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Prostatic Abscess Due to Idiopathic Granulomatous Disease: A Rare Complication of a Chameleon Disorder

—Prostatic Abscess Due to Idiopathic Granulomatous Disease

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Abstract

Background: Prostatic abscesses are usually diagnosed in the setting of bacterial prostatitis. Rarely, they reveal or complicate granulomatous prostatitis (GP). Four cases of idiopathic xanthogranulomatous GP have been described previously and the present case report is the first of typical idiopathic variety. The case: A 60-year-old man presented with urine retention that was associated with pyuria and massively enlarged prostate. Cystoscopy revealed prostatic abscess (PA) that was opened. Urine and prostatic culture were negative for bacteria. Prostatic biopsy revealed multiple non-caseating granulomata surrounded by lymphocytes, plasma cells yet without foamy histiocytes, parasites and vasculitis. Special stains were negative for vasculitis, fungiand acid-fast organisms. The patient was treated with Solumedrol 1 g intravenously daily for 3 days followed by Prednisone 1 mg/kg/day for 1 month followed by gradual tapering till discontinuation by 3rd month. Moreover, he had received Mycophenolate mofetil (MMF) 1 g twice/daily. By the end of 2nd month; he was asymptomatic and without pyuria. Repeat cystourethroscopy and MRI scan of the prostate showed near normal prostate. In Conclusion: Idiopathic GP can present with PA that requires proper drainage and since it is a locally hyperimmune disease with genetic predisposition; MMF therapy will be maintained for a total of 2 years to prevent future disease-relapse.

Keywords

Cystourethroscopy, Granulomatous Prostatitis, MRI, Mycophenolate Mofetil, Prostatic Abscess

1. Introduction

Granulomatous prostatitis (GP) is a rare inflammation of the prostate. It was first described by Tanner and McDonald in 1943 with an incidence of 3.3% of total inflammatory lesions of prostate [1]. The etiology has been classified, based on etiology and histopathology, into the following types: a) idiopathic (nonspecific) with typical and xanthogranulomatous variants, b) infectious due to tuberculosis, fungi, parasites and herpes simplex virus, c) Malakoplakia, d) iatrogenic as postsurgical and radiotherapy as well as BCG bladder immunotherapy, and e) systemic diseases that include; sarcoidosis, rheumatoid arthritis, Wegner's granulomatosis and polyarteritis nodosa [2]. It usually presents as a hard fixed nodule on rectal examination, hyperechoic shadow on ultrasound examination and is may be associated with high level of prostatic specific antigen simulating prostatic cancer [2]. Contrary to the diverse causes of GP that requires specific treatment, the nonspecific GP was considered a non-progressive, mild and self-limited disorder, that hardly requires more than alpha blockers [3]. On the other hand; bladder neck obstruction, due to prostatic abscess, has been reported in 4 cases with xanthogranulomatous variant [4]. However, Prostatic abscess (PA) due to the typical variant has not been reported before. In the present case report, we describe a patient with such rare disease and outline his diagnostic work up and management.

2. The Case

A 54-year-old man presented with recurrent urinary tract infections and suprapubic pain for months. Despite multiple antibiotic courses; he did not improve. Subsequently, he developed poor urine flow that did not improve with Alpha blockers and ultimately had severe urine retention that had required placement of foley catheter. He denied fever, chest and abdominal pain, skin rash and joint pains. Past history was significant for stable kidney transplantation for 25 years. His medications were Cyclosporin A 50 mg twice/daily with Prednisone 5 mg daily. On his initial physical examination, the patient was conscious and oriented X3. He was in distress of suprapubic and perineum pains. Blood pressure was 120/80 mm Hg. He was afebrile with a body weight of 75 kg. He did not have lymphadenopathy, goiter, jugular venous distension or oedema. Systemic examination did not show abnormality except for tenderness in the painful areas. Laboratory investigations showed normal peripheral leucocytic and platelets counts. Hemoglobin was 110 g/L with normal MCV. Serum urea and creatinine were elevated at 16 mmol/L and 201 umol/L, respectively. Serum glucose, electrolytes and liver functions were normal except for albumin at 28 g/L. Serum cholesterol and TSH were normal. Urine routine and microscopy showed 2(+) protein with excess WBCs and RBCs/HPF. Urine culture was sterile for and Zeil Nelson stains were negative. Real-time polymerase chain reaction (PCR) for mycobacterium tuberculosis was negative. Serum complements (C3 & C4) were normal. ANA, anti-ds DNA, ANCA, anti-GBM antibodies, hepatitis B surface antigen and an-

