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Endoleaks after endovascular aneurysm repair lead to non-uniform intra-aneurysm sac pressure

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Abstract

Objective: to study intra-aneurysm sac pressures in patients who developed endoleaks after endovascular repair (EVAR) of abdominal aortic aneurysms (AAA).

Methods: 25 patients with endoleaks (18 men, 7 women; 76 (68-80) years old) underwent 31 direct intra-aneurysm sac pressure measurements (DISP) 16 (14-26) months after EVAR (AAA diameter 59 (52-67) mm). Six patients underwent DISP twice. Tip-pressure sensors were used through direct translumbar puncture of the AAA except in three patients (transabdominal puncture in 2 and endoluminal in 1). Mean pressure index (MPI) was calculated between simultaneously registered intra-aneurysm sac and systemic pressures. Values presented are medians with interquartile range between parentheses.

Results: Type I EL (n=1) showed MPI of 93% in the nidus and 62% in the thrombus. Type II ELs were associated with lower MPIs in the thrombus (35(24-38)%) when AAAs shrank (n=4), compared to when the aneurysms remained unchanged (n=11, MPI 78 (47-85)%) or expanded (n=6, MPI 74 (58-87)%, P=.019). The nidus of type II EL (MPI 79 (70-90)%) had higher pressure than the thrombus (45 (34-85)%, P=.047, n=7). Successful embolization of type II ELs led to AAA shrinkage (n=3, MPI reduction to 13-31%) or diameter stability (n=3, unchanged MPIs of 37-44%). Type III ELs (n=3) had MPIs in the thrombus of 33%-70%.

Conclusions: Endoleaks after EVAR pressurize the AAA-sac non-uniformly with higher, near-systemic, pressure in the EL nidus comparing with the thrombus. Nevertheless, type

II EL in shrinking AAAs have lower intra-sac pressure than expanding or stable aneurysms and successful EL embolization reduces pressure.

Introduction

Endovascular aneurysm repair (EVAR) of abdominal aortic aneurysms (AAA) is successful when the aneurysm-sac is completely excluded.[1] Type I and III endoleaks are considered treatment failures given their association with adverse clinical outcomes.[1] In contrast, the importance of type II endoleaks is controversial. On the one hand they tend to seal spontaneously and are frequently associated with aneurysm stability or shrinkage,[2, 3] but on the other hand, aneurysm expansion, and even sporadic rupture, has been reported with type II endoleaks.[4, 5]

Follow-up after EVAR has focused on the evaluation of endoleaks and AAA size. Imaging techniques allow assessment of flow within the aneurysm sac detecting contrast enhancement and/or Doppler signals. However, intra-sac pressure after EVAR is only indirectly evaluated by these imaging methods by assessing AAA size changes. The intra-aneurysm sac pressure is one of the main determinants of the tension applied on the AAA wall. A high or low tension will lead to aneurysm expansion or shrinkage, respectively. We have previously verified this relationship in the absence of endoleaks by performing direct intra-aneurysm sac pressure measurements (DISP).[6, 7] Pressure has also been measured in patients with endoleaks, but mostly in type II endoleaks and shortly after EVAR.[8, 9]

The aim of this study was to evaluate intra-aneurysm sac pressures in patients who developed endoleaks after EVAR of AAA).

Methods

Patients

553 patients underwent EVAR of AAA at our tertiary university center since 1993. Twenty-five of these patients (18 men, 7 women; 76 (68-80) years old) underwent 31 DISP 16 (14-26) months after EVAR (figure 1). The median duration of DISP was 174 (141-209) minutes. DISP was performed in 21 patients while a type II endoleak was present or had been embolized. Three patients had type III endoleaks at the time of DISP and one patient had a type I endoleak.

Inclusion criteria for the study were based on anatomic suitability for direct sac puncture and the presence of an endoleak after EVAR or the status after endoleak embolization. Patients were grouped according to the type of endoleak or status after its embolization (table I). Our initial experience with DISP in 8 of these patients has been included in a previous report.[7]

The study was approved by the local ethics committee and all patients gave informed consent before the procedure.

Imaging

The imaging protocols included contrast-enhanced spiral-computer tomography (CT) scans preoperatively and yearly after EVAR. More frequent CT-scans were performed when indicated. Preoperative CT scans included two spiral scans, i.e., before and after intravenous non-ionic iodinated contrast injection. Postoperative CTs had an additional delayed spiral scan. CT scans were reconstructed with 0.75-3mm axial slices. An additional CT-scan was obtained the month before DISP (pre-DISP CT-scan).

