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## Diagnosis and Risk Stratification of Acute Pancreatitis

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### **DIAGNOSIS OF ACUTE PANCREATITIS**

Patients with acute pancreatitis (AP) usually present with sudden onset of abdominal pain, nausea, and vomiting. Approximately 80% of patients have interstitial pancreatitis with mild-to-moderate symptoms, and 20% have life-threatening necrotizing disease. Careful clinical assessment and the judicious use of biochemical tests and radiological imaging enables the practitioner to differentiate AP from other causes of acute abdomen and to assess the severity of disease (1–7).

#### ***History and Physical Exam***

AP is typically characterized by abdominal pain located in the epigastric or supraumbilical regions, often radiating to the mid-thoracic portion of the back. Pain usually reaches maximum intensity within 20 minutes but may have a more gradual onset. The pain from AP is usually sharp, constant, lasts hours to days, and is severe enough to force the patient to visit the emergency room. In mild AP, the pain may decrease when sitting or leaning forward in comparison to lying flat.

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Nausea and vomiting with or without low-grade fever are the most commonly associated symptoms (1,4,5).

A recent history of binge drinking may be frequently elicited in patients with alcohol-induced pancreatitis. The concomitant presence of jaundice and high-grade fever strongly suggests choledocholithiasis as the etiology of AP, complicated by coexistent cholangitis (1–6). Less commonly, respiratory failure, confusion, and even coma are the main presenting features, which are frequently manifestations of severe necrotizing pancreatitis. In rare cases, abdominal pain may be absent, leading to a delayed or missed diagnosis (1).

The usual findings on a physical examination are abdominal distension, tenderness, guarding, and absent bowel sounds. Fever associated with AP is generally low grade. High-grade temperature may indicate the development of infected pancreatic necrosis and associated fluid collection or cholangitis, particularly if jaundice is present (1,2,5,6).

Severe acute pancreatitis (SAP) is often complicated by massive loss of fluid into the retroperitoneal spaces. Tachycardia and hypotension are some of the earliest clues for a moderate-to-severe attack of pancreatitis and are markers for significant early depletion of intravascular volume. These may soon progress to hypovolemic shock caused by increased vascular permeability, vasodilatation, and hemorrhage (1). Tachypnea and dyspnea are also common in severe pancreatitis, owing to splinting from the subdiaphragmatic inflammatory process, associated pleural effusions, or pulmonary capillary leak syndrome (adult respiratory distress syndrome). Pleural effusions are mainly found on the left side but can be bilateral.

Rare clinical findings include ecchymoses of the umbilicus or flanks, peripheral subcutaneous fat necrosis, and polyarthritides. Classically, dark skin discoloration of the flanks and periumbilical areas because of hemorrhage is described with severe and hemorrhagic pancreatitis; however these physical findings may result from any type of retroperitoneal bleeding (6).

### ***Laboratory Tests***

The diagnosis of AP is usually suspected based on the appropriate clinical features and is confirmed by laboratory and imaging tests. Leakage of pancreatic enzymes into the circulation is a hallmark of AP. Although amylase and lipase constitute a small fraction of all pancreatic enzymes, they are the easiest and the quickest enzymes to measure. Typically, the elevation of serum amylase in AP is above threefold of the normal values. Amylase levels are usually increased within a few hours of disease onset, but they may be cleared from the serum rather quickly. Serum amylase usually remains elevated for 3–5 days in uncomplicated

Table 1  
Causes of Increased Serum Amylase Activity

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Pancreatic diseases
Acute pancreatitis
Pancreatic cancer
Abdominal emergencies
Acute cholecystitis
Common bile duct obstruction
Perforated viscous
Intestinal ischemia
Acute appendicitis
Ruptured ectopic pregnancy and acute salpingitis
Salivary gland diseases
Renal insufficiency
Macroamylasemia
Diabetic ketoacidosis
HIV infection/AIDS
Sphincter Oddi stenosis or spasm
Drugs: Morphine

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AP. Because many conditions can cause hyperamylasemia (Table 1), the specificity of elevated serum amylase level is less than 70%. Very high elevations of serum amylase (more than fivefold normal), however, are rarely associated with diseases other than AP. Elevations of three- to fivefold normal are commonly seen in the absence of acute pancreatitis in patients with renal failure, as a result of decreased clearance of the enzyme. Measurements of urinary amylase and the amylase-to-creatinine ratio may be helpful to distinguish AP from other causes of hyperamylasemia, but such measurements are infrequently employed (2–6).

