Choosing the Right Option for Treating EVAR Failure: When to Use and Avoid Open Repair

A discussion of patient selection and when open repair should be considered based on the type of endoleak and presence of infection.

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Endovascular aneurysm repair (EVAR) is the most common therapy for infrarenal abdominal aortic aneurysm (AAA) treatment in the United States. Long-term outcomes are comparable to that of open surgical repair, and there is concern that late EVAR mortality may surpass that of open surgical repair. The EVAR reintervention rate of 18% and EVAR rupture rate of 5.4% at 8 years explain why the early mortality advantage with EVAR is lost over time.[1] Therefore, a strategy of lifelong surveillance is mandatory to detect device failures, endoleaks, and AAA sac expansion.

Modes of EVAR failure are often multifactorial, including a combination of endoleaks, graft migration, graft occlusion, progression of aortic or iliac aneurysmal disease, AAA sac expansion, and EVAR rupture. The process of EVAR failure is often dynamic, and most patients have multiple factors contributing to any given scenario. Frequently, a persistent endovascular strategy is employed to treat EVAR failures, partly due to the perceived complexity of open endograft explantation. It is critical to employ a durable solution to prevent AAA rupture after EVAR failure has occurred. Although elective EVAR conversion has yielded acceptable mortality rates, urgent or emergent EVAR conversions result in mortality rates of 29% to 37%.[2,3]

PATIENT SELECTION FOR OPEN REPAIR

When determining the appropriate salvage for EVAR failure, the patient's physiologic risk for reintervention, mode of EVAR failure, and EVAR history dictate therapy. Endoleaks with AAA sac expansion are the leading cause for endograft explantation, followed by infection, thrombosis, and migration. Initially, EVAR explantations were reported to carry mortality rates of 10% to 40% in heterogeneous groups that included infection, urgent presentation, and ruptures. These patients often required complete graft excision and required suprarenal or supramesenteric aortic clamping for repair. Several groups adopted a strategy to reduce perioperative mortality by minimizing the surgical complexity. Both limited endograft explantation and utilization of an infrarenal aortic clamp have demonstrated a lower perioperative mortality and morbidity for patients; this strategy yields perioperative mortality rates of < 3% in selected patients.[4,5]

Figure 1. Multiple coils are visualized in the AAA sac. In addition, a large amount of liquid embolic material has formed a cast within the AAA sac.

The choice of therapy should be driven by the surgeon's ability to achieve a durable solution that will protect patients from AAA rupture over their lifetime. The foundation of this decision is a thorough risk assessment for EVAR explantation. An analysis of the Vascular Quality Initiative demonstrated that age, congestive heart failure, chronic obstructive pulmonary disease, previous leg bypass, previous carotid revascularization, suprarenal aortic cross-clamping, and female sex were associated with 30-day mortality for both EVAR explantations and primary aortic repairs.[6] When preoperative risk factors were controlled, EVAR explantation was not associated with increased mortality. Perioperative mortality was driven by preoperative risk factors and not endograft explantation. For instance, a 71-year-old man with a normal stress test, no other preoperative risk factors, and anatomy favorable for an infrarenal clamp carries an estimated 30-day mortality of 3% after endograft explantation.[6] The perceived anatomic challenges of EVAR explantation do not portend higher mortality, and in low- to moderate-risk patients, EVAR explantation should be considered for EVAR failures.

The leading causes of post-EVAR death are coronary artery disease and cancer. Beyond preoperative risk assessment, it is prudent to ensure that lung, colon, prostate, and breast cancer screening has occurred. A determination of high physiologic risk and short life expectancy may drive the decision for endovascular therapy only or no therapy.

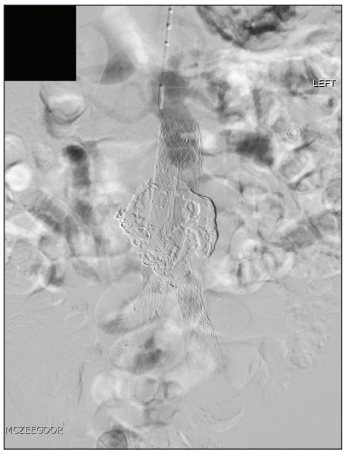

Figure 2. The stent graft is 3 to 5 mm below the lowest renal artery with minor angulation, which likely represents proximal stent graft migration.

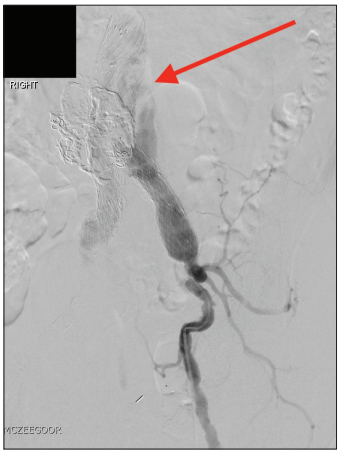

Figure 3. A type Ia endoleak is shown filling the AAA sac. Because of the amount of embolic material in the AAA sac, CT could not clearly identify the endoleak.

ENDOLEAKS

Type Ib and IIIa/IIIb Endoleaks

EVAR failure in the iliac landing zones is caused by continued iliac artery degeneration. Treating these patients with either a branched iliac device or hypogastric artery embolization and limb extension into the external iliac artery is a low-risk, durable endovascular solution. However, in patients who have thoracic aneurysms or thoracoabdominal aneurysms and undergo thoracic EVAR (TEVAR) or who have had previous TEVAR plus EVAR, spinal cord perfusion is paramount. If hypogastric artery perfusion cannot be maintained through endovascular means, open reconstruction can be performed through either a transabdominal or retroperitoneal approach.

