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THE ATLANTA CLASSIFICATION AND DEFINITIONS OF ACUTE PANCREATITIS HAVE BEEN REVISED AND ARE BASED ON WORLDWIDE CONSENSUS

(Review Articles)

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ABSTRACT

Background and Objective: In a complex disease such as acute pancreatitis, correct terminology and clear definitions are important. The clinically based Atlanta Classification was formulated in 1992, but in recent years it has been increasingly criticized. No formal evaluation of the use of the Atlanta definitions in the literature has ever been performed. Deficiencies identified and improved understanding of the disease make a revision necessary. Methods: A Medline literature search sought studies published after 1993. The guidelines, review articles, and their cross-references were reviewed to assess whether the Atlanta or alternative definitions were used. Revisions were made in response to comments. The consensus was reviewed, and only statements based on published evidence were retained. Results: The severity of the disease is classified as mild, moderate, or severe. Mild acute pancreatitis, the most common form, has no organ failure, local or systemic complications, and usually resolves in the first week. Moderately severe acute pancreatitis is defined by the presence of transient organ failure, local complications, or exacerbation of co-morbid disease. Severe acute pancreatitis is defined by persistent organ failure, that is, organ failure lasting more than 48 h. Local complications are peripancreatic fluid collections, pancreatic and peripancreatic necrosis (sterile or infected), pseudocysts, and walled-off necrosis (sterile or infected). We present a standardized template for reporting CT images. There was a large variation in the interpretation of the Atlanta definitions of local complications, especially relating to the content of peripancreatic collections. Conclusion: The Atlanta definitions for acute pancreatitis are often used inappropriately, and alternative definitions are frequently applied. Such a lack of consensus illustrates the need for a revision of the Atlanta Classification. So, the consensus provides clear definitions to classify acute pancreatitis using easily identified clinical and radiological criteria.

Key words: Acute pancreatitis; Predictive severity scoring system; The Atlanta definitions; CECT imaging, Multi-organ failure; Necrosectomy; Pancreatic necrosis.

Introduction

Over the past 50 years, multidisciplinary symposia1-4 have produced a number of classification schemes for pancreatitis. The 1992 Atlanta Symposium, the most recent international conference on the subject, created a classification scheme with a clinical foundation.4.5 Acute pancreatitis was defined, along with its severity, organ failure, and local complications such "acute fluid collection," "pancreatic necrosis," "pseudocyst," and "pancreatic abscess." The Atlanta Classification made an effort to bring about consistency in the evaluation of clinical severity and the numerous illness consequences. The sole classification scheme utilised by radiologists and clinicians is this particular one.

Several writers have identified weaknesses in the Atlanta Classification due to growing understanding of the pathophysiology of pancreatitis and the creation of new interventional techniques.[6–13] Phlegmon and infected pseudocyst, for example, were terms dropped by the Atlanta symposium, but they are still commonly used in the literature. A recent analysis also showed that several new names, like "organised pancreatic necrosis" and "necroma," have been used since 1993.[14] There has never been a thorough analysis of how the Atlanta Classification is used in the literature.

Modern disease concepts were integrated into this revision in in order to clarify some points, improve clinical severity assessment, enable standardised reporting of data, aid in the objective assessment of new therapeutic treatments, and streamline communication between institutions and among treating physicians.

The current review analyses the level of diversity in these definitions' interpretations as well as the acceptance of the Atlanta Classification definitions in the literature. Consequently, this change is not meant to serve as a managerial directive.

Methods

The terms "acute pancreatitis and review" and "acute pancreatitis and guidelines" were used in a Medline search of literature published between 1993 and 2006 to find appropriate articles. Cross-references were collected from the found guidelines and reviews. All publications (reviews, recommendations, initial investigations, case reports, and editorials) were included in the search, however those not written in English were not included.

To determine if the following five elements of the Atlanta Classification were defined using the original Atlanta definitions from 1992 **(Table 1)** or another definition, one author (T.L.B.) did the selection and evaluated all full-text papers. Organ failure (determinants of specific failing organ systems, cut-off levels of determinants, distinction between single-organ failure and multi-organ failure), actual severity (difference between mild and severe pancreatitis, distinction between predicted and actual severity), local complications, and predicted severity (predictive scoring systems, predictive scoring systems, cut-off levels of scoring systems) (pancreatic necrosis and peripancreatic necrosis, infection of necrosis, morphological aspects and distinction of different types of collection).

One of the two other authors verified any instances where the components had differing definitions (H.C.v.S., M.G.B.). The authors discussed each issue to find a solution. Additionally, research findings that provided fresh perspectives and may have affected how the Atlanta Classification was interpreted

were noted and debated. Only the three most current publications for each element of the Atlanta Classification that was evaluated are cited here due to the vast number of references that were obtained.

