Incidental Pancreatic Cysts on Cross-Sectional Imaging

Shannon M. Navarro, MD, MPH[a,*], Michael T. Corwin, MD[a], Douglas S. Katz, MD, FASER, FSAR[b], Ramit Lamba, MD, FSAR[a]

KEYWORDS

• Incidental • Pancreatic cyst • MR imaging • CT • Radiology

KEY POINTS

- Incidental pancreatic cysts are commonly encountered in a radiology practice.
- Although some of these are benign, mucinous cystic lesions have a potential to undergo malignant transformation.
- Characterization of some incidental pancreatic cysts based on imaging alone is limited, and given that some pancreatic cysts have a malignant potential, guidelines exist to help determine management and follow-up based on current evidence and consensus agreements.

INTRODUCTION

Incidental pancreatic cysts (PCs) are commonly encountered in radiology practice. The prevalence rate of PCs is estimated at 2.5%.[1] There is a 9% reported incidence on computed tomography (CT) and a 27% incidence on MR imaging.[2] PCs are a heterogeneous group, including intraductal papillary mucinous neoplasm (IPMN), serous cystic neoplasm (SCN), and mucinous cystic neoplasm (MCN). Non-neoplastic PCs are pancreatic pseudocysts (common), epithelial cysts (uncommon), and lymphoepithelial cysts (rare). The significance in categorizing PCs lies in the potential of the mucinous varieties to develop malignancy. There is substantial variability in the malignant potential of the incidentally detected PC, particularly if they are too small or otherwise cannot be fully characterized by imaging. For this reason, multiple societies have published follow-up imaging guidelines and management plans aimed at detecting early malignant transformation. The guidelines have been complicated with concerns of imaging costs and over-screening.[3] The prevalence rate of pancreatic ductal adenocarcinomas (PDACs) arising in patients with PCs is very low, at 33.2 per 100000; the rate of malignant transformation increases linearly with age.[1] This review provides a practical understanding of PCs because they are commonly encountered in radiology practice. The radiologist has an important opportunity to work with pancreatic surgeons and gastroenterologists to provide optimal multidisciplinary care to the many patients with PCs.

IMAGING TECHNIQUE AND PROTOCOL

The American College of Radiology (ACR) Appropriateness Criteria for initial evaluation of an incidental PC without high-risk stigmata list MR imaging of the abdomen without and with contrast with MR cholangiopancreatography (MRCP) as "usually appropriate" and intravenous (IV) contrast-enhanced CT (CECT) as "may be appropriate"; cyst size cutoff of greater than 2.5 cm adds a recommendation for endoscopic ultrasound (EUS). For initial evaluation of an incidental PC

The authors acknowledge and thank Dr Shiro Urayama, MD, for providing endoscopic ultrasound images.

[a] Department of Radiology, UC Davis, 4860 Y Street, Suite 3100, Sacramento, CA 95817, USA; [b] Department of Radiology, NYU Winthrop, 259 First Street, Mineola, NY 11501, USA

* Corresponding author.

E-mail address: smnavarro@ucdavis.edu

Radiol Clin N Am 59 (2021) 617–629

https://doi.org/10.1016/j.rcl.2021.03.010

greater than 2.5 cm, with worrisome features or high-risk stigmata, EUS and MR imaging of the abdomen without and with IV contrast with MRCP fall into the usually appropriate categories.[4]

For an incidentally detected main pancreatic duct (MPD) dilated beyond 7 mm and suspicion for main duct (MD)-IPMN, EUS, and MR of the abdomen without and with IV contrast with MRCP are considered usually appropriate.

Computed Tomography Protocol

Pancreatic CT is performed in the pancreatic parenchymal and portal venous phases. The pancreatic phase represents peak pancreatic parenchymal enhancement, which occurs 40 seconds to 45 seconds following the IV injection of contrast. The portal venous phase is obtained 70 seconds to 75 seconds following the IV injection of contrast. Because the time of peak parenchymal enhancement can vary based on a patient's physiology, an accurate method for achieving this phase entails triggering imaging at 16 seconds following the acquisition of a threshold of 175 Hounsfield units in the upper abdominal aorta. At UC Davis, the pancreatic CT protocol involves injecting 125 mL of iodinated contrast (iohexol, 350 mg Iodine/mL) at an injection rate of 4 mL/s. This acquisition approach also yields a late arterial phase which can be used for surgical resection planning of PDAC. One set of axial reconstructions is obtained at a slice thickness of 1.0 mm to 1.5 mm, which allows for better evaluation of small mural nodules, side duct branches, and for creating a curved-planar reformation (CPR) along the path of the main pancreatic duct. CPRs can better depict communication of a cyst with the pancreatic ductal system, changes in duct caliber, or intraductal enhancing masses.

MR Protocol

The pancreatic MR imaging protocol consists of multiplanar T2-weighted images, MRCP images, and precontrast and postcontrast 3-dimensional (3-D) –T1-weighted images. Coronal and axial single-shot fast spin-echo T2-weighted images with and/or without fat suppression are acquired because they allow for an anatomic overview and delineation of PCs. Fast spin-echo T2-weighted images are optional. Steady-state free-precession images provide contrast determined by the ratio of T2/T1. Fluid is high signal intensity and can be used as an alternative to single-shot fast spin-echo imaging. MRCP images can be acquired as 2-dimensional, thick (40 mm), heavily T2-weighted slabs and/or high-resolution (1–2 mm) 3-D volumetric acquisitions. The latter can result in superior assessment of the features of PCs and should be acquired routinely.[5] Diffusion-weighted imaging now is a routine part of abdominal MR imaging, although not specifically necessary for PC follow-up.

Nonenhanced T1-weighted MR images are acquired using 3-D fat-suppressed spoiled gradient-echo sequences. The use of IV gadolinium in the follow-up of PCs is controversial. Studies have shown the addition of contrast adds little value in the follow-up of PCs and rarely changes management.[6,7] Nonenhanced MR imaging is faster, entails review of fewer images, and avoids the risks of IV gadolinium, although rarely IV contrast may be helpful in identifying high-risk features. One approach is to selectively administer contrast for larger cysts, cysts with known high-risk features, or for the initial evaluation of cysts, leaving nonenhanced MR imaging for follow-up of cysts known to be small and without high-risk features.

