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Case Report

Classical Renal Tuberculosis Presented As Recurrent Sterile Pyuria and End Stage Kidney

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ABSTRACT

Genitourinary tuberculosis (GUTB) is the most frequent type of extrapulmonary tuberculosis. However the diagnosis is generally missed due to lack of classical signs and symptoms of renal tuberculosis (TB). As the signs and symptoms of renal tuberculosis mimics those of other infections of the kidney such as sterile pyuria, pain in abdomen, blood in urine etc. Clinical suspicion of this rare entity is necessary to prevent unnecessary avoidable complications as end stage renal disease. Evidence as TB is a cause of end stage renal failure worldwide, is very scanty. Herein we report a rare case of 35 years old female presented with recurrent sterile pyuria and end stage kidney. The patient later on histopathologically diagnosed as classical / primary renal tuberculosis, as clinical and imaging modalities suggested right kidney was the primary site of the infection.

Keywords: Genitourinary tuberculosis, primary tuberculosis, sterile pyuria, tuberculosis, end stage kidney.

INTRODUCTION

Tuberculosis (TB) is a common disease with 8-10 million new cases occurring annually worldwide. After lymphadenopathy, the most common form of non pulmonary TB is genitourinary tuberculosis (GUTB), accounting for 27 % (with a range of 14-41%) of several surveys in USA, Canada and the UK. [1] The incidence of GUTB in developing countries as India is still alarming in view of ill health, lower socioeconomic status and high incidence of HIV infection. [1]

GUTB is a relatively uncommon and under recognized disease worldwide.[2] GUTB is often missed by the physicians due to lack of suspicion, negligence and rarity of

its presentation. [1] It usually presents as sterile pyuria, recurrent urinary tract infections, hematuria of unknown origin or abdominal pain. [2]

Classical renal tuberculosis is a well known cause of urinary tract scarring and calcification with sometimes renal dysfunction. [3] However progressive renal failure is an uncommon cause of TB and end stage renal failure by GUTB is rarest, as worldwide data for its evidence is limited and scanty. [4]

We present such rare case of classical (? primary) renal tuberculosis in a 35 year old female who presented with recurrent sterile pyuria and end stage kidney with lack of evidence of primary lesion in lung or anywhere in the body by clinical and imaging modalities of diagnosis.

CASE REPORT

A thirty five year old female was referred to the urologist with chief complaints of recurrent pyuria, urinary tract infection, pain in the abdomen and hematuria of unknown origin since 6 months. The patient was averagely built and averagely nourished. She had two live children with no history of abortion/miscarriage. There was no clinical history or systemic symptoms of tuberculosis. No past history of contact with active TB patient. Her family history was not significant. There was no history of diabetes, hypertension or dialysis to the patient. The patient's clinical examination was unremarkable. Physical examination of the urine showed pale yellow, hazy appearance with trace of albumin present with absent sugar or acetone, bile salts / pigment The serial urine microscopy showed Pus cells – 30 to 35/hpf and RBCs – 14-16/hpf. No cast/crystal was detected. Occult blood was positive. Two urine cultures were collected over a period of 2 weeks but they failed to grow any organism. Biochemical investigations indicated slightly elevated creatinine (1.6 mg/dl). X – ray chest was within normal limits. Ultrasonography (KUB, Pelvis) showed enlarged grade III hydronephrotic right kidney. Left kidney, bladder, uterus, ovaries and adnexa were unremarkable.

PET- CT scan of kidneys were done. Renal perfusion images and static renal scinti images and computer generated renogram curves were obtained after intravenous administration of 5 ml of 99m Tc- DTPA. The study was done pre and post lasix and showed no appreciable perfusion in the region of right kidney, whereas perfusion to the left kidney appeared normal. Conclusion given was-- the right kidney was not visualized throughout the examination s/o a non functioning status. The left kidney showed cortical function and normal excretory pattern. With all above mentioned investigations, clinically diagnosed as right sided end stage kidney and nephrectomy was performed. The resected specimen was sent for histopathology.

