Endoleak and the Role of Embolization

Classifications and diagnosis of aneurysm endoleak and the techniques and technologies available to treat them.

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Endoleak is defined as a persistent blood flow outside the lumen of an endoluminal graft but within the aneurysm sac or adjacent vascular segment being treated by the device used for endovascular aneurysm repair (EVAR).

Endoleaks are caused by incomplete sealing or exclusion of the aneurysm sac. The inflow or reflux of blood flow into the sac causes continued pressurization of the aneurysm and may leave the patient at risk for rupture. Preventing rupture by excluding the aneurysm sac is the main goal of stent graft treatment. Despite the improvements in stent graft technology in the last decade, endoleaks remain a potential problem after endovascular thoracic and abdominal aortic aneurysm (AAA) repair. Different types of endoleaks require different treatment strategies, which are discussed in this article.

TABLE 1. TYPES OF ENDOLEAK

Type	Definition
Type I	Persistent filling of the aneurysm sac due to incomplete seal or ineffective seal at the proximal (type IA) or distal (type IB) end of the stent graft
Type II	Persistent filling of the aneurysm sac due to retrograde branch flow from collateral vessels
Type III	Blood flow into the aneurysm sac due to inadequate or ineffective sealing of overlapping graft joints or rupture of the graft fabric
Type IV	Blood flow into the aneurysm sac due to the porosity of the graft fabric, causing blood to pass through from the graft and into the aneurysm sac
Type V	Aneurysm sac expansion without clear evidence of endoleak origin

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Embolization techniques play an important role in the treatment of type II endoleaks by occlusion of the inflow and outflow branches as well as the nonthrombosed part of the sac. In type I endoleaks, embolization is also used whenever other techniques to seal the proximal or distal end of the stent graft have failed.

CLASSIFICATION

There are five types of endoleaks (Table 1), which are classified according to the source of blood flow into the endoleak. The type of endoleak will determine the patient's treatment and follow-up protocol.[1-3] Type I endoleak usually occurs in the early course of treatment, but may also occur later. A type I endoleak is associated with pressurization of the aneurysm sac with systemic pressure, progressive growth risk, and rupture risk; therefore, it should always be treated.

Type II endoleak may be thought of as an analogy to an arteriovenous malformation in which two or more patent vessels allow blood inflow and outflow within a channel or space created within the aneurysm sac. Examples of inflow vessels are the inferior mesenteric artery (IMA) and the lumbar artery (LA). Type II endoleaks may be classified as transient (spontaneous resolution within 6 months) or persistent (residual endoleak after 6 months of observation), and 60% of these will resolve within 1 month after stent graft placement. According to the EUROSTAR study, which involved 2,463 patients, only 5% to 6% of type II endoleaks induce sac enlargement, and the rupture rate can be as low as 0.52% (1/191 type II endoleaks). Long-term clinical results in patients with type II endoleaks are not significantly different than patients without endoleak.[4]

Marchiori et al studied the potential predictive factors for the development of type II endoleaks. Out of a group of 195 patients with type II endoleak, all patients had four patent LAs (mean diameter, 2.3 mm). At least one LA > 2 mm in diameter was a positive predictive factor for the development of persistent type II endoleak ($P < .001$). Larger-diameter LAs tend to be associated with persistent type II endoleaks, whereas LAs < 2 mm would more likely be seen with a transient type II endoleak.[5]

A type III endoleak usually occurs early after treatment due to technical problems or later due to device component disconnection or material fatigue. Type IV endoleak was relatively common with the first generation of stent grafts. Due to the improvement of the stent graft fabrics (Dacron, polytetrafluorethylene, or polyester) and suture lines, this type of endoleak is practically nonexistent today. Type V endoleak, also known as *endotension*, is a challenge and a diagnosis of exclusion. It is defined as continued enlargement of the aneurysm sac without evidence of a leak site. It is the result of a transudate due to ultrafiltration of blood by the graft membrane or unidentified leak.

