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EARLY PREDICTORS OF ACUTE KIDNEY INJURY FOLLOWING VENOMOUS SNAKEBITE ENVENOMATION: AN OBSERVATIONAL STUDY AT A TERTIARY CARE CENTRE IN SOUTH INDIA

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ABSTRACT

Background: Acute kidney injury (AKI) is a serious and potentially fatal complication of snakebite envenomation, particularly following hemotoxic bites. Early identification of patients at risk for AKI is critical for timely intervention and improaved clinical outcomes. This study aimed to determine the incidence of snakebite-induced AKI and to identify early clinical and biochemical predictors at the time of hospital admission.

Materials and Methods: This observational study enrolled 102 consecutive patients admitted with confirmed venomous snakebite to the Department of General Medicine, Mahatma Gandhi Memorial Government Hospital, Tiruchirappalli, over a one-year period. AKI was defined using the Acute Kidney Injury Network (AKIN) criteria. Demographic characteristics, clinical features, haematological parameters, coagulation profiles, biochemical markers, and treatment variables were compared between AKI and non-AKI groups using independent samples t-tests, chi-square tests, and Fisher's exact tests, with statistical significance set at $p < 0.05$.

Results: AKI developed in 18 of 102 patients (17.6%), exclusively among those with hemotoxic envenomation. Patients with AKI were significantly older (mean age 56.22 ± 8.57 vs. 37.88 ± 5.86 years; $p < 0.001$) and presented with significantly longer bite-to-hospital times (203.33 ± 34.94 vs. 92.21 ± 29.93 minutes; $p < 0.001$). Abnormal urine discolouration (black or brown) was observed exclusively in the AKI group ($p < 0.001$). Significant laboratory predictors included lower haemoglobin, lower platelet counts, elevated serum urea, creatinine, AST, ALT, LDH, creatine kinase, and prolonged coagulation parameters (prothrombin time, INR, WBCT, bleeding time). The overall mortality rate was 9.8%.

Conclusion: Hemotoxic envenomation, advanced age, delayed hospital presentation, abnormal urine colour, and specific haematological, biochemical, and coagulation derangements at admission are significant early predictors of AKI following snakebite. Integration of these parameters into clinical risk stratification protocols may facilitate early identification of high-risk patients and reduce AKI-associated morbidity and mortality.

Keywords: Acute kidney injury; snakebite envenomation; hemotoxic; early predictors; coagulopathy; renal failure.

INTRODUCTION

Snakebite envenomation constitutes a neglected tropical disease of substantial global public health significance. The World Health Organization (WHO) estimates that between 4.5 and 5.4 million snakebite incidents occur worldwide annually, with 1.8 to 2.7 million cases resulting in clinical envenomation and

81,000 to 138,000 fatalities (WHO, 2019). The burden of snakebite disproportionately affects rural populations in tropical and subtropical regions, particularly agricultural workers, herders, fishermen, and children in low-income communities with inadequate housing and limited access to healthcare infrastructure.[1-8]

India bears a particularly heavy burden, accounting for approximately half of all snakebite-related deaths globally. An estimated 3 to 4 million snakebite incidents occur annually in India, with approximately 50,000 fatalities. Data from the Central Bureau of Health Investigation (CBHI) indicate that approximately 300,000 snakebite cases are reported annually, although the true burden is likely underestimated due to substantial underreporting. Approximately 90% of these incidents are attributed to the "Big Four" venomous species: the Russell's viper (Daboia russelii), the common krait (Bungarus caeruleus), the Indian cobra (Naja naja), and the saw-scaled viper (Echis carinatus). Regional epidemiological analyses have demonstrated that states such as West Bengal, Tamil Nadu, Maharashtra, Andhra Pradesh, and Odisha bear the highest per-capita snakebite incidence.[9-16]

Among the multisystemic complications of snake envenomation, acute kidney injury (AKI) represents one of the most clinically consequential outcomes. AKI following snakebite is predominantly associated with hemotoxic and myotoxic venoms, which trigger a cascade of nephrotoxic processes including direct venom-mediated cytotoxicity, intravascular haemolysis, rhabdomyolysis, disseminated intravascular coagulation (DIC), haemodynamic instability, and microangiopathic damage. The pathological spectrum of snakebite-associated AKI (SAKI) encompasses acute tubular necrosis, cortical necrosis, glomerulonephritis, interstitial nephritis, and thrombotic microangiopathy. Published studies report AKI incidence ranging from 1.4% to 44.9% among snakebite patients, with mortality rates in the AKI subgroup reaching 18–39%.[17-22]

