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Death to bite: a case report of dead snake envenoming and treatment

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Background: Envenoming by venomous snakes may induce serious pathophysiological manifestations and death in victims if timely treatment is not provided. Such abilities of snakes remain intact even after death and decapitation. In this article, three cases of envenoming caused by dead snakes that required antivenom therapy are presented from Assam, India.

Case description: Two incidents of envenoming by heads from decapitated *Naja kaouthia* were referred to the Demow Rural Community Health Centre, Sivasagar, Assam. Victims were clinically examined, and the necessary antivenom was administered. Another case of envenoming by a dead black krait was referred to the Boko 30-bed Community Health Centre/First Referral Unit, Kamrup, Assam. The victim exhibited neurotoxic symptoms and required ventilatory support. Antivenom was immediately administered, along with doses of neostigmine, glycopyrrolate, and calcium gluconate. The victim recovered and was discharged after 43 h of hospitalization.

Conclusion: These instances of envenoming by dead or decapitated snakes highlight the potential danger posed by venomous snakes even after death, emphasizing the necessity of extreme caution while handling them.

KEYWORDS

case report, dead snake, envenoming, *Naja kaouthia*, krait

1 Introduction

Snakes have inspired extreme fear and fright in humans since primeval times. Ancient stories involving snakebite and resulting death have been an integral part of various mythological books and folktales (1). Envenoming and death resulting from snakebite and the perception created by such stories have fostered an enormous fear of snakebite in humans. Although approximately 80% of snakes in India are non-venomous (2), a lack of knowledge about snake diversity and distribution, combined with this widespread fear, often leads to snakes being killed in rural households, regardless of the danger they

represent. Additionally, there is a general belief that the snake's head should be smashed as it might bite even after death.

Snakebite is a general concern in rural communities of tropical and sub-tropical countries, causing significant physical, mental, and socioeconomic challenges. An estimated 1.2–5.5 million envenomings occur annually, with 125,000 fatalities and 3–4 times as many permanent disabilities (3). India recorded approximately 1.2 million snakebite deaths alone (58,000 per annum) between 2000 and 2019 (4). Based on the verbal autopsies of a community-based survey (Million Death Study) and a systematic literature review, eight states were marked as highest-burden states, which exhibit high prevalence of the Big-Four snake species (*Naja naja*, *Daboia russelii*, *Echis carinatus*, and *Bungarus caeruleus*), considered to be the most medically significant (4, 5). Northeastern India accounted for a total of 37 fatalities as per the Million Death Study (MDS) from 2001 to 2014 (4); however, there is a dearth of actual epidemiological figures due to underreporting of envenoming cases and deaths in this geographical region (6).

Assam, a state in the northeastern part of India, has a diverse geography and demography as well as rich biodiversity. The state officially recorded approximately 11,000 snakebite cases in 2024, along with 36 deaths (7). Most snakebites have been reported during the monsoon season. Venomous snakes of Assam responsible for these reported incidences are cobras, kraits, and pit vipers. Epidemiological data from the Demow Community Health Centre in Assam show a total of 1,011 snakebite cases between 2018 and 2022. Dry bites and bites from non-venomous snakes accounted for 872 cases. Among the 139 venomous bites, snakes belonging to the family of Elapidae, such as the Indian monocled cobra, banded krait, and lesser/greater black krait, inflicted envenoming in 30 patients (25.5%), which required antivenom treatment (8). These snakes are considered to be crucial from a clinical perspective in Assam, as they are responsible for substantial mortality and morbidity in envenomated patients (8). The clinical manifestations caused by monocled cobras (*Naja kaouthia*) have been well reported in the literature, highlighting their severity post-bite (8, 9). Furthermore, envenomings by various species of krait (*Bungarus* spp.) have also been reported to cause pathophysiological abnormalities in victims (8, 10, 11). Moreover, the lesser-known pit vipers from northeastern India, collectively known as green pit vipers due to their high morphological similarity, account for the highest number of envenoming cases in Assam (66% of total venomous bites) (8, 12). Although non-fatal, the envenoming causes severe pain, ecchymosis, tissue necrosis, and prolonged coagulopathy in victims (12, 13). However, no reports of envenoming caused by dead cobras or kraits are available in the literature so far from this region.

