Pancreaticopleural Fistula*

Diagnosis With Magnetic Resonance Pancreatography

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Pancreaticopleural fistula secondary to chronic pancreatitis is a rare cause of recurrent pleural effusion. The demonstration of the fistula with endoscopic retrograde pancreatography and CT is invasive or limited. We report in two patients the use of magnetic resonance pancreatography as a noninvasive alternative to endoscopic retrograde pancreatography for the diagnosis of pancreaticopleural fistula. (CHEST 2000; 117:912–914)

Key words: diseases; fistula; MRI; pancreas; pancreaticopleural

Abbreviation: MR = magnetic resonance

Pancreaticopleural fistula is a rare complication of chronic pancreatitis. It results from posterior disruption of the pancreatic duct into the retroperitoneal space, leading to the formation of a fistulous tract between the pancreas and the pleural cavity through the aortic or esophageal hiatus. Clinical presentation is often misleading, as respiratory rather than abdominal symptoms predominate. Markedly elevated pleural fluid amylase level is the most important laboratory finding. The pancreatic fistula can be demonstrated by endoscopic retrograde pancreatography. However, selective duct cannulation is invasive, and the entire anatomy of the fistula will not always be delineated. Therefore, there is a need for an accurate and noninvasive procedure that could substitute for diagnostic endoscopic retrograde pancreatography. Visualization of pancreaticopleural fistula in chronic pancreatitis has been reported with CT but the sensitivity of this imaging method for the demonstration of fistulas is low.

We report two cases of pancreaticopleural fistula demonstrated by magnetic resonance (MR) pancreatography.

CASE REPORTS

Case 1

A 50-year-old man with a history of chronic alcoholic pancreatitis complained of cough, dyspnea, and left-sided chest pain. Physical examination revealed a left-sided pleural effusion that was confirmed by chest radiography. Pleural fluid amylase was 30,750 IU/L; total protein, 4.5 g/dL; glucose, 137 mg/dL; and WBC count, 86 cells/µL. CT of the chest and abdomen showed the left-sided pleural effusion and characteristic features of chronic pancreatitis, including parenchymal atrophy with numerous calcifications, and a caudal pseudocyst. Endoscopic retrograde pancreatography demonstrated dilatation of the main pancreatic duct with intraductal calcifications, and leak of contrast material from the tail of the pancreas toward the left pleural cavity.

Having failed to respond to medical treatment including total parenteral nutrition and somatostatin infusion, the patient was transferred to our institution for surgical therapy. MRI was performed for preoperative assessment of the pancreatic anatomy. First, conventional T1- and T2-weighted images of the upper abdomen were obtained. Next, MR pancreatography was performed with a breath-hold, single-shot, fast spin-echo sequence using two acquisition techniques: sequential single-slice acquisition with thick slices (effective echo time, 871 ms; acquisition time, 2 s; slice thickness, 20 mm) and multislice acquisition with thin slices (effective echo-time, 94 ms; acquisition time, 30 s; slice thickness, 3 mm). Single-slice acquisition was obtained in the coronal and coronal oblique planes, and multislice acquisition in the coronal and transverse planes. Coronal projectional images were constructed with the maximal intensity projection algorithm. In addition to features of chronic pancreatitis, MR pancreatography demonstrated the pancreaticopleural fistula that extended from the tail of the pancreas to the left pleural cavity (Fig 1). The presence of the pancreaticopleural fistula was confirmed at surgery, and the patient underwent distal pancreatectomy and longitudinal pancreaticojunostomy.

Case 2

A 32-year-old man with a history of chronic alcoholic pancreatitis was admitted for progressive dyspnea. Physical examination was suggestive of a large left-sided pleural effusion that was confirmed by chest radiography. Pleural fluid amylase measurement was 10,800 IU/L; total protein, 4.3 g/dL; glucose, 43 mg/dL; and WBC count, 6,880 cells/µL. CT of the chest and abdomen showed the left-sided pleural effusion and features of chronic pancreatitis, including parenchymal atrophy with multiple calcifications and dilatation of the main pancreatic duct. Furthermore, a subtle infiltration of the retroperitoneal fat between the pancreas and the crus of the left hemidiaphragm was considered as a possible sign of a fistulous tract between the pancreas and the left pleural cavity. Endoscopic retrograde pancreatography demonstrated a stenosis at the distal part of the main pancreatic duct and a 4-mm obstructive stone inside the duct. Cannulation of the minor papilla resulted in opacification of a dilated main pancreatic duct proximal to the obstructive stone. No fistula could be demonstrated.

