Discussion

This study shows that serum hepatitis B virus-DNA analysis is a very sensitive and specific method of identifying those hepatitis B surface antigen carriers who are at high risk of transmitting infection to their offspring. Hepatitis B virus-DNA was found in all mothers positive for hepatitis B e antigen for whom the risk of perinatal transmission was already established.¹ A consistent proportion (11%) of mothers positive for hepatitis B e antibody, and, therefore, considered at low risk,¹⁻² showed hepatitis B virus-DNA in the serum, indicating the presence of replicating hepatitis B virus and thus a high infectivity.⁵

Perinatal hepatitis B virus transmission occurred in only three offspring, all of whom were born to hepatitis B virus-DNA positive mothers. One of these mothers was also positive for hepatitis B e antibody and the others for hepatitis B e antigen.

In evaluating these results it should be remembered that the maternal transfer of hepatitis B surface antigen in these babies may have been affected by the immunoglobulin administration, and this may explain why no persisting carrier of hepatitis B surface antigen was found among them. Our results suggest that transmission is primarily

related to the presence of hepatitis B virus-DNA in the serum. The lack of a control group, however, and the small number of subjects included do not allow any definitive conclusions on this point.

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Laxative abuse and secretory diarrhoea

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SUMMARY Three children with chronic diarrhoea secondary to laxative abuse are reported. Growth disturbance, a previously unrecognised feature of this form of abuse, is recorded.

Laxative abuse is well recognised as a cause of chronic diarrhoea in adults,¹ but the diagnosis is rarely considered in children. Three children presented with chronic diarrhoea secondary to laxative abuse during a three year period when 600 new referrals were assessed.

Case reports

Case 1. A 3 year old boy was referred with chronic diarrhoea. After an acute infection at 3 months of age, he had had diarrhoea every four to six weeks

and passed up to eight watery stools each day for four to five days. A progressive fall off in growth occurred. The elimination of cows' milk and gluten from his diet had not altered his clinical course. The parents had long standing marital conflicts and his mother had required psychiatric treatment: she denied laxative administration.

The patient was a quiet, pale boy with height (87 cm) and weight (10.8 kg) both below the fifth centile. The remainder of the physical examination was normal. Stool smear contained no neutral fat or fatty acid crystals. Haematological and biochemical assessment, stool examination, and stool culture were all normal. Dietary assessment of caloric intake varied from 32 to 80% of recommended nutritional intake.

On assessment in hospital, stools were initially normal, but two days later became watery. They were negative for reducing substances. Stool elec-

trolyte concentrations were sodium 88 mmol/l, chloride 77 mmol/l, and potassium 57 mmol/l. The addition of sodium hydroxide to the stool and urine produced an intense pink colour and laboratory testing confirmed the presence of phenolphthalein.

When confronted with the diagnosis the mother admitted giving him laxatives. The family was referred to the Child Abuse Service. During psychotherapy it was learned that the mother had herself been the victim of child abuse. The child's diarrhoea resolved when laxatives were stopped and he was discharged to foster care with the aim of eventual family reunification.

Case 2. A 3 year old boy with intractable diarrhoea was admitted for assessment. He had been adopted as an infant and had suffered only minor infections until 18 months of age. From 18 months to 3 years he passed from five to 12 explosive watery stools each day. Growth parameters had shown a progressive fall from the 50th centile. Extensive previous investigations had yielded negative or normal results and ruled out infectious, inflammatory, or metabolic causes of his diarrhoea. Hyponatraemia and acidosis at the time of severe diarrhoea were the only abnormalities ever detected. Cows' milk-free diet, elemental diet, cholestyramine, and disodium cromoglycate treatment failed to control the diarrhoea. Satisfactory weight gain had only been achieved during three periods of total parenteral nutrition.

At the time of admission his height (86 cm) and weight (9.8 kg) were both below the fifth centile. He had diminished subcutaneous tissue, but otherwise the physical examination was normal. Serum sodium was 126 mmol/l and serum bicarbonate 11 mmol/l. Stool sodium was 88 mmol/l, potassium 60 mmol/l, and chloride 93 mmol/l. The addition of sodium hydroxide to stool and urine produced a bright red colour and laboratory assessment identified the presence of phenolphthalein.

The mother was confronted, and admitted administering laxatives to the child. The family was referred to the Child Abuse Service. The boy's diarrhoea ceased promptly and he showed rapid weight gain.

Case 3. A 13 year old girl was referred because of a three month history of passing 15 to 30 watery stools each day. She also complained of vomiting and anorexia and had lost 10 kg in weight. Extensive investigations before referral failed to find any infection or evidence of inflammatory bowel disease. Past history and family medical history were non-contributory. Her parents had been divorced four years before the onset of the symptoms and there was still considerable familial discord.

