

Pancreatic Ascites in a Patient with Severe Acute Pancreatitis: An Unusual Case

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Abstract

Background: Pancreatic ascites is an uncommon clinical entity resulting from the accumulation of pancreatic fluid in the peritoneal cavity. It is caused by the leakage from a pancreatic pseudocyst or injury to the pancreatic duct. Diagnosis is based on elevated amylase levels in ascitic fluid (greater than 1000 U/L) and protein levels above 2.5 g/dL. Chronic pancreatitis (83%), acute pancreatitis (8.6%), and trauma (3.6%) are the most common causes of pancreatic duct disruption. **Case Summary:** We report the case of a 59-year-old male patient referred to our institution for severe acute pancreatitis of biliary etiology, complicated by infected encapsulated necrosis. Necrosis extended through the left paracolic gutter to the pelvis and left inguinal region, with additional subcapsular hepatic fluid collections involving segments VI and VIII, requiring both endoscopic and percutaneous management. During clinical follow-up, the patient developed grade 3 ascites, for which he underwent two diagnostic and therapeutic paracenteses. Analysis of the fluid was consistent with pancreatic ascites.

Conclusions: Patients with pancreatic ascites represent a small and heterogeneous population. Early detection is crucial due to the prognostic and therapeutic implications of this finding. Mild cases typically respond to medical management, while more severe cases require endoscopic treatment, with surgical intervention being rarely necessary.

Keywords

Pancreatitis, ascites, biliary tract, pancreatic duct, surgery.

INTRODUCTION

Pancreatic ascites is an uncommon clinical complication caused by persistent leakage of pancreatic secretions into the peritoneum due to pancreatic duct injury. The epidemiology of this condition remains poorly understood and has primarily been reported in retrospective studies and case series⁽¹⁾. Males account for the majority of cases (75%-85%), with the mean age of onset in the fifth decade of life⁽²⁾.

The presence of ascitic fluid is characterized through imaging, and its volume has been associated with clinical outcomes⁽³⁾. Analysis of ascitic fluid—measuring amylase,

protein, and albumin gradient—is indicated for diagnosing pancreatic ascites⁽⁴⁾. Gram stain and culture are also essential to detect superimposed infection. While peritoneal fluid lipase measurement has been described in some cases, there is no standardized protocol or sufficient evidence to support its routine use⁽⁵⁻⁷⁾.

The severity of this condition varies widely depending on the location and extent of ductal injury, as well as the presence of infected fluid. Most mild cases resolve spontaneously; however, persistent pancreatic ascites—with or without infected fluid—is associated with significant morbidity⁽⁸⁾.

Treatment for mild disease without visible pancreatic duct abnormalities on pancreatogram may involve medical management, including total parenteral or nasojejunal nutrition and octreotide or somatostatin. More severe cases with visible ductal abnormalities require endoscopic intervention with transpapillary stent placement. Surgical pancreatectomy is rarely needed as rescue therapy. However, due to the low incidence, comparative studies between available treatment modalities are lacking^(9,10).

We present the case of a 59-year-old male admitted to our institution with clinical findings consistent with severe acute biliary pancreatitis. Local complications included walled-off necrosis with dissecting pancreatic collections showing signs of infection, as well as paracolic gutter, pelvic, and subhepatic collections. Initial management involved endoscopic cystogastrostomy and percutaneous drainage by interventional radiology. During hospitalization, he developed grade 3 ascites, requiring two therapeutic paracenteses with cytochemical analysis confirming pancreatic ascites.

CASE REPORT

A 59-year-old male patient, initially treated at an outside facility for severe biliary pancreatitis with documented local complications including walled-off necrosis and extraperitoneal dissecting collections, was referred to our institution for interdisciplinary evaluation due to case complexity. Repeat imaging revealed a pancreatic collection measuring $123 \times 119 \times 163$ mm (L \times AP \times T) with a volume of 1240 cm³, consistent with infected encapsulated necrosis extending through the left paracolic gutter to the pelvis and left inguinal region, along with subcapsular hepatic fluid collections in segments VII and VIII (Figure 1). The patient underwent cystogastrostomy, which identified a giant infected pancreatic cystic lesion. A metal stent was placed, yielding copious purulent drainage filling the entire gastric fundus. Cultures confirmed penicillinase-hyperproducing *Escherichia coli*. Subsequent intraperitoneal collection drainage was performed by interventional radiology.

During hospitalization, the patient developed grade 3 ascites requiring diagnostic and therapeutic paracentesis with 4000 cc drainage. Ascitic fluid analysis showed amylase 9579 U/L, total protein 2.69 g/dL, and albumin gradient <1.1 g/dL (Table 1), confirming pancreatic ascites. Given high suspicion for pancreatic duct injury based on ascitic findings, total parenteral nutrition was initiated followed by magnetic resonance cholangiopancreatography (MRCP).

This revealed a poorly defined peripancreatic collection adjacent to the body and tail with partial cystic components. T1/T2 hypointense areas suggested necrotic foci (55 cm³ volume, increased from prior imaging) without identifiable leak. Due to persistent ascites requiring repeat paracentesis

and absence of endoscopically treatable duct disruption, video-assisted retroperitoneal debridement was performed. The procedure removed walled-off necrotic tissue involving 30% of proximal pancreas, with laparoscopic drainage of 2000 cc retroperitoneal fluid. Postoperatively, the patient showed marked clinical improvement without ascites recurrence and tolerated oral intake well with absent abdominal pain.

