

# Mimics, Miscalls, and Misses in Pancreatic Disease

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*The radiologist plays a pivotal role in the detection and characterization of pancreatic disorders. Unfortunately, the accuracy of rendered diagnoses is not infrequently plagued by a combination of “overcalls” of normal pancreatic anomalies and variants; “miscalls” of specific and sometimes pathognomonic pancreatic entities; and “misses” of subtle, uncommon, or inadequately imaged pancreatic abnormalities. Basic understanding of the normal and variant anatomy of the pancreas, knowledge of state-of-the-art pancreatic imaging techniques, and familiarity with the most commonly made misdiagnoses and misses in pancreatic imaging is mandatory to avoid this group of errors.*

Mimics of pancreatic disease, caused by developmental variants and anomalies, are commonly encountered on imaging studies [1–3]. To differentiate these benign “nontouch” entities from true pancreatic conditions, radiologists should be familiar with them, the imaging techniques available to study them, and their variable imaging presentations [1–3]. Classic examples include pseudomasses due to fetal lobulations, aberrant ductal configurations, pancreatic clefts, and intrapancreatic accessory spleens.

Miscalls of pancreatic abnormalities are generally multifactorial and may be induced by lack of individual or generalized knowledge, inadequate imaging technique, and inaccurate interpretation due to overlapping or confusing features of certain entities [1]. Classic examples include misdiagnosis of uneven pancreatic lipomatosis, determination of pancreatic cancer resectability, characterization and accurate measurement of cystic pancreatic lesions, and the misdiagnosis of autoimmune pancreatitis and inflammatory pseudotumors in chronic pancreatitis.

Misses of pancreatic disorders on imaging can typically be classified as perceptual, technical, and communication related [1]. Classic examples include failed detection of small (resectable) pancreatic cancers and subtle unresectable pancreatic malignancies, hypervascular pancreatic neoplasms, early chronic pancreatitis, vascular complications in acute pancreatitis, and adenocarcinomas complicating intraductal papillary mucinous tumor (IPMN).

This chapter will summarize, review, and illustrate the most common and important mimics, miscalls, and misses in pancreatic imaging and thereby improve diagnostic accuracy of diagnoses rendered when interpreting radiologic studies of the pancreas.

## Normal Pancreatic Anatomy

### The Gland

The coarsely lobulated pancreas, typically measuring approximately 15–20 cm in length, is located in the retroperitoneal anterior pararenal space and can be divided in four parts: head and uncinate process, neck, body, and tail [4]. The head, neck, and body are retroperitoneal in location whereas the tail extends into the peritoneal space. The pancreatic head is defined as being to the right of the superior mesenteric vein (SMV). The uncinate process is the prolongation of the medial and caudal parts of the head; it has a triangular shape with a straight or concave anteromedial border. The pancreatic neck is located to the left of the head and ventral to the SMV. The pancreatic body and tail are situated behind the stomach, and the distinction between them is not clearly defined but can be determined using half of the distance between the neck and the end of the pancreatic tail [4]. There is a gradual decrease of the size of the pancreas with age [5]. Moreover, the size of the pancreas is variable. Therefore, overall proportions and features of the gland (including lobular architecture, symmetry, density and signal intensity, enhancement, normal duct, and contour) are considered more important than absolute measurements in assessing the presence of a focal or diffuse pancreatic abnormality.

### The Ducts

The normal anteroposterior diameter of the main pancreatic duct measures maximally 3.5 mm in the head, 2.5 mm in the body, and 1.5 mm in the tail [2]. The main pancreatic duct (MPD) receives approximately 20–30 side branches that enter the duct at right angles [6]. There are approximately 27 different normal ductal configurations, including the common sigmoid configuration and the rare looped configuration. The downstream ductal configuration most commonly (60%) en-

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countered consists of a bifid configuration with patent ducts of Wirsung (ventral) and Santorini (dorsal) present (Fig. 1). Less common configurations include a rudimentary duct of Santorini and a dominant duct of Santorini [2].

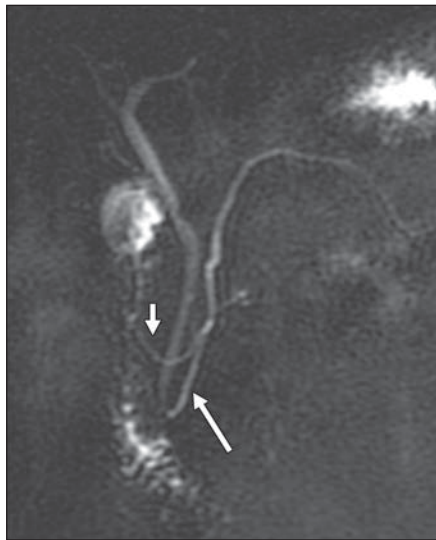
### The Pancreatobiliary Union

The duct of Wirsung joins with the common bile duct (CBD) and drains into the duodenum through the major papilla [2, 3]. Before entering the duodenum, the distal CBD and duct of Wirsung are encircled by the sphincter of Oddi, which typically measures 10–15 mm in length [2]. Sometimes, vivid contraction of the sphincter of Oddi may simulate a stone in the distal CBD on MRCP; this has been referred to as the “pseudocalculus sign.” In most cases, the distal CBD and duct of Wirsung unite within the sphincter of Oddi, forming a short (5 mm) common channel with a distal dilation called the ampulla [2]. The duct of Santorini drains the anterior and superior portions of the head via the minor papilla.

