

Acute and Chronic Pancreatitis: Clinical Features, Diagnosis, and Management

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Abstract

Acute and chronic pancreatitis are inflammatory disorders of the pancreas, associated with significant morbidity, mortality and healthcare burden. Acute pancreatitis is a common cause of hospital admission, ranging from a mild, self-limited disease to a severe condition complicated by pancreatic necrosis and persistent organ failure. In contrast, chronic pancreatitis is characterized by progressive pancreatic fibrosis, irreversible structural damage, and the eventual development of exocrine and endocrine insufficiency. Despite advances in diagnostic imaging, intensive care management, and minimally invasive therapeutic approaches, pancreatitis remains a substantial diagnostic and therapeutic challenge to clinicians. This review provides a comprehensive overview of the epidemiology, etiology, pathophysiology, clinical presentation, diagnostic strategies, and management of both acute and chronic pancreatitis. Emphasis is placed on disease severity assessment, complication recognition and evidence-based treatment strategies, including updated guidelines and expert-opinion recommendations. A comparative approach is adopted to highlight the main differences and overlaps between acute and chronic pancreatitis, aiming to support early diagnosis, appropriate risk stratification and individualized patient management through a multidisciplinary approach.

Key words: *Acute and chronic pancreatitis; systemic inflammatory response syndrome; pancreatic necrosis; Revised Atlanta Classification and Risk Assessment; Acute Physiology and Chronic Health Evaluation II (APACHE II) score; minimally invasive therapeutic strategies*

INTRODUCTION

Pancreatitis represents a spectrum of inflammatory disorders of the pancreas with variable clinical presentation, treatment response and outcomes. Acute pancreatitis is defined by the sudden onset of pancreatic inflammation, most commonly triggered by gallstones or alcohol consumption, and is characterized by local pancreatic injury and a systemic inflammatory response. While most cases follow a mild and self-limiting course, some cases progress to severe disease, complicated by infected pancreatic necrosis, persistent organ failure, prolonged hospitalization and increased mortality.

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Despite improvements in supportive care and early risk stratification, severe acute pancreatitis remains associated with substantial morbidity and mortality.

In contrast, chronic pancreatitis is a progressive inflammatory condition marked by irreversible destruction of pancreatic parenchyma and fibrosis, along with ductal abnormalities that lead to exocrine pancreatic insufficiency, clinically manifested as pancreatogenic diabetes mellitus and chronic abdominal pain. Alcohol abuse and smoking constitute the primary risk factors, although genetic, autoimmune and idiopathic patterns are increasingly recognized. Recurrent episodes of acute pancreatitis may play a role in disease progression from the acute to the chronic form, indicating that these entities are part of a continuous pathological spectrum. Table 1 summarizes the key clinical and pathophysiological differences between acute and chronic pancreatitis.

Table 1. Key Differences between Acute and Chronic Pancreatitis.

Feature	Acute Pancreatitis	Chronic Pancreatitis
Definition	Acute inflammatory process caused by premature activation of pancreatic enzymes	Progressive inflammatory disorder with irreversible structural damage and fibrosis
Disease course	Sudden onset, usually reversible	Chronic progressive disease
Main etiologies	Gallstones, alcohol, hypertriglyceridemia, drugs, ERCP	Alcohol consumption, smoking, genetic mutations, autoimmune pancreatitis
Pathophysiology	Enzymatic autodigestion and systemic inflammatory response	Chronic inflammation and pancreatic stellate cell activation leading to fibrosis
Structural changes	Edema, necrosis, peripancreatic fluid collections	Pancreatic calcifications, ductal strictures, gland atrophy
Clinical presentation	Acute severe epigastric pain, nausea, vomiting	Chronic abdominal pain, malabsorption, diabetes
Exocrine insufficiency	Rare	Common in advanced disease
Endocrine dysfunction	Rare	Pancreatogenic diabetes mellitus (Type 3c)
Complications	Necrosis, pseudocysts, organ failure	Pseudocysts, biliary obstruction, portal vein thrombosis, pancreatic cancer
Treatment	Supportive care, fluid resuscitation, early nutrition	Lifestyle modification, enzyme replacement, endoscopic or surgical interventions

Clinical suspicion, accurate diagnosis, severity assessment and early intervention are crucial determinants of outcome in both acute and chronic pancreatitis. Advances in cross-sectional imaging, endoscopic techniques, and evidence-based management guidelines have refined clinical practice. However, significant challenges remain regarding optimal risk stratification, complication management and long-term follow-up. The aim of this review is to provide a revised and clinically oriented overview of the underlying pathways of acute and chronic pancreatitis, focusing on clinical features, diagnostic approaches and contemporary management strategies, with particular emphasis on practical decision-making in everyday clinical practice.

