Case Report

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Venomous Aftermath: A Case of Chronic Kidney Disease Following a Snake Bite

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Abstract

Background: Snakebite remains neglected public health concern in many tropical and subtropical countries. National epidemiological data on snakebite incidence in Indonesia remain limited, and restricted healthcare access often turns snakebite into a medical emergency due to the potential to cause local tissue damage, hemorrhage, respiratory failure, and particularly renal injury.

Objective: To discuss the management of Acute Kidney Injury (AKI) resulting from snakebite that progressed to Chronic Kidney Disease (CKD) in the rural region of Indonesia.

Case: A 45-year-old female patient from Maumere, East Nusa Tenggara, Indonesia, was bitten by an unidentified snake. The patient experienced pain, edema, and visible bite marks on the left leg, but no reported or identified comorbidities. She was hospitalized for 26 days and was diagnosed with snakebite-induced acute kidney injury (AKI). Antivenom, symptomatic treatment, and hemodialysis were administered due to her conditions. The patient was subsequently followed for 3 months and was diagnosed with chronic kidney disease (CKD) secondary to progression of AKI.

Conclusion: In Indonesia, particularly in rural regions such as East Nusa Tenggara, where snakebite cases are common, the establishment of long-term outpatient programs may be essential. Such programs could help manage late complications, including CKD, and enhance outcomes by avoiding hemodialysis and kidney transplantation.

Keywords: Chronic kidney diseases, renal failure, snakebite

Introduction

Snakebite envenoming is recognized by the World Health Organization (WHO) as the highest priority neglected tropical disease since 2017. WHO set the goal of reducing the incidence of death and disability from snakebite by 50% by 2030.[1] Globally, the incidence causes between 81,000 to 138,000 deaths annually, with four times the number of physical disabilities.[2] This burden is concentrated in impoverished rural areas of Asia, Africa, and Latin America, particularly where access to healthcare is limited.[3] National epidemiological data on snakebite incidents are limited and derive mainly from regional reports. Between 2004 and 2009, Cipto Mangunkusumo National Referral Hospital reported handling 42 cases.[4] Antivenom is the only definitive treatment in snakebite envenomation, but the availability of specific, effective types remains limited in many areas.[5]

Systemic manifestations of snake envenomation include venom-induced consumptive coagulopathy, neuromuscular paralysis, acute renal injury, myotoxicity, and cardiovascular complications.[6] AKI is recognized as a significant contributor to morbidity and mortality.[7] This injury following snake bite envenoming is multifactorial,

including direct nephrotoxicity of venom, coagulopathy, hypotension, disseminated intravascular coagulation, intravascular hemolysis, thrombotic microangiopathy, "capillary leak syndrome," rhabdomyolysis, complement activation, and secondary sepsis.[8] The incidence of AKI varies from 5% to 29% depending on the species of snake and the severity of envenomation.[9] According to Ariga, 2021 in India, 26%–28% of 193 patients with bite and AKI went on to develop Chronic Kidney Disease (CKD), depending on the duration of follow-up. Among patients who required dialysis during hospitalization, 36.8% developed CKD at follow-up.[10] This case specifically outlines the entire clinical progression of snakebite-induced AKI advancing to dialysis-dependent CKD, including crucial post-discharge follow-up. Furthermore, it highlights the practical challenges of managing severe envenomation and implementing long-term patient care in a resource-limited rural setting.

Case(s)

A 45-year-old female from Maumere, East Nusa Tenggara, presented to the hospital four hours after being bitten by an unidentified, blackish-brown snake with white patterns on the left leg. The patient, a teacher with no prior medical history or comorbidities, reported immediate pain, swelling (oedema), and visible bite marks on the left leg. Other symptoms included bleeding gums, bloody urine (hematuria), epigastric pain, and nausea.

On physical examination, the patient appeared fully conscious with a Glasgow Coma Scale score of 15. Vital signs were largely stable with a blood pressure of 110/70 mmHg, heart rate of 93 beats per minute, respiratory rate of 20 breaths per minute, and a temperature of 37.8°C. Initial laboratory investigations showed significant abnormalities, including marked leukocytosis, mild anemia, and thrombocytopenia. Additionally, urinalysis confirmed gross hematuria, and the initial results are summarized in Table 1.

Initial treatment included adequate fluid administration, pain management, and immobilization of the injured limb. It also entailed consulting with an expert clinical toxicologist specializing in snakebites in Indonesia. The patient then received two vials of polyvalent hematotoxin antivenom (*polihemato*), and the surgeon on duty was assigned to manage the care.

