INTRODUCTION

Though many Neurological complications of Krait bite are known, the association of Acute Disseminated Encephalomyelitis (ADEM) following krait bite has seldom been reported. So far in India only two cases have been reported in literature one following treatment for Russell viper bite. This is a rare case where a 42 year old female presented with neurological manifestation which recovered completely initially and then later worsened within a week following treatment for her snake bite with Anti-snake venom (ASV). It was finally diagnosed as a case of Acute Disseminated Encephalomyelitis possibly ASV induced and treated successfully.

CASE REPORT:

We had a 42 year old female who came to the emergency department following snake bite (krait) on her right ankle. Though she was normal at presentation within one hour she started to develop ptosis as well as a swelling at the site of bite. Within the next few hours she developed respiratory distress. She did not have a previous significant medical history. General Examination she was conscious, oriented, afebrile, dyspnoeic, tachypnoeic SBC (Single breath count > 30); Fang marks present in Right ankle. Bilateral ptosis present BP: 110/70, PR-100/min, RR 30/min Spo2 98% with 2L/min O2, CVS: S1 S2+, RS:NVBS, P/A Soft, CNS: PERTL 3mm,EOM full, Ptosis+ HMF normal, Dysarthria+ Motor system Examination: clinically normal.
MANAGEMENT:

CBC, RFT, LFT- WNL, Serial Clotting time < 10 min, ECG, CXR - NAD

TREATMENT GIVEN:

Intensive care Support ,IV fluids, O2, Antisnake venom Injections: 25 vials serially, Antibiotics, Limb elevation.

Further course: As her general condition deteriorated she was intubated and connected to mechanical ventilator. Within a day she recovered and successfully extubated. Neurological Status at this time:- Pt awake, conscious, oriented, communicative, moves all 4 limbs equally, power 4+/5, plantar flexor response, no sensory disturbance. Biochemical parameters: CBC RFT LFT was normal.

Her further stay in hospital was uneventful except for certain hospital acquired infections which were adequately managed. Physiotherapy was continued. During the recovery phase she began to develop certain neurological symptoms in the form of altered mental status and weakness and focal seizures. Examination revealed: Clouding of consciousness, afebrile, vitals were Stable, CVS- S1 S2, RS – VBS, P/A: Soft

Neurological assessment: E2V3M5, PERTL 3mm, EOM full, No neck stiffness, Cranial Nerve: NAD , Fundus: No e/o papilledema, Motor:- weakness more in Left Upper Limb & Left Lower Limb with power 2/5 and 3/5 on the Right side limbs. Tone: Normal in all four limbs. DTR: 2+ Both UL and LL, Plantar: b/l Withdrawal. Impression: Acute Encephalopathy: ?Cause. Her clinical condition deteriorated. We did the following investigation to rule out septic and metabolic causes of encephalopathy. CBC, RFT, RBS, Electrolytes, LFT, TFT, CRP- Normal. Blood, Urine & Sputum C/S were sterile. Fever profile was negative. CXR – WNL. CSF analysis: Glu-79, Prot -21, Acellular, AFB & G/S Culture was negative. No Oligoclonal bands. Conservative Management in the form of bed care and physiotherapy was continued. Further she became unresponsive to our commands, uncommunicative, responded only to painful stimulation, tone all 4 limbs increased, plantar bilateral flexor, DTR were sluggish. She also started to have right lower limb focal fits suggestive of myoclonus. She was started on Tab. Clonazepam 0.5 mg 1HS and responded well. CT Brain: Normal Study. NCS: Slowed conduction velocities, decreased amplitude of motor and sensory action potentials suggestive of

Figure-1 Bilateral white matter hyperintensity with periventricular involvement and both cerebral hemisphere - ADEM.
demyelination. No other cause could be attributed to her altered mental status as all septic and metabolic work up was negative. Hence a MRI brain was taken which revealed T2 weighted white matter hyper intensities in multiple sites – periventricular and bilateral cerebrum suggestive of Acute Disseminated Encephalomyelitis (ADEM) (Figure-1,2).

**FINAL DIAGNOSIS:** Acute Disseminated Encephalomyelitis – Possibly – Anti Snake Venom Induced.

In view of the above diagnosis patient was treated with intravenous Methylprednisolone for 5 days, oral steroids subsequently and physiotherapy. Fitting with the diagnosis her neurological status improved dramatically and she was discharged in good health. She was followed up over the next six months and seen to be neurologically stable.

**DISCUSSION:**

ADEM is an acute widespread demyelinating condition characterised by the rapid development of focal or multifocal neurological dysfunction. It usually follows few days to 3 weeks after a triggering event like vaccination or infection. It is usually monophasic and self-limited. The clinical presentation (Table 1) is characterized by a prodromic phase followed by neurological deficits that peak early and recover gradually3, 4. 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. The hallmark lesions of ADEM are perivascular inflammation and surrounding demyelination within the CNS. MRI in ADEM: Bilateral asymmetric/symmetric involvement (rarely unilateral), White matter > gray matter, but usually both affected, Deep/juxtacortical white matter > periventricular white matter5, 6. **TREATMENT:** Initially with – High dose glucocorticoids7, 8 Duration: 4–8 weeks. Patients who fail to respond within a few days may benefit from a course of plasma exchange or intravenous immunoglobulin It has been successful in some fulminant cases (Kanter et al; Stricker et al) 9, 10.

More than two lakh cases of snake bite are reported in India annually and it is estimated that about 1/4th to 1/5th
of these end fatally. Four clinically important types of snake are found in India: cobras (Naja naja and Naja kaouthia), the common krait (Bungarus caeruleus), Russell’s viper (Daboia russelii), and the saw scaled viper (Echis carinatus). The neurological manifestations of snake bite are primarily due to inhibition of neuromuscular transmission. Literature search does indicate reports of Guillain-Barre’s syndrome and delayed neuropathy following snake bite. To the best of our knowledge, this case is one of the very rarely reported events of demyelination involving central and peripheral nervous system following krait bite. Diagnosis was confirmed with the help of imaging studies, while the nerve conduction study showed demyelinating pattern.

The immunopathogenesis of demyelination following snake bite may be related to molecular mimicry between one of the components of snake venom and myelin and subsequent generation of pathogenetic auto-antibodies causing myelin damage. In this patient, it may also have developed as a consequence of a serum sickness-like reaction to the initial administration of antivenin, as neuroparalysis recurred a few days after definite clinical improvement. Late reactions to antivenin are immune complex diseases and present in the form of serum sickness syndrome usually 5–24 days after antivenom administration. Clinical features include fever, arthralgias, mononeuritis multiplex, and rarely encephalopathy.

CONCLUSION:

The association of Acute Disseminated Encephalomyelitis (ADEM) following krait bite has seldom been reported. Patients who develop unexplained encephalopathy after snake bite should be screened for ADEM and appropriate therapy instituted. Up to two thirds of patients with ADEM treated with corticosteroids benefit clinically, especially those who are treated early. Our case is unique not only because of rarity of the diagnosis but also because of the improvement which the patient showed.

REFERENCES: