Case Report

Krait snakebite mimicking brain death: a case report from central India

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Abstract

Snakebite is a common medical emergency in India. Cobra (*Naja naja*) and common krait (*Bungarus caeruleus*) are important neurotoxic snakes causing neuroparalysis. We are reporting a pediatric case report of krait snakebite showing respiratory failure, areflexia, internal and external ophthalmoplegia, and loss of brain stem reflexes mimicking brain death. Mechanical ventilation was continued despite features suggestive of brain stem dysfunction. After 86 h of ventilation, child was weaned off from mechanical ventilator with excellent clinical outcome.

KEY WORDS: Snakebite, krait snake, mimicking brain death

Introduction

Snakebite is a major public health problem in India. According to toxicity, snakebite is divided into three major categories: hematotoxic, neurotoxic, and myotoxic. Neurotoxic envenomation presents as a broad spectrum starting from ptosis and ophthalmoplegia to respiratory arrest and loss of brain stem reflexes mimicking brain death, thus prompting clinical staff to consider withdrawing ventilator support, which is harmful. Supportive care needs to be continued till the effect of venom wears off with excellent prognosis. In this case report, we are presenting a pediatric case mimicking brain death where proper use of antisnake venom (ASV), ventilator support, and other medical care saved the life of child.

Case Report

An 11-year-old boy got snakebite while sleeping on floor on July 31, 2014, at 1.30 a.m. on left side ring finger. The snake was spotted and killed and carcass was examined by forensic expert at Netaji Subhash Chandra Bose Medical College & Hospital, Jabalpur, Madhya Pradesh, India. The snake was 1.2-m long glistening black with single white arches across the body resembling common krait (*Bungarus caeruleus*). On examination (2.30 a.m.) two fang marks were present on left ring finger with no signs of inflammation. Child was drowsy but arousable and obeying verbal command, dropping of eyelid present, grade 3-4/5 power in all four limbs, and deep tendon reflexes were normal. His pulse rate was 120/min, respiratory rate (RR) 36/min, and blood pressure (BP) 110/70 mmHg, and SpO_2 was maintained. Polyvalent ASV (enzyme refined equine immunoglobulin) 10 vials, neostigmine, and atropine were started immediately. After 2 h, child's respiratory pattern altered with poor respiratory effort and SpO_2 60% at 5 liters of oxygen by face and mask. Child was immediately intubated and put on mechanical ventilator on synchronized intermittent mandatory ventilator mode. Child's heart rate (HR) was 138/min, temperature periphery cold, and BP 90/70 mmHg. After two NS boluses (10 mL/kg), shock was improved. Further 10 vials of polyvalent ASV were repeated. Over a period of 5 hours, descending paralysis was seen. He was comatose and on examination, HR was found to be 118/min, temperature normal, BP 116/80 mmHg, RR on ventilator, no spontaneous movement, no respiratory effort, all deep tendon reflexes and doll's eye reflex absent, pupil was dilated, and fixed. All brain stem reflexes were absent mimicking brain death. He went on full ventilator support. Ventilation was continued despite findings suggestive of brain stem dysfunction and after about 72 h of ventilation, he showed flickering movement of fingers

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and toes. He was weaned off the ventilator and extubated after 86 h of ventilation. His vitals were as follows: HR 102/min, RR 34/min, BP 118/80 mmHg, and SpO_2 95% at 5 liters of oxygen, conscious, bilateral crepts present, pupil was mid-dilated, and sluggish reacting. After 3 days of extubation when child was shifted out of PICU, he had grade 3 power in both upper and lower limbs and was breathing well, weakness present at truncal muscles and lower limbs, and his pupils were mid-dilated and sluggish reacting to light. At time of discharge from hospital, all the vitals were within normal range. He has no dysphonia and dysphagia, grade 4 power in lower limbs, and grade 5 power in the upper limbs, and pupil is normal size and normal reacting.

Discussion

India and south Asian countries constitute the majority of world snakebite deaths. According to the 2008 global snakebite report, the annual mortality due to snakebite is 5.6–12.6 per 100,000 populations. According to the American Society of Tropical Medicine and Hygiene, more than 2.5 lakh cases of snakebites occur in India leading to 46,000 deaths every year.[1] In India, two-third bites are by saw-scaled viper, about one-fourth by Russell viper, and a small proportion by cobra and krait.[1] The snakebites are more common after rains, after flood, during harvest, and at night.[2] Many bites like the one in our case occur at night when the snake enters the house in search of its prey and people sleeping on the floor may be bitten. The snakes are classified into two important families, Elapidae and Viperidae. Elapidae have short, permanently erect fangs, and include cobra, krait, coral snakes, and sea snakes. Viperidae, however, have long fangs folded up against the upper jaw, which are erected when snakes strike. This family consists of snakes such as the typical vipers and pit vipers.[2]

The neurotoxic envenomations have broad range of presentation from ptosis and ophthalmoplegia to respiratory arrest and death. Neuromuscular paralysis occurs due to blockade of neuromuscular transmission. Cobra venom acts post-synoptically whereas krait venom acts pre-synoptically.[2] Bites of the common krait have been associated with profound neurotoxicity but relatively few local signs or symptoms. Cases have been reported with patient having severe neuroparalytic features without any history of snakebite due to lack of local symptoms.[3,4] Neurotoxins cause muscle paralysis by blocking the nicotinic acetylcholine at post-synaptic motor endplates or they may affect the mode of neurotransmitter release at the pre-synaptic motor nerve endings. The binding of the pre-synaptically portions is irreversible, hence clinical recovery occurs slowly and only with the formation of a new neuromuscular junction.[5] Autonomic disturbance in relation to krait bite characterized by hypertension, tachycardia, and mydriasis has been well described in the literature.[6]

This case report highlights absence of inflammation at common krait bite site but loss of brain stem reflex mimicking brain death, thus prompting clinical staff to consider withdrawing ventilator support, which is harmful. In such case, electroencephalography, four-vessel cerebral angiography, transcranial Doppler ultrasonography, or radionucleotide imaging (technetium Tc99m exametazime) should be resorted.[7] Supportive care needs to be continued till the effect of venom wears off with excellent prognosis. Proper use of ASV, ventilator support, and knowledge of different clinical scenarios can prevent the morbidity and mortality of victim.

Conclusion

A pediatric case report of krait snakebite showing respiratory failure, areflexia, internal and external ophthalmoplegia, and loss of brain stem reflexes mimicking brain death was presented. Mechanical ventilation was continued despite features suggestive of brain stem dysfunction. After 86 h of ventilation, child was weaned off from mechanical ventilator with excellent clinical outcome.

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