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Case Study

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Acute Kidney Injury Following Viper Envenomation in One-Year-Old Doberman pinscher: Clinical Features and Management: A Case Report

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A B S T R A C T

Snake envenomation is a frequent veterinary emergency in tropical countries, with viper bites commonly resulting in hemotoxic effects, coagulopathies, and local tissue injury. Secondary organ dysfunction in snakebite cases results from the systemic action of venom and the cytotoxic effects of its metabolites. A one-year-old male Doberman Pinscher was presented with a history of snake bite, confirmed by the owner who presented the dead snake. The dog exhibited visible fang marks on the lateral facial region, accompanied by localized swelling and mild hemorrhage. Initial laboratory evaluation revealed a prolonged whole blood clotting time (Tube method, 25 minutes), elevated prothrombin time (PT) and activated partial thromboplastin time (aPTT), thrombocytopenia, mild anemia, and markedly elevated hepatic enzymes (AST and ALT), while serum creatinine remained within normal limits on day 0. The dog was managed as a viper envenomation emergency with prompt administration of anti-snake venom and intensive supportive therapy. By day 2, hematological parameters including platelet count and anemia showed slight improvement, and hepatic enzyme levels demonstrated a mild downward trend. However, despite stabilization of coagulation parameters, serum creatinine rose sharply to 4.2 mg/dL, indicating the development of acute kidney injury. The case highlights the delayed onset of renal involvement following viper envenomation. Creatinine and BUN returned to normal level by day 10. This case emphasizes that viper snake bite in dogs can result in delayed acute kidney injury despite initial stabilization of clinical and hematological parameters. Continuous monitoring of renal function is essential in the post-envenomation period, even when early biochemical values appear normal.

Introduction

Snake bites are a common and life-threatening emergency, often leading to significant morbidity and mortality. The venom of snakes consists of various toxins that differ depending on the species. Around 375 venomous snake species globally are known to be dangerous (Rolan, 2015). In India, 52 species are identified as poisonous (Turkar *et al.,* 2017). The majority of bites and consequent mortality is mainly caused by Indian cobra (Naja naja), Common Krait (Bungarus caeruleus), Russell's viper (Daboia russelii), and saw-scaled viper (Echis carinatus) (Meenatchisundaram and Michael, 2009).

Snake bite in animals generally occurs during grazing, hunting, or while playing in the garden. Snake bite cases are more common in horses and dogs when compared to other animals such as cattle, sheep, and goats. Snake envenomation in dogs represents a critical emergency in veterinary practice, particularly in regions where venomous vipers are endemic and animal–human contact is frequent (Martinez *et al.,* 2020).

Dogs are commonly presented with extensive oedematous swelling, severe pain, ecchymosis, and discoloration of the skin at the affected site within several hours after the bite. The initial local reactions, such as fang marks, swelling, and mild bleeding, frequently progress within hours and may be accompanied by lethargy and signs of systemic involvement (Saravanan *et al.,* 2017).

Viperid venom is predominantly hemotoxic, composed of proteolytic enzymes and procoagulant toxins that disrupt normal haemostatic mechanisms, often leading to coagulation abnormalities and systemic hemorrhagic tendencies in envenomated patients (Martinez *et al.,* 2020). Clinical manifestations in dogs following viper envenomation are variable and may range from localized swelling at the bite site to severe systemic effects, including coagulopathy, thrombocytopenia, and anemia (Sadhu *et al.,* 2024).

Laboratory abnormalities in canine viper bite patients commonly include prolonged clotting times, such as elevated PT and aPTT, reduced platelet counts, and evidence of venom-induced consumption coagulopathy (Meenatchisundaram and Michael, 2009). These hematological changes reflect the venom's capacity to consume clotting factors and impair normal coagulation pathways (Martinez *et al.,* 2020). Hepatocellular injury, evidenced by elevated enzymes such as AST and ALT, is also a frequently observed effect of systemic venom absorption and tissue damage (Sadhu *et al.,* 2024). Despite these systemic effects, initial renal parameters may be within normal limits at presentation, underscoring the potential for delayed onset of more serious complications (Martinez *et al.,* 2020).

Acute kidney injury (AKI) is a recognized and potentially severe sequela of viper envenomation in dogs and humans, occurring in a significant proportion of cases within 48 hours or more after the bite (Sai *et al.,* 2008). The pathogenesis of AKI following envenomation is multifactorial, including hemodynamic instability due to hypovolemia and shock, direct nephrotoxic effects of venom constituents, and microvascular damage from consumptive coagulopathy (Meenatchisundaram and Michael, 2009). Retrospective data indicate that approximately 29% of dogs with confirmed pit viper envenomation develop AKI, and its development is associated with increased antivenom requirements, higher shock indices, and poorer outcomes (Sai *et al.,* 2008).

These findings highlight the importance of vigilant monitoring of renal function in the post-bite period, even when initial kidney values are normal.

