Dissemination of tuberculosis after biopsy of primary tubercular prostate: a case report

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Only 15–20% of extrapulmonary tuberculosis (TB) has been attributed to TB of the urogenital system and often results from haematogenous spread from an active site of infection. Isolated involvement of prostate by TB is relatively less common. The incidence of primary prostatic TB is unknown and in its truest sense is a very rare entity. Here, we report the case of a patient with primary prostatic TB who was misdiagnosed as nonspecific granulomatous inflammation on transrectal ultrasound-guided biopsy of the prostate, who later presented to our centre after 4 months with disseminated TB. The rarity of the case prompted us to report this case.

Keywords:
disseminated tuberculosis, nonspecific granulomatous inflammation of prostate, primary prostate tuberculosis

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Introduction

The occurrence of genitourinary tuberculosis (TB) is almost always secondary and diagnosed as a part of disseminated TB. It is fairly common and accounts for 15–20% of TB cases [1]. However, the actual prevalence of prostate TB may be much higher than reported. Most patients present to urologists with symptoms of prostatitis, and urologists seek prostatic biopsy histopathology to rule out malignancy and many a time undermine reports of benign granulomatous inflammation in view of the high prevalence of nonspecific granulomatous inflammation in post-transurethral prostatectomy prostate [2,3]. Although prostate TB without evidence of other primary focus is rare and mostly reported in immunocompromised individuals, there have been multiple case reports that have illustrated prostate as the primary foci [1,4,5]. Therefore, a careful consideration of TB in all prostate biopsy with granulomatous inflammation may help in the early detection of such cases, thus preventing future complications. Here, we report the case of a patient who was misdiagnosed as having nonspecific granulomatous inflammation on transrectal ultrasound (TRUS)-guided biopsy of the prostate, who later presented to our centre after 4 months with disseminated TB. The rarity of the case prompted us to report this case.

Case report

A 67-year-old nonsmoker and diabetic man with good glycaemic control presented to our tertiary respiratory care unit with 3-week history of progressive shortness of breath, low-grade fever on and off, cough with expectoration with loss of weight and loss of appetite. There was no history of antitubercular therapy intake, or recent exposure to a TB patient. At presentation, he was underweight (BMI = 16.65) and in respiratory distress, with oxygen saturation at room air being 86%. He was oriented to time, person and place. He had tachypnoea and tachycardia with normal blood pressure and body temperature. The remaining physical examinations were unremarkable except for bilateral crackles on auscultation. His chest radiography revealed diffuse nodular opacity bilaterally (Fig. 1). Initial laboratory parameters revealed elevated erythrocyte sedimentation rate, transaminases and alkaline phosphatase, and blood picture was within normal limits. Ultrasonography of the whole abdomen was normal except for prostatomegaly (43 ml); serum prostate specific antigen (PSA) was within normal limits (1.2 ng/ml). Sputum acid-fast bacilli (AFB) and gram and fungal stains were negative. Pyogenic cultures of urine, blood and sputum showed no growth. Urinary antibodies against Streptococcus and Legionella were negative. Approval have been taken verbally from the patients.

Computed tomography (CT) of the chest was carried out, which showed randomly distributed miliary nodules involving all lobes bilaterally (Fig. 2). Mantoux (12 × 10 mm) and interferon-γ release assay tests were found to be positive. His serum angiotensin-converting enzyme (68.5 μg/l) was also high. Fibre-optic bronchoscopy with BAL, endobronchial biopsy and transbronchial

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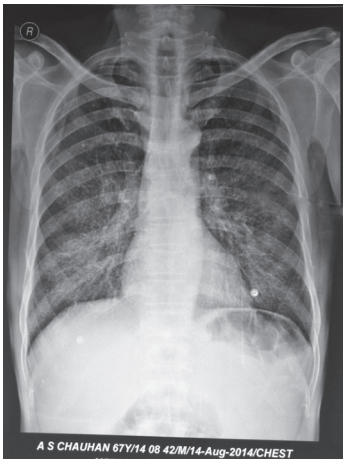
lung biopsy were performed and samples were sent for investigations. Broncho-alveolar lavage (BAL) immunoassay for *pneumocystis carinii* pneumonia was negative. BAL stains were negative for AFB, fungus and pyogenic bacteria. BAL cytospin for malignant cells was negative. BAL Gene Xpert detected *Mycobacterium* TB. Histopathological examination (HPE) of transbronchial lung biopsy showed thickened interstitium with mononuclear infiltration, type-2 pneumocyte hyperplasia and epithelioid cell granuloma without significant necrosis (Fig. 3). HPE of endobronchial biopsy showed nonspecific inflammation.

