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**Case Series**

# Non-hemorrhagic stroke following venomous snakebite: a case series

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## ABSTRACT

Snakebite envenomation remains a major public health issue in India, causing nearly 50,000 deaths annually. Hemotoxic venoms, particularly from viper bites, are well recognized for inducing coagulopathy and hemorrhagic complications; however, ischemic stroke following envenomation is a rare but important clinical entity. We present a case series of three patients who developed non-hemorrhagic ischemic strokes after Russell's viper bites. All three had evidence of systemic envenomation with prolonged clotting times and received prompt treatment with anti-snake venom and fresh frozen plasma. Despite correction of coagulopathy, each developed focal neurological deficits. Neuroimaging revealed acute infarcts: right corona radiata infarct in Case 1, left middle cerebral artery territory infarction in Case 2, and multiple embolic infarcts in Case 3. All patients were managed with antiplatelet therapy, neuroprotective agents, and supportive care, with gradual neurological improvement. This series highlights that neurological deterioration after snakebite is not always attributable to neurotoxic effects or intracranial hemorrhage and should prompt evaluation for ischemic stroke. Early recognition, timely neuroimaging, and institution of stroke-specific management are critical in improving outcomes. Clinicians should maintain a high index of suspicion for ischemic stroke in snakebite victims presenting with new-onset focal deficits, as prompt intervention may prevent long-term morbidity and disability.

**Keywords:** Non-hemorrhagic stroke, Venomous snakebite, Snakebite deaths

## INTRODUCTION

Snakebite deaths are very common in India, with approximately fifty thousand deaths occurring annually due to snakebites. Snake venoms are mainly of two types neurotoxic and hemotoxic. Neurotoxic venom inhibits the conduction of nerve impulses by degrading neurotransmitters and depolarizing the axonal membrane. Hemotoxic venom causes tissue destruction and affects the circulatory system. Post-snakebite ischemic stroke is a rare but serious condition that can occur after a venomous snakebite. In this report, we present a series of non-haemorrhagic strokes that developed following snake envenomation.

## CASE SERIES

### *Case 1*

A 38 years old female was brought to the ER with a history of a Russell's viper bite on her right big toe. On examination, she was stuporous and the bite site was swollen with active bleeding, suggesting local envenomation. Initial investigations revealed prolonged whole blood clotting time (WBCT>20 minutes), prolonged prothrombin time and an elevated INR, indicating systemic envenomation with coagulopathy. Based on these findings, anti-snake venom was administered in a dose of 20 vials intravenously, followed by 7 units of fresh frozen plasma, which corrected the

coagulopathy. During observation, the patient developed left upper limb weakness, prompting an urgent CT brain to rule out an intracranial bleed, which was found to be normal. Over time, her sensorium improved, but she had persistent left-sided weakness, raising concerns of a neurological complication. MRI brain revealed an acute infarct in the right corona radiata. The patient was managed according to stroke protocol, including antiplatelet therapy and supportive care.

***Case 2***

A 56 years old female presented to the emergency department with history of a snake bite on her right wrist while working in a field. The patient arrived at the emergency room approximately two hours after the bite, complaining of severe redness, progressive swelling at the bite site and dizziness. Upon initial evaluation, she was found to have tachycardia (heart rate of 110 bpm) and hypotension (BP: 80/50 mmHg), along with local tenderness and ecchymosis at the site of the bite. Blood investigations revealed prolonged clotting time and elevated prothrombin time, suggestive of hemotoxic snake bite. The patient was immediately administered anti-snake venom along with intravenous fluids and inotropic support to stabilize her hemodynamic. The coagulopathy was corrected with the treatment.

On the third day of hospitalization, the patient developed sudden onset weakness of the right upper and lower limbs, along with facial asymmetry. Neurological examination revealed right-sided hemiparesis. An MRI brain with diffusion-weighted imaging revealed an acute infarct in the left middle cerebral artery territory, indicative of an ischemic stroke. The patient was immediately started on antiplatelet therapy, neuroprotective agents and supportive measures. Adequate hydration and close monitoring of coagulation parameters were continued. Over the following days, the patient showed gradual improvement in limb movements and was transitioned to physiotherapy for motor rehabilitation. She remained hemodynamically stable and was eventually discharged with advice for regular follow-up.

