

CASE REPORT

Case Report: Cryptic Neurotoxic Envenomation Mimicking a Localized Facial Reaction – The Critical Role of Syndromic Triage in Unwitnessed Nocturnal Snakebites

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ABSTRACT

Background: Snakebite Envenomation is a time-limiting, acute medical emergency and a significant public health hazard in rural and suburban India. Neurotoxic Envenomation, predominantly caused by the Common Krait (*Bungarus caeruleus*), presents profound diagnostic dilemmas. Because Kraits are nocturnal and possess fine fangs, their bites are frequently painless, lack significant local signs, and can escape the patient's awareness. When a patient presents with atypical soft tissue swelling or an alternative medical history, significant diagnostic delays and rapid clinical deterioration can occur.

KEYWORDS

• Occult Snake Bite • Krait Envenomation • Neurotoxic Snake Bite • Neuro-Paralytic Snake Bite

INTRODUCTION

Snakebite envenomation is a time-limiting, acute medical emergency and a significant public health hazard in rural and suburban India.¹ Neurotoxic envenomation, predominantly caused by the Common Krait

(*Bungarus caeruleus*), presents profound diagnostic dilemmas.² Because Kraits are nocturnal and possess fine fangs, their bites are frequently painless, lack significant local signs, and can escape the patient's awareness.^{2,5} When a patient presents with atypical soft

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tissue swelling or an alternative medical history, significant diagnostic delays and rapid clinical deterioration can occur.^{2,4}

Globally, snakebite stands as a heavily burdened environmental injury, resulting in massive mortality and leaving hundreds of thousands of survivors with permanent structural or functional disabilities.^{4,6} In India, the public health strain is exceptionally high; data from the nationally representative Million Death Study estimates approximately 45,900 to 58,000 annual snakebite deaths.^{1,6} This crisis is exacerbated by immense underreporting, with direct surveys indicating that only about 7.23% of snakebite fatalities are officially documented through standard administrative channels.² This massive data gap is largely driven by the fact that only 22.19% of victims seek immediate hospital care, while a vast majority initially consult traditional community healers.²

While more than 300 snake species exist in India, only 52 are venomous, with systemic envenomation primarily driven by the “Big Four” medically significant species: the Indian Cobra (*Naja naja*), the Russell’s Viper (*Daboia russelii*), the Saw-scaled Viper (*Echis carinatus*), and the Common Krait (*Bungarus caeruleus*).⁴ Among these, the Common Krait is exceptionally lethal, accounting for an estimated 65.7% of all snakebite fatalities nationally, with a significant surge in cases recorded between the monsoon months of June and September.² Kraits are strictly nocturnal predators that regularly enter human dwellings at night.^{3,7} Because a significant portion of the rural population sleeps on mats or floor beds, the vast majority of Krait envenomations occur while victims are asleep.^{3,7}

Pathophysiologically, envenomation by *Bungarus caeruleus* introduces a profound clinical challenge.^{2,4} The venom contains highly potent presynaptic neurotoxins that target the nervous system, inducing a classic flaccid, ascending or descending paralysis by systematically blocking the release of acetylcholine at the neuromuscular junction.^{1,2,8} Without timely emergency intervention, this progressive neuromuscular blockade advances rapidly to complete respiratory muscle failure.^{1,8} Despite this extreme toxicity, Krait bites are clinically deceptive; they have small, slender teeth that induce virtually no localized pain, tissue necrosis, or identifiable bite puncture marks.^{2,5}

This complete absence of classic local envenomation signs represents a dangerous diagnostic trap for emergency departments.^{2,3} Sleeping victims are routinely unaware of the bite and frequently present with vague, non-specific early morning symptoms—such as severe epigastric pain, throat discomfort, tongue stiffness, or vomiting—causing them to misattribute the encounter to alternative domestic events or pests.^{2,3,7} As a result, clinicians frequently misdiagnose envenomed patients with acute abdominal colic, appendicitis, food poisoning, stroke, or hysteria.^{3,7} This clinical confusion routinely subjects the patient to unnecessary, time-consuming diagnostic pathways (such as brain CT/MRI scans or lumbar punctures), creating catastrophic delays in specific therapy.^{3,4}

