Chronic pancreatitis: Clinical manifestations and diagnosis in adults

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INTRODUCTION

Chronic pancreatitis can result from episodes of acute pancreatitis of any cause, most commonly in those with multiple relapsing episodes of acute pancreatitis [1-3]. While acute pancreatitis can be considered an event, chronic pancreatitis is an ongoing process of pathologic response to pancreatic injury. Acute and chronic pancreatitis can best be thought of as a continuum and two parts of the same spectrum of disease rather than as two completely separate conditions. The syndrome of chronic pancreatitis results from exposure to risk factors (genetic and environmental) and is characterized by derangements in pancreatic function (digestive function or insulin secretion), and structural changes in the pancreas visible on imaging or endoscopic studies [4]. However, many of the clinical features take time to manifest and may be absent in the early stages of chronic pancreatitis [5]. While chronic pancreatitis can be diagnosed easily when end-stage features have developed, the clinical challenge is making an accurate diagnosis early in the clinical course when interventions to prevent progression might be most effective.

This topic reviews the clinical manifestations and diagnosis of chronic pancreatitis. The etiology, pathogenesis, complications, and treatment of chronic pancreatitis are discussed separately. (See "Etiology and pathogenesis of chronic pancreatitis in adults" and "Overview of the complications of chronic pancreatitis" and "Chronic pancreatitis: Management".)

CLINICAL MANIFESTATIONS

Clinical presentation

Abdominal pain — Abdominal pain is the most common clinical symptom in chronic pancreatitis. The pain is most commonly felt in the epigastric region and often radiates to the back. It may be worse when recumbent and patients may experience postprandial exacerbation. Pain is most common in those with chronic pancreatitis due to alcohol or tobacco use and in those with idiopathic pancreatitis occurring at a young age. Nausea, vomiting, and anorexia are commonly associated with the pain.

The pattern of abdominal pain varies among patients. While some patients may experience constant pain of variable severity with periodic exacerbations, others have ongoing relentless and severe pain. Many patients may present with episodes of pain and be pain-free for prolonged periods between exacerbations. In this phase, patients may be labeled as having relapsing acute pancreatitis. Pain patterns can change over time, most commonly evolving from episodic to more continuous pain. In rare patients, pain may actually diminish after years of disease. However, a change in pain pattern or sudden worsening of pain should also prompt a search for a complication of chronic pancreatitis, such as a pseudocyst, duodenal or biliary obstruction, or secondary pancreatic carcinoma. (See "Clinical manifestations, diagnosis, and staging of exocrine pancreatic cancer" and "Overview of the complications of chronic pancreatitis" and 'Complications of chronic pancreatitis' below.)

The presence of pain or its severity does not correlate with the severity of damage to the pancreas on imaging studies (eg, computed tomography [CT] or magnetic resonance cholangiopancreatography [MRCP]) [6]. An important clinical consequence of this lack of correlation is that patients may have severe pain with evidence of only minimal pancreatic damage on CT, or may have dramatic damage on CT and yet be pain-free. Pain is the most common reason for hospitalization or for endoscopic or surgical intervention, and the most deleterious for quality of life (QOL) [7].

The cause of pain in these patients may be due to increased pressure, ischemia, and inflammation in the pancreas; injury and alterations in pain signaling of nociceptive neurons; or complications of chronic pancreatitis. These mechanisms can overlap. The injury and alteration to pain signaling in these patients may be most important, and can produce a centrally sensitized pain state in which central (spinal cord and central nervous system) neural signaling and structural reorganization produce ongoing pain irrespective of local pancreatic events. These patients develop hyperalgesia (magnified pain in response to normally painful stimuli) as

well as allodynia (pain in response to stimuli that are not pathologic) [8]. These changes in nociceptive signaling explain the frequent failure of therapeutic approaches to pain that target only the pancreas or pancreatic duct.

