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CASE REPORTS

Ischaemic stroke with internal carotid artery occlusion following viper bite: A case report

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Abstract

Snake bites are an important cause of morbidity and mortality in tropical countries. Neurological complications are quite common in venomous snake bites. Strokes, mainly haemorrhagic and occasionally ischaemic, have been reported in 2.6% of snake bites. Direct effect of toxin, vasospasm and release of procoagulant and anticoagulant factors have all been postulated as the cause. However thrombotic stroke with involvement of a major cerebral vessel is very rare. We report a case of complete left internal carotid artery occlusion with ischaemic stroke in a young lady after a Russel's viper bite.

INTRODUCTION

India reports the most number of deaths from snake bite in the world with an annual mortality of 46,000.[1] The big four namely Russell's viper, saw-scaled viper, Indian cobra, and the common krait are usually responsible for most of the deaths.[2] The most common clinical manifestations are bleeding from various sites, neurological toxicity, renal failure and local reactions at site of bite. Studies have documented a stroke incidence of 2.6% in snake bite with a predominance of haemorrhagic strokes due to consumption coagulopathy.[3] We present an unusual complication, ischaemic infarct due to left internal carotid artery occlusion after Russell's viper bite.

CASE REPORT

A 32 year old previously healthy female was bitten by a snake (identified as Russell's viper) in the leg while working in the farm. She was taken to a nearby public sector hospital after four hours, where anti snake venom (ASV) was started. Her initial blood investigations revealed a whole blood clotting time of 28 minutes. She developed altered sensorium after six vials of ASV were administered and was then referred to our centre in view of aphasia and decreased movements of the right side of body. In the emergency room, she was drowsy with a Glasgow Coma Scale of 8/15. Her pulse rate and blood pressure were normal. Central nervous system examination revealed a right hemiplegia (grade 0 power in both upper and lower limb) with absent reflexes and extensor plantar responses. Her left foot revealed fang marks and evidence of swelling up to knee associated with warmth, erythema and tenderness.

Laboratory investigations revealed leucocytosis (total count 20,000/cmm), normal liver and renal functions, whole blood clotting time of 13 minutes.

A provisional diagnosis of acute stroke (possible Haemorrhage) involving the left Middle Cerebral Artery territory was made in view of the initial altered clotting profile. The patient underwent a CT scan brain which showed nonhaemorrhagic infarct of left middle cerebral artery and anterior cerebral artery territory. Patient was started on intravenous antibiotics in view of cellulitis and a total of 20 vials of ASV were given according to the Indian snake bite protocol 2007. Due to a drop in the sensorium, a repeat CT scan with angiogram was done on the third day, which revealed a large ischaemic stroke with midline shift (Figure 1). Angiogram revealed complete occlusion of the left internal carotid artery (Figures 2). Further etiological workup for stroke in the young revealed normal ECG, echocardiography, lipid profile, and serum

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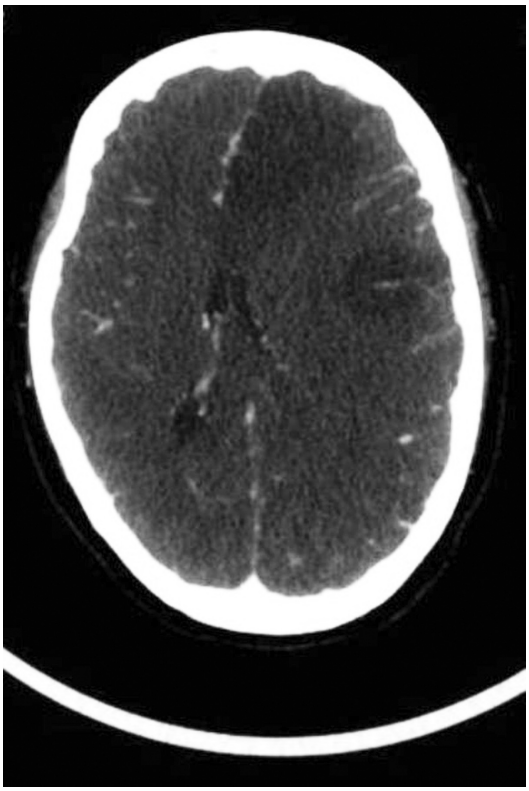

Figure 1. CT brain showing large ischaemic infarct left side with midline shift.

homocysteine level. ANA was negative. Protein C, S and antithrombin III levels were not done as values will be altered following a thrombotic event and ASV therapy.

The patient was started on antiedema measures (mannitol and dexamethasone for 48 hours), antiplatelets (aspirin 150 mg/day) and physiotherapy. Anticoagulants were not initiated due to the potential risk of bleed. Over the next 10 days, her Glasgow coma score improved to 12/15. At discharge (day 12), she was conscious, had motor aphasia and power of grade II in the right upper and lower limb. At a follow-up visit to the outpatient clinic after 3 weeks, she was able to walk with support with a muscle power of 3/5 and was able to make incomprehensible sounds.

