Acute pancreatitis is a common disorder in the USA. Its diagnosis, prognosis and management, both in the short and long term, have long presented significant challenges to clinicians, surgeons, and diagnostic and interventional radiologists. This article reviews historical and current concepts in the diagnosis and management of acute pancreatitis and its complications, including radiological diagnosis and percutaneous intervention, as well as endoscopic and surgical management.

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Keywords: Acute pancreatitis, Interventional gastroenterology, Interventional radiology, Necrosectomy, Peripancreatic collection

Introduction

Acute pancreatitis is a common disorder in the USA, with more than 200,000 hospital admissions each year for management of the disease. Acute pancreatitis in its many forms often presents complex diagnostic and management challenges to physicians, surgeons, and radiologists caring for patients with the disease. In this article we discuss current practices in the diagnosis and management of acute pancreatitis.

Diagnosis and severity assessment of acute pancreatitis

Clinical diagnosis of acute pancreatitis is based on patient symptoms, physical examination, laboratory analysis, and radiological data. According to practice guidelines published in 2006, a diagnosis of acute pancreatitis requires two out of three main features: (1) abdominal pain typical for acute pancreatitis, (2) serum amylase and/or lipase greater than or equal to three times the upper normal limit; and (3) evidence of acute pancreatitis on computed tomography (CT) scans.

Almost all patients with acute pancreatitis have acute upper abdominal pain at onset. The pain is usually severe and constant. The pain may be confined to the mid-epigastrium or may be diffuse throughout the abdomen. Approximately half of patients report pain that radiates to the back that may be relieved by sitting or leaning forward. Patients frequently experience nausea and vomiting as well. However, the differential diagnosis for patients presenting with these symptoms is broad and includes diagnoses ranging from biliary colic, gastric or duodenal ulcer perforation and bowel obstruction, to mesenteric ischemia, aortic aneurysm or dissection, and even inferior wall myocardial infarction.

Physical signs and symptoms often depend on the severity of the attack. Systemic features include fever and tachycardia, and in severe cases patients may be in shock. In mild disease, the epigastrium may be minimally tender on physical examination, whereas patients with severe pancreatitis may have abdominal distention, tenderness, and guarding. Jaundice can occur due to obstruction of the common bile duct secondary to choledocholithiasis or due to extrinsic compression of the common bile duct due to edema within the pancreas head.

Laboratory analysis for work-up of patients with signs and symptoms of acute pancreatitis includes serum amylase and lipase levels, as well as a complete blood count, electrolytes, blood urea nitrogen (BUN), creatinine, liver function tests, and inflammatory markers, such as C reactive protein (CRP). In a recent retrospective analysis, the sensitivity and specificity for lipase levels in the diagnosis of acute pancreatitis were 96.6% and 99.4%, respectively. The sensitivity and specificity of amylase levels in diagnosing acute pancreatitis were 78.6% and 99.1%, respectively. An elevated serum amylase level is less specific as it can also occur in a number of other conditions aside from acute pancreatitis, including diseases of the salivary glands, cholecystitis, bowel obstruction or ischemia, and peptic ulcer disease. In addition, the longer half-life of lipase in comparison to amylase makes it a useful diagnostic measure in

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patients with delayed presentation in whom amylase levels may have already returned to normal. The level of pancreatic enzyme elevation does not correlate with the severity of the disease, and serial measurements should not be used as a tool to assess the prognosis or progress of acute pancreatitis. However, it has been noted that CRP levels >150 mg/dL at 48 hours help to differentiate between severe and mild disease.²

Initial assessment of severity is one of the most important issues in the management of acute pancreatitis. Approximately 15–20% of patients with acute pancreatitis will develop severe disease resulting in a prolonged clinical course, often in the setting of pancreatic parenchymal necrosis. An international symposium held in Atlanta, Georgia, in 1992, established a clinically based classification system for acute pancreatitis (Table 1).³ The revised Atlanta classification of acute pancreatitis established in 2008 identifies two phases of the disease: early and late.⁴ Severity is classified as mild, moderate, or severe (Table 2). Mild acute pancreatitis, the most common form, has no organ failure or 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 lasting longer than 48 hours.⁴

A variety of predictive systems have been developed to assist clinicians in predicting prognosis. These include Ranson’s criteria (Table 3), APACHE II (Acute Physiology and chronic Health Evaluation), and the BISAP (Bedside Index for Severity in Acute Pancreatitis) score.⁵-⁷ Of these, the BISAP score represents a simple way to identify patients at risk of increased mortality and the development of intermediate markers of severity within 24 hours of presentation. The BISAP score provides a single point for each of five parameters: BUN >25 mg/dL, impaired mental status, systemic inflammatory response syndrome (SIRS), age >60 years, and/or the presence of a pleural effusion, for a possible total of 6 points. A BISAP score greater than three is associated with a seven- to 12-fold increase in the risk of developing organ failure (Table 4).⁷ Hemocencentration, indicated by an admission hematocrit of ≥47%, and subsequent failure of the hematocrit to decrease by 24 hours are risk factors for the development of pancreatic necrosis.⁸ Older age (≥55 years) and a body mass index (BMI) >30 are also known risk factors for more severe forms of pancreatitis. In particular, obesity is associated with increased risk of developing both systemic and local complications.⁹ However, according to a recent large population-based study, abdominal adiposity, rather than total body fat, places patients at particular risk.¹⁰

Role of imaging in diagnosis

The Atlanta classification first reported in 1992 and its revision in 2008 provide a radiographic classification system for standardized diagnosis and management of acute pancreatitis.³ The revised imaging classification system reflects advances in knowledge of the disease process and in radiologic techniques since the original classification scheme.¹¹ The ultimate aim of the Atlanta classification is to provide a consistent radiographic lexicon for more precise communication of the severity and complications of acute pancreatitis to clinicians responsible for clinical management of patients.

