Editor's Choice — Type II Endoleak: Conservative Management Is a Safe Strategy CME

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WHAT THIS PAPER ADDS

This study suggests that patients with isolated type II endoleak demonstrate equivalent aneurysm-related mortality and an improved all-cause survival. A conservative approach to the treatment of type II endoleak appears to be safe.

Objective: Type II endoleak is the most common complication after endovascular abdominal aortic aneurysm repair (EVAR); however, its natural history is unclear. The aim of this study was to examine the incidence and outcomes of type II endoleak, at a single institution after EVAR.

Methods: A total of 904 consecutive patients who underwent EVAR between September 1995 and July 2013 at a single centre were entered onto a prospective database. All patients were followed up by duplex ultrasound (DUSS). Patients who developed type II endoleak were compared for preoperative demographics, mortality, and sac expansion.

Results: A total of 175(19%) patients developed type II endoleak over a median follow-up of 3.6 years (1.5—5.9 years); 54% of type II endoleaks spontaneously resolved within 6 months (0.25—1.2 years). No difference was found in preoperative demographics or choice of endograft between the two groups. Survival was significantly higher in the group with type II endoleak (94.1% vs. 85.6%; $p = .01$) and this effect was most pronounced in those with late type II endoleaks (97.7% vs. 85.6% $p = .004$). No difference was seen in aneurysm-related mortality or rate of type I endoleak between the two groups. Freedom from sac expansion (>5 mm from preoperative diameter) was significantly lower in the group of patients with type II endoleak (82.5% vs. 93.2%, $p = .0001$); however, at a threshold of >10 mm from preoperative diameter no difference was seen.

Conclusions: Patients with isolated type II endoleak demonstrate equivalent aneurysm-related mortality and an improved survival.

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INTRODUCTION

Endoleak may be defined as a continued perfusion of the aneurysm sac despite endograft deployment. Type II endoleak is the most common type of endoleak, the most frequent complication of endovascular abdominal aortic aneurysm repair (EVAR) occurring in approximately 10% of patients[1] and may be defined as the backflow of blood from aortic collateral arteries into the aneurysm sac. Despite this, the natural history of type II endoleak is currently unclear, with several retrospective studies suggesting it to be a benign complication[2] while others have associated type II endoleak with adverse late outcomes[3,4] such as continued sac expansion and rupture.

Aortic aneurysm rupture after EVAR secondary to an isolated type II endoleak is rare[5] and the impact of sac expansion on risk of rupture in patients with an isolated type II endoleak is unknown. The decision to intervene is often down to surgeon preference and although vascular centres that do not routinely attempt to abolish the type II endoleak (with or without sac expansion) have reported good outcomes,[2] an optimal strategy for treatment is currently lacking.[6] The aim of this study was to examine the incidence and outcomes of type II endoleak, at a single institution that conservatively manages patients with type II endoleak.

METHODS

Within this study, 904 consecutive patients who underwent EVAR for AAA between September 1995 and July 2013 at a single centre were entered onto a prospective database. EVARs were performed by a dedicated vascular team with choice of

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endograft being dependent on aortoiliac morphology and operator preferences. Collected data included preoperative patient demographics, for example the presence of comorbidities (smoking, diabetes, chronic renal failure, ischaemic heart disease, hypertension, and cerebrovascular disease), preoperative aneurysm diameter, and intraoperative details.

Following EVAR, patients were followed up at regular outpatient appointments with clinical examination and duplex ultrasound scan (DUSS) at 1, 3, and 6 months postoperatively, and at 6-monthly intervals thereafter. At the time of DUSS, data were recorded on sac size, including the presence or absence of endoleak. Computed tomography (CT) scans were performed as necessary to confirm or clarify complications detected by DUSS. All outcomes including the presence of endoleak, stent migration, stent kinking, conversion to open surgical repair, rupture, and death were recorded prospectively on a research database. Those patients in whom DUSS was not possible due to large body habitus were followed up with yearly CT scans with data as above recorded.

Currently DUSSs are performed by a trained vascular technician using a GE Logiq E9 scanner (Fairfield, CA, USA) with a C1—5 MHz broadband curved array transducer (B-mode imaging, colour flow imaging and spectral Doppler modalities); however, over the study period this centre also used a Philips Medical Systems HDI 5000 duplex ultrasound scanner (Bothell WA, USA) with a C5—2 MHz broadband curved array transducer (B-mode imaging, colour flow imaging, and spectral Doppler modalities). B-mode imaging of the aneurysm sac was performed in transverse and longitudinal planes to identify the graft and to examine the sac contents. The maximum sac diameter (outer wall to outer wall) was measured in both anteroposterior and lateral planes (side-to-side diameter from the coronal position) and any changes in sac size were determined. Outer wall to outer wall diameter measurements have recently been demonstrated to have a high repeatability and reproducibility.[7] Colour flow imaging was the principal method used to examine the sac for evidence of endoleak. The colour flow scanner controls were also optimized to detect low-velocity flow from very small endoleaks. Spectral Doppler recordings were taken from any endoleak to examine flow characteristics (pendulum flow suggesting a blind-ending endoleak, or directional flow that might suggest both inflow and outflow). Type II endoleak was defined as blood flow outside the stent graft but within the aneurysm sac caused by retrograde filling from aortic side branches.

