

# Severe Acute Pancreatitis and Necrotizing Pancreatitis



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## KEYWORDS

• Acute pancreatitis • Necrotizing pancreatitis • Mortality • Morbidity

## KEY POINTS

- Acute pancreatitis varies widely in its clinical presentation, from clinically negligible to precipitously fatal despite any intervention.
- Necrotizing pancreatitis is a manifestation of severe acute pancreatitis and is associated with significant morbidity and mortality.
- Having established the diagnosis of pancreatic necrosis, goals of appropriately aggressive resuscitation should be established and adhered to in a multidisciplinary approach involving medical and surgical intensive care.
- In all cases of necrotizing pancreatitis, a multidisciplinary approach is needed, using endoscopic techniques and/or percutaneous drainage.
- Open surgery should be reserved for failure of less invasive techniques.

## INTRODUCTION

Acute pancreatitis results in nearly 250,000 annual admissions at a cost of approximately \$2.2 billion.<sup>1,2</sup> In most cases, acute pancreatitis represents a mild, self-limited disease, but in 15% to 25%, severe acute pancreatitis (SAP) develops, manifested with pancreatic parenchymal and/or peripancreatic tissue necrosis.<sup>3</sup> Pancreatic necrosis accounts for substantial additional morbidity, with mortality remaining as high as 10% to 20% despite advances in critical care.<sup>4,5</sup>

### **Severe Acute Pancreatitis**

Acute pancreatitis is best defined clinically by a patient presenting with 2 of the following 3 criteria: (1) symptoms (eg, epigastric pain) consistent with pancreatitis;

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Disclosures: None.

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(2) a serum amylase or lipase level greater than 3 times the laboratory's upper limit of normal; and (3) radiologic imaging consistent with pancreatitis, usually computed tomography (CT) or MRI. Once the diagnosis of acute pancreatitis is established, patients are classified based on disease severity. The Atlanta Criteria revision of 2012 (**Box 1**)<sup>6</sup> classifies severity as mild, moderately severe, or severe. Severe acute pancreatitis is defined by persistent single or multiorgan failure (lasting >48 hours). Local complications include peripancreatic fluid collections, pancreatic and peripancreatic necrosis (sterile or infected), pseudocyst, and wall-off necrosis (sterile or infected; **Figs. 1** and **2**).<sup>6</sup>

Other acceptable markers of severe pancreatitis include 3 or more of Ranson's II criteria for non-gallstone pancreatitis, and an Acute Physiology and Chronic Health Evaluation score greater than 8. It is important to use precise terms in describing the anatomic complications of acute pancreatitis. Although patients with interstitial pancreatitis have a normally perfused gland, manifesting on contrast-enhanced CT as normal, bright appears as an indication of flow throughout the gland; patients with necrotizing pancreatitis (NP) have greater than 30% of the gland that is not perfused, with low attenuation. Pancreatic necrosis is consistent with focal or diffuse nonviable pancreatic parenchyma and is usually accompanied by peripancreatic fat necrosis. Pancreatic necrosis can be sterile or infected. Peripancreatic necrosis describes necrotic fatty and tissue debris around the pancreas; it is more important to surgeons because this is typically not appreciated on imaging. NP (pancreatic necrosis) is defined, in the absence of laparotomy or autopsy, by the presence of greater than 30% of nonenhancement of the pancreas on a contrast-enhanced CT (or MRI with gadolinium). The determination that a patient has pancreatic necrosis has clinical implications because the morbidity and mortality of NP are higher than that associated with interstitial pancreatitis. Patients with NP may seem ill with single- or multiorgan failure or may seem well with no evidence of organ failure.

