Overview of the complications of chronic pancreatitis

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INTRODUCTION

Chronic pancreatitis is an inflammatory condition that results in permanent structural changes in the pancreas, which can lead to impairment of exocrine and endocrine functions. This topic will review the complications of chronic pancreatitis. The clinical manifestations, diagnosis, and management of chronic pancreatitis are discussed in detail separately. (See "Chronic pancreatitis: Clinical manifestations and diagnosis in adults".)

PANCREATIC DUCT DISRUPTION

Pseudocysts — Pseudocysts develop in approximately 10 percent of patients with chronic pancreatitis. They can be induced by an acute exacerbation of pancreatitis or occur as a result of ductal disruption. Pseudocysts are mature fluid collections, have a well-defined wall (although without a true epithelial lining) visible on computed tomography (CT) or magnetic resonance imaging (MRI), and do not contain solid material or pancreatic necrosis. Most pseudocysts communicate with the pancreatic ductal system and contain high concentrations of digestive enzymes. The walls of pseudocysts are formed by granulation tissue and by adjacent structures such as the stomach, transverse mesocolon, gastrocolic omentum, and pancreas (image 1). (See "Approach to walled-off pancreatic fluid collections in adults", section on 'Etiology and classification'.)

Clinical manifestations — Most pseudocysts are asymptomatic, but may produce

symptoms such as abdominal pain, weight loss, early satiety or jaundice due to gastric outlet, and intestinal or biliary obstruction due to mass effect. Pancreatic pseudocysts are sterile but may become infected. Infection, usually with gut flora, occurs in up to 10 percent of pseudocysts. Signs of an infection include fever, hypotension, and leukocytosis. If left untreated, this may progress to peritonitis and/or systemic sepsis.

Diagnosis — The diagnosis of a pancreatic pseudocyst is usually made by the finding of an intra- or peripancreatic encapsulated fluid collection on imaging in a patient with a history of chronic pancreatitis, or in a patient who has recovered from acute pancreatitis. Pancreatic pseudocysts may be seen on transabdominal ultrasound, but contrast-enhanced CT or MRI is typically obtained to confirm the diagnosis and to further classify the fluid collection. Pseudocysts do not contain solid material on imaging. If the diagnosis is uncertain, either because the clinical setting is unclear or when imaging findings are atypical, diagnostic endoscopic ultrasound can be performed with sampling of the fluid collection and cyst wall. The diagnosis of pancreatic pseudocysts and the differential diagnosis include a variety of other pancreatic cystic neoplasms. (See "Classification of pancreatic cysts" and "Approach to walled-off pancreatic fluid collections in adults", section on 'Differential diagnosis' and "Approach to walled-off pancreatic fluid collections in adults", section on 'Clinical features'.)

Differential diagnosis — The differential diagnosis of a pancreatic pseudocyst includes pancreatic cystic neoplasms, cystic degeneration of a solid pancreatic tumor, and rare non-neoplastic pancreatic cysts (eg, retention cysts). These are discussed in detail separately. (See "Approach to walled-off pancreatic fluid collections in adults", section on 'Differential diagnosis'.)

Management — The management of a pancreatic pseudocyst depends on the patient's symptoms, characteristics and location of the fluid collection, and whether complications such as a pseudoaneurysm or infection have developed. (See 'Pseudoaneurysms' below.)

Asymptomatic pseudocysts do not require therapy. For patients who are symptomatic, have rapidly enlarging pseudocysts, or who have infected pseudocysts that do not improve with medical management, a drainage procedure is often necessary. The indications, risks, and benefits of these treatment options are discussed in detail separately. (See "Approach to walled-off pancreatic fluid collections in adults", section on 'Management'.)

Pancreatic ascites/pleural effusion — Pancreatic ascites and pleural effusion may develop following disruption of the pancreatic duct, leading to fistula formation in the abdomen or chest, or rupture of a pseudocyst with tracking of pancreatic juice into the peritoneal cavity

or pleural space. Less than 10 percent of patients with pseudocysts will develop pancreatic ascites, and even fewer will develop a pancreatic pleural effusion [1].

- **Clinical manifestations** – Patients with pancreatic ascites may be asymptomatic. The most common symptom is abdominal distension. Other less frequent symptoms include abdominal pain, weight loss, or dyspnea. Patients with a pancreatic pleural effusion may mainly complain of dyspnea or chest pain. Patients with pancreatic ascites and pleural effusions frequently do not have a history of a recent flare of pancreatitis or pancreatic-type abdominal pain. Evidence of ascites or a pleural effusion may be present on physical examination. The pleural effusion is almost always left sided.

