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Original Article

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Is there a relationship between endoleak and antiaggregant treatments in the change of the aneurysm sac after endovascular treatment in abdominal aortic aneurysm?

Abstract

Aim: It was aimed to determine the prognosis and treatment in relation to the risk factors of the patient group by recording the changes in aneurysm sac, thrombus diameter and thrombus load in the follow-up of patients treated with endovascular stent-graft for abdominal aortic aneurysm (AAA).

Material and Methods: Preoperative and postoperative 1st, 3rd, 6th and 12th month CT-A's of 112 patients who were followed up regularly were performed and changes in aneurysm sac diameter, thrombus diameter and thrombus percentage were recorded.

Results: When the measurements were evaluated, an increase was detected in the mean thrombus diameter at post-operative month 1 with a gradual reduction over time; however, it was observed to be higher than the pre-operative values even at month 12. These changes were statistically significant ($p<0.001$). Aneurysm sac diameters of the cases in the pre-operative work-up ranged between 50mm and 108mm with a mean of 65.53±12.08mm. Pre-operative thrombus burden was measured to be 28% minimum and 96% maximum with a mean of 61.8±18.69%. In the post-operative measurements, the mean thrombus burden was 86.0±13.47 at month 1, 85.9±15.46 at month 3, 82.94±14.27 at month 6 and 82.27±13.82 at month 12. Similar to the changes in thrombus diameter, there was an increase at month 1 with a gradual reduction over time; however, the values at month 12 were still higher than the pre-operative values. These changes were also statistically significant ($p<0.001$).

Conclusion: The present study showed that the changes in aneurysm sac diameter and endoleak formation may affect prognosis and single antiaggregant treatment would be more suitable compared to the combination treatment.

Keywords: Endoleak, aneurysm sac, endovascular aortic aneurysm repair, antiaggregant treatmentss

INTRODUCTION

Aneurysm sac shrinkage after endovascular aortic aneurysm repair (EVAR) is an important indicator of successfully excluding the aneurysm and resolving the pressure issue there (1). The aim of the aortic aneurysm repairs is to prevent aortic rupture (2). Since early 1990s, EVAR has been an alternative to open surgery for the treatment of abdominal aortic aneurysm (AAA) and substantially reduced the operative mortality (3). The advantages of EVAR procedures include the short intensive care unit and hospital stays, minimal surgical trauma, and being able to be used in patients with high morbidity and in elderly. Several studies have been performed to scrutinize the independent parameters which may affect the aneurysm sac after the endovascular repair (4-6). The main parameters studied include pre-operative aneurysm diameter, the number and diameter of the aortic branches, thrombus burden, the length and shape of the proximal neck, diameter of the minor neck, presence of neck plates/ thrombi, age, sex, antiaggregant and anticoagulant treatment,

and smoking. Endoleak is defined as the "continued or recurring perfusion of the aneurysm sac after stent graft implantation", and while it affects 25% of the patients during follow-up, the aspects of the many cases have yet to be clearly understood (7,8). "Endoleak" classification includes the following: Type I "endoleak", originating from the proximal or distal end of the graft; Type II, originating from the retrograde flow into aneurysm sac from lumbar arteries or other collaterals; Type III, originating from the rupture caused by manufacturing defect or modular joint; and Type IV, caused by increased permeability of the graft (9). Endoleaks are one of the most frequent complications of endovascular aneurysm repair and the most important cause of post-procedural secondary interventions. Studies have shown the correlation between the pre-operative thrombus burden and the changes in sac size following EVAR. In their study, Yeung et al. observed that the odds of aneurysm sac shrinkage is lower in patients with higher thrombus burden compared to the patients with lower thrombus burden.2 There have also been studies evaluating the association between type 2 endoleaks and aneurysm sac size, and it has been shown that it is more frequently associated with sac enlargement (10,11). In some other studies, however, no significant correlation could be shown between pre-operative aneurysm sac thrombus burden and post-operative sac shrinkage (12,13). Significant shrinkage in sac size is an important indicator of the continued treatment success (14). This study aimed to determine prognosis and treatment methods in association with the risk factors of the patient group by recording the changes in the aneurysm sac, thrombus diameter and thrombus burden during the follow-up of the patients treated using endovascular stent-graft for AAA.

