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Uncommon presentations of common pancreatic neoplasms: a pictorial essay

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Abstract

Pancreatic neoplasms are a wide group of solid and cystic lesions with different and often characteristic imaging features, clinical presentations, and management. Among solid tumors, ductal adenocarcinoma is the most common: it arises from exocrine pancreas, comprises about 90% of all pancreatic neoplasms, and generally has a bad prognosis; its therapeutic management must be multidisciplinary, involving surgeons, oncologists, gastroenterologists, radiologists, and radiotherapists. The second most common solid pancreatic neoplasms are neuroendocrine tumors: they can be divided into functioning or non-functioning and present different degrees of malignancy. Cystic pancreatic neoplasms comprise serous neoplasms, which are almost always benign, mucinous cystic neoplasms and intraductal papillary mucinous neoplasms, which can vary from benign to frankly malignant lesions, and solid pseudopapillary tumors. Other pancreatic neoplasms, such as lymphoma, metastases, or pancreatoblastoma, are rarely seen in clinical practice and have different and sometimes controversial managements. Rare clinical presentations and imaging appearance of the most common pancreatic neoplasms, both solid and cystic, are more frequently seen and clinically relevant than rare pancreatic tumors; their pathologic and radiologic appearances must be known to improve their management. The purpose of this paper is to present some rare or uncommon clinical and radiological presentations of common pancreatic neoplasms providing examples of multi-modality imaging approach with pathologic correlations, thus describing the histopathological bases that can explain the peculiar imaging features, in order to avoid relevant misdiagnosis and to improve lesion management.

Key words: Pancreatic neoplasms—Ductal adenocarcinoma—Neuroendocrine neoplasms—Serous cystadenoma—Mucinous cystic neoplasms—Intraductal papillary mucinous neoplasms

Rare solid and cystic pancreatic tumors are heterogeneous neoplasms, infrequently seen in individual clinical practice; distinct pathological entities can be found, according to the World Health Organization (WHO) classification, comprising for example anaplastic carcinoma, pancreatoblastoma, acinar cell carcinoma, mesenchymal tumors, metastases, and lymphoma [1].

Uncommon presentations of common pancreatic tumors are more frequent and clinically relevant than rare pancreatic tumors. Typical features of common pancreatic neoplasms at different imaging modalities are well known and routinely used for diagnosis in everyday clinical practice, but possible uncommon presentations must be also known in order to avoid relevant misdiagnosis.

The purpose of this paper is to show rare clinical presentations and imaging features of common pancreatic neoplasms, through a multi-modality imaging approach with pathologic correlations.

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Solid neoplasms

Pancreatic solid neoplasms are a group of pathological entities with predominant frequency of one cellular histotype arising from the exocrine pancreas. The most common solid pancreatic neoplasm is pancreatic ductal adenocarcinoma. Pancreatic solid neoplasms arising from endocrine pancreas are more rare than those from exocrine pancreas, but of great interest for differential diagnosis.

Ductal adenocarcinoma

Ductal adenocarcinoma is the most common primary malignancy of the pancreas (85%–90% of all pancreatic neoplasms). The vast majority of these tumors are inoperable at time of diagnosis, mostly due to their advanced stage, when they cause symptoms as pain, weight loss, jaundice, diabetes and ascites. Ductal adenocarcinoma usually presents as a solid and firm mass, with infiltrative margins [2].

Common imaging findings

Common imaging features of ductal adenocarcinoma are summarized in Table 1.

Generally, ductal adenocarcinoma presents as a unifocal solid mass with ill-defined and infiltrative margins, causing upstream dilation of the main pancreatic duct and common bile duct when located in the pancreatic head. After contrast medium administration it usually shows hypoenhancement pattern.

At ultrasound (US), it typically presents as a solid hypoechoic mass [3]; at unenhanced computed tomography (CT) or magnetic resonance (MR), the tumor is isodense/hypointense on T1-weighted images and with variable signal intensity on T2-weighted images [4].

After contrast medium administration, this tumor shows poor enhancement during all phases at contrast-enhanced ultrasound (CEUS) [5, 6]; at CT and MR, the tumor becomes better depictable during the pancreatic phase, when there is the maximum difference between the hyperdense/hyperintense normal pancreatic parenchyma and the markedly hypodense/hypointense neoplastic tissue, thus reaching its best conspicuity; delayed scans may show a mild intra-tumoral pooling of contrast medium, reflected by a slightly hyperdense/hyperintense appearance, related to the high amount of fibrotic tissue within the tumor [3].

Uncommon presentations

Ductal adenocarcinoma may have several uncommon presentations.

The most frequent uncommon imaging variants are the isoenhancing pattern, the cystic and the pancreatitis-like appearance of the tumor. Acute pancreatitis is an uncommon but peculiar clinical presentation of this tumor.

Isoenhancing pattern. Ductal adenocarcinoma can be isovascular at imaging, therefore not directly identifiable.

It has been reported that up to 11% of pancreatic adenocarcinomas show no density difference compared to the surrounding pancreatic tissue at CT, the so-called isoattenuating pancreatic adenocarcinomas [7–9] (Fig. 1).

Yoon et al. [8] reported that 27% of small (≤20 mm) pancreatic adenocarcinomas are isoattenuating, therefore not directly identifiable without the help of some secondary signs, as upstream ductal dilation; despite this, dimensional assessment and local staging need the direct visualization of the tumor.

Moreover, well-differentiated pancreatic adenocarcinomas, which have a better survival rate after resection, are reported to be isoattenuating in more than 50% of cases [8, 10].

MR and positron emission tomography (PET), but also US, CEUS and endoscopic ultrasound (EUS) may be useful for detecting invisible lesions at CT or if CT findings are inconclusive [9]. In these cases, in fact, a "simple" US can solve the problem, because the lesion can be usually immediately detected owing to its hypoechoic appearance in contrast to the usual slightly hyperechoic pancreatic parenchyma with significant acoustic impedance difference and subsequent better conspicuity of the tumor [11].

Table 1. Main common imaging aspects of ductal adenocarcinomas and neuroendocrine tumors at different imaging methods

Ductal adenocarcinoma	US/CEUS	CT	MR
Pre-contrast	Hypoechoic	Isodense	Hypointense (T1) Variable (T2)
Pancreatic phase	Hypovascular	Hypodense	Hypointense
Portal phase	Hypovascular	Hypodense	Hypointense
Delayed phase	Hypovascular	Hyperdense	Hyperintense
Neuroendocrine tumors	**US**	**CT**	**MR**
Pre-contrast	Hypoechoic	Isodense or hypodense	Hypointense (T1) Hyperintense (T2)
Dynamic phase	Hypervascular	Hypervascular	Hypervascular

US, ultrasound; CEUS, contrast-enhanced ultrasound; CT, computed tomography; MR, magnetic resonance

