의학박사 학위논문

복부 대동맥류의 혈관 내 치료 후의 동맥류 낭의 크기 감소에 관련된 새로운 예측 인자

New predictors of aneurysm sac behavior after endovascular aortic aneurysm repair

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지도교수 조용필

이 논문을 의학박사 학위 논문으로 제출함

2018년 12월

울산대학교 대학원
의 학 과
정 민 재

정민재의 의학박사학위 논문을 인준함

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Abstract

New predictors of aneurysm sac behavior after endovascular aortic aneurysm repair

Min-Jae Jeong, MD[1]; Hyunwook Kwon, MD[1]; Gi-Young Ko, MD, PhD[2]; Dong Il Gwon, MD, PhD[2]; Min-Ju Kim, MS[3]; Youngjin Han, MD[1]; Tae-Won Kwon, MD, PhD[1]; Yong-Pil Cho, MD, PhD[1]

From the [1]Division of Vascular Surgery, Departments of Surgery, [2]Radiology, and 3Clinical Epidemiology and Biostatistics, University of Ulsan College of Medicine, Asan Medical Center, Seoul, Republic of Korea

Background: The aims of this study were to identify new predictors of sac behavior after endovascular aortic aneurysm repair (EVAR) and to investigate whether the sac behavior is associated with long-term clinical outcomes.

Methods and Results: A total of 168 patients undergoing successful EVAR for abdominal

aortic aneurysms with computed tomography angiography (CTA) follow-up of at least 1 year were included. Predictors of aneurysm sac behavior and its impact on long-term clinical outcomes were retrospectively analyzed. According to the sac behavior, eligible patients were stratified into the sac regression group (n=79, 47.0%) and the sac expansion group (n=89, 53.0%). Patients in the sac regression group were younger (P=0.036) and more likely to take sarpogrelate hydrochloride at follow-up (P=0.011) than those in the sac expansion group. The incidence of postimplantation syndrome (PIS) was significantly higher in the sac regression group (P=0.005). On multivariate analysis, sac regression was more likely to occur in those with PIS (hazard ratio [HR], 1.68; 95% confidence interval [CI], 1.07–2.64; P=0.023) and less likely to occur in those with transient type II endoleaks (HR, 0.43; 95% CI, 0.20–0.95; P=0.037) and higher thrombus density within the sac on follow-up CTA (HR, 0.97; 95% CI, 0.95–0.99; P=0.013). Sac expansion was associated with significantly higher rates of reintervention during the follow-up period (P=0.001).

Conclusions: In addition to type II endoleaks, PIS and thrombus density are new predictors of aneurysm sac behavior, and sac regression is significantly associated with lower rates of

reintervention.

Key words: Aortic Aneurysm, Abdominal; Endovascular Procedures; Treatment Outcome

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Introduction

Endovascular aortic aneurysm repair (EVAR) has become the standard treatment for abdominal aortic aneurysms (AAAs).[1] The goal of EVAR is to prevent aneurysm-related death due to rupture and other complications by removing the aneurysm from the circulation.[2] A decrease or stability in the size of the AAA sac on follow-up imaging is considered a treatment success.[3] Although controversy exists about the effect of aneurysm sac behavior on long-term clinical outcomes, it is generally accepted that sac regression is a marker for the absence of the need for further intervention and of the risk of rupture during long-term follow-up, whereas sac expansion is, by itself, a marker for the potential progression of AAA and has led to indefinite periods of EVAR surveillance.[4,5] To improve the results of EVAR and simplify post-EVAR surveillance, it would be worthwhile to identify any predictive factors of sac regression.[5]

In this study, we aimed to identify new predictors of aneurysm sac behavior and to determine the association between the sac behavior and long-term clinical outcomes in patients who had undergone elective EVAR of an AAA.

Methods

Study design and patient population

This was a single-center, retrospective, observational study using data extracted from a prospectively recruiting AAA registry. The study protocol was approved by the Institutional Review Board of Asan Medical Center (2018-0288), which waived the need for informed consent because of its retrospective nature. All methods were performed in accordance with the relevant guidelines and regulations.

