Preoperative predictive factors of aneurysmal regression using the reporting standards for endovascular aortic aneurysm repair.

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ABSTRACT

Background: Aneurysmal regression is a reliable marker for long-lasting success, following endovascular aneurysm repair (EVAR). The aim of this study is to identify the preoperative factors which can predictably lead to aneurysmal sac regression following EVAR, according to the reporting standards of the Society of Vascular Surgery and the International Society of Vascular Surgery (IVS/ISCVS).

Methods: From a total of 199 patients treated by EVAR between 2000 and 2009, 164 completed computerized tomography angiographies (CTA) and duplex scan follow-ups. Moreover, for any patient to be enrolled in this retrospective study, all of his/her CTAs were required to be analyzable with Endosize software (Therenva®, Rennes, France), in order to provide spatially correct three-dimensional data in accordance with the recommendations of the IVS/ISCVS. All anatomic parameters were graded according to the relevant severity grades. From these grades, a severity score was calculated at the aortic neck, the AAA and the iliac arteries. Clinical and demographic factors were also studied. Patients with aneurysmal regression > 5 mm were assigned to group A, and the others were assigned to group B.

Results: Aneurysmal regression occurred in 66 (40.2%) patients (group A). The mean age was 71.4 ± 8.9 years in group A, and 76.3 ± 8.3 in group B. Univariate analyses showed smaller severity scores at the aortic neck (p=0.02) and the iliac arteries (p=0.002) in group A. In group A, calcifications and thrombus were less significant at the aortic neck (p=0.003 and p=0.02) and at the iliac arteries (p=0.001 et p=0.02), and inferior mesenteric artery patency was less frequent (68.2% vs 82.7%, p=0.04). Two multivariate analyses were carried out, of which one considered the scores, and the other was based on the variables included in the scores. In the first, the patients of group A were younger (p=0.002) and aortic neck calcifications were less significant (p=0.007). In the second, the patients of group A were younger (p<0.001) and the aortic neck scores were smaller (p=0.04). There was no difference between the two groups, in terms of the implanted endoprosthesis, nor in the follow-up (46.4 ± 24 months in group A, and 47.2 ± 22 months in group B, p=0.35).

Conclusion: In this study, the young age of the patients and their aortic neck quality, in particular the absence of neck calcification, appear to have been the main factors affecting aneurysm regression, such that they represent a target population for the improvement of EVAR results.

INTRODUCTION

Endovascular aneurysm repair (EVAR) of an abdominal aortic aneurysm (AAA) does not systematically lead to regression of the AAA sac[1]. However, such regression is a reliable marker for long-term success[2-6]. Regression of the sac is, in particular, a marker for the absence of further surgery, and for the absence of rupture during follow-up. In order to improve the results achieved with EVAR, it would seem logical to try to identify any predictive factors for sac regression. The role of prostheses has been incriminated, particularly in the case of first generation prostheses[7-12]. Anatomic factors were also studied. It appears that less favorable anatomies lead to poorer results[13, 14]. However, in most studies the description of these anatomic factors does not comply with the reporting standards[15], the publishing of which was intended to standardize the outcomes of studies dealing with EVARs. This description has the advantage of analyzing a large number of anatomic factors, and of providing a sufficient level of detail. An exhaustive and more accurate description of these factors, in patients presenting with AAA regression, would perhaps also allow EVAR candidates to be more rigorously selected. The aim of the present study was to identify the preoperative clinical and anatomic factors, which are predictive of aneurysmal regression following EVAR, in accordance with the recognized reporting standard.

MATERIAL AND METHODS

From a total of 199 consecutive patients operated in our unit for an infrarenal AAA, between January 2000 and December 2009, 164 were included in the retrospective study. Image analysis was carried out using the Endosize[16] software (Therenva®, Rennes, France). It was thus possible to make 3D angular measurements and to compute the tortuosity indices according to the reporting standards' recommendations. Whenever the preoperative CT image could not be analyzed with this software, it was consequently not possible to carry out all of

the measurements according to the reporting standards, and these patients were excluded from the study. If the follow-up was incomplete, the patients were also excluded. Finally, 164 patients (147 men, 17 women) could be included in this analysis and in total, 164 aortic necks and aneurysms and 327 iliac arteries were analyzed (one patient had a single iliac occlusion).

The patients were operated on using the endovascular technique, whenever they were not eligible for open repair[17] and whenever the aneurysm diameter was greater than 50 mm, or if its growth rate was greater than 1 cm per year, or if it was painful. Infected, inflamed and ruptured aneurysms were excluded, as were patients operated with a fenestrated or branched endoprosthesis. A preoperative CT angiography was required before surgery, and at 1, 6, 12, 18 and 24 months of follow-up, after the operation. The patients were then followed-up, with images being taken every year, alternately by CT angiography or ultrasonography. For the purposes of the study, the time interval between the last CT image and the operation was taken to represent the follow-up duration of each patient. The mean interval was 46.8 ± 22.6 months (minimum 18 months; maximum 120 months).

