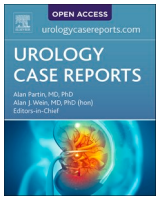
Urology Case Reports 54 (2024) 102714

Contents lists available at ScienceDirect

Urology Case Reports

journal homepage: www.elsevier.com/locate/eucr



Inflammation and infection

Eosinophilic cystitis mimicking bladder tumor: A case report

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ARTICLE INFO

Keywords:
Eosinophilic cystitis
Eosinophilia
Bladder neoplasia
Dysuria
Gross hematuria

ABSTRACT

Eosinophilic cystitis (EC) is a rare inflammatory condition characterized by eosinophilic infiltration into the bladder wall. It often presents symptoms common to urological issues such as urinary tract infections, hematuria, bladder stones, or bladder neoplasms. Here, we describe a case of a 44-year-old male veteran with a history of multiple tuberculosis episodes who presented to the Emergency Department with dysuria, suprapubic pain, and gross hematuria.

Initial imaging and cystoscopy concerned bladder neoplasia; however, subsequent pathological evaluation showed EC. This case underscores the importance of considering EC in the differential diagnosis of bladder tumors, especially when imaging describes bladder wall thickening in a patient without risk factors for bladder malignancy.

1. Introduction

Eosinophilic cystitis is an uncommon inflammatory bladder disease. Due to its rarity and nonspecific symptomatology, EC often poses a diagnostic challenge. Most cases of EC present with mucosal lesions of the bladder, and only a few cases have been reported as mimicking malignancies.[1–3]

Its etiology remains poorly understood; however, it has been associated with various factors, including allergies, asthma, atopy, peripheral eosinophilia, bladder trauma, open bladder surgery, transurethral resection of the bladder tumor, intravesical treatments (mitomycin-C, thiotepa, Bacillus-Calmette-Guerin (BCG)), medications (e.g., sulfonamides, warfarin, penicillin), parasitic infections, eosinophilic enteritis, recurrent urinary tract infections, vesicoureteral reflux, chronic granulomatous disease, surgical sutures, and certain foods (such as tomatoes, coffee, and carrots). To date, only a few of these associations have been verified.[1,4]

2. Case presentation

A 44-year-old male presented to the emergency department (ED) with a one-week history of dysuria, suprapubic pain, and gross hematuria. He is a veteran with a past medical history of kidney stones, post-traumatic stress disorder (PTSD), traumatic brain injury, and several episodes of Tuberculosis contracted during his military service that have been treated. He denied any smoking history or exposure to industrial chemicals. At the time of presentation, he was taking Acetaminophen, Buspirone, Celecoxib, Cyclobenzaprine, Famotidine, Pantoprazole, Prazosin HCl, Quetiapine, Sertraline, Sumatriptan and Topiramate.

Clinical examination in the ED was unremarkable except for the symptoms mentioned. He denied fever, chills, or weight loss. A bladder ultrasound reported diffuse abnormal thickening of the bladder's right lateral wall, with no signs of hydronephrosis or hydroureter. An abdomen and pelvis CT scan identified a thickened bladder mass concerning malignancy (Fig. 1).

Urinalysis reported leukocytosis and red blood cells with negative nitrites. A complete blood count (CBC) showed eosinophilia at 7.7% and absolute eosinophil count elevated at 0.6 x10^9/L (Table 1). The patient was initiated on intravenous fluids, Ceftriaxone, and Azo Urinary Pain Relief, which contains 97.5 mg of Phenazopyridine Hydrochloride per tablet for symptomatic management.

The following week, a cystoscopy showed a sessile-appearing mass, with bullous changes and hypervascularity extending from the right trigone to the right posterior wall and involving the entire right lateral wall. The lesion appeared to involve the area around the right ureteral orifice (UO), which was not visualizable. Meanwhile, the left UO was found in its orthotopic position without abnormalities. The patient subsequently underwent transurethral resection of the bladder tumor (TURBT) with right ureteral stent placement; recovery after surgery was uneventful.