More recently, the 2012 revised Atlanta classification for acute pancreatitis addressed several lingering deficiencies and further developed consistent terminology for acute pancreatitis and its sequelae as highlighted in **Table 1**.<sup>7</sup> The term mild acute pancreatitis is now defined as pancreatitis without organ failure (defined in later discussion, such as renal or pulmonary failure), or complications (such as necrosis or

#### Box 1

#### 2012 Atlanta classification revision of acute pancreatitis

##### *Definitions of grades and severity of acute pancreatitis*

##### Mild acute pancreatitis

- No organ failure
- No local or systemic complications

##### Moderately SAP

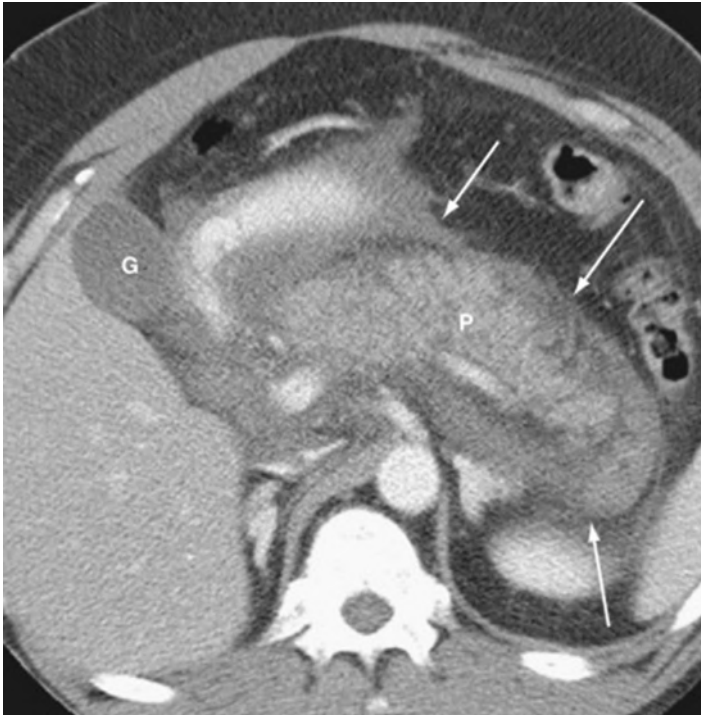
- Transient organ failure (<48 hours) and/or
- Local or systemic complications<sup>a</sup> without persistent organ failure

##### Severe acute pancreatitis

- Persistent organ failure (>48 hours)—single organ or multiorgan

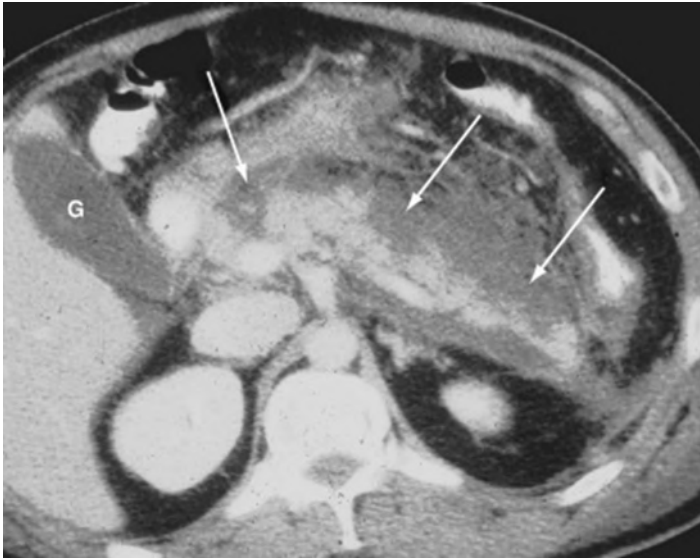
<sup>a</sup> Local complications are peripancreatic fluid collections, pancreatic necrosis, and peripancreatic necrosis (sterile or infected), pseudocyst, and WON (sterile or infected).

From Banks PA, Bollen TL, Dervenis C, et al. Classification of acute pancreatitis—2012: revision of classification and definitions by international consensus. *Gut* 2013;62:102–11; with permission.