- **Diagnosis** – The diagnosis is established by evaluation of ascitic and pleural fluid. Analysis of fluid obtained at paracentesis or thoracentesis is diagnostic. Ascitic fluid amylase concentration in the fluid is very high, typically >1000 IU (international units)/L [2]. The combination of a serum-albumin ascites gradient below 1.1 g/dL, a total protein level >3 g/dL, and elevated ascitic amylase is diagnostic of pancreatic ascites.

  Pleural pancreatic fluid is exudative with albumin levels typically over 3 g, and the amylase level is often more than 4000 IU/L. The diagnosis of an underlying pancreatic fistula requires evidence of pancreatic duct disruption. (See "Pleural fluid analysis in adults with a pleural effusion" and "Chylous, bloody, and pancreatic ascites".)

- **Differential diagnosis** – The differential diagnosis of a pancreatic fistula includes other causes of abdominal pain and peripancreatic fluid collections (eg, pancreatic cystic neoplasm) and ascites. The presentation may mimic intra-abdominal carcinomatosis. Fluid collections due to a pancreatic fistula can be distinguished from these by sampling the fluid and imaging to assess the pancreatic duct. This evaluation is discussed in detail separately. (See "Evaluation of adults with ascites", section on 'Determining the cause of the ascites' and "Approach to walled-off pancreatic fluid collections in adults", section on 'Clinical features'.)

- **Management** – Initial management of pancreatic fistulas and resultant complications of pancreatic ascites and pleural effusion include reduction of pancreatic stimulation with octreotide (a long-acting somatostatin analogue) to decrease pancreatic secretion. Conservative therapy is often unsuccessful, and endoscopic stenting with bridging of the ductal disruption is frequently required [3]. Surgery for a persistent pancreatic fistula is indicated when endoscopic management fails or is technically unfeasible. (See "Surgical resection of lesions of the head of the pancreas", section on 'Pancreatic

fistula'.)

OBSTRUCTION

Symptomatic obstruction of the bile duct and/or duodenum develops in 5 to 10 percent of patients with chronic pancreatitis. These complications can result from inflammation and fibrosis in the head of the pancreas or direct compression from a pseudocyst.

Biliary obstruction

- **Clinical manifestations** – Patients with bile duct obstruction may present with postprandial epigastric abdominal pain, nausea, and jaundice. Patients may have subtle elevations of transaminases for years, and only later do alkaline phosphatase and bilirubin levels begin to rise. Longstanding biliary obstruction may lead to secondary biliary cirrhosis or recurrent cholangitis.

- **Diagnosis** – The diagnosis is suspected based on presenting clinical symptoms of abdominal pain, early satiety, weight loss, and jaundice. However, these symptoms are not specific, and the initial evaluation begins with laboratory testing to identify the cause of jaundice and abdominal imaging. Additional testing is then directed based upon the findings of the initial testing as well as the patient's clinical presentation, prior diagnosis of chronic pancreatitis, and risk factors for pancreatic cancer (as cancer can mimic this clinical presentation). The diagnostic approach to a patient with abdominal pain, early satiety, and jaundice and subsequent evaluation is discussed in detail, separately. (See "Diagnostic approach to the adult with jaundice or asymptomatic hyperbilirubinemia", section on 'Initial laboratory tests and interpretation' and "Clinical manifestations, diagnosis, and staging of exocrine pancreatic cancer", section on 'Diagnostic approach'.)

- **Management** – In patients with bile duct stenosis as a complication of chronic pancreatitis, operative biliary bypass is the treatment of choice with choledochoduodenostomy or a Roux-en-Y jejunal bypass. In patients whose biliary obstruction is associated with severe abdominal pain and a large amount of fibrotic tissue in the head of the pancreas, a pancreatic head resection may be indicated. Endoscopic stenting is generally a temporizing measure, as it frequently requires several stent changes, but the use of large-caliber self-expanding metal stents will resolve the stricture in some patients who do not undergo surgery. Decompression can reverse cirrhosis secondary to bile duct obstruction [4].

In patients whose biliary obstruction is associated with a pseudocyst in the region of the pancreatic head, pseudocyst drainage alone may be sufficient to relieve the obstruction. The management of biliary obstruction is discussed in detail separately. (See "Surgery for chronic pancreatitis", section on 'Surgery for other complications' and "Bile duct resection and reconstruction".)

