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## RISK FACTORS AND CONSEQUENCES OF PERSISTENT TYPE II ENDOLEAKS

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### Abstract

**Objectives**—Type 2 endoleaks are common after endovascular aneurysm repair (EVAR) but their clinical significance remains undefined and their management controversial. We determined risk factors for type 2 endoleak and associations with adverse outcomes.

**Methods**—We identified all EVAR patients in the Vascular Study Group of New England abdominal aortic aneurysm (AAA) database. Patients were subdivided into two groups: 1) those with no endoleak or transient type 2 endoleak and 2) persistent type 2 endoleak or new type 2 endoleak (no endoleak at completion of case). Patients with other endoleak types and follow-up shorter than 6 months were excluded. Multivariable analysis was used to evaluate predictors of persistent or new type 2 endoleaks. Kaplan-Meier and Cox regression analysis were used to evaluate predictors of reintervention and survival.

**Results**—2367 EVAR patients had information on endoleaks; 1977 (84%) were in group 1, of which 79% had no endoleak at all and 21% were transient endoleaks that resolved at follow-up. The other 390 (16%) were in group 2, of which 31% had a persistent leak and 69% had a new leak at follow-up that was not seen at the time of surgery. Group 2 was older (mean age 75 vs. 73 years,  $P<.001$ ), and less likely to have COPD (24% vs. 34%,  $P<.001$ ) or elevated creatinine levels (2.6% vs. 5.3%,  $P=0.027$ ). Coil embolization of one or both hypogastric arteries was associated with a higher rate of persistent type 2 endoleaks (12 vs. 8%,  $P=0.024$ ), as was distal graft extension (12% vs. 8%,  $P=0.008$ ). In multivariable analysis, COPD (OR 0.7, 95% CI 0.5–0.9,  $P=0.017$ ) was protective against persistent type 2 endoleak, while hypogastric artery coil embolization (OR 1.5, 95% CI 1.0–2.2,  $P=0.044$ ), distal graft extension (OR 1.6, 95% CI 1.1–2.3,  $P=0.025$ ) and age 80 (OR 2.7, 95% CI 1.4–5.3,  $P=0.004$ ) were predictive. Graft type was also

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associated with endoleak development. Persistent type 2 endoleaks were predictive of post-discharge reintervention (OR 15.3, 95% CI 9.7–24.3,  $P<.001$ ), however not predictive of long-term survival (OR 1.1, 95%CI 0.9–1.6,  $P=0.477$ ).

**Conclusion**—Persistent type 2 endoleak is associated with hypogastric artery coil embolization, distal graft extension, older age, the absence of COPD, and graft type, but not with aneurysm size. Persistent type 2 endoleaks are associated with an increased risk of reinterventions, but not rupture or survival. This reinforces the need for continued surveillance of patients with persistent type 2 endoleaks and the importance of follow-up to detect new type 2 endoleaks over time.

## INTRODUCTION

Endovascular aneurysm repair (EVAR) of abdominal aortic aneurysms (AAA) is associated with significantly higher short-term survival rates when compared to open AAA repair, but equivalent long-term survival rates.<sup>1–5</sup> Though EVAR imparts an early survival benefit, this benefit is not sustained and EVAR is associated with more aneurysm-related reinterventions than open repair.<sup>6</sup> Endoleaks are the most common complication of EVAR and are a frequent indication for reintervention. While Type 1 and Type 3 endoleaks necessitate reintervention and repair, the clinical significance of type 2 endoleaks remains controversial.

Most type 2 endoleaks resolve spontaneously and the one-year post-operative prevalence ranges from 1–10%.<sup>7–9</sup> However, there is evidence that persistent type 2 endoleaks are associated with an increased risk of adverse outcomes (sac enlargement, aneurysm rupture, need for reintervention, conversion to open repair).<sup>9, 10</sup> Results from the EUROSTAR registry suggest that type 2 endoleaks are associated with aneurysmal growth and reintervention, but not with rupture or conversion to open repair.<sup>10</sup> A study of 832 EVAR patients found persistent type 2 endoleaks accounting for 38% of secondary reinterventions.<sup>11</sup> Other studies have shown no such relationships between type 2 endoleaks and adverse outcomes,<sup>12, 13</sup> but these studies may have been insufficiently powered.

Current recommendations suggest intervention for type 2 endoleaks in the presence of aneurysmal growth and/or endoleak persistence; however the exact criteria for reintervention remain undefined.<sup>7, 8, 14</sup> Some physicians argue for aggressive management, such as prophylactic embolization of patent vessels communicating with the endoleak at the time of graft implantation, while others endorse conservative monitoring approaches.<sup>9, 15, 16</sup> Nevertheless, prophylactic embolization has mixed success in preventing type 2 endoleaks.<sup>17, 18</sup>

Our goal was to study the incidence of type 2 endoleaks, both transient and persistent, and to assess adverse outcomes, including post-operative complications and reinterventions. In addition, we wished to identify independent predictors of persistent and delayed type 2 endoleaks, and to determine whether persistent type 2 endoleaks were associated with late reinterventions and survival.

