Three-dimensional analysis of enlarging aneurysms after endovascular abdominal aortic aneurysm repair in the Gore Excluder Pivotal clinical trial

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Objective: **Recent reports have raised concern about the percentage of enlarging abdominal aortic aneurysms (AAAs) after endovascular repair with the Gore Excluder device. As part of the investigation into this issue, a morphologic analysis was performed on enlarging aneurysms in the Excluder Pivotal clinical trial.**

Methods: **Computed tomographic scans were evaluated on all patients identified with enlarging aneurysms (5-mm increase by Core laboratory or site) and at least 4 years of follow-up in the Excluder Pivotal clinical trial. Three-dimensional reconstruction, a set of 24 standard morphologic measurements, and analysis of potential enlargement mechanisms were performed.**

Results: **Of 112 trial patients with 4 years of follow-up, 38 AAAs (34%) were identified as enlarging. Data were obtained from 196 computed tomographic scans (the mean interval was 47 months from first to last scan). Of the 158 scans with a prior scan for comparison, 41% demonstrated growth relative to the initial scan by diameter criteria, but 79% demonstrated growth relative to the initial scan by 3-dimensional volume criteria ($P < .0001$ vs diameter; χ^2 analysis). This difference was most evident at early time points: at 1 year, diameter criteria indicated that 8% of these AAAs were enlarging, but 56% were already enlarging by volume criteria. On average, enlargement was detected by volume 18 months before it was detected by diameter ($P < .0001$), and at a smaller diameter (55 ± 1 mm vs 60 ± 1 mm; $P < .0001$). Only 19% of scans (39% of patients) had apparent endoleaks. Scans with apparent endoleaks demonstrated a greater interval rate of growth as compared with those without apparent endoleak (3.6 ± 0.8 mm vs 1.9 ± 0.3 mm [$P < .02$] by diameter; 23 ± 4 cm^3 vs 11 ± 1 cm^3 [$P < .001$] by volume). Although the etiology of enlargement may be endotension or device permeability in up to 74% of patients, other potential causes of aneurysm enlargement included neck apposition length less than 15 mm (15 patients; 39%), large aortic diameter relative to device (18%), large iliac diameter (5%), and iliac apposition length less than 15 mm (20%). Multiple potential etiologies of enlargement were present in 53% of AAAs.**

Conclusions: **The etiology of aneurysm enlargement in the Excluder Pivotal trial is likely multifactorial, including endoleak, inadequate attachment site length, and endotension or device permeability. Even by conservative criteria, a substantial percentage of aneurysm growth with the original device is likely due to material permeability. Three-dimensional volume criteria detected aneurysm enlargement more frequently, at a smaller diameter, and on average 18 months sooner than standard diameter criteria, thus suggesting a role in further investigation of this issue. (J Vasc Surg 2006;43:888-95.)**

Recent reports have raised concern about the percentage of enlarging abdominal aortic aneurysms (AAA) after endovascular repair with the Gore Excluder device (WL Gore, Flagstaff, Ariz).[1-3] This led to an extensive investigation into potential causes of AAA enlargement, with the conclusion that graft material permeability in the original Excluder device may be the etiology of aneurysm sac enlargement in a significant number of cases. As part of this investigation, a more detailed analysis of the enlarging aneurysms in the original Excluder clinical trial was also undertaken. This report represents a morphologic analysis of the subset of patients with enlarging aneurysms at the 4-year time point in the Excluder Bifurcated Endoprosthesis 98-03 Pivotal clinical trial,[4] based on computed tomography (CT) scans, three-dimensional reconstruction, and morphologic measurements.

METHODS

Patient population and data supplied. CT scans were evaluated on all patients identified with enlarging AAAs (a 5-mm increase by Core laboratory or site) and at least 4 years of follow-up in the Excluder 98-03 Pivotal clinical trial (2004 Annual Clinical Update Report). All patients participating in the trial received informed consent in institutions with institutional review board approval for the study, and all patients met anatomic selection criteria at the time of enrollment (1998-2000) by using the imaging methodology available at the time. CT scans for this subset of patients with enlarging AAAs were sent to Medical Metrx Solutions (West Lebanon, NH) for three-dimensional reconstruction. A set of 24 standard morpho-

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logic measurements was performed, followed by analysis of potential enlargement mechanisms by using morphologic data, 3-dimensional reconstructions, and data regarding secondary interventions and diameter changes reported by each site relative to the clinical trial.

