



Pancreatic Calcifications and Calcified Pancreatic Masses: Pattern Recognition Approach on CT

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OBJECTIVE. The purpose of this article is to review a spectrum of calcified pancreatic masses and propose an algorithm for diagnostic radiologic evaluation.

CONCLUSION. Pancreatic calcifications are being detected more frequently because of the widespread use of imaging, particularly CT. Pancreatic calcifications are most commonly associated with chronic pancreatitis related to alcohol abuse. Several other pathologic entities, however, can cause pancreatic calcifications. Familiarity with these entities and their CT appearance is helpful in making an accurate diagnosis.

Pancreatic calcifications are key features commonly used to diagnose various types of pancreatic disease. Chronic calcific pancreatitis secondary to alcohol use is the most common cause of pancreatic calcifications [1]. Other pathologic conditions affecting the pancreas also can cause calcifications. Awareness of these entities and their classic features is important in making the correct diagnosis and guiding proper management. We describe various causes of pancreatic calcifications and their imaging appearances at CT.

Inflammatory Etiologic Factors

Chronic calcifying pancreatitis is the most common cause of pancreatic calcifications. Other types of chronic pancreatitis, such as obstructive pancreatitis and autoimmune pancreatitis, rarely cause pancreatic calcifications [2]. Chronic pancreatitis results from continuous or recurrent episodes of inflammation, which in turn cause irreversible parenchymal damage and impairment of the exocrine and endocrine components of the gland. The incidence of chronic pancreatitis is approximately 0.01% in industrialized countries, which means that chronic pancreatitis occurs in approximately 5–12 per 100,000 people [3].

Alcohol consumption is the most common cause of chronic calcifying pancreatitis, accounting for as many as 50–70% of cases [1, 4]. Chronic alcoholic pancreatitis is more common in men than women, 59% compared with 28% [5], which may be due

to the increased frequency of homozygosity of the *CLDN2* gene locus in men, which has been linked to alcoholic pancreatitis [6]. In a meta-analysis, Irving et al. [7] found that the incidence of chronic pancreatitis increases exponentially with increasing alcohol consumption of more than four drinks per day. Calcification is found in 20–40% of patients with chronic alcoholic pancreatitis and can be focal or diffuse [8].

Smoking and genetic factors are other major risk factors for chronic calcifying pancreatitis [2]. Smoking-related chronic pancreatitis, like alcoholic pancreatitis, is dose dependent. The risk of chronic pancreatitis increases considerably when an individual smokes more than one pack per day and decreases after smoking cessation [9].

Several studies have identified genes that are predisposing factors for chronic pancreatitis. The *PRSS1* gene is associated with hereditary pancreatitis, the *SPINK1* gene is associated with tropical calcific pancreatitis, and the *CFTR* gene is associated with cystic fibrosis and idiopathic pancreatitis [10–12].

The formation of pancreatic calcifications is theorized to be due to ductal obstruction by proteinaceous plugs and accumulation of calcium carbonate, which causes ductal ectasia and periductal fibrosis (Fig. 1). Other factors that contribute to ductal injury include sphincter of Oddi obstruction, biliopancreatic reflux, and duodenopancreatic reflux. Ductal calcifications can present in the main pancreatic duct or in side-branch ducts. Parenchymal calcifications can vary morpho-

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TABLE 1: Specificity of Calcification in Chronic Pancreatitis

Location of Calcifications	Specificity (%)
Only parenchymal	67
Only intraductal	88
Diffuse parenchymal	91
Coexisting parenchymal and intraductal	100

logically and in size and may be focal or diffuse throughout the pancreas (Table 1). The degree of calcifications increases with disease progression [13]. Scattered parenchymal or intraductal calcifications occur in approximately 50% of patients, parenchymal atrophy in 54% of patients, and ductal dilatation in 68% of patients [14].

Other key characteristics of interstitial edematous pancreatitis are acute peripancreatic fluid collections and pseudocysts. Acute peripancreatic fluid collections occur less than 4 weeks after an episode of interstitial edematous pancreatitis. Pseudocysts, however, occur after 4 weeks and are identified in 25% of patients [15]. Pseudocysts are well-defined fluid collections with an enhancing capsule that may be present around the pancreas or distant from the pancreas. They can mimic pancreatic cystic neoplasms but rarely calcify. In these cases, features associated with chronic pancreatitis, such as calcifications, can help in confirming the diagnosis of chronic pancreatitis with associated pseudocysts [16] (Fig. 2). Other secondary signs of pancreatitis include splenic vein thrombosis, which is a complication of pancreatitis in approximately 11% of patients [17]. Superior mesenteric vein and portal vein thrombosis and portal hypertension can also be seen in chronic pancreatitis.

Developmental Etiologic Factors

Cystic Fibrosis

Cystic fibrosis (CF) is an autosomal recessive multisystem disease that most commonly involves the pancreas. It results from a mutation in the CF transmembrane conductor regulator gene (*CFTR*). CF more frequently affects white people and is the most common cause of pancreatitis in the pediatric population. With improved survival, there is a growing population of adults with CF. In CF, the chloride-pumping channel in exocrine glands is defective. Viscous epithelial secretions obstruct the proximal pancreatic ducts; functioning acinar cells are replaced with adipose tissue and then

with fibrotic tissue. Exocrine pancreatic insufficiency is found in 85–95% of patients with CF, and 30–50% of CF patients have endocrine pancreatic insufficiency [18].

Lipomatous pseudohypertrophy or fatty replacement of the pancreas is the most common imaging finding in CF. Calcifications are less common with an incidence of 8% of cases. Calcifications in the dilated pancreatic duct are caused by a high serum concentration of calcium and altered calcium-binding characteristics. Diffuse parenchymal calcifications and pancreatic atrophy can also be seen in patients with advanced pancreatic insufficiency [19] (Fig. 3). Associated with calcifications, small unilocular pancreatic cysts measuring 1–3 mm can also be seen; complete replacement of the pancreas with cysts (pancreatic cystosis) rarely occurs.

Shwachman-Diamond Syndrome

Shwachman-Diamond syndrome is a rare autosomal recessive disorder and is the second-most common cause of pancreatic insufficiency in children. Varying degrees of hematologic and skeletal abnormalities have also been described, including leukemia, pancytopenia, metaphyseal dysostosis, and short stature. Histologically, most of the pancreatic acinar tissue is replaced by fat, and the islet cells and ductal architecture are preserved [20, 21]. Diffuse fatty infiltration is commonly seen, and calcifications are rare (Fig. 4). When present, calcifications are typically scattered throughout the pancreatic parenchyma. Fatty replacement of the pancreas can be seen in both Shwachman-Diamond syndrome and CF. The overall size of the pancreas is unchanged in Shwachman-Diamond syndrome but decreased in CF [22].