Despite the tumor-like appearance, histopathological findings confirmed the presence of benign urothelium and submucosa with

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https://doi.org/10.1016/j.eucr.2024.102714
Received 13 March 2024; Accepted 17 March 2024
Available online 22 March 2024

overlapping features of polypoid and eosinophilic cystitis (Fig. 2). Following the diagnosis, the patient was referred to rheumatology and allergy medicine to explore potential underlying systemic conditions contributing to the observed bladder pathology. However, all tests for rheumatoid disease returned negative, and the allergy consultation concluded that the patient's eosinophil levels were not elevated enough to indicate a myeloproliferative disorder or systemic eosinophilic disorder, thus ruling out a systemic reaction. Additionally, the patient has no family history of such conditions.

3. Treatment and evaluation

A follow-up CBC performed five months after tumor resection revealed a normalized eosinophil level of 3.8% and a normalized absolute eosinophil count of 0.2 x10^9/L. Parasite testing returned negative.

Follow-up cystoscopy demonstrated no evidence of tumor recurrence, with the bladder displaying normal texture within the trigone region. However, a small mound or nodule was noted at the right UO. Ureteral orifices were identified bilaterally without obstruction.

The patient's symptoms have improved; he denies pain, dysuria, or gross hematuria since the initial presentation. Nonetheless, mild symptoms of cystitis with urgency were reported during the follow-up assessment. Antihistamine therapy was initiated, and the patient is scheduled for CBC monitoring every six months.

The rheumatology, allergy, and urology services did not recommend steroid therapy in consideration of the patient's clinical course. This decision was based on his improvement in CBC parameters post-tumor resection and the absence of systemic symptoms.

4. Discussion

This case highlights the complex nature of EC, often confused with malignant bladder lesions due to its presentation and imaging findings. Upon reviewing the patient's medical history, two possible risk factors contributing to EC can be identified: multiple episodes of tuberculosis contracted during military service and the long-term use of Celecoxib. Although the exact cause remains undetermined, the association with drug-induced hypersensitivities presents a plausible etiological pathway.[5,6]

There have been case reports of eosinophilic gastroenteritis and eosinophilic pneumonia associated with long-term use of Celecoxib. This selective cyclo-oxygenase-2 inhibitor is a diaryl-substituted pyrazole derivative containing a sulfonamide substituent. Additionally, the antibiotic regimen for tuberculosis treatment, involving Rifampin and sulfa drugs, has been associated with interstitial nephritis, which can contribute to bladder mucosal inflammation.[6]

Table 1
Patient's complete blood count results on presentation to the emergency department.

Component	Lab Value	Reference Range & Units
WBC	7.8	4.67–9.11 x10^9/L
Erythrocytes	5.21	4.07–5.6 x10^{12}/L
Hemoglobin	16.8 (High)	12.7–16.3 g/dL
Hematocrit	48.5	39.4–49.2 %
MCV	93.1	83.7–99.0 fL
MCH	32.2	26.9–32.9 pg
MCHC	34.6	30.8–34.6 g/dL
RDW	12.0	11.2–15.1 %
Platelet Count	197	142 - 396 x10^9/L
Mean Platelet Volume	8.9	8.5–12.3 fL
Neutrophils %	57.8	43.3–71.5%
Lymphocytes %	24.5	18.8–43.0 %
Monocytes %	9.0	2.2–12.8 %
Eosinophils %	7.7 (High)	0.0–5.2 %
Basophils %	0.4	0.0–1.2 %
Neutrophils #	4.47	2.2–5.7 x10^9/L
Lymphocytes #	1.9	1.1–3.2 x10^9/L
Monocytes #	0.7	0.3–0.8 x10^9/L
Eosinophils #	0.6 (High)	0.0–0.3 x10^9/L

WBC: white blood cell count; MCV: mean corpuscular volume; MCH: mean corpuscular hemoglobin; MCHC: mean corpuscular hemoglobin concentration; RDW: red blood cell distribution width; L: liter; g/dL: gram per deciliter; fL: femtoliter; pg: picogram.

Eosinophilic cystitis is often reported as a transmural lesion with eosinophilic infiltrates. It was first reported in 1960, and about 21 cases have been documented in the last five years. It can occur at any age and presents with various clinical manifestations. The etiology is unclear and is generally considered to be related to an allergic reaction.[1,7]

In type 1 hypersensitivity, IgE and IgA induce eosinophils to release Major Basic Protein (MBP), which increases the inflammatory reaction, as observed in eosinophilic cystitis. Consequently, the thickening of the bladder wall mucosa is due to fibrosis and inflammation. Increased MBP and other cationic protein concentrations correlate with atopic diseases, parasitic infections, and eosinophil-associated inflammatory processes.[8]

In this case, EC resembles a bladder tumor, but the difference in imaging is not pronounced.[9] Most literature describes images showing thickening of the bladder wall or tumor-like masses.[9,10] Another finding in this case is that laboratory tests showed microhematuria due to mucosal inflammation and significant eosinophilia.[11] Although it cannot be definitively stated that the patient's EC was caused by the medications taken, this aspect is noteworthy given the drug's potential for eliciting allergic reactions, which could precipitate conditions like eosinophilic cystitis.

