



SILENT BITE: A DIAGNOSTIC CHALLENGE FROM RURAL INDIA

General Medicine

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ABSTRACT

Aim and Background: Neurotoxic snakebite remains an important cause of morbidity and mortality in rural India. Common krait (*Bungarus caeruleus*) bites are particularly challenging to diagnose because they often occur during sleep, leave minimal local signs, and present with nonspecific neurological manifestations. This case report aims to highlight the diagnostic challenges of occult krait envenomation and emphasize the importance of considering snakebite in the differential diagnosis of unexplained neuromuscular paralysis in endemic regions. **Case Description:** We report a case of a middle-aged female from rural Telangana who presented with sudden onset unconsciousness following alleged toddy consumption and was initially managed as unknown poisoning. Clinical features including urinary incontinence, fecal incontinence, respiratory failure, pinpoint pupils, and markedly depressed serum cholinesterase levels led to an initial diagnosis of organophosphorus poisoning. Despite treatment with atropine and pralidoxime, neurological recovery was inadequate. Subsequent consideration of benzodiazepine intoxication and administration of flumazenil also failed to produce significant improvement. Further history revealed that the patient routinely slept on the floor with her children during the summer season, raising suspicion of occult neurotoxic snakebite. Administration of neostigmine and glycopyrrolate resulted in immediate neurological improvement, followed by dramatic recovery after anti-snake venom (ASV) administration. The patient was ultimately diagnosed with probable neurotoxic krait envenomation. **Conclusion:** Occult neurotoxic krait envenomation should be considered in all cases of unexplained neuromuscular paralysis or suspected poisoning in rural India, particularly during summer months. Failure to respond to conventional poisoning treatment should prompt clinicians to revisit the diagnosis and obtain a detailed environmental history. Early recognition, ventilatory support, anticholinesterase therapy, and timely administration of anti-snake venom can be lifesaving. **Clinical Significance:** This case highlights the importance of maintaining a high index of suspicion for occult krait bite in endemic regions, especially when patients present with unexplained neuromuscular paralysis, respiratory failure, or apparent poisoning syndromes that do not respond to conventional therapy. It underscores the value of detailed environmental history-taking and the potential for life-saving intervention when the correct diagnosis is established.

KEYWORDS

Krait Envenomation; *Bungarus Caeruleus*; Neurotoxic Snakebite; Organophosphorus Poisoning Mimic; Benzodiazepine Overdose; Occult Snakebite; Rural India; Anticholinesterase Therapy; Anti-Snake Venom

INTRODUCTION

Snakebite envenomation remains a major neglected tropical disease in India, accounting for a significant proportion of global snakebite-related mortality. Among the medically important snakes, the Common Krait (*Bungarus caeruleus*) is responsible for a large number of neurotoxic envenomations, especially in rural regions.

Kraits are nocturnal snakes that commonly enter human dwellings during warm summer months in search of prey. Individuals sleeping on the floor, particularly with minimal clothing, are at increased risk of accidental bites. Due to their small fangs and painless bite, victims are often unaware of the envenomation. Local signs such as pain, swelling, or fang marks are frequently absent, making diagnosis difficult.

Neurotoxic manifestations including ptosis, bulbar weakness, respiratory paralysis, altered sensorium, and autonomic dysfunction may mimic organophosphorus poisoning, sedative overdose, Guillain-Barré syndrome, brainstem stroke, or other causes of acute neuromuscular paralysis. Delayed diagnosis may result in respiratory failure and death.

We report a case of occult krait envenomation that initially masqueraded as organophosphorus poisoning and benzodiazepine overdose, posing a significant diagnostic challenge.

Case Description

A middle-aged female from a rural area of Telangana, known to be a chronic toddy drinker, chronic tobacco chewer, and habitual NSAID user, was brought to our emergency department with altered sensorium and respiratory failure.

According to relatives, the patient had consumed toddy and an unknown substance at approximately 12:00 PM on 11 May 2026. About one hour later, she developed sudden urinary incontinence and fecal incontinence and was found unconscious, incoherent, and unresponsive.

She was initially taken to a nearby government hospital where she developed cardiorespiratory arrest. Two cycles of cardiopulmonary resuscitation were performed, and return of spontaneous circulation (ROSC) was achieved. During resuscitation, atropine and adrenaline were administered. Following stabilization, she was referred to our tertiary care center for further management.

On arrival, the patient was deeply unconscious with a low Glasgow Coma Scale score and signs of respiratory failure. She was immediately intubated and placed on mechanical ventilation. Due to hemodynamic instability, inotropic support was initiated, and the patient was transferred to the Respiratory Intensive Care Unit.

