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Isolated Distal Ureteral Tuberculosis Presenting as Renal Colic: A Case Report and Literature Review

Ayoub MAMAD[1*], Mohammed Amine ELAFARI[1], Mohammed Amine BIBAT[1], Pr Amine SLAOUI[1], Pr Tarik KARMOUNI[1], Pr Abdelatif KOUTANI[1], Pr Khalid ELKHADER[1]

[1]Urology Department, Ibn Sina Hospital, University Hospital Center IBN SINA, University Mohammed V, Rabat, Morocco

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***Corresponding author:** Ayoub MAMAD
Urology Department, Ibn Sina Hospital, University Hospital Center IBN SINA, University Mohammed V, Rabat, Morocco

Abstract **Case Report**

Genitourinary tuberculosis (GUTB) remains a diagnostic challenge because clinical manifestations are often non-specific, bacilluria may be intermittent, and imaging findings can mimic malignancy or other inflammatory causes of ureteral obstruction. We report an atypical presentation of GUTB as an isolated distal ureteral stenosis revealed by persistent renal colic. A 66-year-old man presented with a two-month history of right-sided renal colic without lower urinary tract symptoms or fever. Computed tomography urography showed right hydronephrosis with circumferential thickening of the renal pelvis and ureter, upstream of a short distal ureteral stricture. Cystoscopy revealed a punctiform right ureteric orifice requiring endoscopic enlargement to permit placement of a double-J stent. Ureteroscopic biopsies demonstrated granulomatous inflammation with caseous necrosis, supporting the diagnosis of urogenital tuberculosis. Mycobacterial urine culture and chest imaging did not show concomitant pulmonary disease. The patient completed a six-month antituberculous regimen with a favorable clinical outcome. This case highlights the importance of considering GUTB in patients with unexplained ureteral strictures, particularly when routine urine cultures are negative, to avoid diagnostic delay and irreversible urological sequelae.

Keywords: Urogenital tuberculosis; Ureteral stricture; Sterile pyuria; Ureteroscopy; Obstructive uropathy; Case report.

INTRODUCTION

Tuberculosis (TB) remains a major global health problem and a leading cause of death from a single infectious agent in many regions. Extrapulmonary TB represents a substantial proportion of the overall disease burden, and the genitourinary tract is a frequent site of extrapulmonary involvement [1–4].

GUTB typically results from hematogenous spread after primary infection, with renal parenchymal seeding followed by descending involvement of the collecting system, ureters, and bladder [2,3]. Because symptoms may be subtle and routine urine cultures are often negative, diagnosis is commonly delayed, especially in low-incidence settings [2–5].

Delayed recognition is clinically consequential: progressive inflammation and healing by fibrosis can cause strictures, hydronephrosis, and irreversible loss of renal function [3,4]. We report a case of distal ureteral tuberculosis presenting as persistent renal colic and obstructive uropathy, emphasizing the diagnostic contribution of endoscopy and biopsy.

CASE REPORT

A 66-year-old man with no notable medical history presented with a two-month history of right-sided renal colic.

He reported no fever, dysuria, urgency, frequency, or other lower urinary tract symptoms. Physical examination revealed right costo-vertebral angle tenderness without peritoneal signs; the remainder of the examination was unremarkable.

Laboratory investigations demonstrated a marked inflammatory response with C-reactive protein of 248 mg/L and leukocytosis of 13,400/mm³ with neutrophil predominance. Renal function was preserved (serum creatinine within normal limits). Urinalysis showed significant leukocyturia with microscopic hematuria; initial routine urine culture was negative.

Renal and bladder ultrasound showed right pyelo-calyceal dilatation with an anteroposterior pelvic diameter of approximately 23 mm. Computed tomography urography confirmed right hydronephrosis (renal pelvis about 21 mm) with circumferential thickening of the renal pelvis and ureteral wall upstream of a regular, short stenosing thickening of the distal pelvic ureter (about 8 mm in length), located immediately proximal to the ureterovesical junction (figure 1).

