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Acute Pancreatitis: An Integrated Approach to Laboratory Diagnosis and Nursing Management-An Updated Review

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Abstract

Background: Acute pancreatitis (AP) is an inflammatory disorder with a variable course, ranging from a self-limited episode to severe disease associated with organ failure, local complications, and death. Early recognition and effective management are essential to improve outcomes and reduce long-term pancreatic damage.

Aim: This review aims to summarize the diagnosis, causes, clinical course, inpatient management, complications, and strategies for preventing recurrent AP.

Methods: This narrative review addresses the main aspects of AP, including diagnostic criteria, etiological assessment, severity classification, supportive treatment, and cause-specific interventions. It also discusses pancreatic necrosis, acute compartment syndrome, endocrine and exocrine dysfunction, and recurrent disease.

Results: Diagnosis is based on at least two of three features: characteristic abdominal pain, serum amylase or lipase levels at least three times the upper limit of normal, and characteristic imaging findings. Gallstones and alcohol are major causes, while hypertriglyceridemia, medications, procedures, and less common conditions also contribute. Management primarily involves monitoring, intravenous fluid resuscitation, analgesia, and early oral or enteral nutrition. Severe disease may require multidisciplinary care and a minimally invasive step-up approach for infected necrosis. Long-term complications include diabetes mellitus and pancreatic exocrine insufficiency. Recurrence prevention depends on addressing the underlying cause through alcohol abstinence, smoking cessation, cholecystectomy, triglyceride control, medication review, and treatment of specific disorders.

Conclusion: Accurate diagnosis, severity-based supportive care, timely management of complications, and targeted prevention are central to reducing morbidity and preventing progression to chronic pancreatitis, while supporting long-term follow-up and patient education.

Keywords: Acute pancreatitis; diagnosis; management; pancreatic necrosis; complications; recurrence prevention.

Introduction

Acute pancreatitis (AP) is an acute inflammatory disorder of the exocrine pancreas characterized by the premature activation of digestive enzymes within pancreatic acinar cells, resulting in pancreatic tissue injury, local inflammation, and, in severe cases, systemic inflammatory responses and multiple organ dysfunction. Clinically, AP typically presents with sudden-onset, persistent upper abdominal pain, often radiating to the back, accompanied by elevated serum pancreatic enzymes, particularly amylase and lipase. The severity of the disease varies considerably, ranging from mild, self-limiting inflammation to severe necrotizing pancreatitis associated with persistent organ failure and potentially life-threatening complications. Acute pancreatitis represents a significant global health concern, with an estimated annual incidence of approximately 30–40 cases per 100,000 individuals. Although less common in children, the reported incidence in the pediatric population is approximately 10–15 cases per 100,000 children annually [1,2]. Despite advances in diagnostic techniques, critical care, and supportive treatment, severe episodes remain associated with substantial morbidity and mortality, with reported mortality rates ranging from 1% to 5%, particularly among patients who develop pancreatic necrosis and multiple organ failure [1]. Beyond its clinical consequences, AP imposes a considerable economic burden on healthcare systems

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and affected individuals. The average healthcare expenditure associated with an acute episode has been estimated at approximately USD 10,000, although the actual cost varies according to disease severity, hospitalization duration, complications, and the need for intensive care or surgical intervention [3]. Patients with severe disease may require prolonged hospitalization, repeated imaging investigations, nutritional support, and multidisciplinary management. Furthermore, some individuals experience recurrent episodes or develop long-term pancreatic complications, including exocrine pancreatic insufficiency, endocrine dysfunction, and chronic pancreatitis. These consequences may interfere with daily activities, nutritional status, occupational productivity, and overall quality of life, while increasing the long-term financial burden on patients and healthcare providers.

The etiology of AP is heterogeneous, with gallstones and excessive alcohol consumption representing the two most frequently identified causes in adults. Other recognized etiological factors include hypertriglyceridemia, hypercalcemia, certain medications, endoscopic retrograde cholangiopancreatography (ERCP), abdominal trauma, infections, genetic susceptibility, and autoimmune disorders. In a proportion of patients, however, the underlying cause remains unidentified despite appropriate investigations, resulting in a diagnosis of idiopathic acute pancreatitis. Identifying the underlying etiology is essential because it directly influences therapeutic decisions, preventive interventions, and the risk of disease recurrence. Although extensive research has investigated the pathophysiological mechanisms, clinical presentation, diagnostic criteria, classification systems, and therapeutic approaches associated with AP, the development of effective disease-specific pharmacological treatments remains a major challenge. Current management primarily relies on supportive interventions, including appropriate fluid resuscitation, analgesia, nutritional management, monitoring of organ function, and treatment of the underlying cause. Despite numerous investigations into potential targeted therapies, no internationally approved pharmacological agent has consistently demonstrated sufficient clinical benefit to become an established disease-specific treatment for AP.

