JoTH Journal of Tropical Health

Case report

Bilateral Cerebellar Infarctions Following Russell's Viper (*Daboia russelii*) Envenoming: A Rare Case Report from Sri Lanka

Sanmuganathan Priyadarshan[1], Namal Herath[2], Hemal Senanayake[3]*

[1]Professorial Medical Unit, Teaching Hospital, Anuradhapura, Sri Lanka.

[2]Department of Physiology, Faculty of Medicine and Allied Sciences, Rajarata University of Sri Lanka.

[3]Department of Medicine, Faculty of Medicine and Allied Sciences, Rajarata University of Sri Lanka.

Abstract

Most snakebites in Sri Lanka are caused by Russell's viper. *Daboia russelii*, one of Sri Lanka's medically most important snakes, typically cause local swelling, coagulopathy, neurotoxicity and acute kidney injury. Cerebral infarction is a recognised but rare complication of Russell's viper envenoming, and posterior circulation involvement is particularly uncommon.

We report the case of a 43-year-old man bitten by a Sri Lankan Russell's viper who subsequently developed bilateral cerebellar infarctions. He initially presented with features of both local and systemic envenomation and received prompt administration of Snake Venom Anti-Serum. On the second day of admission, he developed a severe occipital headache. Computed tomography of the brain revealed bilateral cerebellar infarctions.

This case underscores a rare neurological complication of Russell's viper bite and highlights the importance of early recognition and neuroimaging in patients who develop new, severe headaches or neurological symptoms following Russell's' viper envenomation.

Keywords: Russell's viper envenoming, cerebellar infarction, posterior circulation stroke

Funding: None
Competing interests: None
Received: 02 December 2025 **Accepted:** 01 March 2026 **Published:** 26 May 2026

***Corresponding author:** Email: senanayakehms@med.rjt.ac.lk https://orcid.org/0000-0001-5739-1979

Cite this article as: Priyadarshan S et al. Bilateral Cerebellar Infarctions Following Russell's Viper (*Daboia russelii*) Envenoming: A Rare Case Report from Sri Lanka. Journal of Tropical Health 2026;2 (1): 30-35. DOI: https://doi.org/10.4038/joth.v2i1.45

Introduction

Snake envenoming is an important cause of morbidity and mortality in tropical countries, particularly in rural Sri Lanka. Among venomous species, the Russell's viper (*Daboia russelii*) is one of the most dangerous and frequently encountered snakes [1]. Common manifestations of envenoming include local swelling at the bite site, venom-induced consumption coagulopathy, neurotoxicity, and acute kidney injury [1,3,18].

Cerebral infarctions following Russell's viper envenoming are uncommon, with involvement of the anterior circulation as the predominant pattern [2,3,7]. Posterior circulation infarctions, especially cerebellar involvement, are rare [5,18].

We report a case of bilateral cerebellar infarction following Russell's viper envenomation in a previously healthy Sri Lankan male with a normal coagulation profile.

Case Presentation

A 43-year-old male from the North Central Province of Sri Lanka, previously healthy with no known past medical history, was bitten on his left foot by a Russell's viper on 31 December 2024. He was taken to a local hospital within 1 hour of envenoming. He developed swelling at the bite site, abdominal pain, vomiting, and bilateral eyelid drooping following the envenoming. Based on examination of the snake specimen at the local hospital, the offending snake was identified as a Russell's viper. However, no specimen was available for confirmation at the Teaching Hospital, Anuradhapura. At the local hospital, he was treated with 20 vials of Indian polyvalent snake antivenom without any antivenom allergic reaction and was transferred to Teaching Hospital Anuradhapura for further management.

The patient's only complaint on admission to the Anuradhapura hospital was mild pain at the bite site. Ptosis, ophthalmoplegia, ataxia, bleeding manifestations, and systemic neurological abnormalities were absent on examination. On admission, he experienced no nausea or vomiting, and there was no pain in his abdomen. PT/INR and aPTT were within normal ranges, and the whole blood clotting test (WBCT) revealed clotting in less than 20 minutes.

