The Revised Atlanta Classification of Acute Pancreatitis: Its Importance for the Radiologist and Its Effect on Treatment[1]

Ruedi F. Thoeni, MD

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.

Online CME
See www.rsna.org/education/ry_cme.html

Learning Objectives:

- Define acute pancreatitis in its early phase and later phase, and the persistent organ failure that can accompany its occurance.
- 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.

Accreditation and Designation Statement

The RSNA is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians. The RSNA designates this journal-based activity for a maximum of 1.0 *AMA PRA Category 1 Credit*™. Physicans should claim only the credit commensurate with the extent of their participation in the activity.

Disclosure Statement

The ACCME requires that the RSNA, as an accredited provider of CME, obtain signed disclosure statements from the authors, editors, and reviewers for this activity. For this journal-based CME activity, author disclosures are listed at the end of this article.

[1] From the University of California San Francisco Medical School, Department of Radiology and Biomedical Imaging, P.O. Box 1325, San Francisco, CA 94143-1325. Received May 5, 2011; revision requested June 20; revision received July 19; accepted August 5; final version accepted August 15; final review by the author November 9. **Address correspondence to** the author (e-mail: *ruedi.thoeni@radiology.ucsf.edu*).

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.

Published online
10.1148/radiol.11110947 **Content code:** GI

Radiology 2012; 262:751–764

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

Potential conflicts of interest are listed at the end of this article.

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

multiorgan failure cause a significant increase in mortality (19). The mortality rates for sterile necrosis remain relatively low (5%–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 hematocrit, 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

Marshall Scoring System for Acute Pancreatitis

Organ System	Score = 0	Score = 1	Score = 2	Score = 3	Score = 4
Respiratory*	>400	301–400	201–300	101–200	<101
Renal (mg/dL) †	≤1.5	>1.5 to ≤1.9	>1.9 to ≤3.5	>3.5 to ≤5.0	>5.0
Cardiovascular (mm Hg)‡	>90	<90, fluid responsive	<90, not fluid responsive	<90, pH < 7.3	<90, pH < 7.2

Source.—References 4, 72.

Note.—Organ failure is defined as score ≥ 2 for at least one of the three organ systems. Duration of organ failure is defined as transient, (≤48 hours from time of presentation), or persistent (>48 hours from time of presentation). Persistent multiorgan failure is defined as two or more organs failing during same 3-day period.

* Ratio of partial pressure of arterial oxygen to fraction of inspired oxygen.

† Serum creatinine level. To convert to Système International units (micromoles per liter), multiply by 88.4.

‡ Systolic blood pressure.

Figure 1

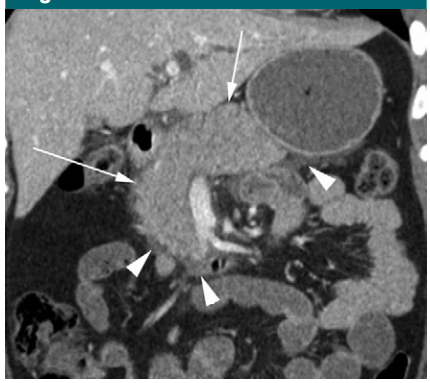


Figure 1: Coronal CT image of Interstitial edematous pancreatitis (IEP) in a 34-year-old man with acute onset of epigastric pain for 12 hours due to gallstones. Pancreas (arrows) is heterogeneously enhanced, with indistinct margins due to inflammation of peripancreatic fat. Some stranding and minimal fluid (arrowheads) are also present.

Figure 2

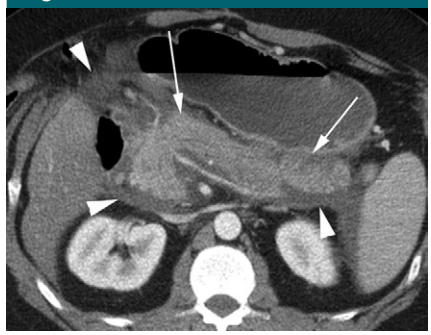


Figure 2: Axial CT image of IEP in a 51-year-old man with persistent organ failure (lipase, 1027 U/L [17.15 microkatals per liter]; Marshall score of 3 with persistently low systolic pressure of <90 mm Hg and pH 7.2; white blood cell count, 7000 cells/mm^3) for 5 days. Clinically, the Atlanta classification system places this in the severe pancreatitis group. At this stage, there is no correlation between morphologic appearance and clinical severity. Nevertheless, compared with Figure 1, edema in the pancreas (arrows) and peripancreatic stranding and fluid (arrowheads) are more extensive.