## METHODS

We undertook a retrospective analysis of the Vascular Surgery Group of New England (VSGNE) database, which is a multidisciplinary quality improvement collaborative between both academic and non-academic hospitals. More detailed information can be found at [www.vascularweb.org/regionalgroups/vsgne/pages/home.aspx](http://www.vascularweb.org/regionalgroups/vsgne/pages/home.aspx). This study contained de-identified data only without any protected health information and is therefore not subject to patient consent or Institutional Review Board approval. We used data from January 2003 through December 2014 and identified all patients undergoing EVAR. The presence or absence of an endoleak was recorded by the surgeon at the time of case completion as well as at each follow-up. Patients were subdivided into two categories: 1) those who either had no endoleak at any time or those with a transient type 2 endoleak (defined as an endoleak at completion of the case that was not seen again at any point during follow-up) and 2) persistent type 2 endoleaks (defined as an endoleak at completion of case that was also detected at follow-up) or those who developed a new type 2 endoleak (defined as no endoleak at completion of the case that developed an endoleak at any point postoperatively). Because the ability to detect a type 2 endoleak at the time of graft implantation is depends on several factors (contrast dose, timing of angiography, body habitus, quality of imaging equipment, and how carefully one examines the completion angiogram) which could not be quantified in this study, we felt patients with a “new” type 2 endoleak should be grouped with those with persistent type 2 endoleaks. Patients who underwent reinterventions for a type 2 endoleak were grouped into the latter category. Patients who developed Type 1 or Type 3 endoleaks at any time, or those without endoleak information were excluded from the analysis. Patients with a follow-up time shorter than 6 months were also excluded from our analysis. (Figure 1)

We analyzed potential predictors of persistent type 2 endoleak including baseline demographics and comorbidities and operative factors including hypogastric artery coverage, both intentional (planned prior to procedure to treat distal aneurysm extent) and unintentional (inadvertent extension of graft not necessary to treat distal aneurysm extent). In addition, both preoperative and intraoperative hypogastric artery coiling were analyzed. Presence of an iliac aneurysm was defined by an iliac diameter  $> 1.5\text{cm}$ , using the maximum diameter of either the common or internal iliac artery. We also evaluated the influence of graft type on persistent type 2 endoleak, but limited this to only those grafts with at least 100 implants.

Outcomes at long-term follow-up were also documented. These included the development of sac growth, migration, limb occlusion, aneurysm-related symptoms, and rupture. Reintervention rates for these aforementioned indications were recorded, as were rates of conversion to open repair.

Patient demographics, comorbidities, perioperative details, and outcomes are reported as proportions of the total. We compared categorical variables between outcome subgroups using the  $\chi^2$  and Fisher’s exact tests. The means of continuous variables were compared using student’s t-test. Multivariable logistic regression using purposeful selection<sup>19</sup> was used to determine independent predictors of type 2 endoleak development. Reintervention

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rates and survival were compared between the two subgroups using Kaplan-Meier analysis, survival curves were compared using the log-rank test, and multivariable predictors of reintervention and survival were identified using Cox regression modeling. All statistical analyses were performed using SPSS statistical software version 20 (IBM Corp, Armonk, NY). Statistical significance was defined as  $P<0.05$ .

## RESULTS

### Baseline Characteristics

At the time of analysis, 2,367 patients in the VSGNE undergoing EVAR had information on endoleaks at both the end of the procedure as well as long-term follow-up, which on average was 463 days. Group 1 included 1977 patients (84% of the total) with no or transient endoleak. Of these, 1560 (79%) had no endoleaks and 417 (21%) were transient endoleaks that had resolved spontaneously at follow-up. Group 2 consisted of 390 patients (16% of the total); of these, 120 (31%) had a persistent leak and 270 (69%) developed a new leak that was not seen at the time of surgery (Figure 1). There were 36 patients undergoing an intervention for type 2 endoleak. The baseline demographics and comorbidities of patients in groups 1 and 2 are shown in Table I. Patients in group 2 were older, less likely to have had high creatinine levels ( $>1.8$ ) preoperatively, and less likely to have chronic obstructive pulmonary disease (COPD), or a history of smoking.

### Anatomic Characteristics and Surgical History

Across all anatomical measured characteristics, no significant differences were found between the two groups (Table II). The mean aneurysm diameter was similar for both groups and there was no difference in iliac aneurysm involvement. Aortic rupture and prior aortic surgery did not differ across groups.