Patients with inflammatory or infective aneurysms were excluded from this study. The primary analysis performed aimed to compare those with type II endoleak against a group with no post-EVAR complications by excluding all patients who died within 30 days of EVAR and those who developed other complications of EVAR, for example endoleak (other than type II endoleak), graft migration, and graft kinking. A subgroup analysis aimed to compare outcomes in those with type II endoleak against those without type II endoleak. The no type II endoleak group included patients who had died within 30 days of EVAR and those who had developed other complications of EVAR, for example endoleak (other than type II endoleak), graft migration, and graft kinking. Dangerous/life-threatening complications of EVAR were recognized at follow-up and treated urgently to prevent death, and all type I endoleaks detected in clinic were admitted with a view to inpatient treatment. Any type I endoleak detected on completion angiogram were treated within the first operation. The primary outcome measures were all-cause mortality, aneurysm-related mortality, and freedom from aneurysm growth. Separate analysis were performed in patients with more than 5 mm of sac expansion (compared to original sac size) and those with more than 10 mm of sac expansion (compared to original sac size). Secondary outcomes included spontaneous resolution of the endoleak, defined as no visible type II endoleak on two consecutive DUSS with no intervention having been performed.

Statistical analysis

Patients were analysed separately according to the presence or absence of a type II endoleak. Those with type II endoleak were further classified according to the nature of type II endoleak including early (first imaged on initial post EVAR DUSS), late (first imaged more than 12 months after EVAR), and persistent (present for more than 6 months). Continuous variables were expressed as mean and standard deviation (SD) or median and interquartile range (IQR) depending on the variance, and were analysed by the Fisher exact test or the Mann—Whitney *U* test where appropriate. Kaplan—Meier analysis was used to estimate all-cause mortality survival rates and freedom from aneurysm sac growth rates with log rank analysis used to test the equality of survival distributions between different factors. Censoring occurred on the last date that a patient was known to be alive or had attended an outpatient appointment if lost to follow-up. Cox proportional hazards regression was used to test for independent associations with risk of type II endoleak and sac expansion with results expressed as odds ratios (OR) and confidence intervals (CI). Stepwise selection of the following covariates was performed: age, sac diameter, ischaemic heart disease, hypertension, diabetes, cerebral vascular disease, hyperlipidaemia, chronic renal failure, current smoking, and gender. All statistical analyses were performed using SPSS v.20 (IBM Corp, Armonk, NY, USA) and assumed to be significant if $p < .05$.

RESULTS

During the study period, 904 consecutive patients underwent EVAR. From these 904 patients, follow-up data were available on 773 patients who were included into the analysis (data completeness 86%) from which 175 (19%) patients developed type II endoleak (Table 1). No differences could be seen in the baseline characteristics of those patients lost to follow-up compared to those included within the analysis (Supplemental Table 1). Median follow-up was 4.1 years (1.8—6.2 years) in the no complication group and 3.6 years (1.5—5.9 years) in the type II endoleak

Table 1. Baseline characteristics of patients with and without type II endoleak.

	Type II endoleak ($n = 175$)	No type II endoleak ($n = 598$)	p
Follow-up (yrs)[a]	3.6 (1.5–5.9)	4.1 (1.8–6.2)	
Age (yrs)	74.9 ± 6.6	74.0 ± 8.4	0.1
Spontaneous resolution	95 (54.2)	–	
Time to resolution (yrs)[a]	0.63 (0.25–1.2)	–	
Male	157 (89.7)	553 (92.5)	0.3
Aneurysm diameter (mm)	64 ± 11	63 ± 10	0.5
Hypertension	70 (40)	254 (42.5)	0.6
Ischaemic heart disease	55 (31.4)	183 (30.6)	0.8
Hyperlipidaemia	63 (36)	210 (35.1)	0.8
Diabetes	18 (10.3)	63 (10.5)	1.0
Chronic renal failure	6 (3.4)	33 (5.5)	0.3
Cerebrovascular disease	11 (6.3)	41 (6.9)	0.9
Smoking	95 (54.3)	334 (55.9)	0.7

Note. Continuous data are shown as the mean ± standard deviation and categoric data are shown as number (%).
[a] Median and interquartile range.

group. No significant differences were found in preoperative demographics between the two groups. Within the group who developed type II endoleaks (Table 2), 59 developed early type II endoleak, 53 developed late type II endoleak, and 81 developed persistent type II endoleak. Within the persistent type II endoleak group were 31 late type II endoleaks and 26 early type II endoleaks. Spontaneous resolution of the endoleak occurred in 95 (54.2%) of all type II endoleaks after a median of 0.63 years (0.25–1.2 years). Comparing the rate of spontaneous resolution between different types of endoleak, there appears to be a higher rate of spontaneous resolution within the early endoleak group (78%) than in the late and persistent type II endoleak groups (53% vs. 53%). Furthermore, the early endoleak group appear to spontaneously resolve earlier (0.26 years, 0.13–0.94 years) than the late (1.04 years, 0.6–2.2 years) and persistent (1.3 years, 0.9–2.2 years) groups.