On the second day, he complained of a severe occipital headache unresponsive to paracetamol. There was no vomiting, visual disturbance, ophthalmoplegia, ptosis, limb weakness, or sensory deficit. Neurological and systemic examinations were normal. Blood pressure was 130/80mmHg, and pulse rate was 80 beats/minute and regular. Examination of the optic fundus was negative for papilledema.

The patient complained of a severe occipital headache for which analgesics were administered. Over the course of the following day, his headache grew worse even though there were no focal or systemic neurological symptoms. The results of the bilateral fundoscopic examination were normal. Intracranial haemorrhage was taken into consideration in the differential diagnosis, even though there were no abnormalities on preliminary investigations, no external bleeding, and no signs of end-organ damage. To rule out intracranial bleeding, a non-contrast Computed Tomography (NCCT) scan of the brain was performed, which instead showed bilateral cerebellar infarctions. The patient's headache gradually subsided with regular paracetamol and tramadol administered as needed. The patient had no headache or any other neurological impairments by the fifth day following envenoming.

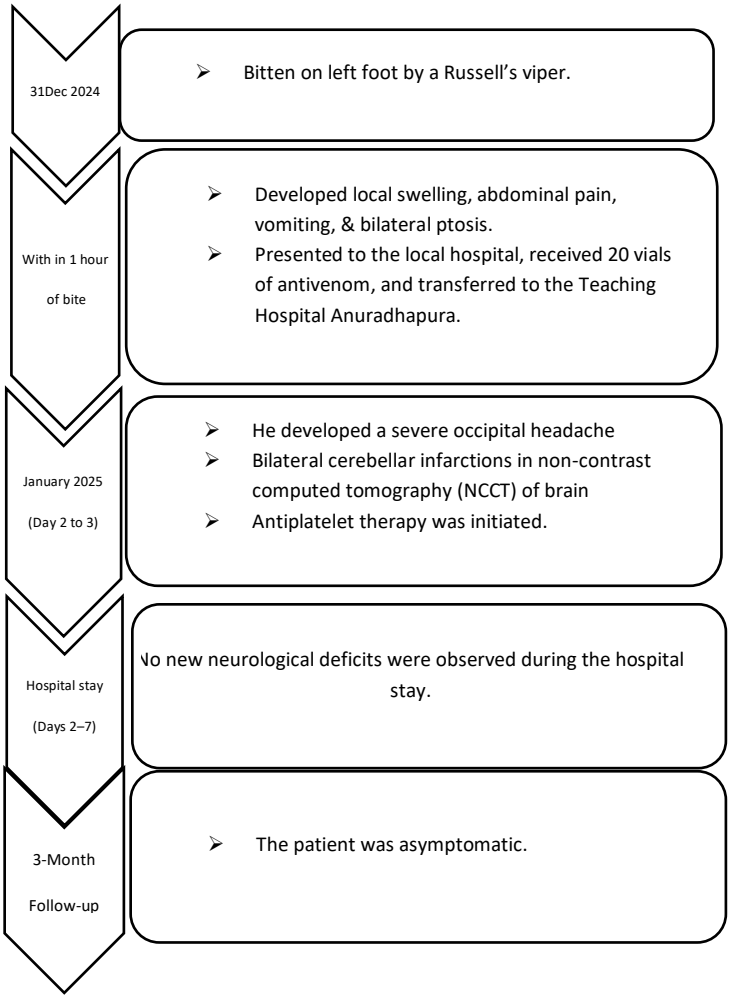

Figure 1: Timeline of events

The results of his laboratory investigations are summarised in Table 1. Chest X-ray was normal, and the 2D echocardiogram was negative for intracardiac thrombi. The electrocardiogram (ECG) showed a sinus rhythm. The blood picture was normal. The blood picture performed on day 2 of admission showed no evidence of microangiopathic haemolytic anaemia. Non-contrast Computer Tomographic (NCCT) brain imaging done on day 2 of admission revealed bilateral cerebellar infarctions (Figures 2 and 3). Surprisingly, he didn't have any cerebellar signs related to the infarctions. He was managed with antiplatelet therapy (aspirin 300mg), intravenous fluids, and close neurological observation. He did not develop further neurological deficits and was discharged on day 7 with complete recovery. He was symptom-free at the 3-month follow-up.