The shortest transverse diameter of the AAA was measured at its widest portion on axial CT-scans by the same observer. Diameter changes were calculated to express the diameter evolution before DISP (Diameter at the time of DISP – Initial diameter).[7] A 5 mm cutoff was used for grouping AAAs into shrinking (≤ -5 mm), unchanged and expanding (≥ 5 mm).[1]

At the time of DISP a digital subtraction aortogram was performed. This included selective angiography of the superior mesenteric and hypogastric arteries whenever the origin of the type II endoleak was not clearly established by CT-scan and non-selective aortography.

Endoleaks were classified according to the recommended reporting standards.[1] Endoleak nidus was defined as the contrast filled area within the aneurysm sac. A pressure measurement was considered within the endoleak nidus when this location was verified by contrast injection and free blood flow was obtained from the needle.

Pressure measurement system and DISP technique

Anatomical suitability was assessed in the pre-DISP CT-scan and defined as the possibility of inserting a pressure sensor in the AAA-sac without damaging any viscera or jeopardizing stent-graft integrity.

The pressure measurement system and DISP technique have been previously described in detail.[6, 7, 10] Wired tip-pressure sensors mounted on .014-inch guide-wires were used (PressureWire4, RADI Medical AB, Uppsala, Sweden). The pressure guide-wire used for intra-aneurysm sac pressure measurements had a shorter tip (1 mm instead of 3 cm) in order to allow precise placement of the sensor. Systemic pressure was measured in the

lumen of the stent-graft. Access to the AAA-sac was obtained by translumbar puncture using fluoroscopic guidance in 28 occasions (including the 6 late embolization controls). Two DISP were done through direct transabdominal ultrasound-guided AAA puncture. During the direct puncture of the AAA-sac efforts were made to pin-point the endoleak nidus (figure 2-A). Measurements in the thrombus were made when the sensor was located approximately mid-way between the nidus and the stent-graft. In one occasion, the pressure guide-wire was passed through a coaxial catheter placed in the ostium of the inferior mesenteric artery via the superior mesenteric artery.

The system recorded systemic and AAA-sac pressures simultaneously. Intra-aneurysm sac pressures were analyzed as systolic, diastolic, mean and pulse pressures. Mean pressure index (MPI) was calculated as the percentage of the mean intra-aneurysmal pressure relative to the simultaneous mean systemic pressure.

MPI = mean AAA pressure / mean systemic pressure * 100

Measurements were only considered of good quality if the drift in recalibration of the pressure sensor did not exceed 5 mm Hg in the end of the measurements. DISP has been previously validated in patients with a median intraobserver variability of MPI of 0 (-7 – 17)%.[10] The sensor was also tested in vitro with an accuracy better than 2 mm Hg.[10, 11]

Endoleak embolization

Embolization of the endoleak was performed once access to the endoleak nidus or its feeding vessels was achieved (figure 2-B). The embolizing materials used were coils,

radioopaque glue (Histoacryl, Braun, Tuttlingen, Germany and Lipiodol, Laboratoire Guerbet, Aulnay-Sous-Bois, France) and/or gel-foam sponge (Spongostan Standard, Johnsson & Johnsson Medical Limited, UK). Late embolization control was defined as DISP being performed on a different occasion than the embolization.

Statistical analysis

Normal distribution was not assumed. Values are presented as medians and interquartile range (IQR) between parentheses, if not stated otherwise. Non-parametric exact tests were used for paired and unpaired comparisons. Results were considered significant when $P < .05$. Statistical analysis was done using SPSS 12.0.1 (SPSS Inc, Chicago, USA).

Results

Type I endoleak

One patient (81 years old male, AAA diameter of 55 mm) with a proximal type I endoleak and unchanged AAA diameter (0 mm) underwent DISP 15 months after EVAR. MPI and pulse pressure were, respectively, 62% and 21 mm Hg in the thrombus and 93% and 66 mm Hg in the nidus. The AAA expanded 9 mm in the 28 months following DISP until the patient consented a reintervention

Type II endoleaks

Nineteen patients (12 men; table I) underwent 21 DISP while a type II endoleak was present. Two patients underwent DISP twice, since no embolization was performed on the first occasion and the aneurysms subsequently expanded (6 mm after 8 months and 11 mm after 11 months, respectively).

