

Imaging of an Indeterminate Pancreatic Mass

Anu Eapen¹ Anuradha Chandramohan¹ Reetu John¹ Betty Simon¹ Tharani Putta¹ A. J. Joseph²

¹Department of Radiology, Christian Medical College, Vellore, Tamil Nadu, India

²Department of Gastroenterology, Christian Medical College, Vellore, Tamil Nadu, India

Address for correspondence Anu Eapen, DMRD, DNB, Department of Radiology, Christian Medical College, Vellore, Tamil Nadu, India (e-mail: anuepn@yahoo.com).

J Gastrointestinal Abdominal Radiol ISGAR 2020;3:75–86

Abstract

Keywords

- CT
- MRI
- EUS
- imaging
- pancreatic masses
- indeterminate

Several conditions ranging from neoplasm to inflammatory disorders can present as a pancreatic mass on imaging. While classic imaging features help us make an accurate diagnosis, we are often confronted by indeterminate pancreatic masses that pose diagnostic dilemma. In this review, we describe the typical imaging features of common mass forming conditions of pancreas followed by description of atypical imaging appearances, which make a pancreatic mass indeterminate. We also emphasize on imaging features useful in differentiating one from the other.

Introduction

A mass lesion in the pancreas makes one consider the possibility of a neoplasm. Pancreatic ductal adenocarcinoma (PDAC) is the most common malignant neoplasm of the pancreas (85–95%) and has a poor prognosis.^{1,2} Typical imaging feature of PDAC on computed tomography (CT) or magnetic resonance imaging (MRI) is an ill-defined hypovascular mass.³ When imaging features are atypical, it could mimic other neoplasms or inflammatory conditions, which have better prognosis than a PDAC. Moreover, 5 to 10% of pancreatectomies performed for clinically suspected PDAC have alternative histopathological diagnosis, leading to needless major surgical procedure and related morbidity.^{4,5} This review aims to highlight ways to differentiate between various “mass forming” conditions of the pancreas that could pose a diagnostic dilemma. A brief summary of typical features of each condition is described first before description of atypical findings that may render the lesion indeterminate.

Imaging Modalities

Ultrasound is the first-line imaging modality but is of limited use in the evaluation of pancreatic mass. The presence of bowel gas obscuring retroperitoneum and body habitus are some of the limiting factors in the use of ultrasound.⁶

Computed Tomography

Multidetector CT (MDCT) is the most useful imaging modality in the evaluation of pancreatic mass lesions. MDCT has excellent spatial resolution and provides wide area of coverage. Scanning in the late arterial or pancreatic parenchymal phase (35–50 S), during which there is peak pancreatic enhancement, increases contrast between hypovascular pancreatic carcinoma and pancreas. The portal venous phase is useful in detecting liver metastasis. Advanced post processing techniques such as multiplanar reformation, maximum intensity projection, and volume rendering help in the assessment of local extent of a mass and its resectability.^{7,8}

Magnetic Resonance Imaging

MRI is a useful modality to identify small lesions that are not well delineated on CT, especially in the liver.⁷ Diffusion-weighted imaging is useful to further characterize a mass. MR cholangiopancreatogram (MRCP) helps to delineate relation of duct with respect to any mass and the presence of any ductal obstruction.

EUS and Biopsy

Endoscopic ultrasound (EUS) helps to examine the pancreas in close proximity, without intervening viscera. The ability to concurrently do a fine needle aspiration provides additional value. Characteristic patterns have been described in various solid and cystic pancreatic lesions (►Fig. 1), using two additional tools, contrast-enhanced

EUS, and elastography. The usefulness of EUS in patients with chronic pancreatitis is limited when there is significant calcification.

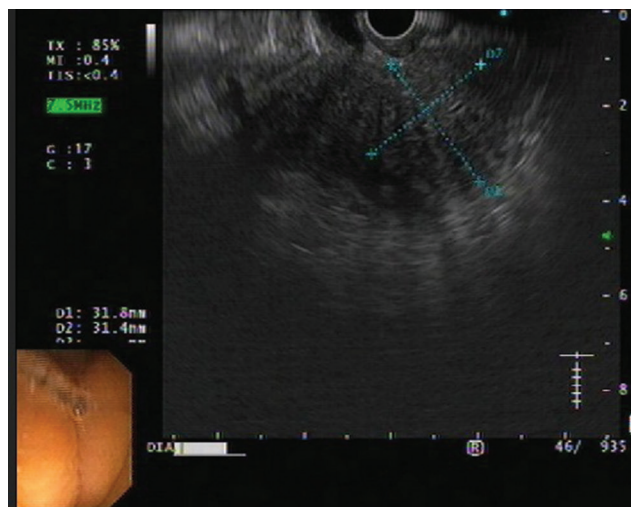


Fig. 1 Endoscopic ultrasound shows an ill-defined hypoechoic mass in the pancreas.

Pancreatic Ductal Adenocarcinoma

On CT, pancreatic carcinoma is a hypoattenuating mass, best appreciated in the pancreatic parenchymal phase, when there is peak parenchymal enhancement. The commonest site of involvement is the head of the pancreas (60–70%) with the remaining (10–20%) in the body and the tail (5–10%).¹ Lesions in the head causes an abrupt narrowing of the bile duct with proximal dilatation. Similarly, there is an abrupt cutoff of the pancreatic duct with upstream dilatation of the duct (double duct sign).¹ With advanced malignancy, tumor encasement of vessels, defined as tumor surrounding vessel, more than 180 degree of circumference, occurs. Similarly, loss of perivascular fat, vessel deformity, and thrombosis are the other features that suggest vessel infiltration.¹ Atrophy of the pancreas, upstream to the mass is common. Peripancreatic infiltration of adjacent organs and distant metastasis may also be seen.² In the presence of extensive calcifications in chronic pancreatitis, a small hypoattenuating mass can be missed. ▶**Fig. 2** and ▶**Fig. 3** are examples of classical imaging appearances of PDAC.

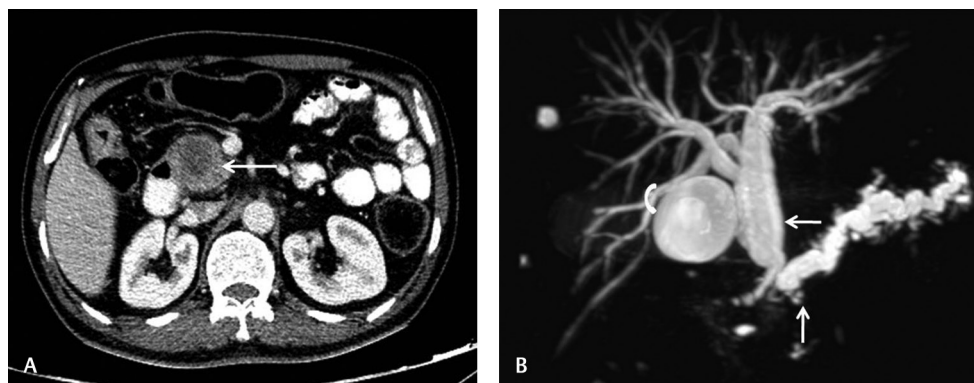


Fig. 2 Pancreatic adenocarcinoma in a 56-year-old man with severe abdominal pain, fever, and jaundice. (A) Postcontrast computed tomography axial sections in the portal phase showing a large hypoattenuating irregular mass (arrow) involving the pancreatic head. (B) Reconstructed magnetic resonance cholangiopancreatography (B) showing resultant abrupt narrowing of the common bile duct (horizontal arrow), and the main pancreatic duct (vertical arrow) with upstream dilatation (double duct sign); curved arrow indicates the gall bladder.

