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Prostatic Sarcoidosis Presenting With Acute Urinary Retention and Detrusor Underactivity: A Case Report

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Abstract

Prostatic sarcoidosis is a rare manifestation of systemic sarcoidosis and may mimic acute bacterial prostatitis. We report a 66-year-old man who presented with fever and acute urinary retention and was initially treated for presumed bacterial prostatitis. Further evaluation, including histopathological examination, supported prostatic sarcoidosis rather than bacterial prostatitis, and corticosteroid therapy resulted in clinical improvement. Pressure-flow study after treatment demonstrated detrusor underactivity without persistent bladder outlet obstruction, suggesting that impaired detrusor contractility may have contributed to urinary retention. This report provides additional urological insight into a rare presentation of prostatic sarcoidosis.

Categories: Urology
Keywords: acute urinary retention, detrusor underactivity, pressure-flow study, prostate, sarcoidosis

Introduction

Sarcoidosis is a systemic granulomatous disease of unknown etiology that most commonly involves the lungs, lymph nodes, skin, and eyes [1]. Genitourinary involvement is uncommon, and clinically apparent prostatic sarcoidosis is exceptionally rare [2]. Because patients may present with fever, lower urinary tract symptoms, prostate enlargement, and elevated serum prostate-specific antigen (PSA), prostatic sarcoidosis can closely mimic acute bacterial prostatitis or prostate cancer, making an accurate diagnosis challenging.

The systemic manifestations and histopathological diagnosis of the present patient have previously been reported from a nephrological perspective, with particular emphasis on interstitial nephritis accompanied by necrotizing vasculitis [3]. The previous report established the diagnosis of systemic sarcoidosis involving the kidney, mediastinal lymph nodes, and prostate. However, detailed assessment of lower urinary tract function, including pressure-flow study (PFS), a urodynamic test that measures bladder pressure and urinary flow during voiding, was not reported, and the mechanism underlying acute urinary retention, defined as the sudden inability to void, was not investigated.

The present report extends the previous nephrological report by providing previously unpublished urological findings, particularly the results of a pressure-flow study, to investigate the mechanism underlying acute urinary retention associated with prostatic sarcoidosis. Urinary retention may result from bladder outlet obstruction (BOO), which increases resistance to urinary flow, detrusor underactivity (DU), which impairs bladder emptying due to insufficient bladder muscle contraction, or a combination of both. We report PFS findings demonstrating DU without persistent BOO after corticosteroid therapy and discuss the potential contribution of both transient inflammatory outlet obstruction and impaired detrusor contractility to urinary retention.

Case Presentation

A 66-year-old man presented to a local hospital with several days of persistent fever and acute urinary retention. He had no apparent lower urinary tract symptoms before the onset of urinary retention. He was diagnosed with acute bacterial prostatitis and treated with oral levofloxacin and silodosin (8 mg/day), and a urethral catheter was placed at the referring hospital, draining 520 mL of urine. Neither urine nor blood cultures were obtained at the referring hospital before the initiation of levofloxacin. However, his fever persisted despite six days of antimicrobial therapy, and he was referred to our department for further evaluation and management.

On admission, his body temperature was 37.5 °C, blood pressure was 139/75 mmHg, and pulse rate was 85 beats/min. Digital rectal examination revealed prostatic tenderness without obvious induration or enlargement. Laboratory investigations revealed leukocytosis with neutrophilia, markedly elevated C-reactive protein and serum PSA levels (Table *1*). Because fluoroquinolone-resistant bacterial prostatitis was suspected, intravenous meropenem (1.0 g/day) was initiated. Nevertheless, high-grade fever and marked

systemic inflammation persisted. Blood and urine cultures remained negative, and the serum procalcitonin level was within the normal range (Table 1), findings that were atypical for bacterial infection.