Contrast-enhanced multi-detector CT (CECT) is the primary imaging modality used for further evaluation when acute pancreatitis is suspected or diagnosed clinically. Its speed and reproducibility, as well as its ability to accurately demonstrate morphologic changes in acute pancreatitis, make it an ideal first step in imaging of patients with acute pancreatitis. The main drawback of CT is its use of ionizing radiation, especially for younger patients who may require several repeat scans over the course of their illness. What remains somewhat unclear is the best time frame in which to perform CT after the patient's initial presentation. It is generally accepted that early in the course of the disease, the clinical severity and morphologic characteristics seen on CT may not directly correlate with each other. Imaging too early, before 48 hours, may significantly underestimate disease severity on the basis of imaging characteristics.¹²,¹³ In general, CT is not indicated in patients who are clinically classified as having mild pancreatitis (no clinical signs of severe pancreatitis) and show rapid improvement with appropriate medical management. CT should be used in patients who are classified as having severe pancreatitis or are at risk of developing severe pancreatitis, ideally after 72 hours, to best assess the full extent of the disease.¹⁴ In addition, while generally not used for evaluation of the pancreas itself, ultrasound (US) is often performed early in the course of the disease, regardless of the severity, to help establish an etiology for the pancreatitis (i.e. the presence of cholelithiasis or choledocholithiasis) and direct the need for further endoscopic or surgical management [endoscopic retrograde cholangiopancreatography (ERCP) or cholecystectomy].¹¹

The revised Atlanta classification subdivides acute pancreatitis into two types: interstitial edematous pancreatitis (IEP) and

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**Table 2 Revised Atlanta Classification**

<table>
<thead>
<tr>
<th>Disease severity</th>
<th>Symptoms</th>
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<tbody>
<tr>
<td>Mild acute pancreatitis</td>
<td>No organ failure</td>
</tr>
<tr>
<td>Moderately severe acute pancreatitis</td>
<td>Organs failure that resolves within 48 h (transient organ failure)</td>
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<tr>
<td>Severe acute pancreatitis</td>
<td>Local or systemic complications without persistent organ failure Persistent organ failure (&gt;48 h) Single organ failure Multiple organ failure</td>
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**Table 3 Ranson's Criteria**

<table>
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<tr>
<td>On admission</td>
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<tr>
<td>Age (y)</td>
<td>&gt;55</td>
<td>&gt;70</td>
</tr>
<tr>
<td>White blood cells (×10⁹)</td>
<td>&gt;16,000</td>
<td>&gt;18,000</td>
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<tr>
<td>Blood glucose (mg/dL)</td>
<td>&gt;200</td>
<td>&gt;220</td>
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<tr>
<td>Serum lactate dehydrogenase (IU/L)</td>
<td>&gt;350</td>
<td>&gt;400</td>
</tr>
<tr>
<td>Serum aspartate aminotransferase (IU/L)</td>
<td>&gt;250</td>
<td>&gt;250</td>
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<tr>
<td>During initial 48 h</td>
<td></td>
<td></td>
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<tr>
<td>Hematocrit decrease (%)</td>
<td>&gt;10</td>
<td>&gt;10</td>
</tr>
<tr>
<td>Blood urea nitrogen increase (mg/dL)</td>
<td>&gt;5</td>
<td>&gt;2</td>
</tr>
<tr>
<td>Serum calcium (mg/dL)</td>
<td>&lt;8</td>
<td>&lt;8</td>
</tr>
<tr>
<td>Arterial po₂ (mmHg)</td>
<td>&lt;60</td>
<td>NA</td>
</tr>
<tr>
<td>Serum base deficit (mEq/L)</td>
<td>&gt;4</td>
<td>&gt;5</td>
</tr>
<tr>
<td>Fluid sequestration (L)</td>
<td>&gt;6</td>
<td>&gt;4</td>
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</tbody>
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**Table 1 Atlanta Criteria for Severity**

<table>
<thead>
<tr>
<th>Feature</th>
<th>Symptoms</th>
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<tbody>
<tr>
<td>Organ failure</td>
<td>Shock (systolic blood pressure &lt; 90 mmHg)</td>
</tr>
<tr>
<td></td>
<td>Pulmonary insufficiency (PaO₂ &lt; 60 mmHg)</td>
</tr>
<tr>
<td></td>
<td>Renal failure (serum creatinine &gt; 2 mg/dL after rehydration)</td>
</tr>
<tr>
<td></td>
<td>Gastrointestinal bleeding (&gt;500 mL/24 h)</td>
</tr>
<tr>
<td>Local complications</td>
<td>Pancreatic necrosis (&gt;30% of the pancreas or &gt;3 cm)</td>
</tr>
<tr>
<td></td>
<td>Pancreatic abscess (circumscribed collection of pus containing little or no necrotic tissue)</td>
</tr>
<tr>
<td></td>
<td>Pancreatic pseudocyst (collection of pancreatic juice enclosed by a wall of fibrous tissue or granulation tissue)</td>
</tr>
</tbody>
</table>
necrotizing pancreatitis. The main distinguishing factor among the two is the presence or absence of necrosis. The detection and classification of necrosis in pancreatitis are of particular importance because mortality of up to 23% is seen in patients with necrotizing pancreatitis and life-threatening complications almost all occur in patients with necrosis. In addition, patients with necrotizing pancreatitis are much more likely to suffer from secondary infection.

