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Acute Pancreatitis: Revised Atlanta Classification and the Role of Cross-Sectional Imaging

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OBJECTIVE. The 2012 revision of the Atlanta Classification emphasizes accurate characterization of collections that complicate acute pancreatitis: acute peripancreatic fluid collections, pseudocysts, acute necrotic collections, and walled-off necroses. As a result, the role of imaging in the management of acute pancreatitis has substantially increased.

CONCLUSION. This article reviews the imaging findings associated with acute pancreatitis and its complications on cross-sectional imaging and discusses the role of imaging in light of this revision.

Acute pancreatitis is the most common gastrointestinal reason for hospitalization in the United States, accounting for $2.6 billion in annual inpatient costs [1]. The disease exhibits a wide range of clinical manifestations, from transient local abdominal discomfort to irreversible systemic complications and, sometimes, death.

The management and study of acute pancreatitis was hindered by confusing and occasionally conflicting terminology [2]. In 1992, a consortium of acute pancreatitis experts developed the Atlanta Classification, the only widely accepted clinically based classification system used by clinicians and radiologists [2, 3]. As knowledge of acute pancreatitis and its sequelae progressed, the 1992 Atlanta Classification became outdated because some of its definitions proved confusing [4, 5]. In response, the Atlanta Classification underwent revision in 2012 [4] to incorporate the latest understanding of the disease.

The Revised Atlanta Classification

Definition and Diagnosis

Acute pancreatitis is an acute inflammatory process affecting the pancreas with variable involvement of local tissues and remote organs [2, 6]. The 2012 revision requires at least two of the following three criteria for diagnosis: abdominal pain consistent with the disease, a threefold increase in serum amylase or lipase levels, and imaging findings consistent with acute pancreatitis [4, 7]. As such, acute pancreatitis is mainly a clinical diagnosis, and imaging should be reserved for ambiguous cases, when the patient fails to improve clinically within the first 48–72 hours after admission, to evaluate suspected complications [7], or for elucidating the underlying cause.

Severity Classification

Disease severity is stratified by organ failure, local complications (fluid collections and necrosis), and systemic complications [4]. Mild disease lacks organ failure and local or systemic complications and is associated with rare mortality (1–2%) [8]. Moderately severe disease has transient organ failure (resolves within 48 hours) or local or systemic complications and is associated with a low mortality rate of approximately 2% [9]. Severe disease has persistent organ failure (persists beyond 48 hours), a mortality rate of approximately 20–30% [10, 11], and typically also local complications [4].

Interstitial Edematous Pancreatitis, Necrotizing Pancreatitis, and Pancreatic Fluid Collections

The distinction between two types of acute pancreatitis—interstitial edematous pancreatitis and necrotizing pancreatitis—was retained, but pancreatic and peripancreatic collections were redesignated according to the presence of necrosis and the time from the onset of symptoms. Collections that are seen less than 4 weeks after presentation are called acute peripancreatic fluid collections in interstitial edematous pancreatitis and acute necrotic collections in necrotizing pancreatitis.

Keywords: acute pancreatitis, Atlanta classification, imaging, necrosis, pseudocyst

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Collections that are seen more than 4 weeks after presentation are called pseudocysts and walled-off necrosis [4] (Fig. 1). Previously used terms such as "pancreatic abscess" and "pancreatic phlegmon" were abolished.

Phases of Disease

The revised Atlanta Classification divides the disease course into an early phase, usually lasting up to 1 week, followed by a late phase. During the early phase, the presence and duration of organ failure primarily determine disease severity, and imaging of local complications such as necrosis is not reliable, especially initially [4].

The late phase is characterized by persistent signs and symptoms with systemic or local complications, thus occurring only in moderately severe and severe disease. During this phase, assessment of local complications by imaging may be crucial for management. The late phase can extend for months, allowing persistent pancreatic collections to evolve, acquiring different characteristics and terminology; acute peripancreatic fluid collections and acute necrotic collections may evolve into pseudocysts and walled-off necroses, respectively.

Acute pancreatitis is a mercurial disease that is difficult to discuss with two phases. To better illustrate its progressively changing appearance, we will discuss the imaging findings in terms of less than 72 hours, 72 hours to 4 weeks, and more than 4 weeks since onset.

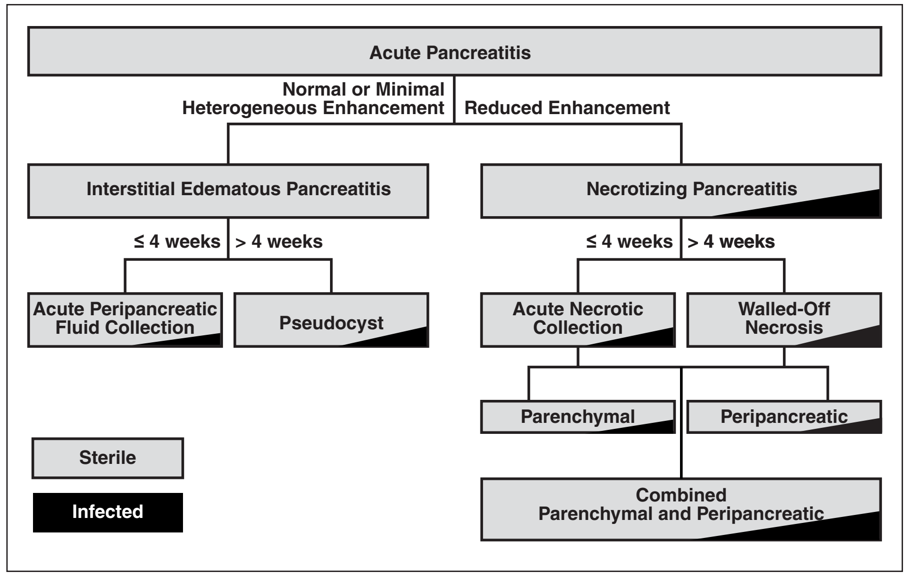


Fig. 1—Classification of acute pancreatitis and associated collections. Necrotizing pancreatitis and all collections arising from acute pancreatitis can undergo secondary infection.

Imaging Less Than 72 Hours After Onset

Establishing Diagnosis and Cause

Acute pancreatitis is readily confirmed by cross-sectional imaging. However, routine imaging is unwarranted, because it is usually a transient mild disease that is diagnosed clinically.

The most common cause of acute pancreatitis is cholelithiasis (35–40%), followed by alcohol (30%) [11, 12]. Therefore, transabdominal sonographic evaluation for cholelithiasis should be performed for all patients at admission [7, 13]. If cholelithiasis is present in the setting of elevated liver enzyme levels, cholecystectomy is indicated for prevention of recurrent biliary pancreatitis [14]. Intervention with ERCP is indicated for persistent choledocholithiasis or acute cholangitis [13]. If sonography is negative for cholelithiasis and another cause for recurrent pancreatitis cannot be found, MRCP or endoscopic ultrasound can further evaluate for other causes, such as pancreas divisum, malignancy, occult choledocholithiasis, or microlithiasis.

Limitations of Early Necrosis Detection

Pancreatic necrosis is a well-established risk factor for morbidity and mortality [15, 16]. Despite this, imaging for the detection of necrosis within the early phase is generally only indicated in deteriorating or critically ill patients and must be interpreted cautiously [7]. Early detection is difficult, because both necrotic and edematous parenchyma exhibit heterogeneous enhancement on contrast-enhanced CT (CECT). This heterogeneity is caused by intrapancreatic edema and ischemia [17, 18]. Over time, the intrapancreatic fluid becomes reabsorbed, and nonviable necrotic tissues liquefy [18, 19]. Frank necrosis may take 24–48 hours to develop [7, 20, 21], and the sensitivity of CECT for detection of pancreatic necrosis within 72 hours of disease onset is approximately 60–70% [22, 23]. Ideally, imaging should be performed at least 72 hours after symptom onset [6].

