

(Dent 1966). The required dose was usually very constant, and he supported the expressed attitude of Parfitt (1970) that published series which suggested otherwise (Hossain 1970) almost certainly illustrated inadequate clinical control.

## REFERENCES

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### Hyperuricæmia and Renal Failure Preceding the Onset of Acute Lymphoblastic Leukæmia

W J Appleyard BM MRCP  
(*Guy's Hospital, London*)

A H, boy aged 6

**History:** Presented with epistaxes and was found to have a pancytopenia (Hb 4.1 g/100 ml, WBC 1,700, platelet count 88,000/mm<sup>3</sup>). Bone marrow normal. He was transfused and a spontaneous rise in the white cells and platelets occurred. One month later he developed pain and swelling in his left middle toe and right foot. He was readmitted in acute renal failure (blood urea 425 mg/100 ml, serum potassium 10.4 mEq/l) which was successfully treated with peritoneal dialysis. The uric acid level was 78 mg/100 ml (Fig 1). IVP showed a normal pelvi-calyceal system and normal-sized kidney. His parents had normal uric acid levels.

**Treatment and progress:** Allopurinol was prescribed until the uric acid level had been normal for 2 weeks. A pancytopenia was again found though blood films and two bone marrow examinations revealed no abnormal cells. He returned to hospital a few weeks later in renal failure with a uric acid of 76 mg/100 ml and white count of 300,000/mm<sup>3</sup>; the bone marrow biopsy confirmed acute lymphatic leukæmia.

Forced alkaline diuresis (Holland & Holland 1968) resolved the hyperuricæmia and renal failure and a remission was induced with pred-

nisolone and vincristine. Allopurinol was started and continued after his discharge (Krakoff 1966) the uric acid has remained normal except during a relapse when it rose to 26.5 mg/ml, returning to normal in his present remission.

## Comment

Raised uric acid levels in leukæmia have been found at the time of diagnosis or after cytotoxic therapy. These have been correlated with the degree of lymphoblastic proliferation (Lynch 1962) and uric acid excretion (Sandberg *et al.* 1956). Secondary gout where these symptoms precede the onset of acute leukæmia is less common (Schultz 1931). When our patient first developed hyperuricæmia and renal failure, intensive search failed to reveal any sign of a proliferative disorder. We were faced with the paradox of diagnosing the complication of a disease before the disease itself was diagnosable.

## REFERENCES

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### Pancreatic Calcification with Pancreatic Insufficiency in an 8-year-old Girl

Caryl W Darby MB MRCP  
(*The Hospital for Sick Children, Great Ormond Street, London WC1*)

S K, girl aged 8

**History:** Born at term weighing 3.17 kg. At 9 months she was treated for two months with a gluten-free diet because of abdominal pain and steatorrhœa, with no improvement. Between the ages of 3 and 7 recurrent attacks of undiagnosed abdominal pain associated with fever (39°C), anorexia, vomiting and steatorrhœa occurred. In June 1969 pancreatic calcification was noted on a plain abdominal X-ray (Fig 1) and further investigations confirmed pancreatic insufficiency and steatorrhœa. At this time her height was 117 cm (25–50th percentile), her weight 21.25 kg (10–25th) and, apart from signs of easy bruising, physical examination was normal.

**Investigations:** Repeated sweat tests were normal, excluding cystic fibrosis. Duodenal juice trypsin 4 units (normal >9), lipase 8.2 units (normal >20), amylase 630 units (normal >85). Five-day fat balance: fat excretion 8.2 g/day.

**Diagnosis:** She had no features suggesting congenital exocrine pancreatic hypoplasia (Shwachman *et al.* 1964) and did not fit into the picture of trypsinogen deficiency (Townes 1965) recently thought to be due to intestinal enterokinase deficiency (Tarlow *et al.* 1970).

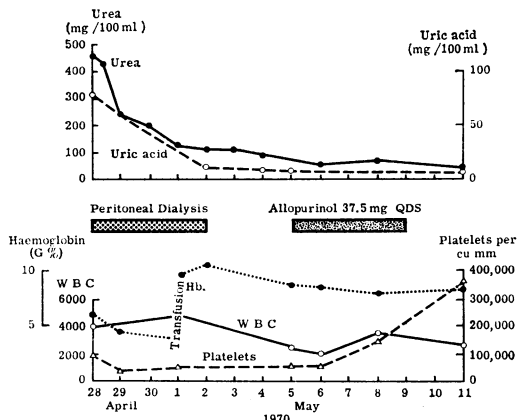


Fig 1 Response of hyperuricæmia to peritoneal dialysis