Definition of Type II Endoleak Risk Based on Preoperative Anatomical Characteristics

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Abstract

Purpose: To define the risk for type II endoleak (EII) after endovascular aneurysm repair (EVAR) based on preoperative anatomical characteristics. **Methods:** Between January 2008 and December 2015, 189 patients (mean age 78.4±7.6 years; 165 men) underwent standard EVAR. Mean aneurysm diameter was 5.7±0.7 cm and mean volume 125.2±45.8 cm^3. Patients were assigned to the "at-risk" group (n=123, 65%) when at least one of the following criteria was present: patency of a >3-mm inferior mesenteric artery (IMA), patency of at least 3 pairs of lumbar arteries, or patency of 2 pairs of lumbar arteries and a sacral artery or accessory renal artery or any diameter patent IMA; otherwise, patients were entered in the "low-risk" group (n=66, 35%). EII rates and freedom from EII reintervention were compared using Kaplan-Meier curves. Preoperative clinical and anatomical characteristics were evaluated for their association with EII and EII reinterventions using multiple logistic regression analysis; results are presented as the odds ratio (OR) and 95% confidence interval (CI). **Results:** Freedom from endoleak was lower in the at-risk group compared with the low-risk group at 36 months after EVAR (p=0.04). Freedom from EII-related reinterventions was significantly lower in the at-risk group (80% vs 100%, p=0.001) at 48 months. Based on the multiple regression analysis, the at-risk group had a higher likelihood of both EII (OR 9.91, 95% CI 2.92 to 33.72, p<0.001) and EII-related reinterventions (OR 9.11, 95% CI 1.06 to 78.44, p=0.04). These criteria had 89.4% (95% CI 83.9% to 93.2%) sensitivity and 48.0% (95% CI 40.7% to 55.3%) specificity for EII; sensitivity and specificity for EII reintervention were 100% (95% CI 93.8% to 100%) and 38.8% (95% CI 31.9% to 46.2%). Within the at-risk group, a sac thrombus volume <35% was an additional predictor for both EII (OR 5.21, 95% CI 1.75 to 15.47, p=0.003) and EII-related reinterventions (OR 8.33, 95% CI 2.20 to 31.51, p<0.002). **Conclusion:** The selection criteria effectively discriminated between low-risk patients and patients at risk for EII and associated reinterventions. A thrombus volume <35% was an additional predictor for EII and EII-related reintervention among patients at risk. These criteria may be useful for preemptively selecting patients who may benefit from EII prevention procedures or a more aggressive surveillance protocol.

Keywords

abdominal aortic aneurysm, aneurysm morphology, endoleak, endovascular aneurysm repair, inferior mesenteric artery, intrasac thrombus, lumbar arteries, reintervention, risk assessment, type II endoleak

Introduction

Type II endoleak (EII) after endovascular aneurysm repair (EVAR) continues to be the most common and also the most controversial issue in terms of outcome and need for treatment. It is described in about 10% to 30% of patients undergoing EVAR[1,2]; many of these leaks are benign, but reintervention may be required in about 10% of cases.[1,2] Furthermore, the success rate of secondary procedures is low (about 45%),[3] and some cases require multiple procedures. For these reasons, EII complications represent one of today's controversial limitations of EVAR, and studies have been performed to recognize patients considered at "high risk" and to identify reliable preoperative anatomical predictors of worse outcomes.[4–6]

However, as pointed by some authors,[7,8] EII is an enigmatic and unpredictable marker of worse outcome after EVAR. In agreement with this concept, it is our

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opinion that prevention could be effective only if the entire EVAR cohort is routinely exposed to a procedure that is effective in EII reduction without negating the advantages of EVAR in terms of number of procedures, patient exposure to radiation and contrast agents, operative time, and costs.

This new approach to EII seems to be feasible today thanks to the concept of aneurysm sac filling and stabilization during EVAR as opposed to preoperative selective feeding branch embolization. It has already been demonstrated that routine EVAR + sac embolization significantly reduces EII-related reintervention at 2 years[9]; similarly, new-generation sac-sealing endografts show promising results in terms of reducing the EII incidence during midterm follow-up.[10] However, routine use of these techniques for all abdominal aortic aneurysms (AAA) undergoing endovascular repair is not justified, not only because of limitations related to technical feasibility and instructions for use but also because it may be a waste of resources for those cases that will never develop an EII.

