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Prostatic Tuberculous abscess masquerading as benign prostatic hyperplasia: A rare incidental finding during transurethral resection

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Abstract
Prostatic tuberculosis is an uncommon manifestation of genitourinary tuberculosis and may remain clinically silent or present with nonspecific lower urinary tract symptoms that closely resemble benign prostatic hyperplasia, chronic prostatitis, or prostate malignancy. We report a 58-year-old man from Northern Karnataka, India, who presented with an eight-month history of progressive lower urinary tract symptoms and a recent episode of acute urinary retention requiring catheterization. Digital rectal examination and ultrasonography suggested benign prostatic enlargement, while serum prostate-specific antigen was mildly elevated at 4.1 ng/mL. Because symptoms persisted despite medical therapy and a trial without catheter was unsuccessful, transurethral resection of the prostate was undertaken. During resection of the left lateral lobe, an unexpected cavity was entered with drainage of approximately 15-20 mL of thick yellowish-white purulent material. The cavity was completely unroofed and drained. Ziehl-Neelsen staining demonstrated acid-fast bacilli, and GeneXpert MTB/RIF detected *Mycobacterium tuberculosis* complex without rifampicin resistance. Histopathology showed necrotizing granulomatous prostatitis characterized by epithelioid granulomas, Langhans-type giant cells, and central caseous necrosis, together with benign prostatic hyperplasia. The patient received two months of isoniazid, rifampicin, pyrazinamide, and ethambutol followed by four months of isoniazid and rifampicin. At six months, he remained symptom free with an IPSS of 4 and maximum urinary flow rate of 19.5 mL/s. This case emphasizes that tuberculous prostatic abscess should remain an important differential diagnosis in patients with unexplained or atypical lower urinary tract symptoms, particularly in tuberculosis-endemic regions, and demonstrates the diagnostic value of histopathological and molecular examination of unexpected intraoperative prostatic lesions.

Keywords: Prostatic tuberculosis, tuberculous prostatic abscess, benign prostatic hyperplasia, transurethral resection of prostate, genitourinary tuberculosis, granulomatous prostatitis, GeneXpert

Introduction
Tuberculosis remains an important infectious disease worldwide, and extrapulmonary involvement continues to present substantial diagnostic challenges. Genitourinary tuberculosis represents an important form of extrapulmonary tuberculosis because it may involve the kidneys, ureters, urinary bladder, prostate, seminal vesicles, epididymis, testes, and other structures of the male or female genital tract. Unlike pulmonary tuberculosis, genitourinary tuberculosis frequently develops with few or nonspecific constitutional manifestations, resulting in delayed recognition and potentially irreversible urological complications. Contemporary urological reviews therefore emphasize the importance of maintaining clinical suspicion and using microbiological, molecular, histopathological, and imaging investigations in combination when genitourinary tuberculosis is considered [1-3].
Tuberculous involvement of the prostate is particularly uncommon. The prostate may become infected through hematogenous dissemination of *Mycobacterium tuberculosis*, descending infection from the urinary tract, or less frequently direct or lymphatic extension from adjacent genital structures. Isolated male genital tuberculosis is especially difficult to diagnose because patients frequently lack a previous history of pulmonary tuberculosis, constitutional symptoms, or obvious radiological evidence of tuberculosis elsewhere [4]. The clinical presentation may include dysuria, urinary frequency, obstructive lower urinary tract

symptoms (LUTS), perineal discomfort, hematospermia, urinary retention, or an abnormal prostate on digital rectal examination. However, none of these findings is specific.

Another important diagnostic difficulty is the ability of prostatic tuberculosis to mimic common prostatic disorders. Prostate-specific antigen (PSA) may be normal or elevated, while ultrasonography and magnetic resonance imaging may reveal abnormalities resembling benign prostatic hyperplasia (BPH), chronic prostatitis, granulomatous prostatitis, or prostate carcinoma [5, 6]. Recent reports continue to demonstrate coexistence of prostatic tuberculosis with nodular prostatic hyperplasia, further complicating clinical interpretation [7].

Tuberculous prostatic abscess formation is an even rarer manifestation. When not identified preoperatively, tuberculosis may occasionally be detected only after prostate biopsy or transurethral resection performed for presumed BPH or malignancy. A recent 2026 case similarly demonstrated incidental prostatic tuberculosis following transurethral resection of the prostate (TURP), reinforcing the continuing relevance of this diagnostic pitfall [8]. We report a case of an unsuspected tuberculous prostatic abscess discovered during TURP for presumed benign prostatic hyperplasia in an immunocompetent 58-year-old man without constitutional or radiological evidence of pulmonary tuberculosis.

Case Presentation

A 58-year-old man residing in rural Northern Karnataka, India, presented to the Department of Urology with progressive obstructive and irritative lower urinary tract symptoms of approximately eight months' duration. His predominant complaints included hesitancy, weak urinary stream, nocturia, and intermittency. Two weeks before presentation, he developed an episode of acute urinary retention requiring urethral catheterization. His International Prostate Symptom Score was 24/35, indicating severe LUTS.