Figure 3

Type of Pancreatitis	Fluid Collections
< 4 Weeks after Onset	
IEP	APFC
	Sterile
	Infected
Necrotizing Pancreatitis	ANC
	Parencymal necrosis alone
	Sterile
	Infected
	Peripancreatic necrosis alone
	Sterile
	Infected
	Pancreatic and peripancreatic necrosis
	Sterile
	Infected
≥ 4 Weeks after Onset	
IEP	Pancreatic pseudocyst
	Sterile
	Infected
Necrotizing Pancreatitis	WON
	Sterile
	Infected

Figure 3: Revised Atlanta classification of fluid collections in acute pancreatitis (4). ANC = acute necrotic collection, APFC = acute peripancreatic fluid collection, WON = walled-off necrosis.

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 extrahepatic 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 parenchyma (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.

Figure 4

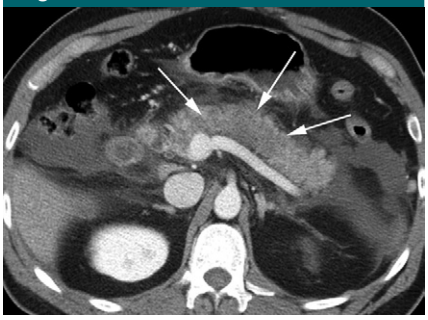
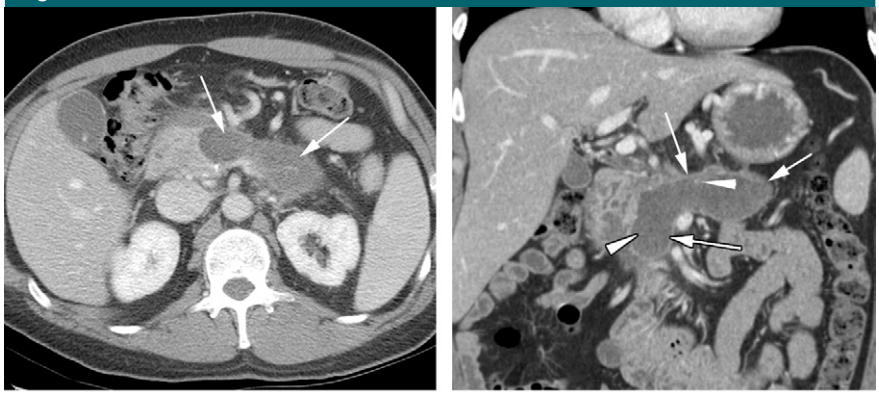


Figure 4: Axial multidetector CT image of IEP in a 39-year-old man with acute onset of epigastric pain obtained 48 hours after onset of pain. Note focal heterogeneous low-attenuation area in pancreas body and neck (arrows). At this stage, the appearance could not be definitively characterized as IEP or patchy necrosis and was classified as indeterminate. Follow-up multidetector CT study did not show any necrosis.

Figure 5



a. **b.**

Figure 5: Acute necrotizing pancreatitis: pancreatic parenchymal necrosis alone. **(a)** Axial CT image in a 38-year-old man obtained 5 days after onset of symptoms. Tail and body of the pancreas are nonenhancing (arrows) and slightly heterogeneous in appearance. **(b)** On coronal reformation CT image obtained 4 weeks after onset, capsule (arrows) is evident and some heterogeneity (arrowheads) is seen within this collection, reflecting presence of nonliquefied material.

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%, 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 no or poor 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).

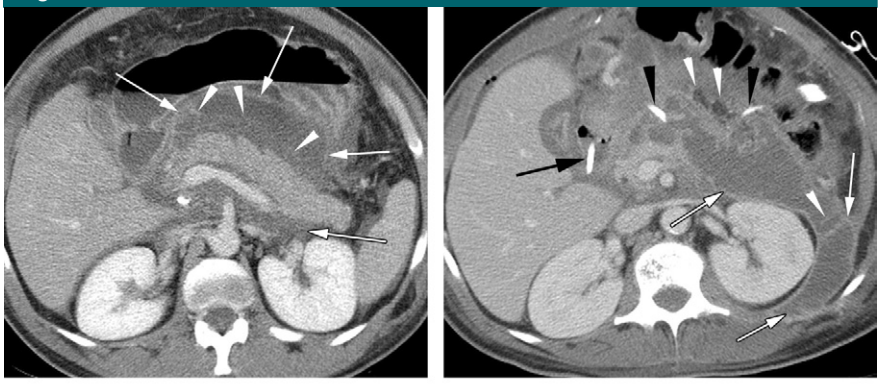
Pancreatic and Peripancreatic Collections

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.

