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Clinical management of common krait (*Bungarus caeruleus*) envenomation in a dog using polyvalent anti-snake venom: A case report

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Abstract

This report details the effective treatment of a 15 kg, one-year-old crossbred canine following a natural envenomation by a Common Krait (*Bungarus caeruleus*). The patient was brought to the clinic roughly one-hour post-bite, presenting with acute distress, localized facial edema, and distinct paired puncture wounds on the right side of the muzzle. A definitive diagnosis of acute elapid envenomation was established via clinical evaluation and the owner's identification of the reptile. Emergency intervention included the immediate administration of intravenous polyvalent anti-snake venom (ASV), alongside supportive fluid resuscitation, atropine, enrofloxacin, meloxicam, tranexamic acid, and furosemide. The canine was monitored continuously for secondary complications, including delayed neurotoxic effects, coagulopathy, and acute kidney injury. The patient demonstrated significant clinical recovery within 24 hours and was discharged with no permanent neurological or local tissue deficits. This case highlights that rapid antivenom deployment paired with aggressive supportive management is critical to securing a favorable prognosis in canine elapid envenomations.

Keywords: Bungarus caeruleus, canine, snakebite, anti-snake venom, neurotoxic envenomation, case report

Introduction

Snakebite envenomation is an important cause of morbidity and mortality in both humans and domestic animals throughout tropical and subtropical regions. In India, companion animals are frequently exposed to venomous snakes because of their outdoor activity and close proximity to agricultural and peri-urban environments (Warrell, 2010; World Health Organization [WHO], 2016) [12, 14]. Dogs are particularly susceptible owing to their inquisitive behaviour, with most bites involving the muzzle, lips or forelimbs while investigating or attacking snakes (Peterson, 2006).

The Common Krait (*Bungarus caeruleus*), one of India's "Big Four" venomous snakes, possesses highly potent presynaptic and postsynaptic neurotoxins that interfere with acetylcholine release at the neuromuscular junction, leading to progressive flaccid paralysis and respiratory failure if untreated (Whitaker & Captain, 2004; Warrell, 2010) [12, 13]. Although local tissue damage following krait bites is generally less severe than that caused by viperid snakes, systemic manifestations may develop rapidly and require immediate veterinary intervention. In addition to neurotoxicity, venom-induced endothelial injury, hypotension, pigment nephropathy and inflammatory responses may contribute to secondary complications, including acute kidney injury (AKI) and multiple organ dysfunction (WHO, 2016; Segev *et al.,* 2004) [10].

Polyvalent anti-snake venom (ASV) remains the only specific treatment capable of neutralizing circulating venom toxins. Nevertheless, successful management depends equally on early supportive care, including maintenance of cardiovascular stability, preservation of renal perfusion, control of inflammation, monitoring for respiratory compromise and prevention of secondary complications (Merck Veterinary Manual, 2024; Peterson, 2006). This report describes the successful clinical management of naturally occurring Common Krait envenomation in a young crossbred dog and highlights the importance of early antivenom therapy and intensive supportive care in preventing life-threatening complications.

Case History

A 12-month-old intact crossbred dog weighing approximately 15 kg was presented to the Veterinary Clinical Complex with a history of snakebite approximately one hour before admission. According to the owner, the dog attempted to attack a Common Krait (*Bungarus caeruleus*) while roaming near agricultural fields. The snake was recognized by its characteristic glossy black body with narrow white cross-bands before escaping. Immediately following the bite, the dog vocalized in pain and developed rapidly progressive swelling over the right side of the muzzle.

No first-aid measures, tourniquets or traditional medications had been applied before presentation. During transportation, the dog became dull and reluctant to move but remained conscious and ambulatory.

Clinical Examination

On presentation, the dog was depressed but responsive. Physical examination revealed marked diffuse oedematous

swelling involving the right upper lip and muzzle. Two distinct fang puncture wounds with mild haemorrhagic oozing were observed on the lateral aspect of the right muzzle (Figure 1). The affected region was warm, painful and firm on palpation.

Physiological examination revealed mild tachycardia, while rectal temperature, respiratory rate and mucous membrane colour were within normal limits. Capillary refill time was approximately two seconds. No ptosis, dysphagia, generalized muscle weakness or respiratory distress was evident at admission, suggesting that systemic neurotoxicity had not yet progressed. Nevertheless, considering the known neurotoxic nature of Common Krait venom, continuous monitoring for respiratory muscle paralysis and cranial nerve dysfunction was instituted. The principal differential diagnoses included acute allergic angioedema, insect sting hypersensitivity, penetrating facial trauma and bacterial cellulitis. However, the confirmed history of Common Krait bite, characteristic paired fang puncture wounds and rapidly developing facial oedema (Figure 2) strongly supported the diagnosis of acute neurotoxic snake envenomation.

Fig 1a & 1b: Paired fang puncture wounds and diffuse oedematous swelling over the right muzzle of the dog following envenomation

Emergency treatment was initiated immediately to neutralize circulating venom and prevent progression to systemic neurotoxicity.

Diagnostic Evaluation

Because the owner witnessed the puppy being bitten by a Common Krait (*Bungarus caeruleus*) and two distinct fang punctures with rapidly progressive muzzle swelling were evident on clinical examination, the case was immediately managed as an acute neurotoxic envenomation. Although neurological deficits were absent at presentation, early intervention was considered critical because krait venom contains potent presynaptic neurotoxins, particularly β-bungarotoxins, which irreversibly damage motor nerve terminals at the neuromuscular junction. Unlike postsynaptic neurotoxins that produce immediate paralysis, presynaptic toxins cause progressive degeneration of nerve endings, resulting in a latent period during which affected animals may appear clinically normal before developing rapidly progressive flaccid paralysis, respiratory muscle weakness, and potentially fatal respiratory failure. Therefore, treatment was initiated without awaiting the onset of neurological signs to prevent irreversible neurotoxicity and improve the likelihood of survival (Warrell, 2010; White, 2013) [12]. Routine haematological and biochemical evaluation was recommended to assess systemic involvement and establish baseline renal and hepatic function. Continuous monitoring of respiratory rate, heart rate, capillary refill time, urine output and neurological status was undertaken throughout hospitalization to detect early deterioration. Particular attention was directed towards signs of respiratory muscle weakness, cranial nerve dysfunction, coagulopathy and venom-associated acute kidney injury (AKI).

