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Predicting the severity of acute pancreatitis

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INTRODUCTION

Approximately 15 to 25 percent of all patients with acute pancreatitis (AP) develop moderately severe (MSAP) or severe AP (SAP). Between 1988 and 2003, mortality from acute pancreatitis decreased from 12 percent to 2 percent, according to a large epidemiologic study from the United States [1]. However, mortality rates remain much higher in subgroups of patients with severe disease. The ability to predict its severity can help identify patients at increased risk for morbidity and mortality, thereby assisting in appropriate early triage to intensive care units and selection of patients for specific interventions.

This topic review will summarize methods for predicting the severity of AP. The clinical manifestations, diagnosis, and treatment of AP are discussed separately. (See "[Clinical manifestations and diagnosis of acute pancreatitis](#)" and "[Management of acute pancreatitis](#)".)

GENERAL CONSIDERATIONS

A multitude of predictive models have been developed to predict the severity of AP based upon clinical, laboratory, and radiologic risk factors, various severity grading systems, and serum markers. Some of these can be performed on admission to assist in triage of patients, while others can only be obtained after the first 48 to 72 hours or later.

However, these predictive models have low specificity (ie, high false-positive rates), which, when coupled with the low prevalence of moderately severe (MSAP) and severe (SAP), results in low positive predictive values [2-8]. Studies that have tried to predict MSAP could not differentiate this from SAP [9]. There is a need to identify predictive markers or tools that are accurate in prognosticating both MSAP and SAP during the initial 24 to 72 hours [10]. In addition, measuring clinical outcomes in groups with and without the use of such tools is also required, but clinically pertinent only if a drug or other specific therapy is available to treat AP. Future predictive models will need to incorporate additional factors (eg, biomarkers, genetic polymorphisms and mutations, and proteomic and metabolomic patterns) and methods of analyses [11].

CLASSIFICATION OF ACUTE PANCREATITIS

The revised Atlanta classification system divides acute pancreatitis into two broad categories ([table 1](#)) [12,13]:

- Interstitial edematous acute pancreatitis, which is characterized by acute inflammation of the pancreatic parenchyma and peripancreatic tissues, but without recognizable tissue necrosis
- Necrotizing acute pancreatitis, which is characterized by inflammation associated with pancreatic parenchymal necrosis and/or peripancreatic necrosis

According to the severity, acute pancreatitis is divided into the following:

- Mild acute pancreatitis which is characterized by the absence of organ failure and local or systemic complications
- Moderately severe acute pancreatitis which is characterized by transient organ failure (resolves within 48 hours) and/or local or systemic complications without persistent organ failure (>48 hours)
- Severe acute pancreatitis which is characterized by persistent organ failure that may involve one or multiple organs

Local complications of acute pancreatitis include acute peripancreatic fluid collection, pancreatic pseudocyst, acute necrotic collection, and walled-off necrosis ([table 1](#)).

Organ failure is defined as a score of two or more for any one of three organ systems

(respiratory, cardiovascular, or renal) using the modified Marshall scoring system ([table 2](#)) [14]. Studies using the revised classification found severe type occurred in less than 5 percent and moderately severe type around 15 percent [9].

CLINICAL PREDICTORS

Older age — Several studies have concluded that older age is a predictor of a worse prognosis, although the age cutoff has varied from 55 to 75 years in different reports [15,16]. In an illustrative study, patients older than 75 years had more than a 15-fold greater chance of dying within two weeks and a more than 22-fold greater chance of dying within 91 days compared with patients aged 35 years or younger [16].

Sex — The patient's sex has not been a predictor of outcome in most reports [15].

Alcoholic pancreatitis — Alcohol as a cause of pancreatitis has been associated with an increased risk of pancreatic necrosis and need for intubation in some reports [16-18].

Short time interval to symptom onset — A time interval between the onset of symptoms and hospital admission of less than 24 hours, as well as rebound tenderness and/or guarding were associated with increasing severity of pancreatitis in at least one report [19].

