Revised Atlanta Classification for Acute Pancreatitis: A Pictorial Essay[1]

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Abbreviations: ANC = acute necrotic collection, APFC = acute peripancreatic fluid collection, IEP = interstitial edematous pancreatitis, WON = walled-off necrosis

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SA-CME LEARNING OBJECTIVES

After completing this journal-based SA-CME activity, participants will be able to:

■ Discuss the revised Atlanta classification system.

■ Distinguish interstitial edematous pancreatitis from necrotizing pancreatitis at imaging.

■ Describe the imaging appearance of pancreatitis-associated collections, including acute peripancreatic fluid collections, pseudocysts, acute necrotic collections, and walled-off necrosis.

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The 2012 revised Atlanta classification is an update of the original 1992 Atlanta classification, a standardized clinical and radiologic nomenclature for acute pancreatitis and associated complications based on research advances made over the past 2 decades. Acute pancreatitis is now divided into two distinct subtypes, necrotizing pancreatitis and interstitial edematous pancreatitis (IEP), based on the presence or absence of necrosis, respectively. The revised classification system also updates confusing and sometimes inaccurate terminology that was previously used to describe pancreatic and peripancreatic collections. As such, use of the terms *acute pseudocyst* and *pancreatic abscess* is now discouraged. Instead, four distinct collection subtypes are identified on the basis of the presence of pancreatic necrosis and time elapsed since the onset of pancreatitis. Acute peripancreatic fluid collections (APFCs) and pseudocysts occur in IEP and contain fluid only. Acute necrotic collections (ANCs) and walled-off necrosis (WON) occur only in patients with necrotizing pancreatitis and contain variable amounts of fluid and necrotic debris. APFCs and ANCs occur within 4 weeks of disease onset. After this time, APFCs or ANCs may either resolve or persist, developing a mature wall to become a pseudocyst or a WON, respectively. Any collection subtype may become infected and manifest as internal gas, though this occurs most commonly in necrotic collections. In this review, the authors present a practical image-rich guide to the revised Atlanta classification system, with the goal of fostering implementation of the revised system into radiology practice, thereby facilitating accurate communication among clinicians and reinforcing the radiologist's role as a key member of a multidisciplinary team in treating patients with acute pancreatitis.

Introduction

Beginning in 2007, the Acute Pancreatitis Classification Working Group polled an international cohort of pancreatic experts, including 11 pancreatic societies, with the goal of revising the original 1992 Atlanta classification system for acute pancreatitis (1,2). Although the original Atlanta classification system established common terms for acute pancreatitis and related complications, the nomenclature proved to be confusing, leading to incorrect use of terms in both clinical practice and research. In addition, advances in imaging and in the understanding of pathophysiology necessitated substantial revision of the classification system (2). A draft was posted on the Pancreas Club Web site in 2008 (3). This draft was referrenced in several initial publications in the radiology literature (4–6). After a total of four Web-based revisions, the revised Atlanta classification system was finalized in 2012 and published shortly thereafter, with updates to the earlier-cited draft (2).

TEACHING POINTS

- In the 1st week after the onset of pancreatitis, imaging findings correlate poorly with clinical severity, and imaging sensitivity for necrotizing pancreatitis is decreased in the first few days. In addition, any local complications that are detected in the 1st week generally do not necessitate intervention because treatment is based on supportive measures and management of organ failure. Initial imaging is most useful when performed 5–7 days after hospital admission, when local complications have developed and pancreatic necrosis (if present) should be clearly distinguishable.
- In the revised classification system, new definitions were created to clearly stratify acute pancreatitis into two subcategories based on imaging findings: IEP and necrotizing pancreatitis. IEP is more common and represents nonnecrotizing inflammation of the pancreas. The entire pancreas will enhance at contrast-enhanced CT or MR imaging, with no unenhanced (necrotic) areas, although enhancement of the gland may be less avid than that of the normal pancreas owing to interstitial edema. IEP usually manifests with focal or diffuse pancreatic enlargement and is typically surrounded by wispy peripancreatic inflammation or a small amount of fluid. In addition, there should be no surrounding peripancreatic necrotic collections in IEP, although there may be surrounding fluid-containing collections. Necrotizing pancreatitis accounts for 5%–10% of cases of acute pancreatitis. It is important to understand that necrosis may involve either the pancreatic parenchyma or the peripancreatic tissues and in both cases is termed necrotizing pancreatitis. There are three subtypes of necrotizing pancreatitis; the subtypes are based on the anatomic area of necrotic involvement: *(a)* pancreatic only, *(b)* peripancreatic only, and *(c)* combined pancreatic and peripancreatic.
- The revised Atlanta classification makes an important distinction between collections that contain purely fluid (those encountered in nonnecrotizing pancreatitis and IEP) and collections that contain necrotic debris in addition to fluid (those encountered in necrotizing pancreatitis). The terms acute pseudocyst and pancreatic abscess have been abandoned. Similarly, the use of the term pseudocyst in radiology reporting to describe any pancreatitis-related collection is misleading to treating physicians, since the term implies that these collections always contain purely fluid, which is not the case in necrotic collections. Instead, the revised classification includes new definitions that more accurately describe the various types of collections encountered: APFC, pseudocyst, ANC, and WON. The important distinctions for classifying collections correctly are the time course (≤4 weeks or >4 weeks from onset of pain) and the presence or absence of necrosis at imaging.
- If an APFC has not resolved within 4 weeks, it becomes more organized, with development of a capsule that manifests as an enhancing wall at contrast-enhanced CT. At this point, the collection is referred to as a pseudocyst, and, since there is no necrosis, it should contain only fluid with no nonliquefied components. If there is even a small area of fat or soft-tissue attenuation in an otherwise fluid-attenuation collection, the diagnosis is not pseudocyst but WON.
- Any collection can be sterile or infected, although infection occurs far more frequently in necrotic collections. Clinically, infection is suspected in a previously stable patient who experiences decompensation with signs of infection. The only imaging finding of an infected collection is the presence of gas within the collection. Wall enhancement is not a reliable indicator of infection, since it is invariably present in mature collections (pseudocyst and WON).

One major component of the revised classification system that is of particular importance to radiologists is the manner in which pancreatitis-associated collections (local complications) are described and named (Table 1). It was well recognized by the working group that previously used terms such as *acute pseudocyst* and *pancreatic abscess* were confusing and had fallen out of favor, in addition to their not being entirely descriptive of the pathologic condition. Therefore, a specific lexicon was needed to distinguish necrotic from nonnecrotic collections (7). Use of standardized reporting schemas and application of a well-defined lexicon to a specific disease are thought to be important for effective communication and quality care and are in fact becoming more common in everyday radiologic practice (8,9). Most important, adoption of such standardized terminology allows the radiologist to be an effective member of a multidisciplinary team in the diagnosis and treatment of acute pancreatitis.

The purpose of this article is to serve as an image-rich practical overview of the revised Atlanta classification system such that the radiologist will have a working understanding of the system and can immediately incorporate the lexicon into clinical practice. To that end, we focus on key imaging characteristics that help differentiate collections that often have a similar appearance, describe various infectious complications, and discuss imaging pitfalls. In addition, we will propose a reporting schema to facilitate accurate communication.