Accurate and timely diagnosis is therefore fundamental to the effective management of AP. Diagnosis generally depends on the presence of at least two of three established criteria: characteristic abdominal pain, serum amylase or lipase levels exceeding three times the upper limit of normal, and imaging findings consistent with acute pancreatic inflammation. Following confirmation, patients require systematic assessment to identify the underlying etiology, evaluate disease severity, and detect local and systemic complications. The Revised Atlanta Classification provides an important framework for categorizing AP into mild, moderately severe, and severe forms according to the presence and duration of organ failure and local or systemic complications. Accordingly, this review examines the current evidence regarding the diagnosis, classification, and management of AP, with particular emphasis on identifying its underlying causes, evaluating disease severity, recognizing potential complications, and selecting appropriate therapeutic interventions. Understanding these aspects is essential for optimizing clinical decision-making, improving patient outcomes, preventing recurrent episodes, and reducing the clinical and socioeconomic burden associated with this potentially life-threatening condition.

Diagnosis

Diagnostic Criteria and Clinical Presentation

The diagnosis of acute pancreatitis (AP) relies on a combination of clinical manifestations, biochemical investigations, and radiological findings. According to the internationally accepted diagnostic criteria, the diagnosis requires the presence of at least two of the following three features: (1) characteristic abdominal pain suggestive of pancreatic inflammation, typically presenting as acute, persistent epigastric pain that may radiate to the back; (2) serum pancreatic enzyme concentrations, particularly lipase or amylase, at least three times the upper limit of the normal reference range; and (3) characteristic findings of acute pancreatic inflammation on imaging studies, including computed tomography (CT) or magnetic resonance imaging (MRI) [4]. These criteria provide a standardized diagnostic approach and help distinguish AP from other conditions presenting with similar abdominal symptoms. However, clinicians should recognize that relying exclusively on clinical manifestations and biochemical findings may occasionally result in missed or delayed diagnoses. Approximately one-quarter of patients may not be adequately identified using the first two diagnostic criteria alone, highlighting the importance of considering radiological assessment when clinical suspicion remains high despite inconclusive laboratory results [5]. Serum lipase is generally preferred over amylase because of its greater diagnostic specificity and longer period of elevation following disease onset. Nevertheless, pancreatic enzyme concentrations do not reliably reflect disease severity and should not be used independently to predict clinical outcomes. , and may extend toward the back. Other common manifestations include abdominal distension, nausea, and vomiting, which collectively occur in approximately 50–80% of affected individuals The most frequently reported symptom is acute epigastric or diffuse abdominal pain, occurring in approximately 80–90% of patients. The pain is usually persistent, moderate to severe

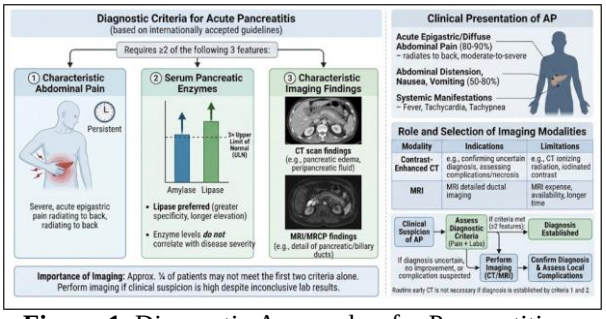


Figure-1: Diagnostic Approaches for Pancreatitis.

Clinical manifestations of AP vary according to disease severity and the presence of associated complications. Systemic manifestations may include fever, tachycardia, and tachypnea, particularly in patients experiencing a pronounced inflammatory response or developing complications [7]. Physical examination may also reveal abdominal tenderness and reduced bowel sounds. In severe cases, hypotension, respiratory distress, and signs of organ dysfunction may indicate systemic involvement. Radiological investigations play an important role in confirming uncertain diagnoses, assessing pancreatic morphology, and identifying local complications. Contrast-enhanced CT is widely used to evaluate pancreatic inflammation, necrosis, fluid collections, and other abdominal abnormalities. However, its use involves exposure to ionizing radiation and, when contrast enhancement is required, potential complications associated with iodinated contrast agents. MRI represents an alternative imaging modality that provides detailed information about pancreatic and biliary structures without ionizing radiation. Nevertheless, MRI is generally more expensive, less readily available in some healthcare settings, and may require longer examination times [6]. The selection of an appropriate imaging modality should therefore consider the patient's clinical condition, renal function, contraindications, radiation exposure, and diagnostic requirements. Importantly, routine early CT is not necessary when the diagnosis is already established through characteristic abdominal pain and elevated pancreatic enzymes. Imaging is particularly valuable when the diagnosis remains uncertain, the patient fails to improve with initial treatment, or complications are suspected. A comprehensive diagnostic approach integrating clinical assessment, laboratory investigations, and selective imaging is essential for establishing an accurate diagnosis and guiding subsequent management decisions.