Neoplastic Etiologic Factors

Neuroendocrine Tumors

Neuroendocrine tumors (NETs) can be divided into hyperfunctioning and nonhyperfunctioning types on the basis of clinical presentation and hormonal production. Patients with hyperfunctioning tumors present with symptoms related to overproduction of a hormone. Hyperfunctioning tumors tend to be small, frequently smaller than 2 cm in diameter, and present earlier owing to symptoms caused by hormone production. Functioning NETs, such as insulinomas, have been associated with calcifications in approximately 20% of cases. Nonhyperfunctioning NETs, however, tend to present later as larger tumors because of the lack of hormone pro-

duction. Large NETs can cause local and vascular invasion [23] and are more likely to outgrow their blood supply, resulting in cystic changes and dystrophic calcifications. Nonhyperfunctioning NETs contain calcifications more commonly than do hyperfunctioning tumors. The calcifications are typically located centrally within the large mass, and the pattern can be coarse, focal, and irregular [23]. In one study, 22% of nonhyperfunctioning NETs contained calcifications [24]. In another study, 7 of 10 NETs with calcifications had malignant features [25].

Solid Pseudopapillary Tumors

Solid pseudopapillary tumors are rare low-grade malignant tumors representing nearly 1% of pancreatic exocrine tumors. Approximately 90% of these tumors occur in young women, particularly those of African American descent [26, 27]. At imaging, these tumors typically present as large (mean size, 9 cm) encapsulated solid and cystic tumors with hemorrhagic degeneration, predominantly in the pancreatic tail. Calcifications are common, reported in 30% of cases, and are characteristically peripheral and punctate [28] (Fig. 5). The capsule and solid portion of a solid pseudopapillary tumor typically are enhancing in a pattern similar to that of normal pancreatic parenchyma in both the arterial and the venous phases. Hyperattenuating areas on CT images correspond to high signal intensity on T1-weighted MR images, representing hemorrhagic components. In one study [28], approximately 18% of tumors had a fluid-debris level representing cystic hemorrhagic cavities. Various imaging modalities, including CT, MRI, and ultrasound, can consistently depict cystic and hemorrhagic areas of the tumor, but punctate peripheral calcifications are visible only with CT [28]. It is difficult to differentiate the benign and malignant forms of solid pseudopapillary tumors at imaging unless more aggressive features are seen, including vascular involvement, distant metastases, and pancreatic duct dilatation [29].

In extremely rare instances, solid pseudopapillary tumors are seen in men, in whom the tumors tend to be larger, have a lobulated contour, and are progressively enhancing. Rather than occurring at a younger age as they do in women, in men solid pseudopapillary tumors occur at a more advanced age [30]. There is no substantial difference in calcification patterns between benign and malignant solid pseudopapillary tumors. In one study of 82 patients

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with solid pseudopapillary tumors [31], 39% of patients had calcifications. Nodular focal calcifications and incomplete rim calcifications were seen in both the group with benign tumors and the group with malignant tumors. In the benign group, one tumor had complete rim calcification and seven had amorphous and scattered calcifications.

Intraductal Papillary Mucinous Neoplasm

Intraductal papillary mucinous neoplasm (IPMN) is a rare cystic tumor arising from the pancreatic duct. IPMN is more prevalent in older men (mean age, 65 years; male-to-female ratio, 2.2). It most commonly develops in the head of the pancreas [32]. The prevalence of IPMN has increased dramatically owing to an increase in use of cross-sectional imaging. In a 2012 article [33], they were reported to constitute as many as 50% of cystic pancreatic neoplasms.

In IPMNs the main or side-branch pancreatic ductal epithelium is replaced by columnar cells that secrete mucin. The mucin distends the orifice and pancreatic duct [34]. IPMNs appear as multilocular cystic masses in the pancreas that communicate with the main or a side-branch pancreatic duct; however, the communication with the pancreatic duct can be difficult to visualize at CT. MRCP is used to visualize the communication with the main or side-branch pancreatic duct [35, 36].

There are three types of IPMN: main duct, side branch, and mixed. Most side-branch IPMNs are benign and asymptomatic; however, when a side-branch IPMN is large, it can compress the main pancreatic duct and cause pancreatic ductal dilation and obstructive pancreatitis [37]. Malignant IPMNs can cause fistulas to the duodenum, stomach, or colon. The presence of a mural nodule increases the likelihood of malignancy [32]. In one study [38], only 8% of side-branch IPMNs with negative cytologic results proved to be malignant after resection. On the contrary, a higher risk of malignancy has been associated with main-duct IPMNs that dilate the main pancreatic duct 1 cm or more and have a solid nodular component larger than 1 cm and thus require surgical management [39].

The most likely explanation for the calcifications in an IPMN is the presence of mucin, which has a tendency to build up calcium salt deposits. The patients also tend to have underlying chronic calcific pancreatitis. The calcification pattern in IPMN is indistinguishable from that in chronic pancreatitis; calcifications can be found in the pancreat-

ic duct, parenchyma, or diffusely throughout the gland [40] (Fig. 6). Calcifications are reported in 20% of IPMNs. Punctate calcification is the most common pattern (87%), followed by coarse calcification (33%). The presence, pattern, or location of calcifications in IPMN has no correlation with malignancy [40, 41]. Taouli et al. [42] calculated a 77% likelihood of malignancy in main-duct IPMNs when intraluminal calcification was present, but most of the lesions had other concurrent features of malignancy. In another study [40], coarse calcification was associated with malignancy in more than 80% of cases, but these lesions also had coexisting malignant features, such as main-duct dilatation, solid nodules, or size larger than 3 cm.

Serous Cystic Neoplasm

Serous cystic neoplasm is a benign microcystic pancreatic tumor. It is a slow-growing tumor and tends to occur in older patients (> 60 years) [43]. The lesions are often discovered incidentally at imaging but can present with nonspecific symptoms. Serous cystadenoma is typically composed of multiple small thin-walled cysts with a central calcified scar. The central scar consists of calcified septations radiating outward with a sunburst or stellate appearance. The calcified central scar seen in 30% of cases is highly specific for the diagnosis of serous cystadenoma [13, 44] (Fig. 7).