Prompt antivenom therapy remains the cornerstone of specific treatment for viper envenomation, aimed at neutralizing circulating venom components and halting the progression of systemic toxicity (Martinez *et al.,* 2020; Vijayakumar *et al.,* 2019). Supportive therapies, including isotonic fluid administration, correction of coagulopathy, and management of secondary complications, are essential adjuncts to improve clinical outcomes (Martinez *et al.,* 2020; Sadhu *et al.,* 2024; Ravi *et al.,* 2025).

Anti-snake venom is known to potentially cause anaphylactic reactions (Vijayakumar *et al.,* 2019); hence, antihistamines are administered to counteract the adverse effects of histamine, as suggested by Ananda *et al.,* (2009). Despite available treatments, standardized protocols for monitoring and managing complications such as AKI in canine snakebite cases are still evolving, and further research is needed to refine therapeutic strategies and prognostic indicators.

Early identification of the snake bite and timely initiation of anti-snake venom can prevent death, localized cell

necrosis, and associated complications in affected patients.

Case report

A One-year-old Doberman Pinscher was presented with a history of snake bite (45 minutes prior to presentation), as confirmed by the pet owner, who brought the dead snake along with the dog (Fig.1). Based on the visible morphological characteristics; including a stout, heavy body; strongly keeled dorsal scales; brownish coloration with distinct dark oval blotches arranged in longitudinal rows; and a broad, triangular head clearly demarcated from the neck; the snake was identified as Russell's viper (*Daboia russelii*) by a wildlife veterinarian. Clinical examination revealed distinct fang marks on the lateral facial region with localized swelling (Fig.2) and mild bleeding from the puncture wounds. The dog was dull and lethargic at presentation, prompting immediate evaluation and emergency intervention considering suspected viper envenomation. The pet owners were advised about the serious and potentially fatal complications of snake envenomation, including multiple organ dysfunction and circulatory failure.

Initial laboratory investigations on day 0 demonstrated leucocytosis, significant coagulation abnormalities, including a prolonged whole blood clotting time (Tube method, 26 minutes), elevated prothrombin time and activated partial thromboplastin time, thrombocytopenia, and mild anemia. Serum biochemical analysis revealed markedly elevated hepatic enzymes, while renal parameters, including serum creatinine, were within normal limits. A diagnosis of viper snake envenomation was made based on history, clinical signs, and laboratory findings. Serial monitoring was performed to assess progression and response to therapy. On day 2, hematological parameters showed gradual improvement, with increasing platelet counts and improved hematocrit. Hepatic enzyme levels exhibited a mild decline. However, despite stabilization of coagulation parameters, serum creatinine levels increased markedly, indicating the onset of acute kidney injury. Mild hyperkalemia was observed, attributable to decreased renal excretion of potassium consequent to acute kidney injury (AKI). Continued monitoring up to day 10 demonstrated further clinical and laboratory changes as summarized below.

Therapeutic management

Based on the clinical presentation and laboratory findings consistent with hemotoxic viper envenomation, immediate specific and supportive therapy was initiated. Polyvalent anti-snake venom (ASV) was administered at a dose of 2 vials, each reconstituted as per manufacturer's instructions and diluted in 500 mL of isotonic normal saline, and infused intravenously over 60 minutes under close monitoring for adverse reactions. The ASV dose was selected based on the presence of significant systemic envenomation, evidenced by prolonged whole blood clotting time, markedly elevated PT and aPTT, thrombocytopenia, and progressive local swelling, rather than on body weight alone, as recommended in standard snakebite management protocols.

To reduce the risk of acute hypersensitivity reactions associated with ASV administration, dexamethasone was administered intravenously at 1 mg/kg prior to ASV infusion. Although routine corticosteroid premedication remains controversial, its use in this case was justified due to the anticipated risk of anaphylactoid reactions and the absence of contraindications, with careful monitoring to avoid masking early signs of adverse responses.

Atropine sulphate (0.06 mg/kg, intramuscularly) was administered to counteract potential vagal-mediated bradycardia and excessive salivation that may occur secondary to venom effects or stress during emergency management, although it is not routinely indicated in all snakebite cases. Aggressive intravenous fluid therapy was instituted to maintain renal perfusion and prevent venom-induced nephrotoxicity. Isotonic crystalloids (normal saline) were administered initially at maintenance to slightly increased rates (4–6 mL/kg/hour), with adjustments made based on hydration status and urine output. Following the detection of acute kidney injury on day 2, fluid therapy was carefully titrated to avoid volume overload while ensuring adequate renal perfusion.

Urine output was closely monitored throughout hospitalization as an essential indicator of renal function, alongside serial measurements of serum creatinine, blood urea nitrogen, and electrolyte levels. This allowed early detection and ongoing assessment of renal involvement and therapeutic response. Broad-spectrum antimicrobial therapy was initiated to prevent secondary bacterial infection at the bite site. Ceftriaxone was administered intravenously at 25 mg/kg twice daily for three days, considering the high likelihood of bacterial contamination associated with snake fangs and local tissue damage.

