Case Report

Russell's Viper Envenomation Complicated by Venom-Induced Consumption Coagulopathy, Myocardial Infarction, and Ischemic Stroke: A Rare Case Report

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Abstract

Background: Snake envenomation is a major medical emergency in tropical regions. Russell's viper envenomation commonly causes venom-induced consumption coagulopathy (VICC), which may lead to severe systemic complications. Although myocardial infarction or ischemic stroke has been reported individually following snakebite, their simultaneous occurrence in the same patient is exceedingly rare. **Case Presentation:** A 46-year-old woman presented one hour after a suspected Russell's viper bite with tachycardia, hypoxemia, reduced Glasgow Coma Scale score, and prolonged whole blood clotting time, consistent with VICC. She required endotracheal intubation and mechanical ventilation due to respiratory compromise, with bloody endotracheal secretions noted on suctioning. She presented with AMI during her hospitalisation, which was confirmed by electrocardiographic changes, with an increase in cardiac biomarkers. Later she had left upper limb weakness and a neuro-image showed an acute ischemic stroke. Anti-snake venom therapy, supportive care and a multidisciplinary management was given in time. She recovered with residual focal neurological deficit. **Conclusion:** The case demonstrates the rare occurrence of simultaneous acute myocardial infarction and ischemic stroke after envenomation by Russell's viper. These life-threatening complications may be due to various factors such as VICC, endothelial injury and venom-induced vascular thrombosis. Timely and multidisciplinary management and close cardiovascular and neurological monitoring are crucial for optimizing patient outcomes, while early recognition is necessary.

Keywords: Snake envenomation, Russell's Viper, Venom-induced consumption coagulopathy, Myocardial infarction; Ischemic stroke.

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Introduction

Envenomation by snakes is a serious public health problem in tropical areas, especially in Africa, Southeast Asia and parts of Latin America, where the Russell's viper Daboia russelii is a major cause of morbidity and mortality because of the very potent envenomation causing hemotoxic type of venom.[1-5] The venom incorporates strong procoagulant factors, which can lead to venom-induced consumption coagulopathy (VICC) with a rapid consumption of the clotting factors causing a delayed clotting time and a mainly bleeding presentation.[6]

In most instances, bleeding is the first appearance of VICC, but there are reports of paradoxical thrombotic events. A number of cases have described acute myocardial infarction (AMI) post Russell's viper bite, probably secondary to coronary thrombosis, vasospasm or direct action of the venom toxin on the endothelial cells.[1,8] Similarly, ischemic strokes—affecting medium- to large-vessel territories—have occurred post-envenomation, attributed to disseminated coagulopathy and toxin-mediated vascular damage.[10,12]

To date, concurrent occurrence of both MI and ischemic stroke following Russell's viper envenomation appears to be unreported and exceedingly rare. We describe a novel case of Russell's viper bite complicated by VICC, acute myocardial infarction, and ischemic stroke, providing insight into the rare but devastating thrombo-haemorrhagic spectrum of hemotoxic snake envenomation.

Case Presentation

A 46-year-old female agricultural worker was brought to the emergency department by her son in a disoriented state following a snakebite sustained over the right ankle at approximately 9:00 AM while working in agricultural fields at Deetupally. Soon after the bite, she developed giddiness that rapidly progressed to loss of consciousness. There was no

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history suggestive of neurotoxic envenomation, including ptosis, dysphagia, dysarthria, diplopia, dysphonia, limb weakness, or respiratory distress. She also denied vomiting or fever. Her medical history was unremarkable, with no known comorbidities, addictions, or previous cardiovascular or cerebrovascular disease.

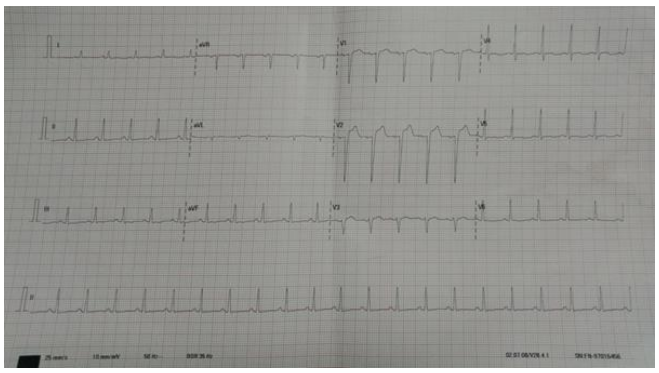
On arrival, she was critically ill with a Glasgow Coma Scale (GCS) score of E2V2M4. Her pulse rate was 120 beats/min, blood pressure was 110/80 mmHg, and oxygen saturation was 40% on room air, improving only to 60% despite administration of oxygen at 15 L/min through a non-rebreathing mask. Owing to persistent hypoxemia and depressed sensorium, she underwent endotracheal intubation and mechanical ventilation.

Examination revealed swelling at the bite site over the right ankle. Endotracheal suctioning yielded blood-stained secretions, and continuous bleeding was observed from venous catheter insertion sites, indicating severe coagulopathy. The 20-minute whole blood clotting test (WBCT20) was grossly abnormal, with failure of blood to clot after 20 minutes.

