ISSN: 2753-7919
British Journal of Healthcare and Medical Research; Vol. 13 No. 02 (2026) (300-315)
https://doi.org/10.14738/bjhr.1302.20265

Acute Pancreatitis Evolved to a New Treatment

Brenda Lizeth Herrera Gómez Tagle[1], María de los Ángeles Carmona Alva[2], Jaime Ricardo López Sixtos[3], José de Jesús Macedo Garduño[4], Rubén Omar Gress Tovar[5], Gabriel Cervera Rodríguez[6], Aldair Cruz Gallardo[7], Alexis Nevid Fuentes Otero[8], Indra Lucía Badillo Campuzano[9], Morelos Adolfo García Sánchez[10]

1. Specialist in Surgery. Attached to the Department of Surgery General Hospital "Dr. Rubén Leñero" of the Ministry of Health of Mexico City. Graduated from the National Autonomous University of Mexico. Mexico City. Country Mexico
2. Specialist and Holder of the Specialty Course in Surgery. Attached to the Department of Surgery General Hospital "Dr. Rubén Leñero" of the Ministry of Health of Mexico City. Graduated from the National Autonomous University of Mexico. Mexico City. Country Mexico
3. Specialist in Surgery. Attached to the Department of Surgery General Hospital "Dr. Rubén Leñero" of the Ministry of Health of Mexico City. Graduated from The National Autonomous University of Mexico. Mexico City. Country Mexico
4. Specialist in Surgery. Attached to the Department of Surgery General Hospital "Dr. Rubén Leñero" of the Ministry of Health of Mexico City. Graduated from the National Autonomous University of Mexico. Mexico City. Country Mexico
5. Specialist in Surgery. Attached to the Department of Surgery General Hospital "Dr. Rubén Leñero" of the Ministry of Health of Mexico City. Graduated from Autonomous University of the State of Hidalgo. Mexico City. Country Mexico
6. Specialist in Surgery. Attached to the Department of Surgery General Hospital "Dr. Rubén Leñero" of the Ministry of Health of Mexico City. Graduated from Autonomous University of San Luis Potosí. Mexico City. Country Mexico
7. Specialist in Surgery. Attached to the Department of Surgery General Hospital "Dr. Rubén Leñero" of the Ministry of Health of Mexico City. Graduated from the National Autonomous University of Mexico. Mexico City. Country Mexico
8. Specialist in Surgery. Attached to the Department of Surgery General Hospital "Dr. Rubén Leñero" of the Ministry of Health of Mexico City. Graduated from La Salle University. Mexico City. Country Mexico
9. Specialist in Surgery. Attached to the Department of Surgery General Hospital "Dr. Rubén Leñero" of the Ministry of Health of Mexico City. Graduated from the National Autonomous University of Mexico. Mexico City. Country Mexico
10. *Specialist in Surgery and with a subspecialty in Colon and Rectal Surgery attached to the Surgery Department of the General Hospital Ministry of Health of Mexico City "Dr. Rubén Leñero". Graduated from the National Autonomous University of Mexico, Mexico City. Country Mexico

Abstract: Introduction: Acute pancreatitis is an inflammatory disease of the pancreas that is characterized by a wide spectrum of clinical severity, from mild and self-limiting episodes to severe necrotizing disease with high morbidity and mortality. Objective: To describe the experience of the results obtained from patients with acute pancreatitis with a statistical, deductive, critical, logical and impartial analysis. Method: A retrospective, longitudinal, observational and descriptive study was conducted; presenting results by descriptive statistics, patients present with acute pancreatitis of probable biliary and/or pancreatic pathology. Results: 81 patients (19.75 %) men and 65 women (80.25 %), with a mean age of 39 years, with acute pancreatitis due to acute lithiasis cholecystitis (77.7 %). Once in remission, laparoscopic cholecystectomy was performed in 71 patients (91.02%). Morbidity was 19.75% and adjusted to 4.93%. With a residual choledocholithiasis of 7.4%. No mortality was reported. Discussion: The etiology of acute pancreatitis is a mystery to date, since there is no proven genesis that causes it, it is a potentially fatal inflammatory disease with well-known etiologies; however, in up to 30% of cases no identifiable cause

is found, resulting in the diagnosis of acute pancreatitis. The Atlanta classification for acute pancreatitis is currently the most reliable that focuses on baseline behavior/behavior/prognosis, organ failure, and protective measures as central issues for developing or not developing acute respiratory distress syndrome and increasing mortality rates. Conclusion: acute pancreatitis is a pathology that for centuries has caused fear, brutal health sequelae/limitations to survivors, which has plagued humanity due to such high rates of morbidity and mortality; that in view of this background, overdiagnosis and excessive treatment have been exercised with an increase in the costs of human resources, material and economic, with precarious results. It is concluded that therapeutic management of mild acute pancreatitis, so basic and unorthodox, is beneficial for medical practice that does benefit the patient and the permanently worn-out impoverished health sector.

Keywords: Acute pancreatitis, Acute cholecystitis, Endoscopic retrograde cholangiopancreatography, Cholecystectomy, Choledocholithiasis, Pancreatic necrosis, Pancreatic pseudocyst.

INTRODUCTION

In humans, the pancreas is an elongated accessory digestive gland located retroperitoneally, crossing the bodies of the L1 and L2 vertebrae in the posterior abdominal wall. The pancreas is located transversely in the upper abdomen between the duodenum on the right and the spleen on the left. [1] The pancreas is an abdominal organ that, due to its anatomical location behind the stomach, colon and mesentery, with a small size and a soft yellowish consistency, were sufficient reasons for the late discovery of its physiology. [2]

The first information on the anatomy and physiology of the pancreas is documented only in the sixteenth and seventeenth centuries, Johann Georg Wirsung from whose description of the pancreatic duct in 1642. The function of the pancreas in digestion began to be protocolized in the eighteenth and nineteenth centuries, Claude Bernard who between 1849-1856 described the pancreatic exocrine function and is considered the father of pancreatic physiology.[3] Lipase was discovered in 1815 by the Englishman Alexander Marcet (1770-1822), Opie (1901) proposes his theory of the common canal, suggesting that a stone can produce obstruction of the ampulla of the toilet, allowing bile to reflux from the common bile duct into the pancreatic duct; [4, 5] in addition, the role of the pancreas in carbohydrate metabolism is described in the nineteenth century, and finally insulin was discovered in the first half of the twentieth century. However, the clinical signs of acute pancreatitis were first described in the early nineteenth century, but the diagnosis and/or treatment of this disease even today in the twenty-first century presents certain rough edges. [6].