ti-HCV antibodies were negative. Chest x-ray and ECG were normal. Abdominal and pelvic ultrasound showed bilateral small native kidneys and normal transplanted one with huge prostate that had heterogenous echotexture. MRI showed enlarged prostate at 50 cc with multiple foci of high signal intensity (necrosis), restricted diffusion weighted images showing globular marked decrease in apparent diffusion coefficient (ADC) map and high signal intensity in diffusion weighted images (DWI) (**Figure 1(Aa-c)**). After relieve of bladder neck obstruction by placement of foley catheter; he underwent cystoscopy which showed extensive necrotic tissue in prostate and bladder neck with pus on incision (**Figure 2(A)**). Subsequent to pus drainage; Transurethral resection of prostate (TURP) was done (**Figure 2(B)**). Histopathological examination of the resected tissue showed diffuse nodular infiltrate with microabscesses within the ducts and acini in addition to epithelioid non-caseating granulomata (**Figure 3**). Those granulomata were composed of histiocytes, lymphocytes and plasma cells. There was no evidence of malignant cells, leucocytoclastic vasculitis and Bilharzia ova. Special stains were negative for fungi and mycobacteria. After establishment of final diagnosis; the patient was treated with Solumedrol 1 g IV for 3 consecutive days followed by Prednisone 60 mg daily and Mycophenolate mofetil (MMF) 1 g twice/daily. The latter induction therapy resulted in disappearance of symptoms and clearance of pyuria within 4 weeks. Pyuria and hematuria disappeared and serum creatinine returned to normal by the 8th week. By the 3rd month; repeat MRI showed significant improvement in prostatic sizeto (30 cc), decrease in size of ADC and DWI signals (**Figure 1(Ba-c)**). Moreover, repeat cystourethroscopy showed clean prostatic bed (**Figure 2(C)**). Prednisone was tapered down and discontinued after 3 months and MMP will be continued for a minimum of 2 years to prevent future disease-relapse. By now; the patient is stable after 1 year of therapy.

3. Discussion

Overall, the incidence of prostatic abscess can be as high as 0.5% of all urologic diseases, and the mortality rate is between 1% to 16%. Approximately 6% of acute bacterial prostatitis patients develop a prostate abscess [5]. Due to increased use of antibiotics; bacterial prostatic abscessesare rare. However, it remains a major complication of urological sepsis, in patients with diabetes, chronic renal failure, uretheral disease and in those on immunosuppression. *E. coli* and staph aureus are the most common culprits [6]. Large abscesses and those associated with bladder neck obstruction requiredrainage viz. transurethral aspiration, ultrasound-guided transrectal or perineal drainage [6]. Moreover, histopathological examination may disclose non-bacterial etiologies viz. GP; which requires different mode of management. PA, associated with GP, has been reported in 3 conditions viz. a) Tuberculosis (3% - 12%), b) Wegner's vasculitis (10%), and c) Xanthogranulomatous one (4 cases so far). The first 2 may present with florid systemic manifestations and/or lung disease [7]. The hallmark of tuberculous GP is