Type II endoleak rates were comparable regardless of the type of endograft used (Table 3). Cox regression analysis adjusted for covariates (age, sac diameter, ischaemic heart disease, hypertension, diabetes, cerebral vascular disease, hyperlipidaemia, chronic renal failure, current smoking, and gender) failed to identify any significant associations with the risk of developing type II endoleak.

The association of type II endoleak with type I endoleak

Within the study period, 27 patients developed type 1 endoleak, six of whom had a type II endoleak. Three of these patients had a persistent and late type II endoleak from which two developed 10 mm of sac expansion. One patient had a persistent type II endoleak and no sac expansion, and two patients had type II endoleak with no sac size changes. Thirty-one patients had both a persistent and late type II endoleak from which only three developed a type I endoleak. These data demonstrate no association between type II endoleak and the development of a type I endoleak ($p = 1.0$).

All-cause and aneurysm-related survival

Analysis of patients with type II endoleak versus a post EVAR, no complication group demonstrated an improved all-cause survival in patients with type II endoleak compared to those with no complication; 94.1% versus 85.6% at 3 years and 80% versus 71.4% at 5 years ($p = .04$),

Table 2. Baseline characteristics of patients with different classifications of type II endoleak.

	Early type II endoleak ($n = 59$)	Late type II endoleak ($n = 53$)	Persistent type II endoleak ($n = 81$)
Median follow-up (yrs)[a]	3.7 (1.7–5.6)	5.6 (3.6–8.6)	5.1 (2.5–7.3)
Age (yrs)	75.3 ± 7.4	74.8 ± 6.7	74.4 ± 6.6
Male	52 (88.1)	43 (81.1)	71 (87.7)
Aneurysm diameter (mm)	64 ± 11	66 ± 12	66 ± 1.2
Spontaneous resolution	46 (78)	25 (47.2)	43 (53)
Time to resolution (yrs)[a]	0.26 (0.13–0.94)	1.04 (0.6–2.2)	1.3 (0.9–2.2)
Hypertension	23 (39)	15 (28)	33 (40.7)
Ischaemic heart disease	15 (25)	9 (17)	19 (23.4)
Hyperlipidaemia	13 (22)	13 (24.5)	25 (30.9)
Diabetes	5 (8.5)	4 (7.5)	8 (9.9)
Chronic renal failure	1 (1.7)	0 (0)	1 (1.2)
Cerebrovascular disease	1 (1.7)	3 (5.6)	9 (11.1)
Smoking	29 (49.1)	20 (37.7)	44 (54.3)

Note. Continuous data are shown as the mean and standard deviation and categoric data are shown as number (%).
[a] Median and interquartile range.

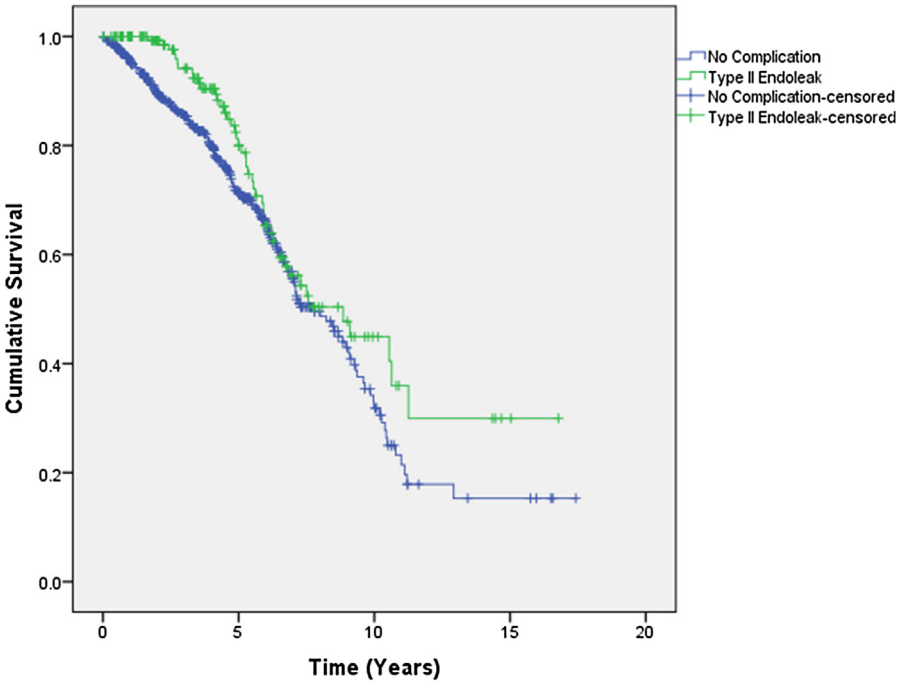
Table 3. Type II endoleak distribution by device models.

