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Clinical and laboratory parameters associated with Acute Kidney Injury in patients with Viperine Snake Bite: A Prospective Observational Study in Central Odisha

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ABSTRACT

Aim: The aim of the present was to determine the clinical and laboratory parameters associated with AKI in patients with viperine snake bite in Central Odisha.

Hypothesis: We hypothesize that there is a significant association between certain clinical and laboratory parameters and the development of AKI in patients with viperine snake bite in Central Odisha.

Materials & methods: The General Medicine department of a tertiary teaching hospital in Central Odisha was the site of this prospective analytical investigation. This study was conducted between September 2020 and August 2021. In our study a total of 262 snake bite patients were scrutinized, out of which 137 snake bites were of neurotoxicity, 65 snake bites were of Viper, and 60 bites were of normal or dry bites. The study included 65 patients who were of viper bites. Out of 65 patients, 40 were male and 25 were female. Patients were included based on the inclusion and exclusion criteria.

Conclusion: The presence of DIC and delay in hospital arrival following a snakebite are predictors of poor outcomes in patients with viperine snake bites developing AKI. Mortality can be prevented by early hospital arrival and administration of ASV.

Keywords: *Acute Kidney Injury; Viperine snake bite; D-dimer levels; anti-snake venom; whole blood clotting test.*

1. INTRODUCTION

Acute Kidney Injury (AKI) is a common complication in patients with viperine snake bite, and early identification of risk factors can help in preventing the development of AKI. The incidence of viperine snake bite and the associated risk of AKI is a significant health concern [1-4]. Previous studies have identified several risk factors for AKI in patients with viperine snake bite, including age, sex, and co-morbidities [5-8]. However, there is a lack of prospective, observational studies on the clinical and laboratory parameters associated with AKI in patients with viperine snake bite in Central Odisha. This study aims to fill this knowledge gap by conducting a prospective, observational study on the clinical and laboratory parameters

associated with AKI in patients with viperine snake bite in Central Odisha. The research question of the present study is what are the clinical and laboratory parameters associated with AKI in patients with viperine snake bite in Central Odisha? Hence, the aim of the present was to determine the clinical and laboratory parameters associated with AKI in patients with viperine snake bite in Central Odisha. We hypothesize that there is a significant association between certain clinical and laboratory parameters and the development of AKI in patients with viperine snake bite in Central Odisha.

2. METHODS

The General Medicine department of a tertiary teaching hospital in Central Odisha was the site of this prospective analytical investigation. This study was conducted between September 2020 and August 2021. In our study a total of 262 snake bite patients were scrutinized, out of which 137 snake bites were of neurotoxicity, 65 snake bites were of Viper, and 60 bites were of normal or dry bites. The study included 65 patients who were of viper bites. Out of 65 patients, 40 were male and 25 were female. Patients were included based on the inclusion and exclusion criteria. The study was approved by the Institutional Ethics Committee of SCB Medical College and Hospital, Cuttack, as evidenced by the letter number ECR/84/Inst/OR/2013/RR-20. The inclusion criteria were a clear history of viperine snake bites, as shown by the victims' and their relatives' identification of the snakes, local cellulitis or blood pouring from the bite site, or a positive 20-minute whole blood clotting test (WBCT). The inclusion criteria were adults who were at least 18 years old and willing to consent. Exclusion criteria were chronic liver or kidney disorders and immunocompromised persons.

Sample size calculations

Sample size was estimated based on the reported incidence of renal involvement in snakebite envenomation which ranges from 1.4 – 28.0 %.[6,22,23] Taking the incidence of renal dysfunction in viperine snake bites to be approximately 3% and an absolute precision of 5%, the minimum sample size was calculated to be 45. The nonparticipation rate was 10%, and the required final sample size was calculated to be 50.

Sampling method

Consecutive sampling of the viperine snake bite cases was performed for recruitment of cases in the medicine wards, where the admitted cases were found during weekly visits by the investigator. It took approximately 7 months to complete recruitment.

