An 8-year experience with type II endoleaks: Natural history suggests selective intervention is a safe approach

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Objective: **The treatment of type II endoleaks remains controversial because little is known about their long-term natural history and impact on changes in aneurysm morphology. This study reviews type II endoleaks occurring in patients after endovascular abdominal aortic aneurysm repair (EVAR) at a single-institution over an 8-year period.**

Methods: **All patients undergoing EVAR who had type II endoleaks documented on follow-up imaging studies at our institution between January 1997 and March 2005 were reviewed. Data regarding patient demographics in addition to aneurysm size, device type, operative complications, and secondary interventions were reviewed. Outcomes evaluated included the rate of spontaneous sealing, freedom from secondary intervention, and aneurysm enlargement, rupture, or conversion.**

Results: **Type II endoleaks were present in 154 of 965 patients (16.0%) undergoing EVAR. Mean follow-up time was 22.0 months (range, 1 to 72 months). Fifty-five patients (35.7%) with type II endoleaks sealed spontaneously in a mean time of 14.5 months. According to Kaplan-Meier analysis, approximately 75% of type II endoleaks sealed spontaneously within a 5-year period. Nineteen patients (12.3%) with type II endoleaks were treated at a mean time of 19.9 months at the operating surgeon's discretion, including 13 with sac enlargement >5 mm. Kaplan-Meier analysis estimated that approximately 65% of the patients remained free of intervention after a period of 4 years. Thirteen patients (8.4%) experienced aneurysm sac enlargement >5 mm. Kaplan-Meier analysis estimated that approximately 80% of patients with type II endoleaks remained free of sac enlargement >5 mm over a 4-year period. No patients with type II endoleaks experienced rupture or required conversion to open repair during their follow-up. Cox regression analysis showed that cancer, coronary artery disease, and chronic obstructive pulmonary disease were associated with earlier spontaneous closure of the type II endoleaks ($P < .05$).**

Conclusions: **We observed that type II endoleaks have a relatively benign course, and in the absence of sac expansion, can be followed for a prolonged course of time without the need for intervention. The rate of spontaneous seal continues to increase with time and, therefore, close follow-up of patients with type II endoleaks who show no signs of aneurysm expansion is a safe approach. For patients in whom the exact etiology of their endoleak is in question, dynamic imaging should be used to exclude the presence of a type I endoleak. (J Vasc Surg 2006;44:453-59.)**

Endovascular aneurysm repair (EVAR) has offered a minimally invasive alternative to open abdominal aortic aneurysm (AAA) repair since its initial description by Parodi et al in 1991.[1] EVAR is based on the same principles as the traditional open AAA repair, which is complete exclusion of arterial perfusion from the aneurysm sac with resultant prevention of aneurysm rupture. Mid-term results using this technique with second-generation and third-generation devices have been excellent.[2] The long-term durability of EVAR continues to be questioned, however, largely because of endoleak, which is the persistence of blood flow outside the graft and within the aneurysm sac.[3-5]

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There is little debate regarding the treatment of type I and type III endoleaks, which involves stent-graft attachment sites or junction points between graft components, respectively. Type I and type III endoleaks signify incomplete exclusion of the aneurysm sac from systemic arterial pressure and, therefore, incomplete aneurysm repair and should be treated promptly. The treatment of type II endoleaks, which are related to retrograde flow into the aneurysm sac via patent collaterals, most commonly lumbar arteries or the inferior mesenteric artery (IMA), remains controversial.

The long-term natural history of type II endoleaks has not yet been fully elucidated. Several reports have related aneurysm rupture to type II endoleaks.[6,7] Additional reports have shown that type II endoleaks may produce systemic arterial pressures within the aneurysm sac, increasing the risk of rupture.[8,9] Conversely, other studies have shown type II endoleaks not to be a risk factor for rupture after EVAR.[10-12] Given this conflicting data, some have advocated immediate treatment, whereas others limit treatment to only those aneurysms with significant increases in size.[13,14]

To gain a better understanding of the natural history of type II endoleaks, we reviewed our 8-year experience with

a variety of different endovascular devices for the treatment of AAAs to determine the incidence and outcomes of type II endoleaks.

