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Unusual ureteral stenosis in an immunosuppressed patient: Case report.

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Abstract

Introduction: Genitourinary tuberculosis (GUTB) is among the most insidious forms of extrapulmonary tuberculosis. In HIV-positive patients, progression to structural complications can be severe, even under stable immunosuppression. The aim of this report is to analyze the progression of GUTB to end-stage renal disease secondary to obstructive nephropathy.

Case report: A 45-year-old male patient with HIV (15-year history, undetectable viral load, and CD4+ count > 250 cells/mm³). He had a recent history of successfully treated pulmonary tuberculosis. He presented with a 3-month history of dysuria and decreased urine output, which progressed to acute urinary retention. Imaging revealed grade IV bilateral hydronephrosis and segmental ureteral stenosis. Laboratory tests revealed critical deterioration of renal function (initial creatinine concentration of 3 mg/dL, which progressed to 19.27 mg/dL, and a urea concentration of 284 mg/dL). Urine culture confirmed the presence of *Mycobacterium tuberculosis*.

Evolution: Despite the initiation of antituberculosis treatment and bladder decompression, the patient developed severe uremic symptoms. The severity of bilateral obstructive nephropathy resulted in irreversible kidney damage (stage V), requiring the initiation of chronic renal replacement therapy (dialysis), with no possibility of surgical intervention due to the extent of fibrosis.

Conclusion: Genitourinary tuberculosis can cause accelerated end-stage renal failure due to obstructive nephropathy, even in patients with controlled HIV and successful antituberculosis treatment. Irreversible ureteral fibrosis necessitates early urological monitoring to detect strictures before functional damage becomes permanent and requires chronic dialysis.

Keywords: Genitourinary tuberculosis, HIV, nephropathy obstruction, ureteral stenosis, end-stage renal disease.

Introduction

Scarring from genitourinary tuberculosis (GTB) results from an inflammatory process secondary to *Mycobacterium tuberculosis* infection. Its most devastating characteristic is not the active infection itself but the hyperscring response it generates [1]. Unlike other infections, TB destroys tissue and replaces it with collagen and calcium, permanently altering the architecture of the urinary tract. The main scarring lesions, classified by their location and functional effect, are, first, ureteral strictures known as "beaded" lesions, in which granulomatous inflammation in the ureteral submucosa heals through fibrosis; multiple areas of narrowing (strictures) alternate with areas of dilation [2]. This gives the ureter a segmental or "beaded" appearance on imaging studies, with sequelae of severe obstructive uropathy, hydronephrosis, and functional renal exclusion. The second is the parenchymal renal lesion known as the mastic kidney or "putty kidney," sometimes called autonephrectomy, because it is the terminal stage of parenchymal scarring in which the renal parenchyma is destroyed and replaced by caseous material that subsequently calcifies [3]. The kidney shrinks, becomes small and dense, and loses all its corticomedullary architecture. On CT scans, a calcified, shapeless mass is observed, with complete cessation of function of that renal unit (nonsurgical autonephrectomy). The third is necrotizing papillitis, which, upon healing, causes retraction of the calyx, making it round. The duct that connects the calyx to the renal pelvis (infundibulum) closes because of fibrosis, isolating a group of calyces and forming retention cysts filled with purulent or caseous material [4]. The fourth is microcystis (tuberculous bladder), in which if the infection progresses, the bladder also undergoes irreversible scarring. Inflammation of the muscular layer (detrusor) is replaced by fibrous tissue. The bladder loses its elasticity and distensibility, becoming a small, rigid structure with thick walls. As a consequence, there is an extreme decrease in urine volume, severe frequency, and vesicoureteral reflux (which aggravates hydronephrosis) [4].

Lesions are more prevalent in men, with a ratio of 2:1, and occur in 5 to 10% of patients with tuberculosis, sometimes appearing in isolation or concurrently with pulmonary involvement [1, 2]. Extrapulmonary tuberculosis is more common in patients with HIV or those who have undergone organ transplantation. Genitourinary involvement accounts for 27% of extrapulmonary tuberculosis cases. Associated clinical manifestations are diverse, ranging from general symptoms such as fever or weight loss, with or without sterile pyuria and hematuria [2].

The diagnosis is made by rescuing the germ in body fluids such as urine or by performing a culture of compromised tissue, with the use of a genetic technique being the most specific, allowing for timely diagnosis and appropriate treatment.

We present the case of an immunocompromised patient who developed genitourinary tuberculosis in the context of structural alterations of the urinary tract.

