CASE REPORT

GRANULOMATOUS PROSTATITIS MASQUERADING AS ADVANCED PROSTATE CANCER: A DIAGNOSTIC CHALLENGE

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Granulomatous prostatitis (GP) is a rare inflammatory condition, accounting for only 3.3% of all benign inflammatory lesions of the prostate. Histologically, it is characterized by granulomas secondary to infection, iatrogenic causes, or systemic granulomatous diseases. Most cases are idiopathic and termed nonspecific granulomatous prostatitis (NSGP). First described in 1943 by Tanner and McDonald, GP has an estimated incidence of 0.65–1.5% among inflammatory prostate lesions.

Keywords: Granulomatous prostatitis; Benign inflammatory lesion; Prostate

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INTRODUCTION

Granulomatous prostatitis (GP) is a rare inflammatory condition, accounting for only 3.3% of all benign inflammatory lesions of the prostate. Histologically, it is characterized by granulomas secondary to infection, iatrogenic causes, or systemic granulomatous diseases. Most cases are idiopathic and termed nonspecific granulomatous prostatitis (NSGP).[1,2] First described in 1943 by Tanner and McDonald, GP has an estimated incidence of 0.65–1.5% among inflammatory prostate lesions.[3] Notably, some studies report a 10–14% coexistence of GP and prostatic carcinoma, further complicating diagnosis. GP is a benign condition that can mimic prostate cancer clinically and radiologically. Findings such as hard nodules on digital rectal examination (DRE), elevated prostate-specific antigen (PSA) levels, hypoechoic lesions on transrectal ultrasound (TRUS), and malignant-appearing features on magnetic resonance imaging (MRI) pose significant diagnostic challenges, necessitating histological confirmation.[4]

We present a case where clinical and radiological findings strongly suggested locally advanced prostate cancer, but histopathology revealed granulomatous prostatitis. This underscores the critical role of tissue diagnosis in managing prostate diseases.

CASE PRESENTATION

A 66-year-old male presented to his primary care physician with constipation and lower urinary tract symptoms (LUTS), including increased urinary frequency, difficulty initiating urination, and a sensation of incomplete bladder emptying. His medical history was unremarkable except for a 40-pack-year smoking history and moderate alcohol intake. Urine microscopy, culture, and sensitivity identified *E. coli*, but his symptoms persisted despite completing a course of antibiotics.

On DRE, the prostate gland was hard and nodular, highly suggestive of malignancy. However, his PSA level was 2.8 ng/mL, within the normal range for his age. He was urgently referred to a urologist, who ordered a bi-parametric MRI of the prostate. The MRI revealed bilateral capsular breach (green arrow), nodular invasion of the left mesorectal fat, possible rectal wall invasion, and left seminal vesicle involvement, suggestive of T3B/T4 disease. The PSA density was 0.04 ng/mL/cc, and the prostate volume was approximately 66 cc.

The patient reported no symptoms of metastatic disease, such as weight loss, bone pain, or decreased appetite, and a bone scan showed no evidence of metastases. A transperineal prostate biopsy was performed, and histopathological examination revealed active chronic inflammatory cell infiltrate with acinar loss and fibrosis, consistent with granulomatous prostatitis. No evidence of neoplasia was found.

The patient was treated with a 4-week course of oral antibiotics per institutional guidelines and has remained asymptomatic to date.

DISCUSSION

This case highlights the diagnostic challenges posed by granulomatous prostatitis, which can closely mimic prostate cancer both clinically and radiologically. The patient's journey from initial LUTS to the final diagnosis of GP emphasizes the complexities of prostate disease management and the pitfalls of relying solely on a single diagnostic modality.

Granulomatous prostatitis can present with features indistinguishable from prostate cancer, including hard nodules on DRE, elevated PSA,

hypoechoic lesions on TRUS, and malignant-appearing changes on MRI. This makes histological confirmation essential for accurate diagnosis.[4]

LUTS are common to both benign prostatic conditions and prostate cancer. Initial antibiotic therapy was appropriate given the patient's recent urinary tract infection (UTI). While the UTI may have contributed to chronic inflammation and GP development, no definitive causal relationship was established. For recurrent or persistent GP, corticosteroids or anti-inflammatory agents may be considered.[5]

The hard, nodular prostate on DRE, combined with the patient's age and symptoms, warranted further investigation with PSA testing, specialist referral, and advanced imaging. However, the relatively low PSA (2.8 ng/mL) and PSA density (0.04 ng/mL/cc) were inconsistent with the aggressive disease suggested by MRI.

The MRI findings extra-prostatic extension, seminal vesicle invasion, and possible rectal involvement are typically associated with high-grade prostate cancer, making the final diagnosis of GP particularly striking. This discrepancy underscores the limitations of imaging in distinguishing GP from malignancy and reaffirms histopathology as the diagnostic gold standard.

The case also raises important questions about interpreting prostate MRI in the setting of active inflammation. Radiologists and urologists should maintain a high index of suspicion for inflammatory conditions, especially in patients with recent genitourinary infections or low PSA levels.

Although rare, the coexistence of GP and prostate cancer has been reported, necessitating careful follow-up after a GP diagnosis. Most GP cases are self-limiting, but oral antibiotics or corticosteroids may be used for symptomatic relief. In refractory cases, surgical interventions such as transurethral resection of the prostate (TURP) may be required to relieve urinary obstruction.[7]

CONCLUSION

A multidisciplinary approach incorporating clinical presentation, laboratory findings, imaging, and histopathology is essential for accurately diagnosing prostate diseases. This case reinforces the importance of considering benign inflammatory conditions like GP in the differential diagnosis of suspected prostate cancer, even when imaging strongly suggests malignancy.

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