Approach to Treatment of Refractory Type II Endoleaks

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ABSTRACT: Endovascular repair of abdominal aortic aneurysms (EVAR) has been well established over the past two decades. However, high reintervention rates and delayed aneurysm rupture have been reported in early trials, necessitating continued surveillance. Endoleaks are the most common complication following EVAR, and management of endoleaks can often be problematic. Technical success is predicated on correct identification of the endoleak type. Type II endoleaks are most commonly seen. Successful treatment of continued aneurysm sac growth related to type II endoleaks can be approached by either transarterial or direct sac injection. However, complete exclusion of the inflow and outflow vessels contributing to backfilling of the aneurysm sac is required for successful long-term outcomes, namely prevention of continued aneurysm sac growth. Techniques to approach such repair of refractory type II endoleaks are discussed.

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BACKGROUND

Endovascular repair of abdominal aortic aneurysm (EVAR) is a preferred alternative to conventional open repair due to reduced postoperative mortality rates, and similar long-term outcomes in 4 large randomized studies.1-4 While endovascular repair is considered less invasive, consistently higher reintervention rates were observed in those studies, ranging from 5.1% to 29.6%. Endoleaks are the most frequent complication followed by graft migration, accounting for roughly half of reinterventions, and have been implicated in up to 68% of reports of delayed abdominal aortic aneurysm (AAA) rupture following EVAR.5,6 Thus, long-term imaging surveillance is a necessity, a feature unique to EVAR, contributing to overall cost of endovascular repair. Failure to prevent aneurysm enlargement is likely responsible for eroding the initial survival benefit of EVAR compared to open repair,1-4 although the specific contribution of type II endoleak (T2E) in this regard is less certain.

DETECTION, CLASSIFICATION, AND SIGNIFICANCE OF ENDOLEAK

Endoleak refers to the continued blood flow in the
aneurysm sac outside the confines of the stent graft, causing persistent arterial pressurization of the aneurysm sac. Endoleaks are classified according to route of flow. Type I refers to attachment site leak, occurring at either the proximal (Ia) or distal (Ib) end of the stent graft. Type II refers to retrograde sac filling via branch vessels, commonly lumbar or inferior mesenteric arteries. Type II endoleaks can occur as a single vessel (with to-and-fro flow into the aneurysm sac), termed type IIa, or as multiple vessels (flow-through hemodynamics), termed type IIb. Type III refers to a junctional defect or modular disconnect in the stent graft, or fabric disruption allowing direct communication with the aneurysm sac. Type IV refers to fabric porosity, allowing seepage of blood products across the graft into the aneurysm sac despite absence of a focal defect. Finally, type V refers to endotension, in which an anatomic or structural cause is not identified on angiographic studies, yet the aneurysm sac fails to regress following repair or demonstrates progressive enlargement (generally defined as a 5-mm increase in axial sac diameter). Endotension is also known as endoleak of undefined origin.

Several modalities are suitable for detection and classification of endoleak, including computed tomography (CT), magnetic resonance (MR), digital subtraction angiography (DSA), and contrast-enhanced ultrasonography (CEUS). Computed tomography angiography (CTA) is the most widely used and has excellent reproducibility. Arterial and delayed post-contrast phase images are generally acquired to assess for contrast opacification within the aneurysm sac. Thin section data are then closely evaluated to identify the presence of patent branch vessels arising from the aneurysm sac, in the case of T2E. In one study, CTA and DSA were in agreement regarding endoleak classification in 86% of patients. CTA incorrectly classified the type of endoleak in 5 of 36 patients, resulting in a significant change in patient management in 4 of these patients. In another study in which a variety of CT protocols were used (several lacking images in the angiographic phase), 9 patients initially thought to have T2E were instead treated for type I or type III endoleak, with the remaining 16 patients in the cohort proceeding to specific treatment for T2E. For this reason, selective DSA is essential prior to treatment of an endoleak.

Magnetic resonance angiography (MRA) is at least as sensitive as CTA, but is best suited to evaluation of nitinol stent grafts because other stent materials cause substantial artifacts, which limit sensitivity. Ultrasonography (US) is also capable of detecting endoleaks, particularly with use of microbubble contrast agents. Both modalities have occasionally demonstrated endoleaks that were occult on CTA, although in many cases the clinical implications of such findings are not well known. Additionally, US evaluation is operator dependent and patient dependent. Although conventional US is considered less sensitive than CTA or MRA, use of spectral Doppler allows identification of aneurysms with bidirectional or low intrasac flow, which are characteristics associated with spontaneous closure and therefore implying conservative treatment.

