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Eosinophilic Pyelo-Ureteritis Mimicking Urinary Calculus: A Rare Cause of Ureteric Obstruction in an Adult

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Abstract

Eosinophilic infiltration of the urinary tract is rare. We report a case of eosinophilic pyelo-ureteritis in a middle aged female presented with features of urinary stone disease. It could be due to a variety of factors, with a high chance to be overlooked. As clinical and imaging studies are not specific, high index of suspicion is necessary to the proper diagnosis and appropriate management.

Keywords

pyelitis; pyelo-ureteritis; eosinophils; ureteric obstruction

Introduction

Flank pain and hematuria are caused by multiple factors, most frequent being urinary stones. Eosinophilic pyelo-ureteritis (EP) is a rare stenosing condition of the renal pelvis and ureter, cause flank pain, hematuria and/ or unilateral hydronephrosis [1]. It is an inflammatory condition, represents a response to a variety of agents and may be overlooked. Atopy, Hypereosinophilic syndrome and prior trauma have been described as possible aetiologies [2,3,4]. As clinical and imaging studies show nonspecific findings, a high grade of suspicion is crucial to the correct diagnosis and proper management of eosinophilic pyelo-ureteritis. We report a case of Eosinophilic pyelo-ureteritis, a rare entity as a cause of ureteric obstruction.

Case Report

A 48 year old female was admitted in urology for pain in the right loin since six months and increasing pain in the last two days. There was a history of dysuria, hematuria and nausea. There was no significant past medical history or drug allergy. A provisional diagnosis of calculus in kidney or urinary tract was made. Complete urine analysis, Urine culture, Urea, Creatinine and Hemoglobin were done.

Her urine examination revealed increased pus cells of 10-12 per high power field (HPF), increased red cells (30-35/HPF), and few epithelial cells. Her Creatinine was 0.8mg/dl, Hemoglobin of 11 g/dl and urea of 32 mg/dl. Urine culture did not reveal any bacterial growth. IgE and eosinophil counts were not done.

Computerised tomography of KUB region with contrast showed gross hydronephrosis with abrupt tapering at the pyelo-ureteric junction and no evidence of calculus (Fig 1). Ultrasound correlation revealed a feeble ureteric jet with no obstructive soft tissue or calculus. Intravenous urogram was

planned, revealed right pyelo-ureteric junction obstruction (PUJ) with ballooned out right renal pelvis measuring 6.4 cm and abrupt narrowing at the PUJ. Delayed images after 2 hrs showed dense filling of the renal pelvis with contrast (Fig 2) and right ureter has not opacified with contrast. Mild calyceal dilatation and blunting of the fornices of the right kidney were noted. Left kidney was normal. There was no evidence of vesico-ureteric reflux in micturating cystourethrogram.

After pre operative preparation, the patient underwent laparoscopic pyeloplasty with excision of the atretic segment. Gross examination revealed dilated funnel like structure measuring 5 cm in diameter and 2.5 cm in length with congested mucosa. Microscopy revealed denuded urothelium at most places, and oedematous sub mucosa with infiltration by many eosinophils along with lymphocytes and congested vessels. There were aggregates of eosinophils. Muscle coat showed patchy fibrosis and scattered eosinophils (Fig 3, 4). With the above morphological findings, a diagnosis of Eosinophilic Pyelo-ureteritis was rendered. She recovered uneventfully and was followed up for 4 months till date with urine and ultrasound examination.

Discussion

Uretero-pelvic obstruction is a functional impairment of urinary flow from the renal pelvis to the ureter leading to hydronephrosis. Majority are congenital, with male preponderance, and bilateral involvement in the paediatric population. Functional obstruction in adults has been reported due to extrinsic compression by an aberrant lower pole vessel. Eosinophilic pyelo-ureteritis is a very rare entity, with very few reports published in the literature. It has unclear aetiology that may go unrecognized, especially in the chronic phase. It can manifest clinically as dysuria, gross or microscopic hematuria (as seen in our patient), increased frequency, gastro intestinal symptoms (nausea, vomiting, and tenesmus), fever, and skin rash. Allergic disorder is usually seen, but our patient did not have any history of allergy.

The clinical and imaging findings are non specific. It is a benign condition which can closely mimic infection and malignancy, difficult to differentiate by imaging techniques. It has to be considered as a differential in a patient with thickened wall of the urinary tract with preserved mucosal lining on CT (as seen in our patient). Peripheral blood eosinophilia, low complement and increased IgE are the coexistent findings [5-7], but our patient did not have these parameters tested. These parameters suggest that eosinophilic infiltration of the urinary tract may be related to allergic or autoimmune phenomenon [6].

