

EOSINOPHILIC CYSTITIS- A DISTINCT CLINICO-PATHOLOGICAL ENTITY

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Eosinophilic cystitis is an uncommon poorly understood clinicopathological condition first described in 1960[1,2]. It usually cause irritative voiding symptoms and can sometimes simulate infiltrative vesical malignancy.

A 58-year old male presented with difficulty in voiding and slow stream of urine for 3 years and dysuria for 2 years. He also had increased frequency with twenty times during the day and nocturnal enuresis for last 6 months. Patient was a known hypertensive on irregular treatment with no other significant past history.

General physical examination was essentially unremarkable except for pallor.

Per abdomen examination showed no organomegaly or ascites. Bladder was palpable. Other systemic examination was normal. On per rectal examination, prostate was enlarged (approximately 40 gms), smooth, firm and, non-tender.

Cystoscopy showed grade I trabeculations in bladder. Both the uretric orifices were normal. Baseline and other relevant investigations were done, findings of which are available in table 1.

Table1: Laboratory findings

Test	Patient's value
Hemoglobin	9.2 gm/dl
PCV	28.9 %
Total leucocyte count	8670 cells/cumm
Differential leucocyte count	Polymorphs: 80%; Eosinophils:1%; Lymphocyte:14%; Monocyte :5%
PSA	1.44 ng/ml
Serum iron	29 µg/dl
Serum creatinine	1.7 mg/dl
BUN	15 mg/dl
Urine protein	+++
Urine sugar	Nil
Urine microscopy	Pus cells 10-15; RBC: 25-30; occasional granular cast +
Urine culture	No growth

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Perurethral catheterization done drained one litre of urine. A diagnosis of voiding dysfunction with chronic urinary retention, anaemia and hypertension was made. A transurethral resection of prostate and bladder biopsy was done and submitted for histopathological examination.

Histopathological examination of bladder biopsy received showed focal ulceration of the mucosa, edema, congestion, few lymphocytes and dense eosinophilic infiltrates in the lamina propria (Figure 1, 2, 3 and 4). Focal areas of hemorrhage was also noted (Figure 3). Muscularis propria showed eosinophilic infiltrates (Figure 5) and focal areas of fibrosis (Figure 6). Histopathological diagnosis of eosinophilic cystitis was made. Prostatic tissue received showed features of benign prostatic hyperplasia.

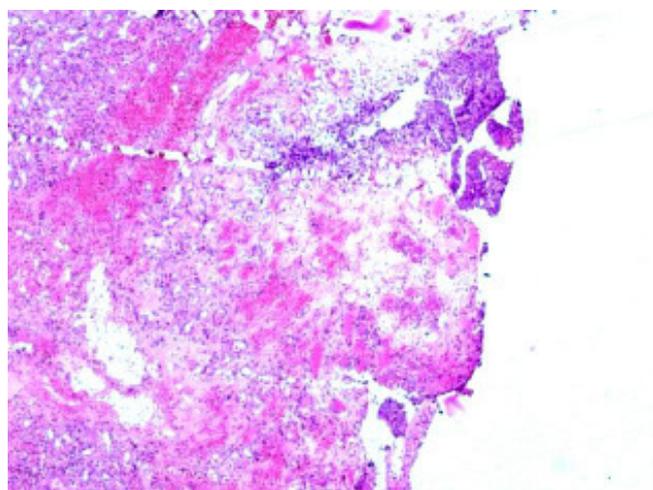


Figure 1: Deep bladder biopsy with denuded epithelium, mucosal edema and infiltration by eosinophils into lamina propria as well as muscularis propria (H&E X 20)

