

Urogenital tuberculosis

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INTRODUCTION

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Urogenital tuberculosis (TB) is the third most common form of extrapulmonary TB (after lymph node involvement and tuberculous pleural effusion) [1]. Urogenital TB occurs in 2 to 20 percent of individuals with pulmonary TB [1-3]. Among patients with miliary disease, hematogenous seeding of the urogenital tract occurs in 25 to 62 percent of cases [1]. In one review including more than 9000 patients with TB, urogenital TB affected two males to each female, with a mean age of 40 years (range 5 to 90 years) [4]. Up to 50 percent of patients have radiographic evidence of past infection and 10 percent may have active infection [5]. Diagnosis of urogenital tuberculosis is often overlooked and a high index of suspicion is necessary for prompt diagnosis.

Issues related to urogenital TB are reviewed here; issues related to pulmonary and other forms of TB are discussed separately. (See related topics.)

Rarely, tuberculous bacilli may enter the urinary tract via intravesical instillation of the attenuated live *Bacillus Calmette-Guérin* (BCG) to treat bladder cancer; this is discussed separately. (See "[Infectious complications of intravesical BCG immunotherapy](#)".)

PATHOGENESIS

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Issues related to pathogenesis of urogenital TB are discussed here; general issues related to pathogenesis of TB are discussed separately (see "[Tuberculosis: Microbiology, pathogenesis, and immunology](#)").

Renal and urologic tuberculosis

— TB involving the kidney and urologic system has two forms. The most common presentations involve the urinary collecting system (including renal pelvis, calyces, ureters, and bladder). Less commonly, kidney lesions occur; these may include ulcers, abscess formation, perinephric spread, caseating necrosis, and calcification. Diffuse parenchymal lesions such as granulomatous interstitial nephritis and glomerulonephritis have also been described [6].

Classical presentation

— Mycobacterial seeding of the urogenital tract via hematogenous spread can occur at the time of primary pulmonary infection, in the setting of reactivation, or miliary disease. Bacilli from regional lymph nodes enter the bloodstream through the thoracic duct and then disseminate to the kidneys.

The inflammatory response induces formation of a cortical granuloma, which may heal and form a microscopic scar. Organisms may remain dormant for many years or rupture into the proximal tubule of the nephron with excretion of tuberculous bacilli into the urinary tract, leading to contiguous spread of infection ([figure 1](#)) [7,8]. The bacilli in the nephron are trapped at the loop of Henle where they multiply. The combination of relatively poor blood flow, hypertonicity, and high ammonia concentration in the renal medulla impair immune responses and favor formation of medullary granulomas or chronic granulomatous tubulointerstitial nephritis.

Subsequently, destruction of renal papilla can lead to abscess formation. Descending spread of infection to the ureter and bladder causes diffuse or multiple ureteral strictures and obstruction, hydronephrosis, perinephric abscess, and impairment of kidney function [9,10]. The radiographic features of ureteric involvement may include segmental ureteric strictures and dilatations ('corkscrew' ureter), or shortened rigid ureter ('pipestem' ureter).

Renal TB can be classified as follows [11]:

- Stage 1 – Nondestructive form (TB of parenchyma)
- Stage 2 – Small destructive form (TB papillitis)
- Stage 3 – Destructive form with one or two caverns (cavernous renal TB)
- Stage 4 – Widespread destructive form (polycavernous renal TB)

Dystrophic calcification may result in a nonfunctional calcified kidney called 'putty kidney' or 'cement kidney.' Since organisms survive in the calcified lesions, calcification reflects active disease and not a sign of healing.

Unilateral kidney involvement is generally more common than bilateral involvement [8,12]. However, both kidneys may be involved, which may result in end-stage kidney disease.

Bilateral renal TB may occur via three mechanisms:

- Hematogenous spread initially affects one kidney with contiguous spread to the ipsilateral ureter and bladder. As a result of fibrosis and contraction of the bladder, patients secondarily develop contralateral vesicoureteral reflux with retrograde spread to the contralateral kidney.
- Hematogenous spread to both kidneys. This occurs in patients with immunodeficiency and miliary TB [13].
- Reactivation of bilateral kidney foci, with spread to the collecting system and bilateral ureteral strictures, in the absence of bladder contraction. This presentation is extremely rare [14,15].