Several studies have sought to identify clinical and laboratory predictors of SAKI. Factors consistently implicated include advanced age, delayed presentation to medical facilities, the type and severity of envenomation, coagulopathy, elevated creatine kinase levels, leucocytosis, thrombocytopenia, and abnormal urine findings. However, the predictive significance of these variables varies considerably across geographic regions, reflecting differences in predominant snake species, venom composition, healthcare access, and management practices. In the South Indian context, where viperine species predominate and rural healthcare access remains challenging, there is a paucity of data specifically characterising early predictors of AKI at the time of hospital admission.[23-26]

The identification of reliable early predictors is of considerable clinical importance because AKI following snakebite is potentially reversible with prompt and appropriate management, including timely antivenom administration, aggressive fluid resuscitation, and renal replacement therapy when indicated. Conversely, delayed recognition of AKI portends poorer outcomes, including progression to chronic kidney disease and increased mortality.[27-30]

The present study was therefore designed to address this research gap by investigating the incidence of AKI among snakebite patients presenting to a tertiary care centre in Tamil Nadu, South India, and by identifying clinical characteristics, haematological parameters, biochemical markers, and coagulation profiles that serve as early predictors of AKI at the time of hospital admission. The findings are intended to inform the development of risk stratification tools that can be readily deployed in resource-limited clinical settings to facilitate early identification and targeted management of patients at high risk for SAKI.[31]

This paper is organised as follows. Section 2 describes the study design, setting, participant selection, data collection procedures, and analytical methods. Section 3 presents the results, including overall descriptive characteristics, AKI incidence, and comparative analyses between AKI and non-AKI groups. Section 4 discusses the findings in the context of existing literature, addresses study limitations, and offers conclusions and directions for future research.[32]

MATERIALS AND METHODS

Study Design, Setting, and Duration: This was a prospective, hospital-based observational study conducted at the Department of General Medicine, Mahatma Gandhi Memorial Government Hospital, Tiruchirappalli, Tamil Nadu, India. This institution is a tertiary care referral centre serving a predominantly rural and semi-urban catchment area in central Tamil Nadu. The study was conducted over a period of one year.

Study Population and Eligibility Criteria: The study population comprised all patients admitted with a confirmed history of venomous snakebite during the study period. Inclusion criteria were: (a) written informed consent to participate, (b) a definitive history of snakebite, and (c) clinical evidence of envenomation including visible fang marks. Exclusion criteria were: (a) pre-existing renal disease, (b) known comorbidities including diabetes mellitus, hypertension, cardiovascular disease, pulmonary disease, liver disease, neurological disease, or haematological disorders, (c) concurrent use of antiplatelet or anticoagulant medications, (d) pregnancy, and (e) refusal to provide informed consent. These exclusion criteria were applied to minimise confounding by pre-existing conditions that could independently contribute to renal impairment.

Sample Size Determination and Sampling Technique: The sample size was calculated using the single proportion formula: $N = Z^2P(1–P)/d^2$, where Z

= 1.96 (for 95% confidence), P = 0.28 (expected AKI prevalence based on David et al., 2012), and d = 0.09 (absolute precision). Incorporating a 10% non-response rate, the required sample size was determined to be 102 patients. Consecutive sampling was employed, enrolling all eligible patients sequentially until the target sample size was achieved.

Definition of Acute Kidney Injury: AKI was diagnosed according to the Kidney Disease: Improving Global Outcomes (KDIGO) 2012 guideline and staged using the Acute Kidney Injury Network (AKIN) classification. AKI was defined as: (a) an absolute increase in serum creatinine ≥0.3 mg/dL within 48 hours of admission, (b) a ≥1.5-fold increase in serum creatinine from the baseline value recorded at admission, or (c) urine output less than 0.5 mL/kg/h for more than 6 hours. Snakebite-induced AKI was further operationally defined as a creatinine clearance (estimated GFR) <60 mL/min/1.73 m² within the first 72 hours following snakebite, calculated using the CKD-EPI formula, in the absence of diabetes, hypertension, or prior renal disease.

