# Mid-term Outcome and Predictor of Transarterial Embolization for Type II Endoleak after Endovascular Abdominal Aortic Aneurysm Repair

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# Mid-term Outcomes and Predictors of Transarterial Embolization for Type II Endoleak after Endovascular Abdominal Aortic Aneurysm Repair

**Abstract**

**Purpose:**

To evaluate the mid-term outcomes of transarterial embolization (TAE) for type II endoleak after endovascular abdominal aortic aneurysm repair (EVAR) and investigate the predictors of sac enlargement after embolization.

**Materials and Methods:**

We conducted a retrospective analysis of 55 patients (48 men and 7 women, median age 79.0 (interquartile ranges 74-82) years) who underwent TAE for type II endoleak from 2010 to 2018. The aneurysmal sac enlargement, endoleaks, aneurysm-related adverse event rate, and reintervention rate were evaluated. Patients' characteristics and clinical factors were evaluated for their association with sac enlargement.

**Results:**

Fifty-five patients underwent TAE with technical success and were subsequently followed for a median of 636 (interquartile ranges 446-1292) days. The freedom from sac enlargement rates at 1, 3, and 5 years were 73.2%, 32.0%, and 26.7%, respectively. After initial TAE, the recurrent type II, delayed type I and occult type III endoleak were identified in 39 (71%), 5 (9%) and 3 (5%) patients, respectively. Although a patient had aorto-duodenal fistula, there was no aneurysm-related death. The freedom from reintervention rates were 84.6%, 35.7%, and 17.0%, respectively. In the multivariate analysis, sac diameter >55 mm at initial TAE (hazard ratios, 3.23; 95% confidence intervals, 1.22-8.58; $P < 0.05$) was a significant predictor of sac

enlargement.

**Conclusion:**

TAE for type II endoleak was not effective in preventing sac enlargement, and reinterventions were required among the mid-term follow-up. The sac diameter >55 mm at initial TAE was a significant predictor of sac enlargement.

**Abbreviations:**

| | |
|---|---|
| *TAE* | Transarterial embolization |
| *T2EL* | Type II endoleak |
| *T1EL* | Type I endoleak |
| *T3EL* | Type III endoleak |
| *ARAE* | Aneurysm-related adverse event |
| *IMA* | Inferior mesenteric artery |
| *LA* | lumbar artery |
| *NBCA* | N-butyl-2-cyanoacrylate |

**Introduction**

Endovascular abdominal aortic aneurysm repair (EVAR) has become an alternative to conventional open surgical repair for abdominal aortic aneurysm (AAA) due to its lower perioperative mortality and shorter hospital stay [1, 2]. Unfortunately, the early advantages of EVAR appear to be lost over time due to complications [3], which is probably largely attributable to endoleaks.

Type II endoleak (T2EL) is the most common endoleak occurring in 8%-44% of patients who underwent EVAR [4, 5]. T2EL occurs due to backflow of blood from aortic collaterals into the AAA sac after EVAR. In general, conservative management is selected for T2EL, as most of them are resolved spontaneously in the natural course [6-8]. However, persistent T2EL that remains >6 months after EVAR has been reported to be associated with aneurysm sac enlargement and aneurysm-related adverse event (ARAE) [9, 10]. In addition, patients with aneurysm sac enlargement caused by persistent T2EL after EVAR can develop delayed type I endoleak (T1EL) or type III endoleak (T3EL), which require additional interventions [11]. Therefore, the indication of intervention is defined commonly as persistent T2EL with sac enlargement according to a systematic review [12], although the latest ESVS (European Society for Vascular Surgery) guidelines stated that there is no evidence for when intervention is indicated for T2EL [13].

Embolization for T2EL is the first choice in terms of minimum invasiveness. Previous studies have reported various techniques of embolization for T2EL including transarterial embolization (TAE),

translumbar embolization, and transcaval and transealing embolization [8, 12, 14]. However, there is little evidence supporting the efficacy of embolization for T2EL [7, 8, 12, 15], and a systematic review and meta-analysis supposed that the clinical course after additional treatments including embolization may not be different from the conservative management of T2EL [12]. On the contrary, these studies have some limitations including lower technical success rates, the different definitions of technical and clinical success, short duration of follow-up, and several different techniques and materials of embolization. Therefore, current evidences might underestimate the efficacy of additional treatments. To optimize the management of persistent T2EL, mid to long-term assessments of single method and larger study groups are required.