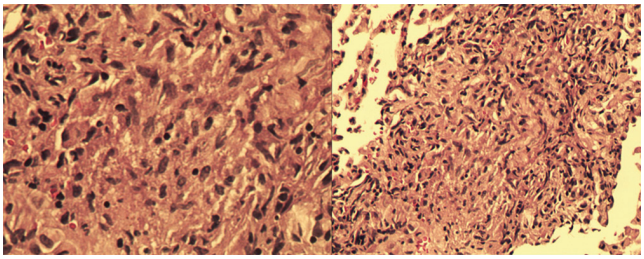
PET-CT was carried out, which showed increased ^{18}F-FDG uptake in bilateral lungs (SUV_{max} 4.7) and prostate (SUV_{max} 7.5) and mild diffuse ^{18}F-FDG uptake in the bone marrow of axial and proximal appendicular skeleton (Fig. 4). In view of ^{18}F-FDG uptake in bone, bone marrow biopsy was carried out, which showed granulomatous infiltration with AFB positive on ZN stain suggestive of dissemination of TB (Fig. 5), and the patient was started on antitubercular therapy (isoniazid, rifampicin, pyrazinamide and ethambutol: HRZE) with LFT monitoring. The patient was given antitubercular therapy for 9 months.

Figure 1



Bilateral diffuse nodular opacities.

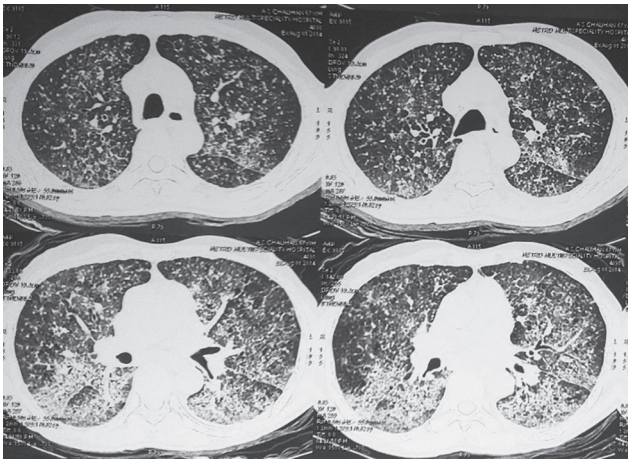
Figure 3



Thickened interstitium with mononuclear infiltration, type 2 pneumocyte hyperplasia and epithelioid cell granuloma without significant necrosis.

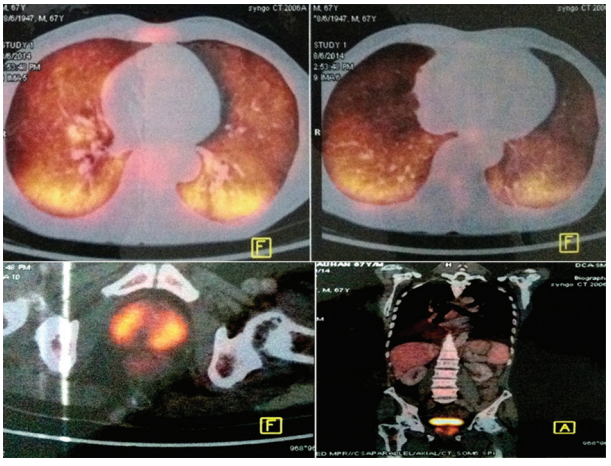
On detailed history taking, it was discovered that he had a history of recurrent prostatitis for a year and 6 months back he had consulted an urologist. His PSA was found to be high (7.5 ng/ml); TRUS-guided 12 segmental biopsies were taken, which was reported as granulomatous inflammation with necrosis with no evidence of malignancy seen. His chest radiograph obtained before biopsy was normal (Fig. 6). He was treated with multiple courses of antibiotics, and also prescribed fluoroquinolones for 2 weeks. The patient had reported relief in his symptoms and was advised to continue tamsulosin. To evaluate whether the patient had prostatic TB as the primary focus, the patient's previous slides and samples of prostate biopsy carried out outside were retrieved for re-evaluation. HPE of biopsy specimen revealed epithelioid cell granuloma with caseation and necrosis (Fig. 7).

Figure 2



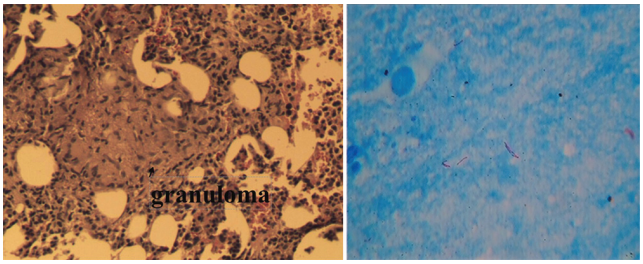
Bilateral randomly distributed miliary nodules more in lower lobes.