MATERIAL AND METHODS

Patient Selection

One hundred and forty-six patients who were assessed using computerized tomography angiography (CT-A) with the pre-diagnosis of AAA and planned to undergo endovascular treatment between 2013 and 2022 in Cardiovascular Surgery Department, Tekirdağ Namık Kemal University Faculty of Medicine Hospital were included into the study. These patients underwent radiological imaging before and 1, 3, 6, and 12 months after the procedure. Patients who underwent redo procedure during the follow-up period, were lost to follow-up or continued their follow-up in an external center were excluded from the study. One hundred and twelve patients meeting our clinical criteria were included into the study. Two groups were formed based on the treatment method. Group 1 received only aspirin for treatment and Group 2 received aspirin+clopidogrel. Among the patients included into the study, 98 were males and 14 were females with a mean age of 76.5±8.7 years. Pre-operative and post-operative CT-A images of the patients were assessed retrospectively. Aneurysm sac diameter, thrombus diameter and thrombus burden measurements were taken using axial, coronal and sagittal images, and the data were recorded. The risk factors and demographics of the patients were recorded. This is a retrospective study with ethics committee approval being taken from Ethics Committee, Tekirdağ Namık Kemal University.

Assessment and Follow-up Protocol

Pre- and post-operative CT-A images taken included the whole abdominal aorta and iliac arteries up to the main femoral artery in all patients. Non-ionic iodinated contrast material with a density of 320-400 mg/dL was used for the work-up. Before the work-up, time-lapse was calculated in the suitable anatomic localization using 12-16 mL of contrast material injection at a rate of 4 mL/sec. Axial images were taken by injection of a total of 100-120 mL of contrast material on average.

Evaluation

One hundred and twelve patients were followed up regularly, and the changes in their aneurysm sac diameter, thrombus diameter and thrombus percentage were recorded using pre-operative and post-operative 1-, 3-, 6- and 12-month CT-A images. These changes were evaluated using the data obtained by several measurements taken by the same investigator using axial, coronal and sagittal CT-A images. Reference points of vertebral level, aortic wall calcifications and arterial orifices were determined in the section with the widest aneurysm, and the same section was used in the post-operative work-up to record the measurements

Statistical Analysis

Statistical analysis was performed using SPSS. Data were expressed as mean±standard deviation (SD), number and percentage. First, the findings were determined. Pre-operative data on the aneurysms were compared with post-operative data. Pearson Chi-square test was used to compare the different parameters. Independent t-test was used for the comparison of the continuous variables between the groups. Patients were followed up regularly. Endoleak development and sac behaviours were analysed and compared during the follow-up visits. A p value of <0.05 was considered to be statistically significant.

RESULTS

The mean age of 112 patients included into the study was found to be 76.5±8.7 years. Ninety-eight of the patients (87.5%) were males and 14 (12.5%) were females. Table 1 shows the demographic characteristics and risk factors of the patients. According to this, 90 patients (80.3%) had hypertension, 64 (57.1%) had hyperlipidaemia, 84 (75%) had chronic obstructive pulmonary disease (COPD), 72 (64.2%) coronary artery disease (CAD) and 4 (35.7%) had peripheral artery disease (PAD). Seventy-four patients (66%) had the history of smoking. The mean body mass index (BMI) was 28.17±2.76. During the follow-up, 70.5% of the patients received Aspirin for treatment and 29.5% Aspirin + Clopidogrel. Table 2 shows the pre- and post-operative changes in aneurysm sac, thrombus diameter and thrombus burden. Aneurysm sac diameters of the cases in the pre-operative work-up ranged between 50mm and 108mm with a mean of 65.53±12.08mm. In the post-operative measurements, the mean aneurysm sac diameter was 64.32±11.24mm at month 1, 59.36±12.40mm at month 3, 56.32±13.24mm at month 6