Etiologies of Acute Pancreatitis

Acute pancreatitis (AP) is a multifactorial inflammatory condition associated with several underlying etiological factors. Identifying the cause of an acute episode is an essential component of the initial clinical assessment because the underlying etiology influences treatment selection, disease recurrence, prognosis, and the need for specific preventive interventions. Therefore, obtaining a comprehensive medical history upon hospital admission is particularly important. This assessment should include previous episodes of pancreatitis, alcohol consumption, smoking habits, medication use, dietary and metabolic disorders, previous abdominal procedures, family history, and relevant comorbidities. The major recognized causes of AP include gallstones, excessive alcohol consumption, hypertriglyceridemia, medication-related pancreatic injury, and post-endoscopic retrograde cholangiopancreatography (ERCP) complications. Other contributing factors include autoimmune disorders, anatomical abnormalities such as pancreas divisum, obesity, hypercalcemia, infections, and abdominal trauma. In some patients, more than one etiological factor may contribute to pancreatic inflammation, making the identification of a single underlying cause difficult.

Gallstones

Gallstones represent the leading cause of AP worldwide and account for approximately 50% of cases in Western populations [8,9]. Biliary pancreatitis usually develops when gallstones or biliary sludge migrate from the gallbladder and temporarily or persistently obstruct the ampulla of Vater. This obstruction interferes with pancreatic juice drainage and may promote premature activation of pancreatic digestive enzymes, resulting in pancreatic inflammation and tissue injury. The likelihood of gallstone formation increases with age, and epidemiological investigations have consistently demonstrated a higher prevalence of cholelithiasis among women. Women account for approximately 66% of reported cases of biliary pancreatitis, reflecting the established association between female sex and gallstone disease [9,10]. The initial evaluation of patients with suspected AP should therefore include an investigation for underlying biliary disease. Transabdominal right upper-quadrant ultrasonography is the preferred initial imaging technique because it is noninvasive, widely available, relatively inexpensive, and effective in detecting gallstones and biliary duct dilatation. Identification of a dilated common bile duct, particularly when accompanied by elevated bilirubin and liver enzymes, may indicate persistent biliary obstruction. When acute cholangitis or ongoing biliary obstruction is suspected, therapeutic ERCP may be indicated to achieve biliary decompression and remove obstructing stones [11]. However, ERCP should not be routinely performed in all patients with gallstone-associated AP because most stones pass spontaneously without requiring endoscopic intervention.

Alcohol Consumption and Smoking

Excessive alcohol consumption is another major etiological factor associated with AP and represents the second most common cause in North America and Europe, accounting for approximately

33% of cases [12,13]. Alcohol-induced pancreatic injury involves several mechanisms, including direct toxic effects on pancreatic acinar cells, altered intracellular calcium signaling, increased oxidative stress, and disturbances in pancreatic enzyme secretion. Alcohol metabolites may also promote the formation of toxic fatty acid ethyl esters, which contribute to mitochondrial dysfunction, impaired cellular energy production, and premature activation of digestive enzymes. The risk of alcohol-associated pancreatitis is influenced by the quantity, frequency, and duration of alcohol consumption. Binge drinking, commonly defined as consuming five or more alcoholic drinks during a single drinking session, represents an additional risk factor. The corresponding alcohol intake has been estimated at approximately 70 g for men and 56 g for women [14,15]. Epidemiological observations have also identified an increase in AP presentations during holiday periods, potentially reflecting changes in drinking patterns and episodes of excessive alcohol consumption. Nevertheless, alcohol-associated pancreatitis frequently occurs in individuals with a history of prolonged alcohol exposure over several years rather than following a single episode of excessive drinking [16]. Smoking is an independent risk factor for AP and is also associated with recurrent acute episodes and progression to chronic pancreatitis [17]. Tobacco exposure may aggravate alcohol-induced pancreatic injury through oxidative stress, inflammatory signaling, and alterations in pancreatic ductal function. The combined presence of alcohol consumption and smoking may further increase pancreatic injury compared with either exposure alone. Some observational studies have suggested that low alcohol consumption may be associated with a protective effect against an initial episode of pancreatitis among nonsmokers, potentially through stimulation of pancreatic ductal secretion. However, this association does not extend consistently to recurrent episodes and should not be interpreted as evidence that alcohol consumption prevents pancreatitis [18,19]. Smoking cessation and avoidance of excessive alcohol consumption remain important preventive measures, particularly in patients with previous episodes of AP.

