J Surg Med. 2025;9(11):251-254.
https://doi.org/10.28982/josam.8139

JOSAM

Journal of Surgery and Medicine

e-ISSN: 2602-2079

Case report
https://jsurgmed.com/

Pancreaticopleural fistula – An unusual indication of intercostal tube drainage

Tarun Kalra [1], Abhinav Agarwal [2]

[1] Department of Surgical Gastroenterology, Motilal Nehru Medical College, Prayagraj, Uttar Pradesh, India
[2] Department of General Surgery, Motilal Nehru Medical College, Prayagraj, Uttar Pradesh, India

ORCID of the author(s)

TK: https://orcid.org/0009-0003-3823-4208
AA: https://orcid.org/0009-0007-6873-2960

Corresponding Author
Tarun Kalra
Department of Surgical Gastroenterology, Motilal Nehru Medical College, Prayagraj, Uttar Pradesh, India
E-mail: drtk2014@gmail.com

Informed Consent
The authors stated that the written consent was obtained from the patient presented with images in the study.

Conflict of Interest
No conflict of interest was declared by the authors.

Financial Disclosure
The authors declared that this study has received no financial support.

Published
2025 October 9

Abstract

Pancreatitis, whether acute or chronic, can lead to a spectrum of local and systemic complications due to the extravasation of inflammatory mediators in the vicinity of the gland. One such rare complication is the development of a pancreaticopleural fistula (PPF), typically presenting as a unilateral or, less commonly, bilateral pleural effusion accompanied by respiratory symptoms. A young male in his thirties with a history of heavy alcohol consumption presented with dyspnea, upper abdominal pain, and a recent episode of an alcohol binge, one month ago. Clinical examination revealed epigastric tenderness in the epigastrium with decreased breath sounds, and a diagnosis of acute pancreatitis was made. This was confirmed by elevated serum lipase and bilateral pleural effusion on chest X-ray. Given his partial response to conservative management, a decision was taken for bilateral tube thoracostomy. Fluid analysis confirmed the diagnosis of PPF. He was subsequently started on somatostatin analogs and parenteral nutrition. He responded well to the above treatment and was discharged after a week. While PPF is a recognized cause of unilateral pleural effusion, bilateral involvement is uncommon. The condition often presents predominantly with chest symptoms, with a paucity of abdominal complaints. It should be suspected that alcoholic pancreatitis responds poorly to usual conservative therapy. The biochemical analysis of the aspirated pleural fluid reveals significantly increased amylase levels, which clinches the diagnosis. Cross-sectional imaging helps in characterizing the primary disease in the pancreas and in delineating the ductal anatomy. Endoscopy is both diagnostic and therapeutic, as it involves placing a stent in the main pancreatic duct (MPD). The management involves a combination of medical, endoscopic, and surgical modalities. Medical management is successful in a majority of cases by using somatostatin analogs with nutritional support. Placement of an intercostal tube hastens recovery and reduces the duration of hospitalization. Surgical management by distal pancreatectomy with pancreatojejunostomy is reserved for a small subset of patients. Hence, patients with PPF rarely present with bilateral pleural effusion and are an indication for tube thoracostomy with successful results.

Keywords: pleural effusion, intercostal tube, pancreaticopleural fistula, octreotide, endoscopy, Contarini syndrome

Introduction

Bilateral pleural effusion is commonly seen in systemic disorders such as congestive cardiac failure, cirrhosis, end-stage renal disease, hypoalbuminemia, malignancy, autoimmune disease, or a combination of these etiologies. In contrast, acute pancreatitis is an uncommon etiology of bilateral effusion [1].

In rare cases, acute or chronic pancreatitis can lead to the formation of internal fistulae. The most common clinical presentation includes dyspnea with or without accompanying abdominal pain. PPF is typically diagnosed by the increased levels of amylase in pleural fluid and cross-sectional imaging such as magnetic resonance cholangiopancreatography (MRCP) and contrast-enhanced computed tomography (CECT). These imaging techniques help visualize the fistulous tract, the anatomy of the MPD, and changes in pancreatic parenchyma [1-3]. Diagnosing PPF requires skilled clinical assessment and is managed using various modalities, including medications, endoscopic stents, and surgery. This article presents a unique case of PPF, which manifested as bilateral pleural effusion and was managed by bilateral thoracostomy.

