Renal tuberculosis in a 4 year old child

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Abstract

A 4 year old female child presented with complaints of low graded fever associated with decreased appetite and failure to gain weight and persistent foul smelling urine. She had a positive history of contact. She was evaluated for pulmonary tuberculosis which came out to be negative. Acid fast bacilli was detected in urine, and ultrasonography of the abdomen revealed renal tuberculosis. The patient was treated with category 1 DOTS therapy.

Keywords: Renal tuberculosis

Introduction

Renal tuberculosis is rare in children [1]. Genitourinary tuberculosis occurs as a complication or late reactivation of pulmonary tuberculosis. It is usually prevalent in the middle aged and young adults [2]. In children, it represents less than 5% of the cases of extrapulmonary disease [3].

Renal tuberculosis usually presents with symptoms such as irritative voiding, hematuria and flank pain. Additionally, presentation as a mass lesion is extremely rare [4]. If diagnosed early, nearly all children can be cured with medical treatment. However, diagnosis is often delayed and a number of children present with non-functioning kidneys, obstructed ureters, shrunken bladders and even chronic renal failure [5].

Case Report

A 4 year old female child presented with complaints of low grade intermittent fever over the past 2 months associated with decreased appetite and failure to gain weight, intermittent abdominal pain and foul smelling urine. There was no history of cough, no history of gross hematuria or dysuria.

The mother was a known case of sputum positive tuberculosis and was on DOTS therapy. Physical examination revealed severe malnutrition (weight for height below -3rd SD for the age). Systemic examination revealed no pathological findings.

Keeping in view the history and presence of a contact the child was evaluated for pulmonary tuberculosis. Gastric lavage was examined 3 times for presence of acid fast bacilli (AFB) which was negative. Tuberculin test was negative as well. Examination of the gastric fluid for MTB by PCR was also negative. Chest x ray was normal. Haemogram showed anemia (Hb = 10.2 gm/dl). ESR was raised (34 mm at the end of 1st hour)

Ultrasonography was done which showed urinary bladder wall thickening of 4.8 mm suggestive of cystitis, and ectasia in the upper pole of the right kidney. Micturating cystourethrogram was normal and showed no reflux or voiding dysfunction. Intravenous pyelography revealed a dilatation of the right pelvicalyceal system.

Serum creatinine and blood urea were within normal limits. Urine examination revealed microscopic hematuria and 4 to 5 pus cells per high power field. Two 24 hour urinary samples were sent for Ziehl-Neelsen staining, which revealed acid fast bacilli.

Discussion

Renal tuberculosis is a secondary tuberculous infection which is caused due to the hematogenous dissemination during primary infection or reactivation. The cortex of the kidney is an appropriate place for lodging of bacilli since the rate of oxygenation is high. Afterwards, the infection is transmitted from the cortex to the medulla and numerous tubercles will form. At this point, cell-mediated immunity (CMI) will stop bacilli proliferation and fibrous tissue and then scar will form.
Different causes such as stress, old age, immune deficiency, etc. will cause reactivation. In our patient, severe malnutrition (growth chart below 3%) and young age, were the predisposing factors for the severe reactivation of tuberculosis [6].

Sterile pyuria is the most common clinical finding. Hematuria, flank pain, dysuria are also reported. Severity of the bladder infection correlates with the severity of kidney infection. But the involvement of bladder is by far more scarce in children than in adults. The positive finding in this child was microscopic hematuria, pyuria and presence of acid fast bacilli in the urine coupled with ultrasonography of the abdomen which revealed cystitis.

A diagnosis was made of extrapulmonary renal tuberculosis and category 1 treatment was started which includes 2 months of intensive phase antitubercular therapy with isoniazid, rifampicin, pyrazinamide and ethambutol, followed by at least 4 months of continuation phase therapy with isoniazid and rifampicin.

In only 3 months this treatment can achieve an 86.4% negativization of previous positive urinary AFB, mycobacterial cultures or PCR tests.

Gloor and May classification of Renal tuberculosis [7]:

1. Miliary form
2. Nodular cavernous form equivalent to chronic renal tuberculosis associated with tissue destruction
3. Pyelitis caseosa – caseation of the mucosa of the renal pelvis, ureter and urinary bladder
4. ‘Kittniere’ – final stage of total destruction of the kidney with occlusion of ureter.

Conclusion

Renal tuberculosis should be considered in a case of hematuria with sterile pyuria especially in a patient with immunocompromise, malnutrition or positive history of contact.

References