There was no history of fever, night sweats, unexplained weight loss, chronic cough, hemoptysis, or documented previous tuberculosis exposure. The patient had no history of intravesical Bacillus Calmette-Guérin therapy. Serological testing for human immunodeficiency virus, hepatitis B surface antigen, and hepatitis C virus was non-reactive.

General and abdominal examinations were unremarkable. Digital rectal examination demonstrated a moderately enlarged, smooth, firm, non-tender prostate with preserved median sulcus and no palpable nodularity. These findings favored benign prostatic enlargement rather than malignancy or acute prostatitis.

Laboratory evaluation showed hemoglobin of 12.4 g/dL and a total leukocyte count of 8, 400/µL. The erythrocyte sedimentation rate was elevated at 48 mm during the first hour. Renal function was preserved, with serum creatinine of 0.9 mg/dL and blood urea of 24 mg/dL. Serum PSA was mildly elevated at 4.1 ng/mL. Urinalysis revealed 4-6 pus cells per high-power field, while routine urine culture showed no bacterial growth after 48 hours.

Transabdominal ultrasonography demonstrated a uniformly enlarged prostate measuring approximately 52 g with apparently homogeneous parenchyma. Post-void residual urine volume was 140 mL. No definite prostatic collection was recognized on the preoperative ultrasound examination. Chest radiography demonstrated no active pulmonary lesion or radiographic evidence of healed tuberculosis.

The patient had previously received an α1-adrenergic blocker, tamsulosin 0.4 mg, without satisfactory improvement. Because of persistent severe LUTS, the episode of acute urinary retention, and an unsuccessful trial without catheter, he was scheduled for standard TURP under spinal anesthesia.

Cystourethroscopy showed significant bilateral lateral lobe enlargement producing bladder outlet obstruction together with mild trabeculation of the urinary bladder. Resection was initiated in the conventional fashion. During deeper resection of the left lateral prostatic lobe, an unexpected cavity was entered, resulting in sudden drainage of approximately 15-20 mL of thick, creamy, yellowish-white purulent material into the prostatic urethra.

The operative field was immediately irrigated. The abscess cavity was completely unroofed to permit adequate drainage, followed by completion of resection of the remaining hyperplastic prostatic tissue and meticulous hemostasis. The purulent material was sent for Gram staining, routine bacterial culture, Ziehl-Neelsen staining, and GeneXpert MTB/RIF analysis. Resected prostatic chips were submitted for histopathological examination.

Gram staining revealed abundant pus cells without identifiable pyogenic organisms. GeneXpert MTB/RIF detected DNA belonging to the *Mycobacterium tuberculosis* complex, with no rifampicin resistance detected. Ziehl-Neelsen staining of the purulent material demonstrated acid-fast bacilli.

Histopathological examination of the TURP chips revealed extensive necrotizing granulomatous inflammation. Multiple confluent epithelioid-cell granulomas containing Langhans-type multinucleated giant cells were identified, surrounded by lymphocytes and prominent central areas of caseous necrosis. Adjacent areas showed benign stromal and glandular hyperplasia consistent with BPH. Taken together, the histopathological and microbiological findings established the diagnosis of tuberculous prostatitis with prostatic abscess formation coexisting with benign prostatic hyperplasia.

Postoperatively, continuous bladder irrigation was maintained and a urethral catheter was retained for 48 hours. The postoperative course was uncomplicated, and the patient achieved satisfactory spontaneous voiding following catheter removal on the third postoperative day.

Following confirmation of tuberculosis, standard antitubercular therapy was initiated, comprising an intensive phase of isoniazid, rifampicin, pyrazinamide, and ethambutol for two months, followed by a continuation phase of isoniazid and rifampicin for four months. Renal and hepatic function tests monitored during therapy remained within reference limits.

At six-month follow-up, the patient reported complete resolution of his lower urinary tract symptoms. His IPSS had decreased from 24 before surgery to 4, and uroflowmetry demonstrated a maximum urinary flow rate of 19.5 mL/s. There was no recurrent urinary retention or development of systemic symptoms suggestive of tuberculosis.

Table 1: Summary of clinical parameters, preoperative investigations, and diagnostic outcomes

Age / Sex	58-year-old Male
Primary Presentation	Progressive LUTS (IPSS: 24/35), acute urinary retention
Digital Rectal Exam	Grade II enlarged, smooth, firm, non-tender, no nodules
Serum PSA	4.1 ng/mL
USG Prostate Volume	52 grams
Chest X-Ray	Clear (No active or old pulmonary TB)
Intraoperative Finding	Sudden egress of ~15-20 mL thick, creamy pus upon chips resection
Histopathology	Necrotizing granulomatous prostatitis with caseous necrosis & Langhans giant cells
Microbiology	GeneXpert MTB positive; ZN Stain positive for Acid-Fast Bacilli (AFB)

Discussion

Prostatic tuberculosis is a rare and frequently underdiagnosed form of genitourinary tuberculosis. Its clinical significance lies in its ability to mimic benign prostatic hyperplasia (BPH), bacterial or granulomatous prostatitis, and prostate carcinoma. Nonspecific urinary symptoms, sterile pyuria, and chronic prostatitis-like presentations may be the only clues to the diagnosis [1-4].

