Chronic Lymphocytic Leukemia Renal Infiltration Masquerading as Tumor Lysis Syndrome

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Introduction

Renal infiltration in chronic lymphocytic leukemia (CLL) is uncommon, reported in 1–2% of patients with renal dysfunction, though autopsy studies suggest subclinical involvement in up to 90% of cases. Diagnosis is often delayed because the presentation mimics tumor lysis syndrome (TLS), drug toxicity, or obstruction. We report a case of biopsy-proven CLL renal infiltration initially suspected to represent TLS.

Objective and Significance

Objective:

- Present a case of biopsy-proven CLL renal infiltration initially misattributed to tumor lysis syndrome.

Significance:

- Highlights the diagnostic pitfalls of acute kidney injury in CLL.
- Emphasizes the need for histopathologic confirmation to distinguish from TLS or obstruction.
- Underscores the clinical importance: therapy may control leukemia, but renal recovery is inconsistent.

Case

A 64-year-old man with chronic lymphocytic leukemia (13q deletion, Rai stage 2) and high-grade urothelial carcinoma presented nine days after right nephroureterectomy with abdominal pain and oliguria. Laboratory studies (Table 1) showed acute kidney injury with severe hyperkalemia, leukocytosis, and elevated uric acid. Imaging revealed no hydronephrosis. Pathologic review of the nephrectomy specimen demonstrated diffuse lymphoid infiltration, and bone marrow biopsy confirmed CLL without Richter transformation. The differential diagnosis included tumor lysis syndrome versus leukemic renal infiltration.

Results

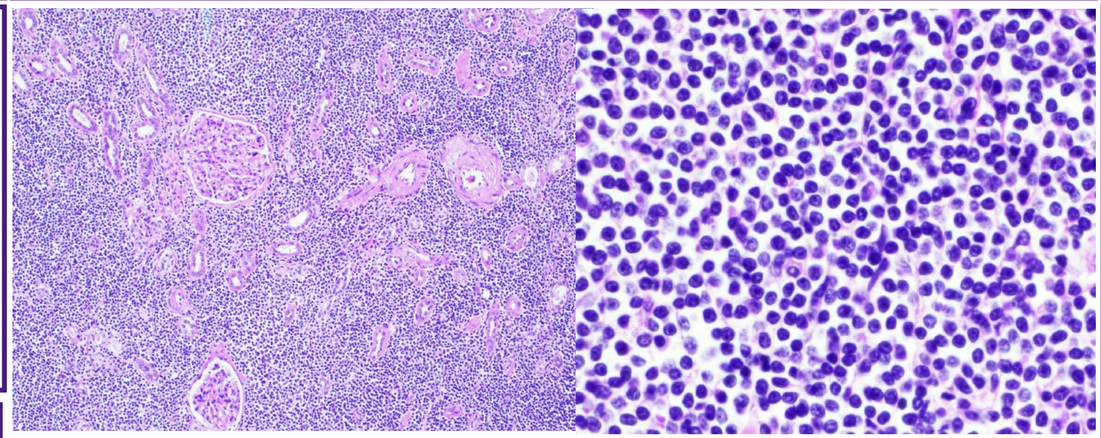

Figure 1. Renal infiltration by chronic lymphocytic leukemia.
Hematoxylin and eosin–stained nephrectomy specimen showing diffuse interstitial infiltration of small, mature lymphocytes with preserved glomeruli and tubules. The cells display clumped chromatin, consistent with CLL/small lymphocytic lymphoma. Immunohistochemistry (not shown) confirmed a $CD5^+/CD20^+/CD23^+$ B-cell phenotype, diagnostic of CLL renal infiltration.

Lab	Result	Normal Range
Creatinine	8.6 mg/dL	0.6–1.3
Potassium	6.4 mmol/L	3.5–5.0
Uric Acid	9.3 mg/dL	3.5–7.2
Phosphate	8.6 mg/dL	2.5–4.7
Calcium	7.7 mg/dL	8.4–10.3
LDH	259 U/L	140–280
WBC	149×10^9/L	4–11 $\times 10^9$/L

Table 1. Admission laboratory results showing severe acute kidney injury, hyperkalemia, hyperuricemia, and leukocytosis. Elevated phosphate and low-normal calcium further complicated interpretation, initially raising concern for tumor lysis syndrome.

Step	Key Findings	Interpretation
Presentation	Abdominal pain, oliguria, ↑Cr, electrolyte derangements	Acute kidney injury (AKI)
Initial Impression	Labs consistent with hyperkalemia, hyperuricemia, hyperphosphatemia, hypocalcemia	Raised concern for TLS
Context Check	No recent cytotoxic therapy, stable CLL on surveillance	TLS unlikely
Imaging	No hydronephrosis or obstruction	Obstructive etiology excluded
Pathology	Nephrectomy specimen: diffuse lymphoid infiltration; bone marrow CLL without Richter	Confirms CLL renal infiltration
Management	Hemodialysis + zanubrutinib 80 mg BID	Targeted therapy initiated
Outcome	No renal recovery	Dialysis dependent

Table 2. Diagnostic reasoning pathway for AKI in CLL. Despite TLS-like laboratory abnormalities, absence of a cytotoxic trigger and histopathology established CLL renal infiltration as the true etiology.

Discussion and Limitations

CLL renal infiltration represents a diagnostic challenge, often mimicking tumor lysis syndrome, drug toxicity, or obstructive nephropathy. Infiltration occurs through interstitial accumulation of malignant B cells, driven by aberrant trafficking and retention mechanisms. While hematologic therapy can lead to renal improvement in approximately 60% of cases, recovery is inconsistent, and many patients progress to end-stage renal disease. Bruton tyrosine kinase (BTK) inhibitors achieve high hematologic response rates in CLL, but renal outcomes in biopsy-proven infiltration remain poorly defined. This case underscores that irreversible renal damage may limit recovery despite targeted therapy. Limitations include the single-patient nature of this report, restricting generalizability and highlighting the need for larger studies to identify predictors of renal response and guide management.

Conclusion

This case highlights CLL renal infiltration as a rare but critical cause of irreversible kidney failure. Despite disease control with targeted therapy, renal function did not recover, emphasizing that histopathologic confirmation is essential and that early recognition may be the only chance to preserve renal outcomes.

References

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