

Imaging of Acute Pancreatitis: Update of the Revised Atlanta Classification

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KEYWORDS

- Acute pancreatitis • Classification • Severity
- Pancreatic necrosis • Peripancreatic collections

INTRODUCTION

Acute pancreatitis (AP) is defined as an acute inflammatory state of the pancreas and is conventionally categorized as either mild or severe disease. Approximately 80% to 85% of patients with AP will have the mild form with an uncomplicated clinical course, whereas 15% to 20% develop a complicated clinical course characterized by organ failure and/or local complications.¹

Worldwide the incidence of AP is increasing, probably related to the rising incidence of gallstones, obesity, and aging of the population; all are well-known risk factors for AP.² Besides gallstones, alcohol abuse is the second most common cause of AP.¹ Although the pathophysiology of AP is incompletely understood, it is believed that in gallstone and alcoholic pancreatitis a chain of events is triggered by a temporary or permanent pancreatic duct obstruction. Locally this leads to activation and release of pancreatic enzymes into the pancreatic interstitium and peripancreatic tissues. When severe, autodigestion and necrosis occur.¹ Systemically, this results in release of inflammatory mediators termed cytokines, such as tumor necrosis factor, which is toxic to acinar cells. Cytokines activate and intensify the inflammatory cascade that may ultimately culminate in (multi)organ failure. Alternatively, the ischemia-reperfusion theory has been proposed as a unifying mechanism for AP; microcirculatory disturbances are the cause of local tissue injury and cytokine-

mediated inflammatory response that may lead to the systemic inflammatory response syndrome (SIRS) and the development of organ failure.^{3,4} There are evolving data that the magnitude of the inflammatory response, mediated by the immune system, is responsible for most of the morbidity and mortality in AP rather than the degree and extent of local pancreatic damage.^{5,6}

Clinically, severe AP is characterized by an early toxic phase with variable organ dysfunction lasting 1 to 2 weeks and a later phase dominated by the effects of local complications (primarily infected necrosis).^{5,7,8} Mortality from AP closely follows this biphasic pattern; in the first weeks, patients with severe AP die from sustained (multiple) organ failure, whereas in the later phase mortality can largely be attributed to infection of necrotic pancreatic and peripancreatic tissues, often superimposed by organ failure.^{5,8}

Over the past decades, several classification systems for AP have emerged, with the 1992 Atlanta Classification (AC) being the latest.⁹ The International Symposium on Acute Pancreatitis (Atlanta, Georgia, September 11–13, 1992) proposed a clinically based classification system that provided definitions for the disease, its severity, organ failure, and the local complications of AP.⁹ Better understanding of the pathophysiology of AP, improved diagnostic imaging of AP, and the development of minimally invasive radiologic, endoscopic, and operative techniques for the management of local complications have made it necessary to revise the AC.

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Important topics to be incorporated in a new state-of-the-art classification are assessment of clinical and morphologic severity, and recognition that there is no direct correlation between clinical severity and morphologic manifestations of AP. In addition, such a classification should make appropriate use of terms relating to peripancreatic collections; should include recognition of distinct entities, such as peripancreatic or extrapancreatic necrosis alone and walled-off necrosis; and should outline other important findings to be evaluated by contrast-enhanced computed tomography (CECT). Computed tomography (CT) is the most widely available imaging modality and is the standard for evaluation of AP. Magnetic resonance (MR) imaging and transabdominal or endoscopic ultrasonography (EUS) may also be used in specific situations. Both latter techniques depict more precisely the heterogeneity of peripancreatic collections and are superior to CECT in detecting the presence of nonliquid tissue components within collections¹⁰; however, because these techniques may not be readily available, the revised classification principally concentrates on CECT. The working group that revised the AC is composed of internationally renowned pancreatologists of different disciplines. These experts first convened in 2006, and many drafts of the revised AC have been circulating on the World Wide Web since then, some of which have been referred to in review articles in the radiologic^{11,12} and nonradiologic literature.^{13,14} The working document of the revised AC is, however, still in progress and probably will be published in its final form in 2012. Therefore, slight changes must be anticipated, in particular regarding definitions of clinical severity. This article reviews the CT features of AP and presents terms found in the latest version of the document describing the revised AC, with emphasis on the radiologic evaluation of AP.

DEFINITION OF ACUTE PANCREATITIS

The clinical diagnosis of AP requires 2 of the following 3 features:

1. Abdominal pain strongly suggestive of AP (epigastric, radiating to the back)
2. Serum amylase and/or lipase activity at least 3 times the upper limit of normal
3. Characteristic findings of AP on imaging, with CT the best, most universally available imaging modality.

If abdominal pain is strongly suggestive of AP but the serum amylase and/or lipase activity is less than 3 times the upper limit of normal, characteristic findings of AP on a CECT or MR imaging are required to confirm the diagnosis.

CT FINDINGS OF ACUTE PANCREATITIS

Most cases of AP are diagnosed clinically and do not require imaging for diagnosis. Sometimes, however, the clinical history and presentation of a patient are not straightforward, and a reliable imaging modality is needed to ascertain the diagnosis. Of all imaging techniques available, CT is the preferred imaging modality in the initial evaluation and in follow-up of AP. The sensitivity of CT for the diagnosis of AP is not known, particularly in mild cases, but a good-quality CECT demonstrates distinct pancreatic and peripancreatic abnormalities in most patients with moderate to severe AP.¹⁵

AP is a dynamic disease with continuously altering appearances on imaging. CECT findings of AP are time dependent. The development of morphologic and clinical complications approximately run along a well-described timetable, as observed in previous studies: parenchymal necrosis is infrequently found within the first day after onset of symptoms^{15,16}; infection of pancreatic and extrapancreatic necrosis needs several weeks to develop, with a peak incidence between the second and fourth weeks¹⁷; and full encapsulation of peripancreatic collections takes approximately 3 to 4 weeks.^{9,16}

On CT the mild, moderate, and severe morphologic forms of AP are staged by the CT Severity Index (CTSI) (Table 1), a radiologic grading system on a 10-point severity scale developed by Balthazar and colleagues¹⁸ that combines quantification of pancreatic/extrapancreatic inflammatory changes (0–4 points) with the extent of pancreatic parenchymal necrosis (0–6 points), both of which can be assessed by CECT. Besides prognostic information on patient morbidity and mortality, the CTSI depicts

Table 1
CT severity index

Characteristics	Points
Pancreatic inflammation	
Normal pancreas	0
Focal or diffuse enlargement of the pancreas	1
Peripancreatic inflammation	
Single acute peripancreatic fluid collection	3
Two or more acute peripancreatic fluid collections	4
Pancreatic parenchymal necrosis	
None	0
Less than 30%	2
Between 30% and 50%	4
More than 50%	6

the order in which morphologic manifestations in AP appear on CT.

In its mildest forms, AP is characterized by a normal (CTSI 0) or minimal increase in size of the pancreas caused by interstitial edema that might be focal or diffuse (CTSI 1) (**Fig. 1**). This feature is often not appreciated, however, when comparison with prior imaging is not available. Release of pancreatic enzymes in the interstitial space results in peripancreatic fat planes becoming blurred and thickened (CTSI 2) (**Fig. 2**). Peripancreatic extension of the inflammatory process is common because the pancreas does not have a capsule. At this stage, pancreatic and peripancreatic alterations generally resolve over time, leaving no residual findings.

As AP progresses, peripancreatic collections accumulate in and around the pancreas (CTSI 3 and 4) (**Fig. 3**). The most common sites of peripancreatic collections are the lesser sac, a potential space located directly anterior to the pancreas and posterior to the stomach, and the left anterior paranrenal space. Early peripancreatic collections lack a well-defined capsule and are confined by the anatomic space in which they arise. The natural course of these early collections is variable: they can persist or enlarge and evolve into encapsulated collections, or they can resolve spontaneously. The significance of peripancreatic collections in AP is their susceptibility to undergo secondary infection or hemorrhage or to cause symptoms attributable to mass effect.

The most severe morphologic forms of AP are denoted by the formation of pancreatic parenchymal necrosis, the extent of which is often classified in terms of less than 30%, between 30% and

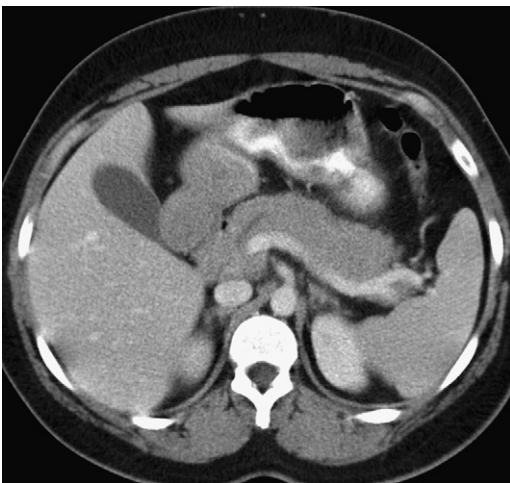


Fig. 1. Mild AP (ethanol-induced) in a 40-year-old man. Diagnosis was made on clinical grounds and elevated serum amylase and lipase. CT shows a slightly enlarged pancreatic gland. Grade B, no parenchymal necrosis; CTSI 1.

