CASE REPORT

All in One – Multi-Organ Involvement of Never-Ending Tuberculosis

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ABSTRACT

Tuberculosis (TB) prevalence has risen over the last few decades, particularly in developing countries. The genitourinary tract is the most prevalent extrapulmonary site for tuberculosis. Genitourinary TB symptoms are frequently ambiguous. To identify multiorgan TB, healthcare personnel must understand TB imaging characteristics on various imaging modalities in different organ systems, as well as how to correlate these results with histologic analysis and clinical assessment.

Keywords: *Tuberculosis, kidneys, radiology, urology, Pakistan.*

INTRODUCTION

Tuberculosis (TB) is one of the most frequent infectious illnesses in underdeveloped nations, with the prevalence increasing owing to HIV and AIDS. Pulmonary TB is the most common kind observed. One of the most frequent locations of extrapulmonary tuberculosis (TB) involvement is the genitourinary area [1]. Twenty percent of cases of extrapulmonary tuberculosis are caused by renal tuberculosis [2]. Genitourinary TB typically coexists with two percent to twenty percent of pulmonary TB patients; this frequency is higher in developing nations [1]. Both the renal parenchyma and the collecting system (calyces, renal pelvis, ureter, bladder, and urethra) may be affected by tuberculosis, which can lead to various radiological and clinical manifestations [2]. The diverse clinical and radiological presentations of genitourinary tuberculosis, which frequently mirror other diseases, make it difficult to diagnose. A strong degree of clinical suspicion and acquaintance with distinctive aspects of tuberculosis imaging are typically necessary for an early diagnosis [3]. To contribute to the body of knowledge on renal TB, we are sharing a case study of renal TB that tested positive.

CASE PRESENTATION

A 32-year-old female, without known comorbidities, presented to the outpatient department with a history of gross hematuria and pyuria persisting for two months, accompanied by increased urinary frequency, burning micturition and left flank pain. She also reported intermittent fever with chills, loss of appetite, nausea, and shortness of breath. Symptoms of increased urinary frequency and left flank pain had been present for approximately 1.5 years. Her past surgical history was unremarkable. Initial laboratory tests revealed significant findings, including urine analysis showing proteinuria, numerous red blood cells and leukocytes. Her complete blood count (CBC) showed a total white blood cell count of 10,280/µL, a hemoglobin level of

12.2 g/dL, and a platelet count of 548,000/µL. Serum creatinine was elevated at 1.48 mg/dL. Pelvic ultrasound revealed a small bladder volume with postvoid residual urine.

Further imaging with CT abdomen and pelvis revealed mild bilateral hydronephroureter and thickened pelvic and ureteric walls with adjacent fat strandings. Focal uneven Calyctasis was also observed bilaterally with cortical thinning on the left (**Fig. 1A** and **1B**). A thick wall urinary bladder was seen with perivesical fat stranding without intraluminal calculus. Calcified granuloma was noted in the right hepatic lobe, suggesting a granulomatous disease process **(Fig. 2)**. Basal lungs demonstrate multiple trees in bud nodular infiltrates concerning for endobronchial spread of acute infective disease process **(Fig. 3)**. Bilateral ureteroscopy with DJ stenting showed reduce bladder capacity with inflamed bladder mucosa and noticeable bleeding and sloughing of tissue. Additionally, ureteral obstructions and beading were noted with calyceal irregularities

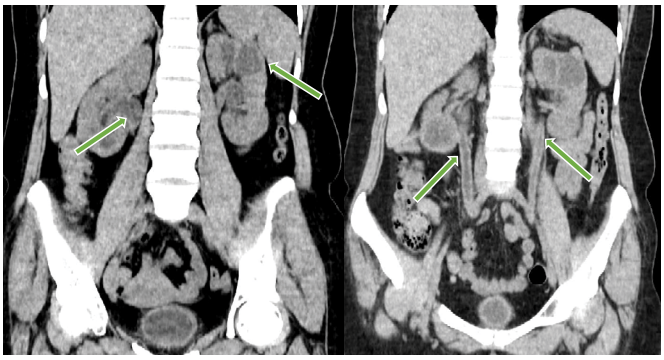

Fig. (1A) (right): CT abdomen coronal reformatted images showing mild hydronephrosis with thickened pelvic walls (right) and uneven calyctasis with focal cortical thinning (left).
Fig. (1B) (left): CT abdomen coronal reformatted images showing mild hydroureters with thickened ureteric walls.

