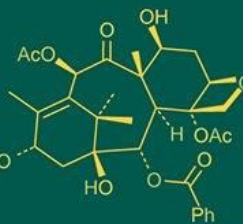
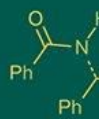


International Journal of Advanced Biochemistry Research



ISSN Print: 2617-4693
ISSN Online: 2617-4707
NAAS Rating (2025): 5.29
IJABR 2025; 9(12): 1141-1144
www.biochemjournal.com
Received: 15-09-2025
Accepted: 18-10-2025

S Kavibharathy
Assistant Professor,
Department of Veterinary
Medicine, Veterinary College
and Research Institute, Salem,
Tamil Nadu, India

PA Enbavelan
Assistant Professor,
Department of Veterinary
Medicine, Veterinary College
and Research Institute, Salem,
Tamil Nadu, India

E Venkatesakumar
Professor and Head,
Department of Veterinary
Medicine, Veterinary College
and Research Institute, Salem,
Tamil Nadu, India

S Sivaraman
Associate Professor, Veterinary
Clinical Complex, Veterinary
College and Research Institute,
Salem, Tamil Nadu, India

S Sunandhadevi
Department of Veterinary
Medicine, Veterinary College
and Research Institute, Salem,
Tamil Nadu, India

A Elango
Dean, Veterinary College and
Research Institute, Salem,
Tamil Nadu, India

V Ramakrishnan
Assistant Professor,
Department of Veterinary
Pharmacology and Toxicology,
Veterinary College and
Research Institute, Tirunelveli,
Tamil Nadu, India

Corresponding Author:
S Kavibharathy
Assistant Professor,
Department of Veterinary
Medicine, Veterinary College
and Research Institute, Salem,
Tamil Nadu, India

Biochemical alterations and hemotoxic effects of Russell's viper (*Daboia russelii*) envenomation in a dog: A case report

S Kavibharathy, PA Enbavelan, E Venkatesakumar, S Sivaraman, S Sunandhadevi, A Elango and V Ramakrishnan

DOI: <https://www.doi.org/10.33545/26174693.2025.v9.i12n.6972>

Abstract

Snake envenomation is a frequent and potentially fatal emergency in dogs, particularly in tropical and subtropical regions where encounters with venomous snakes are common. The severity of envenomation depends on the species of snake, amount of venom injected, bite location and the timeliness of therapeutic intervention. Viperid envenomation, especially by Russell's viper (*Daboia russelii*) is commonly associated with local tissue damage, severe edema, hemorrhagic manifestations, coagulopathies and acute renal injury in dogs.

A four year old male Kombai dog was presented to the Veterinary Clinical Complex, Veterinary College and Research Institute, Salem, with a history of acute swelling in the neck region noticed one day prior to presentation. Clinical examination revealed profuse salivation, marked edematous swelling involving the jaw and neck, serous nasal discharge and signs of respiratory distress. Fang marks were clearly visible on the ventral aspect of the neck. Hematological evaluation revealed a prolonged twenty minute whole blood clotting time, suggestive of venom induced coagulopathy. Serum biochemical analysis demonstrated elevated creatinine (2.5 mg/dL) and blood urea nitrogen (72 mg/dL), indicating renal involvement.

Based on the presence of fang marks, characteristic clinical signs, clotting abnormalities and biochemical alterations, the case was diagnosed as Russell's viper envenomation. The dog was treated promptly with 10 mL of polyvalent snake venom antiserum, along with supportive therapy including broad-spectrum antibiotics, corticosteroids, diuretics and fluid therapy. The animal was closely monitored for clinical and laboratory response.

Marked clinical improvement, evidenced by a reduction in cervical swelling, resolution of respiratory distress and normalization of clotting time, was observed from the following day of treatment. Hemato-biochemical parameters gradually returned to normal by the fifth day of presentation. The dog showed complete clinical recovery without any complications, highlighting the importance of early diagnosis and prompt administration of antivenom combined with appropriate supportive care in canine snake envenomation.

