of gross marrow activity. A few days after the child was admitted to hospital his mother developed a sore throat and follicular tonsillitis. Group A β -haemolytic streptococci were isolated from the mother's throat, the grandmother's nose, and the nose of a nursery nurse in the maternity department. The streptococci from the patient, his mother, and his grandmother were indistinguishable by typing (M3.R3.T3/13/B3264) but were different from the nurse's strain (M4.R-T4).

Comment

Group A streptococcus used to be a major cause of neonatal septicaemia.⁴ The clinical picture is different from the now predominant group B streptococcal disease.⁵ In this case the onset of septicaemia was slow and was obvious only after admission to hospital. The rash that developed in hospital was similar to that seen in meningococcal septicaemia, and new lesions developed as the child was observed. The broad-spectrum antibiotic treatment was maintained only until the identity and sensitivity pattern of the organism isolated from the blood cultures were known. The isolation of indistinguishable strains from the child's mother and grandmother suggested that the infection was acquired at home. There was no evidence of active streptococcal disease in the family immediately before the child's illness.

Group A streptococcal disease is unusual in neonates and it is also uncommon in Guildford to isolate this organism from routine nose and throat swabs from children under 6 months old. The finding of the streptococcus in the nose and throat of this child was regarded as potentially important, and penicillin treatment was started at once. In the absence of any other septic focus possibly the septicaemia followed initial colonisation of the upper respiratory tract.

I thank Dr S J R Macoun for permission to report this case. Typing of the streptococci was done at the Cross Infection Reference Laboratory, Colindale.

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Appendix abscess with intestinal haemorrhage

I report a case of appendix abscess with haemorrhage into the caecum. This rare association should not be managed conservatively if there are also symptoms and signs consistent with appendicitis.

Case report

A previously fit 14-year-old schoolboy was admitted to hospital in June 1974 with a two-week history of intermittent colicky pain in the right iliac fossa and vomiting, associated initially with diarrhoea. Apart from Lomotil (diphenoxylate hydrochloride and atropine sulphate) at first and a soluble aspirin tablet taken on the night of admission there had been no drug treatment. Examination showed an ill-looking, pale, obese boy. The pulse was 140/min, blood pressure 120/80 mm Hg, and temperature 37.5°C. The abdomen was mainly soft with normal bowel sounds, but there was tenderness with guarding and rebound in the right iliac fossa. Rectal examination showed copious melaena mixed with fresh, bloody stool. Haemoglobin was 9.9 g/dl, white cell count $21.4 \times 10^9/l$, (21 400/mm³), and blood urea 7.3 mmol/l (44 mg/100 ml).

After resuscitation with intravenous saline and two units of whole blood operation was performed. Under anaesthesia a large mass was felt in the right iliac fossa. Through a right paramedian incision an extensive retrocaecal abscess was seen that filled the right paracolic gutter and contained foul-smelling blood clot and pus. There was blood in the lumen of the whole of the colon but none proximal to the caecum. A 1-cm perforation was seen in

the posterior caecal wall, with two faecaliths lying free in the abscess cavity. After drainage and ileocaecal resection with anastomosis the patient made good recovery. Postoperative haemoglobin was 11.9 g/dl and blood urea was 2.7 mmol/l (16 mg/100 ml). At follow-up haemoglobin, white cell count, plasma viscosity, blood urea, and results of barium meal and follow-through examination of the small bowel were all normal. He has been well since.

Histology—The specimen was 30 cm of terminal ileum and 12 cm of caecum. The mucosa of the terminal ileum contained many polypoid projections of about 0.3 to 0.5 cm in diameter. The appendix was embedded in fibroadipose tissue with a perforation through it into the caecum 5 cm distal to the ileocaecal valve. The mesenteric lymph nodes were enlarged up to 1 cm in diameter. Sections of the appendix showed inflammation extending through the wall into surrounding tissue where there was granulation and fibrosis. There was fibrosis and inflammation of pericaecal tissue and considerable caecal submucosal oedema and lymphoid hyperplasia. In one place inflammation had spread through the caecal wall causing extensive muscular necrosis. The serosal surface of the ileum was inflamed and hyperplastic mucosal lymphoid tissue formed the polypoid mucosal projections. The appearance was consistent with perforated appendicitis and an appendix abscess.