Table 1 Summary of the 1992 Atlanta Classification

	Definition
Acute pancreatitis	An acute inflammatory process of the pancreas with variable involvement of other regional tissues or remote organ systems Associated with raised pancreatic enzyme levels in blood and/or urine
Severity Mild acute pancreatitis	Associated with minimal organ dysfunction and an uneventful recovery; lacks the features of severe acute pancreatitis. Usually normal enhancement of pancreatic parenchyma on contrast-enhanced computed tomography
Predicted severity APACHE II score ≥ 8 Organ failure and systemic complications	Ranson score ≥ 3 or
Shock	Systolic blood pressure < 90 mmHg
Pulmonary insufficiency	Creatinine ≥ 177 □mol/l or ≤ 2 mg/dl after rehydration
Gastrointestinal bleeding	500 ml in 24 h
Disseminated intravascular coagulation	Platelets ≤ 100, 000/mm3, fibrinogen < 1·0 g/l and fibrin-split products > 80 □g/l
Severe metabolic disturbances	Calcium ≤ 1·87 mmol/l or ≤ 7·5 mg/dl
Local complications	
Acute fluid collections	Occur early in the course of acute pancreatitis, are located in or near the pancreas and always lack a wall of granulation of fibrous tissue. In about half of patients, spontaneous regression occurs. In the other half, an acute fluid collection develops into a pancreatic abscess or pseudocyst
Pancreatic necrosis	Diffuse or focal area(s) of non-viable pancreatic parenchyma, typically associated with peripancreatic fat necrosis Non-enhanced pancreatic parenchyma > 3 cm or involving more than 30% of the area of the pancreas
Acute pseudocyst	Collection of pancreatic juice enclosed by a wall of fibrous or granulation tissue, which arises as a result of acute pancreatitis, pancreatic trauma or chronic pancreatitis, occurring at least 4 weeks after onset of symptoms, is round or ovoid and most often sterile; when pus is present, lesion is termed a 'pancreatic abscess'
Pancreatic abscess	Circumscribed, intra-abdominal collection of pus, usually in proximity to the pancreas, containing little or no pancreatic necrosis, which arises as a consequence of acute pancreatitis or pancreatic trauma Often 4 weeks or more after onset Pancreatic abscess and infected pancreatic necrosis differ in clinical expression and extent of associated necrosis

Grades of severity

- Mild acute pancreatitis
  - No organ failure
  - No local or systemic complications
- Moderately severe acute pancreatitis
  - Organ failure that resolves within 48 h (transient organ failure) and/or
  - Local or systemic complications without persistent organ failure
- Severe acute pancreatitis
  - Persistent organ failure (>48 h)

- Single organ failure
- Multiple organ failure

Results

447 papers in all, including 82 reviews and 12 guidelines, were examined. These articles just used Atlanta definitions while reporting on research that weren't intended to evaluate the Atlanta Classification, such as a randomised trial comparing two treatment modalities with the result "pseudocyst." An evaluation of methodological quality was therefore ruled inappropriate. **Table 2** provides a summary of the papers by the article type and journal impact factor that they were published in. The most significant differences between the 12 guidelines and ***the five components of the Atlanta Classification*** are explored in order:

Diagnosis

Pancreatic enzyme levels have no cut-off value specified by the Atlanta Classification. A distinctive clinical history of stomach discomfort and an elevation of pancreatic enzyme levels to three or more times the upper limit of normal were necessary for the diagnosis of acute pancreatitis in 116 investigations. Although 31 studies utilised a variety of criteria, ranging from two or more[15-17] than four[18-20] and more than five[21-23] times the upper limit of normal, these thresholds were all within this range.

Predicted severity

A total of 283 articles included criteria for estimating acute pancreatitis severity. The severity rating schemes suggested by the Atlanta symposium were applied in about 86 reports.[16,17,23] However, 197 studies employed a different cut-off point to define severity, or they used extra or other scoring systems, such as the Sequential Organ Failure Assessment, Imrie (Glasgow) score, Imrie (CT) severity index, or severity predictors (such as C-reactive protein).[15,24,25] The severity stratification cut-off values varied widely between reports. The most well-known radiological grading system, the CT severity index, was created by Balthazar and colleagues in 1990. The cut-off value to distinguish between mild and severe disease ranged from three to eight points.[27-29]
Acute Physiology and Chronic Health Evaluation (APACHE) II threshold values (other than eight or more) varied between five and more in 32 studies, while the duration for calculating the score ranged from the day of admission to 24, and 48 h after admission.[30–32] Eleven studies, ranging in size from more than three to more than five, used various threshold values for the Ranson criteria (other than three or more).[32 – 34]

Since the Atlanta symposium in 1992, numerous researchers have discovered additional severity determinants, and these have been integrated into a number of guidelines. Obesity (body mass index greater than 30 kg/m2), age (above 55[6], over 70[35], or over 80[36] years), and [11, 24, 37]; left or bilateral pleural effusion on a chest radiograph;[38– 40]; increased haematocrit;[6, 41, 42]; and a C-reactive protein level of more than 150 mg/dl after 48 hours.[43-45]

Table 2 Characteristics of retrieved articles (1993 – 2006) specified according to impact factor of journal

Total no. of studies (*n* = 447)		High (> 5·0) (*n* = 89)	**Impact factor** Intermediate (1·5–4·9) (*n* = 273)	Low (< 1·5) (*n* = 85)
Meta-analyses	3	2	1	0
Randomized controlled trials	34	13	18	3
Prospective series	144	28	99	17
Retrospective series	147	23	95	29
Reviews	82	10	44	28
Guidelines	12	5	5	2
Editorials	5	2	3	0
Other	20	6	8	6

Actual severity

Of the 297 articles that supplied definitions for severe acute pancreatitis, 195 did so in accordance with the Atlanta Classification, while 61 merely noted the adoption of the Atlanta criteria without giving further specifics.[46–48] The Atlanta Classification was not utilized to characterize serious disease in the remaining 102 articles. These definitions were based on various additional or undefined criteria, like intensive care unit admission, hospital stay length, complications requiring medical or surgical intervention, mortality, etc.[17,49,50] The authors of 45 publications used moderate acute pancreatitis and severe acute pancreatitis indiscriminately when talking to existence or absence of pancreatic necrosis.[47,51,52] However, several reports noted that individuals with the morphological diagnosis of interstitial pancreatitis can suffer clinically serious illness.[44,53,54]