Anatomic factors. All of the measurements were made perpendicularly to the centerlines, which were extracted automatically (Fig. 1). For the preoperative scan, in addition to the measurements generally required before ordering an endograft, other parameters were measured and sorted according to the reporting standards[15]. The maximum AAA diameter was always measured at the same cutting level for all of the scans. Each anatomic parameter was classed according to four grades of severity, from which three anatomic severity scores were computed (Table 1). The aortic neck score was the sum of the grades determined for the thrombus, diameter, length, calcifications, and aortic neck angulation. The AAA score was the sum of the grades determined for the thrombus, the angle, and the T1 ratio (tortuosity index of the aorta, i.e. the ratio of the length of the aorta from the renal arteries to the aortic bifurcation, taken along the central line, to the length of a straight line between these points)

of the AAA collaterals. The iliac score was the sum of the diameter grades, the length, the T2 ratio (Fig. 1) (tortuosity index of the iliac axis, taken from the aortic bifurcation to the common femoral artery, computed as for the aortic index), the thrombus, the calcifications and the minimum diameter of the iliac axes.

Non-anatomic factors. In addition to the anatomic parameters, the clinical factors (Table 2), follow-up duration, type of implanted endoprosthesis, and installation (aorto-bi-iliac or uni-iliac) were considered. The patients' clinical factors were classed according to the recommendations[15] (Table 2)

Sub group. Group A was comprised of patients who, during follow-up, presented with > 5 mm^2 [2, 15, 18] regression of the aneurysmal sac. The remaining patients were assigned to group B.

Statistical Analysis. The data is presented in the form of mean ± standard deviation for quantitative variables, unless otherwise noted, and in the form of numbers with corresponding percentages for qualitative variables. The predictive factors (clinical and anatomical) for sac regression were studied using univariate analyses, which were computed using the Kaplan-Meier method and the log-rank test, since the follow up dates were not the same for all patients. Anatomic severity scores were considered as ordinal variables and compared with the Mann-Whitney test. Variables found to be less than 0.1 in the univariate analysis were included in a multivariate analysis, implemented using a Cox model. A stepwise descending procedure was carried out. Two multivariate analyses were carried out, with the variables considered separately or grouped together in scores. We checked the PH assumption for all covariates of the two models using a graphical approach, by plotting the logarithms of the cumulative probabilities. Between endografts, the rate of regression was compared using the Fischer exact test. The rate of endoleaks between group A and B, as the comparison of the

anatomic factors related to endoleaks were also computed using the Kaplan-Meier analysis and the log-rank rank test. Correlation between age and the evolution of the maximum AAA diameter was assessed with the Pearson correlation coefficient. All analyses were performed with the Statistica software 6.0 version (Statsoft, Oklahoma, USA), and the statistical level of significance was 5%.

RESULTS

Demographics. The clinical characteristics of the studied population are provided in Table 3. The regression rate in the present study was 40.2% (group A, n=66). The regression>5 mm of the AAA was observed on the postoperative CTA at 15.9 ± 10 months. Using univariate analysis, age was the only significant factor (Table 1, $P < 0.001$). The Kaplan-Meier curves showed that there is a significant difference in regression rate (P=0.005) between the four age severity grades (Fig. 2) (Table 3). Regression occurred most frequently when the patients' age corresponded to grade 1 (between 55 and 70 years the regression rate was 58%). For the other patients, this rate was 33% for those in grade 0, 46% in grade 2, and 23% for those in grade 3. The postoperative variation of the AAA diameter was found to be correlated with age (Fig. 3) (P=0.001).

Anatomic factors. The anatomic descriptions are summarized in Table 3.. Concerning the aortic neck, calcifications and thrombus were less severe in group A (P=0.003 and P=0.02, respectively). The best regression rate for aortic neck calcifications and thrombus corresponded to grade 0 (51% and 49% respectively). Similarly, the severity score for the aortic neck was lower in group A (Table 4) (3.5 ± 2.3 in group A and 4.4 ± 2.5 in group B, P=0.05). For the AAA score, as well as for all of the parameters used to establish this score, there was no significant difference between the groups. When the inferior mesenteric artery (IMA) was considered as a separate variable (not included in the aortic branches) the patency

of the inferior mesenteric artery (IMA) was lower in group A (P=0.04). In the case of the iliac arteries, calcifications and thrombus were less severe in group A (P=0.001 and P=0.02, respectively). The severity score for the iliac arteries was lower for group A (6.5 ± 2.1 in group A and 7.7 ± 2.3 in group B, P=0.002).

Non anatomic factors. The implanted endoprosthesis distribution is summarized in Table 5. There was no significant difference between the implanted prostheses in the two groups (P=0.40). Seven (10.6%) aorto-uni-iliac endoprostheses were implanted in group A, and 15 (15.3%) in group B (P=0.47). The follow-up duration was 46.4 ± 24 months in group A and 47.2 ± 22 months in group B (P=0.35).

