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CLINICOPATHOLOGICAL SPECTRUM OF PATIENTS WITH SNAKE BITE AND ACUTE KIDNEY INJURY: A PROSPECTIVE INVESTIGATION AT A TERTIARY CARE MEDICAL CENTRE

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Abstract

Introduction: Acute kidney damage (AKI) is a significant complications and leading cause of death following snakebite. The development of nephropathy is largely explained by changes in hemodynamic brought on by direct nephrotoxicity including vasoactive mediators as well as cytokines. AKI is exacerbated by bleeding, hypotension, rhabdomyolysis, intravascular haemolysis, as well as disseminated intravascular coagulation (DIC). Healthcare professionals in must comprehend the unique difficulties presented by snakebite-induced AKI due to the geographic variety in snake species and the makeup of their venoms.

Aims/Objective: To investigate the clinco-pathological manifestations of AKI caused by snakebite.

Materials and Method: This was a prospective study conducted on 19 patients with snake bite and AKI. Every patient underwent a thorough clinical examination and history. To check for coagulation defects, a bedside 20-minute "whole blood clotting test (20WBCT)" had been performed. All patients underwent haematological as well as biochemical testing. The results of the kidney biopsies' histological analysis were also examined.

Results: Patients of snake bite with AKI had significantly greater incidence of leucocytosis, low platelet count, rise in urea/creatinine levels, hyperkalaemia, liver dysfunction and raised LDH levels ($p<0.0001$). Acute tubular necrosis (ATN) and acute interstitial nephritis (AIN) were found in greater than 40% of patients with AKI. There was 1 case of acute cortical necrosis.
Conclusion: Factors linked to death included a low serum albumin, an elevated WBC count, a high level of bilirubin, seizure or encephalopathy, pneumonia or ARDS complications, multi-organ failure, and requirement for ICU assistance. RCN was uncommon on renal histology, whereas ATN and AIN were prevalent.
Keywords: Snakebite, Acute Kidney Injury, Laboratory Parameters, Renal Histopathology.

INTRODUCTION:

In the world, snake bites are major public health concerns that significantly increase morbidity and mortality, especially in tropical regions. The World Health Organization now lists snakebite as a Neglected Tropical Disease [1]. An estimated 5 million people get bitten by poisonous snakes annually, resulting in more than 2 million envenomation, a minimum of 100,000 fatalities, a million amputations, and various other lifelong disabilities, based on WHO estimates [2]. Sub-Saharan Africa and Asia account for the majority of snakebite-related fatalities [1]. India has the highest annual rate of venomous snakebite fatality in the world, reported to be between 35,000 and 50,000 [1, 3]. For a variety of social, cultural, and economic reasons, the death rate from poisonous snakebites in India remains high [4].There are more than three thousand species of snakes in the world, and about 600 of those are thought to be poisonous. In south-east Asia, there are two major families (groups) of poisonous snakes: Elapidae, which have short, permanently erect fangs. The sea snakes, coral snakes, kraits, king cobras, as well as cobras are all members of this family. The large fangs of viperid snakes are erect when they strike, but they are ordinarily folded up towards the upper jaw. The pit vipers (Crotalinae) as well as regular vipers (Viperinae) are the two subgroups [5]. The so-called "Big 4" snakes of India such as the saw-scaled vipers (Echiscarinatus), the cobra (Najanaja), regular krait (Bungarus caeruleus), as well as russell's viper (Daboia russelli) are found all over the nation and are considered medicinally significant. [5, 6]. The neurological system, renal system, liver, cardiovascular system, respiratory system, the blood coagulation mechanism, vascular endothelium, including local effects at the bite site are the principal targets of envenomation [7]. Acute kidney damage (AKI) is a significant side effect and leading cause of death following a snakebite. AKI frequently occurs following a bite from a myotoxic or hemotoxic snake. Vasculitis, glomerulonephritis, interstitial nephritis, tubular necrosis, and cortical necrosis are examples of pathologic alterations in the kidneys. The development of nephropathy is largely explained by changes in hemodynamic brought on by direct nephrotoxicity including vasoactive mediators as well as cytokines. AKI is caused by renal ischemia that is exacerbated by bleeding, hypotension, rhabdomyolysis, intravascular haemolysis, as well as disseminated intravascular coagulation (DIC) [8]. According to the species of the snake along with the level of envenomation, these snakes can cause anywhere from 5% to 29% of AKI cases [9–11]. AKI can appear anywhere from several hours to 96 hours after the bite. After a snake bite, AKI typically lasts two to three weeks. One significant pathological consequence of AKI is tubular necrosis. Following a snake bite, prolonged AKI accompanied by oligo-anuria is suggestive of cortical necrosis and acute tubular necrosis linked to extra-capillary glomerulonephritis or interstitial nephritis [8].Healthcare professionals in areas where snakebites are prevalent must comprehend the unique difficulties presented by snakebite-induced AKI due to the geographic variety of snake species and the makeup of their venoms. Thus, in a tertiary care hospital in eastern India,