and 52.36±13.86mm at month 12. During the post-procedural follow-up, minimum aneurysm sac diameter was measured to be 30mm and maximum to be 102mm. This progressive shrinkage was statistically significant (Table 2). Pre-operative thrombus diameter was measured to be 11mm minimum and 56mm maximum with a mean of 25.36±8.64mm. In the post-operative measurements, the mean thrombus diameter was 31.46±7.84mm at month 1, 28.34±8.32mm at month 3, 26.69±9.22mm at month 6, 26.86±11.26mm at month 12. When the measurements were evaluated, an increase was detected in the mean thrombus diameter at post-operative month 1 with a gradual reduction over time; however, it was observed to be higher than the pre-operative values even at month 12. These changes were statistically significant (p<0.001) (Table 2). Pre-operative thrombus burden was measured to be 28% minimum and 96% maximum with a mean of 61.8±18.69%. In the post-operative measurements, the mean thrombus burden was 86.0±13.47 at month 1, 85.9±15.46 at month 3, 82.94±14.27 at month 6 and 82.27±13.82 at month 12. Similar to the changes in thrombus diameter, there was an increase at month 1 with a gradual reduction over time; however, the values at month 12 were still higher than the pre-operative values. These changes were also statistically significant (p<0.001) (Table 2). In the light of these results, we can say that abdominal aortic aneurysm starts to take form at month 3 following EVAR. Table 3 shows the evaluation of the overall change in the aneurysm sac by antiaggregant use. When the changes in aneurysm sac diameter were evaluated by antiaggregant use, the difference between the measurements was found to be significant (p<0.001). The difference between 2 groups in terms of antiaggregant use was not statistically significant (p=0.79). Similarly, the changes in thrombus diameter were evaluated by antiaggregant use. The difference between the measurements was significant (p<0.001). The difference between 2 groups was not found to be statistically significant in terms of antiaggregant use (p=0.65). While the difference between the measurements of the changes in thrombus diameter in terms of antiaggregant use was significant (p<0.001), the difference between 2 groups by anticoagulant use was not found to be statistically significant (p=0.32) (Table 3). The rate of endoleak occurrence was evaluated by antiaggregant use (Table 4). Endoleak was observed in a total of 14 patients (17.7%) in group 1 and in 24 patients (72.7%) in group 2. None of the patients had type 1 endoleak in group 1, and two patients had type 1 endoleak in group 2 and underwent revision procedure. Spontaneous regression was observed in patients with type 2 endoleak during follow-up and no increase was observed in the aneurysm diameter. Table 5 shows the endoleak development by aneurysm sac diameter and thrombus burden. Aneurysm enlargement was assessed by the changes in sac diameter, as well as the changes in sac volume. The mean change in the thrombus volume was a more sensitive indicator of enlargement than the change in aneurysm sac diameter (Table 5). The mean volume change in patients who developed type 2 endoleak (46.3%±13.1%) was statistically significant (p<0.0001) compared to the patients who did not develop endoleak (23.2%±18.6%). The possibility of type 2 endoleak occurrence was lower in patients with high thrombus burden in the pre-operative CT scan compared to the patients with minimal thrombus burden. While out of 26 patients with >50% thrombus burden, only 4 (15.4%) developed endoleak following EVAR, out of 86 patients with <50% thrombus burden 32 (37.3%) developed endoleak (p<0.0001). Surprisingly, the mean post-operative thrombus burden did not differ significantly between the patients who developed and did not develop type 2 endoleak. Therefore, the percentage of increase in the thrombus burden in patients who developed endoleak (46.3±13.1%) was higher than the patients who did not develop endoleak (23.2±18.6%) (p<0.001). This suggests that patients who had low thrombus burden initially and did not develop post-operative endoleak develop more rapid or complete thrombosis in their aneurysm sac.