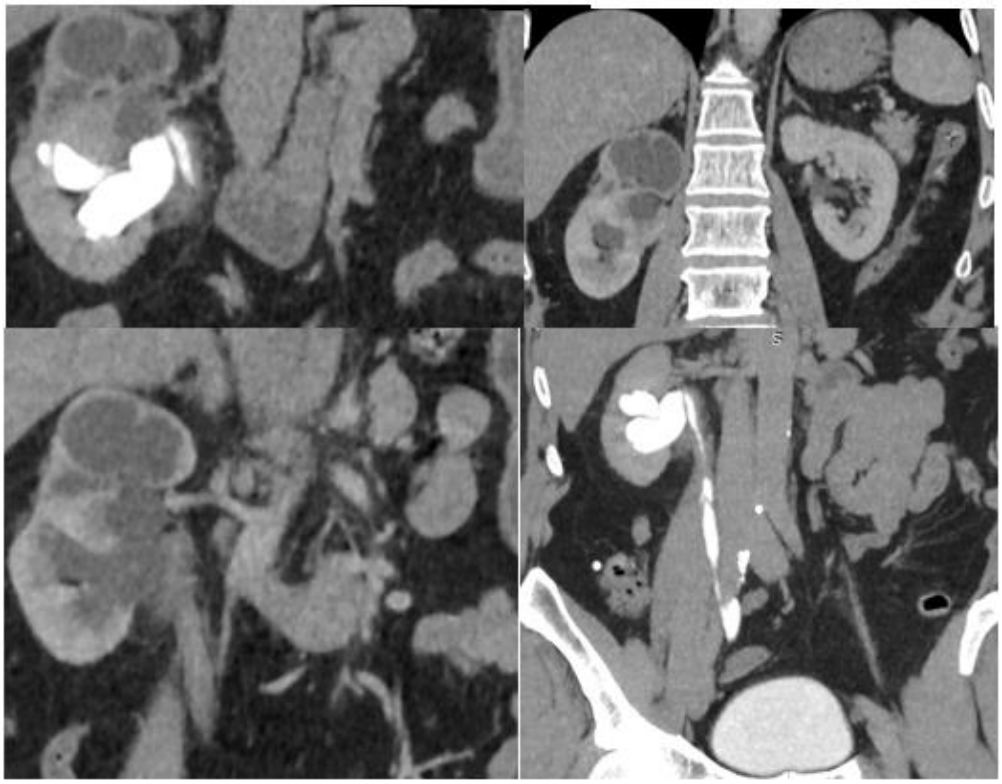

Figure 1. CT urography findings of right hydronephrosis associated with circumferential thickening of the renal pelvis and ureteral wall

Cystoscopy demonstrated a normal bladder mucosa and a normal left ureteric orifice. The right ureteric orifice was punctiform, and endoscopic enlargement was required to allow insertion of a right double-J ureteral stent. Diagnostic ureteroscopy with targeted ureteral biopsies was then performed.

Histopathology revealed granulomatous inflammation with caseous necrosis, highly suggestive of tuberculosis. Additional investigations (urine mycobacterial culture) and chest computed tomography did not identify concomitant pulmonary disease.

A six-month antituberculous regimen for presumed drug-susceptible TB was initiated, consisting of two months of rifampicin, isoniazid, pyrazinamide, and ethambutol, followed by four months of rifampicin and isoniazid. The clinical course was favorable, with resolution of pain and no recurrence of symptoms reported at the end of treatment.

DISCUSSION

The genitourinary tract is among the most frequently involved sites of extrapulmonary TB, yet GUTB remains under-recognized because clinical signs are often non-specific and concomitant pulmonary disease is frequently absent at presentation [1–5]. After hematogenous seeding of the kidneys, ureteral involvement typically occurs by descending spread; fibrotic healing may lead to segmental ureteral stenosis, particularly at anatomical narrowings such as the ureterovesical junction [3].

Symptoms may include irritative voiding symptoms, hematuria, flank pain, or recurrent episodes labeled as bacterial cystitis despite negative standard cultures. Sterile pyuria with microscopic hematuria is a common clue but is not specific; the key is persistence and the combination with suggestive imaging or risk factors (endemic exposure, immunosuppression, or previous TB) [2,3,5,11]. In the present case, the lack of lower urinary tract symptoms and the presentation as renal colic delayed the suspicion of TB.

CT urography is central for evaluating ureteral obstruction and may show urothelial thickening, strictures, hydronephrosis, or parenchymal renal lesions. However, imaging findings are protean and can overlap

with urothelial carcinoma, schistosomiasis, iatrogenic or post-stone strictures, and other inflammatory conditions [6]. Therefore, when imaging shows unexplained ureteral wall thickening or stenosis, tissue sampling is often required.

Mycobacterial culture of multiple early-morning urine specimens remains a reference standard but is limited by prolonged turnaround time and variable sensitivity due to intermittent bacilluria [3,4,11]. Rapid molecular tests (NAATs) are increasingly incorporated into diagnostic algorithms for extrapulmonary TB; urine-based Xpert MTB/RIF can provide timely results and has shown promising pooled accuracy in meta-analysis, although performance varies across studies and patient populations [7,15]. In our patient, urine mycobacterial culture was negative, and ureteroscopic biopsy was decisive, consistent with reports that invasive sampling can be essential when non-invasive tests are non-diagnostic [5].