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Case report

Medical records

This is a 45-year-old male patient who has been receiving appropriate treatment for 15 years and whose immunological status is stable (viral load less than 25 copies and CD4+ T lymphocyte count greater than 250). Seven months ago, he developed pulmonary tuberculosis, for which he received appropriate antituberculosis treatment and experienced clinical improvement. He presents with a three-month history of dysuria and a progressive decrease in urine output, culminating in acute urinary retention.

Diagnostic tests

Laboratory tests revealed impaired renal function, with an initial serum creatinine concentration of 3 mg/dL. Ultrasound showed cortical and medullary abnormalities, with signs of grade IV bilateral hydronephrosis. A noncontrast CT urogram confirmed severe bilateral hydronephrosis with pyelocaliceal dilation and segmental ureteral stenosis. Given that the patient was immunosuppressed, structural abnormalities of infectious origin were suspected, and a urine culture was ordered, which was positive for *Mycobacterium tuberculosis*. The clinical picture was interpreted as chronic kidney disease secondary to segmental ureteral stenosis due to genitourinary tuberculosis (GTB) (Figure 1).

Evolution

A urinary catheter was inserted, resulting in partial resolution of the symptoms; however, uremic symptoms developed, with a progressive increase in blood urea nitrogen (BUN) levels, reaching a urea concentration of 284 mg/dL and a creatinine concentration of 19.2 mg/dL. Therefore, a dialysis catheter was placed, and dialysis therapy was initiated (Table 1). Following the diagnosis of tuberculous ureteritis nodosa (TUUB), antituberculosis treatment was started, and surgical intervention by urology was awaited. However, the patient progressed to stage V chronic kidney disease requiring chronic dialysis, and surgical treatment was no longer possible.

Table 1. Laboratory results.

Parameter	Result	Range
Hematocrit	23%	42 – 45%
Hemoglobin	8.5 g/dl	12.3 – 15.3 g/dl
Platelets	231 mil/mm3	150 – 450 thousand.
Urea	284 mg/dl	21 – 43 mg/dl
Creatinine	19.27 mg/dl	0.6 – 1.1 mg/dl
Sodium	131 mEq/l	136 – 145 mEq/l
Potassium	3.75 mEq/l	3.5 – 5 mEq/l
Glucose	151 mg/dl	80 – 110 mg/dl
TGO	21 UI	31 – 34 U/L
TGP	14 UI	0 – 45 U/L
Phosphorus	4.5 mg/dl	2.3 – 4.5 mg/dl
Albumin	3.0 gr/dl	3.5 - 4.5 gr/dl
Urinalysis	cloudy appearance, density 1009, leukocytes +++	

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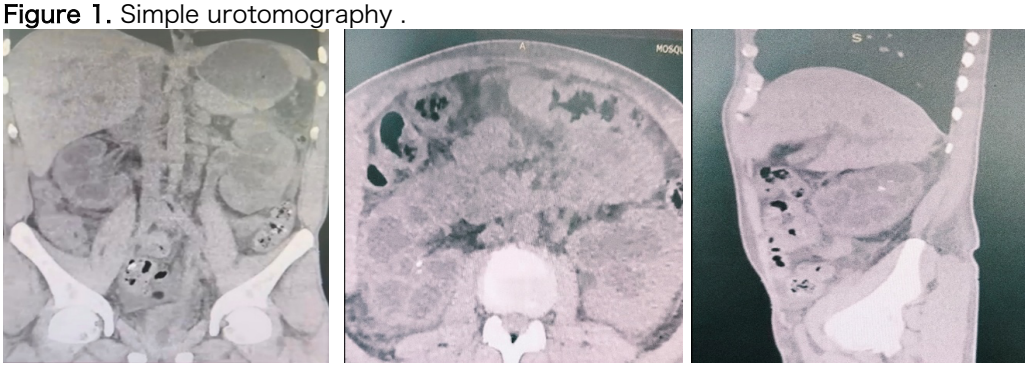
Figure 1. Simple urotomography .



The right kidney shows severe pyelocaliceal dilation , with loss of the renal sinus parenchyma relationship. The left kidney is enlarged, with diffuse cortical thickening and severe pyelocaliceal dilation . A dilated lumbar portion of the ureter is evident at the level of the ureterovesical junction . Changes in perirenal fat density are present, without retroperitoneal lymphadenopathy.

Discussion

This case describes a rare and serious complication of extrapulmonary tuberculosis: genitourinary tuberculosis (GTB) progressing to end-stage renal disease (ESRD) in a patient with controlled HIV. The significance of this report lies in the rapid progression of irreversible kidney damage, despite a stable immune status.