Device	Type II endoleak ($n = 175$) No. (%)	No type II endoleak ($n = 598$) No. (%)	p
Cook Zenith	91 (52)	304 (51)	0.8
Cook Trifab	15 (8.6)	52 (8.7)	1.0
Medtronic endurant	18 (10)	54 (9)	0.6
Talent	18 (10)	57 (9.5)	0.6
Anaconda	2 (1)	14 (2.3)	0.5
Gore excluder	24 (14)	79 (13.2)	0.9
Edwards lifepath	2 (1)	2 (0.3)	0.2
Cook uni iliac	—	9 (>1)	—
Local device	—	5 (>1)	—

a finding which persists out to 9 years; 47.7% versus 41.9% (Fig. 1). This remained significant when including patients with known complications into the non-type II endoleak group: 94.1% versus 85.3% at 3 years, which perseveres to 9 years, 47.7% versus 40.4%; $p = .01$.

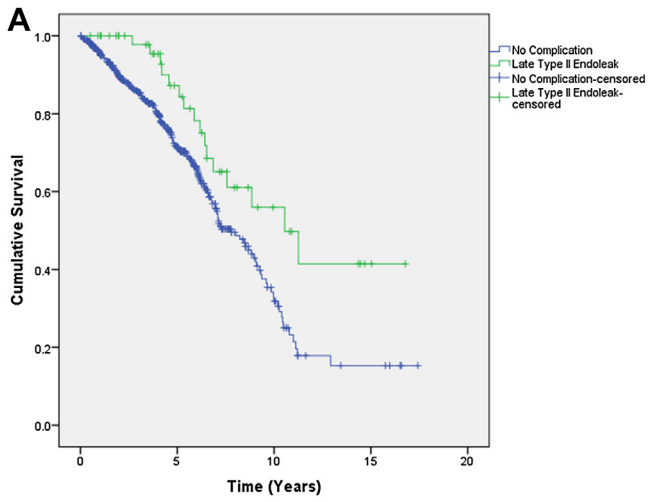
Analysing all-cause mortality within the different classifications of type II endoleaks, those with late endoleaks appear to have a significant survival advantage compared to those with no complication (Fig. 2(A)): 97.7% versus 85.6% at 3 years which persists to 9 years; 56% versus 41.9%, $p = .008$. This again remained significant when including patients with known complications into the analysis: 97.7% versus 86.3% at 3 years and 56% versus 41.4% at 9 years ($p = .004$) over those without type II endoleaks. Analysing survival in patients with persistent and early type II endoleak versus those with no complications demonstrated no difference in all-cause mortality (Fig. 2(B and C)).

No significant difference was demonstrated between aneurysm-related mortality in the group of patients with type II endoleak compared to the group without complication: 98.1% versus 100% at 5 years and 96.8% versus 98.3 at 7 years ($p = .44$). Eleven aneurysm ruptures occurred at a median of 41 months after discharge (IQR 9.5—52.5 months) from which two occurred in the perioperative period and nine occurred after discharge from hospital. The principle complication associated with rupture was a type 1 endoleak ($n = 7$). One patient was reported to have a type III endoleak and consequently ruptured prior to a bridging stent being placed while another patient had a stent strut fracture. One patient who underwent elective EVAR aged 71 for a 90 mm AAA developed a persistent type II endoleak 18 months after EVAR and sac expansion (more than 10 mm from original sac size). The type II endoleak was treated conservatively and the patient was admitted with a proximal type 1 endoleak 56 months after EVAR, treated with proximal cuff. At 72 months this patient was admitted with

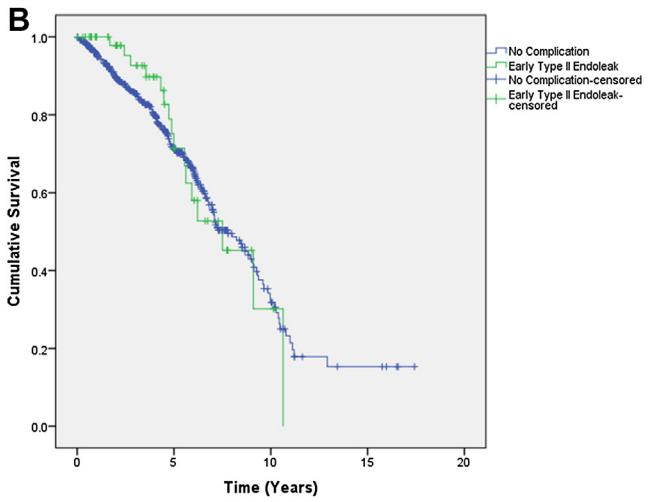


	Years	0.5	1	3	5	7	9
No Complication	N at risk	531	472	334	197	87	40
	Survival %	98.0	95.5	85.6	71.4	55.6	41.9
Type II Endoleak	N at risk	158	143	108	64	31	17
	Survival %	100	100	94.1	80	56.2	47.7

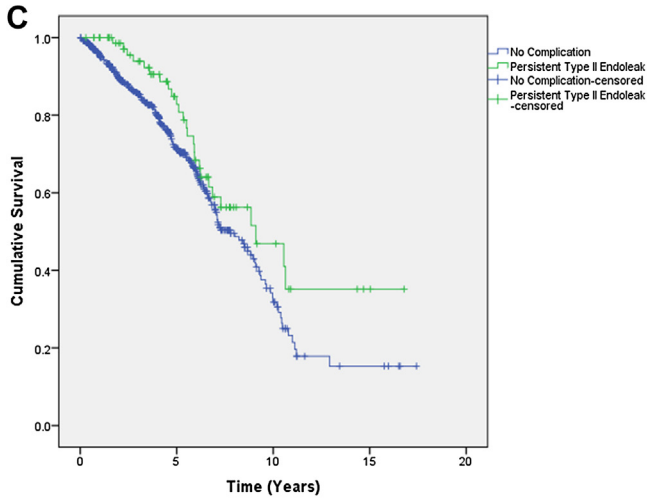
Figure 1. Kaplan—Meier analysis comparing cumulative all cause survival in patients with type II endoleak versus those with no complication. Log rank (Mantel Cox), $p = .04$.



