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RENAL CONSEQUENCES OF VENOMOUS ANIMAL INJURIES: FOUR CLINICAL CASES OF ACUTE KIDNEY INJURY AND SEVERE CHRONIC KIDNEY DISEASE DECOMPENSATION

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Abstract

Background: Venomous animal injuries remain a relevant cause of severe systemic disease in tropical and subtropical settings. Renal involvement may occur both in previously healthy kidneys, leading to acute kidney injury (AKI), and in chronically diseased kidneys, precipitating severe decompensation and dialysis dependence.

Objective: To describe four clinical cases illustrating the spectrum of renal involvement associated with venomous animal injuries, ranging from dialysis-requiring AKI to severe decompensation of pre-existing chronic kidney disease (CKD).

Cases: We report four patients treated at a regional referral hospital in Paraguay. Case 1 involved a 44-year-old man with Loxosceles bite complicated by necrotizing fasciitis, septic shock, and AKI requiring 10 hemodialysis sessions, with fatal outcome after referral. Case 2 was a 33-year-old man with multiple wasp stings who developed anuric AKI, severe metabolic acidosis, hyperkalemia, respiratory failure, and death after 9 hemodialysis sessions. Case 3 involved an 81-year-old man with Bothrops snakebite and severe AKI requiring 12 hemodialysis sessions, with subsequent clinical and biochemical improvement. Case 4 was a 48-year-old man with probable spider bite and soft tissue infection, in whom advanced hypertensive CKD suffered severe metabolic and uremic decompensation, requiring repeated hemodialysis and progressi on toward chronic dialysis support.

Conclusion: Natural venomous injuries can severely affect renal function through different mechanisms, including direct and indirect nephrotoxicity, hemodynamic compromise, sepsis, inflammatory injury, and metabolic derangement. Their impact extends from severe AKI in previously preserved renal function to profound decompensation in pre-existing CKD. Early recognition, laboratory monitoring, and timely nephrology intervention are essential.Venomous animal injuries may cause severe renal dysfunction through different mechanisms, including direct and indirect

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nephrotoxicity, hemodynamic compromise, sepsis, inflammatory injury, and metabolic derangement. Beyond illustrating the spectrum from severe AKI to decompensated CKD, these cases emphasize two clinically relevant lessons: delayed presentation may amplify severity, and preserved diuresis does not exclude advanced renal dysfunction. Early recognition, laboratory monitoring, and timely nephrology intervention are essential.

Introduction:-

Venomous animal bites and stings are a persistent public health problem in many regions of the world and may produce a wide range of local and systemic manifestations. Although skin lesions, coagulopathy, anaphylaxis, hemolysis, and soft tissue destruction are among the best-known complications, renal involvement is one of the most severe consequences because it may determine short-term prognosis and long-term survival (1,2).The kidney is particularly vulnerable to the biological aggression imposed by envenomation. Depending on the offending species and host factors, renal dysfunction may result from direct nephrotoxicity, pigment-mediated tubular injury, hypovolemia, shock, intravascular hemolysis, rhabdomyolysis, coagulopathy, systemic inflammation, or secondary sepsis. In Paraguay, particularly in border and rural regions, Bothrops-related accidents are among the most relevant venomous injuries and have been associated with important renal complications (3–8). Loxosceles bites may be followed by tissue necrosis, hemolysis, and kidney injury, whereas severe hymenoptera exposure may produce systemic toxicity, allergic phenomena, pigment nephropathy, and hemodynamic compromise (9–11).