ULTRASOUND

Ultrasound (US) of the pancreas has been limited by the pancreas' anatomic location as a retroperitoneal organ with overlying bowel. The utility of transabdominal US to assess PCs is inversely related to a patient's weight and abdominal diameter.[8]

Conventional US has a sensitivity of 94% for the differentiation of pseudocysts and cystic neoplasms but a relatively poor specificity, at 44%.[9] The use of IV US contrast agents can increase the specificity to 97%, by demonstrating perfusion to small nodules or septations. IV contrast significantly improves the area under the curve in receiver operating characteristic curve analysis but has not yet gained widespread use.[10]

ENDOSCOPIC ULTRASOUND

EUS utilizes endoscopy to access the upper digestive tract. The endoscope is equipped with a small US transducer with high frequency and corresponding high spatial resolution (**Fig. 1**). EUS has been shown to help differentiate pseudocysts from cystic neoplasms and guides fine-needle aspiration (FNA).[11] SCNs have a heterogeneous appearance on EUS. Mural nodules, thick cyst walls, or intracystic growth are found more commonly in mucinous neoplasms.[11,12] EUS has been shown to be very sensitive in the diagnosis of PDAC during the follow-up of IPMNs, outperforming MR imaging, CT, and US.[13] Variables most predictive of malignancy on EUS include mural nodules and MPD greater than or equal to 10 mm.[14] The interobserver agreement

Fig. 1. EUS image of the pancreas shows a complex PC (*arrow*) with a mural nodule (annotated on the image), highly compatible with malignant degeneration. (*Courtesy of* S. Urayama, M.D., Sacramento, CA.)

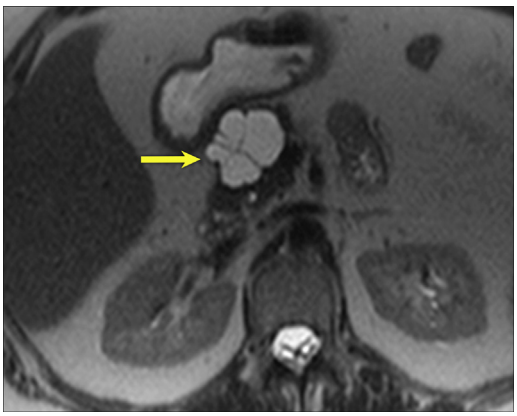

Fig. 2. SCN of the pancreas (macrocystic variant): axial T2-weighted MR image through the pancreatic head shows a multi-locular cystic mass (*arrow*) with thin septations. The largest cyst locule measures greater than 2 cm.

of EUS is low for differentiating neoplastic versus non-neoplastic, type of PC, and EUS features of a PC; EUS is limited in the differentiation of mucinous from nonmucinous cysts.[15,16] US contrast increases the accuracy of detection of mural nodules in branch duct (BD)-IPMNs from 72% to 98%.[17]

APPROACH TO THE INCIDENTAL PANCREATIC CYST

It is critical for radiologists to know if the PC actually is incidental. Any worrisome symptoms, such as pain, jaundice, or mass effect, should encourage further work-up, although PC signs and symptoms often are nonspecific. Clinical history, including patient age and gender as well as any known syndromes, may help in characterization.

DEFINITELY BENIGN

Serous Cystic Neoplasms

SCNs typically are described as having a honeycomb or multilocular appearance with or without a central scar. However, there can be variations in the morphologic appearance with polycystic, oligocystic, and solid patterns described[18,19] (**Figs. 2** and **3**). Microcystic morphology is more common in SCNs[19] (**Fig. 4**). The classic imaging features are a lobulated external contour and central scar with stellate calcification[18] (**Fig. 5**). SCNs rarely demonstrate peripheral enhancing capsule or mural nodules.[19]

The combination of morphologic features, such as location in the body or tail, size, and lobulated contour plus textural analysis, yields a high area under the receiver operator characteristic curve in differentiating SCNs from MCNs.[20] SCNs typically are isolated; however, patients with von Hippel-Lindau (VHL) disease may demonstrate multiple pancreatic masses in addition to cysts and tumors in other organ systems.[18]

Pancreatitis-Associated Fluid Collections

Pancreatitis is an inflammatory condition of the pancreas, which can result in fluid collections with a cystic appearance. The revised Atlanta classification for acute pancreatitis describes *acute peripancreatic fluid collections* (*APFCs*) as fluid collections associated with pancreatitis in the first 4 weeks of inflammation, with *acute necrotic fluid collections* (although the term is still used loosely in practice); used to denote any associated necrosis. Pseudocyst is restricted to evolving peripancreatic fluid collections, although the term is used loosely in practice. If an APFC persists beyond 4 weeks and develops an enhancing capsule the term pseudocyst is used (**Fig. 6**). If an acute necrotic collection lasts beyond 4 weeks and develops an enhancing capsule the term walled off necrosis is used.[21] It is important for the radiologist to consider pseudocyst in the differential given the high prevalence of pancreatitis.

Congenital or Syndromic Pancreatic Cysts

Congenital PCs with an epithelial lining are rare.[22] Pancreatic cystosis is a rare finding in cystic fibrosis in which the entire pancreatic parenchyma is replaced with macrocysts.[23]

Lymphoepithelial Cyst

Lymphoepithelial cysts are rare cysts lined with squamous epithelium and surrounded by

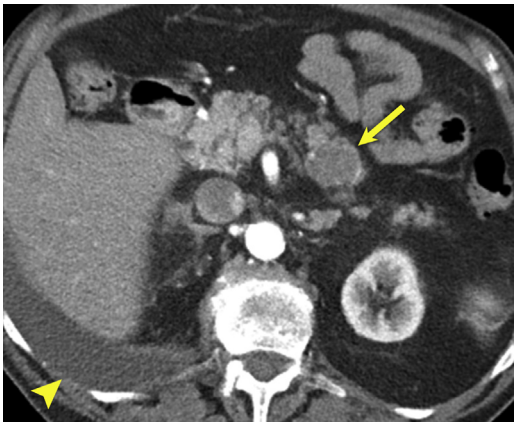

Fig. 3. SCN (solid appearing): IV CECT through the pancreatic tail shows a hypoenhancing cystic mass (*arrow*). The mass has attenuation greater than that of simple fluid compared with the simple right pleural effusion (*arrowhead*). Multiple tiny microcysts and enhancing septations can mimic a solid tumor.

lymphoid tissue. They typically affect men who are middle-aged or older and are exophytic with a higher CT attenuation compared with SCNs and MCNs.[24] The reference standard for diagnosis is excision.[25]

von Hippel-Lindau Disease

VHL disease is an autosomal dominant disorder with tumors affecting multiple organ systems. The pancreas is affected in VHL disease by PCs, endocrine tumors, and SCNs (**Fig. 7**).[26]

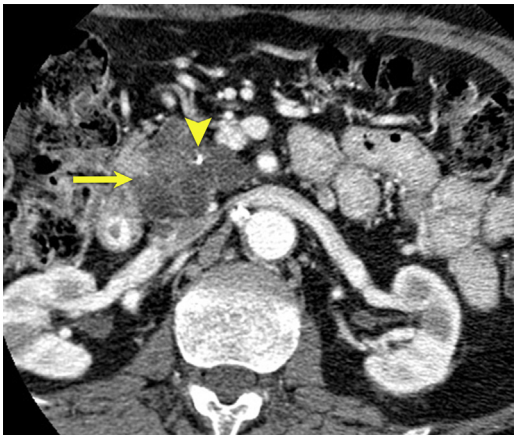

Fig. 4. SCN (microcystic variant): axial CECT image through the pancreatic head shows a cystic mass with multiple small cystic loculations (*arrow*). A focal area of calcification is seen centrally within a septation (*arrowhead*).

