REVIEW ARTICLE

https://doi.org/10.3329/jmcwh.v21i2.81412

UROGENITAL TUBERCULOSIS

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ABSTRACT

Mycobacterial seeding of the urogenital tract by hematogenous dissemination causes tuberculosis affecting the kidney and urologic system; this can happen after a lung infection, reactivation, or miliary disease. Kidney parenchymal lesions, such as glomerulonephritis and interstitial nephritis, are less frequent. Tuberculous bacteria have the ability to penetrate the medullary interstitium and result in development of granuloma. In the absence of obvious kidney disease, they may heal with accompanying fibrosis. Alternatively, years after the initial infection, they may burst and break down into the tubular lumen, releasing tuberculous bacilli into the urinary system and causing the infection to spread continuously. When an infection descends to the bladder and ureter, it results in hydronephrosis, ureteral stricture and blockage, and impaired kidney function.When individuals exhibit pertinent clinical symptoms and pertinent epidemiologic characteristics, urogenital tuberculosis should be suspected.Urinary frequency, hematuria, acidic urine, sterile pyuria, and/or dysuria are important clinical indicators for renal or urologic tuberculosis. Infertility, pelvic or abdominal discomfort, and/or menstrual abnormalities in women; nodular lesions of the scrotum, prostate, and/or testis in males; and nonhealing ulcers of the external genitalia in women are all signs of genital Tuberculosis (TB). Relatively seldom are systemic signs like fever and weight loss. The presence of tubercle bacilli in the urine can confirm the diagnosis of urogenital TB. Additionally, radiographic imaging is necessary for individuals who may have urologic or renal tuberculosis. When possible, contrast-enhanced computerized tomography is the preferable method; intravenous pyelography and high-resolution ultrasonography are other radiography methods.Anti-tubercular treatment is recommended for patients with urogenital TB; the main strategy is the same as for pulmonary TB. Surgical interventions are justified to a certain extent.

Keywords: Tuberculosis, Renal, Genital, Urogenital.

Cite this article: Kader MA, Ansary EAF, Arif NUM. Urogenital Tuberculosis. J Med Coll Women Hosp.2025; 21(2): 166-174.

INTRODUCTION

Mycobacterium tuberculosis is the pathogen that causes tuberculosis. It is still a significant public health issue in developing nations. With the rise in organ transplants and the onset of human immunodeficiency virus infection, extrapulmonary TB became more prevalent and caused immunosuppression in thousands of people[1]. Twenty seven percent of extrapulmonary cases are that of urogenital TB. Both localized genitourinary diseases and widespread infections can impact the kidneys. The majority of healthcare facilities underdiagnose renal involvement by TB infection. Microscopic hematuria and sterile pyuria are common in patients with renal tuberculosis. The presence of pyuria without a common bacterial infection serves as the basis for the diagnosis of urinary tract TB[1].

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After tuberculous pleural effusion and lymph node involvement, urogenital TB is the third most prevalent form of extrapulmonary TB[2]. Two to twenty percent of patients with pulmonary TB develop urogenital TB[3,4]. Between 25 and 62 percent of patients with miliary disease experience hematogenous seeding of the urogenital tract[2]. Urogenital TB affected two males for every female in a survey of over 9000 TB cases, with a mean age of 40 years (range: 5 to 90 years)[5]. Ten percent of patients may have an ongoing infection, and up to fifty percent have radiographic evidence of a previous infection[6]. Urogenital TB is frequently misdiagnosed, and early detection requires a high index of suspicion.Urogenital tuberculosis frequently progresses quickly to a non-functioning kidney due to its subtle onset and non-specific constitutional symptoms. There is a chance that the contralateral kidney may also get involved because of the hematogenous spread of TB[7].

Pathogenesis

Two distinct forms of TB affect the kidney and urologic system. The urine collecting system (containing the renal pelvis, calyces, ureters, and bladder) is the site of the most frequent presentations. Kidney lesions, which are less frequent, can include calcification, ulceration, abscess formation, perinephric spread, and caseating necrosis. Additionally, diffuse parenchymal abnormalities like glomerulonephritis and granulomatous interstitial nephritis have been reported[8].