	Years	0.5	1	3	5	7	9
No Complication	N at risk	530	472	334	197	87	40
	Survival %	98.4	95.5	85.6	71.4	55.6	41.9
Type II Endoleak	N at risk	52	50	43	29	18	10
	Survival %	100	100	97.7	84.3	65.1	56.0



	Years	0.5	1	3	5	7	9
No Complication	N at risk	531	472	334	197	87	40
	Survival %	98.0	95.5	85.6	71.4	55.6	41.9
Type II Endoleak	N at risk	56	47	35	18	7	3
	Survival %	100	100	92.6	71.4	52.7	45.2



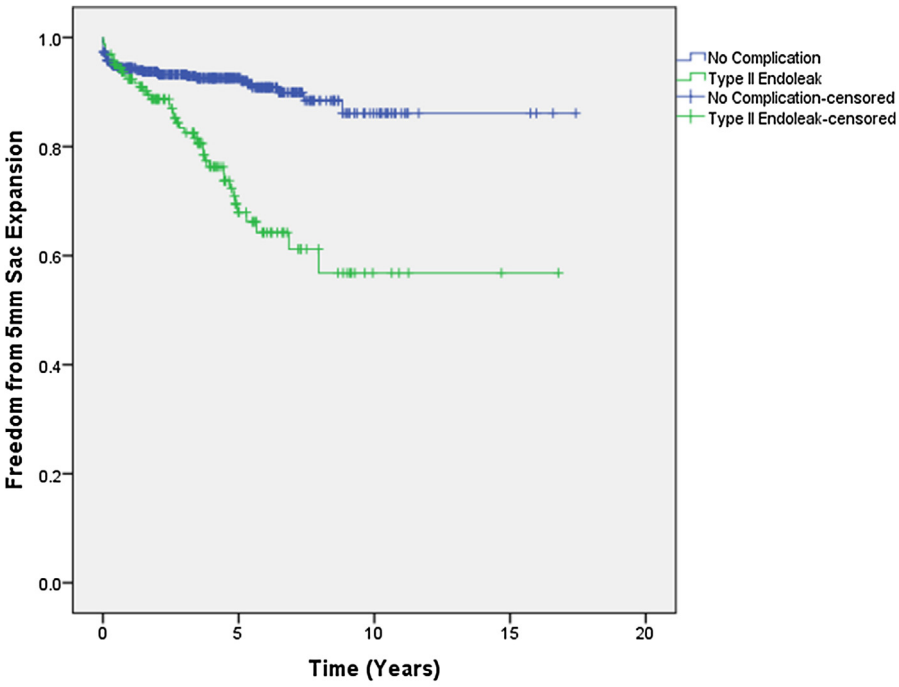
	Years	0.5	1	3	5	7	9
No Complication	N at risk	531	472	334	197	87	40
	Survival %	98	95.5	85.6	71.4	55.6	41.9
Type II Endoleak	N at risk	80	75	58	40	21	10
	Survival %	100	100	93.9	80.8	56.2	46.9

Figure 2. (A) Kaplan–Meier analysis comparing cumulative all cause survival in patients with a late type II endoleak (visualized after 1 year of follow-up) versus those with no complication. Log rank (Mantel Cox), $p = .008$. (B) Kaplan–Meier analysis comparing cumulative all cause survival in patients with a persistent type II endoleak (more than 6 months) versus those with no complication. Log Rank (Mantel Cox), $p = .8$. (C) Kaplan–Meier analysis comparing cumulative all cause survival in patients with an early type II endoleak (visualized on first follow-up) versus those with no complication. Log rank (Mantel Cox), $p = .06$.

a ruptured AAA and died. No patients with an isolated type II endoleak presented with a ruptured AAA.

Sac expansion

Freedom from sac expansion (an increase of 5 mm from preoperative sac size) was demonstrated to be significantly lower in the group of patients with type II endoleak than in the group without complication (Fig. 3) at 3 years (82.5% vs. 93.2%), a finding that can be demonstrated throughout the duration of follow-up ($p = .0001$). Freedom from sac expansion of more than 10 mm from preoperative sac size was however comparable between the two groups ($p = .1$). Cox regression analysis adjusted for covariates (age, sac diameter, ischaemic heart disease, hypertension, diabetes, cerebral vascular disease, hyperlipidaemia, chronic renal failure, current smoking, and gender) confirmed a strong independent association between the presence of type II endoleak and the likelihood of sac expansion (more than 5 mm) after EVAR (OR



	Years	0.5	1	3	5	7	9
No Complication	N at risk	493	440	301	178	75	32
	Survival %	94.8	94.4	93.2	92.6	89.9	86.1
Type II Endoleak	N at risk	149	133	91	42	19	10
	Survival %	95	92.4	82.5	67.9	61.2	56.8

Figure 3. Kaplan–Meier analysis comparing freedom from sac expansion (more than 5 mm) in patients with type II endoleak (visualized after 1 year of follow-up) versus those with no complication. Log rank (Mantel Cox), $p = .0001$.

