

Fungal Aortic Endograft Infection

Increasing application of EVAR and adjunctive procedures for endoleak repair may increase the incidence of this complication.

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Endoluminal stent infection involving an iliac artery was described as early as 1993 by Chalmers et al.¹ Given that there is a very low overall incidence of aortic endograft infection (AEI), conclusive studies of effective treatment regimens have not been reported. As physicians, we rely on experiences drawn from case reports, literature reviews, and one multicenter series by Ducasse et al to direct treatment in these circumstances.^{2,3} Surgical intervention has focused on removal of the infected endograft, debridement of the aorta, and revascularization of the pelvis and lower extremities. Control of sepsis and prevention of aortic rupture remain the principal goals of these procedures.⁴ In this article, we report a case of an AEI with *Candida albicans* after treatment for *Candida* esophagitis and repair of a type III endoleak.

CASE REPORT

A 76-year-old man with a medical history of coronary artery disease, carotid artery stenosis, and a 5.9-cm infrarenal aneurysm underwent endovascular aortic aneurysm repair (EVAR) in 2005 with a bifurcated modular endograft (Zenith, Cook Medical, Bloomington, IN). Two years later, the patient developed an asymptomatic type III endoleak without aneurysm sac enlargement, which was found on a surveillance computed tomography (CT) scan (Figure 1). Angiography confirmed the location

of the endoleak in an extremely tortuous left limb of the graft, and multiple covered stents were used to repair it with a satisfactory radiographic result (Figure 2). Five months later, the patient was helicoptered to our hospital from an outside institution with symptoms of a lower gastrointestinal bleed, fever (39° C), and leukocytosis (19,000 white blood cells per mm) that developed over several days. He had recently received treatment for esophageal candidiasis with intravenous fluconazole. Urine and blood cultures were negative for microorganisms. An abdominal CT scan revealed air surrounding the aortic endograft, which suggests an aortoenteric fistula (AEF) by stent graft erosion into the overlying duodenum (Figure 3). There was no stent migration, recurrent endoleak, or aneurysm sac enlargement.

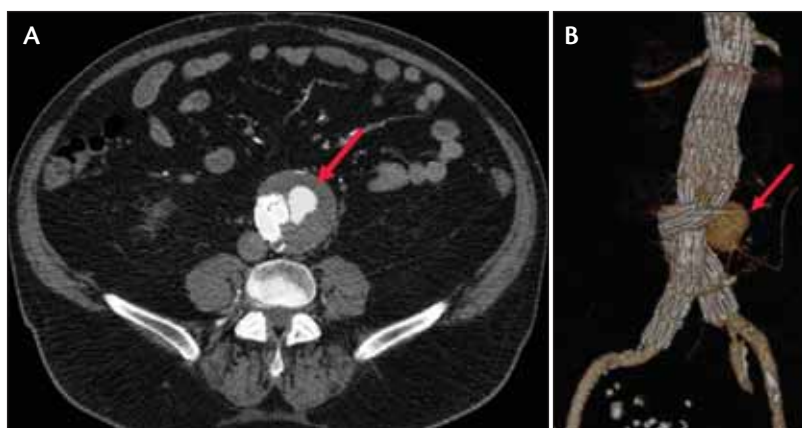


Figure 1. A CT scan of a type III endoleak (A). A CT angiogram with three-dimensional reconstruction of a type III endoleak (B).

The patient was taken to the operating room for an emergency exploratory laparotomy in which the infected endograft was explanted, the infrarenal aorta was resected and covered with an omental patch, and wide retroperitoneal tissue debridement and drainage was performed (Figure 4). Due to the integration of suprarenal barbs within the vessel wall, temporary proximal control was required, necessitating cross-clamping just above the renal arteries.⁵ Extra-anatomic revascularization was performed with an 8-mm, polytetrafluoroethylene (PTFE) heparin-bonded, right axilobifemoral bypass graft (Propaten, W. L. Gore & Associates, Flagstaff, AZ). Despite a diligent search for an enteric defect and blood noted within the lumen of several loops of small bowel, an AEF was not found during exploration. Total operative blood loss was approximately 500 mL.

Postoperatively, the patient's fever and leukocytosis resolved. Intraoperative tissue cultures yielded *C. albicans*, and fluconazole antifungal therapy was continued long term. Forty-eight hours after surgery, the patient suffered a stroke with right-sided paralysis. Carotid duplex ultrasound showed high-grade left external carotid artery stenosis and a stable left internal carotid artery occlusion.

