

Necrotizing Pancreatitis: Diagnosis, Imaging, and Intervention¹

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Abbreviations: ANC = acute necrotic collection, APFC = acute peripancreatic fluid collection, CBD = common bile duct, ERCP = endoscopic retrograde cholangiopancreatography, MRCP = MR cholangiopancreatography, PCD = percutaneous catheter drainage, WON = walled-off necrosis

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SA-CME LEARNING OBJECTIVES

After completing this journal-based SA-CME activity, participants will be able to:

- Recognize the imaging manifestations of necrotizing pancreatitis and related complications, as well as the terminology used in the recently revised Atlanta classification scheme.
- Describe the role of imaging in the management of necrotizing pancreatitis and related complications.
- Discuss current concepts in the management of necrotizing pancreatitis, with an emphasis on the rationale and role of interventional radiology.

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TEACHING POINTS

See last page

Acute necrotizing pancreatitis is a severe form of acute pancreatitis characterized by necrosis in and around the pancreas and is associated with high rates of morbidity and mortality. Although acute interstitial edematous pancreatitis is diagnosed primarily on the basis of signs, symptoms, and laboratory test findings, the diagnosis and severity assessment of acute necrotizing pancreatitis are based in large part on imaging findings. On the basis of the revised Atlanta classification system of 2012, necrotizing pancreatitis is subdivided anatomically into parenchymal, peripancreatic, and combined subtypes, and temporally into clinical early (within 1 week of onset) and late (>1 week after onset) phases. Associated collections are categorized as “acute necrotic” or “walled off” and can be sterile or infected. Imaging, primarily computed tomography and magnetic resonance imaging, plays an essential role in the diagnosis of necrotizing pancreatitis and the identification of complications, including infection, bowel and biliary obstruction, hemorrhage, pseudoaneurysm formation, and venous thrombosis. Imaging is also used to help triage patients and guide both temporizing and definitive management. A “step-up” method for the management of necrotizing pancreatitis that makes use of imaging-guided percutaneous catheter drainage of fluid collections prior to endoscopic or surgical necrosectomy has been shown to improve clinical outcomes. The authors present an algorithmic approach to the care of patients with necrotizing pancreatitis and review the use of imaging and interventional techniques in the diagnosis and management of this pathologic condition.

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Introduction

Acute pancreatitis results in approximately 300,000 hospital admissions in the United States each year, at a cost of approximately \$2.2 billion (1–3). This disease entity is divided into two morphologic subtypes: interstitial edematous pancreatitis and necrotizing pancreatitis. Necrotizing pancreatitis, the more severe form, is defined as necrosis of the pancreatic parenchyma with or without necrosis of the peripancreatic tissues. It occurs as a complication in 20%–30% of patients with acute pancreatitis and historically has been associated with high rates of morbidity (34%–95%) and mortality (2%–39%) (4,5). Mortality within the first 2 weeks of onset is most often due to systemic inflammatory response syndrome, which is an exaggerated inflammatory response associated with systemic organ dys-

function, immunosuppression, and organ failure. Subsequent mortality is typically the result of uncontrolled infection (6,7). Recent developments in the overall medical management of necrotizing pancreatitis and the application of new endoscopic, interventional, and surgical techniques have led to improved outcomes (6).

The management of pancreatitis begins with diagnosis, assessment of severity, and identification of complications. In 1992, the International Symposium on Acute Pancreatitis held in Atlanta developed a multispecialty, consensus-based classification system to define the various manifestations of acute pancreatitis (8). The impetus for developing such a classification system was to establish a standard, useful set of terms that could be used to manage the disease and conduct comparative outcomes research. However, the original classification system was limited and included inconsistencies in the definition of the severity of pancreatitis, imprecise and ambiguous descriptions of fluid collections, and vague radiologic criteria for characterizing complications. Furthermore, the development of new, minimally invasive nonsurgical options for managing pancreatitis and its complications increased the need for a revised classification system.

The revised Atlanta classification system, introduced in 2012, more precisely defined the clinical diagnosis, disease course, and computed tomographic (CT) manifestations of acute pancreatitis (9). According to the revised classification system, acute pancreatitis is defined clinically as a disorder that includes abdominal pain (typically epigastric in location and radiating to the back) and serum amylase or lipase levels more than three times higher than normal. If these findings are present, imaging is not necessary to make the diagnosis. If signs of systemic inflammatory response syndrome or organ failure are not present, imaging is not generally indicated. The revised classification system subdivides the clinical course of acute pancreatitis into early (<1 week) and late (>1 week) phases relative to disease onset; the late phase may persist for weeks to months. **During the early clinical phase, the severity of pancreatitis is determined predominantly by the presence of systemic inflammatory response syndrome and organ failure. The role of imaging is limited during the early phase because early morphologic changes do not correlate with clinical findings or help predict the subsequent clinical course (10). Imaging, typically CT or magnetic resonance (MR) imaging, is performed (a) to confirm the diagnosis of pancreatitis if symptoms are atypical or serum amylase or lipase levels are less than three times higher than normal, (b) when the cause of pan-**

creatitis is uncertain and an underlying neoplasm is suspected as a cause, and (c) to confirm the diagnosis of necrosis when the patient's condition does not improve or deteriorates. During the late clinical phase, severity and treatment are dictated by the presence of clinical and imaging features of persistent systemic signs of ongoing inflammation, persistent organ failure, and local and systemic complications. Imaging is essential in the late phase for (a) diagnosing and evaluating the evolution of necrotizing pancreatitis and its complications, (b) helping determine when to implement interventional radiologic, endoscopic, or surgical treatment, and (c) monitoring treatment response (11).

In this article, we discuss necrotizing pancreatitis in terms of classification and terminology, imaging, complications, diagnostic problems, and management. In addition, we review the management of associated conditions and describe the management algorithm used at our institution for patients with necrotizing pancreatitis.

Classification and Terminology in Necrotizing Pancreatitis

The revised Atlanta classification system divides pancreatic necrosis into three morphologic subtypes, depending on whether it involves pancreatic parenchyma only, peripancreatic tissues only, or both pancreatic parenchyma and peripancreatic tissues (9). The term *parenchymal necrosis* refers to nonviable pancreatic tissue, and this condition occurs in isolation in less than 5% of cases of necrotizing pancreatitis (Fig 1a) (12). It typically develops early in the disease course and is generally established by 48–72 hours after disease onset (10,12). Peripancreatic necrosis involves the peripancreatic fat; isolated peripancreatic necrosis occurs in less than 20% of cases (Fig 1b) (13). Patients with isolated peripancreatic necrosis have a better prognosis than do those with parenchymal necrosis (13). Combined necrosis is the most common morphologic subtype, occurring in approximately 75%–80% of cases of necrotizing pancreatitis (Fig 1c) (11).

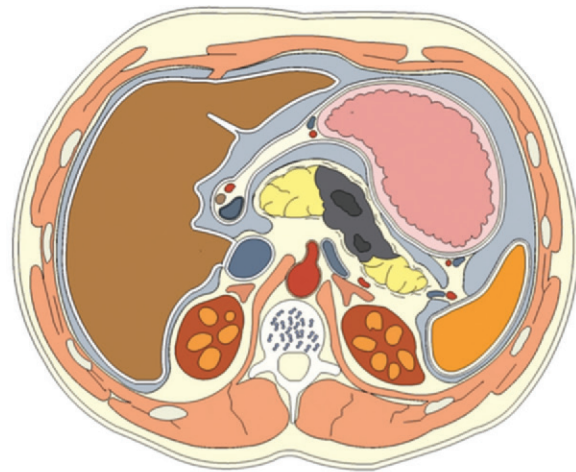
The revised Atlanta classification system subdivides collections associated with necrotizing pancreatitis according to time of disease onset. In the presence of pancreatic necrosis, a collection that develops within 4 weeks of onset and lacks a discrete wall is defined as an acute necrotic collection (ANC) (9,11). A collection that persists after 4 weeks and develops a discrete wall is defined as walled-off necrosis (WON) (Fig 2) (9,11). Both an ANC and WON can be sterile or infected (9).

Terms such as *pancreatic abscess*, *fluid collection*, and *phlegmon* are no longer accepted owing to their ambiguity. A pancreatic abscess virtually

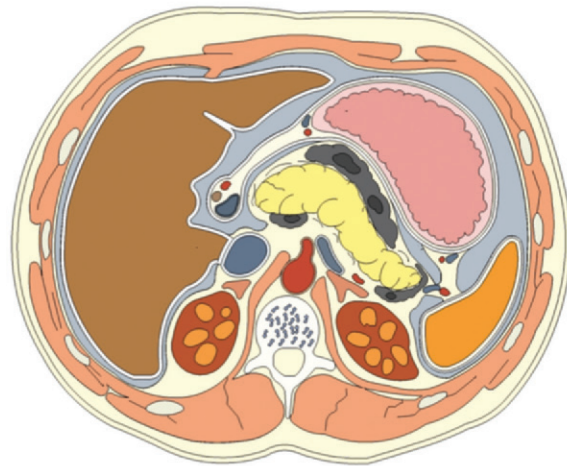
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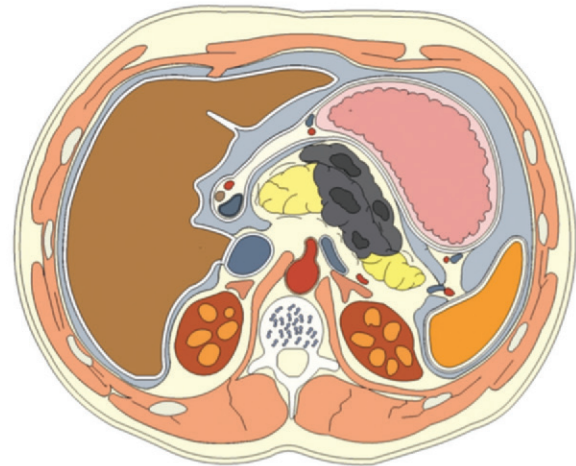
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a.



b.



c.