Methodology

To detect viperine snake bite and learn about any related comorbidities, including chronic kidney disorders, chronic liver diseases, and immunocompromised states , all individuals with a history of snakebite underwent complete clinical examinations. Participants in the study provided approximately 10 ml of blood, which was taken in a simple vial and tested for clotting after 20 minutes (20 minute Whole Blood Clotting Test). Another 10 ml of blood was collected and sent for the following tests: complete blood count, random blood sugar, serum urea and creatinine levels, serum sodium and potassium levels, D-dimer levels, PT, aPTT, and INR. On alternate days, serum urea and creatinine assays were repeated. If the results of the 20-minute whole blood clotting test were positive or if there was any sign of local cellulitis or bleeding at the bite site, 10 vials of anti-snake venom (ASV) [100 ml] were delivered over the course of an hour while being watched for any unusual side responses. No adverse reactions were observed in our study population. After six hours, repeat ASV dosages based on 20 WBCT scans were administered. Repeat doses of ASV were administered if 20WBCT had not clotted after six hours of ASV treatment. As shown in table no., we administered ASV to our research population at doses as low as 10 vials and as high as 40 vials, with an average of 20 vials. To avoid an allergic reaction to ASV, chlorpheniramine was administered first. The timing, location, time of hospital arrival, and time of ASV administration were noted at the time of hospitalization. We then analyzed and condensed all the participants' inquiry reports into Table No. 1.

Calculation of sample size

Based on the reported incidence of renal involvement in snakebite envenomation, which ranges from 1.4 to 28.0%, a sample size was estimated.[6,22,23] The minimum sample size was calculated to be 47 based on the assumption that the incidence of renal impairment in snakebites is approximately 3%.The final sample size was calculated to be 52, with a non-participation rate of 10%.

Statistical Analysis

IBM SPSS version 21 was used to analyze the data after they were entered using Microsoft Excel 2013 and the Statistical Package for Social Sciences. Data were examined for normality using the Shapiro-Wilk test, Z-score (skewness and kurtosis), and visual inspection with histograms and Q-Q plots. If the data had a Z-score value (skewness/SE of skewness) of less than 3.29, a normal bell-shaped curve on the histograms, and a p-value > 0.05, as determined by the Shapiro-Wilk test, the data were considered to be normally distributed. Descriptive statistics were used, and the results are expressed as median (interquartile range) for non-normally distributed data for continuous variables and frequency(percentage) for categorical variables. Pearson's Chi-square test was used for categorical variables. Non-parametric tests, such

as the Mann-Whitney U test, were used for continuous variables of two independent samples. Renal function tests evaluated on days 1, 3, and 5 were correlated with the duration of time elapsed from the snake bite to reaching the hospital using Spearman rank correlation and Spearman's rho correlation coefficient. Kaplan-Meier survival analysis was performed for the outcome of the study, and a time–to–event survival graph was plotted. Line charts were constructed using Microsoft Excel for graphical representation of serum urea and creatinine levels measured on 3 alternate days.

3. RESULTS

During the study period, 262 snakebite cases were admitted to the medical ward, of which 137 were neurotoxic and 60 were normal. The study consisted of 65 subjects (viperine envenomation cases). Male patients comprised 61.5% (n = 40) of the study population, and the rest were female. Our patients ranged in age from 27 to 53.5, with a median age of 40 years. Of the 65 patients, 54 (83.1%) had a snakebite during the day, while 11 (16.9%) had a snakebite at night. Right-foot bites occurred most frequently (n=26/65), followed by left-foot bites (n=22/65), right-leg bites (n=6/65), and left-leg bites (n=5/65). The average amount of time needed to reach the hospital was 60 min (range: 45–120 min). At the snakebite site, cellulitis affected every patient. On days 1, 3, and 5, serum urea levels were 45(39-50), 46(40-65), and 36(30-50), respectively. Similar to this, the serum

The creatinine levels measured on days 1, 3, and 5 were 1, 2, and 0.8(0.6-1.8) correspondingly. The median (range, 20–30) number of ASV vials required was 20. Twenty% of patients (n=13) had oliguric symptoms. Hematuria was observed in 33.8 % (n = 22) of the patients. Acute renal damage was noted in 30.8% (n=20) of the patients. Among the study participants, 10.8% died (n = 7).