The most recent evidence suggests that kidney injury associated with venomous animals should not be understood as a late and isolated complication, but rather as a dynamic biological syndrome in which direct toxicity, inflammation, hemolysis, rhabdomyolysis, and microvascular dysfunction converge. In 2023, **Yu** highlighted that, in wasp envenomation, acute kidney injury may arise from both the direct nephrotoxicity of venom and from hemolysis and rhabdomyolysis, whereas Arshadshowed clinically that such stings may progress to severe acute renal failure requiring renal replacement therapy (12,13). At the mechanistic level, **Li** implicated the NLRP3 inflammasome in wasp venom-induced kidney injury, Cheng **d**ocumented complement activation, and Zho**u** linked BAX pore-mediated mitochondrial DNA release to amplification of tubular damage (14,15). In loxoscelism, Okamoto demonstrated that venom sphingomyelinases D may induce complement-mediated renal cell death, reinforcing the biological plausibility of systemic nephrotoxicity (16). In snakebite envenomation**,** Pushpalatha described in 2024 a substantial clinical burden of acute kidney injury associated with snakebite, Meena summarized a histopathological spectrum ranging from acute tubular injury to cortical necrosis, and both **Rao** and Alvitigalaemphasized in 2025 that snakebite-associated acute kidney injury remains a major cause of morbidity and mortality and may leave chronic renal sequelae when recognition and specialized support are delayed; more broadly, **Su** reminded us that toxin-induced acute kidney injury requires early surveillance, serial follow-up, and treatment directed at the predominant mechanism (17–20).

This distinction is clinically relevant. Nature may injure a previously functional kidney and produce dialysis-requiring acute kidney injury, or it may destabilize a chronically damaged kidney and precipitate severe uremic and metabolic decompensation. The present series brings together four cases managed at a regional referral hospital in Paraguay, showing how venomous animal injuries may affect renal function across a broad spectrum, from severe acute kidney injury to marked decompensation of advanced chronic kidney disease.

CASE PRESENTATIONS

Case 1

A 44-year-old man presented with a history of probable Loxosceles spider bite approximately 15 days before admission. He developed progressive cellulitis of the right lower limb, sought delayed care at another hospital, left against medical advice, and subsequently presented to our institution, where he was admitted to the adult intensive care unit. At evaluation he was conscious, afebrile, eupneic, and initially hemodynamically stable, although vasopressor support was required. Urine output remained preserved at 1,800 mL over 12 hours, with no edema. He received piperacillin-tazobactam and vancomycin adjusted to estimated renal function.The clinical course was marked by severe soft tissue infection requiring surgical debridement and fasciotomy. Admission diagnoses included acute kidney injury, Loxosceles bite, septic shock of skin and soft tissue origin, necrotizing fasciitis of the right lower limb, postoperative status, posterior lower limb cellulitis, and type 2 diabetes mellitus. Initial laboratory evaluation showed severe azotemia with urea 173 mg/dL and creatinine 9.1 mg/dL. Hemodialysis was initiated approximately 48 hours after admission after negative viral screening and was continued daily for a total of 10 sessions. During follow-up, creatinine decreased to 1.5 mg/dL and urea to 69 mg/dL, although later fluctuations occurred. Despite preserved diuresis and partial biochemical improvement, the patient’s overall condition deteriorated because of the severity of the infectious process and systemic compromise; he was transferred to a higher-complexity center and subsequently died.

Case 2

A 33-year-old man was admitted after multiple wasp stings affecting the face, anterior chest, and extremities. According to relatives, the accident occurred approximately 18 days before admission. He sought care four days before hospital presentation because of malaise, nausea, vomiting, and dyspnea, but his condition continued to worsen. On arrival at the emergency department, he had Kussmaul breathing, dry mucous membranes, severe distress, and anuria. Blood pressure was 183/110 mmHg, heart rate 132 beats/min, oxygen saturation 95% on high-flow supplemental oxygen, and temperature 36°C.Laboratory studies showed profound renal and metabolic derangement, including creatinine 34.5 mg/dL, potassium 6.4 mmol/L, severe leukocytosis, and subsequent potassium elevation up to 7.8 mmol/L. Total creatine kinase reached 790 U/L, supporting major systemic injury and possible muscle breakdown. He was transferred to intensive care, where he required sedation, endotracheal intubation, invasive mechanical ventilation, central venous access, and urgent hemodialysis. The main diagnoses were acute kidney injury, severe acute respiratory failure, severe metabolic acidosis, severe hyperkalemia, and severe dehydration. Hemodialysis was initiated urgently and repeated for a total of 9 sessions. Despite aggressive organ support, he experienced progressive deterioration and died after the ninth dialysis session.