Gross findings:

We received right nephrectomy specimen totally measuring 10 x 8 x 4 cms. Externally, kidney appeared swollen, enlarged with dilated pelvis and fibrotic areas. On cutting open, loss of corticomedullary structures by variable areas of necrosis, abscesses and cystic change was seen. Large yellowish, granular, friable, necrotic nodule was noted at the upper pole measuring 1.8 x 1.5 cm. (Figure 1). Ureter measured 2 cm in length and showed gross thickening. Cut section of the ureter also showed yellowish tiny necrotic nodules.

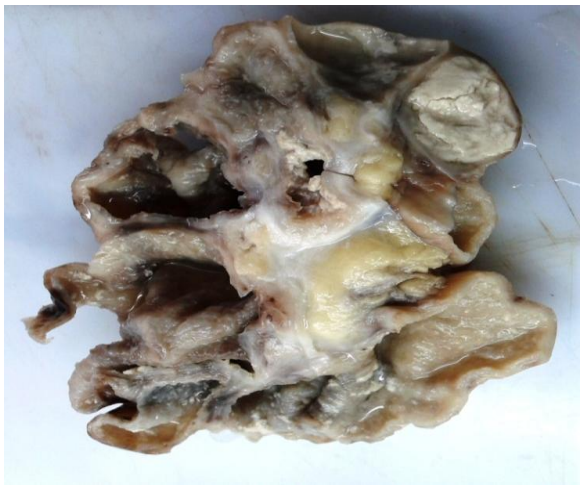

Figure 1: Gross specimen of cut section of kidney showing loss of corticomedullary structures by cystic change, abscess and large yellowish friable necrotic nodule at upper pole.

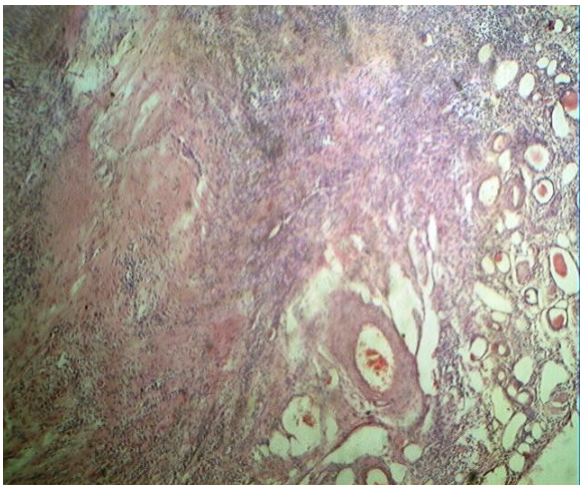

Figure 2: Photomicrograph showed complete effacement of renal architecture by fibrosis and diffuse infiltration by mononuclear cells in tubules, interstitium and glomeruli with changes of chronic pyelonephritis. (H&E, x100)

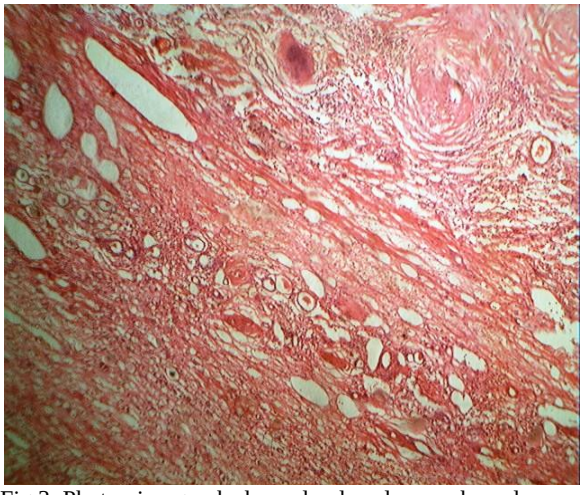

Fig 3: Photomicrograph showed reduced parenchymal cellularity with fibrosis and granulomas formations. (H&E, x100).

