The Revised Atlanta Classification of Acute Pancreatitis: Its Importance for the Radiologist and Its Effect on Treatment

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An international working group has modified the Atlanta classification for acute pancreatitis to update the terminology and provide simple functional clinical and morphologic classifications. The modifications (a) address the clinical course and severity of disease, (b) divide acute pancreatitis into interstitial edematous pancreatitis and necrotizing pancreatitis, (c) distinguish an early phase (1st week) and a late phase (after the 1st week), and (d) emphasize systemic inflammatory response syndrome and multisystem organ failure. In the 1st week, only clinical parameters are important for treatment planning. After the 1st week, morphologic criteria defined on the basis of computed tomographic findings are combined with clinical parameters to help determine care. This revised classification introduces new terminology for pancreatic fluid collections. Depending on presence or absence of necrosis, acute collections in the first 4 weeks are called acute necrotic collections or acute peripancreatic fluid collections. Once an enhancing capsule develops, persistent acute peripancreatic fluid collections are referred to as pseudocysts; and acute necrotic collections, as walled-off necroses. All can be sterile or infected. Terms such as pancreatic abscess and intrapancreatic pseudocyst have been abandoned. The goal is for radiologists, gastroenterologists, surgeons, and pathologists to use the revised classifications to standardize imaging terminology to facilitate treatment planning and enable precise comparison of results among different departments and institutions.

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Learning Objectives:
- Define acute pancreatitis in its early phase and later phase, and the persistent organ failure that can accompany its occurrence.
- List the various fluid collections encountered in acute pancreatitis as defined by the revised Atlanta classification.
- Identify the two phases of acute pancreatitis, the parameters that determine care, and the treatment for an infected walled-off necrosis.

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In 1992, the Atlanta classification for acute pancreatitis was introduced as a universally applicable classification system for the various manifestations of acute pancreatitis (1). This system was designed to facilitate understanding and correlation of findings seen by gastroenterologists, pathologists, radiologists, and surgeons. This approach was to be particularly useful for assessment and treatment of the various fluid collections identified during the course of acute pancreatitis. It defined acute pancreatitis as an acute inflammatory process of the pancreas with variable involvement of other local tissues and remote organ systems. It is associated with elevated pancreatic enzyme levels in blood and/or urine. Mild pancreatitis was described as associated with minimal organ dysfunction and an uneventful recovery. Severe pancreatitis was defined as associated with organ failure and/or local complications such as “acute” pseudocyst, pancreatic necrosis, or pancreatic abscess (2). Both categories were described as having acute fluid collections early in the course of the disease. A Ranson score of 3 or higher or an APACHE II (Acute Physiology and Chronic Health Evaluation II) score of 8 or higher was suggested as clinically predictive of severity. Organ failure and systemic complications were diagnosed on the basis of signs of shock, pulmonary insufficiency, renal failure, gastrointestinal bleeding, disseminated intravascular coagulation, and severe metabolic disturbances.

This initial Atlanta classification system represented major progress, but advancing knowledge of the disease process, improved imaging, and ever-changing treatment options such as minimally invasive radiologic, endoscopic, and laparoscopic procedures soon rendered some of the definitions inadequate or ambiguous (2,3), presenting a need to revise and update the Atlanta classification (4). It was found that the definitions of severity and local complications of acute pancreatitis were not used consistently and that characterization of severity based on presence of organ failure had limitations (2,3). The definition of necrotizing pancreatitis was determined to be inadequate because it included sterile and infected necrosis and did not distinguish between pancreatic and peripancreatic necrosis (2). The initial Atlanta classification system also did not include exact radiologic criteria for local complications, and controversy developed over the natural course of pancreatic and peripancreatic fluid collections.

In 2008, a global consensus statement was developed that included broad and international participation of many experts in the field of pancreatitis and was led by the Acute Pancreatitis Classification Working Group (4). This working group gathered input and revised the Atlanta classification system to improve clinical assessment and management of acute pancreatitis and to clarify appropriate terms for peripancreatic fluid collections, pancreatic and/or peripancreatic necrosis, and their changes over time (4–7). It also recognized that morphologic characteristics and clinical severity might not directly correlate (2). Such a revised classification system facilitates standardized reporting of clinical and imaging data, as well as objective assessment of treatment, which can be used as an effective means of communication among physicians. It also enables comparison of results among different institutions. Precise description of pancreatic collections is particularly important, because treatment varies with collection type. In short, the goal of this revised classification system is to facilitate more objective communication between physicians and institutions through a precise standardized classification system that allows better treatment planning. This revised classification is directly applicable only to adults (>18 years of age).