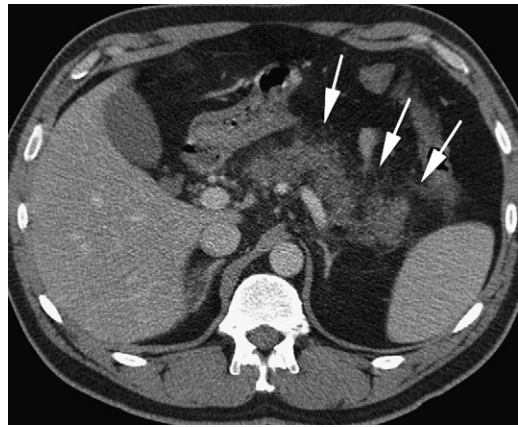


Fig. 2. Mild AP in a 45-year-old man with history of cholelithiasis. CT reveals an edematous pancreas, which is enlarged and heterogeneous. The peripancreatic fat planes are blurred and show increased attenuation, also called fat stranding (arrows). Grade C, no parenchymal necrosis; CTSI 2.

50%, and more than 50% (CTSI 5–6, 7–8, and 9–10, depending on the number of accompanying fluid collections, respectively) (**Fig. 4**). Parenchymal necrosis, defined as diffuse or focal areas of nonviable pancreatic parenchyma, tends to occur early in the course of the disease (within 4 days after onset of disease).^{16,19}

On CECT, the diagnosis of parenchymal necrosis is based on demonstrating a focal or diffuse area of parenchymal nonenhancement. Excellent correlation between the lack of pancreatic enhancement on CT and parenchymal necrosis is observed when the affected region is at least 3 cm or larger in diameter or involves more than one-third of the gland.^{16,20}

Although it is not necessary to perform CT imaging for all patients presenting with AP, it is generally recommended in the following circumstances:

1. When the clinical diagnosis is in doubt
2. For patients with organ failure or other reliable clinical or biochemical predictors of severe AP
3. For patients who are suspected to have a severe local complication (ie, intestinal ischemia or perforation)

Follow-up CT studies are reserved for patients who have severe morphologic findings on the index CT (such as parenchymal necrosis and peripancreatic fluid collections) together with clinical suspicion of complications, mainly infection of necrosis or hemorrhage, or when there is failure of clinical response to treatment.^{15,16} Finally, CT is helpful if intervention is contemplated because the information provided by the scan helps guide percutaneous, endoscopic, and surgical aspiration or drainage.

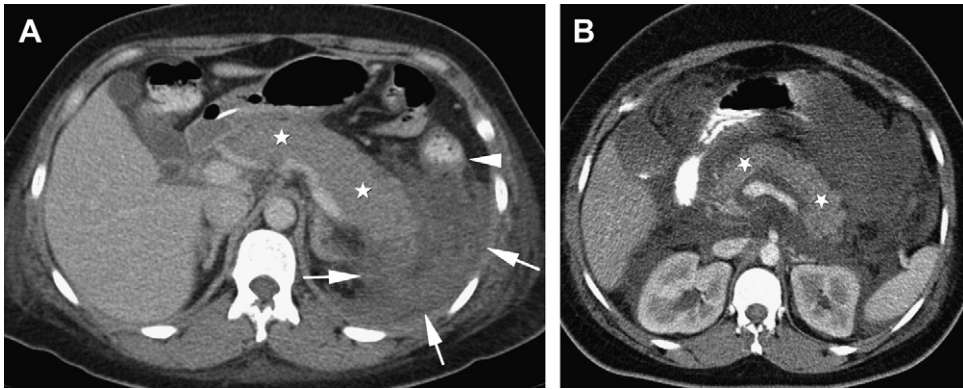


Fig. 3. Morphologically moderate severe AP (both of biliary cause) in 2 different patients. (A) Normal enhancement of the pancreas is observed (asterisks) with one heterogeneous peripancreatic collection in the left anterior pararenal compartment (arrows). There is slight wall thickening of the adjacent splenic flexure of the colon (arrowhead). Grade D, no parenchymal necrosis; CTSI 3. (B) Slightly swollen but normal enhancing pancreas is seen (asterisks), surrounded by multiple heterogeneous peripancreatic collections. Grade E, no parenchymal necrosis; CTSI 4.

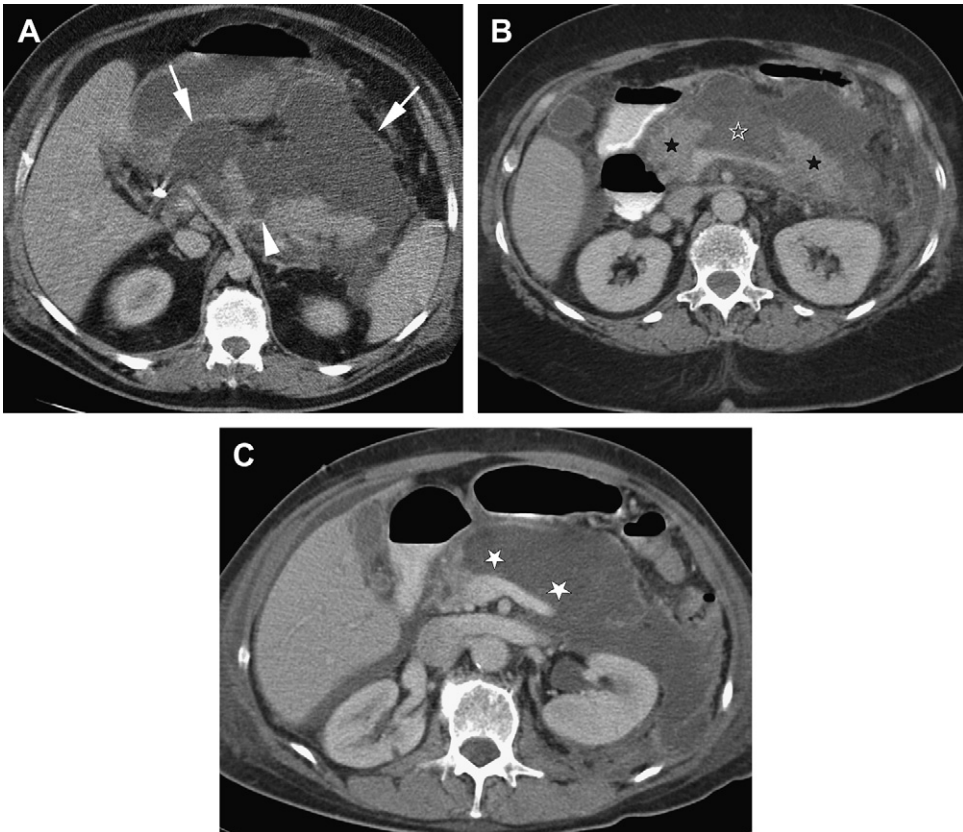


Fig. 4. Necrotizing pancreatitis in 3 different patients. (A) A 57-year-old woman with lack of enhancement (arrowhead) of a small part of the body of the pancreas (<30%) with surrounding acute necrotic collections (arrows) in lesser sac and left anterior pararenal compartment. Grade E, less than 30% parenchymal necrosis; CTSI 6. (B) A 45-year-old woman with biliary pancreatitis. CT reveals perfusion defect of approximately 30% to 50% at the body of the pancreas (white asterisk) with preserved enhancement of head and tail (black asterisks), surrounded by heterogeneous acute necrotic collections. Grade E, 30% to 50% parenchymal necrosis; CTSI 8. (C) A 44-year-old man with extensive necrosis of body and tail (asterisks) with surrounding partially encapsulated acute necrotic collections. Grade E, greater than 50% parenchymal necrosis; CTSI 10.

ATLANTA CLASSIFICATION: THE NEED FOR REVISION

The 1992 Atlanta symposium defined AP and classified complications of AP based on clinical criteria (Table 2).⁹ The AC attempted to offer a universally applicable classification system for AP, and represented an important step forward in defining and classifying the severity of AP. Before this symposium, most terms used to describe the morphologic entities (seen on imaging modalities and at operation) were understood differently among different pancreatologists, especially relating to pancreatic and peripancreatic collections.

