The role of aortic neck dilation and elongation in the etiology of stent graft migration after endovascular abdominal aortic aneurysm repair with a passive fixation device

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Objective: **Endovascular repair of abdominal aortic aneurysm (AAA) is complicated by the potential for stent graft migration over time. Factors including the type of fixation, initial proximal fixation length, and dilation and elongation of the infrarenal aortic neck may contribute to device migration. We sought to determine when device migration is a real phenomenon with actual device movement that compromises aneurysm exclusion.**

Methods: **Computed tomographic (CT) scans and computer reconstructions of all patients undergoing endovascular AAA repair with a passive fixation device at our institution from June 1996 to October 2004 were retrospectively reviewed. The distance from the distal renal artery to the proximal end of the stent graft at the time of initial deployment was determined for each patient. Migration was defined as a distance increase greater than 5 mm in the follow-up period; proximal fixation length, aortic neck enlargement and elongation, and neck angle were then measured. Data were further analyzed with respect to AAA growth, development of endoleak, AAA rupture, and the need for reintervention.**

Results: **A total of 308 patients with endovascular AAA repairs using a passive fixation device had complete postoperative imaging data sets; 48 patients (15.6%) with stent graft migration of 5 mm or more were identified, and 25 (8.1%) of these had a migration of 10 mm or more. Seventeen (35.4%) of 48 migration patients had a total loss of the proximal seal zone (loss patients); their average migration distance was 17.7 ± 12.0 mm, with a mean neck shortening of 13.6 ± 14.2 mm, and the average proximal fixation length loss was 14.0 ± 7.6 mm. Those 31 patients with an intact proximal seal zone (nonloss patients) showed an average migration of 9.4 ± 3.7 mm, with a mean neck lengthening of 9.6 ± 8.4 mm and an average proximal fixation length change of 0.7 ± 8.0 mm. Univariate analysis demonstrated significant differences between the loss and nonloss patients in follow-up duration (65.9 ± 20.4 months vs 45.9 ± 26.4 months; $P = .01$), neck dilatation at the distal renal artery (4.6 ± 4.5 mm vs 1.8 ± 1.9 mm; $P = .026$), stent graft migration distance (17.7 ± 12.0 mm vs 9.4 ± 3.7 mm; $P = .001$), change in aortic neck length (−13.6 ± 14.2 mm vs 9.6 ± 8.4 mm; $P < .0001$), change in proximal fixation length (−14.0 ± 7.6 mm vs 0.7 ± 8.0 mm; $P < .0001$), change in AAA size (1.8 ± 7.1 mm vs −3.6 ± 9.7 mm; $P = .033$), and use of a stiff body stent graft (47.1% vs 19.4%; $P = .043$). However, only change in aortic neck length was statistically significant on multivariate analysis (odds ratio, 0.75; 95% confidence interval, 0.591-0.961; $P = .022$). There were no differences between the loss and nonloss patients in time to migration discovery, initial AAA size, initial aortic neck diameter or length, initial device oversizing, initial neck angle, neck angle increase, type II endoleak, or AAA rupture. Eight of the 17 loss patients have been treated with proximal aortic cuffs; the remainder have refused reintervention, died of unrelated causes, or elected to have open repair.**

Conclusions: **Postoperative elongation of the infrarenal aortic neck may create the radiographic perception of migration without necessarily causing a loss of proximal stent graft fixation. Patients with a total loss of the proximal seal zone actually have infrarenal aortic neck shortening, with a degree of neck dilatation beyond initial device oversizing that may compromise proximal fixation length. Conversely, those with an intact proximal seal zone demonstrate aortic neck elongation equivalent to migration, with no loss of proximal fixation length; these patients have a benign natural history without intervention. Thus, aortic neck dilatation beyond oversizing, aortic neck shortening, and loss of proximal fixation length are more clinically relevant predictors of proximal stent graft failure than simple migration distance. (J Vasc Surg 2006;44:1176-81.)**

