

# A Study of Clinical and Renal Profile in Poisonous Snake Bite Victims Attending a Tertiary Care Hospital in Assam

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## ABSTRACT

**Background:** Snakebite is a significant occupational and environmental hazard in Assam, with one of the highest reported incidences of snakebite in the country. Despite this burden, data on comprehensive clinical and renal profiles from this region remain limited.

**Objectives:** To evaluate clinical and renal parameters in poisonous snakebite patients and compare them with controls at a tertiary care hospital in Assam.

**Methods:** A hospital-based comparative observational study was conducted at Nagaon Medical College and Hospital (NMCH), Nagaon, Assam, over six months following ethics committee approval. Forty clinically confirmed poisonous snakebite patients (cases) were enrolled alongside forty age- and sex-matched healthy controls. Clinical examination was done and serum glucose, urea and creatinine were measured. Statistical analysis was performed using Student's t-test;  $p < 0.05$  was considered significant.

**Results:** The mean age of cases was  $35.45 \pm 13.56$  years and males predominated (57.5%). Haemotoxic envenomation was the most common type (56.7%). Serum urea ( $52.87 \pm 30.25$  vs  $38.16 \pm 13.36$  mg/dL) and creatinine ( $1.93 \pm 1.58$  vs  $0.85 \pm 0.25$  mg/dL) were significantly elevated in cases ( $p < 0.05$ ). Vomiting was seen in 55% of the cases. Elevated renal parameters were seen in 27.5% of cases.

**Conclusion:** Poisonous snakebite in Assam causes significant derangements in renal parameters with high chances of progression to renal failure. Renal involvement is a major morbidity determinant, especially in haemotoxic envenomation. Early biochemical screening and prompt management are essential to reduce snakebite-related mortality in this high-burden region.

**Keywords:** Snakebite, Envenomation, Renal profile, Acute kidney injury

## INTRODUCTION

Snakebite is a serious but neglected public health crisis, particularly in tropical and sub-tropical countries. Globally, an estimated 5.4 million snakebite incidents

occur annually, resulting in 1.8 to 2.7 million cases of envenomation with between 81,410 and 137,880 deaths each year.<sup>1</sup> In India, snakebite-related mortality is estimated at approximately 15,000 deaths

per annum, though underreporting in rural settings likely means the true burden is considerably higher.<sup>2</sup>

Assam and the broader Northeast Indian region carry a disproportionately high burden of snakebite. The terrain - comprising the Brahmaputra floodplain, tea gardens, agricultural fields, and dense forest borders - creates an environment highly conducive to human-snake encounters.

Incidence of snake bite in Assam is 35000 (Mohapatra et al 2011).<sup>3</sup> However, these statistics are inconclusive, as most snakebites occur in villages and forests and all victims may not reach hospital in time for management. The occupational groups most at risk include farmers, tea garden labourers, fishermen, and forest workers, many of whom work barefoot or with minimal protective clothing.

The major families in the India subcontinent are Elapidae which includes common cobra, king cobra and krait, Viperidae which includes Russell's viper, pit viper and saw-scaled viper and Hydrophidae (sea snakes) of the 52 poisonous species in India. Majority of bites and consequent morbidity is attributable to 5 species viz. Ophiophagus Hannah (king cobra), Najanaja (common cobra), Daboia russelii (Russell's viper), Bungarus caeruleus (krait) and Echiscarinatae (saw-scaled viper).<sup>4-6</sup>

Snake venoms are complex mixture of enzymatic and toxic proteins, which include phospholipase A2 (PLA2s), myotoxins, hemorrhagic metalloproteinases and other proteolytic enzymes, coagulant components, cardiotoxins, cytotoxins and neurotoxins.<sup>7,8</sup>

Different species have different types of venom which depends upon its species, geographical location, its habitat, climate, age etc. There are three types of venom according to its effect viz. Haemotoxic, Cytotoxic & Neurotoxic. • Haemo-toxic venoms are one which affects cardiovascular system • Cytotoxic venoms targets specific cellular sites • Neuro-toxic venoms harm nervous system of human body.<sup>9-11</sup>

Renal involvement is one of the most clinically significant and potentially fatal complications following envenomation, particularly by haemotoxic species. Acute kidney injury (AKI) following snakebite may result from direct nephrotoxicity of venom components, haemoglobin-induced renal tubular damage from intravascular haemolysis, disseminated intravascular coagulation (DIC), hypotension, and rhabdomyolysis.<sup>12</sup>

Despite the known severity of snakebite in Assam, there is a paucity of published data from this region on the comprehensive clinical and renal profile of envenomated patients. Existing studies from tertiary centres across India have documented diverse biochemical derangements, but region-specific data from Northeast India are crucial for informing local clinical protocols and resource allocation. This study was therefore undertaken to systematically assess the renal parameters in poisonous snakebite victims attending the emergency department of a tertiary care hospital in Assam, and to compare these findings with healthy controls.