It is unclear how the development of organ failure is impacted by pancreatic necrosis, a significant contributor to severe acute pancreatitis. Several studies found that only 51–55% of patients with pancreatic necrosis had organ failure.[55–57] In the study by Lankisch and colleagues, 53.5% of patients with acute edematous pancreatitis had organ failure. In a recent study, organ failure was discovered to be the main risk factor for mortality regardless of whether pancreatic necrosis was present or absent.[23] On the other hand, numerous studies showed a high correlation between organ failure and the severity of pancreatic necrosis.[16,58,59]

In 38 articles, which compared acute pancreatitis with both conditions, the distinction between "actual severe" disease (systemic or local complications) and "predicted severe" disease (Ranson, Imrie, or APACHE II score) was not readily apparent from the provided data.[17,28,60] The distinction is important because, according to recent studies, fewer than 50% of individuals who were expected to have severe disease really did so, according to the Atlanta criteria.[25,46] This lack of differentiation may account for the variation in incidence of severe acute pancreatitis between institutions.

Organ failure

149 articles contained organ failure criteria. The precise Atlanta definitions for organ failure were stated and used in 35 reports.[23,61,62] Seven publications limited organ failure to the respiratory and renal insufficiencies, two of the four Atlanta determinants for organ failure.[63–65] However, 107 articles used additional criteria for organ failure and systemic complications, including sepsis, leucocytosis, temperature, coagulopathy, nervous system failure, hepatic failure, and systemic inflammatory response syndrome, or they used different cutoffs or adjustments for the Atlanta definitions of organ failure.[52,66,67] The remaining articles either did not define organ failure at all or they only said that the Atlanta criteria were used without more explanation.
Multiorgan failure has been identified as an important predictor of mortality in recent years. Multiorgan failure, however most research did not define it, has been defined in 20 reports as the failure of two or more organ systems,[31,46,49] and eight reports as the failure of three or more organ systems.[23,68,69]

Numerous authors have distinguished between transitory and permanent organ failure, highlighting the dynamic process of organ malfunction.[70–72] Furthermore, numerous investigations revealed that while temporary organ failure typically had an easy course, early and progressive organ failure were associated with increased mortality.[72–74] According to the most recent UK guidelines on acute pancreatitis, organ failure in the first week that settles within 48 hours shouldn't be taken as a sign of a serious illness.[43]

The number of organ systems involved as well as the extent of each organ's dysfunction are taken into account by the Goris score, Marshall or multiple organ dysfunction score, Bernard score, Sequential Organ Failure Assessment, and logistic organ dysfunction syndrome score, all of which are developed since 1993. Some systems additionally require the use of mechanical ventilation, dialysis, and inotropic or vasopressor medications, which even the Atlanta symposium did not take into consideration.
Numerous studies have demonstrated that dynamic scoring systems, such as the delta APACHE II score, or scoring systems that take into account the physiological response to therapy, such as the delta organ failure score or cumulative Marshall score, are superior to static scoring systems as predictors of outcome.[31,32,71]

Local complications

Interobserver agreement was poor in a recent investigation on the Atlanta criteria for the various local complications; just three of the 70 collections visualized by contrast-enhanced CT (CECT) had five radiologists agree on the corresponding Atlanta classification.[8]

Acute fluid collection

In 64 articles, a definition was given for an "acute fluid collection'. The following terms were used to describe acute fluid collections: '(peri)pancreatic fluid collections",[75–77] "peripancreatic effusions",[78] "extra pancreatic fluid collections",[61,79,80] "immature pseudocyst",[81,82] and "exudates".[54] (Peri)pancreatic fluid collection was also used as an overall descriptive term for all types of collection related to acute pancreatitis.[83–85]

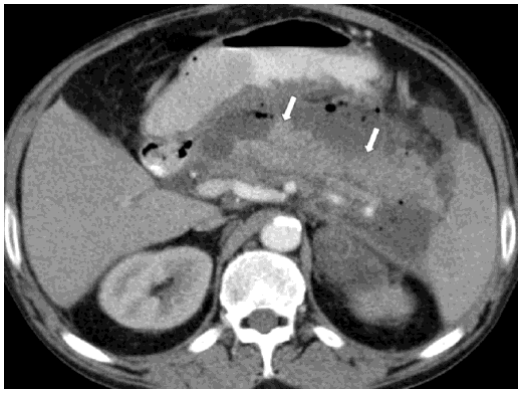
In most reports, the differentiation between acute fluid collection and pseudocysts was made after 4 weeks from the onset of disease (as proposed by the Atlanta Classification). In eight reports, however, a different time period was used as a criterion for this distinction, varying from 3 weeks[75, 86, 87] to 6[88, 89] and even 8[90] weeks. Moreover, they did not adequately describe whether acute fluid collections consisted of fluid alone or whether they may have contained necrotic debris.[85,91,92]

The authors of 17 articles regarded the occurrence of an acute fluid collection as a local complication and so a sign of 'severe disease'.[46,62,93] However, most others did not include acute fluid collection either in the definition of local complication or in that of severe disease.

