Case Report

Tracheo-Bronchial Obstruction and Esophageal Perforation after TEVAR for Thoracic Aortic Rupture

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A 67-year-old man was referred to our hospital for an ascending aortic aneurysm, thoracoabdominal aortic aneurysm and aortic regurgitation. Graft repair of the thoracic aortic arch and aortic valve replacement was given priority and completed, however he developed descending aortic rupture before the second scheduled surgery, and endovascular stent grafting was performed. He subsequently developed tracheobronchial obstruction and esophageal perforation. The patient underwent urgent esophagectomy and enterostomy with continuity later reestablished. However, he died of sepsis 5 months after surgery. Despite the less invasive nature of endovascular treatment, esophageal perforation can nevertheless occur and postoperative vigilance is well warranted.

Keywords: aortoesophageal fistula, tracheobronchial obstruction, thoracic endovascular aortic repair

Introduction

Tracheobronchial obstruction and esophageal perforation secondary to thoracic aortic rupture is rare.1–3 Endovascular treatment is generally considered to be a safe and effective method of repairing thoracic aortic rupture in high surgical risk patients. The incidence of aorto-esophageal fistula (AEF) following open surgery versus endovascular stent graft implantation is reported to be nearly identical,11 but emergent thoracic endovascular aortic repair (TEVAR) is associated with an increased risk.1,3 Despite improvements in endovascular treatment, esophageal perforation remains a fatal complication.1–6 Therefore, early detection and effective therapeutic management are critical.

Case Report

A 67-year-old man previously diagnosed with chronic obstructive pulmonary disease (COPD) presented with dyspnea and general fatigue; laboratory findings were unremarkable. Mediastinal enlargement consistent with aneurysm was visible on chest X-ray, and computed tomography (CT) revealed aneurysms of the ascending aorta and the thoracoabdominal aorta. The patient was then referred to our hospital. As measured using CT the ascending aorta was 7.2 cm in diameter with a thoracoabdominal aortic aneurysm of 7.6 cm (Figs. 1a and 1b). Echocardiography revealed severe aortic regurgitation. Treatment was scheduled to occur in two separate operative procedures, the first being graft repair of the thoracic aortic arch and aortic valve replacement (Fig. 1c). This was accomplished and his postoperative course was uneventful.

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One and half months after the first operation the patient was readmitted for surgical treatment of the thoracoabdominal aortic aneurysm, but he complained of acute chest pain on the day prior to surgery. His condition subsequently deteriorated, and emergent CT revealed a ruptured descending aortic aneurysm (Fig. 2a). Stent grafting was immediately performed, starting at a point just distal to the neck branches of the previously implanted prosthetic vascular graft and ending at the superior mesenteric artery (TAG 34 × 200, 37 × 200, and 40 × 200, WL Gore & Associates, Flagstaff, Arizona; TX2 42 × 42 extension, COOK, Bloomington, Illinois, USA).

The 170-min procedure was uneventful with no leakage or adverse effects observed; the patient was subsequently transferred to the intensive care unit. Post-operative monitoring indicated substantially elevated endotracheal pressure despite efforts to improve ventilation. A bronchoscopy showed narrowing at the lower part of the trachea due to outer compression. CT was again performed and demonstrated no endoleak, but rather a mediastinal hematoma compressing and obstructing the trachea and left bronchus (Figs. 2b and 2c). The right main bronchus was intubated, and he was managed on mechanical ventilation until weaned from the respirator on the tenth postoperative day.

Twenty days after the second surgery, the patient began having episodes of hematemesis. CT indicated air around the hematoma, suggesting esophageal perforation (Fig. 3a). To confirm the diagnosis, esophagography was performed and revealed contrast medium leakage from the mid-esophagus into the mediastinum (Fig. 3b). Total parental nutrition was immediately initiated, and the patient was maintained on antibiotic coverage. This conservative therapy was chosen as his general condition was deemed unsuitable for another major surgery. However, a low-grade fever persisted despite antibiotic administration, and blood tests revealed an elevated leukocyte count and a highly elevated C-reactive protein level, necessitating another urgent operative intervention on the 29th postoperative day.

This surgical examination revealed severe adhesion surrounding the esophagus, to an extent that nearly precluded esophagectomy. The mid to distal esophagus was found to be necrotic and the fistula was identified; distinguishing the aortic aneurysm wall was extremely difficult due to the severity of the adhesion.

