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The diagnostic challenge of the sequelae of acute pancreatitis on CT imaging: a pictorial essay

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Abstract

Purpose: The purpose of the study was to present a pictorial review of the long-term sequelae of acute pancreatitis on CT imaging as these findings can cause diagnostic confusion in the absence of a proper clinical history and/or prior CT imaging.

Methods: We retrospectively identified 81 patients who had an episode of acute pancreatitis with diagnostic findings on CT and also underwent one or more follow-up CT scans at least 1 month beyond the acute episode. The residual findings on all follow-up CT scans were tabulated, including the time interval since the initial bout of acute pancreatitis.

Result: Residual inflammatory changes were present in 19.8% of cases, with a median time period lasting 86 days since the initial episode of acute pancreatitis. Residual fluid collections were seen in 27.2% and persisted for a median of 132 days. Three patients had residual solid-appearing inflammatory masses, which could be mistaken for neoplasms. Other long-term sequelae were also tabulated, including pancreatic ductal dilatation, pancreatic atrophy, new or increased pancreatic calcifications, biliary tract dilatation, central portal venous occlusion, and pseudoaneurysm formation. These residual findings and long-term complications are presented as a pictorial essay.

Conclusion: Recognizing the spectrum of residual findings of acute pancreatitis, some of which can be long term, is important in the correct interpretation of a pancreatic CT. These findings can mimic acute pancreatitis or a pancreatic/peripancreatic neoplasm and often cause diagnostic confusion, especially in the absence of prior CT imaging.

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Key words: Acute pancreatitis—Chronic pancreatitis—Pseudocyst—Residual peripancreatic inflammation—Residual fluid collections—Computed tomography (CT)

Acute pancreatitis (AP) can be divided into interstitial edematous pancreatitis (AIEP, 70–80%) and necrotizing pancreatitis (ANP, 20–30%). AIEP has a mild, self-limiting clinical course lasting usually no longer than 2 weeks. ANP has a more severe course lasting weeks to months leading to many complications including pancreatic/peripancreatic inflammation, necrosis, and fluid collections that may be accompanied by multi-organ failure [1–3]. CT plays a critical role after the first week in diagnosing the morphological complications of the disease and helps identify patients who need intervention [4].

The complications of acute pancreatitis involve the evolutionary changes of pancreatic inflammation and necrosis. These complications can lead to focal or diffuse pancreatic atrophy, exocrine and/or endocrine insufficiency of the gland [5], obstruction of the biliary tract [6], or involvement of the duodenum or transverse colon resulting in bowel obstruction [7, 8]. If the inflammatory changes affect the vasculature, complications such as thrombotic occlusion of the central portal venous system [9] or pseudoaneurysms of adjacent arteries, particularly the splenic artery, can occur [10].

While the short-term sequelae of acute pancreatitis are well known, the persistence of inflammatory changes, fluid collections, or inflammatory masses several weeks to months or even years beyond the acute course of the disease can cause diagnostic confusion, especially without a proper clinical history and/or prior CT imaging for comparison. For example, residual inflammation can mimic acute pancreatitis in a patient with a clinically resolved bout of pancreatitis. Additionally, a residual

fluid collection or soft tissue mass can mimic a pancreatic/peripancreatic neoplasm [11, 12]. This pictorial review will address the spectrum of these residual findings and long-term complications of acute pancreatitis.

Patients and methods

Patients admitted to the George Washington University Hospital with the diagnosis of acute pancreatitis over a 10 years period from 2004 to 2014 were identified from the medical records following IRB approval. Patients were included in the study if they had (1) an abdominal/pelvic CT scan with findings consistent with acute pancreatitis and (2) a follow-up CT scan performed at least 1 month after the first scan.

Eighty-one patients were identified for evaluation, including 61 males and 20 females. The average age of the patients was 48 years with a range of 21–84 years. A total of 334 CT scans were reviewed, of which 299 were performed using IV contrast. CT scans were performed for inpatient or outpatient studies from 1 month up to 10 years following the initial bout of acute pancreatitis. Patients with CT scans performed long after the initial bout of AP had a normal serum lipase, and the CT scans were being performed for other clinical indications, usually abdominal pain. The average number of CT scans performed on each patient was 4.1. This average included one patient who had 48 scans over 6 years due to multiple complications from necrotizing pancreatitis. Excluding this patient, the average number of CT scans performed per patient was 2.5.

All patients had acute pancreatitis based on clinical parameters, an elevated serum lipase, and CT findings consistent with the diagnosis. Acute pancreatitis on the first CT scan was defined by the presence of pancreatic/peripancreatic inflammation and fluid collections, and the location and severity of these findings were documented. All contrast-enhanced CT scans were evaluated for pancreatic necrosis as defined by the lack of enhancement of a portion of the pancreatic parenchyma. The presumed etiology of the initial episode of acute pancreatitis was determined via chart review.

The follow-up CT scans performed at least 1 month after the initial CT scan(s) were evaluated for the presence of the following residual findings:

1. Pancreatic swelling
2. Inflammatory changes involving the pancreas and peripancreatic soft tissues (anterior pararenal space, transverse mesocolon, small bowel mesentery, extension into the pelvis)
3. Fluid collection(s)
4. Solid-appearing inflammatory mass
5. Pancreatic atrophy
6. New or increased pancreatic calcifications
7. Pancreatic ductal dilatation >4 mm
8. Biliary tract dilatation
9. Thrombosis/occlusion of the portal venous system (main portal vein, splenic vein, superior mesenteric vein)
10. Pseudoaneurysm.

On the follow-up CT scans, it was not always possible to distinguish between the different types of fluid collections found more than 1 month after the initial CT, including pseudocysts and walled-off necrosis (WON). Pseudocysts tend to be round to oval in shape with a well-defined wall, while WON is less well defined and is usually multiloculated. For the purposes of this study, they were combined together as "fluid collections." However, distinct types of fluid collections do exist [3].

The time periods between the initial CT scan and subsequent follow-up studies were noted (months, years). These data provided a rough estimate of the duration these findings took to appear and how long they persisted.