Steatorrhea — Patients with steatorrhea may report oily or floating stool due to fat maldigestion but may not have frequent or watery diarrhea. Approximately 90 percent of pancreatic exocrine secretory capacity must be lost before maldigestion due to exocrine pancreatic insufficiency (steatorrhea) develops. This degree of damage takes time to accumulate. Steatorrhea is generally a consequence of long-standing chronic pancreatitis (usually >5 to 10 years) but may also be seen with lesions that obstruct the pancreatic duct and prevent enzymes from reaching the duodenum, and after severe episodes of acute pancreatitis that destroy significant amounts of acinar cells. Weight loss may occur, especially if pain also limits oral intake. Weight loss, especially sarcopenia, is associated with increased mortality risk in patients with chronic pancreatitis [9].

Asymptomatic — While most patients with chronic pancreatitis have significant symptoms, a small subset of patients with chronic pancreatitis will not have symptoms or identifiable clinical consequences. In such patients, the diagnosis of chronic pancreatitis may be made incidentally by cross-sectional imaging (eg, a CT scan demonstrating diffuse pancreatic calcifications). (See 'Imaging findings' below and 'Cross-sectional imaging' below.)

Laboratory findings — Low serum levels of amylase or lipase are often seen in patients with advanced chronic pancreatitis but are not of any diagnostic value. In patients with an episode of acute pancreatitis, serum levels of amylase or lipase are elevated more than three times the upper limit of normal. However, in patients who develop repeated attacks, and who are evolving towards chronic pancreatitis, peak levels of elevation of amylase and lipase tend to decrease with each additional pain flare. In those with established chronic pancreatitis, even severe painful flares are often associated with only minimal or even no elevation in amylase or lipase. This change is likely a consequence of the progressive destruction of acinar cells (the source of these enzymes) over time.

Elevations of serum bilirubin and alkaline phosphatase can suggest compression of the intrapancreatic portion of the bile duct by pancreatic edema or fibrosis. Jaundice or significant cholestasis can also occur from a coexistent pancreatic cancer. (See 'Complications of chronic pancreatitis' below.)

Deficiencies of fat-soluble vitamins frequently develop, with vitamin D in particular, which increases the risk of metabolic bone disease. Deficiencies of water-soluble vitamins and trace

elements are less common but can occur when oral intake is limited or inadequate. (See 'Complications of chronic pancreatitis' below.)

In those with chronic pancreatitis due to hypertriglyceridemia, serum levels of triglycerides are elevated. Although triglyceride levels above 1000 mcg/mL are typically needed to produce the first episode of acute pancreatitis in these patients, relapses may occur with levels as low as 500 mcg/mL [10]. (See "Hypertriglyceridemia-induced acute pancreatitis", section on 'Laboratory findings'.)

In patients with type 1 autoimmune pancreatitis (AIP), elevations in serum levels of immunoglobulin G4 (IgG4) can be seen, and these elevations are part of the diagnostic criteria. In addition, other autoimmune markers may occasionally be elevated (rheumatoid factor, antinuclear antibody, and antismooth muscle antibody titer) [11]. (See "Autoimmune pancreatitis: Clinical manifestations and diagnosis", section on 'Serum IgG4'.)

Imaging findings

- **Plain abdominal radiograph** – The presence of diffuse pancreatic calcifications may be seen incidentally on plain radiographs of the abdomen (image 1). While the presence of pancreatic calcification is specific to chronic pancreatitis, it is not uniformly present and can take decades to develop. Nearby vascular calcifications can also falsely give the appearance of pancreatic calcification.

- **Abdominal ultrasound** – Imaging features of chronic pancreatitis on ultrasound include an increase in echogenicity, atrophy, a dilated pancreatic duct, and pancreatic duct stones.

  Ultrasonography is of limited diagnostic utility in the diagnosis of chronic pancreatitis since the pancreas may not be visible due to body habitus or overlying intestinal gas. The sensitivity of abdominal ultrasound for chronic pancreatitis is approximately 60 percent [12]. However, changes in echotexture of the pancreas similar to chronic pancreatitis may be seen in older individuals and in those with long-standing type 1 or type 2 diabetes mellitus.

