Case Report

Acute Type B Aortic Dissection Associated with Acute Pancreatitis, Pancreatic Pseudocysts, and Acalculous Cholecystitis

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Acute aortic dissection can result in fatal conditions when associated with organ malperfusion. A rare complication of aortic dissection with organ malperfusion is ischemic pancreatitis with cholecystitis. Here, we present the case of acute type B aortic dissection complicated by concurrent acute ischemic pancreatitis and acalculous cholecystitis. Prompt diagnosis and specific multidisciplinary treatment are crucial to improving patient outcomes in cases of visceral ischemia.

Keywords: acute type B aortic dissection, acute pancreatitis, acute acalculous cholecystitis

Introduction

Acute aortic dissection is frequently associated with organ malperfusion, which can be fatal.1) Various visceral complications develop depending on the affected branches of the aorta, but both acute pancreatitis and acute acalculous cholecystitis (AAC) following acute aortic dissection are uncommon.2,3) Herein, we report the rare case of acute type B aortic dissection (ABAD) complicated by acute pancreatitis with pancreatic pseudocysts, which improved after spontaneous formation of a cystogastric fistula, and AAC successfully treated with percutaneous drainage.

Case Report

A 71-year-old man with a history of hypertension and dyslipidemia presented with sudden onset of epigastric and back pain. A contrast-enhanced computed tomography (CT) scan revealed an ABAD, which extended from the proximal descending aorta to the level of the renal arteries. The celiac trunk originated from the false lumen and the superior mesenteric artery (SMA) from both the true and false lumens. The celiac trunk, the distal part of the splenic artery, and the proximal part of the left gastric artery were occluded, and the SMA was partially occluded after the origin of the first jejunal and inferior pancreaticoduodenal arteries (Figs. 1A–1C). No signs of bowel ischemia were apparent on the CT scan. Laboratory data were normal except for white blood cell count (14,600/µL), serum amylase (242 IU/L), CRP (0.95 mg/dL), and D-dimer (179.5 µg/mL). The patient was admitted to an intensive care unit, and antihypertensive treatment was initiated. Two days after admission, exploratory laparotomy was performed for suspected progressive bowel ischemia with aggravation of lactic acidosis and clinical symptoms. No intestinal ischemic changes were noted. On day 15 after admission, laboratory examination indicated worsening inflammation, and a CT scan revealed swelling and a heterogeneous contrast-absent area in the pancreatic parenchyma, leading to the diagnosis of acute pancreatitis. It was complicated by pancreatic pseudocysts, the largest of which measured 6.1 × 5.6 × 6.0 cm in size. Simultaneous AAC and splenic infarction were also observed. A portion of the gallbladder wall was contrast-poor, indicating necrosis (Figs. 1D and 1E). The splenic artery was totally occluded, whereas the SMA was recanalized due to the reduction of the false aortic lumen. Fluid resuscitation and antibiotics were administered for acute pancreatitis, and percutaneous transhepatic gallbladder drainage (PTGBD) was performed for AAC. Clinical symptoms and laboratory data promptly improved. A follow-up CT scan on day
26 after admission revealed that a fistula spontaneously developed between the stomach and the largest pancreatic pseudocyst. The remaining pseudocysts had completely disappeared at 6 months after the onset of ABAD (Fig. 2). The patient did not experience recurrent ischemic or aortic events during the 3-year follow-up period.

Discussion

To date, few cases with either acute pancreatitis or AAC following ABAD have been reported, and, to our knowledge, this is the first report of ABAD associated with simultaneous development of acute pancreatitis and AAC.2,3)

The pancreas is considered resistant to ischemia because of the abundance of feeding arteries. The pancreaticoduodenal arteries predominantly supply the pancreatic head, whereas the great caudal pancreatic arteries, originating from the splenic artery, supply the pancreatic body and tail, which is additionally fed by the transverse dorsal pancreatic arteries. Supplementary circulation from the left gastric and gastroepiploic arteries is also important. The pancreatic circulation supplied by these inflows is rarely damaged in cases of aortic dissection. In our patient, the celiac trunk, the proximal part of the left gastric artery, and the distal part of the splenic artery were occluded on the day of onset of ABAD, which presumably led to ischemia of the pancreas. Additionally, para-aortic inflammation after aortic dissection might have contributed to the development of pancreatitis.

Pancreatic pseudocysts are a complication of acute and chronic pancreatitis, accounting for up to 20%–40% of cases.4) Spontaneous fistulous formation of a pancreatic pseudocyst with the surrounding viscera is uncommon, with the reported incidence of <3%. Most fistulae are created with the colon or duodenum5); however, a cysto-gastric fistula is very rare. In this case, we consider that continuous compression of the stomach wall by a large pseudocyst, in addition to decreased blood flow in the stomach caused by the occlusion of the left gastric artery, contributed to the spontaneous fistula. Rupture of a pancreatic pseudocyst into the stomach is often complicated by abscess formation or bleeding, and uncomplicated spontaneous rupture is very rare.6)

Inflammation of the gallbladder in the absence of gallstones is also uncommon, accounting for 5% of acute cholecystitis.7) It occurs in 1.1% of cases after abdominal aortic aneurysm repair and in 0.08% of cases after open cardiac surgery.8) AAC following aortic dissection is uncommon (Table 1).2,9,10) We presume that occlusion of the celiac trunk and subsequently decreased blood flow in the cystic artery, in addition to prolonged fasting, resulted in the development of ischemic cholecystitis. Considering
the poor general health of the patient, we chose PTGBD rather than cholecystectomy; PTGBD successfully resulted in a rapid improvement of local inflammation.

Conclusion
Acute pancreatitis and AAC following ABAD are rare, difficult to diagnose, and can lead to fatal outcomes. Prompt diagnosis of visceral ischemia in cases with aortic dissection and a suitable multidisciplinary approach specific to patients’ general condition are important to improve prognosis.

Disclosure Statement
The authors declare no conflict of interest.

Additional Note
We obtained the patient’s consent for publication of this case report.

Author Contributions
Study conception: MN, AH
Data collection: MN, AH
Writing: MN, AH
Critical review and revision: all authors
Final approval of the article: all authors
Accountability for all aspects of the work: all authors

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