SHORT REPORTS

Treatment of ethylene glycol poisoning with peritoneal dialysis

Ethylene glycol is commonly available as a constituent of antifreeze solutions and is itself non-toxic. Since, however, the ingestion of over about 100 ml ethylene glycol (1·8 mol; 110 g) in an adult is potentially lethal owing to the production of toxic metabolites, treatment with an inhibitor of ethylene glycol metabolism, of which ethanol is the most convenient, should be started as soon as is practicable. Peritoneal dialysis or haemodialysis is also indicated to enhance the elimination of unchanged ethylene glycol and possibly also its metabolites, especially since renal impairment is one of the first serious manifestations of poisoning with this compound. There have been few reports[1 5] of the use of dialysis in such cases, however, and the amount of ethylene glycol recovered was calculated in only two patients, one a child treated with peritoneal dialysis[4] and one an adult treated with haemodialysis.[5] We report on three adult patients treated with peritoneal dialysis on whom full analytical data were collected.

Details of three patients poisoned with ethylene glycol and treated with peritoneal dialysis

Case No	Age (years) and sex	Approximate amount of ethylene glycol ingested (mol)	Pre-dialysis ethylene glycol plasma concentration (mmol/l)	Duration of peritoneal dialysis (hours)	Amount of ethylene glycol removed by peritoneal dialysis (mol)	Renal excretion of ethylene glycol during peritoneal dialysis (mol)
1	39 F	2·74	46·8	65	0·61	0·66
2	51 M	5·48	106·5	41	2·27	0·34
3	56 M	2·74	40·3	99	0·89	0·31

Conversion: SI to traditional units—Ethylene glycol: 1 mol/l ≈ 62·1 g/l.

Patients, methods, and results

Plasma, peritoneal dialysis fluid, and urinary ethylene glycol concentrations were measured by gas chromatography.[4] All three patients were treated with intravenous sodium bicarbonate and ethanol. Peritoneal dialysis was started as soon as possible after admission and was continued until the plasma ethylene glycol concentration fell below 1·6 mmol/l (0·1 g/l). In all cases ethanol at a concentration of 21·7-43·5 mmol/l (1-2 g/l) was added to the dialysate to prevent removal of the infused ethanol. The results are summarised in the table. Two patients (cases 1 and 3) made full recoveries. The remaining patient required a total of 2 mol (168 g) sodium bicarbonate in order to maintain the arterial pH between 7·2-7·3 and, although haemodialysis and ultrafiltration were performed to remove excess sodium, he died from multiple organ failure. One patient (case 3) had a one-month history of weight loss, polyuria, and polydipsia and required treatment with insulin (blood glucose concentration 14·5 mmol/l (261 mg/100 ml) on admission). In addition, he needed sedation and elective ventilation for the first 18 hours of peritoneal dialysis.

Comment

Peritoneal dialysis for eight days in a 2½-year-old child who had ingested about 1·8 mol (110 g) ethylene glycol removed 0·43 mol (26·9 g),[4] a similar proportion of the total dose to that removed in the cases cited here, and the child made a full recovery. While peritoneal dialysis may not be as efficient as haemodialysis in removing ethylene glycol (Peterson *et al*[5] reported that haemodialysis for six hours in a 51-year-old man who had ingested about 9·7 mol (600 g) ethylene glycol removed 1·8 mol (111 g), while urinary excretion was 0·16 mol (10 g) during this period), clearly appreciable quantities of ethylene glycol were removed by peritoneal dialysis in our patients. Thus, in patients severely poisoned with ethylene glycol we believe that in addition to gastric lavage, supportive measures to combat shock and respiratory distress, the correction of metabolic acidosis and hypocalcaemia, and the use of ethanol, the early application of dialysis will remove substantial amounts of ethylene glycol and may prevent death. If haemodialysis is not immediately available then peritoneal dialysis is an effective alternative and should be instituted without delay when it is certain, on the basis of the clinical features and a plasma ethylene glycol concentration in excess of 8 mmol/l (0·5 g/l), that the patient is at serious risk from ethylene glycol toxicity.