To eliminate these treatment delays and standardize emergency care, the Ministry of Health and Family Welfare (MoHFW), Government of India, established the Standard Treatment Guidelines (STG) for Snakebite Management.^{2,4,9} The STG mandates a strict syndromic approach to diagnosis, dictating that clear, objective clinical indicators of systemic neuromuscular paralysis must instantly override alternative or absent exposure narratives.^{2,4,9}

CASE PRESENTATION

Patient Presentation and Triage

A 44-year-old male was brought by his wife by auto-rickshaw at around 7.00 A.M. to the emergency department with an alleged history of an unknown incident leading to swelling of his left cheek while he was sleeping on the floor at night, followed by drowsiness, throat pain, tongue stiffness and an episode of vomiting. At triage, his airway was patent and protected, RR- 25 breaths per minute, SPO₂ – 100% on room air, HR- 88 bpm, regular, BP- 130/90 mm Hg, Random needle stick glucose was 98 mg/dl.

Patient was assigned to yellow zone bed in emergency ward, shifted with nurse and resident doctor. On further examination by emergency medicine resident doctor a localized swelling on his left cheek was noted which was warm to touch and no local bite mark visible.

Acute Neuroparalytic Crisis and Intervention

A few hours into his observation, the patient suffered rapid, profound neuromuscular deterioration. He acutely manifested severe bilateral ptosis, absolute dysphagia, dysarthria, and escalating respiratory distress. His respiratory rate rose to 30 breaths per minute with SPO₂ dropped to 65 %, while his single breath count dropped to 18. Suspecting impending respiratory failure, emergency endotracheal intubation was performed, and mechanical ventilation was initiated and patient was reverse triaged to emergency ICU/ Red zone.

Targeted Management and Clinical Recovery

Faced with rapid descending flaccid paralysis involving respiratory muscles, a syndromic diagnosis of a neurotoxic snakebite/ occult snake bite was established.

Intact blood coagulation was confirmed via a normal 20-minute whole blood clotting time (20 WBCT). Specific resuscitation protocol consisting of initial loading dose of 10 vials of polyvalent anti-snake venom (ASV) and a structured Atropine-Neostigmine anticholinesterase schedule consisting of three doses spaced at 30-minute intervals was employed along with ventilatory support. Despite initial resuscitation, progressive muscle weakness ensued bringing muscle power down to 1/5 in all four extremities, considering this, a second dose of 10 vials of ASV was successfully infused. To target the presynaptic neural pathway, intravenous Calcium Gluconate was added, and the patient was safely maintained on mechanical ventilation overnight without paralytic agents to preserve accurate neurological tracking.

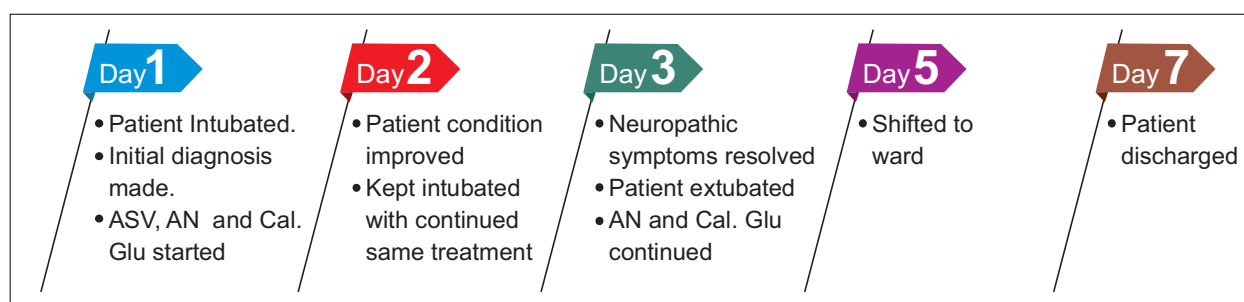


Figure 1: Chain of events



Figure 2: Figures comparing ptosis of the patient immediately after Extubation and during follow up.

The patient's clinical course reversed rapidly over the subsequent days.

By Day 2, his bilateral ptosis began resolving, and his peripheral motor power increased to 3/5 across all limbs while continuing maintenance Atropine-Neostigmine and Calcium Gluconate protocols.

