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Rare Solid Tumor of the Exocrine Pancreas: A Pictorial Review

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Numerous other solid primary neoplasms may arise from the pancreas besides primary ductal adenocarcinomas and neuroendocrine tumors. Although diagnosis can be difficult because of the very low incidence of these tumors, knowledge of several, typical, epidemiologic, biological, and imaging features can help obtain a correct diagnosis. This pictorial review describes the features of solid rare primary pancreatic neoplasms on computed tomography and magnetic resonance imaging focusing on characteristics that can help radiologists differentiate them from classical forms of ductal pancreatic adenocarcinoma and neuroendocrine tumors. Cystic pancreatic neoplasms are beyond the scope of the current review.
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Introduction

Many other solid primary neoplasms may arise from the pancreas besides primary ductal adenocarcinomas (PDA) and neuroendocrine tumors (NETs). This pictorial review is presented in 4 sections. The first section describes tumors that mimic PDA (and that share hypoenhancement on contrast-enhanced images), including acinar cell carcinoma, rare variants of PDA, and primary pancreatic lymphoma. The second part presents tumors that mimic NET (characterized by arterial phase hyperenhancement), including solid pseudopapillary neoplasms (SPNs), "solid" forms of serous cystadenomas, and pancreatic extragastrointestinal stromal tumors (EGIST). Pancreatic metastases can mimic both PDA and NET depending on the primary tumor and are also described in this section. The third section focuses on desmoid and solitary fibrous tumors that are characterized by a fibrotic stroma. Finally, the fourth section will discuss miscellaneous and rare solid pancreatic tumors. Our description of solid pancreatic neoplasms focuses on imaging characteristics that can help radiologists differentiate these entities from PDA and NET. Cystic pancreatic neoplasms are beyond the scope of the current review, and are not discussed.

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Tumors Mimicking Pancreatic Ductal Adenocarcinoma

Classic pancreatic ductal adenocarcinomas are seen as hypoenhanced, ill-defined, infiltrative tumors with vascular, and ductal encasement. Thus, the presence of hypoenhanced focal pancreatic lesions frequently leads to a diagnosis of PDA. Nevertheless, other neoplasms and rare histologic variants of PDA can share these features, and it is extremely important to differentiate these entities from classic PDA because of the therapeutic consequences for patients.

Acinar Cell Carcinoma

Acinar cell carcinomas (ACCs) represent no more than 1% of all pancreatic tumors. ACCs originate from the exocrine pancreas and usually occur earlier than PDA (reported mean age 56 and 70 years, respectively).[1] More than 50% of patients with ACC present with metastatic disease while 25% present with locoregional invasion.

Symptoms are nonspecific and are mostly related to the tumor mass. These include fever, abdominal pain, weight loss, and jaundice. A paraneoplastic presentation may occasionally be found, suggesting the diagnosis, and characterized by lipase hypersecretion,[2] fat necrosis, polyarthralgia, subcutaneous nodules, and eosinophilia. Increased alfa-fetoprotein serum levels may be present, especially in younger patients.[3]

ACC is more slow growing than PDA with a reported overall 5-year survival of 42.8% (vs 3.8% for PDA).[1] When possible, surgery is the best therapeutic option with a 5-year survival rate of between 43.9% and 72% for resected ACC.[1,4]

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Table 1 Characteristics of Acinar Cell Carcinoma

	Tatli[7] *N* = 11	Chiou[5] *N* = 10	Bhosale[3] *N* = 30	Hsu[6] *N* = 6
Age (years)	64 (44-79)	64 (49-75)	58 (23-85)	61 (41-71)
Gender Male/female	5/6	7/3	21/9	5/1
Size (cm)	6 (2-15)	7.2 (3.3-18)	7 (2.6–19)	6.1 (3.5-12)
Location	*H* = 5, *B* = 2, *T* = 4	*H* = 6, *B* = 1, *T* = 3	*H* = 16, *B* = 0, *T* = 14	*H* = 4, *B* = 1, *T* = 1
Capsule	N.A.	6 (60%)	N.A.	4 (67%)
Well circumscribed	10 (91%)	8 (80%)	22 (73%)	4 (67%)
Exophytic	9 (82%)	N.A	N.A.	N.A.
Biliary dilatation	2 (18%)	N.A	5 (17%)	3 (50%)
Pancreatic duct dilatation	3 (27%)	N.A	9 (30%)	1 (16%)
Vascular encasement	2 (18%)	5 (50%)	10 (33%)	1 (16%)
Intravascular tumoral thrombus	N.A.	2 (20%)	1 (3%)	N.A.
Hypovascular appearance	11 (100%)	8 (80%)	14 (47%)	6 (100%)
Calcification	3 (27%)	5 (50%)	2 (7%)	1 (16%)
Cystic/Necrosis	6 (55%)	8 (80%)	7 (23%)	5 (83%)
Hemorrhage	1 (9%)	0 (0%)	N.A.	2 (33%)
Lymph node	0 (0%)	4 (40%)	15 (50%)	3 (50%)
Metastasis	1 (9%)	3 (30%)	20 (66%)	3 (50%)
Increased serum alfa-fetoprotein	N.A.	2 (20%)	4 (13%)	1 (16%)

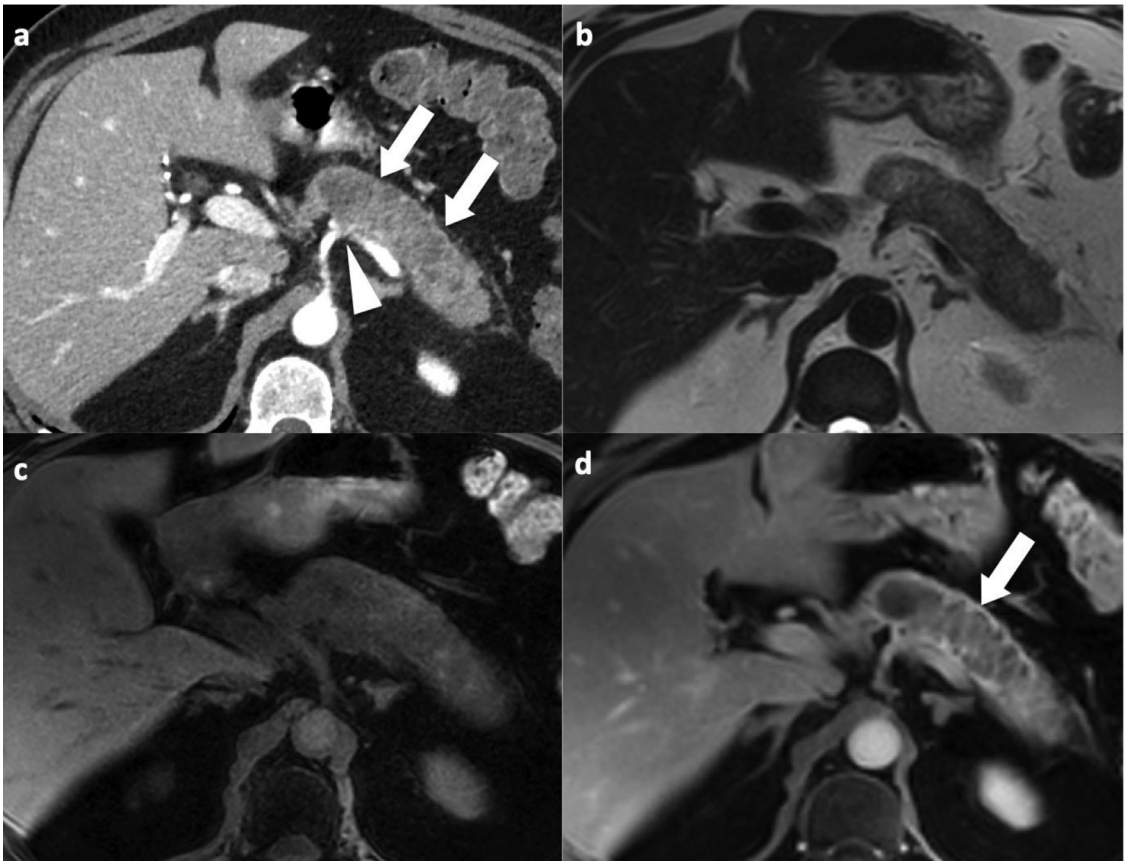

Figure 1 Acinar cell carcinoma in a 57-year-old male patient. Axial CT image obtained during arterial phase (a) shows a well-defined hypoattenuating mass (arrows) in the body and the tail of the pancreas. There is no peripancreatic fat stranding. The splenic artery is compressed by the tumor (arrowhead). The lesion shows mild T2 hyperintensity (b), heterogeneous hypointensity on a precontrast fat-suppressed T1-weighted GRE MR image (c) and is well delineated and hypointense with an enhancing capsule (arrow—d) on a fat-suppressed T1-weighted GRE MR image obtained during the portal venous phase (d).

