

# Multiple Ascending Aortic Mural Thrombi and Acute Necrotizing Mediastinitis Secondary to Acute Pancreatitis

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The formation of aortic thrombi is an extremely rare complication of acute pancreatitis. Here we report a case of acute pancreatitis complicated by a paraesophageal pseudocyst, necrotizing mediastinitis, and the formation of multiple thrombi in the ascending aorta. The patient was successfully treated by surgical therapy, which included extensive debridement of the mediastinum and removal of the aortic thrombi under cardiopulmonary bypass. Although esophageal resection was not carried out concomitantly, the lesions were resolved and the patient remained free of complications over 2 years of follow-up care.

**Key words:** 1. Pancreatitis, Acute Necrotizing  
2. Aorta  
3. Thrombosis  
4. Mediastinitis  
5. Esophageal Diseases

## Case report

A 66-year-old male patient with a history of hypertension and heavy alcoholism was referred to Asan Medical Center for further evaluation of an abdominal distension that had developed 15 days prior. On admission, the patient described mild abdominal pain and anorexia. The patient had experienced 6 kg of weight loss during the previous 2 months. A physical examination revealed fever (38.2°C) and abdominal tenderness. Laboratory investigations revealed an elevated white cell count of  $10.6 \times 10^9/L$  and a C-reactive protein level of 6.69 mg/dL (the reference value is  $<0.6$  mg/dL). Other findings were unremarkable, including serum amylase/lipase levels and the results of liver function tests. Esophagogastroduo-

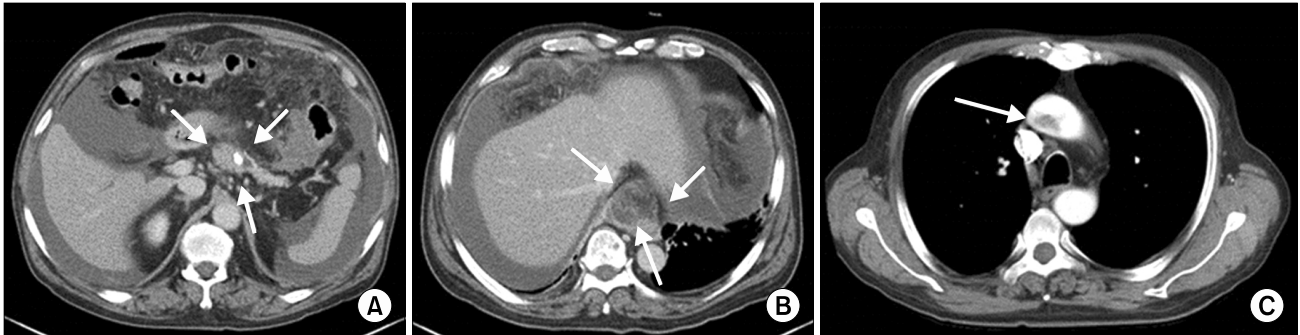
denoscopy (EGD), colonoscopy, and enhanced abdominopelvic computed tomography (CT) were performed under suspicion of a cancer of gastrointestinal origin. Abdominopelvic CT revealed chronic calculous cholecystitis, pancreatic duct stone, and associated peripancreatic fat necrosis accompanied by peri-esophageal fluid collection and a moderate amount of ascites (Fig. 1A, B), which were indicative of acute necrotizing pancreatitis. Subsequent laboratory tests showed elevated pancreatic enzymes (serum levels of amylase and lipase of 773 U/L and 589 U/L, respectively). Additional enhanced chest CT showed an incidental tubular low attenuated lesion within the lumen of the ascending aorta (Fig. 1C). Ascitic fluid examinations showed exudative fluid that was negative for malignant cells. Under the impression of

