

Abdominal Aortic Aneurysm Presenting with Acute Pancreatitis: A Rare Clinical Manifestation

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Abstract

Acute pancreatitis is an inflammatory condition of the pancreas associated with elevated pancreatic enzymes, mainly caused by gallstones and alcohol abuse. In this article, we report a rare case of an 82-year-old woman with abdominal aortic aneurysm (AAA) and acute pancreatitis. The patient presented with typical acute pancreatitis symptoms and AAA was diagnosed by further imaging. This study underlines the mechanical and inflammatory interactions between AAA and acute pancreatitis and highlights the potential for concurrent presentation, including aneurysm rupture or recurrent pancreatitis. It emphasises the importance of advanced imaging and multidisciplinary management in handling such cases. Further research is recommended to uncover pathophysiological interactions and optimise clinical outcomes for such comorbid conditions.

Keywords: Abdominal aortic aneurysms, acute abdomen, pancreatitis

Introduction

Acute pancreatitis (AP) is an inflammatory condition of the pancreas, characterised by abdominal pain and elevated levels of pancreatic enzymes in the blood (1,2). A comprehensive understanding of the etiology of acute pancreatitis is imperative for the effective management and prevention of the disease. The most prevalent aetiological factors contributing to acute pancreatitis are gallstones and alcohol misuse, which collectively account for approximately 75% of cases. Hypertriglyceridaemia (HTG) and endoscopic retrograde cholangio pancreatography (ERCP) have also been identified as significant aetiological factors (3,4). Genetic mutations, including those in the PRSS1 and SPINK1 genes, have been identified as risk factors for the development of pancreatitis. Autoimmune pancreatitis, although rare, is another recognised cause. Certain medications, including azathioprine and valproic acid, have been implicated in causing pancreatitis, though they account for less than 5% of cases (5). The etiology of pancreatitis encompasses a wide spectrum of factors, including hypercalcemia, infections, and abdominal trauma. Acute pancreatitis is characterised by the failure

of protective mechanisms against trypsinogen activation, resulting in the activation of enzymes within the pancreas, auto-digestion, and subsequent inflammation. This process can lead to both local and systemic inflammatory responses, which may ultimately result in multiple organ dysfunction syndrome (MODS) (6,7).

Case Report

An 82-year-old female patient presented to the emergency department with abdominal pain and nausea. The patient had been living with hypertension for 15 years, hypothyroidism for 15 years, and cardiomegaly and atrial fibrillation for 2 years. A physical examination of the patient revealed that their general condition was satisfactory. Their level of consciousness was clear, and they were oriented and coherent. Vital signs revealed a blood pressure of 137/69 mmHg, a pulse rate of 87/min, and finger tip oxygen saturation of 91%, all of which were stable. Abdominal examination revealed tenderness in the upper quadrants of the abdomen. There was no history of previous abdominal operation and no scar. Biochemical tests showed amylase 1152 U/L, lipase 2215 U/L, total bilirubin 0.61, and liver function tests

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and other parameters were within the normal range. White blood cell count was 11,000/ μ L, haemoglobin was 12.7 and platelet count was 184,000/ μ L. The gallbladder exhibited signs of distension, with a diameter measuring 35 mm, and the presence of freefluid was observed in the pericholecystic region. Millimetre-sized hyperdense appearances were detected within the gallbladder lumen (possibly indicating calculi). Intra- and extrahepatic bile ducts are normal (Figure-1). The pancreas manifested normal dimensions and anatomical location, with a smooth internal structure and lobulated external contours. Contamination was observed in the fatty planes of the peripancreatic area (Figure-2). Contrast-enhanced computed tomography revealed that the dilatation of the aneurysm measured up to 76 mm at its widest point at the level of the terminal abdominal aorta (Figure-3). The patient was seen in the gastroenterology, general surgery and cardiovascular surgery (CVS) clinics. Urgent surgical intervention was considered and the patient was admitted to the CVS clinic for aneurysm surgery. The postoperative image, taken one month after the operation, is illustrated in Figure-4.

Discussion

Abdominal aortic aneurysm (AAA) is a serious vascular condition characterised by the dilation of the abdominal aorta. In the event of rupture, this condition can lead to life-threatening complications. It is most prevalent in older adults, particularly men over the age of 65, and is associated with high mortality rates upon rupture. It is important to note that AAAs are frequently asymptomatic and are often discovered incidentally through imaging studies such as ultrasound, CT, or MRI (8). Surgical repair, either through open surgery or endovascular aortic repair, is the primary treatment for large or symptomatic aneurysms. Elective



Figure 2. Contamination in the fatty planes of the peripancreatic area



Figure 3. The aneurysm at the level of the terminal abdominal aorta



Figure 1. Normal intra-extrahepatic bile ducts



Figure 4. The postoperative image, one month after the operation

repair is generally considered to be the preferred option due to its proven track record of superior outcomes when compared with emergency repair. However, it should be noted that both methods carry an inherent risk of morbidity and mortality (9). Anatomically and medically speaking, an AAA and pancreatitis are two distinct pathologies that can result in intricate clinical scenarios. AAA is characterised by the dilatation of the abdominal aorta, and if it were to rupture, would result in a potentially fatal outcome. Conversely, pancreatitis is an inflammation of the pancreas that can range in severity from mild to severe. The intersection of these conditions gives rise to distinctive challenges in terms of diagnosis and management. The co-occurrence of AAA and pancreatitis is an uncommon occurrence, but one that can present significant clinical challenges. A review of the literature reveals a small number of documented cases where these conditions coexist, often complicating the clinical picture and management strategies (10-12). The presence of pancreatitis in patients with AAA has been demonstrated to increase the risk of complications and mortality. For instance, acute pancreatitis (AP) has been associated with increased mortality in cases of ruptured AAA. As is evident in the present case, the presence of AAA has been demonstrated to exert pressure on the pancreatic duct, thereby inducing the onset of pancreatitis. This mechanical compression has been shown to result in recurrent episodes of acute pancreatitis, as documented in case reports (11,13). The inflammatory processes involved in pancreatitis have the potential to exacerbate the weakening of the aortic wall, thereby leading to aneurysm rupture. Elevated pancreatic enzymes, such as elastase, have been shown to degrade the aortic wall thereby contributing to aneurysm formation or rupture (12,14). The diagnosis of pancreatitis in the context of AAA can be challenging due to the presence of overlapping symptoms and the potential for atypical presentations. Consequently, the employment of advanced imaging techniques in conjunction with a meticulous clinical evaluation is imperative for an accurate diagnosis. Further research is required to enhance our understanding of the pathophysiological interactions between aneurysm of the abdominal aorta (AAA) and pancreatitis. This includes the exploration of the role of inflammatory mediators and the impact of surgical interventions on these conditions.

Conclusion

Acute pancreatitis is a multifaceted condition involving numerous aetiological factors. Gallstones and alcohol

use are the most common aetiological factors, but other important contributing and known factors include hypertriglyceridemia, ERCP, genetic predispositions and drugs. It is likely that vascular factors may also play a role among these causes and a comprehensive understanding of the underlying pathophysiological mechanisms is imperative for the development of improved management strategies and optimisation of patient outcomes.

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