The condition was not improved, and hemotoxins caused progressive thrombocytopenia, severe anemia requiring numerous blood transfusions, and gastrointestinal bleeding (melena). In particular, the patient suffered Stage III AKI with anuria (0.4 cc/kg/hour). A chest X-ray and abdomen ultrasound confirmed

Table 1 Initial Laboratory and Urinalysis Findings on Admission (6 March, 2025)

Parameter	Result	Reference Range
Complete Blood Count		
Leukocytes	21.31 x 10^3/µL	4.50 - 11.00 x 10^3/µL
Hemoglobin	11.0 g/dL	12.0 - 16.0 g/dL
Hematocrit	31.0%	36.0 - 46.0%
Platelets	129 x 10^3/µL	150 - 450 x 10^3/µL
Blood Chemistry		
Cr	0.99 mg/dL	0.5 - 1.1 mg/dL
Coagulation		
Clotting Time (CT)	9 minutes	5–15 minutes
Bleeding Time (BT)	3 minutes	<8 minutes
Urinalysis		
Color	Red	Yellow
Blood	+4	Negative
Erythrocytes	Full	0-2/HPF

CT: Clotting Time, BT: Bleeding Time, Cr: Creatinine

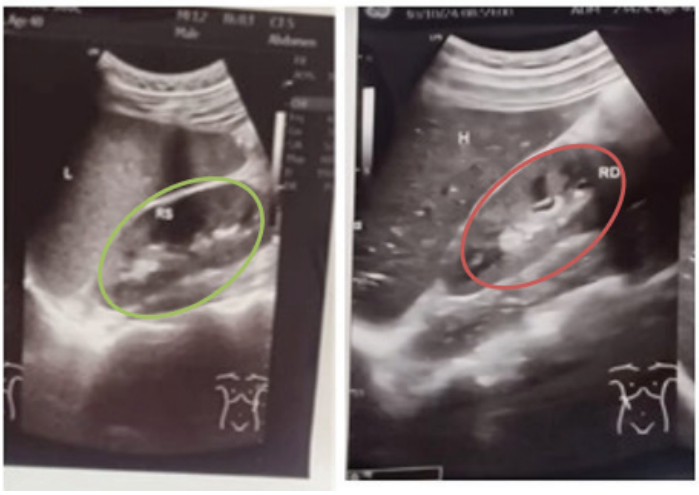

Fig. 1 Abdominal Ultrasound on 10 March 2025. Both Kidneys Show Normal Size and Echotexture, And Normal Morphology Does Not Rule Out Functional Acute Kidney Injury
*Red Circle (RD: Ren Dextra) Green Circle (RS: Ren Sinistra)

generalized oedema, ascites, and acute pulmonary oedema caused by significant fluid overload, as detailed in Figs. 1 and 2. Hemodialysis was recommended on the second day due to the anuria, but the patient and family initially declined. As the condition deteriorated, consent was provided to conduct hemodialysis on the fourth day. Additionally, four more vials of antivenom were administered on the fifth day, making a total of six. Fig. 3 details the patient's clinical progression, lab results, and major treatments in the 26-day hospitalization.

There were increased, hazy vascular markings in both pulmonary hila extending to the lateral basal regions, suggestive of pulmonary oedema, with bilateral pleural effusions that were more pronounced on the left, reaching the level of the fifth anterior rib. The right was confined to the costophrenic sinus, and cardiac size could not be assessed. The tip of the double-lumen catheter was

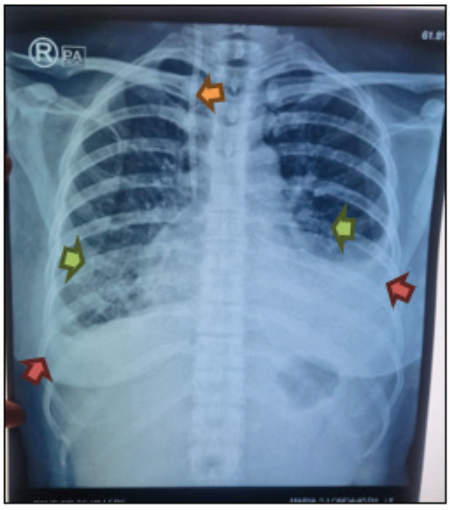

Fig. 2 Chest X-ray on 10 March 2025
*Red arrow (bilateral pleural effusion), Green arrow (increased pulmonary vascularity), Orange arrow (double-lumen hemodialysis catheter)

