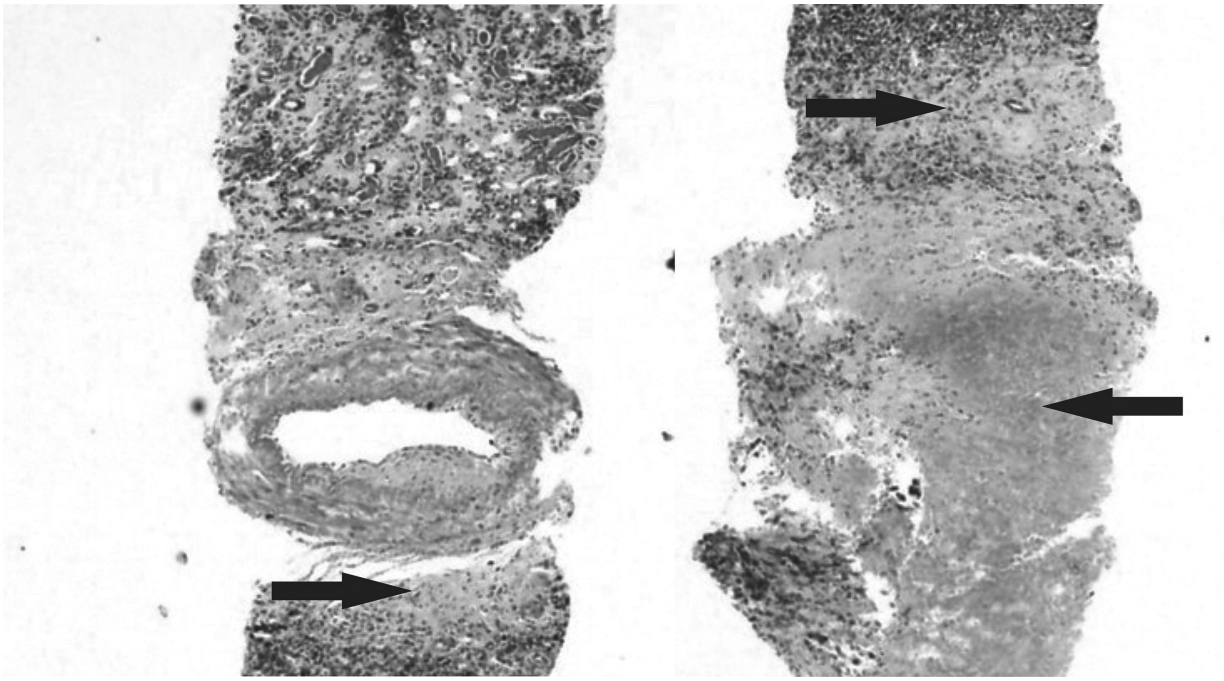

Fig. 1. PAS stain of the renal core needle biopsy (magnification ×4). Histology showed extended caseous lesions (necrotizing granulomas with focal epitheloid cells, arrows) surrounded by leukocytes.

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.

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1. Dye C, Watt CJ, Bleed DM *et al*. Evolution of tuberculosis control and prospects for reducing tuberculosis incidence, prevalence, and deaths globally. *JAMA* 2005; 293: 2767–2775
2. Frieden TR, Sterling TR, Munsiff SS *et al*. Tuberculosis. *Lancet* 2003; 362: 887–899
3. Figueiredo AA, Lucon AM, Junior RF *et al*. Epidemiology of urogenital tuberculosis worldwide. *Int J Urol* 2008; 15: 827–832
4. Linh NN, Marks GB, Crawford AB. Radiographic predictors of subsequent reactivation of tuberculosis. *Int J Tuberc Lung Dis* 2007; 11: 1136–1142
5. Eastwood JB, Corbishley CM, Grange JM. Tuberculosis and the kidney. *J Am Soc Nephrol* 2001; 12: 1307–1314
6. Klote MM, Agodoa LY, Abbott K. Mycobacterium tuberculosis infection incidence in hospitalized renal transplant patients in the United States,1998–2000. *Am J Transplant* 2004; 4: 1523–1528
7. Figueiredo AA, Lucon AM, Gomes CM *et al*. Urogenital tuberculosis: patient classification in seven different groups according to clinical and radiological presentation. *Int Braz J Urol* 2008; 34: 422–432
8. Rui X, Li XD, Cai S *et al*. Ultrasonographic diagnosis and typing of renal tuberculosis. *Int J Urol* 2008; 15: 135–139

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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