Definitions and criteria for evaluation. The maximum AAA sac diameter was measured as the maximum AAA diameter on CT reformat perpendicular to the lumen centerline. AAA size change was measured both relative to the initial postoperative CT scan and as the interval change from the previous scan. Three-dimensional volume was measured from the lowest renal artery to the aortic bifurcation and from the lowest renal artery to the common iliac artery bifurcation (to capture iliac aneurysms). Total volume was based on three-dimensional reconstruction of CT data for the outer boundary of the vessel, including lumen, thrombus, calcified plaque, and metallic stent. For diameter change, 5 mm or more was considered to be a significant change (based on interobserver variability more than 3 mm and Society for Vascular Surgery [SVS] reporting standards).[5-7] For three-dimensional volume change, 5% or more was considered significant, based on SVS reporting standards[6,7] and our interobserver variability of 2.5%.

Other parameters for evaluation included aortic neck length, neck diameter at multiple sites, and achieved aortic neck apposition (100% circumference); initial aneurysm sac diameter; iliac length, diameter (maximum and average), and achieved apposition length (all for both right and left iliacs); aortic neck angulation; device angulation over time; endoleak during the interval being examined; and endoleak at any time. These measurements were defined and reported according to SVS reporting standards.[6]

Techniques. Morphologic assessment was made by using three-dimensional computer-aided measurement, planning, and simulation technology.[8-10] Measurements were made by using electronic calipers on digital CT data with multiplanar CT reformats, including orthogonal reformats for diameter. Length and angle measurements were made along the lumen centerline in three-dimensional space. CT scan techniques and intervals were according to institutional protocols, with guidance within the parameters of the 98-03 study.[4]

Statistical methods. Anatomic measurements were analyzed with a standard statistical software package (StatView; SAS Institute, Cary, NC). Nominal variables were compared by χ^2 or Fisher exact test where applicable. Continuous variables were analyzed by using analysis of variance and linear regression.

RESULTS

Patient population. Of the 235 test patients in the 98-03 trial, 112 patients were identified with 4-year follow-up (site reported; last update August 2005). Of these, 38 AAAs (34%) were identified as enlarging at some point during the course of follow-up, most at 3- to 4-year follow-up by site or Core laboratory (36 of 38 in agreement). Data were obtained from 196 CT scans, with a mean interval of 11.2 months between scans and a mean of 47 months from first to last scan. Only seven scans were unavailable or unusable from routine follow-up. When compared with the overall population of AAAs undergoing endovascular aneurysm repair in the trial, the key baseline anatomic characteristics seem similar for this subset of enlarging aneurysms (Table I).

Mean diameter and volume over time and detection of enlargement. As would be expected in a study of aneurysms identified to be enlarging, both diameter and volume increased over time. The mean diameter change between scans was 2.3 ± 0.3 mm, compared with a 9.0% ± 0.9% mean volume change. The apparent growth relative to the threshold for detection was quite different for the two techniques. Most aneurysms did not enlarge beyond the threshold for detection by diameter until 3 years of follow-up, as shown in Fig 1. This is consistent with data reported for the trial, in which only 6 of these 38 aneurysms were identified as enlarging by 2 years of follow-up. By contrast, most aneurysms enlarged beyond the threshold for three-dimensional volume detection by 1 year of follow-up, as shown in Fig 2. This difference in the two techniques is demonstrated by the percentage of aneurysms identified as enlarging at each time point (Fig 3). In this study, 11 aneurysms (of 38) were identified as enlarging by diameter at 2 years, compared with 6 by site-reported diameter data and 30 by 3-dimensional volume (Fig 3). On average, enlargement was detected by volume 18 months before it was detected by diameter (18 ± 2 months vs 36 ± 2 months; $P < .0001$), and at a smaller diameter (55.4 ± 1 mm vs 59.8 ± 1 mm; $P < .0001$).

Early in the Pivotal trial, a criterion of a 3-mm diameter change was attempted but was found to result in a number of false positives (aneurysms reverting back and forth between enlarging and shrinking on subsequent studies), so the criterion of 5 mm was adopted, according to the SVS reporting standards. Even with a diameter threshold below typical interobserver variability, however, the percentage of AAAs identified as enlarging was lower by diameter (Table II). Volume criteria have always been reported as a percentage, but for comparison, using a 5% threshold for diameter was also significantly worse than using the three-dimensional volume (Table II). Even a volume criterion of 10% change identified 68% of AAAs increasing relative to the first scan, which is still significantly better than the 3-mm or 5% diameter criteria ($P < .03$).

