Successful Management of Esophageal Necrosis After Endovascular Repair of Chronic Type B Aortic Dissection

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We report the case of a 65-year-old patient with esophageal necrosis that developed after thoracic endovascular aortic repair (TEVAR) of a previously stented, ruptured chronic type B aortic dissection. The cause of this complication may have been related to an infected mediastinal hematoma causing esophageal compression. Emergent esophagectomy was performed with success.


Esophageal necrosis is a rare but lethal complication after thoracic endovascular aortic repair (TEVAR). It can occur as a result of extrinsic compression from a mediastinal hematoma or can be due to overstretching of the esophageal arteries [1–3]. Only 3 cases of esophageal necrosis after TEVAR have been reported in the literature, all of which have been fatal [1–3]. We describe a case in which esophageal necrosis occurred as a complication after a TEVAR extension for a previously stented, ruptured chronic type B dissection that was successfully managed by esophagectomy.

A 65-year-old woman had undergone TEVAR at our institution in 2012 to treat a residual distal aortic dissection after successful arch replacement for an acute type A dissection. One year later, she presented to an outside institution with fever and paraplegia. An aortic graft infection was suspected. Magnetic resonance imaging was performed, and a diagnosis of anterior spinal artery syndrome was made by visualization of spinal cord ischemia at the C6 level. She improved with antibiotics and cerebrospinal fluid drainage until the abrupt onset of dysphagia. A computed tomography (CT) scan of the chest showed chronic type B aortic dissection with aneurysm of the false lumen and an associated large mediastinal hematoma compressing the esophagus and trachea (Fig 1). She was intubated and immediately transferred to our hospital for treatment of the acute aortic rupture. Successful TEVAR extension was performed across the reentry of the false lumen. A CT scan performed the next day showed no evidence of residual retrograde false lumen flow but did demonstrate progression of the esophagotracheal compression (Fig 2). A tracheotomy was performed. However, the patient worsened, and 2 weeks after the TEVAR extension, she became septic with Enterococcus faecium. On day 15, a chest CT scan showed air bubbles within the false lumen of the dissected aorta (Fig 3). Esophagoscopy revealed circumferential esophageal necrosis and perforation at the 24-cm to 29-cm level.

The patient was emergently taken to the operating room for exploratory thoracotomy through a right fifth intercostal incision. Dense inflammatory adhesions were dissected. The hematoma was removed, and the infected false lumen of the aneurysm was partially excised. The previously placed graft was left in place. After the hematoma was removed, a 7-cm-long defect of the esophagus was observed. A decision was made to perform esophagectomy in the standard fashion. After dissection of the remaining esophagus, the resulting cavity and the esophageal bed were debrided and irrigated. To cover the graft, we decided to perform gastric pull-up during the same surgery. Through laparotomy, the stomach was mobilized and fashioned into a tube in the usual manner. Gastric pull-up reconstruction was completed by end-to-side cervical esophagogastrectomy. Chest tubes were placed. Pathology examination confirmed transmural ischemic esophageal necrosis.

The operation was complicated by cardiac arrest requiring 15 minutes of resuscitation and defibrillation with subsequent stabilization. The postoperative course was uneventful, although paraplegia persisted. The patient was discharged to a weaning center on postoperative day 28. Currently, at 5 months after the esophagectomy, she is at a residential care facility and is eating a regular diet.

Comment

Esophageal necrosis is a rare complication after TEVAR. The 3 previously reported cases have all been fatal [1–3].

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Fig 1. (A) Native computed tomography scan of the chest demonstrating aneurysm of the false lumen and a mediastinal hematoma (arrows) with associated compression of the esophagus and trachea 1 year after thoracic endovascular aortic repair for a chronic type B aortic dissection. (B) Contrast-enhanced computed tomography scan demonstrating a persistent false lumen perfusion (*). (A = aorta; PE = pleural effusion; T = trachea.)

Fig 2. Computed tomography scan after distal thoracic endovascular aortic repair extension demonstrating reentry occlusion (*) with no evidence of filling of the false lumen (**) and progressive esophagotracheal compression from the mediastinal hematoma (white arrows). (A) Native computed tomography scan of the chest, axial plane. (B) Contrast-enhanced computed tomography scan of the chest, axial plane. (C) Contrast enhanced computed tomography scan of the chest, coronal plane. (A = aorta; Aa = abdominal aorta; K = kidney; L = liver; PE = pleural effusion; S = spleen; T = trachea.)

Fig 3. Computed tomography scan on day 15 after thoracic endovascular aortic repair extension demonstrating air bubbles surrounding the aortic stent graft (arrowheads), indicating an infection of the aortic aneurysm due to esophageal necrosis. Bilateral increasing pleural effusions (PEs) led to lung compression and atelectases. (A) Contrast-enhanced computed tomography scan of the chest, axial plane. (B) Contrast-enhanced computed tomography scan of the chest, coronal plane. (A = aorta; Aa = abdominal aorta; E = esophagus; K = kidney; L = liver; S = spleen; T = trachea.)

Here, we report the successful management of esophageal necrosis that occurred after a TEVAR extension for a previously stented, ruptured chronic type B dissection. Esophagectomy and removal of the infected mediastinal hematoma were performed. Removal of the esophagus led to improvement of the sepsis. The infected graft was left in place. Gastric pull-up was used to cover the graft. Omission of esophagectomy may have contributed to the fatal outcomes of the previously reported cases.
Esophageal necrosis after TEVAR may go unrecognized, or the diagnosis may be delayed because of its rare occurrence. Thus, a high level of suspicion for this complication should exist for patients with dysphagia and a previous thoracic aortic repair; however, the diagnosis is generally not considered until the onset of sepsis. In all reports (including the present), the patients were either in a septic state due to delays in diagnosis or had severe debilitation due to underlying premorbid conditions. In this setting, 2 of the 3 previously reported patients have been deemed unable to tolerate an extensive procedure such as esophagectomy [1, 2].

The cause of the observed necrosis may have been related to extrinsic esophageal compression from a mediastinal hematoma. Unlike open repair, TEVAR does not allow for removal of the associated hematoma. Whether early removal of the hematoma could have prevented this complication remains speculative, however, as esophageal necrosis has also been described after open repair [4]. In addition, this complication may also be related to aortic graft infection. In another case, graft resection and extraanatomic bypass was performed; however, the outcome was fatal [3]. It has been suggested that graft removal is not always required in the treatment of an infection surrounding an aortic graft [5]. The present case also suggests that it may be more appropriate to only perform esophagectomy for the treatment of esophageal necrosis. Our patient is still alive 5 months after the esophagectomy; however, long-term follow-up will be necessary to confirm this initial success. The permanent paraplegia of our patient likely resulted from hypoperfusion of the anterior spinal artery due to the aortic dissection as preoperative magnetic resonance imaging was already consistent with spinal cord ischemia. Moreover, additional segmental artery sacrifice during the TEVAR extension and intraoperative hypoperfusion during cardiac arrest may have contributed to irreversible spinal cord injury.

References