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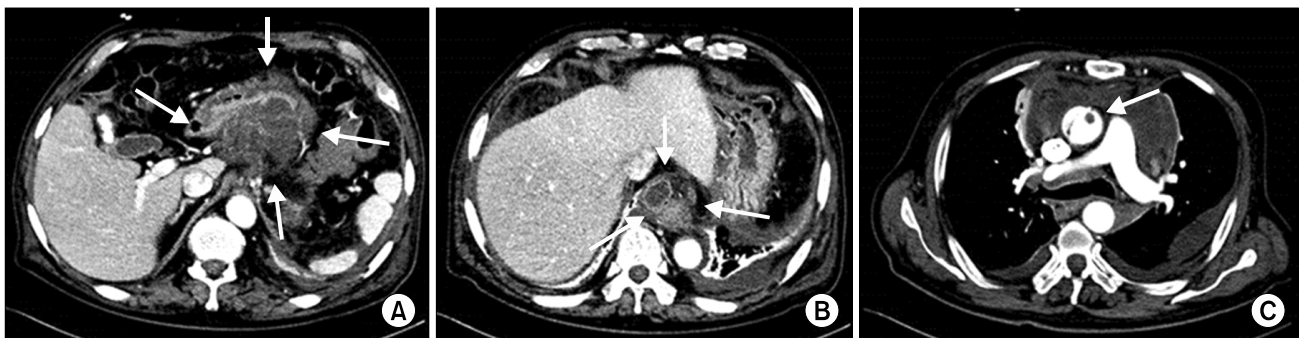
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**Fig. 1.** Axial view of abdominal and chest contrast-enhanced computed tomography on admission. (A) Pancreatic duct stone and peripancreatic fat necrosis was found (arrows). (B) Periesophageal fluid collection and a moderate amount of perigastric ascites can be seen (arrows). (C) A tubular low attenuated portion was found in the ascending aorta (arrows).



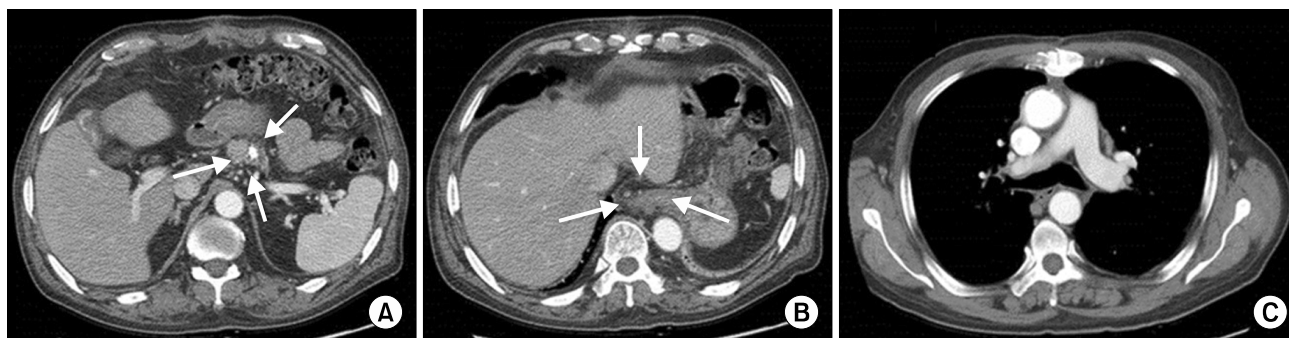
**Fig. 2.** Axial view of aortic contrast-enhanced computed tomography on day 16 of hospitalization. (A) An increased extent of peripancreatic fat necrosis was found (arrows). (B) Marked wall thickening and septation of the distal esophagus was seen (arrows). (C) Multiple floating masses in the ascending aorta (arrows) and acute necrotizing mediastinitis were found.

acute necrotizing pancreatitis complicated by exudative ascites resulting in periesophageal fluid collection, the patient remained in a fasting state, and received intravenous (IV) fluids and broad-spectrum antibacterial therapy.

Due to persistent fever and elevating C-reactive proteins despite these treatments, the patient underwent follow-up abdominal CT 2 weeks later, which revealed a newly developed left kidney infarction, pleural effusion, and pericardial effusion. In addition, marked thickening of the distal esophageal wall and an increased extent of loculated paraesophageal fluid were also found on the CT imaging (Fig. 2). A subsequent chest CT revealed multiple floating masses in the ascending aorta and mediastinal fat necrosis (Fig. 2). The aortic mass was confirmed to be highly mobile on transesophageal echocardiography (TEE). Therefore, we proceeded with urgent surgery, which included mediastinal debridement and removal of aortic throm-

bi under cardiopulmonary bypass. In the operating room, we found severe necrotizing mediastinitis with multifocal abscess formation and 2 of the highly mobile thrombi attached loosely to the aortic intima in the ascending aorta. The total pump and aortic clamping times were 52 minutes and 23 minutes, respectively. After mediastinal debridement and removal of the mobile thrombi, the inflammatory anterior pericardium was further resected, which was followed by massive irrigation (>10 L) of the whole surgical field. Additionally, we found the inflamed distal esophagus to be suggestive of an infected pseudocyst and planned to perform distal esophageal resection; however, the patient's family declined further esophageal procedures in consideration of the additional surgical risks.

