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Acute Pancreatitis: A clinical perspective

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Abstract

Acute pancreatitis is one of the main gastrointestinal disorders that is characterized by an acute inflammatory process of the pancreatic tissue with variability in severity, usually with a mild and self-limiting presentation, and presenting a low mortality. Biliary etiology is the leading cause of acute pancreatitis worldwide, with greater involvement in the female gender. Within the diagnostic approach, acute abdominal pain and intense onset in the epigastrium radiating to the back is usually the most representative symptom that leads to suspicion of this entity, accompanied by elevation of pancreatic enzymes and the presence of typical image findings of inflammation of the pancreas. The objection to identifying which patients are at risk of developing local or systemic complications has led to the creation of different scales predicting the severity of pancreatitis with performances that are still debatable. Therapeutic management has two fundamental pillars, fluid resuscitation to maintain or restore tissue perfusion and adequate nutritional support to counteract the catabolic state and reduce the rate of infectious complications. In the context in which the cause is a biliary origin, its resolution is important to avoid progression to chronic pancreatitis.

Keywords: Pancreatitis; Acute; Amylase; Abdominal Pain; BISAP.

1. Introduction

Acute pancreatitis (AP) is one of the principal gastrointestinal disorders that cause abdominal pain and is a reason for consultation in the emergency room potentially lethal**,** associated with a substantial mortality and morbidity rate (1). It's a complex entity with a variable progression that represents a grand cost to the health system worldwide. In the US, it represents 2.6 billion dollars to the health system annually (2).

2. Epidemiology

In the past decades, the incidence of acute pancreatitis has considerably increased, close to 20% worldwide and it has a general mortality rate that fluctuates between 1% and 5% and up to 30% in severe cases. Besides, around 20% of patients with AP will develop moderate to severe pancreatitis with peripancreatic necrosis and or target organ damage (3) (**Figure 1**). A possible theory that could explain the rise of its incidence around the world is the high prevalence of obesity, sedentarism, and alcoholic beverage consumption, besides the frequent realization of laboratory tests that contribute to the rise of statistics in the detection and diagnosis of acute pancreatitis.

Moreover, the etiology of acute pancreatitis may vary geographically. For example, gallstones represent 26% of cases of AP in the US and increase up to 66% in Latin America (4). In Latin America in 2006 it was reported an incidence of 15.9 cases for every 100,000 habitants in Brazil, a prevalence of 3% in Mexico in 2001, and in Peru the Ministry of Health

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statistics by the year 2009 stated an incidence of 28 cases for every 100,000 habitants. Gallstone etiology is the principal cause of almost 70% of registered pancreatitis cases in the emergency room (5)(6).

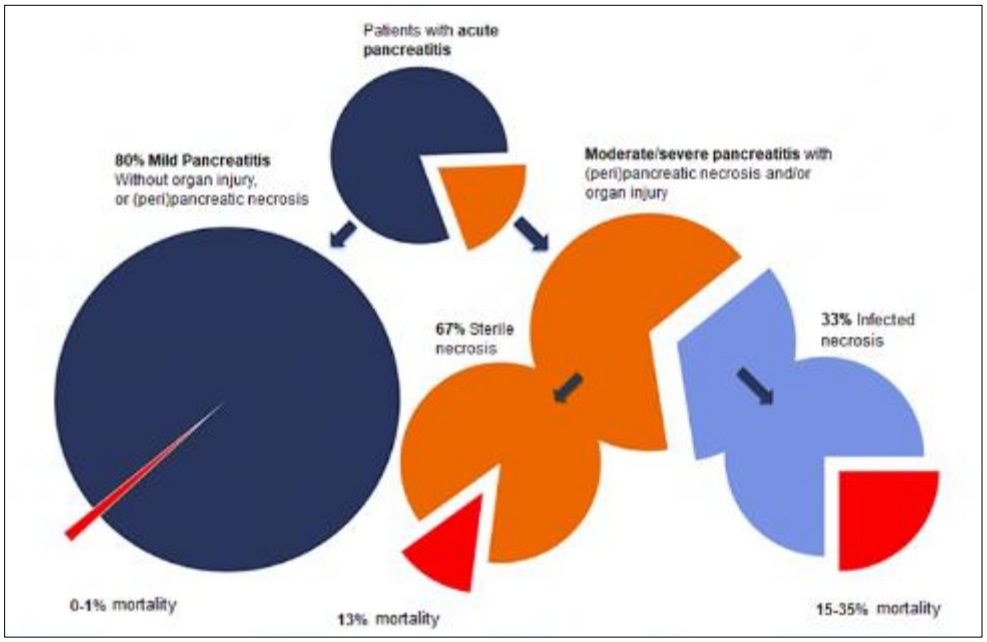

Figure 1 Mortality rate in acute pancreatitis. (Modified from Van Dijk SM et al. Acute pancreatitis: recent advances through randomized trials. Gut. 2017;66(11):2024-32.