At the time of the pressure measurement 4 AAAs had decreased in size, 11 remained unchanged and 6 had expanded. MPI, intra-aneurysm mean and pulse pressures were significantly lower in the thrombus of shrinking AAAs when compared to AAAs that expanded or remained unchanged in size (P of .019, .030 and .019, respectively; table II).

Pressure in the thrombus and nidus of type II endoleaks

Pressure was measured within the thrombus and inside the endoleak nidus during the same puncture in 7 patients (figure 3). Pressure was significantly higher inside the endoleak nidus (MPI of 79 (70-90)% and pulse pressure of 22 (18-57) mm Hg) than

within the thrombus (MPI of 45 (34-85)% and pulse pressure of 11 (7-21) mm Hg; P = .043 and P=.028, respectively). The median MPI difference between the nidus and the thrombus was 32 (4-44)%.

Type II endoleak embolization

Thirteen patients underwent embolization of type II endoleaks at the time of DISP. Glue was used in 12 cases and coils and gel-foam particles in one.

Twelve of the 13 procedures were considered successful and the AAA diameter changed 0 (-7 – 2) mm during the following 13 (6-37) months. In 5 of these patients pressure was measured immediately after the embolization at the same procedure. MPI changed from 70 (57-84)% before the embolization to 53 (30-79)% immediately after (not significant). Intra-aneurysm sac mean and pulse pressure changed from 86 (56-98) and 13 (12-19) mm Hg before the embolization to, respectively, 56 (31-111) and 12 (8-40) mm Hg after the embolization (not significant in both). There was no association between the final MPI and the AAA diameter change afterwards.

One embolization was unsuccessful since the endoleak persisted at the end of the procedure. This patient was considered unfit for any further reintervention and the aneurysm continued to expand (19 mm during the following 26 months).

Late control of type II endoleak embolization

Six patients (5 men, 1 women; 74 (70-79) years old; table I) underwent DISP 16 (6-31) months after successful embolization of type II endoleaks.

Three patients with MPIs of 31% or less at the late embolization control showed AAA shrinkage. The MPIs were lower after the embolization (table III).

Three patients with unchanged AAA diameter after the embolization of the type II endoleak had MPIs between 37% and 44% at the late embolization control. The MPIs had not changed significantly at the late control comparing to before the embolization (table III).

Type III endoleaks

Three patients (3 men, table I) underwent DISP in the presence of a type III endoleak. One patient with a small type III endoleak persisting after EVAR underwent DISP 2 months after the EVAR. MPIs ranged from 70% close to the endoleak to 42% in the periphery of the AAA-sac. The endoleak sealed spontaneously afterwards and the AAA diameter shrank by 12 mm.

The other two patients underwent DISP when late type III endoleaks were identified (one partial separation of the components of an aortic uni-iliac system and one small fabric disruption) and the AAAs remained unchanged in diameter (+2 and +1 mm, respectively). Intra-sac pressure within the thrombus decreased with increasing distance to the endoleak (MPIs ranging from 57 to 35% and 44 to 33%, respectively). No measurements were performed in the endoleak nidus. One patient refused any further reinterventions and died within one month of unknown cause. The other one showed a 6 mm expansion of the AAA in the 4 months following DISP and was converted to open repair.

Discussion

The aim of EVAR is the exclusion of the aneurysm sac from blood flow and pressure. AAA size and endoleak status have been the key points in the evaluation of the treatment success. However, intra-aneurysm sac pressure has, until recently, been mostly indirectly assessed. The use of tip pressure sensors for direct intra-aneurysm sac pressure measurement after EVAR has been previously validated.[6, 7, 10] Shrinking AAAs without endoleaks were shown to have lower intra-aneurysm sac pressure within the thrombus compared to expanding aneurysms.[7] Furthermore, DISP was able to predict future AAA diameter changes in the absence of endoleaks. However, the effect of endoleaks after EVAR in intra-sac pressure is incompletely understood, especially considering the varied clinical outcome associated with the different types of endoleaks. The results of this study show that the endoleak nidus (channel) has consistently higher pressure than the intra-sac thrombus. Type II endoleaks seem to be associated with varying intra-sac pressures in the thrombus relating to the AAA diameter changes. Moreover, type II endoleaks appear to be dynamic since the variation can occur within the same patient at different time points. Successful embolization of a type II endoleak leads to depressurization of the AAA-sac with shrinkage or stabilization of the aneurysm diameter. Endoleaks of type I and III usually lead to a pressurization of the AAA-sac, as it was anticipated.