Acute pancreatitis is an inflammatory disease of the pancreas that is characterized by a broad spectrum of clinical severity, from mild and self-limiting episodes to severe necrotizing disease with high morbidity and mortality. [7] Pathology originating from an intracellular disorder of calcium in pancreatic cells, which can trigger necro inflammatory changes with local and systemic complications; since calcium is an ion whose concentration is robustly regulated in pancreatic cells, generating cytosolic signals as a phenomenon with multiple and complex events that regulate intracellular free calcium levels. [8, 9]

Four phases of the pathophysiology of acute pancreatitis have been distinguished:

- Intracellular
- Intraacinar
- Pancreatic
- Systemic [9, 10]

The family of formyl peptide receptors (FPR), which in humans comprises three main members (FPR1, FPR2 and FPR3), located on chromosome 19, the human FPR1 gene encodes a protein with the canonical structure of seven transmembrane domains, actively modulates inflammatory processes in a ligand-dependent manner, determining the activation or inhibition of intracellular kinase pathways in their development. [11] Adipose tissue plays a crucial role in severe acute pancreatitis due to obesity. Metabolomic analysis revealed abnormal lipid metabolism in obese patients, and several signaling pathways could contribute to the alteration of autophagic flow in acute pancreatitis. [12] In addition, unhealthy, high-sugar, high-fat modern diets and lack of exercise have led to an increase in incidence over the years, and hyper triglyceridemic has overtaken alcohol as the second leading cause of acute pancreatitis. Where excessive production of reactive oxygen species and mitochondrial dysfunction, especially impaired oxidative phosphorylation, play a central role in the spread of inflammatory damage and cellular apoptosis. [13]

It should be noted that the gut microbiome, a dynamic ecosystem comprising bacteria, fungi, viruses, and archaea, plays a central role in maintaining gut homeostasis, modulating immunity, and influencing host metabolism. Bacterial dysbiosis has been the most studied, with an overgrowth of opportunistic pathogens, such as Escherichia coli and Enterococcus spp. [14] Acute pancreatitis is an abrupt onset disease accompanied by profound microbial disruption and systemic inflammation, defining its gut biota landscape becomes particularly important contributing to the severity of pancreatitis where potential pathobionts and host metabolome. [15] Increased intestinal permeability and subsequent translocation of gut-derived bacteria to pancreatic tissue may play a crucial role in aggravating inflammation, bacteria from the gut to the blood or nearby tissues, such as the pancreas, thereby increasing the risk of infection. [16]

Severe acute pancreatitis involves dynamic interactions between immune dysregulation and inflammatory infiltration, inflammatory injury by the underlying mechanisms by which they exacerbate the inflammatory lesion that is still unclear, which represents a life-threatening gastrointestinal emergency characterized by progressive pancreatic necrosis and dysregulated systemic inflammatory response. [17:18]

Acute pancreatitis is a life-threatening inflammatory disease with well-known etiologies; however, in up to 30% of cases no probable identifiable cause is found, resulting in the diagnosis of being idiopathic; Therefore, food allergy is believed to be an immune-mediated hypersensitivity reaction to specific food antigens, considered in an etiological differential diagnosis by inflammatory cytokines. [19]

OBJECTIVE

To describe the experience of the results obtained from patients with acute pancreatitis with a statistical, deductive, critical, logical and impartial analysis. In addition, to carry out the history of art based on an exhaustive search of the medical literature of national and

international acute pancreatitis and its variants in the medical and/or surgical field in terms of its applicability.

METHOD

It is a study with a retrospective, longitudinal, observational and descriptive design. The presentation of the results was carried out through descriptive biostatistics procedures, where the electronic records and files of all patients with acute pancreatitis of probable origin of biliary and/or pancreatic pathology in the services following endoscopic retrograde cholangiopancreatography are reviewed, at the General Hospital "Dr. Rubén Leñero" of the Ministry of Health of Mexico City. Country Mexico. Classified as a second level of health care.

In a study period that spanned from January 2024 to January 2026. Age, sex, associated factors or comorbidities, type of acute pancreatitis, time of remission of the disease, probable specific origin due to biliary pathology, clinical picture were evaluated. antibiotic therapy, complications, morbidity and mortality. And finally special observations.

RESULTS

There was a total of 81 patients, of which 16 patients (19.75%) were men and 65 women (80.25%), with an average age of 39 years with a bimodal value of 34 and 50 years. The following comorbidities were detected: firstly, overweight in 47 patients, representing 58.02%, secondly, type 2 diabetes mellitus in 9 cases, 11.11%, and thirdly, essential arterial hypertension in 6 individuals, the other comorbidities detected were 12%. See table 1.

Table 1: Chronic-Degenerative Diseases/Factors Expressed in Number and Percentage in Patients with Acute Pancreatitis

Chronic-Degenerative Disease/Factors	Number / %
Bronchial Asthma	1 / 01.23
Acute/Chronic Renal Failure	1 / 01.23
Overweight/Obesity	47 / 58.02
Mellitus Diabetes	9 / 11.11
Systemic Arterial Hypertension	6 / 07.40
Chronic Obstructive Pulmonary Disease	1 / 01.23
Dyslipidemias	1 / 01.23
Others	6 / 07.40
Total	72 / 88.88

It should be clarified that a single patient can suffer from more than one disease, so the indicated values are relative, and therefore their interpretation must be considered with criteria. Together, all chronic-degenerative diseases represent up to 35% of the patients under study, but the total number of cases is only 22 with an adjusted percentage of 24%. Regarding the type of pancreatitis, only those patients who presented acute pancreatitis of probably biliary etiology and/or its unresolved consequences due to different circumstances in the medium or long term were involved. Clinical characteristics and previous treatments and decisions on the conduct to be followed are specified. See table 2.

Table 2: Probable Biliary Etiology of Acute Pancreatitis, Clinical Presentation and Initial Therapeutic Management

Etiological Diagnosis	No	Jaundice	Inflammatory Response Syndrome	Sequelae	Therapeutic Conduct
Acute Lithiasic Cholecystitis	63	7	3	No	Cholecystectomy
Primary Choledocholithiasis	03	03	1	0	Ercp+Cholecystectomy
Secondary Choledocholithiasis	02	02	2	No	Ercp+Cholicecystectomy
Residual Cholidocholithiasis	07	06	2	No	Cholecystectomy+Ercp
After An Ercp	02	0	0	0	Medical Management
After To Sphincterotomy	01	0	0	0	Medical Management
Mirzzi Syndrome	02	02	1	0	Cholecystectomy
Acute Alithiasic Cholecystisti	01	0	1	0	Colecistostomy
Total	81	20	10	0	

Of the cases with acute pancreatitis that were diagnosed with probable biliary etiology, they are composed by listing the possible causes such as Mirizzi syndrome, choledocholithiasis and the most common is acute lithiasis cholecystitis that comprises 77.7%, the second diagnosis by incidence is residual choledocholithiasis 8.64%. Jaundice was present in 24.69 %. However, it is clarified that this clinical sign does not translate as ethereal choledocholithiasis, but as an inflation of the pancreas that compresses the intrapancreatic bile duct. 12.34% had data on systemic inflammatory response syndrome [20] There were no complications or sequelae typical of the previous established management, during acute pancreatitis, in the trans operative and postoperative periods. When acute pancreatitis subsided or remitted, laparoscopic cholecystectomy was performed, which was performed in 71 patients, representing 91.02%, and in 7 cases, representing 8%. 97% performed the conventional technique; when primary or secondary choledocholithiasis was corroborated by imaging, endoscopic retrograde cholangiopancreatography (ERCP) was indicated after acute pancreatitis had ended, or in individuals who underwent cholecystectomy and had residual choledocholithiasis. Only one patient underwent reoperation for bile duct exploration, which was used as a third therapeutic option in a conventional manner. 2 patients with Rendez-vous were documented. Patients who underwent ERCP with an adequate response without complications were only admitted to this study.

