

The experience with our two cases shows that patients with sarcoidosis may often not be properly diagnosed. The condition must be suspected clinically in patients with involvement of multiple organ systems. Therapy with corticosteroids in selected patients leads to rapid relief of symptoms and possibly decreases the incidence of long term complications.

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Calcification in Renal Tuberculosis

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Genito-urinary tuberculosis consti-

tutes the single largest group of extrapulmonary tuberculosis in all age groups. It is estimated that 15-20% of

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cases with primary pulmonary tuberculosis are likely to develop genito-urinary tuberculosis later, of which kidney alone or alongwith bladder or ureter is affected in more than 70% cases(1). There is paucity of literature on renal tuberculosis in childhood. We report a case of renal tuberculosis with calcification in a 7-year-old girl.

Case Report

A 7-year-old girl was admitted, with a sudden onset of painless hematuria of three days duration. There was no history of fever, pain abdomen, frequency or urgency of micturition, bleeding from any other site, cough, icterus or recent abdominal trauma. There was history of hematuria 2 months ago which lasted for 3-4 days only. She was not vaccinated. She belonged to poor socio-economic status and shared a small room alongwith other family members. Five years ago, the father was treated for pulmonary tuberculosis at a Government Hospital.

On examination, the weight and height were normal for her age. She was pale, with no pedal edema or significant lymphadenopathy. The blood pressure was 90/70 mm Hg. The examination of cardiovascular, respiratory, nervous systems and abdomen were unremarkable.

The investigations, revealed a hemoglobin of 9 g/dl, total leucocyte count of 9000/cu mm and differential count of 32% polymorphs, 64% lymphocytes. The erythrocyte sedimentation rate was 95 mm in first hour. The blood levels of calcium, phosphorus and alkaline phosphatase were normal. Urine examination showed mild albuminuria, plenty

of red blood corpuscles and 60-70 polymorphs per high power field, without any casts. The 24 hours urinary protein estimation was 400 mg/day. The culture of urine was repeatedly sterile on routine media. The Mantoux test done with 5 TU resulted in an induration of 24 mm at 48 hours. The skiagram of chest showed right parahilar lymphadenopathy. In addition, calcification in the right kidney was evident on plain X-ray of abdomen (*Fig. 1*). The ultrasound examination of abdomen showed a shrunken right kidney (3.0 x 2.5 cm) and hypertrophied left kidney (10.5 x 8 cm). Intravenous urography confirmed, total non-functional right kidney (*Fig. 2*). Three consecutive morning urine samples demonstrated the presence of acid fast bacilli. *Mycobacterium tuberculosis* was grown on culture of urine.

The patient was given pyrazinamide, INH, rifampicin and ethambutol for two months followed by INH, rifampicin and etharrbutol. Her urine was sterile for *Mycobacterium tuberculosis* after three months of therapy. She was considered for total nephrectomy but parents did not give consent.

Discussion

Genito-urinary tuberculosis is not commonly seen in children. In a large series of childhood tuberculosis from India, not a single case of genito-urinary tuberculosis has been reported. However, in four other series encompassing all age groups, only 6 out of 535 patients of genito-urinary tuberculosis were less than 10 years of age(1-4). Among autopsied cases of childhood tuberculosis kidney involvement had been noted in one third of miliary tuberculosis and

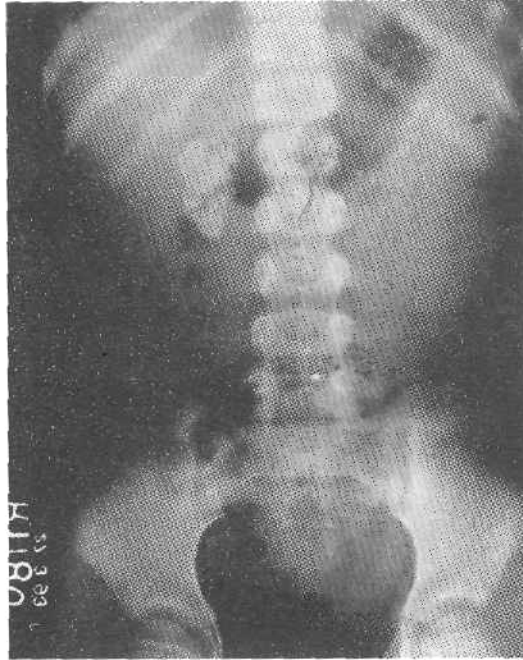


Fig. 1. Plain X-ray abdomen showing calcification in right kidney.

