Case Study

Acute Ischemic Stroke After Russell's Viper Snake Bite, Rare Presentation: A Case Report

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Abstract

Snake bites are a global health hazard and are a noteworthy cause of mortality and morbidity especially in Southeast Asia. Cerebral complications after snake bite are rare. It can be attributed to various factors such as vasculitis, vasospasm, endothelial damage; toxin-induced procoagulant effect, and disseminated intravascular coagulation. We present a case of a previously healthy 22-year-old woman who suffered acute multiple cerebral infarctions following Russell's viper bite. Imaging revealed significant non-hemorrhagic infarctions in the left middle cerebral artery territory and right pons, indicating rare yet severe neurological complications of viper envenomation. The patient experienced serious complications including rhabdomyolysis and acute kidney injury, ultimately leading to her demise due to aspiration pneumonia and septic shock. This case underscores the potential neurological impact of viper envenomation and underscores the challenges in managing delayed procoagulant effects of snake venom, despite the administration of anti-snake venom. Early detection and intervention remain vital in addressing such devastating outcomes.

Keywords:Ischemic stroke; Cerebral infarct; Russell's viper; Snakebite; Polyvalent antisnake venom; daboia russelii; Acute ischemic stroke

Introduction

The World Health Organization (WHO) estimates that about 5 million snakebites occur each year, resulting in up to 2.7 million envenomings. WHO added snakebite envenoming to its priority list of neglected tropical diseases (NTDs) in June 2017[1]. A nationally representative study (Million Death Study) noted 45,900 annual snakebite deaths nationally. Around 90% of snakebites in India are caused by the 'big four' among the crawlers - common krait, Indian cobra, Russell's viper, and saw-scaled viper[1]. Many victims do not attend health centers or hospitals and instead rely on traditional treatments[2] thus the epidemiology of snake bites has not been adequately studied. About 94% of snakebite deaths occurred

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in rural areas, and 77% occurred out of hospitals[3]. Viper being the most common cause of bite [4], local envenomation is the frequent manifestation, followed by hemostatic abnormalities and neurotoxicity [5,6,7]. Coagulopathy if present is diagnostic of viper bites in South Asia. As reported, the most common species were Russell's vipers with a higher incidence of ischemic stroke than intracranial hemorrhage (ICH) [14]. Whereas in Borthrops ICH was frequently reported[8,14]. Ischemic stroke, when present, commonly involves anterior circulation. Here we describe a polyvalent anti-venom treated patient with multiple infarcts in the left MCA territory, right pons, and right parieto-occipital region with mid-line shift.

Case Report

We report a case of multiple ischemic infarctions following a viper bite in a previously healthy person. A 22-year-old pre-morbidly well female was admitted with a history of snake bite on her left foot above the ankle joint from the Satara district of Maharashtra, south India on 25th June 2023. The snake was identified as Russell's viper, as per the photograph shown by relatives (figure 1). A few minutes after the bite, the patient noticed minimal swelling over the Left foot and ankle joint. 30mins later, the patient developed breathing difficulty and consulted a nearby center, from there patient was referred to an ASV center, meanwhile patient deteriorated and became unconscious. The patient was taken to a civil hospital and Received 10 vials of polyvalent anti-snake venom, atropine-neostigmine within three hours of bite. The patient was referred to a tertiary center with intubated status after 8 hours of snake bites, meanwhile patient received 30 vials of polyvalent anti-snake venom& 4 unit FFP. On arrival (8 hours after bite) patient was moving all 4 limbs, off sedation. Complete blood count was normal with Serum Platelet of 1.56L, PT/aPTT / INR was normal, and serum urea 68 creatinine 2.2, CPK 3160. Suspected rhabdomyolysis with acute kidney injury. The next day (22 hours after the bite) patient was desaturated and the endotracheal tube was blocked with multiple large clots. X-ray showed right upper lobe haziness; due to O2 requirement bronchoscopy was withheld.

