REVIEW ARTICLE

2012 revision of the Atlanta Classification of acute pancreatitis

Michael G. Sarr

Department of Surgery, Mayo Clinic, Rochester, Minnesota, United States

KEY WORDS

acute pancreatitis, classification, severity

ABSTRACT

Recently, the original Atlanta classification of 1992 was revised and updated by the Working Group using a web-based consultative process involving multiple international pancreatic societies. The new understanding of the disease, its natural history, and objective description and classification of pancreatic and peripancreatic fluid collections make this new 2012 classification a potentially valuable means of international communication and interest. This revised classification identifies 2 phases of acute pancreatitis – early (first 1 or 2 weeks) and late (thereafter). Acute pancreatitis can be either edematous interstitial pancreatitis or necrotizing pancreatitis, the latter involving necrosis of the pancreatic parenchyma and peripancreatic tissues (most common), pancreatic parenchyma alone (least common), or just the peripancreatic tissues (~20%). Severity of the disease is categorized into 3 levels: mild, moderately severe, and severe. Mild acute pancreatitis lacks both organ failure (as classified by the modified Marshal scoring system) and local or systemic complications. Moderately severe acute pancreatitis has transient organ failure (organ failure of <2 days), local complications, and/or exacerbation of coexistent disease. Severe acute pancreatitis is defined by the presence of persistent organ failure (organ failure that persists for ≥2 days). Local complications are defined by objective criteria based primarily on contrast-enhanced computed tomography; these local complications are classified as acute peripancreatic fluid collections, pseudocyst (which are very rare in acute pancreatitis), acute (pancreatic/peripancreatic) necrotic collection, and walled-off necrosis. This classification will help the clinician to predict the outcome of patients with acute pancreatitis and will allow comparison of patients and disease treatment/management across countries and practices.

Introduction The original Atlanta Classification of acute pancreatitis was derived over 20 years ago.[1] It attempted to provide a common terminology and to define the severity of the disease to provide physicians with a uniform classification. Unfortunately, the actual classification, as written by the Atlanta Conference, has not been accepted or utilized universally.[2] In addition, our understanding of the etiopathogenesis, natural history, various markers of severity, and equally important, the features of cross-sectional imaging have led to confusing and imprecisely used terms. Indeed, there is no common terminology for the disease, its severity, and the pancreatic and peripancreatic fluid collections, which leads to confusion when comparing various institutional experiences, outcomes, therapies, etc. An example of this confusion is the use of the term "pseudocyst", which has many different parochial meanings to pancreatologists, surgeons, radiologists, and pathologists.

The revised Atlanta Classification utilized a new technique of a global, web-based "virtual" consensus conference over the Internet.[3] Although the concept was novel, the global, web-based consensus was only partially successful. The Working Group (Drs. M.G. Sarr, P.A. Banks, and S.S. Vege [United States], H.G. Gooszen and T.L. Bollen [The Netherlands], C.D. Johnson [England], and G.G. Tsiotos and C. Dervenis [Greece]) collated the evidence-based literature whenever available to construct a new classification based on the 2 phases of the natural history of the disease (the first 1 or 2 weeks and the next several weeks/months that follow), and then asked for a global input and suggestions from multiple groups

Correspondence to:
Michael G. Sarr, MD, James C. Masson Professor of Surgery, Department of Surgery, Mayo Clinic, 200 First Street SW, Rochester, MN, USA 55 905,
phone: +1-507-255-5713,
fax: +1-507-255-6318,
e-mail: sarr.michael@mayo.edu
Received: January 11, 2013.
Accepted: January 12, 2013.
Published online: January 25, 2013.
Conflict of interest: none declared.
Pol Arch Med Wewn. 2013; 123 (3): 118-124

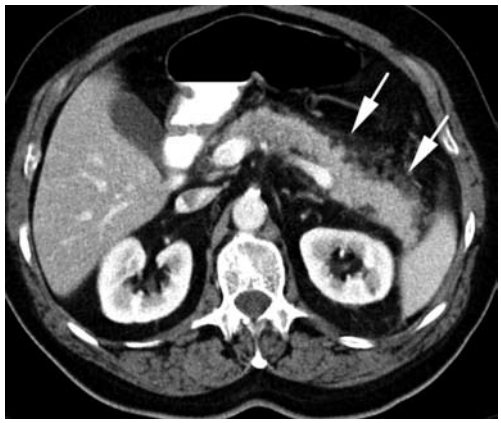

FIGURE 1 Acute interstitial edematous pancreatitis; the pancreas enhances completely; arrows show peripancreatic stranding; reprinted with permission from Sarr et al.[4]