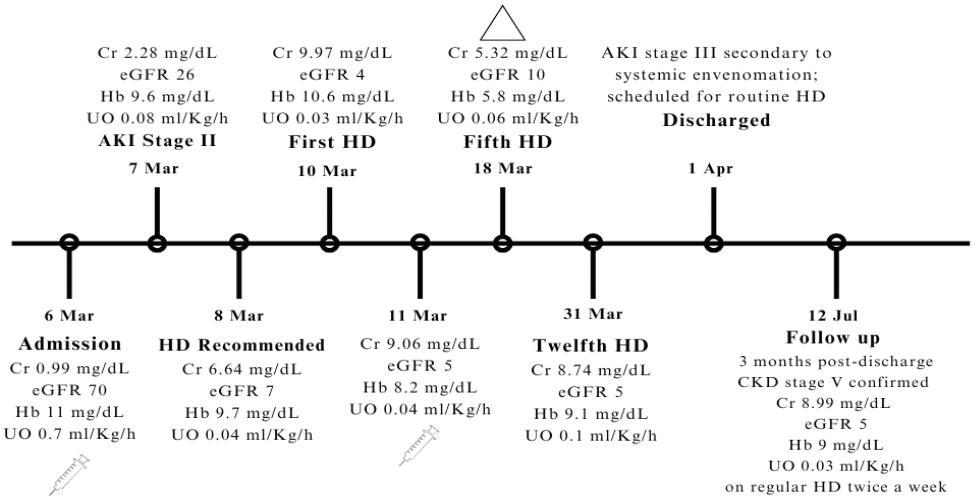

Fig. 3 Patient's Clinical Progression, Lab Results, And Major Treatments In The 26-Day Hospitalization

projected over the right paravertebral region at the level of the sixth thoracic vertebra (T6), and the visualized osseous structures were intact.

The patient was discharged after 26 days of hospitalization in a stable, dialysis-dependent condition and scheduled for regular outpatient hemodialysis twice weekly. At a 3-month follow-up, the patient remained dialysis-dependent, with persistently elevated serum creatinine (8.99 mg/dL), ureum (118 mg/dL), and estimated glomerular filtration rate (5 mL/sec/1,73 m^2). An abdominal ultrasound performed at this time showed bilaterally shrunken kidneys and increased cortical echogenicity, evidence consistent with chronic diffuse parenchymal disease as detailed in Fig. 4. Based on the clinical, laboratory, and imaging results, a final diagnosis of CKD Stage 5 secondary to snakebite envenomation was established. Written informed consent was obtained from the patient in July 2025 during a follow-up visit at T.C. Hillers Hospital, Maumere, for the publication of this case report and any accompanying images.

Both kidneys are reduced in size (right:

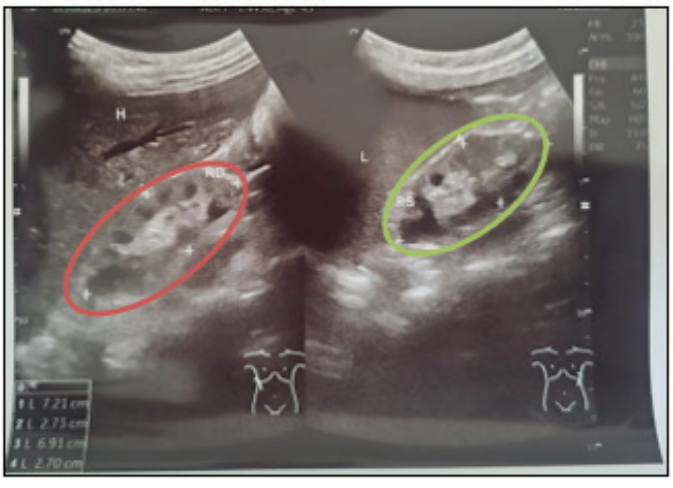

Fig. 4 Abdominal ultrasound on 12 July 2025
*Red Circle (RD: Ren Dextra) Green Circle (RS: Ren Sinistra)

7.2 × 2.7 cm; left: 6.9 × 2.7 cm) and exhibit moderately increased cortical echogenicity, while the corticomedullary border remains distinct. These results are suggestive of early bilateral chronic diffuse parenchymal disease.

