RESEARCH PAPER

Clinical Spectrums of Snake Envenomation: A Case Series on Management and Outcomes

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ABSTRACT
Snake envenomation remains a major medical emergency, particularly in tropical countries, with clinical presentations ranging from mild local reactions to severe systemic complications. This case series describes three patients with distinct clinical spectrums of snake envenomation managed in a tertiary care centre. The first case involved isolated neurotoxic envenomation due to cobra bite causing respiratory distress requiring mechanical ventilation. The second case presented with mixed neurotoxic and hemotoxic manifestations complicated by coagulopathy and antivenom-induced anaphylaxis. The third case demonstrated severe mixed envenomation associated with acute kidney injury and rhabdomyolysis requiring haemodialysis. Early recognition, prompt anti-snake venom administration, and timely critical care interventions were crucial in achieving favourable outcomes in all three cases.
Keywords: *Snake envenomation, Neurotoxicity, Hemotoxicity, Anti-snake venom, acute kidney injury, Rhabdomyolysis.*
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INTRODUCTION

Snakebite envenomation remains a major public health concern, especially in tropical and subtropical regions where human interaction with snakes is common. It predominantly affects rural and agricultural populations due to occupational exposure, poor housing conditions, and delayed access to healthcare facilities. Despite advances in emergency medicine, snakebite continues to cause significant morbidity and mortality worldwide. In addition to deaths, many survivors experience long-term complications such as tissue necrosis, amputations, chronic kidney disease, and persistent neurological deficits [1].India contributes significantly to the global burden of snakebite-related deaths and disabilities. The country's tropical climate, agricultural workforce, and large rural population increase the frequency of human-snake encounters. Snakebites are particularly common during monsoon and harvesting seasons. Although hospital-based data provide some estimates, the true burden remains underestimated because many bites occurring in rural settings are either untreated or unreported. Consequently, snakebite remains an important but often neglected medical emergency in India [2].In India, the majority of clinically significant envenomation cases are caused by the "big four" venomous snakes: Indian cobra (*Naja naja*), common krait (*Bungarus caeruleus*), Russell's viper (*Daboia russelii*), and saw-scaled viper (*Echis carinatus*) [3]. The type and severity of envenomation vary depending on the snake species, amount of venom injected, site of bite, and time to medical intervention. This variability contributes to a wide range of clinical presentations, making early recognition and prompt management essential.

Snake venom is a complex mixture of enzymes, proteins, and toxins that can affect multiple organ systems. Depending on the venom composition, patients may present with neurotoxic, hemotoxic, myotoxic, cytotoxic, or mixed manifestations [4]. The clinical spectrum ranges from mild local swelling to life-threatening systemic complications involving respiratory failure, coagulopathy, acute kidney injury, and multiorgan dysfunction. Neurotoxic envenomation is commonly seen with cobra and krait bites. These venoms interfere with neuromuscular transmission, leading to progressive

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paralysis. Early symptoms often include ptosis, diplopia, dysarthria, dysphagia, and generalized weakness. Without timely intervention, paralysis may progress to involve respiratory muscles, causing respiratory failure. Early recognition of neurological deterioration and prompt airway management are critical to improving survival [5].Hemotoxic envenomation is more frequently associated with viper bites and primarily affects coagulation pathways and vascular integrity. Patients may develop coagulopathy, spontaneous bleeding, thrombocytopenia, and haemorrhagic complications. Clinical manifestations include bleeding from the bite site, gum bleeding, haematuria, melena, and hematemesis. The 20-minute whole blood clotting test remains a simple and useful bedside test for identifying venom-induced coagulopathy, particularly in resource-limited settings [6].