On Day 3, his voluntary muscle power returned to a normal 5/5, and his spontaneous respiratory drive improved sufficiently to permit a successful extubation protocol. A post-extubation neurological assessment confirmed a fully intact swallow reflex and complete resolution of all bulbar and neuropathic signs. The patient was transferred to the general medical ward on Day 5 and was discharged home on Day 7 in excellent, fully stable condition with no residual neurological deficits.

DISCUSSION

The clinical trajectory of this patient presents a deceptive manifestation of a neurotoxic elapid envenomation complicated by atypical localized signs. While the soft tissue swelling on the left cheek initially suggested a localized inflammatory or minor traumatic process, the subsequent development of an acute neuromuscular syndrome shifted the diagnostic focus. The progression from isolated facial edema and progressive drowsiness to absolute bulbar palsy and respiratory collapse matches the classic descending flaccid paralysis characteristic of neurotoxic elapid venoms.^{2,4,8}

The absolute absence of generalized coagulopathy, verified by a normal bedside 20-minute whole blood clotting time (20WBCT), successfully ruled out a primary vasculotoxic envenomation from vipers such as *Daboia russelii* or *Echis carinatus*.^{4,9} Instead, the explosive clinical deterioration focused suspicion directly on a cryptic neurotoxic snakebite, highly indicative of the Common Krait (*Bungarus caeruleus*).^{2,8} Envenomation by *Bungarus caeruleus* is notorious for inducing an "occult" or unwitnessed clinical picture.^{2,3} Because Kraits possess fine, short fangs and hunt nocturnally, their bites are characteristically painless and frequently lack visible puncture marks.^{2,5} This frequently leads victims who are sleeping on floor beds to remain completely unaware of the

encounter, or to misattribute early neurological symptoms to alternative domestic events.^{3,7}

To secure a proper diagnosis, a broad range of neuromuscular conditions had to be systematically evaluated against this patient's presentation:

- **Tetanus Envenoming:** Formally excluded because tetanus toxins block inhibitory spinal interneurons, manifesting exclusively as severe hypertonia, trismus ("lockjaw"), and spastic paralysis. This patient presented with a completely opposite profile of generalized hypotonia and flaccid paralysis.⁹
- **Atypical Rabies:** Ruled out because rabies requires a minimum incubation period of weeks to months. The development of systemic bulbar symptoms within a few hours of an overnight encounter is biologically incompatible with rabies transmission.⁹
- **Myasthenic Crisis:** Considered given the acute onset of ptosis and respiratory failure, but excluded as a primary diagnosis due to the hyper-acute onset in a 44-year-old male with no prior medical history of chronic muscle fatigue or fluctuating ocular weakness.⁹
- **Lambert-Eaton Myasthenic Syndrome (LEMS):** Disregarded because LEMS is a chronic autoimmune condition targeting presynaptic calcium channels where muscle strength characteristically improves with repeated exercise, rather than presenting as a hyper-acute, life-threatening respiratory emergency.⁹
- **Foodborne Botulism:** Ruled out as botulism typically tracks back to the systemic ingestion of contaminated, improperly canned foods or progressive wound colonization over days, rather than an explosive nocturnal paralysis.⁹

The presentation of localized facial swelling directly over the cheek remains an uncommon but recognized anomaly in neurotoxic envenomations. While Krait bites typically feature minimal to no localized reactions, direct envenomation into the highly vascular facial structures can cause atypical soft tissue edema.^{2,5} From a toxicological standpoint, Krait venom is exceptionally rich in presynaptic beta-bungarotoxins.^{2,5} These presynaptic toxins possess phospholipase

activity that structurally alters the motor nerve terminals, permanently halting the release of acetylcholine vesicles into the synaptic cleft.^{5,8} This causes a persistent neuromuscular block that cannot be easily reversed by antivenom alone once structural binding has occurred, necessitating mechanical ventilatory support while the body regenerates new motor endplates.^{1,2,8}