Three CT morphologic cystic patterns have been described: polycystic, honeycomb, and oligocystic. Polycystic lesions are the most common, accounting for 70% of cases. They consist of numerous cysts measuring up to 2 cm. Thin fibrous septations that separate the small cysts can coalesce and cause a central scar. The honeycomb pattern consists of innumerable subcentimeter cysts and is seen in 20% of cases. These small cysts cannot be visualized individually on CT images. They have a spongelike appearance and soft-tissue attenuation. Sometimes further characterization with MRI is needed because of the indeterminate, pseudosolid appearance of these lesions on CT images. The cystic lesions are T2-hyperintense on MR images. The oligocystic type is seen in less than 10% of cases. This type consists of larger, macrocystic lesions that can be difficult to differentiate from mucinous cystadenoma or IPMN [45]. Management of serous cystic neoplasms ranges from imaging surveillance to resection based on its size, clinical presentation, and possible

malignant transformation [46]. Serous cystadenocarcinomas are exceedingly rare [47].

Mucinous Cystic Neoplasm

Mucinous cystic neoplasms are macrocystic neoplasms more commonly seen in women in the sixth decade of life (male to female ratio, < 1:20). These neoplasms arise from the body or tail of the gland in 93–97% of cases and do not communicate with the pancreatic duct [48–50]. These macrocystic tumors can be unilocular or multilocular, individual cystic components measuring 2 cm or greater. These lesions consist of mucin-producing ovarian stroma and may contain hemorrhage or debris [44]. Approximately 48% of the stromal cells have progesterone receptors, and 22% have estrogen receptors [51]. Curvilinear or peripheral eggshell-like calcifications are present in the cyst wall or septa in 15% of cases [52]. Multilocular macrocystic lesions with thick walls and calcification in the wall have a higher risk of malignancy than do unilocular lesions with thin walls without calcification [53] (Fig. 8).

It is important to differentiate mucinous cystic neoplasm from serous cystic neoplasm and IPMN, because mucinous cystic neoplasm is more frequently managed surgically, because of its malignant potential. Management of serous cystic neoplasm or side-branch IPMN is conservative with follow-up imaging [50, 54, 55]. The prognosis of benign and borderline malignant mucinous cystic neoplasms is favorable (5-year survival rate, > 95%) [36].

Pancreatic Adenocarcinoma

Pancreatic adenocarcinoma is the most common pancreatic malignancy and typically does not calcify. Few cases of calcified adenocarcinomas have been reported in the literature [56–60]. Calcifications in adenocarcinoma can be explained by the occurrence of adenocarcinoma on top of pre-existing chronic calcific pancreatitis. The presence of calcifications can also be caused by pancreatic ductal obstruction by adenocarcinoma. In one study [60], only 4% of calcifications were seen in patients with pancreatic adenocarcinoma. In that study, the calcifications were seen within the nonneoplastic pancreatic tissue and not within the adenocarcinoma in three of four patients, which would suggest that adenocarcinoma arises on top of chronic calcific pancreatitis. Calcification within the adenocarcinoma was seen in only one patient without underlying chronic calcific pancreatitis (Fig. 9).

Acinar Cell Carcinoma

Acinar cell carcinoma is a rare malignant exocrine pancreatic tumor. It typically occurs in men in the 5th through 7th decades of life and accounts for less than 2% of primary pancreatic neoplasms [61, 62]. Although the symptoms are nonspecific, abdominal pain and weight loss are the most common presentations, and pancreatitis and obstructive jaundice are uncommon [63, 64]. These tumors can secrete lipase, amylase, trypsin, and chymotrypsin. Fifteen percent of patients may present with a paraneoplastic lipase hypersecretion syndrome, which results in polyarthralgia, subcutaneous fat necrosis, and peripheral eosinophilia due to the increased secretion of serum lipase. Some patients may have elevated α -fetoprotein levels [65]. At histopathologic analysis, the lesions are periodic acid–Schiff positive, and at immunohistochemical analysis they are strongly positive for acinar enzymes, such as trypsin and chymotrypsin [66]. The imaging appear-

ance of acinar cell carcinoma is a large solid well-circumscribed mass, usually larger than 5 cm at presentation. At unenhanced CT, the mass typically has the same attenuation as or lower attenuation than the surrounding parenchyma, has low attenuation on arterial phase contrast-enhanced CT images, and is more enhancing than the pancreatic parenchyma in the venous phase. Most of the tumors are exophytic masses with an enhancing capsule without pancreatic ductal dilatation, which differentiates them from adenocarcinoma [67]. Calcifications have been reported in 6–50% of patients in different studies [63, 68, 69] (Fig. 10). In one study of 10 patients [68], five of the patients had calcifications. In two of these patients the calcification pattern was central punctate, in one it was peripheral punctate, in one it was central stellate, and in one it was peripheral plaque calcifications.

Metastasis at presentation has been reported in 50–67% of the patients, and the liver is the most common site [63, 64]. Acinar cell

carcinoma has a more indolent course and better prognosis than pancreatic adenocarcinoma, but it has a higher rate of recurrence. Resection is the treatment of choice. Results of more recent studies suggest that surgery and chemotherapy have a more favorable outcome than does surgery alone [70].

Calcified Pancreatic Metastases

Metastases are uncommon in the pancreas and rarely calcify. The most common cancers metastasizing to the pancreas are renal (30%), lung (27%), and breast (12%) carcinomas, sarcoma (8%), and less frequently, colon carcinoma and melanoma [71]. Calcified metastases to the pancreas have been reported from primary cancers of the kidney, colon, and ovary and from osteosarcoma [71–73] (Fig. 11).

Pancreatoblastoma

Pancreatoblastoma is a rare primary pancreatic malignancy in children. It presents as a heterogeneous solid mass with mostly well-

TABLE 2: Diagnostic Approach to Pancreatic Calcifications

Pathologic Finding	Incidence of Calcification (%)	Pattern of Calcification	Notes
Cystic			
Pseudocyst	Very rare	Outer rim of calcification, milk of calcium	Associated with other features of chronic pancreatitis
Mucinous cystic neoplasm	15	Curvilinear or punctate calcifications	May be associated with calcifications of the septa
Serous cystic neoplasm	30	Central calcified scar	Typically occurs in elderly women
Intraductal papillary mucinous neoplasm	1–2	Intraductal calcifications without features of pancreatitis	Mucin distends the orifice and duct (can be seen at endoscopy)
Solid pseudopapillary tumor	30	Peripheral and frequently punctate calcifications	Typically occurs in young women
Solid			
Hypoenhancing			
Adenocarcinoma	Rare	Calcification may be seen adjacent to the mass	Can be associated with pancreatitis
Focal pancreatitis	50	Focal calcifications	May be diffuse parenchymal, intraductal, or both
Hyperenhancing			
Neuroendocrine tumor	22	Focal, coarse, irregular central calcifications	Calcifications are more common in nonhyperfunctioning neuroendocrine tumors
Variable			
Acinar cell carcinoma	6–50	Variable, nonspecific	Typically occur in elderly men
Calcified metastasis	Very rare	Variable, nonspecific	Metastases have overall extremely low incidence in pancreas; incidence of calcification is even lower
Pancreatoblastoma	30	Variable, nonspecific	Typically occur in children
Mimics			
Calculus	Variable	Discrete intraductal calculus	Discrete focal calcification not associated with mass
Vascular	Variable	Involving arterial wall or aneurysm	Associated with atherosclerotic disease elsewhere

