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CASE REPORT OPEN ACCESS

A case of cardiac arrest induced by anti-snake venom administration after an unidentified bite

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Abstract

Background: Each year, an estimated 5.4 million snakebites occur globally, resulting in 1.8–2.7 million cases of envenoming and 81,000–138,000 deaths. Sub-Saharan Africa, Southeast Asia, and South Asia are the most affected regions, with India alone reporting nearly 45,900 snakebite-related deaths annually. Patients with clinical signs of envenomation are generally treated with an initial dose of 10 vials of polyvalent anti-snake venom (ASV), with repeat doses administered if symptoms persist. Whole blood clotting time (WBCT 20) is used to monitor treatment response.

Case presentation: We report the case of a 57-year-old female who presented with a history suggestive of snakebite envenomation. Approximately one hour after hospital admission, she developed sudden cardiac arrest. Cardiopulmonary resuscitation (CPR) was initiated, resulting in return of spontaneous circulation (ROSC). She was shifted to the intensive care unit (ICU) and started on dobutamine and furosemide infusions. A 2D echocardiogram revealed regional wall motion abnormalities (RWMA) involving the LAD, RCA, and LCX territories, with a left ventricular ejection fraction (LVEF) of 35%. Laboratory findings showed leukocytosis (WBC count 14,300/mm^3) and elevated cardiac biomarkers. Cardiology consultation was sought, and the patient was managed with norepinephrine, dobutamine, vasopressin infusion, diuretics, and guideline-directed medical therapy (GDMT), following which she gradually stabilized.

Conclusion: This case highlights a rare presentation of cardiac arrest following ASV administration, likely due to severe anaphylaxis. Early recognition of envenomation, prompt initiation of ASV therapy, close monitoring for complications, and timely resuscitative efforts are critical in improving outcomes in snakebite victims.

Keywords: unidentified bite; ASV administration; cardiac arrest; guideline-directed medical therapy; recovery

Introduction

Snakebite envenoming remains a significant public health problem, with more than 100,000 deaths and approximately 400,000 cases of serious disabilities or disfigurements reported annually worldwide [1]. South Asia is the most affected region, with India alone accounting for nearly half of global snakebite-related deaths [2, 3]. The highest mortality rates in India are reported from the states of Uttar Pradesh, Andhra Pradesh, Bihar, Tamil Nadu, West Bengal, and Maharashtra [4].

Snakebite envenoming primarily affects rural populations in tropical and subtropical regions, where farming and agriculture increase the risk of contact with snakes [5]. In recognition of the burden, the World Health Organization (WHO) has prioritized snakebite envenoming as a neglected tropical disease and set a goal in 2019 to reduce deaths and disabilities by 50% by the year 2030 [6].

Cardiac involvement following snakebite may occur either directly (due to cardiotoxic venom] or indirectly (secondary to shock, hypoxia, or electrolyte

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disturbances). It is most commonly associated with viper bites [7]. Cardiac sequelae have also been reported following bites from the hump-nosed viper (*Hypnale hypnale*), whose venom is not neutralized by the currently available polyvalent anti-snake venom (ASV) [8].

Diagnosis of systemic envenomation is often clinical, based on symptoms such as spontaneous bleeding, neurotoxic signs, prolonged clotting time, or acute kidney injury. Laboratory tests, including blood coagulation profile, renal and liver function tests, are useful adjuncts [9]. In resource-limited settings, the 20-minute whole blood clotting time (WBCT20) is a simple, bedside test widely used to detect coagulopathy and guide ASV therapy [10].

ASV remains the mainstay of treatment for systemic envenomation and severe local envenoming involving more than half of a limb [11]. However, ASV administration carries the risk of adverse reactions, with more than 10% of patients experiencing mild to severe hypersensitivity reactions, including potentially life-threatening anaphylaxis [12].

The objective of this case report is to describe a rare presentation of cardiac arrest following ASV administration, likely due to anaphylaxis, and to highlight the importance of early recognition, prompt management, and monitoring for cardiovascular complications in snakebite victims.

Case Presentation

A 57-year-old woman was admitted to the emergency department, KIMS hospital, Secunderabad with complaints of dizziness and blurred vision. On arrival, her blood pressure was not recordable. She was immediately started on norepinephrine (noradrenaline) infusion and shifted to the intensive care unit (ICU).

On further history-taking, it was revealed that she had sustained an unknown bite at her hometown, for which she had received anti-snake venom (ASV) at a nearby clinic. Approximately one hour after hospital admission, she developed cardiac arrest. Cardiopulmonary resuscitation (CPR) was performed, resulting in return of spontaneous circulation (ROSC), which is defined as the resumption of a sustained perfusing heart rhythm following cardiac arrest.

In the ICU, the patient was started on dobutamine and furosemide (Lasix) infusions. Initial laboratory investigations revealed hemoglobin 14.5 g/dL, leukocytosis (WBC count: 14,300/mm^3), and elevated cardiac biomarkers. Cardiology consultation was sought, and treatment was continued with norepinephrine, dobutamine, vasopressin infusion, and diuretics. A 2D echocardiogram showed regional wall motion abnormalities (RWMA) in the LAD, RCA, and LCX territories, with a left ventricular ejection fraction (LVEF) of 35% (Figure 1).

