International Journal of Science and Research (IJSR)
ISSN: 2319-7064
Impact Factor 2025: 7.089

Multiple Bilateral Ischemic Strokes Following Russell's Viper Bite in a Young Woman: A Rare Case Report

Dr. Pinnam Venkata Pavan Kalyan[1], Dr. Challa Venkata Mahesh[2], Dr. G. Shobha Rani[3]

[1]Junior Resident, Department of General Medicine, Government General Hospital & ACSR Government Medical College, Nellore, Andhra Pradesh, India

[2]Junior Resident, Department of General Medicine, Government General Hospital & ACSR Government Medical College, Nellore, Andhra Pradesh, India

[3]Associate Professor, Department of General Medicine, Government General Hospital & ACSR Government Medical College, Nellore, Andhra Pradesh, India (Corresponding Author)

Abstract: ***Snakebite remains a major public health problem in India. Ischemic stroke is an uncommon but potentially serious neurological complication of snake envenomation. Russell's viper (Daboia russelii), which is widely distributed across the Indian subcontinent, causes hemotoxic envenomation and may produce coagulopathy and vascular endothelial injury, potentially contributing to cerebral ischemia. We report a 39-year-old woman from Andhra Pradesh who developed multiple bilateral acute ischemic infarcts involving the frontoparietal lobes, caudate nucleus, centrum semiovale, corona radiata, corpus callosum, bilateral cerebellar hemispheres, and vermis following a Russell's viper bite. She presented with acute left hemiparesis and visual impairment, with concurrent acute kidney injury (AKI). Despite extensive infarction involving multiple vascular territories, she experienced substantial neurological improvement and was discharged after 11 days. Possible mechanisms include venom-associated endothelial injury, vasculopathy, and disturbances of hemostasis. This case highlights the importance of considering cerebrovascular complications in patients presenting with neurological deficits after viper envenomation.***

Keywords: Russell's viper; Snakebite; Ischemic stroke; Acute kidney injury

1. Introduction

Snakebite is a major neglected public health problem worldwide [1]. India accounts for a substantial proportion of the global burden of snakebite and related mortality [2,3]. Among the medically important snakes in India, Russell's viper (Daboia russelii) is a major cause of severe envenomation, particularly in southern states such as Andhra Pradesh, Tamil Nadu, and Karnataka [2,3].

Russell's viper venom has predominantly hemotoxic and vasculotoxic effects. Envenomation may cause local tissue injury, venom-induced consumption coagulopathy (VICC), acute kidney injury (AKI), and, rarely, neurological complications. Ischemic stroke is an uncommon but recognized complication of snakebite, with cases reported in the medical literature [4–6,9,10].

The mechanisms underlying cerebral ischemia after viper envenomation are likely multifactorial. Proposed mechanisms include venom-associated endothelial injury, inflammatory vascular damage, disturbances in coagulation and fibrinolysis, disseminated intravascular coagulation (DIC), and vasospasm [7–10]. Most published reports describe infarction confined to a single vascular territory [4,6]. Bilateral infarcts involving both anterior and posterior circulations are distinctly uncommon. We describe a 39-year-old woman who developed extensive bilateral, multi-territory acute ischemic infarcts following a Russell's viper bite, with marked subsequent neurological recovery.

2. Case Report

A 39-year-old woman, a farmer from a rural area of Nellore district, Andhra Pradesh, was brought to the casualty department of Government General Hospital, Nellore, on 2 September 2025. She reported a snakebite on the lateral aspect of her right foot at midnight on the preceding day. She was initially taken to a private hospital, where she received initial care. Soon after the bite, she developed sudden-onset blurring of vision followed by progressive weakness of the left upper and lower limbs.

At presentation, she complained of weakness of the left upper and lower limbs and blurring of vision. There was no history of seizures, fever, chest pain, or breathlessness. She had no known history of diabetes mellitus, hypertension, coronary artery disease, or chronic kidney disease.

On examination, she was conscious and coherent. Her blood pressure was 140/80 mmHg, pulse rate 110 beats/min (regular), oxygen saturation 98% on room air, and respiratory rate 18 breaths/min. General examination revealed pallor. Neurological examination showed a Glasgow Coma Scale score of E4V5M6. She had bilateral visual impairment, with perception of light preserved but inability to count fingers in either eye. Both pupils were equal (3–4 mm) and reactive to light. Motor power was 5/5 in the right upper and lower limbs; in the left upper limb, it was 3/5 proximally and 2/5 distally, and in the left lower limb it was 3/5. Tone was reduced on the left side. Deep tendon reflexes were present bilaterally, and plantar responses were extensor on both sides.

Selected hematological and biochemical parameters are summarized in Table 1. The recorded bleeding time was 9 minutes and clotting time was 5 minutes. Viral markers (HBsAg, HCV, and HIV) were negative.

