The New Revised Classification of Acute Pancreatitis 2012

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KEYWORDS

• Classification • Acute pancreatitis • Interstitial edematous pancreatitis • Necrotizing pancreatitis

KEY POINTS

- The aim of this study is to update the original 1991 Atlanta Classification of acute pancreatitis to standardize the reporting of and terminology of the disease and its complications.
- Important features of this classification have incorporated the new insights into the disease learned over the last 20 years, including the recognition that acute pancreatitis and its complications involve a dynamic process involving two phases, early and late.
- The accurate and consistent description of the two types of acute pancreatitis (interstitial edematous pancreatitis and necrotizing pancreatitis), its severity, and, possibly most importantly, the description of local complications based on characteristics of fluid and necrosis involving the peripancreatic collections, will help to improve the stratification and reporting of new methods of care of acute pancreatitis across different practices, geographic areas, and countries.
- By using a common terminology, the advancement of the science of acute pancreatitis should be facilitated.

INTRODUCTION

More than 20 years have passed since the first concerted effort to classify acute pancreatitis by the Atlanta Classification, spearheaded by Edward Bradley in 1991.[1] At the time, this classification was an attempt to define a common terminology and

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define the severity of the disease such that physicians around the world would accept and adopt a uniform classification. Although novel at the time, the classification defined and used several terms that never "caught on," and the actual classification as written by the Atlanta Conference, while referred to by many articles, has not been accepted or used universally.[2] Moreover, in these last 20 years our understanding of the etiopathogenesis of acute pancreatitis, its natural history, the various markers of severity, and, equally important, the features of the disease on state-of-the-art cross-sectional imaging, have led to a plethora of often confusing and imprecisely used terms. Indeed, a common terminology for the disease, its severity, and, possibly most importantly, the pancreatic and peripancreatic "fluid" collections, have yet to be acknowledged and adopted. Because of this confusion, a group of researchers decided to revise the Atlanta Classification using a new technique for a global, Web-based "virtual" consensus conference over the Internet. Although the concept was novel, the idea of a Web-based global consensus, as described in this article, was only partially successful. Nevertheless, using this approach initially, with very helpful and insightful input from numerous pancreatologists of many different disciplines (gastroenterology, surgery, pathology, diagnostic and interventional radiology, gastrointestinal endoscopy, and acute care medicine/surgery) around the world, a new classification was developed and vetted through many different international societies dealing with acute pancreatitis. Using this input, the Working Group (the authors of this article) then collated the evidence-based literature whenever available to construct a new classification, in part based on the two phases of the natural history of the disease (the first week or two, and the next several weeks/months that follow). The product of the past 5 years of work culminated in the Classification of Acute Pancreatitis 2012.[3] This classification addresses diagnosis, types of acute pancreatitis, severity, and definition of pancreatic and peripancreatic collections, which are discussed herein. The authors hope that this classification will unify the terminology to allow global consensus and facilitate comparison of studies published in the literature.

DIAGNOSIS OF ACUTE PANCREATITIS

The diagnosis of this disease is usually straightforward and, as described in many studies, involves a combination of symptoms, physical examination, and focused laboratory values. This classification requires 2 of the following 3 features: (1) central upper abdominal pain usually of acute onset often radiating through to the back; (2) serum amylase or lipase activity greater than 3 times the upper limit of normal; and (3) characteristic features on cross-sectional abdominal imaging consistent with the diagnosis of acute pancreatitis (see later discussion). Note that not every patient requires pancreatic imaging; for instance, for the patient with characteristic abdominal pain and increased serum amylase/lipase activity, a contrast-enhanced computed tomography (CECT) or magnetic resonance imaging (MRI) is usually not required on admission or later (if it is mild acute pancreatitis), provided the clinical picture is that of acute pancreatitis.

DEFINITION OF THE TWO TYPES OF ACUTE PANCREATITIS

There are two basically different forms of acute pancreatitis: interstitial edematous pancreatitis and necrotizing pancreatitis.

