

Not Your Everyday Case of Acute Pancreatitis: A Rare Complication of a Common Diagnosis

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Abstract

Pancreaticopericardial fistula is an extremely rare complication of chronic pancreatitis and is virtually unheard of in the setting of acute pancreatitis. A 67-year-old male presented with acute pancreatitis complicated by pancreaticopericardial fistulization. The patient's initial presentation was consistent with tamponade physiology. Computed tomography demonstrated a gas-containing pericardial fluid collection extending into the anterior upper abdomen and ill-defined fluid collections within the pancreas representing necrosis with probable infection. Surgical exploration of the window revealed a pancreatic pericardial fistula and an infected window. The patient ultimately passed away from septic shock. We present this rare complication of pancreatitis and review the relevant literature.

Introduction

A pancreatic fistula is an abnormal communication between the pancreas and other organs, which results from disruption of pancreatic ducts and subsequent leakage of pancreatic enzymes. Pancreatic fistulas can either be external, in which case the communication is with the skin, or internal, with the most common sequela being the development of a pancreatic pseudocyst. Other complications depend on the flow of secretions from a disrupted pancreatic duct or leakage from a pseudocyst. If a pancreatic duct is disrupted such that pancreatic enzymes leak anteriorly into the peritoneal cavity, the result is pancreatic ascites. Similarly, in the event of a posterior disruption, pancreatic enzymes leak into the mediastinum via either the aortic or esophageal hiatus, resulting in a mediastinal pseudocyst if contained. If uncontained, however, pancreatic enzymes would leak into the mediastinum, pleural space, or, rarely, into the pericardium, resulting in enzymatic mediastinitis, pleural effusion, or pericardial effusion, respectively.^{1,2}

Case Report

A 67-year-old male with a complicated past medical history notable for severe rheumatoid arthritis, for which he was receiving tocilizumab, presented with increasing dyspnea and chest pain. Upon presentation, the patient was tachycardic, tachypneic, and hypotensive. His lab values were notable for an elevated white blood cell (WBC) count of $48.6 \times 10^3/\mu\text{L}$, lactic acid of 8.9 mmol/L, and liver enzymes with an SGOT (AST) of 2,552, SGPT (ALT) of 5,198. Electrocardiography showed sinus tachycardia with low voltage. The patient was emergently intubated in the emergency department (ED) after becoming unresponsive. Out of concern for tamponade, the patient underwent emergent pericardiocentesis with removal of 800 mL of cloudy, milky fluid with speckled white flecks. The patient was admitted to the intensive care unit (ICU) with a provisional diagnosis of rheumatoid pericardial effusion resulting in cardiac tamponade and septic shock in an immune-compromised host.

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The patient was aggressively resuscitated, requiring pressor support, and started on broad-spectrum antibiotics. After blood and urine cultures were negative, an indium scan delineated marked WBC accumulation within the upper abdomen, favoring involvement of the pancreas (Figure 1). The patient responded to broad-spectrum antibiotics and aggressive resuscitation and was discharged to a skilled nursing facility.

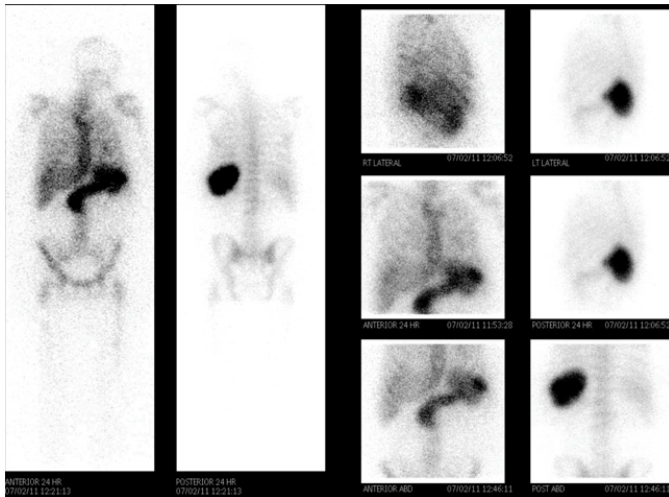


Figure 1. WBC Scan. Marked white blood cell accumulation within the upper abdomen. The anatomic distribution favors involvement of the transverse colon, pancreas, or stomach/proximal duodenum.

