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Case Report

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NEUROTOXIN ENVENOMATION OF SNAKEBITE MIMICKING BRAIN DEATH: LOCKED-IN SYNDROME

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INTRODUCTION

Snakebites remain a public health problem in many countries even though it is difficult to be precise about the actual number of cases. The snake venom consists of different enzymatic and nonenzymatic components loosely categorized as neurotoxins and hemorrhagens. Venomous snakebite has become a global concern. While external ophthalmoplegia (ptosis) is an established association with neurotoxin envenomation, the combination of internal (dilated, non-reacting pupils) and external ophthalmoplegia can mimic brain death and pose a dilemma to the caregivers regarding continuation of therapy.[1] Many cases has been reported in children. We report a case of neurotoxin envenomation in a 35 year old male with such a presentation.

CASE REPORT

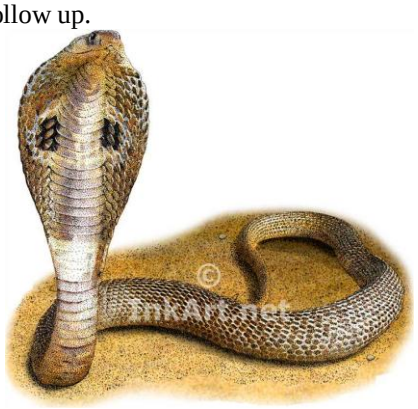
A 35 year old male was brought to emergency department with history of snake bite (viper) over the left upper limb while sleeping outside home. He presented half an hour after the bite. Patient developed ptosis and shallow breathing. In view of respiratory paralysis patient was shifted to ICU and intubated and put on ventilator support. Glasgow coma scale (GCS) score was 3.

Antisnake venom (ASV) was administered in 500 ml normal saline (NS). Patient received intravenous (IV) hydrocortisone 200 mg and IV chlorpheniramine 2 mg (as he developed allergic reactions to ASV) & ASV was continued. Intramuscular tetanus toxoid and Inj Amoxycillin- Clavulanate & Metronidazole was also administered to avoid snakebite related anaerobic and aerobic infections.

The patient was haemodynamically stable but remained unconscious with no response to deep painful stimuli. Pupils were dilated and not reactive to light, absent corneal reflexes, absence of doll's eye movement, generalized hypotonia, mute bilateral planters and absence of deep tendon reflexes (DTRs). A provisional diagnosis of hypoxic brain encephalopathy post-snake bite was made.

Non-contrast computed tomography scan head was advised which revealed a normal study. All biochemical investigations, including kidney and liver functions and coagulation profile were within the normal limits. Urine output was normal and there was no haematuria.

Patient started to respond to painful stimulus on day 4. Tracheostomy was done. Patient was weaned off of the ventilator support day 15. Quadriperesis persisted and was shifted out of ICU. Patient started improving. Decanulation of tracheostomy tube was done. Patient recovered from quadriperesis and started walking with support. Patient was discharged and was not available for further follow up.



Hb: 12.5%	**Calcium: 7.4mEq/L**	**SGOT/AST: 27U/I**
WBC: 22.6T/cumm	**GRBS: 75mg/dl**	**SGPT/ALT: 29U/I**
RBC: 4.04m/cumm	**Bleeding time: 2'05"**	**Sr.Albumin: 2.7g/dl**
Plt: 4.9 l/cumm	**Clotting time: 5'30"**	**Sr.Globulin: 2.8g/dl**
ESR: 20mm/hr	**PT: 20.6 Control: 13.5**	**A:G ratio – 0.9**
Blood Urea: 44mg/dl	**APTT: 28.4 Control: 33.8**	**Chest X-ray: normal**
Sr.Creatinine: 0.65mg/dl	**Ratio- 1.53 INR: 1.76**	**ECG: normal**
Sodium: 144mEq/l	**Sr. Total Bilirubin: 0.6mg/dl**	**HIV : negative**
Potassium: 3.7mEq/ml	**Sr. Direct bilirubin: 0.2mg/dl**	**HBsAG : negative**

DISCUSSION

Snake bite is one of the common medical emergency and an occupational hazard encountered in day to day practice in rural populations of India, where farming is a major source of employment. The morbidity and mortality rates of snake bite patients is high. In India, there are 216 species of snakes of which most important venomous snakes are Cobra, Common Krait, Russell's viper, and Saw scaled Viper.[2] Cobra and krait are neurotoxic.