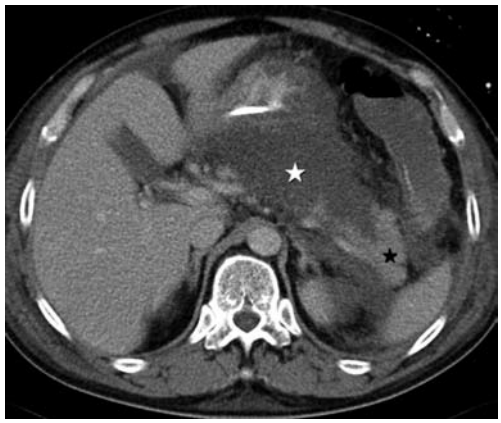

FIGURE 2 Acute necrotizing pancreatitis; extensive nonenhancement of the pancreatic body (white star); normal enhancement of pancreatic tail (black star); reprinted with permission from Sarr et al.[4]

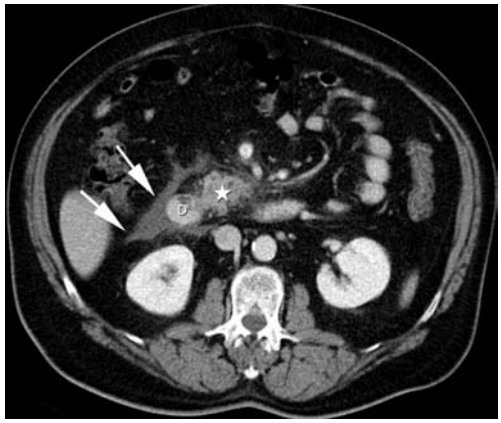

FIGURE 3 Acute interstitial edematous pancreatitis with an acute peripancreatic fluid collection without an encapsulating wall (white arrows); normal enhancement of the pancreatic head (white star); reprinted with permission from Sarr et al.[4]

and individuals via an iterative, web-based process. Helpful and insightful input from pancreatologists of many different disciplines (surgery, gastroenterology, diagnostic and interventional radiology, gastrointestinal endoscopy, pathology, and acute care medicine/surgery) around the world helped to define or refine the development of this new classification. This new classification addresses the diagnosis, types of acute pancreatitis, severity, and definition of pancreatic and peripancreatic collections. The classification will be summarized below.[4]

Diagnosis The diagnosis of acute pancreatitis involves a combination of symptoms, physical examination, and focused laboratory values and requires 2 of the following 3 features: 1) upper abdominal pain of acute onset often radiating through to the back, 2) serum amylase or lipase activity greater than 3 times normal, and 3) findings on cross-sectional abdominal imaging consistent with acute pancreatitis. We stress that not every patient requires pancreatic imaging to make the diagnosis, provided the clinical picture is that of acute pancreatitis.

Types of acute pancreatitis There are 2 different forms of acute pancreatitis: interstitial edematous pancreatitis and necrotizing pancreatitis.

Interstitial edematous pancreatitis The majority of patients with acute pancreatitis (80%–90%) have interstitial edematous pancreatitis, which is a milder form. Acute interstitial edematous pancreatitis lacks pancreatic parenchymal necrosis or peripancreatic necrosis on imaging. Usually diffuse (or on rare occasions localized) enlargement of the pancreas secondary to inflammatory edema is evident (FIGURE 1); there may also be peripancreatic fluid (see "Pancreatic and peripancreatic fluid collections"). While there may be haziness and stranding in the pancreatic parenchyma and peripancreatic tissues secondary to inflammatory edema, no necrosis is evident on cross-sectional imaging. This form of acute pancreatitis usually resolves quickly within a week.

Necrotizing pancreatitis The presence of tissue necrosis, either of the pancreatic parenchyma or the peripancreatic tissues, defines necrotizing pancreatitis. This more aggressive form of acute pancreatitis most commonly involves both the pancreatic parenchyma and the peripancreatic tissue (FIGURE 2), just the peripancreatic tissue alone (FIGURE 3), or rarely the necrosis is limited only to the pancreatic parenchyma. Thus, necrotizing pancreatitis should be classified as pancreatic parenchymal and peripancreatic necrosis, peripancreatic necrosis alone, or pancreatic necrosis alone. The presence of pancreatic parenchyma necrosis is a more severe disease compared with peripancreatic necrosis alone.[5,6]

The differentiation of "necrosis" can be difficult on contrast-enhanced computed tomography (CECT) early in the disease (first week). Nonperfusion of the pancreatic parenchyma is evident, but in the peripancreatic region, loss of "perfusion" of the retroperitoneal fat is not evident several days to a week later. The eventual diagnosis of peripancreatic necrosis becomes evident because of local inflammatory changes with associated fluid and a solid component, which appears as a heterogenous collection of both solid and liquid components.