In this study, we evaluated the mid-term outcomes of TAE in the same manner for persistent T2EL associated with sac enlargement after EVAR. Furthermore, we investigated the predictors of sac enlargement to optimize the criteria and techniques of T2EL embolization.

## Materials and Methods

### Patients

We conducted a review of 69 patients who underwent TAE for T2EL after EVAR from January 2010 to May 2018 at our two related institutions. This retrospective study has obtained approval from the Institutional Review Board in both institutions and the need for informed consent was waived. Eight patients with <6 months of follow-up duration and six patients without technical success were excluded.

Technical success was defined as no detectable endoleaks at the completion angiogram at TAE (details of technical success are available in the next section). No patient had coexisting other types of endoleak than T2EL. A total of 55 patients (48 men and 7 women, median age 79.0 (interquartile ranges (IQR) 74-82) years) who underwent TAE for T2EL after EVAR were enrolled. The indication of TAE for T2EL after EVAR was determined according to persistent T2EL with sac enlargement >5 mm in all cases. Patients' baseline preprocedural demographic and characteristics including comorbidities, smoking status, antiplatelet and anticoagulation history, the diameter and the shape of aneurysm, and details of EVAR were collected from clinical records and operative reports (Table 1).

**TAE procedure**

The treatment strategy is complete embolization of endoleak nidus and all feeding/drainage branches. Embolization of all branches without endoleak nidus or embolization of endoleak nidus without all branches were permitted when embolization of nidus and all branches was technically impossible. The patent aortic branches connecting to the endoleak nidus as feeding or drainage arteries of T2EL were identified on the preprocedural contrast-enhanced CT images with a 1.0-mm slice thickness and angiograms during the TAE procedure. The presence of coexisting other types of endoleak than T2EL was checked by the preprocedural contrast-enhanced CT images and intraoperative angiograms.

T2EL from the inferior mesenteric artery (IMA) were approached by accessing the middle colic

artery through the superior mesenteric artery and retrograde cannulating the IMA via the arc of Riolan or the marginal artery. T2EL from the lumbar artery (LA) were approached by accessing the iliolumbar arteries via the internal iliac arteries and retrograde cannulating the LA. A 1.6- or 1.9-Fr non-tapered microcatheter (Carnelian Marvel; Tokai Medical Products, Aichi, Japan or Carry; UTM, Aichi, Japan) was advanced to the endoleak nidus through a 2.7-Fr microcatheter, which was coaxially introduced through a 4-5-Fr catheter.

The endoleak nidus with branches or all branches without nidus were embolized using coils and NBCA glue. NBCA glue is the mixture of N-butyl-2-cyanoacrylate (Histoacryl; B.Braun, Melsungen, Germany) and iodized oil (Lipiodol; Guerbet, Aulnay-sous-Bois, France). Selection of embolization materials and range of NBCA/Lipiodol ratio (10%-50%) were determined by the attending interventional radiologist according to the target vessel anatomy. The endpoint of procedure (technical success) was no detectable endoleak nidus at the completion angiogram. Patients with residual nidus at the completion angiogram were excluded out of this study as a technical failure. The results and details of the TAE procedures were collected from operative reports (Table 2).

**Follow-up protocol**

After initial TAE, unenhanced computed tomography (CT) or magnetic resonance imaging (MRI) were basically performed at 1, 6, and 12 months and yearly thereafter, if no sac enlargement was identified. Contrast-enhanced CT was performed if sac enlargement, stent-graft migration, or sealing-zone

shortening were identified; however, contrast media administration was avoided in patients with renal dysfunction or allergy. The follow-up duration was defined as the time from initial TAE to the CT or MRI, death, or surgical explant.

**Imaging outcomes**

Preprocedural and follow-up CT and MRI evaluations of the maximum aneurysm sac diameter, the presence of endoleaks and type, stent-graft migration, and sealing-zone shortening were performed by two radiologists ([blinded for review] and [blinded for review] with 8- and 9-years' experience). The final diagnosis was achieved by consensus. Maximum aneurysm sac diameter was defined as the external diameter on the axial images. The aneurysm sac enlargement was defined as >5 mm increase in the maximum diameter compared to the sac diameter at the initial TAE. T2EL detected on follow-up CT after TAE was defined as recurrent T2EL. Other types of endoleak that were newly detected on follow-up CT after TAE was defined as delayed endoleak.

