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Case Series

A case series of posterior circulation ischaemic stroke following viper snake bite

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ABSTRACT

Snakebite is a common problem in Rural India. Viper snake bite is India's most common cause of fatal snake bites. They are usually associated with hemorrhagic complications. Ischemic complications following viper bite are uncommon and have only been reported by a few authors. A posterior circulation stroke is even rarer as it is challenging to diagnose. It is also fatal if there is a delay in diagnosis. We report a series of 5 patients with posterior circulation infarcts following viper envenomation confirmed by MRI Brain. We review published literature and discuss the likely pathogenesis of the ischaemic stroke following a viper bite. This study stresses the need for a higher degree of suspicion for posterior circulation stroke following snakebite and an early radiological diagnosis. Following diagnosis, adequate hydration, maintaining blood pressure, antiplatelets, and ICP lowering measures are essential. Snakebite should be considered a differential diagnosis in a young patient with stroke, especially in rural areas.

1. Introduction

Snakebite is a health hazard often faced by the rural population in tropical and subtropical countries with heavy rainfall and humid climate.[1] Snakebite is a common cause of morbidity and mortality in India. According to older estimates, approximately 2,00,000 fall prey to snake bites annually in India, with an estimated mortality ranging from 20,000 to 50,000 cases. Mohapatra et al. estimated 45,900 annual snakebite deaths nationally or an annual age-standardized rate of 4.1/100,000, with higher rates in rural areas (5.4) and with the highest rate in the state of Andhra Pradesh (6.2).[2]

Viperidae species consisting of Russell's viper (Daboia russelli) is India's most common cause of fatal snake bites.[3,4] Viper bites are more common in South India than in North India.[4] The common clinical characteristics of viper bite include local cellulitis, renal failure, and systemic hemorrhagic manifestation, including intracranial and subarachnoid hemorrhage. The neurological manifestations are mostly confined to the cranial nerves, commonly causing ptosis and external ophthalmoplegia. Only a few authors have described ischemic infarction following viper envenomation.[5–9] In all of these cases, ischemic infarction involved the anterior circulation. Only five cases of posterior circulation infarcts after snakebite have been reported to date,[10–14] out of which only three have been documented after a viper bite.[12–14] Paraplegia following snake bite is a rarely reported manifestation, only reported once previously.[15] We report 5 cases with the unusual presentation of posterior circulation infarct following a viper snake bite.

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2. Case Report

2.1. Patient A

A previously healthy 50-year-old woman was bitten by a snake on her left leg while working in a paddy field. She presented to a nearby primary health centre with progressively worsening sensorium, bilateral lower limb weakness, and respiratory distress. Her left foot had two deep bite marks with local swelling, redness, and cellulitis. Onlookers identified the snake as a Viper. She was intubated, put on ventilator support, and administered 20 equine polyvalent anti-snake venom vials. Her GCS was E2VtM3. Platelet counts, Bleeding time, clotting time, and APTT were within normal limits. Intracerebral haemorrhage was suspected, and CT Brain showed an evolving left PCA territory infarct. MRI Brain done 24 hours later confirmed it to be Left PCA territory Infarct (Figure 1 (1). CT Angiogram showed a significantly attenuated left-sided posterior cerebral artery from the P2 segment. (Figure 1(2)). She was started on anti-platelets, statins, Low molecular weight heparin, and antibiotics. She developed severe pulmonary oedema and died two days later.