Table 1. Demographic data and risk factors

	n (112)	Mean±SD/n (%)
Age		76.5±8.7
Gender		
Male	98	87.5%
Woman	14	12.5%
HT	90	80.3%
HL	64	57.1%
BMI		28.17±2.76
Smoke	74	66%
Chronic obstructive pulmonary disease	84	75%
CAD	72	64.2%
PAD	40	35.7%
Chronic renal failure	16	14.2%
Stroke	10	8.9%
Myocardial infarction	2	1.7%
Cancer history	10	8.9%
Use of aspirin	79	70.5%
Use of Aspirin+Clopidogrel	33	29.5%

BMI: body mass index, CAD: coronary artery disease, PAH: peripheral artery disease, MI: myocardial infarction, HT: hypertension, HL: hhyperlipidemia

Table 2. Preoperative and postoperative evaluation of aneurysm sac and thrombus diameter/burden

(Mean±SD/min-max)	Preoperative values	Postoperative 1st month values	Postoperative 3st month values	Postoperative 6st month values	Postoperative 12st month values
Aneurysm sac diameter (mm)	65.53±12.08 (50-108) (p<0.001)	64.32±11.24 (48-102)	60.36±12.40 (32-84)	58.32±13.24 (32-82)	56.36±13.86 (30-82) (p<0.001)
Thrombus diameter (mm)	25.36±8.64 (11-56) (p<0.001)	31.46±7.84 (18-59)	28.34±8.32 (13-53)	26.69±9.22 (13-52)	26.86±11.26 (14-55) (p<0.001)
Thrombus burden (mm) (%)	61.8±18.69 (28-96) (p<0.001)	86.0±13.47 (39-101)	85.9±15.46 (39-99)	82.94±14.27 (38-98)	82.27±13.82 (38-97) (p<0.001)

Table 3. Preoperative-postoperative comparison of changes in sac diameter, thrombus diameter and thrombus load–subgroup analyzes by antiaggregant use

(mean±SD)	Preoperative values	Postoperative 1st month values	Postoperative 3st month values	Postoperative 6st month values	Postoperative 12st month values	p
Aneurysm sac diameter (mm) Group 1	65±13	65±12	60±10	58±12	56±10	<0.001
Group 2 (p=0.79)	65±10	63±13	60±12	57±14	56±14	<0.001
Thrombus diameter (mm) Group 1	27±12	36±10	32±8	30±8	28±7	<0.001
Group 2 (p=0.65)	24±16	31±9	30±8	28±8	25±10	<0.001
Thrombus burden (%) Group 1	58±16	80±15	78±12	77±12	77±14	<0.001
Group 2 (p=0.32)	62±19	88±6	85±7	83±8	83±8	<0.001

Group 1 (uses only acetyl salicylate), Group 2 (uses acetyl salicylate+clopidogrel)

Table 4. Distribution of endoleak ratio according to antiaggregant use

Endoleak	Group 1 (n/%) (n=79)	Group 2 (n/%) (n=33)
Type I	0 (0%)	2 (6.1%)
Type II	14 (17.7%)	22 (66.6%)
Total	14 (17.7%)	24 (72.7%)

Group 1 (uses only acetyl salicylate), Group 2 (uses acetyl salicylate + clopidogrel)

Table 5. Type 2 endoleak development according to aneurysm sac diameter and thrombus burden

(mean±SD)	Type 2 Endoleak (n: 36)	No Type 2 Endoleak (n: 76)	p
Preoperative mean aneurysm sac diameter	62.36±12.20	64.16±12.70	NS
Postoperative mean aneurysm sac diameter	65.32±14.24	62.47±11.20	NS
Average diameter change (mm)	2.9±8.6	-1.6±12.7	0.007
Preoperative mean thrombus burden(%)	47.8±12.69	60.3±15.46	<0.0001
Postoperative mean thrombus burden(%)	84.1±12.47	83.5±15.64	NS
Mean change in thrombus volume (%)	46.3±13.1	23.2±18.6	<0.0001
Preoperative <50% thrombus burden (n: 86)	32 (37.3%)	54 (62.7%)	<0.0001
Preoperative >50% thrombus burden (n: 26)	4 (15.4%)	22 (84.6%)	<0.0001