**Fig. 1.** CT showing acute interstitial pancreatitis with diffuse swelling of the pancreas (P) and peripancreatic inflammatory changes (*arrows*). The pancreas was well perfused without evidence of necrosis. G, gallbladder. (From Tenner S, Steinberg WM. Acute pancreatitis. In: Feldman M, Friedman LS, Brandt LJ, eds. *Sleisenger and Fordtran's gastrointestinal and liver disease pathophysiology, diagnosis, management*. 10th edition. Philadelphia: Elsevier; 2016; with permission.)

pseudocysts), as discussed later. Moderately severe acute pancreatitis is defined by organ failure lasting less than 48 hours, or by local complications. The term SAP is reserved for cases in which organ failure lasts greater than 48 hours. According to the current classification of acute pancreatitis, interstitial edematous pancreatitis (IEP) is defined by the lack of pancreatic or peripancreatic necrosis on imaging and is distinguished from NP, which is subdivided into 3 categories: parenchymal necrosis, peripancreatic necrosis, or combined necrosis, all 3 of which may be infected or sterile. The disease process is further separated into an early phase and a late phase, with definition of local complications based on characteristics of collections of fluid and necrosis. In the setting acute pancreatitis, typically IEP, a peripancreatic fluid collection occurring within the first 4 weeks is termed an acute peripancreatic fluid collection (APFC) and is characterized by the lack of both a well-defined wall and a pancreatic or peripancreatic necrosis on imaging. When an APFC persists beyond 4 weeks, a well-defined wall will develop, and the term pancreatic pseudocyst (PP) is applied. Similarly, in the setting of NP, a collection of not only fluid but also necrosis involving the pancreatic parenchyma or the peripancreatic tissues is termed an acute necrotic collection (ANC) when seen within the first 4 weeks of the disease. Like APFCs, ANCs lack a well-defined wall. When an ANC persists beyond 4 weeks and becomes encapsulated, the term walled-off necrosis (WON) is used. Concisely, an APFC contains no



**Fig. 2.** CT showing acute pancreatic necrosis with focal areas of decreased perfusion in the pancreatic parenchyma (*arrows*) and surrounding peripancreatic inflammation. The necrosis was estimated to involve less than 30% of the pancreas. G, gallbladder. (From Tenner S, Steinberg WM. Acute pancreatitis. In: Feldman M, Friedman LS, Brandt LJ, eds. Sleisenger and Fordtran's gastrointestinal and liver disease pathophysiology, diagnosis, management. 10th edition. Philadelphia: Elsevier; 2016; with permission.)

<b>Table 1</b>	
<b>Glossary of terminology</b>	
<b>Term</b>	<b>Definition</b>
Mild acute pancreatitis	Pancreatitis without evidence of organ failure or complications
Moderately severe acute pancreatitis	Pancreatitis with a local complication, such as APFC, PP, ANC, or WON (defined below) or with organ failure (defined below) lasting <48 h
SAP	Pancreatitis with a local complication, such as APFC, PP, ANC, or WON (defined below) or with organ failure (defined below) lasting more than 48 h
IEP	Pancreatitis that lacks pancreatic or peripancreatic necrosis on imaging
NP	Pancreatitis with parenchymal, peripancreatic, or combined necrosis, identified by contrast-enhanced imaging
APFC	Peripancreatic fluid collection that occurs within the first 4 wk of pancreatitis in the setting of IEP, without a well-defined wall
PP	APFC that has persisted more than 4 wk and now has evidence of well-defined wall
ANC	Collection of both fluid and necrotic solid material, in NP, within the first 4 wk, without a well-defined wall
WON	ANC that has persisted more than 4 wk and has developed a well-defined wall
Organ failure	A score of 2 or more for any organ system in the Marshall scoring system (see text)

See text for further explanation.

From Sabo A, Goussous N, Sardana N, et al. Necrotizing pancreatitis: a review of multidisciplinary management. JOP 2015;16(2):126; with permission.

necrotic material, whereas ANC contains fluid and necrosis; when these 2 entities persist beyond 4 weeks, they become PP and WON, respectively. Erstwhile terms, such as pancreatic abscess, pancreatic sequestration, necroma, and organized pancreatic necrosis, have fallen out of favor, and their use should be discouraged to avoid confusion.<sup>7</sup>

## PATIENT EVALUATION OVERVIEW

### *Clinical Features*

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#### **History**

Abdominal pain is present at the onset of most attacks of acute pancreatitis. Pain in pancreatitis usually involves the entire upper abdomen. However, it may be epigastric, in the right upper quadrant, or infrequently, confined to the left side. Onset of pain is rapid but not as abrupt as that of a perforated viscus. Usually, it is at maximal intensity in 10 to 20 minutes. Occasionally, pain gradually increases and takes several hours to reach maximum intensity. Pain is steady and moderate to very severe. There is little pain relief with changing position. Frequently, the pain is unbearable, steady, and boring. Bandlike radiation of the pain to the back occurs in half of the patients. Pain is absent in 5% to 10% of attacks, and a painless presentation may be a feature of serious fatal disease.