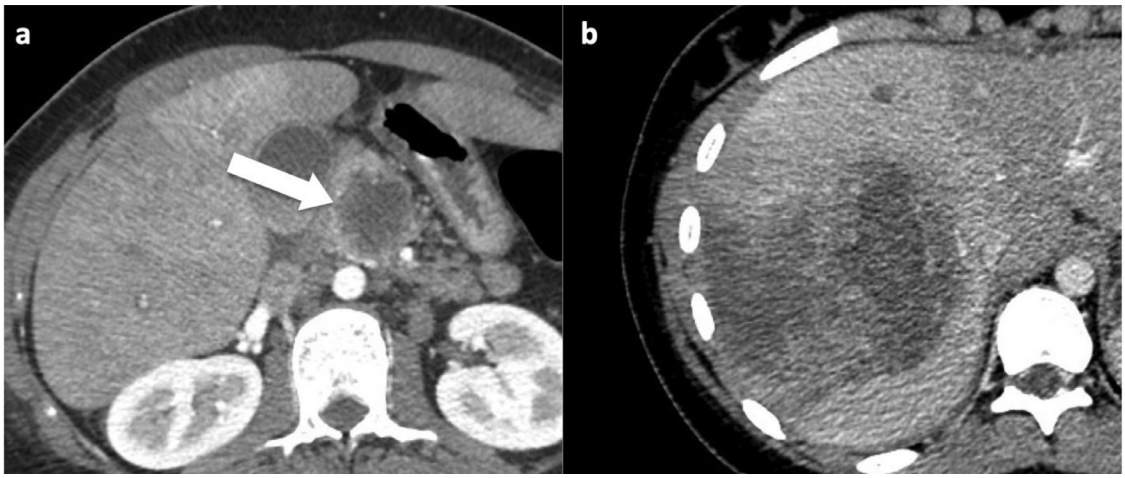

Figure 2 Anaplastic pancreatic adenocarcinoma in a 17-year-old female patient. Axial enhanced CT image obtained during the arterial phase (a) shows a large necrotic mass of the pancreatic body, presenting with predominant peripheral rim enhancement (arrow). Note the large metastasis of the right liver lobe (b) at the time of diagnosis. Biopsy confirmed an anaplastic type of pancreatic adenocarcinoma.

Imaging Features

In most cases ACCs appear as solid, well-circumscribed lesions[3] that are frequently located in the pancreatic head. The tumor is often exophytic. Enhancement tends to be homogeneous and lower than that of the surrounding pancreatic parenchyma, resulting in a hypoattenuating/-intense lesion during pancreatic arterial and portal venous phases on both computed tomography (CT) and magnetic resonance (MR) imaging. An enhancing capsule can be observed in more than 50% of the cases.[5,6]

Cystic portions, necrotic changes, and calcifications are common and have been reported in most published series.[3,5-7]

Although the lesion is large (usually >6 cm at diagnosis), and often located in the head of the pancreas, upstream dilatation of the main pancreatic duct is present in less than one third of patients (Table 1). There are very few published series describing the imaging features of ACC. The most relevant are summarized in Table 1. An example of ACC is presented in Fig. 1.

Thus, a diagnosis of ACC should be suggested in middle-aged men, presenting with large, well-defined, hypoenhanced tumors of the pancreatic head, without upstream ductal dilatation, and with limited or absent peritumoral invasion. Increased serum lipase or alfa-fetoprotein may be additional key features.

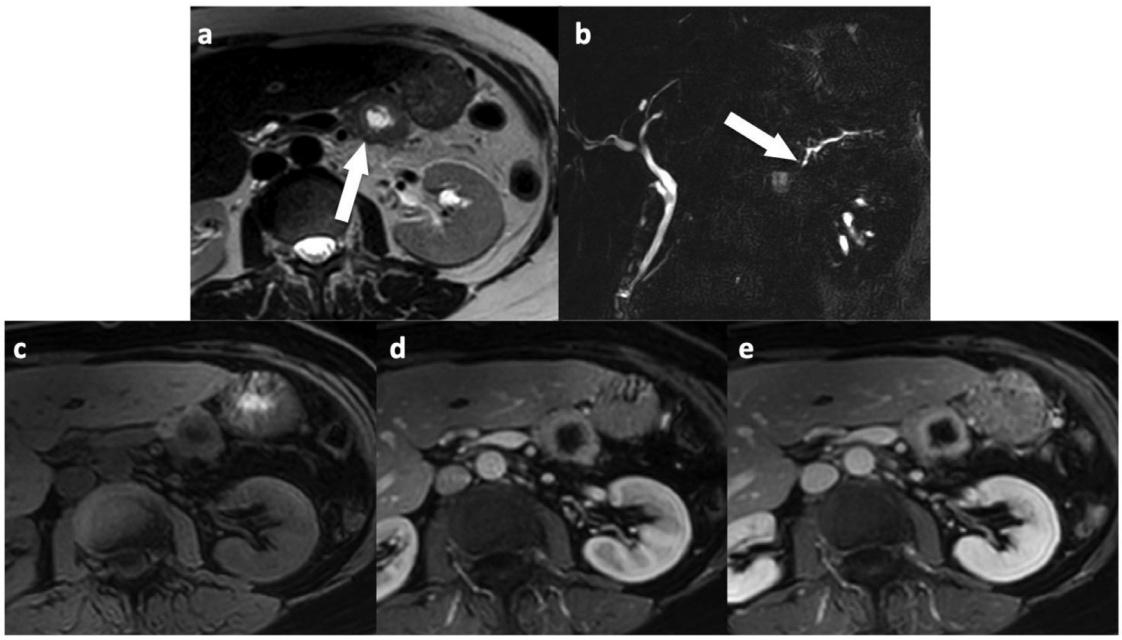

Figure 3 Adenosquamous adenocarcinoma in a 68-year-old female patient. Axial T2-weighted image (a) shows a lesion (arrow) located in the pancreatic body with central hyperintense portion. MR cholangiopancreatography (b) shows the upstream dilatation of the main pancreatic duct (arrow). The lesion is hypointense on precontrast (c) fat-suppressed T1 GRE image and shows a ring-like enhancing appearance on gadolinium-enhanced (d and e) fat-suppressed T1 GRE images obtained during portal venous (d) and delayed (3′) phase (e). Diagnosis of adenosquamous adenocarcinoma was confirmed after resection.

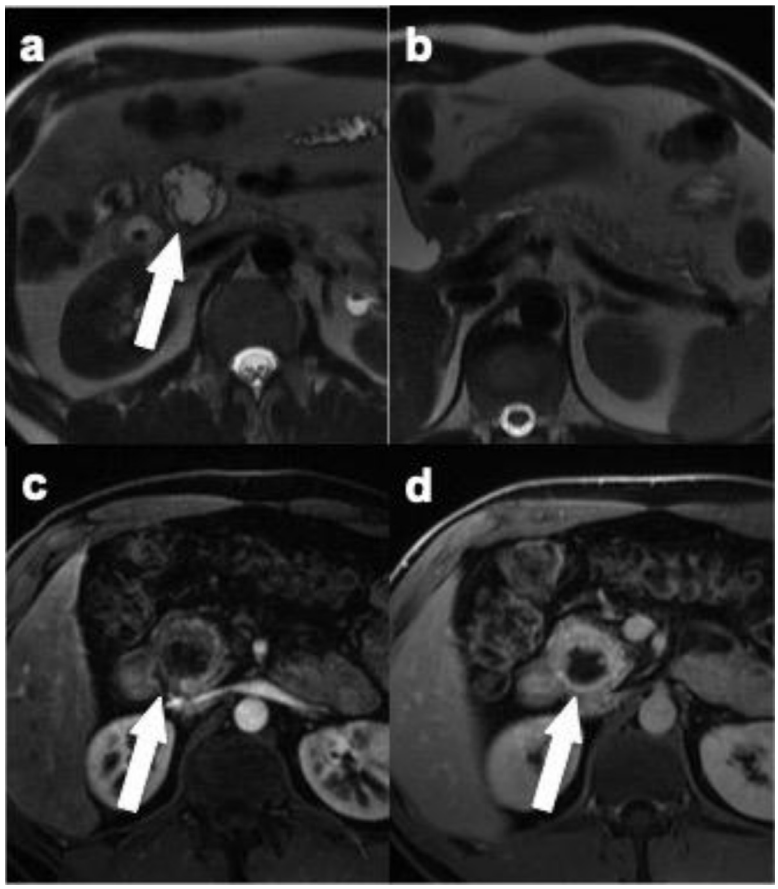

Figure 4 Colloid pancreatic adenocarcinoma in a 36 year-old male. Axial T2-weighted single-shot MR images (a, b) show a lesion (arrow) located in the pancreatic head, presenting with a heterogeneous bright signal, without upstream ductal dilatation (b). The lesion shows heterogeneous enhancement on a fat-suppressed T1-weighted GRE MR image obtained during the arterial phase (c) with progressive appearance during the late venous phase (d). Colloid adenocarcinoma was confirmed after resection.

Variants of Pancreatic Ductal Adenocarcinoma

Undifferentiated (anaplastic) carcinomas (including osteoclast-like giant cell tumors), adenosquamous carcinomas, and mucinous noncystic adenocarcinomas (also known as colloid carcinomas) are considered to be variants of ductal adenocarcinomas because, although they are poorly differentiated, most of these carcinomas contain some foci showing neoplastic glands with ductal differentiation.

Undifferentiated (Anaplastic) Carcinoma With Osteoclastic Giant Cell

The anaplastic form of pancreatic adenocarcinomas represents 2%-7% of pancreatic exocrine tumors.[8] The form presenting with osteoclastic giant cells accounts for less than 1% of all pancreatic malignant tumors.[9] Three different types of osteoclastic cell tumors (osteoclastic, pleomorphic, and mixed) have been regrouped by the World Health Organization into a unique entity, called "undifferentiated carcinoma with osteoclast-like giant cells."

The anaplastic form is usually larger than PDA at diagnosis (45 vs 30 mm), and is more evenly distributed in the pancreatic parenchyma (while classic PDA is frequently located in the pancreatic head).[10] Because of its rarity, there are very few series describing the imaging features of this tumor.[11,12] No specific radiological features have been identified to differentiate anaplastic forms from classic PDA. Intratumoral or peripheral enhancement is the most frequently reported finding on CT (Fig. 2), followed by the presence of cystic areas in the lesion.[13] Despite the poor prognosis, a matched cohort analysis including 192 patients with anaplastic pancreatic carcinomas showed that 5-year survival was not reduced compared to classic PDA (21.6% vs 17.4%) when resection was possible.[10] Moreover, 5-year survival was better in the osteoclastic giant cell form of the disease (50% vs 15%).

