Advanced Research in Gastroenterology & Hepatology
ISSN: 2472-6400

Review Article
Volume 21 Issue 4- April 2025
DOI: 10.19080/ARGH.2025.21.556069

Adv Res Gastroentero Hepatol

Pancreatitis and Its Management in the Inpatient Setting: A Collaborative Approach Between Internal Medicine and Surgery

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Submission: March 04, 2025 **Published:** March 25, 2025

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Abstract

Background: Acute pancreatitis (AP) represents a significant clinical challenge in the inpatient setting, necessitating prompt diagnosis and multidisciplinary care. While supportive therapy remains the mainstay, ongoing debates persist regarding the optimal strategies for fluid resuscitation, antimicrobial use, and adjunctive therapies. Objective: This review aims to provide an updated, evidence-based overview of current inpatient management strategies for acute pancreatitis, emphasizing internal medicine and surgical perspectives and focusing on fluid resuscitation, infection control, anti-inflammatory interventions, and coordinated multidisciplinary care. Methods: A comprehensive review of recent clinical trials, retrospective analyses, and guideline recommendations was conducted to evaluate the comparative efficacy of crystalloid versus colloid resuscitation, aggressive versus controlled hydration strategies, antimicrobial therapy indications, and emerging adjunctive treatments. Results: Lactated Ringer's solution remains the preferred initial fluid in AP, though colloids may offer advantages in specific contexts.

Aggressive fluid resuscitation has not demonstrated a consistent benefit and may increase the risk of complications such as pulmonary edema and organ failure. Timely, culture-guided antimicrobial therapy is essential in managing infected necrosis, while indiscriminate antibiotic use should be avoided. Adjunctive measures, including NSAIDs, probiotics, and selective anticoagulation, contribute to a more comprehensive care strategy. Coordinated involvement of surgical, critical care, and nutritional services improves outcomes, particularly in severe or complicated cases. Conclusion: Inpatient management of acute pancreatitis requires individualized, evidence-based strategies supported by collaborative multidisciplinary care. Fluid resuscitation protocols should balance efficacy with safety; antimicrobial stewardship is critical to optimizing outcomes. Emerging therapies and less invasive interventions continue to reshape the therapeutic landscape, promising more targeted and effective management approaches.

Keywords: Acute pancreatitis; Multidisciplinary care

Abbreviations: AP: Acute Pancreatitis; NSAIDs: Nonsteroidal Anti-inflammatory Drugs; MODS: Multi-Organ Dysfunction Syndrome; SIRS: Systemic Inflammatory Response Syndrome; IL: Interleukin; TNF-α: Tumour Necrosis Factor Alpha; CRP: C-Reactive Protein; ICU: Intensive Care Unit; EUS: Endoscopic Ultrasound; EN: Enteral Nutrition; LPS: Lipopolysaccharide

Introduction

Pancreatitis, an inflammatory condition, exists primarily in two clinical forms, acute and chronic. Acute pancreatitis is characterized by a sudden onset of inflammation, often presenting with severe epigastric pain radiating to the back, nausea, and vomiting. It ranges in severity from mild, self-limited disease to life-threatening systemic illness with complications such as necrosis, organ failure, and sepsis. In contrast, chronic pancreatitis is a progressive disorder marked by persistent inflammation, irreversible pancreatic damage, fibrosis, and the gradual loss of both exocrine and endocrine functions. This condition often manifests as recurrent abdominal pain, malabsorption, and diabetes mellitus due to long-term pancreatic insufficiency [1,2].

The management of pancreatitis, especially in moderate to severe cases, often necessitates inpatient care. Hospitalization allows for close monitoring of clinical status, timely administration of supportive therapies such as fluid resuscitation and pain control, and rapid intervention in the event of complications. Acute pancreatitis frequently requires dynamic clinical decision-making to manage systemic inflammatory responses, nutritional support, and potential progression to necrotizing pancreatitis or infected collections. Similarly, chronic pancreatitis may require inpatient admission during exacerbations, particularly when pain becomes intractable or complications like pseudocysts, biliary obstruction, or pancreatic duct strictures arise [3-5].