### Classic Variants (Mimics) to Know

#### Pancreatic Fetal Lobulations

Pancreatic shape alterations can simulate pancreatic neoplasms. Pancreatic head and neck lobulations are defined as



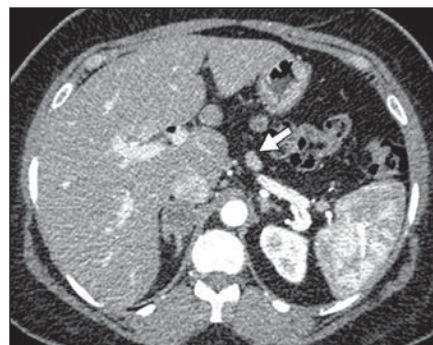
**Fig. 1**—Oblique coronal thick-slab MRCP image obtained in asymptomatic 39-year-old man shows normal bifid ductal configuration with duct of Wirsung (*long arrow*) and duct of Santorini (*short arrow*).

outpouchings of the parenchyma more than 1.0 cm beyond the anterior superior pancreaticoduodenal artery [7]. These lobulations are present in approximately 30% of people and are classified in three main types: type I (anterior), 10%; type II (posterior), 19%; and type III (lateral), 5% [2, 3]. Another well-recognized pseudomass is a prominence of the anterior and superior surface of the pancreatic body that abuts the posterior surface of the lesser omentum; this entity is known as “tuber omentale” or “omental tuberosity” and should be not misinterpreted as a pancreatic neoplasm or lymph node (Fig. 2) [2, 3].

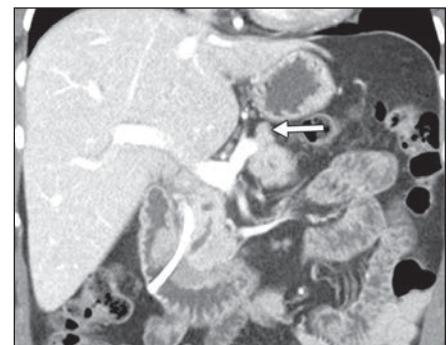
#### Ductal Fusion Anomalies

Discrepancy of the caliber of the MPD at the site of fusion of the dorsal and ventral ducts is a normal variant that may be confused for a site of stricturing [2]. The absence of dilatation of the proximal or upstream ductal system enables differentiation between those entities (Fig. 3). Duplication of the pancreatic ductal system is fairly common, especially in the tail, whereas parenchymal duplication is extremely rare.

Pancreas divisum is reported in approximately 9% of the population [7] and results from nonfusion of the ventral and dorsal pancreatic ducts during embryologic life [8]. The ventral duct (duct of Wirsung) drains only the ventral anlage, whereas most of the gland (dorsal anlage) drains via the minor papilla through the dorsal duct or duct of Santorini [8] (Fig. 4).



**A**



**B**

**Fig. 2**—43-year-old woman with ampullary adenocarcinoma.

**A and B**, Axial (**A**) and coronal (**B**) contrast-enhanced CT images show outpouching of pancreatic body in lesser sac (*arrow*). This omental tuberosity should not be confused with lymph node.

Focal dilation of the terminal portion of the dorsal duct, also called “Santorinicele,” is suggestive of relative obstruction at the minor papilla [8]. MRCP, especially when enhanced by IV secretin, has been shown to be highly accurate for depicting pancreas divisum [9].

### Annular Pancreas

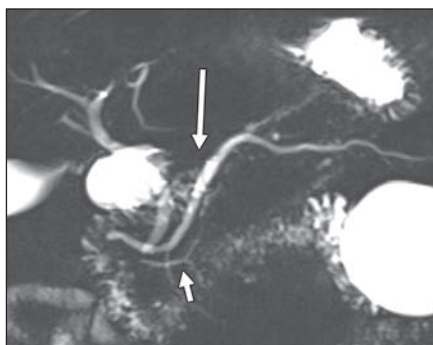
Annular pancreas is a rare (1/2000) congenital migratory anomaly due to incomplete rotation of the ventral anlage around the duodenum that leads to a segment of the pancreas encircling the second part of the duodenum [10]. There are two types of annular pancreas: extramural and intramural. In the extramural type, the ventral pancreatic duct runs around the duodenum to join the main pancreatic duct. In the intramural type, the pancreatic tissue is intermingled with muscle fibers in the duodenal wall and small ducts drain directly into the duodenum [2]. Annular pancreas can be diagnosed on the basis of CT and MRI findings that reveal pancreatic tissue and an annular duct encircling the descending duodenum [11].

### Pancreatic Hypoplasia

Hypoplasia (partial agenesis) results from the absence of the ventral or, more commonly, the dorsal anlage. Hypoplasia of the dorsal anlage, also known as “short or truncated pancreas,” can be partial or complete and may be seen as a solitary finding or in association with heterotaxia syndromes [12]. Patients with dorsal pancreatic agenesis often present with



**Fig. 3**—Oblique coronal thick-slab MRCP image obtained in asymptomatic 26-year-old woman shows discrepancy of caliber of main pancreatic duct at site of fusion of dorsal and ventral ducts (*long arrow*). This is normal variant that may be confused with site of structuring. Also note looping of pancreatic duct (*short arrow*).