Radiologic imaging has become increasingly important in staging and treating acute pancreatitis (8,9). The revision of the Atlanta classification focuses heavily on morphologic criteria for defining the various manifestations of acute pancreatitis as outlined principally by means of

**Essentials**

- The revised Atlanta classification distinguishes an early phase (1st week) in which clinical parameters determine treatment from a later phase (after the 1st week) in which treatment is determined on the basis of clinical parameters and morphologic criteria defined by CT.
- Severe acute pancreatitis is defined in the first phase as organ failure lasting more than 48 hours or death; and during the second phase, as persistent organ failure, death, or complications resulting from acute pancreatitis.
- Fluid collections are defined by presence or absence of necrosis and infection: acute peripancreatic fluid collections (in the first 4 weeks without necrosis), pseudocysts (encapsulated fluid collections after 4 weeks, without necrosis), acute necrotic collections (ANCs; in first 4 weeks, with necrosis), and walled-off necrosis (WON; encapsulated collections after 4 weeks, with necrosis).
- Intraparenchymal fluid collections due to pancreatitis are referred to as ANCs or WONs, not as pseudocysts.
- Pseudocysts rarely become infected or require intervention; for sterile ANC or WON, any need for drainage is based on the clinical information; infected ANCs or WONs usually require intervention.

**Abbreviations:**

- **ANC** = acute necrotic collection
- **APACHE II** = Acute Physiology and Chronic Health Evaluation II
- **APFC** = acute peripancreatic fluid collection
- **FNA** = fine-needle aspiration
- **IEP** = interstitial edematous pancreatitis
- **WON** = walled-off necrosis

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computed tomography (CT). This revision places major emphasis on revised or new criteria for pancreatic fluid collections and revises some of the clinical criteria and terminology (4). This review article will principally address the new definitions for the various manifestations of fluid collections and/or liquefaction and their CT criteria as they occur during the course of acute pancreatitis; it also briefly outlines the revised terminology for description of the clinical course of acute pancreatitis. The goal is to familiarize radiologists with the revisions so that they may adopt these criteria and the terminology in their clinical practice and research. The author has been a consultant to the working group for the description of the radiologic manifestations of the various forms and complications of acute pancreatitis and for the revision of the manuscript. Some of his suggestions may have been included in the final report. This review will also briefly discuss treatment options for various complications of acute pancreatitis on the basis of CT and clinical findings.

**Clinical Definition, Course, and Severity of Disease**

Compared with the original Atlanta classification of acute pancreatitis by the international symposium in 1992 (1), the present revisions much more meticulously delineate the clinical diagnosis, more precisely describe the clinical course, and further define the clinical severity of acute pancreatitis. The major changes in the definitions of the various collections that occur during the course of pancreatitis will be emphasized in the section on Imaging-based Morphologic Classification.

**Clinical Definition**

According to the revised Atlanta classification of acute pancreatitis, acute pancreatitis (regardless of presence or absence of chronic pancreatitis) is clinically defined by at least the first two of three features (4): (a) abdominal pain suggestive of pancreatitis (epigastric pain often radiating to the back), with the start of such pain considered to be the onset of acute pancreatitis; (b) serum amylase and lipase levels three or more times normal (imaging is to be used if the elevated values are <3 times normal); and (c) characteristic findings on CT, magnetic resonance (MR) imaging, or transabdominal ultrasonographic (US) studies. If acute pancreatitis is diagnosed on the basis of the first two criteria with no systemic sign of severe systemic inflammatory response syndrome or persistent organ failure, contrast material–enhanced CT may not be necessary for determining patient care.

**Course and Severity of Disease**

The revised Atlanta classification introduces two distinct phases of acute pancreatitis: a first, or early, phase that occurs within the 1st week of onset of disease; and a second, or late, phase that takes place after the 1st week of onset (4,10–12). During the 1st week of acute pancreatitis, the pathologic conditions in and around the pancreas progress from early inflammation with variable degrees of peripancreatic edema and ischemia to resolution or to permanent necrosis and liquefaction. In this early phase, severity is entirely based on clinical parameters, because the need for treatment in the first phase is determined primarily by the presence or absence of organ failure caused by systemic inflammatory response syndrome and much less by morphologic findings involving the pancreas and peripancreatic areas. For organ failure, the Marshall scoring system (Table 1) is most commonly used, and the respiratory, cardiovascular, and renal systems need to be assessed (13,14). Over the course of the 1st week, organ failure either resolves or becomes more severe. Patients with organ failure that resolves in 48 hours are considered to have mild pancreatitis without complications and have a mortality rate of 0% (11,15). Severe acute pancreatitis in the first phase is defined as organ failure that lasts more than 48 hours or death (10,11). Expansion of systemic inflammatory response syndrome and ensuing multiorgan failure is responsible for many deaths during this phase (10). In this initial time period, there is not always a direct correlation between clinical severity with or without organ failure and extent of morphologic characteristics in and around the pancreas (Figs 1, 2) (16,17).

It is standard clinical practice within the first 3 days of admission of a patient with acute pancreatitis to record markers of severity (eg, hematocrit; score from APACHE II, Ranson, or other system; pulmonary complications on chest radiograph, including pleural effusion; and serum levels of C-reactive protein) (4,8). Other severity markers may also be used (CT severity index or modified CT severity index; serial blood, urea, nitrogen measurements; levels of creatinine, serum lactate dehydrogenase, serum and/or urinary trypsinogen, and cytokines; and other parameters of acute pancreatic injury). Potential risk factors to assess are age, comorbidities, and body mass index (8). Serum amylase and lipase are important for diagnosing acute pancreatitis but are not clinical markers of severity. These latter parameters should be evaluated but are not part of the revised Atlanta classification system, and their discussing is beyond the scope of this review. Moreover, these markers for forecasting severity within the first 24–72 hours are of limited value for predicting the development of pancreatic necrosis, persistent organ failure, or death.

