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CIRSE STANDARDS OF PRACTICE GUIDELINES

Quality Improvement Guidelines for Imaging Detection and Treatment of Endoleaks following Endovascular Aneurysm Repair (EVAR)

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Abstract Major concerns after aortic aneurysm repair are caused by the presence of endoleaks, which are defined as persistent perigraft flow within the aortic aneurysm sac. Diagnosis of endoleaks can be performed with various imaging modalities, and indications for treatment are based on further subclassifications. Early detection and correct classification of endoleaks are crucial for planning patient management. The vast majority of endoleaks can be treated successfully by interventional means. Guidelines for Imaging Detection and Treatment of endoleaks are described in this article.

Keywords Arterial intervention · Endovascular aortic repair · Endoleaks

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Introduction

With the increasing use of endovascular repair of aneurysms (EVAR), the need for pre- and posttreatment imaging is deemed an essential part of the overall patient management. A major concern after aortic aneurysm repair is the presence of endoleaks, which are defined as persistent perigraft flow within the aortic aneurysm sac. Endoleaks represent the most common complication of EVAR with an incidence of 10–50 % [1–9]. Anatomical factors that might increase the risk of endoleaks should be assessed by imaging techniques before EVAR, and interventional techniques optimized during device deployment to prevent or minimize subsequent endoleaks.

Depending on the type and persistency of endoleaks, further enlargement and rupture of the aneurysm sac may occur. Therefore, it is vital to evaluate carefully the follow-up imaging, with correct classification of any endoleaks so that the most appropriate management is instigated. Once detected, endoleaks that require treatment are managed predominantly by endovascular techniques.

Definitions

Endoleak

Endoleak (EL) is a common complication of endovascular aortic repair (EVAR). It is defined as a persistent blood flow outside the endograft but within the aneurysm sac [10–13]. It is the most frequent complication of EVAR [12], although its consequences differ widely, depending on the type of EL. Five different types of endoleaks have been described [5]. Treatment and prognosis depend on the type of EL (Table 1) [8, 13–19].

Table 1 Endoleak classifications

Type of endoleak	Location of leak
Type 1	Attachment site
A	Proximal
B	Distal
C	Iliac occluder
Type 2	Collateral vessel
A	Single vessel
B	Multiple vessels
Type 3	Graft failure
A	Midgraft puncture
B	Junctional
Type 4	Porosity of graft wall
Type 5	Endotension

Adapted and modified from [19]

Type I EL

Type I endoleak is defined as a leak at the attachment site of an endograft, and a manifestation of sealing failure. Type I ELs are further subclassified in type IA, IB, and IC depending on the occurrence at proximal and distal end of the endograft, or iliac occluder, respectively. The incidence of type IA proximal endoleak increases in anatomically difficult situations, such as short (<15 mm) neck, large neck diameter (>32 mm), tapered necks, increased angulations (>60°) and landing zones with calcification, thrombus, or uneven size [5]. Incidence of type I endoleak has been reported in as many as 10 % of EVAR cases [5]. Type I endoleaks increase with time from 3.5 % at 30 days to 6.8 % at 12 months [20].

Type I EL is associated with a significant pressure increase in the aneurysm sac and treatment always should be considered.

Type II EL

Type II EL are branch endoleaks and involve retrograde flow into the aneurysm sac from aortic or iliac branch arteries, such as intercostal, lumbar, inferior mesenteric, hypogastric, arteries. They are further differentiated into type IIA when they are related to only one patent branch and type IIB when they are complex with two or more patent branches and creating a flow-through situation. Type II ELs account for approximately 40 % of all endoleaks and are reported in 20–30 % of EVAR cases at 30 days, 18.9 % at 1 year, and 10 % beyond 1 year [5]. Fifty percent of type II ELs resolve spontaneously, 10–15 % are persistent on long-term follow-up, and new type II endoleaks develop in 5–10 % [3, 4, 21, 22].

Type III EL

Type III EL are defined as a junctional leak or modular disconnection (IIIA) or fabric disruption with midgraft holes (IIIB). Like type I endoleaks, type III endoleaks are considered high-pressure, high-risk leaks and always warrant urgent management. Type III endoleaks are infrequent with an estimated incidence of 4 % beyond 1 year [5]. They generally manifest as large collections of contrast, centrally located in the aneurysm sac and usually distant from landing sites (Table 2). Fabric tears can be hard to detect, but modular disconnections usually are well seen with computed tomography (CT).

Type IV EL

Type IV EL are defined as a porous endograft, which is detected <30 days after graft placement, due to fabric porosity. They present at the time of the operation on completion aortograms, when patients are fully anticoagulated. By definition, an endoleak noted on follow-up imaging should not be considered a type IV endoleak.

Type V EL

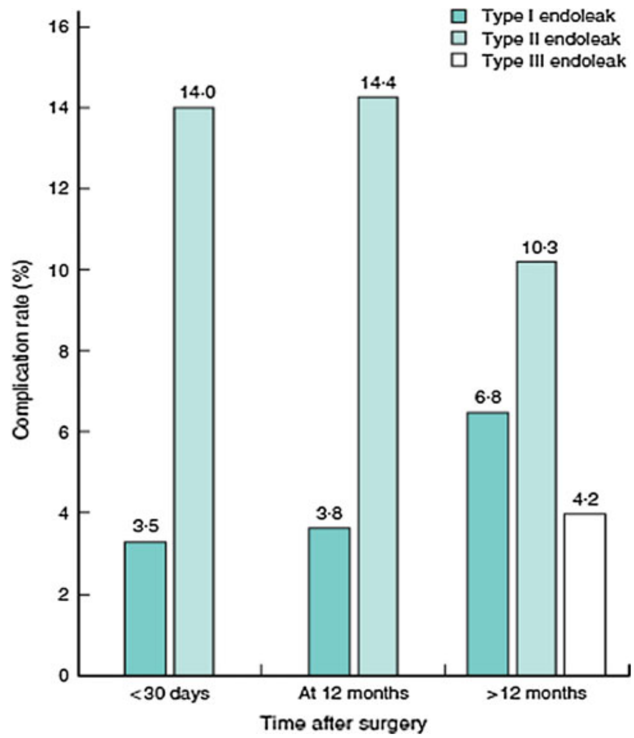
Type V EL refers to the phenomenon of endotension, which represents a persistent or recurrent pressurization of an aneurysm sac without an identifiable type I–IV endoleak on imaging. This may be due to such slow blood flow that it is below the sensitivity limits for detection on current imaging methods. Other alternative explanations that have been suggested for this phenomenon include the development of seroma, infections, and pulsation of the graft wall and thrombus with transmission of the pulse wave through the perigraft space to the native aneurysm wall or by ultrafiltrate across the porous fabric of the endograft. Therefore, endotension may be related to the graft design, including stent structure and fabric compliance (Figs. 1, 2, 3, 4).

