

ISOLATED TUBERCULOUS PROSTATITIS IN A PATIENT WITH BENIGN PROSTATIC HYPERPLASIA COMPLICATED BY EPIDIDYMO-ORCHITIS: A CASE REPORT

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ABSTRACT

Background: Genitourinary tuberculosis (GUTB) is a common form of extrapulmonary tuberculosis but remains diagnostically challenging due to its ability to mimic malignancy and nonspecific inflammatory conditions. Prostatic tuberculosis is particularly rare and often underrecognized, leading to delayed treatment and disease progression.

Case Presentation: A 64-year-old male with benign prostatic hyperplasia presented with recurrent epididymo-orchitis and abnormal digital rectal examination findings suggestive of prostate malignancy. Multiparametric MRI showed indeterminate prostatic lesions. Histopathology from prostate biopsy and transurethral resection revealed caseating granulomatous prostatitis. Initial refusal of anti-tubercular therapy led to disease progression with bilateral epididymo-orchitis and scrotal abscess formation. Urine and pus cartridge-based nucleic acid amplification testing confirmed *Mycobacterium tuberculosis* without rifampicin resistance. The patient required right orchidectomy and was subsequently treated with standard anti-tubercular therapy, with clinical improvement.

Conclusion: This case highlights the protean manifestations of GUTB and underscores the importance of early histopathological and molecular diagnosis to prevent disease progression and avoidable surgical morbidity.

KEYWORDS: Genitourinary tuberculosis; Prostatic tuberculosis; Epididymo-orchitis; Granulomatous prostatitis; CBNAAT

INTRODUCTION

Background

Tuberculosis remains a major global health concern, particularly in TB-endemic regions. Genitourinary tuberculosis (GUTB) constitutes approximately 20–27% of extrapulmonary tuberculosis cases and predominantly affects individuals in their fourth and fifth decades of life, with a male preponderance. GUTB usually arises from hematogenous dissemination of *Mycobacterium tuberculosis* from a primary pulmonary focus and may manifest years after initial infection, even in the absence of active pulmonary disease (1, 2).

Prostatic tuberculosis is a rare and often underdiagnosed form of GUTB, frequently mimicking benign prostatic hyperplasia or prostate carcinoma due to non-specific clinical and radiological features (1, 3). Imaging modalities such as multiparametric MRI may suggest malignancy but lack diagnostic specificity without histopathological correlation. Delayed diagnosis can allow progression to involve adjacent structures, resulting in epididymo-orchitis, abscess formation, and the need for surgical intervention (4). Molecular diagnostics such as CBNAAT have significantly improved detection in smear-negative disease and enable early initiation of appropriate therapy (5).

Case Presentation

A 64-year-old male with a known history of benign prostatic hyperplasia (BPH) for three years, on regular medical management, was evaluated for acute onset pain and swelling of the left hemiscrotum of two days' duration, associated with intermittent low-grade fever. There was no history of dysuria, haematuria, burning micturition, lower urinary tract obstruction, abdominal pain, or constitutional symptoms. The patient denied any prior history of tuberculosis, contact with tuberculosis, or previous anti-tubercular therapy. He was a non-smoker and did not consume alcohol. No other comorbid illnesses were reported.

On local examination, the left hemiscrotum was noted to be enlarged, warm, and tender. The left testis and epididymis were tender on palpation, while the external urethral meatus was normal. Per rectal examination revealed normal anal tone; however, the prostate was found to be hard and nodular with suspected mucosal fixity. Systemic examination findings were otherwise unremarkable.

Baseline laboratory investigations were within normal limits except for mild leukocytosis and an elevated erythrocyte sedimentation rate. Ultrasonography of the scrotum demonstrated features suggestive of left epididymo-orchitis (Figure 1). In view of abnormal digital rectal findings, further evaluation with multiparametric magnetic resonance imaging (mpMRI) of the prostate was performed (Figure 2). The imaging revealed a few defined T2-hypointense nodules in the central gland (base and apex) with diffusion restriction, reported as Prostate Imaging Reporting and Data System (PIRADS) 1 lesions, along with a focal ill-defined T2-hypointense nodule involving the left peripheral zone at the apical region, classified as PIRADS 3 (Figure 3).