Although the AC was useful for the successive 2 decades, many of the definitions proved confusing. A primary reason for this confusion has only become apparent over time: the AC is a clinical classification system that has been used by treating pancreatologists and radiologists to define the morphologic manifestations as depicted by radiologic imaging, in particular CECT. The communication problem that results not only is an issue between clinicians and radiologists but also is evident within the radiology community. A recent interobserver study using the AC to classify local complications found that when 5 abdominal radiologists did a blinded review of CT scans from 70 patients with severe AP, agreement on the appropriate definition occurred in only 3 cases (Fig. 5).²¹ Furthermore, several literature reviews showed that large variation existed in the interpretation of the Atlanta definitions^{22,23}; many reports on AP used alternative schema for predicting severity or terms to categorize actual severity and organ failure, and different definitions were applied to the contents of peripancreatic collections. For example, because of a lack of a precise morphologic description of the Atlanta terms pancreatic pseudocyst and pancreatic abscess, interpretation between pancreatologists varied widely, depending on the presence or

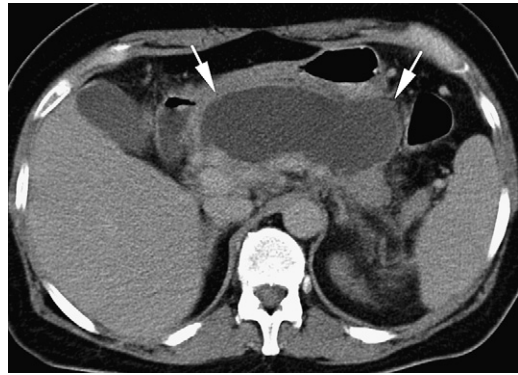


Fig. 5. CECT in a 39-year-old man with acute necrotizing pancreatitis 31 days after onset of symptoms. According to the original 1992 AC, this homogeneous and fully encapsulated collection (arrows) could be classified as pancreatic necrosis, pseudocyst, or even pancreatic abscess (all may have a similar appearance on CT). The patient suffered from fever despite antibiotic treatment. At surgery, infected necrosis was debrided and pus was drained.

absence of necrotic tissue as part of the collection.²³ Also, new descriptive terminology, such as extrapancreatic necrosis, necroma, organized necrosis, and other terms, have been suggested in the time after the AC in an attempt to reconcile the confusion but without success.²²

Insights in pathophysiology and treatment of AP have improved substantially in the past decades. Several studies showed that persistent rather than transient organ failure has a significant impact on patients' morbidity and mortality.^{24,25} With the introduction of new treatment modalities, such as percutaneous catheter drainage and transgastric and retroperitoneal minimally invasive necrosectomy, it has become essential in making decisions for patients with severe AP to provide accurate clinical staging, localization, and classification of morphologic changes in addition to defining the

Table 2
The original 1992 Atlanta Classification: local complications

Acute peripancreatic fluid collection	Occur early in the course of acute pancreatitis; are located in or near the pancreas; and always lack a wall of granulation or fibrous tissue
Pancreatic necrosis	A diffuse or focal area(s) of nonviable pancreatic parenchyma, which is typically associated with peripancreatic fat necrosis
Acute pseudocyst	A collection of pancreatic juice enclosed by a wall of fibrous or granulation tissue that arises as a consequence of acute pancreatitis or pancreatic trauma, or chronic pancreatitis
Pancreatic abscess	A circumscribed intra-abdominal collection of pus, usually in proximity to the pancreas, containing little or no pancreatic necrosis, which arises as a consequence of acute pancreatitis or pancreatic trauma

interrelationships between pancreatic collections and adjacent structures.²⁶

FUNDAMENTAL CONCEPTS

AP is conceptualized as a dynamic, evolving disease process. Patients with mild AP often have only transient SIRS with no significant systemic or local sequelae.²⁷ Two distinct phases, however, can be distinguished in patients presenting with nonmild AP.

In the early phase of nonmild AP, the clinical severity is dominated by the extent and duration of SIRS and organ failure that may result in death.^{5,6,24,27} At this stage, intervention is usually not performed unless an emergency laparotomy is indicated for complications such as abdominal compartment syndrome, bowel ischemia, or bowel perforation.²⁶ In the later phase of nonmild AP, morphologic changes of the pancreatic/peripancreatic region (ie, local complications) can become manifest systemically (ie, infected necrosis giving rise to bacteremia, organ failure, and sepsis), which may result in late mortality.^{7,8} This is the stage at which aggressive interventional (operative or minimally invasive) management has its benefits.²⁶ Thus, clinical severity parameters include death and the persistence rather than transient SIRS and organ failure, whereas morphologic severity is characterized by the presence of pancreatic parenchymal necrosis and peripancreatic collections, especially when superimposed by infection. However, the working group on the revised AC is still debating the clinical aspects of defining the severity of AP, and the reader is advised to consult the final version that will probably be published later in 2012.

Interrelation Between Clinical and Morphologic Severity

It is crucial to acknowledge that clinical and morphologic severity do not necessarily overlap and do not necessarily correlate with one another. Patients with clinically mild AP may show severe morphologic manifestations on CT (Fig. 6). Conversely, patients may sustain clinically severe disease whereas CT only shows minimal inflammatory changes. In these patients, clinical severity is often mainly driven by the presence of significant comorbid disease.²⁷

MORPHOLOGIC CLASSIFICATION

AP is subdivided into 2 morphologic types: acute interstitial edematous pancreatitis and acute necrotizing pancreatitis. Both types can be assessed using CECT or contrast-enhanced MR imaging.

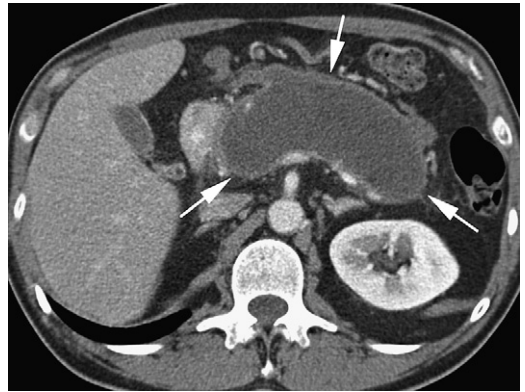


Fig. 6. Clinically mild AP in a 53-year-old man with acute necrotizing pancreatitis. CT was performed for continuing mild discomfort after a clinically mild AP. CT shows an encapsulated collection (arrows) located in the pancreatic bed with extensive parenchymal necrosis (>50% necrosis).

Morphologic Types of Acute Pancreatitis

Acute interstitial edematous pancreatitis

Most patients with AP will have diffuse or localized enlargement of the pancreas as a result of interstitial or inflammatory edema. On CECT, patients with interstitial edematous pancreatitis demonstrate diffuse or localized enlargement of the pancreas and normal enhancement of the pancreatic parenchyma. The pancreas often remains homogeneous, but at times can have a heterogeneous appearance depending on the amount of interstitial fluid (Fig. 7). Similarly, the retroperitoneal and peripancreatic tissues usually appear normal or show mild inflammatory changes characterized by haziness or mild stranding densities and varying but

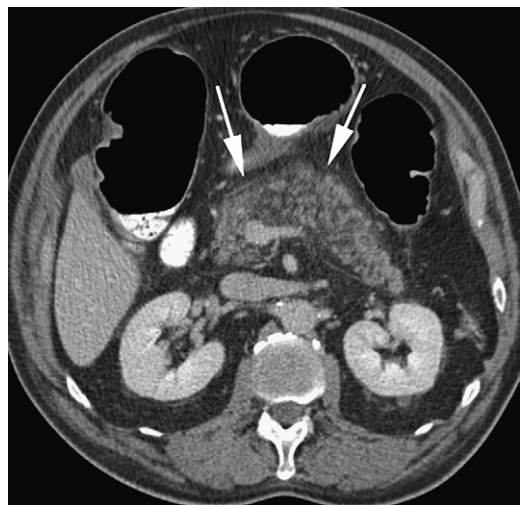


Fig. 7. Acute interstitial or edematous pancreatitis in a 65-year-old man. CT shows heterogeneous enhancement of an enlarged pancreatic gland with little surrounding fat stranding (arrows).

small amounts of nonenhancing areas of fluid density (acute peripancreatic fluid collections are discussed later). On occasion, an early CECT (done within the first days after onset of pancreatitis) exhibits diffuse heterogeneity in pancreatic parenchymal enhancement, which cannot be characterized definitively as interstitial edematous pancreatitis or patchy necrosis; with these findings, the presence or absence of pancreatic necrosis may have to be classified initially as indeterminate. A CECT done 3 to 7 days later should allow definitive classification.