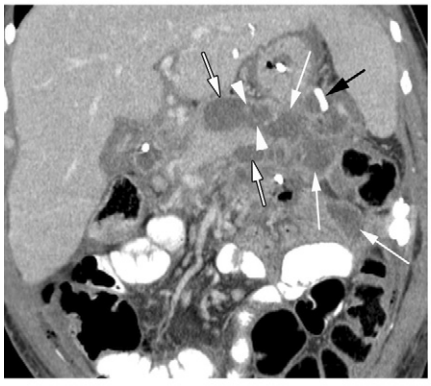
APFCs.—Peripancreatic fluid collections without nonliquefied components arising in patients with IEP during the first 4 weeks are referred to as APFCs (Fig 3). They are caused by pancreatic and peripancreatic inflammation or by rupture of one or more small peripheral pancreatic side duct branches. APFCs 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

Figure 6



a. b.



c.

Figure 6: Acute necrotizing pancreatitis: peripancreatic necrosis alone. **(a)** Axial multidetector CT image in a 58-year-old man obtained 5 days from onset of pancreatitis shows slightly edematous pancreas surrounded by fluid collections (arrows) that contain nonenhancing areas of variable attenuation and loculation (arrowheads). 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).

Figure 7

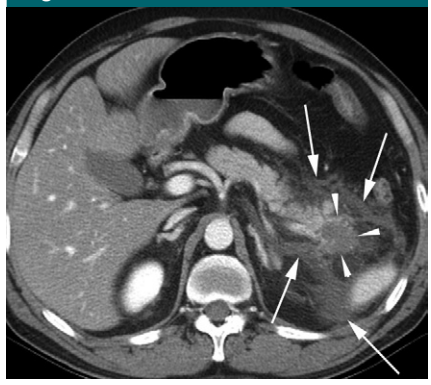


Figure 7: Parenchymal necrosis in tail of the pancreas with ANCs in a 34-year-old man. Axial CT image shows necrosis (arrowheads) in tail of the pancreas as lack of enhancement. Multiple ANCs (arrows) are seen surrounding tail and body of the pancreas.

Figure 8

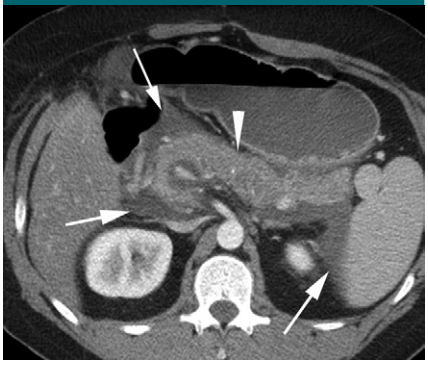


Figure 8: IEP in a 25-year-old woman with alcohol abuse and epigastric pain for 72 hours. Axial CT image shows the pancreas (arrowhead) to be slightly edematous and heterogeneously enhancing. APFCs (arrows) are seen surrounding the pancreas.

drainage or aspiration of fluid could introduce infection. Only the rare infected APFC necessitates drainage.

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

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

Table 2

Fluid Collections as Defined in Revised Atlanta Classification of Acute Pancreatitis and Possible Interventions

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

Source.—Reference 4.

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

† Some homogeneous early in course.

‡ Or endoscopic procedure.

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

Figure 9

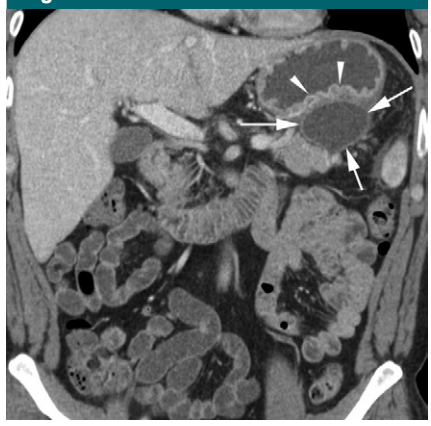
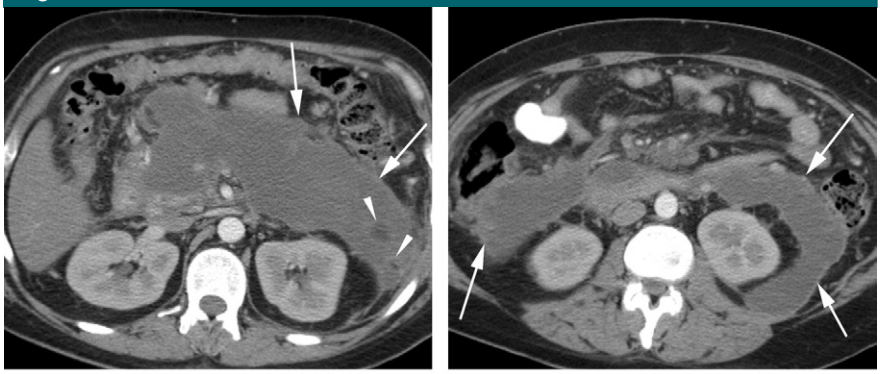


Figure 9: Pancreatitis with pseudocyst in a 27-year-old woman. Coronal CT reconstruction obtained 5 weeks after acute episode shows pseudocyst (arrows) with well-defined rim representing the capsule near the tail of the pancreas. Gastric folds are slightly thickened (arrowheads).

