International Journal of Gastroenterology Research

E-ISSN: 2664-6447
P-ISSN: 2664-6439
www.gastroenterologyjournal.in
Gastro 2025; 7(1): 45-48
Received: 15-05-2025
Accepted: 20-06-2025

Dr. Rakesh Kumar Adi
Department of Medical Gastroenterology, Yashoda Hospital, Secunderbad, Telangana, India

Dr. Akhil Konduru
Department of Medical Gastroenterology, Yashoda Hospital, Secunderbad, Telangana, India

Dr. Anil Kumar Mannava
Department of Medical Gastroenterology, Yashoda Hospital, Secunderbad, Telangana, India

Dr. Vamshidhar Reddy
Department of Medical Gastroenterology, Yashoda Hospital, Secunderbad, Telangana, India

Dr. GP Venkat Choudary
Department of Interventional Radiology, Yashoda Hospital, Secunderbad, Telangana, India

Dr. Vikram
Department of Radiology, Yashoda Hospital, Secunderbad, Telangana, India

Corresponding Author:
Dr. Rakesh Kumar Adi
Department of Medical Gastroenterology, Yashoda Hospital, Secunderbad, Telangana, India

Caught between a rock and a hard place: Impostor in pancreatic fluid collections

Rakesh Kumar Adi, Akhil Konduru, Anil Kumar Mannava, Vamshidhar Reddy, GP Venkat Choudary and Vikram

DOI: https://www.doi.org/10.33545/26646439.2025.v7.i1a.13

Abstract
A 46-year-old female was referred to our hospital with low grade fever, progressive abdominal distension since one month. The patient presented to our hospital with recurrent ascites, despite having undergone multiple diagnostic and therapeutic paracentesis at an outside facility. Imaging revealed a large anterior collection (7 × 21 cm) along with lesser sac and pelvic collections, with severe pancreatic atrophy. Endoscopic ultrasound confirmed chronic pancreatitis with pseudocyst and loculated ascites. Fluid analysis showed neutrophilic predominance (TC:13, 500) but unexpectedly a low amylase (<30) in aspirated collection, suggesting spontaneous bacterial peritonitis (SBP). Subsequent lesser sac aspirate revealed the same. The patient was treated with pigtail drainage of anterior preperitoneal collection, EUS guided CGS of lesser sac collection, pancreatic duct stenting, i.v antibiotics and leading to significant resolution of the collection. Patient previously had multiple admissions for diagnostic and therapeutic tapping of ascitic fluid. This case highlights the diagnostic dilemma of loculated collections mimicking pancreatic fluid collections. Low amylase levels in preperitoneal collections may mislead clinicians, emphasizing the need for comprehensive imaging and fluid analysis to guide management.

Keywords: Chronic pancreatitis, endoscopic retrograde cholangiopancreatography (ERCP), endoscopic ultrasound (EUS)-guided cystogastrostomy (CGS), pseudocyst

Introduction
Chronic pancreatitis is a gradual inflammatory condition marked by permanent harm to pancreatic tissue, impairing both exocrine and endocrine functions. With the passage of time, pancreatic parenchyma is substituted by fibrous tissue, resulting in intense abdominal pain, malabsorption, and diabetes mellitus. As the illness advances, individuals frequently endure crippling symptoms and a notable deterioration in their quality of life. The condition is associated with multiple factors, such as prolonged alcohol consumption, genetic influences and autoimmune diseases, with ongoing inflammation exacerbating its progression [1].

Pancreatic insufficiency occurs when over 90% of the organ is affected. The occurrence varies based on the disease's severity and can reach up to 85% in severe chronic pancreatitis. Conversely, dysfunction of the pancreas endocrine activity ultimately leads to pancreatogenic diabetes (type 3c diabetes) [2]. It is linked to numerous complications, the most prevalent being the development of pseudocysts. In rare cases, it results in pancreatic ascites or pleural effusion [3]. A pseudocyst of the pancreas is a specific fluid accumulation abundant in amylase and other pancreatic enzymes, enclosed by a fibrous tissue wall that lacks epithelial lining [4].

