

A Clinical Profile of Neurotoxic Snakebite in Pediatric Population of Jammu Region

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Abstract

Snake bite is a common neglected emergency in children. Due to lack of adequate epidemiological data its incidence is underestimated. This study analyses the clinical profile of neurotoxic snake bites in children. Retrospective analysis of 37 children with features of neurotoxicity with or without history of snake bite was done. 37 cases of neurotoxic envenomation had a median age of 8 years. 30 were males. Majority of bites occurred during night hours and in upper limbs. Among the 17 who were bitten during night 12 were sleeping on floor. The most common systemic presentation was ptosis (94.59%) followed by difficulty in breathing (78.37%), pain abdomen (64.86%) blurring of vision and diplopia (54.04%), decreasing level of consciousness (45.94%) and vomiting (29.72%). 13 patients (35.13%) with clinical features of neurotoxic envenoming but without any history of snake bite were brought with history of early morning sudden onset of abdominal pain. All patients were given Anti Snake Venom (ASV) with medial dose of 600 units. Twenty patients received ventilator support. Ten (27%) patients died all of whom received ASV after a delay of at least 5 hours.

Key Words

Neurotoxic Envenomation, Snake Bite, Anti Snake Venom

Introduction

Snake bite is a life threatening emergency and a high incidence of snake bite envenomation is reported from rural India but due to inadequate epidemiological data the incidence is underestimated (1). Snakebite is an environmental hazard associated with significant morbidity and mortality. Of more than 3000 known species of snakes worldwide, only about 500 are poisonous (2). In India, of the 216 species only 52 are poisonous, out of which the Saw scaled viper, Russell's viper, the Common Krait, and Cobra are the most common (3).

Although it is difficult to be precise about the actual number of snakebite cases, in India it is estimated that about 2 lakh persons fall prey to snakebite every year with an estimated fatality rate of 35,000-50,000/year (4). This high mortality due to snakebite is attributed to local superstitions, wrong practices, misconceptions regarding management and impractical treatment guidelines that impede doctors, especially those working in primary health

centres (5,6,7). Majority of snake bite related deaths in India's rural population are caused by neurotoxic envenoming by Kraits and Cobras (8,9). The common Krait (*Bungarus caeruleus*) is regarded as the most dangerous species of venomous snake in the Indian subcontinent (10).

Few studies are available regarding the clinical profile of neurotoxic snake bite particularly in the paediatric age group in India. The present study shows the varied clinical presentation, complications and their outcome in neurotoxic snake bite in children in Jammu.

Material and Methods

In this retrospective study we included children admitted over a period of 4 years in the Department of Paediatrics, SMGS Hospital, Government Medical College Jammu with clinical features of neurotoxicity with or without history of snake bite. Clinical features and circumstantial evidence were used to arrive at a diagnosis

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of neurotoxic snake bite, when there was no history of snake bite or the offending snake was not seen by the patient or the attendants. Clinical assessment included a detailed history and physical examination. Ptosis, paralysis of external ocular muscles, power of neck flexors and limb muscles, respiratory rate, chest expansion, strength of speech, ability to protrude the tongue beyond the teeth margin, level of consciousness, muscle tenderness, local effects at the bite site and evidence of autonomic dysfunction were all evaluated. Tidal volume was estimated by assessing the patient's capacity to count out loud in single breath. Depending upon the availability, ventilator support or manual ventilation using endotracheal tube intubation and self inflating bag was instituted in patients with respiratory paralysis.

Results

A total of 138 cases of snake bite were admitted in SMGS hospital between November 2007 and October 2011. Out of these 37(27%) cases were diagnosed as neurotoxic snakebites. The remaining cases were excluded from the study.