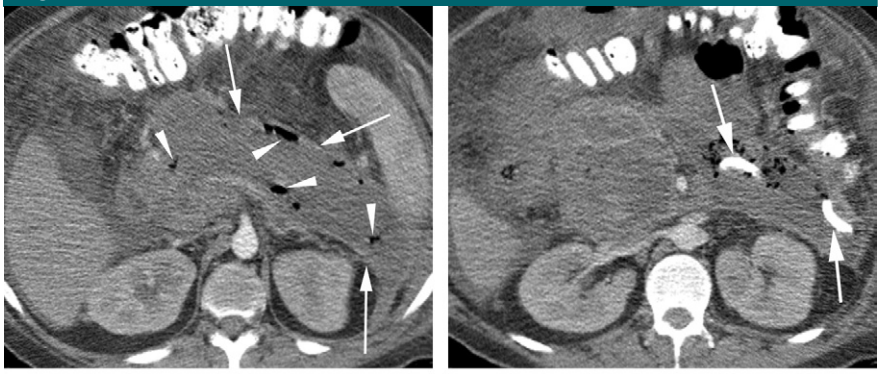
Figure 10



a. b.

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



a. b.

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.

parenchyma should be considered an ANC and not a pseudocyst. An ANC may or may not have a connection to the disrupted pancreatic ductal system within the necrosis.

WON.—Over time (usually at or after 4 weeks), the ANC matures and develops a thickened nonepithelialized wall between the necrosis and the adjacent tissue. This maturing collection is called a WON. Previous terms used for this phenomenon include *pancreatic sequestration, necroma,* and *organized pancreatic necrosis* and are a manifestation of the late stage of an ANC. WON was not recognized in the original Atlanta classification. Like ANC, WON may involve the pancreatic parenchymal tissue and the peripancreatic tissue (Fig 10), the peripancreatic tissue alone or the pancreas alone (Table 2). Any 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,

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.

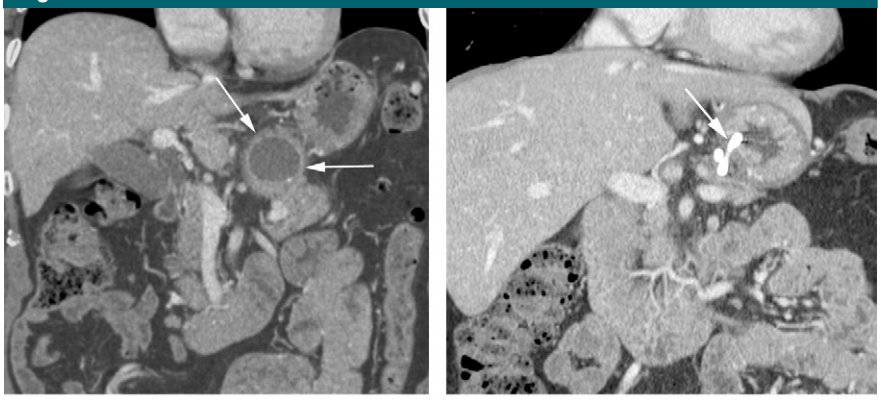
Treatment of Necrotizing Pancreatitis

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

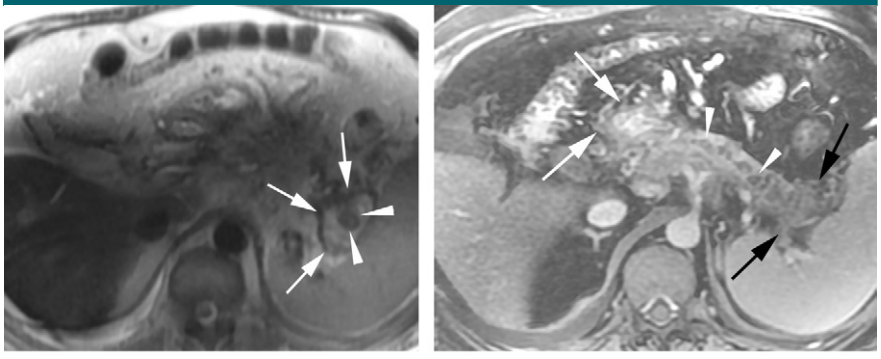
Figure 12



a. b.

Figure 12: Pseudocyst in a 61-year-old man. **(a)** Coronal CT reconstruction shows pseudocyst (arrows) next to body of the pancreas with a well-defined capsule. The patient complained of pain in the midabdomen to left upper quadrant and early satiety. **(b)** Follow-up coronal CT reconstruction was obtained after stent (arrow) had been placed endoscopically through the stomach into the sterile collection. There is no residual collection next to the pancreas.

