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ENDOVASCULAR INTERVENTIONS FOR ENDOLEAKS

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ЕНДОВАСКУЛАРНО ЛЕЧЕНИЕ НА ЕНДОЛИЙКОВЕ

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Abstract. Endoleak represents the most common complication after endovascular aortic aneurysm repair (EVAR) and is defined as persistent perfusion of the aneurysmal sac, which subsequently could lead to its expansion and possibly rupture. There are different types of endoleaks, depending on their inflow source, regardless of the number and type of other vessels involved in the outflow (endoleak type I A/B/C, type II, type III, type IV and type V). The current gold standard for the diagnosis of endoleak is the contrast-enhanced helical computed tomography (CT). Since, there is no generally accepted consensus for the best surveillance and treatment methods for this pathology, in this article we will present the most effective endovascular interventions for the successful management of the different types of endoleaks.

Key words: endovascular aortic aneurysm repair, endoleak, treatment, embolization, computer tomography

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Резюме. Ендолийкът е едно от най-честите усложнения при ендоваскуларното лечение на аортни аневризми и представлява персистираща перфузия на аневризмалния сак, което може да доведе до неговото експандиране и евентуално руптуриране. Има различни типове ендолийкове в зависимост от мястото на захранване с кръв, независимо от броя и вида на другите ангажирани съдове (ендолийк тип I A/B/C, тип II, тип III, тип IV и тип V). Златният стандарт за диагностицирането на ендолийк е компютърната томография (КТ). Тъй като няма общоприет консенсус за лечение на тази патология в тази статия представяме най-ефективните ендоваскуларни методи за справяне с различните видове ендолийкове.

Ключови думи: ендоваскуларно лечение на аортни аневризми, ендолийк, лечение, емболизация, компютърна томография

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Introduction

Endoleak is defined as persistent perfusion of the aneurysmal sac after endovascular aortic aneurysm repair (EVAR) and it represents the most common complication. Endoleak is artificially created state/disease, which leads to a not complete exclusion of the aneurysmal sac from the systemic circulation [1, 2]. This persistent perfusion has been observed in 15% to 21% of the clinical trials for EVAR, but overall, in the literature the incidences reported are up to 50% [2, 3]. According to a report from EURO-STAR registry early endoleak incidences were 18% and sixty-nine percent of these leaks were graft-related [4]. Even though, endoleak presents a common complication it is important to state that most of the endoleaks resolve spontaneously during the first 6 months [5]. Similar data comes from the EURO-STAR registry, where seventy percent of the endoleaks sealed spontaneously during the first 6 months. Still, there is no rational explanation on the cause of spontaneous resolution of some endoleaks and persistent or late occurrence/reoccurrence in others [2, 4]. This phenomenon could be possibly explained with the presence of outflow vessels (mainly lumbar arteries and inferior mesenteric artery) and a leak communicating with them could disap-

pear spontaneously [4]. Since, endoleak is sometimes associated with continuing aneurysmal dilatation or rupture, a prompt monitoring and treatment of this pathology is crucial [6]. We can classify the endoleaks into primary endoleaks, which appear within 30 days post-procedure and secondary endoleaks, which are detected more than 1 month after procedure and after previous negative imaging. Primary and secondary endoleaks may develop due to graft-related factors, such as fabric defects, graft porosity, endotension or inadequate seals at the proximal and distal attachment sites or between endograft components in the case of a modular endograft. Another source of endoleaks could be collateral flow through the aneurysmal sack due to lumbar, mesenteric, or iliac artery collaterals. This type of endoleak is sometimes referred as peri-graft flow and also, can be primary or secondary in nature [7]. The generally accepted classification of endoleaks is defined by their inflow source, regardless of the number and type of other vessels involved in the outflow [2]. Numerous imaging modalities are available to detect and characterize endoleaks, but the gold standard for the diagnosis is contrast-enhanced helical computed tomography (CT). Pre-contrast scan followed by an arterial and delayed phase study is performed, with endoleaks best appreciated on the delayed phase (Figure 1). The common imaging protocol, which we also use is CT angiography at 1, 6, and 12 months, and annually thereafter. In the setting of aneurysm sac shrinkage and absence of an endoleak, some patients may be followed every 2 years. There is no generally accepted consensus for the best surveillance and treatment methods of endoleaks. In this article, we present the different types of endoleaks and review the different endovascular treatment options.