Obesity — Many studies have found obesity (defined as a body mass index >30) to be a risk factor for severe AP. A meta-analysis that included 739 patients made the following estimates [20]:

- Severe acute pancreatitis, odds ratio (OR) 2.9 (95% CI 1.8-4.6)
- Systemic complications, OR 2.3 (95% CI 1.4-3.8)
- Local complications, OR 3.8 (95% CI 2.4-6.6)
- Mortality, OR 2.1 (95% CI 1.0-4.8)

Organ failure — Early and persistent organ failure is a reliable indicator of a prolonged hospital stay and increased mortality. In one report, organ failure within 72 hours of admission was associated with the presence of extended pancreatic necrosis and a mortality rate of 42 percent [21]. Several subsequent studies found that the evolution and clinical course of organ failure was a more accurate predictor of adverse outcomes. In one study, persistent and deteriorating (≥ 48 hours) organ failure were associated with mortality rates of 21 and 55 percent, respectively [22]. On the other hand, early organ dysfunction that was not persistent (<48 hours) was associated with a mortality rate of 0 percent. In a second study,

transient organ failure was associated with a mortality rate of 1.4 percent, whereas persistent organ failure had a 35 percent mortality rate [23]. Persistent organ failure is widely accepted as a reliable criterion for severe AP. A systematic review of predictors of persistent organ failure (severe acute pancreatitis) and infected pancreatic necrosis, found blood urea nitrogen useful for prediction of persistent organ failure after 48 hours of admission and procalcitonin for prediction of infected pancreatic necrosis. However, there is no predictor of persistent organ failure within 48 hours of admission [4].

LABORATORY AND RADIOLOGIC PREDICTORS

Hemoconcentration — Acute pancreatitis results in significant third space losses, resulting in hemoconcentration and a high hematocrit. Studies evaluating the hematocrit as a predictor of the severity of AP have produced variable results [24-28]. The discrepancies may be due to differences in values chosen as a cutoff and the time that they were obtained. Despite these differences, it appears that a normal or low hematocrit at admission and during the first 24 hours is generally associated with a milder clinical course.

C-reactive protein — C-reactive protein (CRP) is one of the acute phase reactants made by the liver in response to interleukin-1 and interleukin-6. Levels of CRP above 150 mg/L at 48 hours discriminate severe from mild disease. At 48 hours, CRP above 150 mg/L has a sensitivity, specificity, positive predictive value, and negative predictive value of 80, 76, 67, and 86 percent, respectively, for severe acute pancreatitis [29].

CRP rises steadily in relation to the severity of pancreatitis, is inexpensive to measure, and testing is readily available [30-32]. As a result, we suggest it be used to help predict the severity of pancreatitis, especially at 48 hours.

Blood urea nitrogen — In a large hospital-based cohort, serial blood urea nitrogen (BUN) measurements were the most reliable routine laboratory test to predict the mortality in AP [33]. For every increase in the BUN of 5 mg/dL during the first 24 hours, the adjusted odds ratio (OR) for mortality was 2.2.

A subsequent study by the same group that included 1043 patients found that a BUN level of 20 mg/dL or higher at admission was associated with an increased risk of death compared with a BUN level of less than 20 mg/dL (OR 4.6) [34]. In addition, any increase in BUN at 24 hours was also associated with an increased risk of death (OR 4.3).

Serum creatinine — An elevated serum creatinine within the first 48 hours may predict the

development of pancreatic necrosis. In one study of 129 patients, a peak creatinine of greater than 1.8 mg/dL during the first 48 hours had a positive predictive value of 93 percent for the development of pancreatic necrosis [28]. However, a study from Germany did not find this association, though it did show that a normal creatinine had a high negative predictive value for the development of pancreatic necrosis [35]. The authors suggested that a normal creatinine in the absence of complications obviated the need for an abdominal computed tomographic (CT) scan. The discrepancy between the two studies may have been due to a lower prevalence of pancreatic necrosis in the German study, which could have resulted in a lower positive predictive value.

Other serum markers — Multiple other serum markers have been studied for predicting the severity of pancreatitis including: urinary trypsinogen activation peptide (TAP); procalcitonin; polymorphonuclear elastase; pancreatic-associated protein; amylase; lipase; serum glucose; serum calcium; procarboxypeptidase-B; carboxypeptidase-B activation peptide; serum trypsinogen-2; phospholipase A-2; serum amyloid protein-A; substance P; antithrombin III; platelet activating factor; interleukins 1, 6, and 8; tumor necrosis factor (TNF)-alpha or soluble TNF receptor; angiopoietin-2, and various genetic polymorphisms [28,36-39].

