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Original Article

Neurotoxic Snakebite in Jammu Region: Is it Cobra or Krait

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Abstract

Background: The aim of this study is to analyze the clinical profile and outcome of the neurotoxic envenomation in children in Jammu region and to identify the species based on the syndromic approach developed by WHO.

Materials and Methods: A retrospective hospital record based descriptive study which analyses the case records of children reporting to pediatric emergency with signs and symptoms of neurotoxic envenomation.

Results: A total of 22 cases of the neurotoxic envenomation reported between April 15 and October 15. These included 14 males and 8 females between the age group of 2.5 years and 16 years. The highest incidence of snakebite was observed in the age group of 4–8 years. A total of seven cases presented neuroparalytic symptoms and local signs suggesting cobra bite. Bite was reported in the afternoon or evening hours between 12.30 pm and 10.30 pm and 83% bites were outdoors. A total of 15 children presented with neuroparalytic symptoms with no local signs suggesting krait bite. 86% of the bites were indoor with onset of symptoms between 12 am and 7 am.

Conclusion: Both cobra and krait cause neurotoxic envenomation in children in Jammu region with krait bite accounting for 68% of the total cases. Most of these cases are brought to the pediatric emergency late. Training of the peripheral doctors regarding early recognition of neurotoxic snakebite, species diagnosis as per the WHO syndromic approach, prompt institution of initial management with neostigmine and after visit summary, endotracheal intubations and AMBU bag ventilation, and quick referral to a center with ventilator facility should help in reducing the morbidity and mortality due to krait and cobra bite in children.

Key words: Cobra bite, Envenomation, Krait bite

INTRODUCTION

Neurotoxic snakebite is a well-known pediatric medical emergency. Neurotoxic snakes belong to the family Elapidae which includes cobra and krait. Russell viper's venom has also been reported to cause neurotoxicity in some patients. Majority of snakebite-related deaths in India's rural population are caused by kraits and cobras. Russell viper's venom has also been reported to cause neurotoxicity such as ptosis, ophthalmoplegia, and respiratory distress in some patients.[1,2]

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The type of snakes found in particular area varies considerably. Furthermore, the symptoms and signs of neurotoxic envenomation vary according to the species of snake responsible for the bite and the amount of venom injected. Diagnosis of species of the snake responsible for bite is, therefore, important for optimal management and can be strongly suspected from the patient's description of the snake, the circumstances of the bite, or from knowledge of the clinical effects of the venom of that species.[2,3]

A syndromic approach has been developed by the WHO for diagnosing the species responsible for snakebite in different parts of the Southeast Asia region. The patient should be observed closely to allow recognition of the emerging pattern of symptoms, signs, and results of laboratory tests, "the clinical syndrome," which together with the circumstantial evidence, may suggest that the species was responsible for envenomation. This information will enable

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the doctor to anticipate the likely complications and manage the patient appropriately, thereby reducing the mortality associated with neurotoxic envenomation.[3]

Cobra bites tend to occur during daytime and early darkness while going to the open toilet, playing near the loose stones, searching ball in bushes, putting sticks in grooves, and improper or careless handling while rescuing the cobra. Bite by cobra results in tender local swelling, blistering, and necrosis. Victims experience severe pain at bite site having fang marks, with rapid progression of swelling. Skin at or around the bite site is ecchymosed. Subsequent formation of tense blebs and massive damage of skin and subcutaneous tissue occurs due to myocytolysis. This rapidity of the onset of symptoms prompts the rural victim in India to seek care quickly after cobra bite.[1,4-6]

The common krait, on the other hand, is nocturnal, terrestrial snake that enters human dwellings in search of prey such as rats, mice, and lizards during the course of hunting activity. It resides in the vicinity of human habitation, near the wattle and daub, mud, and small hut dwelling, hunts nocturnally, and is quick to bite people sleeping on the floor, often without waking their victims since venom is painless and associated with minimal local changes.[1,7]

Cobra venom is rich in postsynaptic neurotoxins called alpha-bungarotoxin and cobratoxin, which bind, especially to postsynaptic acetylcholine receptors, preventing the interaction between Ach and receptors on postsynaptic membrane, resulting in neuromuscular blockade. Cardiotoxin content of cobra venom has direct action on cardiac and smooth muscles causing circulatory failure, cardiac arrhythmias, various heart blocks, and cardiac arrest. Cobra venom is of smaller molecular size and rapidly absorbed into circulation. Cobra unlike the krait deposits its venom deeply. This in combination with hyaluronidase allows spread of the venom rapidly and symptoms to arise abruptly. Absorption is further accelerated by running due to fear and the liberated catecholamines can kill the victim within 8 min. Interestingly, severe, irreparable local tissue is lost at the bite site of cobra envenoming due to myocytolysis. Paralysis is heralded by ptosis followed by ophthalmoplegia. Blurring of vision and loss of accommodation are earliest sign of neurological envenomation. Paralysis of facial, palatal, tongue, and neck muscles follow. Respiratory failure, precipitated by upper airway obstruction and paralysis of intercostals and diaphragm, is the usual cause of death.[1,6,8]

