Case Report

Neurotoxin envenomation mimicking brain death in a child: A case report and review of literature

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ABSTRACT

The spectrum of presentation of a victim of neurotoxic snake bite can range from mild ptosis to complete paralysis and ophthalmoplegia. We report a case of snake bite in a 10-year-old child who was comatosed with bilateral fixed dilated pupils and absent doll's eye movement that was interpreted as brain death. Physicians need to be aware of the likelihood of snakebite presenting as locked in syndrome.

Key words: Locked in syndrome, ophthalmoplegia, snake bite

INTRODUCTION

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] We report a case of neurotoxin envenomation in a child with such a presentation.

CASE REPORT

A 10-year-old boy, 30 kg, was brought to the emergency department (ED) 1 h after sustaining a snake bite (right index finger). The child was unconscious, in shock and gasping. Heart rate was 60/min and blood pressure was not recordable. Pupils were bilaterally dilated and fixed. The trachea was intubated with cuffed orotracheal tube, size - 6.0 and intermittent positive pressure ventilation (IPPV) was instituted. Ringer's lactate solution (400 ml) was infused rapidly. The child became haemodynamically stable, but there was no respiratory effort. Pupils remained dilated and fixed. Glasgow coma scale (GCS) score was 3. IPPV was continued. Antisnake venom (ASV) was administered; 10 vials of ASV in 400 ml normal saline (NS) over 1 h, followed by five vials of ASV in 250 ml NS over 3 h. Patient received intravenous (IV) hydrocortisone 200 mg and IV chlorpheniramine 2 mg (to prevent allergic reactions to ASV). Intramuscular tetanus toxoid and ceftriaxone 1 g IV 12 h was also administered to avoid snakebite related anaerobic and aerobic infections. Arterial blood gas analysis revealed pH: 7.125, PO_2: 60 mmHg, PCO_2: 72 mmHg, HCO_3: 16.6 mEq/L.

The child was transferred to intensive care unit (ICU) and lungs were ventilated with synchronized intermittent mandatory ventilation mode (tidal volume: 260 ml, respiratory rate: 14/min, inspiratory: Expiratory ratio - 1:2, FiO_2: 0.4). The child was haemodynamically stable but remained unconscious with no response to deep painful stimuli. Pupils were bilaterally dilated (4 mm) with no reaction to light. Neurologist consultation was obtained who documented 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

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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. Enteral feeding was started on day 2.

On day 3, the pupils began to react to light and gradually became bilaterally normal in size. However, GCS was still 3. Lorazepam (0.25 mg 12 h) was given through Ryle's tube. On day 4, the child became conscious and responded appropriately to verbal commands. Ptosis was evident. On day 5, respiratory efforts improved. Ventilation mode was changed to continuous positive airway pressure (CPAP) 6 cmH_2O with pressure support of 8 cmH_2O. On day 8, slight motor power improvement was seen (both upper limbs grade 2/5; both lower limbs grade1/5). On day 9, the child could generate a maximum inspiratory pressure (MIP) of − 12 cmH_2O and maximum expiratory pressure (MEP) +15 cmH_2O. Ptosis persisted.

By day 12, motor power was graded 5/5 in all the limbs. MIP was −20 cmH_2O and MEP was + 15 cmH_2O. T-piece trial was attempted, but failed, and CPAP mode was reinstituted. Throughout the stay in ICU, the biochemical parameters were within normal limits and the patient was on enteral feeding. On day 14, T-piece trial of weaning was successful, and the trachea was extubated. The child remained stable on oxygen by facemask. He was transferred to the ward and subsequently discharged home.

DISCUSSION

Progressive descending paralysis is characteristic of systemic envenoming by elapid snakes (cobra and kraits).[2] 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.[4] Lifting the paralysed upper eyelids enables the patient to see their surroundings which reassure the victim. The internal ophthalmoplegia is attributed to autonomic dysfunction.[5]

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. The patient can feel pain but cannot communicate that a snake has bitten him. The snake bite site may be difficult to find. This is exactly what was thought of in this patient who had rapidly deteriorated and showed very little signs of improvement despite receiving ASV therapy. There was reluctance on the part of the doctors to accept the patient in the ICU. The neurologist also had pronounced the patient as 'brain dead.'

Locked in syndrome (LIS) is a neurological syndrome in which despite being conscious patient is unable to communicate. 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). A child with snake bite was almost put on a funeral pyre and there are reports of snake bite victims being almost buried alive.[8] John *et al*.[1] 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.[9]

While there are several reports in the literature of snake bite envenomation, not many mention about the pupillary size and reaction. Dhaliwal[10] described a case of a 6-year-old child with cobra envenomation who was unconscious, responding feebly to deep pain, with dilated pupils reacting to light and bilateral disc oedema. The child subsequently recovered, but cortical blindness persisted. Vir *et al*.[11] 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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