For these reasons a study was designed to evaluate if specific anatomical characteristics seen on the preoperative computed tomography angiogram (CTA) could reliability identify a group of AAA patients "at risk" for EII and EII-related reintervention. Furthermore, multivariate regression modeling was applied within this "at-risk" group to uncover other independent predictors, with an emphasis on aneurysm sac volume, quantity of thrombus, and numbers of feeding branches. Such a preoperative risk assessment could guide operators in judiciously applying EII prevention techniques only in a subpopulation of EVAR cases, thus optimizing resources and costs.

Methods

Study Design and Patient Cohort

The electronic medical records of a tertiary care center were interrogated to identify all patients who underwent percutaneous or open-access EVAR with bifurcated stent-grafts between January 2008 and December 2015. Patients who had associated complex endovascular procedures, such as chimney grafts, branched grafts, and fenestrated grafts, were excluded, as were patients presenting with ruptured AAA, undergoing urgent EVAR, or receiving aortouni-iliac or tubular grafts. The search identified 189 EVAR patients (mean age 78.4±7.6 years; 165 men) matching the inclusion criteria. All demographics, preoperative characteristics, perioperative outcomes, and follow-up data were extracted from the database. Baseline patient characteristics are shown in Table 1, whereas anatomical characteristics of the aneurysms are shown in Table 2. Institutional review board approval and informed consent were waived for this retrospective analysis.

Table 1. Characteristics and Risk Assessment of 189 Patients Who Underwent Standard Endovascular Aneurysm Repair.[a]

Patient characteristics	
Age, y	78.4±7.6
Men	165 (87.3)
Hypertension	164 (86.8)
Diabetes	49 (25.9)
Smoking[b]	111 (58.7)
Ischemic heart disease	83 (43.9)
Arrhythmia[c]	47 (24.9)
CRI	68 (35.9)
COPD[d]	41 (21.7)
Medical therapy	
None	16 (8.5)
Antiplatelet	102 (53.9)
Dual antiplatelet	26 (13.8)
Anticoagulant	31 (16.4)
Antiplatelet + anticoagulant	14 (7.4)
Preoperative risk assessment	
ASA score	2.9±0.3
SVS cardiac score	1.36±1.19
SVS pulmonary score	0.41±0.80
SVS renal score	0.22±0.47
SVS sum score	1.16±0.58

Abbreviations: ASA, American Society of Anesthesiologists; COPD, chronic obstructive pulmonary disease; CRI, chronic renal insufficiency (creatinine >1.5 mg/dL); SVS, Society for Vascular Surgery.
[a]Continuous data are presented as the means ± standard deviation; categorical data are given as the counts (percentage).
[b]Current and former smokers.
[c]Symptomatic or requiring treatment.
[d]Requiring medications.

Treatment

All patients underwent a standard EVAR procedure performed by the same surgeons. Three stent-grafts models were used in the majority of cases: Zenith [n=75 (40%); Cook Medical, Bloomington, IN, USA], Endurant [n=58 (30%); Medtronic, Minneapolis, MN, USA], and Excluder [n=40 (21%); W. L. Gore & Associates, Flagstaff, AZ, USA]; the remaining 16 (9%) cases were treated with different types of endografts. Intraoperative and postoperative medical therapy was the same for all patients, including intravenous heparin (5000 units) during the procedure and single antiplatelet therapy on postoperative day 1. Follow-up included CTA at 3, 6, and 12 months and then yearly. Indications for an EII-related reintervention were a >5-mm increase in AAA maximum diameter on 2 consecutive CTAs.

