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ADVANCES IN THE CLINICAL MANAGEMENT OF ACUTE PANCREATITIS: CURRENT EVIDENCE, DIAGNOSTIC APPROACHES, THERAPEUTIC STRATEGIES, CRITICAL CARE MANAGEMENT, AND EMERGING EVIDENCE-BASED INTERVENTIONS

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ABSTRACT

Background: Acute pancreatitis is one of the most common gastrointestinal emergencies and is associated with substantial morbidity, mortality, prolonged hospitalization, and healthcare expenditures worldwide. Although most patients experience a mild and self-limiting course, approximately 20% develop severe disease characterized by pancreatic necrosis, persistent organ failure, and life-threatening systemic complications. Recent advances in evidence-based management have shifted the focus toward early risk stratification, goal-directed supportive care, optimized nutritional strategies, and selective use of invasive interventions. **Aim:** This review aimed to summarize contemporary evidence regarding the assessment and evidence-based management of acute pancreatitis, with emphasis on severity prediction, fluid resuscitation, pharmacologic and nonpharmacologic therapies, nutritional support, management of underlying etiologies, and prevention of disease recurrence. **Methods:** A comprehensive narrative review of current international guidelines randomized controlled trials, systematic reviews, meta-analyses, and observational studies was conducted. Evidence addressing prognostic assessment, intravenous fluid therapy, antibiotic use, emerging pharmacologic agents, nutritional management, biliary interventions, and recurrence prevention was critically synthesized. **Results:** Simple laboratory markers, particularly blood urea nitrogen, hematocrit, and serum creatinine, demonstrated prognostic performance comparable to complex scoring systems. Early goal-directed fluid resuscitation with Lactated Ringer's solution significantly improved clinical outcomes and reduced complications. Routine prophylactic antibiotics and targeted pharmacologic therapies showed no consistent clinical benefit. **Conclusion:** Contemporary management of acute pancreatitis relies primarily on early supportive care, individualized risk assessment, timely enteral nutrition, and treatment of the underlying cause. Evidence-based implementation of these strategies improves patient outcomes while reducing complications, mortality, and disease recurrence.

KEYWORDS: Acute pancreatitis; fluid resuscitation; enteral nutrition; pancreatic necrosis; antibiotics; severity prediction; Lactated Ringer's solution; recurrence; evidence-based management; supportive care.

INTRODUCTION

Acute pancreatitis is one of the most common gastrointestinal emergencies encountered in clinical practice and represents a major cause of hospital admission worldwide. It is characterized by acute inflammation of the pancreas resulting from the premature activation of pancreatic digestive enzymes within the gland, leading to pancreatic autodigestion, local tissue injury, and a variable degree of systemic inflammatory response. Although the majority of patients experience a mild and self-limiting clinical course, a substantial proportion develop severe disease accompanied by local and systemic complications that contribute to significant morbidity, prolonged hospitalization, increased healthcare costs, and considerable mortalit.[1] Consequently, acute pancreatitis remains an important clinical and public health concern that requires timely diagnosis, appropriate risk

stratification, and evidence-based therapeutic intervention. The global incidence of acute pancreatitis has increased steadily over recent decades, making it one of the leading causes of gastrointestinal-related hospital admissions. Despite advances in diagnostic imaging, intensive care medicine, and supportive management, the disease continues to be associated with an overall mortality rate of approximately 5%, which may increase to nearly 30% among patients who develop severe pancreatitis complicated by persistent organ failure or infected pancreatic necrosis.[2–4] The economic burden associated with acute pancreatitis is equally substantial. In the United States alone, acute pancreatitis was identified as the most common gastrointestinal diagnosis requiring inpatient hospitalization in 2009, accounting for more than 270,000 hospital admissions and healthcare expenditures exceeding 2.6 billion dollars.[5] These epidemiological and economic data underscore the growing importance of optimizing clinical management strategies to improve patient outcomes while reducing healthcare resource utilization.