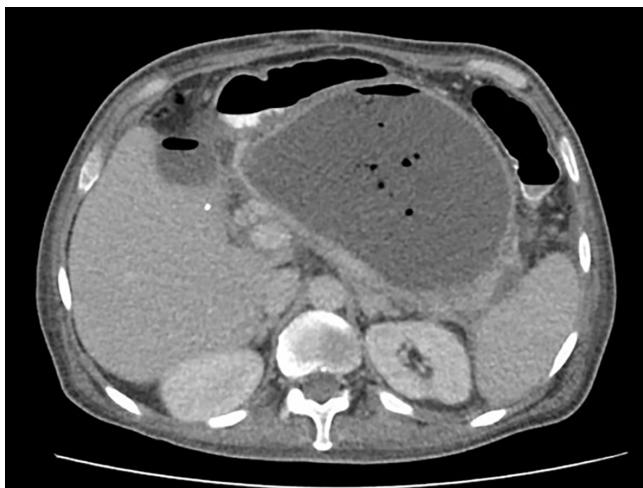


Figure 1. Initial CT scan showing pancreatic collection measuring $123 \times 119 \times 163$ mm (L \times AP \times T). Image property of the authors.

Table 1. Initial Peritoneal Fluid Analysis

| Peritoneal Fluid Study |
|--|
| Appearance: Slightly turbid Color: Yellow |
| WBC: 224 cells/mm ³ <i>Differential</i> - Lymphocytes: 78% - Monocytes: 4% - Neutrophils: 18% |
| Macrophages: 117 per 100 WBC Mesothelial cells: 2 per 100 WBC RBC: 244 cells/mm ³ |
| Biochemical |
| Method: Colorimetric Glucose: 79.32 mg/dL Protein: 2.69 g/dL |
| Amylase |
| Amylase: 9579 U/L |
| Microbiology |
| Gram stain: No organisms Culture: ESBL-producing <i>E. coli</i> |

Table prepared by the authors.

DISCUSSION

Pancreatic ascites is an uncommon complication of pancreatitis, most frequently occurring in the context of pseudocysts or walled-off necrosis, as seen in our patient. In chronic pancreatitis, pseudocysts typically develop less robust fibrin walls, allowing pancreatic secretions to leak from ruptured ducts into the pseudocyst and peritoneal cavity. Alternatively, pancreatic duct rupture without pseudocyst formation may create fistulous tracts.

The clinical manifestations vary depending on fistula location: anterior pancreatic duct disruptions permit direct pancreatic secretion drainage into the peritoneum, causing ascites, whereas posterior duct injuries often form fistulas through the aortic or esophageal hiatus or diaphragmatic dome, leading to pleural effusions. In both scenarios, the ascitic fluid is typically exudative with elevated amylase levels. Some attribute this exudative quality to pancreatic fluid triggering an inflammatory process that increases vascular permeability⁽¹¹⁾.

Diagnosis requires ascitic fluid analysis demonstrating amylase >1000 U/L, protein >2.5 g/dL (in our patient: 9579 U/L and 2.96 g/dL, respectively), and serum-ascites albumin gradient <1.1 g/dL.

Given the high suspicion for pancreatic duct disruption in this pathophysiology, pancreatography is recommended. Treatment approaches include medical, endoscopic, and surgical interventions, often used in combination, with no clear mortality superiority established for any single modality⁽¹²⁾.

Mild cases may resolve with medical management alone (30%-50% of patients), though endoscopic therapy remains first-line due to lower morbidity. Transpapillary pancreatic duct stenting reduces intraductal pressure and diverts secretions to the small intestine. Other endoscopic options—such as injectable glues or fibrinogen sealants for fistula occlusion—lack robust evidence for routine use. Overall, endoscopic approaches show promising outcomes with lower mortality and morbidity than surgery⁽¹³⁾.

EUS-guided endoscopic therapy may be indicated for pancreatic duct disruption syndrome when imaging confirms the lesion and anatomy suggests treatment res-

ponsiveness⁽¹⁴⁻¹⁶⁾. Our patient's anatomy precluded this approach, prompting surgical intervention.

Notably, while repeated endoscopic therapy appears inferior to surgery for long-term abdominal pain control (typically assessed after two years)^(17,18), our patient fortunately reported no such complaints. Following inconclusive pancreatography and failed endoscopic control, we proceeded with video-assisted retroperitoneal debridement.

The overall prognosis for patients with pancreatic ascites has improved with the availability of endoscopic interventions. Endoscopic transpapillary stent placement demonstrates success rates ranging from 82% to 100%. For cases refractory to medical and endoscopic management, surgical intervention is pursued, with reported mortality rates between 15% and 25%. Recent studies indicate that endoscopic treatment has reduced mortality, hospital stay duration, recurrence rates, and hospitalization costs compared to standalone medical or surgical interventions⁽⁸⁾.

Additional strategies—such as long-term pancreatic enzyme replacement—are indicated for severe exocrine pancreatic insufficiency and chronic pancreatitis with malabsorption symptoms or abdominal pain. Exocrine pancreatic insufficiency typically occurs when >90% of pancreatic parenchyma is compromised. In our case, only 30% of proximal pancreas required resection, preserving functionality and preventing malabsorptive symptoms^(19,20).

CONCLUSION

Pancreatic ascites represents an uncommon complication of acute pancreatitis, resulting from peritoneal accumulation of pancreatic fluid due to ductal injury. It occurs most frequently in necrotizing pancreatitis with pancreatic duct involvement, with chronic pancreatitis being the primary risk factor.

Mild cases without visible duct disruption respond to medical management, while severe cases with main pancreatic duct injuries require endoscopic stenting. Surgical intervention is rarely needed as rescue therapy.

Although high-quality evidence remains limited, some studies suggest endoscopic approaches reduce hospitalization duration, morbidity, and mortality compared to medical or surgical management alone.

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