A few reports of envenoming from dead or presumed to be dead snakes have been reported previously. Klouber reported that a rattlesnake head can remain active for 20–60 min after decapitation (14). Freeze-dried fangs of venomous snakes have also been reported to cause envenoming after weeks and months, requiring antivenom treatment (15). Moreover, Suchard et al. reported on five envenoming cases involving freshly killed rattlesnakes that required antivenom treatment (16). Fatality due to a dead snake bite was first reported by Wilhite and his team in

2018, in which the victim was envenomated by a freshly severed prairie rattlesnake (*Crotalus viridis*) and developed a bradysystolic cardiac arrest (17). Another report of coagulopathy by a freshly killed copperhead (*Agkistrodon contortrix*) was reported by Emswiler et al., which required eight vials of antivenom for treatment (18). Although reports of envenoming by presumably dead snakes remain scarce in the scientific literature, a number of press articles have reported such instances, which have been recently reviewed by Naik (19). A fatal envenoming by a freshly decapitated Indochinese spitting cobra while preparing a delicacy soup in a restaurant was reported from China (20). Also, an envenoming by a red-bellied black snake (*Pseudechis porphyriacus*), which had been killed for 45 min, was reported from Australia, which required antivenom treatment (21). Another report from India presented the case of two inebriated individuals who ate a dead and half-burnt *Bungarus caeruleus* (22). Nevertheless, there is a dearth of reported incidents of such envenoming for other venomous snakes of India.

Herein, we report the first case of envenoming resulting from bites by two dead monocled cobras and a lesser black krait from Assam, India, with their symptoms and management. The article is intended to add to the limited knowledge repository of envenoming caused by dead venomous snakes. The article also incorporates the therapeutic interventions associated with the envenoming, which would provide a framework for future management of such cases.

2 Case descriptions and therapeutic interventions

2.1 Case 1

A 45-year-old man encountered a black-colored snake eating chickens in his house at 9:30 p.m. in Sivasagar, Assam. He killed the snake by beheading it, and while trying to discard the body, he was bitten by the snake on his right thumb. The bite was followed by severe pain at the bite site, radiating up to his shoulder. He reported to the nearby community hospital (Demow Rural Community Health Centre, Sivasagar, Assam) at 10:30 p.m. He had multiple episodes of vomiting (five to six times) on the way to the hospital, along with unbearable pain, which was radiating up to his shoulders. Upon initial examination, his pulse rate and blood pressure were observed to be 110/min and 130/80 mmHg. The patient was conscious and responsive to verbal commands. Examination of the bite site revealed blackening of the affected area (Figure 1b). The snake was identified to be a monocled cobra (*N. kaouthia*) based on the photograph shown (Figure 1a). The attending physician immediately administered 20 vials of polyvalent antivenom intravenously along with a dose of paracetamol (1 g) infusion for pain relief. Magnesium sulfate (MgSO₄) glycerin dressing was applied to the wound. Antibiotics, trypsin, and paracetamol infusion were prescribed along with pantoprazole and MgSO₄ glycerin dressing. The pain significantly decreased following this treatment. The patient did not develop any symptoms of neurotoxicity. However, the bite resulted in some

