International Journal Dental and Medical Sciences Research
Volume 6, Issue 4, July. – Aug. 2024 pp 138-140 www.ijdmsrjournal.com ISSN: 2582-6018

A Rare Case Report of Russell's Viper Snakebite with Ischemic Stroke

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Date of Submission: 10-07-2024 Date of Acceptance: 20-07-2024

I. INTRODUCTION:

Snakebite envenomation is a commonly encountered emergency in tropical countries with potentially fatal complications. Russell's viper is the leading cause of fatal snakebite in India. The clinical manifestations include local swelling and necrosis, renal failure, and coagulopathy complicated by hemorrhagic manifestations such as pituitary and intracerebral hemorrhage. There are only a few case reports of snakebites associated with ischemic stroke. We report a case of an18-year-old male who developed expressive aphasia and hemiplegia following a Russell's viper bite. Magnetic resonance imaging of the brain revealed infarcts in the left MCA (middle cerebral artery) territory and watershed zones. In this scenario, the possible mechanisms for cerebral infarction are toxin-induced vasculitis, endothelial damage, and hypotension. Although ischemic stroke is rare in viper bites, if treatment is started early, neurological deficits can be minimized.

II. CASE REPORT:

An 18-year-old previously healthy male patient presented to triage, after 48 hours of Russell's viper snakebite over the right upperlimb. Initially patient went to a local hospital where he was treated with 10vials of ASV and injection of adsorbed tetanus toxoid and was referred to this facility because of ptosis. The patient complained of severe pain and swelling of the right upper limb and bleeding from the bite site. He had spontaneous eye opening, verbal response, and spontaneous limb movements and his GCS was 15/15 (E4V5M6). He had hypotension with BP- 90/60 mmHg, pulse rate- 76/min, SPO2- 99% with room air, and respiratory rate- 16/min. Local examination showed two deep fang marks with erythema and edema. On systemic examination, ptosis was noticed. No other systemic hematological manifestations were observed clinically. Twenty-minute whole blood clotting test (WBCT) was negative. He was started on ASV as per guidelines along with Atropine and Neostigmine. Adequate IV fluids were given and the patient's blood pressure improved (110/90 mmHg). A course of antibiotic injection of Amoxyclav was started. Laboratory investigations on day 1 of admission (day 3 of snakebite) showed elevated serum creatinine(3.0mg/dl) and blood urea(52mg/dl). Complete blood picture and liver function tests were within normal range. ECG and Chest X-ray were normal.

On day 2 of admission(the day of the snakebite), the patient developed expressive aphasia and right hemiplegia. He was not opening his eyes to verbal response with a GCS- 8/15 (E2V1M5). Neurological examination revealed right hemiplegia and expressive aphasia while he remained hemodynamically stable. WBCT 20 was negative. MRI of the brain showed acute infarcts in the left MCA territory and watershed zones with mass effects and midline shift. Biochemical investigations showed elevated serum creatinine (1.4mg/dl) decreased since admission, mild thrombocytopenia (1.21lakh), elevated prothrombin time- 17.40 sec (normal <14), and INR- 1.46 (normal <1.1). Lipid profile, ECG, and 2D Echo were normal. The patient was treated with aspirin and clopidogrel.The patientshowed improvement in general condition, motor power (3/5), and speech after 1week of treatment.