A total of 352 consecutive patients who underwent EVAR of an AAA at our institution from January 2008 to December 2015 were included. Patients were considered suitable for inclusion if they had undergone an initial successful EVAR for asymptomatic, uncomplicated

infrarenal AAAs with CTA follow-up of at least 1 year. Patients treated for thoraco-AAAs (n=1), symptomatic or ruptured aneurysms (n=9), or isolated iliac aneurysms (n=5) were excluded. To limit confounding factors, patients were included only if they met the criteria of treatment success, defined as the absence of the following: loss to follow-up, imaging and clinical follow-up at another institution, lack of 1-year follow-up CTA data or EVAR surveillance with duplex ultrasound imaging (n=139), and type I or III endoleaks on initial post-EVAR angiography and/or follow-up CTA (n=30). Finally, 168 patients (47.7%) were included in the analysis (**Fig 1**).

Index procedure and follow-up

EVAR was indicated when the maximum AAA diameter was at least 50 mm and/or when an increase in the maximum diameter of at least 10 mm was observed over a period of 1 year.[6] EVAR procedures were performed under general or regional anesthesia, complying with the instructions for use and following a standard vascular protocol. Our routine surveillance program included CTA and plain radiography of the abdomen within 1 month of EVAR, 6 and 12 months after EVAR, and annually thereafter to monitor aneurysm sac behavior if the

evaluations showed no abnormalities.[7] All medication adjustments were made according to each patient's atherosclerosis risk factors. The interval between the last follow-up visit and EVAR was taken to represent the follow-up duration for each patient.