METHODS

A retrospective review was performed for all patients undergoing EVAR at our institution between January 1997 and March 2005. Patients gave written informed consent and were treated in accordance with the approval of the institutional review board of the Mount Sinai Medical Center. All patients underwent preoperative contrast-enhanced computed tomography (CT) and arteriography to evaluate AAA anatomy. Patients were prospectively monitored, and data on demographics, aneurysm size, medical comorbidities, complications, and secondary interventions were obtained. Preoperative, perioperative, and postoperative follow-up data were reviewed from these medical records and archived radiology studies.

Follow-up for all patients undergoing EVAR consisted of an office visit with the operating surgeon as well as plain abdominal radiography and three-phase contrast-enhanced computed tomography angiography (CTA) at 1 month, 6 months, 12 months, and annually thereafter. CTA consisted of a noncontrast study to assess for calcium in the sac followed by dynamic and late-phase angiographic assessment of the abdominal aorta. Helical images were obtained from the diaphragm through the femoral heads before, during, and after the intravenous bolus administration of nonionic contrast material at a rate of 4 mL/s for a total volume of 140 mL. Magnetic resonance angiography (MRA) or duplex ultrasonography was substituted for CTA in patients with renal insufficiency. Patients who demonstrated endoleaks underwent more frequent surveillance imaging and follow-up.

Patients who were selected for treatment of their endoleak underwent transfemoral arteriography with selective injection of the hypogastric arteries and superior mesenteric artery to evaluate lumbar and inferior mesenteric artery components of their endoleak before embolization and to exclude the presence of type I or type III endoleaks. Incidence of endoleak type II was calculated for the entire cohort of patients. The study population consisted of all patients with a type II endoleak. Patients who underwent preoperative interventions (IMA or internal iliac artery embolization) and went on to develop a type II endoleak were included in this study. Preoperative embolization of the IMA was performed in 12 of the patients with type II endoleaks, and preoperative embolization of a single internal iliac artery was performed in 31 patients.

The follow-up period was defined as the time from endograft implantation to the most recent imaging study. The time until endoleak seal was calculated from the date of surgery until the date of imaging when the endoleak was no longer present. The time until endoleak treatment was calculated from the date of surgery until the date when the endoleak was treated.

Statistical analysis was performed using the statistical software package SPSS 13.0 (SPSS Inc, Chicago, Ill) and GraphPad Prism (GraphPad Software, Inc, San Diego, Calif). Kaplan-Meier tables were used to estimate the rate of spontaneous seal, aneurysm sac enlargement, and freedom from intervention in patients with type II endoleaks. When rates of sealing were calculated, patients who were treated for their type II endoleaks were considered as "failure to spontaneously seal," and were censored at the time of intervention. The incidence of type II endoleak development related to device type was examined using χ^2 analysis. The association between different comorbidities and seal rate was examined using the Student's t test for continuous variables and χ^2 for categoric variables. Multivariate analysis was done using Cox regression.

Table I. Characteristics and comorbidities of patients with type II endoleaks

	N	*%*
Gender		
Male	133	86.4
Female	21	13.6
Comorbidities		
CAD	75	48.7
Hypertension	111	72.0
Hypercholesterolemia	73	47.4
COPD	33	21.4
Diabetes mellitus	18	12.0
Malignancies	27	17.5

CAD, Coronary artery disease; *COPD,* chronic obstructive pulmonary disease.