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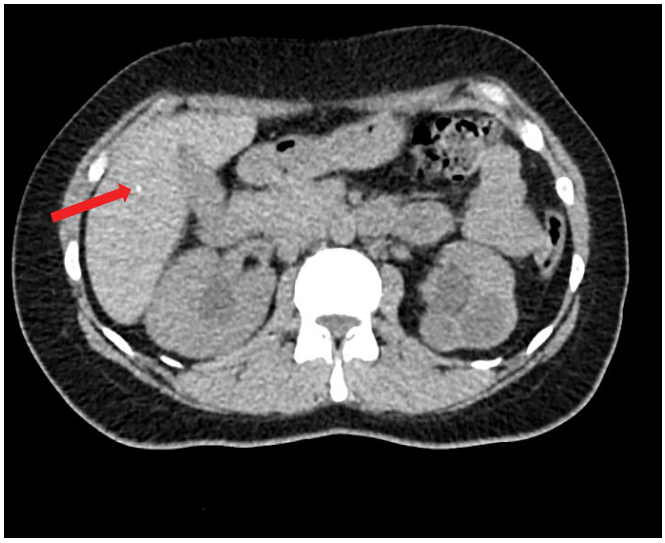

Fig. (2): CT Abdomen Axil view showing tiny calcified granuloma in the right hepatic lobe.

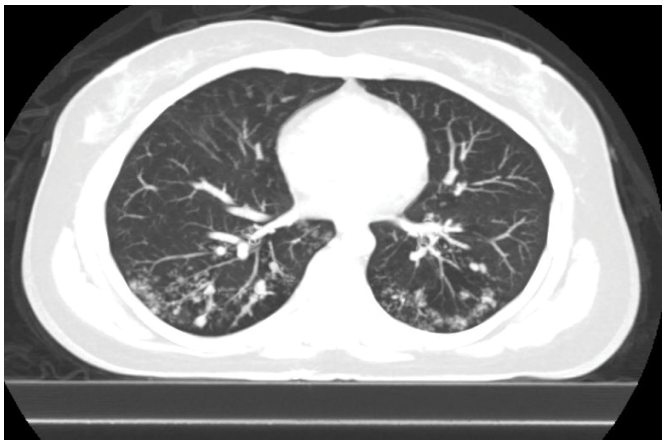

Fig. (3): CT Axial view (lung window) showing multiple trees in bud nodular infiltrates in superior segments of bilateral lower lobes.

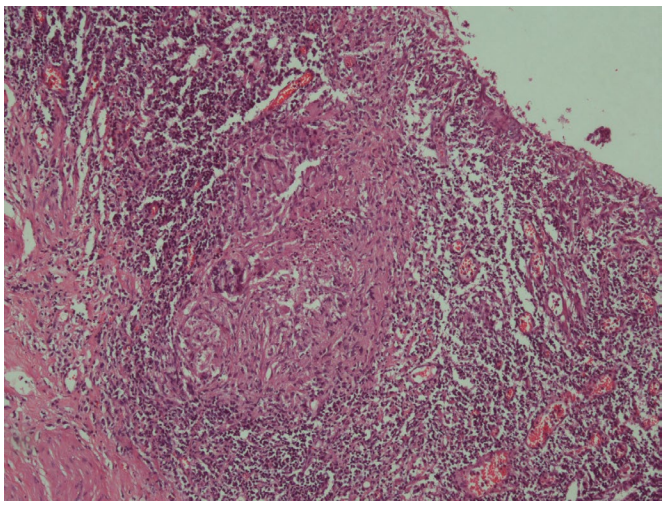

Fig. (4): Bladder wall with dense chronic inflammation and collections of epithelioid histiocytes forming granulomas. Foci of caseous necrosis and langhan type giant cells are also noted (H&E 20X).

and infundibular stenosis. Bladder biopsies were taken during the procedure. Acid-fast *Bacilli* (AFB) were noted in urine and bladder tissue samples, with positive results on MTB gene testing. Histopathological examination of bladder tissue indicated chronic granulomatous cystitis, likely due to tuberculosis, ruling out malignancy **(Fig. 4)**. The patient was promptly initiated on anti-tuberculous therapy and continued follow-up in the outpatient department.

DISCUSSION

Mycobacterium tuberculosis is an acid-fast *Bacillus* that grows in an aerobic environment and is known to be notorious for causing morbidity and even mortality in humans. Because of its slow replication rate, it is resistant to several antibiotics that act during the replication and division phases. It can remain inactive and dormant for a long period, without resulting in any symptoms. It is reactivated mostly within the immunity on the lower side, thus resulting in symptoms [4]. Mostly, primary pulmonary tuberculosis is asymptomatic. Invasion of the renal and prostatic parenchyma is possible when bacillemia spreads from the pulmonary center to other tissues.

The patient enters a latent phase after almost half a year, during which time they may reactivate the illness 5% of the time in the following two years and 5% of their lifetime. After six months, the patient develops spontaneous cicatrization of primary pulmonary TB. Latent foci reactivated in most of the present cases of pulmonary and extrapulmonary disease when immunological function was compromised by low immunity, steroids, diabetes, and drugs that result in immunosuppression [5].