Interstitial Edematous Pancreatitis

The majority (80%–90%) of patients presenting with the clinical picture of acute pancreatitis will have this more mild form. The differentiating characteristic of acute

interstitial edematous pancreatitis is the lack of pancreatic parenchymal necrosis or peripancreatic necrosis evident on imaging. The associated findings are usually diffuse (or, on occasion, localized) enlargement of the pancreas secondary to inflammatory edema (**Fig. 1**); there may also be some peripancreatic fluid (see the section on pancreatic and peripancreatic collections). The pancreatic parenchyma and surrounding tissues may have haziness and stranding secondary to inflammatory edema, but there is no necrosis evident on cross-sectional imaging. The clinical picture of this form of acute pancreatitis usually resolves quickly over the first week.

Necrotizing Pancreatitis

The hallmark of this form of acute pancreatitis is the presence of tissue necrosis, either of the pancreatic parenchyma or the peripancreatic tissues. Necrotizing pancreatitis most commonly involves both the pancreatic parenchyma and the peripancreatic tissue (**Fig. 2**) or the peripancreatic tissue alone (**Fig. 3**); rarely, the necrosis is limited only to the pancreatic parenchyma. Therefore, necrotizing pancreatitis is classified as pancreatic parenchymal necrosis alone, pancreatic parenchymal and peripancreatic necrosis, or peripancreatic necrosis alone. Involvement of the pancreatic parenchyma usually heralds a disease more severe than peripancreatic necrosis alone.[4,5]

Early in the illness (during the first week), the differentiation of "necrosis" can be difficult on CECT. For the pancreatic parenchyma, nonperfusion of the pancreatic gland is usually evident. For the peripancreatic region, obvious loss of "perfusion" of the retroperitoneal fat is not evident (this area has little radiographic "perfusion" even normally), and the diagnosis of necrosis is usually made based on the presence of local inflammatory changes and some element of associated fluid, but also a solid component (see later discussion). Recognition of this peripancreatic necrosis is difficult during the first week of the disease, but thereafter the diagnosis on imaging becomes more apparent, with a more heterogeneous collection of both solid and liquid components.

Infected Versus Sterile Necrosis

Necrotizing pancreatitis should also be labeled either infected or sterile. Infection is rare during the first week.[6,7] Infection can be diagnosed based on ongoing signs of sepsis

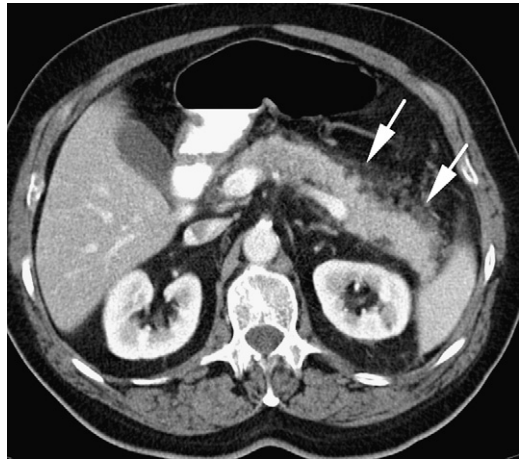

Fig. 1. A 48-year-old man with acute interstitial edematous pancreatitis. There is peripancreatic fat stranding (*arrows*); the pancreas enhances completely.

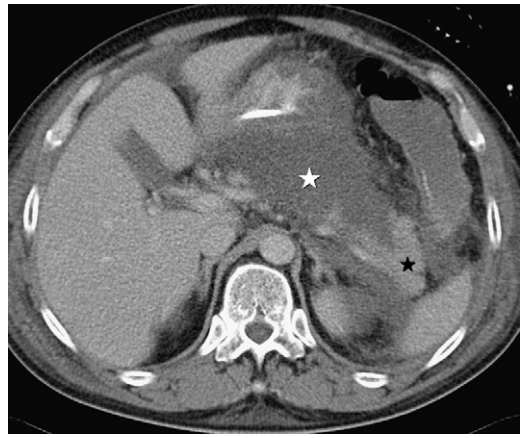

Fig. 2. A 39-year-old woman with acute necrotizing pancreatitis. There is extensive nonenhancement representing parenchymal necrosis (*white star*) of the body of the pancreas. Part of the pancreatic tail shows normal enhancement (*black star*).