Severity Assessment

A prognostic method to identify patients likely to develop severe acute pancreatitis would allow clinicians to triage into an intensive care setting and initiate outcome-improving measures, such as aggressive fluid resuscitation [24, 25]. The CT severity index (CTSI) and subsequent modified CTSI are radiologic grading systems developed to help predict disease severity [20, 26]. Both the CTSI and modified CTSI assign points on the basis of the presence and extent of pancreatic inflammation, parenchymal necrosis, and extrapancreatic complications (modified CTSI only) observed on CT, up to a maximum of 10 points. The score correlates with mortality and indexes of patient morbidity, including occurrence of pancreatic infection, length of hospital stay, and need for invasive intervention [20, 26]. MRI can be comparably used for severity assessment [21, 27]. A major drawback of imaging for severity assessment is its reliance on necrosis characterization, which, as discussed previously, is difficult within 72 hours of disease onset. Therefore, imaging solely for severity assessment at admission is not recommended.

There are several clinically based scoring systems for predicting the severity of acute pancreatitis. The Ranson score, Acute Physiology and Chronic Health Examination II, and Bedside Index for Severity in Acute Pancreatitis are three of the most prevalent. However, these clinical scoring systems provide limited additional information, because they typically require 24–48 hours of clinical data to become accurate, and severe disease is often apparent regardless of score [7]. Clinically based systems for severity assessment are of limited efficacy, similar to imaging-based systems [28–30].

Imaging 72 Hours to 4 Weeks After Onset

Interstitial Edematous Pancreatitis

Interstitial edematous pancreatitis is a diffuse inflammatory process involving pancreatic and peripancreatic tissues and causing interstitial edema and enlargement. It is the milder and more common form of acute pancreatitis, seen in 85% of patients [31], and typically resolves within a week [32].

In mild interstitial edematous pancreatitis, the edematous pancreas can appear enlarged and hypodense on CT, typically diffusely, but occasionally focally. Peripancreatic inflammation may be manifest as an irregular pancreatic contour with peripancreatic fat stranding and fluid and typically involves the anterior pararenal space. After contrast agent administration, on both CT and MRI, the viable edematous parenchyma generally enhances homogeneously, albeit less strongly than normal, but the edema may cause slight heterogeneity.

MRI is more sensitive for pancreatic edema than CECT [27, 33]. On T2-weighted images, the pancreas is enlarged and hyperintense (Fig. 2). On T1-weighted images using fat suppression (a sequence that is not sensitive for acute edema) [34], the parenchyma may appear normal (in mild cases), hypointense, or heterogeneous [27, 33, 35]. Peripancreatic fat stranding is best visualized with T1 gradient-echo and T2 fat-suppressed sequences [34]. DWI can be helpful, because greater diffusion restriction is seen in patients with acute pancreatitis than in healthy control subjects [36]. There is no recognizable tissue necrosis in interstitial edematous pancreatitis. In more severe interstitial edematous pancreatitis, pancreatic fluid may leak out of the edematous parenchyma and form peripancreatic fluid collections.

Acute Peripancreatic Fluid Collection

Acute peripancreatic fluid collections are nonencapsulated collections that typically arise less than 4 weeks after the onset of acute pancreatitis. They are peripancreatic, rarely become infected, and generally resolve spontaneously [4]. Five percent to 15% of acute peripancreatic fluid collections persist beyond 4 weeks and are likely to become pseudocysts [37].

Acute peripancreatic fluid collections have variable size and shape and may be numerous. They are predominantly adjacent to the pancreas, without extension within the pancreatic parenchyma, and commonly reside within the lesser sac or anterior pararenal space [38] (Fig. 3). They are not round, instead taking the contours of the peripancreatic fascial planes containing them. Uncommonly, the fluid may collect in more distant areas, including the pelvis, ligamentum venosum fissures, splenic hilum, and mediastinum [39]. Acute peripancreatic fluid collections exhibit a homogeneous fluid appearance—hypodense on CT and T1 hypointense and T2 hyperintense on MR images—without a well-defined capsule.

Necrotizing Pancreatitis

Necrotizing pancreatitis presents in three configurations: combined pancreatic and peripancreatic necrosis (75%), peripancreatic necrosis alone (20%), and pancreatic necrosis alone (< 5%) [6, 40]. Patients with peripancreatic necrosis alone have lower morbidity and mortality rates [40].

On imaging, necrotizing pancreatitis is similar to interstitial edematous pancreatitis, with additional findings reflecting necrosis. After the first week, areas of impaired perfusion and necrosis have matured, resulting in more confluent nonenhancing areas on imaging [28]. The capacity of CT to diagnose retroperitoneal fat necrosis is limited [17]; therefore, all peripancreatic regions with a heterogeneous appearance on CECT should be considered fat necrosis, unless proven otherwise [17].

On MRI, necrotic parenchyma or peripancreatic tissue is hypointense and nonenhancing on gadolinium-enhanced T1-weighted imaging. On T2-weighted images, necrotic tissue is typically hypointense, though it can be hyperintense if liquefied [41, 42]. Abnormal hyperintensity of the peripancreatic fat on T1-weighted fat-suppressed images, corresponding to hemorrhagic fat necrosis, is associated with extremely poor prognosis [43]. MRI can distinguish between necrotic pancreatic and peripancreatic tissues and adjacent fluid collections or hemorrhage, whereas differentiating between necrosis and adjacent fluid may be difficult with CT [44]. Persistent collections of fluid and necrotic material can develop in the setting of necrotizing pancreatitis and should not be called fluid collections, but rather acute necrotic collections or walled-off necroses, because they contain solid necrotic material.

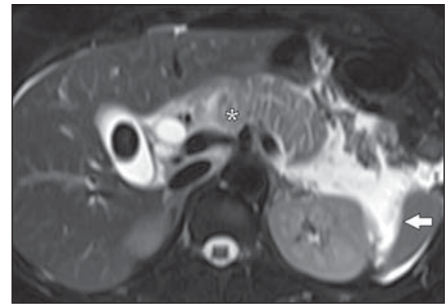
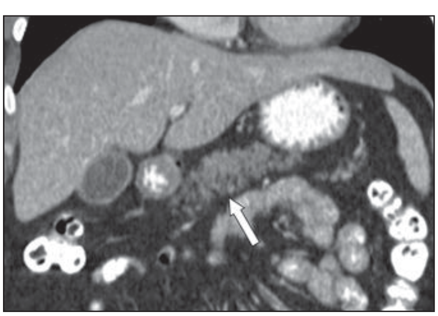


Fig. 3—30-year-old woman with interstitial edematous pancreatitis. Axial T2-weighted fat-suppressed image shows edematous pancreas (*asterisk*) and noncircumscribed hyperintense peripancreatic fluid collection (*arrow*), consistent with acute peripancreatic fluid collection, extending within left pararenal space.



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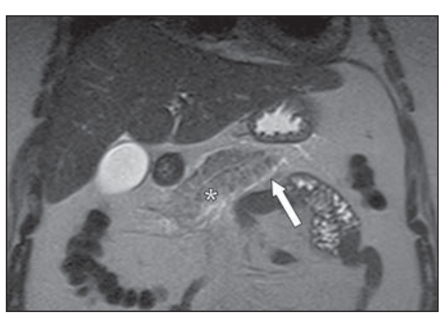


B

Fig. 2—42-year-old woman who presented with acute right upper quadrant pain.
A, Coronal contrast-enhanced CT image shows barely visible minimal free fluid (*arrow*) surrounding body of pancreas.
B, Coronal T2-weighted MR image obtained 37 hours later shows mild pancreatic enlargement and edema (*asterisk*) with minimal surrounding free fluid (*arrow*).

Acute Necrotic Collection

Acute necrotic collections are nonencapsulated collections of inflammatory fluid and necrotic pancreatic or peripancreatic tissue that typically arise within the first 4 weeks of necrotizing pancreatitis (Fig. 4). Acute necrotic collections are usually asymptomatic and undergo progressive liquefaction and decrease in size [45, 46]. Some acute necrotic collections will develop a fibrous capsule and become a walled-off necrosis.