Laboratory parameter	Value	Unit	Reference range
Hematology			
White blood cell count (WBC)	9,700	/μL	4,000 - 8,000
Neutrophils	86	%	45 - 74
Lymphocytes	7	%	20 – 45
Eosinophils	7	%	0 - 6
Basophils	0	%	0 – 3
Red blood cell count (RBC)	4.81	10^6/μL	4.10 - 5.40
Hemoglobin (Hb)	13.7	g/dL	14.0 - 18.0
Hematocrit (Hct)	41	%	39.0 - 50.0
Platelet count (Plt)	189	10^3/μL	120 - 400
Biochemistry			
Total protein (TP)	7.3	g/dL	6.7 - 8.3
Total bilirubin (T-Bil)	0.7	mg/dL	0.2 - 1.2
Aspartate aminotransferase (AST)	34	U/L	8 – 38
Alanine aminotransferase (ALT)	25	U/L	4 - 44
Lactate dehydrogenase (LDH)	287	U/L	120 – 245
γ-Glutamyl transferase (γ-GT)	44	U/L	< 80
Amylase (AMY)	56	U/L	37 – 125
Glucose (Glu)	149	mg/dL	70 - 109
Blood urea nitrogen (BUN)	22.1	mg/dL	8.0 - 20.0
Uric acid (UA)	5.2	mg/dL	2.0 - 7.0
Creatinine (Cre)	1.09	mg/dL	0.66 - 1.11
eGFR	53.8	mL/min/1.73 m²	≥90
Electrolytes			
Sodium (Na)	139	mEq/L	137 – 147
Potassium (K)	3.5	mEq/L	3.5 - 5.0
Chloride (Cl)	101	mEq/L	98 – 108
Calcium (Ca)	8.8	mg/dL	8.4 - 10.2
Phosphorus (P)	2.9	mg/dL	2.5 - 4.5
Inflammatory / tumor markers			
C-reactive protein (CRP)	14.04	mg/dL	< 0.30
Prostate-specific antigen (PSA)	16.31	ng/mL	< 3.52
Microbiology / other			
Blood culture	Negative	—	Negative
Urine culture	Negative	—	Negative
Procalcitonin	0.19	ng/mL	< 0.5

TABLE 1: Laboratory findings on admission to our hospital

Abnormal values are shown in bold.

WBC, white blood cell count; LDH, lactate dehydrogenase; BUN, blood urea nitrogen; eGFR, estimated glomerular filtration rate; CRP, C-reactive protein; PSA, prostate-specific antigen

Contrast-enhanced computed tomography and multiparametric magnetic resonance imaging demonstrated diffuse enlargement of the prostate (estimated prostate volume, 39.1 mL) without evidence of abscess formation or malignancy (Figure *1A*). Prostate volume was manually estimated using the ellipsoid formula: 0.523 × anteroposterior diameter × transverse diameter × longitudinal diameter (all measured in cm). Mediastinal lymphadenopathy prompted lymph node biopsy, while worsening renal function (serum creatinine, 1.1 to 2.6 mg/dL by hospital day 14) prompted renal biopsy.

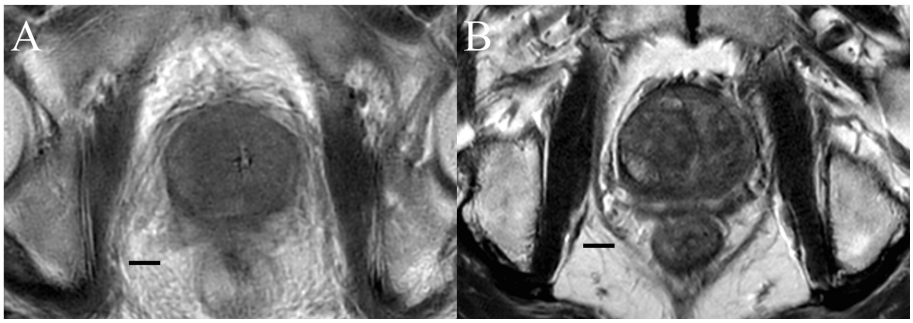


FIGURE 1: Magnetic resonance images of the prostate before and after corticosteroid therapy

(A) Axial T2-weighted magnetic resonance imaging (MRI) at diagnosis, before initiation of corticosteroid therapy, showing diffuse enlargement of the prostate with an estimated prostate volume of 39.1 mL. (B) Axial T2-weighted MRI obtained three months after initiation of corticosteroid therapy. The prostate volume was 39.8 mL, showing no substantial change compared with that before treatment. Black scale bars indicate 1 cm.