Acute Pancreatitis: Overview and New Diagnostic Criteria

Acute pancreatitis is an acute inflammatory condition, with a range of severity as well as various local and systemic complications. Gallstones and alcohol are the first and second most common causes of acute pancreatitis, respectively, and additional variants occur when patients are stratified by sex. In 2009, acute pancreatitis was the most common cause of hospital admission for gastrointestinal disorders in the United States, with approximately 275 000 admissions, nearly double the number in the previous decade. This increase is thought to be secondary not only to nationwide increases in obesity and the incidence of gallstones, but also to more sensitive and more frequently used laboratory testing (10). A majority of patients have mild acute pancreatitis, which carries essentially no risk of mortality. In the subset of patients with organ failure (severe disease) or infected necrosis, however, the mortality rate reaches 30% (2,11). The revised Atlanta classification requires that two or more of the following criteria be met for the diagnosis of acute pancreatitis: *(a)* abdominal pain suggestive of pancreatitis, *(b)* serum amylase or lipase level greater than three times the upper normal value, or *(c)* characteristic imaging findings (2). Contrast material–enhanced CT is most commonly used to fulfill the radiologic

Table 1: Pancreatic and Peripancreatic Collections

Collection	Time after Onset of Pain (wk)	Pancreatitis Subcategory	Location	Imaging Features
APFC	≤4	IEP	Extrapancreatic	Homogeneous, fluid attenuation, conforms to retroperitoneal structures, no wall
ANC	≤4	Necrotizing pancreatitis	Intra- and/or extrapancreatic	Inhomogeneous*, nonliquefied components†, no wall
Pseudocyst	>4	IEP	Extrapancreatic‡	Homogeneous, fluid filled, circumscribed, encapsulated with wall
WON	>4	Necrotizing pancreatitis	Intra- and/or extrapancreatic	Inhomogeneous, nonliquefied components, encapsulated with wall

Sources.—References 2–4.
Note.—Any collection may become infected. ANC = acute necrotic collection, APFC = acute peripancreatic fluid collection, IEP = interstitial necrotizing pancreatitis, WON = walled-off necrosis.
*Early ANCs may be homogeneous; follow-up computed tomography (CT) performed in 2nd week may help clarify status.
†Includes solid-appearing components or fat globules within fluid.
‡Rarely, persistent pancreatic leak or disconnected duct may lead to intrapancreatic pseudocyst.

criterion, but magnetic resonance (MR) imaging is also appropriate. Although many patients will meet the criteria for acute pancreatitis on the basis of symptoms and laboratory results alone and may not require imaging initially, imaging may be performed early in the disease course when the cause of the disease is unclear, to look for causative factors such as choledocholithiasis and pancreatic cancer. Imaging for the diagnosis of pancreatitis is also appropriate when abdominal pain suggests pancreatitis but the amylase or lipase level is not elevated to the threshold value, which is often the case at delayed presentation (12). The onset of pancreatitis is considered to coincide with the 1st day of pain, not the day on which the patient presents for care or the day of hospital admission (2).

Phases of Acute Pancreatitis

In pathophysiologic terms, acute pancreatitis is divided into early and late phases. The early phase occurs in the 1st week after onset, with the disease manifesting as a systemic inflammatory response. At this time, clinical severity and treatment are mainly determined on the basis of type and degree of organ failure. The late phase, which generally starts in the 2nd week and can last for weeks to months, occurs only in patients with moderately severe or severe pancreatitis, as defined by persistent organ failure and by local complications (2).

Grading of Severity of Acute Pancreatitis

To improve the stratification of patients at the time of presentation, the pancreatitis severity scale was updated in the revised Atlanta classification. The original classification categorized patients as having either severe or mild pancreatitis on the basis of the presence or absence of organ failure, respectively (1). However, emerging evidence indicated that a large subset of patients with local complications experienced substantial morbidity but little mortality (11). Therefore, a third category was added to the new classification—moderately severe acute pancreatitis—to describe this patient group (2). Organ failure and local complications are not seen in patients with mild pancreatitis, who are usually discharged within the 1st week, with very low mortality (13). These patients rarely require CT for local complications, and imaging may be useful only in assessing the cause of pancreatitis (eg, ultrasonography [US] or MR cholangiopancreatography for choledocholithiasis) (12).

Moderately severe acute pancreatitis manifests in patients with transient organ failure lasting less than 48 hours and/or local or systemic complications. Systemic complications are generally comorbidities exacerbated by pancreatitis, such as acute kidney injury in the setting of chronic renal failure. Local complications include a variety of pancreatic and peripancreatic collections. Such collections generally develop in the 2nd week (the late phase of pancreatitis) and are clinically suspected in patients with unremitting or recurrent pain, a secondary peak in pancreatic enzyme levels, worsening organ dysfunction, or sepsis (2). These symptoms should prompt imaging studies such as (in order of preference) contrast-enhanced CT, contrast-enhanced MR imaging, or unenhanced MR imaging (12).

Severe disease is characterized by organ failure that persists for more than 48 hours. Because

Table 2: Modified Marshall Scoring System

Organ System	Score 0	Score 1	Score 2	Score 3	Score 4
Respiratory*	>400	301–400	201–300	101–200	≤100
Renal: serum creatinine (mg/dL)	≤1.4	1.5–1.8	1.9–3.5	3.6–4.9	≥5
Cardiovascular: systolic blood pressure (mm Hg)	>90	<90, responding to fluid resuscitation	<90, not responding to fluid resuscitation	<90 with pH <7.3	<90 with pH <7.2

Sources.—References 2,4,14.
Note.—A score of 2 or higher indicates organ failure, with transient failure lasting less than 48 hours and persistent failure lasting more than 48 hours.
*Partial pressure of oxygen/fraction of inspired oxygen, or Pao_2/Fio_2.

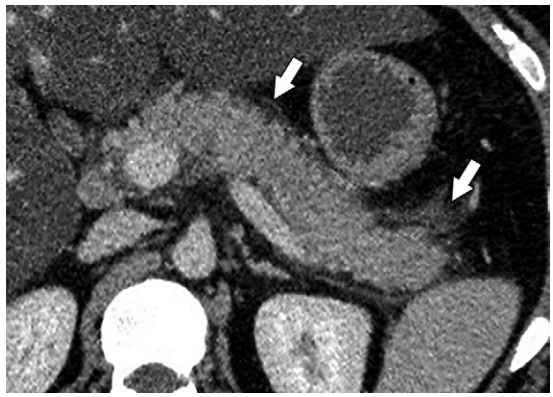


Figure 1. IEP in a 28-year-old man with alcohol-related pancreatitis. Axial contrast-enhanced CT image shows wispy peripancreatic inflammation (arrows) with normal pancreatic enhancement and no collections.

organ failure plays a key role in determining disease severity, an accurate definition is essential for clinical management of acute pancreatitis. The modified Marshall scoring system (Table 2) is endorsed in the revised Atlanta classification as the primary method for determining organ failure. The modified Marshall scoring system incorporates measurements from the respiratory, cardiovascular, and renal systems, with a score of 2 or higher for any system indicating organ failure (2,14). In the 1st week after the onset of pancreatitis, imaging findings correlate poorly with clinical severity, and imaging sensitivity for necrotizing pancreatitis is decreased in the first few days (2). In addition, any local complications detected in the 1st week generally do not necessitate intervention because treatment is based on supportive measures and management of organ failure (15). Initial imaging is most useful when performed 5–7 days after hospital admission, when local complications have developed and pancreatic necrosis (if present) should be clearly distinguishable (2).

IEP versus Necrotizing Pancreatitis

In the revised classification system, new definitions were created to clearly stratify acute pancreatitis into two subcategories based on imaging findings: IEP and necrotizing pancreatitis. IEP is more common and represents nonnecrotizing inflammation of the pancreas. The entire pancreas will enhance at contrast-enhanced CT or MR imaging, with no unenhanced (necrotic) areas, although enhancement of the gland may be less avid than that of the normal pancreas owing to interstitial edema. IEP usually manifests with focal or diffuse pancreatic enlargement and is typically surrounded by wispy peripancreatic inflammation or a small amount of fluid (Fig 1). In addition, there should be no surrounding peripancreatic necrotic collections in IEP, although there may be surrounding fluid-containing collections (Fig 2).

