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Advanced Imaging of Chronic Pancreatitis

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Abstract Chronic pancreatitis is characterized by continuing inflammation, destruction, and irreversible morphological changes in the pancreatic parenchyma and ductal anatomy. These changes lead to chronic pain and/or loss of function. Although these definitions are simple, the clinical diagnosis of chronic pancreatitis remains difficult to make, especially for early disease. Routine imaging modalities such as transabdominal ultrasound and standard CT scans are insensitive for depicting early disease, and detect only advanced chronic pancreatitis. Advances in imaging modalities including CT, MRI with gadolinium contrast enhancement, MRI with magnetic resonance cholangiopancreatography (MRI/MRCP), MRI/MRCP with secretin-stimulation (S-MRCP), endoscopic retrograde cholangiopancreatography (ERCP), and endoscopic ultrasound (EUS) allow earlier diagnosis of chronic pancreatitis. This article reviews the recognized findings, advantages, and disadvantages of the various imaging modalities in the management of chronic pancreatitis, specifically CT, MRI with or without MRCP and/or S-MRCP, ERCP, and EUS.

Keywords Chronic pancreatitis · Computed tomography · Endoscopic retrograde cholangiopancreatography · Magnetic resonnance imaging · Secretin stimulated magnetic resonnance cholangiopancreatography · Endoscopic ultrasound

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Introduction

Irrespective of its etiology, chronic pancreatitis is described by irregular fibrosis, destruction, and distortion of the pancreatic ducts with loss of exocrine and endocrine parenchyma. The most common cause of chronic pancreatitis in the western world is alcohol abuse. Other cited causes include hereditary, tropical, autoimmune, and idiopathic pancreatitis, the latter accounting for 10% to 30% of cases. Conclusive clinical diagnosis of chronic pancreatitis is achieved in advanced disease when exocrine insufficiency occurs and with the destruction of more than 90% of the gland. In mild disease and with absence of a true gold standard, the clinical diagnosis of chronic pancreatitis can be difficult. In chronic pancreatitis, tissue diagnosis is not commonly obtained. Unlike the liver and kidney, random pancreas biopsies are discouraged. The perceived risk of causing acute pancreatitis or causing other pancreatic complications such as fistula, pseudocyst, or hemorrhage discourages sampling of pancreatic tissue. Furthermore, histopathologic specimens are often nonspecific for chronic inflammation, and may reflect nonrepresentative sampling in cases of focal rather than diffuse inflammatory gland changes. In addition, normal aging may induce changes in pancreatic tissue similar to chronic pancreatitis. For these reasons, the use of histopathology in the diagnosis of chronic pancreatitis is rarely used [1, 2•, 3]. Various imaging modalities are then being advocated for the early detection, staging, and management of chronic pancreatitis. Earlier detection of chronic pancreatitis may expedite intervention. In addition to diagnosis, imaging modalities of the pancreas may be used to detect severity and complications of the disease. Screening imaging modalities such as transabdominal ultrasound and standard abdominal CT scan are insensitive and only detect advanced disease. Newer imaging modalities such as pancreatic CT protocol with a multi-

detector CT scanner, MRI with or without magnetic resonance cholangiopancreatography (MRI/MRCP) and/or secretin stimulation (S-MRCP), endoscopic retrograde cholangiopancreatography (ERCP), and endoscopic ultrasound (EUS) have improved sensitivity for detection of early disease, allowing earlier management interventions/decisions [1].

Computed Tomography

The use of CT in chronic pancreatitis was first reported in 1976 [4]. Now, spiral CT is the most widely used modality to image inflammatory and neoplastic diseases of the pancreas [1]. On early noncontrast CT protocols, findings of chronic pancreatitis were insensitive and nonspecific. These included calcification, gland atrophy, irregularities in the pancreatic parenchyma or duct outline, and heterogeneity of the composition of the pancreas. These findings were not helpful in making early management decisions (endoscopic, surgical, or other) because by the time these changes were noted, disease was already advanced [5, 6].

