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Type II Endoleak after Endovascular Aneurysm Repair: natural history and treatment outcomes

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Abstract

Objective: The natural history of type II endoleaks and linkage to aneurysm rupture is unclear. Likewise, treatment recommendations are controversial. The aim of this study was to examine the incidence, factors associated with type II endoleaks and outcomes in an Australia cohort of patients who have undergone endovascular AAA repair (EVAR).

Methods: Data from 693 patients who underwent EVAR between 2009 and 2013 at multiple institutions across Australia were studied. Patients who developed a) type II endoleak and b) type II endoleak with sac expansion, were compared for preoperative demographics, mortality, sac expansion, aneurysm rupture and intervention rates.

Results: A total of 225 patients developed type II endoleak over a mean follow-up of 1.9 years (±1.0 years), out of which 133 spontaneously resolved, 37 were untreated unresolved and 16 underwent intervention. Type I and III endoleaks occurred in 50 and 19 patients respectively. Smoking (p=0.002) and Warfarin (p=0.044) were protective factors for development of type II endoleak whilst age (p=0.034), right iliac artery tortuosity (p=0.031), and right (p=0.008) and left external iliac diameters (p=0.028) were risk factors for endoleak. Three patients suffered aneurysm ruptures in the entire cohort. All ruptures occurred in type II endoleak patients, of which two occurred after reintervention and in the absence of sac expansion (>5mm). Late type II endoleak occurred in 117 patients, out of which 26 had sac expansion. Of those without late type II endoleak, 25 has sac expansion. There was no statistically significant difference in survival between those with and without type II endoleak. Age (p<0.0001) and smoking (p=0.001) were significant independent predictive factors for survival in this patient sample. Treatment outcomes were encouraging with most cases involving endoleak resolution (15 of

16 patients) and no sac expansion post intervention (0 of 8 patients with complete follow-up info on sac size).

Conclusion: Aneurysm rupture in patients with type II endoleak is uncommon in our series. Type II endoleak with sac expansion does not appear to be associated with aneurysm rupture. In this series, most aneurysm ruptures occurred in the absence of documented sac expansion and after reintervention.

Key words: endoleak, type II, endovascular, aneurysm rupture

1. **Introduction**

Type II endoleak (T2EL) is the commonest complication of endovascular aneurysm repair (EVAR) and due to an insufficient understanding of its role in aneurysm sac rupture, optimum treatment recommendations are presently uncertain.

The risk of sac expansion in patients with type II endoleak is unclear[1]. Sac expansion of 5mm has been reported to be associated with aneurysm rupture[2], but according to a more recent review by Sidloff et al, in over one third of patients with type II endoleak who suffer aneurysm ruptures, this occurs in the absence of documented sac expansion[1]. This suggests that there may be other unidentified factors involved in the mechanism of aneurysm rupture in type II endoleak and highlights the concern of using sac expansion as a surrogate marker for risk of aneurysm rupture in management of type II endoleak.

Treatment outcomes for type II endoleak are also suboptimal, with recurrence of type II endoleak found in a third of patients following transarterial embolizations and around one fifth following translumbar embolizations. A large proportion of patients are found to have persistent sac expansion despite either treatment of type II endoleak.[1].

The current guidelines of the European Society of Vascular Surgery recommend a conservative approach to managing type II endoleak which involves reintervention with increased sac diameter ≥10 mm, with conversion to open surgery if endovascular treatment fails (level 2b)[3]. There are studies however, that recommend more aggressive approaches to treatment, such as intervening at ≥5mm[4], or in cases of persistent or recurrent type II endoleak regardless of sac expansion[5, 6] .

It is therefore important to clarify the natural history of type II endoleak and treatment outcomes, which will guide the establishment of more appropriate treatment guidelines.

The key objectives of this present study are to evaluate the incidence, outcomes, predictive factors and treatment success of type II endoleak in patients who have undergone EVAR for infrarenal AAA in

Australia with particular reference to rates of sac expansion, death, interventions, aneurysm rupture and treatment success. This will assist with the development of better treatment recommendations.