In Colombia, it is unknown the real incidence of acute pancreatitis (7). In an observational descriptive study accomplished by Universidad Nacional de Colombia, the characterization of acute pancreatitis patients in the time span between April 2016 and September 2017; they found a higher incidence of AP in the female genre, with an average age of 53 years old where the principal etiology was gallstone of about 70% (8). Another analytic retrospective descriptive study of patients with acute pancreatitis diagnosis that were admitted to a third-level hospital in the time spent between January 1 of 2015 and July 31 of 2017 found that the total studied population had a higher predilection for the female genre among the 4th and 6th decade of life, and yet it was found that ages older than 60 had statistical significance ($p = 0,04$) as a risk factor of severity (9).

3. Definition and classification

Acute Pancreatitis is defined as a disorder characterized by acute necroinflammatory changes in the pancreas, with consequent destruction of acinar cells (10). Physiologically, it can be defined as an acute inflammatory process of the pancreas with variable effects on other regional tissues or distant organ systems (11).

Besides, based on the definitions revised by the 2012 Atlanta classification system, it can be classified according to its severity: **mild, moderately severe, and severe** (**table 1**). Being moderately severe and severe, characterized by the involvement of different organs (respiratory, renal, cardiac) or decompensation of a chronic illness previously diagnosed. Likewise, AP is morphologically classified into 2 subtypes: interstitial edematous pancreatitis and necrotizing pancreatitis (12).

Table 1 Definition of severity in acute pancreatitis. (Revised Atlanta Classification 2012).

Mild acute pancreatitis	Without organ failure Without local or systemic complications
Moderately severe acute pancreatitis	Organ failure < 48 hours and/or Systemic or local complications without persistent organ failure
Severe acute pancreatitis	Persistent organ failure (> 48 hours)

Adapted from: Mederos MA, Reber HA, Girgis MD. Acute Pancreatitis: A Review. JAMA. January 2021;325(4):382.

3.1. Etiology and risk factors

The etiology of acute pancreatitis may be variable. It can even be multicausal depending on the risk factors and the patient's comorbidities **(table 2)**. That being said, the principal causes of AP still are gallstones, followed by excessive alcohol consumption (13).

Table 2 Causes of acute pancreatitis. (Modified from Forsmark CE, et al. Acute Pancreatitis. N Engl J Med. 2016;375(20):1972-81)

Cause	**Approximate frequency**	**Observation**
Gallstones	40%	Alteration of liver enzymes, Hepatobiliary ultrasound.
Alcohol	30%	History of chronic pancreatitis, CAGE* test. .
Hypertriglyceridemia	2-5%	Fasting triglycerides > 1000 mg/dl (11.3 mmol/L).
Genetic cause	Unknown	Acute, chronic and recurrent pancreatitis.
Drugs	<5%	Previous history of atopy, mild presentation. Idiosyncratic.
Autoimmune causes	<1%	Type 1: obstructive jaundice, IgG4 elevation, systemic disease. Type 2: Isolated PA, no IgG4. Both respond to glucocorticoids.
ERCP	5-10% (among ERCP patients)	Pain that improves with NSAIDs, or temporary implant in the pancreatic duct.
Trauma	<1%	Clinical history of contusion or penetrating trauma.
Infection	<1%	Viruses: CMV, mumps, epstein-barr. Parasites: ascaris and clonorchis
Surgical complication	5-10% (among patients with cardiopulmonary bypass)	Severe pancreatitis due to probable ischemic injury.
Obstruction	Rare	Celiac disease, Crohn's disease, pancreas divisum, sphincter of Oddi dysfunction.
Associated condition	Common	Diabetes, obesity, and smoking.

Abbreviations: CMV: cytomegalovirus; ERCP: endoscopic retrograde cholangiopancreatography; CAGE+: Have you ever felt you needed to **Cut down** on your drinking?; Have people **Annoyed** you by criticizing your drinking?; Have you ever felt **Guilty** about drinking?; Have you ever felt you needed a drink first thing in the morning **(Eye-opener)** to steady your nerves or to get rid of a hangover?

An estimation of acute pancreatitis performance in the US during the past decade by *Krishna, SG et al*. showed that gallstones persist as the principal cause of acute pancreatitis but with a decrease in the significant frequency, with an increase in the association with alcohol consumption (2).

In Colombia, the frequency of causes underscore biliary etiology, alcoholic, idiopathic, post-ERCP**,** neoplasms, and hypertriglyceridemia, among others (14).

3.2. Risk factors

The risk factors may be dependent on the etiology supposedly associated with it. In addition, it is known that there is a higher association with men over women when the AP is alcoholic, due to the probable rise in alcoholic consumption in the masculine genre. Other risk factors may be, a history of cholecystitis with or without cholelithiasis, family history, drug consumption such as azathioprine, valproic acid, didanosine, angiotensin-converting enzyme inhibitors, and cardiovascular factors such as morbid obesity, smoking, and diabetes (13).

