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Severe Cucurbitacin Toxicity Following Bitter Bottle Gourd Juice Consumption Presenting With Shock, Toxic Hepatitis, and Dialysis-Requiring Acute-on-Chronic Kidney Disease With Biopsy-Proven Renal Injury: A Case Report

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Abstract

Bottle gourd (*Lagenaria siceraria*) juice is widely consumed for its perceived health benefits; however, bitter bottle gourd containing high concentrations of cucurbitacins can cause severe, potentially fatal toxicity, including gastrointestinal symptoms, hypotension, acute liver injury, and acute kidney injury. Histopathological characterisation of the associated renal injury remains rare. We report a 53-year-old man with hypertension and chronic kidney disease who developed profuse vomiting, generalised rash, facial oedema, oliguria, and circulatory shock within 20-25 minutes of consuming bitter bottle gourd juice. Evaluation revealed marked leucocytosis with peripheral eosinophilia, severe high-anion-gap metabolic acidosis with lactic acidosis, acute hepatocellular injury, and severe acute-on-chronic kidney injury. Despite aggressive supportive therapy, renal function progressively deteriorated, requiring six sessions of intermittent haemodialysis. Renal biopsy demonstrated severe acute tubular injury with vacuolar degeneration, eosinophil-rich acute tubulointerstitial nephritis, and focal segmental glomerulosclerosis, with underlying chronic changes; immunofluorescence and electron microscopy showed no immune-complex deposits. Extensive evaluation excluded infectious, viral, autoimmune, and drug-induced causes. Formal causality assessment using the WHO-Uppsala Monitoring Centre (WHO-UMC) system, together with a Roussel Uclaf Causality Assessment Method (RUCAM) score, supported a probable association with bitter bottle gourd ingestion. The patient improved with supportive care and renal replacement therapy and, at the six-month follow-up, had recovered to a serum creatinine level of 1.23 mg/dL and was dialysis-independent. This case provides rare clinicopathological evidence of severe cucurbitacin toxicity causing dialysis-requiring acute kidney injury through combined direct tubular toxicity, ischaemic injury, and eosinophilic tubulointerstitial nephritis. Early recognition and prompt supportive care with timely renal replacement therapy are essential for favourable outcomes.

Categories: Emergency Medicine, Internal Medicine, Nephrology
Keywords: acute kidney injury, bottle gourd, cucurbitacin toxicity, renal biopsy, toxic hepatitis, tubulointerstitial nephritis

Introduction

Bottle gourd (*Lagenaria siceraria*), a member of the Cucurbitaceae family, is widely consumed as a vegetable and increasingly used as a health supplement because of its perceived benefits in weight reduction, glycaemic control, and cardiovascular health [1,2]. Although generally regarded as safe, consumption of bitter bottle gourd juice has been associated with severe and occasionally fatal toxicity due to high concentrations of cucurbitacins, naturally occurring tetracyclic triterpenoids responsible for the characteristic bitter taste [3,4].

Cucurbitacins possess potent cytotoxic, pro-inflammatory, and membrane-disrupting properties and can induce gastrointestinal mucosal injury, increased vascular permeability, systemic inflammatory responses, and multiorgan dysfunction [4,5]. Clinical manifestations typically develop within minutes to hours of ingestion and may include profuse vomiting, abdominal pain, diarrhoea, gastrointestinal bleeding, hypotension, shock, acute liver injury, and acute kidney injury [3,6]. Owing to reports of severe poisoning and fatalities, the Indian Council of Medical Research (ICMR) has advised against the consumption of bitter bottle gourd juice [7].

Acute kidney injury is a recognised complication of severe cucurbitacin toxicity; however, most published reports describe transient renal dysfunction diagnosed on clinical and biochemical grounds alone [3,6,8]. Histopathological characterisation of renal injury remains exceptionally uncommon, and the precise mechanisms underlying toxin-associated kidney damage have not been fully elucidated. Proposed mechanisms include direct tubular toxicity, ischaemic injury secondary to hypovolaemia and shock, and toxin-mediated inflammatory responses [4,5].

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Similarly, hepatic involvement has been reported in severe cases, with varying degrees of hepatocellular injury and cholestatic dysfunction [6,8]. Because these manifestations can mimic septic shock, acute viral hepatitis, ischaemic hepatitis, drug-induced liver injury, or other causes of multiorgan dysfunction, establishing the diagnosis often requires careful correlation of the clinical history with the systematic exclusion of alternative aetiologies.