Further Definitions

Time of detection On the basis of the time of first detection endoleaks can appear perioperative (first 24 h), early (1–90 days after EVAR), and late (>90 days after EVAR) [1].

Intrasac pressure Endoleaks may be associated with a persistent or recurrent systemic pressure in the aneurysm sac. Intra-aneurysm sac pressure measurements have been performed by invasive techniques post-EVAR; however, with the availability of newer technologies, it may be possible to do this noninvasively in the future [23–25].

Table 2 Incidence of new or persisting endoleak following EVAR



Reprinted from [20]

Sac expansion is defined as an increase of >5 mm in the maximum sac diameter during the interval between successive follow-up imaging [20].

Imaging of Endoleaks

Surveillance is necessary in all patients who undergo EVAR. The frequency of follow-up imaging varies between different centers. Multislice computed tomography (MSCT) and DSA are considered the most accurate imaging techniques for the detection and classification of endoleaks. Most institutions utilize serial CT angiography (CTA) follow-up following EVAR, and a typical protocol is for CTA to be performed at 30 days, 6 months, and annually thereafter [19].

Surveillance should include plain film and/or one type of cross sectional imaging, including 3D reconstructions. Most units continue lifelong surveillance. However, surveillance increases costs by nearly 50 % of the EVAR procedure and 65 % of this due to CT. This also results in a high radiation dose to patients. The vast majority of complications present within 3 years of the initial EVAR [26], and therefore, it has been advocated by some authors that in the absence of complications at 3 years, patients might be discharged [27]. Surveillance protocols are undergoing reevaluation and change in many centers: the 6-month CT scans is being eliminated increasingly, the need for long-term surveillance versus selective surveillance has not been determined, and alternative methods, such as contrast duplex ultrasound (CDU) are being introduced increasingly as an alternative to CT.

Computed Tomography

The current standard for imaging surveillance after EVAR is MSCT. It yields a high sensitivity and specificity for complications following EVAR, including EL detection; however, it is not as specific as DSA for endoleak classification, because CTA has a limited ability to determine blood flow direction, which is critical for endoleak classification [28]. Perigraft flow is seen as a collection of contrast material located anywhere between the aortic wall and the stent-graft. The detection of a small collection in the proximity of metal or calcification, with a similar high attenuation as contrast material may be difficult. When using CT as the primary imaging method, protocols should be optimized to achieve the highest quality imaging while minimizing the dose to the patient [27]. Protocols should be defined to minimize radiation dose. Minimum protocol