***Case 3***

A 64-year-old male was referred from a local hospital with a history of snake bite on the right big toe while walking barefoot in a rural area. The patient reported immediate pain and swelling at the bite site, followed by dizziness and generalized weakness. He arrived at the emergency department approximately three hours post-bite with complaints of nausea, sweating and altered sensorium. On initial evaluation, he was found to be hypotensive (BP: 84/56 mmHg),had tachycardia (HR: 115 bpm) and he had a poor Glasgow Coma Scale score of 7/15 (E2V2M3), necessitating immediate intubation for airway protection. Physical examination revealed severe edema and local tenderness at the bite site and multiple ecchymoses at venepuncture sites.

Laboratory investigations revealed prolonged whole blood clotting test, prolonged prothrombin time (PT Test: 22 sec), international normalized ratio (INR: 2.5) and activated partial thromboplastin time (APTT: 45 sec), indicating venom-induced coagulopathy. Additionally, serum fibrinogen levels were low and D-dimer levels were elevated, suggestive of disseminated intravascular coagulation. In view of these findings, 20 vials of anti-snake venom was administered, along with intravenous fluids and inotropic support for hemodynamic stabilization. Despite ASV therapy, the patient developed worsening coagulopathy with spontaneous bleeding manifestations, including multiple hematomas over the body and haematuria. 10 units of fresh frozen plasma was transfused to correct coagulation abnormalities. Supportive management included broad-spectrum antibiotics and meticulous wound care.