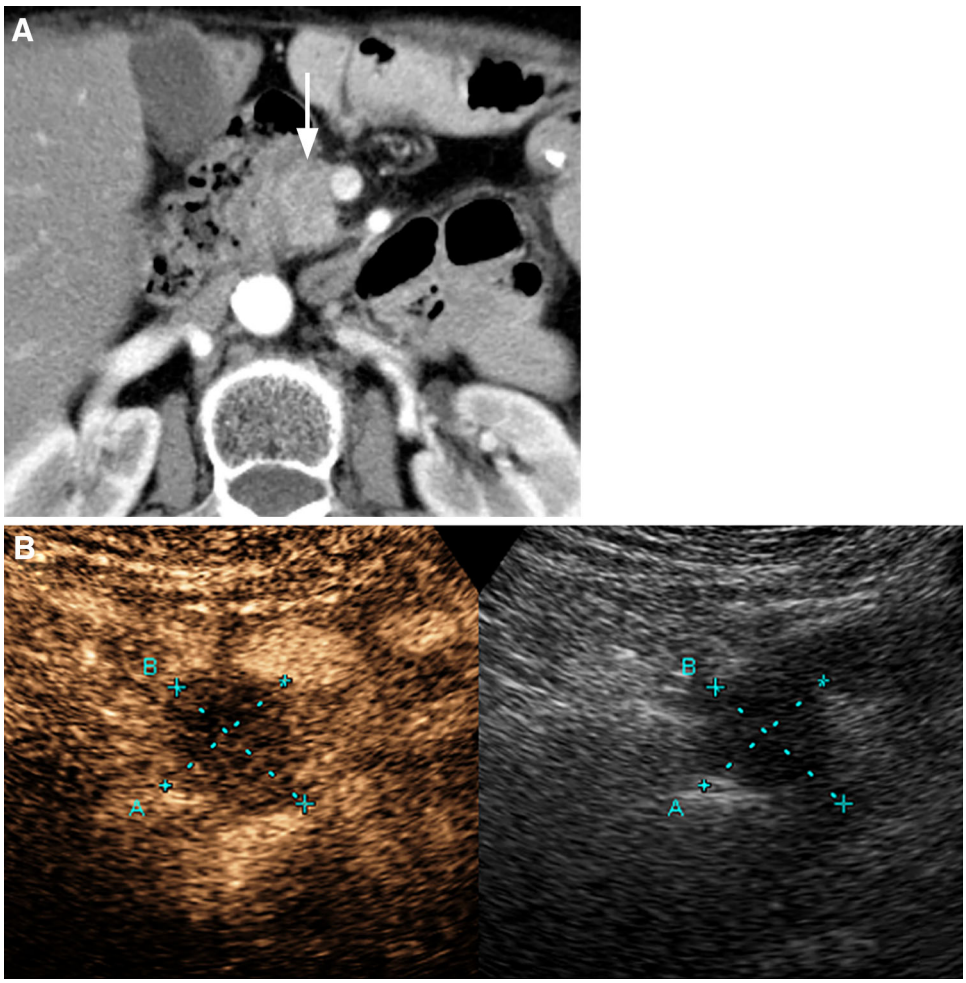

Fig. 1. Isoenhancing ductal adenocarcinoma of the pancreatic head. **A** Pancreatic phase axial CT image does not show clear focal lesions within the pancreas, despite a small inhomogeneously isodense mass can be suspected, due to the lack of a clear-cut margin between pancreatic head and the superior mesenteric vein (*arrow*). **B** Trans-abdominal CEUS examination, transverse scan, clearly shows a hypoechoic (see calipers in B-mode reference image on the right), ill-defined and markedly hypovascular lesion in the pancreatic head. Final pathologic diagnosis was ductal adenocarcinoma.

Cystic/microcystic appearance and cyst epiphenomenon. Ductal adenocarcinoma can show different cystic features, which have different pathologic bases, thus resembling other primary or secondary cystic tumors of the pancreas.

Cystic changes are not a rare occurrence in histologic specimens, since they can be present in up to 8% of ductal adenocarcinomas. These cystic changes differ in nature and pathogenesis: neoplasms can develop cystic changes because of extensive central necrosis (Fig. 2), or cystic structures can represent a true neoplastic component (Figs. 3, 4), or they can be retention cysts located at tumor periphery, or pseudocysts attached to tumoral borders [12].

In less than 1% of cases ductal adenocarcinoma undergoes cystic changes due to necrosis and hemorrhage. Usually a large central cyst develops because of necrosis; but what appears at imaging to be a macrocyst, may often be solid, nonviable tissue surrounded by a cuff of viable carcinoma [13]. The presence of necrotic or cystic degeneration makes the tumor hyperintense on T2-weighted images (Fig. 2) [14]; such cases can sometimes be misdiagnosed as pseudocysts [13]: the absence of a clinical history of acute pancreatitis may strength the hypothesis of ductal adenocarcinoma.

Microcystic appearance is even more uncommon; these forms are usually histologically defined "large duct type adenocarcinomas" or "large gland type adenocarcinomas" and may show a similar imaging appearance to microcystic serous cystadenoma (SCA) (Fig. 4) [12].

Even very small ductal adenocarcinoma can infiltrate and obstruct pancreatic ductal system, thus leading to cystic dilation of the upstream secondary ducts, therefore being similar at imaging to intraductal papillary mucinous neoplasms (IPMNs). Moreover, at histological analysis the dilated ducts can show reactive epithelial changes, which may be indistinguishable from IPMNs or mucinous cystic neoplasms (MCNs) [13, 15].

Acute pancreatitis epiphenomenon. Ductal adenocarcinoma may cause acute pancreatitis due to the infiltration and subsequent obstruction of Wirsung's duct, rather than classical symptoms. This event has been reported in about 3% of cases [16].

Imaging usually shows predominantly signs of acute pancreatitis (size increase of the pancreatic gland, decreased echogenicity/density/signal intensity, hypovascularization, local complications) (Fig. 5).

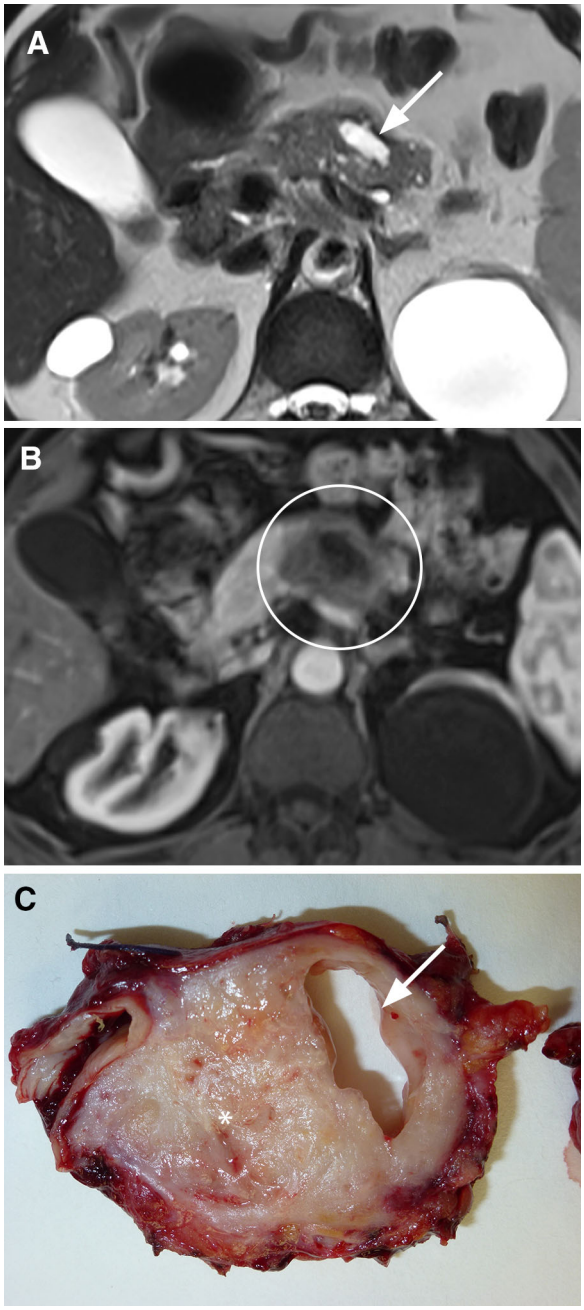

Fig. 2. Ductal adenocarcinoma with intratumoral cystic degeneration. **A** T2-weighted half-Fourier single shot turbo spin echo examination (HASTE) axial image shows a hyperintense (fluid) oval area within pancreatic body (*arrow*). **B** Pancreatic phase T1-weighted gradient echo (GRE) fat-sat 3D (volume-interpolated breath-hold examination—VIBE) axial image shows a hypovascular mass (*circle*), with avascular cystic component. **C** Resection specimen (transverse cut) displays a highly fibrotic ductal adenocarcinoma (*asterisk*), containing a cystic area (*arrow*).

The direct visualization of a focal, well-defined, hypoechoic area at the first examination of a patient with acute pancreatitis should raise the suspicion of an obstructing neoplasm, especially if other causes of acute pancreatitis (biliary stones, alcoholic abuse, etc.) have been excluded and if the lesion is located downstream to the inflammatory changes of the gland. [17]. In these cases the repetition of imaging, especially EUS, after the acute inflammation resolution, is important, as surrounding inflammation can make the diagnosis of ductal adenocarcinoma more difficult [18].

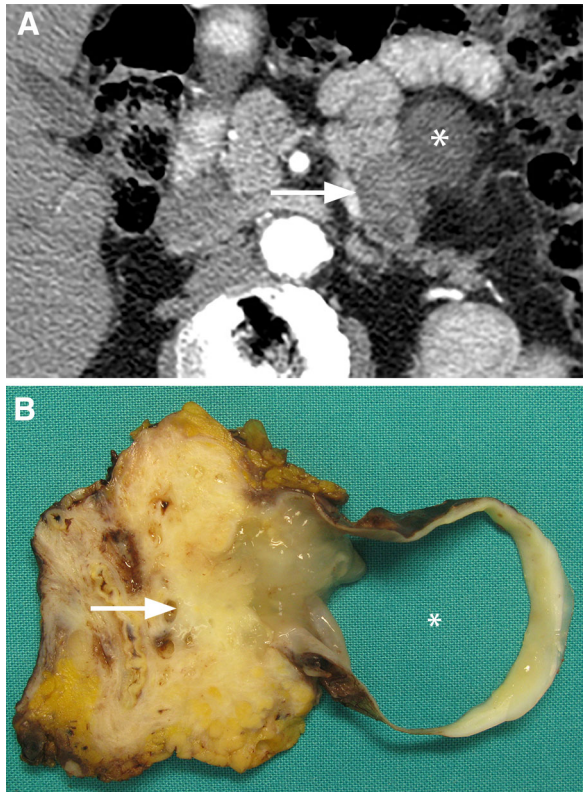

Fig. 3. Ductal adenocarcinoma with huge cystic portion. **A** Pancreatic phase CT axial image displays an oval-shaped hypovascular lesion in pancreatic tail (*arrow*), with a huge exophytic cystic lesion next to its lateral margin (*asterisk*). **B** Resection specimen (longitudinal cut) well demonstrate that the cystic lesion (*asterisk*) is part of a ductal adenocarcinoma (*arrow*).

