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Long-term analysis of standard abdominal aortic endovascular repair using different grafts focusing on endoleak onset and its evolution

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A B S T R A C T

Objective: To report long-term results of standard EVAR focusing on endoleak onset (ELo) and its evolution.
Methods: 880 EVARs using different stentgrafts (1999–2015) were included. Primary outcomes were all-cause mortality and AAA-related mortality. Timing of ELo after EVAR was categorized as follows: ELo1 = 0–2 years, ELo2 = 2–4 years, ELo3 = 4–6 years, ELo4 = 6–8 years, and ELo5 ≥ 8 years. The rate of sac shrinkage/sac expansion and the need to re-intervene were the variables considered to determine EL evolution.
Results: Median follow-up was 60 months (IQR: 36–84). Summary follow-up index was 0.99. Survival rate was 94.5% at 2 years, 57.7% at 10 years, 33.3% at 14 years. Freedom from AAA-related-death rate was 99.3% at 14 years. Freedom from endoleak was 86.4% at 2 years, 68.3% at 10 years, and 48.6% at 14 years. EL rate was 1.9% (n = 19),16.6% (n = 146), 0.8% (n = 7), and 0.4% (n = 4) for type I, II, III and IV, respectively. Only type II EL showed a significant difference in the ELo (Elo1 = 31%; ELo2 = 12.8%; ELo3 = 9.4%; Elo4 = 10.2%; Elo5 = 11.4%; P < .001). Sac shrinkage occurred in 791 (90%) patients while 89 (10%) had a persistent sac expansion at the last follow-up. Freedom from reintervention was 95.6% at 2 years, 86.4% at 10 years, and 80% at 14 years. 48 out of 176 (27.2%) patients with EL underwent reintervention. The re-intervention rate was significantly higher within the first two years of follow-up if compared to the following years (17.6% vs. <10%; P < .001).
Conclusions: An active lifelong surveillance follow-up can guarantee good long-term EVAR outcomes. Reinterventions and type II EL were more frequent in the first two-year of follow-up.

1. Introduction

Endoleak (EL) is the Achille's heel of endovascular aortic aneurysm repair (EVAR). EL is the most common complication after EVAR, and it is frequently related to reintervention. At the time of repair, ELs may be present in up to 25% of patients. Some ELs may resolve without intervention, some require immediate or delayed treatment to prevent aneurysm rupture, and some develop months or years after EVAR [1–7].

Therefore, lifelong surveillance after EVAR is required and recommended [4]. The 2018 Guidelines by the Society for Vascular Surgery reported a significant incidence of postoperative ELs up to 5 years after EVAR, but a detailed evaluation of the EL onset and evolution on a long-term period is still lacking in literature.

This study analyzed long-term results of standard EVAR focusing on EL onset, its evolution, and related reintervention rates using different grafts.

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2. Methods

Consecutive patients operated on for asymptomatic abdominal aortic aneurysm (AAA) larger than 5.5 cm were included in the current study. All patients were treated from 1999 to 2015 at a single tertiary referral Hospital. Patients' characteristics, operative data, and follow-up details were prospectively collected in a specific database. All data were retrospectively analyzed. The local Ethics Committee approved the study and patients gave informed consent before the procedure and during follow-up before each procedure of follow-up (computed tomography angiography: CTA; or contrast enhanced ultrasound: CEUS). The inclusion in the computerized database required the patient consent.

2.1. Definitions

Outcome criteria and definitions were reported according to the Reporting standards for EVAR [8]. ELs were classified as early if they occurred before the 12 month follow-up or as late if they occurred after. Sac shrinkage was considered in any case of stable reduction of both antero-posterior and latero-lateral diameter of the sac of at least 2 mm in the two last consecutive examinations. In case of a transient shrinkage with afterwards growth, this condition has been considered in the group of patients with sac expansion. Sac expansion was considered in any case of growth of both anterior-posterior and latero-lateral diameter of the sac of at least 5 mm at the last follow-up vs. the first post-operative follow-up imaging. Sac evolution (shrinkage or expansion) was evaluated using either CTA or CEUS/color duplex ultrasound (CDU) imaging.

2.2. Endpoints

Primary outcomes were all-cause mortality and AAA-related mortality. Endoleak onset (ELo) after EVAR was categorized as following: **ELo1** when the endoleak was first diagnosed from the beginning of follow-up to year 2; **ELo2** when the endoleak was first diagnosed from year 2 to year 4 of follow-up, **ELo3** when the endoleak was first diagnosed from year 4 to year 6 of follow-up, **ELo4** when the endoleak was first diagnosed from year 6 to year 8 of follow-up, and **ELo5** when the endoleak was first diagnosed after year 8 of follow-up. Sac shrinkage/sac expansion and need to reintervene were the variables in evaluating EL evolution.