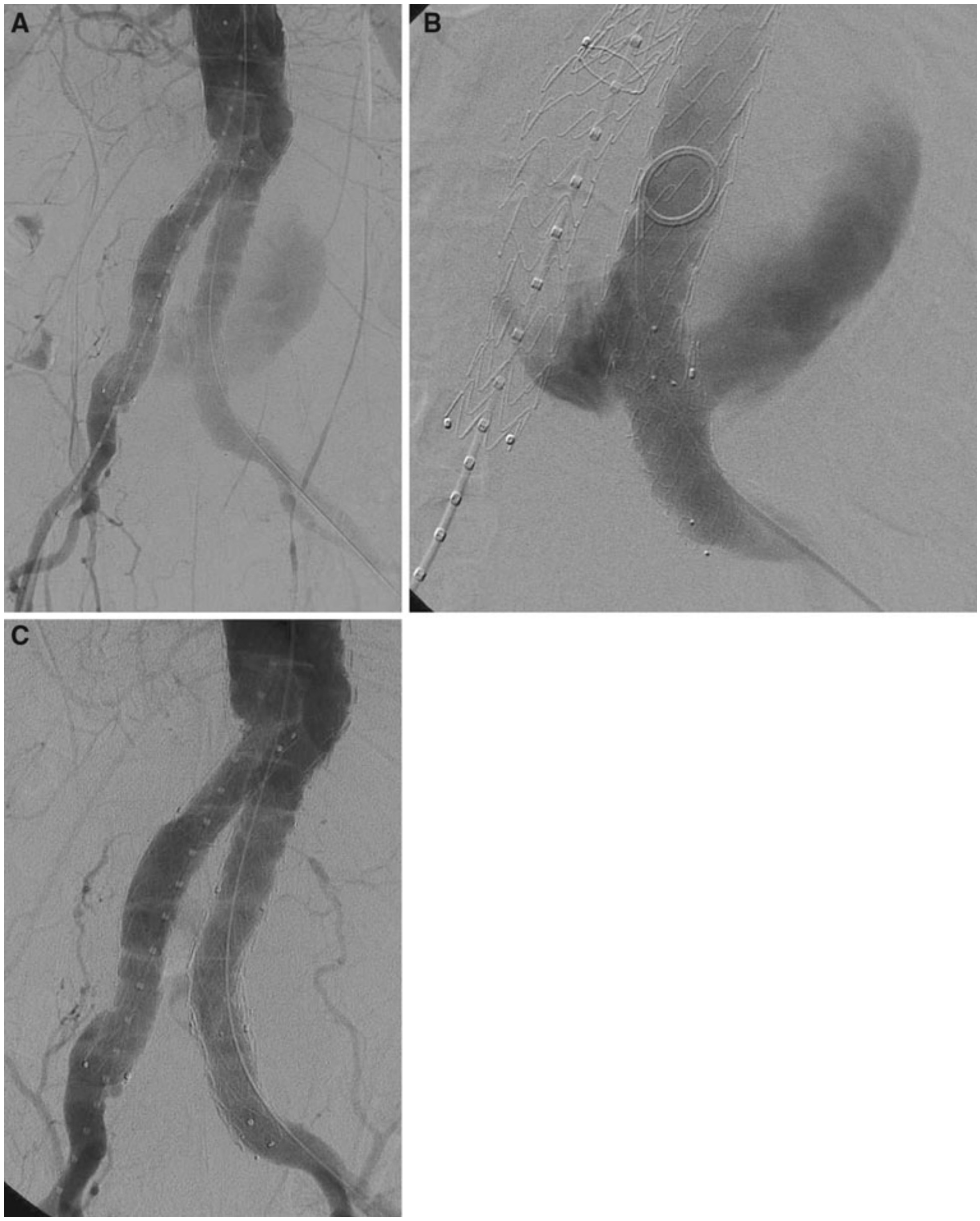


Fig. 1 Type 1b (distal) endoleak at the left distal limb before (**A**, **B**) and after (**C**) endovascular repair (DSA)

is a single arterial phase study. A delayed phase study is of value in those patients with enlarging sacs with no cause on the arterial phase. One alternative technique involves splitting the bolus so that one part is given at a delay before the CT and the other just before, so that the CT is arterial phase but with the benefit of the equivalent of a late phase study. Delayed phase images after contrast injection are helpful to differentiate progressive aneurysm expansion due to low-flow endoleak from endotension. The unenhanced phase rarely contributes to the diagnosis and should be omitted whenever possible. Standard techniques comprise the use of MSCT scanners.

Technical parameters will vary depending on the type and make of the scanner within each unit. With the use of MSCT, 3D imaging reconstructions can be performed [28–30] to classify and to help localize the site of endoleaks. For a 64 MSCT, the following parameters are suggested in Table 3.

Color Duplex Ultrasound and Contrast-enhanced Ultrasound

Avoidance of ionizing radiation and potentially nephrotoxic contrast agent administration are the main advantages

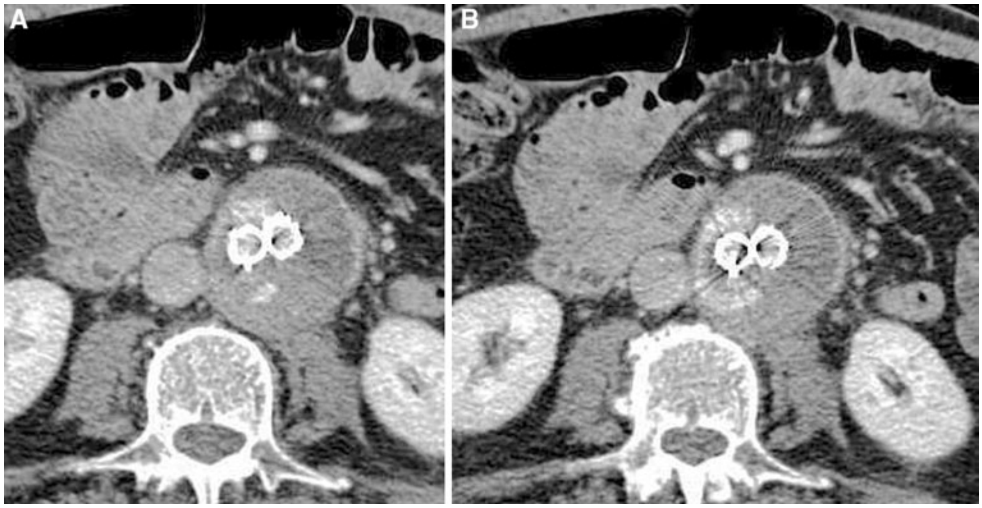


Fig. 2 Type 2 endoleak at contrast-enhanced CT

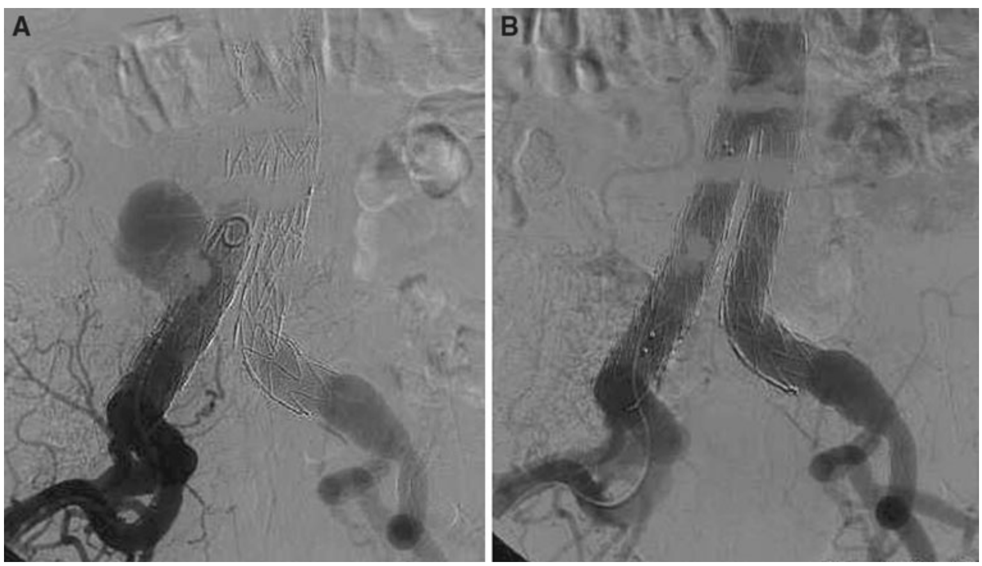


Fig. 3 Type 3 disconnection leak at the right limb, before (**A**) and after (**B**) endovascular repair (DSA)

of color duplex ultrasound (CDU) compared with CTA. CDU also offers the additional advantage to document flow velocity and direction in the aneurysm sac, thus providing dynamic information. Using US contrast media can improve the sensitivity and specificity of CDU [31–33]. Furthermore, it can be used for the guidance of percutaneous treatment or ELs [34].

