Endoleak Management after Endovascular Aortic Repairs (EVAR/ TEVAR)

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Endoleaks after stentgraft

The principle of EVAR/TEVAR consists in the exclusion of the aortic aneurysm) by endoluminal sealing using a stent graft. Due to the nature of procedure, the crevice between the stentgraft and the aortic wall is inevitable. Moreover, the sac of the aneurysm surrounding the stentgraft is left as is even after the procedure. An endoleak is defined as transmission of the blood pressure or residual blood flow within the aneurysmal sac, which can occur in approximately one fourth of the cases after EVAR1). The endoelaks occurring after EVAR are known to be related to late aneurysmal rupture[1)].

There are five types of endoleaks. Type I is defined as residual blood flow that originates from a stentgraft attachment site. Type II is defined as retrograde blood flow through aortic branch vessels into the aneurysm sac. Type III is caused by structural failure of the stentgraft, which includes subtypes III-a and III-b, which correspond to stentgraft holes in the fabric and junctional leaks in modular devices, respectively. Type IV is a endoleak caused by stentgraft porosity. Type V can be explained as expansion of the aneurysm without presence of an apparent endoleak, commonly known for "endotension". The exact cause of endotension is still unknown **(Fig 1)**.

While digital subtraction angiography is the standard method for differentiating endoleaks, time-resolved dynamic contrast CT and/or MRI have also been reported to be useful[2)]. However, the application of MRI is limited by materials because of potential metallic artifact.

Endoleak management

Type I and III endoleaks are classified as high pressure endoleaks, which should be fixed by the end of each procedure. Although type I and III endoleaks occasionally resolve spontaneously, these are highly associated with sac growth or rupture if they persist during follow-up. Therefore, persistent developing type I or III endoleaks should be treated as soon as possible[3)]. The treatment of type I and III endoleaks includes touch-up with balloons or additional stentgrafts to extend the proximal or distal sealing zone. While type I endoleaks from distal sealing zone (type Ib) can be

treated with distal extension of the stentgraft with or without embolization of the internal iliac artery, stentgraft extension in the treatment of Type I endoleaks from the proximal side (type Ia) is often challenging due to the visceral arteries to be preserved. More complex procedures such as the chimney technique, fenestrated stentgraft, surgical explantation, or proximal neck banding may be necessary.

Evidence regarding type II endoleak

Type II endoleaks occur in about 40% of cases after EVAR during the early follow-up period, and they may be resolved spontaneously within 6 months. Accordingly, they are considered fundamentally benign[4]. However, sac growth can be observed in 24% of persistent type II endoleaks over the 6-month follow-up period, in which case the endoleaks may not necessarily be benign[4]. The cohort with persistent type II endoleak shows significantly higher incidence of sac growth than that without type II endoleak[5]. However, rupture of the aneurysm due to type II endoleaks is extremely rare (0.4%)[6]and the morality rate is still not affected by the presence of Type II endoleak[5]. Therefore, around 1-3% of type II endoleaks occurring after EVAR require close attention. Longitudinal elongation of the sac due to type II endoleak can cause type I and III endoleaks. Based on these evidences, the treatment indication of the endoleak in consensus is a residual type II endoleak over 6 months with significant sac growth. Large aneurysms exceeding 6cm in diameter without sac growth can be indicated in the treatment. The threshold of the sac growth is defined as 5mm or 1cm interval change in the maximum diameter.

Treatment option for Endoleak

There are many interventional options for the treatment of type II endoleak. The endovascular treatment includes transarterial embolization and percutaneous direct puncture. Other endovascular options include perigraft access wherein a catheter is retrogradely introduced into the sac via a space between the stentgraft and iliac arterial wall[7]. There are also the transcaval and transgraft approaches, wherein a needle catheter is inserted into the sac through the wall of the IVC or the fabric of the stentgraft, respectively[8].

Surgical options include ligation of aortic tributaries under laparotomy or laparoscopically, plication of aortic aneurysm, and explantation of the stentgraft; however, these are highly invasive when compared to endovascular options.

