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Evidence-based cost-effective management of acute pancreatitis: An algorithm of the Journal of Trauma and Acute Care Surgery emergency general surgery algorithms work group

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(*J Trauma Acute Care Surg.* 2025;98: 850–857. Copyright © 2025 The Author(s). Published by Wolters Kluwer Health, Inc.)

This algorithm was developed by the Journal of Trauma and Acute Care Surgery Emergency General Surgery algorithms working group to provide an evidence-based practical approach to the initial evaluation and management of acute pancreatitis (AP) presenting in the emergency setting. The algorithm is intended to serve as a bedside reference for clinicians. It is annotated with letters linked to corresponding text that provides the rationale and references to support these recommendations. The algorithm is not a substitute for the clinical judgment, and experience of bedside clinicians and should not be considered as the "standard of care." We encourage institutions to use these recommendations to formulate local clinical protocols but recognize that there are patient-specific factors and institutional factors that may require deviation from this algorithm.

Acute pancreatitis is defined as acute inflammation of the pancreas and is a major cause of morbidity and mortality worldwide.[1–4] Acute pancreatitis is a common gastrointestinal disease with 381,741 US emergency department visits reported in 2018. [5] Acute pancreatitis global incidence is 33 to 74 cases per 100,000 person-years, increasing by 2% to 5% per year. From 1990 to 2019, there was a 1.63-fold increase in AP cases globally.[6] Mortality related to AP is 1 to 60 deaths per 100,000 person-years, and optimal medical and surgical management has decreased mortality.[7] The evaluation of patients with possible AP in the emergency setting should be focused on diagnostic confirmation using a systematic approach.

AP DIAGNOSIS AND MANAGEMENT

A. The diagnostic evaluation of a patient with possible AP first requires a thorough history and physical examination (Fig. 1). Common presenting symptoms include constant epigastric or left upper quadrant abdominal pain, which may radiate to the back, sometimes associated with nausea and vomiting. Laboratory evaluation should include serum levels of amylase and lipase, although the diagnostic test accuracy is not high.[8] According to the revised Atlanta classification for the diagnosis of AP, the patient should meet two of the three following criteria: (1) typical abdominal pain consistent with the disease, (2) serum amylase and/or lipase levels greater than three times the upper limit of normal, and/or (3) characteristic findings of AP on abdominal diagnostic imaging (contrast-enhanced computed tomography, magnetic resonance imaging, or abdominal ultrasound) (Table 1).

B. After the diagnosis of AP has been made, the causative etiology of AP should be determined. Gallstone disease, alcohol, and hypertriglyceridemia are the most common etiologies of AP. Biliary disease (gallstone pancreatitis) is the leading cause of AP (50–70%), and alcohol accounts for 25% to 35% of AP cases. Both biliary and alcoholic pancreatitis account for over 80% of all AP cases.[9] Nonbiliary causes of AP include hypertriglyceridemia and other etiologies including hypercalcemia, drug reactions (6-mercaptopurine, azathioprine, and others), procedure associated (post–endoscopic retrograde cholangiopancreatography [ERCP]), structural (tumor or cysts), immune, infectious, and scorpion stings.

Submitted: March 3, 2025, Accepted: March 10, 2025, Published online: April 14, 2025.

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DOI: 10.1097/TA.0000000000004622

