Case Report

Bilateral Renal Cortical Necrosis following Snake Envenomation: A Comprehensive Case Report and Analysis of the Underlying Pathophysiological Mechanisms and Treatment Approaches

Vijay Kumar SS1, Shabbir Shekhli2, Anila Jose3

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Abstract

Acute bilateral renal cortical necrosis (RCN) is a rare but potentially lethal form of renal injury that poses a diagnostic and therapeutic challenge for clinicians. The causes of renal cortical necrosis are multi-factorial. Renal cortical necrosis shows a higher incidence in men than women in non-obstetric cases, and its clinical presentation is heterogeneous, ranging from asymptomatic to severe renal failure. In this case report, we describe the clinical course of a patient who presented with acute renal failure following a venomous snake bite and was subsequently diagnosed with renal cortical necrosis. Despite aggressive management, the patient ultimately succumbed to death on the 24th day of admission. The present case highlights the importance of early diagnosis, prompt initiation of appropriate management, and effective prevention of complications in patients with renal cortical necrosis to improve patient outcomes and reduce morbidity and mortality. A timely and accurate diagnosis of renal cortical necrosis is critical for initiating appropriate interventions, and management should aim to address the underlying cause and prevent potential complications.

Keywords: Snake Bite; Rhabdomyolysis; Acute Kidney Injury; Emergency Medicine.

Author's Affiliation: ¹Assistant Professor, Department of Emergency Medicine, KS Hegde Medical Academy Deralakatte, Mangalore 575018, India, ²Assistant Professor, Department of Emergency Medicine, S. Nijalingappa Medical College, Navanagar 587102, Bagalkot, India, ³Assistant Professor, Department of General Medicine, Sree Narayana Institute of Medical Sciences Chalakka, kunnukara 683594 India.

Corresponding Author: Shabbir Shekhli, Assistant Professor, Dept. of Emergency Medicine, S. Nijalingappa Medical College, Navanagar-587102, Bagalkot, India.

E-mail: vijay8792069357@gmail.com

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INTRODUCTION

Acute bilateral renal cortical necrosis (RCN) is an uncommon yet potentially lethal form of renal injury, characterized by extensive necrosis of the cortical elements, including blood vessels, glomeruli, and tubules. Despite its rarity, renal cortical necrosis represents a diagnostic and therapeutic challenge for clinicians, given its varied etiology, complex pathophysiology, and potential complications. With an incidence of only 2% among cases of acute renal failure, renal cortical necrosis is an elusive entity that requires a high degree of clinical suspicion for early diagnosis and appropriate management.¹

The causes of renal cortical necrosis are multifactorial and vary depending on the geographical region, patient population, and comorbid conditions. Obstetric complications remain the leading cause of renal cortical necrosis, accounting for up to 70% of cases, primarily due to prolonged or severe hypotension during pregnancy or childbirth, resulting in renal ischemia and subsequent necrosis. In contrast, non-obstetric causes contribute to 20-30% of cases and can stem from various etiologies, including infectious diseases, vascular disorders, drug toxicity, and snake bites.

More over, renal cortical necrosis exhibits a higher incidence in men than women in non-obstetric cases, although the underlying mechanisms are unclear. This gender based discrepancy may be attributed to the differential susceptibility of the renal vasculature to various insults, such as endothelial damage, thrombosis, or inflammation, leading to cortical necrosis.⁴ Additionally, the clinical presentation of renal cortical necrosis is heterogeneous, ranging from asymptomatic to severe renal failure, depending on the extent and severity of the cortical damage, as well as the underlying cause.

Thus, a timely and accurate diagnosis of renal cortical necrosis is critical for initiating appropriate interventions, such as supportive care, dialysis, and, in some cases, renal replacement therapy. Furthermore, the management of renal cortical necrosis should aim to address the underlying cause and prevent potential complications, such as hypertension, electrolyte imbalances, and infection. Therefore, a comprehensive understanding of the pathophysiology, diagnosis, and management of renal cortical necrosis is essential for improving patient outcomes and reducing morbidity and mortality.

CASE REPORT

A 51 year old female presented with complaint of alleged H/O snake bite over right upper limb on an evening of July while working in field, brought to our Emergency Room (ER) after 2 hours of bite.

