

CLINICAL RESEARCH STUDIES

Persistent type 2 endoleak after endovascular repair of abdominal aortic aneurysm is associated with adverse late outcomes

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Objective: Type 2 endoleak occurs in up to 20% of patients after endovascular aneurysm repair (EVAR), but its long-term significance is debated. We reviewed our experience to evaluate late outcomes associated with type 2 endoleak.

Methods: During the interval January 1994 to December 2005, 873 patients underwent EVAR. Computed tomography (CT) scan assessment was performed ≤ 1 month of the operation and at least annually thereafter. Sequential 6-month CT scan follow-up was adopted for those patients with persistent type 2 endoleaks, and reintervention was limited to those with sac enlargement > 5 mm. Study end points included overall survival, aneurysm sac growth, reintervention rate, conversion to open repair, and abdominal aortic aneurysm (AAA) rupture. Preoperative variables and anatomic factors potentially associated with these endpoints were assessed using multivariate analysis.

Results: We identified 164 (18.9%) patients with early (at the first follow-up CT scan) type 2 endoleaks. Mean follow-up was 32.6 months. In 131 (79.9%) early type 2 endoleaks, complete and permanent leak resolution occurred ≤ 6 months. Endoleaks persisted in 33 patients (3.8% of total patients; 20.1% of early type 2 endoleaks) for > 6 months. Transient type 2 endoleak (those that resolved ≤ 6 months of EVAR) was not associated with adverse late outcomes. In contrast, persistent endoleak was associated with several adverse outcomes. AAA-related death was not significantly different between patients with and without a type 2 endoleak ($P = .78$). When evaluating patients with no early endoleak vs persistent endoleak, freedom from sac expansion at 1, 3, and 5 years was 99.2%, 97.6%, and 94.9% (no leak) vs 88.1%, 48.0%, and 28.0% (persistent) ($P < .001$). Patients with persistent endoleak were at increased risk for aneurysm sac growth vs patients without endoleak (odds ratio [OR], 25.9; 95% confidence interval [CI] 11.8 to 57.4; $P < .001$). Patients with a persistent endoleak also had a significantly increased rate of reintervention (OR, 19.0; 95% CI, 8.0 to 44.7); $P < .001$). Finally, aneurysm rupture occurred in 4 patients with type 2 endoleaks. Freedom from rupture at 1, 3, and 5 years for patients with a persistent type 2 endoleak was 96.8%, 96.8%, and 91.1% vs 99.8%, 98.5%, and 97.4% for patients without a type 2 endoleak. Multivariate analysis demonstrated persistent type 2 endoleak to be a significant predictor of aneurysm rupture ($P = .03$).

Conclusions: Persistent type 2 endoleak is associated with an increased incidence of adverse outcomes, including aneurysm sac growth, the need for conversion to open repair, reintervention rate, and rupture. These data suggest that patients with persistent type 2 endoleak (> 6 months) should be considered for more frequent follow-up or a more aggressive approach to reintervention. (*J Vasc Surg* 2007;46:1-8.)

Endovascular abdominal aortic aneurysm repair (EVAR) is an increasingly used method of repairing abdominal aortic aneurysms (AAAs) in patients with suitable anatomy.¹ This less invasive technique has been established as a safe and effective method of short-term aneurysm

exclusion.²⁻⁵ However, unique mid-term and long-term graft-specific complications related to EVAR continue to present management dilemmas for clinicians.^{6,7} Principal among these adverse events is the presence of type 2 endoleaks, which occur at some interval after EVAR in 20% to 30% of patients.⁸

The more frequent need for reintervention, the requirement for close, long-term surveillance in patients with type 2 endoleaks and the potential for late conversion to open repair have tempered the enthusiasm for EVAR and focused uncertainty about the long-term durability of the procedure.^{9,10} The lack of data on late outcomes associated with type 2 endoleaks has contributed to the wide variety of approaches to their management currently found in the literature.^{11,12} Some authors advocate intervention on all

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Competition of interest: none.

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leaks persisting >6 months, regardless of change in aneurysm sac size.¹³ The rationale for early reintervention includes risk of rupture with continued endoleak, lack of patient compliance with the close follow-up needed with persistent endoleaks, and the cost and potential negative renal effects of long-term computed tomography (CT) scan surveillance.

