Received: 23 January 2022 | Accepted: 26 February 2022

DOI: 10.1002/rcr2.929

CASE REPORT

Respirology Case Reports OPEN ACCESS APSR WILEY

Acute-on-chronic pancreatitis complicated with mediastinal pseudocysts and cardiac tamponade: A case report and literature review

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Associate Editor: Sita Andarini

Abstract

The clinical course and severity of pancreatitis might vary largely. Pancreatitis-related thoracic complications might be life-threatening but frequently ignored. We report an alcoholic patient who initially presented to the emergency department with community-acquired pneumonia, acute respiratory failure and acute-on-chronic pancreatitis with massive pancreatic pleural effusion. Subsequently, he developed insidiously pancreatitis-related intra-abdominal, mediastinal pseudocysts, and unexpectedly sudden onset of cardiac tamponade. Although tamponade-related haemodynamic instability improved soon after timely diagnosis and emergent pericardial drainage, his recovery period was prolonged. His serum amylase and lipase were persistently elevated until definitive treatment with endoscopic retrograde cholangiopancreatography-assisted removal of pancreatic duct stones. Pancreatitis-related cardiac tamponade is rare but lethal without prompt diagnosis and management. We reviewed pancreatitis-related thoracic complications, particularly for cardiac tamponade, and discussed about the pathophysiology and management options.

KEYWORDS
cardiac tamponade, mediastinal pseudocyst, pancreatitis, pleural effusion

INTRODUCTION

Acute pancreatitis can result in systemic inflammation and multiple organ dysfunction (respiratory, cardiovascular or renal).[1] Thoracic complications associated with acute and/or chronic pancreatitis, such as pleural, pericardial effusion and acute lung injury, vary largely in severity.[2] Unawareness of the potentially life-threatening thoracic complications might lead to poor outcomes. We reported a case who initially presented with acute respiratory failure and acute-on-chronic pancreatitis complicated by massive pancreatic pleural effusion, followed by insidious development of pancreatitis-related cardiac tamponade during the recovery period.

CASE REPORT

A 57-year-old man presented to our emergency department (ED) with high fever (39.4°C) and 3 days of dyspnoea and abdominal pain. He was a heavy smoker and chronic alcoholic. In recent years, he had experienced several hospitalizations for acute pancreatitis and recovered after conservative treatment. However, this time, he developed consciousness disturbance, respiratory distress and haemodynamic instability soon after arrival at ED. He was intubated and admitted to the intensive care unit. Physical examinations revealed diffuse wheezing in bilateral lungs and epigastric rebound tenderness without concurrent guarding of abdomen. Blood tests were unremarkable except for elevated amylase (1127 U/L, reference <125), lipase (1481 U/L, reference <78), C-reactive protein (13.16 mg/dl, reference <0.5) and respiratory acidosis (arterial blood gas: pH 7.165, partial pressure of carbon dioxide [$PaCO_2$] 88.7 mmHg, HCO_3^- 31.3 mmol/L). He was managed as community-acquired pneumonia with acute respiratory failure and acute-on-chronic pancreatitis based on the image findings of chest x-ray (CXR; Figure 1A) and abdominal contrast-enhanced computed tomography (CECT; Figure 2A). Left-sided pleural effusion was percutaneously drained and found to be pancreatic exudate