Abdominal aortic aneurysm (AAA) continues to pose a significant public health risk to the aging population. The patient population encountered with AAA is typically elderly with numerous comorbidities, including diabetes mellitus, coronary artery disease, chronic obstructive pulmonary disease, renal dysfunction, and peripheral vascular disease. This demographic subset results in high-risk surgical patients, particularly for open repair. As an alternative, the feasibility and safety of endovascular AAA repair have been clearly established in trials with several now Food and Drug Administration (FDA)-approved, commercially available devices. Although the endovascular approach affords shorter hospital stays, less postoperative pain, and lower perioperative morbidity and mortality as compared with

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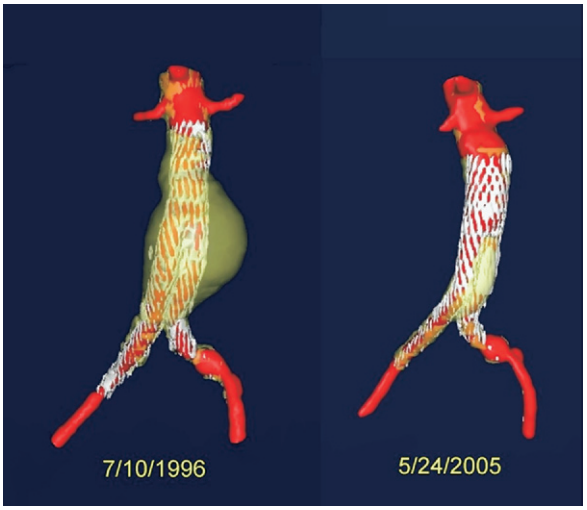

Fig 1. Device migration with aneurysm regression (the intraaortic thrombus has been rendered transparent).

traditional open repair, continual postoperative surveillance is required. Of particular concern is the potential for stent fracture, cloth tears, device migration or dislodgement, endoleak development, loss of stent graft fixation, loss of aneurysm exclusion, aneurysm growth, and aneurysm rupture.[1-3]

Postoperative surveillance is most frequently performed with contrasted computed tomographic (CT) scanning as well as plain film interrogation, with magnetic resonance imaging reserved for patients whose renal function will not tolerate iodinated contrast. The decision for reintervention is complex and involves situations such as development of type I or III endoleak, loss of proximal or distal stent graft fixation, device migration, continued aneurysm expansion with a type II endoleak, or device fatigue with structural failure. Although aneurysm growth and loss of aneurysm exclusion constitute absolute indications for reintervention, the question arises about the clinical significance of stent graft migration without aneurysm reperfusion.

A significant subset of patients is noted to undergo aneurysm regression, sometimes complete, after stent graft repair; however, some of these same patients seem to develop device migration on their postoperative imaging (Fig 1). The aneurysm is gone, but the device seems to be moving caudally. Other patients have less dramatic aneurysm regression, but their devices likewise seem to be migrating. Therefore, the question of reintervention emerges: do these patients require proximal cuff extension of the stent graft? Because the goal of AAA repair is to prevent rupture and death and the likelihood of rupture correlates with aneurysm size, it would seem that the answer is no. However, how do we reconcile stent graft migration with aneurysm shrinkage? It would seem that migration should cause loss of proximal fixation and, thus, loss of aneurysm exclusion leading to growth and potential rupture.

Even in the open vascular literature, it has been recognized that suturing a prosthetic graft in the infrarenal aorta poses a risk of subsequent proximal aortic neck dilation and proximal suture line pseudoaneurysm.[4] The sewn-in graft cannot migrate, yet the infrarenal aortic neck enlarges and elongates, thus creating the appearance of caudal displacement. If this same mechanism were present in endovascular AAA repair, it could explain the appearance of distal stent graft migration without a loss of aneurysm exclusion.

The objective of this study was to determine whether stent graft migration always represents a real phenomenon with actual loss of the proximal stent graft seal zone or whether it is sometimes explained by simple aortic neck elongation. The device used for this study is the AneuRx passive fixation device (Medtronic AVE, Santa Rosa, Calif).

