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 the use of other non-steroidal anti-inflammatory drugs (NSAID), to our knowledge ARFA has not been reported with piroxicam.

A 75-year-old man with pre-existing moderate renal failure discovered in 1980 had intermittently been on methyl dopa 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. Osteoarthritis 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 calcium: 7.5 mg/dl (1.9 mmol/l), serum phosphorus: 4 mg/dl (1.3 mmol/l), serum sodium: 125 mEq/l (125 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 though hydration continued, the urine volume decreased and the patient became anuric on the seventh day (Fig. 1). Hydration was discontinued. The anuria (confirmed twice by bladder catheterisation) lasted for two 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 paraproteinaemia, 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 (hypernatraemia and hyponatraemia, 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 NSAID^{3,4} and the existence of the above mentioned risk factors suggest that piroxicam caused this patient's acute renal failure and anuria.

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Fig. 1 Twenty-four-hour urine output during the first 14 days of the patient's hospitalisation.

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