

## Acute Pancreatitis Update of the Current Diagnostic and Therapeutic Approach

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### Abstract:

**Introduction:** The endocrine pancreas is responsible for producing digestive enzymes such as; lipases, proteases and amylases, which are responsible for the metabolism of fats, proteins and carbohydrates respectively for their absorption. Acute pancreatitis is an intracellular disorder of calcium in pancreatic cells, which can trigger necroinflammatory changes and local and systemic complications.

**Objectives:** Carry out a bibliographic review to assess the level of scientific evidence that exists on visual screening to determine whether or not existing recommendations are adequate.

**Material and methods:** Systematic review of scientific articles consulting the MedLine and The Cochrane Library Plus databases, without date restriction, in Spanish and English. Gray literature was included by manual search. No restrictions were made regarding the type of study. The abstracts and, where necessary, the complete articles are reviewed, finally taking into account all the articles that have recently received recommendations on visual acuity screening and eliminating the rest.

Twenty-four bibliographies of articles related to the subject of pancreatitis were selected, performing a bibliographic review and using the most up-to-date information on the subject.

**Conclusions:** Acute pancreatitis is a highly relevant condition for our environment due to its high incidence, the management of this pathology requires adequate knowledge of the underlying clinical and pathophysiological phenomenon of the patient. The correct diagnostic approach and optimal and timely therapeutic management favor a favorable outcome and are associated with fewer complications, morbidity, and mortality.

### 1. INTRODUCTION

The pancreas is an abdominal organ with numerous functions, secreting digestive enzymes and vital hormones. (1) The endocrine pancreas produces digestive enzymes like lipases, proteases, and amylases, responsible for the metabolism of fats, proteins, and carbohydrates for absorption. (1) Acute pancreatitis is an intracellular calcium disorder in pancreatic cells, which can trigger necroinflammatory changes and local and systemic complications. (4).

### 2. EPIDEMIOLOGY

Acute pancreatitis is a highly prevalent condition, ranking as the 21st leading cause of

hospitalization in the United States and the most common reason for gastrointestinal-related hospital admissions (4). With an incidence of 20-80 cases per 100,000 individuals (3), the economic burden is substantial, with approximately \$2.6 billion spent in the United States, underscoring the significance of this pathology (1). The mortality rate for mild cases ranges from 1% to 35% for severe cases (4).

### 3. PATHOPHYSIOLOGY

Acute pancreatitis's pathophysiology comprises four phases (4):

Intracellular Phase: Dysregulated intracellular calcium due to toxic factors and cellular stress

leads to endoplasmic reticulum injury, abnormal calcium clearance, mitochondrial dysfunction, and mitochondrial permeability transition pore damage. These factors result in abnormal ductal and acinar secretion, intracellular zymogen activation, organelle rupture, and necrosis (4).

**Intra-acinar Phase:** Sustained activation of zymogens by cathepsin B triggers oxidative stress, leading to necrosis, apoptosis, and autophagy, along with mitochondrial injury and rough endoplasmic reticulum stress. This injury releases DAMPs, activates the inflammasome, NFκB, and a local inflammatory response, perpetuating the initial injury (4).

**Pancreatic Phase:** Acinar injury promotes cytokine release, leading to pancreatic leukocyte infiltration, establishing a local positive feedback system that sustains the injury and contributes to systemic complications (4).

**Systemic Phase and Multiple Organ Dysfunction Syndrome:** The extension of the pancreatic inflammatory response causes abnormalities in pancreatic microcirculation, coagulation disorders, increased endothelin, platelet activation, elevated levels of IL-1β, IL-6, IL-17, IL-22, and tumor necrosis factor α, leading to increased intestinal barrier permeability, bacterial translocation, and microbiome imbalance (4).

#### 4. ETIOLOGY

The most common causes of acute pancreatitis are gallstones or biliary sludge in 40-50% of cases, with alcohol being the second most common cause at 20-40%. Less frequent

**Table 1.** Atlanta severity criteria for acute pancreatitis

Grade	Description
<b>Mild.</b>	<ul style="list-style-type: none"> <li>No organ failure.</li> <li>No local or systematic complications.</li> </ul>
<b>Moderate.</b>	<ul style="list-style-type: none"> <li>Organ failure resolving within 48 hours.</li> <li>Local or systemic complications without persistent organ failure.</li> </ul>
<b>Severe.</b>	<ul style="list-style-type: none"> <li>Persistent organ failure &gt;48 hours.</li> </ul>

Organ failure is defined as:

**Respiratory:** Oxygen arterial pressure/fraction of inspired oxygen less than or equal to 300.

**Circulatory:** Systolic blood pressure less than 90 mmHg unresponsive to fluids.

**Renal:** Plasma creatinine concentration greater than or equal to 170 μmol/L (1).

causes include medications, post-endoscopic retrograde cholangiopancreatography, trauma, surgery, hypercalcemia, and hypertriglyceridemia (4,5).