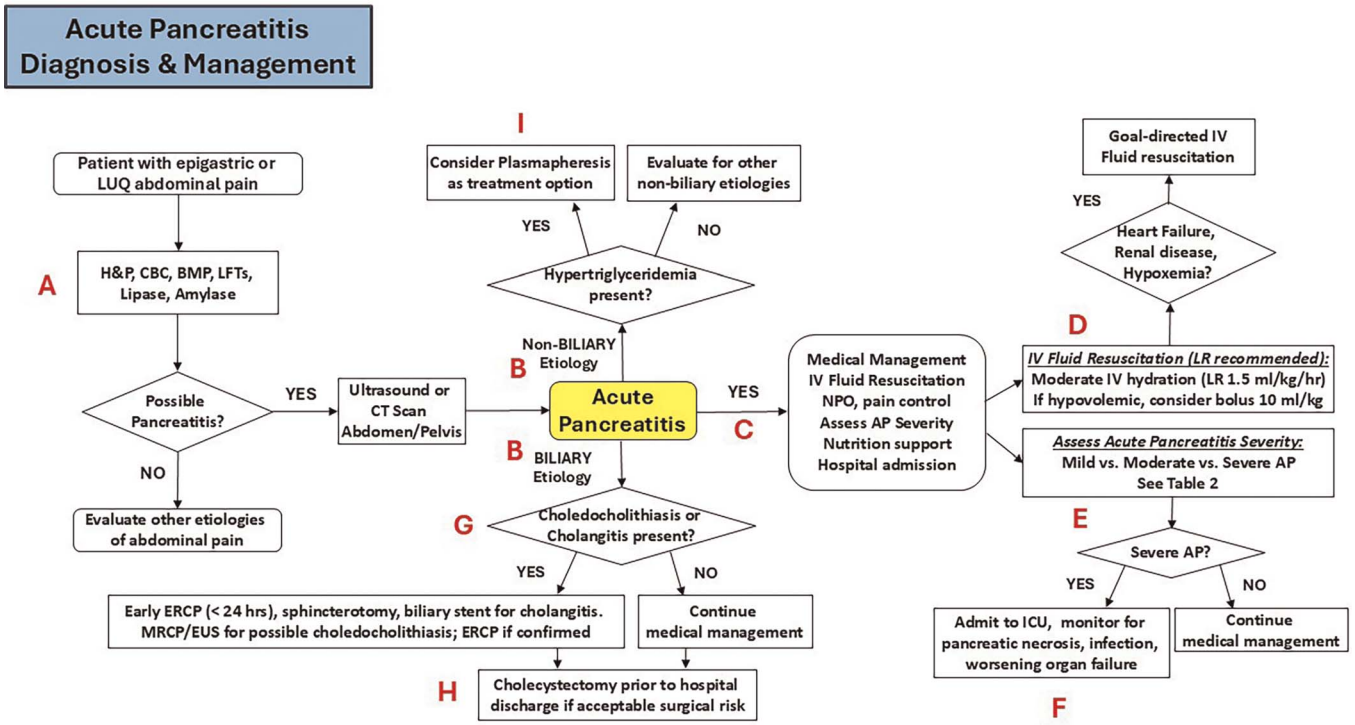

Figure 1. Management algorithm for AP. RUQ, right upper quadrant; H&P, history and physical examination; US, ultrasound; CBC, complete blood count; LFT, liver function test; NPO, nil per os.

A revised classification system is now available to determine which drugs cause AP based on the Grading of Recommendations, Development, and Evaluation criteria.[10] Hypertriglyceridemia-associated AP has increased in some global areas and is an independent risk factor for organ failure and associated with a more severe course of AP.[11–14] There is no definitive threshold of serum triglyceride level that always induces AP. Triglyceride serum levels >1,000 mg/dL at clinical onset of AP are consistent with a definitive diagnosis of hypertriglyceridemia-associated AP, whereas levels <1,000 mg/dL are considered probable, and other potential etiologies of AP should be considered.[15] In about 10% to 15% of cases of AP, the causative etiology may not be definitively established.

The dominant etiology of AP may vary by country. A recent study of over 10,000 patients from China over 15 years reported that cholelithiasis (56.0%), hyperlipidemia (25.3%), and alcohol (6.5%) were the top three etiologies. During this time period, the proportion of biliary AP decreased, while hypertriglyceridemic and alcoholic AP significantly increased and were associated with higher rates of organ failure, necrotizing pancreatitis, and intensive care unit (ICU) admission.[16] Regional epidemiologic studies of AP etiology are important to target AP prevention.