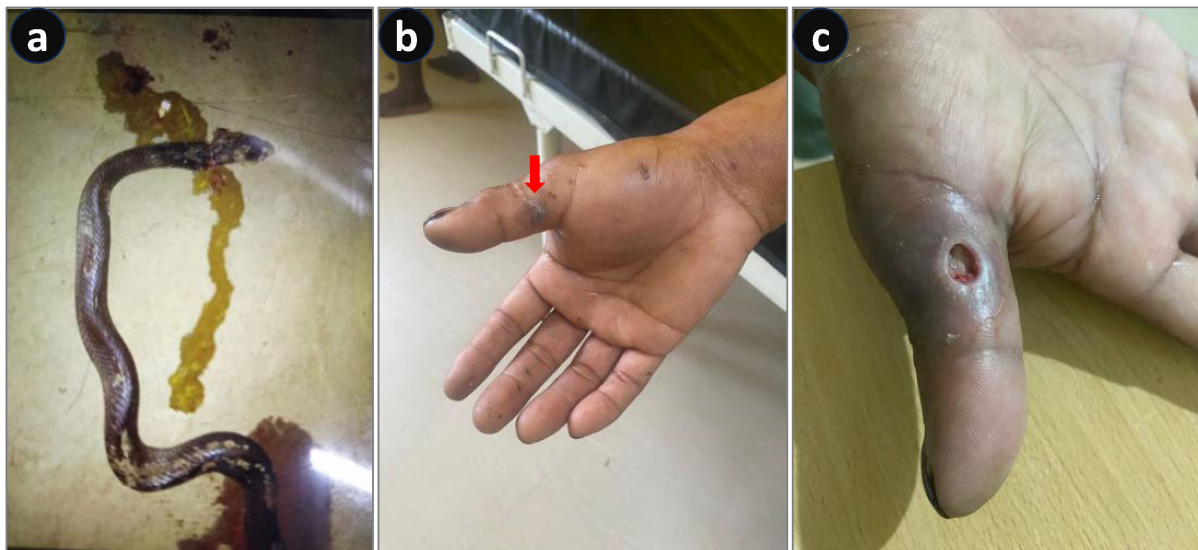


FIGURE 1
Envenoming by a decapitated monocled cobra (*Naja kaouthia*). **(a)** Photograph of a dead snake. **(b)** Blackened bite area. **(c)** A cytotoxic ulcer developed at the bite site.

blebs that were aspirated, followed by sloughing and finally developed in the form of a profound wound. Extensive debridement was performed with hydrogen peroxide and papain-urea ointment, and dressing was continued with Placentrex extract gel over the course of a month (Figure 1c). Upon normalization of all the laboratory parameters on the 20th day, the patient was discharged with a regular follow-up for wound dressing. The wound was completely healed without any residual effect on the 40th day.

2.2 Case 2

A farmer in Sivasagar, Assam, working in a paddy field, unknowingly crushed a snake with the wheels of his tractor. Upon dismounting his vehicle at the end of his work, the farmer was bitten by the snake on his foot at approximately 7:30 a.m. He observed that it was a monocled cobra (locally known as “feti,” Figure 2a), which was presumed to be dead. He started feeling pain at the bite site, which was progressive in nature; hence, he immediately rushed to a nearby hospital (Demow Rural Community Health Centre, Sivasagar, Assam). The patient complained of severe pain, progressive swelling, and noticeable color change at the bite site. He had two episodes of vomiting at approximately 8:30 a.m. in the hospital. Based on the photograph shown by the patient and the vital signs, envenoming was suspected and 20 vials of polyvalent antivenom were immediately administered. The patient was also prescribed antibiotics, trypsin, paracetamol infusion, and pantoprazole, along with MgSO₄ glycerin dressing and limb immobilization. Following the treatment, vomiting gradually stopped and pain was significantly reduced.

Although he did not develop any signs of neurotoxicity, severe cytotoxicity led to an ulcer, which required extensive debridement with hydrogen peroxide and papain-urea ointment and Placentrex extract dressing. The patient was discharged on the 25th day after normalization of all the laboratory parameters. Moreover, he was asked to regularly visit the hospital for wound dressing. The wound was completely healed without any residual effect by the 65th day.

2.3 Case 3

A black-colored snake entered a household at 6:30 p.m. in a village in Kamrup, Assam. The owners killed the snake and discarded the dead body in the backyard of the house. Due to curiosity, one of their neighbors visited the household to see the dead snake at approximately 9:30 p.m. Upon noticing that the snake was dead, he tried to hold its head, but during the process, his little finger of the right hand was envenomed. Since there was no pain or swelling, and considering the snake was already dead, he and his family members initially ignored the bite. However, the victim started to feel uneasiness, sleeplessness, and body pain and gradually became restless by 2:00 a.m. He first sought treatment from a faith healer, but it did not help. He began experiencing difficulty in swallowing and developed drooping eyelids. Finally, at approximately 12:30 p.m. the following day, he reported to a nearby community hospital (Boko 30-bed Community Health Centre/First Referral Unit, Kamrup, Assam). On initial examination, his pulse rate and blood pressure were recorded at 90/min and 140/80 mmHg, respectively. He exhibited ptosis, dysphagia, throat pain, and body aches (Figure 3B). The snake was identified as a black krait (*Bungarus* sp., likely to be either *B. lividus* or *B. niger*)

