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INTERVENTIONAL RADIOLOGY

ORIGINAL ARTICLE

Risk factors for the development of persistent type II endoleaks after endovascular repair of infrarenal abdominal aortic aneurysms

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PURPOSE
To determine, based on preoperative imaging findings, which patients treated with endovascular abdominal aneurysm repair are at high risk for the development of persistent type II endoleaks.

MATERIALS AND METHODS
Preoperative computed tomography (CT) angiographies of 136 patients, treated endovascularly, were retrospectively examined for possible anatomic predictors of type II endoleak development. Specifically, the number of patent aortic branches and thrombus load parameters (i.e., thickness, perimeter, area, and localization) were recorded. Thrombus load parameters were evaluated at the level of maximum aneurysm diameter, at the level of sac lumbar arteries' ostia, and at the level of inferior mesenteric artery ostium. Follow-up CT angiographies were also studied for the presence of persistent type II endoleaks (present at six-month follow-up examination). The association of these anatomic features with the development of persistent type II endoleaks was assessed using logistic regression analysis.

RESULTS
Aortic branch patency increased the risk for persistent type II endoleak development, while thrombus load parameters decreased this risk. In multiple logistic regression analysis, the total number of patent aortic branches (odds ratio=4.23, 95% confidence interval=1.72–10.42, $P = 0.002$ for each additional branch), and the percentage of aortic perimeter covered by a thrombus at the level of the sac lumbar arteries' ostia (odds ratio=0.16, 95% confidence interval=0.06–0.44, $P < 0.001$ for a 15% increase) were independent predictors.

CONCLUSION
Anatomic characteristics of a preoperative aorta can be used to predict patients with higher risk for persistent type II endoleak development.

Key words: • *endovascular procedures* • *abdominal aortic aneurysm* • *endoleak* • *tomography, X-ray computed*

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Endovascular aneurysm repair (EVAR) is universally accepted as an alternative to open surgery for the treatment of infrarenal abdominal aortic aneurysms because of its association with lower perioperative mortality and morbidity (1). The main drawback of EVAR is the relatively high rate of re-interventions (approximately 20%) required during the follow-up (1); endoleaks are usually the culprit responsible for these re-interventions. Type II endoleaks are most common, with a frequency varying from 10% to 25%, in EVAR procedures (2). They are caused by retrograde perfusion of the aneurysm sac from collateral aortic branches.

In the majority of cases, type II endoleaks are benign and resolve spontaneously during follow-up. However, it has been shown both clinically and experimentally that type II endoleaks are associated with increased sac pressure (3). Interestingly, in a multivariate analysis from a series of 873 patients, it was documented that type II endoleak persisting for more than six months was a significant predictor of aneurysm rupture ($P = 0.03$) (4). Considering the lack of regularity in imaging follow-up, especially on a long-term basis, it is obvious that a small but existing minority of patients with untreated type II endoleaks may suffer from aneurysm rupture.

Several studies have investigated potential correlations between preoperative characteristics of the aortic anatomy and the development of type II endoleaks following an EVAR (5–9). Although some studies failed to identify anatomic predictors for type II endoleak development (5), others with larger numbers of patients showed that preoperative patency of the aortic branches increases the possibility of type II endoleak development (6–9). Sampaio et al. (8) have not only focused on preoperative patent arteries but have also studied the aneurysm sac thrombus load in relation to type II endoleak development.

The aim of our study was to define these potentially high-risk patients that are candidates for a more intense follow-up protocol. We have specifically focused on persistent type II endoleaks (present at six months after EVAR) because these of all type II endoleaks are implicated most frequently in late adverse outcomes (4). Based on previous studies, we investigated the role of preoperative patent aortic branches and sac thrombus formation as potential predictors of persistent (longer than six months) type II endoleak development.

Materials and methods

Patient population

One hundred forty-nine patients (144 males, 5 females) with infrarenal abdominal aortic aneurysm (AAA) were treated with EVAR in our institution by a team of interventional radiologists, anesthesiologists, and vascular surgeons. Only patients that strictly adhered to the

imaging follow-up protocol for at least six months after the procedure were included in this study. Two patients died during the first postoperative month (because of myocardial infarction and stroke), four died before their six-month follow-up (two from myocardial infarctions and two from cancer), and seven more were excluded because they missed their regular follow-up examinations or because their six-month computed tomographic angiograms (CTAs) were not available. Hence, our study population consisted of 136 patients (131 males, 5 females) with a mean age of 72.3±8.6 years and a median follow-up period of 13.5 months.