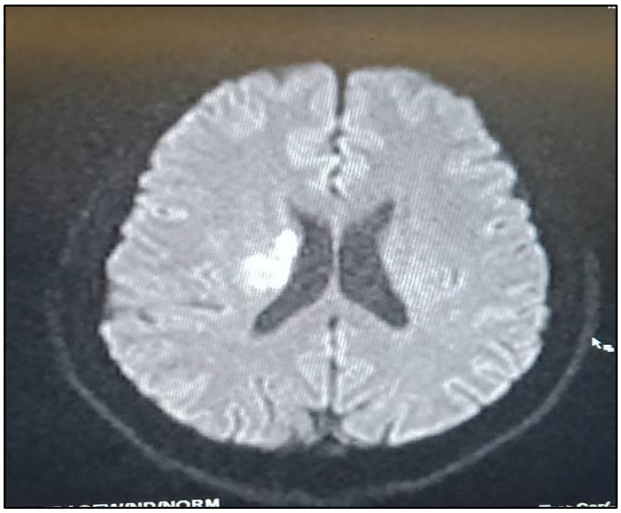

**Figure 1: MRI brain diffusion weighted imaging (DWI) Acute infarct in right corona radiata.**

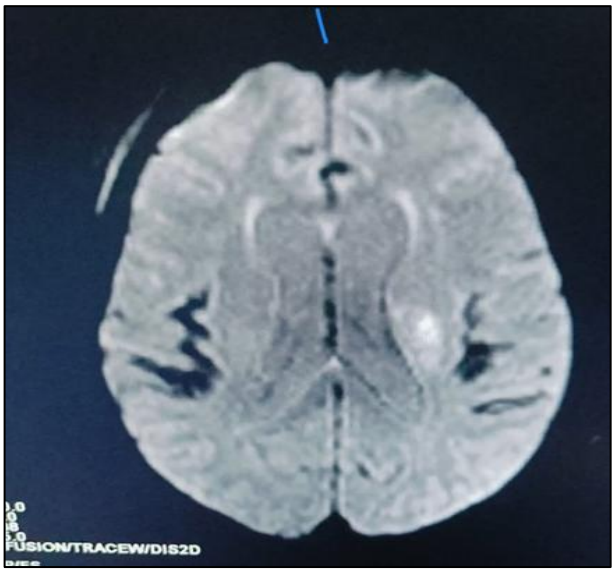


**Figure 2: MRI brain DWI Acute infarct over left MCA territory.**

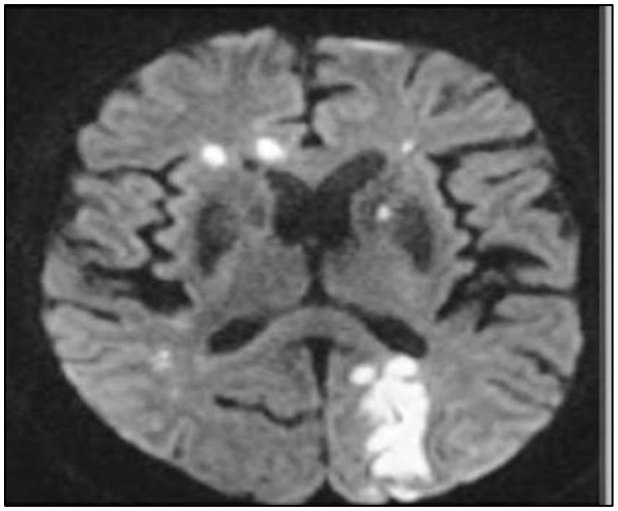

**Figure 3: MRI brain DWI embolic infarcts.**

While in the ICU, the patient remained critically ill with persistent ventilator dependence. An attempt at weaning and extubation was unsuccessful due to poor respiratory effort and fluctuating consciousness. Neurological assessment raised suspicion of central involvement, prompting an MRI brain, which revealed multiple embolic infarcts. Stroke management was initiated, including antiplatelet therapy, neuroprotective agents and aggressive supportive care. Given the prolonged ventilator requirement, a tracheostomy was performed, following which the patient was successfully weaned off mechanical ventilation.

## DISCUSSION

Snakebite envenomation remains a significant public health concern in India, contributing to an estimated 50,000 deaths annually. While hemorrhagic complications are well-documented sequelae of hemotoxic snakebites, non-hemorrhagic neurological manifestations particularly ischemic strokes are relatively rare and underreported. The recognition of cerebral infarction in this context is critical, as it challenges conventional expectations that neurological deterioration following snakebite is solely due to neurotoxicity or intracranial bleeding.

This study is important because it highlights ischemic stroke as a potential and serious complication of snake envenomation, particularly with viper bites. The findings underscore the need for clinicians to maintain a high index of suspicion for ischemic events in snakebite patients presenting with altered sensorium or focal neurological deficits. Timely neuroimaging and stroke-specific interventions can significantly influence outcomes. By documenting a series of such cases, this study aims to expand clinical awareness of this underrecognized phenomenon and emphasizes the need for multidisciplinary vigilance and early intervention in managing snakebite victims with neurological symptoms.

In all three reported cases of ischemic stroke following venomous snakebite, timely intervention and multidisciplinary care were crucial for favorable outcomes. Each patient received prompt administration of anti-snake venom , with doses tailored to clinical severity. Fresh frozen plasma was administered where coagulopathy was evident. In first case, a 38 years old female developed left-sided weakness after snake bite; MRI revealed an acute right corona radiata infarct. She was managed with ASV, FFP, antiplatelets, statins and physiotherapy. In second case, a 56 years old female, developed left middle cerebral artery infarction on day three. She was treated with ASV, inotropes, statins and antiplatelets. Third case involved a 64 years old male with severe systemic involvement. Coagulopathy was corrected with ASV and other supportive measures. However, it was difficult to wean him off the ventilator. MRI brain showed embolic infarcts and he was started on antiplatelets and statins. He underwent tracheostomy and was gradually weaned off the ventilator. All patients improved with supportive therapy, emphasizing the role of early stroke recognition, antiplatelet therapy and neurorehabilitation in post-envenomation stroke management. The development of ischemic stroke in snakebite envenomation is driven by several interrelated mechanisms. Haemostatic abnormalities, collectively termed snake venom-induced coagulopathy, are a major contributor. Many venoms, particularly from viperid species, contain procoagulant enzymes (e.g., thrombin-like enzymes, factor V and X activators) that trigger a DIC-like state resulting in both hemorrhagic tendencies and microthrombus formation, which can obstruct cerebral vessels.[1,2]

Simultaneously, venom metalloproteinases degrade vascular basement membranes, cause direct endothelial injury and induce vasculitis, all of which promote localized thrombosis.[3,4] Systemic hypotension from hypovolemia, shock or autonomic instability further compromises cerebral perfusion, predisposing to watershed infarcts.[5,6] Additionally, certain venoms act as potent procoagulants, leading to hypercoagulability and fibrin deposition in small vessels.[7] Immune-mediated mechanisms such as anaphylaxis to venom or antivenom may result in inflammatory vasculitis and secondary thrombosis.[8]