#### **Aims and Objectives:**

1. To evaluate the clinical profile of poisonous snake bite patients.
2. To measure and compare blood urea and serum creatinine levels in poisonous snakebite cases and healthy controls.
3. To find out if there was any significant increase in the urea and creatinine levels as compared to controls.

## **MATERIALS & METHODS**

### **Study Design and Setting**

A hospital-based case control study was conducted at the Emergency Department of Nagaon Medical College and Hospital (NMCH), Nagaon, Assam, over a period of six months following approval by the Institutional Ethics Committee Ref No: MCI/IEC/NgMCH/2025/14 NMCH.

### Study Population and Sample Size

A total of 80 subjects were enrolled in two groups of 40 each: (a) Case group (n=40): Clinically diagnosed adult patients (aged  $\geq 18$  years) with poisonous snakebite history and positive signs/symptoms of envenomation, admitted via the emergency department. (b) Control group (n=40): Age- and sex-matched healthy volunteers with no history of acute or chronic illness.

### Inclusion Criteria

Cases were hospitalised adults with a history of snakebite and clinical features consistent with envenomation, including one or more of the following: local necrosis, ecchymosis, blistering, painful swelling; neurotoxic features (ptosis, diplopia, dysarthria, dyspnoea, paralysis); vasculotoxic features (bleeding, shock, acute kidney injury); or musculotoxic features (myalgia, muscle swelling, compartment syndrome).

### Exclusion Criteria:

1. Patients bitten by non-poisonous snakes.
2. Patients in whom the biting organism could not be identified.
3. Patients with previous history of co morbid illnesses like Chronic Kidney Disease, Heart Failure, and Diabetes.
4. Patients who had received anti-snake venom (ASV) prior to arrival were also excluded to avoid confounding of biochemical parameters.

**Clinical assessment** (Pulse, BP, general examination and systemic examination) was done upon arrival in the emergency department

### Laboratory Investigations

Blood samples were collected on admission to the emergency department (Day 0, prior to ASV administration). Control samples were collected under identical conditions.

Serum creatinine levels were estimated using modified Jaffes method. The normal range is 0.7-1.4 mg/dl. Blood urea was estimated using Urease –GLDH (Glutamate Dehydrogenase) method. Normal range at our laboratory is 15-40 mg/dL. Plasma Glucose was estimated based on Trinder's GOD/POD method. Normal range of Plasma Glucose (Random): 70 - 140mg/dl.

Demographic data (age, sex etc) were determined by existing standards, medical records, proper history taking.

### Statistical Analysis

Data were entered in Microsoft Excel and analysed using SPSS version 21.0. Continuous variables are presented as mean  $\pm$  standard deviation (SD). Categorical variables are presented as frequencies and percentages. Student's independent t-test was used for comparing continuous variables between cases and controls; Correlation between parameters was assessed using Pearson's correlation coefficient. A p-value of  $<0.05$  was considered statistically significant. Envenomation was classified as haemotoxic, neurotoxic, or mixed based on clinical presentation and laboratory findings.

## RESULT

### Demographic Profile

Of the 40 cases, the mean age was  $35.45 \pm 13.56$  years (range: 18-80 years). The male-to-female ratio was approximately 1.3, with 23 males (57.5%) and 17 females (42.5%).

**Table 1: Mean AGE and Gender distribution of cases and controls**

Parameter	Cases (n=40)	Controls (n=40)
Age (years), Mean $\pm$ SD	35.45 $\pm$ 13.56	37.33 $\pm$ 9.90
Sex - Male, n (%)	23 (57.5%)	23 (57.5%)
Sex - Female, n (%)	17 (42.5%)	17 (42.5%)

