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Management of Endoleaks After Elective Infrarenal Aortic Endovascular Aneurysm Repair
A Review

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IMPORTANCE Endovascular aneurysm repair (EVAR) is the dominant treatment strategy for abdominal aortic aneurysms, encompassing 80% of all repairs in the United States. Endoleaks are ubiquitous and affect 30% of patients treated by EVAR, potentially leading to sac enlargement and increased risk of rupture. The care of EVAR patients requires long-term surveillance by a multidisciplinary team. Accordingly, physicians should be familiar with the fundamentals of endoleak management to achieve optimal outcomes, including timely referral for remediation or providing counseling and reassurance when needed.

OBSERVATIONS PubMed and the Cochrane database were searched for articles published between January 2002 and December 2022 in English, addressing epidemiology, diagnosis, and management of endoleaks after EVAR. Endoleaks can be detected intraoperatively or years later, making lifelong surveillance mandatory. Type I and III have the highest risk of rupture (7.5% at 2 years and 8.9% at 1 year, respectively) and should be treated when identified. Intervention should be considered for other types of endoleak when associated with aneurysm sac growth larger than 5 mm based on current guidelines. Type II endoleaks are the most common, accounting for 50% of all endoleaks. Up to 90% of type II endoleaks resolve spontaneously or are not associated with sac enlargement, requiring only observation. Although the risk of rupture is less than 1%, cases that require reintervention are challenging. Recurrence is common despite endovascular treatment, and rupture can occur without evidence of sac growth. Type IV endoleaks and endotension are uncommon, are typically benign, and primarily should be observed.

CONCLUSIONS AND RELEVANCE Endoleak management depends on the type and presence of sac expansion. Type I and III endoleaks require intervention. Type II endoleaks should be observed and treated selectively in patients with significant sac expansion. Since endoleaks can appear any time after EVAR, at least 1 contrast-enhanced computed tomographic angiogram or duplex ultrasound by an experienced laboratory is recommended every 5 years.

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Supplemental content

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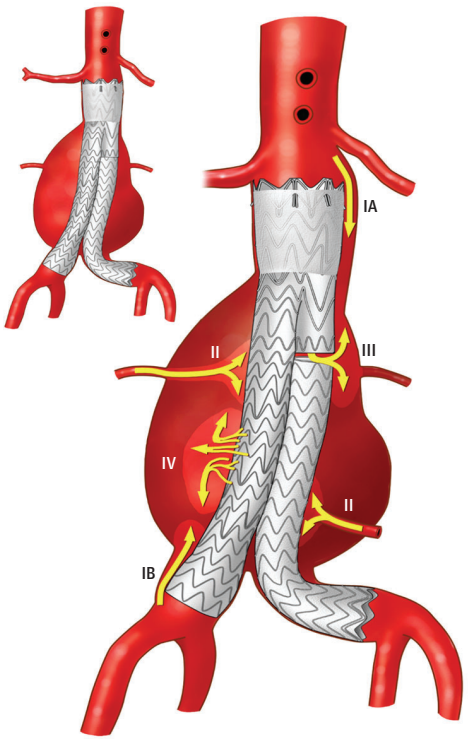
Abdominal aortic aneurysms (AAAs), defined as an aortic diameter larger than 3 cm, affect approximately 1.4% of adults aged 50 to 84 years in the United States.[1,2] Risk factors strongly associated with AAA development include tobacco use, male sex, age older than 65 years, and family history.[3] Several clinical trials have supported elective repair when the aneurysm diameter exceeds 5.5 cm in men and 5.0 cm in women.[4,5] The advent and subsequent adoption of endovascular aortic aneurysm repair (EVAR)[6] over the past 25 years have largely supplanted open treatment in most patients. EVAR is currently the dominant treatment modality used in more than 80% of AAA procedures in the United States.[7,8] This paradigm shift has primarily been due to the consistent results of EVAR, lower 30-day morbidity and mortality rates, faster recovery, and shorter hospital stays compared with open repair in multiple randomized clinical trials (RCTs).[9-11]

Despite these advantages, EVAR does not involve aneurysm resection, and sac perfusion remains a potential repair failure mode. Endoleaks are defined as continued sac perfusion despite endograft deployment and can be detected intraoperatively during EVAR and months or years later during follow-up. Thus, lifelong imaging surveillance is mandatory.[2] Endoleaks are the most frequent indication for secondary intervention after EVAR. Although half of cases resolve spontaneously, 30% of endoleaks require reintervention.[12,13] Endoleaks are classified into 4 types according to the cause of perigraft flow (**Figure 1**). Type II is the most common, accounting for 50% of all subtypes. In certain scenarios, endoleaks can eventually lead to aneurysm rupture.[14]