**Clinical outcomes**

The ARAE and reintervention were evaluated and documented from the clinical records. ARAE included aneurysm-related death, rupture, infection, and fistula. Reintervention after initial TAE included additional T2EL embolization, additional stent-grafting, open surgical ligation of aortic branches, and

explantation of the prosthesis. The occult endoleaks in patients who underwent open surgical explantation were evaluated using data from the operative reports. The occult endoleaks were defined as endoleaks not diagnosed on preoperative images and identified only intraoperatively.

**Predictors of sac enlargement after TAE for T2EL**

Patient characteristics and clinical factors were evaluated for their association with sac enlargement after TAE, and their candidates are listed in Table 3. They included preprocedural demographic, clinical characteristics, smoking status, antiplatelet and anticoagulation history, EVAR device, AAA morphological characteristics, aneurysm sac diameter at EVAR and initial TAE, interval and sac growth between EVAR and initial TAE, follow-up duration after TAE, number of patent aortic branches at initial TAE, embolization level, embolization materials, and endoleaks after TAE. The embolization level was categorized into the following three levels: sac packing with embolization of all patent branches, embolization of all branches without sac packing, or sac packing without embolization of all branches.

**Statistical analysis**

Categorical variables were summarized using frequencies and percentages, and continuous variables were described using median and interquartile ranges. Kaplan-Meier curve was used to estimate the freedom from sac enlargement and reintervention rate, and the log-rank test was used to compare.

Hazard ratios (HR) and 95% confidence intervals (CI) from Cox proportional hazards models were calculated to identify predictors of sac enlargement. A receiver operating characteristics (ROC) curve was used to assess the influence number of the continuous variables at $P < 0.05$ in the univariate analysis. The variables at $P < 0.05$ in the univariate analysis were introduced into the multivariate analysis. Statistical analyses were performed using software (JMP 14, SAS Institute, Cary, NC, USA). $P < 0.05$ was considered statistically significant.

## Results

### TAE procedure

In all patients, there was no detectable endoleak nidus at the completion angiogram after TAE. Complete obliteration of endoleak nidus with all branches was achieved in 30 patients (55%). Endoleak nidus without all branches were embolized in 9 patients (16%), and all branches without endoleak nidus were embolized in 16 patients (29%). The results and details of the TAE procedures are listed in Table 2.

### Imaging outcomes

The median sac diameter changes between pre-EVAR and initial TAE were 6.0 (IQR 2-9) mm, and the median sac diameter at initial TAE was 55 (IQR 49-59) mm. After initial TAE, recurrent T2EL was identified in 39 patients (71%). The coexistence of delayed T1EL and stent-graft migration or sealing-zone

shortening was identified in 5 (9%) and 3 (5%) patients, respectively. Twenty-eight patients (51%) resulted in sac enlargement >5 mm after TAE. Of these, the recurrent T2EL and delayed T1EL were identified in 25 (89%) and 3 (11%) patients, respectively. The freedom from sac enlargement rates at 1, 3, and 5 years were 73.2%, 32.0%, and 26.7%, respectively (Fig. 1a).

**Clinical outcomes**

The median follow-up duration was 636 (IQR 446-1292) days after initial TAE. There was no aneurysm-related death or rupture. One patient had ARAE and underwent open surgical repair due to aorto-duodenal fistula. All nine deaths during the follow-up duration were caused by non-aortic disease such as cancer or pneumonia..

Among 29 patients (53%) who required reintervention after initial TAE, 18 patients underwent single intervention, whereas 11 patients underwent multiple interventions, including additional TAE (n=19; 35%), additional stent-grafting (n=7; 13%), open surgical LA ligation (n=1; 2%), and explantation of the prosthesis (n=11; 20%). The indication of reintervention was sac enlargement (n= 27), delayed T1EL without sac enlargement (n= 2). Among 12 patients with open surgery, the occult T3EL was identified intraoperatively in three patients. One patient with occult T3EL had the coexisting T2EL, which was identified on the preoperative contrast-enhanced CT images. The freedom from reintervention rates at 1, 3, and 5 years were 84.6%, 35.7%, and 17.0%, respectively (Fig. 1b).

