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Endotension – a cause of failure in endovascular repair of abdominal aortic aneurysms

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Summary

The phenomenon of aortic aneurysm enlargement after endovascular repair without detectable endoleak is called "endotension". It is caused by persistent pressurization within the excluded aneurysm sac and may cause subsequent rupture of the aneurysm. We undertake a review of current knowledge about causes, significance and treatment of endotension as a failure in endovascular aortic aneurysm repair. The goal of endovascular abdominal aortic aneurysm repair is to prevent aneurysm ruptures by excluding the aneurysms from the aortic circulation. AAA (Abdominal Aortic Aneurysm) after EVAR (Endovascular Aneurysm Repair) can enlarge even in the absence of detectable endoleak because of persistent pressurization within the excluded aneurysm. There are many theories about the mechanism of pressure transmission into the excluded aneurysmal sac. Some laboratory and clinical research shows that endotension can be connected with attachment side failure, graft fabrics or aneurysm sac geometry. Pressure transmission by the thrombus, poor outflow, osmotic effect or ultrafiltration are the other possible mechanisms causing this phenomenon. Maximal diameter measurement by CT is considered to be the best management method in patients after EVAR. However, lack of aneurysm sac shrinkage observed in some cases does not mean the presence of endotension. The role of pulsatility inside the excluded aneurysm sac remains unclear. Several possible concepts of endotension treatment have been discussed, including both open surgical convention and nonoperative approach. The absence of endoleak after endovascular repair not always means that there is no pressurization within the aneurysm. Success of endovascular repair can be evaluated indirectly by observation of changes in the diameter of the aneurysm sac after EVAR. Thus, it is essential to follow up patients after endovascular repair in order to detect any late complications including endotension.

Key words: Abdominal Aortic Aneurysm • Endovascular Aneurysm Repair • endotension

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Background

Endovascular repair has gained popularity quickly as an alternative to conventional surgical treatment of abdominal aortic aneurysms (AAA). However, the new technique is associated with many new problems, not encountered previously. The success of endovascular treatment of an abdominal aortic aneurysm with stent-graft implantation (EVAR – Endovascular Aneurysm Repair) can be defined broadly as exclusion of the distended segment from circulation without adverse events such as leak, occlusion of the vessel, dislocation or rupture of the stent-graft, or the need for re-operation. The size of most AAA is reduced after

endovascular repair. However, it has been observed that some of them, despite exclusion of the aneurysm sac from circulation, do not cease to expand. This phenomenon, also regarded as a treatment failure [1], associated with persistent or recurrent pressurization exerted on the AAA walls despite the indwelling stent-graft, has been termed „endotension“.

The concept of endotension, defined as AAA enlargement without the presence of endoleak, was first proposed in 1996 by the Sidney group [2]. The correct use of the term „endotension“, the factors responsible for it and its grading was widely discussed. The topic is still controversial

and its explanation is based more on theory than on scientific facts. Enlarging aneurysm sac, even without observable endoleak, poses a risk of rupture [3]. Other changes of aneurysm sac morphology after EVAR are not taken into consideration in this definition.

Theories

There are many theories attempting to explain the origins of endotension. They can be divided into 5 main categories [3]:

1. Transmission of pressure to the aneurysm sac around the ends of the graft through the thrombus layer between the stent-graft and the AAA wall (involving sealed type I endoleak), displacement of the stent-graft module exposing a thrombus in the AAA neck or undetected endoleak;
2. Transmission of pressure through the stent-graft wall; stent-grafts with high porosity demonstrate higher pressurization inside the AAA sac [4]; microleaks, effusion or diffusion through the graft material can be the cause of endotension;
3. Transmission of pressure from the aortic branches occluded by the stent-graft, which can also be mediated by the thrombus;
4. Pressure generated by volume increase of the structures inside the AAA sac, which can be caused e.g. by infection of the stent-graft, thrombus fibrinolysis or another enzymatic activity within the aneurysm;
5. Endotensja without pressure increase, whose causes can also be attributed to the aforementioned phenomena;

Each of the above 5 mechanisms can occur in isolation or in combination with others in the development of endotension.

Classification

The term „endotension“ is currently used for the phenomenon of aneurysm sac enlargement after endovascular treatment. Endotension is strictly defined as the presence of increased pressure in the aneurysm sac after its endovascular repair with stent-graft implantation, in a situation when endoleak is not detectable by an appropriate CT protocol. In a broader sense, the term denotes each situation involving increased pressure in the aneurysm sac after EVAR, including the cases in which endoleaks are observed. Table 1. presents the types of endotension.

Laboratory and Clinical Investigations

The phenomenon of endotension has been observed by numerous authors. Clinical studies have confirmed that patients with enlarging aneurysm sac are at risk of AAA rupture, even if there is no endoleak [2]. There is a consensus that this enlargement is a result of persistent or recurrent pressurization within the aneurysm sac and that it is associated with the risk of rupture. The earlier concepts that AAA enlargement after stent-graft implantation might be associated with an undetected endoleak have been questioned. Gilling-Smith et al. reported that the absence of endoleak demonstrated by conventional investigation techniques does not evidence the absence of endotension. They confirmed that in a group of 55 patients, where 10 out of 44 subjects with no detected endoleaks demonstrated enlarge-

Table 1. Classification scheme for endotension [5].

