Imaging of Acute Pancreatitis

Ruedi F. Thoeni, MD

KEYWORDS

• Acute pancreatitis • Interstitial edematous pancreatitis • Necrotizing pancreatitis
• Acute peripancreatic fluid collections • Acute necrotic collection • Pseudocyst
• Walled-off pancreatic necrosis • Systemic inflammatory response syndrome

KEY POINTS

- The revised Atlanta classification of acute pancreatitis provides a consistent and universally adaptable system for defining acute pancreatitis in its various stages.
- The revised Atlanta classification of acute pancreatitis distinguishes interstitial edematous pancreatitis from necrotizing pancreatitis and defines early and late phases.
- The revised Atlanta classification of acute pancreatitis divides severity into mild, moderately severe, and severe form and describes transient (≤48 hours) and persistent (>48 hours) organ failure, the presence or absence of which defines these stages.
- The revised Atlanta classification of acute pancreatitis provides a new and clear definition for pancreatic fluid collections (local complications) based on the absence or presence of necrosis: acute peripancreatic fluid collection, acute necrotic collection, pseudocyst, walled-off necrosis, and postnecrosectomy pseudocyst. All fluid collections may be sterile or infected.
- In the first week, only clinical parameters are important for treatment planning, but after the first week, morphologic criteria defined by computed tomography combine with clinical parameters to determine care.

INTRODUCTION

Acute pancreatitis represents an acute inflammatory disorder of the pancreas that is initiated by premature activation of digestive enzymes in the pancreatic acinar cells, leading to autodigestion of the pancreas. The process consists of local inflammation of the pancreas and an exaggerated systemic inflammatory response syndrome (SIRS) to the pancreatic injuries, which may result in multisystem organ failure.[1] Its incidence is increasing worldwide related to well-established risk factors, such as obesity, increased aging of the general population, and rising incidence of gallstone disease.[2] In the United States, alcohol abuse and gallstone disease are the most common causes of acute pancreatitis.[2,3] About 80% to 85% of patients suffer only from the mild form of pancreatitis, whereas approximately 15% to 20% develop a severe course with complications that can include organ failure, local complications, and even death.[4]

In 1992, the original Atlanta classification system was introduced as a method for defining terminology of acute pancreatitis, its severity, organ failure, and complications.[5] However, over the years, many researchers and clinicians found this classification system insufficient and at times confusing. The need for revision also was prompted by new insights into the pathophysiology of the disease, markedly improved imaging, and new treatment options. These new treatment options included minimally invasive radiologic, endoscopic, and operative procedures for local complications that

Department of Radiology and Biomedical Imaging, University of California, San Francisco, Medical School, PO Box 2829, San Francisco, CA 94126-2829, USA
E-mail address: remo148@yahoo.com

Radiol Clin N Am 53 (2015) 1189–1208
http://dx.doi.org/10.1016/j.rcl.2015.06.006
0033-8389/15/$ – see front matter

changed the management of patients. Therefore, an international working group on acute pancreatitis was formed to revise the Atlanta classification and published its revision in 2013.[6] The important topics that were included in this revision were a clear assessment and definition of clinical and morphologic severity of disease and clear definition of the various pancreatic and peripancreatic fluid collections that develop over the course of this disease. It also outlined what radiologists should look for when evaluating a patient with acute pancreatitis who undergoes imaging. Importantly, it stressed that in the early phase of the disease, there is no correlation between the severity of clinical disease and morphologic manifestations as seen by imaging, which limits the role of imaging in the early phase.

Because computed tomography (CT) is widely available and considered the standard in evaluating patients with acute pancreatitis, the emphasis of this revision is on CT. Magnetic resonance (MR) was recommended in selected cases to define the nature of a fluid collection or when CT is contraindicated. Both ultrasound and MR are considered superior to CT in diagnosing the heterogeneity of pancreatic collections and visualizing the presence of nonliquefied necrotic material, which is important for management. It remains to be seen if dual-energy CT might improve results in this area.[7] This article addresses the clinical definition of acute pancreatitis, defines the various pancreatic fluid collections and complications as demonstrated by CT and MR, and assesses the role of imaging in these patients.

REVISED TERMINOLOGY AND CLASSIFICATION

Diagnosis of Acute Pancreatitis

The clinical diagnosis of acute pancreatitis is based on 2 of 3 features[6]:

1. Abdominal pain highly suggestive of acute pancreatitis (acute onset of severe epigastric pain, often radiating to the back).
2. Serum lipase or amylase activity at least 3 times the upper limits of normal.
3. Characteristic features of acute pancreatitis on CT and less commonly on MR imaging or ultrasound.

If serum lipase and amylase are not sufficiently elevated but symptoms strongly suggest acute pancreatitis, imaging needs to be used to confirm the diagnosis. Imaging is usually not required in the emergency department or on the first day of admission if acute pancreatitis can be diagnosed based on typical symptoms and elevated lipase/amylase activity. However, imaging may be needed in patients with inconclusive presentations.

Onset of acute pancreatitis is defined by the beginning of acute abdominal pain and not by the time of arrival in the hospital or emergency room.

Phases of Pancreatitis

The revised Atlanta classification distinguishes between an early (within the first week) and a late phase (after the first week).[6]

Early phase

This phase usually takes place in the first week only but occasionally extends into the second week. The early phase is characterized by a systemic response to the pancreatic injury. As a result of a cytokine cascade caused by the inflammatory injury to the pancreas, a SIRS develops. If SIRS persists, organ failure may develop. During the early phase of acute pancreatitis, the severity of the attack is determined by the presence and duration of organ failure. The Atlanta classification defines organ failure as transient if it lasts for 48 hours or less, and as persistent if it lasts for greater than 48 hours. It may affect a single organ or multiple organs (multiorgan failure). In the early stage, possible pancreatic necrosis cannot by diagnosed with certainty. Local complications do not determine severity, because there is no direct correlation between the degree of morphologic changes and the severity of organ failure. In the early phase, the categorization of acute pancreatitis as moderately severe or severe is defined by the presence and duration of organ failure, which is determined based entirely on clinical criteria.

Late phase

The late phase occurs only in patients with moderately severe or severe pancreatitis because it is characterized by persistence of systemic signs of inflammation or by development of local or systemic complications. Local complications evolve over time and CT plays an important role in defining the type and extent of complications for best management. Nevertheless, persistent organ failure is an important factor in determining severity, and this phase is categorized based on both clinical data and morphologic findings.

Types of Pancreatitis

According to the revised Atlanta classification, acute pancreatitis is split into interstitial edematous pancreatitis and necrotizing pancreatitis.[6]

Interstitial edematous pancreatitis

In interstitial, edematous pancreatitis, the pancreas shows diffuse and sometimes localized

enlargement of the parenchyma with heterogeneous and occasionally homogeneous enhancement and peripancreatic fat stranding or fluid collections (see discussion under fluid collections). Usually the symptoms in patients with interstitial edematous pancreatitis resolve within 1 week.[8]

Necrotizing pancreatitis

Necrotizing pancreatitis may involve the pancreatic parenchyma and peripancreatic tissue, the peripancreatic tissue alone, or the pancreas parenchyma alone. The combination of pancreas parenchyma and peripancreatic tissue necrosis is most common, and necrosis of the pancreatic parenchyma alone is the least common.[9] The reduced perfusion and necrosis of the pancreas develop slowly over several days.[10] For this reason, necrosis cannot be accurately diagnosed in the initial stage of acute pancreatitis. In the first few days, decreased enhancement of the pancreas may simply reflect edema and, only when an area of decreased perfusion becomes well demarcated (usually after 72 hours) can it be diagnosed as necrosis. In the patient with peripancreatic necrosis alone, the pancreatic parenchyma enhances in a fashion similar to interstitial edematous pancreatitis, but morbidity and the need for intervention are much higher than in interstitial edematous pancreatitis.[11] Patients with parenchymal necrosis alone have a higher risk of organ failure and death than patients with peripancreatic necrosis alone.[12] Evolution of pancreatic necrosis is variable and may resolve over time, persist, become infected, remain solid, or liquefy. There is no direct correlation between extent of necrosis, symptoms, and risk of superinfection.[13]

Severity of Pancreatitis

According to the revised Atlanta classification, the severity of acute pancreatitis can be divided into mild, moderately severe, and severe (**Table 1**).[6] Classifying the degree of severity is important because of the difference in treatment of the various degrees of severity (eg, patients with suspected severe acute pancreatitis need aggressive treatment, whereas patients with mild acute pancreatitis do not). On CT, the various forms of acute pancreatitis are staged by the CT severity index (CTSI), which is an imaging grading system that combines grading of intrapancreatic and extrapancreatic inflammatory changes with extent of pancreatic necrosis (**Table 2**).[14,15] This grading system can help predict morbidity and mortality in patients with acute pancreatitis and guide treatment planning. A recent retrospective study in 549 patients validated the revised Atlanta classification system for severity of acute pancreatitis and outcome.[16]

Table 1
Grades of severity in acute pancreatitis

Grade	Definition
Mild acute pancreatitis	No organ failure No local or systemic complications
Moderately severe acute pancreatitis	Transient organ failure for ≤48 h and/or local or systemic complications without persistent organ failure
Severe acute pancreatitis	Persistent organ failure for >48 h Single organ failure Multiple organ failure ± Local and/or systemic complications[a]

[a] Some patients with persistent organ failure may recover without local complications.[19]

Adapted from 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;62:108; with permission.

Table 2
Computed tomographic severity index

Characteristics	Grade	Points
Inflammation of the pancreas		
Normal pancreas	A	0
Pancreatic enlargement, focal or diffuse	B	1
Peripancreatic inflammation	C	2
Single peripancreatic fluid collection	D	3
Two or more peripancreatic fluid collections	E	4
Pancreatic parenchymal necrosis		
No necrosis	—	0
<30%	—	2
Between 30% and 50%	—	4
More than 50%	—	6

Points for pancreatic inflammation and necrosis are added for total CTSI score.

Adapted from Sakorafas GH, Tsiotos GG, Sarr MG. Extrapancreatic necrotizing pancreatitis with viable pancreas: a previously under-appreciated entity. J Am Coll Surg 1999;188:643–8; and Balthazar EJ. Acute pancreatitis: assessment of severity with clinical and CT evaluation. Radiology 2002;223:10.