Results

The initial episode of acute pancreatitis was secondary to alcohol in 49 patients (60.5%) and gallstones in 13 patients (16%). Other less common etiologies, as Table 1 lists, including hypertriglyceridemia, medication-induced, pancreas divisum, ERCP, and autoimmune, occurred much less frequently. Nine patients (11.1%) did not have a clear etiology for acute pancreatitis. 47 patients (58.0%) had AIEP with CT evidence of pancreatic swelling and/or peripancreatic inflammatory changes. 14 patients (17.3%) had acute-on-chronic pancreatitis with peripancreatic inflammation superimposed on pancreatic calcifications, pancreatic atrophy, and/or pancreatic ductal dilatation. The remaining 20 patients (24.7%) were diagnosed with ANP and had pancreatic necrosis either at the time of the initial scan or on follow-up CTs. Table 2 summarizes the spectrum of findings that occurred on these follow-up CT scans at least 1 month following the initial CT diagnosis of acute pancreatitis. The distribution of these findings comparing alcohol-induced pancreatitis and gallstone pancreatitis is summarized in Table 3. Although there were significantly more cases of alcohol pancreatitis, the data overall suggest that more long-term sequelae occur with alcohol.

Table 1. Etiology of the acute episode of pancreatitis

Etiology	Number	Percentage
Alcohol	49	60.5
Gallstone	13	16.0
Hypertriglyceridemia	3	3.7
Medication-induced	3	3.7
Pancreas divisum	2	2.5
ERCP	1	1.2
Autoimmune	1	1.2
No clear etiology	9	11.1

Table 2. Follow-up CT findings in patients ($N = 81$) with acute pancreatitis

Finding	Number	Percentage
1. Residual pancreatic swelling	5	6.2
2. Residual peripancreatic inflammatory changes	16	19.8
3. Residual fluid collections	22	27.2
4. Residual solid appearing inflammatory mass	3	3.7
5. Pancreatic atrophy	19	23.5
6. New/increased pancreatic calcifications	15	18.5
7. Pancreatic ductal dilatation	17	21.0
8. Biliary tract dilatation	4	4.9
9. Central portal venous occlusion	7	8.6
10. Pseudoaneurysm	1	1.2

Table 3. Distribution of findings comparing alcohol-induced and gallstone pancreatitis (number of cases)

	Alcohol	Gallstone
Total cases	49	13
Acute necrotizing pancreatitis	10	7
Acute-on-chronic pancreatitis	12	0
Residual inflammatory changes	8	5
Residual pancreatic swelling	5	0
Residual fluid collections	13	5
Residual inflammatory mass	2	0
New/increased calcifications	13	0
Central portal venous occlusion	5	0
Pancreatic atrophy	14	2
Pancreatic ductal dilatation	13	3

Table 4. Length of time (days) that residual findings were present

Finding	Median	Range
Residual peripancreatic inflammatory changes ($N = 16$)	80	22–373
Residual pancreatic swelling ($N = 5$)	73	31–143
Residual pseudocyst(s) and/or walled-off necrosis ($N = 22$)	132	14–640

Pictorial essay

Residual pancreatic swelling, peripancreatic inflammatory changes, and solid-appearing inflammatory mass

Pancreatic swelling was present in 49 patients (60.5%) during the acute episode, but only persisted in five patients (6.2%) on follow-up scans. The median number of days of residual pancreatic swelling was 73 days (Table 4).

Peripancreatic inflammation was present in all patients ($N = 81$) on the initial CT scan. On follow-up, residual inflammatory changes (Figs. 1, 2) were seen in 16 patients (19.8%) and persisted on average for 80 days (Table 4). By themselves, these residual findings could simulate acute pancreatitis or a recurrence, even though the patient has made a full clinical recovery with a normal serum lipase. These imaging findings require correlation with the patient's serum lipase if prior scans are not available.

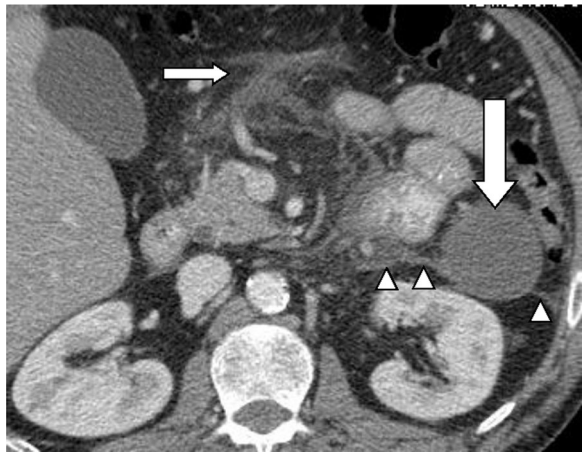

Fig. 1. Clinically resolved acute pancreatitis with residual peripancreatic inflammatory changes in the region of the transverse mesocolon (*small arrow*) and left anterior pararenal fascia (*arrowheads*) 5 weeks after the acute episode. A loculated fluid collection (*large arrow*) is also present along the left pararenal fascia.

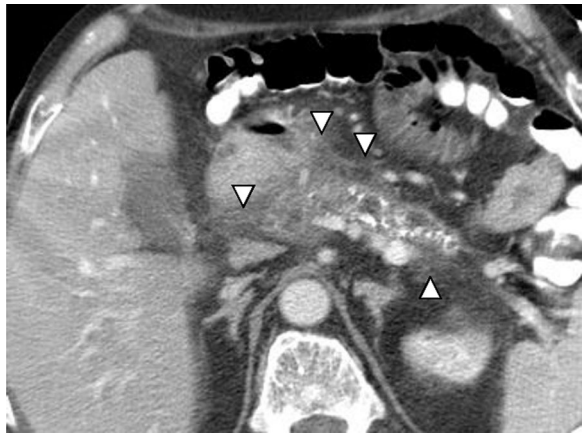

Fig. 2. Chronic pancreatitis with persistent peripancreatic stranding around the head and body of the pancreas (*arrowheads*) 4 months after an episode of acute pancreatitis. The patient had clinically recovered.

