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# Pancreatic ascites: update on diagnosis and management

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**Abstract**

Pancreatic ascites is a rare condition characterized by the accumulation of high-amylase ascitic fluid in the peritoneal cavity. This condition is often associated with chronic pancreatitis, pancreatic trauma, or pseudocyst rupture. Because of its rarity and ill-defined clinical presentation, pancreatic ascites is often a diagnostic and therapeutic challenge in clinical practice. The current diagnostic criteria include an amylase level >1000 mg/dL, a protein level >3 g/dL, and a serum ascites albumin gradient <1.1 g/dL. The clinical features vary, but may include progressive abdominal distension, diffuse abdominal pain, weight loss and peritonitis. The management of pancreatic ascites remains controversial, and there is no consensus regarding the optimal approach. Conservative medical management, which includes nutritional support, pain control, therapeutic paracentesis and the use of somatostatin analogs, has been associated with a high failure rate and significant morbidity. Interventional therapies, such as surgery and endoscopic transpapillary stenting, have shown more promising outcomes. However, the choice between these methods is still debated, with some advocating for endoscopic approaches, because of their minimally invasive nature and reduced morbidity compared with surgical options. Endoscopic approaches remain underutilized in practice, probably because of the need for repeated interventions, the potential risks associated with endoscopic retrograde cholangiopancreatography, or a lack of skilled personnel. Although they show significant perioperative morbidity and mortality, surgical options provide definitive resolution of pancreatic ascites. Herein, we provide an updated review of pancreatic ascites, highlighting advances in diagnostic techniques and therapeutic approaches, and summarizing insights from recent clinical cases and retrospective studies.

**Keywords** Pancreatic ascites, chronic pancreatitis, duct disruption, pseudocyst, endoscopic stenting

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## Introduction

Pancreatic ascites is a rare form of ascites characterized by the accumulation of fluid in the peritoneal cavity as a result of pancreatic disease [1-4]. This condition is typically associated with the disruption of the pancreatic duct or rupture of a pancreatic pseudocyst, leading to the leakage of pancreatic enzymes into the peritoneal cavity [3,5]. Pancreatic ascites is characterized by high levels of amylase enzyme, typically exceeding 1000 U/L, and protein concentration >3 g/dL [1,6]. Patients may present with abdominal distension, pain and weight loss, although some cases may be asymptomatic [3,6,7].

Pancreatic ascites is a diagnostic challenge, given its rarity and ill-defined clinical features [8]. Its management is also disputed and controversial, as a conservative approach is associated with high failure rates, while endoscopic and surgical options carry significant perioperative morbidity [2-4,9]. There are no well-defined, standardized management guidelines for pancreatic ascites, and existing recommendations are often based on limited data [10,11]. Herein, we provide an updated review of pancreatic ascites, with a focus on diagnostic techniques and therapeutic approaches, and we summarize insights from recent clinical cases and retrospective studies.

Conflict of Interest: None

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## Epidemiology

Pancreatic ascites is a form of non-malignant exudative ascites and is a dreaded complication of pancreatic pathologies [1,2,12]. Data on its epidemiology in the US are

heterogeneous, but most studies estimate that it accounts for 0.4-1.0% of all ascites cases [4,11,13]. A higher prevalence of 3.2% has been observed in China, probably because of a higher burden of pancreatic disease and a large population [1]. Pancreatic ascites is more commonly seen in young to middle-aged adults, typically those aged 20-50 years, reflecting the age distribution of chronic pancreatitis and pancreatic cancer [4,9]. Sex-related differences also exist, showing a higher prevalence in males compared to females [11]. This finding is probably due to the higher incidence of pancreatic disease in males. Chronic pancreatitis, which is often linked to chronic alcohol use, is more prevalent in men, thereby contributing to the higher incidence of pancreatic ascites in this demographic [14]. In a targeted review of published cases between 1975 and 2000, Gómez-Cerezo *et al* [6] identified 139 patients with pancreatic ascites, of whom 104 (74.8%) were male and 35 (25.2%) were female. The patients' ages ranged from 3 months to 76 years, with an average age of 41 years. Individual cases continue to be documented in the literature, probably because of the evolving definitions and more advanced diagnostic techniques.

