Mechanisms of EVAR Failure and New Surveillance Strategies

The rationale behind updating our surveillance protocols post-EVAR to decrease patient risks, as well as costs.

BY JILL K. JOHNSTONE, MD, AND GUSTAVO S. ODERICH, MD

Endovascular aneurysm repair (EVAR) has gained widespread acceptance for the treatment of abdominal aortic aneurysms. Prospective randomized studies have shown several short-term advantages compared to open repair, including lower mortality and morbidity rates.[1-3] EVAR surpassed open repair as the most commonly used treatment for abdominal aortic aneurysms, accounting for > 70% of all cases in most large referral centers.[4] Long-term results have been challenged by the presence of endoleaks, persistent aneurysm sac growth, and higher reintervention rates. In some patients, conversion to open repair and aneurysm rupture can occur.[1,2]

Lifelong surveillance after EVAR has been well-accepted to ensure continued clinical success and to prevent life-threatening aneurysm-related complications. Initial surveillance guidelines were based on pivotal trials and consisted of serial four-view abdominal x-rays and CT angiography (CTA) performed at 1, 6, and 12 months after the procedure and annually thereafter for at least 5 years. These recommendations were largely arbitrary and have not necessarily been corroborated by clinical data. Most recently, large amounts of clinical data from prospective studies, national datasets, and single-center experiences have been accumulated with late follow-up. Modes of device failure have been well-defined (Figure 1), which help to identify patients who are at increased risk for late complications or reintervention. New paradigms include change in surveillance schedule and greater utilization of duplex ultrasound (DUS) to avoid the added cost, as well as the increased radiation and contrast exposure, of CTA.[5]

MODES OF FAILURE

EVAR failure is a dynamic process that cannot be attributed to a single cause. Most often, it is a combination of several factors involving the patient, device, and physician

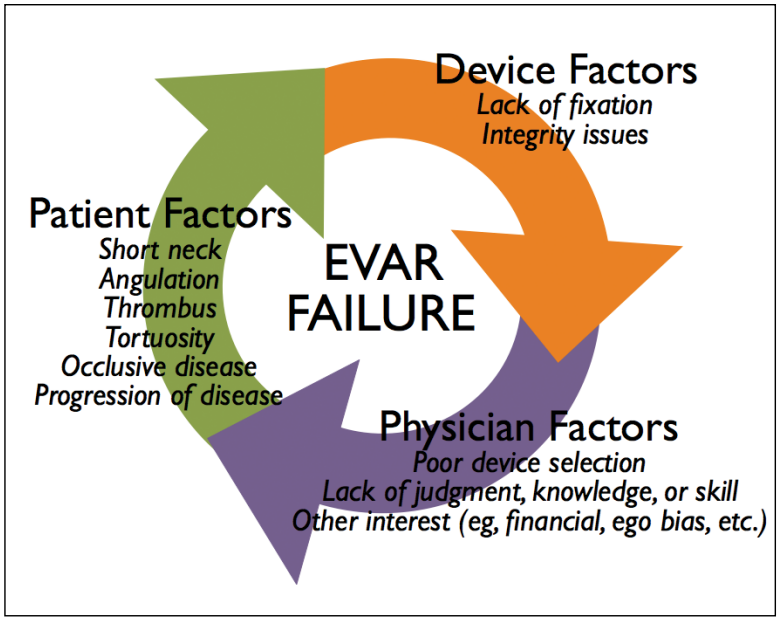

Figure 1. Mechanisms of failure after EVAR.

that result in failure. A classic example is the unexpected migration that led to failure of first-generation devices, which relied solely on radial force and not on active fixation. There is also increasing evidence that the progression of aortic disease results in a loss of proximal seal and late failure from poor proximal attachment, type I endoleak, and pressurization of the aneurysm sac. Lastly, the physician is responsible for treatment selection, including the specific type and extent of repair, device, and intraprocedural technique. All these factors have a great impact on outcomes and may ultimately result in EVAR failure.