The late phase begins after the 1st week, may extend for weeks to months, and is characterized by increasing necrosis, infection, and persistent multiorgan failure (18). Local complications may manifest systemically with bacteremia and sepsis when necrotic tissue becomes infected. The need for treatment in this phase is determined by the presence of symptoms and/or complications of acute pancreatitis, and the type of treatment is based on the imaging findings in the area of the pancreas and peripancreatic region as seen on contrast-enhanced CT or MR images and by the presence of local complications. Morphologic data help guide therapy and must be added to the clinical criteria in this phase. Development of increasing necrosis, persistent systemic inflammatory response syndrome, and
multorgan failure cause a significant increase in mortality (19). The mortality rates for sterile necrosis remain relatively low (3%–10%), but superinfection of the necrosis increases the mortality rate substantially (20%–30%) (20).

There is an ongoing discussion about introducing a third category called “moderate acute pancreatitis.” This category would include disease in patients who have sterile pancreatic or peripancreatic complications or transient organ failure but no persistent systemic complications. This leads to a morbidity rate that is higher than that expected for mild pancreatitis but has very low mortality rate. Therefore it is quite different from severe pancreatitis.

### Imaging-based Morphologic Classification

#### Imaging

According to the revised Atlanta classification, contrast-enhanced CT is the primary tool for assessing the imaging-based criteria because it is widely available for these acutely ill patients and has a high degree of accuracy (21,22). Contrast-enhanced CT is especially suited for staging in patients with acute pancreatitis, helping assess complications, and monitoring of treatment response through follow-up studies. Not all patients with acute pancreatitis need to undergo contrast-enhanced CT. Contrast-enhanced CT is not indicated initially in patients with acute pancreatitis who have no clinical signs of severe pancreatitis and who show rapid clinical improvement. However, contrast-enhanced CT should be performed in patients who develop or are likely to develop severe acute pancreatitis or complications related to acute pancreatitis. The ideal time for assessing these complications with CT is after 72 hours from onset of symptoms. CT should be repeated when the clinical picture drastically changes, such as with sudden onset of fever, decrease in hemocrit, or sepsis. CT also is useful to guide catheter placement for drainage and to assess success of treatment in patients who underwent percutaneous drainage or other interventions.

Furthermore, in patients with their first episode of pancreatitis who are over 40 years of age and have no identifiable cause for pancreatitis, contrast-enhanced CT should be used to exclude a possible neoplasm (23). The radiologist should address whether pancreatic necrosis is present, characterize pancreatic parenchymal and extrapancreatic fluid collections, and describe the presence of ascites and extrapancreatic findings such as gallstones, biliary dilatation, venous thrombosis, aneurysms, and contiguous inflammatory involvement of the gastrointestinal tract.

According to the revised Atlanta classification, MR imaging or transabdominal or endoscopic US may be used for special indications (24–26). MR imaging is reserved for detection of choledocholithiasis not visualized on contrast-enhanced CT images and to further characterize collections for the presence of nonliquefied material (27–29). Nonliquefied material refers to solid and semisolid components.

### Table 1

<table>
<thead>
<tr>
<th>Marshall Scoring System for Acute Pancreatitis</th>
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<tr>
<td><strong>Organ System</strong></td>
</tr>
<tr>
<td>Respiratory*</td>
</tr>
<tr>
<td>Renal (mg/dL)†</td>
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<td>Cardiovascular (mm Hg)‡</td>
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* Ratio of partial pressure of arterial oxygen to fraction of inspired oxygen.
† Serum creatinine level. To convert to SI, multiply by 88.4.
‡ Systolic blood pressure.
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usually pancreatic and extrapancreatic debris and necrotic fatty tissue and may appear on contrast-enhanced CT images as a homogeneous or heterogeneous fluid collection. MR imaging has an important role in patients in whom contrast-enhanced CT is contraindicated (eg, due to allergy to iodinated intravenous contrast agents or pregnancy) (29–31). Transabdominal US can be helpful for determining the presence of stones in the gallbladder, but it is less accurate than contrast-enhanced CT or MR imaging for visualizing distal common bile duct stones and has the disadvantage of being operator dependent (32). In patients with renal insufficiency who cannot undergo administration of iodinated contrast material or gadolinium, unenhanced CT or MR imaging may be used (30,33,34). Endoscopic retrograde cholangiopancreatography has no role in this morphologic imaging–based classification of acute pancreatitis.

The morphologic classification system based on contrast-enhanced CT findings requires close collaboration between the diagnostic radiologist, the “interventionalists” (endoscopist, surgeon, interventional radiologist), and the clinician. The findings identified on CT or MR images allow appropriate staging of acute pancreatitis and help predict complications (21,35). The clinician in turn integrates the reported morphologic findings into the clinical picture to optimize treatment, which should lead to improved outcomes.

