Research Article

A study of systemic envenomation from various types of poisonous snake bites and their effective management to reduce mortality

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Abstract

Introduction: Snake bite is an important occupational and rural hazard because India has always been a land of exotic snakes. All snakes are commonly considered by layman to be poisonous but according to the classical work of Melcon smith in 'fauna of British india-1943' about 216 species belonging to 8 families are poisonous. In Maharashtra, common poisonous snakes are cobra, Russell's viper, Saw Scaled Viper and Krait. It is very difficult to give a definite statistical record of the snake bite mortality of the cases take places in the periphery, which are hardly accessible to the big hospitals. Then too, with urbanization and cutting down of forests, snake bite has become a very important preventable public hazard. **Aims and Objectives:** To study the systemic envenomation from various types of poisonous snakes and their effective management in reducing the mortality rate. **Methodology:** This was a hospital Record based study of various types snake bite patients admitted to Krishna Institute of Medical Sciences University Karad, this was carried out in one year duration. Data was collected using semi-structured proforma. Data is Presented in the forms Percentages and Proportions. **Result:** Ptosis was most common presentation of Neuro-Paralytic Bite Poisoning followed by Respiratory Paralysis, Opthalmoplegia, Dysphagia, Cellulitis, Flaccid Limb Paralysis, Unconsciousness. Local Bleed was the most common Symptoms presented with Viperine bite followed by Cellulitis, Hematemesis, Oliguria, Hematuria, Gum bleed, Epistaxis, Subconjuctival Hemorrhage, IntracerebralHemorrhage.ARF was the most common complication followed by DIC , Surgical intervention and Death. Most of the patients required ASV followed by Diuretics, Hemodialysis, and Blood Transfusion. Maximum patients of Neuroparalytic snake bite patients required Artificial Ventilator, followed by Intubation, Not required any device. Completely recovery was more common in Neuroparalytic Bite, followed by Locally Toxic Bite and Viperine Bite. Death Mostly Observed in Viperine Bite followed by Neuroparalytic Bite **Conclusion:** The symptomatology and complications varies with neuroparalytic and Viperine and Localy toxic snake so adequate treatment specific to the verity should be provided.

Keywords: Neuroparalytic Snake Bite, Viperine Snake Bite, Locally Toxic Snake bite.

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INTRODUCTION

Snake bite is an important occupational and rural hazard because India has always been a land of exotic snakes. All snakes are commonly considered by layman to be poisonous but according to the classical work of dr. Melcon smith in 'fauna of British india-1943' about 216 species belonging to 8 families are poisonous. In Maharashtra, common poisonous snakes are cobra, Russell's Viper, Saw Scaled Viper and Krait[1]. it is very difficult to give a definite statistical record of the snake bite mortality of the cases take places in the periphery, which are hardly accessible to the big hospitals. Then too, with urbanization and cutting down of forests, snake bite has become a very important preventable public hazard. 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]. WHO (1963)

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reports 40,000 annual deaths in tropical countries. Largest number of deaths reported in India is paid Bengal, UP Tamil Nadu, Bihar and Maharashtra[3]. Epidemiology of Snake bite: Incidence In India, The largest number of snake bites are recorded in Maharashtra, Tamil Nadu and Kerala and more than 1000 people die years of snake bites in Maharashtra alone[1]. Observation in all age group of 11-60 years. Male: Female ratio is 3:1 as reported by Banerjee and Siddiqui[4] (1976), Sawai and Hona[4] reported the incidence to be 66% (probably because males in fields). Nearly 75% bites are from outdoor areas. Banerjee[5], Reid and Saini[6] *et al* reported the rural incidence to be 70-80% than urban areas. Snake bites is more common in warm months (April to July) 70-80%[5,6,7] Reid H. A[8]. Sawai[9] *et al* and Benerjee R.N[4]. Observed that about 85% of bites occurred in day light when more people are exposed to the risk. They also should that the commonest site of bite are the lower extremities. Next in frequency are the upper extremities. Parikh[9] (1965) noted 58% bites on the lower extremity. 38% on the upper and 0.5% on the head, face and neck and 0.7% on the trunk. Incidence of poisonous snake bites varies from 10-24%. Benerjee R.N[5]. and Swami[7] *et al* reported an estimate of only 6.1% from hospital records of six states in India. No define statistics are available but it is thought that 25% of all bites are bites are by poisonous snakes in our country. Benerjee and Siddiqui[4] (1976) reported in their study at Safdarjung Hospital that the mortality is 5o-70% in spite of frequent use of polyvalent ASV. Reid H.A[10]. In his study out of 1159 snake bite patients in Malaya had poisonous snake bites but 53% these patients, excepted with slight or no poisoning and the mortality rate was 1.3%. All these findings are based on hospital patients, So they do not reflect a representative pattern of the disease. To decrease the mortality, more emphasis has to be laid on:-Effective treatment of snake bites careful analysis of venoms of various types of snakes as to their effects. Deriving ways and means to combat the harmful effects on human body. To educate doctors and public in the diagnosis of snake bite poisoning.