Type I endoleak

Type I endoleak is presented by leakage at the graft attachment either above, below, or between graft components. It is basically caused by failure to achieve a circumference seal proximally (type IA) or distally (type IB) (Figure 2). There is a type IC endoleak, which is present in patients with aorto-mono-iliac stent and femoral-femoral bypass and it is due to non-occluded iliac artery [1, 2]. Type I endoleak occurs in up to 9% of the patients treated with EVAR. Early type IA endoleak is a common complication (30%) after snorkel/chimney EVAR technique, with high spontaneous resolution in up to 71.8% at 12 months and a low reintervention rate at 3.3% [8]. In the settings of type I endoleak the aneurysm is perfused from the aorta or the iliac arteries. The leak usually communicates with the aneurysmal sac through a channel and there are outflow vessels (lumbar arteries and inferior mesenteric artery (IMA)), which normally communicate with the channel or the sac. Since the pressure from the endoleak type I is systemic, the tension to the aortic wall remains high. The etiology of a primary type I endoleak include unsuitable anatomy, with significantly angulated neck, calcifications in the distal/proximal landing zones, mispositioning of the stent graft and under dilatation of the stent graft. Progressive dilation or migration of the proximal neck leads to aneurysmal remodeling and subsequent secondary type I endoleak. The incidences of secondary endoleak among type I endoleaks is 2.2 to 15% [2, 8].

Since, type I endoleak presents a high risk of rupture (in up to 50%) a reintervention is indicated. The risk of rupture is even higher if there is a combination between type I and type III endoleaks (high pressure leaks) [2, 8]. There are different methods of treatment and the best approach is based on the source of the leak (Table 1). Management of this type of endoleak, requires clear understanding of the mechanics, and the risk of sac enlargement and rupture due to the increased sac pressure. Endovascular techniques are the main treatment approach to manage secondary endoleaks. However, surgery should be considered where endovascular treatments fail to arrest aneurysm growth.

As stated above, endovascular management of type I endoleak is mandatory, because of the docu-