Endoleak

Endoleak implies persistent blood flow outside of the lumen of the endograft and within the aneurysm sac, resulting in incomplete exclusion.[6] Endoleaks of all types have been well-documented to be associated with sac enlargement and rupture, representing the main target of surveillance protocols. Primary endoleaks (present in the first imaging study) occur in 20% to 25% of patients.[7-9] The majority of these are type II endoleaks (> 95%), which are independent of device selection and carry a relatively benign course provided there is no sac growth. Type I endoleak is infrequent (< 1%–3%) in patients with favorable anatomy, such as those treated within the anatomic guidelines.

Schanzer et al reported that 32% of post-EVAR patients developed endoleak and that 21% had sac enlargement.[10] Sternbergh et al found that the presence of endoleak on CTA (performed at 30 days) significantly increased the need for a secondary procedure at 5 years (42% vs 15%).[11] Type I and III endoleaks are associated with higher rates of reintervention. In the EUROSTAR registry, the reintervention rate was 54% for type I, 22% for type III, and 6% for type II endoleaks.[12] Type II endoleaks are the most common form of endoleak, but their clinical importance is less clear. Persistence of a type II endoleak on follow-up imaging correlates with sac enlargement, reintervention, rupture, and the need for conversion to open repair.

Migration

Migration has been defined by Society for Vascular Surgery standards as ≥ 10 mm of movement. A revised report suggests movement of > 5 mm or any movement requiring treatment.[6] Factors associated with migration include lack of active fixation, short sealing zone, progression of aortic disease, and other adverse anatomical features such as angulation, thrombus, calcification, conic neck, and excessive oversizing. In pivotal device trials, migration was < 1% for Zenith (Cook Medical, Bloomington, IN) and Excluder (Gore & Associates, Flagstaff, AZ) endografts, but was noted in 4% of patients who were treated with Talent (Medtronic, Inc., Santa Rosa, CA) or Powerlink (Endologix, Inc., Irvine, CA) endografts.[7,8,13] Zarins et al reported a 9% migration rate using the AneuRx device (Medtronic, Inc.) at 3 years. In other studies, this rate for the AneuRx device reached up to 22% at 3 years.[14-17]

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Iliac Limb Occlusion

The patency rates of endograft iliac limbs are excellent.[2,18,19] Several first-generation devices have undergone improvements with spiral stent technology, which is more forgiving of iliac tortuosity. Anatomical factors have been identified that increase the risk of occlusion, including narrow aortic bifurcation, iliac occlusive disease, tortuosity, extension into the external iliac artery, and excessive oversizing. In a 5-year follow-up study of the Zenith endograft, there were three iliac limb stenoses (2%) and eight iliac limb occlusions out of 143 patients.[13] The Gore Excluder device had no iliac limb occlusions at 5-year follow-up, and at 6-year follow-up, the Endologix Powerlink device had six (3.8%) graft limb stenoses or occlusions.[8,9]

Progression of Aortic Disease

Unquestionably, progression of aortic disease plays a significant role in device failure. This is often noted after 5-year follow-up and therefore is often not well-documented. Brown and associates recently reported on the association of familial history of aneurysm disease with more proximal aortic pathology involving the ascending aorta, arch, thoracic, and visceral aortic segments.[20] Other stigmata of aortic disease include ectasia, thrombus, or synchronous aneurysms. The Southwestern group has also reported that large aneurysms are associated with shorter neck length, emphasizing that growth occurs in the cranial axis.[20] These features affect late failures due to migration and type I endoleak and should be taken into consideration when planning open or endovascular procedures in younger patients with longer anticipated survival.