METHODS

A total of 308 patients undergoing endovascular AAA repair by using the AneuRx device at our institution between June 1996 and October 2004 had fine-cut CT imaging data reconstructed as a 3-dimensional model by Medical Metrx Solutions (West Lebanon, NH). The patient population involved had AneuRx stent grafts implanted during phase I, II, and III clinical trials, as well as after FDA approval of the device. We identified 48 (15.6%) of 308 patients who had evidence of stent graft migration of 5 mm or more; 25 (8.1%) of the 308 patients experienced migration of 10 mm or more. Patient imaging data sets were analyzed by comparing their most recent CT scans with their immediate postoperative CT scans. The 8 of 48 migration patients who required proximal aortic cuff placement were analyzed according to the last CT scan before reintervention. The time to initial discovery of migration was noted for life-table analysis, and the total duration of postoperative follow-up was measured. The device oversizing percentage was calculated by using the neck diameter from the preoperative CT scan (30/48 patients) or intraoperative intravascular ultrasonography measurement (18/48 patients).

The proximal seal zone was defined qualitatively as the region of overlap between the stent graft main body and the infrarenal aortic neck and was measured quantitatively as the proximal fixation length. Total loss of the proximal seal zone was defined as a proximal fixation length equal to zero, and migration was determined to be the change in centerline distance from the lowermost renal artery to the top of the stent graft over time (Fig 2). All longitudinal measurements in this study were made in the vessel centerline. Only 2 of the 48 migration patients had total AAA regression; in their cases, the infrarenal neck length was measured from the distal renal artery to the first discernible point where the aortic diameter increased by 10%. In Fig 2, $a = b + c$, where a is the infrarenal aortic neck length, b is distal renal artery to top of stent graft distance, and c is the proximal fixation length. Patients with infrarenal neck angulation had centerline measurements taken from the distal renal artery to the first CT slice with a full-circumference stent graft. Proximal fixation length was measured as the difference between aortic neck length and the distance

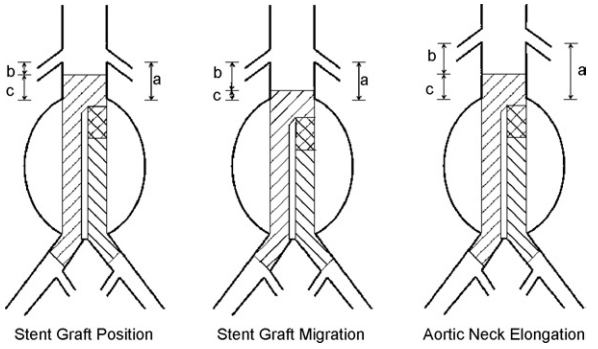

Fig 2. Measuring changes in the infrarenal aortic neck: $a = b + c$, where a is the infrarenal aortic neck length, b is the lowest renal to top of stent graft distance, and c is the proximal fixation length. Δa is aortic neck elongation, Δb is stent graft migration, and Δc is loss of proximal fixation length.

from the distal renal artery to the top of the stent graft ($c = a - b$). In the middle scenario (stent graft migration), if migration is a true phenomenon representing actual downward device displacement without aortic neck elongation (ie, a is constant), the migration distance b should increase by the same amount that the proximal fixation length c decreases. Likewise, if the aortic neck were to shorten (ie, a decreases), then the proximal fixation length c would shorten by the same amount and threaten loss of the proximal seal zone; this problem would be compounded if b also increased (ie, migration occurred). In the far right example (aortic neck elongation), the distance a increases by the same amount as the distance b (renal to stent graft length) without any loss of proximal fixation length (ie, c is constant).

