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Overview of Diagnosis and Treatment of Urinary Tract Tuberculosis

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Abstract: **This review was aiming to overview the urinary tract tuberculosis or as it's called in many studies urogenital TB (UGTB) from many different aspects, and we intended to more discuss the diagnostic and treatment approaches for this urological infectious disease. An electronic search was performed through Midline, and Embase databases for relevant subject to urinary tract tuberculosis that was published up to December 2016 in English language and contain only human subject, we used Mesh terms in searching the Pubmed database which are; "urogenital TB" Or "UGTB" Or "urinary tract tuberculosis" Or "extrapulmonary TB" combined with "diagnosis" AND "screening" Or "therapy" Or "management". We have included most of evidence based trails which were discussing the diagnosis and treatment of UGTB in every population stages. Tuberculosis has a high incidence in developing countries. he insidious beginning and non-specific constitutional symptoms of genitourinary tuberculosis (GUTB) often lead to postponed diagnosis and quick development to a non-functioning kidney. Due to hematogenous dissemination of TB, there is a potential risk of participation of the contralateral kidney too. Imaging plays a crucial function in the making of a prompt medical diagnosis and in the planning of treatment, and therefore helps to prevent complications such as kidney failure. Imaging of GUTB still remains a difficulty, primarily on account of the dearth of literature, especially related to the use of the newer modalities such as magnetic resonance imaging (MRI).**

Keywords: **urinary tract tuberculosis, extrapulmonary TB, diagnosis, screening, therapy, urogenital TB, UGTB.**

1. INTRODUCTION

The Mycobacterium tuberculosis (MTb) bacillus has, because ancient times, called Tuberculosis (TB), is the commonest around the world reason for death from contagious diseases caused by Mycobacterium bacillus [(1)] with 9 million brand-new cases and two million deaths per year [(2)]. Around 95% of cases take place in establishing countries [(1)].

In 1882, the microbiologist Robert Koch unmasked the nameless opponent and discovered the bacillus that causes tuberculosis (TB). The urogenital system is a typical part of extrapulmonary TB in adults, however the true incidence of urogenital TB (UGTB) is less clear (3,4). complications regarding the occurrence of UGTB as a symptom of non-pulmonary TB vary from 4% to 73% [(5,6,7)]. Extrapulmonary TB is uncommon in children, but UGTB is usually common site of TB affectation [(8,9)]. Common sites of extrapulmonary TB in children include the lymph nodes, meninges, intestinal system, bones and joint areas [(10)]. patients with tuberculosis of the urinary tract is constantly in danger of an abrupt fatal spread of the disease. If care is exercised he may go on and never be affected by his status, but without care, it may bring about his fast and total destruction. Urinary tract tuberculosis is typically secondary to a focus elsewhere in the body [(11)]. Comprehensive evaluations of TB impacting the female genital tract were just recently released [(3,4)], showed that the most common gynaecological presentation of TB is infertility, but women with TB peritonitis might present with abdominal pain and distension due to ascites, looking like the presentation of sophisticated ovarian carcinoma [(3)].

Renal disease might be the result of direct infection of the kidney and lower urinary system or might present as secondary amyloidosis. Patients present with dysuria, flank, or hematuria pain [(12,13)]. More than 90 percent of asymptomatic patients have sterilized pyuria with or without tiny hematuria [(12,13)]. Intravenous pyelography might reveal a "motheaten calyx" or

papillary necrosis. abdominal CT scan may reveal kidney calcifications, calculi, scarring, hydronephrosis, or evidence of extrarenal disease (e.g., ureteral strictures; contracted bladder; calcifications in the vas deferens, critical blisters, or prostate). Mycobacterial culture of 3 early morning urine specimens develops the medical diagnosis in 90 percent of patients [(12)]. Male genital tuberculosis generally is related to kidney tuberculosis. It involves the prostate, seminal vesicles, epididymis, and testes, in order of incidence. Patients typically provide with a scrotal mass (**Figure 1**) [(13)], and medical diagnosis is made by surgery. Oligospermia is common and may be persistent. Female genital tuberculosis begins in the endosalpinx and can infect the peritoneum, endometrium, ovaries, cervix, and vaginal area [(13)]. Patients present with pelvic pain, infertility, and vaginal bleeding [(13)].