Imaging protocol

All patients' AAAs were evaluated using CTA prior to the operation. CTA was performed with a 2-mm collimation thickness and using 150 mL of intravenous, low osmolarity iodine contrast medium with a flow rate of 4 mL/s. Follow-up CTAs were scheduled at 1, 6, and 12 months after the procedure and yearly thereafter. The postoperative CTA protocol was completed with acquisitions one minute after the contrast medium injection to detect type II endoleaks that appear at a delayed phase.

Patent aortic branches

In the preoperative CTA, all patent aortic branches located between the ostium of the inferior main renal artery and aortic bifurcation were recorded. These recorded arteries included the lumbar arteries, inferior mesenteric arteries (IMA), accessory renal arteries and median sacral arteries. We separately examined those branches arising from the aneurysm sac. For reasons of simplicity, the median sacral artery was studied together with sac lumbar arteries as one entity. Lumbar pairs were also independently registered.

Wall thrombus assessment

Wall thrombus was evaluated using the following parameters: A, maximum thrombus thickness; B, percentage of the aortic perimeter covered by thrombus; and C, aneurysm area occupied by thrombus expressed both as an absolute value (C1) and as a percentage (C2). These measurements were performed at three levels: at the level of maximum aneurysm diameter (MAD); at the level of the aneurysm sac lumbar arteries' ostia; and at the level of the IMA ostium.

Taking into account that lumbar arteries arise from the posterior part of the aneurysm sac, we specifically examined the thrombus lying posteriorly. The following parameters were measured at the sac lumbar arteries' ostia: D, maximum thickness of the posterior thrombus; and E, percentage of the posterior hemicycle of the perimeter covered by thrombus.

Thrombus localization was finally evaluated at the level of the MAD. Mural sac thrombus (F) was categorized as anterior, posterior, concentric or circumferential, and null (i.e., no thrombus at all or minimal thrombus).

Thrombus thickness (maximum [A] and maximum posterior [D]) at all levels was measured as shown in Fig. 1. The percentages of the perimeter (B) and posterior hemicycle (E) covered by thrombus were calculated as shown in Fig. 2. The covered thrombus area (C) was calculated by subtracting the lumen's area from the total aneurysm area at the defined levels (Fig. 3).

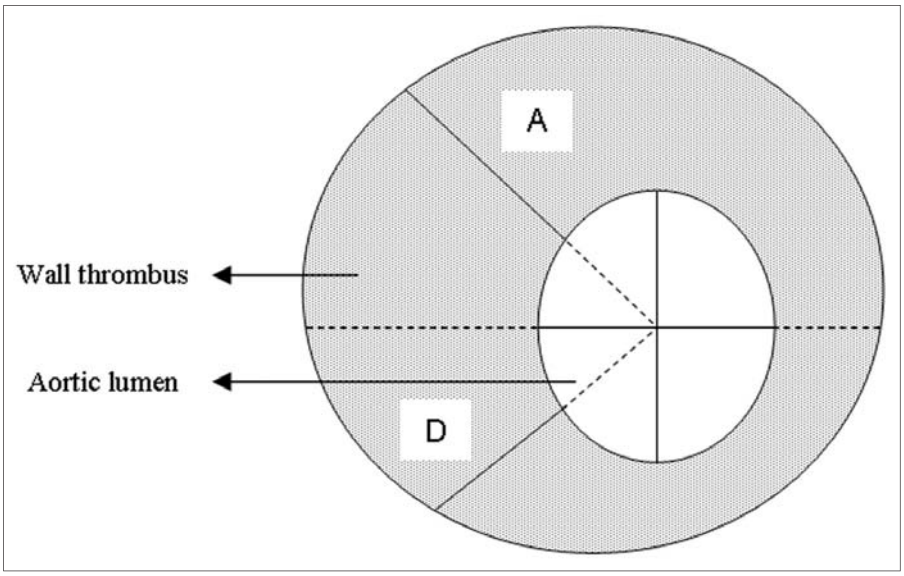

Figure 1. The maximum thrombus thickness (A) and maximum thickness of the posterior thrombus (D) were measured as the maximum extensions of aortic lumen diameter.

