Original Article

Analytical study involving Neuroparalytic-snake Envenomation cases reported to a North Indian tertiary care Hospital

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ABSTRACT

Aims: To study the clinical profile of neuroparalytic snake envenomation in a tertiary care referral north Indian hospital.

Material & methods: Retrospective analysis of neuroparalytic snakebite cases admitted to the ICU (A&E) PGIMS, Rohtak from Jan 2010 to Dec 2014.

Results: Out of total 185 cases of snakebite there were 183 elapid bites with neuroparalytic symptoms and 2 viper bites having haemostatic abnormalities. 65% of cases of snakebite occurred when the patients were asleep. Urban to rural ratio was 1: 2.8 and male to female ratio was 3.1:1 .Median time to arrival at hospital was eight hours. 78.1 % of all elapid bites required mechanical ventilation (MV). Mean duration of MV was 3.8 ± 1.79 days. 21 of 178 patients who received antivenom had an adverse event. The average dose of antivenom was 25.3 ± 0.93 vials. There was no beneficial effect of giving neostigmine. Overall mortality rate was 6%.

Conclusion: Snakebite should be considered in all early morning myasthenic presentation and in patients coming in rainy season with respiratory failure with no prior symptoms. Ventilatory support along with anti-snake venom is life-saving. Significant side effects are seen with anti-snake venom, therefore it should be given under strict monitoring. Mortality due to snakebite depends on time delay in presentation and pretreatment cardiopulmonary arrest and sensitivity to anti-snake venom. There is an urgent need to educate rural population about the hazards and treatment of snakebite. Availability of antivenom at primary health centers and rapid transport facilities may decrease the morbidity with snakebites.

Keywords: snake bite; neuroparalysis; antivenom; neostigmine; mechanical ventilation.

INTRODUCTION

Snake envenoming is a major health problem in rural tropics, with large numbers of envenomations and deaths. An estimated 2 lakh envenomings and 35 000 to 50 000 mortalities occur due to snake bite every year in India.[1] Victims of snake bite are commonly rural population, paddy farmers or farm workers, and people who sleep outdoors. Most human snakebites occur during summer or rainy season. The principle effects of envenomation are on the nervous system, kidneys, heart, blood coagulation, vascular endothelium and locally at the site of bite. In Indian subcontinent most of the snake bites occur in uneducated rural people who believe in witchcraft and use traditional healers, therefore only cases of severe envenomation reach the healthcare centers and there is under reporting of snake bite cases. This study was carried out to describe arrival delays, clinical features, complications and outcome of snake bites seen in an intensive care unit of a tertiary care hospital of north India.

MATERIALS AND METHOD

This is a retrospective analytical study design involving Neuroparalytic-snake envenomation cases reported during the period from January 2010 to December 2014. All data, case-records available for the snake-bite cases admitted to the ICU (A&E) of our hospital; were scanned and compiled according to the inclusion and exclusion criteria. Our hospital is a Post Graduate Institute of Medical Science at Rohtak, catering major part of the state of Haryana (North India). Hence, the sample strength could covered a major parts of the community in North India. Our study included, those cases with the signs of neurotoxicity (ptosis, dysphagia, dyspnoea, limb paralysis, altered sensorium) and the data with the evidence of coagulopathy i.e. increased whole blood clotting time or spontaneous bleeding were excluded from the study. The demographic details and the clinical data were obtained, e.g. age, sex, and occupation, duration of admission, duration of ventilation and outcome for each snakebite cases admitted to the ICU