- **Cross sectional imaging** – Cross-sectional imaging with high-quality CT scan using multidetector technology and a pancreatic protocol, or magnetic resonance imaging (MRI) with MRCP can establish the diagnosis of chronic pancreatitis. (See 'Cross-sectional imaging' below.)

COMPLICATIONS OF CHRONIC PANCREATITIS

Chronic pancreatitis is associated with a variety of complications. The most common complications include metabolic consequences of exocrine insufficiency (particularly osteopenia and osteoporosis), pancreatogenic (type 3c) diabetes, and opioid dependency due to the treatment of pain [13]. Up to two-thirds of patients with chronic pancreatitis will develop osteoporosis or osteopenia and patients are also at increased risk of bone fracture [14,15]. (See "Overview of the complications of chronic pancreatitis".)

Other less frequent complications include pseudocyst formation, bile duct or duodenal obstruction, pancreatic ascites or pancreatic pleural effusion, splenic vein thrombosis, arterial pseudoaneurysms, small intestinal bacterial overgrowth, gastroparesis, and pancreatic ductal adenocarcinoma.

DIAGNOSIS

Approach to evaluation — Chronic pancreatitis should be suspected in patients with chronic abdominal pain and/or a history of relapsing acute pancreatitis, symptoms of pancreatic exocrine (diarrhea, steatorrhea, or weight loss) insufficiency, or pancreatogenic diabetes. Occasionally, patients with chronic pancreatitis are asymptomatic and the diagnosis is suspected incidentally on imaging [16]. (See 'Imaging findings' above and 'Cross-sectional imaging' below.)

Cross-sectional imaging with high-quality computed tomography (CT) scan using multidetector technology and a pancreatic protocol, or magnetic resonance imaging (MRI) with magnetic resonance cholangiopancreatography (MRCP) are the best initial diagnostic tests for chronic pancreatitis. In patients who have undergone a routine abdominal CT (often through an emergency department) but not had a high-quality CT (or MRI with MRCP), we repeat pancreatic imaging. In patients without advanced chronic pancreatitis, especially early in the clinical course, the CT or MRI may not demonstrate conclusive evidence of chronic pancreatitis [5]. As chronic pancreatitis progresses over time, damage visible on cross-sectional imaging will accumulate, as will the risk of exocrine insufficiency and/or diabetes. Most available diagnostic tests will be able to detect advanced chronic pancreatitis.

In patients with suspected chronic pancreatitis but equivocal cross-sectional imaging, we perform either a direct pancreatic function test with secretin, if available, or endoscopic ultrasound (EUS) (table 1). However, neither test should be used as the sole diagnostic criterion for chronic pancreatitis, and the results of these tests should be interpreted in the context of the patient's history (recurrent acute pancreatitis), symptoms, and probable risk for chronic pancreatitis.

If the diagnosis remains in doubt, follow-up over time with periodic reassessment is the best approach. This should include a detailed review of symptoms and repeat cross-sectional imaging with MRCP. Our approach is largely consistent with the 2020 American College of Gastroenterology Guidelines on Chronic Pancreatitis [2].

Cross-sectional imaging

- **Computed tomography (CT)** – CT has become the most frequently utilized test for diagnosis of chronic pancreatitis. It is widely available and is able to image the entire pancreas and pancreatic duct. Features of chronic pancreatitis on CT include atrophy of the pancreas, ductal dilatation, and multiple parenchymal and intraductal calcifications (table 1). The overall sensitivity of CT for chronic pancreatitis ranges from 80 to 90 percent, with a specificity of approximately 85 percent [12]. Like all diagnostic tests for chronic pancreatitis, however, the sensitivity and specificity increase with duration of disease, and the sensitivity is lower in patients with early chronic pancreatitis (as these patients may have a normal or near-normal CT of the pancreas) [17].