A multidisciplinary, collaborative approach is central to optimizing outcomes in patients hospitalized with pancreatitis. The internal medicine team plays a pivotal role in the early recognition, diagnostic evaluation, and initial stabilization of patients. They manage systemic complications, coordinate imaging studies, and initiate supportive care measures. Concurrently, the surgical team becomes integral when complications such as infected necrosis, abscesses, pseudocysts requiring drainage, or biliary obstruction demand procedural or operative intervention. Increasingly, minimally invasive surgical and endoscopic techniques have enhanced the management of complex pancreatitis cases, highlighting the necessity of seamless collaboration between internal medicine physicians and surgical specialists. This integrated model improves clinical outcomes and streamlines the patient's hospital course by aligning diagnostic clarity with timely therapeutic decisions.

Pathophysiology

Several mechanisms have been suggested to play a role in the complex development of acute pancreatitis (AP). These include abnormal calcium signalling, mitochondrial dysfunction, early activation of trypsinogen in acinar cells and macrophages, endoplasmic reticulum stress, unfolded protein response, and disruption in autophagy. Previous research has shown that both acinar and ductal cells contribute to the onset of acute pancreatitis, and more recent studies show that exosomes containing proteins, nucleic acids, and lipids have been implicated in the progression of AP [6-8].

Calcium signaling

In homeostasis, Calcium is released from the endoplasmic reticulum during zymogen exocytosis and ATP production in the mitochondria. This results in a temporary increase in cytosolic calcium, which is quickly counteracted by two ATP-powered calcium channels that lower the calcium levels in the cytosol. The smooth endoplasmic reticulum pumps help move the calcium back, while the plasma membrane calcium pumps calcium out. If calcium levels remain elevated for an extended period in acinar cells, it can trigger pro-inflammatory pathways, such as the premature activation of trypsinogen and NF-kB, and mitochondrial dysfunction, leading to cell death [7-10].

Mitochondrial dysfunction

All forms of pancreatitis come with mitochondrial abnormalities. There are many mechanisms by which they could cause cell death, but the most important one would be cytochrome c leakage into the cytoplasm of the cell [6-11].

Trypsinogen activation

The activation of trypsinogen is one of the most extensively studied mechanisms in the pathogenesis of acute pancreatitis. Trypsin inhibitors and the release of zymogen granules can prevent premature activation of trypsinogen. Alcohol and bile acids promote the synthesis of lysosomal digestive enzymes and suppress the release of zymogen granules into the acinar cells. This results in the fusion of lysosomes and zymogen granules, a process known as co-localization. The trypsin-centered theory originates from discovering mutations in the PRSS1 gene, which encodes trypsinogen, in hereditary pancreatitis. This rare form of pancreatitis follows an autosomal-dominant inheritance pattern [6-12].

Role of the immune system

In the early stages of acute pancreatitis (AP), the pancreas is infiltrated by inflammatory cells, with macrophages and neutrophils being the first to arrive. These cells contribute to pancreatic damage by phagocytosing necrotic tissue. Pancreatitis is a sterile inflammation, meaning that pathogen-associated molecular patterns (PAMPs) are not involved, at least in the initial phases. Instead, immune cell activation is triggered by damage-associated molecular patterns (DAMPs) released from acinar cell necrosis. These DAMPs facilitate the nuclear translocation of NF-kB family members in the infiltrating immune cells, intensifying the cytokine storm [10-15].

The American College of Gastroenterology classifies Acute Pancreatitis into three main categories. Mild Acute Pancreatitis: characterized by transient pancreatic inflammation without organ failure or local complications. Moderately Severe Acute

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Pancreatitis: involves transient organ failure or local complications that resolve within 48 hours. Severe Acute Pancreatitis: Marked by persistent organ failure and/or local complications lasting more than 48 hours. Local complications include pancreatic pseudocyst, necrosis, fluid collection, and abscess [6].

