Brucella Endocarditis Causing Acute Renal Failure

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Dear Sir,

Brucella nephritis has traditionally been classified into three main clinical groups. The first is a transient renal affliction usually occurring during the acute infection [1]. In the second group there is a chronic process that simulates chronic pyelonephritis [2, 3]. Rarely, renal involvement is associated with Brucella endocarditis, which used to carry very high mortality [4].

Case Report

A 45-year-old male shepherd was admitted to the hospital because of heart failure. He was well until 3 weeks prior to admission when he experienced fever, generalised muscle aches and headache. A week later he noted smoky discolouration of urine and became increasingly short of breath. Physical examination on admission revealed a middle-aged male who was orthopneic, his blood pressure was 130/80, the pulse was 90/min, regular, and he had normal temperature. His fundus evaluation was normal. Subungual hemorrhages were noted as well as purpuric rash over the lower extremities. The internal jugular venous pressure was moderately raised. Air entry was diminished on the right base with fine basilar rales. Grade III/VI early diastolic murmur was prominent over the aortic area. The spleen was felt 5 cm below the costal margin and the liver measured 15 cm in the vertical span and was mildly tender. All peripheral pulses were equally felt and minimal pedal edema was present. His neurological evaluation was unremarkable as well as the genital and rectal examination.

Urine examination showed high protein, many red blood cells and granular casts. Chest roentgenogram revealed cardiomegaly, prominent pulmonary veins and right pleural effusion; ECG was unremarkable; Hb was 13.3 g%, WBC was 8,100 with 85% PMNs, 12% lymphocytes and 2% monocytes; platelets were 110,000. ESR was 56 mm in the 1st h. Serum electrolytes were Na⁺ 133, K⁺ 4.8, Cl⁻ 102 and HCO₃⁻ 15.8 mEq/l. Blood urea and serum creatinine were 335 and 15.8 mg%, respectively. Serum values were: calcium 8.4 mg%, phosphorous 5.0, albumin 2.4, protein 6.1, uric acid 18.2, alkaline phosphatase 174 and bilirubin 1.7 mg%; SGOT 57, SGPT 61 and CPK 178; Brucella agglutinin titre was 1/640. C₃ and C₄ were 25 and 8.9 mg%, respectively; antinuclear antibodies and rheumatoid factor were negative. Wright stain of urine sediment was negative for eosinophils. The echocardiogram revealed a thickened aortic valve with a large ball of vegetation on the aortic cusp. Renal ultrasound showed normal-sized kidneys. Blood cultures were taken and patient was begun on gentamicin and penicillin G. On the 6th hospital day he suddenly deteriorated and underwent successful aortic valve replacement.

Brucella sp. was isolated from the vegetations on the damaged aortic cusps, and few days later the same organism was identified from two sets of blood cultures drawn on admission (12 days earlier). He was treated with a 6-week course of co-trimoxazole and rifampicin and did very well.

Four months later, his serum creatinine was 1.5 mg% and his 24-hour urinary protein became 300 mg; urine microscopy however, continued to show 10–12 red blood cells per high-power field and few granular casts. A percutaneous renal biopsy done at that time revealed 10 glomeruli with diffuse mesangial proliferation. The interstitium was diffusely infiltrated by chronic mononuclear cells with focal tubular atrophy. The immunofluorescence microscopy was only positive for IgG in a diffuse granular pattern.

Comment

The renal biopsy in our patient consisted of a tubulo-interstitial nephritis like the picture which has previously been described in *Brucella* nephritis [2]. More interesting were the glomerular changes in the form of mesangial cell proliferation and the immunofluorescent microscopy *Brucella* Endocarditis Causing Acute Renal Failure

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which demonstrated IgG in peripheral capillary loops and mesangium.

These changes are suggestive of an underlying immunological process and support recent case reports of renal brucellosis in which both immunoglobulins (IgG and IgA) and complement (C3) were shown in glomeruli in the absence of endocarditis [5, 6]. Whether the renal affliction in our patient was caused by circulating immune complexes, similar to what has been described in infective endocarditis caused by a variety of organisms, or to in situ antigen-antibody reaction is difficult to ascertain. More sophisticated immunological studies will be needed to elucidate the nature of the underlying immunological mechanism.

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