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.

[1] Wilson, H D, and Eichenwald, H F, *Pediatric Clinics of North America*, 1974, **21**, 511.
[2] Parker, M T, and Ball, L C, *Journal of Medical Microbiology*, 1976, **9**, 275.
[3] Nieburg, P I, and Williams, M L, *Journal of Pediatrics*, 1975, **87**, 453.
[4] Dunham, E C, *American Journal of Diseases of Children*, 1933, **45**, 229.
[5] McCracken, G H, *Journal of Pediatrics*, 1973, **82**, 703.

(*Accepted 8 October 1976*)

Public Health Laboratory, Guildford, Surrey
R Y CARTWRIGHT, MB, MRCPATH, consultant microbiologist

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 $21{\cdot}4 \times 10^9$/l, (21 400/mm^3), 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.

[1] LeBrun, H I, *British Journal of Surgery*, 1958, **46**, 32.
[2] Fowler, G R, *A Treatise on Appendicitis*, p 60. Philadelphia, Lippincott, 1894.
[3] Kelly, H A, and Hurdon, E, *The Vermiform Appendix and its Diseases*, pp 391, 404, 662. Philadelphia and London, Saunders, 1905.
[4] Hawkins, H, *On Diseases of the Vermiform Appendix*, p 100. London, MacMillan, 1895.
[5] Bailey, H, *Emergency Surgery*, 5th edn, p 149. Bristol, John Wright and Sons, 1944.

(*Accepted 12 October 1976*)

Cheltenham General Hospital, Cheltenham, Glos GL53 7AN
P J MILEWSKI, MB, FRCS, surgical registrar (formerly senior house officer, United Bristol Hospitals)

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 questionnaire[2] asking about the occurrence of premenstrual irritability, depression, anxiety, headaches, and