POTENTIALLY OR DEFINITELY MALIGNANT

Intraductal Papillary Mucinous Neoplasm

IPMNs are cystic neoplasms with variable degree of malignant potential. They may evolve into dysplasia or invasive carcinoma and are associated with a higher risk for the development of PDAC in the gland separate from the IPMN sites. The rate of progression increases with time.[27] Low-risk IPMNs have an approximately 8% chance of progression, whereas higher risk IPMNs have an approximately 25% chance of progression to PDAC in 10 years.[28] Even presumed low-risk BD-IPMNs may demonstrate growth after 5 years.[29]

IPMNs may be separated into BD-IPMNs, with a clear connection to the main duct; MD-IPMNs, in which there is either focal or diffuse ductal dilatation; or mixed types[30] (**Figs. 8–10**). Filling defects are worrisome for malignancy.[31] Variable MPD cutoff levels exist in the literature, with MPD dilatation between 5 mm and 15 mm reported as worrisome.[14,31] Other predictors of malignant IPMNs include an enhancing solid component/mural nodule(s) and thickened septae or walls.[14]

Mucinous Cystic Neoplasm

MCNs occur almost exclusively in women and more commonly are found in the pancreatic tail. MCNs are oval or round and can show septations, cyst wall calcifications, enhancing capsules, and occasionally mural nodules[19,32,33] (**Figs. 11** and **12**). MCNs typically do not cause dilatation of the biliary or pancreatic ductal system but can be associated with distal pancreatic atrophy.[34] They may be associated with lymphadenopathy but generally are not associated with peripancreatic fat infiltration or vascular involvement. Predictors

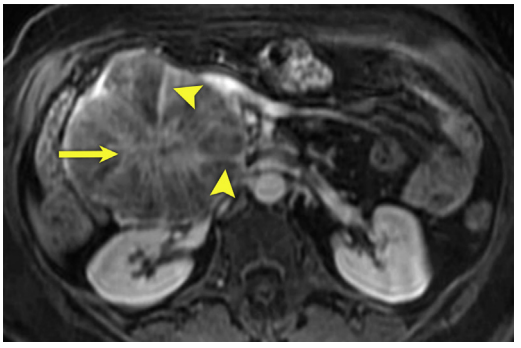

Fig. 5. SCN: axial IV contrast-enhanced MR image through the pancreatic head shows enhancement within the central scar of a SCN (*arrow*). Enhancing fibrous septations are seen radiating out from the central scar (*arrowheads*).

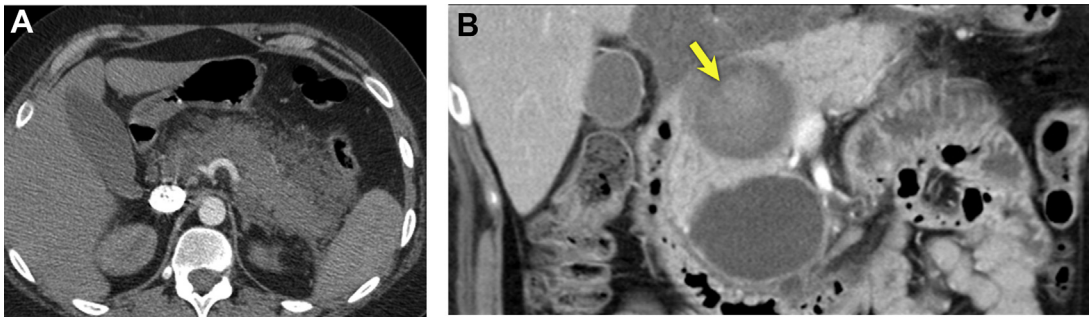

Fig. 6. Pseudocyst: (*A*) Axial CECT image in a patient with epigastric pain reveals peripancreatic fluid and pancreatic edema, compatible with acute edematous interstitial pancreatitis. (*B*) Coronal CECT image through the pancreas obtained 3 months later shows the development of 2 pseudocysts, 1 of which contains hemorrhagic material (*arrow*).

of high-grade dysplasia include size greater 8.5 cm.[32]

INDETERMINATE: REVIEW OF GUIDELINES

Many PCs are indeterminate by imaging and require imaging follow-up and/or EUS-FNA. The management of PCs is controversial and multiple societal guidelines exist (**Table 1**). It is important to work with gastroenterologists and pancreatic surgeons to ensure collaboration in regard to work-up and follow-up.

American Gastroenterological Association

The American Gastroenterological Association has published guidelines for diagnosis and management of PCs. Solid and pseudopapillary neoplasms (SPENs), cystic degeneration of adenocarcinomas, cystic neuroendocrine tumors, and MD-IPMNs were excluded. The guidelines call for involvement with the patient, and discussion of the goals and risks of PC surveillance.

Cysts with at least 2 high-risk features (size >3 cm, dilated MPD or solid component) should have EUS-FNA. After reassuring EUS-FNA, MR imaging surveillance is recommended after 1 year and then every 2 years; substantial changes in the cyst by imaging should result in repeat EUS-FNA. Cysts with solid components and a dilated MPD or concerning EUS-FNA should be offered surgery. After excision of cancer or a cystic/mucin-producing neoplasm with dysplasia, the residual pancreas should be examined using MR imaging surveillance every 2 years.[35]

Cyst surveillance may be ended after 5 years of stability or if a patient no longer is a surgical candidate. Surveillance cessation is controversial because risk of progression of PCs may increase after 5 years. This increased risk with time is reflected in the European Consensus Guidelines, which increased the follow-up time to every 6 months after 5 years.[27,36]

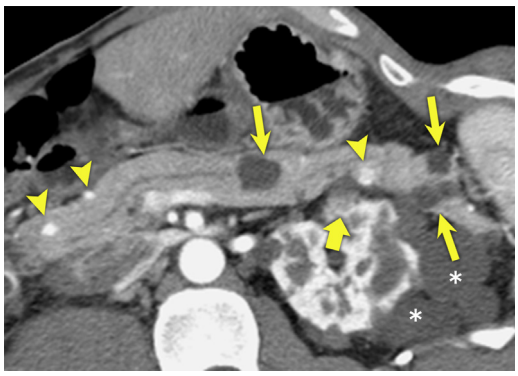

Fig. 7. VHL disease: CPR of a CECT through the pancreas shows cysts in the pancreatic body and tail (*arrows*). Hyperenhancing nodules are seen in the pancreas (*arrowheads*), compatible with neuroendocrine tumors. Cysts are seen in the left kidney (*asterisks*). An enhancing renal mass is in the anterior cortex of the left kidney (*short thick arrow*).