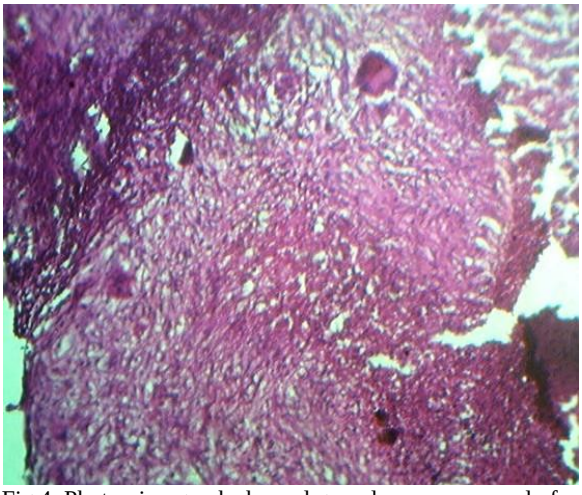

Fig 4: Photomicrograph showed granulomas composed of clusters of epithelioid cells, Langhans giant cells, lymphocytes and central amorphous, pink granular caseation necrosis. (H&E, x400).

Light microscopy:

Multiple sections from the renal tissue showed extensive effacement of architecture by dense, diffuse infiltration by lymphocytes, plasma cells and polymorphs involving tubules, interstitium and glomeruli. Tubules were mainly atrophic, lined by flattened epithelium. The lumina showed dark eosinophilic material s/o thyroidization. Peritubular fibrosis was noted. The glomeruli were reduced with decreased cellularity and showed extensive fibrosis (Figure 2). Renal parenchyma showed areas of granuloma formations. The granulomas composed of small clusters of epithelioid cells, Langhans giant cells, occasional foreign body giant cells and peripheral rim of lymphocytes (Figure 3). Areas of caseation necrosis were also seen (Figure 4). Interstitial stroma showed cystic change, fibrosis and mild diffuse mononuclear cells infiltration. 20 % Zeihl

Nielsen stain revealed few acid fast bacilli. Final histopathological diagnosis given was classical (/ primary) renal tuberculosis with chronic interstitial nephritis and changes of end stage kidney.

DISCUSSION

Tuberculosis is the most important communicable disease in the world. It transcends age, sex and nationality. About 80 % of the world's cases of tuberculosis are found in developing countries like India, where the disease continues a heavy toll in the form of high mortality and morbidity. It is estimated that India alone has got one fifth of the world's total tuberculosis population. [5] TB is possibly the greatest emerging HIV related problem at present. [5]

Tuberculosis (TB) in general and genitourinary tuberculosis (GUTB) in particular can be defined as an infectious, systemic, chronic and granulomatous disease. [6] As all forms of TB, GUTB too affects mostly people in the reproductive age group between 18 and 59 years. [7] Lower urinary tract symptoms have been reported as the most frequent symptoms in GUTB. [7] Renal tuberculosis accounts for the 50 % of cases of GUTB with flank pain as a frequent symptom. [7]

GUTB classically presents as 'painless hematuria' and 'sterile pyuria' as in our case. Positive urine cultures for mycobacterium tuberculosis occur in 5-7 % of the tuberculous patients. [5] Renal TB is often clinically silent in its early stages, marked only by sterile pyuria and microscopic hematuria. Dysuria, flank pain, abdominal pain and gross hematuria develop as the disease progresses. Hydronephrosis or ureteral stricture may complicate the disease as noted in our case.

Renal TB occurs as a result of hematogenous spread following a primary infection in the lung. At the time of presentation, there is frequently no evidence of active pulmonary disease. However there may be clinical or radiographic evidence of past infections suggesting renal involvement has reactivated after a period of dormancy.[1] But as in our case, there was no clinical or radiological evidence of past infection of tuberculosis of lung, fallopian tubes or gastrointestinal tract, so we labeled this as classical(?primary) renal tuberculosis.

The lack of history of previous TB in our patient may be explained by lack of screening and health care in our population, possibly resulting in previous TB going unnoticed and undiagnosed.