- **Magnetic resonance imaging (MRI)** – MRI including MRCP gives more detailed images of the pancreatic duct [18]. Drop in signal on T1-weighted sequences are useful markers for chronic pancreatitis and appear to hold promise for earlier diagnosis than is possible with CT. The ductal findings of early disease can range from a normal-looking main pancreatic duct (MPD) to mild irregularity of MPD and side branches. With more advanced pancreatitis, there is progressive glandular atrophy, irregularity of the pancreatic duct contour with focal areas of narrowing and dilation, and ectasia of the side branches. Unlike CT, MRCP does not show calcifications in the pancreas.

  Secretin may be administered at the time of MRCP to both improve visualization of the pancreatic duct and estimate the pancreatic ductal secretion of fluid in response to secretin. MRCP, particularly with secretin infusion, has replaced diagnostic endoscopic retrograde cholangiopancreatography (ERCP) in these patients, as it is as accurate and far safer. Like ERCP, however, MRCP will be inaccurate in those with minimal pancreatic ductal abnormalities (early chronic pancreatitis).

Additional testing in selected patients

Endoscopic ultrasound — Endoscopic ultrasound (EUS) allows a highly detailed examination of the pancreatic parenchyma and duct. However, EUS features of chronic pancreatitis are not specific. Similar changes in the pancreas can be also be seen in patients who do not appear to have chronic pancreatitis, including older individuals, chronic alcoholics, social drinkers,

smokers, diabetics, and those with chronic renal insufficiency. There is also significant inter-operator variability in interpretation of EUS findings.

Nine diagnostic criteria have been proposed in the conventional criteria (minimal standard terminology [MST]) for diagnosing chronic pancreatitis based on EUS features. These include (table 1):

Parenchymal abnormalities:

- Hyperechoic foci
- Hyperechoic strands
- Lobular contour
- Cysts

Ductal abnormalities:

- Main duct dilation
- Duct irregularity
- Hyperechoic margins
- Visible side branches
- Stones

The accuracy of EUS in diagnosing chronic pancreatitis varies based on the cutoff selected. In many studies, three or more features are considered abnormal [19]. Using this cutoff, sensitivity is high (>80 percent), but specificity is unacceptably low (around 50 percent). A higher cutoff of five or more improves specificity (>80 percent), but sensitivity drops substantially. The diagnosis of chronic pancreatitis cannot be made based on EUS alone, especially when the findings are in the indeterminate range without confirmatory testing (secretin pancreatic function testing) or supportive evidence (eg, genetic testing for hereditary pancreatitis, findings on MRCP, pancreatic steatorrhea by fecal elastase testing). (See "Endoscopic ultrasound in chronic pancreatitis", section on 'Threshold to diagnose or exclude CP by EUS' and "Pancreatitis associated with genetic risk factors".)

Unlike other diagnostic tests, however, pancreatic tissue can be sampled at the time of EUS (picture 1). While pancreatic core biopsy is possible, it is rarely done for diagnostic purposes, with the exception of rare patients with presumed autoimmune pancreatitis based on characteristic abnormalities on MRCP and a more subacute smoldering course [2]. (See "Autoimmune pancreatitis: Clinical manifestations and diagnosis", section on 'Imaging findings'.)

Direct pancreatic function testing — The function of the pancreas can be assessed by administering a stimulatory hormone (cholecystokinin to cause acinar cell secretion of digestive enzymes or secretin to cause ductal secretion of bicarbonate-rich fluid) and collecting the pancreatic secretion as it enters the duodenum. The secreted fluid is collected by an oroduodenal tube or an upper endoscope. While both cholecystokinin and secretin are commercially available, only secretin testing is performed in the United States and testing is limited to centers of expertise. This test involves the intravenous administration of the hormone secretin, which causes the pancreatic duct (and biliary duct) to secrete a bicarbonate-rich fluid. The fluid is collected continuously or sampled periodically, depending on the test procedure utilized. The concentration of bicarbonate in the fluid is measured. In most test protocols, a level >80 mEq/L at any measurement is considered normal. Direct pancreatic function test may be more accurate at identifying earlier stages of chronic pancreatitis [20,21]. (See "Exocrine pancreatic insufficiency", section on 'Direct pancreatic function tests'.)