The patient was treated with an initial dose of 10 vials of polyvalent anti-snake venom (ASV) diluted in 500 mL of normal saline and infused over one hour. During the infusion, she developed an anaphylactic reaction manifested by hypotension, which was managed with intravenous pheniramine (Avil), hydrocortisone (100 mg), and two intramuscular doses of adrenaline (0.5 mg of 1:1000 solution). Following stabilization, ASV therapy was continued. Because the WBCT20 remained persistently prolonged, an additional 18 vials were administered in three divided doses of six vials each, bringing the total ASV dose to 28 vials.

Initial laboratory investigations demonstrated severe venom-induced consumption coagulopathy with plasma fibrinogen <80 mg/dL, prothrombin time (PT) of 600 seconds, international normalized ratio (INR) of 70, markedly elevated D-dimer levels (>100,000 µg/L), and elevated cardiac troponin I (0.80 ng/mL). In view of persistent bleeding and profound coagulation abnormalities, she received four units of fresh frozen plasma (FFP) and one unit of cryoprecipitate. Nine hours after admission, the WBCT20 normalized, and subsequent clotting tests remained within normal limits.

The admission electrocardiogram (ECG) showed sinus tachycardia. On the following day, repeat ECG demonstrated ST-segment elevation in leads V1–V3. Two-dimensional echocardiography revealed regional wall motion abnormalities in the left anterior descending (LAD) coronary artery territory with moderate left ventricular systolic dysfunction (left ventricular ejection fraction, 36%). Based on these findings, a diagnosis of acute anterior wall myocardial infarction complicating hemotoxic snake envenomation was established. After careful assessment of the bleeding risk and improvement of the coagulopathy, loading doses of dual antiplatelet therapy were initiated.



The next day during the process of weaning trial from mechanical ventilation the patient was noted to have no movement of the left upper limb. Non-contrast CT brain revealed acute infarcts in the right fronto-parietal and parieto-occipital regions.Appropriate medical management was initiated in view of acute coronary syndrome, coagulopathy secondary to snake bite, and acute ischemic stroke.

Low molecular weight Heparin along with Dual antiplatelet therapy was initiated. Multidisciplinary consultations were obtained from Cardiology, Neurology, Surgery teams, and their recommendations were followed.

The patient showed gradual symptomatic improvement during hospitalization. She was successfully extubated, maintained stable hemodynamics, and was closely monitored throughout her hospital stay. At discharge, she had improved significantly and was in a hemodynamically stable state. Patient was advised coronary angiogram on follow up and discharged with dual antiplatelet therapy.

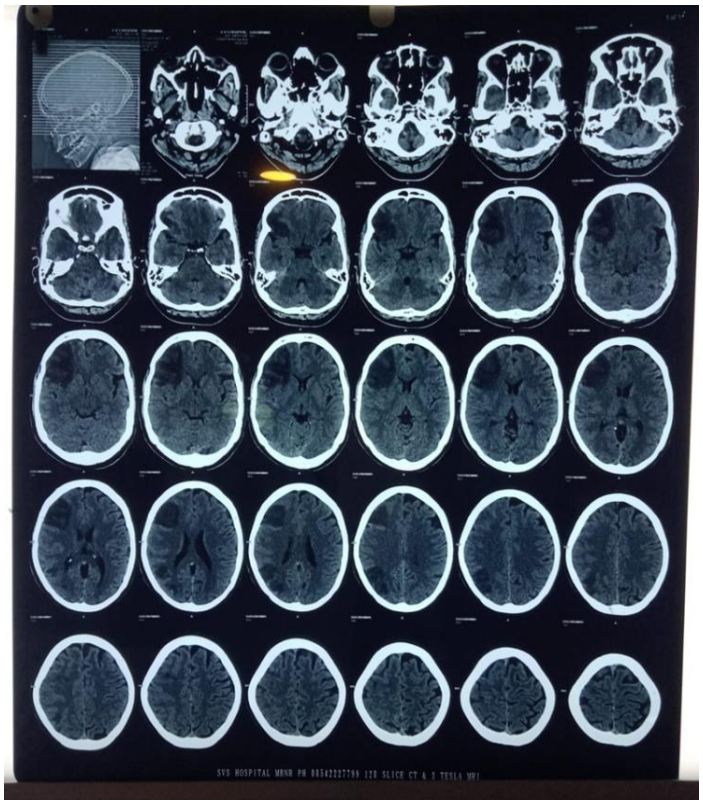


Table 1: Coagulation profile & troponin i levels. (TIMELINE- 3/8/2025 to 11/8/2025)

Date	D-Dimer (µg/L)	Troponin-I (ng/ml)	Fibrinogen plasma (mg/dl)	PT (sec)	INR	APTT (sec)
3/8	>1 lakh	0.8	<80	600	70.8	30.2
4/8	None	None	None	28.8	1.66	23.4
5/8	None	3.1-2.2→1.7	77.8	19.8	1.5-1.56	31.0
6/8	None	None	None	20.3-20.2	None	29.3
11/8	None	0.035	None	None	None	None