On Primary survey she was found to be anxious but oriented. Her airway was patent, spontaneous breathing with a rate of 12 per minute and maintains a saturation of 96% on room air. Her circulation parameters were Bp - 180/100 mm of Hg, pulse -82 bpm, temperature of 100 degree Fahrenheit. On Disability examination GCS - E4V5M6 and pupils bilateral normal and reactive, she was moving

all her limbs. Systemic examination revealed no abnormalities.

Local Examination revealed redness swelling over right hand and local rise of temperature present. Fang marks present over right hand index finger over dorsal aspect (Fig. 1).

Past medical history revealed that she was a known case of diabetic and hypertension on irregular treatment since 1 year.



Fig. 1: Swelling of right upper limb after Envenomation

Investigations

On arrival to ER whole blood clotting time (WBCT) test was more than 20 minutes.

Her capillary glucose level was 202mg/dl, and ECG was normal sinus rhythm. Total Count were 9210 cells per cu.mm with Neutrophilic predominance (86%), PT-INR was more than 3 minutes.

Other investigations like Serum Electrolytes (Sodium, Potassium and chloride), Urine Analysis, Renal function tests and Liver Function tests were within normal limits. Chest X-ray revealed no abnormalities. Ultrasound of the abdomen revealed fatty hepatomegaly, splenomegaly and minimal right side pleural effusion.

Management

The present study reports on the clinical course of a patient admitted to the intensive care unit (ICU) on an emergency basis following a venomous snake bite. Initial management involved the administration of 10 vials of antisnake venom (ASV) stat, followed by 6 vials every 8 hours for

the first three days. In addition, 6 units of fresh frozen plasma were transfused, and antibiotic therapy consisting of piperacillin with tazobactam and metronidazole was initiated parenterally every 8 hours. Other supportive measures were implemented as needed. Due to the development of oliguria, hemodialysis was initiated and continued regularly on a daily basis, with antibiotic dosages adjusted based on creatinine clearance.

By day 3 of admission, the patient's renal parameters had become impaired, with blood urea and serum creatinine levels of 104 and 4.0, respectively, and urine output decreased to 160 ml while the input was 2900 ml. On day 5, the patient developed acute pulmonary edema, as evidenced by chest x-ray, and emergency hemodialysis was continued with Non-invasive ventilation (NIV). (Fig. 2) Antibiotic therapy was stepped up accordingly. General condition improved with NIV and hemodialysis, and the patient was transferred from the ICU to the emergency ward.

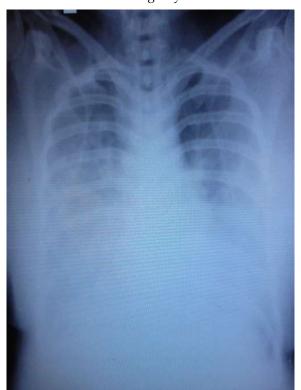


Fig. 2: Chest Xray showing pulmonary edema

However, after 15 days of admission, the patient's condition began to deteriorate, and in view of anemia with hemoglobin of 7.4 g/dL, 3 units of packed red blood cells were transfused. Ultrasound guided renal biopsy was performed, revealing renal cortical necrosis (fig. 3). Despite aggressive management, the patient ultimately succumbed to death on the 24th day of admission.

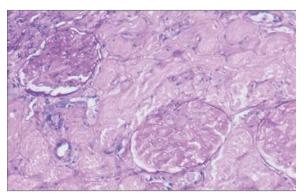


Fig. 3: Renal Biopsy suggesting renal cortical necrosis

DISCUSSION

Snake bite is a medical emergency that can have dire consequences if not treated promptly. It is a preventable public health issue that is particularly prevalent in rural areas of tropical and subtropical countries. Despite a significant body of literature on snake bite morbidity and mortality, it is crucial to recognize that snake bite and envenoming result in more than 20,000 deaths annually worldwide.⁴ This staggering figure is a somber reminder of the importance of timely intervention and appropriate management.