#### 5. CLINICAL PRESENTATION

Acute pancreatitis often presents as an emergency, requiring hospitalization for symptom management and resolution. The typical presentation includes severe, sudden-onset abdominal pain, which may radiate to the back in a belt-like fashion or diffusely. In 80% of cases, it is associated with vomiting (1).

#### 6. DIAGNOSIS

The diagnosis of acute pancreatitis requires the presence of at least two of the following criteria:

Characteristic abdominal pain of acute pancreatitis (1,2,3,4).

Serum lipase levels elevated at least three times the upper limit of normal (1,2,3,4).

Characteristic findings of acute pancreatitis on computed tomography, magnetic resonance imaging, or ultrasound (3,4).

#### 7. CLASSIFICATION AND SEVERITY

Acute pancreatitis can manifest in two ways:

**Edematous interstitial pancreatitis:** The majority of patients exhibit localized or diffuse pancreatic thickening (1,2).

**Necrotizing pancreatitis:** In 5-10% of cases, necrosis develops in acute pancreatitis (1,2). The Atlanta classification (see Table 1) is a useful tool for assessing the severity of acute pancreatitis, with three severity levels (1).

#### 8. COMPLICATIONS

Multiple local or systemic complications can occur in patients with acute pancreatitis. Local complications are categorized into four types of collections based on radiological and pathological findings (Table 2). Systemic complications (Table 3) include pulmonary, cardiac, hematological, renal, metabolic,

central and peripheral nervous system, and miscellaneous issues (4,6).

**Table2.** Local complications of acute pancreatitis.

<b>1.Acute Pancreatic Fluid Collection (Figure 1).</b>	Peripancreatic fluid associated with ANP without associated necrosis, during the first 4 weeks.
<b>2.Pancreatic Pseudocyst (Figure 2).</b>	Encapsulated collection of fluid with a well-defined inflammatory wall, occurring after the first 4 weeks..
<b>3.Acute Necrotic Collection (Figure 3).</b>	Collection with varying amounts of fluid and associated necrotic tissue, occurring within the first 4 weeks..
<b>4.Walled-Off Necrosis (Figure 4).</b>	Mature encapsulated pancreatic collection or peripancreatic necrosis that has developed a well-defined wall, occurring after 4 weeks.

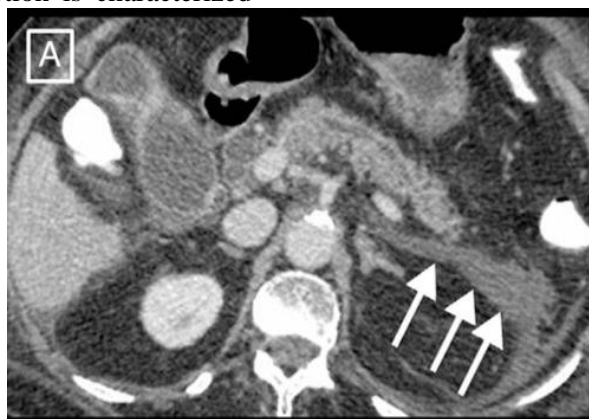
**Table3.** Systemic Complications of Acute Pancreatitis

<b>1. Pulmonary</b>	Hypoxia, atelectasis, pneumonia, pleural effusion, acute respiratory distress.
<b>2. Cardiac</b>	Shock, pericardial effusion, arrhythmias.
<b>3. Hematological</b>	Disseminated intravascular coagulation, hemolytic-uremic syndrome.
<b>4. Metabolic</b>	Hypocalcemia, hyperglycemia, hypertriglyceridemia, acidosis.
<b>5. Central Nervous System</b>	Psychosis, encephalopathy, retinopathy.
<b>6. Peripheral</b>	Fat necrosis, arthritis.
<b>7. Miscellaneous</b>	Rhabdomyolysis.

**9. RADIOLOGY**

Acute peripancreatic fluid collection is a homogeneous fluid collection that adapts to fascial spaces and planes (see Figure 1). A pseudocyst results from the persistence of peripancreatic fluid collection over time, surrounded by granulation tissue (see Figure 2). Acute necrotic collection is characterized

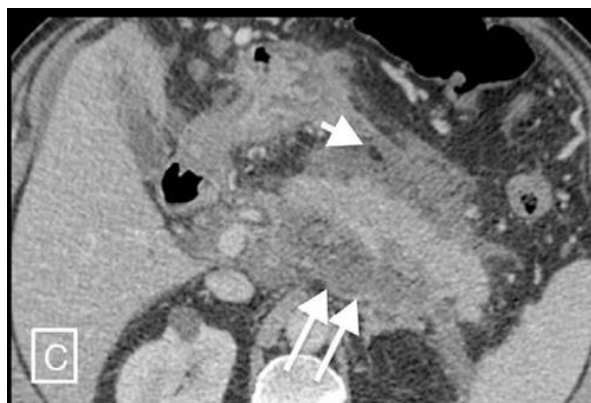
by heterogeneous content, consisting of fluid mixed with solid or semisolid elements (see Figure 3). If a necrotic collection persists for more than 4 weeks and becomes completely encapsulated, it is termed encapsulated necrosis, with solid elements of varying density within the liquid (see Figure 4).