Independent predictors of mortality, endoleak onset, sac shrinkage, and reintervention were investigated.

2.3. Follow-up protocol

From 1999 to 2012 all consecutive EVAR patients were scheduled for post-operative day 1 CDU and plain abdominal radiography (RX), and a 1-month post-operative follow-up consisting of CTA, CDU and clinical examination. The same examinations were performed every 6 months thereafter. In cases with sac shrinkage, CTA was then performed annually. Starting from 2012 EVAR follow-up was modified after the publication of the guidelines of the European Federation of Societies for Ultrasound in Medicine and Biology [9]. Patients were scheduled for a 1-month post-operative follow-up consisting of CDU, RX and clinical examination. Thereafter, clinical examination and CDU were performed every 6 months. CEUS was also performed when: a) any endoleak was detected at CDU; b) significant sac growth >5 mm within 6 months; and routinely for: c) patients suffering from renal insufficiency stage ≥3 Chronic Kidney Disease – CKD; or d) iodine contrast allergy. CTA after 2012 was performed within 3-months of the procedure, but thereafter, a CTA is performed when there is a case of non-diagnostic imaging with CDU or CEUS, when a secondary intervention is planned or after 4-years. More details on the follow-up protocols used in this study have been previously published [10].

The Follow-Up Index (FUI) was used to describe follow-up completeness at a given study end date as ratio between the investigated and the potential follow-up period. Each patient had a FUI value therefore the entire study population had a summary FUI value (i.e.: mean value) [11].

2.4. Statistical analysis

Normal distribution and homogeneity of variance were confirmed by the Kolmogorov-Smirnov and Levene's tests, respectively. Data are expressed as mean and standard deviation (SD) in case of parametric distribution or median and interquartile range (IQR) in case of non-parametric distribution or as absolute frequency and percentage (%). The Chi-square or Fisher's exact tests were used for all comparisons of proportions. The continuous variables were compared using the Student's *t*-test. Survival analysis, freedom from reintervention and endoleak were performed according to Kaplan-Meier and significance was calculated with a log-rank test for pairwise comparisons. Following multivariate logistic regression, odds ratios (OR) were calculated for significant risk of significant variables related to mortality, endoleak onset, sac shrinkage, and reintervention. A P value <.05 (two tailed) was considered statistically significant. All statistical analyses were performed using the SPSS package, version 18.0 (SPSS Inc., Chicago IL, USA).

Table 2
Patients characteristics and intra-operative details. SD: standard deviation; COPD: chronic obstructive pulmonary disease; BMI: body mass index; RX: Fluoroscopy.

	OVERALL Patients n = 880	With Endoleak n = 176	Without Endoleak n = 704	P
Age, years				
Mean (SD)	75.6 (8.4)	75.2 (11.3)	75.6 (7.4)	.463
Range	46–92	50–89	46–92	
AAA Diameter (mm)				
Mean (SD)	60 (4)	59.6 (4)	60.2 (5)	1
Range	55–120	55–120	56–115	
Coronary Artery Disease	446 (51%)	94 (53.4%)	352 (50%)	1
Current smokers	330 (37%)	50 (28.4%)	280 (39.7%)	<.001
Ex-smokers	423 (48%)	98 (55.6%)	325 (46.1%)	.108
COPD	383 (43%)	89 (50.5%)	294 (41.7%)	.934
Renal insufficiency	326 (37%)	63 (35.8%)	263 (37.3%)	.998
Peripheral Arterial Disease	243 (28%)	44 (30%)	199 (28.3%)	.957
Hypertension	651 (74%)	140 (79.5%)	511 (72.3%)	1
Diabetes	106 (12%)	25 (14.2%)	76 (10.8%)	.224
Hypercholesterolemia	459 (52%)	88 (50%)	368 (52.3%)	<.001
BMI				
Mean (SD)	26.6 (3.6)	26.6 (3.5)	26.7 (3.6)	.995
Range	16–39.8	16.2–38	16–39.8	
BMI >30	154 (17.5%)	29 (16.4%)	125 (17.8%)	
RX time				
Mean (SD)	14.5 (6)	14.7 (7)	14.5 (6)	1
Range	5–131	7–120	5–131	
Contrast Agent				
Mean (SD)	116 (42)	116 (41)	116 (42)	.174
Range	10–400	10–250	15–400	
Technical Success	805 (91%)	152 (86.3%)	653 (92.7%)	1
Perioperative adjunctive maneuvers	73 (8%)	16 (9%)	57 (8%)	.865

3. Results

During the study period, 880 patients (mean age 75.6; SD 8.4 years; 824; 93.6% males) underwent EVAR, with a minimum of 1-year follow-up. Implanted stent grafts are reported in Table 1. Seven hundred eighty-nine (89.6%) procedures were performed under the instructions for use (IFU). The characteristics of the patients treated outside the IFU (n = 91;10.4%) were a short (<15 mm), conical or angulated proximal neck >60° (n = 28;31%), the absence of a suitable distal iliac neck (n = 32;35%) or narrowed iliac accesses (n = 31;34%). Population study data are shown on Table 2. Median follow-up was 60 months [IQR 36–84]. Summary follow-up index was 0.99.