In 1983, the ERCP Cambridge criteria were introduced to aid in the diagnosis of chronic pancreatitis based on ductal changes. CT was used as an adjunct to the ERCP criteria by highlighting the parenchymal changes seen with chronic pancreatitis. The Cambridge classification of 1983 for chronic pancreatitis using ERCP and CT includes the following [1, 7•]. Cambridge 1 (normal pancreas): good-quality study visualizing the whole gland without abnormal signs and with a main pancreatic duct less than 2 mm in diameter and no side branch ectasia. Cambridge 2 (equivocal findings): main pancreatic duct 2–4 mm in diameter, a gland one to two times normal size, or heterogeneous appearance of the pancreatic parenchyma with less than three abnormal side branches (ectasia or clubbing). Cambridge 3 (mild disease): normal main pancreatic duct with more than three abnormal side branches. Cambridge 4 (moderate disease): cyst less than 10 mm, duct irregularity, focal acute necrosis/pancreatitis, parenchymal heterogeneity, increased echogenicity of the ductal wall (or abnormalities in the main pancreatic duct), and/or contour irregularity of the pancreatic head/body along with findings of Cambridge 3. And finally, Cambridge 5 (severe disease), which includes all of the changes noted in mild-moderate disease along with one or more of the following: at least one cyst greater than 10 mm, intraductal filling defect(s), calculi/pancreatic calcification(s), duct obstruction/stricture(s), main pancreatic duct dilatation or irregularity, gross gland enlargement (>2 × normal), and/or contiguous (diffuse) organ involvement and/or more than three abnormal side branches. It is important to note that parenchymal changes associated with advanced chronic pancreatitis (especially Cambridge 4 and 5) are more readily seen on CT than on ERCP. Reported sensitivities for the use of CT in the diagnosis of chronic pancreatitis are 60% up to 95%, with CT being more sensitive in advanced disease (Cambridge 5). This implies a normal CT is frequently seen in patients with early chronic pancreatitis (Cambridge 2 and 3). Parenchymal findings for chronic pancreatitis may also be seen on transabdominal ultrasound; however, ultrasound is also insensitive for early disease and is more operator dependent than CT [1, 7•].

With further development of CT technology, including the use of contrast injection and the capture of images pre-contrast, during the portal venous phase, and during the arterial phase and with contiguous thin overlapping slices through the pancreatic bed, the sensitivity for the detection of chronic pancreatitis and pancreatic cancer has improved. Chronic pancreatitis exhibits delayed enhancement on CT because of fibrosis [1]. Moreover, with CT, the main pancreatic duct is sometimes reportedly dilated on its own and/or in association with common bile duct dilatation (double duct sign). One retrospective study showed that in 77 patients with ductal abnormalities on CT, 51 had isolated main pancreatic duct dilatation, of which 37 (73%) had chronic pancreatitis and two (4%) had pancreatic carcinoma. The 26 remaining patients had double duct dilatation, of which 15 (58%) had pancreatic carcinoma and three (12%) had chronic pancreatitis [8].

Minimal literature exists correlating histopathology to diagnosis of chronic pancreatitis using CT. Kusano et al. [9] showed that in patients with chronic pancreatitis diagnosed by contrast-enhanced spiral CT, fibrous stroma is demonstrable on histopathology; however, the sample size was small (N=2). In 56 consecutive patients with documented moderate-severe chronic pancreatitis by histology or ERCP (the gold standard for ductal changes), a retrospective analysis showed that contrast-enhanced CT detects the following changes: dilatation of the main pancreatic duct in 68%, atrophy in 54%, calcifications in 50%, fluid collections in 30%, pancreatic enlargement in 30%, biliary ductal dilatation in 29%, and alterations in the peripancreatic fat and fascia in 16%. The findings were nondiagnostic in 7%.