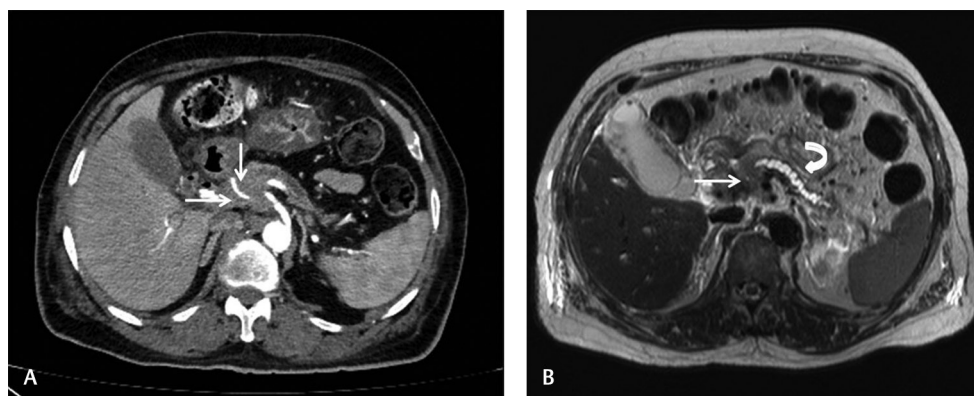


Fig. 3 Unresectable pancreatic adenocarcinoma in a 81-year-old man with abdominal pain. (A) Postcontrast computed tomography axial sections in the arterial phase showing a hypoattenuating ill-defined mass (horizontal arrow) involving the pancreatic head with encasement of the gastroduodenal artery (vertical arrow). (B) T2-weighted axial magnetic resonance imaging showing an intermediate signal intensity mass (arrow) causing upstream main pancreatic duct dilatation and pancreatic parenchymal atrophy (curved arrow).

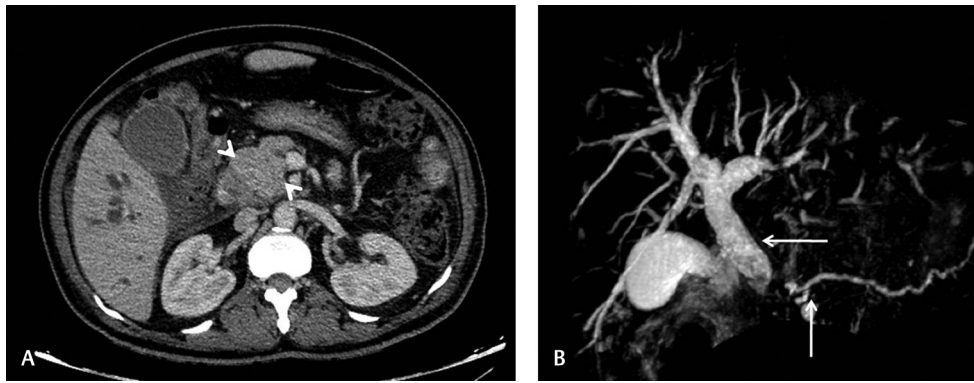


Fig. 4 Isoattenuating pancreatic adenocarcinoma in a 59-year-old man with obstructive jaundice and weight loss. (A) Contrast-enhanced computed tomography axial sections in the portal phase showing an isoattenuating mass (arrowheads) in the pancreatic head. (B) Reconstructed magnetic resonance cholangiopancreatography shows abrupt cutoff of the common bile duct (horizontal arrow) with upstream biliary dilatation and also cutoff of the main pancreatic duct (vertical arrow).

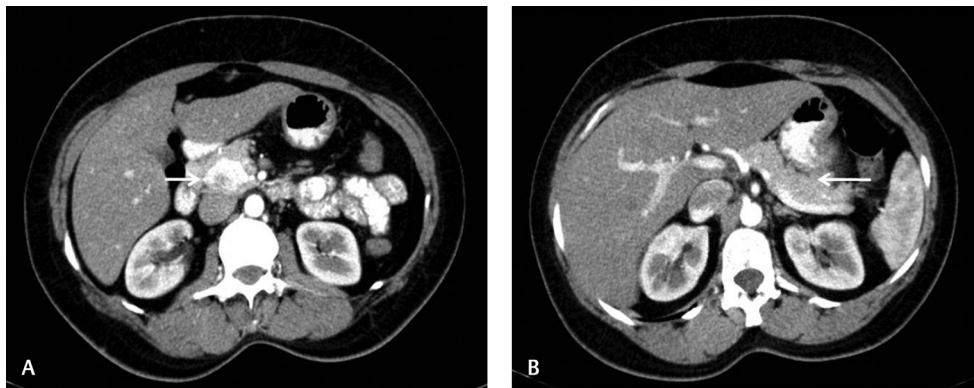


Fig. 5 Neuroendocrine tumor in a 37-year-old lady who presented with abdominal pain, vomiting; and diarrhea; she also had a history of breast malignancy. (A) Postcontrast computed tomography axial sections in the arterial phase show a well-defined intensely enhancing homogenous lesion (arrow) in the pancreatic head. (B) Normal pancreatic body and tail with no main pancreatic duct dilatation (arrow).

Atypical Features

In 10% of patients, the lesion is isoattenuating (► Fig. 4). Indirect evidence of malignancies, when the lesion is isoattenuating or small, are a convex contour of pancreas, ductal obstruction with “double duct sign,” vessel encasement, and mass effect.¹ Isoattenuating masses may be missed in the absence of these features. MRI is useful for detection of isoattenuating masses.

Other atypical features, when lesion is close to pancreaticoduodenal groove, include absence of pancreatic duct obstruction and proximal dilatation and absence of distal atrophy of the pancreas.⁹

Pancreatic Neuroendocrine Tumors

These arise from pluripotent pancreatic cells of the ductal/acinar system.^{10,11} These account for 1 to 2% of all pancreatic tumors. Most tumors occur sporadically, but 1 to 2% of these tumors occur with cancer predisposition syndromes, namely multiple endocrine neoplasia-1, von Hippel Lindau disease, neurofibromatosis-1, and tuberous sclerosis.¹²

Typical Features

On CT, these tumors are well circumscribed and hypervascular, with intense enhancement in the arterial phase (► Fig. 5).

Small tumors, less than 2 cm, are homogenous while larger tumors are heterogeneous with cystic degeneration and necrosis.¹³ Pancreatic neuroendocrine tumors (PNETs) rarely cause pancreatic duct obstruction and proximal duct dilatation.¹³ Liver metastases from PNETs are hypervascular.

Atypical Features

Pancreatic duct obstruction, if present, maybe due to small serotonin producing tumors due to the local effects of serotonin causing fibrosis and stricturing of pancreatic duct. Large tumors may also cause mass effect with ductal obstruction.¹³ ► Figure 6 shows an example of neuroendocrine tumor (NET) showing duct obstruction.

Nonhypervascular tumors may be mistaken for ductal adenocarcinoma. The absence of ductal obstruction and distal pancreatic atrophy favors neuroendocrine neoplasm over PDAC.¹³ ► Table 1 compares the imaging features of nonhypervascular NET and PDAC. Enhancement patterns on MRI have also been studied to differentiate between neuroendocrine neoplasm and PDAC. The presence of hyper or isoenhancement in the portal venous phase and a well-defined margin were useful imaging features of nonhypervascular NETs, to differentiate it from PDAC.¹⁴ Subset of pancreatic NET may be seen better on the portal venous phase and may appear as

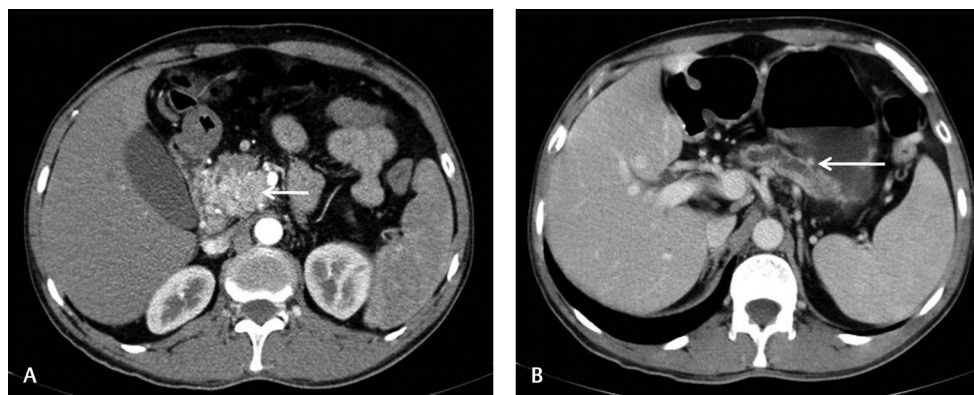


Fig. 6 Pancreatic neuroendocrine tumor in a 52-year-old man with epigastric discomfort and loss of weight. (A) Postcontrast computed tomography (CT) axial sections in the arterial phase shows a large hyperenhancing mass (arrow) involving the uncinate process of the pancreas. (B) Postcontrast CT in the portal phase at a more cranial level shows upstream main pancreatic duct dilatation with pancreatic parenchymal atrophy (arrow).

