Case Report

Clinical Outcomes of Hematotoxic Snakebite Managed with Antisnake Venom–Guided Therapy

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Abstract

Background: Snakebite remains a major medical emergency in rural India, frequently associated with rapidly progressive and life-threatening complications. We report the case of a 45-year-old previously healthy male farmer who sustained a Russell's viper bite. He initially presented with localized limb swelling and prolonged whole blood clotting time and was treated with anti-snake venom (ASV). Six hours later, he developed hematuria, hypotension, respiratory distress, and hemoptysis. Chest imaging revealed bilateral pulmonary infiltrates consistent with alveolar hemorrhage. Laboratory evaluation demonstrated severe coagulopathy and thrombocytopenia, progressing to disseminated intravascular coagulation and acute kidney injury with oliguria, necessitating hemodialysis. With worsening of the patient's condition, a tele-ICU consultation was sought, which advised escalation of ASV therapy, proving to be the critical turning point in his clinical course. The patient showed significant clinical improvement and was discharged in stable condition on day eleven. This case underscores the lifesaving role of guided ASV escalation and the value of collaborative e-critical care in managing severe envenomation in resource-limited settings.

Keywords: Russell Viper Bite, Hematotoxic Envenomation, Venom-Induced Consumption Coagulopathy, Anti-Snake Venom, Whole Blood Clotting Time.

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Introduction

Snakebite is a major public health concern in many tropical and subtropical regions, with an estimated 5.4 million bites occurring annually, leading to 1.8-2.7 million cases of envenomation. The World Health Organization (WHO) recognizes snakebite as a neglected tropical disease.[1] India alone is responsible for nearly half of the estimated 138,000 snakebite-related deaths and 400,000 disabilities that occur worldwide each year. According to the World Health Organization, India is home to approximately 236 snake species, of which 17 are classified as medically important.[2] Of the over 2,000 snake species worldwide, around 300 are found in India, with 52 of them being venomous.

In India, venomous snakes belong to three main families: Elapidae, Viperidae, and Hydrophiidae (sea snakes). The most common species include the Indian cobra (Naja naja) and Indian krait (Bungarus caeruleus) from the Elapidae family are neurotoxic, while Russell's viper (Daboia russelii) and the saw-scaled viper (Echis carinatus) from the Viperidae family are primarily vasculotoxic or hematotoxic. In India, the available anti-snake venom (ASV) is polyvalent, offering efficacy against all four of these common species. The most frequent systemic manifestation of hematotoxic snakebite is venom-induced consumption coagulopathy (VICC).

The dose of ASV in hemotoxic snakebite varies with the amount of venom injected and the type of snake species; however, in an emergency setting, accurately determining either is usually not possible. The optimal protocol for ASV administration remains unclear. Therefore, ASV should be administered at the earliest sign of envenomation to neutralize circulating venom and prevent complications.[3] There are also no definitive guidelines for ASV dosing in cases of persistent coagulopathy.

Case Presentation

A 45-year-old previously healthy male farmer was bitten on his right foot by a russel viper (Figure 1a) while returning home from the field. He had no known history of any chronic medical condition. Soon after the bite, he developed mild pain and progressive swelling at the bite site. The patient reached approximately 6 hours post-bite. On admission, the patient was conscious and well-oriented. His vital signs were as follows: blood pressure (BP) 140/95 mmHg, respiratory rate (RR) 20

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breaths per minute, pulse rate (PR) 110 beats per minute, and peripheral oxygen saturation (SpO_2) of 97% on room air. Local examination of the right foot revealed fang marks with surrounding swelling (Figure 1b).

On presentation, his whole blood clotting time (WBCT) was more than 20 minutes. He was administered 10 vials of ASV. At this point, he had no systemic symptoms other than localized swelling and mild pain. Relevant laboratory investigations are summarized in Table 1. However, six hours after ASV administration, he developed hematuria. Subsequently, the patient developed acute shortness of breath and tachypnea, with a drop in SpO_2 to 88% on room air and hypotension with BP of 80/60 mmHg. He was initiated on vasopressors and oxygen support at 5 L/min via face mask, which improved his SpO_2 to 97%. An additional 10 vials of ASV were administered; however, there was no clinical improvement. The patient developed hemoptysis, prompting an immediate chest X-ray, which revealed bilateral infiltrates consistent with alveolar hemorrhage (Figure 1c). A subsequent 2D echocardiogram demonstrated normal findings.