In the bladder, hyperemia and ulceration result in distortion of the ureterovesical junction; lateral displacement and enlargement of the ureteral orifice may occur ('golf hole' ureter). Ulcers with granulomatous changes involving all layers (pancystitis), bladder fibrosis, and contraction ('thimble bladder') may also occur. Rare sequelae of bladder involvement include fistulae formation.

Uncommon kidney manifestations

— Rare conditions associated with TB include interstitial nephritis, glomerulonephritis, and amyloidosis.

Interstitial nephritis

— The pathogenesis of TB-associated interstitial nephritis is not clear. At least in some cases, interstitial nephritis may be an immunologic phenomenon caused by TB involvement of other organs rather than the direct result of TB infection in the kidney [16]. In a case series from the United Kingdom that included 17 patients with known TB who underwent a kidney biopsy, chronic granulomatous interstitial nephritis was present in all [6]. However, although the biopsy showed interstitial inflammation with eosinophilia and granulomas, none of the biopsy specimens was positive for acid-fast stain or mycobacterial culture; in addition, polymerase chain reaction (PCR) was negative for mycobacterial DNA in all five cases in which it was tested.

Interstitial nephritis that develops after the initiation of antituberculous therapy may be attributable to a paradoxical response (ie, the development of new lesions or the worsening of existing lesions after initiation of therapy), which may occur as a result of improved immunologic function resulting in an increase in inflammatory response to infection [17,18].

Acute allergic interstitial nephritis may also occur as a result of drugs used to treat TB, such as rifampin. (See "Clinical manifestations and diagnosis of acute interstitial nephritis", section on 'Drugs'.)

Glomerulonephritis

— Several case reports describe glomerulonephritis associated with TB [19-21]. Glomerulonephritis appears to be directly related to TB infection of the kidney. In a retrospective study including 46 Chinese patients with tuberculous glomerulonephritis, a positive urine mycobacterial culture was observed in 20 percent of cases, and kidney biopsy was PCR positive for TB in 85 percent of cases [19]. Seventy-six percent had a history of pulmonary or extrapulmonary TB.

Amyloidosis

— TB can also cause renal amyloidosis [22,23]. Amyloidosis is a glomerular lesion that results from amyloid deposition in the kidney (see "Renal amyloidosis"). In TB, renal amyloidosis is due to chronic inflammation, with high circulating levels of the acute-phase reactant, serum amyloid A protein [22,23].

Genital tuberculosis

— TB affects the entire male genital tract, including the prostate, seminal vesicles, vas deferens, epididymis, testicles, Cooper glands, and penis. Genital TB occurs via hematogenous spread to the prostate and epididymis or via the urinary tract to the prostate with spread from the ejaculatory ducts to the seminal vesicles, vas deferens, and epididymis [1].

TB of the female genital tract usually occurs via hematogenous spread from the lungs; less commonly, it occurs via lymphatic spread from other abdominal organs [24,25]. TB affects the fallopian tubes, endometrium, and ovaries but generally spares the myometrium [2].

Primary TB of the female genital tract can develop after sexual intercourse with a man with TB of the penis or epididymis [1]. Female-to-male sexual transmission of TB has not been described in the literature.

Vertical transmission of TB is rare; risk factors include maternal extrapulmonary, miliary, and meningeal TB [1]. Transmission may occur transplacentally via the umbilical veins to the fetal liver and lungs or via aspiration of infected amniotic fluid. The risk of vertical transmission can be reduced with timely administration of maternal antituberculous therapy.

CLINICAL MANIFESTATIONS AND DIAGNOSIS

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The onset of TB of the genitourinary tract is usually insidious. The average period between pulmonary infection and clinical manifestations of urogenital TB is 22 years (range 1 to 46 years) [8]. Urogenital tuberculosis is rare in children; the typical age range is 20 to 40 years and the male to female ratio is 2:1.

Renal and urologic tuberculosis

— An understanding of the pathogenesis of infection is important for understanding the range of clinical manifestations of renal and urologic TB. (See '[Pathogenesis](#)' above.)

Presentation

— Initially, renal and urologic TB are not associated with specific symptoms; pyuria and/or microscopic hematuria may be observed as incidental findings. Once the disease has progressed to involve the bladder, symptoms of frequency, dysuria, urgency, and nocturia occur in approximately half of cases; gross hematuria and low back pain develop in approximately 40 percent of cases [[1,7,8,26,27](#)]. Long-standing parenchymal involvement may result in tubular proteinuria, which is often in subnephrotic range. Systemic symptoms of fever and weight loss are relatively rare [[7,9](#)]. Manifestations of advanced disease include end-stage kidney disease and, occasionally, refractory hypertension [[28,29](#)].

