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Acute Pancreatitis Imaging in MDCT: State of the Art of Usual and Unusual Local Complications. 2012 Atlanta Classification Revisited

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A B S T R A C T

Acute pancreatitis is an inflammatory disease in which most common etiologies are biliary lithiasis and alcoholism. Acute pancreatitis can be classified into 2 groups according to its morphologic features: interstitial edematous pancreatitis and necrotizing acute pancreatitis. The prognosis of this group of diseases and its complications varies significantly and contrast-enhanced computed tomography is the imaging study of choice for the diagnosis and detection of complications. . In this review, we aim to summarize the changes introduced in the revised Atlanta classification and describe other usual and unusual local complications of acute pancreatitis that are not analyzed in that classification. We will also describe early detection signs and provide an accurate interpretation of complications on contrast-enhanced computed tomography that will lead to prompt management decisions which can reduce the morbidity and mortality of these patients.

Introduction

Acute pancreatitis is an inflammatory condition caused by intracellular activation and inappropriate extravasation of proteolytic enzymes causing destruction of the pancreatic parenchyma and peripancreatic tissues. The most common etiologies are biliary lithiasis and alcoholism. Less frequent causes include hypertriglyceridemia, hypercalcemia, drugs, autoimmune disease and parasitosis, among others. In Chile, approximately 75% of acute pancreatitis are caused by biliary lithiasis and 15%-20% by alcoholism, meanwhile in the United States, biliary lithiasis and alcohol are responsible for 40% and 35% of the cases, respectively.[1]

Contrast-enhanced computed tomography (CECT) is the imaging study of choice for the diagnosis and detection of complications of acute pancreatitis.[2-4] In 2012, a new classification for acute pancreatitis was proposed, addressing the most frequent local complications.[5]

The present study aims at summarizing the changes introduced in the revised Atlanta classification and describing other usual and unusual local complications of acute pancreatitis that are not analyzed in that classification.

Revised Atlanta Classification of Acute Pancreatitis

The 1992 Atlanta Symposium proposed a universal definition and classification for acute pancreatitis.[6] Even though the classification played an important role since its creation, the significant development in diagnostic imaging and the growing understanding of organ failure justified an update. In 2012,[7] the classification was reviewed by the Acute Pancreatitis Classification Working Group, who proposed changes related to diagnosis and phases of the disease. These changes redefined the radiological classification, introducing new concepts and removing others regarding local complications of acute pancreatitis.

The most important change introduced was the use of more specific terms to describe fluid collections and foci of necrosis within or around the pancreas. The Acute Pancreatitis Classification Working Group proposed the classification of pancreatic and peripancreatic fluid collections into 4 main categories according to elapsed time since the disease onset and the severity of acute pancreatitis[5] (summarized in Table 1).

Disease Progression Over Time

In acute pancreatitis, it is possible to distinguish 2 phases: an early phase, which lasts approximately 7 days, and a late phase. In the early phase, disease severity is related to systemic complications. While local complications may be identified, it is generally not necessary to document them by imaging examinations during the first week. The reason for this is that the presence and extent of pancreatic and peripancreatic necrosis may not be defined early by imaging during the first few days of disease, and false-negative studies searching for necrosis are not uncommon.[8] When necessary, a CECT 3-5 days after admission is more reliable in establishing the presence and extent of pancreatic necrosis. Second, the extent of morphologic changes and necrosis is not directly proportional to the severity of organ failure. Finally, even if imaging during the first week identifies the presence of peripancreatic fluid collection of pancreatic necrosis, in general no treatments are required for these conditions at that time.[9]

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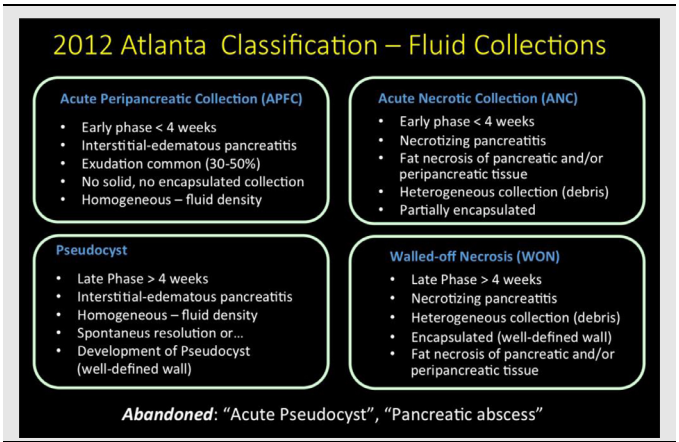
TABLE 1
2012 Atlanta classification—fluid collections



In the late phase of moderate or severe acute pancreatitis, local complications evolve more fully and can be determined by a CECT examination. At this phase, the need for treatment is determined from the basis of clinical progression of the disease, and treatment is guided by morphologic imaging findings. Therefore, CECT plays an important role in determining the therapeutic approach during this phase. Other imaging techniques such as MRI and ultrasonography can also be helpful in this late stage, either if CT is not an option or as an adjunct, but their description is outside the scope of this review.[10]

Imaging Evaluation of Acute Pancreatitis

CT Protocol

The CT scan should be performed at least 3-5 days after the onset of clinical symptoms, since necrosis begins after 48-72 hours from the onset. The protocol differs depending on the local preference, but it should include the use of iodinated contrast agents, with acquisition of arterial (pancreatic phase) and venous phases. The protocol used in this study is summarized in Table 2.

Role of the Dual-Energy CT

Since the introduction of the dual-energy CT, there is a constant search for applicability in the different pathologies, in particular, in pancreatitis may add value in some specific points. Virtual noncontrast sequences help to reduce the time of acquisition and therefore decrease the radiation dose in successive controls. Lower KeV images may improve the detection of necrotic areas and thrombosis by optimizing the difference of density and iodine quantification with the contrast-enhanced images. The detection and characterization of noncalcified lithiasis hold special utility importance in the cause clarification for the disease.[11-13]

TABLE 2
CT protocol

CT pancreatitis protocol: arterial and portal-venous phase	
kVp	120-140
Detectors	64 × 0.5
Slice thickness	0.5 mm
Reconstruction interval	0.3 mm
Pitch	57 mm/s
Scan delay (bolus tracking)	45 s (late arterial) 70-80 s (portal-venous)

Morphologic Classification

Acute pancreatitis can be classified into 2 groups according to its morphologic features: acute interstitial edematous pancreatitis and necrotizing acute pancreatitis (NAP). The prognosis of these 2 entities varies significantly.

Acute Interstitial Edematous Pancreatitis

Acute interstitial edematous pancreatitis (AIEP) consists of inflammation of the pancreatic parenchyma and peripancreatic tissues, but without recognizable tissue necrosis. This is the most frequent presentation of acute pancreatitis[5] (80%-85%) with very low morbidity and mortality. On CECT, AIEP is characterized by pancreatic parenchymal edema, peripancreatic fat stranding, free fluid, and no findings of pancreatic or peripancreatic necrosis (Fig 1). Temporally, collections related to AIEP are defined as acute peripancreatic fluid collections (APFC) or pseudocysts, depending on onset time.[5]

Necrotizing Acute Pancreatitis

The 2012 revised Atlanta classification defines necrotizing pancreatitis as inflammation associated with the presence of necrosis, whether intrapancreatic, isolated peripancreatic (Figs. 2 and 3) or frequently a combination of both pancreatic and peripancreatic soft tissues. This entity represents less than 30% of acute pancreatitis (15%-20%), but has significant morbidity and mortality.[14]On CECT, pancreatic necrosis is defined by the lack of enhancement that represents parenchymal hypoperfusion (Fig 4). The intrapancreatic areas of necrosis can be quantified as follows: <30%, between 30% and 50% or >50% of the pancreas, and can be identified after the first 48-72 hours. This is important because similar findings before the first 48 hours may only represent areas of reversible ischemia and edema rather than necrosis.[15,16]

Pancreatic necrosis, as described in the literature, is identified in the arterial phase.[9] Nevertheless, we suggest using the portal phase as a complement because in some patients it can be challenging to obtain a pancreatic arterial phase of good quality. Some authors even suggest that a portal venous phase can be enough to characterize pancreatic necrosis.[17]

Necrosis is classified according to temporality as an acute necrotic collection (ANC) or walled off necrosis (WON) (Fig 8).

Pancreatic and Extrapancreatic Necrosis

In 2013, Bakker et al[18] proposed that apart from the quantity of necrosis, the site of necrosis also was important to predict the morbidity and mortality of the patient. The researchers suggested that extrapancreatic necrosis and pancreatic necrosis are different entities that confer different prognoses. In their prospective study, they found that patients who only presented with extrapancreatic necrosis had less risk

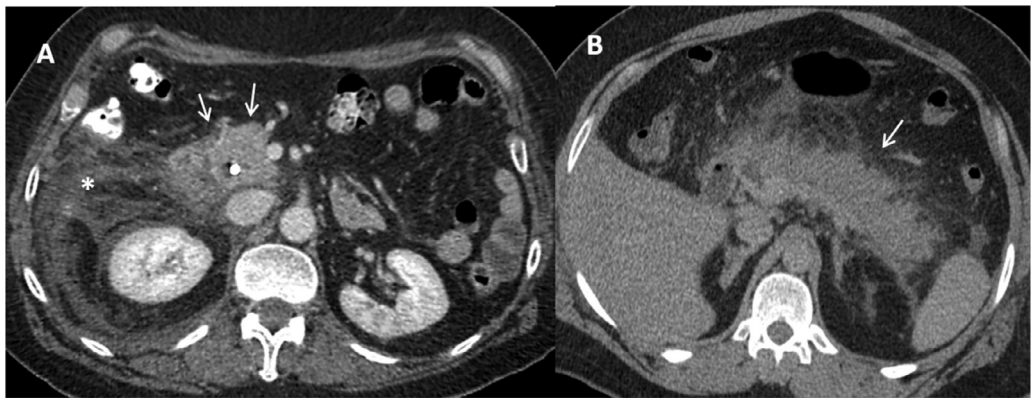


FIG 1. (A) Focal AIEP of the head and uncinate process (arrows). Focal enlargement of the pancreas with extension of the peripancreatic inflammatory changes involving the right anterior pararenal space (*). (B) Diffuse AIEP CECT Equilibrium phase (arrows). Diffuse enlargement of the pancreas with peripancreatic inflammatory changes (edema of the retromesenteric plane with minimal peripancreatic fluid without parenchymal areas of hypoperfusion).