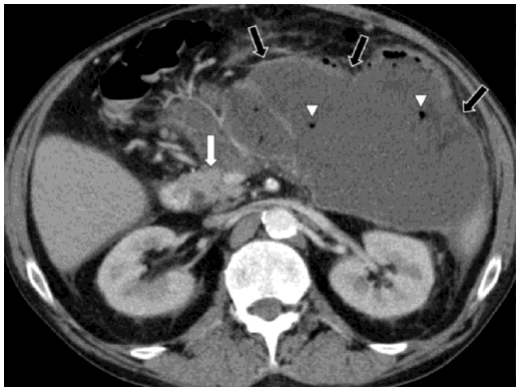
Pancreatic necrosis

47 of the 152 articles ***(Fig. 1)*** that provided a comprehensive definition of "pancreatic necrosis" or "necrotizing pancreatitis" used the Atlanta criterion of more than 30% parenchymal necrosis to do so.[28,61,94] However, necrotizing pancreatitis was defined as any indication of pancreatic parenchymal necrosis, even if it was less than 30%.[47,95,96] The emergence of pancreatic necrosis, extra pancreatic necrosis, or both on CECT (along with a serum C-reactive protein value of more than 150 mg/dl) was a third definition of necrotizing pancreatitis that was cited in 20 studies.[52,86,97]

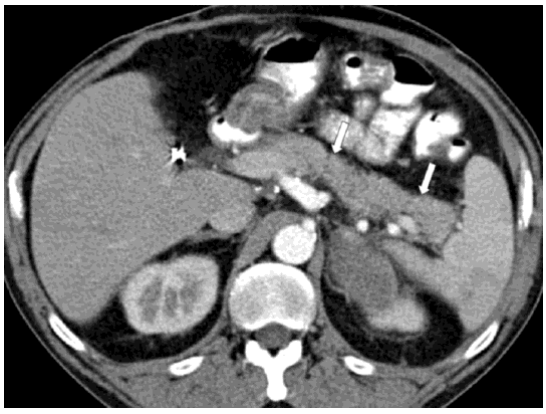
Pancreatic parenchymal non-enhancement on CECT is a requirement for pancreatic necrosis in the Atlanta Classification. [4] However, some healthcare professionals questioned if the absence of enhancement on CECT indicated necrosis and irreversible harm.[86,98,99] For instance, devitalized tissue discovered during surgery was classified as pancreatic necrosis by Traverso and Kozarek86. Takeda and colleagues,[100-102] who pointed out that pancreatic parenchymal perfusion was preserved during intra-arterial angiography whereas CECT revealed pancreatic non-enhancement, provided support for this. On the other hand, multiple investigations showed a strong association between the presence of pancreatic necrosis and parenchymal non-enhancement on CECT (confirmed at operation).[103–105]



a Normal enhancement of the pancreas



b Large fluid collection with gas bubbles



c Follow-up 6 months after operation

Fig. 1 Contrast-enhanced computed tomography (CT) of a patient with acute pancreatitis 22 days after onset of symptoms

a with normal enhancement of the pancreas (white arrows) and

b surrounded by a large heterogeneous and encapsulated fluid collection (black arrows) with gas bubbles (arrowheads) suggesting secondary infection. Some would call this 'necrotizing pancreatitis', but others would call it 'interstitial pancreatitis' because there is no evidence of pancreatic parenchymal necrosis (only peripancreatic necrosis). A large amount of fat necrosis was debrided during operation.

c Follow-up CT 6 months after operation reveals a normal enhancing pancreatic parenchyma (white arrows)

Contradictory information exists on the diagnostic efficacy of CECT in extra-pancreatic or peripancreatic fat necrosis. Several investigations showed a strong association between extra pancreatic findings on CECT and the presence of fat necrosis at operation or autopsy, despite eight groups' claims that fat necrosis could not be consistently detected by CECT.[92,106,107,104,108,109]
According to the Atlanta Classification, "pancreatic necrosis" applies to both infected and sterile necrosis. 4 According to some organizations, pancreatic parenchymal necrosis without infection does not provide a significant morbidity risk.[110–112]
Studies demonstrating an uncomplicated course in the presence of necrosis without infection provided support for this.[23,55,56]

Beger and colleagues[81,113] were the first to point up necrosis as a potential nidus for secondary infection, which affects 40–70% of patients. This was supported by recent research, which showed that infected necrosis was the main reason for late mortality.[58,114,115] Definitions of "infected necrosis," though, varied widely. According to some writers, parenchymal necrosis, peri-pancreatic fat necrosis (in the absence of parenchymal necrosis), or both can be infectious.[67,76,119]

Pseudocyst

In 87 publications, the term "pseudocyst" had a particular definition that was similar to the Atlanta Classification. However, several issues continue to be controversial. The pseudocyst category comprised 38 articles that featured collections of both fluid and necrotic material (Fig. 2, 3).120–122 However, according to Baron123 and others85,124, pseudocysts should be free of substantial necrotic debris. According to the evidence, collections with fluid alone and those with fluid plus necrosis differed in their therapeutic approach and results.[84,125,126] The mistaken identity of (peri)pancreatic fluid collections as pseudocysts by CECT, in Bradley's [127] opinion, is a very frequent mistake in modern diagnostic radiography. This misinterpretation has two potentially harmful effects: ***first***, by instrumentation of a sterile collection containing both fluid and necrosis, infection may be introduced6,[120,128]; ***second***, a delay in appropriate intervention may occur.[33,120,129]