2. Materials and Methods

Data were collected prospectively between 2009 and 2013 in this multicenter study from 67 surgeons at 29 institutions throughout Australia. Institutions included both public tertiary centers and private hospitals. A total of 693 patients who underwent EVAR for infrarenal AAAs were entered onto a prospective database which was analysed retrospectively. Collected data included perioperative demographics and follow-up information such as aneurysm size, detection of type II endoleak, deaths, aneurysm ruptures, and treatments performed. The follow-up period was 3 years, with follow-up intervals of 1, 6, 12, 24 and 36 months. Ultrasound (USS) and Computed Tomographic Angiography (CTA) imaging were used during follow-up. Ethical clearance was obtained from the Queen Elizabeth Hospital Ethics of Human Research Committee, the Australian Institute of Health and Welfare and from all institutions contributing data to the EVAR trial.

Incidence of type II endoleak was defined by the presence of a type II endoleak at any point in the study, and included type II endoleaks combined with other endoleaks, e.g. type I and III endoleaks. Patients with combined type II endoleaks (two types of endoleaks) were included in the analysis of type II endoleak outcomes. The detection of type II endoleak is based on CTA and USS evidence, and the clinical judgment of the surgeon. Type II endoleak was further classified into early (demonstrated within 30 days) and late type II endoleak (type II endoleak occurring after 30 days). Sac expansion was defined as ≥5mm increase in aneurysm diameter. Anything less was regarded as stable sac size. All Australian death data were obtained from the Australian Institute of Health and Welfare National Death Index. Mortality was recorded in terms of overall, 30-day and twelve-month mortality rates. Survival analysis included variables with <10% incomplete data and involved two approaches. One approach was to assess the effects of Type II endoleak on survival reported as an unadjusted model along with a model adjusted for confounding factors. The other approach was to develop a model that contained significant, independent predictors of survival.

Preoperative Computed Tomographic Angiography (CTA) scans were prospectively reviewed for several anatomic variables which included aneurysm diameter, infrarenal neck length and diameter, infrarenal neck shape, aortic and aneurysm angles, diameters of iliac arteries, prevalence of aortic neck thrombus (as judged by the treating surgeon on the preoperative CTA),, presence of saccular aneurysm, occlusive aorto-iliac disease, arteries involved in the aneurysm sac, patency of Inferior Mesenteric Artery (IMA) and iliac tortuosity. Iliac tortuosity was defined as the extent of tortuosity of the common iliac arteries as assessed by the clinician using the following categories: none (i.e. straight), mild, moderate or severe. This scoring is subjective.

2.1 Statistical analysis

Statistical analyses were performed using SAS Version 9.3 (SAS Institute Inc., Cary, NC, USA) -. The data were summarized as means with standard deviations or percentages as appropriate. Group comparisons were made using Pearson's Chi-squared test or Fisher's exact test for categorical variables and Wilcoxon (Mann Whitney) test for continuous variables. The association between any Type II Endoleak and survival was summarized as median survival and assessed using a Cox Proportional Hazards model. Independent predictors of survival were identified using regression modelling. All tests were two-tailed and significance was assessed at $p<0.05$.

3. Results

3.1 Incidence of type II endoleak and preoperative variables

Out of 693 patients in Australia who underwent EVAR, 225 patients developed a type II endoleak. Median follow-up was 1356 days (95% CI: 1303-1404 days) for the type II endoleak group and 1466 days (95% CI: 1288-1576 days) for the no type II endoleak group (p=0.760). Combined type II endoleaks occurred in 9 patients, with 6 patients having combined type I and II endoleaks and the other 3 having combined type II and III endoleaks. Type I and type III endoleaks occurred in 50 and 19 patients respectively. The majority of the grafts were Zenith (Cook) grafts (67%) and the aorto-bi-iliac-bifurcated

configuration was used in over 90% of cases. The prevalence of type II endoleaks was higher in patients treated with the Excluder (Gore) device (54.2%) when compared to Zenith (Cook) (32.2%) and Endurant (Medtronic) (26.0%) devices (P=0.001). While the Excluder device was used in only 8% of patients, it accounted for 14% of type II endoleaks.