Case 3

An 81-year-old man was admitted three days after Bothrops snakebite. At presentation he had acidotic breathing, oxygen requirement by non-rebreather mask, and anuria initially raising concern for obstructive retention because of bladder distension. He reported preserved previous urination and had a history of prostate surgery. Vital signs were blood pressure 184/100 mmHg, heart rate 97 beats/min, respiratory rate 31/min, and oxygen saturation 100%. Initial diagnoses were acute kidney injury, bothropic envenomation, acute urinary retention of undetermined etiology, and arterial hypertension.Initial laboratory values showed hemoglobin 11.3 g/dL, platelets 42,000/mm³, urea 181 mg/dL, and creatinine 7.9 mg/dL. During the following days renal dysfunction worsened, with creatinine peaking at 10.7 mg/dL and urea at 279 mg/dL. Severe anemia and thrombocytopenia were also documented in the early phase. The patient underwent 12 hemodialysis sessions. Over time, clinical and biochemical recovery became evident, with creatinine decreasing to 2.4–2.5 mg/dL and urea to 41 mg/dL. He was discharged with marked improvement, although he did not return for ambulatory nephrology follow-up.

Case 4

Patient information and clinical findings

A 48-year-old man from Belén was admitted with probable spider bite and right lower limb cellulitis. At the time of nephrology evaluation, he carried diagnoses of probable stage V chronic kidney disease of hypertensive etiology, arterial hypertension, moderate anemia, and soft tissue infection associated with a probable arachnid bite. He was conscious, afebrile, hemodynamically stable, eupneic, and initially maintained spontaneous diuresis, with no peripheral edema.

Diagnostic assessment

Initial laboratory results already showed severe renal dysfunction, with urea 237 mg/dL and creatinine 10.4 mg/dL on July 2, rising to urea 292 mg/dL and creatinine 10.1 mg/dL on July 5. Gasometry showed severe metabolic acidosis with pH 7.13 and bicarbonate 7.4 mEq/L. Viral serologies and VDRL were negative. Renal and vesicoprostatic ultrasonography demonstrated bilateral chronic nephropathy and enlarged prostate. One laboratory value of calcium 1.19 corresponded to ionized calcium, not total calcium.

Therapeutic intervention

Because of severe uremic and metabolic decompensation, renal replacement therapy was indicated. Hemodialysis was initiated on July 5 and repeated sequentially during hospitalization. The patient also received antimicrobial therapy for skin and soft tissue infection, initially cefotaxime plus clindamycin and later piperacillin-tazobactam plus vancomycin.

Follow-up and outcomes

Diuresis remained preserved during much of the course, reaching 1,960 mL/24 h, 2,970 mL/24 h, and 2,890 mL/24 h in successive evaluations. Biochemical parameters improved after the first sessions, with creatinine declining to 6.8 mg/dL, then 4.9 mg/dL, and later 4.0 mg/dL, while urea fell to 32 mg/dL. However, this improvement was not sustained. Subsequent values showed renewed deterioration, with creatinine 9.6 mg/dL on July 22 and 10.2 mg/dL on August 2, accompanied by urea 172 mg/dL and potassium 6.36 mmol/L. The patient required at least 12 hemodialysis sessions, and documentation was sent to secure a fixed slot at the national nephrology institute, supporting transition toward chronic dialysis support.Table 1 summarizes the temporal sequence, baseline kidney status, major renal manifestations, key diagnostic findings, therapeutic interventions, and clinical outcomes across the four cases, providing an integrated overview of the spectrum of kidney involvement associated with venomous animal injuries.These four cases illustrate that venomous animal injuries may affect renal function across a continuum, ranging