MRI was performed, and conventional T1- and T2-weighted
images of the upper abdomen were obtained. Next, MR pancreatography was performed using sequential single-slice acquisitions (effective echo-time, 1,100 ms; acquisition time, 5 s; slice thickness, 50 mm) in the coronal plane, and multislice acquisitions (effective echo-time, 90 ms; acquisition time, 11 s; slice thickness, 4 mm) in the transverse and coronal planes. MR pancreatography demonstrated a pancreaticopleural fistula arising at the level of the head of the pancreas toward the left pleural cavity (Fig 2). Endoscopic retrograde pancreatography was repeated and the fistula was confirmed. After sphincterotomy of the minor papilla, a pancreatic stent was placed and the patient received somatostatin, 200 μg/h. The evolution was uneventful with complete regression of the pleural effusion.

**DISCUSSION**

Pleuropulmonary complications are relatively frequent in patients with acute pancreatitis. Pleural effusions are reported to occur in 4 to 17% of patients. The effusion is commonly small, transient, and left sided. In contrast, pleural effusions are less common in patients with chronic pancreatitis. The effusion is often large and recurrent, arising from a fistulous tract between the pancreas and the pleural cavity, with or without pseudocyst formation. It usually predominates on the left side, but may be right sided or bilateral. The pleural fluid is typically an exudate with very high amylase content. The causes of an elevated amylase level in pleural fluid include acute pancreatitis, esophageal perforation, and various types of tumors such as lung carcinoma and female genital tract carcinoma. Clinical presentation, radiographic studies, and determination of the amylase isoenzyme levels usually confirm the diagnosis.

Endoscopic retrograde pancreatography and CT have been used for the diagnosis of pancreaticopleural fistula. Endoscopic retrograde pancreatography is very useful for imaging the pancreatic ductal anatomy, and can demonstrate the fistulous tract that extends to the pleural cavity. CT is recommended to show pancreatic parenchymal atrophy, in addition to dilatation of the pancreatic ducts, calcifications, and pseudocysts. Furthermore, the fistula can sometimes be revealed. However, endoscopic retrograde pancreatography and especially CT may fail to demonstrate the entire anatomy of the pancreaticopleural fistula. In addition, endoscopic retrograde pancreatography is an invasive procedure with a small but substantial complication rate, including acute pancreatitis, sepsis, and bleeding.

MR pancreatography is a noninvasive imaging method to assess pancreatic diseases. MR pancreatography is based on the acquisition of heavily T2-weighted images, which result in high signal intensity of static or slowly flowing fluids. Chronic pancreatitis may be consistently evaluated with this imaging method. MR pancreatography can depict parenchymal and ductal structural changes, but also extrapancreatic complications, including pancreaticopleural fistula as shown in our cases. MR pancreatography provides an overview of the fistulous tract, which appears as a high-signal-intensity structure. No contrast material is injected, and therefore there is no risk of infection. Site of fistulization and anatomic relationships are precisely defined, suggesting that diagnostic endoscopic retrograde pancreatography should be performed only in confusing cases.

In conclusion, MR pancreatography can show pancreaticopleural fistulas, and may be a noninvasive alternative to diagnostic endoscopic retrograde pancreatography.

**REFERENCES**


**Figure 1.** A 50-year-old man with chronic pancreatitis. Coronal projectional MR pancreatographic image shows dilatation of the main pancreatic duct with intraductal calcifications (arrowheads), and pancreaticopleural fistula (arrow) that extends to the left pleural cavity. GB = gallbladder; St = stomach; LPE = left-sided pleural effusion; D = duodenum.