AIMS AND OBJECTIVES

To study the systemic envenomation from various types of poisonous snakes and their effective management in reducing the mortality rate.

METHODOLOGY

This was a hospital Record based study of various types snake bite patients admitted to Krishna Institute of Medical Sciences University Karad, this was carried out in one year duration. Data was collected using semi-structured proforma. Data is Presented in the forms Percentages and Proportions.

RESULT:

Table 1: Showing Incidence of Symptomatology of Neuro Paralytic Bite Poisoning

S/s	No. of Cases	Percentage
Ptosis	21	100%
Dysphasia	9	42.85%
Opthalmoplegia	18	85.71%
Unconsciousness	6	28.57%
Flaccid Limb Paralysis	6	28.67%
Convulsions	0	0%
Cellulites	8	38.09%
Respiratory Paralysis	19	90.47%

Ptosis was most common presentation of Neuro-Paralytic Bite Poisoning followed by Respiratory Paralysis, Opthalmoplegia, Dyspahgia, Cellulitis, Flaccid Limb Paralysis, Unconsciousness.

Table 2: Showing the incidence of symptomatology in viperine snake bite

S/s	No of Cases	Percentage
Local Bleed	35	83.33%
Gum bleed	3	7.14%
Hemoptysis	2	4.76%
Epistaxis	1	4.76%
Hematemesis	2	47.61%
Melena	18	4.76%
Hematuria	20	42.85%
Oliguria	2	47.61%
Ecchymosis	1	4.76%
IntracerebralHaem.	2	2.3
SubconjuctivalHaem.	2	4.76%
Cellulitis	24	57.14%
ARF	20	47.61%

Local Bleed was the most common Symptoms presented with Viperine bite followed by Cellulitis, Hematemesis, Oliguria, Hematuria, Gum bleed, Epistaxis, Subconjuctival Hemorrhage, Intracerebral Hemorrhage.

Table3: Showing incidence of complications of vasculotoxic snake bite

VT	ARF	SUR.INTER	DIC	DEATH
42	20	8	24	4

VT-Vasculotoxicity, NT-Neurotoxicity, LT-Locally toxic, SUR.INTER-Surgical Intervention. ARF was the most common complication followed by DIC, Surgical intervention and Death.

Table 4: Showing type of treatment received in vasculotixic snake bite

ASV	42
Diuretics	13
Hemodialysis (HD)	7
Blood Transfusion	4

Most of the patients required ASV followed by Diuretics, Hemodialysis, Blood Transfusion.

Table 5: Table showing incidence of respiratory assistance given in neuroparalytic snake bite

Device	No	Death
Artificial Ventilator	15	2
Intubation	4	1
Not Required Both	2	0

Maximum patients of Neuroparalytic snake bite patients required Artificial Ventilator, followed by Intubation, Not required any device.