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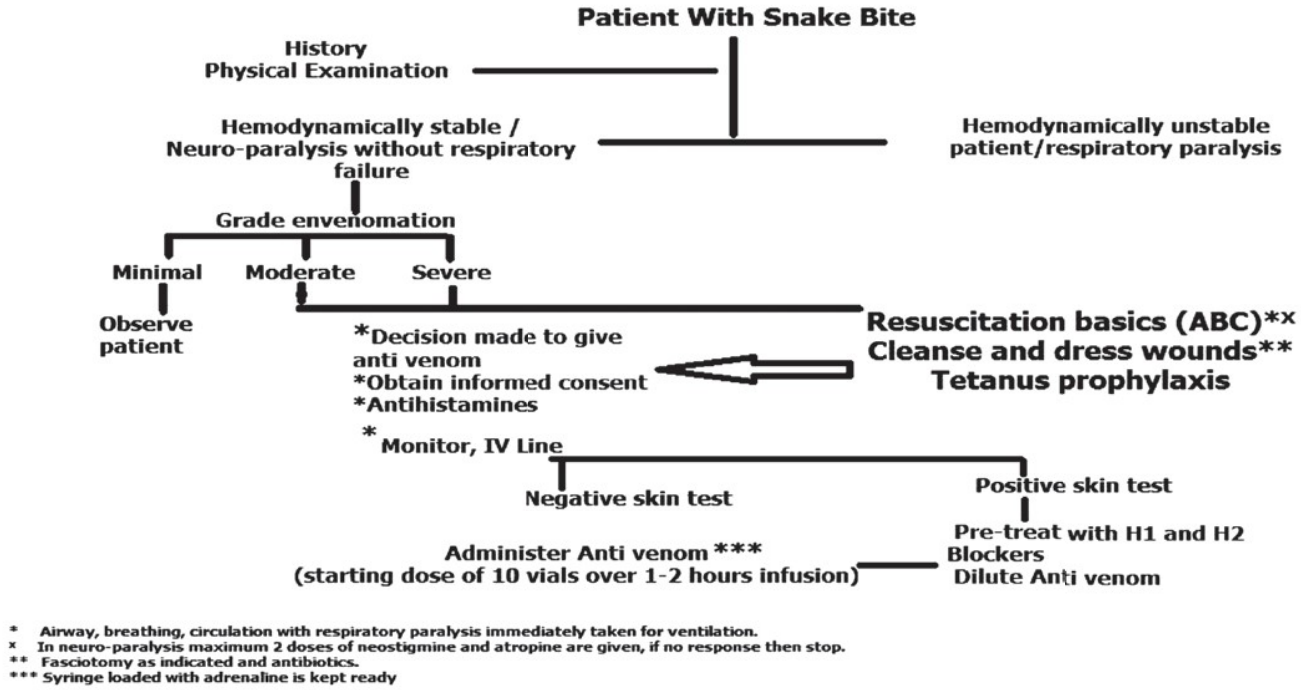
during the period from January 2010 to December 2014, and compiled for further analysis. The statistical analysis was done using SPSS 16 version software. This study, as the case applicable, is in accordance with our institute ethical clearance policies.

Fig. 1: showing Snake de-envenomisation protocol followed in our hospital at Rohtak.



* Airway, breathing, circulation with respiratory paralysis immediately taken for ventilation.
x In neuro-paralysis maximum 2 doses of neostigmine and atropine are given, if no response then stop.
** Fasciotomy as indicated and antibiotics.
*** Syringe loaded with adrenaline is kept ready

Source: A& E Department, Pt. BD Sharma PG Institute of Medical Sciences, Rohtak, India.

RESULTS

There were total 185 patients admitted to ICU of PGIMS for snake bite envenomation during the study period. Of these 183 patients had neuroparalytic features and were selected for study. Fifteen patients were bitten by common krait as it was confirmed by morphologically examining the dead snake provided by the victim. Eighty three patients were confirmed of krait bite by morphological description of snake and the circumstantial evidence. Two patients had features of coagulopathy on admission and were excluded from study and in rest 85 patients the species of snake could not be confirmed. The demographic profile of neuroparalytic snake bite patients is shown in table 1.Most of the snake bites occurred in rural areas (73.77%) and during rainy season July-September(62.84%). Some 65% of snakebites occurred when patients were asleep at night and 23% occurred during afternoon while working in field. Foot remains the most common site of bite seen in 38% cases. There were 140 males (76.5%) and 43 females (23.49%) with male: female ratio of 3.1:1. 78% patients were adults with range of 11-40 years. 80% patients were farmers and laborers. Pre-hospital management was reported in 63 (34.42%) of 183 patients (table 2). Pressure bandages had been applied in 21 patients, incision and drainage was done in 11 patients and tourniquet was applied in 31 patients. The median arrival times at hospital after the bite was 8 hours, with a range of 1- 32 hours and mean of 7 ± 0.76 hrs. Neuroparalytic features, a hallmark of cobra and krait bites were seen in 183 (98.9%) patients and hemostatic abnormalities attributable to viper bites were seen in only 2 (0.01%) patients. There were no patients with both neuroparalytic and hemostatic abnormalities. Minimal local reaction and fang marks are the most common complaint at presentation and ptosis was the most common sign at presentation. 5 patients had complete paralysis