Acute Pancreatitis

Etiology and Pathophysiology

Acute pancreatitis is an acute inflammatory disorder of the pancreas resulting from premature activation of pancreatic digestive enzymes within the acinar cells, leading to autodigestion of pancreatic tissue and a variable degree of local and systemic inflammatory response [1,2]. Gallstone disease and excessive alcohol consumption are the most common etiological factors [1,3]. Other recognized causes include hypertriglyceridemia, drug-induced pancreatic injury, post-endoscopic retrograde cholangiopancreatog-

raphy (ERCP) pancreatitis, metabolic disturbances such as hypercalcemia, pancreatic ductal obstruction, genetic mutations, as well as infectious and idiopathic etiologies [1,4].

At the cellular level, acute pancreatitis is initiated by the intrapancreatic activation of trypsinogen, which triggers a cascade of inflammatory signaling pathways and is characterized by the release of cytokines, chemokines, and other vasoactive mediators [2,5]. The ensuing inflammatory response leads to increased vascular permeability, pancreatic interstitial edema, and, in severe cases, parenchymal necrosis and hemorrhage. When the inflammatory process extends beyond the pancreas, systemic inflammatory response syndrome (SIRS) may develop, predisposing patients to multiple organ dysfunction syndrome (MODS) and significantly increasing disease-related mortality [1,6].

Two principal pathogenic mechanisms are recognized: obstruction of the pancreatic duct, most commonly due to gallstones or biliary sludge and, secondly, direct toxic injury to pancreatic acinar cells, especially in alcohol-related disease [1,3]. These mechanisms may coexist, contributing to the heterogeneity observed in clinical presentation and disease severity. Genetic susceptibility, including mutations in PRSS1, SPINK1, CFTR, and CTSC genes, has been increasingly implicated in both idiopathic and recurrent forms of acute

pancreatitis, further highlighting the complex interplay between environmental and host-related factors [4,7].

Classification and Severity Assessment

The clinical and morphological classification of acute pancreatitis has been standardized by the Revised Atlanta Classification (2012) for disease stratification [8]. Based on morphological criteria, acute pancreatitis is categorized into interstitial edematous pancreatitis, characterized by pancreatic enlargement and interstitial edema without necrosis, and necrotizing pancreatitis, which involves pancreatic and/or peripancreatic tissue necrosis [8,9].

Severity is classified into three categories: mild, moderately severe, and severe acute pancreatitis. Most commonly, mild acute pancreatitis is defined by the absence of organ failure and local or systemic complications, typically resolving with conservative management [8]. Moderately severe acute pancreatitis is characterized by transient organ failure lasting less than 48 hours and/or the presence of local or systemic complications. Severe acute pancreatitis is defined by persistent organ failure exceeding 48 hours and is associated with a substantially higher risk of mortality [8,9].

Assessment of disease severity is critical for early risk stratification and appropriate allocation of healthcare resources. Several clinical scoring systems have been developed, including the Ranson criteria and the Acute Physiology and Chronic Health Evaluation II (APACHE II) score [10,11]. Although historically important, the Ranson score has demonstrated limited predictive accuracy in meta-analyses and requires 48 hours for full assessment [10]. In contrast, the APACHE II score allows dynamic, daily evaluation of disease severity, particularly in critically ill patients, and has been widely adopted in intensive care settings [11]. Current guideline recommendations emphasize the integration of clinical parameters, laboratory markers and imaging findings, rather than adopting a single prognostic model to identify patients at risk of severe disease [1,6].

Clinical Presentation and Diagnostic Evaluation of Acute Pancreatitis

The clinical presentation of acute pancreatitis is variable and depends on the underlying etiology, the extent of pancreatic injury and the presence of local or systemic complications. The hallmark symptom is severe, persistent epigastric pain with acute onset, frequently radiating to the back and often accompanied by nau-

sea and vomiting [1,3]. In severe cases, patients may present with signs of systemic inflammatory response, including fever, tachycardia, hypotension and altered mental status, reflecting the development of systemic inflammatory response syndrome (SIRS) and early organ dysfunction [6].

Physical examination findings range from mild epigastric tenderness to an acute abdomen in severe forms. Abdominal distension, decreased bowel sounds due to paralytic ileus, and signs of pleural effusion may be observed. Rare findings, such as Cullen's and Grey Turner's signs, indicate retroperitoneal hemorrhage and are associated with severe disease [1].

Laboratory Evaluation

The diagnosis of acute pancreatitis is established when at least two of the following three criteria are present: characteristic abdominal pain, serum amylase and/or lipase levels elevated to at least three times the upper limit of normal and imaging findings consistent with acute pancreatitis [1,8]. Among biochemical markers, serum lipase is considered more specific and remains elevated for a longer duration compared with amylase [1]. Serum amylase typically rises within six–ten hours of symptom onset and normalizes within three–five days. However, its diagnostic accuracy is limited in alcohol-related pancreatitis and hypertriglyceridemia-associated disease, where false-negative results are common [1,4].