Mild pancreatitis

Mild acute pancreatitis is present when no organ failure develops and systemic and local complications are absent. Patients with mild acute pancreatitis usually can be discharged in the first week and mortality is rare and associated with comorbidities.[17]

Moderately severe pancreatitis

Transient organ failure, local complications, and exacerbation of comorbid disease typify moderately severe acute pancreatitis. When transient organ failure transitions into persistent organ failure, the condition becomes severe acute pancreatitis. Local complications can consist of peripancreatic fluid collections that result in leukoytosis, fever, or abdominal pain over an extended period of time. Even inability to tolerate food orally can be caused by local complications. Systemic complications brought about by acute pancreatitis include increased symptoms from pre-existing disease (see later discussion under systemic complications). Moderately severe pancreatitis leads to increased morbidity and mortality compared with mild acute pancreatitis, but both are lower than seen in severe pancreatitis.[18] Moderately severe acute pancreatitis may resolve spontaneously or may require prolonged care.

Severe pancreatitis

Severe acute pancreatitis is characterized by persistent organ failure that may involve a single organ or multiple organs (see discussion of organ failure later). As mentioned earlier, SIRS brings about organ failure. SIRS carries an increased risk of persistent organ failure if it continues. Patients with persistent organ failure usually also develop local complications and have a high mortality if persistent organ failure develops early in the disease process.[19] Some patients with persistent organ failure may recover without local complications.[20] The mortality increases further with superinfection of necrosis.[21]

Complications

According to the revised Atlanta classification system, complications from acute pancreatitis can be divided into organ failure, systemic complications, and local complications.[6]

Organ failure

The respiratory, renal, and cardiovascular systems are used for determining organ failure. Organ failure is usually assessed based on the Marshall scoring system, which is a simplified and universally accepted system that can be used initially and repeated daily to gauge disease severity (**Table 3**).[6,22,23] Based on this system, a score of 2 or more for one of these 3 organ systems constitutes organ failure. Persistent multiorgan failure is defined as 2 or more organs failing over the same 3-day period.

Systemic complications

Besides persistent organ failure, other features that define severe acute pancreatitis include systemic complications, which are events that are triggered by acute pancreatitis and represent acute exacerbations of pre-existing comorbidities such as chronic lung disease and coronary artery disease.

Local complications

The term local complication was used in the original Atlanta classification and has been retained as useful to distinguish between acute edematous interstitial pancreatitis without complications and acute pancreatitis with local complications.[5] In the revised Atlanta classification, local complications consist of acute peripancreatic fluid collections (APFCs), pseudocysts, acute necrotic collections (ANCs), and walled-off necroses

Table 3
Marshall scoring system for acute pancreatitis

	Score				
Organ System	**0**	**1**	**2**	**3**	**4**
Respiratory (PaO_2/Fio_2)	>400	301–400	201–300	101–200	<101
Renal[a] (serum creatinine, mg/dL)	≤1.5	>1.5 to ≤1.9	>1.9 to ≤3.5	>3.5 to ≤5.0	>5.0
Cardiovascular (systolic blood pressure, mm Hg)	>90	<90 Fluid responsive	<90 Not fluid responsive	<90 pH < 7.3	<90 pH < 7.2

Organ failure is described as a score ≥2 for at least 1 of these 3 organ systems.
Duration of organ failure is defined as transient (≤48 h) or persistent (>48 h) from the time of presentation.
Persistent multiorgan failure is defined as 2 or more organs failing over the same 3-day period.
[a] If pre-existing chronic renal failure is present, the score depends on further worsening of the baseline renal function.
Data from Refs.[6,22,23]

(WONs).[6] These various collections are defined by CT and are described in detail later under the heading "Imaging Findings." They may be sterile or infected. Other complications caused by necrotizing pancreatitis include pseudoaneurysm, splenic or portal vein thrombosis, obstruction or ileus of the gastrointestinal tract, contiguous inflammation of the colon, biliary stones, cholecystitis, pancreatic duct strictures, involvement of neighboring solid organs, ascites, and pleural effusions.[9] The description of local complications should include their locations and morphologic appearances, such as wall thickness, heterogeneity of the collection, and possible presence of extraluminal gas. Such local complications are suspected when there is a change in the clinical presentation, such as increased abdominal pain, fever, or increasing organ failure.

IMAGING TECHNIQUES

Computed Tomography

Imaging usually is not required in the initial phase of acute pancreatitis or in patients with acute pancreatitis who are rapidly improving clinically. However, imaging is recommended in the initial phase when the diagnosis is indeterminate clinically. Unless contraindicated related to renal function, contrast-enhanced CT (CECT) should be used in patients with SIRS, organ failure, or other clinical or biochemical predictors of severe acute pancreatitis and in patients who clinically are suspected to develop complications due to acute pancreatitis.[24] The best time for scanning these patients by CECT is after 72 hours from onset of symptoms. The patient should be re-examined with CECT when the clinical picture drastically changes for the worse, such as when fever suddenly develops, a drop in hematocrit is encountered, or sepsis ensues. A CECT also is useful for guidance of catheter placement to drain fluid collections and for determining the success of treatment in patients who have undergone percutaneous drainage or other interventions including surgical debridement. In addition, CECT is indicated to exclude a possible pancreatic neoplasm in patients who have a first episode of pancreatitis and are 40 years of age or older with no identifiable cause for pancreatitis.[25]

In assessing for acute pancreatitis, intravenous contrast is needed because the CT diagnosis of pancreatic necrosis relies on lack of enhancement in the necrotic area of the pancreas. Following initial scout images, the upper abdomen is scanned with a low-dose technique to look for stones, calcifications, and possible hemorrhage. At the University of California, San Francisco, a dual-phase pancreatic CECT is preferred for the initial study, which on follow-up studies can be reduced to a single-phase pancreas protocol at 70 to 80 seconds. Using a multidetector row CT, the dual-phase protocol is performed at 45 and 70 to 80 seconds extending from the diaphragm to the iliac crest at a slice thickness of 1.25 to 2.5 mm. Reformations in the coronal and sagittal planes also are performed. The coronal plane is particularly useful for assessing the extent of fluid collections in the abdomen. If the patient can tolerate it, neutral oral contrast (water or VoLumen; Bracco Diagnostics, Inc, Princeton, NJ, USA) is administered for improved visualization of the duodenal sweep and stomach. Dose reduction strategies should be used. In patients with renal insufficiency or contrast allergies, a nonenhanced CT or MR can be performed.[26–28] Subtraction color maps may be helpful for diagnosing pancreatic necrosis in the early stages of acute pancreatitis (within first 72 hours).[29]

Magnetic Resonance

MR generally is not the modality of choice for evaluating patients with acute pancreatitis but is useful in patients with impaired renal function or allergies to iodinated contrast, in young or pregnant patients, in patients with suspected choledocholithiasis not seen on CECT, and when assessment of the composition of a pancreatic fluid collection is needed to determine presence or absence of nonliquefied material (necrotic debris) or superinfection.[30–34] MR is more sensitive than CT for detecting hemorrhage and for demonstrating communication of a collection with the pancreatic duct. Although MR with diffusion-weighted sequences may be equivalent to iodinated contrast material–enhanced CT for the diagnosis of acute pancreatitis, it is superior to nonenhanced CT.[34]

For complete evaluation of the pancreatic duct and parenchyma, the following sequences are used at the University of California, San Francisco: in-phase and opposed-phase T1-weighted gradient echo in the axial plane, T2-weighted single-shot fast spin echo or turbo spin echo in the axial and coronal plane, T2-weighted fast recovery spin echo with fat suppression in the axial plane, and T1-weighted, 3-dimensional dynamic gradient echo before and after gadolinium with fat suppression in the axial plane. Diffusion-weighted MR imaging is added because it enables differentiation between different degrees of severity of acute pancreatitis and between sterile and infected collections associated with acute pancreatitis.[33,34] Heavily T2-weighted MRCP sequences may be added when needed as a slab or 3-dimensional

technique in the coronal plane. Because MR protocols are persistently evolving, the details are not listed here. For more detailed information as a snapshot in time, please refer to the reviews by Barral and colleagues[34] and Tirkes and colleagues.[35]

IMAGING FINDINGS

Acute Interstitial Edematous Pancreatitis

On CECT, acute pancreatitis manifests itself as either focal or diffuse enlargement of the pancreas with homogeneous or slightly heterogeneous enhancement (**Fig. 1**). At times, very subtle inflammatory changes may be missed, especially when no previous CT studies are available. The peripancreatic fat may appear normal, particularly in mild cases, or demonstrate mild stranding (**Fig. 2**) or early peripancreatic fluid collections (described later under the heading "Pancreatic and peripancreatic collections") (**Figs. 3** and **4**). In the early stage of acute pancreatitis, pancreatic necrosis cannot be diagnosed with certainty. Decreased perfusion of the pancreatic parenchyma appears as diffuse or focal heterogeneity related to various degrees of edema that can be mistaken for pancreatic necrosis (**Fig. 5**). This finding is especially the case for areas of poor enhancement that are estimated to be less than 30% in the early phase.[14,15] A definitive diagnosis in these patients necessitates a follow-up study, because these findings should be considered indeterminate at this stage. Pancreatic necrosis cannot be diagnosed accurately before 72 hours from onset of acute pancreatitis and is best assessed between 5 and 7 days after onset. Obtaining a CECT at the start of the late phase is recommended in patients with proven or suspected necrotizing pancreatitis to identify those patients who are increased risk for adverse outcomes.[36] Subtraction color maps have been shown to improve diagnostic results for pancreatic necrosis

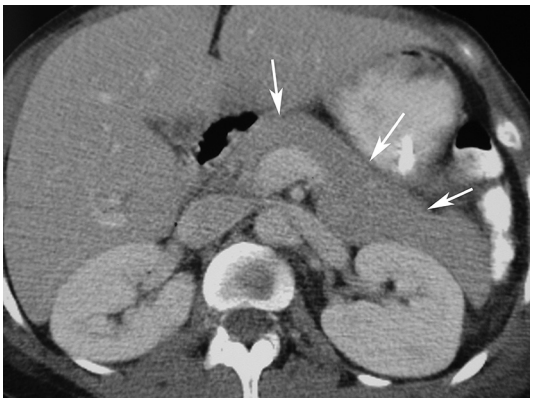

Fig. 1. Acute interstitial edematous pancreatitis, CTSI 1. This 45-year-old man suffered from acute onset of epigastric pain for 12 hours. Acute pancreatitis was diagnosed based on the clinical presentation and elevated lipase and amylase levels. CT shows a slightly enlarged pancreas (*arrows*) without peripancreatic stranding.