### Etiopathogenesis

Pancreatic ascites results from the abnormal accumulation of pancreatic fluid within the peritoneal cavity, often secondary to pancreatic ductal disruption or pseudocyst leakage [6,8]. Approximately 4% of patients with chronic pancreatitis and 6-14% of patients with a pancreatic pseudocyst develop this condition [4,6,15]. Severe acute pancreatitis or blunt abdominal trauma can also cause duct dehiscence, leading to pancreatic ascites [10,15,16]. Other etiologies include congenital obstruction of pancreatic ducts, ampullary stenosis, pancreatic stones, and malignancy [4,8,16-18]. Sporadic cases arising from iatrogenic causes, such as endoscopic ultrasound (EUS)-guided fine needle aspiration (FNA), intraoperative FNA biopsy, and endoscopic retrograde cholangiopancreatography (ERCP), have also been observed [10,19,20].

In acute pancreatitis, ductal dysfunction is usually seen in the necrotizing form, where necrosis of ductal epithelial cells leads to malfunction of the pancreatic duct [6,10,16,21]. In chronic pancreatitis, ductal obstruction and elevated intraductal pressure predispose patients to duct ruptures [6,8]. Leakage from these disrupted ducts or pseudocysts allows pancreatic enzymes, rich in amylase and lipase, to infiltrate the peritoneal cavity, causing an inflammatory response that facilitates fluid accumulation [6,8,22]. Enzymatic activity plays a significant role in the pathophysiology, as pancreatic enzymes within the ascitic fluid induce peritoneal inflammation, leading to increased vascular permeability and continuous fluid leakage [6,10,16]. This is further compounded by impaired lymphatic drainage, which contributes to the persistent accumulation of enzyme-rich fluid in the abdominal cavity. The ongoing release of pancreatic enzymes can also lead to autodigestion and peritoneal irritation, escalating local inflammation and promoting fibrotic changes within the peritoneum. Pancreatic trauma can also contribute to pancreatic ascites through acute pancreatic ductal ruptures [18].

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### Clinical manifestations

Pancreatic ascites typically presents with nonspecific abdominal symptoms, which may delay diagnosis. Patients often report progressive abdominal distension due to fluid accumulation, diffuse abdominal pain and discomfort [2,8,10,13,23]. Ascitic fluid leakage from the pancreas can provoke peritoneal irritation, further contributing to abdominal pain [4,24]. Patients may be pain-free, or may experience sharp abdominal pain in the epigastrium, which can range in severity from mild to severe [4,24]. In addition, patients may exhibit anorexia, emesis, early satiety and weight loss, because of compromised digestive function and persistent inflammation [2,11,23].

Dyspnea may also develop secondary to diaphragmatic elevation, caused by extensive peritoneal fluid accumulation or concomitant pancreatic pleural effusions [11,25]. Montemuro and Roy [13] described a unique case of pancreatic ascites in a male patient with a history of cirrhosis secondary to chronic hepatitis C infection and alcohol abuse. The patient presented with progressive dyspnea on exertion and refractory pancreatic ascites due to disruption of the main pancreatic duct. The patient underwent endoscopic stenting and showed improvement in symptoms and resolution of duct leakage. Physical examination findings in pancreatic ascites include abdominal tenderness, shifting dullness and fluid wave on percussion, indicative of free fluid within the peritoneal cavity [5,9,11,13,24,26,27]. Given its retroperitoneal location and proximity to other visceral organs, pancreatic duct disruption can also lead to pleural effusions, peritonitis and fistulas [6,22,28,29]. Pancreatic disease has also been shown to result in vascular complications, such as venous thrombosis and arterial aneurysms [2,28,30].

## Diagnosis

Pancreatic ascites is a diagnostic challenge because of its rarity and nonspecific clinical features. Therefore, a comprehensive diagnostic approach that incorporates a history of the presenting illness, physical examination, blood tests, imaging, and diagnostic paracentesis with fluid analysis, is crucial. As noted above, the physical findings of ascites may include cachexia, abdominal tenderness, peritonitis, abdominal

distension, shifting dullness, positive fluid wave, decreased bowel sounds and extremity edema [9-11,13,27,31,32]. Patients with concomitant pleural effusion may exhibit decreased breath sounds, crackles or rales, and in severe cases may be in respiratory distress [13].

