ASK THE EXPERTS

Does Treating Type II Endoleak and the Aneurysm Sac Make a Difference?

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Nearly 30 years after the implantation of the first aortic endograft, the natural history and hemodynamic and clinical significance of type II endoleaks remain controversial. There is continued debate regarding the indications for treatment and surveillance, technical approach to treatment, and efficacy of treatment on long-term outcomes after endovascular aneurysm repair (EVAR).[1,2] There are even anecdotal reports that type II endoleaks may simply represent outflow channels for subtle type I and type III endoleaks not detected by current imaging modalities. A variety of different techniques for intervention have been reported, including transarterial, transcaval, and/or translumbar embolization using various embolic agents such as coils, plugs, and polymerizing agents; surgical or laparoscopic ligation; sac imbrication; and endograft explantation.[3-6] Technical success rates and aneurysm-related outcomes after intervention remain widely variable.[7] However, the coexistence and concomitant treatment of other types of endoleaks continue to prevent meaningful conclusions from being drawn regarding the significance of successful treatment of an isolated type II endoleak.

The variable and unpredictable sac behavior pattern observed after technically successful type II endoleak embolization suggests that among patients with aneurysm sac growth in the setting of an isolated type II endoleak, obliteration of the endoleak nidus can potentially alter sac behavior much in the same way that nidus embolization of a high-flow arteriovenous malformation slows its growth but is not curative in the long term. As such, type II endoleaks may simply represent the nidus of a more intricate endoleak flow pattern—one that may involve subtle seal zone leaks not detected by current imaging techniques. Therefore, aggressive treatment of these type II endoleaks may merely represent a temporizing measure until a different endoleak flow pattern manifests. Given that these endoleaks occur in thrombus-ridden aneurysm sacs with unpredictable flow channels, dynamic, high-resolution, real-time, four-dimensional imaging may be the only means of detecting these endoleaks that may pressurize the sac spontaneously and intermittently.[1]

CT or duplex scans may only be providing a single view of a much more intricate, dynamic process. The inability of currently employed surveillance imaging modalities to adequately identify subtle endoleaks is the concept behind the type V endoleak (eg, endotension without identifiable endoleak). Detection of contrast within the inferior mesenteric artery (IMA) and/or lumbar branches during surveillance scans may represent outflow conduits for subtle type I or type III endoleak with origins out of phase with the CTA cuts and, therefore, may be mislabeled as a type II endoleak. This may help explain why persistent aneurysm sac growth has occasionally been encountered despite successful

elimination of a type II endoleak nidus.[3,8,9] Interestingly, both metabolic syndrome and systemic inflammatory disease have been associated with the presence of type II endoleak, as well as sac expansion, suggesting a complex metabolic environment within an aneurysm sac after EVAR.[10,11]

As with detection of slow gastrointestinal bleeding, nuclear medicine using sulfur colloid or tagged red blood cell scans may play a more prominent role in the future to enhance imaging sensitivity to detect subtle endoleaks.[1] Blood pooling techniques in MRI may also prove useful in enhancing imaging sensitivity, but this technology is not yet readily available.[12] Given the growing awareness of flow directionality and spectral waveform analysis in the accurate diagnosis of endoleaks, duplex ultrasound is an increasingly attractive option in evaluation of persistent type II endoleaks.[13] A to-and-fro spectral waveform is more consistent with a focal, isolated type II endoleak as opposed to a mono- or biphasic signal that suggests the presence of an inflow and outflow source.[14] Manning and colleagues have reported excellent sensitivity of duplex ultrasound in detecting endoleaks that went on to require intervention; however, more subtle leaks were frequently missed.[13]

In addition, although an isolated type II endoleak may not be hemodynamically significant enough to cause rupture, it may alter sac morphology over time such that seal zones are compromised and rapid expansion and/or rupture become real possibilities.[15] These type II endoleaks may represent cases in which prompt intervention can be of clinical benefit in preventing or delaying late type I endoleaks and may help explain why a yet-to-be-stratified subgroup of patients benefit from successful type II endoleak intervention. This warrants further experience, longer-term follow-up, and improved imaging technology. For now, close surveillance of all type II endoleaks is warranted, with emphasis on evaluation of sac morphology and further vigilance to detect subtle or delayed formation of type I and/or type III endoleaks.