Data Collection Procedures: A semi-structured questionnaire was used to collect data at the time of admission and during the subsequent hospital course. Demographic variables (age, gender), bite characteristics (site of bite, time of bite, type of envenomation, arrival time to hospital), clinical features (cellulitis, hypotension, bleeding manifestations, urine colour, oliguria, DIC), and treatment variables (number of anti-snake venom vials administered, hospital stay duration, outcome) were documented.

All patients received standard management according to the 2010 WHO guidelines for snakebite management in South-East Asian countries, including serial monitoring with the 20-minute whole blood clotting test (20WBCT), antivenom administration per clinical indications, and supportive care. Antivenom was administered for patients exhibiting local envenomation with progressive swelling or systemic envenomation evidenced by haemostatic abnormalities, neurotoxic signs, cardiovascular disturbances, or renal impairment.

Laboratory Investigations: The following laboratory parameters were assessed at admission (Day 1) and at 48 hours (Day 2) as clinically indicated: complete blood count (haemoglobin, white blood cell count, platelet count), coagulation profile (prothrombin time, international normalised ratio [INR], 20-minute WBCT, bleeding time, fibrinogen), renal function tests (serum urea, serum creatinine), liver function tests (AST, ALT, serum albumin), serum lactate dehydrogenase (LDH), creatine kinase (CK), C-reactive protein (CRP), urine analysis (colour, presence of RBCs/haemoglobin), blood glucose, serum electrolytes, and electrocardiography. Laboratory values obtained on the first day of admission were utilised for early predictor analysis.

Statistical Analysis: Data were entered in Microsoft Excel 2019 and analysed using SPSS version 21 (IBM Corporation, Armonk, NY). Descriptive statistics were generated for all variables: continuous variables were expressed as mean ± standard deviation (SD), and categorical variables as frequencies and percentages. For inferential analysis, comparisons between AKI and non-AKI groups were performed using independent samples t-tests (or Mann-Whitney U tests where normality assumptions were violated) for continuous variables and chi-square tests (or Fisher's exact tests for cells with expected frequency <5) for categorical variables. A two-tailed p-value < 0.05 was considered statistically significant for all analyses.

Ethical Considerations: The study protocol was reviewed and approved by the Institutional Ethics Committee of K.A.P.V. Government Medical College, Tiruchirappalli. Informed oral consent was obtained from all participants (or their legal representatives in cases of incapacitation) prior to enrolment. Patient confidentiality was maintained throughout the study, and all data were anonymised for analysis.

RESULTS

Demographic and Clinical Characteristics of the Study Population: A total of 102 patients meeting the inclusion criteria were enrolled. The mean age was 41.12 ± 9.48 years (range: 28–67 years), with a male predominance (70 males [68.6%] vs. 32 females [31.4%]). The majority of bites involved the lower limb (76 patients, 74.5%), followed by the foot (14, 13.7%), upper limb (8, 7.8%), and hand (4, 3.9%). Hemotoxic envenomation was the most frequent type (64 patients, 62.7%), followed by unknown type (26, 25.5%) and neurotoxic envenomation (12, 11.8%). The mean arrival time to hospital was 111.82 ± 52.48 minutes (range: 30–260 minutes). [Table 1] presents the complete demographic and clinical profile.

Table 1: Demographic and Clinical Characteristics of the Study Population (N = 102)

Variable	Category	n / Mean	% / SD
Age (years)	—	41.12	±9.48
Gender	Male	70	68.6%
	Female	32	31.4%
Envenomation type	Hemotoxic	64	62.7%
	Neurotoxic	12	11.8%
	Unknown	26	25.5%
Bite site	Lower limb	76	74.5%
	Foot	14	13.7%
	Upper limb	8	7.8%
	Hand	4	3.9%
Arrival time (min)	—	111.82	±52.48
Cellulitis	Present	26	25.5%
Hypotension	Present	18	17.6%
Bleeding manifestation	Present	18	17.6%
DIC	Present	16	15.7%
Hospital stay (days)	—	11.41	±3.78
ASV vials administered	—	16.94	±1.31

DIC = disseminated intravascular coagulation; ASV = anti-snake venom; SD = standard deviation.