Table 1 Differentiating features between pancreatic ductal adenocarcinoma and nonhypervascular neuroendocrine tumor

	PDAC	PNET
Portal venous phase enhancement	Hypoenhancing	Iso- to hyper-enhancing
Margins	Ill defined	Well defined
Pancreatic duct obstruction	Common	Rare
Vessel encasement	Common	Rare
Distal pancreatic atrophy	Common	Rare

Abbreviations: PDAC, pancreatic ductal adenocarcinoma; PNET, pancreatic neuroendocrine tumor.

pedunculated or exophytic mass from the pancreas.⁷ Localizing isodense NET may be challenging with CT and MRI. These tumors are best localized using ⁶⁸Ga-Dotatate PET-CT that has a pooled sensitivity of 93% and a specificity of 95% and additionally impacted management in 69% of patients with NET detected on anatomical imaging.^{15,16} Nonfunctioning pancreatic NET present with a large mass and the presence of calcification in a preoperative CT of a well-differentiated NET were associated with intermediate grade and lymph node metastases.¹⁷ Rarely functioning NET may also attain large sizes (►Fig. 7).

Other than NET, there are many other conditions that present as enhancing masses in the pancreas. These include metastases (►Fig. 8A), acinar cell carcinoma, solid pseudopapillary tumor, solid type of serous cystadenoma, vascular malformation (►Fig. 8B), and intrapancreatic spleen to name a few.⁸ It may often be impossible to differentiate these based on imaging appearance alone and EUS-guided biopsy will be useful to confirm the diagnosis.

Metastasis to the Pancreas

These are rare with an incidence of 2 to 5% of all pancreatic masses.¹⁸ The most common primary malignancy with metastasis to the pancreas is renal cell carcinoma (70.5%).^{19,20} Pancreatic metastasis from other sources such as breast, lung, colorectal, and melanoma is also known to occur, although less common. On imaging, a solitary lesion is the commonest presentation (50–73%), but diffusely infiltrative pattern (15–44%) and multiple metastasis (5–10%) may also be seen.¹⁸ Lesions can be homogenous, with larger and more aggressive tumors showing central areas of cystic change. Metastasis are largely discrete, round, or ovoid lesions with smooth margins.²⁰ Diffuse infiltration causes enlargement of the pancreas and is seen in breast and small cell lung carcinoma.¹⁸

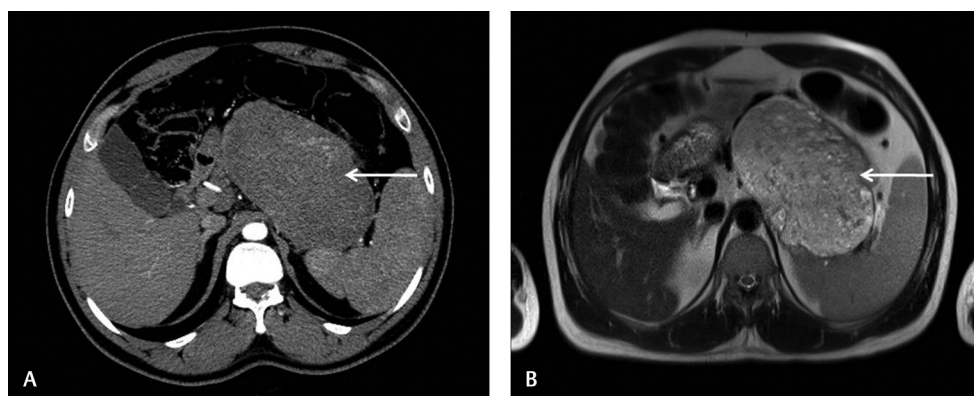


Fig. 7 Insulinoma in a 29-year-old man with recurrent episodes of hypoglycemia. (A) Postcontrast CT axial sections in the arterial phase showing a large hypoattenuating irregular mass (arrow) involving the pancreatic body and tail. (B) T2 HR axial images show a large well-defined T2 hyperintense mass (arrow) involving the body and tail of the pancreas.

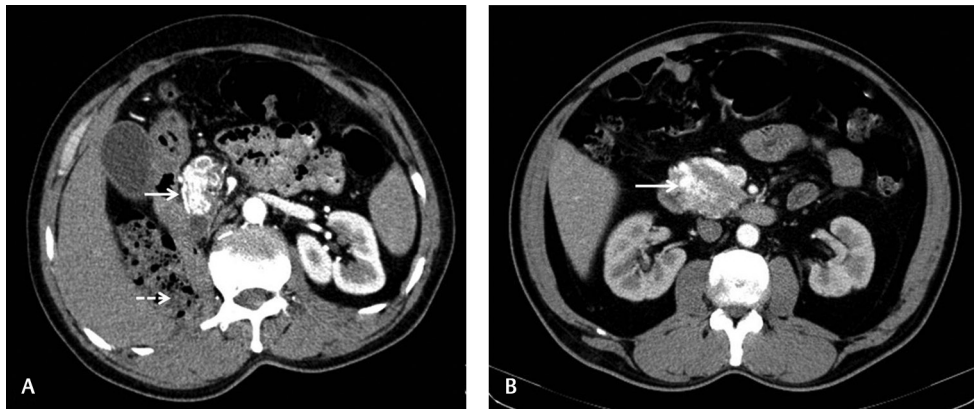


Fig. 8 (A) Metastatic renal cell carcinoma in a 59-year-old man with a history of melena, and a past history of renal cell carcinoma. Postcontrast computed tomography (CT) axial sections in the arterial phase showing an intensely enhancing well-defined mass (arrow) in the head and uncinate process of the pancreas, with associated empty right renal fossa (dashed arrow). (B) Incidental pancreatic arteriovenous malformation in a 51-year-old gentleman with gastric leiomyoma. Postcontrast CT axial sections in the arterial phase show a hyperenhancing area (arrow) in the pancreatic head with prominent vessels.

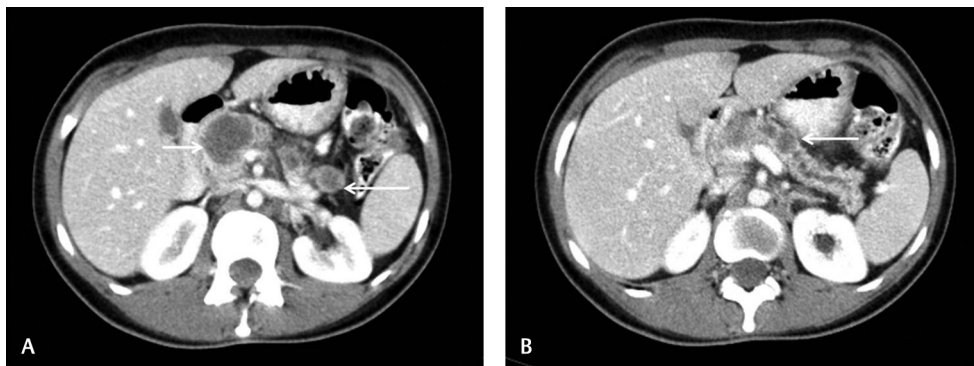


Fig. 9 Multiple pancreatic metastasis in a 29-year-old lady with abdominal pain and history of osteosarcoma. (A) Postcontrast computed tomography (CT) axial sections in the portal phase showing multiple well-defined hypovascular lesions (arrow). (B) Postcontrast CT axial sections in the portal phase showing ductal infiltration and resultant proximal dilatation of the main pancreatic duct (arrow).