this study was carried out to investigate the clinco-pathological manifestations of AKI caused by snakebite.

MATERIALS & METHODS:
This was a prospective study conducted on 19 patients with snake bite and AKI at Rajendra Institute of Medical Sciences, Ranchi over a period of August 2022 to May 2023. The study was conducted after obtaining written informed consent from patients with snake bites and AKI as per recommendations of good clinical practice and the declaration of Helsinki.

Inclusion Criteria:
- Patients with a history of snake bites.
- Patients with clinical features of snake bite such as the presence of bite marks, cellulitis, neuropathy, or coagulopathy.
- Patients with diagnosis of AKI as per KDIGO criteria with rise in serum creatinine more than or equal to 0.3mg/dl in 48 hours or more than or equal to 1.5 times of baseline [15].
- Patients with any indication of renal replacement therapy.

Exclusion Criteria:
- Patients with pre-existing renal disease.
- Patients with hypertension or type 2 diabetes mellitus.
- Patients with recent exposure to nephrotoxic drugs.

Sample Size: Consecutive sampling was done to screen 100 patients with snake bites. After screening for eligibility criteria, 19 patients with snake bite and AKI were included in the study.

Methodology:
Every patient underwent a thorough clinical examination and history. To check for coagulation defects, a bedside 20-minute "whole blood clotting test (20WBCT)" had been performed. All patients underwent haematological as well as biochemical testing. The level of albumin serum bilirubin, serum AST (aspartate aminotransferase), Serum ALT (alanine aminotransferase), alkaline phosphatase, CK (creatine kinase), LDH (lactate dehydrogenase), leukocyte count, platelet count, blood urea, creatinine levels in the serum, the serum electrolytes of potassium and sodium, and ABG (arterial blood gas analysis) results were collected. Additionally, the frequency of anaemia (haemoglobin less than 12 g/dL), leucocytosis, thrombocytopenia, hyperkalaemia, Hypoalbuminemia, liver dysfunction, and severe metabolic acidosis (pH 7.2), haemolysis, as well as rhabdomyolysis were noted. For analysis, the best or peak values for these laboratory variables at the time of admission were employed. Recorded were the complications observed throughout the hospital stay, including low blood pressure, high blood pressure,pulmonary infection, ARDS (acute respiratory distress syndrome), myocarditis, MI (myocardial infarction), bleeding from the gastrointestinal tract, seizures and encephalopathy, DIC, or multi-organ failure (MOF).Using WHO recommendations for "the clinical management of snakebites in Southeast Asia", the study classified the clinical symptoms and sequelae of snake envenomation [5].The main methods of detecting coagulation defects were 20WBCT or apparent spontaneous systemic bleeding that was seen at this hospital or reported by the referring healthcare facility. Ptosis, external ophthalmoplegia, muscular paralysis, incapacity to raise the head, and other symptoms are indicative of neurotoxicity. An arterial SBP (systolic blood

pressure) of less than 90 mmHg was referred to as hypotension and a blood pressure reading of 140/90 mmHg as hypertension.An AST and ALT of more than 60 IU/L were considered to indicate hepatic impairment. Anaemia, jaundice, reticulocytosis, an aberrant peripheral blood smear, and elevated serum LDH levels are indicative of intravascular haemolysis. Elevated serum CK levels 5 times or higher than normal, along with an indicative clinical presentation and the absence of heart and brain damage, were the hallmarks of rhabdomyolysis [11,12].

We employed Yu et al.'s recommended screening test criteria to detect disseminated intravascular coagulation [13]. After three weeks, patients who were still oligo-anuric as well as whose serum creatinine had not decreased to a satisfactory level underwent kidney biopsies, which were analyzed using light as well as immunofluorescence microscopic examination. The results of the kidney biopsies' histological analysis were also examined.

