

Putty kidney without pulmonary tuberculosis

Radiodiagnosis

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KEYWORDS:

SUMMARY

A 40-year-old woman presented with a 2 month history of a dull ache in her right flank along with recent onset of hypertension. There was no history suggestive of tuberculosis and examination was unremarkable. X-ray showed dense opacity in right renal area. CT SCAN UROGRAPHY (PLAIN+CONTRAST) was done.

BACKGROUND

Tuberculosis is one of the most common causes of mortality from infectious diseases especially in developing countries. Though pulmonary tuberculosis is the most common manifestation, renal tuberculosis is not uncommon in areas endemic to tuberculosis; however, presentation usually follows pulmonary tuberculosis. The consideration of renal tuberculosis in absence of any history of pulmonary tuberculosis is difficult and often misleads to the diagnosis of renal stone.

CASE PRESENTATION

A 40-year-old woman presented to us with a 2 month history of a dull aching right flank pain. There were no exacerbating or relieving factors. The pain did not radiate. There was no history of fever, chronic cough, anorexia and weight loss. There was no history of haematuria or dysuria. She did not have any bowel symptoms. She had a history of hypertension of recent onset. There was no family

history of tuberculosis. On examination the only finding was a tender and palpable right kidney and tender right renal angle. Chest examination was unremarkable.



Figure 1

INVESTIGATIONS

Her haemoglobin was 11 g/dl and total leucocyte count was 9100/mm³ of blood with a lymphocyte count of 18%. Her serum creatinine was 0.9 mg/dl and blood urea was 42 mg/dl. Her blood pressure was 156/98 mm Hg while she was on a once daily dose of 5 mg of amlodipine

(calcium channel blocker) and 5 mg of enalapril (ACE inhibitor). Urine examination was unremarkable and a chest x-ray did not reveal any abnormality.

X-ray kidney, ureters and bladder (KUB) showed dense reniform opacity in right lumbar area representing calcified right kidney (figure 1).



Figure 2

Ultrasonography of the abdomen showed large echogenic mass in right renal area with strong posterior acoustic shadow confirming calcified right kidney (figure 2).

A non-contrast CT revealed completely calcified right kidney measuring

(67 x 39) mm².

Left kidney was enlarged measuring (110 x 50) mm² representing compensatory hypertrophy (figure 3A,B).

Contrast-enhanced tomography in arterial phase showed normally enhancing left kidney and non-enhancing right kidney. Films obtained in delayed phase showed normal excretion of dye from left kidney and non-excretory right kidney

(figure 4A,B,C).

A subsequent urine examination for the presence of acid-fast bacilli was unsuccessful and a culture did not yield any growth.

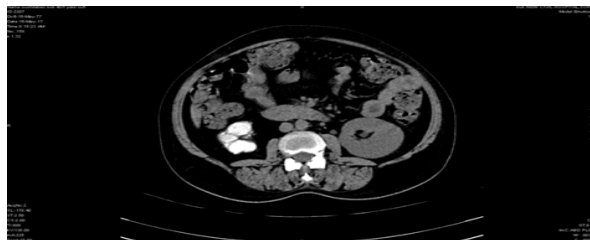


Figure 3 A



Figure 3 B

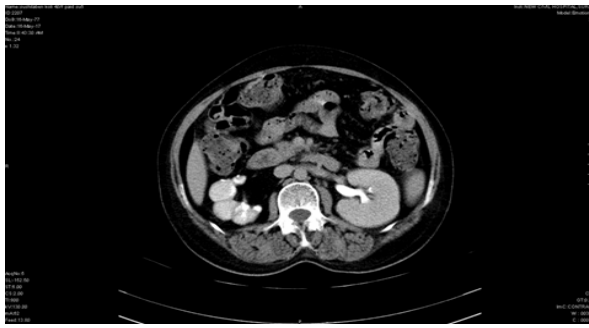


Figure 4A



Figure 4B



Figure 4C

DIFFERENTIAL DIAGNOSIS

A diagnosis of renal tuberculosis leading to putty kidney (autonephrectomy) was made. The only differential considered in this case was a large calculus occupying the whole kidney but was ruled out after CT. Diagnostic difficulty arises when disease is localised with small areas of calcifications. Renal calculus disease is important consideration. Xanthogranulomatous pyelonephritis may present as segmental calcification. Stippled calcification may suggest an underlying transitional cell carcinoma. But confluent parenchymal calcification is almost never found in any of these. Calyceal blunting without scarring and pelvic dilation indicates papillary necrosis and a differential of analgesic nephropathy and haemoglobinopathies must be kept in mind. Both of these usually do not show calcification and calyceal truncation.