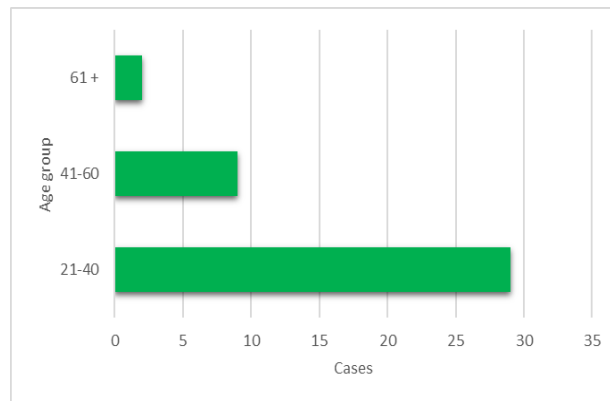


Figure 1: Age Distribution of cases

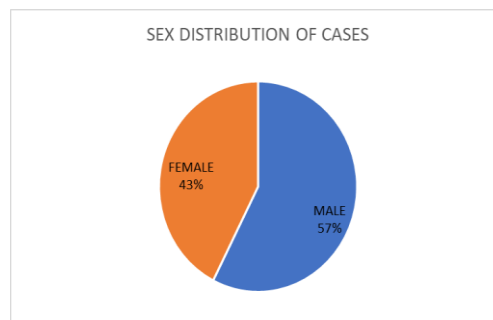


Figure 2: Sex distribution of cases

Haemotoxic envenomation was the most common type (56.7%), followed by Neurotoxic (23.3%) and mixed/unclassified (20%)

Table 2: Distribution of envenomation type in case group

Type of Envenomation	Number (n=40)	Percentage (%)
Haemotoxic	22	56.7%
Neurotoxic	10	23.3%
Mixed / Unclassified	8	20%
Total	40	100%

Table 3: Clinical features at presentation

Presenting complaints	Number (n=40)	Percentage (%)
Fang marks	40	100
Vomiting	22	55
Bleeding	15	37.5
Hypotension	8	20
Ptosis	10	25
Respiratory distress	8	20
Paralysis	8	20

### Renal Parameters

Serum urea was significantly elevated in cases compared to controls ( $52.87 \pm 30.25$

vs  $38.16 \pm 13.36$  mg/dL;  $p < 0.05$ ). Serum creatinine was similarly elevated ( $1.93 \pm 1.58$  vs  $0.84 \pm 0.25$  mg/dL;  $p < 0.001$ ).

**Table 4: Comparison of renal parameters and between cases and controls**

Parameter	Cases (Mean ± SD)	Controls (Mean ± SD)	p-value
Serum Urea (mg/dL)	52.87 ± 30.25	38.16 ± 13.36	<0.05
Serum Creatinine (mg/dL)	1.93 ± 1.58	0.84 ± 0.25	<0.001

## DISCUSSION

This study systematically evaluated the clinical and biochemical profile of 40 poisonous snakebite victims at a tertiary care hospital in Assam, providing region-specific data that has been largely absent from the published literature on Northeast India. The findings confirm that envenomation causes widespread and clinically significant derangements across renal parameters.

The predominance of male patients (57.5%) in the case group is consistent with findings from comparable Indian studies.<sup>13,14</sup> In the Nagaon district of Assam, large-scale cultivation of paddy and jute, together with tea estate work, brings the locals into frequent contact with snake habitats, especially during the monsoon season. The incidence of snakebites in this study was (57.5%) in males compared to females (42.5%). Similarly, Dharod et al.<sup>4</sup> reported the incidence of snake bites in 61% of males and 39% of females. Raghavendra et al.<sup>7</sup> in a similar study reported that 66.4% male victims and male predominant victims were also reported by Vallidevi et al.<sup>8</sup>

In the present study, 72.5% of the victims of snake bites were between 21-40 years of age, the mean age was 35 years compared to Dharod et al.,<sup>4</sup> where mean age of the cases was 42.2 years. Harshvardhan L et al., found majority with AKI had a mean age of 41.2 years.<sup>15</sup> Singh et al found that of the 138 patients of venomous snake bite, 62 developed AKI (44.92%). Patients who developed AKI were older in age.<sup>16</sup>

In our study we couldn't significantly correlate the deranged renal parameters to a particular age group.

In the present study, 100% of the patients showed fang marks. Harshvardhan L et al.,<sup>16</sup> reported fang marks in 93% of cases, followed by tenderness in 76% of cases. The

presence of fang marks is important for the identification of the species of snake that could have been involved and for delivering the treatment appropriately.