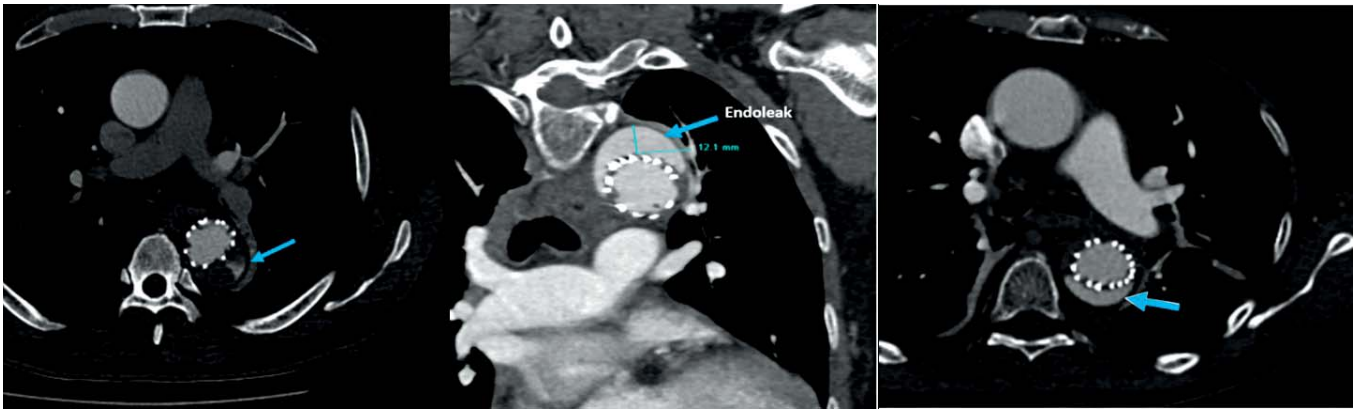

Fig. 1. CT scans of different endoleaks

mented high rupture risk. In the presence of type IA endoleak there are multiple options for reintervention. The early detection of the endoleak is crucial, because the technical success rates are high when managed intra-procedural or early after the initial implantation. The most used technique for the management of type I endoleaks is the placement of a proximal extension endograft, mainly in the cases associated with mispositioning, angulated neck or migration. In the cases, where there is no migration of the stent graft, a simple balloon angioplasty with large balloons (25 to 30 mm) or large Palmaz stent placement could be enough to enclose the stent graft to the aortic wall. Alternative treatment in selected patients can be embolization and coiling of the aneurysmal sac and the outflow vessels. Even though there were some doubts in the success rate of these procedure, numerous studies reported positive and encouraging results [1, 2, 8]. Gorich et al., report 13 successfully treated patients and Sheehan and colleagues have a high clinical success rate in 9 patients treated with coil embolization [9, 10]. Golzarian et al., treated 32 patients with type I endoleak from 1996 to 2003. Embolization was successful in 29 of the patients with the occlusion of the outflow vessels and the aortic channel and/or sac. In 3 of the patients the endoleak persisted, even after several procedures [2]. Since, none of the patients with successful embolization had developed a new endoleak, we can confirm that embolization of the outflow vessels and the sac could be associated with a long-term clinical success rate [2]. In the presence of type IB endoleak, all of the treatment options for type IA endoleak are valid. Standard treatment usually consists of insertion of an additional endograft distally to achieve a distal seal. If there is insufficient space to extend to the origin of the internal iliac artery, then it is necessary to extend endograft coverage into the external iliac artery. Most of the type IB endoleaks could be easily treated with balloon angioplasty or bare stent implantation. Embolization of the internal iliac artery can be performed with coils or plugs in order to prevent retrograde flow. According to study from Massoni et al., who treated 35 patients with late type IB endoleaks by endograft extensions demonstrated a 100% success rate and 100%% freedom from re-intervention at a mean follow-up at 20 months and no requirement for open surgical treatment [2, 11].

Table 1

Treatment options for Type I Endoleak
Extension stent graft
Balloon angioplasty
Bare metal stent
Embolization
Surgical conversion

Type II endoleak

Type II endoleaks are the most common endoleaks following EVAR and remain the main cause of repeated interventions. It basically presents a retrograde filling of the aneurysmal sac from lumbar arteries and/or IMA, but also in rare situations from sacral, gonadal, or accessory renal artery [1, 2] (Figure 2). Since, the natural history of type II endoleak remains unclear, their management is also controversial. Even though, type II endoleaks are associated with aneurysmal sac expansion and potentially rupture, the risk for these complications is much less than with type I and type III endoleaks [12]. Large part of the type II endoleaks resolve spontaneously and never lead to aneurysmal sac expansion, mainly due to compromised outflow. Because of that, if there is a leak present, but in the setting of shrinking aneurysm, it could be followed up and not immediately intervened [2, 13]. In a study with 474 participants with type II endoleaks, there was no aneurysmal sac rupture related to the endoleak, all-cause mortality was the same in the patients who underwent procedure for the endoleak and in those patients who were not treated [14]. As, we can see, it is well established that up to half of the type II endoleaks will seal spontaneously and will not require intervention. If the endoleak is not associated with aneurysmal sac expansion it seems reasonable to just observe it. Despite of that, some advocate intervening in all endoleaks persisting beyond 6 months [2]. Type II endoleaks, can be divided into type IIA endoleaks, where a single vessel is involved with the blood flow in the aneurysmal sac, and type IIB endoleaks, where multiple vessels are involved. Type IIB endoleaks are behaving like arteriovenous malformations (AVM). The type II endoleaks which persist, have a complex architecture and usually have more than one inflow and outflow vessel, typically presenting like AVM. Predictors of persistent type II endoleaks are numerous collaterals, large central nidus (> 15 mm), high blood flow (velocity > 100 cm/s) and anticoagulation [2, 11-15]. The main question here, is when to intervene a type II endoleak. According to a large meta-analysis of 10 EVAR trials, if there is no evidence of type I and type III endoleaks, a reintervention should be done only when type II endoleak occurs after 6 months and persists more than 12 months or when aneurysm sac pressure is > 20% of systolic blood pressure [16, 17]. On the other hand, a more recent meta-analysis of 10 EVAR trials failed to demonstrate a clear threshold for intervention, due to the rarity of rupture and sac expansion associated with pure type II endoleak [17]. There is an interesting approach for minimization of the type II endoleak, which is a preventive IMA embolization. A study, conducted from Biancari et al., found that the rate of endoleaks without preventive emboli-