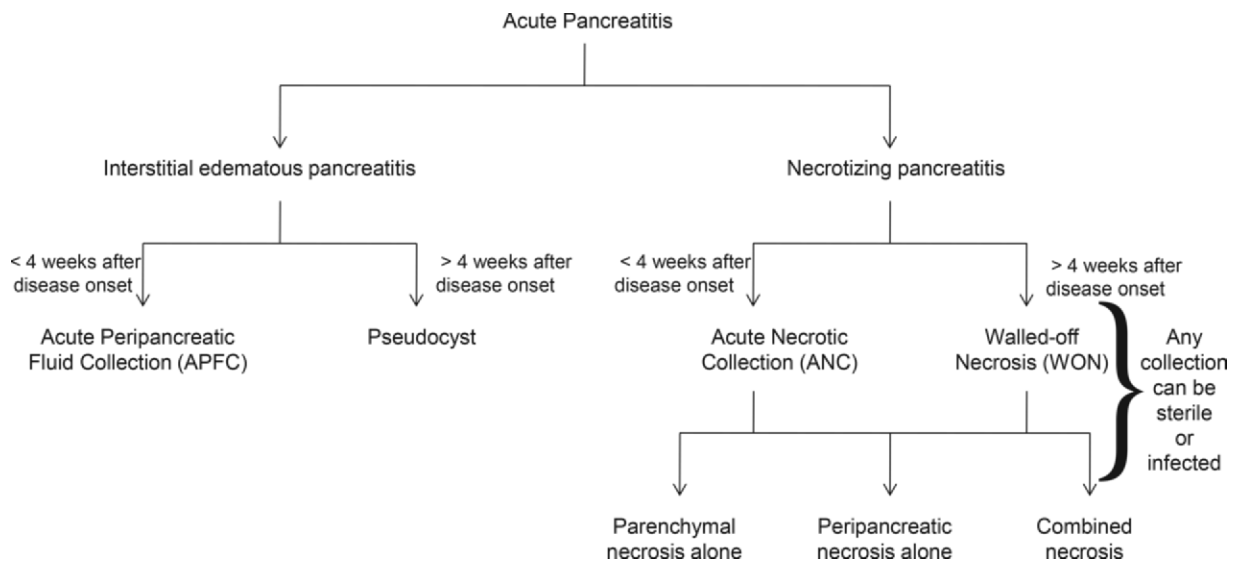


Figure 2. Chart shows the terminology used for collections in patients with acute pancreatitis.

never develops in the absence of necrotizing pancreatitis; it is the necrotic tissue that becomes superinfected, so that *infected necrosis* is a more appropriate term. Collections associated with nec-

rotizing pancreatitis may not consist of fluid alone and can arise from necrosis of both pancreatic and peripancreatic tissue. In addition, a precise definition of the collections associated with pancreatitis

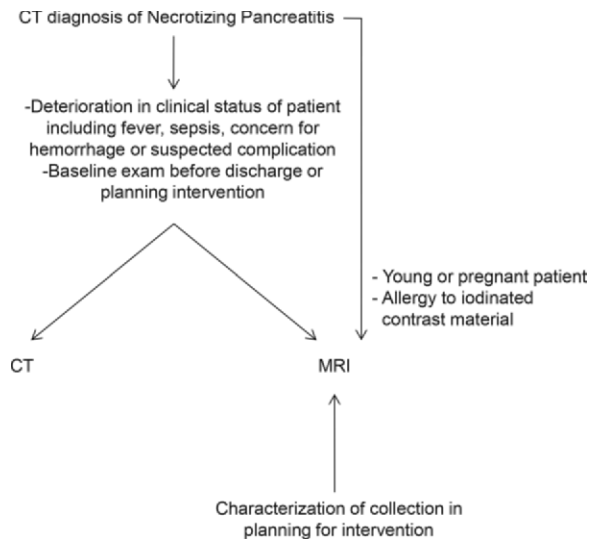


Figure 3. Chart illustrates an imaging approach for evaluating patients with suspected necrotizing pancreatitis.

is important because the treatment varies with the type of collection; for this reason, the nonspecific term *fluid collection* has fallen out of favor (9).

Imaging in Necrotizing Pancreatitis

An imaging approach for evaluating patients with suspected necrotizing pancreatitis is shown in Figure 3.

Computed Tomography

CT is the primary imaging modality used to assess the morphologic features of necrotizing pancreatitis (11). In addition to establishing the diagnosis, CT can be used to define the extent and severity of necrotizing pancreatitis and to evaluate for complications, interval change, and treatment response. CT is the most established imaging technique for characterizing the severity of necrotizing pancreatitis, with findings having been shown to correlate with outcome (14–16). Balthazar et al (14,15) established a CT severity index that graded pancreatitis based on the degree of inflammation, presence of fluid collections, and extent of necrosis. A higher CT severity index score is associated with increased morbidity and mortality. A modified CT severity index by Mortelet et al (16) included extrapancreatic complications (eg, ascites) and vascular complications in the grading system and found that the inclusion of these entities resulted in a stronger correlation with patient outcome.

Although CT can be used to accurately identify necrosis 72 hours after its onset, necrosis cannot be excluded if CT is performed earlier (15). As a result, to determine whether necrosis is present, CT is ideally performed no earlier

than 3–5 days after presentation (Fig 4) (15). At our institution, the CT protocol for suspected or known necrotizing pancreatitis involves administering water orally and scanning the abdomen and pelvis 40 seconds after the intravenous administration of 100 mL of contrast material (370 mg/mL) at a rate of 3–5 mL/sec. Images are reconstructed at 3-mm intervals in the axial, coronal, and sagittal planes. For established cases of necrotizing pancreatitis, follow-up CT is performed when there is deterioration in the clinical status of the patient or a suspected complication, and as a baseline study prior to discharge or planning intervention (17).

At approximately 40 seconds after intravenous contrast material administration, normal pancreatic parenchyma demonstrates maximum enhancement (typically, 100–150 HU); this period is considered the pancreatic parenchymal phase. Pancreatic necrosis is suspected when any region of pancreatic parenchyma demonstrates an attenuation of less than 30 HU during the pancreatic parenchymal phase (15). Although pancreatic necrosis may initially appear homogeneous, the regions of necrosis can become heterogeneous as necrotic tissue gradually becomes liquefied (11). The severity of necrotizing pancreatitis at imaging is determined on the basis of the extent of parenchymal involvement by necrosis (ie, <30%, 30%–50%, and >50%) (14). When the extent of parenchymal involvement is less than 30%, the low attenuation due to decreased enhancement of the small region of necrosis may mimic the low attenuation of the gland seen with acute interstitial edematous pancreatitis, making the diagnosis of necrosis less reliable. In these cases, follow-up CT may be required (14).

Peripancreatic necrosis is a more difficult diagnosis to make at CT, since this modality is not able to demonstrate the presence or absence of fat perfusion. Therefore, the diagnosis of peripancreatic necrosis is suggested by the presence of increased attenuation, linear stranding, and fluid collections interspersed among the peripancreatic fat. Recognition of peripancreatic necrosis is difficult in the first week after onset because the increased attenuation, linear stranding, and fluid collections associated with acute interstitial edematous pancreatitis can have a similar appearance. However, the diagnosis of peripancreatic necrosis may be favored when the regions of increased attenuation have a heterogeneous appearance. After 1 week, the heterogeneous peripancreatic fat and the liquefied components among the fat become more apparent, so that peripancreatic necrosis can be diagnosed with greater confidence (Fig 5) (11,13). Combined necrosis is diagnosed when imaging features of

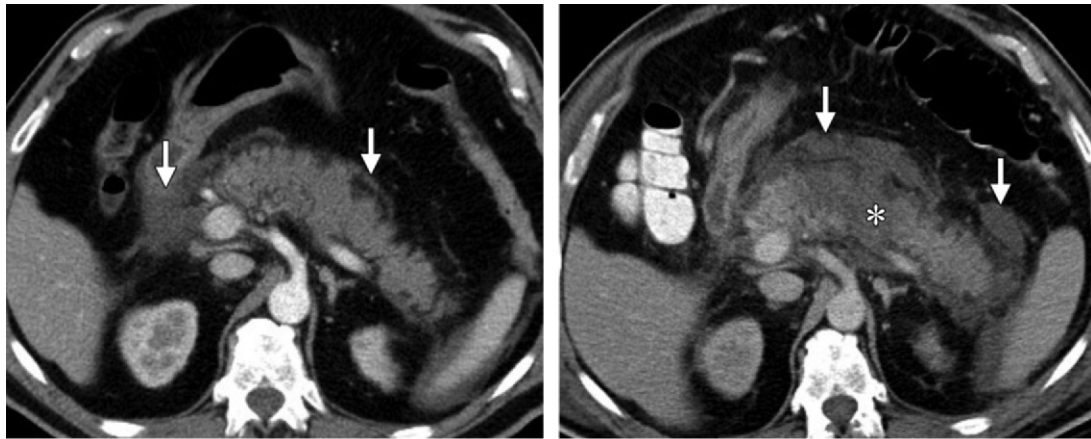


Figure 4. Pancreatic necrosis in a 65-year-old man. **(a)** Axial contrast material–enhanced CT image obtained 2 days after the onset of acute abdominal pain shows peripancreatic fluid and stranding (arrows) and normal-appearing pancreatic parenchyma. **(b)** Axial contrast-enhanced CT image obtained 5 days later owing to the patient's worsening clinical condition reveals an ill-defined hypoattenuating region in the body of the pancreas (*), a finding that suggests pancreatic necrosis. Peripancreatic fluid and stranding (arrows) are also seen.

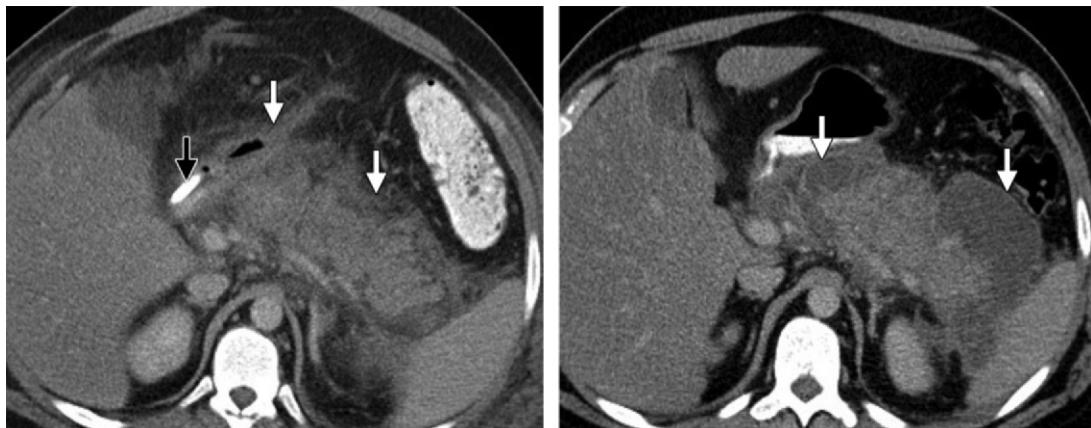


Figure 5. Peripancreatic necrosis in a 22-year-old man. **(a)** Axial contrast-enhanced CT image shows stranding, increased attenuation, and a heterogeneous appearance of the peripancreatic fat around the body and tail of the pancreas (white arrows). A feeding tube is seen in the stomach (black arrow). **(b)** Axial contrast-enhanced CT image acquired 20 days later reveals evolution of the heterogeneous peripancreatic collection into well-defined WON (arrows).

both parenchymal and peripancreatic necroses are present (Fig 6) (11). After 4 weeks, the acute pancreatic or peripancreatic inflammation and collections generally evolve into WON, which appears at CT as a heterogeneous or homogeneous collection with a well-defined wall (Fig 6) (18). Approximately 60% of ANCs evolve into sterile WON, 20% are complicated by infection, and the remaining 20% resolve spontaneously (19).

MR Imaging

Although MR imaging is not the first-line imaging modality for evaluating patients with suspected acute necrotizing pancreatitis, it is an acceptable alternative to CT in patients with an

allergy to iodinated contrast material. Because imaging may be performed repeatedly, MR imaging may be preferred in young or pregnant patients to minimize radiation exposure. Unenhanced MR imaging can be used in patients with renal impairment (20). In addition, MR imaging is more sensitive than CT for detecting gallstones and hence is preferred in patients with suspected choledocholithiasis.