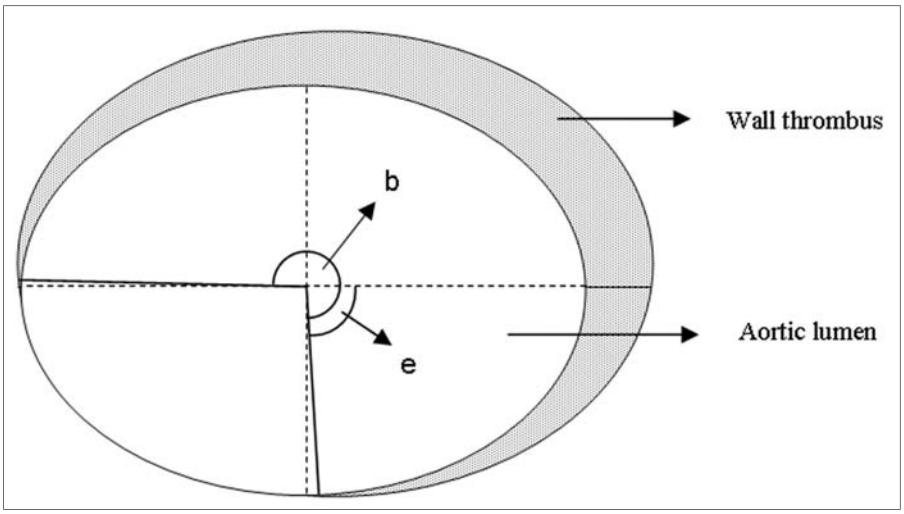

Figure 2. The percentage of aortic perimeter covered by a thrombus (B) and the percentage of posterior hemicycle covered by a thrombus (E) were estimated by measuring angle b and angle e, respectively. Their values were extracted by the equations: B=b/360×100 and E=e/180×100.

Statistical analysis

Continuous variables are presented as the means and standard deviation, while quantitative variables are presented with absolute and relative frequencies. Univariate logistic regression analysis was conducted to explore the association of type II endoleaks with preoperative characteristics. Data were modeled using stepwise multiple logistic regression analysis (i.e., *P* for removal was set at 0.1, while *P* for entry was set at 0.05) with the dependent variable indicating the presence of type II endoleak. Adjusted odds ratios (OR) with 95% confidence intervals (CI) were computed from the results of the logistic regression analyses. Model diagnostics were evaluated using the Hosmer and Lemeshow statistic.

The percentage of the perimeter covered by thrombus was further tested for its ability to predict type II endoleaks using a receiver operating characteristic (ROC) curve. The overall performance of the ROC analysis was quantified by computing the area under the curve (AUC). Using ROC analysis, the optimal sensitivity and specificity of using various cut-off values for the prediction of type II endoleaks was determined. All reported *P* values are two-tailed. Statistical significance was set at $P < 0.05$, and analyses were conducted using STATA statistical software (version 6.0, College Station, Texas, USA).

Results

One hundred thirty-six patients met our inclusion criteria and constituted our cohort. During the follow-up period, twenty-five of them (18.4%) needed reintervention: seven with type I endoleaks, nine with persistent type II endoleaks, six with graft occlusions, one with graft kinking, one with a graft infection, and one with a rupture. At six months' follow-up, we recorded 31 persistent type II endoleaks in our patient cohort (22.8%). The mean number of preoperatively patent lumbar arteries was 5.3±2, while the respective number of lumbar pairs was 2.1±1.1, and the total number of patent branches was 6.3±2.2.

Univariate logistic regression analysis showed that the likelihood for type II endoleak development increases in relation to the total number of patent aortic branches (Table 1). Specifically, for each patent aortic branch the odds for type II endoleaks increased by almost four times (OR=3.94, 95% CI=2.35–6.61, $P < 0.001$). A similar impact was found for each patent lumbar artery (OR=4.28, 95% CI=2.48–7.39, $P < 0.001$) and each patent lumbar pair (OR=5.21, 95% CI=2.71–10.01, $P < 0.001$).

Stronger correlations were found when only the vessels arising from the aneurysm sac were examined. For each patent aneurysm sac branch, the odds for type II endoleak development increased more than six-fold (OR=6.43, 95% CI=2.71–10.01, $P < 0.001$); for each patent aneurysm sac lumbar artery, the odds increased more than four-fold (OR=4.39, 95% CI=2.56–7.5); for each patent aneurysm sac lumbar pair, the odds increased more than seven-fold (OR=7.23, 95% CI=3.48–15.01).

IMA was found to be patent preoperatively in 105 of our 136 patients (77.2%). Patients with preoperatively patent IMA were at greater risk for developing persistent type II endoleaks after an EVAR procedure (OR=12, 95% CI=1.56–91.99, $P = 0.017$).