That being said, with the purpose of analyzing the impact of the risk factors on the morbidity and mortality of hospitalized patients, *Garg SK et al*, determined in a retrospective analysis of the USA- National Emergency Database Sample: 2006 - 2012, that alcoholic beverage consumption was the strongest predictor of the requirement for hospitalization in patients with AP in the emergency room.(OR: 4.53; P<0.0001); followed by elderly patients (age >84 OR: 3.52; P<0.0001), and lastly smocking (OR: 1.75; P<0.0001)(15).

4. Clinical presentation

The most representative symptom is abdominal pain. Usually in the epigastrium or upper left quadrant that radiates to the back or chest, of high intensity and of acute onset (15). This pain is poorly specified and can be exacerbated with food, eating, drinking, or laying down in a supine position. Other associated symptoms can be nausea, emesis, and low-temperature fever.

At the physical exam, it can be identified distention of the abdomen, decreased peristalsis, and peritoneal irritation signs are rare. At the same time, the patient must be evaluated for possible clinical signs that suggest acute cholangitis (fever, abdominal pain, jaundice). Cullen sign can be found, and it is characterized by periumbilical ecchymosis, which suggests peritoneal hemorrhage. In addition, the Grey Turner sign can also be identified, and it is characterized by ecchymosis on one or both abdominal lumbar regions suggesting retroperitoneal bleeding. Although they are only found in 1% of patients and are associated with bad prognosis (11).

4.1. The course of the disease

Acute pancreatitis may have a variable onset progression. Morphologically and based on tomographic criteria, it presents 2 subtypes: Interstitial edematous pancreatitis (80-85% of cases) or necrotizing pancreatitis (15 20% of cases) (16)(13).

Interstitial edematous pancreatitis it's the first morphological presentation and it is characterized by a diffuse and localized augmentation of the pancreas with a homogeneous contrast enhancement shown on the CT scan with contrast. On one hand, the favorable progress of the disease can resolve the pain in the first 72 hours with a benign resolution of the clinical presentation.

On the other hand, collections may appear in the first four weeks after the beginning of the disease, acquiring the denomination of **acute peripancreatic fluid collection**, localized adjacently to the pancreas. It can be single or multiple, homogeneous with fluid density, without associated peripancreatic necrosis, confined to the fascia, undefined capsule, or intrapancreatic extension (4). From there, the collections that appear after the first 4 weeks are denominated **pancreatic pseudocysts**, which are mature collections encapsulated with fluid with a well-defined wall, outside of the pancreas with homogeneous fluid density and no solid components (4)(13).

Necrotizing pancreatitis is the second initial morphological presentation that affects the pancreatic tissue as well as the peripancreatic tissue. Being identified a variable contrast enhancement pattern on the CT scan during the first few days, in fact, the first 4 weeks of its presentation, it is named **acute necrotic consolidation** defined by a heterogeneous density and variable fluid content, necrosis of different stages and locations on the pancreatic and peripancreatic tissue. Nevertheless, in the first 48 to 72 hours the necrotic collection can get infected or remain sterile. Lastly, after 4 weeks of the established necrosis, **a walled-off pancreatic necrosis** is formed, which is characterized by a mature encapsulated necrotic collection that can be pancreatic and or peripancreatic, of well-defined walls, heterogeneous fluid density with no fluid, with different loculations, that yet again can get infected or remain sterile (4).

Patients with acute pancreatitis run the risk of presenting recurrent episodes of acute pancreatitis with a risk of progression to chronic pancreatitis long term, and finally the development of endocrine and exocrine insufficiency in 35% of patients.

The general mortality is approximately 2%, and close to 30% in patients with persistent organ damage. Half of the fatalities that happen in the first two weeks are due to multi-organ failure, meanwhile half of the deaths that happen after the first two weeks are mainly due to pancreatic and extrapancreatic infection (13).

5. Diagnostic approach

The evaluation of the patient with AP requires starting with a clear clinical history and a good physical examination. According to the 2012 Atlanta classifications and definitions revised (updated since 1992), AP diagnosis requires 2 out of the 3 following criteria (17)(18):

- Abdominal pain consisting with pancreatitis.
- Increase of amylase and/or lipase, 3 times the upper normal value.
- Characteristic findings on images of acute pancreatitis (Contrast computed tomography, nuclear magnetic resonance, and less proportion by ultrasonography).

The "gold standard" test is the determination of pancreatic enzymes, mainly amylase and lipase. These proteins present a better precision among the different pancreatic enzymes (elastase and trypsin) for the diagnosis of AP, and they have also been proven to have a sensibility and specificity of 72% and 93% for serum amylase, and 79% and 89% or serum lipase, respectively (17).

There is no standardized range for the pancreatic laboratory levels: lipase and amylase. They differ due to different techniques used in each laboratory, which means, that for patients with pancreatic hypertriglyceridemia and excessive alcohol consumption, the analysis of these enzymes is a challenge and it can be complicated to establish a clinical diagnosis, so it is recommended to perform a CT scan or MRI in cases of diagnostic doubt, unfavorable clinical progress (> 48-72 hours) or patients classified with severe AP (Evaluation of complications)(17).

