A NON-FUNCTIONING LEFT KIDNEY FROM RENAL TUBERCULOSIS: A CASE REPORT

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ABSTRACT

This is a 25-year old petty trader with 2-year history of recurrent left flank pains, an uneventful pregnancy, a ballotable tender, irregular left kidney and a renal sonogram suggestive of chronic pyelonephritis with intra-renal abscesses. Intravenous urogram showed a non-functioning left kidney. She had a left nephrectomy with histology showing focal segmental glomerulosclerosis with extensive areas of necrosis, acute on chronic inflammation with formation of foreign body and Langerhans type giant cells suggestive of renal tuberculosis. She made an uneventful recovery with stable renal function.

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CASE PRESENTATION

Mrs. AA is a 25-year-old petty trader who was referred from a private hospital to the University College Hospital (UCH), Ibadan following complaints of recurrent left flank pains of 2 years duration. Her problem started 2 years earlier with left sided flank pains said to be dull, continuous and non- radiating. There was neither gross haematuria nor other urinary symptoms. At the time, there was a history of recurrent low grade fever. She had had several episodes of left flank pains over the past 24 months treated with antibiotics and analgesia. However, about two months prior to referral to UCH, she noticed a left-sided abdominal swelling necessitating referral. There was no history of chronic cough, night sweats nor contact with anyone with a chronic cough. She admitted to a history of weight loss. She had not been treated for tuberculosis (TB)in the past. She was neither hypertensive nor diabetic and there was no family history of renal disease. During the two-year period, she got pregnant, with an uneventful antenatal period and was delivered of a baby girl six months before presentation.

Examination showed a young lady, pale, afebrile and anicteric. She had no significant peripheral lymphadenopathy. Her abdomen was full with a swelling in the left lumbar area. The left kidney was

ballotable and measured about 8cmx6cm. It was firm, mildly tender and irregular. The liver and spleen were not enlarged. Her chest was clear on auscultation. She had a pulse rate (PR) of 88b/m, blood pressure (BP) was 120/72mmHg and her heart sounds were normal. Her neurologic examination was unremarkable.

A provisional diagnosis of a left renal tumor to rule out left chronic pyelonephritis complicating Renal Cell Carcinoma was made.

Results of her tests showed a haematocrit of 35% and a total white cell count of 4100/mm^3. She had marked leucocyturia with 20-25 pus cells per high power field, 2+ granular casts and calcium oxalate crystals. Urine culture was unremarkable. Serum electrolyte urea and creatinine values were within normal limits as were liver function tests. Her lung fields were clear on chest radiograph and mantoux test was negative. An abdominal sonogram showed a markedly enlarged left kidney that measured 16.2 x 10.1cm with well demarcated hypoechoic multiple round to oval foci (largest 5.2cm x 3.9cm) having ecogenic debris within them. There was splaying of the calyces and lobulation of the renal contour. The surrounding renal tissue was hypoechoic with loss of corticomedullary differentiation. The right kidney was enlarged but was otherwise normal. Liver were unremarkable. Features were suggestive of pyelonephritis with multiple intra-renal abscesses.

An intravenous urogram showed prompt excretion of contrast on the right but none on the left even after 24 hours. Features were in keeping with a nonfunctioning left kidney.

She was then referred to the urologists and subsequently had a left nephrectomy. The post operative period was uneventful. Her creatinine level was 0.8mg/dl (eGFR 94.5mls/min) in the immediate postoperative period. She was discharged and is being followed up.

The nephrectomy specimen weighed 650g, measured 15cmx9cmx9cm and cut section showed multiple abscess cavities containing purulent material. Histology showed focal segmental glomerulosclerosis with extensive areas of necrosis, acute on chronic inflammation with formation of foreign body and langerhans type giant cells. There were areas showing sheets of macrophages and focal aggregates of neutrophil polymorphs. Overall features were in keeping with those of renal TB with background focal segmental glomerulosclerosis as shown in figures 1 and 2 below.