Serum amylase isoenzyme measurements may improve the diagnostic accuracy of serum amylase alone. In healthy people, less than half of all circulating amylase originates in the pancreas, whereas the remainder is of salivary origin. Serum pancreatic isoamylase (P-isoamylase) accounts for the elevated total serum amylase level in AP and tends to persist for several days. However, pancreatic isoamylase can be elevated in some other gastrointestinal disorders and in renal insufficiency, making it difficult to diagnose AP based on P-isoamylase levels alone without additional diagnostic parameters (2,5,8).

The elevation of serum lipase generally parallels the serum amylase level in AP. However, the serum lipase level often remains elevated longer, making it more useful to diagnose pancreatitis after symptoms

have subsided. Lipase is considered more specific than amylase for pancreatic tissue injury, despite that lipase is also produced by numerous other gastrointestinal tissues. Another potential advantage of lipase is that it is generally not elevated in diabetic ketoacidosis or macroamylasemia (1).

Both amylase and lipase are widely available and are, in general, rapidly available from hospital laboratories. In practice, combining the measurement of serum amylase and lipase somewhat enhances the diagnostic accuracy for AP. A normal amylase or lipase level makes the diagnosis of AP unlikely, except in the presence of hyperlipidemia. Very high levels of serum triglyceride (one of the causes of AP) can interfere with the laboratory assay for both amylase and lipase; dilution of the serum may be necessary in this situation to reliably measure the elevations of amylase or lipase. In some patients with chronic pancreatitis, acute abdominal pain can be the result of focal acute inflammation of the gland, and serum amylase and lipase levels may remain normal (5,6). It is important to note that a correlation has not been found between the degree or trend of serum amylase and lipase elevation with the amount of structural damage of the pancreas or severity of AP (9).

Pancreatic enzymes, such as serum trypsin, chymotrypsin, elastase, ribonuclease, and phospholipase A<sub>2</sub> have been all reported to be elevated in AP, but assays to measure these enzymes are not readily available for clinical use, and their specificity has not been defined (2,5,6,9).

The use of other clinically available laboratory tests may have a role in determining the etiology of AP. For example, elevated bilirubin and hepatic transaminases, particularly alanine aminotransferase more than 80 IU/L should raise the suspicion of gallstone pancreatitis (1–5; *see* Chapter 3).

## *Imaging*

### ULTRASONOGRAPHY

Transabdominal ultrasonography is widely available, relatively inexpensive, and quite safe. Unfortunately, pancreatic imaging by ultrasound has limitations from overlying bowel gas and surrounding fat planes, which tend to be exaggerated in the acutely inflamed pancreas owing to ileus and peripancreatic edema. Thus the sensitivity and specificity of this modality for diagnosing AP is low (1). Nonetheless, transabdominal ultrasonography is useful in the early stages of AP to search for gallbladder stones or sludge, evaluate for dilation of the common bile duct caused by choledocholithiasis, and analyze for other possible causes of severe abdominal pain.