Necrotizing pancreatitis accounts for 5%–10% of cases of acute pancreatitis (2). It is important to understand that necrosis may involve either the pancreatic parenchyma or the peripancreatic tissues and in both cases is termed *necrotizing pancreatitis*. There are three subtypes of necrotizing pancreatitis; the subtypes are based on the anatomic area of necrotic involvement: *(a)* pancreatic only, *(b)* peripancreatic only, and *(c)* combined pancreatic and peripancreatic. The latter subtype is the most common, accounting for 75% of cases. The combined subtype demonstrates nonenhancing pancreatic parenchyma, as well as nonenhancing heterogeneous peripancreatic collections, and typically accumulating in the lesser sac and anterior pararenal space (Fig 3a). Peripancreatic necrosis alone, in which the pancreas enhances normally but the peripancreatic tissues show necrosis, with collections containing variable amounts of fluid and nonliq-

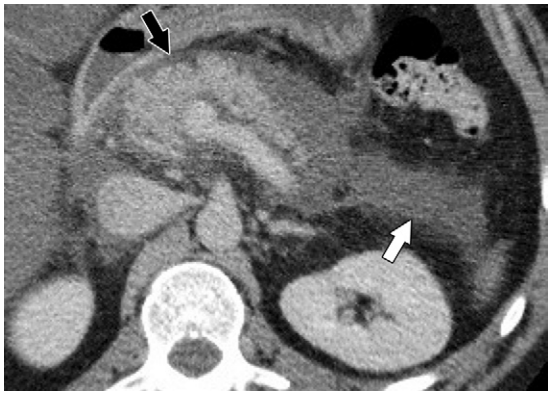


Figure 2. IEP in a 43-year-old man. Axial contrast-enhanced CT image shows peripancreatic inflammation (black arrow) and a homogeneous fluid-attenuation collection in the left anterior pararenal space (white arrow), a finding that is consistent with APFC.

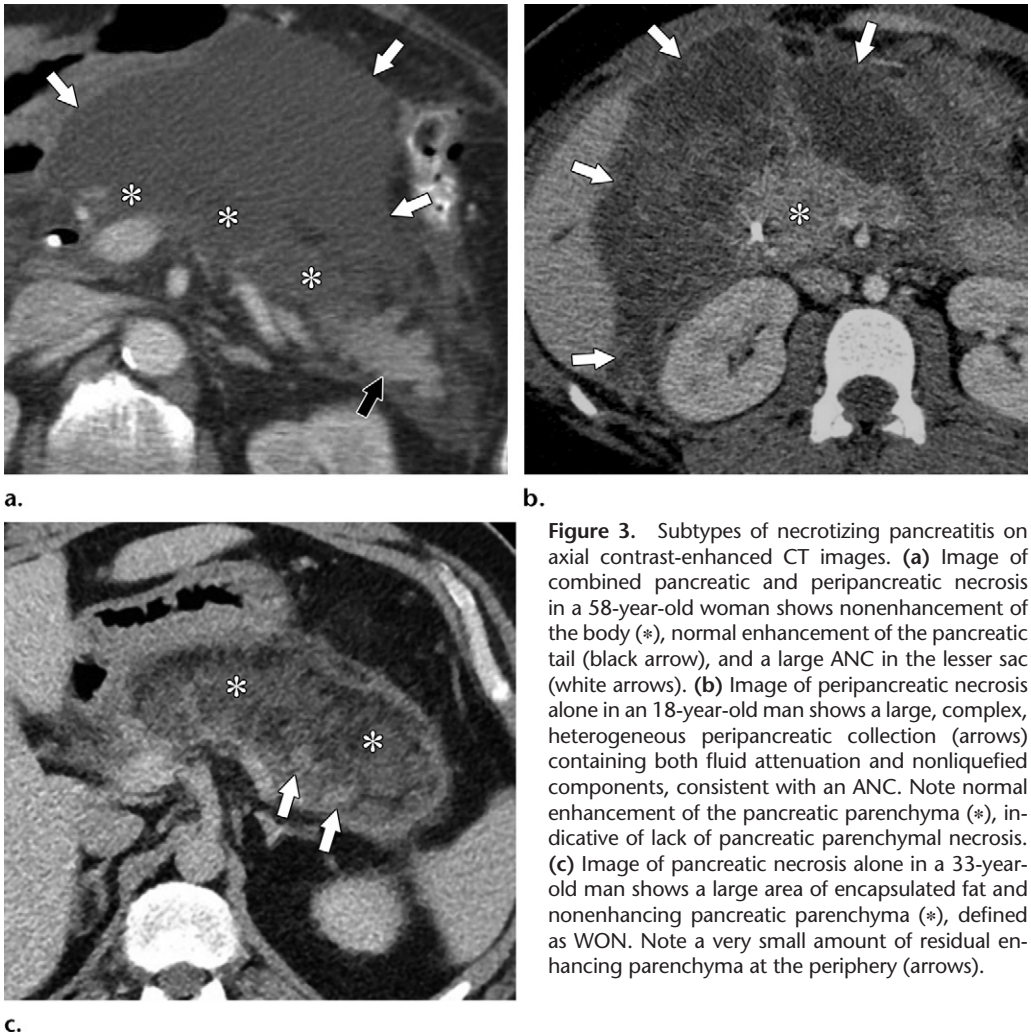


Figure 3. Subtypes of necrotizing pancreatitis on axial contrast-enhanced CT images. **(a)** Image of combined pancreatic and peripancreatic necrosis in a 58-year-old woman shows nonenhancement of the body (*), normal enhancement of the pancreatic tail (black arrow), and a large ANC in the lesser sac (white arrows). **(b)** Image of peripancreatic necrosis alone in an 18-year-old man shows a large, complex, heterogeneous peripancreatic collection (arrows) containing both fluid attenuation and nonliquefied components, consistent with an ANC. Note normal enhancement of the pancreatic parenchyma (*), indicative of lack of pancreatic parenchymal necrosis. **(c)** Image of pancreatic necrosis alone in a 33-year-old man shows a large area of encapsulated fat and nonenhancing pancreatic parenchyma (*), defined as WON. Note a very small amount of residual enhancing parenchyma at the periphery (arrows).

uefied components, occurs in 20% of cases (Fig 3b). Pancreatic necrosis alone is the least common subtype, occurring in 5% of cases, and lacks peripancreatic collections (Fig 3c). When imaging is performed within the first few days of disease onset, necrosis may not be detected because the pancreas can appear edematous and globally or focally hypoenhancing and, as such, may be indistinguishable from IEP. In these cases, repeat contrast-enhanced CT performed 5–7 days later is more accurate for the diagnosis of necrotizing pancreatitis (2). In general, routine contrast-enhanced CT is adequate for diagnosis, and pancreatic protocol multiphase imaging (arterial and portal phase) is typically unnecessary. If renal failure prevents administration of intravenous contrast material, nonenhanced MR imaging is preferred to nonenhanced CT, although limited availability and the difficulty of imaging critically ill patients are significant limitations of MR