C. After the diagnosis of AP has been made, medical management should be promptly initiated. This includes intravenous (IV) crystalloid resuscitation; nil per os and bowel rest initially; adequate pain and symptom control; treatment of nausea, which is common; and hospital admission. The primary approach to AP is supportive care and largely determined by the severity of the inflammatory process. The severity of AP should also be assessed (Tables 2, 3, and 4). Nutrition support is recommended once volume resuscitation is complete. Enteral nutrition is the preferred route unless it is not tolerated or otherwise contraindicated (bowel obstruction, severe ileus), and then parenteral nutrition is considered. Parenteral nutrition is initiated early in critically ill patients with existing malnutrition and high nutritional risk but is delayed to 5 to 7 days in low nutritional risk patients.[20] The multicenter randomized Pancreatitis, Very Early Compared with Selective Delayed Start of Enteral Feeding (PYTHON) trial found that early enteral tube feeding within 24 hours did not reduce infection or mortality compared with oral feeding.[21] If the patient cannot tolerate oral nutrition, then nasogastric or nasojejunal enteral nutrition is recommended within 72 hours after admission.[22,23] The Acute Pancreatitis Task Force on Quality developed 40 quality indicators to ensure that AP patients receive high-quality care and medical management and have optimal outcomes according to current evidence-based best practices.[24]

TABLE 1. Diagnosis of AP According to Revised Atlanta Classification

Patient should meet 2 of the 3 following criteria:
Typical abdominal pain consistent with the disease
Serum amylase and/or lipase greater than 3 times the upper limit of normal
Characteristic findings of AP on abdominal diagnostic imaging (contrast-enhanced computed tomography, magnetic resonance imaging, or abdominal ultrasound)

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TABLE 2. Severity of AP Definitions: Revised Atlanta Classification

Revised Atlanta Classification of Severity of AP		
Mild AP	**Moderately Severe AP**	**Severe AP**
No local* or systemic** complications	Local or systemic complications	Local or systemic complications
No organ failure	Transient organ failure (up to 48 h)	Persistent single- or multiple-organ failure >48 h – Shock, SBP <90 mm Hg – Pulmonary insufficiency, PaO_2 <60 mm Hg – Acute kidney injury (creatinine >2 mg/dL after rehydration) – Gastrointestinal bleeding >500 mL/24 h – Marshall score ≥2 in 3 organ systems

*Local complications: pancreatic or peripancreatic fluid collections, splenic and portal vein thrombosis, intestinal ischemia and gastric outlet dysfunction.
**Systemic complications: exacerbation of preexisting comorbidity.
Adapted from Ref. [17].
SBP, systolic blood pressure.

D. Intravenous fluid resuscitation should be initiated early with lactated Ringer's solution based on the results of randomized trials, which confirmed improved outcomes (decreased systemic inflammatory response syndrome, C-reactive protein, ICU admission, and hospital length of stay) compared with normal saline.[25–27] A recent meta-analysis reported that patients who received lactated Ringer's solution were less likely to develop moderately severe/severe pancreatitis (odds ratio [OR], 0.49; 95% confidence interval [CI], 0.25–0.97) and were less likely to require ICU admission (OR, 0.33; 95% CI, 0.13–0.81) or to develop local complications (OR, 0.42; 95% CI, 0.20–0.88).[28]

Adequate IV fluid resuscitation to maintain pancreatic perfusion and prevent organ failure is required as early treatment in AP, but prevention of fluid overload and pulmonary complications is also important.[29] The recent Waterfall randomized trial in AP confirmed that moderate IV resuscitation (10 mL/kg bolus if hypovolemia, no bolus if no hypovolemia; 1.5 mL/kg body weight per hour) was as effective as aggressive IV resuscitation (20 mL/kg bolus; 3 mL/kg body weight per hour) with no difference in the incidence of moderately severe or severe pancreatitis.[30] Aggressive IV fluid resuscitation was associated with significantly increased fluid overload (20.5% vs. 6.3%; adjusted relative risk, 2.85; 95% CI, 1.36–5.94; $p = 0.004$). A recent systematic review and meta-analysis of 20 studies reported that aggressive IV fluid resuscitation was associated with significantly higher rates of organ failure ($p = 0.009$), including pulmonary ($p = 0.02$) and renal ($p = 0.01$) complications, but no significant difference in mortality (8.3% vs. 6.0%; $p = 0.3$).[31]

Patients with cardiac disease, renal disease, or hypoxemia may not tolerate moderate IV fluid resuscitation and will be more likely to develop pulmonary complications. These patients require a goal-directed approach with frequent reassessment of vital signs, urine output, point-of-care cardiac/IVC ultrasound, and venous or arterial blood gases.