Comment

Presumably the bleeding was caused by infection from a sloughed appendix stump. Crohn's disease was excluded by the clinical picture, subsequent progress, histology, and the negative results of post-operative investigations. A case of considerable haemorrhage associated with appendicitis has not been reported for at least 30 years. Slight bleeding with occult blood in the stool has been reported with "appendicular granuloma."¹ Postoperative secondary haemorrhage, either into the bowel or the wound, was referred to in the pre-antibiotic era.^{2,3} Fistulation between an iliac artery and the caecum has been reported.⁴ The fatal termination of an appendicectomy by sudden haemorrhage has been attributed to forcibly hooking out an inflamed appendix adhering to the iliac vessels.⁵ Intestinal haemorrhage, as in this case, has been mentioned in older texts.^{3,5}

I thank Mr A J Webb, consultant surgeon, Bristol Royal Infirmary, for permission to report this case, and Dr J Tudway, consultant pathologist, Bristol Royal Infirmary, for the pathological report.

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Bromocriptine and premenstrual syndrome: controlled study

We investigated whether prolactin is important in the aetiology of the premenstrual syndrome and depression.¹ Bromocriptine, which reduces plasma prolactin concentrations, was used in a controlled study of patients with the premenstrual syndrome.

Patients, methods, and results

Women attending a group of general practitioners because of minor symptoms were asked if they suffered from the premenstrual syndrome, and if so whether they would take part in a trial of treatment. Those who agreed completed a standardised menstrual questionnaire² asking about the occurrence of premenstrual irritability, depression, anxiety, headaches, and

swelling. Most symptoms were rated on a four-point scale from nil to severe. Women who rated moderate or severe were included in the trial provided that their periods were regular and they were not taking oral contraceptives or sex hormones.

Thirteen patients completed the trial. It had a double-blind, crossover design, and patients were allocated at random to receive either bromocriptine (2.5 mg) or identical placebo tablets. The patients were instructed to take one tablet (bromocriptine 2.5 mg or placebo) daily 10 days before the start of their next period. Ten days before the subsequent period they were given the other tablets.

The total scores for each course of treatment are shown in the table. Statistically there was no significant difference between the two treatments or order of treatments.

Mean age and total scores ($\pm$ SE) of patients taking bromocriptine and placebo for premenstrual syndrome (PMS)

Group	Mean age in years	PMS total scores	
		Placebo phase	Active phase
Placebo first (n=5)	34.2 $\pm$ 3.5	6.0 $\pm$ 1.0	6.4 $\pm$ 1.4
Bromocriptine first (n=8)	33.2 $\pm$ 3.4	6.6 $\pm$ 1.1	5.0 $\pm$ 0.8
Combined series (n=13)	33.6 $\pm$ 2.5	6.4 $\pm$ 0.7	5.5 $\pm$ 0.7

Comment

Our results clearly do not support the theory that prolactin is related to premenstrual mood disturbances or to the symptoms of swelling or headaches. They do not agree with those of Benedek-Jaszmán *et al.*,³ who observed significant alleviation of the premenstrual syndrome after bromocriptine treatment. The dosage used in their study, however, was 2.5 mg twice daily, which is double the dose used in our study. We decided to use a smaller dosage after a pilot study had shown that most of the women were unable to tolerate 2.5 mg twice daily. We also found that a single 2.5 mg oral dose was sufficient to reduce plasma prolactin to a significantly low concentration after two hours, and that the effect lasted for six to eight hours.⁴

We thank Drs E Saphier, S V Kar, O Meerapfel, and G Wills for invaluable help in the trial, and Mr J Bailey for the statistical evaluation of the data. We are grateful to Sandoz Products Limited for supplying the bromocriptine and placebo tablets.