HIV-infected patients are younger and more prone to developing tuberculous kidney and prostate abscess than HIV-uninfected patients [[30](#)]. In one series including 24 patients with urogenital TB, HIV infection was present in two-thirds of cases [[30](#)]. An autopsy study including 46 patients with AIDS noted bilateral renal granuloma in 24 percent of cases [[31](#)].

Characteristic laboratory findings include persistent pyuria and acidic urine in the setting of urine cultures that are repeatedly negative for pyogenic organisms ("sterile pyuria"). Painless macroscopic or microscopic hematuria is present in more than 90 percent of cases [[7](#)]. The plasma creatinine concentration is usually normal in the setting of unilateral kidney involvement. An elevated plasma creatinine concentration may be observed in the setting of bilateral kidney involvement and/or in the setting of interstitial nephritis or glomerulonephritis [[1](#)].

Patients with interstitial nephritis are likely to have white blood cell casts. Patients with glomerulonephritis are likely to have dysmorphic red blood cells and red blood cell casts. (See '[Urinalysis in the diagnosis of kidney disease](#)'.)

Proteinuria is observed in the setting of glomerulonephritis and amyloidosis. In a retrospective study of 46 Chinese patients with tuberculous glomerulonephritis, 70 percent of patients had proteinuria and 11 percent had pyuria [[19](#)]. Patients with amyloidosis often have nephrotic range proteinuria (ie, >3.5 grams per day) [[22,23](#)]. (See '[Clinical manifestations and diagnosis of acute interstitial nephritis](#)', section on '[Clinical features](#)' and '[Glomerular disease: Evaluation and differential diagnosis in adults](#)', section on '[Proteinuria](#)' and '[Renal amyloidosis](#)', section on '[Clinical manifestations](#)'.)

Diagnosis

— The diagnosis of renal and/or urologic TB should be suspected in patients with relevant clinical manifestations (urinary frequency, dysuria, hematuria, and/or sterile pyuria) and relevant epidemiologic factors (history of prior TB infection or disease, known or possible TB exposure, and/or past or present residence in or travel to an area where TB is endemic) [[26](#)].

Patients with concomitant clinical or radiographic findings suggestive of pulmonary involvement should also undergo diagnostic evaluation for pulmonary TB; this is discussed separately. (See '[Pulmonary tuberculosis disease in adults: Clinical manifestations and complications](#)' and '[Diagnosis of pulmonary tuberculosis disease in adults](#)'.)

Diagnostic tools for urogenital TB include urine studies, radiographic imaging, [tuberculin skin test](#) or interferon-gamma release assay, and histopathology (in some circumstances).

The diagnosis of urogenital TB may be established by demonstration of tubercle bacilli in the urine. Three to six early-morning urine samples should be sent for acid-fast stain, mycobacterial culture, and polymerase chain reaction (PCR) for *Mycobacterium tuberculosis* if feasible.

- Urine mycobacterial culture – Urine mycobacterial culture has sensitivity up to 90 percent and specificity of 100 percent; results may take six to eight weeks, although automated broth culture should be positive in two to three weeks [[32-34](#)]. Urine acid-fast bacilli cultures have sensitivity of 80 to 90 percent with high specificity [[35](#)]. False-negative results may occur in the setting of concomitant antituberculous or antibacterial therapy capable of inhibiting mycobacterial growth (particularly fluoroquinolones) [[26](#)]. Mycobacteria Growth Indicator Tube culture uses a nutrient-rich liquid culture medium to amplify the growth of mycobacteria in the sample while inhibiting other organisms; the results are usually available in two weeks [[36](#)].

A positive urine acid-fast stain is not diagnostic for TB, since nontuberculous mycobacteria may be present [[26](#)]. The threshold for urine acid-fast stain is 5000 organisms per mL; it has sensitivity of 42 to 52 percent and specificity of 97 percent [[2,37,38](#)].

- Urine PCR – Urine PCR has sensitivity of 87 to 100 percent and specificity of 93 to 98 percent, respectively [[2,38-40](#)]. Some individual laboratories offer validated PCR testing; thus far, there is no commercial PCR test approved by the US Food and Drug Administration for detection of mycobacterial nucleic acid in urine.

