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Successful management of Russell's viper (*Daboia russelii*) envenomation in a two-year-old non-descript male dog

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Abstract

Snake envenomation is a frequent veterinary emergency in tropical regions, particularly in India, where Russell's viper (*Daboia russelii*) is a major cause of severe systemic toxicity. A two-year-old non-descript male dog was presented in lateral recumbency following a suspected Russell's viper bite, with clinical signs including hemoglobinuria and incoagulable blood confirmed by a positive 20-minute whole blood clotting test (WBCT20). These findings were consistent with systemic envenomation characterised by venom-induced consumption coagulopathy (VICC) and intravascular hemolysis. The dog was promptly treated with polyvalent anti-snake venom, tetanus toxoid, broad-spectrum antibiotics, and intensive supportive therapy. Marked clinical improvement was observed within days, with normalization of coagulation parameters and renal function. The patient achieved full recovery after seven days of hospitalization. This case highlights the effectiveness of early antivenom therapy and supportive care, and underscores the utility of WBCT20 as a rapid bedside diagnostic tool in resource-limited veterinary settings.

Keywords: Russell's viper, WBCT20, canine snakebite, venom-induced consumption coagulopathy, anti-snake venom

Introduction

Snakebite envenomation represents a significant cause of morbidity and mortality in animals in tropical and subtropical regions ^[1]. In India, Russell's viper (*Daboia russelii*) is among the most medically important venomous snakes, responsible for severe hemotoxic and nephrotoxic effects ^[2, 6]. The venom contains a complex mixture of proteases, phospholipases, and procoagulant enzymes that result in venom-induced consumption coagulopathy, endothelial injury, hemolysis, and acute kidney injury (AKI) ^[4, 7]. Dogs are particularly susceptible to viper envenomation due to their inquisitive behavior and outdoor exposure ^[8]. Clinical manifestations commonly include local tissue damage, hemorrhage, anemia, hemoglobinuria, incoagulable blood, and renal dysfunction ^[8, 9]. Early diagnosis and prompt administration of antivenom remain the cornerstone of successful management ^[2, 10]. The WBCT20 is a simple, inexpensive, and reliable test widely used to detect coagulopathy in viper envenomation, especially in rural and resource-constrained settings ^[3, 11].

Case History and Observation

A two-year-old non-descript male dog weighing approximately 18 kg was presented with a history of lateral recumbency and suspected snakebite (Figure 1). The animal passed dark reddish-brown urine, suggestive of hemoglobinuria, a common sequela of viper envenomation. Clinical examination revealed pale mucous membranes, prolonged capillary refill time, tachycardia, and weak peripheral pulses. Although fang marks were not clearly visible, the absence of distinct bite marks does not rule out viper envenomation due to deep tissue penetration and rapid tissue edema.

A WBCT20 performed at presentation showed complete absence of clot formation at 20 minutes, confirming systemic envenomation and severe coagulopathy.

Diagnostic Evaluation

Hematological and biochemical evaluation revealed anemia, mild thrombocytopenia, leukocytosis, elevated liver enzymes, hyperbilirubinemia, and marked azotemia, consistent with venom-induced hemolysis, hepatic involvement, and acute kidney injury.

Table 1: Hematobiochemical parameters before and after treatment

Parameter	Pre-treatment	Post-treatment (Day 7)
Red blood cells ($\times 10^6/\mu\text{l}$)	3.38	4.67
Hemoglobin (g/dl)	7.0	8.0
Packed cell volume (%)	22.2	25.8
Platelet count ($\times 10^3/\mu\text{l}$)	141	144
White blood cells ($\times 10^3/\mu\text{l}$)	15.8	15.2
Alanine aminotransferase (U/L)	154	121
Creatinine (mg/dl)	3.9	1.4
Total bilirubin (mg/dl)	8.6	9.2
Direct bilirubin (mg/dl)	4.8	5.28



Fig 1: Dog with Russell's viper

Treatment and Management

Immediate treatment was initiated with intravenous administration of Polyvalent Anti-Snake Venom (ASV) following standard protocols. Supportive therapy included isotonic intravenous fluids to maintain renal perfusion, broad-spectrum antibiotics to prevent secondary bacterial infections, and tetanus toxoid as prophylaxis. Antihistamines and corticosteroids were administered to mitigate hypersensitivity reactions to antivenom. Serial monitoring of urine output, coagulation status, and renal parameters was performed. Repeat WBCT20 after 24 hours demonstrated restoration of clot formation, indicating reversal of venom-induced coagulopathy.

Outcome and Follow-Up

Clinical improvement was evident within 48-72 hours, with resolution of hemoglobinuria and improvement in mentation. Renal function progressively normalized, as reflected by declining serum creatinine levels. By day 7,

hematobiochemical parameters showed marked improvement, and the dog was discharged with complete clinical recovery. Early antivenom therapy has been shown to significantly reduce mortality and complications in viper envenomation, consistent with the favorable outcome observed in this case.

Discussion

Russell's viper venom induces a complex pathophysiological cascade involving activation and consumption of clotting factors, fibrinolysis, hemolysis, and direct nephrotoxicity [4, 7]. Venom-induced consumption coagulopathy is a hallmark of viper envenomation and is reliably detected using WBCT20, which remains a practical bedside test in veterinary practice [3, 11]. Hemoglobinuria observed in this case is attributed to intravascular hemolysis and has been reported frequently in canine viper envenomation [8, 9]. Early administration of antivenom neutralizes circulating venom, preventing progression of systemic toxicity, while supportive care plays a critical role in managing secondary organ damage [10, 14, 15].

Conclusion

This case report emphasizes that prompt clinical recognition, early use of WBCT20, timely administration of polyvalent antivenom, and comprehensive supportive therapy can lead to successful outcomes in canine Russell's viper envenomation. Veterinary clinicians in snakebite-endemic regions should prioritize rapid diagnosis and immediate antivenom therapy to minimize morbidity and mortality.

Conflict of Interest

Not available.

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