

## CASE SERIES

# Acute Ischemic Stroke Associated with Snake Envenomation: A Reversible Cerebrovascular Event

VEERASIVABALAN S<sup>1,\*</sup>, JACINTH PREETHI<sup>2</sup>

<sup>1</sup>Post Graduate of General Medicine, Department of General Medicine, Government Stanley Medical College and Hospital, Chennai, India

<sup>2</sup>Associate Professor of General Medicine, Department of General Medicine, Government Stanley Medical College and Hospital, Chennai, India

\*Correspondence to: Dr. Veerasivabalan, Department of General Medicine, Government Stanley Medical College and Hospital, Chennai-600001, Tamil Nadu, India. Tel: +919787855960; E-mail: [rubinis478@gmail.com](mailto:rubinis478@gmail.com).

## Abstract

**Introduction:** Neurological complications following snake envenomation are uncommon, and acute ischemic stroke represents a particularly rare but potentially devastating manifestation. Because snakebite is prevalent in many tropical and subtropical regions, atypical neurological presentations may pose significant diagnostic challenges. Early recognition of vascular complications is critical, as prompt and appropriate intervention can result in favorable neurological outcomes.

**Case report:** A 47-year-old male presented with sudden-onset right-sided hemiparesis following a recent history of an unknown snake bite. There was no prior history of cerebrovascular disease or traditional vascular risk factors. Neurological examination revealed features consistent with an acute focal neurological deficit, along with bilateral ptosis. Neuroimaging confirmed the presence of an acute ischemic stroke. The patient was treated with timely administration of polyvalent antivenom along with supportive and neuroprotective care. Over the course of a one-week hospitalization, he demonstrated marked neurological improvement and achieved complete recovery of motor function without residual deficit at discharge.

**Discussion:** Ischemic stroke temporally associated with snake envenomation is an unusual and underrecognized phenomenon. Proposed mechanisms include venom-induced coagulopathy, endothelial dysfunction, vasculitis, platelet aggregation, and thrombotic occlusion of cerebral vessels, although a causal relationship cannot be firmly established from a single case. Differentiating a vascular event associated with envenomation from other neurotoxic sequelae is challenging, particularly in endemic regions where snakebites are common.

**Conclusion:** This case illustrates that acute ischemic stroke occurring in temporal association with snake envenomation can be reversible with early diagnosis and timely treatment. The patient achieved complete neurological recovery without residual deficit over the course of his hospitalization. Awareness of this rare presentation is crucial to ensure appropriate management and improved clinical outcomes.

**Keywords:** Acute ischemic stroke, Anti-snake venom, Hemiparesis, Envenomation

**How to cite this article:** V S, Preethi J. Acute Ischemic Stroke Associated with Snake Envenomation: A Reversible Cerebrovascular Event. Asia Pac J Med Toxicol. 2026; 15(3):141-45.

## INTRODUCTION

Snake bites and related deaths are not uncommon in India. Snake bites cause significant mortality and morbidity and are one of the preventable causes of death. The clinical manifestations of snake bite are diverse and depend on the species of snake, quantity of venom injected, and time to medical intervention. While haematological and local tissue effects are commonly observed, neurological complications are relatively uncommon. In addition, myotoxic manifestations occur after sea snake bite. Snake bites can

produce local envenomation reactions like cellulitis, ulceration, abscess, gangrene, and compartment syndrome that might even require amputation at times. Other systemic features include acute kidney injury, myonecrosis, rhabdomyolysis, coagulopathy, cardiac arrhythmia, myocarditis and ARDS. This case report aims to highlight the various neurotoxic manifestations of snake envenomation and describes a reversible acute ischemic stroke occurring in temporal association with a snake bite. Prompt initiation of anti-snake venom and other supportive measures help mitigate the morbidity and mortality.

## CASE PRESENTATION

An apparently healthy 47-year-old farmer, with no known comorbidities, presented to the hospital with alleged history of being bitten by a snake over the right middle finger at his residence around 4 pm. He had complaints of swelling at the bite site soon after the snake bite. One hour later, the patient had difficulty using the right upper and lower limb with paucity of movements of the right upper and lower limb. He had slurring of speech. He underwent native treatment for the same before coming to the hospital. There was no history of seizure, headache, diplopia, dropping of eyelids or altered sensorium noted by the patient or family prior to arrival. He presented to the hospital around 3 am the following night, approximately 11 hours after the bite.