Several recent studies also have shown superior results of contrast-enhanced ultrasound (CEUS) compared with CTA for the detection and classification of endoleaks. However, these results must be interpreted with caution, because the quality of CDU imaging may be affected by several factors, including patient habitus, bowel gas, and the skill of the operator [28, 32]. CTA cannot currently be completely substituted, because it enables a more precise evaluation of aneurysm morphologic changes, aneurysm sac diameter, graft anchorage, and integrity.

Some investigators have recommended that if no endoleak or sac enlargement is detected on the first-year follow-up CT, CDU should be used as the sole follow-up imaging modality following EVAR. Detection of a new onset endoleak or sac enlargement by CDU would prompt further CT imaging [35, 36]. CDU and CEUS also can be used along with CTA when the latter reveals the presence of endoleak to provide a better characterization of this taking advantage of the angiodynamic behavior of the contrast agent. It also may be useful when there is an increase in the aneurysm sac diameter with a negative CTA

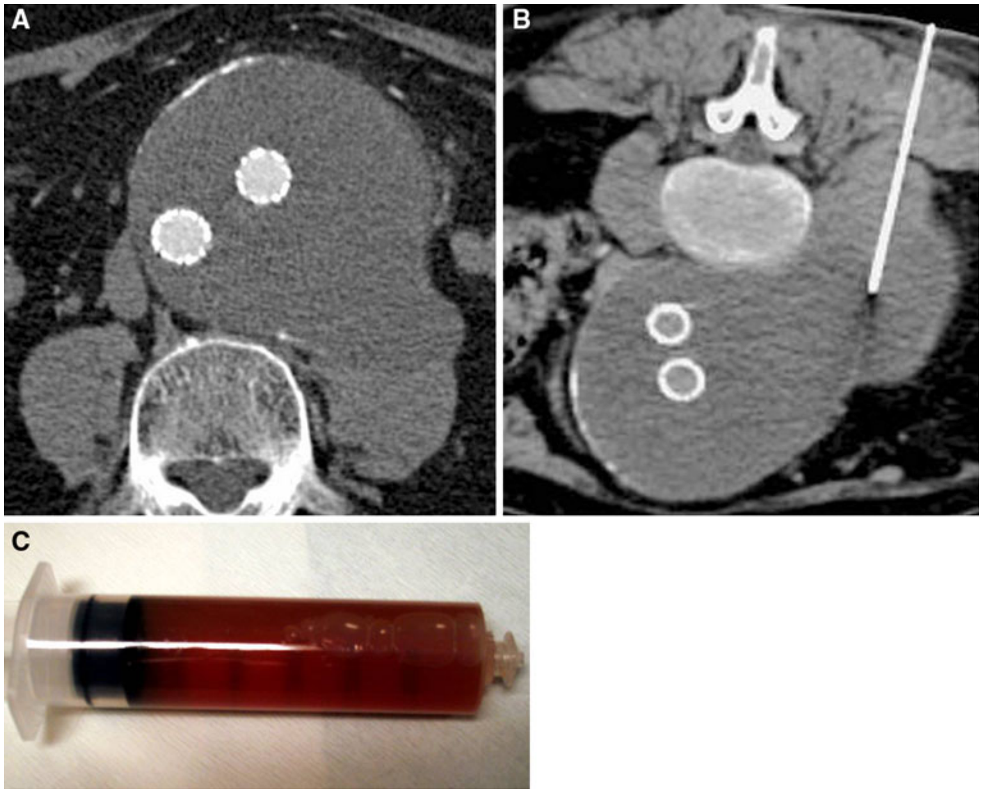


Fig. 4 Type 5 (endotension) endoleak. No evidence of extravasation into the increased aneurysmal sac (**A**). Direct puncture (**B**) revealed a serous, slightly hemorrhagic content (**C**). Courtesy of Lammer J, Vienna, Austria

Table 3 Standard parameters for evaluation of the aorta for endoleaks using 64 MSCT

Abdominal aorta (64-slice scanner), i.e., Siemens Somatom 64						
	kV	mAS	Rot Time	Slice	Coll. Slice	Pitch
Arterial phase	120	200	0.5	3	64/0.6	1
Venous phase	120	200	0.5	3	64/0.6	1
Contrast medium injection parameters						
Total volume: 120 ml						
Flow: 4.5 ml/s						
Care bolus (CB) + 6 s delay						
Saline chaser bolus after injection of the contrast medium						

Region of interest (ROI) in the proximal abdominal aorta at the celiac axis

Special applications MPR multiplanar reconstruction, *MIP* maximum intensity projection, thin MIP, *SSD* (shaded surface display), *VRT* volume rendering techniques, *CPR* curved planar reformation

to show sac reperfusion or to monitor type II endoleaks, thereby reducing the need for CTA with consequent reduction of costs and exposure to radiation.

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) is used less commonly in clinical practice for endoleak detection after EVAR. Although the sensitivity of gadolinium enhanced MRI might be superior to CT for detection of type II ELs, image quality and interpretation is decreased by significant artefacts, coming from the device itself or other sources [37, 38]. MRI with new blood pool contrast agents with longer serum half-life can help to image patients with nonshrinking aneurysms due to endoleaks that are occult on CTA [38]. Further studies and MRI compatible stent-grafts are required to define the exact role of MRI for surveillance after EVAR.

Angiography

CTA and CDU are routinely used methods to detect endoleaks after EVAR. However, DSA may still be required to determine the type and source of an already detected EL after CT evaluation [5]. ELs occurring in the distal sealing zones (IB) may be confused with type II ELs. Balloon blockage techniques and selective angiography can clearly differentiate ELs, if such problems arise. Aortography performed with the diagnostic catheter placed above and within the endograft permit exclusion of a type IA proximal EL vs. type II EL. Additionally, selective angiography of the superior mesenteric and the hypogastric arteries can be assessed to identify collateral pathways to inferior mesenteric and lumbar arteries, respectively, as a prelude to treatment of the EL with embolization during the same session in suitable cases. If the EL cannot be visualized angiographically via a transarterial approach, direct translumbar puncture of the sac percutaneously, can be performed [5, 6].

Plain Radiography

Although they do not image endoleaks directly, plain radiographs (anteroposterior and lateral projections) are very accurate to assess migration, stent fractures, and modular separations, which may result in type I and III endoleaks [12].