There were differences between acute and chronic pseudocysts in terms of incidence, natural history, and management choices. As a result of the lack of distinction made between pseudocysts and acute fluid collections, or between pseudocysts that complicated acute and chronic pancreatitis, several authors emphasized that the results of treatment for pancreatic fluid collections in the literature were challenging to interpret.[122,128,130] Only five of the 31 original studies on the topic of treating pseudocysts that were reviewed specifically addressed those that developed following an episode of acute pancreatitis.[89,120,131] The outcomes of treating pseudocysts that exacerbated acute and chronic pancreatitis were reported in the remaining 26 publications.[121,132,133]

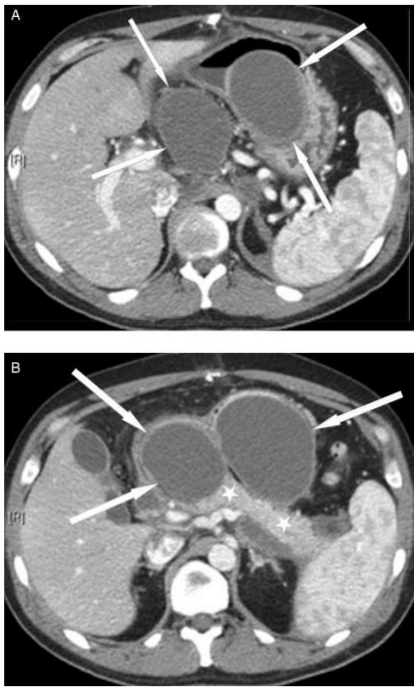


Figure 2 A 40-year-old man with two pseudocysts in the lesser sac 6 weeks after an episode of acute interstitial pancreatitis on CT (A, B). Note the round to oval, low-attenuated, homogeneous fluid collections with a well-defined enhancing rim (white arrows pointing at the borders of the pseudocysts), but absence of areas of greater attenuation indicative of non-liquid components. White stars denote normal enhancing pancreas.

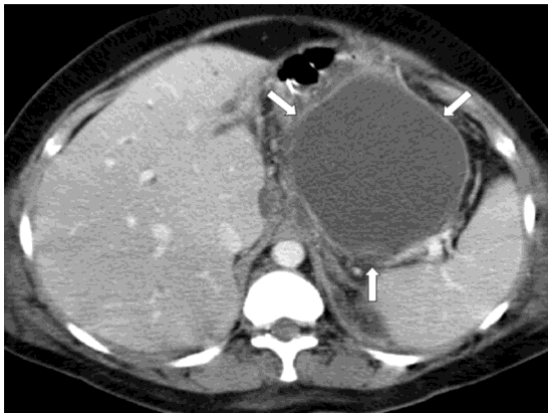


Fig. 3 Contrast-enhanced computed tomography (CT) of a patient with acute pancreatitis 30 days after onset of symptoms. The fluid collection seems to be homogeneous and encapsulated (white arrows) and could be interpreted as a 'pseudocyst' according to the Atlanta Classification. However, at operation the collection was found to contain large amounts of necrotic debris that CT had not show

Pancreatic abscess

A definition of "pancreatic abscess" was given in 68 papers, and it typically matched the original Atlanta definition. Three of the nine original publications published after 1993 that addressed the treatment of "pancreatic abscesses" strictly adhered to the Atlanta definition (collection of pus and almost little necrotic debris, more than 4 weeks after onset).[134–136] The others comprised specimens that were treated within 4 weeks after disease onset[140] or after surgery, or those contained solid necrotic material in addition to pus.[141,142] On CECT, the diagnosis of pancreatic abscess is similarly debatable. The "air bubble" phenomenon was deemed "diagnostic of a pancreatic abscess" in 10 articles.[93,143,144] Gas bubbles in a heterogeneous collection on a CT scan, however, were thought to be a strong indicator of infected pancreatic necrosis ***(Fig. 4)***.[61,67,145] There are numerous theories as to the cause of pancreatic abscess. According to some authors, pancreatic abscesses and "post-acute pseudocysts" are late effects of necrotizing pancreatitis.[146–148] The interstitial pancreatitis with a normal augmenting pancreas on CECT, according to some, is the only condition in which pancreatic abscesses can develop.[117,149,150]

Several authors proposed that pancreatic abscesses arose from the gradual liquefaction of necrotic pancreatic and peripancreatic tissues, which eventually led to full liquefaction, in addition to the "infection of a pseudocyst".[76,123,151] Although some doctors detect "pancreatic abscesses" after 1[50,152],2[153,154] or 3[86,146,147] weeks, the Atlanta Classification states that the majority of pancreatic abscesses develop at least 4 weeks after the onset of symptoms.[4] Interestingly, variable degrees of liquefaction of necrotic tissue were seen during operational necrosectomy performed several months after the beginning of severe acute pancreatitis by Morgan and colleagues[10], Howard and Wagner[155], and others.[156] Given that they found both pus and necrotic material in these (infected) collections, several authors speculated that a collection would be a transitional form from (infected) pancreatic necrosis to a (infected) pseudocyst or pancreatic abscess.[7,12,139]