American College of Radiology

The initial guidelines published by the ACR incidental findings committee were released in 2010, in which they recommended PCs less than 2 cm undergo imaging at 1 year follow-up, and, if the PC is stable, to cease surveillance.[37] Subsequently, a study questioned the safety of stopping surveillance by demonstrating that 27% of PCs grow during the 1-year surveillance, and 11% grow after 1 year of stability.[38] The ACR published revised guidelines for the management of PCs in 2017, which takes a more conservative surveillance approach.[39] High-risk findings requiring EUS-FNA and surgical evaluation include mural nodularity, peripheral calcification, wall thickening, MPD greater than or equal to 7 mm, or

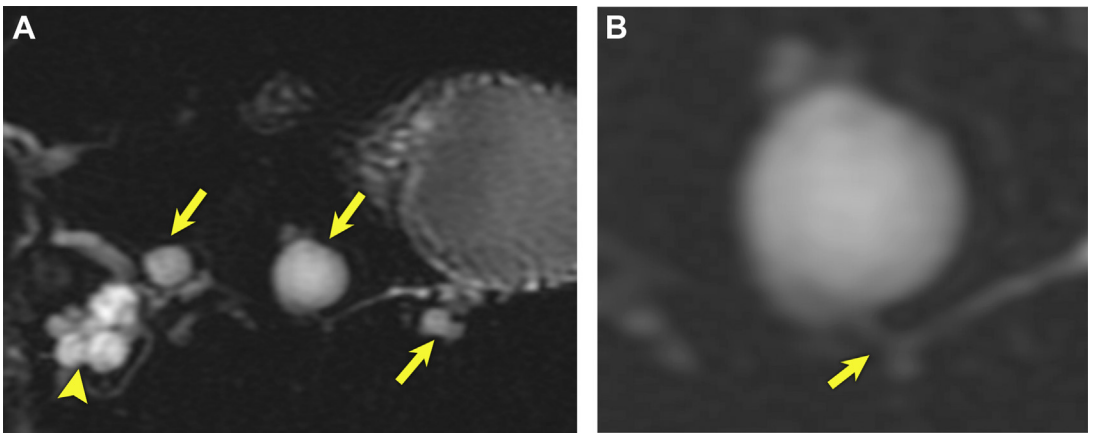

Fig. 8. BD-IPMN. (*A*) A 2-dimensional coronal MRCP image shows multiple unilocular cysts in the pancreatic head, body, and tail (*arrows*). In addition, a multilocular cyst is seen in the pancreatic head (*arrowhead*). (*B*) Magnified image of the cyst in the mid pancreas shows communication of the unilocular cyst in the pancreas body with the MPD (*arrow*).

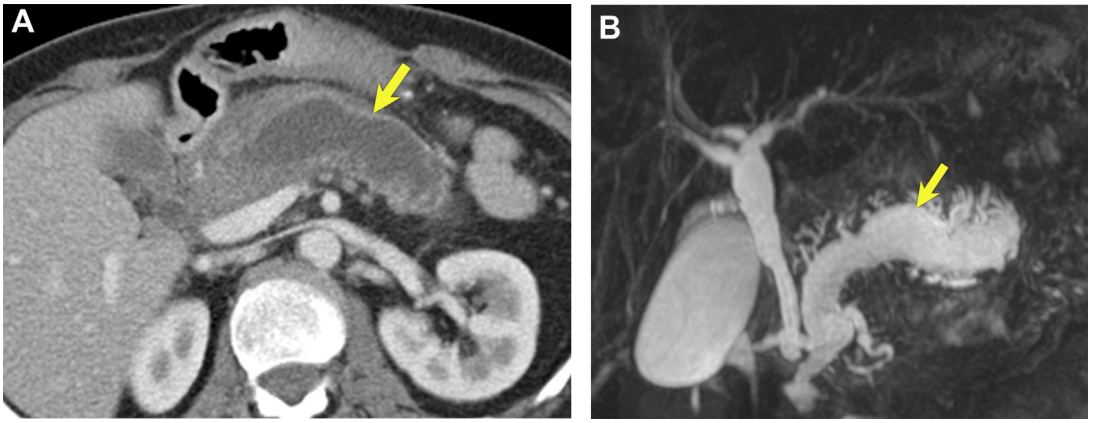

Fig. 9. MD-IPMN and BD-IPMN: (*A*) axial CECT image through the pancreas and (*B*) MRCP of the pancreas shows a markedly dilated pancreatic duct (*arrow*) with dilatation of multiple BD's.

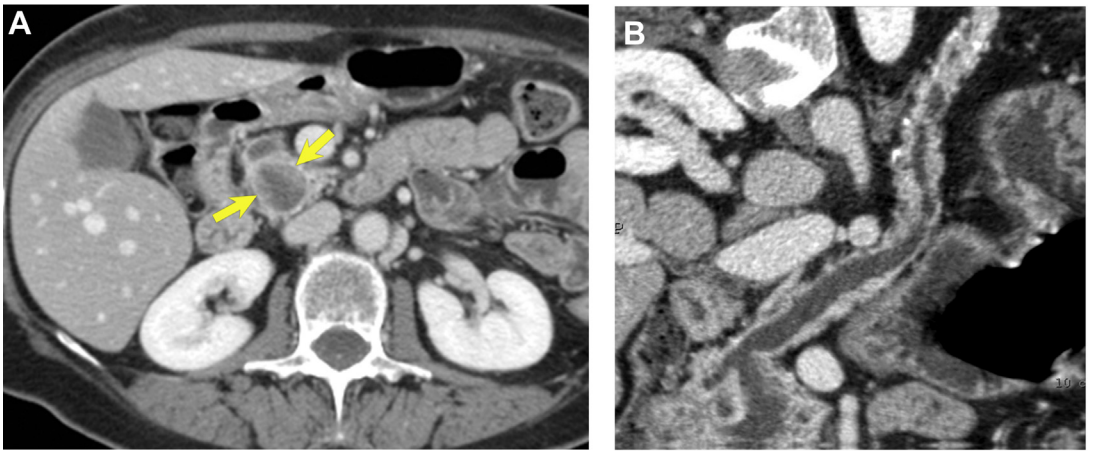

Fig. 10. MD-IPMN: (*A*) Axial CECT image through the pancreatic head shows enhancing papillary projections (*short arrows*) within the dilated pancreatic duct in the head. (*B*) CPR CECT image of the pancreatic duct shows the MPD to be dilated at 1 cm.