The clinical presentation of acute pancreatitis ranges from mild pancreatic inflammation with rapid recovery to life-threatening multisystem disease requiring intensive care management. To facilitate standardized diagnosis, prognostication, and treatment planning, the revised Atlanta Classification categorizes acute pancreatitis into distinct clinical subtypes based on the presence of local complications and organ dysfunction.[6,7] Mild acute pancreatitis, also known as interstitial edematous pancreatitis, is characterized by pancreatic inflammation without evidence of pancreatic necrosis or organ failure. This form accounts for approximately 80% of cases and generally follows a favorable clinical course, with spontaneous resolution occurring within approximately one week following appropriate supportive care. Conversely, approximately one-fifth of patients develop severe disease characterized by extensive pancreatic and peripancreatic tissue injury, resulting in local complications such as pancreatic necrosis, infected necrosis, abscess formation, and pancreatic pseudocysts. The revised classification further distinguishes moderately severe pancreatitis from severe pancreatitis according to the presence and duration of organ failure, with persistent organ dysfunction lasting more than 48 hours representing the defining feature of severe disease.[6,7] Over the past decade, substantial progress has been achieved in understanding the pathophysiology, clinical progression, and optimal management of acute pancreatitis. Contemporary evidence emphasizes the importance of early disease severity assessment, prompt fluid resuscitation, adequate pain control, nutritional support, and timely recognition of local and systemic complications. In parallel, advances in prognostic scoring systems, laboratory biomarkers, and imaging modalities have enhanced clinicians' ability to identify patients at increased risk for adverse outcomes, thereby facilitating early intervention and individualized management strategies. Furthermore, ongoing research has refined the role of intravenous fluid therapy, antibiotic stewardship, pharmacological interventions, minimally invasive procedures, and multidisciplinary critical care in improving survival and reducing complications among patients with severe disease.

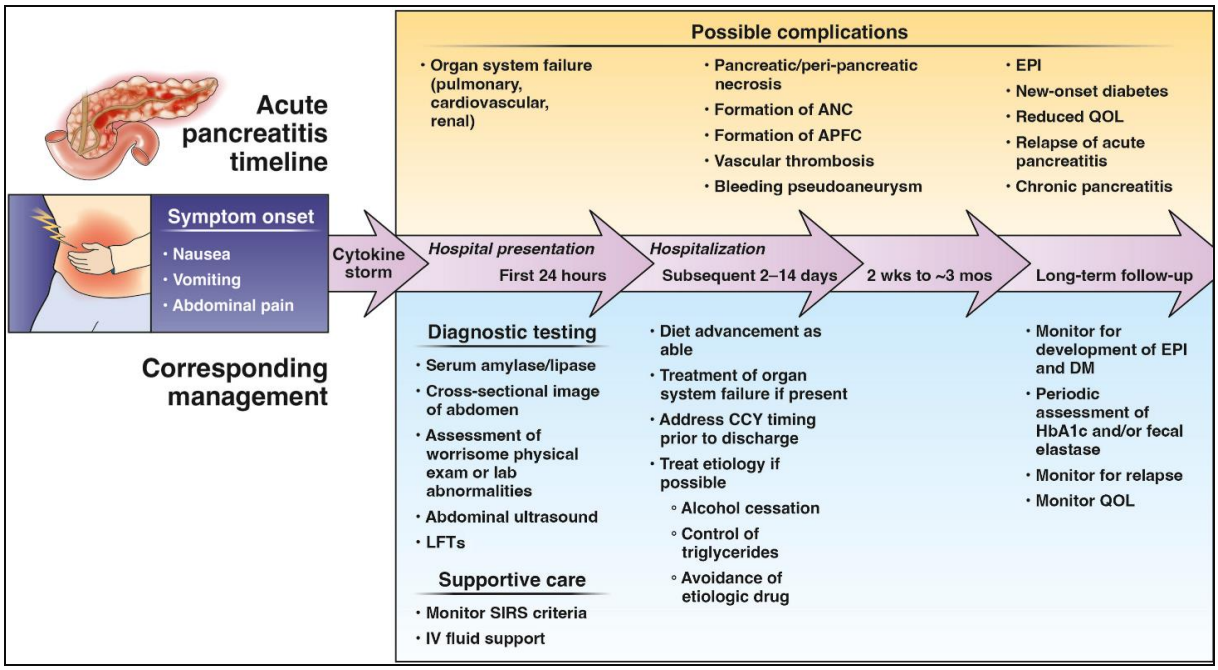

Fig. 1: Acute pancreatitis symptoms, diagnosis, and management.

