Case Report

A rare cause of anuria: Bilateral synchronous isolated mid-ureteric tubercular lesions

Anuj D. Dangi, Thomas Alex Kodiatte[1], Santosh Kumar, Nitin S. Kekre

Department of Urology, [1]Pathology, Christian Medical College and Hospital, Vellore, Tamil Nadu, India

ABSTRACT

A young female presenting with right flank pain, fever, raised creatinine and bilateral hydronephrosis was treated with antibiotics elsewhere, with presumptive diagnosis of bilateral pyelonephritis. She had partial relief in symptoms and her creatinine level showed an improvement. Three months later during evaluation at our center she had anuria, hypertensive crisis and pulmonary edema which were managed with emergency bilateral percutaneous nephrostomies. Cross-sectional imaging and ureteroscopy suggested bilateral synchronous intramural mid-ureteric lesions as underlying pathology. Histopathology of the ureteric segments during laparotomy revealed caseating granulomas suggestive of tuberculosis. This clinical presentation has not been previously described in urinary tuberculosis.

Key words: Anuria, synchronous, tuberculosis, ureteric stricture

INTRODUCTION

Genitourinary tuberculosis can present with a wide variety of symptoms. Bilateral synchronous mid-ureteric tubercular strictures as a cause for anuria is uncommon. We recently encountered one such case.

CASE REPORT

A 47-year-old lady presented to us for evaluation of persistent hydroureteronephrosis on imaging. 3 months earlier, she had been treated at another center for a 3 month history of right colicky flank pain associated with malaise and fever, without any lower urinary tract symptoms. Investigations at that time showed raised creatinine (4.8 mg/dL) and bilateral hydroureteronephrosis on ultrasonography. She was treated with antibiotics for suspected pyelonephritis and serum creatinine showed a downward trend, reaching a nadir creatinine of 1.41 mg/dL over the next 3 months. Her medical history was significant for diabetes for 8 years, hypothyroidism for 4 years and hypertension for 3 years. She was a reformed tobacco chewer.

Evaluation at our centre revealed 2-4 red blood cells and 6-8 white blood cells in the urine. Computed tomography (CT urography) showed bilateral hydroureteronephrosis with global caliectasis, right and left ureter were dilated till S1 and L5 vertebral level respectively, at which point there was a hyperdense enhancing lesion within the ureteric lumen, associated with ureteric wall thickening, periureteric and retroperitoneal fat stranding and normal ureteric caliber below this level. There was no evidence of retroperitoneal fibrosis or retroperitoneal lymphadenopathy. Urinary bladder was normal [Figure 1]. She was planned for cystoscopy and bilateral retrograde pyelography but required emergency bilateral percutaneous nephrostomy for sudden onset anuria, breathlessness, azotemia (serum creatinine 11.1 mg/dL), hypertensive crisis (blood pressure 210/110) and flash pulmonary edema. She had post obstructive diuresis and the creatinine level reached a nadir of 1.53 mg% at 2 weeks. Cystoscopy and retrograde pyelography revealed bilateral mid-ureteric narrowing [Figure 2], which persisted on balloon dilatation. A ureteroscope could be negotiated till the lower level of narrowing on both sides and revealed mildly edematous urothelium; no obvious luminal lesion was identified. Intramural pathology compressing the ureteric lumen symmetrically on both sides was considered, and she was taken up for laparotomy and frozen section histology.

For correspondence: Dr. Anuj Deep Dangi, Department of Urology, Christian Medical College and Hospital, Vellore - 632 004, Tamil Nadu, India.
E-mail: anujdeepdangi@gmail.com

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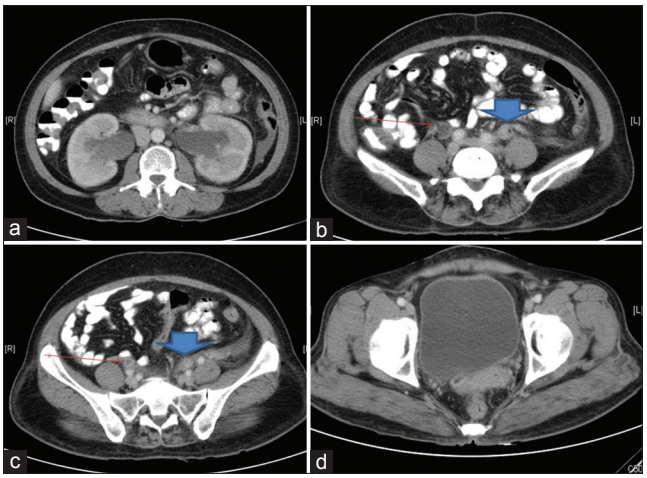

Figure 1: Venous phase of contrast-enhanced computed tomography of abdomen and pelvis showing: (a) bilateral global dilatation of calyces and pelvis, (b and c) red and blue arrows showing right and left ureters respectively, (d) showing normal urinary bladder

