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doi: 10.1093/ndtplus/sfn204

Advance Access publication 16 January 2009

An unconscious 76-year-old woman with renal failure and hyperechoic kidney lesions

Sir,

Overall rates of infection with *Mycobacterium tuberculosis* have declined over recent years in most western countries. However, tuberculosis remains a problem in certain high-risk groups [1,2]. Postprimary tuberculosis is even rarer, especially causing chronic renal failure [3]. Thus, attention to this (once) common disease is most likely low in the present generations of physicians in industrialized countries.

The present case shows that a careful workup of renal insufficiency, particularly considering anamnesis and clinic, can still lead to an unexpected diagnosis. This case report should encourage clinicians to consider tuberculosis in differential diagnosis, especially in the patient at risk.

A 76-year-old white, cachectic female was admitted to the university hospital as an emergency case. On presentation at the emergency department in June 2008, she had a Glasgow coma scale of 5 and a body temperature of 36°C. The respiratory rate was increased despite normal O_2 saturation. Tachycardia and arterial hypertension were detected. Physical examination showed a pale, dehydrated and cachectic woman (BMI 13.3) with normal auscultation of the lung, normal physical abdominal findings, absence of skin abnormalities and meningism, and without focal neurological signs. A CT scan excluding bleeding and raised intracranial pressure was performed. The patient was transferred to a neurological intensive care unit where she underwent lumbar puncture. Cerebrospinal fluid cell count, protein concentration and gram staining were normal. Blood analysis revealed Na^+ 138 mmol/L, K^+ 5.9 mmol/L, Ca^{2+} 2.5 mmol/L (corrected for serum albumin), serum–creatinine 203 μmol/L, blood urea nitrogen 14.6 mmol/L, haemoglobin 75 g/L, prolactin 1000 pmol and CRP 18 mg/L. Although her medication included carbamazepine, it was not traceable in blood analysis on the day of presentation.

Sixteen months previously, our patient had been admitted to the department of neurosurgery in a community hospital due to persistent headache and vomiting. Hypertrophic pachymeningitis was confirmed histologically. After the neurosurgical procedure, the patient suffered from symptomatic epilepsy. For this reason, she was admitted twice at different hospitals (March and April 2008) and treatment with carbamazepine (400 mg bid) was commenced. Serum–creatinine was elevated (177 μmol/L) on both occasions despite a normal creatinine 1 year before. The patient's past medical history also revealed the development of arterial hypertension, anaemia and severe weight loss within the previous year.

Treatment in the department of neurology consisted of rehydration, blood transfusion and carbamazepine saturation. Due to increased serum creatinine, the consultant nephrologist was involved. At presentation, diuresis was 950 mL/day and GFR was 13 mL/min. Urinalysis was negative except for traces of protein. Differentiation of the mild proteinuria revealed moderate elevated albumin, IgG and alpha-1-microglobuline. The urine culture was negative. B-mode kidney ultrasound showed a left kidney of 10.8 × 4.3 cm and a right kidney of 8.5 × 2.2 cm. Both kidneys presented with multiple, disseminated nodulous (2–4 mm) hyperechoic lesions of the parenchyma. There were no hints towards kidney stones or postrenal obstruction. Perfusion was adequate. Mycobacteria were not detected in the urine (PCR and culture). Abnormal ultrasonography qualified the patient for a kidney core needle biopsy. Histology showed the presence of necrosis with focal epithelioid cells as a central proportion of granuloma (caseous lesions, indicative for tuberculosis) whereas interstitial fibrosis was absent (Figure 1). Chest CT scan was not probative for active tuberculosis, yet typical older lesions were seen [4]. Anti-tuberculous therapy was commenced immediately [quadruple regimen with ethambutol 500 mg (tid), isoniazid/vitamin B6 200 mg/day, rifampicin 300 mg/day and pyrazinamide 1500 mg/day)].

The patient stabilized and was discharged into a nursing home, with daily medication consisting of carbamazepine 600 mg (bid), amlodipin, lisinopril, hydrochlorthiazide, torasemide, calcitriol and darbepoetin-alpha 30 μg sc (weekly).

Four months after the initiation of tuberculostatic therapy, she has gained 4 kg of body weight, her well-being improved constantly and kidney function has recovered to 24.2 mL/min (MDRD GFR).