NS: non significant

DISCUSSION

In the present study, we studied whether aneurysm sac behaviours and endoleak development vary by antiaggregant use and explored the correlation between endoleak development and sac behaviours. In our study, 87.5% of the patients were males and 12.5% were females. This ratio was consistent with the literature data (15,16). AAA is observed in 6% of the males over the age of 60 with 1% of the females of the same age being affected (17). A study has shown that while the prevalence of AAA is lower in females, its mortality and morbidity is higher . In the same study, it was found that the incidence of intra-operative aneurysm neck and iliac artery rupture is higher, the mean blood loss is greater and the mean duration of hospital stay is longer in female patients with no statistically significant difference was detected in terms of mortality between EVAR and open surgery in females (3.2%-5.7%); the mortality rate in male patients was 0.96% following EVAR and 4.7% following open surgery with the difference being significant (18). As for the risk factors in the present study, the rate of hypertension was 80.3%, hyperlipidaemia was 57.1%, peripheral artery disease was 35.7%, chronic obstructive pulmonary disease 75%, coronary artery disease 64.2% and

smoking was 66%. Histories of myocardial infarction, stroke and cancer were risk factors in <10% of the patients. BMI of our study group was found to be 28.17±2.76. In the EVAR 1 study, the prevalence of AAA in patients with severe CAD was detected to be 50%. In the same study, while aspirin+statin use following EVAR was reported for 35.9% of the patients, no data was seen on the clopidogrel use (19). In the present study, 64.2% of the patients had CAD with 70.5% of them receiving only aspirin and 29.5% receiving both aspirin and clopidogrel; and Type II endoleak was observed in 66.6% of the patients in group 2 (aspirin+clopidogrel) and 17.7% of the patients in group 1 (aspirin). In their study, Bobadilla et al. (20) found that the incidence of endoleak following EVAR for AAA to be 20-30%, and explored the effect of warfarin on endoleak in this study. In the study including 127 patients, the incidence of endoleak was found to be 54% in the group receiving warfarin and 25% in the group receiving single antiaggregant treatment. In another study, the effect of antiaggregant treatment and type II endoleak development following EVAR for AAA on the aneurysm sac behaviours was studied and it was founded that aneurysm sac was completely excluded and shrunk only in 60% of the patients following EVAR. The correlation of aneurysm sac behaviours with multiple antiaggregant use, hyperlipidaemia, pre-procedural aneurysm sac diameter, aneurysm proximal neck length and type II endoleak was studied, and it was concluded that only multiple antiaggregant use and type II endoleak have effects on sac behaviours (21). In the present study, 66.6% of the patients who developed type II endoleak were receiving aspirin+clopidogrel and 17.7% were receiving only aspirin. Therefore, we believe that the changes in aneurysm sac diameter and the formation of endoleak may affect prognosis and single antiaggregant treatment is more suitable. Sixty-six percent of the cases in our study group were smokers. The most important environmental factor for aortic aneurysm development is smoking. Therefore, CAD is also present in the majority of the patients with AAA even though their physiopathologies differ. In their study, Koole et al. (22) evaluated the effects of smoking on aneurysm sac enlargement following EVAR and found that the rates of percutaneous transluminal angioplasty (PTA) intervention and graft migration are higher in smokers. Moreover, the incidence of type II endoleak was found to be 35.5% in smokers and 58.5% in non-smokers.