To determine which factors cause stent graft migration with a loss of proximal seal zone, the cohort of 48 migration patients was divided into 2 groups for comparison: 17 patients with total loss of the proximal seal zone (loss patients) and 31 patients with intact proximal seal zones (nonloss patients). For both groups, the aortic neck diameter was measured just below the lowermost renal artery; the change over time of these neck diameters was also determined. Infrarenal aortic neck length, a, distal renal to stent graft distance, b, and proximal fixation length, c, were also measured directly, as were their respective changes over time. Change in a is aortic neck elongation, Δb is stent graft migration, and Δc is loss of proximal fixation length. Initial AAA size, change in size with respect to time, aortic neck angle relative to the AAA, and any increase during follow-up were determined. The initial stent graft deployment position below the renal arteries was also measured. All patient imaging studies were reviewed for endoleak development or resolution. All patients charts were reviewed for documentation of aneurysm growth, need for reintervention, and aneurysm rupture. For purposes of life-table analysis, time to discovery of migration was determined for all patients, as was duration of follow-up. Direct comparison between the two patient subgroups was performed for each of these variables.

To determine whether stent graft migration distance is different from aortic neck elongation length, the two measurements were compared within each patient subgroup. Centerline proximal fixation length and its change over time were measured, and the change was independently compared with the migration distance within each patient subgroup.

All length measurements were taken in the vessel centerline of the three-dimensional Medical Metrx Solutions reconstructions. The results of all continuous data measurements are expressed as means ± SD. Univariate analysis was performed for all variables listed previously. The Student t test was used to analyze continuous data, and the Pearson χ^2 analysis was performed for categorical variables. A P value of $<.05$ was determined to be statistically significant. Multivariate logistic regression analysis was performed on all variables determined to be significant on univariate analysis. Life-table analysis was performed to demonstrate the probability of migration with time.

RESULTS

A total of 308 patients undergoing endovascular AAA repair with the AneuRx device at Harbor-UCLA Medical Center had complete postoperative imaging data sets available. Of these 308 patients, 48 (15.6%) were found to have stent graft migration of 5 mm or more; 25 (8.1%) of these 308 patients had migrations 10 mm or more. Seventeen (35.4%) of the 48 migration patients had complete loss of the proximal seal zone (loss patients), whereas 31 (64.6%) of the 48 had an intact proximal seal zone (nonloss patients).

Loss vs nonloss patients. Comparing patients who did and did not have a total loss of the proximal seal zone (Table), there were statistically significant differences in follow-up duration, stent graft migration distance, change in aortic neck length, change in AAA size, change in aortic neck diameter, use of the stiff body AneuRx stent graft, and change in proximal fixation length. There were no significant differences in time to detection of migration, initial aortic neck length, initial aortic neck diameter, initial postdeployment proximal fixation length, initial AAA size, initial aortic neck angulation or its change over time, degree of stent graft oversizing, stent graft main body diameter, rate of type II or III endoleak, or rate of AAA rupture. The initial stent graft deployment distance below the distal renal artery approached, but did not reach, statistical significance between the loss and nonloss patients. By definition, loss patients developed type I endoleaks and required aortic cuff placement, whereas nonloss patients did not.

Multivariate logistic regression analysis was then performed on variables determined to be significant between loss and nonloss patients on univariate analysis. Follow-up duration, stent graft migration distance, change in aortic neck length, change in proximal fixation length, change in AAA size, change in aortic neck diameter, and use of a stiff body stent graft were entered into the multivariate analysis.

