Renal complications of tuberculosis; a mini-review

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Abstract
Tuberculosis (TB) is a chronic infectious disease caused by Mycobacterium tuberculosis, which primarily affects the lungs but can also involve other organs, including the kidneys. TB has the potential to result in the development of chronic renal failure and requires prompt diagnosis and treatment for effective management. Renal TB can lead to various complications, including renal failure, perinephritis, perinephric abscesses, fistulae, psoas abscesses, and non-functioning kidneys. Additionally, TB-associated nephropathy is an infrequent variant of glomerulonephritis characterized by the deposition of immune complexes containing TB antigens in the kidney’s glomeruli.

Introduction
Tuberculosis (TB) is a chronic infectious disease caused by Mycobacterium tuberculosis, primarily affecting the respiratory system. However, it can also affect several organs, such as the renal system (1). Kidney TB is a rare extra-pulmonary disease, accounting for less than five percent of all TB cases (2,3). Kidney TB can lead to significant morbidity and mortality if not diagnosed and treated promptly. The renal complications of TB can be classified into two categories: tuberculous nephritis and obstructive uropathy (4). Tuberculous nephritis is characterized by granulomatous inflammation of the renal parenchyma, which can lead to renal failure. Obstructive uropathy arises from the formation of ureteral strictures or renal calculi due to the scarring and fibrosis caused by the infection. This condition has been reported worldwide, but its prevalence varies depending on geographic location, socioeconomic status, and healthcare accessibility factors (5,6). This mini-review sought to provide a concise overview of the current knowledge on TB-associated nephropathy.

Search strategy
For this review, we conducted a comprehensive search across many academic databases, including PubMed, Web of Science, EBSCO, Scopus, Google Scholar, Directory of Open Access Journals (DOAJ), and Embase, using a range of relevant keywords, including renal tuberculosis, long-term complications, tuberculosis-associated glomerulopathy, focal segmental glomerulosclerosis, minimal change disease, and Embase, using a range of relevant keywords, including renal tuberculosis, long-term complications, tuberculosis-associated glomerulopathy, focal segmental glomerulosclerosis, minimal change disease.

Renal tuberculosis in brief
Renal TB can cause various complications, including fistulae, psoas abscesses, renal failure, perinephritis, perinephric abscesses, and non-functioning kidneys (7,8). According to a study, the prevalence of renal dysfunction is reported to be 24% (4). Several investigations have described several mechanisms of kidney failure following kidney infection, including: first, direct kidney tissue infection which initiates oblitative endarteritis, along with widespread dystrophic calcification or secondary kidney amyloidosis, both of which result in kidney injury. Secondly, post-obstructive atrophy due to numerous strictures and, finally, subtle but progressive...
tubulo-interstitial nephritis are also responsible for renal failure in this disease (9). A short look at the published cases of kidney TB, shows this condition may be asymptomatic or display the disease with damaging the renal parenchyma. In addition, site-dependent nonspecific urinary complications include pelvic or abdominal pain, abdominal mass, urinary frequency, hematuria, and sterile pyuria. Moreover, an unresponsive urinary tract infection to antibiotics is highly suggestive of urinary TB (10,11).

Risk factors for kidney tuberculosis
The risk factors associated with renal TB comprise previous TB infection, immunosuppression (HIV infection, corticosteroids or immunomodulatory drugs, transplant individuals), malnutrition, diabetes, chronic renal and liver disease, alcohol and substance abuse, smoking, homelessness, poor housing, pneumonia, genetics, vitamin deficiency, immunosuppressive drugs, renal transplantation, chronic renal disease, and dialysis or end-stage renal failure. Moreover, a previous study demonstrated that a high creatinine level at the diagnosis reflects the progression of urinary TB and the destruction of the kidney structure (12,13).

Long-term complications of renal tuberculosis
Renal TB can lead to various long-term complications and sequelae. This disease can result in the formation of abscesses in the kidney, which can initiate further damage and complications. Additionally, this condition can result in the formation of strictures or fistulas in the urinary tract, leading to obstruction and urinary complications. Renal TB also can increase the risk of developing kidney stones or calculi, which can cause pain and urinary problems. Likewise, hydronephrosis can occur due to this disease (14,15). Meanwhile, untreated or poorly managed renal TB can lead to chronic renal failure, defined by gradually lessening renal function over time. Finally, in severe cases, renal TB can destroy the renal parenchyma, leading to a non-functioning kidney (16).