How to cite: Kalra T, Agarwal A. Pancreaticopleural fistula – An unusual indication of intercostal tube drainage. J Surg Med. 2025;9(11):251-254.

Case presentation

History

A 30-year-old male presented with complaints of dyspnea for one week, which was aggravated in the supine position. He reported a recent episode of alcohol binge drinking approximately a month back, which was followed by upper abdominal pain. He self-medicated with over-the-counter pain medications, which provided partial relief. However, he continued to experience abdominal discomfort, particularly after meals. He denied vomiting, high-grade fever, or cough. Notably, he had a similar episode of upper abdominal pain six months earlier, which was managed conservatively at a local hospital. There was no other significant past medical or surgical history.

Examination

On examination, he was tachypneic and tachycardic with signs of malnutrition and dehydration. Abdominal examination revealed tenderness and fullness in the epigastrium region. Respiratory examination showed diminished vesicular breath sounds, more on the left side. The remainder of the systemic examination was normal. Based on his symptoms, history of alcohol use, and examination findings, a provisional diagnosis of acute pancreatitis with pleural effusion was made.

Diagnosis

Initial laboratory investigations revealed leukocytosis with elevated serum amylase (1729 IU/L) and lipase (1249.7 IU/L) levels. A chest roentgenogram (X-ray) demonstrated moderate pleural effusion on the left side and mild pleural effusion on the right side, with a tracheal deviation to the right (Figure 1).

Management

The patient was admitted and managed with intravenous fluids, analgesics, and supplemental oxygen via a face mask at 4L/minute. As his oxygen saturation at room air was low, at 88%, he was maintained in a reclining posture.

Despite initial therapy for 3 days, he reported partial symptom relief. He underwent CECT of the thorax and abdomen, which revealed acute pancreatitis with acute pancreatic fluid collection (APFC) near the tail of the pancreas and extending to the perisplenic area and moderate bilateral pleural effusion without atelectasis (Figure 2).

Due to persistent dyspnea, tube thoracocentesis was performed bilaterally, draining 400 ml of fluid on the left side and 200 ml of fluid on the right side (Figure 3). The aspirated fluid was serous and was sent for biochemical and microbiological analysis, which revealed markedly elevated pleural fluid amylase levels: 51416 IU/L on the left side, and 10412 IU/L on the right side. This confirmed the diagnosis of a PPF.

Meanwhile, the patient showed significant improvement in dyspnea following drainage. Subsequently, he was started on subcutaneous octreotide (100 mcg every 8 hours), kept nil by oral, and initiated on total parenteral nutrition (TPN).

Over the next two weeks, there was a progressive decline in his thoracostomy output, and the patient's overall well-being improved. A follow-up chest roentgenogram (X-ray) showed a resolution of effusion on the right side, leading to the removal of the right thoracostomy tube. TPN was gradually weaned off, and oral intake resumed.

The patient was discharged with a left-sided thoracostomy tube in situ, which was subsequently removed two weeks later during follow-up after output was ceased. Repeat imaging confirmed full pulmonary re-expansion (Figure 4).

Figure 1: Chest roentgenogram with bilateral pleural effusion.

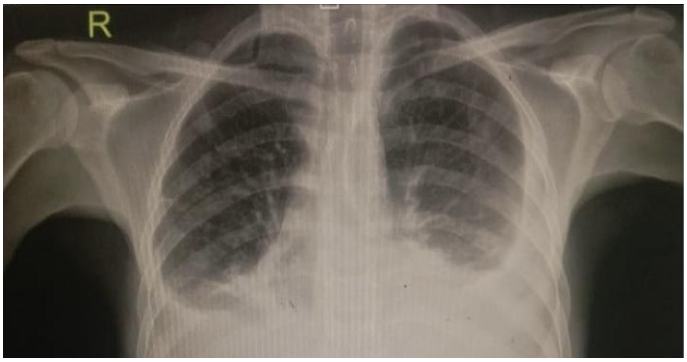


Figure 2: CECT chest and abdomen with bilateral pleural effusion and acute pancreatitis with APFC.