DISP in one patient with a type I endoleak showed a high pulsatile intra-aneurysm sac pressure in the thrombus and even higher and near-systemic in the nidus. This was followed by AAA expansion and reinforces the perception of these endoleaks as treatment failures.

Type II endoleaks were associated with varying degrees of intra-sac pressurization. The previously reported difference in intra-sac pressure between shrinking and expanding AAAs without endoleaks[7] was also verified in type II endoleaks. However, AAA shrinkage with a type II endoleak was associated with higher intra-sac pressures in the thrombus than what was previously seen in AAA shrinkage without endoleaks (MPI of 35% and 19%, respectively).[7]

Reports in the literature have consistently shown near-systemic pressure in patients with type II endoleaks in the early follow-up after EVAR.[8, 9] However, these measurements were performed mainly within the endoleak nidus and, as our results show, there is a pressure difference between the EL nidus and the AAA thrombus. The difference in pressure between the nidus and the thrombus seems to be dependent on several factors including the size of the endoleak nidus,[12, 13] its flow and perfusion pressure[14-16] and thrombus composition. In our experience some cases displayed a discrepancy in the endoleak size as seen on the CT-scan and on the aneurysmogram (with contrast injection into the nidus). The composition and structural properties of the thrombus, and their dependency on the proximity of blood circulation, may also influence the transmission of pressure.[17] Furthermore, the amplitude of the pressure gradient measured between the nidus and the thrombus may also be influenced by the distance between the measurement locations. This varying distribution of sac pressure in patients with endoleaks, although consistently higher in expanding aneurysms, may question the reliability of systems based on single-spot pressure measurements such as implantable pressure sensors.[18]

Recent literature report the use of implantable devices in the intra-operative identification

of endoleaks[19] and in canine models of type II endoleaks.[20] However, the location of the pressure transducer in relation to the endoleak nidus may influence the measurements obtained with those devices. For instance, if the pressure transducer is placed within the endoleak nidus, measurements can be high without necessarily implying AAA expansion. Type II endoleaks seem to be a dynamic entity since many seal spontaneously early after EVAR. This dynamic character of type II endoleaks, the difference in intra-sac pressure between patients with and without endoleaks and the above mentioned pressure gradient between the nidus and thrombus, suggest the need for a cautious interpretation of intra-sac pressure in patients with endoleaks.

Direct percutaneous puncture of the AAA allows not only intra-sac pressure measurement but also embolization of type II endoleaks. This is a significant advantage, as translumbar embolization has been shown to be more efficient[9, 21-23] than endoluminal methods.[24-27] Successful type II endoleak embolization was associated with shrinkage or stabilization of the aneurysm diameter. The diameter stability may be partially explained by the incompressibility of the embolic material injected into the sac. Intra-aneurysm sac pressure appears, nevertheless, to decrease upon successful embolization. In contrast to others[8] we did not find this pressure reduction directly after the embolization but only later during follow-up. It is possible that by performing our initial pressure measurement later in the follow-up after EVAR we have measured a more selected group of patients where the endoleaks that sealed spontaneously in the early follow-up hade been excluded.

In conclusion, the endoleak nidus had high, near-systemic pressure while the degree of thrombus pressurization varied. Thus, pressure is not uniformly distributed through the

AAA sac. Shrinking AAAs with type II endoleaks had lower pressure than those with expanding or unchanged AAA diameter. Successful endoleak embolization leads to a delayed depressurization of the AAA sac. Type I and III endoleaks are associated with high intra-sac pressures.

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References

1. Chaikof EL, Blankensteijn JD, Harris PL, White GH, Zarins CK, Bernhard VM, et al. Reporting standards for endovascular aortic aneurysm repair. J Vasc Surg 2002;35:1048-60.

2. Resch T, Ivancev K, Lindh M, Nyman U, Brunkwall J, Malina M, et al. Persistent collateral perfusion of abdominal aortic aneurysm after endovascular repair does not lead to progressive change in aneurysm diameter. J Vasc Surg 1998;28:242-9.

3. Rhee RY, Eskandari MK, Zajko AB, Makaroun MS. Long-term fate of the aneurysmal sac after endoluminal exclusion of abdominal aortic aneurysms. J Vasc Surg 2000;32:689-696.

4. White RA, Donayre C, Walot I, Stewart M. Abdominal aortic aneurysm rupture following endoluminal graft deployment: report of a predictable event. J Endovasc Ther 2000;7:257-62.

5. van Marrewijk CJ, Fransen G, Laheij RJ, Harris PL, Buth J. 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;27:128-37.