Statistical Analysis: Data from patients bitten by a snake were entered in tabular form using Microsoft Excel 2010 and then transferred to GraphPad version 8.4.3 for further statistical analysis. Categorical data such as age, gender, bite site, and incidence of disorders were compared using chi-square or Fisher's exact test. Continuous data such as haematological and biochemical parameters were compared using an unpaired t-test to determinethe statistical significance of difference at $p<0.05$.

RESULTS:
Out of 100 patients bitten by snake screened, 19 were diagnosed with AKI.

Table 1: Comparison of Baseline Demographic and Clinical Characteristics between Patients with or without AKI

Parameters	Number of Patients (%)		P-Value
	Patients with AKI (n = 19)	Patients without AKI (n = 81)	
Age Group in Years			
<15	3 (15.79)	15 (18.52)	0.95
15-50	14 (73.68)	57 (70.37)	
>50	2 (10.53)	9 (11.11)	
Gender			
Male	17 (89.47)	70 (86.42)	>0.99
Female	2 (10.53)	11 (13.58)	
Bite Site			
Upper Limb	3 (15.79)	15 (18.52)	>0.99
Lower Limb	16 (84.21)	66 (81.48)	
Oliguria	16 (84.21)	13 (16.05)	<0.0001
Haematuria	11 (57.89)	29 (35.80)	0.11
Cellulitis	3 (15.79)	4 (4.94)	<0.0001
Coagulation defect	18 (94.74)	71 (87.65)	0.68

Most of the patients in our study (>70%) were from working age group (15-50). Most of them (>85%) were males. Bite site was in lower limb in more than 90% of the patients. There were significantly more incidence of oliguria and cellulitis in patients with AKI as compared to those

without AKI (P<0.0001). Incidence of haematuria and coagulation impairment was also higher but the difference was not significant at this sample size.

Table 2: Comparison of Haematological and Biochemical Parameters between Patients with or without AKI

Parameters	Value in Mean ± SD		P-Value
	Patients with AKI (n = 19)	Patients without AKI (n = 81)	
Haemoglobin in g/dl	9.58 ± 2.49	10.23 ± 3.01	0.38
Leucocyte count/mm^3	14.86 ± 3.35	12.38 ± 2.86	0.001
Platelets/mm^3	119.13 ± 17.26	133.47 ± 20.19	0.005
Urea in mg/dl	172.57 ± 23.14	69.96 ± 9.52	<0.0001
Creatinine mg/dl	7.31 ± 1.95	1.25 ± 0.29	<0.0001
Sodium in mEq/L	134.42 ± 6.23	137.78 ± 7.06	0.06
Potassium in mEq/L	5.15 ± 0.93	4.25 ± 0.82	<0.0001
Albumin g/dl	3.19 ± 0.37	3.79 ± 0.51	<0.0001
Bilirubin in mg/dl	1.49 ± 0.24	0.89 ± 0.13	<0.0001
AST in U/L	148.76 ± 18.62	73.74 ± 6.59	<0.0001
ALT in U/L	105.57 ± 12.39	56.37 ± 5.86	<0.0001
Alkaline Phosphatase in U/L	95.66 ± 9.85	69.98 ± 8.86	<0.0001
LDHin U/L	1161.32 ± 198.59	765.23 ± 101.39	<0.0001

Patients of snake bite with AKI had a significantly greater incidence of leucocytosis, low platelet count, rise in urea/creatinine levels, hyperkalaemia, liver dysfunction, and raised LDH levels (p<0.0001).

Table 3: Comparison of Incidences of Haematological and Biochemical Disorders between Patients with or without AKI

Parameters	Number of Patients (%)		P-Value
	Patients with AKI (n = 19)	Patients without AKI (n = 81)	
Anaemia (Hb<12 g/dl)	16 (84.21)	49 (60.49)	0.06
Leucocytosis	15 (78.95)	39 (48.15)	0.02
Thrombocytopenia	9 (47.37)	23 (28.4)	0.17
Hyperkalemia	5 (26.32)	7 (8.64)	0.046
Severe metabolic acidosis	8 (42.11)	11 (13.58)	0.008
Hepatic dysfunction	8 (42.11)	26 (32.1)	0.43
Hypoalbuminemia	12 (63.16)	34 (41.98)	0.13
Hemolysis	17 (89.47)	62 (76.54)	0.35
Rhabdomyolysis	14 (73.68)	55 (67.9)	0.78

Patients of snake bite with AKI had significantly greater incidence of leucocytosis, low platelet count, rise in urea/creatinine levels, hyperkalaemia, hypoalbuminemia, hepatic dysfunction and raised LDH levels (p<0.0001).