Hypertriglyceridemia

Hypertriglyceridemia is an important metabolic cause of AP and has been estimated to account for approximately 9% of cases globally, making it one of the leading etiological factors after gallstones and alcohol [20]. However, its contribution varies considerably between populations. For example, a study conducted at a high-volume tertiary healthcare center in China reported that approximately 33% of AP episodes were attributable to hypertriglyceridemia, demonstrating the influence of geographical, metabolic, and population-related differences [21]. According to the classification described by the Endocrine Society, hypertriglyceridemia can be categorized into mild (150–500 mg/dL), moderate (500–1000 mg/dL), and severe (>1000 mg/dL) elevations in serum triglyceride concentrations [22]. The risk of AP increases substantially when triglyceride levels exceed 1000 mg/dL. An estimated 4% increase in pancreatitis incidence has been reported for every additional 100 mg/dL increase above this threshold [23]. The underlying mechanism involves pancreatic lipase-mediated hydrolysis of triglycerides into free fatty acids. At excessive concentrations, these fatty acids may exert direct toxic effects on pancreatic acinar cells and vascular endothelial structures, contributing to inflammation, microcirculatory disturbances, and pancreatic tissue damage. A lipid profile should be obtained early during hospital admission when hypertriglyceridemia is suspected. This is particularly important because serum triglyceride concentrations may decrease rapidly following fasting and fluid administration, potentially obscuring the initial metabolic abnormality. Identifying hypertriglyceridemia also helps distinguish primary metabolic pancreatitis from cases in which elevated triglycerides coexist with other risk factors. Hypertriglyceridemia-associated AP may be associated with a more complicated clinical course, particularly when accompanied by metabolic disturbances such as diabetes mellitus and obesity [24]. Management includes supportive treatment of AP and correction of the underlying metabolic abnormality.

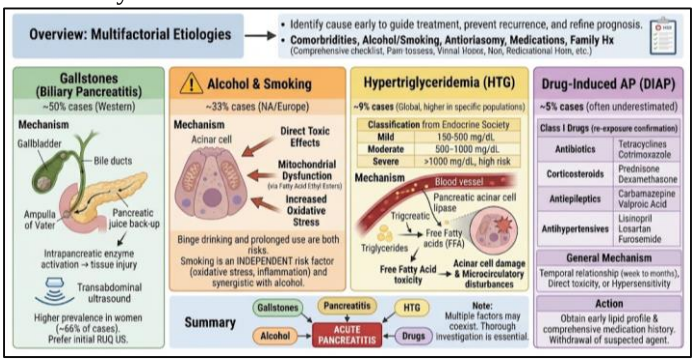


Figure-2: Etiology of Acute pancreatitis.

Drug-Induced Acute Pancreatitis

Medication-related pancreatic injury represents an estimated 5% of AP cases, although its actual prevalence may be underestimated because establishing a causal relationship between a particular medication and pancreatic inflammation is often challenging [25]. A comprehensive medication history is therefore essential during the initial assessment. Clinicians should investigate recently prescribed medications, long-term treatments, dosage changes, over-the-counter preparations, and herbal supplements. Although some drug-associated episodes develop within approximately one week of treatment initiation, others may occur after several weeks or months of exposure. Drug-induced AP has

been classified according to the strength of available clinical evidence. Class I medications include drugs supported by case reports demonstrating recurrence of pancreatitis following re-exposure to the suspected agent, providing relatively strong evidence of a causal association [26]. Reported medications include certain antibiotics, such as tetracyclines and cotrimoxazole; corticosteroids and hormonal agents, including prednisone, dexamethasone, and estradiol; antiepileptic medications, such as carbamazepine and valproic acid; antihypertensive drugs, including lisinopril, losartan, and furosemide; and opioid analgesics, particularly codeine [27]. The mechanisms responsible for drug-induced AP vary between medications. Proposed mechanisms include direct pancreatic toxicity, accumulation of toxic metabolites, hypersensitivity reactions, metabolic disturbances, and alterations in pancreatic ductal secretion. Some medications may produce dose-dependent pancreatic injury through intrinsic toxic effects or the formation of harmful metabolites [28]. When drug-induced AP is suspected, the clinical assessment should consider the temporal relationship between medication exposure and symptom onset, alternative etiological factors, and improvement following drug withdrawal. Discontinuation of the suspected medication is generally recommended when a causal association is considered clinically plausible.

Post-ERCP Pancreatitis

Post-ERCP pancreatitis is a recognized procedure-related complication that develops following endoscopic retrograde cholangiopancreatography. ERCP is commonly performed for the diagnosis and treatment of biliary and pancreatic duct disorders, including choledocholithiasis, biliary obstruction, and selected pancreatic duct abnormalities. Although it provides important therapeutic benefits, manipulation of the pancreaticobiliary system may induce pancreatic inflammation. Estimates of the overall incidence vary according to patient characteristics, procedural techniques, and diagnostic definitions. A systematic review incorporating more than 100 randomized clinical trials reported that approximately 9% of patients developed post-ERCP pancreatitis, with rates reaching approximately 14% among individuals classified as high risk [29]. Several procedural factors increase the likelihood of this complication. These include repeated attempts at biliary cannulation, unintended pancreatic duct cannulation, pancreatic contrast injection, difficult cannulation, and prolonged procedural duration. These interventions may cause mechanical trauma to the papilla, increase intraductal pressure, impair pancreatic juice drainage, and initiate inflammatory responses. Patient-related risk factors are also important. Young women, individuals with relatively small bile ducts, and patients with sphincter of Oddi dysfunction represent recognized high-risk groups [30,31]. Prevention is an essential component of ERCP practice. Appropriate patient selection, experienced endoscopic operators, careful cannulation techniques, and evidence-based pharmacological prophylaxis can reduce the incidence of post-ERCP pancreatitis. Rectal nonsteroidal anti-inflammatory drugs, particularly indomethacin or diclofenac, are commonly recommended for eligible patients. Prophylactic pancreatic duct stenting may also be considered in selected high-risk cases.