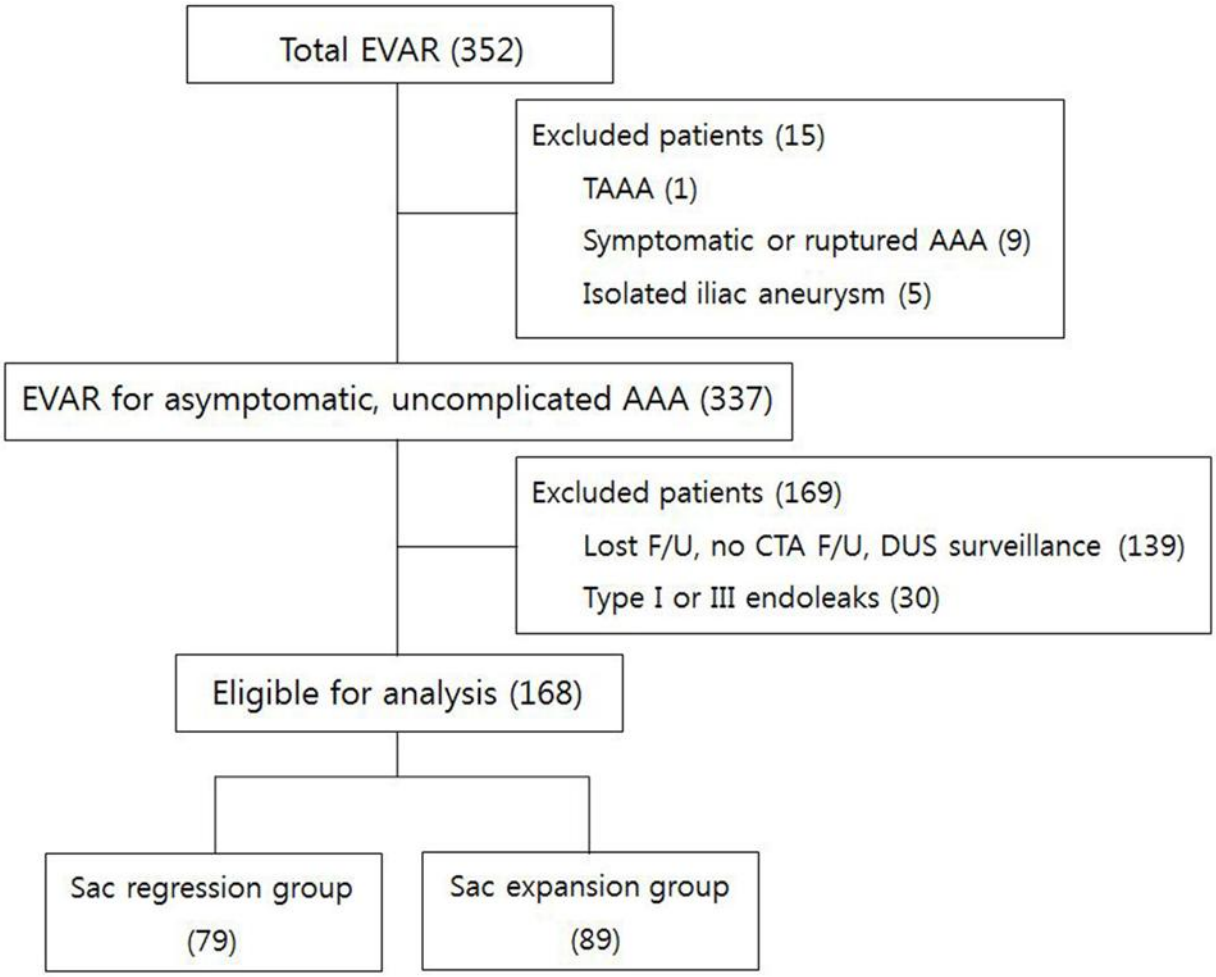

Figure 1 Flowchart of patient inclusion

A total of 168 patients who had undergone an initial successful EVAR for asymptomatic, uncomplicated infrarenal AAAs with CTA follow-up of at least 1 year were included in the analysis

Values in parentheses are number of patients. AAA = abdominal aortic aneurysm; CTA = computed tomography angiography; DUS = duplex ultrasound; EVAR = endovascular aortic aneurysm repair; F/U = follow-up; TAAA = thoraco-abdominal aortic aneurysms.

Definitions and data collection

Initial and follow-up CTAs (120 kVp, 20 mA, and 1-mm reconstructed slice thickness) were obtained after the intravenous administration of 100 mL nonionic iodinated contrast agent (Iomeprol) at a rate of 3.5 mL/s followed by a 50-mL normal saline flush at a rate of 3.5 mL/s. Image acquisition was automatically initiated once a selected threshold (100 HU) was reached at the end of descending thoracic aorta level. Thrombus density was measured in HU on arterial phase. All CTA images for each patient were evaluated by dedicated board-certified radiologists specializing in vascular imaging and intervention, and who were unaware of the patients' general health status, according to the Society for Vascular Surgery reporting standards.[6]

The maximum aneurysm sac diameter was measured in the axial imaging plane on the selected aortic section by positioning 2 calipers (external to external).[8] Aneurysm sac regression was defined as a decrease of at least 5 mm in the maximum aneurysm sac diameter from before EVAR to any post-EVAR CTA evaluation, and sac expansion was defined as an increase of at least 5 mm in the maximum sac diameter6; patients with a

change of <5 mm in either direction in maximum sac diameter were defined as having a stable sac.[2] For each patient, the change of maximal sac diameter was calculated on follow-up CTA by using the aforementioned method.

Intraoperative endoleaks were considered present if any endoleak was noted at any point during the EVAR. Per their definition,[9] endoleaks were subdivided into 2 categories: spontaneously resolved transient endoleaks and new or persistent endoleaks.[7] Transient endoleaks were defined as spontaneously resolved endoleaks at the last CTA imaging without reintervention, whereas persistent endoleaks were defined as persisting or newly developed endoleaks at the last CTA imaging.

PIS was defined in accordance with that of systemic inflammatory response syndrome as previously described.[7] The density of the thrombus within the sac was obtained by calculating the average of 3 mean absolute thrombus HU values at maximal sac diameter level and each of 3 or 4 cuts above and below this level in the follow-up CTA within 1 month of EVAR. The mean absolute thrombus HU values at each level were obtained by calculating the average of 3 regions of interest (ROIs) that were chosen at the mid-point of the aneurysm

sac and the stent graft (**Fig 2**). Thrombus HU values were quantified as described by Puig et al.[10]

Eligible patients were stratified into 2 groups according to the aneurysm sac behavior as follows: sac regression (regression group) and sac expansion or stable sac (expansion group). The regression group comprised patients who presented a decrease of at least 5 mm in the maximum sac diameter during follow-up; the remaining patients were assigned to the expansion group. Body temperature, WBC and platelet counts, and serum CRP concentrations were serially assessed 1 day before EVAR and during hospitalization, depending on the clinical status of the patient.7 Clinical outcomes were defined as all-cause mortality, aneurysm-related mortality, reintervention, late conversion to open surgery, and late rupture during the follow-up period.

Demographics, risk factors of interest, and other data, including clinical presentation and morphological characteristics of the aneurysm and clinical outcomes, were recorded for each patient. All data were prospectively collected in an Excel database (Microsoft Corp., Redmond, WA, USA) for all consecutive patients and retrospectively analyzed.