Data on use of the GeneXpert MTB/RIF molecular system for diagnosis of extrapulmonary TB are limited. One study of 91 urine samples from patients with suspected TB or nontuberculous mycobacteria infections (including five culture-positive samples) noted sensitivity and specificity of 100 and 98 percent, respectively [[41,42](#)]. Point-of-care versions of the GeneXpert system (GeneXpert Omni, GeneXpert Edge) are available; these are portable and battery-operated.

The urine TB lipoarabinomannan assay is an immunochromatographic assay used for diagnosis of TB in people with HIV and CD4+ lymphocyte counts <200 cells/microL. The utility of this assay for routine diagnostic use has not been established [[43](#)]. (See '[Clinical manifestations, diagnosis, and treatment of miliary tuberculosis](#)', section on '[Urine antigen test in HIV infection](#)'.)

Radiographic imaging is also warranted for patients with suspected renal or urologic TB. Radiographic tools include computerized tomography (CT) with contrast, magnetic resonance imaging (MRI), and high-resolution ultrasound; either may be used based on availability and center preference [[7,27,44-47](#)]. CT provides greater anatomic detail so is preferable when feasible.

In general, radiographic evidence of concomitant upper and lower urinary tract involvement is strongly suggestive of TB. Strictures throughout the collecting system (from the renal pelvis to the ureterovesical junction) are observed in 60 to 84 percent of cases ([image 1](#)) [[48](#)]. Additional manifestations include asymmetric caliectasis

(reflecting uneven infundibular scarring), calcification (throughout the urinary tract) and hydronephrosis. Presence of bladder contraction is a specific finding and represents advanced disease [4]. Extensive kidney damage may result in an autonephrectomized kidney (image 2) [28,46,49,50]. In some cases, TB may be identified incidentally during radiographic examination for an unrelated problem.

Intravenous urography or contrast CT scan provides a simultaneous assessment of kidney function, urinary tract anatomy, and drainage; it allows identification of early changes (such as parenchymal lesions, minimal calyceal dilatation), or advanced changes in the urinary system.

Patients with suspected TB, suggestive radiographic findings, and negative urine studies (mycobacterial culture or PCR) warrant fine-needle aspiration of the pathologic tissue, kidney biopsy, or bladder biopsy for mycobacterial culture and histopathology examination [51]. The selection of biopsy site depends on the predominant clinical features and radiographic findings. Biopsy specimens should be sent for histopathological examination, stain for acid-fast bacilli, and mycobacterial culture. The biopsy specimen is macerated before inoculating into culture medium (since organisms are intracellular) to increase yield.

Patients who have evidence of interstitial nephritis or glomerulonephritis (ie, an active urinary sediment and elevated plasma creatinine concentration) commonly undergo kidney biopsy even in the absence of clinical features suggestive of TB. The diagnosis of secondary amyloidosis is established by demonstrating tissue amyloid deposition on biopsy of the abdominal fat pad, rectum, or kidney [22,23]. (See "The kidney biopsy", section on 'Indications'.)

Tuberculin skin testing or interferon-gamma release assay (IGRA) is warranted. These tests are designed for diagnosis of TB infection; a positive result supports (but cannot be used to establish) a diagnosis of active TB disease, and a negative result does not rule out active TB disease [52-54]. (See "Use of interferon-gamma release assays for diagnosis of tuberculosis infection (tuberculosis screening) in adults" and "Tuberculosis infection (latent tuberculosis) in adults: Approach to diagnosis (screening)".)

In the absence of microbiologic or histologic confirmation, a presumptive diagnosis of urogenital TB may be made based on suggestive clinical, laboratory, and radiographic data.

Male genital tract tuberculosis

Presentation

— TB can affect the entire male genital tract, including the prostate, seminal vesicles, vas deferens, epididymis, testicles, Cooper glands, and penis. (See 'Genital tuberculosis' above.)

Epididymitis is the most common clinical manifestation among men with genital TB; the epididymis is affected in 10 to 55 percent cases [1]. Physical findings consist of scrotal nodule, elevated scrotal skin temperature, or epididymal hardening in up to half of cases [55]. Involvement is bilateral in 34 percent of cases. Scrotal fistula occurs in half of cases, and hydrocele occurs in 5 percent of cases [56].