In 1991, D'Egidio and Schein proposed a classification system for pancreatic pseudocysts that considers the cause of pancreatitis (whether acute or chronic), the anatomy of the pancreatic duct and whether the cyst communicates with the ductal system [5]. Pancreatic pseudocysts are categorized into three types. Type I or acute post-necrotic pseudocysts appear after acute pancreatitis, typically involving a normal pancreatic duct with minimal ductal communication. Type II pseudocysts occur after acute-on-chronic pancreatitis, where the duct is abnormal but not narrowed, and communication with the cyst is frequent. Type III known as retention pseudocysts, develop in chronic pancreatitis, showing ductal strictures and a clear connection with the pancreatic duct [6].

Nealon and Walser proposed a classification of pancreatic pseudocysts based on pancreatic duct anatomy. Type I has a normal duct without cyst communication, while Type II also has a normal duct but shows duct-cyst communication. Type III presents with a ductal stricture and no communication and Type IV includes both a stricture and communication. Type V shows complete ductal obstruction. Type VI and VII occur in chronic pancreatitis, without and with duct-cyst communication, respectively [7].

Pancreatic pseudocysts are relatively uncommon, with an estimated incidence of 1.6%-4.5%, corresponding to approximately 0.5-1 case per 100,000 adults annually. Their occurrence is notably higher in chronic pancreatitis compared to acute pancreatitis [8]. Reports in the literature suggest that pancreatic pseudocysts may develop in 30% to 40% of cases. However, precise incidence data from long-term follow-up of patients with chronic pancreatitis are limited. Unlike acute pseudocysts, patients with chronic pancreatitis may live with the disease for 10, 20 or more years, placing them at an increased lifetime risk of developing a pseudocyst at least once during the course of their illness [8]. Pancreatic ascites (PA) most commonly arises as a complication of chronic pancreatitis (CP), though other etiologies have been described. It is characterized by exudative ascitic fluid with an amylase concentration at least six times higher than that of serum or exceeding 1000 IU/L. [9]. Chronic pancreatitis leads to fibrosis, parenchymal calcifications and local inflammation, which increase intraductal pressure and cause pancreatic secretions to leak. Leakage may result from rupture of the main pancreatic (Wirsung) duct but more often arises from a pancreatic pseudocyst, present in about 80% of pancreatic ascites cases. Pancreatic ascites is rare, occurring in roughly 3.5% of chronic pancreatitis patients and 6-14% with pseudocysts. The fluid can track into the retroperitoneum or mediastinum, with pancreatico-pleural effusions reported in 18-38% of cases [9].

We report the case of an elderly woman with a history of chronic calcific pancreatitis, presenting with a large preperitoneal and pelvic fluid collection that mimicked pancreatic ascites.

Case Presentation

A 46-years-old female known diabetic presented with complaints of progressive abdominal distension associated with fever since four weeks. Patient previously had multiple admissions for diagnostic and therapeutic tapping of ascitic fluid at an outside facility. On examination patient was febrile. Vitals were stable, abdomen was distended and tense ascites was present. Hemoglobin (Hb) was 10. 2 g/dL, total leukocyte count was 16,200/mm³ (or) 16.2 × 10⁹/L, C-Reactive protein (CRP) was 9.8 mg/L and liver function tests were within normal limits.

Upon contrast-enhanced computed tomography (CECT) of the abdomen showed large anterior extraperitoneal collection measuring approximately 7 × 21 cm, along with collections in the lesser sac and pelvis. Severe pancreatic atrophy with intraductal calcifications was observed. Endoscopic ultrasound (EUS) revealed an atrophic pancreas with a pseudocyst and loculated ascites. Endoscopic retrograde cholangiopancreatography (ERCP) showed filling defects in the pancreatic head linked to the pseudocyst in the tail region, for which a pancreatic duct stent was placed (Figure 1).

