Aortoesophageal fistula after thoracic endovascular aortic repair and transthoracic embolization

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Endografts are more commonly being used to treat thoracic aortic aneurysms and other vascular lesions. Endoleaks are a potential complication of this treatment modality and can be associated with aneurysmal sac expansion and rupture. This case report presents a patient who developed a type IA endoleak after endograft repair of a descending thoracic aneurysm. The endoleak was successfully treated through computed tomographic-guided transthoracic embolization, although the patient experienced lower extremity paraparesis postprocedurally. The patient's endovascular repair was complicated by the development of an aortoesophageal fistula and endograft infection necessitating operative débridement and endograft explantation. (J Vasc Surg 2007;46:789-91.)

Thoracic endovascular aortic repair (TEVAR) is an alternative to open surgical repair for the treatment of many thoracic aortic lesions, but late complications of this form of therapy can occur.[1] Endoleaks are a potential complication and may indicate persistent systemic pressurization of the aneurysm sac. Aortic endograft infections are rarely reported and have been associated with secondary procedures after endovascular abdominal aortic aneurysm repair (EVAR).[2]

In this case report we present a patient with a type IA endoleak after TEVAR successfully treated by computed tomographic (CT)-guided embolization. An aortoesophageal fistula and endograft infection developed. This report highlights the technical feasibility of a transthoracic approach to endoleak management and raises a word of caution regarding endograft infection as a potential complication of TEVAR and subsequent secondary procedures.

CASE REPORT

A 52-year-old man with diabetes mellitus and hypertension presented with chest pain. Chest radiographic findings were suggestive of a thoracic aortic aneurysm. The patient was treated for bronchitis and referred for further evaluation. CT angiography (CTA) of the chest revealed a 6-cm descending thoracic aortic aneurysm without evidence of inflammation or rupture. The patient underwent TEVAR and received three Gore TAG endografts (W. L. Gore & Associates Inc, Flagstaff, Ariz), which were sized according to the manufacturer's instruction for use and deployed with partial overlap of the left subclavian artery (SCA).

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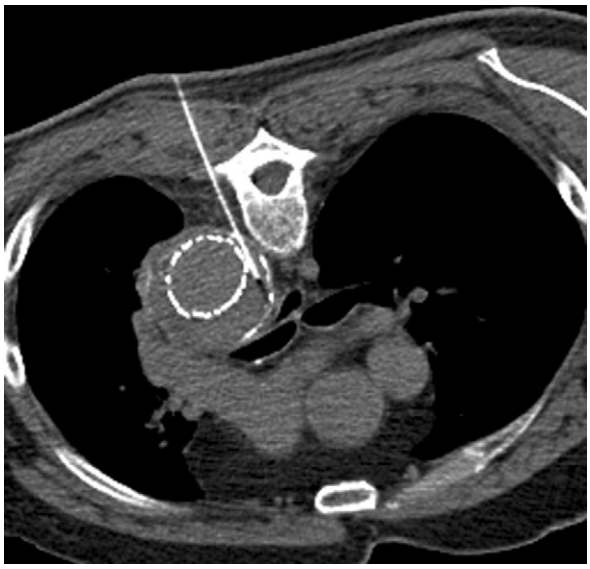

Fig 1. Computed tomographic-guided transthoracic access of the aneurysm sac.

At 1-month follow-up, the patient reported left lateral chest pain radiating to his back, and CTA revealed contrast outside the proximal portion of the endograft on delayed images suggestive of a type IA or type II endoleak. The proximal endograft appeared to be fully expanded with good circumferential apposition. Observational management was initially used, but the patient's back pain became progressively worse during the next 6 weeks.

A repeat CTA demonstrated persistence of the endoleak. Thoracic angiography confirmed a type IA endoleak that communicated with the aneurysm sac through an extremely narrow channel. Attempts to perform transarterial embolization were unsuccessful owing to the inability to sufficiently advance multiple microguidewires and microcatheters to obtain a stable position necessary for embolization.