Other laboratory tests recommended by the World Journal of Emergency Surgery are (19):

- **C-reactive protein ≥ 150 mg/L** on the third day it can be used as a prognosis factor for severe AP (recommendation: 2, evidence level: A).
- **Hematocrit > 44%** represents an independent risk factor of pancreatic necrosis (1B).
- **Urea > 20 mg/dL** as an independent mortality predictor (2B).
- **Procalcitonin** is the most sensitive laboratory test for the detection of pancreatic infection and low serum values appear to be strong negative predictors of infected necrosis (2A).
- **Sérum triglyceride** levels higher than 11.3 mmol/l (1000 mg/dl) indicate the etiology of acute pancreatitis associated with hypertriglyceridemia (2C).

5.1. Stratification of risks and severity

There is no severity score established as the gold standard to p**redict complications** of acute pancreatitis (19). Due to the variability of the clinical progress and the high mortality rate of cases with severe AP, there have been developed multiple predictive and prognosis scales to guide medical conduct. Besides, there is no evidence of Head-to-Head clinical trials that determine the superiority of a clinical scale over others.

The modified Marshall score collects the assessment of three systems: renal, respiratory, and cardiovascular, where a two-point or higher score for each defines the presence of organ failure. Due to its simplicity, this score has an easy application in order to estimate in an objective manner the severity of the disease on its onset (20). However, in an observational study performed recently in Colombia by *Rodríguez A. et al.* where they wanted to establish the concordance between the Marshall, Ranson, and APACHE II scores, found that there is a poor correlation between the different scores of risk classification, so they shouldn't be interpreted equivalently. In addition, this study suggests that between the mentioned scores, the Marshall score could overestimate the risk in cities above 2,000 meters above sea level (21).

Probably the BISAP scale (Bedside index of severity of acute pancreatitis), provides the most reliable score and it is applied in clinical practice every day because its simplicity and capacity to predict the severity, mortality, and organ damage in the same way the APACHE II score and other scales (19). In some cases, it is considered a loss of valuable time (in a critically ill patient) if it is necessary to establish therapeutic conduct with **tools or scores** where the prediction is obtained 24 or 72 hours later.

In **table 3,** it is shown the characteristics and differences of the main principal scales for AP. Likewise, based on the available evidence and recommendations in **Figure 2** is proposed a diagnosis algorithm for patients with acute pancreatitis (5)(19)(22).