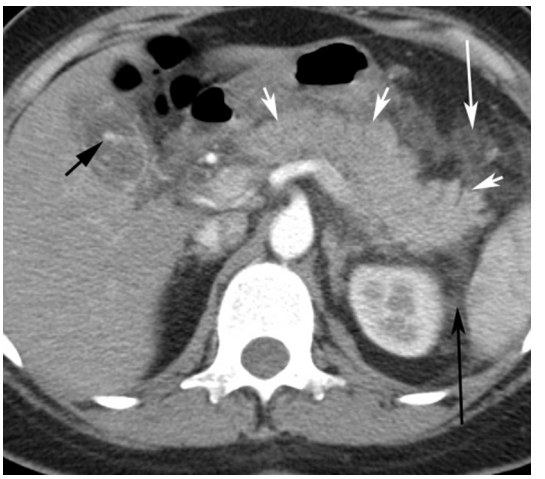

Fig. 2. Acute interstitial edematous pancreatitis, CTSI 2. A CT image in the axial plane of a 34-year-old woman with acute onset of epigastric pain for 48 hours depicts a pancreas that enhances heterogeneously (*short white arrows*) with peripancreatic stranding anterior to the pancreas (*long white arrow*) and in the left anterior pararenal space (*long black arrow*). CT also demonstrates gallstones (*short black arrow*).

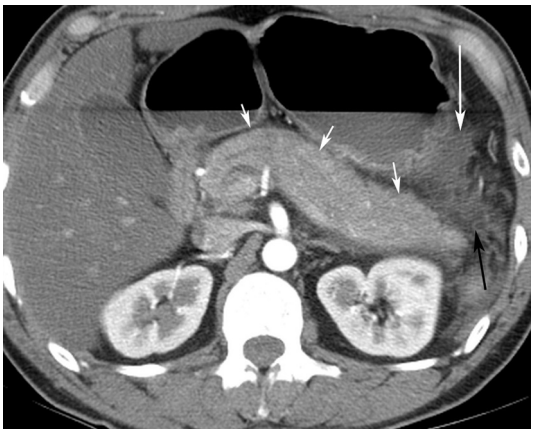

Fig. 3. Acute interstitial edematous pancreatitis with a single peripancreatic fluid collection, CTSI 3. A CT image in the axial plane of a 55-year-old woman demonstrates a swollen pancreas (*short white arrows*), which enhances heterogeneously. Peripancreatic stranding is evident (*black arrow*), and a single peripancreatic fluid collection (APFC) is identified next to the stomach (*long white arrow*).

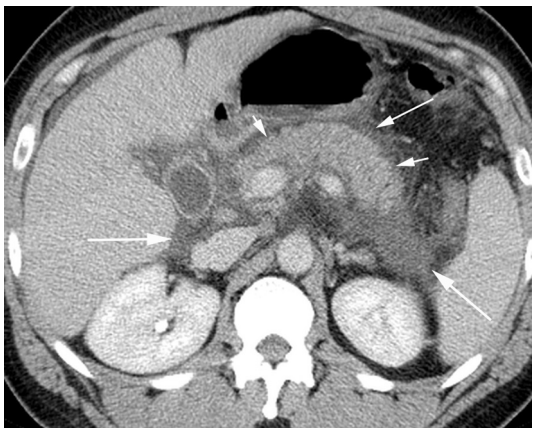

Fig. 4. Acute interstitial edematous pancreatitis with multiple fluid collections, CTSI 4. CT image in the axial plane of a 22-year-old man demonstrates an edematous pancreas (*short arrows*), and multiple fluid collections (APFCs) (*long white arrows*) are present anterior to the pancreas and in both anterior pararenal spaces.

within the first 72 hours,[29] but this has not found general use as yet.

With progression of acute pancreatitis, fluid collections develop around the pancreas, mostly in the lesser sac between the anterior pancreas and the posterior wall of the stomach and in the left pararenal space (see **Figs. 3** and **4**). These collections can persist, enlarge, and become encapsulated or resolve spontaneously. These collections may be superinfected, or hemorrhagic, or they may produce mass symptoms.

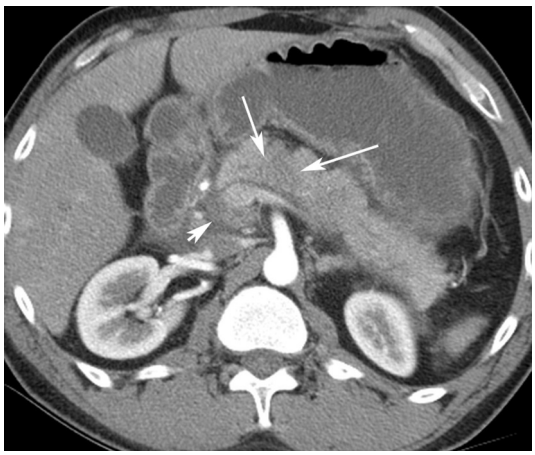

Fig. 5. Acute interstitial edematous pancreatitis for 24 hours. A CT image in the axial plane of a 22-year-old man shows a swollen and edematous pancreas with stranding and a small amount of APFC (*short arrow*). A focal area of decreased perfusion (*long arrows*) is identified in the body of the pancreas. This likely represents focal edema but could be confused with necrosis at this early stage. It should be considered indeterminate. A follow-up CT study after 7 days demonstrated absence of necrosis.

Acute Necrotizing Pancreatitis

The presence of pancreatic necrosis, which CECT depicts as nonperfusion of the pancreatic parenchyma, constitutes necrotizing pancreatitis. It represents the most severe form of acute pancreatitis and occurs in 15% to 20% of patients with acute pancreatitis.[35] This necrotizing process distinguishes necrotizing pancreatitis from interstitial edematous pancreatitis. According to the revised Atlanta classification, 3 forms of acute necrotizing pancreatitis are discerned depending on location of the inflammatory process: involvement of the pancreatic parenchyma alone, the peripancreatic tissues alone, or both.[6] Peripancreatic necrosis generally appears as heterogeneous fluid except in the early stages when CECT may show a homogeneous collection (see later discussion). This characterization by location signifies a distinct change from the original Atlanta classification.[5]

Pancreatic parenchyma necrosis alone

Pancreatic parenchymal necrosis alone is the least common form of necrotizing pancreatitis (<5%).[36,37] Initially, CECT in patients with pancreatic necrosis shows a focally or diffusely enlarged pancreas with the necrosis seen as an area of mostly homogeneous nonenhancement, which over time changes into an area of more heterogeneous attenuation (**Fig. 6**). The changes seen on CECT are caused by the presence of nonviable tissue consisting predominantly of necrotic pancreatic parenchyma and some fat that slowly begins to liquefy. These areas of necrosis traditionally have been divided into 3 categories of involvement of the pancreas parenchyma: less than 30%, 30% to 50%, and greater than 50% (see **Table 2**).[14,15] A newer grading system, the modified CTSI, divided the necrosis into less than 30% and greater than 30% and added other manifestations (such as pleural effusion, ascites, vascular complications, or gastrointestinal complications) to it, but a later analysis did not demonstrate a clear benefit over the original Balthazar grading system.[15,38] Both CTSIs were found to diagnose clinically severe disease more accurately than clinical parameters and to correlate better with the risk of pancreatic infection and the need for intervention.[38]

Peripancreatic necrosis alone

Peripancreatic necrosis alone is the second most common form of necrotizing pancreatitis and may be seen in approximately 20% of patients.[36] It is important to recognize this type of necrosis because these patients have a better prognosis

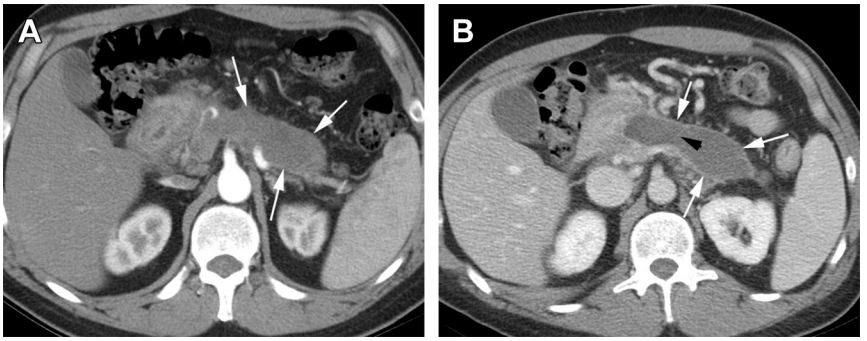

Fig. 6. Acute necrotizing pancreatitis: pancreas parenchyma alone. (*A*) CT image in the axial plane of a 38-year-old woman taken 7 days after onset shows an area of nonenhancement in the tail and body of the pancreas (*arrows*). The ANC is slightly heterogeneous. (*B*) Axial CT image of the same patient as Fig. 6A, 5 weeks later. The ANC has developed into a WON with an enhancing wall (*white arrows*) and septation (*black arrowhead*). It remains an area of heterogeneous attenuation.

than patients with pancreatic parenchymal necrosis.[39] Nonetheless, patients with peripancreatic necrosis alone have a higher morbidity than patients with interstitial edematous pancreatitis only.[17] Peripancreatic necrosis can be diagnosed on CECT when nonenhancing areas of heterogeneous attenuation (Fig. 7) are demonstrated in the lesser sac or retroperitoneum that include liquid and nonliquefied components. The presence or absence of necrotic material in peripancreatic collections can be difficult to determine by CECT, particularly in the early phase, and if clinical concern is strong enough, MR or ultrasound can be used to detect the nonliquefied necrotic components of the fluid collection. More recently it was found that a simple grading system based on the volume of extrapancreatic necrosis and using a threshold of 100 mL provided the best correlation with clinical outcome in patients with necrotizing pancreatitis (predicting organ failure and infection) and was superior to CTSI and measurements of the C-reactive protein level.[39]

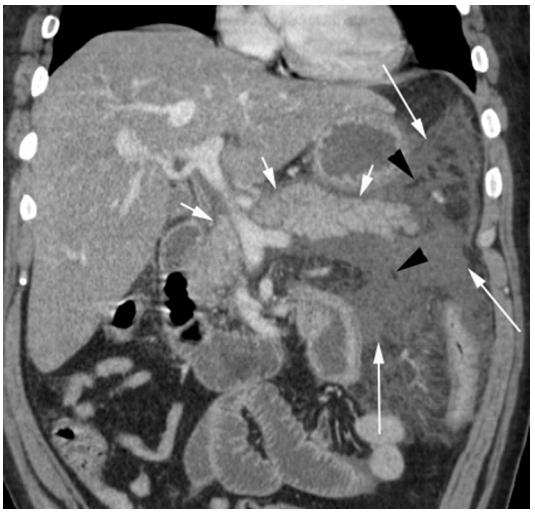

Fig. 7. Acute necrotizing pancreatitis: peripancreatic necrosis alone. A coronal CT image in a 24-year-old man shows an edematous but completely enhanced pancreas (*short white arrows*) with multiple ANCs (*long white arrows*) that are heterogeneous with islands of fat (*black arrowheads*) within the collections.