An inflammatory solid-appearing mass, or "pseudomass," may persist in a region of prior peripancreatic inflammatory change. This was present in three patients (3.7%) and may be adjacent to or away from the pancreas, in the anterior pararenal space, transverse mesocolon, or small bowel mesentery (Fig. 3). One patient with follow-up scans 3 years later demonstrated a stable but persistent pseudomass measuring 40 Hounsfield units along the anterior aspect of the head of the pancreas at the root of the transverse mesocolon (Fig. 4). If the initial scans were not available for comparison, these findings could be misinterpreted as a possible malignancy, as occurred in this case. Occasionally, these residual inflammatory masses can be found in unusual

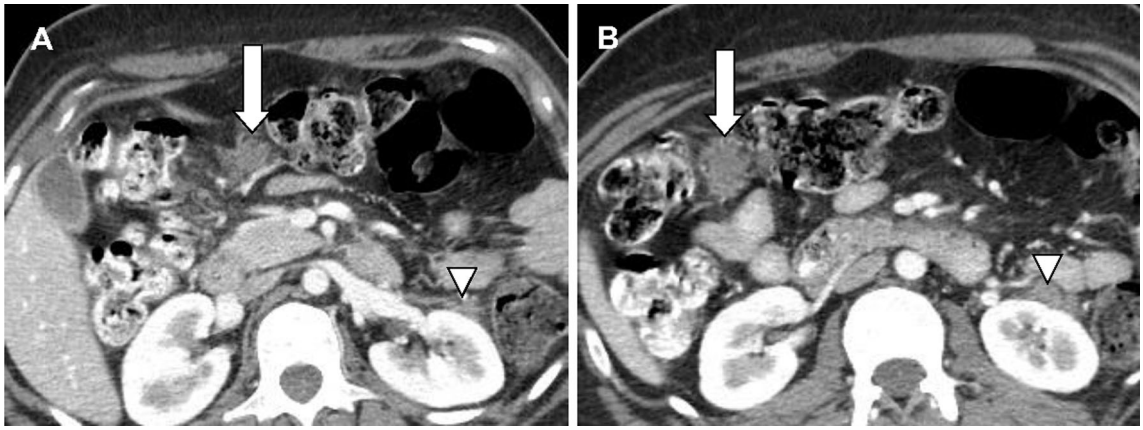

Fig. 3. 2 months after acute necrotizing pancreatitis (**A**, **B**), there are two small residual inflammatory soft tissue masses in the transverse mesocolon (*arrows*). Focal inflammatory changes are also present along the anterior aspect of the left kidney (*arrowheads*).

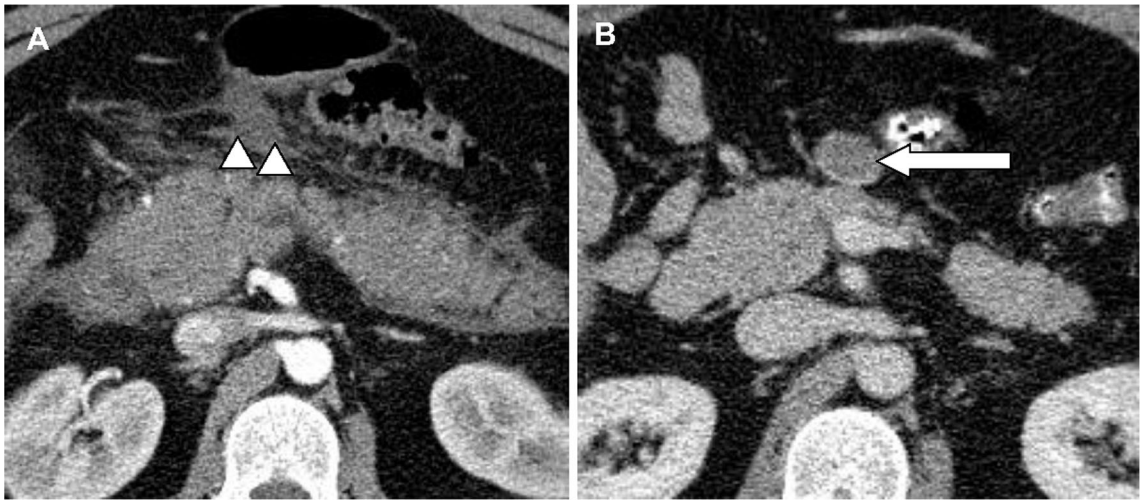

Fig. 4. Initial episode of acute pancreatitis (**A**) with peripancreatic inflammatory stranding and focal inflammatory change anteriorly (*arrowheads*). 3 years and 2 months later, there is a residual "pseudomass" measuring 40 Hounsfield units anterior to the pancreatic body in the area of prior inflammation (**B**). No biopsy was performed, and the mass had not changed on a follow-up CT scan done 1 year later.

places away from the pancreas, such as the right posterior perinephric space (Fig. 5).

Residual pseudocyst(s) and/or walled-off necrosis (WON)

Residual fluid collections representing pseudocysts or WON were found in 22 patients (27.2%) and can persist for months after the initial acute episode of pancreatitis (Figs. 6, 7). They can be single or multiple and can occur in the pancreas and surrounding tissues, anterior pararenal space, wall of the duodenum, or even in the spleen. Over time, some of these can resolve only to have another fluid collection appear in a different location (Fig. 7). They tended to persist for long time periods, with a median time period of 132 days (Table 4). As these residual fluid collections can mimic a cystic pancreatic neoplasm, comparison to prior studies is critical.

Uncommon complications of residual fluid collections were observed, and can occur weeks to months after acute pancreatitis. Some fluid collections can demonstrate focal areas of contrast extravasation on follow-up scanning, indicating active hemorrhage, and such patients can present with an acute abdomen (Fig. 8). They can bleed into the stomach and peritoneal cavity, and may require embolization or even surgical intervention [13, 14].

Two patients developed calcifications within the wall of a fluid collection that arose in an area of WON (Fig. 9). Calcification in a pseudocyst/pancreatic fluid

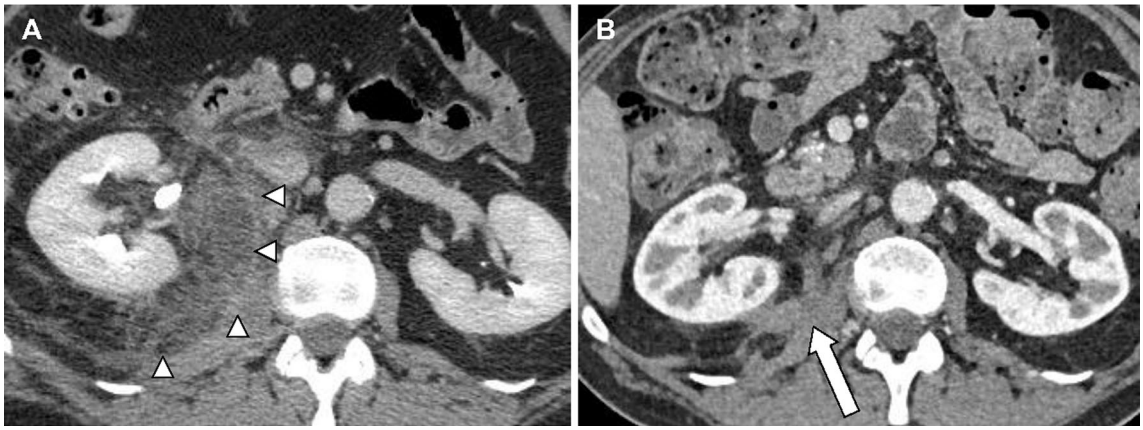

Fig. 5. Acute pancreatitis with inflammatory change (**A**) extending through the medial right perinephric space into the posterior pararenal space (*arrowheads*). Residual inflammatory changes are still present in the right posterior perinephric space 8 months later (*arrow*) **(B)**.