Type	Characteristics
A	Without endoleak
B	With sealed endoleak
C	With type I or III endoleak*
D	With type II endoleak*

* Endoleaks detectable only during open surgery of the aneurysm sac.

ment of the AAA sac. The conclusion from these studies indicates the necessity of systematic monitoring of the changes in maximal AAA diameter and its volume to identify the patients at risk of rupture. Different results were obtained by Meier, who suggests that endotension is a rare phenomenon, associated probably with the presence of an undetected endoleak [5]. He analyzed 476 cases of patients, divided into three groups. The first one consisted of subjects who had never been diagnosed with endoleak, the second – those in whom endoleaks had been detected initially, but then they were sealed. The third one consisted of patients with no endoleaks initially, who developed them later during the observation period. Sanchez et al. demonstrated on a canine model that the pressure within the aneurysm sac after implantation of a tantalum-dacron stent-graft did not differ significantly from the systemic arterial blood pressure despite the lack of endoleaks. They attributed such situation to high porosity of the graft made of this material. When low-porosity polytetrafluorethylene grafts were used in the same model, the measured pressure values were significantly lower, although they still exceeded those obtained after classic procedures utilizing PTFE grafts [4]. Skillern et al. investigated pressure values in the AAA sac in case of incorrect stent-graft hold placement without observable endoleaks. Their studies on an animal model demonstrated that inappropriate placement of the proximal stent-graft hold may, despite no endoleaks observed, result in pressure increase within the AAA sac. The above was additionally confirmed by a notable decrease of that pressure after endovascular correction of proximal fixation [6]. Exclusion of the AAA sac from the circulation by stent-graft implantation usually results in reduction of pressure within the aneurysm, except for the cases where endoleaks are present. Pacanowski et al. presented interesting data indicating that the distribution of pressure inside the AAA depends on its structure. The pressure was the highest in the vicinity of the stent-graft and decreased towards its walls [7]. Other studies demonstrated persistent pressure transmission via the endoleak canal sealed with a thrombus. The pressure exerted in this way was proportional to the width of the occluded canal and inversely proportional to its length. Thus, it can be supposed that in clinical situations wide and short endoleak canals may cause endotension with higher probability, whereas long and narrow ones, such as small lumbar arteries, may be sealed without exerting pressure on the AAA sac [8]. In a few clinical reports it was noted that open surgery revealed the aneurysm sac filled with a semi-liquid, jelly-like thrombus or fluid containing no blood. Such a situation can be caused for instance by degradation of the AAA sac content to osmotically active products and secondary fluid accumulation inside. Other

theories propose the presence of ultrafiltration through the stent-graft wall with consequent development of a hydrocele, hyperfibrinolysis, periodic endoleak or an infection.

It remains unclear to what extent pulsatility of the repaired aneurysm can be regarded as a clinical marker of EVAR failure. Mehta et al. demonstrated that pulsation is dependent on outflow and not on the presence of endoleak, thrombi inside the stent-graft and the material it is made of [9]. They also demonstrated that pulsation is not an indicator of the pressure value inside the aneurysm sac. Nevertheless, palpable pulsation changes can indicate recurrence of pressure increase within the AAA if they were undetectable in the early post-implantation period.

One of the questions that still have to be answered is whether reduction of pressure within the AAA sac is equally effective in case of a small canal in the stent-graft wall sealed by a thrombus in comparison with structural character of the seal. A thrombus or another fibrous material sealing the endoleak canal can transmit higher pressure into the AAA sac lumen. There is also evidence that some coating materials applied in the structure of stent-grafts predispose them for the development of microleaks which can result in increased pressure within the AAA [10].

Current State of Knowledge

There is a consensus that the applicable definition of endotension should include the presence of increased pressure in the aneurysm sac after its endovascular repair in a situation when no endoleak is detectable by an appropriate CT protocol involving the use of contrast [11]. Division of endotension into 4 types has been proposed. The differentiation of specific endotension categories seems justifiable, however, their clinical implications have not been determined yet. Other conclusions concerning endotension are as follows:

1. Maximum AAA diameter measurement by CT is a sufficient indicator of changes in its dimensions, and CT itself has been regarded as more useful for detection of endoleaks than Doppler sonography.
2. An AAA still increasing in size after endovascular repair, even without observable endoleak, should be subjected to surgical treatment or repaired using a new stent-graft.
3. The presence of pulsatility after EVAR, or its absence, does not necessarily indicate the presence or absence of endoleak or endotension. Elimination of pulsatility can

indicate effective exclusion of the AAA sac from the circulation, however, this sign is not completely reliable.

4. Although AAA sac shrinkage after EVAR is a desirable effect, its failure to occur may not indicate the presence of endoleak or endotension. Aneurysms over 6 cm in diameter may not shrink, or this process can take many years, depending on the stent-graft type used.

Management of Endotension

The data concerning effective treatment of patients with detected endotension are scarce. The following options are considered:

1. Conservative treatment with more frequent control examinations,
2. Conservative treatment with later invasive monitoring of pressure inside the AAA sac,
3. Conversion to classic surgery,
4. Repeated stent-graft implantation.

Conclusions

No detectable presence of an endoleak cannot be treated as sufficient evidence for the absence of endotension [12]. Shrinkage of the AAA sac after EVAR implies intuitively its complete exclusion from the circulation, serving as objective, easily obtained evidence for absence of this phenomenon and successive outcome of stent-graft implantation [13]. Direct measurement of pressure within the aneurysm sac by approach via the iliac artery, although possible, seems to be difficult to use in everyday practice [14]. Until new, less invasive methods of pressure measurement within the AAA are introduced, AAA shrinkage will remain the only available criterion allowing to exclude the presence of endotension.

There are numerous theories concerning the causes of endotension, attributing its occurrence to undetected leaks, chronic infections or genetic factors. Monitoring patients by periodic CT scans and Doppler sonography is currently considered to be necessary for detection of potential endotension-related complications. Further research of etiology, pathophysiology and clinical consequences of endotension is essential. The progress in technology should enable non-invasive measurements of pressure within the AAA and establishment of new parameters useful in everyday clinical management of patients after EVAR.

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