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Lupus foot

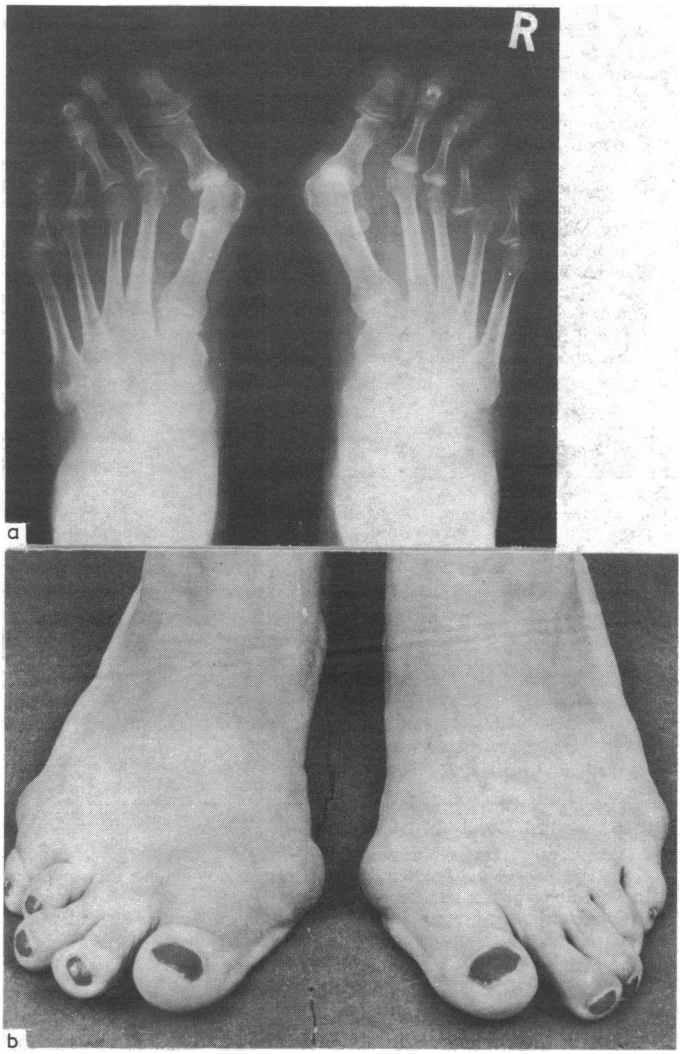
Articular symptoms are the most common clinical manifestation of systemic lupus erythematosus as well as the most frequent manner of presentation.[1] Up to 95% of patients with systemic lupus erythematosus have arthralgia or arthritis at some stage of their disease,[2 3] and in 74% more than two joints may be affected.[4] Symmetrical distribution is typical, and the joints most commonly affected in order of frequency are the finger proximal interphalangeal, knees, wrists, and metacarpophalangeal. Deformity may be severe, but bone and cartilage damage is uncommon and erosions rare.[1 3] Radiologically the main abnormality is subluxation, and the underlying pathology is thought to be contracture of tendons and laxity of the joint capsule.

We report on three patients with deformities and radiological changes in both feet due to systemic lupus erythematosus. We are not aware that this has been previously reported.

Case reports

Case 1—A 35-year-old Cypriot woman had had systemic lupus erythematosus for 14 years; throughout the course of her disease a prominent feature had been synovitis affecting the hands and feet. Deformity of the hands had developed at an early stage, and over the past three years her feet had become progressively abnormal (figure). Radiologically there were subluxa-

tion deformities of the fourth and fifth metatarsophalangeal joints of both feet; the proximal interphalangeal joints of the right third, fourth, and fifth toes; and also the proximal interphalangeal joints of the left fourth and fifth toes.