3.1. Survival

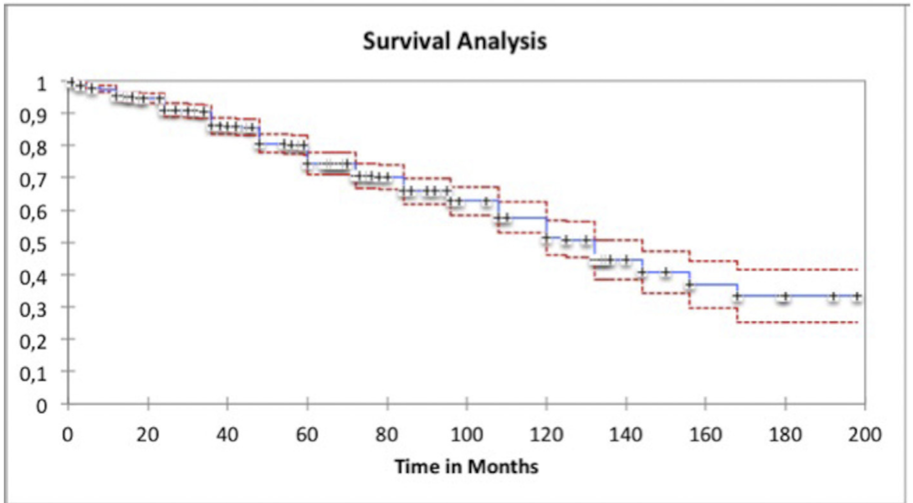
Overall survival was 94.5% at 2 years, 80.6% at 4 years, 74.5% at 6 years, 62.8% at 8 years, 57.7% at 10 years, and 33.3% at 14 years (Fig. 1). The 30-day overall mortality was 0.8% (n = 7). Six aneurysm-related deaths occurred (0.7%) during the entire study period, four of them were with old generation devices. In details we had: two perioperative deaths (Zenith first generation device, and Anaconda second generation device), two sac ruptures for type Ia EL (Endurant at 12 month and Anaconda first generation device at 36 month of follow-up), two deaths caused by reintervention (Zenith first generation device, at 12 months for a type Ia EL, and Excluder first generation device, at 34 months for sac expansion). Freedom from AAA-related death was 99.3% at 14 years of follow-up, 50% of the AAA-related deaths (n = 3) had a type Ia EL and in four cases (67%) sac expansion was present. Significant independent predictors for mortality were renal insufficiency (OR 2.1; 95% confidence interval (CI), 1.3–3.1; P < .001), and statin use (OR 0.47; 95%CI, 0.28–0.77; P = .003). Patients treated outside the IFU and patients treated according to the IFU had a similar freedom from all-cause and AAA-related-death rate. No difference in survival outcome was found for different grafts.

3.2. Endoleaks

One hundred seventy-six ELs were detected during the study period; there were 19 (2.1%) type I ELs, 146 (16.6%) type II ELs, 7 (0.8%) type III ELs, and 4 type (0.4%) IV ELs. Overall freedom from endoleak was 86.4% at 2 years, 81% at 4 years, 79.3% at 6 years, 73.2% at 8 years, 68.3% at 10 years, and 48.6% at 14 years (Fig. 2). The EL onset for type I, III and IV did not show any statistical difference during the five time periods (Fig. 3). Type II EL onset showed a significant difference during the five periods; specifically, it was 31% in the first two years but in the following periods the incidence was constantly 9–12%. Early EL counted for almost half of the EL in the entire follow-up [early EL n = 84 (47%) vs. late EL n = 92 (53%)]. The EL incidence did not depend on any specific device whether old or new. Patient's characteristics (age, gender, comorbidities, year of implant, type of drugs used), type of stentgraft used, and aneurysmal sac characteristics (sac diameter, proximal neck characteristics) were investigated but no significant independent predictor for EL onset was found.