Acute necrotizing pancreatitis

Necrosis can involve the pancreatic parenchyma and/or the peripancreatic tissues. The presence of necrosis in either the pancreatic parenchyma or the peripancreatic tissues defines the process as necrotizing pancreatitis and differentiates necrotizing pancreatitis from interstitial edematous pancreatitis. Necrotizing pancreatitis involves 1 of 3 subtypes: pancreatic parenchymal necrosis with peripancreatic necrosis (most common), pancreatic parenchymal necrosis alone (least common), or peripancreatic necrosis alone (without any discernible pancreatic parenchymal necrosis).

Pancreatic necrosis

Most patients with necrotizing pancreatitis have a variable extent of pancreatic parenchymal necrosis on CECT, evident by the lack of parenchymal enhancement after intravenous contrast administration.¹⁵ Pancreatic necrosis usually is associated with a variable extent of peripancreatic necrosis, but on rare occasions pancreatic necrosis alone is observed. At CECT, the pancreas shows diffuse or localized enlargement with one or more areas of non-enhancing pancreatic parenchyma (Fig. 8). The extent of pancreatic parenchymal necrosis is traditionally quantified in 3 categories: less than 30%, 30% to 50%, and greater than 50% of the total pancreatic parenchyma. CECT within a few days from onset of disease may underestimate the presence and true extent of parenchymal necrosis (Fig. 9) and, therefore, CECT should be preferably performed 3 to 7 days after onset of symptoms in patients with predicted severe AP (based on established clinical and biochemical systems, such as Marshall or APACHE II score, Glasgow criteria, and C-reactive protein level, among others).^{15,28}

Peripancreatic necrosis alone

The presence of (extensive) retroperitoneal peripancreatic fatty tissue necrosis secondary to AP, but with no recognizable pancreatic parenchymal necrosis on imaging and/or at operation, is recognized with increasing frequency.^{29–31} Peripancreatic necrosis may result from spread of inflammation from the surface of the pancreas or disruption of small

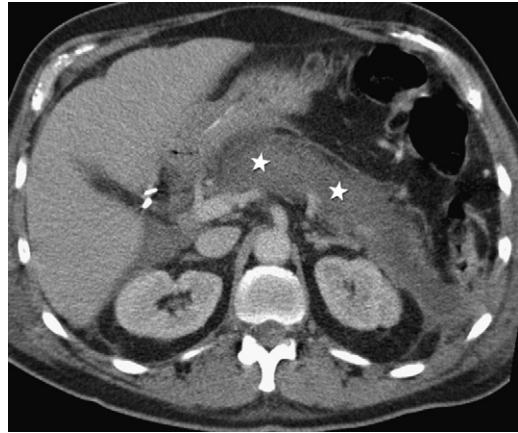


Fig. 8. Acute necrotizing pancreatitis in a 50-year-old woman with a prior history of cholecystectomy and choledocholithiasis. After endoscopic retrograde cholangiopancreatography, the patient developed extensive parenchymal necrosis of body and tail of the pancreas (asterisks) with accompanying acute necrotic collection in the left anterior pararenal compartment.

peripheral ductules with extravasation of activated pancreatic enzymes. This entity is recognized in up to 20% of the patients who require operative or interventional management of necrotizing pancreatitis.³¹ The distinction proves important clinically, because patients without recognizable pancreatic gland necrosis have a better prognosis and outcome than patients with pancreatic parenchymal necrosis.³¹

The presence or absence of necrosis in the peripancreatic tissues is more difficult to ascertain by CECT, especially early in the course of the disease. CECT may suggest peripancreatic necrosis by the presence of heterogeneous areas of variable density containing liquid and nonliquid components in one or more areas, especially in the regions of the retroperitoneum and subperitoneal spaces of the mesenteries (Fig. 10). If concern is great enough, MR imaging or ultrasonography may aid in the recognition of nonliquid, necrotic components within the peripancreatic collection.

Characteristics of necrosis

The relative amount of liquid versus nonliquid components within areas of necrosis may vary with the time from onset of necrotizing pancreatitis. Necrosis should be thought of as a continuum; as time evolves, the necrosis that is initially nonliquid is believed to liquefy or reabsorb gradually. Thus, early in the course of the disease (in the first 1–2 weeks), the necrosis may appear as a non-enhancing area of variable density on CECT. Later in the course of the disease (≥ 4 weeks), it may appear as homogeneous (Fig. 11) and low in attenuation. During this stage, CECT may not be able to

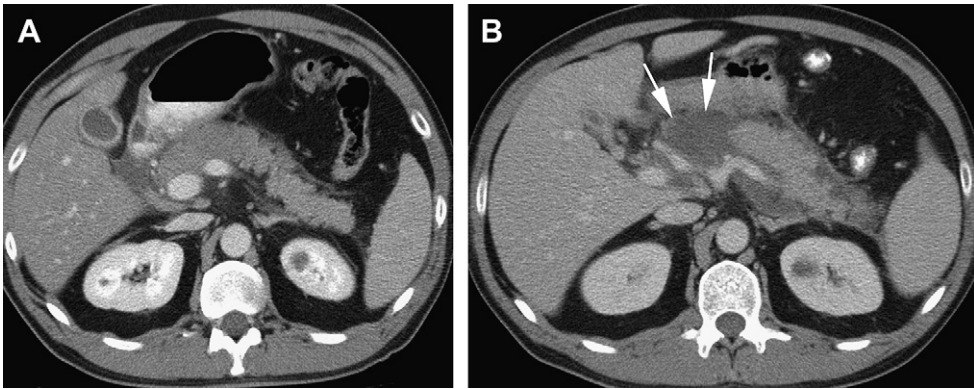


Fig. 9. False-negative parenchymal necrosis on early CT. (A) CECT on day of admission shows minimal hypoperfusion of the neck of the pancreas compared with the remainder of the pancreas. Parenchymal necrosis was not reported in the radiologic report. (B) Follow-up CT examination 7 days later reveals definitive perfusion defects of the neck and small part of the body of the pancreas (arrows), indicating parenchymal necrosis.

differentiate nonliquid, necrotic content from liquid content; MR imaging or ultrasonography may be necessary to do so if deemed necessary clinically. Complete resolution of necrosis (weeks or months to years later) may occur through complete liquefactive necrosis and reabsorption (Fig. 12). In some patients, complete liquefaction or reabsorption may never occur. This phenomenon relates to the amount of necrotic debris; up to 2 cm of necrotic tissue is reabsorbed or liquefied, but necrotic tissue that is larger than 5 cm rarely completely disappears (Fig. 13).³²

Sterile and infected necrosis

According to the absence or presence of infection in the necrotic pancreatic and/or peripancreatic

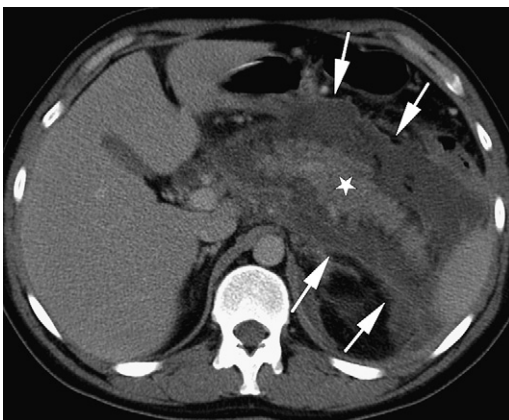


Fig. 10. Acute necrotizing pancreatitis (peripancreatic necrosis alone) in a 40-year-old man. CT shows normal enhancing pancreas (asterisk) surrounded by heterogeneous acute necrotic collections (arrows) with fluid and nonliquid (fat) densities, suggestive for peripancreatic necrosis alone. Two weeks later, operative debridement of necrotic peripancreatic tissues was performed because of infection of peripancreatic tissues.

areas, the entities of sterile and infected necrosis are distinguished. Distinction between sterile and infected necrosis is essential because they have different natural history, treatment, and prognosis.^{8,26} Patients with sterile necrosis rarely need surgery or intervention except in the few patients who may remain persistently unwell after the initial SIRS phase despite optimized intensive medical management. Conversely, most patients with infected necrosis require long-term antibiotic therapy and some kind of interventional treatment.²⁶ Infection of necrosis can be diagnosed based on image-guided fine-needle aspiration and culture, or on the presence of extraluminal gas bubbles within pancreatic or peripancreatic collections on CECT (Fig. 14). This finding is virtually pathognomonic and reflects the presence of a gas-forming organism. In rare cases, perforation and spontaneous fistulization of an adjacent hollow viscus will produce gas locules in peripancreatic collections.¹⁵