To our knowledge, very few reports have documented renal biopsy findings in patients with bottle gourd toxicity, and reports describing dialysis-requiring acute-on-chronic kidney disease with concurrent biopsy-proven acute tubular injury and acute tubulointerstitial nephritis are exceedingly rare. This case highlights a severe presentation of cucurbitacin toxicity resulting in shock, toxic hepatitis, and dialysis-requiring acute-on-chronic kidney disease, and provides rare clinicopathological evidence supporting both direct toxic and inflammatory mechanisms of renal injury.

Case Presentation

A 53-year-old man presented with recurrent vomiting, decreased urine output, generalised erythematous rash, and facial swelling following ingestion of bitter bottle gourd (*Lagenaria siceraria*) juice.

The patient had been consuming freshly prepared bottle gourd juice regularly for approximately two months because of its perceived health benefits. On the day of presentation, he noticed that the juice tasted unusually bitter. Approximately 20-25 minutes after ingestion, he developed profuse vomiting, estimated at nearly 20 episodes, followed by a generalised rash, facial oedema, and progressive oliguria (urine output of 100 mL in 24 hours). There was no associated diarrhoea, haematemesis, melaena, abdominal pain, fever, recent infection, exposure to nephrotoxic medications or herbal preparations, alcohol use, or recreational drug use.

At an outside healthcare facility, he was noted to be hypotensive, with a systolic blood pressure of approximately 70 mmHg, and was subsequently referred to our tertiary care centre for further management.

His medical history was significant for hypertension and chronic kidney disease, with a documented baseline serum creatinine of approximately 2 mg/dL in 2017; however, intervening renal function records were unavailable. There was no prior history of chronic liver disease, autoimmune disease, vasculitis, or recurrent allergic disorders.

On arrival at our institution, the patient was conscious, oriented, and haemodynamically stable following initial resuscitation. Blood pressure was 124/76 mmHg, pulse rate 102 beats per minute, respiratory rate 20 breaths per minute, and oxygen saturation 99% on room air. General physical examination revealed facial puffiness and a residual erythematous skin rash. No lymphadenopathy, icterus, pedal oedema, or features of chronic liver disease were identified. Cardiovascular, respiratory, abdominal, and neurological examinations were otherwise unremarkable.

Laboratory evaluation revealed marked leucocytosis, with a total leucocyte count of 30,000/mm^3. The differential count showed neutrophils of 27 × 10^9/L, lymphocytes of 1.6 × 10^9/L, and monocytes of 0.17 × 10^9/L, with associated peripheral eosinophilia (absolute eosinophil count approximately 1.2 × 10^9/L). Haemoglobin was 15.5 g/dL, and the platelet count was 136,000/mm^3. Renal function tests demonstrated severe acute-on-chronic kidney injury, with blood urea of 153 mg/dL and serum creatinine of 6.8 mg/dL at presentation. Liver biochemistry showed significant hepatocellular injury, with total bilirubin of 4.0 mg/dL (direct bilirubin 2.6 mg/dL), aspartate aminotransferase (AST) of 840 IU/L, and alanine aminotransferase (ALT) of 1789 IU/L. Inflammatory markers were elevated, including CRP of 47.6 mg/L and procalcitonin of 8.3 ng/mL.

Arterial blood gas analysis demonstrated severe high-anion-gap metabolic acidosis with lactic acidosis (pH 7.20, partial pressure of carbon dioxide (PCO_2) 24 mmHg, bicarbonate 9.6 mmol/L, lactate 4.85 mmol/L), consistent with tissue hypoperfusion secondary to shock. The clinical course and key laboratory trends are summarised in Tables *1*-*2*.

Time point	Clinical events and findings
Day 0, 20-25 min after ingestion	Bitter bottle gourd juice consumed; approximately 20 episodes of vomiting, generalised rash, facial oedema, and oliguria.
Day 0 (referring facility)	Hypotension (systolic BP approximately 70 mmHg); intravenous fluid resuscitation; referred to a tertiary care centre.
Day 0 (admission)	BP 124/76 mmHg, HR 102/min; WCC 30,000/mm³ with peripheral eosinophilia (AEC approximately 1.2 × 10^9/L); urea 153 mg/dL, creatinine 6.8 mg/dL; AST 840 IU/L, ALT 1789 IU/L; bilirubin 4.0 mg/dL; CRP 47.6 mg/L, procalcitonin 8.3 ng/mL; arterial pH 7.20, bicarbonate 9.6 mmol/L, lactate 4.85 mmol/L.
During hospitalisation (22 days)	Six sessions of intermittent haemodialysis performed on hospital days 0, 2, 4, 5, 8, and 16.
Day 15	Renal biopsy demonstrating acute tubular injury, eosinophil-rich tubulointerstitial nephritis, and FSGS with chronic changes.
Discharge (Day 22)	Creatinine 3.7 mg/dL; AST 13 IU/L, ALT 18 IU/L; rash resolved. Advised to avoid bitter bottle gourd products and undergo nephrology follow-up.
Six-month follow-up	Dialysis-independent; serum creatinine recovered to 1.23 mg/dL.