Both a localized genitourinary disease and a widespread infection may involve the kidney in tuberculosis[9,10]. Most of the time, the main emphasis is a pulmonary infection. Following exposure, the bacilli are engulfed by the macrophages where they gradually proliferate. The initial infection usually resolves on its own. The kidneys are frequently impacted by miliary TB, a condition in which hematogenic spread, especially in the cortical region, results in miliary lesions in renal tissue[10]. Some patients with disseminated or pulmonary tuberculosis exhibit renal failure without the usual localized renal parenchymal abnormalities. Hematogenic spreading happens when a conduit, usually a pulmonary vein, erodes and releases microorganism-containing emboli into the bloodstream. Kidney, epididymis, uterine tube, bone marrow, and encephalus are among the organs for which Mycobacterium Tuberculosis have the predilection to grow in since it requires specific environmental conditions to do so[9,10].

The medullary area of the kidney is the preferred site for Mycobacterium tuberculosis colonization. Here, granulomatous lesions with caseous necrosis can develop, resulting in local tissue damage. The cortex is where the renal lesion starts, and when the person is resistant to this organism, it usually heals. The bacilli then produce cortical granulomas after migrating to the cortico-medullary junction. During reactivation, the organisms enter the renal medulla and induce papillitis, but these granulomas stay stable for several years. As the illness worsens, large patches of papillary necrosis may result in cavities that erode the renal parenchyma and spread into the collecting system. Papillary necrosis may result from vascular insufficiency brought on by the infection in the renal papillae. Pyonephrosis may develop from tuberculous pyelonephritis brought on by an infection spreading to the renal pelvis. Usually, the infection spreads from the ureter to the bladder, resulting in granulomatous lesions that are linked to fibrosis. These procedures take place gradually over a number of years. One of the most significant TB symptoms visible in the pyelogram is the series of dilations intercalated with strictures caused by the lesions in the ureter. A megaureter may develop when the obstruction is more distal and complete[11].

The formation of mass lesions that mimic a neoplastic lesion can result from renal lesions that spread outside of the renal capsule. Urinary blockage and reflux may result from segmental stenosis and dilatation brought on by ureteral involvement[10]. Pelvic stenosis and infundibular stenosis are signs of advanced illness. One or both kidneys may have a single or multiple renal calcification. Diffuse calcifications, loss of renal function, and organ damage are the ultimate outcomes[9].