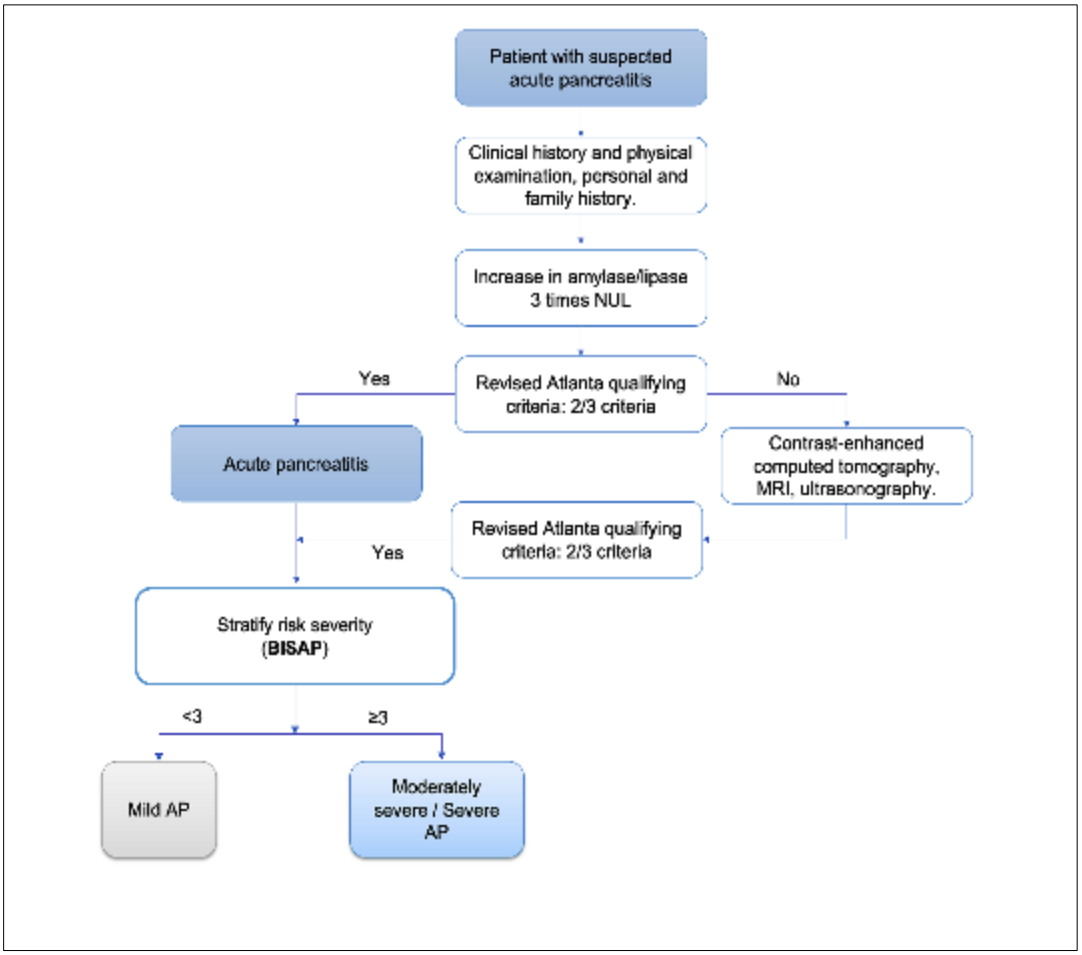

Figure 2 Proposed algorithm for the diagnosis of patients with acute pancreatitis.

Abbreviations: MRI: Nuclear magnetic resonance; AP: Acute pancreatitis. NUL: normal upper limit; BISAP: Bedside Index of Severe Acute Pancreatitis

Other methods used as tools to predict prognoses are the use of computed tomography, and novel predictors based on artificial intelligence. Computed tomography with contrast, with Balthazar scale by degrees of severity, is increasingly falling into disuse due to its limitations, the greater prevalence of mild forms of AP, the requirement of contrast and not having adequate performance in the first 48 hours. On the other hand, there are studies that compared the use of computerized machine learning models compared to APACHE II, RANSON or GLASGOW score, which have concluded greater accuracy in predicting severity in AP (AUC between 0.84 and 0.92). However, their results are lack of validation in clinical trials and do not have standardized algorithms (23).

Table 3 Comparison of risk factors scales: APACHE II, BISAP, Ranson based on the severity definitions of the revised Atlanta classification. (Modified from Mederos MA, et al. Acute Pancreatitis: A Review. JAMA. 2021;325(4):382.)

	APACHE II	**BISAP (2008, Gut)**	**Ranson**
Variables	Age Temperature Mean arterial pressure pH Heart rate Respiratory rate Sodium Potassium Creatinine Acute kidney injury Hematocrit White blood cell count Glasgow Scale FIO2	BUN > 25 mg/dl (>8.9 mmol/L) Alteration of the state of consciousness > 2 Criteria SIRS Age > 60 years Existing pleural effusion	Admission: Age > 55 years Leukocyte count > 16,000/μL LDH > 350 U/L AST >250 U/L Glucose > 200 mg/dl. Within 48 hours: Decrease in hematocrit > 10% Increase in BUN > 5mg/dl Pao2 < 60 mmhg Base deficit > 4 meq/L Fluid loss > 6 L
Original purpose	Disease severity and mortality in ICU patients.	Predictor of mortality in patients with AP	Predictor of mortality in patients with AP.
Severity prediction, AUC (SE)	0.82 (0.03)	Score: ≥3 0.87 (0.16)	0.08)
Severity prediction Sensitivity (95% CI) Specificity (95% CI)	Score: ≥8 0.83 (0.77-0.88) 0.59 (0.56-0,63)	Score: ≥3 0.51 (0.43-0.60) 0.91 (0.89-0.92)	Score: ≥3 0.66 (0.59-0.72) 0.78 (0.76-0.81)
Mortality prediction, AUC (SE)	0.83 (0.16)	Score: ≥3 0.87 (0.03)	0.92 (0.05)
Mortality prediction Sensitivity (95% CI) Specificity (95% CI)	Score: ≥8 0.95 (0.77- 1.00) 0.68 (0.63-0.73)	Score: ≥3 0.56 (4.23-0.7.55) 0.91 (0.90-0.91)	Score: ≥3 0.93 (0.78-0.99) 0.69 (0.65-0.79)
Advantages	It can be calculated in the first 24 hours	5 variables Easy to calculate (1 point/variable) Can be calculated in < 24 hours Specific for PA	Understandable Specific for PA
Limitations	Designed for patients admitted to the ICU Large number of variables It is not specific for AP	Lower sensitivity and specificity than APACHE II	It is calculated after 48 hours Variables not routinely taken in patients - not admitted to the ICU

Abbreviations: AP: acute pancreatitis; APACHE II: Acute Physiology and ChronicHealth Evaluation II; AST: aspartate aminotransferase; AUC: area under the curve; BISAP: Bedside Index of Severe Acute Pancreatitis; BUN: blood urea nitrogen; FIO2: inspired fraction of oxygen; ICU: intensive care unit; PAO2: arterial partial pressure of oxygen; SIRS: systemic inflammatory response syndrome

6. Treatment

The use of score tools are useful to guide the therapy, but they should never overrule the clinical judgment, so the recommendation aims to perform a medical therapy guided by the severity of the disease and concomitant complications. There are 2 fundamental pillars approaching the treatment: 1. Fluid resuscitation to maintain or restore tissue perfusion and 2. Adequate nutritional support to counter the catabolic state and reduce the infectious complication rates.

