ISSN- O: 2458 - 868X, ISSN–P: 2458 – 8687
National Library of Medicine - ID: 101731606

International Journal of Medical Science and Innovative Research (IJMSIR)
IJMSIR : A Medical Publication Hub
Available Online at: www.ijmsir.com
Volume – 7, Issue – 5, October – 2022 , Page No. : 116 – 120

Histomorphological Spectrum of 3 Cases of Renal Tuberculosis and brief review of literature

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Citation this Article: Dr Pragati Upasham, Dr. Hirina Jairath, Dr. Asawari Arwikar, "Histomorphological Spectrum of 3 Cases of Renal Tuberculosis and brief review of literature", IJMSIR- October - 2022, Vol – 7, Issue - 5, P. No. 116 – 120.

Type of Publication: Case Report

Conflicts of Interest: Nil

Abstract

Tuberculosis is a multi-organ disease with varied clinical presentation. Genito-urinary tuberculosis is caused by Mycobacterium Tuberculosis complex. Lung is the most common affected organ, however the 2nd most common presentation of extra pulmonary tuberculosis is genito-urinary tuberculosis accounting to incidence of 30%-40%, with kidney being the most common location. It is a major health problem in the developing countries including India. Haematogeous spread from an active focus of infection results in urogenital tuberculosis. It is a common complication accounting for about 3-4% in patients with pulmonary tuberculosis. It may present with symptoms like fever, haematuria, dysuria, loss of weight, flank pain. Here in we discuss clinico-histological spectrum of 3 cases of renal tuberculosis.

Keywords: Histopathology, Tuberculosis, Extrapulmonary, Renal Tuberculosis, Caseous necrosis, Granulomas, Urogenital Tuberculosis (UGTB)

Introduction

Urogenital tuberculosis is caused due to late reactivation or complication of pulmonary tuberculosis and affects the age group of 25-45 years of age. Haematogeous spread from the pulmonary site of infection results in lodgement of the bacteria in the glomerular and peritubular capillary bed giving rise to tuberculosis of the kidney. [1]. Other parts of urinary tract are affected by direct extension. [2]. The WHO Global report of 2017 declared that of all tuberculosis cases in the world 15% accounted for extrapulmonary tuberculosis.[3]. Among the patients affected with pulmonary tuberculosis the occurrence of genitourinary tuberculosis ranges from 2%-20% depending on where they reside and increases upto 20% in developing countries.[4]. Renal tuberculosis in paediatric population is quite rare and accounts for less than 5% of cases of extrapulmonary tuberculosis.[5] In recent years, prevalence of extrapulmonary tuberculosis has increased in immunocompromised patients. The difficulty in diagnosis of extrapulmonary tuberculosis is sometimes attributed to its varied presentations and wide morphological spectrum. Hence prompt evaluation should be done by doing urine routine examination or urine culture in a patient presenting with pyuria or haematuria. Therefore, early diagnosis and early treatment of tuberculosis is of utmost importance to prevent end stage renal failure. In the present study, selected 3 cases of renal tuberculosis are being discussed

in reference to their clinical and histopathological presentation.

Materials and Method

A study conducted in a tertiary care centre for a period of 3 years.

Results

4 cases of renal tuberculosis of 108 cases of extrapulmonary tuberculosis diagnosed on histopathology were identified.

Case Report

Over a period of 3 years a study was conducted in a tertiary care centre. Here 4 cases of renal tuberculosis of 108 cases of extrapulmonary tuberculosis diagnosed on histopathology were identified. However 3 cases are being discussed, the 4th case excluded as complete clinical details were not available.

Case report 1: A 39 year male came with pain in the left lumbar region radiating to groin, evening rise of temperature, loss of weight and appetite. Patient was a known case of pulmonary tuberculosis and was on AKT. All routine blood investigations were within normal limits. HHH- negative. Urine Analysis- (WBC 5-10/hpf, RBC 15-20/hpf, protein 1+). CT-KUB revealed a left ureteric stricture causing hydronephrosis and thinning of renal parenchyma. Left nephrectomy with a part of ureter was sent for histopathological examination.