**Fig. 4**—Oblique coronal thick-slab MRCP image obtained in 47-year-old woman with acute recurrent pancreatitis shows pancreas divisum characterized by lack of fusion of dorsal (*long arrow*) and ventral (*short arrow*) ducts. Dorsal duct is mildly dilated with presence of Santorinicle at minor papilla.

nonspecific abdominal pain or diabetes mellitus. When the diagnosis of pancreatic hypoplasia is suspected, it is critical to rule out a pancreatic neoplasm with upstream atrophy of the gland.

### Pancreatic Cleft

Peripancreatic fat can invaginate into the pancreas and produce an appearance that is similar to a cleft. Such a pattern on CT in a trauma patient can be confused with a pancreatic fracture. However, serum pancreatic enzyme levels are usually normal and the peripancreatic fat remains clear, which typically excludes the presence of severe pancreatic trauma.

### Intrapancreatic Accessory Spleen

Accessory spleens are common, occurring in approximately 15% of

people [13]. Their location is variable, with few (< 2%) located within the pancreatic tail [13]. Therefore, an accessory spleen may mimic a hypervascular pancreatic mass. Accessory spleens, however, typically have the same density or signal intensity characteristics as the main spleen on CT or MRI, respectively (Fig. 5). In addition, they enhance to the same degree as the spleen on both modalities. However, if CT and MRI findings are still equivocal, then nuclear scintigraphy can be performed with either  $^{99m}\text{Tc}$ -labeled sulfur colloid or  $^{99m}\text{Tc}$ -labeled heat-damaged RBCs.

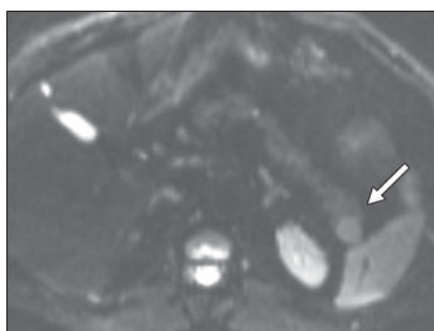
### Anomalous Pancreatobiliary Union

An anomalous pancreatobiliary junction is characterized by fusion of the pancreatic duct and CBD outside the sphinc-

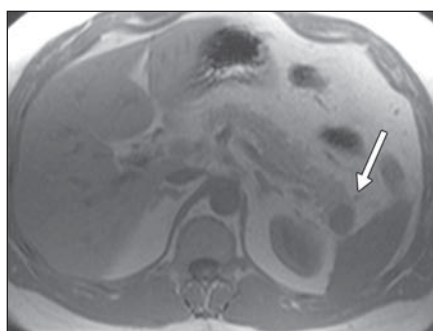
ter of Oddi with formation of a long common channel (usually > 15 mm) [14]. This long common channel allows reflux of pancreatic secretions into the biliary system, possibly resulting in choledochal web and cyst formation. Conversely, reflux of bile into the pancreatic duct can cause acute recurrent pancreatitis.

### Top Five Miscalls to Avoid Calling Uneven Pancreatic Lipomatosis Pancreatic Cancer

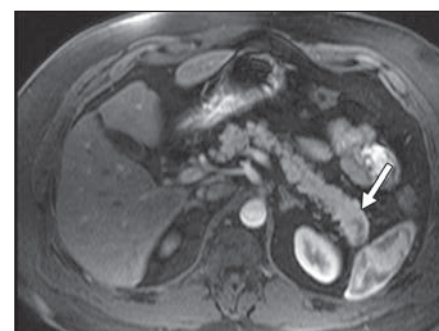
Diffuse pancreatic lipomatosis can be seen in cystic fibrosis; Shwachman-Diamond syndrome; diabetic, obese, and elderly patients; or with chronic steroid intake [15]. Sometimes, fatty replacement is heterogeneous throughout the gland. Focal fatty change of the anterior portion of the pancreatic head only is not rare on CT studies and can be confused with a hypodense mass [15]. MRI, with in- and out-of-phase imaging, can exclude a true mass by showing the presence of intracellular fat (Fig. 6). There are four different recognized types of uneven pancreatic lipomatosis: Type 1a (35%) defines fatty change of the head with sparing of the ventral pancreas and the peribiliary region; type 1b (36%) is replacement of the head, neck, and body with sparing of the ventral pancreas and the peribiliary region; type 2a (12%) is replacement of the head, including the ventral pancreas and sparing of the peribiliary region; and type 2b (18%) is the total replacement of the gland, only sparing the peribiliary region [15].



