought to the emergency department with respiratory distress, ptosis, and quadriparesis. Primary investigations, including arterial blood gas, magnetic resonance imaging, and cerebrospinal fluid studies, were unremarkable. Suspecting an occult neuroparalytic snakebite, she received 20 vials of antivenom with atropine and neostigmine, resulting in gradual improvement. She was extubated on day 6 and discharged on day 10 with full recovery. The case highlights the need to consider snakebites in unexplained neuroparalysis.

Keywords: Occult snake bite, Neuroparalysis, Poisoning, Antisnake venom.

Received: 20-12-2024; **Accepted:** 23-01-2025; **Available Online**: 16-04-2025

1. Introduction

Snakebite envenomation is a major medical emergency in tropical regions, with India contributing to the highest global burden. An estimated 45,900 deaths occur annually due to snakebites, primarily affecting rural populations during monsoon months. Kraits (Bungarus spp.) are responsible for most neurotoxic envenomations, often leading to respiratory paralysis and descending neuroparalysis.[1] Early recognition and timely administration of antivenom along with supportive care are critical to prevent morbidity and mortality.[2] In rare cases, snakebites can be occult, where no bite marks are visible or the history is unclear, complicating diagnosis and delaying appropriate management. This report highlights the challenges and successful management of a case of occult neuroparalytic snakebite, emphasizing the importance of clinical suspicion in unexplained neuroparalysis.

2. Case Report

A 48-year-old woman with a history of hypothyroidism experienced a sudden awakening at 2 am due to generalized itching and the appearance of hives all over her body. She was treated at a local hospital with intravenous pheniramine and hydrocortisone, which relieved her symptoms temporarily. However, she subsequently developed acute reduced responsiveness and quadriparesis, preceded by tingling sensations in her upper limbs.

On arrival at the emergency department, her airway was patent but unstable, with oxygen saturation at 90% on room air. She exhibited significant respiratory distress, with a respiratory rate of 42 breaths per minute and increased work of breathing, though her trachea was midline, and chest rise was symmetrical. Circulatory examination revealed normal findings, including warm skin, capillary refill time of less than three seconds, and a normal pulse. Neurological assessment showed a Glasgow Coma Scale (GCS) of E3V1M6, bilateral reactive pupils measuring 2.5 mm, and equivocal plantar reflexes. Blood sugar was 128 mg/dL. The

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https://doi.org/10.18231/j.ijca.2025.061

patient was immediately intubated for respiratory distress and placed on mechanical ventilation.

During further evaluation, the patient demonstrated progressive flaccid paralysis, including respiratory muscle involvement, within 24 hours. Initial ptosis evolved into worsening weakness, with diminished deep tendon reflexes. Muscle power was graded as 1/5 in the distal upper limbs, 2/5 in the proximal upper limbs, 1/5 in the distal lower limbs, and 3/5 in the proximal lower limbs. Secondary survey findings included a soft neck, absence of meningeal signs, and normal muscle tone and bulk.

The *differential diagnoses* included cerebrovascular accident (CVA), Guillain-Barré Syndrome (GBS), myasthenia gravis, botulism, dyselectrolytemia, poisoning, and myxoedema. Comprehensive investigations, including blood tests, MRI brain and spine, and cerebrospinal fluid analysis were unremarkable. Myasthenia gravis and botulism toxin profiles were negative (**Table 1**).

On the second day of hospitalization, the patient's condition worsened with progressive ptosis and a further decline in Glasgow Coma Scale (GCS). A detailed re-evaluation of her history revealed that she had been normal the previous night but woke up with tingling sensations, which progressed to quadriparesis and ptosis. This clinical picture raised the suspicion of an occult neuroparalytic snake bite. Consequently, the patient was administered 10 vials of antisnake venom (ASV) infusion over 30 minutes, along with intravenous atropine (0.6 mg) and neostigmine (1.5 mg). However, as there was no significant improvement within the first hour, an additional 10 vials of ASV were given, alongside repeated doses of atropine (0.6 mg) and neostigmine (0.5 mg).

The patient exhibited slight improvement in ptosis after the second ASV dose. Atropine and neostigmine were continued for three more doses at 30-minute intervals and then tapered over 12 hours (1 hour, 2 hours, 6 hours, and 12 hours). Gradual clinical improvement was observed, with resolution of ptosis and recovery of muscle power to 4/5. By the sixth day of admission, the patient was extubated and transitioned to face-mask oxygen support. Oxygen therapy was gradually tapered off, and by day 10, the patient's limb power had fully recovered to 5/5. She was subsequently discharged in stable condition.