Indications for Treatment of ELs

Secondary interventions are performed following EVAR in 16.2 % of patients most commonly due to endoleak (Table 4) [20, 27, 39–58]. Type I EL is associated with significant pressure increase in the sac and constitutes a high risk for continued aneurysm expansion and rupture. Therefore, type I ELs are considered clinically significant and should be treated quickly upon detection.

For type II endoleaks, a "wait and see" approach is accepted with regular follow-up for stable aneurysms, because up to 50 % of these endoleaks will eventually thrombose spontaneously [12, 40]. If there is an increase in size of the aneurysm sac, this is indicative of high sac pressure and the endoleak should be treated. There is currently no defined increase in size in the literature at which intervention is recommended, but in the authors practice we use a >5-mm increase in size.

Table 4 Summary of studies retrieving reintervention rates for endoleaks

Study	Year	EVR cases	Secondary interventions (%)	Reintervention for EL (%)
EVAR1	2005	543	20	8
EVAR 2	2005	178	26	8
Ricco	2003	1012		7.5
Eurostar	2006	2846	8.7	4.2
Cao	2009	349	9.5	3.1
Criado	2003	240	3.8	1.3
Etkouri	2003	100	29	10
Flora	2003	108	26	9
Carpenter	2004	192	12	5
Lalka	2005	136	12.5	7.3
Hiramoto	2007	325	8.6	6.7
Abbruzese	2008	565	10.6	7.8
Smih	2008	113	27	9.7
Traul	2008	245	6	3
Black	2009	417	7.4	1.9
Espinosa	2009	337		5.6
Jean Baptiste	2009	447	6.5	4
Pitoulias	2009	617	22.5	11.6
Conrad	2009	832	11	8

Reprinted from [26]

Type III ELs should be treated upon detection. An additional covered extension cuff at the level of the leak is required. Type IV EL represents self-limiting blood flow through the graft material due to porosity and treatment is not usually required. Type V ELs are treated individually. Invasive pressure monitoring may be a useful adjunct; however, treatment of type V ELs is not defined. The risk of rupture from combined types I and III endoleaks is reported to be ten times of that of type II endoleaks (Table 5).

Table 5 Risk for ruptures in EL

Adapted from: The EUROSTAR experience [4]		
Type I + III EL	10/2463	0.4 %
Type II EL	1/2463	0.04 %
Unknown type EL	5/2463	0.2 %
Significant risk factors for rupture		
Type I EL		$p < 0.001$
Type III EL		$p < 0.001$
Migration		$p < 0.001$

Based on the following numbers the risk of rupture from combined types I and III endoleaks is reported to be ten times that of type II endoleaks

Patient Preparation

The majority of cases can be treated endovascularly (82 %) with only a minority requiring surgery (18 %) [3]. In case of a strictly endovascular procedure, patient preparation and management is the same as for any standard endovascular procedure. Where a formal cut down is required, the preparation is as for any surgical case. Prophylactic antibiotics for secondary interventions are advisable, although there is no convincing evidence of benefit for the prevention of infection in the literature.

Procedural Features

Good patient planning with appropriate patient and device selection and adherence to high standards in technique should minimize the development of endoleaks that require treatment.

Type I El

Immediate type I EL may be treated by balloon remodelling or the use of various extender cuffs or other modular devices, such as bare stents [5, 19]. Delayed type I ELs might occur with changes in the configuration of the aorta or graft migration. These ELs can be managed by placement of an extension cuff if any additional landing zone exists, which gives an additional coverage at the end of the stent graft or balloon angioplasty and placement of bare stents to increase the radial strength to the sealing zones. Rarely obliteration of the lumen with coils, glue, thrombin, or other embolic agents, such as N-BCA (Trufill, Cordis, Miami, FL) "glue" or Onyx (ev3, Plymouth, MN) is performed [59, 60]. In these approaches, the graft is probed from the femoral or brachial artery with a curved catheter, i.e., SOS Omni (AngioDynamics). After the tip of the catheter has engaged the channel of the leak, a microcatheter can be advanced and embolization performed by using a variety of agents [5, 59]. In case of failure of endovascular techniques to control type I EL, conversion to open surgery may be the only option.

Type II EL

Common sources for type II ELs are arterial branches that are prone to retrograde flow after having being covered by a stent-graft. Therefore, careful CTA evaluation of vessels should be performed during the planning phase of EVAR cases to reduce the risk of type II ELs. To prevent endoleaks caused by retrograde flow when coverage of the internal iliac arteries (IIA) is indicated due to combined iliac artery aneurysm, occlusion of the internal iliac artery is commonly performed with transarterial embolization using metallic coils or nitinol occlusion plugs. Embolization should involve only the proximal part of the IIA, preserving the branches for collateral circulation. Bilateral IIA occlusions should be avoided if possible, because of the potential risk of ischemic complications, although such complications have been shown to be far less morbid than previously thought [60, 61].

Prominent lumbar arteries or a prominent IMA may be preevaluated as a potential source of ELs and can be embolized by coils prior stent-graft insertion [62–64]. No overall significant reduction in endoleak occurrence has been demonstrated, and pre-EVAR embolization is not generally recommended [65].

In case of type II ELs associated with continuous increase of the size of the aneurysm sac detected in the course of follow-ups after stent-graft insertion, translumbar and transarterial embolization techniques are effective [1, 5]. The use of microcatheters and embolization of the feeding and draining arteries with coils is one method. These procedures can be rather time-consuming and can require advanced technical skills. Also, catheterization and successful embolization may not be possible in all patients because of anatomic limitations. To prevent recurrences after embolization, entering the aneurysm sac with a microcatheter and embolization of both the channels in the sac and the feeding/draining vessels is advised. Clear anatomic background and the knowledge of vessel anatomy are mandatory.

Leaks from lumbar arteries may be treated via catheterization of the hypogastric arteries. Leaks from the IMA may be treated by a passage from the SMA via collaterals (Riolan Anastomosis). Laparoscopic retroperitoneal ligation of the IMA or the lumbar arteries may be used, as an alternate option for treatment of type II endoleaks [66, 67]. However, it requires advanced laparoscopic experience and is more invasive than embolization techniques.