and/or the combination of clinical signs and the computed tomographic imaging when extraluminal gas is present within areas of necrosis in the pancreatic and/or peripancreatic tissues (**Fig. 4**). Similarly, the diagnosis of infected necrosis can be made based on percutaneous, image-guided fine-needle aspiration when bacteria and/or fungi are seen on Gram stain and the culture is positive. Lack of positive Gram stain or culture positivity should be interpreted with some caution. The presence of suppuration (numerous polymorphonuclear cells) is somewhat variable; the longer the duration of the infection, the more suppuration. Infection may also be diagnosed as a secondary event after instrumentation of whatever form (percutaneous, endoscopic, operative); secondary infection is associated with increased mortality and morbidity.[8]

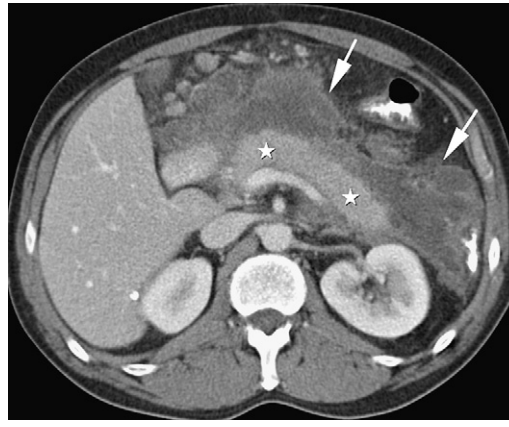

Fig. 3. Acute necrotic collections (ANC) in a 42-year-old man with acute necrotizing pancreatitis involving only the peripancreatic tissues. Note normal enhancement of the entire pancreatic parenchyma (*white stars*) and the heterogeneous, nonliquid peripancreatic components in the retroperitoneum and mesentery of the transverse mesocolon (*white arrows* pointing at the borders of the ANC).

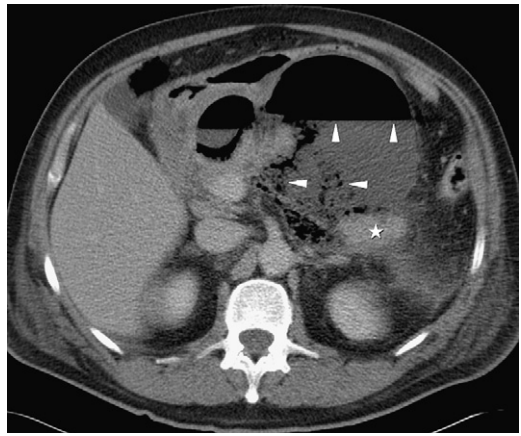

Fig. 4. A 45-year-old man with acute necrotizing pancreatitis complicated by infected pancreatic necrosis. The pancreatic tail (*white star*) enhances normally. There is a large heterogeneous ANC in the pancreatic and peripancreatic area with presence of impacted gas bubbles (*horizontal white arrowheads*) and gas-fluid level (*vertical white arrowheads*), usually a sign of infection of the necrosis.

SEVERITY OF THE DISEASE

Classifying the severity of the disease is important when comparing different institutional experiences, talking with patients about prognosis, planning therapy, and comparing new methods of management.

This classification of severity of acute pancreatitis defines 3 degrees of severity: mild acute pancreatitis, moderately severe acute pancreatitis, and severe acute pancreatitis. These levels of severity are based on the presence and/or absence of persistent organ failure and local and systemic complications (see later discussion). In general, mild acute pancreatitis resolves within several days to a week, moderately severe acute pancreatitis resolves slowly and may require interventions, and severe acute pancreatitis, in addition to longer hospital stay and interventions, is also associated with organ failure and death.