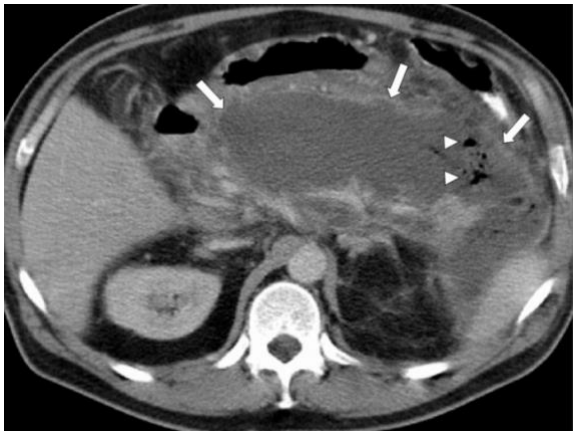


Fig. 4 Contrast-enhanced computed tomography of a patient with acute pancreatitis 36 days after onset of symptoms. The body and tail of the pancreas are largely non-enhancing. Adjacent to the pancreatic bed is a large collection with predominately fluid-like attenuation (white arrows). Because of the gas bubbles (arrowheads), some would call this a ‘pancreatic abscess’ but others would call it ‘infected pancreatic necrosis’

Walled-off necrosis

WON consists of necrotic tissue contained within an enhancing wall of reactive tissue. This collection of pancreatic and/or peripancreatic necrosis is mature, encapsulated, and has a well-defined inflammatory wall ***(figure 5)***; typically, this maturation takes place about 4 weeks following the commencement of necrotising pancreatitis. The term "organised pancreatic necrosis" as well as "necroma," "pancreatic sequestration," "pseudocyst associated with necrosis," and "subacute pancreatic necrosis" were previously proposed names for this condition.

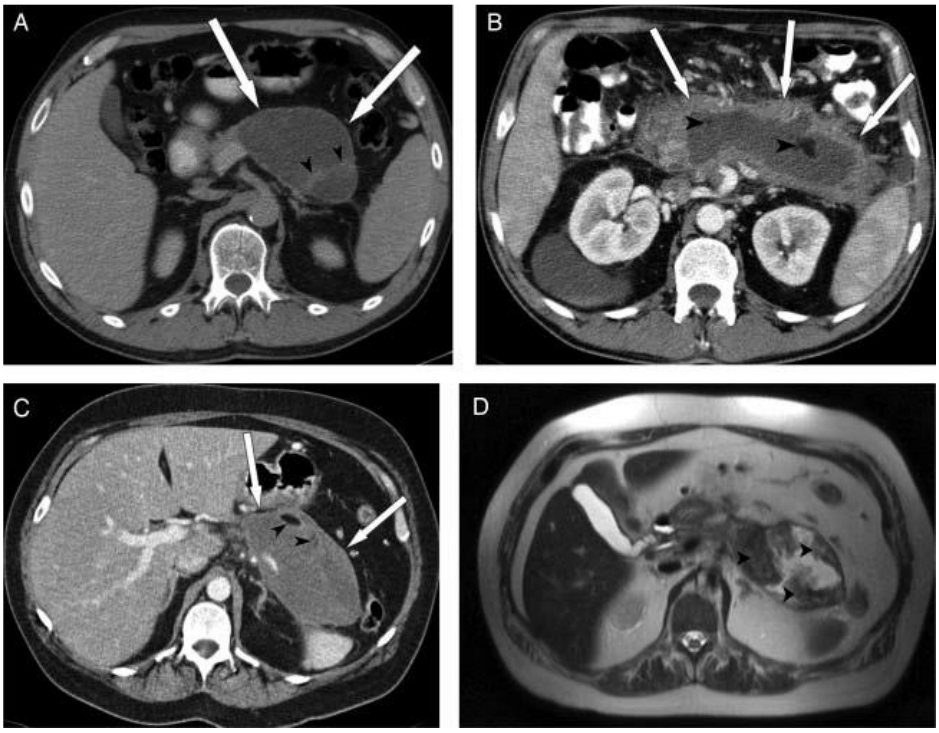


Figure 5 (A–C) Three different patients with walled-off necrosis (WON) after an acute attack of necrotising pancreatitis. In all three patients, a heterogeneous, fully encapsulated collection is noted in the pancreatic and peripancreatic area.

(A) Non-liquid components of high attenuation (black arrowheads) in the collection ar noted. The collection has a thin, well defined, and enhancing wall (thick white arrows).

(B, C) A largely liquefied collection in the bed of the pancreas is observed with non-liquid components representing areas of trapped fat (black arrowheads).

(D) represents the corresponding T2-weighted MRI to (C), showing the tru heterogeneity of the collection. Black arrowheads denote areas of necrotic debris surrounded by fluid (white on T2-weighted image).

The most significant differences in the criteria of organ failure and those of projected severe disease were seen in the 12 guidelines[6,35,36,43,148,157-165] on acute pancreatitis. These are summarized in Table 3.

Table 3 Overview of definitions for organ failure and predicted severe acute pancreatitis in guidelines for acute pancreatitis published after **1993**