Adenosquamous Carcinoma

Adenosquamous carcinoma is a rare variant of PDA (3%-4%), which is defined on pathology by the presence of at least 30% of squamous differentiation in the lesion alongside classic glandular differentiation.[14]

The largest population-based study by Boyd et al including 415 patients showed that this tumor is more frequently located in the pancreatic body and tail (29.2% vs 19%,

respectively), and less frequently in the head (44.6% vs 53.5%, respectively) than classic PDA. It is often poorly differentiated (71.4%) and presents with metastatic lymph nodes (52.8%).[15] Adenosquamous carcinoma is aggressive with a poor outcome and a 2-year overall survival rate similar to classic PDA (11%). However, resected patients were reported to have reduced 2-year overall survival (29% vs 35.8%, respectively).[15]

The main radiological features include the presence of central necrosis with a ring-like enhancing peripheral lobulated portion[16] (Fig. 3). Hemorrhagic changes, calcifications, and a capsule are usually absent.[17] Upstream biliary and pancreatic duct dilatation is common. Importantly, and unlike classic PDA, this tumor may invade the portal vein.

Colloid Carcinoma

The colloid carcinoma, also known as "mucinous noncystic carcinoma," is a rare (1%) variant of ductal adenocarcinoma characterized by the presence of mucin in at least 50% of the tumor.[14] The main differential diagnoses for colloid carcinomas are mucinous cystic lesions (mucinous cystadenomas and intraductal papillary mucinous neoplasms).

The colloid form of pancreatic adenocarcinoma is characterized on imaging by a lobulated appearance with ill-defined margins. Due to the presence of mucin pools, this tumor is very bright on T2-weighted MR images with a "salt and pepper appearance" that is due to the presence of stroma and tumor cells in the pools[18] (Fig. 4). Yoon et al reported that the typical "peripheral and internal sponge-like or mesh-like progressive delayed contrast enhancement" in these tumors is due to enhancement of the stroma in the mucin pools, while the fibrotic enhancement pattern, characterized by persistent and delayed enhancement, is associated with the desmoplastic reaction induced by the tumor.[18,19]

Colloid carcinomas can be distinguished from intraductal papillary mucinous neoplasms by an absence of communication with the main pancreatic duct on MR cholangiopancreatography, a lack of upstream pancreatic ductal dilation and of papillary elements in the ductal system and an absence of papillary bulging into the duodenal lumen.[18,20]

Primary Pancreatic Lymphoma

The pancreas may be primarily or secondarily involved in a lymphoproliferative disorder. Diagnosis of primary pancreatic involvement includes the presence of a pancreatic mass with a normal leukocyte count, without hepatosplenic involvement or palpable enlargement of superficial or mediastinal lymph nodes.[21] The most common type of primary pancreatic involvement is represented by diffuse large B-cell lymphoma (80%).[8] This tumor usually develops between the ages of 50 and 60. Symptoms are nonspecific, and include abdominal pain, an abdominal mass and weight loss, nausea, jaundice, vomiting, diarrhea, pancreatitis, and bowel obstruction.

Primary lymphoma presents with 2 different patterns: a focal, well-circumscribed tumor, or a diffuse infiltrating form that may involve the entire pancreatic gland[22] (Fig. 5). Differentiating primary lymphoma from PDA (localized form) or pancreatitis (diffuse form) may be difficult because of the overlap of imaging findings.[23] Localized forms appear as a hypoattenuating mass on CT with or without moderate enhancement on the delayed phases. The tumor shows marked hypointensity on T1-weighted MR sequences and moderate hyperintensity on T2-weighted sequences. The enhancement pattern is similar to that observed on CT. The lesion can cause biliary and pancreatic ductal dilatation. Upstream pancreatic ductal dilatation is rare and usually mild, while biliary dilatation is more common in non-Hodgkin's lymphoma, with jaundice reported in up to 42% of patients.[22,24] Lack of or mild pancreatic ductal dilatation

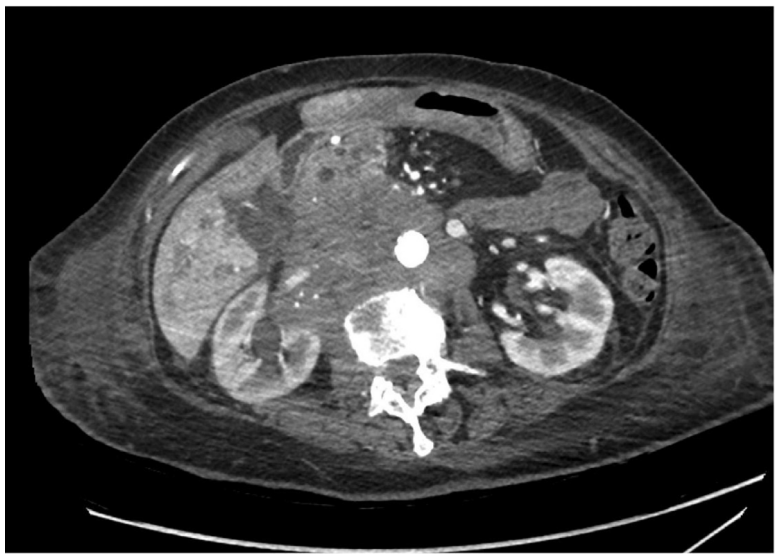

Figure 5 Primary pancreatic lymphoma in 82-year-old female patient. Axial contrast enhanced CT image shows a hypoattenuating infiltrative mass infiltrating the pancreatic head, the retroperitoneal right perirenal space and surrounding the abdominal aorta.

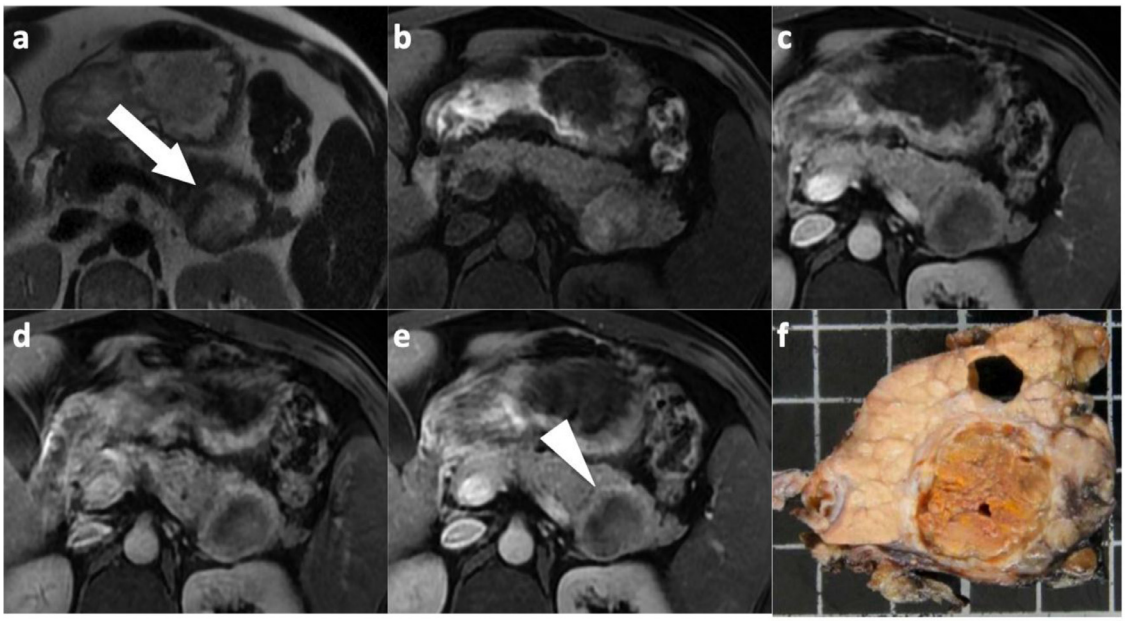

Figure 6 Solid pseudopapillary neoplasm in a 36-year-old female patient. Axial T2-weighted MR image (a) shows a large lesion (arrow) in the pancreatic tail with a heterogeneous central high T2 signal area including cystic portions. The lesion is hyperintense on precontrast (b) fat-suppressed T1-weighted GRE MR image, suggesting hemorrhagic content. Note the peripheral thick well-defined fibrous enhancing capsule (arrowhead), presenting with progressive enhancement on enhanced fat-suppressed T1 GRE MR images (c-e) obtained during portal (c), 3′ (d) and 5′ (e) delayed phase. Diagnosis of a solid papillary neoplasm was confirmed after surgery (f).

associated with mesenteric or retroperitoneal enlarged lymph nodes, may suggest a diagnosis of primary lymphoma. The absence of peripancreatic fluid collections or fat necrosis helps differentiate infiltrating lymphoma from acute pancreatitis. A heterogeneous appearance on enhanced CT and MR imaging as well as the absence of capsule-like rim enhancement can help exclude the diagnosis of autoimmune pancreatitis.[23] Histologic confirmation is essential, because surgical resection is not indicated in primary pancreatic lymphoma.

Mimickers of Pancreatic NETs

NETs are rare neoplasms arising from neuroectodermal cells. The 2017 World Health Organization classification defined pancreatic NETs according to tumor proliferation and differentiation. Low-grade tumors (ie, grades 1 and 2) are small, well-delineated tumors with a rich vascular network showing arterial phase hyperenhancement on imaging. Enhancement is variable during portal and delayed phases, but tumor washout is rare. A peripheral capsule, calcifications, and inner bleeding may be present. These tumors often result in limited locoregional invasion, with no upstream pancreatic ductal dilatation. Although visualization of a hyper-enhanced focal pancreatic lesion strongly suggests a diagnosis of NET, other neoplasms share these features.