Baseline Laboratory Parameters: At admission, the mean serum creatinine was 1.70 ± 0.96 mg/dL, blood urea was 60.22 ± 53.39 mg/dL, haemoglobin was 9.63 ± 0.95 g/dL, platelet count was 195.16 ± 54.67 ×103/µL, and white blood cell count was 13,900 ± 2,352/mm³. The coagulation profile showed a mean prothrombin time of 18.98 ± 7.22 seconds, INR of 1.27 ± 0.48, 20-minute WBCT of 13.43 ± 3.14 minutes, and bleeding time of 9.18 ± 2.05 minutes. The mean LDH was 328.41 ± 56.70 IU/L, AST was 49.90 ± 15.76 IU/L, ALT was 37.33 ± 5.31 IU/L, creatine kinase was 181.57 ± 47.52 IU/L, CRP was 20.43 ± 2.64 mg/dL, serum albumin was 3.40 ± 0.42 g/dL, and fibrinogen was 238.25 ± 51.97 mg/dL. Regarding urine appearance, 90 patients (88.2%) had normal-coloured urine, 10 (9.8%) had black urine, and 2 (2.0%) had brown urine. Table 2 summarises these findings.

Table 2: Baseline Laboratory Parameters of the Study Population (N = 102)

Parameter	Mean	SD	Range
Haemoglobin (g/dL)	9.63	0.95	6.90–11.40
WBC count (/mm³)	13,900	2,352	11,400–19,900
Platelet count (×10³/µL)	195.16	54.67	124–286
Serum urea (mg/dL)	60.22	53.39	10–210
Serum creatinine (mg/dL)	1.70	0.96	0.9–4.6
Serum albumin (g/dL)	3.40	0.42	2.0–4.0
AST (IU/L)	49.90	15.76	20–100
ALT (IU/L)	37.33	5.31	30–55
LDH (IU/L)	328.41	56.70	200–500
Creatine kinase (IU/L)	181.57	47.52	100–350
CRP (mg/dL)	20.43	2.64	17–30
Fibrinogen (mg/dL)	238.25	51.97	197–376
Prothrombin time (s)	18.98	7.22	14–38
INR	1.27	0.48	0.93–2.53
WBCT (min)	13.43	3.14	10–23
Bleeding time (min)	9.18	2.05	7–15

WBC = white blood cell; AST = aspartate aminotransferase; ALT = alanine aminotransferase; LDH = lactate dehydrogenase; CRP = C-reactive protein; INR = international normalised ratio; WBCT = 20-minute whole blood clotting test.

Incidence and Outcome of Snakebite-Induced AKI: Of the 102 patients, 18 (17.6%) developed snakebite-induced AKI. All 18 AKI cases occurred exclusively in patients with hemotoxic envenomation (18/64 hemotoxic patients, 28.1%); no cases of AKI were observed among patients with neurotoxic or unknown envenomation types (p = 0.002). Regarding outcomes, 92 patients (90.2%) were discharged and 10 (9.8%) died during hospitalisation.

Comparison of Demographic and Clinical Characteristics Between AKI and Non-AKI Groups: Patients who developed AKI were significantly older than those who did not (mean age 56.22 ± 8.57 vs. 37.88 ± 5.86 years; p < 0.001). The mean bite-to-hospital arrival time was significantly longer in the AKI group (203.33 ± 34.94 vs. 92.21 ± 29.93 minutes; p < 0.001). No statistically significant difference was observed between the two groups with respect to gender (p = 0.357) or bite site (p = 0.478). Abnormal urine discolouration was exclusively observed in the AKI group: all 10 patients with black urine and both patients with brown urine developed AKI, compared to only 6 of 90 patients with normal urine colour (p < 0.001). The AKI group received a significantly higher mean number of ASV vials (18.44 ± 2.12 vs. 16.61 ± 0.76; p < 0.001).