Table 1: Laboratory Investigations on Admission

Investigation	Result	Reference Range
Hemoglobin	10.2 g/dL	12–16 g/dL (Low)
Total WBC Count	13,100 /cumm	4,000–11,000 (High)
Platelets	2.45 lakhs/cumm	1.5–4.5 lakhs (Normal)
Serum Creatinine	2.8 mg/dL (peak)	0.5–1.2 mg/dL (High)
Blood Urea	84 mg/dL	15–45 mg/dL (High)
Serum Na+	136 mEq/L	135–145 mEq/L (Normal)
Serum K+	3.3 mEq/L	3.5–5.0 mEq/L (Low-normal)
Serum Bilirubin (Total)	2.0 mg/dL	0.2–1.2 mg/dL (High)
SGOT	42 U/L	10–40 U/L (Borderline)
SGPT	23 U/L	7–56 U/L (Normal)
CRP	Positive	Negative (Elevated)
HBsAg / HCV / HIV	Negative	—
GRBS	98 mg/dL	70–140 mg/dL (Normal)
ESR	Elevated	0–20 mm/hr

Electrocardiography showed sinus rhythm at 100 beats/min, with nonspecific T-wave inversions in leads V3–V6. Two-dimensional echocardiography showed preserved left ventricular systolic function (ejection fraction approximately 55%), with no intracardiac thrombus, vegetation, or pericardial effusion. Non-contrast computed tomography of the brain demonstrated acute ischemic infarcts involving the bilateral frontoparietal lobes, head of the right caudate nucleus, right centrum semiovale, corona radiata, body of the corpus callosum, bilateral cerebellar hemispheres, and vermis, without intracranial hemorrhage. Magnetic resonance imaging confirmed multiple acute infarcts involving bilateral middle cerebral artery and posterior cerebral artery territories, indicating a multi-territory pattern [Figures 1–5].

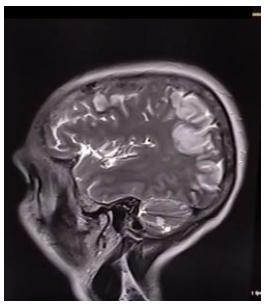
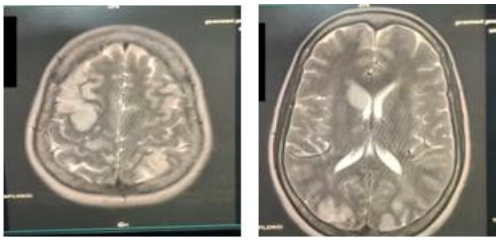
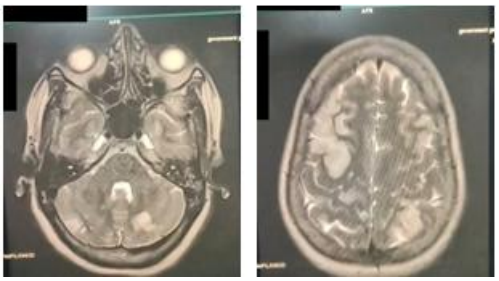


Figure 1: MRI Brain- Sagittal T2 view showing multiple hyperintense infarct areas in bilateral hemispheres



Figures 2–3: MRI Brain- Axial T2 views demonstrating bilateral fronto-parietal infarcts and corpus callosum involvement



Figures 4- 5: MRI Brain- Axial T2 views at the level of posterior fossa and cerebellum showing bilateral cerebellar and vermian infarcts

Neurology review confirmed multiple ischemic strokes in a relatively young patient. Clinical findings included right homonymous hemianopia and left-sided neglect. An antinuclear antibody/vasculitis profile, carotid Doppler study, and further autoimmune evaluation were advised. The final diagnosis was multiple bilateral acute ischemic infarcts with left hemiparesis following Russell's viper envenomation, with concurrent acute kidney injury (AKI).

The patient was admitted to MICU-II. At presentation, she received a total of 20 vials of polyvalent anti-snake venom (ASV) over 12 hours. Other treatment included intravenous fluids, Inj. Ceftriaxone 1 g once daily, tablet Aspirin 150 mg once daily, tablet Atorvastatin 40 mg at bedtime, tablet Amlodipine 5 mg once daily, Inj. Mannitol 100 mL three times daily for cerebral edema, a proton pump inhibitor, and antiemetics. Physiotherapy for the upper and lower limbs was initiated early.

The patient showed progressive clinical improvement during the 11-day hospital stay (2–12 September 2025). Motor power in the left limbs improved from 2–3/5 at admission to 4–5/5 by day 10. Vision also improved, from perception of light to counting fingers at 6 metres, with a normal confrontation test. Renal function improved, and serum creatinine decreased to 1.2 mg/dL. She was discharged ambulant, with substantial neurological and functional recovery, on oral aspirin, atorvastatin, amlodipine, and vitamin supplements, with outpatient follow-up advised.

Discussion

Bashir and Jinkins [4] described cerebral infarction following carpet viper (Echis carinatus) envenomation. In an observational study of 309 patients bitten by Bothrops spp., Mosquera et al. [5] reported cerebrovascular events in eight patients (2.6%); seven were intracranial hemorrhages and one was an ischemic infarction. Narang et al. [6] described an MCA-territory ischemic infarction following a Russell's viper bite.