Fig. 1 (a, b) CT scan revealed aneurysms of the ascending aorta and the thoracoabdominal aorta. The ascending aorta was 7.2 cm in diameter and the thoracoabdominal aortic aneurysm was 7.6 cm in diameter. Postoperative CT demonstrated successful graft replacement in the thoracic arch and successful aortic valve replacement (c). CT: computed tomography.
Debridement was performed through a right thoracotomy, along with lateral cervical esophagostomy and enterostomy, however we did not attempt further aortic surgery because of high rate of mortality in this patient. Three months after the esophagostomy and enterostomy continuity was restored with an anterosternally-guided, pedicled isoperistaltic transverse colonic conduit; hematogenous disorder precluded the use of a gastric tube. Although the patient tolerated these procedures well, he died of sepsis caused by mediastinitis on the 5th month after surgery.

**Discussion**

AEF and its catastrophic consequences are a well-recognized complication of thoracic aortic surgery.1-6)
Due to the various causations of AEF it is difficult to exclude the possibility of damage of descending aortic aneurysm, or of iatrogenic injury such as intraoperative transesophageal echocardiography, in the progression of this condition. Although the exact mechanism of secondary AEF after stent grafting remains unknown, hypotheses include: (1) direct erosion of the stent graft into the esophagus, (2) pressure necrosis caused by the self-expanding endoprosthesis, (3) ischemic esophageal necrosis due to disruption of the arteries that feed the esophagus, (4) infection of the stent-graft prosthesis, (5) pseudoaneurysm development, and (6) endoleakage into the residual aneurysmal sac. In this case, the blood supply to the esophagus was already disrupted because of the previous graft repair of the thoracic aortic arch; relative ischemia of the esophagus may have occurred secondary to endovascular stent-grafting. Bleeding into the mediastinal space instead of the thoracic cavity is also a known cause of thoracic compartment syndrome (TCS). From the information gleaned in this case we hypothesize that extremely elevated mediastinal pressure was compressing the trachea and esophagus, worsening the esophageal ischemia and thus making the esophagus more prone to further necrosis.

AEF has been reported to occur in 1.7%–5% of TEVAR patients; there are no fundamental differences in the frequency of occurrence following open repair surgery versus endovascular methods of management. However, emergent TEVAR has been associated with an increased risk of AEF development and is often performed in cases of aortic rupture; as endovascular stenting does not remove the hematoma or the thrombosis, it is likely to result in TCS. Although TEVAR has been developed as a safe and less-invasive treatment option for patients at high risk for conventional surgical repair, it may not have any advantages with respect to the risk of AEF. Caution and diligent monitoring after surgery, especially in emergent procedures, is therefore suggested.

Abdominal compartment syndrome (ACS) is well-documented in many cases of multiple organ failure and a leading cause of postoperative death. Reports have described ACS incidence of 20 percent in endovascular aortic repair (EVER) for ruptured abdominal aortic aneurysm. This relatively high incidence of ACS after EVER compared to open surgery may be once again attributable to the inability to evacuate retroperitoneal hematomas in endovascular repair. Because the mortality rate is significantly higher in patients who develop ACS, it is important to recognize this complication; if it develops early decompression is necessary. In this case, we did not evacuate the blood clots from the mediastinum because the patient’s hemodynamics were stable, and the airway pressure had gradually improved. Although removing the hematoma may have caused stent graft instability, it might have also reduced the length of time that respirator support was required and the extent of the esophageal lesion caused by compartment syndrome.

Radical treatment for AEF involves exclusion of the infected stent graft and esophageal fistula repair or esophageal resection. However, such radical surgery is not applicable to most AEF patients owing to their poor general condition and/or comorbidities. When such a radical procedure seems too risky, conservative treatment is considered. Although several reports have described the successful conservative treatment of AEF, endovascular stenting in a potentially contaminated or infected lesion is still controversial. In this case, we initially selected conservative treatment because the patient’s general condition was poor and there was no evidence of sepsis, resorting to urgent surgery only after laboratory data showed increasing inflammatory reaction. In addition, it has been previously reported that when an ulcer is caused by ischemia, healing does not occur and the lesion may progress in size. Therefore, when ischemia is considered to be involved with an AEF we should not expect it to spontaneously heal, but rather consider surgical repair or removal of the esophageal lesion as a better treatment option than conservative treatment.

**Disclosure Statement**

None of the authors have anything to disclose with regard to commercial support or financial interests.

**References**

Esophageal Perforation after TEVAR