**Figure 2.** A 32-year-old man with chronic pancreatitis. Coronal MR pancreatographic image shows pancreaticopleural fistula (arrow) arising at the level of the head of the pancreas (arrowheads). See Figure 1 legend for abbreviations.
Oxidant/antioxidant imbalance can occur in obstructive airways disease as a result of ongoing inflammation. Glutathione (GSH) plays a major role in pulmonary antioxidant protection. As an alternative or complement to anti-inflammatory therapy, augmenting antioxidant protection could diminish the effects of inflammation. We describe a case of a patient who had obstructive lung disease responsive to corticosteroids, and low whole blood GSH levels. After 1 month of supplementation with a whey-based oral supplement designed to provide GSH precursors, whole blood GSH levels and pulmonary function increased significantly and dramatically. The potential for such supplementation in pulmonary inflammatory conditions deserves further study.

Key words: glutathione; inflammation; oxidative stress; supplementation

Abbreviations: ELF = epithelial lining fluid; GSH = glutathione; GSH-Px = glutathione peroxidase; PFT = pulmonary function test; ROS = reactive oxygen species

Evidence of oxidant/antioxidant imbalance has been demonstrated in obstructive airway disease. Continued lung inflammation with the mobilization and activation of neutrophils, macrophages, and eosinophils and their release of free oxygen radicals and other reactive oxygen species (ROS) is a source of oxidative stress. In addition to the direct effects of such ROS on cell membranes, DNA, and proteins, breakdown products act as signals perpetuating the inflammatory cascade. Glutathione (GSH) and the GSH system play a key role in protecting against the effects of ROS. Modulation of the oxidant/antioxidant status in obstructive airway disease, primarily aimed at enhancement of the GSH system, has been limited by difficulties in delivery of an effective substrate. We describe the response to an oral, whey-based supplement designed to supply GSH precursors.

Case Report

A 40-year-old woman of North African origin was followed by the pulmonology service at a tertiary care hospital in 1997. Her medical history was significant for Hodgkin’s lymphoma, diagnosed 27 years earlier and treated with radiation and chemotherapy. She had a 25-pack-year smoking history and had quit smoking in 1994. In 1995, she received a diagnosis of mild valvular heart disease (aortic and mitral regurgitation). At that time (time 1), pulmonary function tests (PFTs) suggested mild airflow obstruction (Fig 1); bronchodilators were not prescribed. In 1997 (time 2), she was admitted to hospital with a virally induced exacerbation of obstructive lung disease as well as mild heart failure. She improved with diuretics, bronchodilators (salbutamol and ipratropium bromide), and oral prednisone, 20 mg/d. She was discharged taking a tapering course of prednisone.

When seen in follow-up 1 week later (time 3), she had suffered an exacerbation of her respiratory symptoms (shortness of breath, wheeze, chest tightness, and excessive mucus production) coincident with cessation of prednisone. Prednisone was prescribed again.

She returned 2 weeks later (time 4) with significant symptomatic improvement while still taking systemic corticosteroids and regular bronchodilators (salbutamol metered-dose inhaler and ipratropium bromide metered-dose inhaler qid). An attempt was made to discontinue prednisone. When the patient was seen 1 month later (time 5), her symptoms had returned. At that time, review of her history revealed no environmental insult that could account for her deterioration. Additionally, serum IgE was 52 kU/L (laboratory control, 0 to 100 kU/L), and both allergen skin testing and Aspergillus precipitin testing were negative. A further course of oral prednisone was prescribed (40 mg/d initially; tapering over 1 month).

Four months later (time 6), the patient returned to clinic independently, having begun taking Immunocal (Immunotec Research Ltd; Vaudreuil, Quebec, Canada), a whey-based protein supplement (10 g bid), 1 month before. She had heard that the product could be helpful in inflammatory conditions, and had started taking the product of her own accord. She reported a remarkable improvement in her respiratory status and had discontinued all inhalers and steroids, and was not taking any other supplements, medications, or over-the-counter therapies. She was asked to discontinue the Immunocal, and within 3 months her symptoms returned. PFTs were performed at this time (time 7). She then restarted Immunocal of her own accord, and 1 month later (time 8), PFTs were again assessed. Additionally, whole blood GSH levels were measured before and 1 month after therapy was reinitiated, using a modification of the method previously described. Again, a remarkable improvement in both symptoms and PFTs was noted (Fig 1). In addition, the total lung capacity increased from 3.91 L at time 7 to 5.00 L at time 8, and the residual volume/total lung capacity ratio fell from 33 to...