Because of persistent pain and a 2-mm increase in aneurysm diameter, transthoracic embolization was performed 3 months

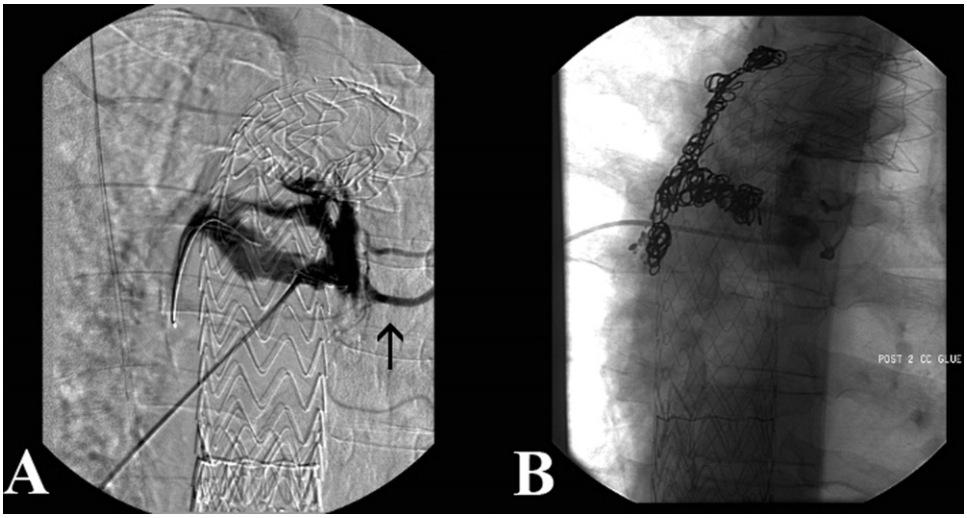

Fig 2. Angiography and embolization of a type IA endoleak. **A,** Direct injection of contrast into the aneurysm sac revealed a type IA endoleak and efferent drainage through intercostal vessels *(arrow)*. **B,** Successful coil embolization.

after TEVAR. The patient was placed in the prone position, and CT guidance was used to insert a 21-gauge needle into the aneurysm (Fig 1). The needle was exchanged for a 6F sheath from an AccuStick introducer system (Boston Scientific, Natick, Mass). Injection of contrast demonstrated communication at the proximal attachment site, confirming a type IA endoleak. Draining efferent intercostal arteries were also observed (Fig 2, *A*).

A 4F catheter was advanced through the 6F catheter into the aneurysm and manipulated into the proximal region of the endograft. Sixteen platinum Nester coils (Cook, Bloomington, Ind) were placed (Fig 2, *B*). Additional embolization was performed with a 1:3 mixture of Trufill glue (Cordis Neurovascular, Inc, Miami Lakes, Fla) and Ethiodol (Savage Laboratories, Melville, NY). A gel-foam slurry was used to embolize the needle tract as the catheters were removed.

After the procedure, the patient presented with progressive bilateral lower extremity paraparesis. Placement of a lumbar drain did not improve symptoms. Magnetic resonance imaging (MRI) of the thoracic and lumbar spine did not demonstrate evidence of spinal cord ischemia. Two weeks after his secondary procedure, the patient was transferred to an acute rehabilitation facility, and his lower extremity symptoms improved.

Six weeks after the endoleak repair, the patient presented with night sweats, fatigue, and chills. He denied chest pain, hematemesis, hemoptysis, and dysphagia. CTA demonstrated perigraft and aneurysmal sac air-fluid levels. A swallow study revealed contrast extending into the aneurysm sac around the endograft.

The patient urgently underwent replacement of his descending thoracic aorta with a Dacron interposition graft (Boston Scientific, Wayne, NJ) soaked in rifampin under cardiopulmonary bypass and hypothermic circulatory arrest. The aorta was found to be densely inflamed from the distal arch to the diaphragmatic hiatus, with gross purulence. In addition, a 4-mm defect in the middle esophagus communicated with the aneurysm. A large defect in the mid-descending aorta with exposed endograft was appreciated (Fig 3). The exposed endograft did not appear to be close enough to the esophagus to have caused mechanical erosion. The aneurysm remained excluded from the systemic circulation. The defect in the esophagus was closed primarily in two layers. Cultures of the aneurysm grew *Staphylococcus aureus, Streptococcus anginosus,* and multiple anaerobic bacteria.