On clinical examination, the patient was conscious, oriented, afebrile and not dyspneic. No bleeding manifestations noted. The patient was hemodynamically stable with: Heart rate 86/minute, all the peripheral pulses were well felt, Respiratory rate-15/minute, SpO<sub>2</sub>: 98% on room air and BP 120/80mmHg, capillary blood glucose was noted to be 109mg/dl. The blood sample drawn for whole blood clotting test [WBCT] clotted within 20 minutes. Urine output was adequate. Normal findings across cardiovascular, respiratory and abdominal systems. CNS examination revealed ptosis of both eyes with preserved extraocular movements and normally reactive pupils, hypotonia on the right upper and lower limb, with paucity of movements of the right upper and lower limb; absent deep tendon reflexes with an extensor plantar response on the right side whereas reflexes were elicitable on the left side with a flexor plantar response; Right UMN type facial palsy was noted. There was no bulbar weakness, dysphagia, or respiratory compromise. Normal higher mental functions as evidenced by MMSE 27/30; bilateral pupils equally reactive to light; there was no nystagmus. Sensory system, autonomic system and coordination was intact. There was a tender swelling limited to the right middle finger with erythema and no discoloration.

Given the bilateral ptosis at presentation, there was initial clinical concern for neurotoxic envenomation, and management priorities were directed accordingly; a formal NIHSS score was therefore not calculated at admission, and clinical course was instead followed with serial neurological examination.

A comprehensive initial laboratory test, including complete blood count, liver function test, renal function test, serum electrolytes, coagulation profile were within normal limits. Lipid profile and HbA1c levels were within normal limits. Chest radiography and electrocardiography did not show any abnormalities. Serology for VDRL, HIV, HBV and HCV were non-reactive. ANA profile, ANCA and Antiphospholipid antibody panel were negative. The work up for thrombophilia including Antithrombin III, Protein C, Protein S, Factor 5 Leiden mutation, Prothrombin gene mutation and Homocysteine levels were not contributory.

The arteriovenous doppler study of the involved upper limb showed adequate vascularity.

In view of right-sided hemiparesis with right UMN type facial palsy, a CT brain scan was done on admission, which showed an ill-defined hypodensity in the left parietal and basi-temporal region. There was no evidence of hemorrhage. MRI brain, performed 24 hours after admission due to logistic constraints, showed an acute infarct in the left frontal, parietal lobe, left caudate lobe and left lentiform nucleus (figure 1). A carotid artery doppler showed a normal study, with no evidence of thrombus or stenosis. Taken together, these findings support a diagnosis of acute ischemic stroke occurring in temporal association with a snake bite in our patient.

The patient was managed with polyvalent anti-snake venom (10 vials, reconstituted and infused over 1 hour in isotonic saline at 5–10 mL/kg body weight, targeting the unbound circulating venom), antiplatelet therapy (aspirin 150 mg) and a statin (atorvastatin 40 mg) — the latter two initiated once neuroimaging had confirmed acute ischemic infarction and excluded intracranial hemorrhage, together with limb physiotherapy. He also received intravenous antibiotics for cellulitis around the bite site. There was spontaneous recovery of the limb weakness over the course of the one-week hospitalization, with no residual neurological deficit at discharge. The patient was symptomatically well and doing well at the time of discharge (table 1).

## DISCUSSION

From a state where snake bite was one of the neglected tropical diseases in the yesteryear, we have come to a level where clinicians act proactively to reduce the morbidity associated with snake bites, as it is one of the preventable causes of mortality when meticulously managed. In this case report, we highlight the various neurological manifestations following a snake bite.

The neurological manifestations following a snake bite include ptosis, extraocular muscle weakness, diplopia, intracerebral hemorrhage, altered sensorium, toxic encephalopathy, early morning neuromuscular syndrome, palatal and pharyngeal muscle palsy, limb weakness, bulbar paralysis leading to dysphasia, dysarthria, dysphagia with subsequent respiratory muscle weakness leading to respiratory failure [1-4].