TABLE 1: Clinical course timeline.

AEC: Absolute eosinophil count; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; BP: Blood pressure; FSGS: Focal segmental glomerulosclerosis; HR: Heart rate; WCC: White cell count.

Parameter	At presentation	Peak derangement	At discharge	Six-month follow-up	Adult reference range
Serum creatinine (mg/dL)	6.8	12	3.7	1.23	0.7-1.3 (adult male)
Blood urea (mg/dL)	153	342	58	Normal	15-40
AST (IU/L)	840	840	13	Normal	10-40
ALT (IU/L)	1789	1789	18	Normal	7-56
Total bilirubin (mg/dL)	4	5.1	0.86	Normal	0.2-1.2
Total leucocyte count (/mm³)	30,000	30,000	Normalising	6,610	4,000-11,000
CRP (mg/L)	47.6	91.8	Declining	Normal	<5
Arterial pH	7.2	7.2	Corrected	-	7.35-7.45

TABLE 2: Trend of key laboratory parameters.

ALT: Alanine aminotransferase; AST: Aspartate aminotransferase.

Further investigations were undertaken to evaluate alternative causes of multiorgan dysfunction. Viral hepatitis serologies were negative. Blood and urine cultures remained sterile throughout the hospital stay. Serum creatine phosphokinase was normal, effectively excluding rhabdomyolysis. Urinalysis demonstrated microscopic haematuria (15-20 RBCs/high-power field) and proteinuria; a 24-hour urinary protein collection performed later during admission, after urine output had improved, showed protein excretion of approximately 1.4 g/day (1384.8 mg/day).

USG of the abdomen demonstrated bilaterally increased renal cortical echogenicity, consistent with chronic kidney disease, and grade I hepatic steatosis. No evidence of biliary obstruction or chronic liver disease was identified.

Despite aggressive fluid resuscitation, correction of metabolic acidosis, and supportive care, renal function progressively worsened during hospitalisation. Serum creatinine increased from 6.8 mg/dL on admission to a peak of 12 mg/dL, accompanied by persistent oliguria and worsening azotaemia. The patient subsequently required six sessions of intermittent haemodialysis for the management of severe acute-on-chronic kidney disease and metabolic acidosis.

In contrast, hepatic dysfunction improved gradually with supportive treatment, with AST decreasing from 840 IU/L to 13 IU/L and ALT from 1789 IU/L to 18 IU/L by discharge. Supportive treatment included intravenous fluids, sodium bicarbonate, N-acetylcysteine, ursodeoxycholic acid, proton-pump inhibitor therapy, and empirical broad-spectrum antibiotics pending exclusion of infection.

Given the severity of renal dysfunction and the presence of underlying chronic kidney disease, a renal biopsy was performed. Light microscopy demonstrated focal segmental glomerulosclerosis involving two glomeruli, severe acute tubular injury characterised by extensive vacuolar degeneration and granular cast formation, focal acute tubulointerstitial nephritis with eosinophil-rich inflammatory infiltrates, tubular atrophy, interstitial fibrosis involving approximately 20-25% of the cortex, and arteriolar hyalinosis with fibrointimal sclerosis (Figure *1*).

