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Right-Sided Pleural Effusion Secondary to Acute Pancreatitis: A Rare Entity

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Abstract

Pleural effusion as a consequence of acute pancreatitis is transient, usually left-sided, and straw colored and accounts for 1% of all cases.[1] Rarely, it can be right-sided, causing difficulty in establishing the diagnosis, especially if new symptoms are disproportionate to the pre-existing symptoms. We present a case of a young chronic alcoholic male patient with gross right-sided pleural effusion and moderate ascites.

Keywords: Pleural effusion, Right sided, Acute pancreatitis

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Introduction

Acute pancreatitis is an acute inflammatory process of the pancreas in its severe form and is complicated by development of multiple organ dysfunction syndrome with a mortality rate of 15–20%.[2] Regardless of etiology, once acute pancreatitis is initiated, the inflammatory events within the acinar cells will progress to a generalized systemic inflammatory response syndrome.[2]

Among the systemic complications, pulmonary complications are the most frequent and potentially most serious.

Pleural effusion is the most common complication. Earlier it was thought to be a marker of acute pancreatitis, but, now, it is noted as a poor prognostic sign.[2]

Chronic pancreatitis is a progressive inflammatory disorder that leads to irreversible destruction of exocrine and endocrine pancreatic parenchyma caused by atrophy and/or replacement with fibrotic tissue.[2]

The most common cause is alcohol consumption. Alcohol increases secretion of proteins from acinar cells, causing the fluid to become viscous, leading to ductal obstruction, acinar fibrosis, and atrophy.[2]

Case Report

A 24-year-old, male patient, presented to the outpatient department with a history of shortness of breath, fever, chest discomfort, and reduced appetite since 7 days. He also gave a history of vomiting (two episodes) since 2 days.

He had been a known alcoholic since 5 years, and a known case of alcoholic pancreatitis.

Before visiting us, he was evaluated under medical gastroenterology for the same issues. High-resolution computed tomography [Figure 1] showed right-sided gross pleural effusion. On admission, he was lean, coherent, and febrile. He had mild pallor, no lymphadenopathy, clubbing, cyanosis, or icterus. His pulse rate was regular - 98/min, and blood pressure was 110/70 mm Hg.

A diagnostic aspiration was done and amber colored fluid was seen which was sent for analysis.

Biochemical analysis of the pleural fluid revealed the following: Proteins – 2.5 g%; Sugars – 90 mg%; and LDH – 240 U/L; albumin – 1.5 g%; amylase – 10718 U/L; lipase – 3394 U/L; and ADA – 15 U/L.

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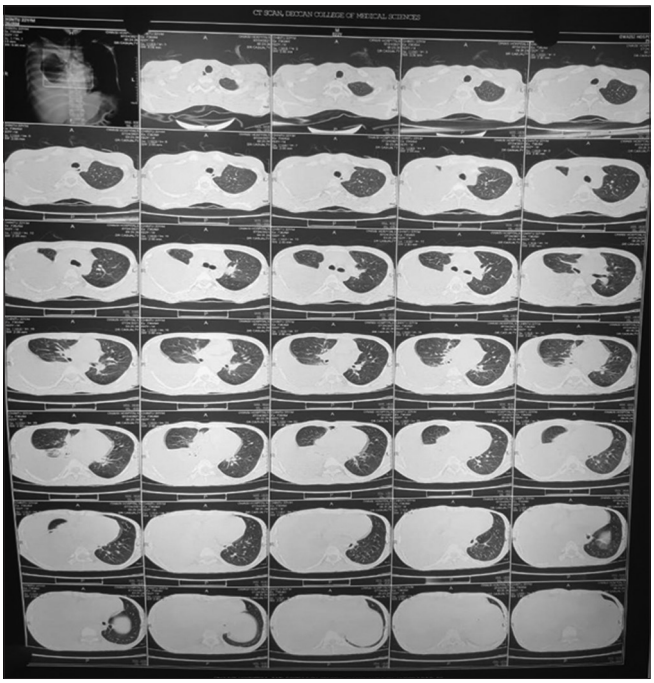