Univariate analysis also revealed that most of the studied thrombus load characteristics had a significant protective role against type II endoleak development (Table 2). An increase of 5 mm of the maximum thrombus thickness (A) at the level of sac lumbar arteries' ostia and IMA ostium was found

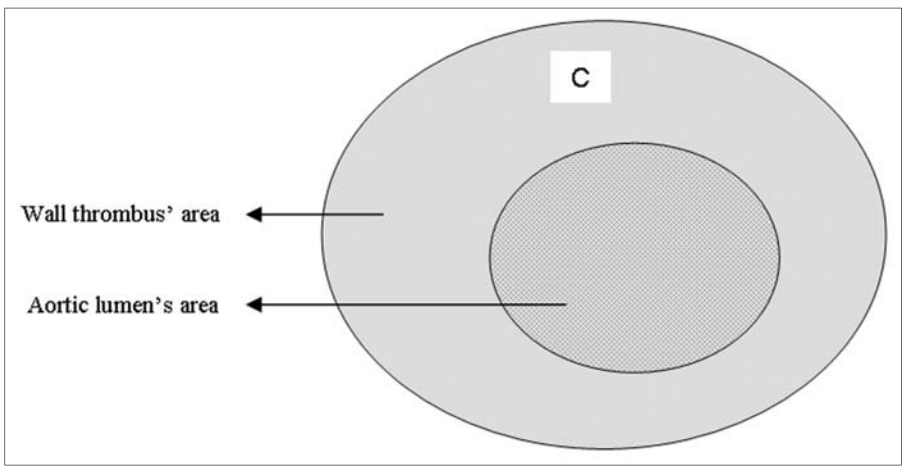

Figure 3. The thrombus-occupied area (C) was expressed as a result of subtraction of the lumen area from the aneurysm sac area.

Table 1. Type II endoleak prediction based on preoperative patency of aortic side branches (univariate analysis)

Patent aortic branches	Increment	OR (95% CI)	*P*
Patent IMA	Yes/No	12 (1.56–91.99)	0.017
Total patent aortic branches	One vessel	3.94 (2.35–6.61)	< 0.001
Patent lumbar arteries	One vessel	4.28 (2.48–7.39)	< 0.001
Patent lumbar pairs	One pair	5.21 (2.71–10.01)	< 0.001
Patent aneurysm sac branches	One vessel	6.43 (3.15–13.14)	< 0.001
Patent aneurysm sac lumbar arteries	One vessel	4.39 (2.56–7.5)	< 0.001
Patent aneurysm sac lumbar pairs	One pair	7.23 (3.48–15.01)	< 0.001

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

Table 2. Type II endoleak prediction using computed tomographic characteristics of aneurysm thrombus load (univariate analysis)

Thrombus load	Increment	OR (95% CI)	*P*
Maximum thrombus thickness at the level of MAD	5 mm	0.85 (0.7–1.04)	0.114
Mean maximum thrombus thickness at the level of sac lumbar arteries' ostia	5 mm	0.62 (0.45–0.86)	0.004
Maximum thrombus thickness at the level of IMA ostium	5 mm	0.85 (0.64–1.11)	0.017
Mean maximum thickness of the posterior thrombus at the level of sac lumbar arteries' ostia	5 mm	0.25 (0.12–0.54)	< 0.001
Percent perimeter covered by thrombus at the level of MAD	25%	0.17 (0.08–0.36)	< 0.001
Mean percent perimeter covered by thrombus at the level of sac lumbar arteries' ostia	15%	0.11 (0.05–0.23)	< 0.001
Percent perimeter covered by thrombus at the level of IMA ostium	5%	0.87 (0.8–0.94)	< 0.001
Mean percent posterior hemicycle covered by thrombus at the level of sac lumbar arteries' ostia	25%	0.04 (0.01–0.13)	< 0.001
Area covered by thrombus at the level of MAD	500 mm^2	0.86 (0.69–1.07)	0.177
Mean area covered by thrombus at the level of sac lumbar arteries' ostia	500 mm^2	0.56 (0.37–0.86)	0.008
Area covered by thrombus at the level of IMA ostium	300 mm^2	0.94 (0.77–1.15)	0.557
Percent area covered by thrombus at the level of MAD	15%	0.75 (0.57–0.98)	0.033
Mean percent area covered by thrombus at the level of sac lumbar arteries' ostia	15%	0.59 (0.42–0.81)	< 0.001
Percent area covered by thrombus at the level of IMA ostium	10%	0.90 (0.74–1.10)	0.312
Thrombus localization at MAD level	Posterior (reference)/ Anterior	2.05 (0.8–5.27)	0.137
Thrombus localization at MAD level	Posterior (reference)/ Not posterior	2.16 (0.88–5.26)	0.092

OR, odds ratio; CI, confidence interval; MAD, maximum aneurysm diameter; IMA, inferior mesenteric artery

to decrease the likelihood of type II endoleak development (OR=0.62, $P < 0.004$; and OR=0.85, $P < 0.017$; respectively).