Enhancement patterns on CT following contrast administration reflect the underlying primary malignancy. Metastasis from renal cell carcinoma is hypervascular with intense arterial phase enhancement (►Fig. 8A) and washout in the portal venous and delayed phases.^{20,21} Hypovascular metastases are seen with lung primary.²² Metastasis to the pancreas can cause ductal infiltration and obstruction with upstream dilatation and pancreatic atrophy.²⁰ Metastasis in the head region can cause bile duct obstruction, along with pancreatic duct obstruction. Distal atrophy of the body and tail of the pancreas may be seen.¹⁸ ►Figure 9 shows an example of multiple hypovascular metastases.

Metastasis versus PDAC

A hypovascular metastasis to the pancreas with ductal infiltration causing proximal dilatation can mimic PDAC. The clinical setting of a known primary malignancy and presence of other metastasis are useful features to differentiate the two conditions.

Pancreatic Lymphoma

Primary lymphoma of the pancreas is very rare, accounting for 0.5% of pancreatic neoplasms. Majority of them are B

cell type of non-Hodgkin's lymphoma with immunocompromised patients being more prone to this condition. On CT and MRI, pancreatic lymphoma is seen as varying sized single or multiple homogeneously hypodense lesions in the pancreas. Morphologically pancreatic lymphoma has a focal mass forming type and a diffuse type (►Fig. 10).²³

Lymphoma versus PDAC

The focal form of pancreatic lymphoma mimics pancreatic adenocarcinoma. Unlike pancreatic adenocarcinoma, lymphoma does not obstruct the pancreatic duct or biliary duct. Despite peripancreatic vessel encasement, there is no vessel attenuation or obliteration in lymphoma.²³ Moreover identification of lesions in other organs like kidneys, spleen, or liver may aid in the correct diagnosis (►Fig. 11).

Autoimmune Pancreatitis

Autoimmune pancreatitis (AIP), is a type of immune-mediated chronic pancreatitis, now recognized to have two main subtypes: type 1 AIP, also known as lymphoplasmacytic sclerosing pancreatitis and type 2 AIP or idiopathic duct-centric pancreatitis.

The more common type 1 AIP forms a part of the spectrum of IgG4-related systemic disease in which pancreatic

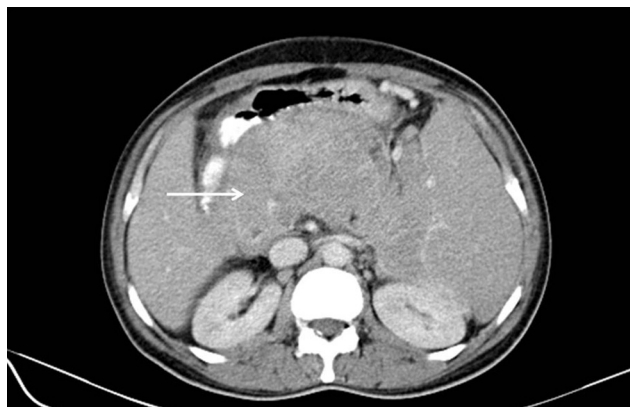


Fig. 10 Plasmablastic lymphoma in a 45-year-old man with human immunodeficiency virus and short duration of abdominal pain. Post contrast computed tomography axial sections in the portal phase show diffuse enlargement of the pancreas (arrow) that can mimic acute pancreatitis. Diffusion-weighted imaging can be used to differentiate between the two entities.

the pancreas and duct, serum levels of Ig G4, involvement of other organs, histology, and response to steroids with the first four of these features, depending on their diagnostic reliability, further grouped as level 1 or level 2 criteria.²⁵ Involvement of the pancreas in AIP can be diffuse, focal or multifocal.

Typical Features

Typical imaging features of AIP are diffuse enlargement of the pancreas with sausage shape, effacement of the normal pancreatic clefts with “featureless pancreas,” and the presence of a low attenuation soft tissue rim on CT (→**Fig. 12**), which is hypointense on T2-weighted MRI. On contrast-enhanced CT or MR, enhancement maybe variable depending on inflammation or fibrosis but typically increases on delayed phases of contrast imaging.^{24,26} These typical features categorized as level 1 criteria are diffuse enlargement of the gland with delayed enhancement and typical ductal involvement, seen on imaging as long segment(>1/3 length of pancreatic duct)

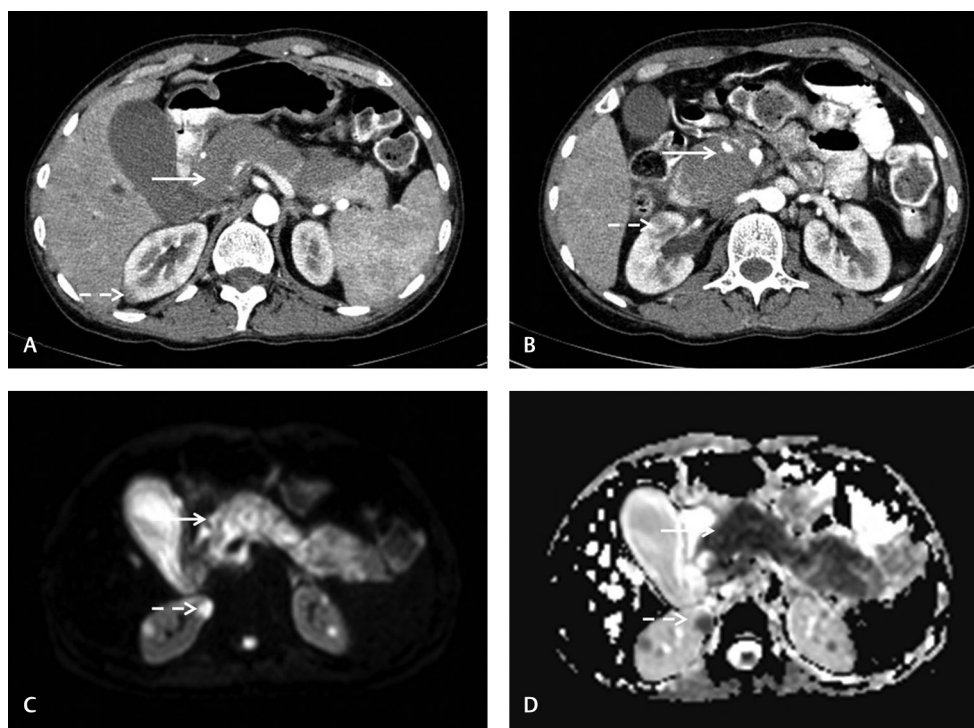


Fig. 11 A 28-year-old man with acute lymphatic leukemia with clinically suspected graft-versus-host disease and pancreatitis. (A) Postcontrast computed tomography axial sections in the arterial phase showing a hypoenhancing mass (arrow) involving the pancreas, and also showing multiple hypodense lesions in the kidneys (dashed arrow). (B) Encasement of the pancreaticoduodenal artery (arrow). (C) Diffusion-weighted imaging shows high signal on diffusion-weighted imaging with corresponding low signal on the apparent diffusion coefficient (D) seen in the pancreatic mass (arrow) and the renal lesions (dashed arrows).

involvement is common. It presents in older men, usually with painless obstructive jaundice. Elevated levels of serum IgG4, response to steroids and a tendency to relapse, are some of the hallmarks of this type.²⁴ Type 2 AIP is uncommon, occurring in younger individuals with no sex predilection. Serum IgG4 markers are normal. An association with inflammatory bowel disease, usually ulcerative colitis, may be seen. Patients with type 2 AIP also show response to steroids with relapses being very rare.²⁴

The International Consensus Diagnostic Criteria for AIP uses five criteria for the diagnosis of AIP, namely imaging features of

or multiple strictures involving pancreatic duct without any significant upstream dilatation.²⁵ Type 1 AIP is often associated with strictures of the bile duct, most commonly involving distal common bile duct (CBD) in the pancreatic head with proximal CBD dilatation.