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defined margins. Clustered calcifications and rim calcifications are reported in 30% of cases [74]. Pancreatoblastoma frequently metastasizes to the liver. In some cases, it is difficult to determine the pancreatic origin of the tumor, because the tumor is large and invades the liver, and it may present as large hepatic metastatic lesions inseparable from the original pancreatic mass. Calcifications in hepatic metastases have also been reported [74]. The clinical presentation can range from asymptomatic to a palpable mass or jaundice due to biliary obstruction. Laboratory values, including levels of α -fetoprotein, α -antitrypsin, and lactate dehydrogenase, can be elevated. The prognosis is favorable in surgically resectable cases. Neoadjuvant therapy and radiation are used in the treatment of metastatic and recurrent cases [75]. This tumor is extremely rare in adults and has a poor prognosis [76, 77].

Miscellaneous Etiologic Factors

Vascular Calcifications, Aneurysms, and Pseudoaneurysms

Vascular calcifications mimicking calcified pancreatic lesions typically involve the splenic, gastroduodenal, and pancreaticoduodenal arteries (Fig. 12). Vascular calcifications are usually linear and have a tram-track appearance. They also can present as a true aneurysm or pseudoaneurysm. A true aneurysm wall can calcify as the result of atherosclerosis; evidence of communication with an artery is essential for correct diagnosis. Pseudoaneurysm formation results from an underlying chronic condition; it is commonly seen in patients with chronic pancreatitis or patients who have previously undergone pancreaticobiliary surgery [78]. Untreated pseudoaneurysms have a high mortality rate owing to their risk of spontaneous hemorrhage [79].

Milk of Calcium

Milk of calcium is due to a fluid-calcium level in pancreatic pseudocysts. The cause of milk of calcium is unclear. It has been suggested, however, that a pseudocyst causes stasis of calcium-containing suspensions, which results in dependent layering of calcium and is seen as high-attenuation material on CT images [80, 81] (Fig. 13).

Mimics of Calcified Lesions

There are several mimics of pancreatic calcifications. Oral contrast medium or hyperdense ingested material retained in duodenal or gastric diverticula can be mistaken

for pancreatic calcifications [13]. A rare case of intrapancreatic gastric duplication cyst with calcification also has been reported [82]. Calcified calculi can be seen within biliary and pancreatic ducts (Figs. 14 and 15). These appear as high-attenuation foci on contrast-enhanced CT images and can be misinterpreted as enhancing masses. Thus, reviewing unenhanced CT images is important to confirm that these are calcified calculi rather than enhancing lesions.

Conclusion

A wide spectrum of pathologic conditions affecting the pancreas can cause calcifications. A pattern recognition approach entailing the morphologic characteristics of the pancreatic lesion and pattern of calcification can be helpful in making a reasonable differential diagnosis and reaching a specific diagnosis, because the management of these lesions varies. Table 2 summarizes the diagnostic approach to pancreatic calcifications. Imaging studies should be interpreted in the clinical context in conjunction with relevant laboratory findings. Occasionally, definitive diagnosis cannot be made with imaging alone, and tissue sampling should be performed because several of these entities can have overlapping features.

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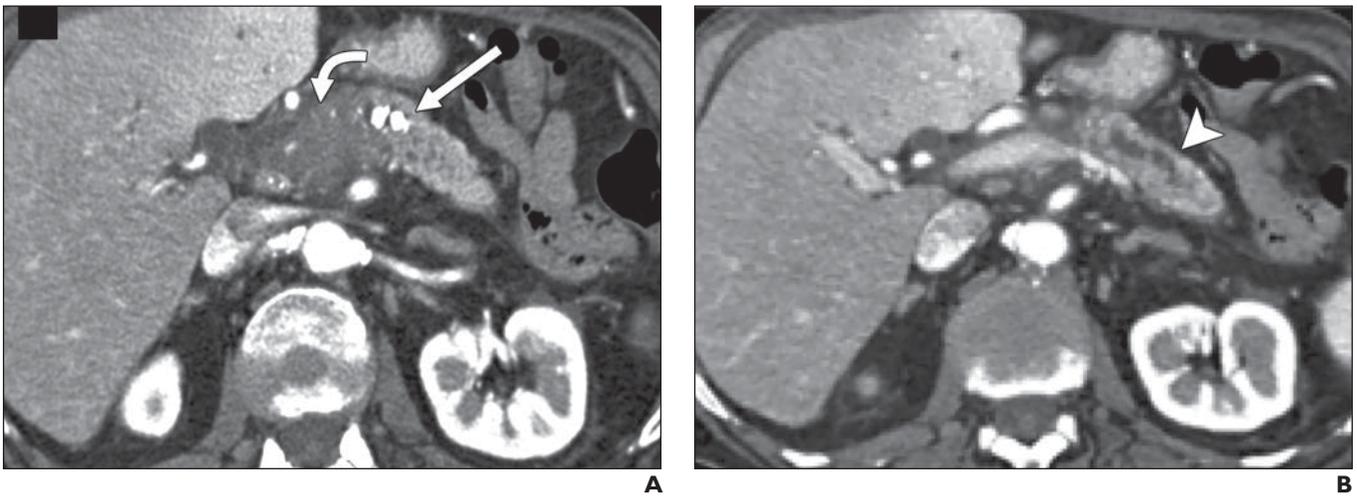


Fig. 1—72-year-old man with focal chronic pancreatitis. **A**, Axial contrast-enhanced CT image shows intraductal calcifications (*straight arrow*) associated with dilatation of main pancreatic duct. Hypoattenuating area of focal pancreatitis (*curved arrow*) within pancreatic head can mimic cancer. Presence of other clues to pancreatitis, namely calcifications, can help in making accurate diagnosis. **B**, Axial contrast-enhanced CT image shows multiple calcifications diffusely scattered within pancreatic parenchyma as result of chronic pancreatitis. Arrowhead indicates dilatation of main pancreatic duct.