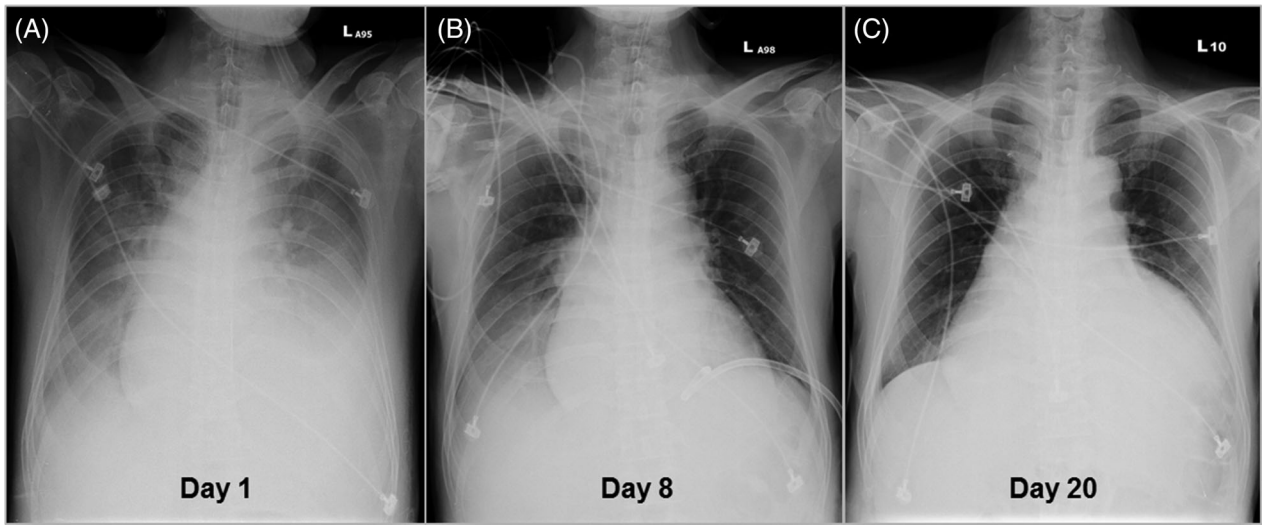

FIGURE 1 Serial changes of supine chest x-ray (CXR). On day 1, CXR reveals endotracheal tube in the trachea and opacification in the right lower lung and massive left-sided pleural effusion (A). On day 8, CXR indicates partial regression of opacification in the right lower lung and marked decline in the amount of left-sided pleural effusion after pig-tail drainage (B). On day 20, CXR shows nearly complete regression of opacification in the right lower lung, but an enlarged cardiac silhouette (C)

(amylase 24,364 U/L, leucocytes 3670/mm^3, neutrophils 76%, lymphocytes, 13%). His vital signs were stabilized after treatment with inotropic agents and empirical broad-spectrum antibiotics. He stayed fasting until relief of abdominal pain on day 5. Workup for malignancy and microorganisms showed negative results. On day 8, CXR improved (Figure 1B) and he was extubated successfully. However, intermittent epigastric tenderness and vague chest tightness gradually developed and serum amylase peaked (3144 U/L; Figure 2E) again on day 10. On day 20, sudden-onset cold sweats, orthopnoea, tachycardia (125/min) and hypotension (55/22 mmHg) occurred. CXR disclosed significantly enlarged cardiac contour (Figure 1C). Blood tests were unremarkable except for persistently high amylase/lipase (1215/968 U/L; Figure 2E). Emergency echocardiography indicated cardiac tamponade (massive pericardial effusion with diastolic right ventricular collapse). He was haemodynamically stabilized after drainage of 1000 ml of pancreatic pericardial fluid (amylase 82,132 U/L). On day 25, chest CECT (for persistent chest distress despite pericardial drainage) revealed a dilated pancreatic duct (PD), swollen pancreas (indicating interstitial oedematous pancreatitis, IEP) and formations of homogenous acute peripancreatic fluid collection (APFC), mediastinal pseudocysts and suspicious pancreaticopericardial fistula (Figure 2B). Because infectious fluid collections were deemed unlikely based on clinical and radiological findings, neither surgical intervention nor endoscopic-guided drainage was performed until well-defined fluid collections were formed. His general condition gradually improved with conservative treatment. On day 47, follow-up abdominal CECT revealed resolution of mediastinal pseudocysts, but persistently dilated PD and formations of large intra-abdominal pseudocysts (Figure 2C). Thus, endoscopic retrograde cholangiopancreatography (ERCP) was performed on day 51, revealing a patent bile duct but an obstructed PD due to stones, which were removed, followed by plastic stenting. On day 58, abdominal CECT revealed regressed pseudocysts (Figure 2D). Moreover, the sustained elevated serum amylase/lipase was finally normalized (Figure 2E). He was discharged without further complications on day 67, and remained uneventful at a 6-month follow-up period.