Two primary classification systems assess disease severity in acute pancreatitis: the Revised Atlanta Classification and the Determinant-Based Classification. The majority of patients with Acute pancreatitis (approximately 80%) experience a mild, self-limiting form of the disease. Mild Acute pancreatitis is characterized by the absence of organ failure and local complications, such as pancreatic necrosis or fluid collections. These patients typically show symptom improvement within 48 hours of admission, can tolerate solid foods, maintain adequate pain control, and have a brief hospital stay. Around 20% of patients develop moderately severe or severe Acute pancreatitis, which carries a higher risk of complications, prolonged hospitalization, and increased mortality. Moderately severe Acute pancreatitis is defined by the occurrence of local complications and/or transient organ failure lasting less than 48 hours. In comparison, severe Acute pancreatitis is marked by persistent organ failure lasting more than 48 hours. Local complications include peripancreatic fluid collections and pancreatic or peripancreatic necrosis, which can remain sterile or become infected. Organ system failure is diagnosed when the modified Marshall scoring system assigns a score of 2 or higher to any of the following organ systems: respiratory, cardiovascular, or renal [6-15].

Initial Assessment and Diagnosis

The clinical presentations of pancreatitis include diffuse or acute upper abdominal pain (80-95%), nausea and vomiting (40–80%), abdominal distension, irritability, fast heart rate and in some patients acute pancreatitis can be complicated by organ failure which include impaired consciousness, fever, tachypnoea, hypotension, abdominal guarding, ileus and/or oliguria. The medical history should include the history of gallstones disease, alcohol abuse, smoking, obesity, hypertriglyceridemia, previous ERCP, or drugs that can induce the disease [16].

The diagnosis for acute pancreatitis requires at least two of the following three criteria: severe epigastric abdominal pain often radiating to the back, laboratory markers like serum lipase activity at least three times greater than the upper limit of typical and imaging abnormalities of the pancreas in patients who has a history of alcohol use or gallstones. Although lipase has greater sensitivity and specificity and is preferred to diagnose AP than amylase, both serum pancreatic enzyme levels peak goes up on the first day and normalize around three to seven days. High glucose levels may result from pancreatic insufficiency and hypocalcaemia due to saponification of peripancreatic fatty tissue [16-19].

The most common clinical symptom of both AP and CP is abdominal pain. AP can present on a spectrum of severity, from mild abdominal pain to severe and multiple organ failure otherwise CP symptoms include abdominal pain, vitamin deficiencies, as chronic pancreatitis progresses, patients may develop exocrine pancreatic insufficiency (steatorrhea and other signs of malabsorption) as well as diabetes due to destruction of pancreatic islet cells. For years, acute and chronic pancreatitis were considered two different diseases. Still, it has been shown that recurrent episodes of acute pancreatitis (AP) can lead to the development of chronic pancreatitis (CP), which is persistent, long-standing inflammation of the pancreas. This condition leads to an irreversible decrease in exocrine and endocrine function and fibrosis of the organ. Approximately 70-80% of AP cases are caused by gallstones and alcohol abuse. Due to the distinctions in management, the differentiation of both etiologist is significant. Ultrasonography is the primary and basic imaging test performed in all patients with suspected acute pancreatitis because it has high accessibility and low cost and gives no radiation exposure [18-23].

For both acute pancreatitis and chronic pancreatitis, imaging methods are part of the diagnosis, this include ultrasound (US), computed tomography (CT), various guidelines recommend both of them as the first-line non-invasive imaging approaches for evaluating patients with pancreatitis, magnetic resonance imaging (MRI), magnetic resonance cholangiopancreatography (MRCP), and endoscopic ultrasonography (EUS); endoscopic retrograde cholangiopancreatography (ERCP) this last one is no longer recommended because is invasive and may result in complications to the patient except in the presence of cholangitis. CT which is the most readily available and widely used, is magnificent diagnostic method, characterized by fast scans with high spatial resolution, presents higher accuracy and sensitivity than ultrasound in diagnosing; is helpful in patients with suspected local complication of AP, such signs of peritonitis, shock, severe abdominal pain or ambiguous ultrasound results and also helps to evaluate the extent of AP; CT also provides essential information for percutaneous management. The method is recommended to perform 48-72 hours after the onset of symptoms rather than at the time of admission because it can distinguish necrotizing acute pancreatitis or interstitial acute pancreatitis, and this can be diagnosed 3 to 4 days from the onset of symptoms [16,17-20].