Imaging Assessment

Aneurysm characteristics assessed on the preoperative CTA scan (1-mm slice thickness) included maximum aneurysm

Table 2. Preoperative Aneurysm Characteristics and Anatomical Spectrum for the 189 Patients in the Study.[a]

Aneurysm sac characteristics	
Volume, cm^3	125.2±45.8
Diameter, cm	5.7±0.7
Anatomical spectrum	
Patent IMA	96 (50.8)
>3 mm	50 (26.4)
<3 mm	46 (24.3)
Patent lumbar arteries	
1	23 (12.1)
2	44 (23.3)
3	57 (30.1)
≥4	21 (11.1)
Accessory renal arteries[b]	28 (14.8)
Thrombus volume, %	
0–25	29 (15.3)
25–50	82 (43.4)
50–75	74 (39.2)
>75	4 (2.1)
Thrombus position	
Absent	15 (7.9)
Anterior	61 (32.3)
Posterior	36 (19.0)
Circumferential	23 (12.2)
Other concomitant aneurysms	
Single iliac aneurysm	8 (6.3)
Bilateral iliac aneurysm	6 (3.2)
Iliac aneurysm diameter, cm	3.1±1.1

Abbreviation: IMA, inferior mesenteric artery.
[a]Continuous data are presented as the means ± standard deviation; categorical data are given as the counts (percentage).
[b]Accessory renal arteries arising from the aneurysm or covered by the endograft.

diameter, sac volume, thrombus volume and distribution, and number and type of patent aortic sac branches. AAA dimensions were measured in standard fashion. Sac volume was calculated from the axial CTA images using Osirix Pro 4.0 software (Pixmeo SARL, Geneva, Switzerland). The same trained physician, blinded for outcomes, manually tracked the regions of interest of the aneurysm external wall every 8 mm on the axial cuts from the distal renal artery proximally to the aortic bifurcation distally. Subsequently the software generated missing regions of interest and computed aneurysm sac volume in cubic centimeters. Thrombus volume was calculated with the same method. Mean aneurysm diameter was 5.7±0.7 cm and mean volume 125.2±45.8 cm^3.

Definitions

The criteria for defining patients "at risk" for EII were selected based on the major anatomical risk factors extracted from the available literature.[4,5,6,9] These criteria were defined after data collection and before the retrospective analysis was performed to be independent from selection and reduce biases.

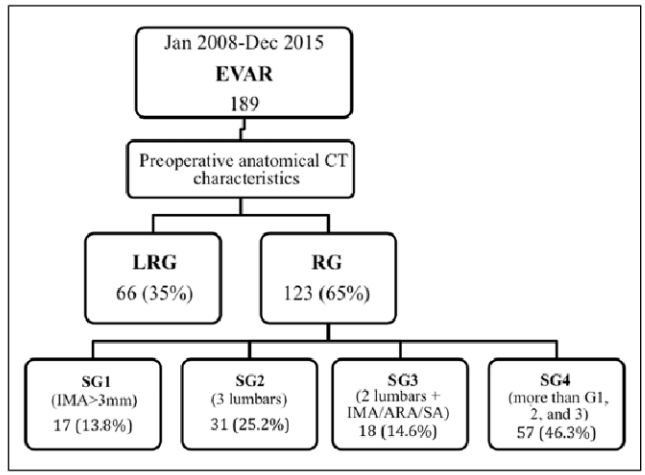

Figure 1. Schematic representation of the criteria utilized to define the risk for type II endoleak. ARA, accessory renal artery; CT, computed tomography; EVAR, endovascular aneurysm repair; IMA, inferior mesenteric artery; LRG, low-risk group; RG, at-risk group; SA, splenic artery; SG, subgroup.

The anatomical characteristics derived from the preoperative CTA selected to define an "at risk" patient were

(1) patency of the inferior mesenteric artery (IMA), with a luminal diameter at the origin ≥3 mm (subgroup 1);
(2) patency of 3 pairs of lumbar arteries (subgroup 2);
(3) patency of 2 pairs of lumbar arteries and either a sacral artery, an accessory renal artery, or any diameter (also <3 mm) patent IMA (subgroup 3); or
(4) any of the above criteria plus any patent aortic branch, eg, a patent IMA >3 mm + 3 pairs of patent lumbar arteries (subgroup 4).