The management strategy utilized in the Emergency Intensive Care Unit (EICU) aligns closely with the Standard Treatment Guidelines instituted by the Ministry of Health and Family Welfare, Government of India.^{4,9} Immediate endotracheal intubation took priority once respiratory compromise was established. Polyvalent anti-snake venom (ASV) remains the cornerstone of definitive therapy, working by binding to and neutralizing unbound, circulating venom molecules.^{4,9} In accordance with the national guidelines, a cumulative limit of 20 vials was infused to maximize neutralization while

reducing the risk of complement-mediated anaphylactic or pyrogenic ASV reactions.^{4,8,9}

The structured administration of anticholinesterase therapy using the Atropine-Neostigmine regimen provided a vital therapeutic bridge by inhibiting acetylcholinesterase, thereby elevating native acetylcholine concentrations within the synaptic cleft to overcome any concurrent postsynaptic blockade. Because the patient's presynaptic blockades do not fully clear with anticholinesterases alone, the integration of intravenous Calcium Gluconate was essential.^{5,10} Calcium gluconate serves as an important adjunct by enhancing the calcium gradient, promoting neurotransmitter release from surviving axon vesicles, and supporting structural recovery.^{5,10} This case reinforces that prioritizing systemic neurosyndromic signs over local anatomical variations prevents catastrophic diagnostic delays and ensures full neurological restoration.^{2,4,9}

Table 1: Clinical Differentials for Neuroparalytic Conditions

Differential Condition	Core Pathophysiology	Clinical Discrepancy / Outcome in This Case
Neurotoxic Elapid Envenomation	Presynaptic/ Postsynaptic blockade of neuromuscular transmission.	Confirmed. Classic chronological match of descending flaccid paralysis (ptosis → dysphagia → respiratory failure), lack of local tissue markings, and full response to guideline therapies.
Tetanus Envenoming	Tetanospasmin-mediated blockade of inhibitory spinal cord interneurons.	Excluded. Tetanus manifests exclusively as severe hypertonia, trismus ('lockjaw'), and spastic paralysis. This patient presented with generalized hypotonia and flaccid paralysis.
Atypical Rabies	Neurotropic viral infection tracking via peripheral sensory axoplasm.	Excluded. Rabies requires a minimum incubation period of weeks to months. The rapid development of systemic symptoms within 2–4 hours of a bite is biologically incompatible with rabies.
Myasthenic Crisis	Autoimmune autoantibody down-regulation of postsynaptic nicotinic ACh receptors.	Excluded. While it presents with acute ptosis and respiratory failure, the hyper-acute onset in a 44-year-old male with a complete absence of preceding ocular or skeletal muscle fatigue is atypical.
Lambert-Eaton Syndrome (LEMS)	Autoimmune IgG targeting of presynaptic P/Q-type voltage-gated calcium channels.	Excluded. LEMS is an ultra-chronic paraneoplastic process where muscle strength characteristically improves with repeated exercise. It does not present as a hyper-acute, life-threatening respiratory emergency.
Foodborne Botulism	Systemic absorption of botulinum toxin blocking presynaptic ACh release.	Excluded. Characteristically tracks back to the ingestion of contaminated, improperly canned foods or wound colonization over days rather than an acute nocturnal puncture history.

CONCLUSION

This case emphasizes that atypical or localized clinical signs, such as isolated facial swelling, must not deter emergency physicians from executing a rapid, syndromic approach to neurotoxic snakebites. While envenomation by the Common Krait (*Bungarus caeruleus*) is classically recognized as an occult crisis lacking significant visible local markings,

direct inoculation into highly vascular facial structures can cause unexpected regional tissue reactions.^{2,5} Most importantly, this case highlights a critical diagnostic rule for emergency clinicians in endemic regions: a neurotoxic snakebite must remain at the top of the differential diagnosis for any patient presenting with unexplained neuromuscular paralysis who has a history of sleeping on a

floor bed inside a house, even in the absolute absence of a visible bite mark.^{3,7}

Adherence to the national standards established by the Ministry of Health and Family Welfare, Government of India, remains the cornerstone of reducing snakebite-related mortality.^{4,9} Securing the airway via early endotracheal intubation, delivering timely and optimally dosed polyvalent anti-snake venom up to the recommended 20-vial neurotoxic ceiling, and initiating structured Atropine-Neostigmine anticholinesterase cycles can successfully arrest descending paralysis.^{4,8,9} Furthermore, the integration of adjunctive intravenous Calcium Gluconate provides vital support to the neuromuscular machinery during presynaptic toxic blockades, preventing prolonged mechanical ventilation and accelerating safe extubation.^{5,10} Ultimately, recognizing the clinical picture of a nocturnal, occult envenomation associated with floor-sleeping prevents catastrophic diagnostic delays and ensures full neurological restoration.^{2,3,7}