Combination of pancreatic parenchymal with peripancreatic necrosis

The most common form of acute necrotizing pancreatitis involves necrosis of the pancreatic parenchyma and peripancreatic tissues. It can be seen in 75% to 80% of patients with acute necrotizing pancreatitis.[40] On CECT, a combination of the imaging findings described above for pancreatic parenchymal necrosis alone and peripancreatic necrosis alone (Fig. 8) can be seen.[9] Full-width necrosis of the pancreatic parenchyma may demonstrate a fistula with the main pancreatic duct and often is combined with a significant amount of peripancreatic necrosis.[41]

Pancreatic and Peripancreatic Collections

The revised Atlanta classification makes an important distinction between (i) fluid and (ii) collections with fluid and nonliquefied or only partially liquefied material (Table 4).[6] This distinction is largely new and based on morphologic characteristics depicted on CECT. The characteristics for the various fluid collections depend on the presence of interstitial edematous pancreatitis or necrotizing pancreatitis, location of the collection (peripancreatic or within the pancreas), presence or absence of a capsule, fluid content (fluid or fluid and nonliquefied necrotic material), presence or absence of superinfection, and history of prior intervention (necrosectomy). These fluid collections represent local complications in nonmild forms of acute

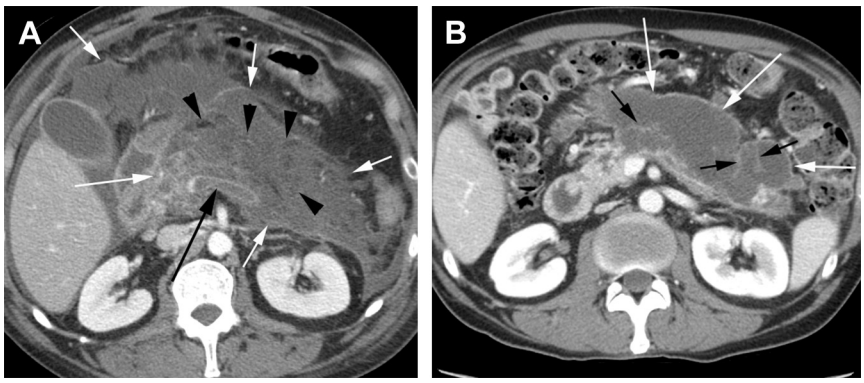

Fig. 8. Acute necrotizing pancreatitis: peripancreatic and parenchymal necrosis, 8 days after onset of symptoms. (*A*) An axial CT image in a 44-year-old woman with heavy EtOH abuse demonstrates necrosis of the pancreatic parenchyma with only the head of the pancreas partially enhancing (*long white arrow*) and extensive ANCs (*short white arrows*) containing areas of fat necrosis and heterogeneity (*black arrowheads*). Also note the splenic vein thrombosis (*long black arrow*). (*B*) Same patient as **Fig. 8**A 5 weeks later. The ANCs have matured into WONs involving the pancreas and peripancreatic areas with an enhancing capsule (*white arrows*) clearly demonstrated. Also, multiple loculations (*black arrows*) are seen.

Table 4
Computed tomographic criteria for local complications of acute pancreatitis based on the revised Atlanta classification

Fluid Collection	CT Features
Interstitial edematous pancreatitis	
APFC	Usually within the first 4 wk from onset **Homogeneous** collection with fluid attenuation Confined by normal peripancreatic fascial planes No fully definable wall surrounding the collection Adjacent to pancreas, no intrapancreatic extension
Pseudocyst (PC)	Usually after 4 wk from onset of symptoms **Homogeneous** collection (round or oval), fluid density Well-defined enhancing wall (complete encapsulation) Absence of nonliquefied material Adjacent to pancreas, no intrapancreatic extension
Necrotizing pancreatitis	
ANC	Usually within the first 4 wk from onset **Heterogeneous** collection with liquid and nonliquid components of various attenuation and varying degrees of loculations No fully definable wall surrounding the collection Located intrapancreatic and/or extrapancreatic
WON	Usually after 4 wk from onset of symptoms **Heterogeneous** collection with liquid and nonliquid components of various attenuations and varying degrees of loculations Well-defined enhancing wall (complete encapsulation) Located intrapancreatic and/or extrapancreatic[a]
Postnecrosectomy pseudocyst	Occurs after necrosectomy for necrotizing pancreatitis **Homogeneous** collection (round or oval), fluid density Well-defined enhancing wall (complete encapsulation) Absence of nonliquefied material Located intrapancreatic and/or extrapancreatic

Note: The term "pancreatic abscess" or "phlegmon" is no longer used.
[a] Often with irregular borders, extending to paracolonic gutters and increasing in size.
Data from Refs.[6,9,24,43]

pancreatitis and require supportive measures or interventional therapy. The original 1992 Atlanta classification distinguished 4 different types of local complications: APFC, pancreatic necrosis, pseudocyst, and pancreatic abscess.[5] The revised Atlanta classification distinguishes 5 types of pancreatic or peripancreatic collections (see **Table 4**). All of these collections can be sterile or infected. The term pancreatic abscess no longer is used because an abscess in the pancreas does not develop without pancreatic necrosis and the infected collection always consists of necrotic material and pus, which has different implications for drainage than a simple pus collection. Also the terms phlegmon and fluid collection are no longer used in describing findings in necrotizing pancreatitis because they are too ambiguous.

Acute peripancreatic fluid collection

In the acute phase of interstitial edematous pancreatitis, usually within the first 4 weeks and in the absence of necrosis, the acute collection is described as an APFC. It is a term new to the revised Atlanta classification. This fluid collection represents an exudate from the inflamed pancreas. It may be associated with rupture of a small peripheral pancreatic side branch duct. At times, there may be no connection to the pancreatic duct. These collections accumulate within the first few days and may resolve spontaneously within the first several weeks after onset of acute pancreatitis.[24] They contain only fluid without any necrotic components (please see **Table 4** for additional morphologic features, see **Figs. 3** and **4**) and no intervention is needed (**Table 5**). APFCs should not be confused with ascites, which is located in the perihepatic and perisplenic areas (**Fig. 9**), in the paracolonic gutters, and in the pelvis.

Pseudocyst

In patients with interstitial edematous pancreatitis and APFC, over time, usually after 4 weeks, pancreatic pseudocysts may develop. This term pseudocyst remains unchanged from the original Atlanta classification. It denotes a fluid collection containing pancreatic juice with a high content of amylase and lipase and has a well-defined wall of granulation tissue (**Fig. 10**). For a detailed description, see **Table 4**. A pseudocyst does not contain any necrotic debris and appears as a homogeneous collection of high signal intensity on T2-weighted images (**Fig. 11**). Occasionally, a connection to the pancreatic duct can be visualized on CECT, particularly if a curviplanar reconstruction is obtained, but MR and endoscopic ultrasound are more accurate in performing this task. Ductal communication is not part of the revised Atlanta criteria for a pseudocyst, but it may be an important finding for deciding on the appropriate treatment (see **Table 5**). Pseudocysts are rare in acute pancreatitis in contrast to chronic pancreatitis. Infection of a pseudocyst is uncommon.

Table 5
Possible interventions in patients with acute pancreatitis and local complications

Type of Collection	Location	Infection	Drainage or Surgery
Interstitial edematous pancreatitis			
APFC	Adjacent to pancreas, extrapancreatic only	Extremely rare	None
Pseudocyst[a]	Adjacent or distant to pancreas	Rare	Rarely (only if symptomatic or infected)
Necrotizing pancreatitis			
Sterile ANC	Intrapancreatic and/or extrapancreatic	No	Based on clinical picture, percutaneous drainage at times, rarely endoscopic or surgical procedure
Infected ANC	Intrapacreatic and/or extrapancreatic	Yes	Percutaneous drainage, endoscopic procedure or surgery later if needed
Sterile WON	Intrapancreatic and/or extrapancreatic	No	Percutaneous drainage based on clinical picture, endoscopic procedure or surgery to follow if needed
Infected WON	Intrapancreatic and/or extrapancreatic	Yes	Percutaneous drainage, endoscopic procedure or surgery to follow if needed

Terminology based on references[6,9].
[a] Can be seen in disconnected duct syndrome and after necrosectomy.

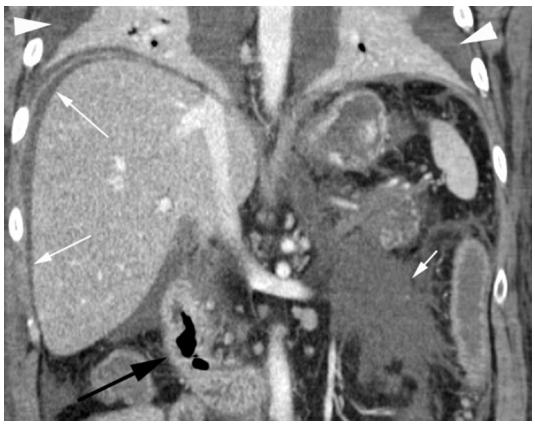

Fig. 9. Acute interstitial edematous pancreatitis with ascites and bilateral pleural effusions. The coronal CT image in this 53-year-old woman demonstrates an edematous duodenum (*black arrow*) and retroperitoneal APFC (*short white arrow*). Perihepatic ascites (*long white arrows*) and bilateral pleural effusions (*white arrowheads*) also are noted.

Acute necrotic collection

In the acute phase of necrotizing pancreatitis, usually within the first 4 weeks, the acute collection is described as ANC. All 3 types of necrotizing pancreatitis can be associated with ANC in the acute phase. The ANC represents a combination of parenchymal and fat necrosis mixed with exudates from the pancreas. The release of activated pancreatic enzymes into the peripancreatic and pancreatic areas leads to saponification of the fat and necrosis.