Additional laboratory investigations are essential for etiological assessment and severity stratification. These include complete blood count, hematocrit, C-reactive protein, liver function tests, renal function parameters, serum electrolytes, calcium levels and arterial blood gas analysis in selected cases [6]. Elevated blood urea nitrogen and hematocrit at presentation, as well as failure of their early normalization, have been associated with increased disease severity and worse outcomes [6,11].

Imaging Modalities

Abdominal ultrasonography is recommended as the initial imaging modality in patients with suspected acute pancreatitis, primarily to identify gallstones or biliary dilatation [1]. Its sensitivity for pancreatic inflammation is limited by bowel gas and patient body mass. Contrast-enhanced computed tomography (CT) of the abdomen is not routinely required for the initial diagnosis and should be reserved for patients with diagnostic uncertainty, without clinical improvement within 48–72 hours, or suspicion of local complications [1,6]. When

performed after the early inflammatory phase, contrast-enhanced CT provides valuable information regarding the presence and extent of pancreatic necrosis, peripancreatic collections and vascular complications [9].

Magnetic resonance imaging (MRI) and magnetic resonance cholangiopancreatography (MRCP) offer superior soft tissue contrast and enhanced detection of early pancreatic inflammation and ductal abnormalities, particularly in patients with contraindications to iodinated contrast agents. Secretin-enhanced MRCP may further delineate pancreatic ductal anatomy and functional abnormalities. Endoscopic ultrasound (EUS) plays an important role in the evaluation of idiopathic or recurrent acute pancreatitis, allowing the detection of microlithiasis, small pancreatic tumors and subtle ductal abnormalities, as well as providing the opportunity for tissue sampling when indicated [1,6].

Differential Diagnosis

The differential diagnosis of acute pancreatitis is broad and includes other causes of intra-abdominal and extra-abdominal pathology, such as perforated peptic ulcer disease, acute cholecystitis, biliary colic, mesenteric ischemia, intestinal obstruction, ruptured abdominal aortic aneurysm, acute myocardial infarction and diabetic ketoacidosis. A systematic diagnostic approach integrating clinical, laboratory and imaging findings is therefore essential to establish the diagnosis and provide appropriate management.

Local and Systemic Complications of Acute Pancreatitis

Complications of acute pancreatitis develop in approximately 20% of patients. According to the Revised Atlanta Classification, they are classified as local or systemic, and they are related to the severity and duration of the inflammatory process and contribute significantly to morbidity and mortality [8].

Local complications include acute peripancreatic fluid collections, pancreatic and peripancreatic necrosis, pseudocysts and walled-off necrosis [8,9]. Acute peripancreatic fluid collections typically occur early in the disease course and often resolve spontaneously. In contrast, pancreatic necrosis may become infected, significantly increasing the risk of sepsis and mortality [6,9].

Pancreatic pseudocysts generally develop several weeks after the onset of acute pancreatitis and are characterized by encapsulated collections of pancreatic fluid without solid necrotic material. Walled-off necrosis represents a mature, encapsulated collection containing

both fluid and necrotic tissue and usually develops after four weeks [8]. Additional local complications include pancreatic ascites due to ductal disruption, hemorrhage secondary to vascular erosion or pseudoaneurysm formation, gastrointestinal bleeding, paralytic ileus, and splenic or portal vein thrombosis.

Systemic complications result from the systemic inflammatory response and may involve multiple organ systems. Respiratory complications, including pleural effusions, atelectasis and acute respiratory distress syndrome (ARDS), are among the most frequent and are associated with poor prognosis. Cardiovascular instability, acute kidney injury, metabolic disturbances such as hypocalcemia and hyperglycemia and hematologic abnormalities may also occur. Persistent organ failure lasting more than 48 hours defines severe acute pancreatitis and is strongly associated with increased mortality, particularly in the presence of infected pancreatic necrosis [6,9].

Management of Acute Pancreatitis

The management of acute pancreatitis is primarily supportive and should be tailored according to disease severity, etiology and the presence of complications. Early recognition of patients at risk for severe disease and prompt initiation of appropriate therapy are essential to improve outcomes.

Initial Supportive Care

Early intravenous fluid resuscitation is a cornerstone of management, with recent randomized trials suggesting that moderate fluid resuscitation strategies may reduce complications compared with aggressive hydration [12]. Balanced crystalloid solutions, especially Ringer's lactate, are generally preferred over normal saline due to their favorable effects on acid-base balance and inflammatory response. Fluid administration, particularly within the first 24 hours, should be guided by frequent clinical and laboratory assessment to avoid both hypovolemia or fluid overload [1,6].

Adequate pain control is essential and is typically achieved using opioid analgesics, such as hydromorphone or fentanyl. Oxygen supplementation and respiratory support should be provided as needed, while careful monitoring of vital signs, urine output, hematocrit, and renal function is required to assess response to therapy [1].