Prostate involvement is usually subclinical; nodularity may be appreciated on digital rectal examination. Histological involvement occurs in 40 to 50 percent of men with urogenital TB [13,57]. Prostate involvement occurs more commonly via hematogenous spread than through the urinary system [58,59]. Urethritis may occur in association with prostate involvement or chronic urethral strictures and fistulae [60]. Prostate abscess is rare but occurs among patients with AIDS [61].

Male infertility may be a first symptom of genital TB; it can occur as a result of ejaculatory duct stricture with obstruction, oligozoospermia, and low-volume ejaculate [2,62].

Penile TB is rare; it manifests as an erythematous papule that may ulcerate [63]. Therefore, it may be mistaken for more common causes of genital ulcers such as herpes simplex virus or syphilis; failure to respond to treatment for such causes should prompt further investigation [64] (see "Approach to the patient with genital ulcers"). An alternative presentation consists of penis deformity and urethral fistulae caused by infiltration of the cavernous bodies; this may be confused with carcinoma.

Diagnosis

— The diagnosis of male genital TB should be suspected in patients with relevant clinical manifestations (eg, nodular lesions of the scrotum, prostate and/or testis, and/or nonhealing ulcers of the external genitalia) as well as relevant epidemiologic factors (history of prior TB infection or disease, known or possible TB exposure, and/or past or present residence in or travel to an area where TB is endemic) [26].

The diagnostic approach to male genital TB begins with evaluation of the urinary tract as described above. (See 'Diagnosis' above.)

In the absence of evidence for urinary involvement, men with suspected genital TB should undergo biopsy of the involved site; biopsy specimens should be sent for histopathological examination, stain for acid-fast bacilli, and mycobacterial culture.

Female genital tract tuberculosis

Presentation

— TB of the female genital tract involves the fallopian tubes, the endometrial cavity, and the ovaries; cervical and vaginal disease are rare [65]. Genital tract TB has been associated with 0.2 to 21 percent of infertility cases, mostly among women in resource-limited settings [24,25]. (See 'Genital tuberculosis' above.)

The fallopian tubes are affected in 90 to 100 percent of cases, usually with bilateral involvement [2]. Lesions occur most commonly in the ampulla (which has abundant vascular supply), followed by the isthmus. Infection may progress from the fallopian tubes to the endometrium in 50 to 70 percent of cases; the myometrium is rarely affected. Presence of endometrial TB always means that the tubes are infected, but tuberculous salpingitis can exist in the absence of associated endometritis. Ovarian TB usually occurs as a sequela of tubal disease, with development of a tubo-ovarian abscess. Vulvar or vaginal TB is exceedingly rare.

Female genital TB presents clinically as infertility (40 to 76 percent), pelvic or abdominal pain or mass (50 percent) and menstrual disorders (25 percent) [1,24,66-71]. Infertility develops as a result of tubal obstruction or adhesions of the uterine cavity [72].

Diagnosis

— The diagnosis of female genital TB should be suspected in patients with relevant clinical manifestations (infertility, pelvic or abdominal pain, and/or menstrual disorders) as well as relevant epidemiologic factors (history of prior TB infection or disease, known or possible TB exposure, and/or past or present residence in or travel to an area where TB is endemic) [26].

Hysterosalpingogram may demonstrate fallopian tube obstruction or constriction and/or uterine cavity adhesion or deformity [73]. Histopathologic diagnosis may be made via endometrial or fallopian tube biopsy for histology and culture or via mycobacterial culture of menstrual fluid [24,25]. Specimens should be sent for histopathological examination, stain for acid-fast bacilli, and mycobacterial culture.

Immune reconstitution inflammatory syndrome

— Immune reconstitution inflammatory syndrome (IRIS) may occur in early stages of tuberculosis treatment. (See "[Overview of immune reconstitution inflammatory syndromes](#)", section on '[Mycobacterial infections](#)'.)