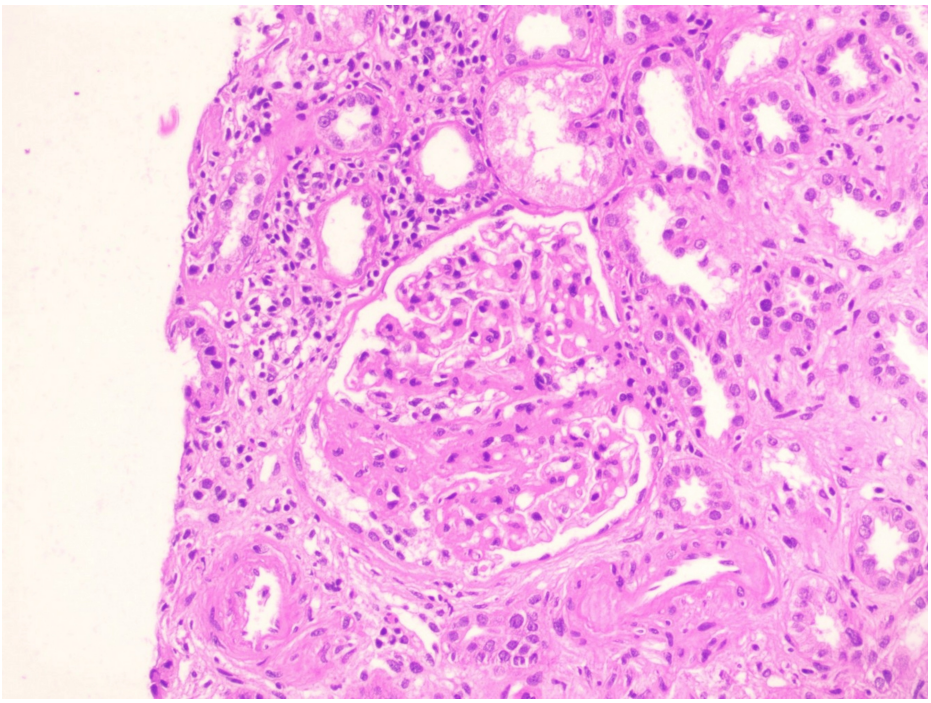


FIGURE 1: Light micrograph of the renal biopsy (H&E staining) showing a glomerulus with segmental sclerosis consistent with focal segmental glomerulosclerosis, together with an interstitial inflammatory infiltrate, tubular injury, and tubular atrophy with early interstitial fibrosis (original magnification, ×400).

Immunofluorescence was negative for IgA, IgG, IgM, C3, C1q, and kappa and lambda light chains, with no immune-complex deposition. Transmission electron microscopy confirmed the absence of electron-dense immune-type deposits and demonstrated a glomerular basement membrane of approximately normal thickness (measured at 231.57 nm and 280.24 nm), with diffuse podocyte foot-process effacement, in keeping with podocyte injury rather than immune-complex-mediated glomerulonephritis (Figures *2*-*4*).