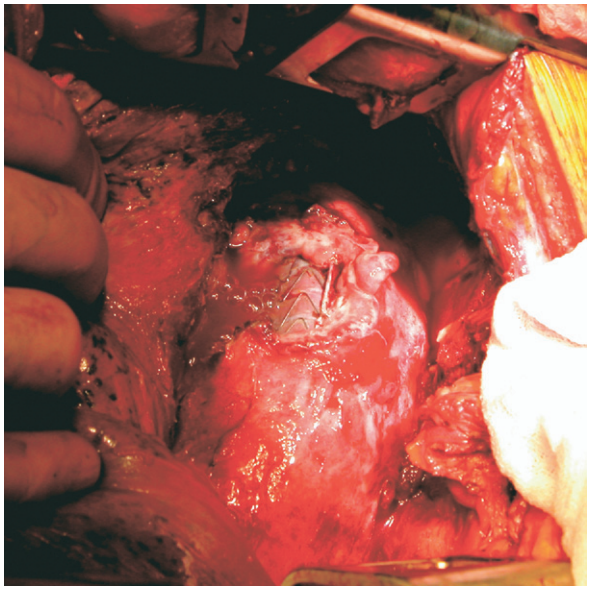

Fig 3. Intraoperative photograph taken during aortoesophageal repair and endograft explantation shows the esophagus is retracted anteriorly and a defect in the aortic wall is appreciated with exposed endograft.

On hospital day 25, the patient went into cardiopulmonary arrest after an acute massive hemorrhage into his left chest. Emergent exploration revealed hemorrhage from the proximal anastomosis. Despite repair of the defect, the patient died intraoperatively.

DISCUSSION

Successful TEVAR of descending thoracic aneurysms involves exclusion of the aneurysm. Endoleaks represent a persistent communication between the aneurysm and the systemic arterial circulation that may contribute to sac enlargement or rupture, or both.[3,4] Type I endoleaks can often be managed through balloon angioplasty or deployment of an additional endograft component when an adequate landing zone is present. In the case reported here, an additional endograft could not be placed because the patient's left common carotid artery was too close to the left SCA. Although an extra-anatomic bypass would have allowed for proximal endograft deployment, this would have necessitated a more invasive procedure. In addition, angioplasty was not felt to be beneficial in resolving a narrow channel endoleak owing to the full expansion and good apposition of the endograft.

CT-guided translumbar embolization of endoleaks after EVAR has been previously described using a variety of embolic and thrombotic agents.[5-8] Translumbar embolization promotes thrombosis to decreasing mean aneurysm sac pressure and eliminates blood outflow from efferent branch vessels. This form of treatment has been advocated instead of a transarterial approach for the treatment of type II endoleaks after EVAR.[7]

The incidence of thoracic endograft infections is unknown, but abdominal endograft infections are rare. A combined survey and review of the literature from 1991 to 2002 by Ducasee et al[2] reported an incidence of 0.43%. When infections occurred after embolization procedures, *S aureus* was identified as the infectious agent in 85% of the cases. The presence of *S aureus* in the intraoperative cultures is consistent with these observations and may suggest a cutaneous source of the infection.

From the history, it is unclear how the patient's endograft became infected; either as a consequence of foreign material deployed after TEVAR or secondary to contamination from the transthoracic procedure. The patient's chest and back pain may have suggested a subacute infectious aortitis, although CT imaging before and after the procedure was not suggestive of this. In addition, although the patient became symptomatic after the transthoracic procedure, his presentation was rather subacute at 6 weeks.

Imaging after the procedure did not reveal ischemic changes to the spinal cord, but the patient's paraparesis was most likely due to distal embolization of glue into efferent aortic branch vessels. We elected to use coils and a mixture of glue and contrast to more reliably embolize the aneurysm sac. A less dilute mixture might have prevented this complication from occurring.

CT-guided transthoracic embolization is a technically feasible treatment approach for endoleaks after TEVAR. Clinicians should also be aware of endograft infection as a potential complication of TEVAR and subsequent secondary procedures.

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