Management of snake-bite in rural Maharashtra: A 10-year experience

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ABSTRACT

Background. *A high incidence of snake-bite envenomation has been reported from Marathwada, Maharashtra. This study analysed the pattern of snake-bites and their management in a rural area of India over a 10-year period.*

Methods. *A total of 633 patients with snake-bite admitted to the Rural Community Centre and Punde Hospital in Mukhed taluka, Nanded district (Marathwada) of Maharashtra, between 1992 and 2001, were analysed retrospectively. The local and systemic manifestations of snake-bite, response to antisnake venom, atropine and neostigmine, the treatment of complications and the outcome were analysed.*

Results. *Of the 633 patients, 427 (67.5%) had been bitten by poisonous snakes and 206 (32.5%) by non-poisonous snakes. The majority of snake-bites (68.9%) occurred between May and November. Those affected were mainly farmers (228 [36%]), students (191 [30.2%]) and housewives (175 [27.6%]). Of the 427 envenomed by poisonous snakes, 274 (64.2%) were by Echis carinatus (saw-scaled viper), 71 (16.6%) by cobra, 42 (9.8%) by krait and 40 (9.4%) by Russell viper. The requirement of antisnake venom for treating neurotoxic envenomation was 40–320 ml and for Echis carinatus and Russell viper bites it was 20–250 ml. Among those envenomed by poisonous snakes, the mortality was 4.7% (n=20).*

Conclusion. *Snake-bite is a common life-threatening emergency in the study area. We observed an occupational risk and a seasonal incidence of snake-bite. Knowledge of the varied clinical manifestations of snake-bite are important for effective management. Ready availability and appropriate use of antisnake venom, close monitoring of patients, institution of ventilatory support and early referral to a larger hospital when required help in reducing the mortality. Most patients with snake-bites can be successfully managed even in small rural hospitals with limited facilities.*

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INTRODUCTION

Snake-bite is a life-threatening medical emergency. It occurs frequently among rural people, especially those working in the fields. The Deccan plateau with its plain agricultural land and hot and dry climate provides an ideal environment for cobras, kraits and vipers.[1] Most houses in rural areas of India are made of mud and have many crevices where rodents flourish. Snakes have easy access to the interiors of such houses and often enter them in search of food.[2] Firewood and dried cowdung, stored in or near the house, provide easy shelter for snakes and rodents.[2,3]

In India, an estimated 35 000–50 000 lives are lost per year due to snake-bite.[4] Though every year about 2000 deaths occur due to snake-bite in Maharashtra, the majority go unreported because many villagers go to traditional healers.[5] Delay in seeking medical aid or ignorance among primary care physicians about the correct treatment of snake-bite is responsible for the high morbidity and mortality.[2] This analysis aims to ascertain the presentation and outcome of patients with snake-bite in a rural setting.

METHODS

This retrospective analysis was based on patients treated at the Rural Community Centre and Punde Hospital, Mukhed in the Nanded district of Maharashtra, India. The data from 1992 to early 1997 is of patients treated at the Rural Community Centre, Mukhed which had facilities for admitting up to 30 patients and was staffed by 2 medical officers and 3 staff nurses. It had emergency services and a small laboratory for investigations but neither ventilators nor ambulances were available.

The data from mid-1997 to 2001 is of patients treated at the Punde Hospital, Mukhed where patients were managed by the author along with a resident medical officer. This is a 12-bedded hospital equipped with an electrocardiogram machine, a Newmon ventilator, a pulse oximeter, an oxygen concentrator and an electricity generator. Blood-banking facilities are not available at Mukhed. Patients requiring blood transfusion were sent to the district hospital at Nanded (a larger referral facility), a distance of 81 km. Since no ambulances were available, patients were transported to Nanded in private vehicles. The resident medical officer or a trained paramedical person, equipped with emergency drugs, accompanied the referred patients.

The diagnosis of snake-bite was established on the basis of a history of snake-bite with examination of the killed snake in 45 cases and, in the remaining, by correlating the clinical manifestations and recognition of the snakes by patients and bystanders. Bites due to cobras and kraits were classified as neurotoxic bites and those due to saw-scaled viper (*Echis carinatus*) and Russell viper as vasculotoxic bites.