#### **Physical examination**

Physical findings vary with the severity of an attack. Patients with mild pancreatitis may not seem acutely ill. Abdominal tenderness may be mild, and abdominal guarding may be absent. In severe pancreatitis, patients look severely ill and often have abdominal distention, especially epigastric, which is due to gastric, small bowel, or colonic ileus. Almost all patients are tender in the upper abdomen, which may be elicited by gently shaking the abdomen or by gentle percussion. Guarding is more marked in the upper abdomen. Tenderness and guarding can be less than expected, considering the intensity of discomfort. Abdominal rigidity, as occurs in diffuse peritonitis, is unusual but can be present, and differentiation from a perforated viscus may be impossible in these instances. Bowel sounds are reduced and may be absent. Additional abdominal findings may include ecchymosis in 1 or both flanks (Grey Turner sign) or about the periumbilical area (Cullen sign) owing to extravasation of hemorrhagic pancreatic exudate to these areas. These signs occur in less than 1% of cases and are associated with a poor prognosis. The general examination, particularly in severe pancreatitis, may uncover markedly abnormal vital signs if there are third-space fluid losses and systemic toxicity. Commonly, the pulse rate is 100 to 150 beats per minute. Blood pressure can be initially higher than normal (perhaps because of pain) and then lower than normal with third-space losses and hypovolemia. Initially, the temperature may be normal, but within 1 to 3 days it may increase to 101° F to 103° F owing to the severe retroperitoneal inflammatory process and to the release of inflammatory mediators from the pancreas. Tachypnea with shallow respirations may be present if the subdiaphragmatic inflammatory exudate causes painful breathing. Dyspnea may accompany pleural effusions, atelectasis, acute respiratory distress syndrome (ARDS), or congestive heart failure. Chest examination may reveal limited diaphragmatic excursion if abdominal pain causes splinting of the diaphragm, or dullness to percussion and decreased breath sounds at the lung bases if there is a pleural effusion. There may be disorientation, hallucinations, agitation, or coma, which may be due to alcohol withdrawal, hypotension, fever, or toxic effects of pancreatic enzymes on the central nervous system. Conjunctival icterus, if present, may be due to

choledocholithiasis (gallstone pancreatitis) or bile duct obstruction from edema of the head of the pancreas, or from coexistent liver disease.

The differential diagnosis of acute pancreatitis is outlined in **Box 2**. The pain of perforated peptic ulcer is sudden, becomes diffuse, and precipitates a rigid abdomen; movement aggravates pain. In mesenteric ischemia or infarction, the clinical setting often is an older person with atrial fibrillation or arteriosclerotic disease who develops sudden pain out of proportion to physical findings, bloody diarrhea, nausea, and vomiting. In intestinal obstruction, pain is cyclical; abdominal distention is prominent; vomiting persists and may become feculent, and peristalsis is hyperactive and often audible.

#### Laboratory Diagnosis

##### Pancreatic enzymes

Amylase

Lipase

Other pancreatic enzyme levels

##### Standard blood tests

Diagnostic imaging

Abdominal plain film

Chest radiography

Abdominal ultrasound

Endoscopic ultrasound

Endoscopic retrograde cholangiopancreatography (ERCP)

CT

MRI

## PHARMACOLOGIC TREATMENT OPTIONS

### *Management*

Patients with acute pancreatitis require early aggressive intravenous (IV) hydration to maintain hemodynamic stability and adequately perfuse the kidneys and pancreas (**Fig. 3**). Patients also need adequate analgesia to eliminate or markedly reduce pain. The patient is usually NPO (nothing by mouth) until any nausea and vomiting have subsided. Nasogastric intubation is not used routinely because it is not