Duodenal obstruction — Duodenal obstruction occurs from fibrosis in the pancreas and surrounding tissues. Duodenal obstruction is characterized by postprandial pain and early satiety. Duodenal stenosis is often associated with biliary obstruction and an inflammatory pancreatic head mass, and may be best managed with resection of the head of the pancreas (classic Whipple procedure) or with a duodenum-preserving pancreatic head resection. The management of duodenal stenosis (ie, gastric outlet obstruction) is discussed in detail in another topic. (See "Gastric outlet obstruction in adults" and "Surgery for chronic pancreatitis", section on 'Surgery for other complications'.)

VASCULAR COMPLICATIONS

Pseudoaneurysms — Pseudoaneurysm formation is a rare complication of chronic pancreatitis. Affected arteries are in close proximity to the pancreas with the most commonly involved being the splenic artery followed by the gastroduodenal and pancreaticoduodenal arteries.

- **Clinical manifestations** – Pseudoaneurysm in a pseudocyst may be complicated by bleeding into the associated pseudocyst. Blood may remain in the pseudocyst, rupture the pseudocyst, and reach the peritoneal cavity, or reach the gut lumen through the pancreatic duct (hemosuccus pancreaticus). Patients usually present with a sudden onset of abdominal pain associated with unexplained anemia or gastrointestinal bleeding. (See "Causes of upper gastrointestinal bleeding in adults", section on 'Hemosuccus pancreaticus'.)

- **Diagnosis** – The diagnosis of a pseudoaneurysm is often made incidentally on a computed tomography scan (with and without contrast) or magnetic resonance imaging in a patient with chronic pancreatitis. A pseudoaneurysm appears as a contrast-enhancing cystic structure in the pseudocyst.

- **Management** – Mesenteric angiography permits confirmation of the diagnosis, and also provides a means of therapy by embolization of the pseudoaneurysm in patients with bleeding [5]. If bleeding persists or is massive, treatment is with

pancreaticoduodenectomy and ligation of the bleeding vessel, which definitively prevents rebleeding. However, surgery for bleeding pseudoaneurysms is difficult and associated with a high morbidity and mortality [6]. (See "Angiographic control of nonvariceal gastrointestinal bleeding in adults", section on 'Embolization'.)

Splenic vein thrombosis and gastric varices — The incidence of splenic vein thrombosis in patients with chronic pancreatitis is estimated to be up to 12 percent [7]. Splenic vein thrombosis develops secondary to inflammation due to chronic pancreatitis as the splenic vein courses along the posterior surface of the pancreas. Affected patients can develop gastric varices as a result of associated portal hypertension. Splenic vein obstruction produces a left-sided portal hypertension with gastric varices in the absence of esophageal varices or gastric varices out of proportion to esophageal varices.

Patients may be asymptomatic. Among patients who are symptomatic, upper gastrointestinal bleeding due to gastric varices is the most common clinical presentation. On physical examination, patients may have evidence of ascites and splenomegaly. The clinical manifestations, diagnosis, and management of splenic and portal vein thrombosis are discussed in detail separately. (See "Chronic portal vein thrombosis in adults: Clinical manifestations, diagnosis, and management".)

PANCREATIC INSUFFICIENCY

Pancreatic diabetes — Diabetes may develop as a consequence of destruction of the pancreatic islets or by lowering the threshold for type 2 diabetes linked to metabolic (obesity, male sex, increasing age) and genetic (family history, polygenic risk scores) co-factors [8,9]. Diabetes due to chronic pancreatitis and destruction of the islets is termed pancreatogenic (or type 3c) diabetes [10]. The destruction or loss of entire islets leads to loss of insulin secretion, as well as loss of secretion of other islet-derived regulatory hormones. In particular, the lack of glucagon, pancreatic polypeptide, and insulin leads to a form of brittle diabetes characterized by insulin deficiency, rare ketosis, and frequent treatment-induced hypoglycemia (due to lack of glucagon) [10]. Like steatorrhea, type 3c diabetes is a complication of long-standing chronic pancreatitis and is especially common in those who also undergo pancreatic surgery.

Pancreatic endocrine and exocrine insufficiency usually takes many years to develop. Endocrine insufficiency ultimately occurs in 37 to 44 percent of patients with chronic pancreatitis [11]. While all patients with chronic pancreatitis are at risk for diabetes, those

with longstanding duration of disease, prior partial pancreatectomy, and early onset of calcific disease may be at higher risk. Patients with chronic pancreatitis should be screened with fasting glucose and HbA1c annually [12]. Diabetes due to chronic pancreatitis (pancreatogenic diabetes) can lead to large fluctuations in blood sugar that are difficult to manage. Impairment in either fasting glucose or HbA1c requires further evaluation. The diagnosis and management of diabetes mellitus is discussed in detail separately. (See "Classification of diabetes mellitus and genetic diabetic syndromes", section on 'Chronic pancreatitis' and "Screening for type 2 diabetes mellitus", section on 'Screening tests' and "Overview of general medical care in nonpregnant adults with diabetes mellitus".)

