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Management of Type II Endoleaks: Available Options for Treating the Most Common Type of Endoleak After EVAR

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Management of Type II Endoleaks

Available options for treating the most common type of endoleak after EVAR.

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According to the National Surgical Quality Improvement Program, 75% of abdominal aortic aneurysms are treated with endovascular techniques compared to open repair,[1] as endovascular aneurysm repair (EVAR) is associated with decreased periprocedural mortality, complications, and length of hospital stay.[2,3] However, some studies have shown an increased rate of reinterventions in EVAR compared to open repair,[4,5] a percentage of which are due to aneurysm growth secondary to an endoleak. Endoleaks can occur in up to 20% to 25% of patients after EVAR,[6,7] with type II endoleaks being the most common.

Type II endoleaks account for at least 40% of all endoleaks.[8] They commonly occur from retrograde collateral blood flow into the aneurysm sac, typically from a lumbar artery or the inferior mesenteric artery (IMA).[9] Less common sources of type II endoleaks include accessory renal and median sacral arteries. In patients who require endograft limb extension across the internal iliac artery, this vessel can also serve as a source of endoleak if it is not adequately embolized. Endoleaks can lead to enlargement and pressurization of the sac, leading to rupture (which is uncommon). The retrograde collateral blood flow may or may not result in sac pressurization, as not all of these endoleaks are associated with aneurysm growth. Type II endoleaks can be subdivided into type IIa, which has a single causative vessel involved with "to-and-fro" flow in the aneurysm sac, and type IIb, in which multiple vessels are involved.

NATURAL HISTORY OF TYPE II ENDOLEAKS

Compared to other endoleaks, type II endoleaks generally have a benign course, and many spontaneously resolve. In our experience, type IIa endoleaks have a greater propensity to spontaneously resolve than type IIb, which tend to be more complex. In one study, only 19% of type II endoleaks were associated with aneurysm growth requiring intervention.[10] In another study, only 20% required intervention.[11] However, it has been demonstrated that the presence of a persistent type II endoleak is associated with aneurysm sac growth, increased reintervention rates, conversion to open repair, and aneurysm sac rupture.[12] A persistent type II endoleak is described as persisting beyond 6 months. Predictors of a persistent type II endoleak include a patent IMA > 2.5 mm in diameter, a lumbar artery > 1.9 mm in diameter, and more than two lumbar arteries that extend from the aneurysm sac.[13,14]

FOLLOW-UP AFTER EVAR AND ENDOLEAK EVALUATION

After EVAR has been completed, our follow-up protocol consists of CT angiography (CTA) at 1, 6, and 12 months, and annually thereafter. Some patients may be followed every 2 years, especially in the setting of a shrinking sac in the absence of an endoleak. Patients with renal insufficiency may be followed with duplex ultrasound and noncontrast CT. Time-resolved magnetic resonance angiography is used selectively at our center, sometimes to better determine the flow dynamics of an endoleak seen on CTA.

It is critical that a proper imaging protocol is utilized for cross-sectional imaging after EVAR to ensure that endoleaks are identified. A three-phase scan consisting of a noncontrast scan, an arterial phase, and delayed imaging is essential, and review of previous studies is mandatory. Once an endoleak is identified, it is critical to determine the endoleak type in order to guide management. Although cross-sectional imaging can diagnose the presence of an endoleak, the type of endoleak is not always evident on cross-sectional imaging alone. In these cases, diagnostic angiography is required to determine the precise etiology in order to guide subsequent therapy. Diagnostic angiography should include an aortogram, as well as selective angiography of the superior mesenteric artery (SMA) and bilateral internal iliac arteries. If a type III endoleak is suspected, angiography performed with a pigtail catheter placed within the endograft