Other Causes of Acute Pancreatitis

In addition to the major etiological factors, several less common conditions may precipitate AP and should be considered when the initial investigations fail to identify a clear cause. These include autoimmune disorders, anatomical abnormalities, hypercalcemia, abdominal trauma, viral infections, cardiovascular surgical procedures, and certain toxic exposures [32]. Autoimmune pancreatitis is an immune-mediated inflammatory disorder that may present with pancreatic enlargement, abdominal symptoms, and biochemical abnormalities. It is commonly associated with elevated immunoglobulin G4 concentrations in type 1 autoimmune pancreatitis, although diagnosis requires integration of clinical, serological, radiological, and histopathological findings. Anatomical abnormalities, particularly pancreas divisum, may also contribute to recurrent pancreatic inflammation in selected individuals. This congenital variation results from incomplete fusion of the dorsal and ventral pancreatic ducts and may impair pancreatic drainage through the minor papilla in susceptible patients. Hypercalcemia is another potential cause of AP. Elevated serum calcium concentrations may promote premature activation of trypsinogen within pancreatic acinar cells, contributing to pancreatic injury. Common underlying conditions include primary hyperparathyroidism and certain malignancies. Consequently, serum calcium measurement is particularly relevant in patients with unexplained or recurrent AP.

Infectious causes include viral pathogens such as coxsackievirus B, cytomegalovirus, mumps virus, and severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Viral infections may induce pancreatic inflammation through direct cellular injury, immune-mediated mechanisms, or systemic inflammatory responses. Nevertheless, the presence of a viral infection does not necessarily establish it as the cause of pancreatic inflammation, and alternative etiologies should be investigated. Abdominal trauma may also produce pancreatic injury through direct mechanical damage, particularly following blunt abdominal trauma. In addition, AP has been reported following cardiac bypass surgery. The proposed mechanisms include pancreatic ischemia, impaired tissue perfusion, and systemic inflammatory responses associated with cardiopulmonary bypass. These cases may improve

as cardiovascular function and pancreatic perfusion recover [33]. Scorpion envenomation represents another uncommon cause of AP, particularly in regions where scorpion stings are prevalent. Venom-induced autonomic stimulation and excessive pancreatic secretion may contribute to pancreatic inflammation and systemic manifestations. Management requires supportive treatment, assessment of systemic toxicity, and consultation with local poison control authorities. Antivenom administration may be considered according to the scorpion species, severity of envenomation, and regional treatment protocols, although evidence supporting its specific effectiveness in preventing or treating AP remains limited.

Pathophysiology

Understanding the pathophysiological mechanisms underlying acute pancreatitis (AP) is essential for developing therapeutic strategies that minimize pancreatic tissue damage and systemic complications. The disease begins with an initial insult that injures pancreatic acinar and ductal cells, disrupting normal intracellular calcium signaling and impairing the physiological coupling between cellular stimulation and digestive enzyme secretion [34,35]. Increased exposure to toxins or other triggering factors can intensify cellular injury, reduce adenosine triphosphate (ATP) production, and interfere with essential cellular processes. These disturbances contribute to defective autophagy, abnormal endolysosomal function, inflammasome activation, inflammatory cytokine release, and ultimately pancreatic cell necrosis [34,36]. The inflammatory response is mediated by several pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF-α) and interleukins IL-1α, IL-1β, IL-6, and IL-18. These mediators amplify the initial pancreatic injury and promote the development of local inflammation and, in some cases, microscopic hemorrhage [37]. The inflammatory cascade may subsequently extend through lymphatic and systemic circulation, affecting multiple organs, particularly the liver, lungs, heart, kidneys, and gastrointestinal tract. This systemic involvement can result in multiple organ injury and systemic inflammatory response syndrome (SIRS), which represents an important early manifestation of severe AP [38]. Furthermore, gastrointestinal inflammation and tissue damage may compromise intestinal barrier integrity, facilitating bacterial translocation into the systemic circulation and potentially contributing to disease progression [39]. The bacterial species involved may influence disease severity, with Enterococcidae frequently associated with severe disease [40]. Obesity is another factor associated with increased disease severity. Excessive adipose tissue and adipocyte lipolysis within pancreatic and surrounding adipose tissues may release free fatty acids, aggravating local inflammation and contributing to systemic complications [41,42].