Nutritional Support

Early enteral nutrition is recommended once hemodynamic stability is achieved, ideally within 48–72 hours

of admission [13]. Enteral feeding has been shown to preserve gut barrier function, reduce bacterial translocation and decrease the risk of infectious complications compared with parenteral nutrition [14]. Nasogastric feeding is generally safe and effective, with nasojejunal feeding reserved for patients who are unable to tolerate gastric feeding.

Antibiotic Therapy

Routine prophylactic antibiotic administration is not recommended in acute pancreatitis, including cases with sterile pancreatic necrosis. Antibiotics should be reserved for patients with confirmed or strongly suspected infected pancreatic necrosis or for the treatment of extrapancreatic infections [1,6]. When indicated, broad-spectrum antibiotics, such as carbapenems or a combination of a third-generation cephalosporin or fluoroquinolone with metronidazole, are commonly used, guided by microbiological culture results whenever possible.

Endoscopic, Radiologic, and Surgical Interventions

Endoscopic retrograde cholangiopancreatography (ERCP) with sphincterotomy is indicated in patients with gallstone-related acute pancreatitis complicated by acute cholangitis or persistent biliary obstruction [1]. In the absence of these indications, routine early ERCP is not recommended [15].

Management of pancreatic and peripancreatic collections follows a minimally invasive, step-up approach. Percutaneous or endoscopic drainage is preferred as first-line therapy for symptomatic or infected collections, with surgical necrosectomy reserved for patients who fail the less invasive interventions above [6,9]. When feasible, interventions should be delayed for at least four weeks to allow maturation of collections and reduce procedural morbidity [16]. In patients with biliary pancreatitis, cholecystectomy is recommended during the index hospitalization for mild disease or after resolution of inflammation in more severe cases to prevent recurrence.

Chronic Pancreatitis

Definition and Pathophysiology

Chronic pancreatitis is a progressive inflammatory disorder of the pancreas characterized by irreversible structural damage, fibrosis and distortion of the pancreatic ductal system, ultimately leading to loss of exocrine and endocrine function. Chronic pancreatitis, as op-

posed to acute pancreatitis, represents a long-standing process marked by repeated or persistent cellular injury and impaired tissue dys-regeneration [17-20].

At the pathophysiological level, chronic pancreatitis is associated with sustained activation of inflammatory pathways and pancreatic stellate cells, resulting in excessive deposition of extracellular matrix components and progressive fibrosis [18-20]. Over time, cumulative damage leads to acinar cell loss, ductal strictures and eventual islet cell dysfunction, manifesting clinically as malabsorption and pancreatogenic diabetes mellitus [21].

Etiology and Risk Factors

Alcohol abuse remains the predominant etiological factor in chronic pancreatitis, accounting for approximately 70–80% of cases [22,23]. Sustained alcohol consumption has direct toxic effects on pancreatic acinar cells and alters the composition of pancreatic secretions, promoting ductal obstruction. Cigarette smoking has emerged as an independent disease modifier, accelerating progression and increasing the risk of complications [23].

Non-alcoholic causes include genetic disorders, such as mutations in PRSS1, SPINK1, CFTR, and CTRC genes, which are associated with early-onset disease [24]. Auto-immune pancreatitis, a distinct immune-mediated entity often associated with elevated IgG4 levels, represents a potentially reversible cause when recognized earlier [25]. Additional etiologies include anatomic abnormalities, such as pancreas divisum, cystic fibrosis, tropical pancreatitis and idiopathic forms, which account for approximately 10–20% of cases [26-29].

Clinical Manifestations

The clinical presentation of chronic pancreatitis is heterogeneous and non-specific. Chronic or recurrent epigastric pain, often radiating to the back, is the most prominent symptom. Pain severity does not consistently correlate with the extent of structural damage and may diminish in advanced disease due to parenchymal loss and pancreatic burnout [29].

As the disease progresses, destruction of pancreatic parenchyma leads to exocrine pancreatic insufficiency, typically manifesting after loss of more than 90% of exocrine function [30]. Clinical features include steatorrhea, weight loss, fat-soluble vitamin deficiencies and malnutrition [31,32]. Endocrine insufficiency develops later in the disease course and results in pancreatogenic diabetes mellitus, characterized by hard-to-manage

glycemic control and an increased risk of hypoglycemia due to impaired glucagon secretion [33].

Diagnostic Evaluation of Chronic Pancreatitis

The diagnosis of chronic pancreatitis is often challenging, particularly in the early stages of the disease, when structural abnormalities may not be evident and laboratory findings are non-specific [19]. A comprehensive diagnostic strategy incorporates clinical history, laboratory evaluation and imaging modalities, with the selection of investigations determined by disease stage and clinical presentation [34].