Infection Necrotizing pancreatitis should be classified as either infected or sterile; interstitial edematous pancreatitis does not become infected. Infection, diagnosed based on ongoing signs of sepsis and/or the combination of clinical signs, is rare in the first 1 or 2 weeks of necrotizing pancreatitis.[7,8] CT findings of extraluminal gas within the areas of necrosis in the pancreatic

TABLE 1 Modified Marshall Scoring System[a]; modified from Banks et al.[3]

Organ system	Score				
	0	1	2	3	4
respiratory (PaO_2/FIO_2)	>400	301–400	201–300	101–200	≤101
renal[b] (serum creatinine, μmol/l)	≤134	134–169	170–310	311–439	>439
cardiovascular (systolic blood pressure, mmHg)	>90	<90 responsive to fluid resuscitation	<90 not responsive to fluid resuscitation	<90 pH <7.3	<90 pH <7.2

a a score of ≥2 in any one organ system defines "organ failure"
b scoring patients with pre-existent chronic renal failure depends on the extent of deterioration over baseline renal function; calculations for baseline serum creatinine >134 μmol/l or >1.4 mg/dl are not available
Off inotropic support

and/or peripancreatic tissues are often seen with infected necrosis. Percutaneous fine-needle aspiration will make the diagnosis when both bacteria and/or fungi are seen on gram stain and the culture is positive. Infection may also occur as a secondary event after percutaneous, endoscopic, or operative intervention and is associated with an increased mortality and morbidity.[9]

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

To allow an international classification appropriate for different practices and geographic locations, a readily usable, objective definition of severity is imperative. This new classification defines 3 degrees of severity: mild, moderately severe, and severe acute pancreatitis. These definitions of severity are based on the presence or absence of persistent organ failure and local and systemic complications (see below). Mild acute pancreatitis usually resolves within several days to a week; moderately severe acute pancreatitis resolves more slowly, may require interventions, and prolongs hospitalization; severe acute pancreatitis obligates a longer hospital stay, usually some form of intervention, and can also be associated with multiple organ failure and death.

Definition of organ failure (persistent or transient) The most reliable marker for disease severity in acute pancreatitis is persistent organ failure for longer than 48 hours.[8,9] After review of the literature, this new classification of acute pancreatitis uses the Modified Marshall Scoring System,[10] a universally applicable scoring system that does not require unique assays or advanced critical-care monitoring; more importantly, this scoring system stratifies disease severity easily and objectively.[11-13] The Modified Marshall System evaluates the 3 organ systems most commonly affected by severe acute pancreatitis: respiratory, cardiovascular, and renal (TABLE 1). Persistent organ failure is defined objectively as a score of 2 or more for longer than 48 hours for 1 (or more) of the 3 organ systems. Transient organ failure is also important in the classification of moderately severe acute pancreatitis and involves a score of 2 or more for 1 (or more) of the 3 organ systems that occurs but is present for longer than 48 hours. The Modified Marshall Scoring System can be re-evaluated during the course of the disease to reclassify severity.

Local complications Unlike in the 1991 Atlanta Classification, the natural history, consequences, and definition of pancreatic and peripancreatic collections are now better understood and defined objectively as acute peripancreatic fluid collections (APFCs), pancreatic pseudocysts, acute necrotic collections (ANCs), and walled-off necrosis (WON) (see "Pancreatic and peripancreatic fluid collections"). Other complications include colonic necrosis, splenic/portal vein thrombosis, and gastric outlet dysfunction. These local complications delay hospital discharge or require intervention but do not necessarily cause death, hence the definition of moderately severe acute pancreatitis. Persistence of abdominal pain, secondary increases in serum amylase/lipase activity, organ failure, or fever/chills usually prompt imaging to search for these complications.

Systemic complications Renal, circulatory, or respiratory organ failure or exacerbation of serious pre-existing comorbidities related directly to acute pancreatitis are examples of systemic complications related to the systemic inflammatory response syndrome (SIRS) that accompanies acute pancreatitis. Examples include exacerbation of underlying heart disease (coronary artery disease or congestive heart failure), chronic diabetes, obstructive lung disease, chronic liver disease, etc.