Subsequently, the patient underwent transrectal ultrasound-guided prostate biopsy, which demonstrated features consistent with benign prostatic hyperplasia along with focal granulomatous prostatitis. The patient was later subjected to transurethral resection of the prostate with meatal dilatation. Histopathological examination of the resected prostatic tissue revealed caseating granulomatous inflammation, suggestive of tuberculosis (Figure 4). Despite being advised to initiate anti-tubercular therapy, the patient declined treatment and was discharged after symptomatic improvement.

Six months later, the patient re-presented with pain and swelling of the right hemiscrotum. Repeat ultrasonography of the scrotum revealed bilateral epididymo-orchitis with a left intra-epididymal collection measuring approximately 3mL. Urine cartridge-based nucleic acid amplification testing (CBNAAT) detected *Mycobacterium tuberculosis* without rifampicin resistance. The patient was promptly initiated on standard first-line anti-tubercular therapy as per weight-based dosing. Due to disease progression and complications, a right orchidectomy was performed. Histopathological examination of the excised testis showed necrotizing granulomatous inflammation (Figure 5). Pus obtained from the scrotal abscess demonstrated acid-fast bacilli on Ziehl–Neelsen staining and tested positive for *Mycobacterium tuberculosis* on cartridge-based nucleic acid amplification testing, with no rifampicin resistance detected (Figure 6). At follow-up, the patient was noted to be in the continuation phase of therapy and was symptomatically improved, with good treatment tolerance.

DISCUSSION

This case demonstrates the complex and evolving nature of genitourinary tuberculosis, a condition well known for its ability to masquerade as malignancy or nonspecific inflammatory disease. The patient initially presented with acute epididymo-orchitis and suspicious prostatic findings, with imaging and clinical examination raising concern for carcinoma. Histopathology revealed granulomatous prostatitis, and subsequent progression to bilateral epididymo-orchitis occurred following delayed initiation of anti-tubercular therapy. This clinical course highlights the diagnostic ambiguity and potential for disease advancement when genitourinary tuberculosis is not promptly treated.

The presentation of prostatic tuberculosis mimicking prostate cancer in this case is consistent with multiple reports in the literature. Eziyi et al. (2020) and Li et al. (2023) both described patients with elevated PSA levels, abnormal digital rectal examination, and MRI features suspicious for malignancy, where definitive diagnosis was achieved only through biopsy demonstrating granulomatous inflammation (6, 7). In those cases, early initiation of anti-tubercular therapy resulted in normalization of PSA and radiologic resolution, contrasting with the present case where initial refusal of therapy likely contributed to disease dissemination. Imaging challenges are further supported by Lee et al. (2022), who demonstrated that granulomatous prostatitis can closely resemble malignancy on mpMRI, although lesion regression over time may aid differentiation (8).

Tuberculous epididymo-orchitis is another entity frequently misdiagnosed, often leading to unnecessary orchiectomy. Mehboob et al. (2022) reported that early recognition and prolonged medical therapy could prevent testicular loss, whereas delayed diagnosis required extended treatment (9). Conversely, Civelli et al. (2020) and Liu et al. (2021) described cases similar to the present report, where orchiectomy was performed due to suspicion of malignancy or non-resolving infection, with tuberculosis confirmed only on histopathology (10, 11). These findings underscore the importance of maintaining tuberculosis in the differential diagnosis of atypical or refractory scrotal infections.

