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Primary Prostatic Lymphoma Presenting as Suspected Complicated Prostatitis

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Abstract

Primary prostatic lymphoma is a rare extranodal non-Hodgkin lymphoma that frequently mimics more common prostatic conditions, leading to diagnostic delay. We report the case of a 50-year-old man with HLA-B27 positive ankylosing spondylitis receiving adalimumab who presented with a six-week history of suprapubic pain, fever, and weight loss. Initial investigations demonstrated a normal prostate-specific antigen (PSA) level and a urine culture positive for *Klebsiella* species. Computed tomography findings were initially interpreted as severe prostatitis with possible abscess formation. However, persistent symptoms despite antibiotic therapy and subsequent magnetic resonance imaging demonstrated extensive solid infiltrative disease involving the prostate, seminal vesicles, bladder base, and pelvic sidewalls with associated lymphadenopathy, raising suspicion for malignancy. Transperineal prostate biopsy revealed diffuse large B-cell lymphoma (DLBCL), activated B-cell subtype, with immunohistochemistry positive for CD20, PAX5, BCL2, MUM1, and MYC expression, and a Ki67 proliferative index of 80% - 90%. Staging investigations excluded distant extranodal disease, supporting the diagnosis of primary prostatic lymphoma. The patient was referred for haematology management and commenced on rituximab-based chemotherapy. This case highlights the importance of reconsidering the diagnosis in patients with presumed prostatitis who fail to improve with appropriate antimicrobial therapy, particularly when PSA levels remain normal despite extensive prostatic disease.

Keywords

Prostatic Lymphoma, MRI Prostate, Prostatitis, Adalimumab

1. Introduction

Primary lymphoma of the prostate is a very rare entity within prostatic neoplasms

and extranodal non-Hodgkin lymphoma (NHL). It accounts for less than 0.1% of all prostate malignancies and represents an uncommon site of extranodal lymphoid malignancy, often leading to challenges in recognition and diagnosis [1]. The most frequently reported histological subtype in this location is diffuse large B-cell lymphoma (DLBCL), although other subtypes have been described (e.g. marginal zone lymphoma, follicular lymphoma) [2]. Patients typically present in the fifth to seventh decade of life, but cases have been documented at younger ages, underlining the broad clinical spectrum of this disease [3]. Clinically, primary prostatic lymphoma often presents with non-specific lower urinary tract symptoms such as dysuria, urinary frequency, or retention, which can closely mimic benign prostatic hyperplasia, complicated prostatitis, or carcinoma [4]. Serum prostate-specific antigen (PSA) levels are frequently normal or only mildly elevated, reflecting the non-epithelial origin of the tumour and limiting its utility in diagnosis [5]. Radiological findings are typically non-specific, and without a high index of suspicion, the diagnosis may be delayed [6] [7]. Prostate biopsy with histopathological and immunohistochemical evaluation remains the gold standard for diagnosis, as demonstrated in multiple case reports and retrospective series [6]. Given its rarity, there is no standardized management pathway, but treatment most often follows systemic lymphoma protocols (such as R-CHOP), similar to nodal DLBCL, with combined chemo-immunotherapy demonstrating favourable outcomes in reported cases [7]. Some reports also highlight the role of radiotherapy and the potential variability in clinical behaviour depending on subtype and stage [8] [9]. We describe a case of primary prostatic DLBCL presenting with features initially suggestive of complicated prostatitis, underlining the importance of considering lymphoma in the differential diagnosis of atypical or refractory presentations.

2. Case Presentation

A 50-year-old male with a past medical history of HLA B27-positive ankylosing spondylitis (managed with adalimumab) and hypertension, presented with a six-week history of suprapubic pain, fever, and unintentional weight loss. Examination revealed a tender prostate on digital rectal examination.

