INTRODUCTION

Neurological deficit following snake bite is not uncommon and is usually due to intracerebral haemorrhage or subarachnoid bleed as a result of depletion of clotting factors. We present the case of a previously healthy young male who developed posterior circulation infarction after the bite of a snake. The mechanisms of cerebral infarction following snake bite are discussed.

CASE REPORT

A 14 years old male was bitten by a snake at 10 AM on 16th September 2006 while he was playing outside his house in a hilly area. He developed bilateral ptosis followed by altered sensorium within 3 hours. He was treated with 10 vials of antisnake venom in a taluk hospital and referred to a Tertiary Care Centre.

At the time of admission, patient was drowsy, pupils unequal, sluggishly reacting to light on both sides, oculocephalic reflex was impaired with normal fundus. Patient had hypotonia of all 4 limbs, plantars were extensor. Bitemark was seen over dorsum of right foot. Patient also had cellulitis of Right Foot. Other systems were normal.

Haemoglobin, white blood cell count, ESR, blood Sugar, Urea and Serum Creatinine were normal. clotting time was 8 minutes. Bleeding time was 7 minutes. Prothrombin time was 11 seconds. Activated partial thromboplastin time was 30 seconds and platelet count was 1,50,000 cells/cumm. Electrocardiogram was normal (Figs. 1a, b, c and d).

CT Brain taken after 48 hours of the bite revealed bilateral cerebellar and right occipital infarct with mass effect. Patient was treated with mannitol, frusemide, aspirin and antibiotics. Patient died at 4 PM on 17.9.2006.

DISCUSSION

The clinical and radiological presentation of our patient strongly suggests a vascular thrombosis as cause for his deficit. The occurrence of vascular thrombosis in vessels adjacent to the site of envenomation is common. It is extremely rare for thrombosis to occur in distant vessels. Cerebral infarction following snake bite is rare and only few cases have been reported since 1966. There are several mechanisms by which cerebral infarction occur in snake envenomation.

1. Hypotension due to hypovolaemia from sweating, vomiting, decreased fluid intake and bleeding tendencies. This leads to low flow state and watershed infarct.
2. Hypercoagulability can be due to procoagulants in the

Abstract

Neurological deficits can occur following snake bite. It is usually due to intracerebral haemorrhage or subarachnoid bleed as a result of depletion of clotting factors. A healthy 14 years old male developed bilateral ptosis and altered sensorium within 3 hours of snake bite. CT Brain revealed bilateral cerebellar and right occipital infarction with mass effect. Clotting time and bleeding time were normal. The possible mechanism for infarction in this patient is discussed.
venom such as hydrolase, consumption coagulopathy phase of DIC.\(^2\)

3. Endothelial injury due to toxic vasculitis by the components of venom can lead to thrombosis.\(^3\)

The infarcts in our patient are not in a classical watershed territory and therefore do not suggest hypotension as a cause. Clotting time was normal and ruled out coagulopathy as a cause. The possible cause of infarct in the posterior circulation is due to toxic vasculitis caused by injury to the endothelium by snake venom toxin.

In this case only the posterior circulation is affected and the anterior circulation is spared. Any pre-existing abnormality in the posterior circulatory blood vessel wall will also have to be considered as a cause for the infarct though we do not have DSA studies to prove this.

**REFERENCES**


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**Book Review**

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