Even in industrialized nations where TB was previously uncommon, the AIDS epidemic resulted in unprecedented global tuberculosis levels. Patients with urogenital TB in developed nations exhibited fewer specific symptoms and a reduced rate of delayed diagnosis as compared to persons in other nations. Merely 50% of patients with kidney tuberculosis had symptoms in postmortem examinations, and only 18% received a clinical diagnosis. Because of this, the disease is not as severe, and a higher percentage of patients arrive at the hospital without any significant upper urinary tract lesions [6]. Renal infection is a highly destructive condition that can cause renal exclusion of the affected side and renal failure as soon as it is diagnosed [7, 8]. Kidney failure can result from the progression of a particular disease, such as the development of renal cavitations, fibrosis, or caseous granuloma formation, or, more frequently, from obstruction of the urine collection system [9-12]. The latter may be proximal in cases of intrarenal stenosis or distal as a result of ureteral stenosis [13]. A descending

infection of the urine collection system is the cause of tuberculosis of the bladder and urethra. Ureteral tuberculosis was discovered in kidney-immunized pigs with total ureter obstruction in an experimental study [12]. Numerous stenoses develop throughout the ureter in ureteral TB, with an emphasis on the vesicoureteral junction [7, 8, 14]. The first inflammatory phase of bladder TB is marked by hyperemia, ulceration, and the formation of tubercles at the ureteral meatus. This phase is followed by bladder wall fibrosis [4-8]. Urethral TB makes up just 1.9-4.5 percent of all urogenital tuberculosis cases due to frequent urethral contact with urine *Bacilli* and it never happens by itself [15]. There are often more symptoms associated with renal TB than fever, weight loss, and night sweats. Individuals may exhibit common symptoms related to the lower urinary tract, which would point to a simple case of bacterial cystitis. Renal colic symptoms are not prevalent. Pyuria and hematuria may be seen in the first urinalysis, but no bacteria. Cultures of urine often yield negative results for common bacterial species. One of the reasons for sterile pyuria is tuberculosis [16]. Renal tuberculosis is often suspected due to renal imaging modalities. On intravenous pyelograms, calyceal deformation and papillary necrosis can be seen. Renal TB is indicated by a markedly abnormal renal shape and many intrarenal calcifications on ultrasonography. Cystoscopy and retrograde pyelography can reveal distinctive anomalies associated with ureteritis cystica, as well as a severely aberrant urinary bladder. Because of ureteral or bladder restriction, hydronephrosis can be seen on any renal imaging test [17]. A trustworthy microbiologic diagnosis can be made by isolating tuberculosis germs from urine or tissue biopsy specimens. On microscopy of centrifuged urine, acid-fast *Bacilli* can be seen. Tubulointerstitial nephritis with granuloma formation is indicated by renal histology and is commonly linked to caseous necrosis.

The gold standard for detecting renal tuberculosis (TB) is urine culture for acid-fast *Bacilli*; this method has 100% specificity but a 30% to 90% sensitivity depending on the number of samples examined. A positive urine culture might come back after six to eight weeks [18]. The outcome of renal TB varies widely. An autonephrectomy, or loss of function on the affected side, may be the outcome of diffuse renal scarring. A cementation or "putty" kidney involving the whole renal pelvis is the hallmark of pyonephrosis, a condition that can result from an intrarenal infection that extends to the renal pelvis. Urine obstruction on that side may develop from broad ureteral strictures brought on by ureteral involvement.

Bladder scarring can block the urine and thus increase the creatinine by deranging the RFTs. It is extremely rare for end-stage renal disease to advance. The cause of fewer than 1% of cases of end-stage renal failure is tuberculosis. Treatment for renal TB involves many drugs. The current four-drug regimen, which consists of an initial two-month intense therapy with rifampin, isoniazid, pyrazinamide, and ethambutol, has evolved from this over time [19]. Rifampin and isoniazid are often part of a four-month maintenance regimen that comes after this. Urologic intervention may be necessary for bladder augmentation or for unilateral sickness characterized by pain or bleeding [20, 21]. When ureteral obstruction is relieved, patients can avoid dialysis by using stenting or percutaneous nephrostomy, especially if their underlying parenchymal renal disease is mild and their residual renal function is greater than 15 mL/min.

CONCLUSION

Thus, it's a very rare case to report to shed light on the never-ending and persistently ruling, the notorious most, tuberculosis, with most of the manifestations in a single patient. Being a biopsy-proven case, it further strengthens the point that in a country like Pakistan, it is still causing morbidity in a large number of patients.

CONSENT FOR PUBLICATION

Informed consent was obtained from the patient of the study.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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Declared none.

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