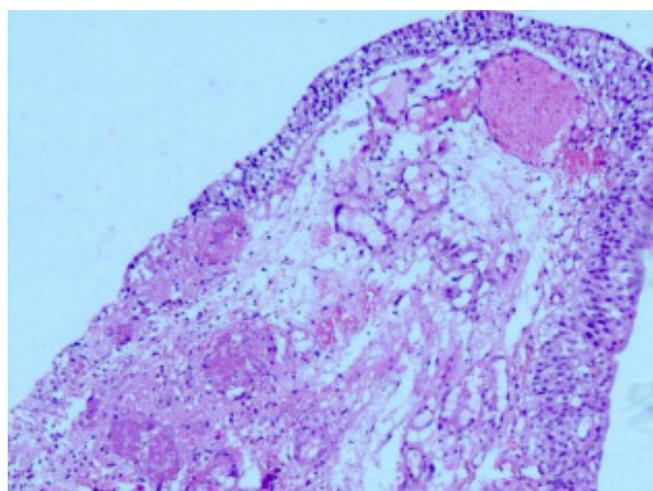


Figure 2: Superficial section of bladder biopsy with focal ulceration of the epithelium, edema and congestion in lamina propria (H&E X 40)

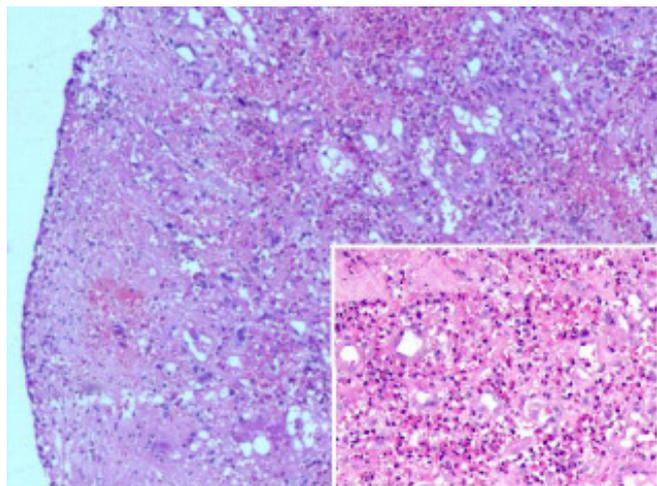


Figure 3: Ulceration of lining epithelium with dense eosinophil infiltrates (H&E X 40); inset show dense collections of eosinophils (H & E X 100)

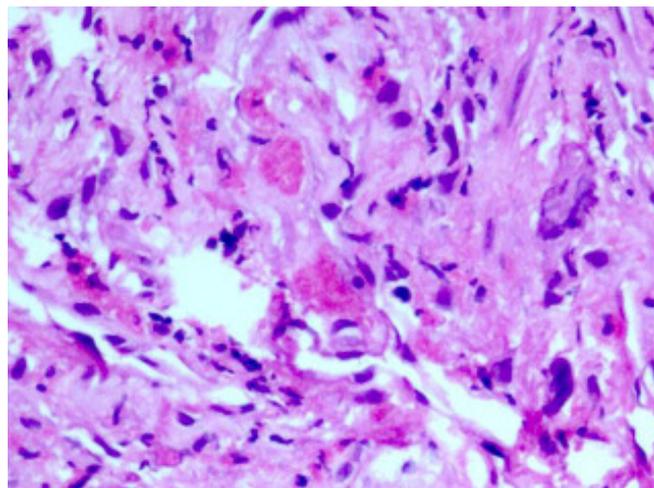


Figure 6: Pale staining areas representing fibrosis seen replacing the muscularis propria which appear darkly stained along with infiltration by eosinophils (H& E X 200)

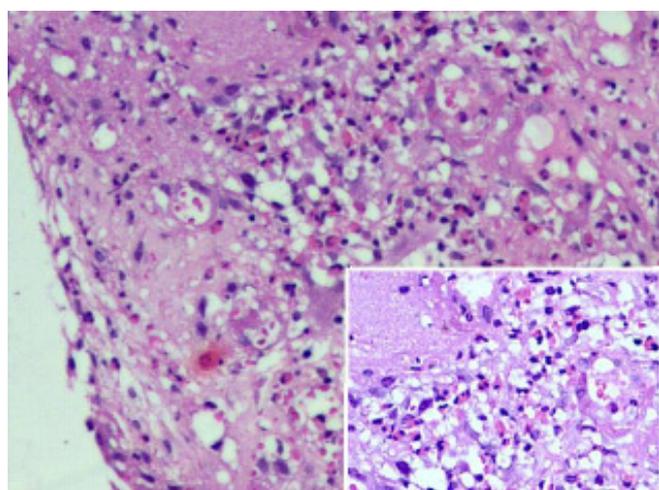


Figure 4: Bladder mucosa with edema, eosinophils and lymphocytes (H&E x 100); inset shows higher magnification of the same (H & E X 200)