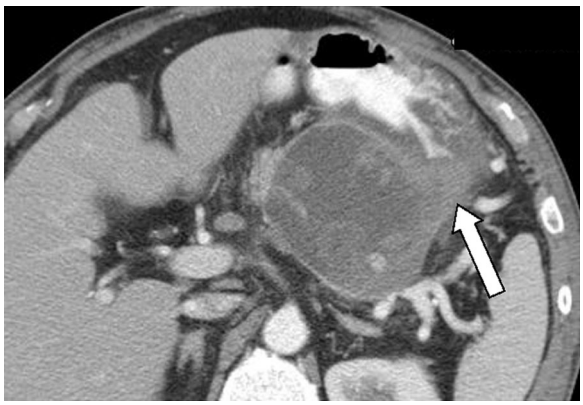

Fig. 6. Persistence of a complex fluid collection in the pancreatic tail 27 weeks after the start of an episode of acute pancreatitis. Notice the involvement of the adjacent posterior wall of the stomach (*arrow*) representing a point of possible marsupialization.

collection is extremely uncommon [15].When present, it raises the possibility of a mucinous cystic pancreatic neoplasm as 10% to 25% of these tumors have calcifications [12]. In our patients, the distinction between the two was easy as we had followed these patients over time and knew the calcifications represented post-inflammatory change.

Another patient had a persistent fluid collection at 1 year with new internal gas and persistent surrounding inflammatory changes (Fig. 10). In the acute phase, the presence of gas in an area of necrosis may indicate the presence of infected necrosis with a sensitivity and specificity of 47.3% and 84.0%, respectively [16]. Since this patient was not in the acute phase of pancreatitis and had no clinical or laboratory findings of infection, this finding was most consistent with sterile necrosis. A follow-up scan 4 months later showed the collection to have decreased in size, and the gas was no longer present. Spontaneous marsupialization of a pseudocyst into the stomach or intestine can also lead to air within a pancreatic pseudocyst.

New or increased pancreatic calcifications

Pancreatic calcifications are a well-known complication of recurrent bouts of alcohol-induced acute pancreatitis and were found in 15 patients (18.5%) in this review (Table 2). The calcifications appeared in as little as 221 days after a bout of AP but usually take much longer (Table 5). In our patients, it took an average of nearly 2 years for the calcifications to appear or increase in size/number. Calcifications may also decrease on long-term follow-up [17].

Pancreatic atrophy

Pancreatic atrophy can be focal or diffuse and occurred in 19 patients (23.5%). Atrophy developed as soon as 67 days following the acute episode of pancreatitis with a median time of 394 days (Table 5). While chronic pancreatitis is associated with atrophy 54% of the time [18], atrophy can occur as the result of pancreatic parenchymal necrosis [19]. One patient had ANP with lack of enhancement in the pancreatic body and adjacent tail (Fig. 11). 7 weeks later, the necrotic portion of the pancreas developed internal gas, followed by a fluid collection in the same region 5 months later. This fluid collection was most likely due to discontinuity of the pancreatic duct (Fig. 11C) [19]. 2 years later, this area went on to focal atrophy of the involved pancreatic parenchyma. A similar but more severe case of ANP with WON demonstrated diffuse pancreatic atrophy on a follow-up CT scan 1 year later (Fig. 12).

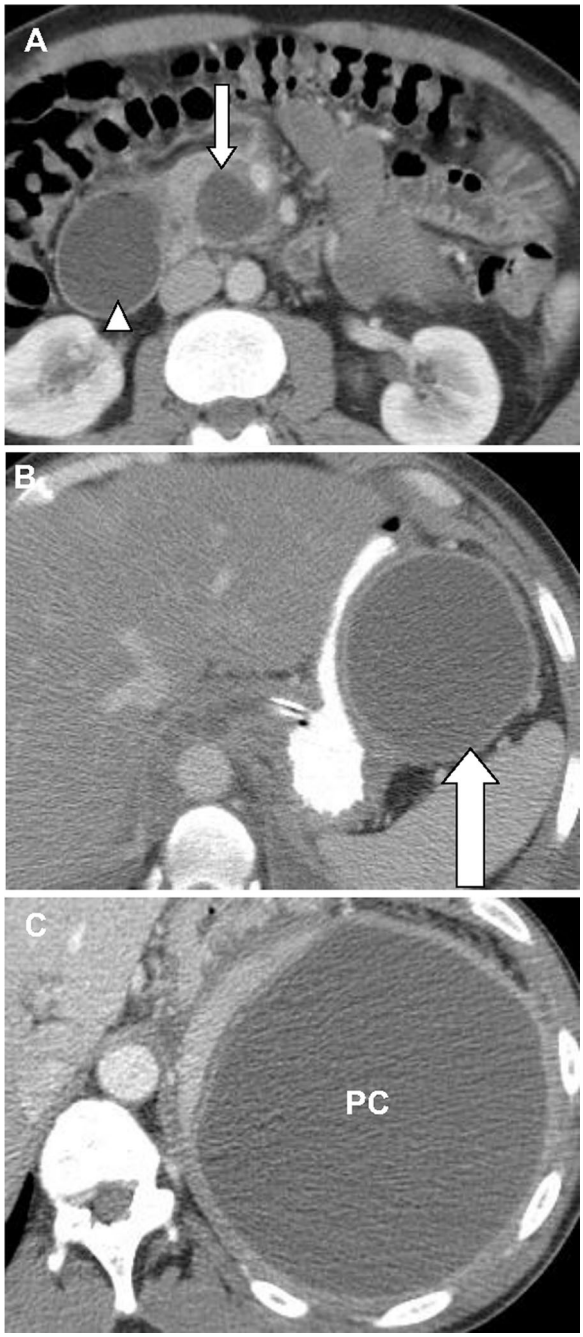

Fig. 7. Residual pancreatic (*arrow*) and peripancreatic (*arrowhead*) fluid collections 2 months after an episode of acute pancreatitis **(A)**. 7 weeks later **(B)**, there is a residual extrapancreatic fluid collection lateral to the stomach (*large arrow*). 6 weeks later **(C)**, an intrasplenic pseudocyst (PC) developed.

