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An Unusual Case of Acute Kidney Injury due to Renal Infiltration by T-cell Lymphoblastic Lymphoma in an 18-Year-Old Male: A Case Report

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Abstract

Background: T-cell lymphoblastic lymphoma (T-LBL) is an aggressive hematological malignancy that primarily affects young adults and adolescents. Renal involvement by T-LBL presenting as acute kidney injury (AKI) is exceedingly rare and often overlooked in clinical practice. The non-specific clinical presentation frequently mimics infectious etiologies, leading to diagnostic delays and inappropriate treatment.

Aims and Objectives: To highlight a rare presentation of AKI caused by renal infiltration of T-LBL, initially mimicking an infectious etiology, and to emphasize the role of renal biopsy in unexplained non-recovering AKI.

Materials and Methods: An 18-year-old male presented with non-oliguric AKI (serum creatinine 6.8–10 mg/dL) along with symptoms of burning micturition and abdominal pain, initially treated as acute pyelonephritis. Despite 14 days of intravenous antibiotics and supportive therapy, there was no renal recovery, and the patient required two sessions of hemodialysis. A renal biopsy was performed to evaluate the persistent AKI. The renal tissue was processed for light microscopy with H&E, PAS, Masson's trichrome, and silver methenamine stains; direct immunofluorescence (DIF); and immunohistochemistry

(IHC) panel including CD3, CD20, CD5, PAX5, TdT, and Ki-67.

Results: Light microscopy revealed glomeruli with non-proliferative morphology without crescents or necrosis. The interstitium was diffusely infiltrated by monomorphic small round blue cells. IHC profile demonstrated CD3 (4+), CD5 (4+), negative CD20 and PAX5, high Ki-67 index (80–85%), and dim patchy TdT positivity. These findings were consistent with T-cell lymphoblastic lymphoma infiltrating the renal interstitium.

Conclusion: Renal involvement by T-cell lymphoblastic lymphoma is a rare and often under-recognized cause of AKI. This case underscores the importance of renal biopsy in evaluating persistent or unexplained AKI, especially in young patients with non-specific symptoms and no recovery after initial therapy. Early recognition allows timely hematology referral and initiation of lymphoma-directed treatment.

Keywords: Acute kidney injury; T-cell lymphoblastic lymphoma; Renal infiltration; Renal biopsy; Atypical lymphoid cells; Immunohistochemistry; CD3; TdT; Ki-67; Young adult

Introduction

T-cell lymphoblastic lymphoma (T-LBL) is an aggressive neoplasm arising from immature T-cell precursors, accounting for approximately 2% of all non-Hodgkin lymphomas in adults.[1] It predominantly affects adolescents and young adults, with a male predominance. The typical clinical presentation includes a mediastinal mass, lymphadenopathy, and bone marrow involvement. However, extranodal manifestations, particularly isolated renal infiltration, are exceptionally rare.[2,3]

Acute kidney injury (AKI) in the context of hematological malignancies can occur through various mechanisms including tumor lysis syndrome, nephrotoxicity from chemotherapeutic agents, sepsis, and direct parenchymal infiltration by malignant cells.[4] Direct renal infiltration by lymphoma cells, although well-documented in autopsy series, rarely presents clinically as the primary manifestation of disease.[5]

The non-specific clinical presentation of renal infiltration by lymphoma often mimics more common conditions such as acute pyelonephritis or acute interstitial nephritis, leading to diagnostic delays. This case report presents an unusual presentation of AKI in a young male caused by renal infiltration of T-LBL, initially misdiagnosed as acute pyelonephritis, highlighting the critical role of renal biopsy in establishing the diagnosis in cases of unexplained non-recovering AKI.

Case Presentation

An 18-year-old male, with no significant past medical history, presented to the emergency department with complaints of burning micturition and bilateral flank pain of one-week duration. He also reported low-grade fever and decreased appetite. There was no history of hematuria, oliguria, edema, joint pain, skin rash, or recent drug intake. Physical examination revealed a young male in mild distress with tenderness in both renal angles. There was no palpable lymphadenopathy or hepatosplenomegaly.

Initial laboratory investigations revealed significantly elevated serum creatinine levels ranging from 6.8 to 10 mg/dL, consistent with severe AKI. Urinalysis showed pyuria with no significant proteinuria. Blood cultures were sterile. Ultrasonography of the kidneys revealed bilaterally enlarged kidneys with increased cortical

echogenicity but no hydronephrosis or calculi. Based on the clinical presentation and initial investigations, a provisional diagnosis of acute pyelonephritis was made, and the patient was started on empirical intravenous antibiotics.

Despite 14 days of appropriate intravenous antibiotic therapy and supportive management, there was no improvement in renal function. The patient required two sessions of hemodialysis due to uremic symptoms. Given the non-recovering nature of AKI and atypical clinical course, a renal biopsy was planned to investigate the underlying etiology.

Materials and Methods

Ultrasound-guided percutaneous renal biopsy was performed from the left kidney using an 18-gauge automated biopsy gun. Two cores of renal tissue were obtained. The biopsy specimen was processed for comprehensive histopathological evaluation.