Predicting Severity

Early prediction of disease severity is a fundamental aspect of the management of acute pancreatitis because it enables clinicians to identify patients at increased risk of complications, organ failure, prolonged hospitalization, and mortality. Accurate risk stratification facilitates

timely admission to higher levels of care, early implementation of intensive supportive therapy, and appropriate monitoring for local and systemic complications. Over the past several decades, numerous prognostic models have been developed to improve the prediction of disease severity. Since the introduction of the Ranson criteria in 1974, several validated scoring systems, including the Acute Physiology and Chronic Health Evaluation II (APACHE II), Bedside Index for Severity in Acute Pancreatitis (BISAP), and the Modified Marshall Scoring System, have been widely used in clinical practice to estimate disease severity and predict adverse outcomes. Although these scoring systems provide valuable prognostic information, their complexity, dependence on multiple clinical variables, and requirement for repeated assessments may limit their routine application in busy clinical settings. Recent evidence has demonstrated that simple laboratory parameters obtained during routine clinical evaluation may offer prognostic accuracy comparable to that of more sophisticated scoring systems. Among these biomarkers, hematocrit, serum creatinine, and blood urea nitrogen (BUN) have emerged as reliable predictors of disease severity. Elevated hematocrit levels at hospital admission or during the first 24 hours have been associated with an increased risk of pancreatic necrosis, reflecting intravascular volume depletion and inadequate tissue perfusion. Similarly, elevated serum creatinine concentrations during the initial 48 hours of hospitalization have been identified as important indicators of severe pancreatic injury and the subsequent development of organ dysfunction.[8–10] Blood urea nitrogen has become one of the most practical and clinically valuable prognostic markers in acute pancreatitis because of its widespread availability, low cost, and strong predictive value. A meta-analysis published in 2011 involving 1,043 patients with acute pancreatitis demonstrated that a BUN concentration of at least 20 mg/dL at hospital admission or a subsequent increase during the first 24 hours was significantly associated with increased risks of persistent organ failure and mortality, with odds ratios of approximately 4.6 and 4.3, respectively.[11] These findings indicate that serial monitoring of BUN provides a simple, objective, and effective method for identifying patients at risk of severe disease progression. Consequently, routine assessment of BUN is recommended as an integral component of the initial evaluation and ongoing monitoring of patients with acute pancreatitis, allowing clinicians to initiate timely therapeutic interventions and optimize clinical outcomes.

Fluid Resuscitation

Early intravenous fluid resuscitation is considered the cornerstone of initial management in patients with acute pancreatitis and remains one of the most effective interventions for preventing disease progression and reducing morbidity and mortality. The principal objective of fluid therapy is to restore intravascular volume, improve pancreatic microcirculation, prevent pancreatic ischemia, and reduce the likelihood of pancreatic necrosis and persistent organ failure. Current evidence supports the initiation of aggressive fluid resuscitation during the first 12–24 hours after hospital admission, with therapeutic goals that include reducing blood urea nitrogen (BUN) and hematocrit levels within the first 24 hours while maintaining adequate tissue perfusion and urine output. These parameters serve as practical indicators of successful volume replacement and improved organ perfusion. The pathophysiology of acute pancreatitis highlights the critical importance of maintaining adequate pancreatic blood flow. During the early stages of the disease, inflammatory processes produce profound intravascular volume depletion through increased vascular permeability, third-space fluid losses, vomiting, reduced oral intake, and systemic inflammatory responses. This hypovolemic state compromises pancreatic microcirculation, which is supplied primarily by branches of the celiac and superior mesenteric arteries. Reduced perfusion of the pancreatic acinar tissue contributes to ischemic injury and promotes the progression from mild interstitial edematous pancreatitis to severe necrotizing pancreatitis.[12] Several mechanisms have been proposed to explain this deterioration, including endothelial injury caused by oxidative stress, increased capillary permeability, hypercoagulability with microvascular thrombosis, and persistent hypovolemia.[13–18] These pathological changes stimulate the release of inflammatory cytokines and other mediators that amplify the systemic inflammatory response syndrome (SIRS), ultimately increasing the risk of pancreatic necrosis, multiple organ dysfunction, and persistent organ failure lasting longer than 48 hours.