**A**



**B**

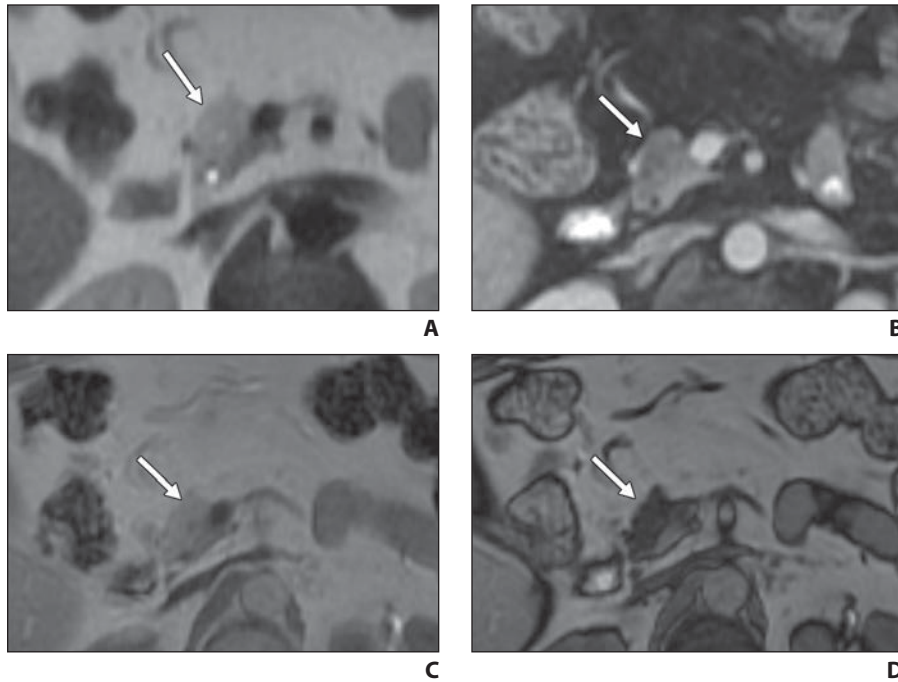


**C**

**Fig. 5**—37-year-old man evaluated for abdominal pain.

**A–C**, Diffusion-weighted (**A**), T1-weighted (**B**), and gadolinium-enhanced fat-suppressed T1-weighted (**C**) images show 1.2-cm pancreatic tail mass (*arrow*). Lesion has same signal characteristics as main spleen on all sequences. In addition, it enhances to same degree as spleen. These features are diagnostic of accessory spleen.





**Fig. 6**—65-year-old man evaluated for pancreatic lesion seen on prior CT study. **A** and **B**, T2-weighted (**A**) and gadolinium-enhanced fat-suppressed T1-weighted (**B**) images show 2.3-cm pancreatic head mass (arrow). **C** and **D**, Axial T1-weighted (**C**) and out-of-phase (**D**) MR images show signal drop (arrow) in anterior pancreatic head consistent with chemical shift effect due to presence of fat (uneven lipomatosis).

#### Measuring Cystic Pancreatic Lesions Inaccurately

Cystic pancreatic lesions are common. At my institution, Lee et al. [16] reported the prevalence of incidental pancreatic cystic lesions detected on MRI to be on the order of 13.5%. Importantly, most cystic pancreatic lesions are neoplastic epithelial lesions and only a few of the lesions are pseudocysts, related to acute or chronic pancreatitis, or nonneoplastic epithelial cysts. The size of the lesion at first diagnosis is an important factor in determining the probability of malignancy: If a lesion measures less than 3 cm, the likelihood of malignancy at that time, regardless of the underlying histologic composition, is less than 3% [17]. Therefore, most of these smaller lesions are not resected but are followed longitudinally with imaging. Several societies, including the American College of Radiology, have proposed guidelines for the follow-up and management of small incidental cystic pancreatic lesions [18] on the basis of single-length measurement, growth, imaging characteristics, and symptoms. However, these lesions

are often pleomorphic in shape and the apparent size can vary significantly depending on the imaging modality, plane of imaging, and pulse sequence used. Therefore, inaccurate measurements could lead to erroneous reporting of growth or unwarranted changes in management. Because currently none of the commonly used guidelines include standards for measurement, it is of utmost importance to measure cystic pancreatic lesions accurately and consistently in a departmentally agreed fashion and be aware of the pitfalls of intermodality and interobserver variability.

In an institutional review board–approved HIPAA-compliant study at our institution (Dunn D et al., 2013, unpublished data), 144 MRI examinations containing an even distribution of pancreatic cysts measuring between 5 and 35 mm were reviewed by four observers before and after (12 weeks) introduction of measurement standards. The interobserver agreement (kappa) increased significantly from 0.41 to 0.67 after introduction of measurement standards. In addition, the frequency

of measurement discrepancies greater than 5 mm decreased from 35.4% to 17.4%, and measurement discrepancies less than 10 mm decreased from 17.3% to 3.4%. Therefore, we believe it is fair to conclude that introduction of a few measurement standards can significantly reduce interobserver variability in the measurement of pancreatic cystic lesions and may prevent erroneous reporting of growth or unwarranted changes in management.

#### Rendering Pancreatic Cancer Resectable When It Is Not

Ductal pancreatic adenocarcinoma, the fourth to fifth leading cause of cancer death in the Western hemisphere, accounts for nearly 95% of all malignant pancreatic neoplasms. At the time of clinical presentation, 65% of patients with pancreatic cancer have locally advanced tumors, with metastatic disease present in approximately 85% of cases [19]. With this information in mind, one has to realize that focusing on unresectability rather than resectability will benefit the patient by avoiding useless and morbid surgery in unresectable cases. Too often, the evaluation of staging imaging studies of patients with pancreatic adenocarcinoma is focused on the possible resectability of pancreatic adenocarcinoma, with elaborate evaluation of the peripancreatic vascular structures and their relationship to the tumor while small hepatic, omental, and peritoneal metastases are overlooked in the process (Fig. 7).