In addition to vascular surgeons, the longitudinal care of patients with AAA involves a multidisciplinary team. These may include primary care physicians, emergency physicians, advanced practice clinicians, diagnostic radiologists, nonvascular surgeons,

Figure 1. Endoleaks Etiological Classification



Type IA, inadequate proximal endograft seal. Type IB, inadequate distal endograft seal. Type II, persistent flow from collateral arteries (inferior mesenteric artery, lumbar). Type III, endograft integrity loss or incomplete attachment between endograft components. Type IV, endograft material porosity. Used with permission of Mayo Foundation for Medical Education and Research.

and other interventionalists (eg, cardiology, radiology). Physicians will likely encounter endoleaks during follow-up or as an incidental finding on abdominal ultrasound or computed tomography angiography (CTA). Endoleaks are a complex topic, and physicians who are not well versed in post-EVAR management may not be familiar with current treatment strategies. Therefore, physicians involved in the care of patients with AAAs must be knowledgeable about this topic to facilitate patient education and initiate appropriate and timely referral. This review aims to describe the types, etiology, pathophysiology, remedial indications, and updated diagnostic and therapeutic alternatives for the management of EVAR-associated endoleaks.

Methods

We conducted a literature search in PubMed and Cochrane databases using the following MeSH terms: "aortic aneurysm, abdominal," "endovascular aneurysm repair," and "endoleak." Other entry terms included "surgery," "complications," and "prevention and control." Search strategy details are available in the eAppendix in the Supplement. Articles were selected based on the following eligibility criteria: date of publication from January 2002 to December 2022, and type of publication (societal management guidelines, RCTs, observational studies, systematic reviews, and meta-analyses). Further assessment was done based on the quality of the evidence and information of interest to a general medical readership. We also considered additional relevant literature from the references of selected articles. The SANRA scale[15] was used to guide the writing process.