Phases of acute pancreatitis In general, there are 2 phases of acute pancreatitis which overlap one another – the early phase and the late phase.

During the early phase, which lasts only a week or so, the systemic manifestations are related to the host response to the cytokine cascade, which manifests as SIRS[14] and/or the compensatory anti-inflammatory syndrome (CARS) that can

TABLE 2 Degrees of severity of acute pancreatitis; modified from Banks et al.[3]

mild acute pancreatitis
lack of organ failure and local/systemic complications
moderately severe acute pancreatitis
transient organ failure – organ failure that resolves within 48 hours and/or
local or systemic complications
severe acute pancreatitis
persistent single or multiple organ failure (>48 hours)

TABLE 3 Terms used in the new classification based on contrast-enhanced computed tomography[a]; modified from Banks et al.[3]

interstitial edematous pancreatitis: "inflammation" or stranding in the pancreatic and/or peripancreatic tissues without tissue necrosis
CECT criteria
– pancreatic parenchyma enhances with the contrast agent
– lack of peripancreatic necrosis
necrotizing pancreatitis: pancreatic parenchymal necrosis and/or peripancreatic necrosis
CECT criteria
– pancreatic parenchymal areas without enhancement by intravenous contrast agent and/or
– peripancreatic necrosis (see below – acute necrotic collection and walled-off necrosis)
acute peripancreatic fluid collection: peripancreatic fluid occurring in the setting of interstitial edematous pancreatitis; this peripancreatic fluid occurs within the first 4 weeks of interstitial edematous pancreatitis
CECT criteria
– homogeneous fluid adjacent to pancreas confined by peripancreatic fascial planes
– no recognizable wall
pancreatic pseudocyst: an encapsulated, well-defined collection of fluid but no or minimal solid components which occurs >4 weeks after onset of interstitial edematous pancreatitis.
CECT criteria
– well-circumscribed, homogeneous, round or oval fluid collection
– no solid component
– well-defined wall
– occurs only in interstitial edematous pancreatitis
acute necrotic collection: a collection of both fluid and solid components (necrosis) occurring during necrotizing pancreatitis. This collection can involve the pancreatic and/or the peripancreatic tissues
CECT criteria
– heterogeneous, varying of non-liquid density
– no encapsulating wall
– intrapancreatic and/or extrapancreatic
walled-off necrosis: a mature, encapsulated acute necrotic collection with a well-defined inflammatory wall; these tend to mature >4 weeks after onset of necrotizing pancreatitis.
CECT criteria
– heterogenous liquid and non-liquid density
– well-defined wall
– intrapancreatic and/or extrapancreatic

Abbreviations: APFC – acute peripancreatic fluid collection, CECT – contrast-enhanced computed tomography

predispose to infection.[15] When SIRS or CARS persist, organ failure becomes much more likely. As described above, severity of acute pancreatitis is defined by the presence and duration of organ failure – transient (<48-hour duration) and persistent (>48-hour duration). When organ failure involves more than 1 organ, the terms of multiple organ failure or multiple organ dysfunction syndrome should be used.

The late phase of acute pancreatitis, which can persist for weeks to months, is characterized by systemic signs of ongoing inflammation, local and systemic complications, and/or by transient or persistent organ failure. This late phase of acute pancreatitis defines moderately severe or severe acute pancreatitis.

Definition of severity Early recognition of disease severity is important to identify patients on admission or during the first 24 to 48 hours who will require aggressive resuscitation/treatment. These patients should be treated in an intensive care unit or transferred to a high-acuity care hospital. Although the definition of severity will not be able to be made definitively in the first 48 hours, patients with SIRS should be treated aggressively as if they had severe acute pancreatitis.

This new classification defines 3 degrees of severity according to the morbidity and mortality of the disease: mild, moderately severe, and severe acute pancreatitis (TABLE 2).

Mild acute pancreatitis lacks organ failure or local or systemic complications. Pancreatitis resolves rapidly, mortality is rare, pancreatic imaging is often not required, and patients are usually discharged in the first week.

Moderately severe acute pancreatitis has transient organ failure, local complications, and/or systemic complications but not persistent (>48 hour) organ failure. The morbidity is increased as is mortality (<8%) compared with that of mild acute pancreatitis, but is not as great as in severe acute pancreatitis.[16] Patients may be discharged within the second or third week or may require prolonged hospitalization because of the local or systemic complications.

