Acute Lymphoblastic Leukemia Presenting as Bilateral Renal Enlargement in a Child

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Key Words
Lymphoblastic leukemia · Renal masses · Magnetic resonance imaging

Abstract

Objective: To report a case with early presentation of acute lymphoblastic leukemia (ALL) as bilateral renal masses and renal failure. Clinical Presentation and Intervention: A 6-year-old boy was admitted with bilaterally enlarged kidneys and severe renal impairment. Magnetic resonance imaging (MRI) showed bilateral renal enlargement with features suggestive of an infiltrative lesion. Accordingly, bone marrow examination was performed, and diagnosis of ALL was made. The patient developed acute renal failure after initiation of chemotherapy, so he received hemodialysis. His renal function normalized and kidney enlargement regressed. Conclusion: This case demonstrates an unusual early renal involvement in ALL in a child. MRI is a valuable imaging modality in the evaluation of renal masses.

Case Report

A 6-year-old Saudi Arabian boy was admitted to the general pediatric ward with a 3-month history of bilaterally enlarged kidneys. He had been complaining of abdominal pain for 3 months, but there was no history of hematuria, oliguria, anorexia, weight loss or night sweats. Apart from a short febrile illness at the start, he had no more febrile episodes thereafter. Recently, he had complained of pain in his right lower limb and he became fatigued easily.

On examination, mild pallor was noted; his blood pressure was 109/67 mm Hg, which was within the normal range for his age. His abdomen was distended, and the kidneys were bilaterally palpable, with smooth surfaces and no tenderness. The liver and spleen were not palpable. There was an enlarged (2 × 2 cm) nontender right inguinal lymph node and several small cervical and axillary lymph nodes. There was no bony tenderness and other systems were within normal ranges. Initial investigations were as follows. A complete blood count, which was carried out by an automated method, showed a hemoglobin level of 98 g/l, the total white blood cell count was 14 × 10\textsuperscript{9} /l, with neutrophils 22% and lymphocytes 68%. The platelet count was 137 × 10\textsuperscript{9} and the erythrocyte sedimentation rate was 98 mm/h. Blood urea nitrogen was 14.4 mmol/l, serum creatinine was 234 μmol/l, phosphorus was 2.39 mmol/l, calcium was 2.51 mmol/l, albumin was...
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24.1 g/l, uric acid was 668 µmol/l and lactate dehydrogenase was 2,441 IU/l. Serum sodium, potassium, liver enzymes and urine analysis were all within reference ranges. A chest X-ray showed no abnormality. Ultrasonography of the abdomen revealed bilaterally enlarged kidneys with a hyperechoic pattern and loss of corticomedullary differentiation. No evidence of hydronephrosis was noted. Magnetic resonance imaging (MRI) of the abdomen (fig. 1) showed bilaterally symmetrical enlargement of the kidneys, which were 15 cm in length, with loss of corticomedullary differentiation. The pelvicalyceal systems were not dilated and, bilaterally, the renal veins and arteries were patent. Lymph nodes, measuring about 1 cm, were noted in the para-aortic and retrocaval area; spleen and liver were normal. The MRI picture was suggestive of an infiltrative lymphoproliferative disorder.

The next day, the patient had a repeat blood count, and a peripheral blood smear was reviewed by the hematologist. The results were: hemoglobin 87 g/l; total white blood cell count 18.9 × 10^9/l, with neutrophils 18%, lymphocytes 32%, monocytes 1%, eosinophils 2%, blast cells 47%, and platelet count 72 × 10^9/l. Bone marrow aspiration was performed the following day and was consistent with ALL of the B-cell type.

The patient was then referred to the hematology-oncology unit, and chemotherapy was started according to the UK Medical Research Council ALL 99 high-risk protocol that included induction chemotherapy, standard consolidation and 3-year maintenance chemotherapy. He developed acute renal failure as his blood urea nitrogen increased to 29.6 mmol/l, phosphate to 5.95 mmol/l, potassium to 7.2 mmol/l and lactate dehydrogenase to 9,124 IU/l, and his blood calcium dropped to 1.73 mmol/l. He was transferred to the intensive care unit and was put on hemodialysis for 16 h. His renal function improved and chemotherapy treatment was resumed without deterioration in renal function. A bone marrow aspiration carried out after the completion of induction chemotherapy showed complete hematological remission. His renal function normalized and both kidneys were clinically unpalpable. A follow-up ultrasound study showed that both kidneys had decreased in size.

Discussion

Renal mass is a commonly encountered clinical problem in pediatrics. The differential diagnosis includes hydronephrosis, congenital anomalies and malignancy. Hydronephrosis is ruled out by ultrasound. Symmetrical enlargement of the kidneys with loss of corticomedullary differentiation, as seen by MRI, denotes an infiltrative lesion [6]. Several neoplastic and inflammatory conditions cause infiltrative renal lesions. Among these are renal medullary carcinoma, renal cell sarcoma, epithelial neoplasms, lymphoproliferative diseases and metastatic diseases [6]. Accordingly, blood count was repeated and a peripheral blood film was requested, which pointed to a possible diagnosis of leukemia. This was confirmed by the bone marrow aspiration.

Renal infiltration has been found in 50% of leukemic children at autopsy [7]. Renal involvement is known to occur late in the course of the disease; however, 3–5% have enlarged kidneys at presentation [8], as in this case. Moreover, renal failure is rarely a presenting symptom in patients with leukemia [2]. Derangement of renal function in ALL can be attributed to several factors: direct invasion by leukemic cells [9], urinary tract obstruction, glomerulonephritis due to immunologic reactions or treatment with nephrotoxic antibiotics, radiation nephropathy and antileukemic nephropathy [10]. Tumor lysis syndrome, either prior to chemotherapy (due to large tumor burden) or after initiation of chemotherapy, can cause renal failure. This is manifested as hyperuricemia, hyperphosphatemia, hyperkalemia and hypocalcemia [2]. Renal impairment in our patient can be explained by the massive infiltration of leukemic cells, as evidenced by the size of the kidneys and tumor lysis syndrome with further deterioration in renal function after initiation of chemotherapy.

Treatment of renal failure in such cases necessitates special attention to hydration, alkalization, electrolyte and metabolic correction, and dialysis support if indicated, together with early initiation of specific antileukemia chemotherapy [9].

The prognostic significance of renal size at presentation in childhood ALL is controversial. Hann et al. [11] related poorer prognosis with increasing renal size. However, Neglia et al. [12] found that kidney size does not affect the outcome, either as a single variable or after adjustment for the known prognostic factors of age, sex and initial white blood cell count.

Fortunately, this patient has made a remarkable recovery as a result of the appropriate management of renal
failure with hemodialysis and chemotherapy for ALL. His renal function is back to normal, the renal masses are not palpable and the kidneys are smaller in size. He is in hematological remission while continuing maintenance chemotherapy.

**Conclusion**

This case demonstrates an unusual early renal involvement in ALL in a child. Bone marrow examination should be included in the workup when an infiltrative lesion is demonstrated by MRI.

**References**