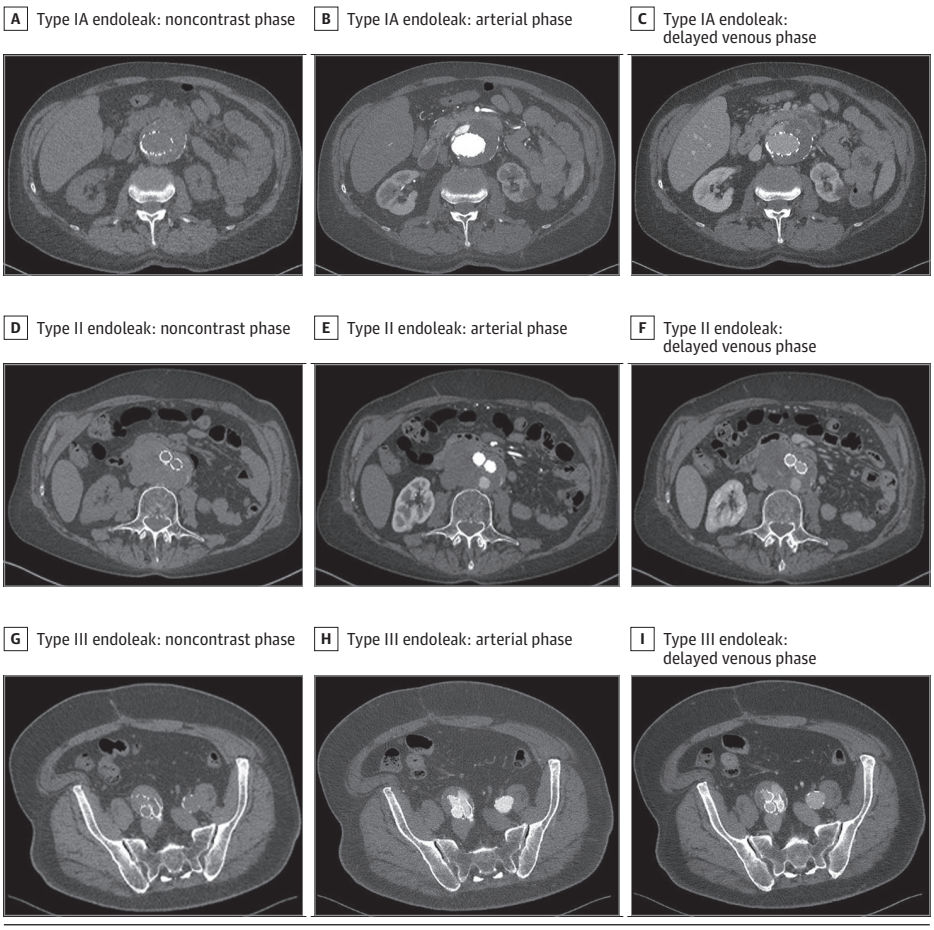
Type I Endoleaks

A type I endoleak occurs when there is persistent blood flow through the attachment zone between the endograft and the artery. Type IA is more common and originates from an incomplete proximal aortic seal. Type IB arises from an incomplete distal iliac seal. Type I endoleaks account for 12% of all endoleaks,[12] with an incidence of approximately 8%.[16-18] They can be identified at the time of repair or during follow-up. Late type I endoleaks are typically detected from 34 to 52 months post-EVAR. Notably, type I endoleaks are associated with higher rupture risk (4%-7.5% at 2 years) and late conversion to open repair.[14,17,19,20] Risk factors for type I endoleaks are primarily related to the anatomy of the aorta at the time of repair, including a short, angulated, or reverse tapered aneurysmal neck, large AAA diameter, mural neck thrombus, or calcification.[21,22] Further, large infrarenal neck diameters (≥30 mm) require 34- to 36-mm endografts and are increasingly recognized to be a risk factor for type IA endoleaks and stent migration.[23] Additionally, short or ectatic common iliac arteries and short distal sealing lengths increase the risk for type IB endoleaks.[24]

Intraoperative angiography during EVAR can detect a type I endoleak. Similarly, during a postoperative imaging surveillance protocol, CTA may identify type I endoleaks and provide the most thorough overall repair assessment.[25] Computed tomography angiography is the preferred method for early postoperative evaluation. Triple-phase imaging (noncontrast, arterial, and delayed venous phase) should be used to rule out endoleaks (**Figure 2**). A baseline CTA 30 days after EVAR is recommended by the 2018 management guidelines from the Society for Vascular Surgery (SVS; Level 1, Quality B) and 2019 guidelines from the European Society for Vascular Surgery (ESVS; Class I, Level B).[2,3] To reduce radiation exposure, costs, and contrast-related nephrotoxic effects, CTA can be replaced by color duplex ultrasound (DUS) or contrast-enhanced duplex ultrasound (CE-DUS) for further follow-up. However, a CTA is warranted if there is evidence of a new endoleak, sac enlargement, or endograft migration.[26] Magnetic resonance angiography is limited to selected cases of post-EVAR sac expansion with inconclusive CTA.[3]

Treatment is recommended for type I endoleaks by the SVS (Level 1, Quality B) and ESVS (Class I, Level B) guidelines, regardless of the detection time due to increased risk of rupture.[2,3] Primary endovascular strategy is balloon angioplasty of the sealing areas. Historically, placement of a Palmaz (Cordis) balloon-expandable stent has been used.[27] If the proximal type IA endoleak persists, additional alternatives include placement of a proximal cuff extension if there is additional sealing zone above the stent-graft with or without endoanchors,[28,29] chimney graft,[30] or fenestrated endograft[30] to extend the seal zone above the renal arteries (**Figure 3**). Some ofthese strategies (eg, fenestrated endograft) may require a staged secondary intervention to manu-

Figure 2. Triple-Phase Computed Tomography Angiography (CTA)



Type IA endoleak secondary to a proximal sealing defect in a short-neck abdominal aortic aneurysm, treated with fenestrated proximal endograft extension (A, B, C). Type II endoleak originating from a lumbar artery (D, E, F). Due to sac enlargement greater than 5 mm, the endoleak was treated with translumbar coil and Onyx embolization (Medtronic). Type III endoleak due to separation of the right internal iliac branch, treated with placement of a bridging stent across the gap (G, H, I). The noncontrast phase (A, D, G) is optimal for evaluation of endograft integrity. Arterial phase (B, E, H) allows visualization of high contrast flow, such as in type I, type III, and large type II endoleaks. Delayed phase (C, F, I) shows late contrast flow, ideal for low-flow type II and IV endoleaks.

