What is the significance of endoleaks and endotension

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The primary purpose of endovascular aneurysm repair (EVR) is to prevent death from aneurysm rupture. Endoleaks are defined as the persistence of blood flow outside the lumen of the endoluminal graft but within the aneurysm sac [1–3]. Thus, the aneurysm sac is not completely excluded from the systemic circulation, and blood may fill the sac either from around the graft (type I) or as back bleeding through small vessel arising from aneurysm sac (type II). Often the pressure in the aneurysm sac in patients with an endoleak is the same as systemic arterial pressure [4], raising concerns of possible aneurysm enlargement and rupture. Endoleaks can be identified in 15% to 52% of patients after endovascular repair [5–13].

Endotension is defined as pressure within the aneurysm sac without evidence of endoleak as the cause. The aneurysm may increase in size under these circumstances. Endotension with sac enlargement raises the possibility of an aneurysm rupture. The phenomenon of endotension is unusual, and is seen in only 2% to 5% of patients after EVR [11,13–15]. At present, the significance of endoleaks and their relationship to aneurysm enlargement and rupture is unclear. Some authors conclude that reduction in the size of the aneurysm is the only absolute indication of stent graft treatment success [16]. However, aneurysm rupture can be seen in aneurysms that decrease in size [14]. Here, we have reviewed the current literature on endoleaks and endotension in relation to their influence on the primary outcome measures of EVR, which are aneurysm-related death and aneurysm rupture.

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Classification and incidence

A widely accepted classification of endoleaks divides endoleaks into four categories according to the origination of flow into the aneurysmal sac [2,3,17]. Type I endoleaks are those in which flow into the aneurysm sac originates at a stent graft attachment site to the infrarenal neck or iliac arteries (Fig. 1). Separation of the space between the arterial wall and the stent graft allows the direct flow of blood from the aorta into the aneurysm sac. Type I endoleaks can be divided further divided into type IA endoleaks, which occur at the aortic neck attachment site, and type IB endoleaks, which occur at the distal iliac attachment sites. Type II endoleaks are those in which blood flows into the aneurysm sac in a retrograde direction through normal branches of the excluded segment of the aorta (Figs. 2 and 3). The most common branches that give rise to type II endoleaks are the inferior mesenteric artery and lumbar arteries. Accessory renal arteries can also give rise to type II endoleaks. In subgroup type IIA endoleaks only, one branch vessel can be detected. In more complex type IIB endoleaks, flow can be seen through two or more branch vessels. Type III endoleaks occur when there is a structural failure of the endovascular device. Type III endoleaks can be subdivided into three groups: type IIIA endoleaks are due to disruptions or holes in the fabric of the device, type IIIB endoleaks arise from separation of modular devices and junctions, and type IIIC endoleaks are due to suture holes in the fabric. Type IV endoleaks are caused by graft porosity, and is usually identified on completion of angiography at the time of implantation while the patient is fully anticoagulated.

Endotension indicates the there is tension exerted on the aneurysm wall with or without an endoleak. The exact cause of endotension, when no
endoleak can be found, is unknown, but some authors think it is a missed or sealed endoleak [7,18,19]. However, there are reports of endotension patients who have undergone surgical conversion for aneurysm enlargement without any evidence of blood inside the aneurysm sac [20]. Pressure may remain high in the excluded aneurysm sac due to transmission of the pressure through thrombus at or around the ends of attachment zones of the prothesis [21,22]. This has been particularly associated with angulation of the proximal aspect of the graft [20]. It has also been suggested that an infectious process may be the cause of endotension, although the mechanism is not clear [23,24]. Endotension has also been noted as a late phenomenon several years after EVR [20].

The incidence of type I and II endoleaks at the time of discharge following endovascular aneurysm repair ranges between 4% to 7% and 27% to 37%, respectively [25,26]. The incidence of type I endoleaks varies between 4% to 6% at 1 month, between 1% and 4% at 12 months, and between 1% to 3% at 24 months follow-up. The incidence of type II

Fig. 2. Type II endoleak is visualized with Doppler ultrasound (top) and computer tomography (bottom).
endoleaks are 8% to 12% at 1 month, 5% to 32% at 12 months, and 13% to 26% at 24 months [23,26–28].