**Morphologic Stages of Acute Pancreatitis**

In the 1992 Atlanta classification, a distinction was made between interstitial pancreatitis and sterile or infected necrosis. In the revised Atlanta classification, these two types are defined similarly as IEP and acute necrotizing pancreatitis, but necrotizing pancreatitis is further subdivided into parenchymal necrosis alone, peripancreatic necrosis alone, and a combined type (peripancreatic and parenchymal necrosis) with or without infection (Fig 3). The imaging-based revised classification involves careful assessment of CT images of collections of fluid and/or nonliquefied material in and around the pancreas (ie, areas of pancreatic parenchymal and peripancreatic necrosis). The terminology for fluid collections is completely revised. It is important for the radiologist to adopt this new nomenclature so that imaging descriptions are standardized and communication with clinical and surgical colleagues is precise. The revised Atlanta classification also outlines other important findings to be evaluated with imaging such as causes of pancreatitis, including cholecystolithiasis and choledocholithiasis, or complications related to acute pancreatitis, including extrapancreatic biliary dilatation; splenic, portal, and mesenteric venous thrombosis; varices; arterial pseudoaneurysm; pleural effusion; and ascites. In addition, other intraabdominal findings caused by pancreatic secretions need to be reported. These are inflammatory changes due to pancreatic secretions in the stomach, duodenum, small bowel, colon, spleen, kidney, ureters and liver.

**Interstitial Edematous Pancreatitis**

In patients with IEP, contrast-enhanced CT demonstrates acute pancreatitis as localized or diffuse enlargement of the pancreas, with normal homogeneous enhancement or slightly heterogeneous enhancement of the pancreatic parenchyma related to edema (Fig 1). The peripancreatic and retroperitoneal tissue may appear normal, usually in early mild disease, or may show mild inflammatory changes in the peripancreatic soft tissue that appear as “mistiness” or mild fat stranding with varying amounts of peripancreatic fluid (see Pancreatic and Peripancreatic Collections). On a contrast-enhanced CT study obtained within the first several days of acute onset of pancreatitis, the pancreas occasionally demonstrates increased heterogeneous enhancement of the pancreatic parenchyma related to edema (Fig 4) that cannot be characterized definitively as either IEP or ill-defined necrosis. With these findings, the presence or absence of pancreatic necrosis needs to be described initially as indeterminate. Contrast-enhanced CT performed 5–7 days later permits definitive characterization.

**Necrotizing Pancreatitis**

The revised Atlanta classification system distinguishes three forms of acute necrotizing pancreatitis, depending on location. This represents a distinct change from the initial classification. All three types can be sterile or infected.
Pancreatic parenchymal necrosis alone.—Pancreatic parenchymal necrosis alone can be seen in fewer than 5% of patients and appears on contrast-enhanced CT images as lack of parenchymal enhancement (36). In the 1st week of necrotizing pancreatitis, contrast-enhanced CT demonstrates necrosis as a more homogeneous nonenhancing area of variable attenuation (Fig 5) and, later in the course of the disease, as a more heterogeneous area. The radiologic changes are the result of a process in which the nonviable and necrotic tissues (primarily pancreatic parenchyma and peripancreatic fat) slowly begin to liquefy. Often the extent of parenchymal necrosis is divided on contrast-enhanced CT studies into three categories: less than 30%, 30%–50%, or greater than 50% of the gland involved (37). In a newer modified CT grading system only two categories are distinguished: less than 30% and greater than 30% (35). At times, areas of non enhancement that are estimated to be less than 30% in the early phase may actually be findings of edema rather than necrosis (9,37). A definitive diagnosis in these patients requires a follow-up study.

Peripancreatic necrosis alone.—Peripancreatic necrosis alone can be seen in approximately 20% of patients and can be difficult to confirm (36). Its presence is diagnosed when heterogeneous areas of nonenhancement (Fig 6) are visualized that contain nonliquefied components. Peripancreatic necrosis is commonly located in the retroperitoneum and lesser sac. The clinical importance of peripancreatic necrosis alone lies in the fact that patients with this condition have a better prognosis than do patients with pancreatic parenchymal necrosis (38). Nevertheless, patients with peripancreatic necrosis have a higher morbidity rate than do patients with IEP only (39).

Pancreatic parenchymal necrosis with peripancreatic necrosis.—Acute pancreatic parenchymal necrosis with peripancreatic necrosis is the most common type and can be seen in 75%–80% of patients with acute necrotizing pancreatitis (36). The radiologic appearance of pancreatic parenchymal necrosis with peripancreatic necrosis is a combination of the findings described above for pancreatic parenchymal necrosis alone and peripancreatic necrosis alone (Fig 7). Peripancreatic necrosis associated with full width necrosis of the pancreatic parenchyma may be connected to the main pancreatic duct (40).

Pancreatic and Peripancreatic Collections

Acute pancreatitis can be accompanied by pancreatic parenchymal or peripancreatic collections (Fig 3). In the revised Atlanta classification, an important distinction is made between fluid and nonliquefied collections (4). The acute collections are referred to as either APFCs or as ANCs, depending on the absence or presence, respectively, of necrosis. IEP can be associated with APFC and, over time, with pancreatic pseudocysts. Necrotizing pancreatitis in its three forms can be associated with ANC and, over time, with WON. All of these collections can be sterile or infected.