Endoleak as a potential cause of AAA enlargement. Only 18% of the scans (36 of 196) had apparent endoleak, but scan protocols, including contrast timing, slice thickness, and a lack of three-phase or delayed contrast studies, likely underestimated the true percentage of cases with endoleak. On the basis of endoleak at any time point, 39% of patients (15/38) had an AAA with an endoleak on at least a single scan. All of the endoleaks seemed intermittent to some degree, but in three patients only a single scan out of the entire series lacked endoleak, and this seemed to be due to poor contrast density rather than a true lack of endoleak. At least some endoleaks were likely transient, however, because 5 of these 15 patients had endoleaks that

Table I. Comparison of baseline measurements for the enlarging group and entire Pivotal trial (EVAR patients)

Measurement (based on first scan applicable)	*Enlarging group (n = 38)*	*EBE Pivotal trial (EVAR patients; n = 235)*
AAA maximum diameter (mm)	53.5 ± 1.4	55.6 ± 0.6
AAA volume renal-aobif (cm^3)	140 ± 11	NA
AAA volume renal-hypo (cm^3)	157 ± 11	NA
Ao neck diameter (mm)	22.4 ± 0.4	22.3 ± 0.1
Ao neck diameter 15 mm distal to lowest renal artery (mm)	23.5 ± 0.5	NA
Ao neck length	NA	28.9 ± 0.7
Achieved aortic neck apposition by stent graft	18.8 ± 2.0	NA
Distance lowest renal to top of graft (mm)	0.5 ± 1.2	NA
Neck angulation (neck to AAA) (°)	30.0 ± 0.6	22.0 ± 1.1
Aortic angle suprarenal aorta to infrarenal aorta	13.2 ± 1.4	NA
R common iliac diameter	12.8 ± 0.4	12.4 ± 0.2
R CIA maximum diameter	16.2 ± 0.5	NA
R iliac apposition length	32.1 ± 4.0	NA
L common iliac diameter	12.7 ± 0.4	11.8 ± 0.2
L CIA maximum diameter	15.9 ± 0.7	NA
L iliac apposition length	28.5 ± 2.6	NA

EVAR, Endovascular aneurysm repair; *AAA,* abdominal aortic aneurysm; *Ao,* aortic; *R,* right; *L,* left; *EBE,* Excludes Bifurcated Endoprosthesis; *CIA,* Common Iliac Artery; NA, Not Available or Not Applicable; *aobif,* aortic bifurcation; *hypo,* hypogastric artery. All diameters and lengths are in mm, all angles in degrees, all volumes in cc (cm^3).

P values are not applicable because measurement methods were not the same. Because of the number of patients without adequate preoperative computed tomography for three-dimensional reconstruction, preoperative neck length was not reported. Baseline measurements are from the 1-month postoperative tomography scan for most cases in the enlarging group.

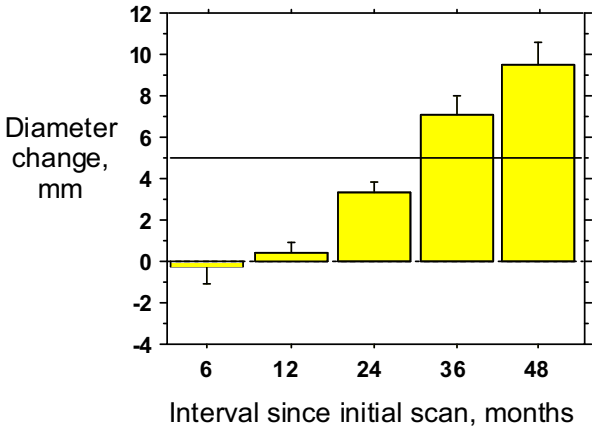

Fig 1. Mean diameter change compared with initial computed tomography.

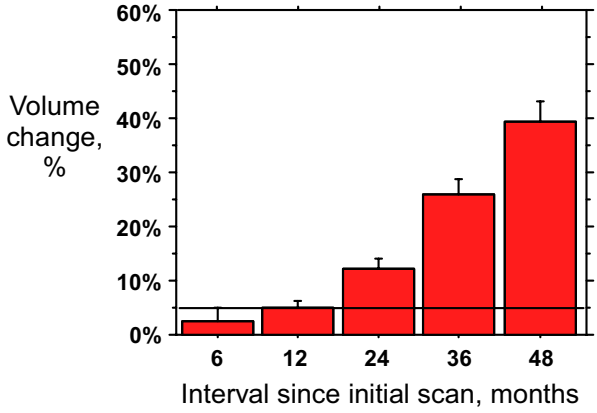

Fig 2. Mean three-dimensional volume change compared with initial computed tomography. Three-dimensional volume is shown from lowest renal to aortic bifurcation. The graph for volume from renal to hypogastric seems similar (eg, the volume change from renal to hypogastric is 45% at 4 years rather than 40%).

were apparent only in the first 1 or 2 scans and were then no longer visible on multiple scans. Almost all endoleaks seemed likely to be type II, and only one potential type I endoleak was reported.