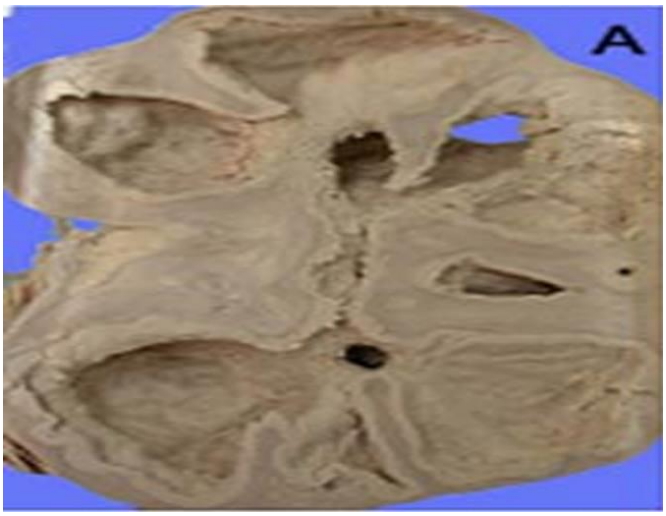
Case report 2: A 60 year female presented with increased frequency of micturition, incontinence since 4 years and has increased since 4 months. All routine blood investigations were within normal limits. HHH - negative. Urine analysis- (WBC 8 -10/hpf, RBC 1-2/hpf, protein 2+). CT-KUB revealed hydronephrosis with small capacity bladder 15-20cc (thimble bladder). Left Nephrectomy with a part of ureter was sent for histopathological examination.

Case report 3: A 7 year old male came with pain in abdomen. No previous history of pulmonary tuberculosis. USG revealed right hydronephrosis with hydroureter. Clinical diagnosis was given as tuberculosis of the kidney. Right nephrectomy with a part of ureter was sent for histopathological examination.

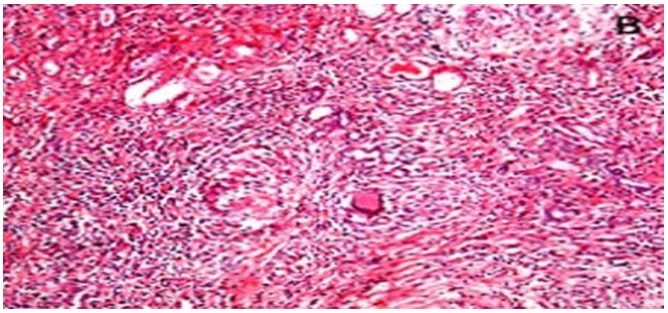
The summary of gross and microscopic findings of presented cases is explained in Table 1.

Case No.	Age	Sex	Gross	Microscopy
1.	39 years	Male	On Gross examination left kidney measured 13x10x5 cm with ureter measuring 3.5 cm. Externally grey brown with multiple cystic nodules. C/S: loss of cortico-medullary differentiation, dilatation of renal pelvis and calyces and areas of necrosis seen. C/S of ureter revealed a stricture. (Figure 1a)	Microscopy revealed granulomas composed of central caseous necrosis, epithelioid cells, langhans cells, lymphocytes and fibroblasts. (Figure 1b)
2.	60 years	Female	Gross examination revealed left kidney measuring 9x6x2 cm with ureter measuring 12 cm. Externally grey brown, firm, irregular, boss elated. Capsule stripped off.	Microscopy revealed granulomas composed of central caseous necrosis, epithelioid cells, langhans cells, lymphocytes and fibroblasts.