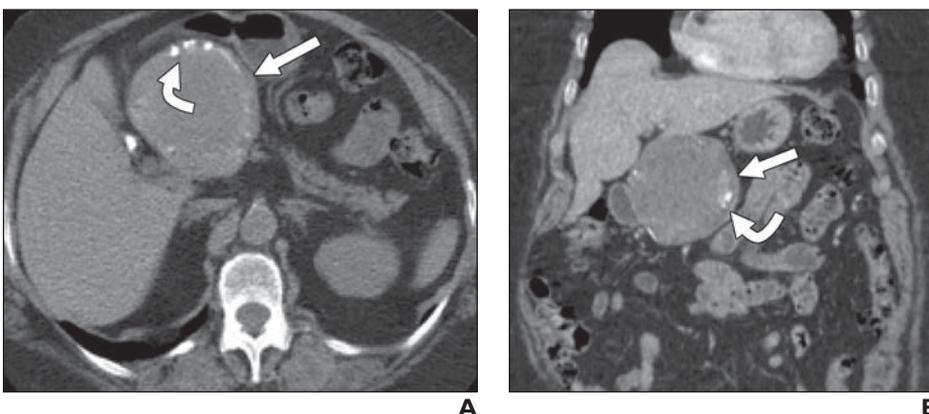
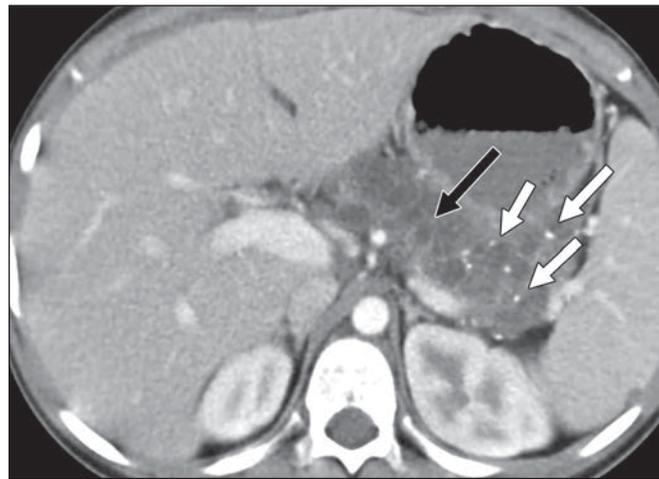


Fig. 2—65-year-old woman with history of chronic pancreatitis. **A** and **B**, Axial (**A**) and coronal reformatted (**B**) contrast-enhanced CT images show well-circumscribed low-attenuation collection (*straight arrow*) and peripheral calcifications (*curved arrow*) consistent with calcified pseudocyst.



A



B

Fig. 3—26-year-old man with cystic fibrosis.

A, Axial contrast-enhanced CT image shows multiple dilated bronchi (*arrows*) in both lungs.

B, Axial CT image through upper abdomen shows innumerable cysts and cystosis (*black arrow*) secondary to cystic fibrosis. Multiple calcific foci (*white arrows*) are scattered throughout pancreatic parenchyma.

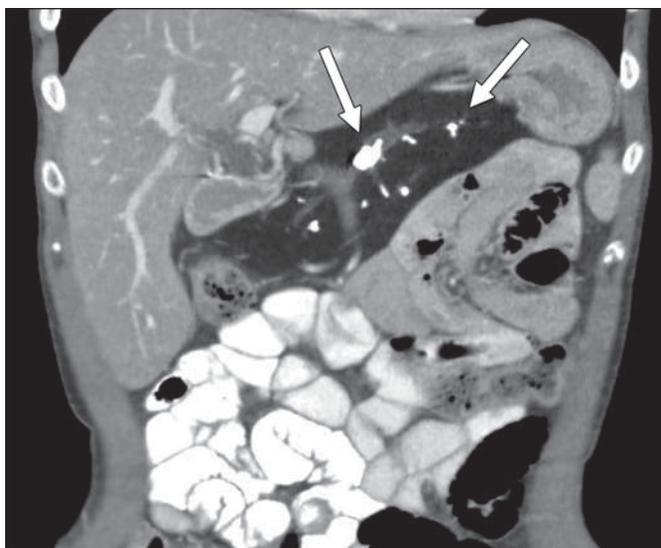
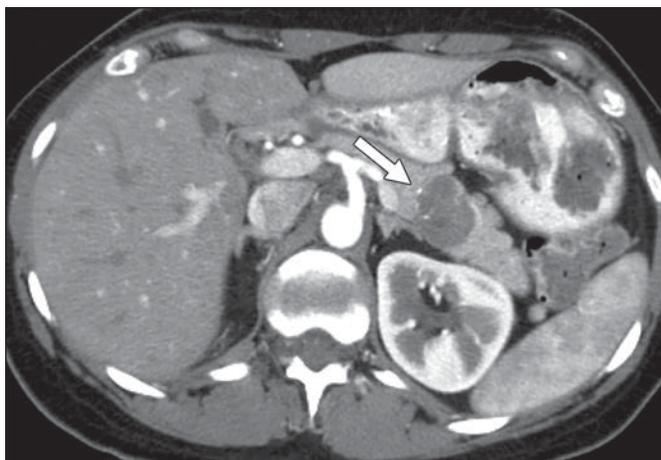
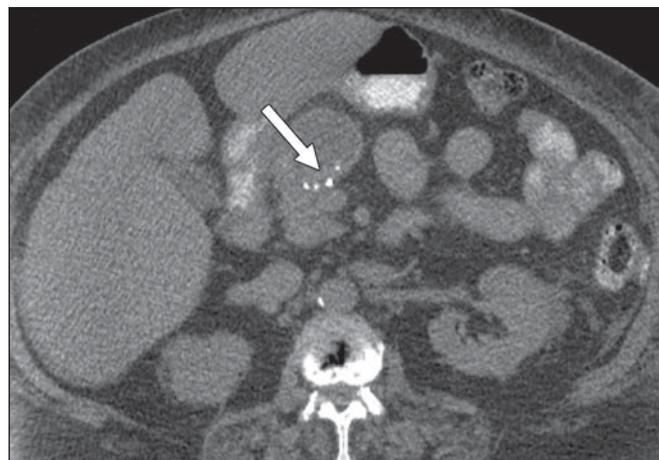


Fig. 4—50-year-old man with Shwachman-Diamond syndrome. Coronal reformatted contrast-enhanced CT image shows diffuse lipomatous infiltration of pancreas with multiple calcifications (*arrows*) secondary to Shwachman-Diamond syndrome.