Atypical Features

Focal involvement of the pancreas in AIP is a less common presentation that can mimic a mass, the so-called mass forming AIP. This usually involves the head of the pancreas (→**Fig. 13**). Focal AIP is also the common imaging feature of type 2 AIP.²⁴

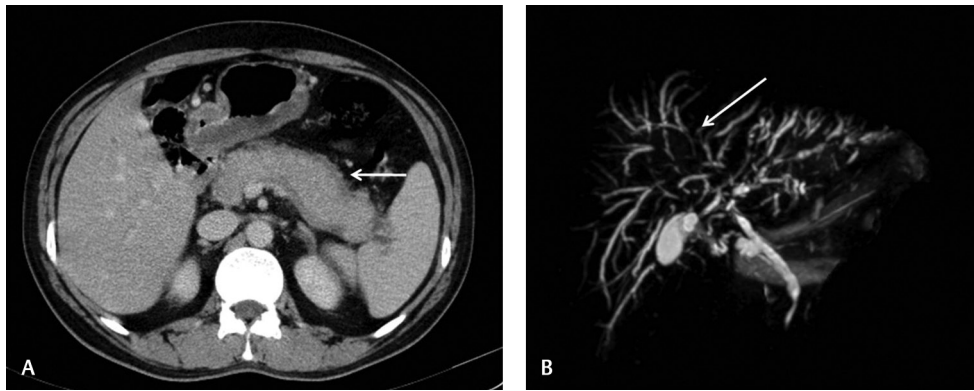


Fig. 12 Autoimmune pancreatitis in a 44-year-old man with a history of jaundice and loss of weight. **(A)** Postcontrast computed tomography axial sections in the portal phase show diffuse enlargement of the pancreas (arrow) with a rim of hypodense soft tissue. **(B)** Reconstructed magnetic resonance cholangiopancreatography showing multifocal long segment intrahepatic strictures (arrow) with narrowing of the terminal common bile duct.

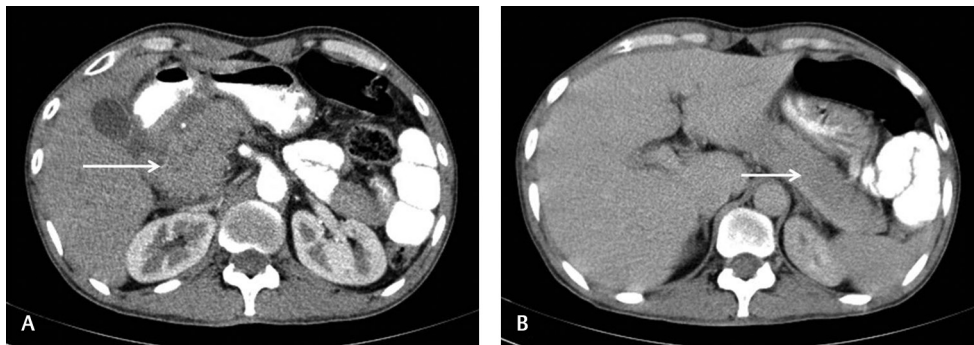


Fig. 13 Mass forming autoimmune pancreatitis in a 44-year-old man with abdominal pain for 5 years and a pancreatic head mass for 3 years. **(A)** Postcontrast computed tomography axial sections in the portal phase showing a bulky isoenhancing mass (arrow) involving the head of the pancreas. **(B)** Axial sections at a more cranial level show normal body and tail of the pancreas with no upstream main pancreatic duct dilatation (arrow).

Atypical features, which are level 2 imaging criteria for AIP, include focal enlargement with delayed enhancement and focal/segmental narrowing of the pancreatic duct without marked upstream dilatation (duct size <5 mm).²⁴

AIP versus Pancreatic Ductal Adenocarcinoma

A mass forming AIP may be difficult to differentiate from PDAC. The presence of other organ involvement and elevated serum IgG4 is useful in differentiating features but this is of limited use in type 2 AIP where extrapancreatic organ involvement does not occur, and serum IgG4 levels are normal.²⁷ Useful imaging features to differentiate between the two conditions are listed below and summarized in ►Table 2.

Enhancement Patterns on CT and MRI

Decreased pancreatic parenchymal phase enhancement with normal enhancement on the hepatic venous phase is seen in AIP. Delayed enhancement is seen in focal AIP. PDAC remains hypoattenuating on pancreatic parenchymal and delayed hepatic venous phase.²⁸ Similarly delayed enhancement pattern is seen on MRI in AIP but not in PDAC.²⁹ Diffusion-weighted MRI is useful as shown in several studies with a significantly lower ADC value in AIP than in PDAC.²⁹⁻³¹

Other findings that favor AIP over PDAC are multiplicity of masses, presence of capsule like rim, multifocal strictures

Table 2 AIP versus PDAC

	AIP	PDAC
Contrast enhancement	Delayed enhancement	Hypoenhancing on delayed phase
DWI	Lower ADC values	
Capsule like rim	Present	Absent
Distal atrophy	Absent	Present
Duct penetrating sign	Present	Absent
Significant upstream dilatation	Absent	Present

Abbreviations: ADC, apparent diffusion coefficient; AIP, autoimmune pancreatitis; PDAC, pancreatic ductal adenocarcinoma; DWI, diffusion-weighted imaging.

of the CBD and main pancreatic duct, absence of significant upstream pancreatic duct dilatation, absence of distal pancreatic atrophy, and stenosis of CBD in patients with lesions not located in head.^{29,32} The duct penetrating sign, which is an unobstructed pancreatic duct penetrating through the mass, is seen with AIP, while complete ductal obstruction is a feature of PDAC.³³

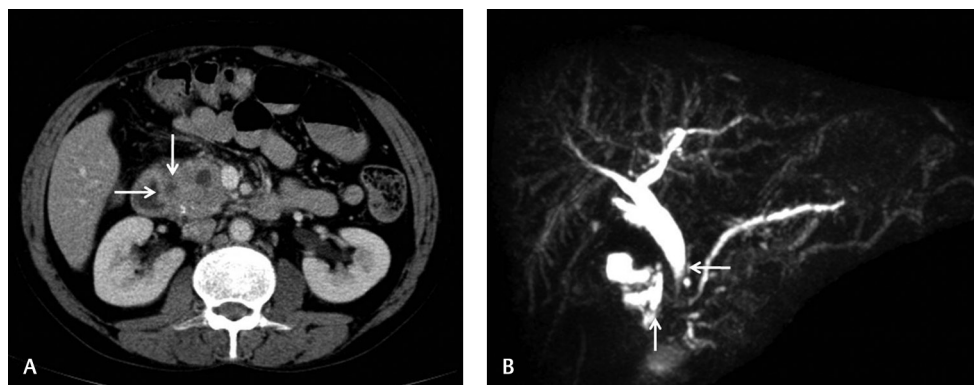


Fig. 14 Groove pancreatitis in a 40-year-old man with abdominal pain. (A) Postcontrast computed tomography axial sections in the portal phase showing thickened duodenal wall (horizontal arrow) and multiple small cystic lesions in the pancreaticoduodenal groove (vertical arrow). (B) Reconstructed magnetic resonance cholangiopancreatography showing smoothly tapered narrowing of the common bile duct (arrow) with cystic lesions in the pancreaticoduodenal groove (vertical arrow).