Diabetes and chronic pancreatitis have complex interactions. Long-standing diabetes is a risk factor for chronic pancreatitis. Long-standing diabetes produces changes in pancreatic structure and function that mimic chronic pancreatitis in many ways, even if the threshold of a diagnosis of chronic pancreatitis is not reached (known as diabetic pancreatopathy) [13]. Finally, the presence of concomitant chronic pancreatitis and diabetes markedly increases the risk of secondary pancreatic carcinoma.

Exocrine pancreatic insufficiency — Patients with mild exocrine pancreatic insufficiency may be asymptomatic or have mild abdominal discomfort and bloating with normal-appearing bowel movements. Patients may not complain of diarrhea despite steatorrhea. Advanced exocrine pancreatic insufficiency results in maldigestion of fat and protein, sarcopenia, and weight loss. Osteoporosis is found in approximately 25 percent of patients with chronic pancreatitis, and osteopenia in an additional 40 percent of patients. Overt steatorrhea does not occur until approximately 90 percent of glandular function has been lost. (See "Exocrine pancreatic insufficiency", section on 'Clinical manifestations'.)

PANCREATIC CANCER

Patients with hereditary pancreatitis and some nonhereditary forms of chronic pancreatitis are at increased risk for pancreatic adenocarcinoma due to the presence of chronic pancreatic inflammation. The risk of pancreatic cancer developing in the background of chronic pancreatitis varies with the duration and etiology of chronic pancreatitis and smoking history [14-16]. Patients with pancreatic cancer may present with similar symptoms of chronic pancreatitis, including weight loss, abdominal pain, and jaundice. The epidemiology, clinical manifestations, and diagnosis of pancreatic cancer, and issues related to screening for pancreatic cancer in high-risk individuals with hereditary pancreatitis are discussed in detail elsewhere. (See "Clinical manifestations, diagnosis, and staging of

exocrine pancreatic cancer", section on 'Diagnostic approach' and "Familial risk factors for pancreatic cancer and screening of high-risk patients", section on 'Hereditary pancreatitis' and "Familial risk factors for pancreatic cancer and screening of high-risk patients", section on 'Pancreatic cancer screening' and "Pancreatitis associated with genetic risk factors", section on 'Cancer screening'.)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Chronic pancreatitis and pancreatic exocrine insufficiency".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient education: Acute pancreatitis (The Basics)")
- Beyond the Basics topics (see "Patient education: Chronic pancreatitis (Beyond the Basics)")

SUMMARY AND RECOMMENDATIONS

- **Pancreatic pseudocysts** – Chronic pancreatitis is an inflammatory condition that results in permanent structural changes in the pancreas, which can lead to impairment of exocrine and endocrine function. Pseudocysts develop in approximately 10 percent

of patients with chronic pancreatitis. Most pseudocysts are asymptomatic. They can, however, produce a wide range of clinical problems depending upon the location and extent of the fluid collection. Symptoms include abdominal pain, duodenal or biliary obstruction, vascular occlusion, or fistula formation into adjacent viscera, the pleural space, or pericardium. (See 'Pseudocysts' above.)

- **Biliary and duodenal obstruction** – Symptomatic obstruction of the bile duct and/or duodenum develops in 5 to 10 percent of patients with chronic pancreatitis. Duodenal obstruction is characterized by postprandial pain and early satiety. Patients with bile duct obstruction may present with postprandial epigastric abdominal pain, nausea, and jaundice. However, patients usually present with jaundice after a very indolent and prolonged clinical course; elevations of transaminases may be subtle for years, and only later do the alkaline phosphatase and bilirubin begin to rise. Patients may develop cirrhosis secondary to chronic bile duct obstruction. (See 'Biliary obstruction' above.)

- **Pancreatic ascites and pleural effusion** – Disruption of the pancreatic duct leading to fistula formation in the abdomen or chest, or rupture of a pseudocyst with tracking of pancreatic juice into the peritoneal cavity or pleural space can lead to pancreatic ascites and pleural effusions. Patients with pancreatic ascites may not have any symptoms or may note abdominal distension. Some patients may have abdominal pain, weight loss, or dyspnea. Those patients with pancreatic pleural effusion may mainly complain of dyspnea or chest pain rather than pancreatic-type abdominal pain. (See 'Pancreatic ascites/pleural effusion' above.)

