Acute Interstitial Nephritis with Septicemia and Erythromycin

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Dear Sir,

Acute interstitial nephritis (AIN) is characterized by the association of acute renal failure (i.e., abrupt clinical onset) and infiltration of the interstitium by inflammatory cells [1]. It is currently stated that the drug-induced form predominates in the list of causes of AIN [1-3]. However, there is no clear information on the prevalence of infectious causes of AIN. It may be difficult to identify the mechanisms involved in patients with systemic infection who receive antibiotics and, therefore, are exposed to the risk of antibiotic-induced AIN [4]. We describe a patient with Echerichia coli septicemia who developed acute renal failure during antibiotic therapy with erythromycin.

A 57-year-old lady without any medical history developed a flu-like syndrome characterized by general malaise, fever and ENT symptoms. 3 days after the prescription of erythromycin, a toxicodermy with an annular erythema was noted and the drug was withheld. She remained ill and febrile and was admitted 2 days later. Physical examination disclosed a moderately ill and obese woman; her temperature was 37.4°C, pulse 110 beats/min, blood pressure 170/90 mm Hg. Apart from the toxicodermy and the painless palpable enlarged kidneys, no other abnormalities were noticed. Laboratory investigation revealed (nonoliguric) renal failure (ureum 96 mmol/l; creatinine 1,360 µmol/l), leucocytosis with eosinophilia (29.8 and 0.76/nl, respectively, leucocyturia (no casts or protein-uria) and hepatic damage (aspartate amino-transferase 35 U/l; alanine aminotransferase 59 U/l). Serological examination (including anti-TBM) was negative. E. coli grew out of 3 blood cultures and urine culture. Renal echo-graphy showed bilaterally enlarged kidneys, the left kidney measuring 12 cm and the right kidney 15 cm, without signs of obstruction. She was treated with tobramycin and dialysis. A skin biopsy revealed perivascular dermatitis with eosinophilia (29.8 and 0.76/nl, respectively, leucocyturia (no casts or protein-uria) and hepatic damage (aspartate amino-transferase 35 U/l; alanine aminotransferase 59 U/l). Serological examination (including anti-TBM) was negative. E. coli grew out of 3 blood cultures and urine culture. Renal echo-graphy showed bilaterally enlarged kidneys, the left kidney measuring 12 cm and the right kidney 15 cm, without signs of obstruction. She was treated with tobramycin and dialysis. A skin biopsy revealed perivascular dermatitis with eosinophil granulocytes. A percutaneous renal biopsy was compatible with a diagnosis of AIN without microabscesses or any involvement of the normal glomeruli. Analysis of the renal cell infiltrate disclosed massive amounts of mononuclear cells including more than 10% eosinophil granulocytes (fig. 1). Upon knowledge of the renal biopsy prednisone was added; renal function recovered after 2.5 weeks, and the patients was discharged with a serum creatinine of 180 µmol/l.

Excluding typical cases, it is difficult to differentiate drug-induced AIN from other types such as E. coli bacteremia. Drug-induced multisystem involvement is rare in patients; its presence in our patient (cutaneous reaction, eosinophilia and hepatic damage) and the renal cell...
infiltrate with many eosinophils are strong pointers to erythromycin as a probable culprit. Up to the time of this study erythromycin-induced AIN has only been established and biopsy proven unequivocally once [5].

Since the drug has regained a revival being the drug of choice in atypical pneumonia and other conditions like gastric paresis, nephrologists should be aware of AIN as serious side effect.

Fig. 1. Renal biopsy: paragon staining showing the presence of interstitial and tubular inflammatory infiltrate. GMA plastic. ×250.

References