**Natural history of aneurysms with endoleak**

The natural history of aneurysms with endoleak after EVR remains poorly defined. Aneurysm sac enlargement is more often seen in patients with endoleak compared with those without an endoleak [4,14,29]. Sac enlargement has been found in 20% of patients with type I/III endoleak compared with 10% of patients with type II endoleak and 5% of patients with no endoleak [4]. However, aneurysm enlargement following endovascular repair may not always be associated with an increased risk of aneurysm rupture [14]. Also, there have been reports on patients with shrinking aneurysms with no endoleaks with aneurysm rupture [30–33],
although type I/III endoleak has been found to be associated with aneurysm rupture [8]. These ruptures have more often been seen in tube grafts or first generation grafts, which were more prone to total graft displacement, graft fractures, and severe kinking [4,9,34]. In the EUROSTAR study of 2862 patients, the risk ratio for late aneurysm rupture for proximal type I endoleak was 7.59 \((P = 0.001)\) and for type III endoleak 8.95 \((P = 0.001)\) [35]. However, distal type I endoleak was not significantly associated with rupture \((P = 0.8)\). In a study of 398 patients who had undergone EVR with the AneuRx stent graft, that the presence or absence of an endoleak did not predict patient survival or aneurysm rupture rate after EVR [16]. Thus, it was felt that the endoleak in and of itself was not a predictor of the primary outcome measure after EVR.

Type II endoleaks are the result of continued blood flow within the aneurysm sac from the inferior mesenteric artery (IMA) or lumbar arteries. Type II endoleaks are often seen immediately after placement of an endovascular device, and are more likely to be seen and persist in patients with large IMA or multiple lumbar arteries [36]. The long-term significance of type II endoleaks is unknown. The risk of aneurysm rupture in patients with pure type II endoleak is not increased compared with patients with no endoleak [4,14]. In a comparison of three groups of patients with type II endoleaks—those with a persistent type II endoleak, those with a transient (<1 month) type II endoleak, and those with no endoleak—Arko et al found that aneurysms with a persistent type II endoleak increased in diameter and volume compared with the two other groups, both of which decreased [36]. There were no aneurysm ruptures in patients with type II endoleak and no correlation between type II endoleaks and stent graft migration, proximal neck enlargement, or aneurysm. Although aneurysm rupture in patients with type II endoleak has been described [37], the overall risk of rupture has not been found to be increased in patients with a pure type II endoleak compared with patients with no endoleak [4].

Intrasac Doppler flow velocities can be used to predict if type II endoleaks will spontaneously seal [38]. Low-velocity endoleaks (<80 cm/s) have been found to be more likely to seal spontaneously compared with high velocity (>100 cm/s) endoleaks. High-velocity endoleak is often related to large-branch vessel diameter or number, and are also often resistant to treatment with transarterial embolization [38].

In the EUROSTAR registry, 91 patients have been reported to have endotension, and no aneurysm ruptures were seen during the mean follow-up of 15.4 months [4].

**Risk factors and prevention**

Short or angulated infrarenal aortic necks are the most significant preoperative risk factors for a type I endoleak [4,39,40]. A neck length less
than 20 mm is significantly more likely to result in a proximal type I endoleak compared with longer neck lengths [40]. Large-diameter aortic necks (>28 mm) can lead to an endograft migration and endoleak [40]. Smaller amounts of mural thrombus in the aneurysm sac also have been found to correlate with device-related endoleaks. Ouriel et al found in a study of 700 patients that patients with a large (>5.5 cm) aneurysm more often developed a late type I endoleak than patients with a small aneurysm [41]. There was no difference in frequency of the type I endoleak seen on intraoperative angiography or postprocedure CT scans. Shames et al compared the endoleak rate between transrenal and infrarenal endograft fixation [42]. They found no difference in the endoleak rate between these two, and concluded that patient selection is more important than the type of proximal fixation in preventing endoleaks.