RESULTS

During a period of 8 years, 965 patients underwent EVAR at our institution. Endovascular stent-grafts used for the initial AAA repair that developed type II endoleaks were the Talent (Medtronic World Medical, Sunrise, Fla), the Excluder (W. L. Gore & Assoc, Flagstaff, Ariz), the AneuRx (Medtronic/AVE, Santa Rosa, Calif), the Ancure (Guidant, Menlo Park, Calif), the Fortron (Cordis/Johnson & Johnson, Warren, NJ), the Teramed (Teramed, Minneapolis, Minn), and the Zenith (Cook, Bloomington, Ind). Of these, 154 were found to have type II endoleaks documented at the time of their initial follow-up visit or at any time thereafter.

The configurations of these grafts were 137 bifurcated, 12 aortouniiliac, and five aortoaortic. The overall type II endoleak rate during this period was 16.0% (154/965); however, this rate should be evaluated with the knowledge that several of our patients were treated preoperatively with coil embolization of the IMA or internal iliac arteries during the period reviewed. Device-specific rates of type II endoleak development during this time were Talent, 17.6% (120/683); Excluder, 15.0% (12/80); AneuRx, 9.2% (9/99); Ancure, 44.4% (4/9); Fortron, 9.3% (4/43); Teramed, 40% (4/10); and Zenith, 14.3% (1/7). Despite the variance in occurrence rates, the difference between devices with regard to type II endoleak development ($P = .52$) was not statistically significant. Mean patient age was 76.1 years

