

Massive Recurrent Pleural Effusion in a 35-Year-Old Non-Smoking Male

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Clinical Summary

A 35-year-old non-smoking male presented with exertional breathlessness of 14 days duration. There was no history of cough or fever. A month earlier he took treatment for an acute abdomen. He had a history of alcohol consumption regularly for at least 15 years until one year back. Respiratory system examination showed features of a massive pleural effusion on left side. Abdomen was mildly distended but there was no tenderness or free fluid.

Investigations

Blood examination showed a total leucocyte count of 12240/mm³. Blood chemistry was normal. A chest radiograph (postero-anterior view; Figure 1) and computed tomographic (CT) scan of chest (Figure 2) showed a massive left-sided pleural effusion with a shift of the mediastinal contents to the opposite side. A

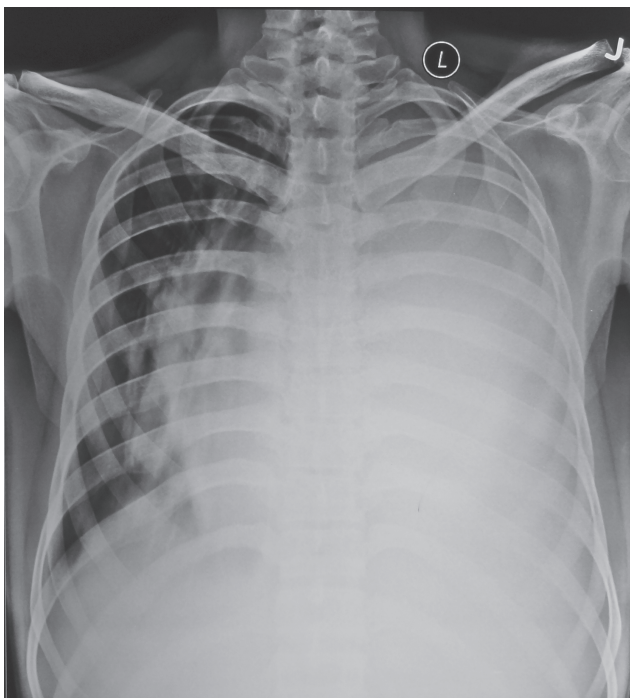


Figure 1. Chest radiograph (postero-anterior view) showing massive left-sided pleural effusion with shift of mediastinal contents to opposite side.

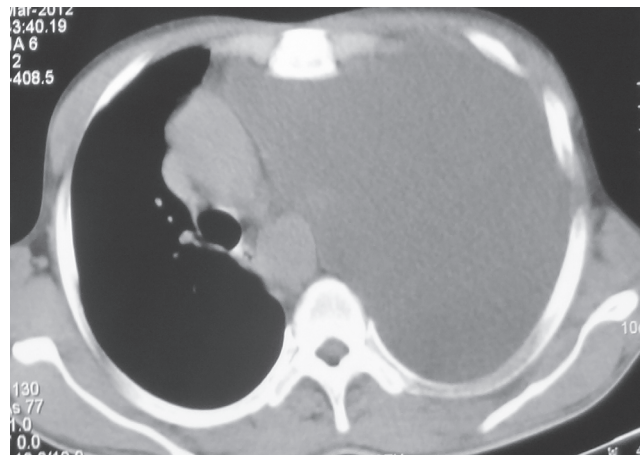


Figure 2. Computed tomographic scan of chest showing massive left-sided pleural effusion with shift of mediastinal contents to opposite side.

diagnostic pleural tap yielded straw-coloured pleural fluid. Pleural fluid protein was 4.5 gm/dL (normal 1-2 g/dL) and lactose dehydrogenase (LDH) was 1639 IU/L (Normal <200 IU/L or <50% of plasma LDH). The cell count of pleural fluid revealed 90% polymorphonuclear leucocytes and 10% lymphocytes. Pleural fluid smears and cultures were negative for pyogenic organisms and acid-fast bacilli. Cytology was negative for malignant cells while adenosine deaminase (ADA) was 69 IU/L (normal <37 IU/L). Therapeutic pleural aspiration was followed by re-accumulation of pleural fluid within three days on two occasions. Abdominal ultrasound done after admission showed an enlarged pancreas with a pseudocyst and ascites. Later, contrast enhanced CT of the abdomen (Figure 3) showed enlarged pancreas with a pseudocyst (7.1 cm × 4.5 cm size) and ascites. Pleural fluid amylase was 2216 IU/L (normal range <150 IU/L) compared to its serum level of 1073 IU/L. Ascitic fluid analysis was not possible as the patient refused further management.

