CASE REPORT

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Ischemic and Hemorrhagic Stroke Consequent to Snake Bite

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ABSTRACT

Snakebite remains a major cause of morbidity and mortality in rural India, particularly due to viper envenomation. While hemorrhagic complications are common, ischemic stroke following viper bite is rare, and concurrent ischemic and hemorrhagic lesions are exceptionally uncommon. We report a case of a 45-year-old male who developed multiple cerebral and cerebellar infarcts with secondary hemorrhagic conversion following a viper bite. The patient presented with vomiting, headache, and diplopia two hours after being bitten on the right foot. Laboratory findings revealed thrombocytopenia, prolonged clotting time, and elevated D-dimer levels. MRI showed multiple acute infarcts at watershed zones in bilateral cerebral and cerebellar hemispheres, with secondary hemorrhagic conversion in the right parieto-occipital region (PCA territory). The patient was managed with intravenous fluids, antibiotics, and anti-snake venom (ASV), followed by antiplatelet therapy after coagulation normalization. Early administration of ASV and careful monitoring led to clinical recovery. This case highlights the rare occurrence of concurrent ischemic and hemorrhagic stroke following viper envenomation and emphasizes the importance of timely diagnosis and intervention to prevent fatal outcomes.

Keywords: Snakebite, Viper, Ischemic stroke, Hemorrhagic stroke, Anti-snake venom

INTRODUCTION

Snakebite is a significant public health issue in tropical and subtropical regions, particularly in India, where approximately 2,00,000 cases and up to 50,000 deaths occur annually. [1,2] Most fatal cases are attributed to viper bites, particularly those of *Daboia russelii* (Russell's viper), known for its potent hemotoxic venom. Common clinical manifestations include local swelling, coagulopathy, and hemorrhagic complications such as intracerebral or subarachnoid hemorrhage.[3,4]

Ischemic complications are exceedingly rare and have been sporadically reported in medical literature. [5,6] The underlying mechanisms include venom-induced hypercoagulability, vascular endothelial damage, and disseminated intravascular coagulation (DIC).[7] Posterior circulation involvement is even rarer and poses diagnostic challenges. This report presents an unusual case of combined ischemic and hemorrhagic stroke following viper envenomation and discusses the potential mechanisms and clinical implications.

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CASE REPORT

A 45-year-old male presented to the emergency department two hours after sustaining a snakebite on the sole of his right foot. The species was unidentified, but fang marks were evident without active bleeding. A tourniquet had been applied above the site immediately after the bite. The patient subsequently developed vomiting, headache, and diplopia. No history of petechiae, hematuria, ophthalmoplegia, ptosis, or limb weakness.

Examination: Vital parameters were stable (BP 120/80 mmHg, pulse 80/min, SpO_2 96%). The patient was conscious and oriented, with mild visual disturbance but no motor or sensory deficit. Local examination revealed swelling and blackish discoloration at the bite site.

Investigations: Laboratory tests showed elevated serum creatinine (1.4 mg/dL), thrombocytopenia (86,000/mm³), prolonged 20-minute whole blood clotting time, and raised D-dimer (4180 ng/mL). Routine work-up for stroke like lipid profile, homocysteine, B12, and ESR are normal. 2-D echo and carotid Doppler unremarkable. MRI brain revealed multiple acute infarcts in bilateral cerebral and cerebellar hemispheres at watershed zones, with secondary hemorrhagic conversion in the right parieto-occipital region (PCA territory).

Management and Outcome: The patient received intravenous fluids, antibiotics, and 100 units of polyvalent anti-snake venom (ASV), followed by an additional 100 units after reassessment. Coagulation parameters gradually normalized, and neurological symptoms improved. After stabilization, single antiplatelet therapy was initiated. The patient was monitored in the ICU and showed progressive recovery with resolution of diplopia and no residual neurological deficit at discharge.

After treatment repeat CT scan shows resolved hemorrhagic stroke.

