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Viper Bite Presenting as Acute Ischemic Stroke

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Abstract: ***We describe a case of 40 year old female patient who was brought to the hospital with history of altered sensorium, 6 hours, following viper bite. The patient had local envenomation with impaired coagulation and right hemiplegia with motor aphasia. MRI of brain showed infarct in the left middle cerebral artery territory and a normal magnetic resonance angiogram. Neurological manifestations following snake bite is often due to hemorrhagic complications. Presentation with infarction is rare and can be attributed to vasculitis, vasospasm, endothelial damage; toxin induced procoagulant effect and disseminated intravascular coagulation.***

Keywords: snakebite, viper, infarct, stroke in young, hemiparesis

1. Introduction

India has the highest number of cases of snake bites worldwide with a mortality of 35,000 to 50,000 cases per year according to WHO direct estimates.[1] Bites are more frequent among males, in the lower limb and during rainy season.[1]-[3] Envenomation occurred in 10-80% of the snake bites.[1] Viper was the most common cause of bite.[1]-[5] Local envenomation is the most common manifestation, followed by hemostatic abnormalities and neurotoxicity.[2][4][5] Coagulopathy, when present, is diagnostic of viper bites in south Asia.[1] Viper bites have been associated rarely with cerebrovascular accidents, most commonly due to hemorrhagic and rarely due to infarct.[6] Here we describe a patient with right hemiplegia with motor aphasia following viper bite, caused due to infarct.

2. Case Report

A 40 year old female patient was brought to the emergency department of the hospital with history of altered sensorium 6 hours after the snake bite, over the left foot. The snake was identified as Russell's viper on the basis of description provided by the informants. The patient was brought to hospital 12 hours after the snake bite. At admission, patient had erythema and swelling of left foot; two fang marks was present over the dorsum of left foot. There was no bleeding from the bite site. The blood pressure was 110/80 mmHg at admission, with a respiratory rate of 16/min. On neurological examination bilateral pupils were reactive and symmetric, with no gaze preference, patient had motor aphasia, hypotonia of the right upper limb and lower limb, with areflexia and extensor plantar on the right side. There was no evidence of neck muscle weakness. There was no evidence of bleeding manifestations.

On investigations, the 20 minute whole blood clotting time was prolonged. The prothrombin time and aPTT was prolonged. The complete blood count, lipid profile and renal parameters was normal. Electrocardiography, echocardiography and Doppler of both carotid was normal. MRI showed infarct in the left tempero-parietal area. Magnetic resonance angiography was normal.

The patient received 20 vials of polyvalent anti-snake venom (ASV) over 24 hours. The coagulation profile normalized over a period of 24 hours. Tetanus toxoid was given, along with symptomatic treatment. The swelling and erythema subsided over the next 4-5 days. Anti-platelets or anticoagulants were not started in the view of derangement of the coagulation parameters. 10 days after admission patient had improved, was ambulatory without support, with nominal aphasia. The patient was discharged with physiotherapy.

3. Discussion

Viper bite is the most common snake bite in the Indian sub-continent. The envenomation by viper bite commonly presents with local envenomation, followed by abnormal coagulation.[6] The various toxins present in the viper venom have both pro-coagulant and anticoagulant effect. The toxins with well-established pro-coagulant / platelet aggregating properties are cerastobin,[7] factor IVa,[8]cerastocytin,[9][10]cerastotin,[11] and afaacytin[12]. These various protein products have thrombinlike enzyme activity; different toxins activate different parts of the coagulation cascade.[7]-[14] There activity is inhibited by monoclonal antibodies against GP1b or GPIIb/IIIa or thrombin receptor. Following viper bite patients are also at risk of disseminated intravascular coagulation and hypotension. Disseminated intravascular coagulation can cause of neurological disorder, due to large vessel occlusion.[15] The toxin itself can cause vasospasm which can precipitate a cerebrovascular accident.

All the case reports of infarct following snake bite is due to viper bite. All these patients had features of local envenomation and most had evidence of disseminated intravascular coagulation. The infarct commonly involved the anterior circulation, with hemiparesis being the most common presentation.[16]-[29] Study by Thomas, et al[30] showed that administration of the anti-snake venom within six hours of the viper bite, would prevent this complication.

4. Conclusion

Neurological manifestations following viper bite can be due to various reasons, such as toxin induced vasculitis, pro-coagulant effect, endothelial damage, disseminated intravascular coagulation and hypotension. The early recognition of the viper bite and administration of anti-snake venom can prevent this rare but debilitating complication.

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