Multivariate analysis. In the first multivariate analysis, the age was lower in group A (P=0.002) and aortic neck calcifications were less severe (P=0.007). The hazard ratios (HR) and 95% confidence intervals (CI) were 0.96 (95% CI: 0.94-0.98) for age and 0.60 (95% CI: 0.41-0.87) for aortic neck calcifications. In the second analysis, the patients were also younger in group A (HR 0.96 [95% CI: 0.94-0.98], P<0.001) and the aortic neck score was lower (HR 0.87 [95% CI: 0.78-0.97], P=0.04).

Postoperative follow-up

Group A was associated with a lower rate of all types of endoleak (Table 6). The occurrence of a type Ia endoleak during follow up was associated with a higher preoperative aortic neck severity score (Table 7). Variables at the aortic neck which were significantly more severe among patients with a type Ia endoleak during follow-up were thrombus (P=0.01) and calcifications (P=0.01). The occurrence of a type Ib endoleak during follow up was associated with more complex iliac anatomies especially in terms of angle, tortuosity index, calcifications and thrombus (Table 8). Patients with a type Ib endoleak had a lower iliac severity score than the others (P=0.001) (Table 8). No difference was found in the aneurysm

related factors between patients with a type II endoleak during follow-up and others patients. When the IMA was considered as a separate variable, the rate of IMA patency was higher among patients with a type II endoleak (P=0.008).

DISCUSSION

Reporting standards have been published, with a view to normalizing EVAR-related data and allowing studies to be compared. Indeed, the description of calcifications or thrombus on the aortic neck varies widely from one study to another. It can be limited to a binary quantification (present or absent) or, at the other extreme, be segmented with a highly accurate quantification of the volume of each segment. Studies reporting on regression of the sac require the analysis of numerous variables. For example, the iliac arteries are rarely included in the analysis factors for aneurysmal regression. However, they can be the source of complications, which compromise regression as a consequence of a preoperative pathology. For this reason, we carried out an exhaustive analysis of all of the preoperative anatomic parameters. We modified some of the risk scores described in the reporting standards since we wished, for example, to unite all of the variables relating to iliac arteries into one single anatomic severity score. Two multivariate analyses were thus necessary, since it was our aim not only to accurately identify each individual factor, but also to evaluate the overall anatomic zone (aortic neck, AAA and iliac arteries) on a global basis, i.e. through the use of a single score.

In this study, age appears to have a non negligible influence on regression of the aneurysmal sac. In most studies, this outcome has not been clearly identified as a factor influencing regression. Quite commonly, various authors have tried to identify factors influencing the progression of the sac following EVAR, and have found an advanced age to be a risk factor, in particular in the meta-analysis of Schanzer *et al.*[19] in which patients having an age > 80

years is one of these factors, and is well correlated with the reporting standard severity grade[15] (grade 3). The same result was found by Ouriel *et al.*[20], who recognized that a lower regression rate was found when the patients' age increased. Although the definitions for regression can vary, Houballah *et al.*[21] found a lower mean age in the group of patients who presented with sac regression. Since age was not found to be a predictive factor for regression in all studies, we tried to corroborate our results using survival analyses, which appear to be the most appropriate approach when the history of each patient is different. It is nevertheless important to note that the highest regression rate is not found in the patients belonging to grade 0. However, a small number of patients (6) in our study had this grade. Moreover, analysis of survival age reveals a difference, especially between grades 1 and 3 and we found a significant correlation between age and the postoperative evolution of the AAA diameter. This outcome thus suggests that there is a relationship between age and postoperative sac evolution, in agreement in agreement with the findings of other authors[19-21]. An explanation for the influence of age on regression could lie in the fact that arterial compliance decreases with age[22], and that calcifications are partly responsible[23]. Furthermore, young patients often have less arterial calcification[24] such that, although it has not been scientifically demonstrated, it would be reasonable to expect that the regression capacity of an aneurysm is partly dependent on these factors, in the absence of any endoleaks. Although we did not quantify calcifications of the AAA itself, it has been shown that the degree of calcification of an AAA is a predictive factor of the natural history of small AAAs[25], which again supports the possible role of calcifications in the sac regression process. In addition, in our study calcification is one of the significant predictive factors for regression, in univariate and multivariate analysis. However, one of the limitations of this study is that of quantifying calcifications. Although we adhered to the reporting standards, it is clear that the evaluation of calcification, based simply on the analysis of four quadrants from a transverse cut, is insufficient for correct evaluation of

the degree of calcification in a given anatomic zone. Segmentation algorithms with better performance, which would not be particularly useful in routine applications, such as EVAR planning, should nevertheless be implemented into the software, in order to provide more accurate quantification.