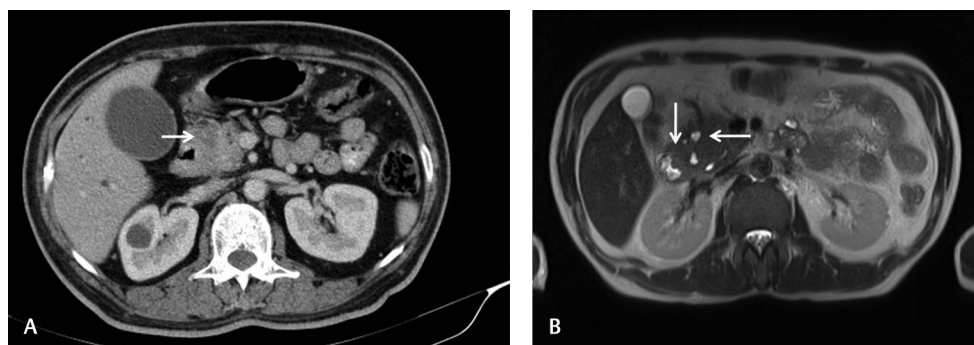


Fig. 15 (A) Pancreatic adenocarcinoma in a 47-year-old man with abdominal pain, jaundice, and fever for 3 months; postcontrast computed tomography axial sections in the portal phase showing an ill-defined hypoattenuating lesion (arrow) close to the pancreaticoduodenal groove. (B) Groove pancreatitis in a 60-year-old man with recurrent abdominal pain; T2-weighted axial magnetic resonance imaging showing hypointense soft tissue thickening with cystic changes in the pancreaticoduodenal groove (horizontal arrow) and duodenal wall thickening (vertical arrow).

Groove Pancreatitis

This is a rare form of chronic pancreatitis, involving the pancreaticoduodenal groove or the space between the head of the pancreas, duodenum, and CBD. This condition, also known as paraduodenal pancreatitis, was first described by Becker in 1973. Various causes have been attributed to this condition, such as functional obstruction of the duct of Santorini or minor papilla, and Brunner gland hyperplasia, but the most important cause is alcohol abuse. It is of two types.⁹

Pure form is confined to the pancreaticoduodenal groove with varying features such as fat stranding or sheet of soft tissue thickening in the groove. Signal intensity on T2-weighted MRI is variable, with relative T2 hyperintensity in the subacute phase due to edema and T2 hypointensity in the chronic form of disease with the onset of fibrosis. Contrast-enhanced dynamic imaging will typically show delayed enhancement due to fibrosis, in the chronic form of groove pancreatitis.³⁴ Segmental form extends from groove to involve the pancreatic head that shows a mass like enlargement and thus may mimic a pancreatic adenocarcinoma.⁹

Other imaging features of groove pancreatitis include thickened wall of duodenum with fibrosis or scar tissue,

which can result in luminal narrowing, cystic lesions in the duodenal wall or in the pancreaticoduodenal groove, smooth tapered narrowing of the CBD (►Fig. 14), and pancreatic duct involvement in the segmental form of the disease with smooth narrowing of the duct in the region of head.³⁴ This may lead to changes of chronic pancreatitis with duct dilatation, beading, and pancreatic calcifications.⁹ Banana-shaped gallbladder is described with groove pancreatitis.³⁴

Groove Pancreatitis versus Pancreatic Adenocarcinoma

PDAC located close to the pancreaticoduodenal region may show absence of pancreatic ductal obstruction with proximal dilatation and atrophy and may be difficult to differentiate from groove pancreatitis (►Fig. 15A). Some of the associated findings typically seen in groove pancreatitis, but rare in pancreatic cancer are cystic changes in pancreaticoduodenal groove or duodenal wall and marked thickening of duodenal wall (►Fig. 15B), resulting in a widened space between distal CBD and pancreatic duct and duodenal wall on MRCP.^{34,35} A smooth tapered narrowing of the distal CBD is seen in groove pancreatitis, while an abrupt narrowing favors malignancy.^{9,34} Encasement of vessels including gastroduodenal artery is a feature of pancreatic adenocarcinoma.³⁵ Similar enhancement pattern may

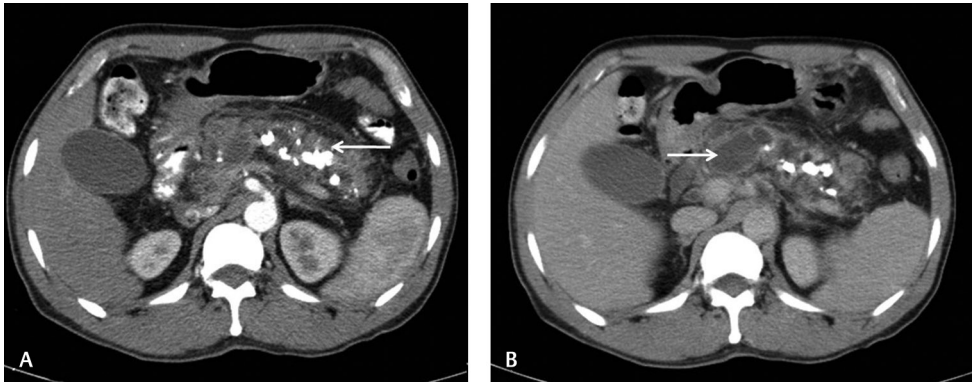


Fig. 16 Chronic pancreatitis in a 46-year-old man. (A) Postcontrast computed tomography (CT) axial sections in the arterial phase showing pancreatic parenchymal atrophy with multiple intraductal and parenchymal calcifications. (B) Postcontrast CT axial sections in the portal phase showing associated irregular dilatation of the main pancreatic duct (arrow).

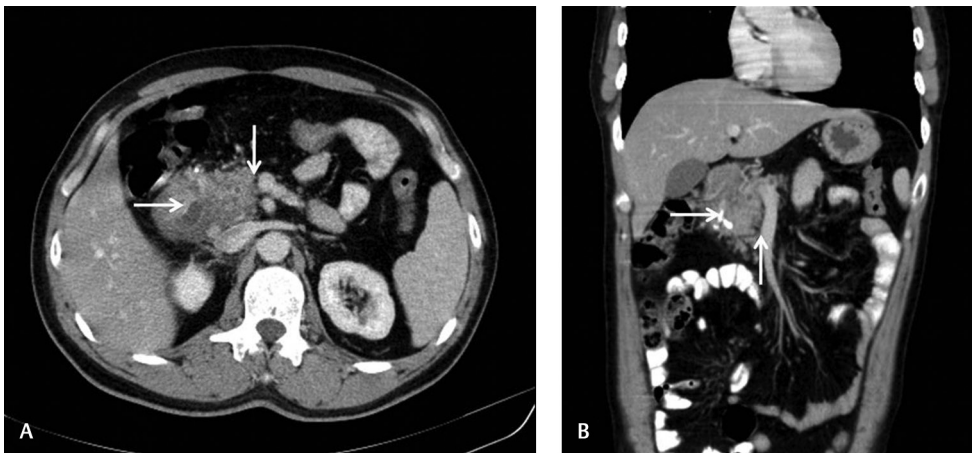


Fig. 17 Focal mass forming chronic pancreatitis in a 44-year-old man with abdominal pain. (A) Postcontrast computed tomography axial and (B) coronal sections in the portal phase showing a bulky heterogeneously hypodense enhancement of the pancreatic head (horizontal arrow) with preserved fat plane with the vessels (vertical arrow).