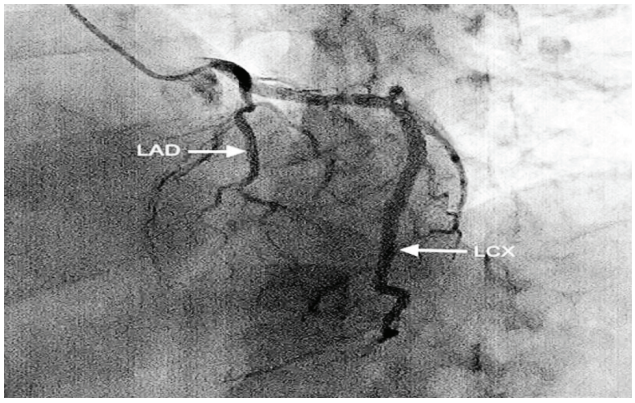

Figure 1: Rudimentary LAD, which is seen to end abruptly. A rudimentary LAD is smaller than normal and may not supply the usual amount of blood to the anterior wall of the heart.

For reference, a normal LVEF is above 50%. In this patient, the LVEF was significantly reduced, indicating heart failure with reduced ejection fraction (HFrEF).

Gradually, inotropic support was tapered, and guideline-directed medical therapy (GDMT) for heart failure was initiated. The patient's hemodynamic status improved, and she was shifted to the ward. Blood pressure remained stable, and there was no shortness of breath or orthopnea. Blood cultures grew *Acinetobacter baumannii*, prompting a change in antibiotics to intravenous tigecycline. She experienced one episode of asymptomatic hypotension, for which GDMT drugs were titrated appropriately. The patient improved gradually and was discharged with advice for outpatient follow-up.

Discussion

Snakebite envenoming is a common yet neglected public health problem in tropical countries and may present with a wide spectrum of clinical manifestations depending on the species involved. Neurotoxic symptoms such as ptosis, dysphagia, dysarthria, facial paralysis, and respiratory distress are frequently associated with elapid bites. In South India, *Daboia russelii* (Russell's viper) bites are the most common cause of morbidity and mortality related to snakebite, often resulting in coagulopathy, acute kidney injury, and multi-organ dysfunction [13].

Snake venom can have direct cardiotoxic effects through disruption of several physiological systems, including

the renin-angiotensin-aldosterone and bradykinin pathways, resulting in hypotension and cardiovascular collapse. Additionally, antivenom itself can occasionally precipitate hypersensitivity reactions ranging from mild urticaria to severe anaphylaxis, which can be life-threatening if not promptly recognized and managed [14].

Electrolyte disturbances, particularly hyperkalemia secondary to rhabdomyolysis, can precipitate fatal arrhythmias and cardiogenic shock. Severe hyperkalemia may develop within hours of envenomation, especially in cases with significant myonecrosis, contributing to cardiac arrest [15]. Cardiac involvement in snakebite is not frequently reported but has been described mainly in viper envenomation. The clinical spectrum ranges from myocarditis and arrhythmias to acute myocardial infarction and cardiogenic shock [16, 17]. Most snake venom components do not cross the blood–brain barrier, and neurological manifestations are largely peripheral, although severe systemic effects such as hypoxia, shock, or cardiac arrest can result in secondary brain injury.

The administration of ASV should always be performed in a monitored setting, preferably in an intensive care or high-dependency unit. The WHO recommends premedication with intramuscular adrenaline in high-risk patients to reduce the incidence of severe anaphylaxis [18].

In the present case, the patient sustained an unknown bite, received ASV, and developed cardiac arrest approximately one hour after hospital admission. She was promptly resuscitated, which resulted in return of spontaneous circulation and good neurological recovery. Subsequent investigations revealed elevated cardiac markers and echocardiographic evidence of global myocardial dysfunction with reduced ejection fraction, consistent with acute myocardial injury. Early institution of inotropic support, diuretics, and GDMT resulted in gradual hemodynamic stabilization. Blood cultures grew *Acinetobacter baumannii*, necessitating modification of antibiotic therapy to tigecycline. The patient ultimately made a good recovery and was discharged with advice for follow-up.

Our case underscores the importance of early recognition and aggressive management of cardiovascular complications following snakebite envenomation or ASV administration. Physicians should maintain a high index of suspicion for cardiac involvement, particularly in patients with hypotension, arrhythmias, or elevated cardiac biomarkers. Close hemodynamic monitoring and prompt initiation of appropriate resuscitative measures are crucial for improving patient outcomes.

Conclusion

Early recognition and prompt management of snakebite envenomation are essential to prevent morbidity and mortality. Even without a confirmed history of snakebite, patients presenting with sudden neurological symptoms or hemodynamic instability should be evaluated for envenomation. Intravenous polyvalent ASV must be administered promptly when systemic signs are present, with close monitoring for potential adverse reactions, including anaphylaxis. Supportive care, including airway management, inotropic support, and treatment of complications such as coagulopathy, is critical. In this case, the patient developed cardiac arrest likely due to ASV-induced anaphylaxis but recovered following timely resuscitation and intensive care. Vigilance and preparedness for such complications can significantly improve outcomes.

Conflicts of interest

Authors declare no conflicts of interest.

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