Concerning the anatomic factors assessed in our study, it is not surprising that in the multivariate analyses, we systematically found the aortic neck to be a significant variable. Here, the absence of calcifications (grade 0) appears to have a particular influence on sac regression. Their presence clearly compromises the proximal attachment zone and increases the risk of an endoleak as shown by the comparison between patients with or without a type Ia endoleak during follow-up. Although the other parameters related to the aortic neck are non significant (except for the case of thrombus, in univariate analysis), it is interesting to observe that the aortic neck score is significant. Taking all factors into account appears to be a more strongly influential element on sac regression, than each parameter considered individually. This is also true and largely accepted in the case of the risk of postoperative complications[26]. Although it is not the case in the present study, in most studies the length of the aortic neck is the factor having the greatest influence on postoperative changes in diameter[9, 14, 19]. The presence of thrombus on the aortic neck has also been identified by other authors as being a factor contributing to non-regression[13, 27]. The indication for an EVAR on complex aortic necks, using current endoprostheses, thus increases the risk of non-regression, such that the manufacturers' instructions must be adhered to, if a satisfactory result is to be achieved. Schanzer *et al.* [19] have revealed a significant rate of disregard for these indications, which could explain the high rate of aortic neck growth observed in their study, which is the largest existing meta-analysis on this subject.

The size and thrombus of an AAA have also been analyzed in several studies, with differing results. In our study, the size and the thrombus of the AAA do not appear to have an influence

on its regression. On the one hand, some authors[11, 28] did not find any influence of preoperative diameter on sac regression, and on the other hand, it appears that the larger the aneurysm, the less it regresses[21, 29]. Finally, Greenberg *et al.*[10] found that the AAAs which are the largest at the surface have the greatest regression in absolute and relative terms. Concerning aneurysmal thrombosis, Yeung *et al.*[27] found that the absence of preoperative thrombosis, associated with the absence of an early endoleak, was a good indicator for sac regression. However, when there is no thrombus in the sac, the IMA as well as a high number of lumbar arteries are often permeable, which is a factor for a type II endoleak[30, 31]. In our study, the permeability of the IMA appears to have an influence on sac regression in univariate analysis and on the occurrence of a type II endoleak. Preoperative embolization could then be considered as a complementary intervention, which would increase the rate of sac regression following EVAR. However, Nevala *et al.*[10] have shown that there was a decrease in the rate of an early type 2 endoleak, but no increase in sac regression. In the present study, we find that patients with a permeable IMA have a lower probability of having a sac regression due to a greater probability of developing a type II endoleak whereas when aneurysm sac collaterals were graded according to the reporting standard, no difference is observed. This result can be explained by the fact that the majority of our patients was classed in severity grades 2 or 3, which may highlight one of the limitations of this grading system, indicating that it could be too severe for the description of the AAA collaterals. Finally, as in our own study, Blankenstein *et al.*[33] did not find an influence of preoperative aneurysmal thrombus on sac regression.

The last point, which has also been studied by several authors, is the influence of the endografts. Here, we were not able to demonstrate any difference between endografts, probably because the vast majority of implanted devices are represented by two different endografts models (Talent and Zenith). Indeed, very few first generation endografts were

implanted, since most of our unit's activities began at the time when these two models were available on the market. Although Ouriel *et al.*[7] found very significant differences between endografts in their series, the Talent and Zenith models had the same regression rate. Greenberg *et al.*[10] found higher regression rates with the Zenith, as compared to the Ancure and Excluder models. Bertges *et al.*[11] found the highest regression rates with the Talent model, at 1 and 2 years, as compared to the Ancure, Excluder and Aneurx models. The Talent and the Zenith are thus likely to be the endografts having the best regression rates or having no statistically significant difference in our series. This result was also found by Badger *et al.* who compared these two endografts[34].

Several limitations can be found in our series. It is a retrospective study, and a certain number of operated patients could not be included in our analyses, which constitutes a selection bias. In addition, the follow-up dates were not the same for all of the patients, which is the reason for which we conducted survival analyses rather than logistical regressions at a date point. Finally, the AAA diameter measurement method and the threshold for defining regression are also subjects of debate. Wever *et al.* [35] have shown that volumetry was more appropriate for the follow-up of EVARs, but when the maximum diameter varies by ± 6%, the correlation with volume is correct. By choosing a threshold of 5 mm[2, 15, 18] on the aneurysm, the mean diameter of which was 55.9 mm in our series, a variation of at least 10% is found, which is considered by several authors to be a reliable figure. In addition, volumetry is not a routine measurement and requires time and the availability of an appropriate workstation. Houballah *et al.*[21] have proposed another definition for shrinkage, with the aim of identifying it as a sensitive and specific marker for successful treatment, on the basis of this definition.

CONCLUSION

The use of the reporting standard and the anatomic severity grades provides a reliable tool for the description of objective anatomic criteria before EVAR and in our opinion should be more widely used in studies dealing with EVAR. In the present study, age seems to be influent on aneurysmal sac evolution. Younger patients could have a higher rate of aneurysmal sac regression than other patients because of a lower rate of endoleaks. The quality of the aortic neck, in particular the absence of calcification, appears to be a determinant factor. The best long-term EVAR results probably occur in this population of relatively young patients having a good anatomy. New endoprostheses (Endurant, Medtronic and Zenith LP, Cook), which are designed for more complex anatomies, will perhaps allow such results to be obtained.

