CLINICAL VIGNETTE

Renal Tuberculosis Presenting as a Renal Mass

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Case Report

An 83-year-old female presented for routine evaluation and was noted to have microscopic hematuria. Her past medical history included: Type 2 Diabetes, Hypertension, Dyslipidemia, and Coronary Artery Disease. She had no history of Pulmonary Tuberculosis and no history of smoking or ethanol use. Medications included: lisinopril, metformin, aspirin, atorvastatin, and metoprolol. Her physical examination was unremarkable. CT Urogram showed a left renal mass and cystoscopy was negative. CT-guided biopsy revealed acid-fast bacilli, with negative subsequent cultures.

The chest x-ray revealed old granulomatous disease and PPD was negative. A QuantiFERON-TB Gold test was positive, early morning urine cultures for acid-fast were negative, sputum cultures were negative for acid fast bacillus. Cystocopy was also negative for lesions; and urine cytology was negative as well.

Treatment

The patient was started on anti-Tuberculosis therapy for latent TB with combination of Isoniazid 300 mg daily, Rifampin 600 mg daily, Ethambutol 800 mg daily, Pyrazinamide 15 mg/kg daily for 2 months, and Pyridoxine 25 mg daily. The patient tolerated the therapy well. She completed a full course of nine months and a subsequent CT Urogram, was performed 3 months after the completion of therapy and showed no residual mass.

Discussion

Renal tuberculosis is usually asymptomatic. Renal cortical foci may occur during all stages of tuberculosis. This occurs in the kidneys when multiple granulomas form at the site of
metastatic focus. These typically occur at bilateral cortical or adjacent to glomeruli. They may remain inactive for decades. Although both kidneys are frequently involved, clinical significant
disease causing capillary rupture usually develops in one kidney. The medullary hypertonic environment impairs the phagocytic function. Clinical features include hematuria, pyuria, and occasional dysuria when the bladders involved. Early radiological findings are usually nonspecific, but more chronic changes would include papillary necrosis, and possible urethral strictures. Advanced disease can also include hydronephrosis, parenchymal cavitation, and pelvic calcification are particularly suggestive. Hypertension is not a feature in renal tuberculosis and renal function is usually preserved. However, a rare condition called Tuberculous Interstitial Nephritis may cause renal failure characterized by interstitial granulomas in normal-sized kidneys usually with the presence of active extra-renal tuberculosis. Hypertension in persons with renal tuberculosis is twice as common as in the general population. Treatment with drug regiments containing isoniazid and rifampin as in pulmonary Tuberculosis is recommended. It is unclear if Tuberculous Interstitial Nephritis is actually caused by Tuberculous infection. Urethral Tuberculosis is an extension of the disease from the kidneys and generally extents to the ureterovesical junction. This process develops in about one-half of patients with renal tuberculosis. Bladder tuberculosis is secondary to renal Tuberculosis and usually starts at the ureteral orifice and clinical findings at cystoscopy are superficial inflammation with both edema and erosions, however, granuloma involvement is rare.

REFERENCES

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