Laboratory Assessment

Routine laboratory tests are of limited diagnostic value in chronic pancreatitis. Serum amylase and lipase levels are frequently normal or only mildly elevated, reflecting progressive pancreatic atrophy rather than active inflammation [20]. Evaluation of exocrine pancreatic function is therefore essential in patients with suspected advanced disease. Measurement of fecal elastase-1 is widely used as a non-invasive screening test for exocrine pancreatic insufficiency [35]. Additional tests, such as fecal chymotrypsin measurement or quantitative fecal fat analysis, may provide supportive evidence of malabsorption.

Direct pancreatic functional tests, involving the collection of pancreatic secretions after secretin or cholecystokinin stimulation, represent the reference standard for assessing exocrine function. However, their invasive nature and limited availability restrict their use in daily clinical practice [36]. Indirect tests, such as fecal elastase measurement, are more commonly undertaken due to their simplicity and acceptable diagnostic performance in advanced disease.

Assessment of endocrine function should include fasting plasma glucose, glycated hemoglobin, or oral glucose tolerance testing to detect pancreatogenic diabetes mellitus [33]. Nutritional evaluation, including serum levels of fat-soluble vitamins, albumin, and prealbumin, may be useful in identifying malnutrition and guiding supportive therapy.

Imaging Modalities

Cross-sectional imaging plays a central role in the diagnosis of chronic pancreatitis. Contrast-enhanced computed tomography (CT) is often the initial imaging modality and may demonstrate pancreatic calcifications, glandular atrophy, ductal dilation, and pseudocyst formation, particularly in advanced disease. However,

CT has limited sensitivity for early-stage chronic pancreatitis [37,38].

Magnetic resonance imaging (MRI) combined with magnetic resonance cholangiopancreatography (MRCP) provides superior visualization of the pancreatic parenchyma and ductal anatomy. MRCP allows detailed assessment of pancreatic ductal irregularities, strictures, and side-branch abnormalities. Secretin-enhanced MRCP further improves diagnostic accuracy by evaluating ductal compliance and exocrine function [37,38].

Endoscopic ultrasound (EUS) is considered the most sensitive modality for the detection of early chronic pancreatitis. EUS enables high-resolution imaging of the pancreatic parenchyma, the ductal system and early fibrosis [39]. In some controversial cases, EUS-guided fine-needle biopsy may be performed to confirm autoimmune pancreatitis or malignancy.

Local and Systemic Complications of Chronic Pancreatitis

Chronic pancreatitis is associated with a wide spectrum of local and systemic complications that impact patients' quality of life. Their incidence increases with disease duration, and they are consequences of progressive pancreatic fibrosis, ductal obstruction and chronic inflammation [20].

Pancreatic pseudocysts are the most common local complications, and they develop because of ductal disruption or persistent inflammation. Large or complicated collections may cause abdominal pain, gastric outlet obstruction, infection, or hemorrhage [39]. Obstruction of the intrapancreatic portion of the common bile duct due to inflammatory mass formation or fibrosis can result in obstructive jaundice and cholangitis [40].

Vascular complications include thrombosis of the splenic or portal vein, which may lead to segmental portal hypertension and the development of gastric or esophageal varices, predisposing patients to gastrointestinal bleeding [39]. Pancreatic ascites and pleural effusions may occur following ductal leakage, whereas duodenal or colonic obstruction may result from inflammatory strictures or extrinsic compression.

A particularly important consideration is the increased risk of pancreatic ductal adenocarcinoma in patients with long-standing chronic pancreatitis. Differentiating inflammatory pancreatic masses from malignancy remains a diagnostic challenge and necessitates careful surveillance, especially in patients with new-onset symptoms or unexplained clinical deterioration [41].

Systemic complications primarily reflect exocrine pancreatic insufficiency, which leads to maldigestion, weight loss, fat-soluble vitamin deficiencies, malnutrition and sarcopenia. Endocrine insufficiency manifests as pancreatogenic diabetes mellitus, characterized by impaired insulin and glucagon secretion and an increased risk of hypoglycemia (type 3 diabetes) [23,43]. Osteoporosis and osteopenia may develop secondary to malabsorption and vitamin D deficiency, further contributing to long-term morbidity [32].

Management Strategies for Chronic Pancreatitis

The management of chronic pancreatitis requires a multidisciplinary approach aimed at symptom control, prevention of complications and preservation of nutritional and metabolic status [34]. Treatment strategies should be individualized according to disease severity, anatomical findings, and the patient's quality of life.

Lifestyle Modification

Abstinence from alcohol and smoking remains fundamental components of management and are strongly associated with improved symptom control and slower disease progression [22,23]. Nutritional counseling is essential, with emphasis on adequate caloric intake and supplementation of fat-soluble vitamins [42].