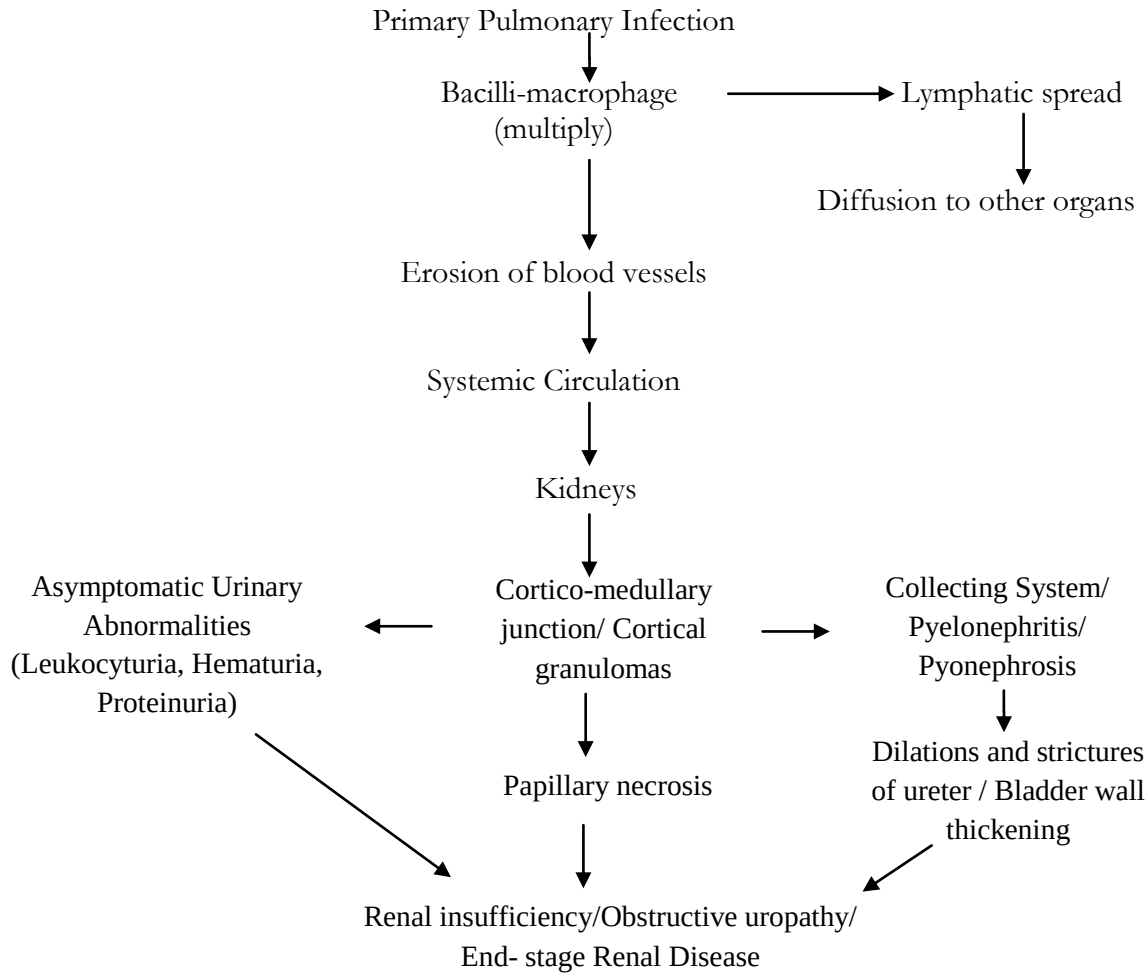

Figure 1: Pathophysiology of renal tuberculosis[1]

Clinical presentation

Pyuria and/or microscopic hematuria may be seen as incidental findings; at first, renal and urologic TB are not linked to any particular symptoms. About half of the patients have frequency, urgency, dysuria, and nocturia after the illness has spread to the urinary bladder; about 40% of cases experience low back discomfort and gross hematuria[2]. Tubular proteinuria, which frequently falls under the subnephrotic range, can be caused by chronic parenchymal involvement. Fever and weight loss are comparatively uncommon systemic symptoms[14].

A cortical granuloma is created by the inflammatory reaction and has the potential to heal and leave a tiny scar. A contiguous spread of infection can result from organisms that either remain latent for many years or burst into the proximal tubule of the nephron, excreting

tuberculous bacilli into the urinary system[12.13]. At the loop of Henle, where they proliferate, the bacilli in the nephron are trapped. The renal medulla's hypertonicity, high ammonia content, and relatively limited blood flow all work together to weaken immune responses and promote the development of medullary granulomas or chronic granulomatous tubulointerstitial nephritis. An abscess may then develop as a result of renal papilla loss. Diffuse or numerous ureteral strictures and blockage, hydronephrosis, perinephric abscess, and impaired kidney function are all consequences of infection spreading downward to the ureter and urinary bladder[14].

A nonfunctional calcified kidney known as a "putty kidney" or "cement kidney" can be the outcome of dystrophic calcification. Calcification is a sign of active disease rather than healing because organisms live in the calcified lesions. Compared to bilateral involvement, unilateral kidney involvement is typically more frequent[13]. End-stage renal disease, however, could arise from involvement of both kidneys.

The ureterovesical junction in the urinary bladder is distorted by hyperemia and ulceration; the ureteral aperture may expand and shift laterally (a "golf hole" ureter). Additionally, there may be urinary bladder fibrosis, contraction (commonly known as "thimble bladder"), and ulcers with granulomatous inflammation affecting all the layers (pancystitis). Fistulae development is an uncommon consequence of urinary bladder involvement[15]. Amyloidosis, glomerulo-nephritis, and interstitial nephritis are uncommon diseases linked to tuberculosis[15].

The pathophysiology of interstitial nephritis linked to tuberculosis is unclear. Instead of being the direct consequence of a kidney TB infection, interstitial nephritis may, at least in certain situations, be an immunologic phenomena brought on by TB involvement of other organs[16]. A paradoxical response (i.e., the development of new lesions or the worsening of existing lesions after therapy initiation) may be the cause of interstitial nephritis that develops after anti-tubercular therapy is started. This could happen because improved immunologic function raises the inflammatory response to infection[17,18]. Drugs used to treat tuberculosis, such rifampicin, can also cause acute allergic interstitial nephritis.

