Long-term sac behavior after endovascular abdominal aortic aneurysm repair with the Excluder low-permeability endoprosthesis

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Purpose: **Sac regression is a surrogate marker for clinical success in endovascular aneurysm repair (EVAR) and has been shown to be device-specific. The low porosity Excluder endograft (Excluder low-permeability endoprosthesis [ELPE]; W. L. Gore & Associates Inc, Flagstaff, Ariz) introduced in 2004 was reported in early follow-up to be associated with sac regression rates similar to other endografts, unlike the original Excluder which suffered from sac growth secondary to fluid accumulation in the sac. The purpose of this study was to determine whether this behavior is durable in mid-term to long-term follow-up.**

Methods: **Between July 2004 and December 2007, 301 patients underwent EVAR of an abdominal aortic aneurysm (AAA) with the ELPE at two institutions. Baseline sac size was measured by computed tomography (CT) scan at 1 month after repair. Follow-up beyond 1 year was either with a CT or ultrasound scan. Changes in sac size ≥5 mm from baseline were determined to be significant. Endoleak history was assessed with respect to sac behavior using χ^2 and logistic regression analysis.**

Results: **Two hundred sixteen patients (mean age 73.6 years and 76% men) had at least 1-year follow-up imaging available for analysis. Mean follow-up was 2.6 years (range, 1-5 years). The average minor-axis diameter was 52 mm at baseline. The proportion of patients with sac regression was similar during the study period: 58%, 66%, 60%, 59%, and 63% at 1 to 5 years, respectively. The proportion of patients with sac growth increased over time to 14.8% at 4-year follow-up. The probability of freedom from sac growth at 4 years was 82.4%. Eighty patients (37.7%) had an endoleak detected at some time during follow-up with 29.6% (16 of 54) residual endoleak rate at 4 years; 13 of the residual 16 endoleaks were type II. All patients with sac growth had endoleaks at some time during the study compared with only 18% of patients with sac regression ($P < .0001$).**

Conclusion: **A sustained sac regression after AAA exclusion with ELPE is noted up to 5-year follow-up. Sac enlargement was observed only in the setting of a current or previous endoleak, with no cases of suspected hygroma formation noted. (J Vasc Surg 2011;53:1178-83.)**

Abdominal aortic aneurysms (AAAs) are routinely treated with endovascular repair (endovascular aneurysm repair [EVAR]) safely and with good short-term results.[1,2] Early data have shown that sac behavior after EVAR is device-specific.[3,4] Reports from the Regulatory Trial using the Original Gore Excluder (OGE; W. L. Gore & Associates Inc, Flagstaff, Ariz) showed that 14% of patients experienced sac growth at 2 years after an EVAR.[5] Subsequently, we reported an alarming sac growth in 37% of patients at 4-year follow-up due to either continuous sac growth or re-expansion of once shrunken aneurysm sacs.[6] Sac growth in several patients occurred in the absence of endoleaks and was attributed to transgraft flow of serous fluid, hygroma formation, and endotension. These findings led to the modification of the OGE with the addition of a new low-permeability layer. The new Excluder low-permeability endoprosthesis (ELPE) was released in mid-2004. It incorporates a low-porosity film into the construct of the device making it impermeable to fluids while maintaining the same luminal and abluminal surfaces, device material thickness, deliverability, and device handling characteristics. Early assessments suggested these modifications enhanced sac regression similar to other endografts.[7,8] However, it has not been established yet whether sac regression is sustainable past 1 year and whether hygroma formation is no longer a threat in the long term. In this study, we sought to evaluate the mid-term to long-term sac behavior after EVAR with the ELPE.