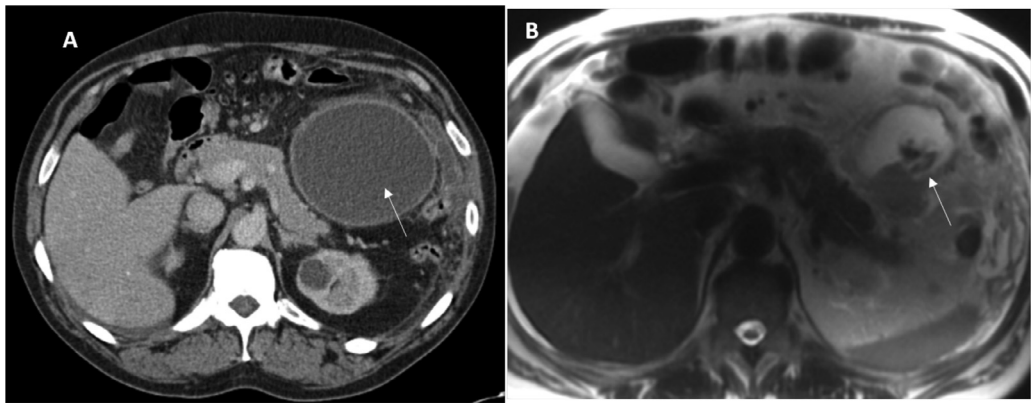


FIG 2. A 65-year-old patient with a history of 7 days of abdominal pain and elevated serum lipase. CECT (A) demonstrates a peripancreatic collection (arrow), and (B) T2-weighted MRI confirms heterogeneous material (debris) within this collection consistent with an acute extrapancreatic necrotic collection (ANC). No necrosis of the pancreas is present.

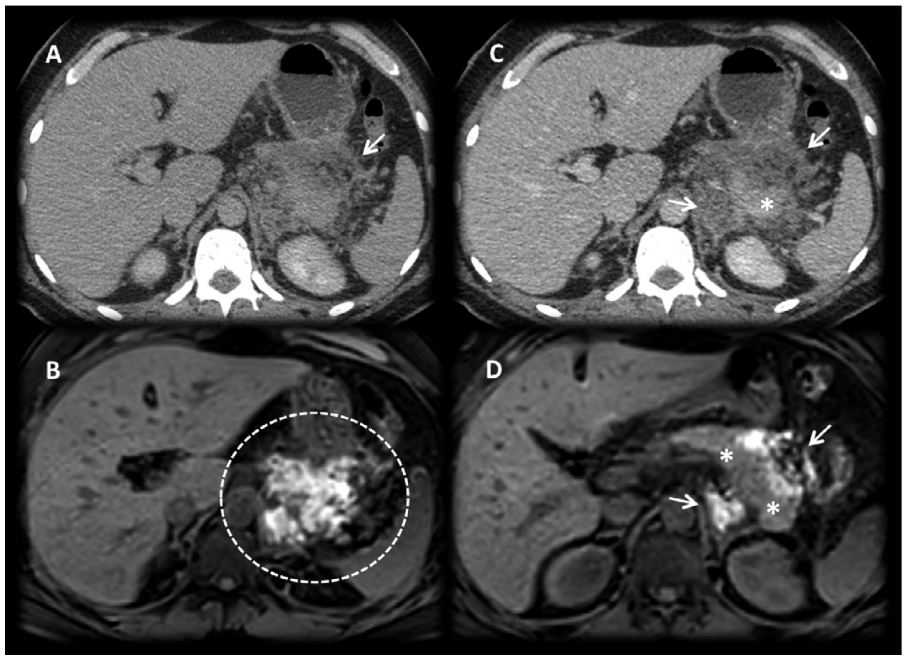


FIG 3. Early NAP with isolated extrapancreatic necrosis. (A, C) CECT demonstrates peripancreatic soft tissue necrosis. Note the tiny dots of macroscopic fat tissue within the hypodense collection (arrows), resembling a "cobblestone" appearance of the necrosis, a very useful sign to depict early extrapancreatic fat necrosis. (B, D) T1 Weighted suppressed MRI confirms heterogeneous hyperintensity of the peripancreatic soft tissue (circle and arrows), consistent with extrapancreatic necrosis. No necrosis of the remaining pancreatic parenchyma is observed (*).

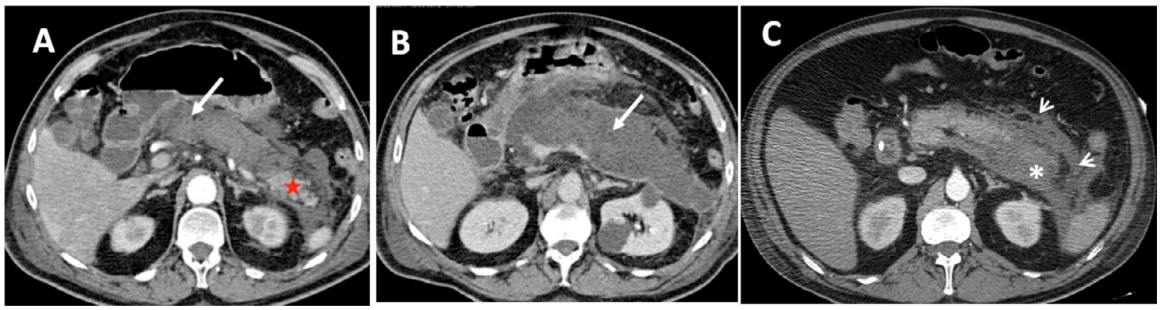
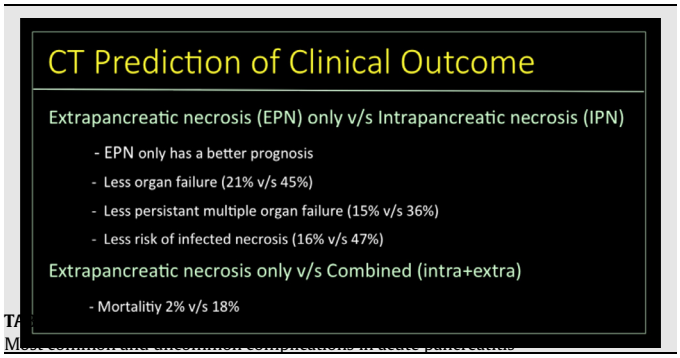


FIG 4. CECT in NAP A) extensive necrosis (white arrows) involving almost 80% of pancreas except for a portion of the tail (red star) that enhances with contrast. (B) Progression within 3 weeks shows an acute necrotic collection. (C) Arterial phase of another case shows lack of enhancement of the body and tail (*) that represents intrapancreatic necrosis (30%-50%) plus extrapancreatic fat necrosis (arrow heads).

TABLE 3
CT prediction of clinical outcome based on localization and involvement of necrosis



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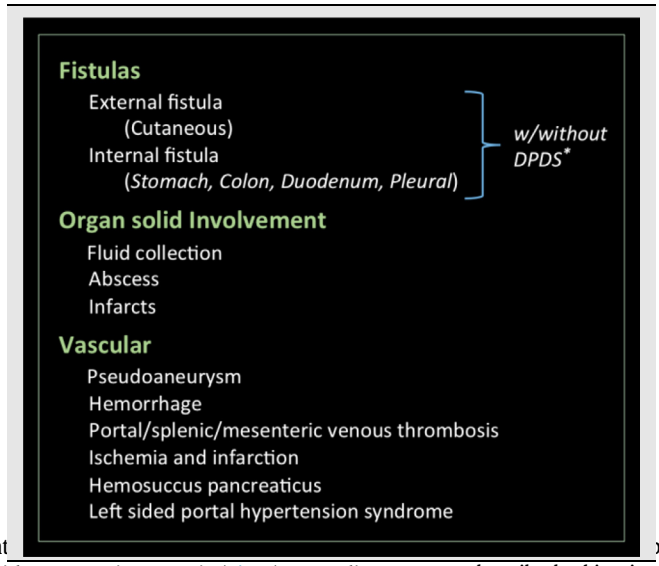

of organ failure, persistent [illegible] presentation time varies, complications will be compared with patients with pancreatic necrosis (Fig 2). Mortality was also significantly different: 2% in the extrapancreatic necrosis group, and 18% in the pancreatic necrosis group (Table 3).

In 2016, Wang et al,[19] in a comparative study of clinical outcomes between combined necrotizing pancreatitis vs extrapancreatic necrosis alone, reaffirmed that extrapancreatic necrosis alone exhibited a significantly better prognosis than combined necrosis and proposed that it be regarded as a separate acute necrotizing pancreatitis entity.

Local Complications of Acute Pancreatitis

Complications of acute pancreatitis have been previously classified according to their temporality into 3 phases: early, intermediate, and late. Systemic complications tend to appear during the early phase (first week), while local complications mainly occur during the intermediate and late phase (>3 weeks), but they can also start appearing in the described taking into account compromised sites instead of onset time.

Fluid Collections

Acute Peripancreatic Fluid Collections

APFC are foci of peripancreatic fluid with no fat necrosis that develop within the first 4 weeks after the onset of interstitial edematous pancreatitis.[5] On CECT, they are characterized as a peripancreatic homogeneous collection with fluid density confined by normal peripancreatic fascial planes with no definable wall encapsulating the collection (Fig 5). They are frequently reabsorbed in a few weeks with no secondary infection.[2,5,7,20]

Pseudocyst

A pancreatic pseudocyst is a late complication that occurs more than 4 weeks after the onset of interstitial edematous pancreatitis