Other investigators advocate a more conservative approach to type 2 endoleaks.¹⁴⁻¹⁷ These authors cite the extremely low incidence of aneurysm rupture associated with type 2 endoleaks. Furthermore, some have demonstrated that the cost of early reintervention is actually greater than sequential radiographic follow-up because many type 2 endoleaks resolve spontaneously within months of EVAR.¹⁵

This study examines the incidence and outcomes of type 2 endoleaks at a single institution during a 12-year experience with 873 patients undergoing EVAR.

METHODS

During the interval January 1994 to December 2005, 873 patients underwent EVAR at our institution. Patients with thoracic aortic aneurysms, anastomotic aortic aneurysms, or isolated iliac aneurysms were excluded from analysis. Clinical and demographic data were collected from hospital records and surgeons' office charts. Radiographic follow-up information was obtained from radiology reports and through the office notes of the primary surgeon. All CT scans reported as demonstrating a type 2 endoleak were reviewed by a vascular surgeon. Any discrepancy in imaging interpretation was adjudicated by an independent surgeon blinded to the study.

An enhanced CT scan with 3-mm cuts and three-dimensional reformatting was used preoperatively to determine if each patient presenting for EVAR met the radiologic criteria for stent graft repair. Ten different stent graft designs were included in this study: custom-made at Massachusetts General Hospital, Vanguard (Boston Scientific, Natick, Mass), Cordis (Cordis, Miami Lakes, Fla), LifePath (Edwards Lifesciences, Irvine, Calif), Ancure/EVT (Guidant, Indianapolis, Ind), Zenith (Cook, Bloomington, Ind), Excluder (W.L. Gore & Associates, Flagstaff, Ariz), AneuRx (Medtronic, Minneapolis, Minn), and Powerlink (Endologix, Irvine, Calif). Overall results from our EVAR experience have been published previously.²

Follow-up. All patients received an enhanced CT scan ≤ 1 month of the procedure at Massachusetts General Hospital. All CT scans were performed with intravenous contrast material and thin collimation. After a nonenhanced scan was performed, a bolus injection of contrast was administered at 4 mL/s, with a 25-second preparation delay. The images were reconstructed with 2.5-mm cuts. Delayed-phase images were also obtained for all patients who underwent EVAR. The average time to the first follow-up CT scan was 12 days. Patients were then clinically evaluated and underwent CT scans at 6 and 12 months after EVAR, and then at least yearly thereafter.

Only early type 2 endoleaks, defined as those present ≤ 1 month of EVAR placement, were evaluated in this study. The presence of a type 2 leak was determined by CT scan in nearly all patients. If the radiologist or surgeon could not differentiate between a type 1, 2, or 3 leak by CT examination, arteriography was performed. Arteriogram was rarely needed to determine type of endoleak, however, and was used in only 11 patients. Eight of these patients demonstrated a type 3 endoleak, and three had type 1 endoleaks from a distal fixation site. None of the indeterminate endoleaks were found to be from a type 2 endoleak.

Patients with early type 2 endoleaks underwent enhanced CT scan every 6 months until leak resolution and then yearly thereafter. Endoleaks were further subdivided into persistent or nonpersistent leaks. Persistent endoleak was defined as a type 2 endoleak that did not resolve ≤ 6 months, whereas a transient endoleak did resolve ≤ 6 months after EVAR.

Outcomes. The primary outcomes examined were AAA-related death, freedom from rupture, and freedom from aneurysm sac growth. Freedom from conversion to open repair and reintervention associated with transient leaks, persistent type 2 endoleaks, and no early endoleak were also evaluated. Deaths were identified through hospital and office charts as well as the Social Security Death Index database. All deaths associated with persistent type 2 endoleaks were confirmed by intraoperative findings or CT scans consistent with rupture.

Aneurysm sac growth was defined as growth >5 mm from the preoperative maximal sac diameter. Preoperative variables previously identified as potentially associated with type 2 endoleaks or our end points, or both, were included in our analysis.¹⁸ Anatomic variables including preoperative aneurysm size, neck width, neck length, presence of concomitant iliac aneurysm, size of iliac vessels, and preoperatively patent lumbar and inferior mesenteric artery vessels were also examined in our data collection and analysis.

