Case Report

# Rhabdomyolysis and acute kidney injury from neurotoxic common krait envenomation: A case report

**Vedavyas Muvva[1], Meroz Pillarisetty[2], Anisha Vattikonda[1]**

*From [1]Student, [2]Assistant Professor, Department of General Medicine, NRI Medical College, Chinakakani, Andhra Pradesh, India*

## ABSTRACT

The common krait is a highly venomous snake endemic to South Asia. It is known primarily for its potent neurotoxicity. Although neurological manifestations such as descending paralysis are well described, rhabdomyolysis and acute kidney injury (AKI) are exceptionally rare. We report a case of rhabdomyolysis and AKI seen in a 36-year-old male with type 2 diabetes who presented 26 h after a confirmed bite by a common krait with dark-colored urine and left-lower-limb weakness. Laboratory investigations revealed marked rhabdomyolysis and myoglobinuria. Progressive renal dysfunction developed despite aggressive hydration, necessitating two sessions of hemodialysis. The patient also developed localized cellulitis requiring surgical fasciotomy. Renal recovery was achieved by day 8, and the patient was discharged with outpatient follow-up. This case broadens the clinical spectrum of common krait envenomation and underscores the need for continued observation after anti-snake venom administration.

**Key words:** Acute kidney injury, Common krait, Dialysis, Rhabdomyolysis, Snakebite envenomation

Snakebite is a significant public health hazard in India and is estimated to cause approximately 58,000 deaths annually [1]. The common krait (*Bungarus caeruleus*), which is widely distributed across the Indian subcontinent, is a highly venomous snake with predominantly neurotoxic effects. It is responsible for a substantial proportion of neurotoxic snake envenomations in South Asia. The venom of the common krait contains phospholipase A2 (including presynaptic β-bungarotoxins), three-finger toxins, κ-neurotoxins, and postsynaptically acting long neurotoxins [2]. These components of the venom collectively contribute to the neurotoxic effects. The neurotoxic manifestations include descending flaccid neuromuscular paralysis, which can progress to respiratory failure [3]. Acute kidney injury (AKI) is a well-recognized complication of snakebite envenomation, with a reported prevalence of up to 32% [1]. However, it is uncommon following common krait envenomation, in which neurotoxicity predominates, rhabdomyolysis is particularly rare.

We report a case of common krait envenomation presenting with delayed neuroparalysis, rhabdomyolysis, and AKI requiring hemodialysis. Although AKI following envenomation by other krait species has been reported, to the best of our knowledge, this is one of the few case reports describing rhabdomyolysis following common krait (*B. caeruleus*) envenomation.

| Access this article online | |
|---|---|
| Received - 07 February 2026<br>Initial Review - 19 February 2026<br>Accepted - 06 April 2026 | Quick Response code |
| **DOI:** *** | |

## CASE REPORT

A 36-year-old male patient with type 2 diabetes mellitus sustained a snakebite to the left foot at approximately 5:50 PM while working in an agricultural field. The snake was captured and presented to a local government teaching hospital, where he received 26 vials of anti-snake venom (ASV), along with neostigmine (1.5 mg) and atropine (0.6 mg). The indication for 26 vials of dosing was not clearly documented in the discharge record of that institution. He was discharged from the government teaching hospital the same day. The government teaching hospital identified the snake as a common krait (*B. caeruleus*). Approximately 18 h post-bite (12:00 PM the following day), the patient developed bilateral ptosis (left greater than right) and diplopia. Over the next several hours, he experienced progressively worsening swelling of the left foot, one episode of non-bilious, non-projectile, non-blood-stained vomiting, and subsequently developed left lower limb weakness along with dark-colored urine.

He presented to our institute's emergency department at 8:00 PM, approximately 26 h after the snakebite. On arrival, he was fully alert and hemodynamically stable. Respiratory examination was normal, and his single-breath count was 25. Neurological examination

**Correspondence to:** Vedavyas Muvva, Department of General Medicine, NRI Medical College, Chinakakani, Andhra Pradesh, India. E-mail: vedavyasmuvva@gmail.com

demonstrated bilateral ptosis. Motor examination revealed decreased strength in the left lower limb. Local examination showed diffuse swelling of the left foot and leg without active bleeding. Cardiovascular, abdominal, and respiratory examinations were unremarkable.

The patient's glycemic parameters were within normal limits. The glycated hemoglobin level was 5.6%, and the random blood glucose level is 120 mg/dL. Initial laboratory testing revealed marked rhabdomyolysis: Creatine kinase 7746 U/L and lactate dehydrogenase 1848 U/L. Urinalysis revealed reddish discoloration with proteinuria, a high red blood cell count (416/HPF), and myoglobinuria, suggesting the coexistence of hematuria and myoglobinuria (Table 1). Renal parameters demonstrated AKI with progressive elevation of creatinine over the first 3 days (Table 2). Coagulation profile, liver function tests, and complete blood count were within normal limits. The 20-min whole blood clotting test was normal. Chest radiograph was unremarkable, and ultrasonography of the left lower limb demonstrated cellulitis involving the middle and lower one-third of the thigh and the entire leg.