When catheter drainage of a fluid collection is contemplated, MR imaging may be helpful in assessing the collection's amenability to drainage by identifying nonliquefied material (eg, debris or necrotic tissue) that is difficult to remove with percutaneous catheter drainage (PCD)

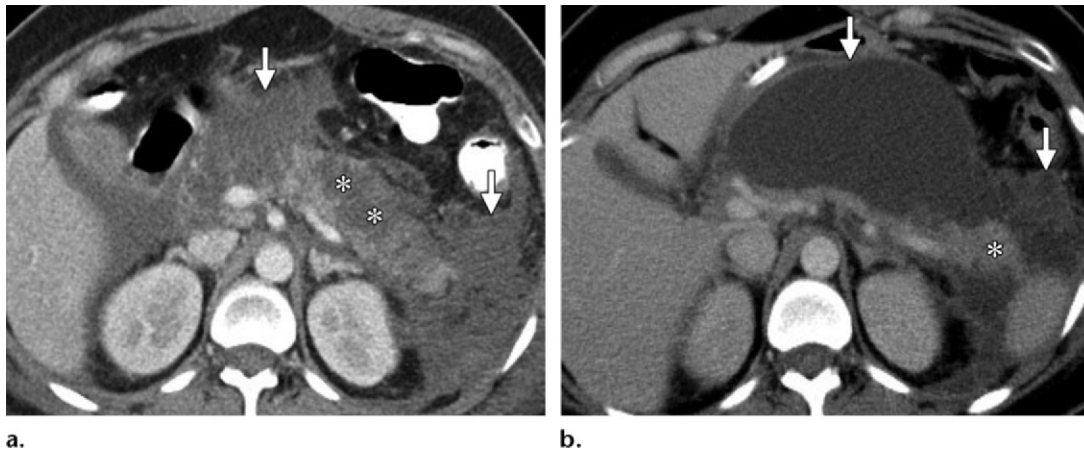


Figure 6. Combined necrosis in a 46-year-old man. **(a)** Axial contrast-enhanced CT image shows an ill-defined hypoattenuating region in the body and tail of the pancreas (*), along with ill-defined heterogeneous peripancreatic fluid, stranding, and increased fat attenuation (arrows). **(b)** Axial contrast-enhanced CT image acquired 6 weeks later reveals evolution of the pancreatic and heterogeneous peripancreatic collection into well-defined WON (arrows). Residual pancreatic parenchyma is seen in the tail (*).

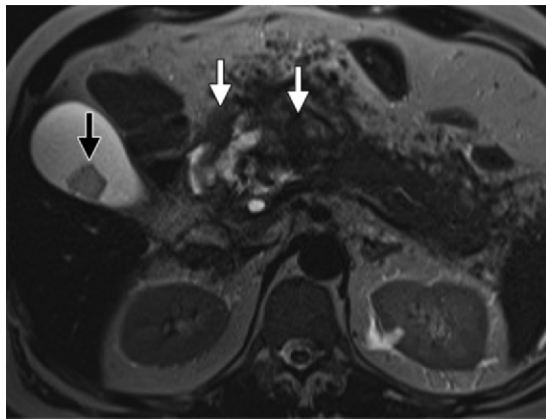


Figure 7. Combined necrosis in a 42-year-old man with gallstones. Axial T2-weighted MR image shows an irregular, heterogeneous pancreatic-peripancreatic collection with hyperintense fluid and hypointense nonliquefied components (white arrows), as well as a gallstone (black arrow) that was not seen at CT.

alone (Fig 7) (9,11,21). If nonliquefied material is present, endoscopic necrosectomy or surgical débridement may be preferred (22). MR imaging is less sensitive than CT for detecting gas in collections, the presence of which can suggest infection (23,24).

At our institution, the MR imaging protocol for evaluating pancreatitis includes axial and coronal single-shot fast spin-echo T2-weighted, axial fat-saturated fast spin-echo T2-weighted, gradient-echo in-phase and opposed-phase, unenhanced fat-saturated gradient-echo T1-weighted, and dynamic gadolinium-enhanced fat-saturated gradient-echo T1-weighted sequences. Heavily T2-weighted coronal two- and three-dimensional MR cholangiopancreatographic (MRCP) images are also acquired prior to intravenous contrast material administration. MRCP can be performed before and after the intravenous administration of secretin (0.2 µg/kg body weight over 1 minute) and involves acquisition of coronal two-dimensional MRCP im-

ages in the plane of the pancreatic duct every 30 seconds for 10 minutes.

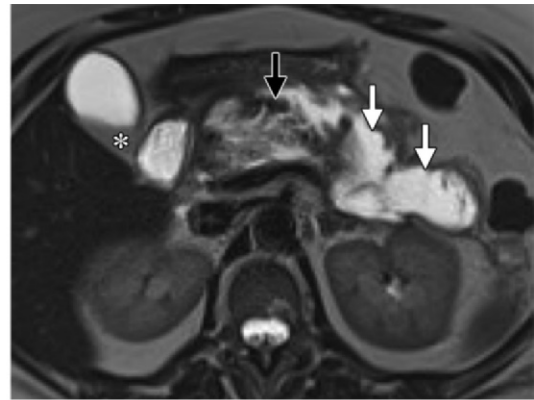
At MR imaging, pancreatic necrosis appears as a region of pancreatic parenchyma that does not enhance (Fig 8). As with CT, the diagnosis of peripancreatic necrosis becomes more certain more than 1 week after disease onset, when the necrosis becomes more defined. Peripancreatic stranding, fat heterogeneity, and necrotic collections such as an ANC and WON are best assessed on T2-weighted images, with liquefied components appearing hyperintense and nonliquefied components appearing hypointense. WON demonstrates a well-defined, T2-hypointense enhancing wall (Fig 9). MR imaging is more sensitive than CT for the detection of hemorrhage, which is best seen on T1-weighted images (Fig 10) (23,25). MRCP is useful for detecting choledocholithiasis and mass effect on the common bile duct (CBD), evaluating the integrity of the pancreatic duct, and detecting communication of a collection with the pancreatic duct (Fig 11). Secretin administration results in increased pancreatic fluid secretion, which distends the pancreatic duct and improves its visibility, as well as the visibility of any duct disruptions or fistulas (26). MRCP with secretin administration is typically performed after resolution of the acute inflammatory changes to avoid exacerbating the inflammation (23).



Figure 8. Pancreatic necrosis in a 62-year-old man. Axial contrast-enhanced fat-saturated T1-weighted MR image shows a nonenhancing focal area in the body of the pancreas (arrow), a finding that suggests necrosis.



a.

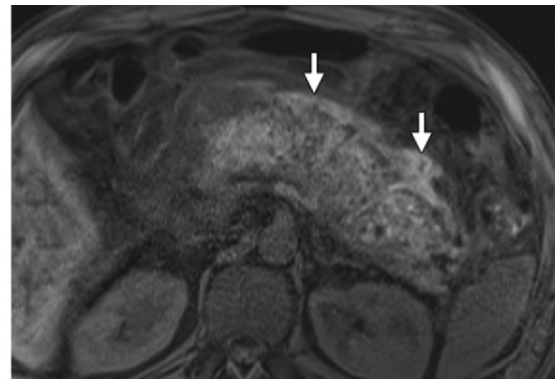


b.

Figure 9. Combined necrosis in a 48-year-old woman. **(a)** Axial contrast-enhanced CT image shows an evolving, mildly heterogeneous pancreatic-peripancreatic necrotic collection involving the neck, body, and tail of the pancreas (arrows). **(b)** Axial T2-weighted MR image shows a well-defined, walled-off, heterogeneous pancreatic-peripancreatic collection, with a fluid component that appears hyperintense (white arrows) and necrotic nonliquefied material that appears hypointense (black arrow). Hypointense sludge is seen in the gallbladder (*).



a.



b.

Figure 10. Combined necrosis with hemorrhage in a 48-year-old woman. **(a)** Axial contrast-enhanced CT image obtained for worsening abdominal pain shows a well-defined, heterogeneous pancreatic-peripancreatic collection (arrows). **(b)** Axial fat-saturated T1-weighted MR image obtained 2 days later for the evaluation of abdominal pain reveals hyperintense signal in the body and tail of the pancreas and in the peripancreatic region (arrows), findings that are suggestive of hemorrhage.

Transabdominal Ultrasonography

Transabdominal ultrasonography (US) has a limited role in the evaluation of patients with necrotizing pancreatitis. Compared with CT, US is

more sensitive for detecting cholelithiasis but less sensitive for detecting distal choledocholithiasis (Fig 12) (27). US has a limited role in evaluating the extent of necrosis and complications, since

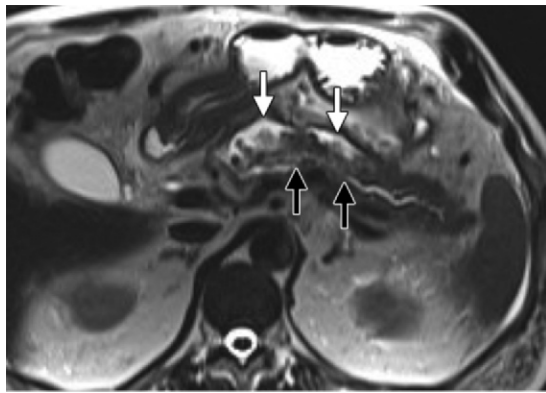
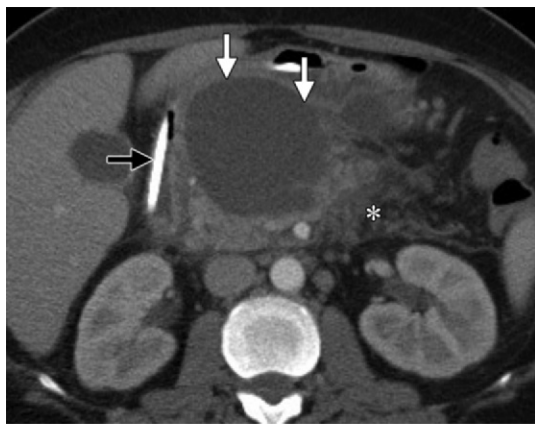
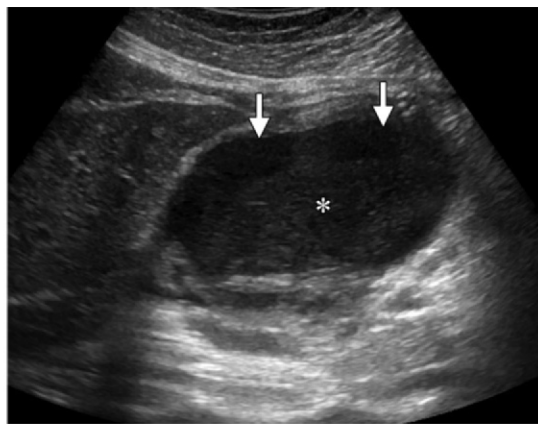


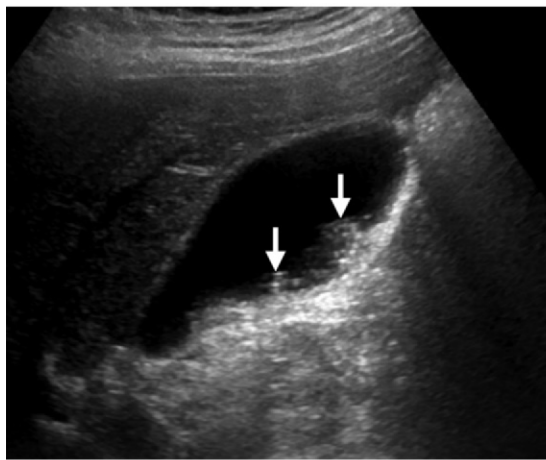
Figure 11. Pancreatic ductal disruption in a 52-year-old man with combined necrosis. Axial T2-weighted MR image shows walled-off pancreatic-peripancreatic necrosis (white arrows) with an internal fluid component and necrotic nonliquefied material involving the body of the pancreas. There is also a 2-cm breach in the continuity of the pancreatic duct (black arrows) in the pancreatic body, a finding that suggests ductal disruption.



a.



b.



c.