Table 1: Comparison of different parameters among the surviving and deceased

Parameters	Survived (n=58)	Deceased (n=7)	P Value
Age (Years)	40 (27 – 52)	46 (26 – 60)	0.783
Male Female	38 (65.6) 20 (34.4)	2 (28.6) 5 (71.4)	 0.097
Time to reach hospital (mins)	60 (30 – 120)	300 (240 – 600)	<0.001
ASV vials received	20 (20 – 25)	40 (40 – 40)	< 0.001
Hb	12 (12 – 13)	12 (10 – 13)	0.142
TLC	9350 (8300 – 10650)	18500 (17400 – 18600)	< 0.001
TPC	1.8 (1.8 – 2)	1.8 (1.5 – 1.9)	0.283
INR	1 (0.9 – 1.1)	1.2 (1.1 – 1.2)	0.001
D – Dimer	225 (150 – 325)	3000 (1200 – 10000)	< 0.001
Urea- day- 1	40 (38 – 50)	60 (60 – 80)	< 0.001
Urea – day -3	44 (40 – 54.5)	96 (90 – 106)	< 0.001
Urea day – 5	34 (30 – 40)	130 (120 – 138)	< 0.001
Creatinine day – 1	1 (0.8 – 1.1)	2.5 (2.4 – 3.1)	< 0.001
Creatinine day – 3	2 (1.8 – 2.7)	8 (7 – 9)	< 0.001

Creatinine day - 5	0.8 (0.6 – 1)	8 (7 – 9)	< 0.001
Hematuria Yes No	 15 (25.9) 43 (74.1)	 7 (100) 0 ())	 < 0.001
Oliguria Yes No	 6 (10.3) 52 (89.7)	 7 (100) 0 (0)	 < 0.001
AKI Present Absent	 13 (22.4) 45 (77.6)	 7 (100) 0 (0)	 < 0.001

Table 2: Comparison of different parameters (Interquartile Range) among the patients with AKI and without AKI (n=65)

Parameters	**AKI Present (n=20)**	**AKI Absent (n=45)**	**P Value**
Age (Years)	41.5 (24.5 – 51.5)	40 (28 – 59)	0.508
Male Female	13 (65) 7 (35)	27 (60) 18 (40)	 0.787
Time to reach hospital (mins)	180 (120 – 240)	60 (30 – 60)	<0.001
ASV vials received	30 (22.5 – 40)	20 (15 – 20)	< 0.001
Hb	12 (11.3 – 13)	13 (12 – 13)	< 0.001
TLC	16050 (11600 – 18575)	8600 (8300 – 9600)	0.194
TPC	1.8 (1.7 – 1.9)	1.9 (1.8 – 2.05)	< 0.001
INR	1.1 (1.06 – 1.2)	1.2 (0.9 – 1.06)	< 0.001
D – Dimer	700 (525 – 2500)	200 (150 – 300)	< 0.001
Urea- day- 1	60 (50 – 66)	40 (37.5 – 47.5)	< 0.001
Urea – day -3	85 (70 – 104.5)	44 (38 – 48)	< 0.001
Urea day – 5	60 (50 – 66)	33 (28 – 37)	< 0.001
Creatinine day – 1	2.3 (1.9 – 2.6)	0.9 (0.8 – 1)	< 0.001
Creatinine day – 3	5.5 (3.1 – 7.4)	1.8 (1.7 – 2)	< 0.001
Creatinine day - 5	2.2 (1.9 – 2.6)	0.7 (0.5 – 0.9)	< 0.001
Hematuria			
Yes	16 (80)	6 (13.3)	< 0.001

No	16 (80)	6 (13.3)	
Oliguria			
Yes	13 (65)	0 (0)	< 0.001
No	7 (35)	45 (100)	
Outcome			
Survived	13 (65)	45 (100)	< 0.001
Deceased	7 (35)	0 (0)	

Table 3: Spearman rank correlation of different parameters (Spearman's rho) w.r.t time of bite to ASV received (n =65).