Figure 1: High-resolution computed tomography

Pleural fluid amylase was correlated with serum amylase which also was highly elevated (Pancreatic enzymes were

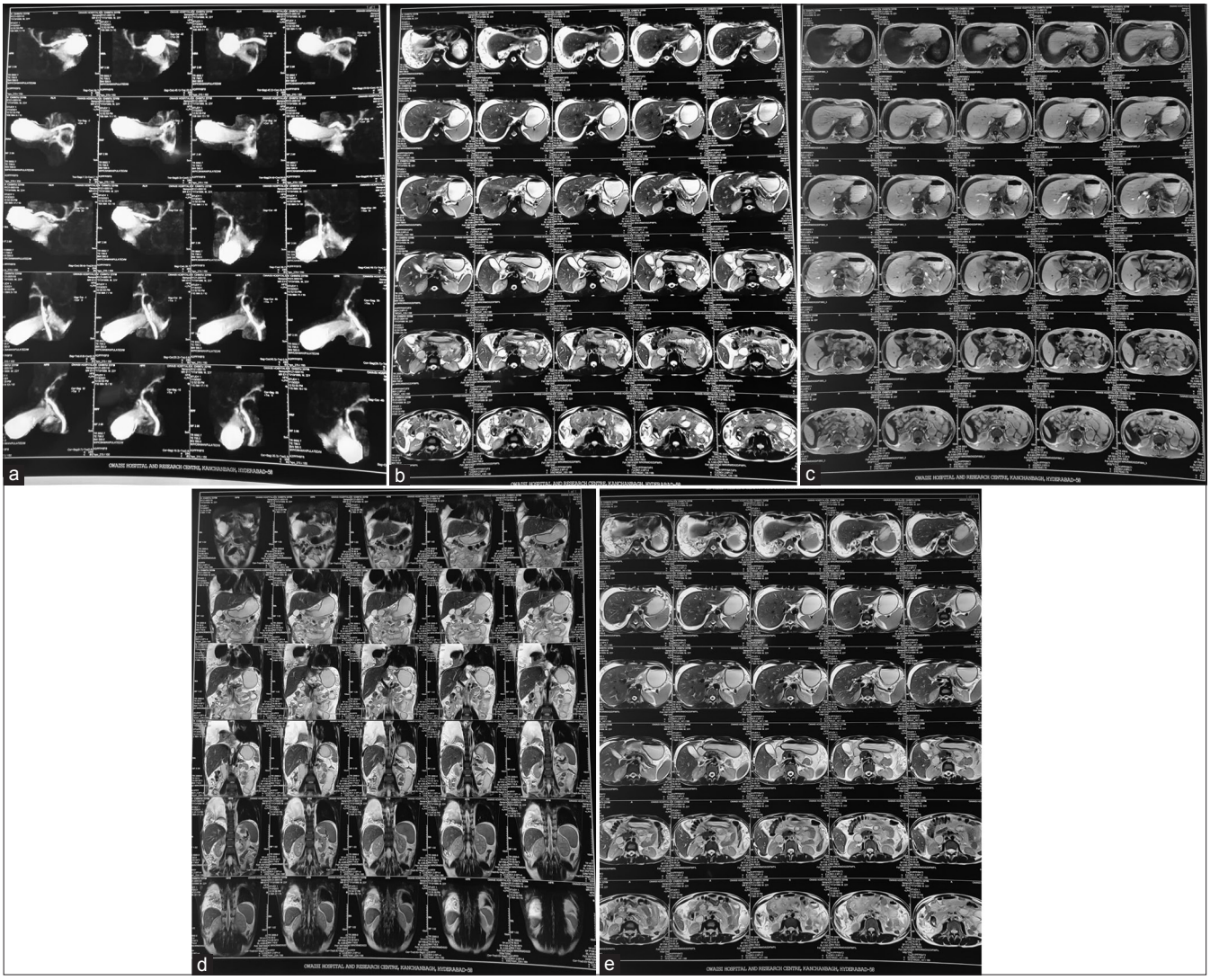

Figure 2: (a) Magnetic resonance cholangiopancreatography, (b-e) MRCP T2 weighted images