In addition to neurotoxic and hemotoxic effects, severe snake envenomation can cause systemic complications such as rhabdomyolysis, shock, and acute kidney injury. Acute kidney injury is an important predictor of poor outcomes and may result from direct nephrotoxicity, hypotension, hemolysis, pigment nephropathy, or rhabdomyolysis. Patients with severe renal impairment may require renal replacement therapy such as haemodialysis. These complications significantly increase morbidity and hospital stay [4].The cornerstone of management is early diagnosis, prompt supportive care, and administration of anti-snake venom (ASV), which remains the only specific treatment for snake envenomation [7]. ASV works by neutralizing circulating venom and is most effective when administered early. Indications include progressive neurotoxicity, coagulopathy, severe local swelling, and systemic manifestations of envenomation. Despite its benefits, ASV administration can be associated with adverse reactions ranging from mild hypersensitivity to severe anaphylaxis. Patients may develop itching, urticaria, hypotension, bronchospasm, or shock during infusion. Such reactions require immediate management with adrenaline and supportive care. Therefore, close monitoring during ASV administration is essential [6].

This case series highlights the varied clinical spectrum of snake envenomation, ranging from isolated neurotoxic manifestations with respiratory distress to mixed neurotoxic and hemotoxic presentations complicated by coagulopathy, acute kidney injury, rhabdomyolysis, and antivenom-related adverse reactions. These cases emphasize the importance of early recognition, timely intervention, and multidisciplinary critical care in improving patient outcomes.

CASE SERIES

Case 1:
A 15-year-old female patient brought to ER with A/H/O snake bite (COBRA) to Left foot at around 9 am on 30/05/2025 near her residence. Patient had no h/o weakness, drooping of eyelid, breathing difficulty. No h/o chest pain, profuse sweating. No h/o abdominal pain, vomiting, loose stools. No h/o bleeding gums, hematuria and melena. No known co morbidities and not on regular medication .No significant past surgical history

On initial assessment, she was hemodynamically stable with heart rate of 110/min, Blood pressure of 120/70mmhg and oxygen saturation of 99% at room air with respiratory rate of 19/min. On systemic examination, no significant abnormality noted. On local examination, fang mark noted over left great toe with no active bleeding and no local reaction. On arrival, Single breath count was 32 and neck holding present. Initial VBG showed pH=7.391, pCO2= 39.8, pO2= 35.9, Na= 130, K=3.1, Creatinine=0.48, Lactate=1.4, HCO3= 23.6.On arrival whole blood clotting test done and sample clotted within 20 minutes

At around 10:30am on Day 0 in ER, patient developed dropping of both eyelids, neck holding was absent and subsequently patient went into respiratory distress for which she was non-tolerant to Non-Invasive Ventilation. Patient underwent emergency endotracheal intubation and started on Anti Snake Venom as per guideline directed protocol. Bedside Chest X-ray done and no significant abnormality noted. Patient subsequently managed in Critical care unit. She was successfully extubated on day 2 and discharged on day 4.

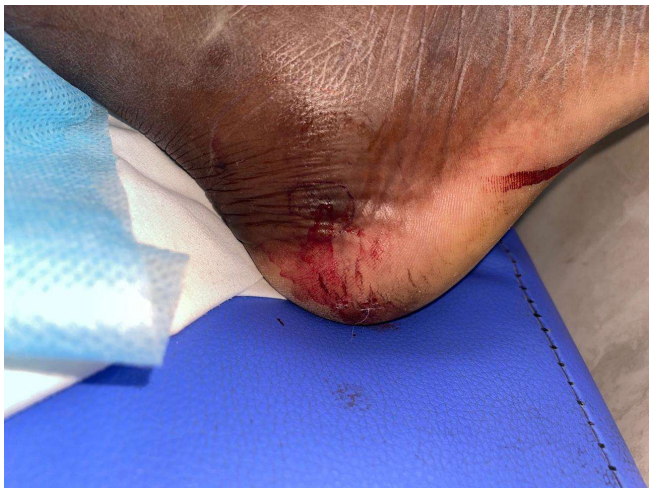
Final diagnosis: Snake bite (COBRA) - Neurotoxic with Respiratory distress

