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A Rare Case of Snake Bite with Acute Ischemic Dual Circulation Stroke

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KEYWORDS:
Viper snake bite
Thrombotic stroke
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ABSTRACT:
We present the case of a 26-year-old male patient who was admitted with a history of los of consciousness for around 1 hour following a Viper snake bite. Subsequent investigatio revealed multiple infarcts in the right frontal, bilateral parietal lobe, bilateral centrun semiovale, head of the right caudate nucleus and bilateral cerebellar hemispheresindicatin involvement of both ACA and PCA territories. He was treated with polyvalent anti-snak venom, a single antiplatelet drug and other supportive treatments. The patient improve symptomatically and was discharged with a single antiplatelet drug. A follow-up visit 1 days later showed a dramatic improvement. The antiplatelet drug was stopped and th patient was reassured.
Although viper bite has been associated with haemorrhagic strokes, thrombotic strok following viper bite is a rare occurrence. To the best of our knowledge, this is one of th very few case reports of thrombotic stroke involving both the ACA and PCA territorie caused by Viper bite. This may be due to a number of potential causes includin endothelial injury, vasospasm, vasculitis, the procoagulant action of toxins and DIC.

Background

The WHO reports that India has between 35,000 and 50,000 snake bite deaths annually, making it the country with the largest number of snake bite cases globally. 10-80% of snake bites result in envenomation.[1] After neurotoxicity and hemostatic problems, viper snake bites are the most common cause of local envenomation. Viper bites have a very uncommon correlation with cerebrovascular accidents in South Asia; these incidents are often hemorrhagic in nature and infarct-related seldom.[1]

Case Report

A26-year-old male patient presented with loss of consciousness for around one Hour after a viper bite in his right dorsum of foot near the 4th and 5th toes while working in his farm at around 5.30pm. Later, his relatives took him to a local hospital and the patient regained consciousness, but he was still drowsy and had four episodes of blood vomitus (hematemesis). He was given five vials of anti-snake venom and other symptomatic measures and referred to our hospital. On examination, the patient had a GCS of 4/15 and had to be intubated in the emergency department of JSS Hospital. Blood pressure was 110/80 mmHg measured in the right arm in the supine position. Pulse rate was 120 bpm, respiratory rate was 26 cpm and SpO2 was 98% on PRVC support. The patient had active oral bleeding and subconjunctival haemorrhage in the left eye. Frank haematuria was also noted. On local examination, fang marks were observed in the right dorsum of the foot near the 4th and 5th toes, along with Local signs of inflammation, multiple blebs, swelling of the right foot up to the knee and ecchymosis over the right thigh and shifted to the ICU complex. The patient was extubated on day 5 and kept for observation for 1 day in the ICU complex to look for respiratory distress. After 6 days, the patient's vitals were stable and he was shifted to the ward. The patient then noticed weakness in the left upper and lower limbs and was also not able to sit on his own as he was swaying. On examination, we noticed that higher motor function was normal with no cranial nerve palsies and a paucity of movements of the left upper and lower limb with poor left-hand grip and left plantar extensor. The bilateral finger-nose-finger test was impaired and truncal ataxia was noticed.

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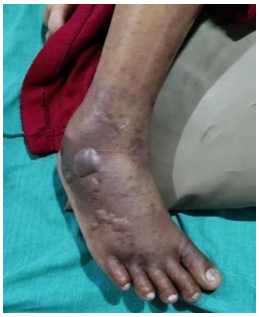

Figure 1 - Site of Snake Bite Showing Fang Marks, Multiple Blebs and Swelling of Right Foot

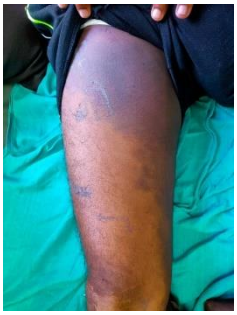

Figure 2 - Showing Ecchymosis over the Right Thigh

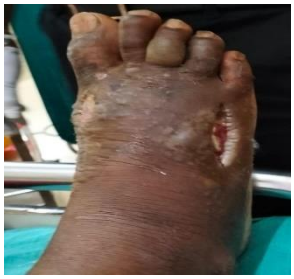

Figure 3 – Showing Fasciotomy done on Day 4 I/V/O Compartment Syndrome

Investigations

Date	28/07/22	30/07/22	31/07/22	02/08/22	04/08/22
Hemoglobin(g/dl)	13.3	10.5	8.7	9.9	10.7
Total leucocyte count (per cubic mm)	24170	21200	17010	12840	8990
Platelet (per cubic mm)	2.03	1.47	1.64	2.75	4.08
Urea/creatinine (mg/dl)	38/1.84	51/1.08	41/0.9	40/0.64	27/0.58
Sodium/ potassium/chloride (mEq/L)	136/3.7/107	139/4.1/105	142/3.9/108	138/4.1/108	134/4/99
Prothrombin time/INR/activated prothrombin time (seconds)	>3mins/-/>3mins	15.9/1.25/28.5	14.5/1.13/27.5	13.3/1.03/27.08	12.5/0.96