3.3, 95% CI 1.87–4.91; $p = .0001$) but failed to demonstrate this with sac expansion of more than 10 mm (Fig. 4, $p = .1$).

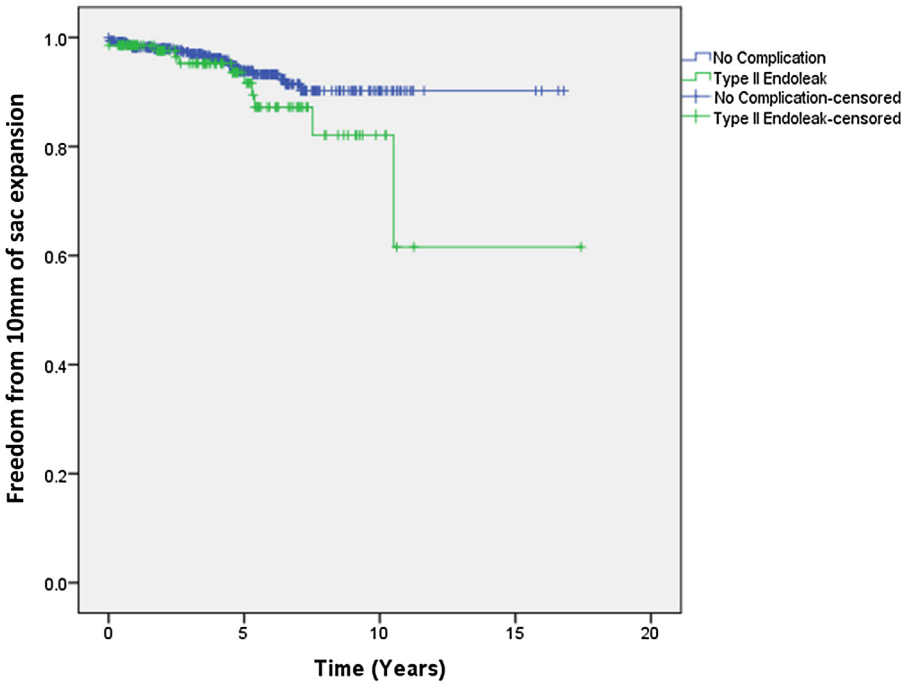
Reintervention after EVAR

Interventions to abolish a type II endoleak were not routinely performed after any specific time period or at any specific sac diameter change; thus, any decision to intervene was down to surgeon/patient preference. A total of nine interventions have been performed to embolize a type II endoleak. (One direct sac puncture and eight transarterial interventions.) Seven of these interventions were in patients who had more than 10 mm of sac expansion and three out of the nine interventions were clinically successful (defined as no type II endoleak present on repeat DUSS). Three patients underwent repeat reintervention: one IMA clipping, one transarterial coil, and one conversion to open surgical repair, all of which were successful. The patient who underwent conversion to open repair had a persistent type II endoleak, failed transarterial intervention and sac expansion from 51 mm to 77 mm. No post-intervention major complications occurred.

DISCUSSION

This study demonstrates that the conservative management of type II endoleak is not associated with an increase in the risk of aneurysm-related mortality, all-cause mortality, 10 mm of sac expansion, or type I endoleak. These data therefore suggest that a conservative approach to the treatment of type II endoleak is safe. Only three out of the nine interventions performed to abolish a type II endoleak were successful, which is in line with previous data suggesting a high risk of treatment failure.[5] Although no complications were noted after intervention in this study, Haulon et al.[8] reported a post-transarterial intervention, mesenteric thrombosis, whereas Uthoff and colleagues[9] reported a pulmonary embolism secondary to leaked embolent and an endograft perforation. The risk of an aggressive approach (treating all type II endoleaks or those with 5 mm of sac expansion) may therefore outweigh any benefit.

In keeping with previous findings,[10] type II endoleak was not associated with an increase in aneurysm related mortality; however, it was associated with an increased survival: 94.1% versus 85.3% $p = .01$. This finding was most pronounced in those with late-onset type II endoleaks (those that developed after 1 year of follow-up): 97.7% versus 86%, $p = .004$. Although these data are not the first to note this unintuitive finding,[10] no clear explanation currently exists. Previous studies[3,4,11] have demonstrated an association between persistent type II endoleaks and adverse outcomes; however, these data demonstrated no increase in aneurysm or all-cause mortality in this group of patients.