Definition of Organ Failure (Persistent or Transient)

Persistent organ failure for at least 48 hours has proved to be the most reliable marker for disease severity in acute pancreatitis.[9,10] Organ failure has been scored by many different systems, and numerous serum markers have also been evaluated. After careful review of the literature as well as consideration of the pathogenesis and the course of acute pancreatitis, the authors chose the modified Marshall scoring system.[11] This scoring system is easy and universally applicable because it does not require any sophisticated assays or monitoring and, most importantly, stratifies disease severity objectively and easily.[12] This scoring system targets the 3 organ systems most commonly affected by the systemic inflammatory response syndrome (SIRS) that accompanies severe acute pancreatitis: respiratory, cardiovascular, and renal (**Table 1**). Persistent organ failure is defined as a score of 2 or more for more than 48 hours for 1 (or more) of the 3 organ systems using the modified Marshall scoring system. By contrast, transient organ failure is a score of 2 or more, but for less than 48 hours. This scoring system is preferred over the Sepsis-related Organ Failure Assessment (SOFA) system,[13] which is used for patients in a critical care unit and also takes into

Table 1
Modified Marshall scoring system

	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, μmol/L	≤134	134–169	170–310	311–439	>439
Serum creatinine, mg/dL	<1.4	1.4–1.8	1.9–3.6	3.6–4.9	>4.9
Cardiovascular (systolic blood pressure, mm Hg)[b]	>90	<90 Fluid responsive	<90 Not fluid responsive	<90, pH <7.3	<90, pH <7.2

A score of 2 or more in any system defines the presence of organ failure.

[a] Scoring patients with preexistent chronic renal failure depends on the extent of deterioration over baseline renal function. Calculations for a baseline serum creatinine ≥134 μmol/L or ≥1.4 mg/dL are not available.

[b] Off inotropic support.

Modified 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;102–11.

consideration other criteria. The modified Marshall scoring system can be used repeatedly, both early and late in the course of the disease, to classify severity.

Definition of Local Complications

Unlike in the prior 1991 Atlanta Classification, the natural history, clinical consequences, and, most importantly, the definition of pancreatic and peripancreatic collections are now better understood. Local complications in the current 2012 classification include acute peripancreatic fluid collections, pancreatic pseudocysts, acute necrotic collections, and walled-off necrosis (see later discussion). Other local complications include splenic/portal vein thrombosis, colonic necrosis, retroperitoneal hemorrhage, and gastric outlet dysfunction. One might think of the local complications as those that delay hospital discharge or require intervention but do not necessarily cause death. Of course, one would expect the presence of a local complication by persistence of abdominal pain, secondary increases in serum amylase/lipase activity, organ failure, fever/chills, and so forth. Such symptoms usually prompt a cross-sectional imaging procedure to search for these complications.

Definition of Systemic Complications

These systemic complications involve de novo occurrence of renal, circulatory, or respiratory organ failure or exacerbation of serious preexisting comorbidities related directly to the acute pancreatitis. Examples of these comorbidities include coronary artery disease, congestive heart failure, chronic obstructive lung disease, diabetes, and chronic liver disease. Note that organ failure as defined by the modified Marshall score is not considered as part of these systemic complications, and a distinction is made between persistent organ failure (a sign of severe acute pancreatitis; see later discussion) and systemic complications. These complications result from the systemic inflammatory response to acute pancreatitis, and may be further exacerbated by the need for fluid resuscitation.

Phases of Acute Pancreatitis

In general there are two phases of this dynamic disease of acute pancreatitis, which overlap one another: the early phase, which usually lasts only 1 week or so, and the late phase, which can persist for weeks to months.