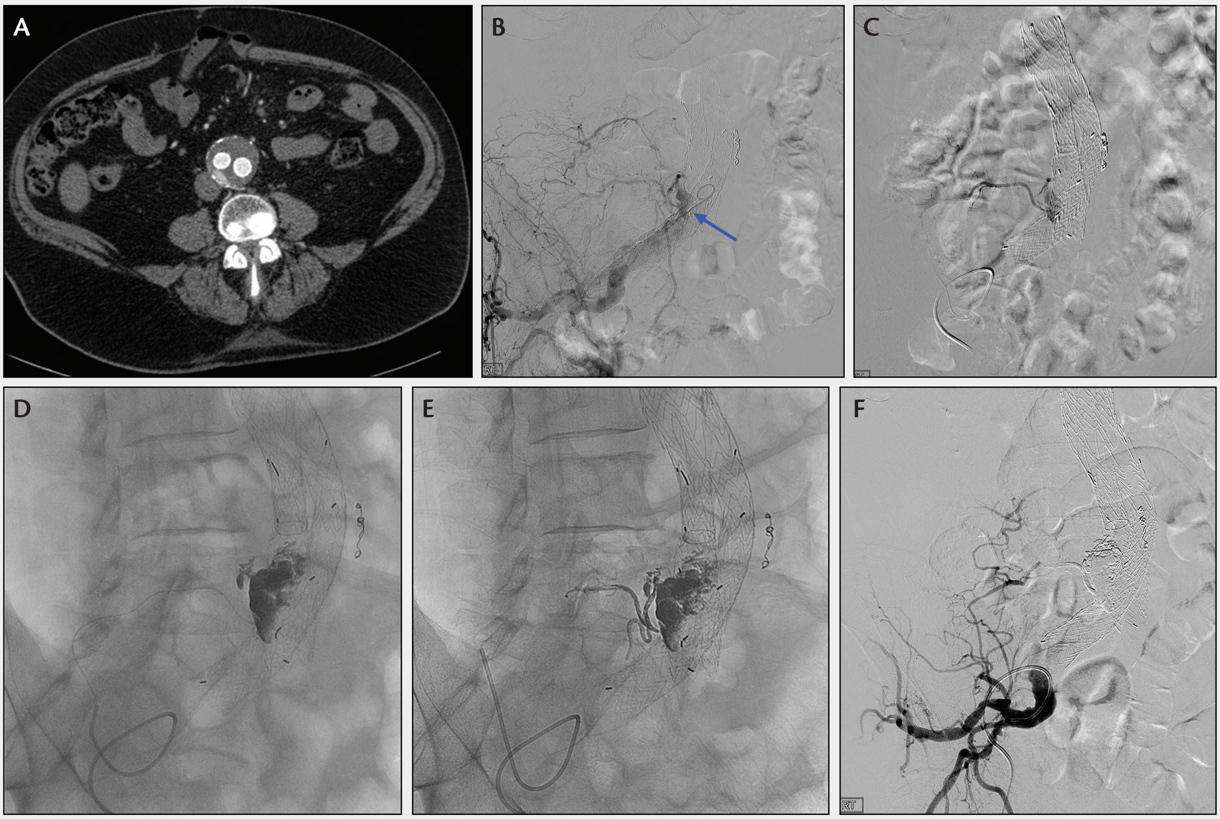


Figure 1. A patient with a type II endoleak via a right lumbar artery (A) and enlarging aortic sac. Angiogram showing a pigtail catheter in the right limb of a bifurcated endograft demonstrates an endoleak being fed by a lumbar artery (arrow) (B). A 5-F Cobra 2 catheter was placed into the right hypogastric artery, and angiography via a microcatheter placed into the culprit lumbar artery through the iliolumbar collateral better delineates the endoleak (C). Onyx was administered through the microcatheter (D). Further embolization was carried out until there was complete filling of the endoleak sac, as well as the feeding artery, with embolic agent (E). Final angiogram after embolization shows no further endoleak (F).

may be useful. Another useful technique is placement of an occlusion balloon above the pigtail catheter in the graft to increase the sensitivity for type III endoleak assessment. The latter strategy is also useful in identifying a type Ib endoleak by deploying a compliant balloon in the iliac limb and performing power injection angiography at the distal seal site. If a significant amount of contrast or radiation dose has been utilized during these diagnostic procedures, it is not unreasonable to stage the therapy with the treatment procedure performed on a later date once the endoleak type and etiology are determined.

It is important to note that the presence of one type of endoleak does not exclude a second type of endoleak. Endoleaks are often complex, and successful treatment of one endoleak does not preclude later development of another. Therefore, longitudinal follow-up of these patients is mandatory. Furthermore, filling of the IMA or lumbar arteries on cross-sectional imaging does not always represent a type II endoleak; we have seen many patients who have a subtle type Ia endoleak with resultant antegrade flow in these vessels (inflow from the proximal attachment zone and outflow from the lumbar/IMA), which is best appreciated on aortography.

