SPECTRUM OF SNAKE BITE POISONING IN JAMMU- AN OBSERVATIONAL STUDY

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ABSTRACT

BACKGROUND

In Jammu region the majority of snake bites occur between May and October, the maximum incidence being in rainy season. The region of Jammu harbours various types of poisonous snakes, mainly belonging to families Elapidae and Viperidae. Snakes belonging to family Elapidae are mainly neurotoxic, though some amount of haemotoxic and cardiotoxic effect have also been reported. Those belonging to family Viperidae are mainly haemotoxic and are commonest ones in Jammu.

MATERIALS AND METHODS

Summer and rainy seasons constitute the time when snakes are out of hibernation. Snake bites during this period comprise a major part of hospital morbidity and mortality, thus a study of poisonous snake bites in Jammu is of interest because of two important reasons-

a. In finding out the behaviour of this accidental ailment;
b. In finding out the best possible approach to handle the victim admitted to the hospital.

There has been a lot of controversy regarding the mode of administration of A.V.S. that has been used in varying schedules and with variable success.

RESULTS

106 cases of poisonous snake bites- 100 haemotoxic and 6 neurotoxic- admitted to male and female wards of Medicine, Government Medical College, Jammu, between May 2002 and April 2003 have been studied.

CONCLUSION

The commonest cause of poisonous snake bite in Jammu region is found to be Echis carinatus. Bleeding manifestation is the commonest disorder caused by viper bites, in the presence or absence of swelling at the site of bite. Incidence of uraemia is high in our setup and increases with the delay in the institution of management. Coagulation studies performed on 30 patients have revealed primary fibrinogenolysis in 27 and disseminated intravascular coagulation in 3 cases. The most effective mode of administration of antivenom serum has been worked up. Clotting time and clot quality have been found to be reliable guides to determination of antivenom therapy on the bed side. It has been found that rapid infusion of antivenom serum not only quickly neutralises the venom but also reduces the rate and magnitude of complications, duration of morbidity and stay in the hospital. Bleeding manifestations or coagulation defect did not occur in cases of neurotoxic snake bites. The response to antivenom therapy in elapid bites has been dramatic with rapid recovery.

KEYWORDS

Elapidae, Viperidae, Clot Quality, Antivenom Serum.

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BACKGROUND

In Jammu region the majority of snake bites occur between May and October, the maximum incidence being in rainy season (Lahori et al, 1961).[1]

Although most snakes (more than 90%) are non-poisonous, yet most of those which bite unprovoked are poisonous. The region of Jammu harbours various types of poisonous snakes, mainly belonging to families of Elapidae and Viperidae. Snakes belonging to these two families are the commonest encountered in this country. Snakes belonging to family Elapidae are mainly neurotoxic, though some amount of haemotoxic and cardiotoxic effect have also been reported. Those belonging to family Viperidae are mainly haemotoxic and are the commonest ones in Jammu Province, (Bhat, 1974).[2]

Since the summer and rainy seasons constitute the time when snakes are out of hibernation, snake bites during this period comprise a major part of hospital morbidity and mortality.

Aims and Objectives

The present study has been conducted with the following aims and objectives-

a. Assessment of clinical profiles;
b. Assessment of renal involvement;

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c. Assessment of coagulation defects; and
d. Standardisation of dosage schedule of A.V.S.

MATERIALS AND METHODS

The present study comprised of 106 cases of poisonous snake bites admitted in male and female wards of Medicine Dept. of Government Medical College, Jammu, between May 2002 and April 2003. The patients were grouped as follows-

Haemotoxic Snake Bite- 100 patients of haemotoxic bite for purposes of treatment were randomly grouped into following categories-

a. 40 patients in group "Q"
b. 25 patients in group "D-2"
c. 35 patients in group "D"

The details of these groups are mentioned under "A.V.S. Therapy" below.

Neurotoxic Snake Bite- This group included 6 patients.