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Table 1. Definition of each anatomic grade according to the reporting standard and the severity scores used

	Anatomic severity grades			
Attribute	0=Absent	1=Mild	2=Moderate	3=Severe
Aortic Neck				
Length (L1), mm	L1>25	15<L1<25	10<L1<15	L1<10
Diameter (D1), mm	D1<24	24<D1<26	26<D1<28	D1>28
Angle (A1), °	A1>150	150< A1<135	135< A1<120	A1<120
Calcifications (Ca1), %	Ca1<25	25 <Ca1<50	Ca1>50	
Thrombus (Th1), %	Th1<25	25 <Th1<50	Th1>50	
Aortic Neck Severity Score (/15)	= L1+D1+A1+Ca1+Th1 grades			
Aneurysm				
Tortuosity index (T1)	T1<1.05	1.05<T1<1.15	1.15<T1<1.2	T1>1.2
Aortic angle (A2), °	160<A2<180	140<A2<159	120<A2<139	A2<120
Thrombus (Th2), %	0	Th2<25	25<Th2<50	Th2>50
Aortic branches (AB)	None	1 lumbar/IMA	2 vessels<4 mm	2 vessels >4 mm
Aneurysm Severity Score (/12)	=T1+A2+Th2+AB grades			
Iliac artery				
Calcification (Ca2), %	None	Ca2<25	25< Ca2<50	Ca2>50
Thrombus (Th3), %	Th3<25	25<Th3<50	Th3>50	
Length (L2), mm	L2<30	20< L2<30	10< L2<20	L2<10
Diameter (D2), mm	D2<12.5	12.5< D2<14.4	14.5< D2<17	D2>17
Tortuosity index (T2)	T2<1.25	1.25<T2<1.5	1.5<T2<1.6	T2>1.6
Iliac angle (A3), °	160<A3<180	121<A3<159	90<A3<120	<90
Access diameter (AD), mm	AD>10	8<AD<10	7<AD<8	AD<7
Iliac artery Severity Score (/21)	=Ca2+Th3+L2+D2+T2+A3+AD grades			

IMA, inferior mesenteric artery

Table 2. IVS/ISCVS medical comorbidity grading system

Major components	
Cardiac status	
Grade 0	Asymptomatic, with normal electrocardiogram
Grade 1	Asymptomatic but with either remote myocardial infarction by history (6 months), occult myocardial infarction by electrocardiogram, or fixed defect on dipyridamole thallium or similar scan
Grade 2	Any one of the following: stable angina, no angina but significant reversible perfusion defect on dipyridamole thallium scan, significant silent ischemia (1% of time) on Holter monitoring, ejection fraction 25% to 45%, controlled ectopy or asymptomatic arrhythmia, or history of congestive heart failure that is now well compensated
Grade 3	Any one of the following: unstable angina, symptomatic or poorly controlled ectopy/arrhythmia (chronic/recurrent), poorly compensated or recurrent congestive heart failure, ejection fraction less than 25%, myocardial infarction within 6 months
Pulmonary status	
Grade 0	Asymptomatic, normal chest radiograph, pulmonary function tests within 20% of predicted
Grade 1	Asymptomatic or mild dyspnea on exertion, mild chronic parenchymal radiograph changes, pulmonary function tests 65% to 80% of predicted
Grade 2	Between 1 and 3
Grade 3	Vital capacity less than 1.85 L, FEV1 less than 1.2 L or less than 35% of predicted, maximal voluntary ventilation less than 50% of predicted, PCO2 greater than 45 mm Hg, supplemental oxygen use medically necessary, or pulmonary hypertension
Renal status	
Grade 0	No known renal disease, normal serum creatinine level
Grade 1	Moderately elevated creatinine level, as high as 2.4 mg/dL
Grade 2	Creatinine level, 2.5 to 5.9 mg/dL
Grade 3	Creatinine level greater than 6.0 mg/dL, or on dialysis or with kidney transplant
Minor components	
Hypertension	
Grade 0	None (cutoff point, diastolic pressure usually lower than 90 mm Hg)
Grade 1	Controlled (cutoff point, diastolic pressure usually lower than 90 mm Hg) with single

	drug
Grade 2	Controlled with two drugs
Grade 3	Requires more than two drugs or uncontrolled
Age	
Grade 0	<55 years
Grade 1	55-69 years
Grade 2	70-79 years
Grade 3	>80 years

Table 3. The clinical and anatomical variables (graded according to the reporting standard) are compared between group A and B by an univariate analysis (log-rank test).