Light microscopy examination was performed using hematoxylin and eosin (H&E), periodic acid-Schiff (PAS), Masson's trichrome (MT), and silver methenamine stains to evaluate glomerular, tubular, interstitial, and vascular compartments. Direct immunofluorescence (DIF) was performed on frozen sections to assess for immune complex deposition using antibodies against IgG, IgA, IgM, C3, C1q, fibrinogen, and kappa and lambda light chains.

Given the unusual interstitial infiltrate observed on light microscopy, an immunohistochemistry (IHC) panel was performed on paraffin-embedded sections. The panel included CD3 (T-cell marker), CD20 (B-cell marker), CD5, PAX5, TdT (terminal deoxynucleotidyl transferase), and Ki-67 (proliferation marker) to characterize the infiltrating cells and establish a definitive diagnosis.

Results

Light microscopy revealed adequate renal tissue with an adequate number of glomeruli for evaluation. The glomeruli showed non-proliferative morphology with no evidence of crescents, necrosis, or segmental sclerosis. The glomerular basement membranes appeared normal without thickening or duplication.

The most striking finding was in the interstitium, which was diffusely infiltrated by a population of monomorphic small round blue cells with high nuclear-to-cytoplasmic ratio, fine chromatin, and inconspicuous nucleoli. These cells were distributed throughout the interstitium, surrounding and separating the tubules. The tubules showed focal acute tubular injury with flattening of epithelium and loss of brush border. Blood vessels appeared unremarkable.

Direct immunofluorescence was negative for all tested immunoreactants, ruling out immune complex-mediated glomerulonephritis.

Immunohistochemistry demonstrated the following profile:

1- CD3: Strongly positive (4+), confirming T-cell lineage
2- CD5: Strongly positive (4+)
3- CD20: Negative, ruling out B-cell origin
4- PAX5: Negative
5- Ki-67: High proliferation index (80–85%), indicating aggressive nature
6- TdT: Dim patchy positivity, consistent with immature T-cell phenotype

The combined histomorphological features and IHC profile were consistent with T-cell lymphoblastic lymphoma (T-LBL) infiltrating the renal interstitium. This rare diagnosis explained the patient's non-recovering AKI despite appropriate antibiotic therapy.

Discussion

This case highlights an unusual presentation of T-cell lymphoblastic lymphoma manifesting primarily as AKI due to direct renal parenchymal infiltration. T-LBL typically presents with mediastinal masses, superior vena cava syndrome, and lymphadenopathy, making isolated renal presentation exceptionally rare.[1,2]

Renal involvement in lymphomas can occur through several mechanisms: direct infiltration, obstruction by retroperitoneal lymphadenopathy, glomerulonephritis (paraneoplastic), or treatment-related nephrotoxicity.[6] While autopsy studies have reported renal infiltration in up to 34% of patients with non-Hodgkin lymphoma, clinically significant AKI from direct infiltration is uncommon and rarely the presenting feature.[5,7]

The initial clinical presentation in our patient with fever, flank pain, and pyuria closely mimicked acute pyelonephritis, leading to empirical antibiotic therapy. This diagnostic pitfall has been reported in the literature, where lymphomatous renal infiltration can present with non-specific symptoms indistinguishable from infectious or inflammatory conditions.[8]

The key to diagnosis in this case was the decision to perform a renal biopsy when the patient's AKI failed to resolve despite appropriate initial therapy. The histopathological findings of monomorphic small round blue cell infiltrate with characteristic IHC profile (CD3+, CD5+, CD20−, high Ki-67, TdT+) were diagnostic of T-LBL.[9] The high Ki-67 index (80–85%) reflected the aggressive proliferative nature of this malignancy.

TdT positivity is a hallmark of lymphoblastic neoplasms, representing immature lymphoid precursors. While TdT positivity is typically strong in T-LBL, the dim patchy pattern observed in our case may reflect tumor heterogeneity or technical factors related to tissue processing.[10]

The differential diagnosis of small round blue cell infiltration in renal biopsy includes various lymphoproliferative disorders, small cell carcinoma metastasis, and Ewing sarcoma. IHC is essential for accurate classification. In our case, the T-cell phenotype (CD3+, CD5+) with absence of B-cell markers (CD20−, PAX5−) and presence of TdT confirmed the diagnosis of T-LBL.[11]

Early diagnosis is crucial as T-LBL is a highly aggressive malignancy that requires prompt initiation of intensive chemotherapy regimens similar to those used for acute lymphoblastic leukemia.[12] With appropriate treatment, T-LBL has a relatively favorable prognosis compared to other aggressive lymphomas, with complete remission rates exceeding 70% in young patients.[13]

Conclusion

Renal involvement by T-cell lymphoblastic lymphoma is a rare and often under-recognized cause of AKI. This case underscores the importance of maintaining a high index of suspicion and pursuing renal biopsy in cases of persistent or unexplained AKI, especially in young patients with non-specific symptoms and lack of response to initial therapy. Early histopathological diagnosis with appropriate IHC panel enables timely hematology referral and initiation of lymphoma-directed chemotherapy, which is essential for favorable outcomes in this aggressive but potentially curable malignancy.

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