EJCRIM
European Journal
of Case Reports
in Internal Medicine

ISOLATED BILATERAL NEPHROMEGALY AS THE INITIAL PRESENTATION OF ACUTE LYMPHOBLASTIC LEUKAEMIA: A RARE CASE AND REVIEW OF THE LITERATURE

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Received: 06/05/2026
Accepted: 18/05/2026
Published: 09/07/2026

Conflicts of Interests: The Authors declare that there are no competing interests.
Patient Consent: Written informed consent was obtained from the patient's family for publishing this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.
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How to cite this article: Hamdan R, Musleh M, Alhamadeh Alswij M, Ibraheem Y, Laflouf M, Mohamed Alyousef A. Isolated bilateral nephromegaly as the initial presentation of acute lymphoblastic leukaemia: a rare case and review of the literature. *EJCRIM* 2026;**13**:doi:10.12890/2026_006944

ABSTRACT

Introduction: Acute lymphoblastic leukaemia (ALL) is an aggressive hematologic malignancy, with T-cell ALL (T-ALL) accounting for approximately 25% of adult cases. Renal infiltration is recognized but rarely presents as isolated bilateral nephromegaly with acute kidney injury (AKI) in adults.

Case description: A 32-year-old previously healthy man presented with progressive bilateral flank pain, abdominal heaviness, fatigue, intermittent fever, night sweats, and unintentional weight loss. Physical examination revealed large bilateral flank masses without peripheral lymphadenopathy. Laboratory tests initially showed normal blood counts but significant renal impairment (creatinine 3.5 mg/dl, urea 82 mg/dl). Imaging demonstrated massive bilateral kidney enlargement (~20 × 20 cm). Percutaneous renal biopsy revealed diffuse infiltration by medium-sized lymphoblasts expressing CD3 and TdT, confirming T-ALL. Within 1-week, hematologic abnormalities developed, including leucocytosis, anaemia, and thrombocytopenia, alongside worsening renal function and tumour lysis syndrome, necessitating dialysis and allopurinol therapy. The patient was started on a pre-phase corticosteroid regimen followed by intensive hyper-CVAD (hyperfractionated cyclophosphamide, vincristine, doxorubicin [Adriamycin], and dexamethasone) induction chemotherapy. Initial radiologic improvement occurred; however, the clinical course was complicated by neutropenic sepsis, ultimately resulting in death.

Conclusion: This case highlights a rare adult presentation of T-ALL in which renal involvement preceded hematologic abnormalities. Clinicians should consider hematologic malignancy in adults presenting with unexplained bilateral nephromegaly and AKI, even if initial blood counts are normal. Early recognition and timely intervention are critical to improve renal and overall outcomes.

KEYWORDS

Kidney infiltration, acute lymphoblastic leukaemia

EFIM
European Federation of Internal Medicine

Publisher
smc media srl

LEARNING POINTS

- Renal infiltration is recognized but rarely presents as isolated bilateral nephromegaly with acute kidney injury (AKI) in adults.
- This report underscores the importance of considering underlying hematologic malignancy in adults presenting with unexplained bilateral nephromegaly and AKI, even when initial hematologic investigations are normal.

INTRODUCTION

Acute lymphoblastic leukaemia (ALL) is an aggressive hematologic malignancy defined by the rapid expansion of lymphoid precursor cells, serving as the most prevalent cancer in children and a significant clinical challenge in adults[1]. T-cell acute lymphoblastic leukaemia (T-ALL), which stems from the malignant transformation of immature thymocytes, constitutes approximately 25% of adult ALL cases[2]. Extranodal lymphoma most commonly affects the gastrointestinal tract (30–40%); however, primary or early renal involvement is uncommon and is often involved in advanced systemic disease. Manifestation of the disease through acute kidney injury (AKI) and significant bilateral renal enlargement as the primary presenting feature is a distinct clinical anomaly[3].