Guideline	Definitions for organ failure	Definitions for predicted severe acute pancreatitis
ACG 1997[157]	Refers to Atlanta Classification 1992	Ranson score ≥ 3 after 48 h APACHE II score > 8 after 48 h
UK 1998[158]	Refers to Atlanta Classification 1992	Ranson/Glasgow ≥ 3 CRP > 210 mg/l (first 4 days) or > 120 mg/l at 1 week APACHE II score ≥ 9 (severe acute pancreatitis) or ≥ 6 (includes all severe cases, but PPV of 50%)
SSAT 1998[159]	Not addressed	Not stated
Santorini 1999[160]	Not addressed	BMI > 30 kg/m^2 Pleural effusion APACHE II score ≥ 6 (at 24 h) APACHE (obesity) score ≥ 6 CRP > 150 mg/l
French 2000[36]	Renal failure: creatinine > 170 μmol/l Shock: systolic BP < 90 mmHg despite fluid replacement Pulmonary insufficiency: Pa_{O2} ≤ 60 mmHg on room air Glasgow Coma Score < 13 Platelets < 80 g/l	At admission Age > 80 years BMI > 30 kg/m^2 Chronic renal failure Pre-existing severe illnesses At 24–48 h Presence of organ failure by using simple measures or use of scoring system (e.g. SOFA) Ranson/Imrie score > 3 CECT: CT severity index ≥ 4 (48–72 h) CRP > 150 mg/l *Note: 'The non-specific scores (APACHE II, SAP II, etc) are not recommended by the Jury'*
WCG 2002[35]	SIRS ≥ 1 vital organ dysfunction ARDS Renal failure: increased serum creatinine > 0·5 mg/dl (44 μmol/l) or 50% above baseline or reduction in calculated creatinine clearance > 50% or need for dialysis Hypotension: mean arterial pressure < 60 mmHg DIC Acute adrenal insufficiency Acute hepatitis Metabolic encephalopathy Ileus	At admission Age > 70 years Clinical assessment BMI > 30 kg/m^2 Pleural effusion/infiltrates CECT: > 30% non-enhancement of the pancreas APACHE II score ≥ 8 Presence of organ failure At 24–48 h Clinical assessment Glasgow score (no cut-off value provided) CRP > 150 mg/l Presence of organ failure
IAP 2002[161]	Not addressed	Not stated: surgical guideline
JSAEM 2002[162]	Not addressed	Clinical signs CRP (48 h: no cut-off value provided) BMI (no value provided) CECT: necrosis Scoring system, like JMHW, APACHE II at 24 h or Ranson/Glasgow at 24–48 h: no cut-off values provided Japanese score ≥ 2
Nathens 2004[148]	Refers to the guidelines for intensive care unit admission, published in 1999[163]	Elderly (age not specified) BMI > 30 kg/m^2 Patients requiring ongoing volume resuscitation CECT: > 30% non-enhancement of the pancreas Clinical assessment *Note: 'Disease-specific scoring systems or severity scores are useful adjuncts to identify patients at high risk of a complication but should not replace serial clinical assessments. In addition, there is a recommendation against the use of markers such as CRP or procalcitonin to guide clinical decision making or predict clinical course of acute pancreatitis or to triage patients'*

Guideline	Definitions for organ failure	Definitions for predicted severe acute pancreatitis
UK 2005[43]	Refers to Atlanta Classification 1992	At admission Clinical assessment BMI > 30 kg/m^2 Pleural effusion APACHE score > 8 At 24–48 h Clinical assessment Glasgow score ≥ 3 APACHE II score > 8 Persistent organ failure for 48 h (especially if multiple and progressive) CRP > 150 mg/l *Note: 'Organ failure present within 1 week, which resolves within 48 h, should not be considered an indicator of a severe attack of acute pancreatitis'*
ACG 2006[6]	Refers to Atlanta classification 1992 *Note: 'Criteria of organ failure will change in the future: gastrointestinal bleeding will undoubtedly be deleted'*	At admission Age > 55 years BMI > 30 kg/m^2 Presence of organ failure Pleural effusion/infiltrates 24–48 h APACHE II score ≥ 8 Serum haematocrit ≥ 44% *Note: 'Ranson signs are no longer advocated, due to a comprehensive evaluation of 110 studies that concluded that Ranson signs provided very poor predictive power of severity of acute pancreatitis'*
JSAEM 2006[164,165]	Pulmonary insufficiency: dyspnoea Shock Central nervous system disorders Bleeding tendency Negative base excess failure: rise of blood urea nitrogen level and creatinine level	Japanese score ≥ 2

ACG, Practice Parameters Committee of the American College of Gastroenterology; APACHE, Acute Physiology And Chronic Health Evaluation; UK, Working Party of the British Society of Gastroenterology, Association of Surgeons of Great Britain and Ireland, Pancreatic Society of Great Britain and Ireland, and Association of Upper GI Surgeons of Great Britain and Ireland; CRP, C-reactive protein; PPV, positive predictive value; SSAT, Society for Surgery of the Alimentary Tract; Santorini, Santorini Consensus Conference; BMI, body mass index; French, French Consensus Conference on Acute Pancreatitis; BP, blood pressure; Pa_{O2}, arterial partial pressure of oxygen; SOFA, Sequential Organ Failure Assessment; CECT, contrast-enhanced computed tomography; SAP, Simplified Acute Physiology; WCG, World Congress of Gastroenterology; SIRS, systemic inflammatory response syndrome; ARDS, adult respiratory distress syndrome; DIC, disseminated intravascular coagulation; IAP, International Association of Pancreatology; JSAEM, Japanese Society of Emergency Abdominal Medicine; JMHW, Japanese Ministry of Health and Welfare; Nathens, Consensus Statement regarding the management of the critically ill patient with severe acute pancreatitis.