Pain Management

Pain control follows a stepwise approach, beginning with non-opioid analgesics and progressing to opioid therapy when necessary. Adjunctive therapies, including antidepressants and neuromodulators, may be beneficial in selected patients. In cases of refractory pain, celiac plexus block or neurolysis may be considered after exclusion of local complications [29].

Management of Pancreatic Insufficiency

Pancreatic enzyme replacement therapy for pancreatic insufficiency should be administered with meals to optimize digestion and nutrient absorption, thereby improving steatorrhea and nutritional status. Endocrine insufficiency is managed with insulin therapy, with careful monitoring due to the increased risk of hypoglycemia in pancreatogenic diabetes [23,43].

Invasive Interventions

Endoscopic therapy plays an important role in selected patients, particularly those with dominant pancreatic duct strictures, intraductal stones or symp-

tomatic pseudocysts. Endoscopic retrograde cholangiopancreatography (ERCP) with ductal stenting or stone extraction may provide symptom relief and delay the need for surgery [43].

Surgical intervention is reserved for patients with refractory pain, complications unresponsive to endoscopic treatment or suspicion of malignancy. Surgical options include decompressive procedures, pancreatic resection, or combined approaches [44,45]. Early referral to specialized centers is recommended to optimize outcomes.

Current Guidelines and Future Perspectives

Over the past decades, the management of both acute and chronic pancreatitis has evolved substantially, guided by evidence-based recommendations issued by international scientific societies. Contemporary guidelines emphasize early risk stratification, judicious use of diagnostic imaging and a shift toward minimally invasive therapeutic strategies, along with balanced crystalloid resuscitation, and avoidance of prophylactic antibiotics in sterile necrosis [1,6].

In acute pancreatitis, current recommendations support early fluid resuscitation with balanced crystalloid solutions, avoidance of prophylactic antibiotics, and early initiation of enteral nutrition. The adoption of a step-up approach for the management of pancreatic necrosis, favoring endoscopic interventions over early surgical necrosectomy, has resulted in reduced morbidity and improved outcomes [43]. Ongoing research focuses on refining prognostic tools, identifying reliable biomarkers for early severity prediction and optimizing individualized treatment pathways.

In chronic pancreatitis, guidelines highlight the importance of etiological classification, lifestyle modification and a multidisciplinary approach to pain management and nutritional support. Advances in endoscopic techniques and surgical strategies have expanded therapeutic options, while emerging insights into the molecular mechanisms of pancreatic fibrosis and inflammation offer potential targets for disease-modifying therapies. Future research is expected to further clarify the role of early intervention in altering disease progression and improving long-term quality of life.

CONCLUSIONS

Acute and chronic pancreatitis represent heterogeneous pancreatic disorders with significant clinical and

healthcare impact. Accurate diagnosis, early assessment of disease severity and timely implementation of evidence-based management are crucial for patient outcomes. Research in diagnostic imaging, supportive care and minimally invasive interventions has improved the prognosis of severe acute pancreatitis. In chronic pancreatitis, prevention of complications, effective symptom control and management of pancreatic insufficiency remain central therapeutic goals. Clinicians' adherence to international guidelines, combined with a personalized approach, is crucial for optimizing patient treatment.

