Imaging characteristics and interventional therapy of type II endoleaks following endovascular repair of abdominal aortic aneurysms

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Learning objectives

Relevant aspects of imaging and assessment of type II endoleaks following endovascular repair of aortic aneurysms are addressed. Different treatment options are discussed.
Background

The aim of endovascular abdominal aortic aneurysm repair (EVAR) is to prevent rupture of the aneurysm by exclusion of the aneurysm sac. Endoleaks represent a common complication following EVAR (1-3) where the blood flow into the aneurysm sac surrounding the stentgraft persists (4).

Endoleaks are classified with regard to their filling mechanism. Type I endoleaks represent discontinuity along the attachment sites, type II endoleaks are fed from aortic branches, in type III the endoleak is caused by defects of graft material or graft component disconnection, graft porosity is the cause in type IV endoleaks (4). Growth of the aneurysm without detection of an endoleak is referred to as endotension which has been reported to occur in 5% to 7%. Endoleaks can be subdivided into transient and persistent (>6 months) (5).

As type I and III endoleaks are significant risk factors for aneurysm rupture, prompt reintervention is required (6). The role of type II endoleaks is still subject to research, the association with continuing aneurysm growth and increased risk of rupture and their impact on management is debated. Type II endoleak is the most common type and has been reported to occur in 14% after EVAR at 1 month. The prevalence decreases to 10.2% after 1 year (6). Rupture rates of 2% for early type II endoleak and 6% for persistent type II endoleak have been reported in a recent study with or without aneurysm enlargement (7).

Common feeding arteries in type II endoleak are lumbar arteries, internal iliac arteries, the inferior mesenteric artery, accessory renal arteries, median sacral artery and other aortic side branches (6, 8). Risk factors for the development of type II endoleaks include size and number of patent branch vessels on preoperative CT (5), age, long aneurysm neck (6), thrombus thickness and percentage of circumference (8). The influence of endograft characteristics is controversial (5, 6).
Because endoleak can occur any time following EVAR, follow-up examinations are performed with periodic imaging. Evaluation of suspected endoleak is predominantly performed with CTA. However, due to radiation exposure and adverse side-effects of contrast agents, the optimal modality and acquisition protocols are still subject to discussion (1).

Biphasic acquisition of arterial and delayed contrast phases has been reported to increase sensitivity of endoleak detection (2). However, it has also been reported that the arterial phase could be eliminated for detection of endoleak (9). On the other hand Hong et al. reported that endoleaks detected only in the delayed contrast phase resolved spontaneously without intervention in a high percentage and that this contrast phase could therefore be eliminated from acquisition protocols to minimize radiation exposure (2).

Furthermore in cases of nonshrinking aneurysms and lacking evidence of endoleak in CT-angiography and delayed CT, MRI with a blood pool contrast agent has been reported to have a higher sensitivity to detect endoleaks. In a study by Cornelissen et al. endoleaks were found in more than 50%. The use of dual-energy CT for reconstruction of virtual unenhanced images has been reported to allow assessment of endoleaks with comparable accuracy to triple-phase CT or non-enhanced and delayed phase CT at considerably lower effective radiation doses (1). Perini et al. reported on the use of contrast-enhanced ultrasound for EVAR follow-up imaging. They found no significant differences in the accuracy of endoleak detection rates and measurements compared to CT imaging (10).

Management of type II endoleaks includes preoperative, intraoperative and postoperative endoleak management. The indications for type II endoleak interventions are subject to controversial discussion. The majority type II endoleaks will seal spontaneously and conservative treatment is thus justified in most cases (6).

Preoperative endoleak management includes embolization in cases of planned coverage of the internal iliac arteries, furthermore preoperative prophylactic embolization of the internal mesenteric artery and lumbar arteries has been reported.

Intraoperative injection of thrombin into the aneurysm sac immediately after initial endograft deployment was performed resulting in 2.4% type II endoleaks during a 2 year follow-up period. Another approach is to pack the aneurysm sac with gelatin sponge via an introducer sheath in cases of patent side branches (6).

Recommendations for postoperative endoleak management vary, usually intervention is performed when the endoleak persists more than 6 months or the aneurysm sac shows...
progression. Transarterial embolization of branch vessels using coils, glue, gelatin, onyx or thrombin has been associated with acceptable initial success rates. However, it can be technically challenging and the endoleak might recur due to communicating vessels or persistent flow through the coils. Techniques have been reported where the aneurysm sac, the feeding and the draining vessel are embolized following microcatheterisation (6). Translumbar embolization using coils, glue and thrombin has been reported. Several authors reported favourable long term results for translumbar embolization compared to transarterial embolization of inferior mesenteric artery (11, 12). Further techniques include transcaval embolization using thrombin, laparascopic clip ligation and open surgery, which should be reserved to very complex cases or failed embolizations. However, it has been reported that a significant number patient require more than one intervention, with coil intervention alone being a predisposing factor, or experience continued aneurysm sac growth. Therefore continued long-term surveillance is necessary (13).
Fig. 1: CTA of Type II endoleak following endovascular repair of abdominal aortic aneurysm. Unenhanced scan (A), arterial (B) and delayed phase (C). The patient presented with persistent endoleak type II. In CTA a prominent arc of Riolan was seen.

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Fig. 2: Transarterial embolization of type II endoleak (same patient as Fig.1). Microcatheterization of the arc of Riolan. Placement of the tip of the microcatheter (red circle) in the proximal inferior mesenteric artery (A), note contrast agent in aneurysm sac (black circle). Advancement of the tip of the microcatheter (red circle) to the ostium of the inferior mesenteric artery (B) and coil embolization.

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**Fig. 3:** Persistent type II endoleak following endovascular repair of abdominal aortic aneurysm. Unenhanced scan (A), arterial (B) and delayed phase (C). In a previous external angiography no major branch vessels feeding the endoleak could be depicted.

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**Fig. 4:** Sagittal reconstruction of delayed phase in persistent type II endoleak (A). Same patient as Fig. 3. Translumbar embolization using thrombin (B), coaxial system (arrow) is used for CT-guided puncture of endoleak.

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Fig. 5: Persistent type II endoleak in arterial phase CTA following transarterial coil embolization of inferior mesenteric artery (A). Translumbar puncture guidance using flat-panel detector CT B. Fluoroscopy showing the contrast agent flow inside the endoleak (C). Following injection of thrombin cessation of contrast agent flow is documented (D).
Conclusion

Type II endoleak is a common complication after EVAR. Imaging is commonly performed using non-contrast enhanced and arterial phase CT. Most type II endoleaks resolve spontaneously. Persistent endoleaks over 6 months and those with increasing aneurysm size should be treated. A variety of treatment options exist. The choice of treatment option should be performed based on imaging findings and experience as no management consensus exists.