Non-neoplastic complications of chronic pancreatitis include pseudocyst(s), arterial pseudoaneurysm(s), splenic vein thrombosis, and/or biliary obstruction/dilatation [3]. CT has been supplanted by high-quality MRI/MRCP for evaluating the pancreatic duct and for diagnosing or detecting the severity of chronic pancreatitis; however, CT continues to be used for detecting its complications [10••]. The advantages of CT are availability, and the ability to scan the entire pancreatic parenchyma with minimal respiratory artifacts. It is also less costly and less operator dependent than other modalities (eg, ERCP and EUS). The disadvantages of CT include ionizing radiation, contrast-

induced nephropathy, and the inability to perform the test in patients with an iodine allergy [1, 11]. The differentiation between pancreatic cancer and focal mass-forming chronic pancreatitis (a well-known risk factor for pancreatic ductal adenocarcinoma) remains difficult [1, 5, 12]. Recent advancement in CT protocols—including targeted triple-phase contrast-enhanced imaging of the pancreas—improved this differentiation [13]. Based on available data, one can safely conclude that CT confidently detects only patient(s) with severe/advanced chronic pancreatitis [3].

Endoscopic Retrograde Cholangiopancreatography

Most pancreatologists have considered ERCP the gold standard for the morphological diagnosis and for the staging of chronic pancreatitis in the absence of histopathology [1]. In 1983, a Cambridge classification was adopted for ERCP staging of chronic pancreatitis at the international workshop (see CT section, above). The ERCP classification included side-branch pathology not previously noted on CT, an earlier feature of the disease. This classification increased sensitivity for earlier detection of chronic pancreatitis [1, 3, 7•]. A retrospective analysis that included 31 patients with chronic pancreatitis who underwent ERCP and histopathology examination after surgical resection demonstrated high correlation with pathologic specimens using the Cambridge classification. The ERCP findings and histopathology reports correlated in 23 (74%) patients, whereas findings did not associate in eight (26%). The early disease group's (9 of 31 classified as normal, equivocal, or mild) ERCP findings correlated with histopathology in six of nine patients (67%). Patients classified as moderate and marked had a correlation of 17 of 22 (77%) [14•]. Early studies also showed greater than 90% sensitivity and specificity for ERCP in the diagnosis of chronic pancreatitis; however, many now feel that these results are overestimated because of the lack of a gold standard for comparison and because of the population of patients investigated [15].

ERCP is a useful test for examining pancreatic ductal changes [3, 11]. It is also useful for the diagnosis of chronic pancreatitis, and for determining the origin of pseudocysts and the location of strictures in neoplasms, the former of which communicate with the main pancreatic duct in 50% of cases [3, 16]. If a main pancreatic duct stricture is seen in the setting of chronic pancreatitis, the differential diagnosis includes malignancy versus benign strictures secondary to fibrosis. In general, benign strictures are shorter, smoother, and more symmetrical than those caused by a carcinoma [17].

ERCP is strongly operator dependent and successful cannulation of the pancreatic duct is only obtained 70% to 91% of the time. ERCP requires sedation, which increases the risk for cardiopulmonary complications in the setting of prolonged (>30 min) small-bowel intubation. It is also expensive, and invasive with a morbidity rate of 1% to 7% (eg, post-ERCP pancreatitis) and a mortality rate of 0.2%. These problems do not occur with the use of MRI-based techniques because of the lack of direct instrumentation of the main pancreatic duct and the lack of need for conscious sedation and/or monitored anesthesia care [1].

Magnetic Resonance Imaging

MRI techniques have made tremendous advances recently and, given their wide versatility, are rapidly emerging as the imaging modalities of choice for pancreatic diseases. Interpretation of MRI images was limited in the past by artifacts caused by bowel peristalsis, respiratory motion, and cardiac pulsation. However, with improved technology, significant improvements have occurred in the length of time for image acquisition and in the signal-to-noise ratio [18]. Pancreatic parenchyma can be best evaluated on noncontrast, T1-weighted, fat-suppressed images and arterial phase postcontrast images. Normal pancreas reveals high signal on T1-weighted sequences because of abundant protein (digestive enzymes and hormones) within the gland. With the use of fat suppression, the contrast between suppressed retroperitoneal fat and the gland increases, increasing sensitivity to detect pathology when present. The pancreatic gland has a rich capillary blood supply; therefore, in the arterial phase images, an arterial capillary blush is expected in the normal gland [1, 2•, 18].