Comparison of Laboratory Parameters Between AKI and Non-AKI Groups: All laboratory

parameters assessed at admission demonstrated statistically significant differences between the AKI and non-AKI groups (all $p < 0.001$). The AKI group exhibited significantly higher mean values for serum urea (174.00 ± 16.23 vs. 35.83 ± 2.33 mg/dL), serum creatinine (3.62 ± 0.76 vs. 1.28 ± 0.16 mg/dL), AST (78.77 ± 8.57 vs. 43.71 ± 8.22 IU/L), ALT (46.55 ± 4.11 vs. 35.35 ± 2.91 IU/L), LDH (435.77 ± 40.96 vs. 305.40 ± 23.07 IU/L), creatine kinase (271.55 ± 50.67 vs. 162.28 ± 9.45 IU/L), CRP (24.77 ± 1.59 vs. 19.50 ± 1.71 mg/dL), WBC count (18,640 ± 945 vs. 12,884 ± 794/mm³), fibrinogen (344.66 ± 24.81 vs. 215.45 ± 13.35 mg/dL), prothrombin time (34.22 ± 2.21 vs. 15.71 ± 1.14 seconds), INR (2.28 ± 0.15 vs. 1.04 ± 0.08), WBCT (19.33 ± 2.47 vs. 12.16 ± 1.25 minutes), and bleeding time (13.11 ± 1.23 vs. 8.33 ± 0.84 minutes). Conversely, the AKI group had significantly lower haemoglobin (8.05 ± 0.65 vs. 9.96 ± 0.60 g/dL), platelet count (138.88 ± 3.37 vs. 207.21 ± 52.92 ×103/µL), and serum albumin (2.58 ± 0.39 vs. 3.57 ± 0.11 g/dL). [Table 3] presents the complete comparative analysis.

Table 3: Comparison of Laboratory Parameters Between AKI and Non-AKI Groups

Parameter	AKI (n=18)	Non-AKI (n=84)	p-value	Test
Age (years)	56.22 ± 8.57	37.88 ± 5.86	<0.001	t-test
Arrival time (min)	203.33 ± 34.94	92.21 ± 29.93	<0.001	t-test
Haemoglobin (g/dL)	8.05 ± 0.65	9.96 ± 0.60	<0.001	t-test
WBC (/mm³)	18,640 ± 945	12,884 ± 794	<0.001	t-test
Platelet (×10³/µL)	138.88 ± 3.37	207.21 ± 52.92	<0.001	t-test
Urea (mg/dL)	174.00 ± 16.23	35.83 ± 2.33	<0.001	t-test
Creatinine (mg/dL)	3.62 ± 0.76	1.28 ± 0.16	<0.001	t-test
Albumin (g/dL)	2.58 ± 0.39	3.57 ± 0.11	<0.001	t-test
AST (IU/L)	78.77 ± 8.57	43.71 ± 8.22	<0.001	t-test
ALT (IU/L)	46.55 ± 4.11	35.35 ± 2.91	<0.001	t-test
LDH (IU/L)	435.77 ± 40.96	305.40 ± 23.07	<0.001	t-test
Creatine kinase (IU/L)	271.55 ± 50.67	162.28 ± 9.45	<0.001	t-test
CRP (mg/dL)	24.77 ± 1.59	19.50 ± 1.71	<0.001	t-test
Fibrinogen (mg/dL)	344.66 ± 24.81	215.45 ± 13.35	<0.001	t-test
PT (seconds)	34.22 ± 2.21	15.71 ± 1.14	<0.001	t-test
INR	2.28 ± 0.15	1.04 ± 0.08	<0.001	t-test
WBCT (min)	19.33 ± 2.47	12.16 ± 1.25	<0.001	t-test
Bleeding time (min)	13.11 ± 1.23	8.33 ± 0.84	<0.001	t-test
ASV vials	18.44 ± 2.12	16.61 ± 0.76	<0.001	t-test

Values are expressed as mean ± SD. PT = prothrombin time; INR = international normalised ratio; WBCT = whole blood clotting test; ASV = anti-snake venom. All p-values computed using independent samples t-test.

Table 4: Comparison of Categorical Variables Between AKI and Non-AKI Groups

Variable	AKI (n=18)	Non-AKI (n=84)	p-value	Test
Gender (Male)	14 (20.0%)	56 (80.0%)	0.357	Chi-square
Hemotoxic bite	18 (28.1%)	46 (71.9%)	0.002	Chi-square
Urine – Black	10 (100%)	0 (0%)	<0.001	Fisher's
Urine – Brown	2 (100%)	0 (0%)	<0.001	Fisher's
Urine – Normal	6 (6.7%)	84 (93.3%)	—	Ref.
Bite: Lower limb	12 (15.8%)	64 (84.2%)	0.478	Chi-square

Values are expressed as n (%). Chi-square or Fisher's exact test as appropriate.