Clinical data including age, sex and occupation of the victims, the site of bite, time of bite, time between bite and presentation, clinical manifestations, complications and outcome were obtained from the case records and entered in a computer database for analysis.

From 1992 to 2001, 633 patients of snake-bite were seen. Of these, 427 (67.5%) had signs of envenomation.

The patients were divided into 3 groups based on their clinical manifestations. These were:

1. No symptoms or signs of envenomation

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2. Presence of local symptoms and signs only
3. Presence of local symptoms and signs along with systemic manifestations of envenomation.

All patients were given tetanus toxoid and non-steroidal anti-inflammatory drugs (NSAIDs).

The antisnake venom (ASV) was obtained from the Haffkine Institute, Mumbai. It was administered by the intravenous route. Prior to administering ASV, a test dose (1 ml of 1:100 diluted ASV) was given subcutaneously and the patient observed for any reaction. ASV was diluted in 200 ml of normal saline in cases of cobra, krait and saw-scaled viper bites, and in 200 ml of 5% dextrose or dextrose–normal saline in cases of Russell viper bite. The initial dose was given over half an hour. Following the resolution of neurological symptoms and signs (in case of neurotoxic bites) or normalization of the whole blood clotting time (in case of vasculotoxic bites), a maintenance drip of ASV was given in 500 ml of intravenous fluids for a further period of 24 hours. The treatment given to each of these groups is detailed below.

No symptoms or signs of envenomation

An intravenous drip was started in all patients to maintain intravenous access. All patients were observed for the development of symptoms and signs of envenomation for 24 hours. If they were asymptomatic at the end of this period of observation, they were discharged. Oral cephalosporins were given for a period of 4 days.

Presence of local symptoms and signs only

All patients were admitted and the tourniquets, if applied, were removed after starting intravenous fluids and administering 20–40 ml of polyvalent ASV.

Patients who had (i) progressive oedema involving more than one joint, or (ii) recurrent bleeding from the site of the bite or evidence of cellulitis, or (iii) systemic signs of envenomation appearing later, were given 50 ml of ASV every 6 hours up to a maximum dose of 240 ml. Oral cephalosporins were given for 7 days.

Presence of local symptoms and signs with systemic manifestations of envenomation

All patients were admitted and whole blood clotting time was done. Patients with Russell viper bite (a diagnosis of Russell viper bite was made in those with massive oedema at the site of the bite, continuous bleeding from the fang marks, lymphadenopathy and rapid development of haemorrhagic disorder), those with hypotension or impending renal failure (urine output <1 ml/kg/hour for 3 consecutive hours in spite of adequate hydration) and those in whom the bleeding did not stop within 30 minutes were given an initial dose of 100 ml of ASV over 30 minutes. If external bleeding was still present an additional 50 ml of ASV was given every 2 hours till the bleeding stopped. Further administration of ASV was guided by the whole blood clotting time, the target for which was <20 minutes. If there was no external bleeding after the first dose of ASV, further doses (20 ml every 6 hours) were given till the whole blood clotting time was <20 minutes. Shock due to envenomation was treated with intravenous fluids and dopamine.

Patients with neurotoxic envenomation were managed with ASV, atropine, neostigmine and were ventilated if respiratory failure developed.

In patients with suspected cobra bite, 100 ml of ASV in 200 ml of normal saline was given within one hour of admission, 40 ml over the next two hours and 20 ml over the next 24 hours. In the absence of neuroparalysis, 40–60 ml of ASV was given initially followed by further doses guided by the clinical features.

Patients with bradycardia, giddiness or hypotension without hypovolaemia (based on clinical evaluation) were given 1.2 mg of atropine every 10 minutes. If the heart rate did not improve more than 20% with this dose of atropine they were considered to be non-responsive and were treated with oral orciprenaline 5 mg thrice daily for 7 days.

Intravenous third-generation cephalosporins were given to all patients in this group for 2–3 days and oral cephalosporins were continued for 2 weeks. Metronidazole was added in patients with sepsis or non-healing ulcers.

Referral policy

Patients with the following conditions were referred to a larger hospital:

1. Excessive or internal bleeding with shock in spite of having received an optimum dose of ASV
2. Shock not responding to inotropes
3. Impending acute renal failure (reduction in urine output to <20 ml per hour)
4. Low oxygen saturation (<90%) despite mechanical ventilation.