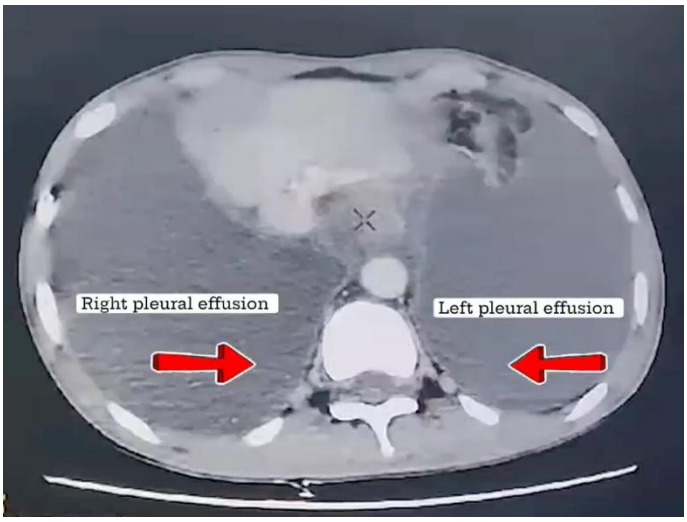


Figure 3: Chest roentgenogram with bilateral intercostal tubes and resolution of pleural effusion.

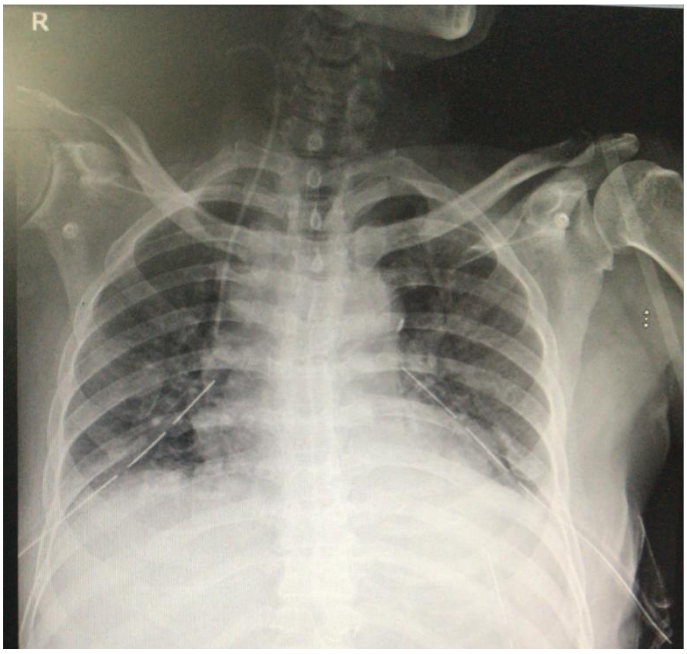
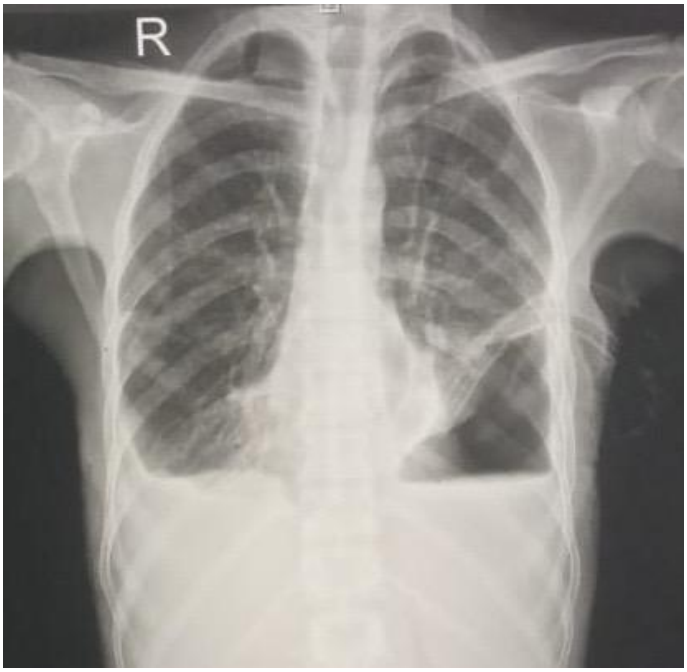


Figure 4: Chest roentgenogram with only the left side intercostal tube.