3.3. Sac evolution

Overall sac shrinkage and stable sac occurred in 791(90%) patients while 89(10%) had a persistent sac expansion at their last follow-up. In the subgroup of patients with sac expansion an endoleak was always



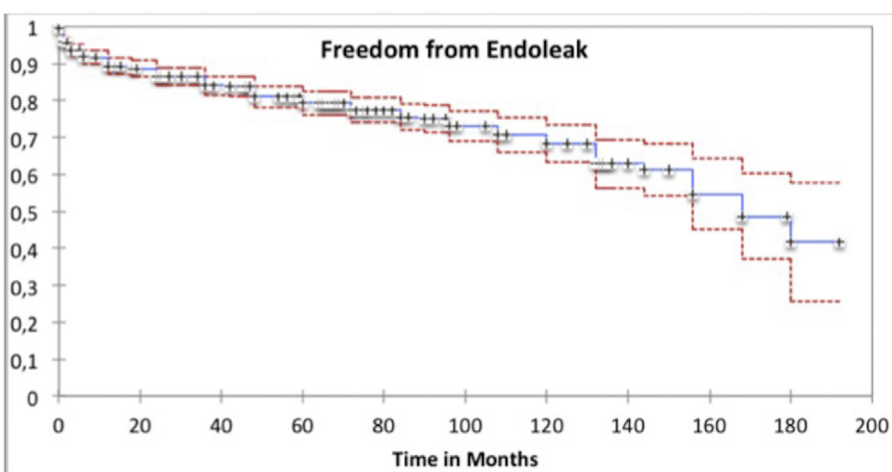
Months of follow-up	24	48	72	96	120	144	168
Patients at risk	724	499	314	193	119	48	20
SE	.016	.023	.043	.045	.051	.058	.066

Fig. 1. Kaplan-Meier curve of overall survival. SE: standard error.

present. Sac expansion during the five time periods did not show any difference in incidence (Fig. 4). Significant independent predictor for sac expansion was a procedure performed outside the IFU (OR 3.2; 95%CI, 1.9–5.6; P < .001). Moreover, any stent graft showed statistical difference in sac evolution even if considered the first generation devices such as the Exlcuder device before 2004 or the AneuRX device.

3.4. Reintervention

Eighty-one reinterventions were recorded during the study period. Causes for reintervention were: presence of an endoleak which was associated with a significant increase in the sac size (n = 48;59%), sac rupture (n = 2;2.5%), aorto-enteric fistula with active gastrointestinal



Months of follow-up	24	48	72	96	120	144	168
Patients at risk	641	415	281	158	90	38	18
SE	.005	.011	.021	.022	.03	.047	.054

Fig. 2. Kaplan-Meier curve of overall freedom from endoleaks. SE: standard error.