Severe acute pancreatitis is defined by persistent organ failure, either early or late in the disease, and patients usually have 1 or more local and/or systemic complications. Patients with severe acute pancreatitis that develops within the early phase are at a markedly increased risk (36%–50%) of death.[10,17,18] Later development of infected necrosis carries an extremely high mortality.[9,19]

While other groups have suggested a 2-tier or 4-tier classification of severity,[9,20,21] this new classification is easier, classifies the 3 tiers of severity (as defined above), and is more appropriate even from a patient's view.

Pancreatic and peripancreatic fluid collections A substantial problem in the past has been the multitude of terms used to describe the pancreatic and peripancreatic fluid collections seen on cross-sectional imaging. The new classification stresses distinction between "collections" consisting of fluid alone vs. those "collections" that arise from necrosis of pancreatic parenchyma and/or peripancreatic tissues; the latter contain

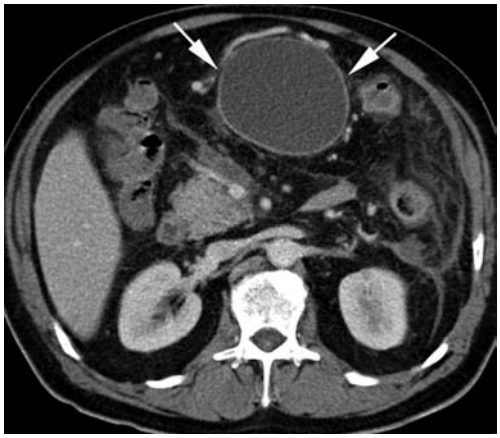

FIGURE 4
A pseudocyst 5 weeks after onset of acute interstitial edematous pancreatitis; the round, homogeneous fluid collection has a well-defined enhancing rim (white arrows point at the borders of the pseudocyst); note absence of areas of greater attenuation representing a non-liquid component; reprinted with permission from Sarr et al.[4]

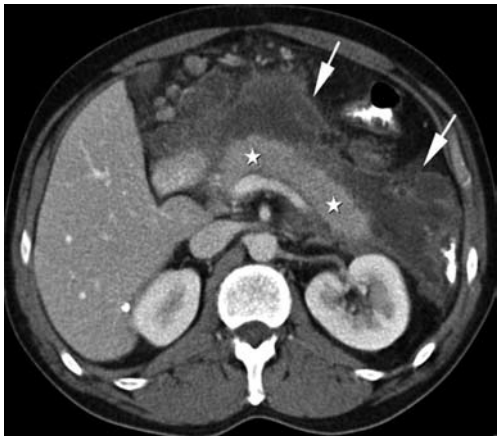

FIGURE 5 Acute necrotic collection (ANC) of the peripancreatic tissues; entire pancreatic parenchyma enhances (white stars) heterogeneous, nonliquid peripancreatic components in retroperitoneum and colonic mesentery (white arrows point at borders of the ANC); reprinted with permission from Sarr et al.[4]

both a solid component and, as the necrotic process evolves, varying degrees of a fluid component (TABLE 3), leading to heterogenous collections that are not solely "fluid," but contain areas of necrosis.

Acute peripancreatic fluid collection APFCs are true fluid collections that develop in the early phase of interstitial edematous acute pancreatitis. APFCs lack a wall but are confined by the fascial planes of the retroperitoneum (FIGURE 3). APFCs remain sterile, usually resolve without intervention, and are not associated with necrotizing pancreatitis.[22,23] APFCs that persist for longer than 4 weeks likely develop into a pancreatic pseudocyst (see below), although the development of a true pseudocyst (i.e., a persistent fluid collection contained by a defined wall and containing little if any solid component) is rare in acute pancreatitis.

Pancreatic pseudocyst This should be a very specific term describing a peripancreatic or, much less commonly, intrapancreatic fluid collection surrounded by a well-defined wall that contains no solid component (FIGURE 4). The term "pancreatic pseudocyst" has been misused repeatedly throughout the literature and in daily dialogue by non-pancreatologists and pancreatologists alike. In fact, the term is often used inappropriately to describe most all peripancreatic collections in the setting of acute pancreatitis. This new classification is designed to stress the importance of defining pancreatic and peripancreatic collections precisely and objectively. The presumed pathogenesis of a true pancreatic pseudocyst occurs from focal disruption of the pancreatic ductal system in the absence of recognizable pancreatic or peripancreatic necrosis on imaging. Magnetic resonance imaging (MRI) or ultrasonography may be necessary to support this diagnosis by showing the absence of solid material within the fluid collection.