Discussion

PPF was first identified as a separate complication of pancreatitis in the 1960s and 70s and has remained an uncommon and difficult entity to treat [1]. The incidence of PPF is 0.4% in patients with chronic pancreatitis, and it usually presents as a left-sided pleural effusion, causing dyspnea [2]. The incidence of right-sided effusion in PPF is around 20%, while bilateral effusion is rarer (<15%) [3]. On the contrary, PPF accounts for 1% of cases of pleural effusion [4].

PPF is caused either by the direct extension of a pseudocyst across the diaphragm or by the formation of a fistulous tract between the pancreas and the pleural spaces. Ethanol is implicated in the secretion of insoluble pancreatic proteins that calcify and occlude the MPD. The pancreatic fluid leaks into the retroperitoneum. From there, it usually moves cranially due to the transdiaphragmatic pressure gradient between the abdominal and pleural cavities [5]. PPF is characterized by the rapid accumulation of significant fluid and its refractoriness [4,6]. Persistent PPF can lead to inadequate lung expansion (trapped lung) and has been reported in a few case reports.

Diagnosing PPF involves differentiating it from reactive and inflammatory pleural effusion in acute pancreatitis. Although PPF is common on the left side, it may also occur on the right side or bilaterally [7]. In cases of bilateral pleural effusion, one must rule out Contarini syndrome.

This syndrome was first described in Francesco Contarini in the 16th century in Venice, who had developed bilateral pleural effusion. His autopsy revealed right-sided pleural effusion due to cardiac failure and a contralateral empyema. The presence of two different etiologies for bilateral pleural effusion occurs in 5% of cases. This condition remains underdiagnosed as experts recommend doing a bilateral thoracocentesis only if [8]:

1. Unilateral lung parenchymal involvement
2. Significant disparity in the size of effusions
3. Markedly different attenuation values on CECT
4. Atypical clinical findings (e.g., fever or pleuritic chest pain in the presence of decompensated cardiac failure)
5. Resolution of pleural effusion only on one side

The typical patient of PPF is a middle-aged, chronic alcoholic male presenting with breathlessness, cough, and chest pain but no abdominal pain [9]. Less commonly, it may occur in cases of gallstone-induced, traumatic, and idiopathic pancreatitis [10]. The predominance of cardiopulmonary symptoms and lack of abdominal features explains the delay in diagnosis (median of 5.6 weeks) [11]. The presented case aligns with this profile, highlighting the importance of clinical suspicion in such scenarios.

Elevated pleural fluid amylase is a hallmark finding and serves as a suitable diagnostic test [12]. In the review by Rockey and Cello [11], the mean recorded value of pleural fluid amylase was found to be 18,450 IU/L (range: 1,830-164,187 IU/L). The value for our patient lies within the range mentioned in the above review. Various other pathologies with increased amylase levels, such as adenocarcinoma of the lung and female genital tract, pleural mesothelioma, and oesophageal perforation, must be ruled out [13].

A high degree of suspicion and clinical acumen is required to accurately diagnose this distinct entity. Imaging modalities help identify PPF and make therapeutic decisions. MRCP offers non-invasive visualization of ductal anatomy, aiding in stratifying further management [10]. MRCP has a sensitivity of 80% for detecting PPF, compared to 78% with endoscopic retrograde cholangiopancreatography (ERCP) [14].

The treatment of PPF is multidisciplinary and involves medical therapy, and endoscopic and/or surgical interventions. The initial conservative approach, including observation and medical therapy, is effective in 31-45% of patients [10]. Somatostatin analogs, such as octreotide, reduce the output from MPD, thereby leading to early fistula closure. The duration of medical treatment is unclear, with a suggested period of 2 to 4 weeks before considering alternative interventions [11]. These patients require frequent thoracocentesis. There are various case reports and series where the patients have been managed with medical therapy with pleural drainage only, without the need for endoscopy or surgery [14]. Our patient was managed similarly by a combination of medical therapy and placement of tube thoracostomy tubes bilaterally.