Discussion

This case report details the severe progression from snakebite-induced AKI to irreversible, dialysis-dependent Stage 5 CKD in a rural region of Indonesia. The clinical progression underscores the significant and chronic nephrotoxic consequences of envenomation and emphasizes the critical challenges in treatment in rural areas. The development of severe AKI in this patient is consistent with the known pathophysiological mechanisms of hemotoxic snakebites.[11] Direct effects of the venom can cause glomerulonephritis, and indirect injuries are attributed to the release of endogenous cytokines and inflammatory mediators that induce apoptosis of kidney cells.[11]

The symptoms that occurred include hematuria, gingival hemorrhage, thrombocytopenia, and anemia requiring transfusions. This strongly shows venom-induced consumptive coagulopathy and thrombotic microangiopathy, which are typical characteristics of envenomation from the Viperidae family, particularly Russell's vipers (Daboia spp.), recognized for their nephrotoxic properties.[12] This case report assumes that the snake is possibly *Daboia siamensis,* based from the patient's clinical manifestations and the species' endemicity in NTT, Indonesia nevertheless, it should be emphasized that this identification remains presumptive and unconfirmed. The systemic effects, combined with potential direct venom nephrotoxicity and hypotension-induced ischemic injury, create a multifactorial assault on the kidneys, leading to acute tubular necrosis or, in more severe cases, acute cortical necrosis, which often causes irreversible renal failure.[11] A crucial aspect of this case is the transition from AKI to CKD, a significant long-term complication. The results correspond to information collected from other regions with high snakebite incidence.

A retrospective case-control study conducted in South Korea showed the long-term renal implications of snake envenomation. The results present that among patients who developed AKI, 33.3% progressed to CKD in a one-year follow-up period.[13] In a prospective follow-up study by Rao *et al*., 19 patients developed thrombotic microangiopathy (TMA), characterized by the triad of microangiopathic hemolytic anemia, thrombocytopenia, and AKI. Approximately 95% of TMA patients necessitated intensive hemodialysis, requiring between 18 and 26 sessions during hospitalization. Although 68% of the cases achieved complete or partial renal recovery, and 10% progressed to CKD. [14] However this rural healthcare facility had limited resources, and therefore, advanced laboratory markers to definitively confirm TMA such as peripheral blood smear for schistocytes, lactate dehydrogenase, and haptoglobin) or rhabdomyolysis (creatine kinase) were not available. Suspicion of TMA in this case was based mainly on the classic clinical trial of acute kidney injury, progressive anaemia and thrombocytopenia.

Jayawardana *et al*. monitored four patients with AKI generated by Russell's viper bites and reported that 50% progressed to CKD.[15] In a prospective observational study of 100 snakebite patients, Kunti *et al*. reported that viper envenomation accounted for 46% of cases. The study underscored a high burden of renal complications, with 60% of patients developing AKI and 12% requiring hemodialysis. The long-term sequelae were significant, as 8% progressed to CKD, while the mortality rate remained at 6%.[16] The literature suggests that the causes of non-recovery were diverse, but several commonalities were identified, including Viperidae bites, extended dialysis duration, consistently elevated creatinine levels, and initial kidney biopsies presenting diffuse or patchy cortical necrosis or acute interstitial nephritis.[11]

The limitation of this case report is the failure to identify the snake species, inability to perform advanced laboratory investigations to definitively confirm the underlying mechanisms of AKI, preventing a conclusive association between a particular venom and the documented clinical progression. Several factors in this case may have contributed to the poor renal outcome, reflecting broader challenges in snakebite management in rural Indonesia. Although antivenom was administered, the definitive efficacy can be hampered when the species is unidentified, as a polyvalent type may have limited parasite-specific coverage. Furthermore, the patient's initial declination of hemodialysis for 2 days, from the second to the fourth day of admission, possibly worsened the uremic state and fluid overload, potentially elevating the ischemic

and toxic injury to the kidneys. This delay, alongside the underlying limits of healthcare facilities in remote regions, underscores the difference between optimal management protocols and the practical implementation in reality.

Future studies should focus on the improved collection of epidemiological data regarding snakebite incidence and outcomes in Indonesia. This will aid in the development of more integrated management protocols. Additionally, greater attention should be paid to the distribution of antivenom to remote areas. Public education and enhanced training in snakebite management for healthcare personnel also need to be prioritized. In conclusion, establishing long-term outpatient programs for snakebite patient in Indonesia, particularly in rural area such as NTT, is critical to managing late adverse events like CKD and preventing the need for hemodialysis or renal transplantation.

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