DIAGNOSIS OF ENDOLEAK

Differing from patients undergoing open surgical aortobifemoral bypass, all patients with AAAs who are treated with stent graft placement need to be followed by some method of imaging. CT angiography (CTA) is probably the most adopted method worldwide. The most common follow-up protocol after EVAR includes a CTA during the first 30 days, 6 months, and 12 months after the procedure. Subsequently, the patient can be followed annually with CTA, noncontrast CT, or a combination of ultrasound (US) and abdominal radiography. In contrast CT exams, the endoleak is defined as the presence of contrast material inside the aneurysmal sac. It is important to acquire at least noncontrast and contrast delayed images. Comparative analysis of the two phases will help in differentiating aneurysmal wall and sac calcifications versus the presence of contrast related to endoleak. A type I endoleak will show the presence of contrast around the proximal (type IA) or distal (type IB) ends of the stent graft, with or without deep extension into the aneurysmal sac. In the majority of the cases of type II endoleak, abdominal CTA is helpful to diagnose the presence and origin of the endoleak. The presence of contrast pool in the left anterior aspect of the AAA sac is more likely related to retrograde filling through the IMA. If the contrast is situated in the posterolateral aspect of the AAA sac, retrograde filling is more likely via the iliolumbar artery. Catheter-based angiography may help to diagnose and to indicate the origin of endoleak in difficult cases. Due to its invasiveness, it is more often reserved for treatment purposes. Angiography should include an aortogram and selective superior mesenteric artery and bilateral internal iliac artery arteriograms to define the origin of the endoleak.

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In type III endoleak, the contrast is typically found adjacent to the connection between the stent graft body and the limbs. Type IV endoleaks are now rare, and they were most frequently seen immediately after stent graft placement, showing as a blush on the completion angiogram. In type V endoleaks, there is an increase in the size of the AAA sac without evidence of a contrast pool within. This is a diagnosis of exclusion.

Contrast-enhanced US may be an alternative to CTA for follow-up after EVAR. Because US reduces exposure to the biologic hazards associated with lifelong annual CTAs, including cumulative radiation dose and nephrotoxic contrast agent load, contrast-enhanced US might be considered as a substitute for CTA in the surveillance of suitable patients after EVAR.[6,7] However, it is frequently considered an imaging method that should be limited to patients with a low body mass index, and it is operator-dependent. Abdominal radiography may be helpful to identify stent graft kinking/migration and modular component separation.[8] MRI is a viable alternative; however, it is more expensive, has a longer acquisition time, and the stent graft alloy, such as nitinol, must be MRI-compatible.[9]

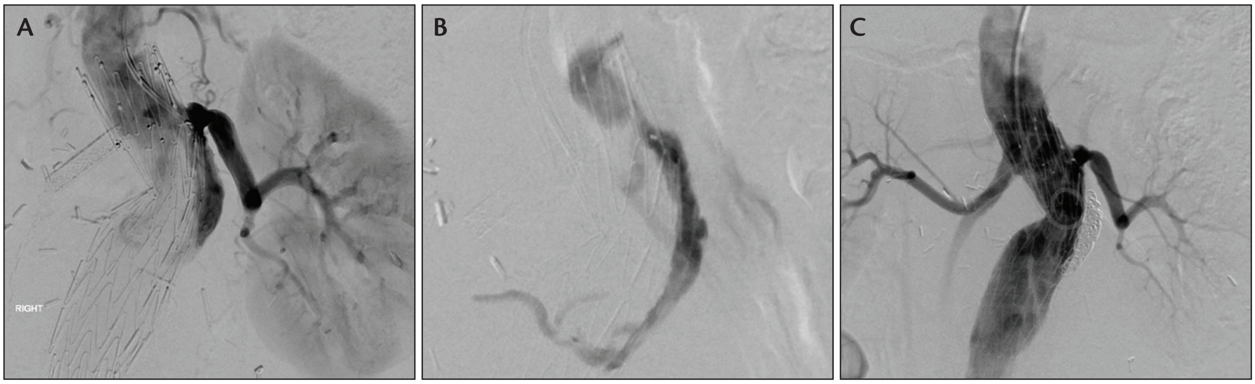

Figure 1. Type I endoleak in a patient with a short and angulated proximal neck due to lack of apposition of the proximal stent (A). Selective microcatheter angiogram showing endoleak anatomy (B). Arteriogram after coil embolization was used to fill the gap between the graft and the aortic neck wall (C).

