SNAKE BITE WITH TOXIC DEMYELINATION – A CASE REPORT

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ABSTRACT

INTRODUCTION

Snakebite is an important cause of mortality and morbidity in India. India has the highest number of deaths due to snake bite[1] Neurotoxicity due to snakebite is well-known with varied presentation.[2] Common cases of snakebites are of saw-scaled viper (Echis carinatus), Russell's viper (a viperidae), krait (Bungarus caeruleus), common cobra (Naja naja) king cobra (Ophiophagus hannah).[3]

KEYWORDS

Snake Bite, Demyelination.

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INTRODUCTION: Snakebite is an important cause of mortality and morbidity in India. India has the highest number of deaths due to snake bite[1] Neurotoxicity due to snakebite is well-known with varied presentation.[2] Common cases of snakebites are of saw-scaled viper (Echis carinatus), Russell's viper (a viperidae), krait (Bungarus caeruleus), common cobra (Naja naja) king cobra (Ophiophagus hannah).[3] The neurological consequences are predominantly the result of inhibition of neuromuscular transmission. In addition, there are reports in the literature of Guillain-Barre syndrome and delayed neuropathy following snake bite. Stroke can occur as a result of coagulopathy.[4] Acute disseminated encephalomyelitis is also reported following treatment with anti-snake venom. Here we are presenting a case of toxic demyelination following snake bite.

CASE REPORT: A 45 years old female patient presented with history of snakebite on her left hand. She had blood oozing from the site of bite. She was treated outside for 5 days with Anti snake venom, FFP and referred. On admission she had history of bleeding gums, decreased urine output, haemoptysis and haematuria of one-day duration. On day-6 she developed weakness of both lower limbs and on day-7 she had upper limb weakness and respiratory distress. There was no history of seizure. In the past history of she was not a known case of DM/HT/PT. No past history of weakness of all four limbs.

On examination the patient was conscious. Her vitals were stable. Higher mental function, cranial nerves examination were normal. Examination of spinomotor system revealed power of 2/5 in shoulder, elbow and wrist joint, with the handgrip-weak bilaterally, power in lower limbs was 0/5.

Abdominal reflex was absent, plantar no response on both sides. DTR-triceps jerk, knee jerk, ankle jerk were absent on both sides, all other DTR were normal. Sensory system examination revealed diminished all modalities of sensations below the level of nipple. Autonomic dysfunction was present. There was no cerebellar sign, no meningeal sign. The spinal vibration was decreased below T2.

Investigations: RBS-234mgs%. Urea-67. Creatinine-1.4 mgs%. Total bilirubin-2.4mgs., Indirect bilirubin-1.2 mgs%. Clotting time>20 minutes. Hb-7.4mgs%. Platelet-21000/cu mm. Prothrombin time 21.6seconds. MRI Cervical spine showed T2 hyperintensity from C5 to D5 level and reported as possibility of toxic demyelination. Patient was treated with IV Methyl prednisolone, blood and FFP transfusion and other supportives. Mechanical ventilation and peritoneal dialysis were done. Following treatment, patient showed improvement in overall clinical features- her power improved up to 4+/5 in both upper and lower limbs. She could also walk without support, able to pass urine and was able to carry out activities of daily living without any problem.

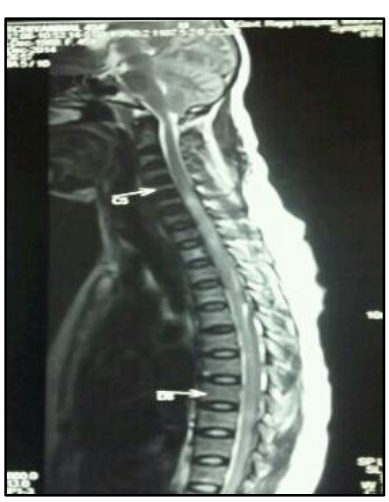

Fig. 1

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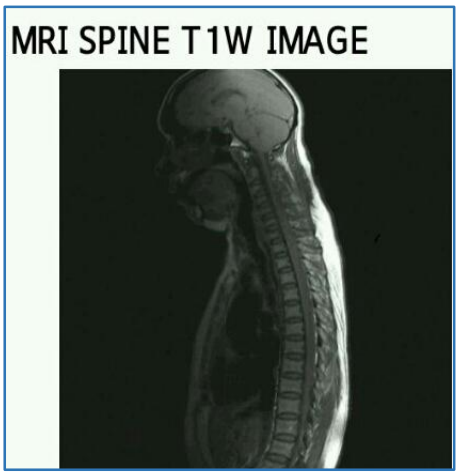