Pancreatitis-like shape. Ductal adenocarcinoma can have a pancreatitis-like shape, both focal and diffuse (Fig. 6). Despite both these entities may present indistinguishable imaging features [19], contrast-enhanced examinations are essential for differential diagnosis. Particularly, CEUS can improve the differential diagnosis between ductal adenocarcinoma, which remains hypoechoic in all contrast-enhanced phases, and inflammatory masses, that display enhancement similar to the adjacent pancreatic parenchyma [20]. It has been reported that a relative demarcation of the mass in respect to background pancreas on MR gadolinium-enhanced images favors the neoplastic hypothesis [21].

The presence of the ''double-duct sign'', particularly at MR cholangiopancreatography (MRCP), is consid-

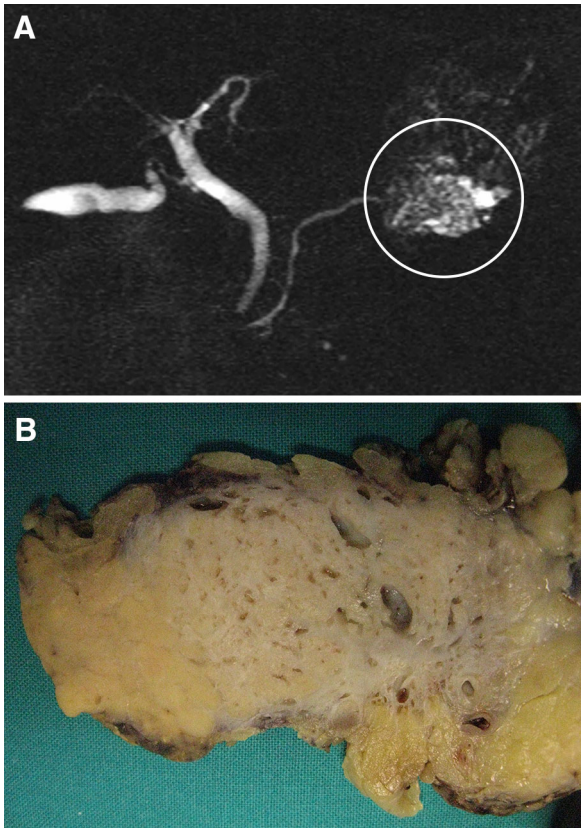

Fig. 4. Ductal adenocarcinoma with microcystic appearance. **A** Para-coronal MRCP image displaying a microcystic lesion in the pancreatic tail (*circle*). **B** Resection specimen (transverse cut), revealing a ductal adenocarcinoma with microcystic composition.

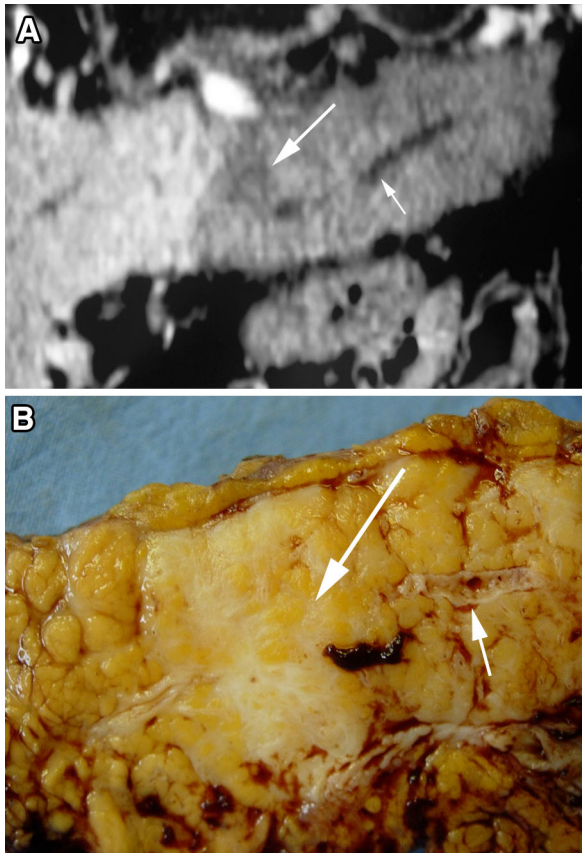

Fig. 5. Mild acute pancreatitis as an epiphenomenon of a ductal adenocarcinoma. **A** Para-coronal reformatted pancreatic phase CT image, showing a marked distinction between a homogeneously vascularized pancreatic body and a poorly-vascularized pancreatic tail; between the two pancreatic portions a small hypodense lesion (*long arrow*), causing a slight upstream dilation of the main pancreatic duct (*short arrow*), can be seen. **B** Resection specimen (longitudinal cut), displaying a small ductal adenocarcinoma (*long arrow*) causing upstream dilation of the main pancreatic duct (*short arrow*).

ered highly suggestive of a pancreatic head tumor [22]; moreover, in inflammatory masses the narrowing of the dilated duct tends to be multiple and gradual, while in pancreatic cancer there is usually a single abrupt interruption [23].

The so-called "duct-penetrating sign", defined as the presence of a stenotic main pancreatic duct coursing through the mass, which enlarges after secretin administration, has a sensitivity of 85% and a specificity of 96% for the distinction between focal inflammatory mass and pancreatic cancer [24].

The presence of a diffuse pancreatic involvement rather than a focal mass-forming appearance can make the radiological differential diagnosis even more difficult.

Neuroendocrine tumors

Pancreatic neuroendocrine tumors (pNETs) arise from neuroendocrine cells and encompass different degrees of malignancy, from well-differentiated lesions (ENETS grades G1 and G2) to poorly differentiated ones (ENETS grade G3—endocrine carcinomas) [25]; they can be generally divided into functioning or non-(hyper)-functioning, based on the presence or absence of symptoms related to an improper hormonal production [26].

Common imaging findings

Common imaging features of pNETs are summarized in Table 1.

Typically, functioning or small pNETs are well-defined, rounded and encapsulated lesions, hypoechoic at US and hypodense-isodense at unenhanced CT; at MR, endocrine tumors are better visualized with fat-suppressed sequences (particularly T2-weighted fat-sat images), and they present as homogeneously hypointense on T1-weighted images and slightly hyperintense on T2-weighted images. Nonfunctioning tumors are frequently larger at presentation, with inhomogeneous appearance mainly due to necrotic areas and calcifications [27].

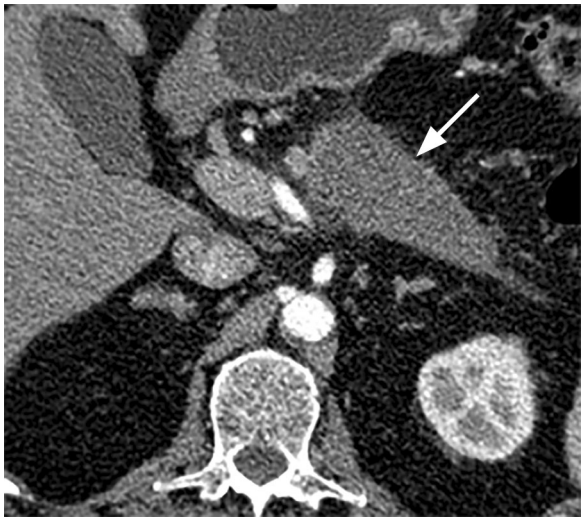

Fig. 6. Pancreatitis-shaped ductal adenocarcinoma. Pancreatic phase axial CT scan, displaying an enlargement of the pancreatic body-tail (*arrow*), with loss of pancreatic lobulated contours and hypovascularity, thus resembling a mild acute pancreatitis.

The feature that generally joins functioning and non-(hyper)functioning endocrine pancreatic neoplasms is their hypervascular appearance at contrast-enhanced examinations (CEUS, CE-CT, CE-MRI) [28].

At diffusion-weighted images, pNETs usually show diffusion restriction, being hyperintense at high b values images. It has been reported that pNETs with different grades of differentiation can be distinguished evaluating their ADC values: endocrine carcinomas have significantly lower mean ADC values compared to normal pancreatic tissue and to well-differentiated pNETs [29, 30].

Uncommon presentations

The most common atypical presentations of pNETs are the hypoenhancing pattern, the intravessels-growing, the cystic and the calcified variants; pancreatic carcinoid is an extremely rare pNET with distinctive imaging features.

