CASE REPORT

Leukemic Infiltration of Kidney in a Case of T-cell Acute Lymphomatous Leukemia

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ABSTRACT

Kidney involvement in acute lymphomatous leukemia (ALL) presenting as acute kidney injury (AKI) is uncommon. We document a case report of a 19-year-old male who presented with loss of weight, weakness, and anorexia. On evaluation, he was found to have T-cell ALL with AKI. Renal biopsy was done, and immunohistochemistry (IHC) confirmed renal infiltration by the leukemic T cells. The patient was started on chemotherapy, and improvement of renal function with subsequent bone marrow showed an initial complete remission. This case illustrates the uncommon presentation of T-cell ALL as AKI.

Keywords: Acute kidney injury, Acute lymphomatous leukemia, Leukemic infiltration.

INTRODUCTION

Acute kidney injury (AKI) is a significant problem in patients with acute leukemia, which can be due to the basic ailment itself or by the medications.¹ Factors that can lead to AKI in this situation commonly are sepsis and decreased perfusion.²,³ Other complications seen are metabolic abnormalities, kidney invasion by malignant cells, obstructive uropathy, glomerulonephritis, and drug toxicity. Infiltration of leukemic cells in the renal parenchyma is mostly occult and is mostly seen in autopsy findings in as many as 33–63% of patients.⁴ AKI due to invasion of kidney in T-cell acute lymphoblastic leukemia (ALL) is rare and is uncommon seen as initial presentation.⁵ We present a case report of T-cell ALL with AKI due to leukemic infiltration of the kidney.

CASE DESCRIPTION

A 19-year-old male reported to us with complaints of lassitude, decreased appetite, and loss of weight lasting for 3 months. He had no significant previous medical ailment. On examination of the patient, he had significant pallor with normotension and normal pulse and respiratory rates. His laboratory findings are detailed in Table 1. Bone marrow biopsy was performed after performing peripheral blood smear. The bone marrow examination revealed characteristics of acute leukemia which was further verified by flow cytometry to be a case of T-cell ALL. The kidney biopsy was performed for determining the cause of AKI. Kidney biopsy revealed diffuse layer-like invasion into interstitium due to monomorphic lymphoid cells and severe tubular atrophy. In renal histopathology, approximately three glomeruli were seen, one of which shows rupture of Bowman’s capsule with infiltration of the Bowman’s space by leukemic cells (Fig. 1). Immunohistochemistry showed these lymphoid cells to be positive for CD7 and Tdt with negative staining for CD20 (Fig. 2). These findings confirm leukemic infiltration of the kidney by T-cell ALL. The treatment protocol for T-cell ALL was initiated, subsequent to which he developed severe tumor lysis syndrome (TLS) that required hemodialysis apart from medical treatment of TLS. Dose reduction was done for daunorubicin and methotrexate. Cyclophosphamide was particularly reduced in dosage to avoid serious complications. Patient improved with renal functions and TLS, and succeeding bone marrow examination demonstrated complete remission (CR).

DISCUSSION

Several factors such as infection, obstructive uropathy, uric acid nephropathy, or leukemic infiltration may contribute to the pathogenesis of renal failure in patients with ALL. Around 10% of patients with ALL develop severe uric acid nephropathy following chemotherapy.⁶ Although kidneys are major extramedullary sites of leukemic invasion, it most frequently is subclinical and usually seen on autopsy findings.⁷ Invasion by leukemic cells are seen diffusely in both kidneys with more involvement of cortex. Interstitial infiltration by leukemia cells is seen with less involvement of glomerulus. This infiltration leads to separation of nephrons and may cause compression and degeneration of tubules.⁸

Our case was unusual in many ways. First, it presented with AKI and bilateral renal enlargement. Leukemic infiltration of the kidneys rarely presents as renal dysfunction and usually is clinically silent. In a study of 938 patients with ALL, 0.4% of patients had clinical evidence of renal dysfunction, while 10% had organ