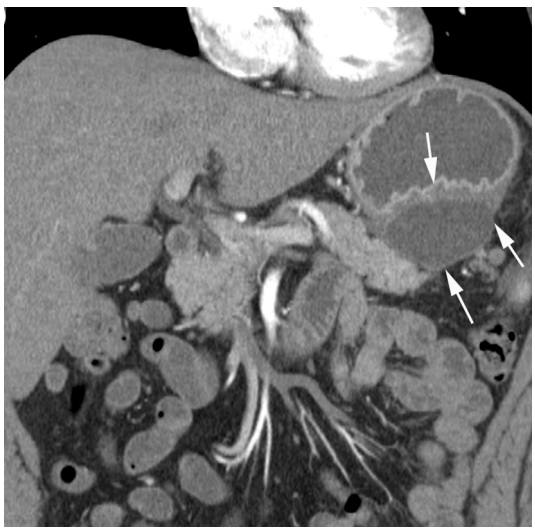

Fig. 10. Pancreatitis with pseudocyst. This coronal CT image demonstrates a well-defined fluid collection with an enhancing capsule (*arrows*) that represents a pseudocyst near the tail of the pancreas. The stomach is slightly elevated by the pseudocyst, and the gastric folds are mildly thickened (*downward arrow*).

Therefore, the collections contain necrotic material and pancreatic fluid to various degrees and within them septa are present that create loculations. Initially, the ANC may appear homogeneous but generally changes to heterogeneous due to the necrotizing process. Please see **Table 4** for the defining features (see **Figs. 6–8**; **Fig. 12**). The term ANC is new in the revised classification. CECT may miss the necrotic components and if a necrotizing process is clinically suspected, MR or ultrasound can be used to confirm the presence or absence of such necrotic material if considered relevant for treatment. Interventional treatment is based on the clinical picture (see **Table 5**). ANCs resolve spontaneously in 20% of patients, become superinfected in about 20%, and develop into sterile WONs (see later discussion) in approximately 60%.[42,43]

Walled-off necrosis

In the subacute phase of necrotizing pancreatitis, the ANC evolves over time into a WON. A thickened, nonepithelialized wall that surrounds the collection containing fluid and nonliquid material defines the interface between the viable fat and the necrosis. It represents the mature stage of an ANC and was known previously under the names necroma, organized pancreatic necrosis, and pseudocyst with necrosis. The term WON is new in the revised classification. It can involve the pancreas alone, the peripancreatic areas alone, or most commonly both, similar to ANC. Please see **Table 4** for a full description of the radiographic features (**Fig. 13**).

Because pseudocysts and WONs can have a similar appearance, they are often confused with each other. Unfortunately, the term pseudocyst often is given to any fluid-containing structure in or near the pancreas, including in some cases cystic pancreatic neoplasms, representing a major management problem. Pseudocysts occur only in patients with interstitial edematous pancreatitis (an exception is discussed later) and are always extrapancreatic. Pseudocysts are also more likely accompanied by a dilated pancreatic duct due to parenchymal compression, whereas with a WON, the pancreas fluid drains into the collection directly without ductal dilation. Pseudocysts do not need drainage unless they are symptomatic and, if needed, it can be a simple percutaneous drainage procedure or a cystogastrostomy placed endoscopically (**Fig. 14**). In WONs, necrotic material often needs to be removed percutaneously or endoscopically, which frequently is followed by surgical debridement (see **Table 5**).

Postnecrosectomy pseudocyst

Following necrosectomy for necrotizing pancreatitis, a pseudocyst can develop within the

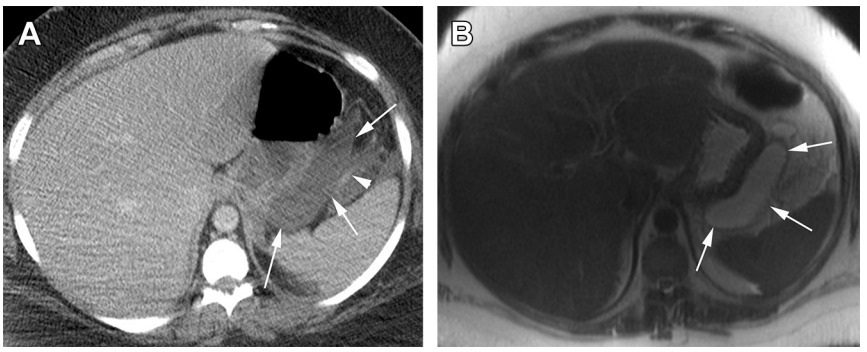

Fig. 11. Early pseudocyst formation in a patient with interstitial edematous pancreatitis. The 35-year-old woman with heavy EtOH abuse had transient organ failure and was not feeling well clinically. (*A*) The axial CT image demonstrates a fluid collection next to the stomach (*arrows*) that shows early partial rim enhancement indicative of early formation of a wall. The presence of nonliquefied material cannot be excluded. The arrowhead indicates the tip of the pancreatic tail. (*B*) Same patient as **Fig. 11**A. MR was obtained to assess if she had developed extrapancreatic necrosis. The axial T2-weighted single-shot fast spin echo MR image obtained a few days later depicts a homogeneous fluid collection of high signal intensity (*arrows*) with a thin early capsule but without evidence of nonliquefied material.

pancreas or in the peripancreatic tissue. It occurs in the so-called disconnected duct syndrome.[41] In these patients, necrosis develops in the body or neck of the pancreas while the tail remains viable. After removal of the necrotic debris via a necrosectomy, the viable pancreatic tail secretes pancreatic juice into the cavity created by the necrosectomy. Please see **Table 4** for complete description. A history of prior necrotizing pancreatitis with necrosectomy needs to be elicited for a correct diagnosis. This collection is called a post-necrosectomy pseudocyst[44] and represents a long-term follow-up complication. A pseudocyst rarely develops after complete liquefaction of a WON or complete reabsorption of necrotic material.

Superinfection of fluid collections

All 5 types of pancreatic fluid collection can become superinfected, but overall infection of APFCs and pseudocysts is rare. Distinction between sterile and infected fluid collections is very

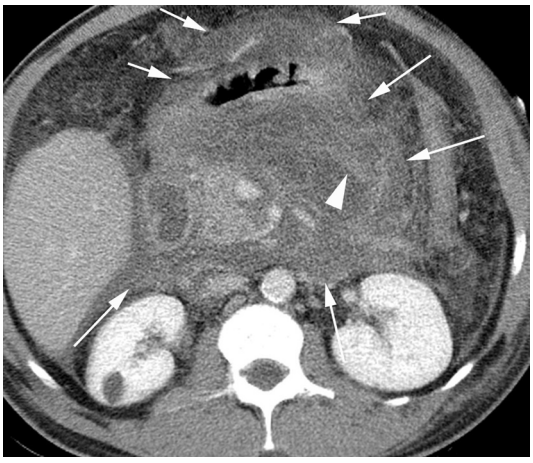

Fig. 12. ANC in necrotizing pancreatitis. The axial image in this 54-year-old woman shows a heterogeneous fluid collection (*long arrows*) without an enhancing capsule and high densities within (*white arrowhead*). The stomach wall is markedly thickened (*short white arrows*) from contiguous inflammation.

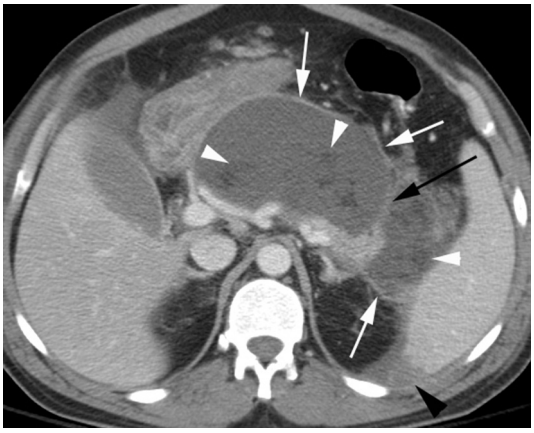

Fig. 13. WON in necrotizing pancreatitis. The axial CT image in this 27-year-old man demonstrates a large fluid collection with an enhancing capsule (*white arrows*) that involves the pancreatic parenchyma and extrapancreatic space. The collection contains areas of necrotic fat (*white arrowheads*) and septations (*black arrow*) and represents the combined form of necrosis after 6 weeks from onset of symptoms. Also, an extrapancreatic complication is seen in the form of a small splenic infarct (*black arrowhead*).

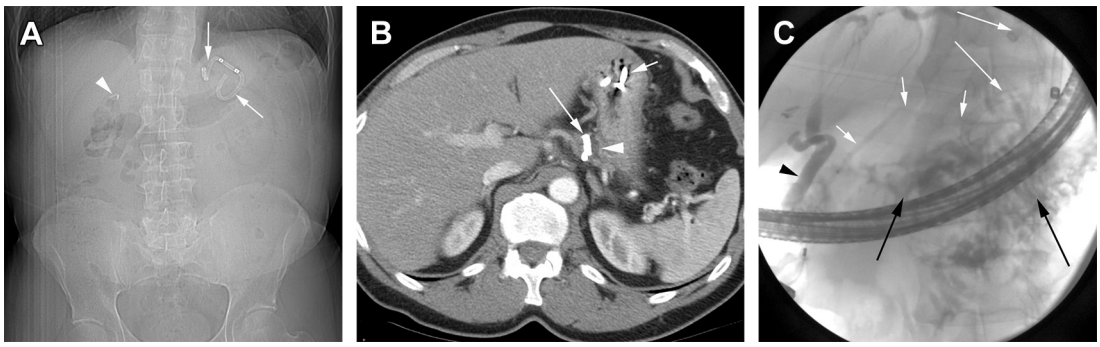

Fig. 14. Cystogastrostomy for a large pseudocyst. (*A*) This 40-year-old man with early satiety and discomfort from a large pseudocyst underwent an endoscopically placed cystogastrostomy. The scout view demonstrates the stent (*arrows*) with the superior coil in the stomach and the inferior coil in the pseudocyst. Surgical cholecystectomy clips (*arrowhead*) also are present. (*B*) Same patient as **Fig. 14**A. The axial CT image reveals the stent (*long arrow*) in the pseudocyst, which shows only a small residual fluid collection (*arrowhead*) following successful drainage. The portion of the stent located in the stomach (*short arrow*) also is seen. (*C*) Endoscopic retrograde cholangiopancreatography (ERCP) in the same patient as **Fig. 14**A. The ERCP demonstrates filling of the pancreatic duct (*short white arrows*) without filling of a fluid collection, but communication with the stomach (*black arrows*) is readily identified demonstrating that the stent (*long white arrows*) is open. The common bile duct (*black arrowhead*) also is filled with contrast.