E. The severity of AP is defined by the revised Atlanta classification (Table 2)[17] and the determinant-based classification (Table 3).[18] About 20% of patients have moderate or severe AP. Severe AP is associated with high mortality rates (20–40%).[32,33] The American Association for the Surgery of Trauma Emergency General Surgery Grading System for AP (Table 4)[19] has undergone initial validation, with increasing American Association for the Surgery of Trauma grades associated with longer hospital and ICU stays and increased readmission rates.[34]

F. Patients with severe AP or AP associated with organ dysfunction should be admitted to the ICU, as they are at higher risk for the development of pancreatic necrosis, infection, and worsening organ dysfunction and/or failure.[35] In addition to IV fluid resuscitation and organ support, these patients may require intubation and mechanical ventilation, renal replacement therapy, and cardiac support with vasopressors and cardiotonic drugs.

G. In patients with biliary etiology of AP, it is important to assess whether choledocholithiasis or cholangitis is present. In patients with AP complicated by cholangitis, early ERCP should be prioritized within the first 24 hours, as it is associated with decreased morbidity and mortality. In patients with suspected choledocholithiasis, Magnetic Resonance Cholangiopancreatography or Endoscopic Ultrasound should be used to confirm the presence of common bile duct stones.

TABLE 3. Severity of AP Definitions: Determinant-Based Classification

Determinant-Based Classification of Severity of AP			
Mild AP	**Moderate AP**	**Moderately Severe AP**	**Severe AP**
No pancreatic or peripancreatic necrosis	Sterile pancreatic or peripancreatic necrosis	Infected pancreatic or peripancreatic necrosis	Infected pancreatic or peripancreatic necrosis
No organ failure	Or transient organ failure (<48 h) or both	Or persistent organ failure (>48 h)	And persistent organ failure (>48 h)

Adapted from Ref. [18].

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TABLE 4. American Association for the Surgery of Trauma Emergency General Surgery Grading System for AP

AAST Grade	Description	Clinical Criteria	Imaging Criteria (CT Findings)	Operative Criteria	Pathologic Criteria
I	Acute edematous pancreatitis	Midepigastric abdominal pain and tenderness; elevated amylase and/or lipase	Pancreatitis without phlegmon, necrosis, peripancreatic fluid collection or abscess	Edematous pancreas	N/A
II	Pancreatic phlegmon or peripancreatic fluid collection or hemorrhage	Midepigastric abdominal pain and tenderness; elevated amylase and/or lipase	Phlegmon or peripancreatic fluid collection or hemorrhage	Pancreatic phlegmon or peripancreatic fluid collection	N/A
III	Sterile pancreatic necrosis	Midepigastric abdominal pain and tenderness; elevated amylase and/or lipase	Pancreatic necrosis without extraluminal air or abscess	Pancreatic necrosis without purulence or abscess	Gram stain and culture of necrosis negative for organisms
IV	Infected pancreatic necrosis or abscess	Severe midepigastric abdominal pain and tenderness; elevated amylase and/or lipase	Pancreatic necrosis without extraluminal air or abscess	Pancreatic necrosis with purulence or abscess	Gram stain and culture of necrosis or abscess positive for organisms
V	Extrapancreatic extension of pancreatic necrosis involving adjacent organs, such as colonic necrosis	Severe midepigastric abdominal pain and tenderness; elevated amylase and/or lipase	Extrapancreatic extension of necrosis involving adjacent organs, such as colonic necrosis	Involvement or necrosis of adjacent organs	Involvement or necrosis of resected adjacent organs

From Ref. [19].
AAST, American Association for the Surgery of Trauma; CT, computed tomography; N/A, not applicable.