The diagnosis of acute pancreatitis was confirmed with three parameters: clinically, elevation of pancreatic enzymes to more than 3 times the baseline value, and by imaging. - With a simple CT scan of the abdomen (there is no radiologist specialized in the area or contrast medium in the hospital).

The management of support or support or medical management is carried out with the criteria of various surgical specialists of the hospital under study, where the diversity is very marked depending on the school and experience of each one, with a diet started in less than 24 hours, without antibiotics, even with jaundice. In some patients even without intravenous solutions, only pain management/control. However, despite a divergent/different and unorthodox treatment, no complications or morbidity are reported due to the natural evolution of the disease due to these therapeutic behaviors mentioned above. Only non-specific morbidity not associated with the underlying pathology was documented. Described in Table 3.

Table 3: Morbidity in Patients with Acute Pancreatitis Expressed in Number and Percentage

Morbidity	Number/%
Pneumonia	1/01.23
Severe Anemia	2/02.46
Mild To Moderate Malnutrition	4/04.93
Urinary Tract Infection	3/03.70
Seroma	2/02.46
Surgical Wound Infection	2/02.46
Upper Gastrointestinal Bleeding	2/02.46
Total	16/19.75

The morbidity associated with patients with acute pancreatitis is reported in 19.75%, and adjusted to 4.93%, since a single case can present two or more morbidities at the same time. The morbid event with the highest incidence was mild to moderate malnutrition, which represented 5%, followed by urinary tract infection with almost 4%. It should be noted that when acute pancreatitis was remitted and the therapeutic course to be followed to avoid recurrence was initiated, patients had the previously specified morbidities; however, there were medium-term complications due to damage or sequelae of pancreatitis, of which three of them were reported and which represented 3.70%:

1. A patient with pancreatic pseudocyst who was surgically treated with the cysto-gastro-anastomosis technique by laparoscopic approach.
2. Another case of infected pancreatic necrosis performing conventional surgical intervention with necrosectomy and continuous cavity lavage by peritoneal system.
3. A third patient who has a pancreatic abscess, with laparoscopic surgical drainage and who later presents pancreatic fistula of low output, and who gave in under conservative treatment. (Sick with initial treatment and referral from another hospital)

No mortality was reported in this group of patients. It should be noted that all acute pancreatitis were classified with the "Revised Atlanta Classification" as mild, moderate, and severe. [21] In this group of patients, 78 patients had mild acute pancreatitis, which accounted for 96.29%, 2 of them as moderate 02.46%, and only 1 case as severe 0.23%.

DISCUSSION

The etiology of acute pancreatitis is to this day a mystery, since there is no proven genesis that causes it. The above only gives theories as to its causal factor. Elevated blood glucose and/or lipid levels trigger the release of several pro-inflammatory mediators, such as tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and monocyte chemoattractant protein-1. [22] Acute hypertriglyceridemia pancreatitis was defined by deduction as having a serum triglyceride level greater than 11.3 mmol/L or 1000 mg/dL at admission, without gallstones or obstruction, alcohol consumption, or other reasons. [13, 23, 24] Acute biliary pancreatitis has been defined as being induced by cholelithiasis or choledocholithiasis combined with abnormal liver function. In addition, biliary microlithiasis induces a local inflammatory reaction in the papilla causing pancreatic inflammatory disease. [25]

Endoscopic retrograde cholangiopancreatography is a tool par excellence for the management of malignant and benign biliary diseases. Post-ERCP pancreatitis is the most common adverse event, affecting between 3.5% and 9.7% of all patients undergoing the procedure. [26] Where precut sphincterotomy and sphincteroplasty were independently associated with a 7.8% increased risk. Post-ERCP pancreatitis remains the main concern. Meta-analyses suggest that incidence rates range from 1.5% to 6%, values consistently lower than those reported in other types of medical literature that can exceed 15% to 20%.[27] In addition, the use of self-expanding, coated metallic biliary stents is associated with an increased risk of post-ERCP pancreatitis compared with patients who did not receive stents or received plastic stents, regardless of the indication or history of prior sphincterotomy. [28] Cholelithiasis with concomitant choledocholithiasis is a common condition in hepatobiliary surgery. Disconnected pancreatic duct syndrome is a rare but serious complication of necrotizing pancreatitis, characterized by disruption of the main pancreatic duct, which separates a viable proximal pancreatic segment from the distal ductal system, resulting in persistent pancreatic secretions and recurrent fluid collections. [29, 30]

Azathioprine is a widely used immunosuppressant, can induce acute pancreatitis, and is generally estimated to be the most common causal of drugs, although it accounts for a relatively small percentage of 2% to 5% of global cases, shows an upward trend in specific populations, and presents significant clinical challenges due to its often-elusive diagnosis and ill-defined mechanisms. [31] On the other hand, there is an origin where genetic pancreatitis is a rare cause of chronic pancreatitis, accounting for 1% of cases. Its genetic origin was suspected as early as 1952 and confirmed in 1996 with the identification of the PRSS1 gene, which predisposes to the disease. Interacting with environmental factors such as alcohol and tobacco consumption. However, the continuous activity of trypsin of genetic etiology leads to a permanent state of acute pancreatitis that will consequently occur in chronicity. [32, 33] IgG4-related autoimmune pancreatitis (IgG4-AIP) has been documented to be a rare autoimmune pancreatic disorder, in which an elevation of carbohydrate antigen 19-9 (CA19-9) is observed in some patients, a chronic systemic fibroinflammatory disorder of immune-mediated origin that can affect multiple organs of the body. [34, 35]

Excessive alcohol consumption often leads to recurrent episodes of acute pancreatitis, which eventually develop into chronic pancreatitis due to addiction. Consequently, alcoholic pancreatitis has seriously impacted patients' quality of life and socioeconomic development, becoming a frequent cause of this disease that oscillates in first and/or second place. [36]

It has been documented that the Atlanta classification for acute pancreatitis is currently the most reliable that focuses on initial behavior/behavior/prognosis, organ failure, and protective measures as central issues for developing or not developing acute respiratory distress syndrome and increasing mortality rates. [37] The patient stratification that continues to be used is the 2012 "Atlanta Revised Classification" and its diagnosis is detailed with typical upper abdominal pain accompanied by serum amylase or lipase levels ≥ 3 times the upper limit of normal and radiological findings with ultrasound, contrast-enhanced computed tomography, or magnetic resonance imaging. [38, 39] Biochemical markers have been stigmatized to determine bacterial infection and sepsis, including procalcitonin, which in all conditions, its elevation of noninfectious types was variable and often overlapped with the ranges reported in bacterial infection, particularly in settings characterized by severe sterile inflammation and tissue damage such as acute pancreatitis.