On examination, she was withdrawn and depressed. Her height (171 cm) was at the 95th centile and weight (48.2 kg) at the 50th centile. Physical examination was otherwise normal. A stool smear was unremarkable, and stool cultures were again negative. Stool electrolytes were sodium 88 mmol/l, potassium 25 mmol/l, and chloride 98 mmol/l. Addition of sodium hydroxide to stool specimens produced a colour change characteristic of phenolphthalein. The diagnosis of laxative abuse was initially denied by the girl and elicited a hostile response from the parents. Laxative tablets were found in the girl's purse, her locker at school, and in her room at home. She later admitted to taking laxatives because of her depression and feelings of worthlessness. Family psychiatric treatment was begun. The diarrhoea ceased abruptly and she gained 4.2 kg in the first month after discharge from hospital.

Discussion

Recognition of factitious illness in children followed some time after the description of Munchausen's syndrome in adults, but is now well described.² These three children had features which, when present, should alert the clinician to the possibility of laxative abuse. All had a history of chronic watery diarrhoea, growth disturbance believed to be secondary to inadequate caloric intake, and disturbed family dynamics. Severe fluid and electrolyte disturbance occurred in one patient and has been previously recognised as a feature of laxative abuse in children.^{3,4} The symptoms that develop with laxative abuse enabled the parent and child to receive attention and to escape from the home environment.

The diarrhoea produced by stimulant laxatives has the features of a secretory diarrhoea. The stools persist despite the cessation of oral intake; they are voluminous, watery, and free of blood, pus, or abnormal amounts of fat and carbohydrate; and the normal stool electrolyte concentrations are altered. There is a noticeable increase in the sodium chloride content and loss of the osmotic gap between stool electrolyte concentrations and osmolality that is seen in osmotic diarrhoea. Alkalinisation of stool or urine containing phenolphthalein or aloes, produces a colour change to red, pink, or mauve. This is shown by the slow addition of NaOH to stool or urine. Senna can be detected by the addition of sulphuric acid and carbon tetrachloride to urine.⁵ A chromatographic method can detect all the phenolic and anthraquinone laxatives in urine up to 32 hours after ingestion.⁶

Our experience suggests that laxative abuse is not rare and should be considered in any infant or older

child with persistent unexplained diarrhoea. Additional clues to the diagnosis are growth disturbance, electrolyte abnormalities, and social and psychological disturbance within the family.

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Diarrhoea due to *Cryptosporidium* in acute lymphoblastic leukaemia

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SUMMARY We describe a case of recurring cryptosporidiosis during immunosuppressive treatment. The infection occurred after contact with an affected kitten and followed a less severe course than expected. After withdrawal of immunosuppressive treatment the infection resolved.

The protozoan parasite *Cryptosporidium* is increasingly recognised as a cause of enterocolitis in humans. In immunologically competent individuals, the commonest manifestation is explosive watery diarrhoea usually accompanied by vomiting, anorexia, cramping abdominal pains, and low grade fever. This is a self limiting illness lasting three to 14 days.¹

Cryptosporidiosis in immunocompromised patients is characterised by intractable watery diarrhoea that is resistant to antimicrobial treatment and is associated with high mortality.¹ We report a case of recurring cryptosporidiosis in a child on maintenance treatment for acute lymphoblastic leukaemia in whom the disease followed a less severe course than is usually recognised in immunocompromised subjects and which followed contact with an infected kitten.

Case report

A 5½ year old boy was diagnosed as having acute lymphoblastic leukaemia (non B, non T, c-ALL⁺^{ve}) at the age of 4 years 9 months and was treated

according to the Medical Research Council UKALL VIII A protocol. Nine months after diagnosis, while on maintenance treatment and in haematological remission, he presented with a brief history of anorexia, nausea, and loose stools. This coincided with similar symptoms in both parents and a sister. There were no abnormal clinical or laboratory findings and treatment was continued. The symptoms rapidly resolved in the rest of the family but continued for the subsequent four weeks in the patient. He became increasingly anorexic with intermittent vomiting, had between one and two profuse, watery, yellow stools per day, and suffered just over 10% weight loss.

Investigations six weeks after the onset of symptoms showed a haemoglobin concentration of 11.6 g/dl, total leucocyte count $3.5 \times 10^9/l$, neutrophils $1.75 \times 10^9/l$, lymphocytes $0.7 \times 10^9/l$, monocytes $0.98 \times 10^9/l$, eosinophils $0.07 \times 10^9/l$, and platelets $384 \times 10^9/l$. Bone marrow aspiration showed a marrow in remission with dyserythropoietic features. Urea, electrolytes, liver enzymes, serum proteins, and iron were normal. Serum folate was 1.4 mg/l (normal 3-12 mg/l), red cell folate 105 ng/ μ l (normal 200-700 ng/ μ l). His one hour D-xylose absorption was 0.49 mmol/l (7.3 mg/100 ml) (normal 1.33 mmol/l (20 mg/100 ml)). Bacterial and viral stool cultures were negative on a number of occasions, but oocysts of *Cryptosporidium* were detected on microscopic examination of stools using a safranin-methylene blue method.² A jejunal biopsy showed noticeable cellular infiltrate but no abnormalities of the villi. Cryptosporidial trophozoites were found in