Type II endoleaks

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Type II endoleaks after endovascular aneurysm repair are the most common type of endoleak and generate the majority of secondary interventions. Their natural history is mostly benign, but they can occasionally lead to sac expansion and eventual rupture. Three-phase computed tomography angiography is the “gold standard” for diagnosis, but duplex ultrasound with or without contrast enhancement and magnetic resonance angiography offer an alternative for endoleak detection or surveillance. Whereas there are concerns as to whether sac expansion can be a dependable marker for risk of rupture, it is currently the best surrogate available and guides the indication for intervention. Obliteration of type II endoleaks can be challenging, and a variety of techniques, endovascular, open, and laparoscopic, have been proposed. The most common approaches are transarterial and translumbar embolization, and they are usually successful, provided the operator is experienced and persistent, targeting both the branches and the nidus of the endoleak. Recurrences and subsequent reinterventions should be anticipated, and on continuing sac expansion, repeated endovascular or open surgical and laparoscopic alternatives may be required. (J Vasc Surg 2014;60:1386-91.)

Endovascular aneurysm repair (EVAR) is currently widely accepted as the standard of care for infrarenal abdominal aortic aneurysm repair for patients with suitable anatomy.1-5 The need for serial surveillance and frequent reinterventions predominantly related to the anticipation or treatment of endoleaks accounts for its weaknesses and continues to limit its cost-effectiveness. Whereas there is little controversy in the management of type I and type III endoleaks, type II endoleaks have generated conflicting reports about their natural history, detection, and follow-up and the optimal timing and type of management. This review summarizes the accumulated evidence on type II endoleaks with particular focus on contemporary detection strategies, treatment criteria, and technical options (Table).

INCIDENCE AND NATURAL HISTORY

Type II endoleaks are “procedure related” as a result of patent lumbar arteries or inferior mesenteric artery branches and may involve various flow patterns.6 They represent the most frequent type of endoleak, and the incidence has been correlated, although not uniformly, with the number and size of patent branches before exclusion.7-10 Earlier studies showed possible endograft dependence, but this does not appear to be the case in later studies with longer follow-up.11,12 The largest published series comes from the EUROSTAR (European Collaborators on Stent/graf Technique for Aortic Aneurysm Repair) registry (3595 patients), reporting a 9% rate of type II endoleak diagnosed any time during follow-up,10 similar to the 8% to 10% rates reported in large systematic reviews and meta-analyses.5,12,13 The rate is generally higher at 1 to 6 months and occurs in around 15% of patients, and although some type II endoleaks may be identified at a later date, the prevalence gradually decreases to <10% at 2 years.8,13 Their natural history is currently considered relatively benign, and evidence suggests that approximately 60% of the immediate postoperative type II endoleaks will resolve spontaneously within 6 months, whereas the majority of the remaining will continue to regress over the years.9,14 Factors that have been linked to spontaneous endoleak resolution are cancer, coronary and peripheral artery disease, chronic obstructive pulmonary disease, and smoking; long-term anticoagulation and dual antiplatelet therapies may cause them to persist.10,14-16 The aneurysm sac may otherwise shrink, despite the presence of a patent type II endoleak (Fig 1). Commonly, however, the sac will remain stable, or in about 5.5% to 24%, it will demonstrate enlargement of >5 mm. The rate of sac enlargement gets even higher, up to 55%, for endoleaks that persist for >6 months.10,14,17,18

Whereas there is no clear evidence that sac expansion in this setting is a surrogate for future aneurysm rupture, aneurysm rupture does occur, although rarely, in patients with type II endoleaks.4,19 Data from the EUROSTAR registry on 2463 patients suggested a cumulative 2-year incidence of rupture after type II endoleak of 1.8% (one of 55 patients)10, however, this rate was no different in patients without any detected endoleak (0.9%; five of 548

EVIDENCE SUMMARY

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In the most recent meta-analysis of Sidloff et al (21,744 EVARs, 1515 type II endoleaks), the rupture rate of all type II endoleaks reviewed was 0.9%; 43% of these ruptures had no sac expansion.12

DETECTION AND FOLLOW-UP

The standard post-EVAR imaging protocols to detect endoleaks or endograft migration rely on computed tomography angiography (CTA), given its availability and easy standardization.21 For CTA to properly detect type II endoleaks, three runs are required, one with and one without contrast material and a third delayed run during the venous phase. Duplex ultrasound (DUS) with or without contrast enhancement and magnetic resonance angiography have been suggested as alternative imaging modalities.21,22 Wireless pressure sensors implanted during