#### **Box 2**

##### **Differential diagnosis**

Biliary colic

Acute cholecystitis

Perforated hollow viscus (eg, perforated peptic ulcer)

Mesenteric ischemia or infarction

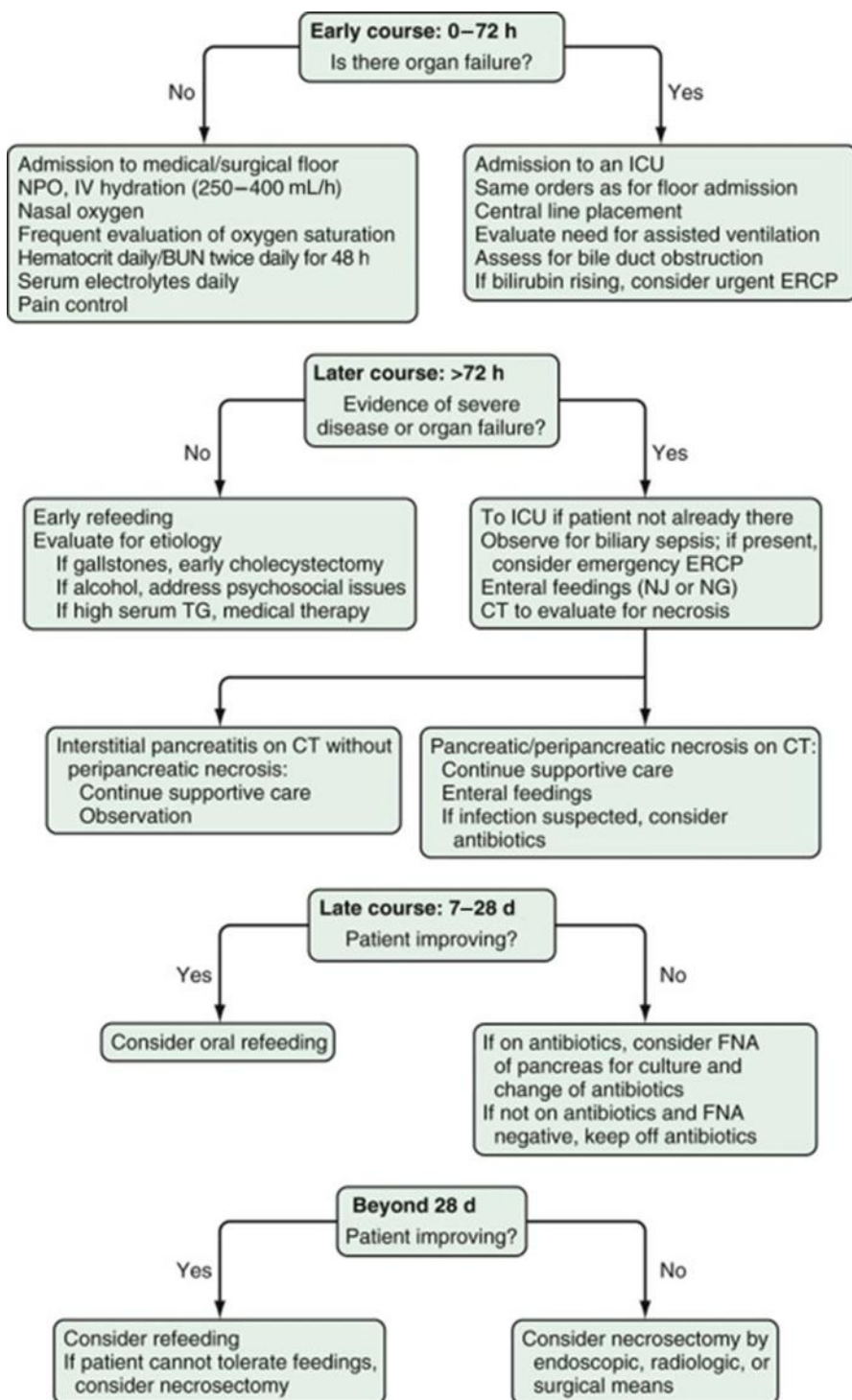
Intestinal obstruction

Myocardial infarction

Dissecting aortic aneurysm

Ectopic pregnancy

*From* Tenner S, Steinberg WM. Acute pancreatitis. In: Feldman M, Friedman LS, Brandt LJ, eds. *Sleisenger and Fordtran's gastrointestinal and liver disease pathophysiology, diagnosis, management*. 10th edition. Philadelphia: Elsevier; 2016; with permission.



