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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 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 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.

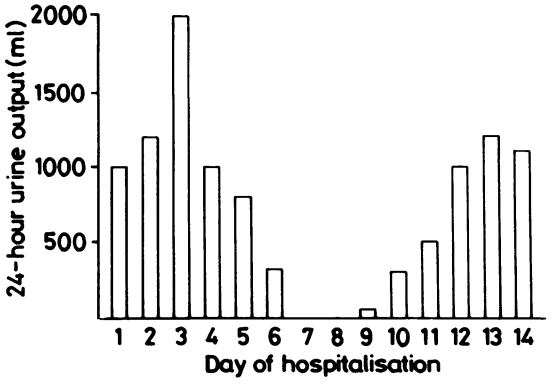

Fig. 1 *Twenty-four-hour urine output during the first 14 days of the patient's hospitalisation.*

A strong association exists between piroxicam ingestion and the development of acute renal failure.[1] Vasconstriction due to inhibition of vasodilator prostaglandin E_2 synthesis by NSAID in patients with pre-existing renal failure appears to precipitate acute renal failure (anuria). Other risk factors[2] (hypereninaemia 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.

University of Athens
Medical School,
Alexandra Hospital,
Vas. Sofias-K. Lourou Str.,
GR 115 28 Athens, Greece

MYRON E MAVRIKAKIS
KONSTANTINOS A GEORGILIS
PANOS N ZIROYANNIS
KYRIAKOS A KEHAYOGLOU
SPYRIDON D MOULOPOULOS

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Unusual case of childhood dermatomyositis

SIR, We read with interest the letter by Foley and Payne,[1] describing an unusual case of dermatomyositis characterised by spontaneous remissions and recurrences separated by long periods of well-being, as we observed a similar

picture in a case of childhood dermatomyositis. A five-year-old girl was first hospitalised in 1971 with muscular pain and weakness accompanied by mild malar erythema and an erythematous/violaceous maculopapular eruption over the extensor surfaces of the interphalangeal joints, elbows, and knees; the electromyogram (EMG) was consistent with myositis. Without treatment the patient presented spontaneous remission during the following months.

In 1975, at 9 years of age, similar symptoms affecting the skin and proximal muscle groups recurred, and a diagnosis of dermatomyositis was confirmed by increase of muscle enzymes (creatine phosphokinase 410 U/l) and typical EMG findings. Again the symptomatology disappeared without treatment.

The child led a normal life and played sports. In January 1984 the skin lesions recurred accompanied by periorbital lilac discoloration, cutaneous arteritic lesions on the medial aspect of the thighs, mild proximal muscular weakness, and moderate increase of muscle enzymes. The symptomatology resolved rapidly with a short period of low dose steroids. Generally, childhood dermatomyositis follows a uniphasic course. The activity of the disease may last from only a few months to two or three years followed by recovery;[2] during this period recurrence of the symptomatology is generally the consequence of premature discontinuation of treatment. On the contrary in our patient, as in the adult reported by Foley and Payne,[1] the clinical course of the disease was polyphasic with recurrences separated by long periods of spontaneous remissions.

Department of Pediatrics
University of Pavia,
Policlinico S. Matteo,
27100 Pavia, Italy

ALBERTO MARTINI
ANGELO RAVELLI

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The lupus anticoagulant, pulmonary thromboembolism, and fatal pulmonary hypertension

SIR, The article by Anderson and Ali[1] is an important contribution which may provide insights into the development of pulmonary hypertension in patients with systemic lupus erythematosus (SLE). While decreased synthesis/release of prostacyclin[2] or diminution of its activity[3] may be responsible for the paradoxical association of the lupus anticoagulant with thrombotic tendencies or states, other mechanisms may be significant. Relative deficiencies of protein C or antithrombin III (ATIII) have been associated with development of a thrombotic diathesis.[4 5] Depression of ATIII levels has been noted in SLE, correlating with disease activity.[6] Perhaps central to the question is the cogent observation by Anderson and Ali[1] that warfarin therapy was ineffective in controlling the thrombotic diathesis in their patient, in spite of maintenance of the prothrombin ratio in the therapeutic range. Oral anticoagulants have been generally noted to augment levels of both protein C[7] and ATIII.[8] One exception has been noted. A subgroup of individuals was identified, by Penner and Hunter,[9] in whom recurrent thrombotic phenomena were noted, even when the prothrombin time was prolonged to the normally 'acceptable' range. That group was categorised by failure of the anticoagulant to produce the expected increase in antithrombin III concentration. As ATIII deficiency-associated phenomena are responsive to ATIII transfusion,[10] identification of deficiency states would appear to be of more than academic interest. It would be of interest to study plasma[8] from such patients as described by Anderson and Ali further, to determine if such potentially treatable phenomena were present.

Rheumatology Division,
Menorah Medical Center, and
Kansas University School of Medicine,
USA.

B M ROTHSCHILD

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Bufexamac crystals in synovial fluid analysis

SIR, Bufexamac, a non-steroidal drug for intra-articular injection, was recently introduced. Its pharmacology and efficacy have been studied by several authors.[1–4] On