A



B

Fig. 5—Two patients with surgically proven solid pseudopapillary tumor.

A, Axial contrast-enhanced CT image shows punctate peripheral calcifications (*arrow*).

B, Axial contrast-enhanced CT image shows punctate peripheral calcifications (*arrow*).

CT of Pancreatic Calcifications

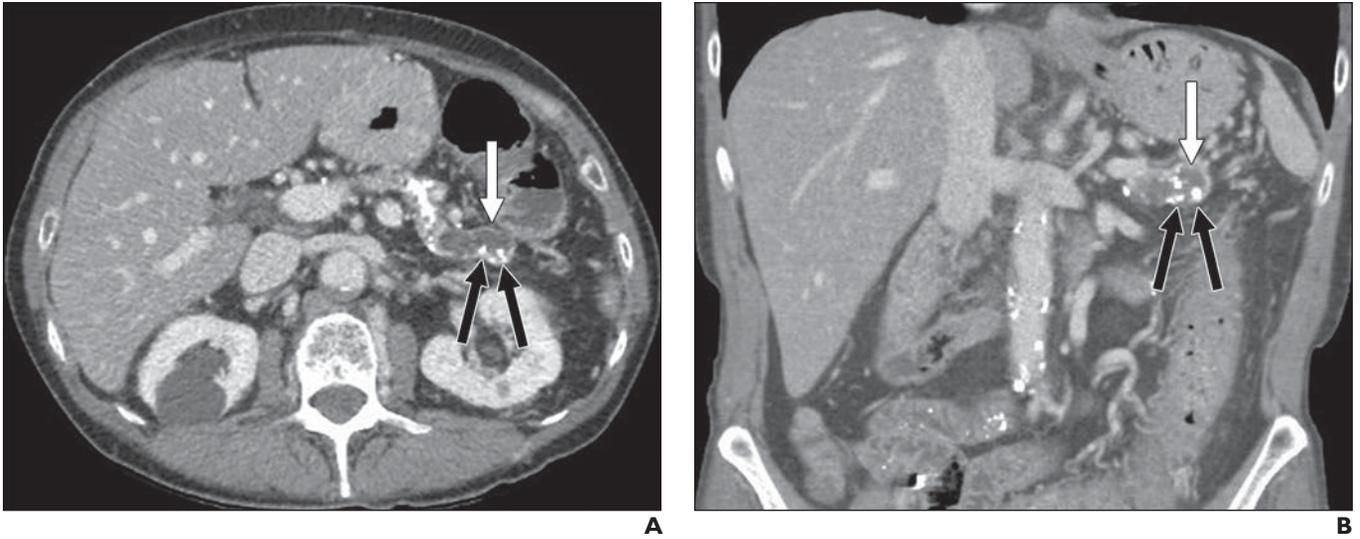


Fig. 6—65-year-old man with intraductal papillary mucinous neoplasm. **A and B**, Axial (**A**) and coronal (**B**) reformatted contrast-enhanced CT images through pancreas show significant segmental dilatation of main pancreatic duct (*white arrow*) at tail secondary to intraductal papillary mucinous neoplasm with small foci of intraductal calcifications (*black arrows*).

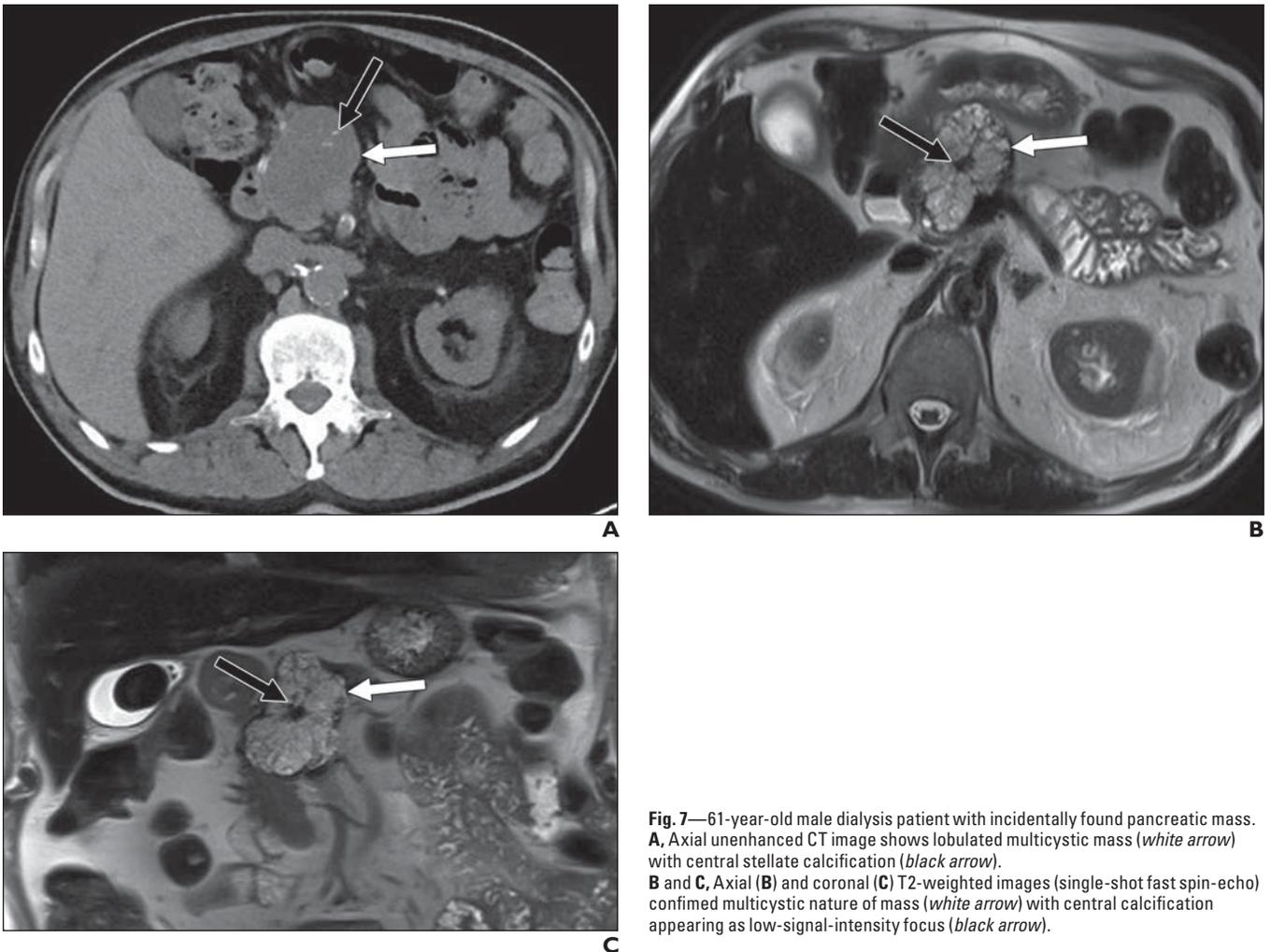
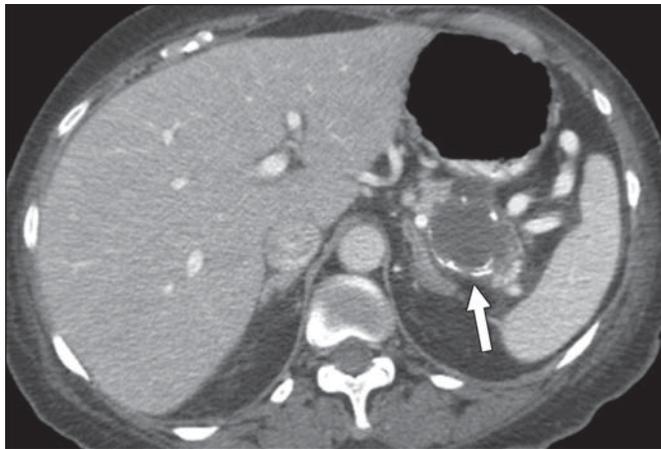
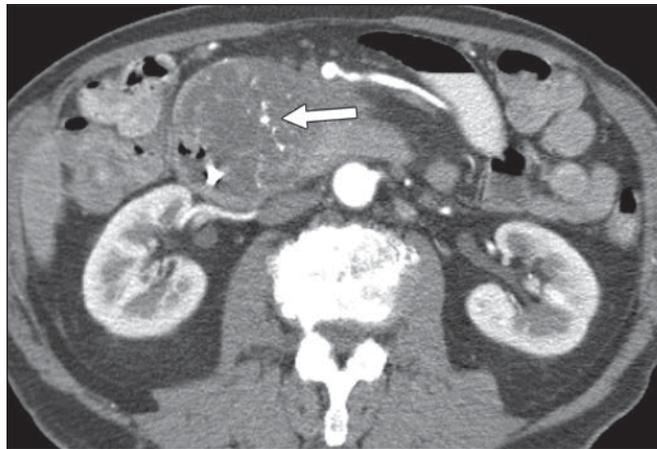