Diagnostic delay has been consistently associated with worse outcomes in genitourinary tuberculosis. Fujimoto et al. (2025) demonstrated that prolonged unrecognized disease led to irreversible renal impairment, reinforcing the need for early molecular testing (12). In the present case, confirmation by CBNAAT aligns with findings by Adhikary et al. (2022) and Gupta et al. (2025), both of whom showed that CBNAAT significantly improves diagnostic yield in extrapulmonary tuberculosis while allowing rapid detection of rifampicin resistance, particularly in pus and necrotic tissue (13, 14).

This case also supports emerging hypotheses regarding local factors contributing to tuberculosis reactivation and progression. Figueiredo et al. (2023) suggested that urinary tract obstruction and local ischemia may promote reactivation of latent disease independent of systemic immunosuppression (15). The coexistence of benign prostatic hyperplasia and tuberculous prostatitis in this patient suggests a plausible, testable hypothesis that chronic outlet obstruction may facilitate local mycobacterial persistence or dissemination within the genitourinary tract.

From a clinical perspective, this case reinforces guideline-based principles advocating early tissue diagnosis and prompt initiation of anti-tubercular therapy to prevent irreversible damage. The need for combined medical and surgical management in advanced disease mirrors observations by Mittal et al. (2020), who reported high surgical intervention rates in delayed presentations but favourable outcomes when therapy was appropriately instituted (16).

CONCLUSION

This case emphasizes that genitourinary tuberculosis remains a diagnostic masquerade with significant consequences if overlooked. Key findings include progressive multisite involvement following delayed therapy and the critical role of histopathology and molecular diagnostics. The broader implication is the need for heightened clinical suspicion, particularly in endemic settings, even in elderly patients without prior tuberculosis history. This report contributes to the

growing evidence that early diagnosis can alter disease trajectory, supports further research into obstruction-related reactivation mechanisms, and calls for proactive diagnostic strategies to reduce preventable surgical morbidity.

Conflict of Interest:

No conflict of interest

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Author Contributions (CRediT Taxonomy):

Jesvanthan Elango: Conceptualization; Data curation; Investigation; Methodology; Visualization; Writing – original draft; Writing – review & editing.

Gangadharan Vadivelu: Conceptualization; Supervision; Validation; Clinical oversight; Writing – review & editing.

Arjun AS: Investigation; Data curation; Resources; Visualization; Writing – review & editing.

All authors read and approved the final manuscript and agree to be accountable for all aspects of the work.

Declaration:**Ethical Approval:**

Ethical clearance was obtained from the Institutional Ethics Committee of Saveetha Medical College. Written informed consent was secured from the patient, who understood the study's procedures before their participation.

Use of Artificial Intelligence and AI-Assisted Technologies:

Artificial intelligence–assisted tools were used during the preparation of this manuscript to support language refinement and manuscript organization. ChatGPT (OpenAI) was utilized to assist in structuring sections of the manuscript and improving clarity of expression. Grammarly was used for spelling and grammatical corrections, and QuillBot was employed for paraphrasing and proofreading. No AI tool was used for data analysis, interpretation of results, or generation of original scientific content. The authors critically reviewed all AI-assisted output and take full responsibility for the accuracy, originality, and integrity of the final manuscript.

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Figures and Tables:

Figures:



Figure 1: Ultrasonography of the scrotum showing features of left epididymo-orchitis.



Figure 2: Multiparametric MRI of the prostate showing T2-hypointense lesions in the central gland (PIRADS 1).



Figure 3: Multiparametric MRI of the prostate demonstrating a focal T2-hypointense lesion in the left peripheral zone (PIRADS 3)