DIFFERENTIAL DIAGNOSIS

- Epididymitis and urethritis – Epididymitis and urethritis are most commonly caused by *Neisseria gonorrhoeae* and *Chlamydia trachomatis*; these infections are diagnosed by urine nucleic acid amplification tests. Additional considerations related to epididymitis and urethritis are discussed further separately. (See "[Urethritis in adults and adolescents](#)" and "[Acute epididymitis in adolescents and adults](#)".)
- Urethral stricture – Urethral stricture in men is most often due to trauma or instrumentation. The diagnosis is established via urethroscopy and/or urethrogram. (See "[Strictures of the adult male urethra](#)".)
- Prostatitis and benign prostatic hyperplasia – Causes of prostatitis include gram-negative bacteria, *N. gonorrhoeae*, and *C. trachomatis*; the diagnostic evaluation includes urine gram stain, urine culture, and urine nucleic acid amplification tests. The diagnosis of benign prostatic hyperplasia is established based on clinical history and laboratory tests including urinalysis and serum prostate specific antigen. (See "[Acute bacterial prostatitis](#)" and "[Clinical manifestations and diagnostic evaluation of benign prostatic hyperplasia](#)".)
- Malignancy (renal cell carcinoma, testicular tumor) – Suspicion for renal cell carcinoma may be raised based on radiographic manifestations; the diagnosis is usually established via nephrectomy. Testicular tumor usually presents with a painless mass; diagnostic evaluation includes imaging, measurement of serum tumor markers, and resection. (See "[Clinical presentation, diagnosis, and staging of renal cell carcinoma](#)" and "[Clinical presentation, diagnosis, and staging of testicular germ cell tumors](#)".)
- Infertility – Identifiable causes of male infertility include hypogonadism, post-testicular defects, and seminiferous tubule dysfunction. Identifiable causes of female infertility include ovulatory dysfunction, tubal damage, endometriosis, and cervical factors. (See "[Approach to the male with infertility](#)" and "[Female infertility: Evaluation](#)".)
- Bacillus Calmette-Guérin (BCG) cystitis – Cystitis can occur following intravesical BCG instillation for treatment of bladder cancer. Urine mycobacterial culture or polymerase chain reaction may demonstrate *M. bovis*. (See "[Infectious complications of intravesical BCG immunotherapy](#)".)

MANAGEMENT

Clinical approach

— Antituberculous drugs form the mainstay of treatment. In addition, in patients with ureteral stricture and hydronephrosis, early stenting or percutaneous nephrostomy is beneficial in the setting of obstructive nephropathy that is potentially reversible. Surgical interventions for management of urogenital TB include nephrectomy, dilatation or reconstruction of ureteral stricture, and bladder diversion.

These issues are discussed further in the following sections.

Antituberculous therapy

— In general, the approach to antituberculous therapy for urogenital TB is the same as that for pulmonary TB (see "[Treatment of drug-susceptible pulmonary tuberculosis in nonpregnant adults without HIV infection](#)" and "[Treatment of drug-susceptible pulmonary tuberculosis in nonpregnant adults with HIV infection: Initiation of therapy](#)" and "[Treatment of drug-resistant pulmonary tuberculosis in adults](#)").

Issues related to timing of antiretroviral therapy in HIV-infected patients are discussed separately. (See "[Treatment of drug-susceptible pulmonary tuberculosis in nonpregnant adults with HIV infection: Initiation of therapy](#)".)

Antituberculous agents achieve high urinary concentrations and provide a cure rate of over 90 percent with sensitive organisms. In general, no bacilli are detectable in the urine after two weeks of appropriate antituberculous therapy. However, relapse of urogenital TB can occur after initial urine sterilization (see '[Follow-up and relapse](#)' below). Antituberculous therapy often does not restore impaired fertility [74].

Ureteral stenting or percutaneous nephrostomy

— Urinary tract lesions may progress despite appropriate treatment; inflammation may lead to obstruction of the collecting system, bladder contraction, worsening of urinary frequency, and development of kidney function impairment [5,44,75,76].

Therefore, early stenting or percutaneous nephrostomy is reasonable for patients with ureteral stricture, especially if hydronephrosis is also present. Areas with stricture may fibrose and scar when antituberculous therapy is instituted; therefore, early stenting protects the kidney from obstruction. In one study including 77 patients with tuberculous ureteral stricture treated with antituberculous therapy with or without additional intervention (early ureteral stenting or percutaneous nephrostomy), the nephrectomy rate was higher among those who received antituberculous therapy alone (73 versus 34 percent) [76].

The optimal duration for stenting or percutaneous nephrostomy is uncertain; one study noted that strictures usually stabilize in 6 to 12 months [77]. The approach to determining timeframe for device removal is easier with percutaneous nephrostomy than with ureteral stenting. A percutaneous nephrostomy tube may be clamped and antegrade nephrostogram may be performed to confirm normal antegrade drainage. In the setting of persistent obstruction, nephrostomy drainage can continue without a period of undrained obstruction. In contrast, while a ureteral stent is in place, it is not possible to know whether obstruction has resolved; the only measure of success is empiric trial of stent removal.