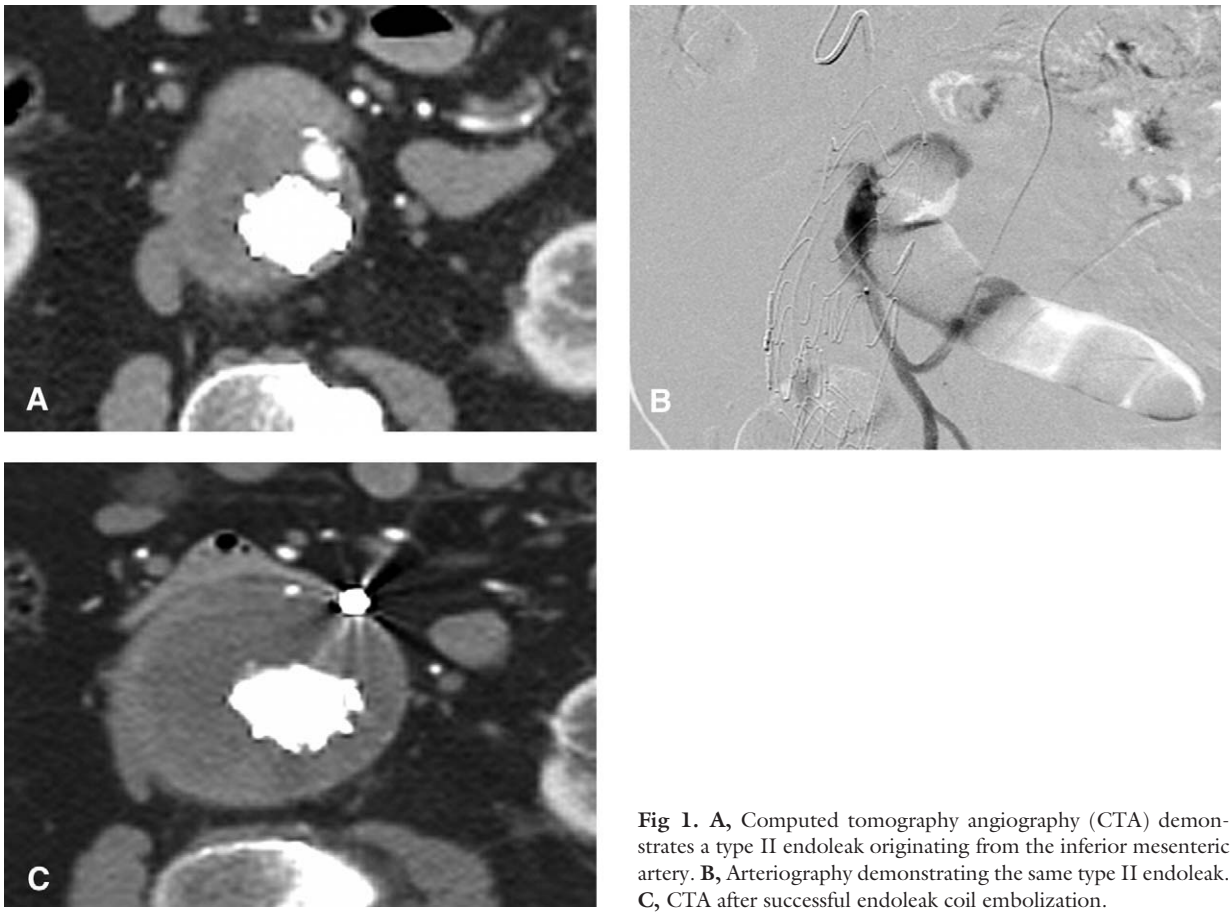

Fig 1. A, Computed tomography angiography (CTA) demonstrates a type II endoleak originating from the inferior mesenteric artery. **B,** Arteriography demonstrating the same type II endoleak. **C,** CTA after successful endoleak coil embolization.

(range, 44 to 95 years), and 133 were men and 21 were women. Patient demographics and comorbidities are documented in Table I. Mean initial sac size was 59.8 mm (range, 31 to 110 mm). The mean follow-up time was 22.0 months (range, 1 to 72 months).

Of the 154 patients reviewed, 55 (35.7%) had type II endoleaks that sealed spontaneously. The mean time until spontaneous seal was 14.5 months (range, 0.3 to 36.3 months). During the study period, 19 patients were treated for type II endoleaks with coil embolization via either transfemoral or translumbar approaches, in select cases. Mean time until treatment was 19.9 months (range, 0.2 to 51.6 months). Of the 19 patients (12.3%) who underwent treatment, 13 had aneurysm sac enlargement >5 mm. The remaining six patients were treated at the discretion of the operating surgeon, but on review, were all noted to have aneurysm enlargement although it failed to reach 5 mm in this subgroup. Three patients required two separate treatments for endoleaks originating from different anatomic locations. A persistent endoleak after treatment developed in eight patients. Type II endoleaks developed from the lumbar arteries in all of the patients in our series who underwent preoperative IMA coil embolization, but none has required secondary interventions.

The mean change in aneurysm size among all the patients in the study was 0.09 mm (range, –37 to 48 mm). The mean change in aneurysm size among patients who were treated was 6.42 mm (range, –6 to 26 mm) from the time preoperatively until their last follow-up. Mean change in aneurysm size among untreated patients was –1.23 mm (range, –37 to 17 mm). Mean change in aneurysm size among the subgroup of patients who underwent preoperative IMA embolization was –1.75 mm (range, –19 to 5 mm). As previously noted, 13 patients experienced sac enlargement >5 mm and all of these patients underwent treatment for their type II endoleaks. (Fig 1). In four patients, aneurysm enlargement continued after interventions. No patients with type II endoleaks experienced rupture or required conversion to a traditional open repair during their follow-up.

The Kaplan-Meier survival curve estimated that approximately 75% of type II endoleaks sealed spontaneously ≤5 years when observed without intervention (Fig 2). Kaplan-Meier analysis estimated that approximately 80% of patients with type II endoleaks remained free of sac enlargement greater than >5mm during a 4 year-period (Fig 3) and that approximately 65% of the patients remained free of intervention after a 4-year period (Fig 4).