Eosinophilic cystitis and pyelo-ureteritis are widely believed to be due to allergy or hypersensitivity to food, drugs, environmental antigens, parasites, bacterial infection, and trauma due to indwelling catheter, although it is still not clearly understood. [6] It is further emphasized by increased IgE, blood eosinophilia, and resolution of bladder symptoms following treatment [7]. The antigens activate the B lymphocytes with resultant IgE production. IgE bind to mast cells, gets sensitized, and release leukotriene and histamine. Antigen-antibody complex formed result in secretion of interleukin 5 (IL-5) and eotaxin by T_H2 lymphocytes. Eotaxin causes aggregation of eosinophils. Eosinophils release Eosinophilic cationic protein in high concentration, IL-5, and IL-12 that serve to enhance local inflammation, oedema and associated myonecrosis in acute phase. The acute phase gets triggered by variety of stimuli with resultant inflammation, oxidative stress and cytokine rich environment [7]. IL-5 and IL-12 activate Natural killer cells and T lymphocytes, creating a transition from TH2 to TH1 immune response and initiate chronic low grade inflammation and fibrosis [8].

Histology gives a definite clue to the diagnosis. Oedematous sub mucosa, eosinophil rich infiltrate along with mast cells and lymphocytes and myonecrosis are characteristic in acute phase. In chronic stages, there can be fibrosis along with many or few eosinophils. Dysplastic changes and malignancy can be excluded by looking for cellular and nuclear atypia, and invasion. It is also to be differentiated from parasitic infection (Schistosomiasis), tuberculosis with the presence of caseating granulomas, Kimura's disease and Angiolymphoid hyperplasia with capillary proliferation along with aggregates of eosinophils and Hypereosinophilic syndrome with peripheral and marrow eosinophilia [6]. It can be associated with eosinophilic infiltration in other parts of the body, including the liver and gastrointestinal tract [5, 7].

Therapy begins with removal of the offending agent if any, that affords 96% cure rate. [4] Pyeloplasty for eosinophilic pyelo-ureteritis might also help in cure as seen in our patient. Antihistaminic and corticosteroid have proven to be efficacious due to their anti-inflammatory effect by decreasing eosinophil action and stabilising lysosomal membranes [7].

Figures

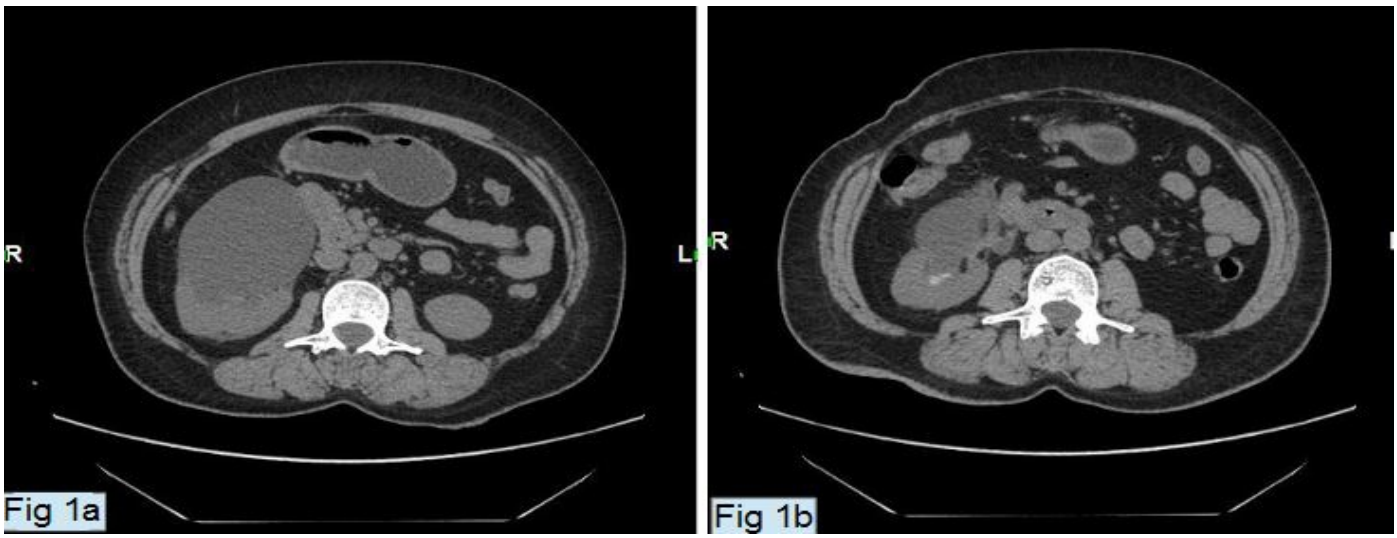

Figure 1: CT images showing dilated renal pelvis and hydronephrosis (1a) and abrupt tapering of the pyelo-ureteric junction (1b).

