CASE REPORT

SEVERE NEUROPARALYTIC SNAKE BITE

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Abstract

Three patients with severe neuroparalytic snake envenomation were admitted in our intensive care unit over one month period. Initially, ptosis and ophthalmoplegia occurred followed by areflexia, bulbar palsy, respiratory muscle weakness and loss of brain stem reflexes mimicking brain death chronologically. They received cardiopulmonary support with mechanical ventilation, anti-snake venom (median dose of 20 vials) and anti-cholinesterase therapy. Mechanical ventilation was continued despite features suggestive of brain stem dysfunction. They required mechanical ventilation for 8, 5 and 2 days respectively. Except one patient suffering from residual weakness of lower limb, the rest survived with complete neurological recovery. Neuroparalysis is known with cobra envenomation but the prolonged loss of brain stem reflexes can be difficult to interpret for the caregivers about the efficacy of the treatment. However, good outcome in such cases is related to early cardiopulmonary support and anti-venom therapy.

Keywords: anticholinesterase, envenomation, polyvalent anti-snake venom

Introduction

Snakebites remain a public health problem in India even though it is difficult to be precise about the actual number of cases. According to toxicity, they are categorized as hematotoxic, neurotoxic and myotoxic. (1) Among the neurotoxic group, the majority of bites are due to Naja naja (common cobra), Ophiophagus hannah (king cobra) and Bungarus caeruleus (Krait) in India. (1) The snake venom consists of different enzymatic and non-enzymatic components loosely categorized as neurotoxins and haemorrhagens. (1) Neurotoxic envenomations have the potency to cause a broad spectrum of presentations starting from ptosis and ophthalmoplegia to respiratory arrest. Timely administered anti-snake venom and ventilator assistance can prevent the mortality and morbidity of the victim. (1) In our intensive care unit (ICU), all of the three cases admitted were from neurotoxic snakebites from the above-mentioned three commonest varieties. No patient presented with any bleeding episode, renal involvement or any cardiac injury.

Case Reports

Case 1

A 3 years old boy who was sleeping on the floor woke up at 4 am with pain and swelling over the right hand. Parents brought the child to the hospital within half an hour of the bite. The patient started complaining of difficulty in breathing and bluishness of body after 2 hours of bite. On examination, he had two bite marks (fang marks) on the right hand with associated swelling. There was no bleeding, blistering or necrosis at the site. On central nervous system (CNS) examination, he was conscious, alert, following verbal commands and localizing pain. After two hours of admission, he developed ptosis with absent power in all 4 limbs and absent reflexes. He was intubated and put on ventilator. Polyvalent anti-snake venom was given (20 vials) within first 6 hours. At six hours of admission, patient was comatose, having dilated pupils with no light response, areflexic and no motor response. Dolls eye movements were absent. Ventilation was continued inspite of brain stem dysfunction. He was given atropine and neostigmine. After 48 hours of ventilation, he showed some flickering movements of eyelids and continued to have improvement in next 3-4 days. On day 7, he developed right sided pneumonia which was managed with antibiotics. The child was extubated on day 8. On day 10, patient had limping gait and right lower limb weakness which was managed with limb physiotherapy. He had no dysphagia or dysphonia. On discharge, patient had grade 4 power in lower limb and grade 5 in upper limb.

Case 2

A 12 year old girl presented with history of snake bite on the right ankle, followed by difficulty in breathing, drowsy and inability to move the limbs. The patient's attendants had brought the dead snake which was identified as krait by forensic experts. She was also found to be hypertensive. She was intubated and put on ventilator, given anti-snake venom, atropine, neostigmine and antihypertensives. She started having movements on day 4. On day 5, she had full consciousness and full range of movements and was extubated. The patient did not have any residual neurological weakness.

Case 3

A 9 year old boy presented with history of snakebite on right thigh followed by sudden weakness and difficulty in breathing. He did not lose consciousness and was awake during the illness. He was intubated and put on ventilator, given anti-snake venom followed by atropine and neostigmine. Patient responded well and was extubated on day 2. He also did not have any residual neurological weakness.

Discussion

There are no reliable reports on the incidence of snake bites in India as many snake bite victims are treated by traditional practitioners and not in hospitals. An estimated 35,000 to 50,000 people die of snake bite each year in India. (2) Snake bites are more common after rains, after floods, during harvest and at night. (2) All the patients presented in monsoon season in our case series. Many bites like the one in our first case occur at night when the snake enters the house in search of its prey and people sleeping on the floor may be bitten. In many cases the history of a snake bite may not be forthcoming.

Snake venoms are complex mixtures of enzymes, low-molecular-weight polypeptides, glycoproteins, and metal ions. Among the deleterious components are hemorrhagins that promote vascular leakage and cause both local and systemic bleeding. (2) Proteolytic enzymes cause local tissue necrosis, affect the coagulation pathway at various steps, and impair organ function. Myocardial depressant factors reduce cardiac output, and neurotoxins act either pre- or post synoptically to inhibit peripheral nerve impulses. Most snake venoms have multisystem effects in their victims. (3) Envenomations by most viperids and some elapids with necrotizing venoms typically cause progressive local swelling, pain, ecchymoses and (over a period of hours or days) hemorrhagic bullae and serum-filled vesicles. (3) In serious bites, tissue loss can be significant. Systemic findings can include changes in taste, mouth numbness, muscle fasciculations, tachycardia or bradycardia, hypotension, pulmonary edema, hemorrhage (from essentially any anatomic site), and renal dysfunction. (3) Envenomations by neurotoxic elapids such as kraits (Bungarus spp.), many Australian elapids [e.g., death adders (Atractaspis spp.) and tiger snakes (Notechis spp.)], some cobras (Naja spp.) and some viperids [e.g., the South American rattlesnake (Crotalus durissus) and some Indian Russell's vipers (Daboia russelii)] cause neurologic dysfunction. Early findings may consist of cranial nerve weakness (e.g., manifested by ptosis) and altered mental status. Severe poisoning may result in paralysis, including the muscles of respiration, and lead to death due to respiratory failure and aspiration. (4) After elapid bites, the time of onset of venom intoxication varies from minutes to hours depending on the species involved, the anatomic location of the bite, and the amount of venom injected. Sea snake envenomation usually causes local pain (variable), myalgias, rhabdomyolysis, and neurotoxicity; these manifestations are occasionally delayed for hours. (3) All the children in our series had neuroparalytic snake bite. The reported incidence of neurological symptoms from two Sri Lankan studies on neuroparalytic snakebites was ptosis in 70% to 85%, respiratory muscle weakness in 18% to 45%, ophthalmoplegia in 53% to 75% and limb weakness in 27% to 54% respectively. (4,5) Our first patient had ophthalmoplegia which mimicked brain death. Withdrawing ventilatory support, in such a case could be disastrous. In such a case, other confirmatory tests of brain death like electroencephalography, four vessel cerebral angiography, transcranial doppler ultrasonography or radionuclide imaging should be done. (6)

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