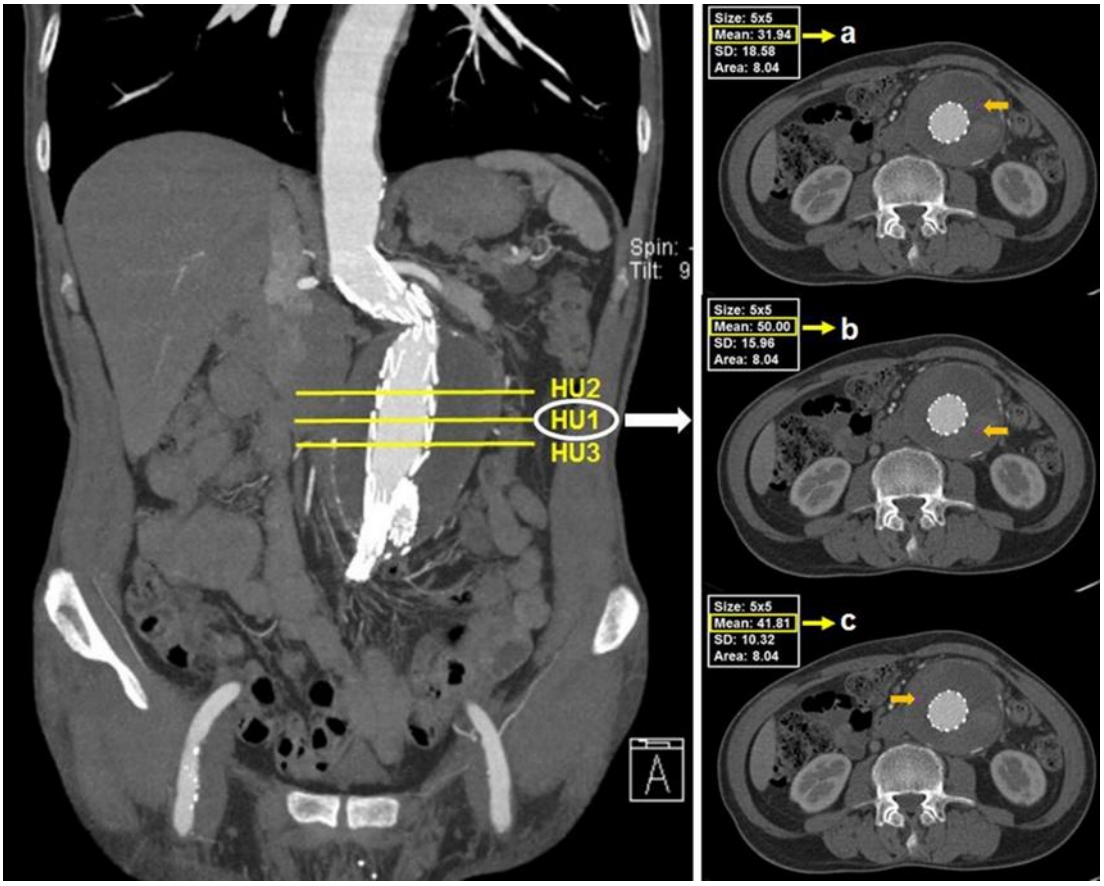

Figure 2 Measurement of the mean absolute thrombus HU values

Thrombus Hounsfield unit (HU) values were obtained by calculating the average of 3 mean absolute thrombus HU values at maximal diameter level (HU1) and each of 3 or 4 cuts above (HU2) and below (HU3) this level in follow-up computed tomography angiography. The mean absolute thrombus HU values at each level were obtained by calculating the average of 3 regions of interest that were chosen at the mid-point of the aneurysm sac and the stent graft. HU1 = (a + b + c) / 3; HU2 = (a' + b' + c') / 3; HU3 = (a'' + b'' + c'') / 3; thrombus HU value = (HU1 + HU2 + HU3) / 3.

Statistical analysis

The demographic and clinical characteristics of the patients are presented as counts and percentages for categorical variables and as means ± standard deviations for continuous variables. Categorical variables were compared using the chi-square test or Fisher's exact test, whereas continuous variables were compared using Student's t test. A Cox proportional hazards model was used to identify possible predictors of aneurysm sac regression. Because the dates of sac regression on follow-up CTA were not the same for all patients, we used a Cox proportional hazard model rather than logistical regression at a date point. Variables associated with sac regression that showed significance with a cutoff P-value of 0.1 in univariate analysis were introduced into a multivariate analysis. HRs and their 95% CIs are reported. A P-value of <0.05 was considered statistically significant. Statistical analyses were performed with SPSS version 21.0 (SPSS Inc., Chicago, IL, USA).

