Acute Renal Failure Secondary to Acute Bacterial Pyelonephritis

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Dear Sir,

Acute bacterial pyelonephritis is rarely a cause of acute renal failure. We report a case of nonobstructive acute pyelonephritis with associated acute renal failure.

A 66-year-old female was admitted to our hospital with a 10-day history of fever, chills, nausea, vomiting, back pain, frequency, dys-uria and decreased urinary output. The patient had a history of mild untreated hypertension, and 1 month prior to admission serum creatinine was 132.6 $\mu\text{mol/l}$. On admission, blood pressure was 114/66 mm/Hg, pulse was 104 beats/min, and oral temperature was 38.6°C. Aside from diffuse abdominal and bilateral costovertebral tenderness, there were no other significant findings. Her urinary output was 35 ml during the first 12 h after her admission. The initial laboratory values were as follows: hemoglobin, 13.0 g/l; hematocrit 38.0%, WBC 22,600 mm^3 ; platelets, 224,000 mm^3 ; prothrombin time 15.2 s, partial thromboplastin time 38.6 s, sodium 129 mmol/l; potassium 4.5 mmol/l; chloride 88 mmol/l; bicarbonate 22 mmol/l; urea nitrogen 31.4 mmol/l; creatinine 627.6 $\mu\text{mol/l}$; glucose 4.9 mmol/l; calcium 2.12 mmol/l; phosphorus 1.7 mmol/l; LDH 470 U/l; CPK 14 U/l; SGOT 123 U/l; SGPT 96 U/l; alkaline phosphatase 136 U/l; total bilirubin 35.9 $\mu\text{mol/l}$; total protein 48 g/l and albumin 21 g/l. Examination of urinary sediment showed white and red blood cells (more than 50/high power field) and numerous bacteria. No eosinophils were found. After admission, aggressive hydration was begun and empiric treatment with ticarcillin-sulbactam for possible bacterial pyelonephritis was started. Renal ultrasound failed to show any obstruction. Other laboratory findings: ASO titers, ANA, cryoglobulins and hepatitis B antigen and antibody were negative; C3, C4 and total complement levels were within the normal range. The urine and blood cultures were positive for *Klebsiella pneumoniae*. A 24-hour urine collection showed 974 mg of protein in 300 ml of urine. A CT scan of the abdomen and pelvis, done the day after admission without contrast, demonstrated increased size of the kidneys bilaterally (fig. 1). Because of persistent oliguria and no improvement in renal function, the possibility of an associated rapid progressive glomerulonephritis was entertained. On the 4th hospital day, a closed biopsy of the right kidney was performed. Microscopic sections showed severe diffuse tubulo-interstitial inflammatory changes. The tubules were distended with acute inflammatory

exudate destroying the epithelium. The inflammation extended into the interstitium producing focal microabscesses. The biopsy did not contain any glomeruli, and there was no evidence of any vasculitis (fig. 2).

The patient had a gradual recovery after 6 weeks of intravenous antibiotic therapy. At the end of that period of time, serum urea nitrogen (BUN) was 16.4 mmol/L, serum creatinine was 300.6 $\mu\text{mol/L}$, urinalysis did not show any WBC, RBC or proteinuria, and the urine culture was negative. Three months later, her BUN was 16.0 mmol/L, and her serum creatinine was 274 $\mu\text{mol/L}$. Again urinalysis was completely negative. An intravenous pyelogram performed at this time showed focal parenchymal scarring and caliceal clubbing in the right kidney, and atrophic changes in the left kidney (fig. 3). A CT scan of the

Fig. 1. Representative frame of the initial CT scan without contrast showing bilaterally enlarged kidneys.

abdomen with oral contrast was also obtained showing bilateral renal atrophy, worse in the left; the ureters and the bladder did not show any abnormality (fig. 4).

The literature contains only a few reported cases of acute renal failure secondary to acute bacterial pyelonephritis in which the diagnosis was supported by histology [1-4].

Our patient presented with a typical history of acute pyelonephritis. However, the cause of her acute renal failure was clinically obscure. She had no prior history of renal disease and was not taking any nephrotoxic medications. There was no evidence of volume depletion and no improvement after hydration. The ultrasound of the kidneys did not show hydronephrosis. It is possible that some underlying nephropathy may have been present prior to her hospitalization, judging from the elevated baseline creatinine of

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Fig. 3. Intravenous pyelography showing severe contraction of the left kidney with marked cortical scarring.

Fig. 2. Renal biopsy showing infiltration of polymorphonuclear leukocytes in the interstitium and in the tubules and a microabscess. HE.

132.6 $\mu\text{mol/L}$, probably related to the history of hypertension. Unfortunately, the renal biopsy did not show any glomeruli, but the predominance of the acute inflammatory infiltrate in the tubules is more in favor of acute pyelonephritis than the interstitial changes that have been described in association with crescentic glomerulonephritis [5]. The enlarged kidneys during the acute phase and the later marked shrinkage in the immediate recovery phase supports the diagnosis of acute pyelonephritis as the cause of the rapid deterioration of the renal function [6,7].

In summary, although a rare cause of acute renal failure this case demonstrates that acute pyelonephritis should also be considered in the differential diagnosis since early antibiotic treatment may result in recovery of the renal function.

Fig. 4. CT scan showing a marked reduction in the size of the left kidney.

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