Correspondence

Piroxicam-induced acute renal failure (anuria)

Sir, We report a case of piroxicam-induced acute renal failure (anuria) (ARFA) of a patient with pre-existing moderate renal failure. Although acute renal failure accompanies failure discovered not has been reported with piroxicam.

A 75-year-old man with pre-existing moderate renal failure discovered in 1980 had intermittently been on methyldopa and frusemide for hypertension since 1975. He discontinued both drugs three weeks before his admission to the hospital. He had a cerebrovascular episode without residua in 1981. He also had congenital subluxation of his hips. Osteoarthrosis secondary to this presented in 1979, for which he had intermittently been treated with paracetamol and sulindac. Initial laboratory data included: blood urea nitrogen: 80–95 mg/dl (57–68 mmol/l), serum creatinine: 457 mmol/l, creatinine clearance: 27.8 ml/min, urine specific gravity: 1003, serum sodium: 7.5 mg/dl (1.9 mmol/l), serum phosphorus: 4 mg/dl (1.3 mmol/l), serum potassium: 3.9 mEq/l (3.9 mmol/l), plasma renin activity: 3.26 ng/ml/h. ECG and chest x-rays were normal. Urine cultures were negative twice. Hydration was instituted and the 24-hour urine volume increased during the first three days, but thereafter the 24-hour urine volume increased progressively and reached the preanuria levels within four days. Computerised tomography of the abdomen detected no abnormalities of the kidneys, ureters, or bladder, nor any abdominal masses. There was no alteration in blood pressure. There was no paraproteinemia, collagen disease, use of other drugs, or administration of contrast material. During the anuria the markers of renal function were significantly impaired in comparison with the preanuria state, but returned to the preanuria levels after the restoration of the 24-hour urine volume above 1000 ml/24 h. The anuria appeared to be attributable to the uncontrolled ingestion of piroxicam (30 mg daily) by the patient himself for relief of his hip pain for the 10 days before the development of anuria.

A strong association exists between piroxicam ingestion and the development of acute renal failure.1 Vasconstriction due to inhibition of vasodilator prostaglandin E2 synthesis by NSAID in patients with pre-existing renal failure appears to precipitate acute renal failure (anuria). Other risk factors2 (hypereninaemia and hypoponatremia, as in our patient) combined with the inhibition of prostaglandin synthesis may in addition precipitate acute renal failure (anuria). Despite the lack of a diagnostic rechallenge for ethical reasons, the many similarities between this patient and those reported with other NSAID3 4 and the existence of the above mentioned risk factors suggest that piroxicam caused this patient’s acute renal failure and anuria.

References


Unusual case of childhood dermatomyositis

Sir, We read with interest the letter by Foley and Payne, describing an unusual case of dermatomyositis characterised by spontaneous remissions and recurrences separated by long periods of well-being, as we observed a similar case of a 1-year-old girl with childhood dermatomyositis. The child presented with a 1-month history of proximal muscle weakness, fever, and rash on the neck and extremities. Laboratory investigations revealed an elevated creatine kinase level and a positive antinuclear antibody. The rash resolved spontaneously after several months, but the muscle weakness persisted. Further investigations, including muscle biopsy, showed evidence of muscle inflammation and inflammation of the subcutaneous tissue. The child was treated with prednisolone, and the muscle weakness improved. The child was followed up regularly, and episodes of muscle weakness were observed, each lasting several months, followed by spontaneous remission. The child is currently well, and the muscle weakness has resolved spontaneously without any treatment. This case highlights the spontaneous nature of childhood dermatomyositis and the importance of monitoring and managing the condition closely.