### Intraoperative Details

Procedural details are shown in Table III. While coverage of the hypogastric artery and graft configuration (AUI vs. bifurcated) had no association with persistent type 2 endoleaks, hypogastric coil embolization was associated with more persistent type 2 endoleaks (12% vs. 8%,  $P=0.024$ ). Of the 390 patients in group 2, 12% had a graft extension during surgery, while only 8% of those in group 1 had an extension ( $P=0.008$ ). Table IV shows proportions of type 2 endoleak development at the procedure and at follow-up per graft type.

### Complications and Reinterventions

At follow-up, data on sac growth were available for 104 patients, where 46% of patients with a persistent/new type 2 endoleak and 6% of patients with no/ transient type 2 endoleak had sac growth ( $P<.001$ ). (Table V) Conversion to open repair was more common in patients with a persistent or new type 2 endoleak (0.8% vs. 0.2%,  $P=0.024$ ). For the 2,330 patients with available follow-up data on any reintervention, those with persistent type 2 endoleaks were more likely to undergo reinterventions (18.6% vs. 1.5%), which was significant by Kaplan Meier ( $P<.001$ ) (Figure 2). All patients who developed sac growth underwent reintervention. Among patients for whom long-term data were available regarding graft migration or symptoms/rupture there were no significant differences in the rates of these

complications. Survival of patients with persistent type 2 endoleaks did not differ from those with no/transient type 2 endoleaks (Figure 3). Among patients with persistent or new type 2 endoleak, there was no difference in long-term survival in those with and without re-intervention (HR 0.8, 95% CI 0.3–1.8, P=0.547).

### Multivariable Predictors

Multivariable predictors of persistent type 2 endoleaks were hypogastric artery coil embolization, distal graft extension, and age 80 or older, while COPD was protective. After accounting for these characteristics, the Powerlink (OR 0.5, 95% CI 0.3–0.9, P=0.012) and Endurant (OR 0.5, 95% CI 0.3–0.9, P=0.026) (which are both no longer available) were protective of persistent or new type 2 endoleaks, while the Excluder with its lower permeability fabric was predictive of persistent or new type 2 endoleaks (OR 1.5, 95% CI 1.1–2.0, P=0.011). (Table VI) Persistent type 2 endoleaks were predictive of post-discharge reinterventions (OR 15.3, 95% CI 9.7–24.3, P<.001), however not predictive of mortality (Table VII). Predictors of mortality were COPD, older age, creatinine level >1.8 mg/dL, and discharge to an institutional facility. There was insufficient power to determine independent predictors of conversion to open repair.

## DISCUSSION

In this large study of 2367 patients who underwent EVAR, we found that persistent type 2 endoleaks are associated with hypogastric coil embolization, distal graft extension, the absence of COPD, age 80 and older, and graft type. Patients with persistent type 2 endoleaks are more likely to have sac expansion and to undergo reintervention during follow-up.

The EUROSTAR registry showed that coil embolization of side branches increased the risk of type 2 endoleak. They showed that blocking one or two hypogastric arteries was predictive on univariate analysis, but not on multivariable analysis.<sup>10</sup> In addition, Conrad et al. found an increased risk for secondary intervention in patients who had coil embolization of any vessel, but did not find an association of hypogastric embolization with persistent type 2 endoleaks.<sup>11</sup> The results from our study provide a possible connection between these two prior findings, suggesting that the increased risk of reintervention, seen in patients with coil embolization, may stem from an increased risk of developing persistent type 2 endoleaks. A possible explanation for the increased risk of developing persistent type 2 endoleaks among patients who receive hypogastric artery coiling is that these patients likely have more extensive aneurysms that extend into the common iliac arteries. Although the presence of iliac aneurysms showed no correlation with persistent type 2 endoleaks, some iliac aneurysms might have a sufficient distal neck that does not require extension into the external iliac artery. Increased blood flow through lumbar arteries and the inferior mesenteric artery (IMA) may be enhanced to provide flow to the pelvis, thus contributing to persistent lumbar and IMA type 2 endoleaks. Coverage without coiling likely preserves some hypogastric branches that would otherwise be lost with embolization, which may reduce the need for enhanced flow from lumbar and IMA branches. In contrast, intraoperative coverage of the hypogastric arteries, whether intentional or not, was not associated with persistent type 2 endoleak development.