METHODS

From July 2004 through December 2007, 301 patients underwent elective EVAR at two institutions using the ELPE device. There were 216 patients with at least 1-year follow-up imaging data available for sac behavior and endoleak analysis. The institutional review boards at the Northwestern University Feinberg School of Medicine and the University of Pittsburgh School of Medicine approved the study protocol. Follow-up schedules were slightly dif-

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ferent between institutions and evolved over time. Patients were universally scheduled to undergo an abdominal computed tomography (CT) scan at 1 and 12 months after implantation with yearly follow-up thereafter. At one institution, Duplex ultrasonography scan replaced CT scanning in some patients with impaired renal function or shrinking aneurysm sacs after the first year of follow-up, and in patients with stable sacs after 2 years.[9] The other institution continued to utilize CT imaging in all patients. In the renal impaired, sac size was determined from noncontrast CT images and endoleaks were sought using Duplex ultrasound scan. The presence or absence of an endoleak was determined either by Duplex scan or by CT scans performed with early and late phase–contrast-enhanced imaging.

The method used for measuring changes in dimension of the aneurysm was in accordance with the Society for Vascular Surgery reporting standards for EVAR.[10] Both major and minor axes were measured from adventitia to adventitia on a digital workstation for CT scans. Similar measurements were obtained on transverse duplex scans perpendicular to the centerline of the aneurysm. AAA sac size change was determined by comparing the minor axis measurements between the baseline and all subsequent imaging; major axis measurements were not used as they do not necessarily represent the true diameter of the AAA, as the AAA sac may not be parallel to the axis of the body. Sac size at 1 month was used as baseline measurement as pretreatment size may not accurately represent preoperative size, as there may be a significant variability in time interval between the last preoperative imaging and repair. Sac diameter size change of ≥5 mm was considered significant. All CT measurements were done by the treating physicians. Statistical analysis was performed using statistical software R (version 2.11.0). Quantitative values were calculated using regression analysis while categorical values were analyzed using the χ^2 test or the Fisher exact test. Values are listed as mean ± SD. Kaplan-Meier curves were used to assess freedom from sac growth over time.

RESULTS

Clinical results. Device implantation was successful in all but 1 patient due to failure to access the contralateral limb. This patient subsequently underwent aneurysm repair with an aorto-uni-iliac device (Renu; Cook Medical Inc, Bloomington, Ind). Perioperative deaths occurred in 6 patients after placement of an ELPE: 1 patient from colonic ischemia, 2 patients from pneumonia, and 3 patients within 30 days from other comorbidities yielding a total mortality of 1.99%. A perioperative stroke occurred in 1 patient, but no patient suffered a perioperative myocardial infarction. One patient with chronic renal insufficiency went on to develop endstage renal disease from contrast-induced nephropathy requiring temporary dialysis.

Total perioperative and late graft-related complications are detailed in the Table. Two patients had early limb complications, one associated with thrombosis of the common femoral artery requiring open repair, and the other was repaired with an iliac stent. Four additional patients had late limb complications requiring subsequent stent placement.

Table. Early and late complications after EVAR with the Excluder low-porosity endoprosthesis

Reported complications	*No. of patients*	*%*
Perioperative death	6	1.9
Failure to deploy device	1	0.3
Stroke	1	0.3
Renal failure	1	0.3
Graft infection	1	0.3
Limb dissection/thrombosis	2	0.7
Migration	4	1.3
Rupture (late)	2	0.7