ENDOLEAK MANAGEMENT

Prevention Through Proper Patient Selection

The best way to manage endoleaks is to prevent them. Proper patient selection is critical to reduce the risk of endoleaks. Due to the less-invasive nature of the endovascular techniques over open surgery and the improvement in stent graft technology, most centers are pushing the limits of the instructions for use of the devices and treating patients off-label with the consequent risk of endoleaks. With the development of more sophisticated techniques to address patients with short necks (eg, snorkel, chimney, and periscope), the treatment of complex AAA anatomy has become a reality. These techniques, however, are associated with a higher incidence of endoleaks. AAA sac expansion in the presence of endoleak warrants treatment. Most of the endoleaks can be managed with endovascular techniques. Open repair is typically reserved for rare cases of endovascular failure. Type I and III endoleaks are the most concerning and should preferably be treated at time of diagnosis. The direct arterial pressure transmitted to the AAA sac is thought to significantly increase the risk of rupture.

Device Options

Different types of tools should be promptly available. Type I endoleaks may respond to simple balloon angioplasty using a Coda balloon (Cook Medical), for example, which can be carefully inflated inside the proximal or distal edges of the stent graft. Insufflation outside of the stent graft should be avoided if at all possible. In cases of persistent type IA endoleak, treatment methods include the deployment of an aortic cuff, use of a balloon-mounted Palmaz stent (Cordis Corporation), microcatheter embolization of the endoleak track, and EndoStaples (Aptus Endosystems, Inc.). Aortic cuffs can be deployed by overlapping over the stent graft body in a way that avoids covering the renal artery ostium. A Palmaz stent may be a good choice in case there is poor apposition of the stent graft against the AAA neck wall and if there is a high risk associated with covering the renal artery origin(s) with an aortic cuff. This technique requires careful manipulation in order to crimp the stent adequately around the Coda balloon. The risk of stent dislodgement during delivery should be kept in mind. Alternatively, a fenestrated cuff or snorkel technique could be considered, which typically requires brachial access to deploy a covered stent in the renal artery(ies) parallel to the body of the stent graft.

Patients with persistent type I and III endoleaks after Coda balloon angioplasty and aortic cuff/Palmaz stent placement may benefit from superselective transarterial embolization. If there is a narrow track of type I endoleak around one side of the stent graft, a superselective microcatheter embolization technique may be helpful in selected cases (Figure 1). N-Butyl cyanoacrylate, or glue (Trufill, Cordis Corporation), dimethyl sulfoxide-ethylene vinyl alcohol (DMSO-EVOH) solution (Onyx, Medtronic), and coils (or a combination of coils and a liquid embolic agent) are the most popular embolic agents.

EndoStaples may also be considered for treating challenging short and angulated aneurysm necks in which there is inadequate contact between the stent graft and the aneurysm neck wall. This might reduce the high reintervention rates after EVAR in this subgroup of patients.[10,11]

Type II endoleaks are considered benign if the patient is asymptomatic and if there is no AAA sac expansion; however, small endoleaks may thrombose spontaneously. If the endoleak is still present after 6 months of observation, there is little chance that it will resolve.

Treatment is warranted in this case or at any point in time if there is evidence of sac enlargement. Similar to a vascular malformation, in the type II endoleak, the blood enters during systole into the endoleak cavity, swirling around and leaving the endoleak cavity during diastole.[12] Type II endoleak can be classified as simple (small cavity and has ingress and egress from a single vessel) or complex (multiple ingress and egress vessels).