#### Calling Autoimmune Pancreatitis Pancreatic Cancer

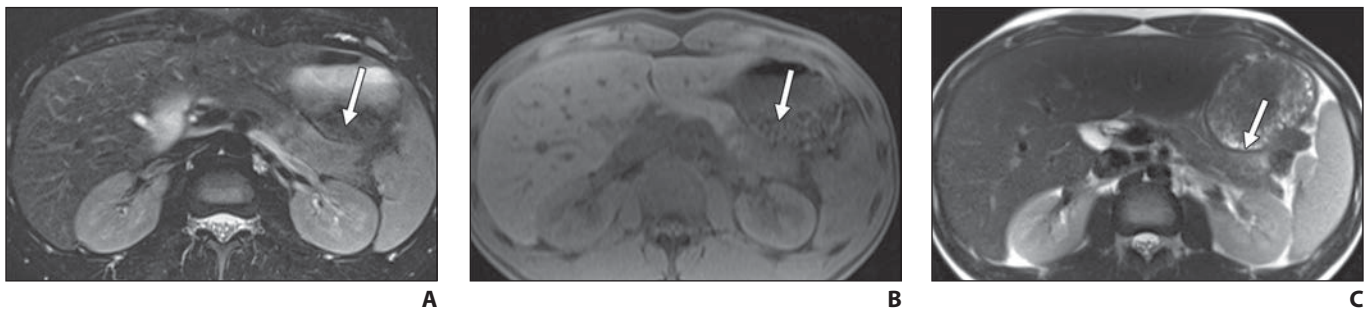
Since its first description by Yoshida et al. [20] in 1995, autoimmune pancreatitis has been increasingly recognized as a rare (5%) but global cause of chronic pancreatitis [21]. Autoimmune pancreatitis refers to a chronic inflammatory condition mediated by an autoimmune mechanism consisting of lymphocytes and plasma cells. It can involve the pancreas focally or diffusely (Fig. 8). Although autoimmune pancreatitis occurs in both sexes, it is most prevalent in men over the age of 50 years. Auto-



**Fig. 7**—67-year-old woman with back pain and weight loss.

**A**, Axial contrast-enhanced CT image shows small pancreatic head mass (arrow). Assessment of local extent failed to show vascular invasion.

**B** and **C**, Axial contrast-enhanced CT images obtained slightly higher (**B**) and lower (**C**) than **A** show subcentimeter metastases (arrow) adjacent to liver capsule and omentum, rendering this patient unresectable.



**Fig. 8**—17-year-old boy evaluated for chronic abdominal pain for over 5 years.

**A** and **B**, T2-weighted (**A**) and fat-suppressed T1-weighted (**B**) images show 5-cm pancreatic tail abnormality (arrow). IgG4 immunostaining was performed on peripanillary biopsies and IgG4-expressing plasma cells were noted, consistent with autoimmune pancreatitis.

**C**, Follow-up axial T2-weighted MR image obtained after 6-week trial of steroids shows near-complete resolution of abnormality (arrow).

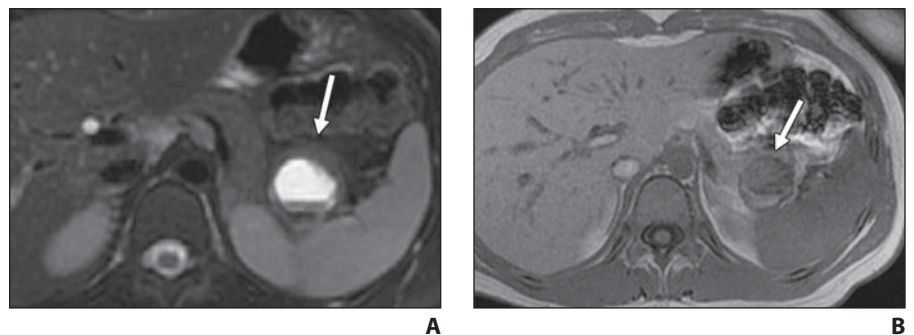
immune pancreatitis has been associated with other autoimmune diseases, including Sjögren syndrome, retroperitoneal fibrosis, primary sclerosing cholangitis, rheumatoid arthritis, and inflammatory bowel disease. A relatively new and uncommon diagnosis, autoimmune pancreatitis, or systemic IgG4-associated sclerotic disease, is not often recognized on initial presentation and is misdiagnosed as a pancreatic malignancy. Of all Whipple procedures performed in the United States, autoimmune pancreatitis is the most common benign entity unexpectedly found. Accurate diagnosis is, therefore, essential because this entity can be effectively managed noninvasively with the use of corticosteroids [21]. Surgery is not indicated in the treatment of this condition, and the prognosis for both pancreatic and extrapancreatic manifestations is generally favorable with medical management alone. The evolving definition of autoimmune pancreatitis and IgG4-associated sclerotic disease

and their increasingly frequent recognition make it essential that the practicing radiologist be aware of their existence and manifestations.