Despite concern about visualizing all endoleaks, AAAs with apparent endoleak demonstrated roughly twice the interval rate of growth as compared with AAAs without apparent endoleak (Table III). The comparison for rate of growth is affected by initial aneurysm size, which was larger in patients with endoleak, but there is still a significant trend even when evaluation is performed by volume percentage (Table III). Aneurysm growth was not proportional to endoleak size, whether for interval AAA diameter change vs endoleak volume ($P = .66$), interval AAA volume change vs endoleak volume ($P = .37$), or diameter or volume change relative to initial scan vs endoleak volume ($P = .4$-$.9$). Thus, interval AAA growth seemed to be related to the presence or absence of endoleak but did not seem to correlate with the size of the endoleak in this series.

Association with other morphologic criteria and possible endotension. Examination of the data in Table I indicates that most patients met the inclusion criteria in terms of preoperative anatomy, even with the three-dimensional computer-aided measurement technology.

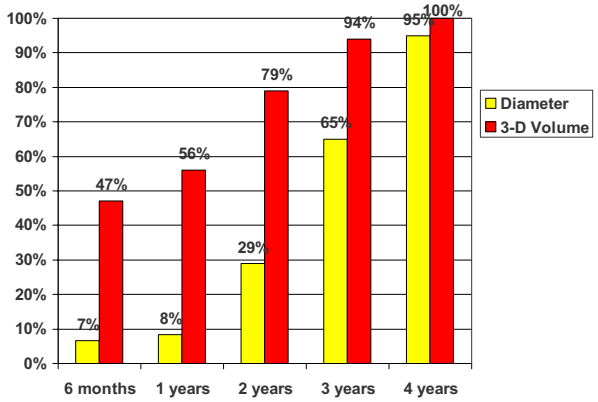

Fig 3. Percentage of enlarging abdominal aortic aneurysms over time. A comparison of the percentage of enlarging aneurysms by diameter and volume criteria is shown at each time point. *3-D,* Three-dimensional.

However, it is also apparent that some patients did not meet desirable goals for fixation and sealing parameters.

In terms of achieved aortic neck apposition length, 15 (39%) of 38 patients had a neck apposition length less than 15 mm at some point during follow-up. Using a criterion of 100% circumference neck apposition greater than 5 mm in length, six patients (16%) may have had poor neck apposition as a factor in aneurysm growth. Many patients did not have a preoperative scan available and adequate for three-dimensional reconstruction, so the percentage who started out with inadequate available neck length is unclear. At least one patient clearly could not have had an acceptable neck initially (Fig 4). The device was implanted immediately below the renal arteries, there was no apparent endoleak, and the AAA diameter was stable for 3 years, but the AAA volume increase was apparent by 1 year and thereafter. It seems that three patients had devices initially implanted more than 1 cm distal to the lowest renal artery despite a neck that met trial inclusion criteria before surgery. In other cases there was thrombus, diameter change (neck enlargement), and/or angulation within the apparent infrarenal neck that seemed to diminish the length of neck apposition. At least one neck apposition length decreased as a result of aneurysm growth over time (Fig 5). This patient had no apparent endoleak on any postoperative study but did have early suboptimal iliac fixation that may have produced sufficient endotension to cause aneurysm growth.

Aortic neck dilation. Infrarenal aortic neck diameter just proximal to the top of the graft seemed to meet inclusion criteria (26 mm) in all but two patients on the initial postoperative scan but exceeded inclusion criteria by at least .5 mm in seven patients (18%) at later time points. The neck diameter 15 mm below the renal arteries also met inclusion criteria on the initial postoperative scan in 36 of 38 patients but was 28 mm or more at later time points in 25 scans (16%) and 9 patients (24%). Both of these parameters suggest a mild dilation of the neck, common with all endografts, and this did not seem to be a common etiology of sac enlargement in this series.