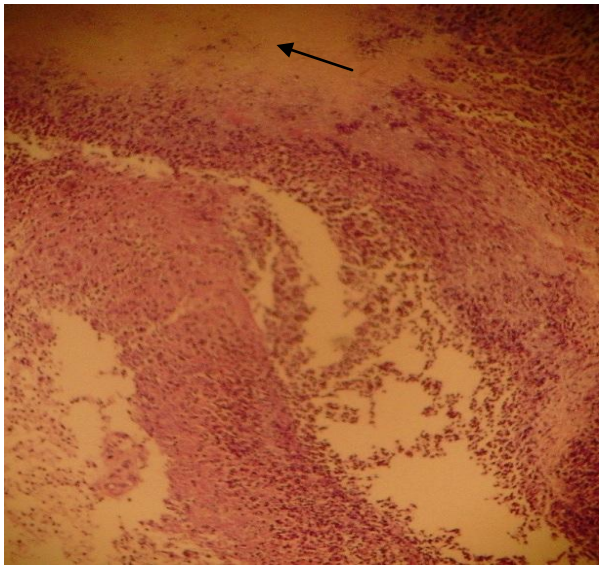

Figure 1. Section of kidney showing areas of necrosis H/E x100

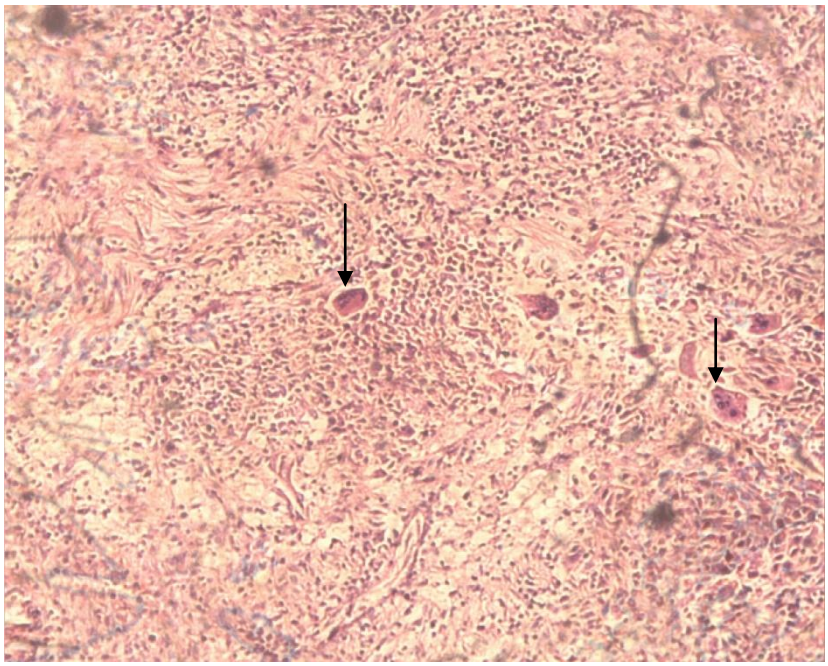

Figure2. Section of kidney showing langerhans giant cells H/E x 100

DISCUSSION

In 2012, over 8.6 million new cases of TB were reported with an estimated 1.3million deaths occurring as a result[1]. Most of these were among people aged 15 -50years in developing countries. Extra pulmonary TB (EPTB) accounts for 10% of all cases of TB while about 30-40% of these cases have urogenital involvement[2,3]. Of those with pulmonary TB (PTB), about 2-20% develop urogenital TB[4,5,6]. Urogenital TB has been reported as the second commonest form of EPTB and is more prevalent in low income countries with high burden of TB[7]. In Nigeria, renal TB has been reported in 16.3% of patients with urogenital TB although hospital based retrospective studies have shown a significantly lower prevalence rate of 1-3% [8,9]. The mean age at diagnosis in a recent review was 40.7years with a range of 5-90 years[1]. In one of the Nigerian series, males were twice more likely to be

affected than females[9]. Although the patient being reviewed was female, she presented at age 25 years which is within the age range.