Results

One hundred and sixty-eight consecutive patients undergoing elective successful EVAR for AAAs with a CTA follow-up of at least 1 year were included in this analysis. The mean patient age was 71 years (range, 50–91 years), and 91.7% of the patients were men. During the mean follow-up period of 44 months (range, 12–104 months), according to the aneurysm sac behavior evaluated using post-EVAR computed tomography angiography (CTA) images, eligible patients were stratified into the sac regression group (n=79, 47.0%) and the sac expansion group (n=89, 53.0%). The regression rate in the present study was 47.0%. Among the sac expansion group, there were 22 patients with sac expansion and 67 patients with a stable sac. The baseline characteristics of the patients in relation to the aneurysm sac behavior are given in **Table 1**. There were no significant differences between the sac regression and sac expansion groups in demographics, atherosclerotic risk factors, anatomical parameters of the AAA, and graft composition, except that patients with sac regression were younger than those with sac expansion (69.6 ± 6.6 vs. 71.8 ± 6.9 years, P=0.036). Those with sac expansion were found to be less likely to smoke (73.4% vs. 59.6%,

P=0.058) and to more frequently have a prior coronary artery disease (24.1% vs. 37.1%, P=0.068). The clinical parameters are presented in **Table 2**. In relation to laboratory findings, patients with sac regression were more likely to have the highest mean white blood cell (WBC) count (P=0.010) and C-reactive protein (CRP) concentration (P=0.019) after EVAR and during hospitalization, although there were no significant differences in the baseline WBC count and CRP concentration between the 2 groups. The incidence of postimplantation syndrome (PIS) (46.8% vs. 25.8%, P=0.005) and the proportion of patients taking sarpogrelate hydrochloride (HCl) at follow-up (38.0% vs. 20.2%, P=0.011) were significantly higher in the sac regression group than in the sac expansion group.

Based on the follow-up CTA evaluations, the incidence of transient (8.9% vs. 27.0%, P=0.003) and persistent (13.9% vs. 41.6%, P<0.001) type II endoleaks was significantly lower in the sac regression group than in the sac expansion group (**Table 2**). The density of the thrombus within the sac measured with the mean Hounsfield unit (HU) values was significantly lower in the sac regression group than in the sac expansion group (43.1 ± 9.6 vs. 48.9 ± 11.4, P=0.001). During the follow-up period, all-cause mortality showed no

significant difference between the 2 groups (19.0% vs. 20.2%, P=0.840), and there was no aneurysm-related mortality. The rates of reinterventions were significantly lower in the sac regression group than in the sac expansion group (0% vs. 13.5%, P=0.001). In our analysis, there was no late conversion to open surgery or late rupture in the 2 groups.

To investigate the independent predictors of aneurysm sac regression, clinical variables associated with sac regression were analyzed using univariate and multivariate Cox proportional hazards regression analyses (**Table 3**). On univariate analysis, transient (Hazard ratio [HR], 0.37; 95% confidence interval [CI], 0.17–0.80; P=0.012) and persistent (HR, 0.29; 95% CI, 0.15–0.56; P<0.001) type II endoleaks and higher-density thrombus within the sac (higher HU values) (HR, 0.96; 95% CI, 0.94–0.98; P=0.001) were independent negative predictors of aneurysm sac regression, whereas use of sarpogrelate HCl (HR, 1.89; 95% CI, 1.20–2.98; P=0.006) and PIS (HR, 1.96; 95% CI, 1.26–3.05; P=0.003) were independent positive predictors of aneurysm sac regression. After adjustment for confounding variables, multivariate analysis indicated that sac regression was more likely to occur in patients with PIS (HR, 1.68; 95% CI, 1.07–2.64; P=0.023) and less likely to occur in those with transient

type II endoleaks (HR, 0.43; 95% CI, 0.20–0.95; P=0.037) and higher-density thrombus within the sac (higher HU values) (HR, 0.97; 95% CI, 0.95–0.99; P=0.013). Use of sarpogrelate HCl at follow-up was associated with an increased occurrence of sac regression with a marginal trend toward significance (HR, 1.60; 95% CI, 1.00–2.56; P=0.052).