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Table 1: Laboratory findings at presentation

| Laboratory parameter | Values with units |
|----------------------|-------------------|
| Hemoglobin           | 7.5 g/dL          |
| Leukocyte count      | 10.35 × 10³/μL    |
| Platelet count       | 187 × 10³/μL      |
| Differential leukocyte count | Neutrophil-59%, lymphocyte 35%, eosinophil-3%, monocytes 3% |
| Peripheral blood smear | Normocytic normochromic anemia with few of blast cells seen |
| ESR                  | 60 mm/AEH         |
| Creatinine           | 2.66 mg/dL        |
| Urea                 | 98 mg/dL          |
| LDH                  | 825 UI/L          |
| pH                   | 7.2               |
| Bicarbonate          | 15 mEq/L          |
| Liver function tests | Normal            |
| Sodium               | 135 mmol/L        |
| Potassium            | 5.6 mmol/L        |
| Calcium              | 6.1 mg/dL         |
| Phosphorous          | 5.4 mg/dL         |
| Uric acid            | 4.5 mg/dL         |
| Chloride             | 111 mEq/L         |
| Total protein; Albumin | 7.68 G/dL; 2.7 G/dL |
| Viral markers (HIV, HBV, HCV) | Nonreactive |
| Urine examination    | Albumin trace; WBC 3–5/HPF; RBC-nil. 24 hours urinary protein excretion 313 mg/day |
| Abdominal ultrasound | Bilateral enlarged bulky hyperechoic kidneys (right, 15 cm; left, 16 cm). Normal corticomedullary differentiation and no hydronephrosis |
| Computed tomography of the abdomen | Bilateral bulky kidneys with left renal calculus. No obstruction seen. |

Patients with excessive tumor load have most tumor cells in the production stage. These cells may get lysed releasing intracellular content. This can lead to hyperkalemia, hyperphosphatemia, and hyperuricemia. Precipitation and deposition of urate and calcium phosphate crystals can impair kidney functions. ALL rarely presents as spontaneous TLS, usually developing after combined chemotherapy. In our patient, AKI on presentation was unlikely because of TLS, as he had no other features of TLS except hypocalcemia and no chemotherapeutic drugs were given prior to the admission.

Around 25% of children with ALL can present with renal enlargement and are associated with earlier relapse. Chemotherapeutic treatment can have differential effect on clearance of leukemic cells from various organs. Kidney can act as a reservoir for leukemic cells with clearance from bone marrow.

In autopsy of patients with ALL who were in remission, microscopic evidence of leukemic cells was seen in kidney. Although these early studies suggested poor prognostic role of kidney enlargement in ALL, the development of more intensive regimen and its role is questionable. A study by Neglia et al. showed no role of kidney enlargement in prognosis of ALL.

Acute kidney injury in ALL patients is associated with poor outcome, less complete remission rate, and higher mortality rates. In our patient, renal impairment necessitated modification of chemotherapy protocol used for treating ALL. Dose reduction was done for chemotherapeutic drugs. Our patient developed TLS after chemotherapy which was treated with hypouricemic drugs and dialysis support.

**Conclusion**

Leukemic cell infiltration into the renal parenchyma is not rare and possibly related to the role of kidney in hematopoiesis during early embryonic stage. AKI, however, is uncommon at the initial presentation of T-cell ALL and renal dysfunction usually is a side effect of treatment. Kidney biopsy is needed for early diagnosis. The renal dysfunction necessitates modification of chemotherapy drugs to avoid further renal damage and to mitigate toxic effects of drugs that can accumulate due to less renal clearance. Kidney function can improve fully with the treatment of ALL.
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Figs 2A to D: (A) Hematoxylin and eosin (H&E) staining (40x) showing tumor cell morphology; (B) IHC showing TdT-positive cells; (C) Immunohistochemistry (IHC) showing CD7-positive tumor cell; (D) IHC showing CD20-negative tumor cell