The use of diagnostic ERCP to confirm choledocholithiasis should be avoided, as it can precipitate AP. The multicenter randomized APEC (Acute biliary Pancreatitis: urgent ERCP with sphincterotomy versus Conservative treatment) trial reported that routine urgent ERCP with biliary sphincterotomy (<24 hours) did not reduce mortality or severe complications in patients with predicted severe biliary pancreatitis (gallstone pancreatitis without cholangitis) when compared with conservative treatment (38% vs. 44%).[36]

H. Cholecystectomy prior to hospital discharge is recommended for patients with biliary etiology of AP, if the surgical risk is acceptable. Patients with mild gallstone pancreatitis should undergo early cholecystectomy, and some studies have shown that it is safe within the first 24 to 48 hours.[37–41] In moderate/severe AP, it is prudent to await resolution of the acute inflammation and edematous stage of AP to facilitate successful laparoscopic cholecystectomy. Current guidelines recommend delaying surgery for 6 weeks in moderate/severe AP, as early cholecystectomy is associated with higher mortality rates.[42] Serial computed tomography scan imaging (CECT) imaging may demonstrate the degree of peripancreatic and portal inflammation to determine when it is safe to perform cholecystectomy. In necrotizing AP with persistent inflammation and/or peripancreatic fluid collections, cholecystectomy should be deferred until resolution. The Dutch Pancreatitis Study Group recommends cholecystectomy within 8 weeks after hospital discharge in necrotizing biliary pancreatitis in the absence of peripancreatic collections, as it was associated with significantly decreased risk of recurrent pancreatitis.[43]

I. If a definitive diagnosis of hypertriglyceridemia-associated AP is made, initiation of plasmapheresis for treatment should be considered, although the current evidence is not definitive. The American Society for Apheresis guidelines recommended plasmapheresis for severe hypertriglyceridemia-associated AP and to prevent relapse despite low quality evidence.[44] Other International guidelines for AP management do not provide recommendations for plasmapheresis for triglyceride-lowering therapy because of the lack of evidence.[45,46]

Complications associated with interstitial edematous AP include peripancreatic fluid collections and pancreatic pseudocysts (PPCs).[47–49] Pancreatic pseudocysts classification is based on its anatomical location and relationship with the pancreatic duct.[50] More than half of all PPCs will resolve spontaneously, so asymptomatic PPCs do not require intervention. Intervention is recommended for symptoms, infection, bleeding, and increased size. Endoscopic ultrasound–guided transmural drainage is the optimal approach, with surgical cyst-gastrostomy/jejunostomy as an alternative approach.[51] Transpapillary drainage by ERCP is considered if the PPC communicates with the main pancreatic duct. Percutaneous catheter drainage is reserved for infected PPCs and in those not amenable to endoscopic or surgical drainage.[52]

NECROTIZING AND/OR INFECTED PANCREATITIS DIAGNOSIS AND MANAGEMENT

Severe AP can progress to necrotizing and/or infected pancreatitis in some patients (Fig. 2). Intervention for drainage or debridement is preferably delayed until the stage of walled off necrosis (WON).[53] It is recommended that each patient with necrotizing infected pancreatitis is discussed and treated by a multidisciplinary team with experience in both endoscopic and surgical approaches. Recently, a core outcome set for necrotizing AP was developed to standardize data collection to facilitate multicenter research and future high-quality studies with data pooling.[54]

J. The diagnosis of necrotizing or infected pancreatitis is best made with contrast-enhanced (IV and enteral) CECT. If WON collections are confirmed, assessment of whether concurrent infection is present is mandatory. If air is present in