TREATMENT APPROACH

At Miami Cardiac and Vascular Institute, type II endoleaks are only treated if there is evidence of aneurysm growth (generally > 5 mm). There are multiple approaches to the management of these endoleaks, including transarterial, translumbar, transcaval, and surgical approaches.

Transarterial Embolization

The transarterial and translumbar approaches are most commonly used to address these endoleaks, and there are conflicting data on which technique is more effective. The transarterial approach is our preferred first-line therapy to manage these endoleaks. It is important to stress that persistent type II endoleaks associated with aneurysm enlargement typically behave like arteriovenous malformations, with multiple ingress and egress vessels. Treatment of the nidus, or endoleak sac, is critical for an effective and durable result. The challenge lies in the ability to advance a microcatheter in a retrograde manner from the SMA (via the arc of Riolan or marginal artery of Drummond) to the IMA or from the internal iliac artery (via the iliolumbar artery) to the culprit lumbar artery. It is essential to advance the microcatheter to the aneurysm sac; however, these collateral pathways can be long and extremely tortuous, which can be difficult and sometimes impossible to navigate. Complete obliteration of the endoleak nidus with elimination of all inflow and outflow vessels is required to prevent recurrence. Proximal embolization is not recommended, as the type II endoleak will recur by recruiting additional aortic branch vessels, possibly making the treatment even more complex.

Stable access in the SMA or internal iliac artery is the first important step in performing transarterial embolization. A 5-F Cobra catheter or reverse-curve catheter (ie, Sos, Simmons) are our initial “go-to” catheters. In the setting of significant tortuosity and an inability to achieve stable access with these catheters, steerable guiding sheaths such as the Destino (Oscor Inc.) or Morph (BioCardia, Inc.) catheter, or the Magellan robotic catheter (Hansen Medical, Inc.) can be valuable. A 150-cm-long microcatheter with a 0.021-inch inner diameter or smaller is recommended. We typically use a dimethylsulfoxide-compatible microcatheter such as Echelon or Rebar (Medtronic) to allow treatment with the Onyx liquid embolic system (Medtronic). Although the Progreat microcatheter (Terumo Interventional Systems) is not validated as dimethylsulfoxide compatible, we have not encountered any problem injecting Onyx through it. We typically use coils and/or Onyx to treat the majority of these endoleaks. One potential benefit of Onyx is the ability to advance beyond the site of delivery and disperse through the endoleak nidus/sac to fill the ingress and egress vessels (Figure 1). Because Onyx is radiopaque, it can be closely followed under fluoroscopic guidance, and injection may be stopped if there is inadvertent nontarget delivery of the embolic agent. The MVP microvascular plug (Medtronic) is a newer device that can be delivered via a microcatheter. An advantage of this device is the presence of polytetrafluoroethylene, which results in immediate flow occlusion with minimal artifact on posttreatment CTA. We typically do not use cyanoacrylate glue, but this is a reasonable embolic alternative. Onyx, glue, and coils can make subsequent endoleak evaluation difficult due to significant streak artifact on CT imaging. In these situations, one can consider magnetic resonance angiography to look for a persistent leak where the artifact from these embolic agents can be minimized. It is possible that new liquid embolic agents may allow better results in terms of imaging.

Translumbar Embolization

If transarterial embolization is either not feasible or unsuccessful in resolving the endoleak, direct translumbar percutaneous access into the aneurysm sac can be performed. CTA is closely evaluated to determine an optimal site for entry such that the access needle will target the endoleak cavity. Our technique involves placing the patient in the prone position and placing a sheath needle into the aneurysm sac using either anatomic landmarks (ie, vertebral body, radiopaque markers on the endograft, calcification) or XperGuide software (Philips Healthcare) with cone beam CT. Another approach is to gain access into the aneurysm sac under CT guidance and subsequently move the patient to a fluoroscopy suite. Care is taken to avoid puncturing the endograft, as this may result in a type III endoleak. We tend to favor a left translumbar approach, if possible, to avoid the course of the inferior vena cava (IVC). However, some endoleaks may necessitate a right-sided transcaval approach. Incidentally, intravasated embolic material into the IVC may result in pulmonary embolism.

