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A retrospective analysis of snake envenomation in the intensive care unit of a tertiary care hospital in Delhi

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ABSTRACT

Objective: To evaluate the epidemiology, clinical profile and treatment for patients with snake bite in the intensive care unit of our hospital.

Methods: A retrospective analysis of patients with snake bite admitted to the intensive care unit of a tertiary care hospital, North Delhi, was conducted between January 2013 and October 2018. Their demographic profile, time and site of bite, clinical manifestations and time interval between the bite and anti-snake venom administration were recorded.

Results: A total number of 102 patients with snake bite were studied, of which 54.9% were males and 45.1% were females. Most patients were presented in the monsoon season and snake bites occurred mainly in the night and early morning. A total of 56.3% of the patients with clinical manifestations received anti-snake venom within 3 h after the snake bite. Most patients presented with painless bites and neuromuscular features with ptosis that was the most common clinical manifestation (50.9%). Anti-snake venom was administered to 55 patients (53.9%) with an average dose of 42 vials per patient and the mortality was 11.7%.

Conclusions: Neuromuscular krait envenomation accounted for the highest incidence of venomous bites in our study. Early medical treatment with judicious anti snake venom administration and related education is crucial.

1. Introduction

Snake bite is a common public health emergency in developing countries. The World Health Organisation estimates worldwide incidence of 1 841 000 snake bites with an annual death incidence of 94 000 with India accounting for the majority of the cases. More than 81 000 snake bites are reported in India annually with a mortality rate of 11 000 cases each year[1].

However, there is widespread underreporting specially in a developing country like India where a significant number of poor people are illiterate and used to resorting to traditional treatment. Snake bites are more common in the rural areas where lack of easy access to medical care facilities. Hence, only a small percentage

of patients with significant clinical symptoms would visit the hospital. Accidental snake bites are usual in population with a habit of sleeping on the floor and open style defecation. Patients with snake bite envenomation may present with symptoms of the central nervous system, coagulation abnormalities with hemolysis and renal failure, disseminated intravascular coagulation or shock[2-4].

We regularly cater to large number of patients of snake envenomation in intensive care unit of our hospital. Majority of the patients belong to poor socio economic strata and have low awareness about proper treatment of snake bite. There is paucity of literature on snake envenomation in National Capital Territory

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of Delhi, so we carried out a retrospective study to investigate the demographic profile, treatment delay, clinical features, medical treatment, complications and mortality of patients with snake bite admitted to the intensive care unit of our hospital.

2. Materials and methods

2.1. Ethic approval and objectives

This retrospective study was carried out at a teaching hospital in the National Capital Territory of Delhi, India, after the approval of the Institutional Ethical Committee (No./HRH/2018/8364 dated 29/11/18). This Institute is a tertiary care hospital catering to the population residing in North Delhi and adjoining areas of Haryana and Uttar Pradesh. We obtained records of patients admitted in our intensive care unit from January 2013 to October 2018.

2.2. Inclusion criteria and exclusion criteria

Patients meeting following criteria were included in our study:

- (1) Definitive history of snake bite with or without presence of fang marks;
- (2) The presence of acute onset neuroparalytic symptoms with a history of snake bite or presence of fang marks;
- (3) The presence of local signs like swelling, inflammation, bleeding from the site with a history of snake bite or presence of fang marks.

Patients with non venomous snake bites and ambiguous history were excluded from the study. Patients who didn't exhibit signs and symptoms of snake bite after a period of observation of 24 h were labeled as non venomous. Clinical envenomation was defined by presence of signs and symptoms of local or systemic toxicity. Local signs were the presence of fang marks, bleeding, swelling or necrosis. Systemic toxicity was defined as presence of neuroparalytic features or hemostatic abnormalities.