Acute necrotic collections, like acute peripancreatic fluid collections, may be single

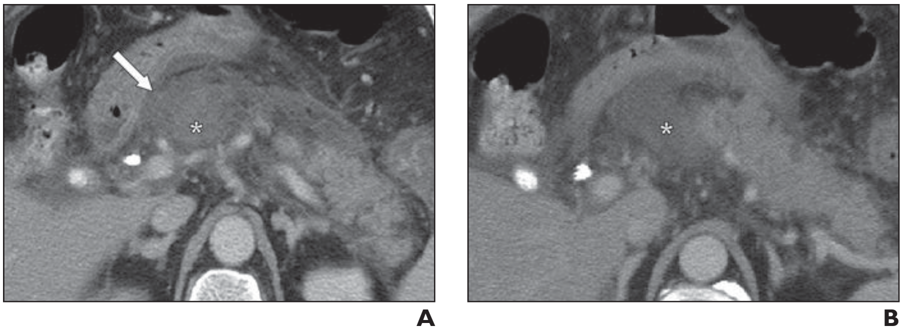


Fig. 4—45-year-old man with acute pancreatitis.
A and **B,** Axial contrast-enhanced CT images obtained early in the disease process show necrotic pancreatic body and head. There is ill-defined heterogeneous peripancreatic collection (*arrow*, **A**) merging with necrotic head of pancreas (*asterisks*, **A** and **B**), which does not have enhancing capsule, consistent with combined pancreatic and peripancreatic acute necrotic collection.

or multiple, variable in size and shape, and present in various anatomic locations. Unlike acute peripancreatic fluid collections, acute necrotic collections may reside within the pancreatic parenchyma and are frequently associated with necrosis of the main pancreatic duct [47, 48]. In fact, any nonencapsulated intrapancreatic collection found within the first 4 weeks after the onset of necrotizing pancreatitis should be considered an acute necrotic collection [6, 49]. MRCP may reveal pancreatic ductal disruption and communication with the acute necrotic collection.

The identification of solid necrotic material on imaging is crucial for differentiating acute necrotic collections from acute peripancreatic fluid collections. Acute necrotic collections appear heterogeneous because of their liquefied and nonliquefied components, seen as irregular hyperdensity among hypodense fluid on CT and as T2-hypointense material among T2-hyperintense fluid on MRI [50]. It is easier to identify the characteristic heterogeneity after the first week of disease [6]. Imaging within the first week may not allow sufficient time for solid necrotic material to liquefy, making differentiation of peripancreatic acute necrotic collections from acute peripancreatic fluid collections on CECT challenging, because both could appear as homogeneous nonenhancing hypodense abnormalities adjacent to the pancreas (which can have normal enhancement when necrosis is purely peripancreatic).

Imaging More Than 4 Weeks After Onset

Pseudocyst

Pseudocysts are encapsulated cystic lesions filled with amylase-rich fluid that complicate 10–20% of cases of acute pancreatitis. They typically evolve from acute peripancreatic fluid collections by forming a capsule, a process that usually requires at least 4 weeks [51]. Pseudocysts lack an epithelial lining and thus are not true cysts [52]. About 50% of pseudocysts remain asymptomatic, and there are no reliable indicators, such as size or duration, to predict which will become symptomatic and require treatment [37, 53]. Approximately 40% of pseudocysts will spontaneously resolve [54].

Pseudocysts are usually round-to-oval fluid collections with a well-defined capsule. Except for some dependent debris, pseudocysts lack internal solid components and appear predominantly homogeneous [55]. They can be single or multiple and are typically unilocular and peripancreatic, though they can be multiloculated [4]. On CT, pseudocysts exhibit fluid density (< 15 HU) within a well-defined capsule. The capsule is smooth and symmetric and varies in appearance from barely perceptible to uniformly thick and may enhance after contrast agent injection. MRI can help confirm the lack of solid material, with pseudocysts generally exhibiting T1 hypointensity and T2 hyperintensity (Fig. 5). Hemorrhage or proteinaceous fluid within the pseudocyst can cause T1 hyperintensity. Dependent debris on MRI is highly specific for pseudocysts [55].

Many pseudocyst-associated complications and symptoms, such as gastric obstruction leading to early satiety and nausea, are secondary to local mass effect. Pseudocysts can also erode into adjacent vessels and cause pseudoaneurysms (discussed below). Hemorrhage within a pseudocyst shortly after an acute attack of pancreatitis is generally not due to pseudoaneurysm rupture but is rather the result of bleeding from intramural capillaries within the pseudocyst or retroperitoneum [56].

Walled-Off Necrosis

Walled-off necroses are well-circumscribed encapsulated cavities containing necrotic pancreatic and peripancreatic tissue that complicate about 1–9% of cases of acute pancreatitis [57]. Walled-off necroses develop when necrotic tissues and acute necrotic collections mature and form a capsule. This process usually requires at least 4 weeks, similar to the process seen in pseudocyst

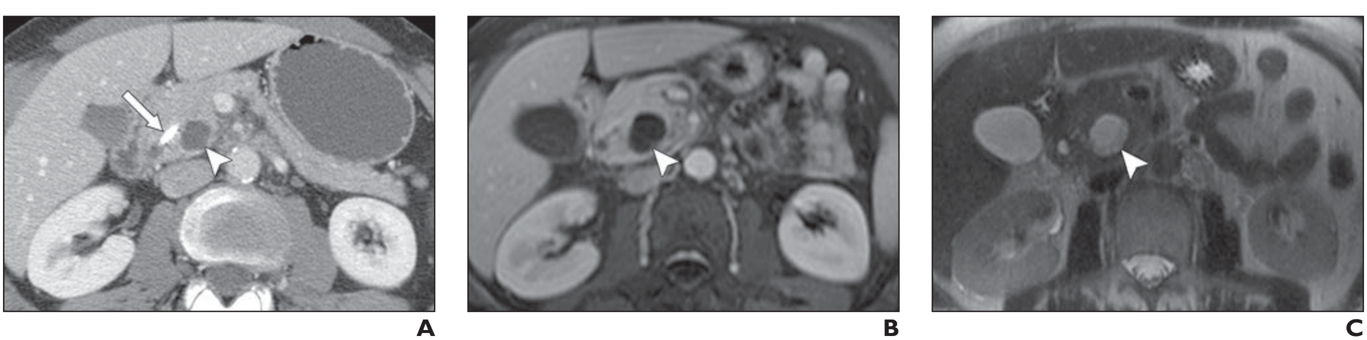


Fig. 5—52-year-old man who presented with pseudocyst 7 months after acute alcoholic interstitial edematous pancreatitis.
A, Axial contrast-enhanced CT shows small fluid collection in uncinate process (*arrowhead*). Pancreatic stent (*arrow*) was placed 8 days before CT. Mild retropancreatic fat stranding is visible, indicating ongoing inflammation.
B and **C,** Corresponding axial contrast-enhanced T1-weighted MR image (**B**) and T2-weighted MR image (**C**) show round nonenhancing T1-hypointense and T2-hyperintense collection (*arrowheads*) with minimal internal debris visible on T2-weighted image, with thin nonenhancing capsule.

formation. The outdated terms "organized pancreatic necrosis," "necroma," "pancreatic sequestration," "pseudocyst associated with necrosis," and "subacute pancreatic necrosis" are not to be used [4]. Patients with walled-off necrosis can be asymptomatic (50%) or present with abdominal pain, fever, nausea and vomiting, early satiety, or weight loss [58, 59]. Similar to pseudocysts, walled-off necroses can obstruct or fistulize adjacent structures, erode into vasculature, and become infected [60]. Radiologic follow-up for growth and complications is recommended, even for asymptomatic walled-off necroses that usually undergo liquefactive necrosis and decrease in diameter over time [45, 46].

Like pseudocysts, walled-off necroses are round, have a nonepithelialized capsule, and may contain septations. Unlike pseudocysts, walled-off necroses more commonly involve the pancreatic parenchyma. Any encapsulated fluid-containing collection within the pancreas, with resultant deformation or discontinuation of the parenchyma, found more than 4 weeks after the onset of acute necrotizing pancreatitis is very likely walled-off necrosis [6]. In fact, most walled-off necroses can be readily diagnosed on the basis of the aforementioned features. Approximately 90% of walled-off necroses are located in the body or tail, but they can also be distant from the pancreas and frequently extend into the paracolic gutters [4, 59, 61].