Tuberculosis-associated glomerulonephritis
Tuberculosis-associated glomerulopathy is a rare form of glomerulonephritis caused by M. tuberculosis infection. This condition can present as rapidly progressive glomerulonephritis or crescentic glomerulonephritis, which are life-threatening diseases. Crescentic glomerulonephritis is associated with poor outcomes if treatment is inadequate and delayed (17,18). In addition, TB-associated IgA nephropathy is reported as a rare form of glomerulonephritis following Mycobacterium tuberculosis infection. There is growing evidence that TB can be associated with IgA nephropathy. This disease is a common form of glomerulonephritis characterized by the accumulation of IgA in the glomerular mesangium. It is a leading cause of chronic kidney disease worldwide (19). A case report described a 59-year-old man with disseminated TB and IgA nephropathy, presenting with microscopic hematuria, red cell casts, and normal renal function (20). Notably, previous case reports of TB-associated IgA nephropathy are from different regions, including Europe and Africa (19). One of the most common glomerular diseases associated with TB is also membranous nephropathy. This glomerulopathy is characterized by thickening of the glomerular basement membrane and deposition of immune complexes, which can lead to proteinuria, hypoalbuminemia, and edema (21,22). Focal segmental glomerulosclerosis (FSGS) is an additional glomerular disease that has been linked to TB. This glomerulopathy is characterized by scarring of the glomeruli, leading to proteinuria and eventual kidney failure. It is thought that TB-induced inflammation and cytokine release may contribute to the development of FSGS (23,24).

Similarly, minimal change disease (MCD) has also been reported in association with TB. MCD is characterized by diffuse loss of podocyte foot processes, leading to proteinuria. Previous studies showed that TB-induced cytokine release may contribute to the development of MCD (25). Finally, acute interstitial nephritis has also been reported in association with TB. This condition is characterized by inflammation of the interstitial tissue of the kidney, leading to acute kidney injury. It is thought that TB-induced inflammation and immune response may contribute to the development of acute interstitial nephritis (26).

Kidney pathology of nephropathy-associated TB
Renal TB can cause various morphologic and pathologic lesions inside the kidneys. These lesions include granulomatous inflammation, caseating necrosis, tubular atrophy, interstitial fibrosis, and calcification (27). Granulomas are a hallmark of TB and are located in the renal parenchyma, renal pelvis, and ureters. The presence of granulomas can lead to the formation of strictures and obstruction of the urinary tract. Caseating necrosis can also occur in the renal parenchyma and lead to renal failure. Tubular atrophy and interstitial fibrosis are caused by chronic inflammation and may result in irreversible damage to the kidneys (28,29). Calcification will occur in the renal parenchyma, renal pelvis, and ureters, leading to the formation of stones and urinary tract obstruction. Overall, renal TB's morphologic and pathologic lesions are diverse and result in significant renal dysfunction if not diagnosed and treated promptly (30).

Clinical presentation of renal TB
The clinical presentation of renal TB can be nonspecific, including symptoms such as fever, fatigue, weight loss, and night sweats. Hematuria, flank pain, and dysuria may also be present. The diagnosis of renal TB requires a high index of suspicion and is based on a combination of clinical, radiological, and microbiological findings (31,32).
Treatment of renal TB
Treating renal TB involves a combination of anti-TB medications for at least six months. Surgical intervention may be necessary in obstructive uropathy or a lack of response to medical therapy (33).

Conclusion
Renal TB can be asymptomatic or present with nonspecific urinary manifestations such as pelvic or abdominal pain, abdominal mass, urinary frequency, sterile pyuria, and hematuria. A resistant urinary tract infection to common antibiotics highly suggests urinary TB. The risk factors associated with this disease include prior TB, previous liver or renal disease, and immunosuppression therapy. It has also been detected that a high serum creatinine at the time of TB detection indicates an advanced stage of urinary TB along with destruction of the kidney structure. Previous investigations also indicated that following treatment of this disease, recovery of a destroyed kidney could be problematic.

Authors’ contribution
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Conflicts of interest
The authors declare that they have no competing interests.

Ethical issues
Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors.

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References