RESULTS

Saw-scaled viper bites were seen throughout the year while cobra bites were common in the months of March, April, July, September and October. Krait bite cases were reported mostly between June and October. The majority of bites (68.9%) occurred between May and November.

Over a 10-year period (1992–2001), 633 patients bitten by snakes were seen. There were 402 men (63.5%) and 231 women (36.5%). Bites were more frequent in younger individuals; 525 were <40 years of age (82.9%) and 218 were <20 years (34.4%).

Patients with no signs and symptoms of envenomation

There were 206 patients (129 men and 77 women) with a history of snake-bite but no evidence of envenomation. Most bites occurred in the fields (54.8%) or at home (41.7%). Seventy-nine patients reported within 60 minutes of the bite, 12 patients between 60 and 90 minutes, 43 patients within 120 minutes and 172 patients reported from 120 minutes to 4 days after the bite.

These patients were anxious and agitated, had mild tachycardia, single or multiple local bite marks or abrasions. There was no local oedema, regional lymph node involvement or systemic signs. None of the patients had brought a killed snake. There were no deaths in this group.

Snake bites with envenomation

Local oedema. A total of 274 patients (178 men and 96 women) presented with local oedema without systemic signs or bleeding. More than half the patients (56.2%) were bitten in the fields and 42.7% at home. Sixty-two patients received first aid (tourniquet and inadequate doses of ASV) while 212 did not. Ninety-seven patients reported within 60 minutes, 73 from 60 to 120 minutes, 63 patients presented from 6 to 24 hours, 40 patients from 24 to 96 hours and 1 patient reported after 8 days with gangrene of the left leg.

Ten patients brought dead snakes that were identified as saw-scaled viper. Two patients had a dry bite (confirmed bite by poisonous snake without envenomation). Two hundred and sixty-

four patients who had a history of snake-bite did not bring the snake but had symptoms and signs similar to those of saw-scaled viper envenomation. Six patients were in shock and were treated with intravenous fluids and dopamine. Two patients had severe bradycardia not responding to atropine and hence were treated with orciprenaline.

The mean duration of hospital stay in these patients was 3 days (range: 2–7 days). The patient with gangrene of the left leg was referred to a larger hospital where an above-knee amputation was done and the patient was given an artificial limb. There were no deaths in this group and all except 1 patient recovered completely.

Local oedema with bleeding. Forty patients (30 men and 10 women) were admitted with a history of snake-bite and rapidly progressive oedema and bleeding diathesis. Fourteen of them had received first aid while 26 had not. Twelve patients reported within 60 minutes, 3 patients from 60 to 90 minutes, 6 patients from 90 to 120 minutes, 11 patients from 2 hours to 6 hours, 3 from 6 hours to 24 hours and 5 patients reported from 24 hours to 15 days later. Ten patients had brought the killed snake which was identified as Russell viper. The remaining 30 patients who did not bring the snake had similar clinical features. They were treated as having Russell viper bites.

Clinical symptoms and signs observed in this group of patients were pain in the abdomen (17), vomiting (20) and bleeding diatheses (18) (such as haemoptysis [11], haematuria, and local and gum bleeding). Fourteen patients were in shock, 1 had ptosis in addition to bleeding and 7 patients had bradycardia.

Of the 40 patients, 20 were referred to a larger hospital. Of these, 12 patients had developed acute renal failure (3 each after 24, 48 and 72 hours, and 1 each after 4, 10 and 15 days). High doses of diuretics were given to patients with acute renal failure, 2 of these patients underwent haemodialysis at larger hospitals. Six patients developed a non-healing ulcer.

Three patients (7.5%) died—1 due to acute renal failure, 1 due to refractory shock and 1 due to disseminated intravascular coagulation.

Neurotoxic envenomation. One hundred and thirteen patients were admitted with neurotoxic envenomation. Of these, 71 had a cobra bite and 42 a krait bite.