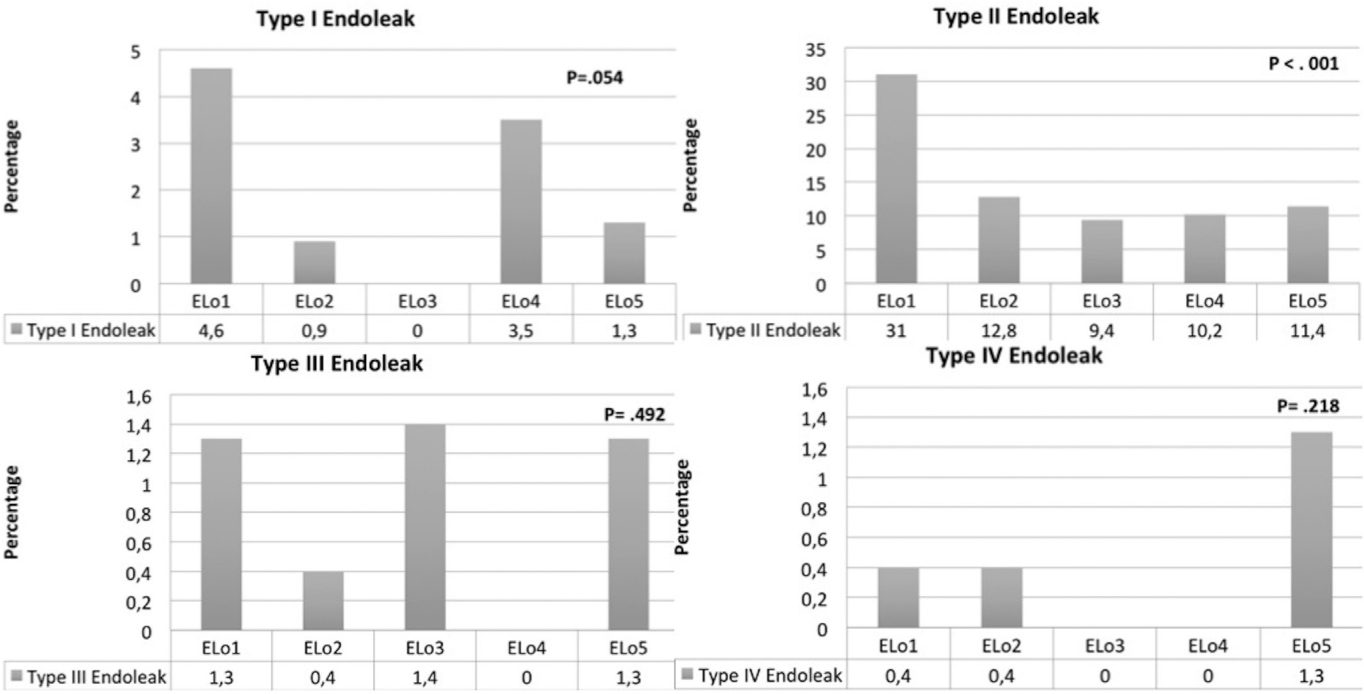


Fig. 3. Distribution of the type I,II, III, and IV endoleaks during the five time periods. Endoleak onset (ELo) after EVAR was categorized as following: **ELo1** when the endoleak was first diagnosed from the beginning of follow-up to year 2; **ELo2** when the endoleak was first diagnosed from year 2 to year 4 of follow-up, **ELo3** when the endoleak was first diagnosed from year 4 to year 6 of follow-up, **ELo4** when the endoleak was first diagnosed from year 6 to year 8 of follow-up, and **ELo5** when the endoleak was first diagnosed after year 8 of follow-up.

bleeding (n = 2;2.5%), limb occlusion causing critical limb ischemia or severe claudication (n = 26;32%), and proximal migration (n = 3;4%). Forty-eight (27.2% out of the total EL) patients with an EL underwent reintervention. Type I EL was treated in 10 (55.6%) cases, type II EL in 31(21.2%), type III in 4 (57.1%), and type IV in 2 (50%). Overall freedom from reintervention was 95.6% at 2 years, 94.1% at 4 years, 90.4% at 6 years, 87.1% at 8 years, 86.4% at 10 years, and 80% at 14 years (Fig. 5). The reintervention incidence was significantly higher within the first two years (17.6%; P < .001); then it declined to a stable value around 5% after 8 years (Fig. 6). Reintervention was performed by means of open surgery (n = 19;23%), endovascular therapy (n = 56;69%), or hybrid therapy (n = 6;8%). Details of the type of reintervention are shown in Table 3. Significant independent predictors for reintervention were: type I EL (OR 10.8; 95% CI, 1.6–73.2; P = .014), limb stenosis or/thrombosis (OR 65.8; 95% CI, 9.5–454; P < .001), age < 65 years (OR 2.9; 95% CI, 1.1–7.6; P = .03), active smoking (OR 2.1; 95% CI, 1.1–3.8; P = .02), and sac expansion (OR 4.9; 95% CI, 1.4–16.8; P = .01). Particularly, persistent sac expansion was the only indication for secondary interventions in case of type II EL. No difference in reintervention rate was seen in early and late EL or with the use of any device.

3.5. Outcomes for EVAR outside the IFU

Need for reintervention, endoleak, limb occlusion, and aneurysm sac enlargement rates for patients treated outside vs. patients treated according to the IFU were 7.7% vs. 9.1% (P =. 648), 23.1% vs. 20.7% (P = .591), 1.1% vs. 3.3% (P = .25), and 3.2 vs. 9.8% (P = .304), respectively (Table 4). The rate of planned perioperative adjunctive maneuvers was more frequent in patients treated outside the IFU (25.3% vs. 6.6%; P < .0001; OR 0.11; 95% CI: 0.032–0.375). Perioperative adjunctive maneuvers for patients treated outside vs. patients treated according to the IFU were: proximal aortic stenting or stent-grafting (8.5% vs. 2.2%;P < .0001), iliac angioplasty and stenting