Keywords: Snake envenomation, polyvalent snake venom antiserum, twenty minutes whole blood clotting time positive

Introduction

The Indian subcontinent is characterized by a remarkable diversity of ophidian fauna, with more than 270 identified snake species, of which approximately 60 are considered venomous [1]. Among these, the "big four" venomous snakes—spectacled cobra (*Naja naja*), Russell's viper (*Daboia russelii*), common krait (*Bungarus caeruleus*) and saw-scaled viper (*Echis carinatus*)—are responsible for the majority of clinically significant and fatal envenomation cases in both humans and animals [2]. Snakebite envenomation represents an important veterinary emergency in India, particularly in rural and semi-urban regions where domestic animals frequently encounter snakes in agricultural fields and peri-domestic environments [3]. Snakebite incidents are reported more commonly in dogs and horses compared to large ruminants such as cattle and sheep, largely due to their inquisitive behavior and increased mobility [4]. Dogs often sustain bites on the head, neck or forelimbs while investigating snakes, leading to rapid venom absorption and severe clinical consequences.

The clinical severity of snake envenomation varies widely and depends on several factors including the species of snake, venom yield, number of bites, anatomical site of envenomation and the time interval between the bite and initiation of treatment [5].

In dogs, viperid envenomation is typically associated with marked local reactions such as extensive edematous swelling, severe pain, ecchymosis, hemorrhage and discoloration of the skin at the bite site [6]. These manifestations result from the cytotoxic, proteolytic and vasoactive components of viper venom, which increase vascular permeability and cause tissue necrosis. Systemic complications may include pulmonary dysfunction, hypotension, shock, cardiac arrhythmias, hemolysis and severe coagulation abnormalities due to venom-induced consumption of clotting factors [7]. Russell's viper venom is particularly known for its potent hemotoxic and nephrotoxic effects, frequently resulting in acute kidney injury secondary to direct tubular damage, hemoglobinuria, myoglobinuria and systemic hypotension [8].

Additional systemic signs commonly observed in dogs include ptyalism, nausea, vomiting, respiratory distress and spontaneous bleeding from mucosal surfaces. Early diagnosis based on clinical signs, identification of fang marks and simple bedside tests such as the twenty-minute whole blood clotting time (WBCT) is essential for confirming viper envenomation [9]. Prompt administration of antivenom, supported by appropriate symptomatic and supportive therapy, remains the cornerstone of successful management. The present paper describes a case of Russell's viper (*Daboia russelii*) envenomation in a dog and its successful therapeutic management.

Case history and clinical observations

A four-year-old male Kombai dog weighing 12.3 kg was presented to the Veterinary Clinical Complex with a history of acute swelling over the ventral aspect of the neck noticed since the previous day. According to the owner, the swelling had developed suddenly and progressively increased in size, accompanied by excessive salivation and discomfort.

On physical examination, the dog was conscious but appeared dull and depressed, with a subdued demeanor. The animal was able to maintain a standing posture and ambulated normally without signs of lameness or ataxia. Body condition was within normal limits. Rectal temperature was recorded at 38.5 °C, which is within the normal physiological range for dogs. A prominent, diffuse edematous swelling was observed over the ventral cervical region extending towards the mandibular area (Figure 1). The swelling was warm, tense and elicited severe pain on palpation. Profuse salivation was evident. Careful inspection of the cervical region revealed distinct fang marks on the ventral neck (Figure 2), strongly suggestive of snake envenomation. The conjunctival mucous membranes were pink and moist, indicating adequate peripheral perfusion. Cardiovascular examination revealed tachycardia with a heart rate of 125 beats per minute, while respiratory examination showed tachypnea with a respiratory rate of 42 breaths per minute, suggestive of mild respiratory distress likely due to cervical edema. The skin and hair coat were smooth and shiny and the facial expression was woebegone. Peripheral lymph nodes were palpated and found to be within normal limits, with no abnormalities detected in posture or gait.



