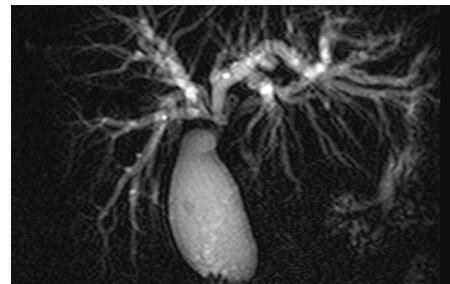


Mistakes in pancreatobiliary imaging and how to avoid them

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Multidetector computed tomography (CT) and magnetic resonance imaging (MRI) with magnetic resonance cholangiopancreatography (MRCP) are cross-sectional imaging modalities largely used for patients with pancreatobiliary diseases.^{1–3} Despite recent technological advances, correct use and interpretation of related radiological findings require good clinical judgment and collaboration between gastroenterologists and radiologists. In this article, we highlight mistakes frequently made during the radiological investigation and interpretation of findings in patients with suspected pancreatobiliary diseases, based on the available literature and on our clinical experience.



Mistake 1 Not describing or looking for anatomical variants

Laparoscopic cholecystectomy is currently the standard procedure for treatment of symptomatic gallstone disease.⁴ Bile duct injury can occur during the procedure with an incidence up to 0.7% and, albeit rare, can be associated with significant morbidity and even mortality.⁴ Biliary anatomical variations can lead to perioperative misinterpretation and are a risk factor for bile duct injury.⁵ If no preoperative MRI is performed or biliary anatomical variations are not taken into account when reporting the MRI findings, the likelihood of bile duct injury and

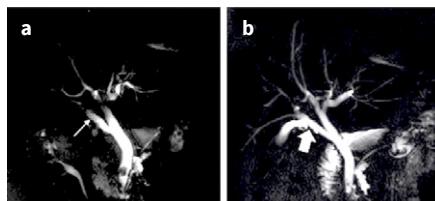


Figure 1 | Imaging the cystic duct. **a** | 2D MRCP showing that what looks like the cystic duct (arrow) is actually the right posterior duct separately originating from the common bile duct. **b** | The cystic duct (thick arrow) is originating from the right posterior duct.

postoperative complications, such as biliary leakage, is increased.

There are several biliary anatomic variations to be aware of that may lead to perioperative biliary injury: perihilar insertion of the **cystic duct** defined as a short cystic duct with an insertion <1 cm from the hilum; posterior insertion of the cystic duct into the common bile duct (CBD); direct insertion of a segmental/sectoral right hepatic duct into the gallbladder or the cystic duct, and insertion of a right sectoral/segmental hepatic duct directly into the CBD (figure 1).^{3,5}

MRCP is considered the gold standard imaging modality for preoperative evaluation of the biliary tree.³ Indeed, in a recent prospective study including 402 patients undergoing preoperative MRCP and subsequent laparoscopic cholecystectomy, **MRCP assisted in the identification of anatomical variations in 105 patients (26%).**³ Performing multiplanar acquisition helps the detection of anatomical variants on MRCP images.

