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Pulmonary embolism and acute pancreatitis: Case series and review

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ABSTRACT

Reports of pulmonary embolism in the setting of acute pancreatitis are rare. We present three cases of acute pancreatitis associated with pulmonary embolism and review the literature. Two of the three patients had severe acute pancreatitis with bilateral pulmonary emboli, and to our knowledge, these cases represent the first report of pulmonary embolism occurring in the setting of pancreatic ascites and pleural effusion. All patients experienced abdominal pain, though in one patient, symptoms suggestive of a pulmonary embolism were lacking. All three patients were successfully treated with unfractionated heparin and conservative management. Pulmonary thrombosis may occur in the setting of severe acute pancreatitis as the result of systemic inflammatory response. We review the literature and provide microvascular explanations for the occurrence of pulmonary complications and thrombosis in the setting of acute pancreatitis. We also review prior cases of pulmonary embolism in acute pancreatitis. Our experience suggests that pulmonary embolism may be an under-recognized complication of severe acute pancreatitis.

Keywords: Pulmonary embolism, pancreatitis, ascites, pleural effusion, thrombosis

Case Report

INTRODUCTION

Severe acute pancreatitis can provoke a systemic inflammatory response which may lead to a number of vascular and pulmonary complications. Pulmonary embolism is a rarely reported complication of acute pancreatitis (1-3). Here we present three cases of acute pancreatitis associated with pulmonary embolism, and propose that pulmonary vascular thrombosis may be an under-recognized complication of severe acute pancreatitis. To our knowledge, two of the three cases represent the first reports of pulmonary embolism occurring in the setting of pancreatic ascites and pleural effusion.

CASE PRESENTATIONS

Case 1

A 28 year old woman presented with six weeks of abdominal pain along with nausea and vomiting. The patient had a history of alcoholic pancreatitis but denied recent alcohol consumption. She reported no chest pain or shortness of breath. Initial vital signs included a temperature of 98.7 F, blood pressure of 122/87 mmHg, and she was tachycardic with a heart rate of 132 beats/min. Oxygen saturation was 95% on room air. Physical examination was remarkable for epigastric tenderness and abdominal distention consistent with ascites. Lipase was 1,453 IU/L, and liver enzymes were normal. Ascitic fluid analysis showed an amylase of 43,400 IU/L. CT scan of the abdomen and pelvis showed ascites and evidence of prior cholecystectomy. Incidental, multiple pulmonary emboli were seen in bilateral lower lobes, along with a right-sided pleural effusion and lower lobe consolidation with abnormal enhancement, consistent with pulmonary infarct (Figure 1). The pancreas appeared normal.

The patient was started on unfractionated heparin along with intravenous hydration and bowel rest. Her abdominal pain and nausea quickly improved, and she tolerated an oral diet within three days. She was discharged with low-molecular weight heparin and warfarin.

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Case 2

A 32 year old male presented to the emergency department complaining of abdominal pain. For 15 years, he was a heavy alcohol abuser and typically drank a pint of vodka three times weekly. Four days prior to admission, he began to experience nausea and vomiting, with non-bloody diarrhea and abdominal distention. He also complained of dyspnea over the prior two days and had a single episode of hemoptysis.

On presentation, the patient had a temperature of 100.1 F, blood pressure 132/87 mm/Hg and a heart rate 105 beats/min. His respiratory rate was 28 breaths/min, and his oxygen saturation was 98% on room air. Breath sounds were decreased bilaterally; abdomen was distended but non-tender. His initial chemistries were notable for a sodium of 129 mEq/L, total bilirubin of 4.0 mg/dL, AST 49 IU/L, ALT 33 IU/L, alkaline phosphatase 89 IU/L, amylase 1105 IU/L and lipase 809 IU/L. His initial WBC was 15,000 cells/microL with 80% neutrophils. On imaging, he was found to have bilateral pulmonary emboli, pleural effusions and ascites (Figure 2). Diagnostic paracentesis showed an amylase of 3,570 IU/L. Diagnostic thoracentesis showed an exudative effusion, with a total protein of 2.4 g/dL, lactase dehydrogenase of 377 IU/L and an amylase of 6,302 IU/L.

The patient was started on unfractionated heparin and treated with intravenous hydration, antibiotics and bowel rest. Following 10 days of conservative management, he began to tolerate enteral nutrition and was discharged after 18 days of hospitalization.

Case 3

A 21 year old woman presented with chest tightness and shortness of breath. At the time of presentation, she also complained of shortness of breath along with mid-back and abdominal soreness. Of note, she had undergone emergent craniotomy one week prior for rupture of a congenital arteriovenous malformation (AVM). Prior to surgery, she had been taking oral contraceptive hormones.

In the emergency department, her initial vitals were as follows: temperature 97.9 F, blood pressure 137/91 mm/Hg, heart rate 118 beats/min, and respiratory rate 20/min. Oxygen saturation on room air was 94%. Physical examination was notable only for mild right upper quadrant and epigastric tenderness to palpation. Abnormal laboratory values included elevated transaminase levels (AST 318 IU/L and ALT 374 IU/L), alkaline phosphatase of 202 IU/L and a lipase of 121 IU/L. D-dimer was 911 mcg/L. A right upper quadrant ultrasound revealed cholelithiasis without cholecystitis. A chest x-ray was unremarkable, and CT of the chest revealed a filling defect in the right lower lobe, consistent with pulmonary embolism. The patient was started on unfractionated heparin and then transitioned to low-molecular weight heparin and warfarin, and was later referred for elective cholecystectomy.