Walled-off necroses and pseudocysts may appear similar on imaging [58]. The distinction of walled-off necrosis from pseudocyst is important, because the solid necrotic component within walled-off necroses may become infected if not removed [62, 63]. On CT, a walled-off necrosis is an encapsulated fluid-density collection within a region that was previously necrotic tissue [19, 64] (Fig. 6). The presence of pancreatic necrosis on imaging from earlier in the disease course may help distinguish between walled-off necrosis and pseudocyst. Specific findings on CT indicative of walled-off necrosis over pseudocyst are larger size, extension to the paracolic or retrocolic space, irregular border, fat attenuation debris, thick or multiple septations, and pancreatic parenchymal deformity or discontinuity [64]. Dilatation of the main pancreatic duct (> 4 mm) favors pseudocyst. The aforementioned findings have an accuracy rate of approximately 80% for differentiating walled-off necrosis from pseudocyst [64]. The solid component can be identified on CT in only approximately 45%

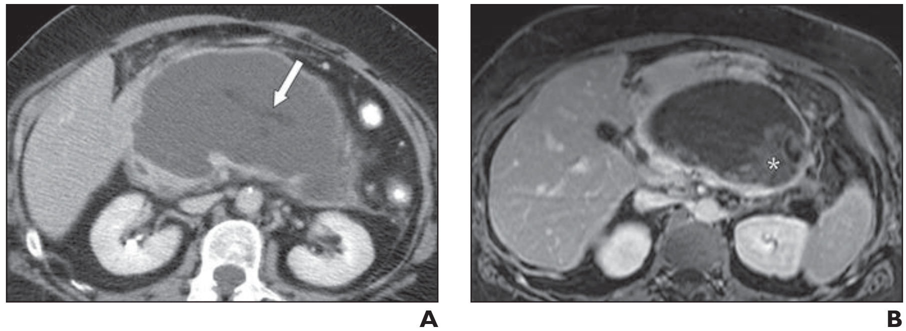


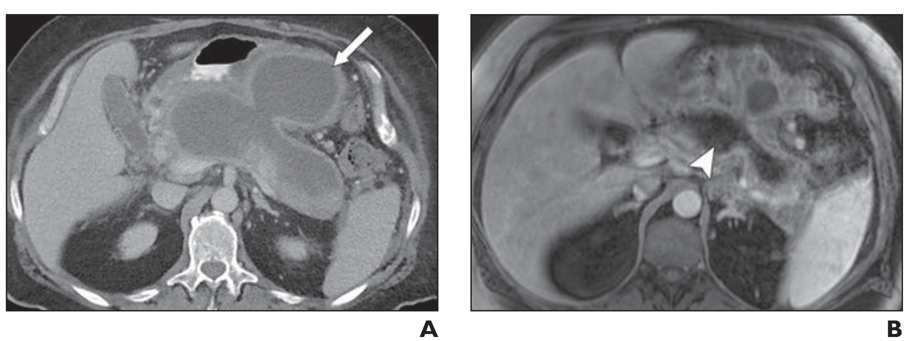
Fig. 6—63-year-old woman with necrotizing pancreatitis.
A, Axial contrast-enhanced CT shows large pancreatic and peripancreatic fluid collection, with residual peripancreatic fat (*arrow*) visible in collection as indication that this is walled-off necrosis rather than pseudocyst.
B, Axial contrast-enhanced MRI is able to show necrotic nonenhancing pancreatic parenchyma within collection (*asterisk*).

of walled-off necroses [64]. When visible, it is seen as a relatively hyperdense component within the fluid, creating a heterogeneous appearance similar to that of acute necrotic collections. MRI is better suited to visualize the necrotic debris, as with acute necrotic collections (Fig. 7). The main difference from acute necrotic collection is a visible capsule. A concomitant MRCP is valuable, because there is strong association (50–70%) between walled-off necrosis and disconnected pancreatic duct syndrome [60, 65].

Other Complications

Disconnected Pancreatic Duct Syndrome

Disconnected pancreatic duct syndrome is a discontinuity of the main pancreatic duct such that the viable tail of the pancreas is isolated and unable to drain into the head and duodenum. Disconnected pancreatic duct syndrome complicates about 30% of cases of acute necrotizing pancreatitis, with the probability directly related to the amount of necrosis [66, 67]. Disconnected pancreatic duct syndrome usually occurs at the pancreatic neck, a watershed region susceptible to perfusion abnormalities [68]. Disconnected pancreatic duct syndrome causes continual leakage of pancreatic juices into the retroperitoneum, resulting in pancreatic and peripancreatic necrosis and formation of fluid collections [66]. In these cases, either permanent drainage (endoscopic or surgical) of the collection or a distal pancreatectomy is required.



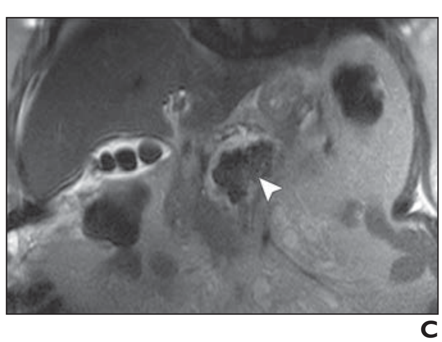


Fig. 7—65-year-old man with walled-off necrosis.
A, Axial contrast-enhanced CT shows encapsulated homogeneous fluid-attenuation collection (*arrow*) consistent with pseudocyst or walled-off necrosis.
B and **C,** Axial contrast-enhanced T1-weighted fat-suppressed (**B**) and coronal T2-weighted (**C**) images show internal heterogeneity (*arrowheads*) indicative of necrotic debris, confirming diagnosis of walled-off necrosis.

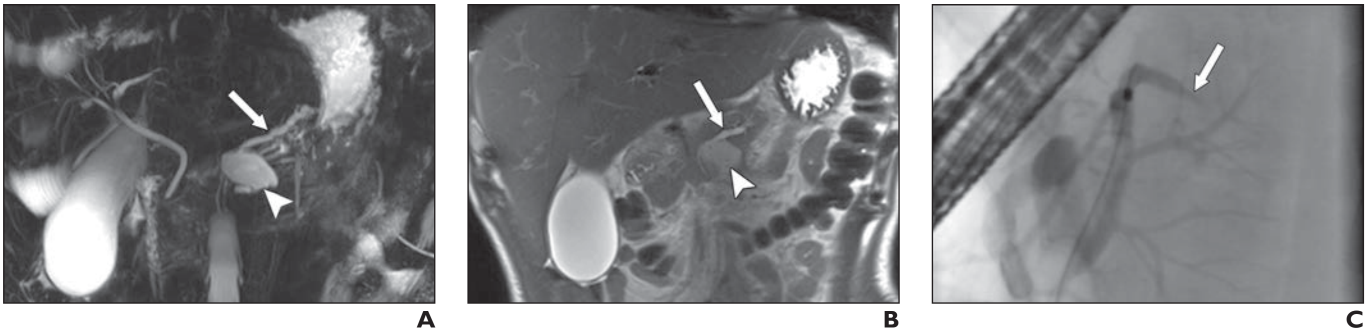


Fig. 8—26-year-old man who underwent transgastric drainage of pseudocyst located near pancreatic neck that recurred after stents migrated into stomach.
A and **B,** Three-dimensional navigator-triggered MRCP maximal intensity projection reconstruction (**A**) and coronal T2-weighted image (**B**) show connection between pseudocyst (*arrowheads*) and pancreatic duct in tail (*arrows*), which is dilated.
C, ERCP shows complete cutoff of pancreatic duct in pancreatic neck (*arrow*).

Contrast-enhanced imaging revealing necrosis within the pancreatic body or neck with intact perfusion of the tail suggests disconnected pancreatic duct syndrome. MRCP can be obtained for ductal evaluation, which can be further enhanced by secretin administration [69]. A discrete ductal cutoff within the upstream pancreatic tail and a fluid collection along the expected course of the pancreatic duct are indicative [66, 69, 70]. ERCP may be useful for confirmation, showing a complete early termination of the pancreatic duct (Fig. 8).

Secondary Infection

Secondary infection of necrotizing pancreatitis is associated with increased mortality and is usually diagnosed at least 2–3 weeks after disease onset [71]. Infected pancreatic necrosis is associated with poor outcomes (mortality rate of 32%) and frequently requires intervention [72], whereas sterile pancreatic necrosis has a mortality rate of about 12% [31]. The risk of infection is increased in prolonged disease with persistent bacteremia [71, 73]. Prophylactic antibiotics are usually ineffective, likely because of poor penetration into necrotic tissue [7, 74].