At the neuromuscular junction, presynaptic beta bungarotoxin of krait inhibits the release of acetylcholine from the synaptic vesicles by blocking the SNARE proteins. Whereas, the postsynaptic alpha bungarotoxin of cobra causes a blockade of acetylcholine receptors, thereby preventing the interaction between acetylcholine and postsynaptic membrane receptors. These lead onto impaired neuromuscular transmission and subsequent development of muscular paralysis. It is important to understand that there is delayed clinical recovery from muscular paralysis following krait bite because recovery depends on regeneration of the terminal axon. In contrast, there is rapid

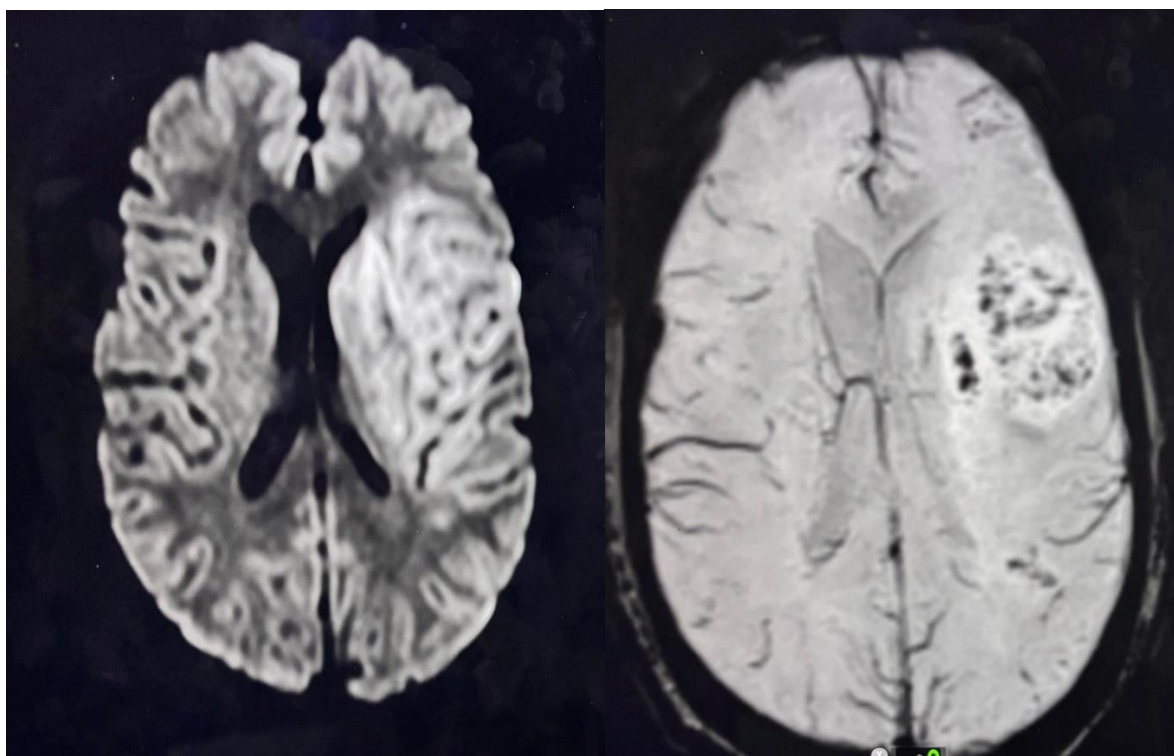


**Figure 1.** MRI brain performed 24 hours after admission (approximately 24 hours after symptom onset). (A) Susceptibility-weighted imaging (SWI/T2\*-GRE) demonstrates susceptibility blooming within the left frontoparietal infarct, consistent with hemorrhagic transformation. (B) Diffusion-weighted imaging (DWI) demonstrates restricted diffusion involving the left frontal and parietal cortex, left caudate nucleus, and left lentiform nucleus, consistent with an acute left middle cerebral artery territory infarction. The corresponding ADC images were unavailable for retrieval from the archived PACS at the time of manuscript revision and are therefore not included.