The principal feature of this case is the extensive bilateral, multi-territory distribution of cerebral infarction, involving both anterior and posterior circulations, followed by substantial neurological recovery. The patient also had concurrent AKI.

Russell's viper venom contains several enzymatic and non-enzymatic proteins. Its major toxin families include phospholipase A2, snake venom serine proteases,

metalloproteinases, and other proteins involved in hemostatic effects. These components can produce venom-induced consumption coagulopathy (VICC), with consumption of fibrinogen and other coagulation factors. The resulting hemostatic disturbance may, in some circumstances, contribute to thrombotic or ischemic complications [7,8].

The precise mechanism of cerebral infarction in this patient cannot be established from the available investigations. Potential mechanisms include venom-associated endothelial injury and vasculopathy, inflammatory vascular damage, vasospasm, and abnormalities of coagulation and fibrinolysis, including DIC [7–10]. These mechanisms are biologically plausible after viper envenomation; however, specific evidence confirming any one mechanism was not available in this case.

The bilateral and posterior circulation involvement in this patient is unusual. Most reported cases describe unilateral MCA-territory infarction [4,6,9]. The involvement of the bilateral frontoparietal lobes, caudate nucleus, cerebellar hemispheres, vermis, and corpus callosum suggests a diffuse vascular process rather than a single-territory event. Echocardiography did not identify an intracardiac thrombus or another evident cardiac source. Nevertheless, the available findings do not completely exclude all conventional stroke mechanisms.

The substantial neurological recovery is notable given the extent of radiological involvement. Possible contributing factors include the patient's relatively young age, supportive treatment, early rehabilitation, and the possibility that some component of the vascular dysfunction was reversible. These explanations remain speculative.

Acute disseminated encephalomyelitis (ADEM) due to polyvalent antisnake venom (ASV) has been infrequently reported in the literature [11]. However, ADEM usually presents late with patchy muscle paralysis, which was not seen in our case.

3. Conclusion

We report an uncommon case of multiple bilateral acute ischemic strokes in a 39-year-old woman following Russell's viper envenomation in Andhra Pradesh. The case is notable for extensive infarction involving multiple vascular territories, concurrent AKI, and substantial neurological recovery. Clinicians in snakebite-endemic regions should consider cerebrovascular complications when patients develop neurological deficits after envenomation. Prompt neuroimaging and multidisciplinary management are important.

Declarations

Declaration of Patient Consent: Written informed consent was obtained from the patient for publication of this case report and associated images.

Financial Support and Sponsorship: Nil.

Conflicts of Interest: There are no conflicts of interest.

References

[1] Kasturiratne A, Wickremasinghe AR, de Silva N, et al. The global burden of snakebite: a literature analysis and modelling based on regional estimates of envenoming and deaths. PLoS Med. 2008; 5(11): e218.
[2] Suraweera W, Warrell D, Whitaker R, et al. Trends in snakebite deaths in India from 2000 to 2019. eLife. 2020; 9: e54076.
[3] Mohapatra B, Warrell DA, Suraweera W, et al. Snakebite mortality in India: A nationally representative mortality survey. PLoS Negl Trop Dis. 2011; 5: e1018.
[4] Bashir R, Jinkins J. Cerebral infarction in a young female following snake bite. Stroke. 1985;16(2):328–330. doi:10.1161/01.STR.16.2.328.
[5] Mosquera A, Idrovo LA, Tafur A, Del Brutto OH. Stroke following Bothrops spp. snakebite. Neurology. 2003;60(10):1577–1580. doi:10.1212/01.WNL.0000061614.52580.A1.
[6] Narang SK, Paleti S, Azeez Asad MA, Samina T. Acute ischemic infarct in the middle cerebral artery territory following a Russell's viper bite. Neurol India. 2009; 57: 479–80.
[7] Sharma M, Das D, Iyer JK, Kini RM, Doley R. Unveiling the complexities of Daboia russelii venom, a medically important snake of India, by tandem mass spectrometry. Toxicon. 2015;107(Pt B):266–281. doi:10.1016/j.toxicon.2015.06.027.
[8] Marsh NA. Snake venoms affecting the haemostatic mechanism. Blood Coagul Fibrinolysis. 1994; 5: 399–410.
[9] Murthy JM, Kishore LT, Naidu KS. Cerebral infarction after envenomation by viper. J Comput Assist Tomogr. 1997; 21: 35–7.
[10] Paul G, Paul BS, Puri S. Snake bite and stroke: Our experience of two cases. Indian J Crit Care Med. 2014;18(4):257–258. doi:10.4103/0972-5229.130585.
[11] Tripathy S, Routray PK, Mohapatra AK, Mohapatra M, Dash SC. Acute demyelinating encephalomyelitis after anti-venom therapy in Russell's viper bite. J Med Toxicol. 2010; 6: 318–21.