Lymph node and renal biopsies revealed non-caseating epithelioid granulomas, prompting subsequent prostate biopsy, which confirmed prostatic involvement and established the diagnosis of systemic sarcoidosis [3]. Prostate biopsy demonstrated non-caseating epithelioid granulomas with surrounding lymphocytic infiltration, without prominent neutrophilic infiltration or evidence of malignancy (Figure *2*). There was no history of intravesical BCG therapy. Although tuberculosis-specific testing was not performed, granulomatous involvement of multiple organs supported the diagnosis of systemic sarcoidosis.

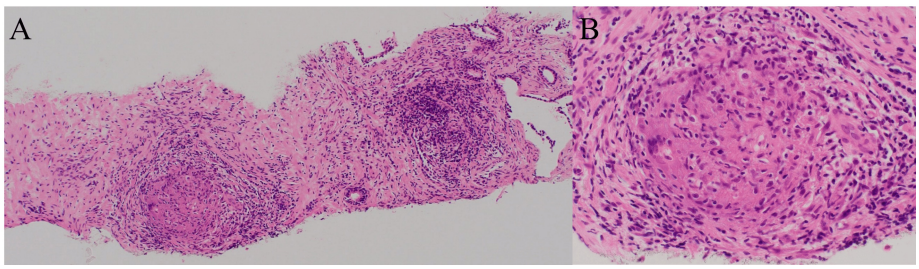


FIGURE 2: Histopathological findings of the prostate biopsy.

(A) Hematoxylin and eosin staining (10× objective) showing non-caseating granulomatous inflammation in the prostatic tissue. (B) Higher-power view (20× objective) of the same granuloma shown in panel A, demonstrating a non-caseating epithelioid granuloma with surrounding lymphocytic infiltration. No prominent neutrophilic infiltration or evidence of malignancy was identified.

These images show different areas of the prostate biopsy specimen from those presented in the previous report [3].

Oral prednisolone was initiated at a dose of 60 mg/day on hospital day 23. The inflammatory response improved rapidly, and renal function recovered. The urethral catheter was removed on hospital day 52, after

which the patient was able to void spontaneously, and he was discharged four days later.

Three months after initiation of corticosteroid therapy, a pressure-flow study (PFS) was performed to evaluate lower urinary tract function and investigate the mechanism underlying the preceding episode of acute urinary retention. The PFS demonstrated no evidence of bladder outlet obstruction (Qmax, 9.1 mL/s; PdetQmax, 19.2 cmH_2O; bladder outlet obstruction index (BOOI), 1.0; Schäfer obstruction grade, 0). In contrast, detrusor contractility was reduced (bladder contractility index (BCI), 64.7; Schäfer contractility grade, W–), consistent with detrusor underactivity (Figure *3*). Follow-up magnetic resonance imaging performed three months after initiation of corticosteroid therapy demonstrated no substantial change in prostate volume (39.8 mL) compared with that at diagnosis (39.1 mL) (Figure *1B*), whereas serum PSA level had normalized to 3.18 ng/mL.

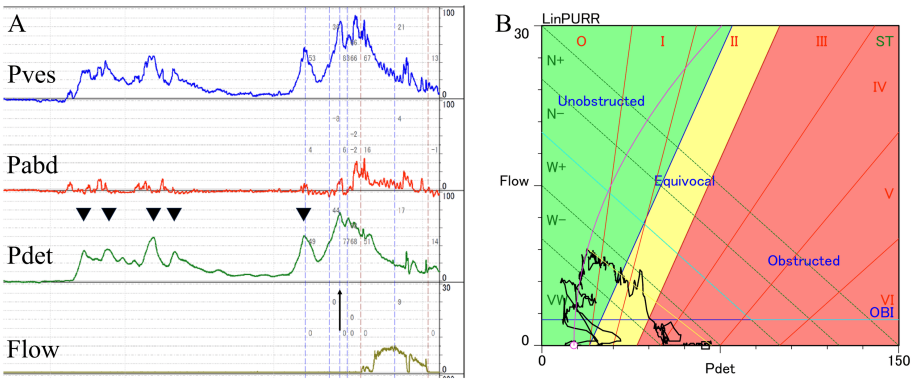


FIGURE 3: Pressure-flow study performed three months after initiation of corticosteroid therapy

