Rapidly progressive glomerulonephritis in a patient with brucellosis

Sir, Subclinical renal involvement in the course of brucellosis is a common scenario in the area of brucella endemi city [1,2]. However, few reports of brucellosis with overt renal failure exist in the literature. Brucella nephropathy may be secondary to glomerulonephritis (GN), interstitial nephritis, renal vasculitis and granuloma or abscess formation [1–4]. Membranous glomerulopathy, mesangial proliferative GN, IgA nephropathy and mesangiocapillary GN have been reported [1,3,5]. Due to the paucity of reports, the nature of brucella nephropathy has remained largely unidentified. We describe a case of brucellosis associated with rapidly progressive renal failure.

A 28-year-old man was admitted to hospital due to rapidly progressive renal failure. He had vomiting, anorexia and weakness and also reported frequent and nocturia for 5 months. A history of surgical correction of ventricular septal defect was present. He had also had serologically proven brucellosis 4 months previously, for which he had received specific antibiotics and apparently was not compliant with them. Examination revealed an ill man with BP of 90/60 mmHg and BT of 37.5°C. A grade V/VI holosystolic murmur was prominent over the pulmonary area. Otherwise, the examination was unremarkable. Remarkable laboratory findings were: haemoglobin 8.8 g/dl, haematocrit 25.6%, CRP +, first-hour ESR 55 mm, serum creatinine 4 mg/dl, urea 101 mg/dl, uric acid 8.3 mg/dl, calcium 8.2 mg%, phosphorus 6 mg/dl. C3 complement level was low at 57 mg/dl (normal 70–170 mg/dl). Hepatitis and HIV serology, P-ANCA, C-ANCA, ANA and antibodies to dsDNA and glomerular basement membrane were all negative. Brucella serum agglutination test was positive at a titer of 1/2560. Urine sediment examination showed many red blood cells casts. There was a proteinuria of 1 g/day. Renal ultrasonography revealed increased parenchymal echogenicity. Echocardiography demonstrated moderate pulmonary stenosis. No valvular vegetations were detected.

Light microscopy of the renal biopsy specimens revealed mesangial cell proliferation, matrix expansion, glomerular basement membrane thickening (double-contour appearance) and accentuation of lobular architecture in some glomeruli (Figure 1). Immunofluorescence study showed intense mesangial staining for C3 and C1q in a granular pattern that was negative for any immunoglobulins and Kappa and Lambda light chains. The diagnosis of mesangiocapillary GN was made.

The patient was placed on rifampin (200 mg/day) and doxycycline (900 mg/day) as well as intravenous methylprednisolone pulses (500 mg/day for three consecutive days) followed by oral prednisolone. One week later, his general condition dramatically improved. At day 12, serum creatinine level and urinalysis returned to normal. Rapid response to steroid and specific antibiotic therapies led us to consider the diagnosis of brucella GN for this patient.

Brucella may involve renal glomeruli, interstitium and/or renal vasculature [1,4,5]. Brucella nephropathy seems to be an underdiagnosed entity. Hence, a high index of suspicion for it is particularly necessary in the area where the brucella is endemic.

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