Indirect pancreatic function testing — Measurement of pancreatic enzymes in stool is not commonly used for diagnosis of chronic pancreatitis, but rather to document possible exocrine pancreatic insufficiency in a patient with known pancreatic disease. Pancreatic enzymes can be measured in stool, and assays for chymotrypsin and elastase are commercially available. Elastase is most widely used and more accurate. Fecal elastase measurement is performed on a random stool sample, which should be solid or semisolid. The assay does not actually measure elastase-1 but instead measures chymotrypsin-like elastases. Levels <100 mcg/g stool are reasonably accurate for exocrine pancreatic insufficiency, while levels >100 but <200 mcg/g stool are indeterminate [22].

No diagnostic role for ERCP — Although ERCP gives the most detailed images of the pancreatic duct, it is no longer used for the diagnosis of chronic pancreatitis because of the availability of alternative imaging modalities and the associated risk of complications. In early-stage disease, the ductal changes may be minimal and the diagnosis may not be clear. In advanced chronic pancreatitis, a dilated and strictured pancreatic duct containing intraductal stones is pathognomonic. ERCP is reserved for therapy in chronic pancreatitis to remove stones or stent pancreatic duct strictures. (See "Chronic pancreatitis: Management", section on 'Endoscopic therapy'.)

Differential diagnosis — Other conditions that may mimic chronic pancreatitis include diseases causing pancreatic ductal obstruction (eg, pancreatic ductal adenocarcinoma, intraductal papillary mucinous neoplasms, and cystic neoplasms) [23]. Cross-sectional imaging can rule out these conditions and provide evidence of chronic pancreatitis. (See 'Cross-sectional

imaging' above and "Clinical manifestations, diagnosis, and staging of exocrine pancreatic cancer", section on 'Diagnostic approach'.)

DETERMINING THE ETIOLOGY

Determining the etiology of chronic pancreatitis requires a combination of the history of underlying risk factors and laboratory testing. However, a significant proportion of cases remain idiopathic. An approach to determining the etiology of chronic pancreatitis is discussed in detail separately. (See "Etiology and pathogenesis of chronic pancreatitis in adults", section on 'Approach to determine the underlying etiology'.)

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".)

SUMMARY AND RECOMMENDATIONS

- The syndrome of chronic pancreatitis results from exposure to risk factors (genetic and environmental) and is characterized by derangements in pancreatic function (digestive function or insulin secretion), and structural changes in the pancreas visible on imaging or endoscopic studies. However, many of the clinical features take time to manifest and may be absent in the early stages of chronic pancreatitis. (See 'Introduction' above.)

- Abdominal pain is the most common symptom in chronic pancreatitis. The pain is most commonly felt in the epigastric region and often radiates to the back. Nausea, vomiting, and anorexia are commonly associated with pain. The pain may be worse when recumbent and may have a postprandial exacerbation. Patients may experience constant pain of variable severity with periodic exacerbations, or ongoing relentless and severe pain. As the chronic pancreatitis progresses, amylase or lipase levels may not be elevated with pain flares. (See 'Abdominal pain' above.)

- Fat maldigestion produces steatorrhea and patients may note oily or floating stool but may not have diarrhea. Weight loss may occur, especially if pain also limits oral intake. While most patients with chronic pancreatitis have significant symptoms, a small subset of patients with chronic pancreatitis will not have symptoms or identifiable clinical

consequences. In such patients, the diagnosis of chronic pancreatitis may be made incidentally by cross-sectional imaging. (See 'Steatorrhea' above and 'Asymptomatic' above and 'Imaging findings' above.)