Figure 13



a. b.

Figure 13: Sterile WON in a 45-year-old man with previous episodes of pancreatitis. MR imaging was performed because the patient had an allergy to iodinated contrast material. **(a)** T2-weighted MR image (1500/92.88) obtained 5 weeks after the acute episode shows encapsulated collection (arrows) near the splenic hilus and next to tail of the pancreas. The collection appears heterogeneous and contains nonliquefied material (arrowheads) and fluid and can be distinguished from a pseudocyst with fluid only. **(b)** Gadolinium-enhanced T1-weighted MR image (4.236/2.032) shows residual thickening of duodenum wall and periduodenal inflammation (white arrows), beaded pancreatic duct (arrowheads), and inflammation at tail of the pancreas (black arrows).

cases, percutaneous drainage has been performed after surgery that has been inadequate for cure, but this has been met with variable success (8,62).

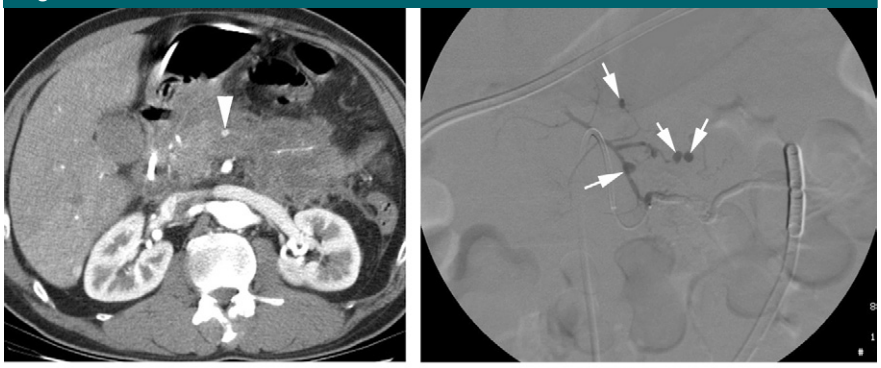
Treatment of Sterile Pancreatic Necrosis

The approaches to sterile and infected pancreatic necrosis are different. In patients suspected of having sterile pancreatic necrosis, CT is performed every 7–10 days to look at the evolution of the pancreatic necrosis and to assess evidence of infection (air bubbles) and complications such as increased peripancreatic necrotic collections or hemorrhage (65). Patients who do not do well clinically, who have clinical instability (eg, tachycardia, leukocytosis, fever, organ failure), and who do not show radiologic evidence of infection, 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

Figure 14



a. b.

Figure 14: Pseudoaneurysm in a 38-year-old woman with alcohol abuse and necrotizing pancreatitis. **(a)** 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 clearly identified in late arterial phase. **(b)** Angiogram obtained for embolization of the pseudoaneurysm seen on **a** shows four pseudoaneurysms (arrows), all of which were successfully treated with coil embolization.

common and carries with it high morbidity and mortality (66). Infected pancreatic necrosis is generally treated with surgical débridement and antibiotics. In patients with infected necrosis, tissue fragments that block the catheter often impair percutaneous catheter drainage. Nevertheless, if a patient is too unstable for surgery, percutaneous catheter drainage may help stabilize the patient by reducing the sepsis through removal of some infected material that is more liquefied (Fig 11). This then can act as a temporizing measure before surgery. The authors of one study described the step-up approach, which consisted of initial percutaneous drainage followed by surgical necrosectomy and then, in some cases, again by percutaneous catheter drainage (67). This approach resulted in fewer new-onset multiple-organ failures and fewer postsurgical complications, but the rate of death was not different, as compared with that for open necrosectomy. In some cases, percutaneous catheter drainage alone can be successful without surgery (57). Success requires careful attention to detail, including that all collections are drained. If collections persist after 2–3 days, additional catheters, often with larger bores, need to be placed. Vigorous catheter irrigation should be performed several times per day, and follow-up CT is needed to monitor treatment response. Recent data suggest that the ultimate outcome may depend more on the presence of multisystem organ failure than on the presence of infection (57).

In recent years, modified procedures have been described that consist of introduction of a percutaneous catheter into the necrotic pancreatic collection to serve as a guide and placement of a single large-port laparoscope along the drain track for necrosectomy with the ability to retrieve necrotic pancreatic tissue (68). Drains were placed at the end of the procedure to allow continuous postoperative lavage. In another study, percutaneous drainage was performed for several days, followed by expansion of the track and eventual introduction of a choledochoscope to retract infected necrotic pancreatic tissue through the sinus tract (69). These novel approaches can lead to a decrease in morbidity and mortality (51).