The decision of whether to reintervene and the method of treatment were made by the attending surgeon. In general, type 2 endoleaks were not treated unless they were associated with aneurysm sac growth ≥ 5 mm. Success at reintervention was defined as resolution of the leak based on CT scan and a maximal sac diameter that did not continue to increase. The Institutional Review Board of the Massachusetts General Hospital approved the study.

Statistical analysis. All statistical analysis was performed using SPSS software (SPSS, Inc, Chicago, Ill). Preoperative and anatomic variables were assessed for patients with transient, persistent, and no endoleak by using analysis of variance with Tukey post hoc analysis. Freedom from rupture, aneurysm sac growth, conversion to open repair, and reintervention were assessed by using Kaplan-Meier life-table analysis, and the log-rank test was used when subgroups were compared. The Cox regression hazards model was performed to identify variables that influenced late outcomes.

Table I. Patient demographics and preoperative variables associated with type 2 endoleaks

Variable	Type 2 endoleak		No early type 2 endoleak	P
	Transient	Persistent		
Patients	131	33	709	
Mean follow-up (months)	32.6	43.1	28.8	
Age, years (mean)	76.2	75.7	75.5	.54
Gender-male (%)	81.6	88.6	81.1	.79
AAA size, mm (mean)	55.6	55.6	56.7	.99
Neck length, mm (mean)	27.1	24.4	24.2	.96
Neck width, mm (mean)	24.6	24.7	24.2	.78
Renal Insufficiency (%)	4.9	2.9	15.0	.07
PVD (%)	5.8	2.9	10.2	.15
Smoking history (%)	47.1	51.4	55.9	.44
COPD (%)	23.5	17.1	25.1	.31
Coumadin* (%)	12.6	20.0	11.3	.28
Plavix† (%)	4.9	8.6	3.2	.13

AAA, Abdominal aortic aneurysm; PVD, peripheral vascular disease; COPD, chronic obstructive pulmonary disease.

*Bristol-Myers Squibb, Princeton, NJ.

†Sanofi-Aventis, Bridgewater, NJ.

RESULTS

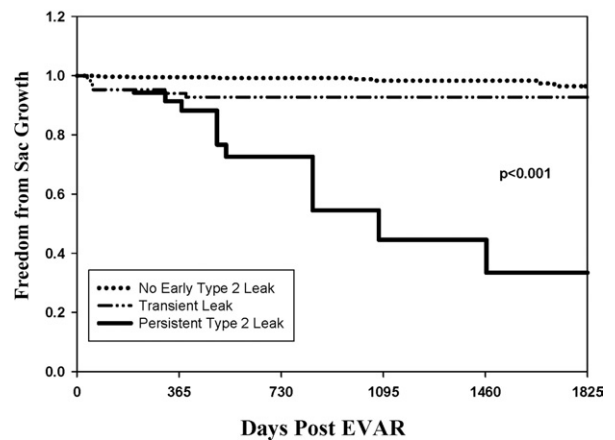
Overall results. An early type 2 endoleak was found in 164 (18.8%) of 873 patients. Of these, 22 (79.9%) early endoleaks spontaneously resolved ≤ 6 months without re-intervention, and 33 patients (20.1% of type 2 leaks; 3.8% of all EVARs) had persistent (>6 months) endoleaks. Only two (6.1%) of the 33 persistent endoleaks resolved spontaneously. The median follow-up for all patients was 28.7 months. For three patients with transient and persistent endoleak, the mean follow-up was 29.6 months and 44.1 months, respectively.

Preoperative patient characteristics and anatomic variables for patients with transient, persistent, and no early endoleaks are summarized in Table I. In addition, no statistically significant difference in number of preoperatively patent lumbar or inferior mesenteric artery vessels was found in patients with transient and persistent type 2 endoleaks ($P = .13$; relative risk [RR], 0.34; 95% confidence interval [CI] 0.22 to 0.52). Furthermore, no other significant differences in anatomic or preoperative variables were found between patients with transient, persistent, and no early endoleak.