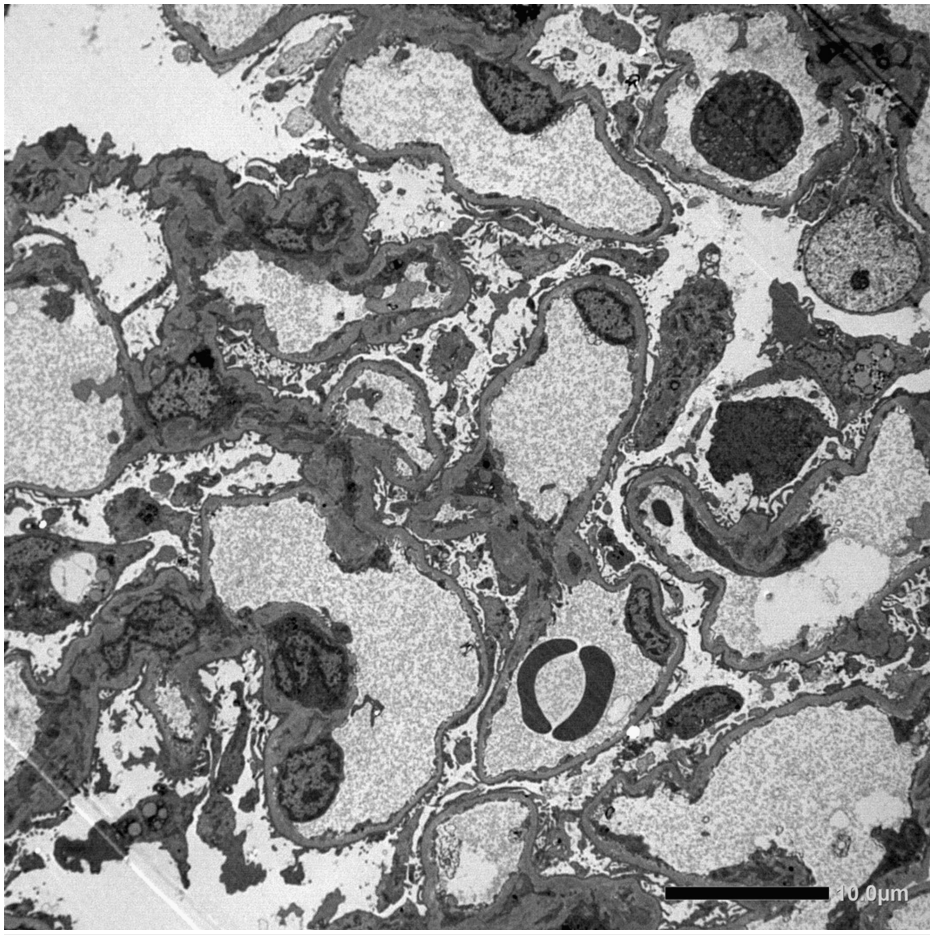


FIGURE 2: Low-power transmission electron micrograph of a glomerular capillary loop demonstrating the glomerular basement membrane, endothelium, and overlying podocytes (original magnification, ×600; scale bar = 10 µm).

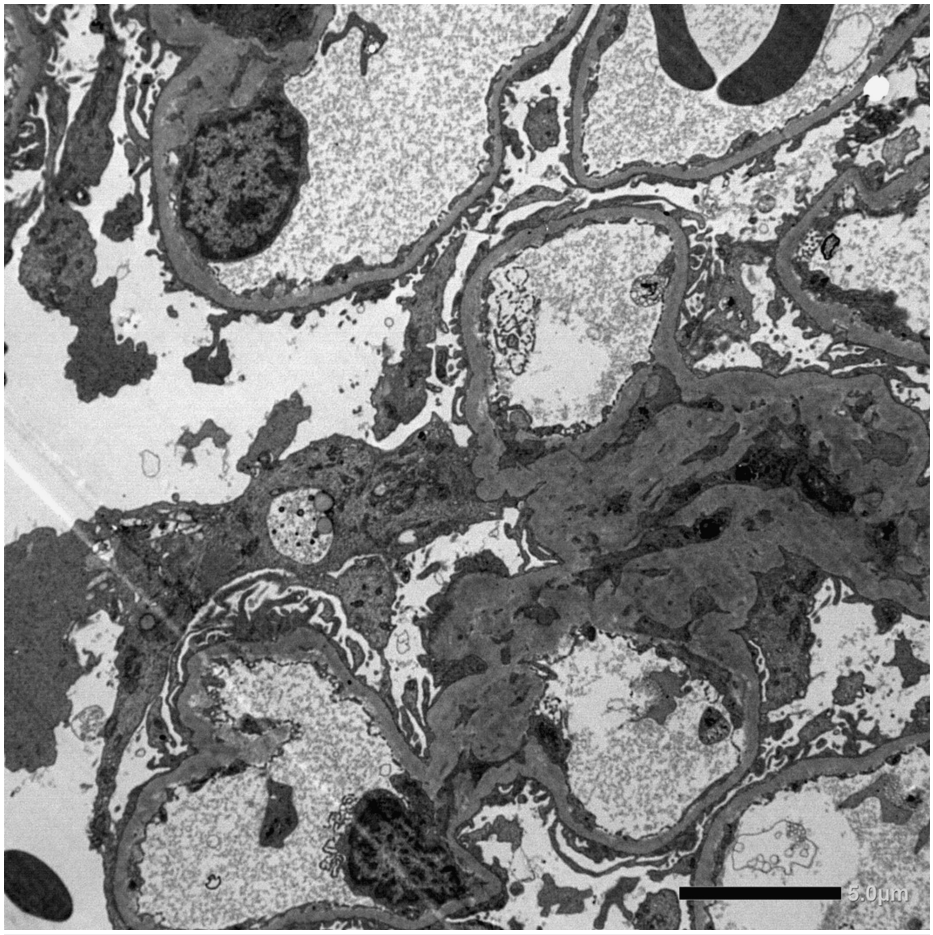


FIGURE 3: Higher-power transmission electron micrograph of the glomerular basement membrane, which is of approximately normal thickness (measured at 231.57 nm and 280.24 nm), with associated podocyte foot-process effacement and no electron-dense immune-type deposits (original magnification, ×1200; scale bar = 5.0 µm).