In patients with acute IEP, CT typically reveals an enlarged pancreas. The parenchymal enhancement is generally normal (homogeneous throughout) or with minimal heterogeneity, reflecting edema within the gland. Early on, the peripancreatic tissues may appear normal or show inflammatory changes such as ground-glass opacity and stranding within the peripancreatic fat (Fig. 1). There may also be varying amounts of peripancreatic fluid. At <4 weeks, these fluid collections are termed acute peripancreatic fluid collections (APFCs) and purely comprise fluid contents (no solid or non-fluid components). They are often the result of leakage of pancreatic enzymes from a ruptured side-branch duct, show no discernible wall, and typically respect the anatomic and fascial boundaries of the retroperitoneum (Fig. 2). The vast majority of these collections are spontaneously resorbed within the first few weeks after onset of pancreatitis.

After 4 weeks, approximately 10–20% of patients with IEP and APFCs can develop pseudocysts as a complication of acute pancreatitis. Pseudocysts are identified on CT by the presence of an identifiable, smooth, and uniform wall. Pseudocysts are also typically round or oval and, depending on their size, may exert a mass effect on adjacent structures rather than insinuate along tissue planes, as observed for APFCs (Fig. 3). Like APFCs, pseudocysts have purely fluid contents and may maintain a connection to the pancreatic ducts, resulting in amylase- and lipase-rich fluid. Once a pseudocyst seals off, it often spontaneously vanishes, as revealed by follow-up imaging.

Secondary infection of pseudocysts is rare but can occur and is often manifest as gas bubbles within the pseudocyst on CT, although this is not always the case. If the patient displays signs and symptoms of infection, fine needle aspiration (FNA) of the cyst for Gram staining and culture may be required for a diagnosis. Even less common than pseudocyst infection is the occurrence of secondary infection in patients with IEP and APFCs. In a retrospective series to evaluate complications and fluid collections in patients with acute mild pancreatitis, Lenhart and Balthazar found no complications at all in patients without peripancreatic fluid collections (IEP without APFC).

If evidence of necrosis is detected on CECT, then the patient is categorized as having necrotizing pancreatitis. This category is further subdivided according to the Atlanta classification based on the location of the necrosis, which in the first 4 weeks is termed post-necrotic pancreatic fluid collection (PNPFC), also sometimes referred to as acute necrotic collection (ANC). Acute necrotizing pancreatitis can involve (1) parenchymal necrosis alone, (2) peri-pancreatic necrosis alone, or (3) mixed parenchymal and peri-pancreatic necrosis.

Pancreatic parenchymal necrosis alone is relatively uncommon in comparison to the other two forms of necrotizing pancreatitis, accounting for only 5% of cases (Fig. 4). On CECT, parenchymal necrosis appears as an area of glandular non-enhancement. The degree of homogeneity in the area of necrosis varies on a continuum, as liquefaction of the necrotic material progresses over time. The extent of parenchymal necrosis is approximated as <30%, 30–50% or >50% of the gland, although estimates of <30% are less reliable and follow-up imaging in 5–7 days is sometimes necessary to confirm true necrosis versus glandular edema, as seen in IEP.

Isolated peripancreatic necrosis in the absence of parenchymal necrosis is seen in up to 20% of cases. This can be more difficult to diagnose on CT alone, but inhomogeneous fluid collections in the peripancreatic spaces and in the lesser sac, and thickening and inflammatory changes at the root of the small bowel mesentery provide clues for diagnosis. These patients typically have a better prognosis than patients with other types of necrotizing pancreatitis owing to preservation of the gland itself.

By far the most common form is mixed parenchymal and peri-pancreatic necrosis, which is seen in 75–80% of cases of acute necrotizing pancreatitis (Fig. 5). PNPFCs (seen <4 weeks from onset) have a variable appearance as the composition of the collection changes with progressive liquefaction of necrotic material. PNPFCs can sometimes be difficult to distinguish from APFCs, especially within the first week after diagnosis, and MRI (or occasionally US) can be useful to visualize the debris and complexity in

<table>
<thead>
<tr>
<th>Table 4 BISAP Scoring System</th>
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<tbody>
<tr>
<td>BUN &gt; 25 mg/dL.</td>
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<tr>
<td>Temperature &lt; 36°C or &gt;38°C</td>
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<tr>
<td>Pulse &gt; 90 beats/min</td>
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<tr>
<td>Age &gt; 60 years</td>
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**Fig. 1.** Axial CT image of interstitial edematous pancreatitis in a 56-year-old man after endoscopic retrograde cholangiopancreatography. Amylase 4650 U/L, lipase > 396 U/L. The image shows homogeneous enhancement of the pancreatic parenchyma (white arrow) without necrosis and mild peripancreatic stranding.

<table>
<thead>
<tr>
<th>Table 5 Revised Atlanta Classification for Imaging</th>
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<tbody>
<tr>
<td><strong>Pancreatitis</strong></td>
</tr>
<tr>
<td>&lt;4 wk</td>
</tr>
<tr>
<td>IEP</td>
</tr>
<tr>
<td>Pseudocyst</td>
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<tr>
<td>Parenchymal necrosis</td>
</tr>
<tr>
<td>Peripancreatic necrosis</td>
</tr>
<tr>
<td>Mixed necrosis</td>
</tr>
<tr>
<td>&gt;4 wk</td>
</tr>
<tr>
<td>IEP</td>
</tr>
<tr>
<td>Pseudocyst</td>
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</table>

APFC, acute peripancreatic fluid collection; IEP, interstitial edematous pancreatitis; PNPFC, post-necrotic pancreatic fluid collection; WOPN, walled-off pancreatic necrosis.
necrotic collections (Fig. 6). Because the necrosis results in breakdown of the pancreatic architecture, PNPFCs may often maintain communication with the main or side-branch pancreatic ducts. MRI can also be useful for delineating this connection.