On postoperative day 7, the patient also developed an intra-abdominal abscess near the aortic stump that required percutaneous drainage and long-term antibiotics for *Klebsiella pneumoniae*, which was found in the cultures. A follow-up barium swallow and small bowel study did not reveal any evidence of enteric erosion. All final urine and blood cultures were negative for bacteria and fungal species.

Once stabilized, the patient was transferred to a long-term care facility and received systemic anticoagulation therapy for his cerebrovascular disease. He later regained limited upper extremity function. The family withdrew care because of his progressive decline in mental status due to dementia, which was a result of multiple small-vessel cerebral infarctions. He died 90 days after endograft explantation without evidence of infection.

DISCUSSION

The true incidence of AEI remains unknown. Ducasse et al reported a 0.4% incidence of AEI in

9,739 procedures.² Similarly, the review by Fiorani et al of the literature and international practitioner survey yielded a 0.4% AEI incidence.⁶ Mortality rates after AEI have been reported as high as 18%.²

Staphylococcus aureus is the predominant pathogenic organism in AEI, showing that the risk of methicillin-resistant *S. aureus* (also known as MRSA) is presently rising.⁷ Other pathogens include *Escherichia coli*, *Klebsiella*, *Enterococci* and *Streptococci*, and rarely, *Listeria monocytogenes*.⁷⁻⁹ Fungal AEI, as shown in our patient, remains extremely rare, with only one other report in the literature in which the patient also died.¹⁰

Many factors have been shown to influence the occurrence of AEI. Extension of any retroperitoneal infection may contaminate an endovascular graft, imparting a very high risk of early mortality.⁷ Periprocedural nosocomial infections are associated with an earlier onset of graft infection at 1.4 years when compared with the average time of prosthetic graft infection at 3 years.¹¹ AEFs have been described in recent studies as a source of graft contamination due to mechanical forces leading to bowel erosion in up to 33% of graft infections.^{2,12,13} Interestingly, EVAR for mycotic aortic aneurysm disease has been performed successfully without the development of AEI.¹⁴ In our patient, the source of the pneumoretroperitoneum remains unclear and may have been the result of gas formation from fungal isolates that translocated from his recent esophagitis or percutaneous inoculation during the type III endoleak repair.

The association of adjuvant endovascular procedures, such as those performed for postoperative endoleaks, and increased incidence of AEI remains unclear. Eliason et al have reported on the development of AEI after coil embolization procedures.¹⁵ In a series of 65 patients, Ducasse et al discussed the relative incidence of infection of aortic endografts placed in the operating room compared with interventional radiology suites (38% vs 63%), stressing the importance of sterile conditions at the time of implantation.² It is of note that EVAR on our patient was performed in the operating room, while the endoleak repair was performed in an interventional radiology suite.

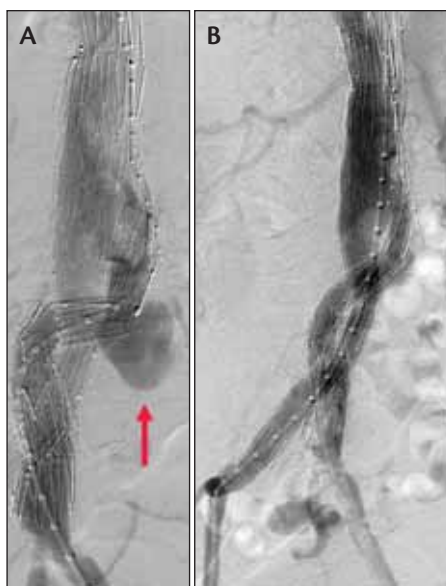


Figure 2. An angiogram with a type III endoleak involving a tortuous left graft limb (A). A successful repair of an endoleak using covered stents (B).

