RESEARCH PAPER

Snakebite-Associated Acute Kidney Injury with Divergent Clinical Courses: A Three-Patient Case Series

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ABSTRACT

Background: Snakebite envenomation remains a major yet preventable cause of acute kidney injury (AKI) in tropical regions, particularly in South Asia. Renal involvement after hematotoxic and myotoxic envenomation spans a broad spectrum, from transient creatinine elevation to dialysis-dependent kidney failure. Clinical severity is often shaped not only by venom toxicity but also by delayed presentation, hemodynamic stress, metabolic derangement, and exposure to nephrotoxic agents.

Case series: We describe three adults with snakebite-associated acute kidney injury (AKI) managed in a tertiary-care emergency department. Case 1 was a 50-year-old woman who presented 10 days after an unidentified snakebite with KDIGO stage 3 AKI, severe azotemia, metabolic acidosis, diabetic ketoacidosis, persistent venom-induced coagulopathy, and need for hemodialysis. Toxicologic analysis of indigenous medication identified mefenamic acid, suggesting a possible nephrotoxic co-exposure within a multifactorial pattern of kidney injury. Case 2 was a 60-year-old man referred after receiving 30 vials of polyvalent antivenom at an outside hospital; he had mild non-oliguric AKI with a normal coagulation profile and improved with supportive care. Case 3 was a 38-year-old man with rhabdomyolysis-associated AKI characterized by myalgia, dark urine, elevated creatine phosphokinase, positive urine myoglobin, hyperkalemia, and metabolic acidosis; he improved with early antivenom, aggressive hydration, urinary alkalinization, and potassium-lowering therapy.

Conclusion These cases demonstrate that snakebite-associated AKI is mechanistically heterogeneous and frequently multifactorial. Early bedside coagulation testing, serial renal monitoring, timely antivenom, judicious fluid resuscitation, and avoidance of nephrotoxic exposures are critical in limiting progression to dialysis-requiring kidney failure. Recognizing secondary insults is essential for risk stratification and outcome optimization.

Keywords snakebite envenomation; acute kidney injury; antivenom; rhabdomyolysis; hemodialysis; India

How to cite this: DR AMEEN UMER P, DR S BALAJI, DR HARI PRASAD, DR RAJARAJESHWARAN K, Snakebite-Associated Acute Kidney Injury with Divergent Clinical Courses: A Three-Patient Case Series, Int J Drug Deliv Technol. 2026;16(52s): 953-960. DOI: 10.25258/ijddt.16.52s.120

Source of support: Nil., **Conflict of interest:** None

Abbreviations

Abbreviation	Expansion
AKI	Acute kidney injury
ASV	Anti-snake venom
CK	Creatine kinase
CPK	Creatine phosphokinase
DKA	Diabetic ketoacidosis
KDIGO	Kidney Disease: Improving Global Outcomes
LDH	Lactate dehydrogenase
NSAID	Non-steroidal anti-inflammatory drug
POCUS	Point-of-care ultrasound

1. Introduction

Snakebite envenomation is a recognized neglected tropical disease that disproportionately affects rural populations in South Asia, where occupational exposure during agricultural activities, delayed access to healthcare facilities, and reliance on informal or

traditional care pathways contribute substantially to morbidity and mortality [1–3]. India accounts for a considerable share of the global snakebite burden, and acute kidney injury (AKI) remains one of the most important and potentially life-threatening systemic complications of envenomation, often contributing to prolonged hospitalization, need for renal replacement therapy, and adverse clinical outcomes [1–4].

Snakebite-associated AKI is increasingly understood as a complex and multifactorial clinical syndrome rather than the result of a single pathogenic insult. Proposed mechanisms include direct nephrotoxicity, venom-induced consumptive coagulopathy, thrombotic microangiopathy, hemolysis, rhabdomyolysis, capillary leak, hypotension, and renal ischemia [4,5]. In routine clinical practice, however, renal injury severity is likely shaped not only by venom-related factors but also by potentially modifiable contributors such as delayed antivenom administration, volume depletion, metabolic stress, superimposed infection, and exposure to nephrotoxic medications [5,6]. As a result, patients with apparently similar envenomation histories may exhibit markedly different renal presentations and outcomes.