After approximately 4 weeks, if the necrotic collections are not resorbed, the necrosis can mature and develop a thick wall, termed walled-off pancreatic necrosis (WOPN) (Fig. 7). Distinguishing WOPN from pseudocysts is important, as management strategies differ for these two types of collections. Both PNPFCs and WOPNs can become infected. This is suggested by the presence of gas within the necrotic collections or extraluminal gas bubbles, although perforation or fistular communication with the gastrointestinal tract should be excluded as a source of gas within the collection. As with pseudocysts, if no gas is seen but there is other

Fig. 2. Axial CT images in a 48-year-old female with interstitial edematous pancreatitis and acute peripancreatic fluid collections. The gland is edematous but enhanced throughout. There is no evidence of necrosis. Homogeneous fluid collections in the mesentery (white arrow) have no discernible wall and no solid or non-fluid components. Amylase 244 U/L, lipase > 396 U/L.

Fig. 3. (A) Axial contrast-enhanced CT (CECT) for a 51-year-old man. (B) T2-weighted MRI for a 48-year-old female. Both show pseudocysts. CECT demonstrates a round, well-circumscribed fluid collection adjacent to the pancreatic tail, with a thin but discernible enhanced wall (white arrow). T2 MRI shows a round fluid collection with a thick wall (black arrow). There is no evidence of solid or non-fluid contents.

Fig. 5. Axial CT image for a 62-year-old male with acute necrotizing pancreatitis with mixed parenchymal and peripancreatic necrosis. Multifocal areas of nonenhancement within the gland parenchyma (white arrow) and peripancreatic fluid collection are present, along with enhancement and thickening of Gerota’s fascia (black arrow).
clinical evidence of infection, FNA for Gram staining and culture should be performed (Fig. 8).11,12

Other CT grading systems have been developed over the last two decades to further refine patient prognosis and quantify morbidity and mortality rates. Most notable is the CT severity index proposed by Balthazar et al in 1990.16 This allows radiologists to grade the severity of pancreatitis on a 10-point scale, with points allotted for the amount of peripancreatic inflammation and fluid collections, as well as the amount of pancreatic necrosis (<30%, 30–50%, or >50%). This was followed in 2004 by a modified CT severity index designed by Mortele et al that incorporates extrapancreatic complications in the scoring system and simplifies analysis of the degree of pancreatic necrosis (<30% or >30%) (Table 6).17

MRI of the pancreas and MR cholangiopancreatography (MRCP) have been increasingly used to evaluate patients with acute pancreatitis. MRI has a distinct advantage over CT: it does not use ionizing radiation, which is important, particularly for younger patients requiring multiple follow-up exams or with recurrent disease. In addition, MRI/MRCP is superior to CT for evaluation of choledocholithiasis and the pancreatic ductal system. It is also important as an adjunct to CECT for evaluating the contents of pancreatic and peripancreatic collections to help distinguish necrotic collections (PNPCCs and WOPNs) from non-necrotic collections (APPCCs and pseudocysts).11,12 In patients older than 40 years for whom no cause of pancreatitis has been identified, MRI can be especially useful in searching for underlying occult neoplasms as a cause of pancreatitis. Drawbacks of MRI include its relatively high cost and its impracticality for critically ill patients who are unable to tolerate long imaging times or effectively hold their breath for many sequences.18

Both CECT and MRI are useful in the detection of other extrapancreatic complications of acute pancreatitis, including venous thrombosis, gastric varices, pseudoaneurysm formation and rupture, hemorrhage, ascites, fistulization and rupture of the GI tract, and pleural effusion.

Medical management of acute pancreatitis

Initial assessment of the severity of acute pancreatitis is the cornerstone in determining further medical management. Aggressive intravenous fluid replacement is very important to treat fluid losses caused by third space shifts, vomiting, diaphoresis, and increased vascular permeability caused by inflammatory mediators. Clinically, the adequacy of fluid resuscitation should be monitored in terms of vital signs, urinary output, and hematocrit at 12 and 24 hours after admission (particularly for patients with hemoconcentration on admission).8 A second important consequence of hypovolemia is intestinal ischemia. Ischemia increases intestinal permeability to bacteria and endotoxins, an important cause of secondary pancreatic infection.1

Adequate pain management with opiate analgesics is important for treatment of severe pain associated with acute pancreatitis. Patient-controlled analgesia is often helpful for good pain control. However, monitoring of oxygenation while on high-dose opiate medications is necessary. According to current American College of Gastroenterology guidelines, initial routine oxygen delivery via a
nasal cannula is recommended for all patients with acute pancreatitis. Nutritional support should be considered when patients are unlikely to be able to eat for 7 days. To compare the safety and clinical outcomes of enteral and parenteral nutrition in patients with acute pancreatitis, a meta-analysis of six studies conducted in 2004 revealed that enteral nutrition was associated with a significantly lower incidence of infections (relative risk 0.45; 95% confidence interval 0.26–0.78, \( P = 0.004 \)), fewer surgical interventions to control pancreatitis (0.48, 0.22–1.0, \( P = 0.05 \)), and shorter hospital stays (mean reduction 2.9 days, range 1.6–4.3 days, \( P < 0.001 \)). However, there was no significant difference in mortality (relative risk 0.66, 0.32–1.37, \( P = 0.3 \)).