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Type II endoleak has been the subject of scientific discussion since EVAR was introduced more than 25 years ago. Still, there is no consensus on the threshold for treatment and controversy surrounding both the optimal methods for diagnosis and treatment. This is quite remarkable given that type II endoleak treatment is the most commonly performed secondary intervention after EVAR.

There are a few well-accepted facts about type II endoleak: most resolve spontaneously; visualization is strongly dependent on imaging modality, operator, timing of contrast, and contrast used; the difference between a type II endoleak in a shrinking, stable, or growing abdominal aortic aneurysm (AAA) sac is unknown; and as EVAR for rupture works well, with almost all patients having immediate type II endoleak with no intact sac to tamponade continuous bleeding, the importance of type II endoleak generally appears very limited. The importance of type II endoleak was further clarified by a systematic review in which the chance of rupture due to type II endoleak was only 0.04% in all 21,744 patients.[1] These results were based on circumstantial evidence at best, and about half of the ruptures occurred in the absence of sac expansion. An interesting question would be: Has it ever been proven that treating type II endoleak does more good than harm to our patients?

We recently conducted a systematic review on the treatment results of persistent type II endoleak. The results were quite shocking—most of the studies were of very low scientific quality, with no consensus on treatment indications, follow-up length was usually minimal, and there was no clear definition of treatment success. When reasonable 2-year treatment success is considered, the firm belief of many interventionalists, as well as the recommendations of official guidelines, to treat type II endoleak in the presence of AAA growth seem to be based on only 27 patients

worldwide! Furthermore, the complications of type II endoleak treatment, including death, greatly exceed the risk of rupture due to type II endoleak. Therefore, one should probably see type II endoleak as "sentinel endoleak" in that it is a guide to a potential problem, but treating it does not cure the patient.

Although type II endoleaks appear to be innocent, it would be ideal to be able to prevent them from occurring, as it would quell the discussions and avoid many secondary interventions. This can be done by active sac management. Preemptive coiling has been shown to make no difference in outcome, but polymer sac filling, with or without the use of endobags (ie, Nellix, Endologix; Arsenal AAA sac-filling technology), has the potential to stabilize the treated aneurysm and thereby potentially reduce surveillance programs. This technology may be the next step in reducing (unnecessary) secondary interventions and improving EVAR results.

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Last November at the VEITHsymposium, I was asked to debate Prof. Verhagen in a session titled, "Type II Endoleaks With AAA Sac Enlargement Can Be Dangerous and Should Be Treated." I argued in support, and he was against this claim. Ultimately, he won the debate because his thorough literature review demonstrated that there is indeed no level 1 evidence to support treating type II endoleaks. However, the proper management of type II endoleaks in everyday practice remains an unanswered question.

We define a growing aneurysm as a diameter increase of > 5 mm during follow-up after EVAR. Most type II endoleaks with aneurysm growth are associated with type I or III endoleaks. It is critical to rule out additional endoleak with careful review of the proximal and distal landing zones and overlap between the graft components on the CT images. Before bringing a patient back to the angiography suite, we recommend performing a contrast-enhanced ultrasound. This allows better classification of endoleaks and has the advantage of providing hemodynamic information on blood flow direction and comparison in real time and on the same screen, as well as at baseline and with and without contrast. In addition to diagnosing an isolated type II endoleak, defining the inflow and outflow vessels perfusing the endoleak is useful in preprocedural planning of embolization.

Once type I and III endoleaks have been excluded, we schedule endovascular repair of the type II endoleak. The technique will be driven by the vessels involved in the type II endoleak. If the IMA is involved, we favor a transarterial approach from the groin. Selective catheterization of the superior mesenteric artery is performed with a 5-F catheter. A microcatheter is advanced all the way to the IMA and most importantly to the nidus of the endoleak. Selective injection of nitroglycerin is mandatory to avoid spasm and false passage of a 0.018-inch guidewire. Because iliolumbar vessels are almost always involved, the microcatheter is pushed to the posterior wall of the aneurysm. We often start the procedure by releasing long microcoils into the nidus of the endoleak to reduce blood flow and provide better control when delivering the liquid embolization agent. Our practice uses Onyx liquid embolic agent (Medtronic). Its release is well visualized and controlled. The goal is to completely fill the nidus and the origin of all involved inflow and outflow vessels. It is mandatory to change the position of the gantry throughout the procedure to ensure that the liquid agent is not migrating into the iliolumbar vessels or the IMA, which can have disastrous outcomes.

