first hour, haemoglobin 13.1 g/dl, and white cell count $11.5 \times 10^9/l$. Anti-nuclear and anti-heart-muscle antibodies were absent and complement concentration normal. Chest x-ray films showed slight cardiac enlargement, and on echocardiography a small amount of pericardial effusion was detected. Treatment with aspirin 2 g daily was started with rapid clinical improvement. Two weeks later the echocardiogram was normal.

He had first suffered from fever and chest pain two years previously; the symptoms had begun 10 days after an influenza vaccination with A/Texas/1/77(H_2N_2), A/USSR/9/77(H_1N_1), and B/Hong Kong 5/72 (Vaccigrip-Institut Marieux). A clinical diagnosis of acute benign pericarditis had been confirmed by echocardiography, and the symptoms had completely resolved after a month of steroid treatment.

Comment

The distinct time interval between the appearance of pericarditis and administration of the vaccine on two consecutive occasions suggests that the recurrent pericarditis was a direct consequence of the vaccinations. As the two vaccines were of different antigenic composition and no signs of autoimmune disease could be detected the exact mechanism of the pericarditis remains obscure. To the best of our knowledge pericarditis has not previously been reported as a complication of influenza vaccine.

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(Accepted 13 May 1981)

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Periampullary adenoma causing pancreatitis

Benign duodenal tumours are rarely of clinical importance, but those in the region of the ampulla may present with obstructive jaundice,¹ the preoperative diagnosis usually being pancreatic carcinoma. We report a case of an adenoma of the duodenal papilla which presented with acute pancreatitis.

Case report

A 68-year-old widow was admitted as an emergency after severe epigastric pain and vomiting for 20 hours. She gave a three-year history of upper abdominal pains, mostly after meals, which had become more severe during the preceding six months, when she had also lost 3 kg. Her epigastrium was very tender, and her serum amylase activity was raised at 6470 IU/l (normal range 86-268 IU/l). Results of liver function tests were normal apart from a raised serum alkaline phosphatase activity of 186 IU/l (normal range 22-92 IU/l). Plain abdominal radiographs and ultrasonography of her upper abdomen showed no abnormality. After three days' conservative management she was much improved, her serum amylase activity had fallen to 698 IU/l, and she went home. An oral cholecystogram three weeks later showed no abnormality, and her serum amylase activity was normal.

Two weeks later her symptoms returned, in particular epigastric pain, usually after meals with occasional vomiting. Findings of barium-meal examination were reported as normal, and examination of the stomach and duodenal bulb with the standard end-viewing gastroscope showed no abnormality. Her symptoms continued to fluctuate in severity over the next two months, and she lost 6 kg. Her serum alkaline phosphatase activity remained raised at 305 IU/l, but she was not jaundiced and at no time was a mass palpable in her epigastrium. A pancreatic cause for her symptoms was strongly suspected, and endoscopic retrograde cholangiopancreatography showed an irregular, mobile mass (2 cm in diameter) in the second part of the duodenum, in the region of the ampulla of Vater. Four biopsy specimens

were taken and these showed fragments of villous mucosa with no evidence of malignancy. Cannulation of the ducts was not possible owing to the mass. At laparotomy (8 July 1980) the duodenum was opened to confirm a tumour 2 cm in diameter arising from the papilla. The tumour was completely excised with diathermy, and the common bile duct and pancreatic ducts were reimplanted as an extended sphincteroplasty. Histology confirmed the mass to be a benign adenomatous polyp with the main ducts running through its centre.

The patient made a good recovery apart from a wound infection, her symptoms have gone, and all investigations have since shown no abnormality.

Comment

This is the first reported case to our knowledge of acute pancreatitis caused by an adenoma of the duodenal papilla. At no time was the patient jaundiced, though the serum alkaline phosphatase activity remained persistently raised. The long history of upper abdominal pain, weight loss, nausea, and vomiting is typical of periampullary adenomas, but the most consistent abnormality is usually jaundice attributed to varying degrees of biliary duct obstruction.

Fourteen cases of duodenal adenomas not affecting the periampullary region have been described; these were similarly associated with upper abdominal pain, nausea, and vomiting, and symptoms persisted in only four patients after excision of the tumour.² This suggests that the symptoms of periampullary adenomas may to some extent be explained by partial obstruction of the duodenum by the tumour.

Post-mortem studies have shown that the prevalence of periampullary adenomas in the elderly may be as high as 4%,³ of which most remain undetected clinically. Symptomatic cases tend to occur in middle to late life with no sex predilection.² Barium studies and cholangiography, though informative, rarely provide a clear diagnosis. Without doubt the best diagnostic tool is endoscopic retrograde cholangiopancreatography, which affords an opportunity to take multiple biopsy specimens. Complete excision is recommended since the condition is premalignant.^{2, 4}

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(Accepted 2 June 1981)

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Pneumocystis carinii pneumonia as presenting feature of lymphoma

Pneumocystis carinii is an opportunistic pathogen that has previously been reported to occur only in patients who are immunosuppressed, either therapeutically to prevent transplant rejection or as a result of treatment of a myeloproliferative disorder. We report on a patient, who presented with pneumocystis pneumonia and had oral candidiasis as the only suggestive evidence of immune depression. Only after histological examination of necropsy specimens was lymphoma diagnosed.

