International Journal of Health Sciences and Research
www.ijhsr.org ISSN: 2249-9571

Case Report

Extensive Renal Calcification due to Tuberculosis

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Received: 21/02//2014 *Revised: 20/03/2014* *Accepted: 29/03/2014*

ABSTRACT

Tuberculosis remains a worldwide scourge and its incidence appears to be increasing due to various factors, such as the spread of Human Immunodeficiency Virus (HIV) and Acquired Immunodeficiency Virus (AIDS). The insidious onset and non-specific constitutional symptoms of genitourinary tuberculosis (GUTB) often leads to delayed diagnosis and rapid progression to a non functional kidney. Due to haematogenous dissemination of TB, there is potential risk of involvement of the contra-lateral kidney too. We report a case of Extensive Renal Calcification due to Tuberculosis in a 37 year old male presenting with symptoms of vague abdominal pain, burning micturition and pyuria, who was treated with AKT and was operated later.

***Keywords*:** Nephrocalcinosis, Genitourinary Tuberculosis, Sterile Acid Pyuria, Langhans Giant Cells

INTRODUCTION

Genito-urinary tuberculosis constitutes the single largest group of extrapulmonary tuberculosis in all age groups. It is estimated that 15-20% of cases with primary pulmonary tuberculosis are likely to develop genito-urinary tuberculosis later, of which kidney alone or along with bladder or ureter is affected in more than 70% cases. [1]

CASE REPORT

A 37 years old male, presented with dull aching right flank pain since last 1 year with burning micturition since 1month, passing purulent urine since 13 days and fever since 7 days. On examination, the patients' weight and height were normal. His vitals were normal. The central nervous system, cardiovascular system and abdomen were normal. USG revealed a hyperechoic shadow of the whole right kidney. X-Ray KUB (Fig. 1) of the patient showed an extensive calcification of the right kidney. Three consecutive morning Urine sample showed acid fast bacilli and culture was positive for *Mycobacterium tuberculosis*. His Blood urea and Serum Creatinine were normal so a CT IVU (Fig. 2) was done which revealed a Non- Functioning Right Kidney with Normal functioning Left Kidney.

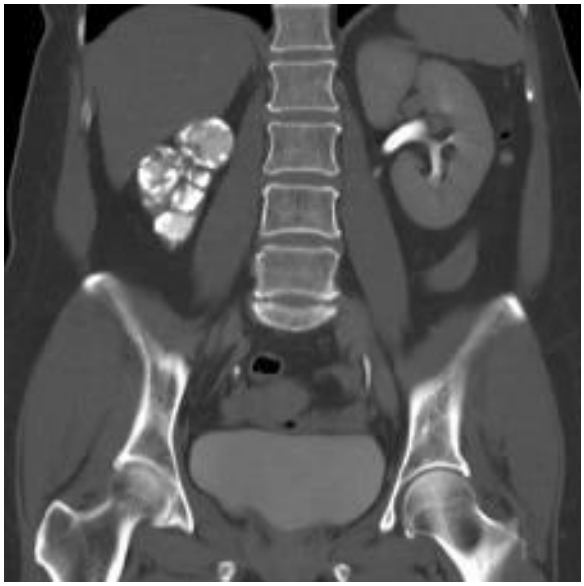

Figure 1. X Ray KUB.

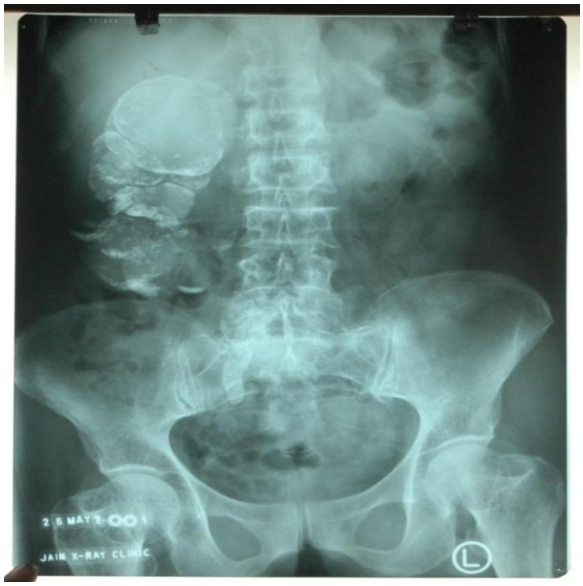

Figure 2. CT IVU.