35 cases (94%) occurred during the months of July - October. The median age of the patients with neurotoxic snake bite was 8 years (range 3-15yrs) and males (30) outnumbered the females. Among those with history of snake bite (24/37), 13 (54.16%) patients were bitten on the upper limbs (arms, 5; hands, 5; fingers, 3) and 11 (45.8%) on the lower limbs (lower legs, 3; ankle, 4; feet, 4). Majority of the bites occurred during the night hours: 0200-0300 for 11 patients (45.8%); 0300-0400 for 4 patients (16.6%); 0400-0500 for 2 patient (8.3%). 6 patients (25%) were bitten between 1200-1600 hrs. Among the 17 patients who were bitten during night, twelve were sleeping on the floor at the time of bite.

Remaining 13 patients (35.13%) with clinical features of neurotoxic envenoming but without any history of snake bite were admitted in the emergency ward in early morning hours with history of sudden onset of abdominal pain starting around 0200 hrs and then gradually developing ptosis, respiratory failure and loss of consciousness. No fang mark was found on clinical examination.

The clinical presentation varied depending upon whether there was history of snake bite or not. Among patients with history of snake bite, the interval between the bite and reaching the hospital ranged from 30 minutes

to 29 hours. There were few local symptoms at the site of bite: pain in 9 cases, numbness in 2 cases. Six patients had clearly visible fang marks, pain at the bite site and local swelling, fourteen patients (64%) had faint fang puncture marks without pain, swelling or ecchymosis while four patients had no visible fang puncture marks. Remaining 13 patients (35%) without history of snake bite had no signs or symptoms of local envenoming. All of them reported in the early hours of morning with history of pain abdomen starting around 0200 to 0400 hours and gradually worsening with or without vomiting and later on developing ptosis, difficulty in breathing and drowsiness. (Table-1)

The predominant systemic presentation in all the patients included ptosis (94%), difficulty in breathing (79%), pain abdomen (65%) blurring of vision and diplopia (53%), decreasing level of consciousness (47%) and vomiting (29%). Less frequently observed findings include head drop seen in 2 patients while pain genitalia, retention of urine and pain throat was seen in one patient each. 82% patients developed respiratory failure requiring mechanical ventilation. Ptosis, the earliest sign of neurotoxicity, was first detected 30 minutes to 8 hours after the bite. Respiratory failure developed between 45 minutes to 10 hours after the bite.

On investigations, 15 patients had moderate anemia, 4 patients had prolonged whole blood clotting time (non clotting at twenty minutes) and 3 patients had deranged renal function tests.

All patients with systemic envenoming received ASV. Four patients had early anaphylactic reactions that were controlled with antihistamines and hydrocortisone. Twenty patients received ventilator support for overt respiratory failure and all survived without any sequelae. The median duration of hospitalisation among these was 8 days (range 5-13 days). Median dose of ASV administered to them was 600 ml (range 400-800 ml). The evidence of recovery was seen as early as 2 hours after starting ASV. The longest time taken for onset of recovery was 18 hours. Ten patients died (27%). All of these had respiratory failure. Six patients were brought in gasping condition and had suffered hypoxic insult to the brain enroute to this hospital as they were referred from areas about 150 kilometres from this hospital with a delay of 8 to 16 hours. Four patients died while on manual ventilation with

Table 1. Clinical Presentation

Clinical Sign	No. of cases	Percentage
Ptosis	35	94.59%
Dyspnea	29	78.37%
Pain abdomen	24	64.86%
Diplopia	20	54.04%
Decreasing level of consciousness	17	45.94%
Vomiting	11	29.72%

endotracheal tube and self inflating bag. In all these patients there was delay in starting ASV by more than 5 hours. All those who survived despite late admission to this hospital had started receiving ASV soon after the bite at the local hospital from where they were referred.

Discussion

In India, nearly two-third of snake bites are due to Saw Scaled viper, about one fourth due to Russel's viper and smaller proportions due to Cobra & Kraits (11). Another study done by Bhat *et al* in 1974 in Jammu attributed 95% of snake bites to saw- scaled viper (12). However, in the present study we found that 27% of the snake bite cases in children in Jammu were due to neurotoxic snakes. This disparity could be due to poor reporting of the patients to the hospitals and higher mortality before reaching the hospitals about three decades back as the medical facilities and the modes of transportation in the rural areas during those times were not as advanced as now. This could also be because many times the diagnosis of neurotoxic snake bite is missed because of lack of history of snake bite and absence of local signs and symptoms particularly in cases of Krait or Cobra bites (10). We also observed paucity of local tissue reaction in majority (82%) of our patients.