Substantial clinical evidence demonstrates that the timing of fluid administration is equally as important as the total volume infused. Early fluid resuscitation has consistently been associated with better clinical outcomes than delayed intervention. One study comparing early and delayed intravenous hydration defined early treatment as administration of more than one-third of the total 72-hour fluid volume during the first 24 hours of hospitalization, whereas delayed treatment involved administration of less than one-third during the same period. Patients receiving early fluid resuscitation experienced significantly lower mortality than those whose hydration was delayed.[20] These findings have been supported by additional investigations, including a retrospective analysis of 436 patients with acute pancreatitis that demonstrated significant reductions in systemic inflammatory response syndrome, persistent organ failure after 72 hours, length of hospital stay, and intensive care unit admission among patients receiving prompt intravenous hydration.[21] Collectively, these studies emphasize that rapid restoration of circulating blood volume during the initial phase of illness plays a crucial role in improving patient outcomes. Despite broad agreement regarding the importance of early fluid therapy, uncertainty remains concerning the optimal fluid type, infusion rate, total

volume, and duration of treatment.[22] Current recommendations from the American College of Gastroenterology advocate administration of isotonic crystalloid solutions at a rate of approximately 250–500 mL per hour during the first 12–24 hours of hospitalization, accompanied by frequent clinical reassessment approximately every six hours to ensure appropriate response to therapy.[23] Additional recommendations suggest administering an initial fluid bolus of 1–2 liters in the emergency department, followed by a maintenance infusion of approximately 250–300 mL per hour or sufficient fluid to maintain a urine output of at least 0.5 mL/kg/hour.[24] During the first 24 hours, the total fluid volume generally ranges between 2.5 and 4 liters, although adjustments should be individualized according to patient age, body weight, cardiovascular status, renal function, physical examination findings, and underlying comorbid conditions.[25] Continuous monitoring of hematocrit and BUN concentrations provides valuable guidance regarding the adequacy of resuscitation, with decreasing values indicating improved intravascular volume and tissue perfusion.

The selection of resuscitation fluid has also received increasing attention. Although comparative clinical studies remain limited, available evidence favors the use of Lactated Ringer's solution over normal saline. The most widely cited prospective study demonstrated that patients receiving Lactated Ringer's solution experienced significantly lower rates of systemic inflammatory response syndrome and reduced serum C-reactive protein concentrations after 48 hours compared with those receiving normal saline.[26] These findings suggest that the balanced electrolyte composition of Lactated Ringer's solution may attenuate systemic inflammation and improve clinical outcomes. Consequently, in the absence of stronger comparative evidence, Lactated Ringer's solution is generally recommended as the preferred crystalloid for initial fluid resuscitation in acute pancreatitis. While aggressive early hydration is essential, clinicians must also recognize the potential hazards of excessive fluid administration. Over-resuscitation may result in pulmonary edema, cardiac overload, and the development of intra-abdominal compartment syndrome, all of which can worsen patient outcomes. One study demonstrated that patients with predicted severe pancreatitis who underwent excessive reduction of hematocrit through overly aggressive fluid administration experienced higher rates of morbidity and mortality than those receiving more carefully titrated therapy.[27]