In our analyses the Powerlink and Endurant stent grafts had lower rates of new or persistent type 2 endoleaks. The Endurant stent graft however is no longer available and was enhanced in 2013 into the Endurant II with a lower profile, additional limb lengths and an aorto-uniiliac option. The number of patients treated with the Endurant II in our study was too small to draw any conclusions. The Powerlink is also no longer available in the US as it was replaced by the AFX device. Powerlink has a longer main body and external billowing fabric that should occupy more space thereby potentially decreasing type 2 endoleaks. However, the Zenith graft, which also has a longer main body was not associated with as low a rate of type 2 endoleak. The low-permeability Excluder was predictive of type 2 endoleaks. The original Excluder was associated with sac expansion despite an absence of radiographic endoleak, which led to an introduction of a new graft with lower permeability in 2004. This stent graft initially had the lowest profile for years and may have allowed an increase in treatment of those with peripheral arterial disease and women, which may have an impact on development of type 2 endoleak, but we are unable to analyze this further.

Our study demonstrates that the development of persistent type 2 endoleaks correlates with an increase in sac growth at follow up. This is consistent with a previous report, which found sac enlargement in 54.5% of patients with persistent type 2 endoleaks during their median follow-up period of 28.7 months.<sup>9</sup> In our analyses, 46% of patients with persistent type 2 endoleaks had sac growth. Along with the risk of sac growth, we found that patients with persistent type 2 endoleaks were more likely to undergo a reintervention. Buth et al. also found an increased risk for intervention and aneurysmal growth, without a change in rupture risk or survival.<sup>20</sup> Jones et al. showed similar findings, but their results also revealed an increased risk of aneurysm rupture, which our study did not confirm.<sup>9</sup> Our analysis includes data with a 1-year mean follow-up period, and thus limits our ability to assess the risk of late rupture. The overall increased risk of reintervention reinforces the need for continued surveillance of patients with persistent type 2 endoleaks and if necessary, intervening for sac expansion.

Our study revealed no relationship between aneurysm size and persistent type 2 endoleaks. This is consistent with some earlier studies,<sup>9, 10</sup> while other reports have demonstrated that the risk of developing type 2 endoleaks is increased with larger aneurysm diameter.<sup>21, 22</sup> Aneurysm sac diameter was also found in one study to be an independent predictor of secondary reintervention.<sup>11</sup> Our results, however, do not demonstrate this associated risk when accounting for persistent type 2 endoleak. Others have noted an association of persistent type 2 endoleak with and an increasing numbers of patent aortic branches.<sup>21, 22</sup> However, these data are not collected in the VSGNE dataset.

Another significant predictor of type 2 endoleaks in our analysis is increased patient age, which is consistent with previous studies.<sup>10, 21</sup> Van Marrewijk et al. similarly showed that patients with persistent endoleaks were two years older than patients without endoleaks.<sup>10</sup> An explanation for this trend has yet to be proposed. Also, gender did not affect the incidence of persistent type 2 endoleak in our analyses.<sup>23</sup> Recent work by Dubois et al. also demonstrated this and showed that the incidence of endoleaks did not vary significantly across gender, despite differences in anatomic characteristics of aneurysms.<sup>24</sup>

Earlier studies have reported an association between COPD and spontaneous resolution of type 2 endoleaks,<sup>21</sup> which is confirmed in our study. This phenomenon may be explained by an increased blood viscosity in COPD leading to increased thrombus formation or atherosclerosis in vessels.<sup>10, 21</sup> We also demonstrated on univariate analysis that smoking was associated with a lower rate of persistent type 2 endoleak, which has been seen in previous studies.<sup>10, 20, 25</sup> However, as smoking is the primary risk factor for COPD, these two variables may be perfectly collinear and in our study smoking was no longer significant after accounting for COPD in multivariable analysis.

To limit the number of reinterventions that occur post-EVAR, further research is needed to explain the association between aneurysms that require hypogastric branch coiling and an increased risk of persistent type 2 endoleak.

Although our study has a large number of patients from multiple institutions, an important caveat is that it is not a randomized controlled trial. One important limitation is that one-year follow-up is not available for a significant percentage of patients in the database. Long-term follow-up in VSGNE is required at one year, accepting anything after nine months. Entry of longer follow-up data is suggested but not required. In this analysis, true one-year follow-up (> 365 days) was 74%. It is possible that entry of further longer-term data is associated with re-interventions. Therefore, analysis of data beyond this time point may overestimate adverse events.

Furthermore, because the variable within the VSGNE is recorded as symptoms and/or rupture, we are unable to distinguish patients who developed symptoms from those who ruptured. This study is also limited by our lack of information on all potentially relevant anatomy, e.g. thrombus burden and patency of side branches. Importantly, long-term follow-up data for several outcome variables are limited (e.g. sac growth, graft migration, conversion to open, and rupture). For example, only 101 patients had data collected on whether they developed symptoms and/or rupture. Also limited are the specifics concerning what postoperative surveillance schedules and imaging modalities were used and what types of reinterventions were performed when undertaken. It is also possible that there is reporting bias, in that those performing re-intervention or noting sac growth may be more likely to carefully inspect for and document the presence of a type 2 endoleak. However, to our knowledge, our study represents the largest American series and may be broadly generalizable due to both academic and community hospitals that the VSGNE incorporates.