The disconnected duct syndrome is a special situation that can lead to a pancreatic pseudocyst in patients with necrotizing pancreatitis.[24] This true fluid collection occurs when necrosis of the neck/proximal body of the pancreas isolates a still viable, secreting distal pancreatic remnant. This usually develops weeks after necrosectomy because of localized leakage of the disconnected duct into the necrosectomy cavity.

Acute necrotic collection ANCs are present in the first 4 weeks of the disease. They contain variable amounts of fluid and solid (necrotic) material secondary to pancreatic and/or peripancreatic necrosis (FIGURE 5). On CT imaging, ANCs may look like an APFC in the first week of acute pancreatitis, but the fluid and solid components become more evident as the necrosis matures. Other imaging procedures, such as MRI or ultrasonography, may image the solid component better. ANCs most commonly involve both the pancreatic parenchyma and the peripancreatic tissues, but less commonly can involve the pancreatic parenchyma alone or the peripancreatic tissue alone. ANCs are either sterile or infected; also, because they may be associated with a disrupted pancreatic duct, leakage of pancreatic juice into the collection is not uncommon, but an ANC is not a pancreatic pseudocyst, because ANCs contain solid material related to tissue necrosis. This distinction is quite important and represents a different pathogenesis.

Walled-off necrosis WON represents the mature phase of an ANC. WON consists of varying amounts of liquid and solid material surrounded by a mature, enhancing wall of reactive tissue (FIGURE 6). WON develops usually 4 or more weeks after the onset of necrotizing acute pancreatitis. The terms used previously have included organized pancreatic necrosis, necroma, pancreatic sequestrum, pancreatic pseudocyst with necrosis, and subacute pancreatic necrosis, but the term WON should be a common, consistent terminology.

As with ANCs, WON may be multiple, occur at sites distant from the pancreas, and have a pancreatic ductal communication; the latter is not necessary in this classification but is of potential clinical importance, because any ductal communication may affect management.

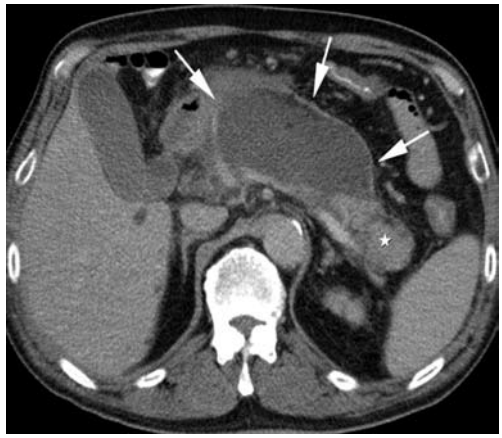

FIGURE 6 Walled-off necrosis (WON) after acute necrotizing pancreatitis; the heterogeneous, encapsulated collection is present in the pancreatic and peripancreatic area; white arrows show borders of the WON); white star shows normal enhancement of the pancreatic tail; reprinted with permission from Sarr et al.[4]

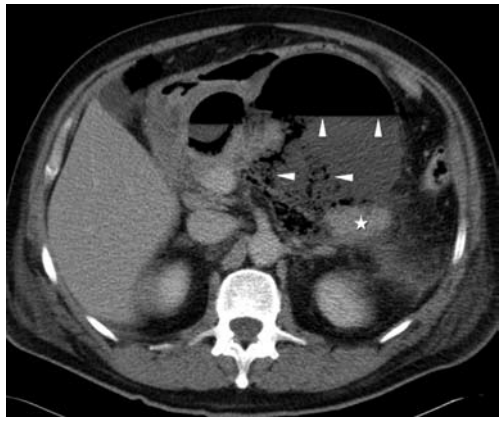

FIGURE 7 Infected pancreatic necrosis; note the large acute necrotic collection in the pancreatic/peripancreatic area with gas bubbles (horizontal white arrowheads) and gas fluid level (vertical white arrowheads); the pancreatic tail (white star) enhances normally; reprinted with permission from Sarr et al.[4]

Sterile vs. infected necrosis Both ANCs or WON can be sterile or infected (infected necrosis). The clinical course of the patient (fever, leukocytosis, tachycardia) or the extraluminal gas within the areas of necrosis evident on CECT (FIGURE 7) usually herald the onset of infection.