Laboratory investigations demonstrated a PSA of 1.07 ng/mL, eGFR 60, CRP 30 mg/L, and WCC 5.5 × 10^9/L; urine culture grew *Klebsiella* species and the patient was treated with ciprofloxacin, although symptoms persisted. CT scan (**Figure 1**) revealed marked periprostatic fat stranding, low-density lesions in the peripheral zone of the prostate and seminal vesicles, and inflammatory extension to both pelvic sidewalls and the bilateral psoas muscles, features reported as consistent with severe prostatitis with possible abscess formation. Subsequent MRI of the prostate (**Figures 2-4**) demonstrated extensive solid tumour-like replacement of the prostate with marked T2 signal loss and diffusion restriction, with direct invasion into the bladder base, seminal vesicles, and pelvic sidewalls, and widespread pelvic and mesorectal lymphadenopathy; these findings were regarded as

more suggestive of malignancy than infection. A transperineal biopsy and aspiration was done under local anaesthesia. The aspiration was unsuccessful due to the solid nature of the tissue. Biopsies revealed diffuse infiltration by atypical pleomorphic lymphoid cells (Figure 5), positive for CD20 (Figure 6), PAX5, BCL2, MUM1, and MYC (60%), with a Ki67 proliferative index of 80% - 90% (Figure 7), and negative for epithelial markers (AE1/AE3, CK8/18) and for CD10, BCL6, and Cyclin D1. EBER was negative, and FISH analysis did not demonstrate MYC rearrangement. The overall features were diagnostic of diffuse large B-cell lymphoma (DLBCL), not otherwise specified, activated B-cell subtype. LDH was at 880 U/l, Baseline viral screening for hepatitis B, hepatitis C, and HIV was negative.

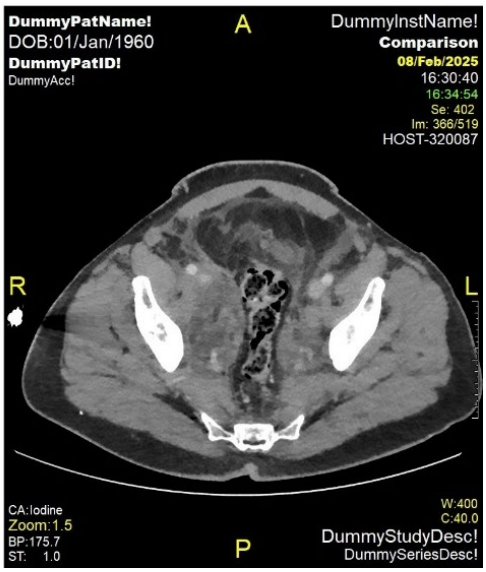

Figure 1. Axial CT showing the bilateral mixed fatty and soft tissue pelvic side wall masses.

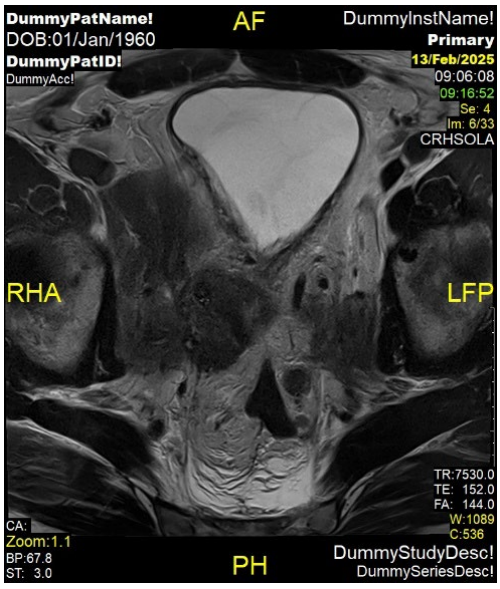

Figure 2. Axial T2 MRI through the pelvis showing bilateral low signal pelvic masses.

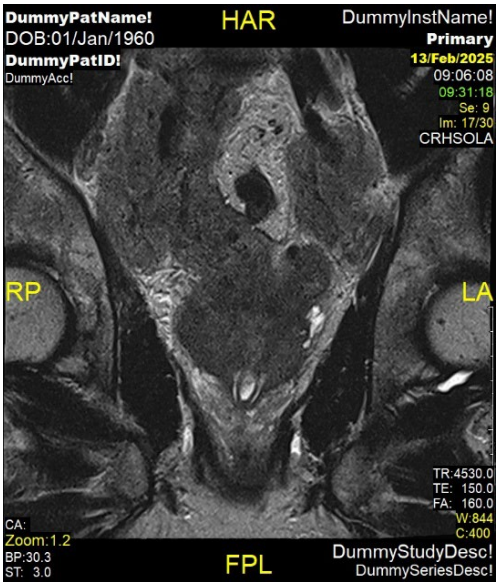

Figure 3. Coronal T2 MRI of pelvis showing prostate mass extending into both pelvic sidewall masses.