The following morphologic changes in the pancreatic parenchyma occur in early chronic pancreatitis and may be well visualized with MRI. First, the anteroposterior dimensions of the gland diminish segmentally or diffusely due to acinar atrophy. Second, the pancreatic signal decreases on T1-weighted fat suppressed images due to loss of exocrine capability of the gland. Finally, the perfusion of the pancreatic gland with gadolinium contrast enhancement is delayed. Normally, the pancreas peaks arterially with contrast enhancement and then contrast washes out in a linear fashion in the subsequent venous phase. Because of the presence of fibrosis in chronic pancreatitis, capillary blood flow is impaired and the gland reaches its maximum enhancement in the venous phases in gradual fashion [19, 20••, 21••]. With gadolinium contrast enhancement, dynamic MRI of the pancreas has a reported 79% sensitivity and 75% specificity for the detection of chronic pancreatitis, a feat that older imaging modalities (CT and transabdominal ultrasound) were poor in achieving [1, 3].

MRCP images are acquired with the use of T2-weighted images. Fluid has a bright signal on T2-weighted images and with the suppression of intraabdominal fat, fluid content of the biliary system becomes more pronounced.

Thus, standard MRCP images are obtained without intravenous or intrabiliary contrast injection. MRCP findings in chronic pancreatitis include one or more of the following: biliary and pancreatic ductal dilatation, strictures, irregularities in the main pancreatic duct, sacculation, and/or ectasia of the secondary (side branches) pancreatic ducts. Modified MRCP Cambridge criteria for chronic pancreatitis are Cambridge 1 (normal pancreas), in which the side branches and main pancreatic ducts are normal. In Cambridge 2 (equivocal findings), dilatation/obstruction of less than three side branches occurs with a normal main pancreatic duct. In Cambridge 3 (mild disease) criteria, the ducts exhibit dilatation/obstruction of greater than three side branches with a normal main pancreatic duct (Fig. 1). Cambridge 4 (moderate disease) criteria include Cambridge 3 criteria plus stenosis and dilatation of the main pancreatic duct. Finally, Cambridge 5 (severe disease) criteria include Cambridge 3 and 4 criteria plus additional obstructions, cysts, stenosis of the main pancreatic duct, and calculi [2•].

Using T1-weighted pancreatic MRI sequences with contrast enhancement and fat suppression also allows for detection of pancreatic adenocarcinoma. Pancreatic adenocarcinoma appears as a low signal-intensity mass relative to a normal background parenchyma. This modality also allows for detection of liver metastases. MRI and CT have relatively similar accuracies (about 90% to 100%) for the detection of pancreatic cancer. MRI is superior to CT in detecting tumor and metastases, whereas contrast-enhanced CT is superior for detecting vascular involvement [22].

MRI/MRCP findings of chronic pancreatitis (including pancreatic size, arterial enhancement, and parenchymal signal) appear to correlate well with exocrine function as measured by fecal elastase-1, as investigated by Bilgin et al. [19] in 81 patients with suspected chronic pancreatitis.

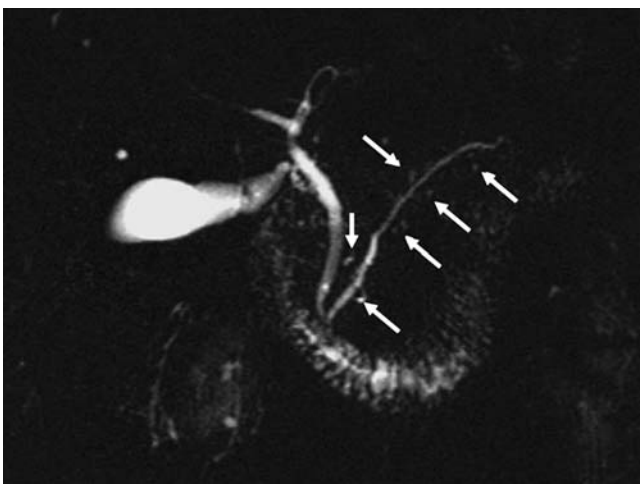

Fig. 1 Magnetic resonance cholangiopancreatography image of a patient with chronic pancreatitis showing multiple side-branch ectasias (>3) of the pancreatic duct consistent with grade 3 Cambridge classification

