Renal Tuberculosis: The Masquerader

Rudi Supriyadi[1], Guntur Darmawan[2,3], Emmy H. Pranggono[4]

[1] Division of Nephrology and Hypertension, Department of Internal Medicine, Faculty of Medicine, Universitas Padjadjaran Hasan Sadikin General Hospital, Bandung, Indonesia.
[2] Department of Internal Medicine, Faculty of Medicine, Universitas Padjadjaran Hasan Sadikin General Hospital, Bandung, Indonesia.
[3] Department of Internal Medicine, Faculty of Medicine, Krida Wacana Christian University, Jakarta, Indonesia.
[4] Division of Respiratory and Critical Illness, Department of Internal Medicine, Faculty of Medicine, Universitas Padjadjaran Hasan Sadikin General Hospital, Bandung, Indonesia.

Corresponding Author:
Guntur Darmawan, MD. Department of Internal Medicine, Faculty of Medicine Universitas Padjadjaran – Hasan Sadikin Hospital. Jl. Professor Eyckman No. 38, Bandung 40161, Indonesia. email: guntur.darmawan@ukrida.ac.id.

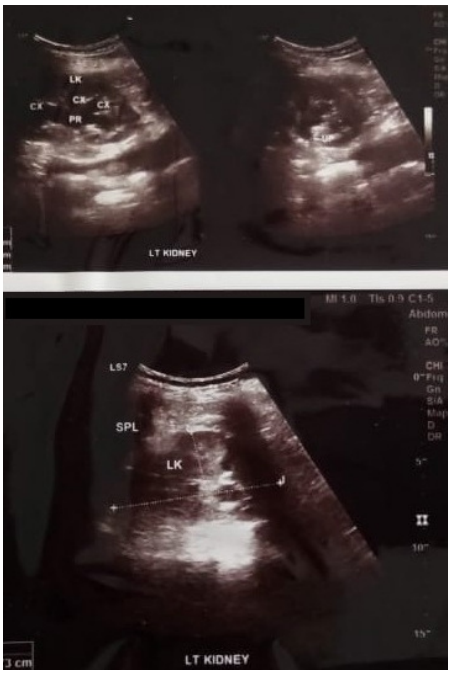

Figure 1.

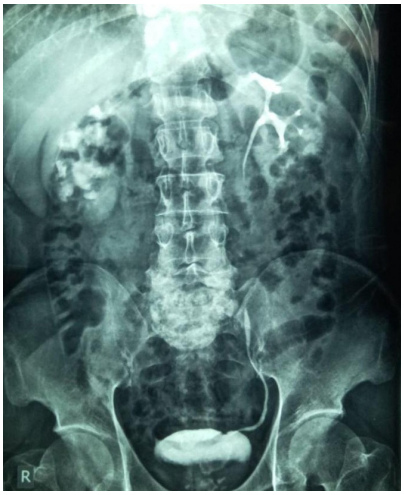

Figure 2.

Tuberculosis (TB) remains a worldwide scourge and the most common cause of mortality from infectious disease. Around 95% of cases occur in developing country. Renal TB is a rare cases that complicates 3-4% of pulmonary TB patients and commonly overlooked in clinical practice due to its symptoms may mimic other diseases.[1-3]

A-39-year-old man was admitted to our institution due to flank pain. He had history of low grade fever and oligouria since 5 months prior. He had no complaint of cough, dyspnea, or night sweat. He was a non smoker and had no past medical history of tuberculosis. Previous 4 months abdominal ultrasound showed left pelvocaliectasis and ureteral dilatation with suspicion of left ureteral stenosis. Ureterolithiasis could not be excluded. No prostate enlargement or vesicolithiasis was seen. Intravenous pyelography (IVP) examination demonstrated similar finding. (**Figure 1 and 2**) Initial laboratory blood examination showed anemia (10.7 g/dl), leukocytosis (14,080/ul), increased in serum creatinin (4.2 mg/dl), ureum (227 mg/dl), and calcium (6.78 mg/dl). Serology

examinations were negative for HIV, HBsAg, anti HCV and blood culture had no growth. Urinary examination revealed severe leucocyturia, hematuria, and negative for bacteria, nitrite and cast. Urine culture was positive for Candida glabrata. Pulmonary X-ray suggested right pleural fibrotic. He was initially diagnosed as multiple myeloma with fungal infection. Nevertheless, additional peripheral blood smear showed neither rouleaux formation nor blast. He underwent percutaneous nephrostomy and got micafungin intravenously. Instead of improving, the patient deteriorated and transferred to intensive room. We then explored the possibility of TB infection. Further examination revealed positive for Mycobacterium tuberculosis in urinary polymerase chain reaction (PCR) test. Tracheal sputum examination was positive for acid fast bacilli staining. There was low level of serum vitamin D2 (5.8 ng/ml). He got TB treatment with rifampicin, isoniazid, pyrazinamide, and ethambutol. Unfortunately, the patient eventually succumbed.