Surveillance Paradigm

Despite a few reports proposing limited surveillance in select patients, there is little question that EVAR necessitates lifelong surveillance to detect endoleak, migration, structural graft failure, change in aneurysm sac size, and limb complications. However, clinical data and modes of failure suggest that the initial surveillance program proposed by trials needs to be revised. These surveillance regimens included four-view abdominal films and CT imaging at 1, 6, and 12 months with annual CT imaging thereafter. Late results of these trials allowed revision of this approach. CT imaging accounts for > 65% of the total cost[21] and has potential late complications due to radiation and contrast exposure.[22-24]

Magnetic resonance imaging is a sensitive modality to detect endoleak but has not gained popularity due to the advantages of CT in assessing device integrity, aortic pathology, and other failure modes. DUS has been increasingly utilized with high specificity (91%) and negative predictive values (91%–100%) in the detection of endoleaks when compared to CT imaging.[25] A meta-analysis that compiled 25 studies comparing DUS to CT for the detection of endoleaks showed that the pooled sensitivity for DUS compared to CT was 0.74, and the specificity was 0.94.

Timing of Surveillance

In the 5-year follow-up of the pivotal United States Zenith multicenter trial, Sternbergh and colleagues retrospectively looked at the presence of endoleak during postoperative surveillance and its affect on aneurysm-related morbidity. They found that the absence of endoleak on CTA at 30 days was associated with an 83% freedom from aneurysm-related morbidity rate at 5 years compared to 55% in the group that demonstrated the presence of endoleak.[11] Kirkpatrick and colleagues suggest that if CTA findings 1 month after EVAR are negative for abnormalities, additional CTA imaging can be delayed for up to 3 years. A normal 1-month CTA was correlated with a 92.9% chance of not developing a complication at a mean of 3.4 years and a 97.1% chance of freedom from undergoing a secondary intervention.[26]

Goncalves et al used the first postoperative CTA to risk stratify patients to determine their postoperative surveillance. They reported that a lack of endoleak and adequate proximal and distal seal zones (length ≥ 10 mm) constituted the low-risk group, and these patients could defer further imaging for up to 5 years.[27]

In 2009, the Society for Vascular Surgery published further guidelines on follow-up surveillance after EVAR in light of these studies and in an effort to contain cost and risk to the patient. Contrast-enhanced CT imaging is recommended at 1 and 12 months. If the CT scan at 1 month shows an abnormality, such as an endoleak, then a 6-month CT scan with contrast is recommended. At the 12-month CT scan, if no abnormality is detected, then imaging can continue with yearly DUS.[5]

A new surveillance regimen can be proposed based on results of the first imaging study performed 1 to 3 months after EVAR.

RECOMMENDATIONS

In the absence of randomized trials to evaluate novel surveillance protocols, a few recommendations can be made based on clinical data from the aforementioned studies. Clearly, routine CTA is unnecessary. A new surveillance regimen can be proposed based on results of the first imaging study performed 1 to 3 months after EVAR. In most centers, CTA is performed to outline morphologic changes after the procedure, allowing high sensitivity to detect endoleaks, migration, or structural problems. In the absence of an endoleak, annual DUS has been widely adopted by several centers, including ours.

Sternbergh and colleagues reported a revised surveillance program on behalf of the Zenith trial investigators, suggesting that the 6-month visit may be omitted for patients with no endoleak at the first visit. At 12 months, if there is a continued lack of endoleak and the aneurysm sac is stable or has shrunk, then further follow-up is performed with yearly DUS. The presence of endoleak or an increase in sac size would require further evaluations with CTA.[11] The benefit of this surveillance regimen is a decrease in cumulative radiation and iodinated contrast exposure and a decrease in cost, without increasing the aneurysm-related mortality rate. ■

Jill K. Johnstone, MD, is a Clinical Fellow in Vascular and Endovascular Surgery, Division of Vascular and Endovascular Surgery, Mayo Clinic College of Medicine in Rochester, Minnesota. She stated that she has no financial interests related to this article.

Gustavo S. Oderich, MD, is Professor of Surgery and Director of Endovascular Therapy, Division of Vascular and Endovascular Surgery, Mayo Clinic College of Medicine in Rochester, Minnesota. He has disclosed that he serves as consultant for W. L. Gore and Cook Medical, with all fees paid to Mayo Clinic and no direct income. Dr. Oderich may be reached at (507) 284-1575; oderich.gustavo@mayo.edu.

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