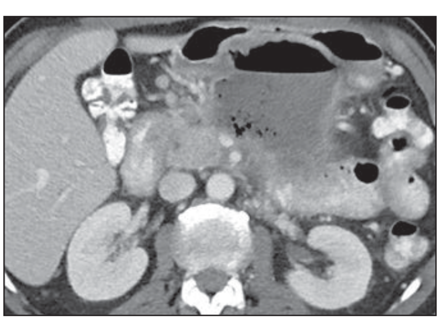


Fig. 9—62-year-old man with walled-off necrosis. Axial contrast-enhanced CT shows air within walled-off necrosis with thickened irregular capsule, concerning for secondary infection versus enteric fistula. Subsequent drainage yielded purulent fluid with positive bacterial culture, confirming infection. There was no fistula.

Any collection complicating acute pancreatitis can become secondarily infected. The diagnosis of infection can be difficult on imaging. Gas within a collection is the most sensitive imaging finding for infection (Fig. 9) but is visible in only 12–18% of cases [75]. Although MRI can reveal large amounts of gas, CT is more sensitive for small quantities [41]. However, gas is not pathognomonic and can alternatively indicate fistulization into an adjacent hollow viscus [76].

The utility of DWI is under investigation, and it has been suggested that central portions of infected acute peripancreatic fluid collections and acute necrotic collections exhibit more restricted diffusion compared with their sterile counterparts, although overlap may be seen [77, 78]. When infection is suspected, aspiration of fluid for culture is necessary [75].

Vascular Complications and Hemorrhage

Venous thrombosis is the most common vascular complication of pancreatitis, usually involving the splenic vein, but possibly also the portal and superior mesenteric veins [79]. Thrombosed vessels are nonenhancing tubular structures on imaging [34].

Pseudocysts and walled-off necroses can erode into adjacent vasculature, resulting in pseudoaneurysms [51, 60]. The splenic, gastroduodenal, and pancreaticoduodenal are the most commonly affected vessels [47, 51]. Pseudoaneurysm formation takes time and therefore does not occur early in the disease [56]. Pseudoaneurysms tend to gradually enlarge and may rupture [51]. They are associated with high mortality rates: 12.5% when detected and treated and more than 90% if untreated [80]. The hemorrhage is most often within the gastrointestinal tract but can be intraperitoneal or, rarely, even within the pancreatic ductal system. Pseudoaneurysms can be detected on both CT and MR angiography as a rounded structure arising from and showing enhancement similar to the donor artery [33, 81].

CT Versus MRI for Pancreatic Imaging

CT is the most widely used modality, because it has a short scan duration (important in acutely ill patients) and is widely available and because radiologists are experienced in its interpretation. Accurate assessment of the pancreatic parenchyma for necrosis on CT requires IV contrast agents, which are potentially nephrotoxic in patients with renal insufficiency and contraindicated with severe renal dysfunction, thus limiting the diagnostic accuracy of CT in potentially renal-impaired patients. MRI is an alternative modality that does not use ionizing radiation, which is particularly important in pregnant women and young patients, for whom the cumulative radiation dose is of concern, but it also has limitations in severe renal dysfunction because of concerns of nephrogenic systemic fibrosis.

For pancreatic fluid and necrotic collections, both CT and MRI can be used for detection, but MRI may be required to differentiate between them (i.e., differentiate acute peripancreatic fluid collection from acute necrotic collection and pseudocyst from walled-off necrosis). The superior tissue contrast of MRI enables visualization of the solid necrotic tissue among the fluid within the collection that is diagnostic of acute necrotic collection and walled-off necrosis [6, 10, 41, 55]. The solid component may be difficult to detect on CT because of volume averaging [47]. In addition to MRI, endoscopic ultrasound and transabdominal ultrasound may also be used for detecting the solid material within acute necrotic collections and walled-off necrosis [10]. MRI may also be superior to CT for evaluation of pancreatic ductal integrity and communication with fluid collections, because disconnected pancreatic duct syndrome is more confidently excluded with MRI than CT [82].

Intervention for Complications of Acute Pancreatitis

Invasive intervention for complications associated with acute pancreatitis is indicated in the presence of symptoms or secondary infection in patients for whom conservative management has failed or who are not sufficiently stable for conservative management [7]. A large proportion of sterile asymptomatic fluid and necrotic collections will spontaneously resolve over time [4, 45, 54], and unnecessary instrumentation of these sterile collections should be avoided because of the risk of introducing infection. A subset of patients with infected pancreatic necrosis can be treated with conservative management using IV antibiotics [83]. When hemorrhage is seen in or adjacent to a collection planned for intervention, a pseudoaneurysm must be excluded because draining a collection complicated by pseudoaneurysm may lead to life-threatening hemorrhage.

Pseudocyst

Endoscopic transenteric (transgastric or transduodenal) drainage, generally with endoscopic ultrasound guidance, is the standard approach for draining pseudocysts [84]. The procedure is performed by identifying the pseudocyst adjacent to the lumen and endoscopically creating a fistula between the pseudocyst and lumen and subsequently placing transluminal stents. Percutaneous ultrasound- or CT-guided drainage is another viable option, but it requires an external drainage catheter [85] and is, therefore, less favored. The transpapillary approach, involving placement of an intraductal stent during ERCP, is the least traumatic approach but is effective only for smaller pseudocysts in the pancreatic head or neck that communicate with the main pancreatic duct [86, 87] or its radicals [88]. ERCP is the most sensitive method for diagnosing a pseudocyst-pancreatic ductal communication, but it may introduce infection [52]. Instead, MRCP is often used as a noninvasive alternative [89]. Pseudocyst aspiration, without continuous drainage (percutaneous, endoscopic, or surgical), is ineffective and should be avoided, except in small indeterminate cysts.

Necrotizing Pancreatitis

Because of the considerable morbidity (34–95%) and mortality (11–39%) of surgical necrosectomy in necrotizing pancreatitis, there is interest in conservative treatment [83, 90] and minimally invasive interventional techniques, such as image-guided percutaneous drainage [60, 90, 91], endoscopic transenteric drainage [60, 91], and endoscopic necrosectomy [92, 93]. When infected pancreatic necrosis is managed conservatively, or by minimally invasive procedures, careful monitoring is needed, because a lack of improvement or clinical deterioration warrants progression to more aggressive measures. Current guidelines support a step-up approach when intervention is necessary, starting with drainage and progressing in invasiveness to surgical necrosectomy [13]. A benefit of the step-up approach is delay of surgery, which has been shown to decrease mortality [94, 95]. As a result, current guidelines recommend delaying invasive intervention for necrotizing pancreatitis until at least 4 weeks after disease onset [13].

In contrast to pseudocyst management, drainage alone is generally ineffective in the management of walled-off necrosis; thus, these entities must be differentiated before intervention, generally via MRI or endoscopic ultrasound. Percutaneous or endoscopic management of necrotic collections often involves multiple catheters or drains with aggressive irrigation or direct endoscopic removal of necrotic tissue [60, 96]. Unsuccessful removal of necrotic debris can lead to infection [62, 63]. Collections that are larger and with a greater proportion of solid debris require more aggressive inter-