During the procedure, 124 type II endoleaks were detected. At first postoperative imaging and on discharge, 71 cases of type II endoleak were detected. At 36 months, 26 type II endoleaks were detected. The number of patients with type II endoleak detected both perioperatively and at some point during follow-up reduced from 23 at discharge to 7 at 36 months.

In terms of preoperative variables, smoking (p=0.002) and Warfarin therapy (p=0.044) were shown to be associated with a significantly lower risk of type II endoleak. Age (p=0.034), right iliac tortuosity (p=0.031), right (p=0.008) and left external iliac diameter (p=0.028) were shown to significantly increase the risk of type II endoleak (Table I).

3.2 Aneurysm ruptures, mortality and survival

A total of three patients in the entire cohort suffered aneurysm rupture in the present study. All of them had type II endoleak reported during the study, of which one had a type I endoleak documented perioperatively. In this patient, the type I endoleak resolved after re-stenting the right limb within the original graft perioperatively, but a type II endoleak was subsequently detected at 1-month follow-up, then at 6 and 12 months until reintervention with Onyx glue at 24 months. Despite post-intervention endoleak resolution and absence of documented sac expansion during follow-up, rupture occurred at 36 months resulting in death. The two other patients with ruptures had isolated type II endoleaks. Of these patients, one had a type II endoleak documented at 1 and 6 months before undergoing reintervention at 12 months with coiling. Despite post-intervention endoleak resolution and absence of documented sac expansion during follow-up, rupture also occurred at 36 months. The other patient with isolated type II endoleak, did not undergo reintervention. In this patient, type II endoleak was detected perioperatively, but not at 1, 6 and 12 months. At 24 months, type II endoleak was detected

again with rupture occurring at 36 months. There was a documented sac expansion of 14mm in the 2 years leading up to the rupture. In summary, of the three patients with ruptures, all had type II endoleak, and two had ruptures in the absence of sac expansion (>5mm), and two had ruptures despite successful endoleak resolution post-reintervention.

A total of 104 (mean follow-up in this cohort was 1.9 years) deaths were recorded over the course of the present study. Of these, 28 deaths were amongst type II endoleak patients and 76 were amongst patients without type II endoleak. Median survival was 1356 days amongst patients who had had a Type II endoleak. This compares to 1466 days amongst those who had not had a Type II endoleak. While the hazards of dying were somewhat lower amongst patients who had had a Type II endoleak, the difference between the two patient groups was not significant (HR = 0.80; 95% CI: 0.51-1.24; p=0.313) (Figure 1).

There was no statistically significant difference in survival between those with and without type II endoleaks (Table II and III). However, older age and active smoking were found to be significant, independent predictors of decreased survival in this patient sample (Table IV).

3.3 Sac expansion

Sac expansion was reported in 35 patients with a type II endoleak. An overall sac expansion (> 5 mm) was recorded in 24 patients, of whom, 5 died and 4 underwent an intervention. Of the 5 deaths in patients with sac expansion, 4 deaths were non-aneurysm related and 1 was of unknown cause. 17 deaths were reported amongst type II endoleak patients without sac expansion, while 20 patients required intervention.

None of the perioperative variables were found to be significantly associated with T2EL and sac expansion (Table V).

Late type II endoleaks were reported in 117 patients (excluding the perioperative type II endoleaks). Of the 117 patients with late type II endoleak, 26 had sac expansion and 91 did not have sac expansion.

Of the 26 patients with sac expansion, 5 died and 10 underwent intervention. Of the 91 patients without sac expansion, 19 died and 15 underwent intervention. Sac expansion was reported in 25 patients who did not have a late type II endoleak; of these, 8 died and 3 underwent intervention. 62 deaths were reported in patients without sac expansion and a further 33 of these patients underwent intervention (Table VI).

