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A STUDY OF ACUTE RENAL FAILURE DUE TO SNAKE BITE ENVENOMATION IN SOUTHERN TIP OF TAMILNADU.

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ABSTRACT :

Background: There are about 2700 species of snakes recognized world over of which about 450 species are venomous..Venomous snakes are broadly classified into 4 families : Elapidae, Viperidae, Hydrophilidae, Colubridae. Snake bite is an important cause of hospital admission and mortality in tropical countries like India due to occupational hazard.

Methods: Data collected from Department of Emergency Medicine of Sree Mookambika Institute of Medical sciences, kanyakumari, tamil nadu, Patients admitted in the emergency department, with snake bite induced renal failure and coogulation abnormalities were examined from 10/2023 - 3/2024 – Total number of patient is 50 Patients.History was elicited about type of snake, site of bite, time of bite, the native treatment taken, symptoms like oliguria, bleeding tendencies and cellulitis.
Site of snake bite is examined for presence of cellulitis , fang marks, bleeding from site of bite, local necrosis, gangrene.

Results: In the study mortality in patients admitted with hypotension was 64%. Mean no of ASV and dialysis required in these patients were more compared to other patients. In our study 13 patients did not required dialysis. They were treated with fluid correction, antibiotics and conservative management. All other patients require peritoneal or hemodialysis. 5 patients expired 3 cases received ASV before admission 2 cases received ASV after admission. One died 1 day after, 2 died 5 days after admission 1 died 6 days after admission, 1 died 11 days after admission.

Conclusion: Renal lesion due to snake bite may be of two types – of which Acute tubular necrosis accounts for 70 to 80%, acute cortical necrosis accounts for 20 to 30%.Oliguria which lasts for 4 to 15 days suggests the possibility of acute cortical necrosis which carries worst prognosis.

Keywords: Anti snake venom, Hemodialysis.Acute renal failure.

INTRODUCTION:

Snakes are a fascinating part of nature. Their colour, movement, and secretive habits make them more mysterious. There are about 2700 species of snakes recognized world over of which about 450 species are venomous..Venomous snakes are broadly classified into 4 families : Elapidae, Viperidae, Hydrophilidae, Colubridae. Snakes are classified on morphological grounds from the arrangement of their scales (Lepidosis), dentition, Osteology, Myology, Sensory Organs and Hemipenis and more recently by immunological studies of their venoms and serum proteins.

The clinical manifestations depend upon the dose of venom injected ; and vary from mild local symptoms to extensive systemic manifestations. Pain and swelling of the bitten part appear within a few minutes and may be followed by blister formation and ecchymosis. Bleeding is seen in 65percent of cases and manifests as continuous ooze from the site of the bite, haematemesis, malena, and haematuria. Bleeding can also occur into the muscles and serosal cavities, and may be severe enough to produce shock. The blood is incoagulable in patients with severe systemic envenomation. Sea-snake bites cause myonecrosis, resulting in severe muscle pains and weakness.

The first indication of renal failure is oliguria or anuria, which develops within a few hours to as late as 96 h after the bite (Chuge et al. 1984). About half the cases give a history of passage of ‘cola-coloured’ urine. Non-oliguric renal failure is seen in less than 10percent of cases. Patients with severe bleeding, disseminated intravascular coagulation or secondary sepsis may present with hypotension. Life-threatening hyperkalemia necessitating immediate dialysis may develop in those with intravascular haemolysis. Oliguria usually lasts for 4-15 days, and its persistence indicates the possibility of acute cortical necrosis(Chugh 1989).

Laboratory investigations show evidence of coagulopathy, there is severe hypofibrinogenaemia, reduction of factors V, X, and XIIIA, protein C, and antithrombin C, depletion of factor V, X and fibrinogen and elevation of fibrin degradation products are frequently observed. Leukocytosis and elevated haematocrit due to haemoconcentration may also be seen.

Eventhough the basic therapeutic approach to renal failure following snake bite is the same as that for ARF due to any other cause, problems such as bleeding, shock, and sepsis complicate management. Early administration of antivenom is vital in patients with systemic envenomation. Experimental studies have shown that delay in administration results in a steep increase in the antivenom dose requirements indications include incoagulable blood, spontaneous systemic bleeding, intra vascular haemolysis, local swelling involving more than 2 segments of the bitten limb, and a serum FDP concentration greater than 80 µg/ml in those reporting within 2h of the bite (Warrell 1989, 1999). Knowledge of the offending snake species allows administration of monovalent antivenom wherever this is available.