Isolated type IIIa and IIIb endoleaks are also favorable for endovascular repair. When detected early without other modes of failure, these endoleaks can be resolved with a bridging stent graft or relining procedure. The AAA sac size, aortic sealing zones, stent graft angulation, and migration should be critically evaluated, as it is very important to resolve all modes of failure.

Type Ia Endoleaks

Inadequate proximal seal zone is the leading cause of EVAR failure among EVAR explantations. Proximal aortic dilation is implicated in failure, which may be due to neck failure over time in the treatment zone or inappropriate selection for EVAR. The foundation of endovascular salvage relies on the ability to extend the proximal seal zone. Palmaz stents (Cordis, a Cardinal Health company) and endostaples do not extend the seal zone, but instead attempt to achieve seal with the stent graft in its current location. An aortic cuff may increase the seal zone if the endograft was deployed ≥ 5 mm from the lowest renal artery. Off-label use of renal chimney grafts and fenestrated EVAR (FEVAR) will extend the seal zone into the paravisceral aorta; however, the long-term durability of this treatment needs to be critically assessed in each patient. Depending on the stent graft design, chimney procedures and FEVAR may be challenged with a short stent graft landing zone proximal to the flow divider, and technically, this makes it very difficult to achieve adequate overlap with available devices. Furthermore, a long endograft body provides a solid landing zone for FEVAR extensions.

Lastly, EVAR explantation eliminates the need for additional seal, late endoleaks, reintervention, and surveillance. As opposed to chimney grafts and FEVAR, the repair can often be achieved at the infrarenal level, mitigating the risk of visceral and renal stents. In addition, open repair can treat all endoleaks will little additional risk to the patient. If a patient has both a type Ia endoleak and also a history of a complicated type II endoleak with AAA sac

expansion, open repair eliminates all mechanisms of failure. Undergoing a complicated FEVAR or chimney procedure to resolve the type Ia but not address the type II endoleak would leave the patient at risk for AAA rupture. Addressing both endoleaks through endovascular means adds significant operative time, fluoroscopic time, and contrast burden. If technical success is achieved, clinical failure may occur with continued sac expansion despite reintervention.

Type II Endoleaks

Type II endoleaks are the most common indication for reintervention after EVAR. Endovascular embolization of type II endoleaks can be extremely challenging and requires large doses of contrast and extended fluoroscopic times. In addition, multiple procedures are often needed to achieve endoleak resolution. Translumbar punctures, transcaval sac access, and accessing the sac behind an iliac limb have been attempted for complete sac ablation. Often, these techniques will halt aneurysm sac enlargement, but in some cases, sac expansion may persist despite type II endoleak treatment. In this setting, it is critical to exclude type I and III endoleaks as the etiology for treatment failure; this is crucial when devising treatment options. Once type I and III endoleaks are excluded, open surgical evaluation is necessary to assess the endograft for persistent sac expansion despite type II endoleak therapy. When addressing these cases, it is important to prepare for EVAR explantation; however, if the proximal seal zone, iliac seal zones, and graft junctions are intact, EVAR explantation is not required. In this setting, the embolic material is removed from the sac, the lumbar arteries and inferior mesenteric artery are ligated, and the AAA sac is then plicated around the endograft.

Isolated type II endoleaks that occur with rapid sac expansion or threaten seal zones should be approached in an aggressive fashion. Often, type II endoleaks are treated with multiple embolizations over several months. Figures 1 to 3 illustrate a patient in whom embolization of the lumbar and inferior mesenteric arteries was employed over the course of four reinterventions. Due to continued sac expansion, translumbar sac access was obtained and embolization was performed using Onyx liquid (Medtronic). The AAA sac continued to expand, leading to a new type Ia endoleak. At this juncture, the decision was made for endograft explantation, as opposed to continued attempts to arrest sac growth and devising an endovascular solution for proximal seal. In retrospect, open surgical evaluation should have been performed prior to the development of a new type Ia endoleak. Identifying these patients can be challenging. When devising a treatment plan for sac expansion, it is critical to evaluate the stability of the overall EVAR repair in the context of a new endoleak with sac expansion.

ENDOGRAFT INFECTION

Endograft infection is a lethal condition. Mortality is 50% to 70% in selected patients treated with antibiotics alone at a follow-up period of 4 months.[7] Early open surgical explantation is necessary to prevent aortic rupture and death. It is critical to employ a supraceliac aortic access approach and be prepared for visceral and renal revascularization. The infected endografts must be removed and the infected aortic wall, abscess cavities, and necrotic tissue must be resected. Cryopreserved aortic allograft, autogenous femoral vein, and antibiotic-soaked aortic grafts may be utilized with an omental wrap. Early mortality rates are 11% to 39% in the best circumstances.[7,8] Chronic infection, continued sepsis, excessive blood loss, and organ failure drive the high early mortality rates; late graft infection accounts for late aortic-related mortality.

SUMMARY

Isolated endograft failures can often be treated with an effective and durable endovascular solution. Patients with complicated proximal failures, acute sac expansion, sac expansion despite multiple reinterventions, or endograft infection should be evaluated for open surgical revision. The preoperative risk profile drives mortality related to endograft explantation after EVAR, while partial graft explantation and employing an infrarenal clamp mitigate the perceived difficulty of open revision. ■

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