Table 1: summary of investigations

Investigations	Day 1	Day 2	Day3	Reference range
White cell count	12.7	10.3	9.22	4-10 × 10^9/L
Hemoglobin	14.2	14	12.9	11.5-15 g/dL
Platelets	211	254	253	150-450× 10^9/L
Hematocrit		41.7	38.5	37-54 %
Erythrocyte sedimentation rate		25		<20 mm/hours
C-reactive protein	25		9.9	< 5 mg/l
Serum creatinine	70.7	97	99	60-115 µmol/L
Aspartate transaminase	24			9-48 u/l
Alanine transaminase	20			10-40 u/l
Serum albumin	44.3			35-52 g/l
Serum globulin	33.2			25-35 g/l
Direct bilirubin	1.05			< 3.4 µmol/L
Indirect bilirubin	7.54			
Prothrombin Time (PT)	12		13.6	9.7-14.7 second

Activated partial thromboplastin time (aPTT)	29.9	28.8	21-30 second
International normalized ratio (INR)	0.97	0.90	0.8-1.10
Sodium	145		135-148 mmol/l
Potassium	4		3.6-5.0 mmol/l
Anti-nuclear antibody (ANA)		38.4	Positive 40-400 AU/mL

Discussion

This case reports a very uncommon cerebellar infarction after *Daboia russelii* envenoming, with no cerebellar symptoms and only a severe occipital headache. Although reports of cerebellar strokes following Russell's viper bites exist, our patient's lack of focal neurological deficits makes this presentation particularly difficult and raises the possibility of a delayed diagnosis. Therefore, in envenomed patients, the isolated, disproportionate occipital headache should be identified as a possible early indicator of posterior circulation ischemia. This case highlights the necessity of early neuroimaging for snakebite victims experiencing severe or worsening occipital headaches to guarantee prompt identification and treatment of major complications.

Our patient developed local effects and features of mild neurotoxicity following envenoming but had no evidence of venom-induced consumption coagulopathy (VICC) or microangiopathic hemolytic anaemia (MAHA). VICC is the most frequent systemic toxicity of Russell's viper venom and is often implicated in both hemorrhagic and thrombotic complications. However, our patient repeatedly demonstrated normal platelet counts, normal coagulation parameters, and no biochemical markers or evidence of MAHA on the blood picture. In the absence of hypotension or systemic coagulopathy, the mechanism of infarction warrants deeper exploration [6,15,16,18].

Cerebral infarction is an uncommon yet described complication of Russell's viper envenoming. [2,3,7,8]. Several pathophysiological mechanisms have been proposed, such as venom-induced consumption coagulopathy (VICC) and cerebral artery thrombus formation, Metalloproteinases and phospholipase A_2 toxins-induced endothelial damage and vasculitis, procoagulant and platelet-aggregating enzymes such as serine proteases, factor X activators and fibrinogenases induced thrombosis and direct vasospasm or immune-mediated vasculopathy [6,9,10,11,12,13,14,15,16.18].