Tests for most of these markers are not widely available, and their test characteristics are incompletely understood. Exceptions are a dipstick test for procalcitonin, an enzyme-linked immunoassay test for urine TAP, and a test for urine anionic trypsinogen, which are likely to become commercially available.

- Procalcitonin is the most rapid general acute-phase reactant. In a validation study, the procalcitonin strip test had an accuracy of 86 percent for predicting severe AP [40].
- TAP is cleaved from the amino-terminal end of trypsinogen when trypsin is activated. TAP is the most studied activation peptide in AP. A European multicenter study found a sensitivity of 58 percent and specificity of 73 percent with urinary TAP within 24 hours of symptom onset [40].

Chest radiographs — A pleural effusion and/or pulmonary infiltrates during the first 24 hours may be associated with necrosis and organ failure [41].

CT scan — CT scan is probably the most frequently used radiologic investigation when severe AP is suspected. It is used to look for pancreatic necrosis and extrapancreatic inflammation. Intravenous contrast-enhanced CT distinguishes between edematous and necrotizing pancreatitis, since areas of necrosis and exudates do not enhance. CT is more

accurate than ultrasonography for the diagnosis of severe pancreatic necrosis (90 versus 73 percent in one report) [42].

After assessment of the patient, a contrast-enhanced CT scan is indicated in patients who are deteriorating or have severe pancreatitis determined clinically and by the Acute Physiology and Chronic Health Examination (APACHE) II score. A CT scan is not required on the first day unless there are other diagnoses being considered. A retrospective analysis of the performance of several CT scoring systems for the severity of AP found that none was statistically superior to the APACHE II or bedside index of severity in acute pancreatitis scoring systems [43]. In addition, it takes time for pancreatic necrosis to develop, and treatment is unlikely to be altered on the basis of CT findings on day one. Although there are some experimental data that suggest ionic contrast may worsen pancreatitis, the association is probably not strong and the information obtained from the CT scan justifies the potential risk.

Perfusion CT can differentiate reversible from irreversible ischemic tissue in the brain and uses less contrast than the conventional contrast-enhanced CT. One report described the utility of perfusion CT in predicting pancreatic necrosis in early stages of AP [44]. Ten of 30 patients with AP had pancreatic ischemia on perfusion CT and nine patients were proven to have pancreatic necrosis subsequently on contrast-enhanced CT. A subtraction color map using subtraction CT with unenhanced and enhanced images also is accurate in the diagnosis of pancreatic necrosis in the early stage of AP [45]. Subtraction CT is easier to perform than perfusion CT and results in less radiation, although perfusion CT requires a smaller amount of iodinated contrast.

MRI and MRCP — Magnetic resonance imaging (MRI) and magnetic resonance cholangiopancreatography (MRCP) are being used increasingly to diagnose AP and to assess its severity [46]. MRI appears to be comparable to CT, if not better, in providing precise information regarding the severity of the disease [47-49]. MRI is as effective as CT in demonstrating the presence and extent of pancreatic necrosis and fluid collections, and is probably superior for indicating the suitability of such collections for nonsurgical drainage [48].

MRI can characterize the "pancreatic necrosis" seen on CT as necrotic pancreatic parenchyma, peripancreatic necrotic fluid collections, or hemorrhagic foci [49]. One study found that MRI was reliable for staging the severity of AP and predicting prognosis with fewer contraindications than CT [50]. It can also detect pancreatic duct disruption, which can occur early in the course of AP.

SCORING SYSTEMS

Many scoring systems have been reported but none has proven to be perfect [5,51]. While they can be useful to group patients for the purpose of interinstitutional comparisons and reporting, none has high accuracy in predicting the severity of AP in a given patient at the bedside.

Many scoring systems (eg, Ranson, Glasgow) take 48 hours to complete, can be used only once, and do not have a high degree of sensitivity and specificity ([table 3](#)) [52-54]. In addition, some have limited utility since they focus on specific complications or are invasive (eg, Leeds diagnostic peritoneal lavage) [52]. As a result, many of these systems are not used routinely.

Ranson's criteria — A score based upon Ranson's criteria is one of the earliest scoring systems for severity in AP [53]. Ranson's criteria consist of 11 parameters. Five of the factors are assessed at admission and six are assessed during the next 48 hours ([table 3](#)) ([calculator 1](#)). A later modification for biliary pancreatitis included only 10 points [55]. Mortality increases with an increasing score. Using the 11 component score, mortality was 0 to 3 percent when the score was <3 , 11 to 15 percent when the score was ≥ 3 , and 40 percent when the score was ≥ 6 [15]. Although the system continues to be used, a meta-analysis of 110 studies found the Ranson score to be a poor predictor of severity [56].