Patients having at least one of these criteria were assigned to the "at-risk" group (n=123, 65%); all other patients were assigned to the "low-risk" group (n=66, 35%). Within the at-risk group, there were 17 (13.8%) patients in subgroup 1, 31 (25.2%) in subgroup 2, 18 (14.6%) in subgroup 3, and 57 (46.3%) in subgroup 4 (Figure 1).

The Society for Vascular Surgery comorbidity grading system and the American Society of Anesthesiologists score were used to assess operative comorbidity risk at baseline. Persistent EII referred to leaks present in 3 or more consecutive CT scans with any increase in aneurysm sac diameter.[11] Freedom from EII-related reintervention included patients who either did not have an EII or had an EII that did not require additional procedures.

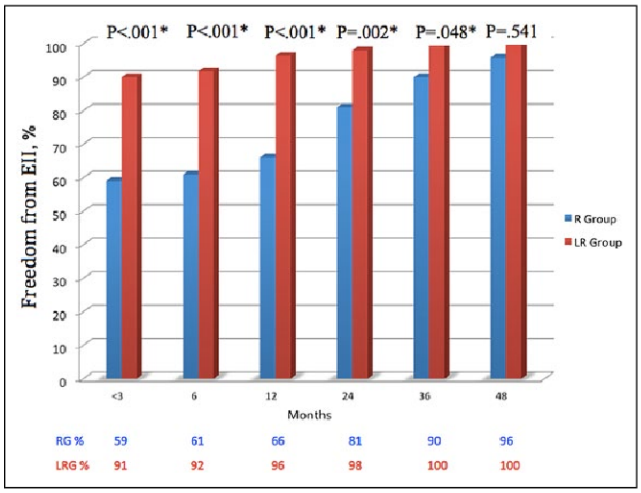

Figure 2. Comparative analysis of freedom from type II endoleak (EII) in the at-risk group (RG) and low-risk group (LRG).

Statistical Analysis

Continuous data are expressed as mean ± standard deviation and were analyzed using the Mann-Whitney *U* test. Categorical data are presented as the number (percentage) and were compared using the chi-square and Fisher exact tests. The Kaplan-Meier method was used to estimate freedom from EII-related reintervention; the log-rank test was applied to test for group comparison.

Preoperative clinical characteristics as well as the number and type of patent aortic branches (IMA, lumbar arteries, accessory renal arteries, sacral artery), sac thrombus characteristics, and sac volume were evaluated for their association with EII and EII-related reinterventions using univariate and multiple logistic regression models. Results are presented as the odds ratio (OR) and 95% confidence interval (CI). Variables with significance ($p<0.05$) in the univariate analysis were entered into the multivariate model in combination with important clinical variables and confounders in order to identify independent predictors. All statistical analysis were performed using R software (version 3.1.2; R Foundation for Statistical Computing, Vienna, Austria); $p<0.05$ was considered to indicate a statistically significant difference.

Results

Overall, the distributions of EII (48% vs 11%, $p<0.001$) and EII-related reinterventions (15% vs 0%, $p<0.001$) were significantly higher in the at-risk vs the low-risk group, respectively. Over an average 38-month follow-up (range 1–96), the EII rate was significantly higher ($p=0.048$) in the at-risk group up to 3 years (mean follow-up 39.2±21.8 months) compared with the low-risk group (mean follow-up 36.6±23.4 months; Figure 2). Based on the anatomical selection criteria, the reliability in identifying patients likely to develop EII during follow-up had a calculated sensitivity of 89.4% (95% CI 83.9% to 93.2%) and a specificity of 48.0% (95% CI 40.7% to 55.3%). The associated positive and negative likelihood ratios for EII were 1.72 (95% CI 1.42 to 2.08) and 0.22 (95% CI 0.11 to 0.46), respectively. In actuality, 89.4% of all EIIs occurred in the at-risk patients. The EII rates in the subgroups (Table 3) were not significantly different ($p=0.08$), though subgroups 1 and 4 had higher EII rates (both ~53%) compared with subgroups 2 and 3 (39% and 44%, respectively).

Table 3. Type II Endoleak (EII) and EII-Related Reintervention Rates for the Risk Groups and Subgroups.

