Images in Aortic Disease

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Esophago-Pleural Fistula Caused by Compression Necrosis In a Patient With Acute Type B Aortic Dissection

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Abstract

Esophago-pleural fistula associated with thoracic aortic aneurysm is a rare and lethal complication. We report the case of a 62-year-old male who suffered from esophago-pleural fistula 56 days after thoracoabdominal aortic surgery. Contrasted CT showed that the fistula occurred at the level of the esophagus compressed by rapid dilatation of thoracic aorta and endoscopy revealed no ischemic signs on esophageal mucosa, demonstrating that the cause of esophago-pleural fistula was compression necrosis due to rapid dilatation of the thoracoabdominal aortic aneurysm.

Key Words

Esophago-pleural fistula · Acute aortic dissection · Complication

Introduction

A 62-year-old male with a history of hypertension presented to the hospital with sudden onset of back pain and paraplegia. Contrasted computed tomography (CT) demonstrated Stanford type B acute aortic dissection with triple barrel of the thoracoabdominal aorta, in which the maximal diameter was 60 mm (Fig. 1). Open repair was considered for prevention of rupture, but his respiratory condition was severely poor in addition to complete paraplegia; hence, conservative treatment was selected. CT at 10 days after the onset of dissection revealed rapid dilatation of the thoracoabdominal aortic aneurysm, in which the maximal diameter reached 105 mm with compression of the esophagus (Fig. 2). Graft replacement of the thoracoabdominal aortic aneurysm was emergently performed. During the operation, proximal anastomosis was made just below the left subclavian artery with-

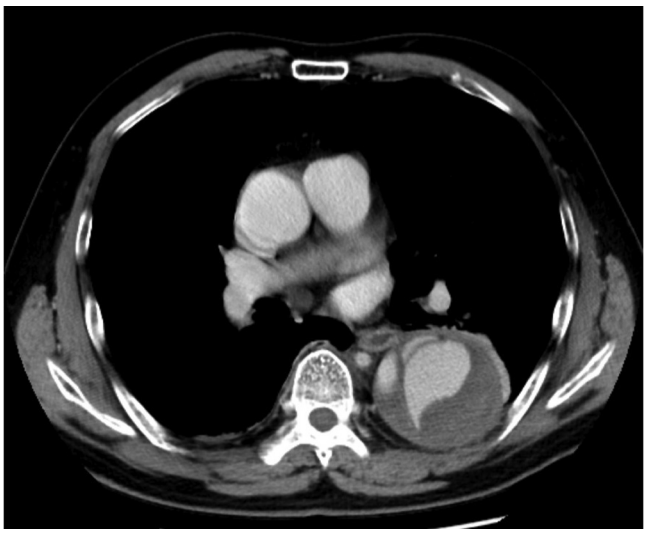

Figure 1. Contrasted computed tomography (CT) on admission demonstrated Stanford type B acute aortic dissection with 60 mm maximal diameter.

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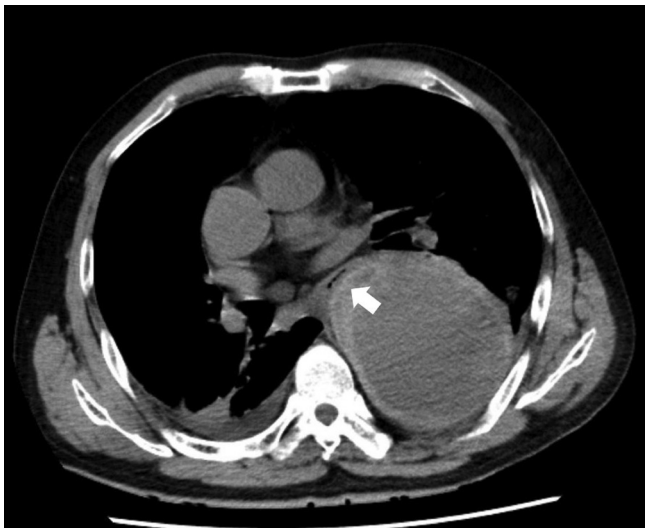

Figure 2. CT at 10 days after the onset of dissection demonstrated compression of the esophagus by the aortic aneurysm with 105 mm diameter (white arrow).

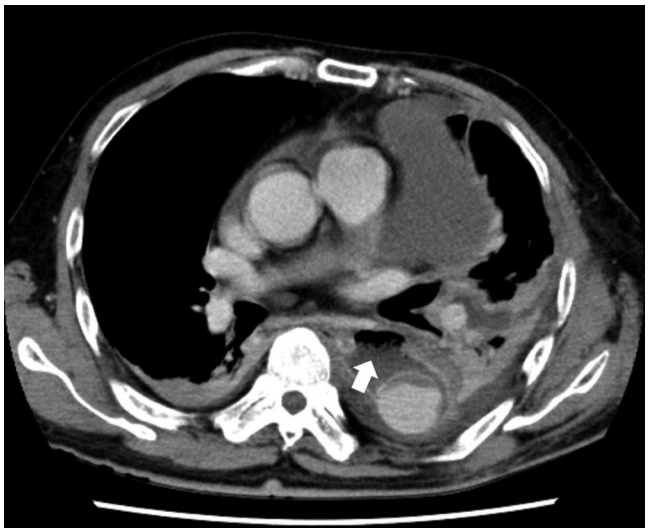

Figure 3. Postoperative contrasted CT at 56 days revealed pleural abscess around the vascular prosthesis, and perforation of the esophagus was suspected (white arrow).