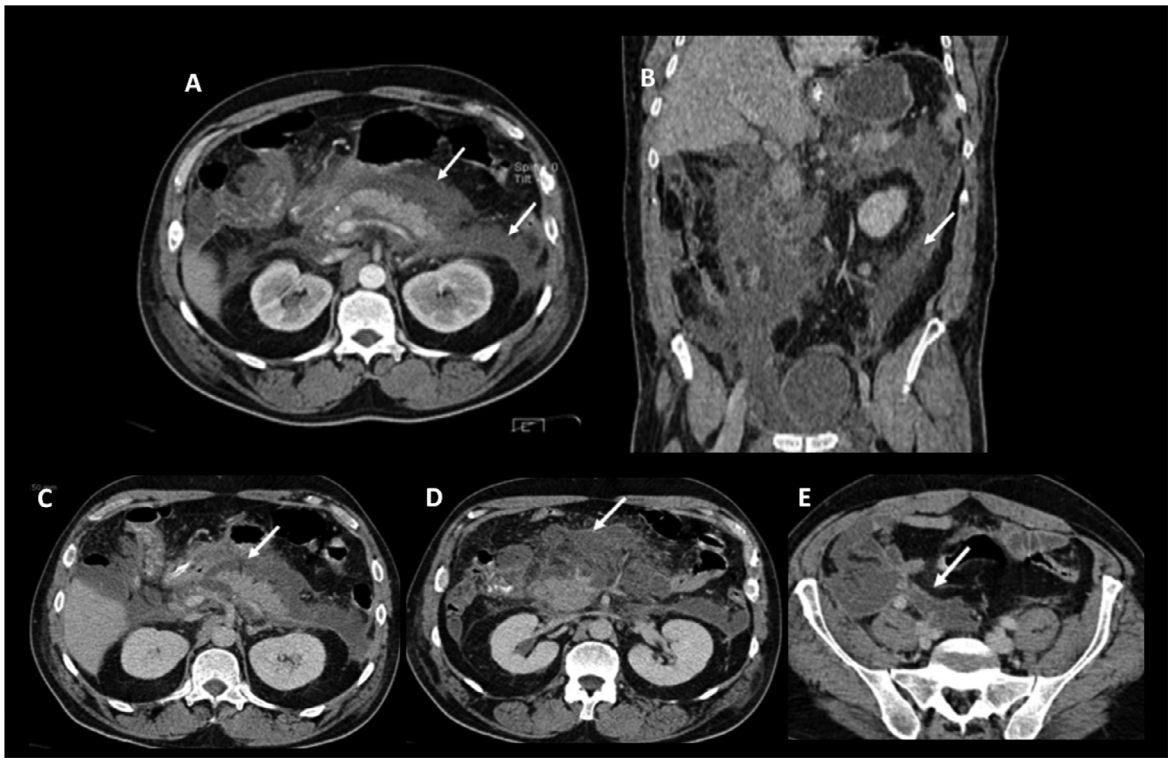

FIG 5. AIEP with APFC. (A) Axial and (B) MPR coronal CECT portovenous phase (PVP) at first (arrows). Peripancreatic fluid collections and inflammatory changes extending through retromesenteric plane involving the left anterior pararenal space and ascites in the left paracolic gutter. (C-E) Follow-up of the previous patient with APFC within 3 weeks. There is more organization of the peripancreatic fluid collections (better defined wall enhancement) (arrows). No pancreatic necrosis is present.

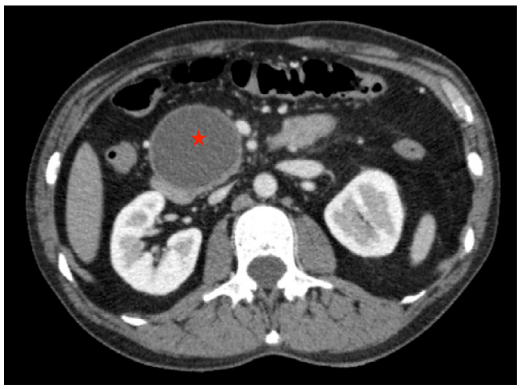

FIG 6. Pseudocyst. Follow-up of AIEP (3 months). CECT in PVP. Pseudocyst of the pancreatic head. (*) Well-defined cystic mass, with no clear debris in fluid collection.

(late phase). It is surrounded by a capsule of granulation tissue with no epithelial lining in an extrapancreatic location.[21] Ten to twenty percent of APC will evolve into pseudocysts rather than being reabsorbed. On CECT, the appearance is a peripancreatic fluid collection with homogeneous attenuation and a well-defined wall. As with APC, there is no detectable pancreatic necrosis (Fig 6).

Acute Necrotic Collection

This complication consists of a collection with variable amounts of both fluid and necrosis. ANC occurs only in the setting of acute necrotizing pancreatitis and is characterized on CECT by the presence of heterogeneous and nonliquid density of varying degrees in a nonencapsulated collection. Necrosis can either be pancreatic, extrapancreatic, or most commonly both[9] (Fig 7).

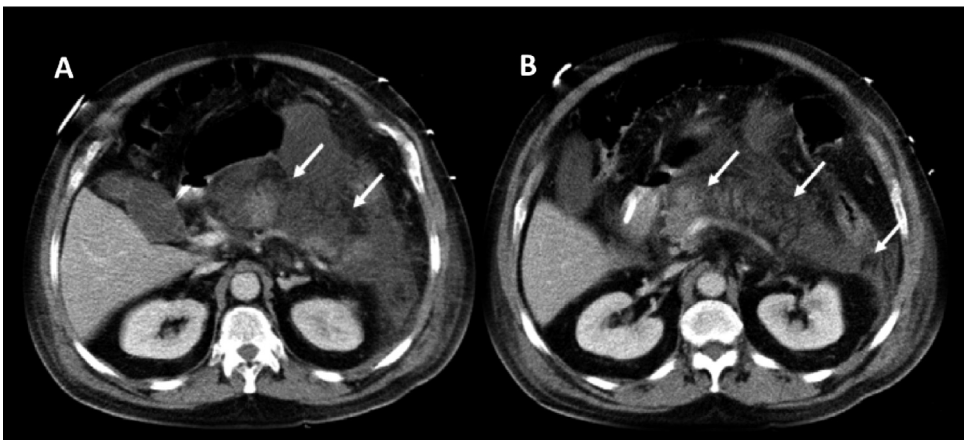

FIG 7. CECT PVP (A, B). Early necrotizing acute pancreatitis (>50%) complicated with an ill-defined pancreatic-peripancreatic ANC (arrows). Necrotic collections with no clear wall. The affected pancreatic fat necrosis demonstrates no enhancement.

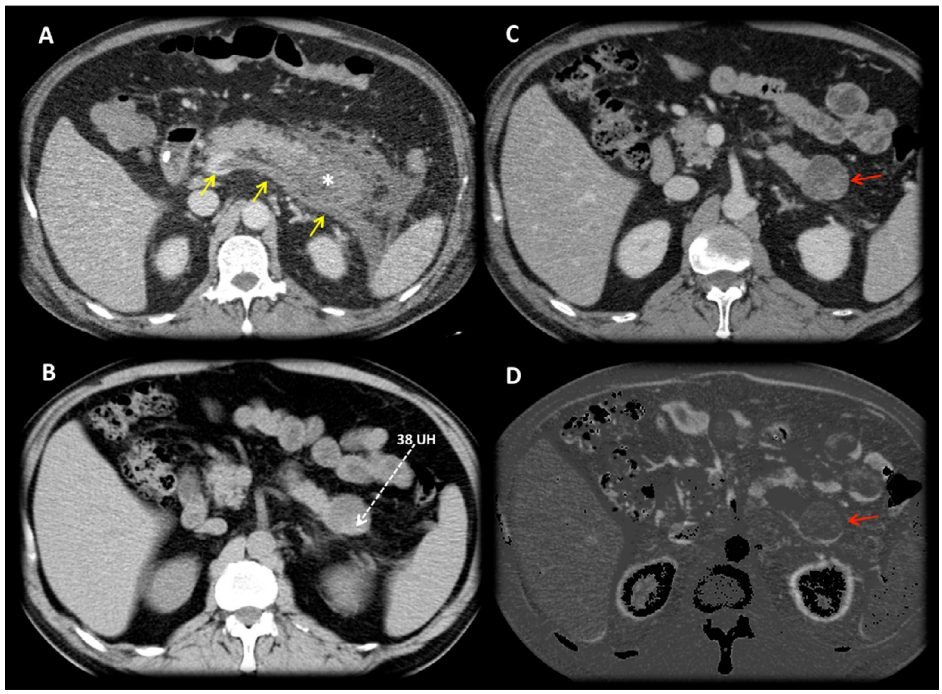

FIG 8. Walled-off necrosis (WON) of the tail of the pancreas. (A) CECT PVP :NAP with necrosis of almost 30% of the body and tail (*), yellow arrows (vascular complication with acute portal and spleen vein thrombosis); (B-D) Follow-up of pancreatitis 6 months later. (B) Nonenhanced image depicts a round dense lesion in the tail of the pancreas. (C, D) PVP and digital subtraction of the tail lesion that denotes no enhancement, representing necrotic collection with debris and avascular pseudocapsule (WON). (Color version of figure is available online.)

Walled-Off Necrosis

WON is a mature, encapsulated necrotic collection, either pancreatic, extrapancreatic isolated or combined (intra + extra pancreatic) that has developed a well-defined inflammatory wall. Usually, it appears in the late phase (>4 weeks) after the onset of necrotizing pancreatitis. Main features on CECT are: heterogeneous collection with fluid and nonfluid density with varying degrees of loculations and a well-defined wall (Fig 8).

Ultrasound (US) and MRI are better techniques to show the presence or absence of debris to distinguish a pseudocyst from walled-off pancreatic necrosis.[20]

Other Fluid Collections

Infected Necrosis

Secondary bacterial infection occurs more frequently in NAP (40%-70%) usually after 3 weeks of the onset of pancreatitis. This is one of the most severe complications of acute pancreatitis and is associated with a high morbidity and mortality rate (50%).[22] On CECT, it is characterized by the formation of extra luminal gas within the collection secondary to enterobacteria, *Escherichia coli* being the most common.[8] The differential diagnoses for extraluminal gas in the pancreas are duodenal reflux in the context of papillotomy, pancreatoenteric fistula, and endoscopic instrumentation.

It is interesting to note that in occasional cases, the presence of gas can be seen in aseptic necrotic collections due to the liquefaction of the pancreatic-peripancreatic tissues, so the clinical context remains key in suggesting infection.[15,16]

Emphysematous Pancreatitis: An Abandoned Concept?

Emphysematous pancreatitis is defined as a necrotizing infection of the pancreas, usually as a complication of a necrotic infected collection in an immune suppressed patient. It is considered one of the most severe complications of acute pancreatitis, and it is associated with a high morbidity and mortality (almost 50%).[23,24]

Early radiographic detection of retroperitoneal gas is critical for the diagnosis of emphysematous pancreatitis.

CT is the modality of choice for detecting parenchymal gas as well as evaluating its extent and location (Fig 9).

It is unclear if this term is still in use, as it is not described in the revised Atlanta classification and its description is the same as infected necrosis. Nevertheless, we suggest that emphysematous pancreatitis should be regarded as a separate infectious complication entity, with a prognosis, morbidity and mortality totally different from an isolated infected collection in NAP.