While EVAR provides many successful outcomes for the treatment of AAA, studies have been performed due to the anticipated effects of the post-procedural changes in thrombus diameter, thrombus burden and aneurysm sac over time on the long-term outcomes of the endovascular method (23). In the present study, the primary parameter evaluated was whether there is a change in the aneurysm diameter early after EVAR. The mean aneurysm sac diameter shrank by 5.1mm during the first three-month period, by 7.2mm at month 6 and by 9.1mm at month 12 with the difference being statistically significant. Our results were consistent with the literature data. Our study is not a randomized study, but was designed by examining the randomized studies in the literature. In a multi-center study involving 676 AAA patients treated using EVAR, aneurysm enlargement was observed in 8% of the patients during 3-year follow-up (24). Aneurysm diameters were shown to be shrunk except for the group showing limited enlargement. In the EVAR TRIAL study, Sandford et al. (25) stated that a mean of 7.5mm post-operative shrinkage was observed in the aneurysm sac diameter during the 5-year follow-up. In their series involving 84 cases, Resch et al. (26) detected a mean of 9mm shrinkage in the aneurysm sac diameter during the first year following EVAR. Similar results were obtained in the present study. While it was stated that the majority of type 2 endoleaks are benign and only require follow-up in some studies (27,28), it was reported that early intervention is necessary to prevent sac enlargement and rupture formation in others (29,30). As is the case with the other studies, there have also been cases of endoleak in our study. Two patients underwent revision procedure due to type I endoleak. Thirty-six patients who developed type II endoleak were followed-up conservatively, and during their routine follow-up, spontaneous regression was observed and no increase in the aneurysm diameter was seen. In our study, the incidence of endoleak in patient who underwent EVAR due to AAA was found to be 33.9%. In the present study, a clear correlation seems to exist between pre-operative thrombus burden, post-operative endoleak presence and aneurysm sac enlargement following EVAR. There are multiple to-and-fro vessels usually in patients with type 2 endoleak (31). Similarly in our study, it was demonstrated that the development of endoleak is more frequent in patients with low thrombus burden. Frequent follow-up of the patients using radiological images is an accepted practice for monitoring the aneurysm morphology and type II endoleak cases (32). The standard method to evaluate the aneurysm changes is to measure the sac diameter. This has been used for reliable monitorization of AAAs after repair and also to decide which patients require a secondary intervention. Some studies have demonstrated that using volume measurements to monitor aneurysm enlargement is a reliable alternative to diameter measurements (33,34). Bargellini et al. have shown that aneurysm volume might be a more sensitive method to monitor the changes in aneurysm sac over time and a better way to predict endoleaks (35). A more significant difference was shown in our study, especially in patients with endoleak. While we believe that volume is a better method to evaluate the changes in aneurysm sac, it did not gain popularity as it is difficult to obtain. With the wider use of 3D imaging technologies, this became an easier-to-obtain measurement and we believe that it should be used more commonly. At the present time, diameter and volume measurements are the only parameters which can be obtained practically.

In the present study, the changes in thrombus diameter and thrombus burden following EVAR were also considered. According to our results, the comparisons made with the pre-operative assessments demonstrated that there is a statistically significant increase in the thrombus diameter and burden during the first post-operative month, however, regression is seen during

the follow-ups at months 3, 6 and 12. With that being said, it was seen that the thrombus diameter and thrombus burden do not return to the pre-operative values after the end of 12-month follow-up. Limited number of studies on this subject were found in our literature search. In the present study, it was seen that post-operative values of thrombus diameter and burden after 12 months of follow-up are close to the pre-operative values, however, did not drop below pre-operative values. The reason for the lack of predicted regression in the thrombus diameter and burden even though there is a regression in the aneurysm diameter could not be determined clearly. The present study has several limitations. Perhaps, the most important one is the relatively short duration of follow-up. As the duration of follow-up is limited to 12 months, it is an early-term study and the obtained results are greatly in line with the literature data. The retrospective collection of the demographic characteristics and risk factors of the patients from the patient files is another limitation. In this sense, our results originate from the patient files.

CONCLUSION

The present study showed that the changes in aneurysm sac diameter and endoleak formation may affect prognosis and single antiaggregant treatment would be more suitable compared to the combination treatment. One of the important contributions of this retrospective review is that it provides an alternative measure of evaluation to help predict the post-procedural aneurysm sac enlargement, stabilization or shrinkage. While the changes in aneurysm sac are commonly being used to monitor aneurysm behaviours, the changes in sac volume was demonstrated to be a more sensitive indicator of the increase in aneurysm size following EVAR. It was demonstrated that the possibility of endoleak development and aneurysm sac enlargement in AAAs with a high pre-operative thrombus burden is lower than the AAAs with low thrombus burden. However, long-term studies including larger patient populations are needed in the literature.

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Ethics Committee Approval: This is a retrospective study with ethics committee approval being taken from Ethics Committee, Tekirdağ Namık Kemal University (Protocol Number: 2021.75.03.15 date: 30.03.2021)