	Years	0.5	1	3	5	7	9
No Complication	N at risk	442	400	294	169	77	37
	Survival %	99.2	98.2	97.0	93.9	91.4	90.2
Type II Endoleak	N at risk	128	108	78	47	22	10
	Survival %	98.5	98.5	95.2	91.6	87.2	82.1

Figure 4. Kaplan–Meier analysis comparing freedom from sac expansion (more than 10 mm) in patients with type II endoleak (visualized after 1 year of follow-up) versus those with no complication. Log rank (Mantel Cox), $p = .1$.

One explanation for the improved survival demonstrated in those with type II endoleak could be differences in patient demographics. Although no differences were seen between patient comorbidities in this study, Van Marrewijk et al.[11] found current smoking and a decreased ankle–brachial index (0.87 or less) to reduce the risk of type II endoleak. El Batti[3] and colleagues similarly demonstrated that the risk of type II endoleak was reduced in active smokers (OR 0.16, CI 95% 0.04–0.71, $p = .01$) and patients with coronary artery disease (OR 0.65, CI 95% 0.45–0.92, $p = .01$). It is plausible that those patients with type II endoleak have less profound atherosclerosis both peripherally and centrally; however, studies to demonstrate a reduced cardiovascular risk in this group of patients would be required to investigate this.

This study confirms an association between type II endoleak and sac expansion of 5 mm, which is in keeping with previous studies[3,4]; however, although growth of more than 5 mm has been shown to represent an actual change in aneurysm size rather than a measurement error,[12] intervention at this point is not evidence based. Current guidelines from the European Society of Vascular Surgery[13] are that a conservative approach is appropriate for type II endoleak without sac expansion. Intervention is recommended in the presence of an increased sac diameter of 10 mm or more, with conversion to open surgery if endovascular treatment fails (level 2b).[13] This study failed to show any association between type II endoleak and sac expansion of more than 10 mm from preoperative sac size.

Within this study, 54.2% of all type II endoleaks spontaneously resolved within a median of 7 months. The number of type II endoleaks that spontaneously resolve is variable between studies; for example, one study[2] demonstrated that 48% will resolve within 4 years while another[4] suggested that 80% would resolve within 6 months. Taken together these studies suggest that given time, type II endoleak have a reasonable chance of spontaneously resolving, a view which has been confirmed by a systematic review which demonstrated that a third of all isolated type II endoleaks spontaneously resolve up to 4 years after EVAR.[5]

Choice of imaging is likely to affect the reported incidence of type II endoleak.[14,15] CT has been reported in some studies to achieve the highest sensitivity and specificity for the detection of endoleak[15]; however, in an effort to reduce the cumulative risk of radiation exposure associated with lifelong follow-up, many vascular centres have evolved to DUSS surveillance with CT only used to confirm suspected complications. Although DUSS surveillance may be a limitation of this study, Schmieder and colleagues[16] recently demonstrated that colour duplex imaging has a higher sensitivity in detecting endoleaks requiring intervention (90% vs. 58%)

and has a better diagnostic accuracy in identifying the type of endoleak than CT, which can be improved further by utilizing contrast-enhanced ultrasound.[17] Contrast-enhanced ultrasound was not utilized during any parts of this study. Gray et al.[18] demonstrated that colour duplex imaging had a sensitivity of 100% (specificity of 85%) and could replace CT as the first-line surveillance tool following EVAR as it was associated with a reduction in the cost of surveillance without any loss of imaging accuracy. A further limitation of DUSS in general is that it is operator dependent and that its sensitivity can be reduced in patients with a raised BMI.[19] Furthermore, it is possible that some type II endoleaks are misdiagnosed type I or type III endoleak.

Some studies have suggested that diameter changes after EVAR correlate poorly with volumetric changes, which may be more informative. Future follow-up may therefore include three-dimensional ultrasound imaging. This study is limited by its retrospective nature; however, all outcomes including the presence of endoleak, conversion to open surgical repair, rupture, and death were recorded prospectively. Not all patients attended follow-up (data completeness = 86%); therefore, outcome data for some patients is unavailable for analysis; however, this study did include data for 773 patients. Within this study, the number of EVARs performed was heavily weighted towards the more recent years. For example in the year 2000, 23 EVARs were performed while in 2012, 98 EVARs were performed. This reflects the increasing use of EVAR with time and skews the follow-up times achieved. Although the results of this study appear to highlight an improved survival in a group of patients with type II endoleak after EVAR, this association does not suggest that type II endoleak or growth of the aneurysm after EVAR is desirable.

A limitation of type II endoleak reporting in general is the varying definitions used by authors when referring to early, late, and persistent type II endoleaks. A classification system for reporting type II endoleaks is necessary to enable standardization of reporting and comparison of results. Furthermore, a randomized clinical trial comparing conservative, selective (sac expansion more than 10 mm), and aggressive management of type II endoleak with adequate follow-up would be required to establish a gold standard treatment strategy for these patients. However, the low outcome event rates in patients with type II endoleaks may preclude funding for, and recruitment to, such a trial owing to the large sample sizes required.[20] Data such as those reported in this study should be gathered on an international database to enable robust analysis of outcomes in patients with endoleak.

CONFLICTS OF INTEREST

None.

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APPENDIX A. SUPPLEMENTARY DATA

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.ejvs.2014.06.035.