Solid Pseudopapillary Neoplasms (SPN)

SPN, formerly known as a "Frantz tumor," represents about 1% of all primary pancreatic neoplasms. This tumor is usually found in young (20-30 years old) female patients (female to

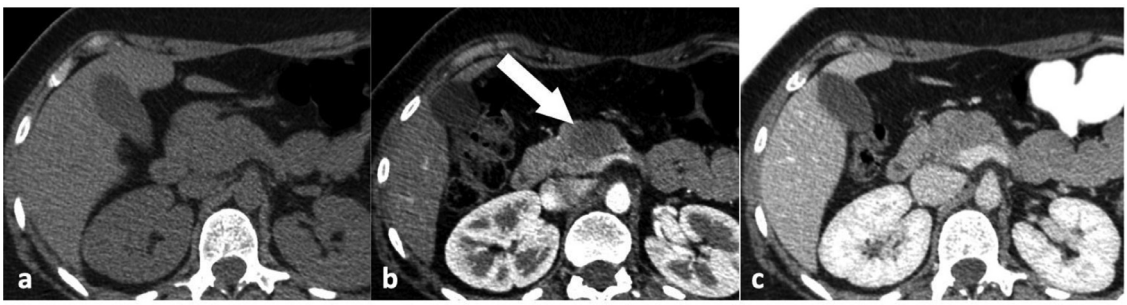

Figure 7 Small solid pseudopapillary neoplasm in a 29-year-old female patient. Precontrast (a) and contrast-enhanced (b, c) axial CT images obtained during the arterial (b) and portal venous phases (c) show a hypo-attenuating solid lesion in the pancreatic head (arrow). Note the progressive enhancement from arterial (b) to portal venous phase (c) and the absence of cystic component.

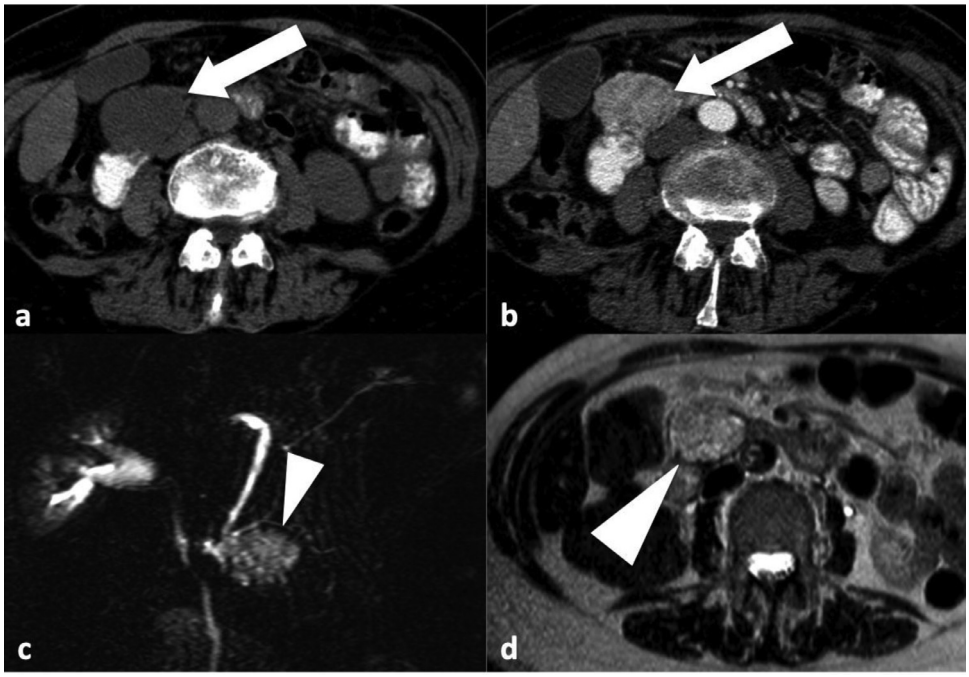

Figure 8 "Solid" variant of serous cystadenoma in a 62 year-old female. Precontrast (a) CT image shows a pancreatic lesion (arrow) with cystic attenuation. Enhanced (b) CT image obtained during the arterial phase shows marked and slightly heterogeneous enhancement of the lesion (b—arrow). MR cholangiopancreatography image (c) and T2-weighted MR image (d) confirmed the microcystic nature (arrowheads) of the tumor.

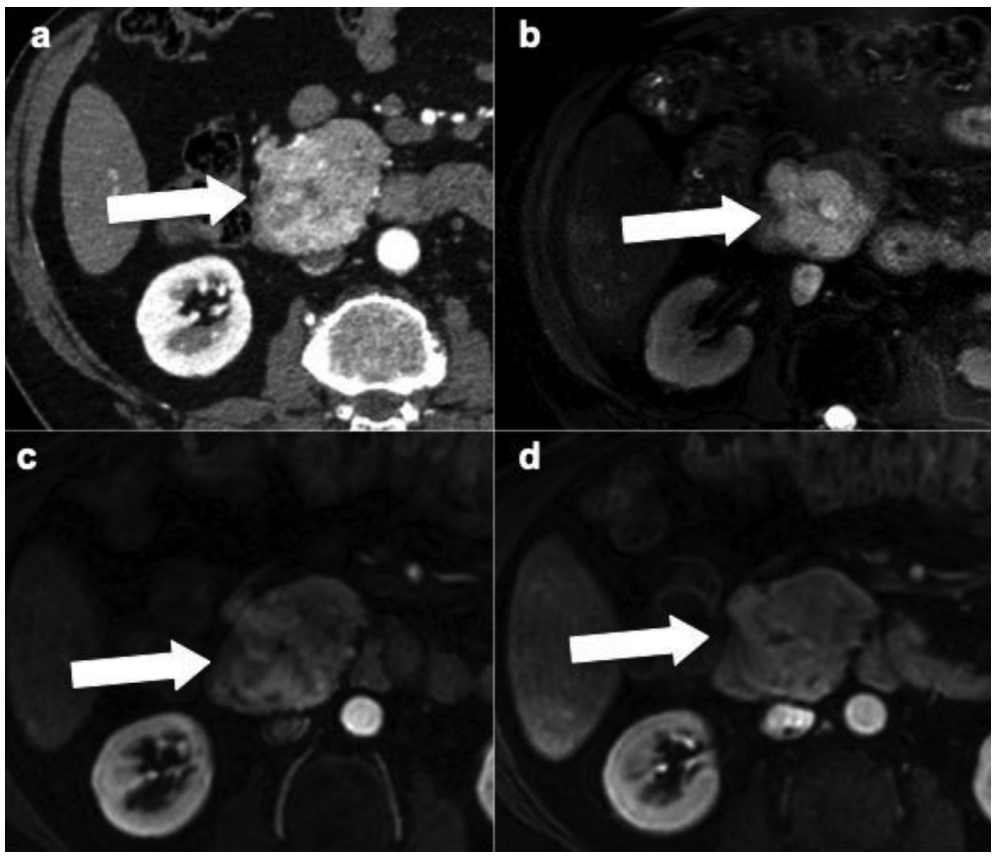

Figure 9 Pancreatic extra gastrointestinal stromal tumor (EGIST) in a 65-year-old male patient. Axial enhanced CT image (a) obtained during the arterial phase shows a hyperenhanced lesion (arrow) in the pancreatic head. The lesion is bright on fat-suppressed T2-weighted MR image (b) and shows enhancement on contrast-enhanced fat-suppressed T1-weighted MR image obtained during the arterial phase (c), which persists in the portal venous phase (d).

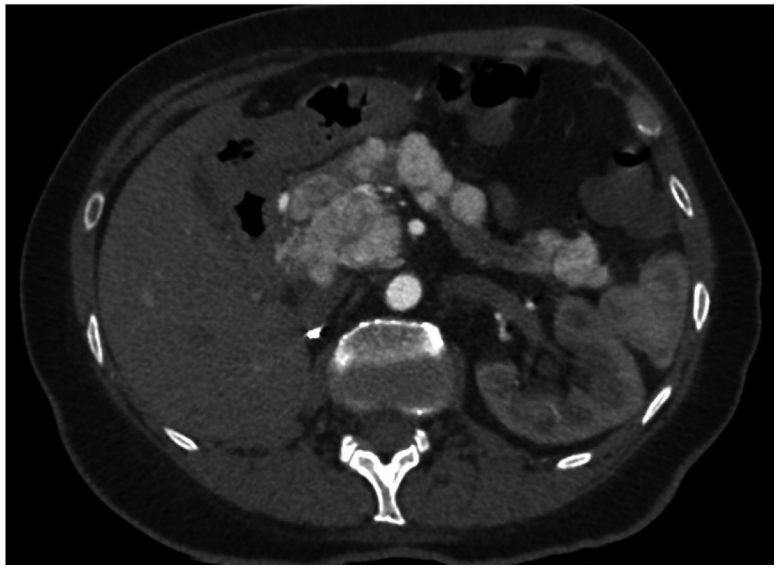

Figure 10 Pancreatic metastases from renal cell carcinoma in a 68-year-old female patient. Contrast-enhanced CT image obtained during arterial phase shows multiple hyperattenuating lesions within the pancreatic parenchyma consistent with pancreatic metastases. The patient had a history of right nephrectomy for renal cell carcinoma 5 years before.

male ratio 9:1).[25] SPNs are frequently benign but malignant characteristics have been reported in 10%-20% of cases including perivascular or pancreatic parenchymal invasion,[26,27] and metastatic disease (main metastatic sites including liver and lungs).[28-30] The clinical presentation is nonspecific, with about one third of patients presenting with abdominal pain[27] or other symptoms such as abdominal discomfort, vomiting, or back pain. In the other cases, patients are asymptomatic and the tumor is discovered incidentally during a routine radiological examination.