	Grade 0		Grade 1		Grade 2		Grade 3		
	n/N	Mean ± SD*	n/N	Mean ± SD*	n/N	Mean ± SD*	n/N	Mean ± SD*	P
Medical comorbidities									
Cardiac status	11/29	13 ± 8	18/44	14 ± 8	21/56	15 ± 11	16/35	20 ± 11	.97
Pulmonary status	7/29	14 ± 6	37/76	14 ± 9	18/48	18 ± 6	4/11	17 ± 8	.36
Renal status	36/92	17 ± 8	21/46	16 ± 7	8/24	19 ± 7	1/2	18	.68
Hypertension	7/16	17 ± 9	10/28	19 ± 8	37/96	16 ± 8	12/24	18 ± 9	.78
Age	2/6	12	21/36	15 ± 7	30/66	16 ± 11	13/56	18 ± 9	.005
Anatomic factors									
Aortic neck (N=164)									
Diameter	36/87	13 ± 7	16/33	15 ± 6	9/32	16 ± 4	5/12	17 ± 3	.41
Angle	30/70	17 ± 11	19/57	16 ± 9	12/28	17 ± 8	5/9	15 ± 4	.72
Length	26/68	12 ± 11	21/51	13 ± 11	19/45	18 ± 6	-	-	.51
Thrombus	39/79	14 ± 8	14/38	15 ± 7	10/34	14 ± 7	3/13	15 ± 3	.02
Calcifications	48/94	14 ± 7	14/41	17 ± 6	3/26	19 ± 7	1/3	18	.003
Aneurysm (N=164)									
Tortuosity	21/39	16 ± 6	35/103	17 ± 9	6/12	16 ± 7	4/10	19 ± 2	.31
Angle	11/26	13 ± 6	28/62	17 ± 9	23/65	17 ± 5	4/11	18 ± 3	.45
Thrombus	3/9	17 ± 9	11/25	16 ± 7	29/49	19 ± 2	23/81	18 ± 3	.20
Aortic branches	-	-	7/15	18 ± 6	34/75	17 ± 5	25/73	16 ± 2	.29
Iliac arteries (N=327)									
Diameter	48/84	12 ± 8	22/91	15 ± 6	33/91	19 ± 7	29/60	17 ± 8	.44
Length	131/325	16 ± 13	1/2	18	-	-	-	-	.81
Angle	0/3	-	69/154	15 ± 8	61/137	16 ± 6	11/33	18 ± 7	.81
Tortuosity	20/38	17 ± 8	22/54	14 ± 7	2/13	19	1/5	18	.35

Thrombus	69/141	11 ± 7	42/128	16 ± 8	11/48	17 ± 8	5/10	16 ± 3	.02
Calcifications	53/94	15 ± 8	59/147	16 ± 8	20/77	15 ± 8	0/10	-	.001
Minimum	10/35	13 ± 11	82/183	17 ± 10	23/74	15 ± 7	18/35	16 ± 9	.48

n/N, number with sac regression/total

*delay (in months) of the occurrence of the regression>5mm

Table 4. Comparison of the anatomic severity score between group A and B.

	Total population (n=164)	Group A (n=66)	Group B (n=98)	P
Aortic neck severity score (/15)	4 ± 2.4	3.5 ± 2.3	4.4 ± 2.5	0.02
Aneurysm severity score (/12)	6.9 ± 1.9	6.6 ± 1.8	7.1 ± 1.9	0.06
Iliac artery severity score (/21)	7.2 ± 2.5	6.5 ± 2,1	7.7 ± 2.3	0.002

Table 5. Endoprosthesis implanted in each group.

	Group A	Group B
Talent (Medtronic)	28 (42%)	62 (63%)
Zenith (Cook)	31 (47%)	25 (26%)
Excluder (Gore)	4 (6%)	6 (6%)
Anaconda (Vascutek)	3 (5%)	1 (1%)
Vanguard (Boston scientific)	0	1 (1%)
Endologix (Bard)	0	1 (1%)
Aneurx (Medtronic)	0	2 (2%)
P value*	0.40	

*from the Fischer exact test

Table 6. Rates of endoleaks in group A and B (log-rank-test).

	Total (n=164)	Group A (n=66)	Group B (n=98)	P
Type Ia endoleak	13 (7.9%)	1 (1.5%)	12 (12.2%)	0.012
Type Ib endoleak	7 (4.3%)	0	7 (4.3%)	0.032
Type II endoleak	33 (20.1%)	4 (6.1%)	29 (29.6%)	<0.001

Table 7. Comparison of the anatomic severity scores between patients with or without an endoleak during follow-up

	Endoleak	No endoleak	P
Aortic neck severity score (/15) –Type Ia endoleak (n=10)	6.9 ± 2.6	3.8 ± 2.3	<0.0001
Aneurysm severity score (/12)-Type II endoleak (n=33)	7.2 ± 1.7	6.8 ± 1.8	0.33
Iliac artery severity score (/21)-Type Ib endoleak (n=7)	9.3 ± 2.2	7.1 ± 2.3	0.001

Table 8. The anatomical variables are compared between patients with or without an endoleak during follow-up by an univariate analysis (log-rank test).