The incidence of neurotoxic snakebite shows a distinct seasonal pattern closely related to rainfall and temperature which compels the reptiles to come out of their shelter. Bawaskar *et al* in their study also found that 85% of neurotoxic cases occurred during June-October months (13). In the present study 94% of the bites occurred during the months of July to October which is the period of maximum rainfall in this region. Kraits are usually nocturnal in habit and usually found around houses and move during night for prey. Majority of the bites in the present study occurred between 0200 and 0500 hours while thirteen patients with no history of snake bite presented with features of neurotoxicity in the early

morning hours indicating that they may also had been bitten by the Kraits during the night. Hati *et al* (14) also observed that most of the identified Krait bites in Raidighi West Bengal were bitten in the third quadrant of the night when episodes of rapid eye movement (REM) sleep, associated with dream anxiety attacks and involuntary movements become more pronounced. These bites occur at night when transport facilities are poor, and the resulting delayed hospitalisation increases fatalities. Various signs of neurotoxicity appear usually within 6 hours of bite (15). Therefore, early recognition of signs of envenomation and timely administration antisanke venom is vital in saving life of snake bite victim as it is the only effective measure to neutralize the circulating toxins (16). We also observed a delay in starting ASV by more than 5 hours in all the patients who died. Snakes usually bite people on the lower limbs (feet and ankles) in response to being trodden on inadvertently; however Kraits bite any part of a recumbent person (14, 17). In the present study 55% of the bites were on the upper limbs while 45% on lower limbs.

Elapid bite envenomation produces a clinical syndrome characterized by abdominal pain, vomiting, negligible local signs and descending paralysis starting as soon as 30 minutes after bite or sometimes as late as 4 hours (18). Mitrakul *et al* (19) in their study observed that the characteristic systemic signs of elapid bite were those resulting from the neuromuscular effects of the venom and included ptosis, frothy saliva, slurred speech, respiratory failure, and paralysis of the skeletal muscles. These episodes occurred within 8 hours in 94% of the cases, and at the latest 19 hours following the bite.(19) Most patients are unable to identify the snake because of ignorance or poor visibility in darkness. Pain abdomen, the cardinal symptom of Krait bite, can precede neurological symptoms by several hours and at times may be the only presenting complaint particularly in the early morning hours.

The most important manifestation in cases of elapid envenoming is the sudden development of respiratory paralysis. Artificial respiration in individuals with respiratory failure can be life saving. This emphasises the importance of anticipation of this complication and timely intervention. A bitten individual with minimal neuroparalytic signs and symptoms should be kept in semi prone position to avoid aspiration, or an endotracheal tube should be inserted to prevent aspiration. Development of respiratory paralysis requiring mechanical ventilation was observed in 82% of patients in the present study while Ariaratnam *et al* (18) and Adhisivam *et al* (20) in their studies reported 64% and 50% of their cases requiring mechanical ventilation respectively.

The most effective treatment for snake bite is the monospecific anti snake venom (ASV), but it is very costly and rarely available in developing countries and it is really difficult to identify the type of snake most of the times. (12) Therefore in developing countries polyvalent ASV is administered on routine basis along with supportive care for management of snake bite cases. The median dose of ASV administered in the present study was 600 ml (400-800 ml).

Conclusion

The results of this study provide a highly distinctive pattern of epidemiology and symptoms of neurotoxic snake bites in children which can be of help in early diagnosis and management of neurotoxic envenomation in which there is no history of snake bite or the snake is not seen, brought in, or correctly identified. Neurotoxic snake bite must be kept as an important differential diagnosis in a child presenting in emergency in early morning hours with abdominal pain, vomiting, ptosis and descending paralysis even in the absence of any history of snake bite or visible bite marks.

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