## COMPUTED TOMOGRAPHY SCAN

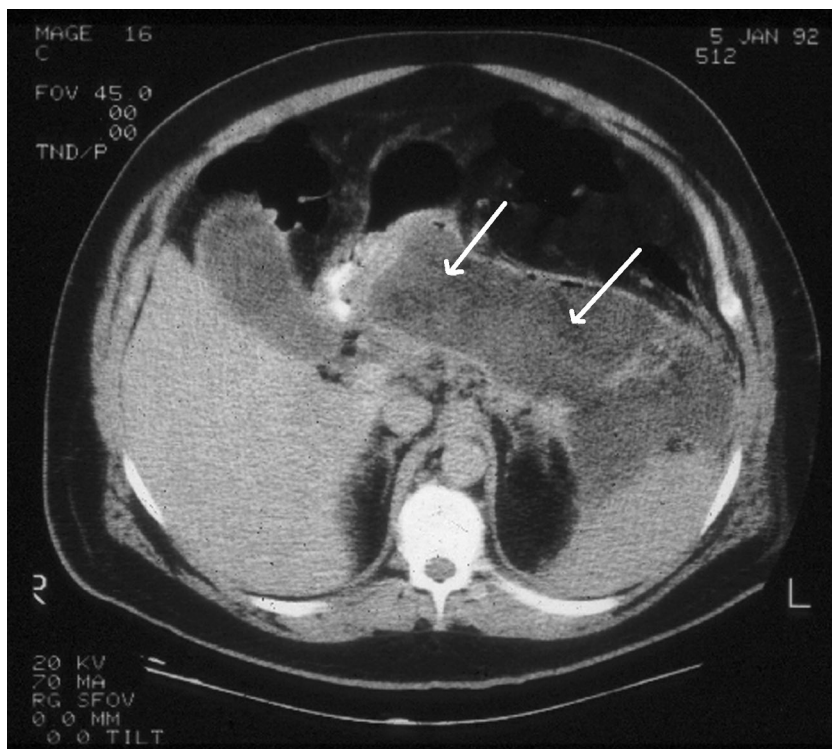
The computed tomography (CT) scan, particularly when done with helical or multidetector technology, is a valuable tool in the diagnosis and management of AP. However, not every patient with AP requires a CT scan. CT is mainly indicated if the initial diagnosis is in doubt or for prognostic purposes in severely ill patients as in the section on Risk Stratification (4). The role of CT is both to document the appropriate findings that confirm the diagnosis of AP and to exclude other intra-abdominal catastrophes that can mimic AP (e.g., a perforated viscus). CT scan findings, which support the diagnosis of AP, include diffuse or segmental enlargement of the pancreas, irregularity of the pancreatic contour with obliteration of the peripancreatic fat planes, areas of decreased density within the pancreas, and ill-defined fluid collections in the pancreas or outside the gland in the lesser sac or pararenal spaces. The frequency of these findings varies according to the severity of pancreatitis, and these findings do not require intravenous administration of contrast material to be identified.

Intravenous contrast-enhanced computed tomography (CECT) is mainly used to differentiate pancreatic necrosis from interstitial pancreatitis or to monitor for pancreatitis complications in selected cases (i.e., to assist in estimating prognosis or managing patients with AP, rather than simply confirming a diagnosis). Normal CT findings have been reported in 24–67% of patients with mild AP (2).

Controversy exists as to whether intravenous contrast early in the clinical course exacerbates the severity of AP. Although deleterious effects of intravenous contrast have been observed in animal models of experimental pancreatitis, studies in humans have yielded conflicting results (10). Many authors agree that CECT scans are unnecessary in patients with mild AP (*see* Risk Stratification in Acute Pancreatitis section) and should be reserved for those patients with a more complicated clinical course. Additionally, early CECT may underestimate the degree of pancreatic necrosis that may develop over time from the disruption of pancreatic microvascular circulation that usually occurs in the first 12–24 hours of SAP (11,12). At present, it is recommended that CECT be obtained 3–4 days after the onset of SAP for optimal assessment of pancreatic necrosis (8).

## MAGNETIC RESONANCE IMAGING

Currently, magnetic resonance imaging (MRI) has no advantage over CT scan in the management of AP. MRI has a comparable specificity and sensitivity for diagnostic and severity assessment of AP (1,2). Its cost, availability, and contraindication in patients with metallic implants has limited the application of MRI in AP to date.



**Fig. 1.** A computed tomography scan demonstrating a large area of necrosis as evidenced by the lack of contrast enhancement (arrows) after intravenous contrast administration.

### ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY

Endoscopic retrograde cholangiopancreatography (ERCP) has no role in diagnosing AP. Therapeutic application of ERCP in moderate-to-severe acute gallstone pancreatitis has been shown by several controlled clinical trials to lower morbidity and mortality when compared to traditional medical treatment alone (*see* Chapter 3). ERCP is also utilized in the differential diagnosis and elective treatment of recurrent unexplained pancreatitis secondary to sphincter Oddi dysfunction, pancreatic divisum, and microlithiasis (13–15).

### ENDOSCOPIC ULTRASOUND

The diagnostic role of endoscopic ultrasound (EUS) in AP is still evolving; it is not readily available in all institutions. In recent studies, the immediate application of EUS for suspected biliary AP may aid in the diagnosis of gallstone pancreatitis, thereby helping to triage

patients for therapeutic ERCP with endoscopic sphincterotomy and stone removal (16).