REFERENCES

1. Mohapatra B, Warrell DA, Suraweera W, Bhatia P, Dhingra N, Jotkar RM, et al. Snakebite Mortality in India: A Nationally Representative Mortality Survey. *PLoS Negl Trop Dis*. 2011;5(4):e1018. Available from: <https://pmc.ncbi.nlm.nih.gov/articles/PMC3075236/> doi:10.1371/journal.pntd.0001018
2. Sinha A, Majumder D, Bhattacharya S, Ram R, Dasgupta U, Ram A. Epidemiological profile of snake bite in South 24 Parganas district of West Bengal with focus on underreporting of snake bite deaths. *Indian J Public Health*. 2014;58(1):17-21. Available from: <https://pubmed.ncbi.nlm.nih.gov/24748352/> doi:10.4103/0019-557x.128158
3. Mazumder A. A Pilot Study on Snake Bite Deaths in the District of Malda, West Bengal, India. *Semantic Scholar*. 2020. Available from: <https://pdfs.semanticscholar.org/144b/2f86c99af7b0b4e232a770b1235e65524a36.pdf>
4. Salim A, Williams HR, Abdel Wahab S, Adeshokan T, Almeida JR, Williams HF, et al. Identifying key factors contributing to treatment costs for snakebite envenoming in private tertiary healthcare settings in Tamil Nadu, India. *PLOS Negl Trop Dis*. 2023;17(11):e0011699. Available from: <https://pmc.ncbi.nlm.nih.gov/articles/PMC10602377/> doi:10.1371/journal.pntd.0011699
5. Sreedevi M, Sreedevi J, Kumar KV, Swamy MN. Clinical Case Control Study of Injection Calcium Gluconate in Krait Snake Bite at Tertiary Care Centre in Tamilnadu. *Rajiv Gandhi J Med Sci*. 2025;15(2):180-183. Available from: <https://www.makhillpublications.co/public/files/published-files/mak-rjms/2025/2-180-183.pdf>
6. Suraweera W, Warrell D, Whitaker R, Menon G, Rodrigues R, Fu SH, et al. Trends in snakebite deaths in India from 2000 to 2019 in a nationally representative mortality study. *eLife*. 2020;9:e54076. Available from: <https://elifesciences.org/articles/54076> doi:10.7554/eLife.54076
7. Kularatne SA. Common krait (*Bungarus caeruleus*) bite in Anuradhapura, Sri Lanka: a prospective clinical study, 1996-98. *Postgrad Med J*. 2002;78(919):276-280. Available from: <https://pmc.ncbi.nlm.nih.gov/articles/PMC1742353/> doi:10.1136/pmj.78.919.276
8. Silva A, Kuruppu S, Othman I, Isbister GK, Hodgson WC. Neuromuscular effects of Common Krait (*Bungarus caeruleus*) envenoming in Sri Lanka. *PLoS Negl Trop Dis*. 2016;10(2):e0004368. Available from: <https://pmc.ncbi.nlm.nih.gov/articles/PMC4734751/> doi:10.1371/journal.pntd.0004368
9. Ministry of Health and Family Welfare, Government of India. Standard Treatment Guidelines: National Snakebite Management Protocol India. New Delhi: MoHFW; 2017. Available from: <https://aiimsrishikesh.edu.in/documents/standard-treatment-guidelines.pdf>
10. Ghorai S, Mandal T, Bhattacharya S. Clinical profile and outcome of patients of common krait bite with features of neurotoxic envenomation, treated with intravenous calcium gluconate infusion in conjunction with AVS, a tertiary care center-based study. *J Indian Med Assoc*. 2019;117(12):14-17. Available from: https://www.researchgate.net/publication/338075794_A_Case_of_Krait_Snake_Bite_Responding_to_Calcium_Gluconate_Therapy