# Nephrotic syndrome and acute renal failure as a presenting symptom of acute lymphoblastic leukemia

## Sir,

A 15-year-old boy presented with low-grade fever, generalized body swelling and decreased urine output. On examination, he had pallor, pitting edema, and facial puffiness with no lymphadenopathy. His blood pressure, pulse rate, and respiratory rate were within normal limit. Hemoglobin was 7.6 g/dl, platelets 57,000/ mm<sup>3</sup>, leukocytes 22,300/mm<sup>3</sup> with 19.2% neutrophils and 64.5% lymphocytes, with blasts cells. Biochemistry revealed serum creatinine 3.16 mg/dl, uric acid 5.4 mg/dl, bilirubin 0.98 mg/dl, total protein 4.2 g/dl with albumin 1.90 g/dl, aspartate aminotransferase 198 IU/L, alanine aminotransferase 154 IU/L, lactate dehydrogenase 564 IU/L, serum cholesterol 325 mg/dl, and triglyceride 365 mg/dl. The electrolytes were within normal limits. Urinanalysis showed proteinuria of 3<sup>+</sup> without hematuria and 24 urinary protein was 3 g. Bone marrow examination showed hypercellular marrow, 34% blast cells, 10% myelocytes and metamyelocytes, 20% lymphocytes, and 10% neutrophils. Immune-phenotyping study showed blast cells positive for CD-3, CD-5, CD-7, cCD-3, CD-45, and TdT markers which were suggestive of mature T-cell acute lymphoblastic leukemia (ALL). He tested negative for HIV-1 and -2, hepatitis B surface antigen, hepatitis C virus, and malaria. Ultrasound of abdomen showed mildly enlarged kidneys and increased cortical echogenicity with the loss of cortico-medullary differentiation without evidence of obstruction in the urinary tract. Hepatosplenomegaly was present. Hemodialysis was done thrice. Dexamethasone, allopurinol, renal safe antibiotics, and blood products were given. He did not respond to treatment and expired 10 days after because of multiorgan dysfunction.

Nephrotic syndrome is commonly reported in Hodgkin and non-Hodgkin lymphomas, chronic lymphoblastic leukemia, and acute myeloid leukemia.<sup>[1]</sup> Acute renal failure (ARF) is a rare presenting symptom of ALL.<sup>[2]</sup> The patients with male sex, advanced age, history of previous kidney disease, refractory leukemia, and hyper-leukocytosis are at higher risk for developing renal complications. The renal dysfunction in leukemia occurs because of preexisting renal disorders, leukemic infiltration of kidneys, nephrotoxic drug exposure, obstructive uropathy, tumor lysis syndrome, and infections.<sup>[3,4]</sup> The renal dysfunction because of the leukemic infiltration of the kidneys is uncommon but should be suspected in patients presenting with ARF and diffusely enlarged kidneys.<sup>[4,5]</sup> The tubular or glomerular injury because of tumor cells or immune complex deposition and paraneoplastic manifestation of leukemia can lead to renal dysfunction.<sup>[5]</sup>

In our case, there was no evidence of hyperuricemia, infection, dehydration, exposure to nephrotoxic drugs, or obstructive pathology of the urinary tract. Anasarca and proteinuria were consistent with the diagnosis of nephrotic syndrome. The renal failure might be due to the leukemic infiltration of the kidney as both kidneys were slightly enlarged with increased cortical echogenicity along with hepatosplenomegaly.

To conclude, the patient presenting with clinical picture suggestive of nephrotic syndrome with anemia and atypical cells in the peripheral blood film should be evaluated for hematological malignancy, and a rare presentation of ALL should be kept in differential diagnosis in children.

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## **Conflicts of interest**

There are no conflicts of interest.

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