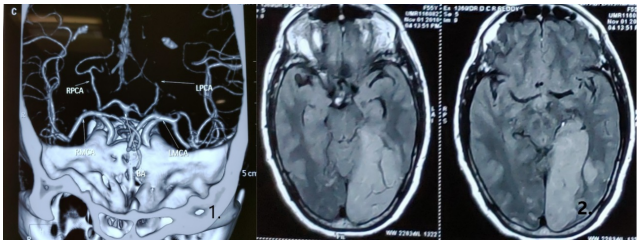

Fig. 1: Patient **A** - 1. Left PCA P2 segment attenuation 2. Left PCA infarct

2.2. Patient B

A 37-year-old lady with no known comorbidities presented with a viper snake bite over her right calf, following which she developed local swelling and pain over the right leg. She was started on ASV but developed an allergic reaction; hence it was stopped. She had seizures, and thus CT brain revealed a large right cerebellar infarction with ventricular dilatation (Figure 2B). The platelet count was low. It was corrected, and she underwent decompressive posterior fossa craniotomy because of the mass effect. She made a good recovery and was discharged without any sequelae.

2.3. Patient C

A 54-year-old male had a viper snake bite while working on the farm on a rainy day. He presented to the hospital in an unconscious state and was intubated. The platelet count was 28,000. Urea and creatine were elevated, and he was started on dialysis. MRI showed multiple infarcts – Right PCA, Left MCA territory lacunar infarcts (Figure 2C). He was tracheostomised. He had a gradual recovery.

2.4. Patient D

A fifty-eight-year-old gentleman presented with a history of snakebite over the left leg. After half an hour, he was found to be unresponsive. He was started on 15 vials of ASV (within 2 to 3 hours), and he was intubated given the low GCS. There was bleeding from the snakebite site. INR was 3.68, and he was transfused 4 units of FFP.CPK was elevated (3717 U/L). D-Dimer was 4.19 g/L. The platelet count was 6,04,000. MRI brain showed acute infarct predominantly involving cerebral cortices in the right parieto-occipital posterior frontal regions, capsulo-ganglionic region, caudate nucleus, hippocampal region, bilateral thalamus, and both cerebellar cortical grey mater (Figure 2D). He recovered over ten days and was extubated. He had residual mild lower limb weakness.

2.5. Patient E

A 55-year gentleman with a viper bite presented to the hospital with seizures, hemiparesis, and bleeding manifestations. He received 11 vials of ASV and was intubated because of prolonged status epilepticus. Baseline investigation revealed prolonged bleeding time (>15 mins), clotting time (>60 mins), leucocytosis, and elevated CPK(3571 U/L). He required increasing doses of inotropes. MRI Brain showed multiple acute infarcts in the brainstem, Cerebellum, and right caudate (Figure 2E). There was no improvement of consciousness, and as brainstem reflexes were absent, he was declared brain dead.

3. Discussion

The most typical cause of snakebite in India is due to the Viper species.[3,4] Clinical manifestations depend on the severity of envenomation. Viper venom contains procoagulant arginine ester hydrolase, an enzyme similar to thrombin that clots fibrinogen and aggregates platelets, forming intravascular fibrin microthrombi.[16] This causes consumptive coagulopathy and leads to bleeding. The viper venom also contains several anticoagulant proteins that activate factors V, IX, X, XIII or cause fibrinolysis and hemorrhage.[17] These hemorrhagins and hemolysins cause damage to the vessel wall and, along with the coagulation defects, may result in severe bleeding. Due to this 'pro-bleeding' background, the common neurological manifestations are due to intra-cerebral or subarachnoid haemorrhage, with a rare occurrence of ischaemic infarctions. Mosquera Et al.[18] have reported strokes in only 8 out of the 309 patients (2.6%) in their series of snake bites. Of these, 7 were hemorrhagic strokes, and only one was ischaemic.

Table 1: Summary of patients

Patient	Age	Sex	Location of stroke	Lab Findings	Outcome
A	50	Female	Left PCA	Normal	Death
B	37	Female	Right cerebellar infarct with mass effect and Hydrocephalus	Thrombocytopenia	Underwent posterior fossa decompression surgery- recovery
C	54	Male	Right PCA + Left MCA lacunar	Thrombocytopenia Elevated urea, creatinine, CPK	Sequalae- Hemianopia
D	58	Male	Right PCA+ Right gangliocapsular+ Bilateral thalamus	INR- 3.68, elevated CPK Thrombocytopenia	Sequalae- Lower limb weakness
E	55	Male	Brainstem, Cerebellum, right caudate	Elevated bleeding time, clotting time and CPK, Thrombocytopenia	Death