Table 1 Baseline characteristics of the study population stratified by aneurysm sac behavior

	Total (n=168)	Regression Group (n=79)	Expansion Group (n=89)	*P*-value
Mean age (yr)	70.8 ± 6.8	69.6 ± 6.6	71.8 ± 6.9	0.036
Male sex	154 (91.7)	74 (93.7)	80 (89.9)	0.376
BMI (kg/m^2)	24.4 ± 3.5	24.0 ± 3.3	24.8 ± 3.6	0.147
Risk factor				
Smoking	111 (66.1)	58 (73.4)	53 (59.6)	0.058
Hypertension	117 (69.6)	56 (70.9)	61 (68.5)	0.741
Diabetes mellitus	26 (15.5)	13 (16.5)	13 (14.6)	0.741
Dyslipidemia	94 (56.0)	43 (54.4)	51 (57.3)	0.708
CKD	2 (1.2)	2 (2.5)	0 (0.0)	0.220
CAD	52 (31.0)	19 (24.1)	33 (37.1)	0.068
CVA	14 (8.3)	8 (10.1)	6 (6.7)	0.428
Malignancy	36 (21.4)	18 (22.8)	18 (20.2)	0.686
Anatomic parameter (mm)				
Neck diameter	23.2 ± 3.2	23.0 ± 3.2	23.3 ± 3.2	0.572
Neck length	35.3 ± 13.4	33.9 ± 11.9	36.6 ± 14.5	0.203
Maximal sac diameter	56.1 ± 9.6	55.7 ± 8.4	56.5 ± 10.6	0.615
Sac length	80.4 ± 25.4	79.5 ± 28.0	81.2 ± 22.9	0.664
Graft composition				0.794
Woven polyester	139 (82.7)	66 (83.5)	73 (82.0)	
Expanded TFE	29 (17.3)	13 (16.5)	16 (18.0)	

Continuous data are presented as means ± standard deviation; categorical data are given as numbers (%).

BMI = body mass index; CAD = coronary artery disease; CKD = chronic kidney disease; CVA = cerebrovascular accident; PTFE = polytetrafluoroethylene.

Table 2 Clinical characteristics of the study population stratified by aneurysm sac behavior

	Total (n=168)	Regression Group (n=79)	Expansion Group (n=89)	*P*-value
Time interval (month)*	12.5 ± 22.4	13.8 ± 23.5	17.0 ± 21.4	0.359
Laboratory data				
Baseline platelet (x10^3/uL)	199.9 ± 55.6	195.7 ± 46.3	203.7 ± 62.8	0.348
Baseline WBC (x10^3/uL)	6.7 ± 1.7	6.9 ± 1.8	6.5 ± 1.6	0.136
Baseline Cr (mg/dL)	1.0 ± 0.9	1.1 ± 1.3	0.9 ± 0.2	0.086
Highest WBC (x10^3/uL)†	11.8 ± 3.8	12.6 ± 4.2	11.1 ± 3.2	0.010
Highest CRP (mg/dL)†	11.7 ± 5.6	12.9 ± 5.4	10.7 ± 5.5	0.019
Medication at follow-up				
Statin	127 (75.6)	62 (78.5)	65 (73.0)	0.412
Single antiplatelet	58 (34.5)	28 (35.4)	30 (33.7)	0.813
Dual antiplatelet	52 (31.0)	26 (32.9)	26 (29.2)	0.605
Sarpogrelate hydrochloride	48 (28.6)	30 (38.0)	18 (20.2)	0.011
Anticoagulation	12 (7.1)	4 (5.1)	8 (9.0)	0.324
PIS	60 (35.7)	37 (46.8)	23 (25.8)	0.005
Follow-up CTA				
Transient type II endoleak	31 (18.5)	7 (8.9)	24 (27.0)	0.003
Persistent type II endoleak	48 (28.6)	11 (13.9)	37 (41.6)	<0.001
Thrombus HU values‡	46.2 ± 10.9	43.1 ± 9.6	48.9 ± 11.4	0.001
Reintervention	12 (7.1)	0 (0)	12 (13.5)	0.001
All-cause mortality	33 (19.6)	15 (19.0)	18 (20.2)	0.840

Continuous data are presented as means ± standard deviation; categorical data are given as numbers (%).

Cr = creatinine; CRP = C-reactive protein; CTA = computed tomography angiography; HU = Hounsfield unit; PIS = postimplantation syndrome; WBC = white blood cell.

* Time interval between the diagnosis of AAA and EVAR.

† The highest WBC count/CRP concentration after EVAR and during hospitalization.

‡ Thrombus HU values within the sac obtained in follow-up CTA obtained within 1 month of EVAR.