facture the device. Endovascular coil or glue embolization of the leak has also been described.[31]

Open surgical repair of the AAA with total or partial explantation of the endograft remains an option based on anatomy and patient risk. Type IB endoleaks are treated with distal endograft extension into the iliac bifurcation or the external iliac artery with internal iliac embolization or using an iliac branch device for hypogastric artery preservation, depending on anatomy (**Figure 4**). This technique allows for a more extended sealing area, excluding ectatic or aneurysmal common iliac arteries.[32] Early imaging is imperative to assess persistence if a type I endoleak is noted at EVAR completion and not corrected. Most cases will resolve spontaneously within 1 year[33]; however, up to 27% can recur during late follow-up.[18] Failure to identify and treat a persistent type I endoleak can lead to adverse outcomes.

There is no consensus about the optimal treatment for type I endoleaks; however, several strategies have demonstrated safety and effectiveness. A meta-analysis reported proximal cuff extension as the most common treatment for type I endoleak, with technical success rates of 98.1% (95% CI, 96.3-99.8), followed by chimney endografts, 93.9% (95% CI, 89.9-97.9); fenestrated endografts, 86.2% (95% CI, 77.3-95.1); open conversion, 96.5% (95% CI, 93-100); embolization, 95.2% (95% CI, 90.4-100); and endoanchors, 57.2% (95% CI, 14.1-100). Overall, the aggregate results of endoluminal procedures maintained endoleak resolution in 399 of 439 patients (91%) at 19 months.[34] Treatment choice should be tailored to patient anatomy, endoleak characteristics, device availability, and the experience of the center and/or surgeon. Patients with type I endoleak and hostile neck anatomy or concomitant endoleaks may benefit more from open repair or a proximal fenestrated-branched endograft.[17] Additionally, persistent type I endoleaks are associated with increased cardiac complications and in-hospital, all-cause, and aneurysm-related mortality.[18,33] Cases refractory to endovascular management usually require endograft explantation.[35,36] The SVS guidelines recommend open repair if endovascular interventions fail to treat a type I endoleak and ongoing sac enlargement is detected (Level 1, Quality B).[2]

Type II Endoleaks

A type II endoleak is defined as persistent aneurysmal sac filling due to retrograde blood flow from collateral vessels, such as lumbar arteries or the inferior mesenteric artery (IMA). Type II is the most common, accounting for more than 50% of all endoleaks[12,16] and the most common indication for reintervention after EVAR.[13] Incidence has been reported from 10% to 15%, with most cases detected within the first year of follow-up.[6,37,38] Although 80% to