mimicking brain death (Fig. 2). General treatment at our hospital consisted of maintenance of airway, breathing and circulation, tetanus toxoid administration and i.v. antibiotics. Specific treatment given to patients has been shown in table 3. Polyvalent equine antisnake venom was administered in 178 patients. Antivenom was withheld in 5 patients due to lack of systemic envenomation. Intradermal sensitivity testing was done in all patients. Mean dose of antivenom administered was 25.3 ± 0.93 vials with a range of 5 - 55 vials. The median dose of antivenom given was 20 vials. Premedication consisting of inj. Hydrocortisone, inj. Avil and inj. Paracetamol was given to all patients requiring antisnake venom. Simple allergic reaction to antivenom occurred in 12 patients and anaphylactic reaction requiring modification of antisnake venom (diluted/ slower administration) or requiring injection adrenaline occurred in 9 patients. Neostigmine was administered in 25 patients not showing improvement with second dose of antivenom. It was given in the dose of 2.5mg at 30 minutes interval for two doses after administration of 0.6mg of atropine. Patients were assessed for any improvement of neuroparalytic features (ptosis, neck muscle weakness, respiratory muscle weakness) at 10 min & 20 min after each neostigmine administration. Assisted ventilation was required for 143 patients, with a range of 1- 25days and mean duration of 3.8 ± 1.79 days. Forty patients were managed without ventilator support. Out of these 12 patients were intubated to prevent airway aspiration. Out of 183 patients with neuroparalysis, 172 patients recovered and 11 died giving a mortality rate of 6%. Of these 11 patients 5 patients had pretreatment cardiorespiratory arrest. Cause of mortality was respiratory arrest in 6/11 patients (54.54%), ventilator acquired pneumonia in 3/11 patients (27.27%) and severe sepsis in 2/11 patients (18.18%).

DISCUSSION

Snakebite is a common medical emergency encountered in India and is often under reported due to occurrence in uneducated rural population and labourers who believe in traditional methods of managing snakebites. World mortality from snake bite is estimated as 50,000 to 1,00,000 annually (McNamee 2001) and the greatest number of reported snake bite death occurring in Indian subcontinent is 10,000 to 15,000 annually.[2] Largest number of deaths reported in India are from Bengal, Uttar Pradesh (UP), Tamil Nadu, Bihar, and Maharashtra.[3] Neurotoxic snake envenoming is one of the most important causes of snake bite fatality and is mainly due to Elapidae family, which includes the Genus Naja and Bungarus, commonly referred to as the cobras and kraits respectively. Snakebites are usually seen in people of rural areas, construction workers and farmers working in fields4 or people sleeping outdoors.[5,6] In this study 74% of snakebites occurred in rural population. Most snakebites (65%) occurred during night time while asleep, in concordance with earlier reports.[7] 23% snakebites occurred in people engaged in field work. The exposed parts of body, mainly hands and feet (38%) were the sites bitten most commonly.[8] Most human snakebites occur during monsoon season because of flooding of the habitat of snakes and their prey. The breeding season of frogs closely follow the monsoons and rats and mice are always in close proximity to human habitat. In our study also most snake bites (63%) occurred during monsoon season. From another Indian study snakebites abound the months of July to September (57%)[9] and May to July (67%).[10, 11]

One important aspect of treatment of snakebite is care of local wound and early administration of antivenin injection. Sixty three of our patients had received local treatment of bitten site – that is pressure dressing, incision and drainage and tourniquet application. These practices are not recommended currently[12,13] and show the lack of awareness of hazards of snakebite, an unrelenting belief in traditional system of medicine and lack of knowledge in the importance of starting early treatment. The median arrival time after snakebite was 8 hours. This can be explained by the fact that access to villages by common mode of transport is still rudimentary, due to disbelieve of rural population, and a lack of proper primary healthcare facilities. Majority of snakebites admitted to our ICU were neuroparalytic with clinical features in descending order of frequency ptosis, respiratory muscle involvement, bulbar weakness followed by ophthalmoplegia as shown in Figure 1. The reported incidence of neurological symptoms in another study of a north west Indian hospital was ptosis (87%), respiratory muscle involvement (75%), bulbar weakness (67%), ophthalmoplegia (49%), paresis (26%), loss of consciousness (14%), giddiness (10%) and headache (8%).7Antivenom was given to 178 patients, although the dose of antivenom is not yet fixed, the indications are still well known. The mean dose of antivenom used in the study was 25.3 ± 0.93 vials. Adverse reactions to antivenom were seen in 21 patients

(11.5%), 12 patients had allergic reaction to antivenom while 9 had anaphylactic reaction requiring modification of antivenom or injection adrenaline but there were no deaths. According to Reid et al., ASV is not very effective against local effects of venom.[14] Neostigmine is shown to be beneficial in postsynaptic neuroparalytic snakebite but its role in presynaptic snakebite is still unclear.[15] Seneviratne U et al and Ahmed SM et al are supporters of this regime but Reid had doubts about the benefits of this mode of therapy.[16,17,14] In our study neostigmine was given to 25 patients. Neostigmine showed no beneficial effect against the neuroparalytic snakebite, similar outcomes have witnessed by some other authors too.[18] This may be due to the fact that common krait is the predominant species found in northwest India and it causes presynaptic neuroparalysis as opposed to snakebite by cobra which causes post synaptic neuroparalysis. The death rate in our study was 6% slightly higher as compared with mortality rate from other studies across India.[7,19,20] This may be due to the fact that cobra venom contains mainly post – synaptic neurotoxins, which have a curare – like effect and can be reversed by snake antivenom after clinical effects have developed, conversely, krait venom contains mainly presynaptic neurotoxins, which are not reversible once paralysis has developed and so respond poorly to delayed antivenom and many of our patients had delayed arrival to hospital. Endotracheal intubation was done in 155 patients, of these 12 were intubated to prevent airway aspiration. Ventilator support was required by 143 patients, with a mean duration of 3.8 ± 1.79 days.