important because prognosis and management of these patients are very different[45] (see **Table 5**). Infection of pancreatic necrosis occurs in about 20% of patients and usually occurs between the second to fourth week from onset of symptoms of pancreatitis.[46] As previously mentioned, sterile fluid collections such as pseudocysts usually only need drainage when they become symptomatic. Patients with sterile necrosis usually do not require an intervention unless they have persistent pain, anorexia, or vomiting or are unable to resume oral feeding. In patients with suspected sterile pancreatic necrosis, CT is recommended in 7- to 10-day intervals to follow the evolution of these fluid collections (increase in size, presence of air bubbles, or hemorrhage).[47,48] Superinfection of a pancreatic fluid collection can only be diagnosed on CT when air bubbles are present within the fluid collection (**Fig. 15**). Patients who clinically are suspected of having a superinfection but for whom CECT is negative without evidence of air bubbles within the collection (see differential diagnosis of air bubbles under Pitfalls in later discussion) may benefit from a fine-needle aspiration of the necrosis to rule out an infection (**Fig. 16**). Care must be taken to avoid contamination of the aspirate by passing through large or small bowel. Other possibly contaminating routes, such as

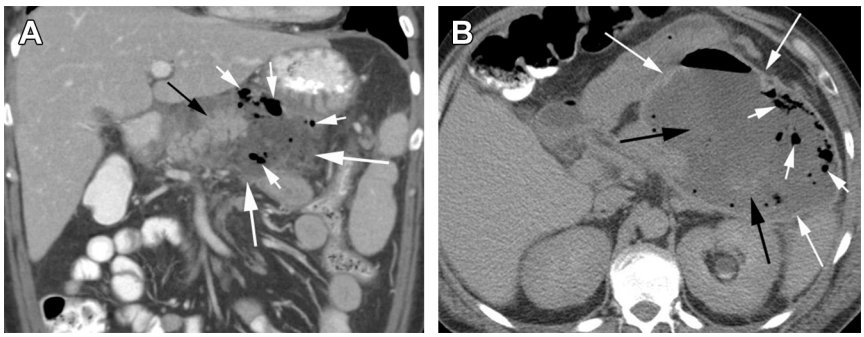

Fig. 15. Necrotizing pancreatitis with infected ANC (*A*) and infected WON (*B*). (*A*) This 20-year-old woman developed fever and sepsis and the CT demonstrates multiple air bubbles (*short white arrows*) in a fluid collection surrounding the edematous pancreas (*black arrow*), which represents the infected ANC. There is no evidence of a capsule but the collection is heterogeneous. (*B*) This 55-year-old man had been in the hospital for 6 weeks when his clinical picture deteriorated and a CT demonstrated multiple air bubbles (*short white arrows*) and an air-fluid level within an encapsulated fluid collection (*long white arrows*) that involved the pancreas and peripancreatic tissues. Also areas of increased attenuation (*black arrows*) are present within the WON.

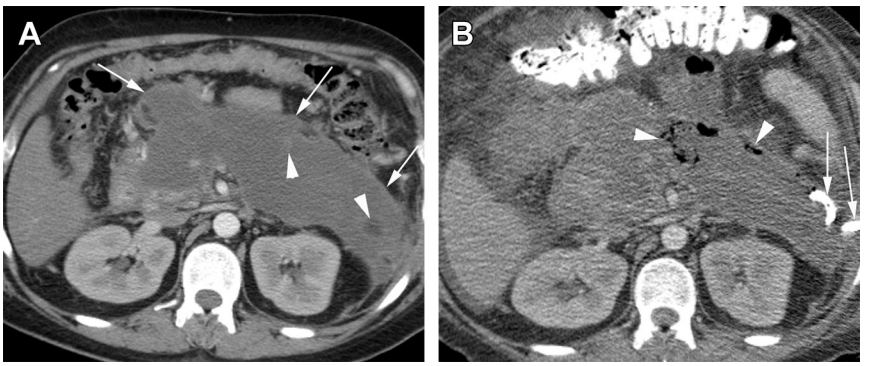

Fig. 16. Necrotizing pancreatitis with infected WON diagnosed with aspiration biopsy. (*A*) This 31-year-old man with a long history of EtOH abuse was diagnosed with necrotizing pancreatitis and became clinically much worse with fever and sepsis during the fifth week from onset. A CT showed a large WON with an enhancing rim (*arrows*) and heterogeneous fluid containing debris and necrotic fat (*arrowheads*) but no air. A CT-guided aspiration was performed that was gram-stain positive. (*B*) Same patient as **Fig. 16**A. Following placement of a percutaneous drainage tube (*long arrows*) by interventional radiology for infected WON, debris and air bubbles (*arrowheads*) are clearly identified. Several exchanges to larger-bore tubes were needed for complete drainage.

transgastric or transduodenal, also should be avoided.[49] A retroperitoneal route via the lateral flank is favored over an anterior approach through the peritoneum. Aspiration of fluid has a false negative rate of less than 10% for diagnosing infection.[45]

A recent study using PET/CT with fludeoxyglucose F 18 –labeled autologous leukocytes found increased tracer uptake in pancreatic fluid collections in 12 of 41 patients, among whom 10 had culture-proven infection with subsequent percutaneous drainage and 2 had unsuccessful aspirations.[50] In the same study, 29 patients with negative PET/CT scans had 25 proven negative cultures, and aspiration was unsuccessful in 4 cases. These results obtained with a noninvasive technique are promising but need to be confirmed in larger series.

Infection of pancreatic necrosis is associated with high morbidity and mortality (25%–70%).[43,51] Suspected superinfected fluid collection containing necrotic debris usually needs to be drained, but this is based on the clinical presentation (eg, presence or absence of clinical instability, such as organ failure, fever, sepsis, or hemorrhage) or the CT demonstration of air bubbles, increase in size, or hemorrhage within the collection. Many patients with infected necrosis can initially be treated with percutaneous catheter drainage as a temporizing measure, with endoscopic or surgical debridement at a later point (step-up approach).[43,51] Some patients may even undergo successful percutaneous drainage after surgical debridement. Several studies have shown that morbidity and mortality are increased with early surgical intervention in these patients.[47,51,52] In some patients, percutaneous catheter drainage alone may be successful. One study suggested that the ultimate outcome may depend more on the presence of multisystem organ failure than on the presence of infection.[53]

DIFFERENTIAL DIAGNOSIS

Usually, the clinical picture combined with laboratory data is strong enough to suggest the correct diagnosis of acute pancreatitis. Clinically, the differential diagnosis of abdominal pain that could be confused with acute pancreatitis includes peptic ulcer disease, intestinal obstruction, abdominal aortic aneurysm, cholangitis or cholecystitis, choledocholithiasis, viral gastroenteritis, mesenteric ischemia, hepatitis, or myocardial infarction (**Box 1**). In all these cases, additional clinical history and the fact that the lipase and amylase are normal should prevent a

Box 1
Clinical differential diagnosis for acute pancreatitis

- Peptic ulcer disease
- Intestinal obstruction
- Abdominal aortic aneurysm
- Cholangitis and/or cholecystitis
- Viral gastroenteritis
- Mesenteric ischemia
- Hepatitis
- Myocardial infarction

misdiagnosis. In these cases, CECT should demonstrate a normal pancreas except in rare instances discussed under the section entitled Pitfalls. The amylase may be elevated in patients with acute abdominal pain and perforated viscus (**Fig. 17**) or mesenteric ischemia, but the amylase level will be less than that seen in acute pancreatitis. Mesenteric ischemia also shows elevation of lactic acid.

Occasionally, when a history of previous pancreatitis cannot be elicited, CT may misdiagnose a cystic neoplasm. In these cases, MR can help demonstrate necrotic debris with or without loculations to confirm that the cystic structure is related to previous acute pancreatitis (**Box 2**, **Fig. 18**).

PITFALLS

Mild acute pancreatitis can be missed on CECT if the patient is scanned on the first day of onset of symptoms because the findings of edema within the pancreas or stranding in the peripancreatic area are not yet evident. Also, necrosis may be missed or misdiagnosed when a patient suffering from acute pancreatitis is scanned with CECT in the first 72 hours because necrosis can be confused with diminished perfusion due to edema (see **Box 2**, **Fig. 5**).[15,54]

Box 2
Imaging pitfalls for acute pancreatitis

Early phase:

- FN for pancreatitis: if imaged within first 24 hours
- FP: confusing pancreatic edema with necrosis

Late phase:

- FP for superinfection:
  - Marsupialization
  - Spontaneous fistula to gastrointestinal tract
  - Previous intervention
- Misdiagnosis of ANC as APFC in first 4 weeks
- Misdiagnosis of WON as pseudocyst after ~4 weeks
- Misdiagnosis of WON as cystic neoplasm

Mimickers:

- Perforated gastric or duodenal ulcer
- Severe peptic ulcer disease
- Mesenteric ischemia
- Overhydration
- Hypoalbuminemia

Abbreviations: FN, false negative; FP, false positive.

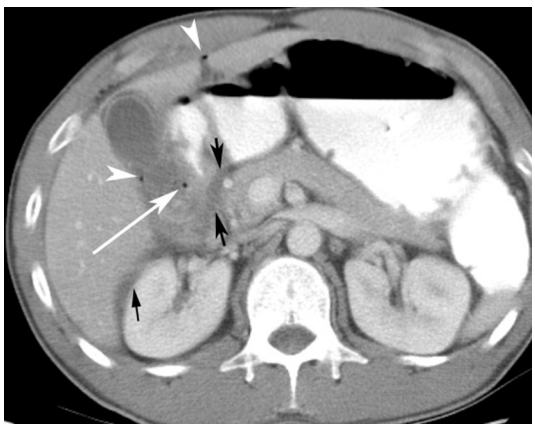

Fig. 17. Perforated duodenal ulcer mimicking acute pancreatitis. This 52-year-old woman came to the Emergency Room with acute onset of severe abdominal pain. The amylase was mildly elevated prompting the clinical diagnosis of acute pancreatitis. The axial CT image demonstrates fluid between the pancreatic head and the duodenum and stranding in the anterior pararenal space (*black arrows*), but the duodenal wall is thickened with free air next to it (*long white arrow*), in the gallbladder fossa, and in the falciform ligament areas (*white arrowheads*), indicative of a perforated duodenal ulcer. Surgery confirmed the perforated ulcer and absence of pancreatitis.

Resuscitation efforts with excessive rehydration can led to generalized edema, ascites, and edema in the pancreas that are associated with stranding and fluid in the retroperitoneum, which mimics acute pancreatitis. The severity of the pancreatic manifestations depends on the degree of hydration (**Fig. 19**). Similarly, hypoalbuminemia with ascites and generalized edema can mimic acute pancreatitis (**Fig. 20**).

