Diffuse Calcification of the Urinary System and Miliary Tuberculosis Due To Delayed Diagnosis of Genitourinary Tuberculosis: A Case Report

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Abstract
Genitourinary tuberculosis (GUTB) is difficult to diagnose in the earlier stage owing to the non-specific symptoms. Usually, renal tuberculous involvement is unilateral and the imaging finding is renal calcification, but associated calcifications of bilateral ureter and bladder are rare. We report a 66-year-old man who presented with diffuse calcification of the urinary system (including bilateral pelvicalyceal system, both ureters and bladder) and disseminated miliary tuberculosis due to GUTB. He had been misdiagnosed with urinary tract infection and urinary lithiasis for two years before the diagnosis of GUTB was confirmed by microbiological examination of the urine. This case highlights the importance of maintaining a high index of clinical suspicion for GUTB.

Keywords: Genitourinary tuberculosis, Calcification, Renal failure, Miliary tuberculosis

Introduction
Tuberculosis (TB) remains a major global problem for public health. It causes ill-health among millions of people each year and ranks as the second leading cause of death from an infectious disease worldwide. In 2011, there were an estimated 8.7 million new cases of TB and 1.4 million deaths from TB (1).
TB is an infectious disease caused by the bacillus Mycobacterium tuberculosis. Almost 20% of all new patients with TB have extrapulmonary TB (2). GUTB is the second most common form of extrapulmonary TB. The development of GUTB arises commonly from the spread of pulmonary TB. Urinary tract as initial infection is infrequent and associated extensive calcification of the whole urinary system is rare. We report a 66-year-old man who presented with diffuse calcification of the urinary system and disseminated miliary TB due to GUTB.

Case Report
A 66-year-old male, without notable medical history, was admitted to a local hospital with complaints of urinary frequency, urgency and urine pain for half a month in January 2010. He was diagnosed with urinary tract infection and a course of treatment with Levofoxacin was recommended. This treatment initially attenuated his symptom. But over the next 1 year he had replacing urinary irritation symptoms and gross hematuria. Plain X-ray abdomen and the ultrasound showed urinary...
lithiasis on the left side of the ureter. Chest radiographs were reportedly negative. Smear and culture for acid-fast bacilli were not requested. He was diagnosed with urinary lithiasis and given extracorporeal shock wave lithotripsy for treatment. The symptoms were abated, but still break out repeatedly. Until March 2012, he was admitted to our hospital. He was also oliguric (urinary volume <400 mL/day). Abdominal CT images showed extensive calcifications within bilateral kidneys, ureters and bladder with walls thickening and contracture malformation (Fig. 1 A, B, D, E), and thoracic CT images showed diffuse inhomogeneous nodules insufficiently integrated in both lungs (Fig. 1 C).

There was no personal history of, or exposure to, TB. He had received Bacillus Calmette-Guerin vaccine as a child. Laboratory test showed serum urea=49.98 mmol/L (normally 3.2~8.2 mmol/L), creatinine=956 umol/L (normally 53~97 umol/L). Blood TB antibody was positive. Urinary and sputum TB–DNA was 790000 and 19000000 copies (normal level <1000 copies) respectively. *M. tuberculosis* had been found through acid-fast bacilli staining, (3+/4+). TB infected T cells detection ESAT-6 and CFP10 was 10 and >50 SFCS (normal level <6 SFCS) respectively. Anti-HIV, HBsAg, anti-HCV were all negative. The patient was diagnosed with GUTB and hematogenous disseminated pulmonary TB, and a decision was made to start antitubercular treatment. The patient had severe renal failure. So he was given the bedside continuous blood purification (CBP) treatment. Bedside radiography of abdomen demonstrated the shaping calcifications of the kindneys expanding along the ureters (Fig. 1 F). Unfortunately, the patient’s condition deteriorated rapidly, and he died 7 days later. After 2 weeks, cultures of urine and sputum samples yielded a strain of *M. tuberculosis* that was susceptible to all antituberculous agents respectively.

**Discussion**

Most of patients who present with tuberculous genitourinary disease have a known history of prior pulmonary TB or have radiographic evidence of prior subclinical pulmonary infection. Our patient with urinary tract as initial infection site and secondary spread to the lungs is relatively rare. The onset of most GUTB is insidious. Some patients are asymptomatic and only found sterile pyuria in routine urine examination, so they are easy to be misdiagnosed as urinary tract infection (3). Some patients present with hematuria. It can be either microscopic or macroscopic. Gross hematuria is seen in only 10%, but microscopic hematuria is present in up to 50% of the cases (4). Imaging findings can support the diagnosis of GUTB. Renal calcifications are common manifestation of TB. However, calcification of the ureter is rare (5). Our patient was characterized by multiple calcifications of the bilateral pelvicalyceal system and associated bladder and bilateral ureteral calcification. The extensive calcification of the whole urinary system just like this patient is very rare.

The definitive diagnosis of TB involves demonstration of *M. tuberculosis* by microbiological or histopathological methods. Urine culture is the gold standard method for establishing the diagnosis. In the case presented here, *M. tuberculosis* has been found through acid-fast bacilli staining and the urine culture was positive for *M. tuberculosis*. The onset of GUTB is often insidious, and there are many limitations of noninvasive testing in establishing the diagnosis (6). We should be alert to the possibility of GUTB when the urinary irritation symptoms occur repeatedly even without a history of TB. TB must be kept on the differential diagnosis for many lesions and pathologies of the genitourinary system. Through this case suggests timely diagnosis and treatment of TB is extremely important to improve prognosis.

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