Diagnosis

Left side massive pleural effusion secondary to pancreatic aetiologia with a probable pancreatico-pleural fistula.

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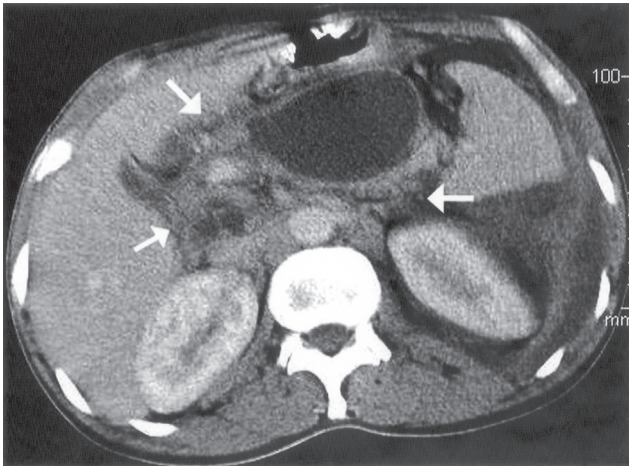


Figure 3. Contrast enhanced CT of abdomen showing enlarged pancreas (white arrows) with pseudocyst and ascites.

Discussion

A massive pleural effusion is a rare complication of pancreatitis due to leakage of pancreatic juice into the pleural cavity from an incompletely formed or ruptured pseudocyst or as a result of direct pancreatic duct leak in some cases.¹ In acute pancreatitis, majority cases of pleural effusions are small and self limiting with amylase levels rarely exceeding 4000 IU/L.² A large recurrent pleural effusion with a high pleural fluid protein (>3 g/dL) and amylase (>1000 IU/L) is characteristic of pancreaticopleural fistula (PPF).³ These were observed in our case. Serum levels of amylase may be raised due to its reabsorption from the pleural fluid across the pleural surface.⁴ A PPF is most often documented in alcohol-induced chronic relapsing pancreatitis with very high level of amylase in pleural fluid and normal or mildly elevated serum amylase.⁵ Our patient was alcoholic and had recovered recently from a possible episode of acute pancreatitis. Ultrasound examination of the abdomen showed pancreatitis with pseudocyst and helped to confirm the cause of massive pleural effusion.

Clinically, a PPF is difficult to suspect unless a large recurrent pleural effusion is associated with history of alcoholism or pancreatitis. The differential diagnosis of high pleural fluid amylase include acute pancreatitis, lymphoma or leukaemia, carcinoma of lung, breast, rectum or female reproductive organs, pneumonia, oesophageal perforation, pulmonary tuberculosis and hydronephrosis.⁶ In immunocompetent individuals, elevated ADA level with lymphocytosis in pleural fluid may suggest tuberculous aetiology. We ignored the raised pleural fluid ADA in our case as it was associated with 90% polymorphonuclear leucocytes in the pleural fluid.

Early abdominal ultrasound in a case of massive pleural effusion and a history of alcoholism should be done, as early diagnosis of pancreatic aetiology is possible. Contrast enhanced CT is the imaging technique

of choice for diagnosis, staging and detecting associated complications of acute pancreatitis.⁷ In our case, contrast enhanced CT showed a diffusely enlarged pancreas and there were no features of chronic pancreatitis like atrophy or ductal calcification.⁸ Ascites may result when the pancreatic secretions collect within the abdomen following disruption of the pancreatic duct or continuous leakage of secretions from the pancreatic pseudocyst. One-third cases have concomitant pleural effusion.⁹ Endoscopic retrograde cholangiopancreatography (ERCP) is useful for both diagnostic and therapeutic purposes including treatment planning and placement of pancreatic stents. The ERCP can confirm the diagnosis of pancreaticopleural fistula in 80% cases and demonstrate a fistulous tract in 59% cases.¹⁰ Magnetic resonance (MR) cholangiopancreatography is a non-invasive alternative to ERCP and can demonstrate the site of PPF and its anatomic relationships as well as the pancreatic pathology.¹¹ These investigations could not be carried out in the present case. The medical therapy of PPF includes adequate drainage of pleural fluid, suppression of exocrine pancreatic secretions by somatostatin or its analogue octreotide and ERCP stenting of the fistulous pancreatic duct.¹² Surgical treatment is indicated for underlying conditions, such as pancreatic stones, ductal strictures or when medical management is unsuccessful.

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