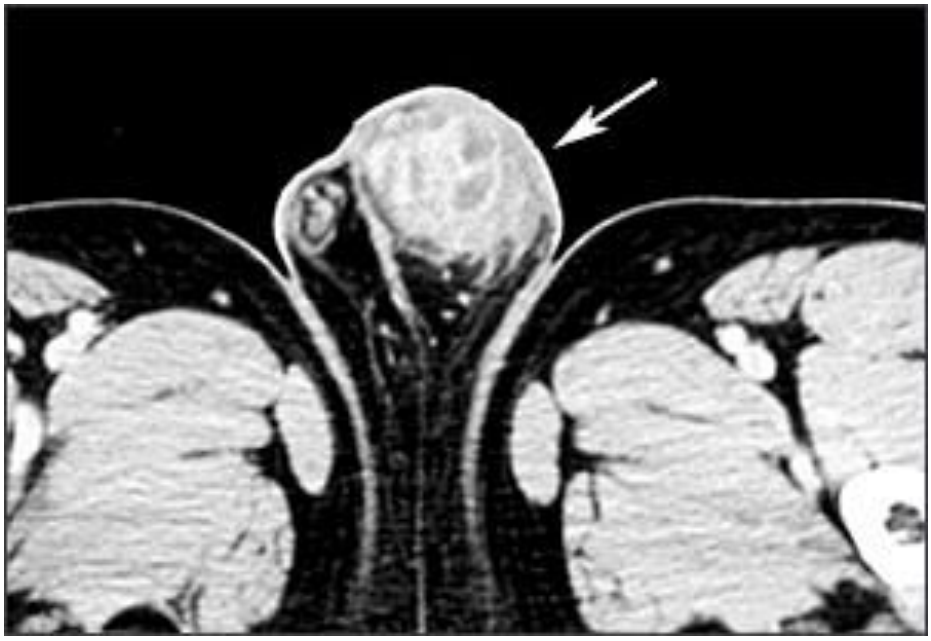

Figure 1: Testicular tuberculosis. Computed tomographic scan of the pelvis showing a large, irregular, mixed solid and cystic left testicular mass *(arrow)*. [(13)]

This review was aiming to overview the urinary tract tuberculosis or as it's called in many studies urogenital TB (UGTB) from many different aspects, and we intended to more discuss the diagnostic and treatment approaches for this urological infectious disease.

2. METHODOLOGY

An electronic search was performed through Midline, and Embase databases for relevant subject to urinary tract tuberculosis that was published up to December 2016 in English language and contain only human subject, we used Mesh terms in searching the Pubmed database which are; "urogenital TB" Or "UGTB" Or "urinary tract tuberculosis" Or "extrapulmonary TB" combined with "diagnosis" AND "screening" Or "therapy" Or "management". We have included most of evidence based trails which were discussing the diagnosis and treatment of UGTB in every population stages.

3. RESULTS

- **Overview of mortality and pathogenesis of urinary TB:**

he genitourinary system is a primary target of hematogenous infections [(14)] and is the most typical site of extra-pulmonary TB, [(15)] comprising 14-41% of the exact same [(16,17)]. Genitourinary tuberculosis (GUTB), or urinary system TB (UTB) a term created by Wildbolz in 1937, [(18)] is an around the world disease, however reveals a more destructive habits in developing nations. The kidney is the most common site of GUTB. An increased incidence of extra-pulmonary TB has been kept in mind in gotten immunodeficiency syndrome (AIDS) [(19)]. Worldwide, 15% of TB patients are co-infected with HIV, and in HIV-endemic areas, as lots of as 75% of patients with GUTB are co-infected with HIV [(20,21)]. For this reason, HIV-positive patients need to be evaluated for TB, and patients with freshly identified TB should be evaluated for HIV infection [(22)]. With the enhancing survival rates of AIDS patients, we can anticipate an increase in the incidence of urinary system TB [(23)].

Spread of tuberculosis to the urinary tract:

Hematogenous dissemination of MTB takes place from a primary TB focus within the lungs, bone, or other organs and can involve both kidneys [24]. Bacille Calmette-Guerin (BCG) a live, vaccine strain-- can cause renal sores by means of reflux, in 0.1% of patients undergoing intravesical instillation of BCG for the treatment of bladder cancer [25,26].

The renal lesions can disseminate beyond the kidney capsule and cause advancement of mass sores, mimicking a neoplastic sore. The ureteral involvement can trigger segmental stenosis and dilations, causing urinary blockage and urine reflux [27]. Advanced disease can cause pelvic and infundibular stenosis. The participation of renal calices can be several or unique in one or both kidneys. The end results are organ destruction, kidney function loss, and scattered calcifications (28). The pathophysiology of kidney TB is shown in (**Figure 2**) [28].