Case 2:
A 26-year-old male patient brought to ER with complaints of abdomen pain, diffuse in nature associated with multiple episodes of vomiting, blood stained and non-bilious since 9pm on 09/05/2026. Patient had H/O unknown bite to Left foot at around 7pm on 09/05/2026 near his residence. Patient had no h/o weakness, drooping of eyelid, breathing difficulty. No h/o fever, chest pain, profuse sweating. No h/o bleeding gums, hematuria and melena. Patient initially went to outside hospital and managed with IV Fluids, IV Analgesic and Tetanus prophylaxis were given. Patient referred to our hospital for further management. No known co-morbidities .No significant past surgical history. On initial assessment, he was hemodynamically stable with heart rate of 100/min, Blood pressure of 110/70mmhg and oxygen saturation of 98% at room air with respiratory rate of 22/min. On systemic examination, no significant abnormality noted. On local examination, fang mark noted below lateral malleolus of right foot with active bleeding and swelling noted. At 11pm on arrival to ER, Single breath count was 28 and neck holding present .Initial VBG showed pH=7.318, pCO2= 50, pO2= 28.5, Na= 137, K=4.0, Creatinine=1.06, Lactate=3.4, Hb= 17.2, HCO3= 23, Glucose= 140 mg/dl.On arrival whole blood clotting test done and sample not clotted within 20 minutes and

At around 12:30am on Day 0 in ER, patient passed frank hematuria of approximately 300ml, neck holding was present. Patient started on Anti Snake Venom as per guideline directed protocol. During administration of 1st ASV, patient BP dropped to 80/50mmhg with complaints

of generalized itching and chest discomfort. Hence patient started on Adrenaline infusion i/v/o anaphylactic reaction to ASV. At around 1:05am, patient developed increased oral secretions and difficulty in swallowing associated with absent neck holding. Hence, subsequently patient underwent elective endotracheal intubation I/v/o threatened airway. On Bedside Chest X-ray done and no significant abnormality noted. Patient subsequently managed in Critical care unit. On Day 2, WBCT sample clotted after administration of 3rd dose of ASV. On Day 3, swelling over the right foot reduced and He was successfully extubated on day 3 with hemodynamically stable and discharged on day 6.

Final diagnosis: Unknown bite – Mixed Neurotoxic and Hemotoxic



Case 3:

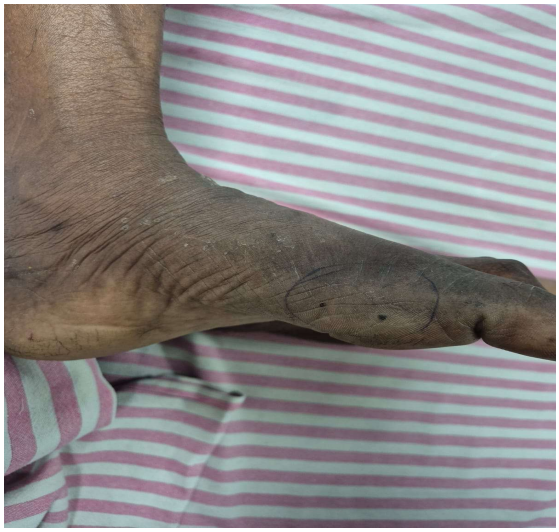
A 63-year-old male patient brought to ER with A/H/O unknown bite to Left foot at around 4 pm on 3/06/2026 near his residence. Patient c/o bleeding from the bite site associated with swelling and pain. Patient had no h/o weakness, drooping of eyelid, breathing difficulty. No h/o chest pain, profuse sweating. No h/o abdominal pain, vomiting, loose stools. No h/o bleeding gums, hematuria and melena. Patient initially went to outside hospital and managed with IV Antibiotics and Tetanus prophylaxis were given. Patient referred to our hospital for further management.

K/C/O T2DM for 12 years and not on regular medications

No significant past surgical history. On initial assessment, he was hemodynamically stable with heart rate of 78/min, Blood pressure of 160/90mmhg and oxygen saturation of 98% at room air with respiratory rate of 18/min. On systemic examination, no significant abnormality noted. On local examination, fang mark noted over medial aspect of left foot with active bleeding and swelling noted. On arrival, Single breath count was 20 and neck holding present with drooping of both eyelids noted. Initial VBG showed pH=7.340, pCO2= 33.3, pO2= 62.5, Na= 136, K=5.1, Creatinine=4.26, Lactate=2.1, Hb= 12.4, HCO3= 17.5, Glucose= 264 mg/dl.On arrival whole blood clotting test done and sample not clotted within 20 minutes and high coloured urine noted. At around 6:30pm on Day 0 in ER, patient developed dysarthria, neck holding was absent and subsequently patient underwent elective endotracheal intubation i/v/o threatened airway and started on Anti Snake Venom as per guideline directed protocol. Bedside Chest X-ray done and no significant abnormality noted. Patient subsequently managed in Critical care unit. On Day 1, Patient underwent 1st cycle of haemodialysis for refractory hyperkalaemia secondary to Rhabdomyolysis (CKMB- 210, CK Total- 6263), 4 units of FFP transfusion done in view of deranged coagulation profile and 4 units of Platelet transfusion done in view of thrombocytopenia. On Day 2, WBCT sample clotted after administration of 3rd dose of ASV. He was successfully extubated on day 5 with hemodynamically stable and improved coagulation profile and discharged on day 7. During treatment, patient underwent totally 4 cycles of Haemodialysis.