(A) Pressure-flow study (PFS) tracing. The arrow indicates the time at which the patient was instructed to void, and arrowheads indicate detrusor overactivity during the filling phase. Pressure and urinary flow rate are expressed in cmH_2O and mL/s, respectively. PFS demonstrated no evidence of persistent bladder outlet obstruction (bladder outlet obstruction index (BOOI), 1.0) but showed reduced detrusor contractility (bladder contractility index (BCI), 64.7). (B) Schäfer/LinPURR nomogram showing Schäfer grade 0 for bladder outlet obstruction and W− for detrusor contractility, consistent with an unobstructed outlet and reduced detrusor contractility.

Multiple episodes of detrusor overactivity were observed during filling. The final episode induced a strong desire to void, and the patient was instructed to void at the point indicated by the arrow. After the initiation of voiding, detrusor pressure gradually decreased.

Pves, intravesical pressure; Pabd, abdominal pressure; Pdet, detrusor pressure (Pves − Pabd); Flow, urinary flow rate; LinPURR, linear passive urethral resistance relation; BOOI, bladder outlet obstruction index; BCI, bladder contractility index

At the three-year follow-up, the patient remained on maintenance prednisolone (3 mg/day) without recurrence of urinary retention, lower urinary tract symptoms, urinary tract infection, or systemic disease progression.

Discussion

Prostatic sarcoidosis is an exceptionally rare manifestation of systemic sarcoidosis, with only a limited number of cases reported in the literature [2,4]. Symptomatic cases appear to be uncommon, and prostatic involvement may be detected incidentally [4]. Fever, lower urinary tract symptoms, and elevated serum PSA may resemble acute bacterial prostatitis, making the diagnosis challenging [4].

The present patient was initially treated as having acute bacterial prostatitis because of fever, acute urinary retention, inflammatory laboratory findings, and a markedly elevated PSA concentration. However, several findings were inconsistent with bacterial infection. Despite empirical broad-spectrum antimicrobial therapy, fever and systemic inflammation persisted, while blood and urine cultures remained negative and the serum procalcitonin level was within the normal range. Furthermore, imaging studies demonstrated no prostatic abscess, and histopathological examination revealed non-caseating epithelioid granulomas with surrounding lymphocytic infiltration, without prominent neutrophilic infiltration or evidence of malignancy. Although negative cultures and a normal procalcitonin level do not exclude localized bacterial infection, the overall clinical and histopathological findings favored prostatic sarcoidosis over bacterial prostatitis. Although infectious and other causes of granulomatous prostatitis could not be completely excluded, the presence of granulomatous lesions in multiple organs supported the diagnosis of systemic sarcoidosis with prostatic involvement. Because antibiotics were administered before microbiological

testing, concomitant acute bacterial prostatitis cannot be excluded and may have independently contributed to urinary retention.

This patient has previously been reported from a nephrological perspective, with emphasis on systemic sarcoidosis associated with interstitial nephritis and necrotizing vasculitis [3]. The present report addresses a different clinical question by focusing on the urological manifestations of the disease. We describe previously unpublished pressure-flow study findings and discuss their implications for understanding the mechanism of acute urinary retention.

The principal novel finding of this report is the demonstration of detrusor underactivity without persistent bladder outlet obstruction on pressure-flow study after corticosteroid therapy. Although the urodynamic study was performed after resolution of the acute inflammatory phase and therefore cannot directly reflect bladder outlet conditions at the onset of urinary retention, it provides important insight into the patient's lower urinary tract function. Persistent anatomical or functional bladder outlet obstruction was not demonstrated, whereas bladder contractility remained impaired. These findings suggest that reduced detrusor contractility may have contributed to the development of urinary retention. Although detrusor pressure gradually decreased during voiding, the contraction was preceded by detrusor overactivity. Therefore, the calculated BCI may not accurately reflect voluntary detrusor contractility and should be interpreted with caution.

