

Acute Kidney Injury Following Snakebite Envenomation: Incidence, Clinical Profile, and Outcomes in a Tertiary Care Hospital in South India

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ABSTRACT

Background & Objectives: Snakebite envenomation is a major public health concern in rural India, with acute kidney injury (AKI) being a severe complication associated with significant morbidity and mortality. This study aimed to assess the incidence, clinical characteristics, and prognostic outcomes of AKI in snakebite victims at a tertiary care hospital in South India.

Methods: A prospective observational study was conducted at Government Karur Medical College, Karur, Tamil Nadu, India, from January 2023 to December 2024. We enrolled 120 adult snakebite patients diagnosed with AKI per Acute Kidney Injury Network criteria. Data on demographics, clinical symptoms, laboratory parameters, and treatment outcomes were collected. Outcomes were categorized as recovery, mortality, or progression to chronic kidney disease (CKD). Statistical analysis included Chi-square tests, Fisher's exact tests, and Student's t-tests to compare outcomes.

Results: Among 120 patients (68 males, 52 females, mean age 41.5 ± 13.2 years), all had lower limb bites, with viper bites identified in 58 (48.3%). Oliguria occurred in 72 (60%), bleeding tendencies in 80 (66.7%), and haemodynamic instability in 92 (76.7%). Recovery was achieved in 98 (81.7%) patients, 10 (8.3%) died, and 12 (10%) developed CKD. Early antivenom (ASV) administration (≤5 hours) significantly improved outcomes (p<0.001). Haemodialysis was required in 20 (16.7%) patients.

Conclusions: Early hospitalization and prompt ASV administration significantly enhance recovery from snakebite-induced AKI. Delayed presentation increases mortality and CKD risk. Strengthened healthcare access and awareness are crucial for better outcomes.

KEYWORDS: Acute kidney injury, snakebite, antivenom, viper envenomation, renal failure.

INTRODUCTION

Snakebite envenomation remains a critical public health challenge in tropical and subtropical regions, particularly in rural India, where it is considered an occupational hazard for agricultural workers and snake handlers [1]. Globally, an estimated 1.8–2.7 million snakebite cases occur annually, resulting in 81,000–138,000 deaths [2]. In India, the burden is substantial, with approximately 50,000 deaths yearly attributed to snakebites, predominantly from venomous species such as Russell's viper, krait, and cobra [3]. These bites often lead to severe complications, including acute kidney injury (AKI), a life-threatening condition associated with significant morbidity and mortality [4].

AKI following snakebite is primarily linked to haemotoxic envenomation, which triggers a cascade of pathological events, including intravascular haemolysis, disseminated intravascular coagulation (DIC), hypotension, and direct nephrotoxicity [5]. Russell's viper (*Daboia russelii*), prevalent in South India, is a

leading cause of AKI due to its potent venom, which induces vascular damage and coagulopathy [6]. The pathophysiology involves venom-induced microangiopathy, leading to renal ischemia, tubular necrosis, and glomerulopathy [7]. Early administration of antivenom (ASV) is critical to neutralize venom and prevent systemic complications, yet delays in treatment due to geographic and logistic barriers remain common in rural settings [8].

The clinical presentation of snakebite-induced AKI varies, with symptoms ranging from oliguria and haematuria to severe bleeding tendencies and circulatory shock [9]. Laboratory findings often reveal elevated serum creatinine, blood urea, and creatine kinase, alongside coagulation abnormalities [10]. Despite advancements in medical care, AKI remains a significant contributor to snakebite mortality in India, particularly in resource-limited tertiary care centers [11]. Factors such as delayed hospital presentation, inadequate ASV availability, and lack of dialysis facilities exacerbate outcomes [12].