The APACHE II score — The Acute Physiology and Chronic Health Examination (APACHE) II score was originally developed for critically ill patients in intensive care units (ICUs). (See "[Predictive scoring systems in the intensive care unit](#)".)

It has 12 physiologic measures and extra points based upon age and presence of chronic disease. It is probably the most widely studied severity scoring system in AP. It has good negative predictive value and modest positive predictive value for predicting severe AP and can be performed daily. Decreasing values during the first 48 hours suggest a mild attack, while increasing values suggest a severe attack. Studies suggest that mortality is less than 4 percent with a score <8 and is 11 to 18 percent with a score >8 [15,29].

Some limitations of the APACHE II score are that it is complex and cumbersome to use, it does not differentiate between interstitial and necrotizing pancreatitis, and it does not differentiate between sterile and infected necrosis. Finally, it has a poor predictive value at 24 hours [15].

The addition of a body mass index (BMI) score to APACHE II (known as APACHE O) improved the prediction of severe pancreatitis compared with the conventional APACHE II score in one study [57]. One point was added for a BMI of >25 to 30 and two points were added for a BMI >30. However, the improved performance of APACHE O was not validated in a second study [58]. A simplified Acute Physiology Score II developed in ICU patients may be comparable to APACHE II [59]. (See "[Predictive scoring systems in the intensive care unit](#)".)

Several additional variables were added to APACHE II to improve its accuracy leading to the development of APACHE III. Both APACHE II and III scores use physiology, age, and chronic health to calculate prognosis; they differ in total score, the number of physiologic variables (12 for APACHE II versus 17 for APACHE III), and the assessment of chronic health status. However, the APACHE III system does not appear to be as useful as APACHE II for distinguishing mild from severe attack [60].

Systemic inflammatory response syndrome score — As noted above, the presence of the systemic inflammatory response syndrome (SIRS) is associated with increased mortality. A score based upon the systemic inflammatory response syndrome has been developed [61]. Initial studies suggest it can reliably predict the severity of pancreatitis and has the added advantage that it can be applied easily at the bedside every day ([table 4](#)) [22]. In one validation study, mortality rates were 25, 8, and 0 percent in those with persistent SIRS from admission, SIRS at admission but not persistent, and no SIRS, respectively [62]. Another study found that the severity of AP was greater among patients with AP and SIRS on day one, particularly in those with three or four SIRS criteria, compared with those without SIRS on day one [63]. Thus, it appears that the SIRS score is inexpensive, readily available, and compares favorably with other more complicated scores.

BISAP score — Development of the bedside index of severity in acute pancreatitis (BISAP) score was based upon 17,922 cases of AP from 2000 to 2001 and validated in 18,256 cases from 2004 to 2005 [64]. Patients are assigned 1 point for each of the following during the first 24 hours: BUN >25 mg/dL, impaired mental status, SIRS (using the same criteria as the SIRS score, ([table 4](#))), age >60 years, or the presence of a pleural effusion ([calculator 2](#)) [64]. Patients with a score of zero had a mortality of less than one percent, whereas patients with a score of five had a mortality rate of 22 percent. In the validation cohort, the BISAP score had similar test performance characteristics for predicting mortality as the APACHE II score. As is a problem with many of the other scoring systems, the BISAP has not been validated for predicting outcomes such as length of hospital stay, need for ICU care, or need for intervention.

A validation study of the BISAP score that included 185 patients found that its performance was similar to APACHE II, Ranson's criteria, and the CT severity index system [65]. While the BISAP score is meant to be easily calculated at the bedside, it has been noted that to do so is not as simple as was initially reported because four variables need to be considered to determine if SIRS is present [66].

Harmless acute pancreatitis score — The harmless acute pancreatitis score can typically be calculated within 30 minutes of admission and takes into account three parameters: lack of rebound tenderness or guarding, normal hematocrit, and normal serum creatinine. It was derived on a cohort of 394 patients and validated in a cohort of 452 patients [67]. This score correctly identified 200 of 204 patients (98 percent) with a harmless course. Presumably patients were considered likely to have a harmless course if none of the three parameters were present, though this was not clearly defined in the report.