If the IMA is not involved, or in the setting of recurrent type II endoleak following IMA embolization, transarterial iliolumbar access through the internal iliacs is an option. We must emphasize that this approach is time-consuming, increases the radiation dose, and is often very challenging. An alternative transarterial route, which can be used in case of IMA embolization, is transsealing access. A stiff 5-F catheter is advanced through a long stiff 6-F

sheath to the distal sealing zone of an iliac limb. A wire is advanced between the arterial wall and the iliac limb and back into the aneurysm sac.

Another option for iliolumbar endoleaks that allows easy nidus access is the translumbar approach. Latest-generation hybrid rooms are equipped with a wide range of new imaging applications, such as fusion imaging (to reduce the risks associated with radiation exposure and iodine contrast medium use) or cone-beam CT (CBCT). CBCT consists of rotational acquisition of a two-dimensional image data set that can be reconstructed similarly to multiplanar CT images on a workstation. Direct puncture of the aneurysm sac is aided by the registration of the preoperative CTA with an intraoperative CBCT, performed with the patient in a supine position. Different views are then used to follow the needle progression from the skin entry point (puncture site, bullseye view) to the target (endoleak, trajectory view).

In appropriate anatomy, a transcaval approach performed under fusion guidance can facilitate access to an endoleak. On the fusion mask, we include the puncture site of the inferior vena cava wall and the endoleak nidus. This three-dimensional guidance helps to accurately position a transseptal needle prior to advancing it into the nidus.

We know from clinical experience that isolated type II endoleaks can be associated with aneurysm growth. We have observed aneurysm shrinking following embolization and impressive backflow from sac branches after opening aneurysm sacs without aortic clamping. Although rare, type II endoleaks can be managed in a variety of ways, as previously described. The key to successful management is access to the sac (whatever the selected route) and complete filling and embolization of the endoleak nidus and origins of all involved sac branches.

Another much more frequent condition is persistent sac flow without significant growth. Although this does not require treatment, both the patient and physician can be dissatisfied with this result. It is not clear whether a local persistent inflammatory process has any effect. However, prevention of type II endoleak may be the best approach. In our practice, we routinely embolize the IMA or any iliolumbar arteries ≥ 4 mm during the index EVAR procedure (mostly with plugs), but at present, there are no available data to support the benefit of this approach.

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Patients who undergo EVAR seek a low-risk treatment with quick return to normal activities, minimal follow-up, no secondary intervention, and knowledge that their aneurysm is "fixed for good." Ideally, EVAR should not be associated with any endoleak, including type II endoleak.

The understanding and management of type II endoleak has evolved since the days of early EVAR but continues to be controversial. Although most agree that type II endoleaks have a relatively benign and indolent course in most patients (sometimes for years), a large body of evidence shows that type II endoleaks are associated with sac enlargement, secondary interventions, added cost, and, in some patients, rupture. At a minimum, closer surveillance is indicated in patients with sac enlargement, which generates cost, radiation exposure, and significant anxiety. From the perspective of the patients, knowledge that their aneurysm is not excluded or "fixed" and that the endoleak is resulting in enlargement is at least troublesome. From the perspective of the physician, it is difficult, if not impossible, to convince patients and their families that the treatment was actually effective in the presence of an enlarging aneurysm.

Type II endoleak is the Achilles heel of EVAR. It is one of the major undermining factors when we compare EVAR to open surgical repair. The true incidence has been underestimated in many studies, including some pivotal trials. Type II endoleaks are not likely device dependent (aside from sac exclusion techniques) and likely occur in > 40% of EVAR patients. Although a conservative approach is recommended and treatment is reserved for those with significant sac enlargement (> 5 mm), results are disappointing in many patients. The cost of embolization material is high, and treatment is not often effective, resulting in persistent type II endoleak in as many as 70% of patients. Therefore, it is no surprise that type II endoleaks are also a common indication for open surgical explantations.

The future of EVAR should include strategies that will minimize surveillance, cost, and secondary interventions, and this will require significant reduction in endoleak rates, including type II endoleaks. ■