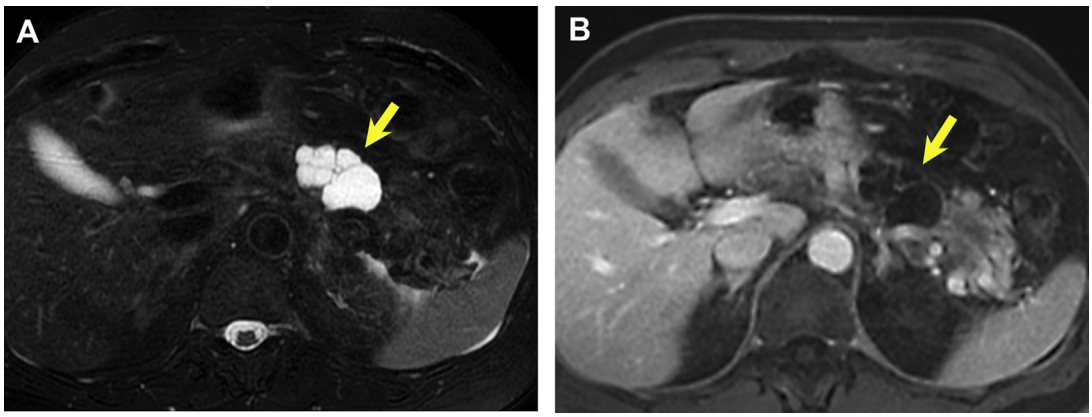

Fig. 11. MCN: (*A*) axial T2-weighted MR image through the pancreas and (*B*) axial contrast-enhanced MR image, show a multilocular cystic mass (*arrow*) in the pancreatic tail without a solid component or ductal dilatation.

extrahepatic biliary obstruction. Imaging follow-up is recommended using either a CT pancreas protocol or contrast-enhanced MR image. Growth is defined as 20% increase in the longest axis diameter on axial or coronal imaging. At the threshold of 1.5 cm to 2.5 cm, or with growth, EUS-FNA may be considered in the evaluation. Surveillance is ended at 10 years or after a patient is greater than 80 years of age, depending on the patient's health status and preferences.

Special consideration has been given to very small (<5 mm) PCs. A 2017 ACR white paper refers to these as white-dots and suggests a single follow-up MR imaging in 2 years with cessation of follow-up if stable. Pandey and colleagues[40] showed that 100% PCs with a baseline size of less than 5 mm were stable at 3 years, although 13% did demonstrate growth with a longer follow-up period. These findings support less frequent follow-up of very small PCs, although the appropriate duration remains controversial.

International Association of Pancreatology

In 2010, in Fukuoka, Japan, a consensus symposium was held in which management of IPMNs and MCNs of the pancreas was established. For PCs larger than 1 cm, a CT or MR imaging/MRCP is recommended to establish high-risk stigmata, including a solid component, enhancement, and MPD greater than or equal to 10 mm, which yield a recommendation for surgery. Worrisome features include cyst size greater than or equal to 3 cm, thickened enhancing cyst walls, mural nodules, MPD 5 mm to 9 mm, change in MPD caliber with distal atrophy, and lymphadenopathy, which yield a recommendation for EUS-FNA.

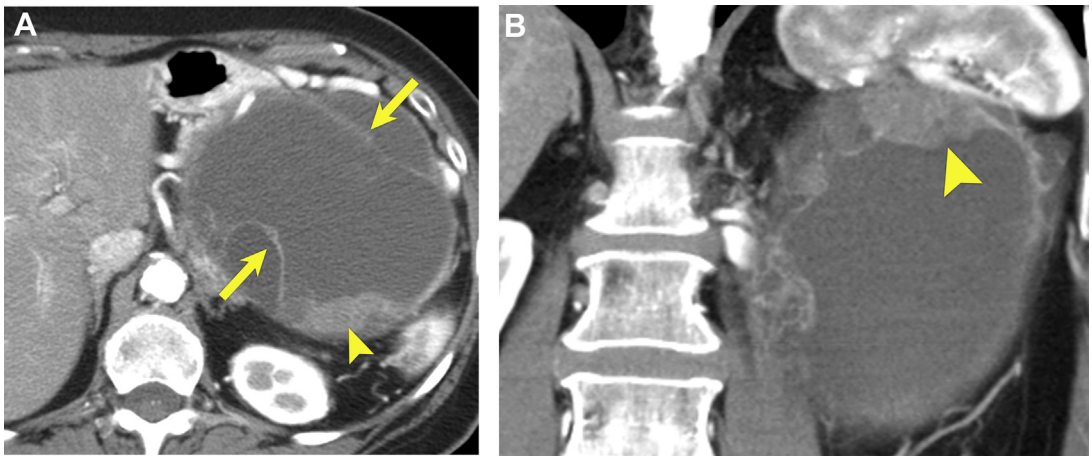

Fig. 12. MCN with malignant degeneration: (*A*) axial CECT image and (*B*) coronal reformation in a middle-aged woman show a cystic mass in the pancreatic tail. Septations (*arrows*) and enhancing solid components are seen (*arrowheads*).

Table 1
Summary of guidelines for follow-up of incidental pancreatic cysts

	High-Risk Criteria	Initial Work-up	Follow-up Modality	Follow-up Interval	Cessation
American Gastroenterological Association	At least 2 of the following: • Size >3 cm • Dilated main duct • Solid component	EUS-FNA	MR imaging	1 y, then every 2 y	After 5 y of stability or when patient is not a surgical candidate
ACR	• Mural nodularity • Peripheral calcifications • Wall thickening • Main duct >7 mm • Extrahepatic biliary obstruction	EUS-FNA and surgical consultation	CT pancreas protocol or IV contrast-enhanced MR imaging	Dependent on cyst size	After 10 y of stability or after patient is >80 y
International Association of Pancreatology (Fukuoka)	• Size >3 cm • Thick/enhancing wall • Mural nodule • Main duct 5–9 mm • Change in duct caliber with distal atrophy • Obstruction • Lymphadenopathy	EUS-FNA and surgical consultation	CT/MR imaging or EUS alternating with MR imaging	Dependent on cyst size	When patient is not a surgical candidate

If the initial imaging examination shows no high-risk stigmata or worrisome features, patients should undergo MR imaging/MRCP or CT after 3 months to 6 months, followed by annual follow-up clinically and by imaging.[41]

The Fukuoka guidelines were revised in regard to the follow-up of IPMNs.[30] Worrisome features now include rate of PC growth and imaging follow-up rate was stratified by size, with continuation until a patient no longer is a surgical candidate or elects to stop.

DIAGNOSTICS

FNA with fluid analysis of viscosity, cytology, and DNA molecular analysis can aid in the diagnostic work up of PC. Cytologic analysis of mucinous neoplasms shows clusters of columnar epithelial cells containing mucin in their cytoplasm.[16] There is low interobserver agreement in cytologic analysis of mucinous neoplasms.[42] A major problem with FNA of PCs is that many samples are limited in their cellularity; in a study of 618 samples, 53% of samples were either "less than optimal" or "unsatisfactory" for cytologic analysis. A majority (98%) of samples, however, were able to undergo molecular analysis.[43]

A combination of carcinoembryonic antigen (CEA) level greater than or equal to 192 ng/mL and molecular analysis, including DNA concentration, K-RAS mutations, and allelic imbalances, improves sensitivity in diagnosing mucinous from nonmucinous neoplasms, although CEA cutoff levels may be specific to the individual laboratory.[16,44] CA 19-9 analysis of cyst aspirate is not useful.[12] Large amounts of PC fluid DNA, high-amplitude mutations, and specific mutation acquisition sequences are predictors of malignancy.[45]

Genes associated with IPMNs include KRAS, GNAS, RNF43, TP53, PIK3CA, PTEN, CDKN2A, and SMAD4.[46] MCNs are associated with genetic alterations in KRAS, RNF43, TP53, PIK3CA, PTEN, CDKN2A, and SMAD4.[46] SCNs have a typical CEA fluid analysis of less than 5 ng/mL and low viscosity and are associated with mutations in the VHL gene.[46–48]

Fluid viscosity can be used to differentiate between mucinous and nonmucinous cysts.[49] The string sign is measured by the maximal length of mucus string between the thumb and index finger of the examiner; a positive string sign is if the mucus measures at least 3 mm.[16]

Given the limitations in adequately obtaining cells by FNA, molecular analysis likely is the direction forward in diagnostic analysis of aspirates of PCs.