Pharmacologic Strategies: Antibiotics

The role of antibiotics in the management of acute pancreatitis has undergone substantial revision over the past two decades as evidence from randomized clinical trials and systematic reviews has challenged previous therapeutic practices. Although infected pancreatic necrosis remains one of the most serious and potentially fatal complications of severe acute pancreatitis, current evidence does not support the routine use of prophylactic antibiotics in patients with sterile pancreatic necrosis. Instead, contemporary management emphasizes careful clinical assessment, early identification of infection, and judicious antibiotic administration based on confirmed or strongly suspected infectious complications. This evidence-based approach aims to maximize therapeutic benefit while minimizing unnecessary antimicrobial exposure and the development of antimicrobial resistance. Infected pancreatic necrosis is recognized as the leading cause of late mortality among patients who survive the initial inflammatory phase of acute pancreatitis, accounting for nearly 70% of deaths associated with severe disease. Although pancreatic necrosis develops in only approximately 5% of patients with acute pancreatitis, those with necrotic pancreatic tissue are at considerable risk of secondary bacterial infection, with reported infection rates ranging from 50% to 70%.[2,6] Infection of necrotic tissue markedly increases the likelihood of systemic inflammatory response syndrome, persistent organ failure, sepsis, prolonged hospitalization, and mortality. Consequently, preventing or effectively treating infected necrosis has remained a major therapeutic objective in the management of severe acute pancreatitis. Historically, prophylactic administration of broad-spectrum antibiotics was widely advocated for patients with pancreatic necrosis because early clinical investigations suggested that antibiotics capable of penetrating pancreatic tissue could reduce bacterial colonization and improve clinical outcomes. Initial randomized controlled trials generated encouraging results, leading to widespread adoption of prophylactic antibiotic therapy in routine clinical practice. A meta-analysis published in 2001, which included three randomized controlled trials, reported reductions of approximately 21.2% in the incidence of sepsis and 12.3% in mortality among patients receiving prophylactic antibiotics compared with those who did not receive antimicrobial therapy. However, despite these apparent benefits, no significant reduction in the actual incidence of infected pancreatic necrosis was observed.[29] These findings generated considerable interest but also raised questions regarding the true clinical value of routine prophylactic antibiotic administration.

Subsequent investigations produced conflicting evidence that gradually altered clinical practice. A later meta-analysis published in 2008, incorporating the same randomized controlled trials together with additional data, demonstrated no significant differences in either pancreatic infection rates or mortality between patients treated with prophylactic antibiotics and those receiving placebo.[30] Similar conclusions were reached by a Cochrane systematic review, which found no overall mortality benefit associated with routine antibiotic prophylaxis. The review did identify a potential reduction in pancreatic infection with imipenem specifically; however, this isolated finding was

insufficient to justify routine prophylactic antibiotic use across all patients with acute pancreatitis.[31] More comprehensive evidence became available in 2011 through an analysis of 14 randomized controlled trials involving a total of 841 patients. This investigation demonstrated no statistically significant differences between antibiotic-treated and placebo groups regarding mortality, infected pancreatic necrosis, extrapancreatic infections, or the need for surgical intervention.[32] Collectively, these studies provided compelling evidence that routine prophylactic antibiotics do not improve clinically meaningful outcomes in acute pancreatitis. In addition to the lack of demonstrated benefit, unnecessary antibiotic administration carries important risks. Broad-spectrum antimicrobial therapy may promote the emergence of multidrug-resistant organisms and has been associated with an increased incidence of pancreatic fungal infections, particularly invasive candidiasis, which further complicates the clinical course and increases mortality.[33] These adverse consequences reinforce the importance of antimicrobial stewardship and highlight the need to reserve antibiotics for patients with documented or strongly suspected infection rather than administering them indiscriminately.