A few weeks following discharge, the patient returned to the ED with recurrent shortness of breath and was noted to have a moderate pericardial effusion on echocardiography without tamponade. A decision was made to place a pericardial window. Shortly after the procedure, the patient began to experience generalized abdominal and back pain with recurrent leukocytosis. Analysis of the pericardial fluid showed an elevated lipase at 24,000 U/L. A subsequent CT demonstrated a gas-containing fluid collection anterior to the heart, contiguous with the anterior upper abdomen. There were fluid collections within the pancreatic head and neck, raising the possibility of necrosis and superimposed infection (Figure 2). Surgical exploration of the subxiphoid window revealed a pancreaticopericardial fistula and an infected pericardial window (Figure 3). A decision was made not to close the fistula but rather irrigate the pericardial space and place a drain.

The patient became increasingly jaundiced and the wound began to show signs of infection and enzymatic breakdown. Subsequently, a wound vacuum was placed and somatostatin started. An EGD and subsequent ERCP revealed organized necrosis with communication to the lumen (Figure 4) and side branch disruption at the head with filling of the lesser sac; ultimately, an 11-cm stent was placed into the pancreatic duct (Figure 5).



Figure 2. CT Abdomen. (A and B) Gas-containing fluid collection anterior to the heart contiguous with a pericardial effusion and extending into the anterior upper abdomen as well as fluid collections (C and D) within the pancreatic head and neck with possibility of pancreatic necrosis and superimposed infection.

The patient became hypotensive and was unable to maintain adequate perfusion despite aggressive pressor support. An emergent family meeting was held to discuss the patient's poor prognosis and goals of care. At this point a decision was made to limit treatment to comfort measures only and the patient expired shortly thereafter.

The etiology of the patient's acute pancreatitis was never fully understood. The common culprits such as gallstone, ethanol, or hypertriglyceridemia-induced pancreatitis were excluded relatively quickly. Tocilizumab has been implicated in the setting of necrotizing pancreatitis, although the literature available is extremely limited.³ It is possible that tocilizumab played a role in this patient; however, there is insufficient evidence to implicate tocilizumab.

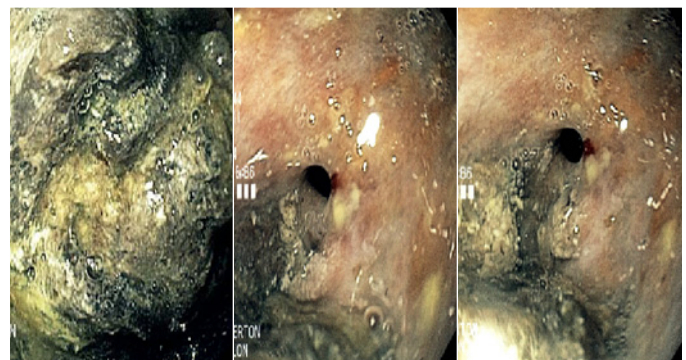


Figure 3. Pancreatic necrosis along with pancreaticoduodenal fistula.

Discussion

A pancreatic pseudocyst is a collection of fluid lacking an epithelial lining arising either within or adjacent to the pancreas, developing in up to 50% of cases of acute pancreatitis.⁴ Typically comprised of pancreatic secretions, pseudocysts tend to form in the lesser sac or extend

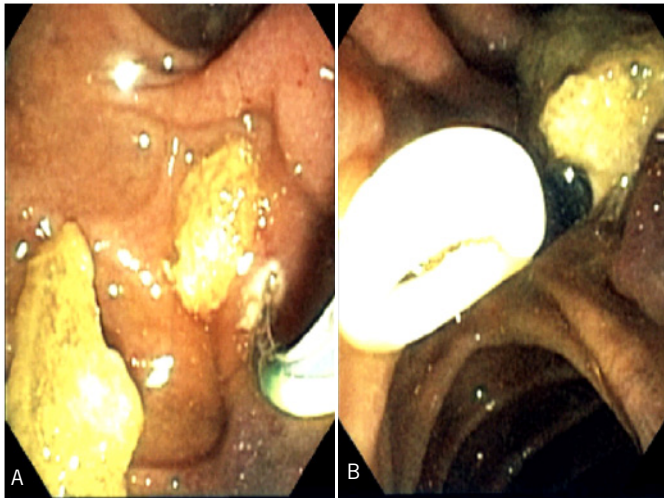


Figure 4. ERCP. (A) Pancreatic stricture (genu). (B) Side branch disruption (head) with large filling of large cyst cavity (lesser sac).

through planes of least resistance, including the aortic and oesophageal hiatus, the foramen of Morgagni, and the diaphragm.⁵ Internal pancreatic fistulas presenting as pleural effusions are a well-known complication of pancreatitis,² but a pericardial effusion that presents as cardiac tamponade in the setting of acute pancreatitis is extremely rare.

