Pleural effusions associated with pancreaticopleural fistula

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Abstract
Two cases of pancreaticopleural fistula are reported. The delay in diagnosis and extensive investigations performed highlighted the need for pleural fluid amylase estimation at an early stage.

(Thorax 1993;48:867–868)

Pancreaticopleural fistula is an uncommon cause of large, recurrent, predominantly left sided pleural effusions. When the underlying pancreatic disease is asymptomatic the diagnosis may not be considered and, as a consequence, extensive unnecessary investigations may ensue. We report two cases in which long delays in diagnosis (four and 13 months) of the pancreaticopleural fistula occurred. These cases emphasise the importance of considering the diagnosis and of estimating the pleural amylase content.

Case 1
A 50 year old man presented to another hospital with exertional dyspnoea and a four month history of weight loss. There was no previous history to suggest pancreatic disease but he drank 40 units of alcohol per week.

Examination indicated a large left sided pleural effusion which was confirmed by chest radiography. The pleural fluid had a protein content of 87 g/l and both cytological and microbiological investigations were negative. The pleural biopsy specimen was normal, as were the results of bronchosopic examination. A computed tomographic (CT) scan of the chest revealed thickening of the left hemidiaphragm, and a CT scan of the abdomen revealed left para-aortic lymphadenopathy. Ultrasound guided biopsy of the lymph nodes showed reactive changes only. A bone marrow aspirate and trephine sample were normal.
Over a 12 month follow up period he had a small persistent effusion. Further investigations were initiated following the development of a right pleural effusion. The pleural fluid was dark and straw-coloured and was assessed for amylase content. Pancreatic hyperamylasaemia (>20 000 units/l) was confirmed. Concurrent serum amylase estimation was normal (159 units/l). An abdominal radiograph, upper abdominal ultrasonographic scan, and a CT scan of the abdomen were normal. He was then transferred to this hospital where endoscopic retrograde pancreatography confirmed a pancreaticopleural fistula with strictureing of the pancreatic duct (fig). A distal pancreatectomy was performed and the patient recovered uneventfully.

**Case 2**

A 38 year old man was admitted to another hospital with right sided chest pain and dyspnoea. There was a history of alcohol excess and three proven episodes of pancreatitis over eight years. At the time of presentation there were no symptoms referable to the pancreas. Examination revealed a right pleural effusion which was confirmed by chest radiography.

He had a normochromic normocytic anaemia with normal liver function tests and serum amylase. The pleural fluid was clear and straw-coloured, its protein content was 47 g/l, and both cytological and microbiological investigations were normal. Examination of pleural biopsy samples revealed reactive changes only. A CT scan of the chest and upper abdominal ultrasonography were normal. He was transferred to another hospital for further investigation where pleural fluid hyperamylasaemia (>20 000 units/l pancreatic amylase) was confirmed. Concurrent serum amylase estimation was normal (140 units/l). Abdominal radiography, repeat ultrasonography, and a CT scan of the abdomen confirmed features of chronic pancreatitis. He was referred to this hospital where endoscopic retrograde pancreatography performed four months after the initial presentation confirmed a pancreaticopleural fistula with a proximal pancreatic duct stricture.

Having failed to respond to medical treatment (total parenteral nutrition and octreotide), he underwent pancreatecojeunal anastomosis and he remains well without recurrence of the effusion.

**Discussion**

The two cases illustrate the delay and unnecessary investigations that may occur when the diagnosis of pancreaticopleural fistula is not considered.

Chest radiographic abnormalities occur quite frequently in the course of upper abdominal disease, particularly subphrenic abscess.

Acute pancreatitis may lead to atelectasis, pneumonitis, the adult respiratory distress syndrome, and pleural effusions. Effusions arising from acute pancreatitis are usually small, left sided and self limiting. The incidence of pleural effusions in acute pancreatitis is between 3% and 17%.1

In chronic pancreatitis, as a consequence of fistula and pancreatic pseudocyst formation, extremely large effusions may be seen which frequently recur following drainage. These result from a fistulous tract between the pancreas and the pleural space or by direct extension of a pancreatic pseudocyst through the mediastinum.

Pleural effusions caused by chronic pancreatitis are usually left sided but may be right sided or bilateral. The amylase content tends to be significantly elevated and the effusion is usually an exudate. The pleural amylase level may be elevated in chronic pleural effusions that are associated with an oesophageal perforation or primary lung neoplasms, but in these instances the amylase is of the S (salivary) type rather than the pancreatic (P) type associated with pancreatic disease.

Some 40–50% of patients with chronic pancreatitis and large effusions respond to conservative management with intercostal drainage and total parenteral nutrition, somatostatin or its analogue octreotide.2 Persistence or recurrence of the effusion(s) is an indication for pancreatic surgery. This is dependent upon delineation of pancreatic anatomy, so endoscopic retrograde pancreatography is mandatory prior to surgery.

Adequate pancreatography may require selective duct cannulation, particularly in the presence of tight strictureing. About 80–95% will have a satisfactory outcome from surgical intervention with a mortality rate of 3%.3 The overall mortality rate from pancreaticopleural fistula is about 5%.2

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