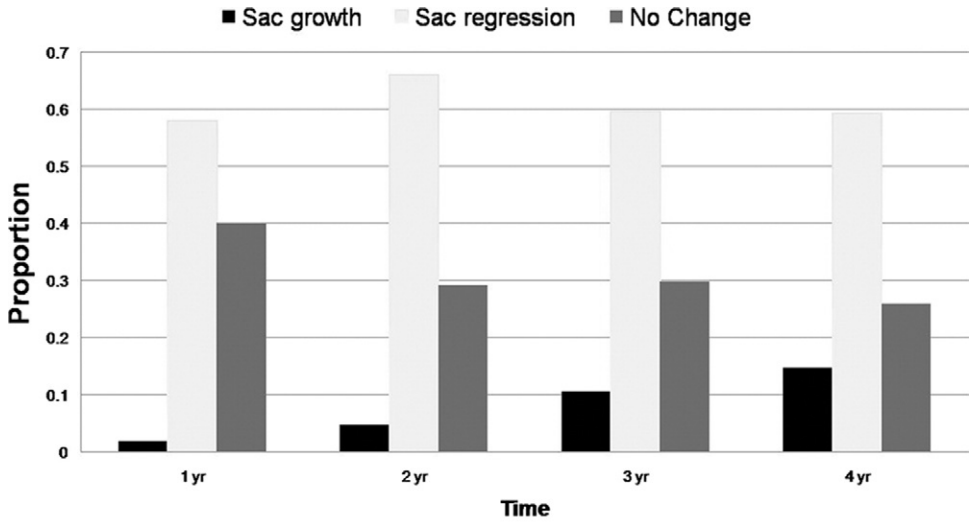
EVAR, Endovascular aneurysm repair.

Sac size change. Baseline AAA sac size was 51.8 ± 8.3 mm. There were 71 patients whose AAA measured <5 cm at baseline, of whom 16 patients had AAAs that measured >5 cm on preoperative imaging studies. For the remaining 55 patients, the indications for repair were as follows: saccular aneurysm, 6 patients; large iliac aneurysm, 7 patients; rapid growth rate, 10 patients; desire for reasons such as strong family history/anxiety, 13 patients; surgeons' discretion for steadily growing AAAs that were almost but not quite 5 cm, 12 patients; symptomatic but not emergent AAA, 7 patients. Follow-up imaging was done by CT scans in the majority of patients (77%). The mean follow-up was 2.6 ± 1.3 years (range, 1-5 years).

Fig 1 illustrates the distribution of patients with different sac behaviors during follow-up. The proportion of patients with sac regression did not differ statistically over the course of follow-up: 58%, 66%, 60%, 59%, and 63% at 1 to 5 years, respectively ($P = .65$). Similarly, the proportion of patients with no change in sac size was equivalent during follow-up: 40%, 29%, 30%, 26%, and 33%, respectively ($P = .84$). However, a steady increase was noted over time in the percentage of patients with sac growth reaching nearly 15% on the 4-year imaging studies.

Sac regression was observed in 134 patients at last follow-up. The mean decrease in sac diameter was 14.1 ± 6.7 mm among patients with sac regression. Thirty-nine patients had a decrease between 5 to 9 mm, 66 patients between 10 to 19 mm, 28 patients between 20 to 29 mm, and 1 patient over 30 mm.

During the course of follow-up, 17 patients manifested an increase in sac size by ≥5 mm compared with baseline. The mean increase in sac size was 13.2 ± 7.7 mm. Increase in size between 5 to 9 mm was noted in 8 patients, between 10 to 19 mm in 7 patients, between 20 to 29 mm in 1 patient and >30 mm in 1 patient. Two patients ultimately required elective open conversion and device explantation. In 1 patient, the AAA sac expanded by 34 mm despite multiple interventions to treat endoleaks (attempted coil embolization, proximal and distal extensions, and banding of a common iliac artery via a retroperitoneal exposure) and ultimately underwent open conversion at 3.5 years after



	1 yr	2 yr	3 yr	4 yr
No change	81	42	28	14
Decrease	117	95	56	32
Increase	4	7	10	8

Fig 1. Percentage of patients with stable, shrinking, or enlarging abdominal aortic aneurysm (AAA) sac at each follow-up interval compared with baseline measurement.