Final diagnosis: Unknown bite – Mixed Neurotoxic and Hemotoxic with Acute Kidney Injury secondary to Rhabdomyolysis.

Bite mark on left mark



High Coloured Urine

DISCUSSION:

Snake envenomation continues to be a major medical emergency in tropical countries, with clinical manifestations ranging from mild local symptoms to severe systemic complications involving respiratory failure, coagulopathy, acute kidney injury, and multiorgan dysfunction. The present case series highlights the diverse clinical spectrum of snake envenomation and demonstrates the importance of early recognition, timely anti-snake venom (ASV) administration, and aggressive supportive care in improving patient outcomes. The three cases in this series illustrate distinct patterns of envenomation: isolated neurotoxic envenomation, mixed neurotoxic-hemotoxic envenomation with antivenom-related anaphylaxis, and severe mixed envenomation complicated by acute kidney injury and rhabdomyolysis. These variations reflect the complex toxicological effects of snake venom and the unpredictable nature of clinical progression following snakebite [8].

The first case involved a cobra bite resulting in predominantly neurotoxic manifestations. Although the patient initially presented without systemic symptoms, rapid progression to bilateral ptosis, loss of neck holding, and respiratory distress occurred within a short period. This emphasizes the dynamic nature of neurotoxic envenomation, where patients may initially appear stable but deteriorate rapidly due to progressive neuromuscular paralysis. Cobra venom contains postsynaptic neurotoxins that interfere with acetylcholine receptors at the neuromuscular junction, leading to paralysis [9]. Early warning signs such as ptosis and reduced neck muscle strength are clinically important indicators of impending respiratory failure. Prompt intubation, mechanical ventilation, and ASV administration in this patient resulted in complete recovery, highlighting the effectiveness of timely intervention. Neurotoxic snakebite remains a leading cause of respiratory paralysis in endemic regions. Studies suggest that early airway management and ventilatory support significantly reduce mortality in patients with respiratory muscle weakness [10]. In the present case, rapid progression from mild symptoms to respiratory distress reinforces the need for continuous

observation even in patients who initially appear clinically stable. Monitoring tools such as single breath count and neck holding assessment are useful bedside indicators of respiratory muscle involvement.

The second case demonstrated mixed neurotoxic and hemotoxic envenomation. The patient presented with abdominal pain, vomiting, active bleeding from the bite site, and later developed frank hematuria. The prolonged 20-minute whole blood clotting test indicated venom-induced coagulopathy. Subsequently, neurological deterioration with excessive oral secretions, dysphagia, and absent neck holding suggested concurrent neurotoxicity. Mixed syndromes are increasingly recognized in snake envenomation and can present significant diagnostic and therapeutic challenges [11].A notable complication in the second case was anaphylaxis during ASV infusion, presenting with hypotension, generalized itching, and chest discomfort. Hypersensitivity reactions to ASV remain an important concern, particularly in settings where equine-derived polyvalent antivenoms are commonly used. Acute reactions may range from mild urticaria to life-threatening anaphylaxis [12]. Immediate recognition and management with adrenaline infusion in this case prevented further hemodynamic compromise and allowed continuation of treatment. This highlights the importance of close monitoring during ASV administration and readiness to manage severe allergic reactions.