Progressive descending paralysis is characteristic of systemic envenoming by elapid snakes (cobra and kraits). Neuroparesis is due to pre- and post-synaptic blockade in krait and post-synaptic blockade in cobra bite. Muscles innervated by cranial nerves are involved earlier. The pupils and diaphragm are the most resistant to toxins.[3] Ophthalmic manifestations of neurotoxin envenomation usually have the following sequence: Ptosis, loss of facial muscle expression, partial ophthalmoplegia (usually VI cranial nerve with loss of eye movement toward midline and diplopia on lateral gaze), complete ophthalmoplegia with a fixed forward gaze and lastly fixed dilated pupils. Lifting the paralysed upper eyelids enables the patient to see their surroundings which reassure the victim. The internal ophthalmoplegia is attributed to autonomic dysfunction.[4]

Presentation of an unconscious, paralysed patient, requiring tracheal intubation and ventilation, in the ED with fixed dilated pupils, no response to deep painful stimuli and absent DTRs leads most physicians to think of hypoxic brain damage.

Locked in syndrome (LIS) is a neurological syndrome in which despite being conscious patient is unable to communicate. The locked-in syndrome (LIS) has been classified into three categories: Classical, Incomplete, and Total. Classical LIS is characterized by quadriparesis and anarthria in conscious patient. Vertical eye movement and blinking are the only means of communication. Incomplete LIS is same as classic but with remnants of voluntary movement other than vertical movement. There is total immobility and inability to communicate in fully conscious patient in case of Total LIS. Each of the three categories have been subdivided into transient and chronic forms.[5]

The LIS is usually described in relation to damage of ventral pons by stroke. Non vascular etiologies of LIS affecting ventral pons are trauma, tumor, inflammatory conditions, etc. In addition to ventral pons, LIS are reported due to involvement of bilateral cerebral peduncle, medulla, etc. Transient LIS due to involvement of brainstem are reported in case of TIA, post-infectious encephalitis, hypoglycemia, hyperglycemia, etc. There are many peripheral causes of LIS, such as severe acute polyneuropathies(GBS), neuromuscular junction blockade(myasthenia gravis, toxins, snake bite) etc. The correct diagnosis is essential as peripheral causes of LIS are treatable conditions and complete recovery can be possible with timely interventions.

Azad *et al*.[6] reported four children with LIS due to snake envenomation all of whom made a successful recovery with polyvalent ASV and supportive management. John *et al*.[1] reported a case of a 6-year-old child who was comatose, areflexic, with internal and external ophthalmoplegia and absent brain stem reflexes, thus mimicking brain death. She received ASV therapy, and mechanical ventilation was continued. After 36 h, she showed improvement in motor power and was weaned off the ventilator after 5 days.

Leeprasert and Kaojarern[7] reported four cases of neurotoxin snake bite (Bungarus candidus) who had fixed and dilated pupils. Of these, three patients recovered following ASV therapy and had fixed non-reacting pupils even at the time of discharge (3-11 days). John *et al*. suggest that in such cases other confirmatory tests of brain death like electroencephalography, four-vessel cerebral angiography, transcranial Doppler ultrasonography or radionuclide imaging should be resorted to.

Vir *et al*.[8] reported a case of a 45-year-old male with neurotoxin envenomation who presented to the ED in a

conscious state with breathing difficulty. Pupils were bilaterally dilated, but reacting to light, but later when the patient became comatose (20 h after snake bite) there was complete ptosis and fixed dilated pupils. The patient responded to ASV therapy. Bhattacharya and Chakraborty.[3] reported dilated pupils in 3 of 13 snake bite patients, which they attributed to respiratory arrest and hypoxia. Of these, two survived with complete neurological recovery and one sustained hypoxic brain injury due to delayed presentation. Therefore, clinicians should be aware that fixed and dilated pupil in neurotoxic snake bite is a sign of envenoming and not a sign of brain death. Aggressive supportive treatment should be given, and the patient may recover completely.

CONCLUSION

Progressive paralysis along with ophthalmic manifestations of neurotoxin is seen commonly in victims of snake bite. However, complete opthalmoplegia (internal and external) with absent doll′s eye movement is not uncommon. Clinicians should be aware of this condition in envenomed patients, which mimics brain death and treat it with supportive measures for a favourable outcome.

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