There are not unified criteria that could determine which patient requires intensive or intermediate care unit management. It can be guided by clinical criteria in conjunction with the complication risk stratification established as moderately severe and severe, in addition to persistent organ failure despite the establishment of an adequate fluid resuscitation (19).

6.1. Fluid resuscitation

The decrease in intravascular volume is not due to a single cause. The interstitial fluid sequestration, reduction of oral consumption, emesis, and peripancreatic inflammation, all contribute to this deficit (24). Consequently, the hydric intake should be initiated early on, preferably with isotonic solutions. According to the AGA, and IPA/APA society recommend goal-guided therapy for fluid management, however, this is with a conditional recommendation that has very low quality (24) (25). Even in some scenarios such as sepsis, goal-guided resuscitation has demonstrated clinical benefit (26).

Randomized clinical trials regarding the type of fluid to use between saline solution at 0.9% and ringer lactate have not demonstrated important outcomes. However, there was a decrease in systemic inflammatory response (SIR) and CRP with the use of ringer lactate but without effect in mortality rate, therefore AGA society has not realized any recommendations (conditional recommendation, very low quality of evidence). Furthermore, retrospective analysis has shown better evidence of the results of using ringer lactate over saline solution aggressively (during the first 24 hours) meaning 20 ml/kg/ bolus and continuing with 3 ml/k/h (5)(22). In addition, it must be considered the possible co-morbidities (heart failure, chronic kidney disease) due to the risk of fluid overload. Subsequently in a recent clinical trial it has been shown early aggressive fluid resuscitation resulted in a higher incidence of fluid overload without improvement in clinical outcomes (27).

Therapy should be guided by clinical monitoring of vital signs such as heart rate, mean arterial pressure, and urine output to determine the individualization of the crystalloid input rate. The goals proposed by IAP/APA (International Association of Pancreatology/American Pancreatic Association) are MAP: 65-85 mmHg, heart rate less than 120 Bpm, urine output 0.5-1 ml/k/h and hematocrit between 34-44%.

6.2. Nutrition

Current guidelines recommend that in acute pancreatitis, feeding should be started within the first 24 hours of diagnosis to reduce complications (28). Based on the high catabolic demand and the risk of infection, the complete resolution of pain is not necessary, nor is the decrease of pancreatic enzymes to their normal level to start enteral nutrition in patients with mild AP. It has been associated with a lower hospital stay (24). In the moderately severe/ severe AP cases with no pain resolution at the fifth (5th) day is recommended to start a low-fat solid or semi-solid enteral nutrition, by nasogastric tube or nasoduodenal tube, because it has been described a beneficial effect by minimizing the exocrine pancreatic secretion.

Parenteral nutrition should be reserved for cases of no tolerance to enteral nutrition or cases without achieving nutritional goals with enteral formulas (22).

6.3. Antibiotics (AB)

In the past few years, the principal gastroenterology societies have recommended with evidence-based knowledge that is not indicated antibiotic use as a prophylactic therapy in patients with AP (19)(22).

The recommendation is antimicrobial coverage against Gram-positive, Gram-negative, aerobic, and anaerobic bacteria in evidence of infection (infected pancreatic and necrosis). Enterobacteriaceae are the principal microorganisms found followed by Staphylococcus aureus, Streptococcus faecalis, Enterococcus and candidas spp (19).

Occasionally it can be difficult to clinically differentiate the inflammatory process from the pancreas and the infection itself, so it is recommended the use of procalcitonin level to discriminate patients that really benefit from antimicrobial medicine. Primarily the scheme should have as first-line medication piperacillin-tazobactam due to its adequate penetration to pancreatic tissue, but other options could be quinolones and carbapenems.

The ideal test to guide the anti-microbial therapy is culture and Gram stain in samples taken by tomography-guided fine needle aspiration, but it is not routinely recommended (19).

Clinical suspicion of infection is based on infection signs (temperature above 38.5° and an increase in the serum inflammatory markers), when there is a new or persistent organ insufficiency that usually is more reliable after the initial phase of IRS or when it is identified CT scan findings of infection such as collections or gas inside the pancreatic collection (4). Besides antibiotics should be administered for extrapancreatic infections such as cholangitis, catheter infections, bacteremia, urinary tract infections, or pneumonia (strong recommendation high-quality evidence) (25)(29).

6.4. Endoscopic retrograde cholangiopancreatography (ERCP)

Generally, ERCP should not be routinely performed. It is recommended during the first 48 hours in cases of severe gallstone AP with concomitant cholangitis and/or persistent gallbladder obstruction. Besides, its use is controversial in cases of severe gallstone pancreatitis in early acute phase (22).

6.5. Other recommendations

Analgesia: Such as the pain is the main presenting symptom, it should be treated as fast as possible and efficiently. Today's evidence about opioid use is limited, and there is not general recommendation so the patient should be individualized, and evaluate the availability of medicine, drug interaction, and local pain control protocols of each Institution. It has been demonstrated a grade of benefit by using epidural analgesia (Bupivacaine) in critically ill patients with a low evidence level to be recommended at the moment (22)(24).