Identification of cause is essential because management is highly dependent on the type of endoleak. Type I and III are frequently repaired at the time of initial graft placement if recognized, as they confer a higher risk of AAA rupture. In contrast, T2Es have traditionally been considered a benign condition and managed conservatively, occurring in approximately a third of patients following EVAR. Nearly 80% of
early T2E (occurring within 6 months of aneurysm repair) can be expected to resolve spontaneously.7 In one series of 754 patients undergoing elective repair, 28.7% developed at least one T2E in a mean follow-up period of 31.1 months.11 Within this group, 40.3% demonstrated aneurysm sac enlargement but only approximately one-fifth required specific treatment for the T2E.

In contrast, patients with T2E persisting beyond 6 months are at increased risk for aneurysm sac growth (odds ratio [OR], 25.9;  P < .001); persistent T2E is also a significant predictor of aneurysm rupture ( P = .03).7 Several studies have failed to show a difference in survival, however, in either patients with T2E or those requiring secondary reintervention, compared to patients without T2E or reintervention.7,11,12 Interestingly, patients with coronary artery disease, chronic obstructive pulmonary disease, and tobacco use may be more likely not to develop T2E and/or require reintervention,11 but this is not consistent across studies.7,12,13

In our institution, if a T2E is identified on the post-EVAR CT, we typically follow these patients more closely, at no greater than 6 month intervals for the first 12 to 18 months. If there is growth of the aneurysm sac, repair is indicated. No specific number is used as an indicator; if the growth is believable (greater than variation of measurement), then treatment is explored.

**APPRAOCH TO REPAIR**

When approaching T2E, an important concept for successful and durable treatment is parallel to that of treatment of an arteriovenous malformation (AVM). When treating an AVM, if the nidus is not treated, additional collateral feeding arteries will be recruited and the AVM will survive.14-16 In a similar fashion, if only the feeding vessel to an endoleak is embolized, new arteries will feed the aneurysm sac; this concept was well demonstrated by Baum et al, whereby the initial success of transarterial IMA embolization was tempered by recanalization in 7 of 8 patients by collaterals.17,18 The need for subsequent reintervention has been demonstrated by others when coil embolization is used alone,13 especially in the T2Es caused by lumbar arteries.19 Therapy is therefore aimed at achieving stasis of flow within the aneurysm sac, either with use of
glue or coils, and embolization of the feeding (inflow and outflow) vessels. This can be accomplished by either transarterial or translumbar routes, which have shown similar durability. In our experience we have employed both treatment routes as an initial mode of therapy, and in some cases we have used both routes in the same patient.

Initially, CT angiography is obtained to determine if there is an endovascular pathway to the aneurysm sac (Figure 1). As the majority of T2Es occur from the inferior mesenteric artery (IMA) or lumbar arteries, the superior mesenteric artery (SMA) to IMA and internal iliac to lumbar collateral pathways are examined. These collateral systems are not always negotiable due to vessel size and caliber, so a percutaneous approach is the only option in many cases. If an appropriate route is identified, the transarterial approach is attempted.

The SMA or internal iliac arteries are selected with a 5 Fr base catheter, often reverse curve for SMA. A microcatheter and microwire are then used to navigate the collateral pathway to the aneurysm sac (Figure 2). Of note, if Onyx (Covidien) is considered, the correct microcatheter should be used because not all are rated for dimethylsulfoxide use (rated catheters include the Marathon Micro Catheter, UltraFlow Micro Catheter, Echelon Micro Catheter, and Rebar Micro Catheter; Covidien).

If the transarterial approach is not feasible or fails, a percutaneous translumbar approach is utilized. The procedure at our institution is similar to that described previously, with some modifications. The typical scenario is with the patient prone under moderate sedation. Antibiotic prophylaxis is typically administered immediately prior to the procedure. Fluoroscopic
guidance is usually adequate when osseous and metallic landmarks are available. Left lumbar is favored, but right-sided transcaval is acceptable, though care must be taken to avoid nontarget embolization of the pulmonary arteries. To minimize traversal of the paraspinal musculature, 30-degree to 60-degree entry is typical. An 18-gauge to 20-gauge needle is used for access. Once blood return is achieved, positioning is confirmed with contrast (Figures 3 and 4). In our experience, directionality is often helpful to direct embolic into specific locations or microenvironments, therefore a system is set up that allows for directional catheters. Over a microwire, the needle is exchanged for a triaxial Neff Percutaneous Access Set (Cook Medical) or Ac-custick (Boston Scientific). The wire and inner dilator are removed and through the outer component, a microcatheter is advanced with or without a 4 Fr angled diagnostic catheter. In addition to directionality, having such a system allows one to easily switch microcatheters

Figure 3. Fluoroscopic guidance during translumbar approach (A). Contrast injection to verify catheter position within the aneurysm sac (B). A feeding vessel is identified, consistent with type II endoleak.