Iliac attachments. Iliac artery diameters were initially at least 1 mm larger than inclusion criteria at the site of apposition in 14 (37%) of 38 patients, on the basis of the outer diameter (including stent). Lumen diameter, not outer wall diameter, was used for inclusion criteria in the trial, and the lumen diameter likely met inclusion criteria in most cases. Thus, iliac endograft sizing was an unlikely cause of aneurysm growth, even considering endotension without endoleak. In terms of iliac apposition length, six limbs had inadequate apposition length (clearly <15 mm) at some point during follow-up, and nine other limbs had possibly inadequate apposition length at some point during follow-up. Thus, at most 15 (20%) of 76 limbs had a possibly inadequate fixation/seal length at some point during follow-up. Two patients with such limbs are shown in Fig 6. Several of these cases could theoretically have had pressure transmission through thrombus, but this was not a likely etiology of aneurysm enlargement in most patients.

Other morphologic factors. Other factors from the literature or trial inclusion/exclusion criteria were present in small numbers. In one patient, mild angle changes allowed the neck-AAA body angle to reach 60°, but this was not a source of endoleak or other problems. There was progressive device deformation (defined as progressive angulation $> 15°$) in only one patient, and it also did not cause endoleak or fixation problems. Angle changes were minimal on average. Angulation from neck to aneurysm body was $30° \pm 2°$ on initial scan and $33° \pm 3°$ at 4 years. Only one case had significant migration ($\geq$5 mm) at any time during follow-up (migration of 7 mm in 3 years), and this did not decrease the aortic neck apposition to less than 15 mm or result in endoleak. For all 38 patients, the mean distance from the lowest renal artery to the top of the graft changed from 0.5 ± 1.2 mm on the initial scan to a mean of 2 ± 0.6 mm on all remaining scans, with no significant change by analysis of variance for distance over time at yearly intervals. There were no stent fractures and no ruptures.

Summary of potential etiologies of enlargement. Each AAA was labeled with potential etiologies of enlargement by the described criteria: 53% had multiple potential etiologies (endoleak or endotension due to transmission of pressure through thrombus at one or more attachment sites), 26% had endoleak as the only clear potential etiology, and 21% of AAAs had no apparent explanation for growth other than endotension due to material permeability issues.

Secondary interventions. In all, 17 of the 38 patients had a secondary intervention of some type. There were 13 coil embolizations, 1 inferior mesenteric artery ligation, 1 case with extensions, and 4 explants (2 patients had more than 1 intervention). In terms of treating endoleaks, only four coil embolizations seemed successful in obliterating the endoleak, but in each case, aneurysm expansion did not stop or slow. In fact, in three of the four cases in which endoleak was no longer apparent, the rate of aneurysm expansion seemed to increase by volume (although not by

Table II. Percentage of scans with increasing abdominal aortic aneurysm sac size at any time

Criterion	*% with interval sac increase (158 scans)*	*% with sac increase since initial scan (158 scans)*
Diameter, 5 mm	22	41
Diameter, 3 mm	36	51
Diameter, 5% change	39	56
Volume, 5% (renal-aobif)	58	78
Volume, 5% (renal-hypo)	56	76
Volume, 5% (either)	61	79

aobif, aortic bifurcation; *hypo*, hypogastric artery.
Data are based on 158 computed tomographic scans with a prior scan for comparison. $P < .0001$ for all diameter criteria vs volume for detection relative to initial scan (χ^2). Volume 5% (either) refers to either volume criterion, to capture changes in iliac aneurysms.

Table III. Rate of aneurysm growth based on apparent endoleak or lack thereof

Variable	*No endoleak*	*Endoleak*	P *value*
Interval diameter increase, (mm)	1.9 ± 0.3	3.6 ± 0.8	.02
Interval volume increase, renal-aobif (cm^3)	11.1 ± 1.3	22.0 ± 4.1	.002
Interval volume increase, renal-hypo (cm^3)	11.3 ± 1.4	23.4 ± 4.4	.001
Interval diameter increase (by %)	3.6 ± 0.5	5.7 ± 1.3	.12
Interval volume increase, renal-aobif (%)	7.9 ± 0.9	12.3 ± 2.2	.04
Internal volume increase, renal-hypo (%)	7.1 ± 0.8	11.9 ± 2.2	.02

aobif, aortic bifurcation; *hypo*, hypogastric artery.
Interval indicates the change compared with the prior scan. The mean interval between scans was 11.2 months.

diameter). The single case of device extensions was in an aneurysm that did not seem to have poor device apposition at the attachment sites or an attachment endoleak. Of all the patients with secondary interventions, 15 of 17 had a follow-up scan. Of these, only the four explants were successful in treating expansion. Notably, in three of the four explants, there was no apparent endoleak at any time, including examination of the sac at the time of explantation.