Although the krait venom is 10 times more lethal than cobra, it is absorbed slowly as skin has poor circulation and reflexes are blunted during sleep. The common Indian krait venom contains both presynaptic beta-bungarotoxin and alpha-bungarotoxin. Beta-bungarotoxin in the krait venom has a great affinity toward presynaptic Ach receptors. These toxins initially release Ach at the nerve endings, at neuromuscular junction, and then damage it subsequently preventing the release of Ach. The tissue having high concentration of these receptors is affected in the following order, such as sphincter pupillae, levator palpebrae superioris, neck muscles, bulbar muscles, subsequently limbs, and, finally, the diaphragm and intercostals muscles. Venom acts as early as 30 min and till 18 h.[9-12]

Optimal management of neurotoxic snakebites depends on the species. Neuromuscular blockade by the short chain neurotoxin present in the cobra venom (cobra toxin, alpha-bungarotoxin) is more readily reversible than with a long chain toxin (beta-bungarotoxin). Most of the patients recover with artificial ventilation, cardiopulmonary resuscitation, and acetylcholinesterase inhibitor (AChEI). Since cobra venom is reversibly attached to postsynaptic receptors, AChEI like neostigmine is important for management. 0.5 mg neostigmine ½ hourly preceded by atropine may help the majority of victim to recover within 24 h. This cycle may not be required >5–6 times. Local wound care is done by intravenous antibiotic, daily dressing and may require plastic surgery.[1-3]

The common krait venom contains both pre- and post-synaptic blocker. Thus, the victims may or may not respond to neostigmine. Envenoming by krait has an early phase profound paralysis which lasts for 30–60 min followed by deep paralysis phase which lasts for 2–3 days and then recovery phase ranging from 2 to 3 weeks. Since the krait venom damages the acetylcholine receptors, prolonged period of ventilatory support, and intensive care requirements essential for recovery.[3,13]

The epidemiology of neurotoxic snakebite in children in Jammu region has not been adequately studied. Many snakebite cases are not treated in hospitals but by traditional healers. The chances of snakebite deaths being missed are perhaps even greater than from deaths occurring from several other causes.

Aims and Objectives

This study aims to study the clinical profile and outcome of the neurotoxic envenomation in children in Jammu region and to identify the species responsible based on the WHO syndromic approach.

Settings

This study was conducted at the Department of Pediatrics, SMGS Hospital Government Medical College, Jammu.

Study Design

This was a retrospective hospital record-based descriptive study.

MATERIALS AND METHODS

Neurotoxic snakebite cases commonly report to the casualty of the Pediatrics Department, SMGS Hospital, Jammu. Since maximum number of snakebite cases occur during summer, monsoon, and post-monsoon months, we decided to carry out retrospective hospital record-based study from April to October 2015. The case records from the record section of SMGS Hospital for all the children who reported to the Pediatric Emergency with signs and symptoms of neurotoxic envenomation were evaluated. The clinical profile of patients including age, sex, residence, site of bite, time interval between snakebite and hospitalization, and clinical symptoms and signs were recorded. An attempt was made to interpret the clinical signs and identify the species based on the WHO syndromic approach. Local envenoming (swelling etc.) with paralysis = cobra or king cobra paralysis with minimal or no local envenoming/Bitten on land while sleeping on the ground = krait

RESULTS

A total of 22 cases of the neurotoxic envenomation who had been hospitalized between April 15 and October 15 were included in this study. Of these, 14 were male and 8 were female. The age group was between 2½ years and 16 years. The highest incidence of snakebite was observed in the age group of 4–8 years.

About 68% of children reported with symptoms suggestive of krait bite while 32% were cobra bites.

A total of seven cases presented neuroparalytic symptoms and local signs suggesting cobra bite. Age range was from 2.5 to 13 years. 66% were male. Bite was reported in the afternoon or evening hours between 12.30 pm and 10.30 pm. 83% bites were outdoors. Patients responded well to neostigmine and after visit summary (AVS). Two patients required mechanical ventilation and mean hospital stay was 5 days. One patient died due to cardiac arrest during transportation to intensive care unit.