SPNs are large, round, and solitary masses. They are usually encapsulated and well separated from the pancreas. These neoplasms are mostly solid, and may contain areas of hemorrhage and necrosis as well as cystic spaces filled with necrotic debris. Occasionally, the hemorrhagic-cystic changes involve nearly the entire lesion and the neoplasm may be misdiagnosed as a pseudocyst. A thick and well-defined fibrous enhancing capsule is possible.[31,32] Fewer than 20% of tumors present with upstream pancreatic ductal dilatation and atrophy, a finding that is not related to lesion size.[31,33]

On CT, SPNs show early moderate heterogeneous enhancement during the arterial phase that usually increases (progressive appearance) during the portal phase. This enhancement is moderate and in most cases the tumor is hypoattenuated or

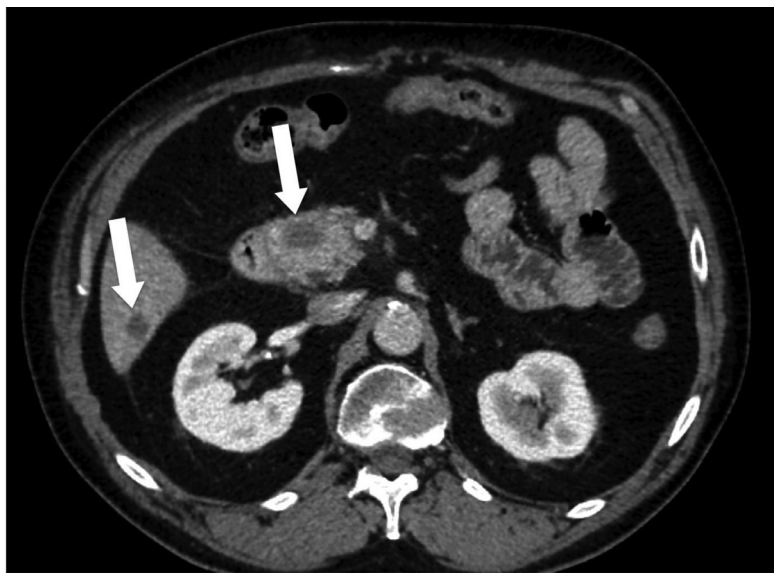

Figure 11 Pancreatic and hepatic metastases from primary lung adenocarcinoma in a 57-year-old male patient. Contrast-enhanced CT image obtained during portal venous phase shows two ill-defined hypoattenuating lesions (arrows) in the pancreatic head and in segment VI of the liver consistent with metastases.

more rarely, isoattenuated compared to the pancreatic parenchyma.[33,34] SPN may also contain calcifications that can be central, peripheral or septal.[34]

SPNs are characterized by a heterogeneous hyperintense signal on T2-weighted MR images with a homogeneous or heterogeneous hypointense signal on T1-weighted images. Enhancement is similar to CT.[31,35] Hemorrhagic components, present in about one third of patients, are seen as heterogeneous hyperattenuating and hyperintense foci on precontrast CT, and T1-weighted images, respectively (Fig. 6).

Park et al have identified different SPN features according to gender.[34] Male patients were older (42 vs 33 years) and presented with larger tumors (6.3 cm vs 4.6 cm).[34] Tumors in male patients were more frequently lobulated with more solid components. Completely solid tumors were described in 42% of the male patients in this cohort (vs 28% in female patients), while a completely cystic appearance was only found in 19% of female patients. The enhancement pattern was not different according to gender.[34,36]

Beak et al described CT features of small (<3 cm) SPN and showed that they were frequently pure solid tumors (Fig. 7) with sharp margins and early progressive enhancement.[33] The early heterogeneous and progressive enhancement pattern is helpful to distinguish small SPN from small NET,[35] which are more likely to have an early persistent homogeneous enhancement pattern.[35,37]

When SPN is suspected on imaging, biopsy should not be performed[38] to prevent the theoretical risk of abdominal dissemination due to tumor rupture. Thus, pancreatic resection should be performed without a preoperative histologic diagnosis.[38] Nevertheless, there are no reported cases of SPN dissemination following endoscopic-ultrasound-guided biopsy,

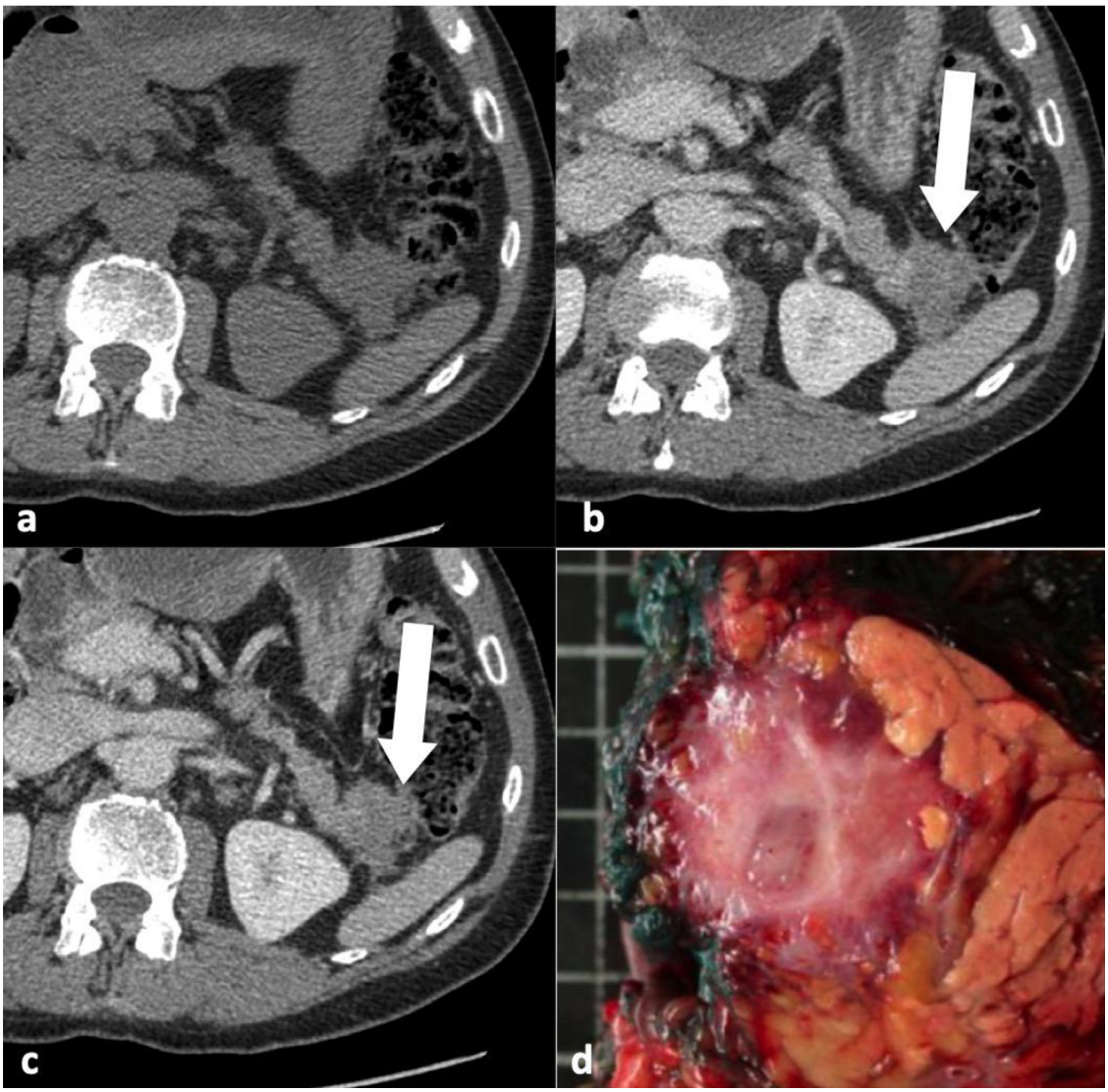

Figure 12 Desmoid tumor in a 67-year-old male patient with history of Hodgkin's lymphoma treated with chemotherapy. Precontrast (a) and contrast-enhanced (b, c) CT images show an infiltrative ill-defined lesion (arrow) of the pancreatic tail with progressive enhancement from the portal (b) to delayed 3′ phase (c). The lesion was resected confirming the diagnosis of desmoid tumor (d).