Mistake 2 Misinterpreting MRCP images regarding intraductal findings

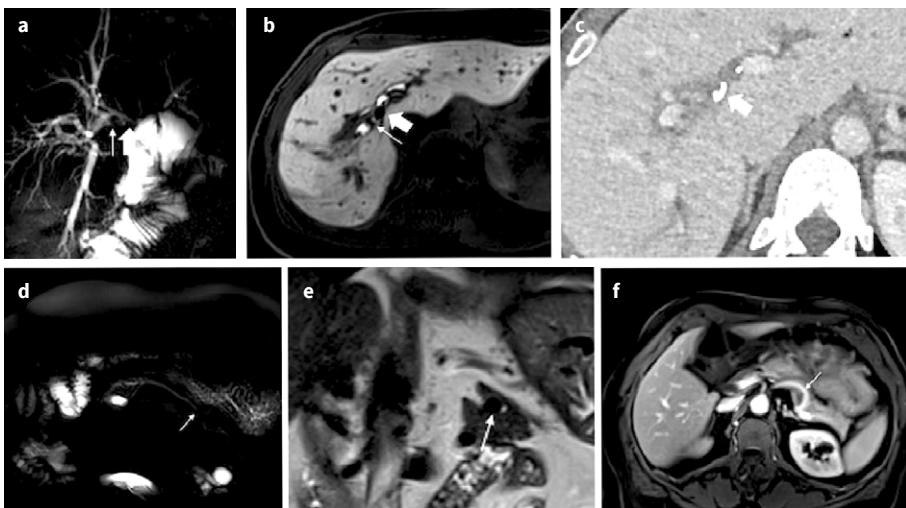


Figure 2 | Interpreting intraductal findings. **a–c** | Intraductal filling defects. **a** | MRCP shows two intraductal filling defects (arrows). **b** | T1-weighted image of the hepatobilary phase shows that the structures have different signals as one is a stone (thin arrow) and the other is a metallic clip (thick arrow) causing a blooming artefact. **c** | CT confirms the presence of a metallic clip (thick arrow). **d–f** | Pulsation artefact. MRCP shows a pancreatic tail duct stricture (arrow)(**d**) that is actually a pseudostricture caused by the adjacent splenic artery, as seen in the T2-weighted image (arrow) (**e**) and the T1-weighted post-contrast image (arrow) (**f**).

MRCP can accurately detect biliary stones and ductal strictures of the biliary and pancreatic ducts.^{1,3} However, bile is a **dynamic fluid and can produce flow voids** that mimic a stone, particularly where the cystic duct joins the CBD. Additional biliary stone mimics include **pneumobilia** (the presence of air) related to previous sphincterotomy, **debris, mucin, haemobilia** (the presence of blood clots), **clips and tumours** within the biliary tree.³ It is important to distinguish between stones and air, as the presence of air does not necessarily imply an indication to proceed to biliary drainage by endoscopic retrograde cholangiopancreatography (ERCP). In challenging cases, concomitant radiological assessment with

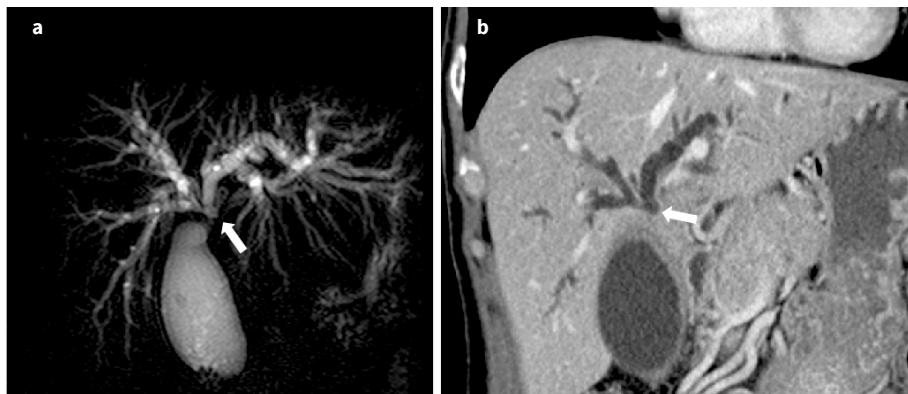


Figure 3 | IgG4 cholangiopathy. **a** | Hilar biliary stricture Bismuth type II (arrow) with upstream dilatation on MR cholangiogram. **b** | No visible mass on contrast enhanced CT (arrow). Cytology brushings were negative but surgical histology revealed IgG4 cholangiopathy.

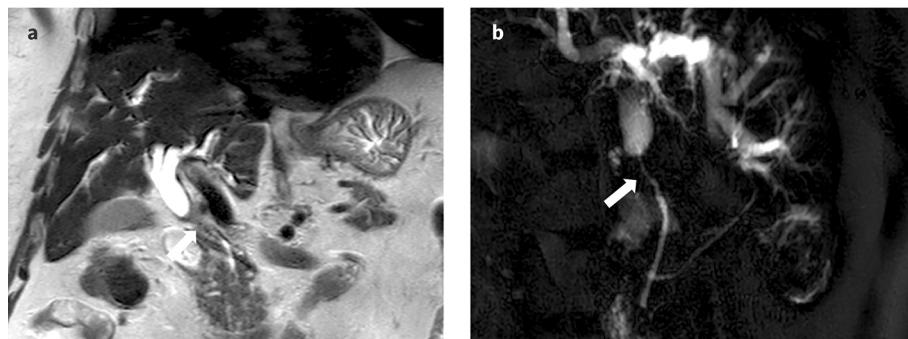


Figure 4 | Cholangiocarcinoma. Long common bile duct stricture (arrow) without mass on MRI T2 W coronal sequence (a) and MR cholangiogram (b). Cytology brushings were negative but surgical resection revealed stage T2 cholangiocarcinoma.

other modalities such as CT can help narrow the diagnosis (figure 2a–c).