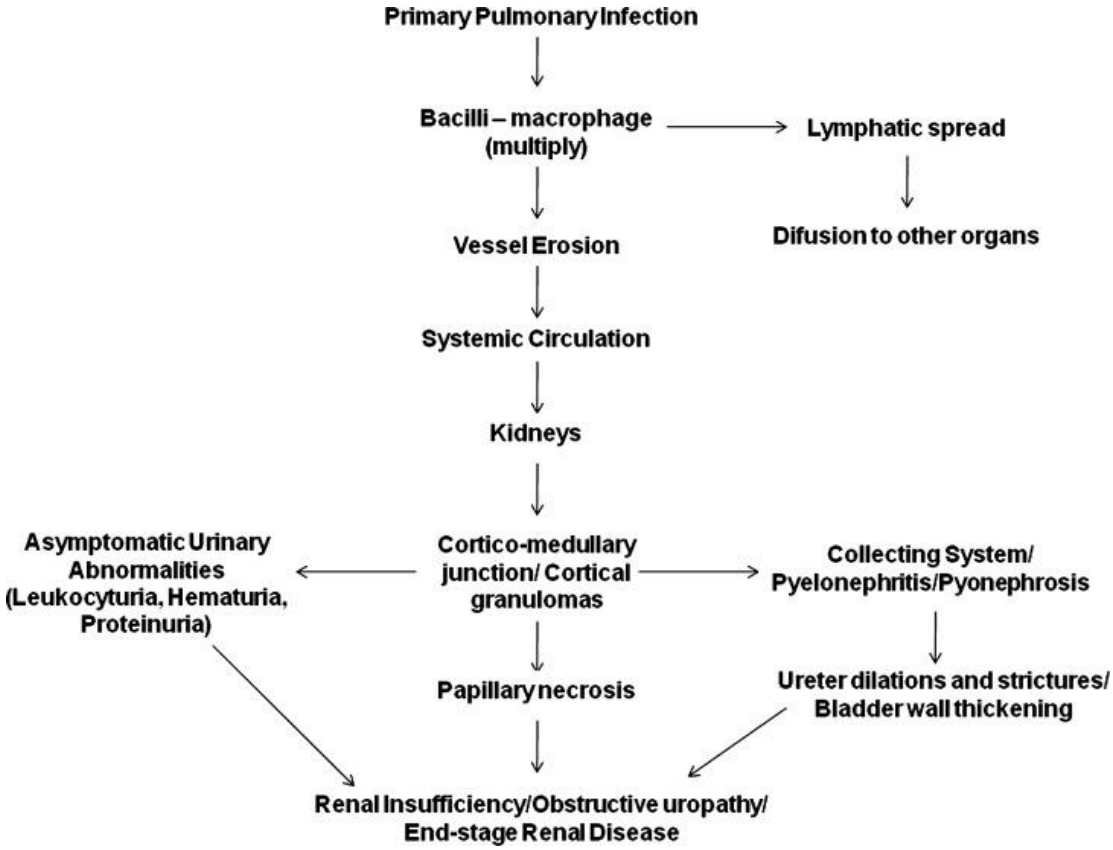

Figure 2: Pathophysiology of renal tuberculosis. [28]

The pathogenesis of kidney TB can be divided into 2 forms: kidney participation during distributed infection and localized genitourinary disease. In both cases, the sores depend essentially on the immunologic status of the person, pathogen virulence, and the site of infection. In milliary TB (distributed kind), a lot of tubercles lie in the renal cortex, and can be as big as 3 mm in diameter [27]. Histologically, milliary TB is identified by epithelioid granuloma, frequently with giant cells. In patients with this form, kidney function is abnormal. Uremia is more related to interstitial nephritis [27]. In immunocompromised patients, granulomas are poor structured, with less caseous necrosis. In more extreme cases, the most common pattern is histiocytes consisting of several bacilli intracytoplasmatic. When kidney TB occurs, it is necessary to think immunologic aggressiveness, and corticosteroids must be consisted of in the treatment [27].