Cholecystectomy: It should be performed prior to the discharge of patients with acute mild gallstone pancreatitis if there are no surgical contraindications (5) (25).

Alcohol: Intervention strategies for alcohol consumption should be performed as early as the moment of diagnosis of alcohol consumption-related AP during hospitalization and alcohol abstinence must be repeatedly reinforced, especially in cases associated with hypertriglyceridemia (30) (31). All of this with educational sessions in order to change the habit of consuming, every 6 weeks after medical discharge for at least 2 years (5).

Table 4 Recommended indications for percutaneous and surgical intervention of pancreatic necrotic collections. (Modified from Leppäniemi A, et al. 2019 WSES guidelines for the management of severe acute pancreatitis. World J Emerg Surg. 2019;14(1):27.)

Indications for percutaneous/endoscopic drainage of pancreatic collection.	**Indications for surgical intervention.**
Clinical deterioration with signs or strong suspicion of infected necrotizing pancreatitis (recommendation 1C). > 4 weeks after onset of illness: Ongoing organ failure without signs of infected necrosis. Continuous obstruction of the gastric, biliary or intestinal outlet due to a large walled necrotic collection. Disconnected duct syndrome. Symptomatic or growing pseudocyst. > 8 weeks after onset of illness: Continuous pain and/or discomfort	As a continuous step in a stepped approach after a percutaneous/endoscopic procedure with established indications. Abdominal compartment syndrome. Acute continuous bleeding when the endovascular approach is unsuccessful. Intestinal ischemia or acute necrotizing cholecystitis during AP. Intestinal fistula extending into the peripancreatic collection.

Abbreviations: AP: acute pancreatitis.

Collection and necrosis treatment: surgical interventions for control of local infected tissue are a debatable point and include invasive and minimally invasive techniques. Pancreatic pseudocysts do not routinely require surgical

interventions if they don't present with clinical signs (persistent abdominal pain, pancreatic secretion obstruction, fluid drain due to the pancreatic duct disconnection, and infection) (11).

In daily practice, there is an effort to delay any invasive intervention for at least 4 weeks to allow the isolation of the necrotic collection (14). The **table 4** presents indications for surgical and percutaneous drainage (19).

7. Conclusions

Acute pancreatitis is an inflammatory disorder of the pancreas that potentially threatens life. The clinician's perspective about this pathology should be centered on the diagnosis and recognizing that there is not always needed to perform initial image studies to establish a diagnosis and stratify its severity.

It is required a good analysis of the associated risk factors, medical record, possible etiology, and risk stratification of each patient as quickly as possible to adequately guide the management stay, the studies to perform, early treatment to avoid complications, inappropriate use of antibiotic schemes and negative outcomes of the disease. In the same way, the pillars of the initial treatment are adequate fluid resuscitation and enteral nutrition once there is no abdominal pain, individualizing the patient's scenario.

The medical follow-up should include programs that support alcohol abstinence as required and monitoring pancreas endocrine function due to the risk of secondary pancreatic insufficiency.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

References

[1] Boxhoorn L, Voermans RP, Bouwense SA, Bruno MJ, Verdonk RC, Boermeester MA, et al. Acute pancreatitis. The Lancet. September 2020;396(10252):726-34.

[2] Krishna SG, Kamboj AK, Hart PA, Hinton A, Conwell DL. The Changing Epidemiology of Acute Pancreatitis Hospitalizations: A Decade of Trends and the Impact of Chronic Pancreatitis. Pancreas. April 2017;46(4):482-8.

[3] Van Dijk SM, Hallensleben NDL, van Santvoort HC, Fockens P, van Goor H, Bruno MJ, et al. Acute pancreatitis: recent advances through randomised trials. Gut. November 2017;66(11):2024-32.

[4] Mederos MA, Reber HA, Girgis MD. Acute Pancreatitis: A Review. JAMA. 26 January 2021;325(4):382.

[5] Valdivieso-Herrera MA, Vargas-Ruiz LO, Arana-Chiang AR, Piscoya A. Situación epidemiológica de la pancreatitis aguda en Latinoamérica y alcances sobre el diagnostic. 19. Acta Gastroenterol Latinoam. 2016 Jun;46(2):102-3. Spanish. PMID: 28703563.

[6] Hellen Jamanca-Milian, Luis Cano-Cardenas. Factores pronósticos de severidad en pancreatitis aguda en un hospital peruano. Rev. Fac. Med. Hum. January 2020; 20(1):14-19.

[7] Nieto Julio Alberto, Rodríguez Samuel Jesús. Manejo de la pancreatitis aguda: guía de práctica clínica basada en la mejor información disponible. rev. colomb. cir. 2010; 25(2): 76-96.

[8] Martinez E. Caracterización de los pacientes con Pancreatitis Aguda que ingresaron al Hospital Universitario Nacional entre Abril de 2016 y Septiembre de 2017. Universidad nacional de Colombia. 2018 .