During the early phase, most of the systemic manifestations of the disease are a consequence of the host response to the pancreatic injury. This early phase is secondary to the cytokine cascade, which manifests as SIRS,[14] and/or the compensatory anti-inflammatory syndrome (CARS), which can predispose to infection.[15] When SIRS or CARS persist, organ failure becomes much more likely. The determinant of the severity of the acute pancreatitis is primarily the presence and duration of organ failure: transient organ failure (<48 hours' duration) and persistent organ failure (>48 hours' duration). If the organ failure involves more than 1 organ, the terms multiple organ failure or multiple organ dysfunction syndrome are appropriate.

The late phase of acute pancreatitis is characterized by the persistence of systemic signs of ongoing inflammation, by the presence of local and systemic complications, and/or by transient or persistent organ failure. By definition, the late phase occurs only in patients with moderately severe or severe acute pancreatitis.

Definition of Severity of Acute Pancreatitis

The need to define severity is important for several reasons. It is important to identify patients on admission or during the first 24 to 48 hours who will require aggressive resuscitation/treatment, either so they are monitored closely in an intensive care unit or so they can be transferred to a high-acuity care hospital. The definition of severity will not be able to be made definitively in the first 48 hours; therefore, patients with SIRS should be treated as if they have severe acute pancreatitis. Second, such stratification allows various practices around the world to compare treatments and experiences in a more objective scoring/classification system.

This classification defines 3 degrees of severity: mild, moderately severe, and severe acute pancreatitis (**Box 1**). These degrees of severity separate patients well into 3 groups according to the morbidity and mortality of the disease.

Mild Acute Pancreatitis

Mild acute pancreatitis is defined as acute pancreatitis without organ failure or local or systemic complications. These patients resolve their symptoms rapidly and are

Box 1
Degrees of severity

Mild Acute Pancreatitis

- No organ failure
- Lack of local or systemic complications

Moderately Severe Acute Pancreatitis

- Organ failure that resolves within 48 hours (transient organ failure) and/or
- Local or systemic complications (sterile or infected) without persistent organ failure

Severe Acute Pancreatitis

- Persistent single or multiple organ failure (>48 hours)

Modified 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;62:102–11.

discharged usually within the first week. Mortality is rare, and pancreatic imaging is often not required.

Moderately Severe Acute Pancreatitis

Moderately severe acute pancreatitis is defined as acute pancreatitis with transient organ failure, local complications, and/or systemic complications, but not associated with persistent (>48 hours) organ failure. The morbidity (longer stay and need for intervention) is increased; mortality is also increased somewhat (<8%) compared with that of mild acute pancreatitis, but not to the extent seen in severe acute pancreatitis. Depending on the complications of the acute pancreatitis, patients may be discharged within the second or third week or may require prolonged hospitalization because of the local or systemic complications. Noteworthy, however, is that the mortality is considerably less than that of severe acute pancreatitis.[16]

Severe Acute Pancreatitis

Severe acute pancreatitis is defined as acute pancreatitis complicated by persistent organ failure, whether the organ failure occurs in the early or late phase of the disease. Patients with severe acute pancreatitis also usually have one or more local and/or systemic complications. Of note, patients with severe acute pancreatitis that develops within the early phase (first week) are at a 36% to 50% risk of death.[9,10,17] Development of infected necrosis later in the course of the disease in patients with severe acute pancreatitis also has an extremely high mortality.[8,18]

Other groups have suggested a 2-tier or 4-tier classification of severity,[8,19,20] singling out infected necrosis as a marker of extreme severity. This system, unfortunately, overlooks a very high-risk subset of patients who have persistent organ failure within the first few days of the disease but lack any infection.

DEFINITION OF PANCREATIC AND PERIPANCREATIC COLLECTIONS

One of the biggest problems with the literature and the dialogue of acute pancreatitis is the multitude of terms used to describe the pancreatic and peripancreatic areas on cross-sectional imaging. In this classification, a crucial and important distinction is emphasized between "collections" consisting of fluid alone versus those "collections" that arise from necrosis of pancreatic parenchyma and/or peripancreatic tissues that consist of a solid component and as the necrotic process evolves, varying the degrees of fluid (**Box 2**).