Alternative strategies aimed at preventing infection have also been investigated. Probiotic supplementation was proposed as a means of reducing bacterial translocation from the gastrointestinal tract and preventing infection of necrotic pancreatic tissue. However, a meta-analysis published in 2009 demonstrated that probiotic therapy did not significantly reduce the incidence of pancreatic infection or improve survival among patients with acute pancreatitis.[34] Another investigational approach involves selective gut decontamination using orally administered antibiotics to eliminate pathogenic Gram-negative intestinal bacteria and thereby reduce bacterial migration into pancreatic necrosis. Although preliminary findings have suggested potential benefits, current evidence remains insufficient to support routine implementation of this strategy, and additional well-designed clinical trials are required before definitive recommendations can be established.[35] Current international guidelines therefore recommend against the routine use of prophylactic antibiotics in patients with acute pancreatitis, including those with sterile pancreatic necrosis. Antibiotics should not be administered during the initial 24 hours of illness unless there is clear clinical suspicion of a concurrent extrapancreatic infection, such as cholangitis, pneumonia, urinary tract infection, or bloodstream infection.[23] Patients with severe acute pancreatitis frequently present with fever, leukocytosis, systemic inflammatory response syndrome, sepsis-like manifestations, or multiple organ dysfunction, all of which may occur in the absence of bacterial infection. Consequently, differentiating sterile inflammation from true infection is essential to avoid unnecessary antimicrobial exposure. When infection is suspected, diagnostic evaluation should include blood cultures, appropriate imaging studies, and, when indicated, image-guided fine-needle aspiration of pancreatic necrosis for microbiological analysis. Antibiotic therapy should be initiated promptly in patients with confirmed infection but discontinued if subsequent evaluation fails to identify an infectious source.[23]

Emerging Pharmacologic Therapies

Despite substantial advances in understanding the pathophysiology of acute pancreatitis, no targeted pharmacologic therapy has consistently demonstrated meaningful clinical benefit beyond standard supportive care. Over the past several decades, numerous medications have been investigated with the aim of interrupting the inflammatory cascade, reducing pancreatic enzyme secretion, preventing pancreatic autodigestion, or limiting systemic inflammatory responses. Although many of these therapies showed promising mechanisms of action in experimental studies, clinical trials have generally failed to demonstrate improvements in mortality, complication rates, or overall patient outcomes. Consequently, current evidence does not support the routine use of any targeted pharmacological agent in the management of acute pancreatitis. One major area of investigation has focused on drugs that suppress pancreatic exocrine secretion, thereby theoretically reducing pancreatic enzyme activation and limiting ongoing tissue injury. Agents such as somatostatin, octreotide, atropine, glucagon, and cimetidine have all been evaluated for this purpose. However, clinical evidence has consistently shown limited therapeutic value. Among these medications, cimetidine has been extensively studied, and a meta-analysis published in 2002 that included five randomized controlled trials demonstrated that cimetidine was no more effective than placebo in reducing pain, preventing complications, or improving clinical outcomes in patients with acute pancreatitis.[36,37] Similar disappointing findings have been reported for other pancreatic secretion inhibitors, leading to the conclusion that suppression of pancreatic exocrine function alone is insufficient to alter disease progression. Another important therapeutic strategy has involved the use of protease inhibitors. Because acute pancreatitis results from the premature activation of pancreatic digestive enzymes that initiate pancreatic autodigestion, inhibition of these proteases was considered a rational therapeutic target. Agents such as gabexate mesilate and aprotinin were therefore investigated in multiple clinical studies. Nevertheless, available evidence has failed to demonstrate significant improvements in mortality, disease severity, organ failure, or overall clinical outcomes among patients treated with these medications.[38,39] As a result, protease inhibitors are not recommended as routine therapy for acute pancreatitis. Several additional pharmacologic interventions have also been explored in an attempt to modulate the inflammatory response associated with severe acute pancreatitis. These include platelet-activating factor antagonists such as lexipafant, antioxidant therapies, corticosteroids, nitroglycerin, and immunomodulatory

agents targeting inflammatory cytokines, including interleukin-10 (IL-10) and tumor necrosis factor-alpha (TNF-α) antibodies. Despite encouraging theoretical mechanisms, randomized clinical trials have consistently failed to demonstrate clinically significant reductions in mortality, complications, or recovery time with these agents.[40] Therefore, current evidence indicates that none of these targeted therapies should be incorporated into routine clinical practice.