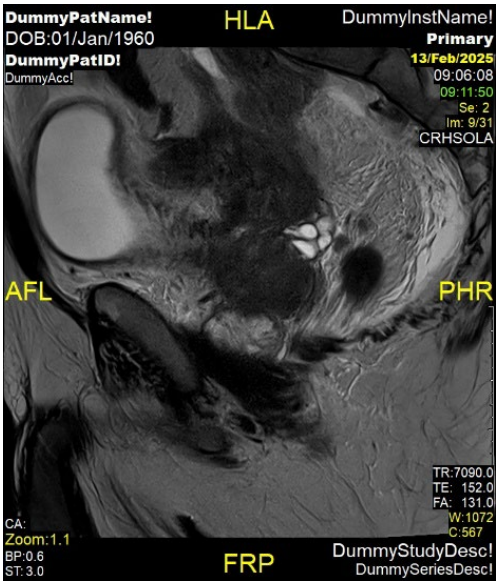

Figure 4. Sagittal T2 of pelvis showing prostate in continuity with large pelvic sidewall mass and ureteric involvement.

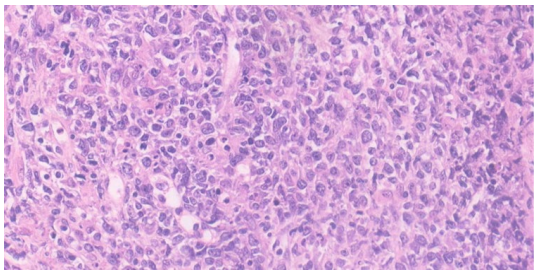

Figure 5. The lymphoid cell infiltrate features a large centrocytic and centroblastic morphology.

Figure 6. The large lymphoid cells feature a diffuse CD20 expression.

Figure 7. Ki67 shows 80 - 90 percent of cells in the cell cycle.

3. Discussion

Primary prostatic lymphoma (PPL) is an exceptionally rare diagnosis, representing fewer than 0.1% of all prostate malignancies and an equally rare subtype of extranodal non-Hodgkin lymphomas—this rarity frequently contributes to delayed recognition and misdiagnosis in clinical practice [10] [11]. Patients often present with non-specific lower urinary tract symptoms (LUTS) such as dysuria, urinary frequency, retention, or hematuria, which closely mimic benign prostatic hyperplasia (BPH), prostatitis, or prostate carcinoma [12] [13]. The positive urine culture for *Klebsiella* species was initially interpreted as supportive of bacterial prostatitis. However, the lack of clinical improvement despite appropriate antimicrobial therapy, combined with progressive infiltrative imaging findings, suggested that bacteriuria may have represented either secondary colonisation or concurrent infection rather than the primary pathological process. This contributed to early diagnostic anchoring toward infection and delayed consideration of malignancy.

Diffuse large B-cell lymphoma (DLBCL) is the predominant histological subtype identified in PPL, but other rare variants including MALT lymphoma have also been described, underscoring the histopathological spectrum of the disease [14]. Clinically, systemic "B-symptoms" (fever, weight loss, night sweats) are uncommon at presentation, further reducing early suspicion for lymphoma [12].

The diagnosis was classified as primary prostatic lymphoma based on predominant prostatic involvement at presentation, with clinical manifestations arising principally from the prostate and adjacent pelvic structures, and no evidence of disseminated nodal or extranodal lymphoma on staging investigations. This approach is consistent with established diagnostic criteria for primary prostatic lymphoma described in previous literature. A bone marrow assessment was not per-

formed and PET CT was not available at the time of reporting.