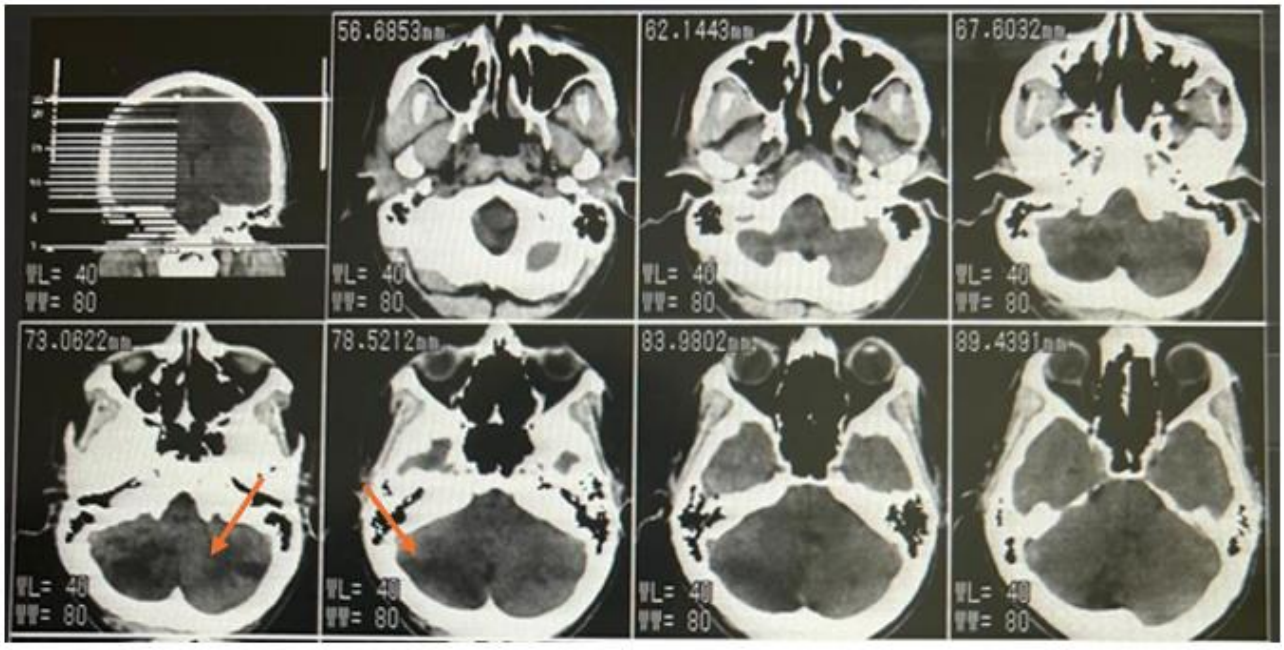

Figure 2: Non-contrast Computed Tomography brain on day 2 of admission showing bilateral cerebellar infarctions (arrows)

Russell's viper venom contains potent procoagulant enzymes and endothelial-damaging toxins that can induce focal thrombosis without producing a detectable systemic coagulopathy. Localised activation of the coagulation cascade, together with endothelial swelling, vasospasm, or direct vascular injury in the posterior circulation, may result in a territorial cerebellar infarction despite normal platelet counts and coagulation tests. This mechanism provides a coherent explanation for how significant cerebral ischaemia can

occur even when conventional laboratory markers of VICC or MAHA remain within normal limits [9,10,11,12,13,14].

The possibility of antivenom-related immune thrombosis deserves consideration. Equine-derived $F(ab')_2$ antivenoms can trigger hypersensitivity reactions, yet there is no established evidence that they induce a VITT-like PF4-mediated thrombosis syndrome. VITT typically presents with thrombosis accompanied by marked thrombocytopenia, which was not seen in this case. Nonetheless, immune-mediated platelet activation remains theoretically possible. If clinical suspicion arises in future cases, PF4 antibody testing and functional platelet activation assays would be informative.

Most previous Sri Lankan case series have reported anterior circulation infarcts (mainly in the middle cerebral artery territory) [2,3,7]. Posterior circulation infarctions, including occipital lobes and cerebellum, are extremely rare [5,17,18]. Nakipuria et al. (2016) described a case series of posterior circulation infarctions following viper bite, proposing microvascular thrombosis as a likely mechanism [17].

In this case, the normal coagulation profile and absence of systemic bleeding suggest a localised vascular thrombotic or vasculitis process rather than classical VICC. The good outcome likely resulted from early recognition and supportive management.

Conclusion

This case highlights a rare neurological complication of Russell's viper envenoming leading to bilateral cerebellar infarction in a patient without venom-induced coagulopathy. Clinicians should be vigilant for this unusual presentation, particularly when patients develop atypical neurological symptoms, such as headache, after apparent recovery from the classical features of envenoming. Early neuroimaging and supportive therapy can result in full recovery.

Patient was quite happy with the care he received during his hospital stay and the recovery of his neurological symptoms at the three-month review.

Consent

Informed written consent was obtained from the patient for publication of the clinical data in this case report.

References

1. Kularatne SA. Epidemiology and clinical picture of the Russell's viper (*Daboia russelii russelii*) bite in Anuradhapura, Sri Lanka: A prospective study of 336 cases. Southeast Asian J Trop Med Public Health. 2003; 34:855–62.