**Fig. 3.** Algorithm for the management of acute pancreatitis at various stages in its course. BUN, blood urea nitrogen; ICU, intensive care unit; NG, nasogastric; NJ, nasojejunal; TG, thyroglobulin. (From Tenner S, Steinberg WM. Acute pancreatitis. In: Feldman M, Friedman LS, Brandt LJ, eds. Sleisenger and Fordtran's gastrointestinal and liver disease pathophysiology, diagnosis, management. 10th edition. Philadelphia: Elsevier; 2016; with permission.)

beneficial in mild pancreatitis. It is used only to treat gastric or intestinal ileus or intractable nausea and vomiting. Similarly, routine use of proton pump inhibitors or H<sub>2</sub> receptor blockers has not been shown to be beneficial. The patient should be carefully monitored for any signs of early organ failure, such as hypotension, pulmonary failure, or renal insufficiency, by closely following hemodynamic parameters, gas exchange, and urinary output. Tachypnea should not be assumed to be due to abdominal pain. Monitoring oxyhemoglobin saturation and, if needed, arterial blood gas measurement is advised, and oxygen supplementation is mandatory if there is hypoxemia. Any patient who exhibits signs of early organ dysfunction should be immediately transferred to an intensive care unit; deterioration can be rapid and fatal.

### ***Fluid Resuscitation***

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Early vigorous IV volume repletion for the purpose of intravascular resuscitation is of foremost importance. The goal is to provide enough intravascular volume to maintain pancreatic perfusion. Indices of hypovolemia and hemoconcentration have been associated with worse outcome; an admission hematocrit of more than 44% and a failure of the admission hematocrit to decrease at 24 hours have been shown to be predictors of NP, and an elevation and/or rising blood urea nitrogen is associated with increased mortality.<sup>6</sup> Maintaining hemodynamic stability and proper urine output (>30–60 mL per hour) is of paramount importance to prevent acute kidney injury.

### ***Respiratory Care***

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Gas exchange abnormalities may complicate the course of severe pancreatitis due to the development of noncardiogenic pulmonary edema secondary due to a systemic inflammatory response or due to decreased chest wall compliance from increased intra-abdominal pressures from severe pancreatic inflammation. In the setting of pulmonary insufficiency and severe lung injury, intubation with mechanical ventilatory support may be necessary. The development of ARDS is typically associated with an extrathoracic pancreatic inflammatory cause, and a low tidal volume ventilatory strategy may be required based on the degree of hypoxemia. Prolonged mechanical ventilatory support in the setting of persistent severe pancreatitis should trigger consideration for tracheostomy.

### ***Cardiovascular Care***

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Cardiovascular complications of SAP include congestive heart failure, myocardial infarction, cardiac dysrhythmia, and cardiogenic shock. The typical hemodynamic derangement is a distributive shock syndrome due to a systemic inflammatory response from pancreatic inflammation, and this physiology is characterized by an increase in cardiac index and a decrease in total peripheral resistance. If hypotension persists even with appropriate fluid resuscitation, vasoactive agent support is indicated. Although there are no evidence-based recommendations for an initial choice of vasoactive agent support, norepinephrine is a reasonable choice, with the addition of low-dose vasopressin as a second agent.

### ***Metabolic Complications***

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Hyperglycemia may present during the first several days of severe pancreatitis but usually disappears as the inflammatory process subsides. Blood sugars fluctuate, and insulin should be administered cautiously. Hypocalcemia is almost uniformly present because of a low serum albumin. Because this calcium low is nonionized, hypocalcemia is largely asymptomatic and requires no specific therapy. However, reduced



serum ionized calcium may occur and cause neuromuscular irritability. If hypomagnesemia coexists, magnesium replacement should restore serum calcium to normal.