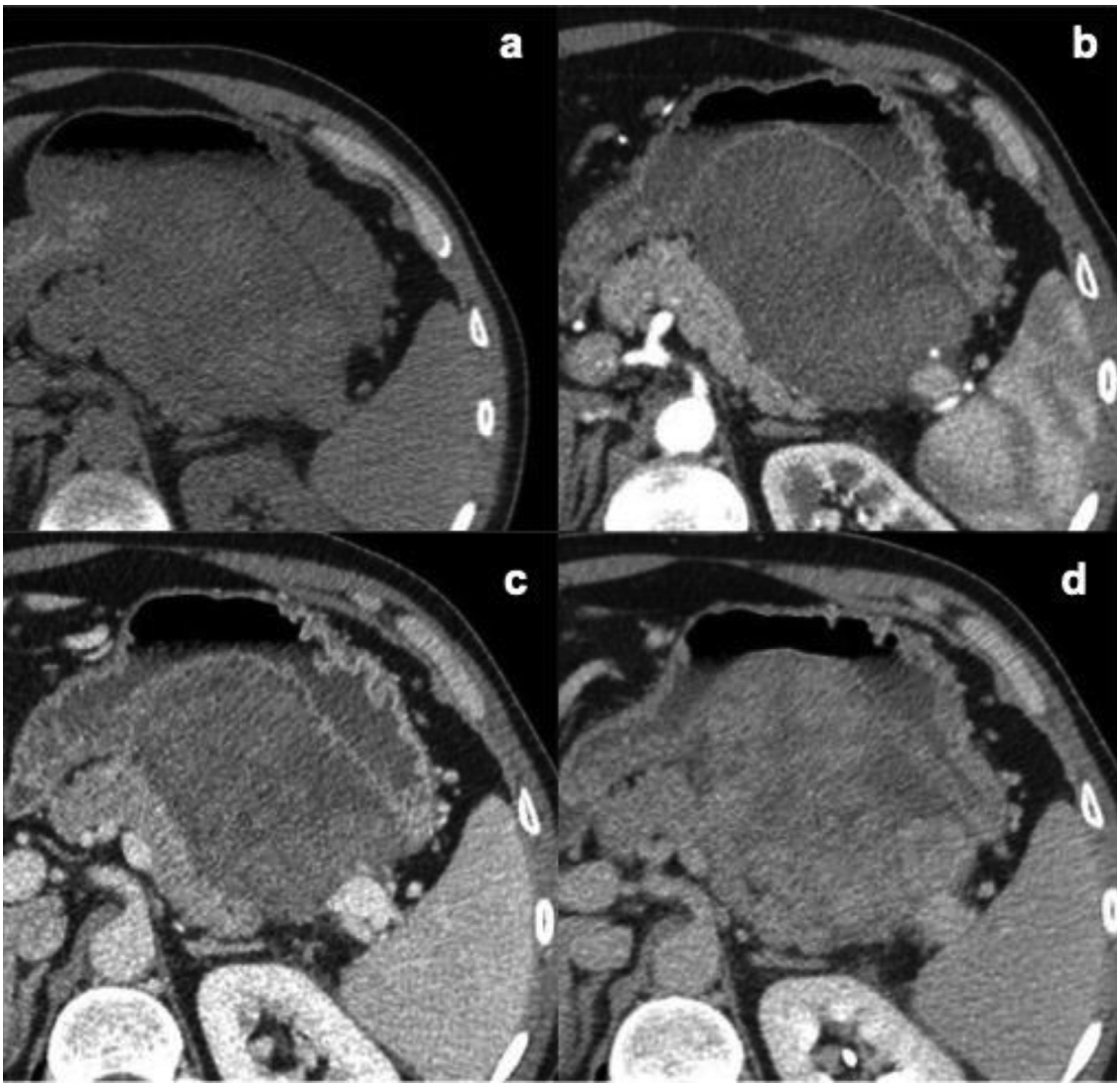

Figure 13 Solitary fibrous tumor in a 55-year-old male patient. Precontrast (a) and contrast-enhanced (b-d) CT images show a large well-delineated lesion of the pancreatic body, presenting with progressive enhancement from the arterial (b) to portal (c) and delayed 3′ (d) phases. The lesion was resected confirming the diagnosis of solitary fibrous tumor.

thus this technique may be considered in doubtful cases. The outcome of patients with SPN is good and disease-free survival after resection is excellent, with almost 100% of patients presenting without tumor recurrence at 5 years.[26]

"Solid" Forms of Serous Cystadenoma

This form is a rare variant of serous cystadenoma. The "solid" appearance on contrast-enhanced CT or MR is due to the avid enhancement of the stroma surrounding the small cysts of the tumor. Because the cysts are microscopic, lesions usually show homogeneous, marked enhancement following contrast administration which can lead to a misdiagnosis, especially on CT. Although the high signal intensity on T2-weighted MR images can mimic NETs, MR cholangiopancreatography clearly visualizes the multimicrocystic appearance[39,40] (Fig. 8). This entity is also confirmed by the high apparent diffusion coefficient value of the lesion.[41] This stresses the importance of using MR imaging to characterize focal pancreatic lesions, especially when they show arterial phase hyperenhancement.

Pancreatic Extragastrointestinal Stromal Tumor

GIST are mesenchymal tumors originating from the Cajal cells, located in the wall of the gastrointestinal tract. These tumors are characterized by mutations in the c-KIT proto-oncogene and are mainly located in the stomach, jejunum or ileum.[42] GISTs rarely arise outside the gastrointestinal tract, and in this case they are called EGIST. Pancreatic locations are extremely rare, and are found in 5% of all EGIST.[43,44] The literature on EGIST is scarce and controversial, and some authors suggest that these entities are GIST with an exophytic growth that has lost the connection with the gastrointestinal tract.[45] The diagnosis of this tumor may be incidental and symptoms are often nonspecific (abdominal pain, bleeding, anemia, weight loss, and obstruction).[46]

There are no published data on the imaging features of pancreatic EGIST, and the radiological appearance has only been described in cases series.[43-48] Tumor size varies from small (2 cm) to very large lesions (>30 cm) and tumors are

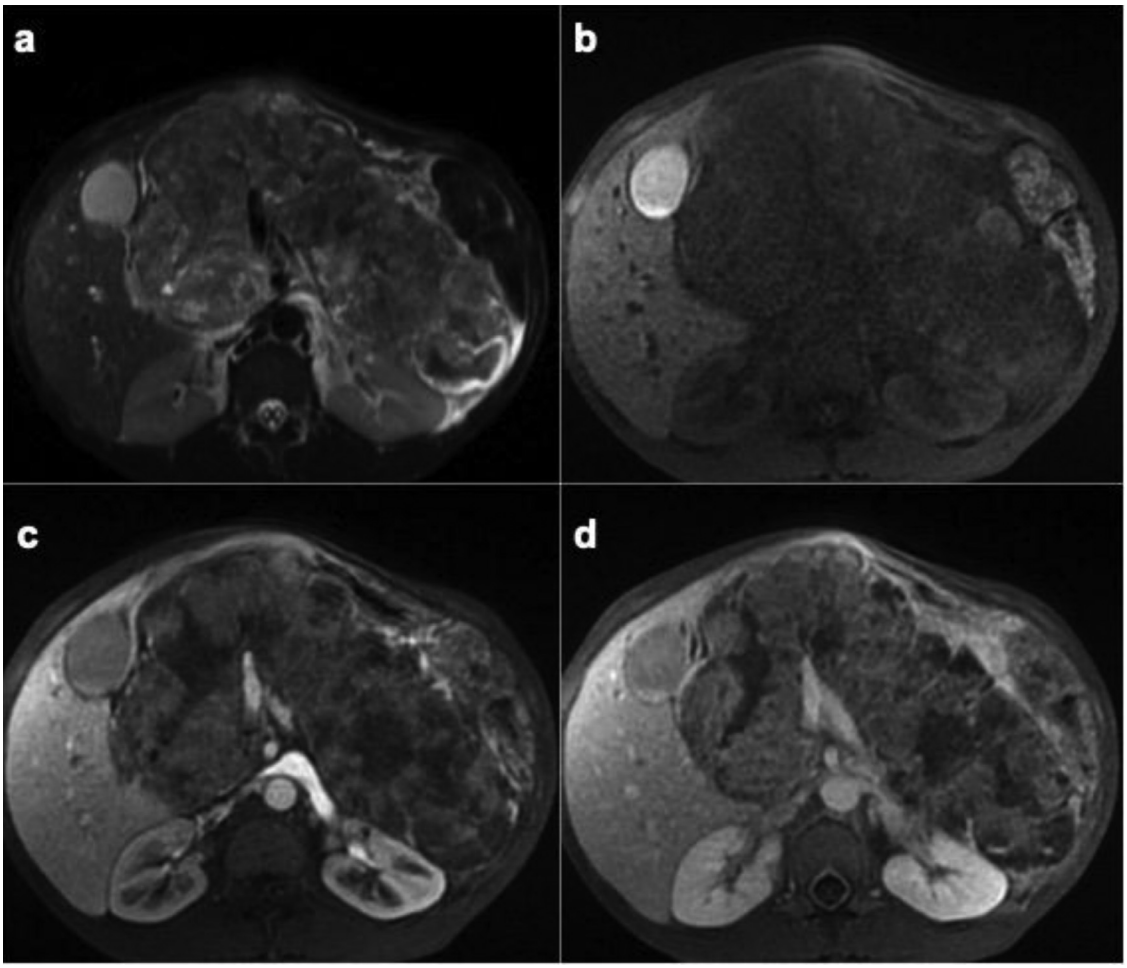

Figure 14 Pancreatoblastoma in a 29-year-old female patient. Fat-suppressed T2-weighted MR image (a) shows a large heterogeneous mass in the entire pancreas. Precontrast (b) fat-suppressed T1 GRE MR image shows that the lesion is heterogeneous with hyperintense foci. Contrast-enhanced fat-suppressed T1 GRE MR images obtained during the arterial (c) and portal phases (d) show the heterogeneous and mild enhancement of the lesion, with hypointense necrotic intratumoral changes.

usually located in the pancreatic head.[46] Half of the lesions are heterogeneous, presenting with necrotic areas or mixed cystic-solid components. The solid portions are typically hyperenhanced on contrast-enhanced CT/MR imaging (Fig. 9), and lack somatostatin receptors at Gallium 68-PET[46] which helps differentiate them from low-grade NET. Nevertheless, because this lesion is so rare, a differential radiological diagnosis with other hyperenhanced pancreatic lesions can be extremely difficult. Because pancreatic EGIST are often biologically aggressive (large tumors with high mitotic count),[43] a surgical approach should be considered when possible.

Pancreatic Metastases

The pancreas is an uncommon site for metastases from other primary malignancies. Renal cell carcinoma is the most common primary tumor that metastasizes to the pancreas, followed by the lung, breast, gastrointestinal adenocarcinoma, melanoma, and soft tissue sarcomas.[49] Other origins are uncommon. While renal cell carcinoma metastases are often multiple, metastases from other primary malignancies are more frequently solitary.[50] Pancreatic metastases may develop several years after the diagnosis of the primary tumor, especially from renal cell carcinomas, with reported cases more than 20 years after the diagnosis of the primary tumor.[50]

The imaging characteristics depend on the primary tumor. Renal cell carcinoma metastases are strongly hyperenhanced during the arterial phase[51] (Fig. 10) with possible washout, mimicking NETs. Metastases originating from other primary tumors are usually hypoattenuating/-intense lesions, mimicking PDA[39,49] (Fig. 11).