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Symptomatic magnesium deficiency after small-intestinal bypass for obesity

The first reported successful massive intestinal resection was in 1881.¹ Since then small intestinal bypass operations have gained a limited place in the management of obesity. Common complications of such operations² include vomiting, diarrhoea, malabsorption, liver failure, pancreatic insufficiency, and disturbances of potassium and calcium balance. Magnesium deficiency, however, is rare^{3 4}; indeed, Shibata⁵

found no overt signs of magnesium deficiency in his series. I report a case of symptomatic hypomagnesaemia occurring after jejunoileal anastomosis for obesity.

Case report

A 40-year-old woman who weighed over 160 kg was admitted to hospital for a small-intestinal bypass operation. Results of all preoperative haematological, biochemical, and endocrine investigations were normal. A 30 cm-30 cm jejunoileal anastomosis was performed, from which she recovered uneventfully, and she was discharged two weeks later. When seen at six weeks as an outpatient she had lost 20 kg but was having five bowel movements daily.

Sixty-two days postoperatively she was readmitted as an emergency case. She complained of weakness and dizziness, with "pins and needles" in her hands and feet. On examination Chvostek's sign and Trousseau's phenomenon were seen, and latent tetany due to hypocalcaemia was diagnosed. Serum calcium, magnesium, and potassium concentrations were 1.35 mmol/l (5.4 mg/100 ml), 0.35 mmol/l (0.85 mg/100 ml), and 2.2 mmol (mEq)/l respectively (see table). She was given 10 ml 10% calcium gluconate intravenously and oral calciferol 1.25 mg (50 000 IU) three times daily, but the serum calcium continued to fall. On day 68 she again developed paraesthesiae and was given a further 10 ml 10% calcium gluconate. The serum potassium was 2.50 mmol (mEq)/l and magnesium was 0.40 mmol/l (0.9 mg/100 ml) and a new diagnosis of hypomagnesaemia was made. Calcium supplements were stopped the next day and intramuscular injections of 2 ml 50% magnesium sulphate five times daily were started. This rapidly controlled her symptoms and corrected her electrolyte imbalance within five days.

Serum electrolyte concentrations in a patient before and after treatment with 10% calcium gluconate, calciferol, and 50% magnesium sulphate

Day	Calcium (normal range 2.25-2.65 mmol/l)	Magnesium (normal range 0.7-0.9 mmol/l)	Potassium (mmol/l)
0	2.22	1.05	3.75
62	1.35	0.35	2.20
65	1.42		2.50
68	1.20		
69	1.22	0.40	2.50
72	1.62	1.40	3.10
76	2.15	0.80	3.30

Conversion: SI to traditional units—Calcium: 1 mmol/l $\approx$ 4 mg/100 ml. Magnesium: 1 mmol/l $\approx$ 2 mg/100 ml. Potassium: 1 mmol/l $\approx$ 1 mEq/l.

She was started on Lomotil (diphenoxylate hydrochloride and atropine sulphate), which controlled her diarrhoea, and then discharged.

At follow-up after six months she was asymptomatic with normal electrolyte concentrations without any further treatment.

Comment

This woman presented with calcium-resistant latent tetany due to magnesium deficiency after jejunoileal anastomosis. Dietary magnesium is uniformly absorbed throughout the small intestine with little or no absorption in the large intestine. The acute magnesium deficiency was probably caused by increased faecal loss of magnesium. After a massive resection the small intestine adapts by dilatation, elongation, and villous hypertrophy. In this case the increase in available surface area for absorption because of adaptation together with control of the diarrhoea presumably resulted in magnesium and calcium homeostasis.

I thank Mr J Leslie for permission to report this case, and Mrs J Cragg for help in preparing the report.

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