Renal TB is an insidious condition with late onset symptoms, late treatment, a high organ destruction rate and therefore an increased incidence of renal failure[10]. The reasons identified for delayed diagnosis include the insidious onset, paucity of symptoms of kidney disease, lack of awareness by physicians and poor care seeking behavior.[11] The average latent period between PTB and clinical renal TB is 22 years (range 1-46 years)[12]. Our index patient had been ill for two years during which time she had an uneventful pregnancy. Although she was seen by several general practitioners, the diagnosis was only made upon referral to our hospital at which time the left kidney had become non functioning as demonstrated by findings of the intravenous urogram. Obviously, this delay may also be explained by the lack of symptoms directly referable to the urogenital system. She had recurrent left flank pains but there were no lower urinary tract symptoms such as dysuria, frequency and nocturia which would have alerted her general practitioners to the possibility of a smoldering urinary tract infection. Repeated infections eventually destroyed much of the kidney with eventual complete loss of function, the stage at which she presented.

A recent comprehensive review has shown that about 26.9% of cases present with unilateral non-functioning kidneys while 7.4% present with renal failure[2]. The prevalence of non-functioning kidneys however varied from 8.0% in the USA to 71.7% in Tunisia[5,13].This may reflect late presentation in a developing country on the one hand and sophistication in laboratory evaluation on the other. Our patient did not present with renal failure because the disease was unilateral allowing the otherkidneyto undergo compensatory enlargement. Renal failure usually results from bilateral generalized parenchymatous destruction, bladder/ureteric involvement with urinary tract obstruction and marked tuberculous interstitial nephritis[14,15], conditions that were absent in this patient.

The clinical features are also variable. Constitutional symptoms such as malaise, fever, night sweats and weight loss are unusual in these patients. Only half of all patients with renal TB are symptomatic at presentation while just about a third of patients have a previous history of PTB[16]. Perhaps our patient had been exposed years ago with the infection becoming reactivated several years later. It is difficult to ascertain the risk factor here in the face of a negative screening test for HIV infection, the absence of diabetes mellitus and malignancy or use of immunosuppressive medications. However, it is documented that only 36.6% of patients have a previous diagnosis of TB or radiologic evidence at time of diagnosis[2].

Current recommendations are that patients presenting with lower urinary tract symptoms with sterile urine cultures and those who fail to respond to conventional antibiotics be evaluated for renal TB[13]. Although our index patient did not have florid lower urinary tract symptoms, she had a sterile pyuria. However, cultures of mycobacteria TB from urine could not be done due to lack of facilities at that time.

Granulomas in the kidneys usually result from implantation of tubercle bacilli in the kidney during haematogenous dissemination following primary infection. They usually appear near the glomeruli where there is high blood flow and relatively high oxygen tension. Cortical granulomas then remain dormant for years till there is a breakdown in host immunity whereupon they proliferate. The inflammatory process is highly destructive

and is characterized by extensive necrosis and fibrosis seen histologically in figure 1. Cortical granulomas may then enlarge, coalesce and frequently cavitate with formation of ulcero cavernous lesions[14]. This may have been responsible for the multicystic abscess containing cavities seen grossly in the nephrectomy specimen of the patient our index patient. Classical epitheloid granulomas were not seen in this patient perhaps owing to an unexplained blunted immune response. However, the presence of foreign body and langherhans giant cells on a background of extensive necrosis is in keeping with tuberculosis.

Treatment entails anti tuberculosis chemotherapy (Isoniazid, Rifampicin, Ethambutol and pyrazinamide) for a period ranging from 12 to 18 months. Surgical intervention is imperative where complications like obstruction and caseating destruction of the kidneys have occurred[17]. In our case, the patient had nephrectomy, a procedure that has been recommended for nonfunctioning or poorly functioning unilateral tuberculous kidney[10,18].

A very close differential diagnosis here is xanthogranulomatous pyelonephritis. This condition also presents with fever, malaise, loin pains and weight loss with palpable renal mass and multiple abscesses. However, it is commoner in elderly women and histology typically shows xanthoma cells (large foamy, lipid-containing macrophages) in addition to neutrophils, plasma cells and foreign body giant cells.[16]

CONCLUSION

In conclusion, this case highlights two important points.Firstly, the clinical symptomatology of renal tuberculosis has not changed much largely due to late presentation by patients. Secondly, there is need to often investigate patients with sterile pyuria for renal tuberculosis.

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