Organ failure-based scores — As noted above, organ failure is a marker of the severity of pancreatitis. While there are several scoring systems for organ failure, they do not directly measure the severity of AP, but rather measure the severity of the organ failure itself. (See '[Organ failure](#)' above.)

This distinction is potentially important as reflected in the limitations of Atlanta classification system described above [68,69]. Organ failure was listed as being present or absent, without specifying the number of organs failing or the severity of each organ failure, issues that are potentially clinically relevant. (See '[Classification of acute pancreatitis](#)' above.)

Organ failure scoring systems such as the Goris multiple organ failure score [70], the Marshall (or multiple) organ dysfunction score [14], the Bernard score [71], the sequential organ failure assessment (SOFA) [72], and the logistic organ dysfunction system score [73] have been described. All these scores take into account the number of organ systems involved and the degree of dysfunction of each individual organ. Some also include the use of inotropic or vasopressor agents, mechanical ventilation, or dialysis [14]. As noted above, the presence of persistent organ failure lasting for more than 48 hours appears to be important [23]. (See '[Organ failure](#)' above.)

CT severity index — A CT severity score (the Balthazar score) has been developed based upon the degree of necrosis, inflammation, and the presence of fluid collections ([table 5](#)) [74]. In an initial validation study, mortality was 23 percent with any degree of pancreatic necrosis and 0 percent with no necrosis. In addition, there was a strong association between necrosis >30 percent and morbidity and mortality [74].

The finding of necrotizing pancreatitis (or even infected necrosis) does not necessarily predict the occurrence of organ failure, but may alter the therapeutic approach [75]. In a retrospective study of 268 patients with AP assessed by the CT severity index, patients with a CT severity index >5 were eight times more likely to die, 17 times more likely to have a prolonged hospital course, and 10 times more likely to undergo necrosectomy than the patients with scores <5 [76]. While necrosis on CT predicts a severe attack, there was no uniform correlation with the extent of necrosis and organ failure and/or mortality. Such a correlation may become more apparent with newer scoring systems that measure the number of failing organ systems and the severity of each failing organ system. (See '[Organ failure-based scores](#)' above.)

Guidelines

- **International Association of Pancreatology (IAP)/American Pancreatic Association (APA)** – IAP/APA guidelines recommend the following for prognostication and predicting the severity of AP [6]:
 - Systemic inflammatory response syndrome (SIRS) is advised to predict severe AP at admission and persistent SIRS at 48 hours.
 - During admission, a three-dimensional approach is advised to predict the outcome of AP combining host risk factors (eg, age, comorbidity, body mass index), clinical risk stratification (eg, persistent SIRS), and monitoring response to initial therapy (eg, persistent SIRS, blood urea nitrogen, creatinine).
- **American College of Gastroenterology (ACG)** – ACG guidelines recommend the following clinical findings associated with a severe course for initial risk assessment [77]:
 - Patient characteristics:
 - Age >55 years
 - Obesity (body mass index >30 kg/m²)
 - Altered mental status
 - Comorbid disease
 - The systemic inflammatory response syndrome:
 - Presence of >2 of the following criteria:
 - Pulse >90 beats/min

- Respirations >20/min or PaCO₂ <32 mm Hg
 - Temperature >38 or <36°C
 - White blood cell count >12,000 or <4,000 cells/mm³ or >10 percent immature neutrophils (bands)
- Laboratory findings:
 - Blood urea nitrogen (BUN) >20 mg/dl
 - Rising BUN
 - Hematocrit (HCT) >44 percent
 - Rising HCT
 - Elevated creatinine
 - Radiology findings:
 - Pleural effusions
 - Pulmonary infiltrates
 - Multiple or extensive extrapancreatic collections

The presence of organ failure and/or pancreatic necrosis defines severe acute pancreatitis.

- **American Gastroenterological Association** – AGA guideline on the initial management of acute pancreatitis [78].