**Predictors of sac enlargement after TAE for T2EL**

We analyzed 26 factors associated with sac enlargement >5 mm after initial TAE. Univariate analysis identified COPD, sac diameter at initial TAE, and sac growth from pre-EVAR to initial TAE were associated with sac enlargement (Table 3). The other factors were not associated; however, the recurrent T2EL has a statistical tendency (P <0.1). The ROC curve identified that the optimal cutoff values of sac diameter at initial TAE, and sac growth between pre-EVAR and initial TAE were 55 mm and 6 mm, respectively. Multivariate analysis of three variables with P values <0.05 in the univariate analysis identified sac diameter >55 mm at initial TAE (HR, 3.23; 95% CI, 1.22-8.58; P= 0.02) was a significant predictor of sac enlargement (Table 4). Among 28 patients with large sac diameter >55 mm, 21 patients (75%) resulted in sac enlargement and T2EL after initial TAE were identified in 22 patients (79%). Conversely, seven (26%) of 27 patients with small sac diameter <55 mm had sac enlargement, and the recurrent T2EL were identified in 17 patients (63%). The freedom from sac enlargement rates at 3 years in patients with small sac diameter <55 mm or large sac diameter >55 mm were 58.0% and 8.2%, respectively (Fig.2). There was a significant difference between the two groups (P <0.0001).

**Discussion**

TAE for T2EL does not seem to be effective in preventing sac enlargement during the mid-term

follow-up in this study. Similarly, a systematic review and meta-analysis showed that the technical success rate of T2EL treatment including TAE is high, ranging from 84% to 100%; however, sac enlargement after treatment occurred in 31.6% of patients [12]. Sarac et al. reported that the freedom from sac enlargement >5 mm at 5 years was as low as 44% after embolization [15]. However, it is premature to decide that embolization for T2EL is meaningless because the definition of technical and clinical success, technical methods, or treatment indication are variable depending on reports.

In this study, the recurrent T2EL after TAE had a statistical tendency for sac enlargement in the univariate analysis. Although the aneurysm rupture by isolated T2EL rarely occurs, continuous sac enlargement with T2EL may cause the delayed T1EL or T3EL, stent-graft migration, and sealing-zone shortening [11]. Therefore, the management of T2EL should be important to prevent sac enlargement after EVAR. There are a few possible reasons that the recurrent T2EL was not statistically significantly associated with sac enlargement in this study. A previous study reported that 21% of patients with continuous sac enlargement had not only T2EL but also occult T1EL or T3EL [16], which is similar to our results. This indicates that some AAAs with T2EL might increase not due to T2EL but due to coexisting occult endoleaks. Additionally, recurrent T2EL may not always cause sac enlargement.

TAE is one of the common treatments of T2EL. The technical success of TAE for T2EL was reportedly as high as 77.2%-89.8% [12]. However, the definition of clinical success varied, and no reports revealed the prevalence or recurrence of T2EL after TAE in the follow-up imaging. In this study, although

there were no detectable endoleaks at the completion angiogram of the TAE procedure, T2EL was identified in 71% of the patients on the follow-up contrast-enhanced CT images. The recurrent T2EL was caused by recanalization from non-embolized, thrombosed branches or vasa vasorum may occur [17]. Contrarily, the angiogram during procedures were limited to detect T2EL due to poor spatial resolution with two-dimensional imaging, and T2EL might not be embolized completely and remain. Contrast-enhanced CT or other examinations should be performed to evaluate the prevalence of endoleak immediately after TAE to precisely evaluate the technical and clinical success of TAE for T2EL.

In this study, sac diameter >55 mm at initial TAE was associated with sac enlargement after embolization in the multivariate analysis. Additionally, a large sac diameter is reportedly a significant and independent risk factor for rupture and sac enlargement of AAA and after EVAR [9, 13]. TAE should be performed before the sac diameter exceeds 55 mm based on our results. However, in the ESVS guidelines, the elective treatment indication of AAA was >55 mm in diameter, and T2EL embolization has been a sac enlargement >10 mm regardless of preoperative sac diameter [13]. Therefore, most aneurysms with sac enlargement >10 mm after EVAR should exceed 55 mm in diameter. We suggest that TAE for T2EL should be performed before sac enlargement >10 mm which was recommended in the current guideline. Furthermore, especially in patients with large sac diameter, the prevention of T2EL occurrence including intraoperative embolization of branches or sac of AAA and endovascular aneurysm sealing should be more important [18-20] and other reliable treatments, such as surgical explantation, instead of TAE may be

considered if persistent T2EL occurs.