Abdominal aortic aneurysm sac enlargement. AAA sac enlargement was noted in 5.9% of patients without an identifiable type 2 endoleak at the time of their first post-operative CT scan compared with 17.1% of patients with an early endoleak ($P < .001$). Eight patients with a transient endoleak experienced sac enlargement, and all of these occurred ≤ 1 year after EVAR. In contrast, 54.5% of patients (18/33) with a persistent endoleak demonstrated sac enlargement during the follow-up period.

As shown in Fig 1, the freedom from sac enlargement at 1, 3, and 5 years is 99.2%, 97.6%, and 94.9% for those without a type 2 endoleak vs 88.1%, 48.0%, and 28.0% for patients with a persistent endoleak ($P < .001$). The median time to aneurysm sac expansion >5 mm for patients with a persistent leak was 17.7 months (range, 4.7 to 76 months).

Freedom from Aneurysm Sac Growth



Persistent	Year-1	2	3	4	5
# at risk	33	28	12	8	6
SE	5.6	9.3	10.2	10.6	10.9
Transient	Year-1	2	3	4	5
# at risk	108	69	48	29	12
SE	2.6	3.0	3.0	3.0	3.0
No Leak	Year-1	2	3	4	5
# at risk	447	291	198	124	91
SE	0.5	0.6	1.1	1.1	2.2

Fig 1. Kaplan-Meier analysis of freedom from aneurysm sac growth.

As summarized in Table II, patients with persistent endoleak had a significantly higher rate of sac enlargement compared with those without early type 2 leaks (odds ratio [OR], 2.5; 95% CI, 1.7 to 3.8; $P = .01$).

Reinterventions. Patients with persistent type 2 endoleaks had a significantly higher reintervention rate than those without endoleak, as is summarized in Table II (RR,

Table II. Significance of persistent type 2 endoleaks on late outcomes after multivariate analysis

Outcome	RR	95% CI	P
Rupture	3.9	1.7-8.8	.04
Aneurysm sac growth	2.5	1.69-3.78	.01
Reintervention rate	5.5	3.5-8.8	.001
Conversion to open repair	5.3	2.0-13.5	.04
AAA-related death	2.6	0.77-8.8	.12

RR, Relative risk; CI, confidence interval; AAA, abdominal aortic aneurysm.

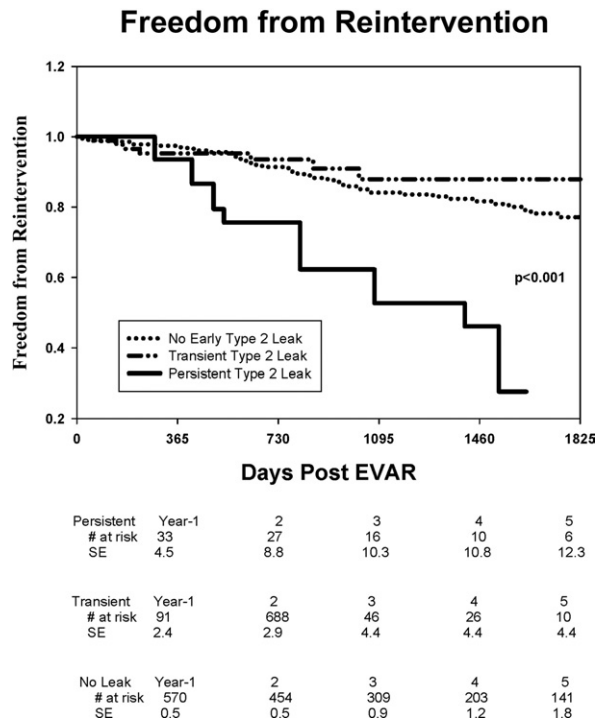


Fig 2. Kaplan-Meier analysis of freedom from reintervention.

5.5; 95% CI, 3.5 to 8.8; $P = .001$). Fig 2 represents a Kaplan-Meier analysis of the influence of persistent endoleak on the reintervention rate. The freedom from reintervention rate for patients without a type 2 endoleak at 1, 3, and 5 years was 97.5%, 84.5%, and 77.9% vs 93.4%, 51.7%, and 24.4% for patients with a persistent endoleak ($P < .001$).