DISCUSSION

In this study, three-dimensional morphologic analysis indicates that multiple factors may be contributing to AAA enlargement after endovascular repair with the Gore Excluder device. The importance of each potential factor is open to interpretation, however, including the presence of endoleak. It thus seems important that the data in this study indicate that the interval rate of aneurysm growth was 50% to 100% greater in the scans with apparent endoleak compared with the scans with no apparent endoleak. This suggests that endoleak is important in at least some cases, despite some controversy on this issue. Nonetheless, only 39% of these patients had an endoleak at any time during 4 years of follow-up, and in 5 of the 15 AAAs with endoleak, the endoleak was apparent only in the first 1 or 2 scans and then was no longer visible on multiple subsequent scans. Either the endoleak was missed on later scans or the late aneurysm growth seen in each of these patients was due to another cause. In three of the four cases that went on to open surgical explantation of the endograft, no endoleak was detected upon direct examination of the sac (and in the fourth, endoleak was demonstrated on CT before explantation). Thus, it seems that endoleak can explain only a minority of the cases of aneurysm enlargement in these patients.

Multiple patients in this study seem to have inadequate attachment site apposition length according to current recommendations of appropriate pretreatment anatomy, which are of course somewhat arbitrary. The values chosen for adequate postoperative apposition length have been found to be important relative to endoleak and stable fixation for other devices, however.[11,12] To the extent that there is no apparent endoleak, the potentially inadequate apposition may be only a theoretical concern, because this requires endotension by transmission of pressure through thrombus at the attachment site, without endoleak. Of course, these may simply be cases of missed endoleak, but that does not explain the lack of endoleak at the time of explantation in this trial and in multiple other studies.[13-15] It is interesting that the aneurysm growth in these endotension cases was significantly slower than in the cases with visible endoleak. Perhaps this means that slow endoleaks undetected on CT without delayed contrast runs have a lower pressure than endoleaks visible on arterial-phase contrast-enhanced CT. Perhaps it means that pressure transmission through thrombus or by material permeability and transmigration of fluid occurs at a lower pressure than type II endoleaks. This analysis cannot answer these questions.

Very little is known about aneurysm enlargement due to material permeability, sac hygroma, or transmigration of fluid. It seems that this phenomenon can happen after endovascular or open repair, with polytetrafluoroethylene or polyester fabrics, and can even occur via the wall of the aneurysm in the absence of graft material.[13-18] Although believed by many to be benign, it can cause aneurysm

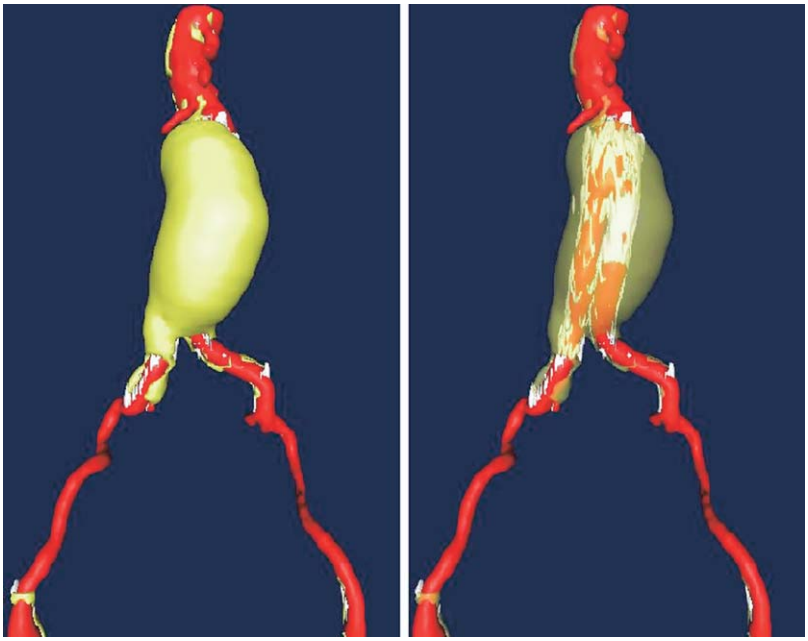

Fig 4. Initially inadequate infrarenal aortic neck with thrombus both solid and transparent in the three-dimensional model.

