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ACUTE ISCHEMIC STROKE FOLLOWING A RUSSELL'S VIPER BITE- A RARE NEUROLOGICAL COMPLICATION

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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. Ischemic stroke following viper bite is rare. Strokes, mainly hemorrhagic and occasionally ischemic, 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. We report a case of 38 years male who developed left hemiplegia following a Russell's viper bite. The possible mechanisms of infarction in this scenario are discussed. Patient was treated with anti-snake venom and showed a good recovery. Early imaging and early treatment with antisnake venom is important for a favorable outcome.

INTRODUCTION

Snake bites are common cause of morbidity and mortality in India. On an average, 200 000 fall prey to snake bite per year in India and an estimated mortality may range from 20 000 to 50 000 cases. Viperidae species consisting of Russell's viper (Daboia russelli) and saw scaled viper (Echiscarinatus) are the leading cause of fatal snake bite in India.[1-3] The common clinical characteristics of viper bite include local cellulites, renal failure and systemic hemorrhagic manifestation.

Neurologic deficit following viper bite is not uncommon and is usually due to intracerebral or subarachnoid bleed. Ischemic infarction following viper envenomation has been described by only few authors.[3-7] In majority of the cases reported, ischemic infarction involved the anterior circulation. Korean viper bite resulting in brain stem infarction has also been reported.[8] In this report we present an unusual complication, cerebral infarction following Russell's viper bite.

CASE REPORT: A 38 years old previously healthy male was bitten by a snake (identified as Russell's viper, as per the descriptions given by relatives) in the left foot while he was working in the paddy field.A few minutes after the bite; he noticed severe pain, redness and ecchymosis over the left foot. Immediately he was taken to a nearby government hospital, at the time of admission, his pulse rate was 86 beats per minute, blood pressure was 128/78mmhg and respiratory rate was 20 per minute. Systemic examinations were normal except mild ptosis. Later Antisnake Venom (ASV) 10 vials started. The patient became drowsy and was not able to move his left upper limb and left lower limb in the morning (approximately 15 hours after the bite) and referred to Bapuji hospital Davangere.

At the time of admission in our hospital, he was drowsy with a Glasgow Coma scale of 9/15. His pulse rate, blood pressure and respiratory cycles were normal. Local examination showed two deep bite marks with surrounding redness, edema and cellulitis over the dorsum of the left foot. Central nervous systemic examination revealed a complete left hemiplegia (grade 0 power in the upper limb and grade 2 power in the lower limb with UMN type left facial nerve palsy) and he had exaggerated deep tendon reflexes in the left side of the body and extensor left plantar response. Detailed neurologic examination could not be done due to altered mentation. Examinations of all other systems were normal.

Laboratory investigations: Hemoglobin was 12.2gm/dl, total leukocyte count was 15,450/mm3, platelet count 1, 70,000/mm3, blood urea was 22mg/dl, serum creatinine was 1.1mg/dl and microscopic hematuria with pus cells seen in routine urine examination. Serum electrolytes and liver function tests were normal. Coagulation profile showed prolonged bleeding time and clotting time during first day and they were normalized by the third day of admission. Prothrombin time and activated plasma thromboplastin time were normal.

Brain computed tomography showed right middle cerebral artery territory nonhaemorrhagic infarct with

mild cerebral edema (figure 1). Further etiological workup for stroke in the patient revealed normal electrocardiogram, echocardiography, carotid Doppler, lipid profile and serum 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 managed with Antisnake venom (ASV), antibiotics and antiedema measures. An injection of tetanus toxoid was given. Over the next 3 days his Glasgow Coma scale score improved to 15/15. At discharge (after 10 days of admission) the patient was able to walk without support and muscle power in the left upper limb 3/5 and lower limb 5/5. The patient was advised to continue physiotherapy.

DISCUSSION

The manifestations following viper bites depend upon the severity of envenomation. In cases of minimal envenomation, local swelling, ecchymosis, and blisters will be noticed. In case of severe envenomation, local signs will be profound with tissue necrosis and systemic manifestations. Systemic complications are primarily related to bleeding due to depletion of fibrinogen and clotting factors. This is manifested by hemorrhage in different parts of the body including gums, nose, central nervous system, gastrointestinal and urinary tract bleeding. Hemorrhagins and hemolysins present in snake venom destroy the walls of blood vessel and cause coagulation defect. Myoglobinuria from muscle destruction can cause renal failure. Cardiotoxins present in venom may lead to arrhythmias, decreased cardiac contractility and hypotension. The neurological feature of viper bite includes drowsiness, confusion, fainting, dizziness, blurred vision, loss of muscle coordination, and convulsions.[9,10] The mechanisms by which cerebral infarction occurs in snake envenomation can be multi-factorial and are as follows:

• The venom exhibits anticoagulant and procoagulant effects that can lead to small and even large vessel occlusions due to the microthrombi resulting in cerebral infarction.[11]

•Haemorrhagins are complement mediated toxic components of snake venom that may result in severe vascular spasm, endothelial damage and increasedvascular permeability. All of these may lead to toxicvasculitis and result in thrombosis.[12] Hyper coagulation due to procoagulants in venom, such as arginine, esterase, andhydrolase, and hyper viscosity caused by hypervolemia andhypo perfusion secondary to hypotension may also contributeto vessel occlusion.[3, 10]

Our patient was relatively young and had no premorbid illness. Carotid and vertebral Doppler did not show evidence of atherosclerosis. Echocardiography was negative for cardiogenic emboli. The possibility of disseminated intravascular coagulation was not considered, as prothrombin time, activated plasma thromboplasin time and platelet counts were normal. Severe hypotension secondary to anaphylaxis can also predispose to an ischemicstroke.[6] As this patient developed neurological symptoms only after initiating ASVtherapy, a direct association with ASV is possible. But hypotension as the cause of cerebral infarction was not considered, as the infarction did not involve the typical watershed zone and his blood pressure recordings were normal. Toxic vasculitis or toxin induced vascular spasm and direct toxin induced endothelial damage may be the cause in this case.

In conclusion, our patient developed significant neurological morbidity despite of early treatment. The exact pathogenesis remains unclear. We report this case as a rare neurological complication of viper bite and illustrate further study is needed to identify the possible exact cause for cerebral infarction and focal neurological deficit.

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