- Chronic pancreatitis should be suspected in patients with chronic abdominal pain and/or a history of relapsing acute pancreatitis, symptoms of pancreatic exocrine (diarrhea, steatorrhea, or weight loss) insufficiency, or pancreatogenic diabetes. Occasionally, patients with chronic pancreatitis are asymptomatic and the diagnosis is suspected incidentally on imaging. (See 'Diagnosis' above.)

- Cross-sectional imaging with high-quality computed tomography scan using multidetector technology and a pancreatic protocol, or magnetic resonance imaging with magnetic resonance cholangiopancreatography are the best initial diagnostic tests for chronic pancreatitis. In patients with suspected chronic pancreatitis, but inconclusive cross-sectional imaging, we perform either a direct pancreatic function test with secretin, if available, or endoscopic ultrasound. (See 'Diagnosis' above.)

- The most common complications of chronic pancreatitis include metabolic consequences of exocrine insufficiency (particularly osteopenia and osteoporosis), pancreatogenic (type 3c) diabetes, and opioid dependency due to the treatment of pain. Other less frequent complications include pseudocyst formation, bile duct or duodenal obstruction, pancreatic ascites or pancreatic pleural effusion, splenic vein thrombosis, arterial pseudoaneurysms, small intestinal bacterial overgrowth, gastroparesis, and pancreatic ductal adenocarcinoma. (See 'Complications of chronic pancreatitis' above.)

REFERENCES

1. Majumder S, Chari ST. Chronic pancreatitis. Lancet 2016; 387:1957.
2. Gardner TB, Adler DG, Forsmark CE, et al. ACG Clinical Guideline: Chronic Pancreatitis. Am J Gastroenterol 2020; 115:322.
3. Vege SS, Chari ST. Chronic Pancreatitis. N Engl J Med 2022; 386:869.
4. Whitcomb DC, Frulloni L, Garg P, et al. Chronic pancreatitis: An international draft consensus proposal for a new mechanistic definition. Pancreatology 2016; 16:218.
5. Whitcomb DC, Shimosegawa T, Chari ST, et al. International consensus statements on early chronic pancreatitis. Recommendations from the working group for the international consensus guidelines for chronic pancreatitis in collaboration with The International

Association of Pancreatology, American Pancreatic Association, Japan Pancreas Society, PancreasFest Working Group, and European Pancreatic Club. Pancreatology 2018; 18:516.

6. Wilcox CM, Yadav D, Ye T, et al. Chronic pancreatitis pain pattern and severity are independent of abdominal imaging findings. Clin Gastroenterol Hepatol 2015; 13:552.
7. Machicado JD, Amann ST, Anderson MA, et al. Quality of Life in Chronic Pancreatitis is Determined by Constant Pain, Disability/Unemployment, Current Smoking, and Associated Co-Morbidities. Am J Gastroenterol 2017; 112:633.
8. Drewes AM, Bouwense SAW, Campbell CM, et al. Guidelines for the understanding and management of pain in chronic pancreatitis. Pancreatology 2017; 17:720.
9. Olesen SS, Büyükuslu A, Køhler M, et al. Sarcopenia associates with increased hospitalization rates and reduced survival in patients with chronic pancreatitis. Pancreatology 2019; 19:245.
10. de Pretis N, Amodio A, Frulloni L. Hypertriglyceridemic pancreatitis: Epidemiology, pathophysiology and clinical management. United European Gastroenterol J 2018; 6:649.
11. Hart PA, Zen Y, Chari ST. Recent Advances in Autoimmune Pancreatitis. Gastroenterology 2015; 149:39.
12. Issa Y, Kempeneers MA, van Santvoort HC, et al. Diagnostic performance of imaging modalities in chronic pancreatitis: a systematic review and meta-analysis. Eur Radiol 2017; 27:3820.
13. Hart PA, Bellin MD, Andersen DK, et al. Type 3c (pancreatogenic) diabetes mellitus secondary to chronic pancreatitis and pancreatic cancer. Lancet Gastroenterol Hepatol 2016; 1:226.
14. Duggan SN, Smyth ND, Murphy A, et al. High prevalence of osteoporosis in patients with chronic pancreatitis: a systematic review and meta-analysis. Clin Gastroenterol Hepatol 2014; 12:219.
15. Martínez-Moneo E, Stigliano S, Hedström A, et al. Deficiency of fat-soluble vitamins in chronic pancreatitis: A systematic review and meta-analysis. Pancreatology 2016; 16:988.
16. Dominguez-Munoz JE, Drewes AM, Lindkvist B, et al. Recommendations from the United European Gastroenterology evidence-based guidelines for the diagnosis and therapy of chronic pancreatitis. Pancreatology 2018; 18:847.
17. Frøkjær JB, Akisik F, Farooq A, et al. Guidelines for the Diagnostic Cross Sectional Imaging and Severity Scoring of Chronic Pancreatitis. Pancreatology 2018; 18:764.
18. Tirkes T, Shah ZK, Takahashi N, et al. Reporting Standards for Chronic Pancreatitis by Using CT, MRI, and MR Cholangiopancreatography: The Consortium for the Study of Chronic