DISCUSSION

Incidence of AKI: The present study identified an AKI incidence of 17.6% among snakebite patients admitted to a tertiary care centre in Tamil Nadu, South India. This finding is consistent with the widely reported range of 8–44.9% documented in the international literature and aligns closely with figures from comparable Asian studies. Moon et al. (2024) reported a 14.1% incidence in South Korea, Li et al. (2016) observed 13.4% in China, and Gadwalkar et al. (2014) documented 15.5% in Karnataka, India. Conversely, higher incidence rates have been reported in centres with greater proportions of hemotoxic envenomation or delayed presentations, including 43.3% by Paul et al. (2012) in West Bengal, 44.9% by Singh et al. (2016) in Uttar Pradesh, and 43.8% by Priyamvada et al. (2019) in Puducherry. These variations likely reflect regional differences in predominant snake species, venom nephrotoxicity profiles, healthcare access, and antivenom administration timeliness. The exclusive occurrence of AKI among hemotoxic envenomation cases in the present study (28.1% of hemotoxic patients) strongly underscores the nephrotoxic propensity of viperine venoms, which act through multiple synergistic pathways including direct cytotoxicity, coagulopathy-mediated microvascular injury, intravascular haemolysis, and rhabdomyolysis.

Age as a Predictor: Advanced age emerged as a robust predictor of AKI development, with the AKI group being significantly older (56.22 ± 8.57 vs. 37.88 ± 5.86 years; $p < 0.001$). This finding corroborates observations by Albuquerque et al. (2014), who reported higher mean age among AKI patients (43 ± 20 vs. 34 ± 21 years; $p = 0.015$), and Li et al. (2016), who documented a substantial age differential (66 ± 7 vs. 43 ± 9 years). The age-related vulnerability to AKI is biologically plausible, as

ageing is associated with progressive nephron loss, reduced renal functional reserve, impaired autoregulatory capacity, and heightened susceptibility to nephrotoxic insults. These factors collectively diminish the kidney's ability to withstand the haemodynamic and toxic challenges imposed by snake venom. Gender was not a significant predictor in the present study ($p = 0.357$), consistent with the broader literature suggesting that gender differences in AKI incidence primarily reflect occupational exposure patterns rather than intrinsic biological susceptibility.

Delayed Hospital Presentation: Delayed hospital arrival was strongly associated with AKI development (203.33 ± 34.94 vs. 92.21 ± 29.93 minutes; $p < 0.001$). This finding aligns with consistent evidence from multiple studies. Athappan et al. (2008) identified a bite-to-needle time exceeding two hours as an independent risk factor for AKI, while David et al. (2012) demonstrated significantly higher mortality among patients with pre-hospital delays exceeding 24 hours (18% vs. 5%; $p < 0.001$). Prolonged interval between envenomation and antivenom administration permits sustained systemic venom effects, including progressive coagulopathy, ongoing haemolysis, and renal hypoperfusion, all of which compound the risk of irreversible renal injury. In the rural Indian context, delays are frequently attributable to geographic remoteness, limited transportation infrastructure, reliance on traditional healing practices, and insufficient awareness of the urgency of hospital-based management. These findings emphasise the critical importance of community-level health education programmes and decentralised antivenom availability to reduce bite-to-treatment intervals.

Urine Discolouration as a Clinical Indicator: Abnormal urine colour (black or brown) was observed exclusively in the AKI group ($p < 0.001$), representing one of the most discriminating early clinical features identified in this study. Dark urine indicates the presence of haemoglobinuria or myoglobinuria, which are pathognomonic of intravascular haemolysis and rhabdomyolysis, respectively—both established mechanisms of AKI in snake envenomation. Paul et al. (2012) similarly reported dark or brown urine as an independent predictor of AKI, and Vikrant et al. (2017) noted that 55% of AKI patients in their cohort had a history of passing red or dark urine. These observations highlight the value of simple bedside urine colour assessment as a cost-free, immediately available screening tool for renal involvement in resource-limited settings.