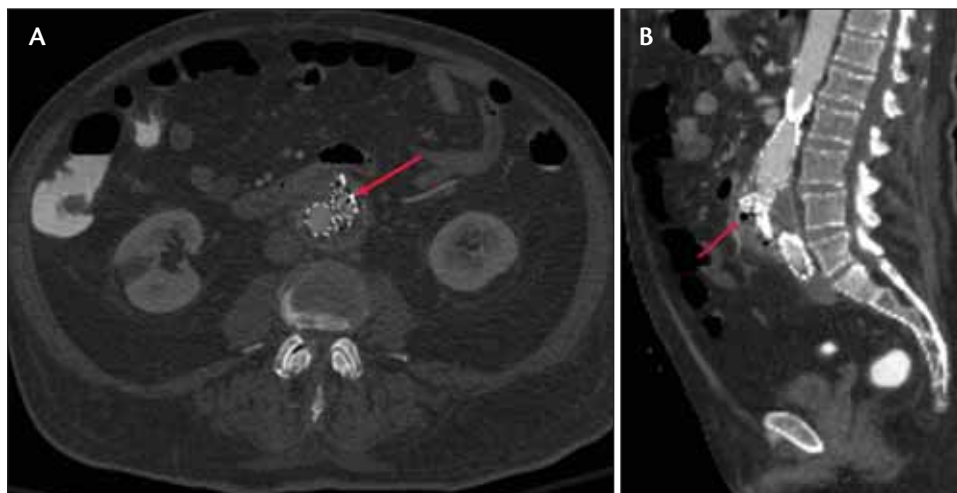


Figure 3. A CT scan revealing retroperitoneal air surrounding the aortic endograft (A). A sagittal CT scan showing retroperitoneal air surrounding the aortic endograft (B).

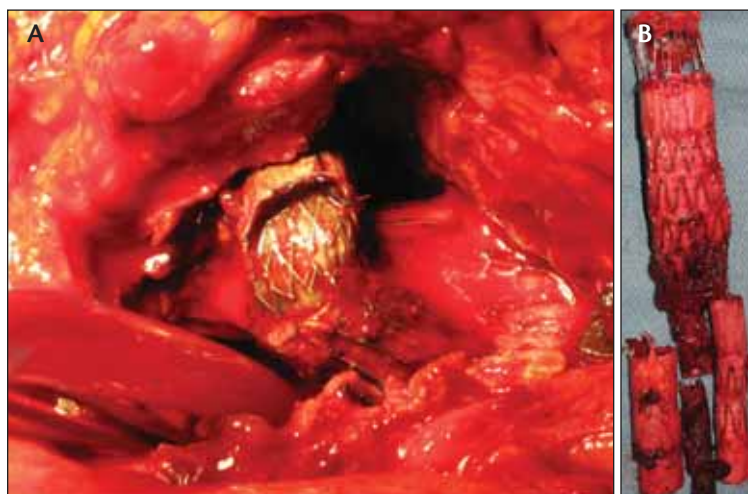


Figure 4. An intraoperative photograph of aortic endograft with purulent fluid in aortic sac (A). An explanted infected aortic graft (B).

Although definitive treatment of AEI is not yet established, it is likely that the clinical course of such infections will parallel that of aortic prosthetic infections after open repair. Therefore, treatment should be based on the patient's clinical condition and comorbidity at the time of presentation. In cases in which the patient cannot tolerate endograft removal, percutaneous or surgical drainage plus intravenous broad-spectrum antibiotics may be the only option to locally control the source of sepsis. Even though anecdotal survival has been reported following a conservative approach toward AEI,^{2,16} others have documented the mortality associated with such measures and suggest the removal of the endograft whenever possible.^{2,3}

In our patient, a high index of suspicion was present preoperatively for an AEF, which we attributed to the patient's antecedent history of esophageal candidiasis and gastrointestinal erosion. This suspicion guided our decision to explore the abdomen to eliminate AEF as a cause of infection and perform definitive resection and revascularization.

Additionally, a large amount of purulent fluid, later found to be fungal in nature, was encountered at exploration. These factors persuaded the surgical team to revascularize the lower extremities using an extra-anatomic bypass, as opposed to in situ reconstruction with either autogenous femoral vein or PTFE. A Propaten graft was chosen due to physician preference, although early published results of patency using a heparin-bonded PTFE conduit appear promising.¹⁷ We extend the possibility of these excellent results to all patients unless contraindicated (history of heparin-induced thrombocytopenia). Lower mortality rates have been recently reported with the use of in situ reconstruction using PTFE or rifampin-treated grafts after endograft explantation compared with extra-anatomic procedures.² Low mortality and amputation rates have been established using the neoaortoiliac system and may be an excellent option when reconstruction is required.^{18, 19}

CONCLUSION

AEI continues to be rarely reported; however, the widespread application of EVAR and adjunctive procedures for endoleak repair will likely be associated with an increase in incidence of this complication. To our knowledge, our patient is the second report of a fungal infection of an endograft. Definitive therapy for AEI should be guided by the patient's clinical condition, and endograft removal should be performed when possible. Additional experience with the management of this complication may influence future treatment and outcomes. ■

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