Pancreatic ductal dilatation

Pancreatic ductal dilatation can appear over time, and is seen with chronic pancreatitis 68% of the time [18]. In our study, main pancreatic ductal dilatation occurred in 17 patients (21%). One patient developed diffuse pancreatic ductal dilatation related to acute pancreatitis

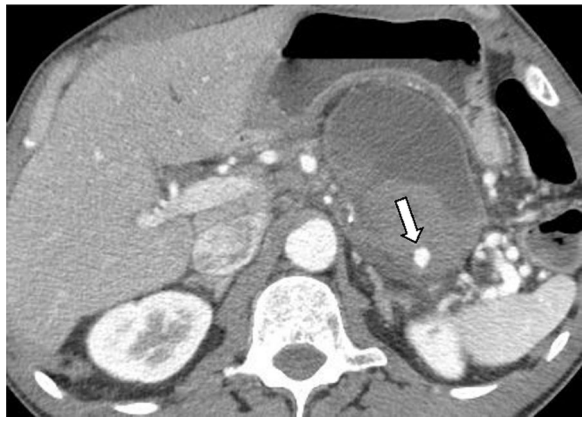

Fig. 8. Fluid collection 3 months after acute pancreatitis with a focus of IV contrast extravasation (*arrow*) surrounded by blood clot, indicative of acute hemorrhage.

9 weeks earlier (Fig. 13). Another patient had dilatation of a small segment of the pancreatic duct in the body in the absence of pancreatic calcifications. Follow-up studies were performed on this patient due to the worry that the ductal dilatation could be secondary to a small intraductal papillary mucinous neoplasm (IPMN) [20]. This proved not to be the case, and the duct remained unchanged on a CT performed 7 years later for unrelated reasons (Fig. 14). It should be noted that in the absence of pancreatic calcifications, pancreatic ductal dilatation in conjunction with common bile duct obstruction is associated with a high likelihood of carcinoma (79%) [21].

Biliary dilatation

Biliary tract dilatation can occur in AP from edema, necrosis, or a fluid collection. With repeated bouts of AP, fibrosis occurs and constricts the duct. A common bile duct stricture may be found as an incidental finding on ERCP in asymptomatic patients (pain, jaundice) with chronic pancreatitis [22]. On ERCP, the duct has a smooth, tapered appearance with no evidence of mass effect or shouldering to suggest carcinoma. There will also be no evidence of encasement of adjacent vessels or metastasis. In our group of patients, four (4.9%) developed biliary tract dilatation on follow-up CT scans. One patient with multiple follow-up studies over several years developed chronic pancreatitis with diffuse parenchymal calcifications and pancreatic ductal dilatation (Fig. 15). The calcifications in the head resulted in common bile duct obstruction with intra-hepatic biliary tree dilatation. A common bile duct stone was suspected on the CT scan; however, no obstructing stone was found on an ensuing ERCP (Fig. 15). If the patient is jaundiced, a biliary stent may be required and be used as a bridge to definitive surgery [23].

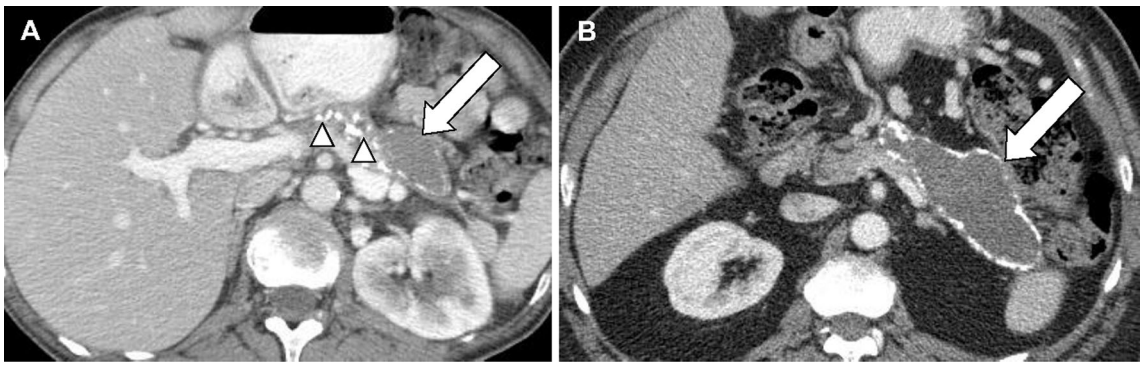

Fig. 9. Two different patients with hypodense collections/pseudomasses (*arrows*) involving the body and tail with calcified walls resulting from acute necrotizing pancreatitis 5 months ago **(A)** and 1 year ago **(B)**. The patient in **A** also had had chronic pancreatitis with pancreatic parenchymal calcifications (*arrowheads*).

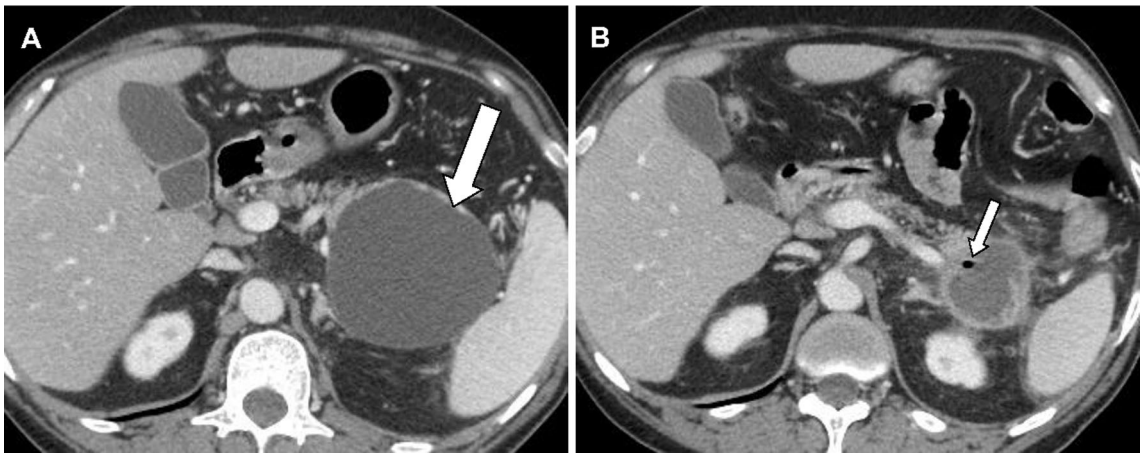

Fig. 10. Following an episode of acute pancreatitis, there is a residual fluid collection (*large arrow*) in the tail of the pancreas **(A)**. 1 year later, the fluid collection had become smaller but had developed a focus of gas (*small arrow*), and there were mild surrounding inflammatory changes **(B)**. The patient had no clinical or laboratory findings to suggest AP or an abscess.