Statistical data indicates that while radiographic nephromegaly is observable in roughly 30% of ALL patients at diagnosis, overt renal failure primary to direct leukemic infiltration is documented in fewer than 1% of cases[4]. This report describes an exceptionally rare clinical presentation in a 32-year-old adult, highlighting a diagnostic challenge that occurs when the complete blood count (CBC) remains unremarkable despite extensive organ involvement[2]. In such instances, the renal interstitium is densely infiltrated by malignant lymphoblasts, which exert mechanical pressure on the tubules and microvasculature, ultimately leading to acute organ dysfunction[3].

CASE DESCRIPTION

A 32-year-old previously healthy man presented to the emergency department with a 2-month history of progressive bilateral flank pain and generalized fatigue. He also reported a sensation of abdominal heaviness that was minimally responsive to analgesic therapy. His symptoms were accompanied by fever, drenching night sweats, and unintentional weight loss during the same period.

On physical examination, large bilateral flank masses were palpable. No peripheral lymphadenopathy was noted at initial presentation. Abdominal ultrasonography revealed markedly enlarged kidneys bilaterally, each measuring approximately 20 × 20 cm, without evidence of hydronephrosis.

Initial laboratory evaluation demonstrated a white blood cell count of $10\times10_9$/l, haemoglobin of 13 g/dl, and platelet count of 240 $\times10_9$/l. However, renal function was significantly impaired, with a serum creatinine of 3.5 mg/dl and blood urea nitrogen of 82 mg/dl. The parathyroid hormone level was 15.8 pg/ml.

A non-contrast-enhanced computed tomography scan of the abdomen confirmed massive bilateral renal enlargement *(Fig. 1)* without retroperitoneal lymphadenopathy or evidence of obstructive uropathy.

Given the unexplained nephromegaly and progressive renal dysfunction, a percutaneous biopsy of the right kidney was performed. Histopathological examination demonstrated diffuse infiltration of the renal parenchyma by medium-sized atypical lymphoid cells with a high nuclear-to-cytoplasmic ratio and frequent mitotic figures, consistent with acute T-cell lymphoblastic lymphoma/leukaemia (T-ALL/LBL) involving the kidney. Immunohistochemical analysis showed strong positivity for CD3 and terminal deoxynucleotidyl transferase (TdT) as shown in *Figure 2*, confirming T-lineage lymphoblastic neoplasia.

One week later, the patient experienced rapid clinical deterioration and a rapid decline in laboratory markers. Repeat laboratory studies revealed marked leucocytosis (white blood cell count 60 $\times10_9$/l), with a differential count demonstrating neutrophils 64% and lymphocytes 46%, anaemia (haemoglobin 8 g/dl), and thrombocytopenia (platelet count 100 $\times10_9$/l). Renal function worsened further (creatinine 4.4 mg/dl; urea 98 mg/dl). Additional findings included hyperuricemia (uric acid 13 mg/dl) and elevated lactate dehydrogenase (760 U/l), reflecting high tumour burden and rapid cellular turnover. Liver function tests were within normal limits (alanine aminotransferase 16 U/l, aspartate aminotransferase 17 U/l, total bilirubin 0.6 mg/dl, alkaline phosphatase 84 U/l), and serum calcium

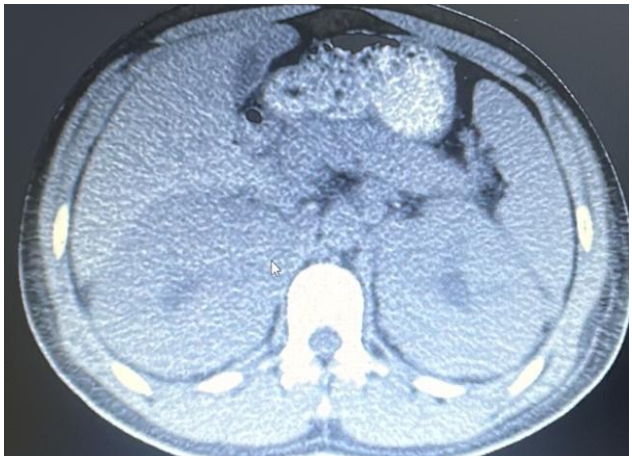


Figure 1. *Contrast-enhanced computed tomography scan of the abdomen showing massive bilateral kidney enlargement, each measuring approximately 20 cm.*