Fig. 7—61-year-old male dialysis patient with incidentally found pancreatic mass. **A**, Axial unenhanced CT image shows lobulated multicystic mass (*white arrow*) with central stellate calcification (*black arrow*). **B and C**, Axial (**B**) and coronal (**C**) T2-weighted images (single-shot fast spin-echo) confirmed multicystic nature of mass (*white arrow*) with central calcification appearing as low-signal-intensity focus (*black arrow*).



A



B

Fig. 8—Two middle-aged women with surgically proven mucinous cystic neoplasms.
A, 47-year-old woman. Axial contrast-enhanced CT image shows well-circumscribed fluid-attenuation mass in pancreatic body and tail with peripheral eggshell calcification (*arrow*).
B, 54-year-old woman. Axial contrast-enhanced CT image shows fluid-attenuation lobulated pancreatic head mass with septal calcifications (*arrow*).

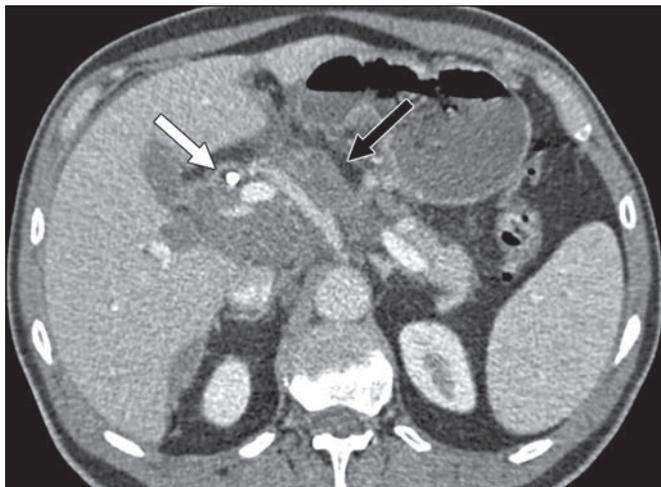


Fig. 9—52-year-old man with ductal adenocarcinoma. Axial contrast-enhanced CT image shows locally advanced pancreatic carcinoma (*black arrow*) encasing multiple vessels with few calcifications (*white arrow*).

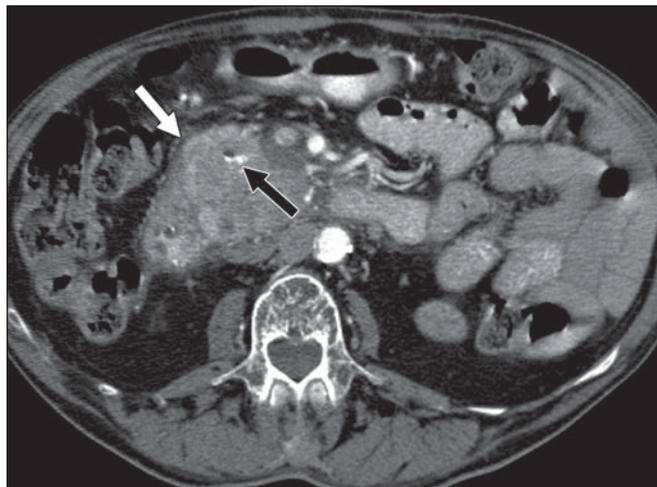


Fig. 10—57-year-old man with acinar cell carcinoma. Axial contrast-enhanced CT image shows large well-circumscribed heterogeneously enhancing mass (*white arrow*) involving head and uncinate process of pancreas and containing dystrophic calcifications (*black arrow*). Findings are consistent with acinar cell carcinoma.