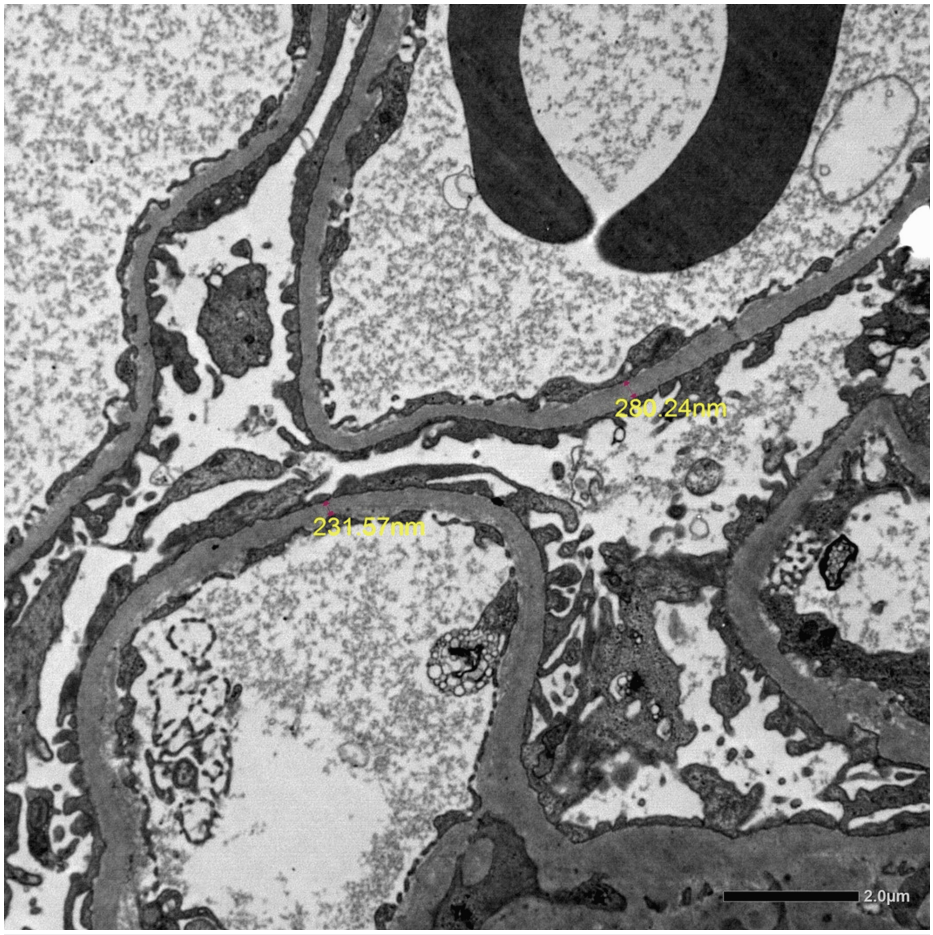


FIGURE 4: Transmission electron micrograph showing glomerular capillary loops, mesangium, and podocytes with diffuse foot-process effacement and absence of electron-dense deposits, in keeping with podocyte injury (original magnification, ×2500; scale bar = 2.0 µm).

No crescents, necrotising lesions, or thrombotic microangiopathy were identified. The chronic changes (focal segmental glomerulosclerosis, arteriolar hyalinosis, and interstitial fibrosis) were consistent with the patient's pre-existing hypertensive chronic kidney disease, whereas the acute tubular injury and eosinophil-rich tubulointerstitial nephritis represented the superimposed acute insult.

The temporal relationship between ingestion of bitter bottle gourd juice and symptom onset, the characteristic clinical manifestations, exclusion of infectious and alternative aetiologies, supportive histopathological findings, a "probable/likely" WHO-Uppsala Monitoring Centre (WHO-UMC) causality grading [9], and a Roussel Uclaf Causality Assessment Method (RUCAM) score [10] in the probable range for the hepatic component together established the diagnosis of severe cucurbitacin toxicity resulting in shock, toxic hepatitis, and dialysis-requiring acute-on-chronic kidney disease.

The patient showed gradual clinical improvement with supportive management and renal replacement therapy. Urine output progressively recovered, liver function tests normalised, and inflammatory markers declined. At discharge, serum creatinine had improved to 3.7 mg/dL, and the patient was advised to strictly avoid bitter bottle gourd products and undergo close nephrology follow-up. At outpatient review six months later, he remained dialysis-independent, with serum creatinine recovering to 1.23 mg/dL, better than the documented 2017 baseline, indicating substantial and largely complete recovery of renal function.

Discussion

Bottle gourd (*Lagenaria siceraria*) toxicity is an uncommon but potentially life-threatening manifestation of cucurbitacin poisoning. Although bottle gourd is widely consumed as a vegetable and health supplement, ingestion of bitter bottle gourd juice may result in severe systemic toxicity due to high concentrations of cucurbitacins, naturally occurring tetracyclic triterpenoids with potent cytotoxic and pro-inflammatory properties [1-5]. Clinical manifestations typically develop within minutes to hours of ingestion and may

include profuse vomiting, abdominal pain, gastrointestinal bleeding, hypotension, shock, acute liver injury, and acute kidney injury [3,6-8].