- **Splenic vein thrombosis** – Inflammation due to chronic pancreatitis can lead to splenic vein thrombosis in up to 12 percent of patients. Affected patients can develop gastric varices as a result of associated portal hypertension. Patients may be asymptomatic. Among patients who are symptomatic, upper gastrointestinal bleeding due to gastric varices is the most common clinical presentation. (See 'Splenic vein thrombosis and gastric varices' above.)

- **Pseudoaneurysm formation** – Pseudoaneurysm in a pseudocyst is a rare complication of chronic pancreatitis and may be complicated by bleeding into the associated pseudocyst. Blood may remain in the pseudocyst, rupture the pseudocyst, and reach the peritoneal cavity, or reach the gut lumen through the pancreatic duct (hemosuccus pancreaticus). Patients usually present with a sudden onset of pain associated with unexplained anemia or gastrointestinal bleeding. (See 'Pseudoaneurysms' above.)

- **Endocrine insufficiency** – Diabetes due to chronic pancreatitis (pancreatogenic diabetes) occurs in 30 to 50 percent of patients with chronic pancreatitis and can lead to large fluctuations in blood sugar that are difficult to manage. (See 'Pancreatic diabetes' above.)

- **Cancer risk** – Patients with hereditary pancreatitis and nonhereditary forms of chronic pancreatitis are at increased risk for pancreatic adenocarcinoma due to the presence of chronic pancreatic inflammation. The risk of pancreatic cancer developing in the background of chronic pancreatitis varies with the duration and etiology of chronic pancreatitis and smoking history. (See 'Pancreatic cancer' above.)

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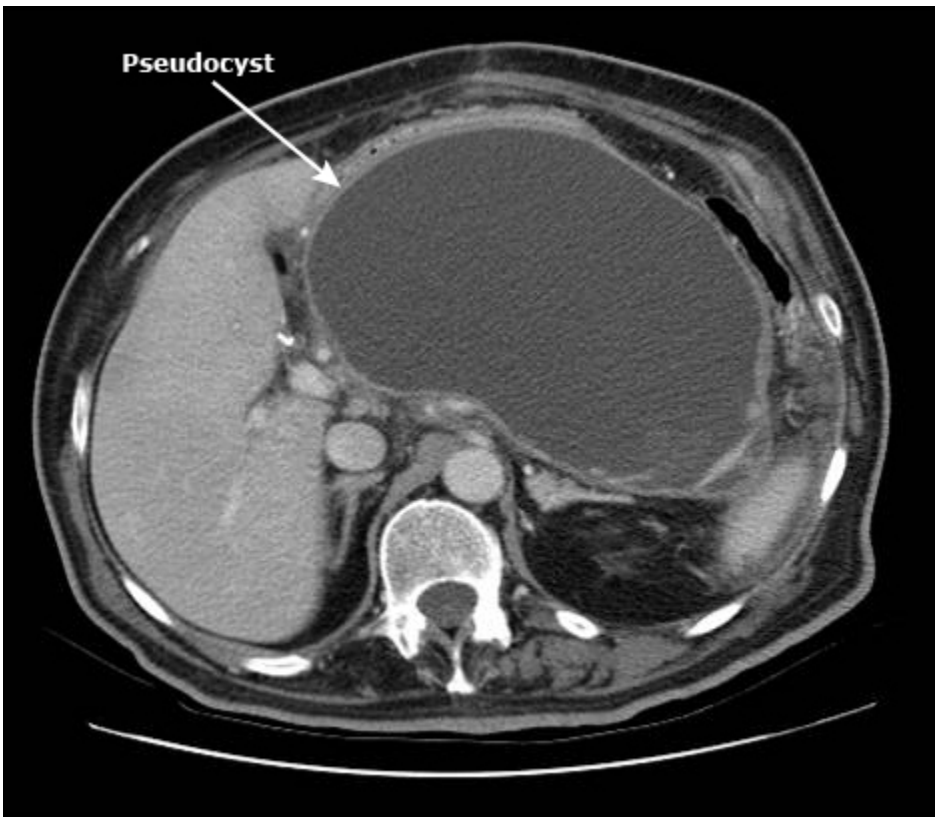
GRAPHICS

Pancreatic pseudocyst



Computed tomographic scan showing a massive pancreatic pseudocyst compressing the stomach and obliterating the pancreas.

Graphic 80720 Version 5.0

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