Tuberculosis of the genitourinary tract: imaging features with pathological correlation

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ABSTRACT

The prevalence of pulmonary and extrapulmonary tuberculosis (TB) has been increasing over the past decade, due to the rising number of people with acquired immunodeficiency syndrome and the development of drug-resistant strains of Mycobacterium tuberculosis. The genitourinary tract is the most common site of extrapulmonary TB. Diagnosis is often difficult because TB has a variety of clinical and radiological findings. It can mimic numerous other disease entities. A high level of clinical suspicion and familiarity with various radiological manifestations of TB allow early diagnosis and timely initiation of proper management. This pictorial essay illustrates the spectrum of imaging features of TB affecting the kidney, ureter, bladder, and the female and male genital tracts.

Keywords: bladder infection, genitourinary tract, genitourinary tuberculosis, kidney infection

INTRODUCTION

Tuberculosis (TB) remains the most common worldwide cause of mortality from infectious diseases. Approximately 95% of cases occur in developing countries. However, the prevalence of TB has increased over the past decade in most developed countries due to human immunodeficiency virus (HIV) infection, immigration, and the development of drug-resistant strains of Mycobacterium tuberculosis. Extrapulmonary TB represents a progressively greater proportion of new cases and the genitourinary tract is the most common site of extrapulmonary TB. Diagnosis is often difficult and delayed because TB has a variety of clinical and radiological presentations. It can mimic numerous other disease entities. A high level of clinical suspicion and familiarity with various radiological features of TB allow early diagnosis and timely initiation of proper management, hence reducing patient morbidity. This pictorial essay illustrates the spectrum of imaging features of TB affecting the kidney, ureter, bladder, and female and male genital tracts.

RENAL TB

Although renal TB is usually spread haematogeneously from the lung, radiographical evidence of pulmonary TB is present in less than 50% of the patients and in only 10% of these cases is the disease active. Renal TB is usually the sequelae of a primary pulmonary infection that had occurred as long as 10-15 years earlier. Tubercle bacilli lodge in the corticomedullary junction and form cortical granulomas. These granulomas remain stable for many years, but if reactivation occurs, the organisms spread into the medulla causing papillitis. As the disease progresses, extensive papillary necrosis may develop with the formation of frank cavities destroying the renal parenchyma and may extend into the collecting system. Advanced disease leads to cortical scarring, and infundibular and pelvic strictures. Single or multiple calyces may be involved in one or both kidneys. The end-result of diffuse disease is destruction, loss of function, and calcification of the entire kidney.
patterns. Diffuse, uniform, extensive parenchymal calcifications forming a cast of the kidney with autonephrectomy are called “putty-like calcifications” (Fig. 2), characteristic of end-stage renal TB(3,4). Early findings are best demonstrated on intravenous urography (IVU) or retrograde pyelography (RP). IVU remains the initial imaging of choice in most patients although 10-15% of patients may have normal IVU findings(3-5). The earliest radiographical abnormality is irregularity (moth-eaten appearance) of the calyx (Fig. 3) due to papillary necrosis. Infundibular stricture results in calyceal dilatation (Figs. 3-8). The entire calyx may not be seen (phantom calyx) (Fig. 3) if the infundibular stricture is complete. The renal pelvis is typically small and contracted (Figs. 3-8).
Fig. 5 (a) Unenhanced axial CT image of the same patient as in Fig 4 shows enlargement of the left kidney, markedly dilated calyces, parenchymal thinning with small calcifications (arrows), and a left psoas abscess (asterisk). (b) Enhanced axial CT image shows enhancement of the thin left renal parenchyma with severe caliectasis and a contracted renal pelvis. Peripheral enhancement of the left psoas abscess (asterisk) is also shown. The right kidney shows good excretion of contrast agent, with no excretion from the left kidney. (c) Enhanced axial CT image obtained more distally shows extension of the left psoas abscess to the left flank (arrow).

