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Back pain and renal failure

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A 40-year-old plumber was admitted to the emergency room of our hospital in October, 2003, with bilateral backache for 1 week. He had no other complaints. He had no previous medical history and was not on any treatment. On examination, his temperature (36·5°C), heart rate (80 beats per min), and blood pressure (140/70 mm Hg) were normal. There was no rash and no lympha-denopathy. Lungs were clear and heart sounds were normal. There was no oedema and he was passing urine normally. Abdomen was unremarkable except for tenderness of both costovertebral angles. Laboratory tests showed normal blood-cell counts but a severe renal failure: serum creatinine 390 µmol/L (normal: 35-106), with raised uric acid (2268 µmol/L: 200-500). Urinalysis showed microscopic haematuria and moderate proteinuria (600 mg/L). Neither crystals nor amorphous material was detected in the urinary sediment. Abdominal ultrasound showed enlarged kidneys (14 cm in length). A renal biopsy was done and haemodialysis started.

The biopsy specimen showed a massive interstitial tumour infiltrate (figure, A) composed of a monomorphous population of small to medium-sized cells with round to oval nuclei, very fine chromatin, and scanty cytoplasm (figure, B). The widened interstitium compressed the tubular luminae but neither urate crystals nor tubular necrosis were seen; there were some mitotic figures. Immunohistochemical studies identified these cells as precursor T lymphoblasts because they were positive for several T-cell antigens, including CD2, CD3, and CD5, as well as for terminal deoxynucleotidyltransferase (TdT) (figure, C and D). The diagnosis of precursor T-lymphoblastic lymphoma was then made. Cytological examination of a bone-marrow aspirate showed massive lymphoblastic involvement, indicative of acute lymphoblastic leukaemia. A positron emission transaxial tomography (PET) scan showed increased metabolic activity in all bone-marrow areas, liver, spleen, and kidneys (figure, E). High doses of corticosteroids (intravenous methylprednisolone, 80 mg twice a day for 1 week) led up to a rapid return to normal of renal function. Tumour lysis secondary to corticosteroids could have been responsible for worsening of hyperuricaemia. Rasburicase (0·2 mg/kg/day for 1 week) was given. Once the final diagnosis was established, standard chemotherapy was started. As of October, 2004, he is on maintenance treatment, feels well, is in complete remission, and his serum creatinine is normal. Severe acute renal failure is a rare presentation of malignant haemopathies. There are two pathophysiological mechanisms. Hyperuricaemia due to increased nucleic-acid catabolism is the most frequent. Uric-acid nephropathy is characterised by tubular necrosis due to uric-acid crystallisation in the tubular lumen. In this setting, urate crystals are easily detectable by light microscopy, the size of the kidneys is within normal range, and patients are usually oliguric.[1] Rasburicase is an effective treatment. As shown in this patient, the other cause is neoplastic-cell infiltration. Whereas renal infiltration by leukaemias and lymphomas is common (in an autopsy series 30-80% of all cases),[2] it rarely leads to clinically significant renal failure. When the renal infiltration is responsible, it is mediated through a tubular compression more than by a tubular necrosis, causing increased kidney volume, moderate proteinuria, and sometimes microscopic haematuria.[35] Increased kidney volume explains the lumbar pain reported by our patient. In the case of massive renal infiltration by neoplastic cells, rapid tumour regression induced by corticosteroids can lead to the return to normal renal function. Acute renal failure is a rare presentation of malignant haemopathies. Kidney biopsy is useful for diagnosis.

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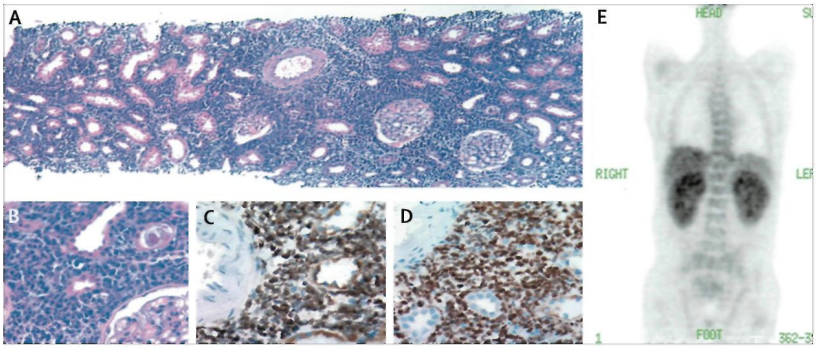
Figure: *A. Low-power view of renal biopsy specimen showing a dense interstitial tumour infiltrate (haematoxylin and eosin). B Higher magnification showing lymphoblasts with round, oval, or irregular nuclei and scanty cytoplasm (haematoxylin and eosin). C. Cytoplasmic positivity of lymphoblasts for CD3 (immunoperoxidase. D. Nuclear positivity of lymphoblasts for TdT (immunoperoxidase). (E) PET-scan.*



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