The pathophysiology behind the development of a pericardial effusion in acute pancreatitis is poorly understood. There have been several proposed theories that attempt to explain the possible mechanism, including chemical pericarditis as a result of lymphatic or hematologic transport of pancreatic enzymes and necrosis of vascular walls in the areas of fat necrosis in the subpericardium.⁶ Maringhini et al performed an echocardiogram on 100 patients with a diagnosis of acute pancreatitis, 24 of whom had severe pancreatitis and 3 of whom eventually succumbed to their illness. Maringhini et al noted 17% of patients with acute pancreatitis do have a pericardial effusion; however, the effusion in all 97 survivors dissipated spontaneously.⁷ Viyam et al studied 15 patients with alcohol-induced pancreatitis by M-mode echocardiography and noted 47% to have a pericardial effusion compared to 11% in the control population, which he attributed to left ventricular dysfunction as a result of chronic alcohol abuse.⁸ Pezzilli et al studied 21 patients with M-mode and B-mode echocardiography to establish the prevalence of pericardial effusion and simultaneously

evaluate left ventricular function in patients with acute pancreatitis. Pezzilli et al concluded the presence of pericardial effusion or left ventricular asynergy may be observed in the setting of acute pancreatitis; however, these findings seem to be unrelated to the severity of the disease in contrast to the presence of pleural and abdominal effusions.⁹

In a patient with a suspected pancreaticopericardial fistula, CT imaging is a useful technique for the overall assessment of pancreatitis and its complications. ERCP or MRCP may be helpful in delineating ductal anatomy. In the presence of a fistula, serum amylase levels are of little or no benefit, as they may or may not be elevated, as was the case in our patient.¹⁰

Conservative approaches may lead to closure rates of up to 50%, thus an initial 2–4 week trial of conservative management is indicated. Bowel rest and somatostatin or its analogue octreotide can be used to reduce pancreatic secretions. A surgical approach, most commonly with a Roux-en-Y with or without partial pancreatic resection, is warranted if the fistula does not close within 2–4 weeks. The role of ERCP and stenting to bridge the site of duct disruption serves as an alternative to surgery. Current recommendations are that the stent remain in situ for 6 weeks; however, long-term data is necessary to determine the role for stenting in the management of internal pancreatic fistulas.¹⁰

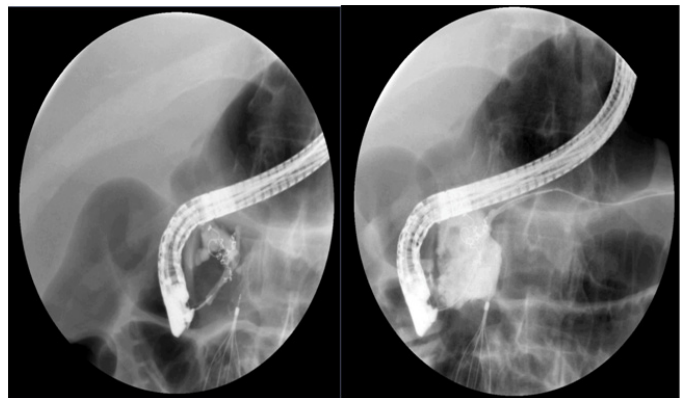


Figure 5. Pancreatic sphincterotomy performed with insertion of 7 Fr 11 cm pancreatic duct stent (Freeman).

Lamparter et al recently published the first case of a successful endoscopic treatment of a pancreaticopericardial fistula. Their patient was a 57-year-old with a diagnosis of acute alcoholic pancreatitis complicated by pericardial fistula and tamponade. Their patient underwent emergent pericardiocentesis and was initially treated with intrapericardial triamcinolone and octreotide, which was unsuccessful. Next, they performed an ERCP and were able to identify and successfully stent the culprit disrupted pancreatic duct, resulting in eventual resolution of the pancre-

aticopericardial fistula.¹¹ The role of ERCP and pancreatic duct stenting in the setting of a pancreaticopericardial fistula is not completely understood; however, this case illustrates its success and argues the necessity for further data.

Disclosures

Author contributions: P.J. Parekh and D.A. Johnson conceptualized, initiated, and wrote the article; D. Howerton initiated and wrote the article. P.J. Parekh is the article guarantor.

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