Microleaks, Ultrafiltration, and Endotension: What to Do about Them

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Microleaks

Microleaks are endoleaks associated with persistent transgraft blood flow through holes in the fabric after endovascular aneurysm repair (EVAR). They occur in thin polyester endografts after 6 to 30 months and are classified as type III endoleaks. Microleaks occur at a discrete focus as opposed to type IV endoleaks owing to diffuse graft porosity. They are associated with aneurysm enlargement and rarely development of symptoms.

Diagnosis

MDR computed tomography (CT) with noncontrast, dynamic contrast, and delayed contrast is frequently used to monitor aneurysms after EVAR. CT often identifies endoleak, but the diagnosis of microleak requires specific understanding of the nature of microleaks and their radiographic appearance. Specifically, microleaks may appear as contrast enhancement closely associated with the surface of the thin polyester endograft that does not track towards a branch vessel or attachment site. The contrast-enhanced area is often very small with the dynamic imaging owing to low flow rates but has larger appearing volume on delayed imaging. Microleaks appear more frequent in areas of curvature and regions of the graft with more suture points.

Color flow duplex ultrasonography can identify microleaks as small jets of color flow directly adjacent to the endograft. These jets often occur at regular spaced intervals along the graft. Pulsed-wave Doppler analysis confirms that there is flow in the microleak and that the jet is not an artifact of graft or aneurysm pulsatile motion.

It is important to note that standard methods of digital subtraction aortography do not demonstrate microleaks, and special techniques are necessary. Microleaks may be imaged by injection in the proximal end of the iliac limb with longer injection rates (8 for 24) and imaging in multiple oblique projections. Small jets of contrast can be seen projecting perpendicular to the endograft, often at the tips of the diamond shapes where the graft had been sutured to the stent. Hand injection of contrast through the guidewire lumen with balloon occlusion of the distal endograft can demonstrate microleak without a doubt. Specific attention to detect microleaks is typically not part of most surveillance protocols and may result in under diagnosis of microleaks. These specific imaging techniques are not necessary for all patients but are useful for the subgroups of patients with endoleak of undetermined origin, unexplained aneurysm enlargement, and before reinterventional therapy is contemplated for endoleak. They can be used in conjunction with selective injections of the superior mesenteric artery and hypogastric arteries, and translumbar aortography. Direct observation during explantation of the endograft provides confirmation of focal blood flow through the wall of the endoprosthesis. Opening the aneurysm sac before applying the aortic crossclamp shows the "sprinkler" effect of microleaks. Often, the thrombus within the sac is described as loose, gelatinous, or fresh.

Mechanism

Etiology for holes in the fabric are vacated suture holes after suture breakage, chronic wear against metal stents or calcified plaque, and polyester fabric weave deformation. Broken sutures or fabric weave deformation may occur during manufacture, forceful deployment maneuvers, or balloon dilation of the endograft with an oversized angioplasty balloon. Microleaks may be more common with specific endografts, particularly those with many suture points to thin polyester graft material, but it is unlikely that microleak is unique to one type of endograft, and transgraft type III endoleaks and material fatigue issues have been identified with many endografts.

Some surgeons may assume that fabric holes the size of microleaks will spontaneously thrombose as they do following standard open surgical repair. Microleak patency after EVAR may be related to the biology of the intraaneurysmal thrombus that has cellular elements and thrombolytic environment.

Significance

The clinical significance of microleaks continues to be debated. Small diameter endoleaks lead to significant aneurysm sac pressure in experimental models, and our patients with microleak for more than a year after EVAR all had significant aneurysm enlargement. Clearly, microleak is a primary cause of abdominal aortic aneurysm (AAA) enlargement in a few patients. It may also be a permissive factor with other types of endoleaks or may be an incidental finding in other individual patients. Although identification of patients with microleak is important, it seems that micoleaks are relatively infrequent compared with caudal migration as a mechanism of aneurysm rupture after EVAR.