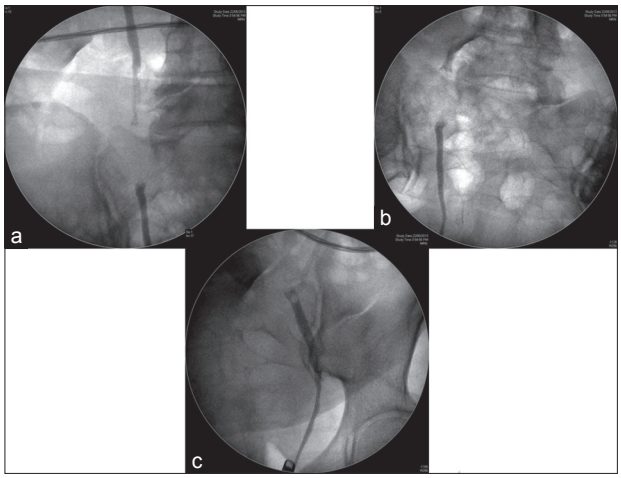

Figure 2: Retrograde pyelography done before ureteroscopy: (a and b) showing filling defect in right ureter at level of upper sacroiliac joint, both proximal and distal ureter are well opacified with radio contrast, (c) showing filling defect in left ureter, faint opacification of proximal ureter is seen at upper sacroiliac joint

On laparotomy, the left ureter was found to be thickened and fibrotic from L5 to S1 vertebral level; similarly, right ureter was thickened and necrotic from L5 to S3 level, retroperitoneal tissue around these ureters was grossly normal. Frozen sections from the ureteric wall on both sides were reported as caseating granulomatous inflammation and later confirmed on routine histopathology. Special stains for acid fast bacilli (AFB) were negative. An ileal segment was used to replace the involved ureteric segments from L3 level to the bladder and she was started on 4 drug antitubercular therapy (ATT), with which she showed clinical improvement (absence of fever, malaise and flank pain). Ureteric splints were removed at 6 weeks. Six months later, creatinine was 0.66 mg% and follow up ultrasound of abdomen 6 months and 18 months post surgery showed resolution of bilateral hydronephrosis. She was continued on ATT beyond 6 months as evaluation for headache revealed an intracranial right cavernous sinus lesion which was not amenable to biopsy and was presumed to be tubercular in etiology.

DISCUSSION

Bilateral synchronous mid-ureteric lesions are rare and may be congenital,[1] or iatrogenic, following radiotherapy[2] or aortic aneurysmal surgery.[3] Bilateral ureteric involvement with acute renal injury has been described due to radiation-induced mid- and lower ureteric strictures; however, bilateral synchronous mid-ureteric tubercular strictures without obvious radiological renal or bladder involvement as a cause for acute renal injury has not been described in literature.

Urinary system involvement in tuberculosis is commonly unilateral,[4] probably due to delayed reactivation of a solitary focus.[5] Ureteric involvement is considered secondary to renal involvement, with the vesicoureteric junction being the most commonly involved. Tuberculosis usually involves the intramural part of the ureteric wall and is characterized by ureteric thickening and post contrast ureteric wall enhancement on contrast-enhanced computed tomography.[6] Bilateral involvement with renal failure is commonly seen after descending infection from one renal unit involving the bladder and subsequent contralateral unit damage due to vesicoureteric reflux and/or small capacity poorly compliant bladder.[4]

This case is unique in many respects and cannot be easily explained by accepted theories on the etiopathogenesis of urinary tuberculosis. There was bilateral, synchronous involvement of the mid-ureter, which, according to existing literature, is the least commonly involved ureteric segment in urinary TB. This can only be explained by taking into account the following facts: bilateral pathological involvement of the urinary system via hematogenous route is not unusual; in fact, a seminal study by Medlar found bilateral pathological involvement to be the rule, rather than the exception and renal tuberculosis can be clinically silent.[7] Involvement of bilateral ureteric segments either by direct hematogenous spread or following bacilluria from an undetectable renal lesion and subsequent simultaneous activation can explain the findings of the case. The interaction between host immunity and pathogenicity decides the clinical presentation. At one end of spectrum is the activation of a single focus of tuberculosis even in the presence of multiple tubercular pathological foci and at the other end is disseminated tuberculosis where more than one tubercular foci become clinically apparent simultaneously. In the case described above, the simultaneous clinical activation of two pathological foci and later on presentation of an intracranial lesion suggests this patient's immune response lay somewhere midway along the spectrum. The presence of caseating necrotizing granulomatous inflammation is almost always associated with tuberculosis and rarely with

nocardiosis (where granuloma formation is rare); however, the clinical features (no solid organ abscesses, response to ATT) are not consistent with the latter.

If tuberculosis had been entertained as a differential diagnosis, appropriate microbiological work up could have been initiated and if proven positive, the patient would have benefited from a 4 to 6 week course of anti-tubercular therapy prior to treatment for ureteric stricture, if it failed to resolve with medical therapy.

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