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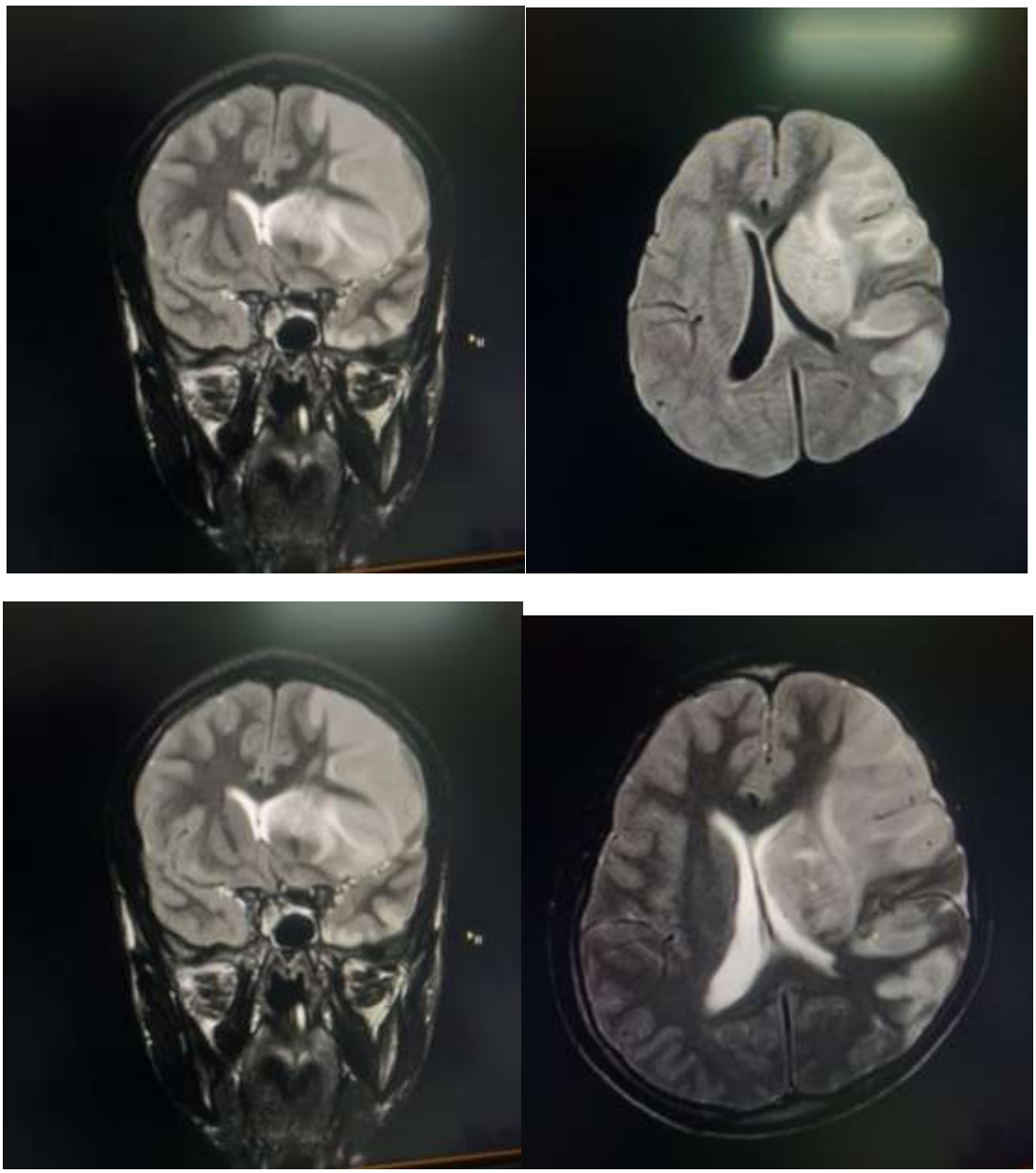

III. DISCUSSION:

Cerebral complications, particularly ischemic complications after snakebite are rare. Viper venom is a complex toxin with rich components principally affecting hemostatic mechanisms. The mechanisms by which cerebral infarction occurs in snake envenomation can be multifactorial and are as follows:

- The venom has anticoagulant and procoagulant effects

which leads to small and large vessel microthrombi leading to cerebral infarction.

- Haemorrhagins are complement-mediated toxic components of snake venom, which cause severe vascular spasm, endothelial damage, and increased vascular permeability. All these lead to toxic vasculitis and result in thrombosis.
- Hypotension can occur due to hypovolemia from sweating, vomiting, decreased fluid intake, and bleeding tendencies leading to low flow state and watershed infarcts.
- Hyperviscosity caused by hypovolemia and hypoperfusion secondary to hypotension and hypercoagulation may lead to vascular occlusion.
- Direct cardiotoxic effects of venom can lead to dysrhythmias causing cardiac thromboembolism.

- Preexisting procoagulant state, due to deficiencies of protein C or S or antithrombin IIIand antiphospholipid antibodies could account for thrombosis in large vessels.

Our patient was young, had no vascular risk factors and the MR angiogram was normal. Cerebral infarction in this patient could haveresulted from

1. Toxic vasculitis or toxin-induced vascular spasm and endothelial damage.
2. Hypotension leading to hypoperfusion, as MRI brain showed involvement of watershed zones (as the patient had hypotension initially).

IV. CONCLUSION:

Ischemic stroke following snakebite is rare. The learning point from this case is that snakebite should be one of the differential diagnoses for any young patient with a stroke. Early administration of ASV is essential to neutralize the total circulating venom before it gets fixed to the tissues. Physicians working in rural areas should look for such less recognized etiologies of stroke in tropical countries, as early administration of ASV may have a role in the prevention of thrombotic complications like ischemic stroke.

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