Since this is a retrospective and small study, there are several limitations. Firstly, the number of patients in the subgroups is too small to allow meaningful statistical analyses. Some factors reported in previous studies, such as completely embolizing the endoleak nidus and branch vessels, and current smoking [14, 21] were not associated with sac enlargement after TAE in this study. In contrast, sac diameter was not noted previously. A larger and prospective study needs to be conducted for further evaluation. Secondly, the incidence of recurrent T2EL may be not accurate. Contrast-enhanced CT scans after TAE were not acquired in patients with renal dysfunction, iodine allergy or without sac enlargement. Furthermore, artifacts from embolization materials including coil and NBCA glue may hide endoleak on CT images. However, it was not associated with outcomes except prevalence of endoleaks at the current stage because endoleaks without sac enlargement were observed conservatively. Thirdly, the size of AAAs treated with EVAR in this study was smaller than that of ESVS guideline recommendation. In the Japanese guidelines, EVAR for patients with small AAA (45-55 mm) who had risk factors of rupture including saccular aneurysms, women, rapid aneurysm growth, and symptomatic cases is recommended relatively [22]. Therefore, the sac diameter tended to be smaller than 55 mm. However, natural growth rate of untreated AAA increases with aneurysm diameter [13, 22], and it may affect the poor result of T2EL for AAA with >55 mm sac diameter. Further studies for large AAA may be needed.

**Conclusion**

TAE for T2EL was not effective in preventing sac enlargement, and reinterventions were required among the mid-term follow-up. The sac diameter >55 mm at initial TAE was a significant predictor of sac enlargement. TAE should be performed before the sac diameter exceeds 55 mm.

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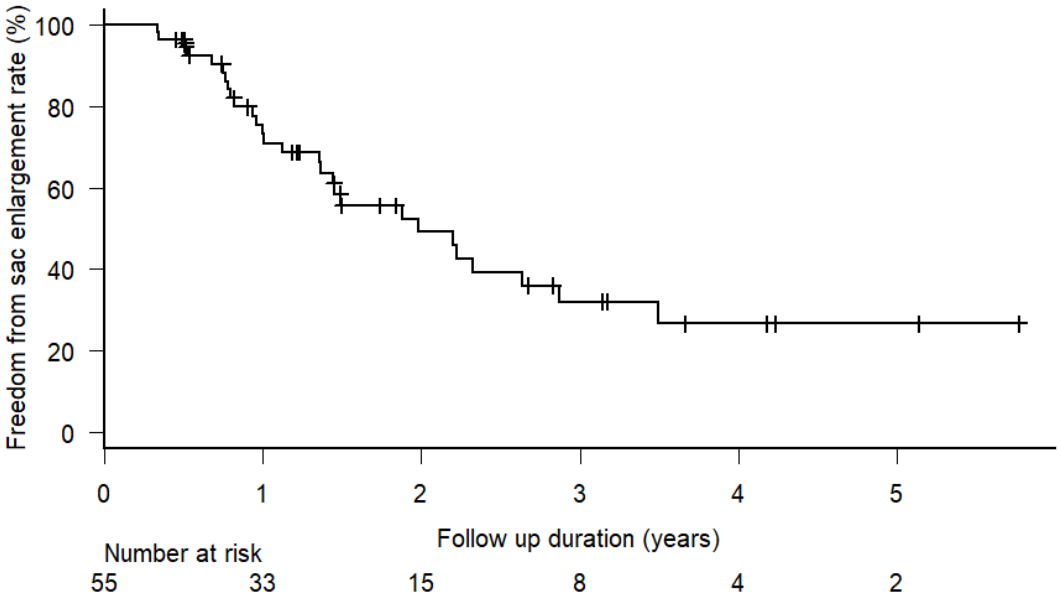
**Figure legends**

**Fig. 1 Freedom from sac enlargement and reintervention rates in all patients**

Kaplan–Meier curve of freedom from sac enlargement (a) and reintervention (b) in 55 patients.