The patient received neostigmine (1.5 mg) and atropine (0.6 mg) every 30 min for three doses, with subsequent improvement in ptosis and diplopia. Aggressive intravenous hydration (125 mL/h) was initiated. Due to progressive renal dysfunction with rising serum creatinine and urea levels despite adequate hydration, the patient underwent two sessions of hemodialysis on days 4 and 6 (Table 2) on the recommendation of the nephrology department of our institute. Given the presence of significant limb swelling with concern for evolving compartment syndrome, the surgical team was consulted, and the patient underwent fasciotomy with relieving incisions of the left lower limb.

Renal function progressively improved (creatinine decreased to 2.1 mg/dL by day 8), and neurological deficits resolved completely. The creatine kinase trends are referenced in Table 3. The patient was discharged on day 8 with instructions for outpatient nephrology follow-up.

**Table 1: Complete urine analysis of the patient**

| Parameter | Result | Reference range |
|---|---|---|
| Color | Reddish | Straw |
| Appearance | Slightly turbid | Clear |
| Urobilinogen | Normal | Normal |
| Protein | + | Nil |
| Glucose | ++ | Nil |
| Ketone bodies | Negative | Negative |
| Bilirubin | Negative | Negative |
| pH | 5.5 | 4.6–8 |
| Specific gravity | 1.007 | 1.003–1.035 |
| RBC | 416/HPF | 0–2/HPF |
| WBC | 42/HPF | 0–5/HPF |
| Epithelial cells | 2/HPF | 0–5/HPF |
| Casts | Nil | Occasional Hyaline Casts |
| Bacteria | Absent | 0–1200 |

**RBC: Red blood cell, WBC: White blood cell**

## DISCUSSION

Krait envenomation typically produces early neuroparalysis due to presynaptic blockade at the neuromuscular junction by the β-bungarotoxins. This presynaptic binding causes ptosis, ophthalmoplegia, bulbar weakness, and respiratory paralysis [4]. In the present case, the patient exhibited delayed (biphasic) neurotoxicity despite initial improvement following ASV and anticholinesterase therapy. This may be because, while circulating venom is neutralized by ASV, presynaptic toxins that have already bound to the nerve terminals are not rendered inactive by antivenom. As a result, ongoing neuromuscular dysfunction may occur due to progressive degeneration of affected nerve terminals. Clinical recovery depends on regeneration of these terminals, which explains the delayed progression and subsequent recovery of neurological symptoms [3,4]. Similar delayed neurological deterioration after initial improvement has been reported in a pediatric case of krait envenomation despite ASV therapy [5].

The myotoxic effects of *B. caeruleus* venom are thought to be mediated primarily by phospholipase A2 enzymes, which disrupt muscle cell membranes, leading to myonecrosis. Although neurotoxicity predominates clinically, experimental studies have demonstrated that these toxins can induce skeletal muscle damage [6-8]. In this patient, the markedly elevated creatine kinase levels and myoglobinuria suggest that myotoxic mechanisms contributed significantly to the clinical presentation.

The development of AKI following snakebite involves a combination of mechanisms: prerenal factors

**Table 2: Serial renal function tests of the patient**

| Day | Serum urea (mg/dL) | Serum creatinine (mg/dL) | Serum sodium (mmol/L) | Serum potassium (mmol/L) | Serum chloride (mmol/L) |
|---|---|---|---|---|---|
| 1 (day of presentation) | 113 | 4 | 133 | 4.7 | 104 |
| 2 | 153 | 5.5 | 133 | 4.3 | 107 |
| 3 | 154 | 5.8 | 133 | 4.3 | 107 |
| 4 | 154 | 5.7 | 133 | 4.2 | 107 |
| 5 | 119 | 3.7 | 137 | 4 | 99 |
| 6 | 82 | 3.0 | 136 | 3.5 | 101 |
| 7 | 54 | 2.3 | 134 | 3.8 | 100 |
| 8 | 41 | 2.1 | 133 | 3.8 | 98 |

**Reference ranges: Serum urea: 18–42 mg/dL; Serum creatinine: 0.66–1.25 mg/dL; Sodium: 137–145 mmol/L; Potassium: 3.5–5.1 mmol/L; Chloride: 98–107 mmol/L**

**Table 3: Serial creatine kinase estimation**

| | |
|---|---|
| Day 1 | 7746 U/L |
| Day 2 | 3159 U/L |
| Day 3 | 2922 U/L |
| Day 4 | 205 U/L |
| Day 5 | 156 U/L |

**Reference range: 55–170 U/L**

such as hypovolemia and hemodynamic instability; direct intrinsic renal damage from venom toxins; immune-mediated injury; and systemic effects, including venom-induced consumptive coagulopathy and capillary leak syndrome [9-11]. Interestingly, in the present case, urinalysis demonstrated significant red blood cells, in addition to myoglobin positivity. While pigment nephropathy typically presents with heme-positive urine without significant hematuria, the findings in this case suggest a combination of myoglobinuria and true hematuria. This may reflect additional renal or vascular injury, although the exact mechanism remains unclear.