Extrapulmonary tuberculosis affecting the genitourinary system is usually caused by haematogenous dissemination of *M. tuberculosis* from the lungs (reasonably responsible for the renal tuberculosis of our patient). However, <50% of these patients give radiographical evidence for pulmonary tuberculosis, whereas active disease is present in only 5% of such patients [5]. Presumably 10% of all patients with tuberculosis develop reactivation. Their risk is highest within the first 2 years or during periods of immunosuppression [2]. Indeed our patient showed pulmonary radiographical findings consistent with older tuberculous lesions. Notably, even in high-risk patients with a history of tuberculosis who received a renal transplant postprimary renal tuberculosis is rare [6]. In a recent report on 80 patients with

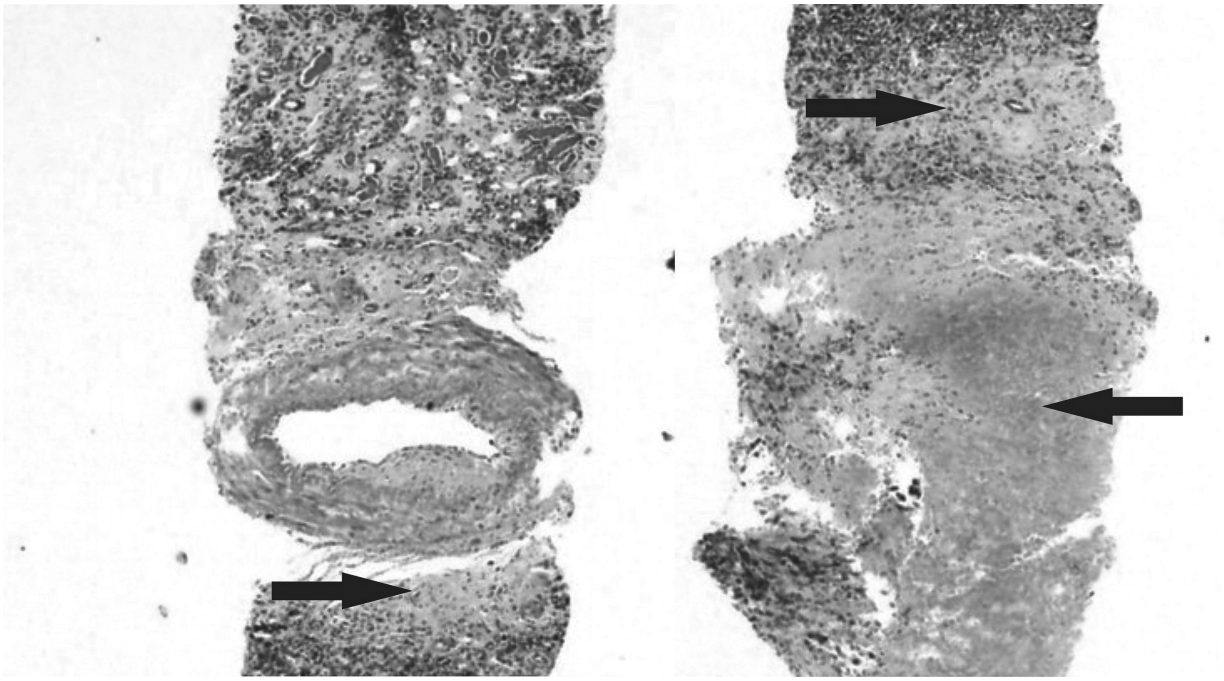

Fig. 1. PAS stain of the renal core needle biopsy (magnification ×4). Histology showed extended caseous lesions (necrotizing granulomas with focal epitheloid cells, arrows) surrounded by leukocytes.

urogenital tuberculosis, only 8.8% suffered from bilateral parenchymatous renal lesions (renal miliary tuberculosis) [7]. Clinical symptoms of renal tuberculosis are microscopic haematuria, flank pain (resistant to the usual antibiotics) and (sterile) pyuria. All of these symptoms were absent in our patient. Constitutional symptoms, such as fever, weight loss and night sweats, are reported to be less common [5].

The laboratory diagnosis of renal tuberculosis is difficult, because identification of the organisms in the urine is hard and clinical and radiological presentations vary. However, ultrasound examination of the urinary tract reveals features of tuberculosis in over 50% of the cases [8]. A recent work published 152 cases of correctly diagnosed renal tuberculosis classified into six types. This classification takes the variability of the ultrasonographic appearance of renal tuberculosis into account, whereas nephrectasia, distension of renal pelvis and calyces, empyema or calcification were the most prominent visible signs [8]. In the case presented here, ultrasonographic findings and kidney biopsy results are primarily compatible with renal tuberculosis.

Renal insufficiency accompanied by wasting should lead physicians to take tuberculosis into account.

Conflict of interest statement. None declared.

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doi: 10.1093/ndtplus/sfn207

Advance Access publication 29 January 2009

Disseminated cryptococcosis, an unusual cause of gross proteinuria in an HIV-infected patient

Sir,

A 52-year-old HIV-positive, ARV-naive African man was admitted with cryptococcal meningitis. Pertinent laboratory investigations included a serum creatinine of 170 mcmol/l, a urinary albumin creatinine ratio of 50 mg/mmol and urinary