Although snakebite-associated AKI has been well recognized, bedside reports that clearly contrast differing renal phenotypes within a clinically interpretable framework remain limited. Case-based descriptions can therefore be valuable in illustrating how contextual and management-related factors may modify the course of renal injury beyond venom toxicity alone. In particular, highlighting these differences may help clinicians identify early warning signs, recognize potentially aggravating exposures, and anticipate the need for escalation of supportive care.

In this case series, we describe three adult patients with snakebite-associated AKI who demonstrated distinctly different clinical trajectories: severe dialysis-requiring AKI following delayed presentation and probable nephrotoxic co-exposure, mild and fully reversible AKI after relatively earlier treatment, and pigment nephropathy in the setting of rhabdomyolysis. By presenting these contrasting patterns, this series aims to emphasize clinically actionable determinants of renal outcome and to underscore the importance of timely recognition, early supportive management, and careful evaluation of coexisting modifiers of kidney injury [7,8].

Case Series Design and Reporting

We conducted a retrospective descriptive case series of three selected adult patients with snakebite-associated acute kidney injury (AKI) managed in the Emergency Department of SRM Medical College Hospital and Research Centre between January 2025 and January 2026. These cases were intentionally chosen to reflect distinct clinical trajectories and renal injury patterns following snakebite envenomation, and therefore should be interpreted as illustrative rather than consecutive cases. Clinical information was retrospectively compiled from contemporaneous case records, serial laboratory data, bedside monitoring documentation, treatment charts, and discharge summaries. Post-discharge follow-up data were not available. No major missing data were identified in the records reviewed for the present series. In none of the cases was the snake species definitively identified.

Summary Tables

Table 1. Baseline characteristics and dominant clinical phenotype across the three cases.

Variable	Case 1	Case 2	Case 3
Age/sex	50/F	60/M	38/M
Major comorbidities	Type 2 diabetes mellitus	Hypertension, hypothyroidism, remote cerebrovascular accident	None reported
Time from bite to tertiary presentation	10 days	5 days	18 hours
Bite site	Right middle finger	Dorsum of right foot	Left calf
Probable dominant syndrome	Hematotoxic with persistent coagulopathy	Treated hematotoxic envenomation with mild renal dysfunction	Myotoxic syndrome with rhabdomyolysis
Key presenting symptoms	Oliguria, abdominal distension, constipation, exertional dyspnea	No bleeding, hematuria, oliguria, or neurotoxic	Myalgia, cola-colored urine, reduced urine

Variable	Case 1	Case 2	Case 3
		symptoms	output
Local examination	Healed fang mark; minimal edema; no necrosis	Residual bite mark; minimal swelling; no necrosis	Fang marks with edema to mid-leg; no compartment syndrome
Bedside coagulation test	WBCT non-clotting at 20 min	WBCT clotted at 20 min	WBCT clotted at 18 min
Urine output status	<25 mL over prior 12 h	Preserved	Initially 0.4 mL/kg/h
Most likely AKI mechanism	Multi-hit injury: venom effect + DKA + NSAID exposure + delayed care	Mild reversible tubular/prerenal insult after treated envenomation	Pigment nephropathy from rhabdomyolysis

Table 2. Key laboratory and imaging features at presentation.

Parameter	Case 1	Case 2	Case 3
Serum creatinine at presentation	20.6 mg/dL	1.4 mg/dL	2.2 mg/dL
Highest documented creatinine	20.6 mg/dL	Approx. 1.6 mg/dL (outside report)	2.6 mg/dL (Day 1)
Blood urea / serum urea	201 mg/dL	43 mg/dL	62 mg/dL
Acid-base status	Severe high-anion-gap metabolic acidosis with ketonemia	Near-normal ABG	Metabolic acidosis (pH 7.31, HCO3 17 mEq/L)
Potassium	Not specified in source notes	3.0 mEq/L	5.8 mEq/L
Hemolysis / myolysis markers	LDH 966 U/L; fragmented RBCs on smear	No biochemical evidence of hemolysis	CPK 18,500 IU/L; LDH 920 U/L; urine myoglobin positive
Urinalysis	Abundant RBCs; no significant proteinuria	No proteinuria, hematuria, casts, or myoglobinuria	2+ protein; 10-15 RBC/HPF; granular casts
Imaging	Bilateral grade II renal parenchymal changes; no obstruction	CT: cystitis; kidneys preserved. USG day 3 normal	USG: normal kidneys; no obstruction
Other notable findings	Blood ketones 1.3 mmol/L; persistent coagulopathy	Stable platelets and coagulation profile	Peaked T waves on ECG; volume depletion on POCUS

Table 3. Management strategies and short-term outcomes.