At her two-week post-discharge follow-up, the patient demonstrated full neurological recovery, with no residual weakness or respiratory compromise, highlighting the efficacy of timely ASV administration and supportive care in managing neuroparalytic snake bites.

Table 1: Key laboratory findings

Parameter	**Patient Value**	**Reference Range**
Complete Blood Count		
Hemoglobin (g/dL)	12.2	12–15
WBC (×10³/µL)	11.01	4–10
Platelet Count (×10³/µL)	150	150–450
Electrolytes (meq/L)		
Sodium	136.7	135–145
Potassium	4.3	3.5–5
Kidney function tests(mg/dl)		
Urea	35.2	13- 43
Creatine	0.81	0.7- 1.3
Calcium	8.91	8.6- 10
CSF Analysis		
Cells (cells/µL)	5	0–5
Glucose (mg/dL)	73	50–80
Protein (mg/dL)	50	15–60
Myasthenia Gravis profile (nmol/l)		
Acetylcholine receptor antibody (ACHR-Ab)	0.04	Normal: <0.4 Borderline: 0.4-0.5 Positive: >0.5
Anti- Musk Ab	0.01	Negative: <0.4

3. Discussion

India accounts for the highest global incidence of snakebites, with an estimated 45,900 annual deaths, predominantly affecting males aged 15–29 years in rural areas.[3] Krait bites, commonly occurring during the monsoon season, are often occult, lacking visible fang marks or a clear bite history, leading to diagnostic challenges and delayed treatment.[4] A mnemonic for identifying symptoms is the "5 Ds and 2 Ps": dysarthria, dysphagia, dyspnea, diplopia, dysphonia, ptosis, and paralysis.[4,5] Previous studies have reported nocturnal krait bites presenting with early morning paralysis, often misdiagnosed as Guillain-Barré Syndrome (GBS) due to overlapping features like flaccid paralysis and respiratory failure.[3,4] Our case exemplifies these diagnostic challenges, with descending paralysis, ptosis, and dyspnea serving as critical markers for early recognition.

Krait venom contains α-bungarotoxins and β-bungarotoxins, which impair neuromuscular transmission by binding to nicotinic acetylcholine receptors and causing presynaptic axon destruction. This pathophysiology explains the delayed recovery observed in many cases despite antivenom administration.[5] While ASV neutralizes circulating venom, its efficacy diminishes once irreversible damage occurs, emphasizing the importance of early intervention. Studies indicate a sensitivity of 85%–90% for ASV therapy in halting the progression of neurotoxicity, although specificity decreases in cases of delayed presentation.[6,7]

Early administration of antivenom (ASV) is critical for neutralizing venom and preventing neurotoxicity progression. ASV efficacy decreases once neuromuscular damage becomes irreversible. Severe cases require intubation and mechanical ventilation. Adjunctive treatments, such as neostigmine and atropine, can improve neuromuscular transmission by counteracting neurotoxic effects.[6-8]

Public health recommendations focus on the need for increased community awareness, improved access to healthcare resources, and the development of standardised protocols to effectively address the challenges posed by snakebite envenomation (**Table 2**).[9,10]

Table 2: Public health recommendations for neuroparalytic snakebite management

Recommendation	Description
Awareness Programs	Educate rural communities and healthcare workers about krait bites and their clinical features to improve early recognition and prompt management.
Strengthening Healthcare Systems	Ensure adequate availability of ASV and ventilatory support in rural and resource-limited health centres to manage envenomation effectively.
Community Education	Promote preventive measures, such as using mosquito nets, to reduce the risk of nocturnal snake bites, particularly from kraits.
Standardized Protocols	Develop and disseminate evidence-based guidelines for the diagnosis and treatment of snakebite envenomation to ensure uniform and effective care.
Training for First Responders	Train primary care providers and first responders in the early recognition and management of neurotoxic snakebites to reduce delays in treatment.

4. Conclusion

Occult neurotoxic snakebites pose unique diagnostic and therapeutic challenges. Early recognition through strong clinical suspicion, timely administration of ASV, and supportive care are paramount to reducing mortality and morbidity. Raising awareness, addressing gaps in healthcare infrastructure, and adhering to standardized protocols can significantly improve outcomes. This case highlights the need for vigilance among primary care providers in endemic areas to identify and treat occult snakebites promptly.

5. Source of Funding

None.

6. Conflict of Interest

None.

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Cite this article: Kumar D, Das U, Vaibhaw V, Kumar S, Bhushan D, Gupta L, Rahul A. Occult neuroparalytic snakebite: A diagnostic challenge. *Indian J Clin Anaesth*. 2025;12(2):390–392.