A pigtail catheter was inserted under ultrasound guidance to drain the extraperitoneal fluid, which showed thick greenish pus-filled fluid. Analysis of the initial drain (anterior collection) revealed a total cell count of 13,500 cells/mm³ predominantly neutrophilic, adenosine deaminase (ADA) level of 190 U/L, normal amylase levels and culture was positive for *Escherichia coli.* Endoscopic ultrasound (EUS)-guided aspiration from the lesser sac demonstrated a total cell count (TC) of 72,000 /mm³, Amylase: 72 U/L.

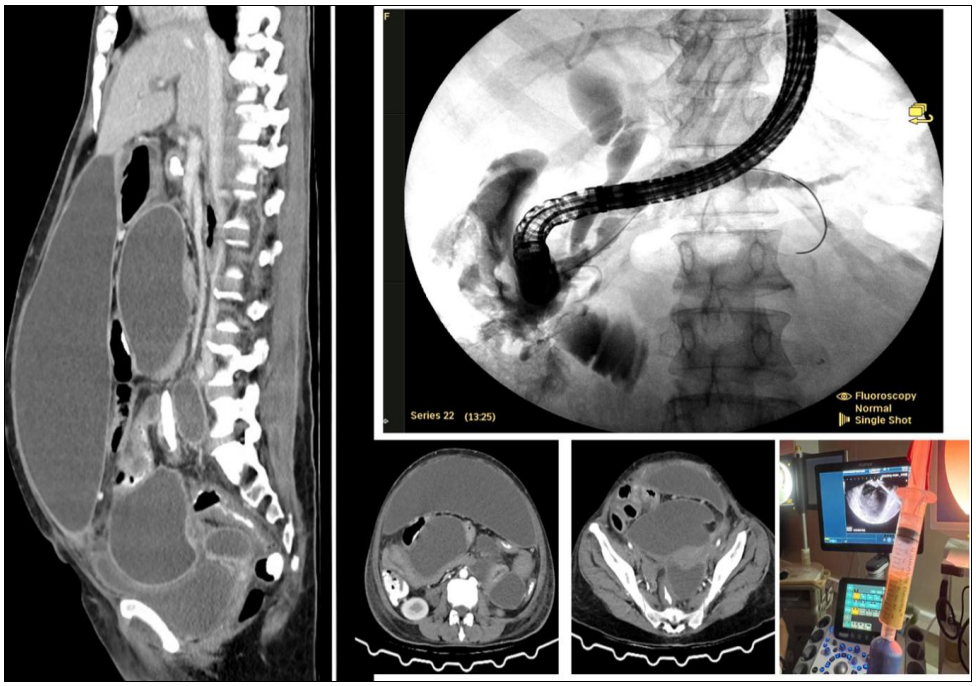

Fig 1: CECT Abdomen showing Extraperitoneal, Lesser sac and Pelvic collections. ERCP fluoro image suggestive of possible duct communication at tail region. EUS guided aspirate from lesser sac.

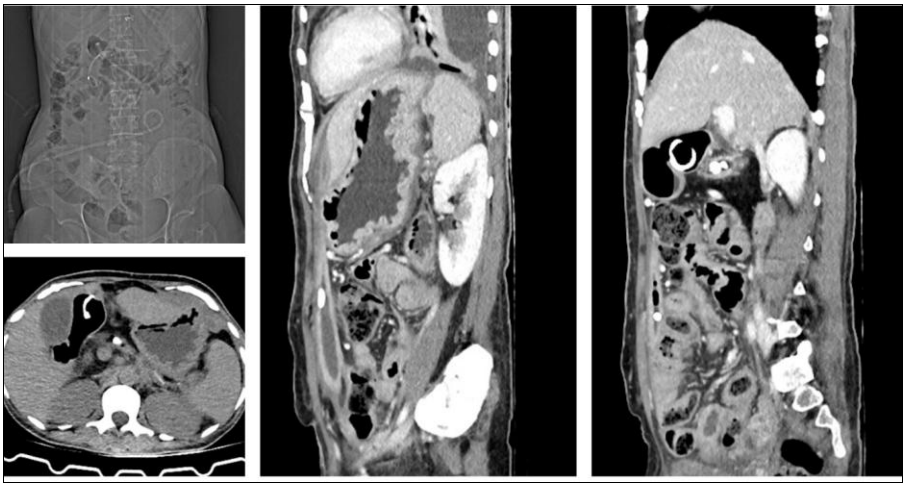

Fig 2: Repeat CT Abdomen suggestive of thin rim of collection at anterior infraumbilical location, along with significant reduction in pelvic, left hypochondriac, lesser sac collections