Figure 4. Translumbar access using the triaxial Neff Percutaneous Access Set (Cook Medical) (A), with subsequent sac embolization using N-butyl cyanoacrylate (B, C). Use of Onyx in a similar case with translumbar access, delivering sufficient liquid embolic to fill the patent portion of the aneurysm sac (D, E), as well as a feeding vessel (F).
if the first is “burned” because of embolic clogging.

Again, whether a transarterial or translumbar approach is utilized, the critical goal is the embolization of the sac itself (akin to the nidus of an AVMs) and the feeding vessel or vessels, rather than only the aneurysm sac or a feeding vessel. In an ideal treatment, embolic agent should fill the patent portion of the sac and extend partially into the feeding and draining vessels (Figure 5).

Both Onyx and N-butyl cyanoacrylate have been used with success. Some advantages of Onyx have been described previously, although there is a learning curve with both agents (Table 1).

If percutaneous or endovascular options fail or are not technically feasible, operative treatment may be con-
sidered. Generally, the perceived risk of aneurysm rupture should exceed expected perioperative morbidity and mortality in this circumstance. Despite generally favorable long-term technical success in treatment of endoleaks, operative risks are significant and delayed open conversions are associated with worse patient outcomes, although the subset of patients treated electively for ligation of culprit vessels (typically IMA or lumbar branches) with endograft preservation fare much better.\textsuperscript{23}

**FUTURE DIRECTIONS**

Endovascular techniques will continue as a favored approach in the treatment of AAA. Even with currently available devices, late complications including persistent endoleak and delayed aneurysm sac enlargement will occur, necessitating strict imaging follow-up for the post-EVAR patient population, frequently in the form of CT angiography at initial follow-up. Typically, patients with stable or decreasing aneurysm sac diameter will transition to noncontrast CT surveillance. In contrast, patients identified with T2E are followed more closely and are treated if there is evidence of sac enlargement. This may be accomplished with transarterial or translumbar routes, or a combination of the two, with the goal of therapy being embolization of the aneurysm sac and feeding (inflow and outflow) vessels. Two commercially available liquid embolics were discussed, N-butyl cyanoacrylate glue and Onyx. There has been early work looking at the use of sclerosing gels for treatment of enlarging aneurysm sacs secondary to T2Es; however, this work remains in preliminary studies requiring further evaluation.\textsuperscript{23}

Recently, a novel sac-anchoring endoprosthesis developed by Endologix promises to substantially reduce the incidence of T2E following EVAR. The Nellix aortic sealing device consists of a stainless steel, balloon expandable endoframe that is deployed in a kissing

*Figure 5. Use of liquid embolic agents in the treatment of type II endoleaks will allow delivery of sufficient embolic to the patent portion of the aneurysm sac as well as the contributing feeding vessels (A, B).*
configuration at the aortic neck proximally with extension into the common iliac arteries. An “endobag” constructed of nonporous expanded polytetrafluoroethylene-based material surrounds the endoframe components, and is subsequently filled with a biocompatible, nonbiodegradable polyethylene glycol-based polymer. The endobag conforms to the aneurysm sac to provide a seal at the aortic and iliac ends, but also seals side branch flow and eliminates the endoleak space within the aneurysm sac.24 To date, published data on 34 patients treated with the Nellix device have demonstrated no AAA sac enlargement at 1 year (32 patients) or at 2 years (5 patients).25 Furthermore, one-half of the patients were not candidates for endovascular repair, based on the IFU of current FDA-approved aortic endografts. The Nellix sac-anchoring endoprosthesis is currently on trial but not FDA-approved in the United States.

Innovations such as these will continue to advance the endovascular treatment of AAA. Factors such as the rate of aneurysm sac enlargement following EVAR, the need for continued surveillance, and the delayed risk of AAA rupture likely explain, in part, the similarity in long-term survival between EVAR and traditional open repair. With the use of currently available devices, identification and comprehensive treatment of T2E in the setting of enlarging sac diameter remains an important component of endovascular management of AAA. ■

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