The oncological history is the key to suggesting a diagnosis, especially when other organs are involved simultaneously. In patients with a history of renal cell carcinoma, all hypervascular pancreatic lesions should be considered to be metastases until they are proven otherwise, especially when they are multiple. Kang et al have also recently suggested that relative washout between the arterial and portal phases is higher in renal cell carcinoma metastases than in low-grade NETs on CT.[52] Von Hippel-Lindau disease is a specific situation. Because of the increased risk of both renal cell carcinoma and

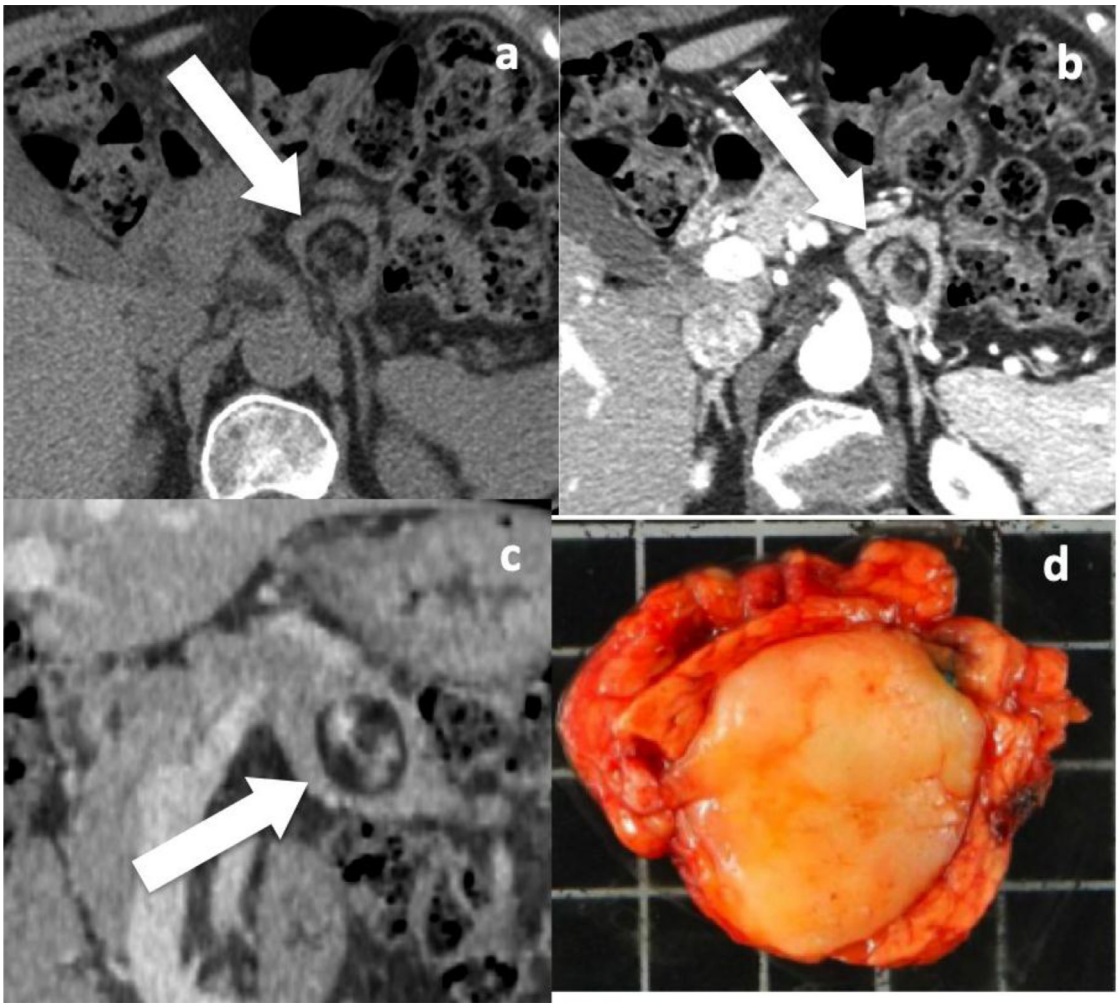

Figure 15 Pancreatic hamartoma in a 50-year-old female. Axial precontrast (a), axial (b) and coronal (c) contrast-enhanced CT images show a heterogeneous lesion in the pancreatic body. The lesion (arrows) presents with fatty hypoattenuating portions mixed with solid enhancing portions. The lesion was resected; the diagnosis of pancreatic hamartoma was confirmed on pathologic examination of the surgical specimen (d).

primary pancreatic NET in these patients, the differential diagnosis between these 2 entities can be difficult and a diagnosis of NET should be considered even in the presence of coexisting renal cell carcinoma.[53,54]

Fibrous Tumors

Desmoid tumors and solitary fibrous tumors are extremely rare neoplasms characterized by the presence of abundant fibrous tissue in the lesion. These lesions are usually benign with no metastatic potential. Nevertheless, they are associated with a high risk of local growth and recurrence after surgery.

Desmoid Tumors

Pancreatic desmoid tumors are an extremely rare benign neoplasm and the literature on imaging is based on case reports.[55] Desmoid tumors more frequently occur in young (third decade) patients with a history of "familial adenomatous polyposis,"[56] abdominal surgery, or trauma. On pathology desmoid tumors show a fibroblastic proliferative tissue with a collagen matrix. Nuclear β-catenin immunostaining helps distinguish this form from fibroblastic and myofibroblastic tumors.[57] Although the tumor has no metastatic potential, local recurrence after resection is not uncommon.[58]

Imaging features are nonspecific, the tumor is usually solid, nonencapsulated and located in the pancreatic tail.[55,59] Enhancement is progressive and delayed because of the fibrotic component of the tumor (Fig. 12). Signal intensity on T2-weighted images varies depending on the amount of fibrosis in the lesion.[55]

Solitary Fibrous Tumors

Solitary fibrous tumors are mesenchymal neoplasms that mainly arise from the pleura. A pancreatic location is extremely rare with fewer than 20 published cases in the

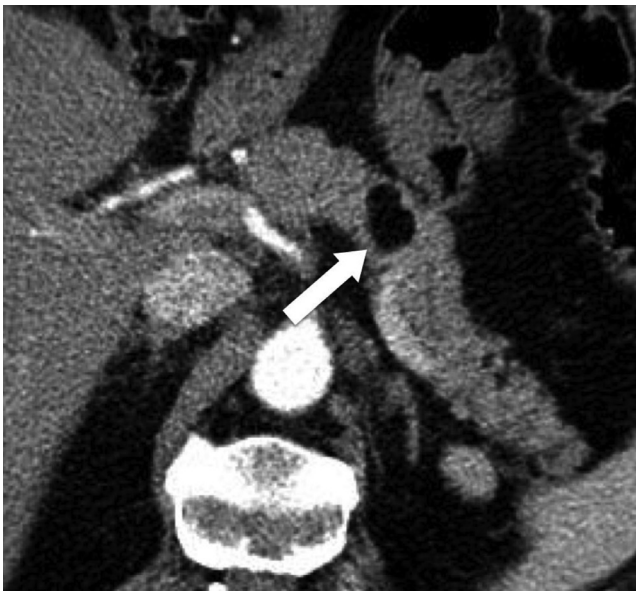

Figure 16 Pancreatic lipoma in a 73-year-old man. Contrast-enhanced CT image obtained during arterial phase shows a homogeneous nonenhancing lesion with fatty attenuation in the pancreatic body (arrow). The lesion is surrounded by pancreatic parenchyma and is clearly separated from the retroperitoneal fat. Note the absence of pancreatic duct dilatation. Case courtesy of Prof. Giuseppe Brancatelli, Palermo, Italy.

literature.[60] This tumor often develops in middle-aged patients (40-50 years old), and is more frequently found in women.[60-62]

At diagnosis, tumor size varies from small (2 cm) to very large (18 cm), located in the head or in the body of the pancreas.[60,61] A malignant potential is reported in around 12% of patients.[62,63]

Solitary fibrous tumors are usually well circumscribed with moderate enhancement during the arterial phase (tumor hypoenhanced compared to pancreatic parenchyma). Enhancement is often progressive and delayed due to the presence of fibrosis. The preoperative diagnosis is based on pathology. Indeed, because of the rarity of the disease and the tumor enhancement pattern, this entity is commonly misdiagnosed as a nonfunctional NET[60-62] (Fig. 13). Data regarding recurrence and outcome after resection are limited. Nevertheless, because of the malignant potential of some tumors, a surgical approach should be considered when possible.

Highly Uncommon Solid Tumors

Pancreatoblastoma

Pancreatic pancreatoblastoma typically occur in children (<10 years old), and rarely in adults. This tumor is extremely aggressive, with a very poor prognosis. At diagnosis, the lesion is usually large involving the entire pancreas in certain cases, and frequently associated with the presence of distant metastases.[64] On CT, foci of calcifications can be depicted. On MR imaging, the tumor is heterogeneous due to the presence of necrotic and hemorrhagic components.[65] Tumor margins are multiloculated and well-delineated. Enhancement is usually mild compared to the noninvolved pancreas (Fig. 14). The carcinoembryonic antigen and alfa-fetoprotein serum levels may be increased.