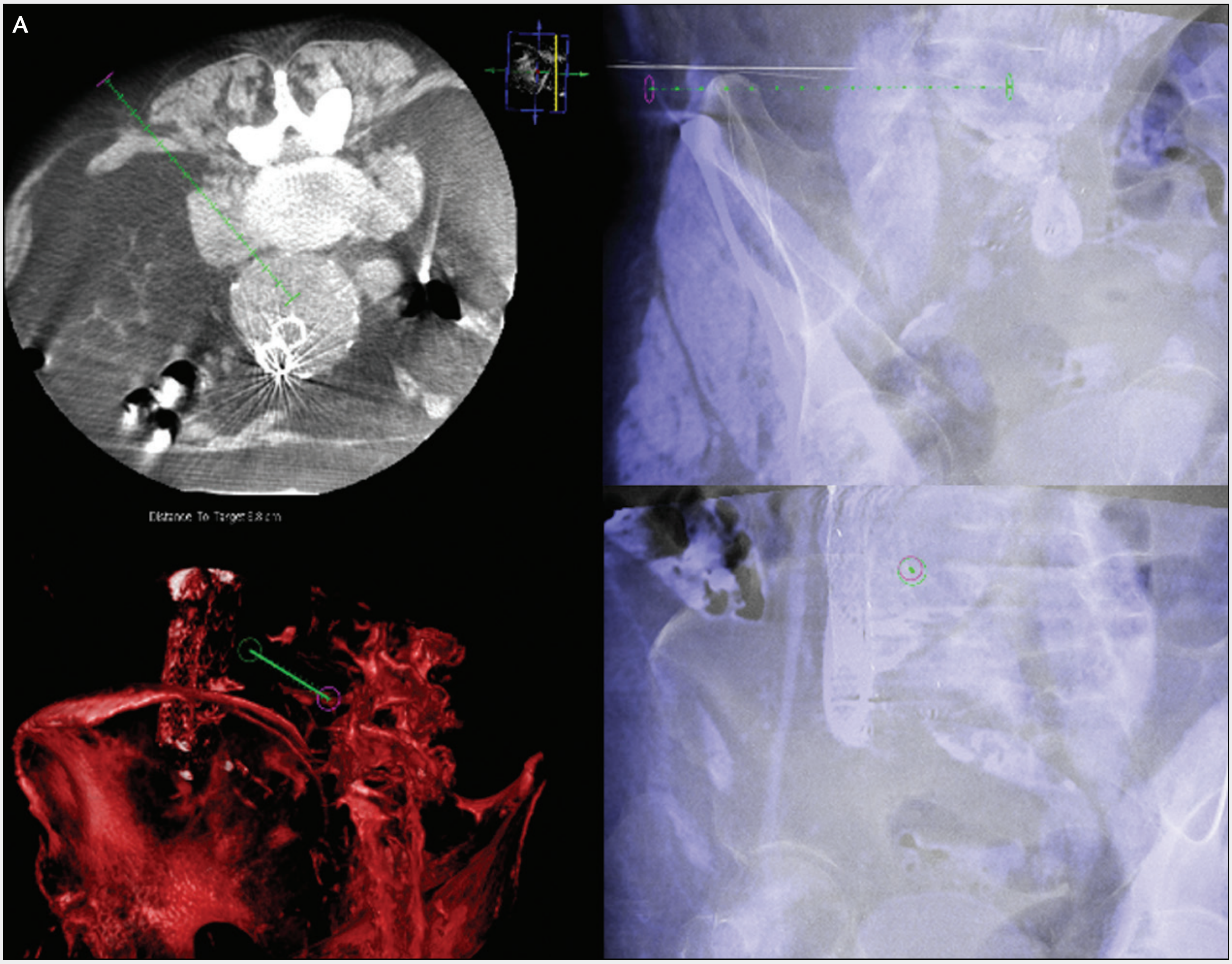