The most important issue in preventing a type I endoleak is careful patient selection. Aortic neck dimensions and quality of proximal and distal fixation sites are the most critical factors. Patients should have infrarenal necks longer than 15 mm, a common iliac artery diameter smaller than 18 mm, with minimum continuous length of 15 mm. Other factors that must be evaluated are neck angulation, tortuosity and transmural calcification, and thrombus. Patients with an aortic neck less that 15 mm in length should be treated with caution because of concern about security of proximal fixation and a type I endoleak. Furthermore, the aortic neck contour and angulation are of particular concern because angulation increases the risk of a type I endoleak, particularly when neck length is short. Security of the proximal endograft, graft fixation, is the most important factor in preventing acute and late type I endoleaks (Fig. 4). Thus, it is recommended that the

Fig. 4. Example of poor proximal fixation. Optimal place for proximal stent graft is immediately under renal arteries (white dashed line). Insufficient proximal fixation leads to migration and a type I endoleak in many cases.
endograft be placed as close to the renal arteries as possible and extended to the hypogastric arteries. The use of suprarenal fixation may be beneficial in patients with short and angulated necks to improve proximal fixation. Type I/III endoleak can sometimes occur after mechanical trauma. Krajcar and Dougherty (2003) reported four patients in whom mechanical trauma was identified as a factor in the development of late complications after EVR [43]. Two patients had sustained blunt abdominal trauma in a car accident, one had suffered a traumatic fall, and one had been participating in vigorous rowing activity. However, a clean relationship between traumatic events and endograft complication has not been established.

A type II endoleak is more likely to occur and persist if there is patent IMA or two or more patent lumbar arteries. To avoid type II endoleak, IMA or large lumbar arteries can be embolized before stent grafting [15,16] (Fig. 5). However, because the risk of an aneurysm rupture with a type II endoleak is so small, preoperative embolization is rarely performed.

Fig. 5. Preoperative coil embolization of patent inferior mesenteric artery (IMA). Preoperative angiogram shows patent IMA (white arrow, top left), which has been selectively cannulated (top right) and coil-embolized (bottom). After embolization, endografting is performed as usual.


**Diagnosis**

The diagnosis of an endoleak is based on the observation of contrast medium or blood flow within the aneurysm sac on angiography, computer tomography (CT), or duplex scanning. In the operating room after EVR, completion angiography is performed. A high-flow power-injected run and filming through the venous phase usually shows if there are any endoleaks present. When an endoleak is seen, further analysis on the type of endoleak is needed. With selected injections near attachment sites and with multiple projections, the source of the endoleak usually can be found. Sometimes, if there are patent lumbar arteries an attachment site leak can mimic a type II endoleak. Thus, careful evaluation of the direction of flow in the branch vessel will show the true source of the leak. Sometimes contrast material does not disappear from the aneurysm sac until after the first postoperative day. Thus, postprocedure CT scans can mimick an endoleak, due to residual contrast material in the aneurysm sac from the previous day’s angiograph procedure and endograft placement. A noncontrast CT scan will identify such a circumstance (Fig. 6).

Duplex ultrasound (US) and spiral CT are the most important tools for recognizing endoleaks during postoperative surveillance [44,45]. High-quality duplex US scanning has been found to be comparable to CT angiography for the assessment of aneurysm size and endoleaks after EVR [45]. As an adjunct to CT in evaluating endoleaks, duplex US provides hemodynamic information that enables further characterization of the type of endoleak [44,45]. CT and US are more sensitive than angiography for the detection of endoleaks [46]. Angiography can be employed selectively to define the source of the endoleak when it is uncertain and when further procedures are considered.