zation is 41.4%, while the rate is 19.9% if the IMA was preventively embolized. Even though, type II endoleak treatment is needed in less than 20% of the patients and the aneurysmal rupture is < 1%, this preventive technique, presents a different safe approach [18]. The inflow and outflow vessels communicate with the sac through a channel. If a treatment approach is adopted, embolization of this channel is needed in order to successfully interrupt the leakage. Embolization is the gold standard for treatment and it presents two possible routes – translumbar and transarterial [2]

Transarterial approach

Transarterial embolization is the approach of first choice and the target is the dominant feeding vessel. The most important task is to access the channel, regardless the route chosen. The crucial part is to disrupt the network between the involved vessels. Permanent embolic agents, such as coils are usually used and with this embolic agent all of the vessels should be cannulated and embolized. It is preferred to deposit the coil as close as possible to the origin of the involved vessel [2]. Transiliac Paraendograft Embolization (TIPE) is a novel technique for treating type II endoleaks. The procedure is done by passing a catheter and hydrophilic wire into the potential space between the iliac limb endograft and the vessel wall. Once access into the paraendograft space is obtained, the catheter and wire are advanced superiorly using a standard catheter. The nidus and any visible and accessible feeding vessels are embolized with a liquid embolic and coils or a combination of these agents. Ameli-Renani et al., routinely performed this type of embolization with Ethylene vinyl alcohol copolymer 34 (Onyx – Covidien, Irvine, California, USA), delivered through a microcatheter. The procedure is performed until there is complete filling of the nidus [19].

Translumbar approach

In the past was speculated that this approach would carry a high rate of periprocedural complications, but previous studies showed that this puncture carries a low risk for the patient [20]. The translumbar puncture is performed under CT or fluoroscopy guidance and the puncture site is typically 8 to 10 cm from the midline. There are different approaches after the initial puncture with aiming at the vertebra body of angulating the needle. After successful access, pressure measurement is obtained within the sac and then coil embolization is performed. There are reports on using different agents, such as thrombin, Onyx or Ethibloc [21-23]. Direct sac puncture could be performed via a translumbar approach with the patient positioned prone on the operating table or transabdominally when there is an anterior endoleak. An 18 or 20 G coaxial needle is advanced until there is brisk, pulsatile blood flow, after that the needle is exchanged for a 4,5 or 6 Fr sheath over a stiff guidewire wire and a short selective catheter (e.g. KMP, Bolia, Cobra). This approach has a very high successful rate (97%), but with limited experience [24]. Another interesting approach is the transcaval embolization. Transcaval access into the endoleak cavity is achieved by using an angled-tip catheter and an angled sheathed needle (e.g. TIPSS set) to penetrate the IVC wall and enter the endoleak cavity. The largest cohort included 29 patients, reported by Giles et al., with technical success achieved in 90% and no significant adverse events, 5 patients required reintervention [25]. Surgical ligations is an option, but there are often more vessels involved than initially expected and the surgical approach will present a high risk of failure [2]

Type III endoleak

Type III endoleaks present as a leak through a structural defect of the stent graft, which could be caused by component modular disconnection (type IIIA endoleak) or secondary to a fabric tear (type IIIB endoleak) [1] (Figure 2). It presents the so called high flow endoleaks, similar to type I endoleaks, resulting in sac pressurization. Even though type III endoleaks are relatively uncommon, they need to be treated aggressively since there is an acute pressurization of the sac [1,2]. Cases of type III endoleak are decreasing with the modern stent graft designs. According to a recent study of 965 EVAR cases with reported type 3 endoleaks, 12.7% of the endoleaks occurred with first and second generation endografts and only 1.3% in third generation endografts [26]. Since, the acute nature of type III endoleaks, early and prompt diagnosis is crucial. Enhanced CT alone can show endoleaks, but differentiation between type II and type III endoleaks is difficult, and it is usually confirmed by an angiogram. The treatment approach for type III endoleaks is relining the endograft by deploying a new endograft within the preexisting graft. Embolization is almost never needed, because of the etiology of the endoleak [2, 26].