## CONCLUSION

Type II endoleaks that persist or develop during follow-up are associated with future sac growth and reintervention. Persistent type 2 endoleaks are predicted by coil embolization of hypogastric arteries, distal graft extension, older age, the absence of COPD, and graft type; allowing heightened awareness in those patients at increased risk. Future analysis with longer follow-up and more thoroughly documented outcome data on contemporary grafts will improve our understanding of persistent type 2 endoleak.

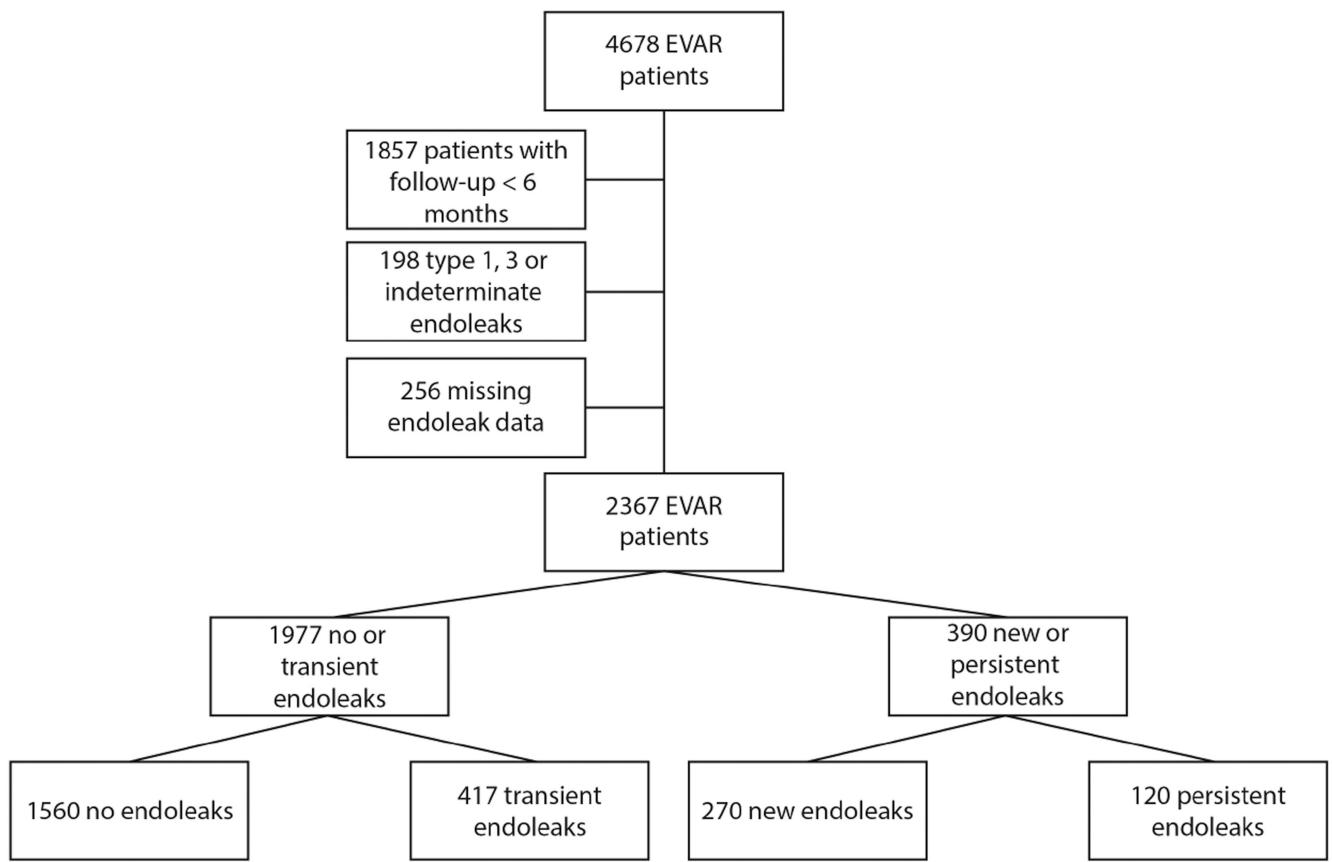
## Acknowledgments

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**Figure 1.**  
Consort diagram of inclusion- and exclusion criteria for this study.