DISCUSSION

Apart from neuromuscular complications like ptosis and respiratory paralysis, strokes are the second most common neurological manifestation of snake bites.[4] Hemorrhagic strokes occur due to release of metalloproteases, phospholipase A2, phosphomono and diesterases which cause

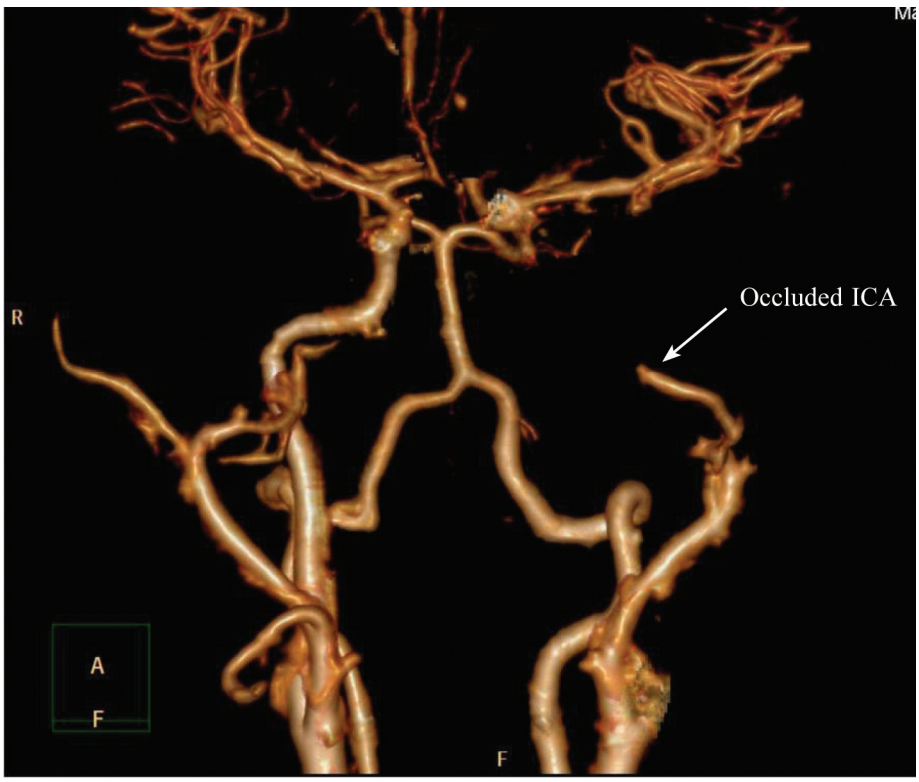

Figure 2. CT angiography showing complete occlusion of the left internal carotid artery.

destruction of basement membrane of capillary wall, interfere with the coagulation system, antagonize platelet action at specific sites such as GpIIb/IIIa, and cause consumptive coagulopathy.[5] In vitro studies on mice injected with Russell's viper venom have revealed low platelets, fibrinogen, increased fibrinolytic activity and presence of fibrin degradation products.[6]

Few ischaemic strokes have been reported following Russell's viper bites. The ones reported are mainly from South Asia.[7-9,11] The majority of these have involved the anterior cerebral circulation. There has been considerable variation in the time of onset of stroke after the snake bite, response to treatment and outcome. Most have revealed normal MR/ CT angiogram. Our patient had evidence of a complete occlusion of the internal carotid artery.

Viper venom has components with both pro and anti coagulant effect. The enzyme that has been implicated in ischaemic stroke is arginine esterase hydrolase which has a mode of action similar to thrombin.[8,10] Other clotting factors including factor V and X are also activated by other enzymes. The postulated mechanisms of ischaemic stroke include massive intravascular coagulation causing small and large vessel occluson[10], toxic vasculitis[8], direct effect on vascular endothelial cells[9], or severe vascular spasm due to hemorrhagins.[8,9]

ASV has been associated with multiple complications. This may be classified into early reactions such as anaphylaxis and pyrogenic reactions characterized by fever with chills and itching and late reactions such as serum sickness, with the incidence being as high as 81% in some studies.[12] Severe hypotension secondary to anaphylaxis can also predispose to an ischaemic stroke.[9] As this patient developed neurological symptoms only after initiating ASV therapy, a direct association with ASV is possible. A hypotensive etiology though possible, is unlikely since hypotension usually tends to cause watershed territory infarcts and is not strongly associated with occlusion of a major cerebral artery.[13] Besides the patient's blood pressure was normal and she did not have any features of anaphylaxis.

In conclusion, in our patient, though ischemic stroke causation includes many possibilities, we suggest that toxic vasculitis or massive intravascular coagulation may be the most likely cause of internal carotid artery occlusion. We report this case to highlight this uncommon presentation of viper bite.

DISCLOSURE

Conflict of interest: None

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