NONPHARMACOLOGIC STRATEGIES

Nutrition

Nutritional support is a fundamental component of the contemporary management of acute pancreatitis and has undergone significant evolution over the past two decades. Historically, patients with acute pancreatitis were routinely maintained on a strict nothing-by-mouth regimen to provide "pancreatic rest" and minimize pancreatic enzyme secretion. Although this approach was widely practiced, subsequent research demonstrated that prolonged bowel rest offered no significant clinical benefit and, in fact, contributed to adverse outcomes. The absence of enteral stimulation leads to intestinal mucosal atrophy, impairment of the gut barrier, increased intestinal permeability, and bacterial translocation, thereby increasing the risk of infectious complications and systemic inflammation.[41] Consequently, current evidence strongly supports enteral nutrition as the preferred method of nutritional support because it preserves gastrointestinal integrity, reduces bacterial translocation, and improves overall clinical outcomes.[42,43] For patients with mild acute pancreatitis, early oral feeding is now recommended once gastrointestinal symptoms begin to resolve. Clinical studies have demonstrated that initiation of a low-fat soft solid diet is generally well tolerated and is as effective as enteral tube feeding in this patient population.[44] Oral intake is usually commenced after improvement in abdominal pain, resolution of nausea and vomiting, discontinuation of parenteral opioid analgesics, and restoration of bowel function. Current recommendations advise initiating enteral nutrition within the first week of hospitalization, as early feeding has been associated with shorter hospital stays, improved nutritional status, and faster clinical recovery without increasing disease-related complications.[45]

The benefits of early enteral nutrition are even more pronounced in patients with severe acute pancreatitis. These individuals are at increased risk of persistent systemic inflammatory response syndrome, pancreatic necrosis, organ failure, and infectious complications, making adequate nutritional support particularly important. Current evidence recommends initiating enteral feeding through a nasoenteric tube within the first 72 hours of hospitalization whenever feasible. A meta-analysis published in 2012 involving 381 patients with severe acute pancreatitis demonstrated that enteral nutrition significantly reduced mortality, infectious complications, organ failure, and the need for surgical intervention when compared with total parenteral nutrition.[46] These findings strongly support early enteral feeding as the preferred nutritional strategy in critically ill patients. Traditionally, nasojejunal feeding has been favored because it bypasses pancreatic stimulation by delivering nutrients beyond the ligament of Treitz. However, more recent evidence suggests that nasogastric feeding provides similar clinical efficacy, safety, and tolerance in many patients, making it a practical and less technically demanding alternative.[47] Nevertheless, when enteral feeding cannot be tolerated or fails to achieve nutritional requirements despite appropriate attempts, parenteral nutrition should be considered. In such cases, current recommendations encourage maintaining low-rate enteral feeding whenever possible while supplementing nutritional deficits with parenteral nutrition to preserve gastrointestinal function and reduce complications.[23]

Management of Underlying Etiology

Successful management of acute pancreatitis extends beyond supportive care and requires prompt identification and treatment of the underlying cause to prevent recurrent attacks. Gallstone disease remains the most common etiology, accounting for approximately 40% to 70% of cases, followed by excessive alcohol consumption, which is responsible for approximately 25% to 35% of episodes.[48–50] Additional causes include hypertriglyceridemia, certain medications, endoscopic procedures, metabolic disorders, and less common genetic or autoimmune conditions. Determining the underlying etiology is therefore an essential component of the initial diagnostic evaluation and long-term management plan. Because gallstones represent the leading cause of acute pancreatitis, abdominal ultrasonography is recommended for all patients presenting with the disease to evaluate the gallbladder and biliary tract for cholelithiasis or biliary obstruction.[23] Ultrasonography is widely available, noninvasive, and highly effective for detecting gallstones and biliary sludge. In patients diagnosed with gallstone pancreatitis, definitive treatment requires removal of the gallbladder to eliminate the source of recurrent biliary obstruction. Accordingly, elective cholecystectomy is recommended during the same hospital admission or before discharge once the acute inflammatory process has resolved sufficiently to permit safe surgical intervention.[23] Early cholecystectomy has been shown to substantially reduce the risk of recurrent pancreatitis and other biliary complications. Endoscopic retrograde cholangiopancreatography (ERCP) also has a well-defined but limited role in the management of acute biliary pancreatitis. Routine ERCP is not recommended because it has not demonstrated clinical benefit in uncomplicated cases and may itself induce procedure-related complications, including post-ERCP pancreatitis.[23] Instead, ERCP should be reserved for carefully selected patients with evidence of persistent biliary obstruction or ascending cholangitis. Indications include worsening liver function tests accompanied by