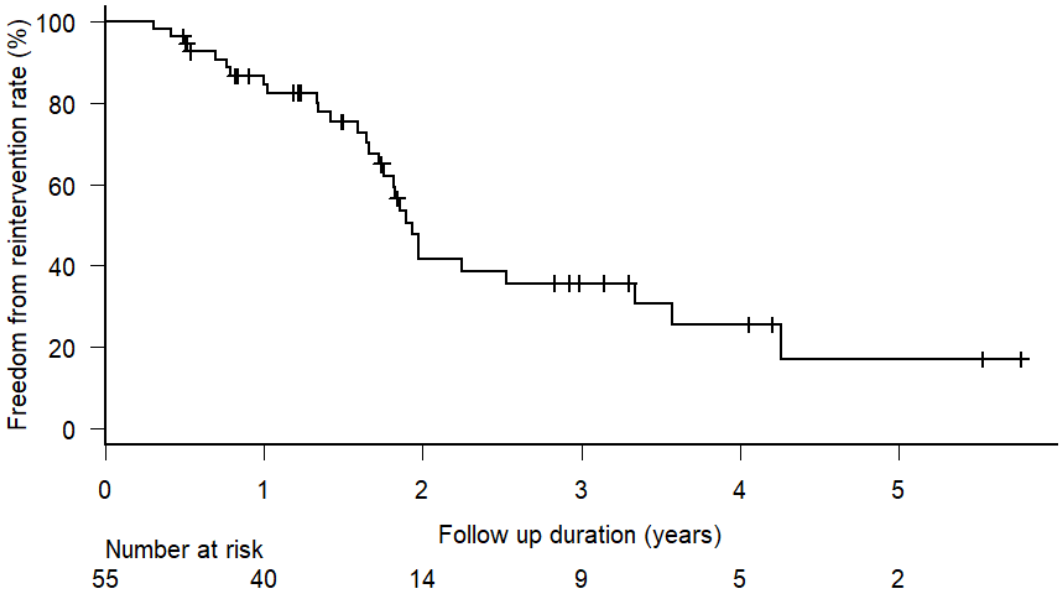

Freedom from reintervention rate (%)
100
80
60
40
20
0
0
1
2
3
4
5
Follow up duration (years)
Number at risk
55
40
14
9
5
2

**Fig .2 Freedom from sac enlargement rate according to sac diameter**

Kaplan–Meier curve of freedom from sac enlargement according to sac diameter at the initial transarterial embolization.