DISCUSSION

Thoracic complications of pancreatitis might involve the pleura, lungs, pericardium, mediastinum and great vessels. Acute respiratory distress syndrome is the most well-known lethal complication. Other thoracic complications include pleural effusion, pericardial effusion, thoracopancreatic fistula, mediastinal pseudocyst, mediastinitis, pulmonary embolism and thoracic aortic aneurysm.[2] The occurrence of ascites, pleural and pericardial effusion is common in pancreatitis, but they are rarely clinically significant and usually resolve spontaneously as the inflammation subsides.[2,3] Pancreatic pleural effusion, the most easily recognized effusion, has two types of clinical manifestations.[2,4] The first type is usually associated with acute pancreatitis and the pleural effusion is reactive, left-sided, small to moderate and might subside during recovery. The second type is usually found in chronic pancreatitis. The effusion could be single-sided or bilateral, large and recurrent if there is no definite treatment. The main pathogenesis includes transdiaphragmatic lymphatic blockage and the formation of pseudocyst or pancreaticopleural fistula following the disruption of PD resulting from trauma, pancreatitis, stone- or stenosis-related increase in ductal pressure.[2,5,6] The leakage of pancreatic enzymes through the ruptured duct might contribute to ascites (if peritoneal cavity is involved) or pleural effusion (if there is upward communication to pleural cavity). Pancreatic ascites and pleural effusion were reported to be associated