Management / outcome	Case 1	Case 2	Case 3
Antivenom	10 vials IV on arrival	30 vials IV before referral	10 vials IV on arrival
Renal-supportive strategy	DKA protocol, dialysis catheter, urgent hemodialysis	Cautious isotonic fluids and serial monitoring	Aggressive isotonic fluids and urinary alkalinization
Other key interventions	Insulin infusion, bicarbonate, nephrology consultation	Antibiotics, glucose control, antihypertensive continuation	Calcium gluconate, insulin-dextrose, potassium monitoring

Management / outcome	Case 1	Case 2	Case 3
Need for dialysis	Yes; 3 sessions	No	No
Short-term response	Improved urine output and metabolic status after dialysis	Creatinine fell from 1.4 to 1.3 mg/dL with preserved urine output	Potassium normalized and urine output improved by day 2
Discharge status	Discharged day 6 with improving renal function	Discharged after clinical and biochemical stabilization	Discharged day 7; creatinine 1.6 mg/dL
Take-home message	Secondary insults can convert envenomation into dialysis-requiring AKI	Some cases remain mild and fully reversible with supportive care	Rhabdomyolysis should be actively sought and treated early

Case Presentations

Case 1. Severe Dialysis-Requiring AKI after Delayed Presentation, Diabetic Ketoacidosis, and Inadvertent NSAID Exposure

A 50-year-old woman with type 2 diabetes mellitus presented 10 days after an unidentified snakebite to the right middle finger sustained during agricultural work. She did not seek formal medical attention immediately after the bite. On the second day after envenomation, after visible fang marks were noted, she consulted a traditional Siddha practitioner and received indigenous oral medication for four consecutive days. During this period, her routine oral hypoglycemic therapy was discontinued. She subsequently developed progressive oliguria, abdominal distension, constipation, and exertional dyspnea, and was referred to the tertiary emergency department after renal dysfunction was identified at a peripheral facility. At presentation, she was conscious, oriented, and febrile, with pallor and minimal bilateral basal crepitations. Examination of the bite site revealed a healed fang mark over the right middle finger with minimal residual edema and no evidence of local necrosis or compartment syndrome. Urine output during the preceding 12 hours was less than 25 mL, consistent with severe oliguric AKI. Initial arterial blood gas analysis demonstrated severe high-anion-gap metabolic acidosis with positive blood ketones (1.3 mmol/L), consistent with diabetic ketoacidosis. Serum creatinine was 20.6 mg/dL and blood urea was 201 mg/dL. Whole blood clotting time remained non-clotting at 20 minutes despite delayed presentation, suggesting persistent venom-induced coagulopathy. Lactate dehydrogenase was elevated to 966 U/L, peripheral smear showed occasional fragmented red cells, and urinalysis demonstrated numerous red blood cells without significant proteinuria. Renal ultrasonography with Doppler evaluation showed bilateral grade II renal parenchymal changes without evidence of renal artery stenosis or obstructive uropathy.

She received 10 vials of intravenous polyvalent antivenom over 1 hour, after which the whole blood clotting time normalized on reassessment. Concurrently, a diabetic ketoacidosis protocol was initiated with insulin, intravenous fluids, and bicarbonate for severe acidosis. In view of refractory metabolic derangement, severe azotemia, and persistent oliguria, a right internal jugular dialysis catheter was inserted and urgent hemodialysis was initiated in the emergency department. Two hemodialysis sessions were performed on the day of admission, followed by a third session on ICU Day 3, after which urine output and biochemical parameters gradually improved. Medication containers supplied by the Siddha practitioner were retrieved and submitted for toxicologic analysis. Gas chromatography identified mefenamic acid in one sample. Although a direct causal contribution cannot be established from a single case, this finding suggests a plausible nephrotoxic co-exposure in a patient already at high risk of kidney injury because of delayed presentation, persistent coagulopathy, severe azotemia, and diabetic ketoacidosis. Other submitted samples showed predominantly hydrocarbon- and solvent-type compounds without a more specific explanatory link to the severity of renal dysfunction. Overall, the case was interpreted as severe multifactorial AKI in which venom-mediated injury likely interacted with metabolic stress and possible nephrotoxic exposure.

She was transferred out of intensive care on day 3 and discharged on hospital day 6 with improving renal function, recovering urine output, and resolution of the

most severe metabolic abnormalities.