Secondary complications like rhabdomyolysis, acute kidney injury and sepsis also contribute to a prothrombotic state through metabolic derangements and hypercoagulation. Other factors include venom-induced vascular spasm, increased vascular permeability, microthrombi, haemoconcentration, hyper viscosity and venom-related cardiac toxicity leading to arrhythmias and embolic stroke.[9,10] Together, these processes demonstrate the complex, multifaceted pathophysiology of ischemic stroke following snakebite and underscore the importance of early neurological assessment and supportive care in at-risk patients. Studies regarding the initiation of secondary prophylaxis in ischemic stroke following venomous snakebite remain exceedingly scarce, with evidence

limited to a handful of case reports and small case series. Neither clinical trials nor consensus guidelines exist to guide secondary prevention in snake venom-triggered ischemic stroke, As such, secondary prophylaxis remains empirical, varies case by case and underscores the urgent need for systematic observational studies and trials to define optimal timing, agent selection and safety monitoring.

## CONCLUSION

This case series highlights the need for a high index of suspicion for ischemic stroke following hemotoxic snake bites. While haemorrhage is the typical presentation, the possibility of ischemic stroke should also be considered in patients presenting with neurological deficits. Early imaging and timely intervention can help prevent irreversible neurological damage. This series underscores the importance of multidisciplinary care, the judicious use of antiplatelets and the role of early imaging in achieving favourable outcomes.

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## REFERENCES

1. Je L, Ae F, De K. Harrison's principles of internal medicine; 2022.
2. Tednes M, Slesinger TL. Evaluation and treatment of snake envenomations. In StatPearls; 2024.
3. Silvestrelli GG. The Interest in the Pathology and Pathophysiology of Vascular Lesions; 2017.
4. Panicker JN, Madhusudanan S. Cerebral infarction in a young male following viper envenomation. J Assoc Phys. 2000;48:744–5.
5. Kay E. A history of herbalism: Cure, cook and conjure; 2022.
6. Thomas L, Tyburn B, Ketterlé J, Biao T, Mehdaoui H, Moravie V, et al. Prognostic significance of clinical grading of patients envenomed by Bothrops lanceolatus in Martinique. Trans R Soc Trop Med Hyg. 1998;92:542–5.
7. Kaufman DM, Geyer HL, Milstein MJ, Rosengard J. Kaufman's Clinical Neurology for Psychiatrists-E-Book. Elsevier Health Sciences; 2022.
8. Brief HP. Post Category. Emergency Medicine; 2020.
9. Wing EJ, Schiffman FJ. Cecil Essentials of Medicine E-Book: Cecil Essentials of Medicine E-Book. Elsevier Health Sciences; 2021.
10. Boviatsis EJ, Kouyialis AT, Papatheodorou G, Gavra M, Korfias S, Sakas DE. Multiple hemorrhagic brain infarcts after viper envenomation. Am J Trop Med Hyg. 2003;68:253–7.
11. Paul G, Paul BS, Puri S. Snake bite and stroke: Our experience of two cases. Indian J Crit Care Med. 2021;2:87-9.
12. Dabilgou AA, Sondo A, Dravé A, Diallo I, Kyelem JMA, Napon C, et al. Hemorrhagic stroke following snake bite in Burkina Faso (West Africa): A case series. Trop Dis Travel Med Vaccines. 2021;7:25.
13. Ansoumane H, Dabilgou A, Ladgani A, Nascimento E, Pérez-Gómez F, Sachett J, et al. Snake venom and cerebrovascular events: insights and public health implications. PLoS Negl Trop Dis. 2024;18:6729.
14. Ghosh S, Lahiri D, Martínez-Villota A, Namal Rathnayaka RM. Cerebrovascular events induced by venomous snake bites. Heliyon. 2025;11:856.
15. Vasconez-Gonzalez J, Delgado-Moreira K, Izquierdo-Condoy JS, de Lourdes Noboa-Lasso M, Gamez-Rivera E, Lopez-Molina MB, et al. Cerebrovascular events induced by venomous snake bites: A systematic review. Heliyon; 2025.
16. Delgado ABT, Gondim CCVL, Reichert LP, da Silva PHV, Souza RMDCE, Fernandes TMP, et al. Hemorrhagic stroke secondary to Bothrops spp. venom: a case report. Toxicon. 2017;132:6–8.
17. Pérez-Gómez AS, Monteiro WM, João GAP, Sousa JDB, Safe IP, Damian MM, et al. Hemorrhagic stroke following viper bites and delayed antivenom administration: three case reports from the Western Brazilian Amazon. Rev Soc Bras Med Trop. 2019;18:52.

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