TREATMENT

After reviewing the available literature, she was started on antituberculous therapy (ATT) along with antihypertensive agents and advised monthly follow-up. When 2 months of treatment failed to resolve her flank pain, it was decided to go ahead with the surgical nephroureterectomy. The kidney was approached by a left lumbar incision. Intraoperatively the left kidney and ureter was filled with calcific caseous material. There was dense fibrosis around the renal pedicle but separate control of renal artery and vein was achieved. There was no breach of peritoneal cavity during the procedure. The postoperative period was uneventful. The histopathology showed the kidney with extensive necrosis, fibrosis, thyroidisation of tubules and focal collection of foamy macrophages. No definitive granulomas were seen. Sections of ureter also show luminal calcification.

OUTCOME AND FOLLOW-UP

The patient was advised to continue ATT for 1 year and monthly

follow-ups for initial 2–3 months. Her flank pain disappeared after surgery. By the end of 1 year, the antihypertensive agents were withdrawn. She was able to maintain her blood pressure at around 140/90 mm Hg without any medication in subsequent follow-up visits

DISCUSSION

Tuberculosis is still one of the most common causes of mortality from infectious diseases across all age groups worldwide especially in low-income and middle-income countries.¹ The emergence of *Mycobacterium tuberculosis* resistant to conventional multidrug therapy as well as opportunism in HIV infection has further complicated the issue.

Renal tuberculosis is common in areas endemic to tuberculosis and accounts for 25–30% cases of extra pulmonary tuberculosis⁴ but presentation is usually preceded by a history of pulmonary tuberculosis. The consideration of renal tuberculosis in absence of any history of pulmonary tuberculosis is difficult and often misleads to the diagnosis of renal stone and there are reported incidences of the same. Genitourinary tuberculosis is believed to be because of haematogenous dissemination of *M tuberculosis* from lungs and involves both the kidneys where the bacteria get seeded in the glomerular and peritubular capillary bed. Bacteria then proliferate and form granulomas in renal cortex, which may remain dormant for a long period. The medullary hypertonicity impairs the phagocytic function. The clinically apparent disease develops in the kidney when capillaries rupture and the bacteria is then delivered into the proximal tubules.

Advanced disease leads to cortical scarring and strictures may develop involving the infundibulum and pelvicalyceal system. The end result is destruction, loss of function and calcification and if not timely intervened, the entire kidney may be involved.

Usual presentation closely mimics cystitis or lower urinary tract infection. Suspicion arises when these symptoms fail to respond to usual treatment and pyuria persists and no organism grows on routine culture media. In absence of pulmonary tuberculosis, patients usually present late and a diagnosis is made very late when whole kidney is already involved, as it happened in this case.

The radiological findings in renal tuberculosis depend on the extent of the disease process and may vary from amorphous to granular to lobar patterns.

Early findings are best demonstrated on contrast-enhanced tomography which is replacing intravenous urography and retrograde pyelography as the investigation of choice.

The earliest imaging abnormality arises because of papillary necrosis leading to calyceal irregularity providing moth-eaten appearance. Subsequent findings include dilation of one or more calyces because of infundibular stricture, phantom calyx (non-visibility of calyx) owing to complete infundibular stricture, small and contracted renal pelvis with sharp angulations (Kerr Kink⁸) and later on putty-like calcification characteristic of terminal stage of renal tuberculosis.

Cavities lined with granulations, discharging sinuses opening into renal pelvis, areas of caseous necrosis, fibrosis and pyonephrosis are the usual histopathological findings. The end stage kidney shows large cavities filled with inspissated putty-like substance mixed with calcium salts. Renal tuberculosis should be managed with standard short

course ATT which includes 2 months of extensive phase with four drugs (isoniazid, rifampicine, pyrazinamide, ethambutol: HRZE) followed by 4 months of maintenance phase with two drugs (HR). This alone should be able to sterilise the kidney and prevent the reactivation.^{15 16} However, routine follow-up

visits have been suggested to see if there is any increase in the size of calcification. Partial or polar nephrectomy is indicated if there is a localised lesion in one pole of kidney that has failed to respond to 6 weeks of standard ATT. Partial nephrectomy is still justified because the histopathological evidence of *Mycobacterium* have been found in the resected specimens even after adequate course of ATT. Management of putty kidney with autonephrectomy is yet not well established and highly controversial. The available literature suggests that medical management

can avoid the surgical trauma and the proponents say a long-term (2 year) course of ATT will sterilise the kidney preventing the chances of reactivation. Those who favour surgical management propose the zero chances of reactivation and

no issue of non-compliance to medical therapy as the total duration is shortened and may help a hypertensive patient to bring to normal if it is owing to unilateral non-functioning kidney. Currently the indications for nephrectomy

include the secondary infection and abscess formation, suspicion of malignancy, hypertension attributed to the pathology and recurrent urinary tract infection.

• **LEARNING POINTS**

- ▶ Diagnosis of extra pulmonary tuberculosis is often delayed if there is no evidence of pulmonary tuberculosis and should always be kept in mind especially in areas endemic to tuberculosis.
- ▶ A standard regimen of anti tuberculous therapy should be initiated in all cases of diagnosed genitourinary tuberculosis.
- ▶ Surgical management should be instituted if there is no response to chemotherapy, in presence of persistent symptoms and hypertension or if there are complications.

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