3.4 Outcomes of treatment of type II endoleaks

Out of 225 patients with type II endoleak, 186 patients had adequate follow-up information regarding treatment or resolution outcomes. Of these, 133 had spontaneous resolution, 37 had an unresolved type II endoleak but were being observed untreated, 28 underwent a diagnostic or other procedure to fix a complication but 16 underwent reintervention specifically for the type II endoleak, and thus form the treatment group.

Of the 16 patients treated for type II endoleak, at the time of the procedure, endoleak resolution occurred in 15 patients. For the one patient without endoleak resolution, intervention was indicated for a persistent type II endoleak with sac expansion of 17mm. The intervention involved embolization at 3 years which was close to the end of the follow-up period. This gave little time for observing endoleak resolution and assessing post-intervention reduction in sac size.

Indications for intervention in the present study included persistent type 2 endoleak (endoleak for longer than 6 months) in 8 patients, persistent type II endoleak with sac expansion in 5 patients, and sac expansion alone in 3 patients. All sac expansions in these patients were >5mm, except for one patient with 2mm sac expansion (5-5.2cm) with abdominal pain. Three patients had sac expansion exceeding 10mm prior to intervention. For the patients with sac expansion alone as their indication for treatment, type II endoleak was present at the time of documented sac expansion. No resolution of endoleak occurred prior to the onset of documented sac expansion or prior to intervention.

Following endoleak treatment, death occurred in 3 patients, with 2 being non-aneurysm related. The one patient with aneurysm-related death, died of a ruptured aneurysm at 3 years, described under 'Aneurysm ruptures, mortality and survival.' Only 8 patients with treated type II endoleaks had sufficient follow-up information regarding sac size, out of which none had sac expansion (6 had sac shrinkage and 2 had stable sac sizes). These 8 patients had undergone a range of interventions, including 1 with laparoscopic ligation of inferior mesenteric artery, 3 with coiling, 2 with embolization with embolic agent unspecified, 1 with open repair, and 1 with a bifurcated graft placed into the old tube graft. Indications for intervention in these 8 patients included 5 for persistent type II endoleak, 1 for persistent type II endoleak with sac expansion >5mm, and 2 for sac expansion alone, one being >5mm and the other >10mm.

4 Discussion

Our study demonstrates that not all aneurysm ruptures occur in the setting of sac expansion. Other unknown factors are involved. Although aneurysm rupture is uncommon in type II endoleak, the occurrence of rupture has been shown to be unpredictable. This explains the uncertainty in knowing when to intervene to prevent such a fatal outcome. Despite the unclear treatment indications, the present study shows that treatment outcomes are encouraging

The number of perioperative type II endoleaks of the present study appears much higher than that of the earlier Australian ASERNIP study. The current study identified 124 type II endoleaks out of 693 patients compared with 69 out of 959 patients respectively[7]. This could be due to improved procedural imaging.

Our study found a significantly lower incidence of type II endoleak with smoking (p=0.002) and anticoagulation with warfarin (p=0.044). Smoking has been reported as a protective factor for type II endoleak in other studies[5, 8-10], including the study by Koole et al.[11]. Accelerated atherosclerosis from smoking might narrow or occlude the inferior mesenteric and lumbar arteries. Warfarin has not been

reported a protective factor in other studies. Studies specifically looking at Warfarin and its association with type II endoleak have either reported it to be a risk factor[12] or as insignificant[8, 13, 14]. Older age (p=0.034) was found to be a significant risk factor for type II endoleak in the present study, which is supported by several studies[5, 8-10]. Nonetheless, there are also several studies which report it to be insignificant[6, 13-16].

Other significant anatomical variables such as right iliac tortuosity, right and left external iliac diameters have not been evaluated by other studies to date. The extent of right iliac tortuosity in the present study is a subjective measure based on its determination by clinician assessment using the four categories of severity. In our study, the graft type used was relatively homogenous, and therefore a comparison of endoleak incidence with each graft type is unlikely to be meaningful. Pre-emptive coiling of the inferior mesenteric artery was performed in only one patient, and they did not develop any endoleak over the three years.