Diagnosis- The criteria for labelling a patient as a case of poisonous snake bite were-

i) History of bite by a poisonous snake, if the patient had seen the snake and could provide its satisfactory description;
ii) Recognition of the snake in case the patient had brought the snake along with dead or alive;
iii) Clinical features of envenomation;
iv) Results of investigations favouring systemic poisoning.

The clinical profile of each case was maintained in detail as per the proforma. Before the tests for diagnosis were undertaken, the tourniquet was released in all cases. Following tests were undertaken before and after completion of therapy-

1. Blood: Hb, TLC, DLC, Platelet count.
2. Bleeding time.
3. Clotting time.
4. Clotting quality.

In case the clot forms, it was kept for observation for clot quality for 4 - 6 hours. The clot was graded according to the classification given by Reid et al (1963)[3,4] as follows-

Grade-1 Normal- Cell deposit not above bottom curve of the tube clot approximately 50% of the original blood volume.

Grade-2 Slight defect- Cell deposit increased above the bottom curve of the tube up to 30% of the original whole blood volume; clot size diminished in proportion.

Grade-3 Moderate defect- cell deposit 30% - 50% of the whole blood volume. Clot size about half the size of a contracted normal clot.

Grade-4 Severe defect- cell deposit 50% or more of original volume. Clot becoming a small speck.

Grade-5 No clot.

5. Urine R/E:
6. E.C.G.:
7. Serum urea
8. Serum creatinine
9. Serum electrolytes
10. Coagulation Studies-

Following coagulation studies were conducted on 30 randomly selected patients:-

i) Prothrombin time index.
ii) Thrombin time.
iii) P.T.T.K. (Partial Thromboplastin Time on Kaolin).
iv) Fibrinogen levels.
v) E.L.T. (Euglobulin Clot Lysis Time).
vi) Protamine sulphate test.

In all these cases, a P.B.F. was examined to observe the morphology of cells, especially the red blood cells. These tests were carried out in collaboration with Haematology Section of Medical College Hospital.

11. C.S.F. Examination- A C.S.F. sample obtained by lumbar puncture was subjected to biochemical and cytological analysis in patients having meningism.
12. Stool Examination- Stool examination for occult blood was conducted, reportedly in patients who had abdominal cramps.

Patients with poorly coagulable or incoagulable blood were labelled as cases of haemotoxic bites; the diagnosis of neurotoxic bites was made on the basis of clinical data.

A.V.S. Therapy- All patients were treated with lyophilised A.V.S. produced at Haffkine Institute, Bombay. The patients for A.V.S. therapy were kept in three groups and were selected randomly before treatment. The A.V.S. therapy was decided according to C.T. and clot quality, which were determined at the outset of treatment.

The regimens of A.V.S. therapy used were-

1. In patients who were on "Quick Regimen" or "One Hourly Regimen" (Group Q) if the blood was incoagulable, 100 mL A.V.S. was infused I.V.; if the blood was poorly coagulable or clot started re-dissolving within 30 minutes or clot grade after assessment was 4 or 3, 50 mL A.V.S. was infused. The clotting time was noted 1 hour after first infusion and therapy was decided similarly again. The process was repeated till the clotting time and clot quality came to normal.
2. In patients treated 12 hourly or twice daily (Group D-2), the clotting time was repeated 1 and 12 hours after the first infusion (of 100 or 50 mL A.V.S. depending upon clot quality). If the clotting was poor after 12 hours, the A.V.S. was administered as described under (1) above. The clotting time was repeated "1" and "12" hourly and A.V.S. infused till coagulation returned to normal.

3. In patients treated 24 hourly once daily (Group D), the clotting time was repeated "1" and "24" hours after the first infusion. In cases of poor clotting, 24 hours after the first infusion A.V.S. administration was carried out as described under (1) above and the process was repeated till the correction of coagulation defect.

Patients were discharged one day after the clotting time came to normal and a clotting time was done just prior to the discharge from the hospital.