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "[Society guideline links: Acute pancreatitis](#)".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are

comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Beyond the Basics topics (see "[Patient education: Acute pancreatitis \(Beyond the Basics\)](#)")

SUMMARY AND RECOMMENDATIONS

- **Scoring systems** – During the initial 24 hours, severe acute pancreatitis (AP) can be predicted using clinical, laboratory, and radiologic risk factors, many of which have been incorporated into scoring systems such as the systemic inflammatory response syndrome (SIRS) score, the Acute Physiology and Chronic Health Examination (APACHE) II score, the bedside index of severity in acute pancreatitis score, and the computed tomography (CT) severity index. However, these predictive models have low specificity (ie, high false-positive rates), which, when coupled with the low prevalence of severe AP, result in low positive predictive values. However, of the scoring systems, we favor the SIRS score because it is simple, cheap, readily available, and as accurate as any other complex scoring system, especially persistent SIRS. (See '[Scoring systems](#)' above.)

- **Abdominal imaging**

- **Indication and timing of abdominal CT** – We suggest a contrast-enhanced CT be performed in patients considered to have severe AP based upon clinical criteria or possibly the APACHE II score to determine if necrotizing pancreatitis is present.

A CT scan is not required on the first day unless other diagnoses are being considered. It takes time for pancreatic necrosis to develop and thus CT may be normal in the first 48 to 72 hours. Although there are some data that ionic contrast may worsen pancreatitis, the association is probably not strong, and the information obtained from the CT scan justifies the potential risk. (See '[CT scan](#)' above.)

- **Role of MRI** – Magnetic resonance imaging is a useful imaging tool in pancreatitis, especially to identify stones in the bile duct, visualize pancreatic duct, and to distinguish the contents of fluid collections seen on CT. However, the limitations in the setting of acute pancreatitis include high cost and need for prolonged

examination time. (See ['MRI and MRCP'](#) above.)

- **Predictors of severe disease** – Clinical signs and laboratory tests have an adjunctive role in predicting the severity of acute pancreatitis. We agree with the guidelines issued by the American Gastroenterology Association, American College of Gastroenterology, and International Association of Pancreatology/American Pancreatic Association, which suggest that advanced age, comorbidities, body mass index >30, pleural effusion or pulmonary infiltrates, hematocrit >44, blood urea nitrogen (BUN) >20, rising BUN, high creatinine, initial SIRS score ≥ 2 , persistent SIRS, and persistent organ failure are moderately useful as predictors of severe disease. (See ['Laboratory and radiologic predictors'](#) above and ['Guidelines'](#) above.)

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Topic 5639 Version 22.0

GRAPHICS

Revised definitions of morphological features of acute pancreatitis

1. Interstitial edematous pancreatitis

Acute inflammation of the pancreatic parenchyma and peripancreatic tissues, but without recognizable tissue necrosis

Contrast-enhanced computed tomography criteria:

- Pancreatic parenchyma enhancement by intravenous contrast agent
- No findings of peripancreatic necrosis

2. Necrotizing pancreatitis

Inflammation associated with pancreatic parenchymal necrosis and/or peripancreatic necrosis

Contrast-enhanced computed tomography criteria:

- Lack of pancreatic parenchymal enhancement by intravenous contrast agent, and/or
- Presence of findings of peripancreatic necrosis (refer to below—acute peripancreatic fluid collection and walled off necrosis)

3. Acute peripancreatic fluid collection (APFC)

Peripancreatic fluid associated with interstitial edematous pancreatitis with no associated peripancreatic necrosis. This term applies only to areas of peripancreatic fluid seen within the first four weeks after onset of interstitial edematous pancreatitis and without the features of a pseudocyst.

Contrast-enhanced computed tomography criteria:

- Occurs in the setting of interstitial edematous pancreatitis
- Homogeneous collection with fluid density
- Confined by normal peripancreatic fascial planes
- No definable wall encapsulating the collection
- Adjacent to pancreas (no intrapancreatic extension)

4. Pancreatic pseudocyst

An encapsulated collection of fluid with a well-defined inflammatory wall usually outside the pancreas with minimal or no necrosis. This entity usually occurs more than four weeks after onset of interstitial edematous pancreatitis to mature.

Contrast-enhanced computed tomography criteria:

- Well circumscribed, usually round or oval
- Homogeneous fluid density
- No non-liquid component
- Well-defined wall (ie, completely encapsulated)
- Maturation usually requires >4 weeks after onset of acute pancreatitis; occurs after interstitial edematous pancreatitis

5. Acute necrotic collection (ANC)

A collection containing variable amounts of both fluid and necrosis associated with necrotizing pancreatitis; the necrosis can involve the pancreatic parenchyma and/or the peripancreatic tissues

Contrast-enhanced computed tomography criteria:

- Occurs only in the setting of acute necrotizing pancreatitis
- Heterogeneous and non-liquid density of varying degrees in different locations (some appear homogeneous early in their course)
- No definable wall encapsulating the collection
- Location—intrapancreatic and/or extrapancreatic

6. Walled-off necrosis (WON)

A mature, encapsulated collection of pancreatic and/or peripancreatic necrosis that has developed a well-defined inflammatory wall. WON usually occurs >4 weeks after onset of necrotizing pancreatitis.