### Laboratory tests

Routine blood tests, including a complete metabolic panel, brain natriuretic peptide level, lactic acid, uric acid and complete blood cell count, can help clinicians narrow down the differential diagnosis of new-onset ascites [4,11]. Hyponatremia is the most common electrolyte abnormality observed in pancreatic ascites cases and is likely to be dilutional, due to fluid retention [8-10]. Hypoalbuminemia is also reported in most cases, and is often secondary to severe malnutrition in the setting of chronic pancreatitis or pancreatic ascites [9,10,25,33].

On complete blood cell count, patients typically show low hemoglobin with a normal to mildly elevated white blood count, even in the absence of an underlying infection [7,10,11,31]. Platelet count and coagulation factors, such as prothrombin time, partial thromboplastin time and international normalized ratio, are usually normal [11,12]. C-reactive protein and erythrocyte sedimentation rate are typically elevated, especially in patients with chronic pancreatitis. Serum lipase and amylase levels are often normal or mildly elevated in pancreatic ascites, thereby rendering these tests non-diagnostic [7,10,23].

### Diagnostic paracentesis

Paracentesis is considered the gold standard for diagnosing pancreatic ascites and has a therapeutic effect on patients. The diagnostic criteria for pancreatic ascites include an amylase concentration >1000 mg/dL, a protein level of >3 g/dL, and a serum ascites albumin gradient (SAAG) <1.1 g/dL [5,6,8,10,13,29]. First proposed by Dr. John C. Hoefs in 1981, SAAG can help clinicians determine whether ascites is due to portal hypertension or to other causes [1,8,34]. The SAAG is calculated by subtracting the ascitic albumin concentration from the serum concentration [4]. SAAG >1.1 g/dL is transudative in nature and indicates portal hypertension etiology, which may include cirrhosis, heart failure, portal vein occlusion, Budd-Chiari syndrome and hepatic sinusoidal obstruction syndrome [1,5,8]. In contrast, an SAAG <1.1 g/dL is considered exudative, insidious, and often due to peritoneal disease [32]. Common causes include infectious peritonitis, chylous ascites, malignancy, nephrotic syndrome and pancreatic ascites [1,32,34]. However, SAAG <1.1 g/dL alone is not diagnostic for either condition.

Similarly to amylase and albumin levels, lipase enzyme level tends to be elevated in pancreatic ascites [25,29,34]. Lactate dehydrogenase is typically elevated in pancreatic ascites; however, it can also be elevated in malignant ascites and tuberculous peritonitis [1,8,10]. Tumor markers such as alpha-fetoprotein, carcinoembryonic antigen, and cancer antigen 125 help rule out peritoneal carcinomatosis and malignancy [1,4]. Fluid cytology and culture are usually negative in pancreatic ascites, particularly in the absence of abscesses or infected pseudocysts [11,26,32]. Pancreatic ascites is typically straw-colored in appearance, although isolated cases of black or bloody ascites have also been reported [5,32,34].

### Diagnostic imaging

Imaging studies play a pivotal role in identifying the source of pancreatic leaks and assessing ductal integrity. Contrast-enhanced computed tomography of the abdomen and pelvis is often the initial modality, revealing peripancreatic fluid collections, ductal dilatation, or pseudocysts [4,7,16,19,32] (Fig. 1, 2). Abdominal ultrasound plays a supportive yet limited role in the diagnosis of pancreatic ascites. It is often used at the point of first contact to rule out biliary pathology, or during diagnostic paracentesis [3,4,12,13]. Doppler ultrasound can also be performed if there is concern about venous thrombosis or hypercoagulability [2].