Pancreatitis, Diabetes, and Pancreatic Cancer. Radiology 2019; 290:207.

19. Conwell DL, Lee LS, Yadav D, et al. American Pancreatic Association Practice Guidelines in Chronic Pancreatitis: evidence-based report on diagnostic guidelines. Pancreas 2014; 43:1143.
20. Kothari D, Ketwaroo G, Freedman SD, Sheth SG. The Impact of Risk Factors of Chronic Pancreatitis on Secretin Pancreatic Function Testing: Results of a 20-Year Study. Pancreas 2017; 46:887.
21. Kothari D, Ketwaroo G, Sawhney MS, et al. Comparison of Combined Endoscopic Ultrasonography and Endoscopic Secretin Testing With the Traditional Secretin Pancreatic Function Test in Patients With Suspected Chronic Pancreatitis: A Prospective Crossover Study. Pancreas 2017; 46:770.
22. Vanga RR, Tansel A, Sidiq S, et al. Diagnostic Performance of Measurement of Fecal Elastase-1 in Detection of Exocrine Pancreatic Insufficiency: Systematic Review and Meta-analysis. Clin Gastroenterol Hepatol 2018; 16:1220.
23. Whitcomb DC, Shimosegawa T, Chari ST, et al. International consensus statements on early chronic Pancreatitis. Recommendations from the working group for the international consensus guidelines for chronic pancreatitis in collaboration with The International Association of Pancreatology, American Pancreatic Association, Japan Pancreas Society, PancreasFest Working Group and European Pancreatic Club. Pancreatology 2018; 18:516.

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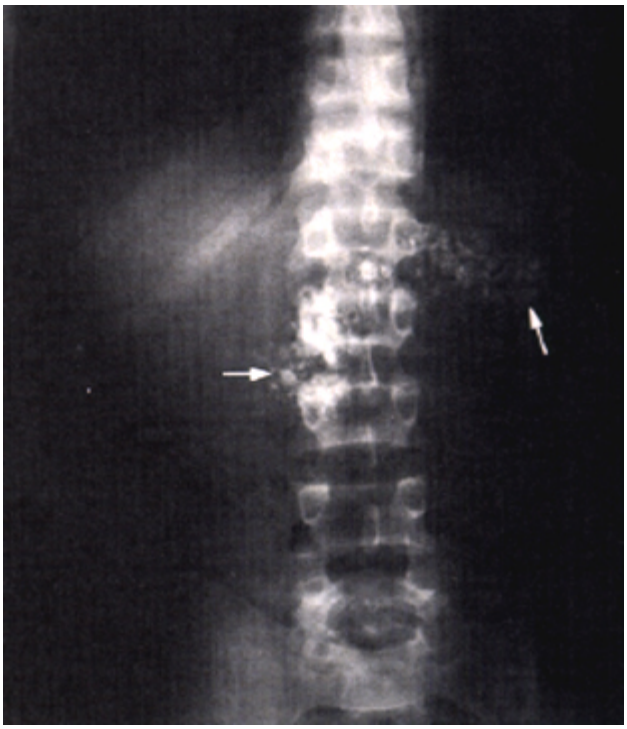
GRAPHICS

Pancreatic calcifications



Plain film of the abdomen shows pancreatic calcifications (arrows) in a patient with chronic pancreatitis.