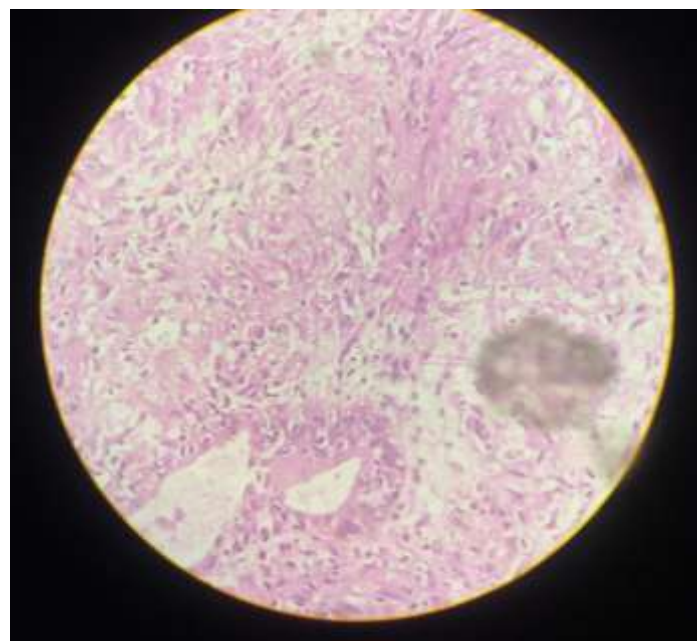


Figure 4: Histopathology of resected prostatic tissue showing caseating granulomatous inflammation consistent with tuberculous prostatitis (H&E stain)