Group/subgroup	EII	p	Reintervention	p
Low risk	7/66 (10.6)	<0.001	0/66	<0.001
At risk	59/123 (47.9)		19/123 (15.4)	
1	9/17 (52.9)	0.08	5/17 (29.4)	0.10
2	12/31 (38.7)		2/31 (6.5)	
3	8/18 (44.4)		4/18 (22.2)	
4	30/57 (52.6)		8/57 (14.0)	

The mean duration of the EII was 18.2±14.4 months in the at-risk patients and 8.2±5.4 months in the low-risk group ($p=0.07$); the number of persistent EII was significantly higher in the at-risk group [31/112 (27.6%)] vs the low-risk group [1/58 (1.7%), $p<0.001$]. Furthermore, during follow-up there were 12 (9.7%) new-onset EII in patients without endoleak at the first CTA in the at-risk group vs 1 (1.5%) in the low-risk group ($p=0.036$). The spontaneous EII resolution rate was higher in the low-risk group (5/7; 71%) compared to the at-risk group (36/59; 61%), but this was not statistically significant ($p=0.7$). None of the anatomical factors analyzed were predictors of spontaneous resolution of EII. There was no statistically significant difference in the distribution of stent-grafts used between the low-risk and at-risk groups ($p=0.68$).

The univariate analysis showed that being in the at-risk group (OR 7.1, 95% CI 3.17 to 18.24, $p<0.001$) and particularly in subgroup 4 (OR 3.55, 95% CI 1.83 to 6.98, $p<0.001$) was significant for the risk of EII. However, in the multiple logistic regression model (Table 4) for all 189 EVAR patients, only being in the at-risk group was an independent predictor of EII (OR 9.91, 95% CI 2.92 to 33.72, $p<0.001$) and EII-related reintervention ($p=0.04$). This latter result was corroborated by the Kaplan-Meier estimates of freedom from EII-related reinterventions (Figure 3), which showed a significantly higher rate of reintervention in the at-risk (19.4%, 95% CI 9% to 26%) compared to the low-risk group (0%) after 48 months ($p=0.001$). No rupture or AAA-related death was reported in either group.

Nineteen at-risk patients needed selective embolization of the feeding vessels causing the EII (including sac embolization in 5 cases) owing to aneurysm sac enlargement >5

Table 4. Final Multiple Logistic Regression Model for Type II Endoleak (EII) in the 189 Patients in the Study.

Variable	OR	95% CI	Coefficient	p
Predictors of EII				
IMA >3 mm	1.32	0.45–3.86	0.28	0.60
>3 lumbar artery pairs	1.16	0.40–3.42	0.15	0.78
Thrombus volume <35%	2.16	1.01–5.09	0.77	0.09
At-risk group	9.91	2.92–33.72	2.29	<0.001
Subgroup 4	0.41	1.16–1.14	−0.73	0.10
Predictors of reintervention				
IMA >3 mm	2.87	0.84–9.81	1.05	0.10
>3 lumbar artery pairs	1.73	0.40–7.46	0.55	0.45
Thrombus volume <35%	2.96	1.01–8.76	0.55	0.08
Aneurysm volume >200 cm^3	0.44	0.04–4.19	−0.81	0.47
At-risk group	9.11	1.06–78.44	2.21	0.04
Subgroup 4	0.31	0.08–1.20	−1.10	0.10

Abbreviations: CI, confidence interval; IMA, inferior mesenteric artery; OR, odds ratio.

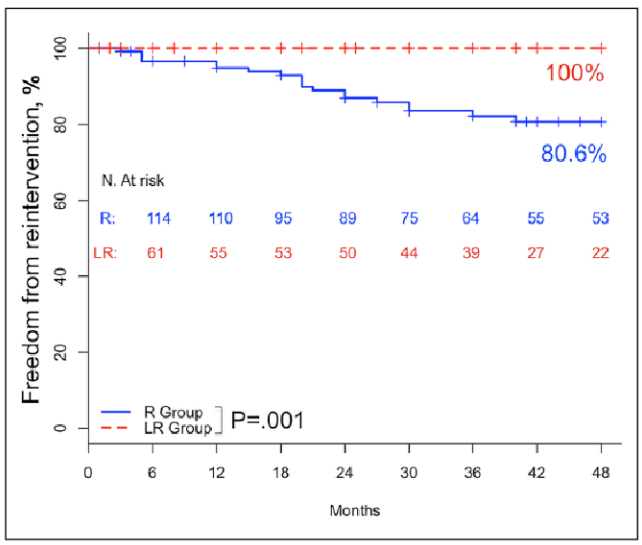

Figure 3. Kaplan-Meyer estimates of freedom from type II endoleak–related reintervention in the at-risk (R) and low-risk (LR) groups. Standard error <10% at 48 months.