Definition of Peripancreatic Collections

In the nonmild forms of AP, peripancreatic collections arise that may develop into local complications requiring supportive measures or interventional treatment. The morphologic characteristics are well depicted by CECT, and this modality forms the basis of the new definitions. The latest version of the revised AC discerns 4 distinct types of peripancreatic collections in the acute and subacute phase of AP and 1 type of collection that may occur during long-term follow-up after an episode of AP (Table 3). Differences between each of the 5 peripancreatic collections depend on:

1. The morphologic type of AP (interstitial edematous or necrotizing pancreatitis)

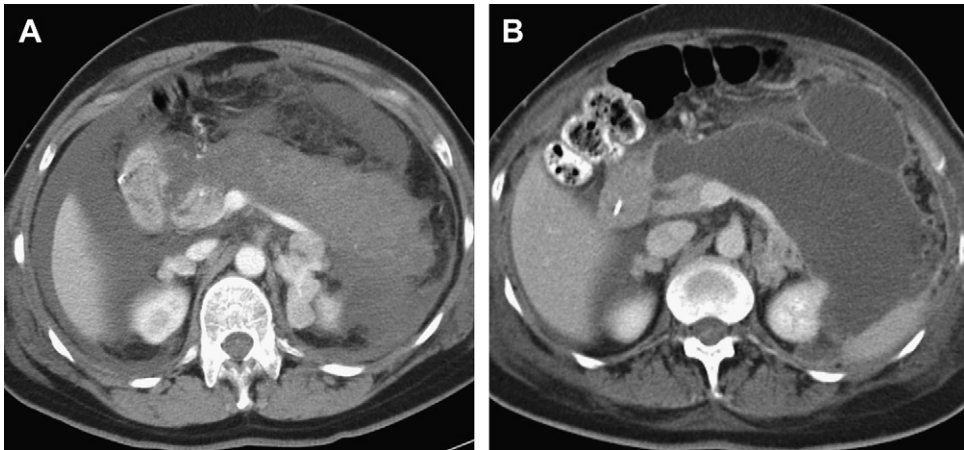


Fig. 11. Characteristics of parenchymal necrosis in a 35-year-old woman with drug-induced pancreatitis, on serial CT scans. (A) Initial CT study shows a heterogeneous appearance of the necrotic pancreatic neck, body, and tail caused by severe hypoperfusion (necrosis). Note the hyperenhancement of hyperplastic adrenal glands, probably stress related. (B) Follow-up examination performed 17 days later reveals a more homogeneous appearance, likely attributable to liquefaction necrosis.

2. The location (pancreatic, peripancreatic, or both)
3. The presence and degree of encapsulation (none, partially, or complete)
4. The contents (fluid and/or nonliquid necrotic debris)
5. The bacterial status (sterile or infected)
6. Prior interventional therapy (necrosectomy)
7. The age of the collection, a factor that relates to the degree of encapsulation.

For assessment at CECT, the most important determinants distinguishing each definition relate to the collections' contents (fluid alone vs a combination of fluid and nonliquid, necrotic debris) and the degree of encapsulation.

In interstitial edematous pancreatitis, collections composed only of fluid are termed acute peri-

pancreatic fluid collection and pseudocyst, depending on the age of the collection. In acute necrotizing pancreatitis, collections composed of necrotic material and fluid in varying degrees are termed acute necrotic collection and walled-off necrosis, depending on the age of the collection. The collection termed postnecrosectomy pseudocyst occurs in the setting of prior necrosectomy. All of these entities can be either sterile or infected, although it is highly unusual for an acute peripancreatic fluid collection to be infected.

Acute peripancreatic fluid collection

Acute peripancreatic fluid collections result from local edema related to parenchymal and/or peripancreatic inflammation (exudate); they either have no connection with the ductal system or they occur

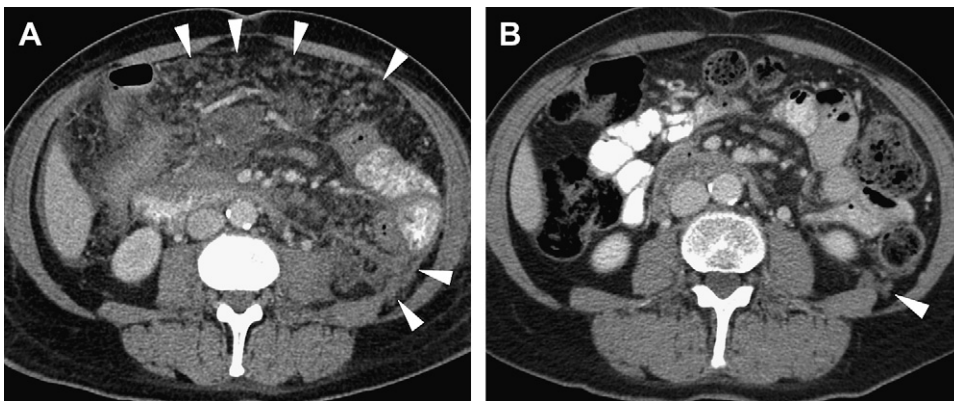


Fig. 12. Reabsorption of fat necrosis. (A) Multiple ill-defined nodules are seen scattered throughout the mesenteries, greater omentum (4 arrowheads), and retroperitoneal region, particularly on the left (2 arrowheads). (B) Follow-up CT 2 weeks later reveals resolution of the nodules, except for small residual nodules in the left anterior pararenal compartment (arrowhead).

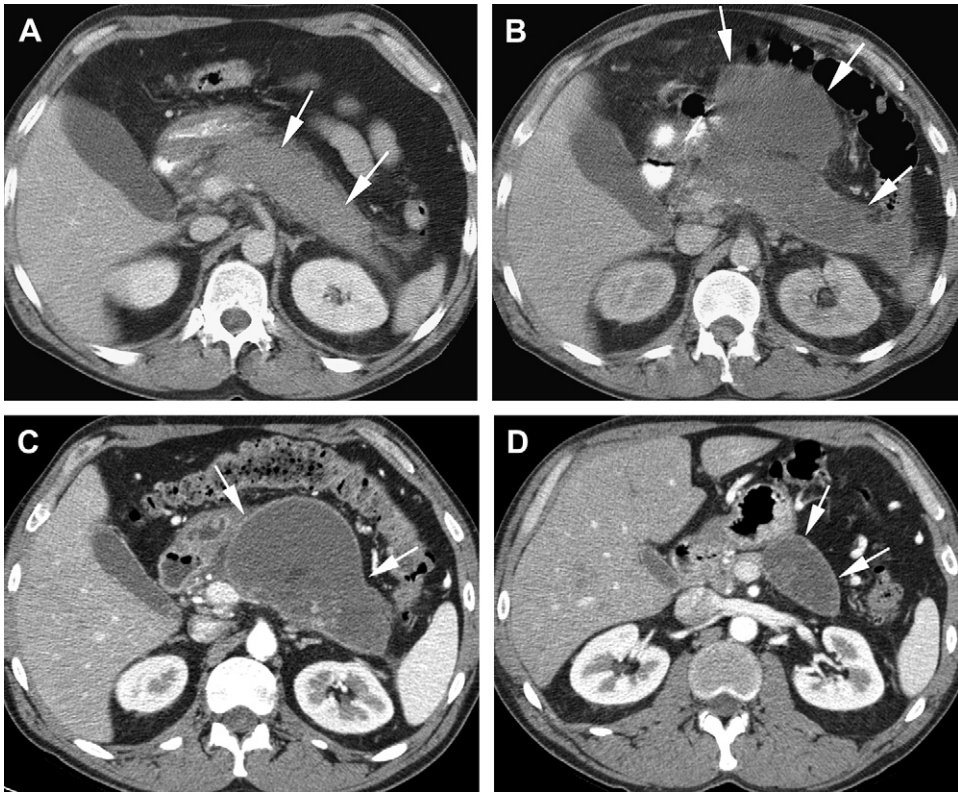


Fig. 13. Serial CT scans in a patient with persistent necrotic collections. (A) Initial CT shows extensive necrosis of body and tail (arrows). (B) Two weeks later, progressive peripancreatic collections are observed (arrows). Patient recovered without need for intervention during hospitalization. (C) One year after onset of acute necrotizing pancreatitis a large, slightly heterogeneous peripancreatic collection is seen with a well-defined thin wall (arrows). The patient was asymptomatic. (D) Two years after the acute attack a persistent heterogeneous collection is observed (arrows), although remarkably diminished in size.