The use of prophylactic antibiotics to prevent pancreatic infection is not recommended for patients with necrotizing pancreatitis. This guideline is based on two randomized studies, the latter of which was a multicenter, prospective, double-blind, placebo-controlled study conducted in 32 centers in North America and Europe. Pancreatic or peripancreatic infection developed in 18% (9 of 50) of patients in the group treated with meropenem, compared with 12% (6 of 50) of patients taking placebo (\( P = 0.401 \)). The overall mortality rate was 20% (10 of 50) in the meropenem group and 18% (9 of 50) in the placebo group (\( P = 0.799 \)). Surgical intervention was required in 26% (13 of 50) of the patients taking meropenem and 20% (10 of 50) of the patients taking placebo (\( P = 0.476 \)). This study demonstrated no significant differences between the treatment and placebo groups for pancreatic or peripancreatic infection, mortality, or requirement for surgical intervention, and therefore did not support early prophylactic antimicrobial use in patients with severe acute necrotizing pancreatitis.

### Radiologic intervention

The role of interventional radiology in the management of acute pancreatitis complications falls into two main categories.

#### Percutaneous drainage of peripancreatic fluid collections in IEP

Peripancreatic fluid collections develop in the early phase of acute pancreatitis within or around the pancreas in approximately 40% of cases. These can be found in the peritoneal and retroperitoneal spaces and less frequently in the mediastinum. Early in the course of the disease (<4 weeks), the APFCs do not have definite epithelial walls and boundaries are limited by the natural fascial barriers of the retroperitoneum and peritoneum. In most cases, spontaneous resolution occurs without intervention. If APFCs do not resolve spontaneously and persist beyond 4 weeks to develop an identifiable wall and become pseudocysts, approximately 25% can cause a spectrum of symptoms mostly related to mass effects. Pseudocysts can also become infected, necessitating treatment.

Intervention for both APFCs and pseudocysts is indicated for infected collections or when they cause severe symptoms. However, neither clinical nor radiological signs are highly specific for the diagnosis of infected collection contents, and therefore FNA with subsequent microbiological analysis remains the gold standard to confirm the presence of underlying infection. Pancreatic necrosis and fluid collections can be aspirated using either CT or US guidance and samples are collected under sterile conditions to evaluate underlying infection (Fig. 9). The size of the aspiration needle required depends on the viscosity and thickness of the aspirated material but usually varies between 22 and 18 gauge. False negative rates for aspiration to diagnose or confirm infection are very low (<10%) and repeat FNA should be performed if there is persistent clinical suspicion of infection in the face of a negative result.

Percutaneous drainage of peripancreatic collections is a well-established and common procedure and simple drainage will often suffice for these collections. The drainage catheter is predominantly placed under CT guidance and to a lesser extent under US guidance. Strict sterility is maintained and local anesthesia is applied. For both diagnostic aspiration and therapeutic drainage, a retroperitoneal approach is preferred over an anterior transperitoneal approach, if possible. Moderate conscious sedation or general anesthesia can be used, as the clinical situation demands.
Two different image-guided techniques are used for drainage catheter insertion. The Seldinger technique, named after the Swedish radiologist Sven-Ivar Seldinger, consists of advancing an 18-gauge needle into the target collection under CT or US guidance. A 0.035-inch guide wire is advanced through the needle, and the needle is exchanged for the catheter over the wire and secured in the drained collection (Fig. 10). Alternatively, the tandem-trocar technique involves similar initial access to the collection via a smaller needle (20–22 gauge) (Fig. 11). Then a trocar-loaded catheter is advanced along a parallel trajectory to a measured depth and a pigtail tip is formed in the cavity. The collection can also be accessed using combined US and fluoroscopic guidance. After access is obtained and confirmed by US, contrast injection under fluoroscopy can be performed to evaluate the extent of the collection and elucidate any fistulous connection to the main pancreatic duct or adjacent bowel. The needle can then be exchanged over a wire for the drainage catheter under fluoroscopic guidance.

After initial access to the collection using either technique, samples are routinely aspirated for analysis, regardless of the degree of suspicion of infection. Drainage catheters typically remain in place until the drained fluid is clear and <10–30 mL per day. Follow-up imaging to confirm drainage catheter position and document collection resolution is often necessary.

**Percutaneous drainage of PNPC and WOPN**

Acute necrotizing pancreatitis accounts for 10–15% of all cases of acute pancreatitis and can be complicated by infected necrosis and multisystem organ failure. On CECT, WOPN can be misdiagnosed as a pseudocyst and unsuccessfully managed by conventional percutaneous drainage. MRI plays a crucial role in the correct diagnosis of WOPN since it provides better appreciation of the amount of fluid and non-liquid necrosis in the collection, resulting in appropriate management.

Management of necrotizing pancreatitis has evolved over the past two decades from more aggressive and traditional surgical necrosectomy to more conservative management relying on minimally invasive percutaneous and endoscopic necrosectomy. This change was prompted by higher mortality in patients who underwent early surgical necrosectomy compared to patients with delayed surgery performed more than 28 days following the onset of symptoms. The percutaneous drainage and necrosectomy procedure is performed using the same techniques as for conventional intra-abdominal collection drainage, as discussed above. However, double-sump large-bore catheters of up to 30 French are used for better evacuation of the necrotic debris. Initially the catheters are irrigated using up to 1.5 L of sterile normal saline per drain daily, depending on the size of the collection. The clinical success of percutaneous necrosectomy varies from 20% to 64%. An alternative nonsurgical method for treatment of WOPN is endoscopic necrosectomy and drainage.