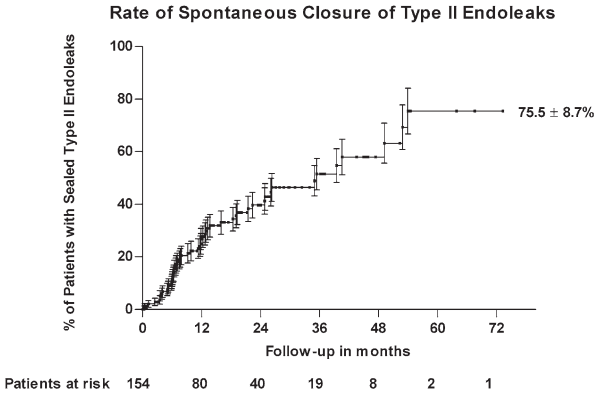

Fig 2. Kaplan-Meier analysis demonstrates the rate of spontaneous closure of type II endoleaks.

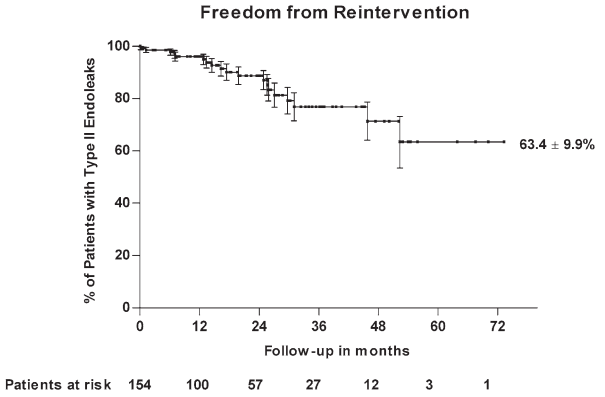

Fig 4. Kaplan-Meier analysis demonstrates freedom from reintervention.

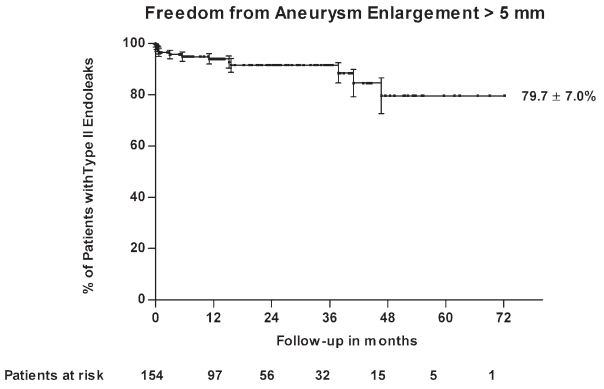

Fig 3. Kaplan-Meier analysis demonstrates freedom from aneurysm enlargement.

Association between age, initial sac size, gender, comorbidities, and spontaneous seal of type II endoleaks is summarized in Table II. A higher rate of spontaneous sealing was found in patients with cancer, coronary artery disease (CAD), and chronic obstructive pulmonary disease (COPD) (Fig 5).

DISCUSSION

The incidence of type II endoleaks after EVAR has been reported to be 8% to 45%.[14-18] The natural history of type II endoleaks is of great importance to physicians and patients both because the presence of a type II endoleak after EVAR frequently leads to reintervention. Only limited information about the natural history of type II endoleaks is available, however, because of the variability of surgeons' decisions to intervene, patient follow-up, and patient life expectancies, along with the constantly evolving technology of devices used for EVAR and imaging techniques used for follow-up. Because much of the natural history of type II endoleaks is not yet known, the exact clinical significance of these endoleaks is also unknown.

There is general consensus in the literature on the management of type I and type III endoleaks; however, the significance and management of type II endoleaks remains controversial. Several authors have reported a benign course of these endoleaks.[10,11,19] Intrasac pressure has been shown to decrease over time in the presence of type II endoleaks, although to a lesser degree than those patients without endoleaks.[20] Conversely, ruptured AAAs with documented type II endoleaks have been reported.[6,7] Furthermore, some studies have shown type II endoleaks to be associated with aneurysm growth,[21,22] but others have demonstrated no change in aneurysm volume.[23]

Given these reports and the premise that a persistent endoleak may cause continuous pressurization of the aneurysm sac and ultimately lead to sac expansion and possible rupture, some authors have supported a more aggressive approach to obliterating type II endoleaks.[8,24] On the other hand, a more conservative approach in which type II endoleaks are observed over time and treated only if sac expansion occurs has been reported to be safe as well as cost-effective.[13]

