20 mg/day), and his creatinine started to increase 1 year after the drug was introduced and returned to baseline 5 months after LFM was withdrawn (positive dechallenge). This, and the previous safety record for LFM, suggests that the interstitial nephritis was related to the chronic overdosage which persisted for many months especially as LFM has a very long half-life lasting for 15 days. Furthermore, the time course of the deterioration in renal function was very long, which argues against an idiosyncratic response.

The patient had uncontrolled hypertension which was the cause of his initial elevated creatinine. In addition, the renal biopsy showed mainly interstitial fibrosis and minimal glomerulosclerosis which is a typical lesion in hypertension-induced renal dysfunction.

Steroids are not proven in uncontrolled trials to be efficacious in the treatment of interstitial nephritis. The evidence for their benefit comes from two small trials where they induced quicker recovery, lower creatinine on follow-up, and less interstitial fibrosis in repeat renal biopsy [6,7].

This case emphasizes the importance of physicians supervising the treatment of patients having a precise knowledge of the type and dosage of drugs their patients are actually taking.

Conflict of interest statement. None declared.


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Ethambutol-induced acute renal failure

Sir,

Ethambutol can cause ocular toxicity and hyperuricaemia, but ethambutol-associated acute renal failure during treatment of pulmonary tuberculosis is extremely rare [1,2]. Here we report the case of a man who manifested with oliguric acute renal failure and jaundice associated with ethambutol.

Case. A 33-year-old man was referred to our university hospital from a local clinic because of oliguria for the previous 2 days. Five years previously he had been treated for pulmonary tuberculosis with the antituberculosis medications rifampicin, pyrazinamide, isoniazid and ethambutol. Three
days prior to admission, he was diagnosed as having a recurrence of pulmonary tuberculosis and was started on the same four medications at a local clinic. One day after the initiation of this treatment, he noted jaundice, abdominal pain and oliguria. Admission laboratory data were as follows: blood urea nitrogen, 71.6 mg/dl (25.5 mmol/l); creatinine, 5.2 mg/dl (459 µmol/l); albumin, 4.0 g/dl (40 g/l); total bilirubin, 14.0 mg/dl (238 mmol/l); direct bilirubin, 9.8 mg/dl (166 mmol/l); aspartate aminotransferase, 170 IU/l; alanine aminotransferase, 28 IU/l; urine pH 7.0, specific gravity 1.010, proteinuria 3þ; and the urine sediment contained many red cells and 3–5 white cells per high-power field. Sputum stained positive for acid-fast bacilli. All four antituberculosis medications were stopped on the day of admission.

Anuria persisted for 4 days. On the 8th and 15th day after initiation of antituberculosis medication, isoniazid and rifampicin were reintroduced without development of anuria or other intolerance reactions. On the 24th day after initiation of antituberculosis medication, ethambutol was reintroduced, and abdominal pain, fever and general weakness occurred on the same evening. The serum creatinine level increased to 8.6 mg/dl and urine output decreased; therefore, haemodialysis was initiated (Figure 1). A renal biopsy performed on the 39th day after initiation of antituberculosis medication showed features characteristic of acute tubular necrosis that were more prominent than interstitial infiltration. The patient completely recovered his renal function after receiving isoniazid, rifampicin, pyrazinamide and cycloserine.

Comment. Ethambutol-induced acute renal failure is very rare; we know of only three reported cases of tubulointerstitial nephritis due to ethambutol administration [1,2]. In those cases, the renal function deteriorated only after administration of antituberculosis medication over several months, and liver function tests were normal. The clinical characteristic findings of ethambutol-induced acute renal failure in the present case were sudden onset of oliguria and renal failure, association with hepatotoxicity, and development after re-exposure to ethambutol. We observed six cases of dobutamine-induced myoclonia in four women and two men (68.6±6.35 years old) hospitalized for congestive cardiac failure responsible for severe renal insufficiency (creatinine clearance: 15.5±2.5 ml/min/1.73 m²). Mean arterial pressure was 62±5 mmHg. Serum ASAT and ALAT were increased by 25% above normal range and total bilirubin and alkaline phosphatases were normal. The patients had moderate hypotraemia (127±3 mmol/l). Plasma potassium was normal, mean plasma total calcium was decreased (2.17±0.05 mmol/l) and serum magnesium was slightly increased (1.3±0.1 mmol/l). All patients had metabolic acidosis, with

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Department of Internal Medicine
Soon Hyo Kwon
Soonchunhyang University
Cheonan Hospital
Cheonan
Korea
Email: eylee@sch.ac.kr


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**Dobutamine-induced myoclonia in severe renal failure**

Sir,

Dobutamine is a catecholamine with β1-, β2- and α1-adrenergic properties. This drug is marketed as a racemic mixture containing two stereoisomers having different distribution and elimination properties. Pharmacological activity also differs: the L-enantiomer is a potent α1-agonist whereas the D-enantiomer is a potent β1-agonist [1]. Dobutamine is widely used in the treatment of severe heart failure. Tachycardia is the most common effect. To our knowledge, no neuromuscular disturbances with dobutamine administration have been reported.

We observed six cases of dobutamine-induced myoclonia in four women and two men (68.6±6.35 years old) hospitalized for congestive cardiac failure responsible for severe renal insufficiency (creatinine clearance: 15.5±2.5 ml/min/1.73 m²). Mean arterial pressure was 62±5 mmHg. Serum ASAT and ALAT were increased by 25% above normal range and total bilirubin and alkaline phosphatases were normal. The patients had moderate hypotraemia (127±3 mmol/l). Plasma potassium was normal, mean plasma total calcium was decreased (2.17±0.05 mmol/l) and serum magnesium was slightly increased (1.3±0.1 mmol/l). All patients had metabolic acidosis, with