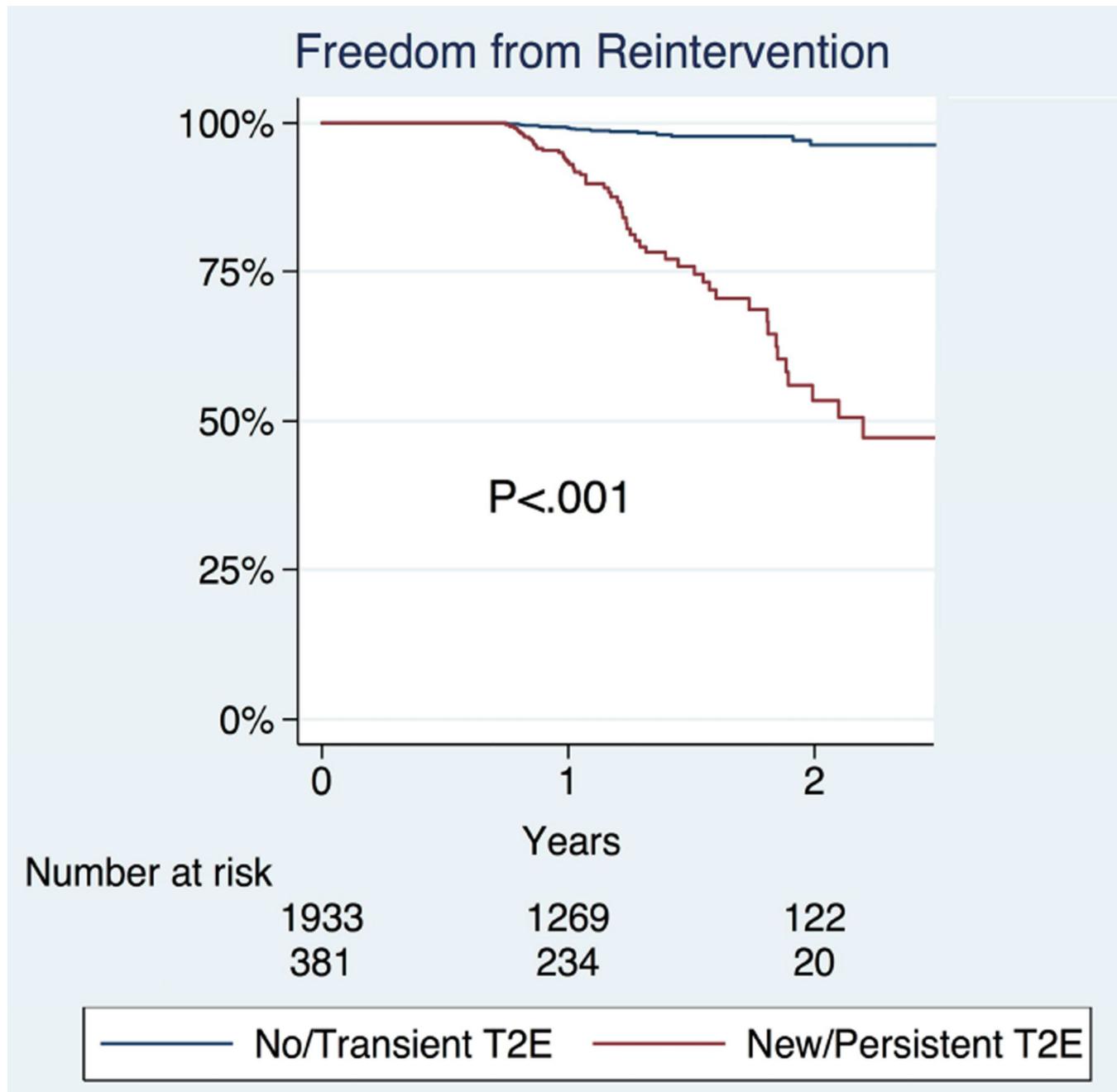
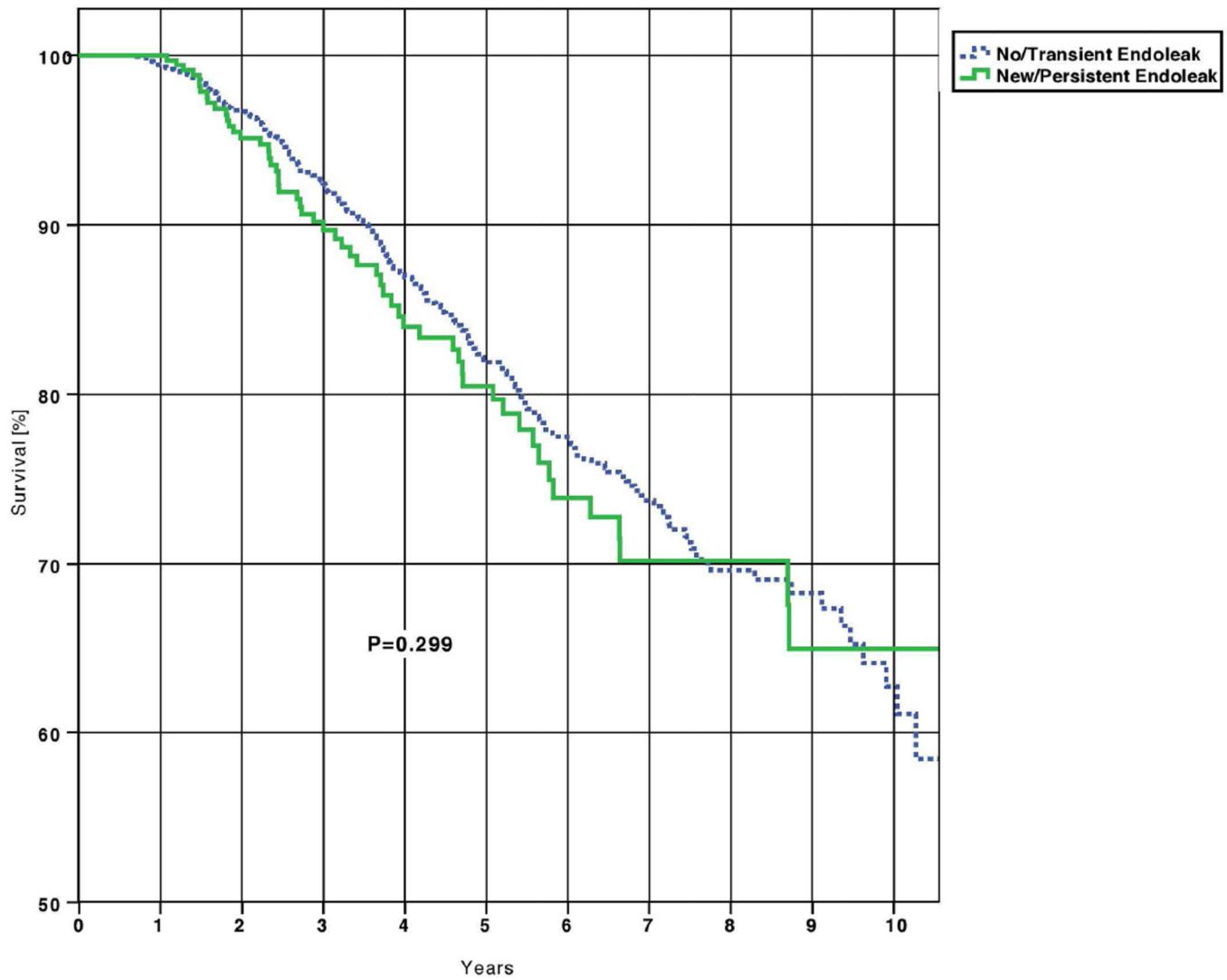