Table 2: Summary of case reports of patients with posterior circulation infarcts after snake bite

Case Reports	Age sex	Presentation	Snake	Labs	Diagnosis	Outcome
Krishnan et al[10]	14 M	Ptosis + Altered sensorium. Unequal & Sluggish pupils, Hypotonia, Plantar-Extensors	Not Known	BT,CT, Platelets - Normal	CT Brain - Bilateral Cerebellar +Rt Occipital Infarct with mass effect	Death
Gouda et al.[12]	40 F	Altered sensorium, GCS 9/15 Hypotonia, Plantar-Extensor	Viper	PT, APTT prolonged INR-4.44 CPK Elevated	CT & MRI – B/L cerebellum + Occipital Infarct	Recovered with residual gait ataxia
Gunchan et al.[11]	36 M	Seizures, Anisocoria, Gaze palsy, Hypotonia, plantars Extensor	Not Known	Normal coagulation profile	MRI- Left Cerebellum + Bilateral occipital+ Bilateral Thalamus	Recovered
Gupta et al.[13]	48 F	Hematuria, hematemesis. Drowsiness (3^{rd} day), Hypotonia, Plantar extensor	Viper	Prolonged BT, CT Xray – Pulmonary edema	CT- Left Cerebellum with mass effect + Hydrocephalus	External Ventricular Drain Recovered
Deepu et al.[14]	48 F	Altered sensorium, respiratory distress GCS 10	Viper	Normal coagulation	Ct- Right Cerebellum with mass effect and midline shift	Death

Previous studies have proposed the following mechanisms for the occurrence of infarctions.

1. Hypotension - Sweating, vomiting, reduced fluid intake, and bleeding tendencies lead to hypovolemia and hypotension. Loss of vasomotor tone due to viper toxin leads to hypotension. This hypotension leads to poor perfusion and watershed infarcts.
2. Hypercoagulability – due to the procoagulant activity of viper venom or consumptive coagulopathy causing procoagulant state.
3. Endothelial injury - due to toxic vasculitis can lead to thrombosis.
4. Cardiotoxicity – Dysarrythmia due to the effect of viper venom may lead to cardiac thromboembolism.

The infarcts in our patients are not in the classical watershed territory and therefore do not suggest hypotension as a cause. Bleeding and Clotting time was normal and ruled out coagulopathy as a cause in Patient A. The possible reason for infarct in this patient's posterior circulation is toxic vasculitis caused by injury to the endothelium by snake venom toxin. Only the posterior circulation is affected, and the anterior circulation is spared. Ct Angiogram ruled out any pre-existing abnormality in the posterior circulatory blood vessel wall that could be considered as a cause for the infarct. However, we do not have DSA studies to prove this. Previous case reports have discussed similar cases where the patient died despite normal coagulation parameters.[10,14] Patient A also had paraplegia that could be explained as an MRI of the spine could not be done. Singh et al.[15] have