If the endoleak cannot be reached by endovascular route, translumbar embolization with direct puncture of the aneurysm sac can be performed with a percutaneous approach [34, 68, 69]. It can be performed under CT, fluoroscopy, or ultrasound guidance. A 18- to 22-gauge needle is inserted directly into the aneurysm sac with imaging guidance (US and/or CT) and then the embolization is performed by using embolic agents, such as coils, glue, thrombin, and/or onyx (see equipment) [5, 70, 71].

Type III EL

Extreme angulation of the neck or iliac segments increases the risk of type III endoleaks. Most fabric failures have been found to be associated with specific graft designs and materials that have subsequently been modified or withdrawn from the market. Some failures related to modular separation are preventable by ensuring adequate overlap of modular elements of the graft. Existing type III ELs are treated by placement of additional extensions or cuffs. When endoleak is due to perforation of the fabric, relining of the entire stent graft with a new bifurcated one or converting to aortouniiliac graft with femoro-femoral cross-over bypass graft and deployment of an occluder to the contralateral side can be performed. Solitary limb tears may be treated by relining of the individual limb only. If these techniques fail to treat the leak, surgical conversion is the final treatment option.

Type IV EL

Type IV endoleaks have been described particularly with the first generation of stent-grafts; however, with new generation low-porosity graft fabrics, these endoleaks are uncommon.

Type V EL

Endotension seemed more common with expanded polytetrafluoroethylene (ePTFE) fabric grafts rather than polyester covered ones. New-generation ePTFE grafts include a second layer of low permeability ePTFE to decrease this risk [72]. Usually endotension is considered a low-risk complication of EVAR in the short-term; however, continued enlargement of the sac usually requires surgical conversion, although relining of the entire stent-graft also has been attempted.

Medication and Periprocedural Care

Where a larger access to the groin is essential with surgical cut downs, to deliver proximal or distal cuffs or in case of stent in stent techniques (type I and III ELs), anticoagulation with 3–5,000 U of heparin and antibiotics should be given as an adjunct to the procedure.

Postprocedural Follow-up

DSA will be performed as part of the secondary intervention. If this shows a satisfactory result, then short-term investigations can be skipped and further follow-up imaging should be performed as for primary EVAR with the use of MSCT or other adequate alternative imaging modalities.

Outcome

Effectiveness For the repair of type I and III Els, the described procedures are highly effective, although a small incidence of recurrence is recognized. For type II Els, failure and recurrences have been reported in up to 80 % because of multiple communicating vessels [26, 41–58].

Complications

Potential complications after EL repair resemble those from the individual endovascular technique used. These represent the hazards of embolization techniques for the treatment of type II endoleaks and all hazards of stent displacement for the repair of types I and III ELs using proximal and distal cuffs or stent-in-stent techniques. Other complications are those generally encountered in endovascular therapies, such as puncture-associated bleeding complications, infections, or an inflammatory or thrombotic origin.

Conclusions

Endoleaks are common complications in EVAR. With the introduction of improved techniques and stent-graft designs, the incidence of ELs has been reduced. Type II ELs are still by far the most common type. The vast majority of endoleaks that require treatment can be managed with endovascular techniques with only a minority requiring surgical intervention.

Conflict of interest The authors have no conflict of interest.

References

1. Veith FJ, Baum RA, Ohki T et al (2002) Nature and significance of endoleaks and endotension: summary of opinions expressed at an international conference. J Vasc Surg 35(5):1029–1035
2. Solis MM, Ayerdi J, Babcock GA et al (2002) Mechanism of failure in the treatment of type II endoleak with percutaneous coil embolization. J Vasc Surg 36(3):485–491
3. Harris PL, Dimitri S (1999) Predicting failure of endovascular aneurysm repair. Eur J Vasc Endovasc Surg 17(1):1–2
4. van Marrewijk CJ, Fransen G et al EUROSTAR Collaborators (2004) Is a type II endoleak after EVAR a harbinger of risk? Causes and outcome of open conversion and aneurysm rupture during follow-up. Eur J Vasc Endovasc Surg 27(2):128–137
5. Cao P, De Rango P, Verzini F et al (2010) Endoleak after endovascular aortic repair: classification, diagnosis and management following endovascular thoracic and abdominal aortic repair. J Cardiovasc Surg 51:53–69
6. Van Bindsbergen L, Braak SJ, van Strijen MJ et al (2010) Type II endoleak embolization after endovascular abdominal aortic aneurysm repair with use of real-time three-dimensional fluoroscopic needle guidance. J Vasc Interv Radiol 21:1443–1447