In cases of neurotoxic snake bites after taking blood samples for preliminary tests and recording an electrocardiogram, an I.V. infusion with 5% dextrose was setup. The lyophilised A.V.S. was reconstituted and injected through the tubing of the infusion set, one ampule at a time. The chest expansion was measured at intervals of 5 - 10 minutes; when it came to 3.5 to 4 cms on inspiration (and usually by that time patient could respond to oral command) the A.V.S. therapy was ceased. The patient was observed in the ward for 1 - 2 days. The three patients who were markedly paralysed and had very little respiratory effort were intubated in the Intensive Care Unit.

RESULTS

Of the 100 cases of haemotoxic snake bites that were included in the present study, age distribution is shown in Table 1.

Sl. No.	Age in Years	No. of Cases
1.	14-20	32
2.	21-30	25
3.	31-40	21
4.	41-50	13
5.	51 and Above	9

Table 1. Age Distribution of 100 Cases of Viper Bites

Sl. No.	Occupation	No. of Cases
1.	Field Workers (Farmers and Labourers)	48
2.	Housewives and Maidens	27
3.	Students (Of Schools and Colleges	16
4.	Sedentary Workers Office Bearers	6
5	Shopkeepers	3

Table 2. Occupation of 100 Cases of Viper Bites

The commonest site of bite was feet around or distal to ankle joints, commonly over the toes (Table 3).

Sl. No.	Site of Bite	No. of Cases
1.	Feet	74
2.	Hands	23
3.	Legs (Shin and Calf)	2
4.	Buttock	1

Table 3. Sites of Bites of 100 Cases of Viper Bites

Measures undertaken immediately after bite are shown in Table 4.

Sl. No.	Measure	No. of Cases
1.	Tourniquet (Two Tourniquets)	89 (13)
2.	Local Incisions	4
3.	Local Herbs	1
4.	Medical Aid at Health Centres (consisting of analgesics, antihistaminics, steroids and A.V.S. from 10 - 200 mL)	16

Table 4. Measures Adopted in 100 Cases of Viper Bites before Admission

The reporting time after bite ranged from 16 minutes to 11 days and is shown in Table 5.

Sl. No.	Time after Bite	No. of Cases
1.	0 - 30 minutes	9
2.	½ - 5 hours	57
3.	5 - 24 hours	14
4.	24 - 48 hours	9
5.	2 - 11 days	11

Table 5. Reporting Time in 100 Cases of Viper Bites

Local symptoms are shown in Table 6.

Sl. No.	Features	No. of Cases
1.	Fang marks	100
2.	Swelling	89
3.	Tingling, numbness, discomfort	79
4.	Pain	63
5.	Oozing of blood	47
6.	Lymph node enlargement	13
7.	Blisters	8
8.	Bluish discolouration around bite site	5
9.	Gangrene (of bitten finger)	1

Table 6. Local Features in 100 Cases of Viper Bites

Sl. No.	Features	No. of Cases
1.	Nausea	16
2.	Nausea and Vomiting	45
3.	Giddiness	5
4.	Frank Haematuria	25
5.	Bleeding gums	11
6.	Haematemesis	10
7.	Haemoptysis	6
8.	Epistaxis	3
9.	Haematomas	3
10.	Bleeding from piles	1
11.	Blood in stools (not from piles)	2
12.	Subconjunctival haemorrhage	2
13.	Melena	2
14.	Abdominal cramps	20
15.	Ecchymoses	29
16.	Severe headache	2
17.	Hiccup	3
18.	Progressive drowsiness	7
19.	Oliguria	17
20.	Anuria	5
21.	Irrelevant Talk	2

Table 7. Systemic Features in 100 Cases of Viper Bites

Of the systemic symptoms (Table 7) commonest were nausea and vomiting. Physical Signs (Table 8).