All three aneurysm ruptures in the present study occurred in patients with type II endoleak. This may suggest that aneurysm ruptures are more likely to occur in type II endoleak. Sac expansion is commonly used as a surrogate marker for risk of aneurysm rupture[1], and sac expansion of 5mm has been previously reported as a risk factor for rupture[2]. The European Society of Vascular Surgery guidelines recommend intervention at 10mm sac expansion[3]. As all ruptures occurred in patients without documented sac expansion, it may be that sac expansion per se is not a surrogate for sac pressurization. Alternatively, sac re-expansion may have occurred between the time of last follow-up and presentation with rupture. Causes for aneurysm rupture other than sac expansion have not been clearly identified to date. More studies are needed to determine other factors in type II endoleak that could lead to rupture.

There was no statistically significant difference in survival between those with and without type II endoleaks Age and smoking were found to be significantly associated with decreased survival in this patient sample. Age has already been reported as a factor associated with poorer long-term (5-year)

survival in patients following EVAR in other studies[17, 18]. Smoking has not been reported as a predictive factor for survival following EVAR to date. A review paper by Lottman et al, has found that mortality does not significantly differ between smokers and non-smokers after EVAR[19]. A systematic review by Khashram et al, however, reports that patients with Chronic Obstructive Pulmonary Disease (COPD) requiring supplemental oxygen is associated with poor long-term survival following abdominal aortic aneurysm repair, i.e. either open or endovascular[20]. COPD mostly occurs in chronic smokers. Perhaps the impact of smoking on survival after EVAR becomes move evident when smoking occurs for long enough to impair respiratory function.

Treatment outcomes seemed satisfactory, given that most type II endoleaks resolved and had sac shrinkage rate post intervention. Both currently used techniques (translumbar vs transarterial), embolic agents (glue, onyx, coils), and ligation (open, laparoscopic) used seemed to obtain satisfactory results. Limitations to this study include variations in criteria used for treating the type II endoleaks, as the data came from multiple institutions with EVARs performed by different surgeons. Another limitation is that only 186 out of 225 patients with type 2 endoleak had adequate follow-up information due to some patients being lost to follow-up, some surgeons failing to supply follow-up data, and some patients being non-compliant with follow-up. A recent published study examined rates of compliance to post-EVAR surveillance and showed only 43% of patients were compliant[21], which is a similar result to that of our study.

5 **Conclusion**

In summary, the present study suggests that type II endoleak with sac expansion does not appear to be associated with aneurysm rupture. Treatment outcomes were encouraging. Aneurysm ruptures in type II endoleak are uncommon but occurred in the absence of documented sac expansion raising the need to identify factors other than sac expansion that can cause aneurysm rupture in type II endoleak.

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Tables

Table I: Baseline characteristics of patients with and without Type II endoleak

Preoperative variables[*]	*Type II endoleak = 225 (32.4%)*[†]	*No Type II endoleak = 468 (67.5%)*	*p-value*
Aneurysm diameter, mm (mean, ±sd)	58.55 (±10.22)[‡]	58.09 (±9.46)	0.995
Infrarenal neck length, mm (mean, ±sd)	27.67 (±12.46)	29.08 (±13.80)	0.390
Infrarenal neck diameter, mm (mean, ±sd)	23.27 (±3.03)	23.85 (±3.59)	0.106
Aortic neck angle, degrees (mean, ±sd)	20.04 (±18.90)	16.84 (±16.48)	0.078
Thrombus in neck (number, %)	39 (25.83)[§]	98 (32.45)	0.148
Occlusive aorto-iliac disease (number, %)	11 (7.19)	50 (15.38)	0.435
Saccular aneurysm (number, %)	22 (14.10)	16 (5.35)	0.712
Artery involved in aneurysm sac (number, %)	122 (77.71)	242 (72.89)	0.254
Aneurysm angle, degrees (mean, ±sd)	27.53 (22.05)	25.48 (21.79)	0.308
RCIA diameter, mm (mean, ±sd)	15.64 (5.86)	15.58 (6.06)	0.890