7. Buth J, Laheij RJ (2000) Early complications and endoleaks after endovascular abdominal aortic aneurysm repair: report of a multicenter study. J Vasc Surg 31:134–146
8. van Marrewijk C, Buth J, Harris PL et al (2002) Significance of endoleaks after endovascular repair of abdominal aortic aneurysms: the EUROSTAR experience. J Vasc Surg 35(3):461–473
9. Liaw JV, Clark M, Gibbs R et al (2009) Update: complications and management of infrarenal EVAR. Eur J Radiol 71(3): 541–551
10. White GH, Yu W, May J et al (1997) Endoleak as a complication of endoluminal grafting of abdominal aortic aneurysms: classification, incidence, diagnosis, and management. J Endovasc Surg 4(2):152–168
11. Rosen RJ, Green RM (2008) Endoleak management following endovascular aneurysm repair. J Vasc Interv Radiol 19:S37–S43
12. Moll FL, Powell JT, Fraedrich G et al (2011) Management of abdominal aortic aneurysms clinical practice guidelines of the European Society for Vascular Surgery. Eur J Vasc Endovasc Surg 41:S1–S58
13. Baum RA, Stavropoulos SW et al (2003) Endoleaks after endovascular repair of abdominal aortic aneurysms. J Vasc Interv Radiol 14:1111–1117
14. Baum RA, Carpenter JP, Tuite CM et al (2000) Diagnosis and treatment of inferior mesenteric arterial endoleaks after endovascular repair of abdominal aortic aneurysms. Radiology 215(2): 409–413
15. Silverberg D, Baril DT, Ellozy SH et al (2006) An 8-year experience with type II endoleaks: natural history suggests selective intervention is a safe approach. J Vasc Surg 44(3):453–459
16. Cynamon J, Federman AS, Veith FJ (1999) Repair of junctional stent-graft leaks with use of a bare metal stent. J Vasc Interv Radiol 10(3):275–280
17. Heikkinen MA, Arko FR, Zarins CK (2004) What is the significance of endoleaks and endotension. Surg Clin N Am 84(5): 1337–1352
18. Gilling-Smith G, Brennan J, Harris P et al (1999) Endotension after endovascular aneurysm repair: definition, classification, and strategies for surveillance and intervention. J Endovasc Surg 6(4): 305–307
19. Shah A, Stavropoulos SW (2009) Imaging surveillance following endovascular aneurysm repair. Semin Interv Radiol 26:10–16
20. Drury D, Michaels JA, Jones L et al (2005) Systematic review of recent evidence for the safety and efficacy of elective endovascular repair in the management of infrarenal abdominal aortic aneurysm. Br J Surg 92:937–946
21. Baum RA, Carpenter JP, Golden MA et al (2002) Treatment of type 2 endoleaks after endovascular repair of abdominal aortic aneurysms: comparison of transarterial and translumbar techniques. J Vasc Surg 35:23–29
22. Solis MM, Ayerdi J, Babcock GA et al (2002) Mechanism of failure in the treatment of type II endoleak with percutaneous coil embolization. J Vasc Surg 36:485–491
23. Springer F, Günther RW, Schmitz-Rode T (2008) Aneurysm sac pressure measurement with minimally invasive implantable pressure sensors: an alternative to current surveillance regimes after EVAR? Cardiovasc Interv Radiol 31:460–467
24. Dias NV, Ivancec K, Malina M et al (2004) Intra-aneurysm sac pressure measurements after endovascular aneurysm repair: differences between shrinking, unchanged, and expanding aneurysms with and without endoleaks. J Vasc Surg 39:1229–1235
25. Baum RA, Carpenter JP, Cope C et al (2001) Aneurysm sac pressure measurements after endovascular repair or abdominal aortic aneurysms. J Vasc Surg 33:32–41
26. Nordon IM, Karthikesalingam A, Hinchcliffe RJ et al (2010) Secondary interventions following endovascular aneurysm repair (EVAR) and the enduring value of graft surveillance. Eur J Vasc Endovasc Surg 39:547–554
27. Stavropoulos SW, Charagundla SR (2007) Imaging techniques for detection and management of endoleaks after endovascular aortic aneurysm repair. Radiology 243(3):641–655
28. Fan CM, Rafferty EA, Geller SC et al (2001) Endovascular stent-graft in abdominal aortic aneurysms: the relationship between patent vessels that arise from the aneurysmal sac and early endoleak. Radiology 218(1):176–182
29. Iezzi R, Cotroneo AR, Filippone A et al (2008) Multidetector-row computed tomography angiography in abdominal aortic aneurysm treated with endovascular repair: evaluation of optimal timing of delayed phase imaging for the detection of low-flow endoleaks. J Comput Assist Tomogr 32(4):609–615
30. Van der Laan MJ, Bartels LW, Viergever MA et al (2006) Computed tomography versus magnetic resonance imaging of endoleaks after EVAR. Eur J Vasc Endovasc Surg 32(4):361–365
31. Henao EA, Hodge MD, Felkai DD et al (2006) Contrast enhanced duplex surveillance after endovascular abdominal aortic aneurysm repair: improved efficacy using a continuous infusion technique. J Vasc Surg 43:259–264
32. Golzarian J, Murgo S, Dussaussois L (2002) Evaluation of abdominal aortic aneurysm after endoluminal treatment: comparison of colour Doppler sonography with biphasic helical CT. AJR Am J Roentgenol 178(3):623–628
33. Bakken AM, Illig KA (2010) Long-term follow-up after endovascular aneurysm repair: is ultrasound alone enough? Perspect Vasc Surg Endovasc Ther 22(3):145–151
34. Gorlitzer M, Mertikian G, Trnka H et al (2008) Translumbar treatment of type II endoleaks after endovascular repair of abdominal aortic aneurysm. Interact Cardiovasc Thorac Surg 7(5):781–784
35. Sternbergh WC, Greenberg RK, Chuter TA et al (2008) Redefining postoperative surveillance after endovascular aneurysm repair: recommendations based on 5-year follow-up in the US Zenith multicenter trial. J Vasc Surg 48:278–284
36. Pitton MB, Schweitzer H, Herber S et al (2005) MRI versus helical CT for endoleak detection after endovascular aneurysm repair. AJR Am J Roentgenol 185(5):1275–1281
37. Schwope RB, Alper HJ, Talenfeld AD et al (2007) MR angiography for patient surveillance after endovascular repair of abdominal aortic aneurysms. AJR Am J Roentgenol 188:334–340
38. Cornelissen SA, Prokop M, Verhagen HJ et al (2010) Detection of occult endoleaks after endovascular treatment of abdominal aortic aneurysm using magnetic resonance imaging with a blood pool contrast agent: preliminary observations. Invest Radiol 45(9):548–553
39. Steinmetz E, Rubin BG, Sanchez LA et al (2004) Type II endoleak after endovascular abdominal aortic aneurysm repair: a conservative approach with selective intervention is safe and cost effective. J Vasc Surg 39:306–313
40. EVAR trial participants (2005) Endovascular aneurysm repair and outcome in patients unfit for open repair of abdominal aortic aneurysm (EVAR trial 2): randomised controlled trial. Lancet 25:2187–2192
41. Ricco JB, Goëau-Brissonnière O, Rodde-Dunet MH et al (2003) Use of abdominal aortic endovascular prostheses in France from 1999 to 2001. J Vasc Surg 38(6):1273–1281
42. Hobo R, Buth J, EUROSTAR collaborators (2006) Secondary interventions following endovascular abdominal aortic aneurysm repair using current endografts. A EUROSTAR report. J Vasc Surg 43(5):896–902
43. Cao P, De Rango P, Parlani G et al (2009) Durability of abdominal aortic endograft with the Talent Unidoc stent graft in common practice: core lab reanalysis from the TAURIS multicenter study. J Vasc Surg 49(4):859–865