Complications related to percutaneous drainage such as injury of the surrounding organs and bleeding are rare (2%). More commonly, the drain can become clogged or dislodged, requiring replacement. A late complication of percutaneous drainage is the development of pancreatic fistulae to the skin or gastrointestinal tract, but most close spontaneously.

**Endoscopic and surgical intervention**

**Indication and timing of ERCP for gallstone pancreatitis**

Several studies investigated the effect of early ERCP in acute biliary pancreatitis. Neoptolemos et al found that ERCP within 72 hours decreased morbidity in patients with severe pancreatitis.

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**Fig. 10.** Seldinger technique in a 37-year-old male patient with necrotizing acute pancreatitis. (A,B) The tip of a 15-cm-long 18-gauge needle was advanced under CT guidance into the target cavity (white arrow). A 0.035 guide wire was advanced through the needle until its floppy tip was coiled within the cavity (black arrow). (C) The needle was removed and a 10 French multipurpose drainage catheter was advanced over the guide wire.

**Fig. 11.** Tandem trocar technique in a 60-year-old female patient with a retrogastric lesser sac pseudocyst. Initial access into the cyst was gained using a 15-cm-long 20-gauge needle. After the position of the needle tip was confirmed inside the pseudocyst, a 10 French multipurpose drainage catheter was advanced in tandem with the needle to the measured depth and a pigtail formed within the pseudocyst.
defined by Ranson’s criteria.33 There were, however, no benefits of early ERCP in patients with mild acute pancreatitis. Fan et al showed that ERCP within 24 hours in patients with acute severe pancreatitis decreased the incidence of biliary sepsis. Survival benefit of early ERCP was also seen in patients with acute pancreatitis if biliary sepsis was present.34 Folsch et al excluded jaundiced patients and focused on the role of early ERCP in patients with severe acute pancreatitis in the absence of cholangitis and found no benefit of early ERCP.35 According to the American Society for Gastrointestinal Endoscopy (ASGE) guidelines, there is no role for early ERCP in the evaluation and management of patients with mild acute pancreatitis in the absence of clear evidence of a retained stone. Conversely, in patients with acute pancreatitis and concomitant cholangitis, early ERCP (within 24–72 hours) is strongly recommended.36

**Indication and timing of cholecystectomy for gallstone pancreatitis**

For both open and laparoscopic cholecystectomy there has been debate about the best timing of cholecystectomy after an episode of gallstone pancreatitis. There is now a consensus that cholecystectomy should be delayed until the clinical symptoms of acute pancreatitis subside, but not so long that a second attack occurs, for the best clinical outcome. In milder cases of acute pancreatitis, it is not clear if surgery needs to be delayed until amylase and lipase levels have normalized, or even until all abdominal pain has gone. Many clinicians and clinical systems advocate that patients hospitalized with an attack of acute gallstone pancreatitis undergo a cholecystectomy once they have clinically recovered but prior to hospital discharge since the likelihood of a subsequent attack is approximately 30% over the next 3 months. It is clear that endoscopic sphincterotomy also substantially reduces the likelihood of subsequent attacks of gallstone pancreatitis, probably to the same degree as cholecystectomy does. However, sphincterotomy does not eliminate the risk of subsequent cholecystitis and has its own suite of procedural complications. Consequently, laparoscopic cholecystectomy is currently considered the default procedure to prevent subsequent attacks in mild or moderate acute gallstone pancreatitis, while endoscopic sphincterotomy is an alternative procedure typically chosen for reasons specific to an individual patient to achieve the same end.

**Endoscopic management of pancreatic fluid collection**

Endoscopic transmural drainage of pancreatic fluid collections is a form of natural orifice transluminal endoscopic surgery (NOTES), which is minimally invasive and has high success rates. It has been used for drainage of pseudocysts, pancreatic abscesses, and necrosis. Indications and criteria for endoscopic drainage include pseudocysts with symptoms such as pain and mechanical obstruction of the gastric outlet or biliary system. Drainage of enlarging pseudocysts that do not resolve after 6 weeks is also indicated to prevent subsequent complications such as hemorrhage, perforation, or secondary infection. Pancreatic abscesses and infected necrosis also require drainage for effective control of infection and sepsis.

The first endoscopic drainage of pancreatic necrosis was performed in 1995.37 Since then the technique has been modified from an irrigation approach to direct endoscopic debridement or necrosectomy in 2000.38 Endoscopic necrosectomy is typically performed at least 3–4 weeks after the onset of pancreatic necrosis. This allows for encapsulation and demarcation of peripancreatic collections, known as WOPN, thereby decreasing the risk of bleeding and perforation. Treatment is performed in a multi-stage procedure involving cystgastrostomy followed by repeated necrosectomy sessions. Cystgastrostomy is the first step, where a fistula tract is created between the collection of pancreatic necrosis and the gastric wall. Endoscopic US is used to locate the collection, which is then accessed by puncturing through the gastric wall, followed by balloon dilatation of the tract (Fig. 12). Two or more double pigtail stents are then placed into the tract for continual drainage. More recently, a new fully covered self-expandable metal stent has been used successfully in infected WOPN.39 Necrosectomy is then typically performed 2–4 weeks after cystgastrostomy. During necrosectomy, the endoscope is advanced through the tract into the necrotic cavity and repeated removal of necrotic tissue is performed (Fig. 13). Double pigtail stents are inserted into the tract at the end of the procedure. Multiple sessions are typically required for complete resolution of the necrotic collection. More recently, the creation of two or three transmural tracts between the necrotic cavity and the GI lumen using endoscopic US (EUS) guidance yielded encouraging initial results.40 Treatment was successful in 92% of the 12 patients who underwent this multiple transmural gateway technique compared to 52% in 48 patients treated with conventional one-tract drainage.