[9] Puerto Horta LJ, Medina Rojas R, Nuñez Romero LR, Jiménez Sánchez HC, Olaya Ramírez JG, San Juan JF, Puerto Horta SA, Martínez Montalvo CM, Medina Cabrera MM. Manejo y desenlaces de la pancreatitis aguda en un hospital de cuarto nivel (Huila, Colombia), 3 años de experiencia. Rev. colomb. Gastroenterol. 2019;34(1):10-6.

[10] Garber A, Frakes C, Arora Z, Chahal P. Mechanisms and Management of Acute Pancreatitis. Gastroenterol Res Pract. 2018;2018:1-8.

[11] Scott T, Steinberg W. Pancreatitis aguda. Sleisenger y Fordtran. Enfermedades Digestivas y Hepáticas. 11 ed. Elsevier. 2020. cáp 58.

[12] Lankisch PG, Apte M, Banks PA. Acute pancreatitis. The Lancet. July 2015;386(9988):85-96.

[13] Forsmark CE, Swaroop Vege S, Wilcox CM. Acute Pancreatitis. Campion EW, editor. N Engl J Med. 17 November 2016;375(20):1972-81.

[14] Gomez A, Rodriguez A. Caracteristicas de los pacientes con diagnóstico de pancreeatitis aguda atendidos entre enero de 2008 y diciembre de 2011 en un centro de cuarto nivel. Rev Col Gastroenterol. 2015; 30 (2):171-176.

[15] Garg SK, Sarvepalli S, Campbell JP, Obaitan I, Singh D, Bazerbachi F, et al. Incidence, Admission Rates, and Predictors, and Economic Burden of Adult Emergency Visits for Acute Pancreatitis: Data From the National Emergency Department Sample, 2006 to 2012. J Clin Gastroenterol. March 2019;53(3):220-5.

[16] Aguilar PAÁ, Ramírez CTD. Pancreatitis aguda: fisiopatología y manejo inicial. Acta Médica Costarrica. 16 July 2020;61(1).

[17] Tenner S, Baillie J, DeWitt J, Vege SS. American College of Gastroenterology Guideline: Management of Acute Pancreatitis. Am J Gastroenterol. September 2013;108(9):1400-15.

[18] Gardner TB. Acute Pancreatitis. Ann Intern Med. February de 2021;174(2):ITC17-32

[19] Leppäniemi A, Tolonen M, Tarasconi A, Segovia-Lohse H, Gamberini E, Kirkpatrick AW, et al. 2019 WSES guidelines for the management of severe acute pancreatitis. World J Emerg Surg. December 2019;14(1):27.

[20] Banks PA, Bollen TL, Dervenis C, et al. Classification of acute pancreatitis—2012: revision of the Atlanta classification and definitions by international consensus. Gut 2013;62:102-111.

[21] Rodríguez-Varón A, Muñoz-Velandia OM, Agreda Rudenko D, García-Consuegra E. Concordancia entre las escalas Marshall, Ranson y APACHE II como estimadoras de la morbimortalidad en pancreatitis aguda. Rev Colomb Gastroenterol. 2020;35(3):298-303.

[22] Lee PJ, Papachristou GI. New insights into acute pancreatitis. Nat Rev Gastroenterol Hepatol. Agost 2019;16(8):479-96.

[23] Lee DW, Cho CM. Predicting Severity of Acute Pancreatitis. Medicina (Mex). June 2022;58(6):787.

[24] García G, Cárdenas K, De-Madaria E. Towards evidence-based and personalised care of acute pancreatitis. United European Gastroenterology Journal. 2020;8(4):403-409

[25] Crockett S, Wani S, Gardner T, et al. American Gastroenterological Association Institute Guideline on Initial Management of Acute Pancreatitis. Gastroenterology 2018;154:1096–1101

[26] Rivers E, Nguyen B, Havstad S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. N Engl J Med 2001;345:1368–1377.

[27] de-Madaria E, Buxbaum JL, Maisonneuve P, García García De Paredes A, Zapater P, Guilabert L, et al. Aggressive or Moderate Fluid Resuscitation in Acute Pancreatitis. N Engl J Med. September 2022;387(11):989-1000.

[28] Chaitoff A, Cifu AS, Niforatos JD. Initial Management of Acute Pancreatitis. JAMA. June 2020;323(22):2331.

[29] Vege S, DiMagno M, Forsmark CE, et al. Initial medical treatment of acute pancreatitis: AGA Institute Technical Review. Gastroenterology 2018.

[30] Garg R, Rustagi T. Management of Hypertriglyceridemia Induced Acute Pancreatitis. BioMed Res Int. July 2018;2018:1-12.

[31] K. Bessembinders, J. Wielders, and A. van de Wiel, “Severe hypertriglyceridemia influenced by alcohol (SHIBA),” Alcohol and Alcoholism, vol. 46, no. 2, Article ID agq088, pp. 113–116, 2011.