Similarly, images depicting ductal strictures may be false and these findings can be attributable to patient-related factors, secondary to the MR imaging technique used or due to post-processing-related factors.⁶ Frequent causes suggestive of a false image of a biliary stricture include the ‘blooming artifact’ (a susceptibility artifact due to cholecystectomy metal clips) and the ‘pulsation artifact’ of the hepatic artery.⁶ The pulsation artifact can also give the impression that there is a stricture of the pancreatic duct, related to the splenic artery (figure 2d–f). When assessing a possible stricture, careful review of axial and coronal images obtained during MRI help to avoid misinterpretation.

Mistake 3 Assuming all biliary strictures are malignant

Despite the fact that the majority of biliary strictures are malignant (only 5–25% have a benign cause), the potential morbidity and even mortality related to unnecessary surgical resection should be taken into consideration.⁷ Causes of benign biliary strictures include iatrogenic biliary injury following hepatobiliary surgery, primary sclerosing cholangitis, IgG4-related cholangiopathy, ischaemic cholangitis, recurrent pyogenic cholangitis, AIDS-related cholangitis, and eosinophilic cholangitis.^{8,9} The clinical context (pain, weight loss, previous medical and surgical history, laboratory tests and associated diseases) is crucial to improve the diagnostic yield.⁸

The specificity of MRCP for differentiating between benign and malignant biliary strictures varies widely, from 30% to 98%.³ In general, benign strictures tend to have smooth borders with tapered margins, whereas malignant strictures are suggested by the presence of an irregular, asymmetric, longer (>12mm) stenosis with shouldered margins, increased enhancement and an indistinct outer margin.⁸ Furthermore, a diffuse or multifocal character mostly relates to autoimmune or inflammatory causes.³ Cross-sectional T1-weighted and T2-weighted MRI sequences add specificity, and the presence of an associated mass lesion is suggestive of a malignant cause.³ Conversely, mass-forming IgG4-related sclerosing cholangitis resembling hilar cholangiocarcinoma has been described, thus potentially leading to unnecessary surgery owing to suspected malignancy (figure 3).⁹

Mistake 4 Considering all biliary strictures without a mass and negative brushings as benign

As already stated, the presence of a mass adjacent to a biliary stricture is suggestive of malignancy. Nevertheless, the absence of a mass does not always mean the cause is benign. Indeed, cholangiocarcinoma can be categorized into different growth types on the basis of morphologic features and growth patterns—mass-forming, periductal infiltrating and intraductal.⁶ On MR imaging, the periductal infiltrating and intraductal growth types appear as single or multifocal biliary strictures, with focal or diffuse ductal thickening with or without contrast enhancement, and intraductal polypoid growth.⁶ These findings are nonspecific and may mimic a wide spectrum of inflammatory conditions involving the bile ducts (figure 4).

ERCP has an important role in the diagnosis of cholangiocarcinoma as intraductal brush cytology and forceps biopsies may establish the diagnosis. Nevertheless, the sensitivity of the techniques described, even combined, does not exceed 60%, leaving the possibility of false-negative results.¹⁰ Therefore, an intraductal brushing or forceps biopsy revealing no malignant cells should be repeated or complemented with additional

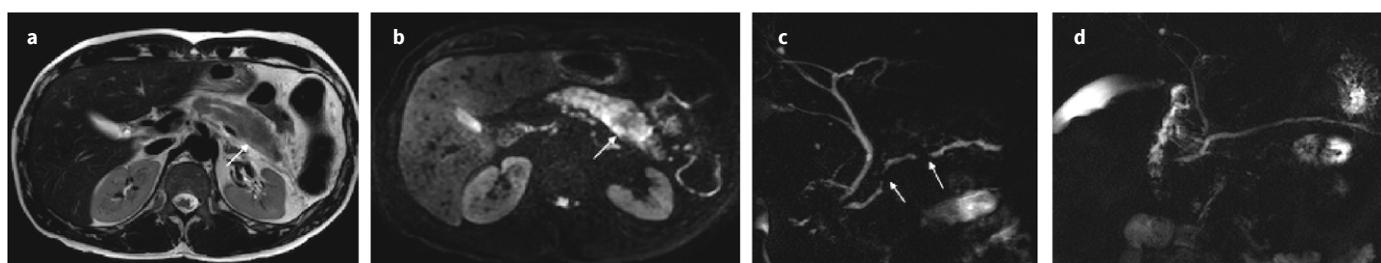


Figure 5 | Autoimmune pancreatitis. **a** | T2-weighted image shows focal pancreatic gland enlargement (arrow) with mild peripancreatic infiltration. **b** | Irregular hypersignal in DWI (arrow). **c** | MRCP shows multifocal duct narrowing (arrows) and diffuse irregularity suggesting autoimmune pancreatitis. **d** | Complete resolution of abnormalities of the duct after steroid treatment.