APFCs.—Peripancreatic fluid collections without nonliquefied components arising in patients with IEP during the first 4 weeks are referred to as APFCs (Fig 3). They are caused by pancreatic and peripancreatic inflammation or by rupture of one or more small peripheral pancreatic side duct branches. APFCs conform to the anatomic boundaries of the retroperitoneum (especially the anterior pararenal fascia), are usually seen immediately next to the pancreas (Table 2, Fig 8), and have no discernable wall. Fluid collections in the pancreatic parenchyma should be diagnosed as necrosis and not as APFCs. Most APFCs are reabsorbed spontaneously within the first few weeks and do not become infected. Intervention at this stage is to be avoided, because
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Pseudocyst.—Within 4 weeks from onset of acute IEP, an APFC may gradually transition into a pseudocyst. Pseudocyst occurs as a complication of acute pancreatitis in approximately 10%–20% of cases (41). On contrast-enhanced CT images, pseudocysts can be diagnosed as well-circumscribed, usually round or oval peripancreatic fluid collections of homogeneously low attenuation that are surrounded by a well-defined enhancing wall (capsule consisting of fibrous or granulation tissue). According to the revised Atlanta classification, pseudocysts contain no nonliquefied components within the fluid collection (Table 2, Fig 9). Prior to 4 weeks, a definite enhancing wall has usually not formed, and such a collection should be categorized as an APFC. In the rare event in which an APFC develops an enhancing capsule earlier than 4 weeks after onset of acute IEP, it should be characterized as a pseudocyst. The pseudocyst contains fluid with increased amylase and lipase activity due to communication with the pancreatic ductal system. However, many pseudocysts seal off such a communication and vanish spontaneously. Demonstrating the presence or absence of communication with the pancreatic duct may be difficult or impossible, because both collections may appear as areas of nonenhancement. If nonenhancing areas of variable attenuation are seen in these collections, the diagnosis of peripancreatic necrosis with nonliquefied components is suggested. Nonliquefied components are primarily hemorrhage, fat, and/or necrotic fat. Such findings are not compatible with IEP, and, in these cases, the process should be diagnosed as acute necrotizing pancreatitis with peripancreatic necrosis alone. A diagnosis of peripancreatic necrosis based on contrast-enhanced CT findings often cannot be made specifically but can be suspected when slightly heterogeneous peripancreatic collections are seen. After 1 week from onset, the collection usually becomes clearly heterogeneous, and necrosis can be diagnosed on contrast-enhanced CT images.

Figures 6 and 7: Acute necrotizing pancreatitis: peripancreatic necrosis alone. (a) Axial multidetector CT image obtained 5 days from onset of pancreatitis shows slightly edematous pancreas surrounded by fluid collections (arrows) that contain nonenhancing areas of variable attenuation. Collections contain nonliquefied material, which at times may be difficult to discern and are referred to as ANCs. (b) Axial multidetector CT image obtained 5 weeks after onset shows peripancreatic WON anterior to pancreas and extending around the Gerota fascia with a well-defined wall (white arrows), heterogeneous content with debris and loculations (white arrowheads), and two percutaneous drains (black arrowheads). Feeding tube also is seen in the duodenum (black arrow). (c) Coronal CT reconstruction shows extent of the peripancreatic WONs (white arrows) with percutaneous drain (black arrow) and debris (arrowheads).

Figures 8: IEP in a 25-year-old woman with alcohol abuse and epigastric pain for 72 hours. Axial CT image shows the pancreas (arrowhead) to be slightly edematous and heterogeneously enhancing. APFCs (arrows) are seen surrounding the pancreas.

In the 1st week of acute pancreatitis, distinction between APFC and ANC necessitates drainage.

Figure 8: IEP in a 25-year-old woman with alcohol abuse and epigastric pain for 72 hours. Axial CT image shows the pancreas (arrowhead) to be slightly edematous and heterogeneously enhancing. APFCs (arrows) are seen surrounding the pancreas.
be important since it may help determine management. Persistent communication with the pancreatic duct can be shown on contrast-enhanced CT images and curved planar reconstructions, but MR cholangiopancreatography is usually more accurate (42,43). In the rare case when a pseudocyst becomes infected, it contains purulent liquid but no nonliquefied material. An infected pseudocyst is diagnosed on CT images by the presence of gas within the pseudocyst or, in absence of gas, by means of fine-needle aspiration (FNA) with Gram staining and culture for bacteria or fungal organisms (38).

In rare instances, a pseudocyst can develop in patients after pancreatic resection due to necrosis and subsequent leakage of pancreatic secretions from the remaining duct or in patients with disconnected duct syndrome (44).

