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Recurring pleural effusion secondary to walled-off pancreatic necrosis

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ABSTRACT

Recurring, exudative, lymphocytic-predominant pleural effusions have not been previously reported in association with walled-off pancreatic necrosis. We present a case of chronic pancreatitis complicated by a large pancreatic fluid collection and recurrent pleural effusion. Endoscopic drainage of the walled-off pancreatic necrosis was the definitive treatment for both fluid collections.

KEYWORDS Chronic pancreatitis; lymphocytic-predominant pleural effusion; pleural fluid collection; walled-off pancreatic necrosis

Chronic pancreatitis is uncommonly associated with massive symptomatic pleural effusions. Furthermore, analysis of the pleural fluid is typically characterized by high levels of amylase.[1] In this report, we present a case of chronic pancreatitis complicated by a large walled-off pancreatic necrosis (WOPN) and large, recurring lymphocytic-predominant pleural effusion with normal amylase levels.

CASE REPORT

A 48-year-old alcoholic Hispanic man presented with a 1-month history of dyspnea, abdominal pain, and nonproductive cough. His pulse was 114 beats/minute, with all other vital signs normal. Lung examination revealed decreased respiratory sounds throughout all left lung fields. The abdomen was distended with mild tenderness to palpation diffusely. Computed tomography (CT) of the chest with contrast revealed a large left pleural effusion resulting in collapse of the left upper and lower lobes *(Figure 1a)*. CT of the abdomen and pelvis revealed a large multiloculated exophytic cystic lesion centered in the pancreatic tail measuring up to 14.6 cm and a large left hepatic subcapsular collection. Thoracentesis obtained 1.5 L of fluid. Analysis was consistent with a lymphocytic-predominant exudate (933 white cells/mL with 86% lymphocytes). The fluid amylase level was 150 U/L (upper limit of normal, 200 U/L). Pleural fluid cultures were negative for bacteria and fungi. Pleural cytology showed abundant macrophages and mixed inflammation and was negative for malignancy. Cultures and polymerase chain reaction testing for *Mycobacterium tuberculosis* were negative.

The left hepatic lobe subcapsular collection was drained. Its analysis was negative for *Coccidioides, Entamoeba histolytica,* and *Echinococcus*. The patient's fluid amylase level was low at 29 U/L. During hospitalization, he required two additional thoracenteses and placement of a chest tube. Analysis of the fluid obtained from these procedures was consistent with a lymphocytic-predominant, exudative pleural fluid with low to normal amylase levels.

Upper endoscopy with endoscopic ultrasound (EUS) confirmed a large pancreatic tail fluid collection with a mature wall and significant internal layering debris, consistent with a WOPN. An EUS-guided cyst gastrostomy was created with a cautery-enhanced, lumen-apposing metal stent (LAMS) (AXIOS, 15 mm, Boston Scientific, Marlborough, MA), followed by endoscopic pancreatic necrosectomy, debridement, and placement of two transgastric pigtail stents into the WOPN cavity to facilitate entry of gastric acid for sanitation of necrotic debris *(Figure 1b, 1d–1f)*.

A month after discharge, repeat CT of the chest and abdomen revealed a significant decrease in the size of the WOPN and minimal left pleural effusion. Complete resolution of the subcapsular hepatic fluid collection was also