investigations when clinical features or outcome suggest malignancy.¹⁰

Mistake 5 Failing to identify autoimmune pancreatitis

Two subtypes of autoimmune pancreatitis (AIP) have been described: type 1 is an IgG4-related lymphoplasmacytic sclerosing pancreatitis, whereas type 2 is an idiopathic, duct-centric pancreatitis.⁹ Clinical presentation (jaundice, abdominal pain, weight loss) and imaging features (focal parenchymal enlargement) can falsely suggest the presence of a malignant lesion and erroneously lead to surgery.¹¹ Typical features of AIP include diffuse enlargement of the gland with loss of globular contours, described as a ‘sausage-like’ appearance, minimal or absent peripancreatic infiltration, a capsule-like rim enhancement surrounding the pancreas, and the absence of calcifications or vascular encasement.¹¹

Dynamic contrast-enhanced CT or MR imaging may help differentiate focal AIP from pancreatic adenocarcinoma. Both lesions appear hypovascular in the pancreatic arterial phase compared with the surrounding parenchyma, but during the portal venous phase, focal AIP may show some enhancement while pancreatic adenocarcinoma usually remains hypodense.¹¹ MR imaging can further assess pancreatic and biliary ducts, which may present with narrowing, irregularity and multifocal strictures with mild upstream dilatation in patients with AIP.¹¹

MRI diffusion-weighted imaging (DWI) is a functional technique that reflects the characteristics of tumour tissues, such as cellular density and the integrity of cell membranes, based on the erratic motion of water molecules that is quantitatively expressed as an apparent diffusion coefficient (ADC).¹ Pancreatic adenocarcinoma provokes a dense fibrotic process, which can give a lower ADC value due to the restriction diffusion often associated with fibrosis.¹ Similarly, AIP will also present with a low ADC, due to the increased density of the pancreatic parenchyma associated with periductal inflammation.¹² A distinctive feature that can differentiate the two diseases is the impressive response to steroid treatment that is

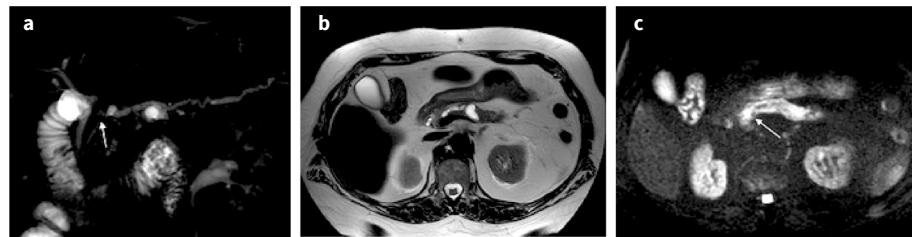


Figure 7 | Pancreatic duct stricture. An MRCP image reveals a focal pancreatic duct stricture (arrow) (a) without any measurable mass in axial T2W image (b) but with focal restriction in DWI (arrow) (c). EUS-guided fine needle aspiration confirmed a pancreatic ductal adenocarcinoma of less than 2 cm. Reproduced with permission from Zamboni GA, et al. *Abdom Radiol (NY)* 2020; 45: 1410–1419 © (2020) Springer Nature Switzerland AG.

experienced by 90% of patients with AIP, regarding both clinical and imaging features (figure 5).⁹

Mistake 6 Thinking all hypervascular pancreatic lesions are neuroendocrine neoplasms

Pancreatic neuroendocrine neoplasms (PNENs) are rare tumours that constitute approximately 1–3% of all pancreatic neoplasms.¹³ They are often well-circumscribed solid lesions that appear hyperattenuated on arterial and portal venous phase CT images because of their rich vascularity.¹³ These same features may also be encountered in cases of intrapancreatic accessory spleen (IPAS).^{11,14} Accessory spleen is a benign condition in which splenic tissue is found outside of the spleen, elsewhere in the abdomen and pelvis, and it is found in approximately 10% of adult patients in autopsy studies.¹⁴

The second most common location for IPAS is the tail of the pancreas (16.7%), following the perihilar area of the spleen.¹⁴ There are three CT and MR signs in dynamic studies that may help in differentiate IPAS from PNENs: location in the dorsal surface of the pancreas, heterogeneous enhancement in the arterial phase, and a similar enhancement pattern to the spleen in venous and late phases.¹⁵ Similarly, DWI or T2-weighted MRI can likewise suggest IPAS if the lesion is isointense with the spleen (figure 6).¹⁴