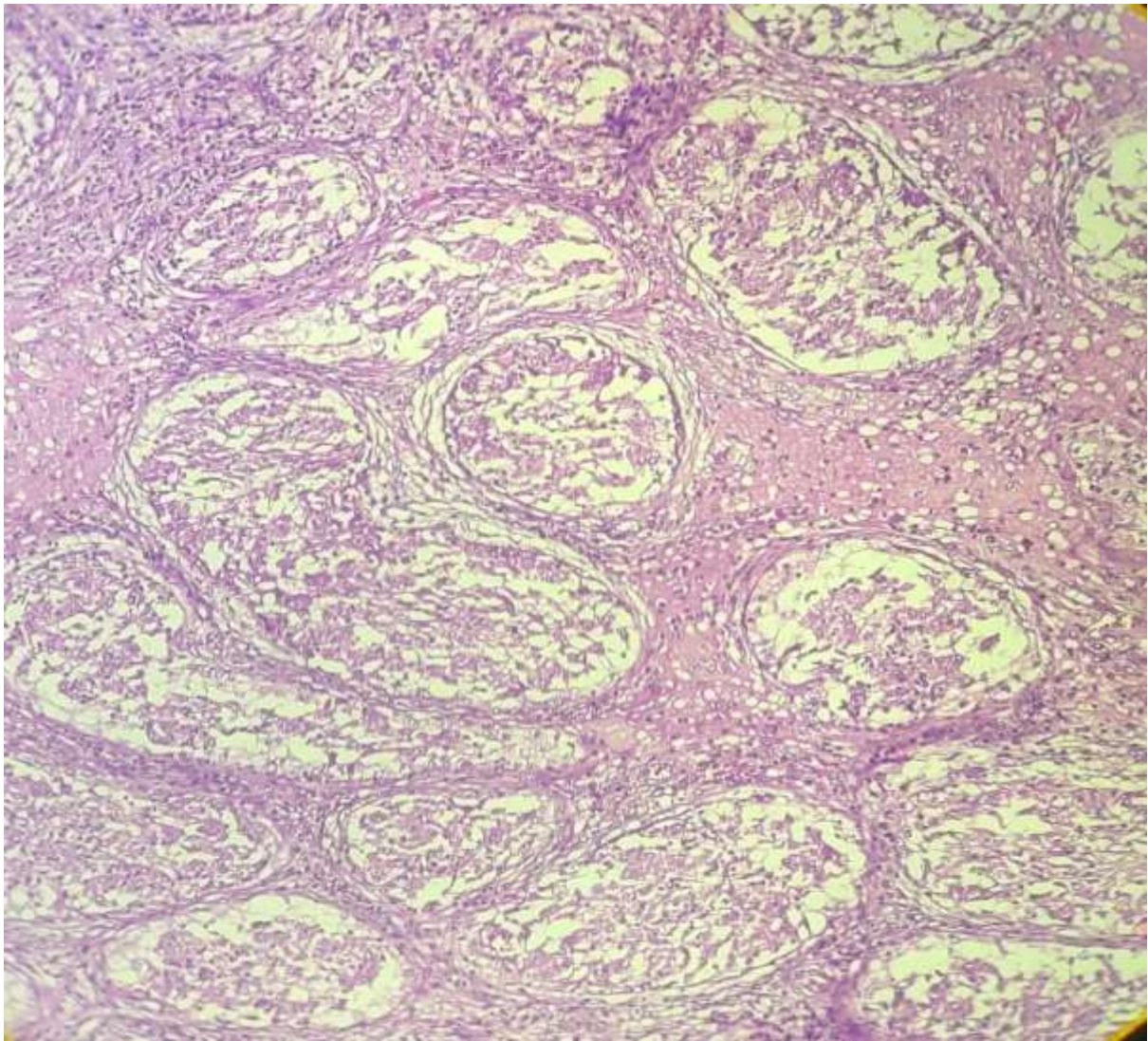


Figure 5: Histopathology of excised testis showing necrotizing granulomatous inflammation (H&E stain)

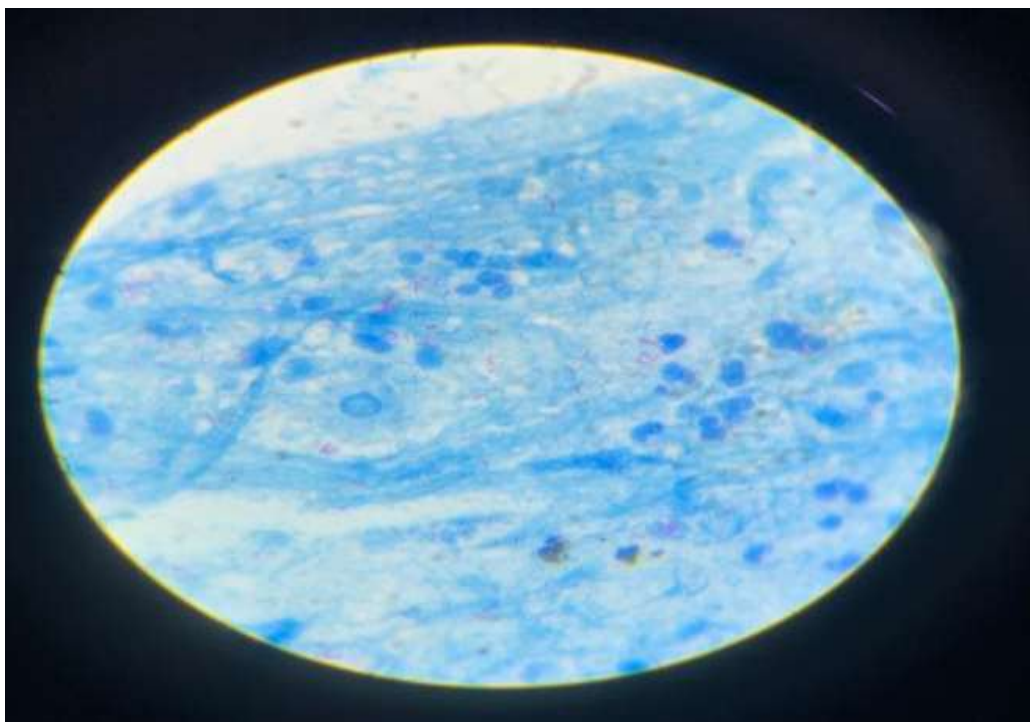


Figure 6: Ziehl–Neelsen–stained smear of scrotal abscess pus demonstrating acid-fast bacilli, with corresponding CBNAAT confirming *Mycobacterium tuberculosis*