Several mechanisms may explain urinary retention in this patient. Prostatic inflammation may affect bladder outlet resistance, as suggested by studies of benign prostatic hyperplasia [5,6]. However, whether granulomatous inflammation associated with sarcoidosis produces a similar effect remains uncertain. Proinflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6) are increased in sarcoidosis and have been implicated in granulomatous inflammation [7]. IL-6 has also been associated with acute urinary retention [8], while TNF-α has been reported to modulate penile smooth muscle tone [9]. Although speculative, cytokine-mediated prostatic inflammation may have contributed to bladder outlet obstruction in this patient. Notably, prostate volume remained essentially unchanged after corticosteroid therapy despite normalization of serum PSA and resolution of urinary retention, suggesting that prostate enlargement alone was unlikely to account for the urinary retention. However, whole-gland volume measurements cannot exclude changes in local inflammation or edema. Moreover, impaired detrusor contractility could reduce the patient's ability to compensate for even mild outlet resistance. Therefore, acute urinary retention may have resulted from impaired detrusor contractility combined with a possible transient increase in outlet resistance. However, transient bladder outlet obstruction remains unconfirmed because PFS was not performed during the acute episode. Notably, no bladder outlet obstruction was demonstrated on PFS after corticosteroid therapy, although outlet conditions during the acute episode remain unknown.

Only a limited number of cases of prostatic sarcoidosis have been reported in the literature, and prostatic involvement has frequently been detected incidentally [4]. Case reports describing the detailed lower urinary tract functional characteristics of prostatic sarcoidosis are rare, and pressure-flow study findings in such patients have been scarcely reported. Accordingly, the present case expands the current understanding of lower urinary tract dysfunction associated with prostatic sarcoidosis by providing objective urodynamic evidence.

This case also has important clinical implications. In patients presenting with presumed acute bacterial prostatitis who show an atypical clinical course, including poor response to antimicrobial therapy, negative microbiological investigations, or unexplained systemic manifestations, clinicians should broaden the differential diagnosis to include systemic inflammatory diseases such as sarcoidosis. Furthermore, urodynamic evaluation may provide valuable information regarding the mechanism of urinary retention after resolution of the acute phase and may help guide subsequent management.

Several limitations should be acknowledged. First, this is a single-case report, and the findings may not be generalizable. Second, the pressure-flow study was performed after corticosteroid treatment; therefore, lower urinary tract function during the acute phase could not be evaluated directly. Finally, although impaired detrusor contractility was demonstrated after treatment, whether it predated the onset of sarcoidosis or developed secondary to inflammation cannot be determined. Therefore, the post-treatment urodynamic findings should not be interpreted as direct evidence of the mechanism underlying acute urinary retention at its onset. Future accumulation of similar cases will be necessary to clarify the pathophysiology of urinary retention associated with prostatic sarcoidosis.

Conclusions

This case highlights two important clinical messages. First, prostatic sarcoidosis should be considered in patients with presumed acute bacterial prostatitis who exhibit an atypical clinical course, particularly when antimicrobial therapy is ineffective and microbiological investigations are negative. Second, a pressure-flow study performed three months after initiation of corticosteroid therapy demonstrated detrusor underactivity without bladder outlet obstruction, suggesting that impaired bladder contractility may have contributed to acute urinary retention. Although a transient inflammation-related increase in bladder outlet resistance

may also have played a role, this mechanism remains unconfirmed because urodynamic evaluation was not performed during the acute episode.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Yuki Kyoda, Naoya Masumori

Acquisition, analysis, or interpretation of data: Yuki Kyoda, Yuito Momma, Tetsuya Shindo, Reiko Watanabe

Drafting of the manuscript: Yuki Kyoda, Yuito Momma

Critical review of the manuscript for important intellectual content: Yuki Kyoda, Tetsuya Shindo, Naoya Masumori, Reiko Watanabe

Supervision: Tetsuya Shindo, Naoya Masumori

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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