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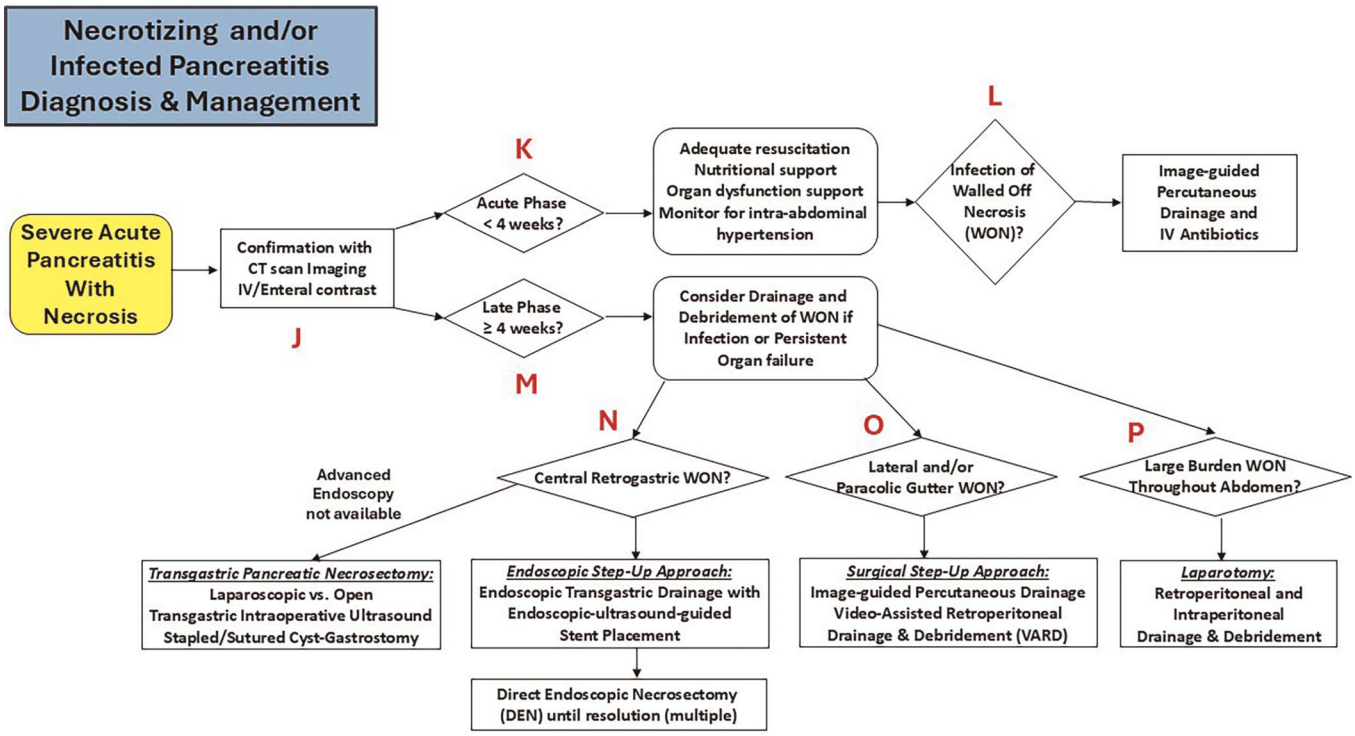

Figure 2. Management algorithm for necrotizing and/or infected AP.

WON (about 50% of patients), then infection is presumed, and IV antibiotics are initiated.[55]

K. If necrotizing pancreatitis is confirmed by CECT in the acute phase (<4 weeks), medical management is continued with adequate resuscitation, nutritional support, organ dysfunction support, and monitoring for intra-abdominal hypertension. Many patients with sterile pancreatic necrosis can be successfully treated conservatively without intervention. Patients with persistent severe pain, gastric outlet obstruction, or persistent organ failure related to WON may require earlier intervention for drainage and/or debridement. However, all attempts should be made to wait until ≥4 weeks after the diagnosis of AP or until diagnostic imaging confirms a well-contained process.[56]

L. If there is evidence of secondary infection of the WON in the acute phase (<4 weeks), either by the presence of air in the WON or clinical signs and symptoms of infection, then empiric IV antibiotics should be initiated, and image-guided percutaneous drainage of the WON should be performed for source control. Fluid should be sent for gram stain and culture for definitive diagnosis of causative pathogens and to provide pathogen-directed appropriate antimicrobial therapy. Fine-needle aspiration of WON can be considered if infection is uncertain (no CECT or clinical signs of infection), but some studies report a 25% false-negative rate.[57] Prognostic factors for the development of infected pancreatic necrosis include older age, gallstone etiology, greater than 50% pancreatic necrosis, multiple or persistent organ failure, invasive mechanical ventilation, and delayed enteral nutrition.[58]