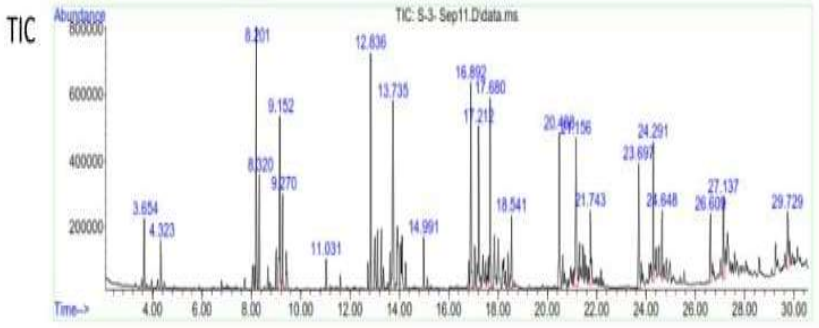

Table 4. Condensed toxicologic findings from indigenous medication used in Case 1.

Submitted sample	Notable reported compounds	Clinical interpretation
Sample 1	Mefenamic acid; diethyl phthalate; bis(tridecyl) phthalate; other minor compounds	Mefenamic acid is the most clinically relevant finding and provides a plausible nephrotoxic co-factor that may have amplified venom-related renal injury.
Sample 2	Predominantly hydrocarbon/solvent-type compounds (e.g., heptane, dodecane, undecane, tetradecene derivatives)	Qualitative profile does not establish a specific alternative explanation for AKI; likely of secondary relevance compared with venom effect and NSAID exposure.
Sample 3	Predominantly hydrocarbon and aliphatic compounds with uncertain toxicologic significance in this setting	Findings are exploratory and best interpreted as supportive toxicology rather than definitive causation.

Case 2. Mild Non-Oliguric AKI after Prior High-Dose Antivenom and Subacute Referral

A 60-year-old man with hypertension, hypothyroidism on replacement therapy, and a remote cerebrovascular accident with full recovery presented on the fifth day after a snakebite to the dorsum of the right foot. He had initially been admitted to a peripheral hospital for cellulitis and suspected hematotoxic envenomation, where he received a total of 30 vials of polyvalent antivenom over four days before referral for evaluation of renal dysfunction. At presentation, he denied bleeding manifestations, hematuria, oliguria, neurotoxic symptoms, or progressive limb swelling. Structured primary assessment showed a patent airway, spontaneous breathing with preserved oxygen saturation on room air, blood pressure of 170/100 mmHg, and a Glasgow Coma Scale score of 15/15. The bite mark remained visible with only minimal local swelling and no necrosis, blistering, or compartment tension. Whole blood clotting time was clotted at 20 minutes, indicating no active consumptive coagulopathy. Arterial blood gas values were essentially normal apart from hypokalemia (potassium 3.0 mEq/L). Admission hemoglobin was 14.2 g/dL, leukocyte count 10,570/mm3, platelet count 211,000/mm3, serum urea 43 mg/dL, blood urea nitrogen 23 mg/dL, and serum creatinine 1.4 mg/dL. CK-MB, liver function tests, and coagulation parameters were within normal limits. Urinalysis showed no proteinuria, hematuria, casts, or myoglobinuria. POCUS demonstrated relative euvolemia with inferior vena cava collapsibility below 50% and preserved left ventricular systolic function. Computed tomography showed cystitis with bladder wall thickening but no renal infarction, obstruction, or perinephric collection. Follow-up abdominal ultrasonography on day 3 showed no features of medical renal disease. He was managed conservatively with carefully titrated isotonic fluids, antimicrobial therapy for cystitis and recently treated cellulitis, continuation of antihypertensive therapy, and insulin-based glucose control. No indication for dialysis emerged. Renal indices remained stable and improved gradually, with creatinine decreasing to 1.3 mg/dL and urine output remaining preserved throughout admission. He was stepped down to the ward on day 3 and discharged after continued clinical improvement.

This case is most consistent with mild, reversible AKI in the setting of treated envenomation, possibly reflecting limited tubular injury or a transient prerenal component rather than established intrinsic renal damage.