2. Ameratunga B. Middle cerebral occlusion following Russell's viper bite. J Trop Med Hyg. 1972; 75:95–97.

3. Gawarammana I, Mendis S, Jeganathan K. Acute ischemic strokes due to bites by *Daboia russelii* in Sri Lanka – first authenticated case series. Toxicon. 2009; 54:421–28. doi:10.1016/j.toxicon.2009.05.006.

4. Subasinghe CJ, Sarathchandra C, Kandeepan T, Kulatunga A. Bilateral blindness following Russell's viper bite – a rare clinical presentation: A case report. J Med Case Rep. 2014; 8:99. doi:10.1186/1752-1947-8-99.

5. Rathnayaka RMMKN, Ranathunga PEAN. Late-onset of bilateral cerebral infarction following Russell's viper (*Daboia russelii*) bite. Sri Lankan J Anaesthesiol. 2017; 25:35–38. doi:10.4038/slja.v25i1.8165.

6. Hung DZ, Wu ML, Deng JF, Yang DY, Lin-Shiau SY. Multiple thrombotic occlusions of vessels after Russell's viper envenoming. Pharmacol Toxicol. 2002; 91:106–110. doi: 10.1034/j.1600-0773.2002.910303.x.

7. Kumar RM, Babu RP, Agrawal A. Multiple infarctions involving cerebral and cerebellar hemispheres following viper bite. J Med Soc. 2015; 29:51–53. doi:10.4103/0972-4958.158938.

8. Pothukuchi VK, Chepuri VR, Natta K, Madigani N, Kumar A. A rare case report of Russell's viper snakebite with ischemic stroke. Hong Kong J Emerg Med. 2018;25:95–7. doi:10.1177/1024907917735071.

9. Farid TM, Tu AT, el-Asmar MF. Effect of cerastobin, a thrombin-like enzyme from Cerastes viper venom, on human platelets. Haemostasis. 1990; 20:296–304. doi: 10.1159/000216141.

10. Basheer AR, el-Asmar MF, Soslau G. Characterization of a potent platelet aggregation inducer from *Cerastes cerastes* venom. Biochim Biophys Acta. 1995; 1250:97–109.

11. Dekhil H, Wasner A, Marrakchi N, Ayed M, Bon C, et al. Molecular cloning and expression of a functional snake venom serine proteinase with platelet aggregating activity from Cerastes viper. Biochemistry. 2003; 42:10609–18. doi: 10.1021/bi034790b.

12. Marrakchi N, Barbouche R, Guermazi S, Bon C, el-Ayeb M. Procoagulant and platelet-aggregating properties of cerastocytin from *Cerastes cerastes* venom. Toxicon. 1997; 35:261–72. doi: 10.1016/s0041-0101(96)00116-x.

13. Marrakchi N, Barbouche R, Guermazi S, Karoui H, Bon C, et al. Cerastotin, a serine protease from *Cerastes cerastes* venom, with platelet-aggregating and agglutinating properties. Eur J Biochem. 1997; 247:121–8. doi: 10.1111/j.1432-1033.1997.00121.x.

14. Laraba-Dejabri F, Martin-Eauclaire MF, Mauco G, Marchot P. Afaacytin, an alpha beta-fibrinogenase from *Cerastes cerastes* venom, activates purified factor X and induces serotonin release from human blood platelets. Eur J Biochem. 1995; 233:756–65. doi: 10.1111/j.1432-1033.1995.756_3.x.

15. 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. doi: 10.4103/0028-3886.55594.

16. Kalana M, Geoffrey KI. Current treatment for venom-induced consumption coagulopathy resulting from snakebite. PLoS Negl Trop Dis. 2014;8: e3220. doi: 10.1371/journal.pntd.0003220.

17. Nakipuria M, Shah A, Easwarappa VS, Selvapandian S, Ghosh S. A case series of posterior circulation ischaemic stroke following viper snake bite. J Neurosci Rural Pract. 2016; 7:462–5.doi.org/10.18231/j.ijn.2023.020.

18. Prasad UDN, Bandara JMRP, Senanayake HMS. Multiple cerebral and cerebellar infarctions following Russell's viper (*Daboia russelii*) envenomation - A case report. Clin Med Rev Case Rep.2020;7(6). doi.10.23937/2378-3656/1410313.