Case Report

Successful Endoscopic Treatment of Mediastinal Pseudocysts

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ABSTRACT

Mediastinal pseudocysts can pose a diagnostic and therapeutic challenge to the clinician and surgeon. Recognizing their presence and instituting appropriate therapy can reduce morbidity and mortality. This report describes unusual clinical features in a patient presenting with multiple mediastinal pseudocysts due to pancreatic duct leak secondary to pancreatic duct stenosis and an entrapment of a pancreatic duct stone. Successful endoscopic therapy averted the need for surgery.

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INTRODUCTION
Mediastinal pancreatic pseudocyst is a rare complication of acute and chronic pancreatitis and pancreatic trauma. Treatment options for mediastinal pseudocysts include conservative management with surgery reserved for refractory or progressive disease. On rare occasions, spontaneous resolution with conservative therapy has occurred. Medical management includes parenteral or enteral nutrition through nasal jejunal tube feeding and somatostatin or its analogues. Transpapillary pancreatic stent placement with or without initial transpapillary duct drainage has been shown to be a useful nonsurgical modality for definitive treatment of this condition. We report our experience in a patient with multiple pancreatic pseudocysts, cardiac compression by a pancreatic pseudocyst, possible central lymphatic blockade due to mediastinal pseudocysts and resolution with endoscopic transpapillary stent placement and somatostatin LA.

CASE REPORT
A 69-year-old male with a history of recurrent alcoholic pancreatitis presented with a 2-week history of progressive dyspnea, bilateral pleuritic chest pain, and mild dysphagia with solids. Vital signs included body temperature of 36.5°C, blood pressure of 120/60 mmHg, and pulse of 68 bpm. Physical examination revealed diminished breath sounds and dullness to percussion bilaterally involving the lower one-third of the posterior lung bases.

A chest radiograph showed bilateral pleural effusions and interstitial pulmonary edema (figure 1). Echocardiography showed normal left and right ventricular size and function, mild pulmonary hypertension, with mild tricuspid and mitral regurgitation. Initial computed tomography (CT) of the chest and abdomen showed bilateral pleural effusions, a 4.2 cm x 1.7 cm periesophageal fluid collection, mass effect on the atrium and esophagus, a pseudocyst measuring 3.7 cm x 2.4 cm in the body of the pancreas, a large pancreatic calcified duct stone, and multiple pancreatic calcifications in the body of the pancreas. Follow-up CT scan one week later showed persistent periesophageal cyst, fluid tracking along the entire length of the thoracic esophagus with three small fluid collections ranging in size from 1.0 to 2.0 cm near the gastroesophageal junction, and increased dilation of the main pancreatic duct at the level of the body and tail measuring up to 0.9 cm (figure 2). Right-sided thoracentesis obtained 865 cc of yellow, minimally hazy fluid with 424 red blood cells/µL, 1730 white blood cells/µL, 44% neutrophils, 1% eosinophils, 39% monocytes, 13% lymphocytes, and 3% mesothelial cells. No organisms or malignant cells were identified. The pleural fluid amylase was 9327 U/L, lipase 9570 U/L, cholesterol 67 mg/dL, pH 7.45, PCO2 32 mmHg, PaO2 46 mmHg, and HCO3 22 mmol/L. Serum chemistry was significant for a serum amylase of 1483 U/L and lipase of 1778 U/L.

Endoscopic retrograde cholangiopancreatography (ERCP) revealed a 7 mm elliptical, free-floating stone within the main pancreatic duct in the body of the pancreas (figure 3). The duct in the body and tail of the pancreas was dilated to approximately 8 mm. Injection of contrast media revealed changes of chronic pancreatitis in the head, body and tail. The contrast freely refluxed into the common bile duct, normally calibrated to 3 mm, with leakage of contrast media in the tail of the pancreas. A maximal, wire-guided pancreatic traction sphincterotomy was performed.

Figure 1. Chest radiograph shows bilateral interstitial edema and pleural effusions.

Figure 2. Computed tomography of the chest. There are multiple small fluid collections representing pancreatic pseudocysts with mass effect on the atrium and large bilateral pleural effusions.
Initial attempts to extract the stone using a 12 mm balloon and a Flower basket alongside the guide were unsuccessful. A 7 French x 7 cm stent was placed in the main pancreatic duct proximal to the stone. Enteral hyperalimentation through a nasal jejunal tube and somatostatin LA once weekly for 6 weeks was instituted.

ERCP performed 7 days later revealed the leak persisting in the pancreatic tail. The stone was successfully removed using a 12 mm biliary balloon, and the stent replaced with an 8.5 French x 10 cm Amsterdam stent (figure 4).

CT scan of the abdomen and pelvis performed 41 days after the first ERCP showed resolution of the mediastinal pseudocysts, pleural effusions, and dilated pancreatic duct. A small 1.0 cm residual pseudocyst was identified in the pancreatic tail (figure 5). The pancreatic stent was subsequently removed, and the patient made a complete recovery that persisted at the 6-month follow-up.

DISCUSSION
Mediastinal pancreatic pseudocyst is a rare thoracic complication of pancreatitis. In acute pancreatitis, the pseudocyst represents the inflammatory response to tissue injury resulting in the formation of fibrous or granulation tissue surrounding an inflammatory exudate of fluid. However, in chronic pancreatitis the pseudocyst arises in the setting of ductal obstruction with subsequent disruption of the pancreatic duct due to increased intraductal pressure. Pancreatic pseudocysts are typically formed and confined to the lesser omental sac. Complications related to pseudocysts include hemorrhage, infection, fistulization, extension, or rupture.