Hypoenhancing pattern. Non-functioning neuroendocrine tumors can be hypovascular, thus mimicking a ductal adenocarcinoma.

This appearance is directly related to the amount of stroma, which is dense and hyalinized, and to the small size of the lesion or of its vascular network [27, 31].

It is crucial to carefully evaluate other imaging findings, as size, margins, growing pattern and Wirsung's duct aspect, to differentiate them from ductal adenocarcinoma, because these two entities may have a completely different management.

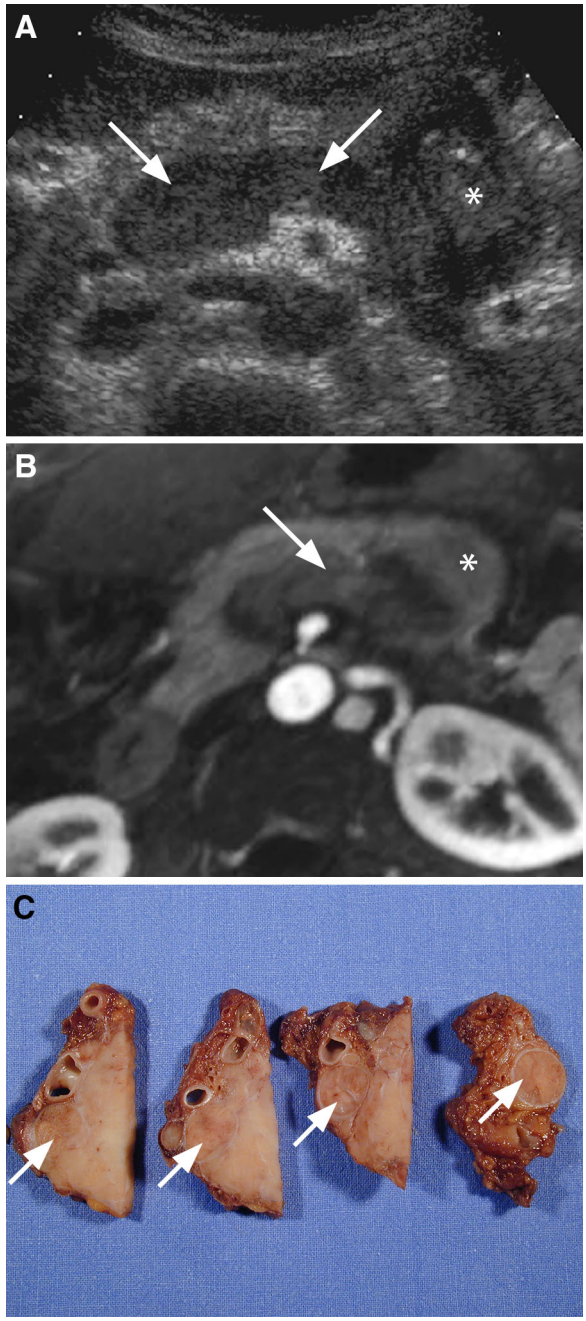

Fig. 7. Intravessel-growing neuroendocrine tumor. **A** Transabdominal B-mode ultrasound, transverse scan, showing a solid, slightly hyperechoic tissue within the lumen of the splenic vein and the spleno-mesenteric confluence (thrombus, *arrows*). A inhomogeneously hypoechoic pancreatic body mass is also visible (*asterisk*). **B** Pancreatic phase VIBE axial image, showing a thrombus with mild enhancement within the lumen of the splenic vein (neoplastic thrombus, *arrow*). A pancreatic body mass is also visible (*asterisk*). **C** Resection specimen (sequential transverse cuts), showing the growth of the endocrine tumor within the lumen of the splenic vein (*arrows*).

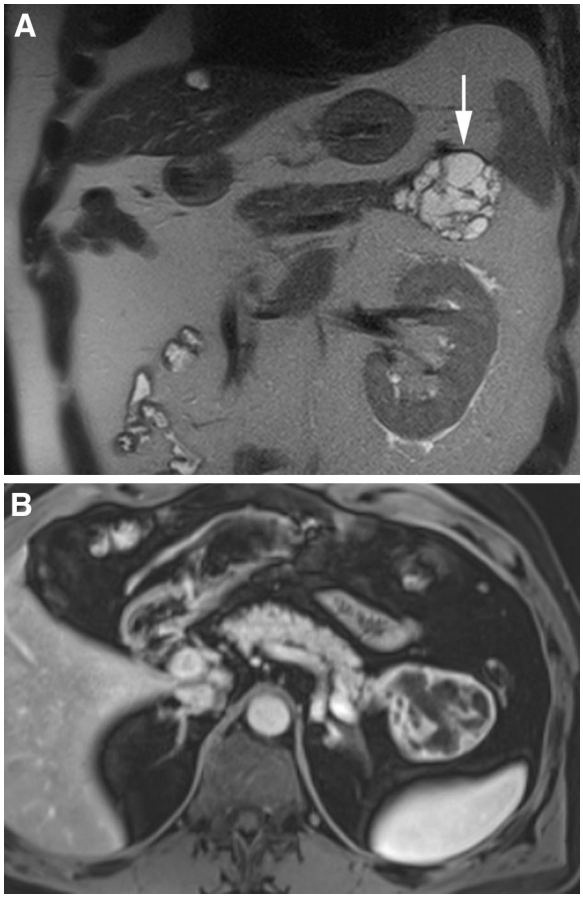

Fig. 8. Endocrine tumor with micro-/macro-cystic aspect. **A** T2-weighted HASTE coronal image showing an oval, well-defined multilocular cystic lesion within the pancreatic tail (*arrow*); cysts content is homogenously hyperintense. **B** Pancreatic phase T1 GRE fat-sat 3D (VIBE) axial image shows marked enhancement of cyst's walls and septa.

Intravessels-growing. In some cases, neuroendocrine tumors, especially carcinomas, can present a peculiar intravascular growth (Fig. 7). This particular growing pattern is usually confined to peri-pancreatic vessels, such as splenic and portal veins, producing a neoplastic thrombus.

Cystic. Neuroendocrine neoplasms may present different cystic features (Figs. 8, 9, 10).

Larger non-functioning endocrine tumors may show cystic degeneration in up to 10% of cases [32].

In contrast to cystic changes that may develop in other solid tumors, cysts formation in these neoplasms does not appear to be entirely due to necrosis. Rather, the cysts are lined by a cuff of well-preserved neoplastic endocrine cells, hypervascular at dynamic imaging, and filled with a clear fluid instead of necrotic debris. Cyst is usually unilocular and centrally located within the NET [32].

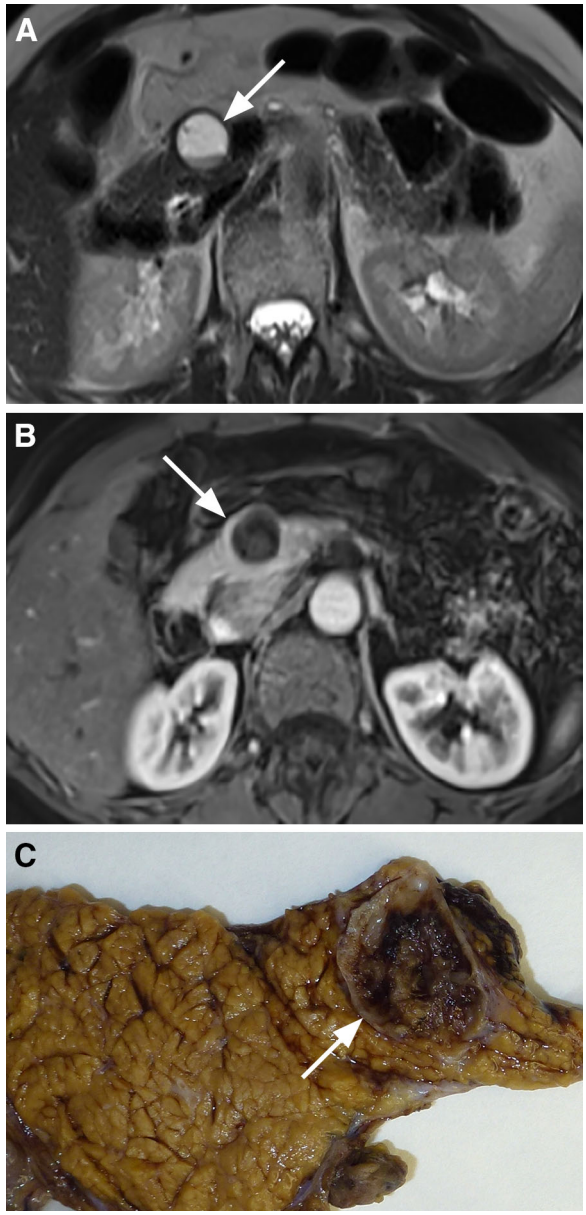

Fig. 9. Small cystic endocrine tumor. **A** T2-weighted HASTE axial image showing a small rounded, well-defined cystic lesion with a fluid–fluid level in the pancreatic head (*arrow*). **B** Pancreatic phase VIBE axial image, displaying the presence of hypervascularization of the well-defined, thin wall of the cystic lesion (*arrow*). **C** Resection specimen (longitudinal cut), showing a cystic endocrine tumor with a well-defined thin wall (*arrow*).