M. If necrotizing pancreatitis is confirmed by CECT in the late phase (≥4 weeks), drainage and debridement of WON are recommended if infection or persistent organ failure is present. Intervention for drainage or debridement is preferably delayed until the stage of WON, as it facilitates safe interventions and lowers the risk of complications. The "step-up approach" is the current standard treatment for necrotizing infected pancreatitis and requires image-guided percutaneous catheter drainage or endoscopic transluminal drainage (Fig. 3), followed by minimally invasive necrosectomy only when the initial step does not fully resolve the WON. The PAncreatitis, Necrosectomy versus sTEp up appRoach (PANTER) randomized trial[60] confirmed that a step-up approach for the management of necrotizing infected pancreatitis compared with open necrosectomy via laparotomy significantly reduced the composite endpoint of major complications and mortality (40% vs. 69%, respectively). Importantly, 35% of patients were successfully treated with simple catheter drainage only. The long-term follow-up of this study confirmed the superiority of the step-up approach (44% vs. 73%), without an increased need for additional invasive interventions.[61] A meta-analysis compared retroperitoneal versus open intraperitoneal necrosectomy in step-up therapy for necrotizing infected pancreatitis (21 studies, 2,177 patients). The retroperitoneal approach was associated with significantly decreased mortality and complications, shorter operative time, shorter hospital stay, higher technical success rates, and no difference in reintervention rates.[62]

The step-up approach can be done both surgically and endoscopically, and these approaches have been compared in randomized trials.[63,64] The multicenter Transluminal Endoscopic Step-Up Approach Versus Minimally Invasive Surgical Step-Up

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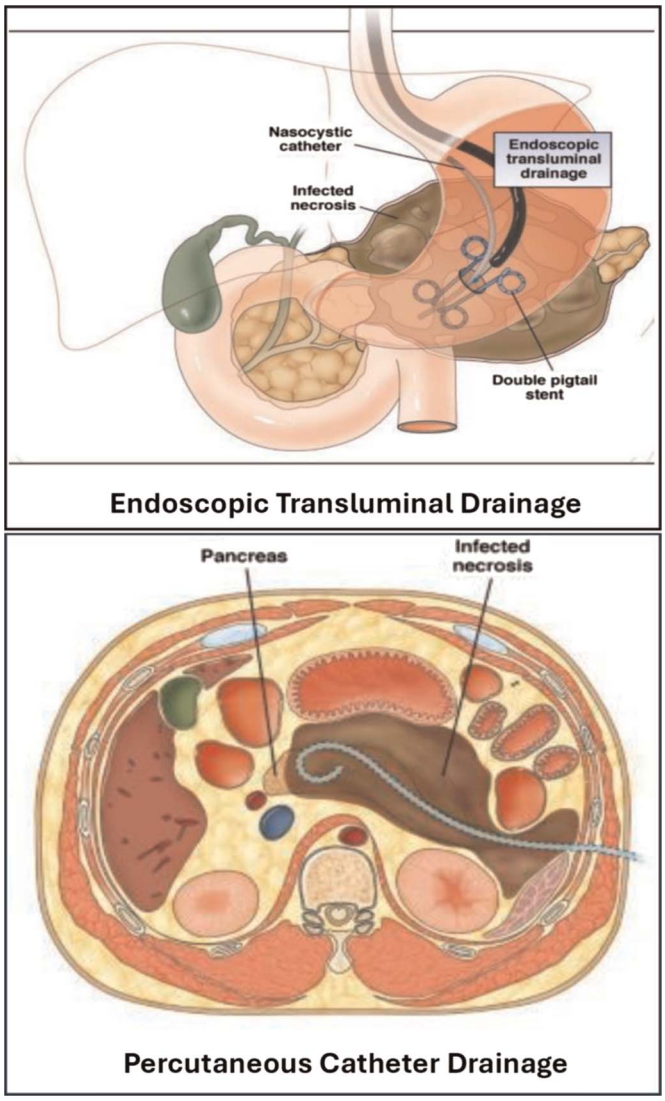

Figure 3. First steps in endoscopic versus surgical step-up approach for necrotizing infected pancreatitis. From Ref. [59].