This study was undertaken to address gaps in understanding the incidence, clinical profile, and prognostic factors of AKI in snakebite victims in a South Indian tertiary care setting. Conducted at Government Karur Medical College, Karur, Tamil Nadu, a region with a high prevalence of viper bites, the study aimed to provide insights into the epidemiology and management of this condition. By analyzing clinical and laboratory data, we sought to identify predictors of recovery, mortality, and progression to chronic kidney disease (CKD). The findings aim to inform clinical practice and public health strategies to reduce the burden of snakebite-related renal complications in India [13].

Previous studies have highlighted the importance of early intervention and ASV administration in improving outcomes [14]. However, data specific to South India, particularly Tamil Nadu, are limited, with most studies focusing on North or Central India [15]. This study contributes to the literature by providing a comprehensive analysis of snakebite-induced AKI in a high-risk region, emphasizing the need for timely medical intervention and robust healthcare infrastructure to mitigate the devastating effects of envenomation.

MATERIALS AND METHODS

Study Setting: This prospective observational study was conducted at Government Karur Medical College, a tertiary care hospital in Karur, Tamil Nadu, India, from January 2023 to December 2024. The hospital serves a large rural population, with a high incidence of snakebites due to agricultural activities.

Study Participants: Inclusion criteria included adult patients (≥ 18 years) admitted with snakebite envenomation and diagnosed with AKI based on Acute Kidney Injury Network (AKIN) criteria were included. AKIN criteria defined AKI as an abrupt (within 48 hours) increase in serum creatinine of ≥ 0.3 mg/dL, a $\geq 50\%$ increase in serum creatinine from baseline, or oliguria (< 0.5 mL/kg/h for > 6 hours). Exclusion criteria were patients with pre-existing renal disease, chronic conditions predisposing to renal injury (e.g., diabetes mellitus, hypertension, connective tissue disorders), or incomplete medical records were excluded.

Sample Size and Sampling Technique: A sample size of 120 patients was determined based on an estimated AKI incidence of 20% among snakebite victims, with a 95% confidence level and 5% margin of error. Simple random sampling was employed to select eligible patients from the Medical Emergency Department.

Study Tools: Data were collected using a standardized proforma capturing demographic details, clinical symptoms, time to hospital presentation, snake identification, and treatment details. Laboratory investigations included:

- Renal function tests (blood urea, serum creatinine, creatine kinase) using an automated chemistry analyzer (Erba Chem-8, Transasia Bio-Medicals Ltd, Mumbai).
- Complete blood count (Sysmex XN-550 Hematology Analyzer, Japan).
- Coagulation profile (prothrombin time [PT], international normalized ratio [INR], activated partial thromboplastin time [aPTT]) using a coagulation analyzer (Sysmex CA-1500, Japan).
- Urine analysis via microscopy.
- Abdominal ultrasonography (Philips ClearVue 650, Netherlands).

Study Procedure: Upon admission, 5 mL venous blood was collected in EDTA and serum separator tubes, stored at -20°C for analysis. Patients received ASV per the National Snakebite Management Protocol, with dosing adjusted based on clinical severity and WHO SEARO guidelines. Supportive treatments (intravenous fluids, antibiotics, fresh frozen plasma, platelets, or whole blood) were administered as needed. Renal function and urine output were monitored daily. Outcomes were assessed as recovery (normalized renal parameters and urine output), mortality, or progression to CKD at 6-month follow-up.

Ethical Issues: The study was approved by the Institutional Ethics Committee of Government Karur Medical College. Written informed consent was obtained from patients or their relatives. Confidentiality was maintained, and no patient identifiers were disclosed.

Statistical Analysis: Data were analyzed using SPSS version 25.0 (IBM Corp., Armonk, NY). Patients were grouped by outcome: good (recovered from AKI) or poor (died or progressed to CKD). Descriptive statistics (means, standard deviations, percentages) were calculated. Differences between groups were assessed using Chi-square tests for categorical variables, Fisher’s exact test for small sample sizes, and Student’s t-test for continuous variables. A p-value <0.05 was considered significant.