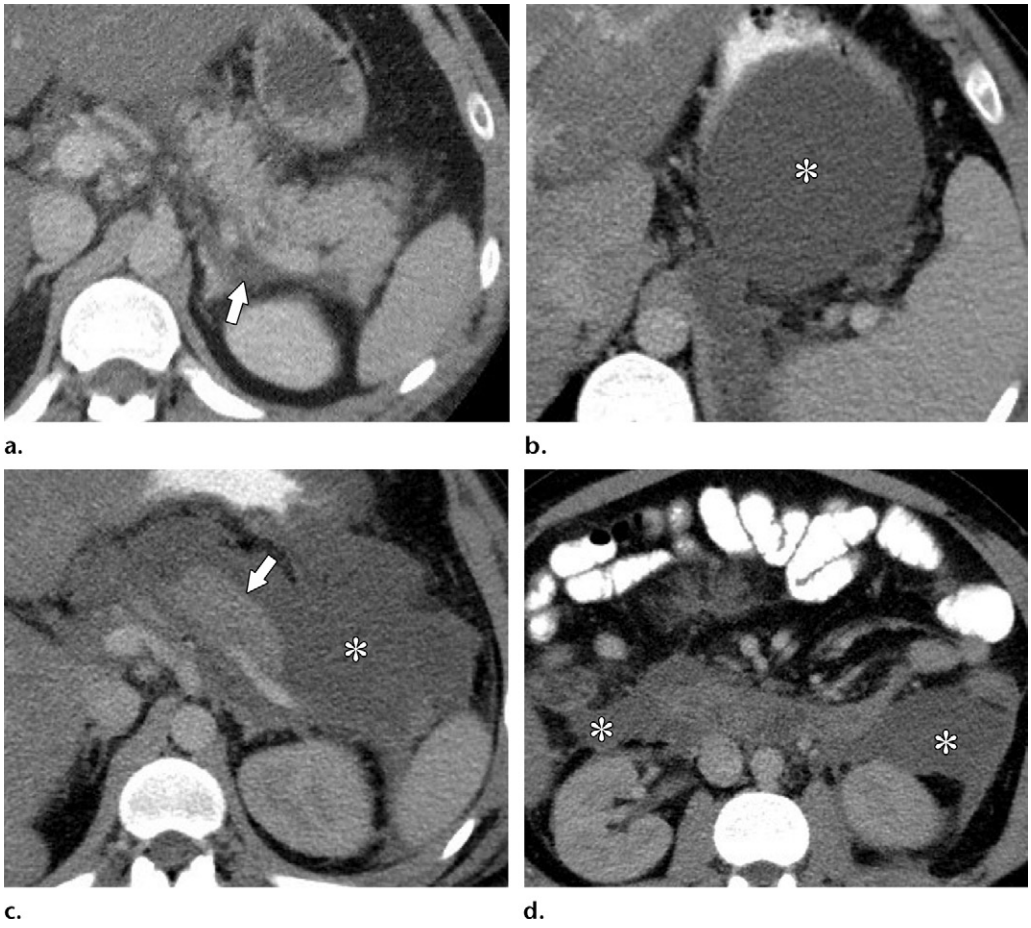


Figure 4. IEP with APFCs on axial contrast-enhanced CT images in a 49-year-old man. **(a)** One day after the onset of pain, IEP is seen with a small, homogeneous, fluid-attenuation collection in the left anterior pararenal space (arrow). The pancreas demonstrates normal enhancement. **(b–d)** At 10 days after onset of pain, multiple homogeneous fluid collections (*) can be seen in the lesser sac **(b)** and peripancreatic **(c)** and bilateral anterior pararenal spaces **(d)**, findings consistent with APFCs. Arrow on **c** = normally enhancing pancreatic tail.

imaging, and nonenhanced CT may be easier in practice (12).

Pancreatic and Peripancreatic Collections

The revised Atlanta classification makes an important distinction between collections that contain purely fluid (those encountered in IEP) and collections that contain necrotic debris in addition to fluid (those encountered in necrotizing pancreatitis). The terms *acute pseudocyst* and *pancreatic abscess* have been abandoned. Similarly, the use of the term *pseudocyst* in radiology reporting to describe any pancreatitis-related collection is misleading to treating physicians, since the term implies that these collections always contain purely fluid, which is not the case in necrotic collections. Instead, the revised classification includes new definitions that more accurately describe the various types of collections encountered: APFC, pseudocyst, ANC, and WON. The important distinctions for classifying collections correctly are the time course (≤4 weeks or >4 weeks from onset of pain) and the presence or absence of necrosis at imaging (Table 1) (2).

APFC and Pseudocyst

APFCs occur during the first 4 weeks and are present only in patients with IEP. Because the pathogenesis involves inflammation without necrosis, APFCs contain only fluid and are visualized as homogeneous fluid-attenuation collections that lack a wall and tend to conform to the retroperitoneal spaces (Fig 4). APFCs are always peripancreatic in location. If a similar-appearing collection is seen within the pancreatic parenchyma, it is by definition an ANC, and the diagnosis is no longer IEP but necrotizing pancreatitis (2). Most APFCs resolve spontaneously, and drainage should be not be performed because of the risk of infecting an otherwise sterile collection (16). Just as some cases of IEP and necrotizing pancreatitis can be difficult to distinguish at contrast-enhanced CT during the 1st week, it may also be difficult to distinguish between an APFC and an ANC. In general, repeat imaging at

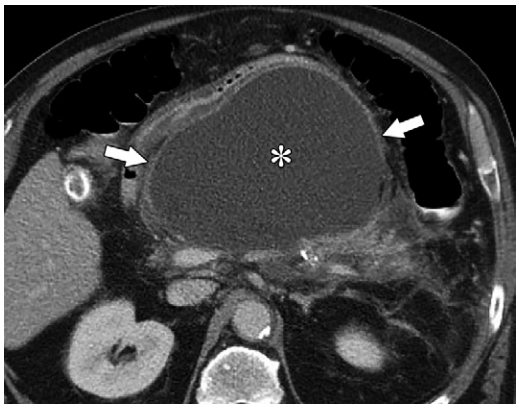


Figure 5. Pseudocyst in a 36-year-old man. Axial CT image obtained 6 weeks after onset of gallstone pancreatitis shows a large homogeneous fluid collection in the lesser sac (*) with no nonliquefied components and a thick enhancing wall (arrows), findings that are consistent with pseudocyst.

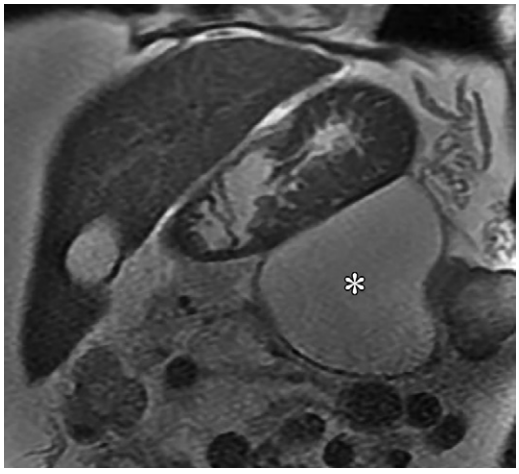


Figure 6. Pseudocyst in a 48-year-old woman. Coronal T2-weighted MR image obtained 8 weeks after onset of pancreatitis shows a large homogeneous collection in the lesser sac (*) with uniformly hyperintense fluid signal and no necrotic components, findings that are consistent with pseudocyst.

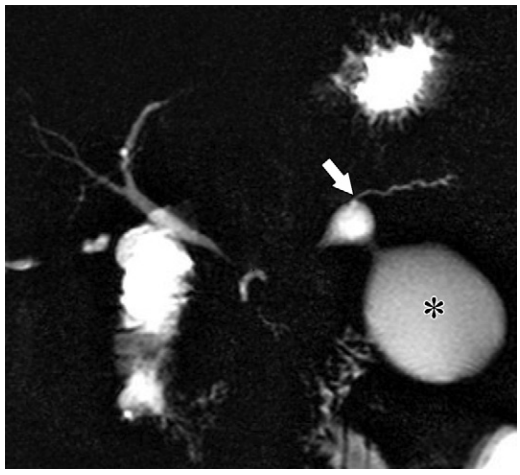


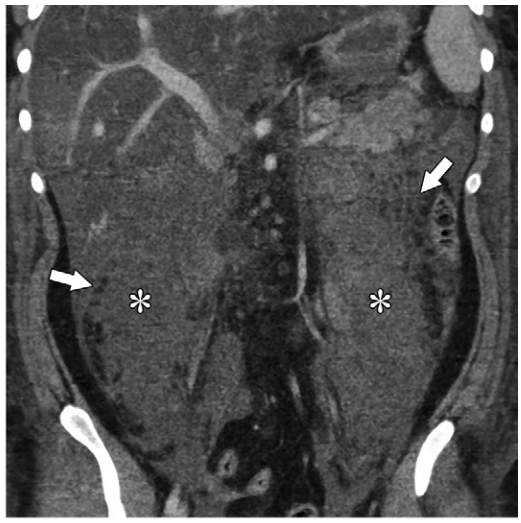
Figure 7. Pseudocyst in a 55-year-old woman with disconnected duct syndrome. Coronal two-dimensional MR cholangiopancreatographic image shows a large bilobed peripancreatic and intrapancreatic pseudocyst (*) that communicates with the pancreatic tail duct (arrow), which is mildly dilated. Note the disconnection of the tail duct from the ampulla owing to segmental pancreatic body necrosis.