Case report

A 53-year-old engineer who had been working in Saudi Arabia for eight years had deteriorated in health over the past 18 months. Shortly after

returning to work after a holiday in England he was repatriated severely ill. He had had a progressive dry cough, drowsiness, anorexia, weight loss, and recurring fevers. Examination showed drowsiness and confusion, tachypnoea, tachycardia, and fever of 40°C. Apart from mild oral candidiasis examination showed no abnormality and there was no lymphadenopathy or splenomegaly.

He underwent full investigations with negative results apart from a mild leucopenia (white cell count $3.8 \times 10^9/l$ with 93% neutrophils, 7% lymphocytes). Chest radiography showed slight diffuse upper-zone infiltration. Immunoglobulin and immune complex concentrations, T-cell assays, and repeated marrow biopsy specimens were all normal. Appearances at fiberoptic bronchoscopy were normal, but transbronchial biopsy disclosed cysts of *P. carinii*. He was treated with co-trimoxazole without therapeutic response and on the 12th day developed profound pancytopenia necessitating a change to pentamidine. Again there was no response and he gradually deteriorated and died a week later.

At necropsy there was confluent consolidation of the left lung and patchy consolidation in all the lobes of the right. The reticuloendothelial system was normal macroscopically, with no enlargement of glands or the spleen. Microscopy confirmed *P. carinii* to be the infective agent in the lungs, and examination of the spleen and lymph nodes showed a histiocytic medullary reticulosis.

Comment

P. carinii is a protozoan parasite with some fungal features initially classified in 1914.¹ It has a particular affinity for pulmonary alveoli, causing a pneumonic illness. Impaired host resistance appears to be important in the development of pneumonia as all previous cases have been in immunosuppressed patients. The organism occurs widely, and humoral antibodies to pneumocystis may be found in a high proportion of normal children.²

Diagnosis of *P. carinii* infection requires identification of the organism in lung tissue, and transbronchial biopsy via a fiberoptic bronchoscope provides a way of obtaining this without the disadvantages of open-lung biopsy. Treatment with pentamidine isethionate has been successful but has a high incidence of toxic reactions,³ and more recently co-trimoxazole has been advocated as the treatment of choice.⁴

Lack of evidence of immunosuppression should not necessarily exclude the possibility of infection with *P. carinii*, and this case emphasises the need to perform transbronchial biopsy in a pneumonia of unknown aetiology.

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(Accepted 2 June 1981)

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Richter's hernia: an unrecognised complication of chronic ambulatory peritoneal dialysis

Chronic ambulatory peritoneal dialysis has gained widespread acceptance as a method of treating patients with end-stage renal failure. The technique was initially described by Popovich *et al* in 1976,¹ and the first report of its use in a large series in the United Kingdom was recently published²; increasing use seems certain. Although peritonitis is by far the most common complication, other problems are likely to become evident as experience increases. We use chronic ambulatory peritoneal dialysis in patients with chronic renal failure as part of an integrated dialysis and transplant programme³ and describe here a rare complication of this treatment.

Case report

In 1976 a 26-year-old woman presented with malignant hypertension and chronic renal failure. Her medical history included tuberculous meningitis at the age of 4 years, which had resulted in mild mental retardation, and pulmonary tuberculosis at age 19. Despite reasonable control of the hypertension her renal function continued to deteriorate, and in March 1980 chronic ambulatory peritoneal dialysis was started. A Tenckhoff catheter was inserted under general anaesthesia through a midline abdominal incision and the greater omentum excised to prevent blockage of the catheter. Dialysis was started a week later using the Travenol system and four 2 l exchanges each day.

Her general health improved, haemoglobin concentration returned to normal, and serum biochemistry was well controlled with a mean serum creatinine concentration of 800 $\mu\text{mol/l}$ (9 mg/100 ml). Four episodes of peritonitis were successfully treated with intraperitoneal antibiotics. Eight months after the start of dialysis several brief episodes of upper abdominal pain and vomiting were noted. Symptoms settled rapidly on two successive

Richter's hernia with dilated small bowel proximally and collapsed bowel distally.

admissions to hospital and there was no evidence of peritonitis. On the first admission, however, a tender mass, 2 cm in diameter and under the laparotomy scar, was noted by two observers but resolved spontaneously within a few hours of admission.

In late November she was readmitted with central abdominal pain and copious vomiting. Erect and supine abdominal x-ray films were normal, and peritonitis was diagnosed on the basis of slightly cloudy peritoneal fluid and a peripheral leucocytosis. Intraperitoneal cefuroxime was started and 50 mg pethidine administered intramuscularly to relieve the pain. Shortly thereafter she fainted, inhaled gastric contents, and died despite intensive resuscitation. Postmortem examination showed small-bowel obstruction at the level of the distal jejunum due to a Richter's hernia through a 1 cm defect in the anterior abdominal wall at the upper end of the laparotomy scar (figure). The jejunum at this site showed early necrotic change. The immediate cause of death was severe bilateral aspiration pneumonia.

Comment

As peritonitis is by far the most common cause of abdominal pain in patients receiving chronic ambulatory peritoneal dialysis it is tempting to ascribe any episode of pain without an immediately obvious origin to peritonitis. This case shows the danger of such an approach. We believe that this is the first description of a Richter's hernia as a specific complication of catheter insertion.

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(Accepted 2 June 1981)

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