Further studies that can help refine the diagnosis and avoid unnecessary surgery are Technetium-99m sulfur colloid radiolabelled heat-damaged red blood cells and endoscopic ultrasound (EUS) guided fine needle aspiration (FNA).^{11,14} On EUS IPAS appears hypoechoic or

isoechoic, round or oval shaped, homogenous with a smooth, well-demarcated border. The echogenicity is similar to the splenic echogenicity and the size is usually <2 cm.¹⁴ FNA typically reveals a heterogeneous population of lymphocytes with traversing small vessels over a background of blood and mixed inflammatory cells with CD8 positive immunostaining.¹⁴

Mistake 7 Relying on contrast enhanced dynamics alone to detect pancreatic cancer

Pancreatic cancer can be overlooked on imaging studies for various reasons such as lesion size or perfusion dynamics.¹¹ While the sensitivity for detection of pancreatic adenocarcinoma >2 cm is about 67–100%, it drops to 50–78% for the detection of smaller tumours. This can be worrisome because up to 30% of pancreatic cancers are <2 cm at presentation.¹⁶ Furthermore, although the majority of pancreatic adenocarcinomas appear hypoenhanced, in 11% of cases they may appear isoenhanced to the surrounding parenchyma.¹⁷

In the aforementioned situations, detection may rely on secondary findings such as pancreatic duct dilatation, glandular atrophy, and abrupt ductal cut-off. Complete cut-off of a portion of the pancreatic duct, as identified on MRCP, in patients who have no other signs of chronic pancreatitis, should always suggest the possibility of malignancy. As mentioned before, DWI can help in increasing diagnostic accuracy.¹ Finally, EUS-FNA directed to the transition zone may help to establish the final diagnosis, with a sensitivity, specificity, positive predictive value, and accuracy of 87.3%, 98.3%, 98.5%, and 92.1%, respectively (figure 7).¹⁸

Mistake 8 Incorrectly identifying pancreatic ductal anatomy (ex pancreas divisum)

The dorsal duct drains the superior and anterior portion of the pancreatic head, usually as a separate duct terminating at the minor papilla, which is located 10–15 mm above and to the right of the major papilla. In approximately 60–70% of the population, the dorsal and ventral pancreatic ducts have fused, resulting in a

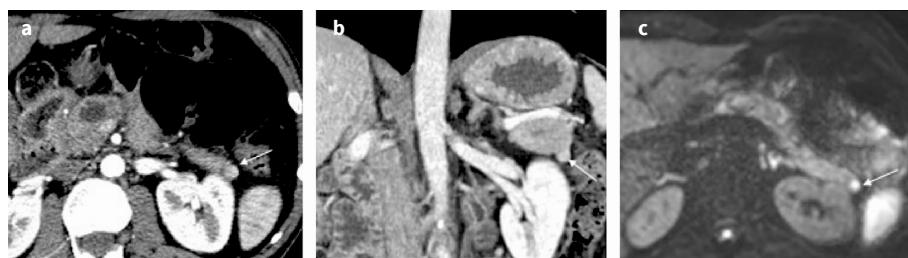


Figure 6 | Intrapancreatic accessory spleen. An intrapancreatic accessory spleen (arrow) is seen as a focal nodular lesion showing the same enhancing pattern of adjacent splenic parenchyma in arterial (a) and venous (b) phase as well as the same signal intensity in DWI (c).

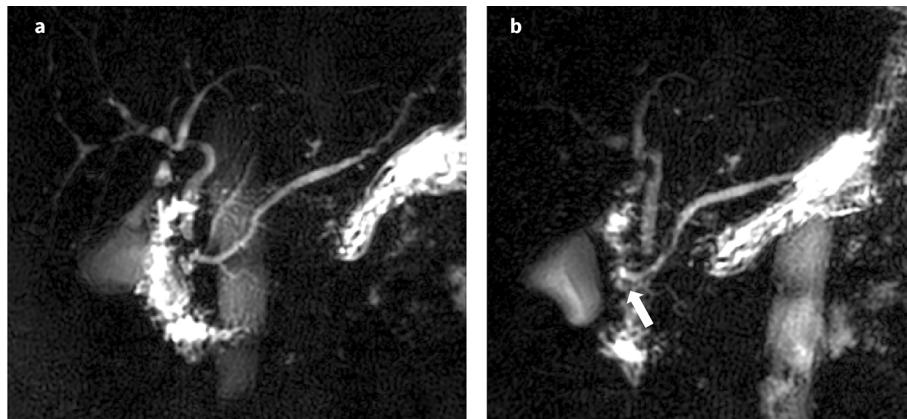


Figure 8 | Pancreas divisum. a | 2D MRCP displays the pancreatic duct anatomy in a selected plane, which does not correctly identify pancreas divisum. b | An additional plane shows the pancreatic duct crossing the common bile duct (arrow) to reach minor papilla and confirms pancreas divisum.