Table 2: Hemogram reports -3/8/2025-12/8/2025

Date	Hb (g/dl)	RBC (millions/cu mm)	WBC (cells/cu mm)	PCV/HCT (%)	MCV (fl)	MCH (pg)	MCHC (g/dl)	Platelet (lakh/cu mm)
3/8	13.5	4.6	18100	42.0	90.2	29.3	32.9	0.9
4/8	13.2	4.5	24700	40.5	92.0	29.3	32.9	1.0
5/8	13.1	4.3	24200	40.5	90.9	30.4	32.2	1.3
6/8	12.4	4.1	19500	37.6	90.0	30.2	33.3	1.6
9/8	11.2	4.2	17200	35.0	91.4	30.0	33.1	2.8
12/8	12.1	4.0	10200	36.6	90.3	30.0	33.2	3.1

Discussion

Russell's viper envenomation is a major cause of severe hemotoxic snakebite in South Asia and is frequently associated with venom-induced consumption coagulopathy (VICC), characterized by rapid depletion of coagulation factors, hypofibrinogenemia, prolonged clotting times, and secondary fibrinolysis.[6,7] More recently, however, an increasing number of cases of VICC have been reported to coexist with subsequent thrombotic events such as AMI and ischemic stroke, and bleeding manifestations were more common than thrombotic phenomena, suggesting a complex procoagulant effect of venom.[10,12] In our patient, the severe coagulopathy, elevated D-dimer levels, prolonged PT/INR, and hypofibrinogenemia were consistent with VICC; after bleeding, she experienced both AMI and ischemic stroke.

The exact mechanism of arterial thrombosis after Russell's viper envenomation is still not fully understood and may be multifactorial. These can activate factors V, X, and prothrombin, creating widespread generation of thrombin and intravascular formation of fibrin, which ultimately leads to consumption of coagulation factors and bleeding, but can also result in localized thrombus formation before coagulation factors are depleted. During early acute envenomation or post-anaphylaxis from anti-snake venom, there could be episodes of hypotension, which could further exacerbate vasoconstriction in susceptible areas of the vascular bed. In susceptible vascular territories, early episodes of hypotension during severe envenomation or following anti-snake venom anaphylaxis can further compromise tissue ischemia. The presence of both mechanisms could account for the patient's coexistence of both hemorrhagic and thrombotic symptoms.

Hemotoxic snake bite has been associated with ischemic stroke in a few isolated case reports, which is considered as one of the less frequently observed phenomena of the bite of the Russell's viper, occurring simultaneously with AMI has not been reported.The simultaneous occurrence of AMI and ischemic stroke following a bite of a Russell's viper is exceptionally rare, and the pathogenesis of AMI remains unclear; however, possible mechanisms include cerebral vasospasm, embolic phenomena, endothelial injury, and hypercoagulable state. This work, as best we know, is unpublished and is therefore unique and a useful addition to the literature.

Thrombotic complications are a therapeutic challenge in VICC. Antiplatelet agents and anticoagulants should not be used in active coagulopathy as they can cause life-threatening bleeding, but they are recommended in acute coronary syndrome and ischemic stroke. Hence, correction of coagulopathy before administering anti-snake venom, if necessary, and the use of blood component therapy, was the foundation for treatment in our patient; antithrombotic therapy was only started after the coagulation parameters were normalised and the balance between the thrombotic and hemorrhagic risks was carefully evaluated. The active involvement of emergency physicians, intensivists, cardiologists, neurologists, and hematologists played a crucial role in clinical decision-making and decision outcomes.

In this case the wide spectrum of thrombohemorrhagic involvement of Russell's viper envenomation is demonstrated and the fact that severe coagulopathy does not preclude arterial thrombosis is emphasized. Patients with hemotoxic snake bite should be evaluated early for cerebrovascular and cardiovascular complications when there are any new onset neurological deficits, electrocardiographic abnormalities or elevated cardiac biomarkers. Early diagnosis, continuing neurological and cardiac monitoring, correction of coagulopathy, and personalized, multidisciplinary treatment are all essential to better outcomes in survival and functional outcomes.

Conclusion

Russell's viper envenomation can manifest with a wide range of thrombohemorrhagic disorder that can be more or less severe and may encompass the classic symptoms of venom-induced consumption coagulopathy. In this case, the authors demonstrate the rare overlap of acute anterior wall myocardial infarction and ischemic stroke in the setting of severe VICC in the paradoxical context of hemotoxic snake envenomation. It is important to have a high level of clinical suspicion for cardiovascular and cerebrovascular complications in patients with new neurologic deficits, electrocardiographic changes, or

elevation of cardiac biomarkers despite apparent correction of coagulopathy. Promptly applying anti-snake venom to intervals of coagulation abnormalities and timely correction; active monitoring and individualized multidisciplinary management are crucial to achieve the best results. Reporting these rare presentations adds to the body of knowledge on the clinical spectrum of Russell's viper envenomation, and can help to ensure prompt diagnosis and treatment in any future occurrences.

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Conflicts of interest
There are no conflicts of interest.

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