Overall, 20 catheter-based interventions and one open reintervention were performed on 16 patients. Also performed were 13 transarterial embolizations, 5 translumbar embolizations, 2 graft revisions/stents, and 1 lumbar artery ligation. Nine (56.3%) of 16 patients eventually had successful treatment of their persistent endoleak.

Conversion to open repair. Patients with a persistent type 2 endoleak had a significantly higher rate of conversion to open repair compared with those without an early endoleak (RR, 5.3; 95% CI, 2.0 to 13.5; $P = .001$), as

Freedom from Conversion to Open Repair

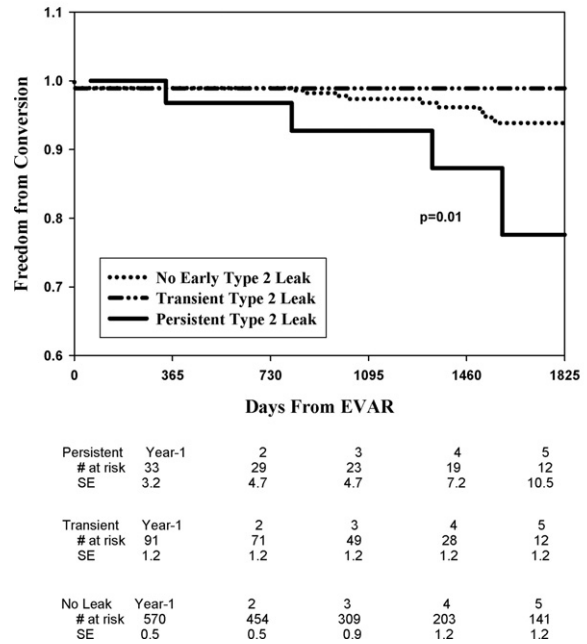


Fig 3. Kaplan-Meier analysis of freedom from conversion to open repair.

described in Table II. The median time to conversion to open repair in these patients was 20 months (mean, 24 months; range, 9 to 33 months). Figure 3 depicts a Kaplan-Meier analysis of freedom from conversion to open repair for patients with transient, persistent, and no type 2 endoleaks. The respective rate for freedom from conversion to open repair at 1, 3, and 5 years was 98.8%, 97.3%, and 93.7% for no endoleak patients vs 96.7%, 93.1%, and 78.6% for patients with persistent leak ($P = .01$).

Three of 33 patients with a persistent type 2 endoleak required a conversion to open repair. Two patients had failed prior transarterial embolizations in that the leak was evident after the procedure. However, the aneurysm sac size did not increase further after the interventions, so they were followed expectantly. The AAA in one of these patients subsequently ruptured. The other patient presented with rapid expansion (>5 mm during 6 months) and required conversion to open repair. The third patient was being monitored for a persistent endoleak without sac expansion. However, the patient presented with a significant increase in aneurysm sac size approximately 41 months after EVAR and an open repair was performed.

Aneurysm rupture. Aneurysm rupture occurred in four patients with an early type 2 endoleak. As summarized in Table II, patients with a persistent type 2 endoleak had a higher rate of rupture vs those without early endoleak (RR, 3.9; 95% CI, 1.7 to 8.8; $P = .03$). Fig 4 illustrates the Kaplan-Meier analysis for freedom from aneurysm rupture for patients with transient, persistent, and no early type 2 endoleaks. The freedom from rupture rates at 1, 3, and 5

