

Extensive nephroureteric calcification presenting with renal failure: A rare case report

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Abstract

The diagnosis of renal Tuberculosis (TB) can be hypothesized in the setting of non-specific bacterial cystitis associated with a therapeutic failure or a urinalysis with a persistent leukocyturia and absence of bacteriuria. Renal TB is an important cause of kidney disease, mainly in tropical areas of the globe, which can lead to end-stage renal disease if not diagnosed early and treated correctly. We report a case of a 58 year man with a past history of pulmonary TB treated for six months with extensive nephroureteric calcification presenting with renal failure.

Key Words: End-stage renal disease, nephroureteric calcification, renal tuberculosis

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INTRODUCTION

Tuberculosis (TB) is a current public health problem, and the most common world-wide cause of mortality from infectious disease, with an estimated global incidence of 8–10 million/year.^[1-3] The difficulty in diagnosing extra-pulmonary TB can be attributed to the poor access of disseminated lesions, the fact of patients being usually paucibacillary (very often causing a negative smear), histopathologic findings are not pathognomonic (granulomatous reaction can be found in other diseases) and there are lower rates of bacteriological positivity (only in a quarter of the cases).^[4] The diagnosis of renal TB can be hypothesized in the setting of nonspecific bacterial cystitis associated with a therapeutic failure or a urinalysis with a persistent leukocyturia and absence of bacteriuria.^[1,4-6] We report a case of a 58-year-old man with a past history of pulmonary TB treated for 6 months with

extensive nephroureteric calcification presenting with renal failure.

CASE REPORT

A 58-year-old man who presented with anuria was incidentally found to have calcified left kidney on ultrasound examination with right side mild hydrourteronephrosis. His current chest radiography [Figure 1], complete blood count was normal, serum creatinine was 6 mg/dl, and there was a history of pulmonary TB. A plain radiography [Figure 2] revealed a lobulated completely calcified left kidney with calcification of the left ureter. Noncontrast computed tomography (CT) [Figures 3 and 4] showed dense calcification replacing left kidney and confirmed left ureteral calcification with a small (4 mm) obstructing calculus in right mid ureter. Intra-operatively during ureterorenoscopy, a long segment right mid ureteric stricture with a small calculus proximal to the stricture was identified, guide wire was passed, and double-J stenting [Figure 5] was done. Three-dimensional reconstructed computed tomography scan image showing calcified left kidney and left upper ureter [Figure 6].

DISCUSSION

Genitourinary tract is the second most common site of TB


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Figure 1: X-ray chest PA view showing normal findings



Figure 2: X-ray kidney-ureter-bladder showing a lobulated completely calcified left kidney with calcification of the left ureter

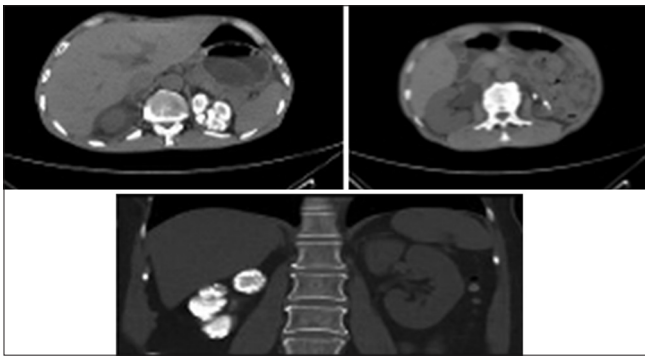


Figure 3: Noncontrast computed tomography scan (axial and sagittal views) showing dense calcification replacing left kidney and confirmed left ureteral calcification

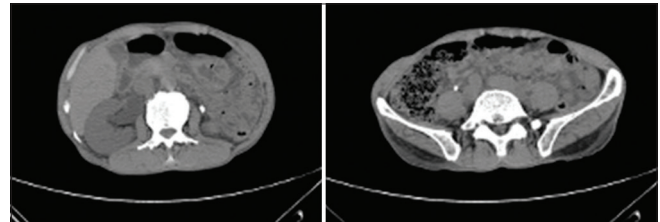


Figure 4: Noncontrast computed tomography scan (axial views) showing Right hydronephrosis with a small (4 mm) obstructing calculus in right mid ureter