POTENTIAL PITFALLS

There are several pancreatic masses in particular that may appear cystic and which are potential pitfalls to consider when assessing a PC mass.

Solid and Pseudopapillary Epithelial Neoplasms

SPENs are relatively rare low-grade malignant tumors that typically affect young women. These tumors can be solid, cystic, or mixed and frequently develop internal hemorrhage (**Fig. 13**). A well-defined thick enhancing capsule is typical. These usually are large (average 9 cm) and more often in the tail.[50]

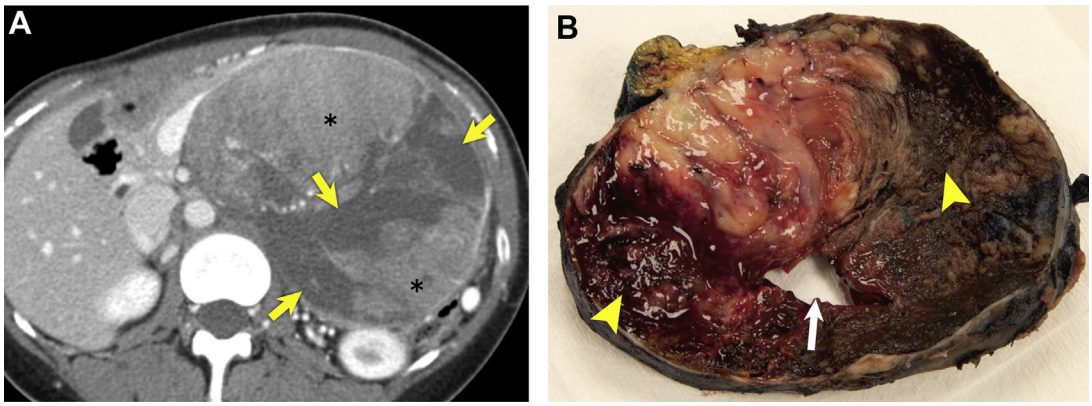

Fig. 13. SPEN. (*A*) Axial CECT image shows a large pancreatic mass with enhancing solid components (*asterisks*) and areas of cystic degeneration (*arrows*). (*B*) The gross resected specimen reveals areas of hemorrhage (*arrowheads*) and cystic degeneration (*arrow*).

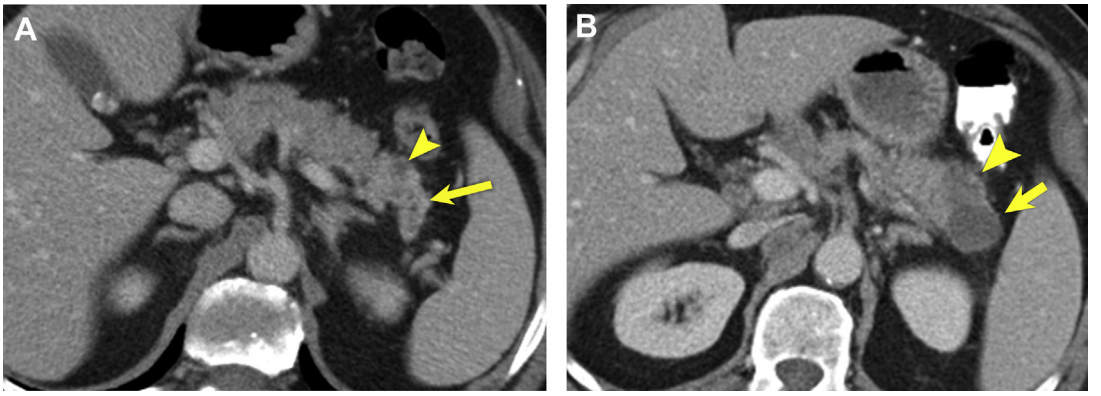

Fig. 14. PDAC with cystic component. (*A*) Axial CECT image shows focal dilatation of the MPD in the tail (*arrow*). A hypoenhancing focus (*arrowhead*) is seen proximally (*B*) Axial CECT image 3 months later shows a hypoenhancing mass in the pancreatic tail (*arrowhead*). Distal to this mass a cystic mass (*arrow*) is present that represents a cystic component of the adenocarcinoma and/or a pseudocyst after duct obstruction.

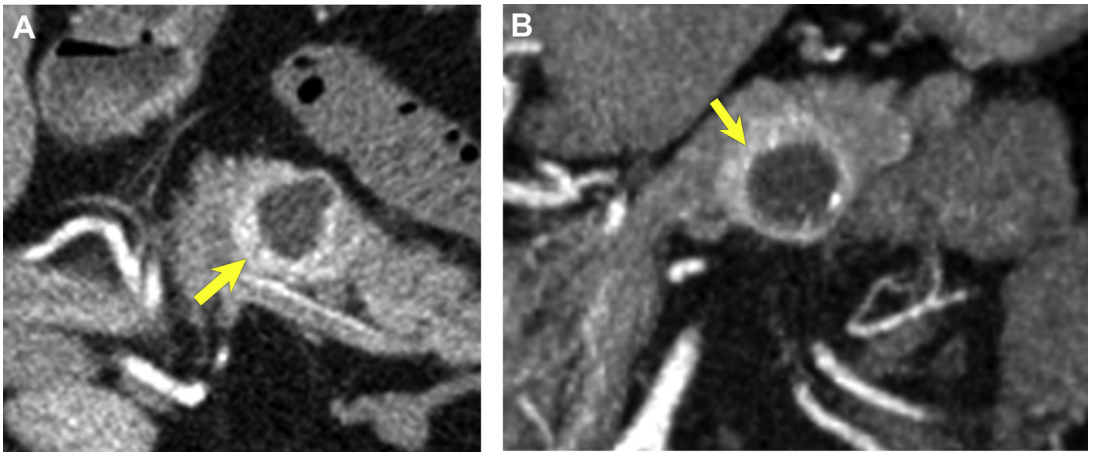

Fig. 15. (*A*) Neuroendocrine tumor with cystic degeneration: (*A*) Axial CECT image through the pancreatic neck with (*B*) coronal reformation shows a PC surrounded by peripheral enhancement (*arrows*). An arterial enhancing component almost always is present in a cystic neuroendocrine tumor.