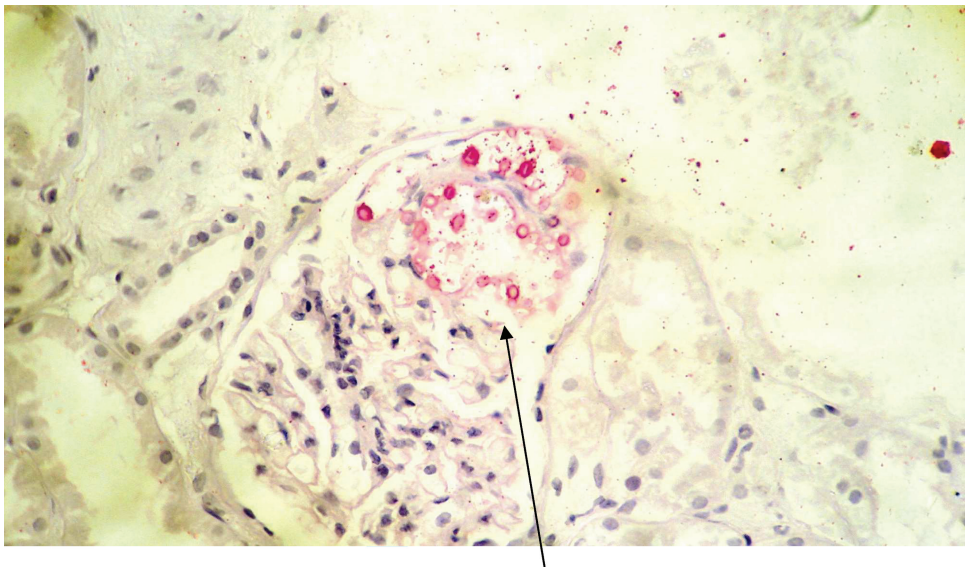

Fig. 1. Glomerulus showing yeast forms of cryptococcocus in one of the capillary loops. Note the relatively normal background (mucicarmine ×20).

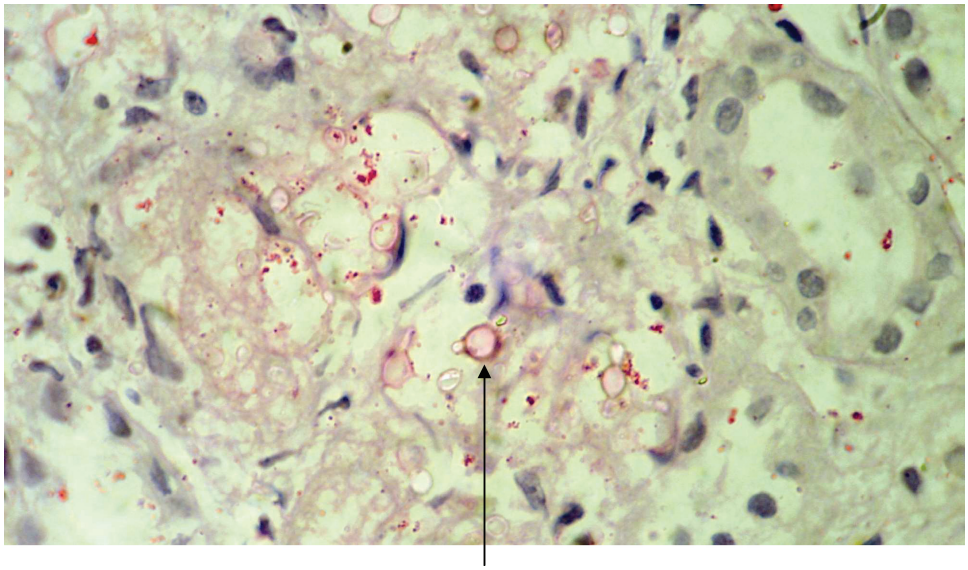

Fig. 2. Mucicarmine stain highlighting the capsule of the budding yeast forms tubular lumina (×40).

sodium of 268 mmol/24 h. His CD_4 count was 15 cells/ml. Both hepatitis B and C serology were negative.

Renal ultrasound showed normal-sized kidneys with increased renal parenchymal echogenicity suggestive of HIV-associated nephropathy. A renal biopsy, which yielded eighteen glomeruli, showed cryptococcal yeasts in some of the glomerular capillaries, tubules and within the interstitium with minimal inflammatory response. There was no evidence of collapsing glomerulopathy. The final diagnosis was cryptococcal meningitis with fungaemia and nephritis.

The patient was initially started on antifungal therapy and both his clinical state and serum creatinine improved. He was planned for highly active antiretroviral therapy as an outpatient, however, was lost to follow up on discharge.

Discussion of diagnosis

Renal involvement in disseminated cryptococcosis has been described in autopsy studies in the pre-HIV era to occur at rates ranging from 26 to 45% [1]. Following the advent of the HIV pandemic, there have been several case reports and series reporting the occurrence of cryptococcal nephritis [2,3]. Direct renal glomerular, tubular or interstitial invasion are potential causes of renal dysfunction in patients with disseminated cryptococcosis.