Discussion

The current review has revealed that the Atlanta definitions of acute pancreatitis severity and local complications are applied inconsistently and that various aspects of the classification have drawn a lot of criticism. The Atlanta conference improved the management of acute pancreatitis and clinical research pertaining to the condition by providing definitions, the outcome of consensus among more than 40 scientists based on the evidence available in 1992. The last 20 years, however, have seen advancements in imaging methods in addition to new understandings of pathophysiology and therapeutic approaches. It seems obvious that it is time to change how acute pancreatitis is categorised. Since the Atlanta symposium, the various predictive scoring systems have not much improved. When forecasting severe disease in a particular patient, they are only moderately accurate. Predictive systems were initially created to assign patients to clinical trials, not to determine the severity of an individual, as McKay and Imrie[166] have highlighted. There are additional drawbacks to determining severity based on the presence or absence of organ failure. The most significant predictor of morbidity and death, which are primarily correlated with the number of organ systems failing, the severity of the organs implicated, and the length of organ failure, is now widely acknowledged to be persistent organ failure (lasting longer than 48 hours). Because it includes both sterile and infectious necrosis, as well as pancreatic parenchymal necrosis and peripancreatic fat necrosis, the definition of necrotizing pancreatitis is questionable. Because the necrotic debris present in pseudo-cysts and pancreatic abscesses is frequently disregarded, interpretations of these collections differ considerably. This could be explained by CECT's inability to distinguish between sterile and infectious collections as well as its inability to detect necrotic material in collections predominately holding fluid.[7,10,12,92,167] Although endoscopic ultrasonography and magnetic resonance imaging (MRI) may be of further benefit in identifying these collections, their utility in critically unwell patients has been questioned.[168,169. 92,170]. The Atlanta Classification includes morphological and pathological descriptions of various local consequences, but it does not give precise radiological criteria for each. The necessity for new descriptive morphological terminology to describe CECT results is highlighted by the recently found low interobserver agreement on the Atlanta Classification of local complications.[8] The CT severity index, an existing radiological grading system, utilizes a numerical score system to quantify extra pancreatic alterations and the degree of pancreatic necrosis.[26] The CT severity score does not identify the regional consequences of acute pancreatitis, although having a clear predictive value in terms of morbidity and mortality.[26,171–174] The paucity of prospective evidence from extensive patient series is largely to blame for the ongoing discussion regarding the normal progression of (peri)pancreatic collections. Consequently, in order to better understand the etiology, natural history, and ideal care of (peri)pancreatic collections, the authors of this paper call for an international collaboration. The goal of the current review is to provide a summary of the controversies around the Atlanta Classification in the literature. The Atlanta Classification's recommended definitions have rarely been the subject of study. As a result, there are not many original data points on this subject that can be examined. Therefore, this analysis has only classified uses and readings of the Atlanta definitions. For effective communication in clinical settings and for comparing inter-institutional data for clinical research, correct terminology and standard definitions are crucial. The development of evidence-based recommendations has been impeded by the ongoing inability to employ standardised definitions for expected and actual severe acute pancreatitis, organ failure, local consequences, and patient inclusion criteria in clinical studies. Numerous research that have advanced understanding of the disease's natural course have been cited in this study. These fresh perceptions should be used to design new classification.

The author proposes the following recommendations for revision of the classification of acute pancreatitis:

First, two of the following four components must be present: upper abdominal pain, amylase and/or lipase levels that are at least three times above normal (this cut-off is most frequently used in the literature), and CT or MRI results that are compatible with acute pancreatitis.

Second, determining the severity of acute pancreatitis should take into account persistent organ failure (for at least 48 h).

Third, based on a thorough analysis of the information provided, it should be chosen which predictive scoring system(s), including cut-off value, should be used to characterise predicted severe acute pancreatitis.

Fourth, future research should always distinguish between anticipated severe and actual severe disease, with validation of the severity of the disease.

Fifth, a systematic review should show which organ failure scoring system should be utilised, and definitions for organ failure should take into account the number of failing organ systems, the length of time the organ has been failing (less than or greater than 48 hours), and the requirement for a particular therapy (such as inotropic or vasopressor agents, mechanical ventilation, and dialysis).

Sixth, necrotizing pancreatitis or peripancreatic fat necrosis should be considered if there is no pancreatic parenchymal necrosis.

Seventh, infected necrosis must be handled differently than other conditions. Moreover, infected necrosis needs to be treated as a distinct condition.

Eighth, enclosed collections that include both fluid and necrotic debris should be given a label.

Ninth, in order to rule out necrotic material in a collection that contains just fluid (such as a pseudocyst), MRI or (endoscopic) ultrasonography should be carried out first.

Tenth, to describe local problems on CECT, a new set of descriptive morphological terminology should be created.

Such a new classification system should be evaluated in high-quality interobserver and prospective clinical studies. Adjustments should be made every few years, based on new data. Most importantly, clinicians and radiologists worldwide should comply with the new classification in clinical practice and research. Progress in the field of acute pancreatitis is hampered greatly when various author groups use their own idiosyncratic definitions. When journal referees are requested to peer-review manuscripts, they should pay special attention to the correct use of definitions as defined by a new classification.

CONCLUSION

The definitions of acute pancreatitis from the Atlanta Classification have been revised and updated in this classification. The understanding that acute pancreatitis is a growing, dynamic condition and that the severity may evolve over the course of the disease is a crucial component. SIRS or organ failure at an early stage of the illness signal potentially serious illness.

- The condition is referred to as mild acute pancreatitis if the patient improves quickly during the early stages without experiencing organ failure or other local or systemic consequences.
- The condition is referred to as moderately severe acute pancreatitis if the patient experiences local or systemic consequences but does not have permanent organ failure.
- The condition is classified as severe acute pancreatitis and is linked to extremely high morbidity and mortality rates if the patient experiences prolonged organ failure.

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Finally, we hope that this work will prove to be useful and beneficial to all medical staff, paramedics and allied professionals. Indeed, we dedicate this work to all junior doctors who will take this effort to practice, develop and lead this demanding but rewarding profession.

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