TB-related glomerulonephritis has been reported in a number of case studies[20]. It seems that tubercular infection of the kidney is directly linked to glomerulonephritis. Twenty percent of the 46 Chinese patients with tubercular glomerulonephritis in a retrospective investigation had a positive urine mycobacterial culture, and 85 percent of the kidney biopsy results were Polymerase Chain Reaction (PCR) positive for tuberculous glomerulonephritis[19].

Amyloidosis

Renal amyloidosis can also be brought on by tuberculosis[21]. Amyloid deposition in the kidney causes amyloidosis, a glomerular lesion. The whole male genital system, including the prostate, seminal vesicles, vas deferens, epididymis, testicles, Cowper's glands, and penis, is impacted by genital tuberculosis. Hematogenous spread to the prostate and epididymis or urinary tract spread to the prostate from the ejaculatory ducts to the seminal vesicles, vas deferens, and epididymis are the two ways that genital tuberculosis can arise[2].

Hematogenous spread from the lungs is the most prevalent way that tuberculosis of the female genital tract spreads; lymphatic dissemination from other abdominal organs is less common[22]. Tuberculosis often spares the myometrium but damages the ovaries, endometrium, and fallopian tubes[3].

Sexual contact with a man who has tuberculosis of the penis or epididymis can

result in the development of primary tuberculosis of the female genital tract[2]. There are no documented cases of tuberculosis being sexually transmitted from female to male in the literature.

Diagnosis

Patients with relevant epidemiologic factors (history of prior tuberculosis infection or disease, known or suspected tuberculosis exposure, and/or past or present residence in or travel to an area where tuberculosis is endemic) and relevant clinical manifestations (urinary frequency, dysuria, hematuria, and/or sterile pyuria) should be suspected of having renal and/or urologic tuberculosis[23].

Urine tests, radiographic imaging, the tuberculin skin test or interferon-gamma release assay, and histopathology (in certain cases) are diagnostic methods for urogenital tuberculosis. The presence of tubercle bacilli in the urine can confirm the diagnosis of urogenital tuberculosis. If possible, three to six mornings urine samples should be tested for Mycobacterium tuberculosis by PCR, acid-fast staining, and mycobacterial culture. The automated broth culture should be positive in two to three weeks, but urine mycobacterial culture has a sensitivity of up to 90% and a specificity of 100%. Results may take six to eight weeks[24]. Cultures of urine acid-fast bacilli have high specificity and sensitivity of 80%–90%[25].

Urine PCR for Mycobacteria has a sensitivity of 87%–100% and a specificity of 93%–98%. There is little information available on the application of GeneXpert MTB/RIF molecular system (used to detect Mycobacterium Tuberculosis and resistance to Rifampicin) in extrapulmonary tuberculosis diagnosis. Sensitivity and specificity of 100 percent and 98 percent, respectively, were reported in one analysis of 91 urine samples from patients with suspected tuberculosis or nontuberculous mycobacterial infections, including five culture-positive samples[26].

Additionally, radiographic imaging is necessary for individuals who may have urologic or renal tuberculosis. Depending on availability and center desire, radiographic methods such as high-resolution ultrasonography, magnetic resonance imaging (MRI), and computed tomography (CT) with contrast may be utilized[27]. When possible, CT is preferred because it gives more anatomic information.

Fine-needle aspiration of the pathologic tissue, kidney biopsy, or urinary bladder biopsy is necessary for mycobacterial culture and histopathology investigation in patients with suspected tuberculosis, suggestive radiographic symptoms, and negative urine testing (mycobacterial culture or PCR)[28]. The primary clinical characteristics and radiographic results determine the biopsy location. Biopsy specimens have to be submitted for mycobacterial culture, staining for acid-fast bacilli, and histological analysis. Interferon-gamma release assay (IGRA) or tuberculin skin testing is necessary. The purpose of these tests is to diagnose tuberculosis infection. A negative result does not rule out active tuberculosis illness, and a positive result supports (but cannot be used to prove) a diagnosis of active tuberculosis disease[29].