[40] Several scoring systems such as Ranson, Glasgow (Imrie), BISAP, and APACHE-0, I, and II have been developed to predict disease severity. Current literature suggests an association between laboratory markers of inflammatory response, including C-reactive protein (CRP), white blood cell (WBC) count, and lactate dehydrogenase (LDH) levels, disease activity and clinical course. [41] Among the diagnoses and/or etiology, eosinophilic pancreatitis is extremely difficult to confirm, which represents less than 1% of pancreatitis cases, poses important diagnostic challenges due to its heterogeneous manifestations, ranging from mild to life-threatening. The overlap of clinical symptoms and radiological features with other types of pancreatitis often hinders and the rarity of the disease alone leads to high rates of misdiagnosis in clinical practice. See Figure 1. [42]

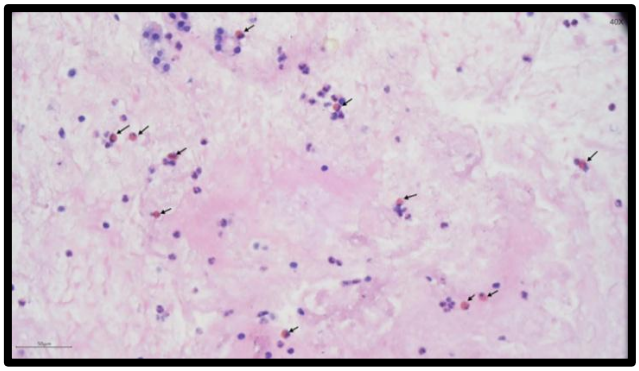

Figure 1: Histopathological analysis of pancreatic tissue revealed eosinophilic infiltration (15/HPF), with eosinophils indicated by arrows (H&E staining; original magnification, 400×). Figure taken from the reference: Li X, Zhang D, Li Y, Zhang K, Li N. Recurrent Eosinophilic Pancreatitis With Eosinophilic Gastroenteritis: A Case Report. Am J Case Rep. 2026 Mar 9;27: e950992. doi: 10.12659/AJCR.950992. PMID: 41801837. [42]

Abdominal pain is one of the most prominent symptoms of acute pancreatitis, it is associated with an increased risk of severe acute pancreatitis and systemic or local complications, the most used analgesics have shown that NSAIDs and opioids are equally effective. [43] The use of opioids with mild acute pancreatitis is documented, although the relative paucity of high-quality trials and data in this setting is notable. [44] The authors differ in the conventional use of opioids in the 1st line due to the various effects already corroborated, such as contraction and increased pressure, simulating acute clinical symptoms due to spasm of the sphincter of Oddi, adding the contraction of the gallbladder, which causes alteration of liver enzymes due to the high pressure of the biliary tree and can cause necrosis of hepatocytes. resulting in an increase in ALT/AST transaminases, alkaline phosphatase and bilirubin, simulating lithiasis obstruction, because on long-term motility. Chronic opioid use may contribute to the dilation of the bile and pancreatic ducts due to their inhibitory effects on motility already described. [45]

Acute pancreatitis is one of the most common gastrointestinal disorders requiring hospitalization, in the scenario of a mild and self-limiting illness, but approximately 20% will develop necrotizing pancreatitis. Mortality rates associated with infected necrosis range from 15% to 35%, compared to 0%-1% for the disease. [46] Two high-quality, double-blind, randomized controlled studies investigating prophylaxis with ciprofloxacin and

metronidazole (n = 114) and meropenem (n = 100) initiated antibiotics early (≤72–120 hours) using strict criteria for predicted or confirmed severe necrotizing pancreatitis, and found no reduction in infected necrosis.[47] However, this high-quality study showed that it was significantly reduced when prophylaxis was applied. Therefore, prophylactic antibiotics are likely to reduce the overall incidence of infectious complications, with greater mitigation of extra pancreatic infections and sepsis than of infection by the same pancreatic necrosis. [48] Another meta-analysis suggested that the cumulative incidence of infectious complications was significantly decreased in the antibiotic prophylaxis group compared with the control group. [49] Acute pancreatitis at its onset behaves sterilely and may mimic a diagnosis of "sterile abdominal sepsis," which results in a systemic inflammatory response, multiorgan failure, and death; this occurs in chemical aseptic peritonitis, which occurs when irritants enter the abdominal cavity. Inflammation of the peritoneal serosa is initially sterile but exponential and is usually fatal. [50]

Of the complications of the acute pancreatic aseptic inflammatory event, 20% progress to acute necrotizing pancreatitis, and about one-third of these develop infected pancreatic necrosis. And only 38% of these patients require intervention. [51] These cases undergoing endoscopic necrosectomy are at risk of bleeding associated with the debridement devices used, and out of 969 procedures in 447 patients, intraoperative bleeding is reported in 175 cases (18.1%). [52] Pancreatic pseudocyst is a well-encapsulated accumulation of peripancreatic fluid that occurs at least 4 weeks after acute pancreatitis ranging from 8% to 41%. The option is an endoscopic ultrasound-guided drainage, which is safe and effective, and is preferable to surgery. [53] Exocrine pancreatic insufficiency, on the other hand, can develop after acute pancreatitis, affecting one in five cases in the long term. Alcoholic etiology and necrosectomy are risk factors for induction. [54] Another connotation is chronic pancreatitis as a repetitive result of a causal or misdiagnosed or defined etiologic factor, estimating that 50% to 90% of patients with chronic pancreatitis may develop it, so lateral pancreatic jejunostomy or the surgical technique called the Frey procedure is the panacea for its treatment. [55] See Figure 2.

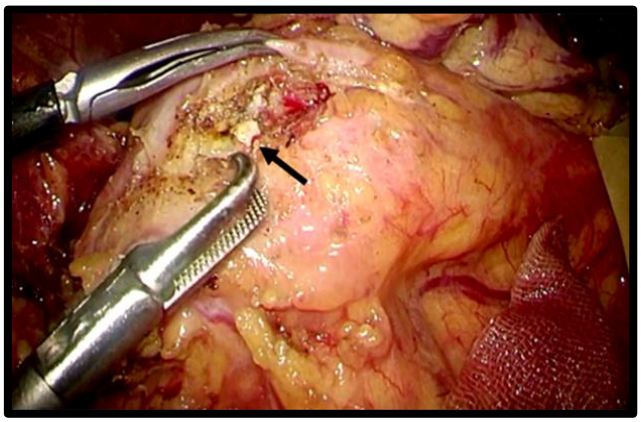

Figure 2: The main pancreatic duct was opened longitudinally on the ventral aspect of the pancreas with an ultrasonic dissector. Ductal stones (black arrow) were removed by opening the pancreatic duct distally and proximally. Figure taken from the reference: Nakajima T, Fukumoto T, Tsukamoto T, Kanazawa A, Kodai S, Mori Y. Laparoscopic Frey's Procedure for Chronic Pancreatitis in a Japanese Patient. Am J Case Rep. 2020 Jun 15;21:e924206. doi: 10.12659/AJCR.924206. PMID: 32541646; PMCID: PMC7319076. [56]