Magnetic resonance cholangiopancreatography is a noninvasive approach for visualizing ductal disruptions

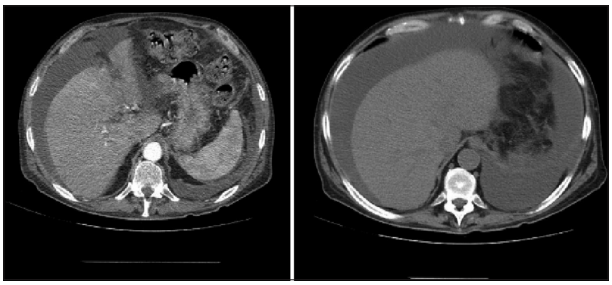

**Figure 1** A 67-year-old man was admitted to our hospital for severe necrotizing pancreatitis with pancreatic ascites and pleural effusion. Contrast-enhanced computed tomography of the chest, abdomen, and pelvis showing diffuse pancreatic ascites (axial view)

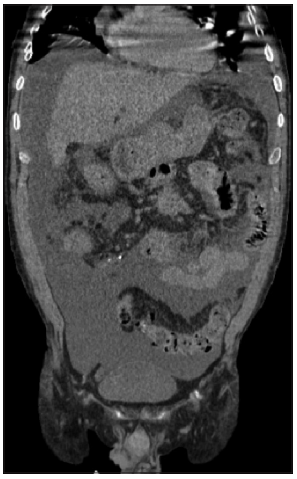

**Figure 2** Contrast-enhanced computed tomography of the chest, abdomen, and pelvis showing complex pancreatic ascites (coronal view)

or leaks [13,35]. ERCP remains the gold standard for ductal imaging, allowing both diagnostic and therapeutic interventions; however, it carries a risk for biliary sepsis and pancreatitis [4,5,10,17]. In practice, ERCP is particularly valuable in cases where pancreatic ductal disruptions or pseudocyst leakage are suspected. By injecting contrast into the pancreatic duct, ERCP can visualize ductal anatomy in high resolution, allowing for precise identification of leaks, strictures or disruptions [3,17]. In chronic pancreatitis, ERCP with a pancreatogram may reveal areas of ductal irregularity, strictures or calculi, further elucidating the pathophysiology of the ascites [17,22,36]. Notably, patients with chronic pancreatitis are often difficult to cannulate, because of calcification and fibrous stenosis of the pancreatic duct [29].

### EUS

EUS is a valuable diagnostic tool for evaluating pancreatic ascites and broader pancreatic pathology. It provides high-resolution imaging of the pancreas, allowing for detailed assessment of the pancreatic parenchyma, ductal anatomy, and surrounding structures [37-39]. This precision makes EUS particularly useful for identifying subtle ductal leaks or disruptions contributing to pancreatic ascites, which may be challenging to detect on conventional imaging [38]. In addition to imaging, EUS can be coupled with FNA, permitting cytological analysis of pancreatic lesions and pseudocysts, which can help differentiate benign from malignant pathology [39]. EUS-FNA also enables fluid sampling from suspected pancreatic collections, allowing for amylase level measurement to confirm the pancreatic origin of ascitic fluid [38,39].

Although EUS is not a first-line modality in every case of pancreatic ascites, its combination of diagnostic and tissue-sampling capabilities makes it invaluable in complex cases, or when standard imaging fails to yield a conclusive diagnosis [39]. Recently, researchers demonstrated the superiority of EUS augmented with deep learning-based models in evaluating solid pancreatic masses compared with EUS alone [39]. This innovation may provide a new frontier for the prompt diagnosis of pancreatic diseases, including ascites. The American Gastroenterological Association recommends EUS as the preferred diagnostic test for unexplained acute and recurrent pancreatitis, which can be associated with pancreatic ascites [36].

EUS and EUS-FNA are generally safe procedures, but are associated with certain risks. The most common adverse events are abdominal pain, pancreatitis and intracystic hemorrhage [40]. Other potential complications include infection, bleeding and perforation, with the risk of perforation being relatively low but noteworthy [41,42]. Lu *et al* [43] reported a case of duodenal perforation during EUS-FNA of a pancreatic head lesion in an older adult. The perforation was successfully managed with endoscopic suturing, endoclips, and aspiration of tissues and ascites. When nonsteroidal anti-inflammatory drugs (NSAIDs) and antibiotics are used in conjunction with EUS, additional adverse events, such as renal impairment, hypersensitivity reactions and allergic reactions, should be considered [40,44].