Table 5. Length of time (days) to develop long term sequelae of acute pancreatitis

Finding	Mean	Median	Range
New or increased calcifications ($N = 15$)	1097	732	221–3734
Central portal venous occlusion ($N = 7$)	217	136	0–640
Pancreatic ductal dilatation ($N = 17$)	460	0	0–3734
Pancreatic atrophy ($N = 19$)	771	394	67–3226

Thrombosis of the central portal venous system

Thrombosis of the central portal venous system may occur at the time of the acute episode, or it may appear weeks later [2]. In one study, the median time to detection was 17 days with a range of 11–40 days [9]. It occurs in 18–35% of patients, especially in ANP (50%) [9, 24]. The splenic vein (86–93%) is most commonly involved, followed by the portal vein (34–36%) and superior mesenteric vein (24–27%) [25]. Involvement of one vein often leads to involvement of another, e.g., thrombosis of the splenic vein or superior mesenteric vein with extension into the main portal vein. In our series, seven patients (8.6%) developed this finding. In one patient, a thrombus of the splenic vein was present at the time of the initial episode of acute pancreatitis (Fig. 16). 1 month later, the thrombus extended into the main portal vein on a background of ongoing residual inflammatory changes and fluid collections. While the thrombus may resolve, persistent complete occlusion of the vein can lead to cavernous transformation of the main portal vein and ultimately portal hypertension with varices, gastrointestinal bleeding, splenomegaly, and ascites [25]. Figure 17 shows the sequelae of splenic vein occlusion with development of gastroepiploic collaterals.

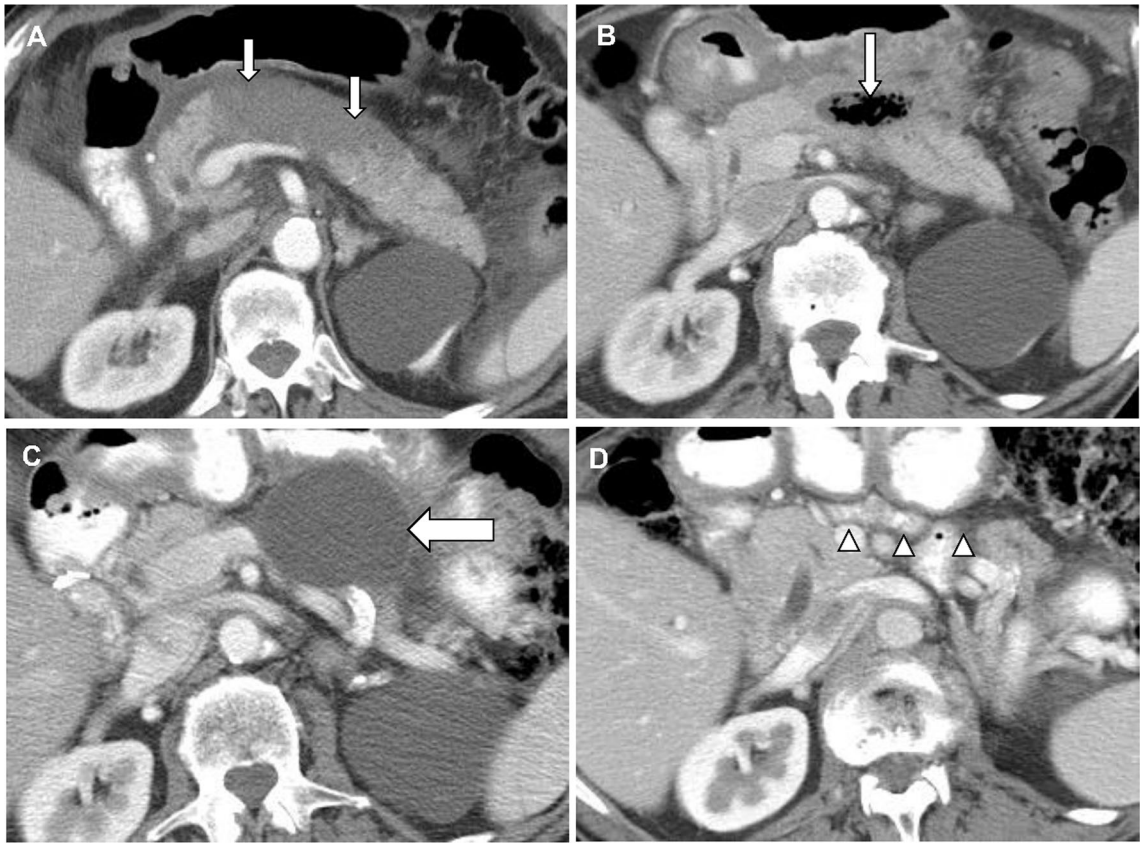

Fig. 11. Acute necrotizing pancreatitis **(A)** with lack of enhancement in the pancreatic body and tail (*small arrows*) with peripancreatic inflammatory changes. 7 weeks later **(B),** the area of necrosis contained gas (*arrow*). The patient had no symptoms of an abscess. 5 months later **(C)**, there is a fluid collection in this area (*large arrow*), and on a 2 years follow-up **(D),** there is atrophy of the body and tail (*arrowheads*).

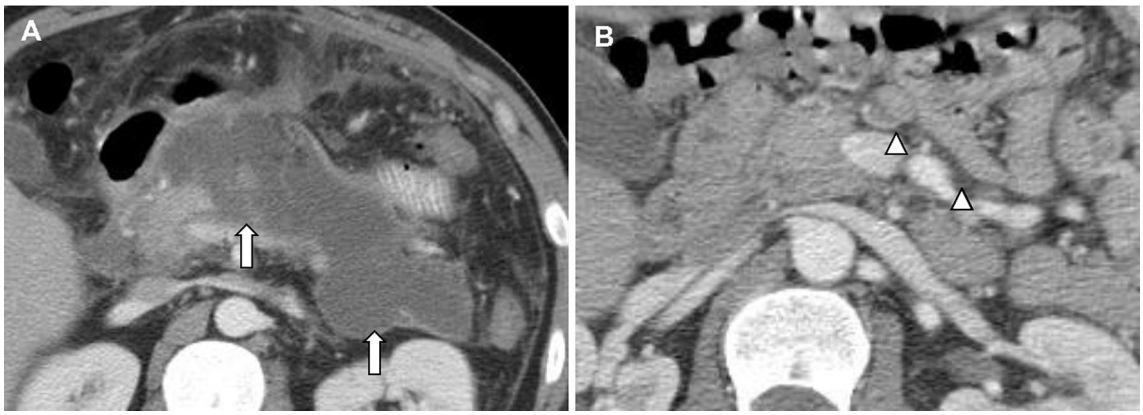

Fig. 12. Acute necrotizing pancreatitis (**A**) with walled-off necrosis involving the body and tail of the pancreas (*arrows*). 1 year later (**B**), there is parenchymal atrophy of the body and tail (*arrowheads*).