Patients with suspected pancreatitis should also undergo magnetic resonance imaging (MRI) before other evaluations using invasive investigations to rule out carcinoma. MRI and MR cholangiopancreatography (MRCP) are recommended, especially in patients without specific changes detected on CT. MRI and MRCP are superior to CT for the identification of mild CP. MRI and MRCP are useful for non-invasive evaluation. These imagine methods have many advantages, such as no exposure to radiation, no use of a contrast agent, and no premedication, which is safe for patients with kidney failure or allergies. It can also be used during an acute attack of pancreatitis and cholangitis. Furthermore, MRCP can diagnose local complications as necrotic material or localized fluid-filled lesions and stage the AP. Chronic pancreatitis is diagnosed using imaging scans showing ductal distortion,

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glandular atrophy, and calcifications or histopathologic studies demonstrating widespread fibrosis. Endoscopic ultrasound (EUS) is one of the most sensitive modalities for detecting fibrosis in patients with CP. EUS has better accuracy than non-invasive imaging methods in diagnosing parenchymal and ductal changes, especially during the early stage of the disease. Therefore, when CT and MRI show negative results in patients suspected of having CP, EUS should be performed. However, this procedure is invasive, observer-dependent, and prone to false positive results [16-23].

Inpatient Management: Internal Medicine vs. Surgery Approach

Internal medicine perspective

In the inpatient setting, the internal medicine team is typically at the forefront of managing patients with pancreatitis, especially during the initial phase of hospitalization. Their primary focus centres on supportive care, preventing complications, and determining the need for escalation to procedural or surgical interventions.

Supportive care: fluid resuscitation strategies: Aggressive intravenous fluid resuscitation remains the cornerstone of early management in acute pancreatitis, particularly within the first 24–48 hours. The goal is to maintain adequate intravascular volume, support organ perfusion, and reduce the risk of systemic inflammatory response syndrome (SIRS) and pancreatic necrosis. Current guidelines Favor the use of isotonic crystalloids, most commonly lactated Ringer's solution, due to its favourable effect on acid-base balance compared to normal saline. A goal-directed approach is recommended, guided by clinical parameters such as urine output (>0.5mL/kg/h), haematocrit levels, and blood urea nitrogen (BUN), with frequent reassessments to avoid the complications of over-resuscitation, such as abdominal compartment syndrome and pulmonary edema [24,25].

Pain management: Effective pain control improves patient comfort, reduces metabolic stress, and facilitates early mobilization. Opioids remain the mainstay for moderate to severe abdominal pain in acute pancreatitis, with options including hydromorphone, fentanyl, or patient-controlled analgesia when necessary. Although concerns have been raised about opioids inducing sphincter of Oddi spasm, this has not been conclusively demonstrated in clinical practice. Non-steroidal anti-inflammatory drugs (NSAIDs), such as ketorolac, may be used in mild cases or as adjuncts, provided there are no contraindications like renal dysfunction. Adjunctive therapies, including acetaminophen or regional blocks (e.g., transversus abdominis plane block), may help reduce opioid requirements and enhance multimodal pain control strategies [26,27].

Nutritional support: Nutritional management plays a vital role in the recovery from pancreatitis. EN is preferred over parenteral nutrition in patients who cannot tolerate oral intake, as it maintains gut integrity, reduces bacterial translocation, and lowers the risk of infectious complications. Nasogastric or Naso jejunal feeding can often be initiated within 48–72 hours in patients with moderate to severe pancreatitis. Total parenteral nutrition is reserved for patients with prolonged ileus, high-output fistulas, or intolerance to enteral feeding. Early initiation of nutrition is associated with improved outcomes and reduced length of hospital stay [28,29].