## Management and outcomes

Prompt and effective management of pancreatic ascites is crucial to prevent complications and improve patient outcomes. However, an evidence-based treatment approach has yet to be established, given the low prevalence and high mortality rates [6,10,12]. Depending on patient-specific factors, provider preference or the availability of local expertise, pancreatic ascites and its associated complications can be managed medically, or with invasive techniques, such as pancreatic duct stenting, pseudocyst drainage or pancreatectomy.

### Medical management

Medical management of pancreatic ascites involves a multimodal approach, including nutritional support, pain control, pharmacologic therapy and fluid management. Nutritional optimization is critical in patients with pancreatic ascites, as they often experience malabsorption and weight loss. Initiating a low-fat diet through enteral feeding reduces pancreatic exocrine stimulation, while in severe cases total parenteral nutrition may be indicated to rest the pancreas entirely [4,6,10,29,35]. Analgesics are central to symptomatic management, with a focus on non-opioid options to reduce the risk of narcotic dependence and complications [4]. For patients with persistent pain, adjunctive therapies, such as nerve blocks, may be considered [36]. Alanís Naranjo *et al* [32] reported a unique case of black ascitic fluid in a male patient with a history of alcohol abuse and chronic pancreatitis. The patient was found to have pancreatic ascites on further investigation and was managed conservatively with analgesics and bowel rest.

Somatostatin analogs, such as octreotide, are commonly used to reduce pancreatic exocrine secretion and thus decrease ascitic fluid production [13]. By inhibiting pancreatic secretion, octreotide may limit further leakage of enzyme-rich fluid into the peritoneal cavity, providing a symptomatic benefit and often reducing the need for repeated paracentesis [5]. Octreotide is typically administered as a continuous infusion, followed by subcutaneous injection, although monthly intramuscular injections are also available [10,24,45]. Its efficacy and the optimal dosing and duration of treatment are yet to be established, as current studies have yielded a variety of data [6]. Diuretics, such as spironolactone or furosemide, may help manage fluid overload in patients with significant ascites, although their efficacy in pancreatic ascites is limited [13,32]. High doses can also cause intravascular volume depletion and renal dysfunction. Diuretic therapy is generally reserved for patients with concurrent cirrhosis or portal hypertension [13].

Therapeutic paracentesis remains a cornerstone of symptomatic relief, particularly in patients with large-

volume ascites causing discomfort or respiratory compromise [4,8,10,15]. Although paracentesis is effective for immediate symptom relief, it does not address the underlying ductal disruption and may need to be repeated [24]. Combining paracentesis with somatostatin analogs may prolong the interval between procedures. Although effective in mild to asymptomatic cases, conservative treatment of pancreatic ascites has a failure rate of 40-60% and is associated with high mortality [3,10-12]. It is also costly and involves prolonged hospitalization, as current wisdom advises waiting 3-4 weeks before considering alternative treatments [3,6,8]. Although a cutoff of 3 weeks appears arbitrary, O'Toole *et al* [29] argued that it is the optimal duration for nutritional therapy and octreotide treatment to take effect. Long-term total parenteral nutrition carries risks, including infection and liver dysfunction, and may not be feasible for all patients [9,29]. In patients with severe hyponatremia, very low albumin levels and multiple sites of pancreatic duct disruption, conservative therapy has a higher chance of failure [9].

## Endoscopic interventions

In pancreatic ascites, endoscopic therapies can be used as an adjunct to medical therapy, salvage intervention after failed conservative treatment, or as a primary intervention [4,24]. The endoscopic approach primarily involves the use of ERCP with transpapillary pancreatic duct stenting [8,12] (Fig. 3). This intervention aims to decompress the pancreatic ductal system and facilitate the resolution of ascites [8]. The procedure typically involves performing a pancreatic sphincterotomy followed by the placement of a pancreatic stent, usually 5F or 7F in size, to bridge the site of ductal disruption [3,16,21].

