

CASE REPORT

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Multidisciplinary management of a Spondyloarthritis presenting with bladder involvement as the initial clinical symptom: a rare case report

Lingmin Song^{1*}, Shengdong Li², Jingjing Yu³ and Guobin Weng¹

Abstract

Background Spondyloarthritis (SpA) is a group of chronic inflammatory rheumatic diseases that can present with diverse extra-articular manifestations. However, bladder involvement as the primary clinical presentation has not been previously reported.

Case presentation : We present a case report of a 55-year-old male with a 20-year history of recurrent left lower abdominal pain and lower urinary tract symptoms (LUTS). Despite multiple treatments for presumed chronic prostatitis and cystitis, symptoms persisted. Imaging revealed bladder wall alterations and inflammatory changes in bilateral sacroiliac joints. Laboratory tests showed positive HLA-B27 expression. Histopathological examination of bladder tissue demonstrated chronic inflammation with eosinophilic infiltration and vasculitis. These findings led to a diagnosis of SpA, despite the absence of typical musculoskeletal symptoms. Treatment with nonsteroidal anti-inflammatory drugs (NSAIDs) and tumor necrosis factor (TNF) inhibitors resulted in complete resolution of urological symptoms and normalization of bladder morphology. After one month of continuous management, the patient experienced significant relief from left lower abdominal pain (NRS 2–3) and LUTS. The patient reported complete alleviation of pain (NRS 0) and LUTS at the three-month follow-up. No recurrence was observed during a 1-year follow-up period.

Conclusions This case highlights the potential for SpA to manifest primarily with urological symptoms, emphasizing the need for clinicians to consider systemic inflammatory conditions in cases of refractory LUTS. The successful diagnosis and management underscore the importance of interdisciplinary collaboration between urology and rheumatology.

Keywords Spondyloarthritis, Lower urinary tract symptoms, Bladder inflammation, Chronic pelvic pain, Extra-articular manifestations, Multidisciplinary approach, Case report

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Background

Spondyloarthritis (SpA) encompasses a group of chronic inflammatory rheumatic diseases with a global prevalence of 0.5–1.9%, placing a significant burden on the healthcare system [1, 2]. SpA is characterized by shared clinical, genetic, and imaging features, and typically presents with axial skeletal pain, peripheral arthritis, and tenosynovitis [3]. However, the insidious onset and diverse clinical manifestations often lead to diagnostic challenges, particularly in primary care settings [2]. The average delay in diagnosis is estimated to be 8–11 years from symptom onset [4]. A positive HLA-B27 expression, coupled with clinical indications of SpA, can substantiate the diagnosis [4]. Extra-articular manifestations are common in SpA, affecting various organ systems including the skin, eyes, and gastrointestinal tract [1, 3]. However, direct involvement of the urinary system, particularly the bladder, as a primary clinical presentation has not been previously documented in the literature.

This case report describes an unusual presentation of SpA where significant bladder dysfunction was the primary clinical symptom. The effective management of SpA was found to mitigate these bladder issues, highlighting a potentially underrecognized association between SpA and urological symptoms.

Case presentation

In March 2020, A 55-year-old male non-smoker presented with a 20-year history of recurrent, progressively worsening blunt pain in the left lower abdomen. The pain was associated with lower urinary tract symptoms (LUTS), including increased frequency and urgency of micturition, soreness (particularly at the end of urination), and occasional urinary incontinence during episodes of abdominal pain. Pain severity fluctuated between 3 and 6 on the Numerical Rating Scale (NRS). Notably, the patient reported no back pain, morning

stiffness, reduced spinal mobility, or joint pain. Over the years, he had been tentatively diagnosed with chronic prostatitis, pelvic pain syndrome, and chronic cystitis. Previous pelvic computerized tomography (CT) scans and ultrasound examinations revealed only a mild prostate enlargement with no significant bladder abnormalities. Urine tests yielded negative results, and the post-void residual volume (PRV) was 0 ml. Treatment with non-steroidal anti-inflammatory drugs (NSAIDs), α -blockers, and M-blockers provided moderate symptom relief. However, symptoms typically recurred and worsened within two months of discontinuing treatment.

