mouth. Electroencephalography and brain scanning showed a focal lesion in the left temporal region. At the same time she developed conjunctivitis and her plasma calcium was found to be 3.4 mmol/l (13.6 mg/100 ml). Intravenous treatment restored the plasma urea to normal without correcting the hypercalcaemia. She remained drowsy, confused, and hypotensive, and had light, generalised pigmentation. Her plasma sodium concentration remained normal, plasma phosphate was 1.2 mmol/l (3.7 mg/100 ml), alkaline phosphatase 7 KA units, and urinary calcium 10 mmol/24 h (400 mg/24 h). Radiography showed erosions in the phalanges of the hands and feet (see figure). The leucocyte count was $7.1 \times 10^9/l$ (7100/mm³), with 12 % eosinophils.

Subperiosteal erosions on radial side of proximal and middle phalanges of right index finger and middle phalanges of middle and ring fingers.

We thought that the patient was in hypercalcaemic crisis. To allow time to determine its cause we gave her phosphate and prednisolone by mouth. The plasma calcium fell to 2.4 mmol/l (9.6 mg/100 ml) in four days and she improved. Further investigation showed no evidence of sarcoidosis, leukaemia, paraproteinaemia, or other malignant disease or of vitamin D ingestion. The radiological findings seemed to indicate hyperparathyroidism.

Phosphate and prednisolone were stopped and the patient went to a surgical ward for neck exploration. With induction of anaesthesia she was given 100 mg hydrocortisone because of the recent exposure to steroids. Exploration of the neck and mediastinum showed four parathyroid glands that were normal on microscopy of frozen sections. After surgery she remained well for 24 hours but then developed mild fever and tachycardia. Plasma calcium, urea, and electrolyte values were normal. Deterioration continued over the next 10 days with increased fever, hypotension, tachycardia, and confusion. We diagnosed adrenocortical insufficiency because of the hypotension, pigmentation, and eosinophilia and the improvement when treated with prednisolone, which was earlier attributed to the phosphate treatment. At this stage the plasma chemistry was: Na⁺ 122 mmol(mEq)/l, K⁺ 6.6 mmol(mEq)/l, Cl⁻ 88 mmol(mEq)/l, HCO₃⁻ 17 mmol(mEq)/l, and urea 15.7 mmol/l (94.6 mg/100 ml). The plasma 11-hydroxycorticosteroid concentration was 196 nmol/l with no rise after tetracosactrin. Treatment was started with intravenous saline and hydrocortisone, and within 12 hours her temperature, pulse, and blood pressure were normal. She remained well on hydrocortisone and fludrocortisone with no neurological symptoms and normal electroencephalograms and brain scans. The radiological abnormalities healed in two years. Her serum contained adrenal autoantibodies.

Comment

We know of no case of adrenocortical insufficiency presenting with hypercalcaemic crisis. Our case is also unique because the bone lesions seen on radiography resembled the typical erosions of hyperparathyroidism. Bone resorption has not been noted previously in

adrenocortical insufficiency. Indeed, increased bone resorption is characteristic of cortisol excess.⁴ A wide range of neurological and psychiatric symptoms occur in hypercalcaemia.¹ EEG abnormalities are well known⁵ but the abnormal brain scan is difficult to explain.

Several features of our case might have made us question the diagnosis of hyperparathyroidism. These included the eosinophilia and the normal plasma phosphate and plasma alkaline phosphatase values in a patient with bone lesions. The presenting symptoms, however, were those of severe hypercalcaemia of any cause, and the diagnosis of hyperparathyroidism was apparently confirmed by the radiological findings. Though our case is exceptional, we feel that adrenocortical insufficiency should be remembered as an important treatable cause of hypercalcaemia.

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Neonatal septicaemia due to group A beta-haemolytic streptococcus

Streptococcal septicaemia in neonates is usually associated with strains of Lancefield's group B.^{1,2} Though no longer the scourge of maternity units, group A streptococci may still cause severe neonatal infection.³ I report a case of group A streptococcal septicaemia in a 2-week-old boy in which the probable source of infection was the mother or grandmother.

Case history

A full-term baby boy was delivered with forceps at St Luke's Hospital, Guildford, on 24 April 1976. He weighed 4080 g. Apart from mild, transient jaundice the immediate neonatal period was uneventful and both mother and child were discharged home the third day after delivery.

Ten days later the child developed an erythematous lesion on his left cheek, which spread to the trunk during the next two days. The lesion was slightly raised but not typical of erysipelas. The cheek became swollen and the eyelid oedematous. The baby, who had been breast-fed since birth, began to take feeds poorly.

On admission to hospital the erythematous rash covered the left half of the face, the neck, the trunk, and the legs up to the knees. The rash was indurated over the neck but not elsewhere. The rectal temperature was 39°C. The white cell count was $9.3 \times 10^9/l$ (9300/mm³) with 35 % polymorphs and 61 % lymphocytes. No cells or bacteria were seen in a urine specimen. Nose and throat swabs were taken and blood was cultured.

During the next 24 hours the child deteriorated rapidly and the rash became confluent. As a β -haemolytic streptococcus had been isolated from the nose and throat swabs, intravenous penicillin 125 000 U six-hourly was given. He continued to worsen and developed a generalised petechial rash. Septicaemia was diagnosed and ampicillin and gentamicin were given in addition to the penicillin. Hydrocortisone treatment was also started. Progress was stormy, with many episodes of respiratory arrest.

On the second day a group A β -haemolytic streptococcus was isolated from the blood cultures. Penicillin was continued at an increased dose of 250 000 U four-hourly and ampicillin and gentamicin were withdrawn. The penicillin was given for 14 days. The child made an uneventful recovery and was discharged four weeks after admission.

The peripheral white cell count rose from $9.3 \times 10^9/l$ on admission to $44.3 \times 10^9/l$ (44 300/mm³) in seven days with 74 % polymorphs and evidence

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}

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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