6% of disseminated tuberculosis(5).

Renal tuberculosis is believed to be secondary to hematogenous dissemination of *Mycobacterium tuberculosis* from the primary focus in lungs. The bacilli get implanted in the blood vessels close to glomeruli, lead to destruction of renal parenchyma, by caseation necrosis, which slough out into a calyx, reach bladder via ureter or heal spontaneously. Healing occurs by formation of reticula around lesions which mature into fibrous tissue, and later attract calcium salts(6).

The manifestations of urogenital tuberculosis have been reported to vary from one to ten years after the onset of primary in the lungs(1). The diagnosis of renal tuberculosis is often a problem

because of its protean manifestations and the problem is further compounded because the duration of the history may not always have any relationship to the severity of the disease(1). The symptoms most commonly seen are frequency of micturition (44%), renal pain, burning micturition in 24% and hematuria in 20%. Irregular fever and constitutional symptoms are seen in about 20% of the cases(4). Still rarely, one might see a presentation as a renal cortical mass(7). Persistence of pyuria with sterile culture on routine media should lead to a strong suspicion of renal tuberculosis.

A definite diagnosis is established by isolation of tubercle bacilli in the early morning specimens of urine. Plain X-ray abdomen may show calcification in

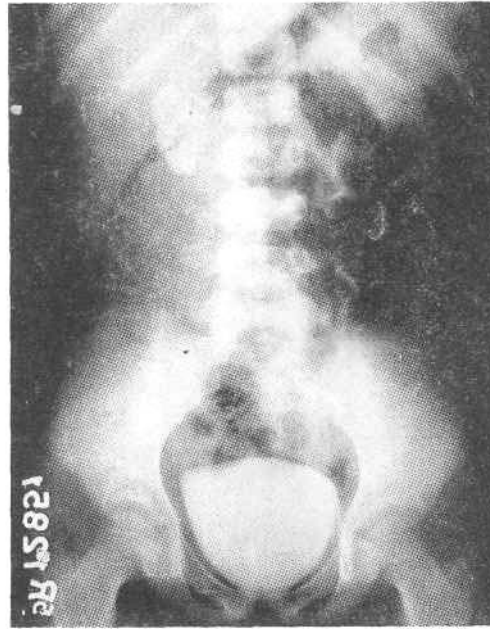


Fig. 2. Intravenous urography showing calcification in right kidney and non-functional right kidney.

renal areas as seen in our patient. Intravenous urography is required for assessment of lesions and renal functions(4,7).

Renal calcification has been reported in 7.5-24% cases of renal tuberculosis(1,6-8). The incidence has been on the rise due to increased survival of these patients following therapy or due to secondary infections or stricture formation(8,9).

Three types of calcifications have been noticed in renal tuberculosis: (i) Diffuse spotty calcification; (ii) Large calcified abscess; and (iii) Medium sized calcification causing deformity of calyx(10). Calcification is more common

with severe cases of renal tuberculosis. The bacilli persist even in non-functioning, calcified kidney though urine may be negative for *Mycobacterium tuberculosis*(11,12).

The exact pathogenesis of calcification in renal tuberculosis is not known. It has been postulated that damage of tubular cell membrane results in intracellular accumulation of cations like calcium. This further impairs the already compromised cellular respiration and oxidation, thus resulting in cell death. The disintegration of nucleoprotein and phospholipins from dead cells liberates phosphate ions which forms lime salts of calcium phosphate and carbonate in proportion similar to that of bone(13).

Chemotherapy and surgery have been the mainstay of treatment of renal tuberculosis. Antitubercular drugs have to be given for one year even if kidney is non-functional(14). Nephrectomy has been indicated in a non-functioning, calcified, tuberculous kidney or a grossly compromised kidney due to destructive ureteric pathology or repeated secondary infections(15).

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