Figure 3. Endovascular Treatment Modalities for Type IA Endoleaks

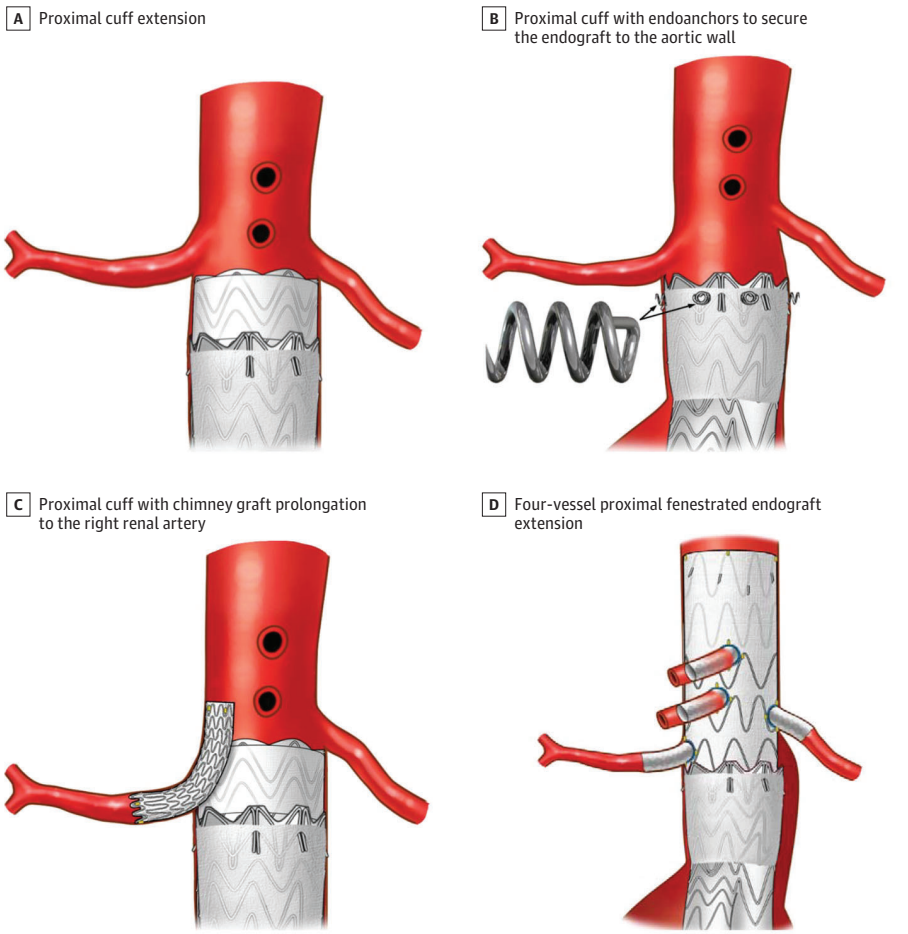


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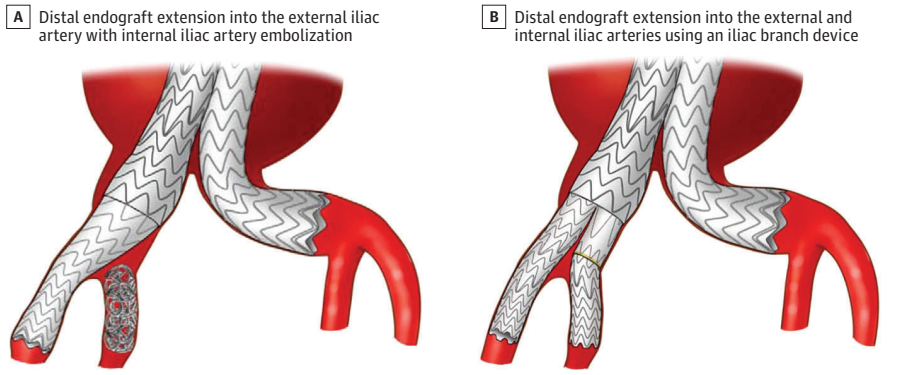
Figure 4. Endovascular Treatment for Type IB Endoleaks



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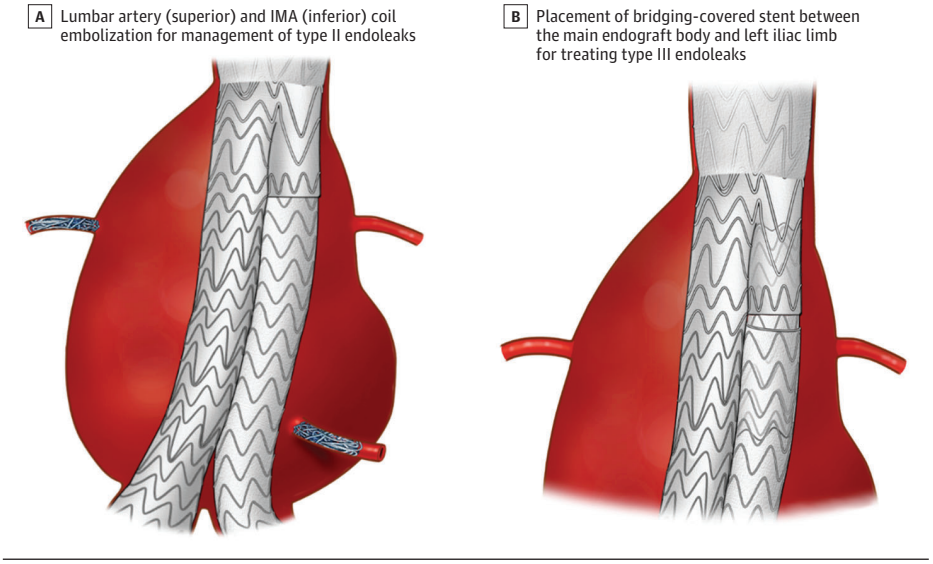
90% of type II endoleaks resolve spontaneously,[39] some may cause aneurysm-related complications such as sac expansion, reintervention, conversion to open repair, and rupture.[40] Risk factors associated with persistent type II endoleak are patent IMA (eg, ≥3 mm), small volume of preoperative mural thrombus, numerous and large caliber lumbar arteries, and anticoagulation.[41-44] Type II endoleaks are generally considered benign, with a risk of rupture less than 1% at 4.8 years.[14,45,46] However, debate remains on the optimal management of type II endoleak. There are no high-quality data about when intervention is required because most cases of rupture occur without sac expansion.[46]