from dialysis-requiring acute kidney injury in apparently preserved kidneys to severe metabolic decompensation in kidneys already compromised by chronic disease. Beyond the descriptive value of the series, two practical insights emerge: delayed presentation may amplify systemic and renal severity, and preserved diuresis does not exclude advanced renal dysfunction.The first three cases belong predominantly to the acute kidney injury end of this spectrum, but their mechanisms were not identical. In Case 1, probable loxoscelism was followed by necrotizing soft tissue infection and septic shock, suggesting a mixed pathway of direct toxic injury, systemic inflammation, hypoperfusion, and sepsis. In Case 2, multiple wasp stings were associated with anuria, severe acidosis, hyperkalemia, and elevated creatine kinase, a profile more consistent with fulminant toxic-inflammatory injury compounded by possible rhabdomyolysis and pigment nephropathy. In Case 3, Bothrops envenomation was accompanied by thrombocytopenia, anemia, and progressive azotemia, supporting the contribution of hemotoxicity, microvascular dysfunction, coagulopathy, and ischemic tubular damage. These patterns are broadly consistent with prior clinical and mechanistic reports on venom-related renal injury (3–7,9–20).

The timing of presentation deserves specific attention. Cases 1 and 2 reached hospital care only after substantial delay, approximately 15 and 18 days after exposure, respectively. Although causality cannot be quantified in a four-case series, this delay likely contributed to the advanced metabolic derangement and poor outcomes observed in both patients. The contrast with Case 3, who presented after three days and survived with biochemical recovery, reinforces the probable clinical importance of earlier recognition and referral.The fourth case expands the conceptual framework but also requires interpretive caution. This patient already had advanced probable hypertensive chronic kidney disease, bilateral chronic nephropathy on imaging, anemia, and a later course compatible with progression toward maintenance hemodialysis. Therefore, the envenomation-related event should not be framed as a proven isolated cause of renal failure. Rather, the probable spider bite and associated skin and soft tissue infection appear more plausibly to have acted as a precipitating stressor that unmasked or accelerated terminal renal decompensation in a kidney with markedly reduced reserve.Another relevant lesson is that urine output alone may underestimate severity. In both Cases 1 and 4, patients maintained preserved diuresis despite substantial azotemia and need for renal replacement therapy. From a practical standpoint, this supports routine biochemical surveillance, acid-base assessment, and serial nephrologic evaluation in envenomation cases, especially when there is systemic toxicity, soft tissue infection, oliguria or anuria, hyperkalemia, or unexplained clinical deterioration.

This report has limitations. It is a small single-center case series, baseline renal function was unavailable in several patients, urine findings were incompletely documented, and long-term follow-up was limited, particularly for Case 3. These constraints reduce generalizability and preclude firm causal inference. Nevertheless, the series remains clinically useful because it highlights real-world renal presentations, underscores the importance of timing and renal reserve, and shows that venom-related injury may present either as severe acute nephrologic emergency or as decompensation of pre-existing chronic kidney disease.

DISCUSSION:-

These four cases illustrate that the relationship between nature and the kidney is neither simple nor uniform. Venomous animal injuries may affect renal function across a continuum. At one end, they may provoke true dialysis-requiring acute kidney injury in kidneys that were apparently functional before the insult. At the other, they may act as a destabilizing trigger in kidneys already structurally and functionally compromised, precipitating severe metabolic and uremic collapse.The first three cases belong predominantly to the first scenario. In the patient with probable Loxosceles bite, renal injury occurred in the context of necrotizing soft tissue infection, septic shock, and extensive inflammatory aggression. In the patient with multiple wasp stings, the picture was fulminant: anuria, hyperkalemia, profound acidosis, respiratory failure, and extremely elevated creatinine indicated overwhelming acute systemic toxicity with catastrophic renal consequences. In the patient with Bothrops envenomation, hemotoxicity, microvascular dysfunction, coagulopathy, and ischemic tubular damage probably converged to produce severe but partially reversible acute kidney injury (3–7,9–11).

The fourth case expands the conceptual framework. Here, the renal insult cannot be explained as a purely acute nephrotoxic event. The patient already had advanced probable hypertensive chronic kidney disease, bilateral chronic nephropathy on imaging, anemia, and a later course compatible with progression toward maintenance hemodialysis. Nevertheless, the probable spider bite and associated skin and soft tissue infection coincided with abrupt and severe metabolic decompensation. Thus, the natural insult appears not as the sole origin of renal failure, but as the precipitating stressor that unmasked or accelerated terminal renal decompensation.This distinction has practical implications. Clinicians should not limit their attention to whether the kidney was previously normal or abnormal. What matters is recognizing that venomous injury may overwhelm renal reserve at any point along the spectrum. In a previously functional kidney, that means early suspicion of acute kidney injury when oliguria, anuria, acidosis, hyperkalemia,

rising creatinine, or systemic toxicity appear. In a chronically diseased kidney, it means appreciating that even a seemingly localized toxic or infectious event may precipitate dialysis-requiring decompensation.