2.3. Data collection

The data was collected from medical record department of our hospital. Data was collected and entered in Excel sheet. The demographic profile, time of bite, site of bite, month of bite, clinical features, time interval elapsed between the bite and medical treatment including administration of anti-snake venom (ASV), the dosage of ASV, adverse reactions to ASV, administration of anticholinesterase, complications and clinical outcomes were recorded. Laboratory results of hemoglobin, total leucocyte count, platelet count, serum creatinine, blood urea, serum alanine aminotransferase and aspartate aminotransferase, alkaline phosphatase, arterial blood gas of patients with respiratory difficulty were recorded. Urine routine microscopy, prothrombin time, International Normalized Ratio and 20 min whole blood clotting test and electrocardiography were recorded. We also recorded the complications, survival and mortality.

Table 1. Different clinical manifestations of patients.

Clinical feature	Number of patients	Percentage of patients (%)
Ptosis	52	50.9
Double vision	46	44.9
Difficulty in breathing	29	28.9
Difficulty in deglutition	22	21.7
Local signs	32	31.8
Muscle weakness	23	23.1
Slurring of speech	6	5.7
Aphasia	9	8.6
Epistaxis	3	2.8

2.4. Diagnosis

Neurotoxicity was defined as presence of ptosis, ophthalmoplegia, weakness of the muscles of extremities, difficulty in breathing and inability to lift the head. Hemolytic toxicity was defined as deranged 20 min whole blood clotting test (WBCT) or the presence of spontaneous visible bleeding signs. Hypotension was defined as systolic blood pressure lower than 90 mm Hg. Oliguria was defined as urine output lower than 0.5 mL/kg/h. Serum fibrinogen levels, fibrin degradation products and D dimers levels were not uniformly available in our hospital during the period of our retrospective study. Disseminated intravascular coagulation was therefore defined by a deranged 20 min WBCT, deranged prothrombin time and International Normalized Ratio.

3. Results

A total of 102 patients were selected, in which 56 were male (54.9%) and 46 were female (45.1%). A total of 39.2% (40/102) patients were 21-30 years, 27.4% (28/102) were 11-20 years patients, and 15.6% (16/102) patients were 31-40 years. A total of 35.3% patients were referred from peripheral health centres and private hospitals and clinics, while 64.7% were admitted directly to the emergency department of our hospital.

Majority of the bites occurred in the monsoon season with June (19.6%), July (31.3%) and August (15.6%) and more snake bites incidence occurred in young patients. Most patients were bitten on the lower limb (56%) followed by the upper limb (36%) and majority of the bites occurred in the night and early morning time

A total of 31.8% (32/102) patients had local signs at the site of the bite. The neuroparalytic symptoms were predominant (Table 1). Epistaxis along with neuroparalytic symptoms was seen in 3 patients. The most common clinical manifestation was ptosis followed by double vision and difficulty in breathing.

The mean hemoglobin concentration was (9.9±2.3) g/dL, white blood cell count was 12 700±6 100 and platelet count was 128 000±45 000. The mean serum urea was (42±6) mg/dL and serum creatinine was (1.3±0.3) mg/dL. The mean serum albumin concentration was (3.2±0.4) g/dL. The mean serum bilirubin

concentration was (0.9 ± 0.4) mg/dL, serum aspartate transaminase was (45 ± 3.7) IU/L, serum alanine transaminase was (46 ± 2.6) IU/L, alkaline phosphatase was (102 ± 17.8) IU/L. Hemoglobinuria was seen in 3 patients.

Twenty-nine patients developed difficulty in breathing, out of which 26 required ventilatory support. The mean ventilator days were 3.8 d. Six patients required inotropic support during the course of ICU stay. Five patients developed oliguria and 3 progressed to acute renal failure requiring dialysis. Three patients required platelet transfusion and 2 required fresh frozen plasma transfusion in the intensive care unit. The mortality was 11.7% (12 patients). Out of these 12 patients, 5 died due to acute respiratory distress syndrome, 4 due to ventilator associated pneumonia and 3 due to acute renal failure. Three patients who were referred from private hospitals, and were admitted in our hospital 6 h after the snake bite and hence were administered ASV after 6 h, out of whom 2 patients deteriorated further in the ICU and died. ASV was administered to 55 patients (53.9%). The average dose of ASV administered was 42 vials per patient. Majority of the patients received ASV within 3 h after the snake bite. No patient developed any adverse reaction to ASV administration.