Table 4: Kidney Biopsy Results in Patients with AKI (n =19)

Findings	Number of Patients	Percentage of Patients (n = 19)
Acute tubular necrosis	9	47.37
Acute interstitial nephritis	8	42.11
Acute cortical necrosis	1	5.26

Acute tubular necrosis (ATN) and acute interstitial nephritis (AIN) was found in greater than 40% of patients with AKI. There was 1 case of acute cortical necrosis.

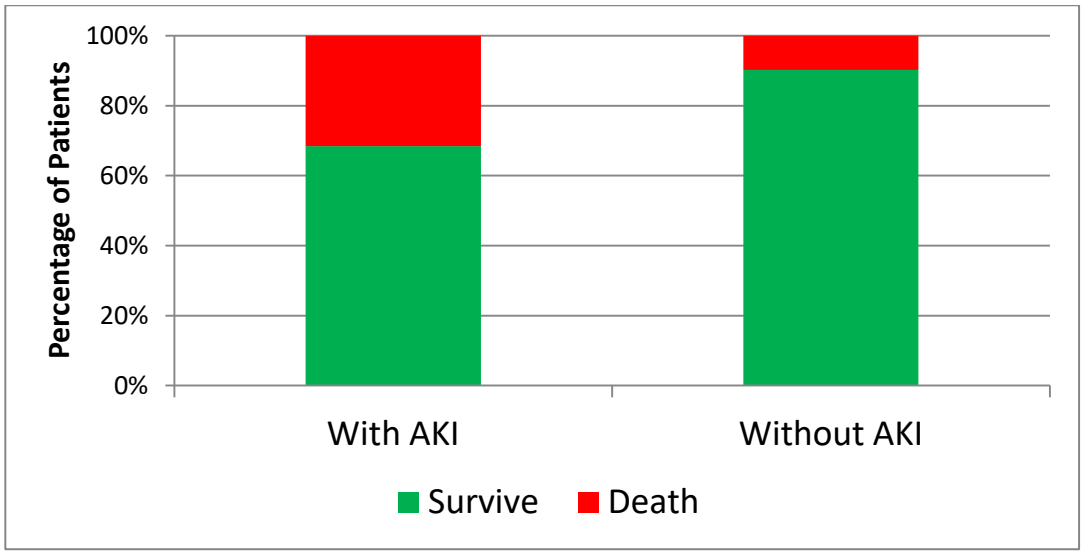

Figure 1: Comparison of Outcome between Two Groups

DISCUSSION:

In various studies, the symptoms of oliguria have been reported to be 13.8%-100%[9,11, 14-21] and hematuria/black colored urine 24.6%-80%[9,11,14,16,18,19,22] as compared to that of 84.21% and 57.89% respectively in our study. Cellulitis was 39%-98.7%[14,17-21] as compared to the15.79% observed in our study. Bleeding/coagulopathy which is a major symptom of systemic viper poisoning was observed in 94.74% of patients as compared to 27.8%-65.8% in other studies of snake bite-induced AKI[11, 14, 17-22].Among laboratory investigations, hemoglobin ranged from 7-13.3 g/dL in other studies[9, 13, 14, 17] as compared to 9.58 g/dL in this study, WBC count ranged from 11.5-19.3 × 103/mL [15, 20, 22] as compared to 14860/mL in current study, serum urea 85-233 mg/dL [9, 14, 15, 17, 18] as compared to 172.57 mg/dL and serum creatinine 2-9.2 mg/dL[9, 14, 15, 17, 18] as compared to 7.31 mg/dL in this study have been reported. Intravascular hemolysis has been reported in various studies to be 13.8%-54%[9, 14, 18], and thrombocytopenia 9.7%-60%[9, 14, 15, 21], as compared to that of 89.47% and 47.37% respectively in our study.Rhabdomyolysis is well well-known cause of AKI [12]. Most of the studies of snake bite-induced AKI have only looked at the hematological and coagulation profile[9, 14-23]. Every AKI patient in our study had their blood and rhabdomyolysis examined for signs of hemolysis. Pinho et al. reported that after a viper bite, rhabdomyolysis was seen in