The patient was administered 4 units of fresh frozen plasma and 1 unit of single donor platelets. Intravenous medications included piperacillin-tazobactam 4.5 g three times daily, pantoprazole 40 mg once daily, ondansetron 3 mg three times daily, tranexamic acid 1 g three times daily, and vitamin K 10 mg once daily, along with intravenous fluids (normal saline) at a rate of 60 mL/hour. Despite these measures, the patient's condition deteriorated, with a progressive drop in platelet count to 20,000/µL, and he developed features of disseminated intravascular coagulation (DIC) as shown in [Table 2]. As the condition progressed, he developed hematuria and hypotension, leading to acute kidney injury (AKI).

At this critical juncture, a tele-ICU consultation was sought with the hub at SGPGI, Lucknow, an advanced center where complex, multispeciality cases are routinely discussed for expert guidance. Based on their recommendations, the patient was administered an additional 10 vials of ASV, followed by 6 vials every 6 hours, continued until normalization of the 20-minute WBCT, resolution of swelling, or cessation of bleeding.

He responded well to the advised ASV regimen, leading to a marked improvement in his condition. The patient's hemodynamic status improved, and his shock began to resolve. He underwent two sessions of hemodialysis due to decreased urine output. Subsequently, his platelet count increased to 1.75 lakh/µL, coagulation parameters normalized, and hemoglobin levels remained stable at 11 g/dL. The patient was successfully discharged on day eleven in a hemodynamically stable condition.

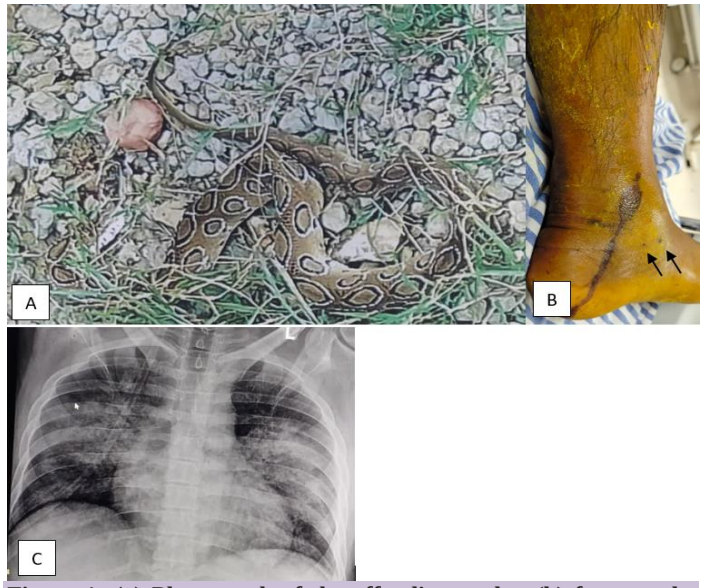

Figure 1: (a) Photograph of the offending snake; (b) fang marks (black arrows) with associated leg swelling; (c) bilateral chest infiltrates on chest X-ray.

Table 1: Laboratory parameters at the time of admission. HB: Hemoglobin; TLC: Total Leukocyte Count; TPC: Total Platelet Count; S. Na: Serum Sodium; S. K: Serum Potassium; Cl: Serum Chloride; AST: Aspartate Aminotransferase; ALT: Alanine Aminotransferase; CRP: C-Reactive Protein; CPK: Creatine Phosphokinase; APTT: Activated Partial Thromboplastin Time; PT: Prothrombin Time; INR: International Normalized Ratio.

Analyte	Patient result	Reference value
HB	14	13-17 g/dL
TLC	12	4 -11 x 10^9/L
TPC	210	150 – 400 x 10^9/L
S. Na	138	135-145 mEq/L
S.K	4.5	3.5-5 mEq/L
CL	100	96-115
AST	300	5-40 U/L
ALT	50	5-40 U/L
T. Bilirubin	1.2	0.0-2.0 mg/dL
Alkaline phosphatase	120	40-129 IU/L
T. Protein	6.8	6.0-8.3 g/dL
S. Albumin	3.5	3.3-5.2 g/dL
Urea	45	13-45 mg/dL
Creatinine	1.2	0.5-1.5 mg/dL
Calcium	7.6	8.6-10.3 mg/dL
Procalcitonin	.02	<0.05 mcg/L
CRP	16	0.0–1.0 mg/dL
CPK	450	39–308 U/L (male)
APTT	40	25–35 sec
PT	16	11–15 sec
INR	1.4	0.8–1.2

Urine for myoglobin and hemoglobin	Negative	Negative

Table 2: Coagulation profile after 24 hours of admission. FDP: Fibrin Degradation Products

Analyte	Patient result	Reference value
HB	11	13-17 g/dL
TLC	16	4-11 x 10^9/L
TPC	20	150-400 x 10^9/L
APTT	52	25–35 sec
PT	21	11–15 sec
INR	1.8	0.8–1.2
D-Dimer	15000	<500 ng/mL
Fibrinogen	<100	200 – 400mg/dL
FDP	7.4	<5 µg/mL

Discussion

Snakebites are frequent among individuals engaged in outdoor and agricultural work in Southeast Asia. Snake venom is a complex natural toxin composed of various biologically active components, including neurotoxins, hemorrhagins, coagulants, nephrotoxins, myotoxins, and necrotoxins.[4] Its composition varies with species, season, geographical region, and diet, resulting in a wide range of local and systemic effects.