Other Local Complications

Development of Internal and External Fistulas

Fistulous complications of pancreatitis are explained by the proteolytic action of the pancreatic enzymes. The exudative pancreatic fluid initially located around the pancreas within the anterior pararenal space and retromesenteric plane, spreads first into the lesser sac and tends to dissect along fascial planes, into the transverse mesocolon, phrenocolic ligament and small bowel mesentery. Therefore, most of the complications affect the stomach, duodenum, and transverse colon.[25]

The development of fistulas between the pancreas and other organs is an infrequent complication. Fistulas can be classified as external or internal. External fistulas are more common, and most of the time secondary to therapeutic drainage or surgical procedures. Less frequent, internal fistulas can affect a wide range of organs including the colon, pericardium and pleura, among others (Fig 10).

Pleuropancreatic fistulas are a very rare complication, affecting 0.4%-4.5% cases of pancreatitis. They result from the posterior disruption of the pancreatic duct or a pancreatic cyst into the retroperitoneal space, the diaphragm and subsequently the pleura (Fig 11). These fistulas are more common in chronic pancreatitis, in men, and in pancreatitis of alcoholic etiology.[26,27] They are generally silent, but can be symptomatic in approximately 20% of patients presenting with dyspnea, pleural effusion, chest, and abdominal pain. Among laboratory findings, an elevated amylase level in the pleural fluid is the most characteristic. ERCP and MRCP can be useful, but frequently

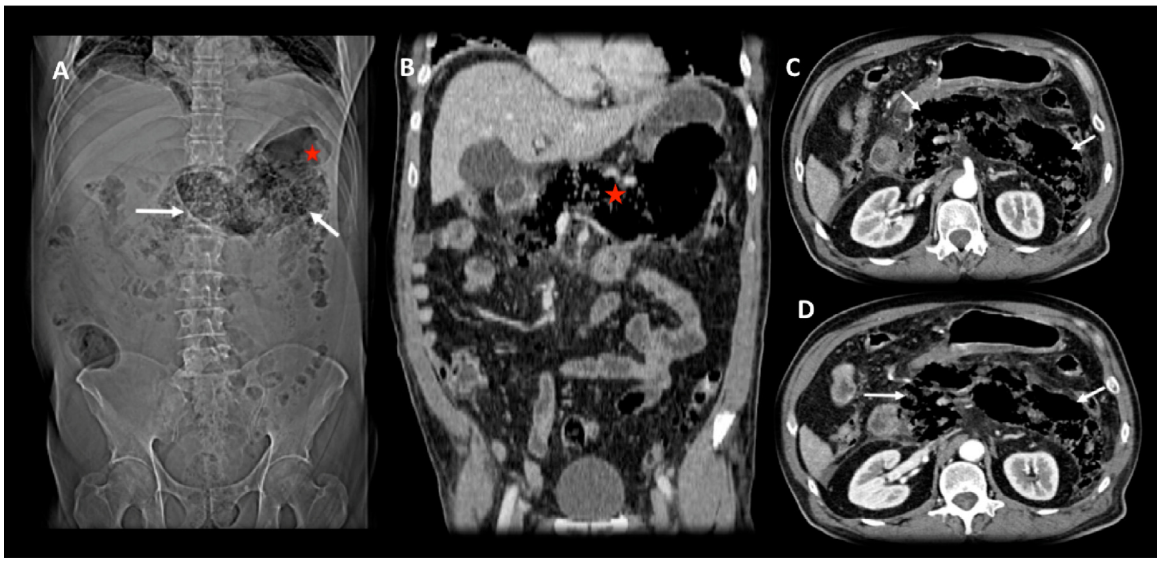

FIG 9. Diffuse acute emphysematous necrotizing pancreatitis. Extensive gastrosplenic necrosis with complete gas replacement of the pancreas and spleen (stars) with extension of the gas collection to the retromesenteric plane and mesenteric secondary to arterial-venous thrombosis of pancreatic and spleen vascular pedicle. (A) Scout view; CECT PVP (B) MPR coronal, (C, D) axial (arrows). Gas collection that extends up to the second portion of the duodenum and retromesenteric planes.

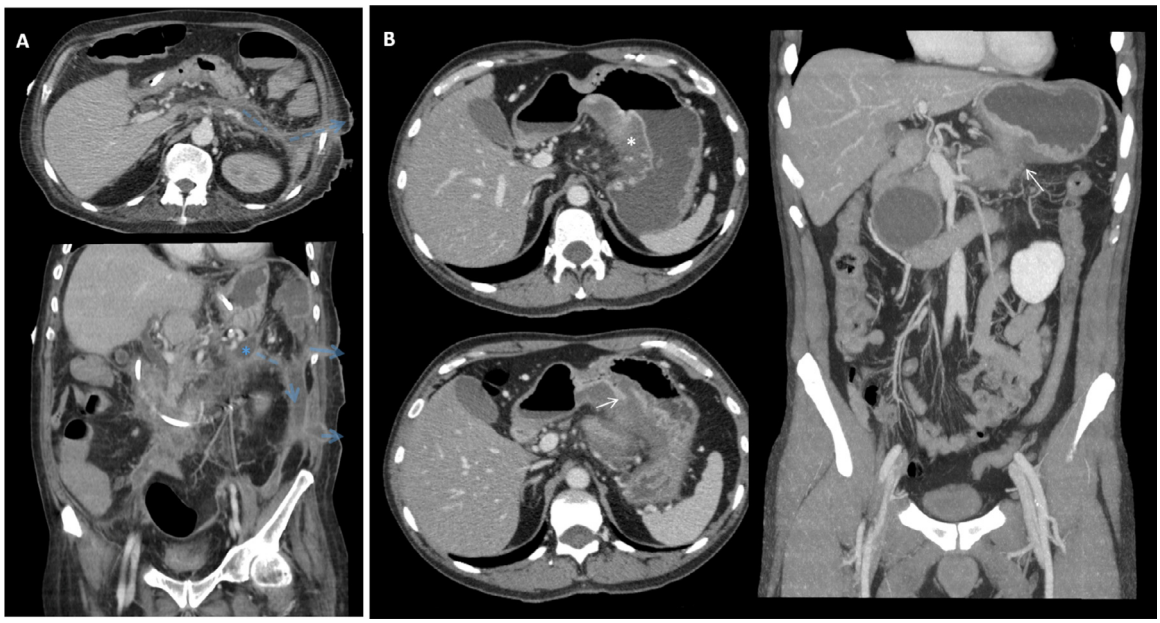

FIG 10. CECT PVP axial and MPR coronal. (A) NAP complicated with peripancreatic WON that required percutaneous drainage. Follow-up after catheter removal, depicted an external fistula (blue arrows) between the residual collection (*) and the skin. (B) NAP complicated with ANC (arrow). Fistulous tract between the main pancreatic duct and stomach. Note the edematous thickened wall of the lesser gastric curvature wall (*). (Color version of figure is available online.)

fail to describe a fistulous tract. CT is quite effective for mapping complex fistula anatomy.[26]

Disconnected Pancreatic Duct Syndrome

There is a subgroup of patients with necrotizing pancreatitis where necrosis affects mostly the ductal epithelium rather than the glandular tissue. Disconnected pancreatic duct syndrome (DPDS) is defined by a complete discontinuity of the main pancreatic duct (MPD), such that the viable segment of the pancreas remains isolated from the gastrointestinal tract. The most frequent causes are acute pancreatitis and pancreatic trauma. The incidence of DPDS in acute pancreatitis is 10%-30%.[28] It occurs predominantly in the pancreatic neck, because it is an area susceptible to ischemia due to its perfusion.[28] It is important to recognize this complication because if exocrine function of the viable segment side of the pancreas is preserved, this may lead to an intra or peripancreatic collection that will not resolve with drainage or can cause an internal or external persistent fistula, which will require surgery in most cases[28,29] (Fig 12).

Diagnosis is difficult and often overlooked with imaging tests. In order to diagnose DPDS, it is necessary to demonstrate the three following features: (1) necrosis of at least 2 cm of the pancreas; (2) viable pancreatic tissue upstream from the site of necrosis; (3) extravasation of contrast material injected into the MPD (by ERCP, endoscopic ultrasonographically guided or by surgical pancreatography).[29]

On CECT, the classic image is a fluid collection in the neck or body that replaces the glandular tissue. If the MPD is visible in the viable