ANCs.—In the first 4 weeks after development of necrotizing pancreatitis, a persistent collection is to be diagnosed as ANC that contains both fluid and necrotic material of various amounts (some of which are loculated) and is to be distinguished from APFC. The revised Atlanta classification carefully avoids the term fluid collection for this stage to emphasize the fact that these collections contain more than fluid. In these ANCs, liquefaction of the necrotic tissue occurs gradually (usually within 2–6 weeks). More and more liquefaction develops as the necrotic tissue breaks down. Within the 1st week, both APFCs and ANCs can manifest as homogeneous nonenhancing areas. Usually, the distinction on contrast-enhanced CT images should become possible after the 1st week, because these collections with necrotic debris appear more complex on images (Table 2; Figs 5b, 6). Within the first 4 weeks of onset of acute necrotizing pancreatitis, any collection in the pancreas that replaces pancreatic

**Table 2**

<table>
<thead>
<tr>
<th>Type of Collection</th>
<th>Time (wk)</th>
<th>Necrosis</th>
<th>Location</th>
<th>Appearance</th>
<th>Infection</th>
<th>Drainage or Surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>IEP</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>APFC</td>
<td>≤4</td>
<td>No</td>
<td>Adjacent to pancreas, extrapancreatic only</td>
<td>Homogeneous, fluid attenuation, no liquefaction (debris), not encapsulated</td>
<td>Extremely rare</td>
<td>None</td>
</tr>
<tr>
<td>Pseudocyst*</td>
<td>&gt;4</td>
<td>No</td>
<td>Adjacent or distant to pancreas</td>
<td>Homogeneous, fluid attenuation, no liquefaction (debris), encapsulated</td>
<td>Rare</td>
<td>Rarely (for infection or symptoms)</td>
</tr>
<tr>
<td>Necrotizing pancreatitis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Sterile ANC</td>
<td>≤4</td>
<td>Yes</td>
<td>In parenchyma and/or extrapancreatic</td>
<td>Heterogeneous¹, nonliquefied material, variably loculated, not encapsulated</td>
<td>No</td>
<td>Based on clinical, percutaneous drainage at times, surgery rarely ²</td>
</tr>
<tr>
<td>Infected ANC</td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>Sterile WON</td>
<td>&gt;4</td>
<td>Yes</td>
<td>In parenchyma and/or extrapancreatic</td>
<td>Heterogeneous¹, nonliquefied material, variably loculated, encapsulated</td>
<td>No</td>
<td>Percutaneous drainage based on clinical, surgery to follow if needed ²</td>
</tr>
<tr>
<td>Infected WON</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Percutaneous drainage/surgery to follow if needed ²</td>
</tr>
</tbody>
</table>

Source.—Reference 4.

* Rarely in necrotizing pancreatitis after resection or in disconnected duct syndrome.

† Some homogeneous early in course.

‡ Or endoscopic procedure.

Figure 9: Pancreatitis with pseudocyst in a 27-year-old woman. Coronal CT reconstruction obtained 5 weeks after acute episode shows pseudocyst (arrows) with well-defined rim representing the capsule near the tail of the pancreas. Gastric folds are slightly thickened (arrowheads).
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apparent fluid collection that occupies or replaces portions of the pancreatic parenchyma should be called a WON after 4 weeks from onset of necrotizing pancreatitis. This WON may or may not be infected. Demonstrating a communication of the WON with the pancreatic duct is not necessary for the Atlanta classification, but it may change management. In contradistinction to a pseudocyst, WON contains necrotic pancreatic parenchyma or necrotic fat. Most nonliquefied components need to be removed by means of a percutaneous image-guided approach, a laparoscopic or endoscopic procedure, or surgery. A pseudocyst can be treated effectively by draining the fluid in most cases. Therefore the distinction between a collection containing fluid only and a collection containing fluid and nonliquefied material is very important.

Complications of Acute Pancreatitis

All four types of pancreatic fluid collections can be sterile or infected. Collections that contain nonliquefied material are more likely to become infected. Distinction between a sterile and an infected collection is important because treatment and prognosis are different, as outlined below (45). Infection can be suggested on contrast-enhanced CT images if gas bubbles are present in the collection owing to the presence of gas-forming organisms (Fig 11) (46). Spontaneous drainage into the gastrointestinal tract can lead to an erroneous diagnosis of infected pseudocyst or necrosis. Careful analysis of the adjacent gastrointestinal walls can help prevent this diagnostic pitfall. Gas can also be present in a collection after marsupialization or other drainage procedures. In the absence of gas in the collection, definitive proof can be obtained only by performing FNA of the collection with a positive Gram stain and culture for bacteria or fungal organisms (38). Owing to the fear of introducing infection through the aspiration needle, FNA should be performed only when there is a high clinical suspicion of superinfection or if imaging results suggest the collection is infected.

Figure 10

WON of pancreatic body, tail, and portion of the head in a 45-year-old man with alcohol abuse and necrotizing pancreatitis. (a) Axial CT image obtained 6 weeks after acute onset shows some areas of lower attenuation (arrowheads) in a heterogeneous collection with a well-defined rim (arrows), representing WON with fat necrosis involving pancreas and peripancreatic tissues. (b) Axial CT image obtained several centimeters caudal to a shows WOns extending into right anterior pararenal and left anterior and posterior pararenal space (arrows).