Clinical examination revealed bilateral pinpoint pupils. In view of the history suggestive of poisoning and severe cholinergic manifestations, serum pseudocholinesterase levels were measured and found to be markedly reduced. Based on these findings, a provisional diagnosis of organophosphorus poisoning was made.

The patient was treated with atropine infusion and pralidoxime according to standard organophosphorus poisoning protocols. Despite two days of therapy, significant neurological improvement was not observed. Respiratory dependence persisted, and the clinical picture remained unexplained.

Considering the patient's history of chronic toddy consumption, the possibility of benzodiazepine intoxication was entertained. In many parts of rural India, toddy is occasionally adulterated with sedative medications such as diazepam to enhance intoxicating effects. Consequently, flumazenil was administered as a therapeutic trial. However, no appreciable improvement occurred following administration.

The absence of response to both organophosphorus and benzodiazepine-directed therapies prompted a thorough reassessment of the case. Detailed questioning of family members revealed an

important environmental clue: the patient had been sleeping on the floor of her house along with her children during the preceding nights. The event had occurred during the peak summer season, a period associated with increased krait activity in rural India.

In view of the possibility of occult neurotoxic snakebite, a therapeutic trial of neostigmine and glycopyrrolate was administered. Remarkably, the patient demonstrated rapid neurological improvement with increased responsiveness and improvement in sensorium shortly after administration.

Encouraged by this response, 10 vials of polyvalent anti-snake venom were administered intravenously. Within one hour, a significant improvement in Glasgow Coma Scale and neurological status was observed. An additional 10 vials of ASV were subsequently administered, resulting in further clinical recovery.

The patient's neurological status continued to improve, strongly supporting the diagnosis of neurotoxic snake envenomation. The absence of local bite marks and the atypical presentation initially obscured the diagnosis. Based on the epidemiological setting, clinical features, delayed neuromuscular presentation, dramatic response to anticholinesterase therapy, and subsequent improvement following ASV administration, a diagnosis of probable Common Krait envenomation was established.

DISCUSSION

This case illustrates several classical yet frequently overlooked features of krait envenomation.

First, the patient had no witnessed snakebite and no obvious local manifestations. Such presentations are characteristic of krait bites, which are often painless and occur while victims are asleep. Consequently, patients frequently present with unexplained neurological symptoms without any recollection of envenomation.

Second, the patient initially mimicked organophosphorus poisoning. Pinpoint pupils, urinary incontinence, fecal incontinence, respiratory failure, and reduced cholinesterase levels strongly suggested a cholinergic toxidrome. However, the lack of meaningful response to atropine and pralidoxime necessitated reconsideration of the diagnosis.

Third, benzodiazepine intoxication was considered because of the history of toddy consumption. However, failure to respond to flumazenil further supported an alternative etiology.

The crucial diagnostic clue emerged only after obtaining a detailed environmental history. Sleeping on the floor during summer months is a well-recognized risk factor for krait bites in rural India. During hot weather, kraits frequently enter homes in search of rodents and may accidentally bite sleeping individuals. Because the bite is painless and often unnoticed, patients may present hours later with progressive neuromuscular paralysis and respiratory failure.

The dramatic improvement following neostigmine and glycopyrrolate suggested a reversible neuromuscular junction disorder consistent with neurotoxic envenomation. Subsequent rapid neurological recovery following ASV administration further strengthened the diagnosis.

This case highlights the importance of maintaining a high index of suspicion for occult krait bite in endemic regions, especially when patients present with unexplained neuromuscular paralysis, respiratory failure, or apparent poisoning syndromes that do not respond to conventional therapy.



CONCLUSION

Occult neurotoxic krait envenomation should be considered in all cases of unexplained neuromuscular paralysis or suspected poisoning in rural India, particularly during summer months. Individuals sleeping on the floor are at increased risk of nocturnal krait bites, which frequently occur without pain or visible local signs. Failure to respond to conventional poisoning treatment should prompt clinicians to revisit the diagnosis and obtain a detailed environmental history. Early recognition, ventilatory support, anticholinesterase therapy, and timely administration of anti-snake venom can be lifesaving.

Clinical Significance

This case underscores several critical learning points for clinicians practicing in snakebite-endemic regions:

- Krait bites frequently occur during sleep and may go unnoticed.
- Absence of fang marks or local swelling does not exclude snakebite.
- Neurotoxic krait envenomation may mimic organophosphorus poisoning and sedative overdose.
- Summer months and floor sleeping are important epidemiological clues in rural India.
- Failure to improve with standard poisoning treatment should prompt diagnostic reassessment.
- Early ASV administration and respiratory support significantly improve outcomes.

List of Abbreviations

ASV – Anti-Snake Venom

GCS – Glasgow Coma Scale

ICU – Intensive Care Unit

NSAID – Non-Steroidal Anti-Inflammatory Drug

ROSC – Return of Spontaneous Circulation

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