10.9 mg/dl. Physical examination at that time revealed generalized lymphadenopathy involving bilateral axillary, supraclavicular, and inguinal regions, with the largest lymph node measuring approximately 5 cm. Persistent bilateral flank masses were noted. The patient also developed grade 2 bilateral lower limb oedema and right-sided peripheral facial nerve palsy, raising suspicion for possible central nervous system involvement.

Peripheral blood smear demonstrated leucocytosis with circulating blasts. Bone marrow aspiration revealed a markedly hypercellular marrow with approximately 80% infiltration by lymphoid blasts as shown in *Figure 3*.

Flow cytometric immunophenotyping confirmed a T-cell lymphoblastic phenotype. The blast population expressed TdT (80%) and showed strong positivity for CD2 (98%), CD5 (100%), CD7 (97%), CD8 (74%), and CD10 (99%), with partial expression of CD3 (35%). These findings were consistent T-ALL/LBL. Conventional cytogenetic analysis demonstrated a normal karyotype.

Brain magnetic resonance imaging was performed to evaluate possible central nervous system involvement and demonstrated no evidence of intracranial pathology. Cerebrospinal fluid analysis was negative for malignant infiltration. Despite the absence of radiologic or cytologic evidence of central nervous system disease, the patient received prophylactic intrathecal chemotherapy consisting of dexamethasone, cytarabine, and methotrexate according to intrathecal injection protocol in ALL.

Pending final diagnostic confirmation, the patient was started on pre-phase corticosteroid therapy with prednisolone 100 mg daily for 7 days. During this period, he developed laboratory and clinical features consistent with tumour lysis syndrome, including worsening renal impairment and hyperuricemia, necessitating initiation of renal replacement therapy (haemodialysis) along with allopurinol and aggressive supportive management.

Following confirmation of T-ALL/LBL, induction chemotherapy was initiated using the hyper-CVAD regimen. This included cyclophosphamide 600 mg/m_2, doxorubicin 50 mg/m_2, and dexamethasone 40 mg daily for 4 days, followed by vincristine 1.4 mg/m_2 administered on day 8, according to institutional protocol.

The patient initially demonstrated clinical and radiological improvement, with significant regression of renal size to approximately 13 × 12 cm. However, his clinical course was complicated by severe neutropenic fever leading to septic shock. Despite intensive supportive care, the patient's condition deteriorated, ultimately resulting in death.