In employing the term Primary renal tuberculosis, one refers to a state of primarity of the infection, only in respect to the site of its appearance in the urogenital system. [8] These findings were noted in our case. Tuberculosis of the urinary tract is easily overlooked. Many patients present with lower urinary tract symptoms typical of 'conventional' bacterial cystitis and suspicious of tuberculosis are aroused only when there is no response to the usual antibacterial agents or when urine reveals pyuria in the absence of a positive culture on routine media. [4] Other symptoms that sometimes occur include flank, back or suprapubic pain, hematuria, frequency and nocturia. These might also suggest conventional bacterial urinary tract infection. Renal colic is uncommon, occurring in fewer than 10 % of patients and constitutional symptoms like fever, weight loss and night sweats are unusual. [4] Most of the times, renal tuberculosis is either ignored or missed due to above mentioned findings.

Renal TB tends to present in an indolent fashion. Advanced tissue destruction may occur long before the diagnosis is made. [1] Ultimately a tuberculous kidney may calcify, rupture into adjacent calyx or form a cavity that communicates with the collecting systems.

[3] Hydronephrosis, stricture due to fibrosis or obstruction involving infundibulum, pelvis or ureter may occur. [1, 4] An obstructed renal lobe or entire kidney may progress to significant dysfunction or end stage failure. [1, 3, 4] This was happened in our case where undiagnosed renal tuberculosis leads to hydronephrosis, obstruction and fibrosis, later on leading to significant dysfunction and end stage kidney.

Tuberculosis may involve the kidney as a part of generalized disseminated infection or as a localized genitourinary disease as in our case. The morphology of the lesions depends on the site of the infection, the virulence of the organisms and the immune status of the patient. [4] TB, although an uncommon cause of progressive renal failure is an important one because unlike many other renal conditions, it is potentially preventable and easily treatable. [4] Evidence as to the extent to which tuberculosis is a cause of end stage renal failure worldwide is limited and scanty. [4] This was noted in our case.

Light microscopy of renal TB showed epithelioid granulomas with or without caseation, leading to tissue destruction and fibrosis. Organisms can be seen in the lesions by Zeihl-Nielsen (ZN) staining but sometimes difficult to find if cell mediated immunity is good. [1]

The diagnosis of GUTB is based on combination of clinical signs and ideally on a positive ZN stained smears or urine culture or a positive biopsy. [6]

Disease related morbidity is lower today due to significant advances in treatment of tuberculosis by antitubercular drugs. [1]

CONCLUSION

The diagnosis of GUTB is often missed. Hence suspicion of GUTB must be considered if the patient has the symptoms of recurrent urinary tract infection (UTI) or hematuria associated with sterile pyuria. Renal TB is a disease with preventable complications if timely diagnosis and treatment is established. In order to avoid outcomes such as renal failure, clinicians must be aware of the signs and symptoms of GUTB while treating patients of urinary problems such as sterile pyuria and recurrent UTI.

REFERENCES

1. Chun HM, Hale B. Renal tuberculosis: Three cases and a review of the literature. Infectious Diseases in Clinical Practice. 2004; 12(2) : 117- 122.
2. Chiang LW, Jacobsen AS, Ong CL, Huang WS. Persistent sterile pyuria in children. Don't forget tuberculosis ! Singapore Medical J. 2010; 51(3): 48-50.
3. Eastwood JB, Corbishley CM, Grange JM. Tuberculosis and tubulointerstitial nephritis: an intiguing puzzle. Kidney International.2011 ; 79 (2) : 579-581
4. Eastwood JB, Corbishley CM, Grange JM. Tuberculosis and the kidney. J Am Soc Nephrol. 2001 ; 12 : 1307-1314
5. Deodhar SG. Bacterial infections: Tuberculosis In: Deodhar SG, Deodhar SS (edi). General Pathology and pathology of systems. 6th edi, Popular pub. 2001.Vol 2 : 924-926
6. Buchholz NP, Haque R, Salahuddin S. Genitourinary Tuberculosis: A profile of 55 in patients. Journal of Pakistan Medical Association (JPMA) .2000; 50(8):265-269.
7. Garcia – Rodriquez JA, Garcia Sanchez JE, Munoz Belido JL et al.Genito- urinary Tuberculosis in

Spain : Review of 81 cases. CID. 1994; 18 : 557-61
8. Herman L. Some difficulties in the diagnosis and treatment of unilateral renal tuberculosis. Ann Surg. 1919; 70(2):202-209.

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