4. Discussion

We evaluated the epidemiology of snake bite envenomation, clinical features, treatment and complications of patients admitted to the intensive care unit of a tertiary care hospital in North Delhi. Majority of patients were 21-30 years (39.2%). Patients in this age group belonged to the working class, and are mainly involved in outdoor activities.

Snake bite is a common life threatening medical emergency in the Indian subcontinent. In India the most common venomous snakes is the elapid family (cobra, krait) and Viper family (Russell Viper, Saw scaled Viper). Snake bite poisoning caused by the cobra and krait predominantly affects the neurological system. The clinical manifestation of cobra and krait bite include paralysis of the ocular, bulbar and limb girdle muscles. It is reported that ptosis is the commonest neurological manifestation in 85.7% of the cases as reported by Seneviratne and Dissanayake, followed by ophthalmoplegia (75%), limb paresis (26.8%), respiratory failure (17.9%), palatal weakness (10.7%) and weakness of neck muscles (7.1%)[5].

Most cases admitted to our institution presented with early morning neuroparalytic features, and ptosis was presented in majority of the patients (50.9%) followed by ophthalmoplegia (44.9%), difficulty in breathing (28.9%) and muscle weakness (23.1%). There was no definitive history of pain in patients with neuroparalytic features. So predominance of neurotoxic features combined with painless bites in our study suggested predominance of krait envenomation in this part of North Delhi. Sharma *et al.* also reported increased incidence of neurotoxic bites in North India with predominance of Krait[6].

Similarly, Chauhan *et al.* reported increased incidence of

neuroparalytic envenomation in North India and haematotoxic envenomation in South India[7]. The hemolytic manifestation of snake envenomation are common in bites caused by the Viper family. These manifestations are persistent oozing from the bite mark, epistaxis, haematemesis, hemoptysis and cutaneous ecchymoses. Mehta *et al.* reported subconjunctival, retroperitoneal and intracranial haemorrhage following snake bite[8]. However, in our study 3 patients presented with epistaxis along with neuroparalytic features and these 3 patients progressed to acute renal failure requiring dialysis.

A total of 56% patients in our study were bitten on the lower limbs, 36% on the upper limbs and 7% had axial bites. Sharma *et al.*[9] reported 38% bites on the lower limbs, 47% bites in the upper limbs and 14% axial bites.

In our study, most patients were from poor socioeconomic status with habits of sleeping on the floor, which could have predisposed them to the snake bites. A total of 53.9% of the snake bites in our study occurred during the night and early morning. Sharma *et al.* have reported an increased incidence of snake bites while the patients were sleeping on the floor (92.5%)[6]. Krait bites are more common during the night where as cobra and viper bites are more common in the day time[10], and similar observation in our study reinforces the fact that krait was the predominant cause of snake envenomation.

The study by Sharma *et al.*[6] reported increased incidence of snake bites during the monsoon period with maximum number of cases seen in July. Another study from the Indian subcontinent also showed higher incidence of snake bites during the monsoons[10]. We also observed similar trend with increased incidence in the monsoon season. In our study, 31% patients had received tetanus toxoid before presentation in our hospital and 23% had a tourniquet in situ. A total of 19% patients in our study had been administered with ASV prior to presentation in our hospital and had been referred to a tertiary care hospital for further treatment. Kalantri *et al.* had reported ASV administration in 18% patients as a first aid measure and tourniquet use in 37% of patients[11].

ASV is definitive treatment of snake envenomation, and intensive care in the form of ventilatory support might be required for patients who develop severe neuroparalytic features. In our study, 29 patients developed difficulty in breathing, out of which 26 required ventilatory support with a mean of 3.8 ventilation days. Three patients required platelet transfusion and 2 required fresh frozen plasma transfusion in the ICU. Five patients developed oliguria and 3 patients progressed to acute renal failure requiring dialysis treatment. We noted a mortality of 11.7% (12 patients) in the study. Out of these 12 patients, 5 died due to acute respiratory distress syndrome and 4 due to ventilator associated pneumonia and 3 due to acute renal failure.