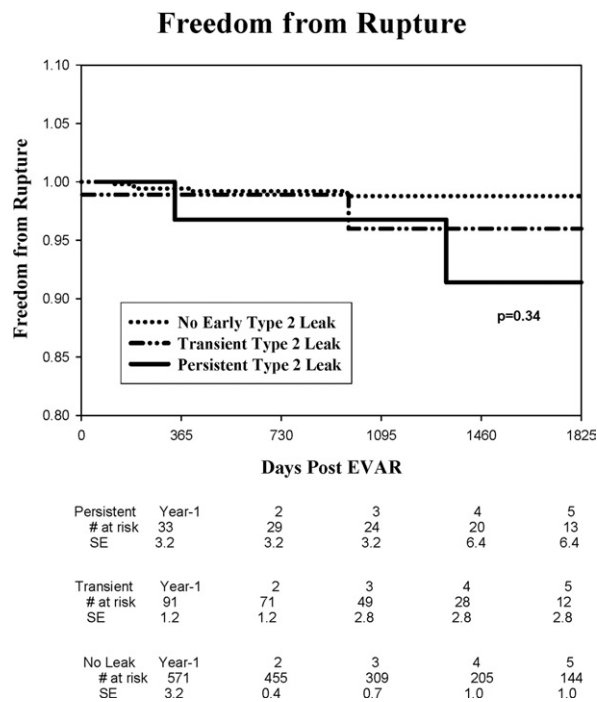


Fig 4. Kaplan-Meier analysis of freedom from rupture.

years for patients without an early type 2 endoleak was 99.8%, 98.5%, and 97.4% vs 98.7%, 96.2%, and 96.2% for patients with transient endoleaks, and 96.8%, 96.8%, and 91.1% for patients with persistent type 2 endoleaks. The median time to rupture for patients with early type 2 endoleaks was 31.6 months (range, 2 days to 37 months).

Two patients with early type 2 endoleaks experienced aneurysm rupture ≤ 6 months of EVAR placement. The first patient experienced back pain 2 days after EVAR with a AneuRx endograft, and a CT scan demonstrated a contained rupture. In the operating room, four lumbar arteries and a middle sacral artery were bleeding briskly. The endograft was replaced with a tube graft, but the patient eventually died of postoperative complications. The next patient had an 8-cm aneurysm that was repaired using a Zenith stent graft. A type 2 leak was noted on the postoperative CT scan. At 3 months after EVAR, a CT scan revealed a continuing type 2 endoleak, but the sac had decreased 2 mm in size. One month later, the patient presented with aneurysm rupture. In the operating room, several lumbar arteries were back-bleeding briskly, and these were oversewn. The aneurysm sac was closed and the endograft was left intact. The patient did well and was discharged 7 days later.

Persistent type 2 endoleaks also ruptured in two patients. One patient was being followed up for an endoleak that was not associated with sac enlargement. She presented to our hospital with back pain and a CT scan demonstrating a contained rupture. In the operating room, the aneurysm sac was opened, revealing two bleeding lumbar arteries. The proximal and distal attachment sites were

intact. The bleeding vessels were oversewn from within the sac, and the graft was left in place. The patient did well postoperatively and no further endoleaks have been noted on follow-up CT scans.

The second patient had an enlarging aneurysm sac associated with an endoleak after placement of a custom-made endograft. A transarterial embolization was unsuccessful. The patient had several comorbidities and did not want another attempt at embolization. Her aneurysm sac did not increase further, remaining 5 mm larger than her preoperative aneurysm size. The patient presented with aneurysm rupture 2.5 years after EVAR placement, with a type 2 endoleak on CT scan. The family chose not to intervene, and the patient died.

Aneurysm-related mortality. Although associated with several adverse outcomes, persistent type 2 endoleaks were not associated with an increase in aneurysm-related mortality compared with patients without a type 2 endoleak, as summarized in Table II (RR, 2.6; 95% CI, 0.77 to 8.8; $P = .12$).

DISCUSSION

The clinical impact of type 2 endoleak after EVAR is not well established and remains controversial.^{19,20} Our data suggest that persistent type 2 endoleaks are significant contributors to late adverse outcomes after EVAR. Events such as aneurysm rupture, conversion to open repair, aneurysm sac growth, and the need for reintervention were significantly increased in patients with persistent endoleak in our large series. The small sample size may have limited this study's ability to evaluate the impact of endoleak on AAA-related mortality. The incidence of type 2 endoleak varies between 6% and 30% in large series of EVAR patients.^{7,11} The European Collaborators on Stent-Graft Techniques for AAA and Thoracic Aortic Aneurysm and Dissection Repair (EUROSTAR) database reports a 9% incidence of early (at first postoperative CT scan) type 2 endoleak.⁸ Zarins et al²¹ reported a 20% incidence of endoleak at 1 year with the AneuRx device, with 72% of leaks being type 2 endoleaks. Our overall rate of type 2 endoleak was 18.8%, with spontaneous resolution occurring ≤ 6 months in 79.9% of patients. This rate of resolution is higher than found in other published reports. For example, Sheehan et al²² recently evaluated the effect of endograft brand on the incidence of type 2 endoleak in 1909 patients and reported a spontaneous resolution at 6 months of only 33%. However, their initial postoperative CT scan was performed at 1 month after EVAR, whereas we often perform postimplant CT scans at 1 day after EVAR, before discharge. Thus, we may be identifying a large number of endoleaks that would have resolved by the 1-month time point used in the Sheehan study. This variation may falsely elevate our spontaneous resolution rate ≤ 6 months after EVAR.