Table.1 Sequential hematological and biochemical changes observed during hospitalization

Parameter	Reference Range	Day 0	Day 2	Day 10
Total Leucocyte Count (× 10^9/L)	5.0–14.1	17.8	22.6	12.9
Hemoglobin (g/dL)	11.9-19.9	10.8	9.7	10.1
Hematocrit	35-57	31.8	28.4	30.4
Platelet count (×10^3/µL)	211-621	85	160	310
ALT (U/L)	10-109	124	242	65
AST (U/L)	13-15	92	260	84
ALP (U/L)	1-114	185	165	120
Serum creatinine (mg/dL)	0.5-1.7	1.1	4.2	2.0
Blood urea nitrogen (mg/dL)	8-28	18	62	32
Prothrombin time – PT (sec)	6 – 9	24	12	8
Activated partial thromboplastin time – aPTT (sec)	10 – 15	48	22	14
Potassium (mmol/L)	3.9-5.1	4.3	5.4	4.7

(Reference ranges adapted from MSD Veterinary Manual—Hematology and Serum biochemistry Reference Ranges for dogs.)

Fig.1 The dead snake

Fig.2 The fang marks and swollen bite region

Supportive therapy also included gastroprotective agents (Pantoprazole at 1mg/kg intravenous route) to prevent stress-related gastric mucosal injury and uremic gastritis associated with systemic illness and renal dysfunction. The dog was maintained under continuous clinical observation with serial monitoring of hematological, coagulation, and biochemical parameters. Progressive improvement in clinical status and laboratory values was observed, and the animal made a successful recovery from acute kidney injury with appropriate supportive care.

Result and Discussion

Thrombocytopenia observed in the present case may be attributed to venom-induced vasculitis, sequestration of platelets within inflamed tissues, and increased platelet consumption associated with the potential development of disseminated intravascular coagulation (DIC) (Segev *et al.,* 2004). Local swelling and tissue necrosis at the bite site were primarily due to the action of proteolytic enzymes such as collagenase, phospholipase A_2, and 5′-nucleotidase present in viper venom. The bleeding observed from the wound further indicated venom-mediated interference with multiple components of the haemostatic system (Wolff, 2006).

Lyophilized polyvalent anti-snake venom has been reported to occasionally induce anaphylactic reactions (Sai *et al.,* 2008). To mitigate this potential adverse

reaction, dexamethasone was administered prophylactically in the present case. According to Klaassen (2008), hyaluronidase cleaves internal glycosidic bonds in acid mucopolysaccharides, resulting in reduced viscosity of connective tissues and facilitating the diffusion of other venom components. The cyanotic oedema observed at the bite site may therefore be attributed to the action of hyaluronidase, which functions as a venom spreading factor.

Viper venom is predominantly haemotoxic in nature and is known to cause cardiopulmonary dysfunction, coagulation abnormalities, local tissue swelling, oedema, necrosis, and, in severe cases, gangrene formation. Broad-spectrum antibiotics were administered, as snake fangs are frequently contaminated with diverse bacterial flora, predominantly gram-negative Enterobacteriaceae, increasing the risk of secondary bacterial infection at the bite site (Blaylock, 2001).

Marked elevations in hepatic enzymes, including aspartate aminotransferase (AST) and alanine aminotransferase (ALT), observed in the present case suggest hepatocellular injury secondary to systemic venom absorption, hypoxic insult, or inflammatory processes. Similar hepatic involvement has been documented in previous veterinary case reports of snake envenomation (Sadhu *et al.,* 2024).

In conclusion, Viper snake envenomation in dogs represents a life-threatening medical emergency with the potential for severe systemic complications. The present case illustrates that the haemotoxic effects and coagulation abnormalities associated with viper envenomation can be successfully managed through prompt administration of polyvalent anti-snake venom and appropriate supportive therapy. However, the subsequent development of acute kidney injury, despite initial stabilization and normal renal parameters at presentation, underscores the risk of delayed organ involvement following envenomation. This case highlights the critical importance of early intervention, continuous clinical and laboratory monitoring, and extended post-envenomation surveillance; particularly of renal function to improve outcomes in canine snakebite cases.

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Availability of Data and Materials

All data generated or analysed during this study are included in this published article.

Competing interests

The authors declare no competing interests.

Authors Contribution

Abhijith S.P. conceived and designed the case report, framed the manuscript structure, performed the primary writing, and critically revised and corrected the manuscript for intellectual content. Abdul Kalam A. was responsible for clinical management of the case, including patient monitoring, treatment administration, and follow-up, and contributed clinical inputs to the manuscript. Apoorva H.J. assisted in data collection, literature review, and contributed to manuscript drafting. Chandrakumar D. contributed to data acquisition, literature compilation, and assisted in manuscript preparation. Sanjay H.V. assisted in data collection, literature support, and manuscript writing. All authors read and approved the final manuscript.

Consent for publication

Informed consent for publication was obtained from the owner of the dog involved in the study.

Data Availability

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethical Approval Not applicable.

Consent to Participate Not applicable.

Consent to Publish Not applicable.

Conflict of Interest The authors declare no competing interests.

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