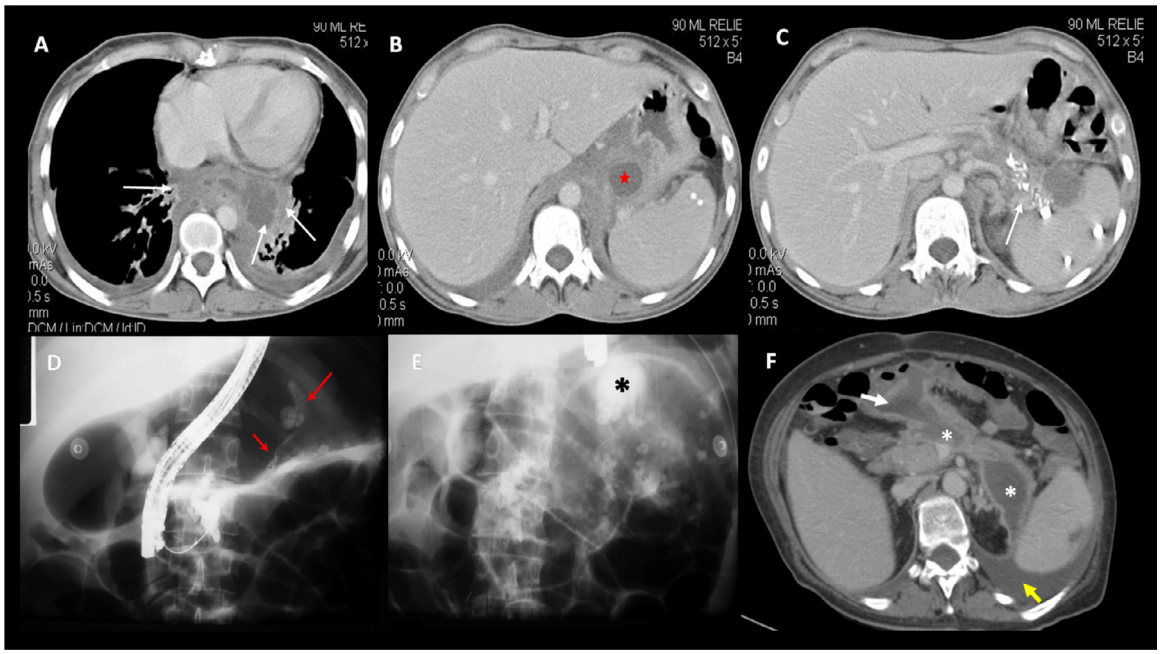

FIG 11. Internal fistula—pleuropancreatic fistula. A 42-year-old female patient with prior history of recurrent acute pancreatitis, last episode 6 months before, complaining of hemoptysis and dyspnea in the emergency room. Chest x-ray and physical examination demonstrated bilateral pleural effusion. The pleural fluid study revealed high levels of amylase, which confirmed the presence of a pleuropancreatic fistula. (A-C) CECT PVP axial images; (*) Pancreatic WON; (Arrows)→Pleuropancreatic communication through a fistulous tract ending in bilateral pleural space through pleurodiaphragmatic tracts. (C) Coarse calcifications in an atrophic pancreas, consistent with chronic pancreatitis complicated with WON and pleuropancreatic fistula that extends to the posterior mediastinum and both pleural spaces. (D, E) Selected images of ERCP depicting the irregular enhance of the Wirsung duct with a fistulous tract that communicates the main pancreatic duct at the body level with WON proven on CT (D); (E) fluoroscopic images during the ERCP at the left pulmonary base depicted a filling cavity (*) that belongs to the posterior mediastinum, consistent with proven pleuropancreatic fistula through pleurodiaphragmatic tract communications. No disconnected pancreatic duct was proven on ERCP. (F) Another patient with extensive peripancreatic collections (WON) that communicate with the transverse colon and the left pleural space through colopancreatic (white arrow) and pleuropancreatic fistula (yellow arrow); (*) WON. (Color version of figure is available online.)

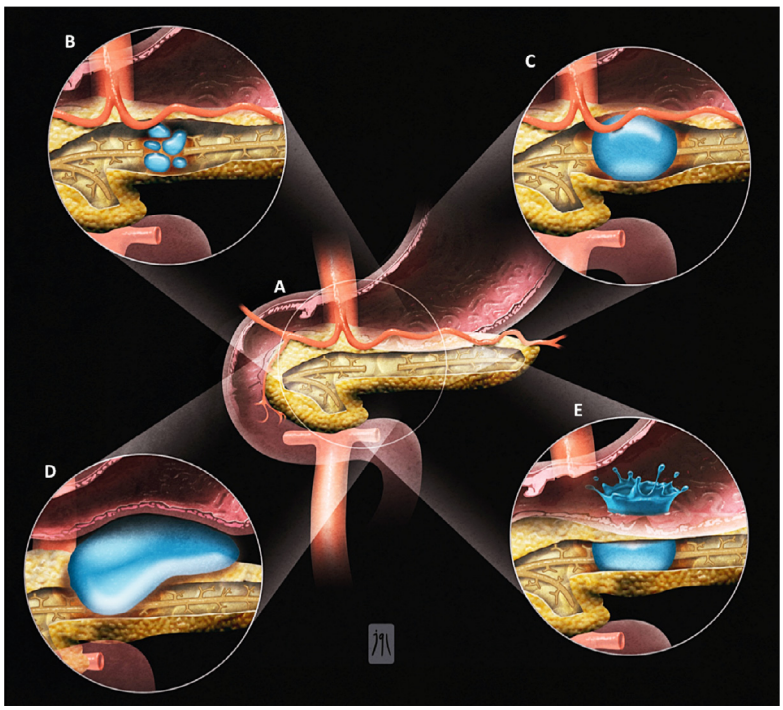

FIG 12. Scheme representation of DPDS and development of collections and/or fistulas. (A) Disconnected duct; (B) early development of phlegmonous intrapancreatic collection; (C) intrapancreatic WON; (D) intrapancreatic WON extension to peripancreatic soft tissue; (E) gastropancreatic fistula.

upstream segment of the pancreas, it shows an abrupt cut off and an angle of 90° with the collection[28] (Fig 13).

Not all peripancreatic collections signify ductal disconnection; therefore, it is important to recognize for thin bridges of viable glandular tissue that are compressed by fluid collections in multiplanar images.[29]

Solid Organ Involvement

The inflammatory exudates and peripancreatic collections can dissect through fascial planes and affect adjacent solid organs, including the liver, spleen, and kidneys. Splenic involvement is most

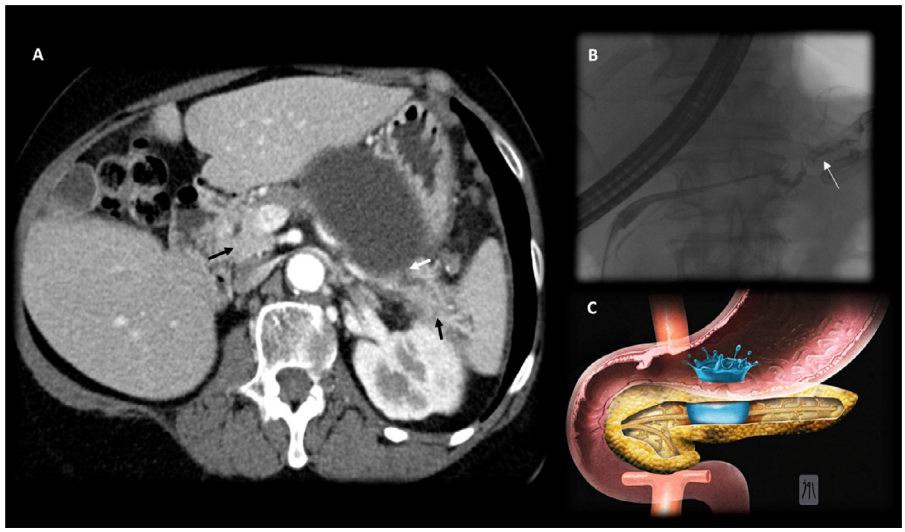

FIG 13. A 54-year-old female patient with necrotizing pancreatitis. (A) CECT image demonstrates an intact pancreatic head and tail (black arrows) with necrosis of the body. A disconnected duct is demonstrated on CT by the pancreatic duct in the tail draining into a collection at a 90° angle, without intervening pancreatic parenchyma between the tail and head (white arrow). (B) ERCP image demonstrates extravasation of contrast into a cyst gastrostomy tube from the dorsal (main) pancreatic duct in the body, with failure to opacify the duct in the tail consistent with a disconnected duct (white arrow). (C) Schematic representation of pancreatogastric communication.

common, facilitated by the location of the tail of the pancreas in the splenic hilum[30] (Fig 14). In most patients, Gerota's fascia forms a protective barrier against enzymatic and inflammatory action of pancreatic exudates. Occasionally, however, pancreatic secretions can traverse fascial planes and involve either kidney in a complex renal and perirenal inflammatory process.[25]

Vascular Complications

Vascular complications are related to the severity of the episode. While they are infrequent in mild acute pancreatitis, they are reported in up to 50% of necrotizing acute pancreatitis. They can result in high mortality rates if not detected and managed early.

The range of vascular morphologic changes includes inflammation and perivascular fibrosis causing stenosis or thrombosis, and vascular erosions leading to ischemia, infarction, pseudoaneurysms, and hemorrhage.

Hemorrhage and Pseudoanerysms

Massive hemorrhage is an uncommon complication of pancreatitis and it is associated with a high rate of mortality. Hemorrhage is

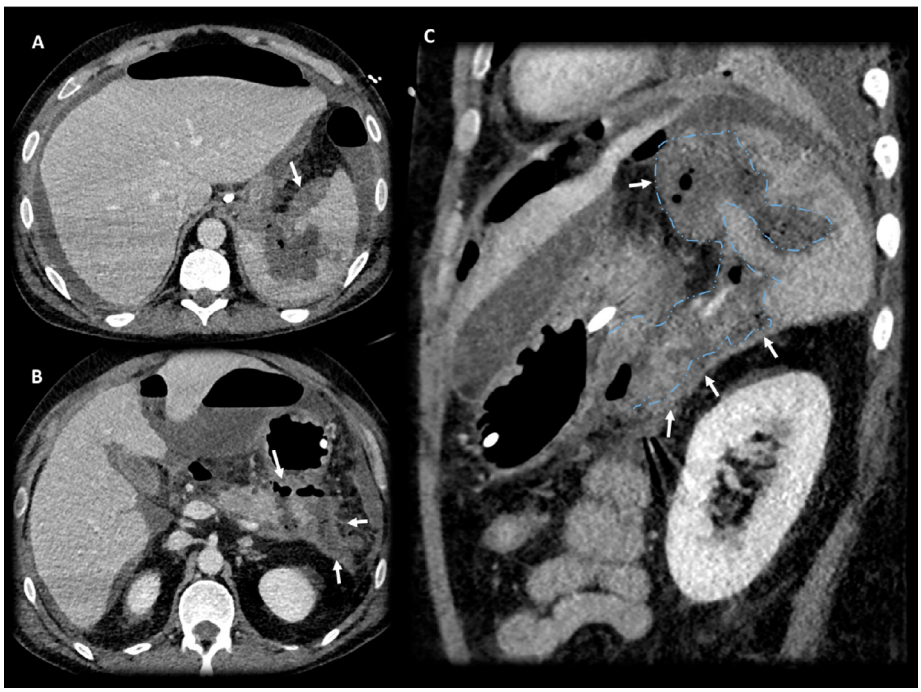

FIG 14. NAP complicated with extensive peripancreatic infected fluid collections (WON) that extends through the splenic hilum involving the spleen. CECT PVP axial (A, B) and MPR sagittal at the necrotic tail of the pancreas (C) (arrows and detached lines) → depicting the boundaries of the infected peripancreatic WON involving the spleen.