An alternative to early stenting or percutaneous nephrostomy consists of periodic ultrasonography (eg, monthly for the first 3 months after initiation of therapy and then every 6 months for the next 18 months) [1,76-78]. Progression of radiographic findings while on antituberculous therapy should prompt ureteral stenting or percutaneous nephrostomy.

The likelihood of kidney function recovery in the setting of urinary collecting system obstruction is low [79]. Positive prognostic factors for recovery of kidney function include distal ureteral stricture, cortical thickness greater than 5 mm, and glomerular filtration rate > 15 mL/min; intrarenal stricture is a negative prognostic factor [80].

Surgery

— Surgical interventions for management of urogenital TB include nephrectomy, dilatation or reconstruction of ureteral stricture, and bladder diversion. In general, when warranted, surgery should be deferred until after at least four weeks of antituberculous therapy have been administered [33,81,82].

For patients with unilateral kidney involvement, nephrectomy can be curative of hypertension (in up to 65 percent of cases), eliminate irritative voiding symptoms, and reduce the likelihood of relapse [82,83]. A functional study (such as a nuclear renal scan) may be helpful to guide consideration of nephrectomy. Indications for nephrectomy include [82]:

- Nonfunctioning kidney
- Extensive disease involving the whole kidney, together with hypertension and ureteropelvic junction obstruction
- Coexisting renal carcinoma

For patients with ureteral stricture, management consists of dilatation or reconstructive surgery. Options for reconstruction depend on the location and length of stricture and include ureteral reimplantation, ureteroureterostomy, pyeloplasty, or ureteral substitution [77,84].

For patients with contracted bladder, bladder augmentation or creation of an orthotopic neobladder is warranted to create a low-pressure, high-capacity reservoir [1,57]. There are no studies comparing these approaches; neobladder is favored for patients with pain (suprapubic or perineal) or very small bladder (<15 to 20 mL) [57,85]. (See "Urinary diversion and reconstruction following cystectomy".)

For women with genital TB, surgery is warranted for large tubo-ovarian abscesses. (See "Tubo-ovarian abscess: Management and complications".)

Follow-up and relapse

— Relapse of urogenital TB can occur after initial urine sterilization. Relapse occurs in up to 6 percent of cases after a mean of 5 years of treatment (range 11 months to 27 years); relapse rates among patients who require nephrectomy appear to be relatively low (<1 percent) [5,86-88].

Therefore, surveillance is warranted for patients with renal TB who do not undergo nephrectomy [1,78]. Surveillance should continue for 10 years following completion of antituberculous therapy and should include visits every 6 to 12 months for urine mycobacterial culture and/or urine polymerase chain reaction for *M. tuberculosis* as well as ultrasonography. Patients should also be instructed to seek evaluation for urinary changes or recurrence of presenting symptoms.

SOCIETY GUIDELINE LINKS

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Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Diagnosis and treatment of tuberculosis".)

SUMMARY AND RECOMMENDATIONS

- Renal and urologic TB
 - Pathogenesis (See 'Pathogenesis' above.)
 - Tuberculosis involving the kidney and urologic system occurs via mycobacterial seeding of the urogenital tract via hematogenous spread; this occurs at the time of pulmonary infection, in the setting of reactivation, or miliary disease. Less commonly, kidney parenchymal lesions occur, including

interstitial nephritis and glomerulonephritis.

- Tuberculous bacilli may enter the medullary interstitium and cause formation of granulomas. These may heal with associated fibrosis (in the absence of overt kidney disease) or, many years after initial infection, may break down and rupture into the tubular lumen with excretion of tuberculous bacilli into the urinary tract, leading to continuous spread of infection. Descending spread of infection to the ureter and bladder causes ureteral stricture and obstruction, hydronephrosis, and kidney function impairment.

- **Clinical presentation** (See '[Presentation](#)' above.)