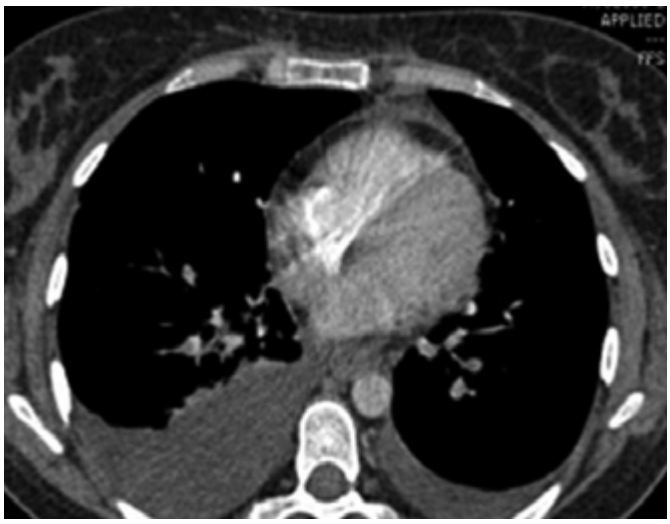

Figure 1. Bilateral pleural effusions and pulmonary emboli in multiple vessels of bilateral lower lobes.

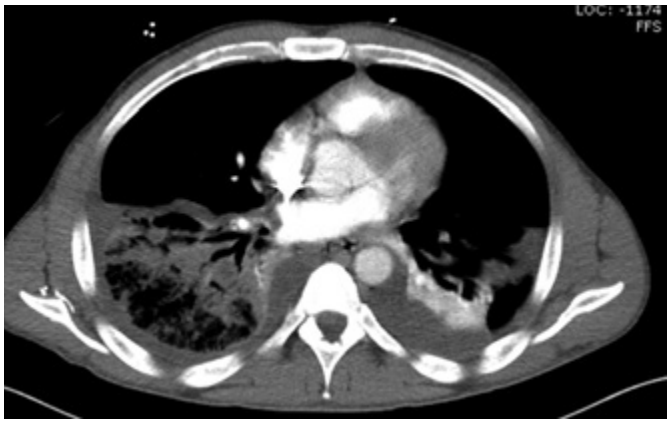

Figure 2. Pulmonary embolus in right lung with bilateral pleural effusions.

DISCUSSION

In this report, we have described three cases of acute pancreatitis with concomitant pulmonary embolism, and our experience suggests that pulmonary embolism may be an underrecognized complication of acute pancreatitis. Indeed, in two of our cases, extensive pulmonary emboli were found incidentally after the diagnosis of severe acute pancreatitis was already made. Also, to our knowledge, this is the first reported series of patients with pulmonary embolism in the setting of pancreatic ascites and pleural effusion.

Vascular complications of pancreatitis are a poorly understood but commonly described phenomenon (4,5). Venous thrombosis, a known complication of acute pancreatitis, most commonly involves the portal, splenic and splanchnic veins (5,6) Much less commonly, extrasplanchnic involvement has been reported in the pulmonary vasculature (2,3,7), renal vein (2,8,9), and in a remarkable case, multiple sites including the superior and inferior vena cavae, bilateral subclavian veins, internal jugular and iliac veins (10). In two post-mortem studies, intravascular thrombosis has been detected among pathologic specimens from the lung (1,11), though the significance of these thromboses is unclear.

Several explanations have been proposed for these occurrences. The systemic inflammatory response associated with severe acute pancreatitis results in a pro-thrombotic state which makes vascular events more likely. At a microvascular level, this mechanism of vascular injury is likely multifactorial and may reflect intrinsic endothelial damage from inflammatory mediators along with extrinsic damage from adjacent edema or fibrosis (6). The release of pancreatic enzymes may in turn further promote a hypercoagulable state from pancreatic enzyme release, activating clotting processes within the inflamed pancreas and leading to thrombophilic changes in venous blood. In acute pancreatitis, levels of fibrinogen and D-Dimer are raised, and the overall platelet level is increased (11,12). Furthermore, disturbances in the coagulation cascade likely lead to further hypercoagulability and activation of fibrinolysis (13).

In cases of pulmonary embolism, several mechanisms of injury have been found. Several reports have described extension from a renal vein thrombosis into the inferior vena cava, both in the absence and the presence of pulmonary emboli (2,7,8,14). In one of these cases, the renal vein thrombosis occurred secondary to a pseudocyst rupture (2). Elsewhere, Asanuma described bilateral pulmonary emboli in post-operative patients with acute necrotizing pancreatitis, though this was a late finding and may have reflected a post-operative complication (1). Finally, in a recent report of severe acute pancreatitis with symptomatic bilateral pulmonary emboli, no clear mechanism was elucidated (3).

Two of the three patients reported here were found to have pulmonary emboli in the setting of pancreatic ascites and pleural effusion. Pulmonary complications of acute pancreatitis are among the most common of systemic complications, ranging from hypoxemia to the adult respiratory distress syndrome (15). In one prospective study (16), 50% of individuals with pancreatitis also had pleural effusions, though elsewhere a much lower prevalence (10%-20%) has been noted (15). Together, pancreatic ascites and pleural effusion, which results from transdiaphragmatic lymphatic blockage or pancreaticopleural fistulae, are both associated with a mortality rate of 20%-30% (17), reflecting the severe inflammatory response in which these conditions arise. We hypothesize that this severe response may have in turn led to the pulmonary emboli we observed in our patients, though predisposing factors remain unclear in this unusual presentation.

Pulmonary embolism is an infrequently reported manifestation of severe acute pancreatitis. Newer and improved imaging modalities may already be leading to increased diagnosis of thromboembolic events, as recent case reports suggest. Nonetheless, thromboembolic events are likely an under-recognized complication of acute pancreatitis, and a high index of suspicion is warranted for diagnosing pulmonary embolism in patients with severe acute pancreatitis.

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