Gastrointestinal tuberculosis (GUTB) is the second most common cause of extrapulmonary tuberculosis. In this patient, dysuria and acute urinary retention were alarm signs of segmental ureteral stricture, a classic sequela of *Mycobacterium tuberculosis* infection. Unlike other reported cases in which GUTB presents as "sterile pyuria," here, the presentation was severely obstructive, precipitating grade IV hydronephrosis.

A critical point of discussion is the coexistence with HIV. Although the patient maintained a CD4+ count above 250 cells/mm³ and an undetectable viral load—factors that theoretically suggest a good immune response—the literature indicates that patients with HIV have a significantly higher risk of reactivation of latent tuberculous foci and more aggressive fibrotic progression in the urinary collecting system.

The patient's renal failure was not purely parenchymal but postrenal. CT urography confirmed that segmental ureteral stenosis caused bilateral obstructive uropathy. The differential diagnosis initially revealed urothelial neoplasms or retroperitoneal fibrosis; however, a history of recent pulmonary tuberculosis and a positive urine culture for *M. tuberculosis* confirmed the infectious etiology.

Despite the initiation of antituberculosis treatment and attempted urological management, the patient developed chronic uremia (creatinine concentration: 19.27 mg/dL; urea concentration: 284 mg/dL; BUN/creatinine ratio: 6.8). This poor clinical course suggests that, at the time of diagnosis of genitourinary tuberculosis (GT), the scarring and ureteral fibrosis were already irreversible, resulting in obstructive nephropathy. A determining factor in the patient's poor

clinical course was the development of severe bilateral obstructive nephropathy. In the context of genitourinary tuberculosis, chronic inflammation caused by the bacillus not only destroys the parenchyma but also induces a fibrotic response that scars and narrows the ureters (segmental stenosis) [6]. This mechanical obstruction generates hydrostatic backpressure that is transmitted to nephrons, causing tubular atrophy and a drastic decrease in the glomerular filtration rate [7 , 8]. Unlike other causes of renal failure, obstructive nephropathy in this case manifested as irreversible postrenal nephropathy. The critical creatinine levels (19.27 mg/dL) and grade IV hydronephrosis reported on CT urography confirm that the mechanical damage from the stenosis was so severe that it overwhelmed any possibility of renal compensation, resulting in stage V chronic kidney disease that did not respond to partial resolution with a urinary catheter. Early intervention with urinary diversions (such as double-J stents or nephrostomies) may be the only window of opportunity to avoid chronic dialysis dependence in cases of segmental stenosis.

To ensure a sustainable and humane healthcare system, identifying these risks in subclinical stages is vital. The transition from a productive patient to one with a permanent need for renal replacement therapy critically affects both the individual's quality of life and the costs to the healthcare system.

Conclusions

This case offers valuable lessons for managing patients with complex comorbidities such as HIV and tuberculosis: obstructive nephropathy was a point of no return, where progression to stage V chronic kidney disease in this patient was not solely due to the *Mycobacterium tuberculosis* infectious load but also to severe and irreversible bilateral obstructive nephropathy. This implies that mechanical damage can be more devastating and rapid than direct inflammatory damage. Despite optimal HIV immunological control ($CD4+ > 250$), the patient's urinary system underwent silent degradation, indicating that immunological stability does not preclude serious extrapulmonary complications. Dysuria and urinary retention should not be overlooked, as in tuberculous nephropathy (TUN), they are often signs of established obstruction that is difficult to reverse surgically. Antituberculosis treatment, while essential for sterilizing urine, is insufficient to treat structural sequelae.

Abbreviations

TBGU: Genitourinary tuberculosis.
HIV: human immunodeficiency virus.

Supplementary information

The supplementary materials have not been provided.

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Authors' contributions

Roberto Gutiérrez Gómez: Conceptualization, data curation, research, visualization, original draft writing.
Pedro Gutiérrez Gómez: Conceptualization, data curation, research, visualization and writing of the original draft.
Sofía Gutiérrez Gómez: Conceptualization, formal analysis, methodology, project management, resources, software, supervision, validation, writing–review and editing.
Alex Gutiérrez Gómez: Conceptualization, data curation, research, visualization and writing of the original draft.

All the authors read and approved the final version of the manuscript.

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Clinical cases are not needed.

Consent for publication
The authors have the patient's authorization to publish the images and the clinical case.

Conflicts of interest
The authors declare that they have no conflicts of interest.

Use of generative AI
The authors declare that they did not use generative AI in this document.

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