Angiography during EVAR can detect a type II endoleak with late sac filling, often with visualization of the feeding collateral vessel. In the short term, no intervention is needed in the perioperative setting. If a type II endoleak is observed on the 30-day triple-phase CTA after EVAR, the SVS guidelines recommend surveillance with CTA

Figure 5. Endovascular Treatment for Type II and Type III Endoleaks



IMA indicates inferior mesenteric artery. Used with permission of Mayo Foundation for Medical Education and Research.

and/or color DUS at 6 months (Level 2, Quality B). If the type II endoleak persists but is associated with a shrinking or stable aneurysm sac, guidelines recommend continued surveillance with color DUS every 6 months for 24 months and then annually (Level 2, Quality C). Surveillance is strongly recommended for type II endoleaks not associated with aneurysm expansion (Level 1, Quality B).[2] Natural history studies have shown that 5 years after EVAR, 25% of persistent type II endoleaks will exhibit sac regression, 50% to 70% will remain stable (diameter change <5 mm), and 15% to 25% will enlarge.[38,47] There is evidence that magnetic resonance angiography is more sensitive than CTA for detecting type II endoleaks and should be considered in cases of indeterminate CTA findings.[48] Endoleak differentiation can be difficult and, if not performed correctly, may miss concurrent type I or type III endoleak. Therefore, it is imperative that CTA is performed with triple-phase protocol and that additional modalities (eg, contrast-enhanced ultrasound and/or dynamic CTA) be used for patients with unresolved questions after standard CTA.

Indication for treatment depends on the presence of aneurysmal sac expansion and if any other endoleak is identified. The SVS guidelines recommend treating type II endoleak associated with aneurysm expansion more than 5 mm (Level 2, Quality C).[2] In comparison, the ESVS guidelines recommend treating type II endoleak associated with aneurysm growth more than 10 mm (Class IIa, Level C).[3] Before pursuing treatment, concomitant type I or type III endoleak should be ruled out with CTA because occult endoleaks are associated with refractory type II endoleak.[49] If indicated, initial management is usually embolization of the artery that supplies retrograde blood flow to the aneurysm (**Figure 5**A). Endovascular approaches for embolization include transarterial, translumbar, and transcaval.

These techniques may use coils or glue-like embolic liquid such as cyanoacrylate or the Onyx Liquid Embolic System (Medtronic), consisting of ethylene vinyl-alcohol copolymer, dimethylsulfoxide, and micronized tantalum powder.[50] Transarterial embolization of the IMA can be performed via femoral artery access. A microcatheter is advanced into the superior mesenteric artery, meandering mesenteric artery, and IMA until reaching the aneurysm sac. Transarterial embolization of the lumbar arteries is technically more challenging because of the small caliber arterial branches via internal iliac artery navigation and has lower success rates.[50] An alternative to failed transarterial embolization is "transseal" or "perigraft" embolization, in which a catheter is advanced between the iliac endograft and the artery wall to access the sac and the leaking vessel; however, limited evidence is available.[51] Less frequently, transgraft embolization may be performed by creating an endograft defect using laser to access the endoleak nidus, with subsequent coverage of the fabric hole using stent-graft.[52]

Translumbar embolization allows direct percutaneous puncture of the aneurysm sac. It has higher technical success rates and lower endoleak recurrence than other strategies.[46,53] Access is obtained with fluoroscopic and/or CT guidance, and the sac is punctured with a needle. A sheath and microcatheter are then advanced into the endoleak cavity. Through angiographic visualization (eg, "saccography"), the leaking vessel is depicted and embolized using coil, glue, or both. Transcaval embolization consists of puncturing the inferior vena cava wall to penetrate the aneurysmal sac and reach the target vessel.[54] Despite high technical success rates, 60% of aneurysms continue to expand after transcaval embolization and may require multiple reinterventions.[55] Refractory type II endoleaks should be critically evaluated and closely followed since they are associated with occult or misdiagnosed type I and type III endoleak and the development of late type I endoleak.[17,56] Alternative surgical options for type II include laparoscopic or open ligation of the branch arteries but are rarely performed.