Type II endoleaks may be treated by an endovascular or percutaneous approach. The endovascular approach may be quite challenging, as it requires the microcatheter tip placement inside the AAA sac. Typically, access is achieved via the superior mesenteric artery (with retrograde microcatheterization of the IMA through the marginal/arc of Riolan arteries) (Figure 2) or via an iliolumbar branch approach (via retrograde microcatheterization of a lumbar branch). Access may also be possible through a catheter placed in between the distal end of the graft and the vessel wall. After microcatheter sacography, the AAA sac is typically embolized, and thrombosis is generated using glue, thrombin, DMSO-EVOH solution, or coils. Again, similar to the principles of endovascular treatment of a vascular malformation, if the egress vessel is identified, proximal occlusion of this vessel should be attempted, as well as occlusion of the distal ingress artery(ies) as close to the opening to the AAA sac, if possible.

Transarterial embolization of the artery that feeds the endoleak cavity has proven ineffective, providing only short-term response if the sac cannot be reached by the embolic agent. Ultimately, the endoleak will recur by recruiting additional aortic branch vessels. In this situation, direct percutaneous aneurysmal sac access is a great alternative in association with or instead of the endovascular techniques in case the sac is not reachable via an endovascular approach. This usually requires the combination of CT and fluoroscopic guidance.

Initial abdominal CT with contrast is critical to identifying the location of the endoleak. Direct percutaneous translumbar access to the aneurysmal sac is achieved with an 18-gauge needle under CT guidance. Over a stiff 0.035-inch wire, the needle is exchanged for a 5-F short semicurved catheter (Kumpe, Cook Medical). After CT confirms that the catheter tip is in position, embolization of the aneurysmal sac is accomplished under fluoroscopic guidance. Sacography is commonly performed to define the endoleak anatomy, understand the size of the nonthrombosed sac, and identify potential egress vessels. After sacography, the aneurysmal sac is embolized or thrombosis is generated using glue, thrombin, DMSO-EVOH solution, or coils (Figure 3). Depending on the aneurysmal sac configuration and the correlation to adjacent organs, alternative accesses (eg, transcaval or via the stent graft) may be considered.

Type III endoleak is typically treated by overlapping a stent graft limb at the leakage site (body-limb junction or limb-extension junction). Type IV endoleak commonly does not require treatment, because in most