Parameters		**Time to reach hospital**
Age	Correlation coefficient	0.205
	P value	0.101
ASV vials received	Correlation coefficient	0.753
	P value	< 0.001
Hb	Correlation coefficient	0.306
	P value	0.0103
TLC	Correlation coefficient	0.567
	P value	< 0.001
TPC	Correlation coefficient	- 0.236
	P value	0.059
INR	Correlation coefficient	0.634
	P value	< 0.001
D - dimer	Correlation coefficient	0.682
	P value	< 0.001
Urea day - 1	Correlation coefficient	0.511
	P value	< 0.001

Urea day - 3	Correlation coefficient	0.546
	P value	< 0.001
Urea day - 5	Correlation coefficient	0.694
	P value	< 0.001
Creatinine day - 1	Correlation coefficient	0.794
	P value	< 0.001
Creatinine day - 3	Correlation coefficient	0.625
	P value	< 0.001
Creatinine day - 3	Correlation coefficient	0.728
	P value	< 0.001

Figure 1: Line chart demonstrating serum urea levels measured on day- 1,day-3 and day-5 (N=65)

Figure 2: Line chart demonstrating serum creatinine levels measured on day-1,day-3 and day-5 (N=65)

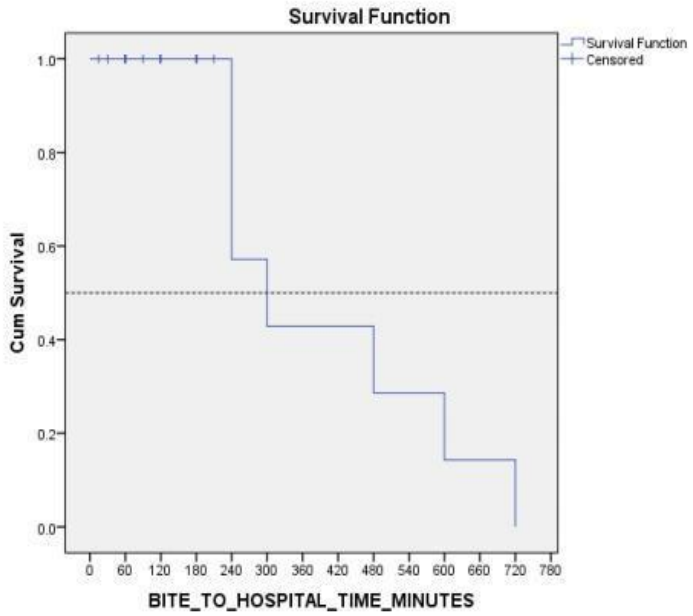

Figure 3: Kaplan Meier survival plot

Table 1 compares the variables with respect to the study outcomes. Of the 65 patients included, 58 survived the event, whereas 7 patients did not survive. The median age of the patients who survived was 40 years (27-52) and that of those who did not survive was 46 years (26-60). In the deceased group (n=7), the majority of the patients were female (n=5/7) compared to males (n=2/7). The median duration of time elapsed in reaching the hospital was 60 minutes (30-120) in the survival group and 300 minutes (240-600) in the deceased group with a p-value of < 0.001. Serum urea measured on day-1, day-3, and 5 in the survival group were 40 mg (38-50), 44 mg/dl (40-54.5), and 34 mg/dl (30-40), respectively. In the deceased group, the serum urea levels were 60 mg/dl (60-80), 96 mg/dl (90-106), and 130 mg/dl (120-138) on day-1. Day-

3, and day-5, respectively. Likewise, the serum creatinine values among the survived vs. deceased group on day-1, day-3, and day-5 were 1 vs. 2.5 mg/dl, 2 vs. 8 mg/dl, and 0.8 vs. 8 mg/dl, respectively with p-value of < 0.001. The median number of ASV vials received by the surviving group was 20, compared to 40 vials in the deceased group ($p < 0.001$). All patients who did not survive (n=7) had oliguria, hematuria, or acute kidney injury. However, among the surviving patients, 10.3% had oliguria (n=6/58), 25.9% had hematuria (n=15/58), and 22.4% had acute kidney injury (n=13/58).