Moreover, in a retrospective analysis, exocrine pancreatic function testing (ePFT) with secretin stimulation during upper endoscopy of 32 patients also suspected to have chronic pancreatitis clinically and normal standard imaging (CT or ultrasound) showed that in 8 of the 23 with normal ePFT, an abnormal MRI/MRCP was seen. This implies that MRI/MRCP findings of chronic pancreatitis (whether focal or diffuse) may be present even with normal ePFT. This is a testament to the high sensitivity of this imaging modality [19, 20••]. Agreement between MRCP and ERCP was noted in several studies with regard to ductal narrowing/stricturing of the main pancreatic duct and its side branches, ductal dilatation, pseudocysts, and filling defects [3]. MRI was poor in the detection of calcifications because of their signal void. Calcifications are more readily seen on CT and/or ERCP [1, 3, 18]. Finally, MRI/MRCP is superior to CT in the detecting complications of chronic pancreatitis, specifically, pseudocysts, fistula formation (with the peritoneal cavity or with the pleural space), biliary dilatation (especially distally in the pancreatic head), and vascular complications (frequently associated with higher morbidity and mortality) [1].

Diffusion-weighted MR imaging (DWI) is a T2-weighted modality that assesses the random motion of protons in water. This modality recently made advancements in pancreatic imaging [23•]. In this modality, the term “apparent diffusion coefficient” (ADC) value is used to quantify the mean volume diffusion contributions of intracellular, extracellular, and vascular water protons [23•]. For instance, in pancreatic cystic lesions, the ADC is high owing to the large presence of water molecules with increased diffusion. In chronic pancreatitis, because of the presence of fibrosis, there is diffusion restriction and the ADC is low. In most instances, diffusion restriction is not discernible because the entire gland is homogeneously fibrotic. When a focal region is involved, the finding is more prominent. On the other hand, DWI and ADC values are variable in cancer, allowing for this modality to differentiate between areas of mass forming focal pancreatitis and pancreatic carcinoma. The differentiation between focal chronic pancreatitis and pancreatic cancer is a drawback in other imaging modalities (eg, CT, ERCP, or transabdominal ultrasound) [1, 23•, 24•].

In summary, MRI has several advantages over other modalities, including increased sensitivity for detection and characterization of early chronic pancreatitis, differentiation of focal fatty infiltration of the pancreas from focal pancreatitis and tumor, characterization of complex peripancreatic fluid collections, characterization of any associated liver lesions or fluid collections, evaluation of biliary ductal and pancreatic ductal changes associated with chronic pancreatitis, lack of ionizing radiation, and relatively safe use in renal impairment and in the setting of an iodinate contrast allergy [1, 3].

MRI/MRCP with Secretin Stimulation

Older imaging modalities such as ERCP and CT were limited in their ability to detect early chronic pancreatitis; however, advances have been made in evaluation of the ductal morphology and pancreatic function with the use of secretin stimulation [3, 18]. In contrast to ERCP, S-MRCP highlights ductal changes and provides diagnostic information about pancreatic exocrine function [1]. S-MRCP involves human secretin given as an intravenous bolus based on body weight with T2-weighted MRCP sequences taken every 15–30 s for 15 min after the infusion. Secretin is a hormone produced by S-cells in the small intestine that stimulates the pancreas to secrete bicarbonate and fluid. This allows improved anatomical delineation of the main pancreatic duct and side branches and permits assessment of the synthetic capacity of the exocrine pancreas based on the duodenal filling volume [3, 21••]. There appears to be good correlation between bicarbonate concentration in the duodenum and duodenal filling volume after secretin-stimulation [24•]. For exocrine function, grading of duodenal filling is as follows: grade 0, no fluid is observed; grade 1, filling is limited to the duodenal bulb; grade 2, fluid fills to the second duodenum; and grade 3, fluid fills beyond the second duodenal knee. Decreased exocrine function is suggested by any duodenal filling grade less than 3. In addition, patients with reduced duodenal filling are 17.6 times more likely to have depressed pancreatic exocrine function than those patients with normal duodenal filling.