6. Sonesson B, Dias N, Malina M, Olofsson P, Griffin D, Lindblad B, Ivancev K. Intra-aneurysm pressure measurements in successfully excluded abdominal aortic aneurysm after endovascular repair. J Vasc Surg 2003;37:733-8.

7. Dias NV, Ivancev K, Malina M, Resch T, Lindblad B, Sonesson B. Intra-aneurysm sac pressure measurements after endovascular aneurysm repair: differences

between shrinking, unchanged, and expanding aneurysms with and without endoleaks. J Vasc Surg 2004;39:1229-35.

8. Baum RA, Carpenter JP, Cope C, Golden MA, Velazquez OC, Neschis DG, et al. Aneurysm sac pressure measurements after endovascular repair of abdominal aortic aneurysms. J Vasc Surg 2001;33:32-41.

9. Rial R, Serrano Fj F, Vega M, Rodriguez R, Martin A, Mendez J, et al. Treatment of type II endoleaks after endovascular repair of abdominal aortic aneurysms: translumbar puncture and injection of thrombin into the aneurysm sac. Eur J Vasc Endovasc Surg 2004;27:333-5.

10. Dias NV, Ivancev K, Malina M, Hinnen JW, Visser M, Lindblad B, Sonesson B. Direct intra-aneurysm sac pressure measurement using tip-pressure sensors: In vivo and in vitro evaluation. J Vasc Surg 2004;40:711-6.

11. Kälvesten E, Smith L, Tenerz L, Stemme G. The first surface micromachined pressure sensor for cardiovascular pressure measurements. Proceedings of the 11th Annual International Workshop on Micro-Electro-Mechanical Systems, Heidelberg, Germany 1998:574-579.

12. Timaran CH, Ohki T, Rhee SJ, Veith FJ, Gargiulo NJ, 3rd, Toriumi H, et al. Predicting aneurysm enlargement in patients with persistent type II endoleaks. J Vasc Surg 2004;39:1157-62.

13. Timaran CH, Ohki T, Veith FJ, Lipsitz EC, Gargiulo NJ, 3rd, Rhee SJ, et al. Influence of type II endoleak volume on aneurysm wall pressure and distribution in an experimental model. J Vasc Surg 2005;41:657-63.

14. Arko FR, Filis KA, Siedel SA, Johnson BL, Drake AR, Fogarty TJ, et al. Intrasac flow velocities predict sealing of type II endoleaks after endovascular abdominal aortic aneurysm repair. J Vasc Surg 2003;37:8-15.

15. Parent FN, Meier GH, Godziachvili V, LeSar CJ, Parker FM, Carter KA, et al. The incidence and natural history of type I and II endoleak: a 5-year follow-up assessment with color duplex ultrasound scan. J Vasc Surg 2002;35:474-81.

16. Li Z, Kleinstreuer C. Computational analysis of type II endoleaks in a stented abdominal aortic aneurysm model. J Biomech 2006;39:2573-82.

17. Vallabhaneni SR, Gilling-Smith GL, Brennan JA, Heyes RR, Hunt JA, How TV, et al. Can Intrasac Pressure Monitoring Reliably Predict Failure of Endovascular Aneurysm Repair? Journal of Endovascular Therapy 2003;10:524-530.

18. Ellozy SH, Carroccio A, Lookstein RA, Jacobs TS, Addis MD, Teodorescu VJ, et al. Abdominal aortic aneurysm sac shrinkage after endovascular aneurysm repair: correlation with chronic sac pressure measurement. J Vasc Surg 2006;43:2-7.

19. Ohki T, Ouriel K, Silveira PG, Katzen B, White R, Criado F, et al. Initial results of wireless pressure sensing for endovascular aneurysm repair: The APEX Trial--Acute Pressure Measurement to Confirm Aneurysm Sac EXclusion. J Vasc Surg 2007;45:236-42.

20. Chaer RA, Trocciola S, Derubertis B, Hynecek R, Xu Q, Lam R, et al. Evaluation of the accuracy of a wireless pressure sensor in a canine model of retrograde-collateral (type II) endoleak and correlation with histologic analysis. J Vasc Surg 2006;44:1306-13.

21. Baum RA, Carpenter JP, Golden MA, Velazquez OC, Clark TW, Stavropoulous SW, et al. Treatment of type 2 endoleaks after endovascular repair of abdominal aortic aneurysms: comparison of transarterial and translumbar techniques. J Vasc Surg 2002;35:23-9.