The percentage of the aortic perimeter covered by thrombus (B) was also negatively correlated with persistent type II endoleaks. Specifically, a 25% increase of the percentage of the perimeter covered by thrombus at the level of the MAD, a 15% increase at the level of sac lumbar arteries' ostia, and a 5% increase at the level of IMA ostium decreased the odds for type II endoleak development (OR=0.17, $P < 0.001$; OR=0.11, $P < 0.001$; OR=0.87, $P < 0.001$; respectively).

The area of the aneurysm sac that was covered by thrombus (C) was additionally found to protect against type II endoleak development. An increase of 15% of the percentage area covered by thrombus (C2) at the level of the MAD and a 15% increase at the level of sac lumbar arteries' ostia decreased the odds for type II endoleak development by 25% and 41%, respectively (OR=0.75, $P = 0.033$; OR=0.59, $P < 0.001$; respectively). No significant correlation was found at the level of IMA ostium.

When examining the amount of thrombus of the posterior aneurysm wall, our results showed it clearly played a protective role against type II endoleak development. An increase of 5 mm of the maximum thickness of the posterior thrombus (D) at the level of sac lumbar arteries' ostia was found to decrease the likelihood of type II endoleak development (OR=0.25, $P < 0.001$). A 25% increase in the percentage of the posterior hemicycle covered by thrombus at the level of the sac lumbar arteries' ostia also significantly decreased the odds of type II endoleak development (OR=0.04, $P < 0.001$). Thrombus localization at the MAD level (F) was found to have no significant correlation with type II endoleaks.

When multiple logistic regression analysis with a stepwise method was performed, it was found that the percentage of the aortic perimeter covered by thrombus (B) at the level of sac lumbar arteries' ostia and the total number of patent aortic branches were independent predictors of type II endoleak development. Specifically, for a 15% increase in the percentage of the thrombus-covered perimeter, the odds for type II endoleak development decreased by 84% (OR=0.16, 95% CI=0.06–0.44, $P < 0.001$). Moreover, for each additional patent aortic branch, the odds for type II endoleak development increased four-fold (OR=4.23, 95% CI=1.72–10.42, $P = 0.002$). For example, in patients with five patent aortic branches, the risk for persistent type II endoleak development was less than 2%, while in patients with 10 patent aortic branches the risk was more than 90% (Fig. 4).

ROC curve analysis showed that a thrombus-covered perimeter value of 66.67% has the best accuracy in the prediction of protection against type II endoleak development. Specifically, our ROC curve analysis (Fig. 5) showed that a thrombus-covered perimeter value of 66.67% is optimal for the prediction of type II endoleaks, having a sensitivity of 96.2% and specificity of