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REFERENCES

- Boxhoorn L, Voermans RP, Bouwense SA, Bruno MJ, Verdonk RC, Boermeester MA, et al. Acute pancreatitis. *Lancet*. 2020;396(10252):726–34.
- Trikudanathan G, Wolbrink DRJ, van Santvoort HC, Mallory S, Freeman M, Besselink MG. Current concepts in severe acute and necrotizing pancreatitis: An Evidence-Based Approach. *Gastroenterology*. 2019;156(7):1994–2007.e3.
- Tenner S, Baillie J, DeWitt J, Vege SS. American College of Gastroenterology guideline: management of acute pancreatitis. *Am J Gastroenterol*. 2013;108(9):1400–15.
- Nawaz H, Mounzer R, Yadav D, Yabes JG, Slivka A, Whitcomb DC, et al. Revised Atlanta and determinant-based classification: application in a prospective cohort of acute pancreatitis patients. *Am J Gastroenterol*. 2013;108(12):1911–7.
- Bollen TL, Singh VK, Maurer R, Repas K, van Es HW, Banks PA, et al. A comparative evaluation of radiologic and clinical scoring systems in the early prediction of severity in acute pancreatitis. *Am J Gastroenterol*. 2012;107(4):612–9.
- Working Group IAP/APA Acute Pancreatitis Guidelines. IAP/APA evidence-based guidelines for the management of acute pancreatitis. *Pancreatol*. 2013;13(4 Suppl 2):e1–15.
- Whitcomb DC. Genetic risk factors for pancreatic disorders. *Gastroenterology*. 2013;144(6):1292–302.
- Banks PA, Bollen TL, Dervenis C, Gooszen HG, Johnson CD, Sarr MG, et al. Classification of acute pancreatitis—2012: revision of the Atlanta classification and definitions by international consensus. *Gut*. 2013;62(1):102–11.
- Van Santvoort HC, Besselink MG, Bakker OJ, Hofker HS, Boermeester MA, Dejong CHC, et al. A step-up approach or open necrosectomy for necrotizing pancreatitis. *N Engl J Med*. 2010;362(16):1491–502.
- De Bernardinis M, Violi V, Roncoroni L, Boselli AS, Giunta A, Peracchia A. Discriminant power and information content of Ranson's prognostic signs in acute pancreatitis: a meta-analytic study. *Crit Care Med*. 1999;27(10):2272–83.
- Knaus WA, Draper EA, Wagner DP, Zimmerman JE. APACHE II: a severity of disease classification system. *Crit Care Med*. 1985;13(10):818–29.
- de-Madaria E, Buxbaum JL, Maisonneuve P, García de Paredes AG, Zapater P, Guilabert L, et al. Aggressive or moderate fluid resuscitation in acute pancreatitis. *N Engl J Med*. 2022;387(11):989–1000.
- Yao Q, Liu P, Peng S, Xu X, Wu Y. Effects of immediate or early oral feeding on acute pancreatitis: a systematic review and meta-analysis. *Pancreatol*. 2022;22(2):175–84.
- Kumar A, Singh N, Prakash S, Saraya A, Joshi YK. Early enteral nutrition in severe acute pancreatitis: a prospective randomized controlled trial. *J Clin Gastroenterol*. 2006;40(5):431–4.
- Schepers NJ, Hallensleben NDL, Besselink MG, Anten MP, Bollen TL, da Costa DW, et al. Urgent endoscopic retrograde cholangiopancreatography with sphincterotomy versus conservative treatment in predicted severe acute gallstone pancreatitis (APEC): a multicentre randomised controlled trial. *Lancet*. 2020;396(10245):167–76.
- Boxhoorn L, van Dijk SM, van Grinsven J, van Grinsven J, Verdonk RC, Boermeester MA, et al. Immediate versus postponed intervention for infected necrotizing pancreatitis. *N Engl J Med*. 2021;385(15):1372–81.
- Stevens T, Conwell DL, Zuccaro G Jr. Pathogenesis of chronic pancreatitis: an evidence-based review of past theories and recent developments. *Am J Gastroenterol*. 2004;99(11):2256–70.
- Whitcomb DC, Frulloni L, Garg P, Greer JB, Schneider A, Yadav D, et al. Chronic pancreatitis: an international draft consensus proposal for a new mechanistic definition. *Pancreatol*. 2016;16(2):218–24.
- Majumder S, Chari ST. Chronic pancreatitis. *Lancet*. 2016;387(10031):1957–66.
- Vege SS, Chari ST. Chronic pancreatitis. *N Engl J Med*. 2022;386(9):869–78.
- Hart PA, Bellin MD, Andersen DK, Bradley D, Cruz-Monserrate Z, Forsmark C, et al. Type 3c (pancreatogenic) diabetes mellitus secondary to chronic pancreatitis and pancreatic cancer. *Lancet Gastroenterol Hepatol*. 2016;1(3):226–37.
- Lugea A, Gerloff A, Su HY, Xu Z, Go A, Hu C, et al. The combination of alcohol and cigarette smoke induces endoplasmic reticulum stress and cell death in pancreatic acinar cells. *Gastroenterology*. 2017;153(6):1674–86.
- Coté GA, Yadav D, Slivka A, Hawes RH, Anderson MA, Burton FR, et al. Alcohol and smoking as risk factors in an epidemiology study of patients with chronic pancreatitis. *Clin Gastroenterol Hepatol*. 2011;9(3):266–73.