Figure 2.

Figure 2. Freedom from reinterventions among patients with no/transient type 2 endoleaks vs. patients with persistent/new type 2 endoleaks



**Figure 3.**

Survival of patients with no/transient type 2 endoleaks vs. patients with persistent/new type 2 endoleaks.

**Table I**

Comparison of baseline characteristics and comorbidities between patients with no/transient type 2 endoleaks vs. patients with persistent/new type 2 endoleaks.

	No/Transient Type II Endoleak N=1977	Persistent/New Type II Endoleak N=390	P-value
Age (mean in yrs $\pm$ SD)	73 $\pm$ 8.1	75 $\pm$ 8.1	<.001
Male	82%	79%	0.157
White race	97%	97%	0.876
Smoking history	87%	80%	<.001
Hypertension	85%	82%	0.078
Diabetes Mellitus	19%	19%	0.947
Coronary Artery Disease	33%	32%	0.479
CABG/PCI	30%	28%	0.264
Congestive Heart Failure	8.4%	9.2%	0.613
COPD	34%	24%	<.001
Dialysis-dependent	0.8%	0.5%	0.538
Creatinine, >1.8 mg/dL	5.3%	2.6%	0.027
Preoperative Medication			
Aspirin	74%	70%	0.087
Statin	71%	69%	0.344
Plavix	7.4%	7.7%	0.862

CABG/PCI, coronary artery bypass graft/percutaneous coronary intervention; COPD, chronic obstructive pulmonary disease;

**Table II**

Aortic surgical history and anatomic characteristics.