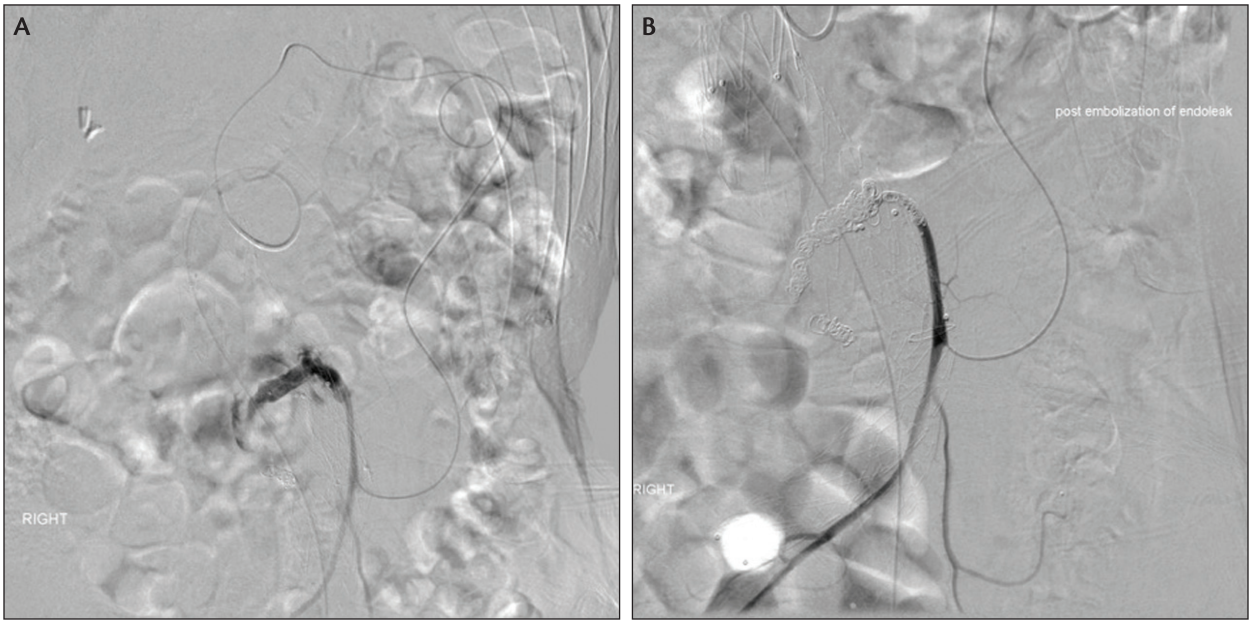

Figure 2. Type II endoleak from the IMA. A microcatheter was advanced inside the aneurysmal sac in a retrograde fashion through the arc of Riolan (A). Postembolization imaging with microcoils filling the aneurysmal sac and the origin of the IMA (B).

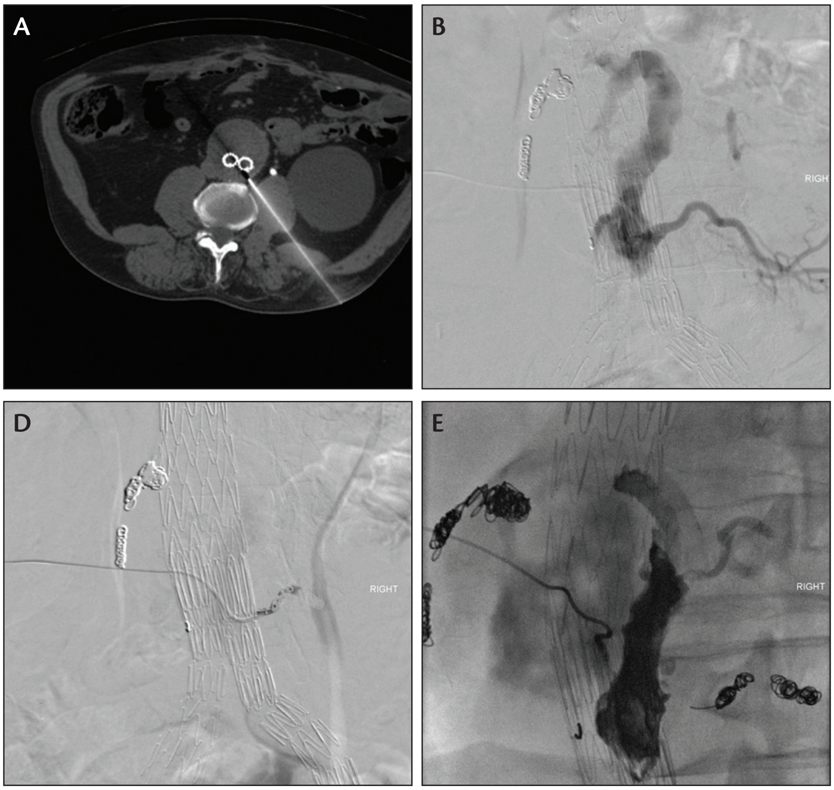

Figure 3. A patient with a type II endoleak and growing aneurysm. CT-guided translumbar access of the sac at the level of the endoleak (A). A 5-F Kumpe catheter was advanced over the wire, and sacography was performed to define the endoleak anatomy (B). Selective catheter placement inside a lumbar artery (C). The lumbar artery was embolized with microcoils (D). The rest of the aneurysm sac was embolized with Onyx, filling the sac and a lumbar artery in a retrograde fashion (E).

cases, there is autosealing of the stent graft porosity (more relevant with the first stent graft generation) after cessation of the intraprocedure heparin anticoagulant effect.

CONCLUSION

Aortic aneurysms treated with stent grafts require long-term imaging follow-up. The type of endoleak will guide the best way to manage it, but careful patient selection is still the best way to prevent it. The treatment of endoleaks using endovascular and percutaneous techniques, including embolization using different techniques to access the aneurysm sac, has helped to reduce aneurysmal sac rupture after endovascular repair. ■

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