Infection can be suggested on CECT if gas bubbles are present within the pancreatic collection due to the presence of gas-forming organisms (see **Figs. 15** and **16**).[55] Pitfalls for erroneously diagnosing a pseudocyst or necrosis as infected are caused by spontaneous drainage of the collection into the gastrointestinal tract. To avoid this diagnostic pitfall, the adjacent gastrointestinal walls need to be carefully analyzed. Marsupialization or other drainage procedures also can lead to the introduction of gas into a collection and erroneous interpretations (**Fig. 21**).

A perforated gastric or duodenal ulcer may be confused on CECT with pancreatitis because of stranding in the peripancreatic and duodenal

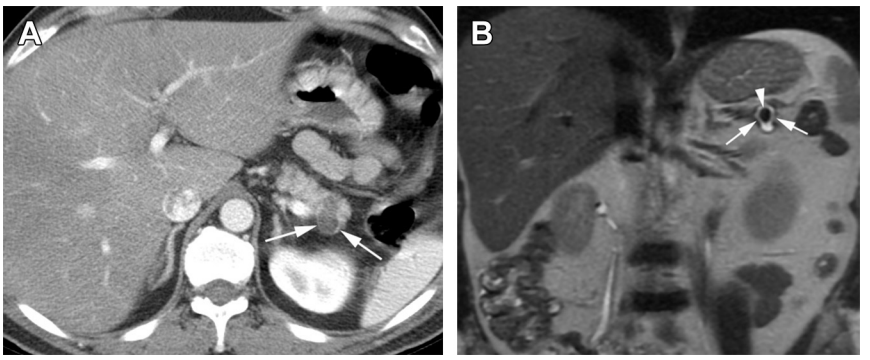

Fig. 18. Residual small WON after necrotizing pancreatitis confused with cystic neoplasm on CT. (*A*) This 77-year-old man presented with right lower quadrant pain, and a CT was performed to assess for acute appendicitis. CT confirmed acute appendicitis (not shown) but also noted a cystic mass (*arrows*) arising from the tail of the pancreas. The patient denied ever having had acute pancreatitis. A possible cystic neoplasm was diagnosed based on an oval-shaped structure of fluid density. For further more definitive evaluation, an MR was recommended. (*B*) Same patient as **Fig. 18**A. The MR imaging shows a well-encapsulated structure of high signal intensity (*arrows*) that contains an area of low signal intensity (*arrowhead*), which could represent hemosiderin or debris. A diagnosis of WON was made, and no intervention was recommended after the chart from a remote admission to an outside hospital confirmed previous necrotizing pancreatitis without intervention.

areas but usually detection of even small pockets of free intraperitoneal air prevents such an error (see **Fig. 17**). Clinically, elevated amylase in these patients adds to the diagnostic dilemma clinically, but the elevation is usually less marked than in acute pancreatitis.

Similarly, severe peptic ulcer disease caused by *Helicobacter pylori*, nonsteroidal anti-inflammatory drug use, or Zollinger-Ellison syndrome can produce enough stranding near the pancreatic bed that it could be confused with acute pancreatitis on CECT. Usually the gastric wall is thickened, the lipase and amylase are normal, and the epigastric pain does not generally radiate into the back, which should enable a correct diagnosis.

Mesenteric ischemia can produce an elevation of the amylase with a normal lipase. A history of atrial fibrillation or peripheral vascular disease in an elderly patient usually raises the index of suspicion for bowel ischemia, and CECT can be diagnostic without confusion with acute pancreatitis unless there is extensive mesenteric stranding in the upper abdomen.

REFERRING CLINICIAN: POINTS OF KNOWLEDGE

- The old Atlanta classification of acute pancreatitis from 1992 is no longer valid.
- Previous descriptions of pancreatic and peripancreatic fluid collection were confusing

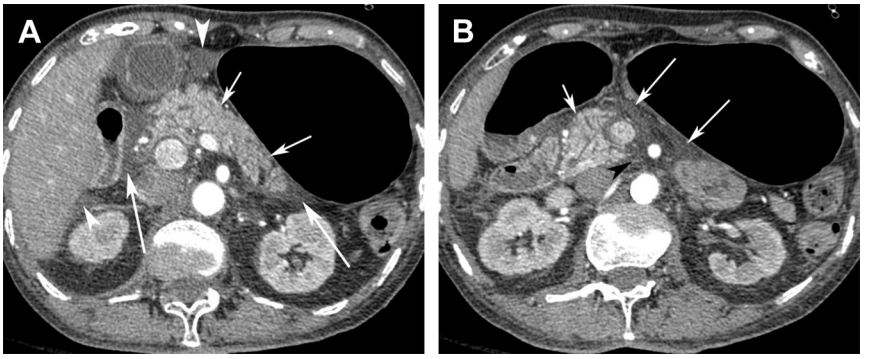

Fig. 19. Edematous pancreas after overhydration. (*A*) The pancreas is swollen and edematous (*short arrows*) with fluid and stranding in the peripancreatic area and between duodenum and head of pancreas (*long arrows*) mimicking acute pancreatitis. Ascites (*arrowheads*) is seen in Morrison pouch and surrounding the thick-walled gallbladder. (*B*) Same patient as **Fig. 19**A. Edema is shown in the head of pancreas (*short white arrow*) on this axial CT image with stranding and fluid in the lesser sac (*long white arrows*) and surrounding the superior mesenteric artery (*black arrowhead*), typically seen in excessive hydration.

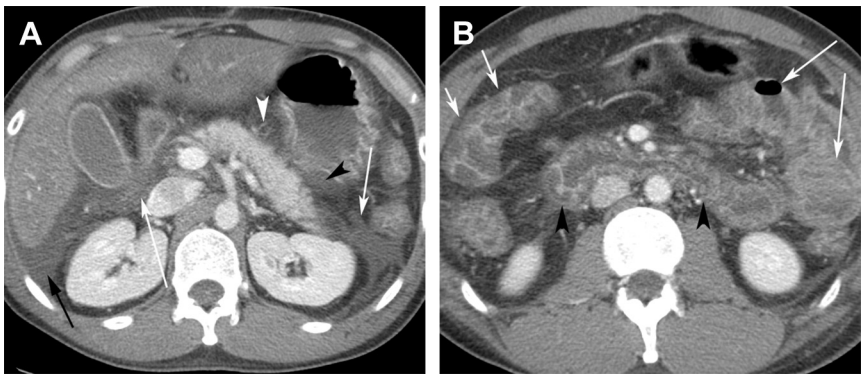

Fig. 20. Hypoalbuminemia in a 45-year-old woman with cirrhosis and diffuse edema. (*A*) The pancreas is mildly edematous with stranding and fluid in the peripancreatic space (*arrowheads*) and anterior pararenal spaces bilaterally (*long white arrows*). Ascites (*black arrow*) also is noted. (*B*) Same patient as **Fig. 20**A. Marked wall thickening is noted in the duodenum (*black arrowheads*), small bowel (*long white arrows*), and hepatic flexure (*short white arrows*) due to the low albumin.

and not used worldwide in a consistent manner.

- The revised Atlanta classification divides acute pancreatitis into interstitial edematous pancreatitis and necrotizing pancreatitis, outlines the clinical definition, defines early and late phases, assesses degrees of severity of pancreatitis, and depicts the definition of organ failure and its significance for outcome.
- Most importantly, the pancreatic fluid collections are now clearly defined based on CECT.
- Based on presence or absence of necrosis, 5 categories are described: APFC and ANC in the first 4 weeks from onset; pseudocyst and WON after 4 weeks; and postnecrosectomy pseudocyst as a long-term complication after necrosectomy.
- The terms pancreatic abscess, phlegmon, or simply fluid collection are no longer used.
- In the first week after onset, only clinical parameters are relevant for treatment, whereas after the first week, morphologic criteria defined by CT are combined with clinical parameters to determine care.
- The revised Atlanta classification system provides a universally understandable and

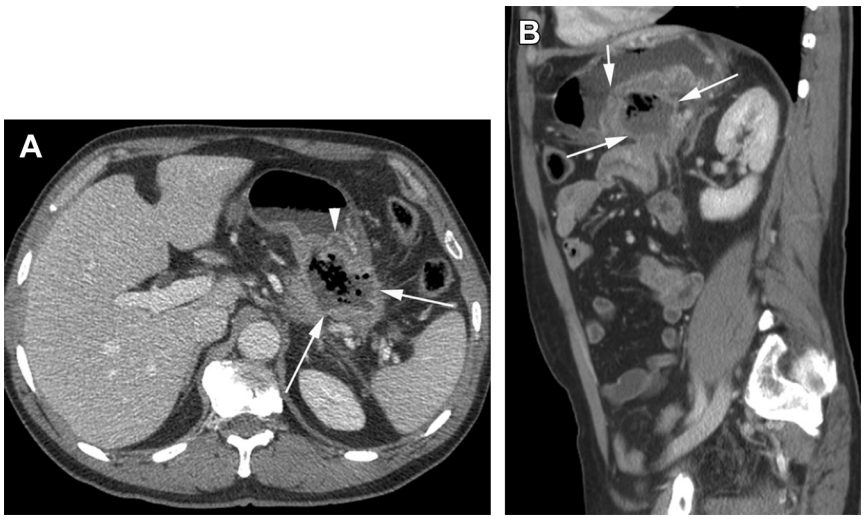

Fig. 21. Marsupialization of a pseudocyst. (*A*) The axial CT image demonstrates a fluid-filled structure with air bubbles and an enhancing rim (*white long arrows*) inseparable from the thickened gastric wall (*white arrowhead*). (*B*) Same patient as **Fig. 21**A. The sagittal CT image shows the same air-filled structure (*long white arrows*) with a communication to the lumen of the stomach (*short white arrow*) due to a marsupialization. This could be confused with infected pseudocyst or WON.

consistent categorization of acute pancreatitis and its complications that facilitates more objective communication between physicians and among institutions, which allows better treatment planning.

SUMMARY

Based on improved knowledge of the disease pathophysiology, superior diagnostic imaging, and expanded treatment options that include minimally invasive procedures, the revised Atlanta classification system provides a common language for clinicians, surgeons, and radiologists in diagnosing and staging acute pancreatitis. CT is declared the method of choice for imaging the pancreas with MR imaging or ultrasound used for clarification of the presence or absence of nonliquefied components within a fluid collection, for detection of gallstones not seen on CT, or when CT is contraindicated. The clinical presentation of acute pancreatitis is clearly defined and a precise description of pancreatic fluid collections is introduced. In the first week, only clinical parameters are used for treatment planning, whereas after the first week, morphologic criteria defined by CT combine with clinical parameters to determine care. Based on the presence or absence of necrosis, a new terminology based on CECT was introduced: ANC are distinguished from APFCs in the first 4 weeks after onset of acute pancreatitis, and WONs are distinguished from pseudocysts after the initial 4 weeks. The term postnecrosectomy pseudocyst in patients with disconnected pancreatic duct syndrome also was presented. These distinctions are important, because management of these patients is different based on the clinical and imaging findings.