44. Criado FJ, Fairman RM, Becker GJ et al (2003) Talent LPS AAA stent graft: results of a pivotal clinical trial. J Vasc Surg 37(4): 709–710
45. Elkouri S, Gloviczki P, McKusick MA et al (2003) Endovascular repair of abdominal aortic aneurysms: initial experience with 100 consecutive patients. Mayo Clin Proc 78(10):1234–1242
46. Flora HS, Chaloner EJ, Sweeney A et al (2003) Secondary intervention following endovascular repair of abdominal aortic aneurysm: a single centre experience. Eur J Vasc Endovasc Surg 26(3):287–292
47. Carpenter JP, Endologix Investigators (2004) Midterm results of the multicenter trial of the powerlink bifurcated system for endovascular aortic aneurysm repair. J Vasc Surg 40(5):849–859
48. Lalka S, Dalsing M, Cikrit D et al (2005) Secondary interventions after endovascular abdominal aortic aneurysm repair. Am J Surg 190(5):787–794
49. Hiramoto JS, Reilly LM, Schneider DB et al (2007) Long-term outcome and reintervention after endovascular abdominal aortic aneurysm repair using the Zenith stent graft. J Vasc Surg 45(3):461–465
50. Abbruzzese TA, Kwolek CJ, Brewster DC et al (2008) Outcomes following endovascular abdominal aortic aneurysm repair (EVAR): an anatomic and device-specific analysis. J Vasc Surg 48:19–28
51. Smith S, Mountcastle S, Burridge A et al (2008) A single-institution experience with the AneuRx stent graft for endovascular repair of abdominal aortic aneurysm. Ann Vasc Surg 22:221–226
52. Traul D, Street D, Faught W, Eaton M et al (2008) Endoluminal stent-graft placement for repair of abdominal aortic aneurysms in the community setting. J Endovasc Ther 15:688–694
53. Black SA, Carrell TW, Bell RE et al (2009) Long-term surveillance with computed tomography after endovascular aneurysm repair may not be justified. Br J Surg 96:1280–1283
54. Espinosa G, Ribeiro Alves M et al (2009) A 10-year single-center prospective study of endovascular abdominal aortic aneurysm repair with the talent stent-graft. J Endovasc Ther 16:125–135
55. Jean-Baptiste E, Batt M, Azzaoui R et al (2009) A comparison of the mid-term results following the use of bifurcated and aorto-uni-iliac devices in the treatment of abdominal aortic aneurysms. Eur J Vasc Endovasc Surg 38(3):298–304
56. Pitoulias GA, Schulte S, Donas KP et al (2009) Secondary endovascular and conversion procedures for failed endovascular abdominal aortic aneurysm repair: can we still be optimistic? Vascular 17(1):15–22
57. Conrad MF, Adams AB, Guest JM et al (2009) Secondary intervention after endovascular abdominal aortic aneurysm repair. Ann Surg 250(3):383–389
58. Rosen RJ, Green RM (2008) Endoleak management following endovascular aneurysm repair. J Vasc Interv Radiol 19:37–43
59. Sheehan MK, Barbato J, Compton CN et al (2004) Effectiveness of coiling in the treatment of endoleaks after endovascular repair. J Vasc Surg 40(3):430–434
60. Schoder M, Zaunbauer L, Hölzenbein T et al (2001) Internal iliac artery embolization before endovascular repair of abdominal aortic aneurysms: frequency, efficacy, and clinical results. AJR Am J Roentgenol 177(3):599–605
61. Bratby MJ, Munneke GM, Belli AM et al (2008) How safe is bilateral internal iliac artery embolization prior to EVAR? Cardiovasc Interv Radiol 31:246–253
62. Gould DA, McWilliams R, Edwards RD et al (2001) Aortic side branch embolization before endovascular aneurysm repair: incidence of type II endoleak. J Vasc Interv Radiol 12:337–341
63. Axelrod DJ, Lookstein RA, Guller J et al (2004) Inferior mesenteric artery embolization before endovascular aneurysm repair: technique and initial results. J Vasc Interv Radiol 15:1263–1267
64. Bonvini R, Alerci M, Antonucci F (2003) Preoperative embolization of collateral side branches: a valid means to reduce type II endoleaks after endovascular AAA repair. J Endovasc Ther 10(2): 227–232
65. Görich J, Rilinger N, Sokiranski R et al (2001) Endoleaks after endovascular repair of aortic aneurysm: are they predictable?. Initial results. Radiology 218(2):477–480
66. Wisselink W, Cuesta MA, Berends FJ et al (2000) Retroperitoneal endoscopic ligation of lumbar and inferior mesenteric arteries as a treatment of persistent endoleak after endoluminal aortic aneurysm repair. J Vasc Surg 31:1240–1244
67. Richardson WS, Sternbergh WC 3rd, Money SR (2003) Laparoscopic inferior mesenteric artery ligation: an alternative for the treatment of type II endoleaks. J Laparoendosc Adv Surg Tech A 13(6):355–358
68. Ellis PK, Kennedy PT, Collins AJ et al (2003) The use of direct thrombin injection to treat a type II endoleak following endovascular repair of abdominal aortic aneurysm. Cardiovasc Interv Radiol 26(5):482–484
69. Stavropoulos SW, Carpenter JP, Fairman RM et al (2003) Inferior vena cava traversal for translumbar endoleak embolization after endovascular abdominal aortic aneurysm repair. J Vasc Interv Radiol 14(9):1191–1194
70. Barbiero G, Baratto A, Ferro F et al (2008) Strategies of endoleak management following endoluminal treatment of abdominal aortic aneurysm in 95 patients: how, when and why. Radiol Med 113(7):1029–1042
71. Kasthuri RS, Stivaros SM, Gavan D (2005) Percutaneous ultrasound-guided thrombin injection for endoleaks: an alternative. CVIR 28(1):110–112
72. Trocciola SM, Dayal R, Chaer RA et al (2006) The development of endotension is associated with increased transmission of pressure and serous components in porous expanded polytetrafluoroethylene stent-grafts: characterization using a canine model. J Vasc Surg 43(1):109–116