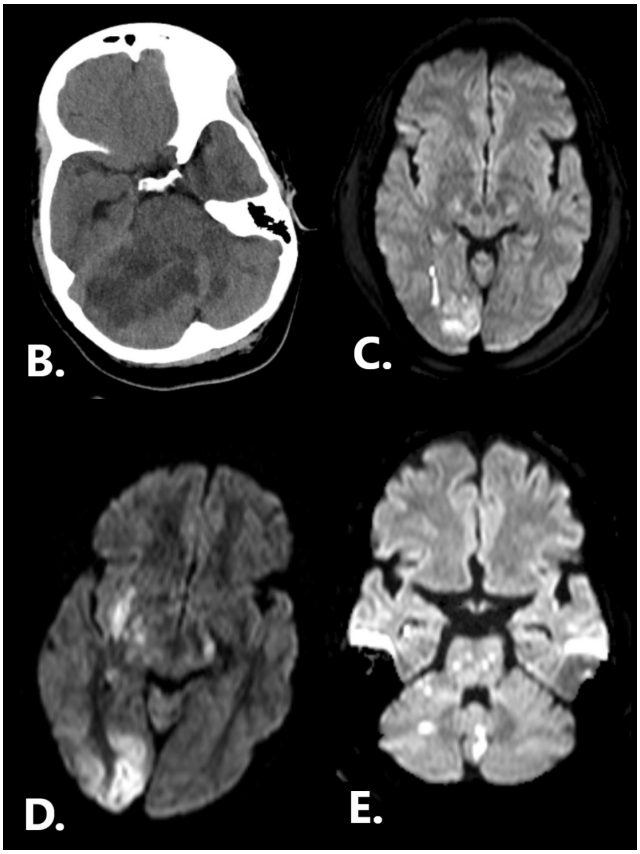

Fig. 2: B - (Patient B) Right cerebellar infarct with mass effect on fourth ventricle and brainstem, **C** - (Patient C) Right PCA territory Acute Infarct, **D** – (Patient D) Right PCA and Right Gangliocapsular acute infarct, **E**- (Patient E) Multiple Brainstem and cerebellar infarcts

reported a case of paraplegia following a viper bite due to dorsal spinal cord involvement.

Four of the five patients had some form of coagulopathy (80%). All Four had thrombocytopenia at presentation, out of which two (Patients D & E) had both thrombocytopenia and elevated clotting time suggestive of consumptive coagulopathy (DIC) as the cause of the infarct. The other two patients (B, C) had only thrombocytopenia with normal clotting time suggestive of early/mild DIC; hence a combination of toxic vasculitis and DIC could explain the infarct.

In our series, two patients presented with seizures despite having only posterior fossa infarcts (B, E). Previously Guncha et al.[11] have reported a similar patient who presented with seizures. Most patients in our series and literature have presented with altered sensorium, drowsiness, and local limb swelling. Altered sensorium after a snake bite may be related to the vasculitis caused by the venom resulting in cerebral oedema or toxic encephalopathy. Thus it may be difficult to diagnose a patient with a posterior circulation stroke based on clinical presentation alone, and a high degree of suspicion is needed.

The high mortality rate among patients with posterior circulation infarcts after snake bites has been documented in the literature. In our series, mortality was 40%. This corresponds to earlier case reports.[10–14] The clinical outcome is better in patients with supratentorial infarcts following snake bite than posterior fossa infarcts.[5–9] This may be due to the rapid rise of intracranial pressure associated with posterior fossa infarcts. Patient B underwent a Decompressive craniectomy due to the mass effect and recovered well after normalisation of ICP. Deepu et al.[14] reported a patient presenting with a cerebellar infarct 20 hours after a snake bite. Here despite surgery, the patient died. This could be due to the late presentation and higher envenomation.

This case series emphasises the need for high suspicion of posterior circulation infarcts following snake bite as they can be fatal. Early diagnosis and reduction of ICP help decrease mortality. Hydration, blood pressure, and CVP should be kept optimal in all snakebite patients. ASV, if indicated, administered early may help prevent coagulopathies and prevent infarcts. Thomas et al.[19] discussed that thrombotic complications were prevented by administering ASV within 6 hours. Local infection should be treated aggressively with antibiotics and antiseptic dressings. Normal platelet counts and coagulation profiles may conceal the early stages of DIC. Snakebite should be considered an essential differential in young patients with stroke, especially in rural areas.

4. Conclusion

Posterior Circulation Stroke is a rare but lethal complication after Russel Viper's snake bite. Due to the non-specific presentation, a high degree of suspicion is necessary to diagnose it. Early diagnosis and reduction of ICP help in improving survival. Snakebite should be considered an important differential in young patients with stroke, especially in rural areas.

5. Conflict of Interest

None.

6. Source of Funding

None.

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