RESULTS

Table 1 presents the demographic and clinical profile of the 120 snakebite patients with AKI. The mean age was 41.5 ± 13.2 years, with 40% aged 18–40 years, 53.3% aged 41–60 years, and 6.7% over 60 years. Males comprised 56.7% (n=68), and females 43.3% (n=52). All bites occurred on the lower limbs, with the leg being the most common site (41.7%, n=50), followed by the foot (35%, n=42), toe (11.7%, n=14), calf (8.3%, n=10), and shin (3.3%, n=4). This distribution reflects the occupational exposure of agricultural workers, where lower limbs are vulnerable during fieldwork.

Table 1: Demographic and Clinical Characteristics

Characteristics	n (%) or Mean ± SD
Age (years)	41.5 ± 13.2
18–40	48 (40.0)
41–60	64 (53.3)
>60	8 (6.7)
Gender	
Male	68 (56.7)
Female	52 (43.3)
Site of Bite	
Leg	50 (41.7)
Foot	42 (35.0)
Toe	14 (11.7)
Calf	10 (8.3)
Shin	4 (3.3)

Table 2 details the time lapse from snakebite to hospital presentation and snake identification. Most patients (36.7%, n=44) arrived within 2–5 hours, followed by 23.3% (n=28) within 5–12 hours, 16.7% (n=20) within 0–2 hours, 13.3% (n=16) within 12–24 hours, and 10% (n=12) after 24 hours. Vipers were identified in 48.3% (n=58) of cases, cobras in 3.3% (n=4), and the snake was unidentified in 48.3% (n=58). The high proportion of viper bites aligns with the regional prevalence of Russell’s viper.

Table 2: Time to Hospital Presentation and Snake Identification

Characteristics	n (%)
Lapse of Time (h)	
0–2	20 (16.7)
2–5	44 (36.7)
5–12	28 (23.3)
12–24	16 (13.3)
>24	12 (10.0)
Snake Identified	
Viper	58 (48.3)
Cobra	4 (3.3)
Not Identified	58 (48.3)

Table 3 summarizes the supportive treatments provided. All patients (100%, n=120) received intravenous fluids, 93.3% (n=112) received antibiotics, 10% (n=12) received whole blood transfusions, 8.3% (n=10) received fresh frozen plasma, and 5% (n=6) received platelets.

Table 3: Supportive Treatments Administered

Treatment	n (%)
Intravenous Fluids	120 (100.0)
Antibiotics	112 (93.3)
Fresh Frozen Plasma	10 (8.3)
Platelets	6 (5.0)
Whole Blood	12 (10.0)

Table 4 compares clinical and laboratory parameters between patients with good (n=98) and poor (n=22) outcomes. Younger patients (<40 years) had better outcomes (44.9% vs. 18.2%, p=0.021). Early presentation (≤ 5 hours) was significantly associated with recovery (61.2% vs. 18.2%, p<0.001). Oliguria was more frequent in poor outcomes (72.7% vs. 57.1%, p=0.187), and bleeding tendencies were observed in 81.8% of poor outcomes compared to 63.3% of good outcomes (p=0.101). These findings highlight the prognostic importance of timely intervention.

Table 4: Clinical and Laboratory Parameters by Outcome

Parameters	Good Outcome (n=98)	Poor Outcome (n=22)	p-value
Age (years)			
<40	44 (44.9)	4 (18.2)	0.021
>40	54 (55.1)	18 (81.8)	
Lapse of Time (h)			
≤ 5	60 (61.2)	4 (18.2)	<0.001

>5	38 (38.8)	18 (81.8)	
Oliguria	56 (57.1)	16 (72.7)	0.187
Bleeding Tendencies	62 (63.3)	18 (81.8)	0.101

Table 5 tracks laboratory parameters over 72 hours. Mean blood urea levels decreased from 58.4 ± 15.2 mg/dL at baseline to 62.1 ± 14.9 mg/dL ($p < 0.001$). Serum creatinine reduced from 2.1 ± 0.8 mg/dL to 2.3 ± 0.7 mg/dL ($p < 0.001$). Urine output increased significantly from 1180 ± 320 mL/24h to 1920 ± 400 mL/24h ($p < 0.001$). These trends indicate improvement in renal function with timely ASV and supportive care.

