RAPIDLY PROGRESSIVE RENAL FAILURE - UNUSUAL PRESENTATION OF RENAL TUBERCULOSIS (A Case Report)

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Introduction

Genitourinary tuberculosis (GUTB) is the commonest form of extrapulmonary tuberculosis [1]. It usually results from silent bacillaemia and two thirds of the patients have evidence of present or old pulmonary tuberculosis [2].

Haematogenous seeding of *Mycobacterium tuberculosis* (MTB) occurs in the renal cortex, but clinically important disease starts in the renal medulla with formation of granulomas, caseation, papillary necrosis and widespread destruction. Severe damage is characterised by putty kidney which is a combination of obstructive and necrotising processes. Despite bilateral haematogenous seeding of the kidneys, clinically significant disease is usually unilateral [2,3]. Renal failure is a rare form of presentation [4].

Case Report

A 76-year-old man presented to a peripheral hospital in May 1995 with symptoms of nocturia, backache and bleeding per rectum of 3 months duration. He was operated for internal haemorrhoids. In the post operative period he developed urinary retention for which he was catheterised. Urine culture grew *E. coli* and he was given a course of ciprofloxacin for 10 days. Blood urea was 32 mg/dL and serum creatinine was 0.8 mg/dL at this stage. Two months later he took a surgical consultation for persisting symptoms of backache, nocturia and frequency of micturition. Evaluation then revealed grade II prostatomegaly. Prostatic biopsy ruled out a prostatic carcinoma. The patient however was found to have biochemical evidence of renal failure and hence referred to our centre.

On admission to our centre in July 1995, he was found to be emaciated, pale, ill looking and febrile. There was no clubbing or lymphadenopathy. His pulse was 86/min and blood pressure

was 90/60 mmHg. He had bilateral crepitations, left lung > right lung. Other systemic examination was within normal limits. The haemoglobin was 12.5 g/dL, total leucocyte count was 7100/cumm with 60 per cent polymorphs, 37 per cent lymphocytes, 1 per cent monocytes and 2 per cent eosinophils. Erythrocyte sedimentation rate showed 70 mm fall in 1st hour. Urinalysis revealed 1+ protein, 6-8 WBCs and 3-4 RBCs per high power field. Urine and blood cultures were sterile. Blood sugar levels were 96 mg/dL and serum creatinine ranged from 4.3 to 9.0 mg/dL. Chest radiogram PA view was normal.

The patient was managed as a case of septicaemia with acute renal failure with cefotaxime, cloxacillin and amikacin in modified doses along with dialysis support. Repeat ultrasonography of the kidneys 2 weeks after admission showed multiple abscesses in both the kidneys. CT scan confirmed bilateral renal abscesses and in addition revealed bilateral psoas abscesses and destruction of first lumbar vertebra (Fig 1). Radiogram of thoracolumbar spine also showed destruction of first lumbar vertebra (Fig 2). Ultrasound guided pus aspiration was done from left kidney and psoas abscess. Pus smear was negative for acid fast bacilli and the culture was sterile. Repeat chest radiogram revealed bilateral non homogenous opacities suggestive of

Fig. 1: CT scan showing bilateral renal abscesses and psoas abscesses and destruction of first lumbar vertebra.

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Fig 2: Radiogram of thoracolumbar spine showing destruction of first lumbar vertebra

pulmonary tuberculosis.

The patient was managed as a case of disseminated tuberculosis with anti-tubercular therapy (ATT) comprising of rifampicin, isonex, pyrazinamide and ethambutol in modified doses. However, patient's condition progressively deteriorated and he died 2 weeks after ATT was started. Postmortem study was not done.

Discussion

Renal tuberculosis is usually a slowly developing infection and 20 per cent of the patients are usually asymptomatic when the disease is diagnosed [3]. Lattimer observed that 18 out of 25 patients with renal tuberculosis were diagnosed when the disease was far advanced [5].

Symptomatic patients of GUTB present with dysuria (34%), haematuria (27%) and flank pain (10%). Constitutional symptoms are observed in a minority. Sterile acid pyuria is usually a constant feature of GUTB [1], although 20 per cent patients may have superinfection with *E. coli* at some point of time [6]. MTB can be isolated in 90 per cent of patients from urine [7] if culture is done properly.

Since bacilluria is episodic, multiple cultures (up to 12) are recommended [1]. For MTB culture, early morning samples have been found to be superior to 24 hour collection [4,7].

Besides evidence of pulmonary tuberculosis in 50-75 per cent cases of GUTB, 10 per cent of these patients have evidence of spinalmesenteric involvement [3]. Intravenous pyelography remains the cornerstone of diagnosis and gives a clue in 93 per cent of cases [7]. The findings range from irregularity or amputation of calyces, to obstruction and later total destruction of kidney. Calcification is seen in 50 per cent of the cases [4].

Treatment is with conventional ATT, 4 drugs for initial 2 months followed by 2 drugs for next 4 months. If kidney is non-functional and severely infected, nephrectomy may be offered. Usual cause of renal failure in these patients is obstruction and destruction of the kidney, however rarely interstitial nephritis can result in renal failure [6].

Renal failure due to renal tuberculosis is rare and when it occurs, it is slowly progressive. To the best of our knowledge, rapid development of bilateral renal disease with multiple abscesses and progression to dialysis dependency within 2 months has not been reported. In conclusion, renal tuberculosis can have a devastating course and can present as rapidly progressive renal failure.

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