Figure 4



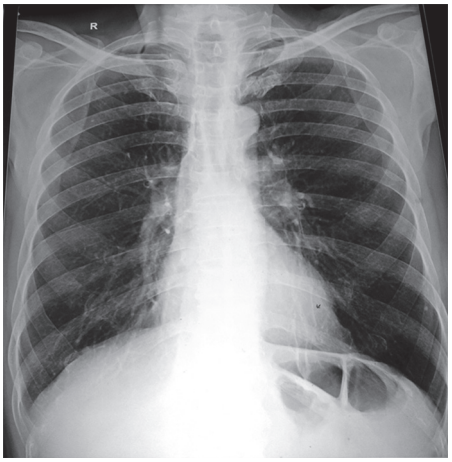
Increased ^{18}F-FDG uptake in bilateral lungs (SUV_{max} 4.7) and prostate (SUV_{max} 7.5), and mild diffuse ^{18}F-FDG uptake in the bone marrow of axial and proximal appendicular skeleton.

Figure 5



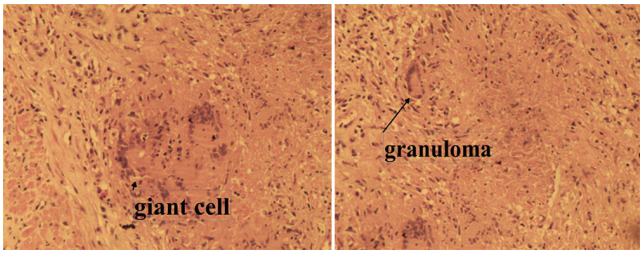
Granulomatous infiltration with AFB positive on ZN stain suggestive of dissemination of tuberculosis.

Figure 6



Normal chest radiography.

Figure 7



Epitheloid cell granuloma with caseation and necrosis.

Discussion

Epidemiology of prostate tuberculosis

Only 15–20% of extrapulmonary TB has been attributed to TB of the urogenital system and often results from haematogenous spread from an active site of infection [1]. Its development precedes the primary infection by at least 5–25 years [6]. Moreover, isolated involvement of prostate by TB is relatively less common as it usually coexists with renal/urethral/epididymal TB [7]. The incidence of primary prostatic TB is unknown and in its truest sense a very rare entity [1,4,5]. In our case, the patient had only symptoms of prostatitis and no other clinical or radiological features at the initial onset of illness to suggest involvement at any other site in the body. The patient complained of respiratory symptoms, fever and weight loss only in the past 3 weeks before presenting to us. Our patient did not have any past history of pulmonary tuberculosis, or any recent exposure of TB. In addition, chest x-ray before prostate biopsy was normal. Although it is difficult to rule out with utmost certainty in a country like India, that our patient might have had a primary elsewhere, we would like to prudently believe that the tubercular focus in prostate was primary and hypothesize that prostatic needling and delay in treatment both were inciting incidents for dissemination of TB in our patient. In our case, PET-CT helped in elucidating the dissemination of the disease and in taking representative biopsies for confirmation of diagnosis without much delay, thus highlighting the importance of PET-CT in the evaluation of nonmalignant diseases as well.

Diagnostic dilemma

Diagnosis of prostatic TB is challenging, as clinical presentation of prostatic TB is insidious and nonspecific. Therefore, maintaining a high index of suspicion when elucidating clinical history from patients presenting with chronic prostatitis is the most critical step. This is because in most clinical practice, prostatic biopsy is carried out to only rule out prostate malignancy as it is a much common entity in this age group. Identifying the cause of granulomatous prostatitis is the second crucial point and is possible only in limited instances. Nonspecific granulomatous prostatitis is usually an incidental finding and is presently the most frequent variety of granulomatous prostatitis observed at histological examination [8]. It accounts for most cases of granulomatous prostatitis (up to 69%) [9]. However, in a study by Epstein and Netto [2] on 25 387 benign prostate specimens, the incidence was reported to be 0.5%. Extensive pathological studies have been able to differentiate the nonspecific (lobulocentric, mixed with variable eosinophils in vicinity of granuloma and without much necrosis or caseation) from the infective ones (scattered, mixed with many multinucleated giant cells and epithelioid histiocytes, with significant necrosis) [2,9]. Therefore, it is important to differentiate infective granulomatous prostatitis from nonspecific granulomatous variety as it is a self-limiting benign condition, whereas the former requires specific treatment.

Our case is an excellent example of these facts, as the patient had presented with nonspecific prostatic symptoms to the urologist, who effectively ruled out carcinoma but undermined the granulomatous inflammation in the biopsy sample, thus leading to delay in diagnosis, dissemination and increase in

morbidity. We also believe that PET-CT should be more frequently utilized to evaluate disease spread [10].

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Conflicts of interest
There are no conflicts of interest.

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