Pseudoaneurysm formation

Pseudoaneurysm formation may involve arteries that are adjacent to the pancreas [1, 26], usually the splenic artery (40%), branches of the gastroduodenal artery (30%), or pancreaticoduodenal artery (20%) [25]. The local inflammation, infected or non-infected, with proteolytic activity of the pancreatic enzymes weakens the arterial wall, leading to pseudoaneurysm formation. In our series, one patient developed a splenic artery pseudoaneurysm adjacent to the pancreatic tail that persisted after a bout

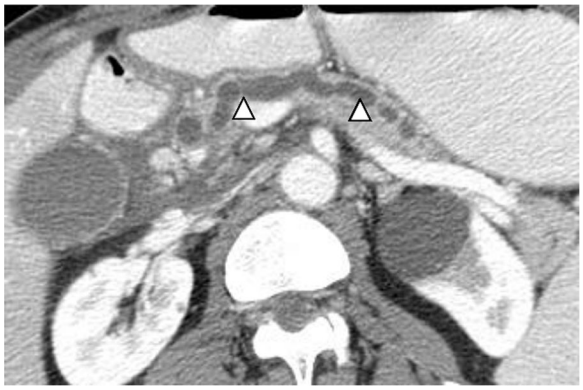

Fig. 13. Development of diffuse pancreatic ductal dilatation (*arrowheads*) following a bout of acute necrotizing pancreatitis nine weeks earlier.

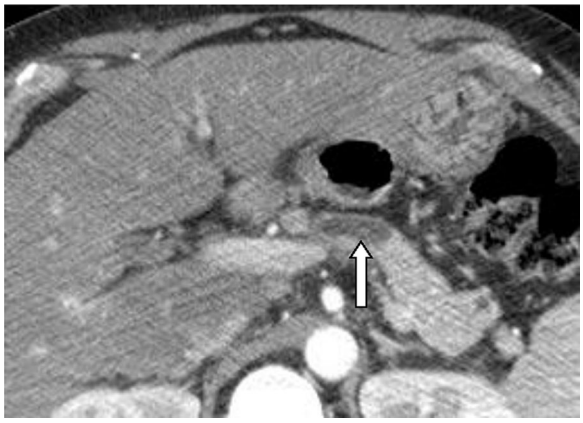

Fig. 14. Chronic pancreatitis with stable dilatation of the pancreatic duct (*arrow*) for 7 years in the absence of pancreatic calcifications, mimicking an intraductal papillary mucinous neoplasm of the main duct or an obstructing neoplasm. There was no evidence of a pancreatic head mass.

of acute pancreatitis (Fig. 18). Pseudoaneurysms can rupture into a pseudocyst, into the gastrointestinal tract, often the stomach, or into the peritoneal cavity. Clinically, the patient presents with acute severe abdominal pain and symptoms of blood loss. Acute hemorrhage in pancreatitis is very uncommon (1.3%) and is due to rupture of a pseudoaneurysm (61%) or bleeding into an area of pancreatic necrosis (19.5%) [27, 28]. On a contrast-enhanced CT scan, there may be active extravasation of contrast at the site of a pseudoaneurysm, usually requiring prompt treatment with arterial embolization (75%) or surgical ligation (25%) [25, 29].

Summary

Recognizing the spectrum of residual sequelae of acute pancreatitis on CT imaging is crucial in correctly interpreting a CT scan of the pancreas, especially if no prior CT scans are available for comparison. Residual inflammatory changes in the adjacent pancreatic soft tissues can persist for months. This can lead to the erroneous CT diagnosis of ongoing acute pancreatitis, when in fact the patient has clinically fully recovered. Without prior studies, interpreting a CT that shows peripancreatic stranding requires correlation with the clinical findings and serum lipase.

It is important to be aware that residual inflammation can appear as fascial thickening or as a "pseudomass." Inflammation can occur in the area of the anterior pararenal fascia, transverse mesocolon, or adjacent small bowel mesentery. Residual fluid collections (pseudocysts, WON) following an acute episode of pancreatitis can remain for months or years and can be single or multiple. The collections may resolve in one area only to have another fluid collection appear over time in a different location. Development of gas within the fluid can simu-

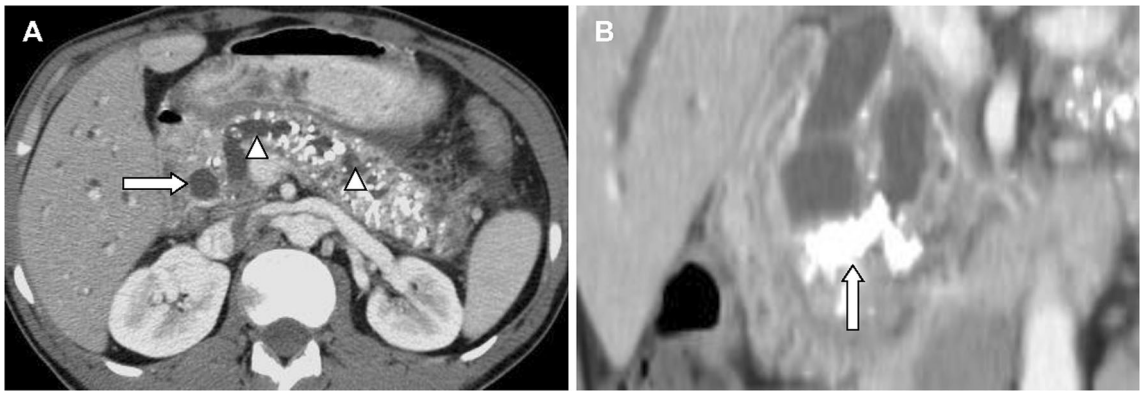

Fig. 15. Axial (**A**) and coronal (**B**) images demonstrate chronic pancreatitis with diffuse pancreatic calcifications, dilatation of the main pancreatic duct (*arrowheads*), and dilatation of the common bile duct (*arrow*) and intra-hepatic bile ducts due to obstruction in the pancreatic head. The calcifications in the head mimicked an obstructing common bile duct stone (*arrow*) on the coronal images (**B**). No stone was present on a follow-up ERCP.