Immuno diagnostic techniques are helpful in the easy and rapid identification of the venom antigen. ELISA has been used extensively in the rural Thailand, but the currently available test is not quick enough for the clinicians. Precise identification of the snake is not essential for management in regions where only polyvalent antivenom is available. Indian authorites recommend until the effects of systemic envenomation disappear (Tariang ET AL.1999). A simple way is to monitor efficacy is by monitoring whole blood clotting time three to four times a day. Coagulability is generally restored within 6 h of an adequate dose. The clotting time must be done for at least 3 more days, as delayed absorption of the venom could lead to recurrence of the coagulopathy. Immunoassays permit serial estimation of venom levels, and are usefull in guiding antivenom therapy. In sea – snake envenomation, patients require from 100 to 1000 units of Enhydrina schistose
antivenom. Other therapeutic measures include rerpalcement of blood loss with fresh blood or plasma, maintenance of electrolyte balance, administration of tetanus immunoglobulin, and treatment of pyogenic infection with antibiotics. The overall mortality rate is about 30 percent (Chug ET AL. 1984).

The kidneys are normal or slightly enlarged, and the surface may show petechial haemorrhages. Light microscopy shows acute tubular necrosis in 70-80 percent of patients (Chugh 1989). The tubules are lined by flattened epithelium and the lumina contain desquamated cells and hyaline or pigment casts. Varying degrees of interstitial oedema, inflammatory cell infiltration with eosinophils, mast cells and hyperplastic fibroblasts, and scattered areas of haemorrhage may be seen. Electron microscopy reveals dense intracytoplasmic bodies representing degenerated organelles in the proximal tubules, and electron-dense mesangial deposits. Acute interstitial nephritis necrotizing vasculitis involving interlobular arteries, and crescentic glomerulonephritis may be seen occasionally (Sitprija et al. 1982). Acute cortical necrosis (Fig 7) carries the worst prognosis and is seen in about 20 – 25 percent of ARF cases following Russell's viper and E. carinatus bites(Chug ET AL. 1984).
A number of clinical and experimental studies have provided insights into the pathogenetic mechanisms that lead to ARF in snake-bitten patients. These include direct nephrotoxicity of venom, hypovolaemia, haemolysis, myoglobinuria, and disseminated intravascular coagulation.

Renal lesions can develop as a result of direct cytotoxic effects of the snake venom on the kidney. Rats injected with the venoms of B.Jararaca, Agkistrodon piscivorus and rattlesnake developed increased excretion of tubular enzymes and histopathological changes of acute tubular necrosis (Burdmann et al.1983). Administration of Russell's viper venom leads to a dose- dependent decrease in inulin clearance in the isolated perfused rat kidney. Willinger et al.(1995) showed extensive destruction of the glomerular filter lysis of vessel wall, and epithelial cell injury in all segments of the tubule following administration of Russell's viper venom to experimental animal. The structure of some of the snake venoms, including the saparatoxin of the Israeli burrowing wasp, is similar to endothelin – I, one of the most potent vasoconstrictor substances known. Vasculotoxic factors have been isolated from the venoms of several snakes, including E.carinatus, Vipera palastinae, Agkistrodon halys, B. Jararaca, and Habu snake. Studies using the Habu snake venom have shown development of mesangiolysis.
Hypotension and circulatory secondary to bleeding and release of kinins and depression of the medullary

vasomotor center or the myocardium play a significant pathogenetic role. Kinin-forming enzymes (kininogenases) are present in crotalid venom, V. oakastubae deoresses the medullary vasomotor center, and Bitis arientans venom causes hypotension through a combination of myocardial depression, arteriolar dilatation, and increased vascular permeability.

The fibrinolytic activity is either due to direct action of the venom or a physiological response to fibrin deposition. Phospholipase A2 also leads to platelet aggregation. The demonstration of fibrin thrombi in the renal microvasculature, both in clinical and experimental studies, confirms the role of disseminated intravascular coagulation in the genesis of renal lesions (Chug et al.1984).