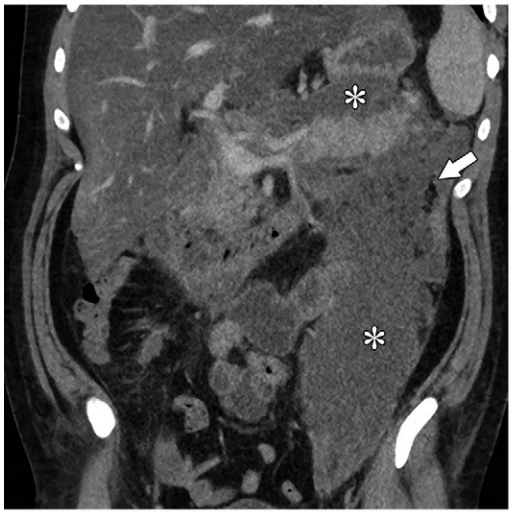
2 weeks better shows the internal heterogeneity of an ANC (2).

If an APFC has not resolved after 4 weeks, it becomes more organized and develops a capsule that manifests as an enhancing wall at contrast-enhanced CT. At this point, the collection is referred to as a pseudocyst (Fig 5), and, since there is no necrosis, it should contain only fluid with no nonliquefied components. If there is even a small area of fat or soft-tissue attenuation in an otherwise fluid-attenuation collection, the diagnosis is not pseudocyst but WON. At MR imaging, pseudocysts are uniformly hyperintense on T2-weighted images, with no solid components or debris in the fluid (Fig 6). Pseudocysts may have a connection to the pancreatic ductal system, which is best seen at MR cholangiopancreatography owing to superior contrast resolution (Fig 7) (2). These ductal connections can seal off, often leading to cyst resolution. Pseudocysts develop in fewer than 10% of cases of IEP (16). A pseudocyst is typically peripancreatic in location, although it can, on rare occasions, be intrapancreatic in cases of prior necrosectomy with a persistent pancreatic duct leak into the necrosectomy bed. In such cases, a pseudocyst forms because of disconnected duct syndrome, whereby a viable pancreatic tail remains after necrosectomy or pancreatic body necrosis, with the pseudocyst forming as a result pancreatic juice leakage from the disconnected duct (Fig 7) (2,17).

ANC and WON

ANCs are present within the first 4 weeks of symptom onset and are poorly organized necrotic collections that occur only in necrotizing pancreatitis. ANCs are often found in the lesser sac and pararenal spaces and may extend into the pancreas within areas of parenchymal necrosis. They are often multiple, with a loculated appearance, and may extend inferiorly as far as the pelvic sidewalls. ANCs typically demonstrate a variable amount of fluid and can be distinguished from APFCs by the presence of nonliquefied debris, such as solid-appearing components or fat globules within the fluid (Figs 3, 4, 8). When intravenous contrast material is contraindicated (eg, in a patient with acute renal failure), the presence of fat attenuation within a collection at nonenhanced CT is helpful for identifying necrosis and diagnosing ANCs (Fig 9). In the early phase of pancreatitis, differentiating between an APFC and an ANC can be





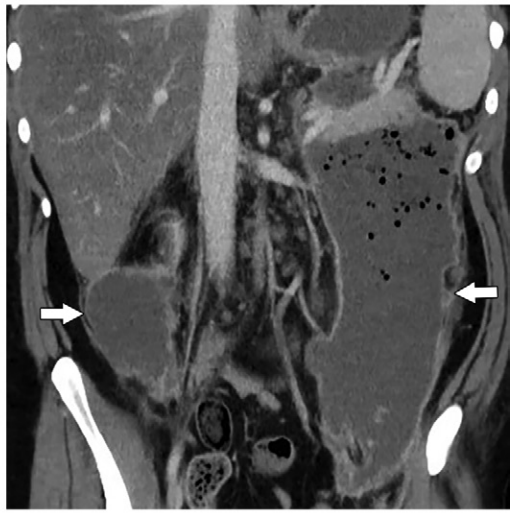


Figure 8. Infected WON on contrast-enhanced coronal CT scans in a 25-year-old man with idiopathic pancreatitis. **(a)** On day 7 after symptom onset, extensive bilateral ANCs are visible in the pararenal spaces. The ANCs extend to the pelvis (*), demonstrate heterogeneous fluid attenuation, and contain nonliquefied debris, including fat globules (arrows). **(b)** On day 15, the collection (*) has a more organized appearance, and it is easier to identify necrotic fat in the collection (arrow). **(c)** On day 42 image obtained after readmission for sepsis, organized, heterogeneous, gas-containing collections with thick enhancing walls are present in the pararenal space (arrows), findings that are consistent with infected WON.

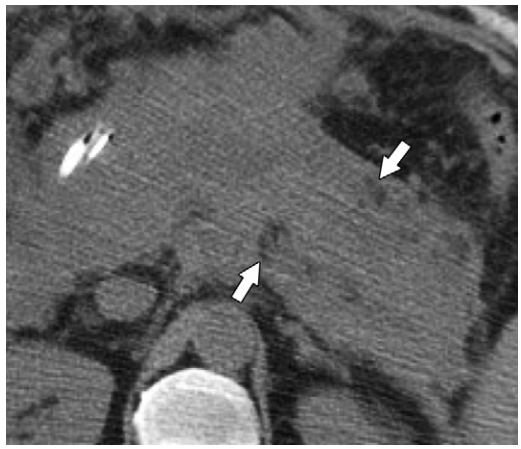


Figure 9. ANC in a 41-year-old woman with acute pancreatitis. Nonenhanced CT image obtained 9 days after symptom onset shows poor contrast resolution between pancreas and peripancreatic collections, such that necrosis in the pancreas is uncertain. However, the presence of small fat globules (arrows) in the peripancreatic collections and the overall heterogeneity are consistent with necrotizing pancreatitis with an ANC.

difficult, and the diagnosis of necrosis may be uncertain. Imaging in the 2nd week is usually helpful for distinguishing an APFC from an ANC. Any peripancreatic collection associated with known pancreatic parenchymal necrosis should be termed an ANC, even if it is homogeneous and contains no nonliquefied debris (2).

After 4 weeks, an ANC typically develops a thick mature wall, at which point the collection is referred to as WON. Like pseudocysts, a WON contains fluid and has a thick enhancing wall. Unlike pseudocysts, however, WON contains necrotic fat and/or pancreatic tissue, which are well demonstrated at both contrast-enhanced CT and MR imaging as nonliquefied debris within the fluid (Figs 3c, 10). WON may be confined to the pancreatic parenchyma but more commonly occurs in the peripancreatic space and can often