#### **Calling “Nontouch” Cystic Pancreatic Lesions “Surgical” and Vice-Versa**

Because most cystic pancreatic lesions are neoplastic, accurate diagnosis

via a combination of clinical information, imaging, and endoscopic ultrasound with cyst fluid analysis is of utmost importance. Cystic pancreatic neoplasms themselves are a diverse group of tumors that vary in aggressiveness from benign to dysplastic or premalignant to frankly invasive cancers [22]. Because treatment and man-



**Fig. 9**—17-year-old girl evaluated for abdominal pain.

**A** and **B**, T2-weighted (**A**) and T1-weighted (**B**) images show 3-cm pancreatic tail encapsulated cystic mass (arrow) with layering blood products. Combination of presence of capsule and hemorrhage in cystic mass in young girl highly suggests solid pseudopapillary tumor of pancreas, which is surgical lesion. Subsequent resection confirmed presumptive diagnosis.

agement of specific cystic pancreatic tumors are markedly different on the basis of histopathologic subtype, familiarity with the imaging appearances of these tumors is of utmost importance to help guide management and prevent unnecessary surgical interventions (Fig. 9). Fortunately, many, especially when large, have specific imaging and demographic features that enable differentiation from one another. Essential imaging features to evaluate include presence of a capsule, presence of intralesional hemorrhage, presence of communication with the main pancreatic duct, lesion contour, presence and location of calcifications, and lesion vascularity [22].

### Top Five Misses to Avoid

A recent internal review of our departmental quality assurance and peer-review databases was performed and highlighted the type of errors encountered in pancreatic imaging at our institution (Table 1). Most (approximately 50%) misses were perceptual false-negative readings (true misses) followed by misses because of technical errors (wrong protocol, technical failure). Review of the individual cases classified most errors as one of the following.

#### Detection of Small (Resectable) Pancreatic Cancers

Ductal pancreatic adenocarcinoma is the fourth to fifth leading cause of cancer death in the Western hemisphere and has a devastating prognosis unless detected when small and resectable [19]. When the correct protocol is used, the newer MDCT and MRI scanners result in accuracies of 96% for detecting pancreatic cancer. Maximum tumor conspicuity can be achieved during the late arterial or pancreatic parenchymal (40 seconds after contrast administration) phase of a dynamic contrast-enhanced CT or MRI examination [23]. Probably more important, it is essential to screen the pancreas for small lesions on all cross-sectional studies obtained for other indications. Incidental detection of a small pancreatic cancer may allow complete resection (R0) and improved survival (Fig. 10).

#### Detection of Hypervascular Pancreatic Lesions

One of the challenges pancreatic imaging still faces is to detect small hypervascular tumors, such as pancreatic endocrine tumors and hypervascular metastases from primary tumors, such as renal cell carcinoma. On multiphasic contrast-enhanced CT and MRI, hypervascular lesions are best seen, and not infrequently only seen, in the late arterial phase after contrast administration [24]. An optimized technique coupled with increased suspicion in certain clinical scenarios will undoubtedly improve detection (Fig. 11). More recently, diffusion-weighted imaging has been proposed as a beneficial method to improve pancreatic endocrine tumor detection.

#### Diagnosis of Early Chronic Pancreatitis

Chronic pancreatitis leads to irreversible parenchymal and ductal changes in the pancreas. MRI may enable an early diagnosis of chronic pancreatitis so patients can be given treatment options that may prevent progression [25]. MRI is highly sensitive and specific for the diagnosis of chronic pancreatitis in patients with advanced disease. When applying a standard MRI/MRCP pro-

tol, radiologists should look, from a ductal perspective, for changes that are induced by periductal fibrosis and the resultant duct ectasia. These changes, including side-branch abnormalities, main duct dilation and strictures, or presence of intraductal stone and intraparenchymal cyst formation can be graded using the Cambridge classification. In addition to evaluating the ductal changes, MRI is also sensitive for detecting parenchymal abnormalities [26]. What radiologists specifically should look for is subtle signal intensity decreases within the gland, especially on fat-suppressed T1-weighted images. Another important conventional MRI feature of chronic pancreatitis is delayed and diminished enhancement of the gland after gadolinium chelates administration. It is very important to realize that these parenchymal abnormalities may precede any ductal abnormalities (Fig. 12).

#### Detection of Vascular Complications in Acute Pancreatitis

Pseudoaneurysm formation occurs in approximately 10% of cases of pancreatitis [27]. The time interval is variable, ranging from days to several years after

**TABLE 1: Types of Radiologic Errors Related to Pancreas as Submitted to Quality Assurance (QA) Database and Through Peer Review at Beth Israel Deaconess Medical Center**

Radiologic Error	QA Database (n = 91)	Peer Review (n = 23)
Perceptual		
Technical	2 (2)	
False-negative (true miss)	37 (41)	13 (57)
Interpretive		
False-positive	3 (3)	
Misclassification	10 (11)	9 (39)
Communication		
Input	2 (2)	
Output (report)	2 (2)	1 (4)
Procedural		
Complications	4 (4)	
Nonrelated	1 (1)	
Technical		
Protocol (radiologist)	11 (12)	
Study (technologist)	19 (21)	

Note—Data are number with percentage in parentheses.