Sl. No.	Signs	No. of Cases
1.	Apprehension	95
2.	Tachycardia	84
3.	Bradycardia	4
4.	Hypotension	4
5.	Mild dehydration	17
6.	Fever	1
7.	Signs of meningism	2
8.	Acidotic breathing	6
9.	Disorientation and confusion	8
10.	Signs of C.C.F.	1
11.	Distension abdomen	1
12.	Absent bowel sounds	1

Table 8. Physical Signs in 100 Cases of Viper Bites

Sl. No.	Parameter	No. of Cases
1.	Low haemoglobin (Less than 12 G%)	69
2.	Leucocytosis (10,000 - 17,000/cu mm)	81
3.	Thrombocytopaenia (40,000 - 1,20,000/cu mm)	9
4.	Prolonged bleeding time	11
5.	**Clot quality -** • Grade 5 • Grade 4 • Grade 3 • Grade 1	 80 8 10 2
6.	**Urine R/E** • Microscopic haematuria • Haematuria and Proteinuria • Isolated Proteinuria • Nephrotic proteinuria • Hyaline and granular casts • Pyuria	 53 18 9 1 4 1
7.	E.C.G. changes	20
8.	Raised serum urea	34
9.	Raised serum creatinine	27
10.	Hyponatraemia	40
11.	Hypokalaemia	6
12.	Occult blood in stool	3
13.	Haemorrhagic C.S.F.	2

Table 9. Routine Parameters on Admission in 100 Cases of Viper Bites

Sl. No.	Serum Urea Level (in mg%)	No. of Cases
1.	20 - 40	66
2.	41 - 80	25
3.	81 - 200	4
4.	201 - 326	5

Table 10(A). Urea and Creatinine Levels in 100 Cases of Viper Bites at the Time of Admission

Sl. No.	Serum Creatinine Level (in mg%)	No. of Cases
1.	1 - 2	73
2.	2.1 - 4.0	17
3.	4.1 - 7.0	6
4.	7.1 - 16.0	4

Table 10(B). Urea and Creatinine Levels in 100 Cases of Viper Bites at the Time of Admission

Definite E.C.G. changes which were noticed before treatment and disappeared after treatment were seen in 20 patients and are mentioned in Table 11.

Sl. No.	E.C.G. Change	No. of Cases
1.	ST-depression with T-inversion	10
2.	ST-elevation	4
3.	Sinus bradycardia	4
4.	Tented T-waves	2
5.	Short PR-interval	1
6.	Sinus tachycardia	84

Table 11. E.C.G. Changes in 100 Cases of Viper Bites

Sl. No.	Name of Test	No. of Cases	Before Treatment	After Treatment
1.	P.T.I.	11 19	No clot 30 - 80%	76 - 100%
2.	P.T.T.K. (Normal : 35 - 65 secs)	11 19	No clot 50 - 118 secs	36 - 80 secs
3.	Thrombin time (Normal: 6 - 11 secs)	2 10 18	Normal No clot 10 - 20 secs	6 - 11 secs
4.	Fibrinogen level (Normal: 200 – 400 mg%)	1 1 28	Normal No clot 32-170 mg% (18- Less than 100 mg%)	200 - 310 mg%
5.	E.L.T. (Normal: More than 2½ hours)	1 4 25	Normal No Clot 20 - 105 mins	More than 3 hours
6.	Protamine sulphate test	3 27	Positive Negative	Negative

Table 12. Results of Coagulation Studies Before and After Treatment in 30 Cases of Viper Bites

Results of coagulation studies conducted in 30 patients, before and after treatment are shown in Table 12.

Sl. No.	Complication	No. of Cases
1.	Uraemia	34
2.	Subarachnoid haemorrhage	2
3.	Septicaemic shock	2
4.	C.C.F.	1
5.	Peritonitis	1
6.	Local Gangrene	1

Table 13. Complications in 100 Cases of Viper Bites

The doses of A.V.S. used in cases of haemotoxic bites are shown in Table 14.

The recovery period in cases of neurotoxic snake bites is shown in Table 14.

Sl. No.	Treatment Group	No. of Cases	Dose Used (in mL)	Mean Dose (in mL)
1.	Q	40	50 – 600	185
2.	D-2	25	50 – 550	220
3.	D	35	50 – 550	257.1

Table 14. Dose of A.V.S. required in 100 Cases of Viper Bites

Sl. No.	Treatment Group	No. of Cases	Dose Used (in mL)	Mean Dose (in mL)
1.	Q	38 2	1½ - 18 26 - 28	6.17
2.	D-2	25	2 - 50	21.48
3.	D	35	2 - 9 Days	50.1

Table 15. Recovery Period in 100 Cases of Viper Bites

The present study includes 6 patients who presented with neurotoxicity after the snake bite. Their clinical profile is shown in Table 16.