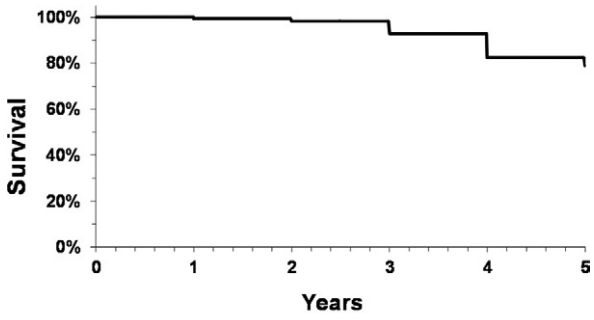
EVAR. The other patient who underwent initial EVAR with a very short neck of approximately 8 mm was lost to follow-up after 1 month. He presented with a right limb occlusion and distal migration of the endograft into the sac 3 years later. He underwent an uneventful open surgical reconstruction.

Re-expansion beyond baseline after initial shrinkage of the AAA sac occurred in 2 of these 17 patients. They both had a decrease in sac size by 5 mm initially but the sac re-expanded by 6 mm above baseline at 3 years in 1 patient and by 5 mm at 4 years in the other despite an earlier coil embolization of a lumbar artery; they both had type II endoleaks.

The probability of freedom from sac growth compared to baseline measurements for the entire cohort of patients was 98.3% at 2 years, 92.8% at 3 years, 82.4% at 4 years, and 78.9% at 5 years based on Kaplan-Meier estimates (Fig 2).

Endoleak. Of the 216 patients, 212 (98%) had endoleak data available for analysis. Eighty patients (37.7%) had evidence of endoleak at some point during follow-up, while at 4-year imaging, 16 of 54 patients still demonstrated an endoleak (29.6%); 3 were type I and 13 type II. At last follow-up, 56 patients (26.5%) had endoleaks: 8 patients had type Is and 48 type IIs. No type III endoleaks were noted. It is of interest to note that of the 8 patients with type I endoleak, 4 patients had no sac change, 2 patients had sac regression, and 2 patients had sac growth. Both patients with sac growth required open conversion with device explantation as detailed above.

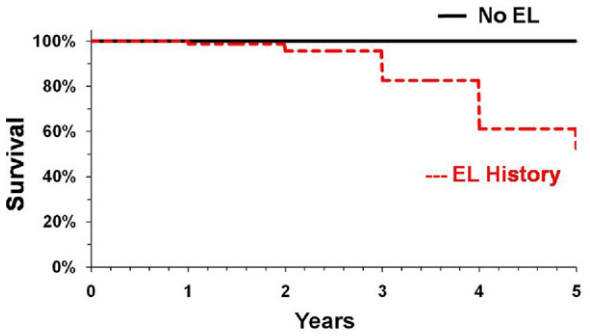
All 17 patients with sac growth had endoleaks at some time during follow-up. In comparison, only 18% patients



Time (years)	# at risk	Survival	SE
1	216	.995	.0046
2	161	.983	.0098
3	108	.928	.024
4	62	.824	.043
5	24	.789	.053

Fig 2. Probability of freedom from abdominal aortic aneurysm (AAA) sac enlargement compared with baseline after endovascular repair of AAA with low permeability Excluder endoprosthesis.

(24 of 134) who exhibited sac regression had an endoleak at any time ($P < .0001$). All but 1 of 17 patients with sac expansion continued to have evidence of an endoleak at last follow-up. Patients without significant sac size change had an even split between the presence and absence of endoleaks. The probability of freedom from sac growth at 4 years was 100% for patients without and 61.2% for those with history of endoleaks (Fig 3).



	No Endoleak			Endoleak		
Time (years)	# at risk	Survival	SE	# at risk	Survival	SE
1	132	1	0	80	.988	.013
2	95	1	0	64	.957	.026
3	63	1	0	44	.826	.065
4	35	1	0	27	.612	.13
5	17	1	0	7	.525	.21

Fig 3. Probability of freedom from abdominal aortic aneurysm (AAA) sac enlargement in patients with and without current or previous endoleaks compared with baseline after endovascular repair of AAA with low permeability Excluder endoprosthesis. *EL*, Endoleak.