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.

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 in the first 4 weeks) and thereafter a WON, which has an enhancing capsule. All four types of collection

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 nonliquefied 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 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.

Acknowledgment: I thank David B. Franklin, JD, for critically reviewing and editing this manuscript.

Disclosures of Potential Conflicts of Interest: No potential conflicts of interest to disclose.

References

1. Bradley EL 3rd. A clinically based classification system for acute pancreatitis. Summary of the International Symposium on Acute Pancreatitis, Atlanta, Ga, September 11 through 13, 1992. Arch Surg 1993;128(5):586–590.
2. Bollen TL, Besselink MG, van Santvoort HC, Gooszen HG, van Leeuwen MS. Toward an update of the Atlanta classification on acute pancreatitis: review of new and abandoned terms. Pancreas 2007;35(2):107–113.
3. Vege SS, Chari ST. Organ failure as an indicator of severity of acute pancreatitis: time to revisit the Atlanta classification. Gastroenterology 2005;128(4):1133–1135.
4. Sarr MG, Banks PA, Bollen TL, et al. Revision of the Atlanta classification of acute pancreatitis. Acute Pancreatitis Classification Workgroup, April 2008. http://www.pancreasclub.com/resources/AtlantaClassification. Accessed April 8, 2011.
5. van Santvoort HC, Bollen TL, Besselink MG, et al. Describing peripancreatic collections in severe acute pancreatitis using morphologic terms: an international interobserver agreement study. Pancreatology 2008;8(6):593–599.
6. Nealon WH, Walser E. Surgical management of complications associated with percutaneous and/or endoscopic management of pseudocyst of the pancreas. Ann Surg 2005;241(6):948–957; discussion 957–960.
7. Takahashi N, Papachristou GI, Schmit GD, et al. CT findings of walled-off pancreatic necrosis (WOPN): differentiation from pseudocyst and prediction of outcome after endoscopic therapy. Eur Radiol 2008;18(11):2522–2529.
8. Banks PA, Freeman ML; Practice Parameters Committee of the American College of Gastroenterology. Practice guidelines in acute pancreatitis. Am J Gastroenterol 2006;101(10):2379–2400.
9. Balthazar EJ. Acute pancreatitis: assessment of severity with clinical and CT evaluation. Radiology 2002;223(3):603–613.
10. Buter A, Imrie CW, Carter CR, Evans S, McKay CJ. Dynamic nature of early organ dysfunction determines outcome in acute pancreatitis. Br J Surg 2002;89(3):298–302.
11. Johnson CD, Abu-Hilal M. Persistent organ failure during the first week as a marker of fatal outcome in acute pancreatitis. Gut 2004;53(9):1340–1344.
12. Besselink MG, van Santvoort HC, Witteman BJ, Gooszen HG; Dutch Acute Pancreatitis Study Group. Management of severe acute pancreatitis: it's all about timing. Curr Opin Crit Care 2007;13(2):200–206.
13. Marshall JC, Cook DJ, Christou NV, Bernard GR, Sprung CL, Sibbald WJ. Multiple organ dysfunction score: a reliable descriptor of a complex clinical outcome. Crit Care Med 1995;23(10):1638–1652.
14. Halonen KI, Pettilä V, Leppäniemi AK, Kemppainen EA, Puolakkainen PA, Haapiainen RK. Multiple organ dysfunction associated with severe acute pancreatitis. Crit Care Med 2002;30(6):1274–1279.
15. Whitcomb DC. Clinical practice. Acute pancreatitis. N Engl J Med 2006;354(20):2142–2150.
16. Lankisch PG, Pflichthofer D, Lehnick D. No strict correlation between necrosis and organ failure in acute pancreatitis. Pancreas 2000;20(3):319–322.
17. Malangoni MA, Martin AS. Outcome of severe acute pancreatitis. Am J Surg 2005;189(3):273–277.
18. Abu-Zidan FM, Bonham MJ, Windsor JA. Severity of acute pancreatitis: a multivariate analysis of oxidative stress markers and modified Glasgow criteria. Br J Surg 2000;87(8):1019–1023.
19. Dervenis C, Johnson CD, Bassi C, et al. Diagnosis, objective assessment of severity, and management of acute pancreatitis. Santorini consensus conference. Int J Pancreatol 1999;25(3):195–210.
20. Beger HG, Rau B, Isenmann R. Natural history of necrotizing pancreatitis. Pancreatology 2003;3(2):93–101.
21. Balthazar EJ. Staging of acute pancreatitis. Radiol Clin North Am 2002;40(6):1199–1209.
22. Mortele KJ, Ip IK, Wu BU, Conwell DL, Banks PA, Khorasani R. Acute pancreatitis: imaging utilization practices in an urban teaching hospital—analysis of trends with assessment of independent predictors in correlation with patient outcomes. Radiology 2011;258(1):174–181.
23. Mujica VR, Barkin JS, Go VL. Acute pancreatitis secondary to pancreatic carcinoma. Study Group Participants. Pancreas 2000;21(4):329–332.
24. Bollen TL, van Santvoort HC, Besselink MG, van Es WH, Gooszen HG, van Leeuwen MS. Update on acute pancreatitis: ultrasound, computed tomography, and magnetic resonance imaging features. Semin Ultrasound CT MR 2007;28(5):371–383.
25. Kiriyama S, Gabata T, Takada T, et al. New diagnostic criteria of acute pancreatitis. J Hepatobiliary Pancreat Sci 2010;17(1):24–36.
26. Papanikolaou IS, Adler A, Neumann U, Neuhaus P, Rösch T. Endoscopic ultrasound in pancreatic disease—its influence on surgical decision-making. An update 2008. Pancreatology 2009;9(1-2):55–65.
27. 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(5):1051–1057.
28. Xiao B, Zhang XM, Tang W, Zeng NL, Zhai ZH. Magnetic resonance imaging for local complications of acute pancreatitis: a pictorial review. World J Gastroenterol 2010;16(22):2735–2742.
29. Hirota M, Kimura Y, Ishiko T, Beppu T, Yamashita Y, Ogawa M. Visualization of the heterogeneous internal structure of so-called "pancreatic necrosis" by magnetic resonance imaging in acute necrotizing pancreatitis. Pancreas 2002;25(1):63–67.
30. Kim YK, Ko SW, Kim CS, Hwang SB. Effectiveness of MR imaging for diagnosing the mild forms of acute pancreatitis: comparison with MDCT. J Magn Reson Imaging 2006;24(6):1342–1349.
31. Swaroop VS, Chari ST, Clain JE. Severe acute pancreatitis. JAMA 2004;291(23):2865–2868.