Outcomes of endoscopic necrosectomy have been investigated in multiple recent studies. In the multicenter GEPARD study, 93 patients underwent a mean of six endoscopic necrosectomy sessions, starting at a mean of 43 days after an attack of severe acute pancreatitis. Repeated sessions were performed at intervals of 1–4 days until all necrotic material had been removed. The success rate was 81%, with 84% of those successfully treated surviving without recurrence at a mean follow-up of 43 months. The mortality rate was 7.5% at 30 days. Major complications occurred in 26% patients, including bleeding, perforation of necrosis into the abdominal cavity, fistula formation, and air embolism (which is caused by prolonged procedures, and can be minimized using CO2 for insufflation).41 In another large multicenter retrospective series in the USA, endoscopic necrosectomy was successful in 91% of 104 patients. A median of three procedures with two debridements were performed, with the first debridement performed at a mean of 63 days after the initial onset of acute pancreatitis. The mean time to resolution from the initial procedure was 4.1 months. Complications occurred in 14%, with five perforations or pneumoperitoneum that were managed nonoperatively. A high BMI of >32 was identified as a risk factor for failure of endoscopic necrosectomy.42 In a systematic review of 10 studies with 260 patients with pancreatic necrosis, resolution of necrotic fluid collections by endoscopic intervention alone was achieved in 76% after an average of four endoscopic sessions. The overall mortality for endoscopic necrosectomy was 5% and the mean procedure-related morbidity rate was 27%. Bleeding was the most common complication, followed by exacerbation of sepsis and perforation.43 In another large retrospective study of 211 patients who underwent endoscopic transmural drainage of peripancreatic fluid (45% pseudocyst, 28% abscess, 27% necrosis), overall treatment success with complete resolution or decrease in size of the pancreatic fluid collection to <2 cm was 85%. The procedure was more successful for patients with a pseudocyst or abscess than for those with necrosis (93% vs. 63%). Complications were seen in 16% of the necrosis cases versus 5% of the patients with a pseudocyst or abscess, and included infection, perforation, bleeding, and stent migration.44

**Surgical management of complications of acute pancreatitis**

The indication for surgery in acute pancreatitis is best described as management of specific complications of acute pancreatitis. These complications almost always occur in patients with severe or necrotizing pancreatitis, typically more than 10 days from the onset of the attack and sometimes more than 3 months later. However, some complications can occur early in the course of severe acute
pancreatitis, such as abdominal compartment syndrome, requiring emergency decompressive laparotomy and an open abdomen in the intensive care unit. In addition, infarction of the transverse colon caused by inflammatory thrombosis of the mesocolic vessels requires emergency colon resection and colostomy.

Strictures of the bile ducts, duodenum, or other segments of the bowel caused by chronic scarring are managed with surgical bypass or resection. This typically occurs many months after the acute attack. Bleeding from a ruptured pseudoaneurysm or GI bleeding secondary to portal gastropathy or esophageal varices due to thrombosis of the splenic vein are complications that tend to occur later in the course of acute severe necrotizing pancreatitis and may require surgical management. However, interventional vascular techniques using embolization and sometimes stents are often better first management choices for these problems. This is especially true for unstable patients since the surgery to access the bleeding site or resect the spleen to manage sinistral portal hypertension can be prolonged and arduous.

It is well established that acute pseudocysts and fluid collections are best managed by expectant observation since they mostly resolve on their own. Larger cysts that persist for more than 6 weeks and are symptomatic benefit from internal drainage into the gut. Traditionally, this has been accomplished by an open surgical cystgastrostomy, cystduodenostomy, or Roux Y cystjejunostomy, depending on the location of the cyst. These operations are effective at producing symptom relief and preventing recurrence. They are now often accomplished laparoscopically when surgery is required. However, internal drainage of these cysts is far more commonly managed by endoscopic stenting of either the pancreatic duct or through the stomach wall into the cyst, or both, as discussed previously. These maneuvers are certainly quicker and less invasive than open surgery, although it is not clear whether they are as cost effective, and vascular structures in the wall of a cyst may inhibit the ability of the endoscopist to safely place a stent or pigtail drain into the cyst. Occasionally, a cyst is surgically resected as definitive management of symptoms, usually when there are no good drainage options or if there are concerns that the cyst may be neoplastic rather than a pseudocyst. Infected cysts that require treatment early in the course of pancreatitis are best drained externally by image-guided radiologic interventions rather than surgical approaches.