ERCP with stent placement has revolutionized nonoperative therapy for PPF [15]. Stents aid by mechanically occluding the fistulous communication and also dilating duct strictures [4]. Success rates vary, depending on patient selection and ductal anatomy. Surgical intervention, though definitive, is typically reserved for cases unresponsive to medical or endoscopic treatments but has a higher curative rate of up to 90% [4]. It includes pancreatojejunostomy with or without pancreatic resection.

Conclusion

In patients with a history of acute or chronic pancreatitis, all pleural effusions may not be assumed to be reactive. PPF may be considered in the list of differential diagnoses, especially in cases of bilateral effusions. Elevated pleural fluid amylase levels, coupled with imaging methods such as ERCP, MRCP, and CECT, confirm the diagnosis. Initially, a conservative, non-operative approach by use of drugs and pleural drainage remains effective in many cases, without the need for endoscopic intervention.

Surgical intervention is considered the last resort for patients who fail both conservative and endoscopic approaches.

References

1. Anderson WJ, Skinner DB, Zuidema GD, Cameron JL. Chronic pancreatic pleural effusions. Surg Gynecol Obstet. 1973;137:827–30.
2. Tay CM, Chang SK. Diagnosis and management of pancreaticopleural fistula. Singapore Med J. 2013;54(4):190–4.
3. Pottmeyer EW 3rd, Frey CF, Matsuno S: Pancreaticopleural fistulas. Arch Surg. 1987;122:648-54.
4. Dhebri AR, Ferran N. Nonsurgical management of pancreaticopleural fistula. JOP. 2005;6(2):152–61.
5. Akahane T, Kuriyama S, Matsumoto M, Kikuchi E, Kikukawa M, Yoshiji H, et al. Pancreatic pleural effusion with a pancreaticopleural fistula was diagnosed by magnetic resonance cholangiopancreatography and cured by somatostatin analogue treatment. Abdom Imaging. 2003;28:92-5.
6. Vyas S, Gogoi D, Sinha SK, Singh P, Yadav TD, Khandelwal N. Pancreaticopleural fistula: an unusual complication of pancreatitis diagnosed with magnetic resonance cholangiopancreatography. JOP. 2009;10:671–3.
7. Reechaipichitkul W, Bowornkitiwong T, Utchariyaprasit E. Chronic pancreatitis presenting with right pleural effusion: a case report. J Med Assoc Thai. 2010;93:378-82.
8. Porcel JM, Civit MC, Bielsa S, Light RW. Contarini's syndrome: bilateral pleural effusion, each side from different causes. J Hosp Med. 2012;7:164-5.
9. Hastier P, Rouquier P, Buckley M, Simler JM, Dumas R, Delmont JP. Endoscopic treatment of wirsungo-cysto-pleural fistula. Eur J Gastroenterol Hepatol. 1998;10:527–9.
10. King JC, Reber HA, Shiraga S, Hines OJ. Pancreatic-pleural fistula is best managed by early operative intervention. Surgery. 2010;147(1):154–9.
11. Rockey DC, Cello JP. Pancreaticopleural fistula. Report of 7 patients and review of the literature. Medicine (Baltimore). 1990;69:332-44.
12. Miller JA, Maldjian P, Seeff J. Pancreaticopleural fistula. An unusual cause of persistent unilateral pleural effusion. Clin Imaging. 1998;22:105–7.
13. Iglesias JI, Cobb J, Levey J, Rosiello RA. Recurrent left pleural effusion in a 44-year-old woman with a history of alcohol abuse. Chest. 1996;110:547-9.
14. Cazzo E, Apodaca-Rueda M, Gestic MA, Chaim FHM, Saito HPA, Utrini MP, et al. Management of pancreatico-pleural fistulas secondary to chronic pancreatitis. Arq Bras Cir Dig. 2017;30(3):225–8.
15. Safadi BY, Marks JM. Pancreatic-pleural fistula: the role of ERCP in diagnosis and treatment. Gastrointest Endosc. 2000;51:213–5.