Case 3. Rhabdomyolysis-Associated AKI with Pigment Nephropathy

A 38-year-old previously healthy male agricultural worker presented approximately 18 hours after a snakebite to the left calf sustained while working in paddy fields. He had not received antivenom before arrival. After initial local pain and swelling, he developed generalized myalgia, cola-colored urine, and falling urine output over the 8 hours before presentation. On arrival, airway and breathing were stable, oxygen saturation was 98% on room air, pulse rate was 104/min, and blood pressure was 110/70 mmHg. He was fully conscious, neurologically intact, and had diffuse muscle tenderness. Two fang puncture marks with edema extending to the mid-leg were visible, without blistering, necrosis, or compartment syndrome. Urine passed in the emergency department was dark brown. Whole blood clotting time was clotted at 18 minutes. Arterial blood gas analysis showed metabolic acidosis (pH 7.31; bicarbonate 17 mEq/L) with partial respiratory compensation and mild hyperkalemia. Serum creatinine was 2.2 mg/dL, urea 62 mg/dL, potassium 5.8 mEq/L, CPK 18,500 IU/L, LDH 920 IU/L, AST 145 IU/L, and ALT 62 IU/L. Urine myoglobin was positive. Urinalysis showed 2+ protein, 10-15 red blood cells per high-power field, and granular casts without dysmorphic red cells. Electrocardiography demonstrated peaked T waves consistent with hyperkalemia. Ultrasonography showed normal-sized kidneys without obstruction. POCUS suggested relative intravascular volume depletion.

He received 10 vials of intravenous polyvalent antivenom, aggressive isotonic saline hydration, sodium bicarbonate infusion for urinary alkalinization, and standard potassium-lowering therapy including calcium gluconate and insulin with dextrose. Strict hourly urine monitoring was instituted. Although creatinine rose modestly to 2.6 mg/dL on day 1, potassium normalized and urine output improved to 1.1 mL/kg/h by day 2. CPK declined from a peak of 21,400 IU/L to 6,800 IU/L by day 4, urine color normalized, and renal function improved without renal replacement therapy. He was discharged on day 7 with serum creatinine of 1.6 mg/dL and planned nephrology follow-up.

This case illustrates a myotoxic phenotype of snakebite-associated AKI in which early recognition of rhabdomyolysis, prompt volume resuscitation, urinary alkalinization, and potassium control likely prevented progression to dialysis-requiring kidney injury.

Discussion

This case series illustrates the marked clinical heterogeneity of snakebite-associated acute kidney injury (AKI) and suggests that renal outcome may be determined not only by venom-related toxicity but also by secondary, and in some instances potentially modifiable, aggravating factors. The three patients described here followed distinct clinical trajectories: severe dialysis-requiring AKI in the setting of delayed definitive care and multiple metabolic and treatment-related stressors, mild non-oliguric and reversible AKI after relatively earlier treatment, and pigment nephropathy associated with rhabdomyolysis. Taken together, these cases underscore that snakebite-associated AKI should be approached as a multifactorial clinical syndrome rather than a uniform complication of envenomation.

The pathophysiology of AKI following snakebite is complex and likely reflects the interaction of several overlapping mechanisms. Direct tubular toxicity, venom-induced consumptive coagulopathy, thrombotic microangiopathy, capillary leak, hemolysis, rhabdomyolysis, hypotension, and renal ischemia have all been implicated, with acute tubular necrosis reported as the most common histopathologic finding in many series [4–8]. Observational and histopathologic data from India further suggest that cortical necrosis and thrombotic microangiopathy may be associated with more severe renal injury and poorer renal recovery [7]. In practice, however, the final renal phenotype is often shaped by additional clinical modifiers that are not directly venom-mediated.

Case 1 is notable for the convergence of several high-risk features that may plausibly have contributed to severe renal dysfunction. In addition to delayed presentation and persistent coagulopathy consistent with ongoing systemic envenomation, this patient had diabetic ketoacidosis, likely volume depletion, and exposure to an indigenous preparation in which mefenamic acid was identified. Although a direct causal contribution of the NSAID cannot be definitively established in a single case, it represents a biologically plausible nephrotoxic co-factor in a hemodynamically stressed patient. In such settings, prostaglandin inhibition may reduce afferent arteriolar vasodilation and thereby exacerbate renal hypoperfusion and ischemic tubular injury. This case therefore highlights the importance of medication reconciliation, early recognition of coexisting metabolic stressors, and careful assessment of therapies received prior to referral, particularly in patients initially managed outside formal healthcare systems.