REFERENCES

1. Rjee RY, Eskandari MK, Zajko AB, Makaroun MS. Long-term fate of the aneurysmal sac after endoluminal exclusion of abdominal aortic aneurysms. J VascSurg 2001;32:689e-96.
2. Norwood M, Lloyd G, Bown M, et al. Endovascular abdominal aortic aneurysm repair. Postgrad Med J. 2007;83:21-7.
3. Brown LC, Thompson SG, Greenhalgh RM, Powell JT; Endovascular Aneurysm Repair trial participants. Incidence of cardiovascular eventsand death after openor endovascular repair of abdominal aortic aneurysm in therandomized EVAR trial 1. Br J Surg. 2011;98:935-42.
4. Houbballah R, Majewski M, Becquemin JP. Significant sac retraction after endovascular aneurysm repair is a robustindicator of durable treatment success. J Vasc Surg. 2010;52:878-83.
5. Schanzer A, Greenberg RK, Hevelone N, et al. Predictors of abdominal aortic aneurysm sac enlargement after endovascular repair. Circulation. 201121;123:2848-55.
6. Hogg ME, Morasch MD, Park T, Flannery WD, Makaroun MS, Cho JS. Long-term sac behavior after endovascular abdominal aortic aneurysm repair with the Excluderlow-permeability endoprosthesis. J Vasc Surg. 2011;53:1178-83.
7. Cao P, De Rango P, Verzini F, Parlani G. Endoleak after endovascular aortic repair: classification, diagnosis, and management following endovascular thoracic and abdominal aortic repair. J Cardiovasc Surg 2010;51:53-69.
8. White GH, Yu W, May J, Chaufour X, Stephen MS. Endoleak as a complication of endoluminal grafting of abdominal aortic aneurysms: classification, incidence, diagnosis, and management. J Endovasc Surg. 1997;4:152-68.
9. Eikelboom BC, Blankenstejin. How and when to treat an endoleak after endovascular abdominal aortic aneurysm repair. In: Whittemore AD, Bandyk DF, CronenwettJL, Hertzer NR, White RA, editors. Advances in Vascular Surgery. St. Louis: Mosby, 1999;105-22.
10. Fairman RM, Nolte L, Snyder SA, Chuter TA, Greenberg RK; Zenith Investigators. Factors predictive of early or late aneurysm sac size change following endovascular repair. J Vasc Surg. 2006;43:649-56.
11. Ouriel K, Clair DG, Greenberg RK, Lyden SP, O'Hara PJ, Sarac TP, et al. Endovascular repair of abdominal aortic aneurysms: device specific outcome. J Vasc Surg. 2003;37:991-8.
12. Fujita S, Resch TA, Kristmundsson T, Sonesson B, Lindblad B, Malina M. Impact of intrasac thrombus and a patent inferior mesenteric artery on EVAR outcome. J EndovascTher. 2010;17:534-9.
13. Sampaio SM, Panneton JM, Mozes GI, Andrews JC, Bower TC, Kalra M, et al. Aneurysm sac thrombus load predictstype II endoleaks after endovascular aneurysm repair. AnnVasc Surg. 2005;19:302-9.
14. Sadek M, Dexter DJ, Rockman CB, Hoang H, Mussa FF, Cayne NS, et al. Preoperative relative abdominal aortic aneurysm thrombus burden predicts endoleak and sac enlargement after endovascular anerysm repair. Ann Vasc Surg. 2013;27:1036-41.

15. Prinssen M, Verhoeven EL, Buth J, Cuypers PW, van Sambeek MR, Balm R, et al. Dutch Randomized Endovascular Aneurysm Management (DREAM) Trial Group. A randomized trial comparing conventional and endovascular repair of abdominal aortic aneurysms. N Engl J Med. 2004;351:1607-18.

16. Wu XJ, Jin X, Zhang SY, Chong ZY, Zhang JY, Dong DN, et al. Mid-term results of endovascular aneurysm repair for infrarenal abdominal aortic aneurysm. Zhonghua Yi Xue Za Zhi. 2010;90:1743-6.

17. Powell JT, Brown LC. Thelong-term results of the UK EvarTrials: the sting in the tail. Eur J Vasc Endovasc Surg. 2010;40:44-6.

18. Mehta M, Byrne WJ, Robinson H, Roddy SP, Paty PS, Kreienberg PB, et al. Women derive less benefit from elective endovascular aneurysm repair than men. J Vasc Surg. 2012;55:906-13.

