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Autonomic Dysfunction Following Neurotoxic Snake Envenomation: Does Site of Bite Predispose?

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ABSTRACT

South Asian subcontinent is among the highest burden areas in respect of snake bite. India is commonest place in this subcontinent with mortality following snake envenomation. The four most important venomous snakes in India are cobra (Naja naja) and common krait (Bungarus caeruleus), which are neurotoxic, and the saw-scaled viper (Echis carinatus) and Russell's viper (Daboia russelii), which are hemotoxic[1]. Various autonomic dysfunction following neuro-paralytic envenomation with krait and cobra has been reported. Timely recognition and appropriate management of snake envenomation along with autonomic dysfunction helps to reduce morbidity and mortality.

Keywords: Autonomic dysfunction, hypertension, antisnake venom, blood pressure

INTRODUCTION

With an estimate of mortality of 35,000–50,000 people per annum, snake envenomation is commonest medical emergency in India [2]. Neuroparalysis due to envenomation by common cobra (Naja naja) and common krait (Bungarus caeruleus) is a common life threatening medical emergency [11]. The commonly encountered venomous snakes in India include common cobra (Naja naja), common krait (Bungarus caeruleus), Russell's viper (Daboia russelii) and saw scaled viper (Echis carinatus), against which a locally prepared equine polyvalent ASV antitoxin is available [3]. Neurological manifestations that follow envenoming by elapids (cobras and kraits) and less commonly Russell's viper include ptosis, external ophthalmalgia, paralysis of pharyngeal muscles, followed by respiratory and generalized muscle paralysis [3]. The acute clinical effects of snakebites vary from mild local reactions to severe, life-threatening systemic responses depending on the species and size of the snake; the location of the bite; the amount of venom inoculated; and the age, weight, and well-being of the patient [4]. Due to higher venom volume to body surface ratio in children, they are more prone to severe envenomation and autonomic instability. Here we present a case of 5 years old male child who presented neurotoxic snake bite with autonomic dysfunction in the form of hypertension.

CASE REPORT

A 5 years old male child presented with history of abdominal pain, vomiting, pooling of secretions from mouth, drooping of eyelids, difficulty in breathing to hospital during morning hours. These all symptoms develop while child was sleeping. As per history provided by parents, child develops these symptoms soon after waking up. At the time of physical examination, child was drowsy with regular heart rate of 160 beats per min, shallow irregular respiratory efforts and blood pressure of 110/60 mm Hg (> 99 percentile) in right arm supine position. His

oxygen saturation was 50% on room air. Fang marks of snake bite were noted behind right ear. On further evaluation of symptoms, history and examination diagnosis of neurotoxic snake bite was made. Child was immediately intubated and was started on mechanical ventilation. There was no abnormal finding on abdominal and respiratory system. Investigations including complete blood count, serum electrolytes, renal function test, liver function test, coagulation profile, whole blood clotting time and urine routine and microscopic examination were normal. Child was administered with ten vials of polyvalent ASV. In view of no improvement in condition of child after 30 minutes again ten vials of ASV were administered with a total dose of 20 vials. Even after giving adequate sedation there was no improvement in hypertension. As there was no prior history of episodes of hypertension, we kept a possibility of autonomic dysfunction secondary to neurotoxic snake bite. Child was started on nitroglycerine infusion for hypertensive urgency. Patients remain on infusion for 12 hours and then infusion tapered off gradually. After 48 hours the neurological status of patient was improved and patient was extubated on 3rd day of mechanical ventilation. To rule out secondary causes of hypertension different investigations done including chest radiograph, echocardiography, ultrasound abdomen, renal Doppler and urine catecholamine. All investigations came out to be unremarkable. After five days of hospitalization, patient was discharged. Blood pressure remains normal during follow-up.

DISCUSSION

Approximately 35,000 people die every year following snake envenomation in India, in which most common cause is delay in early diagnosis and medical treatment[2]. Presynaptic blockade by krait toxin and post synaptic blockade by cobra toxin are attributed in Neuroparalysis caused by these snakes [1]. Envenomation with these snakes may presents as drooping of eyelid, external ophthalmalgia, difficulty in swallowing, respiratory and generalized muscle weakness and associated with an overall mortality of 3.5% [5]. Autonomic dysfunction in snakebite may present as abdominal pain, vomiting, mild-to-moderate hypertension or hypotension and cardiac arrhythmia. Cause of autonomic dysfunctions is not clear and still under study. However, Alpha-2 adrenergic blockade at presynaptic level which block release of norepinephrine can be attributed to the cause [6]. Hence, this process gives rise to sympathetic overactivity and decreased parasympathetic stimulation [6-8]. The α- and β-bungarotoxin of krait mainly inhibits the release of acetylcholine causing paralysis, but cases have been reported with the possibility of pulmonary edema along with ventricular fibrillation and also fulminant myocarditis [12,13]. Hypertension without neurotoxic symptoms was observed in patients with Western Russell's viper envenomation [10], and neurotoxic signs without hypertension were observed in an episode of the Eastern Russell's viper envenomation. It therefore appears that different toxins are responsible for cardiovascular and neurological symptoms [9]. In Our case definite diagnosis about type of snake cannot be made but as per presentation and bite mark it seems to be krait. In a study of common krait bites, 139 of 210 victims (66%) exhibited autonomic dysfunction, which was more marked in those with severe envenomation [6]. Although snake was not seen by any family member but it is well known that kraits are active and agile at night, and during the rainy season, they frequently seek refuge in dry places, such as those inside a house or dwelling place [14]. Additionally, if humans are bitten by krait during their sleep, they are seldom aware of it, as their experience of the bite generally resembles that from an ant or a mosquito [14].

CONCLUSION

Early diagnosis and management is important to reduce morbidity and mortality

in snake envenomation. In children, lack of proper history makes it difficult for physician to ascertain the diagnosis. High dose of venom in comparison to body make them more prone to envenomation and autonomic dysfunction. Site of bite may also correlate with autonomic instability in our case, though not mentioned anywhere and need further research. For better outcome, limb immobilization, prompt ASV administration and ventilator support should be there along with vigilant monitoring for autonomic dysfunction.

Declaration of patient consent:

The authors certify that they have obtained all appropriate patient consent forms regarding images and other clinical information to be reported in the journal.

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