We observed a coexistent hemoglobinuria in patients with acute renal failure. Neuroparalytic symptoms mixed with hemolytic features was seen in these patients. Krait envenomation can present as neuroparalysis with dark brown urine and acute kidney injury (Syndrome 5)[12].

Most patients with clinical manifestations were administered ASV within 3 h after the snake bite (31/102). There was no case fatality in these patients as they were administered anti-snake venom early. The short time interval of ASV administration may be attributed to maximum number of cases presenting from nearby catchment areas. Increased complications and mortality was seen in patients who presented after three hours of snake bite and received anti-snake venom late.

ASV was administered to 55 patients with clinical manifestations (53.9%). The toxin present in cobra (cobratoxin) and krait venom (α bungarotoxin) binds to the acetylcholine receptors on the post synaptic motor end plate^[13]. Anti-snake venom acts by neutralizing the effects of snake venom in the circulation. ASV should be administered as early as possible in symptomatic envenomation as snake venom which has been already bound to the target receptors is not neutralized by the effects of anti-snake venom. The average dose of ASV in our study was 42 vials per patient. We used initial high doses of ASV in our institution in the hope to neutralize the snake venom in the circulation before it gets bound to the acetylcholine receptors.

It has been reported that neuroparalytic snake envenomation requires higher doses of ASV as compared to hemotoxic envenomation^[14]. Raina *et al.* used a mean dose of (292.6±196.27) mL (range 50-950 mL) in the management of snake envenomation^[15]. Agarwal *et al.* have used high doses of ASV with median dose of ASV 90 vials (40-140 vials) for neuroparalytic snake bites^[16]. However, recent trials^[17-19] have concluded that low dose ASV is equally efficacious as high dose ASV in the management of snake envenomation. As ASV is a costly drug, the cost effectiveness should be kept in mind especially in a developing country like India. Further trials are needed to ascertain the optimum dose of ASV in patients of snake bite. The WHO guidelines recommend an initial trial with anticholinesterases for patients of neurotoxic envenomation. The trial would continue if patients show improvement in the neurotoxic symptoms. Anticholinesterases can improve symptoms of patients with bite from cobra envenomation and krait (α bungarotoxin), which act post synaptically. However, the predominant action of krait venom is presynaptically (β bungarotoxin)^[20] and prevents the release of acetylcholine at the neuromuscular junction. Therefore, treatment with anticholinesterase drugs might not improve symptoms in some patients of krait envenomation. As most patients in our study presented with neurotoxic symptoms, we administered neostigmine 0.5 mg twice hourly to all the patients and observed them closely for clinical improvement. Neostigmine was discontinued in 16 patients who did not show improvement in signs and symptoms.

As neuroparalytic symptoms are predominant in our study, we presumed the bites to be cobra and krait. Majority of the patients in our study had history of sleeping on the floor and presented with early morning neuroparalytic features and painless bites. We presumed that majority of the bites were caused by the krait. However, we could not confirm as many patients did not give a positive history of seeing the snake or give a proper description of

the snake nor did we have provision of snake venom detection kits in our hospital which can confirm the presence of venom in the blood or identify the species of the snake. Special investigations like D dimers, fibrin degradation products and serum fibrinogen levels were not done in all the patients during the retrospective period.

Another limitation of our study is that a large number of patients with snake bite are treated through traditional healing methods in India and might not turn to the tertiary care centres. This might lead to an unwanted bias in our study as it might not reflect the true incidence in the community.

To conclude, there is an increased prevalence of krait envenomation in catchment areas of North Delhi and is commonly seen in young adults in the monsoon season. There is a need to educate the masses and healthcare providers about the identification of early morning neuroparalytic symptoms and treatment with ASV administration to avoid fatal complications.

Conflict of interest statement

The authors report no conflict of interest.

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