19. Greenhalgh RM, Brown LC, Kwong GP, Powell JT, Thompson SG; EVAR trial participants. Comparison of endovascular aneurysm repair with open repair in patients with abdominal aortic aneurysm (EVAR trial 1). 30-day operative mortality results: randomised controlled trial. Lancet. 2004;364:843-8.

20. Bobadilla JL, Hoch JR, Leverson GE, Tefera G. The effect of warfarin therapy on endoleak development after endovascular aneurysm repair (EVAR) of the abdominal aorta. J Vasc Surg. 2010;52:267-71.

21. Aoki A, Suezawa T, Sangawa K, Tago M. Effect of type II endoleaks and antiplatelet therapy on abdominal aortic aneurysm shrinkage after endovascular repair. J Vasc Surg. 2011;54:947-51.

22. Koole D, Moll FL, Buth J, Hobo R, Zandvoort H, Pasterkamp G, van Herwaarden JA; EUROSTAR collaborators. The influence of smoking on endovascular abdominal aortic aneurysm repair. J Vasc Surg. 2012;55:1581-6.

23. van Keulen JW, van Prehn J, Prokop M, Moll FL, van Herwaarden JA. Potential value of aneurysm sac volume measurements in addition to diameter measurements after endovascular aneurysm repair. J Endovasc Ther. 2009;16:506-13.

24. Leurs LJ, Hobo R, Buth J; EUROSTAR Collaborators. The multicenter experience with a third-generation endovascular device for abdominal aortic aneurysm repair. A report form EUROSTAR database. J Cardiovasc Surg (Torino). 2004;45:293-300.

25. Sandford RM, Bown MJ, Sayers RD, Fishwick G, London NJ, Nasim A. Endovascular abdominal aortic aneurysm repair: 5-year follow-upresults. Ann Vasc Surg. 2008;22:372-8.

26. Resch T, Ivancev K, Brunkwall J, Nirhov N, Malina M, Lindblad B. Midterm changes in aortic aneurysm morphology after endovascular repair. J Endovasc Ther. 2000;7:279-85.

27. Zarins CK, White RA, Hodgson KJ, Schwarten D, Fogarty TJ. Endoleak as a predictor of outcome after endovascualar aneurysm repair: AneuRx multicenter clinical trial. J Vasc Surg. 2000;32:90-107.

28. Tuerff SN, Rockman CB, Lamparello PJ, Adelman MA, Jacobowitz GR, Gagne PJ, et al. Aretype II (branchvessel) endoleaks really benign? Ann Vasc Surg. 2002;16:50-4.

29. van Marrewijk C, Buth J, Harris PL, Norgren L, Nevelsteen A, Wyatt MG. Significance of endoleaks after endovascular repair of abdominal aortic aneurysms: the EUROSTAR experience. J Vasc Surg. 2002;35:461-73.

30. Gould DA, McWilliams R, Edwards RD, Martin J, White D, Joekes E, et al. Aortic side branch embolization before endovascular aneurysm repair: incidence of type II endoleak. J Vasc Interv Radiol. 2001;12:337-41.

31. Veith FJ, Baum RA, Ohki T, Amor M, Adiseshiah M, Blankensteijn JD, et al. Nature and significance of endoleaks and endotension: summary of opinions expressed at an international conference. J Vasc Surg. 2002;35:1029-35.

32. Marchiori A, von Ristow A, Guimaraes M, Schönholz C, Uflacker R. Predictive factors for the development of type II endoleaks. J Endovasc Ther. 2011;18:299-305.

33. Bley TA, Chase PJ, Reeder SB, François CJ, Shinki K, Tefera G, et al. Endovascular abdominal aortic aneurysm repair: nonenhanced volumetric CT for follow-up. Radiology. 2009;253:253-62.

34. Lee JT, Aziz IN, Lee JT, Haukoos JS, Donayre CE, Walot I, et al. Volume regression of abdominal aortic aneurysm and its relation to successful endoluminal exclusion. J Vasc Surg. 2003;38:1254-63.

35. Bargellini I, Cioni R, Petruzzi P, Pratali A, Napoli V, Vignali C, et al. Endovascular repair of abdominal aortic aneurysms: analysis of aneurysm volumetric changes at mid-term follow-up. Cardiovasc Intervent Radiol. 2005;28:426-33.