- **Diagnosis of urinary tract TB:**

Unique examinations Urinalysis is the least invasive approach of detecting UGTB. The classically explained "sterile pyuria" is neither specific nor sensitive enough for UGTB, however persisting sterilized pyuria in a private at risk (endemic location, immunocompromised patient) should increase the clinician's index of suspicion [29,30]. Urine Ziehl-Neelsen (ZN) staining for acid-fast bacilli (AFB) is frequently incorrect unfavorable [31]. Urine culture for TB takes 6-- 8 weeks for a conclusive result, and since the MTb organisms are excreted intermittently, a minimum of three (ideally five) consecutive morning urine samples (EMUs) must be sent for culture [30].

- **Polymerase chain reaction (PCR) as diagnostic method of UGTB**:

Nucleic acid amplification can be used to discover small numbers of MTb organisms in body fluids. Although sputum PCR has been studied extensively, less research studies have specifically evaluated urine PCR for the medical diagnosis of UGTB. PCR seems more delicate than urine culture (level of sensitivity is 37% to 79% for TB culture and 75% to 94% for PCR) [32]. Whereas a culture takes about six weeks for a final result, PCR supplies confirmation within [33] hours [34]. However, PCR is far more costly than conventional urine culture, and it needs rigid lab strategy to reduce incorrect unfavorable and false favorable outcomes. It is reasonable to consider carrying out PCR on the urine when the clinician strongly presumes UGTB despite unfavorable microbiological and histopathological investigations. It should not be the sole method for the diagnosis of UGTB [35].

- **Role of Magnetic Resonance Imaging (MRI) in Urinary Tract TB diagnosis:**

MRI provides good morphological details of the kidneys along with excellent delineation of the ureters [36]. It allows characterization of various kidney masses and can provide important information contributing to their medical management [37]. When ionizing radiation and iodinated contrast cannot be administered, it is especially beneficial in pregnant or pediatric patients or. Non-contrast MRI is specifically useful in patients with renal failure [36]. When CT and/or ultrasound are equivocal [38], MRI is practical for additional differentiation of lesions. MR urography (MRU) comprises a developing group of methods with the potential for ideal non-invasive examination of urinary tract irregularities [38]. Both static-fluid (non-contrast, greatly T2W sequences) and excretory MRU (carried out during the excretory phase of improvement after intravenous gadolinium) can be combined with traditional MRI for comprehensive assessment of the urinary tract. Cine MRU demonstrates the ureters in their whole and is useful for validating the existence of stenosis [38]. It is most effective in patients with reasonably to seriously dilated obstructed gathering systems and in impaired renal function situations MRU performed with a distended urinary bladder permits much better visualization of the upper urinary tract. Time-resolved dynamic contrast-enhanced MRU has been utilized in the examination of ureteral peristalsis in GUTB [38]. In view of the possibility of nephrogenic systemic fibrosis/nephrogenic fibrosing dermopathy, [39] caution should be worked out while administering gadolinium in patients with compromised kidney function.

- **Most devastating complications of urinary TB:**

Parenchymal renal system changes:

When bacilli are shed into the urine, the disease spreads out antegradely to include the urothelium of the kidney hips, ureter, bladder and, at times, the nearby genital tract [40]. Infection in the walls of the calyces, hips, and ureter produces considerable inflammatory mucosal thickening, a typically ignored imaging finding. Multiple or single calyces may be involved in one or both kidneys. Microscopic granulomas might form here too. Ulceration quickly follows. In advanced disease, in addition to loss of parenchyma by caseation, intra-renal scars and strictures result in obstruction and dilatation of sections of the PCS. Strictures are more common at sites of typical constricting, such as the calyceal neck, the pelvi-ureteric junction, and the ureterovesical junction. Early scarring is obviously reversible by suitable steroid treatment; however, end-stage fibrotic strictures are permanent. Urinary obstruction from strictures along with renal parenchymal caseation destroys all or part of the kidney. The pattern of destruction depends upon the relative rates of development of parenchymal disease and urinary-flow obstruction. Parenchymal caseation, necrosis, and calcification may predominate, which causes the kidney to be damaged (**Figure 3**) [41]. Blockage might predominate, in which case massive hydronephrosis or hydrocalicosis may be the last phase. TB of the kidney thus shows completing procedures: (a) The destructive effects of the bacilli, causing fistulization, ulceration, and cavitation and (b) the host's secondary defense and healing system resulting in the development of granulomas together with fibrosis, calcium deposition, and strictures, which may worsen the obstruction causing progressive renal dysfunction [41]. The final result is thus very variable [42]. The majority expose evidence of peripheral enhancement [40]. The nodules are variable-sized, well-defined parenchymal sores on cross-sectional images and might mimic kidney neoplasms, which may cause unneeded surgical treatment; these are for that reason labeled as the 'pseudo-tumoral' type [40,41,42]. The lesions can occasionally grow to a very large size [43]. Bigger granulomas might form masses of combined density due to the presence of areas of calcification.