Summary This new and markedly different revision of the original 1991 Atlanta Classification of acute pancreatitis should standardize the terminology used in describing acute pancreatitis and its complications. Although not necessarily commissioned by any one society, this new classification of acute pancreatitis received input and support in principle by the American Pancreatic Society, International Association of Pancreatology, European Pancreatic Club, pancreas section of the American Gastroenterological Association, Society for Surgery of the Alimentary Tract, the Pancreas Club, and several other international societies and associations interested in pancreatic disorders.

This classification incorporated new insights into the disease learned over the last 20 years, especially the concept that acute pancreatitis and its complications involve a dynamic process involving 2 phases – early and late. The accurate description of 2 types of acute pancreatitis (interstitial edematous pancreatitis and necrotizing pancreatitis), definition of severity, and the description of local fluid and solid pancreatic and peripancreatic collections based on the characteristics of fluid and necrosis will improve the communication between different groups and practices worldwide. By adopting a consistent and objective common terminology, the advancement of our understanding of new treatments of acute pancreatitis will be facilitated.

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: 586-590.

2 Bollen TL, van Santvoort HC, Besselink MG, et al.; Dutch Acute Pancreatitis Study Group. The Atlanta Classification of acute pancreatitis revisited. Br J Surg. 2008; 95: 6-21.

3 Banks PA, Bollen TL, Dervenis C, et al.; Acute Pancreatitis Classification Working Group. Classification of acute pancreatitis – 2012: revision of the Atlanta classification and definitions by international consensus. Gut. 2012; 62: 102-111.

4 Sarr MG, Banks PA, Bollen TL, et al. The new revised classification of acute pancreatitis 2012. Surg Clin N Am. In press.

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

6 Bakker OJ, van Santvoort HC, Besselink MG, et al.; Dutch Pancreatitis Study Group. Extrapancreatic necrosis without pancreatic parenchymal necrosis: a separate entity in necrotizing pancreatitis? Gut. 2012 Jul 7. [Epub ahead of print].

7 Besselink MG, van Santvoort HC, Boermeester MA, et al.; Dutch Acute Pancreatitis Study Group. Timing and impact of infections in acute pancreatitis. Br J Surg. 2009; 96: 267-273.

8 van Santvoort HC, Besselink MG, Bakker OJ, et al.; Dutch Pancreatitis Study Group. A step-up approach or open necrosectomy for necrotizing pancreatitis (PANTER trial). N Engl J Med. 2010; 362: 1491-1502.

9 Petrov MS, Shanbhag S, Chakraborty M, et al. Organ failure and infection of pancreatic necrosis as determinants of mortality in patients with acute pancreatitis. Gastroenterology. 2010; 139: 813-820.

10 Marshall JC, Cook DJ, Christou NV, et al. Multiple organ dysfunction score: a reliable descriptor of a complex clinical outcome. Crit Care Med. 1995; 23: 1638-1652.

11 Mofidi R, Duff MD, Wigmore SJ, et al. Association between early systemic inflammatory response, severity of multiorgan dysfunction and death in acute pancreatitis. Br J Surg. 2006; 93: 738-744.

12 Johnson CD, Abu-Hilal M. Persistent organ failure during the first week as a marker of fatal outcome in acute pancreatitis. Gut. 2004; 53: 1340-1344.

13 UK guidelines for the management of acute pancreatitis. Gut. 2005; 54 Suppl 3: iii1-iii9.

14 Muckart DJ, Bhagwanjee S. American College of Chest Physicians/Society of Critical Care Medicine Consensus Conference definitions of the systemic inflammatory response syndrome and allied disorders in relation to critically injured patients. Crit Care Med. 1997; 25: 1789-1795.

15 Cobb JP, O'Keefe GE. Injury research in the genomic era. Lancet. 2004; 363: 2076-2083.

16 Vege SS, Gardner TB, Chari ST, et al. Low mortality and high morbidity in severe acute pancreatitis without organ failure: a case for revising the Atlanta classification to include "moderately severe acute pancreatitis". Am J Gastroenterol. 2009; 104: 710-715.

17 Buter A, Imrie CW, Carter CR, et al. Dynamic nature of early organ dysfunction determines outcome in acute pancreatitis. Br J Surg. 2002; 89: 293-302.

18 Mofidi R, Duff MD, Wigmore SJ, et al. Association between early systemic inflammatory response, severity of multiorgan dysfunction and death in acute pancreatitis. Br J Surg. 2006; 93: 738-744.