Transarterial embolization

Transarterial feeder occlusion has been widely accepted for type II endoleaks after EVAR; However, feeder occlusion has now proven to be associated with the recurrence of endoleak and sac expansion even after embolization. These unsatisfactory results are thought to be due to collateral supply from multiple

sources[9]. Therefore, occlusion of both intrasaccular channels and feeding arteries is required for the successful treatment of type II endoleaks. Recent application of the double coaxial technique can easily enhance intrasaccular access, and acceptable results have been reported[10]. The ideal goal of embolization should be eradicating all inflows or outflows. The embolic materials to be used are coils and liquid embolic agents like NBCA-LPD or Onyx. NBCA is a low-cost embolic material, but the precise control of its distribution is often difficult. Embolization for longer durations increases risks related to catheter adhesion. Onyx can fill the aneurysmal sac more easily, but a relatively large amount is often required for successful occlusion of the aneurysmal sac, which can cause significant beam-hardening artifact on follow-up CT examinations and increase the cost[11].

The frequent feeders of type II endoleaks includes the inferior mesenteric artery (IMA) and lumbar arteries, which are followed by the middle sacral artery and accessory renal artery. Common access arteries are the IMA via Riolan's arch from the superior mesenteric artery (SMA) or lumbar arteries via the iliolumbar artery from the internal iliac artery. Uncontrolled liquid embolization should be limited because severe adverse such as mesenteric ischemia or neurological complication can occur in the embolization from the IMA or from the lumbar artery, respectively.

Percutaneous embolization

Percutaneous direct embolization includes a translumber approach via the perivertebral space in the prone position and a transabdomnal approach via the peritoneal cavity in the supine position. These approaches provide easier access to the endoleak cavity and enable occlusion of the entire sac compared to the transarterial approach. Technical success of percutaneous embolization has been obtained in around 90% of cases, and clinical success rates of translumbar embolization reported varied from 25% to 92%. A limitation of the percutaneous approach is that the placement of the needle within the endoleak is difficult due to the anatomical setting among the surrounding organs, stentgraft and endoleak cavity. Another drawback of the translumbar approach is a potential risk of hemorrhagic complication during the procedure. Other complications have includeding migration of embolic materials causing bowel necrosis, or aneurysm rupture into the inferior vena cava have also been reported[12, 13].

Tips for liquid embolization

Uncontrolled embolization using liquid embolic materials for type II endoleaks potentially causes bowel necrosis or neurological complications. Unintended occlusion of the Riolan's arch or the superior rectal artery can cause bowel ischemia because these arteries are major feeders for the rectum after EVAR. Occlusion of the inferior mesenteric artery should be confined to the segment proximal to the orifice of the

first bifurcation. Untargeted embolization of the segmental artery can cause neurological complications. Since the communication to the spinal artery or radicular arteries can originate distally to the bifurcation of dorsal branch, the embolization should be confined to the proximal somatic part of the segmental artery even with non-visualization of the spinal artery.

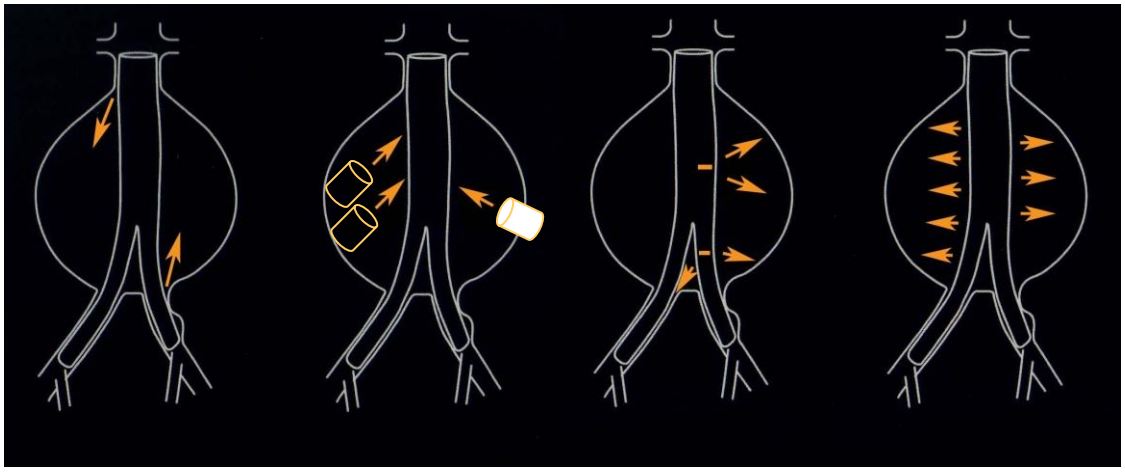
Prophylactic embolization for type II endoleak