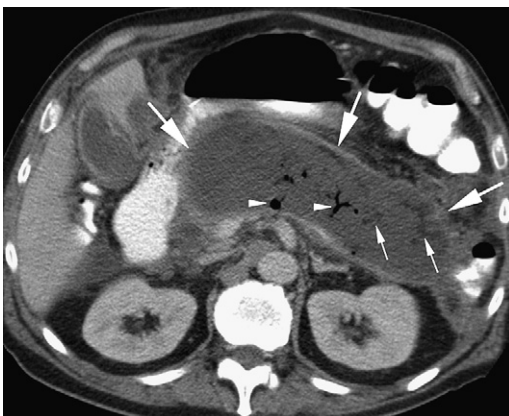


Fig. 14. Infected pancreatic necrosis. A 53-year-old woman with necrotizing pancreatitis developed spiking fever and impending organ failure 29 days after onset of symptoms. CT shows extensive necrosis (>50%) and an encapsulated heterogeneous collection (arrows) with liquid and nonliquid (fat) densities (small arrows), as well as impacted gas bubbles (arrowheads), indicative of infected necrosis.

from rupture of a small, peripheral, pancreatic duct side branch. These collections arise within a few days of onset of interstitial edematous pancreatitis and may persist for several weeks. Acute peripancreatic fluid collections contain fluid only and thus, per definition, have no necrotic components. Most acute peripancreatic fluid collections remain sterile and are reabsorbed spontaneously within the first several weeks after onset of AP.³³

At CECT, acute peripancreatic fluid collections exist predominantly adjacent to the pancreas (peripancreatic), have homogeneous fluid density, have no complete definable wall (partial encapsulation is, however, allowed), and are confined by the normal peripancreatic fascial planes (Fig. 15). These acute peripancreatic fluid collections should be differentiated from areas where ascites resides (perihepatic and perisplenic spaces, paracolic gutters, and pelvic region).

Acute necrotic collection

Early pancreatic and peripancreatic collections in patients with acute necrotizing pancreatitis are

Table 3
Revised Atlanta Classification: CT criteria for local pancreatic complications

Local Complication	Morphologic CT Criteria
Acute peripancreatic fluid collection	Typically less than 4 weeks old after onset of symptoms Occurs in interstitial edematous pancreatitis Homogeneous collection with fluid density Confined by normal peripancreatic fascial planes No fully definable wall encapsulating the collection Adjacent to pancreas (no intrapancreatic extension)
Pseudocyst	Typically older than 4 weeks after onset of symptoms Occurs in interstitial edematous pancreatitis Homogeneous collection (round or oval) with fluid density Well-defined wall (ie, completely encapsulated) Absence of nonliquid component Adjacent to pancreas (no intrapancreatic extension)
Acute necrotic collection	Typically less than 4 weeks old after onset of symptoms Occurs in necrotizing pancreatitis Heterogeneous collection with liquid and nonliquid density and varying degrees of loculation No fully definable wall encapsulating the collection Located intrapancreatic and/or extrapancreatic
Walled-off necrosis	Typically older than 4 weeks after onset of symptoms Occurs in necrotizing pancreatitis Heterogeneous collection with liquid and nonliquid density and varying degrees of loculations Well-defined wall (ie, completely encapsulated) Located intrapancreatic and/or extrapancreatic
Post-necrosectomy Pseudocyst	Occurs after necrosectomy for necrotizing pancreatitis Homogeneous collection (round or oval) with fluid density Well-defined wall (ie, completely encapsulated) Absence of nonliquid component Located intrapancreatic and/or extrapancreatic

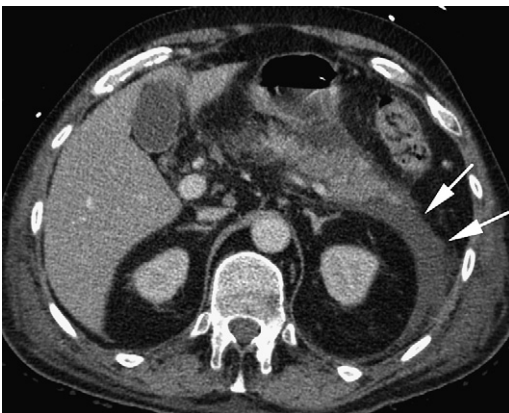


Fig. 15. Acute peripancreatic fluid collection (<4 weeks). CT shows normal enhancement of the pancreas with a fluid collection extending into the left anterior pararenal compartment (*arrows*). The fluid collection lacks a well-defined wall and conforms to the anatomic space in which it arises. The acute peripancreatic fluid collection resolved spontaneously.

termed acute necrotic collections. Acute necrotic collections represent a combination of parenchymal and/or peripancreatic fat necrosis together with pancreatic secretions. It is believed that the necrotic component of acute necrotic collections occurs following the liberation of activated pancreatic enzymes in the pancreatic and peripancreatic area, leading to (peri)pancreatic fat saponification and necrosis. Thus, the contents of acute necrotic collections are a spectrum ranging from predominantly necrotic (nonliquid) to both fluid and necrotic material (with varying degrees of each component), and generally also contain areas of loculations or septa.

On CECT, acute necrotic collections are depicted as homogeneous (fluid density) or heterogeneous (combination of fluid and nonliquid densities) in the pancreatic and/or peripancreatic area (**Fig. 16**). An acute necrotic collection has no capsule or is only partially encapsulated. It remains uncertain as to what proportion of peripancreatic fluid collections contains necrotic debris because CECT, the most

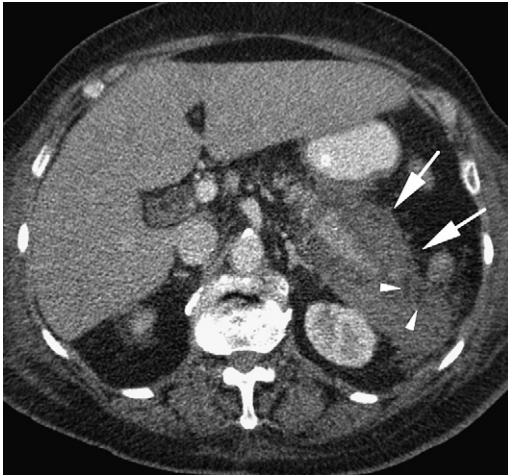


Fig. 16. Acute necrotic collection (<4 weeks). At the tail of the pancreas a heterogeneous, poorly encapsulated collection is observed (arrows) with fluid and nonliquid (fat) densities (arrowheads), indicative of peripancreatic tissue necrosis.

commonly used imaging tool in AP, does not reliably depict necrotic debris inside collections, especially within the first week after onset of AP. This shortcoming implies that peripancreatic collections often cannot be categorized readily into an acute peripancreatic fluid collection or an acute necrotic collection. Both may appear as homogeneous, non-enhancing areas of low (fluid) density; these collections should then be termed indeterminate peripancreatic collections. After the first week or two, however, acute necrotic collections should become evident on CECT, MR imaging, transabdominal ultrasonography, or EUS.

Pseudocyst

The definition of a pseudocyst remains the same: a collection of pancreatic juice (analysis usually shows increased amylase and lipase level) enclosed by an encapsulated wall of granulation tissue, with little or no associated necrotic material (<5%). Pseudocysts develop from acute peripancreatic fluid collections (not from acute necrotic collections). Thus they occur in the setting of interstitial edematous pancreatitis. In general, more than 4 weeks are required for formation of a well-defined wall that differentiates a pseudocyst from an acute peripancreatic fluid collection.

At CECT, a pseudocyst is depicted as well-circumscribed, usually round or oval, homogeneous peripancreatic collection with fluid attenuation, surrounded by a well-defined wall (Fig. 17); an MR image or ultrasonography may be required to confirm the absence of necrosis within the fluid collection. The identification of a recognizable ductal communication can sometimes, but not reliably, be determined by

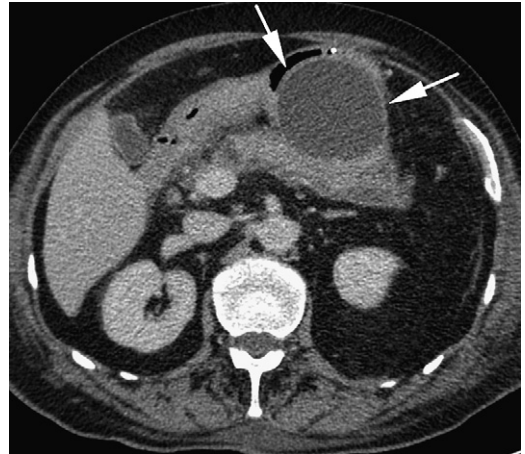


Fig. 17. Acute pseudocyst (>4 weeks). CT shows a normal enhancing pancreas with a homogeneous fully encapsulated collection, with fluid density in the lesser sac abutting the stomach (arrows).