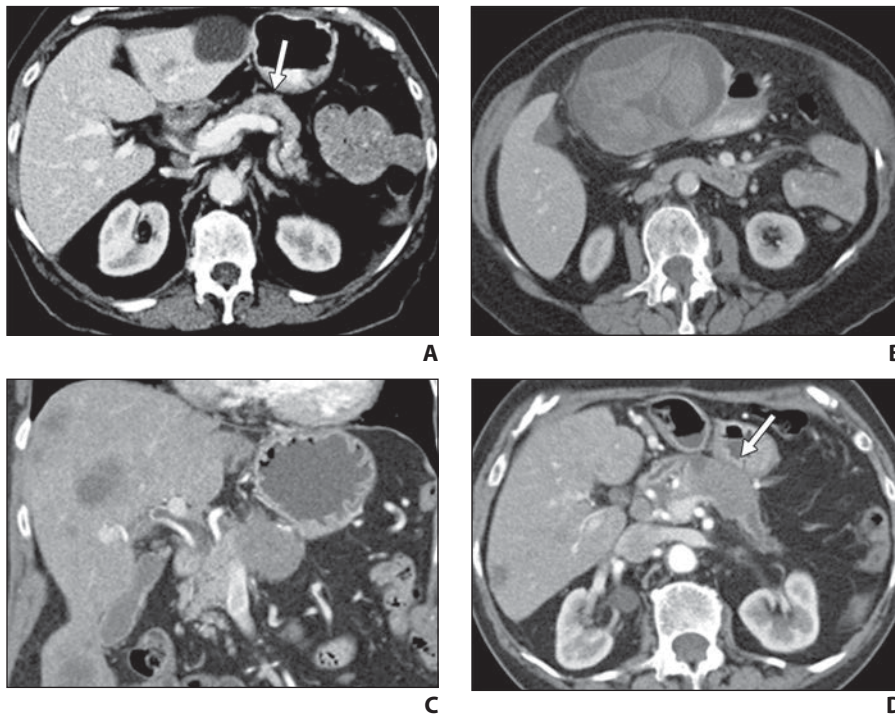
the acute inflammatory event. The vessels most commonly affected include the gastroduodenal and splenic arter-

ies. Early detection and management are paramount given the high mortality associated with rupture. The latter can

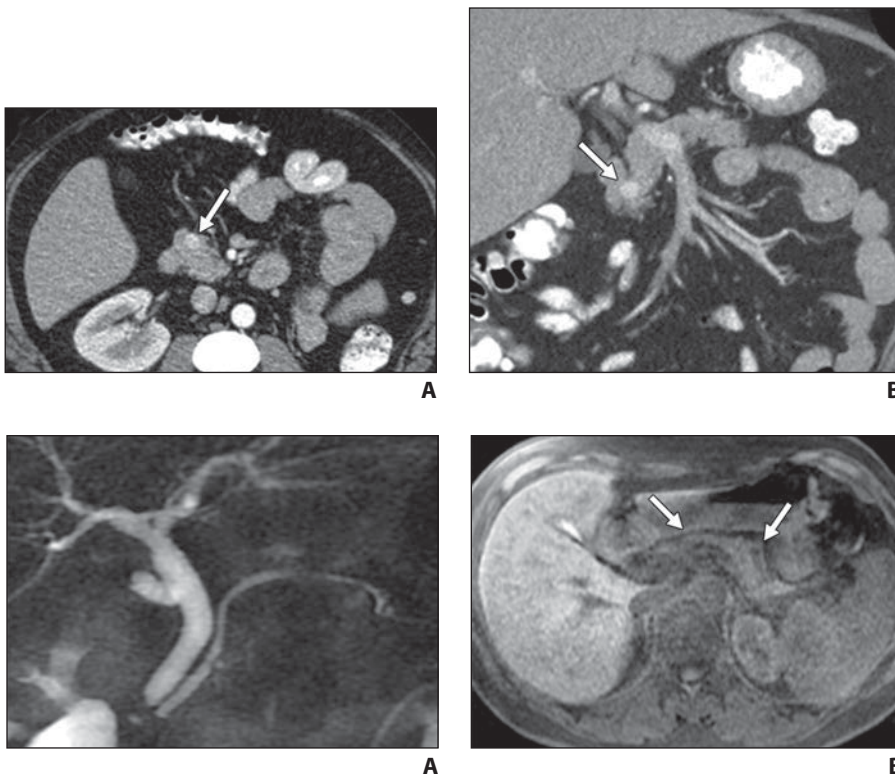
occur into the peritoneum, adjacent hollow organs, pseudocyst, or pancreatic duct (also known as hemosuccus pancreaticus) (Fig. 13). Dedicated MDCT or MR angiography can elegantly depict the pseudoaneurysm as a well-delineated rounded structure originating from the donor artery [27]. High-attenuation or high-signal-intensity thrombus may be seen within the aneurysm on unenhanced CT scans and fat-suppressed T1-weighted MR images. After contrast administration, the aneurysm may fill with contrast material if it is not completely thrombosed.