Stones in the pancreatic duct develop in the late stage of chronic pancreatitis. However, in the case of complex stones in the pancreatic duct or stones in the body and tail of the pancreas, endoscopic treatment is of little use and ultimately requires surgical intervention. This, if early, allows effective drainage of the pancreatic juice, delays pancreatic atrophy and protects pancreatic function. [57] Surgery is the cornerstone of pain management in chronic pancreatitis, in patients with a dilated pancreatic duct, with the use of techniques such as lateral pancreatojejunostomy or the surgical technique called Frey's procedure, with a decompressive goal in the pancreatic ducts, by open or laparoscopic techniques. [58] However, in stones in the body and tail of the pancreas, treatment with stones that respond poorly to pharmacotherapy or endoscopic therapy are often ineffective and ultimately require surgical intervention, which a chronic course increases the risk of acute exacerbations of pancreatitis and poses long-term nutritional risks. Another option being extracorporeal shock wave lithotripsy as a common treatment for pancreatic stones in chronic pancreatitis. In contrast, lithotripsy guided by peroral pancreatoscopy: comparatively, extracorporeal plasma lithotripsy yields the same results as extracorporeal shock wave lithotripsy. [59] Another option, which is described, is nerve blocks that can provide short-term pain relief; however, they often require frequent repeated treatments, provide short-term pain relief, do not address the underlying problem, and the pain gradually returns, thus it has fallen into disuse. [60]

SPECIAL OBSERVATIONS

1. Patients who present acute pancreatitis have been transformed over the years, from being a highly morbid pathology, feared and with high mortality rates. Currently its presentation is mild, with a very light, tenuous and rapid clinical course with an average remission of 72 hours.
2. All of the above known for the initial management in cases with mild acute pancreatitis, both supportive or posterior, has lost value at present or has been overestimated, given that today, diet is started (trial-and-error test) if there is non-specific abdominal pain the diet is not given or vice versa, no antibiotic therapy, there is no concern about a high amount of fluids or intravenous fluids, no continuous cardiac monitoring, no strict fluid control, no nasogastric tube, no Foley tube, etc. And despite these basic or crude changes, the results are excellent
3. Multiple laboratory/cabinet studies have become obsolescence in this specific pathology: procalcitonin. - non-specific, C-reactive protein. - It is not specific. Leukocytosis. - are non-specific. Erythrocyte sedimentation rate. - it is not specific etc. The same happens with radiological studies that are dependent on operators such as ultrasonography, specific contrasted computed tomography such as dynamic contrasted pancreatography. These resources are not available and the only thing we have is a simple CT study. And the connotation is made that there is no difference in not having them, since very good results are evident.
4. In patients with acute pancreatitis, no morbidity typical of pancreatic pathology is reported, per se only from elective surgical intervention after cholecystectomy or endoscopic retrograde cholangiopancreatography.

5. The therapeutic approach of performing laparoscopic cholecystectomy or conventional technique as elective surgery before hospital discharge continues, increasing operating costs and use of day bed.
6. Diagnosis in cases of acute pancreatitis has been expedited, thanks to the ease of emergency room physicians. - for fear of reporting omission or error - they carry out "Defensive Medicine" they routinely request amylase and lipase reagents in the blood in any patient with abdominal pain.
7. The previous observation is the high point of the title "evolved" of this manuscript: the answer is to have the infrastructure (laboratory reagents at a lower price-amylase-lipase-) that from the beginning makes the diagnosis together with the clinical picture and some sensitive available radiological study. Managing to identify the onset of the disease early, and not its complications or sequelae or its own morbidity. A situation that was not possible 20 years ago, due to the lack of reagents in public hospitals and/or the costs of the same in private hospitals in Mexico.
8. These two previous numerals 6 and 7 are a synergy to evidence the results obtained in this research. In addition, it is confirmed that the process of acute pancreatitis is aseptic. All the above can be summarized that using evidence-based research (this study), deductive and inductive methodological research, *the current therapy in patients with mild acute pancreatitis is:*
    - A 24- to 48-hour fast
    - Exclusive use of analgesia
    - Daily enzyme monitoring of amylase, lipase and blood count
    - Once the disease has subsided, priority cholecystectomy vs. endoscopic retrograde cholangiography, depending on the case, is completed.
9. This translates into commendable benefits, such as cost reduction, avoiding the promotion of antibiotic resistance, reducing morbidity and zero mortality.

CONCLUSIONS

Acute pancreatitis is a pathology that for centuries has caused fear, brutal health sequelae/limitations to survivors, which has plagued humanity due to such high rates of morbidity and mortality; that in view of this background, overdiagnosis and excessive treatment have been exercised with an increase in the costs of human, material and economic resources, with truly very precarious results.

It is concluded that therapeutic management of mild acute pancreatitis, so basic and unorthodox, is beneficial in medical practice that does benefit the patient and the permanently worn-out impoverished health sector.

Conflict of Interest

The authors stated that they had no potential conflicts of interest regarding the research, authorship, and/or publication of this article.

REFERENCES

1. Talathi SS, Omole AE, Young M. Anatomía, abdomen, pelvis y páncreas. [Actualizado el 13 de diciembre de 2025]. En: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; enero de 2026. Disponible en: https://www.ncbi.nlm.nih.gov/books/NBK532912/
2. Busnardo A, Didio L, Tidrick R & Thomford Neil. History of the Pancreas. Am J Surg. 1983; 146: 539-543
3. Navarro-Soto S. A brief history of the anatomy and physiology of a mysterious and hidden gland called the pancreas. Gastroenterología y Hepatología.Volume 37, Issue 9, November 2014, Pages 527-534.
4. Leach S, Gorelick F. & Modlin I. Acute pancreatitis at its centenary: the contribution of Reginald Fitz. Ann Surg 1990; 212:109-113
5. E. L. Opie, "The Relation of Cholelithiasis to Pancreatic Disease and Fat Necrosis," American Journal of the Medical Sciences 121, No. 1 (1901): 27–42,
6. Chávez Rossell Miguel. Historia del Páncreas y de la evolución de los conceptos y la clasificación de Pancreatitis. Rev. gastroenterol. Perú [Internet]. 2002 Jul [citado 2026 Mar 12]; 22(3): 243-247. Disponible en: http://www.scielo.org.pe/scielo.php?script=sci_arttext&pid=S1022-51292002000300008&lng=es.
7. Wang CF, Tariq A, Chandra S. Pancreatitis aguda. [Actualizado el 2 de agosto de 2025]. En: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; enero de 2026. Disponible en: https://www-ncbi-nlm-nih-gov.translate.goog/books/NBK482468/?_Acute pancreatitis. [Updated August 2, 2025]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; January 2026. Available in: https://www-ncbi-nlm-nih-x_tr_sl=en&_x_tr_tl=es&_x_tr_hl=es&_x_tr_pto=tc
8. Wen L, Mukherjee R, Huang W, Sutton R. Calcium signaling, mitochondria and acute pancreatitis: avenues for therapy. Pancreapedia. 2016. doi:10.3998/panc.2016.15
9. Álvarez-Aguilar Pablo Andrés, Dobles-Ramírez Carlos Tobías. Pancreatitis aguda: fisiopatología y manejo inicial. Acta med. costarricense [Internet]. marzo de 2019 [consultado el 11 de marzo de 2026]; 61(1): 13-21. Disponible en: http://www.scielo.sa.cr/scielo.php?script=sci_arttext&pid=S0001-60022019000100013&lng=en.
10. Li J, Chandrashekar A, Ravikumar R, Afghani E, Singh VK, Akshintala V. Glucagon-like Peptide-1 receptor agonists rarely cause de novo acute pancreatitis but often result in serum lipase elevations of unclear clinical significance. Pancreatology. 2026 Mar;26(2):299-301. doi: 10.1016/j.pan.2025.12.021. Epub 2025 Dec 25. PMID: 41521121.
11. Bai Y, Hu Y, Li L, Qian H, Wang X, Ma Y, Lu F, Qin K, Wang J, Ye Q. The dual role of formyl peptide receptor 1 (FPR1) in gastrointestinal inflammation and Carcinogenesis: Mechanisms and therapeutic implications. Biochem Pharmacol. 2026 Mar 7:117870. doi: 10.1016/j.bcp.2026.117870. Epub ahead of print. PMID: 41802684.
12. Sheng LP, Shang GC, Han CQ, Ling X, Guo XW, Lin R, Ding Z. Impaired Autophagic Flux in Adipose Tissue Aggravates Pancreatic Injury in Obesity-Related Severe Acute Pancreatitis. Immun Inflamm Dis. 2026 Mar;14(3):e70395. doi: 10.1002/iid3.70395. PMID: 41801557; PMCID: PMC12970485.
13. Wang C, Wei S, Tang Q, Deng K, Jin H, Weng H, Zan X, Li Q. Procyanidin Capsules Relieve Inflammation in Hypertriglyceridemic Acute Pancreatitis via Regulating Oxidative Phosphorylation Signaling. ACS Omega. 2026 Feb 16;11(8):13275-13291. doi: 10.1021/acsomega.5c09867. PMID: 41799146; PMCID: PMC12961546.