Management of urogenital tuberculosis (UGTB) is multimodal, combining antitubercular chemotherapy with measures to preserve urinary drainage and, when needed, definitive reconstruction. For drug-susceptible disease, most guidance supports a 6-month regimen with an initial 2-month intensive phase (isoniazid, rifampicin, pyrazinamide and ethambutol), followed by a 4-month continuation phase (isoniazid and rifampicin) [10,11,14,15].

From a urological standpoint, it is crucial to recognize that tuberculous ureteral strictures can be present at diagnosis but may also emerge or worsen after treatment initiation because of cicatricial fibrosis during healing. This “paradoxical” evolution justifies close functional and radiological follow-up of the upper urinary tract throughout therapy [11,12].

When obstruction is present and the kidney remains salvageable, urgent decompression is recommended to protect renal function, either via retrograde double-J (JJ) stenting or percutaneous nephrostomy [13]. Several authors report that early JJ stenting combined with chemotherapy may delay or even avoid major reconstructive surgery or nephrectomy in selected cases; however, stents are a temporary solution and may act as a nidus for infection and become encrusted/calcified [12]. Consequently, prolonged stenting often requires iterative JJ exchanges (commonly every 3–6 months depending on the clinical context) and close monitoring; conversion to nephrostomy is an alternative when retrograde access is not feasible or when stenting fails to control obstruction.

Definitive repair depends on stricture length, location and bladder condition and is generally planned once the acute inflammatory phase has been controlled medically (often after several weeks of chemotherapy) unless sepsis or complete obstruction mandates earlier intervention [12]. For short, isolated strictures, endoscopic balloon dilatation or endoureterotomy can be attempted when the ureter is catheterizable, with variable success reported in the literature [12]. For focal stenoses amenable to excision, surgical resection with end-to-end ureteroureterostomy (resection-anastomosis) is a durable option [12]. Distal juxtavesical/pelvic strictures can be managed by ureteroneocystostomy when isolated [12]; if additional length is required, adjuncts such as a psoas hitch and/or Boari flap may bridge longer distal defects while maintaining a tension-free, well-vascularized anastomosis [16].

For long-segment or multiple strictures not amenable to reimplantation or ureteroureterostomy, intestinal ureter substitution (entero-ureteroplasty) using appendix or ileum may be considered, typically as a last resort because mucus production and graft atony can compromise drainage and potentially worsen renal function [12]. Reconfigured ileal techniques based on the Yang–Monti principle have been used to replace longer ureteral segments while limiting the length of bowel harvested; double or even triple reconfigured segments can be combined to obtain the required tube length [18]. When bladder tuberculosis leads to a severely contracted bladder, augmentation or replacement cystoplasty may be necessary to protect the upper tracts [12,13]. In such settings-particularly if urethral catheterization is difficult-a continent catheterizable channel can be created using an appendix (Mitrofanoff) or an ileal Monti channel; the “double Monti” configuration (two segments in series) can provide additional length when needed (e.g., obesity or a high stoma site) [17].

The role of adjunctive corticosteroids remains debated. While routine steroid therapy is not recommended, some teams consider it in the setting of ureteral stenosis that worsens or fails to improve after a few weeks of effective chemotherapy; the benefit has not been consistently demonstrated compared with endoscopic management, and dosing must account for rifampicin-related enzyme induction that lowers steroid exposure [11-13].

The punctiform ureteric orifice and short distal ureteral stricture in this case are consistent with the tendency of tuberculous strictures to involve the ureterovesical junction [3]. This presentation reinforces that isolated ureteral disease may occur without clear pulmonary involvement and may mimic other causes of obstructive uropathy. Prompt endoscopic assessment with biopsy allowed timely antituberculous treatment and likely prevented progression to irreversible urological damage.

CONCLUSION

Genitourinary tuberculosis should be considered in the differential diagnosis of ureteral strictures and obstructive uropathy when routine urine

cultures are negative. Early suspicion, appropriate imaging, and timely microbiological or histological confirmation are crucial to prevent irreversible urinary tract damage. In isolated ureteral disease, endoscopic evaluation with biopsy may be pivotal to secure the diagnosis and guide combined medical and urological management.

DECLARATION

Ethics approval and consent to participate: Ethical approval is not applicable. The case report does not contain any personal information.

Consent for publication: Written informed consent for publication of the clinical details and images was obtained from the patient.

Declaration of interests: The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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