Figure 11

Large infected WON in a 57-year-old man with necrotizing pancreatitis. (a) Axial CT image obtained 5 weeks after acute onset shows pancreas replaced by low-attenuation collection with well-defined rim (arrows) and multiple pockets of gas (arrowheads). (b) Axial CT image obtained 3 days after placement of percutaneous drainage catheters (arrows) shows large residual WON with air bubbles, indicative of incomplete drainage of an infected WON. The patient did not show notable improvement, and drainage catheters were replaced with larger caliber catheters; eventually the patient underwent surgical débridement, which was successful.
and care must be taken to avoid a possibly contaminating route such as a transgastric or transduodenal approach (47). A retroperitoneal route via the lateral flank is preferred over an anterior approach through the peritoneum. Aspiration of fluid for the purpose of diagnosing infection has a false-negative rate of less than 10% (48). Therefore if the FNA result is negative but clinical suspicion of infection persists, FNA should be repeated.

Any infected necrosis has varying amounts of necrotic material and pus, and the pus increases with increased liquefaction. Since a localized collection of purulent material without substantial necrotic material is rare in infected pancreatic necrosis, the term pancreatic abscess is no longer used. Patients with infected necrosis usually need percutaneous, laparoscopic, endoscopic, or surgical intervention. Patients with sterile necrosis usually do not require any intervention unless they have persistent pain, anorexia, or vomiting or are unable to resume oral feeding.

**Treatment Options**

In addition to fostering better communication among physicians, the revised Atlanta classification is designed to aid patients' treatment through appropriate triage to intervention or conservative medical care. The severity or stage of acute pancreatitis dictates the type of treatment that the patient needs.

**Treatment of IEP**

IEP is usually self-limited, and supportive measures alone suffice (Table 2). Most APFCs resolve spontaneously or mature into pseudocysts. The majority of these pseudocysts disappear spontaneously over time and do not require any treatment. About 25% become symptomatic or infected and necessitate drainage (49,50). Once the presence of nonliquefied material and infection has been excluded, simple percutaneous drainage is usually sufficient for large and/or symptomatic pseudocysts (Fig 12). In cases when superinfection is clinically suspected, CT images may show air bubbles in the collection, but FNA is needed for a definitive diagnosis of many infected pseudocysts. Most infected pseudocysts are drained percutaneously rather than surgically (51,52). Several percutaneous approaches can be taken, but generally a retroperitoneal approach through the lateral flank, which carefully avoids solid organs and bowel, is preferred over an anterior approach through the peritoneal cavity (53). A cystogastrostomy can be successful in experienced hands when an image-guided percutaneous route is used (54). In uninfected pseudocysts, a transgastric approach may increase the risk for superinfection and should be reserved for targets that cannot be easily approached via other routes. Endoscopic drainage of pseudocysts should be performed only for cysts that have a mature wall and are in proximity to the gastrointestinal lumen (55). The advantages of an endoscopically placed cystogastrostomy include that it can be performed in patients who are not candidates for general anesthesia and surgery and that a pancreaticocutaneous fistula does not develop (56). However, this endoscopic type of procedure is not currently suitable for patients with complex (infected) pseudocysts.

**Treatment of Necrotizing Pancreatitis**

Necrotizing pancreatitis requires close monitoring, and minimally invasive radiologic procedures or laparoscopic, endoscopic, or surgical techniques often are needed to improve the outcome in these patients (Table 2). Once the diagnosis of necrotizing pancreatitis (with or without peripancreatic necrosis) has been established on the basis of contrast-enhanced CT findings, a treatment plan can be developed. Whereas clinical scoring systems (eg, APACHE II) accurately correlate with systemic complications and mortality, the CT severity index or modified CT severity index more accurately helps establish the presence of clinically severe disease and more precisely relates to pancreatic infection and need for intervention (35). Since contrast-enhanced CT may demonstrate some WONs as relatively homogeneous fluid collections, necrotic debris in such a collection may be difficult to diagnose, and MR imaging or US should be used for confirmation (Fig 13).

No universally accepted treatment algorithm currently exists. The approach often is dictated by the expertise of the surgeon and the interventional radiologist. A consensus has been reached as to the indications for interventional procedures versus those for surgery in patients with acute necrotizing pancreatitis with or without peripancreatic necrosis (57). The clinical status of the patient (eg, presence of sepsis or acute hemorrhage) often determines the approach to be taken.

In patients with acute necrotizing pancreatitis, a shift in treatment approach has emerged from early surgical débridement to supportive therapy during the first 2 weeks after onset of symptoms. This was largely brought about by reports from several studies that demonstrated a high mortality rate in patients after early surgical intervention. In one prospective randomized study, the authors reported a mortality rate of 58% in patients who underwent surgery 48–72 hours after onset of symptoms versus a mortality rate of 27% in patients whose surgery was delayed for more than 12 days after onset of symptoms (58). Other investigators confirmed these results (59). Others have suggested that surgery is best delayed for at least a month after the onset of acute pancreatitis (57) and should then be performed only if the acute necrotizing pancreatitis is confirmed to be infected and/or if the patient has persistent pain and cannot eat after systemic inflammatory response syndrome has resolved.