Table 1

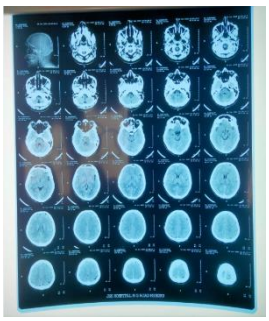

Figure 4 - Showing CT Brain done on Day 1 – Normal

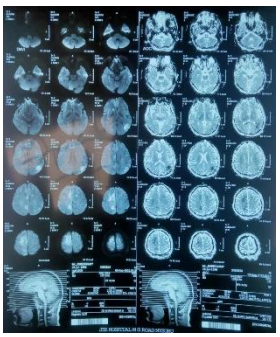

Figure 5. Showing dADC done on day 7 - acute infarct in right frontal, bilateral parietal lobe, bilateral centrum semiovale, head of right caudate nucleus and bilateral cerebellar hemispheres

Management

The patient was intubated, catheterised and Ryle's tube was inserted in the EMD red zone JSSH and stabilized. He was given 5 vials of polyvalent anti-snake venom and other symptomatic treatment and shifted to the ICU complex. The patient was monitored with WBCT and after giving a total of 30 vials of polyvalent anti-snake venom, repeat WBCT was< 6minutes.Patient was kept Nil by mouth in view of hematemesis and started on intravenous dextrose 25%. The Electrocardiogram showed sinus tachycardia. Echocardiography showed normal valves and chambers with an ejection fraction of 65 percent and no regional wall motion abnormality. The initial CT brain scan on day 1 revealed a normal study. A complete hemogram showed neutrophilic leucocytosis and on the 4th day, the patient's Hb had dropped to 8.7 g/dl. He was given 2 units of PRBC and Hb improved to 10.7 g/dl. Patient PT/INR and APTT were deranged and transfused around 8 units of FFP. Repeat PT/INR and APTT were normalised on day 4and

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his active bleeding was stopped. Started on RT feed 200ml Q 3rd hourly and later allowed orally on day 6.The patient had local signs of envenomation in the right dorsum of the foot and a surgery opinion was taken. Regular MgSO4 dressing and right limb elevation were done and later patient underwent a fasciotomy in view of compartment syndrome on day4 . Patient had AKI at the time of presentation and RFT was monitored serially and normalized on day 8 . As the patient noticed weakness in the left upper and lower limbs, an MRI brain was done on day 7 that showed infarcts in the right frontal, bilateral parietal lobe, bilateral centrum semiovale, head of the right caudate nucleus and bilateral cerebellar hemisphere. A neurology consultation was obtained and advised to start on a single antiplatelet drug. Patient was discharged on day 10 of admission with single antiplaletet drug and other symptomatic treatment and advised to get regular wound dressing every alternate day.

Outcome and Follow-Up

After 10 days of discharge, the patient was followed up on an OPD basis. Patient symptoms were completely resolved in the form of normal power of the left upper and lower limbs and he was able to walk without any support. The antiplaletet drug was stopped and the patient was reassured and advised to continue regular wound care and dressing.

DISCUSSION

In the Indian subcontinent, vipers are the most common snake bites. When a viper bite causes envenomation, local envenomation occurs first, followed by aberrant coagulation. Pro and anticoagulant toxins are two different categories of the many toxins found in viper venom. [1] The toxins cerastobin, factor IVA cerastocytin, cerastotin and afaacytin have shown procoagulant/platelet aggregating characteristics. These diverse protein products have enzymatic activity akin to that of thrombin. Monoclonal antibodies against the thrombin receptor, GPIIb/IIIa, or GP1b block their action. The correlated risk factors also include hypotension and disseminated intravascular coagulation.

It is unusual to get an ischemic stroke after a snake bite. There are surprisingly few documented incidences of ischemic strokes brought on by viper bites. Following Russell's viper bite, Bashir and Jinkins described a patient who had hemiplegia and aphasia, which is compatible with a middle cerebral artery infarction.[1] Mosquera et al. documented cerebrovascular consequences in eight patients (seven hemorrhagic strokes and one ischemic stroke) in an observational analysis of 309 snake bite cases.[2] An instance of ischemic cerebral infarction following a viper bite was documented by Narang et al.[3]

Mayank Nakipuria et al.[4] reported a case series of five patients with posterior circulation stroke, one of whom suffered strokes both in PCA and MCA territories. They came to the conclusion that snakebite should be taken into consideration as a significant differential in young stroke patients, particularly in rural regions because of the non-specific presentation, which makes a high degree of suspicion essential to diagnosing such strokes.

Learning Points

- Neurological symptoms following a viper bite might have several causes such as toxin-induced vasculitis, the procoagulant effect, endothelial damage, cardiac thromboembolism due to the direct cardiotoxic effect – dysrhythmias, disseminated intravascular coagulation and hypotension.
- In cases where a viper bite is suspected, it is imperative to assess the risk of ischemic stroke or other neurological disorders.
- Treating the patients early with polyvalent anti-snake venom should be emphasized among the primary health care providers to cover both the neurotoxic and haematotoxic manifestations of the Viper snake bite.

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