Acute Ischemic Pancreatitis Associated with Acute Type B Aortic Dissection: A Case Report

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A 47 year-old man, presenting with sudden back pain in the absence of abdominal discomfort, was diagnosed with acute type B aortic dissection which extended to the celiac and the splenic arteries. Antihypertensive treatment was initiated. However, he subsequently complained of upper abdominal pain with increased amylase levels. Computed tomography scan (CT) revealed new accumulation of peripancreatic fluid with no signs of further aortic or visceral dissection. A protease inhibitor was administered for mild acute pancreatitis. Follow-up CT demonstrated disappearance of thrombosed false lumen of the splenic artery and reduction of the effusion. The patient was discharged without any surgical interventions.

Keywords: acute type B aortic dissection, acute pancreatitis

INTRODUCTION

Hospital outcomes from effective antihypertensive therapies are generally acceptable in patients with uncomplicated acute type B aortic dissection (ABAD).\(^1\) In contrast, those with complicated ABAD, such as malperfusion syndrome, retrograde dissection and aortic rupture, are at a higher risk of mortality. In the International Registry of Acute Aortic Dissection (IRAD) database, malperfusion is recognized in 21% of patients with ABAD, mesenteric ischemia in 5.3%, renal failure in 13.5% and limb ischemia in 7.1% of patients. Acute pancreatitis is not described as an ischemic complication of acute aortic dissection in the IRAD.\(^2\)

Herein, we report our experience with a case of acute pancreatitis complicated with ABAD, which was probably caused by ischemic changes of the pancreas. We described the mechanism of acute pancreatitis associated with ABAD.

CASE REPORT

A 47 year-old man, with untreated hypertension, presented to a regional hospital with sudden onset of severe back pain. He had not consumed alcohol heavily just prior to the onset. Contrast-enhanced computed tomography (CT) demonstrated an acute type B aortic dissection, which involved the celiac trunk and the splenic artery (Fig. 1). The true lumen of the celiac trunk and the splenic artery became narrow because of the thrombosed false lumen. The superior mesenteric artery and the right renal artery arose from the true lumen, and the left renal artery was supplied from the false lumen. True aneurysm with intramural thrombus was not found. Laboratory data were normal except for slight elevation of alanine transaminase (56 IU/L) and D dimer (3.1 µg/mL). Conservative medical management using antihypertensive drugs was started under the judgment of preserving visceral perfusion because he had no abdominal discomfort. Twelve hours later he complained of upper abdominal pain with a slight increase of total blood amylase level (from 50 IU/L at admission to 90 IU/L twelve hours later). A follow-up CT scan revealed no remarkable changes of the aortic and celiac dissection but evident fluid accumula-
tion near the pancreas, which had not been observed at the onset of the dissection. Progressive ischemic change was suspected despite the unchanged appearance of the aortic dissection, and he was transferred to our hospital for further treatment.

Upon arrival in the emergency room, the patient was in a normal conscious state and presented with mild upper abdominal pain without any rebound tenderness, distension, or diminished bowel sounds. The blood pressure was 121/67 mmHg, regular pulse rate 80 beats per minute, and the body temperature 38.0°C. Laboratory examination showed elevation of total blood amylase (239 IU/L) and pancreatic amylase level (193 IU/L), and hepatobiliary tract enzyme was within normal limit. Contrast-enhanced CT at transfer revealed similar findings regarding the aortic and visceral dissection, which was observed from the celiac trunk to the middle part of the splenic artery with almost complete occlusion of the false lumen by the thrombus, resulting in the narrowing of the true lumen. There was a small amount of peripancreatic fluid accumulation with no areas of reduced or absent contrast enhancement, and edematous change of the pancreatic parenchyma (Grade III assessed by the CT severity index"). There was no sign of gallstones. We diagnosed this case as acute pancreatitis associated with aortic dissection, based on the symptom, elevated serous amylase level and CT images. The severity of acute pancreatitis was stage 0, mild acute pancreatitis based on the Japan severity scoring system.

We continued conservative medical therapy, including administration of a protease inhibitor, antibiotics, maximum fluid replacement, and bowel rest, in addition to treatment for controlling blood pressure.

These conservative managements decreased the level of pancreatic amylase to normal level after 2 days with steady alleviation of the abdominal discomfort. Follow-up CT scan showed disappearance of the thrombosed false lumen of the splenic artery and reduction of the peripancreatic effusion (Fig. 2). The patient was discharged without any surgical interventions.

**DISCUSSION**

We have described our experience with a rare clinical case of mild acute pancreatitis associated with ABAD.
We made the diagnosis based on the following findings: 1) the patient presented with upper abdominal pain, several hours after the onset of acute aortic dissection, 2) the concentration of serous pancreatic amylase was increased, 3) the CT scan revealed fluid accumulation around the pancreas with the progression of the aortic dissection into the splenic artery, suggestive of hypoperfusion of the pancreas, 4) the patient was not a heavy drinker and had no sign of gallstones.

