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Chronic Lymphocytic Leukemia Renal Infiltration Masquerading as Tumor Lysis Syndrome

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 (Arora et al., 2016; Poitou-Verkinder et al., 2015; Strati et al., 2015). Diagnosis is often delayed as 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.

Case

A 64-year-old man with CLL (13q deletion, Rai stage 2) and high-grade urothelial carcinoma presented nine days after right nephroureterectomy with abdominal pain and oliguria. His post operative course was complicated by hydronephrosis of the left kidney requiring ureteral stent placement with subsequent improvement in kidney function. His CLL, diagnosed in 2017, had been briefly treated with acalabrutinib but was discontinued due to hematuria; surveillance white blood cell (WBC) counts remained 140–200 × 10^9/L. Admission labs showed creatinine 8.6 mg/dL (baseline 1.3), potassium 6.4 mmol/L, uric acid 9.3 mg/dL, lactate dehydrogenase 259 U/L, and WBC 149 × 10^9/L. CT and ultrasound imaging did not show hydronephrosis. TLS was suspected; however, the absence of recent cytotoxic therapy and overall stable disease argued against this diagnosis. Review of the recent nephrectomy specimen revealed extensive lymphoid infiltration of the renal parenchyma consistent with CLL/small lymphocytic lymphoma. Bone marrow biopsy confirmed CLL without Richter transformation. Renal function failed to recover, and hemodialysis was initiated. Zanubrutinib (80 mg twice daily) was started for CLL-related renal infiltration, however the patient remained dialysis dependent at discharge.

Discussion

This case illustrates the diagnostic challenge of CLL renal infiltration in the setting of multiple potential causes of acute kidney injury. Infiltration occurs via interstitial accumulation of malignant B lymphocytes, driven by aberrant trafficking and retention mechanisms. In a multicenter biopsy series, approximately 60% of patients experienced renal improvement after hematologic therapy, although many progressed to end-stage renal disease in the absence of response (Poitou-Verkinder et al., 2015). Bruton tyrosine kinase (BTK) inhibitors achieve hematologic response rates of 89–92% in CLL, yet renal outcomes in biopsy-proven infiltration remain poorly defined (Yin et al., 2024). This case underscores the therapeutic rationale for BTK inhibition, while highlighting that irreversible renal damage may limit recovery despite treatment.

Conclusion

Renal infiltration in CLL, though uncommon, is an important cause of acute renal failure. Diagnosis requires a high index of suspicion and histopathologic confirmation. While targeted therapies may offer benefit, renal recovery is inconsistent, highlighting the need for prospective studies to identify predictors of renal response and establish evidence-based management strategies.