Equally unusual was the development of significant local swelling and cellulitis. Krait bites are classically described as painless with minimal local tissue reaction [4]. The presence of microorganisms in snakes' oral cavities, which are also transmitted with snake venom, has been well documented in the literature. This is probably the culprit for cellulitis.

This case broadens the clinical spectrum of common krait envenomation and highlights the importance of continuous monitoring despite initial resolution of neurological symptoms. This case is particularly relevant to emergency physicians, who serve as the first point of contact for snakebite victims. Timely recognition of life-threatening complications is essential for optimizing clinical outcomes.

## CONCLUSION

This case highlights an atypical and severe presentation of *B. caeruleus* envenomation characterized by delayed neurotoxicity, significant rhabdomyolysis, and AKI requiring hemodialysis. It underscores that clinical deterioration may occur despite early administration of antivenom, likely due to irreversible binding of presynaptic neurotoxins. This case emphasizes the need for prolonged observation, vigilant monitoring for delayed complications, and a multidisciplinary approach to management. Early recognition and timely intervention for complications such as rhabdomyolysis and AKI are crucial to improving outcomes. Awareness of such atypical manifestations is particularly important for emergency physicians, especially in resource-limited settings where snakebite remains a significant public health concern.

## REFERENCES


1. Meena P, Bhargava V, Gupta P, Panda S, Bhaumik S. The kidney histopathological spectrum of patients with kidney injury following snakebite envenomation in India: Scoping review of five decades. BMC Nephrol 2024;25:112.
2. Oh AF, Tan CH, Ariaranee GC, Quraishi N, Tan NH. Venomics of *Bungarus caeruleus* (Indian krait): Comparable venom profiles, variable immunoreactivities among specimens from Sri Lanka, India and Pakistan. J Proteomics 2017;164:1-18.
3. Gutiérrez JM, Calvete JJ, Habib AG, Harrison RA, Williams DJ, Warrell DA. Snakebite envenoming. Nat Rev Dis Primers 2017;3:17063.
4. Bittenbinder MA, Van Thiel J, Cardoso FC, Casewell NR, Gutiérrez JM, Kool J, *et al*. Tissue damaging toxins in snake venoms: Mechanisms of action, pathophysiology and treatment strategies. Commun Biol 2024;7:358.
5. Sawhney S, Vagha K, Lohiya S, Mishra N, Vagha JD, Varma A. Delayed neurological manifestation in krait bites despite anti-snake venom therapy. Cureus 2022;14:e29849.
6. Al-Mamun MA, Rahman MA, Hasan RH, Rahmann Z, Hoque K. Histopathological alterations induced by common krait *Bungarus caeruleus* venom on hepatic, renal and cardiac tissues of albino mice. Int J Pharm Pharm Sci 2015;7:239-42.
7. Alam MJ, Maruf MM, Iqbal MA, Hasan M, Sohan MS, Shariar MR, *et al*. Evaluation of the properties of *Bungarus caeruleus* venom and checking the efficacy of antivenom used in Bangladesh for its bite treatment. Toxicon X 2023;17:100149.
8. Charoenpitakchai M, Wiwatwarayos K, Jaisupa N, Rusmili MR, Mangmool S, Hodgson WC, *et al*. Non-neurotoxic activity of Malayan krait (*Bungarus candidus*) venom from Thailand. J Venom Anim Toxins Incl Trop Dis 2018;24:9.
9. Zager RA. Rhabdomyolysis and myohemoglobinuric acute renal failure. Kidney Int 1996;49:314-26.
10. Rao PS, Priyamvada PS, Bammigatti C. Snakebite envenomation-associated acute kidney injury: A South-Asian perspective. Trans R Soc Trop Med Hyg 2025;119:780-7.
11. Moon J, Chun B, Cho Y, Park K. Clinical characteristics of snake envenomation-related acute kidney injury in South Korea. Sci Rep 2024;14:23503.


*Funding: Nil; Conflicts of interest: Nil.*

**How to cite this article:** Muvva V, Pillarisetty M, Vattikonda A. Rhabdomyolysis and acute kidney injury from neurotoxic common krait envenomation: A case report. Indian J Case Reports. 2026; April 12 [Epub ahead of print].