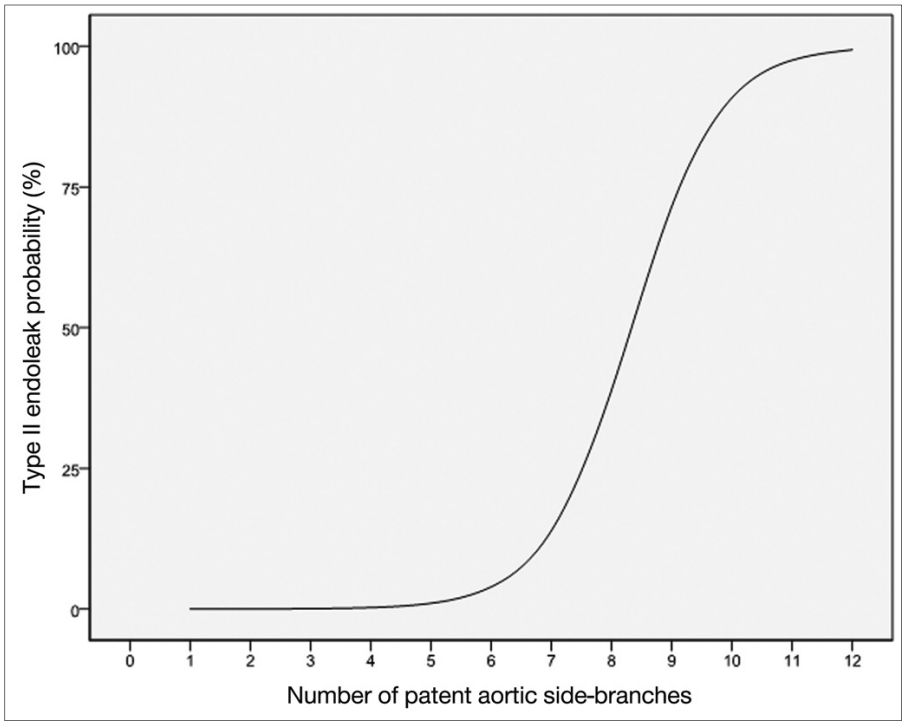

Figure 4. A quantitative correlation of the total number of patent aortic branches with the probability (%) of developing a persistent type II endoleak (extracted by multivariate analysis).

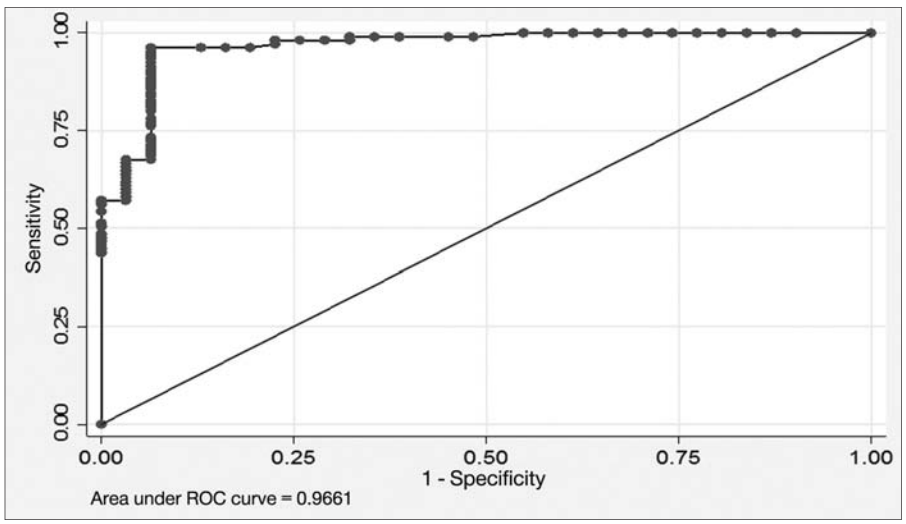

Figure 5. ROC curve defines the percentage of the perimeter covered by a thrombus (B) at sac lumbar arteries' ostia for the prediction of type II endoleaks with maximum sensitivity and specificity.

93.5%. The AUC value was 0.97 (95% CI=0.93–1.00, $P < 0.001$).

Discussion

Although the treatment was introduced more than 15 years ago, type II endoleak development after an EVAR procedure is still a debated issue. The incidence of such endoleaks remains high, according to recent multicenter randomized trials (1). Additionally, they do not seem to be dramatically influenced on a long-term basis by new developments in stent graft technology (10). Most of the endoleaks are reported as transient and resolve spontaneously during follow-up. Nevertheless, there is evidence that persistent type II endoleaks for at least six months after an EVAR may be associated with aneurysm sac growth and potential rupture (11). Hence, predictability based on preoperative imaging parameters would be useful in defining high-risk patients that should be subjected to a frequent follow-up protocol after an EVAR.

Management of patients with persistent type II endoleaks is another issue of controversy (12). A prudent approach consists of close imaging-based follow-up of these patients. If aneurysm sac expansion occurs, however, they should be considered for a more aggressive management protocol, including translumbar sac thrombosis or feeding vessel embolization. Jones et al. (4) have evaluated the late outcomes associated with type II endoleaks. They found that patients with persistent endoleaks were at increased risk for aneurysm sac growth (OR=25.9) and also had a significantly increased rate of reintervention (OR=19.0) vs. patients without an endoleak. Freedom from rupture at five years for patients with a persistent type II endoleak was 91.1% compared to 97.4% for patients without a type II endoleak. The authors concluded that patients with a persistent type II endoleak (at more than six months) should be considered for more frequent follow-up or a more aggressive approach to reintervention. However, Rayt et al. (13) advocate surveillance for type II endoleaks even in patients with sac enlargement.