14. Shkoporov AN, Hill C. Bacteriophages of the Human Gut: The "Known Unknown" of the Microbiome. Cell Host Microbe. 2019 Feb 13;25(2):195-209. doi: 10.1016/j.chom.2019.01.017. PMID: 30763534.

15. Liu J, Yan Q, Li S, Jiao J, Hao Y, Zhang G, Zhang Q, Luo F, Zhang Y, Lv Q, Zhang W, Zhang A, Song H, Xin Y, Ma Y, Owusu L, Ma X, Yin P, Shang D. Integrative metagenomic and metabolomic analyses reveal the potential of gut microbiota to exacerbate acute pancreatitis. NPJ Biofilms Microbiomes. 2024 Mar 21;10(1):29. doi: 10.1038/s41522-024-00499-4. PMID: 38514648; PMCID: PMC10957925.

16. Liu J, Yan Q, Li S, Jiao J, Hao Y, Zhang G, Zhang Q, Luo F, Zhang Y, Lv Q, Zhang W, Zhang A, Song H, Xin Y, Ma Y, Owusu L, Ma X, Yin P, Shang D. Integrative metagenomic and metabolomic analyses reveal the potential of gut microbiota to exacerbate acute pancreatitis. NPJ Biofilms Microbiomes. 2024 Mar 21;10(1):29. doi: 10.1038/s41522-024-00499-4. PMID: 38514648; PMCID: PMC10957925.

17. Zhang H, Wang Z, Lu J, Li J, Jia Y, Xie X, Ding Y, Cao F, Li F. Neutrophil extracellular traps prime the ZBP1-cGAS sensor complex, triggering necroptosis and inflammatory injury in acute pancreatitis. Int J Biol Sci. 2026 Feb 4;22(5):2398-2417. doi: 10.7150/ijbs.122290. PMID: 41800237; PMCID: PMC12965145.

18. Liu M, Wang L, Liu J, Yuan Q, Zhang Y, Wu S, Zhang Y, Guo R, Zhang Y, Lu T, Yan Q, Li S, Xing G, Dong B, Zheng N. Gut virome and metabolic associations in patients with acute pancreatitis. mSystems. 2026 Mar 6:e0140025. doi: 10.1128/msystems.01400-25. Epub ahead of print. PMID: 41789894.

19. Balaban YA, Inan MI, Kalkan F, Sonmez E, Buyukturan G, Demirel F, Selcuk A, Yesillik S, Sakin YS, Kartal O. Can food allergy trigger acute pancreatitis? Exploring an unusual etiology. Allergol Immunopathol (Madr). 2026 Mar 1;54(2):45-50. doi: 10.15586/aei.v54i2.1544. PMID: 41814603.

20. Orsatti VN, Ribeiro VST, de Oliveira Montenegro C, Costa CJ, Raboni EA, Sampaio ER, Michielin F, Gasparetto J, Telles JP, Tuon FF. Sepsis death risk factor score based on systemic inflammatory response syndrome, quick sequential organ failure assessment, and comorbidities. Med Intensiva (Engl Ed). 2024 May;48(5):263-271. doi: 10.1016/j.medine.2024.03.005. Epub 2024 Apr 4. PMID: 38575400.

21. Wang CF, Tariq A, Chandra S. Pancreatitis aguda. [Actualizado el 2 de agosto de 2025]. En: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; enero de 2026-. Disponible en: https://www.ncbi.nlm.nih.gov/books/NBK482468/

22. Al-Mansoori L, Al-Jaber H, Prince MS, Elrayess MA. Role of Inflammatory Cytokines, Growth Factors and Adipokines in Adipogenesis and Insulin Resistance. Inflammation. 2022 Feb;45(1):31-44. doi: 10.1007/s10753-021-01559-z. Epub 2021 Sep 18. PMID: 34536157; PMCID: PMC8449520.

23. De Marchi G, de Pretis N, Capurso G, Pezzilli R, Zambon A, Agnolon C, Conti CB, Gianotti L, Massironi S, Stegagnini M, Galli A, Innocenti T, Apadula L, Giampaolo L, Farinati F, Giannini EG, Checchin D, Giacomin A, Canalis C, Covotta F, Nista EC, Zagari RM, Carrara S, Frulloni L. Clinical outcomes of hypertriglyceridemia-associated acute pancreatitis: a prospective multicenter Italian cohort. Pancreatology. 2026 Mar 10:S1424-3903(26)00124-9. doi: 10.1016/j.pan.2026.03.004. Epub ahead of print. PMID: 41832120.

24. Li JB, Zhu YJ, Liao YJ, Wang JY. Triglyceride-Related Indices and Risk of Acute Pancreatitis: A Prospective Study in UK Biobank. Kaohsiung J Med Sci. 2026 Mar 10:e70189. doi: 10.1002/kjm2.70189. Epub ahead of print. PMID: 41805055.

25. Sirtl S, Ahmad M, Allawadhi P, Metzler T, Buchstab O, Ormanns S, Rudelius M, Beyer G, Krupa L, Staron R, Schulz C, Fahr L, Sendelhofert A, Schulz C, Steiger K, Sendler M, Lerch MM, Regel I,

Żorniak M, Mayerle J, Mahajan UM. NLRP3 Inflammasome Activation-Induced Acute Papillitis as a Trigger of Acute Pancreatitis - A Novel Mechanism of Microlithiasis-Induced Acute Pancreatitis. United European Gastroenterol J. 2026 Mar;14(2): e70198. doi: 10.1002/ueg2.70198. PMID: 41830600.