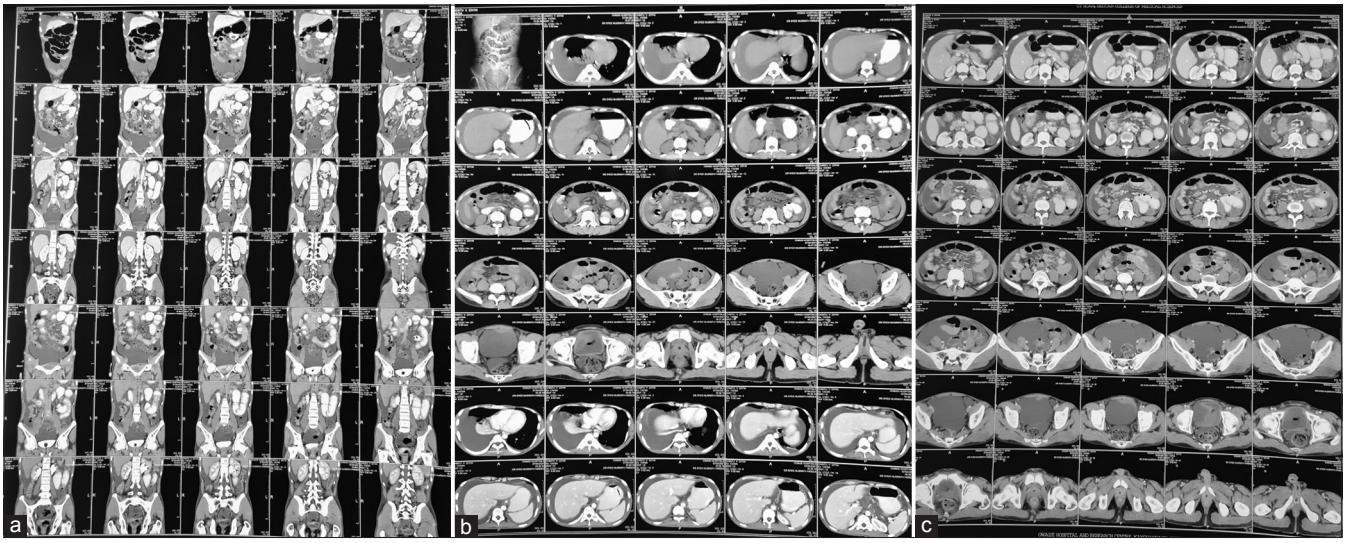

Figure 3: (a) Contrast-enhanced computed tomography abdomen, b and c films show moderate right, left, and non-enhancing well-defined hypodense area (12 × 8 mm) in the neck of the pancreas?

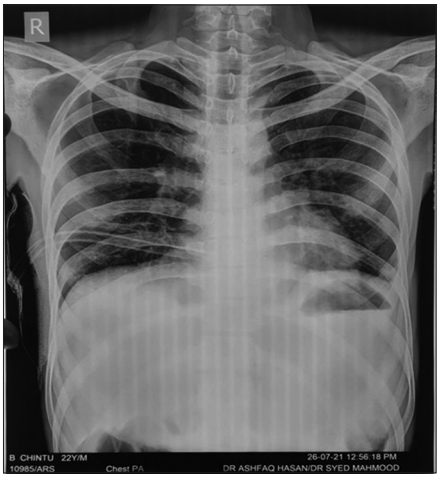

Figure 4: Post-procedure chest X-ray

elevated, with a serum amylase level of 846 U/L and serum lipase level of 401 U/L).

Microbiological analysis of the pleural fluid showed occasional presence of few lymphocytes and no bacteria were seen on gram stain. Smear was negative for AFB and fungal elements.