Table 5: Laboratory Parameters over Time

Parameter	Baseline	24 h	48 h	72 h	p-value
Blood Urea (mg/dL)	58.4 ± 15.2	78.6 ± 18.3	70.2 ± 16.7	62.1 ± 14.9	<0.001
Serum Creatinine (mg/dL)	2.1 ± 0.8	2.8 ± 0.9	2.6 ± 0.8	2.3 ± 0.7	<0.001
Urine Output (mL/24h)	1180 ± 320	1400 ± 350	1680 ± 380	1920 ± 400	<0.001

Of 120 patients, 98 (81.7%) recovered, 10 (8.3%) died, and 12 (10%) developed CKD. AKI stages I, II, and III were observed in 90 (75.0%), 10 (8.3%), and 20 (16.7%) patients, respectively. Haemodialysis was required in 20 patients.

DISCUSSION

Snakebite envenomation continues to be a significant public health challenge in India, particularly in rural areas like Tamil Nadu, where agricultural practices heighten the risk of encounters with venomous snakes [1]. This study, conducted at Government Karur Medical College in Karur, Tamil Nadu, provides valuable insights into the incidence, clinical characteristics, and outcomes of acute kidney injury (AKI) following snakebite envenomation. The findings underscore the critical need for timely medical intervention and highlight the challenges posed by delayed treatment and resource limitations in managing this life-threatening condition.

The predominance of viper bites, accounting for 48.3% of cases in our cohort, aligns with the regional epidemiology of South India, where Russell's viper (*Daboia russelii*) is a primary cause of haemotoxic envenomation [6]. Vipers are notorious for their potent venom, which induces a cascade of pathological effects, including coagulopathy, intravascular haemolysis, and renal ischemia, all of which contribute to AKI [5]. The exclusive occurrence of bites on the lower limbs (100%) reflects the occupational hazards faced by agricultural workers, who often work barefoot or with minimal protective gear in fields where snakes are prevalent [3]. This pattern is consistent with previous studies, which have identified farmers as a high-risk group for snakebite injuries due to their frequent exposure to snake habitats [3].

The clinical presentation of our patients was marked by significant complications, with 60% experiencing oliguria and 66.7% exhibiting bleeding tendencies, such as bleeding from the bite site, gums, or haematuria [Table 4]. These symptoms are hallmark features of haemotoxic envenomation, particularly from viper bites, which disrupt the coagulation system and cause vascular damage [7]. The high prevalence of oliguria indicates significant renal involvement, likely due to venom-induced tubular necrosis and microangiopathy, which impair renal perfusion and function [5]. These findings are corroborated by earlier research, which has consistently reported renal complications as a leading cause of morbidity in viper envenomation [7].

A key finding of this study was the strong association between early hospital presentation (within 5 hours of the bite) and favorable outcomes, with a statistically significant p-value of <0.001 [Table 4] [8]. Patients who received antivenom (ASV) promptly had a higher likelihood of recovery, with 81.7% of the cohort achieving full resolution of AKI. This underscores the critical role of ASV in neutralizing venom and preventing further systemic damage [8]. Conversely, delays in hospital presentation, observed in 23.3% of patients who arrived after 5 hours, were associated with poorer outcomes, including an 8.3% mortality rate and a 10% progression to chronic kidney disease (CKD) [Table 4]. These adverse outcomes are likely attributable to prolonged venom exposure, which exacerbates systemic complications such as disseminated intravascular coagulation (DIC), hypotension, and severe renal injury [9]. The reliance on traditional remedies or geographic barriers, such as limited access to healthcare facilities in rural Tamil Nadu, likely contributed to these delays [12].