Table. Comparison of AAA characteristics between loss and nonloss patients

Variable	*Loss patients*	*Nonloss patients*	P *value*
Follow-up duration (mo)	65.9 ± 20.4	45.9 ± 26.4	.006
Migration detected (mo)	23.9 ± 10.3	25.6 ± 16.5	.70
Initial distance below distal renal (*b*) (mm)	7.7 ± 10.9	3.1 ± 5.4	.054
Stent graft migration distance (Δ*b*) (mm)	17.7 ± 12.0	9.4 ± 3.7	.013
Initial aortic neck length (*a*) (mm)	21.9 ± 13.2	20.1 ± 7.5	.35
Δ Aortic neck length (Δ*a*) (mm)	-13.6 ± 14.2	9.6 ± 8.4	<.0001
Initial proximal fixation length (*c*) (mm)	14.0 ± 7.6	17.0 ± 7.5	.19
Δ Proximal fixation length (Δ*c*) (mm)	-14.0 ± 7.6	0.7 ± 8.0	<.0001
Initial AAA size (mm)	51.5 ± 8.9	55.9 ± 12.3	.20
Δ AAA size (mm)	1.8 ± 7.1	-3.6 ± 9.7	.033
Initial aortic neck angle (°)	31.7 ± 18.3	35.2 ± 17.4	.51
Δ Aortic neck angle (°)	9.8 ± 13.8	5.9 ± 13.5	.34
Initial aortic neck diameter (mm)	23.2 ± 3.0	22.4 ± 3.1	.38
Δ Aortic neck diameter (mm)	4.6 ± 4.5	1.8 ± 1.9	.026
Stent graft oversizing	12.7% ± 7.9%	14.6% ± 9.0%	.47
Stent graft main body diameter (mm)	26.9 ± 1.6	26.6 ± 2.4	.65
Stiff body stent graft	8	6	.04
Type I endoleak	5	0	.001
Type II endoleak	7	6	.11
Type III endoleak	0	0	NS
Aortic cuff placement	8	0	<.0001
AAA rupture	0	0	NS

AAA, Abdominal aortic aneurysm.

After stepwise regression, the change in aortic neck length was the only variable with a significant association with the loss of the proximal seal zone (odds ratio, 0.75; 95% confidence interval, 0.591-0.961; $P = .022$).

Migration vs elongation and loss of proximal fixation. Within the loss patient subgroup of 17 patients, the stent graft migration distance was significantly different from the change in aortic neck length (17.7 ± 12.9 mm vs −13.6 ± 14.2 mm; $P < .0001$). The migration distance was not significantly different from the loss of proximal fixation length (17.7 ± 12.9 mm vs 14.0 ± 7.6 mm; $P = .30$). In this subgroup, significant infrarenal aortic neck dilatation (4.6 ± 4.5 mm) and shortening (−13.6 ± 14.2 mm) occurred and led to a loss of proximal fixation. Furthermore, this degree of neck dilatation (19.8%) exceeds initial oversizing (12.7% ± 7.9%) and allows for a free-floating proximal main body. Of the 17 loss patients, only 5 had type I endoleaks with visible perigraft blood flow, and the remaining 12 patients showed a main body loosely sealed against intra-aortic thrombus with no perigraft flow.

By contrast, the nonloss patient subgroup of 31 patients demonstrated a migration distance that was nearly identical to the increased aortic neck length (9.4 ± 3.7 mm vs 9.6 ± 8.4 mm; $P = .89$). Migration was significantly different from the loss of proximal fixation length (9.4 ± 3.7 mm vs −0.7 ± 8.0 mm; $P < .0001$). As noted previously, if *c* is constant (as in this subgroup), then the change in *a* is equivalent to a change in *b*; in other words, because there is no change in proximal fixation length, migration is equivalent to aortic neck elongation. This subgroup demonstrated aortic neck dilatation (1.8 ± 1.9 mm, an 8% increase) that was below the degree of initial oversizing (14.6% ± 9.0%). Over a mean follow-up duration of 20 months after discovery of their migration, none of these 31 patients developed a type I endoleak, required reintervention, or experienced AAA rupture. In fact, these patients had a significant trend toward AAA regression.

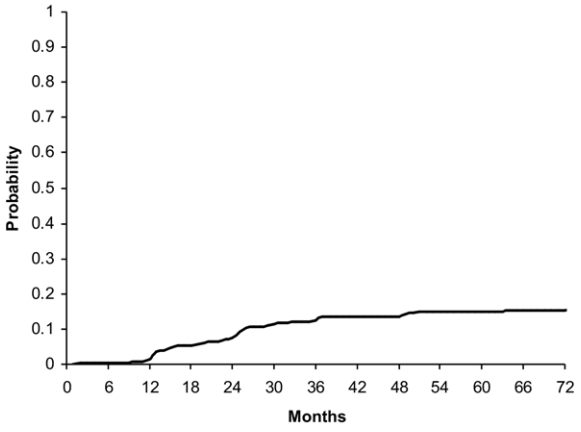

Fig 3. Life-table analysis of the probability of stent graft migration more than 5 mm. There were 223 patients at risk at 12 months, 137 patients at 24 months, 74 patients at 36 months, 30 patients at 48 months, 7 patients at 60 months, and 2 patients at 72 months.