The patient underwent with pancreatic duct stenting, endoscopic ultrasound (EUS)-guided cystogastrostomy (CGS) for drainage of the lesser sac collection and interventional radiology (IR)-guided placement of a 12 Fr pigtail catheter for anterior collection drainage. Intravenous antibiotics were initiated targeting the Gram-negative infection. A follow-up contrast-enhanced CT scan of the abdomen showed complete resolution of the collections (Figure 2). The pigtail catheter was subsequently removed at one-month follow-up once the drainage had reduced to less than 5 mL/day.

Discussion

Patients with chronic pancreatitis, particularly those with risk factors like diabetes can present with progressive abdominal distension, fever and ascites [10]. In this case report our patient was presented with abdominal distension and fever.

Imaging in these cases frequently shows fluid collections around the pancreas, less common locations include extraperitoneal and preperitoneal fluid collections. In another case report it was described that an anterior extraperitoneal collection that occurred after necrotizing pancreatitis, resembling the significant extraperitoneal fluid observed in our case [11]. The existence of pseudocysts, ductal calcifications, and ductal disruptions corresponds with the mechanisms that allow for the leakage of pancreatic secretions, which may extend into atypical areas. These observations highlight that persistent ductal alterations play a role in the development of complications [12].

In these cases, amylase mainly serves as a sign of reduced pancreatic function instead of being a quick marker for inflammation. Typically, low or normal amylase levels are seen in chronic conditions, particularly in the later stages of the calcified type, which reflects the reduction of functional pancreatic tissue [13]. In our case, it was observed that patient's amylase levels were normal. Elevated levels of adenosine deaminase (ADA) might suggest inflammation in the pancreas and could serve as a marker for pancreatic conditions such as pancreatitis [14]. Endoscopic retrograde cholangiopancreatography (ERCP) plays a crucial role in evaluating ductal anatomy and managing complications of chronic pancreatitis [15]. In our case, ERCP demonstrated filling defects in the pancreatic head communicating with a tail pseudocyst, indicating ductal disruption.

Endoscopic transpapillary pancreatic duct stenting is becoming more common for addressing duct disruptions and minimizing leakage. Endoscopic ultrasound-guided cystogastrostomy is regarded as the standard treatment for symptomatic or infected pseudocysts and walled-off collections, providing high success rates and lower morbidity in comparison to surgical drainage [16]. IR-guided pigtail catheter drainage is an effective option for large or loculated collections not suited to endoscopic access. A "step-up" approach, starting with minimally invasive drainage and escalating only if needed, improves outcomes while lowering complications and hospital stay [17].

Conclusion

This case demonstrates the diagnostic difficulty of extraperitoneal pancreatic collections that resemble ascites. Normal amylase levels in infected collections can confuse clinicians, emphasizing the need for thorough imaging and fluid analysis. A comprehensive strategy that includes ERCP with ductal stenting, EUS-guided cystogastrostomy and IR-guided drainage led to complete resolution. Timely identification, precise differentiation of fluid types and minimally invasive step-up interventions are vital for managing complex collections and avoiding unnecessary procedures, ultimately enhancing patient outcomes in chronic pancreatitis.