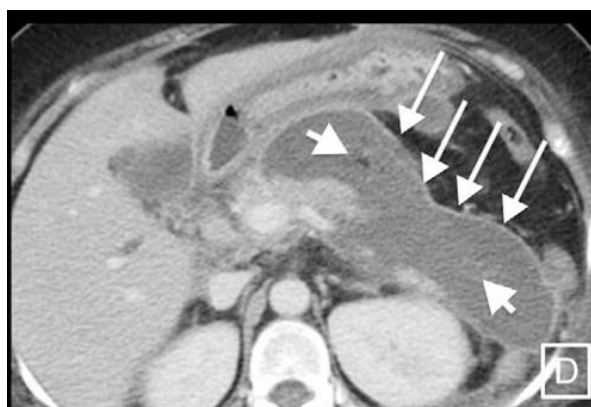
**Figura1.** Acute Pancreatic Fluid Collection.



**Figura2.** Pancreatic pseudocyst.



**Figura3.** Acute necrotic collection.



**Figura4.** Walled-off necrosis.

## 10. MEDICAL MANAGEMENT

The management of acute pancreatitis is based on fluid resuscitation, analgesia, assessing the etiology of acute pancreatitis, early nutrition, evaluating infection suspicion, and surgical management if necessary (4,8,9). Fluid management in mild cases involves an initial 20 mL/kg bolus within the first hour, followed by an infusion of 2-3 mL/kg/hr for mild cases and 1-2 mL/kg/hr for severe cases (4,8,9).

Analgesia options include paracetamol, NSAIDs, and opioids, with a recommendation for multimodal analgesia. Epidural blockade is associated with reduced mortality, less extensive necrosis, acidosis control, splanchnic vasodilation, and improved microcirculation in the pancreatic bed (4,10,11).

Regarding nutrition, it is not necessary to completely resolve pain or normalize pancreatic enzyme levels before resuming the diet. Early initiation of the diet is associated with a 64% reduction in mortality and a 61% reduction in the frequency of multiorgan failure. A solid, low-fat diet is recommended. In the presence of infection, patient mortality is significantly affected (4,12).

In patients with acute pancreatitis, the presence of infection should be suspected, and during the first week, extrapancreatic causes of infection should be sought, and antibiotic use should be considered based on clinical findings and local epidemiology. Between the second and third weeks, consideration should extend to pancreatic-associated infections (4).

## 11. SURGICAL MANAGEMENT

Open necrosectomy is associated with high morbidity and mortality rates of up to 25%, making it a last resort. Less invasive techniques involve catheter drainage followed by minimally invasive necrosectomy methods, which are associated with reduced risk of diabetes, organ failure, abdominal wall injuries, exocrine pancreatic insufficiency, ICU stays, and hospitalization duration (4).

Various endoscopic drainage techniques, such as stents or catheters between the collection and gastric cavity, and direct endoscopic necrosectomy, are currently employed. For endoscopic approaches, the lesion should be larger than 3 cm in diameter, have a mature wall (>4 mm), and be within 10 cm of the gastrointestinal lumen (4).



Regarding cholecystectomy in cases of biliary pancreatitis, a systematic review of nine studies involving 998 patients with biliary pancreatitis demonstrated that early cholecystectomy is associated with a lower incidence of recurrent admissions for pancreatitis, cholecystitis, and biliary colic, with reports of these complications in up to 25-30% of patients (4). Performing this procedure is not associated with higher intraoperative complications, conversion to open cholecystectomy, or mortality (4). Cholecystectomy should be performed after recovery in all patients with biliary pancreatitis, including those who underwent endoscopic sphincterotomy during hospitalization. In mild cases, it should be performed within the first 7 days, and in cases of greater severity or local complications, when the patient is stable (4).

### 12. CONCLUSION

Acute pancreatitis is a highly relevant condition in our environment due to its high incidence. Managing this pathology requires a thorough understanding of the underlying clinical and pathophysiological phenomena in patients. Proper diagnostic approaches and optimal, timely therapeutic management contribute to favorable outcomes, reduced complications, morbidity, and mortality.

### 13. ACKNOWLEDGMENTS

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