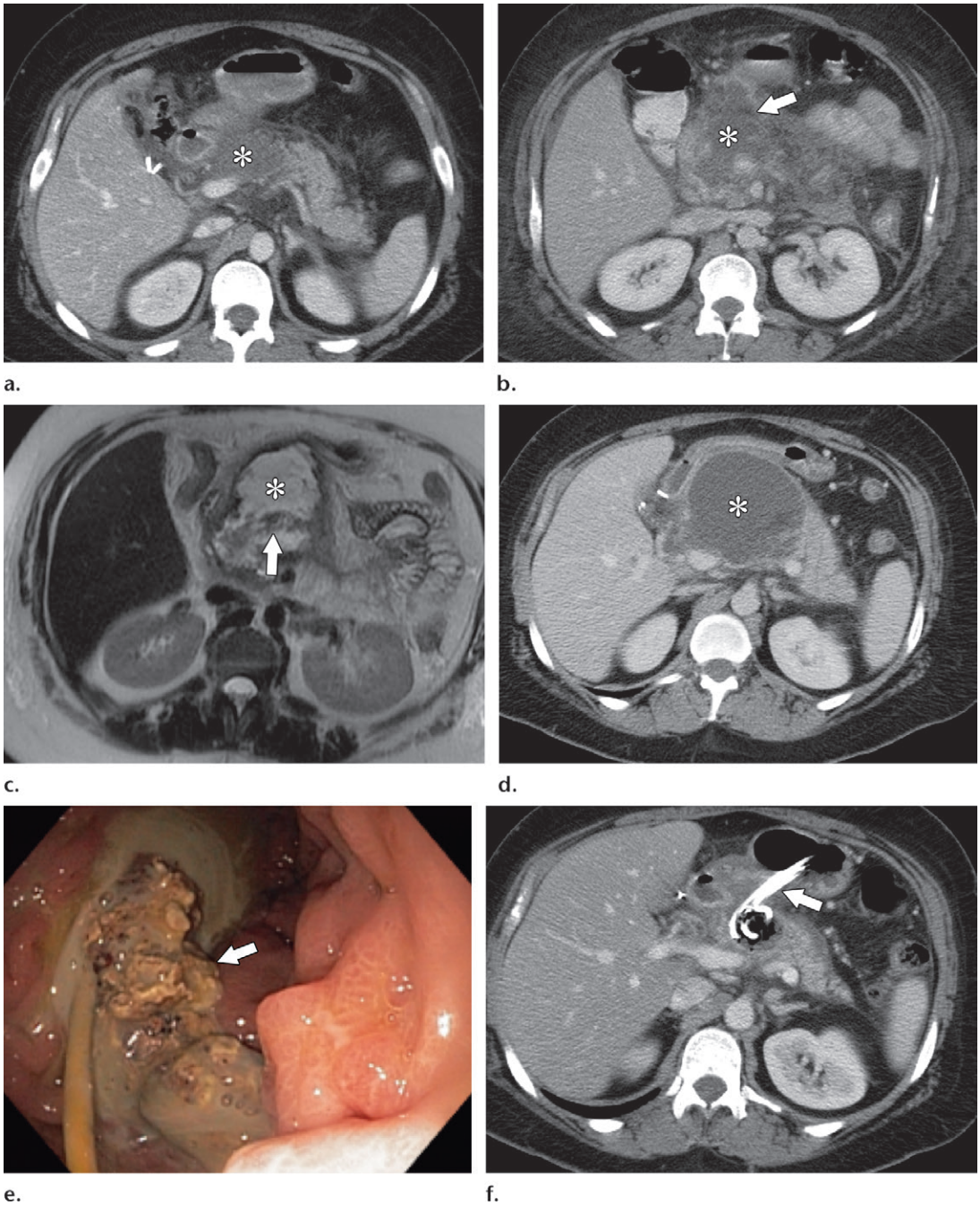
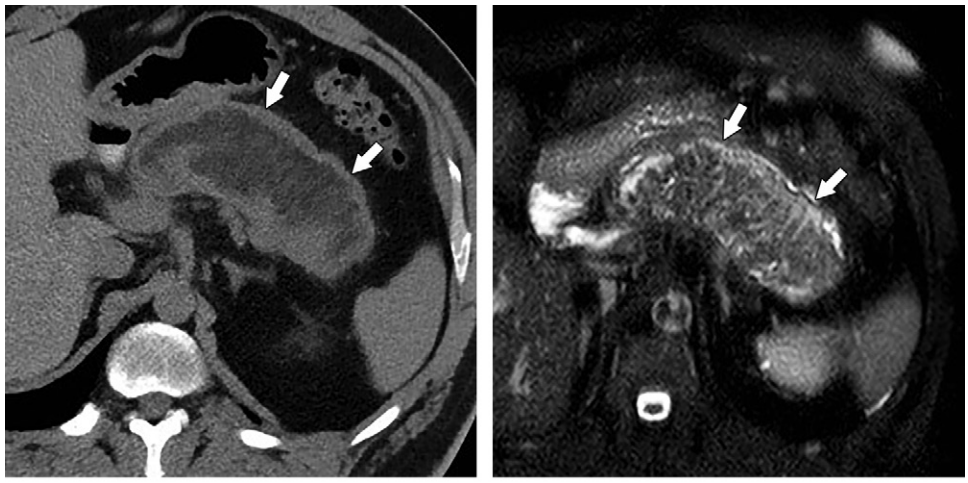


Figure 10. Evolution of necrotizing pancreatitis during 2 months in a 48-year-old woman. **(a)** Week 1: Axial contrast-enhanced CT image shows a necrotic pancreatic neck (*). **(b)** Week 2: Axial contrast-enhanced CT image shows a new heterogeneous necrotic peripancreatic collection (arrow) that is inseparable from the pancreatic necrotic collection (*); both findings are consistent with an ANC. **(c)** Week 3: Axial nonenhanced T2-weighted MR image better shows the contents of the ANC (*), including hyperintense fluid and nonliquefied debris, including necrotic pancreatic neck and body (arrow). Note the developing partial wall. **(d)** Week 5: Axial contrast-enhanced CT image shows maturation of the wall and a more round appearance of what is now referred to as WON (*). **(e)** Week 6: Endoscopic image from the gastric body, obtained during cystogastrostomy and debridement, shows necrotic debris (arrow) from the WON. **(f)** Week 7: Postprocedure axial contrast-enhanced CT image shows successful decompression of the WON. Multiple double-pigtail stents traversing the cystogastrostomy (arrows) can be seen.

occur in both locations, with a coalescent collection extending from the lesser sac into a portion of parenchyma (Fig 10) (2). There is evidence that MR imaging outperforms CT, with higher interreader agreement, in the assessment of the ratio of fluid to necrotic debris in collections older than 4 weeks. Therefore, MR imaging is a valuable alternative to contrast-enhanced CT for planning interventions because it allows determination of the amount of necrotic debris that must be removed by means of more aggressive interventions (Fig 11) (18).



a. b.

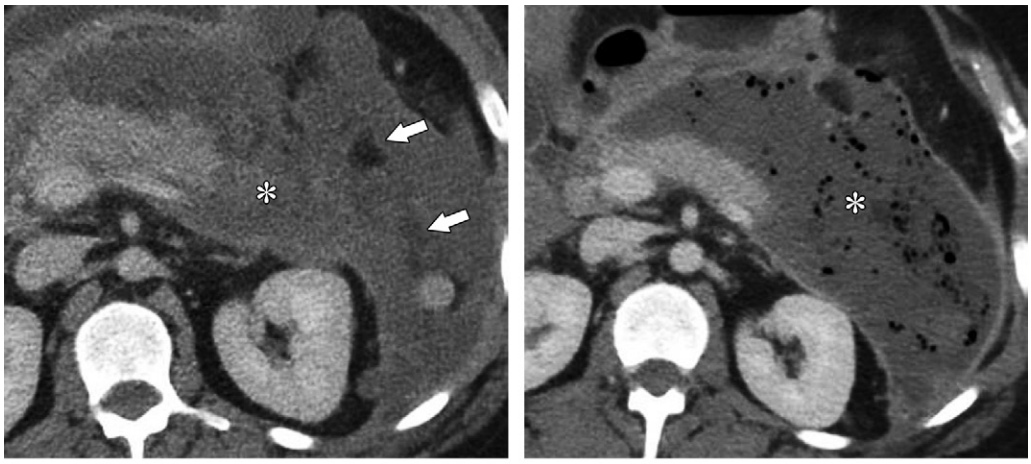
Figure 11. Necrotizing pancreatitis in a 47-year-old man in week 16 of persistent symptoms. **(a)** Axial nonenhanced CT image shows pancreatic WON involving nearly the entire pancreas (arrows); at least half of its volume appears to contain fluid. **(b)** Subsequent axial fat-saturated T2-weighted MR image shows the WON (arrows) containing mostly nonliquefied debris and pancreatic necrosis, with little fluid present. Such a collection would respond poorly to percutaneous or endoscopic drainage and would require more aggressive therapy.