In HIV-infected patients with cryptococcal nephritis, macroalbuminuria is rare. In a case series of 26 HIV patients with proteinuria >1.5 g/24 h, only 2 had histological evidence of cryptococcal nephritis [4]. Varma *et al*. [5]

also reported in their series of 25 HIV-positive patients presenting with proteinuria, 2 patients with cryptococcal renal infiltration. The patient described in the case report had evidence of macroalbuminuria and tubular dysfunction as evidenced by significant urinary sodium loss on the 24-h urine specimen. This may be attributable to both glomerular and tubulo-interstitial cryptococcal invasion. Unlike the index patient who had a minimal inflammatory response, attendant inflammation evoked by the organisms may also contribute to the proteinuria in patients with cryptococcal nephritis.

Conclusion

This case report highlights the varied aetiology of proteinuria in the HIV-infected patient and brings to the fore the fact that not all gross proteinuria in an HIV-infected patient is secondary to HIV-associated nephropathy.

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1. Lewis J, Rabinovich S. The wide spectrum of cryptococcal infections. *Am J Med* 1972; 53: 315–322
2. Chuang YM, Ho YC, Chang HT *et al*. Disseminated cryptococcosis in HIV-uninfected patients. *Eur J Clin Microbiol Infect Dis* 2008; 27: 307–310
3. Van Der Reijden H, Schipper M, Danner S *et al*. Glomerular lesions and opportunistic infections of the kidney in AIDS: an autopsy study of 47 cases. *Adv Exp Med Biol* 1989; 252: 181–188
4. Praditpornsilpa K, Napathorn S, Yenrudi S *et al*. Renal Pathology and HIV infection in Thailand. *Am J Kidney Dis* 1999; 33: 282–286
5. Varma PP, Prasher PK, Deshpande GU *et al*. Spectrum of renal lesions in HIV patients. *J Assoc Physicians India* 2000; 48: 1151–1154

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Advance Access publication 20 January 2009

Diabetic glomerular disease: pitfalls in diagnosis

Sir,

Diabetic nephropathy is a common but not an invariable complication of type 1 and 2 diabetes mellitus. However, diabetes mellitus itself is very common, and therefore, other causes of renal impairment co-exist in this population. Even with a renal biopsy, the correct diagnosis may not always be immediately evident as other pathologies can also mimic diabetic nephropathy. We present a case where diabetes mellitus was incorrectly assumed to be the cause of the patient's renal impairment.

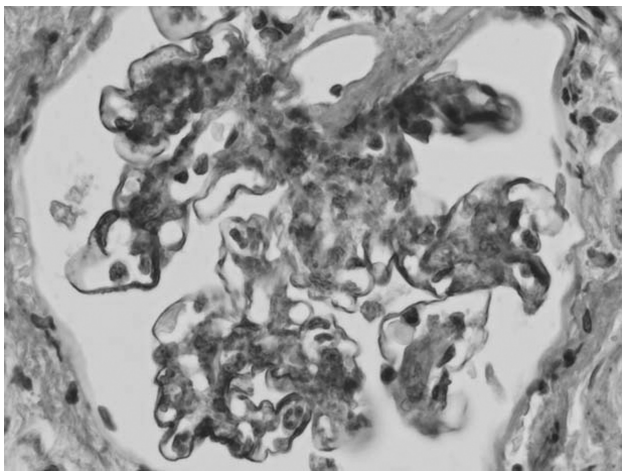

Fig. 1. Mesangial deposits of IgA × 400.

The case is of a 54-year-old man diagnosed with type 1 diabetes mellitus 20 years earlier. He had proliferative retinopathy and autonomic neuropathy but no proteinuria and no hypertension. He developed sudden onset nephrotic syndrome with eGFR >60 ml/min/1.73 m^2. On renal biopsy, the light microscopic appearance was of a diffuse and nodular diabetic glomerulosclerosis with arteriolar hyalinosis. However, the degree of mesangial and endocapillary proliferation was atypical. EM examination disclosed large mesangial, para-mesangial and small extramesangial sub-endothelial electron-dense deposit but no specific features of light chain deposition disease. Over the next 6 months, his eGFR deteriorated to 9 ml/min/1.73 m^2. The repeat biopsy revealed a new focal necrotising and crescentic glomerulonephritis. On immunohistochemistry, there were IgA and C3 deposits in the glomerular mesangium and extra-mesangial capillary walls (Figure 1). The final histological diagnosis was that of diffuse (endocapillary) proliferative glomerulonephritis–IgA nephropathy superimposed on diffuse and nodular diabetic glomerulosclerosis that later evolved into a focal and necrotising crescentic glomerulonephritis. The patient's renal function deteriorated further and he started dialysis.

The sudden onset of nephrotic syndrome in our case should suggest an alternative diagnosis to diabetic nephropathy, as this alone would usually be preceded by lesser degrees of proteinuria.

Despite diabetic nephropathy being the leading cause of end-stage renal failure in the Western world, we must remain suspicious for alternative non-diabetic causes of renal impairment that may co-exist with diabetic nephropathy. Furthermore, other diseases, in particular light chain deposition disease, can mimic the typical nodular lesions of diabetic glomerulosclerosis. Our case emphasizes the importance of immunochemical and ultrastructural evaluation of biopsy tissue submitted from all such patients [1].

Conflict of interest statement. None declared.