The third case represented the most severe clinical presentation in this series, involving mixed neurotoxic and hemotoxic envenomation complicated by acute kidney injury and rhabdomyolysis. On arrival, the patient already demonstrated local bleeding, swelling, ptosis, coagulopathy, and significantly elevated serum creatinine. Clinical deterioration with dysarthria and respiratory compromise necessitated early airway protection. Laboratory findings and dark-coloured urine indicated severe systemic toxicity. Acute kidney injury is a well-recognized complication of severe snake envenomation and is associated with increased morbidity and mortality [13]. The mechanisms are multifactorial and include hypotension, intravascular hemolysis, direct nephrotoxicity, pigment nephropathy from rhabdomyolysis, and venom-induced coagulopathy. In this case, rhabdomyolysis likely played a major role, as evidenced by markedly elevated creatine kinase levels and refractory hyperkalaemia. Early initiation of haemodialysis contributed significantly to stabilization and recovery. Rhabdomyolysis following snakebite is an important but sometimes under recognized complication. Myotoxic venom components can cause skeletal muscle destruction, leading to release of myoglobin and intracellular electrolytes into circulation. This can precipitate acute kidney injury, metabolic disturbances, and life-threatening hyperkalaemia [14]. Early recognition of pigmenturia, electrolyte imbalance, and rising creatine kinase levels is essential for timely intervention. In severe cases, renal replacement therapy becomes necessary.

Coagulopathy was observed in both Case 2 and Case 3, emphasizing the clinical importance of monitoring coagulation parameters in snakebite victims. Venom-induced consumption coagulopathy is common in viper envenomation and can lead to significant bleeding complications [15]. In resource-limited settings, bedside WBCT remains a practical and reliable tool for monitoring coagulation status and guiding treatment response. Restoration of clotting after the third dose of ASV in both patients indicated effective venom neutralization.

An important observation across all three cases was the favourable outcome achieved through early recognition, rapid escalation of care, and multidisciplinary management. Despite severe presentations involving respiratory failure, coagulopathy, anaphylaxis, and acute kidney injury, all patients survived without major long-term complications. This reflects the critical role of timely intensive care support in modern snakebite management.

The cornerstone of successful management in severe envenomation includes rapid clinical assessment, timely ASV administration, respiratory support, management of complications, and continuous monitoring in critical care settings [16]. Early identification of high-risk features such as ptosis, respiratory distress, coagulopathy, bleeding manifestations, oliguria, or altered sensorium is essential to reduce morbidity and mortality.

This case series also underscores the importance of individualized management based on clinical presentation rather than relying solely on snake identification. In many cases, the offending snake is unknown, and treatment decisions must be guided by evolving clinical signs and laboratory findings. Syndromic management remains particularly relevant in such scenarios [17].

Overall, the cases presented here demonstrate the wide clinical spectrum of snake envenomation, from isolated neurotoxicity to severe mixed envenomation with systemic complications. They emphasize the need for early diagnosis, prompt intervention, protocol-based management, and critical care support. Strengthening awareness, improving access to healthcare, and ensuring timely availability of ASV remain essential for reducing snakebite-related morbidity and mortality.

CONCLUSION:

Snake envenomation remains a potentially life-threatening medical emergency with a wide spectrum of clinical presentations, ranging from isolated neurotoxicity to severe mixed envenomation with systemic complications. This case series highlights the diverse manifestations of snakebite, including neurotoxic respiratory paralysis, hemotoxic coagulopathy, antivenom-related anaphylaxis, acute kidney injury, and rhabdomyolysis.

The cases demonstrate that early recognition of clinical deterioration, timely administration of anti-snake venom, and prompt supportive interventions such as mechanical ventilation, hemodynamic stabilization, blood product transfusion, and renal replacement therapy are crucial in

improving outcomes. Continuous monitoring and protocol-based management in critical care settings play a vital role in preventing mortality and reducing complications. This series also emphasizes the importance of syndromic management, particularly when the offending snake species is unknown, as treatment decisions are often guided by clinical features and laboratory findings. Strengthening awareness among healthcare providers, ensuring early referral, and improving access to emergency care and antivenom remain essential for reducing snakebite-related morbidity and mortality.

A multidisciplinary approach with timely intervention can significantly improve survival and lead to favourable clinical outcomes even in severe envenomation cases.

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