Fig 1: Diffuse, tense edematous swelling over the ventral cervical and mandibular region of the dog.



Fig 2: Distinct fang marks visible on the ventral neck, indicative of snake envenomation.



Fig 3: Twenty-minute whole blood clotting time (WBCT) test showing failure of clot formation, indicating venom-induced coagulopathy.



Fig 4: Collection of blood samples in EDTA and plain tubes for hematological and serum biochemical analysis.

Whole blood was collected and subjected to the twenty-minute whole blood clotting time (20-minute WBCT) test to assess coagulation status. The collected blood failed to clot within twenty minutes, indicating a severe coagulation defect consistent with viper envenomation (Figure 3). Based on the history, presence of fang marks, local and systemic clinical signs and a positive WBCT, the case was provisionally diagnosed as Russell's viper (*Daboia russelii*) envenomation.

Blood samples were subsequently collected in EDTA tubes for hematological evaluation and in plain tubes for serum biochemical analysis (Figure 4). Hematological examination revealed leukocytosis with marked neutrophilia, indicating an acute inflammatory response. Thrombocytopenia was also observed, further supporting venom-induced coagulopathy. Other erythrocytic indices were within normal physiological limits. Serum biochemical analysis revealed elevated blood urea nitrogen (72 mg/dL) and creatinine (2.5 mg/dL), suggestive of renal involvement. Serum lactate dehydrogenase levels were increased, reflecting tissue damage, while total protein, albumin and globulin concentrations remained within normal reference ranges.

Treatment and clinical outcome

Based on the diagnosis, immediate treatment was initiated. On the first day of presentation, the dog was administered 10 mL of lyophilized polyvalent snake venom antiserum intravenously, guided by the positive 20-minute WBCT. Supportive therapy included intravenous isotonic normal saline (300 mL) to maintain hydration and renal perfusion. Enrofloxacin was administered at 7.5 mg/kg body weight subcutaneously to prevent secondary bacterial infection. Furosemide was given at 1 mg/kg body weight intravenously to support renal function and promote diuresis and dexamethasone at 0.2 mg/kg body weight intravenously was administered to reduce inflammation and edema (Figure 5).

On the second day, the animal showed noticeable clinical improvement, with a reduction in cervical swelling and respiratory distress. Treatment was continued with isotonic normal saline (400 mL, IV), enrofloxacin (7.5 mg/kg body weight, SC), furosemide (1 mg/kg body weight, IV) and dexamethasone (0.2 mg/kg body weight, IV). From the third day onwards, supportive therapy was continued for a total duration of four days. Progressive clinical improvement was observed, with marked reduction in edema and restoration of normal activity. The twenty-minute WBCT normalized and the dog showed complete clinical recovery (Figure 6). Hemato-biochemical parameters returned to normal by the fifth day of presentation and the recovery was uneventful.



Fig 5: Intravenous administration of polyvalent snake venom antiserum and supportive therapy in the affected dog.



Fig 6: Complete clinical recovery of the dog with resolution of cervical edema following treatment.

On the third day of treatment, the dog was administered isotonic normal saline (400 mL, IV) to maintain hydration and renal perfusion. Enrofloxacin was continued at a dose of 7.5 mg/kg body weight subcutaneously to prevent secondary bacterial infection. Furosemide was administered at 1 mg/kg body weight intravenously to support renal function and promote diuresis. Dexamethasone was continued at 0.2 mg/kg body weight intravenously to control inflammation and edema. This therapeutic regimen was maintained for a total duration of four days.

Results

The animal exhibited marked clinical improvement within 24 hours of initiation of treatment. There was a noticeable reduction in cervical swelling and a gradual resolution of respiratory distress. The twenty-minute whole blood clotting time improved significantly and returned to less than ten minutes, indicating restoration of normal coagulation status. The dog became alert, active and resumed normal feeding behavior. Complete clinical recovery was observed by the fifth day of treatment, with normalization of hematological and biochemical parameters. The recovery was uneventful and no adverse reactions to antivenom therapy were recorded (Figure 7).