Case 1. Radiograph and photograph of the feet.

Case 2—A 40-year-old housewife had developed systemic lupus erythematosus at the age of 28 with arthritis, pericarditis, pleurisy, and focal proliferative glomerulonephritis. Over the past five years her hands had become progressively deformed with pronounced ulnar deviation and flexion contractures, and for two years she had had severe clawing of the toes. Radiography of the feet showed subluxation of the right third, fourth, and fifth metatarsophalangeal joints; the left third and fourth metatarsophalangeal joints; and the third left proximal interphalangeal joint.

Case 3—A 29-year-old girl had systemic lupus erythematosus for 16 years which had presented with thrombocytopenic purpura, fever, pleurisy, rash, and symmetrical arthritis affecting her hands, knees, and ankles. During the previous 13 years of her disease she had remained clinically and serologically active, with average DNA binding values of 75% (upper limit 30% in the Farr technique), and the main feature of her disease had been widespread arthralgias. Her hands had become grossly deformed with severe flexion contractures, and over the past two years she had developed similar changes in her feet. Radiography of the feet showed bilateral subluxation of the interphalangeal joints of the fourth and fifth toes and the interphalangeal joint of the right hallux.

Comment

Clinical and radiological abnormalities of the hands in systemic lupus erythematosus have been well documented.[1] The changes described here in the feet are similar to those observed in the hands, and the underlying disease is likely to be the same. The prevalence of foot deformity due to systemic lupus erythematosus is impossible to calculate accurately but is much lower than that of hand deformity. In all three patients the hands were particularly deformed and affected several years before the feet. While the patients we describe had had serologically active systemic lupus erythematosus for several years there did not appear to be a correlation between the development of arthritis and any clinical or serological pattern.

The process that results in the deformity is not understood but may result from inflammatory changes in tendons or periarticular structures. Occasionally a patient with systemic lupus erythematosus is noted to have pain and erythematous tenderness along the path of tendons. Inflammation and fibrosis have been described in tendon sheaths,[5] and this may in turn result in acute or insidious contracture of the tendon.

We thank Dr N B Bowley, radiology department, and Dr G R V Hughes, rheumatology unit, for help and advice.

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Hyponatraemia in legionnaires' disease

Hyponatraemia is common in legionnaires' disease, and has been attributed to the syndrome of inappropriate secretion of antidiuretic hormone.[1] Our results suggested, however, that an alternative explanation is more likely.

Patients, methods, and results

Between December 1976 and May 1979, 24 patients with acute pneumonia were found to have legionnaires' disease. Thirteen were hyponatraemic on admission (serum sodium concentration lower than 130 mmol(mEq)/l), and none was diabetic or receiving diuretics or corticosteroids. All had normal serum sodium concentrations (135-145 mmol/l) by the time of discharge. Before treatment five patients had measurements of sodium and osmolality, using standard methods, on simultaneous blood and urine samples.

Findings on admission in the five patients studied

Case No	Osmolality (mmol/kg)		Sodium (mmol/l)		Hypovolaemia
	Serum	Urine	Serum	Urine	
1	268	400	127	4	Present
2	265	817	129	5	Present
3	259	806	128	7	Present
4	270	615	129	6	Present
5	268	530	129	7	Present

Conversion: SI to traditional units—Osmolality: 1 mmol/kg = 1 mosmol/kg. Sodium: 1 mmol/l = 1 mEq/l.

Patients were considered to be hypovolaemic if jugular venous pulsation became visible only with the patient lying at an angle of less than 5° and systolic blood pressure was at least 20 mm Hg greater in the supine than in the standing position.

The table shows results of investigations on admission. All five patients were hypovolaemic, with high urine osmolality and urinary sodium concentrations below 8 mmol/l. In a group of 12 patients with legionnaires' disease 10 were hypovolaemic. One of the two exceptions had normal circulating