### Table. Summary of evidence on type II endoleaks

<table>
<thead>
<tr>
<th>Incidence</th>
<th>Natural history</th>
<th>Detection</th>
<th>Decision to treat</th>
<th>Treatment</th>
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<tbody>
<tr>
<td>8%-10% overall5,10,12,13</td>
<td>Predominantly benign</td>
<td>Three-phase CTA is the “gold standard.”</td>
<td>Lack of robust evidence1,2,12</td>
<td>Endovascular techniques are preferred as minimally invasive.</td>
</tr>
<tr>
<td>15% at 1-6 months after EVAR8,14</td>
<td>60% resolve within 6 months9,14</td>
<td>Contrast-enhanced ultrasound and magnetic resonance angiography may have accuracy equivalent to if not better than that of CTA. Yet, they are not widely available, and diagnosis is highly operator dependent.21,22</td>
<td>Intervention is warranted only on sac enlargement &gt;5 mm but not for endoleak persistence.1,2</td>
<td>Percutaneous transarterial embolization is the most common intervention. Average success rate is 63% (range, 15%-89%).12</td>
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<td>&lt;10% at 2 years after EVAR8,13</td>
<td>5.5%-24% will promote sac enlargement10,14,17,18</td>
<td>CTA, computed tomography angiography; DUS, duplex ultrasound; EVAR, endovascular aneurysm repair.</td>
<td>Translumbar embolization is a reasonable alternative, particularly when there is no transarterial access. Average success rate is 81% (range, 67%-100%).12</td>
<td>Endovascular techniques are preferred as minimally invasive.</td>
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<td>Not device related in current practice8,11</td>
<td>For those persisting &gt;6 months, higher rates of sac enlargement (up to 55%) should be anticipated.10,14,17,18</td>
<td>Evidence on ventral or transcaval sac puncture is insufficient16,27</td>
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<td>~1% risk of rupture10,12,19,20</td>
<td>Embolization should target the nidus and the feeder branches of the endoleak.12,26,28</td>
<td>Operator should be persistent and cautious to potentially uncover a type I or type III endoleak.19,29,30</td>
<td>Microcoils are mainly used, but glue, thrombin, and Onyx can also be delivered, with no proven superiority of one agent over the other.</td>
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<td>No evidence that sac enlargement is related to rupture4,12,19,20</td>
<td>Embolization should target the nidus and the feeder branches of the endoleak.12,26,28</td>
<td>If the endoleak persists and the sac continues to grow after “technically” successful embolizations or when embolization is impossible, more invasive approaches are required.</td>
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<td>No evidence that sac enlargement is related to rupture4,12,19,20</td>
<td>DUS is the most cost-effective follow-up test and can be used for monitoring aneurysm sac growth. Type II endoleaks can be differentiated, but diagnosis is highly operator dependent.6,22,24-26</td>
<td>If the endoleak persists and the sac continues to grow after “technically” successful embolizations or when embolization is impossible, more invasive approaches are required.</td>
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Fig 1. After endovascular aneurysm repair (EVAR), the abdominal aortic aneurysm can shrink despite the presence of a type II endoleak, which may otherwise show late spontaneous resolution. a, Type II endoleak at year 1. b, Persistent type II endoleak at year 2 with sac shrinkage. c, No endoleak and aneurysm continues to shrink at year 3.
EVAR have shown some value in sac pressure monitoring, but sufficient data are still lacking.23 DUS is not only cheaper and safer than other modalities but may actually be more sensitive in the diagnosis of endoleaks, although the latter assertion remains controversial.6,22,24-26 Nonetheless, detection of type II endoleaks can be challenging, and not infrequently they can be missed (low flow) or may overlap with other types of endoleaks and cannot be differentiated by any imaging modality. DUS has the specific advantage of detecting flow direction of endoleaks, facilitating type identification, and Doppler waveforms may even predict the natural history of a type II endoleak.6 Contrast-enhanced ultrasound may have even better accuracy, particularly in the detection and classification of endoleak.22 Unfortunately, accuracy is highly operator dependent, and technical difficulties in patients with large body habitus still limit its universal application. DUS can be an excellent test for monitoring the aneurysm sac growth and, in combination with endoleak evaluation, provides sufficient data to identify complications requiring intervention.

Post-EVAR surveillance recommendations have undergone significant updates. Original practice guidelines included a postoperative 30-day CTA study, repeated at 6 months, 1 year, and annually thereafter. There is increasing evidence to decrease the imaging frequency, to eliminate the 6-month follow-up study, and to substitute CTA beyond or even at 1 year with DUS.1,2,22,24-26 Whereas there is uniform agreement to generally treat type I or type III endoleaks on detection, the follow-up protocol remains ill-defined when a type II endoleak is diagnosed. Although current guidelines suggest CTA at 6 months on type II endoleak detection at the postoperative CTA study,1,2 accumulating evidence suggests omission of this follow-up visit and repeated imaging at 12 months with either CTA or DUS (combined with radiographs) or non-contrast-enhanced computed tomography to check for sac growth with subsequent annual DUS, provided the sac does not expand.24-26 Such DUS follow-up protocols should be implemented only in accredited vascular laboratories with experienced technologists and internal quality controls.

**MANAGEMENT**

**Decision to treat a type II endoleak.** On the basis of their benign natural history and very rare association with rupture (~1%), the decision to treat a type II endoleak has changed steadily over the years, gradually favoring a more conservative approach. Criteria for intervention vary across the literature; the most common have been a persistent type II endoleak or an associated sac expansion >5 mm.1,2 Whereas there are concerns as to whether sac expansion can be a surrogate marker for risk of rupture, this is currently the best guide available; thus, intervention is warranted only on sac enlargement but not for endoleak persistence alone.1,2 A treatment algorithm is summarized in Fig 2.