Figure 12. Combined necrosis in a 36-year-old man with gallstones. **(a)** Axial contrast-enhanced CT image shows a well-defined, mildly heterogeneous pancreatic-peripancreatic collection (white arrows) with peripancreatic stranding (*). Note the feeding tube in the stomach (black arrow), which was inserted to bypass the mass effect on the stomach by the collection. **(b)** Transverse transabdominal US image through the upper abdomen shows a well-defined hypoechoic collection (arrows) that corresponds to the CT findings, with internal echogenic material (*) representing the nonliquefied components. **(c)** US image shows echogenic gallstones and sludge (arrows), findings that were not seen at CT.

these findings are often obscured in patients who are large or have large amounts of bowel gas. However, in patients with contraindications for both CT and MR imaging, US may be useful for demonstrating the presence of nonliquefied material within a collection (28).

Endoscopic Retrograde Cholangiopancreatography

Endoscopic retrograde cholangiopancreatography (ERCP) has no primary role in characterizing the morphology of necrotizing pancreatitis (11) and

could lead to complications such as pancreatitis exacerbation, bleeding, and bowel perforation. Because of its less invasive nature, MRCP is preferred for detecting choledocholithiasis and pancreatic ductal strictures or disruptions (29). Hence, ERCP is generally reserved for therapeutic applications such as CBD stone removal or pancreatic duct stent placement used to treat strictures and disrupted ducts (Fig 11) (22).

Endoscopic US

Endoscopic US involves using an echoendoscope that generates high-frequency sound waves, which pass through the wall of the stomach or

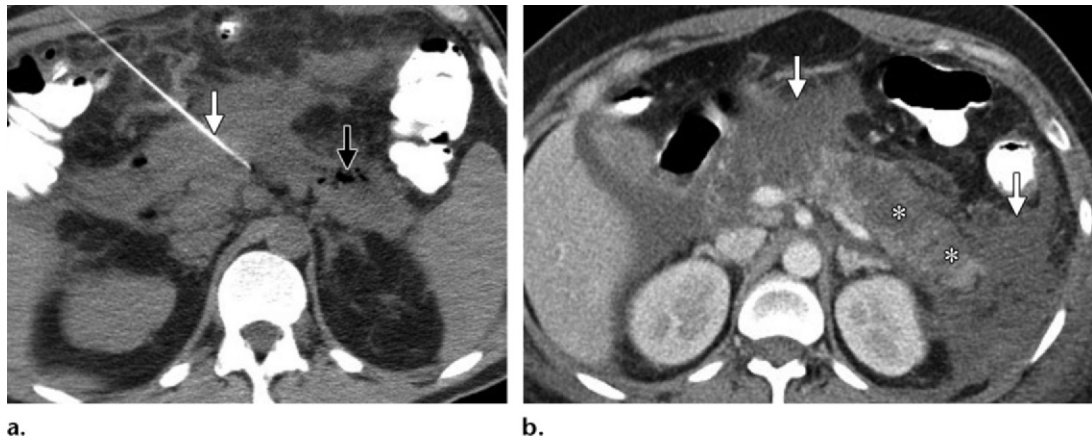


Figure 13. Infected pancreatic collection in combined necrosis. **(a)** Axial nonenhanced CT image obtained in a 42-year-old woman during CT-guided percutaneous needle aspiration shows the needle (white arrow) within a suspected infected collection, as evidenced by the presence of gas (black arrow). **(b)** Axial contrast-enhanced CT image in a 56-year-old man with combined necrosis involving the body and tail of the pancreas (*) shows a heterogeneous peripancreatic collection (arrows). No gas locules were seen at CT despite the patient's fever and elevated white blood cell count. Infection was confirmed at CT-guided needle aspiration.

duodenum to help evaluate the pancreatic parenchyma and ductal system. Endoscopic US combines the diagnostic capabilities of US with the interventional advantages of endoscopy. Like transabdominal US, endoscopic US can be used to identify the nonliquefied components of collections in preparation for endoscopic drainage and débridement. Endoscopic US is also sensitive for detecting CBD stones, without the risks associated with ERCP (22).

Complications of Necrotizing Pancreatitis

Infection

Infection occurs as a complication in 20% of patients with necrotizing pancreatitis and is thought to result from bacterial translocation from the gut to adjacent necrotic pancreatic parenchyma. The most common bacterial organisms include *Escherichia coli*, *Staphylococcus aureus*, and *Enterococcus faecalis*, although several other organisms may be found (5). Infection can occur at any time during the course of the disease but most commonly occurs 2–4 weeks after presentation (30). Patients with infected necrosis typically present with fever, tachycardia, and an elevated white blood cell count. Clinical presentation alone is not diagnostic for infection, and patients with sterile necrosis may present with similar symptoms. At imaging, the presence of gas within a collection suggests infection, although gas is found in a minority of cases of confirmed infection (12%–22%), and the absence of gas does not signify the absence of infection (30,31). Gas can also be found in uninfected collections as a result of gastrointestinal

fistulas (32). Because there are no symptom constellations or imaging findings that are diagnostic for infection, imaging-guided percutaneous needle aspiration is indicated in patients suspected of being infected (Fig 13). Infected necrosis carries a high mortality rate of 25%–70%; therefore, the diagnosis needs to be pursued aggressively when infection is suspected. When infection is confirmed, some form of intervention is usually indicated (19).

Inflammation and Mass Effect on Adjacent Organs

Necrotizing pancreatitis, with its associated inflammatory changes and collections, may displace and compress adjacent organs. Obstruction of the stomach or bowel and hydronephrosis are possible complications of the mass effect caused by nearby collections and inflamed fat (Fig 14). Inflammatory changes may also secondarily cause bowel wall thickening, mural hyperenhancement, and adjacent fat stranding (Fig 14). Patients with severe gastrointestinal tract obstruction or large abdominopelvic fluid collections are at risk for abdominal compartment syndrome, in which increased intraabdominal pressure results in organ ischemia and further tissue necrosis (33).

Biliary Obstruction

Biliary obstruction can result from choledocholithiasis, mass effect from pancreatic inflammation or a collection, or biliary stricture from exposure to pancreatic proteolytic enzymes. A strictured bile duct may appear tapered, compressed, or simply occluded with upstream biliary dilatation, and it may or may not demonstrate mural en-



Figure 14. Gastric outlet obstruction in a 58-year-old woman with combined necrosis. Axial contrast-enhanced CT image demonstrates evolving pancreatic-peripancreatic necrosis (black arrow) causing inflammatory circumferential thickening of the second part of the duodenum (white arrow), with resultant gastric outlet obstruction and a distended stomach (*).

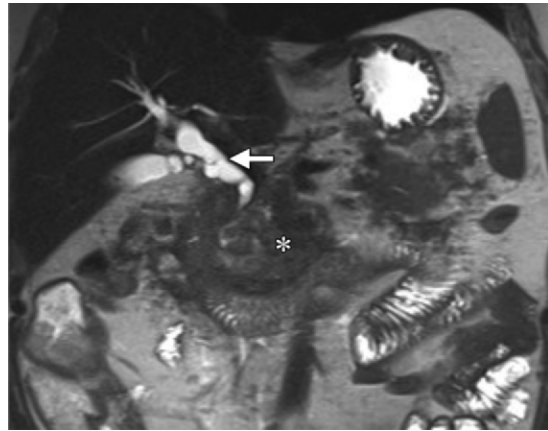


Figure 15. Biliary obstruction in a 57-year-old woman with combined necrosis. Coronal T2-weighted MR image shows combined necrosis in the region of the pancreatic head (*), with 15-mm dilatation of the proximal CBD (arrow) due to mass effect on the distal CBD.

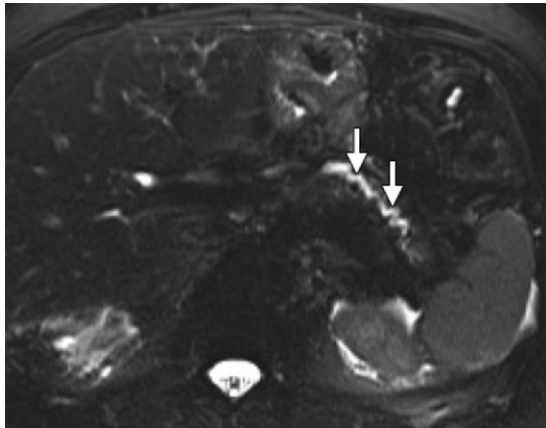


Figure 16. Pancreatic duct stricture in a 56-year-old man with combined necrosis. Axial fat-saturated T2-weighted MR image shows pancreatic ductal dilatation of 6 mm in the body and tail (arrows), a finding caused by a pancreatic duct stricture in the region of the proximal body.

hancement (Fig 15). MRCP is particularly helpful for delineating the biliary system, identifying the narrowed or occluded segment, and identifying the cause of the obstruction (34).

Pancreatic Duct Stricture

Main pancreatic duct stricture is a late complication of necrotizing pancreatitis. Strictures develop secondary to fibrosis from resolving inflammation, or as a result of healing after the successful interventional drainage of a necrotic collection (35). Strictures may be single or multiple, may result in upstream dilatation of the pancreatic duct, and can be diagnosed with CT, MRCP, ERCP, or endoscopic US (Fig 16) (35–37).

Disconnected Pancreatic Duct

Disconnected pancreatic ducts result from necrosis of the central pancreas (commonly the neck or body) or from a therapeutic intervention that disrupts the main pancreatic duct, and they occur in approximately 40% of patients with pancreatic necrosis (38). When there is residual upstream functioning pancreatic tissue, a disrupted duct results in persistent leakage of pancreatic fluid from the viable upstream pancreas and leads to accumulation of fluid around the pancreas, pancreatic ascites, or a pancreaticopleural fistula. Although most, if not all, fluid collections are the result of some form of communication with the pancreatic ductal system, disruption of the main duct often leads to persistent and growing collections around the pancreas. At CT or MR imaging, a disrupted duct is suggested by a large or growing collection involving the neck or body of the pancreas and a viable segment of upstream body or tail. The duct in the upstream pancreas may or may not be dilated and may be seen communicating directly with the collection; disruption is suggested when the duct is oriented perpendicular to the collection (39). A disrupted duct and the presence of a fistula can also be suspected following PCD when there is persistent catheter drainage of amylase-rich fluid, despite the resolution of the fluid collection. Both ERCP and MR imaging with MRCP can help confirm the disruption and identify the site of the fistula (Fig 11) (38).

Pseudoaneurysm

A pseudoaneurysm develops when an arterial vessel wall is weakened by pancreatic proteolytic enzymes and is a typically late and potentially life-threatening complication of pancreatic necrosis



Figure 17. Pseudoaneurysm in a 32-year-old man with necrotizing pancreatitis. Coronal reformatted CT image demonstrates an outpouching from the left gastric artery (arrow), a finding that is consistent with pseudoaneurysm. The pseudoaneurysm was treated surgically owing to the inability to access the left gastric artery with a percutaneous endovascular approach.

(40). At CT, MR imaging, or angiography, a pseudoaneurysm appears as a focal outpouching of a vessel within the necrotic region (Fig 17). A mural thrombus may also be seen (40). At US, turbulent arterial flow may be seen within an anechoic structure (41). The artery that is most frequently involved by pseudoaneurysm formation in the setting of necrotizing pancreatitis is the splenic artery (up to 10% of patients), followed (in descending order) by the gastroduodenal, pancreaticoduodenal, hepatic, and left gastric arteries (41,42). Pseudoaneurysms can rupture into the necrotic collection, gastrointestinal tract, peritoneum, or pancreatic parenchyma (41).