Neuroendocrine neoplasms, especially if small, can also be quite-exclusively cystic, making the differential diagnosis with cystic neoplasms, especially MCNs or unilocular SCA, extremely difficult.

Calcified. Calcifications may occur in NETs (Fig. 11), but intra-tumoral calcifications are not seen in ductal adenocarcinoma. More commonly calcifications occur

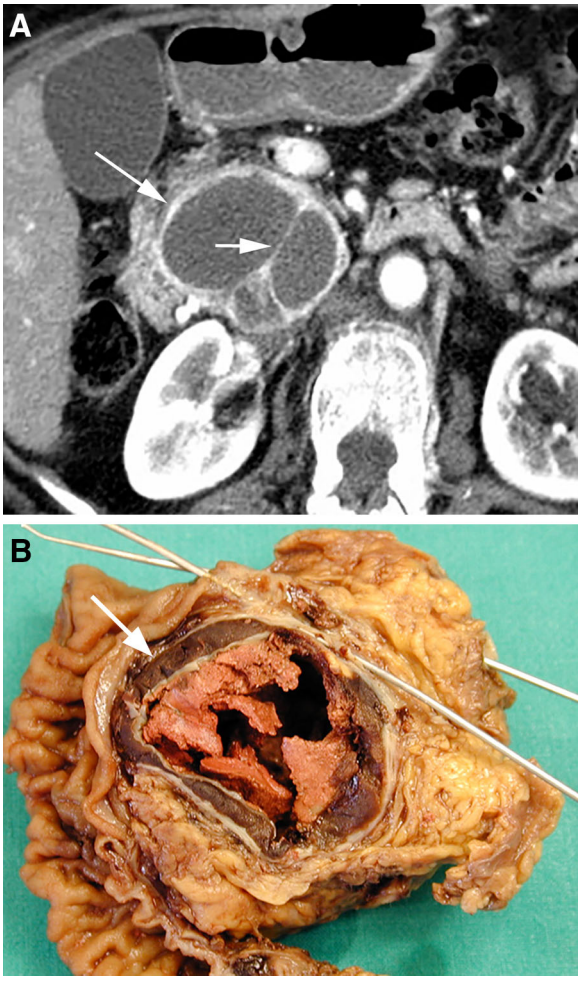

Fig. 10. Cystic endocrine tumor. **A** Pancreatic phase axial CT image, showing a multilocular, well-defined pancreatic head lesion with thick enhancing walls (*long arrow*) and thin vascularized septa (*short arrow*). **B** Resection specimen (transverse cut), showing the complex cystic appearance of the endocrine tumor, with a typical thick wall (*arrow*).

within non-functioning and larger neoplasms [33]: a direct correlation has been reported between tumor size and increasing likelihood of cystic necrosis with subsequent development of dystrophic calcifications within the mass. However, insulinoma, which is the most common functioning islet cell tumor and is generally small at diagnosis, may contain calcifications in up to 20% of cases. Overall, calcifications can be found in NETs in up to 16% of cases; these calcifications tend to be focal, coarse, irregular, and centrally located [34].

Pancreatic carcinoid. Serotonin-producing neuroendocrine tumors are extremely rare in the pancreas (Fig. 12).

They arise from pancreatic enterochromaffin cells and they secrete serotonin [35].

Although classified as neuroendocrine tumors, serotonin-producing tumor is a separate entity and is more properly known as carcinoid tumor of the pancreas [36].

In his large reviews of 8305 cases of carcinoid tumors, Modlin et al. [37] found only 46 (0.55%) carcinoids in the pancreas. Pancreatic carcinoids are associated with a high malignancy rate [38]; despite this, functioning carcinoids associated with carcinoid syndrome due to liver metastases are overall extremely rare [39].

Carcinoids are commonly associated with pancreatic duct stenosis: this could be due to serotonin-induced fibrosis, with subsequent upstream ductal dilation and parenchymal atrophy, which are usually disproportionate to tumor size [40], but may cause an imaging overlap with small ductal adenocarcinomas. Regarding clinical presentation, Wirsung's duct obstruction can also lead to acute pancreatitis episodes [14].

Few papers described imaging findings of pancreatic carcinoid: marked contrast enhancement, inner calcifications, cystic changes, and at times hepatic metastases; however, some of these tumors are not hypervascular or not identifiable at all at imaging, due to very high amount of intratumoral fibrosis [40–44].

Cystic neoplasms

Pancreatic cystic neoplasms are a wide group of pathological entities

Epithelial cystic neoplasms, primarily represented by serous and mucinous tumors, represent the majority, followed by other tumors potentially presenting with cystic changes. While serous tumors are quite exclusively represented by SCA, mucinous tumors can be divided into IPMNs and MCNs.

SCA, MCNs and solid-pseudopapillary tumors never communicate with pancreatic ductal system: this is a key feature to distinguish them from IPMNs.

Serous cystadenoma

SCA is a cystic tumor with a typical multilocular "honeycomb" architecture due to the presence of multiple microcysts (<20 mm), thin walls and multiple septa oriented toward a central scar [45].

Common imaging findings

Common imaging features of SCA are summarized in Table 2.

The typical lobulated, "cloud-like" morphology is usually clearly depictable at imaging. The cystic content is anechoic at US, hypodense at CT and hypointense on T1-weighted images at MR; T2-weighted images clearly demonstrate the microcystic pattern with hyperintense content.

After intravenous administration of contrast material, the calcified central scar in SCA is usually