With permission from: Steer ML, Waxman I, Freedman SD. N Engl J Med 1995; 332:1482.

Graphic 52624 Version 7.0

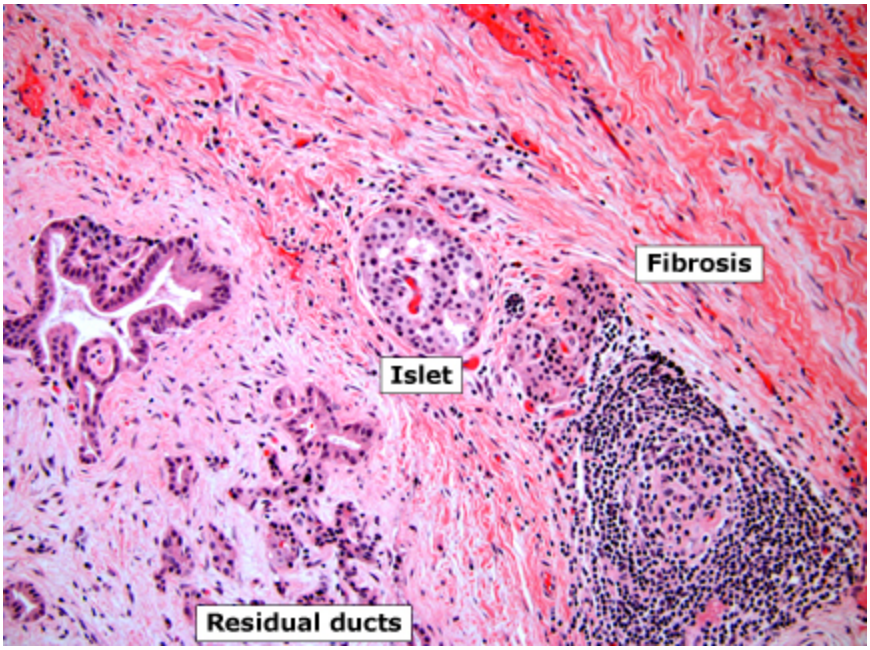
CT findings in chronic pancreatitis

Grade	CT findings
Normal	No abnormal findings on a good-quality study visualizing the entire gland
Equivocal	One of the following: ▪ Mild dilatation of the pancreatic duct (2 to 4 mm) in the body of the gland ▪ Gland enlargement ≤2-fold normal
Mild-moderate	One of the preceding findings plus at least one of the following: ▪ Pancreatic duct dilatation (>4 mm) ▪ Pancreatic duct irregularity ▪ Cavity (ies) <10 mm ▪ Parenchymal heterogeneity ▪ Increased echogenicity of duct wall ▪ Irregular contour of the head or body ▪ Focal necrosis or loss of parenchyma
Severe	Mild/moderate features plus one or more of the following: ▪ Cavity (ies) >10 mm ▪ Intraductal filling defects ▪ Calculi/pancreatic calcification ▪ Ductal obstruction (stricture) ▪ Severe duct dilatation or irregularity ▪ Contiguous organ invasion

CT: computed tomography.

Graphic 127361 Version 3.0

Chronic pancreatitis



H and E stained section of pancreas from a patient with chronic pancreatitis. Pancreatic biopsy showing extensive fibrosis, chronic inflammation, residual ductal structures, and a residual islet. There is no acinar tissue remaining.

Courtesy of Larry Brown, MD.

Graphic 76728 Version 2.0

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