Treatment

Prevention is perhaps the best strategy for treating microleaks. Manufacturing improvements have been made to AneuRx endografts that have microleaks. Avoiding anatomic situations where forceful deployment (tortuous segments) maneuvers may be required is prudent. Endografts for aneurysm treatment are not the same as uncovered stents for stenotic lesions, and balloon angioplasty should only be performed when necessary and with the appropriately sized balloon and pressure. We have found that microleaks are less common with these preventive strategies.

When a patient is identified with microleak and sac enlargement, there are several options. Conversion in a reasonable-risk patient is sometimes performed. When the microleak is in the midportion of an iliac limb, then re-treatment with a second coaxial endograft is relatively simple. There is more challenge when the microleaks are in the short main trunk or flow divider region, and a customized approach can be attempted. We have observed aneurysm diameter reduction after endovascular re-treatment with a coaxial stent graft.

Ultrafiltration
Ultrafiltration has been demonstrated with different types of graft material and is well studied in the original Excluder endoprosthesis. It is associated with aneurysm enlargement and, rarely, clinical symptoms.

Diagnosis
Multiple imaging studies are performed to exclude endoleak. These may include MDR-CT with dynamic and delayed injections, contrast-enhanced or standard duplex ultrasound, and arteriography with selective injections of the superior mesenteric artery and hypogastric arteries. Translumbar aneurysm catheterization may reveal grayish, nonhemorrhagic fluid and a sac pressure that decreases with aspiration. When an endoleak is present, repressurization can occur over a few minutes, but with pure ultrafiltration, an interval of days may be needed to repressurize the sac after aspiration.
Direct observation during explantation of the endograft provides indirect confirmation of transgraft ultrafiltration with two findings: absence of endoleak and fibrinous outgrowths from the endograft. Typically, these growths are present where there is a single layer thickness and absent in the overlap zones. Gadolinium-enhanced magnetic resonance imaging can demonstrate in vivo ultrafiltration as a ring of enhancement around the endograft on delayed imaging, but this is an uncontrolled observation.

Mechanism
Ultrafiltration occurs through the porosity of the fabric when the natural hemostatic pathways do not thrombose the fabric. This seems to be particularly susceptible within an aneurysm sac compared to in the neck regions or extravascular locations where tissue ingrowth is more robust.

Significance
The clinical significance of ultrafiltration is debated. Clearly, it leads to significant aneurysm sac pressure and is a primary cause of AAA enlargement in many patients. The interaction of ultrafiltration with endoleaks is the subject of much speculation.

Treatment
Prevention is perhaps the best strategy for treating ultrafiltration. Manufacturing improvements have been made to Excluder endografts that have ultrafiltration. We have found that ultrafiltration is less common with these new graft materials. When a patient is identified with endotension after an older endograft, there are several options. Conversion in a reasonable-risk patient is sometimes performed. Re-treatment with a second coaxial endograft is possible, and aneurysm diameter reduction after relining has been measured. Observation is another option (see below).

Endotension: New Concepts
Aneurysm size change after endovascular repair has been considered an indicator of success since endografts were developed over a decade ago. This was a natural assumption based on established knowledge that sac size predicts rupture risk in untreated aneurysms. This assumption was the basis for management of many patients after endovascular repair, including conversion for patients with sac enlargement, even though this can be a hazardous procedure. Most physicians agree that those patients who continue to have enlargement with endoleak, particularly type I endoleaks, are at risk of a fatal rupture. Nearly all patients with rupture after endovascular repair have had endoleak at the time of rupture. However, there is less consensus about the management of patients with enlargement without identified endoleak—endotension. Some regard endotension as evidence of endoleak, even if it is not identified on imaging studies. There was also concern for neck instability as sacs enlarge. These assumptions and past algorithms of management with open conversion for all enlarging aneurysms are being challenged.
The mechanisms of endotension could include graft material defects such as microleak, inadequate seal zone that results in sac thrombus in direct contact with the aortic flow channel, undiagnosed endoleak, and ultrafiltration. Transgraft ultrafiltration leading to nonhemorrhagic rupture has been described after open repair by Thoo. Risberg and colleagues and van Sambeek and colleagues have described treatment of endotension with laparoscopic and open sac fenestration, and they have confirmed that iatrogenic rupture of the sac does not lead to massive hemorrhage. There are scattered case reports of coaxial relining as another alternative treatment for endotension, but it remains unknown if this will durably reduce aneurysm sac size or be clinically safer than open conversion or observation.
Recently, Mennander and colleagues reported a key observation—radiographic evidence and direct observation of spontaneous nonhemorrhagic rupture. This occurred in multiple patients after endovascular repair with three different devices, including polyester and polytetrafluoroethylene grafts. In two cases, the aneurysm rupture without fatal hemorrhage was followed by aneurysm sac shrinkage. Mennander also noted no correlation between increasing sac size and decreasing proximal neck length or iliac fixation. Based on these findings, they propose that non-operative approach is indicated for patients with endotension. Before embarking on observation, Mennander and colleagues emphasize the importance of assessing the seal zone and excluding endoleak by multiple techniques. This requires knowledge of the failure modes, imaging options and specific nuances of sac enlargement associated with each device.