Severity of Pancreatitis

Assessing disease severity is essential for determining prognosis, selecting appropriate treatment, and identifying patients who require intensive monitoring. Although an initial prediction can be made at hospital admission, the definitive classification often requires observation over time because organ dysfunction and local complications may develop as the disease progresses. The Revised Atlanta Classification (RAC) is the most widely accepted system and categorizes AP into three severity levels [4]. Mild AP is characterized by the absence of organ failure and local or systemic complications. Moderately severe AP involves transient organ failure lasting less than 48 hours and/or local complications, including pancreatic fluid collections and necrosis. Severe AP is defined by persistent organ failure lasting more than 48 hours. Approximately 65–70% of patients experience mild disease with symptom resolution within several days, whereas 20–25% develop moderately severe disease associated with pancreatic injury, fluid collections, or necrosis. Approximately 10% develop severe AP, often requiring prolonged hospitalization and intensive critical care [4,43]. An alternative approach is the Determinant-Based Classification, which identifies four categories: mild, moderate, severe, and critical. This system considers pancreatic necrosis, its infection status, and the presence and duration of organ failure [43,44]. However, accurately predicting disease progression remains challenging. Capillary leakage, reflected by endothelial dysfunction and plasma protein loss, has been associated with multiple organ failure. Serum albumin concentrations may therefore provide useful prognostic information [46–48]. Genetic variations have also been investigated as potential determinants of disease severity, although available evidence remains limited [49].

Acute Pancreatitis in Special Populations

In elderly patients, gallstones represent the most common cause of AP. Frailty, multiple comorbidities, polypharmacy, and underlying malignancies may complicate diagnosis and worsen clinical outcomes. Idiopathic AP is also relatively common, accounting for approximately 30–40% of cases in this population. Medication-related pancreatitis, pancreatic tumors, and autoimmune pancreatitis should therefore be considered during evaluation. Serum IgG4 measurement may help investigate suspected autoimmune pancreatitis [50–55]. In children, AP is less common, but the diagnostic criteria remain the same as those used in adults. Typical manifestations include abdominal pain, distension, nausea, and vomiting. Transabdominal ultrasonography is the preferred initial imaging modality because it avoids radiation

and contrast exposure [56]. Pregnancy is another clinical setting associated with AP, with an estimated incidence of approximately one case per 5,000 pregnancies. Hormonal changes may alter biliary function and increase gallstone formation. Estrogen exposure and hypertriglyceridemia may also contribute to disease development. Despite these risks, maternal and fetal outcomes have improved through advances in early diagnosis, imaging techniques, and clinical management [57–61].

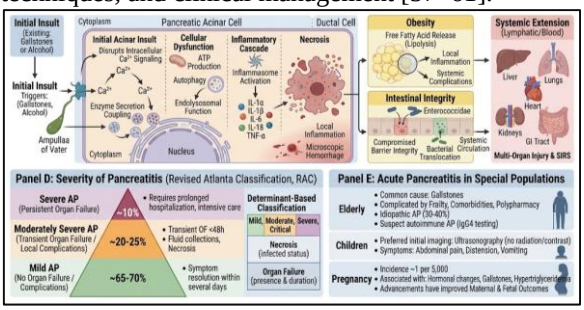


Figure-3: Pathophysiology of acute pancreatitis.

Inpatient Management of Acute Pancreatitis

The inpatient management of acute pancreatitis (AP) focuses on controlling symptoms, maintaining physiological stability, preventing complications, and supporting pancreatic recovery. Patients require regular clinical monitoring, including assessment of vital signs, oxygen saturation, fluid balance, urine output, and organ function. Initial investigations generally include serum amylase and lipase, a lipid profile with triglyceride measurements, complete blood count, comprehensive metabolic panel, serum electrolytes, hemoglobin A1c, and transabdominal ultrasonography. Initial treatment primarily involves oxygen supplementation when indicated, intravenous fluid resuscitation, adequate analgesia, and appropriate nutritional support.

Respiratory monitoring is an important component of inpatient care because severe AP may produce systemic inflammation, respiratory impairment, and hypoxemia. Oxygen supplementation should be adjusted according to the patient's oxygen saturation (SpO_2), with a general target range of 94–99%. However, patients with chronic obstructive pulmonary disease (COPD) or other conditions associated with an increased risk of carbon dioxide retention may require a lower target range of 88–92%. The oxygen delivery device, flow rate, and saturation measurements should be documented regularly to evaluate treatment effectiveness. Patients presenting with oxygen saturation below 85% require prompt oxygen administration, with the delivery rate adjusted according to their clinical response [62]. Arterial blood gas analysis should be considered when significant desaturation occurs or respiratory deterioration is suspected.

Intravenous fluid administration is a fundamental component of early AP management, particularly during the first 24 hours following disease onset. Appropriate fluid replacement helps compensate for intravascular volume depletion caused by inflammatory capillary leakage and fluid accumulation within the third space. Maintaining adequate circulating volume improves tissue perfusion and helps counteract pancreatic and systemic microcirculatory disturbances [63]. Fluid administration should be individualized according to the patient's hemodynamic condition, hydration status, renal function, and response to treatment. The source recommendations describe initial infusion rates of approximately 5–10 mL/kg/h [24]. Treatment goals include maintaining a heart rate below 120 beats/min and achieving urine output greater than 0.5 mL/kg/h. However, excessive fluid administration should be avoided because volume overload may worsen respiratory, cardiovascular, and renal dysfunction and increase mortality risk [64]. Lactated Ringer's solution has demonstrated potential advantages over normal saline during initial resuscitation. Clinical trials have suggested that it may reduce inflammatory responses and improve clinical outcomes [65,66]. Fluid management should involve continuous reassessment through the resuscitation, optimization, stabilization, and evacuation phases of the ROSE approach.