Postoperatively, vital signs were stable and the patient fasted for 2 weeks until the absence of an esophageal fistula or perforation on EGD was confirmed.



**Fig. 3.** Axial view of aortic contrast-enhanced computed tomography one year after the operation. A marked decrease of (A) peripancreatic fat necrosis and (B) the distal esophageal pseudocyst were found (arrows). (C) Aortic mural thrombi were not seen in the ascending aorta (arrows).

The patient was given IV antibacterial therapy for 4 weeks. In addition, the patient underwent anticoagulation with IV heparin, which was switched to oral warfarin to prevent potential thromboembolism. The patient was discharged without complication on postoperative day 29. Postoperative echocardiography demonstrated normal ventricular function and no remnant aortic thrombi. The patient was able to ingest full meals, and a follow-up CT confirmed significant improvement of the lesions in the distal esophagus, which had been thickened diffusely with fat necrosis (Fig. 3). Two years after surgery, the patient was doing well clinically, without any complications.

## Discussion

This was an extremely rare case of a patient with multiple aortic mural thrombi and necrotizing mediastinitis caused by acute pancreatitis. Although most cases of acute pancreatitis are self-limited, one-fourth of patients have been reported to develop vascular complications, such as hemorrhage into a pseudocyst, erosion of the peripancreatic arteries, splanchnic venous thrombosis, and formation of pseudoaneurysms [1]. Arterial thrombus formation, however, is very uncommon, and aortic involvement is extremely rare. In a review of the literature, we were only able to find 3 cases of aortic mural thrombi formation secondary to acute pancreatitis, all of which involved the retropancreatic aorta [2,3]. To our knowledge, this is the first report in the literature describing thrombi in the ascending aorta. Initially, we suspected a cancer of gastrointestinal origin as the etiology of ascites. After performing abdominal CT, acute

necrotizing pancreatitis with paraesophageal fluid collection was diagnosed. We initially thought that the intramural aortic mass on the chest CT was a pseudolesion due to motion artifacts that turned out to be multiple floating thrombi in the ascending aorta according to subsequent CT evaluations and TEE.

Primary aortic thrombosis is defined as the development of thrombosis without underlying atheromatous lesions in the aorta [2]. The etiologies of primary aortic thrombosis are diverse, ranging from chemotherapy to treat cancer to heparin-induced thrombocytopenia and inflammatory bowel disease [4]. In addition, primary aortic thrombosis has been reported to occur in extremely rare cases of acute pancreatitis. Although the pathophysiology of acute pancreatitis leading to aortic thrombosis is not clear, several hypotheses have been suggested. First, acute severe pancreatitis emits proteolytic enzymes that injure the vascular wall directly, which may result in its acting as a nidus for intraluminal thrombus formation [4,5]. Second, the activation of thrombogenic factors from pancreatitis, such as fibrinogens and platelets, can also be triggered by the release of trypsin into the blood stream [5]. Lastly, acute pancreatitis possibly can lead to hypovolemia, which can increase the risk of vascular stasis and vasospasm [5].

After the second set of imaging evaluations in the present case, we assumed that the acute pancreatitis had progressed and was complicated by a paraesophageal pseudocyst, which subsequently developed into phlegmonous esophagitis and resultant acute mediastinitis. These inflammatory processes in the mediastinum might have been facilitated by the thrombogenic environment in the proximal aorta.

In conclusion, we experienced a rare case of acute pancreatitis resulting in necrotizing mediastinitis, paraesophageal pseudocyst, and multiple aortic thrombi. It was successfully treated by medical therapies combined with an urgent operation in which mediastinal debridement and complete removal of the aortic thrombi were performed.

### Conflict of interest

No potential conflict of interest relevant to this article was reported.

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