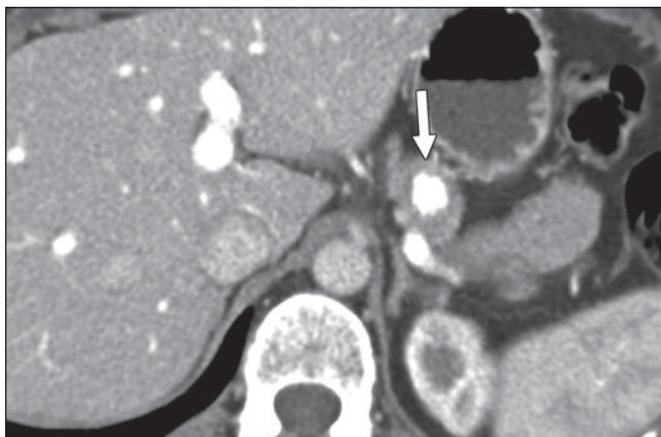
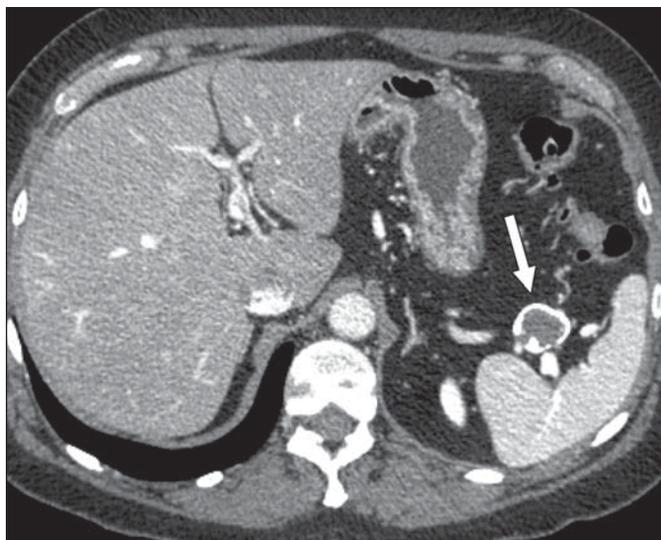
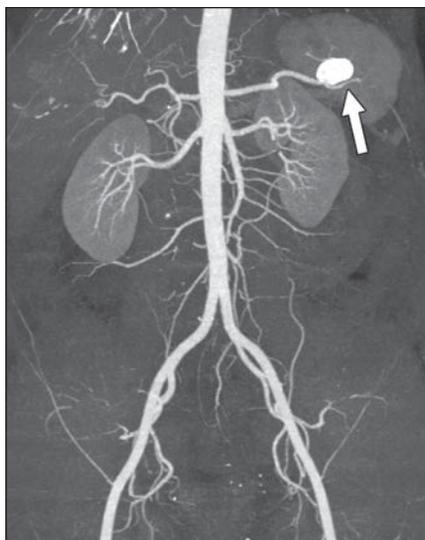


Fig. 11—47-year-old woman with osteosarcoma. Axial contrast-enhanced CT image shows small calcified focus within pancreatic body representing metastatic deposit (*arrow*) secondary to osteosarcoma.

CT of Pancreatic Calcifications

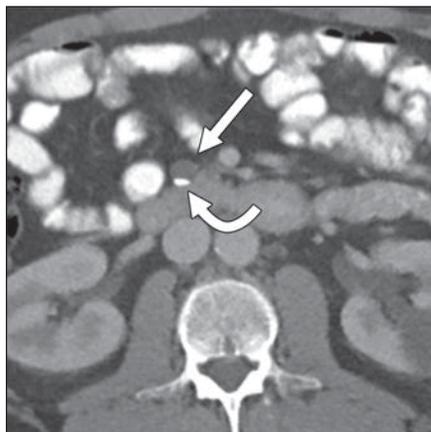


A



B

Fig. 12—Vascular calcification mimicking calcified pancreatic lesion. **A** and **B**, Axial contrast-enhanced CT image (**A**) and volume-rendered CT angiogram (**B**) show calcified small aneurysm (*arrow*) involving splenic artery mimicking mass at pancreatic tail.



A



B

Fig. 13—58-year-old man with history of chronic pancreatitis. **A**, Axial contrast-enhanced CT image (**A**) shows small cystic lesion (*straight arrow*) representing pseudocyst with layering of high-attenuation material (*curved arrow*) representing milk of calcium. **B**, Axial T2-weighted fast spin-echo MR image shows cyst as area of high signal intensity (*straight arrow*) and milk of calcium as area of low signal intensity (*curved arrow*).

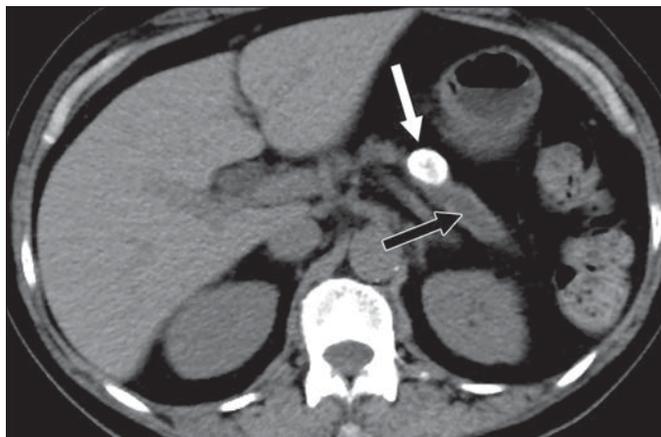


Fig. 14—51-year-old man with pancreatic duct calcified calculus. Axial unenhanced CT image shows calcified calculus (*white arrow*) within pancreatic duct causing marked dilatation of distal pancreatic duct (*black arrow*), which can mimic enhancing mass at contrast-enhanced CT.

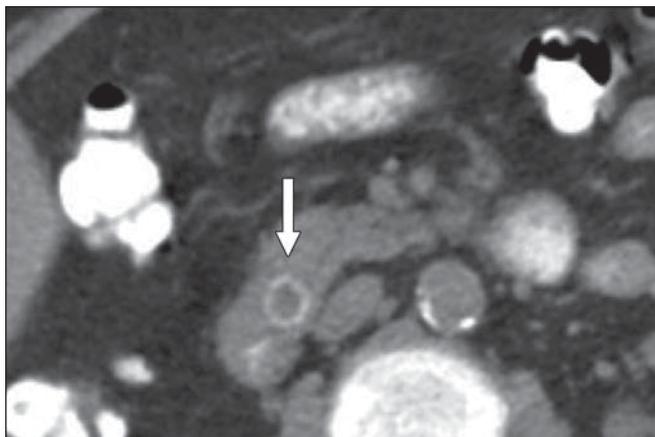


Fig. 15—78-year-old woman with common bile duct calculus. Axial unenhanced CT image shows calculus with calcified rim (*arrow*) within common bile duct, which can mimic rim-enhancing mass on contrast-enhanced CT images.