REFERENCES

1 Gelfand DV, White GH, Wilson SE. Clinical significance of type II endoleak after endovascular repair of abdominal aortic aneurysm. *Ann Vasc Surg* 2006;**20**:69—74.
2 Rayt HS, Sandford RM, Salem M, Bown MJ, London NJ, Sayers RD. Conservative management of type 2 endoleaks is not associated with increased risk of aneurysm rupture. *Eur J Vasc Endovasc Surg* 2009;**38**:718—23.
3 El Batti S, Cochennec F, Roudot-Thoraval F, Becquemin JP. Type II endoleaks after endovascular repair of abdominal aortic aneurysm are not always a benign condition. *J Vasc Surg* 2013;**57**:1291—7.
4 Jones JE, Atkins MD, Brewster DC, Chung TK, Kwolek CJ, LaMuraglia GM, et al. Persistent type 2 endoleak after endovascular repair of abdominal aortic aneurysm is associated with adverse late outcomes. *J Vasc Surg* 2007;**46**:1—8.
5 idloff DA, Stather PW, Choke E, Bown MJ, Sayers RD. Type II endoleak after endovascular aneurysm repair. *Br J Surg* 2013;**100**:1262—70.
6 Karthikesalingam A, Thrumurthy SG, Jackson D, Phd EC, Sayers RD, Loftus IM, et al. Current evidence is insufficient to define an optimal threshold for intervention in isolated type II endoleak after endovascular aneurysm repair. *J Endovasc Ther* 2012;**19**:200—8.
7 Chiu KW, Ling L, Tripathi V, Ahmed M, Shrivastava V. Ultrasound measurement for abdominal aortic aneurysm screening: a direct comparison of the three leading methods. *Eur J Vasc Endovasc Surg* 2014;**47**:367—73.
8 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.
9 Uthoff H, Katzen BT, Gandhi R, Pena CS, Benenati JF, Geisbusch P. Direct percutaneous sac injection for postoperative endoleak treatment after endovascular aortic aneurysm repair. *J Vasc Surg* 2012;**56**:965—72.
10 Cieri E, De Rango P, Isernia G, Simonte G, Ciucci A, Parlani G, et al. Type II endoleak is an enigmatic and unpredictable marker of worse outcome after endovascular aneurysm repair. *J Vasc Surg* 2014;**59**:930—7.
11 van Marrewijk CJ, Fransen G, Laheij RJ, Harris PL, Buth J. EUROSTAR Collaborators. 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.
12 Lederle FA, Wilson SE, Johnson GR, Reinke DB, Littooy FN, Acher CW, et al. Variability in measurement of abdominal aortic aneurysms. Abdominal Aortic Aneurysm Detection and Management Veterans Administration Cooperative Study Group. *J Vasc Surg* 1995;**21**:945—52.
13 Moll FL, Powell JT, Fraedrich G, Verzini F, Haulon S, Waltham M, et al. European Society for Vascular Surgery. Management of abdominal aortic aneurysms clinical practice guidelines of the European society for vascular surgery. *Eur J Vasc Endovasc Surg* 2011;**41**(Suppl. 1):S1—58.

14 Wolf YG, Johnson BL, Hill BB, Rubin GD, Fogarty TJ, Zarins CK. Duplex ultrasound scanning versus computed tomographic angiography for postoperative evaluation of endovascular abdominal aortic aneurysm repair. *J Vasc Surg* 2000;**32**:1142—8.
15 Ashoke R, Brown LC, Rodway A, Choke E, Thompson MM, Greenhalgh RM, et al. Color duplex ultrasonography is insensitive for the detection of endoleak after aortic endografting: a systematic review. *J Endovasc Ther* 2005;**12**:297—305.
16 Schmieder GC, Stout CL, Stokes GK, Parent FN, Panneton JM. Endoleak after endovascular aneurysm repair: duplex ultrasound imaging is better than computed tomography at determining the need for intervention. *J Vasc Surg* 2009;**50**(1012):7 [discussion 1017—8].
17 Gurtler VM, Sommer WH, Meimarakis G, Kopp R, Weidenhagen R, Reiser MF, et al. A comparison between contrast-enhanced ultrasound imaging and multislice computed tomography in detecting and classifying endoleaks in the follow-up after endovascular aneurysm repair. *J Vasc Surg* 2013;**58**:340—5.
18 Gray C, Goodman P, Herron CC, Lawler LP, O'Malley MK, O'Donohoe MK, et al. Use of colour duplex ultrasound as a first line surveillance tool following EVAR is associated with a reduction in cost without compromising accuracy. *Eur J Vasc Endovasc Surg* 2012;**44**:145—50.
19 Elkouri S, Panneton JM, Andrews JC, Lewis BD, McKusick MA, Noel AA, et al. Computed tomography and ultrasound in follow-up of patients after endovascular repair of abdominal aortic aneurysm. *Ann Vasc Surg* 2004;**18**:271—9.
20 Sidloff DA, Stather PW, Choke E, Bown MJ, Sayers RD. Authors' reply: type II endoleak after endovascular aneurysm repair (Br J Surg 2013; 100: 1262—1270). *Br J Surg* 2014;**101**:143.