	No/Transient Type II Endoleak N=1977	Persistent/New Type II Endoleak N=390	p-value
Aneurysm Diameter (mean, mm $\pm$ SD)	57.0 $\pm$ 24.2	57.7 $\pm$ 11.1	0.615
Iliac Aneurysm			0.412
None	77%	76%	
Unilateral	13%	11%	
Bilateral	11%	13%	
Prior Aortic Surgery			0.775
None	98%	98%	
AAA	1.1%	0.5%	
SAAA	0.2%	0.3%	
Bypass	0.3%	0.5%	
Other	0.8%	0.5%	
EVAR	0.2%	0.0%	
Rupture	2.4%	2.8%	0.605

AAA, infrarenal abdominal aortic aneurysm repair; SAAA, suprarenal abdominal aortic aneurysm repair

**Table III**

Intraoperative details.

	No/Transient Type 2 Endoleak (N=1977) %	Persistent/New Type 2 Endoleak (N=390) %	P-value
Graft Configuration			0.755
Aorto-bi-iliac	94.0%	94.9%	
Aorto-uni-iliac	4.7%	3.8%	
Aorto-aortic	1.3%	1.3%	
Hypogastric Artery Coiling	8.3%	12.0%	0.024
Hypogastric Artery Coverage	12.4%	14.4%	0.287
Graft Extension	7.7%	12.0%	0.008

\* Hypogastric artery coiling and graft extension data were missing for 183 patients

Development of persistent or new type 2 endoleaks at procedure and at follow-up by graft type.

**Table IV**

	Type II Endoleak at procedure			Type II Endoleak at Follow-up		
	N	total	%	N	total	%
AneuRx	86	257	33.5%	27	257	10.5%
Endurant	61	194	31.4%	17	194	8.8%
Excluder Low-Permeability	175	747	23.4%	167	747	22.4%
Powerlink	60	289	20.8%	23	289	8.0%
Talent	61	147	41.5%	8	147	5.4%
Zenith	124	537	23.1%	76	537	14.2%

Grafts with fewer than 100 implants were not included in this analysis (Aneure, Excluder Original, MEGS/V1, Cordis, Aorfix, Zenith Low Profile, AFX, Endurant II, Zenith Flex, Other)

**Table V**

Complications and reinterventions at Follow-up.

	Data Available For	No/Transient Type 2 Endoleak	Persistent/New Type 2 Endoleak	P-value
			#	%
Any Reintervention	2,330	1.5%	18.6%	<.001
Conversion to Open Repair	2,298	0.2%	0.8%	0.024
Sac Growth	104	5.9%	45.7%	<.001
Graft Migration	103	11.4%	2.9%	0.082
Symptoms or Rupture	101	0.0%	4.5%	0.210

**Table VI**

Multivariable predictors of persistent/new type 2 endoleaks.

Variable	Odds Ratio	95% CI	P-value
Age			
<60	-	-	-
60–69	1.5	0.7–2.9	0.271
70–79	1.7	0.9–3.3	0.119
80	2.7	1.4–5.3	0.004
History of Smoking	0.7	0.5–1.0	0.076
COPD	0.7	0.5–0.9	0.017
Preop ASA	0.8	0.6–1.1	0.202
Hypertension	0.8	0.6–1.0	0.093
Creatinine, >1.8 mg/dL	0.5	0.3–1.0	0.059
Hypogastric coiling (preop or intraop)	1.5	1.0–2.2	0.044
Graft extension	1.6	1.1–2.3	0.025
Grafttype			
AneuRx	0.8	0.5–1.3	0.357
Endurant	0.5	0.3–0.9	0.026
Excluder Low Permeability	1.5	1.1–2.0	0.011
Powerlink (Intuitrak)	0.5	0.3–0.9	0.012
Talent	0.6	0.3–1.1	0.119
Zenith	ref	ref	ref

**Table VII**

Multivariable predictors of reintervention and survival

Variable	Odds Ratio	95% CI	P-value
<i>Predictors of Post-discharge Reintervention</i>			
Female	1.0	0.6–1.8	0.899
Smoking History	1.1	0.6–1.9	0.814
Creatinine, >1.8 mg/dL	0.6	0.2–2.5	0.454
COPD	1.2	0.8–2.0	0.394
Persistent/New Type 2 Endoleak	15.3	9.7–24.3	<.001
<i>Predictors of Mortality</i>			
Age, years			
<60	-	-	-
60–69	1.4	0.6–3.2	0.424
70–79	2.5	1.1–5.4	0.025
80	3.4	1.5–7.5	0.003
Female	1.1	0.8–1.5	0.551
Not Home Discharge	1.6	1.0–2.4	0.032
CHF	1.4	1.0–2.1	0.078
COPD	1.6	1.2–2.1	<.001
Creatinine, >1.8 mg/dL	1.7	1.1–2.8	0.031
Persistent/New Type 2 Endoleak	1.1	0.9–1.6	0.477