

**HEMOGLOBIN CAST NEPHROPATHY WITH DIALYSIS REQUIRING ACUTE KIDNEY INJURY FOLLOWING SNAKEBITE: A CASE REPORT****Geeta Sheth**

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**ABSTRACT** Snakebite-associated acute kidney injury (AKI) is an important cause of morbidity in tropical countries and may result from hemodynamic disturbance, venom-induced coagulopathy, intravascular hemolysis, rhabdomyolysis, direct nephrotoxicity and microvascular injury. Pigment nephropathy is a less frequently emphasized biopsy finding. We report a 62-year-old farmer presented approximately 20 hours after an unidentified snakebite with hypotension, anuria, coagulopathy, serum creatinine of 12 mg/dL and potassium of 6.8 mEq/L. Creatine kinase was 6,280 U/L, and myoglobinuria was reported. Antivenom was administered and hemodialysis initiated. Persistent anuria prompted planning of kidney biopsy on day 18. The biopsy report described severe acute tubular injury with hemoglobin-positive pigmented casts, supporting hemoglobin cast nephropathy despite concurrent muscle injury. Urine output improved after approximately four weeks of dialysis, and dialysis independence was achieved after eight weeks. Serum creatinine decreased to 1.8 mg/dL at ten weeks. This case highlights pigment-specific staining and the possibility of substantial renal recovery despite prolonged dialysis dependence.

**KEYWORDS :** Snakebite; Acute Kidney Injury; Pigment Nephropathy; Hemodialysis**INTRODUCTION**

Snakebite envenomation is a major neglected tropical health problem, particularly in South Asia. Acute kidney injury is one of its most serious systemic complications. The renal lesion is heterogeneous: acute tubular injury/necrosis is most frequently reported, while acute interstitial nephritis, cortical necrosis, thrombotic microangiopathy and pigment-associated tubular injury are also recognized. In an Indian retrospective series, acute tubular necrosis was the predominant lesion among patients undergoing biopsy, illustrating the importance of tubular injury in this setting (Vikrant et al., 2017).

Pigment nephropathy results from tubular exposure to heme pigments, principally hemoglobin released during intravascular hemolysis and myoglobin released during rhabdomyolysis. These pigments may cause direct tubular toxicity, intratubular cast formation and obstruction, and renal vasoconstriction. Immunohistochemical identification of hemoglobin or myoglobin in renal casts can help establish the mechanism (Prema & Kurien, 2023). We report dialysis-requiring AKI after snakebite in which kidney biopsy demonstrated acute tubular injury with hemoglobin-positive pigmented casts, followed by recovery of urine output and dialysis independence.

**Case Presentation**

A 62-year-old man, a farmer with no known comorbidities, presented approximately 20 hours after a snakebite to the left foot sustained while working in a field. The snake was not identified. He had progressive swelling of the left foot and reduced urine output. On arrival, he was drowsy and hemodynamically unstable, with blood pressure of 88/60 mm Hg and respiratory rate of 25 breaths/min. Pallor and tachycardia were present. The left foot and leg were diffusely swollen, without active bleeding.

In the emergency department, 10 vials of antivenom were administered along with neostigmine (1.5 mg) and atropine (0.6 mg).

Initial investigations showed anemia, coagulopathy, serum creatinine of 12 mg/dL and potassium of 6.8 mEq/L. Creatine kinase was 6,280 U/L and lactate dehydrogenase was 2,542 U/L. Urinalysis was positive for myoglobin and showed 2+ albumin, 20–30 red blood cells/high-power field, and 1–2 pus cells/high-power field indicating coexistence of haematuria and myoglobinuria. Abdominal ultrasonography demonstrated normal-sized kidneys with preserved corticomedullary differentiation. Left lower-limb ultrasonography was reported to show cellulitis involving the middle and lower thirds of the thigh and the entire leg.

Hemodialysis was initiated for anuria and hyperkalemia. Seven sessions were performed on days 1, 2, 3, 5, 7, 9 and 11. Renal biochemical parameters improved, but anuria persisted. Therefore, renal biopsy was planned on day 18.