Published reports demonstrate that spontaneous resolution of endoleaks persisting > 6 months occurs in 5% to 33% of patients.^{22,23} Only 6.1% of our persistent endoleaks resolved spontaneously. Similar to our results, Parent et

al²³ demonstrated no spontaneous resolution of type 2 endoleaks after 12 months.

Evaluation of the natural history of persistent endoleaks is difficult because of the varying management strategies used to address these leaks. From the EUROSTAR database, van Marrewijk et al⁸ found type 2 endoleak to be significantly associated with aneurysm sac growth over time. Kaplan-Meier analysis revealed that 24% of patients with type 2 endoleak had sac enlargement at 48 months compared with 13% without endoleak. At 3 years, 52% of our patients with persistent endoleak had sac enlargement. However, aneurysm sac growth in the EUROSTAR series was defined as enlargement >8 mm vs >5 mm in our report. Our higher incidence of sac growth may be explained by this criteria difference. Moreover, patients with type 2 endoleaks in the EUROSTAR report were not divided into transient or persistent. Combining both groups could mask the clinical impact of persistent endoleaks.

The EUROSTAR series also reported a significantly higher rate of reintervention required by patients with a type 2 endoleak ($P = .001$). Our data support these findings. However, the EUROSTAR series did not find an association between type 2 endoleak and conversion to open repair or rupture, which our study demonstrated.

Other groups have demonstrated adverse events related to type 2 endoleak.²⁰ Timaran et al²⁴ specifically evaluated their experience with persistent type 2 endoleaks in 32 (9.2%) of 348 patients, and 13 (41%) exhibited aneurysm sac enlargement, occurring at a mean of 9.7 months after EVAR (mean follow-up, 31 months). In our series, 54.5% of persistent endoleaks were associated with sac enlargement, but the mean time to aneurysm growth was much greater (21 months post-EVAR).

It is unclear why our mean time to aneurysm growth was significantly longer than the Timaran group. However, in their cohort of persistent endoleaks with stable or shrinking sac size, their mean follow-up was only 25 months. Perhaps they missed the patients with late sac growth that we observed in our series. One patient in the Timaran series died of a confirmed aneurysm rupture. The 5-year actuarial freedom from rupture for patients with a persistent endoleak in the Timaran series was 92%, which is similar to our rate of 91.1%. Timaran et al also demonstrated a high rate of conversion to open repair in these patients of 75% at 5 years, which mirrors our experience of 78.6% at 5 years based on Kaplan-Meier analysis.

Although several groups have identified adverse late outcomes associated with type 2 endoleaks, other reports challenge this assertion. Arko et al²⁵ published their experience with 46 consecutive patients undergoing EVAR, 16 of whom were identified as having persistent endoleaks. Persistent endoleak had no significant effect on proximal neck morphology or migration. Although they demonstrated a significant effect on change in sac size compared with no endoleak, the mean increase in sac size for persistent endoleaks was only 1.8 mm. No association between persistent endoleak and either rupture or conversion to

open repair was identified. Thus, these authors concluded from their short follow-up of 19.7 months that persistent type 2 endoleak did not pose a significant risk to patients.

In a review of the current literature, there are only five reports of rupture secondary to type 2 endoleak.^{24,26,27} Harris et al²⁸ described nine cases of rupture related to type 2 endoleaks from the EUROSTAR database; however, eight of these ruptures were attributed to concomitant type 1 endoleaks. We report four cases of aneurysm rupture associated with type 2 endoleak. Two patients had persistent leaks, and two were not associated with aneurysm sac growth before rupture. The most plausible explanation for the latter two patients is that they experienced a sudden, rapid sac expansion leading to rupture that occurred in the interval between CT scans.