Snake bite is an important cause of hospital admission and mortality in tropical countries like India due to occupational hazard. (Eg. Agriculture) An estimated 15,000 – 20,000 people die each year from snake bite due to its complications like ARF, DIVC etc. (Oxford text book 5th edition 2005). Incidence of ARF following viper bites ranges 10 – 30% (Chugh et al) Most important of this it is an preventable cause of ARF in India . Hence several studies sprout regarding ARF and snake bite envenomation.

AIM AND OBJECTIVES OF THE STUDY:

Clinical presentation of patients with snake bite induced renal failure,Coagulation abnormalities. Outcome in renal failure due to snake bite.

MATERIALS AND METHODS:

Patients admitted in the emergency department, with snake bite induced renal failure and coogulation abnormalities were examined from 10/2023 - 3/2024 – Total number of patient is 50 Patients. History was elicited about type of snake, site of bite, time of bite, the native treatment taken, symptoms like oliguria, bleeding tendencies and cellulitis.
Site of snake bite is examined for presence of cellulitis , fang marks, bleeding from site of bite, local necrosis, gangrene.
Vital signs like pulse, blood pressure, respiratory rate, sub conjunctival hemorrhage, echymosis, bleeding from bite site, periorbital edema, ptosis, extra ocular movement, cardiovascular system, respiratory system, abdomen, centralb nervous system examined.

Coogulation profile – Clotting time, bleeding time, hemoglobin, total count, differential count, prothrombin time, Platelet count, fibrinogen, fibrin degradation products,Liver function tests - serum bilirubin, SGOT, SGPT,. Alkaline phosphatase, total protein , albumin, globulin,Renal Parameters – blood urea, serum creatinine, electrolytes Urine – Albumin, Sugar, RBC, WBC, Deposits,Serum Cholesterol,Serum Uric Acid,Serum Calcium,Electrocardiogram.

Statistical analysis was done using the statistical package for social sciences (SPSS).Different statistical methods were used as appropriate. Mean ± SD was determined for quantitative data and frequency for categorical variables. The independent t- test was performed on all continuous variables. The normal distribution data was checked before any t-test. The Chi-Square test was used to analyze group difference

for categorical variables.A p- value < 0.05 was considered significant.

RESULTS:

RENAL PARAMETERS

BL.UREA	NO. OF PTS	PERCENTAGE
< 50	9	18
50 – 75	13	26
76 – 100	10	20
101 – 150	10	20
150 – 200	2	4
201 – 250 (Maximum)	6	12

From the above table almost 60% of patients had blood urea levels in the range 50 – 150mg%.

CREATININE

CREATININE mg/dl	NO. OF PTS	PERCENTAGE
1 - 2	10	20
2 - 3	12	24
3.1 - 5	10	20
5.1 - 7	7	14
7.1 - 9	5	10
9.1 - 11	4	8
11.1 – 13 (Maximum)	2	4

CT PROLONGATION IN PATIENTS

CT	NO. OF PTS	%
< 10	12	24
11 - 15	17	34
16 - 20	14	28
> 20	5	10
> 1 Hr	2	4

From the above tables in our study 60% patients had creatinine levels in the range of 2 – 7mg%..

NO.OF DIALYSIS IN PATIENTS

NO. OF DIALYSIS	P.D	H.D	NO.OF PTS
0	-	-	13
1	30	8	22
2	2	3	2 + 3
3	-	2	2
4	-	2	2
8	-	8	1
13	-	13	2
22	-	22	1

22 Hemodialysis - In a case of patchy cortical necrosis proved by renal biopsy.

DISCUSSION:

Snake bite envenomation is a major cause of death and disability in developing countries particularly in India on South East Asia. The percentage of hospitalization of all snake bite cases is only 10% of total snake bite. Snake bite has seasonal variation the incidences more common during rainfall and summer. This is shown in a study of clinico epidemiological study in Nepal by Handsak and callor. In study of 50 cases of snake bites the number of cases and mortalilty both were more during March, April, September, and October.The commonest snake causing renal failure was viper (CHUGH et al, 1989) In the study only in 27 cases snakes were identified. 20 of them were vipers.

In a study conducted by Chugh (1989) mean age of snake bite victim was 27years. In this study age group of patients were between 6 years to 70 years. Commonest age group was between 21 – 50 years. Mean Age was 43 years. Males were commonly affected. In a study by Chugh out of 70 cases 58 cases were males. In this study 30 cases were male patients.