Sl. No.	Complication	Number of Cases	
1.	Male Females	1 5	All Rural
2.	Sites of bite • Scrotum • Hand • Foot/ Leg	 1 3 2	
3.	Tourniquet	1	
4.	Incision and Tourniquet	1	
5.	Fang marks	5	
6.	Local swelling	3	
7.	Blister	1	
8.	Progressive drowsiness	6	
9.	Coma	2	
10.	Nasal twang of voice	4	Could be tested in 4 cases only
11.	Regurgitation of food	4	
12.	Dysphagia	4	
13.	Abdominal pain and distension	2	
14.	Ptosis	6	
15.	Weak palate reflex	3	
16.	Absent palate reflex	1	
17.	Hypotonia	6	
18.	Dilated pupils	4	
19.	Cyanosis	1	
20.	Leucocytosis	5	
21.	Respiratory rate	10 - 16/ min	
22.	Chest expansion	0.5 - 1.5 cms	
23.	Time of presentation	2½ - 14 hours after bite (Mean 8.1 hours)	
24.	Onset of symptoms	½ - 10 hours after bite (Mean 4 hrs.)	
25.	Recovery period • Respiratory activity • Ptosis • Local swelling	 • 30 - 90 minutes (mean 46 minutes) • 24 - 50 hours • 3 - 7 days	

Table 16. Clinical Profile of 6 Cases of Neurotoxic Snake Bites

DISCUSSION

In the Jammu region of J and K State, snake bites are very common during the summer and rainy seasons. The commonest poisonous snake bite in this region is undoubtedly due to Echis carinatus.

As expected majority of the patients were males belonging to labour class; in our study 70% were males. Most of these labourers work and walk bare footed; hence, distal parts like feet are bitten more frequently than proximal parts. Similar observations have been made by others (Bhat 1974[2]; Warrel et al 1977).[5,6]

Local swelling is an important feature of poisonous bites; 89% of our patients had local swelling, which started immediately after bite. This was contributed partly by envenomation and partly by the tourniquet, which sometimes was applied very tightly.

Careful search for fang marks is rewarding. Fang marks were seen in all of our cases. These could be visualised, though faint, even in those who reported quite late. Russel (1968)[7] observed fang marks in 100% and Lahori et al (1981).[1] In 94% cases Bhat (1974)[8] observed fang marks in 73% cases, since many (27%) of his patients had given local incisions.

Blisters at the site of bite were seen in 8% cases. Warrel et al (1977) in cases of E. carinatus bite recorded blisters in 13% and Lahori et al (1981).[1] In about 14% cases, Bhat (1974)[2] however recorded blisters in 42% cases.

Of the bleeding manifestations, the commonest detected clinically was ecchymoses seen in 29% cases. Various other bleeding manifestations after Viper bites include bleeding from gums (11%), haematemesis (10%), bleeding per rectum (3%), subconjunctival haemorrhage (2%), epistaxis (3%), melena (2%) and haematomas (3%). None of the patients having haematemesis had history of acid peptic disease. Bleeding per rectum may occur in the absence of an anorectal abnormality.

One of the serious bleeding manifestations is intracranial haemorrhage. The reported incidence of cerebral haemorrhage is about 2% (Bhat 1974[2]; Warrel et al[6] 1977); 2 of our patients who initially complained of severe headache and later became drowsy were proved to have subarachnoid haemorrhage. The patient may be disoriented and confused due to uraemia, subarachnoid haemorrhage, blood loss or severe dehydration due to persistent vomiting or low intake.