Fig 7: Complete clinical recovery of the dog following treatment.

Discussion

Snakebite envenomation is commonly encountered in companion animals, particularly dogs, due to their inquisitive nature and frequent exposure to snake habitats. Bite sites are most often observed on the head, neck, muzzle, trunk and extremities, as dogs tend to investigate snakes with their face. In the present case, the bite was located on the ventral aspect of the neck, resulting in

marked local edema and respiratory distress due to proximity to the upper airway.

The diagnosis of snake envenomation was supported by the presence of fang marks, characteristic clinical signs and a positive twenty-minute whole blood clotting time (WBCT20), which is a simple and reliable bedside test for detecting venom-induced coagulopathy. Failure of blood to clot within 20 minutes strongly indicates hemotoxic envenomation and necessitates prompt administration of anti-snake venom.

Snake venoms are broadly classified into neurotoxic and hemotoxic types. Viper venom is predominantly hemotoxic and contains a complex mixture of biologically active components such as proteases, metalloproteinases, phospholipase A₂, phospholipase B, amino acid esterases and neurotoxic fractions (Aroch and Harrus, 1999). These toxins induce extensive local tissue damage, edema, hemorrhage and systemic effects including cardiopulmonary dysfunction and coagulation abnormalities. Hemorrhagins present in viper venom cause endothelial damage, increased vascular permeability, coagulation factor consumption and extravasation of plasma into surrounding tissues, resulting in severe swelling and edema (Garg, 2002).

Administration of tetanus toxoid is recommended to protect against *Clostridium tetani*, which may be introduced through contaminated snake fangs. Broad spectrum antibiotics were administered in the present case to prevent secondary bacterial infection, as snake oral cavities are known to harbor diverse pathogenic bacteria. The WBCT 20 played a critical role in guiding antivenom therapy, as persistent failure of clot formation beyond 20 minutes indicates the need for repeated doses of polyvalent snake venom antiserum until coagulation normalizes.

Conclusion

Administration of antivenom remains the only specific and definitive therapy for snake envenomation. Polyvalent snake venom antiserum neutralizes circulating venom and prevents further progression of systemic toxicity, although it cannot reverse tissue damage that has already occurred. The timing of antivenom administration following snakebite is a crucial determinant of clinical outcome and survival. Therefore, early recognition of snake envenomation, prompt diagnosis using simple tests such as the twenty minute whole blood clotting time and immediate initiation of antivenom therapy along with appropriate supportive care are of paramount importance for successful therapeutic management and favorable prognosis in canine snakebite cases.

References

- Whitaker R, Captain A. Snakes of India: The Field Guide. Chennai: Draco Books; 2004.
- Warrell DA. Guidelines for the management of snakebites. New Delhi: World Health Organization, Regional Office for South-East Asia; 2016.
- Mohapatra B, Warrell DA, Suraweera W, *et al.* Snakebite mortality in India: a nationally representative mortality survey. PLoS Negl Trop Dis. 2011;5(4):e1018:1-8.
- Garg SK. Snakebite and its management in animals. Indian Vet J. 2000;77:1-6.
- Klassen KM. Snake envenomation in dogs and cats. Compend Contin Educ Vet. 2008;30:206-216.
- Peterson ME, Talcott PA. Small Animal Toxicology. 3rd ed. St. Louis: Elsevier; 2013.
- Hodgson E, Levi PE. A Textbook of Modern Toxicology. 4th ed. Hoboken: Wiley-Blackwell; 2010.
- Sitprija V. Nephrotoxicity of snake venoms. Kidney Int. 2008;74:24-33.
- Warrell DA. Snakebite envenoming: a neglected tropical disease. PLoS Negl Trop Dis. 2010;4(6):e607:1-7.