Cobra bite. Out of 71 patients, 10 had brought the killed cobra and 61 patients had similar clinical features. Out of the 71 bites, 42 (59.2%) occurred at home, 22 (31%) in the fields, 4 (5.6%) on the road, 1 (1.4%) in a shop and 2 (2.8%) in a temple. In 43 patients a tourniquet had been applied. Nine patients had been treated by quacks. Forty patients reported within 1 hour of the snake-bite, 17 reported between 1 and 2 hours, 13 between 2 and 6 hours and 1 patient after 6 hours. Neurological manifestations included bulbar paresis in 65 patients, respiratory paralysis in 36 and loss of consciousness in 28. All patients were vigorously treated and monitored closely. The amount of ASV infused varied from 40 to 320 ml (mean 156 ml). Patients were given neostigmine 50 mg per kg; atropine was given as required. A maximum of 6 doses of neostigmine, given at half-hourly intervals, were required for recovery from neuroparesis. Patients with respiratory paralysis were intubated immediately and ventilated with either an Ambu bag (26 patients) or a ventilator (8 patients). Two patients were brought dead to the hospital.

In 50 patients, the neuroparesis reversed completely within 8 hours. In 8 patients, reversal took longer. One child again developed respiratory arrest 5.5 hours after initial complete recovery. This could be because of an inadequate dose of ASV as compared to the body surface area or a sudden release of poison from local blebs. The patient recovered completely after further ventilation and administration of ASV. A total of 320 ml of ASV was infused in this patient.

In this group 13 patients died.

Krait bites. Forty-two patients with krait bites were admitted. Fifteen of these had brought a killed krait. The remaining 27 had features similar to the others and were treated as having krait bites. Two patients had dry bites (bites by a poisonous snake without injection of venom). Out of 42, 31bites (73.8%) occurred at home, 6 (14.3%) in the fields, 1 each (2.4%) in a classroom and on the road, and in 3 (7.1%) there was no history of a bite though clinical features of krait bite were present.

Six patients reported within 1 hour of the snake-bite, 5 between 1 and 2 hours, 15 between 2 and 6 hours and 4 between 6 and 16 hours. In 12 cases the time since the snake-bite was not known. One patient had krait bite on the nape of the neck and developed neuroparesis after 16 hours.

Fifteen patients received first aid and 14 others were treated by quacks. Thirty-five patients (83.3%) presented with pain in the abdomen and 8 had vomiting. One patient who had chest pain and sweating was referred with a suspicion of acute myocardial infarction. There was no history of a snake-bite. This patient subsequently developed neuroparesis. Neurological manifestations included bulbar paresis in 34 patients, respiratory paralysis in 13 and loss of consciousness in 11.

All patients with respiratory paralysis were intubated and ventilated with either an Ambu bag (8) or a ventilator (5). ASV was infused in a dose of 40–250 ml (mean 154 ml). Neostigmine, 50 mg/kg in half-hourly doses, and atropine (as clinically required) were administered. Usually 3 doses of atropine and 6 doses of neostigmine were needed. Neostigmine did not produce any effect on the neuroparesis and further doses of this drug were omitted.

Three patients (7.1%) in this group died at 31, 72 and 120 hours. One patient died while being taken to a larger hospital.

Cobra versus krait bites

Patients with cobra bite presented with early neuroparalysis and rapid onset of respiratory failure (15 minutes to 7 hours, mean 96.4 minutes). Recovery in these patients was also fast (1–24 hours, mean 4.7 hours).

Patients with krait bite presented with late neuroparalysis and delayed onset of respiratory failure (30 minutes to 16 hours, mean 246.5 minutes). They did not show marked improvement with neostigmine and atropine. Recovery of neuroparalysis and respiratory failure in krait bite was delayed (4–72 hours, mean 22.9 hours). Respiratory paralysis lasted less than 12 hours in 3 patients, between 12 and 24 hours in 4 patients and 36, 42 and 43 hours in 1 patient each.

Complications of snake-bite

Table I lists the complications of snake-bite observed in this study. Respiratory paralysis was the commonest acute complication. It was seen in 50.7% of patients bitten by a cobra and in 31% of those bitten by a krait. Following a Russell viper bite, shock developed in 35% of patients, bradycardia in 17.5% and acute renal failure in 30%. Anaphylaxis due to ASV occurred in 11.7% of the patients to whom ASV was administered. The commonest delayed complication was a non-healing ulcer at the site of the bite. This was seen in 35.2% of the victims of cobra bite and in 15% of those bitten by a Russell viper.