Laboratory Predictors: All laboratory parameters examined demonstrated highly significant differences between the AKI and non-AKI groups (all $p < 0.001$). The constellation of findings—elevated renal function markers (urea, creatinine), liver enzymes (AST, ALT), tissue damage markers (LDH, creatine kinase), coagulation derangements (prolonged PT, elevated INR, prolonged WBCT and bleeding time, elevated fibrinogen), haematological abnormalities (anaemia, thrombocytopenia, leucocytosis), hypoalbuminaemia, and elevated CRP—collectively reflect the multisystemic nature of severe envenomation leading to AKI. These findings are concordant with those of Aye et al. (2017), who identified elevated creatine kinase, DIC, leucocytosis, and microscopic haematuria as significant predictors in Myanmar, and Dharod et al. (2013), who demonstrated that haemoglobin, total bilirubin, bleeding time, prothrombin time, and hypotension were independent predictors of AKI on multivariate analysis. The elevated creatine kinase and LDH levels suggest that rhabdomyolysis and intravascular haemolysis contribute substantially to the pathogenesis of AKI in this population, consistent with the known mechanisms of viperine venom-induced nephrotoxicity. The coagulation abnormalities, particularly the markedly prolonged prothrombin time and elevated INR in the AKI group, reflect venom-induced consumption coagulopathy (VICC), which predisposes to microvascular thrombosis and renal ischaemia. The utility of the 20-minute WBCT as a bedside predictor, as also reported by Paul et al. (2012), is particularly noteworthy given its simplicity and applicability in resource-limited primary healthcare settings.

Practical and Theoretical Implications: The present findings have several practical implications. First, the identification of advanced age, delayed presentation, hemotoxic envenomation, urine discolouration, and admission-day laboratory derangements as early predictors enables the development of simple, point-of-care risk stratification protocols. Clinicians in emergency settings can rapidly identify high-risk patients by assessing urine colour, performing a bedside WBCT, and obtaining basic haematological and biochemical panels. Second, the strong association between delayed presentation and AKI underscores the need for community education campaigns emphasising immediate hospital attendance after snakebite, reduction of reliance on traditional remedies, and strengthening of referral pathways. Third, the exclusive association of AKI with hemotoxic envenomation reinforces the importance of early, adequate antivenom administration and aggressive supportive care for viperine bites. From a theoretical perspective, the findings reinforce the multifactorial pathogenesis of SAKI, involving the interplay of direct nephrotoxicity, coagulopathy-mediated microvascular injury, haemolysis, rhabdomyolysis, and haemodynamic compromise.

Limitations: This study has several limitations that must be acknowledged. First, as a single-centre study conducted at a tertiary referral hospital, the findings may not be generalisable to other geographic regions or healthcare settings with different snake species profiles, management protocols, or patient demographics. Second, the relatively small AKI

subgroup (n = 18) may have limited the statistical power to detect more subtle associations or to perform robust multivariate regression analysis to identify independent predictors after adjusting for confounders. Third, although patients with known comorbidities were excluded, subclinical pre-existing conditions such as undiagnosed early-stage chronic kidney disease or latent diabetes may have influenced the results. Fourth, the observational design precludes establishing causal relationships between predictors and AKI outcomes. Fifth, the study relied on admission-day laboratory values and did not incorporate novel biomarkers (such as NGAL, cystatin C, or KIM-1) that may provide earlier and more sensitive detection of renal injury.

CONCLUSION

In conclusion, AKI developed in 17.6% of snakebite patients in this South Indian tertiary care cohort, occurring exclusively in the context of hemotoxic envenomation. Advanced age, delayed hospital presentation, abnormal urine discolouration, and a comprehensive panel of haematological, biochemical, and coagulation derangements at admission were identified as significant early predictors of AKI. These readily assessable clinical and laboratory parameters can be integrated into simple risk stratification tools to facilitate the early identification of high-risk patients, enabling prompt targeted management and potentially reducing the substantial morbidity and mortality associated with snakebite-induced AKI. Future research should include larger, multicentre prospective studies with adequate statistical power to perform multivariate logistic regression and develop validated predictive scoring systems. The incorporation of novel renal biomarkers such as neutrophil gelatinase-associated lipocalin (NGAL), serum cystatin C, kidney injury molecule-1 (KIM-1), and urine clusterin, as explored by Ratnayake et al. (2019), may enable earlier detection of subclinical AKI. Long-term follow-up studies are needed to characterise the trajectory of renal recovery and the incidence of chronic kidney disease following snakebite-induced AKI. Additionally, interventional studies evaluating the impact of structured risk stratification protocols and early renal protective strategies on AKI outcomes would provide valuable evidence to inform clinical practice guidelines.

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