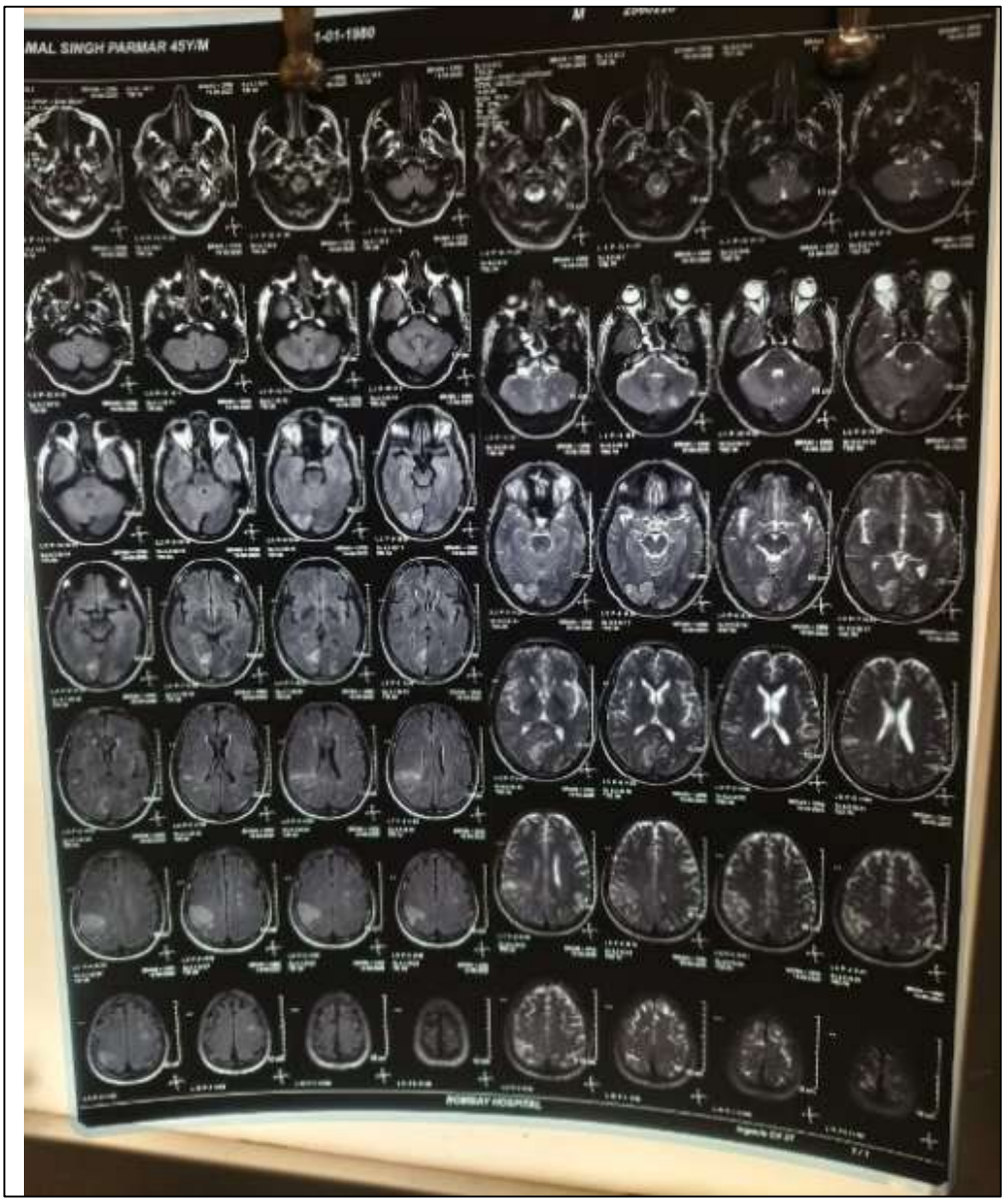

Figure 1: MRI brain showing multiple acute infarcts in bilateral cerebral and cerebellar hemispheres (watershed zones) with secondary hemorrhagic conversion in the right parieto-occipital region (PCA territory)