Table 2 compares these variables with respect to the presence of acute kidney injury. Of the 65 patients included, 20 (30.8%) had acute kidney injury. The median age of the patients with AKI was 41.5 years (24.5-51.5). Among the patients with AKI group (n=20), majority of the patients were male (n=13/20) as compared to females (n=7/20). The median duration of time elapsed in reaching the hospital was 180 minutes (120-240) in the AKI group and 60 minutes (30-60) in the non-AKup with ($p < 0.001$). The serum urea measured on day-1, day-3, and 5 in the AKI group vs. non-AKI group were 60 mg (50-66) vs. 40 (37.5-47.5), 85 mg/dl (70-104.5) vs. 44 (38-48), and 60 mg/dl (50-124.5) vs. 33 (28- 37), respectively. Similarly, the serum creatinine values in the AKI vs. non-AKI group on day-1, day-3, and day-5 were 2.3 vs. 0.9 mg/dl, 5.5 vs. 1.8 mg/dl, and 2.2 vs. 0.7 mg/dl, respectively, with p p-value < 0.001. The median number of ASV vials received by the AKI group was 30 vials (22.5-40) as compared to 20 vials (15-20) in the non-AKI group ($p < 0.001$). Among patients with AKI, 65% had oliguria (n=13/20), 80% had hematuria (n=16/20), and 65% did not survive (n=13/20).

Table 3 demonstrates the correlation of different parameters with the time elapsed to reach the hospital. The correlation coefficient and Spearman's rho for serum urea measured on day-1, day-3, and day-5 against the time to reach hospital were 0.511 ($p < 0.001$), 0.546 ($p < 0.001$), and 0.694 ($p < 0.001$), respectively. Likewise, Spearman's rho for serum creatinine measured on day-1, day-3, and day-5 against time to reach hospital were 0.794 ($p < 0.001$), 0.624 ($p < 0.001$), and 0.728 ($p < 0.001$), respectively. The number of ASV vials required was positively correlated with the time to reach the hospital (Spearman's rho = 0.759, $p < 0.001$).

Figure 1 depicts a line chart showing the median values of serum urea levels measured on day-1, day-3, and day-5 among the survivors and deceased groups. There was a linear increase in the serum urea on day-1, day-3, and day-5 (60, 96, and 130 mg/dl) in the deceased group. However, in the survival group, there was a minimal increase in serum urea level on day-3 prior to a decrease in serum urea levels on day-5 (40, 44, and 34 mg/dl).

Figure 2 depicts a line chart showing the median values of serum creatinine levels measured on day-1, day-3, and day-5 among the survivor and deceased groups. There was a steep increase in the value of serum creatinine from day-1 to day-3, and thereafter remained static from day-3 to day-5 (2.5, 8, and 8 mg/dl) in the deceased group. However, in the survival group, there was a mild increase in serum creatinine level on day-3 prior to a decrease on day-5 (1, 2, 0.8 mg/dl).

Figure 3 shows the Kaplan-Meier survival plot. According to the survival analysis, the probability of survival following a viperine snake bite decreases to less than 50% if the time to reach the hospital is ≥5 h.

4. DISCUSSION

A prospective observational study in Central Odisha examined clinical and laboratory variables related to acute kidney injury in patients with viperine snake bites. In tropical regions, where farmers, plantation workers, environment workers, and those working barefoot are at risk for snakebites, snakebites are a medical emergency [1,2]. According to global estimates, approximately 20, 000 people die from snakebite envenomation each year [2,3]. Most snakebite sufferers seek local quacks and adopt outdated treatments, which can have disastrous results. The primary reason for this was the lack of healthcare facilities at the appropriate time. Even when accessible, primary care doctors sometimes lack the expertise to conduct complete examinations and manage a snakebite, leading to unfavorable results.

Acute kidney injury (AKI), with a death incidence of 8.0-39%, is the most prevalent snakebite-related injury in the state of Odisha, where Russell and Pit Vipers are two common vasculo-toxic snakes.[4- 15] The length of time between a snakebite and hospital admission, the amount of time between a snakebite and the administration of anti-snake venom (ASV), presence of cellulitis at the bite site, incoagulable blood on a 20-minute whole blood clotting test (WBCT), prolonged prothrombin time (PT), and dark or brown urine are some of the factors linked to AKI [4,5,6,7,8]. Although the precise pathogenesis of AKI after a vasculo-toxic snake bite is unknown, certain factors, including hypotension, intravascular hemolysis, disseminated intravascular coagulation (DIC), microangiopathic hemolytic anemia, and direct nephrotoxicity of the venom, which manifests as tubular and cortical necrosis, contribute to AKI [4,11]. Numerous proteins found in vascular-toxic snakes, such as Russell's viper venom factor V activating serine proteinase (RVV-V) and Russell's viper venom factor X activator (RVV-X), interact with clotting cascade components and the fibrinolytic pathway to cause DIC [4,16,17]. Additionally, there is evidence that Phospholipase A2, an enzyme found in almost all vasculo-toxic snakes, directly induces hemolysis by hydrolyzing phospholipids in red blood cell membranes [4,18].