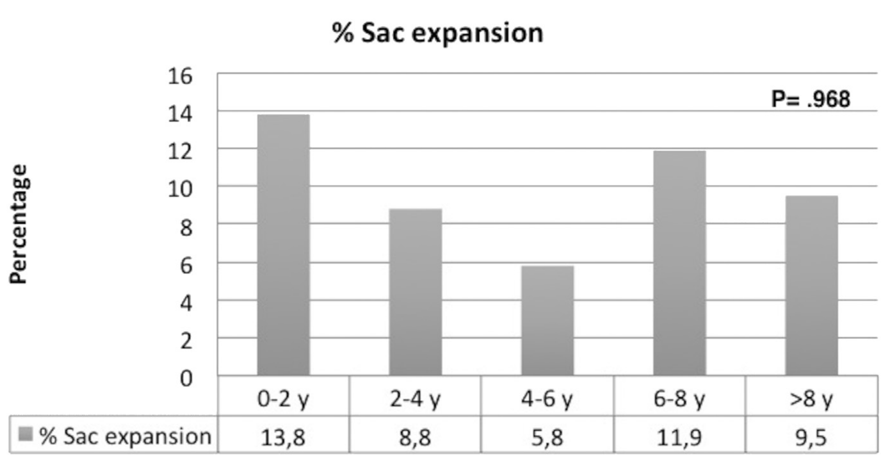
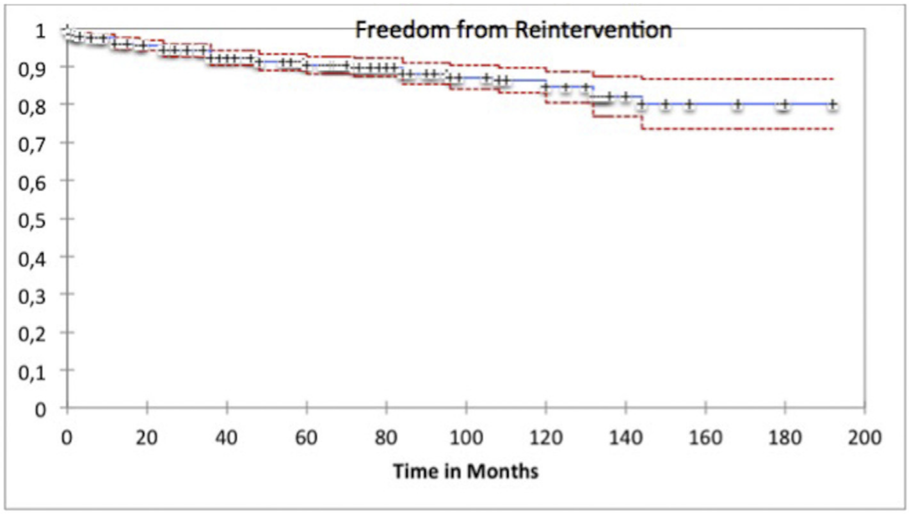


Fig. 4. Percentage of sac expansion during the five time periods. (y = years).



Months of follow-up	24	48	72	96	120	144	168
Patients at risk	698	492	362	156	90	43	19
SE	.003	.009	.017	.023	.032	.044	.055

Fig. 5. Kaplan-Meier curve of overall freedom from reintervention. SE: standard error.

or femoral endarterectomy or femoral crossover (30.8% vs. 7.5%;P < .0001), and hypogastric or inferior mesenteric artery embolization (6.6% vs. 12.2%; P = .116). No statistical differences were observed regarding the type of stentgraft used. When a procedure was performed outside the IFU overall early EL was more frequent than late EL (37.4% vs. 22.3%, P = .021). No differences were seen specifically in the type of EL subgroups. Endoleak onset during the five time periods showed no differences if EVAR was performed according to or outside the IFU.

4. Discussion

The most frequent complication of EVAR is EL occurrence. Literature has shown that EL can be manifested at any time [4]. This study confirmed the detailed onset of any kind of EL during a study period of fourteen years, demonstrating the importance of an active lifelong surveillance for all EVAR patients. Our patients had an optimal compliance to the follow-up and in fact the summary follow-up index of this study was almost perfect [11]. This is a very important point in favor of our study since patients are not always compliant with their surveillance programs. Non-compliance rate has been reported high as 42%–50%, which means that the annual imaging follow-up compliance after EVAR worldwide has been significantly below recommended levels and this can severely alter the results of EVAR papers [12,13]. Considering patients outside clinical trials these levels can be even lower [14]. We have depicted the incidence of EL in our patients: respectively type I EL occurred in 2.1%, type II EL in 16.6%, type III EL in 0.8%, and type IV EL 0.4%. Moreover, we showed how the incidence of type II EL was more frequent in the first two years, then it declined over time. No differences were seen for the other types. Our results were similar to those reported by Chang et al. [15] where the follow-up covered 10 years and endoleak rates were 3.5%, 27.2%, 0.9%, and 0.1% for type I, II, III, and IV EL respectively. The most frequent type of EL is the type II EL whose incidence in literature ranges from 6% to 30% [16–20]. In the study from Jones et al. [16] the perioperative incidence of type II EL was 18.9%, which is significantly lower than the 31% found in our