#### Diagnosing Pancreatic Cancer in Intraductal Papillary Mucinous Tumor

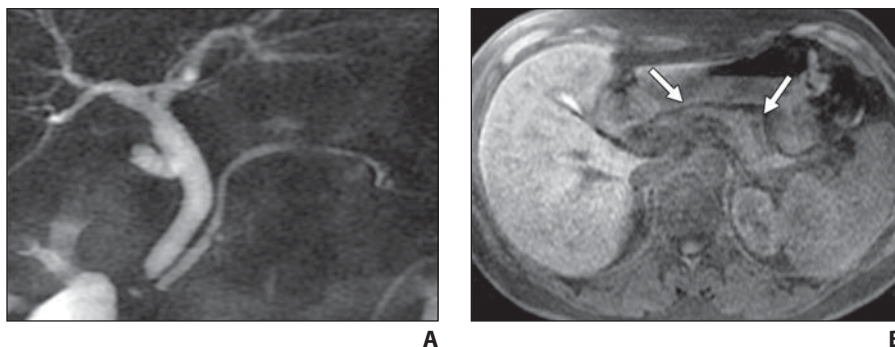
The 5-year risk of malignancy in main duct IPMN is 63% but only 15% in isolated side-branch IPMN. Suggested risk factors for malignancy on imaging include main duct dilation more than 10 mm in width, increasing size and complexity of side-branch lesions, side-branch lesions measuring more than 3 cm in size, presence of internal calcifications, CBD dilatation, thick septations, or mural nodules [22]. Familiarity with these features may enable detection of



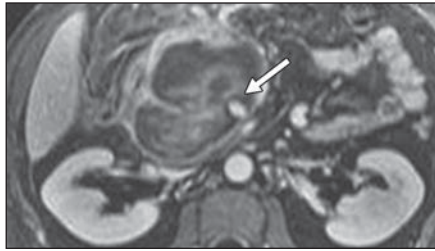
**Fig. 10**—62-year-old woman who was evaluated in emergency department after motor vehicle accident. **A and B**, Axial contrast-enhanced CT images show small pancreatic body mass (arrow, **A**) that was missed. Large left hepatic lobe hematoma was identified. **C and D**, Axial contrast-enhanced CT images obtained 1 year after **A** and **B** because of epigastric pain show unresectable pancreatic body cancer (arrow, **D**) with multiple liver metastases.



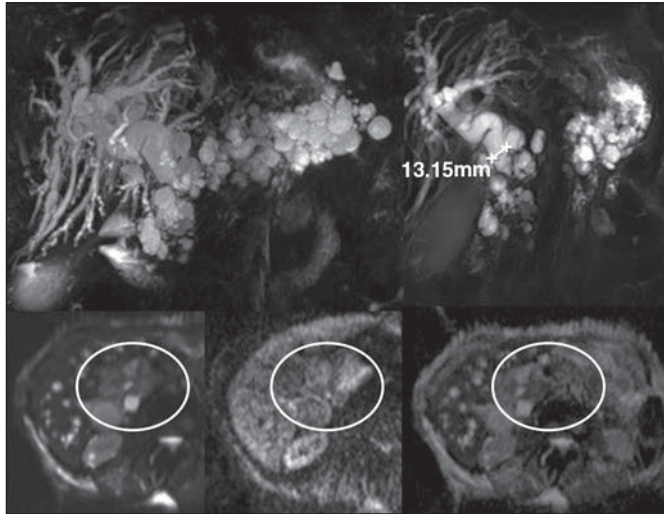
**Fig. 11**—57-year-old woman who underwent left nephrectomy 7 years ago for renal cell carcinoma (RCC). **A and B**, Axial (**A**) and coronal (**B**) contrast-enhanced CT images show small pancreatic head metastasis (arrow) that was missed, although present, on 12 consecutive torso CT scans. Awareness of possible spread of RCC to pancreas and specific screening of gland for small hypervascular lesions probably could have avoided this error.



**Fig. 12**—40-year-old woman with chronic abdominal pain. **A**, Oblique coronal MRCP image shows normal pancreatic duct. **B**, Axial fat-suppressed T1-weighted image shows atrophy of pancreas (arrows) and decreased signal intensity due to chronic pancreatitis.



**Fig. 13**—Axial fat-suppressed T1-weighted image in 34-year-old man evaluated for abdominal pain in setting of smoldering acute pancreatitis shows large hemorrhagic pseudocyst in head of pancreas (arrow) with small enhancing pseudoaneurysm.



**Fig. 14**—Coronal oblique MRCP images (top row) in 76-year-old patient with combined-duct IPMN show multiple cystic lesions scattered throughout all segments of pancreas. Note common bile duct dilation up to 13.15 mm. Diffusion-weighted images (bottom row) show increased conspicuity of small pancreatic cancer (circles) on b1000 image (middle) versus b0 image (left) with restricted diffusion on apparent diffusion coefficient map (right).

pancreatic cancer complicating IPMN with subsequent optimal patient management (Fig. 14).

## Conclusions

Mimics, miscalls, and misses in pancreatic disease are common and frequently multifactorial. Familiarity with the normal and variant anatomy of the pancreas, understanding of state-of-the-art pancreatic imaging techniques, and knowledge of the most commonly made misdiagnoses and misses in pancreatic imaging is essential to avoid this group of errors.

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