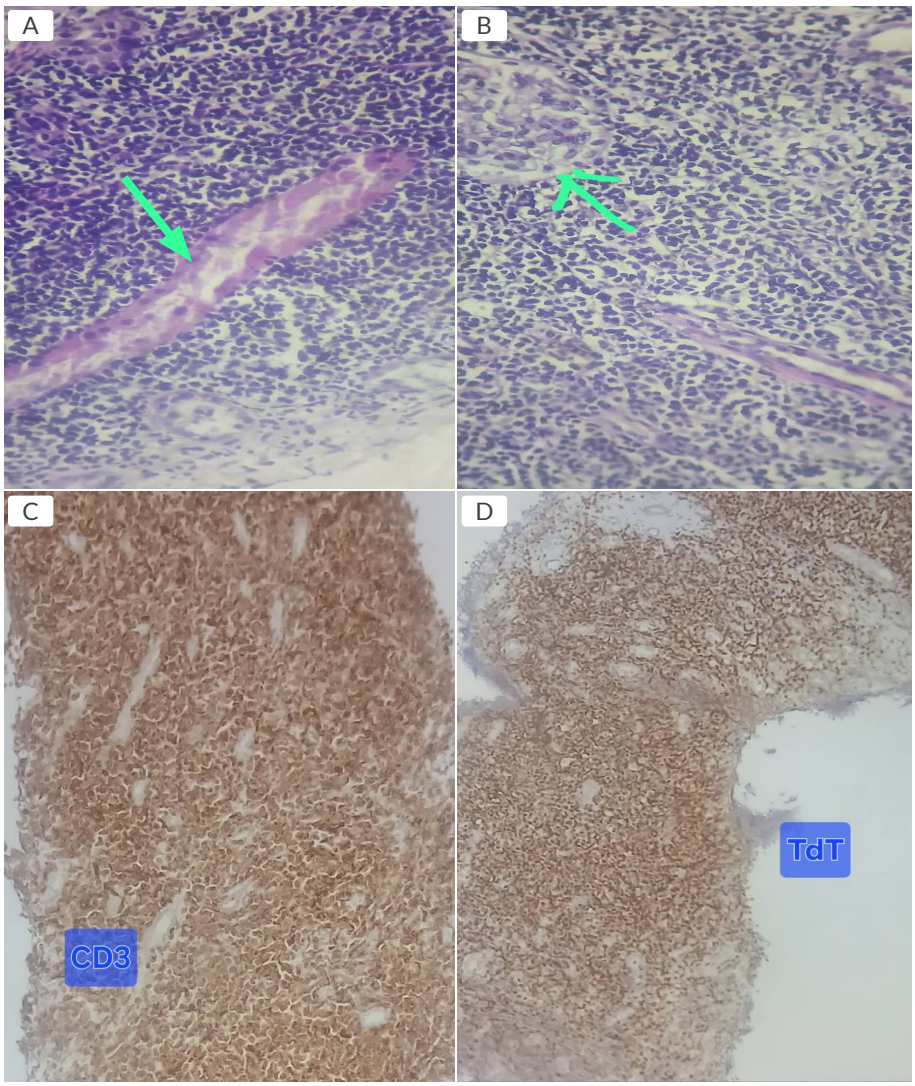


Figure 2. *Kidney biopsy demonstrating diffuse infiltration of the renal parenchyma by medium-sized atypical lymphoid cells with a high nuclear-to-cytoplasmic ratio and frequent mitotic figures (a, b), consistent with T-cell acute lymphoblastic leukemia/lymphoblastic lymphoma (T-ALL/LBL). Immunohistochemical analysis shows strong positivity for CD3 (c) and TdT (d), confirming T-lineage lymphoblastic neoplasia.*

DISCUSSION

ALL, particularly, typically presents with symptoms related to bone marrow involvement, including anaemia, thrombocytopenia, and leucocytosis, as well as lymphadenopathy and mediastinal masses. Common extramedullary sites of involvement include the liver, lymph nodes, spleen, and testis. Bilateral renal infiltration resulting in massive nephromegaly is a rare manifestation of T-ALL, predominantly reported in the paediatric population[5–7].

In several paediatric cases, nephromegaly was observed before the development of overt hematologic abnormalities, with initially normal or only minimally altered complete blood counts[6]. In our patient, bilateral nephromegaly was accompanied by AKI, consistent with direct renal infiltration.

Renal function at presentation can vary widely, ranging from preserved function to severe AKI requiring renal replacement therapy[5,6]. Only a few cases have documented AKI resulting directly from lymphomatous infiltration at diagnosis[9,10]. Although leukemic or lymphoma cells can infiltrate both the glomeruli and interstitium, the incidence of AKI and bilateral nephromegaly is considerably higher in interstitial infiltration (87% and 91%, respectively) than in intraglomerular involvement (45% and 10%, respectively) [7]. Interstitial infiltration is thought to cause AKI primarily through obstruction of the renal tubules by malignant cells, leading to increased intratubular pressure and secondary glomerular collapse[9].