mm in 17 cases and endoleak persistence with a <5-mm sac enlargement in 2. Technical success of endovascular embolization was 68.4%; 2 or more secondary procedures were required in 6 patients. Among all reinterventions, only 1 case evolved to open elective conversion because of persistent sac expansion associated with abdominal pain after 2 previous embolization attempts. Notably, no patients in the low-risk group needed reintervention during follow-up. The sensitivity and specificity of the selection criteria to identify EII-related reintervention were 100% (95% CI 93.8% to 100%) and 38.8% (95% CI 31.9% to 46.2%), respectively.

Table 5. Final Multiple Logistic Regression Model for Type II Endoleak (EII) and EII-Related Reintervention Within the At-Risk Group.

Variable	OR	95% CI	Coefficient	p
Predictors of EII				
IMA >3 mm	1.67	0.54–4.79	0.47	0.39
>3 lumbar artery pairs	0.76	0.41–1.41	−0.26	0.39
Sac volume	1.01	0.99–1.02	0.01	0.19
Thrombus volume <35%	5.21	1.75–15.47	1.65	0.003
Predictors of reintervention				
IMA >3 mm	2.08	0.61–7.15	0.73	0.24
Sac volume	1.01	1.00–1.02	0.01	0.09
Thrombus volume <35%	8.33	2.20–31.51	2.12	0.002
Subgroup 4	0.61	0.19–2.02	−0.48	0.42

Abbreviation: CI, confidence interval; IMA, inferior mesenteric artery; OR, odds ratio.

Finally, multiple logistic regression performed within the at-risk group showed that none of the analyzed clinical variables, type of medical therapy, or endograft model was a significant predictor of EII or reintervention. Among the additional anatomical criteria included in the model (Table 5), a sac thrombus volume <35% was a strong predictor of both EII (OR 5.21, 95% CI 1.75 to 15.47, p=0.003) and EII-related reintervention (OR 8.33, 95% CI 2.20 to 31.51, p=0.002).

Discussion

Endoleaks are the most common complication of EVAR and represent a frequent indication for reintervention. Although type I and III endoleaks necessitate reintervention and repair, the clinical significance of EII remains controversial. Many EII are innocuous because they resolve spontaneously. Reversal of anticoagulation is sometimes effective in promoting thrombotic occlusion of the side branches.[12]

The EUROSTAR registry showed that EII are associated with aneurysmal growth and reintervention but not with rupture or conversion to open repair.[13] Persistent EII conversely seems to be associated with an increased risk of adverse outcomes (sac enlargement, aneurysm rupture, reintervention, and conversion to open repair).[14] Recently, the Open Versus Endovascular Repair (OVER) trial reported the association of delayed EII with sac enlargement over the long term,[8] while other studies did not show this type of relationship between EII and adverse outcomes.[15,16]

As more experience with the diagnosis and treatment of EII has been reported, it is now well recognized that they are not always benign, and close surveillance is mandatory. This approach is justified because it is still unknown which EII will result in aneurysm sac expansion, thereby increasing the risk of reintervention or rupture.

Many authors have tried over 2 decades to identify reliable predictors of EII so as to apply selective prevention in those cases considered at "high risk." Arko et al[4] in 2001 identified a large patent IMA or 2 lumbar arteries as predictors of persistent EII; Sampaio et al[17] in 2005 described sac thrombus load as a predictor of EII.