Table 3 Factors associated with aneurysm sac regression after EVAR

	Univariate analysis		Multivariate analysis	
	HR (95% CI)	*P*-value	HR (95% CI)	*P*-value
Age	0.97 (0.94–1.01)	0.095	0.98 (0.95–1.01)	0.197
Smoking	1.46 (0.89–2.41)	0.136	NA	NA
Sarpogrelate hydrochloride	1.89 (1.20–2.98)	0.006	1.60 (1.00–2.56)	0.052
Statin	1.35 (0.77–2.37)	0.296	NA	
NA				
CKD	1.12 (0.96–1.31)	0.146	NA	
NA				
Transient type II endoleak	0.37 (0.17–0.80)	0.012	0.43 (0.20–0.95)	0.037
Persistent type II endoleak	0.29 (0.15–0.56)	<0.001	NA	NA
PIS	1.96 (1.26–3.05)	0.003	1.68 (1.07–2.64)	0.023
Thrombus HU values	0.96 (0.94–0.98)	0.001	0.97 (0.95–0.99)	0.013

CI = confidence interval; CKD = chronic kidney disease; HR = hazard ratio; HU = Hounsfield unit; NA = not applicable; PIS = postimplantation syndrome.

Discussion

Although type II endoleak is a well-known predictor of aneurysm sac behavior, the main findings of this study were that PIS is a significant positive predictor of sac regression whereas a higher thrombus density within the sac is a significant negative predictor of sac regression. Use of sarpogrelate HCl is associated with sac regression with clinical significance. Moreover, our analysis showed that sac expansion is associated with significantly higher rates of reinterventions, and no patients with sac regression experienced reintervention during the follow-up period.

PIS was considered a kind of acute systemic inflammatory response after EVAR.[7] Several studies have demonstrated that there was a substantial incidence of PIS after EVAR and PIS was not associated with long-term clinical outcomes.[7,11] Recently, PIS has been proposed to have a protective effect against the development of type II endoleaks after EVAR; acute systemic inflammatory response, namely PIS, could result in a high rate of obliteration of relatively low-pressure small arteries, followed by a reduced risk of the development of type II endoleaks and rates of reinterventions during follow-up.[7] In our study, we found that PIS

was a significant positive predictor of sac regression during follow-up, although some authors suggested that there was a statistical time-related association between CRP level increase and AAA sac expansion after EVAR in the absence of proved endoleaks, and chronic systemic inflammatory response was therefore a potential critical pathogenic pathway in aneurysm progression.[12,13] In our analysis, the highest WBC count and CRP concentration after EVAR and during hospitalization, but not the baseline values and the relative rate of increase of CRP concentration, were significantly higher in the sac regression group. Although chronic systemic inflammatory response might be involved in the formation of degenerative aneurysms and sac expansion after EVAR,[14] acute systemic inflammatory response, namely PIS, could be a new predictor of aneurysm sac regression.

Several studies have demonstrated the prognostic value of the anatomical characteristics on CTA for aneurysm sac regression after EVAR[15,16]; however, little is known about the properties of the thrombus within the sac, which determine the success of sac regression in these patients. CTA detects thrombi in large arteries and provides information about thrombus composition based on HU values.[17] Based on other studies of acute ischemic stroke

concerning thrombus density,[10,18] we hypothesized that low clot density (lower HU) within the sac on post-EVAR CTA images could indicate thrombi that are more resistant to spontaneous thrombolysis, and a higher chance of successful sac regression after EVAR. In our analysis, we did not find an association between sac regression and any other anatomical characteristics; however, thrombus density, measured using HU values, was strongly predictive of successful sac regression after EVAR. However, we could not prove the exact mechanism of aneurysm sac behavior after EVAR in relation to thrombus density within the sac in the current human study, and the factor contributing to the thrombus density is unclear, even among patients without identifiable endoleaks. One possible explanation is that patients with higher thrombus density have increased graft porosity with endotension; however, we were unable to account for this issue in this analysis. There may also be patients with endoleaks not detected on follow-up CTA who had higher thrombus density and lower probability of sac regression, although the number of these patients may be extremely small.

Conflicting and limited data exist about the role of medication in aneurysm sac behavior after EVAR.[2] Although statin therapy had no effect on AAA sac regression in our analysis,

we found that the use of sarpogrelate HCl promoted sac regression with clinical significance. Sarpogrelate HCl, a 5-hydroxytryptamine2A-selective inhibitor, has been used clinically as an antiplatelet drug for preventing thrombosis in patients with atherosclerotic diseases.[19] A recent study suggested that this drug improves endothelial function and vascular remodelling.[19,20] Considering that matrix metalloproteinases have been shown to play a major role in progressive extracellular matrix degradation in the development of AAA,[21] and sarpogrelate HCl could stabilize vulnerable plaque by reducing the expression of enzymes that degrade the arterial extracellular matrix in animal models,[19] sarpogrelate HCl might be helpful in preventing AAA progression. However, the detailed mechanism by which this may occur, and whether it has antiatherosclerotic action or not, is unknown.[20] Furthermore, in our analysis, the proportion of patients taking sarpogrelate HCl was relatively small (28.6%) and the effect on sac regression did not reach statistical significance; therefore, future multicenter prospective trials with larger cohorts are warranted for a better understanding of the effect of sarpogrelate HCl on aneurysm sac behavior after EVAR.