The natural history and the rate of spontaneous sealing of type II endoleaks over time have not yet been fully elucidated. Steinmetz et al[13] reported that 35 (38%) of 90 patients with type II endoleaks had endoleaks that persisted ≥6 months.[13] In other words, the endoleaks sealed spontaneously within the first 6 months in 62% of their patients with type II endoleaks. Parent et al[25] reported their experience in patients with type II endoleaks and determined that in 13 (36%) of 36 patients, the endoleaks underwent spontaneous sealing by 6.2 months.

Given the conflicting data on the management and natural history of type II endoleaks, it is unclear what interventions should be performed in the preoperative management of patients with AAAs who are found to have patent IMAs or lumbar vessels. Several authors have noted that a patent IMA or lumbar artery preoperatively is a risk factor for developing a type II endoleak after EVAR.[23,26] Thus, preoperative embolization of either the IMA or lumbar arteries has been studied in relation to type II endoleaks. Our own institution has reviewed its experience with preoperative embolization of the IMAs.[27] Thirty pa-

Table II. Influence of factors on spontaneous seal of type II endoleaks

	No seal	*Seal*	*Univariate* P	*Multivariate* P
Mean age (years)	76.9	74.9	.14	N/A
Initial sac size (mm)	60.2	59.2	.62	N/A
Sex: patients, n (%)			.81	N/A
Male	86 (86)	47 (83)		
Female	13 (14)	8 (17)		
Cancer, n (%)			.05	.004
Yes	13 (14)	14 (25)		
No	86 (86)	41(75)		
CAD, n (%)			.08	.013
Yes	43 (43)	32 (58)		
No	56 (57)	23 (42)		
COPD, n (%)			.01	0
Yes	15 (15)	18 (32)		
No	84 (85)	37 (68)		
Diabetes mellitus, n (%)			.81	.46
Yes	13 (14)	5 (9)		
No	86 (86)	50 (91)		
Hypercholesterolemia, n (%)			.1	.152
Yes	42 (42)	31 (56)		
No	57 (58)	24 (44)		
Hypertension, n (%)			.17	.922
Yes	75 (75)	36 (65)		
No	24 (25)	19 (35)		

CAD, Coronary artery disease; *COPD,* chronic obstructive pulmonary disease.

tients with patent IMAs underwent successful embolization, and at 6 months, this group had a type II endoleak rate of 17%. In comparison, 54 patients who had patent IMAs preoperatively and no embolization demonstrated a type II endoleak rate of 48% at 6 months. Additional reports have shown similar decreases in the rate of type II endoleaks with preoperative embolization.[22,28]

With still little known about the long-term natural history of type II endoleaks, these may not be necessary procedures. At present, our approach has been to preoperatively embolize IMAs only in patients with large-caliber IMAs in whom the IMA is easily cannulated and the procedure is easily performed. Although many of these may spontaneously seal, for those that do not, preoperative treatment avoids the riskier, more technically challenging procedure of postoperative embolization.

Our institution has adapted a less aggressive approach to the management of type II endoleaks. Persistent type II endoleaks are not routinely embolized unless sac expansion has been documented. This has allowed us to observe the rate of spontaneous seal over a prolonged period of time. In our patients with type II endoleaks, the rate of spontaneous closure continues to climb over the entire period of the follow-up and has not been associated with an increased risk of sac expansion or other adverse outcome over the same time period. Furthermore, there were no documented ruptures associated with type II endoleaks monitored over this time period. The reintervention rate was similar to other reports, indicating that this approach did not result in a greater need for late interventions.

Although our institution has adapted a less aggressive approach to interventions, we have been liberal with our use of dynamic studies to rule out the possibility of a type I or even a type III endoleak. It is not unusual for patients to initially be diagnosed with a type II endoleak on CTA but ultimately determined to have a type I or type III endoleak as determined by arteriography or cine MRA. These modalities are invaluable to definitively locate the source of the endoleak.