22. Martin ML, Dolmatch BL, Fry PD, Machan LS. Treatment of type II endoleaks with Onyx. J Vasc Interv Radiol 2001;12:629-32.

23. Schmid R, Gurke L, Aschwanden M, Stierli P, Jacob AL. CT-guided percutaneous embolization of a lumbar artery maintaining a type II endoleak. J Endovasc Ther 2002;9:198-202.

24. Kasirajan K, Matteson B, Marek JM, Langsfeld M. Technique and results of transfemoral superselective coil embolization of type II lumbar endoleak. J Vasc Surg 2003;38:61-6.

25. Haulon S, Tyazi A, Willoteaux S, Koussa M, Lions C, Beregi JP. Embolization of type II endoleaks after aortic stent-graft implantation: Technique and immediate results. J Vasc Surg 2001;34:600-5.

26. Karch LA, Henretta JP, Hodgson KJ, Mattos MA, Ramsey DE, McLafferty RB, et al. Algorithm for the diagnosis and treatment of endoleaks. Am J Surg 1999;178:225-31.

27. Chuter TA, Faruqi RM, Sawhney R, Reilly LM, Kerlan RB, Canto CJ, et al. Endoleak after endovascular repair of abdominal aortic aneurysm. J Vasc Surg 2001;34:98-105.

Figure Legends:

Figure 1 – Patients undergoing direct intra-aneurysm sac pressure measurements (DISP) according to the type of endoleak and AAA diameter change. Six patients underwent DISP after successful embolization of type II endoleaks. *Two patients with type II endoleaks underwent DISP twice. Initially after AAA diameter shrinkage and thereafter once the AAA had expanded.

Figure 2 – A – Digital subtraction angiography with contrast injection inside a type II endoleak nidus (arrow heads) through a translumbar puncture of the AAA at the time of direct intra-aneurysm sac pressure measurement. Two lumbar arteries are filled (arrows). B – Status after embolization of the endoleak with Hystoacril-Lipiodol. The glue fills the endoleak nidus and the proximal part of two lumbar arteries (arrow).

Figure 3 – Recording from direct intra-aneurysm pressure measurement. A – Measurement within the thrombus. B – Measurement in the endoleak nidus. Red curves represent the aortic pressure, and green curves represent the intra-aneurysm-sac pressure. Each figure has three tracings. The large one to the left is the tracing obtained during each recording where the aortic and intra-aneurysm sac pressures are registered simultaneously. The pressure recorded during 10 heart cycles is gathered separately in two different tracings, one for the aortic (upper smaller tracing to the right) and the other intra-aneurysm sac pressures (lower smaller tracing to the right). The system calculates also the mean of the systolic, diastolic and mean pressures for both the aortic and intra-

sac pressures (values bellow the small tracings). In the smaller tracings the scale is adjusted by the software to the amplitude of the curves."

Table I – Characteristics of patients

Group	No. of patients	Age	AAA Ø (mm)	Time between EVAR and DISP (mo)
Type I EL	1	81	55	15
Type II EL	19*	77 (68-81)	62 (53-73)	16 (14-22)
Embolization control after type II EL	6†	74 (70-79)	58 (53-62)	30 (20-48)
Type III EL	3	73 (64-75)	55 (52-62)	41 (2-97)

*Two patients of the 19 patients with type II endoleaks were measured twice. The first time after diameter shrinkage and thereafter once the diameter had expanded.

†Four of these patients were also measured before the embolization of the endoleak and are, therefore, also included in type II endoleak group (ie, 2 patients were measured only after a previous embolization of the endoleak).

Values are shown as median with interquertile range between parenthesis when not stated otherwise.

AAA Ø – AAA diameter; EL – endoleak, DISP – direct intra aneurysm-sac pressure measurement.

Table II – Intra-aneurysm sac pressure in the presence of type II endoleaks.

Diameter change before DISP	N	Follow-up before DISP (mo)	ΔØ AAA (mm)	AAA mean pressure (mm Hg)	AAA pulse pressure (mm Hg)	MPI (%)
Shrinking	4	19 (7-43)	-6 (-9, -5)	40 (24-47) *	7 (6-10) †	35 (25-38) ‡
Unchanged	11	15 (13-16)	2 (0, 3)	86 (53-97) *	21 (12-22) †	78 (47-85) ‡
Expanding	6	22 (20-31)	7 (6,10)	76 (59-109) *	17 (11-31) †	74 (58-87) ‡

Grouping was done according to the AAA diameter change. *P = .019, †P = .030, ‡P = .019. N – number of DISP performed. ΔØ – diameter change; MPI – Mean Pressure Index.