Hemorrhage

Spontaneous hemorrhage in necrotizing pancreatitis can occur from erosion of vasculature by necrosis or from rupture of a pseudoaneurysm or varices. Hemorrhage can occur within the pancreatic parenchyma, fluid collections, or the gastrointestinal tract (43). Although its overall rate of occurrence in pancreatitis is not known, spontaneous hemorrhage probably occurs in approximately 1%–5% of cases; mortality rates of 34%–52% have been reported (40–43). The splenic artery, portal vein, splenic vein, and other smaller peripancreatic vessels are the most common sources of bleeding (44). Hemorrhage manifests at CT as a region of high attenuation, typically in an area of necrosis (Fig 18). At MR imaging, the appearance of hemorrhage on T1- and T2-weighted images varies with the age of the bleeding; subacute hemorrhage appears T1 and T2 hyperintense (Fig 10b) (44).

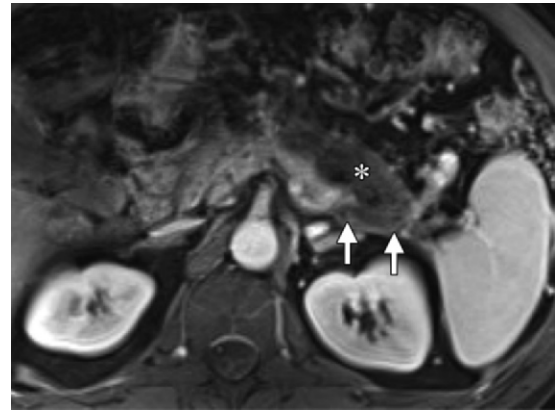


Figure 19. Splenic vein thrombosis in a 49-year-old woman with combined necrosis. Axial contrast-enhanced fat-saturated T1-weighted MR image demonstrates a chronically thrombosed splenic vein (arrows) and combined necrosis in the body and tail of the pancreas (*).

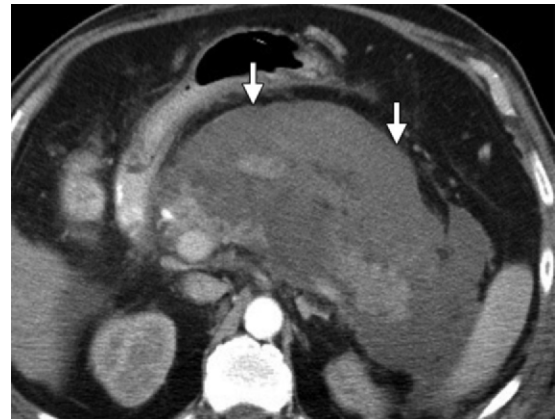


Figure 18. Hemorrhage in a 44-year-old man with combined necrosis. Axial contrast-enhanced CT image obtained for the evaluation of worsening abdominal pain shows hyperattenuating material (arrows) within the region of combined necrosis, a finding that is suggestive of hemorrhage.

Venous Thrombosis

Venous thrombosis results from a multifactorial process involving local prothrombotic inflammatory factors, reduced venous flow, and mass effect on a venous structure from adjacent necrotic tissue and collections. Acute venous thrombosis appears as focal or complete nonenhancement of an expanded venous structure. In chronic cases, scarring results in a diminutive, less well-visualized vein and multiple collateral vessels (Fig 19) (45). The splenic vein is the most common site for thrombosis (up to 23% of cases of acute pancreatitis); the superior mesenteric and portal veins are less commonly affected (45,46). Splenomegaly may result, and collateral vessels may increase the risk of bleeding during subsequent intervention or surgery (45).

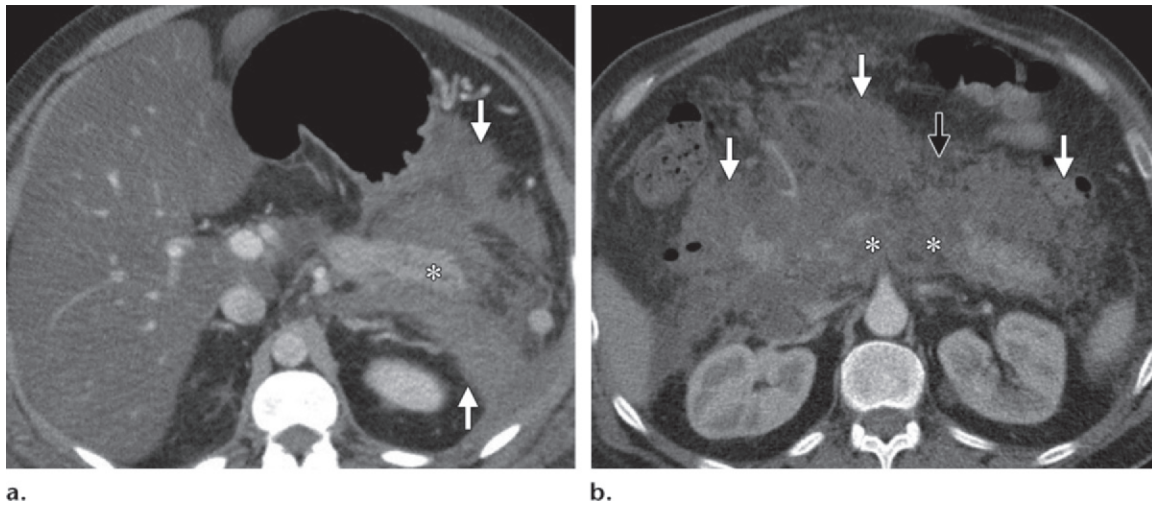


Figure 20. Acute interstitial edematous pancreatitis versus acute necrotizing pancreatitis. **(a)** Axial contrast-enhanced CT image in a 36-year-old man with acute interstitial edematous pancreatitis shows an ill-defined peripancreatic collection and stranding around the body and tail of the pancreas (arrows) with mild heterogeneity of the pancreatic tail (*). **(b)** Axial contrast-enhanced CT image in a 42-year-old man with acute necrotizing pancreatitis shows ill-defined hypoattenuating areas in the head and body of the pancreas (*) and an ill-defined, heterogeneous peripancreatic collection (white arrows) with interspersed fat (black arrow) and stranding around the head, body, and tail.

Diagnostic Problems in Necrotizing Pancreatitis

Acute Peripancreatic Fluid Collection versus ANC

An acute peripancreatic fluid collection (APFC) is a collection that develops within 4 weeks after onset of acute interstitial edematous pancreatitis, whereas an ANC is a collection that develops within 4 weeks after onset of acute necrotizing pancreatitis (Fig 2). Both collections have no discernable walls (Fig 20). An APFC contains amylase- and lipase-rich fluid and develops as a result of pancreatic or peripancreatic inflammation, or from a ruptured pancreatic ductal side branch (11). On the other hand, an ANC is a collection that contains both liquefied and nonliquefied necrotic material (11). Distinguishing an APFC from an ANC in the first week after onset may not be possible with CT; both collections may be homogeneous and nonenhancing and demonstrate fluid attenuation (47). If a collection appears heterogeneous, or if hemorrhage or fatty tissue is present, it can be classified as an ANC (47). Moreover, if pancreatic parenchymal necrosis is present, an associated collection is classified as an ANC. Distinguishing an ANC from an APFC is more challenging when necrosis is solely peripancreatic. Beyond 1 week after onset, collections associated with peripancreatic necrosis become more heterogeneous and are more readily distinguished from APFC (11). MR imaging is more helpful than CT in this re-

gard, since it can be used to detect nonliquefied components that allow classification of a collection as an ANC (47).

Pseudocyst versus WON

Pseudocysts and WON are both late-phase (>4 weeks after onset) collections that develop over time from nonnecrotic (APFC) and necrotic (ANC) collections, respectively (Fig 2). Both pseudocysts and WON have well-defined, nonepithelialized enhancing walls. Pseudocysts contain homogeneous fluid (hypoattenuating at CT, T2 hyperintense at MR imaging) and are only peripancreatic (Fig 21) (48). WON contains necrotic material—often a mixture of fat and fluid—and can involve both pancreatic and peripancreatic tissue. The diagnosis of WON is also favored when a pancreatic collection grows, extends to the paracolic space, and has an irregular border (18). However, any collection that occupies or replaces pancreatic parenchyma is classified as WON, regardless of its appearance (18). Pseudocysts are more likely to be associated with main pancreatic ductal dilatation (>3 mm), possibly as a result of the compression of pancreatic parenchyma. In patients with WON, ductal dilatation is less likely to occur because the pancreatic fluid simply leaks into the collection (18).

Differentiating a pseudocyst from WON is important because WON typically does not respond to endoscopic cyst gastrostomy or PCD with small-bore (10-F or smaller) catheters. Treatment of WON typically requires surgical or endoscopic

Teaching Point

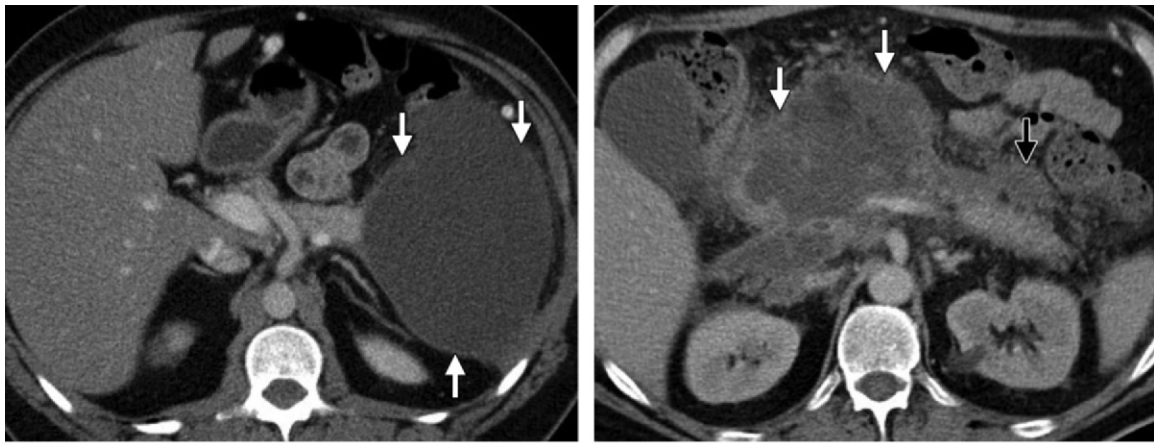


Figure 21. Pseudocyst versus WON. **(a)** Axial contrast-enhanced CT image acquired 4 weeks after the onset of disease in the same patient as in Figure 20a shows a well-defined, homogeneous peripancreatic collection around the tail of the pancreas (arrows), a finding that is compatible with a pseudocyst. **(b)** Axial contrast-enhanced CT image acquired 4 weeks after the onset of disease in the same patient as in Figure 20b reveals evolving, relatively well-defined, walled-off combined necrosis in the region of the head and body of the pancreas (white arrows), with peripancreatic necrosis seen around the tail (black arrow).