The urinary tract is evaluated as the first step in the diagnostic process for male genital tuberculosis. Men with suspected genital tuberculosis should have a biopsy of the affected area if there is no indication that the urine is implicated. The biopsy specimens should then be sent for histopathological analysis, acid-fast bacilli staining, and mycobacterial culture. In case of female genital tuberculosis hysterosalpingogram may show adhesion or deformity in the uterine cavity, as well as restriction or constriction of the fallopian tube. A mycobacterial culture of

menstrual fluid or an endometrial or fallopian tube biopsy and culture can be used to make a histopathologic diagnosis[22]. Samples have to be submitted for mycobacterial culture, acid-fast bacilli staining, and histopathology analysis. A presumptive diagnosis of urogenital tuberculosis can be obtained based on suggestive clinical, laboratory, and radiographic evidence in the absence of microbiologic or histologic confirmation.

Management

The cornerstone of treatment consists of anti-tubercular medications. Furthermore, early stenting or percutaneous nephrostomy is helpful in the context of possibly reversible obstructive nephropathy in patients with ureteral stricture and hydronephrosis. Nephrectomy, ureteral stricture dilatation or repair, and urinary bladder diversion are surgical procedures used to treat urogenital tuberculosis.

Anti-tubercular therapy: Generally speaking, the treatment for urogenital tuberculosis follows the same strategy as that for pulmonary tuberculosis .When applied to susceptible species, anti-tubercular drugs produce high urine concentrations and a cure rate of more than 90%. After two weeks of proper anti-tubercular treatment, no bacilli are often seen in the urine. Urogenital tuberculosis can, however, recur after first urine sterilization. Impaired fertility is frequently not restored by anti-tubercular treatment[30].

Surgery: Nephrectomy, ureteral stricture dilatation or repair, and urinary bladder diversion are surgical procedures used to treat urogenital tuberculosis. A percutaneous nephrostomy or ureteric stenting may be warranted even with the right care, urinary tract lesions can worsen; inflammation can cause the collecting system to become obstructed, the urinary bladder to contract, the frequency of urination to increase, and kidney function impairment to occur[6,31]. For patients with ureteral stricture, early stenting or percutaneous nephrostomy is therefore appropriate, particularly if hydronephrosis is also present. Early stenting protects the kidney against blockage because areas with stricture may develop fibrosis and formation of scar when anti-tubercular medication is started. The nephrectomy rate was higher among patients who received anti-tubercular therapy alone (73 versus 34 percent) in one study that included 77 patients with tubercular ureteral stricture treated with anti-tubercular therapy with or without additional intervention (early ureteral stenting or percutaneous nephrostomy)[31].

Generally speaking, surgery should be postponed until at least four weeks of anti-tubercular treatment have been given when necessary[32].

Relapse and follow-up

Urogenital tuberculosis may recur following first urine sterilization. Recurrence rates among patients who need a nephrectomy seem to be quite low (<1 percent); recurrence occurs in up to 6 percent of cases after a mean of 5 years of therapy (range 11 months to 27 years)[6,33].

Thus, for individuals with renal tuberculosis who do not have a nephrectomy, observation is necessary[2]. For ten years after anti-tubercular treatment is finished, surveillance should be conducted, which should include visits every six to twelve months for urine mycobacterial culture and/or urine PCR testing for Mycobacterium tuberculosis, as well as ultrasonography[34]. Additionally, patients should be told to get evaluated if their symptoms worsen.

CONCLUSION

The prevalence of tuberculosis is high in poorer nations. Urogenitaltuberculosis is worrisomely underdiagnosed, which can result in renal insufficiency, chronic kidney disease, end-stage renal disease, sexual dysfunction and infertility. All of which can be avoided with appropriate and timely targeted treatment. New studies are required to identify the primary clinical manifestations of renal tuberculosis, as well as to provide less harmful anti-tubercular medications and more effective diagnostic techniques. Due to the evolution of human immune-deficiency virus and the rise in iatrogenic immunosuppression brought on by new medical developments, doctors need to be on the lookout for suspected urogenital tuberculosis.

CONFLICT OF INTEREST

There is no conflict of interest.

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