Therapeutic Management

Considering the potentially fatal nature of krait envenomation, treatment was instituted immediately without awaiting the development of systemic neurological manifestations. Polyvalent anti-snake venom (ASV) (Figure 2A) was administered intravenously following dilution in normal saline and infused slowly under close observation for possible hypersensitivity reactions. Early administration of ASV remains the only specific therapy capable of neutralizing circulating venom toxins before irreversible binding at the neuromuscular junction (WHO, 2016). Intravenous isotonic crystalloid fluid therapy was initiated to maintain circulatory volume, improve tissue perfusion and preserve renal blood flow. Adequate hydration was considered essential for minimizing venom-associated nephrotoxicity and preventing pigment-induced renal injury secondary to haemolysis or myotoxicity. Supportive medications included atropine sulphate to counter excessive cholinergic manifestations where indicated, meloxicam for analgesia and control of inflammation, enrofloxacin to reduce the risk of secondary bacterial infection associated with puncture wounds, tranexamic acid to support haemostasis in the event of venom-induced bleeding tendencies and furosemide (Figure 2A) based on clinical assessment of facial oedema and urine output. The patient was maintained under intensive clinical observation with serial assessment of respiratory effort, neurological function and cardiovascular stability (Figure 2B). Renal support formed an integral component of case management.

Intravenous fluid therapy was continued to maintain adequate urine production, while renal function was monitored throughout hospitalization. Oral nephroprotective supplementation containing antioxidants and renal supportive nutrients was instituted during recovery to facilitate renal rehabilitation and minimize delayed renal injury. Although evidence regarding specific renal supplements following snakebite is limited, maintenance of renal perfusion and early recognition of AKI remain fundamental principles of venom management (Segev *et al.,* 2004; Merck Veterinary Manual, 2024) [10].

Fig 2a: Polyvalent anti-snake venom (ASV) Supportive medications & 2B Clinical monitoring and intravenous administration of anti-snake venom during hospitalization

Clinical Outcome and Follow-up

Marked clinical improvement was evident within 24 hours of treatment. Facial oedema decreased substantially, and the patient became brighter, more responsive and resumed voluntary feed and water intake. No evidence of ptosis, dysphagia, generalized weakness or respiratory compromise developed during hospitalization (Figure 3).

Follow-up examination one week later revealed complete resolution of facial swelling with no neurological deficits, respiratory abnormalities or delayed complications.

Fig 3: Complete clinical recovery at follow-up with resolution of local lesions

Discussion

Snake envenomation remains one of the most important emergency conditions encountered in veterinary practice, particularly in tropical countries where venomous snakes are widely distributed. Dogs commonly sustain bites on the muzzle because of their instinctive tendency to investigate moving objects. Early veterinary intervention is one of the most significant factors influencing prognosis.

The present case was unusual in that the offending snake was identified by the owner as a Common Krait (*Bungarus caeruleus*), allowing anticipation of predominantly neurotoxic effects. Krait venom contains β-bungarotoxins that irreversibly impair acetylcholine release at the neuromuscular junction, resulting in progressive paralysis and respiratory failure if untreated (Warrell, 2010) [12]. Interestingly, despite the highly neurotoxic nature of the venom, this patient primarily exhibited localized facial oedema without clinically significant neurological dysfunction. The rapid presentation to the hospital and immediate administration of ASV likely prevented progression to systemic neurotoxicity.

Prompt administration of polyvalent anti-snake venom remains the cornerstone of treatment because it neutralizes circulating venom before irreversible tissue binding occurs. Delayed administration has been associated with increased mortality, prolonged hospitalization and greater risk of neurological complications (WHO, 2016). In the present case, ASV was administered within approximately one hour after the bite, which probably contributed to the favourable outcome.

Supportive therapy plays an equally important role in snakebite management. Maintenance of adequate intravascular volume through intravenous crystalloids

supports tissue perfusion and renal function while reducing the likelihood of venom-associated acute kidney injury. Although AKI is more frequently associated with viper envenomation, haemodynamic disturbances, haemolysis and direct nephrotoxicity may also contribute to renal injury following elapid bites. Consequently, monitoring of urine output, serum creatinine and blood urea nitrogen is recommended in all clinically significant envenomation cases. The absence of delayed neurological deficits, respiratory failure, tissue necrosis and renal dysfunction in the present case underscores the importance of early diagnosis, prompt ASV administration and comprehensive supportive management. The favourable outcome observed is consistent with previous veterinary reports demonstrating significantly improved survival when antivenom is administered during the early phase of envenomation (Segev *et al.,* 2004) [10].

Conclusion

Acute Common Krait (*Bungarus caeruleus*) envenomation should be regarded as a medical emergency because of its potential to cause rapidly progressive neurotoxicity and respiratory failure. Early diagnosis based on history and characteristic fang puncture wounds, followed by immediate administration of polyvalent anti-snake venom and intensive supportive care, resulted in complete clinical recovery in the present case. Preservation of renal perfusion, vigilant monitoring for delayed complications and comprehensive nursing care were important components of successful management. This report reinforces the value of early therapeutic intervention in improving the prognosis of canine snake envenomation.

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