LCIA diameter, mm (mean, ±sd)	14.71 (4.20)	14.73 (4.71)	0.874
REIA diameter, mm (mean, ±sd)	*9.48 (1.52)*	*11.43 (44.50)*	*0.008*
LEIA diameter, mm (mean, ±sd)	*9.43 (1.65)*	*9.24 (2.94)*	*0.028*
RCIA tortuosity (number, %)			
None	34 (22.22)	94 (28.75)	*0.031*
Mild	90 (58.82)	145 (44.34)	
Moderate	23 (15.03)	71 (21.71)	
Severe	6 (3.92)	17 (5.20)	
Patency IMA (number, % patent)	86 (60.14)	172 (58.11)	0.055
Age, years (mean, ±sd)	*75.21 (±7.77)*	*74.02 (±7.73)*	*0.034*
Gender (number, %)			
Male	200 (88.89)	421 (90.00)	0.847
Female	24 (10.67)	48 (10.26)	
Medications (number, %)			
Warfarin	*15 (6.73)*	*53 (11.73)*	*0.044*
Statin	154 (68.75)	315 (68.78)	0.941
Beta-blocker	82 (36.94)	165 (36.11)	0.800
Smoking (number, %)			
Current	29 (13.30)	108 (24.00)	*0.002*
Ex-smoker	141 (64.68)	274 (60.89)	
Never	48 (22.02)	68 (15.11)	

*For each categorical variable, the number and proportion of patients with that variable out of all patients with and without type II endoleak (who had sufficient data for that variable) are recorded. LCIA tortuosity, RCIA and LCIA calcification, ASA, BMI, CIA isolated, and infrarenal neck shape were also all insignificant. ASA: American Society of Anesthesiologists grading; BMI: Body Mass Index, CIA: common iliac artery; RCIA: Right common iliac artery; LCIA: Left common iliac artery; REIA Right external iliac artery; LEIA: Left external iliac artery; IMA: Inferior mesenteric artery; sd: standard deviation

†Number, % of patients with and without type II endoleak are reported in the header row.

‡Continuous data are shown as the mean ± standard deviation (sd)

§Categorical data are shown as number and %.

343 **Table II: Univariate associations: survival analysis**

Parameter	Label	HR*	Lower CL	Upper CL	P
Age		1.05	1.02	1.08	0.0008
Gender	Male	1.00	-	-	-
	Female	1.58	0.85	2.91	0.146
Statin	Statin (yes)	1.00	-	-	-
	Statin (no)	1.64	1.07	2.52	0.024
Beta blocker	Beta blocker (yes)	1.00	-	-	-
	Beta blocker (no)	0.94	0.61	1.45	0.774
ASAII	ASAII 1-2	1.00	-	-	-
	ASAII 3-4	1.56	0.86	2.82	0.143
Smokes	Never	1.00	-	-	-
(p=0.052)	Ex-smoker	1.77	0.90	3.49	0.100
	Current	2.50	1.19	5.29	0.016
Warfarin	Warfarin (yes)	1.00	1.00	1.00	-
	Warfarin (no)	0.86	0.46	1.61	0.644
Aneurysm diameter		1.01	0.99	1.04	0.1669
RCIA diameter		0.99	0.95	1.03	0.7188
LCIA diameter		1.01	0.97	1.06	0.5070
REIA diameter		0.96	0.85	1.08	0.4789
LEIA diameter		0.96	0.86	1.08	0.5398

344 *HR: hazards ratio; CL = confidence level; ASA: American Society of Anesthesiologists grading;
345 BMI: Body Mass Index; CIA: common iliac artery; RCIA: Right common iliac artery; LCIA: Left
346 common iliac artery; REIA Right external iliac artery; LEIA: Left external iliac artery;
347

348 **Table III: Adjusted and unadjusted models for association between Type II endoleak and survival**