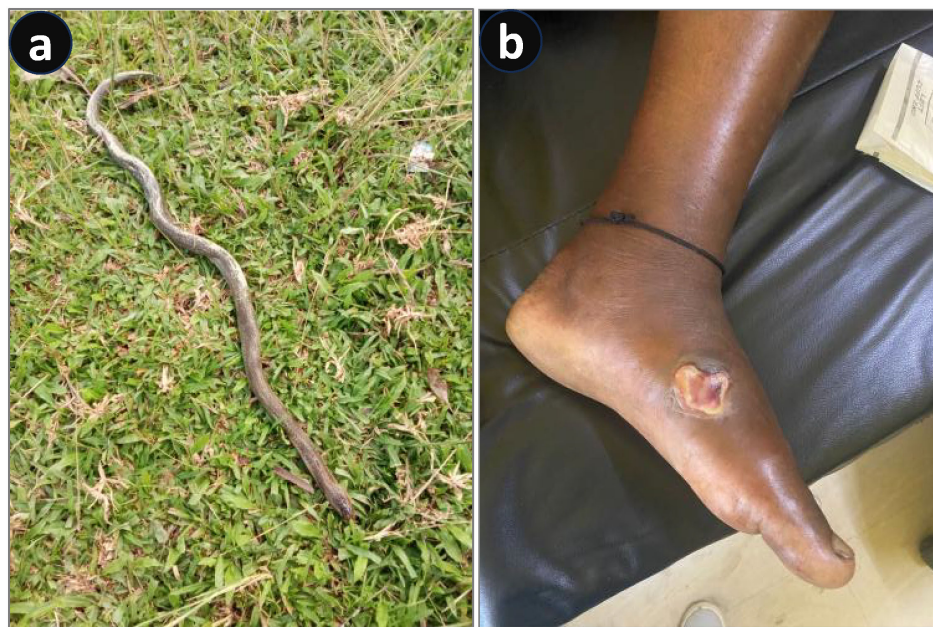


FIGURE 2

Envenoming by a dead monocled cobra (*Naja kaouthia*). (a) Photograph of a dead snake. (b) Bite area showing tissue necrosis.

(Figure 3A), as the dead snake had been brought to the hospital by the family members. Twenty vials of polyvalent antivenom were immediately administered, along with a loading dose of AN [atropine (0.01 mg/kg) and neostigmine glycopyrrolate (0.04 mg/kg)], followed by two maintenance doses of the same. Non-responsiveness to three AN doses confirmed the photographic identification of a krait bite (Standard Treatment Guideline 2017, Ministry of Health and Family Welfare). The patient was infused with calcium gluconate IV (2 mg/kg) every 6 h. However, his condition further deteriorated, and he was moved to the ICU for monitoring. He gradually became quadriplegic and, after 1 h of ASV infusion, became unresponsive to verbal commands. Hypercapnia (PCO_2 was 70 mmHg) developed, and mechanical ventilation of the lungs was immediately initiated. His condition improved over time, and after 43 h of respiratory support, the trachea was extubated, meeting all the vital extubation criteria. However, ptosis and dysphagia persisted for 3 and 5 days, respectively. The patient was later discharged in good health on the 6th day.

3 Discussion

Snakes are ectothermic limbless reptiles and feed on small rodents, frogs, lizards, insects, fish, birds, eggs, etc. (23). They are mostly shy creatures and avoid human interaction while hunting for their prey. However, frequent encounters with farmers do happen during rainy seasons when the digs and burrows are flooded with water (24). An accidental bite from a venomous snake can cause severe complications, requiring emergency medical treatment and sometimes leading to fatalities. Such incidents lead to human–snake

conflict, with the latter being killed whenever spotted. Furthermore, accidental death of snakes is also common due to road accidents, agricultural practices, etc. (25). Despite sustaining fatal injuries, venomous snakes retain the capacity to envenom, posing severe complications.

The monocled cobra (*N. kaouthia*) is the most prevalent cobra species found in northeast India. It has an “O”-shaped mark on its hood, which serves as the identification mark of the snake. It is popularly known as “feti” in the local Assamese language. The monocled cobra is responsible for the majority of envenomings in northeast India and Bangladesh (26, 27). Clinical manifestations of *N. kaouthia* bite include neurotoxicity causing bilateral ptosis, dysphagia, hypotension, and cranial neuropathy, along with cytotoxic symptoms like local blistering, tissue necrosis, ecchymosis, etc. (9). In the present article, envenoming by a monocled cobra presumed to be dead was reported in cases 1 and 2 (Table 1). Case 1 described an instance of envenoming by a beheaded snake while discarding the freshly killed body. The victim began to develop symptoms of envenoming; however, early administration of antivenom prevented neurotoxicity. Nevertheless, cytotoxic wounds developed, which required prolonged care (Figure 1c). The most lethal protein of monocled cobra venom is the non-enzymatic three-finger toxins (3FTXs), which constitute various neurotoxic, cytotoxic, cardiotoxic, and myotoxic components (28). The cytotoxins (CTXs) are known to disrupt the cell membrane, thereby causing tissue necrosis and wounds in victims (28, 29). Similarly, a case of a bite from a dead monocled cobra was described in case 2. Despite being crushed and presumed to be dead for several hours, the snake was capable of delivering a venomous bite, requiring antivenom treatment along with extended wound care (Figure 2b).