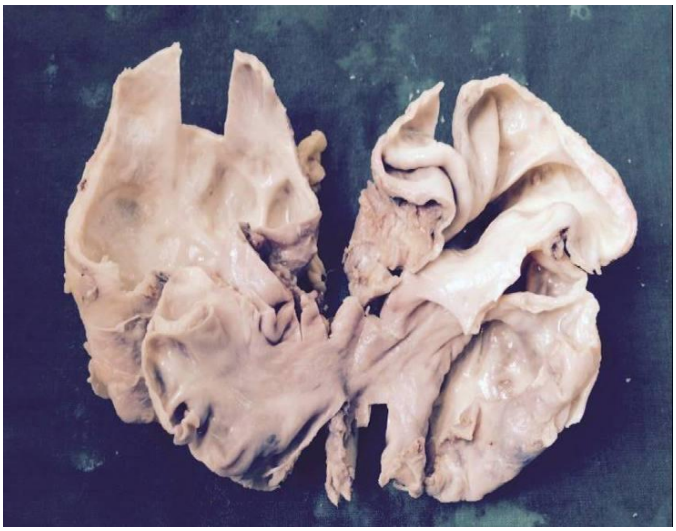
			Cut surface showed cheesy material and loss of cortico-medullary differentiation. Multiple dilated cystic spaces seen. (Figure 2a)	Interstitium showed fibrosis and mixed inflammation and thyroidisation of tubules was noted. (Figure 2b)
3.	7 years	Male	Kidney showed extensive necrosis (Fig 3a)	Histopathological sections from Case no 3 showed extensive caseation. However glass slide of case 3 could not be retrieved.



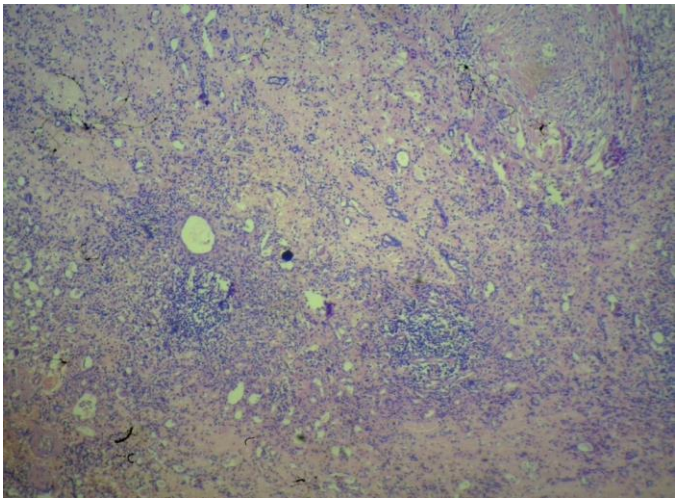
Case 1 - Figure 1a: C/S: Cortico-medullary differentiation lost. Renal pelvis and calyces are dilated. Multiple cystic cavities seen.



Case 1 - Figure 1b: Multiple granulomas composed of central caseous necrosis, epithelioid cells, langhans giant cells, lymphocytes and fibroblasts.



Case 2 - Figure 2a: Cortico-medullary differentiation is lost. Multiple non- communicating cysts seenshowing white raised granular areas Cheesy material seen.



Case 2 Figure 2b: Multiple granulomas composed of central caseous necrosis,epithelioid cells, langhans giant cells ,lymphocytes and fibroblasts.

Case 3 Figure 3a: Kidney showed extensive necrosis.