	Grade 0		Grade 1		Grade 2		Grade 3		
	n/N	Mean ± SD*	n/N	Mean ± SD*	n/N	Mean ± SD*	n/N	Mean ± SD*	P
Aortic neck-Type Ia endoleak									
Diameter	0/87	-	1/33	12	8/32	54 ± 20	4/12	36 ± 24	.10
Angle	0/70		5/57	31 ± 30	3/28	20 ± 12	5/9	25 ± 7	.10
Length	2/68	24	5/51	36 ± 11	6/45	44 ± 35	-	-	.08
Thrombus	0/79	-	2/38	41	5/34	48 ± 12	6/13	22 ± 7	.01
Calcifications	0/94	-	0/41	-	11/26	19 ± 7	2/3	18	.01
Aneurysm-Type II endoleak									
Tortuosity	11/39	5 ± 6	15/103	6 ± 8	3/12	6 ± 7	4/10	6 ± 4	.75
Angle	4/26	5 ± 9	12/62	6 ± 4	13/65	7 ± 10	4/11	4 ± 4	.44
Thrombus	3/9	3 ± 3	6/25	6 ± 7	7/49	4 ± 4	17/81	7 ± 9	.25
Aortic branches	-	-	2/15	3	15/75	3 ± 4	16/73	7 ± 9	.21
Iliac arteries-Type Ib endoleak									
Diameter	1/84	21	1/91	28	2/91	51	3/60	24 ± 17	.14
Length	6/325	34 ± 27	1/2	28	-	-	-	-	.06
Angle	0/3	-	1/154	48	2/137	21	4/33	18 ± 7	.01
Tortuosity	0/38	-	2/54	24	2/13	19	3/5	18 ± 8	<0.001
Thrombus	0/141	-	1/128	36	5/48	28 ± 8	1/10	48	.02
Calcifications	0/94	-	0/147	-	5/77	31 ± 16	2/10	29	<0.001
Minimum	1/35	21	3/183	26 ± 12	2/74	21	1/35	37	.26

n/N, number with endoleak/total

*Delay (in months) of the occurrence of the endoleak

Figures

Figure 1. Aorto-iliac mesurements with Endosize: iliac tortuosity index measurement.