Prophylactic embolization of aortic tributaries for type II endoleak can be considered if the embolization after EVAR is difficult. The typical target is the internal iliac artery prior to the EVAR with the extension of the external iliac artery. Coils or vascular plugs are frequently used in the embolization of internal iliac artery. If the internal iliac artery is not dilated, embolization should be conducted at the proximal segment of the internal iliac artery, and the communication of the superior and inferior gluteal arteries should be preserved because an excessive embolization of the distal tributaries of the internal iliac artery is related to buttock claudication.

The accessory renal artery is also a subject for prophylactic embolization. There haves been several reports of prophylactic embolization of the IMA or lumbar arteries diminishing the incidence of the occurrence of type II endoleaks. However, these procedures still remain controversial in terms of its costs, risks, and benefits, because efficacy for preventing sac growth or
rupture has not yet been proved[14].

Conclusion

While a majority of cases with type II endoleaks can be observed conservatively, some type II endoleaks can contribute to sac growth after EVAR, and this fact substantially compromises the durability of EVAR for AAA. However, type II endoleaks are not life-threatening diseases, and they should be fixed in safe and effective ways.



Endoleak type	Descripton
I	Attachment-site leak—proximal or distal
II	Collateral-vessel leak
III	Graft failure—midgraft hole, junctional leak, or disconnect
IV	Graft-wall porosity
V	Endotension

Figure 1. Types of endoleak

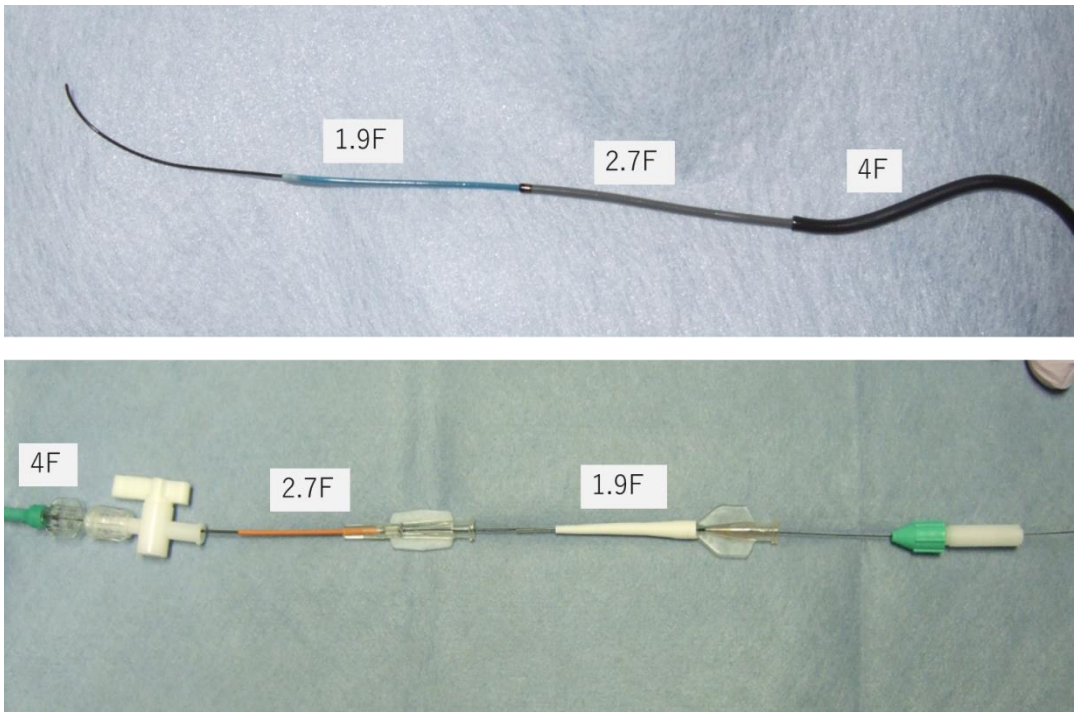


Figure 2. Double coaxial microcatheter system

The system is comprised of a 1.9F non-tapered microcatheter and a 2.7F high flow microcatheter, which can be introduced through a 4F diagnostic catheter.