Fig. 6 32-year-old man with TB of the left urinary tract, urinary bladder and right ureter. (a) Enhanced axial CT image shows a heterogeneous low-density mass in the left kidney (arrow) and a dilated right pelvocalyceal system. (b) Enhanced coronal MIP reconstructed CT image shows dilated left calyces, thickened wall of the left ureter and urinary bladder (arrows). The right renal pelvis and right ureter are dilated. (c) Cystography shows a mildly irregular bladder wall and vesicoureteric reflux of contrast agent up an irregular right ureter. Left vesicoureteric reflux (arrow) is also faintly opacified.
Fig. 7 38-year-old man with TB of the left kidney and ureter. Left RP shows marked dilatation of the calyces with a contracted renal pelvis and irregularity of the ureter.

Fig. 8 40-year-old man with late-stage TB of the left urinary tract, urinary bladder, and seminal vesicle. (a) Enhanced coronal MIP reconstructed CT image shows markedly dilated calyces with thinned parenchyma of the left kidney due to contracted renal pelvis and infundibular stenoses. The right renal pelvis and right ureter (arrows) are markedly dilated due to stenosis at the right ureteric orifice. (b) Gross specimen of the left kidney shows marked calyceal dilatation with thickened irregular walls (arrows). The renal pelvis is contracted. (c) Enhanced axial CT image of the lower pelvis shows a contracted urinary bladder, dilated right distal ureter, and a stricture of the right ureteric orifice (arrow). (d) Unenhanced axial CT image of the lower pelvis shows a calcified left seminal vesicle (arrow).
Computed tomography (CT) is the most sensitive method to demonstrate renal calcification (Fig. 5a), which occurs in approximately 40-70% of cases. Other CT features include parenchymal scarring, low-attenuation parenchymal lesions (Fig. 6a), and hydronephrosis (Figs. 5b, 6b & 8a). CT is also helpful in determining the extent of renal and extrarenal spread of disease (4-9) (Figs. 5b-c). However, CT is less sensitive than urography in the detection of early uroepithelial mucosal change. Ultrasonography (US) is useful in measuring the renal size and showing the dilated calyces with irregular walls and debris (Fig. 4b). However, US is less sensitive than either urography or CT in the evaluation of renal TB(3,5).

URETERIC TB
Ureteric TB is the result of renal TB that spreads down the collecting systems from the renal pelvis. Early ureteric infection produces ulcerations causing mucosal irregularity (Fig. 7). Healing of these ulcers results in ureteric fibrosis (Fig. 6b). Multiple strictures may produce alternating segments of dilatation and narrowing, giving a beaded appearance which is the characteristic appearance of ureteric TB. The ureter may be shortened and straightened, producing a pipe-stem appearance. Ureteric abnormalities are radiologically demonstrated in approximately 50% of patients with renal TB. The lesions may be demonstrated on IVU if renal function is sufficient; otherwise, retrograde or antegrade pyelography may be necessary(6,7,9).

BLADDER TB
Early TB cystitis produces mucosal ulceration and oedema. Urography or US may demonstrate diffuse irregular wall thickening. Mucosal oedema at the trigone can cause ureteric obstruction. In advanced disease, inflammation progresses to involve the muscularis layer. Subsequent mural fibrosis leads to a thickened and contracted bladder (Fig. 8c). Therefore, the most common manifestation of TB cystitis is a reduced bladder capacity with wall thickening. Ureterovesical reflux (Fig. 6c) may also be seen due to gaping of the ureteric orifice secondary to fibrosis in the region of the trigone(6,10). In rare cases, calcifications of the bladder wall may be seen, which must be differentiated from other causes of bladder calcifications, such as schistosomiasis of bladder, cytoxan cystitis, radiation-induced bladder calcification, calcified bladder carcinoma, or encrusted foreign body(9,11).

FEMALE GENITAL TB
The fallopian tubes are affected in 94% of women with genital TB. There is almost always bilateral involvement. Salpingitis results from haematogenous infection. Spread from TB salpingitis can cause peritonitis, endometriosis, or rarely, cervicitis and vaginitis. Hysterosalpingography is recommended to identify the lesions. The characteristics of TB salpingitis include obstruction of the fallopian tube, multiple constrictions of the fallopian tube, endometrial adhesions with deformity and obliteration of the endometrial cavity (Fig. 9), and calcified lymph nodes in the adnexal region(9).