The problem with any collection drainage procedure in this setting is the amount of necrotic debris in the collection. When there is only a small amount of necrotic debris within the cyst and a large volume of fluid, drainage procedures are quite effective. When the fluid collection consists mostly of gelatinous, semisolid, necrotic debris, it often will not drain successfully by internal or external methods. Over the past 30 years, mechanical surgical debridement of this pancreatic necrosis has been the mainstay treatment and has dramatically improved what was previously the lethal natural history of this condition. The tactics and tools used for surgical debridement have steadily evolved, and outcomes measured as conventional morbidity and mortality have also steadily improved. The surgery for this condition was originally open, with sequential trips to the operating theater every second or third day for debridement with gauze packing of the cavity to aid both debridement and hemostasis. This process required prolonged stays in the intensive care unit (ICU) and was associated with multiple complications related to the sequelae of severe pancreatitis and repeat surgery (e.g., enterocutaneous fistulae, iatrogenic bleeding and vascular injury, hernias) and ICU support (e.g., ventilator-associated pneumonia, line infections). Early controversies revolved around whether outcomes could be improved by debridement of the necrosis earlier in the course of severe acute necrotizing pancreatitis. Several small trials demonstrated quite clearly that earlier compared to delayed debridement of pancreatic necrosis was associated with much poorer survival outcomes. The reasons for the adverse outcomes associated with early
debridement are related to the much greater operative difficulty in separating necrotic tissue from surrounding inflamed and highly vascularized tissue. With delay, the necrotic tissue becomes more demarcated, has less vascular adhesion to the surrounding viable tissue, and can be debrided much more easily with blunt dissection. In patients for whom debridement could be delayed, it became clear that a single open operative debridement with the use of multiple drains could resolve the condition most of the time, although repeat operative debridements or subsequent interventional radiologic drains were still required in approximately 20% of these patients.

**Endoscopic versus surgical necrosectomy**

In the 2010 randomized PANter trial (Pancreatitis, Necrosectomy vs. Step-up Approach), the Dutch Pancreatitis Study Group noted that a stepped-up approach involving percutaneous catheter drainage and subsequent minimally invasive surgical necrosectomy was superior to primary open necrosectomy, with significantly fewer complications. In addition, 35% patients were adequately treated with percutaneous drainage alone.

In the more recent randomized PENGUIN trial (Pancreatitis, Endoscopic Transgastric vs. Primary-Necrectomy in Patients with Infected Necrosis), the Dutch Pancreatitis Study Group compared endoscopic necrosectomy \((n = 10)\) with surgical necrosectomy \((n = 10)\) using video-assisted retroperitoneal debridement \((n = 6)\) or laparotomy \((n = 4)\). Serum levels of the proinflammatory cytokine IL-6 were lower for the endoscopic compared to the surgical approach. The endoscopic approach also resulted in lower rates of new-onset multiorgan failure \((0\% \text{ vs. } 50\%)\), fistula formation \((10\% \text{ vs. } 70\%)\), and mortality \((10\% \text{ vs. } 40\%)\).

**Integrated management approach for pancreatitis necrosis**

A major distinction was drawn between the natural history of sterile and infected pancreatic necrosis, and significant diagnostic efforts (e.g. image-guided FNA of the necrosis) have been used to distinguish between them. It was believed that urgent surgery was required for all patients with infected necrosis whereas patients with sterile necrosis could be managed expectantly. However, it has been found that many patients with persistently symptomatic sterile pancreatic necrosis can also benefit from debridement. Furthermore, the pre-debridement diagnosis of sterile necrosis was incorrect 70% of the time. More importantly, management of infected necrosis has evolved through the integration of other interventional techniques. It is now clear that a graduated or stepped-up approach using antibiotics and multiple drains can bridge critically ill patients to more optimal physiological conditions and optimize the timing for mechanical debridement of infected necrosis. Surgical intervention has also become less invasive with the adaptation of laparoscopic techniques to access and debride the necrotic debris.

There are currently two laparoscopic approaches: the standard transabdominal technique via a pneumoperitoneum and the so-called retroperitoneal approach in which percutaneous drain tracts to the cavity are dilated and used to place trocars and instruments into the necrotic cavity. Although a properly timed, single open debridement may define the standard for outcomes in this condition, morbidity and mortality appear to be significantly improved by a stepped-up approach compared to proceeding straight to a more definitive open debridement.

One of the complications that can follow any of these operative debridement techniques for pancreatic necrosis is a pancreatic fistula from a viable pancreas segment disconnected from the main pancreatic duct. This problem is often surgical; requiring another operation to achieve internal drainage of the fistula into the intestine. This problem does not occur after transgastric endoscopic debridement since an internal drainage route for any disconnected pancreatic segment is a byproduct of the method for accessing the necrotic cavity. In addition, incorporation of transgastric endoscopic debridement in a stepped-up approach to pancreatic necrosis appears to yield improved morbidity and mortality compared to a stepped-up approach using laparoscopic debridement. Open surgical debridement may still be occasionally necessary for patients with fulminant infected necrotic necrosis unresponsive to less invasive maneuvers and occurring earlier in the course of severe necrotizing pancreatitis before the necrotic tissue is clearly demarcated. However, patients whose infected pancreatic necrosis can be managed by a stepped-up approach should probably undergo debridement by transgastric endoscopic techniques if local expertise exists and the necrotic cavity can be accessed in this way.

**Conclusion**

Many diagnostic and treatment options are available for acute pancreatitis. However, astute evaluation of the patient on presentation is vital for appropriate initial management of the disease. Likewise, both the timing and interpretation of imaging will significantly impact the patient triage. The Atlanta classification was introduced in 1992 and its subsequent revision in 2008 were devised to create a standardized radiographic lexicon to better classify patients according to the type of acute pancreatitis and characterize associated fluid collection and necrosis. This in turn can better direct treatment options, whether percutaneous drainage by intervention radiology or endoscopic management versus surgical necrosectomy. Overall, successful management of acute pancreatitis requires a multidisciplinary approach with coordination of multiple subspecialty services including diagnostic and interventional radiology, gastroenterology, and surgery.

**Conflict of interest**

None to declare.

**References**