Life-table analysis. Life-table analysis of all 308 patients at risk for stent graft migration is presented in Fig 3. For the various time points, there were 223 patients at risk at 12 months after implantation, 137 patients at 24 months, 74 patients at 36 months, 30 patients at 48 months, 7 patients at 60 months, and 2 patients at 72 months. The probability of migration was 3.6% at 12 months, 9.4% at 24 months, and

13.6% at 36 months, with prominent peaks of detection corresponding to traditional follow-up time points at 1, 2, and 3 years after surgery.

DISCUSSION

Stent graft proximal fixation in the endovascular repair of AAA with a passive fixation device such as the AneuRx is dependent on adequate overlap between native aortic neck tissue and the metallic stent structure of the device. The radial force generated by the expanded stent drives the stent against the aortic wall and prevents slippage. This requires a certain degree of oversizing to provide secure apposition through a friction seal. Over time, the durability of this apposition affords long-term aneurysm exclusion. Of course, this overlap between stent and aorta is compromised by aortic neck thrombus, plaque, insufficient neck length, and high degrees of angulation.

Nonetheless, stent graft migration has been elucidated as one of the potential complications of endovascular AAA repair.[5-7] The mechanisms and etiology of this migration have been extensively studied, although there are fewer data in the literature concerning the clinical significance of this radiographic finding.

Zarins et al[5] published prospective data looking at migration in patients undergoing endovascular AAA repair as part of the multicenter AneuRx clinical trial. In their analysis, a detailed consideration of the potential causative factors for migration was presented. Short proximal fixation length and low initial deployment of the stent graft were identified as risk factors for later migration. Aortic neck length and diameter, aneurysm size, degree of oversizing, use of proximal cuffs at initial implantation, postprocedure endoleak, demographic factors, and comorbid conditions were not shown to be predictive of stent graft migration.

Similarly, Cao et al[6] published data on a series of patients with AneuRx devices implanted to treat AAAs. An analysis of variables potentially causing device migration was also reported. They found that aortic neck dilatation and large aneurysm size were predictors of later migration. Low deployment position, neck angulation, length, diameter, and presence of endoleak were not determined to be significantly predictive.

At our own institution, patients have had the AneuRx stent graft device implanted for endovascular AAA repair during phase I, II, and III of the US multicenter trial, as well as after FDA approval. In reviewing our cumulative database, 308 patients with complete postoperative imaging data sets were identified. We noted 48 of 308 patients with device migrations of 5 mm or more, of which 25 patients demonstrated migration of 10 mm or more. In this migration group, 17 (35.4%) of 48 experienced a total loss of the proximal seal zone and were referred to as loss patients. Proximal seal zone failure was determined radiographically by the lack of complete apposition of the proximal stent with the aortic neck or by the presence of proximal perigraft blood flow. Thirty-one (64.6%) of 48 patients demonstrated intact proximal seal zones and were referred to as nonloss patients. The following question then arose: do the nonloss patients simply have intermediate degrees of loss of proximal fixation length, or is there another mechanism producing the appearance of migration without any actual loss of proximal fixation length? The latter notion was supported by the observation (Fig 1) that a certain number of patients had total disappearance of their AAA, yet the stent graft seemed to be migrating. In such patients, in whom the aneurysm is gone, there would be no indication to intervene with a proximal aortic cuff. However, in patient with intermediate degrees of AAA regression, the answer is not so simple. In other words, when is stent graft migration a clinically significant event necessitating surgical reintervention?