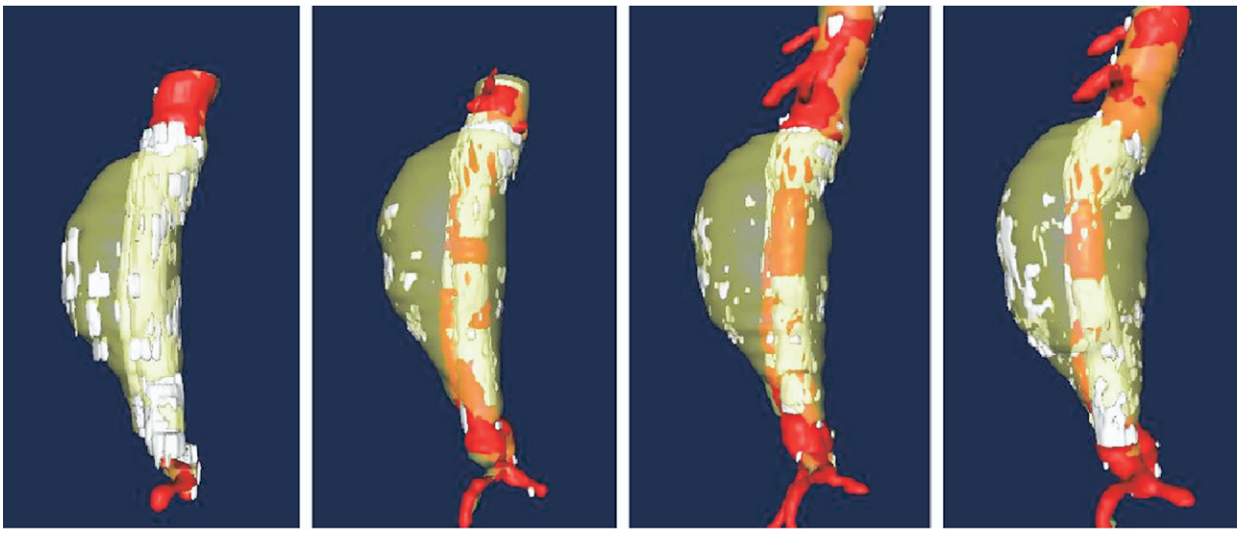

Fig 5. Decrease in neck apposition length due to aneurysm growth. This patient also had suboptimal iliac fixation.

rupture in rare cases.[14] Given all of the above, is there any way to infer or even estimate how many cases of enlargement might be due to graft permeability, in part or in whole? At a minimum, 21% of the cases in this study have no obvious explanation for aneurysm growth, even with a detailed three-dimensional analysis including numerous morphologic factors over time. Logically, enlargement in these cases is very likely due to graft permeability and transmigration of fluid. In the aneurysms with multiple potential etiologies, permeability may have at least a partial influence, because many cases with suboptimal apposition length (<15 mm) are unlikely to have so little apposition as to allow pressure to be transmitted at the attachment site. Thus, one might set an upper limit of 74% in which permeability may be in important issue. This assumes that permeability is not even a partial factor in the 26% of cases with persistent or "late" type II endoleak and no attachment site issues. The latter assumption is likely true, but it should be noted that the rate of aneurysm enlargement did not correlate with endoleak size in this study as it has in others,[19]