Endoleaks have a dynamic natural history and may have changing branch vessel involvement and flow patterns.[25] We noted several patients in our review who were initially thought to have type II endoleaks but were found to have type I endoleaks by dynamic studies. It is difficult to know whether these patients had type I endoleaks over the course of their follow-up that were misinterpreted as type II endoleaks on their studies or whether there was some degeneration to a type I endoleak. Regardless of the mechanism, patients should be evaluated promptly with further imaging whenever there is a question of a type I endoleak.

We found an association between cancer, CAD, COPD, and spontaneous closure of endoleaks. The association with COPD and spontaneous closure of type II endoleaks has been documented previously.[14] Why COPD would increase seal rate is unclear. We found no significant difference in hematocrit levels between COPD and non-COPD patients, but it is possible that blood viscosity is higher in smokers, thus contributing to earlier thrombosis of the collateral flow into the sac. In addition, the higher rate of spontaneous closure of type II endoleaks in the presence of malignancy might be explained by the prothrombotic tendencies of patients with malignancies. Although there is no means of proving this, it has been shown that, conversely, type II endoleaks may be less likely to

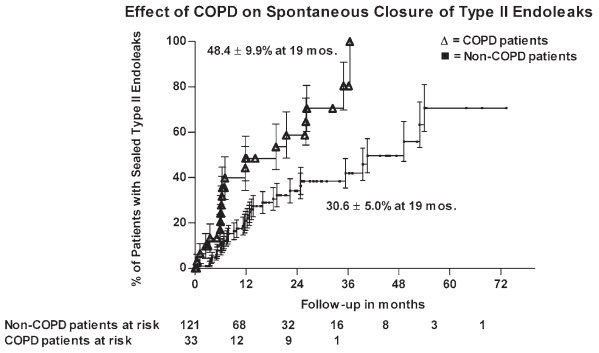

Fig 5. Kaplan-Meier analysis demonstrates effect of chronic obstructive pulmonary disease *(COPD)* on closure of type II endoleaks.

undergo spontaneous thrombosis in patients on warfarin therapy.[29]

Our study is limited in part by its retrospective nature. We determined the time of endoleak seal based on the first follow-up study in which the leak was absent. It is quite reasonable to assume the endoleak had sealed at any point in time before that study. In addition, a few patients with type II endoleaks without evidence of aneurysm enlargement were treated early in their course at the operating surgeon's preference. Furthermore, preoperative inferior mesenteric artery embolization was performed more frequently later in the time period of this study. It should also be noted that almost all of our patients had a Talent device placed. It appears, however, from our own data and that of the largest series examining the role of endograft type on the development of type II endoleaks[30] that the incidence does not appear to be related to device type. Despite these limitations, this study represents one of the largest series of patients with type II endoleaks who have been observed over a significant period of time.

CONCLUSION

We have observed that type II endoleaks have a relatively benign course and, in the absence of sac expansion, can be monitored for a prolonged course of time without the need for intervention. We recommend that close follow-up of patients with type II endoleaks who show no signs of aneurysm expansion is a safe approach, thus sparing the patients from unnecessary secondary procedures and the inherent risk of complications from these interventions. However, for patients in whom there is a question of the exact etiology of their endoleak, dynamic imaging in the form of arteriography or cine MRA should be used to exclude the presence of a type I endoleak.

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AUTHOR CONTRIBUTIONS

Conception and design: DS, DB, SE, AC, MM
Analysis and interpretation: DS, DB, SE, RL
Data collection: DS, DB, SG
Writing the article: DS, DB, SE
Critical revision of the article: DS, DB, SE, AC, RL, MM
Final approval of the article: DS, DB, SE, SG, AC, RL, MM
Statistical analysis: DS, DB, SE
Obtained funding: MM
Overall responsibility: DS

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INVITED COMMENTARY

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Endoleak, the Achilles heel of endovascular aortic aneurysm repair (EVAR), is correlated with aneurysm sac expansion, the need for conversion, aneurysm rupture, and death. The presence of endoleak is the most common reason for readmission to the hospital after EVAR and increases the secondary procedure rate, cost, and length of stay.