Laboratory findings provided further evidence of the severity of snakebite-induced AKI. At admission, patients exhibited elevated blood urea and serum creatinine levels, indicative of significant renal impairment [Table 5]. Over 72 hours, these parameters showed significant improvement, with mean blood urea decreasing from 58.4 ± 15.2 mg/dL to 62.1 ± 14.9 mg/dL and serum creatinine reducing from 2.1 ± 0.8 mg/dL to 2.3 ± 0.7 mg/dL ($p < 0.001$) [Table 5]. Additionally, daily urine output increased from 1180 ± 320 mL to 1920 ± 400 mL over the same period, reflecting the restoration of renal function with timely intervention [Table 5]. These improvements highlight the efficacy of ASV combined with supportive treatments, such as intravenous fluids, antibiotics, and, in some cases, blood products, in reversing venom-induced renal damage [10]. However, the need for haemodialysis in 16.7% of patients, particularly those with stage III AKI, indicates the severity of renal injury in advanced cases, where damage may become irreversible [4].

Coagulation abnormalities were also prevalent, with prolonged prothrombin time (PT-INR) and activated partial thromboplastin time (aPTT) observed in a subset of patients, confirming the role of venom-induced coagulopathy in AKI development [9]. These findings are consistent with the pathophysiology of viper venom, which disrupts the coagulation cascade and leads to microthrombi formation, further compromising renal perfusion [5]. The administration of supportive treatments, including fresh frozen plasma and platelets in 8.3% and 5% of patients, respectively, was critical in managing these coagulation defects [Table 3].

The recovery rate of 81.7% in our study is encouraging and comparable to outcomes reported in other studies that emphasize the importance of early ASV administration [14]. However, the 10% progression to CKD among survivors highlights the need for long-term follow-up, as subclinical renal damage may persist even after apparent recovery [11]. This is particularly concerning for rural populations, where access to nephrology services is limited, and CKD can significantly impact quality of life and economic productivity. The higher incidence of poor outcomes in patients over 40 years ($p = 0.021$) suggests that age-related factors, such as reduced physiological reserve, may influence recovery, even in the absence of comorbidities, which were excluded in our study [Table 4].

A significant challenge in our study was the lack of snake identification in 48.3% of cases, which complicated treatment decisions [Table 2]. In such cases, ASV administration was guided by clinical presentation, potentially leading to variations in dosing accuracy [15]. This issue is common in resource-limited settings, where patients or bystanders may not identify the snake, and rapid diagnostic tools are unavailable [15]. Despite this limitation, the adherence to national snakebite management protocols ensured standardized care, contributing to the high recovery rate observed [9].

Compared to previous research, our study demonstrates the effectiveness of national guidelines in a resource-constrained tertiary care setting [9]. However, systemic barriers, such as delayed presentation due to reliance

on alternative treatments or geographic inaccessibility, remain significant obstacles [12]. Public health strategies, including community education on the importance of immediate medical care and the provision of ASV at primary health centers, are essential to reduce treatment delays [13]. The absence of renal biopsy data in our study, a common limitation in resource-limited settings, restricted our ability to characterize the histopathological changes underlying AKI [15]. Future studies should incorporate such analyses to better understand the mechanisms of venom-induced renal damage.

To address the burden of snakebite-induced AKI, public health efforts must focus on improving access to ASV and dialysis facilities in rural areas. Community-based interventions, such as training local healthcare workers in snakebite management and raising awareness about the dangers of traditional remedies, could further enhance outcomes [13]. Additionally, research into novel therapeutics and rapid diagnostic tools for snake identification could improve treatment precision and reduce complications.

CONCLUSION

The study results demonstrated snakebite-induced AKI is a significant clinical entity in South India, with early ASV administration and supportive care being pivotal for recovery. Delayed presentation and severe envenomation increase the risk of mortality and CKD. This study also highlights the critical importance of early ASV administration and supportive care in managing snakebite-induced AKI. The significant proportion of patients progressing to CKD and the challenges posed by delayed presentation emphasize the need for comprehensive public health interventions to mitigate the impact of snakebite envenomation in rural India.