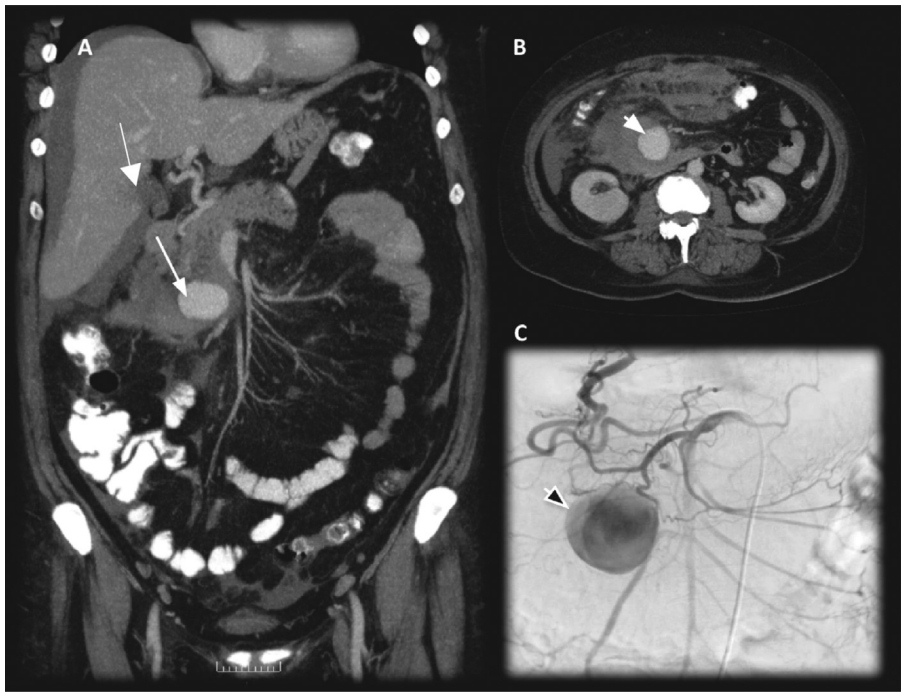

FIG 15. Pseudoaneurysm of the inferior pancreatoduodenal artery secondary to acute pancreatitis, in direct relation with the uncinate process. CECT late arterial phase MPR cor (A) axial; (B) homogeneous hypervascular structure that follows the arterial vascular pool, in the head of the pancreas (arrows); (C) Angiography with digital subtraction confirmed the diagnosis.

usually caused from a bleeding pseudoaneurysm or less frequently from direct erosion into major blood vessels.[31]

Pseudoaneurysms develop in approximately 3.5%-10% of patients with acute pancreatitis, with a higher incidence in severe pancreatitis.[32] On CECT, they are characterized as a completely or partially vascular rounded lesion with transient vascular enhancement that follows arterial blood pool (Fig 15).

The splenic artery is the most frequently involved vessel because of its contiguity with the pancreas (40%), followed by the gastroduodenal artery (20%), pancreaticoduodenal artery (20%), gastric arteries (5%), hepatic arteries (2%), and others (1%-3%).[33,34] Pseudoaneurysms may rupture into the peritoneal cavity, lesser sac, pancreatic parenchyma, or into a hollow viscus.[35]

Arteriovenous Fistula

Arteriovenous fistula (AVF) is a very rare complication of acute pancreatitis whose pathogenesis remains unclear. The incidence is unknown and there are only a few case reports of splenic AVF secondary to acute or chronic pancreatitis.[36] Angiography demonstrates an intense hypervascularity and arteriovenous shunting arising from this hypervascular area (Fig 16).

Portomesenteric/Spleen Vein Thrombosis

Thrombosis secondary to acute pancreatitis is relatively rare. In order of frequency, the splenic vein is involved more frequently, approximately in 1%-3% of the cases, followed by the portal and superior mesenteric vein. In chronic pancreatitis, splenic vein thrombosis is much more frequent and develops in about 10%-40% of the patients.

CECT can detect splenic vein thrombosis with a sensitivity of 71%.[37,38] Findings suggestive of splenic thrombosis include an expanded vessel with an irregular endoluminal nonenhancing filling defect within the vein on an appropriately timed venous phase CECT (Fig 17). Angiography with venous phase is the gold standard for diagnosis of splenic vein thrombosis.[38]

Ischemia and Infarction

The erosion of the arterial territory can lead to ischemia and infarction. As the vascular structures that are near the pancreas irrigate the duodenum, colon and spleen, they are affected more frequently (Fig 9).

Hemosuccus Pancreaticus

The term hemossucus pancreaticus refers to bleeding from the ampulla of Vater via the pancreatic duct.[39] It is a rare and potential life-threatening cause of upper gastrointestinal bleeding. Bleeding is often intermittent and repetitive, but not severe enough to cause hemodynamic instability.[40,41] It is most often caused by rupture of a pseudoaneurysm of peripancreatic arteries in the setting of acute or chronic pancreatitis. The most frequently affected arteries are: splenic, gastroduodenal, pancreaticoduodenal, gastric and hepatic arteries[35,39] (Fig 18).

Other causes include pseudocysts, pancreatic tumors, pancreas divisum with chronic pancreatitis, vascular malformations, trauma, and iatrogenicity.[39,40]

Patients present with history of recurrent abdominal pain, gastrointestinal bleeding, and high amylase levels.[39] Diagnostic modalities include CECT, esophagogastroduodenoscopy, selective angiography, EUS, and ERCP.[39] Angiography is the gold standard for diagnosis and can be used as a therapeutic modality.[39] Coil embolization is the most frequent interventional treatment.[39,40] Stent placement can be used previous to surgery[39] (Fig 19).

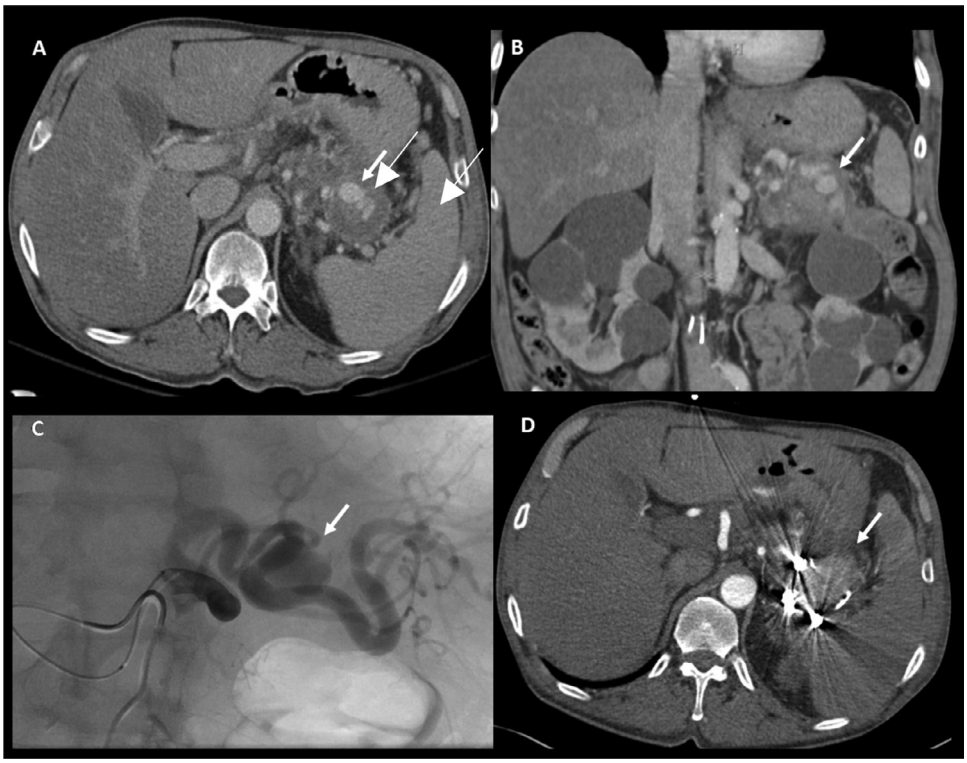

FIG 16. A 59-year-old male patient with alcoholic pancreatitis and splenic artery arteriovenous fistula (AVF). (A, B) CECT images demonstrate a lobulated hypervascular focus in the pancreatic tail initially thought to represent a splenic artery pseudoaneurysm (arrow). (C) Digital subtraction angiography of selective splenic artery injection demonstrates a lobulated vascular lesion, with early filling of the splenic vein (not shown) consistent with an arteriovenous fistula, treated by coil embolization. (D) Postembolization CT demonstrates residual hyperattenuation within the coiled AVF from prior contrast administration, but the attenuation is less than that of the aorta and precontrast imaging confirms no enhancement, consistent with successful treatment.

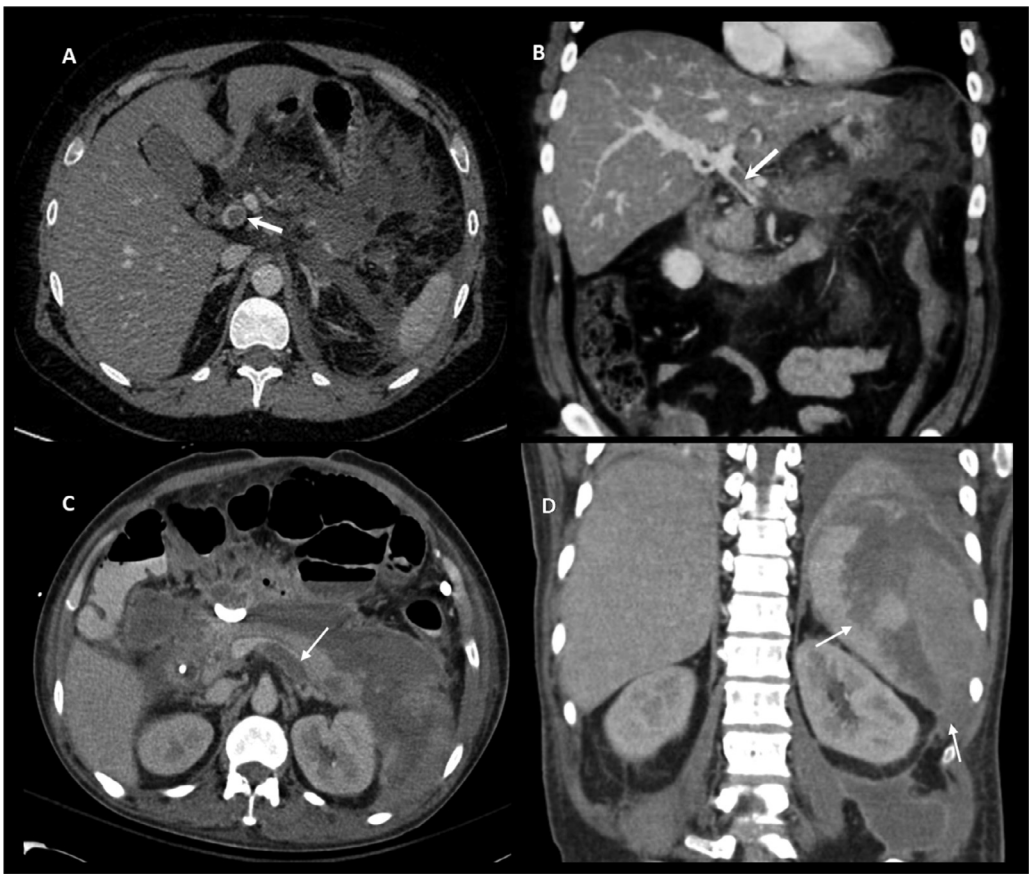

FIG 17. NAP complicated with portal vein thrombosis. CECT PVP axial (A) and MPR (B) (white arrows) depict a partial occlusive thrombus in the lumen of main portal vein. (C, D) A 35-year-old male patient with necrotizing pancreatitis complicated by splenic vein thrombosis (C), splenic infarct (D), and splenic hemorrhage. Axial CECT demonstrates a nearly completely thrombosed splenic vein in the setting of necrotizing pancreatitis. Coronal CECT demonstrates thrombosis of a large volume of the spleen and associated perisplenic hematoma.