It is well-established that the longer the venom remains in the bloodstream before administration of anti-snake venom, the greater the likelihood of complications such as acute renal failure, tubular necrosis, cortical necrosis, and glomerulonephritis. Thus, the administration of anti-snake venom is the only specific treatment for snake bite envenoming that has been shown to be effective in treating this condition.⁵ In light of this, it is imperative to emphasize the importance of early recognition of symptoms and prompt treatment with anti-snake venom, which can significantly improve the prognosis of patients with snake bite envenoming.

Bilateral cortical necrosis of the kidneys is a rare entity that was first described by Juhel-Renoy in the year 1886.⁶ It accounts for only 2% of all cases of acute renal failure and is typically associated with severe renal failure at presentation. Oligoanuria of five to 12 days duration was observed in all patients, and acute kidney injury (AKI) is typical in patients with renal cortical necrosis, with associated complications such as hyperkalemia and fluid overload.⁷

Ramchandra et al. conducted a study that showed that acute cortical necrosis due to snake bite (sea snake, cobra, green pit viper, Russell viper) was observed in 14.2% of cases.⁸ Sonography typically

shows enlarged kidneys with reduced blood flow, and as the disease progresses, a hypoechoic circumferential band on sonography is seen in the subcapsular area. Contrast enhanced computed tomography (CT) scanning is the most sensitive imaging modality for diagnosis.

Renal biopsy remains the mainstay of diagnosis for cortical necrosis. It is indicated if the diagnosis is unclear. The tubular cells show necrosis with infiltration of leukocytes. The glomeruli may be necrotic, and thrombosis of the arterioles can be seen. Diffuse cortical necrosis is the dominant lesion in 71.9% of patients, while the remaining 28% of patients have patchy cortical necrosis.¹ These findings underscore the importance of prompt recognition and management of snake bites, as delayed or inadequate treatment can lead to severe and potentially life-threatening complications.

CONCLUSION

In conclusion, renal cortical necrosis (RCN) is a significant contributor to chronic kidney disease, and its etiology is primarily related to obstetric complications. The timely diagnosis of RCN is crucial, and an early renal biopsy, particularly in cases of anuric renal failure, can aid in establishing a definitive diagnosis. It is noteworthy that acute tubular necrosis is the most common cause of acute kidney injury in snake bites, and pigment nephropathy is a common outcome. In cases of snake bite, it is important to consider the possibility of RCN if renal dysfunction persists despite more than two weeks of hemodialysis. Given the rarity of the condition, the use of imaging techniques such as contrast enhanced computed tomography (CT) and ultrasound guided renal biopsy remains crucial for the definitive diagnosis of RCN. Therefore, further research is necessary to better understand the pathogenesis and management of this debilitating condition.

REFERENCES

- Prakash J, Tripathi K, Pandey L, Sahai S, Usha, Srivastava P. Spectrum of renal cortical necrosis in acute renal failure in eastern India. Postgraduate Medical Journal. 1995;71(834):208-210.
- Sahay M, Swarnalata, Swain M, Padua M. Renal cortical necrosis in tropics. Saudi Journal of Kidney Diseases and Transplantation. 2013;24(4):725.
- 3. Indraprasit S, Boonpucknavig V. Acute interstitial nephritis after a Russell's viper snake bite. Clin. Nephrol. 1986; 15: 111.
- 4. Kasturiratne A, Wickremasinghe AR, De Silva N, Gunawardena NK, Pathmeswaran A, Premaratna R, et al. The global burden of snakebite: A literature analysis and modelling based on regional estimates of envenoming and deaths. PLOS Med. 2008 Nov 4;5(11):e218.
- 5. Ahmed S, Nadeem A, Islam M, Agarwal S, Singh L. Retrospective analysis of snake victims in Northern India admitted in a tertiary level institute. J Anaesthesiol Clin Pharmacol. 2012;28(1):45.
- Juhel-Renoy, E. (1886) De l'annurie precoce scarletinuese. Archives generales de Medicine, 1, 385.
- 7. Shastry JCM, Date A, Carman RH, Johny KV. Renal failure following snake bite. Am. J. Trop. Med. Hyg. 1977; 26: 1032–8.
- 8. Ramachandran, S., & Perera, M. V. F. (1974). Survival in renal cortical necrosis due to snake bite. Postgraduate Medical Journal, 50(583), 314–316.