Acute pancreatitis is a common disease with major etiological factors including alcohol consumption and cholelithiasis. Although ischemia is an infrequent pathogenesis of pancreatitis, the pancreas is an organ that is highly susceptible to ischemia and hypoxia. The ischemic injury with consecutive reperfusion, so-called ischemia/reperfusion (I/R) injury, is considered to be a potentially damaging factor in the initiation and progression of acute pancreatitis. I/R injury of the pancreas has been observed in several clinical situations, such as shock, cardiac or aortic surgery using cardiopulmonary bypass (CPB), and pancreatic transplantation. However, I/R injury of the pancreas, associated with acute aortic dissection, has not been fully recognized, and its precise incidence is unclear due to its rarity.

The incidence of acute pancreatitis after cardiac surgery using CPB is reported to be less than 1%. The pathogenesis of postcardiac surgical pancreatitis is associated with hypoperfusion of the whole pancreas caused by low cardiac output, prolonged bypass time, vasoconstrictor use, and hypothermia. Hypoperfusion evoked by aortic dissection could be a common factor in the pathogenesis of ischemic pancreatitis between pancreatitis related to CPB and dissection. In our case, we speculated that I/R injury-induced acute pancreatitis, related to the ABAD, was caused by the sudden and transient deterioration of the pancreatic circulation, as a result of the dissected splenic artery. The pancreatic head would be fully perfused through arterial arcades, such as the

**Fig. 2** Serial contrast-enhanced computed tomography (CT) images.

A: At onset, CT demonstrated aortic dissection that progressed into the celiac trunk (white arrow) and splenic artery (white arrow head). Peripancreatic effusion was not recognized. B: At the 12-hour follow-up, when this patient complained of abdominal pain and the amylase level was increased, the CT showed mild effusion around the pancreatic body (multiple white arrows). C: At the 9-day follow-up, CT revealed preservation of the blood supply to the celiac trunk (white arrow), which likely occurred via the collateral branches from the superior mesenteric artery. The dissection of the splenic artery disappeared (white arrow head) and the volume of the peripancreatic effusion decreased. D: At the 16-day follow-up, the peripancreatic effusion diminished considerably. P: pancreas
pancreaticoduodenal arteries from the superior mesenteric artery. In contrast, the blood supply to the pancreatic body and tail, which was perfused mainly via branches of the splenic artery (great pancreatic artery and caudate pancreatic artery), would be insufficient because of the dissected splenic artery. However, complete ischemia is unlikely to occur because the pancreatic body and tail have other arterial supplies from the dorsal pancreatic artery and transverse pancreatic artery, originating from celiac or superior mesenteric arteries. The pancreatic tail also has significant collateral circulation through the splenic hilum perfused from the perigastric arteries including the left gastric artery, the short gastric artery, and the left gastroepiploic artery. Therefore, pancreatitis may not deteriorate significantly and remains mild. Umeda et al. reported a case of severe necrotic pancreatitis caused by ABAD. In this report, they described that hypoperfusion was not the only factor in the development of severe pancreatitis because anatomically there is significant collateral circulation to the pancreas. They, therefore, speculated that the cholesterol embolism from the dissected aorta, in addition to hypoperfusion of the celiac trunk, caused the pancreatic necrosis. In our case, the thoracic true aneurysm with intramural hematoma was not detected on CT. In consideration of this finding, it is unlikely that the aortic dissection embolized cholesterol crystals into the arterial circulation.

The management of I/R-induced pancreatitis is similar to that of the pancreatitis of other etiologies. In our case, we selected medical management because the severity of pancreatitis was mild (stage 0) and the perfusion of the pancreas was maintained via the celiac, splenic, and the superior mesenteric artery, as confirmed by the contrast-enhanced CT scan. Surgical management is the favorable treatment for those patients who have recurrent symptoms or persistent elevation of pancreatic enzymes despite appropriate medical management. Surgical treatments are mainly divided into two strategies; revascularization for visceral ischemia and/or necrosectomy or drainage of the damaged pancreas. The strategy of surgical or endovascular revascularization for dissection-related acute pancreatitis has not been well established. These interventional treatments may be reserved for patients with associated complications such as arterial rupture, aneurysm formation, or broad multiorgan ischemia including liver, stomach, and spleen in addition to pancreas. Conservative measures were effective for our patient suffering from mild pancreatitis, and surgical interventions were unnecessary.

**CONCLUSION**

We should be aware of ischemic pancreatitis complicated by acute aortic dissection if the dissection involves the celiac trunk. Conservative therapies would be recommended first for this kind of pancreatitis.

**DISCLOSURE STATEMENT**

The author declares no conflict of interest.

**REFERENCES**

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