

Severe Acute Kidney Injury Secondary to Lawsonia Inermis (Henna) Intoxication Associated with Acute Pancreatitis: A Case Report

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Abstract

Case Report

Severe acute kidney injury (AKI) is a medical emergency requiring prompt management. Toxic causes, particularly those related to the consumption of medicinal plants, are often underestimated [1,2]. We report the case of an 18-year-old patient with no significant medical history, admitted for severe AKI following oral ingestion of *Lawsonia inermis*. On admission, the patient was anuric. Laboratory investigations revealed severe AKI with profound metabolic acidosis, hypokalemia, moderate proteinuria, and elevated serum lipase. Abdominal computed tomography showed stage I acute pancreatitis [3]. Kidney biopsy confirmed severe acute tubulointerstitial nephritis of probable toxic origin, without immune deposits [4–6]. Hemodialysis was required. The outcome was favorable, with gradual recovery of diuresis and complete restoration of renal function. This case highlights the nephrotoxic and systemic potential of certain medicinal plants, even in young individuals without comorbidities.

Keywords: severe acute kidney injury, *Lawsonia inermis*, acute tubulointerstitial nephritis, acute pancreatitis, medicinal plants.

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INTRODUCTION

Severe acute kidney injury (AKI) is characterized by a rapid and significant decline in renal function, potentially requiring urgent renal replacement therapy [7,8]. Common causes include glomerular, vascular, and tubulointerstitial disorders, the latter accounting for 15–27% of biopsied cases [5,6].

In many regions, the use of medicinal plants is widespread. *Lawsonia inermis*, used for cosmetic or therapeutic purposes, contains compounds such as lawsone that may cause renal and systemic damage [2]. We report a rare case of severe anuric AKI secondary to this plant, complicated by acute pancreatitis.

CASE PRESENTATION

An 18-year-old female patient with no prior medical history was admitted for general deterioration and anuria. History-taking revealed oral ingestion of a preparation containing *Lawsonia inermis* 14 days prior to admission [2].

On examination, the patient was conscious, dyspneic, normotensive, afebrile, and showed no signs of fluid overload or dehydration. Cardiovascular and pulmonary examinations were unremarkable.

Laboratory Findings

- Serum creatinine: 197 mg/L
- Urea: 2.24 g/L
- Bicarbonate: 9 mmol/L (severe metabolic acidosis)
- Potassium: 2.2 mmol/L
- Hemoglobin: 8.2 g/dL (microcytic hypochromic anemia)
- Proteinuria: 0.48 g/24 h
- Serum lipase: 1,230 IU/L [3]

Complement C3 was decreased at 0.66 g/L, and serum protein electrophoresis showed a moderate elevation of the beta-2 globulin fraction. Immunological testing (ANA, anti-dsDNA, ANCA, anti-GBM) was negative [4,5].

Imaging

Abdominal computed tomography revealed stage I acute pancreatitis without necrosis or local complications [3].

Kidney Biopsy

Biopsy showed severe acute tubulointerstitial nephritis of probable toxic origin, without significant glomerular involvement. Direct immunofluorescence was negative [4–6].

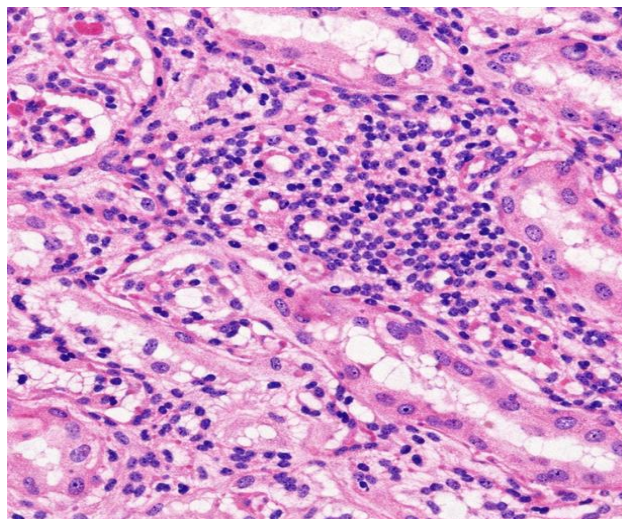


Figure 1: Renal biopsy

Management and Outcome

Due to anuria and the severity of metabolic disturbances, the patient underwent three sessions of hemodialysis [7]. Acute pancreatitis was managed conservatively with bowel rest, analgesia, and biological monitoring [3].

From day 4, diuresis progressively resumed, allowing discontinuation of hemodialysis. The patient continued to improve, with complete recovery of renal function at discharge: serum creatinine 19 mg/L, preserved urine output, and normalized lipase levels [3,7].

DISCUSSION

Severe AKI of toxic origin is often underestimated [4,5]. Medicinal plants may cause serious renal complications. Our observation illustrates a rare case of severe AKI secondary to ingestion of *Lawsonia inermis*, complicated by acute pancreatitis, suggesting a poorly documented systemic toxicity.

Renal involvement related to *Lawsonia inermis* is exceptional. Patil *et al.*, [2] reported that the plant may cause serious complications, although most documented cases involve cutaneous or hepatic reactions. The combination of severe AKI and acute pancreatitis is particularly rare and makes our observation noteworthy in the nephrology literature.

In this case, management consisted of immediate discontinuation of the toxic substance, hemodialysis, and conservative treatment of pancreatitis. Complete recovery of renal function demonstrates that

AKI due to *Lawsonia inermis* may be reversible if managed promptly [2,3,7,8].

This case also highlights the importance of thorough history-taking in any case of severe AKI, particularly in regions where medicinal plants are commonly used. Even young patients without comorbidities may develop severe complications, emphasizing the need to raise awareness among healthcare professionals and the public.

CONCLUSION

Ingestion of *Lawsonia inermis*, even for therapeutic purposes, may cause severe acute kidney injury and systemic involvement. Accurate history-taking and prompt management are crucial to prevent serious complications. Raising awareness among healthcare professionals and the public is essential to limit the risk of toxicity.

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