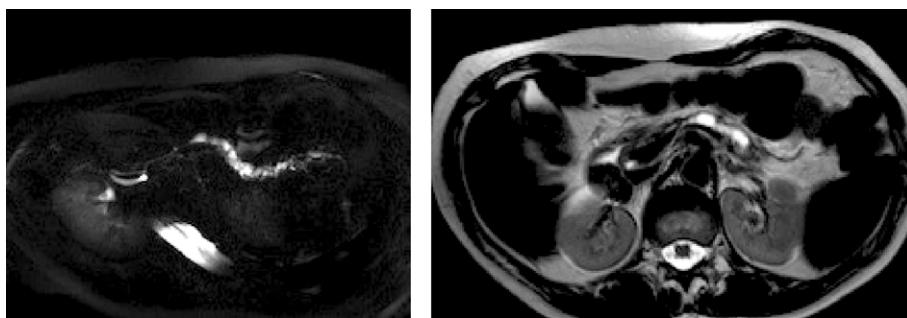


Figure 9 | Main duct IPMN mimicking obstructive chronic pancreatitis. Irregular dilatation of corporeo-caudal main pancreatic duct with parenchymal atrophy as shown by axial 2D MRCP imaging (a) and T2-weighted imaging (b).

communicating dual drainage of the main pancreatic duct, either with a patent or obliterated minor papilla. Variations during the embryological process regarding fusion of the dorsal and ventral pancreas can lead to various congenital variants of the pancreatic ducts.¹⁹

Pancreas divisum is the most common congenital variation and occurs when the ventral and dorsal ducts fail to fuse together. This finding presents with an incidence of 3–7% in patients who are undergoing ERCP and is found in approximately 9% of autopsy cases.¹⁹ MRCP can demonstrate ductal anatomy with precision, and visualization can be enhanced by intravenous secretin, which increases fluid within the duct and therefore better delineates anatomy.¹ It is important to determine pancreatic duct anatomy before pancreatic endotherapy, such as pancreatic sphincterotomy and pancreatic duct drainage in patients with chronic pancreatitis, as preprocedural imaging allows the decision to be made with regards to accessing either the major papilla or the minor papilla (figure 8).²

Mistake 9 Considering every pancreatic duct dilation as chronic pancreatitis

Chronic pancreatitis is an inflammatory process of the pancreas characterized by progressive parenchymal destruction.² Typical morphological

features include parenchymal atrophy, the presence of calcifications, cysts and pancreatic duct irregularity characterized by strictures and dilations.² Both CT and MRI provide accurate diagnosis, but pancreatic duct dilation and cysts can be encountered in other pancreatic diseases.

Intraductal papillary mucinous neoplasms (IPMN) of the pancreas are potentially malignant intraductal epithelial neoplasms that are composed of mucus-producing columnar cells and harbour varying degrees of atypia. The

lesions may involve the side branches (branch type), the main pancreatic duct (main duct type) or both (mixed type).²⁰ The risk of developing cancer is significantly different when comparing main duct/mixed type IPMN with branch duct type IPMN. In patients who have undergone surgical resection, the mean rate of invasive cancer is 43% for main duct/mixed type IPMN compared with 16.5% for branch duct type IPMN.²⁰ Therefore, it is important to accurately diagnose IPMN and provide adequate management.

MRI/MRCP is the imaging technique of choice to confirm the diagnosis of IPMN, as well as defining the presence of high-risk stigmata (mural nodules, thickened cyst wall, main pancreatic duct dilation >5mm) (figure 9).¹ Finally, DWI can increase diagnostic accuracy for the presence of solid malignant components within IPMN (such as mural nodules), with invasive lesions having a lower ADC.¹

Mistake 10 Missing vascular abnormalities

Abdominal pain is a symptom that frequently leads to admittance to the emergency department. Indeed, acute pancreatitis presenting with abdominal pain is one of the leading causes of hospitalization. For patients in whom acute pancreatitis is suspected, contrast-enhanced CT should be performed on admission if there is diagnostic doubt.²¹

Although rare, acute splanchnic venous thrombosis can occur in noncirrhotic patients and is frequently associated with abdominal infections (such as acute pancreatitis), myeloproliferative diseases, or pre-existing coagulation disorders.^{22,23} Furthermore, abdominal pain is the most frequently reported symptom in case of acute splanchnic vein thrombosis. Therefore, it is important to fully study vascular structures and their permeability with CT in patients with abdominal pain of unclear aetiology and pre-existing conditions,