The present patient demonstrated this diagnostic challenge. He presented with progressive lower urinary tract symptoms (LUTS) and acute urinary retention but had no constitutional symptoms suggestive of tuberculosis. Digital rectal examination showed a smooth, non-tender enlarged prostate, while transabdominal ultrasonography demonstrated homogeneous enlargement, strongly favoring BPH preoperatively.

Prostatic tuberculosis may also closely resemble malignancy. Li *et al.* reported prostate tuberculosis mimicking prostate cancer, with considerable overlap in clinical and radiological features [5]. Similarly, William *et al.* described prostatic tuberculosis with multiparametric MRI findings highly suspicious for malignancy [6]. Thus, neither PSA nor imaging can reliably differentiate tuberculosis from malignant or other inflammatory prostatic conditions in all patients.

The mildly elevated PSA of 4.1 ng/mL in the present case was also nonspecific, as PSA may rise due to BPH, urinary retention, infection, inflammation, instrumentation, or malignancy. Granulomatous inflammation and prostatic tissue destruction may further elevate PSA, contributing to diagnostic confusion.

An important finding in this case was the coexistence of BPH and prostatic tuberculosis. Melis *et al.* similarly reported isolated prostatic tuberculosis associated with nodular prostatic hyperplasia [7]. Histopathology in the present patient demonstrated benign stromal and glandular hyperplasia adjacent to tuberculous granulomatous inflammation, suggesting that both BPH and tuberculosis may have contributed to the obstructive symptoms.

The prostatic abscess was not identified on preoperative transabdominal ultrasonography. Tuberculous lesions may appear as granulomas, areas of caseation, or abscesses, and transrectal ultrasonography or multiparametric MRI may provide better characterization when clinically suspected [5, 6]. However, the typical BPH-like presentation in this patient provided little indication for advanced imaging before TURP.

The unexpected drainage of thick purulent material during TURP was therefore the key diagnostic event. Gram staining revealed pus cells without pyogenic organisms, while conventional culture was negative. The combination of sterile pyuria, prostatic abscess, and negative bacterial studies should raise suspicion of *Mycobacterium tuberculosis*, particularly in tuberculosis-endemic regions.

Incidental detection of prostatic tuberculosis during TURP continues to be reported. Su *et al.* described a 72-year-old patient in whom prostatic tuberculosis was diagnosed histopathologically after TURP, with subsequent identification of disease at other sites [8]. Such cases demonstrate that prostatic tuberculosis may remain unsuspected until surgical tissue is examined.

Histopathology remains fundamental to diagnosis. The presence of epithelioid granulomas, Langhans-type giant cells, and caseous necrosis strongly suggested tuberculosis in the present case. Ziehl-Neelsen staining demonstrated acid-fast bacilli, while GeneXpert MTB/RIF confirmed *M. tuberculosis* complex without rifampicin resistance. Molecular testing is particularly useful because it provides rapid microbiological confirmation and information regarding rifampicin resistance [1-3].

Management required treatment of both the abscess and the underlying tuberculosis. Endoscopic unroofing during TURP provided effective drainage, relieved bladder outlet obstruction, and supplied tissue for diagnosis. However, surgical drainage alone is insufficient to eradicate tuberculosis. The patient therefore received two months of isoniazid, rifampicin, pyrazinamide, and ethambutol followed by four months of isoniazid and rifampicin, consistent with recommended therapy for drug-susceptible urogenital tuberculosis [1, 2].

At six-month follow-up, the patient showed complete symptomatic improvement, with IPSS decreasing to 4 and Qmax improving to 19.5 mL/s. This favorable outcome indicates successful management of both the obstructive and infectious components of the disease.

This case emphasizes that absence of constitutional symptoms, pulmonary abnormalities, or known tuberculosis exposure does not exclude prostatic tuberculosis. The disease may coexist with true BPH and remain undetected until surgery. In tuberculosis-endemic settings, unexplained sterile pyuria, granulomatous prostatitis, atypical prostate lesions, or unexpected purulent material during TURP should prompt comprehensive histopathological and mycobacterial evaluation.

Conclusion

Tuberculous prostatic abscess is a rare manifestation of genitourinary tuberculosis that can closely mimic benign prostatic hyperplasia. In this case, a 58-year-old man with refractory LUTS and acute urinary retention underwent TURP for presumed BPH, during which an unsuspected prostatic abscess was discovered. Histopathology demonstrated caseating granulomas with Langhans-type giant cells, while Ziehl-Neelsen staining and GeneXpert confirmed *M. tuberculosis*. Endoscopic drainage followed by six months of antitubercular therapy resulted in complete clinical recovery. Prostatic tuberculosis should therefore be considered in atypical or unexplained prostatic disease, particularly in endemic regions, and unexpected prostatic pus or granulomatous tissue encountered during endoscopic

procedures should undergo appropriate microbiological and histopathological evaluation.

Ethics Approval & Patient Consent: Informed written consent was obtained from the patient for publication of this clinical case report and any accompanying intraoperative data.

Conflict of Interest: The authors declare no conflicts of interest.

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