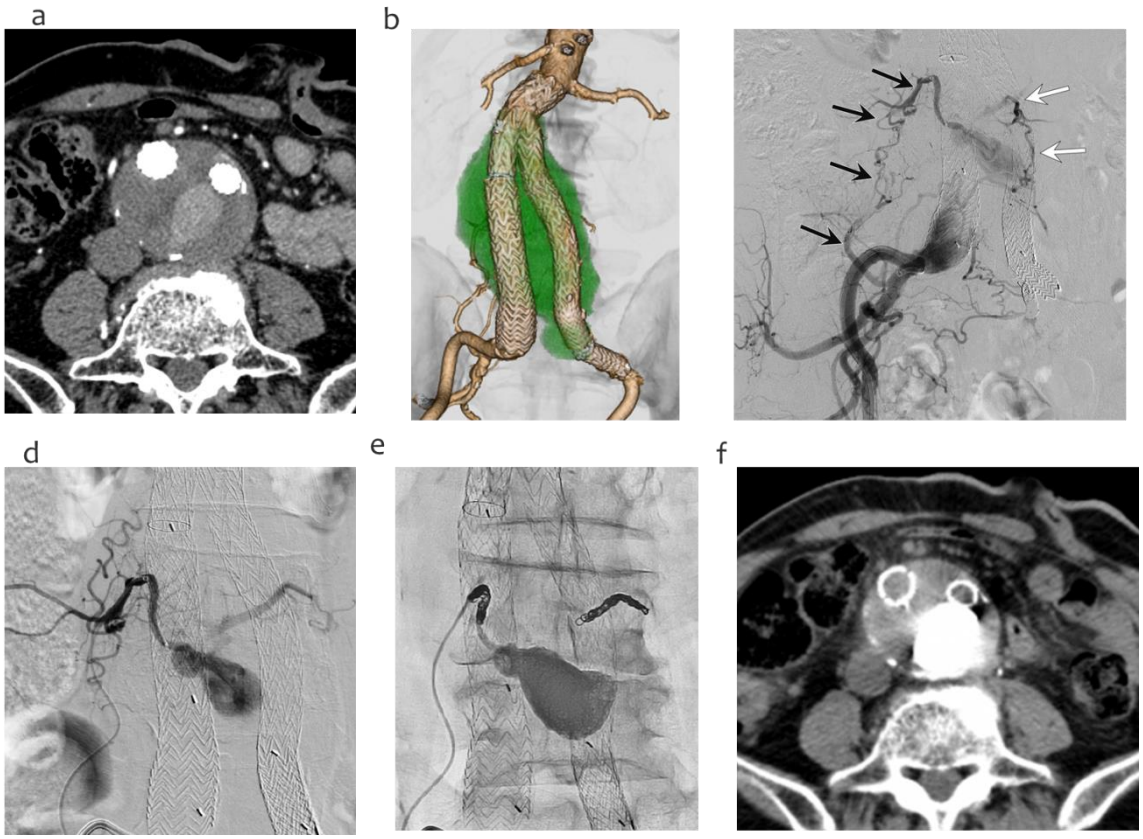


Figure 3. A man in his 80's with a type II endoleak with a growing aneurysmal sac following EVAR.

(a,b) Postcontrast CT obtained 6 months after the EVAR showed an AAA of 57 mm in diameter with persistent type II endoleak supplied by the right iliolumbar artery.

(c) Right internal iliac arteriogram showed type II endoleak supplied by the right 4th lumbar artery via tortuous collaterals (black arrows) from the right ileolumbar artery. The left 4th lumbar arteries were visualized via the aneurysmal sac.

(d) Right 4th lumber arteriogram showed an endoleak channel within the sac and the left 4th lumbar artery as the outflow.

(e) Fluoroscopic image immediately after the selective embolization of lumber arteries using coils and intrasaccular injection of 9.8mL of 25% NBCA-LPD

(f) Precontrast CT after endoleak embolization showed complete replacement of the type II endoleak by NBCA-LPD.

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