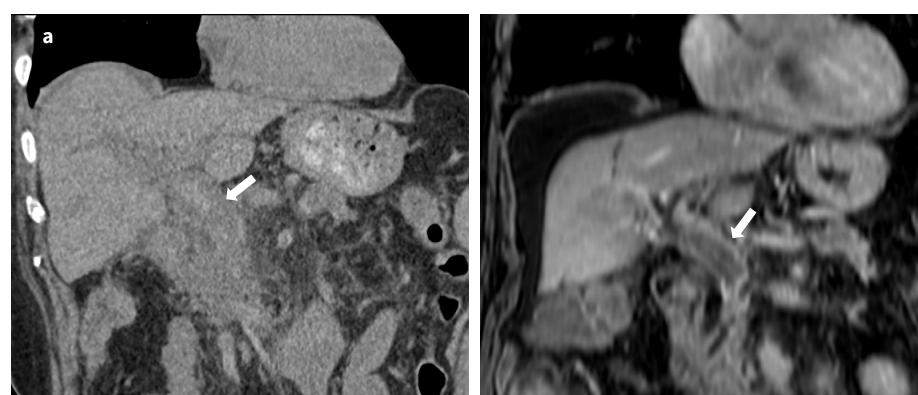


Figure 10 | Acute portal vein thrombosis. a | Non-contrast CT imaging of a patient with acute abdominal pain and renal function impairment in the coronal plane shows peri-hepatic hilar infiltration and a spontaneously hyperdense portal vein (arrow), suggestive of the presence of an intravascular clot. b | T1-weighted contrast enhancement MR confirms acute portal vein thrombosis (arrow).

if renal function allows contrast injection (figure 10).

Additional vascular abnormalities that should not be neglected include peripancreatic arterial pseudoaneurysms, which may develop as a complication of acute pancreatitis or pancreatic surgery, and the locations most frequently involved are the splenic artery (60%) and the hepatic artery (20%).¹¹ On contrast-enhanced CT, a pseudoaneurysm appears as an oval or round lesion with similar enhancement to the abdominal aorta. Diagnosis of pseudoaneurysms is crucial to allow prompt management by angiography, as they may otherwise lead to potentially fatal bleeding.

References

1. Harrington KA, Shukla-Dave A, Paudyal R, et al. MRI of the pancreas. *J Magn Reson Imaging* Epub ahead of print April 17, 2020. DOI: 10.1002/jmri.27148.
2. Zamboni GA, Ambrosetti MC, Pezzullo M, et al. Optimum imaging of chronic pancreatitis. *Abdom Radiol (NY)* 2020; 45: 1410–1419.
3. Yam BL and Siegelman ES. MR imaging of the biliary system. *Radiol Clin North Am* 2014; 52: 725–755.
4. Brunt LM, Deziel DJ, Teleni DA, et al. Safe cholecystectomy multi-society practice guideline and state of the art consensus conference on prevention of bile duct injury during cholecystectomy. *Ann Surg* 2020; 272: 3–23.
5. Rhaiem R, Piardi T, Renard Y, et al. Preoperative magnetic resonance cholangiopancreatography before planned laparoscopic cholecystectomy: is it necessary? *J Res Med Sci* 2019; 24: 107.
6. Katabathina VS, Dasyam AK, Dasyam N, et al. Adult bile duct strictures: role of MR imaging and MR cholangiopancreatography in characterization. *Radiographics* 2014; 34: 565–586.
7. Novikov A, Kowalski TE and Loren DE. Practical management of indeterminate biliary strictures. *Gastrointest Endosc Clin N Am* 2019; 29: 205–214.
8. Seo N, Kim SY, Lee SS, et al. Sclerosing cholangitis: clinicopathologic features, imaging spectrum, and systemic approach to differential diagnosis. *Korean J Radiol* 2016; 17: 25–38.
9. Kamisawa T, Zen Y, Nakazawa T, et al. Advances in IgG4-related panreatobiliary diseases. *Lancet Gastroenterol Hepatol* 2018; 3: 575–585.
10. Fernandez YVM and Arvanitakis M. Early diagnosis and management of malignant distal biliary obstruction: a review on current recommendations and guidelines. *Clin Exp Gastroenterol* 2019; 12: 415–432.
11. Vernuccio F, Borhani AA, Dioguardi Burgio M, et al. Common and uncommon pitfalls in pancreatic imaging: it is not always cancer. *Abdom Radiol (NY)* 2016; 41: 283–294.
12. Klauss M, Maier-Hein K, Tjaden C, et al. IVIM DW-MRI of autoimmune pancreatitis: therapy monitoring and differentiation from pancreatic cancer. *Eur Radiol* 2016; 26: 2099–2106.
13. Lee NJ, Hraban RH and Fishman EK. Pancreatic neuroendocrine tumor: review of heterogeneous spectrum of CT appearance. *Abdom Radiol (NY)* 2018; 43: 3025–3034.
14. Renno A, Hill M, Abdel-Aziz Y, et al. Diagnosis of intrapancreatic accessory spleen by endoscopic ultrasound-guided fine-needle aspiration mimicking a pancreatic neoplasm: a case report and review of literature. *Clin J Gastroenterol* 2020; 13: 287–297.
15. Coquia SF, Kawamoto S, Zaheer A, et al. Intrapancreatic accessory spleen: possibilities of computed tomography in differentiation from nonfunctioning pancreatic neuroendocrine tumor. *J Comput Assist Tomogr* 2014; 38: 874–878.
16. Bronstein YL, Loyer EM, Kaur H, et al. Detection of small pancreatic tumors with multiphasic helical CT. *AJR Am J Roentgenol* 2004; 182: 619–623.
17. Prokesch RW, Chow LC, Beaulieu CF, et al. Local staging of pancreatic carcinoma with multi-detector row CT: use of curved planar reformations initial experience. *Radiology* 2002; 225: 759–765.
18. Wang W, Shpaner A, Krishna SG, et al. Use of EUS-FNA in diagnosing pancreatic neoplasm without a definitive mass on CT. *Gastrointest Endosc* 2013; 78: 73–80.
19. Yu J, Turner MA, Fulcher AS, et al. Congenital anomalies and normal variants of the pancreaticobiliary tract and the pancreas in adults: Part 2, pancreatic duct and pancreas. *AJR Am J Roentgenol* 2006; 187: 1544–1553.
20. Crippa S, Arcidiacono PG, De Cobelli F, et al. Review of the diagnosis and management of intraductal papillary mucinous neoplasms. *United European Gastroenterol J* 2020; 8: 249–255.
21. Arvanitakis M, Dumonceau JM, Albert J, et al. Endoscopic management of acute necrotizing pancreatitis: European Society of Gastrointestinal Endoscopy (ESGE) evidence-based multidisciplinary guidelines. *Endoscopy* 2018; 50: 524–546.
22. Keil R, Kozeluhova J, Dolina J, et al. Acute portal vein thrombosis in noncirrhotic patients different prognoses based on presence of inflammatory markers: a long-term multicenter retrospective analysis. *Scand J Gastroenterol* 2019; 54: 1379–1384.
23. Junare PR, Udgirkar S, Nair S, et al. Splanchnic venous thrombosis in acute pancreatitis: does anticoagulation affect outcome? *Gastroenterology Res* 2020; 13: 25–31.