Collateral vessel endoleaks (type II), the most prevalent form of endoleaks, do not behave as a uniform class even though they share a common etiology—back bleeding from an aortic branch. As Silverberg et al and others have demonstrated, some of these leaks will spontaneously thrombose, and some will persist. Some will transmit systemic pressure to the aneurysm sac and to lead to rupture of the aneurysm, whereas in other cases, the aneurysm sac regresses and the patients seem to be protected from aneurysm rupture despite the presence of an endoleak. Unlike type I and type III endoleaks, which mandate repair upon their discovery, there is no clear consensus on how type II endoleaks should best be treated, or even monitored.

Why are type II endoleaks so different and unpredictable in their behavior? Vascular surgeons readily understand that type II endoleaks could not remain patent if only supplied by a single vessel, because end arteries with no outflow rapidly thrombose. This explains why most type II endoleaks that are detected immediately postoperatively have disappeared by the time of the first postoperative computed tomography (CT) scan. For a branch vessel endoleak to remain patent, it must have both inflow and outflow. Duplex Doppler ultrasound criteria, which have been found to be predictive of type II endoleak thrombosis, include a high-resistance type of flow, whereas what is most predictive of continuation of the endoleak is a low-resistance, continuous type flow, indicative of a patent outflow tract. The flow patterns in these endoleaks are variable, changing with blood pressure, position, respiration, and other dynamic factors.

This changing flow pattern presents a great challenge to current imaging modalities and explains the frequent observation of "intermittent appearance" of a type II endoleak. CT angiography (CTA) has been shown repeatedly to lack specificity for determination of leak type and vessel origin of type II leaks compared with arteriography or direct sac injection. Injection of the sac in patients who have been found to have a type II endoleak often reveals multiple pairs of lumbar arteries in communication with each other as well as with other branch vessels, when only a single vessel was suspected by the screening CTA images.

The emerging understanding of these endoleaks is a picture analogous to our view of arteriovenous malformations. They are associated with multiple vessels, which may serve as inflow or outflow, depending on the prevailing physiologic state at the moment. The involved vessels share a nidus of communication that maintains the patency of the type II endoleak.

Treatment of type II endoleaks should focus on disruption of this nidus, resulting in end arteries without the possibility of outflow, leading to thrombosis. For this reason, it is no wonder that type II endoleaks related to patent lumbar arteries developed in all 12 (100%) of the patients Silverberg et al treated by means of preoperative inferior mesenteric artery (IMA) embolization! Their prophylactic strategy addressed individual feeding arteries rather than the root cause of the endoleak, which is the communication between multiple arteries. If a prophylactic approach to type II endoleak is to be developed, the greatest likelihood for success would likely be to focus on obliteration of the sac and the paths between branch vessels.

Silverberg et al have taken a mostly selective approach to intervention for type II endoleaks, with the notable exception of the previously mentioned preoperative IMA embolizations, generally reserving intervention for patients who demonstrate significant sac expansion. They have shown that in the short term (22 months' mean follow-up), this approach has not resulted in any aneurysm ruptures. Because they did not apply any standard protocol to the treatment of type II endoleak, and in the absence of a control group for comparison, it is difficult to determine if the behavior of their endoleak patients is different from that of EVAR patients in general or even from the natural history of untreated aneurysms over a 22-month mean follow-up interval. No clear treatment recommendations emerge.

What is clear is that our understanding of type II endoleak and its treatment is evolving. Patients remain at risk for development of endoleaks at all times after EVAR. This is an absolute mandate for continued, careful surveillance of patients who have undergone EVAR.