Acute Renal Failure Caused by Two Single Doses of Rifampicin with a Year of Interval

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Dear Sir,

Numerous side effects have been reported in rifampicin-treated patients. Renal effects of such treatment are not common and may manifest themselves clinically in different ways [1-3], with acute renal failure (ARF) being the most frequent manifestation. In most instances, this complication is encountered in patients treated with an intermittent regimen or discontinuously (resumption of therapy after a medication-free period), but there are also cases associated with continuous daily therapy. When treatment is discontinuous, the medication-free period and dose involved tend to vary, but a prior prolonged treatment with rifampicin for tuberculosis has been reported in every case [4-6]. We describe a severe case of ARF caused by the ingestion of 2 minimal doses of rifampicin separated by the interval of an entire year, not related with antituberculous treatment.

A 50-year-old man was admitted for diffuse abdominal pain, vomiting and oliguria of 4-day duration. A few hours before onset of symptoms, he had taken of his own accord a dose of 300 mg of rifampicin for influenzalike symptoms. One year previously, rifampicin had been prescribed for urethritis, but the patient had only taken a single dose of 600 mg. He did not refer to prior treatment for tuberculosis. On admission, the patient's blood pressure was normal, his temperature was 37.8 °C and his pulse rate was 80/min. He presented with diffuse abdominal pain without signs of peritonitis. Laboratory data included hematocrit 36.6%, WBC count 12,500/μl with no eosinophils, and platelet count 80,000/μl. No schistocytes were observed and Coombs's test was negative. Coagulation was normal. Serum creatinine was 11.9 mg/dl (1,052 μmol/l), BUN 133 mg/dl (47.4 mmol/l), uric acid 14 mg/dl (0.83 mmol/l), SGOT 36 U/l, SGPT 64 U/l, γGT 88 U/l, LDH was 1,231 U/l, and glucose, serum ions, bilirubin and alkaline phosphatase were within the normal range. Immunoelectrophoresis was normal except for IgE of 149 U/ml (normal < 120); tests for complement, antinuclear antibodies, antineutrophil cytoplasmic auto-antibodies and crioglobulins were negative. HBsAg was negative. During the first 24 h, 320 cm³ of urine were excreted. Urinalysis showed proteinuria of 2.36 g/l, more than 100 RBC/hpf and 7-8 WBC/hpf, with no eosinophils. Renal ultrasound was normal. A percutaneous renal biopsy showed severe interstitial edema with lymphocytic inflammatory infiltrate and focal detachment of tubular epithelium with some mitosis, compatible with acute interstitial

nephritis and tubular necrosis. No evidence of glomerular and/or vascular pathology was found. The patient required 4 sessions of hemodialysis, but recovered normal renal function after 4 weeks.

Although a test for rifampicin-specific antibodies could not be performed, the clinical, analytical and histological features (abdominal pain, oliguric renal failure, low-grade fever, slightly depressed platelet count and slightly elevated hepatic enzymes) were consistent with rifampicin-induced renal failure [5]. To date, no cases of ARF secondary to rifampicin treatment for other reasons than tuberculosis have been reported. The extremely low doses and the long interval between them lend support to the hypothesis that a hypersensitivity mechanism is involved

in the pathogenesis of ARF, even though the histological expression can be identical to that of acute tubular necrosis in some cases [6], but the diagnoses can be difficult because ingestion of the drug can go unnoticed.

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