Antibiotic use: Routine antibiotic prophylaxis is not recommended in acute pancreatitis, even in cases of sterile necrosis. Antibiotics are indicated only when there is clinical or radiographic evidence of infected necrosis, cholangitis, or extra pancreatic infections. In such scenarios, agents with good pancreatic tissue penetration, such as carbapenems, fluoroquinolones, and metronidazole, are typically selected. Blood cultures, imaging, and fine-needle aspiration (if needed) may assist in identifying the source of infection and guiding therapy duration [30].

Monitoring for systemic complications: Continuous monitoring is essential to detect and manage systemic complications such as SIRS, multi-organ dysfunction syndrome (MODS), acute kidney injury, and respiratory failure. Vital signs, urine output, and laboratory markers (including hematocrit, creatinine, and lactate) should be assessed frequently. In moderate to severe cases, ICU-level care may be required. Internal medicine physicians are also responsible for coordinating multidisciplinary care with intensivists, nutritionists, and infectious disease specialists to prevent progression to irreversible organ failure and optimize recovery [31,32].

Surgical perspective

The surgical perspective in managing pancreatitis requires careful consideration of when to intervene and how aggressively to approach treatment. Surgical consultation becomes imperative in cases of gallstone pancreatitis, particularly when there is evidence of cholangitis, persistent biliary obstruction, or when patients fail to improve despite conservative management. Additionally, surgical evaluation is warranted when complications such as infected pancreatic necrosis, abdominal compartment syndrome, or perforation of a hollow viscus are suspected, as these conditions significantly increase morbidity and mortality if left untreated [33-35].

Minimally invasive procedures represent a cornerstone of modern surgical management of pancreatitis. Endoscopic retrograde cholangiopancreatography (ERCP) plays a dual role in both diagnosis and treatment, particularly in biliary pancreatitis, where it facilitates removing obstructing gallstones and placing biliary stents to ensure adequate drainage. Similarly, percutaneous drainage has revolutionized the management of pancreatic collections and necrosis, allowing for controlled evacuation of infected material while avoiding the physiological stress of open

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surgery in already compromised patients. These techniques, when applied early and appropriately, can significantly reduce the need for more invasive surgical interventions [32-36].

The timing and indications for necrosectomy or surgical debridement remain subjects of ongoing refinement in surgical practice. The current consensus favors a delayed approach, typically 4-6 weeks after disease onset, allowing for the demarcation of necrotic tissue from viable pancreas. Indications for necrosectomy include documented infected necrosis unresponsive to percutaneous or endoscopic drainage, persistent organ failure despite maximal medical support, or worsening clinical parameters despite conservative management. The step-up approach, progressing from less invasive to more invasive interventions as dictated by clinical response, has demonstrated improved outcomes compared to primary open necrosectomy [35,36].

Post-surgical management requires a multidisciplinary approach focusing on nutritional support, pain management, and prevention of further complications. Long-term management strategies include lifestyle modifications such as alcohol cessation, smoking cessation, and dietary adjustments. For patients with gallstone pancreatitis, cholecystectomy is typically recommended during the same hospitalization or within 2-4 weeks after discharge to prevent recurrence. Follow-up imaging is essential to monitor for the development of chronic pancreatitis, pseudocysts, or pancreatic duct disruption that may require additional intervention. Through this comprehensive approach to post-surgical care and prevention, surgical teams contribute significantly to reducing readmission rates and improving long-term quality of life for patients recovering from severe pancreatitis [37].

Pharmacological Management of Pancreatitis

Effective pharmacological management of pancreatitis is essential to alleviate symptoms, prevent complications, and improve patient outcomes. Among the various therapeutic targets, pain control remains a priority due to the severe abdominal discomfort typically associated with both acute and chronic forms of the disease. Optimal pain management requires a tailored approach that considers the severity of symptoms, the underlying form of pancreatitis, comorbidities, and the risks of long-term pharmacologic therapy [33-39].