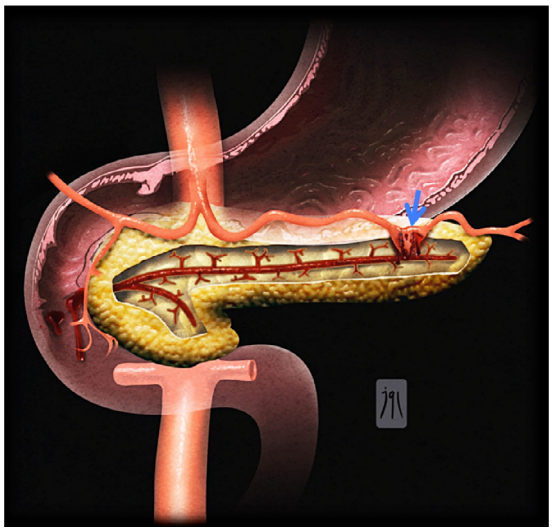

FIG 18. Scheme representation of hemosuccus pancreaticus (blue arrow) → ruptured pseudoaneurysm of the splenic artery communicating with MPD. (Color version of figure is available online.)

CECT can show the source of bleeding and provide crucial planning information for angiographic embolization.[39]

Left-Sided Portal Hypertension

Also known as sinistral or splenoportal hypertension, is a rare, but life-threatening cause of upper gastrointestinal bleeding. It occurs secondary to an isolated splenic vein thrombosis. This results in increased blood flow to short gastric veins which drain to the stomach and consequently, an increase in blood flow and pressure in submucosal gastric veins with development of gastric varices[42] (Fig 20).

The most important causes of isolated splenic vein thrombosis include acute and chronic pancreatitis, pancreatic pseudocysts, and pancreatic carcinomas.[42,43] Most patients are asymptomatic, although it is known that it accounts for less than 5% of all patients with portal hypertension.[42] This diagnosis should be considered in patients with upper gastrointestinal bleeding, splenomegaly, normal liver function, and a patent extrahepatic portal vein.[42] Management in symptomatic patients includes splenectomy and removal of the primary cause. Due to its low incidence, expectant management is recommended in asymptomatic patients.[38,43]

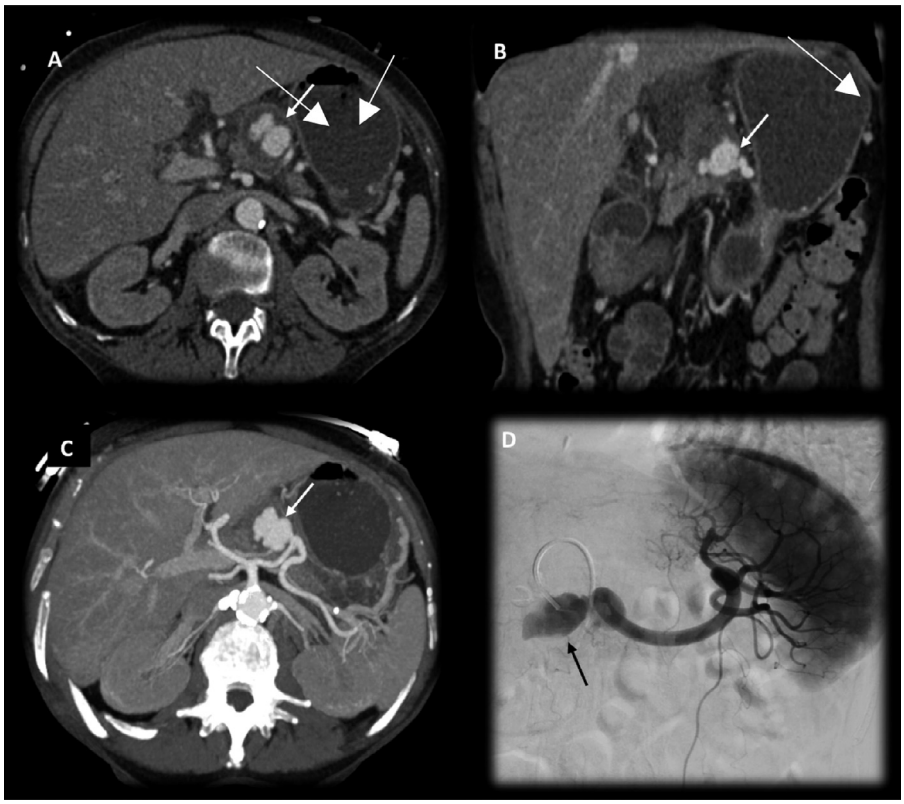

FIG 19. 1. A 61-year-old female patient presenting with right upper quadrant pain and nausea. CECT images demonstrate a large partially thrombosed splenic artery pseudoaneurysm (arrows). (A) Late arterial phase axial CECT; (B) coronal CECT; (C) axial MIP CECT; (D) digital subtraction angiography from selective injection of the splenic artery demonstrates a large splenic artery pseudoaneurysm, which was subsequently treated with covered stent placement to exclude flow to the pseudoaneurysm. **2**. Upper endoscopy of the same patient demonstrates fresh blood oozing from the papilla in the second portion of the duodenum. No ampullary mass was depicted. (A) Lower third esophagus; (B) gastric body; (C-E) second portion of the duodenum and mayor papilla.

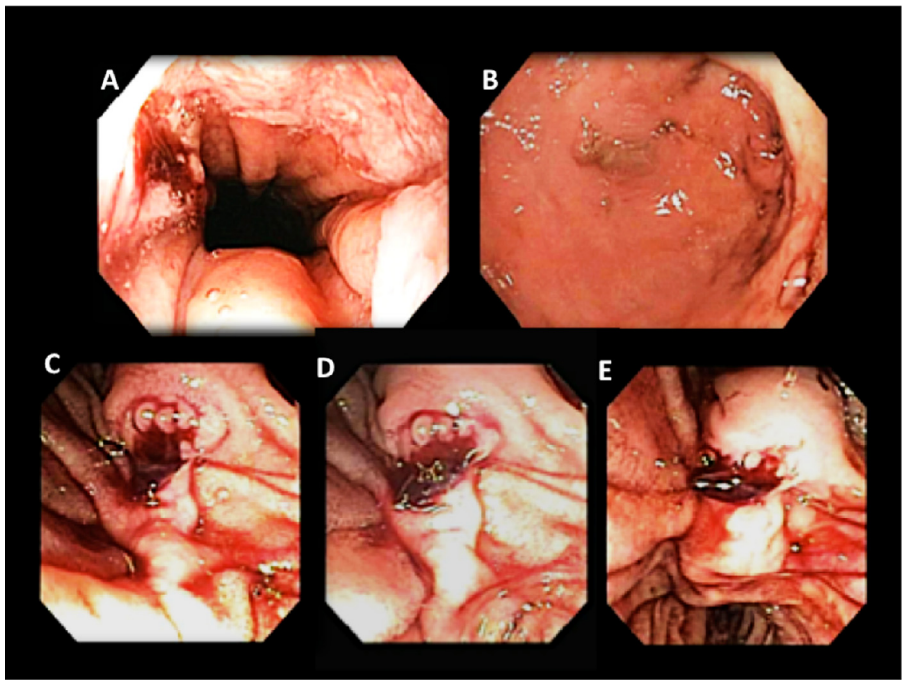

FIG 19. Continued.

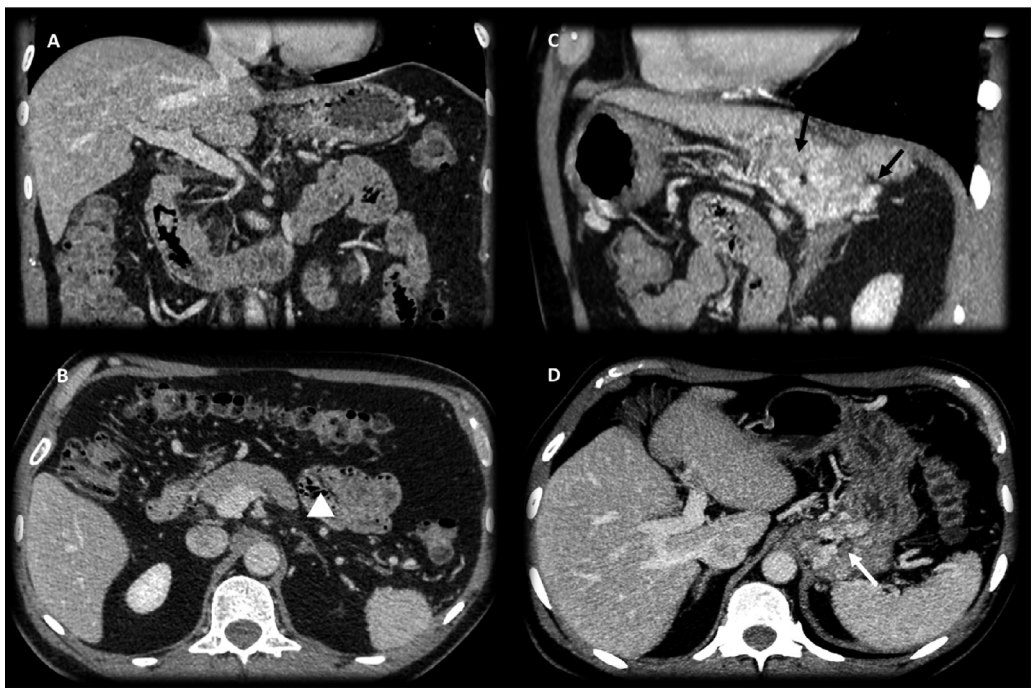

FIG 20. A 65-year-old female patient with history of prior complicated acute pancreatitis with infected collections managed with necrosectomy and distal pancreatectomy 20 years ago. CECT PVP (A, B) MPR cor and axial, (C, D) MPR MIP cor and axial. As a consequence of lack of the splenic vein, a left-sided portal hypertension developed through the years as a compensatory hemodynamic phenomenon secondary to the increase in blood flow and pressure in submucosal gastric veins with subsequent development of gastric varices (arrows). Note patent extrahepatic portal vein and no morphologic chronic liver changes with cirrhosis.