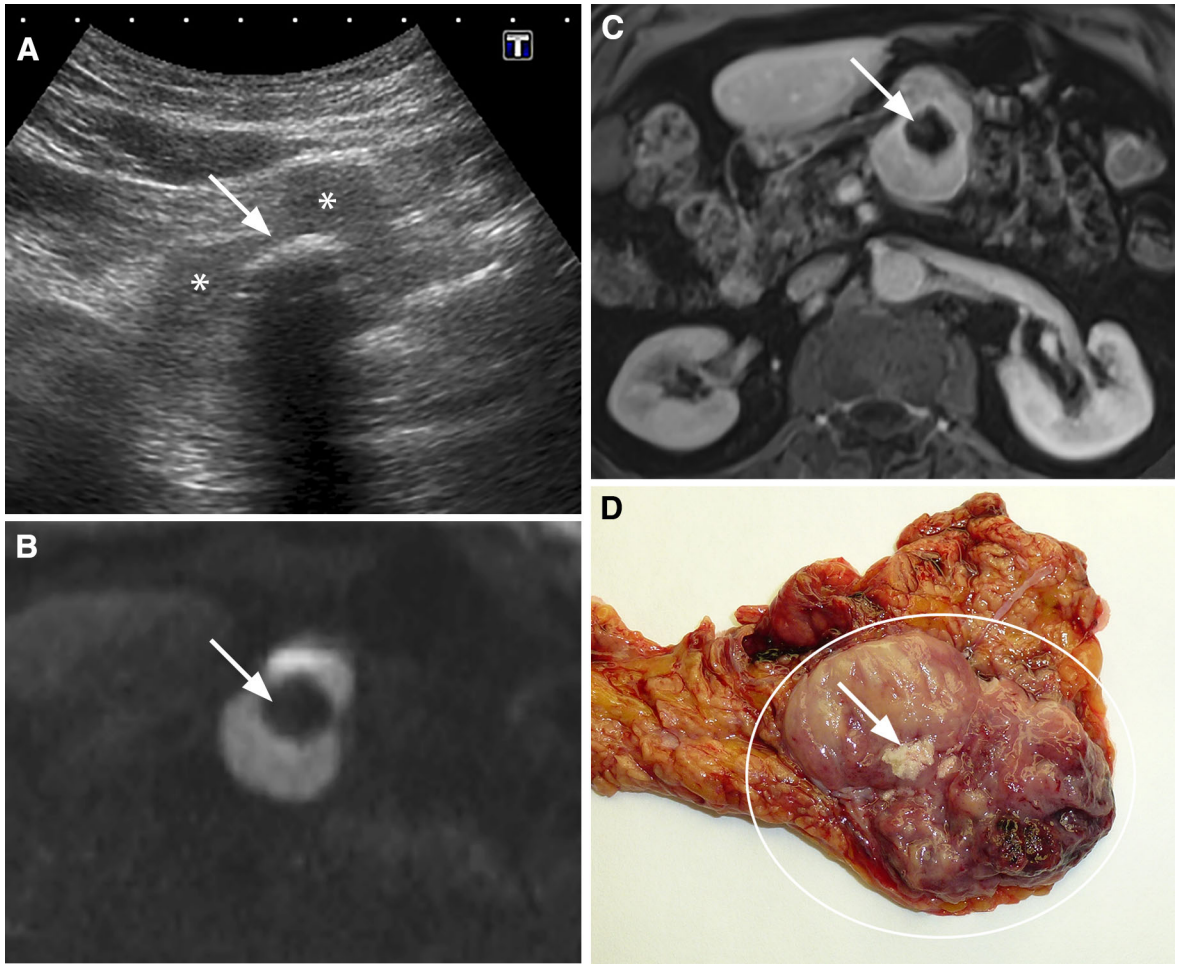

Fig. 11. Endocrine tumor with calcification. **A** Trans-abdominal B-mode ultrasound examination, transverse scan, showing a hyperechoic area with posterior shadow (calcification, *arrow*) within a slightly hypoechoic pancreatic body lesion (*asterisks*). **B** Diffusion-weighted image ($b = 800$), showing diffusion restriction of the lesion, except for the central hypointense area (calcification, *arrow*) **C** Pancreatic phase VIBE axial image, showing a hypervascular pancreatic lesion, with central hypointense area (calcification, *arrow*). **D** Resection specimen (transverse cut), showing the endocrine tumor (*circle*), with a central calcification (*arrow*).

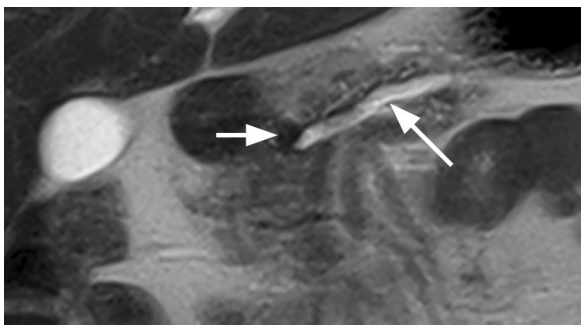

Fig. 12. Pancreatic carcinoid. Coronal T2-weighted TSE image, showing marked fibro-adipose involution of the pancreas; a small markedly hypointense solid lesion is visible in the pancreatic neck (*short arrow*) causing upstream dilation of the main pancreatic duct (*long arrow*).

hypo- or non-enhancing; internal septa may be hypervascular.

SCA does not communicate with the pancreatic ductal system and this can be well demonstrated at MRCP: this finding remains crucial for the differential diagnosis in respect to branch duct IPMNs [45–48].

Uncommon presentations

The atypical presentations of SCA are the macrocystic/unilocular aspect, the pseudosolid appearance and the presentation with huge dimension.

Macrocystic and unilocular. SCA may be macrocystic and unilocular at imaging, thus resembling a mucinous cystadenoma, which has a completely different management.

Table 2. Main common imaging aspects of cystic pancreatic tumors at imaging

	US	CT	MR	Contrast-enhanced imaging
SCA	"Honeycomb" aspect, thin wall, lobulated contours, ±central calcified scar	Hypodense, ±central calcified scar	"Honeycomb" aspect hyperintense on T2w-images, T2w-hypointense thin wall, septa, central scar, lobulated contours	Enhancing septa ± central scar
MCN	Rounded, complex content, thick wall, septa, ±parietal nodules, ±calcifications	Hypodense or slightly hyperdense content, ±peripheral calcifications	Hypointense or slightly hyperintense T1w content, thick wall, septa/nodules	Enhancing wall/septa/nodules
IPMN	Cystic dilation of pancreatic ductal system	Hypodense cystic areas with involvement or communication with ductal system	T2w hyperintense cystic areas with involvement or communication with ductal system	Enhancing solid portions
SPT	Round with heterogeneous content, thick capsule	Heterogeneous content, ±calcifications	T1w hyperintense content	Heterogeneous enhancement of solid components

US, ultrasound; CT, computed tomography; MR, magnetic resonance; SCA, serous cystadenoma; MCN, mucinous cystic neoplasm; IPMN, intraductal papillary mucinous neoplasm; SPT, solid pseudopapillary tumor

Fig. 13. Macrocystic unilocular serous cystadenoma. **A** Axial T2-weighted TSE fat-suppressed image, showing a lobulated homogenously hyperintense cystic lesion in the pancreatic tail. **B** Pancreatic phase VIBE axial image, showing no enhancing portions within the lesion. At resection, final diagnosis was macrocystic serous cystadenoma.

Macrocystic SCA is macroscopically composed of few but numerable cysts, usually larger than 2 cm in diameter, which can reach 15–20 cm. The macroscopically visible cysts sometimes are separated by broad septa without a central stellate scar.

When unilocular, the lesion may resemble a pseudocyst or a mucinous cystadenoma (Fig. 13). Due to the absence of a central scar, the frequent large size and the ill-defined tumoral border, radiologic distinction from mucinous neoplasms or pseudocysts can be difficult [14]; moreover, in about 40% of cases the epithelial lining may be lost, making difficult even the histologic differentiation with a pseudocyst: a clinical history negative for acute pancreatitis favors the neoplastic hypothesis.

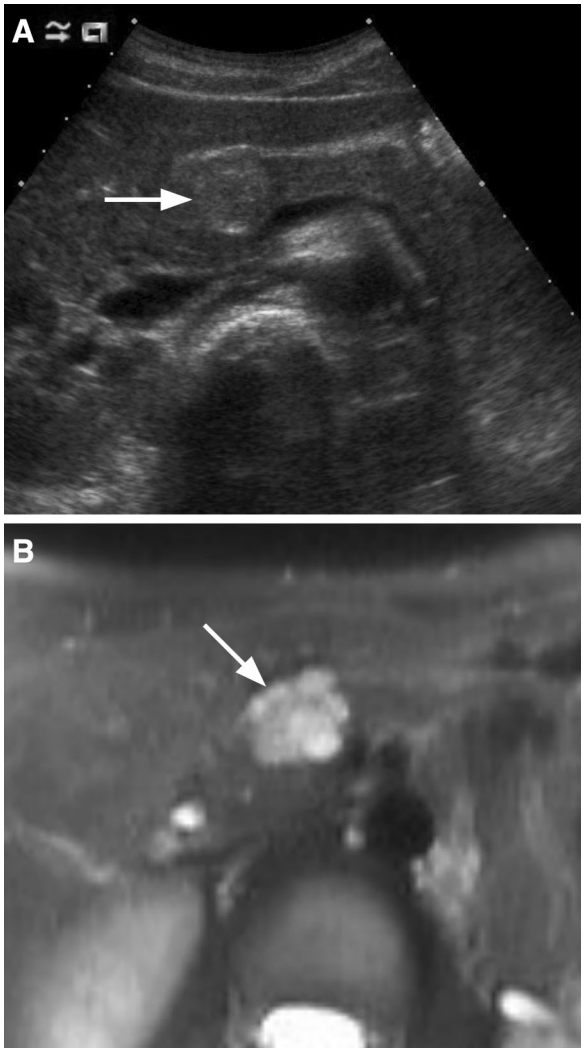

Fig. 14. Pseudosolid serous cystadenoma. **A** Trans-abdominal B-mode ultrasound examination, transverse scan, displaying a solid rounded, well-defined hyperechoic lesion (*arrow*). **B** Axial T2-weighted TSE fat-suppressed image, showing the true microcystic nature of the lesion (*arrow*). Final imaging diagnosis was microcystic serous cystadenoma.

Pseudosolid. Extremely microcystic SCA may mimic a solid lesion both at US and contrast-enhanced US, appearing as a slightly hyperechoic, hyperenhancing lesion owing to the presence of extremely compact multiple thin septa, thus resembling a neuroendocrine tumor (Fig. 14).

MRI can solve the problem, as the lesion is cystic in nature, albeit extremely microcystic, and typically appears hyperintense on T2-weighted images. Moreover, the confirmation of lack of communication with the main pancreatic duct allows a noninvasive differential diagnosis between SCA and small branch-duct IPMNs [29, 49–52].

Huge dimensions. SCA may be of huge dimensions at diagnosis (Fig. 15).