Cystic Features of Pancreatic Ductal Adenocarcinoma

Although the classic appearance of PDAC is a solid, infiltrating mass, it may develop cystic features (**Fig. 14**), including large duct cysts, neoplastic mucinous cysts, colloid carcinomas, and degenerative cystic change. An obstructing mass can cause retention cysts or pseudocysts from pancreatitis.[51] There rarely can be a combination of these processes with the same patient, that is, areas of cystic degeneration/necrosis, as well as cystic changes related to secondary pancreatitis. Clear ductal obstruction should raise concern for PDAC. Careful assessment of the pancreas for a hypoattenuating infiltrative mass or clear ductal obstruction should raise concern for cystic degeneration of PDAC.

Cystic Neuroendocrine Tumor

Although typically solid and hyperenhancing, pancreatic neuroendocrine tumors can be mixed cystic and solid and, rarely, almost entirely cystic with a thick hyperenhancing rim or mural nodularity[52] (**Fig. 15**). These tumors can be multifocal, and, although they usually are sporadic, they can be associated with neurofibromatosis 1, multiple endocrine neoplasia type 1, or VHL disease. There is a relatively high degree of metastatic disease, either to lymph nodes or liver.[52]

SUMMARY

Incidental PCs commonly are encountered in a radiology practice. Some cystic masses of the pancreas, in particular pseudocysts, usually can be characterized accurately and adequately by a combination of imaging, history, and follow-up. Other PCs require further evaluation with EUS with FNA. Because some have malignant potential, many PCs require clinical and imaging follow-up. There are several available societal guidelines to help plan patient follow-up, with recent updates. The care of patients with PCs ideally is a multidisciplinary effort among radiologists, pathologists, surgeons, and gastroenterologists for optimal patient management.

CLINICS CARE POINTS

- Imaging alone cannot always differentiate benign pancreatic cysts from pancreatic cysts with malignant potential.
- Small indeterminate pancreatic cysts need to be followed-up, since invasive testing and resections are typically reserved for larger or growing cysts or definitively malignant cysts.

DISCLOSURE

The authors have nothing to disclose.

ACKNOWLEDGMENTS

The authors acknowledge and thank Dr Shiro Urayama, MD, for providing the endoscopic ultrasound images.

REFERENCES

1. Gardner TB, Glass LM, Smith KD, et al. Pancreatic cyst prevalence and the risk of mucin-producing adenocarcinoma in US adults. Am J Gastroenterol 2013;108(10):1546–50.
2. Mella JM, Gómez EJ, Omodeo M, et al. Prevalence of incidental clinically relevant pancreatic cysts at diagnosis based on current guidelines. Gastroenterol Hepatol 2018;41(5):293–301.
3. Rosenkrantz AB, Xue X, Gyftopoulos S, et al. Downstream costs associated with incidental pancreatic cysts detected at MRI. AJR Am J Roentgenol 2018;211(6):1278–82.
4. Fabrega-Foster K, Kamel IR, Horowitz JM, et al. ACR Appropriateness Criteria Pancreatic cyst. Available at: https://acsearch.acr.org/docs/3127236/Narrative/. American College of Radiology. Accessed August 3, 2020.
5. Liu K, Xie P, Peng W, et al. Magnetic resonance cholangiopancreatography: Comparison of two- and three-dimensional sequences for the assessment of pancreatic cystic lesions. Oncol Lett 2015;9(4):1917–21.
6. Macari M, Lee T, Kim S, et al. Is gadolinium necessary for MRI follow-up evaluation of cystic lesions in the pancreas? Preliminary results. AJR Am J Roentgenol 2009;192(1):159–64.
7. Pozzi-Mucelli RM, Rinta-Kiikka I, Wünsche K, et al. Pancreatic MRI for the surveillance of cystic neoplasms: comparison of a short with a comprehensive imaging protocol. Eur Radiol 2017;27(1):41–50.
8. Sun MRM, Strickland CD, Tamjeedi B, et al. Utility of transabdominal ultrasound for surveillance of known pancreatic cystic lesions: prospective evaluation with MRI as reference standard. Abdom Radiol (New York) 2018;43(5):1180–92.
9. Beyer-Enke SA, Hocke M, Ignee A, et al. Contrast enhanced transabdominal ultrasound in the characterisation of pancreatic lesions with cystic appearance. Jop 2010;11(5):427–33.
10. Chen F, Liang JY, Zhao QY, et al. Differentiation of branch duct intraductal papillary mucinous neoplasms from serous cystadenomas of the pancreas using contrast-enhanced sonography. J Ultrasound Med 2014;33(3):449–55.
11. Song MH, Lee SK, Kim MH, et al. EUS in the evaluation of pancreatic cystic lesions. Gastrointest Endosc 2003;57(7):891–6.
12. Leung KK, Ross WA, Evans D, et al. Pancreatic cystic neoplasm: the role of cyst morphology, cyst fluid analysis, and expectant management. Ann Surg Oncol 2009;16(10):2818–24.
13. Kamata K, Kitano M, Kudo M, et al. Value of EUS in early detection of pancreatic ductal adenocarcinomas in patients with intraductal papillary mucinous neoplasms. Endoscopy 2014;46(01):22–9.
14. Choi SY, Kim JH, Yu MH, et al. Diagnostic performance and imaging features for predicting the malignant potential of intraductal papillary mucinous neoplasm of the pancreas: a comparison of EUS, contrast-enhanced CT and MRI. Abdom Radiol (New York) 2017;42(5):1449–58.
15. Ahmad NA, Kochman ML, Brensinger C, et al. Interobserver agreement among endosonographers for the diagnosis of neoplastic versus non-neoplastic pancreatic cystic lesions. Gastrointest Endosc 2003;58(1):59–64.
16. Oh SH, Lee JK, Lee KT, et al. The Combination of Cyst Fluid Carcinoembryonic Antigen, Cytology and Viscosity Increases the Diagnostic Accuracy of Mucinous Pancreatic Cysts. Gut Liver 2017;11(2):283–9.