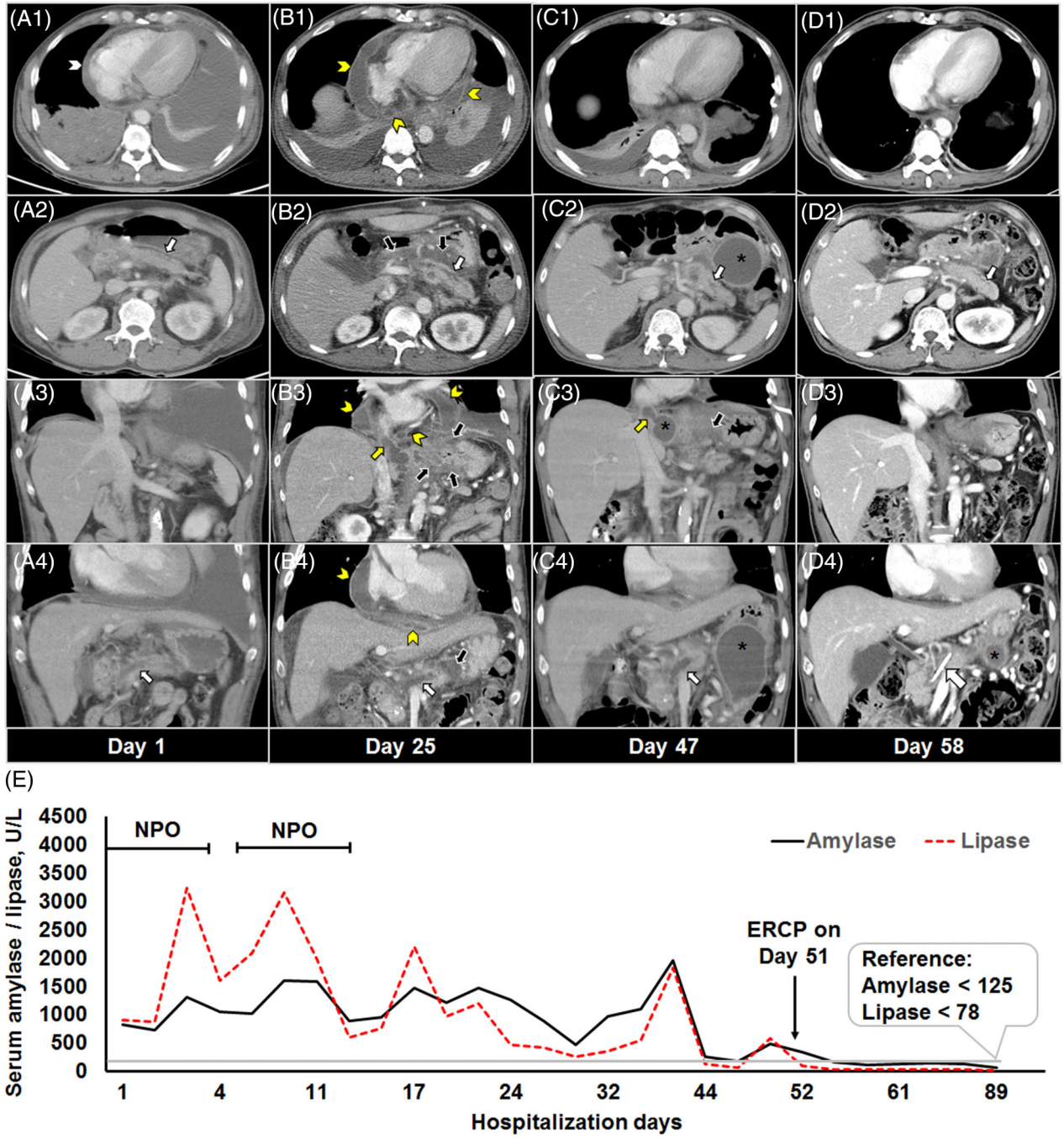

FIGURE 2 Serial CECT images of axial and coronal views at different timings. The right lower lobe is consolidated (A1). The peripancreatic fat stranding (A2–A3–A4) and some tiny punctate calcifications in the pancreatic head (not shown) indicate acute-on-chronic pancreatitis. The amount of left-sided pleural effusion was massive with nearly total collapse of the left lower lobe, while pericardial effusion (white arrowhead, A1) was small on day 1, but was considerably increased and encapsulated on day 25, indicating the formation of mediastinal pseudocysts (yellow arrowheads, B1, B3, B4), which later gradually resolved on day 47 (C1) and day 58 (D1). The PD (white arrow) was persistently dilated from day 1 to day 47 (A2–B2–C2, A4–B4–C4); PD dilation subsided on day 58 (D2, D4) after the placement of a stent (large white arrow, D4) in the place of PD on day 51. The pancreas was swollen with APFC (black arrows, B2–B3–B4) accumulation on day 25, and subsequent formation of multiple separate intra-abdominal pseudocysts (*, C2–C3–C4), which resolved on day 58 (D2–D3–D4). Note that the fluid component was homogenous without solid or air density. Some intra-abdominal pseudocysts extended upward to the mediastinum (yellow arrows, B3, C3), suggesting pancreaticopericardial fistula formation along the inferior vena cava. Sustained elevation of serum amylase and lipase definitely subsided after ERCP on day 51 (E). APFC, acute peripancreatic fluid collections; CECT, contrast-enhanced computed tomography; ERCP, endoscopic retrograde cholangiopancreatography; NPO, nothing per os; PD, pancreatic duct

with disease severity. By contrast, it seemed not the case for pancreatic pericardial effusion.[3,7] However, pancreatitis-related cardiac tamponade is extremely rare but life-threatening.[8,9] Its pathogenesis remains poorly elucidated. The hypothesized mechanism includes pericarditis due to irritation by pancreatic enzymes, formation of pancreaticopericardial fistula and