Viper envenomation, in particular, may produce extensive local and systemic complications. Local effects include tissue necrosis and cellulitis, while systemic manifestations can range from neuroparalysis, coagulopathy, AKI, rhabdomyolysis, shock, spontaneous systemic bleeding, DIC, thrombotic microangiopathy, diffuse alveolar hemorrhage, and acute respiratory distress syndrome (ARDS). Central nervous system involvement may occur in the form of intracranial hemorrhagic or ischemic stroke, and endocrine complications including hypopituitarism and adrenal insufficiency.[5-8]

VICC, once mistaken for DIC in snakebite cases, is now recognized as a distinct condition caused by venom-induced depletion of clotting factors through procoagulant toxins. Unlike DIC, which involves widespread coagulation activation and high mortality, VICC develops and resolves rapidly within 24-48 hours, and lacks nonrenal organ damage.[9] It presents with abnormal coagulation tests but follows a more benign course.[10]

Anti-snake venom is the cornerstone of snakebite management, effectively binding to venom toxins and neutralizing their harmful effects. The primary role of ASV is to halt the ongoing consumption process in coagulopathy; however, it cannot reverse already established irreversible toxic effects. Since the body requires approximately 48 hours to resynthesize clotting factors, the patient remains at risk of bleeding during this period. This underlies the rationale for administering fresh frozen plasma (FFP).[11] Although administration of FFP alone without antivenom can sometimes produce satisfactory outcomes, concerns remain that FFP might exacerbate coagulopathy by supplying additional substrate for the activation of procoagulant venom toxins.[12] The benefit of FFP in VICC without active bleeding is still uncertain, and further research is needed to clarify its role in this specific patient group.

Renal impairment after snakebite arises from multiple mechanisms, including acute tubular necrosis, interstitial nephritis, glomerulonephritis, rhabdomyolysis, microthrombi, and direct nephrotoxicity. Mortality from snakebite-induced AKI ranges from 1to 20%. Plasma exchange shows no clear benefit, though it may be considered in severe, refractory cases.[13] Early dialysis improves survival, with many patients requiring it during recovery.[14]

Early administration of FFP may help prevent AKI, and evidence suggests that immunoglobulins present in FFP can protect against toxin-induced primary renal injury in VICC through immunomodulatory effects, despite the potential risks associated with introducing clotting factors.[15] Observational data indicate that therapeutic plasma exchange (TPE) offers no major outcome benefit and is linked to higher transfusion and dialysis needs, though it may improve hematologic parameters faster. Snakebite-related AKI often progresses to chronic kidney disease (CKD), with 37-41% developing renal abnormalities within a year.[16]

ARDS following a snakebite is uncommon and may result from DIC, aspiration, or direct venom effects.[17] In viper bites, AKI may contribute to pulmonary edema and subsequent ARDS. However, in our case, ARDS developed before the onset of AKI, suggesting that immune complex deposition and DIC were the primary mechanisms rather than fluid overload.

The time between a venomous snakebite and the administration of ASV, known as the bite-to-needle interval, is a crucial factor influencing outcomes in hemotoxic envenomation. Initiating ASV within the first 4 to 6 hours markedly lowers the risk of complications and mortality, whereas delays beyond this window permit venom spread through the vascular and lymphatic systems, causing irreversible endothelial damage, organ dysfunction, and coagulopathy. The site of a snakebite influences the rate of venom spread within the body, thereby affecting the onset and severity of symptoms as well as the effectiveness window for ASV.[18]

High-dose ASV is often preferred in cases of severe systemic envenomation or delayed presentation. Despite ASV being the cornerstone of poisonous snakebite management, there is no universally accepted guideline for its optimal dose or administration schedule, leading to considerable regional variation.

Hemotoxic snake envenomation requires a higher dose of ASV compared to neurotoxic envenomation.[19]

The role of steroids in snakebite management remains controversial; they are beneficial in managing complications such as anaphylaxis or allergic reactions to ASV.[20]

Conclusion

Despite significant progress, standardized protocols for ASV dosing and the management of persistent coagulopathy remain insufficient, highlighting the need for further research. Collaborative critical care models, such as tele-ICU consultations, have demonstrated a meaningful impact on improving outcomes in severe cases, especially in resource-limited settings.

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Conflicts of interest
There are no conflicts of interest.

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