Table 6: Distribution of the patients as per the condition at discharge

	LT	NP	VT
Complete Recovery	13	17	10
Complication	0	1	28
Death	0	3	4

From Table 6 and Table7. Completely recovery was more common in Neuroparalytic Bite, followed by Locally Toxic Bite and Viperine Bite. Death Mostly Observed in Viperine Bite followed by Neuroparalytic Bite

Table 7: Showing comparative mortality of in poisonous snake bite

	No. Cases	No. Death	%
Neuroparalytic	21	3	3.94%
Vasculotoxic	42	4	5.26%
Locally Toxic	13	0	0%

DISCUSSION

In Clinical Features Among the 150 cases studied, 74 were non-poisonous cases and 76 were poisonous cases. Out of 76 poisonous cases, 42 (55.26%) were vasculotoxic, 21 (27.63) neuroparalytic and 13 (17.10%) were locally toxic. Regarding vasculotoxic bites, the presents study shows close resemblance with the studies of safdarjang Hospital study and Saini[6] *et al*. On the contrary, the incidence of neuroparalytic bites are very less in most of the studies, than present study. This shows that elapids are more common in our locality than their localities. Vasculotoxicity is more in our locality because saw scaled viper is he common snake found here. In the present study, maximum neuroparalytics presented within first 6 hours (76%) i.e. 16/21. Maximum vasculotoxic presented within first 6 hours i.e. (50%) 21/42 and maximum patients with local toxicity within 6 hours i.e. (7.69%) 1/13. In Systematic Manifestations in Viperine Snake Bite: In the present study, 3(7.14%) patients had gum bleeding but purohit (1944) described gum bleeding as the commonest manifestation of viperinebite. In Bombay, Meeankshi Mehta (1984)[11], in the study of 104 cases, reports hematuria in 47.6% of cases which is correlating with the present study.Reid[12] *et al* in his study of 281 cases of viper bite had not found a single case with fundal hemorrhage. Above study shows that bleeding manifestation in viper bite are variable, in the present study local bleeding with haematuria were the commonest manifestations. In Clinical Manifestations of Neuroparalytic Snake bite: The diagnostic signs of elapid bite are ptosis and glossopharyngealpalsy. Reid observed drowsiness as the first systematic toxicity appearing within 1-5 hours after the bite. In the present study, 100% patients developed ptosis among 21 cases of Neuroparalytic bite. Opthalmaoplegia was seen in 18 (85.71%) patients of Neuroparalytic bite. 6 (28.57%) patients became unconscious, 6 (90.47%) patients with flaccid limb paralysis, 19 (90.47) patients developed respiratory patients among which 15 patients needed respirator. In Complications**:** ARF- Was seen in 20/42,47.61% of patients of vasculotoxic bite. (In all the cases of ARF except one, DIC was cause the cause for it.).All 3 patients died because of delay in receiving respiratory assistance. Among 4 deaths of vasculitoxic bites, 3 patients died of acute renal failure and one patient died of DIC and intracerebralhemorrhage. Maximum survival period in the present study in neuroparalytic snake bite was 2 days. Banerjee[5] *et al* (1978) reporeted survival period of 32 hours in neurotoxic bites and 3 days in case of vasculotoxicbite as 4% .If used correctly, it can reverse systemic poisoning even when given hours or days after the bite. It is therefore not only safe but highly desirable to wait for clear clinical evidence of systematic poisoning before giving ASV. But according to Bhat[6] *et al* (1974), delay in administration, delayed the reversal of coagulation defects and more quantity of ASW was required for it. Choice of antivenom[8]: Mono specific antivenins are more effective and is likely to cause reactions than polyspecificantivenoms. In Australia, 5 different monospecificantivenoms are available for land snake bite and a capibility tube ELISA kit has recently become are available for determining the snake species. If the biting species is known, the ideal treatment may be with a monovalentantivenom, as this may be less expensive and may involve administration of a lower dose of antivenom protein than with a polyvalent antivenom. However, immunization of a horse or sheep with venoms of several related spices of snakes of snakes (e.g. Viperidae) may produce an enhanced antibody response to common antigens, making the resultant