Effective pain control is essential because abdominal pain represents one of the principal manifestations of AP. Opioid analgesics have demonstrated safety and effectiveness in randomized clinical trials and may be administered initially to control moderate-to-severe pain [67]. Nonsteroidal anti-inflammatory drugs (NSAIDs) represent an alternative option in appropriate patients but should be avoided when renal impairment is present [68]. Pain intensity, analgesic requirements, and treatment response should be assessed regularly. Early oral nutrition, when tolerated without worsening abdominal discomfort, may also contribute to reducing pain intensity and duration [69]. In addition to physical symptoms, psychological distress and anxiety require attention. Providing reassurance, clear information, and psychological support can improve patient confidence and facilitate recovery.

AP induces a hypermetabolic and catabolic state characterized by weight loss, insulin resistance, increased protein breakdown, and enhanced lipolysis. Inadequate nutritional intake and associated infections may further aggravate these metabolic disturbances [71]. Therefore, early nutritional support is an essential component of inpatient management. Oral feeding should be initiated as soon as tolerated. When patients cannot maintain adequate oral intake, liquid nutritional supplements or enteral tube feeding should be considered, preferably within 48 hours of admission [72]. Enteral nutrition helps preserve gastrointestinal barrier integrity, reduces bacterial translocation, and may attenuate systemic inflammatory responses [73]. Parenteral nutrition should be reserved for patients in whom oral and

enteral approaches cannot provide adequate nutritional requirements. Although combined enteral and parenteral nutrition may be considered in selected cases, available evidence has not established its superiority over oral or enteral nutrition alone [74].

Complications of Acute Pancreatitis

Pancreatic necrosis and infected peripancreatic collections are important local complications of acute pancreatitis (AP), particularly in patients with severe or persistent symptoms. Patients experiencing severe abdominal pain lasting more than three days require further clinical assessment, including inflammatory markers such as C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR). Computed tomography (CT) is an important imaging modality for identifying pancreatic necrosis, fluid collections, and other local complications. When intervention becomes necessary, endoscopic or percutaneous drainage is generally considered before more invasive surgical procedures [75]. Evidence of infection, including gas within a necrotic collection on CT, may indicate infected pancreatic necrosis and the need for therapeutic intervention. Management may involve appropriate antibiotic therapy and drainage, followed by necrosectomy when necessary. However, the timing and extent of intervention should depend on the patient's clinical condition. The step-up approach has become the preferred strategy for managing infected pancreatic necrosis. It involves beginning with minimally invasive procedures, such as percutaneous or endoscopic drainage, and progressing to more invasive interventions only when the initial treatment fails. A landmark controlled trial by van Santvoort and colleagues demonstrated that this strategy reduced morbidity and mortality compared with primary open necrosectomy [76]. Subsequent research comparing early and delayed catheter drainage in infected necrotizing pancreatitis highlighted the importance of individualizing intervention timing according to clinical status [77]. Recent reviews have further examined the indications for endoscopic necrosectomy, drainage techniques, intervention timing, and multidisciplinary management [78,79]. Overall, conservative management and a step-up strategy are preferred whenever clinically appropriate because they can reduce treatment-related complications and improve patient outcomes.

Acute compartment syndrome (ACS) is a serious complication of severe AP. Extensive pancreatic and peripancreatic inflammation can cause tissue edema and fluid accumulation within the abdominal cavity. This increases intra-abdominal pressure and may compromise blood flow to vital organs. Reduced organ perfusion can subsequently contribute to organ dysfunction and multiple-organ failure [80]. Early recognition and intervention are essential to prevent further deterioration. Management requires close monitoring and appropriate measures to control intra-abdominal pressure. Surgical and minimally invasive interventions may be considered in selected patients, depending on the severity of the condition and response to initial management [81–84].

Repeated episodes of AP may cause progressive pancreatic parenchymal damage and fibrosis, impairing insulin-producing capacity and contributing to insulin resistance. Consequently, patients may develop impaired glucose tolerance or diabetes mellitus following an acute attack. Tu et al. reported that approximately 60% of patients developed impaired glucose tolerance within five years of their first AP episode [85]. The risk may be greater among patients undergoing pancreatic necrosectomy because of the associated loss of pancreatic tissue. Regular assessment of glucose metabolism is therefore important during follow-up. Patients experiencing recurrent hypoglycemia while receiving antidiabetic treatment may require adjustment of their therapeutic regimen. Continuous subcutaneous insulin infusion is one possible option in selected cases [86].