Contrast-enhanced computed tomography criteria:

- Heterogeneous with liquid and non-liquid density with varying degrees of loculations (some may appear homogeneous)
- Well-defined wall, that is, completely encapsulated
- Location—intrapancreatic and/or extrapancreatic
- Maturation usually requires four weeks after onset of acute necrotizing pancreatitis

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Modified Marshall scoring system for organ dysfunction

Organ system	Score				
	0	1	2	3	4
Respiratory (PaO ₂ /FiO ₂)	>400	301-400	201-300	101-200	≤101
Renal*					
(serum creatinine, micromol/L)	≤134	134-169	170-310	311-439	>439
(serum creatinine, mg/dL)	<1.4	1.4-1.8	1.9-3.6	3.6-4.9	>4.9
Cardiovascular (systolic blood pressure, mmHg) [¶]	>90	<90, fluid responsive	<90, not fluid responsive	<90, pH <7.3	<90, pH <7.2
For nonventilated patients, the FiO ₂ can be estimated from below:					
Supplemental oxygen (L/min)	FiO₂ (percent)				
Room air	21				
2	25				
4	30				
6-8	40				
9-10	50				

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

* A score for patients with pre-existing chronic renal failure depends on the extent of further deterioration of baseline renal function. No formal correction exists for a baseline serum creatinine ≥134 micromol/L or ≥1.4 mg/dL.

¶ Off inotropic support.

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Ranson criteria to predict severity of acute pancreatitis

0 hours	
Age	>55
White blood cell count	>16,000/mm ³
Blood glucose	>200 mg/dL (11.1 mmol/L)
Lactate dehydrogenase	>350 U/L
Aspartate aminotransferase (AST)	>250 U/L
48 hours	
Hematocrit	Fall by ≥ 10 percent
Blood urea nitrogen	Increase by ≥ 5 mg/dL (1.8 mmol/L) despite fluids
Serum calcium	<8 mg/dL (2 mmol/L)
pO ₂	<60 mmHg
Base deficit	>4 MEq/L
Fluid sequestration	>6000 mL

The presence of 1 to 3 criteria represents mild pancreatitis; the mortality rate rises significantly with four or more criteria.

Adapted from Ranson, JHC, Rifkind, KM, Roses, DF, et al, Surg Gynecol Obstet 1974; 139:69.

Graphic 56775 Version 1.0

Defining features of systemic inflammatory response syndrome (SIRS)

Two or more of the following conditions:
Temperature $>38.3^{\circ}\text{C}$ or $<36.0^{\circ}\text{C}$
Heart rate of >90 beats/minute
Respiratory rate of >20 breaths/minute or PaCO_2 of <32 mmHg
WBC count of $>12,000$ cells/ mm^3 , <4000 cells/ mm^3 , or >10 percent immature (band) forms

WBC count: white blood cell count.

Data from: Annane D, Bellissant E, Cavaillon JM. Septic shock. Lancet 2005; 365:63.

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CT findings and grading of acute pancreatitis (CT severity index [CTSI])

Grading based upon findings on unenhanced CT		
Grade	Findings	Score
A	Normal pancreas - normal size, sharply defined, smooth contour, homogeneous enhancement, retroperitoneal peripancreatic fat without enhancement	0
B	Focal or diffuse enlargement of the pancreas, contour may show irregularity, enhancement may be inhomogeneous but there is no peripancreatic inflammation	1
C	Peripancreatic inflammation with intrinsic pancreatic abnormalities	2
D	Intrapancreatic or extrapancreatic fluid collections	3
E	Two or more large collections of gas in the pancreas or retroperitoneum	4
Necrosis score based upon contrast-enhanced CT		
Necrosis (%)		Score
0		0
<33		2
33-50		4
≥50		6

CT severity index equals unenhanced CT score plus necrosis score: maximum = 10, ≥ 6 = severe disease.

CT: computed tomography.

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

Graphic 68191 Version 3.0

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