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References

1. Adolph R, Vorp DA, Steed DL, et al. Cellular content and permeability of intraluminal thrombus in abdominal aortic aneurysm. J Vasc Surg 1997;25:916–26.
2. Bernhard VM, Mitchel RS, Matsumura JS, et al. Ruptured abdominal aortic aneurysm after endovascular repair. J Vasc Surg 2002;35:1155–62.
3. Bohm T, Soldner J, Rott A, Kaiser WA. Perigraft leak of an aortic stent graft due to material fatigue. AJM Am J Roentgenol 1999;172:1355–7.
4. Matsumura JS, Ryu RK, Ouriel K. Identification and implications of transgraft microleaks after endovascular repair of aortic aneurysms. J Vasc Surg 2001;34:190–7.
5. May J, White GH, Stephen MS, Harris JP. Rupture of abdominal aortic aneurysm: concurrent comparison of outcome of those occurring after endovascular repair versus those occurring without previous treatment in an 11-year single-center experience. J Vasc Surg 2004;40:860–6.
6. May J, White GH, Yu W, et al. Conversion from endoluminal to open repair of abdominal aortic aneurysms: a hazardous procedure. Eur J Vasc Endovasc Surg 1997;14:4–11.
7. Mennander A, Pimenoff G, Heikkinen M, et al. Nonoperative approach to endotension. J Vasc Surg 2005;42:194–9.
8. Ouriel K. Abdominal aortic aneurysm. Images in medicine. N Engl J Med 2002;346:1467.
9. Riepe G, Heilberger P, Umscheid T, et al. Frame dis location of body middle rings in endovascular stent tube grafts. Eur J Vasc Endovasc Surg 1999;17:1355–7.
10. Risberg B, Delle M, Eriksson E, et al. Aneurysm sac hygroma: a cause of endotension. J Endovasc Ther 2001;8:447–53.
11. Risberg B, Delle M, Lonn I, et al. Management of aneurysm sac hygroma. J Endovasc Ther 2004;11:191–5.
12. Schurink GW, Aarts NJ, Van Baalen JM, et al. Experimental study of the influence of endoleak size on pressure in the aneurysm sac and the consequences of thrombosis. Br J Surg 2000;87:71–8.
13. Thoo CHC, Bourke BM, May J. Symptomatic sac enlargement and rupture due to seroma after open abdominal aortic aneurysm repair with polytetrafluoroethylene graft: Implications for endovascular repair and endotension. J Vasc Surg 2004;40:1089–94.
14. van Sambeek MRHM, Hendriks JM, Tseng L, et al. Sac enlargement without endoleak: when and how to convert and technical considerations. Semin Vasc Surg 2004;17:284–7.
15. White GH, Yu W, May J, et al. Endoleak as a complication of endoluminal grafting of abdominal aortic aneurysms: classification, incidence, diagnosis and management. J Endovasc Surg 1997:4:152–68.
16. Zarins CK, White RA, Fogarty TJ. Aneurysm rupture after endovascular repair using the AneuRx stent graft. J Vasc Surg 2000;31:960–7.

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