73.68% of patients, whereas AKI was detected in 100% of cases. [11] Some of them may have missed it since they arrived at our facility later than expected. The majority of our patients (over 60%) experienced both rhabdomyolysis as well as hemolysis. Hemoglobinuria along with myoglobinuria both have a role in the onset of AKI. In those participating in our study, rhabdomyolysis as well as intravascular hemolysis were the primary causes of AKI. The risk factors associated with the development of AKI in snake bite are native treatment, bite-to-needle time more than 2 h, age, a bite to hospital/ASV time, black or brown urine, bleeding manifestations, hypotension/shock], cellulitis, regional lymphadenopathy, abdominal pain/tenderness and vomiting, prolonged bleeding time, prolonged prothrombin time, low hemoglobin, high WBC count, low serum albumin, and a high serum bilirubin, CK > 2000 U/L, albuminuria, intravascular hemolysis, DIC, complications of septicemia and ARDS, and a longer DOHS [11, 14-18]. ATN along with patchy or diffuse RCN are the renal abnormalities of clinical importance in envenomed patients. Rare cases of papillary necrosis, interstitial nephritis, as well as glomerulonephritis have been documented. Seventy to eighty percent of patients with AKI have tubulointerstitial lesions, primarily ATN [9]. Early biopsies show intra-tubular pigment granular casts and other morphological signs of extensive acute tubular damage. There may be interstitial edema along with acute tubulointerstitial nephritis of varying degrees. The tubular epithelium is seen to be regenerating in later biopsies [9].High proportion (47.37%) of patients subjected to kidney biopsy had ATN in our study. In addition, 42.11% of them had associated findings of mild to moderate AIN.Although AIN following a snake bite was thought to be uncommon, more recent research indicates that it is not [24, 25]. 5.7%–11.9% of AKI due to snakebite were caused by AIN. At the time of presentation, each patient had severe envenomation, a high ASV demand, and protracted renal failure [24, 25]. A mixed infiltration consisting mostly of lymphocytes with varying amounts of other cells, such as eosinophils, neutrophils, and plasma cells, was discovered during the kidney biopsy. Although the exact cause of this pathology's emergence is unknown, direct venom-related effects may contribute to the growth of interstitial inflammation through the actions of several cytokines, mediators, as well as adhesion molecules [24, 25]. It was discovered that corticosteroid therapy could reverse AIN [40]. However, it was shown that the majority of patients experienced CKD during follow-up, making the long-term outcome worse than it was for those without AIN. In our investigation, AIN was found in 45 percent of renal biopsies overall. In our study, one patient had just moderate AIN on biopsy, and after receiving corticosteroids, she recovered fully. Comparing ATN versus AIN patients, there was no variation in their clinical picture. Kidney biopsy should be performed on patients who are still oligoanuric and whose creatinine levels in the serum did not drop significantly after three weeks. In these patients, the kidney biopsy offers therapeutic, prognostic, as well as diagnostic relevance. It is likely that patients having ATN or ATN linked to AIN will recover. Steroid treatment may be administered to patients with AIN undergoing renal biopsy in order to speed up their recuperation and stop fibrosis from developing. The prognosis of renal recovery is poor in RCN patients. Following snake envenomation, CKD is a serious AKI consequence that is likely anticipated by the duration of RRT [44]. All but two of the patients who made it through the current study's short-term follow-up or hospital stay experienced full renal recovery; however, the absence of such follow-up precluded the assessment of the patient's long-term outcomes.

CONCLUSION:

Factors linked to death included low serum albumin, an elevated WBC count, a high level of bilirubin, seizure or encephalopathy, pneumonia or ARDS complications, multi-organ failure, and the requirement for ICU assistance. RCN was uncommon on renal histology, whereas ATN and AIN were prevalent.AKI is a serious side effect of a snake bite that can be fatal. AKI from a snake bite is mostly caused by intravascular hemolysis and rhabdomyolysis, which are frequent occurrences. Alkaline diuresis along with prompt ASV administration may aid in the prevention of AKI. For a positive patient outcome, appropriate supportive treatment following ASV delivery is crucial. RCN shows poor renal recovery, while ATN or AIN are more likely to recover.

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