Acute Peripancreatic Fluid Collection

This type of fluid collection develops in the early phase of interstitial edematous acute pancreatitis. On CECT, acute peripancreatic fluid collections (APFCs) lack a well-defined wall and are confined by the normal fascial planes in the retroperitoneum (**Fig. 5**). These fluid collections are not associated with necrotizing pancreatitis, remain sterile, and usually resolve without intervention.[21,22] When an APFC persists past 4 weeks, it is likely to develop into a pancreatic pseudocyst (see later discussion), although such development of a pseudocyst is a rare event in acute pancreatitis.

Pancreatic Pseudocyst

This term refers very specifically to a peripancreatic or, less commonly, intrapancreatic fluid collection or collections surrounded by a well-defined wall, and contains essentially no solid material (**Fig. 6**). The term pancreatic pseudocyst is very specific, and has been misused repeatedly throughout the literature and in daily dialogue. The

Box 2
Revised definitions used in this new classification

Interstitial edematous pancreatitis: Inflammation of pancreatic parenchyma and peripancreatic tissue, but without obvious tissue necrosis.

CECT Criteria

- Enhancement of the pancreatic parenchyma by contrast agent
- No evidence of peripancreatic necrosis (see below)

Necrotizing pancreatitis: Inflammation with pancreatic parenchymal necrosis and/or peripancreatic necrosis.

CECT Criteria

- Areas of pancreatic parenchymal lacking by intravenous contrast agent and/or
- Findings of peripancreatic necrosis (see below—ANC and WON)

APFC (acute peripancreatic fluid collection): Peripancreatic fluid with interstitial edematous pancreatitis and no peripancreatic necrosis. This term applies to peripancreatic fluid seen within the first 4 weeks after onset of interstitial edematous pancreatitis.

CECT Criteria

- Homogeneous collection with fluid density adjacent to pancreas confined by normal peripancreatic fascial planes
- No recognizable wall encapsulating the collection
- Occurs only in interstitial edematous pancreatitis

Pancreatic pseudocyst: Encapsulated fluid collection with minimal or no necrosis with a well-defined inflammatory wall usually outside the pancreas. This entity occurs more than 4 weeks after onset of interstitial edematous pancreatitis.

CECT Criteria

- Round or oval well circumscribed, homogeneous fluid collection
- No nonliquid component
- Well-defined wall
- Occurs after interstitial edematous pancreatitis

ANC (acute necrotic collection): A collection of both fluid and necrosis associated with necrotizing pancreatitis involving the pancreatic parenchyma and/or the peripancreatic tissues

CECT Criteria

- Heterogeneous, nonliquid density of varying degrees
- No definable encapsulating wall
- Location: intrapancreatic and/or extrapancreatic
- Occurs in setting of acute necrotizing pancreatitis

WON (walled-off necrosis): A mature, encapsulated collection of pancreatic and/or peripancreatic necrosis with a well-defined inflammatory wall occurring more than 4 weeks after onset of necrotizing pancreatitis.

CECT Criteria

- Heterogeneous liquid and nonliquid density with varying degrees of loculations
- Well-defined encapsulating wall
- Location: intrapancreatic and/or extrapancreatic
- Occurs only in setting of necrotizing pancreatitis

Modified 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;62:102–11.