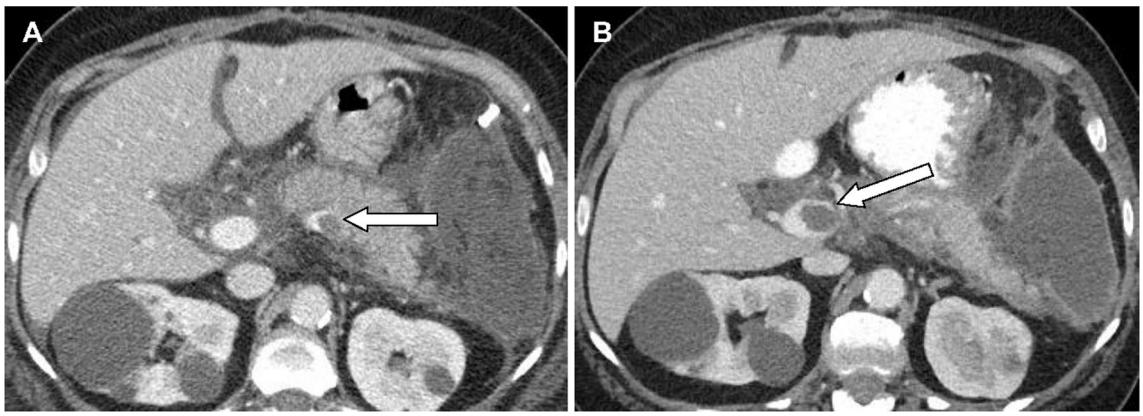

Fig. 16. Acute pancreatitis (**A**) with thrombus in the splenic vein (*arrow*). 1 month later (**B**), thrombus extended into the main portal vein (*arrow*).

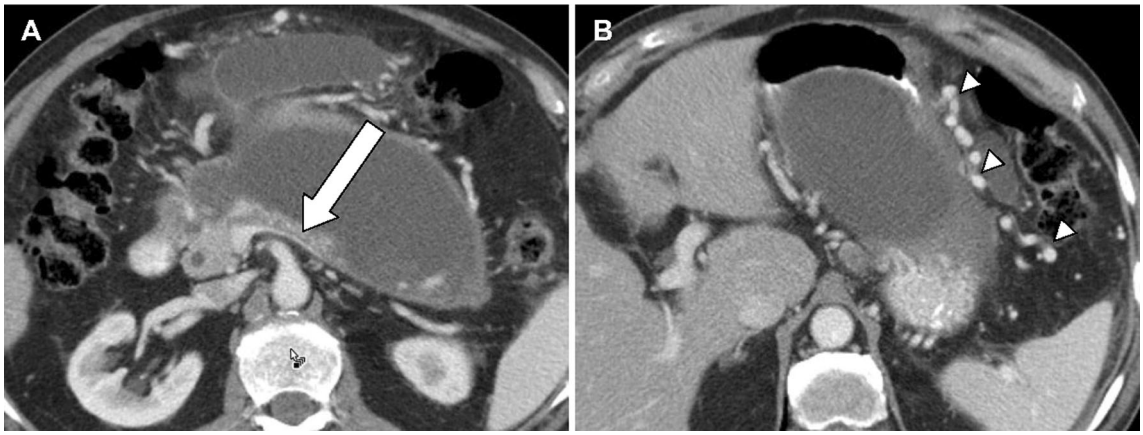

Fig. 17. Obliteration of the splenic vein (*arrow*) (**A**) and development of gastroepiploic collateral veins along the gastric wall (*arrowheads*) 3 months following acute necrotizing pancreatitis (**B**).

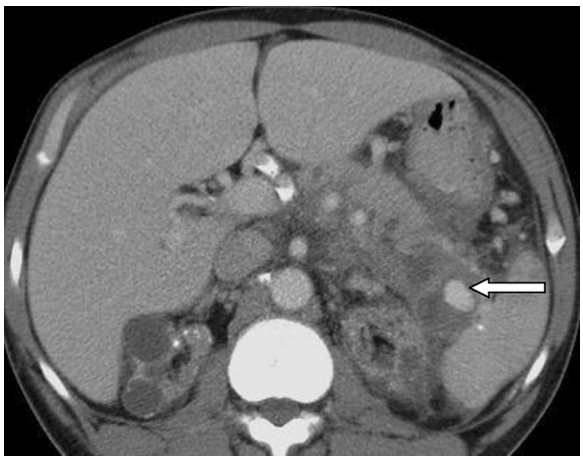

Fig. 18. Acute pancreatitis with development of a splenic artery pseudoaneurysm (*arrow*) on follow-up. This patient had end-stage renal disease.

late an abscess. Similarly, pancreatic necrosis can evolve into a gas and/or fluid-containing collection before going on to focal or diffuse pancreatic atrophy. Calcification within the wall of a fluid collection can lead to the mistaken diagnosis of a cystic pancreatic neoplasm. Rarely, bleeding into the collection or into the stomach/peritoneal cavity can cause an acute abdomen with clinical signs of blood loss requiring immediate embolization or surgery.

Biliary tract dilatation and focal/diffuse dilatation of the pancreatic duct can be the result of chronic pancreatitis even in the absence of parenchymal calcifications, leading to the search for an obstructing neoplasm or a common bile duct stone. ERCP may be required to resolve these issues. In the absence of prior studies for comparison with an obstructing neoplasm or mucinous cystic neoplasm in the differential diagnosis, a follow-up CT, at a minimum, may be warranted to establish stability. If indicated, further work-up may be necessary.

AP can also cause thrombosis of the central portal venous system leading to portal hypertension, gastroesophageal varices, and bleeding. Likewise, bleeding can also result from a pseudoaneurysm of one of the peripancreatic arteries, usually the splenic artery.

This study is limited due to its retrospective nature and the fact that the follow-up CT scans were performed at varying intervals. Thus, the numbers quoted are rough estimates and have to be viewed in that light. Even so, the point has to be made that the sequelae of acute pancreatitis can cause diagnostic confusion in the absence of an accurate clinical history and/or a prior CT scan.

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Compliance with ethical standards

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Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. For this type of study formal consent is not required.

Informed consent Statement of informed consent was not applicable since the manuscript does not contain any patient data.

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