Case Report

Acute renal failure of unknown origin. Don’t forget renal tuberculosis

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Introduction

Renal tuberculosis is not mentioned in the current literature among the various causes of acute renal failure (ARF) [1,2]. We report for the first time that obstructive ARF may be caused by tuberculosis and reversed by medical therapy.

Case report

A 46-year-old woman was admitted with epigastric pain and dyspepsia. She reported symptoms of gastritis, occasionally treated with anti-H2 drugs. Past medical history included insulin-dependent diabetes mellitus for about 20 years associated with marked diabetic retinopathy, slight reduction of renal function (plasma creatinine level of 106 µmol/l, creatinine clearance of 69 ml/min), proteinuria less than 1 g/day, moderate erythrocyturia and leukocycturia. On admission, no other symptom was detected and gastroprotective drugs relieved the gastric pain. The biochemical assessment showed increased plasma creatinine (177 µmol/l) with a creatinine clearance of 28 ml/min, urea (11.5 mmol/l), glycaemia (5.5 mmol/l), and diuresis (900 ml/day), with normal blood pressure (130/80 mmHg). Urinalysis revealed erythrocyturia (10 cells per high-power field) and leukocycturia (10 cells per high-power field) with a pH of 6.0 and a specific gravity of 1.015. An increase in erythrocyte sedimentation rate (56 at the 1st hour) was associated with a moderate degree of anaemia (Hct = 35%). All other examinations including white blood-cell count and chest radiography were within normal limits.

Prerenal and toxic causes of renal failure were excluded by careful anamnesis and clinical and laboratory assessment. To investigate postrenal causes of impaired renal function, the patient underwent sequential radiographic examinations (echography, urography, and computerized tomography). These examinations revealed a right kidney of increased size with gross cavities in which the renal parenchyma was reduced to a thin shell; a moderate dilatation of the collecting system was demonstrated in the left kidney. After contrast media injection, the right kidney was not visualized, while the left kidney revealed pyelocaliectasis secondary to the presence of a stricture at the level of the pyeloureteral junction, which was associated with a moderately restricted left ureter (Figure 1). There were no genitourinary calcifications. The right kidney was removed with no change in the renal function. The histopathological examination led to the diagnosis of tuberculous pyonephrosis. We started treatment with isoniazid (300 mg/day), rifampicin (600 mg/day), and ethambutol (400 mg/day). Six months of this therapy progressively reduced the values of plasma creatinine and urea levels to 106 µmol/l and 6.5 mmol/l respectively, associated with improvement of creatinine clearance (71 ml/min) and urinary abnormalities (5 red blood cells and 5 white blood cells per high-power field). The renal echography demonstrated complete remission of the obstruction.

Comment

This case depicts renal tuberculosis (TB) as a cause of obstructive ARF. In our patient the lack of a previous diagnosis of renal TB was probably due to the absence of urinary and systemic symptoms of TB and to the urinary and renal function abnormalities that could be attributed to the long-term diabetic nephropathy. Indeed, more insights were attained only after the incidental discovery of ARF. The unilateral ureteral stricture, which was probably secondary to the presence of mucosal tuberculous nodules at the level of the ureteropelvic junction, and the combined absence of contralateral renal function, accounted for the development of ARF. The short-term antituberculous therapy led to the remission of ARF. Notably, no previous reports of similar cases appear in the current literature. The clinical manifestations of renal TB are generally unilateral and involve about 3% of all TB patients [3]. Bilateral advanced renal TB, similar to the case presented, is considered as a usual cause of chronic renal failure [4], and it is not even mentioned among the various causes of ARF [1,2]. It is important to underline that TB is a re-emergent infection. Indeed,
it is predicted that there will be 90 million new cases of TB by the end of this decade [5]. In conclusion, renal TB is a possible cause of obstructive ARF, which can be reversed with pharmacological intervention.

References


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