32. Rickes S, Treiber G, Mönkemüller K, et al. Impact of the operator's experience on value of high-resolution transabdominal ultrasound in the diagnosis of choledocholithiasis: a prospective comparison using endoscopic retrograde cholangiography as the gold standard. Scand J Gastroenterol 2006;41(7):838–843.

33. De Waele JJ, Delrue L, Hoste EA, De Vos M, Duyck P, Colardyn FA. 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(2):185–190.

34. Spitzer AL, Thoeni RF, Barcia AM, Schell MT, Harris HW. Early nonenhanced abdominal computed tomography can predict mortality in severe acute pancreatitis. J Gastrointest Surg 2005;9(7):928–933.

35. 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(2):386–392.

36. Sakorafas GH, Tsiotos GG, Sarr MG. Extrapancreatic necrotizing pancreatitis with viable pancreas: a previously under-appreciated entity. J Am Coll Surg 1999;188(6):643–648.

37. Balthazar EJ, Robinson DL, Megibow AJ, Ranson JH. Acute pancreatitis: value of CT in establishing prognosis. Radiology 1990;174(2):331–336.

38. Ashley SW, Perez A, Pierce EA, et al. Necrotizing pancreatitis: contemporary analysis of 99 consecutive cases. Ann Surg 2001;234(4):572–579; discussion 579–580.

39. Bruennler T, Hamer OW, Lang S, et al. Outcome in a large unselected series of patients with acute pancreatitis. Hepatogastroenterology 2009;56(91-92):871–876.

40. 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(4):577–582.

41. Memiş A, Parildar M. Interventional radiological treatment in complications of pancreatitis. Eur J Radiol 2002;43(3):219–228.

42. Desser TS, Sommer FG, Jeffrey RB Jr. Value of curved planar reformations in MDCT of abdominal pathology. AJR Am J Roentgenol 2004;182(6):1477–1484.

43. Sahani DV, Kadavigere R, Blake M, Fernandez-Del Castillo C, Lauwers GY, Hahn PF. Intraductal papillary mucinous neoplasm of pancreas: multi-detector row CT with 2D curved reformations—correlation with MRCP. Radiology 2006;238(2):560–569.

44. Pelaez-Luna M, Vege SS, Petersen BT, et al. Disconnected pancreatic duct syndrome in severe acute pancreatitis: clinical and imaging characteristics and outcomes in a cohort of 31 cases. Gastrointest Endosc 2008;68(1):91–97.

45. Harris HW, Barcia A, Schell MT, Thoeni RF, Schecter WP. Necrotizing pancreatitis: a surgical approach independent of documented infection. HPB (Oxford) 2004;6(3):161–168.

46. Vege SS, Fletcher JG, Talukdar R, Sarr MG. Peripancreatic collections in acute pancreatitis: correlation between computerized tomography and operative findings. World J Gastroenterol 2010;16(34):4291–4296.