Image-guided drainage procedures have proved to be effective alternatives to surgery, particularly early in the course of complications from severe acute pancreatitis with necrosis (57,60–63). Some of these percutaneous procedures are performed to stabilize seriously ill patients before surgery (bridge care), and others are intended to cure (7,60,64). In some
may benefit from FNA of the necrosis to rule out infected necrosis. Care must be taken not to traverse bowel with the needle, to prevent contamination of the sample or the aspirated area. If the pancreatic fluid sample is sterile, the patient is diagnosed as having sterile necrosis. Some patients with sterile necrosis recover rapidly, while toxicity remains in others and they must stay in the intensive care unit for weeks (57). This lack of improvement may be due to pancreatic duct disruption and development of additional peripancreatic necrotic collections. In these patients, percutaneous drainage and supportive measures are preferred rather than surgery because of the higher morbidity and mortality associated with surgery (Fig 6) (61,63). If CT demonstrates residual collections, and little or no drainage from the percutaneous catheter is observed, several drainage catheters may have to be placed and irrigated to achieve percutaneous necrosectomy and reduce toxicity (56). Percutaneous drainage of sterile necrosis remains controversial and has the potential of infection by means of colonization of the indwelling catheter (62). While such infection is possible, complete drainage of fluid and material within 2–3 days of catheter placement should prevent this complication (56). Follow-up CT is used to ensure adequate drainage has been achieved, and additional larger catheters may have to be placed in cases of residual necrotic fluid. In some patients, percutaneous catheter drainage is used to stabilize the patient before surgical débridement. To the author’s knowledge, no studies are available in which weekly fine-needle sampling to assess for infection was compared with direct drainage with indwelling catheters in patients with sterile necrosis who clinically do not do well.

Treatment of Infected Pancreatic Necrosis

In patients with infected necrosis, CT rarely demonstrates gas in the pancreas, lesser sac, or retroperitoneum, but the presence of gas is the only CT sign that permits the diagnosis of infected necrosis (see above). Infection of necrotic pancreatic tissue by bacteria is
Pseudoaneurysm in a 38-year-old woman with alcohol abuse and necrotizing pancreatitis.

**REVIEW:**

Revised Atlanta Classification of Acute Pancreatitis

Thoeni

Angiogram obtained for embolization of the pseudoaneurysm seen clearly identified in late arterial phase. (b) Axial CT image shows poor enhancement in neck and head of the pancreas, whereas no notable enhancement is present in pancreatic body and tail, indicating necrosis. Small pseudoaneurysm (arrowhead) can be seen in the pancreatic head. Small pseudoaneurysms (arrows) all of which were successfully treated with coil embolization.

Conclusions

The revised Atlanta classification is designed to precisely describe patients with acute pancreatitis, standardize terminology across specialties, and help in treatment planning. It defines acute pancreatitis as IEP or necrotizing pancreatitis and distinguishes between an early phase (1st week) and a late phase (after the 1st week). The first phase is defined by clinical parameters, and the second phase is defined morphologically on the basis of contrast-enhanced CT findings combined with clinical staging.

The most important change in the Atlanta classification is the categorization of the various pancreatic collections. In acute IEP, collections that do not have an enhancing capsule are called APFCs; after development of a capsule, they are referred to as pseudocysts (usually after the first 4 weeks). In necrotizing pancreatitis, a collection without an enhancing capsule is called an ANC (usually after the first 4 weeks). These novel approaches can lead to a decrease in morbidity and mortality.

**Related Treatment**

Interventional radiology also is called on for ancillary procedures. Pseudoaneurysms or active bleeding related to acute pancreatitis are usually diagnosed on the basis of contrast-enhanced CT findings, and images should be obtained in late arterial and portal venous phases.

Pancreatitis-associated pseudoaneurysms are treated on the basis of their location and morphology. Most commonly, coil embolization is used. This approach is used in patients with pseudoaneurysms that have a narrow neck and are located in an area where coils can be safely deployed without the risk of nontarget embolization (Fig 14). In some cases where coil embolization is too risky or not feasible, a covered stent can be placed. Embolization may also be performed in selected instances of a hemorrhaging vessel caused by pancreatitis. Because the authors of several studies have supported the use of enteral rather than parenteral nutrition in patients with acute pancreatitis, nasojejunal or percutaneous jejunal feeding tubes often must be placed, with the tip of the tube beyond the ligament of Treitz (70,71). If a percutaneous route is chosen, a transgastric approach best achieves this goal.

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can be sterile or infected. The most important distinction between collections in necrotizing pancreatitis and those associated with acute IEP is the presence of nonliquefied material in collections due to necrotizing pancreatitis. In the early phase of pancreatitis, distinction between APFC and ANC by CT may be impossible and, if clinically needed for treatment planning, MR imaging or US may be used to determine the presence of non-liquefied material. Depending on the time from onset of acute pancreatitis, any collection within the pancreatic parenchyma should be considered an ANC and not an APFC if less than 4 weeks have passed since the onset of symptoms or a WON and a not a pseudocyst if a well-defined capsule has developed. Determination of superinfection is based on clinical presentation and on presence of air observed in collections by CT and if air is absent on CT, by percutaneous needle aspiration. Treatment planning is based on severity of pancreatitis and presence or absence of infection combined with clinical signs. The revised Atlanta classification system with CT helps guide management and monitor the success of treatment.

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References