## RISK STRATIFICATION IN ACUTE PANCREATITIS

Early evaluation of AP severity is essential to allow the clinician to predict the patient's clinical course, estimate prognosis, and determine the need for intensive care unit admission. Severe pancreatitis can be defined by various systems that predict complications and mortality or by the development of the complication itself. Thus, there is a difference between a predictive system that suggests complications may develop and the actual development of a complication. (This issue is discussed in more detail in Chapter 6.) This section focuses on methods to predict morbidity and mortality. Severe pancreatitis can be predicted by clinical criteria, multiple factor scoring systems, serum markers, and radiographic features. The ability of a seasoned clinician's ability to detect severe pancreatitis is similar to the accuracy of the multiple factor scoring systems. Several of these scoring systems have been developed to assist the clinician in the assessment of the severity of AP. The most commonly used systems are the Ranson criteria, the modified Glasgow scoring system, and the Acute Physiology And Chronic Health Evaluation II (APACHE II; 2,8,16–19).

The Ranson Criteria and the Modified Glasgow System rely on a collection of clinical and biochemical variables measured within the first 48 hours of admission, as shown in Table 2. Clearly, from looking at these systems, many of the variables are factors that any clinician would be attuned to in managing a critically ill patient, and the scoring systems merely place these variables within a numerical framework. Using these systems, it is only possible to predict severity after 48 hours have passed. Higher Ranson or Glasgow scores predict severe disease with reasonable sensitivity. Mortality is less than 5% in patients with Ranson score of 0, in comparison to 10% for those with a criteria of 3–5, and 60% for those with a Ranson score greater than 6. Thus, many patients with higher Ranson scores do not die and, in fact, do not develop organ failure or other complications. The same is true for the modified Glasgow scoring system. Therefore, the Ranson and modified Glasgow scoring systems lack specificity. It should also be noted that there are separate Ranson scoring systems for alcohol-induced and biliary pancreatitis, and the total score cannot be calculated unless all factors are measured after 48 hours of observation. The most important roles of the Ranson and Glasgow scoring may be to exclude severe disease. A Glasgow or Ranson score of 0 or 1 virtually guarantees that complications will not develop and that mortality will be negligible. A second



Table 2  
Variables of the Ranson Criteria and Modified Glasgow System

<i>Ranson Criteria</i>		<i>Modified Glasgow System</i>	
For Acute Non-Gallstone Pancreatitis			
Upon admission:		Arterial PO <sub>2</sub>	<60 mmHg
1. Age	>55 years	Serum albumin	<3.2 g/dL
2. WBC	>16,000/mm <sup>3</sup>	Serum Ca	<8 mg/dL
3. Glucose	>200 mg/dL	WBC	>15,000/mm <sup>3</sup>
4. LDH	>350 IU/L	AST	>200 IU/L
5. AST	>250 IU/L	LDH	>600 IU/L
Within 48 hours:		Glucose	>180 mg/dL
1. Drop in HCT	>10%	BUN	>45 mg/dL
2. Serum Ca	<8 mg/dL		
3. Base deficit	>4 mEq/L		
4. Increase BUN	>5 mg/dL		
5. Fluid deficit	>6 L		
6. Arterial PO <sub>2</sub>	<60 mmHg		

For Acute Gallstone Pancreatitis			
Upon admission:			
1. Age	>70 years		
2. WBC	>18,000/mm <sup>3</sup>		
3. Glucose	>220 mg/dL		
4. LDH	>400 IU/L		
5. AST	>440 IU/L		
Within 48 hours:			
1. Drop in HCT	>10%		
2. Serum Ca	<8 mg/dL		
3. Base deficit	>5 mEq/L		
4. Increase BUN	>2 mg/dL		
5. Fluid deficit	>6 L		
6. Arterial PO <sub>2</sub>	<60 mmHg		

HCT, hemoconcentration; WBC, white blood count; LDH, lactate dehydrogenase; AST, aspartate aminotransferase; BUN, blood urea nitrogen.

important use of these scoring systems is for clinical research, in characterizing disease severity for comparison between studies.

The APACHE II scoring system is considered more specific and accurate when compared to clinical assessment and Ranson/modified Glasgow system (Table 3). The APACHE II may be applied at any time point in the course of disease, which is an advantage over the Ranson and Glasgow criteria. The APACHE II system is quite complex (9), making it unwieldy for everyday clinical use. Many free downloadable programs for PDA use are available on the Web, which has markedly