On March 24, 2020, severe pain (NRS 6–8) and LUTS prompted the patient's visit to our hospital. Enhanced pelvic magnetic resonance imaging (MRI) disclosed a mild prostate enlargement and a notable thickening of the right posterior bladder wall measuring 1.0×0.5 cm, raising suspicions of a bladder tumor (Fig. 1). The patient was consequently admitted to our department. The patient denied any history of allergic diseases, hypertension, diabetes mellitus, joint pain/stiffness, sacroiliac stiffness, or chronic pain. No family history of hereditary disorders, malignancies, or similar conditions was reported. Physical examination revealed local muscle tension in the left lower abdomen and inguinal area, accompanied by mild deep tenderness but no other significant findings. The patient underwent multiple prior clinical evaluations, including repeated screenings for sexually transmitted diseases (STDs), urinalysis, and urine cultures, all of which returned negative results. During the current hospitalization, urinalysis and urine cultures remained negative. Additionally, stool routine tests revealed no objective evidence of infectious pathogens (e.g., leukocytes, erythrocytes, or pathogenic bacteria), and the patient exhibited no clinical manifestations of GI abnormalities, such as diarrhea, bloody stools, or abdominal pain. Collectively, the patient was not considered to be infected by bacterium such as *Chlamydia trachomatis*, *Salmonella* spp, *Shigella*, *Campylobacter jejuni*, and *Yersinia enterocolitica*, or a previous GI illness prior to presentation. Bleeding and clotting times, liver function and renal function assessments were also negative. C-reactive protein (CRP) levels were slightly elevated at 14.6 mg/L, and the erythrocyte sedimentation rate (ESR) was recorded at 12.0 mm/h. Tumor markers, including a total prostate-specific antigen (TPSA) at 2.93 ng/ml and others such as AFP, CEA, and CA199, were within normal ranges. Urine exfoliative cytology and cell-detected pathology did not identify any tumor cells. CT urography depicted a different bladder lesion than that observed in the MRI, revealing a thickened, rough, and enhanced region on the left anterior bladder wall measuring 1.0×0.5 cm, with no significant abnormalities in the upper urinary tract (Fig. 2). Inflammation in the bilateral

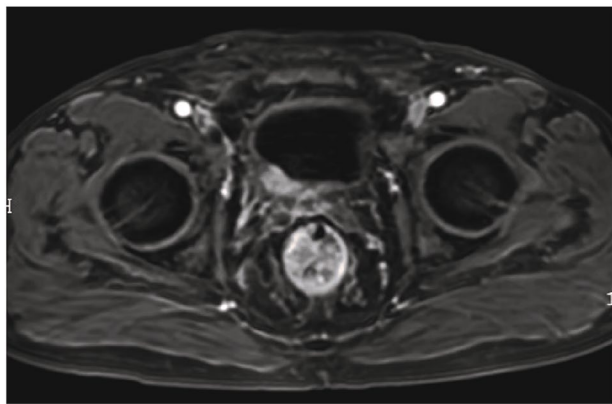


Fig. 1 Enhanced pelvic MRI image of the bladder. Enhanced pelvic MRI revealed a rough bladder wall and thickening enhanced right posterior bladder wall

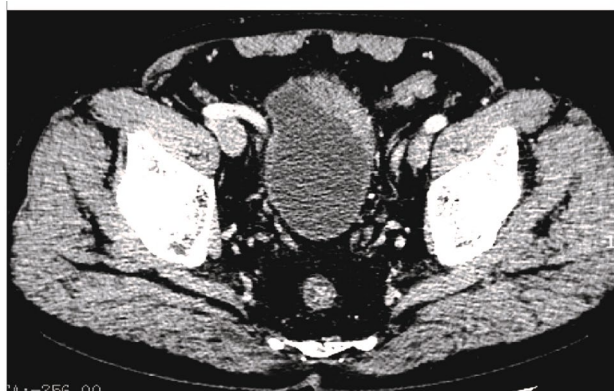


Fig. 2 CTU image of the bladder. CTU revealed the thickened, rough and enhanced region measuring 1 × 0.5 cm located on the left anterior bladder wall