CECT. MR imaging or EUS may allow this communication to be identified; the presence or absence of ductal communication may be important in determining appropriate therapy but is not required as a criterion for a pseudocyst in this classification. It must be stressed that in AP, as opposed to chronic pancreatitis, pseudocysts are rare because most persistent peripancreatic collections contain necrotic material.

Walled-off necrosis

As the necrosis in necrotizing pancreatitis matures, the interface between the necrosis and the adjacent viable fatty tissue becomes established by an encapsulating thickened wall without an epithelial lining. The term walled-off necrosis is introduced to describe this evolution of necrosis to an encapsulated, well-defined collection of pancreatic fluid and necrotic debris. This entity, referred to previously in the literature often as organized pancreatic necrosis,^{10,34} necroma,^{35,36} or pseudocyst associated with necrosis,³⁷ represents the end stage of an acute necrotic collection. This entity was not recognized in the original AC. Just as with acute necrotic collections, walled-off necrosis more commonly involves the pancreatic parenchyma with areas of peripancreatic necrosis as well, the peripancreatic tissues alone or, on rare occasion, the pancreatic parenchyma alone.

On CECT, walled-off necrosis manifests as a full encapsulated collection with varying densities (fluid and/or nonliquid attenuation) in, around, or remote from the pancreatic area (Fig. 18). A complete encapsulating wall (often requiring more than 4 weeks) differentiates walled-off necrosis from an acute necrotic collection.

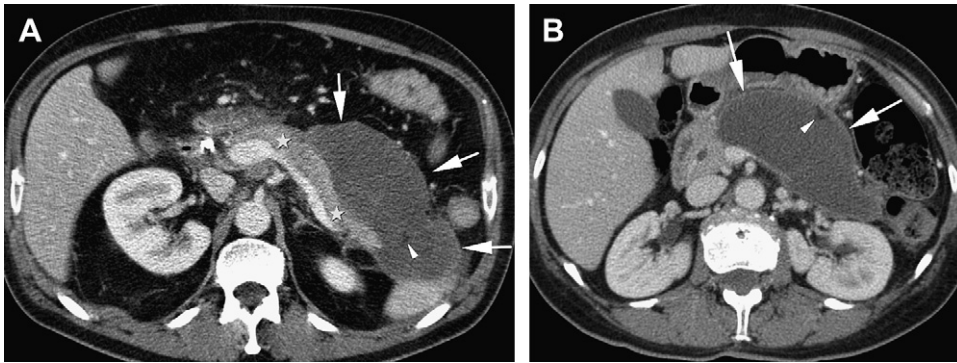


Fig. 18. Walled-off necrosis in peripancreatic necrosis alone (>4 weeks) (A) and pancreatic necrosis (B). (A) CT depicts adjacent to a normally perfused body and tail of the pancreas (*asterisks*), a well-defined heterogeneous collection (*arrows*) with primarily fluid density and scattered areas of fat density (*arrowhead*), indicative of necrosis of peripancreatic tissues. (B) A large, heterogeneous fully encapsulated collection is seen (*arrows*) with predominantly fluid density mixed with nonliquid (fat) density (*arrowhead*). The collection replaces the greater part of the pancreatic parenchyma. Because of the heterogeneity and presence of pancreatic necrosis, this is not a pseudocyst.

The CECT differentiation between a pseudocyst and walled-off necrosis is probably the most difficult, and a source of common mistakes made by radiologists because both entities may have a similar appearance.^{35,36} The term pseudocyst is often applied to every collection in and around the pancreas, even to cystic pancreatic neoplasms, leading to significant problems in patient management; symptomatic pseudocysts require only simple drainage, whereas walled-off necrosis frequently but not always requires removal of necrotic material. The term pseudocyst should be applied only to patients with interstitial edematous pancreatitis (with one exception, discussed later)

who have an acute peripancreatic fluid collection evolving into a round or oval collection with a well-defined wall, whereas walled-off necrosis arises in necrotizing pancreatitis. MR imaging, transabdominal ultrasonography, or EUS may be a valuable complementary test for differentiation between pseudocysts and walled-off necrosis by documenting the presence of necrotic material within the collection (**Fig. 19**).

Postnecrosectomy pseudocyst

An exception of a pseudocyst arising in acute necrotizing pancreatitis is in the setting of a condition termed disconnected duct syndrome.³⁸ These

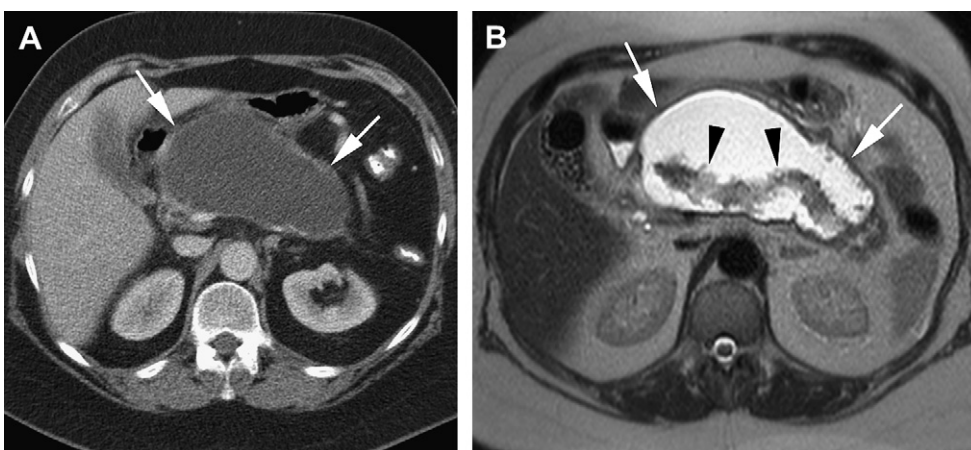


Fig. 19. CT (A) and corresponding MR imaging (B) in a 45-year-old woman who was thought to have a large symptomatic pseudocyst after acute gallstone-induced pancreatitis. (A) CT reveals a well-circumscribed homogeneous collection (*arrows*) in the pancreatic region exerting mass effect on the stomach. (B) Corresponding T2-weighted MR imaging discloses the heterogeneity of the thin-walled collection (*arrows*) with fluid intensity and dark material, most likely representing necrotic pancreatic tissue (*arrowheads*), which was not recognized by CT (note also the presence of gallstones). Consequently, endoscopic necrosectomy (rather than simple drainage) was performed, with subsidence of patient's symptoms.