Fig. 2

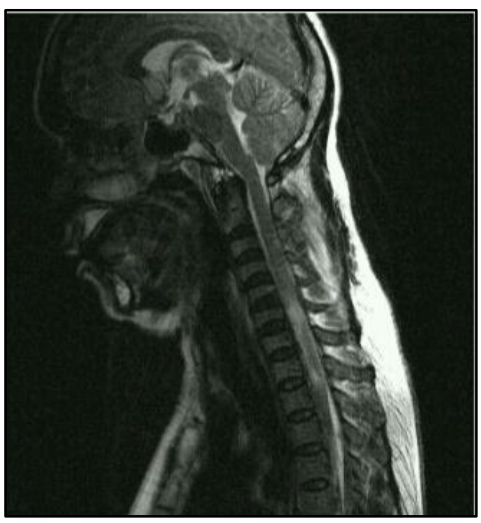

Fig. 3

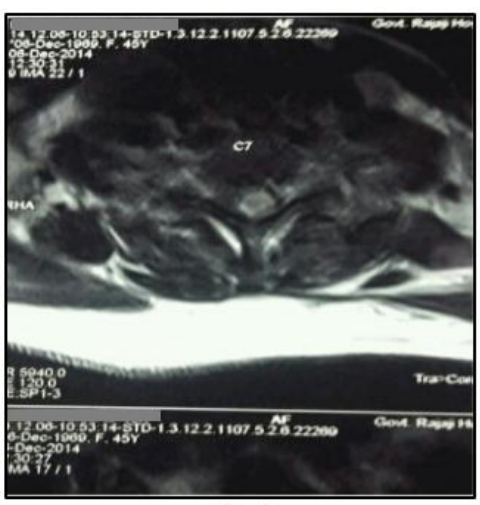

Fig. 4

DISCUSSION: Snake bite envenoming is a neglected tropical disease affecting millions of people living in the developing world. Four clinically important types of snake are found in India: cobras (Naja naja and Naja ka-outhia), the common krait (Bungarus caeruleus), Russell's viper (Daboia russelii), and the saw scaled viper (Echis carinatus)[5] Common neurological symptoms in decreasing order of frequency include ptosis (85.7%), ophthalmoplegia (75%), limb weakness (26.8%), respiratory failure (17.9%), palatal weakness (10.7%) and neck muscle weakness (7.1%) and ischemic stroke [6,7]These are experienced usually within 6 hours of the bite. Snake envenomation following bites from cobras, kraits, coral snakes, pit vipers and others can produce neuromuscular junction abnormality, distal sensory, sensorimotor, and pure motor polyneuropathies.[8]

Among the various neurotoxin, majority of them act by competitive binding to the post synaptic nicotinic and muscarinic Ach receptors, which prevents muscle contraction. Some of them act by presynaptic inhibition of Ach release.[9] Examples are krait alpha bungarotoxin-inhibits post synaptically. Inhibition of ACh release is accomplished effectively by phospholipase A2 toxins in snakes such as krait beta-bungarotoxin. Phospholipase A2 neurotoxins are responsible for severe paralysis in bite victims. In some snakes, such as mamba dendrotoxin, which facilitates Ach release. Some toxins act directly to inhibit cardiac contractility.

Snake bite can be painful or painless. Kraits bite usually occurs during night, often goes unnoticed. Initial symptom from elapid envenomation may include local swelling and necrosis, euphoria, headache, confusion, and nausea. Cranial nerve paralysis often ensues, including ptosis, ophthalmoplegia, and dysphagia. Patients may experience severe peripheral paralysis and localized paraesthesia. The paralysis may progress to respiratory failure and death.

ADEM is an acute condition characterized by widespread demyelinating condition leading to the rapid development of focal or multifocal neurological dysfunction. It usually follows few days to 3weeks after a triggering event like vaccination or infection.[3] It is usually monophasic and self-limited. The clinical presentation is characterized by a prodromal phase followed by neurological deficits that peak early and recover gradually later. The mechanism explained is an autoimmune response due to molecular mimicry against myelin or other auto-antigens. Sometimes it may be due to unintended activation of an auto-reactive T cell clone.

Our case presented with toxic demyelination following snake bite. Our patient had coagulopathy and renal failure following hemotoxic snake bite. At the end of first week she developed weakness of all 4 limbs with respiratory distress. MRI showed T2 hyper intense signals in cervical and dorsal spinal cords. ADEM can occur following treatment with Anti snake venom. But our case had direct toxic demyelination of spinal cord after snake bite. This case is presented for its rarity.

CONCLUSION: The association of Demyelination following snake bite has been seldom reported. Patients who develop unexplained weakness with spinal cord involvement features following snake bite should be screened for demyelination. Up to two thirds of patients with ADEM treated with corticosteroids benefit clinically, especially those who are treated early. Our case is presented for its rarity and also for the rapid improvement following treatment.

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