sacroiliac joints suggested a potential diagnosis of SpA (Fig. 3). Further rheumatologic and immune-related testing was planned, revealing only a positive HLA-B27 expression. Cystoscopy showed a localized mucosal bulge on the left parietal bladder wall without evident neoplastic changes (Fig. 4). The lumbar MRI revealed no signs of compression in the dura mater or abnormal signals in the lumbar cord and cauda equina [5]. Consequently, a diagnostic transurethral resection of the bladder lesions, accompanied by a randomized biopsy of the bladder mucosa, was scheduled. The mucosal bulge lesion was excised deep into the muscular layer. Histopathological examination revealed chronic inflammation of both the bladder mucosa and muscle layer, characterized by a significant presence of eosinophils in the interstitium and signs of vasculitis. No neoplastic changes were identified (Fig. 5).

Given the chronic nature of symptoms, positive HLA-B27, inflammatory changes in sacroiliac joints and

characteristic inflammatory pathology in the bladder, a diagnosis of SpA was established in consultation with a rheumatologist. An interdisciplinary team, including a urologist and a rheumatologist, developed a comprehensive management and follow-up plan with the patient. Upon obtaining his consent for treatment, we initiated daily administration of NSAIDs (Etoricoxib 30 mg orally, once daily) and a Tumor Necrosis Factor (TNF) inhibitor (Adalimumab 40 mg biweekly).

The follow-up protocol includes weekly evaluations of symptoms and physical condition, monthly CRP and ESR tests, quarterly urine Cell-Detect pathology examinations, cystoscopy as needed, and CT urography after six months. After one month of continuous management, the patient experienced significant relief from left lower abdominal pain (NRS 2–3) and LUTS, accompanied by a reduction in local muscle tension in the left lower abdomen and inguinal area. Inflammatory biomarkers, including CRP and ESR, returned to normal levels. Consequently, we discontinued the use of NSAIDs and maintained Adalimumab monotherapy (40 mg biweekly). At the three-month follow-up, the patient reported complete alleviation of pain (NRS 0) and LUTS. Urine Cell-Detect pathology revealed no tumor cells. CT urography, conducted at six months, showed a well-shaped bladder with no signs of thickening, roughness, or enhanced mass-like structures (Fig. 6). Thus, Adalimumab monotherapy was extended for an additional month. Over the following six months, the patient reported no discomfort, and Adalimumab was discontinued after a year of continuous management. A biannual follow-up plan was subsequently established. This case report adheres to the CARE guidelines.



Fig. 3 CTU images of bilateral sacroiliac joints. CTU also detected inflammatory changes in the bilateral sacroiliac joints