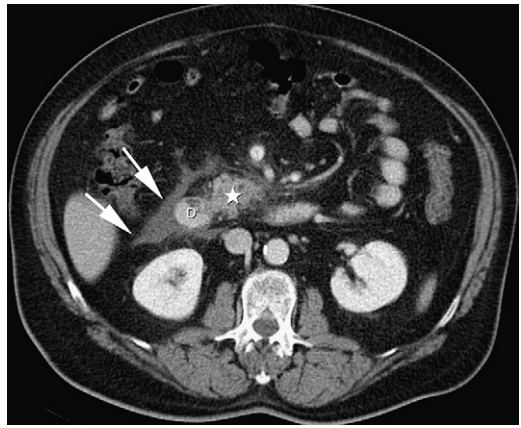

Fig. 5. A 63-year-old man with acute interstitial edematous pancreatitis and acute peripancreatic fluid collection (APFC) in the right anterior pararenal space (*white arrows*). The pancreatic head enhances normally (*white star*). APFC has fluid density without an encapsulating wall. D, descending part of the duodenum.

presumed etiopathogenesis of pancreatic pseudocyst is related to a disruption of the main pancreatic duct or its intrapancreatic branches without any pancreatic or peripancreatic necrosis evident on cross-sectional imaging. It must be stressed that development of a pancreatic pseudocyst is extremely rare in acute pancreatitis. The absence of solid material within a presumed pancreatic pseudocyst may require MRI or ultrasonography to support this diagnosis.

A special situation that can lead to a pancreatic pseudocyst in patients with necrotizing pancreatitis involves the "disconnected duct syndrome."[23] This true fluid

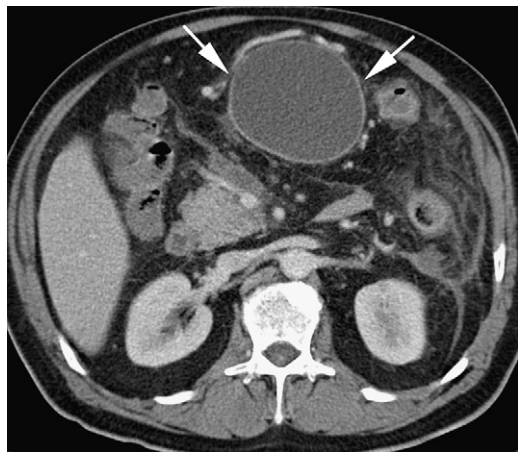

Fig. 6. A 39-year-old man with a pseudocyst 5 weeks after an episode of acute interstitial pancreatitis. Note the round, low-attenuated, homogeneous fluid collection with a well-defined enhancing rim (*white arrows* pointing at the borders of the pseudocyst) in the inferior recess of the lesser sac. There is absence of areas of greater attenuation, indicating nonliquid components.

collection can occur in patients when necrosis of the neck/proximal body of the pancreas isolates a still viable distal pancreatic remnant. A true pancreatic pseudocyst may develop many weeks after operative necrosectomy secondary to localized leakage of the disconnected duct into the necrosectomy cavity.

Acute Necrotic Collection

These collections occur within the first 4 weeks of the disease and contain variable amounts of fluid and solid (necrotic) material related to pancreatic and/or peripancreatic necrosis (see **Fig. 3**; **Fig. 7**). On CECT, acute necrotic collections (ANCs) can closely resemble an APFC in the first few days of the acute pancreatitis, but as the necrosis evolves, both fluid and solid components become evident. MRI or ultrasonography may be useful to image the solid component. An ANC is not an APFC, because it arises in patients with necrotizing pancreatitis. ANCs can be multiple, and may involve the pancreatic parenchyma alone, the peripancreatic tissue alone, or, most commonly, both the pancreatic parenchyma and the peripancreatic tissues. An ANC may be infected or sterile, and may be associated with disruption of the pancreatic ductal system with leakage of pancreatic juice into the collection, but this type of ANC is not a pancreatic pseudocyst, because an ANC contains solid material.

Walled-Off Necrosis

This type of collection consists of varying amounts of liquid and solid material surrounded by a mature, enhancing wall of reactive tissue (**Fig. 8**). A walled-off necrosis (WON) represents the mature encapsulated ANC that develops usually at least 4 weeks after onset of necrotizing acute pancreatitis. Previous terms used intermittently and inconsistently to describe this type of collection include organized pancreatic necrosis, necroma, pancreatic sequestrum, pancreatic pseudocyst with necrosis, and subacute pancreatic necrosis. Use of the term WON gathers all these terms into a common, consistent terminology.

