Endograft Infection After EVAR

An uncommon complication leads interventionists to opt for graft removal.

BY JENNIFER A. STABLEFORD, MD; THOMAS S. MALDONADO, MD; TODD BERLAND, MD; BILLY KIM, MD; MARK A. ADELMAN, MD; H. LEON PACHTER, MD; AND FIRAS F. MUSSA, MD

Graft infection and aortoenteric fistula after endovascular aneurysm repair (EVAR) is an underrecognized and underreported event. Although rare, it may have devastating consequences. Traditional treatment with extra-anatomic bypass and either simultaneous or subsequent graft removal has been challenged by in situ reconstructions with autologous femoral/popliteal veins, cadaveric homograft, or rifampin-impregnated synthetic grafts. With variable results on patency and reinfection rates, a standardized approach to those critically ill patients does not exist. We, herein, present a case of endograft infection that was unmasked after a secondary intervention.

CASE REPORT
A 73-year-old man with hypertension and atrial fibrillation underwent EVAR in September 2008 for a 6-cm infrarenal aortic aneurysm. Three months later, he presented with right leg claudication, and a computed tomography (CT) scan demonstrated thrombosis of the right iliac limb of the endograft. Mechanical thrombectomy failed to reestablish patency, and the patient was discharged. The next day, he developed massive upper gastrointestinal bleeding, but no source was demonstrated on upper and lower gastrointestinal endoscopy. A few days later, he developed fever of 105ºF and was transferred to our institution for management of possible endograft infection. On presentation, he was afebrile but tachycardic to 126 bpm.

A CT scan (Figure 1) demonstrated an air-fluid level around the endograft as well as within the aneurysm sac. Infection was confirmed with percutaneous drainage of purulent fluid and contrast drain study (Figure 2). Over the next few days, his tachycardia stabilized and full medical work up was completed. He underwent a staged procedure with extra-anatomic bypass (left axillofemoral-femoral) followed by total endograft explantation and primary repair of the duodenum (Figures 3 through 5).

The postoperative course was remarkably uneventful; the patient was discharged, tolerating oral and tube feeds. He was maintained on long-term oral antibiotic therapy.

DISCUSSION
With the wide acceptance of EVAR as the primary mode of treatment of aortic aneurysm, endograft infections have been reported more in recent literature (Table 1). Although the true rate of this complication is difficult to determine due to the rarity of occurrence, single-center case series estimate an incidence of 0.5% to 1.3% with a mortality rate ranging from 0% to 60%.

Table 2 lists some of the predictors of mortality due to graft infection.

Open conversion of these cases is a surgical tour de force. It often requires a large thoracoabdominal inci-
sion, supraceliac clamping, endograft removal from the aortic wall, and eventual reconstruction to an attenuated aortic stump. A particular problem with newer endografts is the suprarenal barbs used for proximal fixation. This represents an additional level of complexity that did not exist with infected traditional synthetic grafts.

Calligaro et al\(^4\) reported a series of nine patients with infected aortic grafts who could not tolerate surgical intervention and thus were treated with percutaneous drainage, instillation of antibiotic through the drain, as well as intravenous antibiotics for at least 6 weeks. Seven of the nine patients survived hospitalization, and no recurrent infections developed over a mean follow-up of 7.6 years. In a recent report, Ali et al\(^5\) reviewed the results of two centers at which in situ reconstruction with autologous tissue was the primary method of repair. A total of 187 patients with aortic graft infections underwent repair using femoral popliteal vein grafts, with an overall mortality rate of 14% and a 5-year survival rate of 52%. The primary patency rate was 81%, with an assisted primary/secondary patency rate of 91% and a limb salvage rate of 89% at 7 years.

Ducasse et al\(^3\) surveyed 40 international centers regarding their experience with endograft infection. Ducasse et al found a total of 65 cases of infected endografts on a survey of medical centers that perform
EVAR. Off those 65, 22 were not previously reported, whereas 43 were presented in previous publications. The overall infection incidence was 0.43%, with two-thirds classified as severe infection, and the overall mortality rate was 18%. Eighteen percent underwent nonoperative treatment with 36% mortality. The best treatment option was endograft removal with in situ prosthetic reconstruction and a mortality rate of 5.8%.

This case highlights a seldom-recognized complication of EVAR. Aortoenteric fistula is rare given the lack of direct communication between the endograft and bowel. The etiology is unclear and may be related to an initial infection of the graft, which subsequently leads to inflammation and fistulization to the duodenum. Furthermore, the role of secondary interventions on endograft infection remains to be speculative but worth studying at this time.

CONCLUSION
As follow-up continues for patients undergoing EVAR, endograft infection will be a more recognized complication with well-defined predisposing factors. It remains a challenging problem, and treatment should always be individualized. Although percutaneous drainage with antibiotic irrigation may be beneficial in patients who cannot tolerate surgical intervention, complete graft removal, when possible, with immediate or staged aortic reconstruction provides patients with the best chance of survival.

Jennifer A. Stableford, MD, is a fellow in the Division of Vascular & Endovascular Surgery at New York University Langone Medical Center in New York. She has disclosed that she holds no financial interest in any product or manufacturer mentioned herein. Dr. Stableford may be reached at jennifer.stableford@nyumc.org.

Todd Berland, MD, is a fellow in the Division of Vascular & Endovascular Surgery at New York University Langone Medical Center in New York. He has disclosed that he holds no financial interest in any product or manufacturer mentioned herein.

Billy Kim, MD, is a fellow in the Division of Vascular & Endovascular Surgery at New York University Langone Medical Center in New York. He has disclosed that he holds no financial interest in any product or manufacturer mentioned herein. Dr. Kim may be reached at billy.kim@nyumc.org.

Mark A. Adelman, MD, is Associate Professor and Chief of the Division of Vascular & Endovascular Surgery in New York University Langone Medical Center in New York. He has disclosed that he holds no financial interest in any product or manufacturer mentioned herein. Dr. Adelman may be reached at mark.adelman@nyumc.org.

H. Leon Pachter, MD, is Professor and Chair in the Department of Surgery at New York University Langone Medical Center in New York. He has disclosed that he holds no financial interest in any product or manufacturer mentioned herein. Dr. Pachter may be reached at leon.pachter@nyumc.org.

Firas F. Mussa, MD, is Assistant Professor in the Division of Vascular & Endovascular Surgery at New York University Langone Medical Center in New York. He has disclosed that he holds no financial interest in any product or manufacturer mentioned herein. Dr. Mussa may be reached at (212) 263-7311; firas.mussa@nyumc.org.