clinical deterioration, persistent biliary obstruction, or signs of acute cholangitis requiring urgent biliary decompression.[23,51] Outside these specific circumstances, conservative management combined with definitive cholecystectomy remains the preferred approach.

Recurrence

Recurrence of acute pancreatitis remains a significant clinical concern because repeated episodes increase the risk of chronic pancreatic injury, exocrine and endocrine insufficiency, recurrent hospitalizations, and diminished quality of life. Current evidence indicates that approximately one-quarter of patients experience at least one recurrent episode following recovery from an initial attack. This recurrence rate may increase to nearly 50% among patients in whom the underlying cause has not been accurately identified or appropriately managed.[52] Consequently, prevention of recurrent pancreatitis depends primarily on establishing the correct etiology and implementing definitive therapeutic interventions to eliminate or control the precipitating factor. The most common causes of recurrent acute pancreatitis include untreated gallstone disease, continued alcohol consumption, hypertriglyceridemia, anatomical abnormalities of the pancreaticobiliary system, and certain metabolic or genetic disorders. Therefore, comprehensive etiological evaluation should be performed during or shortly after the initial episode to reduce the likelihood of recurrence. In patients with gallstone pancreatitis, timely cholecystectomy is highly effective in preventing future attacks, whereas sustained alcohol abstinence and management of metabolic risk factors are essential for patients with alcohol-related or hypertriglyceridemia-induced pancreatitis. When recurrent episodes occur despite appropriate initial management, further diagnostic evaluation becomes necessary to identify occult or previously undetected causes. Advanced imaging techniques provide detailed assessment of pancreatic and biliary anatomy and facilitate detection of structural abnormalities that may contribute to recurrent inflammation. Contrast-enhanced computed tomography (CT), magnetic resonance cholangiopancreatography (MRCP), and endoscopic ultrasound (EUS) are valuable diagnostic modalities for evaluating pancreatic morphology, biliary and pancreatic ductal anatomy, microlithiasis, biliary sludge, ductal strictures, pancreatic divisum, obstructive pancreatic calculi, and early chronic pancreatitis.[23] Selection of the most appropriate imaging modality depends on the patient's clinical presentation, previous investigations, and suspected underlying pathology. Overall, prevention of recurrent acute pancreatitis requires a comprehensive approach centered on accurate etiological diagnosis, definitive treatment of the underlying cause, lifestyle modification, and appropriate long-term follow-up. Early recognition and management of recurrent disease are essential for minimizing complications, preventing progression to chronic pancreatitis, and improving long-term clinical outcomes.[23,52]

CONCLUSION

Acute pancreatitis remains a major cause of gastrointestinal morbidity despite significant advances in supportive management. Current evidence emphasizes that early severity assessment, prompt goal-directed fluid resuscitation, appropriate nutritional support, and timely identification of the underlying etiology are the most effective interventions for improving clinical outcomes. Routine prophylactic antibiotics and targeted pharmacologic therapies have not demonstrated consistent benefits and should not replace evidence-based supportive care. Early enteral nutrition and definitive treatment of causative conditions, particularly gallstone disease, reduce complications and recurrence. Continued adherence to international guidelines and individualized patient-centered management remains essential for minimizing mortality, preventing long-term sequelae, and optimizing recovery in patients with acute pancreatitis.

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