Hamartoma

Hamartoma can be considered to be a malformation rather than a tumoral proliferation. Pancreatic involvement is rare with around 30 cases reported in the literature.[66] The lesion is mostly described in middle-aged patients (50 years old), with no difference in gender. Half the patients are asymptomatic and the lesion is discovered incidentally.[66] Hamartoma may have a solid, cystic or mixed appearance. The lesions are well-delineated and the solid portions are composed of fibrous and adipose tissue. Cystic portions are composed of dilated acini and ducts. This explains the presence of hypo-attenuating areas on CT (< −30 HU) with decreased signal intensity on fat-saturated T1-weighted images on MR imaging (with spontaneous hyperintensity on T1-weighted nonfat saturated images), due to the fatty component. Nonadipose solid portions are enhanced on contrast-enhanced images[65] (Fig. 15). Recently, Matsushita et al suggested that progressive shrinking of the cystic components over time could be used as an additional finding to suggest the diagnosis.[66]

Lipoma

Pancreatic lipoma is an asymptomatic and benign lesion. It is typically discovered incidentally. The imaging features reflect the purely fatty composition with homogeneous and diffuse negative attenuation on CT (Fig. 16) a signal drop on fat-saturated MR sequences, hyperintense T1-weighted non−fat-saturated MR images, and an absence of enhancement. MR imaging can help distinguish lipoma from focal fat deposition. Indeed, the latter shows contact with the retroperitoneal fat.[67] Short-term follow-up imaging should be suggested to differentiate lipoma from early forms of liposarcoma.[68]

Leiomyosarcoma

Leiomyosarcoma is an extremely rare pancreatic neoplasm, representing 0.1% of all malignant pancreatic exocrine neoplasms. It arises from the smooth musculature of the pancreatic duct or vessels.[69] Imaging features are nonspecific, including a large heterogeneous mass, with solid, cystic, and mixed portions. Necrotic and hemorrhagic changes may be associated.

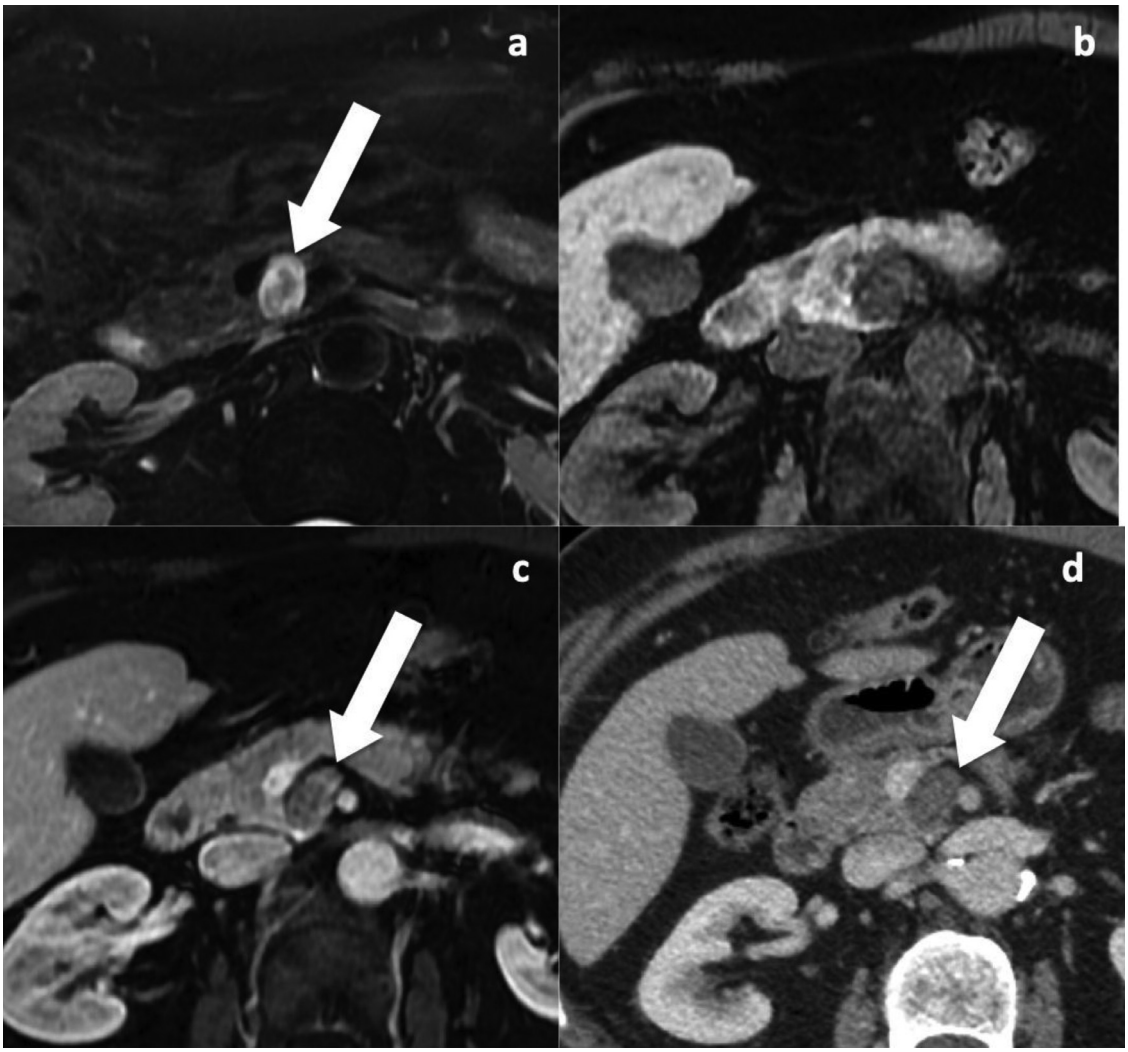

Figure 17 Pancreatic schwannoma in a 73-year-old male patient. Fat-suppressed T2-weighted MR image (a) shows a hyperintense lesion of the pancreatic head (arrow). The lesion is well delineated and hypointense on precontrast fat-suppressed T1 GRE MR image (b) and shows mild-moderate enhancement on enhanced fat-suppressed T1 GRE MR image (C) and on contrast-enhanced CT image obtained during the portal venous phase (d).

The high rate of metastases at diagnosis in contrast to rare lymph node involvement[70] may help suggest a diagnosis.

Schwannoma

Schwannomas are tumors originating from the Schwann cells of peripheral nerve sheaths. A pancreatic location is very rare and tumors are more often located in the pancreatic head. Imaging features are different depending on the microscopic structure. Two patterns of cellular organization have been described. The "Antoni A" pattern is hypercellular with closely packed spindle cells, while the "Antoni B" is hypocellular with degenerative cystic changes. Thus, Antoni A tumors are more likely to be solid (Fig. 17) and moderately enhancing, while Antoni B tumors present with cystic or mixed cystic-solid appearance.[71]

Although most of these tumors are benign, malignant transformation has been reported in 11%-12% of cases. Size (>7 cm), the presence of ill-defined margins, vascular encasement, and adjacent organ invasion suggest malignant schwannoma.[72]

An Evidence-Based Approach

Several rare solid pancreatic neoplasms may mimic PDA and NET. The most typical radiological, clinical, and biological features characterizing these rare neoplasms are listed in Table 2. Among these rare lesions, SPN presents with specific epidemiologic and imaging features, often making correct preoperative diagnosis possible. In the presence of atypical imaging, clinical or biological features of classical PDA or NET, a rare mimicking tumor should be considered. Cases should be discussed by multidisciplinary team meetings for this purpose including surgeons, gastroenterologists, radiologists, pathologists, and interventional radiologists. As resection, when possible, can improve survival in most cases, a preoperative histologic diagnosis must be obtained, except when SPN is suspected.

Table 2 Rare Solid Tumors of the Pancreas With Main Mimickers and Main Suggestive Features

Tumor	Main Mimicker	Suggestive Imaging Feature	Biological	Context
Acinar cell carcinomas (ACC)	Pancreatic ductal adenocarcinoma	Well-defined margins	Lipase and AFP secretion	X
Solid pseudopapillary neoplasms (SPN)	Neuroendocrine tumor	- Capsule - Cystic and solid if >3 cm - Early progressive enhancement - No MPD dilatation or upstream atrophy	X	Young female (20-30 years old)
Anaplastic adenocarcinoma	Pancreatic ductal adenocarcinoma	Nonspecific (cystic changes within the lesion)	x	x
Adenosquamous adenocarcinoma	Pancreatic ductal adenocarcinoma	- Central necrosis, with rim like enhancement - Portal vein invasion - Enlarged lymph node	x	x
Colloid adenocarcinoma	Mucinous tumors	- "Salt and pepper" appearance on T2 - Peripheral and internal sponge-like or mesh-like appearance - Progressive delayed contrast enhancement - Absence of communication with main pancreatic duct on MRCP	X	X
Solid serous cystadenoma	Neuroendocrine tumor	- Very bright on T2- with high ADC values - Multicystic appearance on heavily T2 and MRCP images	x	x
Pancreatic extra gastrointestinal stromal tumor	Neuroendocrine tumor	- Lack of somatostatin receptors at Gallium 68 PET - Radiological differential diagnosis with other hypervascular lesion extremely difficult	x	x
Hyperenhanced metastases	Neuroendocrine tumor	Relative washout		History of renal cell carcinoma
Hypoenhanced metastases	Pancreatic ductal adenocarcinoma	Diffuse metastatic disease	x	History of malignancy
Desmoid tumor	Pancreatic ductal adenocarcinoma	- Well-limited nonencapsulated enhancing lesion. - Delayed enhancement	x	Gardner syndrome / FAP History of abdominal surgery or trauma
Solitary fibrous tumor	Neuroendocrine tumor (nonfunctional form)	- Well-limited nonencapsulated lesion. - Delayed enhancement due to fibrosis	x	x
Primary lymphoma	Pancreatic ductal adenocarcinoma	- Mild or no pancreatic duct dilatation - Enlarged locoregional lymph node	x	Invaliding chronic abdominal pain is less frequent in lymphoma than in ductal adenocarcinoma

ACC, acinar cell carcinoma; ADC, apparent diffusion coefficient; AFP, alfa-fetoprotein; FAP, familial adenomatous polyposis; MPD, main pancreatic duct; MRCP, magnetic resonance cholangiopancreatography; SPN, solid pseudopapillary neoplasm.

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