Previous studies have also investigated the role of patent aortic side branches in type II endoleak development. It should be noted, however, that not all studies agree on which patent aortic branches are suitable predictors of type II endoleak development. Gorich et al. (6) examined a relatively small sample of patients (7 type-II endoleaks in 68 EVAR patients) and managed to associate an increased risk of early type II endoleak with four or more patent lumbar arteries in the preoperative CTA without finding an association with the patency of other sac branches. Fan et al. (7) studied immediate (i.e., at 72 hours) postoperative endoleaks and found that a patent IMA was a predisposing risk factor for type II endoleak development. In addition, they found that when zero to three sac lumbar arteries were patent, the type II endoleak rate was 13%, while when more than six were patent, the type II endoleak rate was 50%. Sampaio et al. (8), studying type II endoleaks observable after the 30th postoperative day, found that

IMA patency is positively correlated with type II endoleak development (OR=6.84, $P < 0.01$). Univariate analysis also showed that the risk for type II endoleak development increased with the number of aortic side branches (OR=1.36 for each additional patent aortic branch, $P = 0.002$). The number of patent branches remained an independent predictor (OR=1.31 for each additional patent aortic branch, $P = 0.009$) in the multivariate analysis. In a more recent study, Warrier et al. (9) were able to identify the patency of IMA, but not of the lumbar arteries, as a predictor for type II endoleak development. Our study demonstrated a clear positive correlation (Table 1) of preoperative patency of aortic branches with persistent (i.e., greater than six months) type II endoleak development. A patent IMA increased the odds by twelve-fold. The risk (i.e., OR) was 3.94 or 4.28 times greater for each additional patent aortic branch or patent lumbar artery, respectively. When patent lumbar pairs were examined, it was found that the risk for a type II endoleak increased 5.21 times for each patent lumbar pair. This risk association was even stronger when only branches arising from the sac were examined: 6.43, 4.39, or 7.23 times greater risk for each sac branch, sac lumbar artery, or sac lumbar pair, respectively, was observed. According to our multiple logistic regression analysis, the total number of patent aortic branches was demonstrated to be a positive independent predictor (OR=4.23, 95% CI=1.72–10.42, $P = 0.002$ for each additional branch).

Armon et al. (14) tried to correlate thrombus with the presence of type II endoleaks without achieving any statistical significance. However, they implied that there is a protective role of a thick circumferential and posteriorly lying thrombus. In our study, an aneurysm thrombus was clearly found to provide protection against persistent type II endoleak development (Table 2). The thickness of a thrombus and the percentage of the aortic sac perimeter that was covered by a thrombus, especially at the levels of IMA and sac lumbar arteries' ostia, were inversely correlated with persistent type II endoleak development. According to our univariate analysis, posterior thrombus load at sac lumbar arteries' ostia was also significantly protective; the mechanism of this protection is not known. We believe that the presence of a thrombus at the side branch ostium may prevent retrograde filling of the aneurysm sac. In addition to that "occluding effect," an old sac thrombus may provide less empty space to be filled by collateral retrograde flow.

To our knowledge, the quantitative protective effect of thrombus load has been previously reported only by Sampaio et al. (8). They independently correlated the percentage of the thrombus-covered area at the level of the MAD (OR=0.74 for a 10% increase, $P < 0.0005$) with the development of type II endoleaks after the 30th postoperative day. Although our results differ in that we found that the percentage of the thrombus-covered perimeter at sac lumbar arteries' ostia (OR=0.16 for a 15% increase, $P < 0.001$) was an independent predictor of type II endoleaks after the sixth postoperative month, both studies have shown that thrombus load may be measured, and the risks for type II endoleak development can be estimated with confidence.

The percentage of the sac area covered by a thrombus was also inversely associated with persistent type II endoleak development in our univariate analysis. This finding was significant at the level of the MAD (OR=0.75 for 15% increase, $P = 0.033$) and at the level of sac lumbar arteries' ostia. However, measurement of the area is more time-consuming than measurement of the percentage of the perimeter that can be easily calculated from the axial CTA images without the use of any sophisticated software (Fig. 2). Our ROC curve analysis (Fig. 5) defined the value of our independent thrombus predictor with the best sensitivity and specificity to be 66.67% (i.e., the value of aortic thrombus-covered perimeter at the level of sac lumbar arteries' ostia).

The main limitation of our study is its weakness to determine preoperatively which patients will need postoperative intervention. Consequently, we are not able to suggest an interventional method of preventing type II endoleaks, such as intraoperative embolization of lumbar arteries, IMA, or the aortic sac itself. All of these techniques have been attempted (15), but the facts that this strategy is time-consuming and that the results are not encouraging have limited this practice from becoming a standard procedure. However, the importance of defining preoperative risk factors for type II endoleaks is crucial for the possible prevention of aneurysm sac expansion or rupture by applying a closer imaging follow-up protocol for high-risk patients.