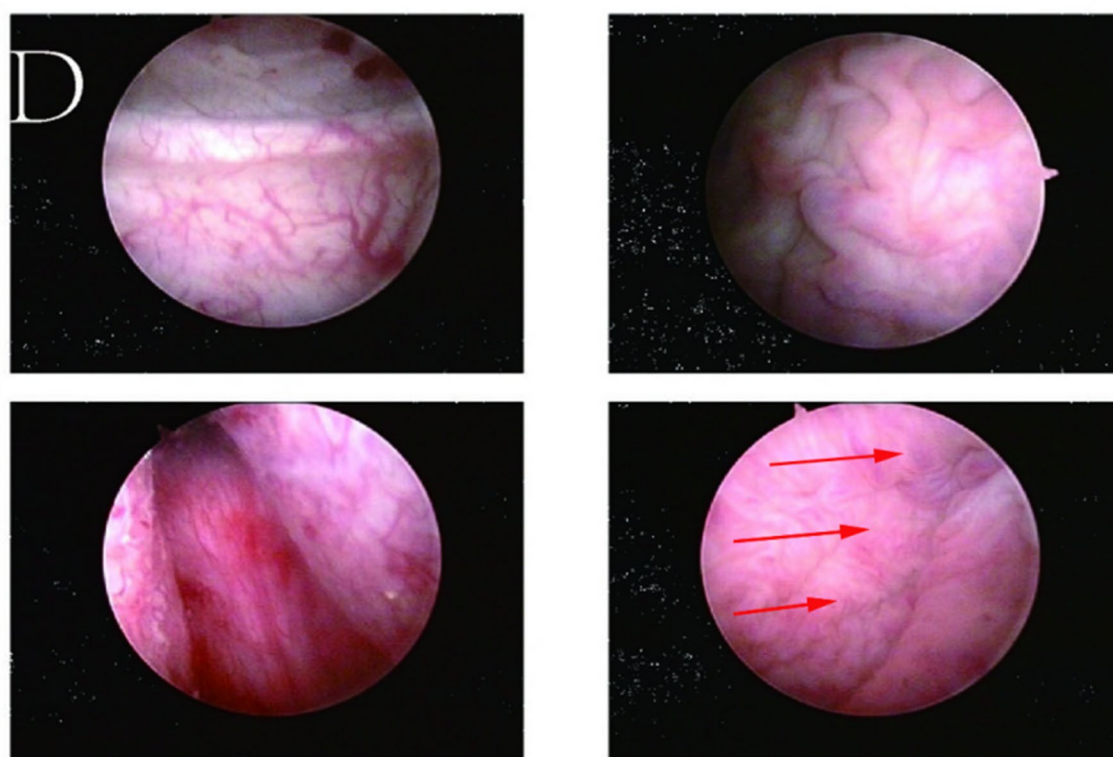


Fig. 4 Examination image of cystoscopy. Cystoscopy showed a local mucosal bulge on the left parietal wall of the bladder without obvious mucosal neoplastic changes, mild enlargement of the prostate

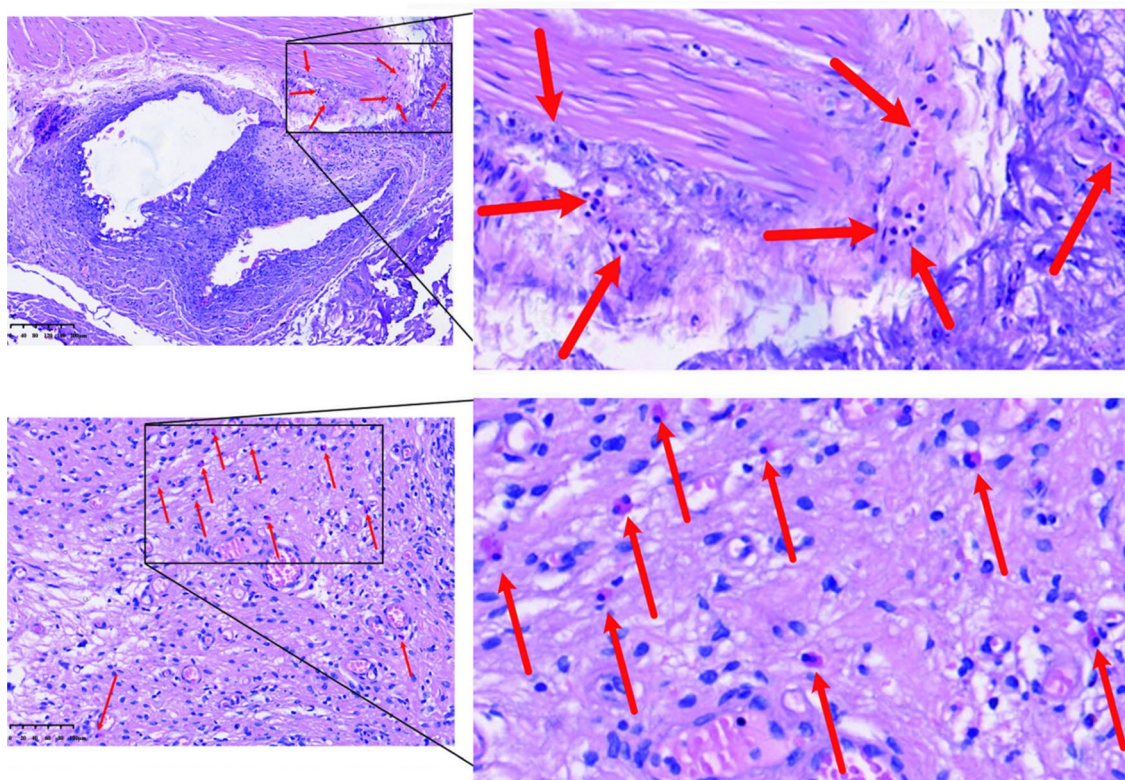


Fig. 5 Images of histopathology. Histopathological examination revealed chronic inflammation of both the bladder mucosa and muscle layer, characterized by a significant presence of eosinophils in the interstitium and signs of vasculitis. No neoplastic changes were identified