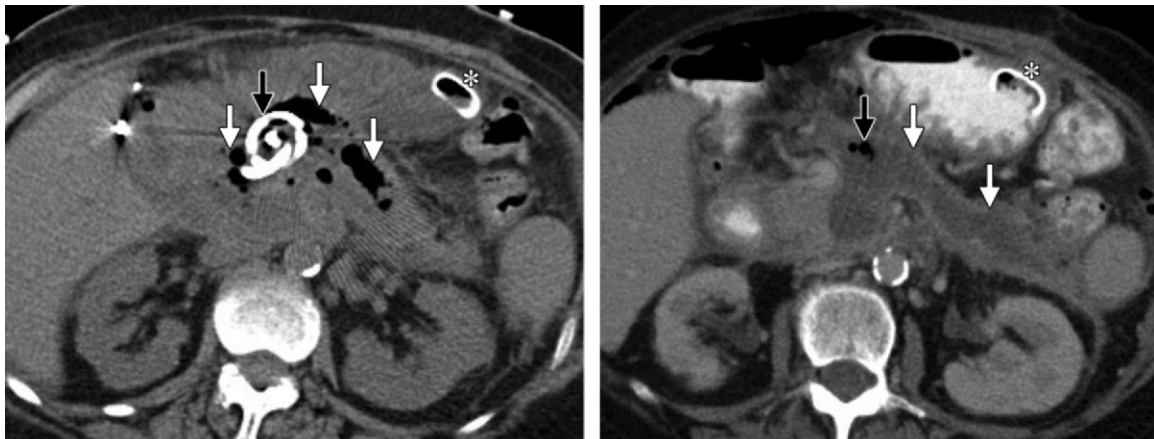


Figure 22. Postnecrosectomy changes in a 53-year-old man with combined necrosis. **(a)** Axial nonenhanced CT image obtained 20 days after necrosectomy shows fluid and gas (white arrows) and a loop drainage catheter (black arrow) in the endoscopic necrosectomy bed. Note also the presence of a percutaneous gastrostomy catheter (*). **(b)** Axial contrast-enhanced CT image obtained 60 days after necrosectomy shows a decrease in the amount of gas (black arrow) in the necrosectomy bed (white arrows). The loop drainage catheter has been removed, but the percutaneous gastrostomy catheter (*) remains in place.

necrosectomy, or PCD using large-bore catheters with frequent irrigation to evacuate the nonliquefied components (18).

Sterile versus Infected Necrosis

As described previously, the presence of gas in an area of necrosis, in the absence of previous intervention or spontaneous communication with the bowel, may indicate the presence of infection. CT attenuation measurements cannot be used to distinguish sterile from infected necrosis (30). With any signs or symptoms of infection, imaging-guided percutaneous needle

aspiration for Gram staining and culture is needed to definitively diagnose infection within a region of necrosis (Fig 13) (49).

Postnecrosectomy Changes versus Infected Collection

Necrosectomy is a procedure in which necrotic pancreatic or peripancreatic tissue is removed, along with drainage of accompanying fluid collections. Postnecrosectomy imaging findings may mimic infection, since the surgical bed and residual collection may contain tissue, fluid, and gas (Fig 22). The presence of gas is expected

when there are indwelling percutaneously placed or surgical catheters or if a stent has been placed between the collection and the stomach following endoscopic necrosectomy. Postnecrosectomy changes are expected to resolve; any interval increase in the size of a collection or formation of a new collection raises the possibility of infection. Microbiologic analysis is necessary to confirm infection (49).

Management of Necrotizing Pancreatitis

Patients with necrotizing pancreatitis are best managed by a team of gastroenterologists, radiologists, and surgeons. Management depends on several factors, including disease severity and phase and the presence of complications.

Initially, management of necrotizing pancreatitis is primarily conservative and includes fluid resuscitation, nutritional support, prophylactic antibiotics, and, in severely ill patients, admission to an intensive care unit. Enteral feeding is recommended for patients who are not expected to resume oral feeding within 5–7 days of disease onset, since it helps maintain the intestinal barrier and prevent translocation of gut bacteria. Enteral feeding is typically accomplished by bypassing the bowel in the immediate region of inflammation, typically by means of a nasojejunal tube or jejunostomy. Compared with parenteral feeding, enteral feeding has been associated with decreased mortality, lower rates of infection, and reduced organ failure (6). Prophylactic antibiotic therapy is often implemented early, although its efficacy has been debated (6).

The usage of interventional radiologic, endoscopic, and surgical interventions for necrotizing pancreatitis varies among institutions (22). Historically, open surgical necrosectomy has been considered the definitive treatment for symptomatic pancreatic necrosis, including infected necrosis. The need for open surgery was largely predicated on the fact that necrotic tissue and other nonliquefied material could not be adequately drained percutaneously or endoscopically with small (10-F or smaller) catheters. However, a variety of minimally invasive non-surgical techniques have been developed to treat necrotic collections. These procedures include imaging-guided percutaneous drainage with large (>10-F) catheters, endoscopic necrosectomy (typically via the stomach), laparoscopic necrosectomy, video-assisted retroperitoneal débridement, and hybrid techniques (50). Relative to open surgical necrosectomy, these minimally invasive techniques are shorter procedures, carry less risk of complications, and result in shorter hospital stays. Most important, they increase the

number of patients who are able to undergo a therapeutic intervention (51).

All interventions, particularly when performed in acutely ill patients, are associated with high morbidity and mortality rates due to heightened systemic inflammatory response during this time period, as well as an increased risk of hemorrhage (22,52). In general, delaying intervention until at least 4 weeks after onset also allows necrotic pancreatic parenchyma to be more clearly distinguished from viable pancreatic parenchyma, thereby preserving viable pancreatic tissue and reducing the risk of pancreatic insufficiency (53). However, interventional radiologic procedures such as percutaneous needle aspiration and PCD may still be performed earlier in patients with suspected or proved infection (52). There is a growing trend to treat infected necrosis with supportive care and antibiotics alone when there are no signs of sepsis, until the necrotic collection is more walled off (28). Asymptomatic sterile WON does not require interventional management (22).

Imaging-guided Percutaneous Methods

Imaging-guided percutaneous methods for necrotizing pancreatitis include fine-needle aspiration, catheter drainage of collections, and débridement of necrosis (54).

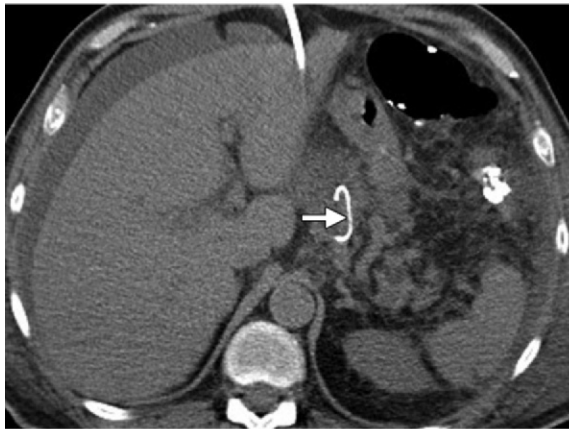
Fine-Needle Aspiration.—Fine-needle aspiration of the necrotic collection is performed to obtain a sample for microbiologic analysis when empiric antibiotics are not effective in patients with signs of infection (11). Needle aspiration is typically performed using a fine (20-gauge) needle under CT guidance. The most liquefied portion of the collection is typically targeted, with care taken to avoid transgressing bowel (Fig 13) (55). Collections are aspirated to assess for infection, direct antibiotic therapy, and help determine the need for further intervention. It is helpful for the care team to decide the course of action before the procedure; if an infection is found and endoscopic or surgical necrosectomy is planned, the procedure can be discontinued after fine-needle aspiration. Alternatively, fine-needle aspiration can be performed preparatory to PCD during the same session.

Percutaneous Catheter Drainage.—PCD is performed to control the signs and symptoms of necrotizing pancreatitis, such as sepsis due to infected necrosis, mass effect, obstruction, inability to tolerate feeding, failure to thrive, and persistent pain (54). It can either represent definitive therapy or serve as bridging therapy in a “step-up” approach to endoscopic or surgical necrosectomy (51). PCD

Figure 23. PCD of WON in a 56-year-old man. **(a)** Axial nonenhanced intraprocedural CT image shows an 18-gauge Chiba needle (arrow) placed within a combined WON (*) in the region of the pancreatic neck and body. **(b)** CT image obtained after removal of the stylet shows a 0.035-inch guide wire (arrow) that was advanced through the needle into the collection. **(c)** CT image shows a 12-F locking looped catheter (arrow) that was placed using modified Seldinger technique after dilating the track using 10- and 12-F fascial dilators. Axial contrast-enhanced CT performed 4 months later revealed complete resolution of the WON.



a.



b.



c.

can be used to treat infected or symptomatic necrosis before the collection becomes WON. It may be the only therapeutic option for patients who are too ill for surgery, or who are not candidates for endoscopy because the collection is too distant from the stomach. PCD can also be performed to treat residual collections after endoscopic or surgical necrosectomy (22).

Percutaneous drainage catheters are placed with a peritoneal or retroperitoneal approach (22,50). The retroperitoneal approach is generally favored because it avoids peritoneal contamination and, depending on the access route, may reduce the risk of bowel injury. CT is preferred to US because nearby bowel and other critical structures can be seen throughout the procedure. CT also allows an immediate intraprocedural postdrainage assessment of the need for additional catheters. Either a tandem trocar or modified Seldinger technique may be used for catheter placement. Catheters needed to drain pancreatic collections typically range from 12 F to 30 F; multiple catheters are often needed during the initial drainage procedure (22,50). At follow-up, additional procedures are often needed to place additional catheters or exchange

indwelling catheters for larger ones during the course of treatment (22). Catheters are usually left to gravity drainage, and frequent irrigations of the cavity are performed to facilitate drainage of nonliquefied material (Fig 23) (55,56). Daily visits on the ward by a dedicated interventional radiology team are essential to the care of patients with pancreatic necrosis.

Although PCD is often used as a temporizing intervention before surgical or endoscopic necrosectomy, randomized controlled trials have shown that PCD alone may be sufficient in some patients. In the PANTER trial (51), authors investigated a step-up approach that involved (a) controlling infected necrosis initially with minimally invasive techniques such as percutaneous drainage, and (b) deferring or possibly avoiding necrosectomy, depending on the patient's clinical course. This management algorithm resulted in improved morbidity rates compared with patients who were managed with open surgical necrosectomy (40% vs 69%). The mortality rates in the two groups were not significantly different (51). In another study, the use of PCD alone proved successful in treating approximately 50% of cases of necrotizing pan-

creatitis; the rate of successful outcomes was not significantly different between cases of sterile (50%) and infected (46%) necrosis (57). The number of catheters used, catheter diameter, and amount of fluid drained did not affect outcome (57).

Disadvantages of PCD include the need for multiple CT scans and procedures and a failure rate of approximately 50%, likely because nonliquefied material may not drain percutaneously (57). Patients with central gland necrosis and pancreatic duct disruption require prolonged drainage and may eventually require endoscopic stent placement or surgical repair of the duct leak (58).

Percutaneous Necrosectomy.—To address the limitations of PCD, percutaneous débridement of necrotic pancreatic collections has been investigated in a limited number of cases (59). With this technique, pancreatic and peripancreatic necrotic tissue is débrided and removed using snares and baskets placed over guide wires advanced via the percutaneously accessed tract. Drainage catheters are then inserted and frequently irrigated. In one study, 20 patients were successfully treated with débridement without the need for surgical or endoscopic necrosectomy (59). However, percutaneous necrosectomy has been associated with an approximately 50% rate of fistula formation to the bowel or main pancreatic duct (54,59).