The biopsy report described diffuse severe acute tubular injury with a

few coarse pigmented casts that stained positively for hemoglobin (Figure 1). Twice-weekly hemodialysis was subsequently continued. Urine output improved after approximately four weeks of dialysis, and the patient became dialysis independent after eight weeks. At the end of 10th week, serum creatinine was 1.8 mg/dL.

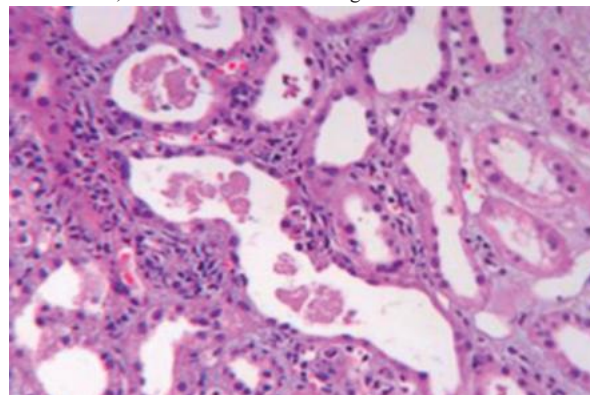


**Figure 1. Renal Biopsy Showing Tubular Injury and Intraluminal Casts (Hematoxylin and Eosin)**

**DISCUSSION**

The principal teaching point in this case is the demonstration of pigment-associated acute tubular injury after snakebite. A 2024 scoping review of 28 studies comprising 534 renal biopsies from snakebite-associated kidney injury found acute tubular necrosis to be the most common lesion, followed by acute interstitial nephritis, cortical necrosis and thrombotic microangiopathy (Meena et al., 2024). A large Indian clinicopathological series also documented frequent hemolysis and rhabdomyolysis among patients with snakebite-associated AKI (Vikrant et al., 2017). These observations support the concept that renal injury after envenomation is often multifactorial rather than attributable to a single mechanism.

Hemoglobin cast nephropathy is an important manifestation of intravascular hemolysis. In this case, anemia and increased lactate dehydrogenase are compatible with hemolysis. The reported hemoglobin staining identifies a hemoglobin component within the casts. Hemoglobin-mediated injury involves tubular toxicity and cast formation (Mahmud et al., 2020). Hypotension at presentation provides a further plausible ischemic contribution in this patient.

Myoglobin cast nephropathy is similarly associated with acute tubular injury, and snake envenomation is a recognized cause of rhabdomyolysis-associated myoglobin cast nephropathy (Prema & Kurien, 2023). The increased creatine kinase and reported myoglobinuria in our patient suggest concurrent muscle injury.

In their 56-case biopsy series, Prema and Kurien (2023) documented snakebite among the causes of hemoglobin cast nephropathy and also reported combined hemoglobin and myoglobin positivity in two snakebite-associated cases. Snake envenomation can cause both rhabdomyolysis and intravascular hemolysis, so both pigments may contribute to renal injury.

The diagnosis is therefore best expressed as hemoglobin cast nephropathy associated with severe acute tubular injury, with possible additional injury from rhabdomyolysis.

Snakebite-associated AKI can require dialysis while retaining the potential for recovery. Vikrant et al. (2017) reported renal replacement therapy in 81.8% of 121 patients; tubular necrosis was present in 20 of 22 biopsied patients. Our patient's recovery of urine output after approximately four weeks and later dialysis independence illustrate that prolonged anuria did not preclude substantial renal recovery in this instance.

## CONCLUSION

Snakebite-associated AKI may involve pigment-mediated tubular injury from hemolysis and/or rhabdomyolysis. This case demonstrates dialysis-requiring anuric AKI with biopsy-proven acute tubular injury and hemoglobin-positive pigmented casts, followed by recovery of urine output and dialysis independence. Pigment-specific staining helped characterize the tubular lesion despite evidence of concurrent muscle injury. In patients with persistent AKI after snakebite, renal biopsy may provide important diagnostic and prognostic information, particularly when the mechanism of injury remains uncertain. Persistent anuria after snakebite does not invariably imply irreversible renal failure when biopsy shows acute tubular injury.

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