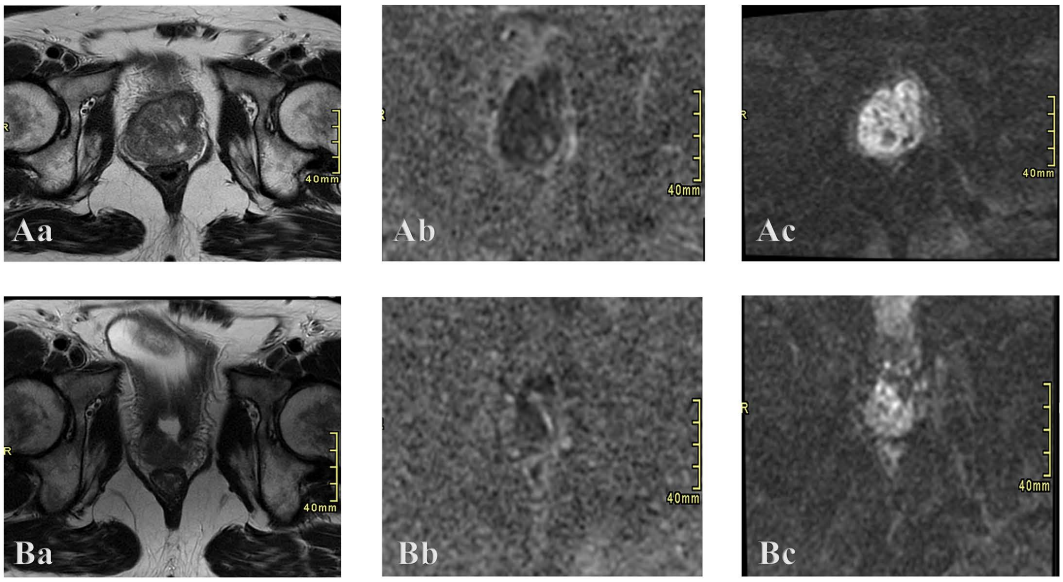

Figure 1. Axial view of MRI of the pelvis showing enlarged prostate (volume: 50 CC) with heterogeneous signal intensity showing multiple foci of high signal intensity necrosis (Aa), decrease in ADC (Ab) and high signal intensity (Ac). Repeat MRI images 3 months later shows decrease prostatic size to 30 CC with resolution of necrotic foci (Ba), decreased in size of ADC (Bb) and DWI (Bc) signals.

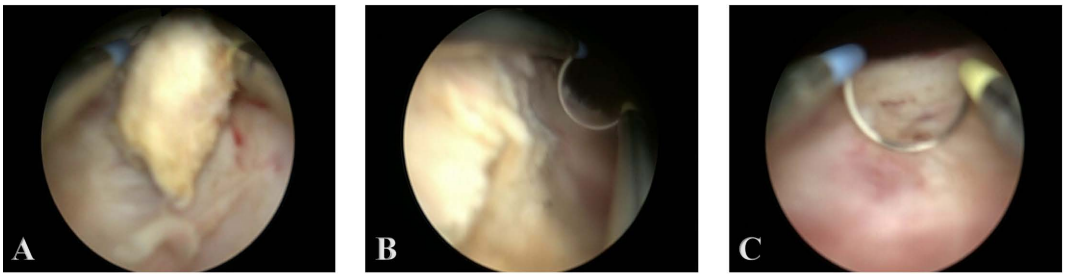

Figure 2. Cystoscopic picture showing pus pouring from prostate on initial assessment (A), after TURP (B), and finally; clean prostatic bed after treatment (C).

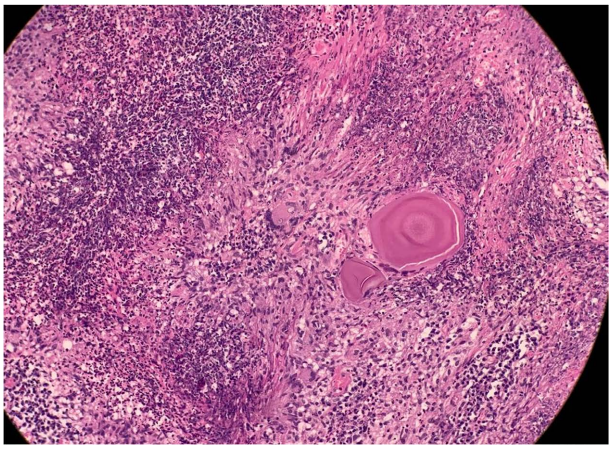

Figure 3. Photomicrograph of a prostatic biopsy showing diffuse nodular infiltrate with microabscesses within the ducts and acini in addition to epithelioid non-caseating granulomata (H & E stain, 200 × magnification).