Pancreatic duct compliance (PDC) or the change in the pancreatic duct diameter before and after secretin stimulation is also a reliable indirect measure of chronic pancreatitis. An increase in pancreatic duct diameter is expected with secretin stimulation. PDC expressed as a percentage of variation of duct diameter from baseline is reduced in chronic pancreatitis secondary to fibrosis (32.2%) as compared with normal controls (66.5%) [1, 3, 21••, 25, 26]. Rapid injection of secretin gives a secretory peak after 4 min, morphologically documented by transient dilatation of the main pancreatic duct. The main duct then returns to baseline after 10 min. In addition, from 3 min to 9 min after injection, the frequency and amplitude of contractions of the sphincter of Oddi increase. Persistent dilatation of the main pancreatic duct for more than 15 min after secretin injection has been reported to indicate sphincter of Oddi dysfunction and/or early dysfunction in the distal main pancreatic duct as with chronic pancreatitis [1, 27]. In patients with equivocal CT or transabdominal ultrasound findings and with clinical suggestion of chronic pancreatitis, early disease may be suggested by reduced duodenal filling and/or diminished pancreatic duct compliance [3].

Estimated pancreatic exocrine function on S-MRCP may be abnormal in patients with a normal ERCP. Discordance can be found in 27% of cases. However, follow-up of the discordant cases has shown evolution to chronic pancreatitis, suggesting that this imaging modality may be helpful in the diagnosis of early chronic pancreatitis [1, 3]. Secretin-stimulation enhances the diagnostic accuracy of MRCP by enhanced detection of minor changes in the pancreatic duct, pancreatic parenchyma, and pancreatic outflow dynamics. The visualization of the pancreatic duct side branches improved from 71% to 100% in severe chronic pancreatitis and from 4% to 63% in mild-moderate disease. Moreover, 92% specificity is reported with S-MRCP for mild chronic pancreatitis [27]. In 33% of asymptomatic patients with suspected chronic pancreatitis and with abnormal chemistries, an abnormal S-MRCP was noted. However, the clinical significance is still unknown because some of these changes may be attributed to age or may be nonspecific. More prospective analyses are needed [28].

Endoscopic Ultrasound

Endoscopic ultrasound is a diagnostic test developed in the 1980s for improving imaging of the pancreas and evaluating patients with chronic pancreatitis. EUS criteria for chronic pancreatitis can be divided into parenchymal and pancreatic ductal findings (Table 1) [29••, 30•, 31].

The capability of EUS criteria in diagnosing chronic pancreatitis was rigorously compared with the ERCP Cambridge criteria. A prospective analysis in 126 patients found that EUS sensitivity was greater than 85% and specificity was less than 60% when fewer than three EUS criteria were met. However, when more than five criteria were met, then specificity increased to greater than 85% [30•]. In a prospective analysis, Varadarajulu et al. [32•] evaluated 21 of 42 patients who underwent pancreatic surgery and were diagnosed with chronic pancreatitis by histopathology. CT was nondiagnostic in all patients. EUS revealed 90.5% sensitivity, 85.7% specificity, and 88.1% accuracy when more than four EUS criteria for chronic pancreatitis were met [32•]. Another retrospective analysis showed that EUS exhibited features of chronic pancreatitis in 13 patients who had a nondiagnostic prior CT, MRCP, and/or ERCP. All patients subsequently had a repeat CT after the EUS, and all CT scans were diagnostic for chronic pancreatitis, indicating progression of their disease [33].