Discussion

Tuberculosis remains the most common cause of mortality worldwide from infectious diseases. A higher perfusion and oxygenation rate increase the probability of proliferation of bacteria in glomerular and peritubular capillary bed. Patients with good cellular immunity inhibit bacterial duplication, further resulting in confinement of the disease to the cortex. [1]. Failure of host defense mechanism leads to reactivation of cortical granulomas and futher coalesce to form ulcers which may discharge pus and mycobacteria in the urine. Rupture of capillaries result in spread of organism to proximal tubule and loop of Henle leading to development of large caseating granulomas along with papillary necrosis. [1]. Disease progresses with granuloma formation, caseous necrosis and cavitation which ultimately leads to destruction of the entire kidney. Regional spread of the organism into renal pelvis, ureters, bladder can occur due to communication of granulomas with the collecting system. Healing response of the host results in fibrosis (scar formation), calcification and stricture formation contributing to obstruction or progressive renal dysfunction. If the lesions are left untreated, they may form a tubercular abscess in the parenchyma. Subsequently it leads to the formation of perinephric abscess and the entire kidney is substituted by caseous material (putty kidney), which may further undergo calcification (cement kidney), leading to renal failure. [6]In our case series case 1 and 3 didn't have any prior history of pulmonary tuberculosis, whereas patient was a known case of pulmonary tuberculosis and was on AKT in case 3. All three cases we HIV negative. The most common way of clinical presentation is in the form of irritative voiding symptoms (frequency, urgency and dysuria). Other common presentations of urogenital tuberculosis are : Recurrent urinary tract infection, sterile pyuria with or without hematuria, renal mass (hydronephrosis/pyonephrosis), infertility, pelvic inflammatory disease, renal failure. Other constitutional symptoms are flank pain, non-healing wounds, sinuses or fistulas, fever weight loss, fatigue, anorexia, abdominal pain.[1] For the detection of bacilli in urine routine examination or culture, at least 3 first- morning-void urine samples are collected. As viability of mycobacteria decreases with prolonged exposure to acidic urine, so morning void samples are preferred. [1] Our study revealed more or less similar clinical features as given in the literature such as mild to moderate flank pain, loin to groin pain, loss of weight & appetite, evening rise of temperature, increased frequency of micturition and incontinence. Finding mycobacterium tuberculosis in urine or tissue culture confirm the diagnosis of UGTB[4]

All 3 cases were suggestive of pyuria but details about urine culture examination could not be obtained. CT findings in renal tuberculosis reveal changes such as focal caliectasis, calcifications, hydronephrosis, cortical thinning and soft tissue masses. Commonly a renal mass indicates renal cell carcinoma, metastasis, lymphoma or an abscess.

Genitourinary tuberculosis can rarely manifest as pseudotumors, which are commonly due to hypertrophied column of Bertin, unusual shaped kidney or renal dysmorphism [6] . Ureteric tuberculosis is characterised by thickened ureteric wall and structures and distal third of the ureter is commonly involved. Bladder tuberculosis is characterised by reduced bladder volume with wall thickening, ulceration and filling defects. [6] CT findings in all three cases mainly revealed hydronephrosis and thinning of renal parenchyma.

Gross of our cases revealed some significant findings on gross and microscopic examination. On gross examination, external surface of the kidney showed grey brown multiple cysts and on cut surface showed loss of cortico-medullary differentiation, dilatation of renal calyces and pelvis and caseous material along with areas of necrosis. Microscopy of our cases revealed granulomas composed of central caseous necrosis, epithelioid cells, lymphocytes, langhans giant cells and fibroblast. Also thyroidisation of dilated tubules was seen. Caseous necrosis was seen in all the cases. However, we could not retrieve glass slides for photographic purpose in case number 3.

The differential diagnosis include: Chronic pyelonephritis, papillary necrosis, medullary sponge kidney, caliceal diverticulum, renal cell carcinoma, and transitional cell carcinoma and xanthogranulomatous pyelonephritis. [1] However the diagnosis in all of our cases was very evident based on gross and microscopy findings which were pointing towards renal tuberculosis.

Conclusion

Early pre-operative diagnosis is advisable of genitourinary tuberculosis is essential to prevent its complications and surgical removal of the kidney [5]. Biopsy can be used as a diagnostic tool in cases of doubtful kidney lesions and histopathological examination can confirm the diagnosis. Thus pre-operative diagnosis can save the kidney. In tuberculosis endemic areas, possibility of renal tuberculosis should be considered in patients presenting with atypical renal masses and urinary symptoms like pyuria, haematuria or urinary incontinence.

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