In the present case, the abrupt onset of profuse vomiting, generalised rash, facial oedema, oliguria, and circulatory shock within 20-25 minutes of ingestion strongly suggested acute cucurbitacin toxicity. The temporal relationship between exposure and symptom onset, together with subsequent severe metabolic acidosis, toxic hepatitis, and acute-on-chronic kidney disease, provided compelling evidence of severe multisystem involvement. Similar rapid-onset presentations have been described previously, although most have demonstrated predominantly gastrointestinal manifestations with less severe end-organ dysfunction [3,6,8].

Acute kidney injury is a recognised complication of severe cucurbitacin toxicity; however, most reported cases describe transient renal dysfunction diagnosed on clinical grounds alone [3,6,8]. In contrast, our patient developed severe dialysis-requiring acute-on-chronic kidney disease, with serum creatinine rising from 6.8 mg/dL to a peak of 12 mg/dL despite aggressive supportive therapy. The biopsy findings are best understood as a "two-hit" process: the chronic lesions (focal segmental glomerulosclerosis, arteriolar hyalinosis, and interstitial fibrosis) reflect long-standing hypertensive chronic kidney disease and probably explain the heightened susceptibility to injury, whereas the acute tubular injury and eosinophil-rich tubulointerstitial nephritis represent the superimposed acute toxic insult. Notably, the marked recovery of renal function at six months (serum creatinine 1.23 mg/dL, better than the 2017 baseline) indicates that the dominant contributor to the presenting renal failure was the reversible acute toxic and inflammatory injury, superimposed on milder underlying chronic disease than the admission creatinine of 6.8 mg/dL alone would have suggested.

The pathogenesis of renal injury in cucurbitacin toxicity remains incompletely understood. Experimental studies have demonstrated that cucurbitacins induce cellular apoptosis, oxidative stress, mitochondrial dysfunction, and membrane disruption, resulting in direct cytotoxic injury [4,5]. In our patient, renal biopsy revealed severe acute tubular injury with extensive vacuolar degeneration and granular cast formation, providing pathological evidence supporting direct tubular toxicity, while profound hypotension at presentation is likely to have contributed additional ischaemic injury through renal hypoperfusion.

An important and unusual finding was the eosinophil-rich acute tubulointerstitial nephritis, accompanied by peripheral eosinophilia (absolute eosinophil count approximately 1.2×10^9/L). Eosinophilic interstitial nephritis is classically a hypersensitivity reaction; rather than attributing this to a conventional culprit medication, none of which had been taken, the most parsimonious explanation is a hypersensitivity reaction to the ingested bitter juice itself, plausibly facilitated by prior sensitisation over the preceding two months of regular consumption, occurring in parallel with its direct cytotoxic effects. This interpretation is reinforced by the accompanying peripheral eosinophilia and by the generalised rash and facial oedema that developed within minutes of ingestion, and it supports a multifactorial pathogenesis involving toxic, ischaemic, and immune-mediated pathways.

Hepatic involvement was another prominent feature, with AST and ALT levels of 840 IU/L and 1789 IU/L, respectively. The injury was hepatocellular in pattern, and the rapid biochemical improvement after removal of the offending exposure strongly favoured toxin-mediated hepatitis over chronic liver disease or viral hepatitis. Applying the RUCAM for hepatocellular injury, short latency, more than a 50% fall in transaminases within days of withdrawal, exclusion of viral and biliary causes, and a previously reported hepatotoxic agent, yielded a score in the "probable" range (approximately 7).

The differential diagnosis included septic-shock-associated acute kidney injury, acute viral hepatitis, rhabdomyolysis, drug-induced interstitial nephritis, and ischaemic acute tubular necrosis. The combination of marked leucocytosis (30,000/mm^3), an elevated CRP level (47.6 mg/L), and a raised procalcitonin level (8.3 ng/mL) initially raised strong concern for bacterial sepsis, and empirical broad-spectrum antibiotics were commenced pending culture results. Several lines of evidence, however, argued against an infectious aetiology. First, procalcitonin is not specific for bacterial infection: clinically significant elevations are well recognised in a range of non-infectious conditions characterised by severe sterile inflammation and tissue injury, including shock, major tissue damage, and toxic systemic inflammatory responses, mediated by cytokine-driven (TNF-α, IL-1β, IL-6) extrathyroidal procalcitonin transcription [11]. Furthermore, procalcitonin undergoes renal clearance, so the patient's severe acute-on-chronic kidney injury would independently have raised circulating concentrations irrespective of infection [11]. Second, the extraordinarily rapid onset of shock within 20-25 minutes of ingestion is incompatible with the time course expected of septic shock arising from a fresh gastrointestinal source. Third, no infective focus was identified, and blood and urine cultures remained sterile throughout admission; antibiotics were accordingly de-escalated and discontinued once cultures returned sterile and the clinical picture stabilised, with no subsequent infective relapse. Viral hepatitis was excluded serologically, and rhabdomyolysis was excluded by a normal creatine phosphokinase level. Applying the WHO-UMC causality assessment system to the overall reaction, the case satisfied the criteria for a "probable/likely" association: a plausible temporal relationship to ingestion, manifestations unlikely to be attributable to concurrent disease or other drugs, and clinical improvement following withdrawal of the offending exposure (positive dechallenge), with rechallenge neither performed nor ethically appropriate.