Bleeding tendencies were seen in 37.5 % of cases. Vomiting was seen in 55% cases. Pushpalatha, et al accounted bleeding tendencies to be 79.4% of cases.<sup>17</sup> Athappan G et al. and Harshvardhan L et al. demonstrated bleeding manifestations among 27.7% and 38.8% subjects with AKI respectively.<sup>18,15</sup>

The elevated renal parameters in 27.5% of the cases in our study is consistent with data from other Indian tertiary centres and reflects the nephrotoxic potential of both haemotoxic and neurotoxic venoms. The factors for snake bite induced AKI is likely attributable to the combined effects of disseminated intravascular coagulation (DIC), haemoglobin-induced tubular toxicity from intravascular haemolysis, and direct nephrotoxic venom components. This pattern aligns with reports from Kapoor's observational study from the sub-Himalayan region, which documented progressive elevation of creatinine and urea over the first four days following haemotoxic snakebite.<sup>19</sup>

Acute kidney injury proposed by Acute Kidney Injury Network [Modified RIFLE Criteria] [6] which defines AKI as an "abrupt (within 48 hours) absolute increase in the serum creatinine concentration of  $\geq 0.3$  mg/dl (26.4 $\mu$ moles/l) from baseline, a percentage increase in the serum creatinine concentration of  $\geq 50$  % or oliguria of  $<0.5$  ml/kg/hr  $> 6$  hours. Glomerular Filtration Rate (GFR)  $<60$  mL/min/1.73 m<sup>2</sup> with in the first 72 hr after snakebite was defined as AKI.<sup>20</sup>

Snakebite-induced AKI has been reported in several Chinese centers. Suchithra et al.<sup>10</sup> showed the prevalence of AKI in 25.5% of

cases and Dharod et al.<sup>4</sup> showed AKI in 30.96% of snakebite cases. However, Panchalwar et al.<sup>11</sup> reported a higher prevalence of 31.5%, while Harshvardhan et al.<sup>15</sup> reported 41% of snakebite cases. Variations in the frequency of AKI could be due to the type of snake venom involved. Some snake species are more likely to cause AKI because they are more venomous than others. The second factor could be variations in access to healthcare, antivenom administration, and supportive care, which can impact patient outcomes, including the development of AKI.

Our finding aligns with the emerging concept of the AKI-to-CKD continuum, where survivors of severe AKI remain at risk of long-term renal impairment.<sup>21</sup> Snakebite nephropathy, therefore, contributes not only to acute mortality but also to the burden of chronic kidney disease in endemic regions.<sup>22</sup>

As per Kakati H et al 66.19% of venomous snake bites accounted for viper bites in Assam.[23] Following the viper bites, various systemic symptoms occur as a result of coagulopathy, hemolysis, acute kidney injury, a generalized increase in capillary permeability, rhabdomyolysis, neurotoxicity etc.<sup>24</sup>

The kidney being a highly vascularized organ with excretory function is prone to toxicity of the venom and acute kidney injury is the most significant of all the renal manifestations.<sup>24-26</sup> AKI in snakebite constitutes 3.0% of total AKIs in India.<sup>27</sup> As a result of intravascular hemolysis and rhabdomyolysis, hemoglobinuria and myoglobinuria develops contributing to the development of AKI after snake bite.<sup>25</sup> Tubular and cortical necrosis are the important causes of AKI and is usually reversible.<sup>5</sup> Bleeding and circulatory collapse in snake-bite victims is usually due to DIC.<sup>18</sup> Increased time intervals between bite to the administration of ASV (bite to needle time) increases the risk of developing AKI. Until the venom is neutralized, it continues the damage.<sup>16</sup>

A strength of this study is its systematic case-control design with clearly defined inclusion and exclusion criteria and standardised laboratory protocols. Limitations include the single-centre design, cross-sectional measurement of most parameters and absence of snake identification data in a subset of cases. Future multicentre studies from Northeast India with longer follow-up provide more comprehensive evidence base for clinical guidelines specific to this region.

## CONCLUSION

Poisonous snakebite in Assam causes profound multi-systemic biochemical derangements. Renal involvement is particularly pronounced in haemotoxic envenomation, with AKI occurring in nearly one-third of such patients. Early and comprehensive biochemical assessment is essential for risk stratification and timely management in the emergency setting. Region-specific data such as this study provides are critical to informing local clinical protocols and training initiatives for snakebite management in Northeast India.

## Declaration by Authors

**Ethical Approval:** Approved by Institutional Ethics Committee, Nagaon Medical College and Hospital, Assam No: MCI/IEC/NgMCH/2025/14

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**Conflict of Interest:** The authors declare no conflict of interest.

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