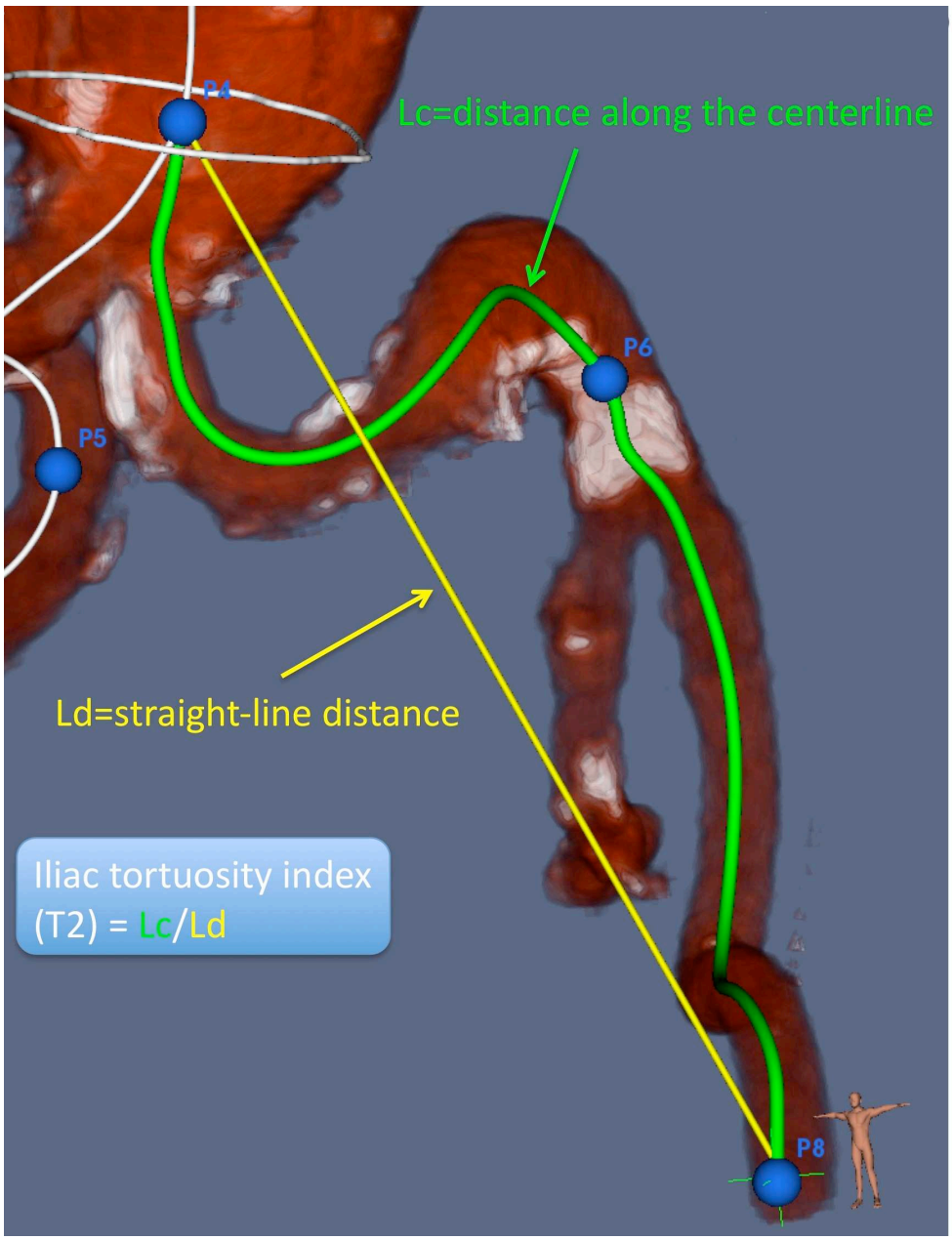
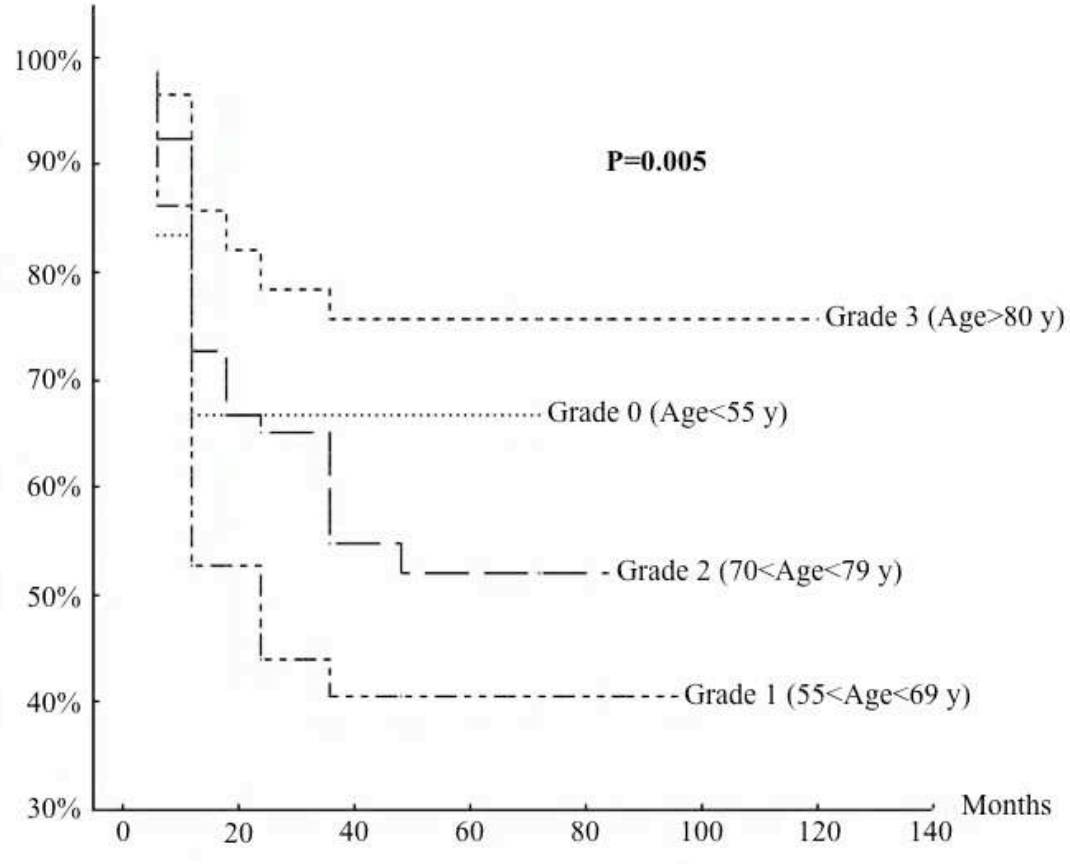


Figure 2. Proportion of patients with aneurysm shrinkage (>5mm) according to the grading age defined in the reporting standard.



	Months	6	18	36	48	60	72	84	96	120
Grade 0	N at risk	6	3	2	2	2	2	0		
	Regression[a]	2	0	0	0	0	0	0		
	Censorship	1	1	0	0	0	2	0		
	%[b]	100	67	67	67	67	67	67		
Grade 1	N at risk	35	17	12	9	6	2	1	1	0
	Regression	17	3	1	0	0	0	0	0	0
	Censorship	1	2	2	3	4	1	0	1	0
	%	100	51	41	37	37	37	42	42	42
Grade 2	N at risk	78	49	45	27	18	11	4	0	
	Regression	25	2	6	1	0	0	0	0	

	Censorship	4	2	12	8	7	7	4	0	
	%	100	67	65	55	53	53	53	53	
Grade 3	N at risk	45	35	24	12	10	7	4	1	1
	Regression	7	1	1	0	0	0	0	0	0
	Censorship	3	10	11	2	3	3	3	0	1
	%	100	84	81	77	77	77	77	77	77

[a]number of patients with sac regression, [b]percent of patients without sac regression

Figure 3. Correlation between age and postoperative variation of the AAA diameter