	Unadjusted			Adjusted*		
	HR†	95% CI	P	HR	95% CI	P
Endoleak						
No	1.00	-		1.00	-	
Yes	0.74	0.46-1.20	0.222	0.78	0.48 – 1.28	0.329

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350 *Adjusted for age, sex, and smoking status

351 †HR: hazards ratio; CL = confidence level

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353 **Table IV: Independent predictors of survival**

Predictor	HR*	95% CI	p
Age	1.07	1.04-1.11	<0.0001
Smoking status (p=0.001)			
Never smoked	1.00	-	-
Ex-smoker	2.33	1.17 – 4.66	0.017
Current smoker	4.27	1.95 – 9.34	0.0003

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355 *HR: hazards ratio; CL = confidence level

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357 **Table V: Baseline characteristics in type II endoleak patients with vs without sac expansion**

*Preoperative variables**	Type II endoleak with sac expansion at any time= 35 (23.3%)[†]	Type II endoleak without sac expansion at any time = 115 (76.7%)	*p-value*
Aneurysm diameter, mm (mean, ±sd)	57.16 (±9.60)[‡]	58.82 (±10.35)	0.416
Infrarenal neck length, mm (mean, ±sd)	28.10 (±13.27)	27.60 (±12.38)	0.447
Infrarenal neck diameter, mm (mean, ±sd)	23.75 (±2.39)	23.17 (±3.14)	0.326
Aortic neck angle, degrees (mean, ±sd)	18.87 (±20.39)	20.25 (±18.71)	0.426
Thrombus in neck (number, %)	4 (20.00)[§]	35 (26.72)	0.523
Occlusive aorto-iliac disease (number, %)	2 (9.09)	9 (6.87)	0.660
Saccular aneurysm (number, %)	3 (13.04)	19 (14.29)	1.000
Artery involved in aneurysm sac (number, %)	18 (78.26)	104 (77.61)	0.945
Aneurysm angle, degrees (mean, ±sd)	27.96 (±19.81)	27.45 (±22.48)	0.727
RCIA diameter, mm (mean, ±sd)	15.28 (±6.16)	15.71 (±5.81)	0.557
LCIA diameter, mm (mean, ±sd)	15.09 (±6.67)	14.64 (±3.54)	0.206
REIA diameter, mm (mean, ±sd)	9.45 (±1.51)	9.48 (±1.53)	0.718
LEIA diameter, mm (mean, ±sd)	9.51 (±1.52)	9.42 (±1.67)	0.860
Age, years (mean, ±sd)	76.09 (±6.48)	75.04 (±7.99)	0.845

RCIA tortuosity (number, %)	-	-	
None	9 (39.13)	25 (19.23)	
Mild	12 (52.17)	78 (60.00)	0.200
Moderate	2 (8.70)	21 (16.15)	
Severe	0 (0.00)	6 (4.62)	
Patency IMA (number, % patent)	11 (50.0)	75 (61.98)	0.167
Gender (number, %)			
Male	31 (88.5)	169 (89.42)	0.774
Female	4 (11.43)	20 (10.58)	
Medications (number, %)			
Warfarin	3 (8.82)	12 (6.38)	0.708
Statin	24 (68.57)	130 (69.15)	0.946
Beta-blocker	13 (37.14)	69 (37.10)	0.996
Smoking (number, %)			
Current	3 (8.57)	26 (14.21)	
Ex-smoker	23 (65.71)	118 (64.480	0.617
Never	9 (25.71)	39 (21.31)	

*For each categorical variable, the number and proportion of patients with that variable out of all patients with and without type II endoleak (who had sufficient data for that variable) are recorded. LCIA, ASA, BMI, CIA isolated, and infrarenal neck shape were all insignificant. ASA: American Society of Anesthesiologists grading; BMI: Body Mass Index; CIA: common iliac artery; RCIA: Right common iliac artery; LCIA: Left common iliac artery; REIA Right external iliac artery; LEIA: Left external iliac artery; IMA: Inferior mesenteric artery; sd: standard deviation

†Number, % of patients with and without type II endoleak are reported in the header row.

