Clin Kidney J (2013) 6: 448–449

CKJ

From the Clinic

Infiltrative acute myeloid leukaemia as a cause of acute kidney injury

A 75-year-old male was admitted for investigation of acute kidney injury. His medical history was significant for hypothyroidism and transfusion-dependent myelodysplastic syndrome. A bone marrow biopsy 1 month prior to admission had categorized him as having refractory anaemia with excess blasts (RAEB) Stage-II. His medications included thyroxine, cholecalciferol and the cytotoxic agent azacitadine.

Over the preceding 6 months, he had noticed progressive lethargy, loss of weight and functional decline. Examination was unremarkable. Biochemistry demonstrated a serum creatinine level of 350 µmol/L, which had markedly worsened over 2 months. This was associated with 1.06 g/day proteinuria but no haematuria. Haematology showed normocytic anaemia (Hb 70 g/L), thrombocytopaenia (Plt 48×10^9/L) and marked leucocytosis (WCC 38.8×10^9/L). Dysplastic changes and occasional blasts were noted on the blood film (Figure 1). Serum protein electrophoresis, uric acid, autoimmune studies, hepatitis and HIV serology were unremarkable. Ultrasound demonstrated normal-sized kidneys of normal echogenicity and no evidence of obstruction.

A renal biopsy was performed, which showed renal cortex with heavy infiltration of atypical cells within the interstitium and lumina of small vessels. These cells had irregular nuclear membranes, prominent nucleoli and granular cytoplasm, and showed positive immunoperoxidase staining with myeloperoxidase, confirming the diagnosis of acute myeloid leukaemia (AML) with renal infiltration (Figures 2 and 3).

A repeat bone marrow biopsy was subsequently performed which showed transformation into AML. The cause of the progressive kidney dysfunction was thus attributed to renal infiltration from AML. In view of the patient's poor prognosis and functional state, community-based palliative care was instituted. He passed away several weeks later.

Malignant infiltration of the kidneys as a cause of renal failure has been well described [1–3]. The commonest causes are low-grade non-Hodgkin's lymphoma and acute lymphocytic leukaemia [4]. AKI due to diffuse kidney infiltration from AML has been reported in the setting of other precipitants [5]. Our case is unique, in that it demonstrates acute kidney injury resulting directly from malignant infiltration of renal parenchyma from AML.

Conflict of interest statement. None declared.

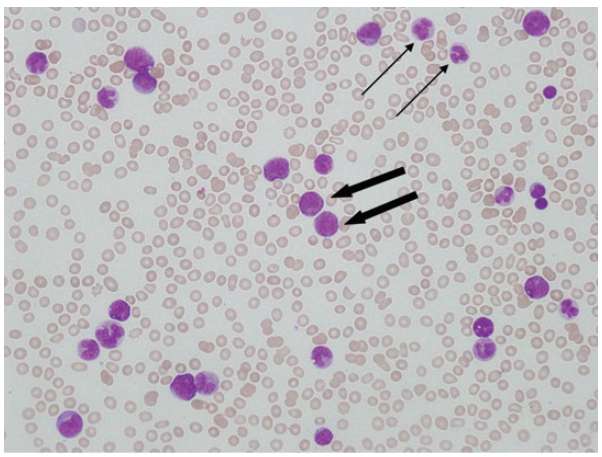

Fig. 1. May–Grunwald–Giemsa staining of the peripheral blood film shows pleomorphic population of blasts with an open chromatin and high nuclear:cytoplasmic ratio (thick arrows). Dysplastic neutrophils are also noted with abnormal segmentation and hypogranular forms (thin arrows).

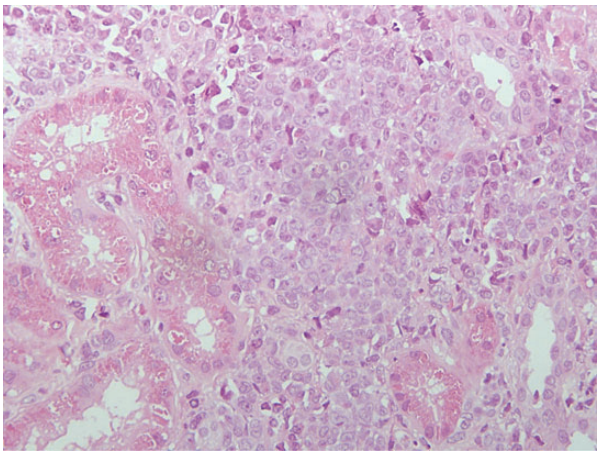

Fig. 2. Highly cellular infiltration in the renal interstitium, atypical myeloid blasts with irregular nuclear membranes, prominent nucleoli and granular basophilic cytoplasm (H&E ×200).

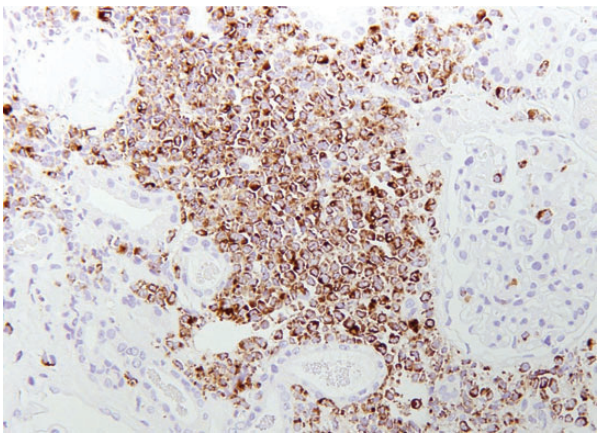

Fig. 3. Positive immunohistochemical staining for myeloperoxidase (MPO IHC ×100).

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doi: 10.1093/ckj/sft066