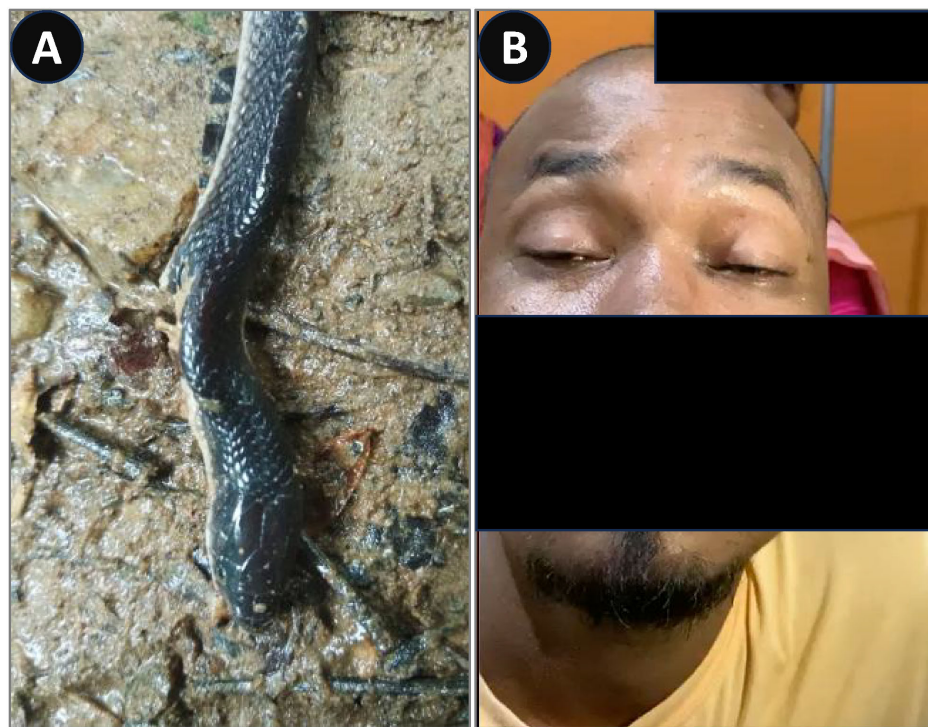


FIGURE 3
Envenoming by a black krait (*Bungarus* sp.). (A) Photograph of the dead snake. (B) Ptosis on the day of hospital admission.

Northeastern India is home to various species of kraits that are medically important; among them, the banded krait (*Bungarus fasciatus*), the lesser black krait (*B. lividus*), and the greater black krait (*B. niger*) are found in Assam. *Bungarus fasciatus* is a morphologically distinct krait species with alternate black and yellow bands on its triangular cross-sectioned body (30). The lesser and greater black kraits are nocturnal snakes characterized by their shiny black coloration and a slender body with a triangular cross-section (31). *Bungarus lividus* shows high morphological similarity with *B. niger*; the only visible difference lies in the relative size of hexagonal vertebral scales on the dorsal surface of the body, which is larger than the bordering scales in *B. niger* compared to *B. lividus* (31, 32). Such resemblance makes them difficult to be identified at the species level, and these species are therefore referred to as “black” krait when spotted. Very few cases of envenoming by greater and lesser black kraits have been reported in

the literature (8, 10, 11, 33, 34). Bites from these snakes are generally characterized by abdominal and chest pain, ptosis, burning sensation, vomiting, slurred speech, and progressive neuromuscular paralysis leading to respiratory distress (33, 34). Although the specific venom composition of the greater or lesser black krait is not known, their venom largely consists of presynaptic neurotoxins belonging to the phospholipase A₂ superfamily, which causes severe neuromuscular paralysis in victims (35). The victims usually remain unaware of the envenoming, as there is usually no pain or insignificant pain during the bite (35). However, patients may experience restlessness, speechlessness, and ptosis followed by respiratory distress (33, 34). In the present report, envenoming by a dead black krait has been presented in case 3 (Table 1). Based on the geographical distribution of kraits, the snake responsible for envenoming could be either the greater or the lesser black krait; however, a close observation of the dead body, especially the

TABLE 1 Summary of cases presenting dead snake envenoming and treatment.