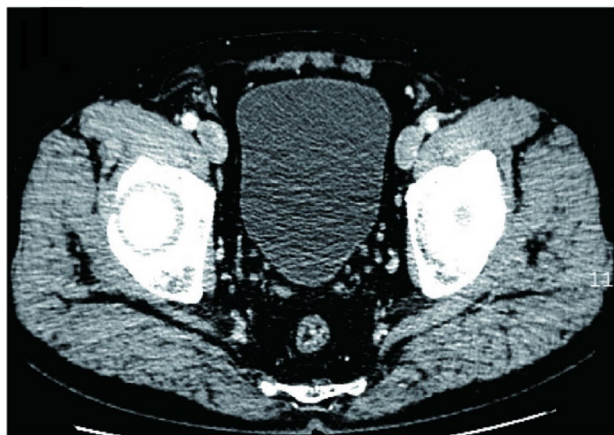


Fig. 6 CTU examination images after 6 months of treatment. Follow-up CTU, conducted six months post-management, demonstrated a well-shaped and filled bladder without any sign of a thickened, rough, or enhanced mass-like structures

Discussion and conclusion

This case report presents a unique manifestation of SpA where bladder dysfunction was the primary clinical symptom. The patient's 20-year history of LUTS and chronic lower abdominal pain, initially misdiagnosed as chronic prostatitis or cystitis [6], was ultimately attributed to SpA. Notably, the patient lacked typical SpA symptoms such as back pain or morning stiffness. The diagnosis was supported by positive HLA-B27 expression, inflammatory changes in sacroiliac joints, and histopathological findings of chronic inflammation with eosinophilic infiltration in the bladder tissue. Treatment targeting SpA led to complete resolution of urological symptoms and normalization of bladder morphology.

To our knowledge, this is the first reported case of SpA presenting primarily with bladder symptoms. While extra-articular manifestations of SpA are well-documented [1, 2], direct involvement of the urinary bladder as an initial and predominant symptom has not been previously described in the literature. This case highlights the potential for SpA to manifest in atypical ways, underscoring the need for clinicians to consider systemic inflammatory conditions when confronted with refractory urological symptoms.

This case underscores the critical importance of interdisciplinary collaboration in managing complex medical presentations. The initial misdiagnosis and ineffective treatments highlight the challenges faced when symptoms cross traditional specialty boundaries. The successful diagnosis and management were achieved through collaboration between urology and rheumatology, emphasizing the need for a holistic approach to patient, especially in cases of chronic, treatment-resistant symptoms.