the presence of confluent foci of caseous necrosis surrounded by epithelioid histiocytes. Moreover, PCR testing for mycobacterial DNA, culture, and stains for acid-fast bacilli are effective tools in diagnosing tubercular prostatitis. On the other hand; in a retrospective analysis of Wegner's disease; a) urogenital involvement was present atonset of WG in 9 of the 11 cases reported (82%), b) the first clinical evidence of WG in 2 cases (18%), c) a symptom of WG relapse in 6 cases (54%), d) symptomatic prostatitis 4 cases (36%), and e) and with suspicion of an abscess in 1 case (9%). In those cases, necrotizing granulomatous vasculitis was the hallmark of involvement. Lastly, Xanthogranulomatous disease was reported in the kidneys yet has been observed in rarer cases in the gallbladder and prostate [8]. The disease is characterized by lipid-laden macrophages, or "foamy histiocytes", with giant cells, lymphocytes, fibroplasia and (+)immunohistochemistry for CD68 (+) and CD68 (+) infiltrates [9]. In general, GP accounts for 1% of prostatic biopsies yet the nonspecific variety accounts for 69% - 77.7% of such cases and is often diagnosed incidentally [10]. Nowadays, its incidence has increased due to; a) post-TURP reaction to cautery and thermal alterations to prostatic epithelium and stroma, b) prostatic biopsy, and c) intravesical BCG immunotherapy for bladder cancer [11]. The previous etiologies for PA have been excluded in our study and biopsy has confirmed non-specific GP. It represents the first case of severe PA which had required surgical intervention and immunosuppressive therapy for minimum of 2 years to avoid future local inflammatory spread, fibrosis and bladder neck obstruction. The etiology of idiopathic GP is uncertain, but it is hypothesized to result from foreign body response to colloidal substance, bacterial products, or refluxed urine. It is now believed that nonspecific granulomatous prostatitis is autoimmune based with HLA-DR15-linked T cell response against proteins in prostatic secretions, especially PSA [11]. Similar phenomenon has been seen with severe idiopathic granulomatous ureteritis in which the inflammatory disease had recurred and even had progressed to severe ureteral stenosis with further involvement of the urinary bladder with masses and bladder neck obstruction. Similar induction therapy with Corticosteroids and MMP followed by MMP as a maintenance therapy was efficacious in that case. The phenomenon of disease development, recurrence and progression, of idiopathic granulomatous disease, indicates local hyperimmune response with malformation of the transitional epithelium in a genetically predisposed patient [12]. Our hypothesis of genetic predisposition is based on analysis of molecular pathology of ureteritis causing hydronephrosis in laboratory rodents [13]. Their stenotic lesions showed extensive infiltration with B-cell lymphocytes and comprehensive gene profiling revealed elevated expression of genes associated with hyperimmune responses through activation of B cells. Furthermore, their diseased ureters showed dramatically higher gene expression of chitinase 3-like 3, known as Ym1, which was associated with formation of adenomas in the transitional epithelium and of eosinophilic crystals in inflammatory conditions. The Ym1 protein was mainly localized to the cytoplasm of the transitional epithe-

lium, infiltrated cells, and eosinophilic crystals in diseased ureters. In our patient, we preferred MMF as the maintenance immunosuppressive drug for its safe and effective profile as well as its potent antiproliferative action via suppression of both B and T cells via stimulation of CD3/CD28 that inhibits T cell IL-17, IFN-γ and TNF-α production [14].

4. Conclusion

Idiopathic GP can present with severe PA that indicates surgical intervention and long-term immunosuppressive therapy to prevent local complications.

Statement of Ethics

The case was reported according to World Medical Association Declaration of Helsinki. There was no new or investigational drug added to the patient's maintenance therapy and they were not subjected to any harmful or injurious investigation.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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