24. Mounzer R, Whitcomb DC. Genetics of acute and chronic pancreatitis. *Curr Opin Gastroenterol*. 2013;29(5):544–51.
25. Okazaki K, Uchida K. Current Concept of Autoimmune Pancreatitis and IgG4-related Disease. *Am J Gastroenterol*. 2018;113(10):1412–6.
26. Romagnuolo J, Talluri J, Kennard E, Sandhu BS, Sherman S, Cote GA, et al. Clinical profile, etiology, and treatment of chronic pancreatitis in North American women: analysis of a large multicenter cohort. *Pancreas*. 2016;45(7):934–40.
27. Wilcox CM, Sandhu BS, Singh V, Gelrud A, Abberbock J, Sherman S, et al. Racial Differences in the Clinical Profile, Causes, and Outcome of Chronic Pancreatitis. *Am J Gastroenterol*. 2016;111(10):1488–96.
28. Yadav D, Lowenfels AB. The epidemiology of pancreatitis and pancreatic cancer. *Gastroenterology*. 2013;144(6):1252–61.
29. Drewes AM, Bouwense SAW, Campbell CM, Ceyhan GO, Delhaye M, Ekin Demir I, et al. Guidelines for the understanding and management of pain in chronic pancreatitis. *Pancreatol*. 2017;17(5):720–31.
30. Sankaran SJ, Xiao AY, Wu LM, Windsor JA, Forsmark CE, Petrov MS. Frequency of progression from acute to chronic pancreatitis and risk factors: a meta-analysis. *Gastroenterology*. 2015;149(6):1490–500e1.
31. Whitcomb DC, Buchner AM, Forsmark CE. AGA Clinical Practice Update on the Epidemiology, Evaluation, and Management of Exocrine Pancreatic Insufficiency: Expert Review. *Gastroenterology*. 2023;165(5):1292–301.
32. Duggan SN, Smyth ND, Murphy A, Macnaughton D, O’Keefe SJD, Conlon KC. High prevalence of osteoporosis in patients with chronic pancreatitis: a systematic review and meta-analysis. *Clin Gastroenterol Hepatol*. 2014;12(2):219–28.
33. Mohapatra S, Majumder S, Smyrk TC, Zhang L, Matveyenko A, Kudva YC, et al. Diabetes Mellitus Is Associated With an Exocrine Pancreatopathy: Conclusions From a Review of Literature. *Pancreas*. 2016;45(8):1104–10.
34. Gardner TB, Adler DG, Forsmark CE, Sauer BG, Taylor JR, Whitcomb DC. ACG Clinical Guideline: Chronic pancreatitis. *Am J Gastroenterol*. 2020;115(3):322–39.
35. Vanga RR, Tansel A, Sidiq S, El-Serag HB, Othman MO. Diagnostic Performance of Measurement of Fecal Elastase-1 in Detection of Exocrine Pancreatic Insufficiency: Systematic Review and Meta-analysis. *Clin Gastroenterol Hepatol*. 2018;16(8):1220–8e4.
36. Kothari D, Ketwaroo G, Sawhney MS, Freedman SD, Sheth SG. Comparison of Combined Endoscopic Ultrasonography and Endoscopic Secretin Testing With the Traditional Secretin Pancreatic Function Test in Patients With Suspected Chronic Pancreatitis: A Prospective Crossover Study. *Pancreas*. 2017;46(6):770–5.
37. Frøkjær JB, Akisik F, Farooq A, Akpinar B, Dasyam A, Drewes AM, et al. Guidelines for the Diagnostic Cross Sectional Imaging and Severity Scoring of Chronic Pancreatitis. *Pancreatol*. 2018;18(7):764–73.
38. Issa Y, Kempeneers MA, van Santvoort HC, Bollen TL, Bipat S, Boermeester MA. Diagnostic performance of imaging modalities in chronic pancreatitis: a systematic review and meta-analysis. *Eur Radiol*. 2017;27(9):3820–44.
39. Mutignani M, Dokas S, Tringali A, Forti E, Pugliese F, Cintolo M, et al. Pancreatic leaks and fistulae: An Endoscopy-Oriented Classification. *Dig Dis Sci*. 2017;62(10):2648–57.
40. Hammel P, Couvelard A, O’Toole D, Ratouis A, Sauvanet A, Fléjou JF, et al. Regression of liver fibrosis after biliary drainage in patients with chronic pancreatitis and stenosis of the common bile duct. *N Engl J Med*. 2001;344(6):418–23.
41. Xiao AY, Tan ML, Wu LM, Asrani VM, Windsor JA, Yadav D, et al. Global incidence and mortality of pancreatic diseases: a systematic review, meta-analysis, and meta-regression of population-based cohort studies. *Lancet Gastroenterol Hepatol*. 2016;1(1):45–55.
42. Martínez-Moneo E, Stigliano S, Hedström A, Kaczka A, Malvik M, Waldthaler A, et al. Deficiency of fat-soluble vitamins in chronic pancreatitis: a systematic review and meta-analysis. *Pancreatol*. 2016;16(6):988–94.
43. Cahen DL, Gouma DJ, Nio Y, Rauws EAJ, Boermeester MA, Busch OR, et al. Endoscopic versus surgical drainage of the pancreatic duct in chronic pancreatitis. *N Engl J Med*. 2007;356(7):676–84.
44. Issa Y, Kempeneers MA, Bruno MJ, Fockens P, Poley JW, Ahmed Ali U, et al. Effect of early surgery vs endoscopy-first approach on pain in patients with chronic pancreatitis: the ESCAPE randomized clinical trial. *JAMA*. 2020;323(3):237–47.
45. Zhao X, Cui N, Wang X, Cui Y. Surgical strategies in the treatment of chronic pancreatitis: An updated systematic review and meta-analysis of randomized controlled trials. *Medicine (Baltimore)*. 2017;96(9):e6220.

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