A total of 15 children presented with neuroparalytic symptoms with no local signs suggesting krait bite. The age group was 5–16 years and 9 were male. Eight patients belonged to Jammu district, four to Kathua district, one to Samba, two to Reasi district, and one from Doda district. 86% bites were indoor. All the children started with the symptoms at night or early morning between 12 am and 7 am. Of these, eight children were brought >6 h after the onset of symptoms while seven were brought within 6 h. In seven patients, fang marks could not be located, three children had fang marks in the upper limbs, four in lower limbs, and one in head and neck (Post-auricular region). Five children had history of sleeping on floor and two had history of sleeping outdoors. However, in 13 patients, there was no history of snakebite or having seen the snake. However, in all these cases, circumstantial evidence along with clinical signs and symptoms of a neurotoxic snakebite were found. These patients had mostly sequence of events, starting with pain throat and pain abdomen, followed by drowsiness, ptosis, and difficulty in breathing. Abdominal pain (60%), ptosis (100%), dysphagia (60%), dyspnea (50%), drowsiness (40%), blurring of vision (25%), pain throat (25%), dryness of mouth (15%), diplopia (10%), and vomiting (10%) were the predominant clinical presentations [Table 1-4].

DISCUSSION

Neurotoxic snakebite is a common pediatric emergency during the summer months. In the present study, 68% of the cases of neurotoxic envenomation appeared to be due to krait bite as per the WHO syndromic classification. Digra and Singh in their study of 37 children with neurotoxic envenomation from Jammu between 2007 and 2011 observed that 94% of the bites occurred during the months of July–October. They further observed that majority of the bites occurred during the night hours and 12 children were sleeping on the floor at the time of bite. 13 children with clinical features of neurotoxic envenomation but without a history of snakebite were admitted in emergency in early morning hours with a history of sudden onset of the abdominal pain and then gradually developing ptosis, neuromuscular paralysis, and respiratory failure.[14]

Table 1: Age- and sex-wise distribution of neurotoxic snakebite patients

Age (years)	Male	Female	Total
0–3	1	0	1
4–8	4	4	8
9–12	4	1	5
13–15	4	2	6
16–18	1	1	2
Total	14	8	22

Table 2: Distribution of the cases studied according to the clinical syndrome

Paralysis with minimal or no local envenoming/Bitten on land while sleeping on the ground=krait	15
Local envenoming (swelling etc.) with paralysis=cobra or king cobra	7

Table 3: Characteristics of suspected cobra bite patients

Age	Sex	District	Time of bite (PM)	Time gap of bite and hospitalization	Indoor/outdoor	Site of bite	Hospital stay days	Outcome
12	M	Rajouri	4.30	4.5 h	Outdoor	LL	5	Recovered
5	F	Rajouri	10.30	5.5 h	Indoor	LL	6	Recovered
2.5	M	Jammu	12.50	2 h	Outdoor	UL	4	Recovered
7	M	Samba	1	9 h	Outdoor	LL	5	Recovered
13	M	Jammu	3	45 min	Outdoor	LL	9	Recovered
8	F	Reasi	5	6 h	Outdoor	LL	-	Died
13	M	Kathua	1	2 h	Outdoor	LL	11	Recovered

LL: Lower limb, UL: Upper limb

Table 4: Characteristics of suspected krait bite patients

Age	Sex	District	Time of bite/onset of symptoms	Time gap of hospitalization (h)	Indoor/outdoor	Site of bite	Hospital stay	Outcome
13	F	Doda	7 AM	10	Indoor	LL	15	REC
5	F	Jammu	1.30 AM	2.5	Indoor	UL	6	REC
10	M	Reasi	3 AM	12	Indoor	Left ear	4	REC
12	M	Samba	7 PM	2	Outdoor	LL	13	REC
7	M	Jammu	7 AM	4	Indoor	?	7	REC
13	M	Jammu	3 AM	4	Indoor	?	13	REC
8	F	Jammu	5 AM	2.5	Indoor	UL	15	REC
14	M	Reasi	9 AM	24	Indoor	UL	4	REC
16	M	Jammu	5.30 AM	7	Outdoor	?	24	REC
5	M	Kathua	6 AM	6.5	Indoor	?	10	REC
8	F	Kathua	5 AM	4	Indoor	?	10	REC
14	F	Kathua	4 AM	6	Indoor	?	8	REC
16	F	Kathua	1.30 AM	12.5	Indoor	UL	5	REC
8	M	Jammu	3 AM	6	Indoor	?	7	REC
10	M	Jammu	12 AM	2	Indoor	LL	10	REC

