months previously the patient had begun a course of Ferrogradumet (slow-release iron), after being found to have a haemoglobin of 11.2 g/dl.

On admission her abdomen was rigid and a plain x-ray film showed free air under the right diaphragm but no other fluid levels. Intestinal perforation was diagnosed. At operation severe generalised peritonitis was found, though with little free fluid. The upper jejunum contained several diverticula along the mesenteric border. The largest of these (1.7 cm in diameter) was perforated, with a tablet extruding through the hole. The affected bowel was resected and an end-to-end anastomosis performed. Initially after the operation the patient was unable to maintain normal blood gas tensions without mechanical ventilation and she did not regain full consciousness. For a short period she could breathe without assistance but after a day her condition deteriorated and, despite antibiotic treatment and further mechanical ventilation, she died 72 hours after the operation. At necropsy the cause of death was found to be severe bronchopneumonia. Histological examination of the resected jejunum showed the perforated diverticulum to be only minimally inflamed, but at the site of the perforation the tissues were heavily stained with iron, with increasing density up to the actual perforation. The perforation appeared to be due to focal necrosis from local iron toxicity. The tablet lodged in the hole was shown by section and staining to be a slow-release matrix containing some unreleased iron. The tablet had the appearance of the Ferrogradumet previously prescribed for the patient.

Discussion

Slow-release tablets may consist of either multiple small particles of differing rates of dissolution or single entities in which the active principle leaches out from the matrix which remains unabsorbed. Local toxicity has not been reported with the first type, but has been reported when tablets of the second type have been retained in contact with the intestinal wall for prolonged periods. This has been most frequently described with potassium preparations. One case involving Ferrogradumet has been reported,⁴ where a tablet lodged in a Meckel's diverticulum caused local gangrene.

Since it is not normally possible to predict which patients are likely to have gut abnormalities it would seem wise to restrict this type of tablet formulation to patients unable to tolerate oral medication in any other form.

I would like to thank Mr R T Burkitt for allowing me to report this case.

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Transient hypocalcaemia and T-cell deficiency in an infant

Hypocalcaemia occurs commonly in the first two weeks of life. In the first three days this is a feature particularly affecting sick low-birth-weight infants and later in the first week well infants taking high phosphate feeds.¹ Serum parathyroid hormone concentrations are frequently low.²

Hypocalcaemia in association with T-cell deficiency was first described by Di George. In this syndrome there is abnormal development of structures derived from the third and fourth pharyngeal pouches with absence of the thymus and parathyroids, abnormalities of the great vessels, and characteristic facies.³

The finding of temporary hypocalcaemia and T-cell deficiency has not to our knowledge been reported.

Case report

A 6-week-old Pakistani boy was admitted with a two-day history of convulsions. He had been a full-term baby (birth weight 3000 g), entirely breast-fed, and physical findings were normal. He continued to have intermittent

convulsions for seven days. The results of initial investigations showed a definite hypocalcaemia (serum concentrations of calcium 1.32 mmol/l (5.3 mg/100 ml), phosphate 2.8 mmol/l (8.7 mg/100 ml), and magnesium 0.68 mmol/l (1.66 mg/100 ml)). He was treated with intravenous and oral calcium supplements, together with anticonvulsants, and after the seventh hospital day no more fits occurred. The serum calcium concentration, however, remained below 2 mmol/l (8 mg/100 ml) for almost three weeks.

Immunological values after correction of hypocalcaemia

Age (weeks)	Serum calcium (2.35-2.65) (mmol/l)	T cells (50-70)	B cells (12-25)	Lymphocyte transformation ratio of tritiated thymidine in test/control		
				PHA 2.0 µg/ml (93-918)	PHA 0.2 µg/ml (30-700)	Pokeweed mitogen
10	2.44	25	39	1.0	0.9	161
14	2.58	29	40	17	21	77
22	2.61	26	30	241	45	14
32		51	25	155	63	37

Conversion: SI to traditional units—Calcium: 1 mmol/l $\approx$ 4 mg/100 ml.

Serum parathyroid hormone estimation on the tenth day was 0.28 ng/ml (upper limit in an adult 0.20 ng/ml), when the serum calcium was 1.67 mmol/l (6.7 mg/100 ml). The results of numerous laboratory and radiological investigations were normal, with no evidence of rickets, but there was doubt about the presence of a thymic shadow on the initial chest x-ray film—while the result of the serum parathyroid hormone estimation was not immediately available. No cardiovascular abnormalities were evident, but Di George's syndrome seemed a distinct possibility. Lymphocyte function tests were, therefore, carried out. The lymphocytes showed an impaired response to stimulation with phytohaemagglutinin (PHA) and an exaggerated response to pokeweed mitogen. In addition, lymphocytes giving spontaneous sheep erythrocyte rosettes (T cells) were reduced, while complement rosetting cells (B cells) were increased. These abnormalities were still present at 14 weeks of age, had shown improvement by 22 weeks, and were absent by 32 weeks.

Discussion

Hypocalcaemia presenting at 6 weeks of age is unusual. Our patient had no findings to suggest associated gastrointestinal, renal, or parathyroid disease. The raised serum parathyroid hormone concentration was expected in the presence of hypocalcaemia and excluded parathyroid insufficiency. We found normal concentrations of calcium, phosphate, and alkaline phosphatase in the mother six weeks after delivery. The possibility that vitamin D deficiency in the mother and infant was the cause of the hypocalcaemia is, therefore, unlikely. The initial hypomagnesaemia was corrected with a single dose of magnesium sulphate, and, although the convulsions continued, subsequent serum magnesium concentrations were normal.

Impairment of T-cell function has been shown in hypocalcaemic rats after parathyroidectomy. Lymphocytes in the thymus showed definite reduction in mitotic activity, leading to atrophy of thymic tissue.⁴ B-cell function was not affected and there was no alteration in the number of antibody-producing cells in the spleen.⁵ Partial correction of the hypocalcaemia by increasing dietary calcium reduced significantly the degree of thymic atrophy.

Possibly the hypocalcaemia in our patient, in the presence of normal parathyroid function, may have been responsible for the apparent delay in maturation of immunological function under control of the thymus.

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