After viper bites clotting defect is the most consistent finding, which may appear as early as half hour after bite. Blood was non-clotting in 72% of our cases at the time of admission. Although, clot did form in certain cases, the clot quality deteriorated to grade 5 in 82%, grade 4 in 8% and grade 3 in 10% cases after sometime.

An important observation in our study has been that clot qualities of the blood samples collected 12 and 24 hours after finishing the A.V.S. infusion have not been significantly different from those collected one hour after infusion. Once the clot quality had come to normal (Grade 1 clot) the prothrombin time, thrombin time and fibrinogen levels were mostly within normal range. This supports the view of Reid et al,[9,10,11] (1963) that the clot quality closely parallels the thrombin titre. This also supports the view of Adelson et al (1960)[12] and Lewis et al[13] (1961) that after neutralisation of venom the fibrinogen levels come to normal within hours, although thrombocytopenia may take longer to correct. Fibrinogen levels in 86% of patients was subjected to coagulation studies 10 - 24 hours after the correction of clotting defect were normal; in others the fibrinogen was 190 mg%.

Coagulation studies revealed primary fibrinogenolysis in 90% and D.I.C. in 10% of our cases. D.I.C. was evidenced by low fibrinogen levels, thrombocytopenia and a positive protamine sulphate test; F.D.P's were not assessed. The predominant coagulopathy reported after Russel's viper envenomation (Basu et al[14] 1977; Sarangi et al[15] 1977) is disseminated intravascular coagulation. Warrel et al[5,6] (1977) found evidence of D.I.C. in all cases of Echis carinatus bites. This wide difference in the coagulation abnormalities found by other workers and by us reflects a different behaviour of the local fauna.

The incidence of neurotoxic snake bites is much less in our area. In contrast to 100 cases of viper bites, only 6 cases of neurotoxic bites presented during the same period. Such incidence seems to vary from place to place. Banerjee and Siddiqui[8] (1976) have recorded a higher incidence of neurotoxic bites- 29.41 to 62.5%.

Ptosis seems to be the most important feature of neurotoxicity, for it occurs in all cases and is the last to disappear; 2 of our patients were in deep coma. Respiratory excursion was reduced in all cases and 1 patient was cyanosed. All our patients had hypotonia of all the four limbs. Banerjee and Siddiqui[8] (1976) recorded flaccid paralysis in 26% cases, while 19% patients seen by them had convulsions, none of our patients had convulsions, encephalitis or meningitis. One patient developed pain abdomen which was diffuse and progressive associated with distension and absent bowel sounds which persisted for about 14 hours. We assume that this is due to autonomic neuropathy. Most of our patients had leucocytosis with total count of 12,000 to 14,000/cu mm and W.B.C.'s of 65% - 79%. Coagulation abnormalities do not occur in neurotoxic bite in our area.

After a snake bite it is advisable to tie a light tourniquet proximal to the area of bite, immobilise the bitten limb and transport the patient to the nearest centre where expertise with the knowledge of management of snake bite is available. Application of a tourniquet would not reduce the dose of A.V.S. required (as this was no different in cases who tied and who did not tie a tourniquet), but can apparently delay the absorption of venom and onset of symptoms. One patient of neurotoxic bite had tied a very tight tourniquet proximal to the bite site and was received 12 hours after bite. Similarly we noticed that after the release of tourniquet, the deterioration in clot quality after viper bites was quicker.

All patients who have local cellulitis should be given an antibiotic cover. Patients of shock need to be managed aggressively, as profound shock is commonly associated with renal shut down, fluid and electrolyte therapy needs to be appropriately planned and administered.

Patients with subarachnoid haemorrhage need to be managed on conservative lines. If the venom is quickly neutralised by the use of specific antivenom and further bleeding is stopped, prognosis in such cases is good; whereas, 33% of Bhat's[2] (1974) patients and 66% of those seen by Warrel et al[5,6] (1977) died. There was no mortality in our cases of subarachnoid haemorrhage. However, we had a rather small number of cases to make any generalisations.