TABLE I. Complications of snake-bite

Complication	Type of snake-bite		Total
	Neurotoxic (*n*=113)	Vasculotoxic (*n*=314)	
Acute			
Respiratory paralysis	49	0	49
Cardiac complications			
Shock	2	20	22
Bradycardia	3	9	12
Pulmonary oedema	3	0	3
Bleeding diathesis	0	18	18
Acute renal failure	0	12	12
Gangrene	0	1	1
Subcutaneous emphysema	1	0	1
Therapy-related			
Antisnake venom anaphylaxis	11	39	50
Severe	8	28	36
Delayed			
Non-healing ulcer	25	6	31
Contracture	2	0	2
Vocal cord adhesions	1	0	1

Referral

Table II shows the number of patients from each group referred to a larger hospital for management. Those with Russell viper bite had the highest rate of referral due to a high rate of acute renal failure and bleeding diatheses in this group.

Mortality

Mortality was highest due to cobra bites (18.3%) followed by krait bites (9.5%) and Russell viper bites (7.5%). The site of bite was the upper limb in 9, the lower limb in 10 and the nape of the neck in 1 patient. The highest mortality was in the 0–10 years' age group (10/25, 40%) followed by the 11–20 years (4/23, 17.4%) and then the group of patients >20 years of age (6/65, 9.2%).

None of these patients had received primary care and the bite to hospitalization time interval was within 30 minutes in 4, 30–60 minutes in 6, 1–2 hours in 4, 2–4 hours in 3 and >4 hours in one. In two cases the time of the snake-bite was not known. Seventeen patients with neurotoxic envenomation died due to respiratory failure. Of the 3 patients with bleeding diathesis, 1 each died of acute renal failure, refractory shock and disseminated intravascular coagulation. Two patients arrived dead at the hospital, 10 died in the hospital, 1 patient died on the way to the referral centre and 7 patients died after being treated at the referral centre.

DISCUSSION

Farming is the main occupation in the study area. Farmers walk barefoot while ploughing their fields, which makes them prone to snake-bites. Their houses, built of mud, provide easy access and shelter to snakes.[2,5] Some students in rural areas walk barefoot to school and are bitten by snakes. Snake-bites can also occur in shops while handling old storage material where snakes may have taken shelter.

TABLE II. Patients referred to a larger hospital

Type of snake-bite	Total patients	Referred patients (%)
Cobra	71	10 (14.1)
Krait	42	8 (19)
Russell viper	40	20 (50)
Saw-scaled viper	274	8 (2.9)

Snakes usually bite the distal parts of the body.[6] The maximum number of cases was in the 11–40 years' age group (82.93%). Individuals in this age group are actively involved in farming and handling farm debris.

Complications of snake-bite most frequently encountered in this study were respiratory paralysis, shock, bleeding diatheses and acute renal failure. Anaphylaxis due to ASV occurred in 11.7% of those who received ASV, a finding similar to that reported by others.[7]

The antihaemostatic effects of venom explain the haemorrhagic manifestations of saw-scaled and Russell viper bites. However, haemorrhagic manifestations due to saw-scaled viper bites were not common in this study. This can be explained by the fact that the severity of envenomation by a particular species can vary from place to place.

Acute renal failure in Russell viper bites is due to toxic nephropathy[8,9] and prolonged hypotension due to delay in seeking treatment.[2]

Krait venom contains both postsynaptic neurotoxins and toxic phospholipase A2 acting presynaptically.[10–12] ASV does not neutralize toxins attached to the presynaptic receptors. The presynaptic blockade produced by krait β-bungarotoxin is resistant to anticholinesterase.[7,11,12] Krait-bite victims thus need prolonged ventilatory support.

Appropriate first aid is of vital importance for snake-bite victims. Many measures which were advocated earlier have now been abandoned as being harmful. These include the tying of tight (arterial) tourniquets, incising and bleeding the wound, sucking the wound to remove the venom from it and the local application of ice.[13]

Recommended first-aid measures[14] include the use of a wide, flat constriction band applied proximal to the bite to block only superficial venous and lymphatic flow. This should be left in place until antivenom therapy, if indicated, is begun. The band should be loose enough to permit one or two fingers to slide easily beneath it. Excess constriction could impair arterial blood flow and lead to tissue death. Excess activity, such as walking, should be avoided. The bitten extremity should be immobilized and kept in a dependent position. The victim should be rapidly transported to the nearest hospital.