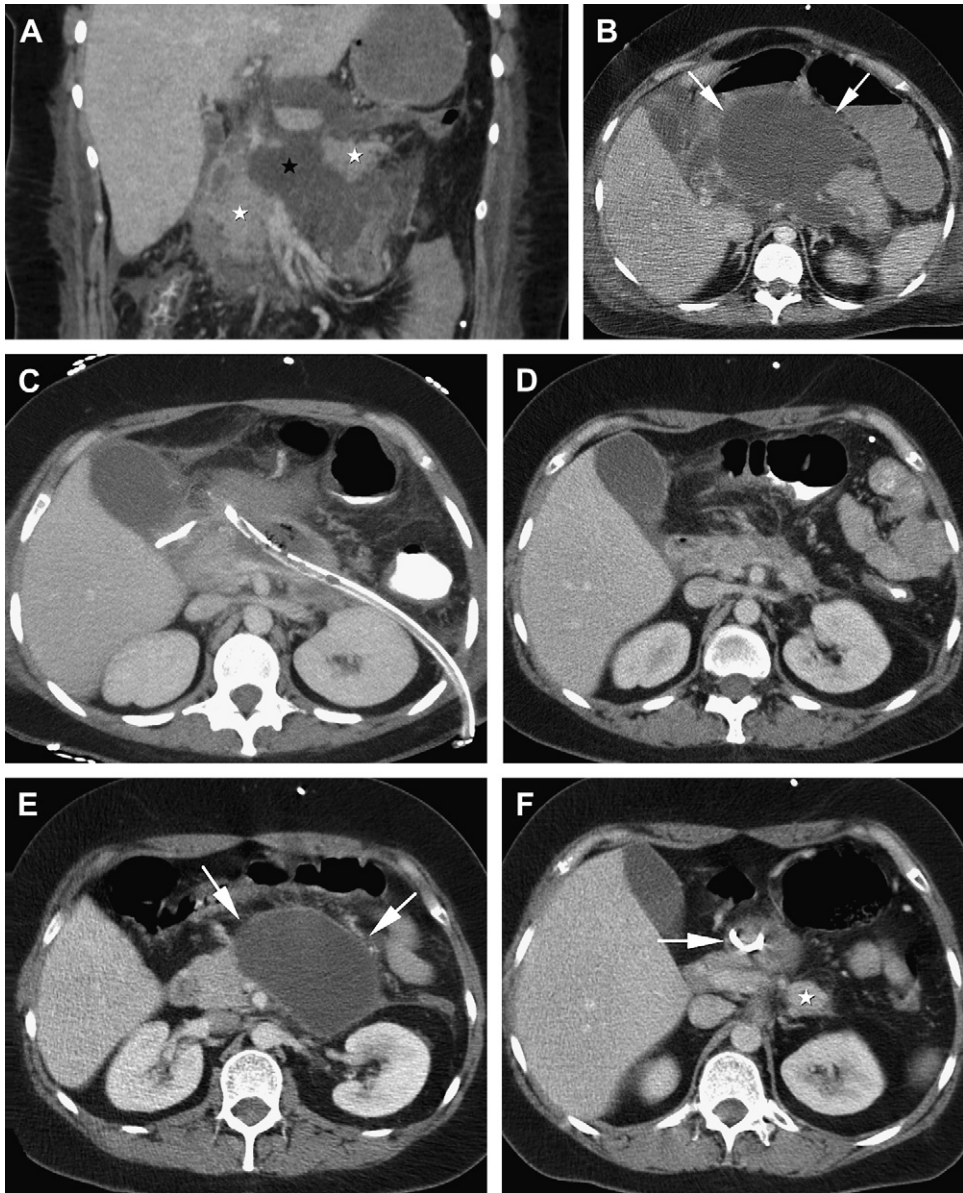


Fig. 20. Typical sequence of events in a case of central gland necrosis and disconnected duct syndrome with post-necrosectomy pseudocyst formation. (A) Coronal reconstructed CT depicts necrosis of the pancreatic neck and part of the body (*black asterisk*) among the viable head and tail (*white asterisks*). (B) Axial CT, performed 12 days later, shows an increase of the partially encapsulated acute necrotic collection (*arrows*). The patient developed fever and elevated white blood cell count, suggestive of infected necrosis. Subsequently, CT-guided percutaneous drainage was performed. (C) Axial thick-slab maximum-intensity projection CT shows the percutaneous drain from the left retroperitoneal compartment into the area of infected necrosis. After additional endoscopic necrosectomy, the patient recovered completely. (D) Axial CT obtained 6 months after removal of the drains shows pancreatic necrosis has resolved. (E) Follow-up CT 1 year later shows a recurrent collection, which is homogeneous and surrounded by a thin wall; a postnecrosectomy pseudocyst (*arrows*). (F) Endoscopic pigtail drainage (*arrow*) was performed, with complete resolution of the collection. Note the viable remnant pancreatic tail (*asterisk*).

patients have necrosis of the central portion of the pancreatic gland with a viable pancreatic tail remnant. Initially, a walled-off necrosis is formed and potentially requires necrosectomy. When the residual cavity (devoid of necrotic material)

continues to have ductal communication with the remnant secreting pancreatic tail, the cavity fills with pancreatic fluid, forming a postnecrosectomy pseudocyst (**Fig. 20**). These recurrent collections appear months to years after the onset of AP,

and thus do not occur during the acute or subacute phase of AP. A prior history of some kind of (surgical or endoscopic) necrosectomy is a prerequisite for diagnosis of this type of collection. Rarely, a pseudocyst may develop during long-term follow-up when walled-off necrosis undergoes complete liquefaction or reabsorption of necrotic material.

RADIOLOGIC EVALUATION ON CECT: PANCODE SYSTEM

Prior studies have shown that interobserver agreement for characterization of the original Atlanta definitions by CECT was poor,²¹ as opposed to the excellent interobserver agreement when applying morphologic terms for describing findings in and around the pancreas using the PANCODE system (Table 4).³⁹ PANCODE is an acronym that stands for pancreatic nonenhancement, collections, and description, and can be used to assist in complete and systematic reporting of all pancreatic and extrapancreatic abnormalities in patients with AP.

Pancreatic Nonenhancement

The first assessment is the presence or absence of areas of pancreatic nonenhancement (indicative of parenchymal necrosis). Note the sites of perfusion defects and estimate the percentage of involved necrotic parenchyma.

Collections

The next assessment concerns the presence of pancreatic and peripancreatic collections. Peripancreatic collection refers to any (fluid) collection that exceeds peripancreatic fat stranding.

Description

Describe the sites of collections and their relation with the pancreas or other organs, the degree of encapsulation, the homogeneity and attenuation, mass effect on surrounding structures, and their shape and size, and note the presence of gas bubbles. Note and describe extrapancreatic complications, such as gastrointestinal, biliary, and vascular complications, as well as involvement of adjacent parenchymal abdominal organs

Table 4
CT description using the PANCODE system

1. Pancreatic nonenhancement	
Assess perfusion defects	No pancreatic perfusion defects, uniform contrast enhancement Pancreatic perfusion defect(s): Location: head, neck, body, tail Extent: <30%, 30%–50%, >50% Patchy or full width
2. Collections	
Assess presence of peripancreatic collections	No collections, only enlarged pancreas with or without peripancreatic fat stranding Presence of one or more peripancreatic collections
3. Description	
Peripancreatic collections	Assess: Location: intrapancreatic and/or extrapancreatic or separate Anatomic site(s) (eg, lesser sac, anterior pararenal space) Shape and maximum size (if possible) Homogeneity and attenuation (explicitly note hemorrhage) Degree of encapsulation (none, partial, or complete) Presence of (impacted) gas bubbles or gas-fluid levels
Extrapancreatic complications	Gastrointestinal (gastric outlet obstruction, bowel ischemia, ileus) Vascular (venous thrombosis/compression with collateralization or segmental portal hypertension, hemorrhage, and arterial pseudoaneurysm) Biliary (cholecystolithiasis, choledocholithiasis, dilation of biliary system, signs of cholecystitis) Solid organ involvement (eg, liver, spleen, kidney) Ascites Pleural effusion

(liver, spleen, and kidney) by the inflammatory process and effects of systemic complications (ascites and pleural fluid).

SUMMARY

This review presents an update rather than a final version of the revised AC that replaces the 1992 Atlanta definitions on AP; in any case, terms such as pancreatic abscess, and other antiquated terms such as phlegmon and hemorrhagic pancreatitis, are not advised for use, and therefore should no longer appear in radiologic reports. Incorporation of fundamental concepts on clinical and morphologic grounds, new insights into the pathophysiology, and the recognition of the dynamic nature of severe AP will be among the important features of this revised classification.

The clinical severity of AP is still under debate by the members of the working group, and the reader is advised to consult the final version of the revised AC, likely to be published later in 2012.

Fluid and/or necrotic collections developing in the pancreas and peripancreatic region as shown on imaging, notably CECT, are classified as early (<4 weeks) and late (>4 weeks) collections. The proposed terms of the local pancreatic complications use morphologic descriptors based on CECT criteria and age.

The early collections are classified as either an acute peripancreatic fluid collection, which develops in the course of interstitial edematous pancreatitis and represents a collection of fluid without associated necrosis, or an acute necrotic collection, which occurs in acute necrotizing pancreatitis that may involve necrosis of the pancreatic parenchyma plus the peripancreatic tissues, the peripancreatic tissue alone, or the pancreatic parenchyma alone. In the first week after onset of AP, it may be difficult to accurately classify collections located exclusively in the peripancreatic region on CECT, and these are then referred to as indeterminate peripancreatic collections. A persistent, late collection with a complete encapsulating wall on imaging should be called a pseudocyst if the collection contains only fluid; it should be called walled-off necrosis if it arose in the setting of necrotizing pancreatitis and contains variable amounts of necrosis and fluid involving the pancreatic parenchyma and the peripancreatic tissues, the peripancreatic tissues alone, or the pancreatic parenchyma alone. Post-necrosectomy pseudocyst is a recurrent fully encapsulated collection, composed only of fluid that arises after necrosectomy for necrotizing pancreatitis.

Imaging is an important feature of this revised AC, and radiologists are important members of the multidisciplinary team of specialists involved in the care and treatment of patients with this potentially devastating disease. Correct use of the revised AC definitions should allow objective communication and conveyance of CT findings to clinicians. In the end, patients with severe AP are best served by close collaboration between radiologists and clinicians for planning the appropriate treatment.

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