Some problems with using EUS criteria to diagnose chronic pancreatitis include the use of ERCP as the gold standard for head-to-head comparison when some studies showed better sensitivity with EUS, implying irreproducible data. Second, the criteria have variable importance, for instance, the presence of calcifications is diagnostic without the presence of any other criteria. More disadvantages include the need for conscious sedation and/or monitored anesthesia care, interobserver variability in interpreting EUS images, operator dependence, and the need for a

Table 1 Endoscopic ultrasound criteria for the diagnosis of chronic pancreatitis

EUS finding	Description	Correlation	Strength
Parenchymal criteria			
Hyperechoic foci with shadowing	Length or width > 2 mm	Calcifications	Major A
Lobularity with honeycombing (>3 contiguous lobules)	Structures >5 mm with an enhancing rim and echo-poor center	None	Major B
Lobularity without honeycombing	As above	None	Minor
Hyperechoic foci without shadowing	As above	None	Minor
Hyperechoic strand(s)	Lines >3 mm long in at least two different directions with respect to the image plane	None	Minor
Intraparenchymal cyst(s)	None	None	Minor
Ductal criteria			
Main pancreatic duct calculi with shadowing	None	None	Major A
Irregular main pancreatic duct/ectatic contour	None	None	Minor
>3 dilated pancreatic duct side branches	None	Side branch ectasias	Minor
Main pancreatic ductal dilatation	>4 mm in head, >3.5 mm in body, and >1.5 mm in tail	None	Minor
Hyperechoic main pancreatic duct margin	Echogenic, distinct structure covering>50% of entire main pancreatic duct in body and tail	Ductal fibrosis	Minor
Stricture(s)	None	None	Minor

Findings "consistent" with chronic pancreatitis include: 1 major A criterion plus >3 minor criteria; 1 major A plus 1 major B criterion; and 2 major A criteria. Findings "suggestive" of chronic pancreatitis include: 1 major A criterion plus <3 minor features; 1 major B criterion plus >3 minor criteria; and >5 minor criteria. Findings "indeterminate" for chronic pancreatitis include: 3 or 4 minor criteria; and 1 major B criterion alone or with <3 minor criteria. "Normal" findings, not suggestive of disease in the pancreas, include < 2 minor criteria

well-trained on-site cytopathologist. These limitations increase the risks and/or costs of the procedure [1, 30•].

Agarwal et al. [34] showed that EUS can differentiate mass-forming chronic pancreatitis from pancreatic cancer by EUS with fine needle aspiration (EUS-FNA). In their retrospective analysis, 110 study patients underwent EUS or EUS-FNA for abnormal CT or MRI with an enlarged head of the pancreas or dilated pancreatic duct with or without dilation of the common bile duct. The study revealed an accuracy of 99.1% for EUS and/or EUS-FNA in diagnosing pancreatic neoplasm with a sensitivity of 88.8% and specificity of 100% [34, 35].

Conclusions

Despite advances in imaging modalities, early diagnosis of chronic pancreatitis remains elusive. CT remains a good study for complications of chronic pancreatitis and to visualize pancreatic calcifications in moderate-severe chronic pancreatitis. ERCP remains the gold standard for most pancreatologists for the evaluation of duct morphology and staging of chronic pancreatitis severity, to which newer modalities are still compared. EUS is an emerging modality for the evaluation of chronic pancreatitis but is limited by the need for endoscopy/sedation with their complications, operator dependence, lack of assessment of exocrine function, interobserver variability, and lack of reproducibility of study data. With EUS-FNA, however, it is possible to differentiate between focal mass-forming chronic pancreatitis and pancreatic cancer. MRI and MRI/MRCP are rapidly emerging as important tools for the evaluation of parenchymal and ductal abnormalities for the diagnosis, staging, and evaluation of complications of chronic pancreatitis. With the addition of secretin-stimulation and diffusion-weighted imaging sequences, exocrine function and a capability for differentiating between focal chronic pancreatitis and cancer are also respectively assessed, all in a noninvasive, "one-stop" fashion with good sensitivity in early disease. No other modality offers such versatility in the early management and possible early intervention for patients with suspected chronic pancreatitis.

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