LL: Lower limb, UL: Upper limb, REC: Recovered

In the present study, most of the children with krait bite were bitten during unprovoked encounters, indoors, and mostly at night and were brought to the hospital quite late, with eight patients reporting 6 h after the onset of symptoms. This could be due to a delayed diagnosis and delayed referral as pain abdomen in early morning hours was the cardinal symptom in 60% of the cases. Timsinha *et al.* in a study of 91 cases of the snakebite observed that 46% of the patients reported to the hospital 6 h after the bite and 92.3% of the patients did not receive any first aid measures before hospitalization, which could be due to lack of transport facilities or awareness. Definite protocol for the primary health care of snakebite cases needs to be considered.[15]

Snakes like krait, on envenomation, may not present with local signs, thereby misleading the physician to think of others possibilities and in the process allowing the golden hour to pass by. Newly posted or inexperienced doctors and inadequate facilities at primary health centre, ignorance of conventional treatment of neurotoxic snakebite by doctors, further delays appropriate treatment of victims and contribute to increasing morbidity and mortality.[9-13,16] Studies of snakes with presynaptic neurotoxins such as kraits suggest that antivenom does not reverse established neurotoxicity, but early administration may be associated with decreased severity or prevent neurotoxicity.[17]

In cobra bite anticholinesterases and supportive care as cornerstones of management. Faridi *et al.* reported complete reversal of envenomation symptoms in a cobra bite patient following the administration of anticholinesterase neostigmine methyl sulfate and emphasized that anticholinesterase drugs may reverse the potentially lethal neurological effects of venom.[18] In our study, none of the children reporting to the hospital with cobra bite had received neostigmine as a part of prehospital treatment. This signifies a lack of popular awareness about current prehospital management recommendations for snakebites. Bomb *et al.* in their study reported that 12 patients of Elapid ophitoxemia with neuromuscular paralysis were administered anticholinesterase (neostigmine). In four of these patients, no antivenom and all survived. Of eight who received antivenom three were given <50 units and all three survived. Of the remaining five, despite the use of AVS in higher doses (>50 units of AVS), two died. The authors concluded that antivenom has no definite role in Elapid ophitoxemia and anticholinesterase drugs alone, with good supportive care can result in satisfactory outcome.[19]

Availability of ventilators in tertiary care centers has improved the outcome of the snakebite patients. 65% of our patients required ventilator support as they had developed respiratory paralysis. Usually, neurotoxicity occurs within 60 min of envenomation, rapidly progressing to respiratory paralysis requiring early ventilator support.[20]

Rapid referral to hospital for definitive care and antivenom administration is the cornerstone of the management of neurotoxic snakebite. Singh *et al.* in a study of 21 cases of the neurotoxic snakebite from a military hospital, where reporting time was between 30 min and 180 min, median dose of AVS was 180 ml and 11 (52%) of patients received neostigmine, only two patients needed ventilation. The median time of recovery from envenomation was 8 h and all patients REC.[21]

Case fatality rate in our series was 9%. The recorded figures of snakebite deaths in hospital are regarded as underestimated of the total fatality from this cause. Data from million deaths study in India estimate that snakebite deaths are >30-fold higher than recorded in the hospital returns.[22,23]

Limitations of syndromic approach: The more carefully the clinical effects of snakebites are studied, the more it is realized that the range of activities of a particular venom is very wide. For example, some elapid venoms, such as cobras, can rarely cause hematotoxic effects, formerly thought to be an effect only of viper venoms. In Sri Lanka and South India, Russell's viper venom causes paralytic signs (ptosis, etc.) suggesting elapid neurotoxicity. Ptosis, bulbar palsy, internuclear ophthalmoplegia, and respiratory paralysis due to presynaptic neuromuscular block in Russell viper's bite poisoning are often seen and reported from Kerala and Sri Lanka.[24] Although there may be considerable overlap of clinical features caused by venoms of different species of the snake, a "syndromic approach" may still be useful. Medical personal throughout the region would benefit from more formal instructions on all aspects of the subject. This should include identification of medically important species of snakes, clinical diagnosis and appropriate use of antivenoms, and ancillary treatments.

CONCLUSION

Both cobra and krait cause neurotoxic envenomation in children in Jammu region with krait bite accounting for 68% of the total cases. Most of these cases are brought to the pediatric emergency late. Pain abdomen, usually during night hours without any abdominal cause, should arouse a strong suspicion of neurotoxic krait bite. Training of the peripheral doctors regarding early recognition of neurotoxic snakebite, species diagnosis, prompt institution of initial management with AChEI and AVS, endotracheal intubations and AMBU bag ventilation, and quick referral to a center with ventilator facility should help in reducing the morbidity and mortality due to krait and cobra bite in children.

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