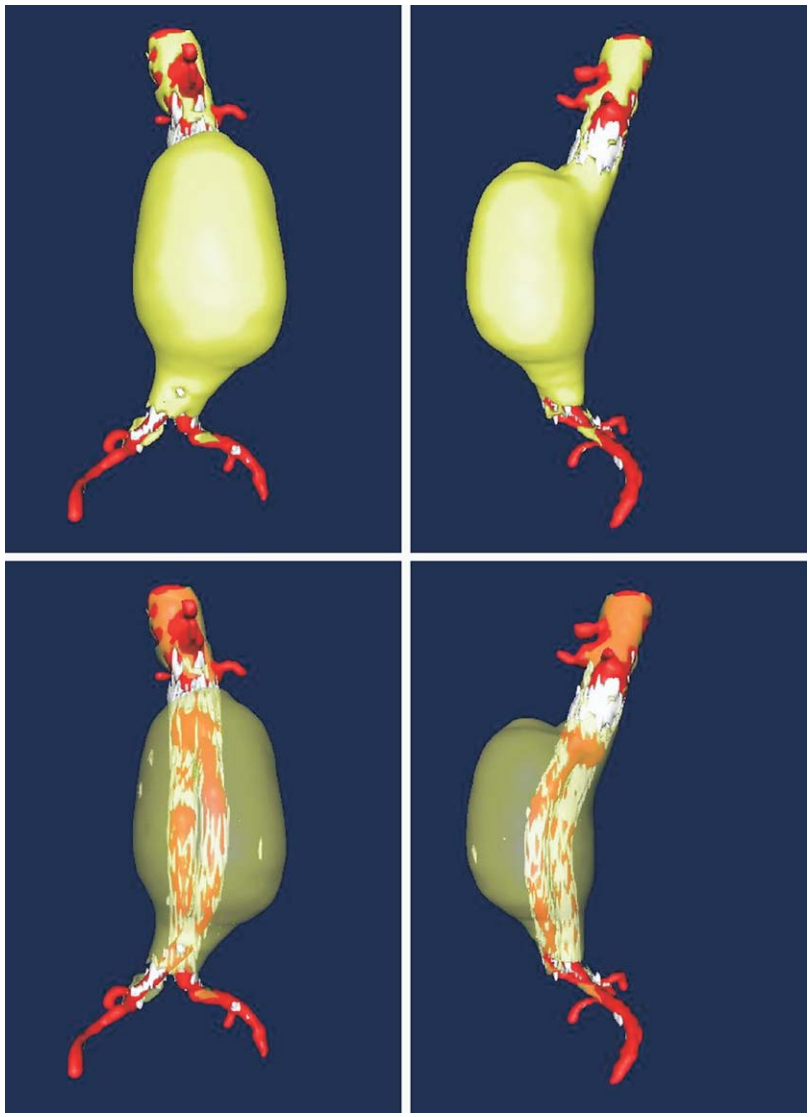

Fig 6. Potentially inadequate iliac apposition length for fixation and sealing. This patient went on to conversion to open repair for aneurysm growth in the absence of endoleak.

so we cannot rule out that permeability affects even endoleak cases.

In terms of other issues, the problems demonstrated in this analysis related to initial imaging, patient selection, device sizing, intraoperative device placement, and attachment site enlargement should be similar from one device to another. These problems can be minimized with good preoperative imaging for patient selection and evaluation of potential device issues,[8,10] as well as proper intraoperative imaging (eg, gantry adjustment for neck angulation).[8,9] There is no reason to believe that these issues should be significantly different for the Excluder device, however. Certainly, the data in this study for placement relative to the renal arteries, our own experience,[10,20] and the trial results[4] suggest that the device is readily placed at the desired location. Data from a meta-analysis of four clinical trials indicate that neck dilation can be associated with migration, but this is uncommon and is not significantly different between devices.[21] Thus, the difference in the percentage of aneurysm enlargement, or even lack of shrinkage, between the original Excluder and other devices could be due to permeability issues. Bertges et al[1] found that the rate of aneurysm enlargement for the original Gore Excluder was not significantly different from Ancure (Guidant, Indianapolis, IN) or Talent (Medtronic, Minneapolis, MN) at 2 years, but the incidence of shrinking aneurysms was 30% lower at 2 years with the Excluder device. Kong et al[15] studied 16 Excluder explants and found 7 with endoleak (2 had been assumed endotension) and 6 documented endotension cases. Both studies fit within our estimate of the

percentage of cases that might be related to material permeability.

One of the problems in evaluating this problem is that nearly all reports from clinical trials report size changes by using diameter. It is clear from this study that three-dimensional volume criteria detected aneurysm enlargement more frequently, at a smaller AAA size, and on average 18 months sooner than standard diameter criteria. Notably, site-reported diameter data detected enlargement at an even lower rate than the standardized diameter data derived from orthogonal CT reformats in this study. Thus, current reports based on diameter assessment of any kind seem to underestimate the true percentage of enlarging AAAs with this endograft and other endografts, especially at early time points. This has obvious implications not only for routine clinical care, but also for clinical trials. Clinically, three-dimensional reconstruction is commercially available from multiple sources and remains the gold standard, as has been suggested by ourselves and others.[9,10,22,23] With regard to clinical trials of new devices, these data indicate that in the absence of three-dimensional reconstruction, longer-term follow-up might be justified before commercial release.

CONCLUSIONS

The etiology of aneurysm enlargement in the Excluder Pivotal trial is likely multifactorial, including endoleak, endotension due to inadequate attachment site length, and endotension due to material permeability. Even by conservative criteria, a substantial percentage of aneurysm growth with the original device is likely due to material permeability. Three-dimensional volume criteria detected aneurysm enlargement more frequently, at a smaller diameter, and on average 18 months sooner than standard diameter criteria, thus suggesting a role in further investigation of this issue. At this point, data are being collected in an ongoing study of the newer low-permeability Excluder device, which will likely answer the questions regarding material permeability with the original Gore device.

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