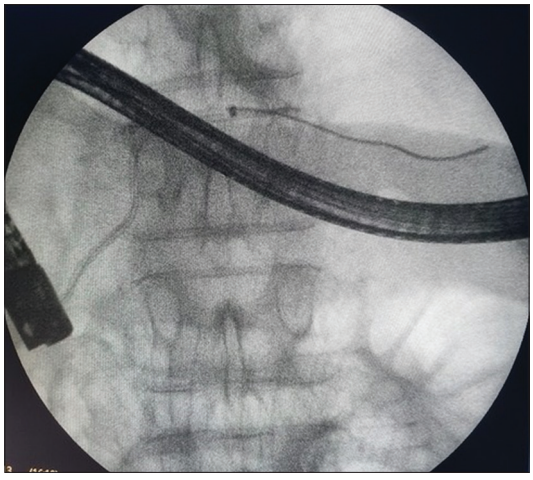

**Figure 3** Endoscopic retrograde cholangiopancreatography: endoscopic transpapillary stent placement in the main pancreatic duct. Adapted from [46]



The sphincterotomy reduces the pancreatic duct's outflow resistance, decreasing ductal pressure and facilitating internal drainage of pancreatic secretions [6,9]. Stent placement across the site of ductal disruption further promotes ductal healing, allowing continuous drainage of pancreatic fluid into the duodenum and reducing ascitic fluid accumulation [12,16] (Fig. 4). This approach is effective in reducing recurrence rates and often eliminates the need for repeated paracentesis. A study by Bracher *et al* [3] demonstrated that transpapillary pancreatic duct stenting effectively resolved pancreatic ascites in 88% of patients within 6 weeks, with no recurrence at a mean follow up of 14 months. No immediate or long-term complications were reported. In cases in which standard plastic stents are insufficient, expandable metallic stents may be considered, particularly in patients who are poor surgical candidates. Generally, somatostatin or octreotide is not required after endoscopic therapy.

Although ERCP is generally well tolerated, it can induce pancreatic inflammation, especially in patients with pre-existing pancreatic disease [10,17]. Given these potential risks, ERCP is often reserved for cases in which imaging strongly suggests a pancreatic leak, while less invasive diagnostic modalities are inconclusive. Aggressive hydration with lactated ringer solution, rectal administration of NSAIDs, or prophylactic pancreatic duct stenting can lower the incidence of post-ERCP pancreatitis [17,44]. Nevertheless, the optimal stent duration has yet to be established, because of the low prevalence of pancreatic ascites and the lack of prospective studies [28,30,47]. In most reported cases, plastic stents were removed or exchanged after 1-6 months or after the resolution of pancreatic ascites [3,5,8,35,36]. In patients with complete pancreatic duct disruption due to acute necrotizing

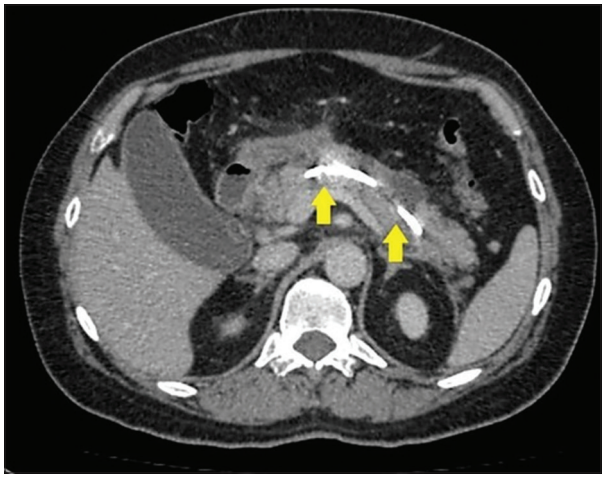

**Figure 4** Computed tomography image after endoscopic transpapillary stent placement in the main pancreatic duct in patients with disconnected main pancreatic duct syndrome (stents in the main pancreatic duct and common hepatic duct are indicated by arrows). Adapted from [46]

pancreatitis, Rana *et al* [16] recommend leaving the stents *in situ* indefinitely. These patients are likely to need close follow up, given the potential risk of biliary sepsis, stent migration, ductal or luminal perforation, bleeding and pancreatitis [48].