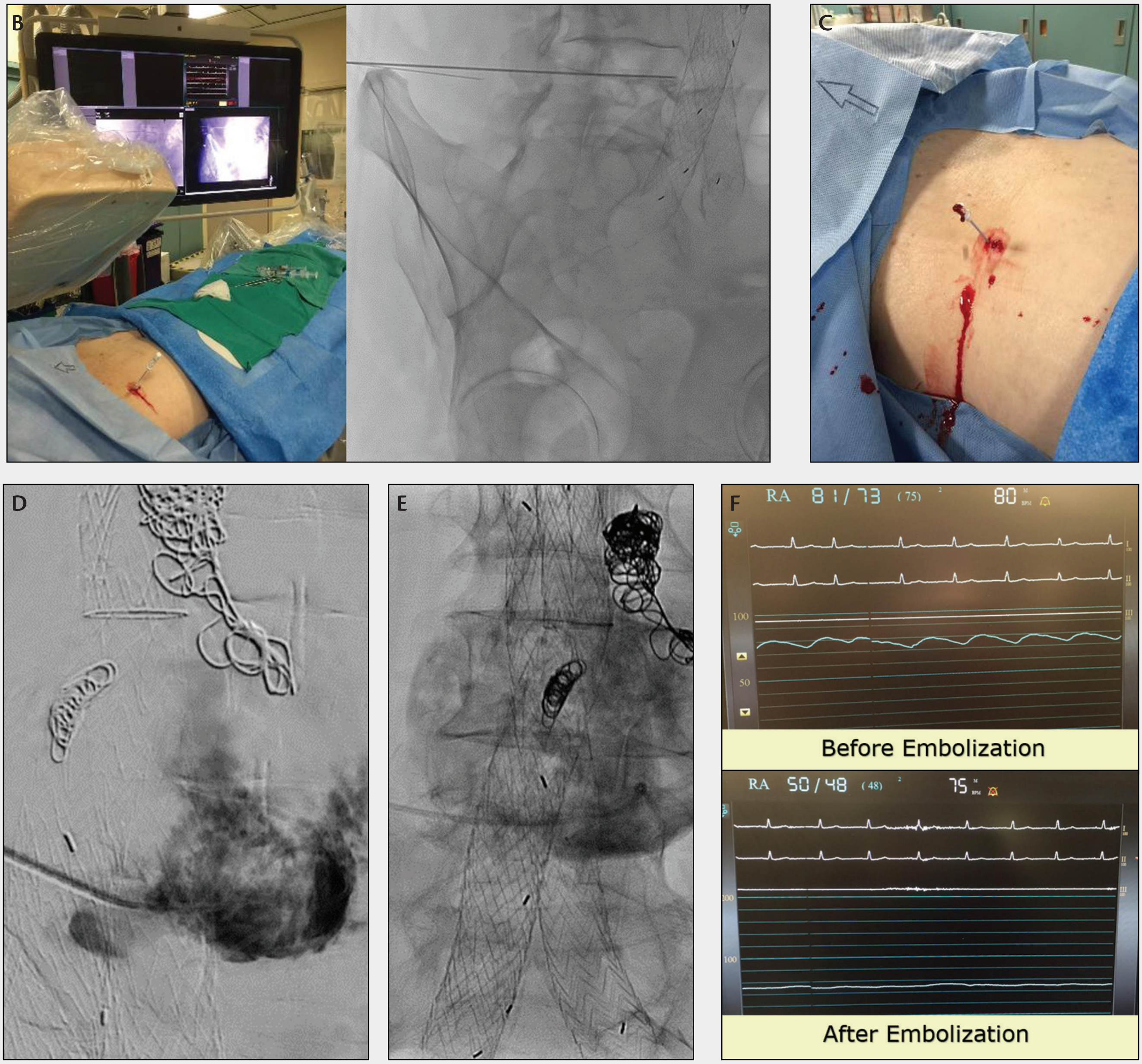