polyvalentantivenom more rather than less potent than a monovalent antivenom[13]. In the present study, ASV was given to all patients showing clinical , laboratory or systematic envenomation. Abnormal coagulation or neurological manifestations were the absolate indications for ASV. In the present study, two patients developed anaphylaxis for ASV and they were treated with steroids and adrenaline. In the present study. 76/150 (50.66%) patients received ASV. According to Reid *et al*, ASV is not very effective against local effects of venom. These local effects may be coursed either directly or indirectly by release of kinins, activation of complement etc. by different venom factors. In the present study, 69/76 recovered competly, 3 deaths in neuroparalytics were because of delay in administration of ASV producing paralysis and delay in ventilatory support who were transferred from other hospital to our hospital. Early administration of ASV prevents respiratory paralysis after elapid snake bite. Patients with evidence of respiratory paralysis after neurotoxic venom poisoning require rapid intubation and artificial ventilation[14] (Bawaskar *et al*). Blood Transfusions: In the present study, four (9.52%) patients of viperine snake bites received blood transfusions. Saini[6]*et al* (1984) and reid *et al* feel that blood transfusions is not required routinely. Heparin therapy in E.carinatus bite[15]: The venom of this snake activates pro-thrombin directly. This procoagulant effect was enhanced by a calcium lipid mixture and by factor V. In platelet rich plasma, heparin inhibited both venum induced platelet aggregation and clotting and this may explain effectiveness of heparin in correcting the coagulation defects. Heparin was not given to any patient in the present study because of controversies. David Warrell[16]*et al* were also against the use of heparin. **Antibiotics:** Antibiotics were given to those patients who had poisonous snake bite with cellulites. Therefore, Gentamicin was added wherever necessary. **Neostigmine and Atropine**

Neostigmine acts by reversal of N-M block at the myoneural junction. It acts by anticholinesterase like action. Atropine is given before neostigmine to block the muscarinic action of neostigmine on glands, smooth muscles and heart. In the present series, neostigmine with atropine was given to 13 patients out of 21 patients of neuroparalytic snake bite. Most of the patients responded to it within 4-6 hours. In these patients, to improve was respiratory paralysis, then opthalmoplegia and lastly ptosis.G.K. Dubay[17]*et al* (1981), Banerjee[4] *et al* Dash S.C. *et al* (1976) are strong supporters of this regime.Krait venom acts both on pre and post synaptic junctions while cobra venom acts only on post synaptic junctions. Kraits have been responsible for all or some of the bites reported in previous evaluation of anticholinesterase and although beneficial effects have been described in individual patients, the results from a series of cases have been consistent.

Treatments of Complications

ARF: In the present series, ARF was observed in 20 patients among 42 (47.61%) patients of vasculotoxic snake bite. 11 patients were treated conservatively with diuretics, renal died and fluid restriction. 7 patients needed hemodialysis. 3 patients died of renal failure among which 2 patients died after hemodialysis and one died because of acute renal failure.Saini[6] *et al* 8 cases of ARF and 7 cases recovered with conservative treatment. Meenakshi Mehta[11] in a series of 104 cases reported only 2 cases of ARF who improved with conservative treatment. **Steroids in Viper bite:** Acco**rding** to Hoback and Green 1953[19], Grody 1954, steroids have been vaunted for bite. Russell and Emery (1961)[18] could confirm any benefit experimentally.

Respiratory Support

In the present study, among 21 patients of neuroparalytic bite (71.42%), 15 needed ventilatory support and 4 (19.04%) patients needed only intubation. Among which 3 patients died. Total duration of ventilator was less than 24 hours for most of the patients except one. Not a single patient needed tracheostomy. Early deaths due to cobra and krait bite occur as a result of generalized paralysis of respiratory muscles, aspiration of vomitus and delay in seeking medical care.

SUMMARY AND CONCLUSION

Time of onset of systemic manifestation was earlier in neuroparalytic snake bites than vasculotoxic snake bite. vasculotoxic snake bites (55.26%) were more common than neuroparalytic bites (0.63%) Commonest vasculotoxic manifestation was Local bleed (83.33%) and next on the list was cellulites (57.14%). Haematuria (42.85%) was the commonest manifestation in those patients who developed ARF. Ptosis (100%) was the commonest and earliest manifestation of neuroparalytic snake bite. Out of vasculaotoxic snake bites, 20 were of ARF, 7 required haemodialysis. 4 DIC patients received blood transfusion. 13 patients of ARF received diuretics and recovered completely.13 cases were locally toxic, out of them 4 patients developed cellulitis.19 patients with neuroparalytic bite required artificial ventilation.7 patients died because they came late to seek medical treatment and by that time complications were beyond control and were associated with systemic infection.

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