Case Report

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Atypical Presentation of Hematotoxic Snake Bite with Acute Ischemic Stroke and Acute Blindness

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Abstract

A 71 year old nil comorbid, referred from government hospital after unknown snake bite with acute vision loss 7 hours after snake bite.

Neurological deficits following hematotoxic snake bite are usually due to intracranial hemorrhage as a result of depletion in clotting factors. Ischemic strokes and blindness followed by snake bites are rare.

Presentation with cerebro-vascular infarction and blindness are rare and can be attributed to vasculitis/optic neuritis, vasospasm, toxin induced endothelial damage, toxin induced procoagulant effect and disseminated intravascular coagulation.

Keywords: Snakebite; Hematotoxicity; Acute Ischemic infarcts; Acute blindness.

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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] Envenomation occurs in 10–80% of the snake bites.[1] Bites are more frequent among field workers, in the exposed extremeties during damp and rainy seasons. local envenomation is the frequent manifestation, followed by hemostatic abnormalities and neurotoxicity.[2,3,4] In the past decade several cases of Ischemic stroke following Russell viper and Saw scale viper envenoming have been reported.[5,6]

Viper bites have been associated rarely with cerebrovascular accidents, most commonly due to hemorrhagic and rarely due to infarct.[7]

Here we describe a patient with no deficits and acute vision loss, caused due to watershed territory infarcts.

Case Report

A 71 years old nil comorbid elderly farmer while working in his field had a unknown snake bite on the medial aspect of the right heel.

He was immediately rushed into local hospital where Anti Snake Venom was given after 3.5 hours of snake bite.

Patient had acute vision loss after 4 hours of ASV. This patient was immediately repeated with another quantum of ASV. He didn't regain his lost vision and was referred to a teritiary centre and the patient was brought into our emergency medicine department after 12 hours of second quantum of ASV.

On examination of patient

Bite mark in medial aspect of the right heel,no bleeding from the site or no local toxicity.

Patient is conscious, cooperative but not oriented.

Vitals on examination-pulse rate 90bpm, blood pressure-130/90mm of Hg, SaO_2-99% on RA, respiratory rate 12 cpm.

Systemic examination-ptosis was present, there was no respiratory distress, no bleeding manifestations like hematemesis/hematuria/ bleeding gums, local swelling was present at the bite site.

On investigation

20 minutes whole blood clotting time was prolonged, PT/Aptt and INR were within normal range.

Total lucocytes count was 13110 cells (predominantly neutrophils), other investigations were in normal limits.

Acute blindness was investigated with CT Brain which showed no abnormality, followed by MRI brain which revealed multifocal areas of acute infarcts in bilateral frontal, parietal, thalami, left temporal region and both cerebellar hemispheres-watershed territory infarcts and swollen retrobulbar intra-orbital segment of the optic nerve.

Ophthalmic examination revealed only light perception.

Conclusion

With the background history of snake bite leading to hematoxicity and acute blindness with no focal neurological deficits; MRI showing watershed territory infarcts and swollen retrobulbar intra-orbital segment of the optic nerve; Ophthalmic examination revealed only light perception; suggesting of OPTIC NEURITIS by the toxins of snake bite.

Patient was administered with total 30 vials of ASV over the period of 36 hours due to prolonged 20 minutes whole blood clotting time. Patient was also started on dexamethasone 4mg TID which was suggesting of optic neuritis.

Patient perceived his vision back after 30 hours of snake bite and 20 vials of ASV administration.

Anti Snake Venom will remain the main stay of treatment for snake bites with the complications arising from the snake bite toxins.

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