The SVS guidelines recommend open repair when endovascular intervention fails to treat a type II endoleak with ongoing aneurysm enlargement (Level 2, Quality C).[2] Open options include ligation of the lumbar arteries or IMA, aneurysmectomy with suture of the leaking vessel ostium, or total or partial endograft explantation.[3] There is no high-quality evidence to recommend a particular treatment for type II; however, selective intervention appears safe and cost-effective.[57] Unless criteria for intervention are met, the management of most isolated type II endoleaks should be conservative

and rarely emergent. A recent multicenter retrospective cohort study including 2018 patients with isolated type II endoleak found no difference in overall survival at 1, 5, and 10 years in patients who underwent reintervention compared with those managed conservatively.[58] Similarly, data from a large registry study reported no difference in health scores at 1 year between patients with and without type II endoleak.[38] Notwithstanding these data, the natural history of type II endoleak is poorly characterized, and further studies are needed to guide proper management.

Management of type II endoleak is challenging because of the heterogeneous clinical course and potential refractoriness to treatment. Preemptive embolization of the aneurysmal sac or branch vessels has been advocated to prevent type II endoleak. A recent systematic review, including 1 RCT and an extensive Vascular Quality Initiative database review, found that patients who underwent prophylactic embolization had lower incidence of sac enlargement (4.3% vs 6.8%; odds ratio [OR], 0.38; 95% CI, 0.26-0.55), type II endoleak (19.7% vs 37.4%; OR, 0.38; 95% CI, 0.30-0.47), and reintervention for type II endoleak (1.2% vs 11.2%; OR, 0.12; 95% CI, 0.06-0.23) compared with controls.[59] Preemptive embolization can be done as a separate procedure (up to 28 days before) or during EVAR. In most cases, coils or vascular plugs are used to embolize the aneurysm sac or feeding vessels (IMA, lumbar, or accessory renal arteries). Results are promising, and evidence favoring preemptive embolization is increasing. Nonetheless, most available evidence comes from retrospective reviews. Further studies are needed to refine indications, procedural details, and cost-effectiveness aspects.

Type III Endoleaks

A type III endoleak occurs because of perigraft flow from either separated endograft components or integrity defects. Type III endoleaks account for 3%[12] of all endoleaks and have an incidence between 2% and 4.5%.[60] They have a 1-year aortic rupture risk of 8.9%[19] and, together with type I, are considered the most dangerous endoleak subtypes. Causes include inadequate endograft deployment overlap, stent migration, or material defects. Type III endoleaks are classified into component separations (type IIIA) or fabric tear (type IIIB). Unfavorable anatomy, such as aneurysm tortuosity, is associated with late type III endoleak development.[61,62] Type III endoleaks secondary to endograft rupture or fabric tear were seen more frequently with the first generations of endografts.[63] However, fabric erosion or stent fracture can spontaneously occur and lead to a hole in the fabric.[64]

In October 2018, the US Food and Drug Administration (FDA) issued a Class I recall pertaining to the AFX Endovascular AAA System endograft (Endologix) due to increased risk of type III endoleak.[65] The cause was attributed to both the AFX with Strata endograft material, which was prone to fabric tears in the region of the aortic bifurcation as a result of the interaction with the endoskeleton, as well as progressive uncoupling of the main endograft body and proximal extension component. The FDA recommends at least yearly follow-up for patients with any AFX endovascular graft (AFX with Strata, AFX with Duraply, AFX2). Currently, the only Endologix available endograft is AFX2, made of Duraply, a thicker material intended to help prevent fabric tears. Endologix has updated instructions for use to avoid uncoupling. In December 2022, the FDA approved a new labeling for the AFX2 product and requested a postmarket study to evaluate the 10-year risk of type III endoleak and compare it with other commercially available endografts.[66]

Type III endoleak can be identified during EVAR or follow-up. If the 30-day follow-up triple-phase CTA shows normal findings, annual surveillance with DUS or CE-DUS can accurately detect type III endoleak.[67,68] Similar to type I, treatment of type III endoleak is strongly recommended by guidelines from the SVS (Level 1, Quality B) and ESVS (Class I, Level B)[2,3] soon after identification, given the risk of rupture if left untreated. However, emergent treatment is only needed in the presence of symptoms or radiographic evidence of rupture or impending rupture. Endovascular options include balloon angioplasty for minor sealing defects between overlapping stents. More commonly, an additional overlapping covered stent is needed when a gap between components is identified (Figure 5B). Despite successful intervention, type III endoleaks have a 25% recurrence rate within 10 years, particularly with the first endograft generations.[60] If endovascular interventions fail and there is ongoing aneurysm enlargement, the SVS guidelines recommend conversion to open repair (Level 2, Quality C).[2]