Endoscopic Methods

Endoscopic necrosectomy and drainage is now an established alternative to surgical necrosectomy. Endoscopic interventions involve the transoral placement of a flexible endoscope, followed by transgastric or transduodenal placement to access the necrotic collection. When present, a transmural bulge can help identify the region of the collection. Endoscopic US, particularly with the aid of color Doppler technique, is increasingly being used to visualize the collection and determine an optimal trajectory that avoids intervening blood vessels. After puncturing the collection with a 19-gauge needle, a guide wire is advanced into the collection and the tract is dilated with hydrostatic balloons 0.5–1.8 cm in diameter and 2 cm in length. Mechanical débridement is then performed using baskets and forceps. The contents are sent for microbiologic analysis, the cavity is copiously irrigated (sometimes with antibiotic solution), and transgastric or transduodenal stents are placed (Fig 24) (22,50). A nasocystic catheter or a percutaneous gastrostomy with jejunal extension can also be used for continuous lavage of the collection (22,50). Endoscopic necrosectomy has proved highly successful in treating symptomatic WON. In a series of 104 patients at six medical centers, suc-

cessful resolution of WON was achieved in 91% of patients; only 4% required surgical necrosectomy (60). In addition, relative to surgical necrosectomy, endoscopic necrosectomy has been shown to result in less postprocedural inflammatory response, less organ failure, and fewer other major complications (61). However, as with PCD, multiple (up to six) débridement sessions may be needed to completely remove necrotic tissue. Complications such as hemorrhage, stent migration, peritonitis, air embolism, and bowel perforation may occur (22). In addition, not all patients are candidates for endoscopic necrosectomy. The collection generally needs to be adjacent to the gastric or duodenal lumen without intervening critical structures such as vessels (22,50). The collection also needs to be walled off because gas insufflation might otherwise spread the infected contents. Endoscopic US is now being used to facilitate necrosectomy; it can be used to visualize the necrotic region and assess the amount of nonliquefied material intraprocedurally (62). Endoscopic US also allows the user to determine the best access route to the collection and avoid vascular structures and bowel (63). Endoscopic necrosectomy and drainage has better success rates and fewer complications when performed with endoscopic US than without endoscopic US (63).

Laparoscopic Necrosectomy

Laparoscopic necrosectomy involves laparoscopic visualization and hand-assisted débridement via a percutaneous port. The necrotic material is more likely to be removed completely and in a single session with laparoscopic necrosectomy than with percutaneous and endoscopic methods (54). Because persistent necrosis is a cause of poor outcomes and continued sepsis, thorough removal of necrotic tissue is important (64). Laparoscopic cholecystectomy can be performed simultaneously in patients with gallstone pancreatitis. Disadvantages of the laparoscopic method include greater invasiveness compared with other minimally interventional methods and a 36% risk of spread of retroperitoneal infection into the peritoneum (50,54). Induction of pneumoperitoneum may also have adverse effects on critically ill patients, including decreased venous return, compression of the lung bases, and decreased renal perfusion (54). For these reasons, other minimally invasive treatments are preferred over laparoscopic necrosectomy (22).

Video-assisted Retroperitoneal Débridement

Video-assisted retroperitoneal débridement involves accessing the necrotic region using a rigid or flexible retroperitoneoscope or accessing the

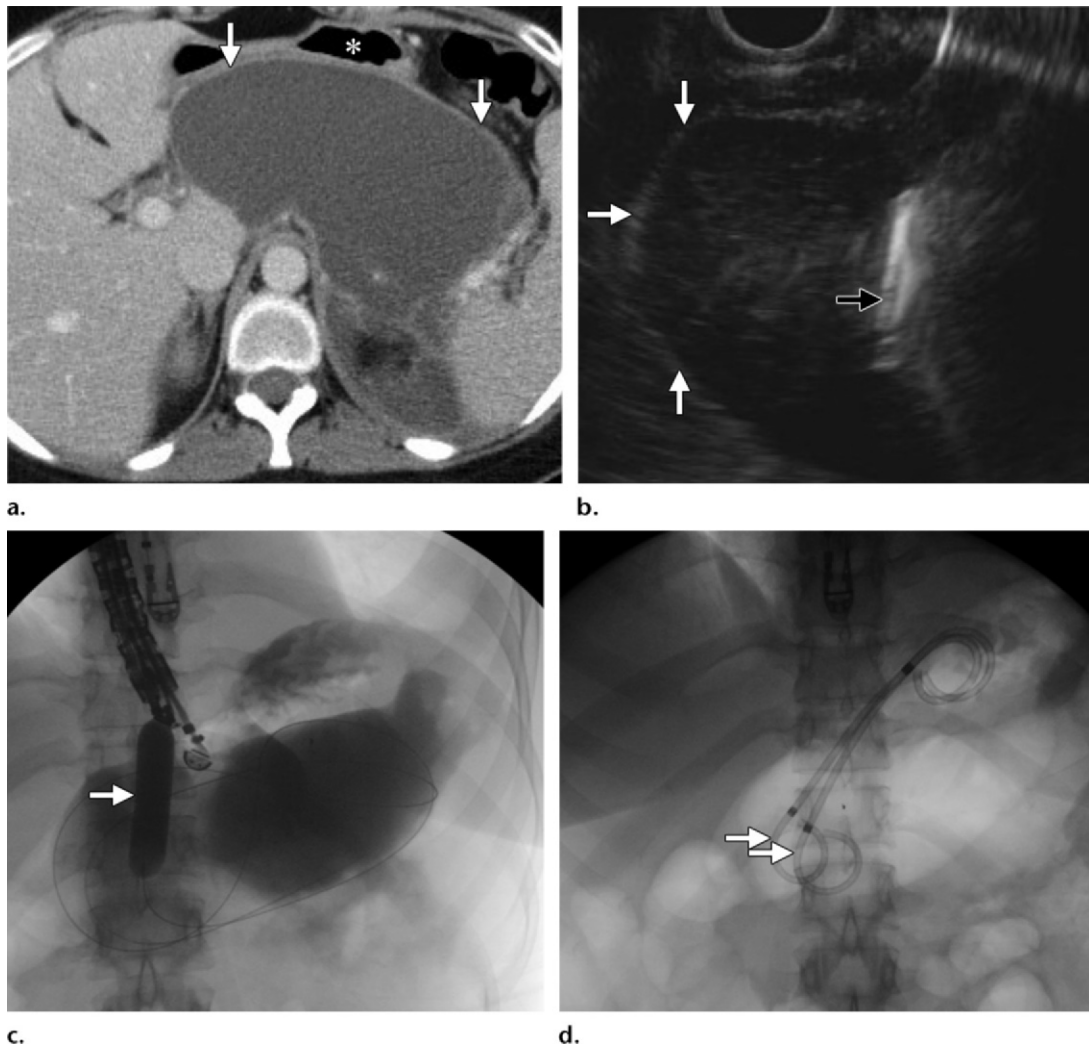


Figure 24. Endoscopic necrosectomy of WON in a 43-year-old woman. **(a)** Axial contrast-enhanced CT image shows combined WON around the body and tail of the pancreas (arrows) abutting and compressing the stomach (*). **(b)** Endoscopic US image shows a 19-gauge needle that was inserted through the stomach (black arrow) to gain access to the WON (white arrows). Purulent fluid was aspirated and sent for culture. **(c)** Fluoroscopic image shows the track serially dilated over the wire using balloons with a diameter of 0.6–1.8 cm (arrow). Fluid and necrotic material were evacuated from the cavity with gastroscopy (which replaced endoscopic US), and the cavity was irrigated with saline solution. **(d)** Fluoroscopic image shows two 5-F double pigtail stents 4 cm in length (arrows) that were placed between the cavity and the stomach.

collection via a tract, typically from a previously placed percutaneous catheter (22,50). Retroperitoneal approaches have been successfully used either to drain a collection after liquefaction has taken place, or to débride necrotic tissue and nonliquefied material. Direct visualization of the collection, débridement using long-grasp forceps, and lavage of the necrotic cavity are performed. Drainage catheters are placed in the retroperitoneum under direct videoscopic guidance. Advantages over laparoscopic methods include avoiding pneumoperitoneum and potentially avoiding infecting the peritoneal cavity; however, other spaces may be at risk for infectious seeding. As with some other techniques, a disadvantage is the

need for multiple interventions to achieve complete drainage (22). Open surgical necrosectomy can be avoided in more than 90% of patients, although there is only a limited amount of evidence comparing outcomes (22,65).

Hybrid Techniques

Necrotizing pancreatitis and its complications often require a multidisciplinary approach. Combinations of surgery, endoscopy, and interventional radiology are often needed. For example, a patient may benefit from PCD of the liquefied component of a collection and endoscopic necrosectomy of the nonliquefied material coupled with internal stent placement between the collection

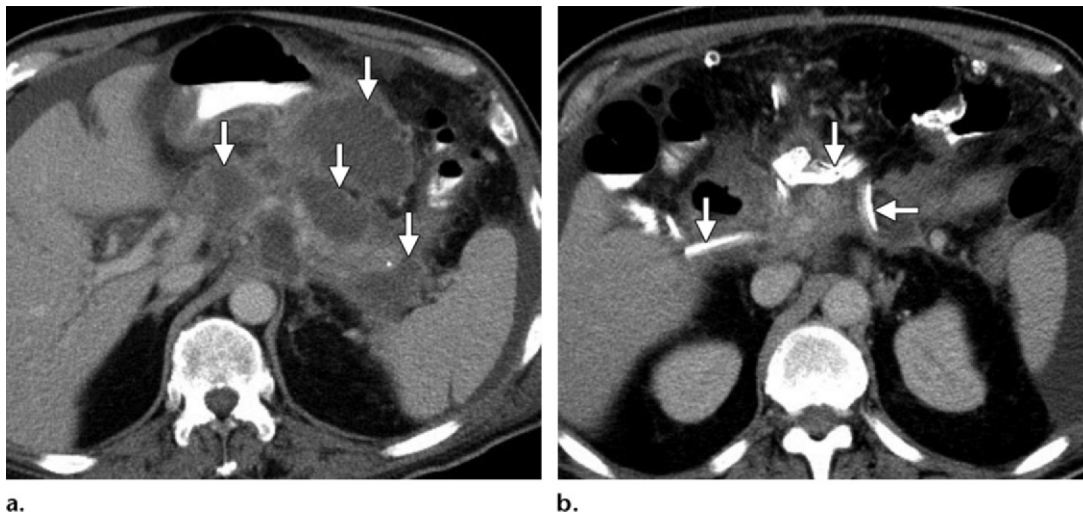


Figure 25. Surgical necrosectomy of WON in a 49-year-old man. **(a)** Axial contrast-enhanced CT image shows multiple pockets of WON in the region of the neck, body, and tail of the pancreas (arrows). Because of the extent of involvement and the presence of multiple pockets, surgical necrosectomy was performed. **(b)** Axial contrast-enhanced CT image obtained 15 days after surgery reveals resolution of the necrotic material, with surgical drainage catheters that were left in the necrosectomy bed (arrows).

and stomach. The endoscopic approach allows internal drainage. However, if a nasocystic catheter is not placed, there is no mechanism for irrigating the cavity to remove the debris, and multiple endoscopic sessions may be required for complete débridement. The addition of PCD allows cavity irrigation and egress of the material via the endoscopic stent. This combined approach has been shown to improve outcomes compared with endoscopic drainage or PCD alone (66).

