Case Report

Acute Disseminated Encephalomyelitis: A rare complication of snake bite

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ABSTRACT

Acute disseminated encephalomyelitis (ADEM), is a demyelinating condition with significant morbidity and mortality, following infectious fevers and vaccinations. It can be diagnosed with certainty using advances in neuroimaging techniques. Following snake bite and Anti Snake Venom (ASV) administration, few cases of ADEM were reported. One such case is reported here.

Key words : Anti - Snake Venom, Methylprednisolone, Magnetic Resonance Imaging.

Introduction

Poisonous snake bite is a common community health problem in India. WHO estimated that among half a million snake bites, about 30,000 to 40,000 snake bite deaths occur every year in the world.[1] Commonly encountered poisonous snakes in India are Indian Cobra, Krait, Russell's Viper, and Saw Scaled Viper. The poisonous snake venom affects circulatory system, neuromuscular junction, and myocardium apart from local effects. The polyvalent Anti - Snake Venom (ASV) is the specific life saving measure for most venomous snake bites along with other supportive measures. ASV is an immunoglobulin raised in horse's serum and has its own complications. Encephalopathy, a rare, complication of ASV, may occur due to late anaphylactic reaction.[1]

Case Report

A seven year old boy presented to emergency department with history of snakebite (cobra) on right forearm. He received a tetanus toxoid injection and one vial of anti-snake venom (ASV) before reaching our hospital. Within two hours, the boy developed ptosis and labored breathing. At admission, he was in a state of altered sensorium with Glasgow Coma Scale of 3/15, sluggishly reacting pupils, severe respiratory distress with oxygen saturation of 60% at room air (Pulse Oxymeter), with poor peripheral pulses, bradycardia, and systolic blood pressure of 60 mm of Hg. Immediately, the child was intubated and connected to a ventilator. After sending necessary investigations, 10 vials of ASV was administered as an infusion along with Inj. Neostigmine, inotropes (Dopamine, Dobutamine), antibiotics (Clindamycin, Cefotaxime) and intravenous fluids. His whole blood clotting time was within normal limits and WBC count was 14,000 cells/mm^3. Renal and liver function tests were within normal limits. Another 15 vials of ASV were given later.

Ventilator support was withdrawn after 18 hours as he recovered. On day 5, the child developed high grade fever, and was in a state of confusion. He was unable to sit and searching movements were noticed. On CNS examination, the tone was decreased in both upper and lower limbs with a power of 3/5, with brisk reflexes and up-going plantars. His bladder and bowel functions

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were normal. Ophthalmic examination revealed decreased visual acuity in both the eyes (not even perception of light) with normal optic disc indicating the possibility of acute bilateral retro bulbar neuritis.

An MRI scan of brain was done which showed hyperintense signals in the sub-cortical white matter involving frontal, parietal and occipital lobes on both right and left sides (Fig. 1) suggestive of "Acute Disseminated Encephalomyelitis (ADEM)".

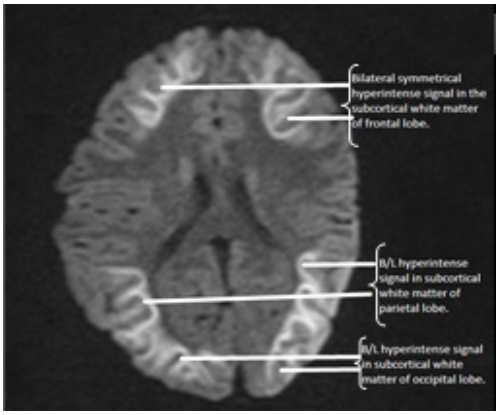

Fig. 1 : MRI of brain – before treatment

A clinical diagnosis of ADEM was made and the patient was given a high dose IV Methyl Prednisolone (30mg/kg). After two doses of I.V. Methyl Prednisolone, the patient showed an improvement in tone and power with no improvement in vision. Methyl Prednisolone was given for five days followed by oral prednisolone. Repeat MRI scan after a week revealed clearing of earlier lesions with hyperintense signals in basal ganglia (Fig. 2). On day 15, the child developed choreoathetoid movements involving both upper and lower limbs and unsteadiness of gait for which oral haloperidol was given. Gradual recovery of his vision and subsidence of abnormal movements was noticed in the next two weeks. Oral prednisolone and haloperidol were slowly tapered and withdrawn over a period of three weeks. An MRI scan taken six weeks later showed minimal persistence of hyperintense signals in basal ganglia. However clinically, the child had recovered.

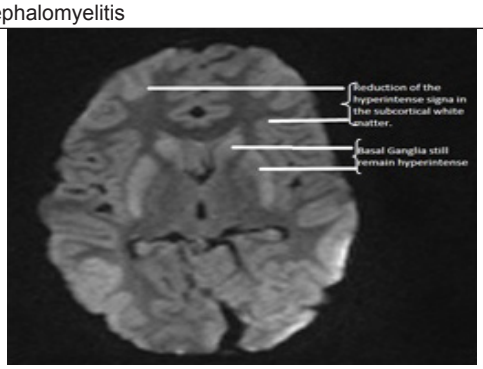

Fig. 2 : MRI of brain – 1 week after treatment

Discussion

Acute disseminated encephalomyelitis is traditionally considered a monophasic inflammatory demyelinating disorder with pleotropic clinical manifestations which usually include encephalopathy, but variably include other focal or multifocal syndromes suggestive of inflammatory demyelinating disorder including optic neuritis and myelitis. The hallmark of ADEM is its monophasic course.[2]

It is known to occur following the infectious illnesses and vaccines. So far, only few cases of ADEM following the snake bite were reported.[3 - 5]

The immunopathogenesis of demyelination following snake bite may be related to "molecular mimicry" between the components of snake venom and myelin, and subsequent generation of pathogenic autoantibodies. It may also develop as a consequence of serum sickness type of reaction to initial administration of anti-venom.[3] Serum sickness type reaction is a late reaction and develops 1 to 12 days after treatment and includes encephalopathy rarely.[1] However, it is difficult to establish what is responsible for demyelination.

Clinical manifestations include moderate to high grade fever, encephalopathy, and visual disturbances due to bilateral acute retro bulbar neuritis. Weakness is more commonly observed. They may have ataxia and chorea due to involvement of cerebellum and basal ganglia.[2] Our case had encephalopathy and visual disturbances on day 5 and ataxia as well as

chorea on day 15.

Investigations may show leukocytosis, increase in ESR, and an increase in the number of cells in Cerebro Spinal Fluid. The lack of pathognomonic, epidemiologic, clinical, and laboratory features in ADEM and the insensitivity of CT imaging indicate the use of MRI with T2 and FLAIR sequences to distinguish ADEM from other causes of acute childhood encephalopathy.[6] In our case MRI showed involvement of frontal, parietal, occipital lobes and basal ganglia.

The pathological hall mark of ADEM is perivenular inflammation with limited sleeves of demyelination,[7] in contrast to confluent areas of macrophagic infiltration in completely demyelinated regions that are typical of multiple sclerosis plaque.[7]

ADEM has been shown to respond to high doses of I.V. Methyl Prednisolone.[8,9] Alternatively I.V Immunoglobulin, and Plasmapheresis may also be used.[9] Our case responded to high doses of (30 mg/kg/day) Methyl Prednisolone.

Conclusion

Acute Disseminated Encephalomyelitis is a rare complication following snakebite. It can be diagnosed by MRI, and early administration of intravenous methyl prednisolone benefits the patient.

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