Type IV endoleak

Type IV endoleak is due to porosity of the endoprosthesis fabric and they are normally visible at the time of device implantation, as a faint blush on the post implantation angiogram. This type of endoleak should be labeled only after ruling out all the other types of leakage. The problem is resolved in new generation endoprosthesis and type IV endoleak is rarely seen and if existing will seal spontaneously [1, 2] (Figure 2).

Type V endoleak

Type V endoleak presents as increase in the sac size in the absence of an identifiable endoleak (also

termed endotension) [1] (Figure 2). Type V endoleak may be due to such slow blood flow that it is below the sensitivity limits for detection on current imaging methods or pulsation of the graft wall and thrombus with transmission of the pulse wave through the perigraft space to the native aneurysm wall. However, development of seroma corresponds to almost all of the endotensions [2, 27]. Since, these endoleaks are not directly associated with high pressure, observation of the leakage may be a valid option. In the cases of increasing aneurysmal sac size, options for interventions are conversion to open repair or the use of extension cuffs, relining the endograft [2].

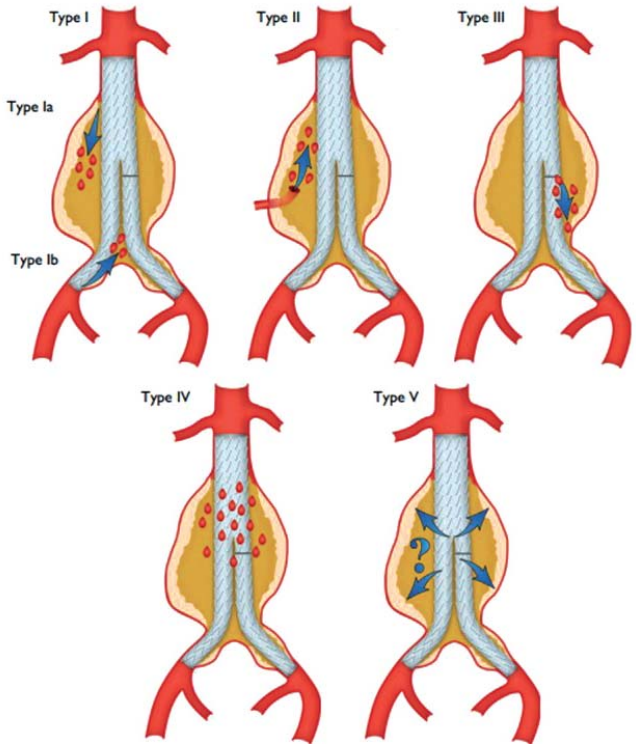

Fig. 2. Classification of endoleaks (no [1])

Our experience

A 6-year (January 2016 to January 2022) retrospective analysis of all patients treated with EVAR in our center was performed. For this period, we have 323 EVAR procedures and 44 (13,62%) of the patients had some kind of endoleak. Type I endoleak was seen in 4 (9,09%) of these 44 patients and type IA endoleak was detected in 3 patients, while type IB was observed in 1 patient. A total of 38 (86,36%) patients with type II endoleak and also 2 patients (4.55%) with type III endoleak were reported. All endoleaks were identified with computed tomography (CT) and they occurred over the range of 10 to 48 months of follow-up. Interestingly enough, one of our patients had presented with both type IA and later with type III endoleak. All of the endovascularly treated endoleaks in our group were successful.