With CTA, the direction of the flow cannot always be identified. Thus, the type of endoleak that is present might be misdiagnosed. Time-resolved magnetic resonance angiography (TR-MRA) has been found to be as sensitive as CTA for detection of endoleaks [47,48]. Furthermore, TR-MRA

![Fig. 6. Contrast material can be sometimes seen in the aneurysm sac at first postoperative day mimicking endoleak (left), but image without contrast shows that there is no endoleak (right).](image)
can visualize the direction of flow in the aneurysm sac, making the endoleak characterization more accurate with TR-MRA than CT [49]. However, MRI imaging is limited to patients with nitinol-based supported or unsupported devices, and cannot be used in patients with a pacemaker. Patients with stainless steel devices are unable to be followed with MRA because of the significant metallic artifact caused by the stainless steel components. Similarly, patients who have undergone embolization procedures with stainless steel coils may be difficult to follow with MRA because of the artifact from the coils [50]. Platinum embolization coils are nearly invisible for MRA, and create no artifact [49].

Endotension can cause sac enlargement, and the diagnosis is made by measuring the intrasac pressure. Intraaneurysm sac pressure assessment is performed using direct translumbar access through the aneurysm sac or selective cannulation of the inferior mesenteric or lumbar arteries via either the superior mesenteric artery or hypogastric arteries, respectively [20,51].

Treatment

The primary objective in treating patients with aortic aneurysms is to prevent aneurysm rupture and death from aneurysm rupture. Thus, treatment of the endoleaks should be based on that same basic principle: if the endoleak increases the risk of rupture, it should be repaired as soon as possible, but the risk of death or severe complications of the chosen treatment should never exceed the risk related to the endoleak itself. Unfortunately, decision making is seldom straightforward due to several factors. The difficulty lies in the fact that the true natural history of aneurysms with an endoleak is poorly understood, and there is not a clear consensus on treatment guidelines. Also, predicting the risk associated with secondary intervention for each individual patient is difficult. The risk of aneurysm rupture depends primarily on the size of the aneurysm [14], the pressure within it, and the force applied to the aneurysm wall [52]. The clinical consequence of rupture, however, depends on the potential for life-threatening hemorrhage. The amount of hemorrhage in the case of an aneurysm rupture should depend on the flow rate to aneurysm sac, that is, the endoleak volume. Thus, clinical significance of the endoleak or endotension is based on the risk of rupture and on the possibility of life-threatening hemorrhage if rupture occurs.

Most endoleaks will seal spontaneously without any treatment. Two of three endoleaks that are seen at the time of the discharge seal during the first month after the procedure [8,16]. Arko et al found that low velocity type II endoleaks (<80 cm/s in Doppler ultrasound) were likely to have thrombosis without treatment [38]. However, 20% of leaks that spontaneously seal reopen at 12 to 18 months [53]. New type II endoleaks can occur at any time.
after the procedure. The timing of endoleak occurrence does not significantly influence the rate of spontaneous endoleak resolution [54].

Type I/III endoleaks have been found to be associated with aneurysm rupture in several studies [4]. Thus, treatment of these endoleaks is indicated.

If a type I endoleak is recognized at completion of angiography, it should be treated during the initial operation with endovascular techniques, if possible. If the endoleak is from the proximal attachment site, balloon angioplasty, Palmaz stent, or stent graft extension to the proximal site will often eradicate the leak. Sometimes, however, a proximal leak is resistant to immediate procedures, and in these cases it can be followed some time after operation, because most of these endoleaks seal spontaneously [8].

With the development of a late type I endoleak, there often can be treatment with additional stent graft modules to improve the proximal fixation. There should be sufficient native aorta available proximal to allow placement on an extender module. Type I endoleaks have been treated with coil or glue embolization with fairly good results [47,55–58]. At the same time of the embolization of the perigraft space, patent outflow vessel, including lumbar arteries and IMA, can be embolized [59]. However, recanalization of the endoleak site or transmission of systemic pressure through thrombus has been seen [57]. Acute complications, such as colon ischemia, has been reported in the treatment of a type I endoleak with glue [57,60].

When there seems to be a proximal endoleak despite proximal restenting, periaortic ligature can seal the leak [61,62]. Although aortic clamping can be avoided in this procedure, it is more invasive than the above described endovascular techniques, and 30-day mortality has been even 30% [61].