Pathophysiology
It is postulated that early in the course, pancreatic fluid tracts from the retroperitoneal space into the mediastinum through the esophageal or aortic hiatus and rarely through foramen of Morgagni, directly through the diaphragm, or inferior vena cava hiatus. The location of the pseudocyst in the posterior mediastinum surrounding the esophagus suggested that the pancreatic fluid traversed the esophageal hiatus. The presence of multiple cysts surrounding the esophagus supports the theory that pancreatic proteolytic enzymes transverse the mediastinal compartment. Later in the course, a matured fistula tract may develop from the
Diagnosis

The diagnosis of mediastinal pancreatic pseudocysts should be suspected in the setting of a clinical history of pancreatitis, a chest radiograph showing a space-occupying mass in the posterior or middle mediastinum with an associated pleural effusion, and the finding of a thin-walled, low, attenuated cystic mass in the lower part of the posterior mediastinum on chest CT. Definitive diagnosis of mediastinal pancreatic pseudocysts is made either by demonstrating continuity with the pancreas structures or analysis of cyst content. CT is the diagnostic test of choice compared to ultrasound since it is a more sensitive method for imaging mediastinal masses. The finding of an elevated amylase in the pleural fluid, as in this case, also helps to confirm the diagnosis. In cases where there is no communication with the pancreas, differentiating mediastinal pseudocysts from other causes of mediastinal cysts can be a diagnostic challenge. Magnetic resonance imaging (MRI) and magnetic resonance cholangiopancreatography (MRCP) are helpful in such cases as they can identify the connection between the mediastinal and abdominal pseudocyst that may not be evident on a CT scan. MRCP has similar sensitivity to ERCP and has the advantage of producing images of the ducts in their natural state, because it does not involve the distention of the ducts by the injected contrast media.

Symptoms

Symptoms due to pancreatic mediastinal pseudocysts reflect either compression or invasion of adjacent structures and include chest or abdominal pain, dyspnea, and/or dysphagia. Retrocardiac compression by posterior mediastinal pseudocysts can result in elevated filling pressure and possibly congestive heart failure. The findings of left atrial compression by the mediastinal pseudocysts, interstitial pulmonary congestion, bilateral pleural effusions, and normal spirometry in the presence of severely reduced diffusion capacity for carbon monoxide and hypoxemia is suggestive of the possible contribution of central lymphatic blockade and left atrial compression due to mediastinal pseudocysts. Abdominal symptoms may be characteristically absent in patients who develop a mediastinal pseudocyst because the formation of a pancreaticomedial fistula allows for decompression of the pancreatic pseudocyst into the lower pressure thoracic cavity.

Complications

The etiology of the exudative pleural effusion is believed to be multifactorial, including obstructive lymphatic interstitial edema due to compression of lymphatic flow from the lungs, left atrial compression from the pseudocyst, trans-diaphragmatic extension of pancreatic fluid through the diaphragm into the pleural space, and sympathetic pleural effusion caused by a secondary inflammatory response resulting from pancreatic fluid irritating the diaphragmatic pleural. Other causes include a sinus tract running from the pancreas to the pleural space or bronchi or rupture of mediastinal pseudocysts into the pleural space. Chronic pancreatic pleural effusions typically require drainage due to their large size and their tendency to become organized and complicated by the development of a bronchopleural fistula or empyema.

Management

Approach to management comes primarily from case reports and case series and is dictated by the severity of symptoms caused by the mediastinal pseudocyst. There are several approaches including conservative, medical treatments, endoscopic stenting, external or internal drainage, and surgery. Medical management is supportive and includes bowel rest, nutritional support, repeated thoracentesis, and somatostatin analogues. This is particularly important early in the course when there are signs of acute pancreatic inflammation to allow the pseudocysts to mature for operative intervention. Regression of the pseudocysts, similar to our case, has been reported with bowel rest, nutritional support and the use of octreotide.

Nasal jejunal enteral feeding has been shown to be a safe and effective method for providing nutritional support to patients with acute or chronic pancreatitis. By reducing the volume of pancreatic secretion, somatostatin or its analogues may facilitate or accelerate the closure of mediastinal pancreatic fistulas. Other nonoperative strategies include CT-guided percutaneous drainage, endoscopic transmural drainage, or for cases similar to ours with pancreatic duct disruption, ERCP with transpapillary duct drainage and endoscopic stent placement. Endoscopic ultrasonography can be used to guide transmural internal drainage of pseudocysts. Patients whose symptoms progress or fail to resolve or develop infection despite medical treatment should undergo surgical intervention.
Surgery should be considered in symptomatic patients if there are associated complications such as infection, obstruction, rupture, or hemorrhage. Approaches include cystogastrostomy or cystojejunostomy, Roux-en-Y pancreaticocolodenostomy, pancreaticojejunostomy, or rarely external percutaneous drainage.8,26

Transpapillary stent placement is an attractive therapeutic option that can be used in patients with pancreatic leakage due to a pancreatic duct leak or disruption. Stent placement is also useful therapeutically in that it can be used to facilitate stone extraction or passage or to dilate a stricture. These nonoperative approaches can be associated with a lower morbidity compared to traditional surgical interventions, although formal comparative studies have not been performed.

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