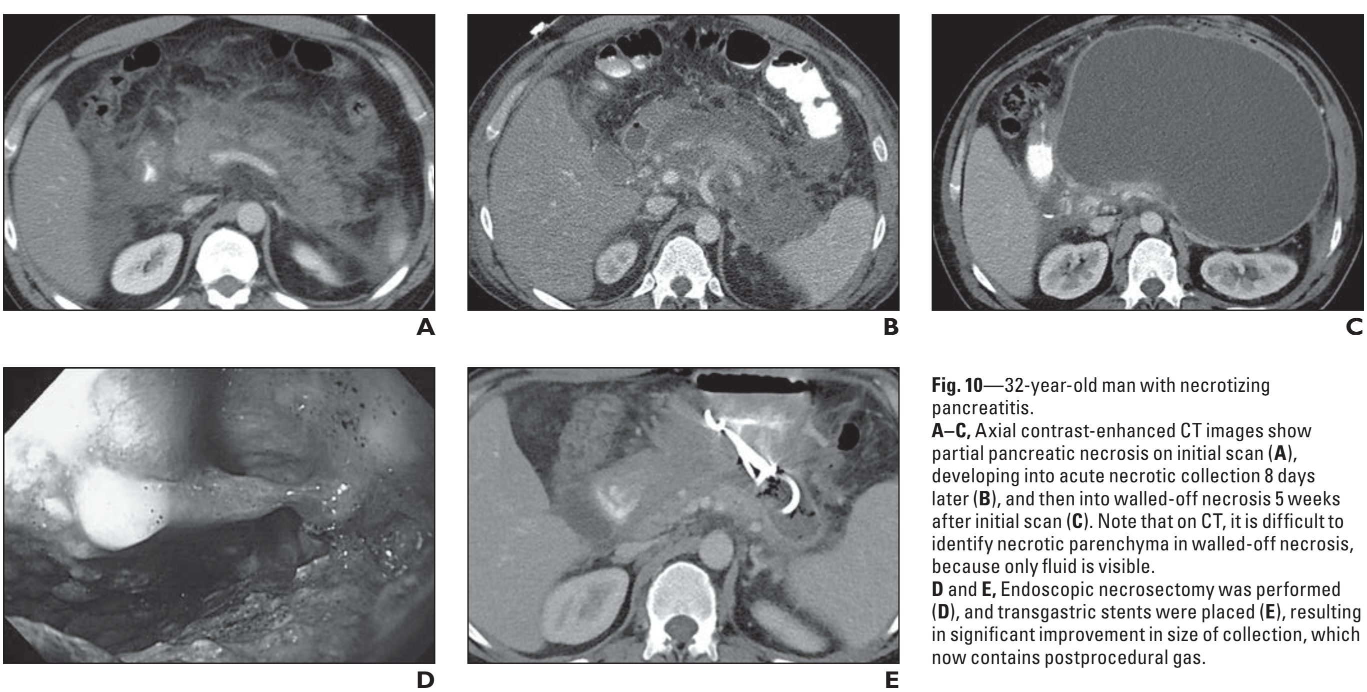


Fig. 10—32-year-old man with necrotizing pancreatitis.
A–C, Axial contrast-enhanced CT images show partial pancreatic necrosis on initial scan (**A**), developing into acute necrotic collection 8 days later (**B**), and then into walled-off necrosis 5 weeks after initial scan (**C**). Note that on CT, it is difficult to identify necrotic parenchyma in walled-off necrosis, because only fluid is visible.
D and **E,** Endoscopic necrosectomy was performed (**D**), and transgastric stents were placed (**E**), resulting in significant improvement in size of collection, which now contains postprocedural gas.

vention, such as endoscopic débridement or surgical necrosectomy, for adequate treatment [97] (Fig. 10).

Postintervention Considerations

A major pitfall of percutaneous and surgical procedures is the subsequent development of pancreaticocutaneous fistulas, either to the surgical incision or in the path of the drain. These are not rare, developing in 25–45% of patients who receive percutaneous or surgical intervention [98, 99], and may be associated with severe electrolyte disturbances and sepsis [100]. The presence of gas is a common imaging finding after invasive procedures. Therefore, finding gas after intervention is not as indicative of infection and should be interpreted with caution.

Conclusion

Acute pancreatitis is a complex and challenging disease. Both clinicians and radiologists should be aware of the current recommendations for appropriate imaging timing and modality. Although CT is often the first choice in acutely ill patients, MRI is better suited for diagnosing necrotic debris that may alter management. Using the correct terminology for reporting imaging findings of the disease and its complications is crucial for appropriate clinical decision making.

References

1. Peery AF, Dellon ES, Lund J, et al. Burden of gastrointestinal disease in the United States: 2012 update. *Gastroenterology* 2012; 143:1179–1187
2. Bradley EL 3rd. A clinically based classification system for acute pancreatitis: summary of the International Symposium on Acute Pancreatitis, Atlanta, GA, September 11 through 13, 1992. *Arch Surg* 1993; 128:586–590
3. Bollen TL, van Santvoort HC, Besselink MG, et al. The Atlanta Classification of acute pancreatitis revisited. *Br J Surg* 2008; 95:6–21
4. Banks PA, Bollen TL, Dervenis C, et al. Classification of acute pancreatitis: 2012—revision of the Atlanta classification and definitions by international consensus. *Gut* 2013; 62:102–111
5. Besselink MG, van Santvoort HC, Bollen TL, et al. Describing computed tomography findings in acute necrotizing pancreatitis with the Atlanta classification: an interobserver agreement study. *Pancreas* 2006; 33:331–335
6. Thoeni RF. The revised Atlanta classification of acute pancreatitis: its importance for the radiologist and its effect on treatment. *Radiology* 2012; 262:751–764
7. Tenner S, Baillie J, DeWitt J, Vege SS. American College of Gastroenterology guideline: management of acute pancreatitis. *Am J Gastroenterol* 2013; 108:1400–1415
8. Triester SL, Kowdley KV. Prognostic factors in acute pancreatitis. *J Clin Gastroenterol* 2002; 34:167–176
9. Vege SS, Gardner TB, Chari ST, et al. Low mortality and high morbidity in severe acute pancreatitis without organ failure: a case for revising the Atlanta classification to include "moderately severe acute pancreatitis." *Am J Gastroenterol* 2009; 104:710–715
10. Lerch MM. Classifying an unpredictable disease: the revised Atlanta classification of acute pancreatitis. *Gut* 2013; 62:2–3
11. Forsmark CE, Baillie J; AGA Institute Clinical Practice and Economics Committee; AGA Institute Governing Board. AGA Institute technical review on acute pancreatitis. *Gastroenterology* 2007; 132:2022–2044
12. Du BQ, Yang YM, Chen YH, Liu XB, Mai G. N-acetylcysteine improves pancreatic microcirculation and alleviates the severity of acute necrotizing pancreatitis. *Gut Liver* 2013; 7:357–362
13. Working Group IAP/APA Acute Pancreatitis Guidelines. IAP/APA evidence-based guidelines for the management of acute pancreatitis. *Pancreatology* 2013; 13:e1–e15
14. Trna J, Vege SS, Pribramska V, et al. Lack of significant liver enzyme elevation and gallstones and/or sludge on ultrasound on day 1 of acute pancreatitis is associated with recurrence after cholecystectomy: a population-based study. *Surgery* 2012; 151:199–205
15. Götzinger P, Sautner T, Kriwanek S, et al. Surgical treatment for severe acute pancreatitis: extent and surgical control of necrosis determine outcome. *World J Surg* 2002; 26:474–478
16. De Waele JJ, Hoste E, Blot SI, et al. Perioperative factors determine outcome after surgery for severe acute pancreatitis. *Crit Care* 2004; 8:R504–R511
17. Balthazar EJ. Acute pancreatitis: assessment of severity with clinical and CT evaluation. *Radiology* 2002; 223:603–613
18. Wu BU, Banks PA. Clinical management of patients with acute pancreatitis. *Gastroenterology* 2013; 144:1272–1281
19. Türkvatan A, Erden A, Türkoğlu MA, Seçil M, Yüce G. Imaging of acute pancreatitis and its complications. Part 2. Complications of acute pancreatitis. *Diagn Interv Imaging* 2015; 96:161–169
20. Mortele KJ, Wiesner W, Intriere L, et al. A modified CT severity index for evaluating acute pancreatitis: improved correlation with patient outcome. *AJR* 2004; 183:1261–1265
21. Stimac D, Miletic D, Radic M, et al. The role of nonenhanced magnetic resonance imaging in the early assessment of acute pancreatitis. *Am J Gastroenterol* 2007; 102:997–1004
22. Tsuji Y, Takahashi N, Fletcher JG, et al. Subtraction color map of contrast-enhanced and unenhanced CT for the prediction of pancreatic necrosis in early stage of acute pancreatitis. *AJR* 2014; 202:[web]W349–W356
23. Casas JD, Diaz R, Valderas G, Mariscal A, Cuadras P. Prognostic value of CT in the early assessment of patients with acute pancreatitis. *AJR* 2004; 182:569–574
24. Gardner TB, Vege SS, Chari ST, et al. Faster rate of initial fluid resuscitation in severe acute pancreatitis diminishes in-hospital mortality. *Pancreatology* 2009; 9:770–776
25. Warndorf MG, Kurtzman JT, Bartel MJ, et al. Early fluid resuscitation reduces morbidity among patients with acute pancreatitis. *Clin Gastroenterol Hepatol* 2011; 9:705–709
26. Balthazar EJ, Robinson DL, Megibow AJ, Ranson JH. Acute pancreatitis: value of CT in establishing prognosis. *Radiology* 1990; 174:331–336
27. Viremouneix L, Monneuse O, Gautier G, et al. Prospective evaluation of nonenhanced MR imaging in acute pancreatitis. *J Magn Reson Imaging* 2007; 26:331–338
28. Bollen TL, Singh VK, Maurer R, et al. A comparative evaluation of radiologic and clinical scoring systems in the early prediction of severity in acute pancreatitis. *Am J Gastroenterol* 2012; 107:612–619
29. Papachristou GI, Muddana V, Yadav D, et al. Comparison of BISAP, Ranson's, APACHE-II, and CTSI scores in predicting organ failure, complications, and mortality in acute pancreatitis. *Am J Gastroenterol* 2010; 105:435–441
30. Schepers NJ, Besselink MG, van Santvoort HC, Bakker OJ, Bruno MJ; Dutch Pancreatitis Study Group. Early management of acute pancreatitis. *Best Pract Res Clin Gastroenterol* 2013; 27:727–743
31. Banks PA, Freeman ML; Practice Parameters Committee of the American College of Gastroenterology. Practice guidelines in acute pancreatitis. *Am J Gastroenterol* 2006; 101:2379–2400
32. Singh VK, Bollen TL, Wu BU, et al. An assessment of the severity of interstitial pancreatitis. *Clin Gastroenterol Hepatol* 2011; 9:1098–1103
33. O'Neill E, Hammond N, Miller FH. MR imaging of the pancreas. *Radiol Clin North Am* 2014; 52:757–777
34. Miller FH, Keppke AL, Dalal K, Ly JN, Kamler VA, Sica GT. MRI of pancreatitis and its complications. Part 1. Acute pancreatitis. *AJR* 2004; 183:1637–1644
35. Sica GT, Miller FH, Rodriguez G, McTavish J, Banks PA. Magnetic resonance imaging in pa-