Another relevant lesson is that preserved urine output does not exclude severe renal compromise. In both the first and fourth cases, patients maintained diuresis while still manifesting substantial azotemia and need for renal replacement therapy. This underscores the value of biochemical surveillance, acid-base assessment, and serial nephrologic evaluation beyond simple urine volume.This report has limitations. It is a small single-center case series, follow-up was incomplete in some patients, and detailed pre-envenomation renal baselines were not always available. In the fourth case, causality between the probable spider bite and dialysis dependence cannot be established with certainty because pre-existing chronic kidney disease was already advanced. However, this limitation is also part of the clinical reality the manuscript seeks to illuminate: nature does not always attack a pristine organ; sometimes it strikes a vulnerable one and accelerates its collapse.

CONCLUSION:-

Venomous animal injuries can severely affect renal function in more than one way. They may produce true dialysis-requiring acute kidney injury in patients with apparently preserved baseline kidney function, as observed after Loxosceles bite, multiple wasp stings, and Bothrops snakebite, or they may precipitate profound metabolic and uremic decompensation in kidneys already affected by chronic disease, as illustrated by the fourth case. In all scenarios, the venom-related insult becomes nephrologically decisive when it overwhelms renal reserve. Early recognition, repeated laboratory monitoring, prompt nephrology involvement, and timely hemodialysis when indicated are essential to improve outcomes.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This case series had prior approval from the corresponding institutional ethics committee before manuscript preparation and submission. All procedures were conducted in accordance with institutional ethical standards and with the principles of the Declaration of Helsinki.

CONSENT FOR PUBLICATION

Clinical data were anonymized before analysis and reporting. No directly identifiable personal information is included in this manuscript. Publication was prepared under prior ethical approval and with preservation of confidentiality.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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Tables

Table 1. Consolidated timeline of the four clinical cases

Case	Exposure	Baseline kidney status	Time to hospital evaluation	Main renal presentation	Key diagnostic findings	Renal support	Outcome
Case 1	Probable *Loxosceles* spider bite	Unknown baseline renal function; no pre-envenomation creatinine available	~15 days after bite	Severe AKI with preserved diuresis	Urea 173 mg/dL, creatinine 9.1 mg/dL; necrotizing soft tissue infection; septic shock	Hemodialysis started ~48 h after admission; 10 sessions	Transferred to higher-complexity center; died
Case 2	Multiple wasp stings	Unknown baseline renal function	~18 days after stings; worsened before admission	Anuric severe AKI with metabolic derangement	Creatinine 34.5 mg/dL, K 6.4–7.8 mmol/L, severe metabolic acidosis, respiratory failure, CK 790 U/L	Urgent hemodialysis; 9 sessions	Died during hospitalization after 9 sessions
Case 3	*Bothrops* snakebite	No baseline creatinine available; no	3 days after bite	Severe AKI with progressive azotemia	Urea up to 279 mg/dL, creatinine up to 10.7 mg/dL;	Hemodialysis; 12 sessions	Clinical and biochemical improvement;

Case	Exposure	Baseline kidney status	Time to hospital evaluation	Main renal presentation	Key diagnostic findings	Renal support	Outcome
		known history of chronic dialysis			thrombocytopenia; anemia		discharged; long-term follow-up unavailable
Case 4	Probable spider bite with soft tissue infection	Advanced probable hypertensive CKD with bilateral chronic nephropathy on ultrasound	Early nephrology evaluation during admission	Severe decompensation of advanced CKD with metabolic acidosis	Urea 237–292 mg/dL initially, creatinine 10.4–10.1 mg/dL, pH 7.13, bicarbonate 7.4 mEq/L	Repeated hemodialysis; at least 12 sessions	Progression toward chronic dialysis support

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