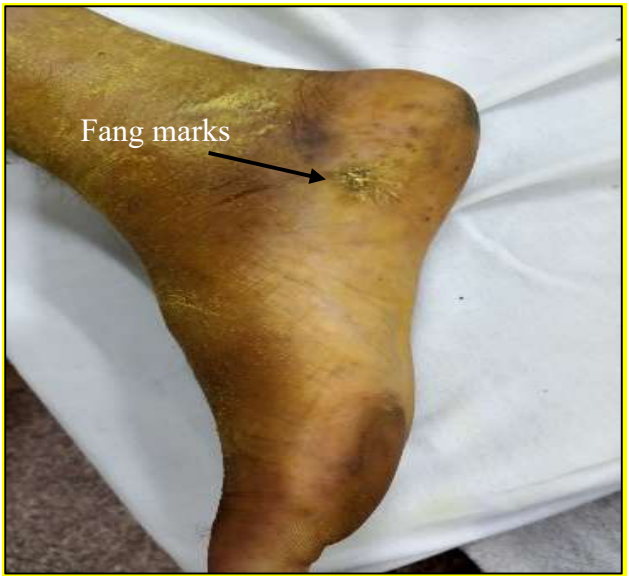

Figure 2: Fang marks on the sole of the right foot showing local swelling and blackish discoloration at the site of envenomation

DISCUSSION

Viper envenomation is known to cause systemic complications due to its complex venom composition, which includes proteolytic enzymes, phospholipase A_2, and metalloproteinases. These components lead to endothelial damage, activation of coagulation cascades, and subsequent tissue injury. [8,9] While hemorrhagic complications are frequently reported, ischemic strokes remain rare and are attributed to venom-induced hypercoagulability, vascular spasm, and toxin-mediated endothelial dysfunction. [5,10]

Cerebral infarction following viper bite may occur due to a combination of factors such as venom-induced DIC, microthrombi formation, and hypotension from hypovolemia leading to watershed infarcts.[11] The present case demonstrated bilateral watershed infarcts and a secondary hemorrhagic lesion, indicating an initial hypercoagulable phase followed by hypocoagulability. Similar cases have been reported by Sowmya AN et al.[12] and Jeevagan V et al.[13], who emphasized that early administration of ASV significantly improves neurological outcomes by neutralizing circulating venom toxins.

Clinically, distinguishing between venom-induced neurotoxicity and ischemic events is challenging. Symptoms like diplopia or cranial nerve involvement may initially mimic neurotoxic effects rather than stroke, delaying diagnosis. Therefore, clinicians must maintain a high index of suspicion and promptly perform neuroimaging in snakebite victims presenting with neurological deficits.

Early ASV therapy remains the cornerstone of management and can prevent irreversible vascular and neurological injury. Supportive measures, correction of coagulopathy, and careful timing of antiplatelet initiation are essential to avoid secondary hemorrhagic complications. This case underscores the importance of early recognition, timely ASV administration, and vigilant monitoring in achieving favourable outcomes in such rare presentations.

CONCLUSION

Concurrent ischemic and hemorrhagic stroke following viper envenomation is a rare but serious complication. Prompt administration of anti-snake venom, correction of coagulopathy, and early neuroimaging are crucial for favorable outcomes. Clinicians should remain alert to neurological manifestations in all snakebite cases for timely intervention and prevention of fatal consequences.

Individual Author's Contribution: AT contributed to study conception, design, and manuscript preparation. **VJS** participated in all stages, including data collection and analysis. **PPB** contributed in study design.

Availability of data: The data that support the findings of this study are available from the corresponding author on reasonable request.

Declaration of Non-use of generative AI Tools: The authors declare that no Artificial intelligence tools were used during the preparation of this manuscript including writing. All work is the original contribution of the authors.

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