Conservative management of acute renal failure seems to be justified. Although, 5% of our patients had been completely anuric for 24 - 96 hours and their renal functions continued to deteriorate for further 1 - 5 days, all the patients started passing adequate urine and showing improvement with forced diuresis and other conservative measure. The role of dialysis appears controversial in the management of acute renal failure following viper bites. In a controlled study by Basu et al[14] (1977), the mortality rate in non-dialysed group was 57.2% and in dialysed group was 51.6%. The situation is further complicated by the fact that whereas Chugh et al[16] (1975) and Russel[17,18] (1967) advocate haemodialysis, Basu et al[14] (1977) in their

experience found peritoneal dialysis superior to haemodialysis. We did not subject any of our patients to dialysis. Favourable response to conservative management may reflect a milder form of renal injury in our cases of uraemia. But a poor response to dialysis as seen by Basu et al[14] (1977) and Sarangi et al[15] (1980) may reflect that the natural course of renal disease in snake bite cannot be much altered.

Of the specific treatment in snake bite A.V.S. therapy stands on the top, whereas most others have used A.V.S. in empirical doses and in variable schedules. The present study has been designed to institute A.V.S. therapy under different schedules and to find out the most effective method that would neutralise the injected venom in shortest duration of time. Rapid A.V.S. therapy not only reduces the duration of coagulation defect, but also abates the chances of spontaneous haemorrhage. After the clotting time and clot quality came to normal, none of our patients continue to bleed. We advocate that with incoagulable blood, 100 mL A.V.S. should be infused rapidly and clotting time repeated after one hour. If the blood is incoagulable, another 100 mL should be infused. If it clots, the clot should be kept for observation. In case of grade 3 or 4 clot, 50 mL A.V.S. should be infused. This should be repeated till the coagulation defect is reversed. We have used up to a maximum of 600 mL of A.V.S. with success. Infusion of 100 mL of lyophilised A.V.S., however, rapidly corrected the coagulation abnormality. Even in cases of D.I.C. response to A.V.S. is dramatic. Considering the views of Rechnic et al (1962), Chang et al,[19,20] (1963) and Chugh[16,21] (1964) regarding the mechanisms of incoagulability of blood in snake envenomation, observation of transient benefit of fibrinogen therapy by Reid et al[3,4,22] (1978) and futility of heparin in cases of D.I.C. (Warrel et al[5,6] 1976) and efficacy of specific antivenom therapy we neither used fibrinogen nor heparin.

Persisted with rapid infusion of A.V.S. in a single dose till the spontaneous respiratory activity returned to a satisfactory level. We believe that the best antidote to neurotoxic venom is the specific antivenom and we have seen recovery from neuroparalytic features in a short period of time (mean 46 minutes), such a short recovery period has not been reported earlier. Whereas others have reported very high mortality in neurotoxic bites (Banerjee and Siddiqui[8] 74.4%; Sarangi et al[15] 60%) all our patients survived. Although, 6 is too small a number to make generalisations, considering consistently favourable and uniform response in all these cases we believe that antivenom in proper doses is the most effective remedy for cases of neurotoxic snake bites.

CONCLUSION

A total of 106 cases of poisonous snake bites- 100 haemotoxic and 6 neurotoxic- admitted to male and female wards of Medicine, Government Medical College, Jammu, between May 2002 and April 2003 have been studied. Echis carinatus was found to be the most common cause of poisonous snake bite in Jammu region. Bleeding tendency was found to be the most commonest manifestation of Viper bites. Incidence of uraemia is high in our setup and increases with the delay in the institution of management. Coagulation studies performed in 30 patients have revealed primary fibrinogenolysis in 27 and disseminated intravascular coagulation in 3 cases. The most effective mode of administration of antivenom serum has been worked up. Clotting time and clot quality have been found to be reliable guides to determination of antivenom therapy on the bedside. It has been found that rapid infusion of antivenom serum not only quickly neutralises the venom but also reduces the rate and magnitude of complications, duration of morbidity and stay in the hospital. Bleeding manifestations or coagulation defect did not occur in cases of neurotoxic snake bites. The response to antivenom therapy in elapid bites has been dramatic with rapid recovery.

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