In cases involving pseudocysts that communicate with the pancreatic duct, endoscopic cystogastrostomy is a valuable intervention. By creating a direct anastomosis between the pseudocyst and gastric lumen, cystogastrostomy allows for the drainage of cystic contents, reducing pressure and promoting the resolution of ascites [4,27]. EUS guidance enhances the safety and precision of this procedure by enabling the visualization of vascular structures, minimizing procedural risks [37]. EUS is associated with a low risk of luminal perforation, bacteremia and tumor seeding, and requires a skilled endoscopist to optimize outcomes and minimize complications [41].

### Surgical options

Surgical management of pancreatic ascites is considered when endoscopic and medical treatments fail, or when significant pancreatic ductal disruption persists, requiring definitive intervention [4]. Preoperative ERCP is associated with better surgical outcomes, as surgeons can locate the site of ductal disruption beforehand [4,6]. Surgical cystogastrostomy involves creating an anastomosis between a pancreatic pseudocyst and the posterior wall of the stomach, allowing direct drainage of cystic contents into the gastric lumen [49]. This procedure effectively reduces intraductal pressure and prevents further leakage of pancreatic fluid into the peritoneal cavity, thereby promoting resolution of the ascites.

Roux-en-Y cystojejunostomy is another option, particularly in patients with complex or distal pseudocysts [15]. In this approach, a jejunal limb is connected to the pseudocyst, creating a drainage pathway into the small intestine and thereby reducing the risk of pancreatic fluid accumulation [32]. Roux-en-Y pancreaticojejunostomy is a variant technique used in cases where direct pancreatic ductal disruption requires decompression; it involves anastomosing the pancreatic duct to the jejunum to reroute pancreatic secretions [6,50,51]. In a prospective cohort study of patients with chronic pancreatitis, 8/14 underwent lateral pancreaticojejunostomy as the initial therapy for pancreatic ascites [15]. This surgical technique was noted to be superior compared to other surgical approaches.

Surgical cystoduodenostomy, a less commonly used procedure, creates a drainage route between a pseudocyst and the duodenum, serving as an alternative in select cases based on anatomical considerations [4,52]. Pediatric surgeons at Boston Children's Hospital successfully performed transduodenal cystoduodenostomy in 2 pediatric patients with chronic pancreatitis complicated by pseudocysts [52]. Similarly, cystojejunostomy may be used in patients with pseudocysts that are not amenable to gastric drainage, providing an effective drainage route into the jejunum [4]. In severe or refractory cases, where significant pancreatic pathology or necrosis is present, surgical resection of the affected portion of the pancreas may be indicated. Pancreatic resection is a more radical approach, and is typically reserved for patients with chronic or extensive ductal damage who are unresponsive to decompressive procedures [8,53].

Surgical interventions for pancreatic ascites involve major procedures that may lead to complications, including infection, anastomotic leaks, fistulae and delayed gastric emptying [9,10,12,51,53,54]. These surgeries also carry the risk of pancreatic endocrine and exocrine insufficiency, particularly in the case of pancreatic resection, which can result in long-term digestive and glycemic control issues. Additionally, the invasiveness of these procedures means that their use is limited to patients who can tolerate major surgery, and they may not be suitable for those with advanced disease or poor general health. Finally, a laparoscopic approach requires a higher level of manual dexterity and technical expertise, limiting its use to high-volume academic centers [50]. The choice of surgical procedure depends on the specific anatomical and pathological findings of each patient. The American Pancreatic Association and the International Association of Pancreatology recommend considering surgical intervention when conservative management fails, emphasizing the importance of individualized treatment plans [55].

## Concluding remarks

Treatment protocols for the management of pancreatic ascites are still being investigated, in view of its rarity. However, prompt diagnosis and early intervention are crucial in order to achieve better patient outcomes. Based on this extensive literature review, we deduced that pancreatic endotherapy is the safest and most effective intervention for pancreatic ascites. This was demonstrated through a series of cohort studies and a targeted review of published cases. Therefore, we propose that this modality should be considered as initial therapy for pancreatic ascites. Conservative approaches entailing nutritional therapy, the use of octreotide, diuretics and therapeutic paracentesis, have high failure and mortality rates. Surgical interventions, on the other hand, carry significant perioperative morbidity and mortality and should be reserved for refractory cases only.

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