Once the patient is brought to the hospital, a high index of suspicion and knowledge of the manifestations of snake-bite is essential for early and accurate diagnosis. For example, neurotoxic snake-bite must be excluded in a patient presenting early in the morning with chest pain in whom the electrocardiogram is normal and mild ptosis is present.

Doctors working in peripheral areas should be trained in diagnosing poisonous snake-bites and in managing victims with ASV, Ambu bags and, where available, ventilators. Even in the absence of symptoms, all patients should be admitted and observed for at least 24 hours for signs of envenomation. Timely institution of ventilatory support and its continuation till complete recovery from respiratory paralysis are critical in patients with neurotoxic envenomation.

The requirement of expensive ASV was high in krait, cobra and Russell viper bites. This may be due to the fact that the geographical differences in the venom from the same species necessitate the use of snakes from the same region for preparing ASV. The setting up of regional ASV banks would address this problem. Making ASV readily available in peripheral hospitals and providing it free of cost to patients would also help save lives.

The local population should be educated in ways to protect themselves from snakes and rapidly seek treatment in case of a

bite. Farmers should be educated about the use of torches, sticks, footwear and appropriate clothing to protect themselves. Education would also help to dispel myths associated with snake-bite. Regular repair and cleaning of mud houses would prevent rats and snakes from entering them.

Provision of facilities for rapid transportation to the hospital and for subsequent referral (if required) would reduce the time from snake-bite to treatment and is essential for reducing mortality.

In this study, mortality following envenomation was 4.7%. Only 46 patients (10.8%) needed referral to a larger hospital for management. This experience indicates that the majority of patients with snake-bite can be successfully managed in a rural hospital with limited facilities. Many lives can be saved with appropriate administration of ASV and timely use of the Ambu bag, a simple device available in most hospitals.

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Snake bite is exclusively accidental in nature. Suicide by snake bite is very rare and homicidal snake bite is not reported. In the present case, a contract killer was hired, who used a poisonous snake to kill an elderly couple by way of direct snake bite. We believe this to be the rst case reported where a snake was directly used for the murder of two victims through a contract killer. Med Sci Law 2012; 52: 40â€“43. DOI: 10.1258/msl.2011.011020. Introduction. Snake bite is an important and serious problem in many parts of the world, especially in South Asian countries.Â Management of snake bite in rural Maharashtra: a 10-year. experience. Nat Med J India 2005;18:71 â€“ 5 7 Auerbach PS, Norris RL. Disorder caused by reptile bites and marine. Profile of snakebite envenoming in western Maharashtra, India. Trans R Soc Trop Med Hyg. 2002;96:79-84. 9. Sankar J, Nabeel R, Priyambada L, Mahadevan S . Factors affecting outcome in children with snake envenomation: A prospective observational study. Arch Dis Child. 2013;98:596-601. 10. Punde DP. Management of snake-bite in rural Maharashtra: A 10-year experience. Natl Med J India. 2005;18:71-5. Record based study of snake bite cases admitted at shri vasantrao naik government medical college and hospital, Yavatmal (Maharashtra). Indian J Public Health. 2006;50(1):35-7. David S, Matathia S, Christopher S. Mortality predictors of snake bite envenomation in southern India a ten-year retrospective audit of 533 patients. J Med Toxicol. 2012;8(2):118-23.Â Management of snakebite in rural Maharashtra: A 10-year experience. Nat Med J Ind. 2005;18(2):71. Agrawal PN, Aggarwal AN, Gupta D, Behera D, Prabhakar S, Jindal SK. Management of respiratory failure in severe neuroparalytic snake envenomation. Neurol Ind. 2001;49(1):25-8. Management of snake-bite in rural Maharashtra: a 10-year experience. National Medical Journal of India, (Vol. 18) (No. 2) 71-75.Â Profile of snakebite envenoming in Western Maharashtra, India. Trans R Soc Trop Med Hyg. 2002;96(1):79-84 [19].