### ***Infectious Disease and Antibiotics***

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In the absence of infection, antibiotics are not indicated in mild pancreatitis. However, antibiotics would be appropriate in pancreatic sepsis (eg, infected necrosis and, less often, abscess) and nonpancreatic sepsis (eg, line sepsis, urosepsis, or pneumonia). These septic conditions are major sources of morbidity and mortality in patients with SAP, and clinicians should be aware that these infectious complications account for many early and late deaths from the disease. Imipenem, fluoroquinolones (ciprofloxacin, ofloxacin, pefloxacin), and metronidazole are the drugs that achieve the highest inhibitory concentrations in pancreatic tissue, whereas aminoglycosides do not. Based on the latest 2 placebo-controlled studies, routine use of antibiotics is questionable in the absence of biliary sepsis or obvious pancreatic or peripancreatic infection.<sup>6</sup> Although previous practice guidelines recommend the use of prophylactic antibiotics in patients with severe NP, more recent reviews and guidelines state that prophylactic antibiotics should not be used for the purpose of preventing infection in patients with NP.<sup>6</sup> Infection of necrosis typically occurs after the 10th day of hospitalization. Infection of the pancreatic necrosis is thought to occur from translocation of bacteria from the colon. This finding may help explain why enteral feeding, decreasing the pathogenic intestinal flora, prevents infection of the necrosis. When infection is suspected, the diagnosis is readily established by CT-guided fine-needle aspiration (FNA). The procedure is safe and effective in establishing the diagnosis. The Gram stain alone has a sensitivity of almost 95% if carefully examined in a fresh specimen. The procedure is also safe, rarely introducing infection into a sterile field in the abdomen. If negative, an aspiration can be repeated every 4 to 7 days if infection continues to be suspected. In the past, the diagnosis of infected necrosis implied the urgent need for surgical debridement, but this is no longer always the case.

## **NONPHARMACOLOGIC TREATMENT OPTIONS**

### ***Endoscopy***

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The question of early removal of a possibly impacted gallstone in improving the outcome of gallstone pancreatitis remains a controversial issue. There is a consensus that severe acute gallstone pancreatitis with ascending cholangitis (jaundice and fever) is an indication for urgent ERCP. Until randomized studies are performed, it is not clear whether potential advantages of early pancreatic duct stenting outweigh the risks.

### ***Nutrition***

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Patients with SAP, especially with pancreatic necrosis, may need 4 to 6 weeks of artificial nutrition support. Formerly, total parenteral nutrition was the standard of care for feeding patients with SAP. Because enteral nutrition can stimulate pancreatic and intestinal secretions, the pancreatic rest concept has been a dogma in managing severe acute pancreatitis. However, bowel rest is associated with intestinal atrophy and bacterial overgrowth and is responsible for elevated endotoxin and cytokines levels, bacterial translocation, and systemic inflammatory response syndrome induction and is associated with a higher risk of infected pancreatic necrosis. Therefore, because of its beneficial effects on tissue of the intestinal mucosa and the splanchnic blood flow, the concept that enteral nutrition “worsens” pancreatitis has diminished greatly over recent years. In a recent meta-analysis including 8 randomized controlled

studies and 381 patients, enteral nutrition compared with parenteral nutrition decreased infectious complications and mortality.<sup>8</sup> The use of early enteral nutrition (within 48 hours of admission) has proven to be beneficial in patients with acute pancreatitis because it improves clinical outcomes by reducing the number of infections, particularly pancreatic infections.<sup>9</sup> On the basis of the assumption that gastric food administration increases the risk of abdominal pain exacerbation, nasojejunal feeding has long been favored. However, exclusive gastric feeding succeeds with the delivery of nutritional targets in 90% of patients.<sup>10</sup>

## RADIOLOGIC AND SURGICAL TREATMENT OPTIONS

Cholecystectomy is routinely performed with gallstone pancreatitis, and a consensus conference suggested that in mild or severe gallstone pancreatitis, cholecystectomy should be performed as soon as the patient has recovered and the acute inflammatory process has subsided. A second potential role for surgery in pancreatitis is to debride pancreatic necrosis (necrosectomy) or drain a pancreatic abscess.<sup>6</sup>