Pain management

Pain in pancreatitis is primarily visceral, resulting from inflammation, ductal hypertension, and in some chronic cases, pancreatic neuropathy. The intensity and duration of pain often dictate the choice of analgesic regimen [36].

Opioid vs. non-opioid options: Opioids are the most commonly used agents for moderate to severe pain in acute pancreatitis, given their effectiveness in controlling visceral pain. Medications such as morphine, hydromorphone, and fentanyl are frequently administered, often via intravenous or patient-controlled analgesia (PCA) for immediate relief. While historical concerns existed regarding morphine's potential to cause sphincter of Oddi spasm, studies have shown this effect is not clinically significant, and morphine remains a safe and effective option [40]. However, due to the risk of dependency, constipation, and altered mental status, especially in chronic pancreatitis, long-term opioid use must be approached with caution [39,40].

Non-opioid analgesics can be valuable as adjuncts or alternatives in milder cases or in efforts to reduce opioid requirements. Non-steroidal anti-inflammatory drugs (NSAIDs), such as ibuprofen or ketorolac, offer anti-inflammatory and analgesic effects but are contraindicated in patients with renal impairment or gastrointestinal bleeding risk. Acetaminophen is another option, particularly when NSAIDs are not advisable, provided liver function is normal. For chronic pancreatitis, medications targeting neuropathic pain-such as tricyclic antidepressants (e.g., amitriptyline), selective serotonin and norepinephrine reuptake inhibitors (e.g., duloxetine), and anticonvulsants like pregabalin-may be introduced as part of a multimodal regimen [41,42].

Regional anesthesia: In cases of refractory or severe pain, regional anesthesia techniques have emerged as valuable adjuncts in inpatient management. Epidural analgesia, commonly using a combination of local anesthetics and opioids, has been shown to provide superior pain control compared to systemic opioids alone and may improve splanchnic circulation and reduce the systemic inflammatory response in severe acute pancreatitis [43]. This approach can also reduce the incidence of opioid-related side effects and support early mobilization and improved pulmonary function.

Nerve blocks, including celiac plexus block or splanchnic nerve block, are often considered in chronic pancreatitis patients who suffer from persistent, intractable pain despite optimal pharmacologic therapy. These interventions, performed under fluoroscopic or endoscopic ultrasound guidance, offer targeted relief by interrupting afferent pain pathways from the pancreas. Their use has been associated with improved quality of life and reduced opioid dependency, although benefits may be temporary and vary between individuals [44]. Pain management in pancreatitis requires a multimodal strategy, combining systemic pharmacologic therapy with regional techniques when indicated.

Fluid resuscitation

The primary aim of fluid therapy is to limit or prevent pancreatic necrosis. Any patient with AP has the potential to progress to a severe disease. Patients with mild interstitial pancreatitis are commonly kept under observation in the emergency room, and once their pain settles, they can be discharged. Patients with underlying comorbidities would, however, require closer

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observation. The Revised Atlanta guidelines allow patients to be triaged and evaluated for severity. Patients with moderate and severe AP require observation for organ failure and local or systemic complications and should be started on fluid therapy. It must be recognized that at the time of first interface with the patient, it may not be possible to gauge severity, which may evolve over the next 24-48 hours [45,47].

Choice of fluid: Fluid resuscitation is a cornerstone in managing acute pancreatitis (AP) in hospitalized patients. Both colloids and crystalloids are commonly used, each with distinct advantages and limitations. Colloids-such as dextran, hetastarch, and albumin-are favored for their ability to enhance hemodynamic stability and improve circulatory flow. Due to their larger molecular size, colloids remain within the intravascular compartment, effectively maintaining vascular volume and correcting hypovolemia. However, they may cause complications, including volume overload, coagulopathy, and renal impairment [45].