Figure 2. A patient with persistent type II endoleak despite previous embolization via a transarterial route. Translumbar direct sac puncture of the aortic sac is performed utilizing XperGuide software in conjunction with cone beam CT and fluoroscopic guidance (A). A 20-cm sheath needle is advanced into the endoleak sac until pulsatile arterial blood flow is noted (B, C). A contrast injection (saccogram) is performed through an access needle (D). Note that coils are from the previous embolization procedure. Because ingress and egress vessels are not easily visualized, thrombin was injected into the sac (E). Pressure measurement via the sheath needle demonstrates a decrease in pressure from 81/73 to 50/48 mm Hg after embolization, with loss of pulsatility (F).

Once the aneurysm sac is accessed and pulsatile blood is seen, baseline pressure measurements are obtained, which are subsequently compared to postembolization pressures. A diagnostic angiogram or "saccogram" is then obtained via the sheath needle to delineate the endoleak cavity and inflow and outflow vessels. A microcatheter is typically introduced coaxially to negotiate the nidus, and an attempt is made to catheterize every inflow and outflow vessel. If the latter vessels can be catheterized, coil embolization is performed. The endoleak sac is finally embolized with coils, Onyx, and/or thrombin. If it is not technically possible to select all of the vessels contributing to the endoleak, Onyx is administered under real-time fluoroscopy until there is embolization of the sac as well as the vessels. Thrombin injection into the sac is another effective alternative, but the inability to visualize and control the thrombin injection makes it less ideal. The benefit of thrombin is the absence of artifact on cross-sectional imaging, which allows for better detection of residual endoleak, but it might lead to a higher rate of recanalization. A final intrasac pressure measurement is obtained after embolization (Figure 2).

Results using this technique have been published, and the authors found it to be a useful bailout technique for persistent endoleaks after EVAR; however, endoleak recurrence was relatively high (50%) at a median follow-up of 39 months, and reintervention was deemed necessary in 33% of cases. Repeat endoleaks following embolization were associated with the use of dual-antiplatelet therapy.[15]



Transcatheter Transcaval Approach

The transcatheter transcaval approach to the aneurysm sac is uncommonly used, but it does have some utility, especially in the setting of a posterior endoleak in close proximity to the IVC, which is otherwise difficult to access. This technique is an alternative to the translumbar or transcaval approach. A prerequisite for utilizing this approach is CT evidence of close proximity or adhesion of the caval wall to the aneurysm sac. Furthermore, there must be sufficient space between the wall of the aneurysm abutting the IVC and the endograft to allow for entry into the endoleak cavity without puncturing the graft. The common femoral vein or internal jugular vein may be utilized for access. A 10-F, 40-cm reinforced sheath, identical to that used for transjugular intrahepatic portosystemic shunt procedures, is then placed. Using a combination of landmarks from preprocedural imaging, fluoroscopic imaging, and/or intravascular ultrasound, the sheath is wedged against the posterior caval wall. The presence of aortic wall calcification may limit intravascular ultrasound evaluation; however, it can serve as an important fluoroscopic target. A small amount of contrast is injected to confirm that the sheath is against the caval wall. The endoleak is then accessed with a Colapinto needle (Cook Medical), and a 5-F cannula with catheter is advanced into the endoleak sac. Once arterial blood flow is seen, intrasac pressure measurements are obtained, followed by contrast administration (sacogram). Embolization is then performed similarly to translumbar embolization. A cavagram is obtained at the conclusion of embolization.

Potential complications related to this technique include pulmonary embolism from nontarget delivery of embolic material, retroperitoneal hemorrhage, and aortocaval fistula. If there is adhesion between the caval wall and aorta, retroperitoneal hemorrhage is unlikely to occur. According to a study published by Mansueto et al, these complications were not encountered.[16]

Surgical Treatment

Laparoscopic, robotic, and open surgical ligation of mesenteric, lumbar, and other offending arteries are options for patients in whom the endovascular approach fails, with persistent endoleak with aneurysm growth. Other surgical options include plication of the aneurysm and graft explantation.

RESULTS OF PREVENTION OF TYPE II ENDOLEAK

In an attempt to prevent subsequent type II endoleaks, one institution employed a protocol of embolizing all IMAs if they were successfully visualized and accessed prior to EVAR. They noted decreased rates in type II endoleak incidence, aneurysm sac enlargement, and reintervention rates at 24 months.[17] Another institution performed intraprocedural abdominal aortic aneurysm sac embolization, which was shown to result in freedom from type II endoleak at 6 months and type II endoleak–related reinterventions, but without demonstration of differences in aneurysm sac size.[18] Given the time constraints, lack of robust data, and the fact that the majority of type II endoleaks regress, aggressive preventive techniques have not been practical or adopted at our institution and are not routinely employed. That being said, we occasionally preemptively embolize the IMA or lumbar arteries if they are unusually large and/or if there is an "empty sac" with minimal luminal thrombus. Newer technology, such as the Nellix endograft (Endologix, Inc.), which fills the aneurysm sac with a polymer-filled endobag, may decrease the incidence of type II endoleaks and reintervention rates.