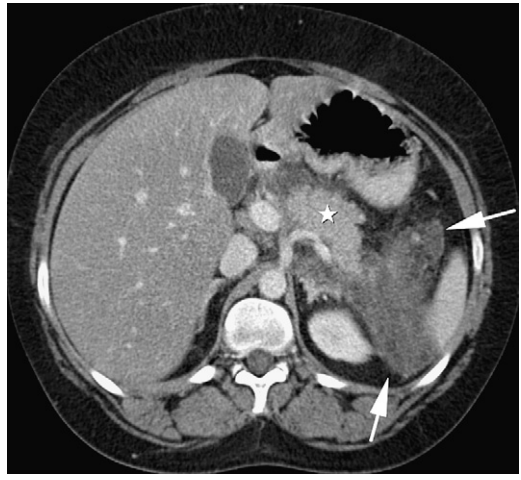

Fig. 7. ANC in a 44-year-old man with acute necrotizing pancreatitis involving only the peripancreatic tissues. The pancreatic parenchyma (*white star*) enhances normally, surrounded by a heterogeneous collection containing liquid and nonliquid components in the left retroperitoneum (*white arrows* pointing at the borders of the ANC). The ANC is not yet fully encapsulated.

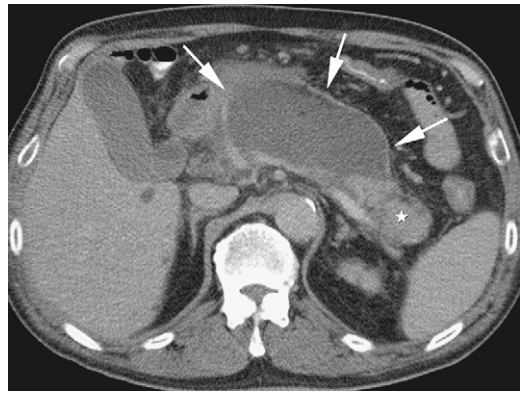

Fig. 8. A 51-year-old man with walled-off necrosis (WON) after an acute attack of acute necrotizing pancreatitis (*white star* shows normal enhancement of the pancreatic tail). A heterogeneous, fully encapsulated collection is noted in the pancreatic and peripancreatic area (*white arrows* pointing at the borders of the WON).

WON may be multiple and present at sites distant from the pancreas, and may or may not become infected. Demonstration of the presence or absence of a pancreatic ductal communication is not necessary in this classification but is of potential clinical import, because any ductal communication may affect management.

Sterile Versus Infected Necrosis

An ANC or WON can remain sterile or become infected (infected necrosis). Infection can be suspected by the clinical course of the patient (fever, leukocytosis, tachycardia) or by the presence of extraluminal gas within the areas of necrosis evident on CECT (see **Fig. 4**).

SUMMARY

The aim of this study was to update the original 1991 Atlanta Classification of acute pancreatitis to standardize the reporting and terminology of the disease and its complications. Although not necessarily commissioned by any one society, the concept of revising the prior 1991 classification received support in principle from the American Pancreatic Society, International Association of Pancreatology, European Pancreatic Club, pancreas section of the American Gastroenterological Association, Society for Surgery of the Alimentary Tract, the Pancreas Club, and several other international societies and associations interested in pancreatic disorders.

Important features of this classification have incorporated the new insights into the disease learned over the last 20 years, including the recognition that acute pancreatitis and its complications involve a dynamic process involving two phases, early and late. The accurate and consistent description of the two types of acute pancreatitis (interstitial edematous pancreatitis and necrotizing pancreatitis), its severity, and, possibly most importantly, the description of local complications based on characteristics of fluid and necrosis involving the peripancreatic collections, will help to improve the stratification and reporting of new methods of care of acute pancreatitis across different practices, geographic areas, and countries. By using a common terminology, the advancement of the science of acute pancreatitis should be facilitated.

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