47. 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(5):697–702.

48. Büchler MW, Gloor B, Müller CA, Friess H, Seiler CA, Uhl W. Acute necrotizing pancreatitis: treatment strategy according to the status of infection. Ann Surg 2000;232(5):619–626.

49. Balthazar EJ, Freeny PC, vanSonnenberg E. Imaging and intervention in acute pancreatitis. Radiology 1994;193(2):297–306.

50. Johnson MD, Walsh RM, Henderson JM, et al. Surgical versus nonsurgical management of pancreatic pseudocysts. J Clin Gastroenterol 2009;43(6):586–590.

51. Vosoghi M, Sial S, Garrett B, et al. EUS-guided pancreatic pseudocyst drainage: review and experience at Harbor-UCLA Medical Center. MedGenMed 2002;4(3):2.

52. Habashi S, Draganov PV. Pancreatic pseudocyst. World J Gastroenterol 2009;15(1):38–47.

53. Neff R. Pancreatic pseudocysts and fluid collections: percutaneous approaches. Surg Clin North Am 2001;81(2):399–403, xii.

54. Curry L, Sookur P, Low D, Bhattacharya S, Fotheringham T. Percutaneous cystgastrostomy as a single-step procedure. Cardiovasc Intervent Radiol 2009;32(2):289–295.

55. Seewald S, Ang TL, Teng KC, Soehendra N. EUS-guided drainage of pancreatic pseudocysts, abscesses and infected necrosis. Dig Endosc 2009;21(Suppl 1):S61–S65.

56. Bharwani N, Patel S, Prabhudesai S, Fotheringham T, Power N. Acute pancreatitis: the role of imaging in diagnosis and management. Clin Radiol 2011;66(2):164–175.

57. Mortelé 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(1):110–116.

58. Mier J, León EL, Castillo A, Robledo F, Blanco R. Early versus late necrosectomy in severe necrotizing pancreatitis. Am J Surg 1997;173(2):71–75.

59. Rodriguez JR, Razo AO, Targarona J, et al. Debridement and closed packing for sterile or infected necrotizing pancreatitis: insights into indications and outcomes in 167 patients. Ann Surg 2008;247(2):294–299.

60. Freeny PC, Hauptmann E, Althaus SJ, Traverso LW, Sinanan M. Percutaneous CT-guided catheter drainage of infected acute necrotizing pancreatitis: techniques and results. AJR Am J Roentgenol 1998;170(4):969–975.

61. Segal D, Mortele KJ, Banks PA, Silverman SG. Acute necrotizing pancreatitis: role of CT-guided percutaneous catheter drainage. Abdom Imaging 2007;32(3):351–361.

62. Shankar S, vanSonnenberg E, Silverman SG, Tuncali K, Banks PA. Imaging and percutaneous management of acute complicated pancreatitis. Cardiovasc Intervent Radiol 2004;27(6):567–580.

63. Schneider L, Büchler MW, Werner J. Acute pancreatitis with an emphasis on infection. Infect Dis Clin North Am 2010;24(4):921–941, viii.

64. van Baal MC, van Santvoort HC, Bollen TL, et al. Systematic review of percutaneous catheter drainage as primary treatment for necrotizing pancreatitis. Br J Surg 2011;98(1):18–27.

65. Mortele KJ, Banks PA, Silverman SG. State-of-the-art imaging of acute pancreatitis. JBR-BTR 2003;86(4):193–208.

66. Perez A, Whang EE, Brooks DC, et al. Is severity of necrotizing pancreatitis increased in extended necrosis and infected necrosis? Pancreas 2002;25(3):229–233.

67. van Santvoort HC, Besselink MG, Bakker OJ, et al. A step-up approach or open necrosectomy for necrotizing pancreatitis. N Engl J Med 2010;362(16):1491–1502.

68. Bucher P, Pugin F, Morel P. Minimally invasive necrosectomy for infected necrotizing pancreatitis. Pancreas 2008;36(2):113–119.

69. Tang LJ, Wang T, Cui JF, et al. Percutaneous catheter drainage in combination with choledochoscope-guided debridement in treatment of peripancreatic infection. World J Gastroenterol 2010;16(4):513–517.

70. Windsor AC, Kanwar S, Li AG, et al. Compared with parenteral nutrition, enteral feeding attenuates the acute phase response and improves disease severity in acute pancreatitis. Gut 1998;42(3):431–435.

71. Petrov MS, Kukosh MV, Emelyanov NV. A randomized controlled trial of enteral versus parenteral feeding in patients with predicted severe acute pancreatitis shows a significant reduction in mortality and in infected pancreatic complications with total enteral nutrition. Dig Surg 2006;23(5-6):336–344; discussion 344–345.

72. 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(4):966–971.