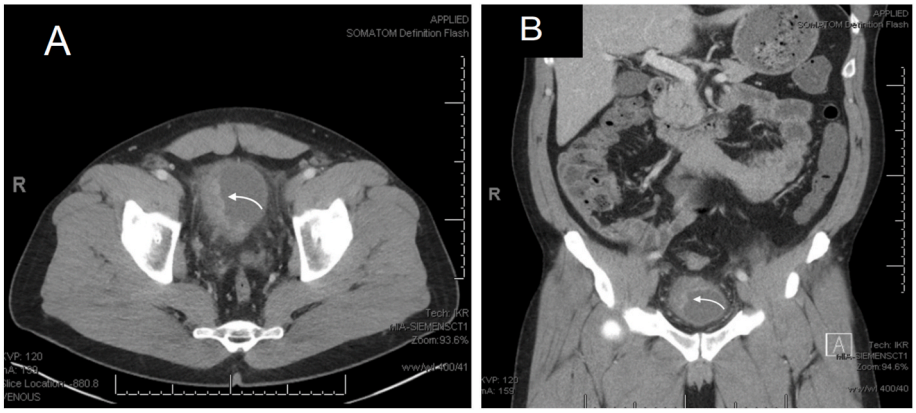

Fig. 1. A) Pelvic axial CT scan showing diffuse abnormal thickening of the bladder's right lateral wall (arrow). B) Abdominal and pelvic coronal CT scan showing diffuse abnormal thickening of the bladder's right lateral wall (arrow)
CT: computed tomography; R: right.

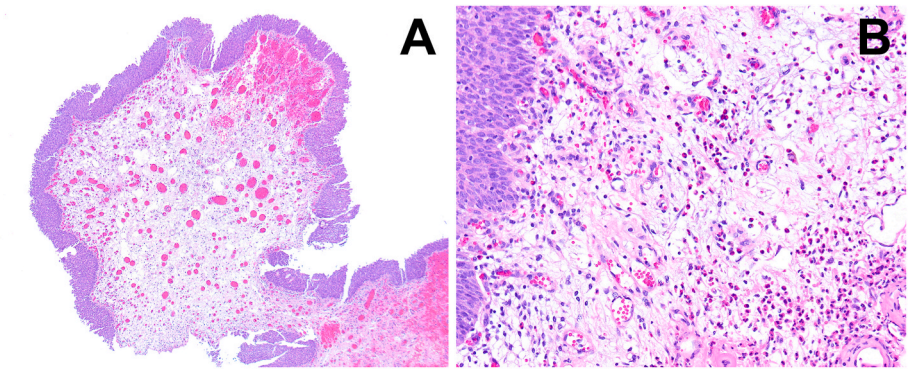

Fig. 2. A) Low-power magnification showing edematous polypoid projects of benign urothelial mucosa with congestion of the vessels in the submucosa (i.e. polypoid cystitis). B) High-power magnification showing numerous eosinophils in the submucosal underlying benign urothelium (i.e. eosinophilic cystitis).

5. Conclusion

Eosinophilic cystitis represents a diagnostic challenge due to its rarity and the overlap presentation with bladder cancer. In clinical practice, it is frequently underdiagnosed or misdiagnosed and should be included in a differential diagnosis, especially in patients with atypical history of bladder malignancy. This case accentuates the necessity for thorough histopathological examination in patients presenting with unusual bladder lesions, particularly when there is a background of potential eosinophil-inducing factors. A multidisciplinary approach, integrating urological, rheumatological, and allergological evaluations, is crucial for accurate diagnosis and effective management of this elusive condition.

CRediT authorship contribution statement

Laura Angulo-Llanos: Investigation, Resources, Writing – original draft. **Ruben Blachman-Braun:** Writing – review & editing. **Thomas A. Masterson:** Supervision.

Declaration of competing interest

The authors declare no conflict of interest.

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