Infection and Local Complications

Any collection can be sterile or infected, although infection occurs far more frequently in necrotic collections (2). Clinically, infection is suspected in a previously stable patient who experiences decompensation with signs of infection (19). The only imaging finding of an infected collection is the presence of gas within the collection. Wall enhancement is not a reliable indicator of infection, since it is invariably present in mature collections (pseudocyst and WON). An infected pseudocyst still lacks solid components that, if present, should instead lead to the diagnosis of infected WON. The gas often appears as multiple small bubbles scattered throughout the collection owing to the complex nature of necrotic collections (Figs 8, 12) (2). Infected collections can also manifest with gas bubbles due to a pancreatic-enteric fistula, which can occasionally be seen when necrotic collections erode through the bowel wall, most commonly in the colon and duodenum (Fig 13) (20). In one series, an enteric fistula occurred in 4% of patients hospitalized for acute pancreatitis (21). Gas within the pancreatic duct can also mimic gas within a pancreatic collection but generally has an identifiable linear distribution and typically occurs in the clinical context of recent endoscopic pancreatography or pancreatic duct stent placement (Fig 14).

The use of imaging-guided fluid collection aspiration or necrotic tissue fine-needle aspiration to help diagnose infection prior to invasive therapeutic necrosectomy has both advantages and disadvantages, and the topic is still widely debated. At some institutions, fine-needle aspiration has fallen out of favor in recent years, partly due to a shift in preference toward early conservative management with percutaneous drainage, which may delay or even obviate surgical intervention. If percutaneous drainage is performed, culture of the fluid can be performed at the same time (19,22). Another argument against fine-needle aspiration is that one must consider the substantial false-negative (sampling too early) and false-positive (contamination) results of the procedure (25% and 15%, respectively). At some institutions, fine-needle aspiration is thought to be helpful when clinical signs and imaging findings are confusing or complicated and the benefits of diagnosis of infection outweigh the risk of iatrogenic introduction of infection (22).

In addition to infection, vascular complications are common, occurring in a quarter of patients with acute pancreatitis, and can cause substantial morbidity and mortality. Two separate pathophysiologic processes lead to vascular complications. First, inflammatory reactions can lead to splenic vein thrombosis, the most common vascular complication (23). Second, pancreatic enzymes can cause vessel erosion and lead to either spontaneous arterial hemorrhage or pseudoaneurysm of (in order of decreasing frequency) the splenic, gastroduodenal, and pancreaticoduodenal arteries (24). A detailed discussion of these complications is beyond the scope of this article, as their evaluation is not specified under the revised Atlanta classification. We direct the reader to other excellent publications that describe these complications (25).



a. b.

Figure 12. Necrotizing pancreatitis in a 37-year-old woman. **(a)** Axial contrast-enhanced CT image obtained in week 3 shows the pancreatic tail (*) and a peripancreatic ANC containing nonliquefied debris with foci of fat attenuation (arrows). **(b)** Axial contrast-enhanced CT image obtained in week 6 because the patient experienced decompensation and was readmitted shows organization of the collection (*) with multiple new foci of gas, findings that are consistent with infected WON.

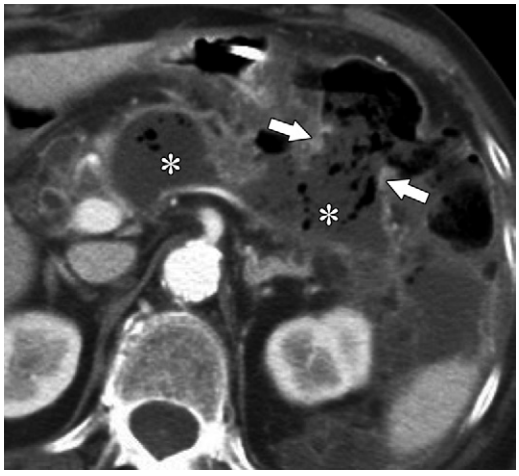


Figure 13. Necrotizing pancreatitis in a 74-year-old woman. Axial contrast-enhanced CT image obtained at 5 weeks shows peripancreatic WON with multiple foci of gas (*). A large fistula is seen to the distal transverse colon and contains gas and fluid (arrows), a finding that explains the development of gas in the WON.

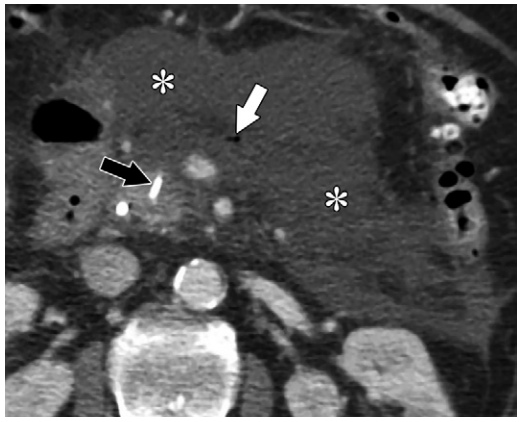


Figure 14. Necrotizing pancreatitis at week 3 in a 59-year-old man. Axial contrast-enhanced CT image obtained 3 weeks after disease onset shows necrosis of pancreatic neck, body, and tail (*), as well as peripancreatic necrosis. A small focus of gas (white arrow) in the pancreatic duct in the necrotic pancreatic body is secondary to recent stent placement in the duct (black arrow) and thus does not represent an infected ANC.

Management Implications

Various interventions are available for managing local complications of acute pancreatitis, with a range of invasiveness and, therefore, morbidity; these include percutaneous drainage, endoscopic cystogastrostomy, endoscopic débridement (Fig 10), and surgical necrosectomy (19). Newer approaches have focused on a combined "step-up" approach in which percutaneous drainage catheters are placed in necrotic collections, followed by minimally invasive débridements along the catheter tract if the patient fails to improve within 72 hours (26). Although these minimally invasive techniques have been replacing open surgical débridement, the previous standard of care, more recent studies in which open necrosectomy has been reanalyzed have found low mortality rates and results comparable to those achieved with minimally invasive techniques (27). While exact treatment regimens vary among institutions, collections generally do not warrant intervention unless there are persistent symptoms, inability to maintain nutrition, or signs of infection (28). By implementing the revised Atlanta classification, the radiologist is able to help the care team prescribe the appropriate therapy according to the type of collection. For instance, in the case of a lesser sac pseudocyst necessitating drainage, the gastroenterologist may create a cystogastrostomy with the use of endoscopic US, a procedure with

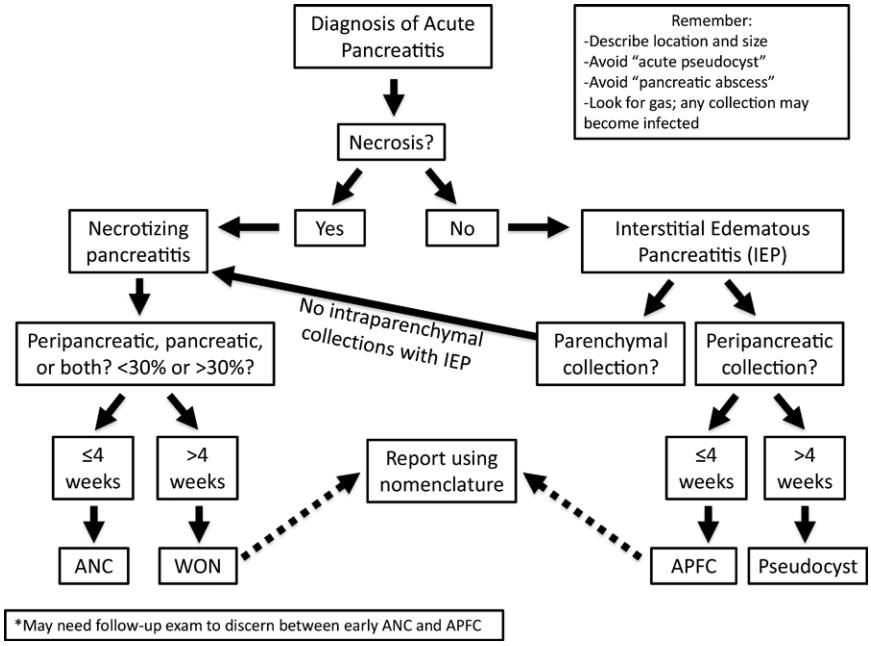


Figure 15. Decision tree for reporting according to the revised Atlanta classification.

a high success rate (29). However, if the collection represents WON, adequate drainage may not be achieved with this approach owing to the nonliquefied components and, therefore, necessitating more aggressive débridement through the endoscope as a first-line therapy (Fig 11).

