CASE REPORT

Prostatic tuberculosis in an HIV infected male

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The patient was a 36 year old white man with recently diagnosed AIDS. At HIV diagnosis, his HIV viral load was > 500 000 copies/ml and his CD4 cells were 40 cells/μl. Six weeks after his AIDS diagnosis, he presented with fevers, night sweats, chills, and dysuria. An *Escherichia coli* urinary tract infection was diagnosed and he was treated with levofloxacin for 14 days. Symptoms continued, he was found to have *E coli* bacteraemia, and he was referred for inpatient evaluation.

He was exclusively heterosexual, denied injecting drug use, and had over 400 sexual partners in the past year. He travelled extensively in the United States, and had lived in Key West, Florida, and Los Angeles.

On admission to hospital, he was afebrile, had bilateral temporal wasting, and leucoplakia but no adenopathy. The respiratory, cardiovascular, abdominal, and central nervous systems were unremarkable. A rectal examination demonstrated multiple prostatic areas that were asymmetric and tender, with induration at 3 o’clock. His white blood cell count was 1900 cells × 10⁶/l (absolute neutrophil count 1693), haemoglobin was 10.1 g/dl, and platelets were 82 000 × 10⁶/l. Electrolytes and coagulation studies were normal. Liver function tests revealed an AST of 278 (normal 0–37), ALT 123 (normal 0–40), and an alkaline phosphatase of 1200. Urinalysis had 5–10 white blood cells/HPF, and moderate bacteria, but culture was initially negative. The chest radiograph was normal. Computed tomography of the lower abdomen and pelvis revealed hydrouretonephrosis in both kidneys. There was an enlarged 5.0 cm prostate gland with multiple 1–1.5 cm intraprostatic collections with enhancing rims (fig 1) and enlarged retroperitoneal and coeliac nodes. A transurethral prostatectomy was performed and histology of the prostate revealed caseating granulomas (fig 2) with numerous acid fast bacilli on Ziehl-Nielsen stain. Subsequently, urine culture from the prostate was positive for *Mycobacterium tuberculosis* on pyelogram is often diagnostic of disease. Culture of urine and blood are necessary if urine cultures are negative and there is a high suspicion of disease. The clinical findings in prostatic tuberculosis are often nonspecific. The most common findings are scrotal lesions, lower urinary tract symptoms, and painless haematuria. The similar findings on digital rectal examination of indurated masses and the transurethral ultrasound findings of diffuse hypoechoic lesions within the peripheral zone of the prostate often makes the distinction between prostatic cancer and tuberculosis difficult.

Although sterile pyuria is a classic feature of genitourinary tuberculosis, positive cultures for pyogenic organisms may lead to misdiagnosis, as happened in this case. Focal calcification on pyelogram is often diagnostic of disease. Culture of three morning urines establishes the diagnosis in approximately 85% of cases, though cytological examination is necessary if urine cultures are negative and there is a high suspicion of disease.

Once diagnosed, genitourinary tuberculosis is treated with regimens recommended for extrapulmonary tuberculosis and urinary concentrations of isoniazid, rifampin, pyrazinamide, and streptomycin are high. Corticosteroid therapy has been recommended if obstruction develops strictures or obstruction of the renal tract and ureteral reimplantation if the obstruction does not resolve. Recent literature suggests that surgical intervention is required rarely.
While we have been unable to identify any positive sexual partners, the location and immune status of the patient does raise the potential for sexual transmission of *M. tuberculosis*.

This case demonstrates the need for vigilance and continued testing in patients who are unresponsive to seemingly appropriate antibiotic therapy with prostatic abscess.

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