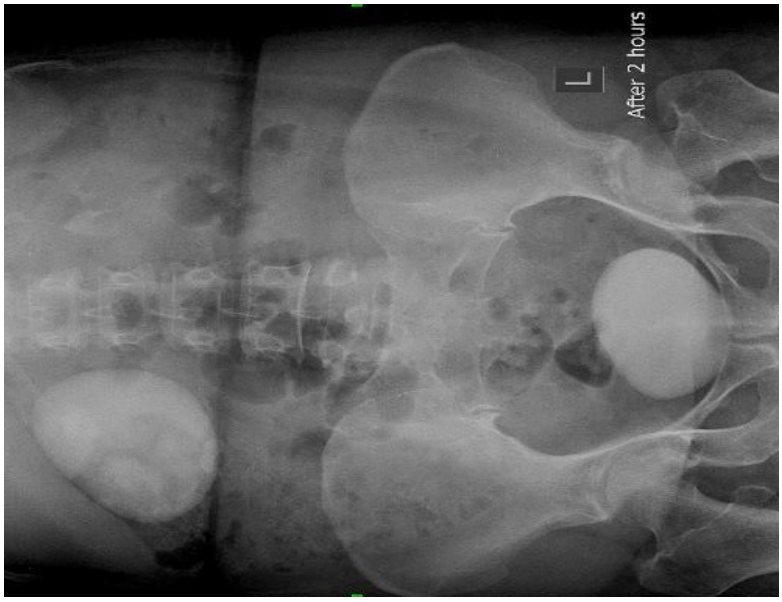

Figure 2: Intravenous Pyelogram, delayed image show dense filling of the renal pelvis with contrast and nonopacified ureter.

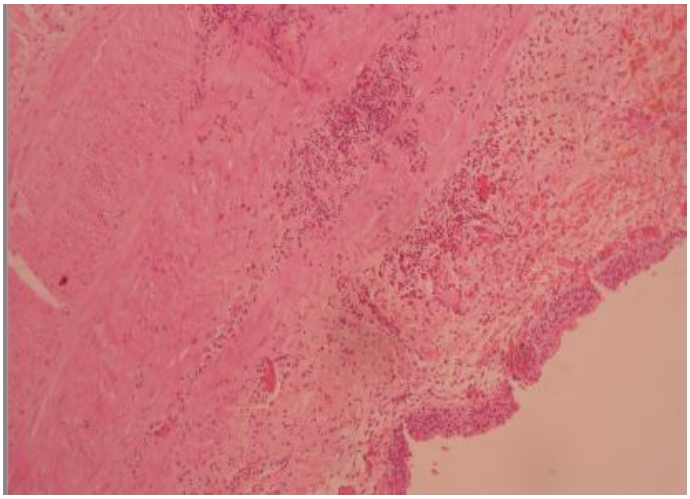

Figure 3: Intravenous Pyelogram, delayed image show dense filling of the renal pelvis with contrast and nonopacified ureter.

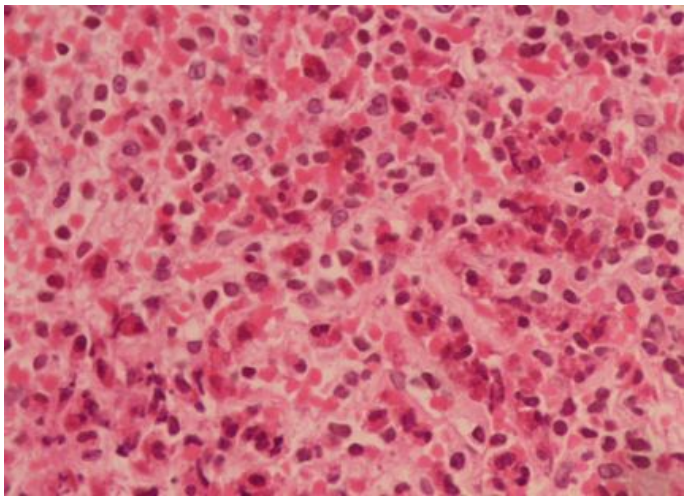

Figure 4: Sub mucosa with eosinophil rich infiltrate. (H & E 40X)

Conclusion

In summary, eosinophilic pyelo-ureteritis has to be considered as a differential, when a functional impairment is encountered with intact mucosa on CT or Ureteroscopy in an adult. Accurate diagnosis is essential as therapy is unique, with identification and withdrawal of the precipitating cause. Treatment is curative in most individuals [4].

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