Suggestions for Reporting

When reviewing a patient's history, it is important to note the time elapsed since the 1st day of abdominal pain, as this defines the time course used to stratify local complications. A statement about the presence or absence of necrosis should be made, and the location (peripancreatic, pancreatic, or both) and amount of necrotic gland (<30% or >30%) should be documented. Local complications should be described in terms of location (lesser sac, anterior pararenal space, transverse mesocolon, etc), size, appearance, and presence or absence of a mature wall. Specifically, the contents of local complications should be described either as homogeneous with fluid attenuation (APFC or pseudocyst) or as having nonliquefied necrotic components (ANC or WON). Finally, the collection should be specifically named according to the revised Atlanta classification lexicon, with the subtype of pancreatitis and the number of weeks since the onset of abdominal pain (<4 or ≥4 weeks) taken into account (Fig 15). We suggest that the findings be succinctly summarized in the impression section of the report (eg, IEP or necrotizing pancreatitis with type and location of collection) and a statement that the revised Atlanta classification was used so that multidisciplinary team members recognize the expertise and the standardization of terminology.

Conclusion

Incorporation of the revised Atlanta classification system into everyday practice updates and standardizes terminology, which facilitates accurate documentation of the range of imaging findings in acute pancreatitis. It is important to remember that pancreatitis-related collections are not always fluid filled, and evaluation for nonliquefied components is essential for differentiating collections that contain only fluid (APFCs and pseudocysts) from those that contain necrotic nonliquefied debris (ANCs and WON). In general, imaging findings combined with the time course of the disease allow clear differentiation between the collections and enable stratification among different treatment plans, facilitating the radiologist's seamless integration into a multidisciplinary team of gastroenterologists, intensivists, interventionalists, and surgeons.

References

1. Bradley EL 3rd. A clinically based classification system for acute pancreatitis: summary of the International Symposium on Acute Pancreatitis, Atlanta, Ga, September 11–13, 1992. Arch Surg 1993;128(5):586–590.
2. 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(1):102–111.
3. The Pancreas Club, Inc. Revision of the Atlanta classification of acute pancreatitis. Available at: http://pancreasclub.com/wp-content/uploads/2011/11/AtlantaClassification.pdf. Published April 9, 2008. Accessed March 28, 2015.
4. Thoeni RF. The revised Atlanta classification of acute pancreatitis: its importance for the radiologist and its effect on treatment. Radiology 2012;262(3):751–764.
5. Bollen TL. Imaging of acute pancreatitis: update of the revised Atlanta classification. Radiol Clin North Am 2012;50(3):429–445.
6. Sheu Y, Furlan A, Almusa O, Papachristou G, Bae KT. The revised Atlanta classification for acute pancreatitis: a CT imaging guide for radiologists. Emerg Radiol 2012;19(3):237–243.

7. Bollen TL, Besselink MG, van Santvoort HC, Gooszen HG, van Leeuwen MS. Toward an update of the Atlanta classification on acute pancreatitis: review of new and abandoned terms. Pancreas 2007;35(2):107–113.
8. Dunnick NR, Langlotz CP. The radiology report of the future: a summary of the 2007 Intersociety Conference. J Am Coll Radiol 2008;5(5):626–629.
9. Ellenbogen PH. BI-RADS: revised and replicated. J Am Coll Radiol 2014;11(1):2.
10. Yadav D, Lowenfels AB. The epidemiology of pancreatitis and pancreatic cancer. Gastroenterology 2013;144(6):1252–1261.
11. Petrov MS, Windsor JA. Classification of the severity of acute pancreatitis: how many categories make sense? Am J Gastroenterol 2010;105(1):74–76.
12. Baker ME, Nelson RC, Rosen MP, et al. ACR Appropriateness Criteria acute pancreatitis. Ultrasound Q 2014;30(4):267–273.
13. Singh VK, Bollen TL, Wu BU, et al. An assessment of the severity of interstitial pancreatitis. Clin Gastroenterol Hepatol 2011;9(12):1098–1103.
14. Marshall JC, Cook DJ, Christou NV, Bernard GR, Sprung CL, Sibbald WJ. Multiple organ dysfunction score: a reliable descriptor of a complex clinical outcome. Crit Care Med 1995;23(10):1638–1652.
15. Spanier BW, Nio Y, van der Hulst RW, Tuynman HA, Dijkgraaf MG, Bruno MJ. Practice and yield of early CT scan in acute pancreatitis: a Dutch observational multicenter study. Pancreatology 2010;10(2-3):222–228.
16. Lenhart DK, Balthazar EJ. MDCT of acute mild (nonnecrotizing) pancreatitis: abdominal complications and fate of fluid collections. AJR Am J Roentgenol 2008;190(3):643–649.
17. 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(5):1389–1400.
18. Kamal A, Singh VK, Akshintala VS, et al. CT and MRI assessment of symptomatic organized pancreatic fluid collections and pancreatic duct disruption: an interreader variability study using the revised Atlanta classification 2012. Abdom Imaging 2015;40(6):1608–1616.
19. Freeman ML, Werner J, van Santvoort HC, et al. Interventions for necrotizing pancreatitis: summary of a multidisciplinary consensus conference. Pancreas 2012;41(8):1176–1194.
20. Tüney D, Altun E, Barlas A, Yegen C. Pancreatico-colonic fistula after acute necrotizing pancreatitis: diagnosis with spiral CT using rectal water soluble contrast media. JOP 2008;9(1):26–29.
21. Kochhar R, Jain K, Gupta V, et al. Fistulization in the GI tract in acute pancreatitis. Gastrointest Endosc 2012;75(2):436–440.
22. van Baal MC, Bollen TL, Bakker OJ, et al. The role of routine fine-needle aspiration in the diagnosis of infected necrotizing pancreatitis. Surgery 2014;155(3):442–448.
23. Türkvatan A, Erden A, Türkoğlu MA, Seçil M, Yüce G. Imaging of acute pancreatitis and its complications. II. Complications of acute pancreatitis. Diagn Interv Imaging 2015;96(2):161–169.
24. Memiş A, Parildar M. Interventional radiological treatment in complications of pancreatitis. Eur J Radiol 2002;43(3):219–228.
25. Shyu JY, Sainani NI, Sahni VA, et al. Necrotizing pancreatitis: diagnosis, imaging, and intervention. RadioGraphics 2014;34(5):1218–1239.
26. van Santvoort HC, Besselink MG, Bakker OJ, et al. A step-up approach or open necrosectomy for necrotizing pancreatitis. N Engl J Med 2010;362(16):1491–1502.
27. Madenci AL, Michailidou M, Chiou G, Thabet A, Fernández-del Castillo C, Fagenholz PJ. A contemporary series of patients undergoing open debridement for necrotizing pancreatitis. Am J Surg 2014;208(3):324–331.
28. Wu BU, Banks PA. Clinical management of patients with acute pancreatitis. Gastroenterology 2013;144(6):1272–1281.
29. Varadarajulu S, Christein JD, Tamhane A, Drelichman ER, Wilcox CM. Prospective randomized trial comparing EUS and EGD for transmural drainage of pancreatic pseudocysts (with videos). Gastrointest Endosc 2008;68(6):1102–1111.