**Treatment of a type II endoleak.** Obliteration of type II endoleaks can be difficult. The principle of treatment is
to eliminate the branches at their junction with the aneurysm. A variety of endovascular, open, and laparoscopic techniques have been proposed to abolish side branch perfusion. Whereas all alternatives may have a role, endovascular techniques are preferred, given their minimally invasive nature.

Percutaneous transarterial embolization is the most common intervention for type II endoleaks. Targeting to embolize the nidus and the feeder branches of the endoleak, transfemoral or transbrachial access is obtained and a coaxial system with microcatheters is created to reach the inferior mesenteric or lumbar arteries; proximal lumbar arteries may be difficult to reach. The approach is generally feasible through the internal iliac (superior gluteal) or superior mesenteric (middle colic and marginal) arteries, depending on the target and the respective collateralization (Fig 3). Microcoils are mainly used, but glue, thrombin, and Onyx (ethylene vinyl–alcohol copolymer) can also be delivered, with no proven superiority of one agent over the other.

Translumbar embolization is a reasonable alternative, particularly when there is no transarterial access (occluded internal iliac or inferior mesenteric arteries, prior transarterial embolization) left. Given the rarity of the technique, not all vascular surgeons are familiar, so some technical details merit attention, despite several variations. The endoleak is identified on prior computed tomography scans and referenced to fluoroscopic landmarks (eg, localization of the endoleak to specific vertebral bodies or radiopaque markers on the stent graft). With the patient in prone position, access is obtained under fluoroscopy, typically from a left paraspinal approach (~4 fingerbreadths from midline). Over a wire, a 6F 30-cm sheath is introduced, and following a sacogram, the system is positioned directly within the endoleak, which is signaled with brisk blood return. Subsequently, similar to the transarterial approach, a coaxial system with microcatheters is created to reach the nidus and the target arteries (Fig 4). Some authors have also suggested ventral or transcaval sac puncture, but data are still insufficient.

In the most recent systematic review, the success rate, defined as no recurrence during follow-up, averages 62.5% (range, 15%-89%) for the transarterial approach (120 reviewed interventions; average follow-up, 4-25 months) and 81% (range, 67%-100%) for the translumbar (57 interventions; average follow-up, 3-22 months). All studies that compare transarterial with translumbar approaches show

![Fig 3. Transarterial embolization. a, A 6F sheath has been placed in the internal iliac artery, and a 5F selective catheter is used to image the endoleak. b, A microcatheter is guided over a microwire (coaxial system) toward the lumbar artery origin. c, Microcoils are used, and there is no residual endoleak. d, Six months later, the endoleak persists and the abdominal aortic aneurysm is expanding; more feeders are identified (arrow). e, The microcatheter is again used to reach the abdominal aortic aneurysm sac. f, Nidus and branches are coiled and endoleak resolves.](image-url)
superior results for the translumbar method; however, in the majority of cases, translumbar was a second-line treatment, after the transarterial had failed; thus, such comparisons may not be valid. A more important aspect of success, irrespective of approach taken, is gaining access and embolizing the sac (nidus) and as many feeders (inflow and outflow) as possible, a task requiring advanced endovascular skills. It should otherwise be expected that recurrences and subsequent reinterventions are going to be frequent, the sac may continue to grow, and even occult type I or type III endoleaks may be revealed up to 5 years after EVAR; thus, the operator needs to be cautious and persistent. Should the endoleak persist and the sac continue to grow after a “technically” successful embolization or when embolization is impossible, more invasive approaches are required. Laparotomy or laparoscopy with ligation of the feeding side branches, suturing of the side branch ostia within the aneurysmal sac but leaving the stent graft intact, and finally conversion to open repair are all feasible alternatives that carry higher complexity, morbidity, and mortality.

CONCLUSIONS

The natural history of type II endoleaks has been recognized as predominantly benign. DUS can detect endoleaks, but three-phase CTA remains the “gold standard” for accurate diagnosis and eventual treatment planning on sac expansion. The most common approaches are transarterial and translumbar embolization, and they are usually successful but often require persistence and advanced endovascular skills. Access and embolization of both the flow channel and the nidus are essential for a successful outcome.

AUTHOR CONTRIBUTIONS

Conception and design: MM
Analysis and interpretation: EA, RC
Data collection: EA

Fig 4. Translumbar embolization. a, With the patient in the prone position, access has been obtained through the left paraspinal approach, and a 6F 30-cm sheath has been introduced into the abdominal aortic aneurysm sac. b, A sacogram is obtained that reveals the endoleak. c, A 5F catheter is used to guide embolization. d, The nidus is embolized with multiple coils.
Writing the article: EA
Critical revision of the article: MM, RC
Final approval of the article: MM
Statistical analysis: Not applicable
Obtained funding: Not applicable
Overall responsibility: MM

REFERENCES