**Table 1.** Clinical timeline from snakebite to hospital discharge

| Time                                  | Event                                                                                                                                                                                                                                                                  |
|---------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Day 0, ~4:00 pm                       | Snake bite to right middle finger at home; local swelling noted                                                                                                                                                                                                        |
| Day 0, ~5:00 pm (1 hour post-bite)    | Onset of right hemiparesis and slurred speech; native treatment sought                                                                                                                                                                                                 |
| Day 1, ~3:00 am (~11 hours post-bite) | Presentation to hospital                                                                                                                                                                                                                                               |
| Day 1, on admission                   | Clinical examination: bilateral ptosis with preserved eye movements, right hemiparesis, right UMN facial palsy; WBCT clotted at 20 minutes; CT brain shows ill-defined hypodensity in the left parietal and basi-temporal region. There was no evidence of hemorrhage. |
| Day 1, promptly after admission       | Polyvalent anti-snake venom initiated (10 vials infused over 1 hour in isotonic saline, 10 mL/kg)                                                                                                                                                                      |
| Day 1, after neuroimaging             | Antiplatelet therapy (aspirin 150 mg) and statin (atorvastatin 40 mg) started once acute infarction was confirmed and hemorrhage excluded                                                                                                                              |
| Day 2 (24 hours post-admission)       | MRI brain confirms acute infarct in left frontal, parietal, caudate and lentiform regions                                                                                                                                                                              |
| Day 7                                 | Complete resolution of limb weakness; discharged with no residual neurological deficit                                                                                                                                                                                 |

recovery of paralysis following cobra bite as there is dissociation of the toxin-receptor complex following

administration of anti-snake venom and anticholinesterase drugs [3, 4].

Patients having decreased visual acuity following snake bite may have ocular complications such as subconjunctival hemorrhage, vitreous hemorrhage, CRAO (central retinal artery occlusion), macular infarction and PRES [3, 5].

PRES (posterior reversible encephalopathy syndrome) is a clinical entity characterized by headache, diminished visual acuity, altered level of sensorium and focal neurological deficits. This could occur due to toxin-mediated infarction of the vessels of the posterior circulation of the brain [5].

Early morning neuromuscular syndrome [EMNS] is one of the rare clinical entities following krait bites. Patients who succumb to krait bite may not have any bite marks, making the diagnosis complicated. Such patients may go to bed healthy and may be found paralyzed in the morning following a krait bite. In such a scenario, only a high degree of suspicion and clinical acumen fetches the right diagnosis [6].

The peripheral nervous system involvement following a snake bite leads to acute onset flaccid quadriplegia and quadriparesis which causes weakness of the pharyngeal and respiratory muscles, culminating in respiratory failure [3-6].

Polyvalent anti-snake venom itself can cause immune complex-mediated catastrophic disease of the central nervous system namely, ADEM (acute demyelinating encephalomyelitis) and AHL (acute hemorrhagic leucoencephalitis), both of which are characterized by fulminant inflammation of the cerebral white matter [3, 4].

Although bilateral ptosis at presentation raised initial concern for a primary neurotoxic envenomation, several features in our patient argued against this. Neurotoxic envenomation typically evolves as a symmetric, often descending, process — progressive ophthalmoplegia, bulbar weakness with dysphagia and dysarthria, and eventual respiratory muscle involvement usually accompany or follow ptosis. In contrast, our patient had preserved extraocular movements, normally reactive pupils, no bulbar weakness, and no respiratory compromise at any point, while the hemiparesis, UMN-type facial palsy, and lateralized reflex and plantar changes were strictly unilateral and confined to a single vascular territory, corroborated by CT and MRI evidence of an acute left-sided infarct. This dissociation — isolated, non-progressive bilateral ptosis without other neuromuscular junction features, alongside a lateralized corticospinal deficit favored a vascular rather than a primary neurotoxic mechanism, although a mild, self-limiting neurotoxic contribution to the ptosis cannot be entirely excluded.