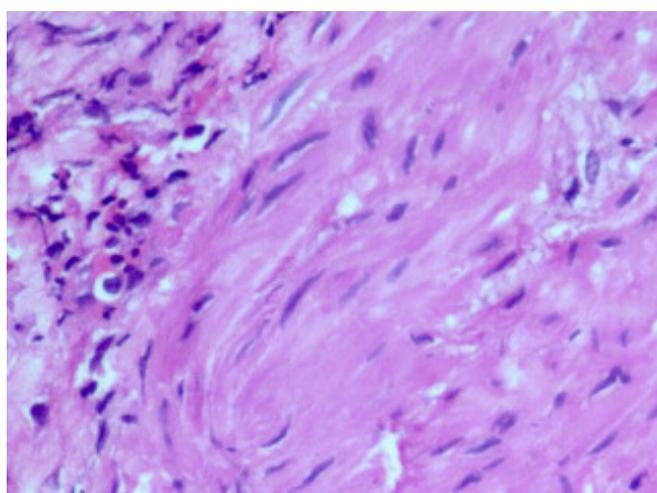


Figure 5: Sections show muscle fibres with infiltration by eosinophils (H & E X 200)

Eosinophilic cystitis, an unusual disease process presents clinically with episodes of marked dysuria, hematuria, suprapubic pain, diurnal and nocturnal urinary frequency[3]. Patient in the study case had several of these symptoms. Although mostly encountered in children and women, can also occur in men[4].

The disease has been associated with vesical injury, chronic vesical irritation, surgery, parstitis, food / drug allergy, tuberculous cystitis and malignancies[3]. Acute flare of eosinophilic cystitis has also been noticed after bladder instillation with dimethyl sulfoxide for presumed interstitial cystitis[5]. Pathogenesis of eosinophilic cystitis still remains unclear. It could possibly be caused by antigen-antibody reaction leading to production of various immunoglobulins which in turn cause activation of eosinophils that initiates inflammatory process[6]. Another reason suggested is dysregulation of cytokines predominantly involving interleukin 4 and 5 [3].

Cystoscopy findings may vary considerably with edema, erythema, ulceration to papillary lesions in bladder mucosa. Sometimes an infiltrative mass-like lesion may also be visualized[4]. Imaging studies of patients with eosinophilic cystitis are not very specific. It can show thickening of the wall and sometimes mimic tumoral mass. Other laboratory findings include proteinuria, microscopic hematuria and at times peripheral eosinophila[6]. The present case had microscopic hematuria and proteinuria without blood eosinophila.

The gold standard for diagnosing this lesion is histopathological examination of bladder biopsy[6]. It is mandatory to obtain a deep biopsy otherwise the diagnosis can be missed.

Histological changes can be acute, chronic or both. The acute changes are characterized by edema, congestion

with inflammatory infiltrate composed of eosinophils and lymphocytes. Eosinophils are more prominent in cases with muscle necrosis. The chronic stage shows few / absent eosinophils, few lymphocytes with fibrosis in the lamina propria and interspersed among superficial muscle layers. The overlying epithelium can show proliferative changes or squamous metaplasia.

There is no specific curative treatment available for this condition[6]. However, patients have responded to treatment modalities such as transurethral resection of the lesion in the bladder and combination of corticosteroids and antihistaminic. A regular follow up is essential as this lesion though self limiting, at times can recur. The knowledge of this distinct clinicopathological entity is essential for urologist since the symptoms are not very specific for the clinician to suspect this lesion and can often mimic malignancy.

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