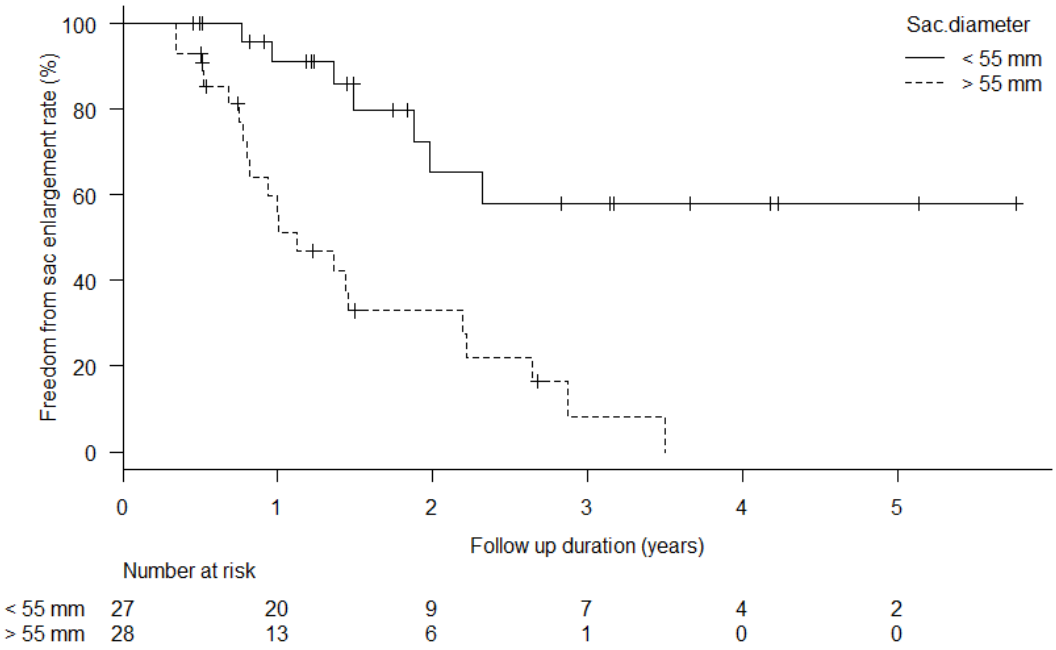


**Table 1. Patient demographics and comorbidities**

| Variables | No. % |
|---|---|
| Sex | |
| Male | 48 (87%) |
| Female | 7 (13%) |
| Coronary artery disease | 15 (27%) |
| Diabetes mellitus | 12 (22%) |
| Chronic kidney disease | 26 (47%) |
| Hypertension | 41 (75%) |
| Dyslipidemia | 24 (44%) |
| Peripheral artery disease | 4 (7%) |
| Smoking | |
| Current | 12 (22%) |
| Former | 25 (45%) |
| Never | 18 (33%) |
| Anticoagulation | 11 (20%) |
| Antiplatelet | 22 (40%) |
| Sac diameter at EVAR | 49 (45-53) mm |
| Aneurysm shape | |
| Fusiform | 47 (85%) |
| Saccular | 8 (15%) |
| EVAR | |
| Within Instructions for use | 48 (87%) |
| Outside Instructions for use | 7 (13%) |
| EVAR device | |
| Excluder | 27 (49%) |
| Zenith | 14 (25%) |
| Endurant | 12 (22%) |
| Powerlink | 2 (4%) |

Data are presented as counts (percentages) for the categoric variables or median (interquartile ranges) for

the continuous variables.

**Table 2. Results of TAE procedure**

| **Variables** | |
|---|---|
| Interval between EVAR and 1st TAE | 990 (657-1440) days |
| Sac diameter at 1st TAE | 55 (49-59) mm |
| Number of patent aortic branches at 1st TAE | |
| 1 | 15 (27%) |
| 2 | 19 (35%) |
| 3 | 12 (22%) |
| 4 | 6 (11%) |
| 5 | 3 (5%) |
| Embolization level | |
| Nidus with all branches | 30 (55%) |
| All branches without nidus | 16 (29%) |
| Nidus without all branches | 9 (16%) |
| Embolization materials | |
| Coil alone | 9 (16%) |
| NBCA glue with or without Coil | 46 (84%) |

Data are presented as counts (percentages) for the categoric variables or median (interquartile ranges) for the continuous variables.

**Table 3. Univariable comparison of factors associated with sac enlargement**

| Variable | No. (%) | HR (95%CI) | P |
|---|---|---|---|
| Age (year) | | 1.01 (0.96-1.07) | 0.70 |
| Sex | | | |
| Male | 48 (87%) | 1.00 | |
| Female | 7 (13%) | 2.19 (0.82-5.83) | 0.12 |
| Coronary artery disease | | | |
| No | 40 (73%) | 1.00 | |
| Yes | 15 (27%) | 1.41 (0.59-3.38) | 0.44 |
| Diabetes mellitus | | | |
| No | 43 (78%) | 1.00 | |
| Yes | 12 (22%) | 1.01 (0.41-2.50) | 0.99 |
| Chronic kidney disease | | | |
| No | 29 (53%) | 1.00 | |
| Yes | 26 (47%) | 1.20 (0.57-2.52) | 0.63 |
| Chronic obstructive pulmonary disease | | | |
| Yes | 10 (18%) | 1.00 | |
| No | 45 (82%) | 7.92 (1.07-58.44) | 0.04 |
| Hypertension | | | |
| No | 14 (25%) | 1.00 | |
| Yes | 41 (75%) | 1.39 (0.61-3.17) | 0.44 |
| Dyslipidemia | | | |
| No | 31 (56%) | 1.00 | |
| Yes | 24 (44%) | 1.24 (0.59-2.61) | 0.57 |
| Peripheral vascular disease | | | |
| No | 51 (93%) | 1.00 | |
| Yes | 4 (7%) | 1.50 (0.4-4.93) | 0.52 |
| Smoking | | | |
| Never | 18 (33%) | 1.00 | |
| Former | 25 (45%) | 0.78 (0.34-1.78) | 0.55 |
| Current | 12 (22%) | 0.63 (0.21-1.84) | 0.39 |
| Anticoagulation | | | |
| No | 44 (80%) | 1.00 | |
| Yes | 11 (20%) | 0.49 (0.15-1.64) | 0.25 |
| Antiplatelet | | | |
| No | 33 (60%) | 1.00 | |