‡Continuous data are shown as the mean ± standard deviation (sd)

§Categorical data are shown as number and %.

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Table VI: Influence of sac expansion on death and intervention in patients with late type II endoleak

	All			Sac expansion >5mm†			No Sac expansion >5mm		
	Total	**Mortality**	**Intervention**	**Total**	**Mortality**	**Intervention**	**Total**	**Mortality**	**Intervention**
Late type 2 endoleaks*	117‡	24	25	26	5	10	91	19	15
No late type 2 endoleaks	458	70	36	25	8	3	433	62	33

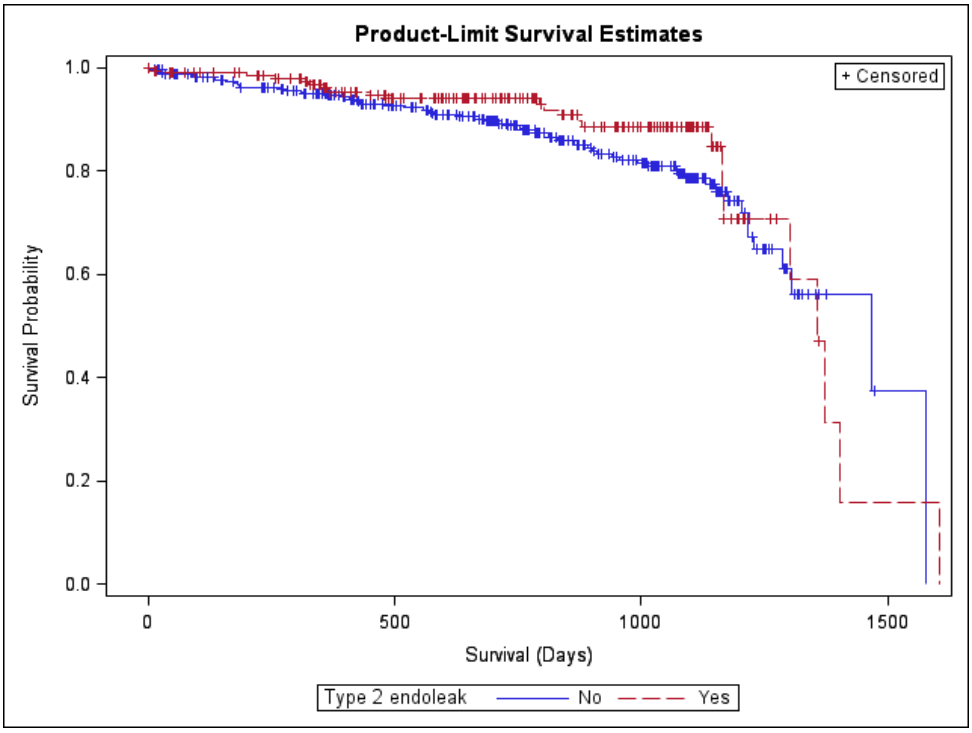
*Late type 2 endoleaks: type 2 endoleaks detected after 30 days

†'Sac expansion >5mm' refers to patients with sac expansion of greater than 5mm at any point following EVAR

‡Data on sac expansion, mortality, intervention are all reported as number of patients in the table

**Note: this table above is required to be printed in color*

Figure 1: Kaplan-Meier survival curve comparing survival between patients with type II endoleak and without type II endoleak



Median survival amongst patients who had and had not had a Type II endoleak

Type II endoleak	*Median survival*	*Lower Limit*	*Upper Limit*
No	1466.00	1289.00	1576.00
Yes	1356.00	1167.00	1404.00

Parameter estimates for survival amongst patients who had had an endoleak compared to those who had not.

Parameter	*Class*	*Hazard Ratio*	*Lower CL*	*Upper CL*	*P*
Type II endoleak	No	1.00	-	-	
	Yes	0.80	0.51	1.24	0.313

**Note: this figure above is required to be printed in color*