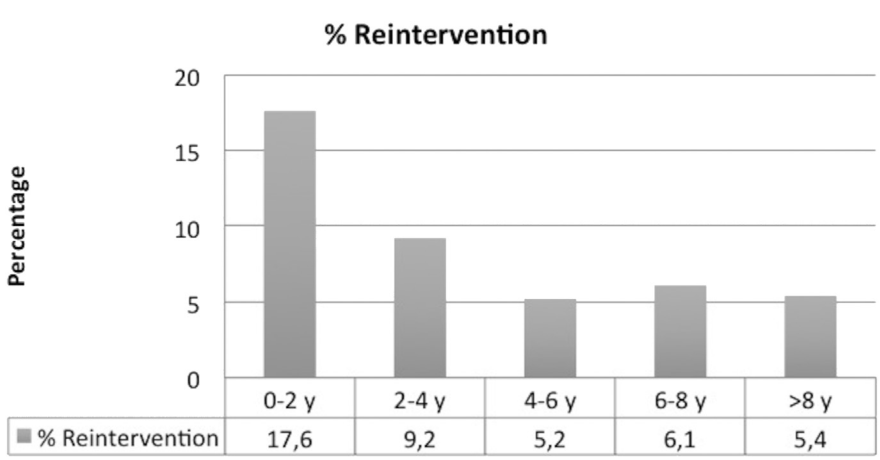


Fig. 6. Percentage of reintervention during the five time periods. (y = years).

Table 3
Type of reintervention. PTA: percutaneous angioplasty.

OVERALL n = 81 reinterventions	
N = 19 (23%) Open reinterventions	
(n = 13)	Open conversions
(n = 4)	Femoro-femoral crossover bypass
(n = 1)	Femoral thromboendarterectomy
(n = 1)	Video Laparascopic Inferior Mesenteric artery clipping
N = **6 (8%) Hybrid reinterventions**	
(n = 5)	Femoro-femoral crossover bypass and iliac PTA/stenting ± Fluoroscopy-assisted Fogarty thrombectomy
(n = 1)	Femoral thromboendarterectomy and iliac Fluoroscopy-assisted Fogarty thrombectomy
N = **56 (69%) Endovascular reinterventions**	
(n = 2)	Complete Relining
(n = 15)	Percutaneous Thrombolysis (Urokinase) ± iliac PTA and stenting
(n = 21)	Coil embolization of lumbar/hypogastric artery
(n = 5)	Iliac extension + hypogastric artery embolization
(n = 3)	Iliac extension
(n = 10)	Proximal cuff