CONCLUSION

Neuroparalytic snake envenomation with minimal local sign or fang marks is the commonest presentation in ICU. Snakebite should be considered in all early morning myasthenic presentation and in patients coming in rainy season with respiratory failure with no prior symptoms. Ventilatory support along with antisnake venom is lifesaving. Significant side effects are seen with antisnake venom, therefore it should be given under strict monitoring. Neostigmine is found to be of no benefit in presynaptic neuroparalytic snakebite due to common krait (Bungarus caeruleus). Mortality due to snakebite depends on time delay in presentation and pretreatment cardiopulmonary arrest and sensitivity to antisnake venom. There is an urgent need to educate rural population about the hazards and treatment of snakebite. Availability of antivenom at primary health centers and rapid transport facilities may decrease the morbidity associated with snakebites.

Competing interest: the authors declare there are no competing interests.

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Fig. 2: Distrbution of Clinical presentations of neuroparalytic snake envenomation

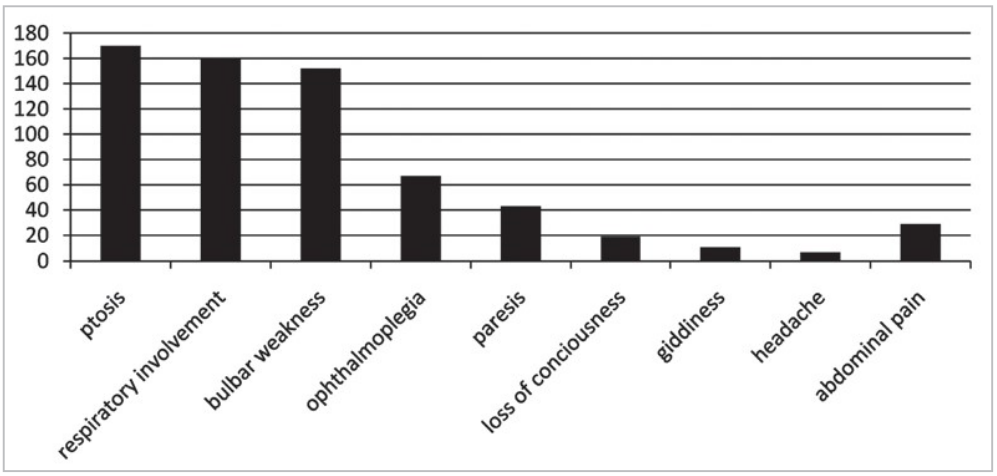

Table 1: Demographic profile of neuroparalytic snake bite patients

Parameter	Number (%)
Residence Rural urban	 135 (73.77%) 48 (26.23%)
Season of snake bite Rainy(July – September) Summer (April- June) Winter (December-March) Autumn(October- November)	 115 (62.84%) 68(37.15%) - -
Sex distribution Male female	 140 (76.50%) 43 (223.50%)

Parameter	Number (%)
Age distribution (Years) 11-20 21-30 31-40 41-50 51-60	 10 (5%) 78(42.62%) 55(30%) 29(15.84%) 11(6.01%)
Occupation Labourer Farmer Skilled worker House maker	 106 (57.92%) 40 (21.85%) 27(14.75%) 10 (5%)

Table 2: Pre hospital management of neuroparalytic snake bite patients

Parameter	Number (%)
Pressure bandage	21 (11.47%)
Tourniquet	31 (16.93%)
Incision and drainage	11 (6.01%)

Table 3: Treatment given to neuroparalytic snake bite patients

Treatment given	Number of patients (%)	Mean ± SD	Range
Antivenom	178 (97.26%)	25.3 ± 0.93 vials	5-55 vials
Neostigmine	25 (13.66%)	-	-
Mechanical ventilation	143 (78.14%)	3.8 ± 1.79 days	1-25 days
Outcome Recovery Mortality	 172 (93.98%) 11 (6.01%)	3.8 ± 1.79 days	1-25 days
Causes of mortality Respiratory arrest Ventilator acquired pneumonia Severe sepsis	 6(54.54%) 3(27.27%) 2(18.18%)		

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