first hour, haemoglobin 13·1 g/dl, and white cell count 11·5 × $10^9$/l. Antinuclear 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/($H_3N_2$), 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.


[1] Hildebrandt HM, Maassab HF, Willis PW. Influenza virus pericarditis. *Am J Dis Child* 1962;**104**:179.

[2] White CS III, Adles WH, McGann VG. Repeated immunizations: possible adverse effects. *Ann Intern Med* 1974;**81**:594.

[3] Center for Disease Control. Influenza vaccine 1980-1981. *Ann Intern Med* 1980;**93**:466-8.

[4] Clinical studies of influenza vaccines 1976: a conference held at the National Institutes of Health, Bethesda, Maryland, 20-21 January 1977. *J Infect Dis* 1977;**136**, suppl:S341-742.


(*Accepted 13 May 1981*)


**Beilinson Medical Centre, Petach Tikva, Tel Aviv University Medical School, Tel Aviv, Israel**

JONATHAN J STREIFLER, MD, junior registrar
SHLOMO DUX, MD, senior registrar
MOSHE GARTY, MD, deputy chief, lecturer
JOSEPH B ROSENFELD, MD, chief of department


# 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]


[1] George P. Disorders of the extrahepatic bile ducts. *Clin Gastroenterol* 1973;**2**:127-46.

[2] Charles RN, Kelley ML, Campeti F. Primary duodenal tumours. *Arch Intern Med* 1963;**111**:23-33.

[3] Baggenstross AH. Major duodenal papilla. *Archives of Pathology (Chicago)* 1938;**26**:853.

[4] Seifert E. Endoscopic polypectomy: upper gastrointestinal tract. *Clin Gastroenterol* 1978;**7**:749-55.


(*Accepted 2 June 1981*)


**Surgical Unit, King's Mill Hospital, Sutton-in-Ashfield, Nottinghamshire**

S H WHITE, BM, BS, surgical house officer (present appointment: house officer, City Hospital, Nottingham)
N A NAZARIAN, FRCS, surgical registrar
A McEWEN SMITH, FRCS ED, consultant surgeon

**Department of Surgery, City Hospital, Nottingham**

T W BALFOUR, FRCS, consultant surgeon and senior lecturer in surgery


# 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