Patients with vasculo-toxic snake bites can have a wide variety of clinical symptoms, from modest local symptoms such as pain and swelling to severe systemic manifestations such as DIC, oliguria, hypotension, and severe hemorrhage, such as

hematemesis, melena, hemoptysis, hematuria, and hematoma [4,19,20]. When a patient exhibits one or more of the following symptoms following a proven or suspected vasculo-toxic snake bite, anti-snakebite treatment is advised. Hemostatic abnormalities, such as spontaneous systemic bleeding that occurs away from the site of the bite, incoagulable blood on 20 WBCT, an INR greater than 1.2, a prothrombin time greater than 4-5 seconds longer than the control value, or thrombocytopenia (TPC less than 1.0 lakh/ cubic millimeter). Oliguria, defined as the production of less than 400 ml of urine in 24 hours, or anuria, which is defined as producing no urine in 24 hours; a spike in serum creatinine of more than 0.3 mg/dL within 48 hours or an increase of more than 1.5 the baseline are all symptoms of acute kidney injury. Hemoglobinuria manifests as a dark brown urine [6,8,21]. ASV should be administered at an initial dose of 8–10 vials (100 ml) over the course of an hour. Recurring doses were based on the six-hour guideline. Thirty to forty vials were advised [8,21]. The objective of this study was to evaluate the clinical and laboratory factors that contribute to acute kidney damage after vasculo-toxic snake bites.

Snakebite envenomation is a major public health problem in tropical and subtropical countries, particularly Southeast Asia [6,24,25,26]. It has been reported that approximately 1.4-28.0% of snakebite envenomation is associated with renal involvement, which manifests as hematuria, oliguria, and AKI [22,23]. Renal involvement is commonly observed among patients with vasculo-toxic snake bites, particularly Russel's vipers [7,8,27,28]. In our study AKI was present in 30.8% which is comparable to other reported studies in India [22,23]. The clinical presentation varies from local symptoms, such as cellulitis of the bitten part, to severe systemic manifestations, including oliguria, hematuria, and DIC. In our study, cellulitis was present in all patients, oliguria was observed in 20% of patients, hematuria was observed in 33.8% of patients, and DIC manifested as elevated D-dimer levels in 30.8% of patients. In our study, most snake bites occurred during the daytime and mainly involved the lower limbs. Therefore, protective footwear use may be beneficial for reducing snakebites.

In this study, the mortality rate was 10.8%. This is comparable to other reported studies (8.0-39.0%) among patients with snakebite envenomation [9-11,13,14]. Clinical and laboratory parameters such as D-dimer, serum urea, creatinine, hematuria, and oliguria were highly significant predictors of mortality in our study. Furthermore, the comparison between the patients who survived and those who died showed a significant difference in the duration from bite to hospital arrival. Our study showed that a delay in hospital arrival following a snakebite increased the risk of AKI and mortality with an ap value <0.001, which was highly significant. Our study also showed that the number of ASV vials required was positively correlated with the time to reach the hospital [Table 3].

5. CONCLUSION

This study concluded that AKI occurred in 30.8% of patients with vasculotoxic snake bite envenomation. Clinical and laboratory parameters associated with AKI include cellulitis,oliguria,hematuria, DIC as evidenced by elevated D-dimer levels, abnormal Renal Function Test results (elevated urea and creatinine levels), and delay in hospital arrival. The overall mortality rate of AKI after vasculotoxic snake bites was 10.8%. The presence of DIC and delay in hospital arrival following a snakebite are predictors of poor outcomes in patients with viperine snake bites developing AKI. Mortality can be prevented by early hospital arrival and administration of ASV.

Conflict of interest: The authors declare that they have no conflict of interest.

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