Renal TB is the second most common form of extra-pulmonary TB after lymph node TB. The non specific clinical presentation of renal TB may result in delayed diagnosis and management of the disease, which might worsen morbidity and mortality. Sterile pyuria is the most common clinical finding as seen in this case. There are no specific clinical symptoms or imaging finding, and low yield of culture techniques; therefore, a combination of clinical symptoms, imaging and laboratory examinations contribute to establish the diagnosis of renal TB. In addition, PCR-based test are promising and can significantly increase case detection. Examination of urinary TB PCR gave the diagnostic clue in our case. Urinary TB PCR test may demonstrate up to 100% in sensitivity.[4-7] Other interesting finding in our case was low level of serum vitamin D2 and hypercalcemia. Many studies showed vitamin D deficiency is associated with the risk of TB infection, since vitamin D play important role in human immunity system.[8-10] However, as in other granulomatous diseases, PTH independent-hypercalcemia may occur in TB through ectopic production of 1α, $25(OH)_2D_3$. Pulmonary alveolar macrophages and lymphocytes upregulate expression of $25(OH)D_3$-1α-hydroxylase. Moreover, pleural fluid contains substances such as γ-interferon that might potentiate the expression of $25(OH)D_3$-1α-hydroxylase. The laboratory examination may show suppressed PTH, elevated 1α, $25(OH)_2D_3$, and normal or low 25(OH)D concentrations.[11-13] Whether vitamin D supplementation will be beneficial to TB treatment is still inconclusive. Looking to the risk of hypercalcemia and a study by Xia demonstrating no significant benefit of vitamin D supplementation in TB treatment, further rigorously controlled studies are suggested to fully answer the controversy in vitamin D supplementation.[14]

Renal TB is a great masquerader in daily practice. The diagnosis of extra pulmonary TB is not easy and it needs high level of clinical suspicion, especially when encounters sterile pyuria. Complementary finding of hypercalcemia and low serum vitamin D could help clinician in directing to TB. Learning from this case, we should not overlook fibrotic finding in chest X-ray which could be a starting point of renal TB.

REFERENCES

1. de Oliveira JL, da Silva Junior GB, Daher EDF. Tuberculosis-associated chronic kidney disease. Am J Trop Med Hygiene. 2011;84(6):843-4.
2. Kumar S, Shankaregowda SA, Choudhary GR, Singla K. Rare presentation of genitourinary tuberculosis masquerading as renal cell carcinoma: A histopathological surprise. J Clin Imag Sci. 2014;4.
3. Lima NA, Vasconcelos CC, Filgueira PHO, et al. Review of genitourinary tuberculosis with focus on end-stage renal disease. Revista do Instituto de Medicina Tropical de São Paulo. 2012;54(1):57-60.
4. Abbara A, Davidson RN. Etiology and management of genitourinary tuberculosis. Nature Rev Urol. 2011;8(12):678.
5. Daher EDF, da Silva Junior GB, Barros EJG. Renal tuberculosis in the modern era. Am J Trop Med Hygiene. 2013;88(1):54-64.
6. Ramana K. Pulmonary tuberculosis disseminating and presenting as bilateral hydronephrosis and renal abscess: a potential threat in the era of multi-drug resistant tuberculosis MDR-TB. Am J Infect Dis Microbiol. 2014;2:48-50.
7. Rui X, Li XD, Cai S, Chen G, Cai B. Ultrasonographic diagnosis and typing of renal tuberculosis. Int J Urol. 2008;15(2):135-9.
8. Huang S-J, Wang X-H, Liu Z-D, et al. Vitamin D deficiency and the risk of tuberculosis: a meta-analysis.

Drug design, development and therapy. 2017;11:91.
9. Iftikhar R, Kamran SM, Qadir A, Haider E, Bin Usman H. Vitamin D deficiency in patients with tuberculosis. J Coll Physicians Surg Pak. 2013;23(10):780-3.
10. Kearns MD, Tangpricha V. The role of vitamin D in tuberculosis. J Clin Transl Endocrinol. 2014;1(4):167-9.
11. Araujo CA, Araujo NA, Daher EF, et al. Resolution of hypercalcemia and acute kidney injury after treatment for pulmonary tuberculosis without the use of corticosteroids. Am J Trop Med Hygiene. 2013;88(3):592-5.
12. Peces R, de la Torre M, Alcázar R, Tejada F, Gago E. Genitourinary tuberculosis as the cause of unexplained hypercalcaemia in a patient with pre-end-stage renal failure. Nephrology, dialysis, transplantation: official publication of the European Dialysis and Transplant Association-European Renal Association. 1998;13(2):488-90.
13. Tebben PJ, Singh RJ, Kumar R. Vitamin D-mediated hypercalcemia: mechanisms, diagnosis, and treatment. Endocrine Rev. 2016;37(5):521-47.
14. Xia J, Shi L, Zhao L, Xu F. Impact of vitamin D supplementation on the outcome of tuberculosis treatment: a systematic review and meta-analysis of randomized controlled trials. 2014.