Type IV Endoleaks

A type IV endoleak is defined as the transudation of blood elements through the endograft or suture lines due to fabric porosity, usually detected intraoperatively on completion angiography. These occur immediately after the device is implanted and resolve with reversal of intraoperative coagulation. Historically, they account for 3% of all endoleaks[12,69]; however, they are rarely found with modern endografts. Since type IV endoleaks are self-limited[70] and do not persist on follow-up, there is no associated risk of rupture.[14] A recent retrospective study of 29 783 patients undergoing EVAR using the Vascular Quality Initiative database found that 1.2% of cases had a late type IV endoleak at 1-year post-EVAR. Risk factors for late type IV endoleak included the presence of type IV endoleak during EVAR (OR, 1.45; 95% CI, 1.26-1.66; $P < .001$) and post-EVAR anticoagulation (OR, 1.88; 95% CI, 1.43-2.49; $P < .001$).[69] Type IV endoleaks are infrequent and resolve spontaneously. They are underreported because they are almost universal with polyester-based endografts on completion angiography. Guidelines from the SVS recommend no treatment (Level 2, Quality C).[2]

Endotension

Endotension, sometimes called type V endoleak, is defined as sac enlargement without a detectable endoleak. Incidence is reported between 1% and 5%,[71,72] but the 4-year risk of rupture is less than 1%.[20,73] Endotension is invoked occasionally as the cause of persistent AAA sac enlargement without an identifiable endoleak but remains poorly understood. Proposed mechanisms for the increased sac tension include blood flow that is not detectable by contemporary imaging techniques, direct pressure transmission through the endograft into the excluded sac that modulates thrombus biology, a microleak through a microporous endograft, and activated fibri-

nolysis inside of the aneurysmal sac, among others.[71] Endotension is a diagnosis of exclusion and can be caused by an undetected endoleak. Further diagnostic evaluation with alternative imaging modalities is recommended to exclude unidentified endoleaks. Delayed phase CTA is ideal for low-flow endoleaks, helping to differentiate type II endoleak from endotension.[2,3]

The lack of high-quality evidence prevents current guidelines from providing robust recommendations on the optimal treatment of endotension. Nonetheless, it is reasonable to observe endotension until sac enlargement reaches the SVS (>5 mm) or ESVS (>10 mm) threshold before considering reintervention. Available evidence supports endograft relining with an additional aortic stent-graft as acceptable treatment for endotension,[74] especially for earlier-generation endografts with higher fabric porosity. Definitive management is open repair with stent-graft removal; however, it is rarely required. A retrospective cohort study reported endotension as the cause of post-EVAR endograft explantation in 6 of 100 cases (6%).[75] Therefore, the treatment approach for endotension must be individualized, and excluding undetected endoleaks should remain imperative.

Conclusions

Endoleaks are the most common complication of EVAR. Management strategies include imaging surveillance, endovascular remediation, or open conversion with or without endograft removal. The optimal approach depends on consideration of the endoleak type, complexity of the proposed reintervention, perioperative risk, and patient and physician preferences. Type I and type III endoleaks have a higher risk of rupture and generally mandate treatment, preferably using endovascular techniques, when anatomically feasible and if the reintervention is predicted to have long-term durability. Type II endoleak, type IV endoleak, and suspected endotension can usually be observed while considering reinter vention if aneurysm sac growth is detected. The eTable in the Supplement provides a comprehensive summary of endoleaks after EVAR.

The natural history and optimal management of type II endoleak remain uncertain and continue to be debated and studied. Although most cases resolve spontaneously, type II endoleak can recur despite endovascular management. When a new endoleak is detected during follow-up, type I and type III endoleaks should be referred for urgent management. In contrast, patients with type II endoleak, type IV endoleak, or presumed endotension do not require immediate evaluation. Finally, endoleaks frequently appear within the first postoperative year after EVAR but can be detected several years later; therefore, all patients should undergo CT imaging evaluation every 5 years[2] while noncontrast CT and/or CE-DUS can be used for surveillance after the first postoperative year if no endoleak and/or sac enlargement is identified.

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