S. no.	Name of snake	Status of biting snake	Time taken to report to the hospital post-bite	Treatment	Duration of hospital stay
1	<i>Naja kaouthia</i>	Freshly decapitated	1 h	20 vials of ASV, one dose of paracetamol	20 days
2	<i>Naja kaouthia</i>	Long crushed by tractor wheels	1 h	20 vials of ASV	25 days
3	<i>Bungarus lividus</i>	Dead for 3 h	14 h	20 vials of ASV, two doses of neostigmine glycopyrrolate and calcium gluconate, ventilation support	6 days

midventral scale of the snake, suggests it to be a lesser black krait (*B. lividus*). The victim was envenomated by a snake that had been dead for 3 h. Symptoms of envenoming occurred approximately 5 h after the bite and were treated with the administration of 20 vials of antivenom and respiratory support (ventilation) for 43 h.

The potential to bite even after death and decapitation lies in the structural organization of the venom apparatus and fangs of front-fanged snakes (families Elapidae, Viperidae, and Atractaspididae) (19). The venom gland consists of a large basal lumen for storage of secreted venom, which is connected to a long hollow fang (36). Venom is rapidly injected into the target, either like a hypodermic syringe or via the groove of the fang by contraction of the compressor muscle (37). An inadvertent envenoming may occur due to accidental pressing of the venom gland of the dead snake while handling the severed head. Additionally, some residual venom, expelled from the venom glands but retained within the lumen of the fangs, might just seep into the pierced tissue and cause envenoming (15). Furthermore, the delivery of venom is controlled by the snake, which may modulate the amount of venom injected based on the size and type of the target (38). They can pierce through the target tissue and inject the entire venom present in the venom sac, or when needed, they might just retrieve the fangs to avoid envenoming (dry bites) (39). This property is completely lost in a dead snake; hence, the entire quantity of venom present in the venom delivery system may just be transferred to the target tissue, leading to severe envenoming (40). Additionally, bites from dead snakes can be attributed to their inherent potential to respond reflexively even after death (14, 41). In the cases highlighted here, such envenomings lead to clinical symptoms comparable to those produced by live snakes, hence requiring antivenom to mitigate the pathophysiological complications. Furthermore, the extent of envenoming might vary depending on the amount of venom present in the sac as well as the structural stability of the venom components. Therefore, vigilant clinical observation is required during antivenom administration and management of the patient.

Previous studies have reported the prolonged stability of snake venom in the absence of air exposure, emphasizing that the pharmacological stability of venom of a dead snake remains virtually similar when compared to live snakes (19, 42). This indicates that venom retained within the venom delivery system remains biologically active for several hours after death and may cause envenoming. In case of pooled venom, factors like air exposure, temperature, and storage condition reduced venom stability over time, thereby reducing its potency (43, 44). However, exactly how long dead and/or decapitated snakes retain the ability to involuntarily deliver a venomous bite remains unknown.

4 Conclusion

Accidental envenoming may occur while handling the corpse or the severed head of a snake. Such envenoming can lead to clinical complications similar to those caused by live snakes requiring antivenom therapy. Such cases of envenoming by a dead

venomous snake or its severed head highlight the critical necessity for utmost care while handling the snake, whether dead or alive. Moreover, the post-mortem envenoming ability of a venomous snake constitutes a critical element of snakebite-related knowledge and should be integrated as a core component into awareness and training programs targeting both the general public and the healthcare providers for effective management of snakebite. Furthermore, research oriented toward understanding the duration and effectiveness of venom activity after snake death would provide a comprehensive understanding of dead snake envenoming and its management.

Data availability statement

The original contributions presented in the study are included in the article/supplementary material. Further inquiries can be directed to the corresponding author.

Ethics statement

The studies involving humans were approved by the Joint Director of Health Services, Sivasagar, Govt. of Assam vide letter no. JT.DHS/SIV/Snake Bite/2025/2614 dated 27/03/2025. The studies were conducted in accordance with the local legislation and institutional requirements. Written informed consent for participation was not required from the participants or the participants' legal guardians/next of kin in accordance with the national legislation and institutional requirements. Written informed consent was obtained from the individual(s) for the publication of any potentially identifiable images or data included in this article.

Author contributions

ST: Writing – original draft, Writing – review & editing, Formal Analysis, Investigation, Methodology. SG: Data curation, Resources, Writing – review & editing. GC: Data curation, Writing – review & editing. HN: Data curation, Writing – review & editing. RD: Conceptualization, Supervision, Validation, Writing – original draft, Writing – review & editing.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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