In the present case, the combination of systemic inflammatory features, normal prostate-specific antigen (PSA), and imaging suggestive of diffuse prostatitis initially favoured a diagnosis of complicated bacterial prostatitis. This is understandable as epithelial prostate malignancies typically present with elevated PSA and focal structural abnormalities, unlike prostatic lymphomas which may cause significant gland enlargement with minimal PSA elevation [10] [15]. PSA is derived from prostatic epithelial cells, and its interpretation can be misleading in non-epithelial malignancies such as lymphoma, illustrating a key diagnostic pitfall [6]. Cross-sectional imaging in PPL may demonstrate homogeneous glandular enlargement and diffuse tissue infiltration without clearly defined abscess or focal tumor mass, which can resemble inflammatory disease on conventional modalities [10] [12].

Lack of clinical and biochemical improvement with broad-spectrum antibiotics, coupled with radiological features disproportionate to infection severity, should prompt reassessment of the working diagnosis. Definitive diagnosis requires histopathological and immunohistochemical confirmation via prostate biopsy, which remains the gold standard in cases where clinical suspicion persists despite non-specific imaging and laboratory findings [12] [13].

This patient was referred to the haematologists, received Rituximab, Polatuzumab, Cyclophosphamide, Doxorubicin and Prednisolone 6 cycles. He developed had residual mass in psoas muscle for which he received radiotherapy.

Management of PPL typically aligns with protocols for systemic DLBCL, with rituximab-based combination chemotherapy (e.g., R-CHOP) forming the backbone of treatment. Several case series and reports demonstrate favourable outcomes with this approach, sometimes in combination with local radiotherapy for symptom control or bulky disease [11] [12]. However, evidence remains limited due to the disease's rarity and the absence of randomized trials.

An important aspect in this case was the history of the patient's tumour necrosis factor-α (TNF-α) inhibitor exposure. While TNF-α inhibitors are effective in chronic inflammatory diseases, their relationship with lymphoma risk is complex. Meta-analyses of randomized trials and observational studies suggest either no significant association or conflicting findings regarding increased lymphoma risk with TNF-α inhibitors compared with conventional therapy, highlighting ongoing debate and methodological limitations in this literature [12] [13]. Some population-based studies outside rheumatology have reported modest associations between TNF-α inhibitor use and hematologic malignancies, though confounding by underlying inflammatory disease activity remains a significant challenge [15]. Consequently, although causality cannot be established in a single case, clinicians should remain vigilant for atypical malignancies in patients receiving long-term TNF-α blockade.

Furthermore, a modest increase in the relative risk of haematopoietic and lymphoid malignancies has been reported in patients with ankylosing spondylitis, although findings across studies remain inconsistent [16]. The pathophysiological

basis of this association is not definitively established; however, the most biologically plausible explanation centres on chronic immune activation. Ankylosing spondylitis is characterised by sustained immune activation and systemic inflammation, with elevated pro-inflammatory cytokines contributing to oxidative stress and increased cellular turnover. This milieu promotes chronic B-cell activation and repeated germinal centre reactions, processes that inherently involve somatic hypermutation and programmed DNA double-strand breaks. An increased frequency of such DNA breaks heightens the probability of oncogenic mutations and malignant transformation. Over time, the cumulative effect may facilitate the acquisition of mutations such as BCL6 rearrangements, MYC translocations, and disruption of the p53 pathway, which are characteristic of diffuse large B-cell lymphoma (DLBCL). Finally, the risk of lymphoma development may be further potentiated by treatment with adalimumab, given that TNF-α plays a recognised role in tumour surveillance and apoptotic regulation; its inhibition may theoretically impair anti-tumour immune mechanisms [15] [16].

In summary, PPL illustrates the diagnostic complexity of rare non-epithelial prostatic malignancies. Persistent, atypical, or antibiotic-refractory prostatitis warrants prompt reassessment, and clinicians should not be reassured by a normal PSA in the context of progressive disease. Early MRI and timely histological sampling are essential in diagnostically ambiguous cases, as delayed recognition of lymphoma can preclude timely curative-intent therapy.

Written informed consent for publication of clinical details and imaging was obtained from the patient.

4. Conclusion

Primary prostatic DLBCL is an uncommon but important differential diagnosis in patients presenting with refractory prostatitis-like symptoms, especially when imaging reveals extensive solid infiltration with lymphadenopathy. Multidisciplinary evaluation and early biopsy are essential to avoid diagnostic delay and initiate appropriate systemic therapy.

Conflicts of Interest

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

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