out aortic clamping under deep hypothermic circulatory arrest. Distal anastomosis was made just above the renal arteries in the retroperitoneal space. Reconstruction of the intercostal arteries was not done because of preoperative paraplegia. Although left recurrent laryngeal nerve paralysis occurred as a complication, the operation was successful. The patient underwent rehabilitation up until discharge while on nasal tube feeding. At 39 days postoperatively, spiking fever occurred a few times per day. White blood cell count and C-reactive protein level were $7500 \times 10^3/\mu$L and 9.97 mg/dL, respectively. *Pseudomonas aeruginosa* was cultured from blood, but there was no obvious focus of infection identified on CT. Antibiotic treatment was initiated, and spiking fever gradually abated. Antibiotic treatment was continued for prophylaxis of graft infection. At 56 days postoperatively, contrasted CT as part of periodic inspection revealed pleural abscess around the vascular prosthesis, and perforation of the esophagus was suspected (Fig. 3). Endoscopy demonstrated the vascular prosthesis through a large esophago-pleural fistula (Fig. 4). The patient underwent emergent video-assisted thoracoscopic esophagectomy and thoracic drainage. Cervical salivary fistula was then established by use of the oral end of the esophagus, and an enteral feeding tube was placed in the jejunum. After the operation, intrathoracic irrigation from the thoracic tube was performed and antibiotic treatment

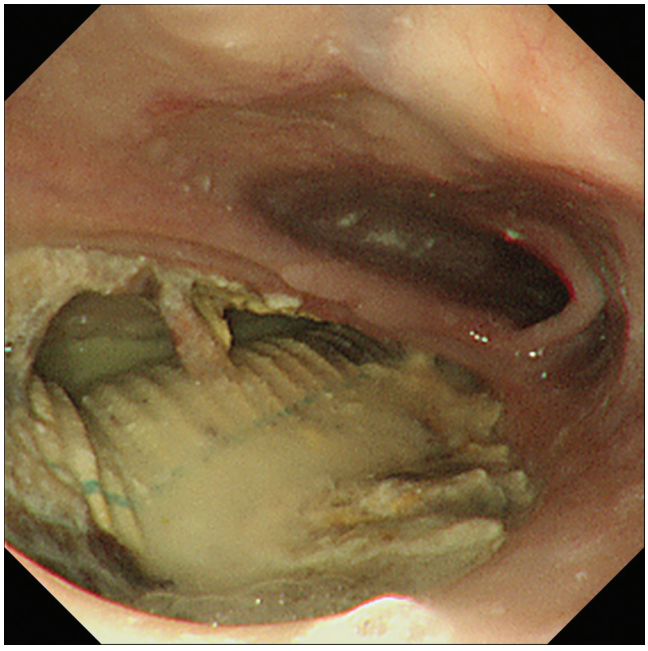

Figure 4. Endoscopy demonstrated the large esophago-pleural fistula.

was maintained. However, *Pseudomonas aeruginosa* was again detected in the culture of thoracic drainage. We recommended performing a graft replacement of the infected vascular prosthesis with omental plombage, but his family did not consent. He suffered from sepsis due to infection of the vascular prosthesis and died at 4 months after the surgery.

This case is our experience of a large esophago-pleural fistula in a patient with acute Type B aortic dissection. The causes of the esophago-pleural fistula were not believed to be due to surgical trauma during the operation or mechanical stimulation of the vascular prosthesis. Surgical procedures which could induce damage to the esophagus, such as resection of the aortic wall, aortic clamping, and reconstruction of the intercostal arteries, were not performed, and the vascular prosthesis was not in contact with the fistula as demonstrated by postoperative CT. Esophageal ischemia due to malperfusion may have influenced the perforation of the esophagus. However, endoscopy did not show necrotic or ulcerated esophageal mucosa around the fistula. Therefore, it is considered that the cause of the esophago-pleural fistula was the compression necrosis due to rapid dilatation of the thoracoabdominal aortic aneurysm. A small esophago-pleural fistula occurred on the 39th postoperative day when spiking fever appeared. However, it was discovered only after the fistula became large because infection was controlled initially by nasal tube feeding and appropriate antibiotic treatment.

Comment on this Article or Ask a Question

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EDITOR'S QUESTIONS

1. Did you consider intervening initially with fenestration or endograft to try to reverse the paraplegia?

No, I didn't. Actually, when he was admitted to our hospital, 8 hours had passed since the paraplegia had occurred. Then, cerebrospinal fluid drainage was performed for 72 hours, but the paraplegia was not recovered.

2. You intervened at ten days to replace the aorta, so the native aorta was then "depressurized". How, then, could the aortic wall produce pressure to result in esophageal necrosis?

There are some possibilities that could cause perforation of the esophagus such as rupture of the aneurysm to the esophagus, surgical trauma during the operation, esophageal necrosis due to malperfusion, or compression necrosis due to rapid dilatation of the aneurysm. During the operation, there were no signs of aneurysmal rupture. On the other hand, any surgical procedure which could induce damage to the esophagus, such as resection of the aortic wall, aortic clamping, and reconstruction of the intercostal arteries, was not performed. Therefore, it is not considered that the cause of esophageal perforation was the rupture to the esophagus from the surgical trauma. It is possible that esophageal ischemia due to malperfusion may have influenced the perforation of the esophagus because the intrathoracic esophagus is mainly supplied by the intercostal arteries which were occluded by the thrombosed false lumen of aortic dissection. However, endoscopy did not show necrotic or ulcerated esophageal mucosa around the fistula. Therefore, it is considered that the cause of esophageal perforation was the compression necrosis due to rapid dilatation of the thoracoabdominal aortic aneurysm.