Table III – Pressure in six patients undergoing late endoleak embolization control

Patient	MPI before embolization (%)	MPI after embolization (%)	Intra-aneurysm sac pressure mm Hg			Follow-up after embolization (mo)	ΔØ AAA after embolization (mm)
			Systolic	Diastolic	Mean		
1	70	31	34	28	30	44	-7
2	NA	13	22	19	20	72	-18
3	67	19	25	18	21	50	-6
4	NA	42	53	40	45	39	-1
5	45	44	45	38	40	11	1
6	39	37	60	57	58	9	-1

The follow-up after the embolization includes even the imaging follow-up after the second DISP.

MPI – Mean Pressure Index; ΔØ – diameter change.

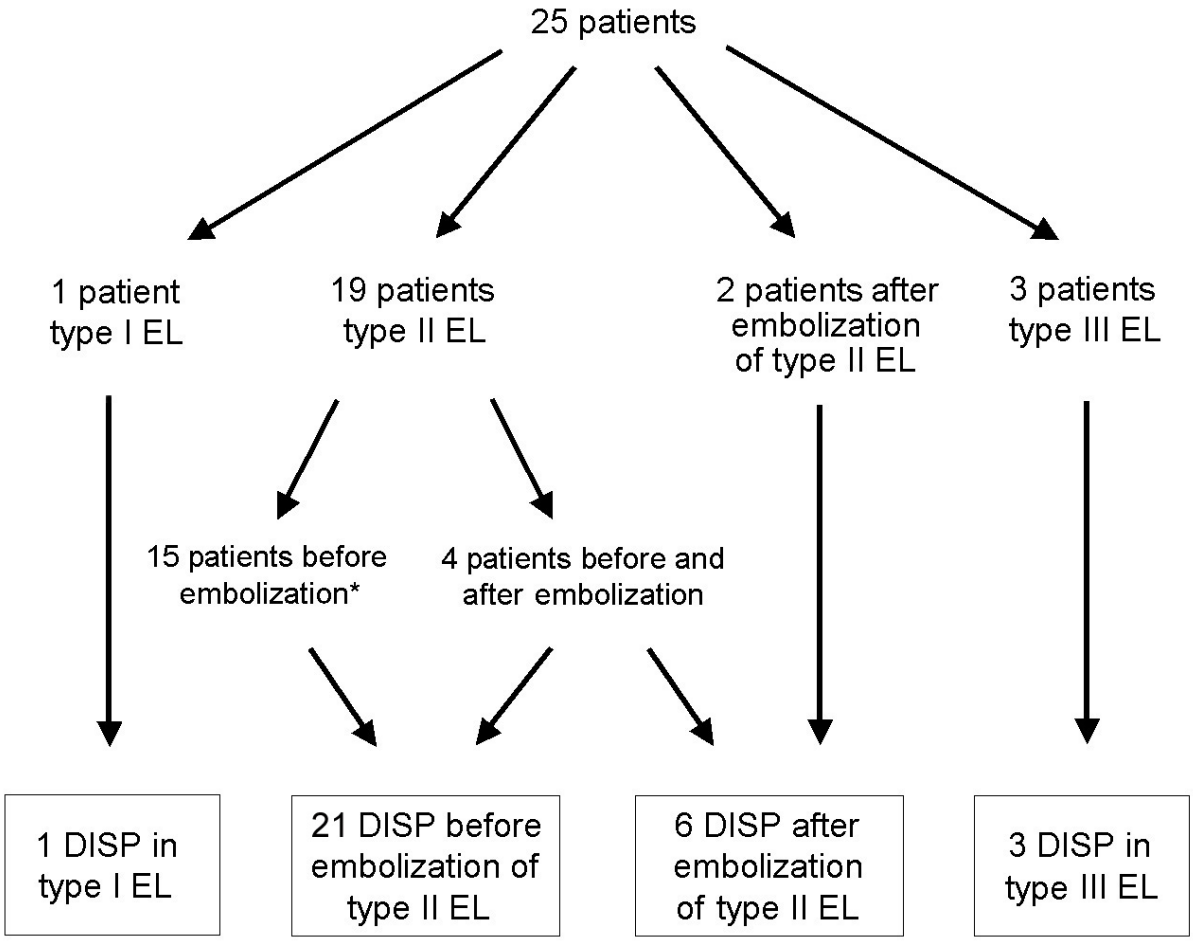

Figure 1

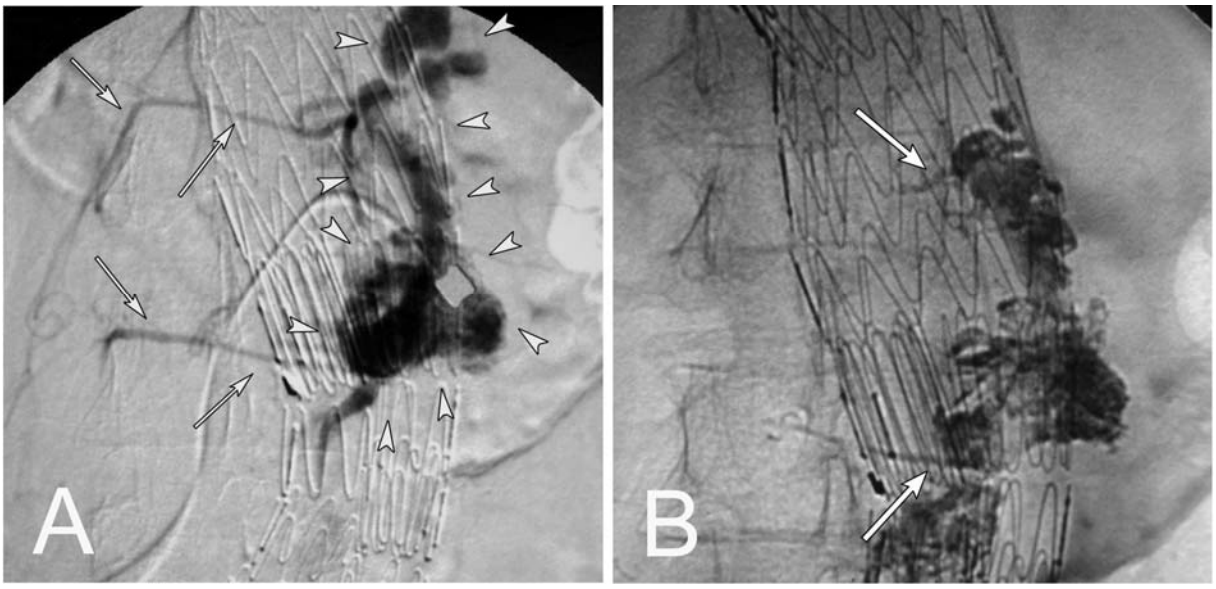

Figure 2

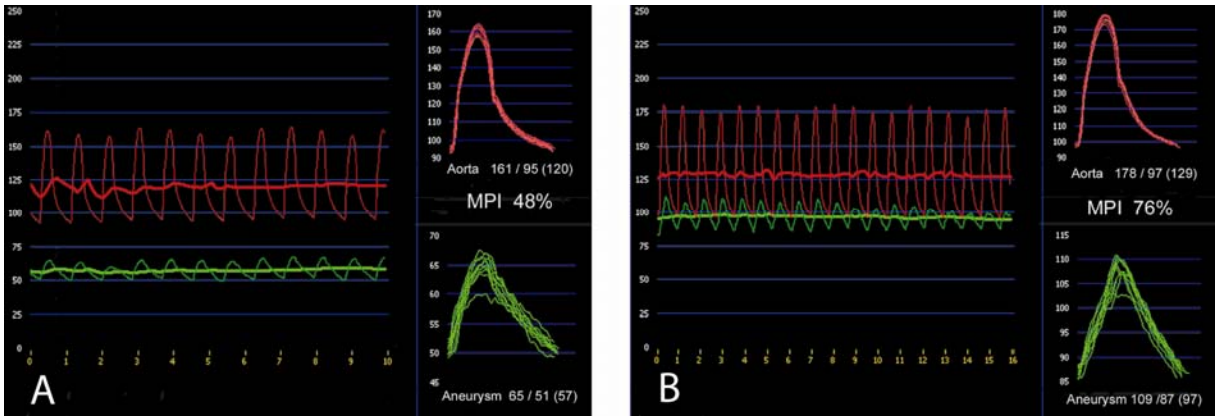
A
Aorta 161 / 95 (120)
MPI 48%
Aneurysm 65 / 51 (57)
B
Aorta 178 / 97 (129)
MPI 76%
Aneurysm 109 /87 (97)

Figure 3