Figure 3: Pathology specimen of end-stage renal tuberculosis: The basis for the 'lobar caseation pattern' is evident post-content [41]

- **Treatment approach of UTB:**

All patients with active or latent TB requires specific treatment. The first-choice drugs consist of isoniazide, rifampicin, pirazinamide, streptomycin, and ethambutol. After diagnosis, chemotherapy with 3 or four drugs ought to be right away started and last for a minimum of six months [43,44]. Two regimens can be used. The first-line regimen, which is utilized for 6 months, is ethambutol, rifampicin, and isoniazide or pirazinamide daily for two or three 3 months; and isoniazide and rifampicin twice a week for three of four months [44]. The second-line regimen, which is utilized for nine months, is isoniazide, ethambutol, pirazinamide, or rifampicin daily for two or 3 months, followed by isoniazide and rifampicin, twice a week, for six to seven months [45]. Rifambutin can substituted for rifampicin to reduce drug interactions with drugs used in the treatment of HIV infection (protease inhibitors and transcriptase reverse non-nucleoside inhibitors) [45].

According to the brand-new guidelines by the Brazilian Ministry of Health, it is suggested that TB patients be treated with four drugs: rifampicin, isoniazide, pirazinamide and ethambutol for 6 months [46] (**Table 1**). The second-line routine is less efficient than the first-line program cited above and is recommended for patients who cannot tolerate the first-line regimen. Second-line anti-TB drugs include capreomycin, ciprofloxacin, clofazimine, cycloserine, ethionamide, kanamycin, levofloxacin, ofloxacin, and aminosalicylic acid [44].

There was a boost in the resistance to tuberculostatic drugs in HIV patients. Isolated resistance to isoniazide ought to be treated with rifampicin, pirazinamide, and ethambutol for 6 months. If rifampicin resistance is presumed, treatment with isoniazide and ethambutol for 18 months or isoniazide, pirazinamide, and streptomycin for 9 months should be used [45].

Patients with loss of renal function must get the typical doses of rifampicin, isoniazide, pirazinamide, and ethionamide due to the fact that these drugs have billiary excretion properties and are metabolized into compounds that are not excreted by the kidneys [47]. However, care ought to be utilized when administering streptomycin, other aminoglycosides, and ethambutol due to the fact that these drugs have kidney excretion residential or commercial properties [47]. Ethambutol can cause optic neuritis, which is reversible, if the dose is lowered according to the GFR: 25 mg, three times a week, if the GFR is 50-- 100 mL/minute and twice a week if the GFR is 30 - 50 mL/minute. Streptomycin and other aminoglycosides can cause ototoxicity and nephrotoxicity and should not be used in patients with reduced kidney function [47].

Table 1: Treatment for adults and adolescents with tuberculosis, adapted from ref# [46]

Drugs, body weight (kg)	Units/dose	Duration of treatment, months
Rifampicin (150 mg), isoniazide (75 mg), pirazinamide (400 mg), ethambutol (275 mg)†		2
20–35	2 pills	
36–50	3 pills	
> 50	4 pills	
Rifampicin/isoniazide 300 mg/200 mg or 150 mg/100 mg		4
20–35	1 pill (300/200 mg)	
36–50	1 pill (300/200 mg plus 150/100 mg)	
> 50	2 pills (300/200 mg)	

†A pill is available that contains all four amounts of these drugs.

4. CONCLUSION

Tuberculosis has a high incidence in developing countries. he insidious beginning and non-specific constitutional symptoms of genitourinary tuberculosis (GUTB) often lead to postponed diagnosis and quick development to a non-functioning kidney. Due to hematogenous dissemination of TB, there is a potential risk of participation of the contralateral kidney too. Imaging plays a crucial function in the making of a prompt medical diagnosis and in the planning of treatment, and therefore helps to prevent complications such as kidney failure. Imaging of GUTB still remains a difficulty, primarily on account of the dearth of literature, especially related to the use of the newer modalities such as magnetic resonance imaging (MRI). There is an uneasy underdiagnoses of UTB, which results in advancement of renal deficiency, persistent kidney disease, and, all preventable circumstances with correct and early specific treatment.

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