Study Limitations

This study has several limitations. First, this was a retrospective analysis of a prospectively maintained database, and thus is subject to selection and information biases; hence, the number of excluded patients was considerable. Second, our current findings were obtained at a single center, resulting in a small sample size, which limits the overall relevance of our results. Finally, the thrombus HU values at each level were gained by calculating the average of 3 ROIs that were manually chosen at the mid-point of the aneurysm sac and the stent graft, and manual ROI placement may be subject to operator bias.

Conclusion

In addition to previously identified predictors of sac regression, we found PIS and thrombus density on follow-up CTA to be new predictors of aneurysm sac behavior after EVAR. PIS and lower thrombus density within the sac were significant positive predictors of successful sac regression, and the rates of reinterventions were significantly lower in patients with sac

regression than in those with sac expansion. Such information might prove helpful in selecting patients undergoing EVAR who should be more rigorously followed.

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국문 요약

연구 배경

본 연구에서는 복부 대동맥류의 혈관 내 치료 후의 동맥류 낭의 크기 감소에 관련된 새로운 예측인자를 확인하고 동맥류 낭의 크기 변화와 장기적인 임상결과와의 관련성에 대해 알아보고자 하였다.

연구방법 및 결과

성공적인 복부 대동맥류의 혈관 내 치료를 시행 받고 최소 1년 이상의 컴퓨터 단층촬영 혈관조영술이 확인된 총 168명을 대상으로 하였다. 동맥류 낭의 크기 감소에 관련된 예측인자와 동맥류 낭의 크기 변화가 장기적인 임상결과에 미치는 영향을 후향적으로 분석하였고, 동맥류 낭의 크기 변화에 따라 대상 환자들을 동맥류 낭의 크기가 감소된 군(n=79, 47.0%)과 증가된 군(n=89, 53.0%)로 나누어 연구를 진행하였다. 동맥류 낭의 크기가 증가된 군에 비해 감소된 군에서 환자의 연령이 낮았고 (P=0.036), 추적관찰 기간 동안 sarpogrelate hydrochloride이 처방된 환자의 수가 많았다 (P=0.011). 복부 대동맥류의 혈관 내 치료 후 발생하는 이식 후 증후군의 발현은 감소된 군에서 의미 있게 많았다 (P=0.005). Cox proportional hazards model을 사용하여 동맥류 낭의 크기 감소에 영향을 주는 인자들에 대해 다변량 분석을 하였을 때, 복부 대동맥류의 혈관 내 치료를 시행 후 이식 후 증후군이 있었던 환자에서 동맥류 낭의 크기가 의미 있게 감소되었고 (hazard ratio [HR], 1.68; 95% confidence interval [CI], 1.07- 2.64; P=0.023), 일시적인 2형 내강누출이 있었던 환자 (HR, 0.43; 95% CI, 0.20- 0.95; P=0.037)와 한달 이내에 시행된 컴퓨터 단층촬영 혈관조영술에

서 동맥류 낭의 높은 혈전 밀도를 보인 환자 (HR, 0.97; 95% CI, 0.95- 0.99; P=0.013)에서 동맥류 낭의 크기가 의미 있게 증가된 것을 확인 하였다. 또한 동맥류 낭의 크기의 증가된 군에서 추적관찰 기간 중 추가적인 시술의 비율이 의미 있게 높은 것을 확인 하였다 (P=0.001).

결론

본 연구에 따르면 복부 대동맥류의 혈관 내 치료 시행 후 이식 후 증후군의 발현과 동맥류 낭의 낮은 혈전 밀도가 동맥류 낭의 크기 감소와 관련된 새로운 예측인자로 확인되었으며 동맥류 낭의 크기 감소가 추가적인 시술의 비율을 낮추어 좋은 장기적인 임상결과를 보이는 것으로 나타났다.

중심단어

Aortic Aneurysm, Abdominal; Endovascular Procedures; Treatment Outcome