26. Boškoski I, Costamagna G. How to Prevent Post-Endoscopic Retrograde Cholangiopancreatography Pancreatitis. Gastroenterology. 2020 Jun;158(8):2037-2040. doi: 10.1053/j.gastro.2020.03.019. Epub 2020 Mar 18. PMID: 32197979.
27. Helou M, Steele E, Savu A, Franz A, Liu D, Liu N. Cutting Edge Access: Needle Knife Fistulotomy for Difficult Biliary Cannulation. Curr Gastroenterol Rep. 2026 Mar 9;28(1):13. doi: 10.1007/s11894-026-01038-1. PMID: 41801579; PMCID: PMC12971757.
28. Zhang LY, Tikhe D, Shrigiriwar A, Ermerak G, Lee HH, Sin L, Turner I, Edwards P, Abi-Hanna D, Hourigan L, Kostalas S, Bassan M. Fully covered metal biliary stents confer a higher risk of post-endoscopic retrograde cholangiopancreatography pancreatitis. World J Gastrointest Pharmacol Ther. 2026 Mar 5;17(1):112872. doi: 10.4292/wjgpt.v17.i1.112872. PMID: 41809211; PMCID: PMC12968636.
29. Shi B, Feng J, Hu P, Lv S, Wang B, Wang Q, Fang Z, Wang A, Zhang W, Zhang F. Clinical Outcomes and Safety of Ureteral Catheter-Assisted Antegrade Guidewire Intubation in the Laparoscopic-Endoscopic Rendezvous Procedure for Cholelithiasis with Choledocholithiasis: A Retrospective Cohort Study. Ther Clin Risk Manag. 2026 Jan 8;22:569440. doi: 10.2147/TCRM.S569440. PMID: 41859199; PMCID: PMC12998649.
30. Duarte AF, André A, Trindade CM, Ferreira A, Carapinha J. Disconnected Pancreatic Duct Syndrome: A Rare Complication of Acute Pancreatitis. Cureus. 2026 Feb 16;18(2):e103729. doi: 10.7759/cureus.103729. PMID: 41859611; PMCID: PMC12998971.
31. Xie Z, Lei H, Zhang P, Li Z, Tang W. Unraveling the Link Between Azathioprine and Acute Pancreatitis: Integrating Network Toxicology, Machine Learning, and Mendelian Randomization. CPT Pharmacometrics Syst Pharmacol. 2026 Mar;15(3): e70178. doi: 10.1002/psp4.70178. PMID: 41832938.
32. Rivera Rivera Edgardo D. Pancreatitis, genes y autotrasplante de islotes: actualizaciones y nuevos horizontes. Rev. gastroenterol. Perú [Internet]. 2017 Abr [citado 2026 Mar 25]; 37(2): 156-161. Disponible en: http://www.scielo.org.pe/scielo.php?script=sci_arttext&pid=S1022-51292017000200008&lng=es.
33. Hayashi H, Hamada S, Matsumoto R, Takikawa T, Xu Y, Jie R, Nakasuji H, Abe T, Kikuta K, Motohashi H, Ohmuraya M, Masamune A. Spink1 promotes the survival of Kras-mutant pancreatic acinar cells. Pancreatology. 2026 Mar 19:S1424-3903(26)00134-1. doi: 10.1016/j.pan.2026.03.014. Epub ahead of print. PMID: 41876325.
34. Han DG, Ying CL, Cai ZP, Tong QY, Liu W. Comparative Analysis of Clinical Characteristics in Multi-organ and Single-organ Involvement of IgG4-Related Disease: A Single-center Retrospective Study. Arch Iran Med. 2025 May 1;28(5):303-312. doi: 10.34172/aim.34027. Epub 2025 May 1. PMID: 40751524; PMCID: PMC12305410.
35. Han DG, Li SY, Zhou RT, Tong QY, Liu W. Elevated CA19-9 in IgG4-Related Autoimmune Pancreatitis: A Single-Center Retrospective Cohort Study. Int J Gen Med. 2026 Mar 16;19:596352. doi: 10.2147/IJGM.S596352. PMID: 41867657; PMCID: PMC13003990.
36. Zhang J, Qi Y, Jiang Y, Wei T, Ma S, Fu K, Shen J, Chen C, Li X, Wang G, Leng A, Shang D, Qu J. Qingyi Decoction Alleviates Alcoholic Pancreatitis by Improving Glycerolipid Homeostasis via the AMPK/SREBP-1c/PPARα Pathway. J Inflamm Res. 2026 Mar 17;19:537259. doi: 10.2147/JIR.S537259. PMID: 41867464; PMCID: PMC13005595.

37. Du D, Ma G, Li L, Hao H, Wu L, Zhou B. Bibliometric Analysis of Published Articles on Acute Pancreatitis and Acute Lung Injury from 2004 to 2024. Clin Lab. 2026 Feb 1;72(2). doi: 10.7754/Clin.Lab.2025.250242. PMID: 41670512

38. Zhang L, Cao X, Wang B, Ji P. Optimization of modified CT severity index by incorporating lymphocyte-to-monocyte ratio for early stratification in acute pancreatitis: a single-center validation study. Front Med (Lausanne). 2026 Jan 26;13:1752594. doi: 10.3389/fmed.2026.1752594. PMID: 41669352; PMCID: PMC12883383.

39. Souza GD, Souza LR, Cuenca RM, Jerônimo BS, Souza GM, Vilela VM. UNDERSTANDING THE INTERNATIONAL CONSENSUS FOR ACUTE PANCREATITIS: CLASSIFICATION OF ATLANTA 2012. Arq Bras Cir Dig. 2016 Jul-Sep;29(3):206-210. doi: 10.1590/0102-6720201600030018. PMID: 27759788; PMCID: PMC5074676.

40. Popa SL, Incze V, Ismaiel A, Surdea-Blaga T, Grad S, Turtoi DC, Amarie DS, David L, Brata VD, Leucuta DC, Abdelghafar A, Gherman CD, Zahan MR, Dumitrascu DI. Non-Infectious Causes for Elevated Procalcitonin. Medicina (Kaunas). 2026 Feb 28;62(3):464. doi: 10.3390/medicina62030464. PMID: 41901546; PMCID: PMC13028110.

41. Yalcinkaya G, Caltepe G. Acute Pancreatitis in Children: Retrospective Analysis of 133 Patients. Children (Basel). 2026 Mar 15;13(3):407. doi: 10.3390/children13030407. PMID: 41897119; PMCID: PMC13024780.

42. Li X, Zhang D, Li Y, Zhang K, Li N. Recurrent Eosinophilic Pancreatitis With Eosinophilic Gastroenteritis: A Case Report. Am J Case Rep. 2026 Mar 9;27:e950992. doi: 10.12659/AJCR.950992. PMID: 41801837.

43. Gao L, Zhang R, Xiao Y, Ma C, Li X. High Serum Lipase Level is Associated With Increased Analgesic Use in Hospitalized Patients With Acute Pancreatitis. Pain Res Manag. 2026 Mar 8;2026:6462957. doi: 10.1155/prm/6462957. PMID: 41808845; PMCID: PMC12968432.