CONCLUSION

In summary, type II endoleaks after EVAR are a common finding but are often of no clinical significance. However, in the setting of a persistent endoleak with concomitant aneurysm growth, secondary interventions are indicated. Further investigation is required to determine the most effective approach and optimal embolic agent(s) to manage this complication. ■

1. Malas M, Arhuidese I, Qazi U, et al. Perioperative mortality following repair of abdominal aortic aneurysms: application of a randomized clinical trial to real-world practice using a validated nationwide data set. JAMA Surg. 2014;149:1260-1265.
2. Jackson R, Chang D, Freischlag J. Comparison of long-term survival after open vs endovascular repair of intact abdominal aortic aneurysm among Medicare beneficiaries. JAMA. 2012; 307:1621-1628.
3. Speicher P, Barbas A, Mureebe L. Open versus endovascular repair of ruptured abdominal aortic aneurysms. Ann Vasc Surg. 2014;28:1239.
4. Schermerhor M, Buck D, O'Malley J, et al. Long-term outcomes of abdominal aortic aneurysm in the medicare population. N Engl J Med. 2015;373:328-338.
5. Dangas G, O'Conner D, Firwana B, et al. Open versus endovascular stent graft repair of abdominal aortic aneurysms: a meta-analysis of randomized trials. JACC Cardiovasc Interv. 2012;5:1071-1080.
6. Hellinger J. Endovascular repair of thoracic and abdominal aortic aneurysms: pre- and postprocedural imaging. Tech Vasc Interv Radiol. 2005;8:2-15.
7. Veith F, Barum R, Ohki T, et al. Nature and significance of endoleaks and endotension; summary expressed at an international conference. J Vasc Surg. 2002;35:1029-1035.
8. Tolia A, Landis R, Lamparello P, et al. Type II endoleaks after endovascular repair of abdominal aortic aneurysms: natural history. Radiology. 2005;235:683-686.
9. Lee K, Forbes T. Current understanding of the significance and treatment of type II endoleaks. Ital J Vasc Endovasc Surg. 2012;19:191-197.
10. Jouhannet C, Alsac J, Julia P, et al. Reinterventions for type 2 endoleaks with enlargement of the aneurismal sac after endovascular treatment of abdominal aortic aneurysms. Ann Vasc Surg. 2014;28:192-200.
11. Sarac T, Gibbons C, Vargas L, et al. Long-term follow-up of type II endoleak embolization reveals the need for close surveillance. J Vasc Surg. 2012;55:33-40.
12. Jones J, Atkins M, Brewster D, et al. Persistent type 2 endoleak after endovascular repair of abdominal aortic aneurysm is associated with adverse late outcomes. J Vasc Surg. 2007;46:1-8.
13. Otsu M, Ishizaka T, Watanabe M, et al. Analysis of anatomical risk factors for persistent type II endoleaks following endovascular abdominal aortic aneurysm repair using CT angiography. Surg Today. 2016;46:48-55.
14. Löwenthal D, Herzog L, Rogits B, et al. Identification of predictive CT angiographic factors in the development of high risk type 2 endoleaks after endovascular aneurysm repair in patients with infrarenal aortic aneurysms. Rofo. 2015;187:49-55.
15. Uthoff H, Katzen BT, Gandhi R, et al. Direct percutaneous sac injection for postoperative endoleak after endovascular aortic aneurysm repair. J Vasc Surg. 2012;56:965-972.
16. Mansueto G, Cenzi D, Scuro A, et al. Treatment of type II endoleak with a transcatheter transcaval approach: results at 1-year follow-up. J Vasc Surg. 2007;45:1120-1127.
17. Ward T, Cohen S, Fischman A, et al. Preoperative inferior mesenteric artery embolization before endovascular aneurysm repair: decreased incidence of type II endoleak and aneurysm sac enlargement with 24-month follow-up. J Vasc Interv Radiol. 2013;24:49-55.
18. Mascoli C, Freyrie A, Gargiulo M, et al. Selective intra-procedural AAA sac embolization during EVAR reduces the rate of type II endoleak [published online ahead of print February 6, 2016]. Eur J Vasc Endovasc Surg.

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