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Identification of renal infiltration based on urinary findings in a child with Burkitt leukemia/lymphoma

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A 9-year-old boy was admitted to hospital with oliguria, hypertension and periorbital edema. A complete blood count showed hemoglobin concentration 83 g/L, platelet count 43 × 10^9/L and leukocyte count 46 × 10^9/L. Biochemical investigations revealed elevated uric acid (9 mg/dL) and lactate dehydrogenase (2400 IU/L). The diagnosis of precursor B acute lymphoblastic leukemia (ALL) (CD10, CD19, CD22, CD34, terminal deoxynucleotidyl transferase (TdT), CD79a, HLA-DR positive) was made based on the finding of 60% L3 Burkitt-type lymphoblasts in the bone marrow (Figure 1A,B). The patient was diagnosed with t(8;14)(q24;q32) positive precursor B ALL with central nervous system involvement and tumor lysis syndrome (Figure 1C). The patient's electrolyte imbalance resolved but his urine output remained inadequate despite effective hydration and hemodialysis for tumor lysis syndrome. Radiological imaging and urinalysis suggested renal infiltration secondary to acute leukemia. Macroscopically, the patient's urine resembled lipemic plasma (Figure 1D), and microscopic examination revealed abundant blast cells (Figure 1E). These observations were supported by positron emission tomography and magnetic resonance imaging findings consistent with renal infiltration, including bilateral renal enlargement and pathological increased uptake of 18F-fluoro-2-deoxy-D-glucose. Epstein-Barr virus PCR was negative at diagnosis.

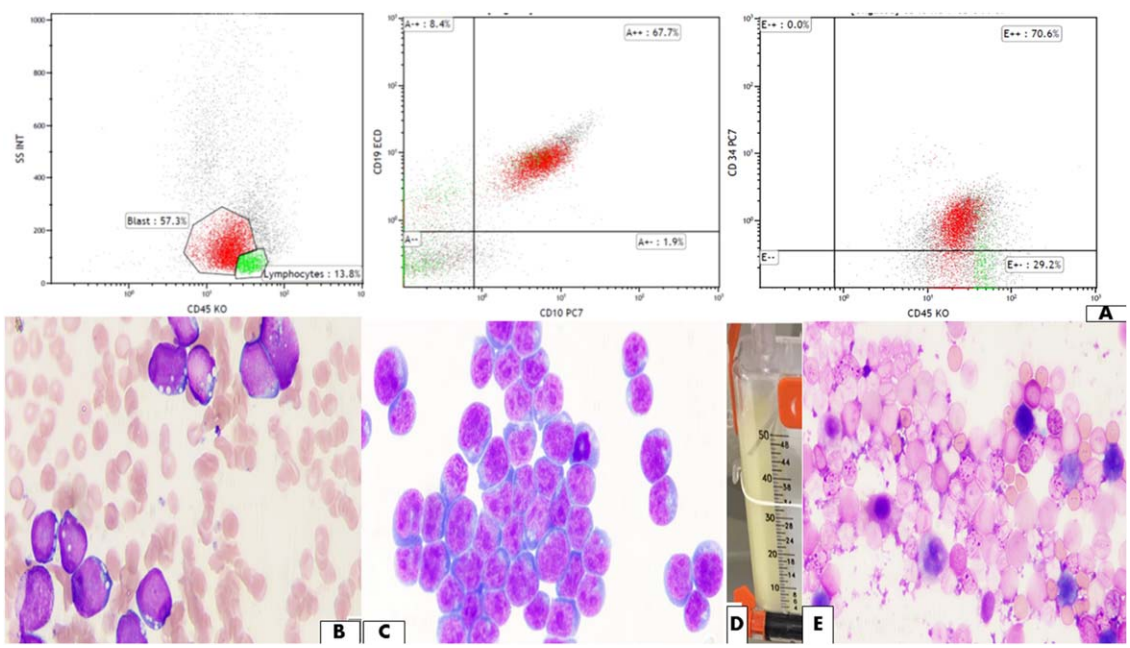


FIGURE 1 Demonstrating precursor B-cell populations by flow cytometry, (A); Widespread blastic infiltration of the bone marrow, (B), in the central nervous system, and (C) in a urine sample as macroscopic (D) and microscopic findings (E) in a patient with precursor B-cell ALL.

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The great majority of patients with t(8;14) express a mature B-cell phenotype with absence of CD34 and TdT, and presence of cytoplasmic heavy *mu* chain.[1] However, this patient's blast cells were positive for CD34 and TdT, and negative for surface immunoglobulin; no testing for cytoplasmic heavy *mu* chain was performed. Based on these findings, the patient's immunophenotyping was consistent with precursor B ALL. Although the boy was diagnosed with B lymphoblastic leukemia (a precursor lymphoid neoplasm) by flow cytometry immunophenotyping, the morphology, presence of t(8;14), and evidence of tumor lysis syndrome raise the possibility of Burkitt leukemia/lymphoma, a mature B-cell neoplasm. Coexistence of t(8;14)(q24;q32) and precursor B-cell phenotype was reported in approximately 0.1% of children with ALL in a study by the Pediatric Oncology Group.[2] Renal infiltration occurs in 7% to 42% of children with leukemia[3]; however, diagnosing renal infiltration in children who have recently been diagnosed with acute leukemia is very challenging. A literature search for this case type suggested radiological evidence of bilateral kidney enlargement as means of identifying malignant infiltration.[4] Renal infiltration in patients with Burkitt lymphoma has been reported previously, and blastic infiltration of the urine has been demonstrated by fluorescence *in situ* hybridization (FISH) and flow cytometry immunophenotyping.[5] Renal infiltration was confirmed by urinalysis, including macroscopic and microscopic urine examination, in a patient with leukemia. We did not perform advanced tests, such as FISH or immunophenotyping investigations, to identify blast cells in the urine because the patient had widespread leukemic infiltrations. As our case indicates, bilateral renal enlargement, presence of blast cells in urine, and kidney dysfunction are strong predictors of leukemic infiltration of the kidney.

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