In summary, we have investigated the predictive value of anatomic features in the preoperative CTAs of 136 EVAR patients with a 22.8% persistent (i.e., greater than six months) type II endoleak rate. Our results corroborated previous efforts that associated aortic branches' patency with type II endoleaks and added the aspect of thrombus formation as a protective predictor. We specifically demonstrated a practical, easy-to-measure, thrombus-load predictor and defined its cut-off value with maximum sensitivity and specificity.

In conclusion, we have identified preoperative imaging parameters of the aorta that seem to predict persistent type II endoleak development. The total number of patent aortic branches and the percentage of the aortic perimeter covered by a thrombus at the level of sac lumbar arteries' ostia are independent positive and negative predictors, respectively. A value of 66.67% of the latter predicts, with high probability, against the development of persistent type II endoleaks.

Conflict of interest disclosure

The authors declared no conflicts of interest.

References

1. The EVAR trial participants. Endovascular aneurysm repair versus open repair in patients with abdominal aortic aneurysm (EVAR trial 1): randomized controlled trial. Lancet 2005; 365:2179–2186.
2. Baum RA, Stavropoulos SW, Fairman RM, Carpenter JP. Endoleaks after endovascular repair of abdominal aortic aneurysms. J Vasc Interv Radiol 2003; 14:1111–1117.
3. Schurink GW, Aarts NJ, Wilde J, et al. Endoleakage after stent-graft treatment of abdominal aneurysm: implications on pressure and imaging: an in vitro study. J Vasc Surg 1998; 28:234–241.
4. Jones JE, Atkins MD, Brewster DC, et al. Persistent type 2 endoleak after endovascular repair of abdominal aortic aneurysm is associated with late adverse outcomes. J Vasc Surg 2007; 46:1–8.
5. Back MR, Bowser AN, Johnson BL, Schmacht D, Zwiebel B, Bandyk DF. Patency of infrarenal aortic side branches determines early aneurysm sac behavior after endovascular repair. Ann Vasc Surg 2003; 17:27–34.

6. Gorich J, Rillinger N, Sokiranski R, et al. Endoleaks after endovascular repair of aortic aneurysm: are they predictable? Initial results. Radiology 2001; 218:477–480.
7. Fan CM, Rafferty EA, Geller SC, et al. Endovascular stent-graft in abdominal aortic aneurysms: the relationship between patent vessels that arise from the aneurismal sac and early endoleak. Radiology 2001; 218:176–182.
8. Sampaio SM, Panneton JM, Mozes GI, et al. Aneurysm sac thrombus load predicts type II endoleaks after endovascular aneurysm repair. Ann Vasc Surg 2005; 19:302–309.
9. Warrier R, Miller R, Bond R, Robertson IK, Hewitt P, Scott A. Risk factors for type II endoleaks after endovascular repair of abdominal aortic aneurysms. Aust N Z J Surg 2008; 78:61–63.
10. Sheehan MK, Ouriel K, Greenberg R, et al. Are type II endoleaks after endovascular aneurysm repair endograft dependent? J Vasc Surg 2006; 43:657–666.
11. van Marrewijk CJ, Fransen G, Laheij RJ, Harris PL, Buth J and EUROSTAR Collaborators. Is a type II endoleak after EVAR a harbinger of risk? Causes and outcome of open conversion and aneurysm rupture during follow up. Eur J Vasc Endovasc Surg 2004; 27:128–137.
12. Maldonado T, Gagne P. Controversies in the management of type II endoleaks following endovascular abdominal aortic aneurysm repair. Eur J Vasc Endovasc Surg 2003; 37:1–12.
13. Rayt HS, Sandford RM, Salem M, Bown MJ, London NJ, Sayers RD. Conservative management of type 2 endoleaks is not associated with increased risk of aneurysm rupture. Eur J Vasc Endovasc Surg 2009; 38:718–723.
14. Armon MP, Yusuf SW, Whitaker SC, Gregson RH, Wenham PW, Hopkinson BR. Thrombus distribution and changes in aneurysm size following endovascular aortic aneurysm repair. Eur J Vasc Endovasc Surg 1998; 16:472–476.
15. Gould DA, McWilliams R, Edwards RD, et al. Aortic side branch embolization before endovascular aneurysm repair: incidence of type II endoleaks. J Vasc Interv Radiol 2001; 12:337–341.