Case 2 demonstrates that not every post-snakebite elevation in serum creatinine necessarily reflects severe intrinsic renal damage. The preserved urine output, absence of marked urinary abnormalities, normal coagulation profile at presentation, lack of evidence of rhabdomyolysis, and spontaneous improvement in renal indices with supportive care suggest a limited and reversible insult. This distinction is clinically relevant because it supports a measured approach centered on serial reassessment, supportive therapy, and close monitoring rather than premature escalation based solely on a modest creatinine rise. The case reinforces the need to interpret renal dysfunction in the context of the overall clinical picture rather than as an isolated laboratory abnormality.

Case 3 illustrates a predominantly myotoxic pattern of

envenomation complicated by rhabdomyolysis, pigmenturia, hyperkalemia, metabolic acidosis, and AKI. In pigment nephropathy, myoglobin may contribute to tubular obstruction, intrarenal vasoconstriction, and oxidative injury, particularly in the setting of aciduria and volume depletion. The management priorities in such cases extend beyond antivenom administration and include aggressive fluid resuscitation when clinically appropriate, close biochemical surveillance, and prompt treatment of electrolyte derangements, especially hyperkalemia [4,6,8]. This case broadens the clinical message of the series by demonstrating that snakebite-associated AKI may also arise through a predominantly myotoxic route rather than through coagulopathic or ischemic mechanisms alone.

Across all three cases, a consistent practical message is the value of structured early assessment in the emergency setting. Initial stabilization should be accompanied by targeted evaluation for systemic envenomation and evolving renal injury, including bedside coagulation assessment, serial renal biochemistry, urine output monitoring, electrolyte surveillance, and focused hemodynamic evaluation. Delayed presentation and delayed antivenom administration have been associated with worse renal outcomes in observational studies [5,6], and our cases are broadly consistent with that pattern. They also emphasize the need for clinicians to actively inquire about traditional, indigenous, or over-the-counter remedies, as such exposures may complicate the clinical picture and introduce avoidable nephrotoxic risk.

An additional point of clinical importance is that apparent short-term improvement does not necessarily exclude longer-term renal sequelae. Follow-up studies suggest that a proportion of survivors of snakebite-associated AKI subsequently develop chronic kidney disease, hypertension, or persistent impairment in glomerular filtration, particularly after severe or dialysis-requiring episodes [6,9,10]. Although follow-up data were not available in the present series, these observations support the need for post-discharge surveillance of serum creatinine, blood pressure, and urinalysis, especially in patients with severe AKI or other high-risk features during admission.

This case series has several limitations. The offending snake species could not be definitively identified in any of the three cases, which limits species-specific interpretation. The sample size is small and the cases were selected for illustrative value; accordingly, the findings should be interpreted as descriptive rather than generalizable. No kidney biopsy was performed in the patient with severe dialysis-requiring AKI, precluding histopathologic confirmation of the underlying renal lesion. Pre-bite baseline renal function was also unavailable, limiting assessment of pre-existing renal susceptibility. In addition, outcomes were confined to the in-hospital course, as no post-discharge follow-up data were available. Interpretation is further constrained by potential confounding from comorbid diabetes and treatment exposures prior to presentation, including outside or non-standard therapies. Despite these limitations, the contrasting trajectories observed in these three patients provide a clinically useful framework for recognizing distinct presentations of snakebite-associated renal injury, including dialysis-requiring multi-hit AKI, mild reversible AKI, and rhabdomyolysis-associated pigment nephropathy.

In conclusion, this series highlights that snakebite-associated AKI is not a uniform entity but a spectrum of renal syndromes shaped by both venom effects and contextual clinical modifiers. Early recognition, timely supportive care, prompt antivenom administration when indicated, and identification of potentially aggravating co-exposures may influence renal trajectory in meaningful ways. Framing snakebite-associated AKI through this broader clinical lens may assist emergency physicians and nephrologists in anticipating severity, refining management, and planning follow-up after hospital discharge.

Declarations

Ethics approval:

Consent for publication:

Funding:

Conflicts of interest: The authors declare no competing interests.

Author contributions:

Data availability:

Figure Legends

Figure 1. Bite-site findings in Case 1 showing a visible fang mark over the right middle finger at delayed presentation.

Figure 2. Photograph of the indigenous medication containers consumed in Case 1 before hospital presentation and later submitted for toxicologic analysis.

Figure 3. Composite gas chromatography figure from Case 1 toxicologic analysis.

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