44. Cai W, Liu F, Wen Y, Han C, Prasad M, Xia Q, Singh VK, Sutton R, Huang W. Pain Management in Acute Pancreatitis: A Systematic Review and Meta-Analysis of Randomised Controlled Trials. Front Med (Lausanne). 2021 Dec 17;8:782151. doi: 10.3389/fmed.2021.782151. PMID: 34977084; PMCID: PMC8718672.

45. Berger Zoltán, Arcos Mario, Matamala Fernanda, Rojas Claudia. Cólico biliar inducido por morfina. Rev. méd. Chile [Internet]. 2017 Mar [citado 2026 Abr 10] ; 145(3): 406-409. Disponible en: http://www.scielo.cl/scielo.php?script=sci_arttext&pid=S0034-98872017000300018&lng=es. http://dx.doi.org/10.4067/S0034-98872017000300018.

46. Iannuzzi JP, King JA, Leong JH, Quan J, Windsor JW, Tanyingoh D, Coward S, Forbes N, Heitman SJ, Shaheen AA, Swain M, Buie M, Underwood FE, Kaplan GG. Global Incidence of Acute Pancreatitis Is Increasing Over Time: A Systematic Review and Meta-Analysis. Gastroenterology. 2022 Jan;162(1):122-134. doi: 10.1053/j.gastro.2021.09.043. Epub 2021 Sep 25. PMID: 34571026.

47. Pauw HS, Schwarz R, Beij A, Sieswerda E, Voermans RP, van Santvoort HC, van Den Berg FF. Antimicrobial therapy for infected necrotizing pancreatitis: microbiology, antimicrobial resistance and pharmacokinetics. J Antimicrob Chemother. 2026 Mar 4;81(4):dkag092. doi: 10.1093/jac/dkag092. PMID: 41808660; PMCID: PMC13016968.

48. Ding N, Sun YH, Wen LM, Wang JH, Yang JH, Cheng K, Lin H, Chen QL. Assessment of prophylactic antibiotics administration for acute pancreatitis: a meta-analysis of randomized controlled trials. Chin Med J (Engl). 2020 Jan 20;133(2):212-220. doi: 10.1097/CM9.0000000000000603. PMID: 31929369; PMCID: PMC7028185.

49. Jiang X, Shi JY, Wang XY, Hu Y, Cui YF. The impacts of infectious complications on outcomes in acute pancreatitis: a retrospective study. Mil Med Res. 2020 Aug 16;7(1):38. doi: 10.1186/s40779-020-00265-5. PMID: 32799919; PMCID: PMC7429726.

50. García-Hernández, J. L., García-Sánchez, M. A., Méndez-Barrón, G., Suárez, I. A. L., & Cabello, J. de J. U. (2025). Abdominal Sepsis in Surgery and does this "Diagnosis" Really Exist? Effective Surgical Therapy. British Journal of Healthcare and Medical Research, Vol - 12(02). 449-469.

51. Sandra van Brunschot, Janneke van Grinsven, Hjalmar C van Santvoort, et al.,Endoscopic or surgical step-up approach for infected necrotising pancreatitis: a multicentre randomised trial, The Lancet, Volume 391, Issue 10115, 2018, Pages 51-58,ISSN 0140-6736

52. Alsaleh T, George J. Targeted therapeutics for pancreatitis. Front Physiol. 2026 Mar 13;17:1795419. doi: 10.3389/fphys.2026.1795419. PMID: 41907532; PMCID: PMC13021450.Rao J, Yang M, Liu H, Yang K, Wu J, Zhu Y, Xia L, Lei Y, Huang X, Xiong H, Luo L, Liu Z, Shu X, Zhu Y, Lu N, He W. Predictors of bleeding during endoscopic necrosectomy: The critical role of device selection favoring snare monotherapy. Pancreatology. 2026 Mar 25:S1424-3903(26)00133-X. doi: 10.1016/j.pan.2026.03.013. Epub ahead of print. PMID: 41905840.

53. Yaseen A, Salman J, Kadir S, Asim M, Tahseen MU, Zakaria N, Altaf A, Ahmed N, Siyal M, Kakar F, Qureshi S, Niaz SK. Endoscopic ultrasound-guided pancreatic pseudocyst drainage in pediatric patients. World J Gastrointest Pharmacol Ther. 2026 Mar 5;17(1):112068. doi: 10.4292/wjgpt.v17.i1.112068. PMID: 41809209; PMCID: PMC12968623.

54. Eperjesi O, Bucur M, Rancz A, Zolcsák Á, Engh MA, Obeidat M, Szentesi A, Hegyi P, Bunduc S. Risk factors for pancreatic exocrine insufficiency after acute pancreatitis: A systematic review and meta-analysis. Pancreatology. 2026 Mar 3:S1424-3903(26)00121-3. doi: 10.1016/j.pan.2026.03.001. Epub ahead of print. PMID: 41807143.

55. Kumar S, Sharma AK, Tomar L, Ahmad S, Kumar S, Mandal M. Comparative analysis of early outcomes in laparoscopic and open surgery for chronic pancreatitis: a retrospective study. Updates Surg. 2026 Mar 10. doi: 10.1007/s13304-026-02589-8. Epub ahead of print. PMID: 41806173.

56. Nakajima T, Fukumoto T, Tsukamoto T, Kanazawa A, Kodai S, Mori Y. Laparoscopic Frey's Procedure for Chronic Pancreatitis in a Japanese Patient. Am J Case Rep. 2020 Jun 15;21:e924206. doi: 10.12659/AJCR.924206. PMID: 32541646; PMCID: PMC7319076.

57. PARTINGTON PF, ROCHELLE RE. Modified Puestow procedure for retrograde drainage of the pancreatic duct. Ann Surg. 1960 Dec;152(6):1037-43. doi: 10.1097/00000658-196012000-00015. PMID: 13733040; PMCID: PMC1613865.

58. Iwata K, Iwashita T, Mukai T, Iwasa Y, Okuno M, Yoshida K, Maruta A, Uemura S, Yasuda I, Shimizu M. Peroral Pancreatoscopy-Guided Lithotripsy Compared with Extracorporeal Shock Wave Lithotripsy in the Management of Pancreatic Duct Stones in Chronic Pancreatitis: A Multicenter Retrospective Cohort Study. Diagnostics (Basel). 2024 Apr 24;14(9):891. doi: 10.3390/diagnostics14090891. PMID: 38732306; PMCID: PMC11082964.

59. Gou HX, Deng C, Wen Y, Yin ZL, Yang TY, Wang T, Luo H, Cheng L. Pancreatic head resection alongside side-to-side pancreatic duct-jejunostomy for pancreatic stones: A case report and review of literature. World J Gastrointest Surg. 2026 Feb 27;18(2):115072. doi: 10.4240/wjgs.v18.i2.115072. PMID: 41809362; PMCID: PMC12968710.

60. Abdel Aziz AM, Lehman GA. Current treatment options for chronic pancreatitis. Curr Treat Options Gastroenterol. 2007 Oct;10(5):355-68. doi: 10.1007/s11938-007-0036-9. PMID: 17897574.