Figure 1: Photograph of Russel's viper taken from an attendant phone

48 hours after the bite ICU doctors noticed right hemiplegia with right plantar mute. D-dimer >69000, FDP 217. The 2D echo was normal.MRI+MRA showed an acute Large Non-haemorrhagic infarct in the Left MCA territory with acute infarct in right pons, a right parieto-occipital region with 7mm midline shift to the right side without herniation (figure 2).suspected hypercoagulable state andthe patient received LMWH, anticoagulants, and other supportive measures. The patient was discharged at the request& took the patient to Assam by road for 3 days.

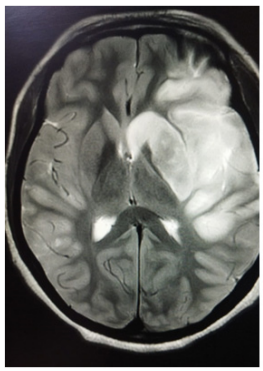
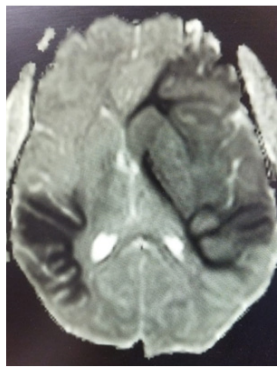


Figure 2 (a)



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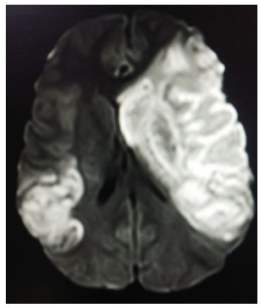

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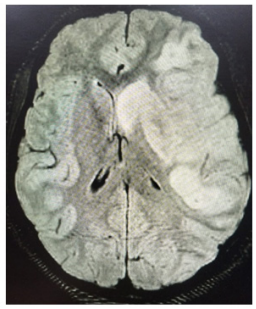

Figure 2 (d)

Figure 2 (e)

The patient received our hospital with a poor Glasgow coma scale (GCS) score of 4/15, increased breathing effort, Temperature of 104*F, coarse crepitation bilaterally, and hypotension. The patient was intubated and a repeat CT scan was taken showed hypo density in the left fronto-parieto-temporal lobes (figure 3),The chest X-ray showed bilateral patchy consolidation. Repeat the Routine investigation shown in Table 1.

Patient admitted to ICU with mechanical ventilator support, ionotropic, and antibiotics in view of aspiration pneumonia and septic shock.

Table 1: Laboratory investigations

Sl.no.	Laboratory parameters (units)	Value
1	Hb (g/dL)	10.9
2	PCV (%)	34.9
3	Platelets (x10³/ul)	452
4	TLC (x10³/ul)	28.80
5	PT (sec)	14.1
6	INR	1.24
8	D-dimer (mcg/mL)	4.28
9	Fibrinogen (mg/dL)	<200
10	LDH (U/L)	412
11	Bilirubin(mg/dL) Direct	0.9
		0.7
12	SGOT(IU/L)	73
13	SGPT(IU/L)	33
14	Albumin(g/dL)	2.7
16	Urea (mg/dL)	235
17	Creatinine(mg/dL)	4.6
18	CPK	230

However, she died on the 14th day of admission despite our best efforts.

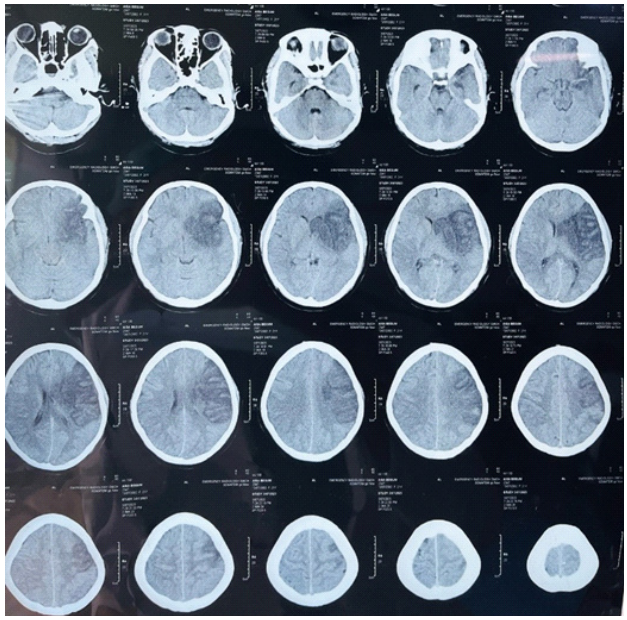

Figure (3): NCCT Brain-Area of hypodensity noted in left fronto-parieto-temporal lobes with loss of gray white matter. There is presence of mass effect as evident by midline shift of 4.5 mm towards right side & compression of ipsilateral lateral ventricle & dilatation of contralateral ventricle