17. Harima H, Kaino S, Shinoda S, et al. Differential diagnosis of benign and malignant branch duct intraductal papillary mucinous neoplasm using contrast-enhanced endoscopic ultrasonography. World J Gastroenterol 2015;21(20):6252–60.
18. Kim HJ, Lee DH, Ko YT, et al. CT of serous cystadenoma of the pancreas and mimicking masses. AJR Am J Roentgenol 2008;190(2):406–12.
19. Manfredi R, Ventriglia A, Mantovani W, et al. Mucinous cystic neoplasms and serous cystadenomas arising in the body-tail of the pancreas: MR imaging characterization. Eur Radiol 2015;25(4): 940–9.
20. Yang J, Guo X, Zhang H, et al. Differential diagnosis of pancreatic serous cystadenoma and mucinous cystadenoma: utility of textural features in combination with morphological characteristics. BMC Cancer 2019;19(1):1223.
21. Thoeni RF. The revised Atlanta classification of acute pancreatitis: its importance for the radiologist and its effect on treatment. Radiology 2012;262(3):751–64.
22. Gerscovich EO, Jacoby B, Field NT, et al. Fetal true pancreatic cysts. J Ultrasound Med 2012;31(5): 811–3.
23. van Rijn RR, Schilte PP, Wiarda BM, et al. Case 113: pancreatic cystosis. Radiology 2007;243(2): 598–602.
24. Kim WH, Lee JY, Park HS, et al. Lymphoepithelial cyst of the pancreas: comparison of CT findings with other pancreatic cystic lesions. Abdom Imaging 2013;38(2):324–30.
25. Osiro S, Rodriguez JR, Tiwari KJ, et al. Is preoperative diagnosis possible? A clinical and radiological review of lymphoepithelial cysts of the pancreas. Jop 2013;14(1):15–20.
26. Mortelé KJ, Rocha TC, Streeter JL, et al. Multimodality imaging of pancreatic and biliary congenital anomalies. Radiographics 2006;26(3):715–31.
27. Del Chiaro M, Ateeb Z, Hansson MR, et al. Survival analysis and risk for progression of intraductal papillary mucinous neoplasia of the pancreas (IPMN) under surveillance: a single-institution experience. Ann Surg Oncol 2017;24(4):1120–6.
28. Choi SH, Park SH, Kim KW, et al. Progression of unresected intraductal papillary mucinous neoplasms of the pancreas to cancer: a systematic review and meta-analysis. Clin Gastroenterol Hepatol 2017;15(10):1509–20.e4.
29. Kayal M, Luk L, Hecht EM, et al. Long-term surveillance and timeline of progression of presumed low-risk intraductal papillary mucinous neoplasms. AJR Am J Roentgenol 2017;209(2):320–6.
30. Tanaka M, Fernández-Del Castillo C, Kamisawa T, et al. Revisions of international consensus Fukuoka guidelines for the management of IPMN of the pancreas. Pancreatology 2017;17(5):738–53.
31. Irie H, Honda H, Aibe H, et al. MR cholangiopancreatographic differentiation of benign and malignant intraductal mucin-producing tumors of the pancreas. AJR Am J Roentgenol 2000;174(5): 1403–8.
32. Garces-Descovich A, Beker K, Castillo-Angeles M, et al. Mucinous cystic neoplasms of the pancreas: high-resolution cross-sectional imaging features with clinico-pathologic correlation. Abdom Radiol (New York) 2018;43(6):1413–22.
33. Lee JH, Kim JK, Kim TH, et al. MRI features of serous oligocystic adenoma of the pancreas: differentiation from mucinous cystic neoplasm of the pancreas. Br J Radiol 2012;85(1013):571–6.
34. Lv P, Mahyoub R, Lin X, et al. Differentiating pancreatic ductal adenocarcinoma from pancreatic serous cystadenoma, mucinous cystadenoma, and a pseudocyst with detailed analysis of cystic features on CT scans: a preliminary study. Korean J Radiol 2011;12(2):187–95.
35. Vege SS, Ziring B, Jain R, et al. American gastroenterological association institute guideline on the diagnosis and management of asymptomatic neoplastic pancreatic cysts. Gastroenterology 2015;148(4):819–22 [quize: 12-3].
36. Del Chiaro M, Verbeke C, Salvia R, et al. European experts consensus statement on cystic tumours of the pancreas. Dig Liver Dis 2013;45(9):703–11.
37. Berland LL, Silverman SG, Gore RM, et al. Managing incidental findings on abdominal CT: white paper of the ACR incidental findings committee. J Am Coll Radiol 2010;7(10):754–73.
38. Brook OR, Beddy P, Pahade J, et al. Delayed Growth in Incidental Pancreatic Cysts: Are the Current American College of Radiology Recommendations for Follow-up Appropriate? Radiology 2016;278(3):752–61.
39. Megibow AJ, Baker ME, Morgan DE, et al. Management of Incidental Pancreatic Cysts: A White Paper of the ACR Incidental Findings Committee. J Am Coll Radiol 2017;14(7):911–23.
40. Pandey P, Pandey A, Luo Y, et al. Follow-up of incidentally detected pancreatic cystic neoplasms: do baseline MRI and CT Features Predict Cyst Growth? Radiology 2019;292(3):647–54.
41. Tanaka M, Fernández-del Castillo C, Adsay V, et al. International consensus guidelines 2012 for the management of IPMN and MCN of the pancreas. Pancreatology 2012;12(3):183–97.
42. Sigel CS, Edelweiss M, Tong LC, et al. Low interobserver agreement in cytology grading of mucinous pancreatic neoplasms. Cancer Cytopathol 2015; 123(1):40–50.
43. Nikiforova MN, Khalid A, Fasanella KE, et al. Integration of KRAS testing in the diagnosis of pancreatic cystic lesions: a clinical experience of 618 pancreatic cysts. Mod Pathol 2013;26(11):1478–87.

44. Sawhney MS, Devarajan S, O'Farrel P, et al. Comparison of carcinoembryonic antigen and molecular analysis in pancreatic cyst fluid. Gastrointest Endosc 2009;69(6):1106–10.
45. Khalid A, Zahid M, Finkelstein SD, et al. Pancreatic cyst fluid DNA analysis in evaluating pancreatic cysts: a report of the PANDA study. Gastrointest Endosc 2009;69(6):1095–102.
46. Theisen BK, Wald AI, Singhi AD. Molecular Diagnostics in the Evaluation of Pancreatic Cysts. Surg Pathol Clin 2016;9(3):441–56.
47. Elta GH, Enestvedt BK, Sauer BG, et al. ACG clinical guideline: diagnosis and management of pancreatic cysts. Am J Gastroenterol 2018;113(4):464–79.
48. Farrell JJ. Pancreatic Cysts and Guidelines. Dig Dis Sci 2017;62(7):1827–39.
49. Khamaysi I, Abu Ammar A, Vasilyev G, et al. Differentiation of pancreatic cyst types by analysis of rheological behavior of pancreatic cyst fluid. Sci Rep 2017;7:45589.
50. Buetow PC, Buck JL, Pantongrag-Brown L, et al. Solid and papillary epithelial neoplasm of the pancreas: imaging-pathologic correlation on 56 cases. Radiology 1996;199(3):707–11.
51. Youn SY, Rha SE, Jung ES, et al. Pancreas ductal adenocarcinoma with cystic features on cross-sectional imaging: radiologic-pathologic correlation. Diagn Interv Radiol 2018;24(1):5–11.
52. Kawamoto S, Johnson PT, Shi C, et al. Pancreatic neuroendocrine tumor with cystlike changes: evaluation with MDCT. AJR Am J Roentgenol 2013;200(3):W283–90.