MALE GENITAL TB
TB involvement of the prostate and seminal vesicles are usually secondary to infection from the upper genitourinary tract and may cause a variety of changes such as necrosis, calcification (Fig. 8d), caseation, and cavitation(11). TB epididymo-orchitis
occurs either from haematogeneous spread or retrograde extension from the prostate and seminal vesicles. It may less frequently be transmitted sexually or disseminated during intravesical bacillus Calmette-Guerin therapy for superficial bladder cancer. Infection usually affects the epididymis first. The testicular involvement occurs by contiguous spread from epididymitis, particularly if appropriate treatment is not initially given. TB epididymo-orchitis may be either unilateral or bilateral\(^{(12-14)}\).

On US, TB epididymitis is seen as diffusely enlarged homo- or heterogeneously hypoechoic, and nodular enlarged heterogeneously hypoechoic lesions (Figs. 10, 11a & 13b). US features of TB orchitis include diffusely enlarged homo- or heterogeneously-hypoechoic testis, nodular enlarged testis with a heterogeneously-hypoechoic texture (Figs. 10, 11a, 13b & 14b), and multiple small hypoechoic nodules in the enlarged testis (Fig. 12) producing a miliary pattern. Other US features of scrotal TB include thickened scrotal skin, hydrocoele (Figs. 12, 13a & 14b) calcifications of the epididymis (Fig. 13b) and tunica vaginalis (Fig. 14b), scrotal abscesses, and scrotal sinus tract (Fig. 14).

The US features of TB epididymo-orchitis must be differentiated from non-tuberculous infection and tumour. The presence of skin thickening and epididymal involvement in conjunction with testicular lesion are suggestive of an infection rather than a tumour because orchitis is almost always caused by epididymitis, while even advanced testicular tumour may only partially involve the epididymis. Non-tuberculous
epididymitis is more likely to be homogeneous (Fig. 15), whereas TB epididymitis is usually heterogeneous or nodular. Failure of conventional antibiotic therapy with the presence of the above-mentioned US features is also suggestive of TB epididymo-orchitis.

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REFERENCES
Multiple Choice Questions (Code SMJ 200510B)

Question 1. Concerning renal TB:
(a) It is usually spread haematogeneously from pulmonary TB. □ □
(b) Evidence of pulmonary TB is present in most cases. □ □
(c) Tubercle bacilli lodge in the corticomedullary junction and form granulomas. □ □
(d) Cortical granulomas may remain stable or cause papillitis. □ □

Question 2. Concerning renal TB:
(a) The earliest radiographical abnormality is irregularity of the calyx. □ □
(b) Calyceal dilatation with infundibular stricture is suggestive of TB. □ □
(c) Abdominal radiograph is the most sensitive method to demonstrate renal calcification. □ □
(d) CT is the most sensitive method to detect early uroepithelial mucosal change. □ □

Question 3. The following statements are correct:
(a) Ureteric infection produces mucosal irregularity. □ □
(b) Ureteric TB typically causes a hydroureter. □ □
(c) The most common manifestation of TB cystitis is a contracted bladder. □ □
(d) TB cystitis may cause ureterovesical reflux. □ □

Question 4. The following statements are correct:
(a) Approximately 30% of patients with renal TB have a normal IVU. □ □
(b) US is less sensitive than IVU and CT in the evaluation of renal TB. □ □
(c) “Putty-like calcifications” are characteristic of end-stage renal TB. □ □
(d) Fallopian tube is the most common affected site of genital TB in women. □ □

Question 5. Concerning male genital TB:
(a) TB epididymo-orchitis may result from haematogeneous spread or retrograde extension from the prostate and seminal vesicles. □ □
(b) Intravesical bacillus Calmette-Guerin therapy for superficial bladder carcinoma may also cause TB epididymo-orchitis. □ □
(c) Heterogeneous nodular enlargement of the epididymis with calcification is suggestive of TB. □ □
(d) Isolate orchitis is common in TB due to haematogeneous spread. □ □

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