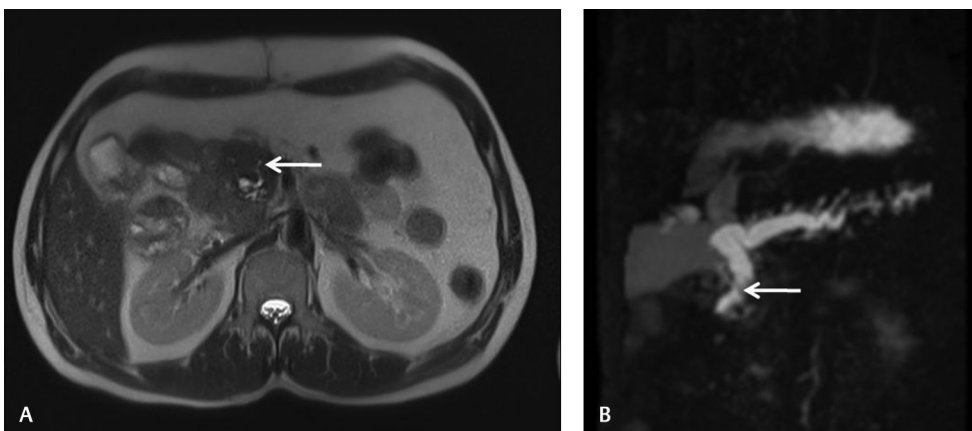


Fig. 18 (A) Chronic pancreatitis with a focal mass in a 45-year-old gentleman. (A) T2-weighted axial images showing a T2 isointense mass (arrow) in the pancreatic head. (A) Reconstructed magnetic resonance cholangiopancreatography shows duct penetrating sign with the main pancreatic duct (arrow) coursing through a pancreatic head lesion.

be seen in groove pancreatitis and the scirrhus form of PDAC with delayed enhancement in both conditions on contrast-enhanced CT or MRI.^{34,35}

Other Mimics of Groove Pancreatitis

Duodenal carcinoma, periampullary neoplasm, and acute pancreatitis involving the groove are some of the other

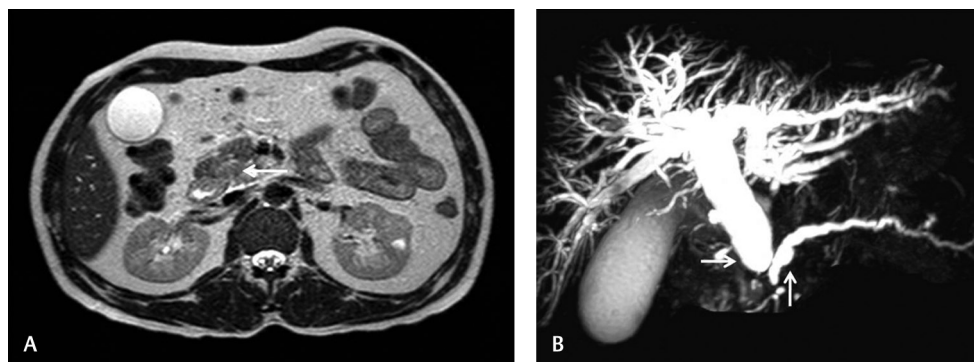


Fig. 19 Pancreatic adenocarcinoma in a 48-year-old man with obstructive jaundice. **(A)** T2-weighted axial images show an ill-defined mildly hyperintense lesion in the pancreatic head. **(B)** Reconstructed magnetic resonance cholangiopancreatography shows abrupt change in the caliber of the distal common bile duct (horizontal arrow) and the distal main pancreatic duct (vertical arrow) with upstream dilatation suggestive of double duct sign.

mimics of groove pancreatitis. The presence of cysts in the groove/duodenal wall and lesion centered in groove rather than in the duodenal wall/ampulla are some useful ways to differentiate. Retroperitoneal inflammation and fluid are seen in acute pancreatitis, but uncommon in groove pancreatitis.⁹

Chronic Pancreatitis

Chronic pancreatitis is a chronic inflammatory process of the pancreas resulting in irreversible damage to the pancreas with permanent exocrine and endocrine pancreatic dysfunction.

Typical Features

Typical findings on CT are dilatation of the main pancreatic duct that can be irregular or beaded with dilated side branches, parenchymal and intraductal calcifications, and parenchymal atrophy. The presence of obstructed main duct or side branches can result in retention cysts or pseudocysts (►Fig. 16). Thrombosis of the splenic and portal veins can occur. MRI with MRCP shows dilated main duct and side branches and calculi seen as intraductal filling defects.³⁶

Atypical Features

Focal mass forming pancreatitis (►Fig. 17) can mimic a pancreatic malignancy. Duct penetrating sign (►Fig. 18), which is seen as an unobstructed pancreatic duct penetrating through the mass, is seen in focal chronic pancreatitis.^{33,37} On the other hand, the double duct sign (►Fig. 19), which is the simultaneous dilatation of the pancreatic duct and bile duct due to obstructing mass, is seen in pancreatic head malignancy. Retroperitoneal infiltration and vessel encasement are seen in PDAC.³⁵

Pancreatic Tuberculosis

Pancreatic tuberculosis, especially isolated involvement of pancreas, is very rare. It has a higher incidence in immunodeficient individuals and those with military tuberculosis.³⁸ Spread of tuberculosis to the pancreas has been attributed to different mechanisms such as hematogenous spread, lymphatic spread, reactivation of a previously latent focus of pancreatic tuberculosis, and contiguous involvement from adjacent nodes.³⁸

Table 3 Differential diagnosis of pancreatic masses

Hypoenhancing lesions	Hyperenhancing lesions	Multiple or diffuse pancreatic masses
Pancreatic ductal adenocarcinoma	Neuroendocrine tumors	Acute pancreatitis
Chronic pancreatitis	Metastases (RCC most commonly)	Lymphoma
Focal form of autoimmune pancreatitis	Solid pseudopapillary neoplasm	Metastases
Groove pancreatitis	Solid type or microcystic serous cystadenoma of pancreas	Autoimmune pancreatitis
Lipomatous hypertrophy	Intrapancreatic spleen	IPMN
Metastases (breast, lungs, melanoma)		Multiple NET
Lymphoma	Acinar cell carcinoma	Diffuse serous cystadenoma of pancreas
Pseudolymphoma		
Sarcoidosis, tuberculosis		
Adenomatous hyperplasia of ampulla of Vater		
Annular pancreas		
Hamartoma of pancreas		
Solid fibrous tumor		

Abbreviations: IPMN, intraductal papillary mucinous neoplasm; NET, neuroendocrine tumor; RCC, renal cell carcinoma.

In the nonimmunocompromised individual, the most common presentation is a focal mass in the pancreas, usually in the head, but may also involve the body and tail. On CT, the mass is hypodense. Peripheral enhancement of the hypodense lesion or a multiloculated appearance due to foci of central enhancement may be seen. On T2-weighted MRI,

lesion is heterogenous and shows similar enhancement pattern as on CT.³⁸ It may be associated with low attenuation periportal and peripancreatic adenopathy.³⁹ Bile duct obstruction is rare, but, if present, may be due to compression by mass in the head or by lymphadenopathy.^{38,39}

Pancreatic tuberculosis in the immunocompromised individual can present as focal mass, multiple small intra-pancreatic low attenuation nodules, or diffuse enlargement of the gland.³⁹ Pancreatic tuberculosis cannot be reliably differentiated from PDAC. However, in the appropriate clinical setting, a mass in the pancreas without pancreatic and bile duct dilatation and presence of low attenuation lymphadenopathy could raise suspicion of pancreatic tuberculosis.

► **Table 3** summarizes the differential diagnosis of hypovascular, hypervascular, multiple and diffuse indeterminate pancreatic masses.

Conclusion

Knowledge of incidence of various pancreatic neoplastic and non-neoplastic conditions and their clinical presentations and typical and atypical imaging appearances will help in optimizing imaging and interpretation of imaging findings to make an accurate diagnosis, in a setting of pancreatic mass.

Conflict of Interest

None declared.