study. In our center low weight molecular heparin is administrated for at least two weeks after the procedure as a predetermined post-operative protocol of anticoagulation. This additive anticoagulation regimen in our center can explain the higher incidence of type II EL reducing therefore the rate of early sac thrombosis. Spontaneous resolution of ELs occurs in 5 to 33% of patients with a type II EL [21,22]. Similarly, our incidence of type II EL after 2 years of follow-up dropped to a 10% rate. Many authors assume type II EL as a generic marker of negative prognosis [19,23]. In any case, no relevant modifiable predictors of type II EL have been found for example advanced age, intraluminal thrombus presence, and patency of aortic branches. According to some Authors [24], preventive coil/embolization of patent aortic branches before/during EVAR have been shown to be of little benefit regarding the occurrence of type II EL and aneurysm shrinkage, but other Authors [25] reported that sac embolization during EVAR, using a sac volume-dependent dose of fibrin glue and coils, could be a valid method to significantly reduce type II EL and its complications during early and midterm follow-up in patients considered at risk. According to Zhou et al. [26], almost 71% of all ELs were diagnosed after one year following EVAR and associated with aneurysm sac growth [13,27,28]. The Eurostar registry showed that around 65% of type I,II and III ELs were detected after the first post-operative assessment [28]. Since clinical significance of delayed EL is still unclear, and long-tern follow-up is often limited outside of clinical trials, Cieri et al. [23] reported that any EL can jeopardize the durability of EVAR, persistent EL is the worst case. Moreover, the absence of an EL during the early phase of follow-up is not a reliable predictor of a problem free future [26]. In our study, early or late ELs did not have any significantly different outcomes; the worst outcomes were always related to sac expansion caused by EL, whatever type they were. Aortic related death was extremely low (0.7%) and did not increase in the follow-up. In major randomized controlled trials there was an increase in the aneurysm-related mortality after 3 years of follow-up, which was five times higher than the open group [20]. This increase in mortality was due to late secondary interventions and late sac ruptures, which did not occur in our experience. We strongly think that these good outcomes depended on the active long-life surveillance that was established at the beginning of the EVAR experience. Moreover, in our center, open surgery is still offered in fit patients with 40% of open patients treated each year. The population study for EVAR was a very frail cohort of patients demonstrated by a poor freedom from death result at 5 (74.3%) and 10 years (57.7%) following EVAR. This data is inline with that of recent trials [20]. We can also postulate that the good outcomes of our study are supported by the fact that the non-adherence to IFU was very low (10%). Only one patient out of ten was treated outside the IFU in this paper, while literature reports a range from 38% to 68.9% [29–33]. Graft-related adverse events are associated to the non-adherence to the IFU: Schanzer et al. [29] reported a 41% rate of sac growth after EVAR in patients treated outside the IFU and the only predictive factor of sac shrinkage was a procedure under IFU (OR 3.2) in our study. Our study confirmed that renal insufficiency is a marker of no benefit from EVAR as reported in a recent meta-analysis [20] where the open repair in this subgroup of patients had better outcomes. Statin therapy was protective against mortality as reported previously [34]. We found that aneurysm-related mortality was associated to type I EL in one patient out of two making this kind of EL the most risky in terms of mortality. Sac can rupture with all types of ELs [16,35,36] type I and III being the most threatening [4]. Our deaths in the follow-up were all due to significant sac expansion related to endoleak presence. This data is in accordance to what was reported [12] in literature highlighting that sac expansion occurred before any sac rupture. Sidloff et al. reported approximately a risk of sac rupture <1% in case of a type II EL after EVAR [35]. In our study, any type II or IV ELs caused sac rupture. For type IV EL, Matsumura et al. [37] reported that microleakage could occur even after 2–3 years of follow-up with an intact graft. Reintervention occurred during all periods of follow-up with a mean reintervention rate of 14.5% at 10 years. Literature reports a 12% to 27% reintervention rate [38–40]. In our experience, EL evolved to reintervention in nearly one third of patients and in all cases, sac expansion was present. Moreover, EL presence was the primary cause of reintervention (almost 60%), limb occlusion being the second in term of incidence (32%). Literature reports type I and III ELs as absolute indication for reintervention [4]. In this study 44.4% type I ELs and 42.9% type III ELs were not operated on. There are not many type I or III ELs; we observed these patients who were very sick (all these patients developed metastatic cancers), therefore we decided not to intervene. As reported in the EVAR 1 trial [41] as well, in a long-term analysis there could have been some pressing clinical reasons not to re-intervene for some patients: old age, frailty and metastatic cancer. Recent data suggests that type Ia EL may not have such a bad prognosis as previously believed [42]. Several studies have demonstrated low rates of rupture and aneurysm-related mortality and high rates of spontaneous sealing of early type Ia EL in a range of 86–100% in selected patients [42–45] even if follow-up and sample size were limited. In the article from O'Donnell et al. [42] 43 type Ia ELs after EVAR were followed up (median 4 years), only 14% persisted after 1 year of follow-up, 7% aneurysm sac ruptured, and reintervention rate was only 30%. Reintervention has been stated to be reserved for patients who showed continued sac growth on serial imaging. Therefore early type Ia EL according to the Authors' conclusions can be safely observed in most patients. Venermo et al. [46] reported that early type Ia EL was associated with a lower rate of rupture than that of patients with an untreated AAA. Whereas early ELs are usually secondary to hostile neck anatomy or a poorly placed graft, those that appear during follow-up, the so called late ELs, can be the results of distal migration of the graft or aneurysmal degeneration of the proximal seal zone and have different clinical outcomes that tend to behave differently. Those ELs may expose the sac that has been depressurized and likely weakened over time to a new level of aortic pressure. Early type with high flow or delayed ELs are not benign either [42]. However, in this study we did not observe significant differences in any outcome for early vs. late ELs; maybe this is due to the relatively low numbers of events detected which can generate a type II error. Reintervention rate for type II ELs has been reported to be 19% in a recent meta-analysis [19]. This data is in concordance to the 21.2% of our study. Management of type II EL is still debated because clinical outcomes are variable [19,23–25,47,48] Persistent type II EL is the most threatening in term of long-term EVAR success [23]. A higher incidence of type III EL has been reported in case of old generation devices [49], where the underlying mechanism was disconnection of the stent graft components. The incidence of any adverse events after EVAR with third generation device demonstrated a clear reduction in reintervention [50,51]. In this study we did not observe this trend for old vs. new generation devices but the length of follow-up was not similar for these two cohorts of devices. Our study is limited by its retrospective nature, the relatively small number of adverse events and for some variables by the low incidence in the population study. Moreover, data from a single center may not be generalized due to the potential of site-specific selection criteria adopted locally.

5. Conclusions

Our 14-year study shows that an active lifelong surveillance follow-up can guarantee good long-term EVAR outcomes with a very low aneurysm-related death. ELs occurred during the entire follow-up. The only type of EL more frequent in the follow-up was the type II EL in the first two years of follow-up; the others had a constant and similar onset over time. EL evolved to reintervention in nearly one third of patients. Reinterventions were more frequent in the first two-year of follow-up.

Conflict of interest

The authors report no relationships that could be construed as a conflict of interest.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijcard.2018.11.009.

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