Crystalloids, such as normal saline, lactated Ringer's, and hypertonic saline, are widely used because of their availability, lower cost, and established safety profile. These fluids distribute across plasma and interstitial spaces, necessitating higher volumes for effective resuscitation. Excessive use can lead to adverse outcomes, including pulmonary edema. Evidence from animal studies suggests that colloids may provide superior outcomes, particularly in stabilizing pancreatic microcirculation and improving survival, as reported by Schmidt et al. and Klar et al. [46] In human studies, dextran has shown potential in reducing pancreatic necrosis and mortality, although results are inconsistent. Conversely, lactated Ringer's solution has demonstrated significant benefits in reducing systemic inflammation and C-reactive protein (CRP) levels, as shown in the study by Wu et al., making it a strong candidate for early resuscitation [45-47].

Despite ongoing debate, a consensus has yet to be reached regarding the optimal fluid choice. Some studies suggest combining crystalloids and colloids-using a crystalloid-to-colloid ratio of 1:3-may yield superior outcomes regarding abdominal pressure and organ function. Hypertonic saline has also shown promising results in preclinical studies, improving microcirculation and reducing pulmonary edema. However, safety concerns remain regarding its use in human resuscitation due to risks such as renal dysfunction. In conclusion, while lactated Ringer's is currently recommended by the American Gastroenterological Association for initial fluid resuscitation, colloids may be considered in cases of severe hypovolemia. Further high-quality studies are needed to determine the optimal fluid strategy tailored to individual patient profiles [45-47].

Volume and rate of fluid resuscitation: what is "aggressive"?: Aggressive fluid resuscitation has been variably defined in the literature. According to the Mayo Clinic, it involves administering ≥33% of the 72-hour fluid volume within the first 24 hours. Chinese studies propose a rate-based definition, with aggressive hydration defined as ≥15mL/kg/h and controlled hydration as 5-10mL/kg/h. Retrospective studies from the Mayo Clinic have suggested improved outcomes with aggressive hydration. In one study, the aggressive group (n=17) had 0% mortality compared to 17.9% in the non-aggressive group (n=28). A second study demonstrated that non-aggressive resuscitation patients had higher SIRS scores. Wall et al. reported an increase in mean fluid administration rates over two decades (234mL/h vs. 194mL/h in the first 6 hours) and noted decreased mortality and necrosis [45-47].

However, other studies challenge the safety of aggressive hydration. De-Madaria et al. found that patients receiving >4.1L in the first 24 hours experienced more persistent organ failure and acute collections. In contrast, volumes between 3.1 and 4.1L were associated with better outcomes. Eckerwall et al. observed that patients receiving >4.0L in 24 hours had significantly more respiratory complications (66% vs. 53%) and higher ICU requirements. Similarly, a Japanese nationwide study found higher fluid volumes within 48 hours correlated with increased respiratory complications and mortality. Studies by Mao et al. further support a cautious approach, reporting higher complication rates, increased sepsis within 28 days, and lower survival in the aggressive hydration group compared to controlled hydration [45-47]. These findings highlight the need for individualized fluid resuscitation strategies. Overly aggressive hydration may increase the risk of complications, while insufficient resuscitation may fail to prevent hypoperfusion-related organ damage.

Antimicrobial therapy

Antimicrobial therapy should be initiated promptly when infection is suspected, even as diagnostic investigations proceed. Clinical signs-such as retroperitoneal gas-may warrant empirical treatment, particularly if percutaneous drainage is planned. If cultures remain negative and no infection source is identified, antibiotics should be discontinued to prevent resistance and adverse effects [48,49]. Empiric therapy should cover both Gram-positive and Gram-negative organisms. Options include monotherapy with carbapenems or combination regimens such as piperacillin-tazobactam or cefepime/ceftazidime with metronidazole. Once microbiologic data are available, therapy should be tailored to ensure efficacy and reduce resistance [50]. Prophylactic antifungal therapy may be considered in patients receiving prolonged broad-spectrum antibiotics. The risk of fungal superinfection, particularly in necrotizing pancreatitis, increases with prolonged antibiotic use. Antibiotic duration should be based on clinical response, tissue damage extent, and systemic inflammation resolution. Typically, antibiotics are discontinued 48 hours after the removal of the final drain if cultures are negative [51].