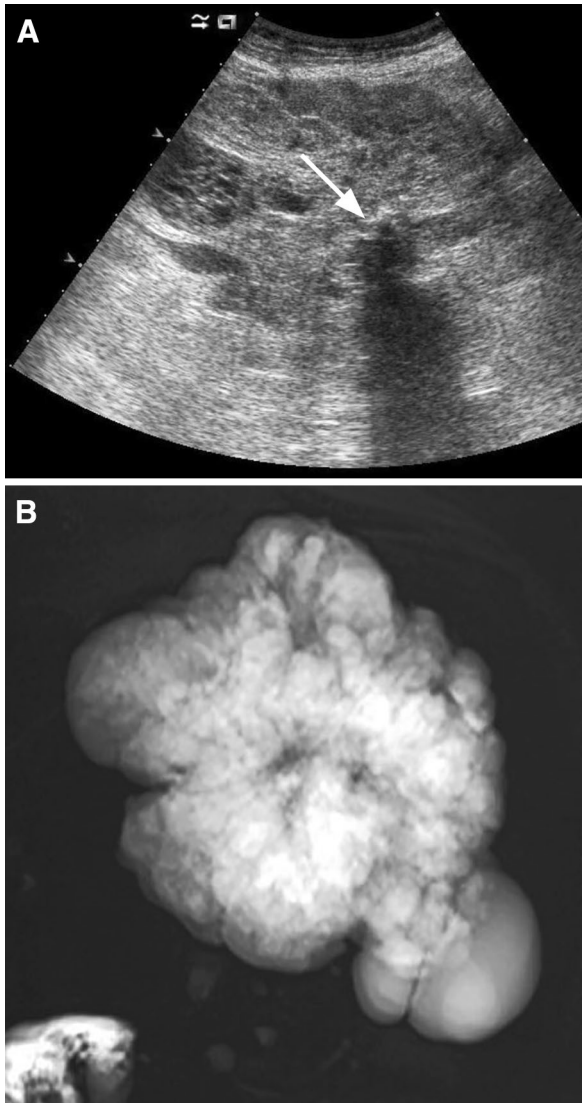

Fig. 15. Huge serous cystadenoma. **A** Trans-abdominal B-mode ultrasound examination, transverse scan, displaying a big ill-defined inhomogeneously hyperechoic lesion, with small cystic areas and a central calcification (*arrow*). **B** Transverse MRCP image, showing the true microcystic nature of the lesion. Final imaging diagnosis was microcystic serous cystadenoma.

SCAs are slow-growing tumors [53], and are generally incidentally found during abdominal studies as quite small lesions. In some instances, they can grow up to significant dimensions, causing abdominal compression-related symptoms; this represents one of the few surgical indications for SCA.

Mucinous cystic neoplasms

MCNs are pancreatic cystic tumors with different degrees of malignancy. They show clear female sex predilection and usually appear as a single lesion with a

rounded "ball-like" morphology, usually located in the body-tail of the pancreas probably due to its close position in respect to the rudimental ovary, and without communication with the pancreatic ductal system [54].

Common imaging findings

Common imaging features of MCNs are summarized in Table 2.

Mucinous cystadenoma (MCA) usually presents as a macrocystic lesion, with irregular septa, thick walls and complex content that can be corpuscolated, viscous and dense mainly owing to mucinous content. This content makes very often the lesion heterogeneously hypoechoic at US, hypodense at CT and slightly hyperintense on T2-weighted images. On T1-weighted images, the signal intensity can vary from hypointensity, more common, to hyperintensity depending on mucin concentration. MRCP clearly demonstrates the lack of communication with the pancreatic ductal system.

Differing from SCA, on post-contrast images, the intralesional septa are disorganized and peripherally located, describing a "bridge" along the cystic wall with a "pseudonodular" appearance. Peripheral calcifications along the thick wall can be detected, especially at CT [31].

Uncommon presentations

The atypical presentations of MCNs can be related to uncommon site and gender and the presence of intralesional disepithelized walls.

Uncommon site and gender. MCNs may rarely arise in the pancreatic head or may rarely develop in males.

As previously reported, pancreatic body-tail is the most common site for MCNs, maybe as a consequence of the closeness of these regions to the rudimental ovary.

Due to the possible presence of ovarian cells heterotopia MCNs can also arise in different sites, as the pancreatic head in up to 7% of patients (Fig. 16) [54–56].

Zamboni et al. [53, 55] hypothesized that MCNs arise either as the consequence of the endodermal immature stroma exposure to female sex hormones or as the consequence of ectopic primary yolk cells. In male patients, however, female hormones or primary lutein cells have theoretically no chance of affecting the development of ovarian stroma in MCNs: one possibility is that ovarian stroma is a secondary change in the growth of the tumor [57, 58].

Moreover, Regi et al. [57] found that hormonal and/or sexual dysfunction was present in the majority of patients affected by uncommon hormone-related pancreatic tumors; in their series, 16/188 MCNs were found in males. Wilentz et al. [58] reported that 18 of 61 patients (29.5%) were men, and also Borgne [59] reported a relatively high prevalence of male patients (50/228, 21.9%).

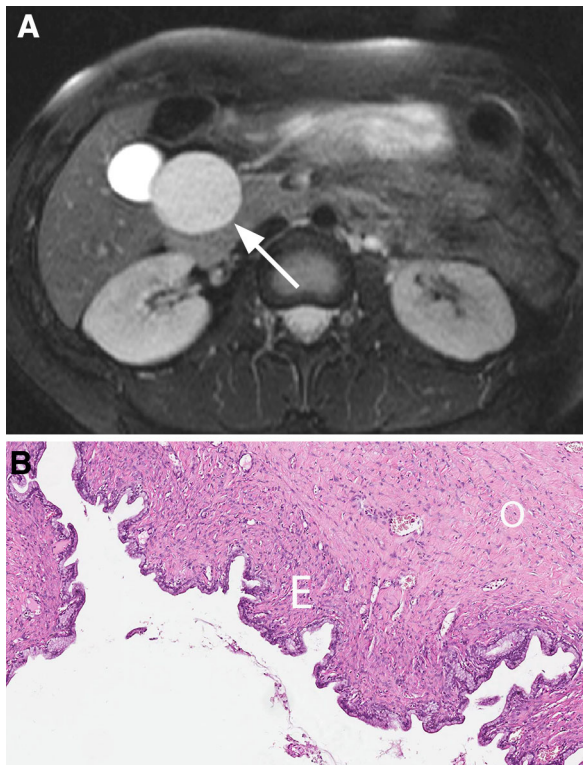

Fig. 16. Mucinous cystic neoplasm in the pancreatic head. **A** T2-weighted fat-sat TSE image, showing a rounded, well defined hyperintense cystic exophytic lesion of the pancreatic head (*arrow*). **B** H&E stain showing tall, columnar mucin-producing cells (E) with distinctive subepithelial ovarian-like stroma (O).

Disepithelized walls. As sometimes happens in SCAs, the epithelial lining within huge MCNs may be lost.

As a consequence, disepithelized MCNs become lined only by a fibrous capsule, making difficult the distinction with pseudocysts, not only at imaging; again, the absence of a clinical history of acute pancreatitis favors the neoplastic hypothesis [14].

Intraductal papillary mucinous neoplasms

IPMNs are exocrine mucin-producing neoplasms. Three types of IPMNs have been described: the branch duct type (BD-IPMN), the main duct type (MD-IPMN) and the mixed type (Mix-IPMN). They have different degrees of malignancy, but there are some imaging findings, as segmental or diffuse involvement of the main pancreatic duct, that can help in identifying those lesions with higher risk of malignancy.

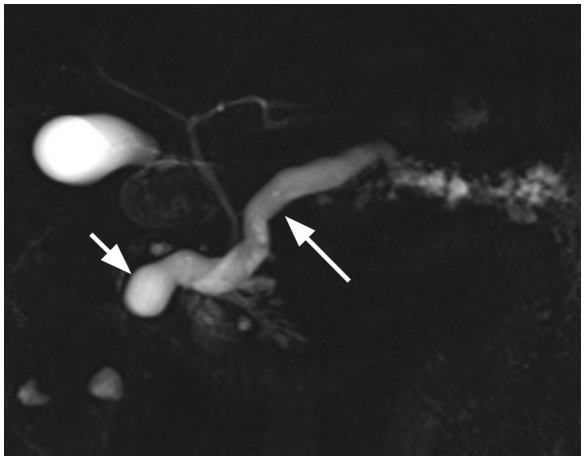

Fig. 17. Intraductal papillary mucinous neoplasm (IPMN) with pan-ductal-ectasia. Para-coronal MRCP image showing a pancreas divisum, with marked dilation of the entire dorsal pancreatic duct (*long arrow*) associated with saccular dilation of the ampullary portion (Santorinicele, *short arrow*).

Common imaging findings

Common imaging features of IPMNs are summarized in Table 2.

The demonstration of the communication with the ductal system is needed for an appropriate diagnosis; MRI with MRCP is the imaging modality of choice for the noninvasive diagnosis of IPMNs.

At imaging, one of the most indicative feature of malignancy is the presence of solid enhancing component appearing as mural nodules or septa [60, 61].

The management of IPMNs is mainly based on the location (MD- and Mix-IPMN vs. BD-IPMN), on the size of the largest cyst in BD-IPMN and on the presence of "high-risk stigmata" or "worrisome features". Moreover, risk stratification for age and fitness for surgery must be considered.