Commonest site of bite was lower limb in 2/3 of patients in a study by Silveria. In our study in 43 patients site of bite was lower limb(86%). Fang mark present in 47% of cases by Silveria et al (1982). In our study fang mark present in 72% of patients. The mean time delay between bite and Medical help was 3 hours by Silveria et al. Increased time interval was associated with increased mortality .

In our study 35% reached the hospital before 6 hours, 38% reached between 6 – 24 hours, 7% between 24 – 48 hours, 14% after 48 hours. Mortality was less in patients admitted before 6 hours and after 4 days associated with increased mortality.

Chugh et al (1989) reported earliest symptom after snake bite is pain and swelling. Blister and local necrosis were present in 40% of cases. Haematemesis, Hematuria, Malena was present. In our study pain swelling and cellulitis was present in all cases. Local necrosis, gangrene was present in 5 cases. Sub conjunctival hemorrhage in 16% cases. Haematemesis in 10%.
In a study of 70% patients Chuge reported Oliguria in 66 patients. In our study 64% of patients had Oliguria. Hematuria was present in 70%. 2 patients had Anuria. 2 patients had seizures. 1 patients had Hemiplegia. Updhuya and Moorthi has reported the cases of ischemic cerebro vascular accident, ARF, DIVC following snake bite.

In our study 7 patients had ptosis out of them only 3 recovered 7 patients had hypotension at time of admission only 4 patients survived. Chugh reported hypotension in 11 out of 70 patients. Hypotension was attributed to bleeding, hypovolemia, depression of medullary vasomotor center. Myocardial depression, Arteriolar vasodilatation and increase vascular permeability.

In the study mortality in patients admitted with hypotension was 64%. Mean no of ASV and dialysis required in these patients were more compared to other patients.Mean Blood urea level in study by Chugh 233 + 10. Mean creatinine level was 9.2

+ 3.4. In our study mean urea was 146.4. Mean creatinine was 6.6. There was no correlation between peak urea level and severity. This difference may be due to earlier intervention by renal replacement therapy.
There are many studies showing DIC in snake bite. Acharya, Kanna had reported DIC in 24 out of 50 cases. Chugh reported 56% of patients having DIC. Desilva also reported DIC following snake bite. In our study 38% of patients had prolonged clotting time. Prothrombin time was prolonged 50% of patients. APTT was prolonged in 64% patients. Patients with prolonged APTT required more number of dialysis and ASV required was more compared to other patients.

In our study 13 patients did not required dialysis. They were treated with fluid correction, antibiotics and conservative management. All other patients require peritoneal or hemodialysis. Mean dialysis required was 2. 1 patient required 22 hemodialysis. 13 patients required blood transfusion.

Duration of hospital stay was between 5 to 30 days among patients survived. 1 patient was hospitalized for nearly 3 months. According to Moorthy mortality was 10%. In our study mortality was also 10%. Patients presenting with bleeding tendency had increased mortality. Patients presenting with hypotension and coagulation abnormalities were also associated with increased mortality.
5 patients expired 3 cases received ASV before admission 2 cases received ASV after admission. One died 1 day after, 2 died 5 days after admission 1 died 6 days after admission, 1 died 11 days after admission.

CONCLUSION:

Seasonal variation was present in snake bite.Incidence and mortality more during January, February, March, April.Males were commonly affected, lower limb being commonest site of snake bite.Of the identified snakes viper bite was the commonest cause of acute renal failure following snake bite.ARF due to snake bite may be both Oliguric and Non – Oliguric of which Oliguric accounts for 90% and Non – Oliguric accounts for 10%.

Renal lesion due to snake bite may be of two types – of which Acute tubular necrosis accounts for 70 to 80%, acute cortical necrosis accounts for 20 to 30%.Oliguria which lasts for 4 to 15 days suggests the possibility of acute cortical necrosis which carries worst prognosis.Hypotension, Sub conjunctival hemorrhage, disorientation, Seizures, Hemiplegia, anuria, during presentation were associated with increased mortality.

Mortality was less in patients who came with in 6 hours. Mortality was more in patients who came after 6 hours - 4 days of snake biteMajority of patients had hypoalbuminemia and this was correlated with increased morbidity and mortality.Mortality rate was 10%. Early adequate dosage of ASV was associated with better prognosis. Early detection of renal failure and institution of dialysis was associated with better

outcome.Commonest cause of mortality was coagulation abnormalities.

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