Approach in Patients with Infected Pancreatic Necrosis (TENSION) trial reported no significant difference in mortality/major morbidity between the endoscopic and surgical step-up approach (43% vs. 45%), but the endoscopic approach was associated with shorter hospitalization and fewer pancreatic fistulas. The long-term (mean, 7 years) follow-up of the TENSION trial reported no difference in mortality/major morbidity but significantly decreased pancreatic fistulas in the endoscopy group (8% vs. 34%) and significantly fewer reinterventions than the surgery group (7% vs. 24%). No difference in pancreatic insufficiency or quality of life was noted between groups.[59]

The single-center Minimally Invasive Surgery vs Endoscopy Randomized (MISER) Trial compared the endoscopic step-up approach with the minimally invasive surgical step-up approach (laparoscopic or video-assisted retroperitoneal debridement) and found no difference in mortality or new-onset organ failure. However, the endoscopic step-up approach had a significantly lower rate of major complications (12% vs. 41%) and pancreatic fistulas (0% vs. 28%). Based on this evidence, the endoscopic step-up approach is now the preferred treatment for necrotizing infected pancreatitis, if possible based on the WON location by CECT imaging.[65]

N. To determine the optimal personalized treatment strategy of necrotizing infected pancreatitis, the careful assessment of the location of the WON by CECT is critical. If WON is present in the central retrogastric region, then the endoscopic step-up approach is recommended, which includes endoscopic transgastric drainage with endoscopic ultrasound–guided stent placement for drainage of the fluid component. Direct endoscopic necrosectomy is then required if necrotic tissue is also present in the WON and commonly requires multiple endoscopic procedures until resolution. Although surgical techniques of transgastric drainage of WON have largely been supplanted by endoscopic approaches, laparoscopic or open transgastric drainage and pancreatic necrosectomy can be performed if there is sufficient local expertise. These surgical approaches can be aided by transgastric intraoperative ultrasound to facilitate creation of a stapled/sutured cystgastrostomy and complete surgical drainage/debridement in one operation.[66–68]

O. If WON collections extend to the flank or pelvic region, the surgical step-up approach is recommended, with image-guided percutaneous drainage (most common in the retroperitoneal left paracolic gutter), followed by video-assisted retroperitoneal debridement along the percutaneous drain tract if complete resolution does not occur with percutaneous drainage alone. Some patients may benefit from combined endoscopic transluminal and percutaneous catheter drainage (dual-modality drainage) for large collections extending into the paracolic gutters or the pelvic region.[69–72] A review of 4,605 necrotizing infected pancreatitis patients (2016–2019) in the United States reported that treatment with drain only (38%) and minimally invasive (32%) approaches significantly increased over time, while open cases declined (47% 2016 to 25% in 2019). Open necrosectomy was associated with higher mortality, respiratory failure, prolonged ventilation, and acute kidney injury.[73]

P. For patients with large burden WON throughout the abdomen, including both retroperitoneal and intraoperitoneal collections not accessible to percutaneous or endoscopic drainage, open laparotomy can be considered but is associated with high morbidity/mortality and should primarily be undertaken as a damage-control procedure in patients with persistent systemic inflammation when less invasive approaches have failed. In some cases, intestinal fistulae may develop related to necrotizing infected pancreatitis (particularly in the colon) and may also require open laparotomy. These can be managed with proximal intestinal diversion. Rarely, severe AP can result in free perforation of the small intestine or colon with development of peritonitis requiring a laparotomy.

Overall, the management of severe pancreatitis with extensive WON requires coordinated care and a well-planned approach that carefully balances the type and extent of invasive procedures against the patient's physiologic state in order to minimize long-

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term morbidity and mortality. In patients with few symptoms and minimal signs of systemic inflammation, invasive interventions should rarely be driven by imaging studies alone.

DISCLOSURE

Conflicts of Interest: Author Disclosure forms have been supplied and are provided as Supplemental Digital Content (http://links.lww.com/TA/E381).

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