tients with pancreatitis: evaluation of signal intensity and enhancement changes. *J Magn Reson Imaging* 2002; 15:275–284

36. Thomas S, Kayhan A, Lakadamyali H, Oto A. Diffusion MRI of acute pancreatitis and comparison with normal individuals using ADC values. *Emerg Radiol* 2012; 19:5–9
37. Cannon JW, Callery MP, Vollmer CM Jr. Diagnosis and management of pancreatic pseudocysts: what is the evidence? *J Am Coll Surg* 2009; 209:385–393
38. Stringfellow GV, Casola G, Wittich GR, Shankar S, Shamos R. Management of fluid collections in acute pancreatitis. In: Beger HGW, Büchler MW, Kozarek RA, et al., eds. *The pancreas: an integrated textbook of basic science, medicine, and surgery*, 2nd ed. Malden, MA: Blackwell, 2009:344–355
39. Balthazar EJ, Freeny PC, vanSonnenberg E. Imaging and intervention in acute pancreatitis. *Radiology* 1994; 193:297–306
40. Sakorafas GH, Tsiotos GG, Sarr MG. Extrapancreatic necrotizing pancreatitis with viable pancreas: a previously under-appreciated entity. *J Am Coll Surg* 1999; 188:643–648
41. Morgan DE, Baron TH, Smith JK, Robbin ML, Kenney PJ. Pancreatic fluid collections prior to intervention: evaluation with MR imaging compared with CT and US. *Radiology* 1997; 203:773–778
42. Ward J, Chalmers AG, Guthrie AJ, Larvin M, Robinson PJ. T2-weighted and dynamic enhanced MRI in acute pancreatitis: comparison with contrast enhanced CT. *Clin Radiol* 1997; 52:109–114
43. Martin DR, Karabulut N, Yang M, McFadden DW. High signal peripancreatic fat on fat-suppressed spoiled gradient echo imaging in acute pancreatitis: preliminary evaluation of the prognostic significance. *J Magn Reson Imaging* 2003; 18:49–58
44. Hirota M, Kimura Y, Ishiko T, Beppu T, Yamashita Y, Ogawa M. Visualization of the heterogeneous internal structure of so-called “pancreatic necrosis” by magnetic resonance imaging in acute necrotizing pancreatitis. *Pancreas* 2002; 25:63–67
45. Rana SS, Bhasin DK, Reddy YR, et al. Morphological features of fluid collections on endoscopic ultrasound in acute necrotizing pancreatitis: do they change over time? *Ann Gastroenterol* 2014; 27:258–261
46. Easler J, Papachristou GI. The morphologic evolution of necrotic pancreatic fluid collections and their management: asymptomatic—delay, defer and don’t panic! *Ann Gastroenterol* 2014; 27:191–192
47. Brun A, Agarwal N, Pitchumoni CS. Fluid collections in and around the pancreas in acute pancreatitis. *J Clin Gastroenterol* 2011; 45:614–625
48. Bharwani N, Patel S, Prabhudesai S, Fotheringham T, Power N. Acute pancreatitis: the role of imaging in diagnosis and management. *Clin Radiol* 2011; 66:164–175
49. Türkvatan A, Erden A, Seçil M, Türkoğlu MA. Fluid collections associated with acute pancreatitis: a pictorial essay. *Can Assoc Radiol J* 2014; 65:260–266
50. Morgan DE. Imaging of acute pancreatitis and its complications. *Clin Gastroenterol Hepatol* 2008; 6:1077–1085
51. Memiş A, Parildar M. Interventional radiological treatment in complications of pancreatitis. *Eur J Radiol* 2002; 43:219–228
52. Aghdassi AA, Mayerle J, Kraft M, Sielenkamper AW, Heidecke CD, Lerch MM. Pancreatic pseudocysts: when and how to treat? *HPB (Oxford)* 2006; 8:432–441
53. Nguyen BL, Thompson JS, Edney JA, Bragg LE, Rikkers LF. Influence of the etiology of pancreatitis on the natural history of pancreatic pseudocysts. *Am J Surg* 1991; 162:527–530
54. Kim KO, Kim TN. Acute pancreatic pseudocyst: incidence, risk factors, and clinical outcomes. *Pancreas* 2012; 41:577–581
55. Macari M, Finn ME, Bennett GL, et al. Differentiating pancreatic cystic neoplasms from pancreatic pseudocysts at MR imaging: value of perceived internal debris. *Radiology* 2009; 251:77–84
56. Lenhart DK, Balthazar EJ. MDCT of acute mild (nonnecrotizing) pancreatitis: abdominal complications and fate of fluid collections. *AJR* 2008; 190:643–649
57. Stamatakos M, Stefanaki C, Kontzoglou K, Stergiopoulos S, Giannopoulos G, Safioleas M. Walled-off pancreatic necrosis. *World J Gastroenterol* 2010; 16:1707–1712
58. Ramia JM, de la Plaza R, Quinones-Sampedro JE, Ramiro C, Veguillas P, Garcia-Parreno J. Walled-off pancreatic necrosis. *Neth J Med* 2012; 70:168–171
59. Papachristou GI, Takahashi N, Chahal P, Sarr MG, Baron TH. Peroral endoscopic drainage/debridement of walled-off pancreatic necrosis. *Ann Surg* 2007; 245:943–951
60. Gluck M, Ross A, Irani S, et al. Endoscopic and percutaneous drainage of symptomatic walled-off pancreatic necrosis reduces hospital stay and radiographic resources. *Clin Gastroenterol Hepatol* 2010; 8:1083–1088
61. Munene G, Dixon E, Sutherland F. Open transgastric debridement and internal drainage of symptomatic non-infected walled-off pancreatic necrosis. *HPB (Oxford)* 2011; 13:234–239
62. Kozarek R. Endotherapy for organized pancreatic necrosis. *Gastroenterol Hepatol (N Y)* 2013; 9:98–127
63. Hariri M, Slivka A, Carr-Locke DL, Banks PA. Pseudocyst drainage predisposes to infection when pancreatic necrosis is unrecognized. *Am J Gastroenterol* 1994; 89:1781–1784
64. Takahashi N, Papachristou GI, Schmit GD, et al. CT findings of walled-off pancreatic necrosis (WOPN): differentiation from pseudocyst and prediction of outcome after endoscopic therapy. *Eur Radiol* 2008; 18:2522–2529
65. Baron TH, Harewood GC, Morgan DE, Yates MR. Outcome differences after endoscopic drainage of pancreatic necrosis, acute pancreatic pseudocysts, and chronic pancreatic pseudocysts. *Gastrointest Endosc* 2002; 56:7–17
66. Fischer TD, Gutman DS, Hughes SJ, Trevino JG, Behrns KE. Disconnected pancreatic duct syndrome: disease classification and management strategies. *J Am Coll Surg* 2014; 219:704–712
67. Paulson EK, Vitellas KM, Keogan MT, Low VH, Nelson RC. Acute pancreatitis complicated by gland necrosis: spectrum of findings on contrast-enhanced CT. *AJR* 1999; 172:609–613
68. Pelaez-Luna M, Vege SS, Petersen BT, et al. Disconnected pancreatic duct syndrome in severe acute pancreatitis: clinical and imaging characteristics and outcomes in a cohort of 31 cases. *Gastrointest Endosc* 2008; 68:91–97
69. Tirkes T, Sandrasegaran K, Sanyal R, et al. Secretin-enhanced MR cholangiopancreatography: spectrum of findings. *RadioGraphics* 2013; 33:1889–1906
70. Larsen M, Kozarek R. Management of pancreatic ductal leaks and fistulae. *J Gastroenterol Hepatol* 2014; 29:1360–1370
71. Besselink MG, van Santvoort HC, Boermeester MA, et al. Timing and impact of infections in acute pancreatitis. *Br J Surg* 2009; 96:267–273
72. Petrov MS, Shanbhag S, Chakraborty M, Phillips AR, Windsor JA. Organ failure and infection of pancreatic necrosis as determinants of mortality in patients with acute pancreatitis. *Gastroenterology* 2010; 139:813–820
73. Maher MM, Lucey BC, Gervais DA, Mueller PR. Acute pancreatitis: the role of imaging and interventional radiology. *Cardiovasc Intervent Radiol* 2004; 27:208–225
74. da Costa DW, Boerma D, van Santvoort HC, et al. Staged multidisciplinary step-up management for necrotizing pancreatitis. *Br J Surg* 2014; 101:e65–e79
75. Triantopoulou C, Delis S, Dervenis C. Imaging evaluation of post-pancreatitis infection. *Infect Disord Drug Targets* 2010; 10:15–20
76. Rerknimitr R, Lakananurak N, Prueksapanich P, et al. A fatal case of a colonic fistula communicating with a walled-off area of pancreatic necrosis. *Endoscopy* 2014; 46(suppl 1):E30–E31