An endoleak was detected at last follow-up in 46 of 151 patients (30%) of CT scans compared with 10 of 65 patients (15%) of Duplex scans. This may be related to the fact that follow-up imaging modality was switched to Duplex scans in patients with stable or shrinking AAA sacs.

Late ruptures. There were two late fatal ruptures in the present series. One initially underwent EVAR with an unfavorable proximal neck anatomy due to prohibitive cardiac condition for an open repair. He was noted to have a type I endoleak with slow sac enlargement by 11 mm at 4.5 years for which coil embolization was attempted. By 5-year follow-up, the AAA sac had grown 14 mm larger than baseline; he underwent proximal extension with the use of a "chimney" graft to preserve one of the two renal arteries at 5.5 years. He presented 2 months later with a rupture and died on the operating table. The other rupture occurred in a patient who had a stable AAA sac with no endoleaks during her first 2 years of follow-up. Unfortunately, she had just missed her 3-year follow-up appointment when her AAA ruptured. She was noted to have a type II endoleak from lumbar arteries and underwent surgical graft implantation, but died in the hospital.

DISCUSSION

Several studies have reported on a short-term sac behavior after EVAR using the Excluder device.[7,8,11] We previously reported that sac regression rate 1 year after EVAR with ELPE was significantly higher compared with the OGE (63.9% vs 25%, respectively) and that it was similar to that of the Zenith (Cook Medical Inc, Bloomington, Ind) endograft (65.3%).[8] Tanski et al[7] observed a significantly decreased sac size and 3-dimensional volume with ELPE (−5.1 mm and −20%, respectively) compared with OGE (−2.1 mm and −6%, respectively). Broker et al,[11] on the contrary, noted a significantly lower absolute sac regression with the ELPE device (34%) as compared with the Zenith (63%) and AneuRx (Medtronic Vascular, Santa Rosa, Calif) endografts (65%) at 18-months follow-up. Later follow-up on the ELPE device, however, has not yet been reported until now.

The present study illustrates a significant rate of AAA sac regression that is sustained during the first 5 years of follow-up. The 59% 4-year sac regression rate with ELPE seen in this study compares favorably to a 4-year 21% sac regression rate we reported with the original Excluder[6] and it remains comparable to the 1-year sac shrinkage rate we reported with ELPE.[8]

While a rare event, this study again shows that a once shrunken sac may re-expand.[6] This was observed in 2 patients, at 3-year and 4-year follow-up, and highlights the need for continued surveillance even in the setting of sac regression.

As with all EVAR procedures and all endograft devices, the presence of endoleaks may ultimately lead to enlargement of the sac. The sac growth rate in this study was 15% at 4 years after EVAR. Although not as high as was seen with the OGE (37% at 4 years), it remains significant. It should be noted, however, that no significant sac enlargement to date has been observed in the absence of a previous or current endoleak. No patients were suspected of having a sac expansion due to hygroma in the absence of an endoleak. It thus appears that the modification of the Excluder device with the addition of the low porosity layer may have been successful in eliminating transgraft flow that accounted for 74% of sac growths in the OGE trial.[12] The incidence of history of endoleak in the present study was 34%, within the range currently reported in the literature.[8,13-17] As with all endografts, however, incomplete exclusion may lead to a continued expansion of the sac, which has to be monitored carefully through continued imaging. It is possible that the increasing number of patients with growing sacs over the course of follow-up may be related to increasing incidence of endoleak development as follow-up duration increases or delayed onset of transgraft flow of serous fluid. The decrease in sac growth rate observed at 5 years may reflect a type II statistical error related to the small number of observations at that time period.