Persistent type I endoleaks resistant to endovascular repair may require open surgical conversion. However, surgical conversion is associated with a high (20%) mortality rate [35,63,64]. Patients who are at especially high risk for open surgery may, on occasion, be observed with careful, ongoing monitoring. However, the ongoing potential for aneurysm rupture must be recognized.

Although the long-term significance of a type II endoleak is unknown, thus far they have been associated with low risk of an aneurysm rupture [4,38]. However, patients with type II endoleaks associated with an aneurysm enlargement are of concern, and should be treated and carefully observed.

Endovascular treatment of a type II endoleak includes selective catheterization of feeding vessels and embolization with glue or coil [65]. (Fig. 7). Another method is to use a translumbar approach via either a left-sided or right-sided (transcaval) approach [66]. Type II endoleaks are often resistant to embolization, and new type II endoleaks can occur after treatment [67,68]. Baum et al compared transarterial and direct translumbar embolization in the treatment of a type II endoleak [69]. In their series, 80%
(16 of 20) of endoleaks treated with transarterial embolization were unsuccessful with recanalization of the original endoleak cavity over time compared with 8% (1 of 13) in the translumbar group during the median follow-up of 8 months ($P = 0.0001$). High velocity (>100 cm/s in Doppler US) type II endoleaks are more persistent and also resistant to coil embolization [38]. A lower recurrence rate can be achieved if the entire aneurysm sac is glue- or coil-embolized [69]. Also, thrombin injection into the leak by a percutaneous approach directly into the aneurysm sac using Doppler US has been described [70]. A more effective, but also more invasive, treatment method is feeding vessel ligation or clipping, which can be performed using laparoscopy or laparotomy [71–74].

Treatment of type II endoleaks is not complication-free, and severe consequences, although rare, have been associated with migration or distal embolization of the agent being used. Paraplegia, colonic necrosis, and ischemic colitis subsequent to embolotherapy for type II leaks have been reported [75–77].

A type III endoleak increases the risk of aneurysm rupture significantly [8]. With the first-generation endografts, severe migration has been a relatively common problem, causing kinking and graft separation followed by a type III endoleak. In many cases, these can be treated with an extender cuff or an entirely new endograft inside the old one [78]. If endovascular means are unsuccessful or are not appropriate, then open conversion is indicated.

Type IV endoleaks, which are seen at the time of completion of angiography when the patient is fully anticoagulated, are self-limited, and no treatment is needed [79].

In the case of endotension, the clinical consequences of rupture are unclear. Some authors favor open surgical conversion in the case of endotension and aneurysm enlargement [20]. Endotension is clinically

Fig. 7. Patent inferior mesenteric artery has been selectively cannulated (left) and successfully coil-embolized (right).
significant if associated with the potential for hemorrhage in the event of aneurysm rupture, or if it causes progressive enlargement of the aneurysm and aneurysm neck so that the security of endograft fixation is compromised [53]. Because of the absence of an endoleak, the only treatment method in the case of endotension is open surgical conversion. However, the risk of death and severe complications of open repair may be greater than the risk of observation of the endotension. Our conclusion from currently available data is that open conversion in endotension is rarely justified. In the case that the aneurysm enlargement leads to compromised proximal fixation and a type I endoleak, treatment is indicated.

Summary

Endoleaks remain a challenging issue following endovascular aneurysm repair. Careful patient selection is the most important factor in preventing endoleaks. The natural history of aneurysms with different types of endoleaks is unclear, and endoleaks may not be reliable indicators of the success of endovascular repair. The primary objective of aneurysm repair is prevention of aneurysm rupture, and endoleaks are only indirect measures of success. An endoleak, however, is a good secondary outcome measure after EVR. Treatment of type I and III endoleaks is indicated, and should be done with minimally invasive technique, if possible. If open conversion is required, risks and benefits must be carefully evaluated. Type II endoleaks are associated with aneurysm enlargement, but may not be indicators of an increased risk of rupture.

References