Your panreatobiliary imaging briefing

UEG week

- ‘Video Case Session I: Diagnosis and management of biliary and pancreatic strictures’ session at 25th UEG Week 2017 [<https://ueg.eu/library/session/video-case-session-i-diagnosis-and-management-of-biliary-and-pancreatic-strictures/149/1809>].
- ‘Malignant biliary obstruction: Diagnostic and therapeutic approach’ session at 25th UEG Week 2017 [<https://ueg.eu/library/session/malignant-biliary-obstruction-diagnostic-and-therapeutic-approach/149/1826>].
- ‘Advanced panreato-biliary imaging’ session at UEG Week 2016 [<https://ueg.eu/library/session/advanced-panreato-biliary-imaging/144/1621>]
- ‘Diagnosis and management of bile stones and its complications’ session at UEG Week 2015 [<https://ueg.eu/library/session/diagnosis-and-management-of-bile-stones-and-its-complications/109/1363>].

Standards and Guidelines

- Manes G, et al. Endoscopic management of common bile duct stones: European Society of Gastrointestinal Endoscopy (ESGE) guideline. *Endoscopy* 2019; 51: 472–491 [<https://ueg.eu/library/endoscopic-management-of-common-bile-duct-stones-european-society-of-gastrointestinal-endoscopy-esge-guideline/231354>].
- Williams EJ, et al. Updated guideline on the management of common bile duct stones (CBDS). *Gut* 2017; 66: 765–782 [<https://ueg.eu/library/updated-guideline-on-the-management-of-common-bile-duct-stones-cbds/174756>].
- Tanaka M, et al. Revisions of international consensus Fukuoka guidelines for the management of IPMN of the Pancreas. *Pancreatology* 2017; 17: 738–753. [<https://www.sciencedirect.com/science/article/pii/S1424390317305161>].
- Madden A, et al. National Institute for Health and Care Excellence Clinical Guideline 188. Gallstone disease: diagnosis and management of cholelithiasis, cholecystitis and choledocholithiasis. October 2014 [<https://ueg.eu/library/gallstone-disease-diagnosis-and-management-of-cholelithiasis-cholecystitis-and-choledocholithiasis/141805>].