Prevention applied only to "high-risk" patients using preoperative selective branch embolization has been reported by several authors employing different types of approaches and materials.[18–20] These patients are exposed to procedures that may require multiple access, longer operative time, and higher costs, which negatively impact the advantages of the EVAR procedure itself. Furthermore, these approaches may be effective in the treatment of single cases carrying an evident increased risk of developing an EII-related complication, but they do not prevent EII complications in unpredictable cases.

Our group has reported its experience with sac filling for routine EII prevention during EVAR.[9] The study demonstrated a significantly higher freedom from EII-related reintervention at 2 years in the group undergoing volume-dependent sac embolization (96% vs 82%, p=0.04) compared to standard EVAR. Recently, new devices for endovascular aneurysm sealing have been reported as a promising technology for AAA treatment. The Nellix system is the only commercially available device that incorporates endobags able to fill the free space of the aneurysm sac.[10] Independent from the technique or device utilized, the concept of sac filling and stabilization has the advantage of being applicable to all AAAs selected for endovascular repair besides guaranteeing good midterm results in terms of reducing EII and EII-related complications. With rapid advances in techniques and material, the near future may hold the opportunity for the operator to easily apply sac filling during EVAR and effectively prevent EII in the entire cohort. Nevertheless, this approach can benefit from excluding those cases that will never develop EII.

The criteria utilized in this study are based on anatomical aneurysm characteristics easily definable at the preoperative CTA. Using this risk assessment profile, two-thirds of the EVAR cases in our study were at risk of developing EII. This group had a 1.7-fold higher chance of developing EII, but even more significant was the 5-fold lower chance of EII in the low-risk group. What is really interesting is that persistent EII were more prevalent in the at-risk group, as were reinterventions. Ultimately, the only predictor of EII and EII-related reintervention was inclusion in the at-risk group. Thus, patients eligible for the low-risk group can undergo EVAR with a standard surveillance protocol, though it is still crucial to continue regular screening to exclude type I or III endoleaks. On the contrary, patients in the at-risk group may benefit from EII preventive treatment or a strict follow-up protocol.

According to our multiple logistic regression model, thrombus volume <35% was the only associated adjunctive anatomical factor, which is in line with Hiraoka et al,[21] who reported that an intraluminal thrombus volume <30% was a predictor of EII (OR 3.52, p=0.011). It is also interesting to note in our study that once a minimum number of patent aortic branches was reached, the presence of any adjunctive patent branches (subgroup 4) was not associated with an independent increased risk of EII or reintervention. A possible explanation may be that once a minimum number of aortic branches have allowed the formation of the EII, then the lack of thrombus creates a "free space" between the endograft and the sac wall that maintains the through-and-through mechanism responsible of EII persistence and sac enlargement. In this scenario, sac filling alone during EVAR, which has been demonstrated to reduce EII and reintervention rates, may play a role.

Limitations

This was a retrospective analysis, and the criteria utilized for group selection were not standardized; however, they were extrapolated from the available literature and clearly defined. Also, the number of cases is limited, and other fundamental subanalysis, such as identifying factors that may predict EII in the low-risk group, were not statistically feasible. The clinical utility of this definition of low risk is primarily related to excluding patients at low risk from the routine application of preventive technique in the EVAR population. Despite these limitations, our study included an appropriate multivariate analysis necessary to confirm the independence of predictors. Indications for secondary endovascular procedures were based on a previously established single-center protocol; causes of reintervention have also been carefully described. In addition, the midterm follow-up supports robust correlation between group risk and EII consequences over time. Finally, the classification of "EII risk" should be utilized not only to define which patients should be considered for preventive treatment but also to identify the subpopulation of EVAR patients that should be enrolled in a strict surveillance program for early EII detection or complication prevention.

Conclusion

This study was focused on the definition of anatomical criteria that could reliably identify EVAR candidates at risk for EII. The reported criteria seem to be effective in discriminating EVAR patients as "at low risk" or "at risk" of developing EII causing aneurysm expansion or requiring reintervention. In patients at risk, a thrombus volume <35% was an additional strong predictor for EII and EII-related reintervention. These criteria may be useful in identifying

those EVAR patients who may benefit from EII preventive procedures or a more aggressive EII screening protocol rather than standard surveillance.

Declaration of Conflicting Interests

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