77. Islim F, Salik AE, Bayramoglu S, Guven K, Alis H, Turhan AN. Non-invasive detection of infection in acute pancreatic and acute necrotic collections with diffusion-weighted magnetic resonance imaging: preliminary findings. *Abdom Imaging* 2014; 39:472–481
78. Inan N, Arslan A, Akansel G, Anik Y, Demirci A. Diffusion-weighted imaging in the differential diagnosis of cystic lesions of the pancreas. *AJR* 2008; 191:1115–1121
79. Sahni VA, Mortele KJ. The bloody pancreas: MDCT and MRI features of hypervascular and hemorrhagic pancreatic conditions. *AJR* 2009; 192:923–935
80. Chu KE, Sun CK, Wu CC, Yang KC. Complete remission of pancreatic pseudoaneurysm rupture with arterial embolization in a patient with poor risk for surgery: a case report. *Case Rep Gastroenterol* 2012; 6:254–259
81. Saad NE, Saad WE, Davies MG, Waldman DL, Fultz PJ, Rubens DJ. Pseudoaneurysms and the role of minimally invasive techniques in their management. *RadioGraphics* 2005; 25(suppl 1):S173–S189
82. 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* 2014 [Epub ahead of print]
83. van Santvoort HC, Bakker OJ, Bollen TL, et al. A conservative and minimally invasive approach to necrotizing pancreatitis improves outcome. *Gastroenterology* 2011; 141:1254–1263
84. Varadarajulu S, Bang JY, Sutton BS, Trevino JM, Christein JD, Wilcox CM. Equal efficacy of endoscopic and surgical cystogastrostomy for pancreatic pseudocyst drainage in a randomized trial. *Gastroenterology* 2013; 145:583–590
85. Bennett S, Lorenz JM. The role of imaging-guided percutaneous procedures in the multidisciplinary approach to treatment of pancreatic fluid collections. *Semin Intervent Radiol* 2012; 29:314–318
86. Barthet M, Sahel J, Bodiou-Bertei C, Bernard JP. Endoscopic transpapillary drainage of pancreatic pseudocysts. *Gastrointest Endosc* 1995; 42:208–213
87. Catalano MF, Geenen JE, Schmalz MJ, Johnson GK, Dean RS, Hogan WJ. Treatment of pancreatic pseudocysts with ductal communication by transpapillary pancreatic duct endoprosthesis. *Gastrointest Endosc* 1995; 42:214–218
88. Lin H, Zhan XB, Jin ZD, Zou DW, Li ZS. Prognostic factors for successful endoscopic transpapillary drainage of pancreatic pseudocysts. *Dig Dis Sci* 2014; 59:459–464
89. Varghese JC, Masterson A, Lee MJ. Value of MR pancreatography in the evaluation of patients with chronic pancreatitis. *Clin Radiol* 2002; 57:393–401
90. Mouli VP, Sreenivas V, Garg PK. Efficacy of conservative treatment, without necrosectomy, for infected pancreatic necrosis: a systematic review and meta-analysis. *Gastroenterology* 2013; 144:333–340
91. Ross AS, Irani S, Gan SI, et al. Dual-modality drainage of infected and symptomatic walled-off pancreatic necrosis: long-term clinical outcomes. *Gastrointest Endosc* 2014; 79:929–935
92. Cirocchi R, Trastulli S, Desiderio J, et al. Minimally invasive necrosectomy versus conventional surgery in the treatment of infected pancreatic necrosis: a systematic review and a meta-analysis of comparative studies. *Surg Laparosc Endosc Percutan Tech* 2013; 23:8–20
93. Kumar N, Conwell DL, Thompson CC. Direct endoscopic necrosectomy versus step-up approach for walled-off pancreatic necrosis: comparison of clinical outcome and health care utilization. *Pancreas* 2014; 43:1334–1339
94. Besselink MG, Verwer TJ, Schoenmaeckers EJ, et al. Timing of surgical intervention in necrotizing pancreatitis. *Arch Surg* 2007; 142:1194–1201
95. Mier J, Leon EL, Castillo A, Robledo F, Blanco R. Early versus late necrosectomy in severe necrotizing pancreatitis. *Am J Surg* 1997; 173:71–75
96. Bang JY, Wilcox CM, Trevino J, et al. Factors impacting treatment outcomes in the endoscopic management of walled-off pancreatic necrosis. *J Gastroenterol Hepatol* 2013; 28:1725–1732
97. Rana SS, Bhasin DK, Sharma RK, Kathiresan J, Gupta R. Do the morphological features of walled off pancreatic necrosis on endoscopic ultrasound determine the outcome of endoscopic transmural drainage? *Endosc Ultrasound* 2014; 3:118–122
98. Boerma D, Rauws EA, van Gulik TM, Huibregtse K, Obertop H, Gouma DJ. Endoscopic stent placement for pancreaticocutaneous fistula after surgical drainage of the pancreas. *Br J Surg* 2000; 87:1506–1509
99. Fotoohi M, D'Agostino HB, Wollman B, Chon K, Shahrokni S, vanSonnenberg E. Persistent pancreatocutaneous fistula after percutaneous drainage of pancreatic fluid collections: role of cause and severity of pancreatitis. *Radiology* 1999; 213:573–578
100. Saadia R. Fistulas of the pancreas. In: Holzheimer RG, Mannick JA, eds. *Surgical treatment: evidence-based and problem-oriented*. Munich, Germany: Zuckschwerdt, 2001. Available at http://www.ncbi.nlm.nih.gov/books/NBK7003/