The relationship between patent collateral flow and the risk of aneurysm growth and rupture has been described. In a series from the Albany Medical Center group,²⁹ 1218 AAAs were repaired using an exclusion and bypass open technique. Of these, 64% of patients with persistent sac flow demonstrated sac enlargement, and 26% ruptured.²⁹ The applicability of this study to EVAR and endoleaks has been questioned, but our data related to sac enlargement (and ongoing potential rupture risk) are similar.

Several treatment options are available for the management of type 2 endoleaks. Transarterial chemical or coil embolization techniques, as well as translumbar sac embolization, have been described.³⁰ Laparoscopic or open ligation of feeding vessels have also been advocated as potential options.³¹ The success of these techniques varies widely in the literature, however.

Baum et al³² compared transarterial coil embolization with translumbar embolization. In the transarterial group, 16 of 20 patients failed treatment (80% failure rate), but 12 (92%) of 13 patients were successfully treated with translumbar embolization.³² Timaran et al²⁴ had similar results in catheter-based treatment of persistent endoleaks. A transfemoral approach achieved only a 38% success rate, although a 71% success rate was noted with a translumbar approach.²⁴ In our series, we used both a transarterial and translumbar approach to the treatment of type 2 endoleaks and achieved success in 60% of patients. The number of techniques and varying success rates of reintervention reflects the difficulty of treating type 2 endoleaks after EVAR. As more experience is gained, a consensus on the best treatment option may emerge.

Our data and others' suggest that the natural history of untreated persistent type 2 endoleaks is not necessarily benign. Our mean follow-up of 43 months in patients with persistent endoleaks allowed us to demonstrate that the risk of aneurysm growth and its associated adverse events continue over an extended follow-up period. In addition, to our knowledge, our report is the first to demonstrate a significant relationship between persistent type 2 endoleaks and aneurysm rupture.

We did not identify any preoperative factors or anatomic variables predictive of persistent endoleak; however, we did identify a trend towards increased persistent type 2

endoleaks in patients who were taking Coumadin (Bristol-Myers Squibb, Princeton, NJ) or Plavix (Sanofi-Aventis, Bridgewater, NJ). Our small sample size prevents us from determining whether anticoagulants significantly contribute to persistent endoleaks.

Previous work from our institution has demonstrated that the preoperative number of patent lumbar vessels significantly correlates with the presence of early type 2 endoleaks.³³ Zero to three patent lumbar arteries was associated with a 13% type 2 endoleak rate, whereas 50% of patients with six or more patent lumbar arteries had a type 2 endoleak. Similarly, inferior mesenteric artery patency was associated with an increase in early endoleak. In this study, we did not find a significant difference in number of preoperatively patent lumbar vessels between patients with transient and persistent endoleaks.

Our study is limited by its retrospective nature and the relatively small number of adverse events. The relatively low number of patients with persistent endoleaks made Kaplan-Meier analysis less robust when evaluating late outcomes >3 years. Furthermore, although multivariate analysis demonstrated persistent endoleak as a significant predictor of rupture in our series, the Kaplan-Meier *P* value did not reach a level of significance. This is likely due to the large variation in time to rupture in the persistent (591 days) vs no-leak groups (1175 days), which can skew Kaplan-Meier log-rank test.

CONCLUSION

Our data demonstrate the negative impact of type 2 endoleaks on patients undergoing EVAR. Patients with a persistent endoleak are at a significantly increased risk of aneurysm rupture, sac growth, conversion to open repair, and reintervention compared with patients without endoleak. To prevent these adverse outcomes, a more aggressive approach to management of persistent type 2 endoleak may be warranted.

AUTHOR CONTRIBUTIONS

Conception and design: JJ, DB, RC, GL

Analysis and interpretation: JJ, TC, CK, GL

Data collection: JJ

Writing the article: JJ, DB

Critical revision of the article: JJ, DB, RC, GL

Final approval of the article: JJ, DB, RC, CK

Statistical analysis: JJ, TC

Obtained funding: Not applicable

Overall responsibility: JJ, DB, RC

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