19 van Santvoort HC, Bakker OJ, Bollen TL, et al.; Dutch Pancreatitis Study Group. A conservative and minimally invasive approach to necrotizing pancreatitis improves outcome. Gastroenterology. 2011; 141: 1254-1263.

20 Petrov MS, Windsor JA. Classification of the severity of acute pancreatitis: how many categories make sense? Am J Gastroenterol. 2010; 105: 74-76.

21 Dellinger EP, Forsmark CE, Layer P, et al.; Pancreatitis Across Nations Clinical Research and Education Allisance (PANCREA). Determinant-based classification of acute pancreatitis severity: an international multidisciplinary consultation. Ann Surg. 2012; 256: 875-880.

22 Balthazar EJ, Robinson DL, Megibow AJ, Ranson JH. Acute pancreatitis: value of CT in establishing prognosis. Radiology. 1990; 174: 331-336.

23 Lenhart DK, Balthazar EJ. MDCT of acute mild (nonnecrotizing pancreatitis): abdominal complications and fate of fluid collections. AJR Am J Roentgenol. 2008; 190: 643-649.

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

ARTYKUŁ POGLĄDOWY

Ostre zapalenie trzustki – rewizja 2012

Klasyfikacja z Atlanty

Michael G. Sarr

Department of Surgery, Mayo Clinic, Rochester, Minnesota, Stany Zjednoczone

SŁOWA KLUCZOWE

ciężkość, klasyfikacja, ostre zapalenie trzustki

STRESZCZENIE

Klasyfikacja ostrego zapalenia trzustki opracowana pierwotnie w 1992 r. w Atlancie została ostatnio przez specjalnie powołaną grupę roboczą poddana rewizji i aktualizacji w internetowym procesie obejmującym wiele międzynarodowych towarzystw pankreatologicznych. Dzięki nowej koncepcji patogenezy choroby, jej przebiegu naturalnego i obiektywnego opisu oraz nowemu podziałowi trzustkowych i okołotrzustkowych zbiorników płynu ta klasyfikacja stanowi wartościowe narzędzie komunikacji na poziomie międzynarodowym. Nowa klasyfikacja wyróżnia 2 fazy ostrego zapalenia trzustki: wczesną (pierwszy tydzień lub dwa) i późną (dalszy okres). Ostre zapalenie trzustki może mieć charakter obrzękowego śródmiąższowego zapalenia trzustki albo martwiczego zapalenia trzustki; w tym drugim przypadku martwica może obejmować miąższ trzustki i tkanki okołotrzustkowe (najczęściej), tylko miąższ trzustki (najrzadziej) albo tylko tkanki okołotrzustkowe (~20%). Klasyfikacja ciężkości choroby obejmuje 3 poziomy: łagodny, umiarkowany i ciężki. Łagodne ostre zapalenie trzustki cechuje się niewystępowaniem zarówno niewydolności narządowej (klasyfikowanej wg zmodyfikowanej skali Marshalla), jak i powikłań miejscowych lub ogólnoustrojowych. W umiarkowanym ostrym zapaleniu trzustki może występować przemijająca (<2 dni) niewydolność narządowa, powikłania miejscowe i/lub zaostrzenie chorób współistniejących. Ciężkie ostre zapalenie trzustki zdefiniowano jako występowanie przetrwałej (≥2 dni) niewydolności narządowej. Powikłania miejscowe definiuje się wg obiektywnych kryteriów opartych głównie na tomografii komputerowej z użyciem kontrastu; wyróżnia się ostre okołotrzustkowe zbiorniki płynu, torbiele rzekome (bardzo rzadkie w ostrym zapaleniu trzustki), ostre (trzustkowe/okołotrzustkowe) zbiorniki pomartwicze oraz martwicę oddzieloną. Ta klasyfikacja pomoże lekarzom określać rokowanie chorych na ostre zapalenie trzustki i pozwoli na porównywanie pacjentów oraz sposobów leczenia w różnych krajach.

Adres do korespondencji:
Michael G. Sarr, MD, James C. Masson Professor of Surgery, Department of Surgery, Mayo Clinic, 200 First Street SW, Rochester, MN, USA 55 905, tel.: +1-507-255-5713, fax: +1-507-255-6318, e-mail: sarr.michael@mayo.edu

Praca wpłynęła: 11.01.2013.
Przyjęta do druku: 12.01.2013.
Publikacja *online*: 25.01.2013.
Nie zgłoszono sprzeczności interesów.
Pol Arch Med Wewn. 2013; 123 (3): 118-124
Tłumaczył lek. Łukasz Strzeszyński