Conclusions

Acute pancreatitis is a common cause of acute abdominal pain with varying degrees of severity. Imaging plays a key role during the late phase of the disease in nonmild pancreatitis, facilitating characterization of disease severity and complications and aiding in prognostication. Early detection and accurate interpretation of complications on CECT lead to prompt management decisions which can reduce the morbidity and mortality of these patients. Knowledge of pancreatitis and its complications is vital for any radiologist that works in the acute care environment.

References

1. Whitcomb DC, Yadav D, Adam S, et al. Multicenter approach to recurrent acute and chronic pancreatitis in the United States: The North American

Pancreatitis Study 2 (NAPS2). Pancreatology 2008;8:520–31 https://doi.org/10.1159/000152001.
2. Bharwani N, Patel S, Prabhudesai S, et al. Acute pancreatitis: The role of imaging in diagnosis and management. Clin Radiol 2011;66:164–75 https://doi.org/10.1016/j.crad.2010.09.003.
3. Morgan DE. Imaging of acute pancreatitis and its complications. Clin Gastroenterol Hepatol 2008;6:1077–85 https://doi.org/10.1016/j.cgh.2008.07.012.
4. Trout AT, Elsayes KM, Ellis JH, et al. Imaging of acute pancreatitis: Prognostic value of computed tomographic findings. J Comput Assist Tomogr 2010;34:485–95 https://doi.org/10.1097/RCT.0b013e3181d344ca.
5. Banks PA, Bollen TL, Dervenis C, et al. Classification of acute pancreatitis—2012: Revision of the Atlanta classification and definitions by international consensus. Gut 2013;62:102–11 https://doi.org/10.1136/gutjnl-2012-302779.
6. Bradley EL. A clinically based classification system for acute pancreatitis. Summary of the International Symposium on Acute Pancreatitis, Atlanta, GA, September 11 through 13, 1992. Arch Surg Chic Ill 1960 1993;128:586–90.
7. Sheu Y, Furlan A, Almusa O, et al. The revised Atlanta classification for acute pancreatitis: A CT imaging guide for radiologists. Emerg Radiol 2012;19:237–43 https://doi.org/10.1007/s10140-011-1001-4.
8. Balthazar EJ, Freeny PC, vanSonnenberg E. Imaging and intervention in acute pancreatitis. Radiology 1994;193:297–306 https://doi.org/10.1148/radiology.193.2.7972730.
9. Shyu JY, Sainani NI, Sahni VA, et al. Necrotizing pancreatitis: Diagnosis, imaging, and intervention. Radiographics 2014;34:1218–39 https://doi.org/10.1148/rg.345130012.
10. Thoeni RF. The revised Atlanta classification of acute pancreatitis: Its importance for the radiologist and its effect on treatment. Radiology 2012;262:751–64 https://doi.org/10.1148/radiol.11110947.
11. George E, Wortman JR, Fulwadhva UP, et al. Dual energy CT applications in pancreatic pathologies. Br J Radiol 2017;90:20170411 https://doi.org/10.1259/bjr.20170411.
12. Matos C. Will dual-energy CT become the reference standard to evaluate gallstone disease? Radiology 2019;292:407–8 https://doi.org/10.1148/radiol.2019191172.
13. Murray N, Darras KE, Walstra FE, et al. Dual-energy CT in evaluation of the acute abdomen. Radiographics 2019;39:264–86 https://doi.org/10.1148/rg.2019180087.
14. Munsell MA, Buscaglia JM. Acute pancreatitis. J Hosp Med 2010;5:241–50 https://doi.org/10.1002/jhm.574.
15. Saokar A, Rabinowitz CB, Sahani DV. Cross-sectional imaging in acute pancreatitis. Radiol Clin North Am 2007;45:447–60. viii https://doi.org/10.1016/j.rcl.2007.04.002.
16. Merkle EM, Görich J. Imaging of acute pancreatitis. Eur Radiol 2002;12:1979–92 https://doi.org/10.1007/s00330-001-1235-8.
17. Kwon Y, Park HS, Kim YJ, et al. Multidetector row computed tomography of acute pancreatitis: Utility of single portal phase CT scan in short-term follow up. Eur J Radiol 2012;81:1728–34 https://doi.org/10.1016/j.ejrad.2011.04.061.
18. Bakker OJ, van Santvoort H, Besselink MGH, et al. Extrapancreatic necrosis without pancreatic parenchymal necrosis: A separate entity in necrotising pancreatitis? Gut 2013;62:1475–80 https://doi.org/10.1136/gutjnl-2012-302870.
19. Wang M, Wei A, Guo Q, et al. Clinical outcomes of combined necrotizing pancreatitis versus extrapancreatic necrosis alone. Pancreatology 2016;16:57–65 https://doi.org/10.1016/j.pan.2015.10.010.
20. Brun A, Agarwal N, Pitchumoni CS. Fluid collections in and around the pancreas in acute pancreatitis. J Clin Gastroenterol 2011;45:614–25 https://doi.org/10.1097/MCG.0b013e318213ef3e.
21. Andrén-Sandberg A, Dervenis C. Pancreatic pseudocysts in the 21st century. Part I: Classification, pathophysiology, anatomic considerations and treatment. JOP J Pancreas 2004;5:8–24.
22. Mazaki T, Ishii Y, Takayama T. Meta-analysis of prophylactic antibiotic use in acute necrotizing pancreatitis. Br J Surg 2006;93:674–84 https://doi.org/10.1002/bjs.5389.
23. Wig JD, Kochhar R, Bharathy KGS, et al. Emphysematous pancreatitis. Radiological curiosity or a cause for concern? JOP J Pancreas 2008;9:160–6.
24. Grayson DE, Abbott RM, Levy AD, et al. Emphysematous Infections of the abdomen and pelvis: A pictorial review. Radiographics 2002;22:543–61 https://doi.org/10.1148/radiographics.22.3.g02ma06543.
25. Larsen M, Kozarek R. Management of pancreatic ductal leaks and fistulae: Pancreatic leak management. J Gastroenterol Hepatol 2014;29:1360–70 https://doi.org/10.1111/jgh.12574.
26. Ali T, Srinivasan N, Le V, et al. Pancreaticopleural fistula. Pancreas 2009;38:e26–31 https://doi.org/10.1097/MPA.0b013e3181870ad5.
27. Khan AZ, Ching R, Morris-Stiff G, et al. Pleuropancreatic fistulae: specialist center management. J Gastrointest Surg 2009;13:354–8 https://doi.org/10.1007/s11605-008-0699-0.
28. Gámez del Castillo JM, Garcés Albir M, Fernández Moreno MC, et al. Disconnected pancreatic duct syndrome: Complete pancreas transection secondary to acute pancreatitis. Rev Esp Enfermedades Dig 2016 https://doi.org/10.17235/reed.2016.3997/2015.
29. Sandrasegaran K, Tann M, Jennings SG, et al. Disconnection of the pancreatic duct: An important but overlooked complication of severe acute pancreatitis. Radiographics 2007;27:1389–400 https://doi.org/10.1148/rg.275065163.
30. Lenhart DK, Balthazar EJ. MDCT of acute mild (nonnecrotizing) pancreatitis: Abdominal complications and fate of fluid collections. Am J Roentgenol 2008;190:643–9 https://doi.org/10.2214/AJR.07.2761.
31. Mallick IH, Winslet MC. Vascular complications of pancreatitis. JOP J Pancreas 2004;5:328–37.
32. White AF, Baum S, Buranasiri S. Aneurysms secondary to pancreatitis. AJR Am J Roentgenol 1976;127:393–6 https://doi.org/10.2214/ajr.127.3.393.
33. Suzuki T, Ishida H, Komatsuda T, et al. Pseudoaneurysm of the gastroduodenal artery ruptured into the superior mesenteric vein in a patient with chronic pancreatitis. J Clin Ultrasound JCU 2003;31:278–82 https://doi.org/10.1002/jcu.10170.
34. Toscano RL, Ruiz OR, Gerace CA. Rupture of splenic artery pseudoaneurysm. Am Surg 1995;61:940–2.
35. Ferreira J, Tavares AB, Costa E, et al. Hemosuccus pancreaticus: A rare complication of chronic pancreatitis. BMJ Case Rep 2015;2015 https://doi.org/10.1136/bcr-2015-209872.
36. Holsbeeck AV, Dalle I, Geldof K, et al. Acquired pancreatic arteriovenous malformation. J Belg Soc Radiol 2015;99:37–41 https://doi.org/10.5334/jbr-btr.863.
37. Hofer BO, Ryan JA, Freeny PC. Surgical significance of vascular changes in chronic pancreatitis. Surg Gynecol Obstet 1987;164:499–505.
38. Butler JR, Eckert GJ, Zyromski NJ, et al. Natural history of pancreatitis-induced splenic vein thrombosis: A systematic review and meta-analysis of its incidence and rate of gastrointestinal bleeding. HPB 2011;13:839–45 https://doi.org/10.1111/j.1477-2574.2011.00375.x.
39. Anil Kothari R, Leelakrishnan V, Krishnan M. Hemosuccus pancreaticus: A rare cause of gastrointestinal bleeding. Ann Gastroenterol Q Publ Hell Soc Gastroenterol 2013;26:175–7.
40. Mandaliya R, Krevsky B, Sankineni A, et al. Hemosuccus pancreaticus: A mysterious cause of gastrointestinal bleeding. Gastroenterol Res 2014;7:32–7 https://doi.org/10.14740/gr596w.
41. Yu P, Gong J. Hemosuccus pancreaticus: A mini-review. Ann Med Surg 2012 2018;28:45–8 https://doi.org/10.1016/j.amsu.2018.03.002.
42. Pereira P, Peixoto A. Left-sided portal hypertension: A clinical challenge. GE Port J Gastroenterol 2015;22:231–3 https://doi.org/10.1016/j.jpge.2015.10.001.
43. Singhal D, Kakodkar R, Soin AS, et al. Sinistral portal hypertension. A case report. JOP J Pancreas 2006;7:670–3.