REFERENCES

1. Bhatia M, Brady M, Shokuhi S, et al. Inflammatory mediators in acute pancreatitis. J Pathol 2000;190:117–25.
2. Spanier BW, Dijkgraaf MG, Bruno MJ. Epidemiology, aetiology and outcome of acute and chronic pancreatitis: an update. Best Pract Res Clin Gastroenterol 2008;22:45–62.
3. Frossard J, Steer ML, Pastor CM. Acute pancreatitis. Lancet 2008;371:143–52.
4. Toouli J, Brooke-Smith M, Bassi C, et al. Guidelines for management of acute pancreatitis. J Gastroenterol Hepatol 2002;17:S15–39.
5. 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;128:586–90.
6. Banks PA, Bollen TL, Dervenis C, et al. Classification of acute pancreatitis – 2012: revision of the Atlanta classification and definitions by international consensus. Gut 2013;62:102–11.
7. Yuan Y, Huang ZX, Li ZL, et al. Dual-source dual-energy computed tomography imaging of acute necrotizing pancreatitis–preliminary study. Sichuan Da Xue Xue Bao Yi Xue Ban 2011;42:691–4 [in Chinese].
8. Sing VK, Bollen TL, Wu BU, et al. An assessment of the severity of interstitial pancreatitis. Clin Gastroenterol Hepatol 2011;9:1098–103.
9. Thoeni RF. The revised Atlanta classification of acute pancreatitis: its importance for the radiologist and its effect on treatment. Radiology 2012;262:751–64.
10. Spanier BW, Nio Y, van der Hulst RW, et al. Practice and yield of early CT scan in acute pancreatitis: a Dutch observational multicenter study. Pancreatology 2010;10:222–8.
11. Sakorafas GH, Tsiotos GG, Sarr MG. Extrapancreatic necrotizing pancreatitis with viable pancreas: a previously under-appreciated entity. J Am Coll Surg 1999;188:643–8.
12. van Santvoort HC, Bakker OJ, Bollen TL, et al. A conservative and minimally invasive approach to necrotizing pancreatitis improves outcome. Gastroenterology 2011;141:1254–63.
13. Perez A, Whang EE, Brooks DC, et al. Is severity of necrotizing pancreatitis increased in extended necrosis and infected necrosis? Pancreas 2002;25:229–33.
14. Balthazar EJ, Robinson DL, Megibow AJ, et al. Acute pancreatitis: value of CT in establishing prognosis. Radiology 1990;174:331–6.
15. Balthazar EJ. Acute pancreatitis: assessment of severity with clinical and CT evaluation. Radiology 2002;223:603–13.
16. Acevedo-Piedra NG, Moya-Hoyo N, Rey-Riveiro M, et al. Validation of the determinant-based classification and revision of the Atlanta classification systems for acute pancreatitis. Clin Gastroenterol Hepatol 2014;12:311–6.
17. Bruennler T, Hamer OW, Lang S, et al. Outcome in a large unselected series of patients with acute pancreatitis. Hepatogastroenterology 2009;56(91–92):871–6.
18. 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;104:710–5.
19. Johnson CD, Abu-Hilal M. Persistent organ failure during the first week as a marker of fatal outcome in acute pancreatitis. Gut 2004;53:1340–4.

20. de-Madaria E, Banks PA, Moya-Hoyo N, et al. Early factors associated with fluid sequestration and outcomes of patients with acute pancreatitis. Clin Gastroenterol Hepatol 2014;12:997–1002.
21. Petrov MS, Shanbhag S, Chakraborty M, et al. Organ failure and infection of pancreatic necrosis as determinants of mortality in patients with acute pancreatitis. Gastroenterology 2010;139:813–20.
22. Marshall JC, Cook DJ, Christou NV, et al. Multiple organ dysfunction core: a reliable descriptor of a complex clinical outcome. Crit Care Med 1995;23: 1638–52.
23. Singh VK, Wu BU, Bollen TL, et al. A prospective evaluation of the bedside index for severity in acute pancreatitis score in assessing mortality and intermediate markers of severity in acute pancreatitis. Am J Gastroenterol 2009;104:966–71.
24. Lenhart DK, Balthazar EJ. MDCT of acute mild (nonnecrotizing) pancreatitis: abdominal complications and fate of fluid collections. AJR Am J Roentgenol 2008;190:643–9.
25. Mujica VR, Barkin JS, Go VL. Acute pancreatitis secondary to pancreatic carcinoma. Study Group Participants. Pancreas 2000;21:329–32.
26. Kim YK, Ko SW, Kim CS, et al. Effectiveness of MR imaging for diagnosing the mild forms of acute pancreatitis: comparison with MDCT. J Magn Reson Imaging 2006;24:1342–9.
27. De Waele JJ, Delrue L, Hoste EA, et al. Extrapancreatic inflammation on abdominal computed tomography as an early predictor of disease severity in acute pancreatitis: evaluation of a new scoring system. Pancreas 2007;34:185–90.
28. Spitzer AL, Thoeni RF, Barcia AM, et al. Early nonenhanced abdominal computed tomography can predict mortality in severe acute pancreatitis. J Gastrointest Surg 2005;9:928–33.
29. 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 Am J Roentgenol 2014;202:W349–56.
30. Moon JH, Cho YD, Cha SW, et al. The detection of bile duct stones in suspected biliary pancreatitis: comparison of MRCP, ERCP, and intraductal US. Am J Gastroenterol 2005;100:1051–7.
31. Xiao B, Zhang XM, Tang W, et al. Magnetic resonance imaging for local complications of acute pancreatitis: a pictorial review. World J Gastroenterol 2010;16:2735–42.
32. Hirota M, Kimura Y, Ishiko T, et al. Visualization of the heterogeneous internal structure of so-called "pancreatic necrosis" by magnetic resonance imaging in acute necrotizing pancreatitis. Pancreas 2002; 25:63–7.
33. Islim F, Salik AE, Bayramoglu S, et al. Non-invasive detection of infection in acute pancreatitis and acute necrotic collections with diffusion-weighted magnetic resonance imaging: preliminary findings. Abdom Imaging 2014;39:472–81.
34. Barral M, Taouli B, Guiu B, et al. Diffusion-weighted MR imaging of the pancreas: current status and recommendations. Radiology 2015;274:45–63.
35. Tirkes T, Menias CO, Sandrasegaran K. MR imaging for pancreas. Radiol Clin North Am 2012;50:379–93.
36. Brand M, Götz A, Zeman F, et al. Acute necrotizing pancreatitis: laboratory, clinical, and imaging findings as predictors of patient outcome. AJR Am J Roentgenol 2014;202:1215–31.
37. Isenmann R, Büchler M, Uhl W, et al. Pancreatic necrosis: an early finding in severe pancreatitis. Pancreas 1993;8:358–61.
38. Bollen TL, Singh VK, Maurer R, et al. Comparative evaluation of the modified CT severity index and CT severity index in assessing severity of acute pancreatitis. AJR Am J Roentgenol 2011;197: 386–92.
39. Ashley SW, Perez A, Pierce EA, et al. Necrotizing pancreatitis: contemporary analysis of 99 consecutive cases. Ann Surg 2001;234:572–9.
40. Meyrignac O, Lagarde S, Bournet B, et al. Acute Pancreatitis: Extrapancreatic Necrosis Volume as Early Predictor of Severity. Radiology 2015;276: 119–28.
41. Tann M, Maglinte D, Howard TJ, et al. Disconnected pancreatic duct syndrome: imaging findings and therapeutic implications in 26 surgically corrected patients. J Comput Assist Tomogr 2003;27:577–82.
42. Baron TH, Harewood GC, Morgan DE, et al. Outcome differences after endoscopic drainage of pancreatic necrosis, acute pancreatic pseudocysts and chronic pancreatic pseudocysts. Gastrointest Endosc 2002;56:7–17.
43. Shyu JY, Sainani NI, Sahni VA, et al. Necrotizing pancreatitis: diagnosis, imaging, and intervention. Radiographics 2014;34:1218–39.
44. Bollen TL. Imaging of acute pancreatitis; update of the revised Atlanta classification. Radiol Clin N AM 2012;50:429–45.
45. Büchler MW, Gloor B, Müller CA, et al. Acute necrotizing pancreatitis: treatment strategy according to the status of infection. Ann Surg 2000;232:619–26.
46. Triantopoulou C, Delis S, Dervenis C. Imaging evaluation of post-pancreatitis infection. Infect Disord Drug Targets 2010;10:15–20.
47. Segal D, Mortele KJ, Banks PA, et al. Acute necrotizing pancreatitis: role of CT-guided percutaneous catheter drainage. Abdom Imaging 2007;32:351–61.
48. Mortele KJ, Girshman J, Szejnfeld D, et al. CT-guided percutaneous catheter drainage of acute necrotizing pancreatitis: clinical experience and observations in patients with sterile and infected necrosis. AJR Am J Roentgenol 2009;192:110–6.

49. Linder JD, Geenen JE, Catalano MF. Cyst fluid analysis obtained by EUS-guided FNA in the evaluation of discrete cystic neoplasms of the pancreas: a prospective single-center experience. Gastrointest Endosc 2006;64:697–702.
50. Bhattacharya A, Kochhar R, Sharma S, et al. PET/CT with 18F-FDG-labeled autologous leukocytes for the diagnosis of infected fluid collections in acute pancreatitis. J Nucl Med 2014;55:1267–72.
51. van Santvoort HC, Besselink MG, Bakker OJ, et al. A step-up approach or open necrosectomy for necrotizing pancreatitis. Dutch Pancreatitis Study Group. N Engl J Med 2010;362:1491–502.
52. Schneider L, Büchler MW, Werner J. Acute pancreatitis with an emphasis on infection. Infect Dis Clin North Am 2010;24:921–41.
53. Guo Q, Li A, Xia Q, et al. The role of organ failure and infection in necrotizing pancreatitis: a prospective study. Ann Surg 2014;259:1201–7.
54. Bollen TL, van Santvoort HC, Besselink MG, et al. Update on acute pancreatitis: US, CT, and MRI features. Semin Ultrasound CT MR 2007;28:371–83.
55. Vege SS, Fletcher JG, Talukdar R, et al. Peripancreatic collections in acute pancreatitis: correlation between computerized tomography and operative findings. World J Gastroenterol 2010;16:4291–6.