Cytology report showed 50% lymphocytes, no neutrophils, and 50% non-hematopoietic cells. Centrifuged cytosmears studies showed sheets, clusters of mesothelial cells with reactive changes with nucleomegaly, irregular nuclear margin, prominent nucleoli, and cytoplasmic vacillations. Few mesothelial cells showing window effect were seen in lymphocyte rich proteinaceous background.

Pleural Effusion for CBNAAT was Negative

Blood investigations revealed a total leukocytes count of 12,600/mm^3, RBC count of 4.6 million/mm^3, and platelet count of 3 lakhs/mm^3. The hemoglobin level was 14.1 g/dL.

Viral markers were found to be negative. CRP level was 24 mg/L.

Serum electrolytes were within normal range (Sodium – 140 mmol/L; potassium – 4.2 mmol/L; and chloride – 102 mmol/L). Pancreatic enzymes were elevated, with a serum amylase level of 846 U/L and serum lipase level of 401 U/L. Serum creatinine was 0.8 mg/dL.

Complete urine examination showed presence of pus cells (6–8/HPF) and the other parameters were normal.

Results of his LFT were as follows: Total proteins – 5.2 g/dL ↓; serum albumin – 2.5 g/dL ↓; total bilirubin – 2.2 mg/dL ↑; conjugated bilirubin – 1.7 mg/dL ↑; serum aspartate transaminase – 24 U/L; serum alanine transaminase – 30 U/L; and serum alkaline phosphatase – 246 U/L.

Magnetic resonance cholangiopancreatography [Figure 2] was performed that showed minimal altered signal intensity with suspicious T2 hyperintense area in neck region? Necrotic area? Cyst?

Upper GI endoscopy revealed erosive antral gastritis.

Contrast-enhanced computed tomography abdomen [Figure 3] showed moderate right, left, and non-enhancing well-defined hypodense area (12 × 8 mm) in the neck of the pancreas? Necrotic area? Cyst? laceration, mild prominent distal main pancreatic duct maximum diameter (2.5 mm), gross ascites, adhesions, and internal hernia.

Eventually, an intercostal chest drainage tube [Figure 4] was placed, under aseptic conditions, in the right 2nd intercostal space. Procedure went uneventful, with cessation of symptoms. Approximately 2 L of amber colored fluid was drained.

Discussion

Pleural effusion often occurs as a complication of pancreatic disorders such as acute pancreatitis, pancreatic abscess, pseudocyst, and chronic pancreatitis.[3] The incidence of pleural effusion with acute pancreatitis in older reports was about 3–7%.

Alcoholism is the most common cause of acute pancreatitis in males, accounting for 90% of the cases.[3] Pleural effusion is more commonly observed in men than women with chronic alcoholism.[3] The pathogenic mechanism involved in the formation of the pleural effusion include direct contact of pancreatic enzymes with the diaphragm, hematogenous transfer of pancreatic enzymes into pleura, transfer of pancreatic secretions through transdiaphragmatic lymphatics, and formation of pleuropancreatic fistula which results in direct communication of pancreatic pseudocyst with pleural cavity.[4] Rarely, there may be spontaneous rupture of the pseudocyst into the pleural cavity causing massive pleural effusion.[5] PPFs have been noted in 2.3–4.5% of patients presenting with pancreatic pseudocyst.

In our case also, the diagnosis was initially overlooked as the patient gave a history of predominantly chest symptoms with few abdominal symptoms, and that too on direct interrogation.

The treatment with drainage by an intercostal chest tube, with concomitant conservative treatment of pancreatitis to decrease pancreatic exocrine secretion (nasogastric suction, total parental nutrition, and somatostatin analogs), is usually effective in massive pancreatic pleural effusions.[5]

Surgery is usually resorted to when medical or endoscopic therapy fails.[6]

Conclusion

Right-sided pleural effusion in the setting of pancreatitis is rare. Pancreatitis should be taken into consideration when the serum amylase and serum lipase levels are elevated in the pleural fluid. A delay in the diagnosis will increase the morbidity and mortality. Hence, timely diagnosis is needed.

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