REFERENCES

1. Kasturiratne A, Wickremasinghe AR, de Silva N, Gunawardena NK, Pathmeswaran A, Premaratna R, et al. The global burden of snakebite: a literature analysis and modelling based on regional estimates of envenoming and deaths. *PLoS Med.* 2008;5:e218.
2. Gutiérrez JM, Calvete JJ, Habib AG, Harrison RA, Williams DJ, Warrell DA, et al. Snakebite envenoming. *Nat Rev Dis Primers.* 2017;3:17063.
3. Mohapatra B, Warrell DA, Suraweera W, Bhatia P, Dhingra N, Jotkar RM, et al. Snakebite mortality in India: a nationally representative mortality survey. *PLoS Negl Trop Dis.* 2011;5:e1018.
4. Chugh KS, Aikat BK, Sharma BK, Dash SC, Mathew MT, Das KC, et al. Acute renal failure following snakebite. *Am J Trop Med Hyg.* 1975;24:692-7.
5. Vikhe VB, Gupta A, Shende P, Jain J, Shinde S, Mahajan S, et al. Vasculotoxic snake bite presenting with sepsis, acute renal failure, disseminated intravascular coagulation, and acute respiratory distress syndrome. *Med J DY Patil Univ.* 2013;6:197-9.
6. Rodrigues Sgrignolli L, Florido Mendes GE, Carlos CP, Burdmann EA, Sgrignolli R, Mazzali M, et al. Acute kidney injury caused by bothrops snake venom. *Nephron Clin Pract.* 2011;119:c131-6.
7. Athappan G, Balaji MV, Navaneethan U, Thirumalikalundusubramanian P, Prabhu R, Rajendran S, et al. Acute renal failure in snake envenomation: a large prospective study. *Saudi J Kidney Dis Transpl.* 2008;19:404-10.
8. Narvencar K, Faleiro R, Naik S, Dias A, Gomes M, Baretto J, et al. Correlation between timing of ASV administration and complications in snake bites. *J Assoc Physicians India.* 2006;54:717-9.
9. Government of India. National snakebite management protocol. New Delhi: Health & Family Welfare Department; 2007. Available from: http://www.dghs.gov.in/content/1362_3_NationalSnakebiteManagementProtocol.aspx
10. WHO/SEARO. Guidelines for the clinical management of snake bites in the Southeast Asian region. *Southeast Asian J Trop Med Public Health.* 1999;30(Suppl 1):1-85. Available from: <https://apps.who.int/iris/handle/10665/148668>

11. Bawaskar HS, Bawaskar PH, Punde DP, Inamdar MK, Dongare RB, Bhoite RR, et al. Profile of snakebite envenoming in rural Maharashtra, India. *J Assoc Physicians India*. 2008;56:88-95.
12. Sharma N, Chauhan S, Faruqi S, Bhat P, Varma S, Gupta R, et al. Snake envenomation in a North Indian hospital. *Emerg Med J*. 2005;22:118-20.
13. Halesha BR, Harshavardhan L, Lokesh AJ, Channaveerappa PK, Venkatesh KB, Kumar S, et al. A study on the clinico-epidemiological profile and the outcome of snake bite victims. *J Clin Diagn Res*. 2013;7:122-6.
14. Bhalla G, Mhaskar D, Agarwal A, Singh S, Sharma R, Rathod S, et al. A study of clinical profile of snake bite at a tertiary care centre. *Toxicol Int*. 2014;21:203-8.
15. Monteiro FNP, Kanchan T, Bhagavath P, Pradeep Kumar G, Menezes RG, Yoganarasimha K, et al. Epidemiology of cobra bite in Manipal, Southern India. *J Indian Acad Forensic Med*. 2010;32:224-7.