necrosis of vascular walls in fatty necrosis area in the subpericardium.[10] Cardiac tamponade can be a complication of acute or chronic pancreatitis, and the risk increases in patients with a history of alcoholism, chronic pancreatitis and pseudocyst formation.[11] The presence of ascites, pleural and pericardial effusion earlier is related to an increased incidence of pseudocysts later.[3] Pseudocysts, often associated with chronic pancreatitis, are mainly located in peripancreatic regions and are rarely seen in the mediastinum. Mediastinal pseudocysts are usually the consequence of pancreaticopericardial fistulous communication and are associated with an increased incidence of cardiac tamponade.[2,11,12] The common symptoms of mediastinal pseudocyst include chest tightness, atypical chest pain and dysphagia. If cardiac tamponade occurs, orthopnoea, dyspnoea and palpitation may develop, followed by unexpected haemodynamic compromise.[12] The management of cardiac tamponade depends on rapid diagnosis of emergency pericardial drainage and treatment of the underlying condition.

The revised Atlanta classification[13] categorizes acute pancreatitis as IEP and necrotizing pancreatitis (NP) according to the absence or presence of necrosis, respectively, which is observed as non-enhancing areas in the pancreas on CECT. Intra-abdominal acute fluid collection, termed APFC or acute necrotic collection (ANC) in IEP or NP, respectively, may occur within 4 weeks after the onset of symptoms. Furthermore, encapsulated fluid, termed pseudocyst or walled-off necrosis (WON) in IEP or NP, respectively, may develop after 4 weeks. The fluid consistency is homogeneous in APFC/pseudocyst but heterogeneous, possibly accompanied by non-liquefied components, in ANC/WON. The levels of serum pancreatic enzymes and the extent of pancreas necrosis or fluid collection do not directly correlate with the severity of organ failure. Usually, the fluid collection does not require treatment unless symptomatic or infected. Progressive abdominal pain, fever, leucocytosis and presence of air bubbles in the necrotic tissue or fluid collection are characteristic of infection. APFC, pseudocyst and early-stage ANC (<2 weeks) are commonly sterile. ANC may become infected after 2 weeks. Management of fluid collection is usually delayed for 4 weeks to allow the formation of a well-circumscribed wall, which demarcates the boundary between the healthy and diseased tissues. Pseudocyst/WON rarely resolves spontaneously. Compared with traditional open surgery, minimally invasive endoscopic interventions, such as endoscopic ultrasound-assisted endoscopic drainage or ERCP-assisted transpapillary stenting, have fewer major complications and are currently the main treatment modalities.[1,14]

In the present case, alcoholism and chronic pancreatitis were the risks for cardiac tamponade. The sudden-onset cardiac tamponade during recovery from respiratory failure implied an unexpected clinical course. Serial images revealed the insidious formation of pseudocysts. Thus, clinicians should develop a high awareness of this life-threatening complication and monitor images frequently for early detection and management. Once pseudocysts are formed, early intervention can help avoid pseudocyst-related complications and shorten the length of hospital stay.

ACKNOWLEDGMENT

We thank the Wallace Academic Editing for English editing of this manuscript.

CONFLICT OF INTEREST

None declared.

AUTHOR CONTRIBUTION

Ying-Ting Liao: Draft writing. Nai-Chi Chiu and Chun-Ku Chen: Interpretation of images and preparation of figures. Kang-Cheng Su: Critical revision of the manuscript for important intellectual content.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ETHICS STATEMENT

The authors declare that appropriate written informed consent was obtained for the publication of this manuscript and accompanying images.

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How to cite this article: Liao Y-T, Chiu N-C, Chen C-K, Su K-C. Acute-on-chronic pancreatitis complicated with mediastinal pseudocysts and cardiac tamponade: A case report and literature review. Respirology Case Reports. 2022;10:e0929. https://doi.org/10.1002/rcr2.929