Some investigators have reported that it is important to differentiate sterile necrosis from infected necrosis by FNA of the pancreas. Sterile necrosis can be managed non-operatively because the mortality of this condition without surgery is less than 5%. On the other hand, infected necrosis (as documented by FNA of the pancreas with Gram stain and cultures) has been historically regarded as an indication for surgical debridement because of the previous thinking that infected necrosis treated medically has a nearly uniform fatal outcome. Surgical debridement of sterile pancreatic necrosis has also been shown not to be helpful in the vast majority of cases. Early surgical debridement is exceedingly difficult and avoided within the first 4 to 8 days because of the cementlike nature of the necrosis.

Currently, the management of NP has undergone a paradigm shift toward minimally invasive techniques for necrosectomy, obviating open necrosectomy in most cases.<sup>11</sup> There is increasing evidence that minimally invasive approaches, including a step-up approach that incorporates percutaneous catheter or endoscopic transluminal drainage followed by video-assisted retroperitoneal or endoscopic debridement,<sup>12</sup> are associated with improved outcomes over traditional open necrosectomy for patients with infected NP. A recent international multidisciplinary consensus conference emphasized the superiority of minimally invasive approaches over standard surgical approaches.<sup>3</sup>

However, in a stable patient with infected necrosis, maximal supportive care and the use of pancreatic-penetrating antibiotics such as fluoroquinolones, metronidazole, and imipenem or meropenem should be administered. Antibiotics have been shown to successfully treat infection of necrosis in many patients so it is possible that no other intervention will be needed. Even if intervention is needed owing to persistent symptoms, the antibiotics will allow time for the formation of a fibrous wall, creating WON. This fibrous wall assists in a successful minimally invasive approach to draining the pancreatic necrosis. Although early debridement of pancreatic necrosis within the first 4 to 5 weeks of an attack will require surgery, WON can be treated laparoscopically, percutaneously, or endoscopically. The timing and method of debridement require a clear discussion between the surgeon, gastroenterologist, and interventional radiologist, but should be left at the discretion of the pancreatic surgeon.

## EVALUATION OF OUTCOME AND LONG-TERM RECOMMENDATIONS

For sterile NP, the mortality of conservative treatment remained between 0% and 15.3%, which is the same as reported before 2000. Despite some studies' reports

of single-digit mortality using surgical necrosectomy, high mortality (20.0%–63.9%) is reported in most series. Except in a few centers, surgical outcome has not changed much, and the surgical risk is high. A nationwide study in the United States of 1783 patients from 1998 to 2010 indicated that the incidence of pancreatic debridement significantly decreased from 0.44% to 0.25% and that in-hospital mortality (overall 22.0%) significantly decreased from 29.0% to 15%. Minimally invasive necrosectomy, mainly transluminal endoscopic necrosectomy with drainage, has shown remarkable results combined with percutaneous drainage (PCD) or using a metallic stent. The success rate of PCD varies. Some series report that it remains unchanged at 35% to 49%, but most have reached a higher success rate of 76% to 93%. The transluminal endoscopic drainage rates are about 80%, and even 100% when using single transluminal gateway transcystic multiple drainage methods. Single-digit mortality was reported in most series, and zero mortality is a reality.<sup>13</sup>

## SUMMARY

Acute pancreatitis varies widely in its clinical presentation, from clinically negligible to precipitously fatal despite any intervention. Progression to multiorgan failure can occur rapidly and portends a life-threatening course. NP is a manifestation of SAP associated with significant morbidity and mortality. The extent of necrosis correlates well with the incidence of infected necrosis, organ failure, need for debridement, and morbidity and mortality. Having established the diagnosis of pancreatic necrosis, goals of appropriately aggressive resuscitation should be established and adhered to in a multidisciplinary approach involving medical and surgical intensive care. Intervention for infected pancreatic necrosis should be based on a minimally invasive approach. In all cases of NP, a multidisciplinary approach is needed, using endoscopic techniques and/or PCD. Open surgery should be reserved for failure of less invasive techniques.

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