Open Surgical Necrosectomy

Despite the development of multiple minimally invasive techniques, open necrosectomy remains the standard of reference for the treatment of necrotizing pancreatitis. However, when open necrosectomy is performed in acutely ill patients, high rates of surgical morbidity (34%–95%) and mortality (10%–25%) have been observed, largely owing to increased systemic response and risk of hemorrhage. Therefore, surgical necrosectomy is now typically delayed until the acute phase has passed and the necrosis has become walled off. One study demonstrated decreased rates of morbidity (from 89% to 72%) and mortality (from 10% to 4%) using the delayed approach (22,50,67). Indications for open surgical necrosectomy include infected necrosis with uncontrolled sepsis despite conservative and minimally invasive measures, symptomatic WON that is not accessible via percutaneous or endoscopic routes, symptomatic extensive and multifocal collections, persistent collections after percutaneous or endoscopic intervention, and the presence of other complications such as pseudoaneurysm,

vessel rupture, perforation, obstruction of or fistula to a viscus, and abdominal compartment syndrome (68).

Open surgical necrosectomy is performed by making a midline abdominal incision or retroperitoneal flank incision, followed by the manual removal of necrotic tissue. Necrosectomy involves blunt dissection rather than sharp cutting resection; the latter increases the risk of removing normal tissue, bleeding, and iatrogenic fistula formation with the bowel or pancreatic duct (53). The cavity is irrigated with saline solution and packed with dressings, and multiple drains are left in situ (Fig 25) (53). If necessary, the surgical procedure can be repeated in stages to remove residual necrotic tissue (53). The operation may also include cholecystectomy to prevent recurrence of gallstone-associated pancreatitis, diverting ileostomy if necrosis extends to the retrocolic area, placement of feeding tubes, and treatments for other conditions such as bowel perforation and vascular injury (53).

Advantages of open surgical necrosectomy include the best chance of complete removal of necrotic tissue and the possibility of treating multiple associated complications in a single procedure. Because of its invasiveness, open surgical necrosectomy is typically reserved for patients in whom minimally invasive methods have failed (56). Despite improvements in perioperative care and surgical technique, open surgical necrosectomy is still associated with a number of complications, including incisional hernias, gastrointestinal and pancreatic fistulas, delayed collections, biliary stricture, and exocrine and endocrine insufficiency. One

study reported complications in 62% of patients, with 16% of these patients requiring further surgical or endoscopic intervention (69).

Management of Associated Conditions

Mass Effect on Adjacent Organs

For bowel obstruction secondary to mass effect or inflammation, management includes temporizing measures such as decompressing obstructed bowel by means of enteric tube placement and drainage of fluid collections (Fig 12). Abdominal compartment syndrome resulting from bowel obstruction by large or multiple collections is an indication for emergent laparotomy (33).

Biliary Conditions

In gallstone pancreatitis, the gallbladder may also be removed during laparoscopic or surgical necrosectomy. ERCP has a role in treating choledocholithiasis by means of stone retrieval (29,70). Biliary obstruction can be relieved by decompressing the biliary system with use of endoscopic biliary stent placement or PCD of collections (Fig 15) (71).

Pancreatic Duct Complications

For pancreatic duct leaks, prolonged PCD can be successful if the collection is fully drained, a controlled fistula is achieved, and the fistula and disrupted duct eventually heal. Healing is possible when a short (<2-cm) segment of the pancreatic duct is involved (39). If the pancreatic duct and the fistula do not heal, ERCP stent placement across the pancreatic duct may be attempted. For pancreatic duct leaks in which the collection is in communication with the downstream duct, ERCP stent placement through the disrupted duct into the collection has been reported (38). This procedure may not be possible if the collection communicates with the upstream duct only; in such cases, prolonged PCD or endoscopic transmural stent placement can be attempted. When left in place indefinitely, endoscopic transmural stents have been shown to significantly reduce the recurrence of pancreatic fluid collections compared with stent removal for a disrupted pancreatic duct. Surgery may be needed when minimally invasive therapies fail (35,38).

Treatment of pancreatic ductal stricture without a leak includes ERCP-guided dilation of and stent placement in the strictured segments. Patients with a single stricture at the pancreatic head respond well to stent placement. In patients with multiple strictures along the main pancreatic duct or strictures in the body and tail that are not amenable to endoscopic stent placement, surgery may be required (35).

Vascular Conditions

Management of a pseudoaneurysm includes endovascular methods such as embolization and stent placement, as well as surgical methods such as bypass surgery and ligation (72). Management of hemorrhage depends on the presence of active extravasation, in which either surgical or endovascular therapies may be indicated to halt the bleeding. Anticoagulation therapy may be indicated in cases of portal and superior mesenteric vein thrombosis, particularly if there is concern for hepatic decompensation (73).

Management Algorithm

Although there is no universally adopted treatment algorithm for the management of necrotizing pancreatitis (22), the management algorithm used at our institution is shown in Figure 26.

Conclusion

Acute necrotizing pancreatitis is a common but serious condition that can result in substantial morbidity and mortality. The revised Atlanta classification system introduced new terminology to better characterize the morphologic and imaging features of necrotizing pancreatitis and its complications. Imaging, primarily CT and MR imaging, plays an important role in diagnosing necrotizing pancreatitis, assessing its severity, and identifying complications. Imaging findings are also key determinants in guiding management. Imaging-guided PCD can serve either as the definitive treatment for infected or symptomatic necrosis, or as part of a step-up approach to control infected necrosis, with endoscopic or surgical necrosectomy either deferred or avoided altogether.

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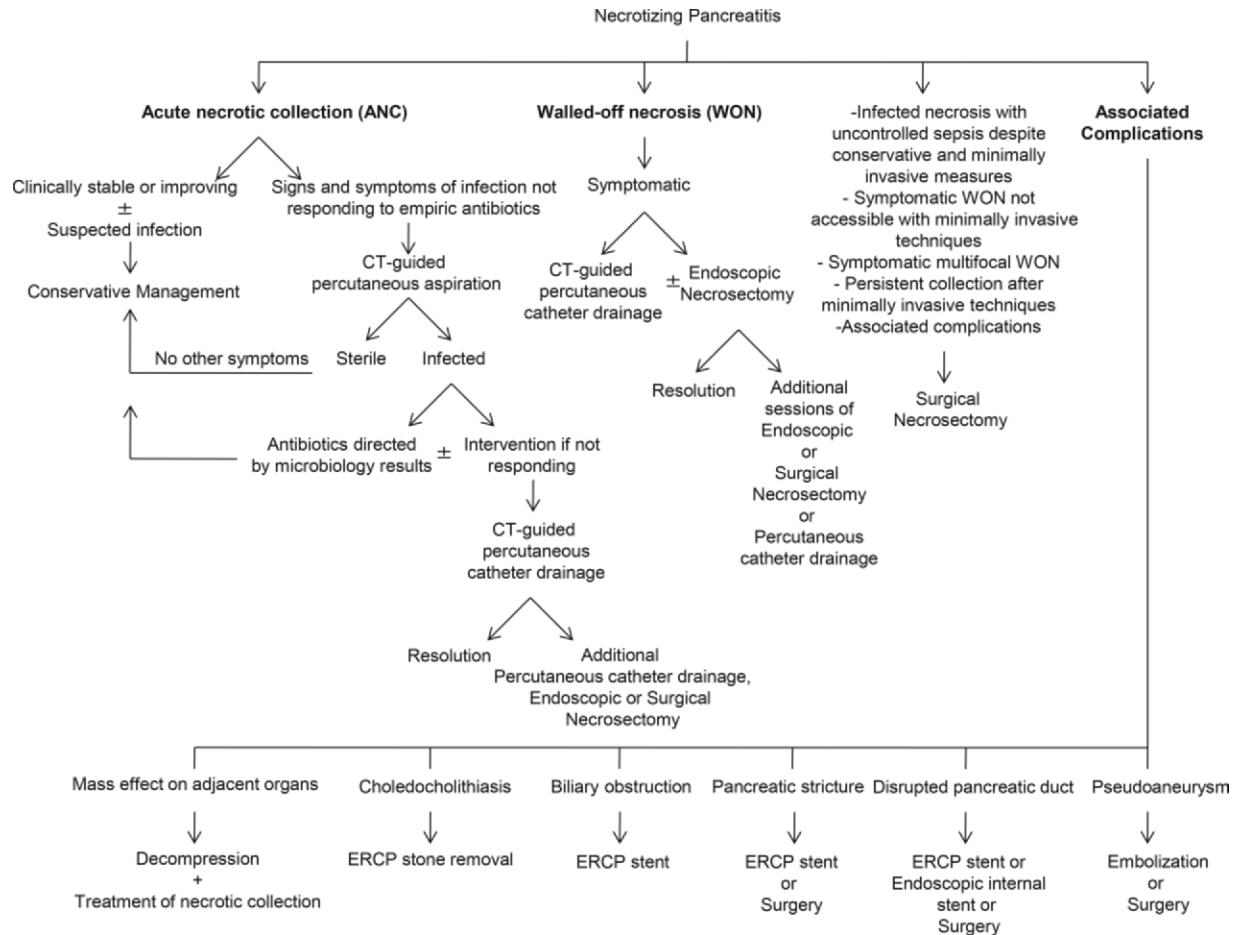


Figure 26. Chart illustrates a management algorithm for treating patients with necrotizing pancreatitis.

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Necrotizing Pancreatitis: Diagnosis, Imaging, and Intervention

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During the early clinical phase, the severity of pancreatitis is determined predominantly by the presence of systemic inflammatory response syndrome and organ failure. The role of imaging is limited during the early phase because early morphologic changes do not correlate with clinical findings or help predict the subsequent clinical course.

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The revised Atlanta classification system subdivides collections associated with necrotizing pancreatitis according to time of disease onset. In the presence of pancreatic necrosis, a collection that develops within 4 weeks of onset and lacks a discrete wall is defined as an acute necrotic collection (ANC). A collection that persists after 4 weeks and develops a discrete wall is defined as walled-off necrosis (WON).

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Terms such as *pancreatic abscess*, *fluid collection*, and *phlegmon* are no longer accepted owing to their ambiguity. A pancreatic abscess virtually never develops in the absence of necrotizing pancreatitis; it is the necrotic tissue that becomes superinfected, so that *infected necrosis* is a more appropriate term. Collections associated with necrotizing pancreatitis may not consist of fluid alone and can arise from necrosis of both pancreatic and peripancreatic tissue. In addition, a precise definition of the collections associated with pancreatitis is important because the treatment varies with the type of collection; for this reason, the nonspecific term *fluid collection* has fallen out of favor.

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Pseudocysts and WON are both late-phase (>4 weeks after onset) collections that develop over time from nonnecrotic (APFC) and necrotic (ANC) collections, respectively. Both pseudocysts and WON have well-defined, nonepithelialized enhancing walls. Pseudocysts contain homogeneous fluid (hypoattenuating at CT, T2 hyperintense at MR imaging) and are only peripancreatic. WON contains necrotic material—often a mixture of fat and fluid—and can involve both pancreatic and peripancreatic tissue. The diagnosis of WON is also favored when a pancreatic collection grows, extends to the paracolic space, and has an irregular border (18). However, any collection that occupies or replaces pancreatic parenchyma is classified as WON, regardless of its appearance. Pseudocysts are more likely to be associated with main pancreatic ductal dilatation (>3 mm), possibly as a result of the compression of pancreatic parenchyma. In patients with WON, ductal dilatation is less likely to occur because the pancreatic fluid simply leaks into the collection.

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All interventions, particularly when performed in acutely ill patients, are associated with high morbidity and mortality rates due to heightened systemic inflammatory response during this time period, as well as an increased risk of hemorrhage. In general, delaying intervention until at least 4 weeks after onset also allows necrotic pancreatic parenchyma to be more clearly distinguished from viable pancreatic parenchyma, thereby preserving viable pancreatic tissue and reducing the risk of pancreatic insufficiency.