- Initially, renal and urologic TB are not associated with specific symptoms; pyuria and/or microscopic hematuria may be observed as incidental findings. Once the disease has progressed to involve the bladder, symptoms of frequency, dysuria, urgency, and nocturia occur in approximately half of cases; hematuria and low back pain develop in one-third of cases. Systemic symptoms (fever, weight loss) are relatively rare. Manifestations of advanced disease include end-stage kidney disease and, rarely, refractory hypertension.
- Laboratory findings include persistent pyuria and acidic urine in the setting of urine cultures that are repeatedly negative for pyogenic organisms (sterile pyuria). Painless macroscopic or microscopic hematuria is present in more than 90 percent of cases. The plasma creatinine concentration is usually normal in the setting of unilateral kidney involvement. An elevated plasma creatinine concentration may be observed in the setting of bilateral kidney involvement and/or in the setting of interstitial nephritis or glomerulonephritis.

- **Diagnosis** (See '[Diagnosis](#)' above.)

- The diagnosis of renal and/or urologic TB should be suspected in patients with relevant clinical manifestations (urinary frequency, dysuria, hematuria, acidic urine, and/or sterile pyuria) and relevant epidemiologic factors (history of prior TB infection or disease, known or possible TB exposure, and/or past or present residence in or travel to an area where TB is endemic). The diagnosis of urogenital TB may be established by demonstration of tubercle bacilli in the urine. Three to six early-morning urine samples should be sent for urine mycobacterial culture or urine polymerase chain reaction (PCR) for *M. tuberculosis*.
- Radiographic imaging is also warranted for patients with suspected renal or urologic TB. Computerized tomography with contrast is preferred when feasible; other radiographic tools include high-resolution ultrasound and intravenous pyelography. In general, radiographic evidence of concomitant upper and lower urinary tract involvement is strongly suggestive of TB. Strictures throughout the collecting system (from the renal pelvis to the ureterovesical junction) are observed in 60 to 84 percent of cases ([figure 1](#)). Other findings are discussed above.

- **Genital TB**

- **Presentation**

- **Male genital tract** - In the male genital tract, TB infection may involve the prostate, seminal vesicles, vas deferens, epididymis, testicles, Cooper glands, and penis. Epididymitis is the most common clinical manifestation among men with genital TB; the epididymis is affected in 10 to 55 percent of cases. Physical findings consist of scrotal nodule or epididymal hardening in up to half of cases. Involvement is bilateral in 34 percent of cases. (See '[Presentation](#)' above.)
- **Female genital tract** - In the female genital tract, TB infection may involve the fallopian tubes, endometrium, and ovaries but generally spares the myometrium. The fallopian tubes are affected in 90 to 100 percent of cases, usually with bilateral involvement. Female genital TB presents clinically as infertility (40 to 76 percent), pelvic or abdominal pain or mass (50 percent), and menstrual disorders (25 percent). (See '[Presentation](#)' above.)

- **Diagnosis** – The diagnosis of genital TB should be suspected in patients with relevant clinical manifestations (in men: nodular lesions of the scrotum, prostate and/or testis and/or nonhealing ulcers of the external genitalia; in women: infertility, pelvic or abdominal pain, and/or menstrual disorders) as well as relevant epidemiologic factors (history of prior TB infection or disease, known or possible TB exposure, and/or past or present residence in or travel to an area where TB is endemic). (See '[Male genital tract tuberculosis](#)' above and '[Female genital tract tuberculosis](#)' above.)

- **Male genital tract** - The diagnostic approach to male genital TB begins with evaluation of the urinary tract as described above. In the absence of evidence for urinary involvement, men with suspected genital TB should undergo biopsy of the involved site; biopsy specimens should be sent for histopathological examination, stain for acid-fast bacilli, and mycobacterial culture. (See '[Diagnosis](#)' above.)
- **Female genital tract** - In women with suspected genital TB, hysterosalpingogram may demonstrate fallopian tube obstruction or constriction and/or uterine cavity adhesion or deformity. Histopathologic diagnosis may be made via endometrial or fallopian tube biopsy for histology and culture, or via mycobacterial culture of menstrual fluid. Specimens should be sent for histopathological examination, stain for acid-fast bacilli, and mycobacterial culture. (See '[Diagnosis](#)' above.)

- **Management** – Patients with urogenital TB should be treated with antituberculous therapy; in general, the approach is the same as that for pulmonary TB. In patients with ureteral stricture and hydronephrosis, early stenting or percutaneous nephrostomy is beneficial in the setting of obstructive nephropathy that is potentially reversible. Indications for surgical intervention and guidelines for follow up are discussed above. (See '[Management](#)' above.)

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