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Adjunctive therapies

Anti-inflammatory agents: NSAIDs and acetaminophen (paracetamol), especially intravenous forms such as dexketoprofen and diclofenac, serve as effective alternatives to opioids for pain management. They offer anti-inflammatory benefits and have demonstrated comparable analgesic efficacy to opioids in pancreatitis. These agents are generally well tolerated but should be used cautiously in patients with renal impairment, gastrointestinal bleeding risk, or coagulopathy [48].

Probiotics and gut microbiota modulation: Probiotics may modulate the inflammatory response in pancreatitis by reducing proinflammatory cytokines such as IL-6, IL-8, IL-1β, and TNF-α while promoting anti-inflammatory cytokines like IL-10. Clostridium butyricum, in particular, has shown promise in animal models. Probiotics also help maintain intestinal mucosal integrity and may prevent bacterial translocation, reducing the risk of infected necrosis. Early enteral nutrition ("pancreatic rest") is now favored over parenteral nutrition and has been associated with reduced hospital stays, complications, and mortality [48-50].

Anticoagulation in severe cases: Severe pancreatitis may lead to splanchnic vein thrombosis involving the splenic or superior mesenteric veins. Though no definitive guidelines exist, treatment generally follows protocols for other venous thromboembolic diseases, using agents like low-molecular-weight heparin, vitamin K antagonists, fondaparinux, or direct oral anticoagulants (DOACs) such as apixaban. The decision to anticoagulate should be individualized based on thrombosis extent, bleeding risk, and clinical status [51].

Multidisciplinary Coordination in Pancreatitis Care

Effective management of pancreatitis requires close collaboration among internal medicine, surgical, critical care, and nutrition teams. While internal medicine typically initiates diagnostic workup and supportive care, surgical consultation is essential for complications such as infected necrosis, pseudocysts, or biliary obstruction. Escalation to interventional or surgical treatment is warranted in persistent deterioration, failure of percutaneous drainage, or radiologic evidence of complications. Critical care input is vital for patients with SIRS, MODS, or shock requiring ventilatory and hemodynamic support. Nutritional teams ensure the timely initiation of enteral or parenteral support and metabolic stabilization. This multidisciplinary approach enhances coordination, expedites decision-making, and improves outcomes [20-47].

Future Directions and Emerging Therapies

Emerging strategies in pancreatitis management focus on targeted anti-inflammatory therapies and minimally invasive interventions. Experimental agents, including IL-1 and TNF-α inhibitors, aim to blunt the systemic inflammatory cascade in acute pancreatitis, while antioxidants and protease inhibitors are evaluated in chronic disease settings. Technological advances are transforming procedural care. Endoscopic ultrasound (EUS)-guided drainage, percutaneous catheter drainage, and other minimally invasive techniques replace traditional open surgery for necrosectomy and fluid collection management. These techniques are associated with reduced morbidity, shorter recovery times, and improved patient outcomes. As research advances, personalized and organ-sparing approaches are expected to redefine pancreatitis care, supporting earlier intervention and targeted therapies [33-39].

Conclusion

Acute pancreatitis remains a dynamic clinical entity demanding timely, nuanced, and multidisciplinary inpatient care. While initial management often begins under internal medicine, complex cases benefit substantially from surgical, critical care, and nutritional collaboration. The evolving evidence base urges caution against overly aggressive fluid resuscitation and supports the preferential use of balanced crystalloids such as lactated Ringer's. Antimicrobial therapy should be promptly initiated when infection is suspected, then tailored or withdrawn based on culture results to avoid unnecessary exposure. Adjunctive measures, including anti-inflammatory agents, probiotics, and anticoagulation in select scenarios, provide additional tools in improving patient outcomes. As minimally invasive procedures become more prevalent and novel anti-inflammatory therapies continue to emerge, the future of pancreatitis management lies in personalized, pathology-driven interventions guided by a cohesive, multidisciplinary approach.

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DOI: 10.19080/ARGH.2025.20.556069

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