The mechanism underlying cerebrovascular events after snake envenomation remains incompletely understood but is thought to be multifactorial, involving toxin-mediated vasculitis, endothelial injury, and procoagulant and platelet-aggregating effects of venom components that activate the coagulation cascade and promote thromboinflammation. A recent systematic review and individual patient data meta-analysis of strokes following snakebite envenomation found

that both ischemic and hemorrhagic strokes occur, frequently involving more than one vascular territory, with venom-induced coagulopathy, endothelial injury, and thromboinflammation implicated as key contributors [7]. Similar mechanisms — toxin-mediated vasculitis and endothelial damage — have been proposed in case reports of ischemic stroke following Russell's viper envenomation, including instances of multi-territorial infarction and middle cerebral artery territory infarction despite timely antivenom administration [8, 9]. Taken together with our case, these reports suggest that ischemic cerebrovascular events, although rare, should be recognized as a possible complication of envenomation across multiple snake families, warranting a high index of suspicion in patients presenting with focal neurological deficits after a snake bite. Our patient was managed with prompt initiation of anti-snake venom, antiplatelets, statins and limb physiotherapy, which was followed by complete clinical recovery over the course of hospitalization.

---

## CONCLUSION

This case describes the occurrence of an acute ischemic stroke in temporal association with a snake bite, with complete clinical recovery achieved during a one-week hospitalization and no residual neurological deficit at discharge. Prompt neuroimaging, and timely initiation of ASV, antiplatelets, statins and limb physiotherapy appear important for mitigating morbidity and mortality in such cases. Clinicians providing service to the rural population must remain vigilant for such rare neurological manifestations to improve outcomes in snake bite cases.

## CONSENT

Written Informed consent for publication of the case and images has been obtained from the patient.

---

## ACKNOWLEDGMENTS

Not applicable.

**Conflict of interest:** The authors declare no conflict of interest.

**Funding and Support:** This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

---

## REFERENCES

1. Sahoo AK, Sriramka B. Acute reversible ischemic stroke after snake bite. *Indian J Crit Care Med.* 2018;22(8):611–12. doi:10.4103/ijccm.IJCCM\_455\_17.
2. Paul G, Paul BS, Puri S. Snake bite and stroke: Our experience of two cases. *Indian J Crit Care Med.* 2014;18(4):257–8. doi:10.4103/0972-5229.130585.
3. Ranawaka UK, Lalloo DG, de Silva HJ. Neurotoxicity in snakebite—the limits of our knowledge. *PLoS Negl Trop Dis.* 2013;7(10):e2302. doi:10.1371/journal.pntd.0002302.
4. Huang YK, Chen YC, Liu CC, Cheng HC, Tu AT, Chang KC. Cerebral complications of snakebite envenoming: Case studies. *Toxins (Basel).* 2022;14(7):436. doi:10.3390/toxins14070436.

5. Ibrahim AM, ElSefi TT, Ghanem M, Fayed AM, Shaban NA. A horned viper bite victim with posterior reversible encephalopathy syndrome. *Case Rep Neurol Med*. 2017;2017:1835796. doi:10.1155/2017/1835796.
6. Samprathi M, Gupta V, Jayashree M, Bansal A, Baranwal A, Nallasamy K. Epidemiology and outcomes of early morning neuromuscular syndrome following snake bite—a retrospective study. *J Trop Pediatr*. 2020;66(4):435–40. doi:10.1093/tropej/fmz084.
7. Almeida T, Priante SP, João GP, Oliveira DN, Mouta G, Sachett J, et al. Strokes following snakebite envenomations: A systematic review and individual patient data meta-analysis. *PLoS Negl Trop Dis*. 2025;19(12):e0013789. doi:10.1371/journal.pntd.0013789.
8. Yadav P, Kumar M, Sundarsingh V, Charlotte A, Thomas M. Multi-Territorial Ischemic Stroke Following Russell's Viper Envenomation: A Case Report. *Cureus*. 2025;17(7):e87988. doi:10.7759/cureus.87988.
9. Vijayakumaran Y, Mayurathan P, Sabrina S. Middle Cerebral Artery Territory Ischemic Infarction Secondary to Russell's Viper Envenomation: A Case Report. *Cureus*. 2025;17(11):e96290. doi:10.7759/cureus.96290.