Introduction

Pulmonary complications following acute pancreatitis are well known, and respiratory dysfunction is the leading cause of death following severe acute pancreatitis [1]. Various pulmonary complications of acute pancreatitis include pleural effusion, atelectasis, consolidation, pulmonary infiltrates, ARDS, respiratory failure. Haemoptysis has been reported less frequently after acute pancreatitis. We report on one such case.

Case report

A 40-year-old male presented with right-sided chest pain, breathlessness, cough, and haemoptysis for one week. There was no associated palpitation, sweating, abdominal pain, or loss of appetite. There was no other relevant history except that the patient was an alcoholic and known to have pancreatitis. The patient was well-oriented, with vitals stable and within normal limits (pulse rate and blood pressure being 76/min and 110/72mmHg respectively). There was decreased air entry in the right lung base with occasional ronchi at the same place. The patient was anaemic (haemoglobin 11mg/dl) with normal white cell and platelet counts. He was hypoxemic with PaO2 of 82mmHg. Abdominal sonography was unremarkable except for minimal right-sided pleural effusion. A chest X-ray showed opacity in the right lower zone (Figure 1). A CT scan showed normal abdominal organs, encysted hydropneumothorax along the mediastinal border on the right side (Figure 2), and patchy ground-glass opacity in the middle and right lower lobes, suggesting alveolar haemorrhage (Figure 3). Renal and liver functions were within normal limits. Serum c-ANCA and p-ANCA levels were normal. Anti-glomerular basement membrane antibody and ANA (anti-nuclear antibody) were negative. Urine examination was normal. The patient underwent fibreoptic bronchoscopy, which revealed active bleeding through bronchus intermedius. No mass was seen. Bronchoalveolar lavage (BAL) was negative for acid-fast bacilli, and the culture of the BAL fluid was sterile. No malignant cells were seen on the cytology of the BAL fluid. Blood coagulation profile was normal. There was no history of any drug intake. Serum amylase was also within the normal limit. A small amount of pleural fluid was aspirated,
which showed raised amylase level (469 U/L). The patient had a history of recurrent pancreatitis over the last three to four years, the last reported incidence being about five or six months ago, during which an MRI of the abdomen was done and showed acute pancreatitis with right pancreatico-pleural fistula (Figure 4 (a & b)) and right pleural effusion. The amylase level in the pleural fluid at that time was 55380 U/L. In the meantime (from five or six months ago to the present episode of haemoptysis), the patient was absolutely symptom-free. As no other cause for alveolar haemorrhage and haemoptysis could be ascertained for the present episode, it was attributed to previous pancreatitis. Though the patient reported recurrent previous episodes of pancreatitis, there were no changes of chronic pancreatitis (like atrophy, dilatation of the pancreatic duct, or calcification) in the present CT scan. The patient was given octreotide and observed. Haemoptysis and other symptoms subsided in a week. The patient was discharged and has been doing well since then.

Discussion
Pulmonary dysfunction is the most important systemic manifestation of severe acute pancreatitis. It occurs in 30% to 50% of patients and ranges from pleural effusion, atelectasis, consolidation, pulmonary infiltrates, and ARDS to respiratory failure [2].

Pancreatic enzymes and inflammatory mediators have been implicated in pulmonary complications of acute pancreatitis, with current research focusing more on the latter. Activated trypsin damages pulmonary vasculature and increases endothelial permeability. Serum level of activated phospholipase A2 (PLA2) correlates strongly with pulmonary insufficiency. PLA2 releases platelet-activating factor (PAF) from membrane lipids. PAF antagonist lexipafant reduces organ failure by reducing oxidative injury, morphologic changes, white cell infiltration, and vascular permeability in pancreas and lungs, thereby implying the role of PAF in organ failure. Free fatty acids released from triglycerides damage capillary alveolar wall membranes. Activated neutrophils are attracted to the pulmonary vasculature, and chemoattractants such as TNF-alpha, IL-1, IL-6, IL-8, fMet-Leu-Phe and complement factor c5a upregulate the beta-2-integrin expression on neutrophils and ICAM-1 receptor on endothelial cells. The binding of ICAM-1 to beta-2-integrin can increase permeability of the pulmonary vasculature [3]. These circulating inflammatory mediators cause damage to pulmonary vasculature, and can readily explain haemoptysis following acute pancreatitis.

Haemoptysis has also been reported following chronic pancreatitis [4,5], but the cause in both reports has been vascular complication (formation of inferior phrenic-pulmonary artery anastomosis in one, and splenic artery pseudoaneurysm communicating with the lung in another). Alveolar haemorrhage was observed in neither of them.

Our case is quite unique. Though the patient experienced recurrent episodes of acute pancreatitis in the past, there were no morphologic features of chronic pancreatitis. Also, at the time of the present episode of haemoptysis, there was no clinical sign of acute pancreatitis. Nor was there any rise in serum amylase level. Hence, our patient had neither evidence of acute pancreatitis nor morphologic features of chronic pancreatitis at the time of the haemoptysis episode. He showed only diffuse alveolar haemorrhage. So although the aetiology was pancreatitis, the pathophysiology remained elusive. Probably some entrapped inflammatory mediators in the remaining right encysted hydro pneumothorax (reminiscent of previously reported pancreaticocpleural fistula) may have caused delayed alveolar haemorrhage (also on right side) and haemoptysis. This was substantiated by a raised level of amylase in the tapped pleural fluid.

Figure 2. Axial section of contrast-enhanced CT scan at cardiac level showing encysted hydro pneumothorax along the mediastinal border of the right lung (arrow).

Figure 3. Axial CT section at the same level as Figure 2 showing patchy ground-glass opacity in the middle and right lower lobes.
In conclusion, as exemplified by our case, haemoptysis can also occur as a delayed complication of acute pancreatitis. In cases of haemoptysis where no cause can be ascertained after a thorough work-up, a history of pancreatitis should be sought: acute or chronic, recent or remote.

**References**


**Figure 4 (a & b).** Axial single shot T2W and coronal balanced TFE MR images showing right-sided pancreaticopleural fistula (arrow).