International consensus guidelines [62] recommend resection in presence of "high-risk stigmata" (main duct diameter >10 mm for MD- and Mix-IPMNs and presence of solid enhancing nodules within the cyst in BD-IPMN, or obstructive jaundice in presence of a cystic lesion of the pancreatic head), while in presence of "worrisome features" (cyst >3 cm, thickened cyst walls, MPD size of 5–9 mm, non-enhancing mural nodules, abrupt change in the main pancreatic duct (MPD) caliber with distal pancreatic atrophy and lymphadenopathies) the lesion should be evaluated by means of EUS. Patients with BD-IPMN, with cysts >3 cm and no "worrisome features" can also be considered for EUS to verify the absence of thickened walls or mural nodules, particularly if the patient is elderly, while if patient is young and fit for surgery, surgery should be strongly considered. BD-IPMN with cysts <3 cm and no "worrisome features" should be considered for observation [62]. MD-IPMN with MPD dilation of 5–9 mm can also be considered for further evaluation, but no immediate resection [62].

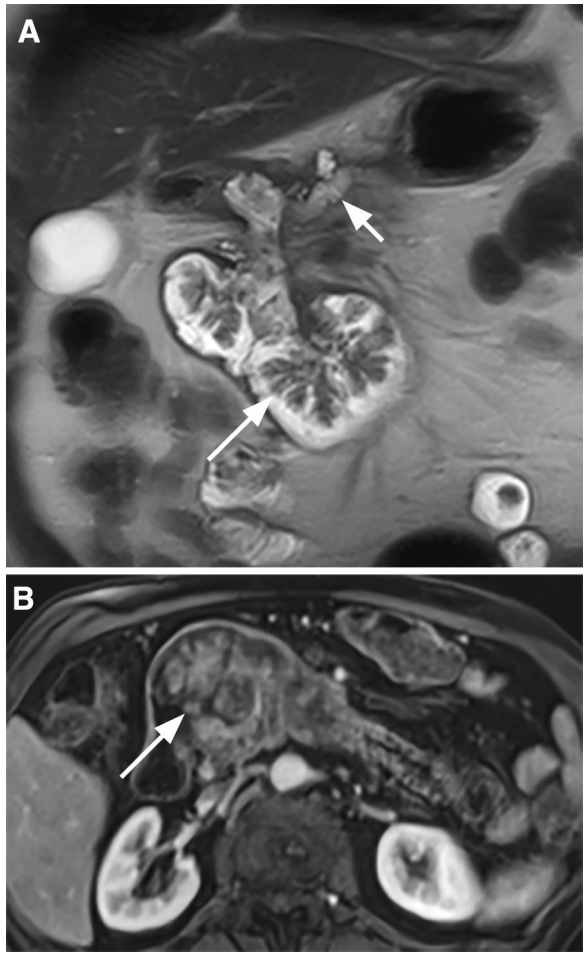

Fig. 18. Colonizing and multifocal mixed intraductal papillary mucinous neoplasm (IPMN). **A** Coronal T2-weighted TSE image, displaying the massive filling of the duodenal lumen by solid, hypointense "frond-like" projections (*long arrow*), with associated dilation of the main pancreatic duct (*short arrow*). **B** Pancreatic phase T1 GRE fat-sat 3D (VIBE) axial image, showing enhancement of the solid portions (*arrow*).

Uncommon presentations

The atypical presentations of IPMNs can be due to dilation of the entire ductal system resulting in a pan-ductal-ectasia or peculiar colonizing growth pattern of this tumor.

Pan-ductal-ectasia. IPMN may present with dilation of the entire ductal system (Fig. 17).

This rare appearance makes difficult the differential diagnosis between a Mix- or a MD-IPMN and a duct dilation secondary to mucin production from an IPMN with Wirsung's duct overfilling.

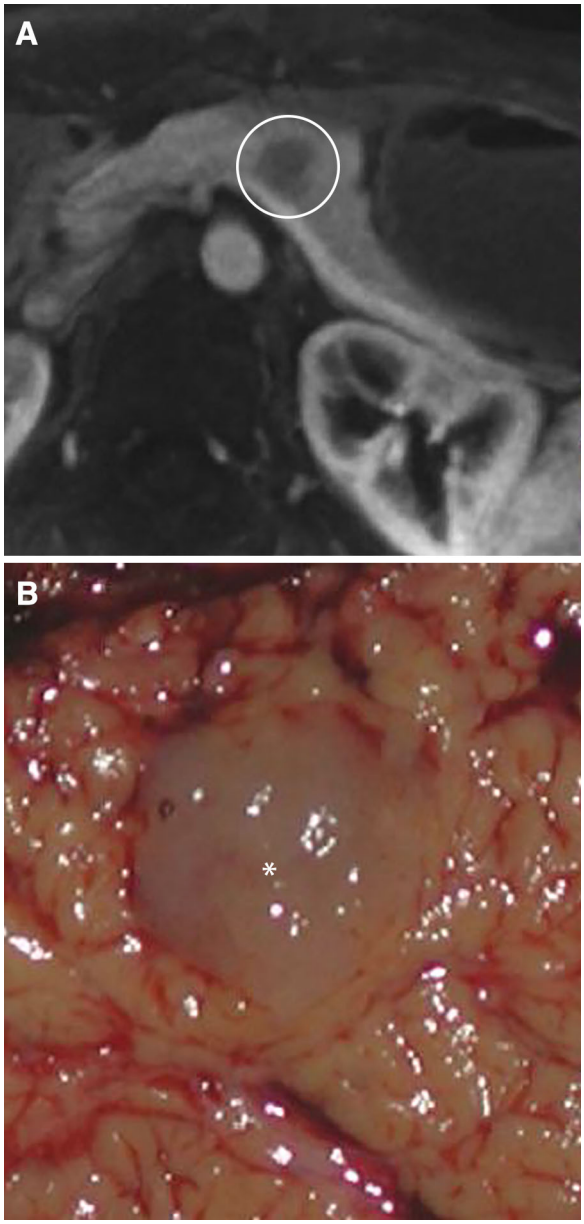

Fig. 19. Small solid pseudopapillary tumor. **A** Pancreatic phase axial CT image showing a small hypovascular solid rounded lesion in the pancreatic body-tail (*circle*), without upstream dilation of the main pancreatic duct. **B** Resection specimen (longitudinal cut) showing a small solid pseudopapillary tumor (*asterisk*) with expansive growth.

Colonizing growth. IPMN may invade adjacent structures (Fig. 18) due to the exposure of the neoplastic epithelium to high pressure produced by mucin, resulting in perforation and fistulization mainly into the duodenum, stomach and choledochus. Autodigestion by enzyme-rich fluids may play an important role for direct invasion and fistulization of IPMN into other organs.

Infiltration usually has the histological appearance of a colloid carcinoma with gelatinous stromal masses. This explains, in advanced/bigger tumors, the presence of fistula with the common bile duct and extrapapillary duodenum with solid colonization, or the peritoneal diffusion as pseudomyxoma peritonei even if exceedingly rare [14, 63].

Solid-pseudopapillary neoplasms

Solid-pseudopapillary tumors (SPTs) are epithelial neoplasms with low malignant potential, predominantly occurring among young women. They usually develop as solid tumors and then undergo massive hemorragic/necrotic degeneration, giving rise to a large mass with cystic appearance [64–66].

Common imaging findings

Common imaging features of SPTs are summarized in Table 2.

At diagnosis, SPTs are often quite large, with a mean size between 7.5 and 11 cm [65]. Cystic areas often present hemorragic content, usually well depicted at CT and, even better, at MRI as respectively hyperdense or T1-weighted hyperintense areas.

At dynamic imaging, homogeneous enhancement of the solid components and the peripheral thick wall is well demonstrated [66–68].

Uncommon presentations

An atypical presentation of SPTs is the small and solid pattern.

Small and solid. Solid-pseudopapillary tumors arise as small and solid lesions (Fig. 19).

Due to the improved possibilities of imaging there have been published several studies describing imaging findings of small (<3 cm) solid pseudopapillary neoplasms [68–70]. Unlike typical large pseudopapillary neoplasms, small SPTs, incidentally detected, appear less sharply circumscribed, often unencapsulated, and completely solid without hemorrhage, necrosis or cystic changes [70]. After contrast medium administration, the tumors usually demonstrate mild, gradually increasing enhancement [69]. These imaging features may overlap with those of ductal adenocarcinomas, except to the well-defined margins due to an expansive rather than infiltrative growing. Owing to this growth pattern, a peripheral pseudocapsule can develop, explaining the typical rim enhancement that can be seen around this lesion, and can be useful for differential diagnosis [71].

Conclusions

Common pancreatic neoplasms, both solid and cystic, may rarely present with atypical appearance; however,

their histopathological bases and imaging features must be known in order to avoid relevant misdiagnosis.

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