| | | | |
|---|---|---|---|
| Yes | 22 (40%) | 2.03 (0.95-4.36) | 0.07 |
| Aneurysm shape | | | |
| Fusiform | 47 (85%) | 1.00 | |
| Saccular | 8 (15%) | 0.18 (0.02-1.31) | 0.08 |
| EVAR device | | | |
| Excluder | 27 (49%) | 1.00 | |
| Zenith | 14 (25%) | 1.41 (0.63-3.17) | 0.40 |
| Endurant | 12 (22%) | 0.68 (0.19-2.41) | 0.56 |
| Powerlink | 2 (4%) | 5.40 (0.00-1.76) | 0.13 |
| AAA | | | |
| Within Instructions for use | 48 (87%) | 1.00 | |
| Outside Instructions for use | 7 (13%) | 2.00 (0.76-5.28) | 0.16 |
| Sac diameter at EVAR (mm) | | 1.02 (0.97-1.07) | 0.37 |
| Sac diameter at 1st TAE (mm) | | 1.05 (1.003-1.09) | 0.03 |
| Sac growth between pre-EVAR and 1st TAE (mm) | | 1.09 (1.01-1.17) | 0.03 |
| Interval between EVAR and 1st TAE (day) | | 1.00 (0.99-1.00) | 0.29 |
| Follow up duration after 1st TAE | | 0.99 (0.99-1.00) | 0.09 |
| Number of patent aortic branches at 1st TAE | | 0.92 (0.63-1.31) | 0.66 |
| Embolization level | | | |
| Nidus with all branches | 30 (55%) | 1.00 | |
| All branches without nidus | 16 (29%) | 0.85 (0.36-2.02) | 0.85 |
| Nidus without all branches | 9 (16%) | 0.65 (0.24-1.81) | 0.41 |
| Embolization materials | | | |
| Coil alone | 9 (16%) | 1.00 | |
| NBCA glue with or without Coil | 46 (84%) | 1.71 (0.59-4.98) | 0.33 |
| Reccurent type II endoleak | | | |
| No | 16 (29%) | 1.00 | |
| Yes | 39 (71%) | 2.90 (0.87-9.63) | 0.08 |
| Type I endoleak | | | |
| No | 50 (91%) | 1.00 | |
| Yes | 5 (9%) | 0.66 (0.20-2.19) | 0.50 |
| Type III endoleak | | | |
| No | 52 (95%) | 1.00 | |
| Yes | 3 (5%) | 2.26 (0.67-7.49) | 0.19 |

Data are presented as counts (proportion) for the categorical variables.

Hazard ratios (HR) and 95% confidence intervals (CI) were calculated from cox proportional hazards

models. $P < 0.05$ was considered statically significant.

**Table 4. Multivariable comparison of factors associated with sac enlargement**

| Variable | No. (%) | HR (95%CI) | P |
|---|---|---|---|
| Sac diameter at 1st TAE | | | |
| <55 mm | 27 (49%) | 1.00 | |
| >55 mm | 28 (51%) | 3.23 (1.22-8.58) | 0.02 |
| Chronic obstructive pulmonary disease | | | |
| Yes | 10 (18%) | 1.00 | |
| No | 45 (82%) | 6.47 (0.87-48.12) | 0.07 |
| Sac growth between pre-EVAR and 1st TAE | | | |
| <6 mm | 27 (49%) | 1.00 | |
| >6 mm | 28 (51%) | 1.75 (0.68-4.47) | 0.24 |

Data are presented as counts (proportion) for the categorical variables.

Hazard ratios (HR) and 95% confidence intervals (CI) were calculated from cox proportional hazards models. $P < 0.05$ was considered statically significant.