The natural history of AAA sac change after EVAR in the presence of a type II endoleak remains to be defined. Discrepancies exist in the literature regarding adverse sequelas as a result of blood flow within the aneurysm sac. Just as sac behavior has been determined to be device-specific, the percentage of postoperative endoleaks after EVAR has been reported to vary from 18% to 65%.[14-17] Ouriel et al[3] showed that type II endoleaks are graft-dependent with the highest rate of endoleak observed with the OGE (58%) compared to the Talent device (Medtronic Vascular) which had the lowest rate (19%). Sheehan et al,[18] however, in a later study showed that this initial variability in the incidence of endoleaks were obliterated over time

with similar rates of spontaneous endoleak resolution and late endoleak development. The proportion of patients with endoleaks at last follow-up was 25% in the current study, in accordance with the findings of other studies[7,11] that showed a higher incidence of endoleak in patients with sac growth than in those with sac regression.

Data on the effects of endoleak on sac growth and their potential graft-specific nature are conflicting. Fairman et al[14] in a multicenter study of 351 patients treated with the Zenith endograft noted that endoleaks were predictive of sac growth. Greenberg et al,[13] observed that, in the presence of a small endoleak, the aneurysms treated with a Zenith endograft would decrease in volume, whereas those treated with the OGE would expand. Jones et al,[15] in a series of 873 patients undergoing EVAR with 10 different stent grafts at a single institution, reported that endoleaks were associated with sac growth and rupture. Statistical analysis breaking the cohort into subgroups based on stent graft was not performed to determine whether these findings were procedure-specific or device-specific.[15] The European Collaborators on Stent/graft Techniques for aortic Aneurysm Repair (EUROSTAR) experience, with 2463 patients from 87 European centers, had enough patients to perform a power analysis based on endoleak type combining patients with type I and III endoleaks.[16] They found that type I and III, but not type II, endoleaks correlated with increased risk of rupture, but changes in aneurysm sac size was not included in this analysis. The absence of endoleak, however, does not always imply an uncomplicated course. Zarins et al[19] reported 7 ruptures in 1046 patients who underwent EVAR using the AneuRx device; 5 had no evidence of endoleak. In a series from the University of Pittsburgh of ruptured AAA with antecedent EVAR, most patients had no evidence of endoleak at last follow-up imaging before the rupture.[20]

There are several limitations in this study. It is a retrospective analysis of one device without another device for comparison. A significant number of patients were either lost to follow-up or had no imaging for review and may have introduced a selection bias. Being tertiary referral centers, a significant portion of our patients are referred from great distances. Unfortunately, these patients do not want to travel such great distances for follow-up. This makes it difficult for complete follow-up of these patients, explaining a relatively high attrition rate in follow-up. A selection bias may also have been introduced by following more closely patients with endoleaks, increasing their representation in the sample. In addition, patients with sac growth may be more apt to get serial imaging than those who have had partial or complete sac regression. Finally, not all patients underwent CT evaluation with contrast enhancement as the only imaging modality. Patients with compromised renal function or contrast allergy or those with stable or shrinking sacs were switched to Duplex scans at one institution introducing a possible error in size comparisons. The accuracy of Duplex relative to CT imaging, however, is well validated and has been shown in recent studies.[9,21]

CONCLUSIONS

The ELPE has shown a sustained sac regression rate up to 5-year follow-up. The addition of a low porosity layer in the ELPE seems to have eliminated the transgraft flow of serous fluid and hygroma formation. Although AAA sac enlargement continues to be observed, it seems to be restricted to patients with current or previous endoleaks. Longer-term follow-up data would be necessary to place this finding into proper perspective. As with all EVAR patients, continued surveillance is mandatory even in patients with stable or shrinking AAA sacs to better understand late performance and help prevent late complications.

AUTHOR CONTRIBUTIONS

Conception and design: MDM, MSM, JC
Analysis and interpretation: MH, MDM, MSM, JC
Data collection: MH, WF, MDM, JC
Writing the article: MH, MDM, MSM, TP, JC
Critical revision of the article: MH, MDM, MSM, JC
Final approval of the article: MDM, JC
Statistical analysis: TP
Obtained funding: Not applicable
Overall responsibility: JC

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