Table 3  
The APACHE II Severity of Disease Classification System

<i>Physiologic variable</i>	<i>High abnormal range</i>					<i>Low abnormal range</i>				<i>Points</i>
	+ 4	+ 3	+ 2	+ 1	0	+ 1	+ 2	+ 3	+ 4	
Temperature—rectal (°C)	≥41°	39–40.9°		38.5–38.9°	36–38.4°	34–35.9°	32–33.9°	30–31.9°	≤29.9°	
Mean arterial pressure—mmHg	≥160	130–159	110–129		70–109		50–69		≤49	
Heart rate (ventricular response)	≥180	140–179	110–139		70–109		55–69	40–54	≤39	
Respiratory rate (nonventilated or ventilated)	≥50	35–49		25–34	12–24	10–11	6–9		≤5	
Oxygenation: A-aDO <sub>2</sub> or PaO <sub>2</sub> (mmHg)	≥500	350–499	200–349		<200					
a. FIO <sub>2</sub> ≥ 0.5 record A-aDO <sub>2</sub>										
b. FIO <sub>2</sub> < 0.5 record PaO <sub>2</sub>					PO <sub>2</sub> >70	PO <sub>2</sub> 61–70		PO <sub>2</sub> 55–60	PO <sub>2</sub> <55	
Arterial pH (preferred)	≥7.7	7.6–7.69		7.5–7.59	7.33–7.49		7.25–7.32	7.15–7.24	<7.15	
Serum HCO <sub>3</sub> (venous mEq/L) (not preferred, but may use if no ABGs)	≥52	41–51.9		32–40.9	22–31.9		18–21.9	15–17.9	<15	
Serum sodium (mEq/L)	≥180	160–179	155–159	150–154	130–149		120–129	111–119	≤110	
Serum potassium (mEq/L)	≥7	6–6.9		5.5–5.9	3.5–5.4	3–3.4	2.5–2.9		<2.5	
Serum creatinine (mg/dL) Double point score for acute renal failure	≥3.5	2–3.4	1.5–1.9		0.6–1.4		<0.6			
Hematocrit (%)	≥60		50–59.9	46–49.9	30–45.9		20–29.9		<20	

*Continued*

Table 3 (Continued)

	High abnormal range					Low abnormal range				
Physiologic variable	+ 4	+ 3	+ 2	+ 1	0	+ 1	+ 2	+ 3	+ 4	Points
White blood count (total/mm <sup>3</sup> ) (in 1000s)	≥40		20–39.9	15–19.9	3–14.9		1–2.9		<1	
Glasgow coma score (GCS) Score = 15 minus actual GCS										
A. Total acute physiology score (sum of 12 above points)										
B. Age points (years)	≤44 = 0;		45–54 = 2;		55–64 = 3;		65–74 = 5;		≥75 = 6	
C. Chronic health points (see below)										
Total APACHE II score (add together the points from A + B + C)										

Chronic Health Points: If the patient has a history of severe organ system insufficiency or is immunocompromised as defined below, assign points as follows:

- 5 points for nonoperative or emergency postoperative patients
- 2 points for elective postoperative patients

*Definitions:* organ insufficiency or immunocompromised state must have been evident prior to this hospital admission and conform to the following criteria: *Liver*—biopsy proven cirrhosis and documented portal hypertension; episodes of past upper gastrointestinal bleeding attributed to portal hypertension; or prior episodes of hepatic failure/encephalopathy/coma. *Cardiovascular*—New York Heart Association Class IV. *Respiratory*—Chronic restrictive, obstructive, or vascular disease resulting in severe exercise restriction (i.e., unable to climb stairs or perform household duties; or documented chronic hypoxia, hypercapnia, secondary polycythemia, severe pulmonary hypertension (>40 mmHg), or respirator dependency. *Renal*—receiving chronic dialysis. *Immunocompromised*—the patient has received therapy that suppresses resistance to infection (e.g., immunosuppression, chemotherapy, radiation, long term or recent high-dose steroids, or has a disease that is sufficiently advanced to suppress resistance to infection, e.g., leukemia, lymphoma, AIDS).

#### Interpretation of score

0–4 = ~4% death rate	10–14 = ~15% death rate	20–24 = ~40% death rate	30–34 = ~75% death rate
5–9 = ~8% death rate	15–19 = ~25% death rate	25–29 = ~55% death rate	Over 34 = ~85% death rate

ABGs, arterial blood gases.