Two of our endoleak type IA patients were initially with infrarenal abdominal aneurysm and we implanted Cardiatis MFM 30/150 mm with iliac extensions Cardiatis MFM 16/120 mm into both of the iliac arteries in the first patient and Endurant II 25/16/166 mm stent in the abdominal aorta with iliac extension Endurant II 16/16/124 mm into the left iliac artery in the second patient. In the first patient we detected the endoaleak on the first postprocedural CT scan, but there was no aneurysmal sac expansion and because of that we decided on closely following it, rather than treating it, since type IA endoleak have a tendency to disappear spontaneously. Unfortunately, the next CT revealed an aneurysmal sac expansion and our patient was symptomatic with abdominal pain and discomfort. Taking into account the systemic pressure and the high risk of rupture we decided on endovascular approach to treat the endoleak. We implanted an extension endograft – Endurant II x 2 16/16/156 mm in the abdominal aorta to the iliac arteries with fully isolated leakage. The procedure was successful without any complications. The second patient with type IA endoleak presented to us 36 months post EVAR with complains of abdominal pain and discomfort which were present for more than a year, but have gotten worse in the past week. The CT revealed a correctly positioned stent graft without evidence of bending or any structural defects, but there was a persistent blood flow into the aneurysmal sac leading to its expansion. We suspected that the patient had a secondary type IA endoleak and because of that we implanted a stent graft Endurant II 36/49 mm with chimney into both renal arteries with two stent grafts Bentley 6.0/38 mm. The leakage was successfully isolated. The third patient was a female with a thoraco-abdominal aneurysm, who presented with a sharp pain in the back, radiating to the scapula and vomiting. The pain was present in the last couple of months, but had gotten worse in the last hours. Unfortunately, the patient had two high pressure endoleaks – type IA endoleak and type III endoleak. Since, in these cases the risk of rupture is very high, an immediate procedure is required. We did an embolization at the level of the overlapping of the prothesis (type III endoleak) with embolization coils Nester 20/20 mm, Nester 20/8 mm x 2, Nester 20/12 mm, Nester 20/5 mm, Nester 20/14 mm and Nester 7/10 mm. Also, we did a balloon molding at the proximal site of the prothesis. Unfortunately, as stated above the combination of these two endoleaks presents a severe complication, because of the high pressure that they apply to the aneurysmal sac and logically despite of our attempts the patient's aneurysmal sac was so big, that it was compressing the left atrium, left superior pulmonic vein, left main bronchus and the esophagus. The patient later died in hypovolemic shock. In one patient we had detected an

endoleak type IB from a branch of the common iliac artery. The patient initially was with impending infrarenal aortic aneurysm. The endoleak was detected on the control CT scan at the 12th month postprocedural. Because there was an enlargement of the aneurysmal sac, we decided on endovascular treatment of the endoleak as an appropriate approach for this patient. Since, the leakage was a canal from the common iliac artery we implanted an Endurant II 16-16/156 mm stent graft in the right common iliac artery with successful isolation of the endoleak. Type II endoleak was detected in 38 patients from our EVAR group. Most of the patients – 30 (78,95%) presented with endoleak type II at the first post procedural CT scan and in the other 8 (21,05%) patients, we detected the type II endoleak at the 6-month post procedural CT scan. Since, the endoleaks were small, we did not intervein and decided to closely follow up the evolution of the leakage. The endoleaks sealed spontaneously in the next year in all of the patients without causing an aneurysmal sac expansion. One of our patients had a type III endoleak after EVAR. The patient had an implantation of stent graft in the abdominal aorta with iliac extensions. After the initial procedure the patient did not have attended the control CT scans and presents 20 months after TEVAR with severe abdominal pain. From the CT scan at presentation the patient's aneurysmal sac was severely enlarged with impending rupture and so that we decided on immediate endovascular treatment. Two Endurant II 16/10/124 mm stent grafts were implanted with successful isolation of the leakage.

Conclusion

Endoleak remains a problematic complication associated with EVAR and while some types of endoleaks seal spontaneously without causing aneurysmal sac expansion, others can be very serious with bad prognosis. Type I and type III endoleaks are the ones, that need to be treated aggressively. There are different endovascular options available for the treatment of persistent endoleaks. Extension stent graft, bare stent, balloon angioplasty are some of the invasive treatment options. Persistent type II endoleaks, leading to sac expansion are best treated with the method of embolization. Even though there are different endovascular options for treatment, the crucial part is early detection, which leads to high technical success rates when managed early after the initial implantation.

No conflict of interest was declared

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