The differential diagnosis for patients with refractory LUTS and chronic pelvic pain requires a systematic, stepwise approach to balance diagnostic feasibility and accuracy, particularly when advanced testing (e.g., HLA-B27, biopsies) is unavailable. Our proposed framework includes: (1) Initial Evaluation: ① Clinical History: Duration, triggers, and progression of LUTS (e.g., urinary frequency, urgency, nocturia). Pain characteristics (location, timing, relation to bladder filling/voiding). Allergy history, autoimmune disorders, or prior infections. ② Basic Investigations: Urinalysis, urine culture, and cytology to exclude infection/malignancy. Blood tests: CBC (eosinophil count), CRP, ESR, and renal function. (2) Imaging and Non-Invasive Testing: ① Pelvic MRI/CT: To assess structural abnormalities (e.g., bladder wall thickening, masses) and rule out pelvic floor dysfunction or musculoskeletal causes. ② Ultrasound: Evaluate for hydronephrosis, bladder residuals, or urothelial lesions. (3) Key Differentiators for Overlapping Conditions: 3.1 Interstitial Cystitis (IC): ① Diagnostic Clues: Symptom correlation with bladder filling (worsening pain relieved by voiding). Cystoscopy Findings: Glomerulations (petechial hemorrhages) after hydrodistension or Hunner's ulcers (patches of inflamed mucosa). Exclusion of mimics (e.g., infection, malignancy). ② Limitations: Requires cystoscopy under anesthesia, which may not be universally accessible. 3.2 Eosinophilic Cystitis (EC): ① Diagnostic Clues: History of allergic diseases (e.g., asthma, eczema) or parasitic infections. Laboratory Findings: Peripheral eosinophilia on CBC. ② Cystoscopy: Polypoid or ulcerative lesions, typically localized to the bladder dome or trigone. ③ Histopathology: Dense eosinophilic infiltration in bladder mucosa/submucosa (definitive diagnosis). 3.3 Spondyloarthropathy (SpA)-Associated Cystitis: ① Diagnostic Clues: Concurrent axial/peripheral arthritis, enthesitis, or extra-articular manifestations (e.g., uveitis). ② Sacroiliac joint imaging (MRI/CT) for axial involvement. ③ HLA-B27 testing if accessible (supports but does not confirm diagnosis). (4) Pragmatic Diagnostic Algorithm: ① Exclude infection, stones, and malignancy via urinalysis, imaging, and cytology. ② For allergy-prone patients or eosinophilia, prioritize EC evaluation (CBC, cystoscopy if indicated). ③ In patients with pain linked to bladder filling/emptying, pursue cystoscopy for IC (if feasible). ④ For those with systemic inflammatory features (e.g., arthritis, elevated CRP), investigate SpA (imaging, HLA-B27). (5) Therapeutic Trials as Diagnostic Tools: ① IC: Response to intravesical therapies (e.g., heparin, hyaluronic acid). ② EC: Trial of corticosteroids/antihistamines with monitoring of eosinophil counts. ③ SpA: Improvement with NSAIDs or biologics (e.g., TNF inhibitors). While HLA-B27 and biopsies provide definitive insights, resource constraints necessitate reliance on clinical phenotyping, targeted imaging, and therapeutic

response. Multidisciplinary collaboration (urology, rheumatology) is critical to optimize diagnostic accuracy and avoid prolonged diagnostic odysseys. This approach aligns with our case management strategy, emphasizing adaptability to local diagnostic capabilities.

There may be a certain number of similar patients in the clinic. Urologists should think more about these patients suffering lower abdominal pain and LUTS, especially when the treatment of CPPS fails and symptoms recur in a short time. The exact mechanism by which SpA affects the bladder remains unclear. The histopathologic manifestations of eosinophilic infiltration and vasculitis in bladder tissue are consistent with the known pathophysiology of SpA, suggesting that the mechanism may be related to dysregulation of the innate and adaptive immune response. However, Further researches and clinical studies are needed to uncover the underlying mechanisms.

As a single case report, this study has inherent limitations in terms of generalizability. It is unclear whether bladder involvement is a rare manifestation of SpA or an under-recognized phenomenon. Additionally, the long-term prognosis and potential for symptom recurrence remain uncertain and warrant further follow-up.

Abbreviations

SpA	Spondyloarthritis
LUTS	Lower urinary tract symptoms
NSAIDs	Nonsteroidal anti-inflammatory drugs
TNF	Tumor necrosis factor
NRS	Numerical Rating Scale
CT	Computerized tomography
PRV	Post-void residual volume
MRI	Magnetic resonance imaging
CRP	C-reactive protein
ESR	Erythrocyte sedimentation rate
TPSA	Total prostate-specific antigen

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None.

Author contributions

LMS and GBW were responsible for the conception and design of the work. LMS, SDL and YJJ conducted the acquisition, analysis and interpretation of data for the work. LMS performed drafting the article. SDL and GBW reviewed it critically for important intellectual content. GBW provided project administration and supervision. All authors contributed significantly to the work and approved the final version of the manuscript for submission.

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Data availability

All data generated or analysed during this study are provided within the manuscript or supplementary information files.

Declarations

Ethics approval code and issuing institution(s)

Written informed consent had been obtained from patient prior to submission of case report. The Ethics Committee of Ningbo Yinzhou No 2 Hospital, Zhejiang, China granted approval for this report (2022-P-016).

Consent for publication

Verbal and written consent had been obtained from the patient reported in this article for publication.

Study registration

Not applicable.

Competing interests

The authors declare no competing interests.

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