Table 4  
Computed Tomography Grading System

Grade A:	Normal findings
Grade B:	Focal or diffuse pancreatic enlargement
Grade C:	Inflammation of the pancreas and pancreatic fat
Grade D:	Peripancreatic fluid collection in single location usually within the anterior para-renal space
Grade E:	Two or more fluid collections or the presence of peripancreatic gas

improved the ease in using the APACHE II scoring system. Predicted SAP is defined by a Ranson score of 3 or greater or an APACHE II score of 8 or greater (8,9). Actual SAP is defined by the presence of organ failure or local pancreatic complications (e.g., necrosis, infected necrosis, pseudocyst, and abscess).

CT has also become routinely used in the prediction and determination of disease severity. The initial CT grading system, which did not require intravenous contrast administration, was developed by Balthazar and Ranson (Table 4; 20). However, using CT alone also has a relatively high false-positive rate (i.e., many patients with grade C and even D pancreatitis recover without developing organ failure or dying). Combining the CT grading system with Ranson prognostic signs further improves the prognostic capacity when compared to either system alone. Patients with grade D or E are almost certain to develop complications, and they have a significantly increased risk of mortality, and this risk is augmented by the coexistence of a high Ranson score. Those patients with grade C pancreatitis and a Ranson score less than 3 routinely do well, whereas those with grade C pancreatitis and a Ranson score more than 3 are much more likely to develop complications and/or die. A grade of A or B strongly predicts an uncomplicated outcome (1,20). These grading systems are based on non-CECT scans. CECT can also be used to determine the presence of pancreatic necrosis. Interstitial pancreatitis (the absence of necrosis) is defined by homogeneous and uniform intravenous contrast enhancement of the pancreas, which requires rapid scanning over the pancreas timed to the infusion of intravenous contrast. Necrosis is defined by inhomogeneous enhancement with intravenous contrast, especially when large areas of the pancreas are entirely devoid of enhancement. Pancreatic necrosis *per se* is not always associated with other clinical features of severe disease (e.g., organ failure or infected necrosis), but the presence of necrosis markedly increases the chance of developing these severe clinical markers. Particularly, pancreatic necrosis puts

patients at risk for infection of the devitalized tissue, one of the most severe complications of AP (*see* Chapter 7). CT scans with intravenous contrast enhancement is our only method currently available to identify necrosis.

Given that the multiple factor scoring systems are complex and that CT scans are expensive, there has been continued interest in identifying simpler or less expensive methods to predict severity. Several clinical and serum markers of disease severity have been proposed, which include routine laboratory tests and novel markers of disease severity. Despite the diagnostic importance of elevated serum amylase and lipase in AP, numerous studies have demonstrated that elevated levels of these enzymes have no prognostic value in AP (2,8,9). This is the reason why they are excluded in any AP severity scoring system. Hemoconcentration more than 44% at presentation has been demonstrated by several investigators to be a reasonably accurate early marker that predicts pancreatic necrosis and organ failure (21–23). In contrast, Whitcomb et al. showed that an admission hematocrit of 40% or below predicts a low risk of pancreatic necrosis and may reduce the need for diagnostic CT scans (24).

More novel serum tests have also been evaluated. C-reactive protein (an acute-phase reactant) is cheap, widely available, and commonly used in Europe as a measure of severity. A level of 150 mg/L of C-reactive protein has been proposed as a criterion for distinguishing mild AP from SAP (9). Other markers, such as trypsinogen activation peptide, interleukin-6, and polymorphonuclear elastase, have been shown in research studies to be of value to predict severe necrotizing pancreatitis, but commercial assays are not yet available for clinical use (2–7).

Clinical or demographic features may also predict disease severity. Obesity has been shown in several studies to be a risk factor for the severe outcome of AP, and it is associated with an increased risk of mortality (9). Advanced age and comorbid diseases are also risk factors for morbidity and mortality from AP. Other clinical parameters like hypovolemic shock, massive pleural effusion, prolonged hypoxia, and body echymosis are indicative of a complicated course and a higher risk of mortality (1).

Many steps have already been taken to guide the clinician's goal of predicting the severity of AP. The ability to accurately predict outcome would allow the improved use of intensive and intermediate care unit beds and would allow specific therapy (once available) to be directed at those patients most likely to benefit. However, the ideal grading system or the predictive marker of choice does not yet exist. Careful and repeated clinical evaluation by skillful clinicians remains an important part of detecting complications early. Multiple factor

scoring systems are useful adjuncts but remain complex, difficult to use, and all have a high false-positive rate. CT scans are widely used and seem to provide the best addition to clinical assessment, both to confirm the diagnosis and/or rule out alternative diagnoses and estimate the disease severity.

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