To date, very few reports have documented renal histopathology in bottle gourd toxicity, and reports describing dialysis-requiring acute-on-chronic kidney disease with biopsy-proven acute tubular injury and eosinophilic tubulointerstitial nephritis remain exceptionally rare [3,6,8]. The present case therefore expands current understanding of cucurbitacin-associated renal injury by providing direct clinicopathological evidence supporting multiple mechanisms of kidney damage. A comparison with previously published reports is provided in Table *3*.

Study	Age and sex	Shock/ hypotension	Acute hepatic injury	Acute kidney injury	Dialysis required	Renal biopsy
Khatib KI et al., (2014) [3]	55 years, male	Yes	Mild transaminitis	No significant AKI	No	Not performed
Verma A and Jaiswal S (2015) [6]	52 years, female	Yes	Not clearly documented	Not clearly documented	No	Not performed
Chavda DM et al., (2022) [8]	64 years, female	Yes	Present (AST 882 IU/L, ALT 633 IU/L)	AKI (serum creatinine 2.44 mg/dL)	No	Not performed
Present case	53 years, male	Yes	Severe (AST 840 IU/L, ALT 1789 IU/L)	Severe AKI on CKD (peak creatinine 12 mg/dL)	Yes (six sessions)	FSGS with severe ATI and eosinophilic ATIN

TABLE 3: Comparison of published reports of severe bottle gourd (cucurbitacin) toxicity.

AKI: Acute kidney injury; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; ATI: Acute tubular injury; ATIN: Acute tubulointerstitial nephritis; FSGS: Focal segmental glomerulosclerosis.

A limitation of this report is the inability to directly quantify serum or tissue cucurbitacin concentrations, as such testing is not routinely available. The remote baseline creatinine (2017) without interval data also limits precise staging of the chronic component. Although the initial inflammatory-marker elevation means sepsis cannot be excluded with absolute certainty, the sterile cultures, the implausibly rapid onset, the recognised non-specificity of procalcitonin in renal failure and sterile inflammation, and the uneventful course after antibiotic withdrawal collectively render infection highly unlikely. The characteristic history, rapid symptom onset, systematic exclusion of alternatives, compatible clinicopathological findings, and probable causality grading strongly support cucurbitacin toxicity as the underlying aetiology.

Conclusions

Cucurbitacin toxicity following ingestion of bitter bottle gourd juice is an uncommon but potentially life-threatening condition that can cause severe multiorgan dysfunction, including shock, toxic hepatitis, and acute kidney injury. Severe dialysis-requiring acute-on-chronic kidney disease with histopathological characterisation of renal injury remains exceptionally rare.

This case demonstrates that cucurbitacin toxicity may produce profound renal dysfunction through direct tubular toxicity, ischaemic injury secondary to shock, and a hypersensitivity-type inflammatory response, suggesting a possible multifactorial mechanism of acute kidney injury. The biopsy-proven acute tubular injury and eosinophilic acute tubulointerstitial nephritis, together with peripheral eosinophilia, provide rare clinicopathological evidence supporting these mechanisms and expand the recognised spectrum of renal manifestations associated with bottle gourd poisoning. The substantial recovery of renal function at six months further underscores that aggressive, timely supportive care can achieve favourable outcomes. Clinicians should maintain a high index of suspicion in patients presenting with abrupt gastrointestinal symptoms, circulatory collapse, acute liver injury, and acute kidney injury after ingestion of bitter bottle gourd juice and should institute early supportive management, including renal replacement therapy when indicated.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. All India Institute of Medical Sciences, Bilaspur HP issued approval AIIMS/BLS/IEC-BHR/W-CERT/26-50. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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