Several paediatric cases have described massive nephromegaly in ALL. Prada Rico et al. proposed two mechanisms for renal enlargement in paediatric ALL: (I) Diffuse or nodular infiltration, often asymptomatic and incidentally detected at diagnosis by ultrasound, typically involving the renal cortex and frequently bilateral and symmetrical; and (II) Hypertrophy or hyperplasia of parenchymal cells, representing a secondary response rather than direct leukemic infiltration[11].

In our patient, the presence of nephromegaly and AKI warranted early intervention with allopurinol and dialysis for tumour lysis syndrome. In adults, cases of ALL presenting primarily with renal manifestations are exceedingly rare. Reported adult cases typically exhibit bilateral nephromegaly accompanied by AKI and concurrent hematologic abnormalities at diagnosis. AKI due to lymphomatous infiltration is uncommon; in a series of 48 patients with aggressive lymphomas and renal involvement, only three developed AKI[9]. Other potential contributors to renal failure in lymphoma include ureteric obstruction, hypercalcemia, urate nephropathy, sepsis, radiation nephritis, and paraproteinemia.

Management of such cases follows the 2024 European LeukemiaNet recommendations, which emphasize immediate initiation of intensive ALL therapy. Accordingly, our patient received a pre-phase corticosteroid regimen followed by intensive induction chemotherapy with hyper-CVAD, resulting in partial regression of renal enlargement.

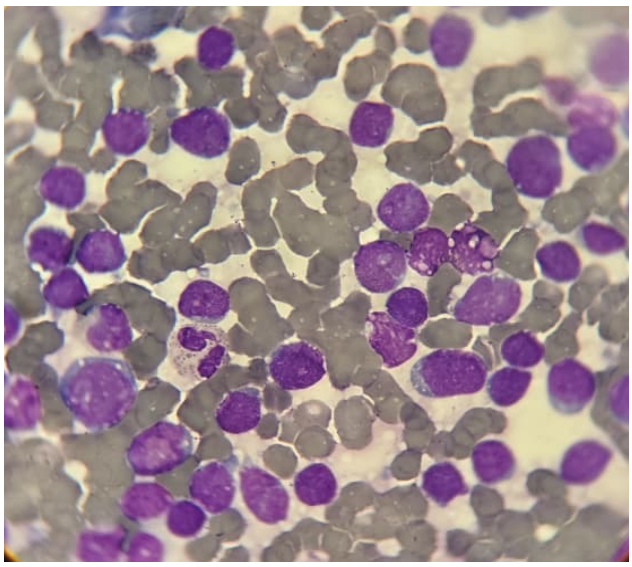


Figure 3. Bone marrow aspiration showing a markedly hypercellular marrow with approximately 80% infiltration by lymphoblasts. Giemsa staining, 100×magnification.

This case is particularly notable because it involves a 32-year-old adult who presented with rapidly progressive bilateral nephromegaly and AKI in the absence of hematologic abnormalities, with a completely normal complete blood count at initial presentation. Hematologic abnormalities developed approximately one month later, ultimately leading to the diagnosis of T-ALL. This delayed hematologic presentation distinguishes our case from most previously reported adult cases and parallels a small subset of paediatric cases in which renal involvement preceded marrow findings[5,6].

This report underscores the importance of considering underlying hematologic malignancy in adults presenting with unexplained bilateral nephromegaly and AKI, even when initial hematologic investigations are normal. Early recognition of this rare presentation may facilitate timely diagnosis and treatment, potentially improving both renal and overall outcomes.

CONCLUSION

Although renal involvement in ALL is a recognized phenomenon, isolated bilateral nephromegaly with AKI as the initial manifestation of adult T-ALL remains exceptionally rare. This case adds to the limited literature and highlights that delayed hematologic manifestations do not exclude leukaemia in patients with rapidly progressive renal enlargement.

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