Discussion

Viper bite is the most frequent snake bite in the Indian subcontinent. The presentation of envenomation by viper bite leads to local envenomation, followed by abnormal coagulation [6]. A systematic review yielded 87,590 snakebite cases in India (both fatal and non-fatal) from 2000 to 2019 based on screening 1417 papers and including 78 studies from 24 states or union territories in India, Russell's viper (*Daboia russelii*) constituted 43% of Case-fatality followed by other species [15]. The various toxins present in the viper venom can be categorized both as pro-coagulant and anticoagulant. The toxins with well-established pro-coagulant/platelet aggregating properties are cerastobin [9], factor IVa [10], cerastocytin [11], cerastotin [12], and afaacytin [13]. These various protein products have thrombin-like enzymatic activity. Different toxins activate different parts of the coagulation cascade [9-13]. Their activity is inhibited by monoclonal antibodies against GP1b or GPIIb/IIIa or thrombin receptors.

In a Scoping Study on stroke associated with snake envenomation between Jan 1995 to Oct 2018, 83 published cases were reviewed. 66.3% of the cases were younger than 50 years of age. The mean time for the onset of the symptoms is 23.8±10.9 hours after exposure(In our patient cerebral infarction was noticed 48 hours after the snake bite) 77.1% of the cases were found to have an ischemic stroke, 20.5% with intracranial hemorrhage, and both infarction and hemorrhage in 2.4%[14]. The infarct commonly involves the anterior circulation, with hemiparesis being the frequent presentation[16-19]. Cerebral infarction after snake envenomation is a complex multifactorial mechanism. It includes direct cardiotoxic effects of venom leading to dysrhythmias. This may cause cardiac thromboembolism and hyperviscosity due to hypovolemia and hypofusion secondary to hypotension.In this patient, ischemic infarctions due to hypotension are unlikely as she was normotensive. The possibility of a cardiac source of embolization was excluded.

Disseminated intravascular coagulation is also an associated risk factor in viper bite patients. Disseminated intravascular coagulation can be a cause of neurological disorders, largely due to vessel occlusion [20]. The toxin itself can cause vasospasm which can lead to a cerebrovascular accident.

In a study by Thomas, et al [21] of the 33 patients with envenomation by Bothrops lanceolatus who had not received ASV or received ASV after 8 hours of envenomation, 14% developed thrombotic complications and 4 of the 14 patients who had not received ASV died. Of the 70 patients who received ASV within 6 hours of envenomation, no thrombotic complication. Our patient, despite treatment with ASV within 3 hours of envenomation developed delayed cerebral infarction on the second day. Thrombin-like activities of venoms are not inhibited by heparin [22]. It has never been proven effective in clinical trials [23], even in victims of Russell's vipers (*D.russelii* and *D. siamensis*) envenoming in whom there is some slight theoretical basis for its use [24] Heparin increases the risk of hemorrhage in victims of viper bites and should never be risked in the treatment of these patients[25].

Conclusion

After a viper bite, neurological manifestations can be attributed to various reasons, such as toxin-induced vasculitis, procoagulant effect, endothelial damage, disseminated intravascular coagulation, and hypotension. Though large-vessel thrombosis and infarction are rare clinical entities associated with viper envenomation, they should be considered in Indian patients.Furthermore, this case depicts the fact that even after the early administration of polyvalent antisnake venom, delayed procoagulant properties of snake venom can't be completely preventable. The early detection of the viper bite and the administration of anti-snake venom may prevent this rare but devastating complication.

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Consent to Publish: Consent was obtained from the patient's parents for the publication of this Case Report and any accompanying images.

Consent form is available on request.

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