Acknowledgements

The authors would like to thank Management Yashoda Hospitals and Dr. Amidyala Lingaiah (Director - Medical Sciences) for the continuous support

Declarations

Funding

No funding sources

Conflict of interest

None declared

Ethical approval

Not required

References

1. Benjamin O, Lappin SL. Chronic Pancreatitis. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2025. PMID: 29493950.
2. Ashouri M, Tadbir Vajargah K, Mohammadzadeh N, Sahraian S. A huge preperitoneal collection following acute necrotizing pancreatitis: A case report and the management approach. Ann Med Surg (Lond). 2022;78:103843.
3. Noh AM, Abdul Rashid AM, Norafida B, Hazeman ZM, Idris I, Suraini MS. Chronic pancreatitis with extra peritoneal fluid collection misleadingly presenting as ascites. Imaging Med. 2016;8(4).
4. Bradley EL 3rd. A clinically based classification system for acute pancreatitis. Summary of the International Symposium on Acute Pancreatitis, Atlanta, Ga, September 11 through 13, 1992. Arch Surg. 1993;128(5):586-590.
5. Habashi S, Draganov PV. Pancreatic pseudocyst. World J Gastroenterol. 2009;15(1):38-47.
6. D'Egidio A, Schein M. Pancreatic pseudocysts: a proposed classification and its management implications. Br J Surg. 1991;78(8):981-984.
7. Nealon WH, Walser E. Main pancreatic ductal anatomy can direct choice of modality for treating pancreatic pseudocysts (surgery versus percutaneous drainage). Ann Surg. 2002;235(6):751-758.
8. Ammann RW, Akovbiantz A, Largiader F, Schueler G. Course and outcome of chronic pancreatitis. Longitudinal study of a mixed medical-surgical series of 245 patients. Gastroenterology. 1984;86(5 Pt 1):820-828.
9. Schneider Bordat L, El Amrani M, Truant S, Branche J, Zerbib P. Management of pancreatic ascites complicating alcoholic chronic pancreatitis. J Visc Surg. 2021;158(5):370-377.
10. Bathobakae L, Farhan H, Mücahit D, Rohira D, Chauhan K, Cavanagh Y, *et al*. Pancreatic ascites: update on diagnosis and management. Ann Gastroenterol. 2025;38(3):247-254.
11. Ashouri M, Tadbir Vajargah K, Mohammadzadeh N, Sahraian S. A huge preperitoneal collection following acute necrotizing pancreatitis: A case report and the management approach. Ann Med Surg (Lond). 2022;78:103843.
12. Misra D, Sood T. Pancreatic Pseudocyst. [Updated 2023 Aug 8]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK557594/
13. Jalal M, Gbadegesin SA, Tehami N, Nakajima K. What is the clinical significance of low serum amylase? Systematic review of the conditions associated with low serum amylase. Frontline Gastroenterol. 2023;15(2):154-161.
14. Parida SK, Pottakkat B, Raja K, Vijayahari R, Lakshmi CP. Bacteriological profile of pancreatic juice in patients with chronic pancreatitis. JOP. 2014;15(5):475-477.
15. Wilcox CM, Bang JY, Asombang A, Bennett C, Bi Y, Blogowski W, *et al*. Management of the Disconnected Pancreatic Duct in Pancreatic Necrosis. Clin Gastroenterol Hepatol. 2025:S1542-3565(25)00586-5.
16. Renelus BD, Jamorabo DS, Gurm HK, Dave N, Briggs WM, Arya M. Comparative outcomes of endoscopic ultrasound-guided cystogastrostomy for peripancreatic fluid collections: a systematic review and meta-analysis. Therapeutic Advances in Gastrointestinal Endoscopy. 2019;12:2631774519843400.
17. Pattarapuntakul T, Charoenrit T, Wong T, Netinatsunton N, Ovartlarnporn B, Yaowmaneerat T, *et al*. Clinical Outcomes of the Endoscopic Step-Up Approach with or without Radiology-Guided Percutaneous Drainage for Symptomatic Walled-Off Pancreatic Necrosis. Medicina (Kaunas). 2023;59(3):569.

How to Cite This Article
Kumar RA, Konduru A, Mannava AK, Reddy V, Choudary GPV, Vikram. Rakesh Kumar Adi, Akhil Konduru, Anil Kumar Mannava, Vamshidhar Reddy, GP Venkat Choudary and Vikram. International Journal of Gastroenterology Research. 2025; 7(1): 45-48.