References

- de la Santa LG, Retortillo JAP, Miguel AC, Klein LM. Radiology of pancreatic neoplasms: an update. *World J Gastrointest Oncol* 2014;6(9):330–343
- Lee ES, Lee JM. Imaging diagnosis of pancreatic cancer: a state-of-the-art review. *World J Gastroenterol* 2014;20(24):7864–7877
- Gandhi NS, Feldman MK, Le O, Morris-Stiff G. Imaging mimics of pancreatic ductal adenocarcinoma. *Abdom Radiol (NY)* 2018;43(2):273–284
- Adsay NV, Basturk O, Klimstra DS, Klöppel G. Pancreatic pseudotumors: non-neoplastic solid lesions of the pancreas that clinically mimic pancreas cancer. *Semin Diagn Pathol* 2004;21(4):260–267
- Abraham SC, Wilentz RE, Yeo CJ, et al. Pancreaticoduodenectomy (Whipple resections) in patients without malignancy: are they all 'chronic pancreatitis'? *Am J Surg Pathol* 2003;27(1):110–120
- Shrikhande SV, Barreto SG, Goel M, Arya S. Multimodality imaging of pancreatic ductal adenocarcinoma: a review of the literature. *HPB (Oxford)* 2012;14(10):658–668
- Brennan DDD, Zamboni GA, Raptopoulos VD, Kruskal JB. Comprehensive preoperative assessment of pancreatic adenocarcinoma with 64-section volumetric CT. *Radiographics* 2007;27(6):1653–1666
- Zaky AM, Wolfgang CL, Weiss MJ, Javed AA, Fishman EK, Zaheer A. Tumor-vessel relationships in pancreatic ductal adenocarcinoma at multidetector CT: different classification systems and their influence on treatment planning. *Radiographics* 2017;37(1):93–112
- Raman SP, Salaria SN, Hruban RH, Fishman EK. Groove pancreatitis: spectrum of imaging findings and radiology-pathology correlation. *AJR Am J Roentgenol* 2013;201(1):W29–39
- Vortmeyer AO, Huang S, Lubensky I, Zhuang Z. Non-islet origin of pancreatic islet cell tumors. *J Clin Endocrinol Metab* 2004;89(4):1934–1938
- Humphrey PE, Alessandrino F, Bellizzi AM, Morteale KJ. Non-hyperfunctioning pancreatic endocrine tumors: multimodality imaging features with histopathological correlation. *Abdom Imaging* 2015;40(7):2398–2410
- Lewis RB, Lattin GE Jr, Paal E. Pancreatic endocrine tumors: radiologic-clinicopathologic correlation. *Radiographics* 2010;30(6):1445–1464
- Raman SP, Hruban RH, Cameron JL, Wolfgang CL, Fishman EK. Pancreatic imaging mimics: part 2, pancreatic neuroendocrine tumors and their mimics. *AJR Am J Roentgenol* 2012;199(2):309–318
- Jeon SK, Lee JM, Joo I, et al. Nonhypervascular pancreatic neuroendocrine tumors: differential diagnosis from pancreatic ductal adenocarcinomas at MR imaging-retrospective cross-sectional study. *Radiology* 2017;284(1):77–87
- Geijer H, Breimer LH. Somatostatin receptor PET/CT in neuroendocrine tumours: update on systematic review and meta-analysis. *Eur J Nucl Med Mol Imaging* 2013;40(11):1770–1780
- Hofman MS, Kong G, Neels OC, Eu P, Hong E, Hicks RJ. High management impact of Ga-68 DOTATATE (GaTate) PET/CT for imaging neuroendocrine and other somatostatin expressing tumours. *J Med Imaging Radiat Oncol* 2012;56(1):40–47
- Poultides GA, Huang LC, Chen Y, et al. Pancreatic neuroendocrine tumors: radiographic calcifications correlate with grade and metastasis. *Ann Surg Oncol* 2012;19(7):2295–2303
- Ahmed S, Johnson PT, Hruban R, Fishman EK. Metastatic disease to the pancreas: pathologic spectrum and CT patterns. *Abdom Imaging* 2013;38(1):144–153
- Sweeney AD, Fisher WE, Wu M-F, Hilsenbeck SG, Brunicki FC. Value of pancreatic resection for cancer metastatic to the pancreas. *J Surg Res* 2010;160(2):268–276
- Klein KA, Stephens DH, Welch TJ. CT characteristics of metastatic disease of the pancreas. *Radiographics* 1998;18(2):369–378
- Ascenti G, Visalli C, Genitori A, Certo A, Pitrone A, Mazziotti S. Multiple hypervascular pancreatic metastases from renal cell carcinoma: dynamic MR and spiral CT in three cases. *Clin Imaging* 2004;28(5):349–352
- Coakley FV, Hanley-Knutson K, Mongan J, Barajas R, Bucknor M, Qayyum A. Pancreatic imaging mimics: part 1, imaging mimics of pancreatic adenocarcinoma. *AJR Am J Roentgenol* 2012;199(2):301–308
- Merkle EM, Bender GN, Brambs H-J. Imaging findings in pancreatic lymphoma: differential aspects. *AJR Am J Roentgenol* 2000;174(3):671–675
- Khandelwal A, Shanbhogue AK, Takahashi N, Sandrasegaran K, Prasad SR. Recent advances in the diagnosis and management of autoimmune pancreatitis. *AJR Am J Roentgenol* 2014;202(5):1007–1021
- Shimosegawa T, Chari ST, Frulloni L, et al; International Association of Pancreatology. International consensus diagnostic criteria for autoimmune pancreatitis: guidelines of the International Association of Pancreatology. *Pancreas* 2011;40(3):352–358
- Bodily KD, Takahashi N, Fletcher JG, et al. Autoimmune pancreatitis: pancreatic and extrapancreatic imaging findings. *AJR Am J Roentgenol* 2009;192(2):431–437
- Lee-Felker SA, Felker ER, Kadell B, et al. Use of MDCT to differentiate autoimmune pancreatitis from ductal adenocarcinoma and interstitial pancreatitis. *AJR Am J Roentgenol* 2015;205(1):2–9
- Takahashi N, Fletcher JG, Hough DM, et al. Autoimmune pancreatitis: differentiation from pancreatic carcinoma and

- normal pancreas on the basis of enhancement characteristics at dual-phase CT. *AJR Am J Roentgenol* 2009;193(2):479–484
- 29 Hur BY, Lee JM, Lee JE, et al. Magnetic resonance imaging findings of the mass-forming type of autoimmune pancreatitis: comparison with pancreatic adenocarcinoma. *J Magn Reson Imaging* 2012;36(1):188–197
 - 30 Muhi A, Ichikawa T, Motosugi U, et al. Mass-forming autoimmune pancreatitis and pancreatic carcinoma: differential diagnosis on the basis of computed tomography and magnetic resonance cholangiopancreatography, and diffusion-weighted imaging findings. *J Magn Reson Imaging* 2012;35(4):827–836
 - 31 Kamisawa T, Takuma K, Anjiki H, et al. Differentiation of autoimmune pancreatitis from pancreatic cancer by diffusion-weighted MRI. *Am J Gastroenterol* 2010;105(8):1870–1875
 - 32 Sun G-F, Zuo C-J, Shao C-W, Wang J-H, Zhang J. Focal autoimmune pancreatitis: radiological characteristics help to distinguish from pancreatic cancer. *World J Gastroenterol* 2013;19(23):3634–3641
 - 33 Ichikawa T, Sou H, Araki T, et al. Duct-penetrating sign at MRCP: usefulness for differentiating inflammatory pancreatic mass from pancreatic carcinomas. *Radiology* 2001;221(1):107–116
 - 34 Blasbalg R, Baroni RH, Costa DN, Machado MCC. MRI features of groove pancreatitis. *AJR Am J Roentgenol* 2007;189(1):73–80
 - 35 Mittal PK, Harri P, Nandwana S, et al. Paraduodenal pancreatitis: benign and malignant mimics at MRI. *Abdom Radiol (NY)* 2017;42(11):2652–2674
 - 36 Ishigami K, Tajima T, Nishie A, et al. Differential diagnosis of groove pancreatic carcinomas vs. groove pancreatitis: usefulness of the portal venous phase. *Eur J Radiol* 2010;74(3):e95–e100
 - 37 Ahualli J. The double duct sign. *Radiology* 2007;244(1):314–315
 - 38 Pramesh CS, Heroor AA, Gupta SG, et al. Pancreatic tuberculosis: an elusive diagnosis. *HPB (Oxford)* 2003;5(1):43–45
 - 39 Rao RN, Pandey R, Rana MK, Rai P, Gupta A. Pancreatic and peripancreatic tuberculosis presenting as hypoechoic mass and malignancy diagnosed by ultrasound-guided fine-needle aspiration cytology. *J Cytol* 2013;30(2):130–135