RESULT

Patient was managed by AKT followed by Right NephroUreterectomy. The Specimen (Fig. 3) was sent for Histopathological Examination. It showed Langhans Giant Cells (Fig. 4).

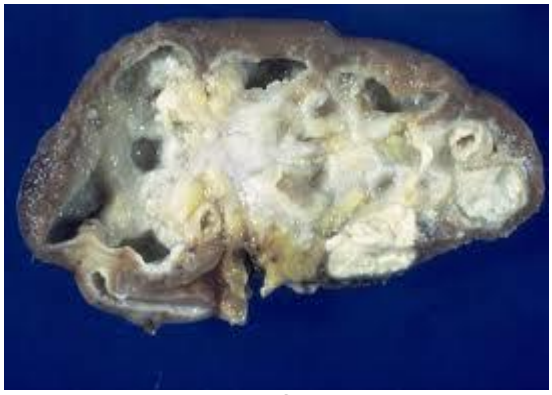

Figure 1. Specimen.

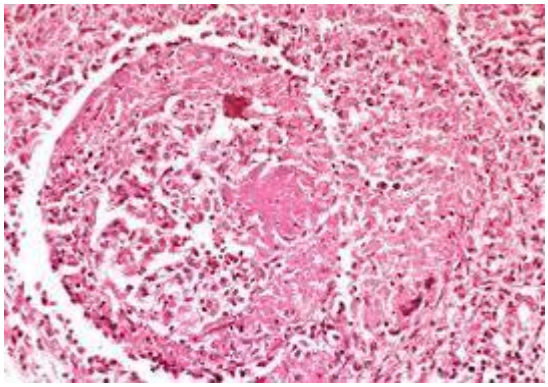

Figure 4. Histopathological Slide.

DISCUSSION

Today, urinary tuberculosis is infrequent and renal calcification is not uncommon, but associated ureteral calcifications are rare. [1] IVU has traditionally been the gold standard tool to diagnose and evaluate urinary tuberculosis, but CT is the most sensitive method of identifying renal/ureteral calcification. [1] In our case, owing to extensive disease, the calcification was clearly visible on the CT IVU. Calcification in the ureteral wall may also be seen in schistosomiasis, but calcification of the kidney extending along the ureter is virtually diagnostic of tuberculosis. [2] Ureteral calcification in tuberculosis is intraluminal and appears as a cast of the ureter, which is thickened and not dilated. [2] This patient had received complete ATT for pulmonary tuberculosis. Despite this, he developed extensive renal disease. Therefore, in patients with a history of treated primary tuberculosis, physicians should be highly suspicious for tuberculosis

in other organs to avoid irreversible damage, and in case of 'Sterile Acid Pyuria', culture for tuberculosis must be done.

CONCLUSION

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. [3] 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. 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. [5] 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 renal area as seen in our patient.

Intravenous urography is required for assessment of lesions and renal function. [4,5]

Renal calcification has been reported in 7.5-24% cases of renal tuberculosis. [1,3,5,6] The incidence has been on the rise due to increased survival of these patients following therapy or due to secondary infections or stricture formation. [6,7] 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. [8] Calcification is more common in severe cases of renal tuberculosis. The bacilli persist even in non-functioning, calcified kidney though urine may be negative for *Mycobacterium tuberculo*sis. [9,10] 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 phospholipids from dead cells liberate phosphate ions which forms lime salts of calcium phosphate and carbonate in proportion similar to that of bone. [11]

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. [12] 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. [13]

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How to cite this article: Gindodia KR, Ansari AAA, Siddiui MM. Extensive renal calcification due to tuberculosis. Int J Health Sci Res. 2014;4(4):240-243.

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