Pancreatic exocrine insufficiency may occur following AP, affecting more than 50% of patients in some reports, although its frequency decreases during subsequent months. Extensive pancreatic necrosis, particularly involving more than 50% of the gland, increases the likelihood of persistent enzyme deficiency. Pancreatic enzyme replacement therapy may therefore be required to improve digestion and nutrient absorption. Commercial preparations include Creon® and Zenpep®, which contain pancreatic lipase and other digestive enzymes. Treatment may be prescribed in moderately severe and severe AP when oral feeding resumes, with fecal elastase monitoring used to assess exocrine function. A fecal elastase concentration above 200 µg/g indicates normal exocrine function [87]. The healthy adult pancreas normally secretes approximately one to two million units of lipase daily, together with proteases, carbohydrate hydrolases, lipid hydrolases, and nucleases [88]. The recommended starting dose of pancrelipase is approximately 50,000 lipase units with each main meal and half this amount with snacks. Doses can subsequently be adjusted according to clinical response and the persistence of steatorrhea.

Prevention of Recurrence of Acute Pancreatitis

Preventing recurrent acute pancreatitis (AP) is an important component of long-term patient management. Approximately 20% of patients experience another episode following their initial attack, and nearly half of those with recurrent episodes may eventually develop chronic pancreatitis. This progression is particularly associated with male patients who consume alcohol and smoke [89].

Therefore, identifying the underlying cause of the initial episode and implementing appropriate preventive measures are essential to reducing recurrence and limiting progressive pancreatic damage. Alcohol abstinence and smoking cessation are fundamental strategies for patients whose pancreatitis is associated with these risk factors. Continued alcohol consumption increases the likelihood of further pancreatic injury, whereas complete abstinence can substantially reduce recurrence. Evidence from an Icelandic follow-up study demonstrated that approximately one-third of patients who continued drinking experienced recurrent AP over five years, compared with none among those who abstained [90]. These findings highlight the importance of counseling, behavioral interventions, and sustained lifestyle modification during follow-up. For patients with biliary pancreatitis, cholecystectomy represents the most effective method of preventing recurrent episodes. Removing the gallbladder reduces the risk of further gallstone-related pancreatic obstruction and subsequent inflammation. Appropriate identification and management of the biliary cause are therefore essential, particularly in patients with a history of recurrent attacks.

In hypertriglyceridemia-associated AP, recurrence prevention primarily depends on controlling serum triglyceride concentrations. The target is to reduce triglycerides below 1000 mg/dL, with an ideal level below 500 mg/dL. Initial management focuses on lifestyle modifications, including regular physical activity, dietary control, and omega-3 fatty acid supplementation. Pharmacological treatment with fibrates, with or without statins, may be introduced when lifestyle measures are insufficient [22]. In patients with treatment-resistant hypertriglyceridemia, plasmapheresis may provide an additional option for reducing triglyceride concentrations and potentially preventing further attacks [91]. Drug-induced AP requires immediate discontinuation of the suspected causative medication. Reintroducing the drug should be considered only when alternative treatments have proved unsuccessful and after careful assessment of the potential risks. In autoimmune pancreatitis, oral prednisolone is recommended at a dose of 2 mg/kg daily, up to a maximum of 60 mg. The dose is gradually reduced by 5–10 mg weekly, followed by a maintenance dose of 5 mg daily for six months, with regular clinical monitoring [92]. Patients with steroid-resistant or relapsing disease may require additional immunosuppressive treatment, including rituximab or azathioprine [93]. Patients with pancreas-sufficient cystic fibrosis also represent a population at risk of developing AP. Treatment with cystic fibrosis transmembrane conductance regulator (CFTR) modulators, including ivacaftor and tezacaftor, may reduce this risk by improving CFTR function [94]. Overall, recurrence prevention requires a cause-specific approach that combines medical treatment, lifestyle modification, and regular follow-up. Early identification of modifiable risk factors and appropriate intervention may reduce repeated pancreatic injury and the likelihood of progression from acute to chronic pancreatitis. The proposed progression of pancreatitis from acute episodes to chronic disease is illustrated in Figure 7 [95].

Conclusion:

Acute pancreatitis is a heterogeneous inflammatory condition that requires prompt diagnosis, assessment of severity, and management tailored to its underlying cause and clinical course. Applying diagnostic criteria, identifying the etiology, and recognizing signs of systemic or local complications help guide treatment. Most patients benefit from supportive care, including fluid resuscitation, pain control, monitoring, and early oral or enteral nutrition. Patients with severe disease may require multidisciplinary management when pancreatic necrosis, infection, or acute compartment syndrome develops. A minimally invasive step-up approach is preferred for managing infected necrotic collections when intervention is indicated. Follow-up is important because pancreatic injury may result in diabetes mellitus or exocrine insufficiency. Preventing recurrence requires addressing modifiable and disease-specific causes through alcohol abstinence, smoking cessation, cholecystectomy for biliary disease, triglyceride control, and appropriate medication or immunosuppressive management. Overall, coordinated acute care and individualized long-term prevention can ultimately reduce recurrent attacks, complications, and progression to chronic pancreatitis.

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