Figure 5: X-ray kidney-ureter-bladder showing placement of right double-J stent following ureteroscopic lithotripsy



Figure 6: Three-dimensional reconstructed computed tomography scan image showing calcified left kidney and left upper ureter

after lungs. Males more commonly infected than females. Active pulmonary TB seen in only 4-8% of cases. Begins as small tubercle and extends to renal tubules and medulla as necrotizing lesions, produce larger cavities which communicate with collecting system. Strictureing eventually results in a fibrotic

and small kidney. Genitourinary TB (GUTB) is caused by metastatic spread of organisms through the blood stream during the initial infection. Active disease results from the reactivation of the initial infection.^[7] TB may involve the kidney as part of generalized disseminated infection or as localized genitourinary disease. Clinically, renal TB usually presents unilaterally, but postmortem studies showed that the disease is often bilateral. The healing process results in fibrous tissue and calcium salts being deposited, producing the classic calcified lesion.^[7] In this case, the patient had unilateral renal involvement. He

had a past history of pulmonary TB, which was treated over a 6-month period. One has to be aware that the latency between pulmonary manifestation and GUTB is enormous. In some cases, it could take more than 30 years before GUTB becomes evident.^[8] Clinical presentation varies from hematuria which may be microscopic or macroscopic, Calculus formation, frequency, dysuria and urgency, anuria, sterile pyuria. The diagnosis was possible because the serum adenosine diaminase was compatible, despite cultures being negative. Furthermore, the typical sterile pyuria was found, and skin tests were strongly positive. 75% of patients have an abnormal chest roentgenogram on admission. 88% of patients tested have positive skin tests and 63% tested have abnormal excretory urography. 16% show renal calcification.^[9] The diagnosis of GUTB can be based on culture studies. Tubercular granulomas in the renal pyramids coalesce to form ulcers which discharge mycobacteria and pus in the urine. Untreated lesions enlarge, and a tubercular abscess may form in the parenchyma. Later on, perinephric abscess is formed, and the kidney is replaced by caseous material (putty kidney) which may become calcified (cement kidney) and nonfunctional leading to renal failure.^[2] In patients with healed or chronic TB, calcifications may be noted. Renal calcifications are a common manifestation of TB at conventional radiography, occurring in 24-44% of patients. Extensive parenchymal calcification in a nonfunctioning, autonephrectomized kidney is characteristic of end-stage TB.

Ultrasonography may show hypoechoic masses with hydronephrosis. Plain X-ray kidney-ureter-bladder film may show large globular, amorphous calcifications or smaller nondescript stones. Intravenous pyelography or, more recently, CT-urograms can be diagnostic, which may show the following findings:

- Affected kidney may show contrast enhancement on CT
- Renal calcification is common (24-44%). Stones, focal or extensive globular calcification, ring-like calcifications of papillary necrosis
- Cortical scarring
- “Smudged” papillae which appear irregular (moth-eaten appearance) due to inflammation and necrosis
- Several cysts surrounding a calyx with cortical thinning
- Infundibular strictures
- Hydrocalyces without dilatation of the renal pelvis or hydronephrosis
- “Putty kidney” – sacs of caseous, necrotic material
- Small, shrunken kidney with dystrophic calcification (autonephrectomy)

Whenever a pattern of chronic renal inflammatory disease is recognized, particularly in the setting of periureteric or peripelvic fibrosis, TB must be considered.^[2] The differential diagnosis for the imaging appearance of renal TB includes chronic pyelonephritis, papillary necrosis, medullary sponge kidney, calyceal diverticulum, renal cell carcinoma, transitional cell carcinoma, and xanthogranulomatous pyelonephritis.

Anti-tubercular treatment (ATT) must be given for 6 months. With ATT, mortality is low (2%). About 55% of patients with GUTB will require surgical intervention in the form of ureteral stenting, percutaneous nephrostomy, partial nephrectomy, nephroureterectomy. The rate is lower in areas where the disease is diagnosed early while still asymptomatic.^[10] If untreated, end result is autonephrectomy. It may also lead to sinus and fistula tract formation and infertility in females.

CONCLUSION

Renal TB is an important cause of kidney disease, mainly in tropical areas of the globe, which can lead to end-stage renal disease if not diagnosed early and treated correctly.

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