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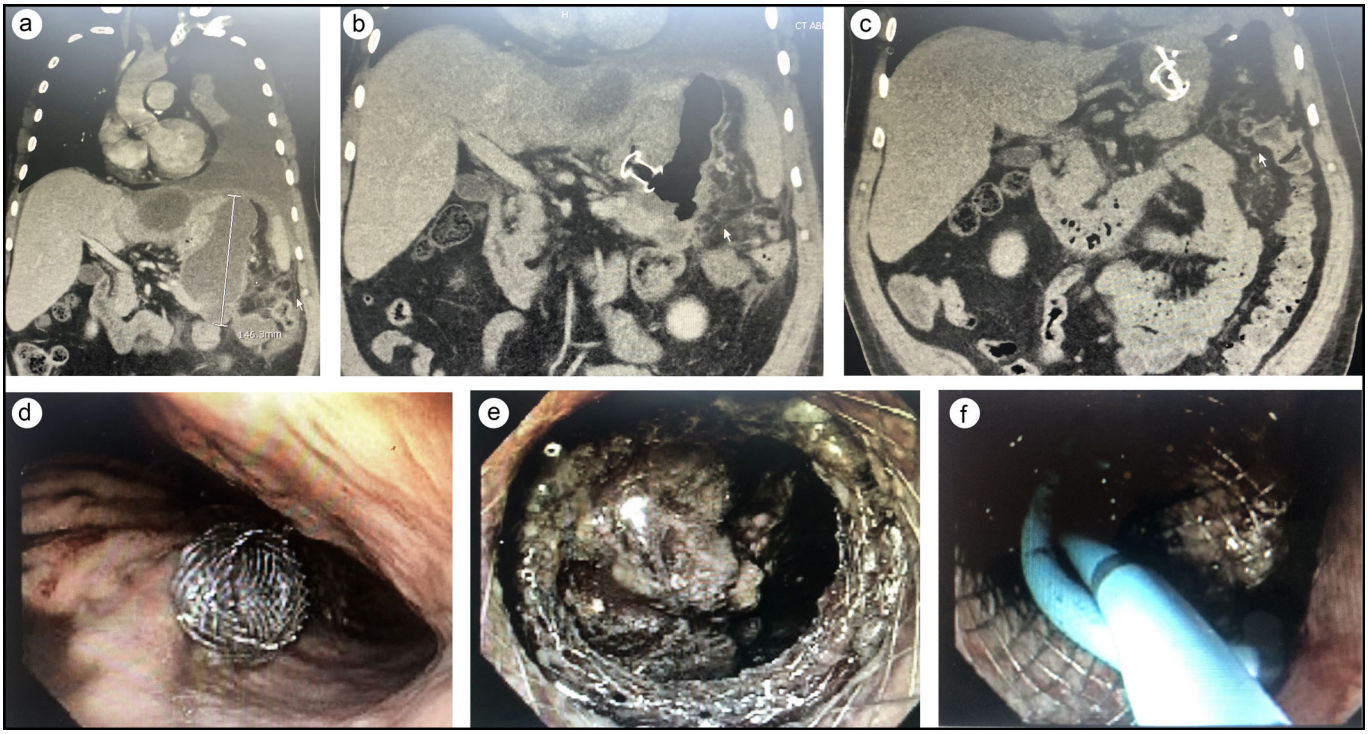

Figure 1. **(a)** Admission CT scan showing massive left-sided pleural effusion with concurrent pancreatic and hepatic subcapsular fluid collections. **(b)** Scan after endoscopic ultrasound-guided cyst gastrostomy with a cautery-enhanced, lumen-apposing metal stent, followed by endoscopic pancreatic necrosectomy, debridement, and placement of two transgastric pigtail stents into the walled-off pancreatic necrosis cavity. **(c)** Three-month follow-up showing complete resolution of the pancreatic and pleural fluid collections. **(d)** A transgastric lumen-apposing metal stent in the stomach. **(e)** Necrotic tissue within the cavity, seen via the lumen-apposing metal stent. **(f)** Placement of two double pigtail stents into the walled-off necrosis cavity to facilitate entry of gastric acid/sanitation of contents.

noted. Three months after EUS, the patient underwent a repeat endoscopic intervention with removal of the LAMS. The transgastric double pigtail stents were left in place to prevent recurrence of the pancreatic fluid collection. Repeat CT scan showed resolution of the pancreatic and pleural fluid collection *(Figure 1c)*.

DISCUSSION

The revised Atlanta criteria classify pancreatic fluid collections as acute or chronic, with chronic fluid collections subdivided into pancreatic pseudocysts and WOPN.[2] Differentiating between pancreatic pseudocysts and WOPN remains a clinical challenge due to the poor sensitivity of CT in detecting necrotic debris within the collection.[3] Although no universal guideline has been developed, it is important to keep in mind that pancreatic pseudocysts are connected with the pancreatic duct system, either as a direct communication or indirectly via the pancreatic parenchyma. Thus, the fluid collected from pancreatic pseudocysts is typically rich in amylase and other pancreatic enzymes and tends to respond to conservative medical management, including bowel rest or octreotide.[4] Therefore, our patient's pancreatic fluid collection is most consistent with WOPN in the setting of chronic pancreatitis.

Pleural fluid lymphocytosis, particularly with counts representing 85% to 95% of the total nucleated cells, has been associated with tuberculosis, lymphoma, sarcoidosis, rheumatoid arthritis, yellow nail syndrome, and chylothorax. Malignant pleural effusions are lymphocyte-predominant in over half of the cases; however, the percentage of lymphocytes is usually between 50% and 70%.[5,6]

We believe that our patient's clinical syndrome is the first documented case of a lymphocytic-predominant pleural effusion associated with WOPN in the setting of chronic pancreatitis. The pathophysiology may be explained by direct lymphatic drainage or a fistula connecting the individual anatomical spaces with the WOPN. A causative role might also be supported by the sustained resolution of the effusion only after the WOPN was drained.

In conclusion, lymphocytic-predominant recurrent pleural effusions characterized by low fluid amylase levels can be caused by WOPN. The resolution of all conditions requires the drainage of the pancreatic collection.

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