From the open vascular literature, it is well known that even after open AAA repair with a sutured aortic graft, the infrarenal neck is likely to continue enlarging in both length and diameter over time.[4] This causes caudal displacement of the proximal suture line and may predispose to the development of proximal suture line pseudoaneurysm. Perhaps a similar phenomenon may be occurring in stent graft repair of AAA, particularly in patients with low initial deployment of the stent graft, thus subjecting the neck to further biomechanical deterioration.[8]

To determine which factors may render migration a true and clinically significant event, we compared critical aneurysm and clinical characteristics between loss and nonloss patients. Patients with migration who lose their proximal seal zone (loss patients) are those who have the longest follow-up, a low initial stent graft deployment, subsequent aortic neck dilatation and shortening, and ultimately loss of all proximal fixation length. It is possible that low initial deployment allows deterioration of the remaining uncovered neck with dilatation and effective shortening. In loss patients, the degree of aortic neck dilatation was found to be significantly greater than the initial device oversizing, thus allowing for a free-floating main body of the stent graft. Time to migration detection, initial aortic neck length, initial proximal fixation length, initial AAA size, initial aortic neck angle and its change, initial aortic neck diameter, degree of stent graft oversizing, rate of type II and III endoleaks, and stent graft main body diameter were no different in loss vs nonloss patients and do not help predict who will lose their proximal seal zone.

The loss subgroup were also the earlier patients in our experience, many of whom had the early stiff body AneuRx device implanted. It is possible that this device was unable to tolerate postoperative conformational changes in the aneurysm sac, which in turn may have compromised its ability to maintain a secure proximal seal. Of course, these loss patients developed type I endoleaks, and all required proximal aortic cuff placement. Eight patients received cuffs, and the remaining nine patients died of unrelated causes, refused reoperation, or had elective open AAA repair. No loss patient experienced AAA rupture.

By contrast, the nonloss subgroup of patients demonstrated a lesser overall degree of migration, but with no loss in proximal fixation length. To elucidate the other mechanism that may be causing the appearance of migration

within the nonloss subgroup, stent graft migration was compared with aortic neck elongation as well as with loss of proximal fixation length. Figure 2 displays these two distinct phenomena. In the first aneurysm on the left, the stent graft has been deployed a distance *b* below the distal renal artery in a neck that is *a* in length. Thus, *c* becomes the proximal fixation length, as the difference between *a* and *b*. If stent graft migration is a true phenomenon with actual slippage of the device downward within the aortic neck, then the increase in *b* over time should equal the loss of *c*, assuming no change in *a* (although in the loss subgroup of patients, aortic neck length *a* actually decreases while migration distance *b* increases, thus resulting in the proximal fixation length *c* going to 0). If, however, aortic neck elongation indeed occurs (*a* increases), *b* may increase (creating the appearance of migration) without any loss of proximal fixation length *c*. In this circumstance, the increase in *b* should equal the increase in *a* (aortic neck length) without any significant change in *c*. This example is precisely what we see in the nonloss subgroup of patients. If stent graft migration has occurred, but without loss of proximal fixation length, it can be explained only by aortic neck elongation.

As for the question of whether complete AAA regression may explain aortic neck elongation, only 2 (4.2%) of the 48 migration patients demonstrated total aneurysm collapse; most patients had much more modest degrees of shrinkage, and some aneurysms actually grew. If aneurysm regression were the sole explanation for neck elongation, we would expect that proximal fixation length would increase by an amount equal to the elongation length. This was not the case in either the loss or nonloss patient groups.

Clinically, none of the 31 nonloss patients experienced type I endoleak, a need for aortic cuff placement, or AAA rupture during the average of 20 months' follow-up after initial migration discovery. One patient later required endovascular treatment for development of a large hypogastric artery aneurysm, obviously unrelated to a discussion about migration. In fact, these nonloss patients actually had a significant trend toward AAA regression. Thus, patients showing stent graft migration due to aortic neck elongation without a loss of proximal fixation length have a benign natural history that does not warrant aortic cuff placement. However, continued close surveillance is indicated.

CONCLUSION

Stent graft migration without a loss of proximal fixation length may simply represent infrarenal aortic neck elongation. Migration becomes clinically significant when there is a concurrent loss of proximal fixation length, which occurs when the aortic neck shortens and dilates beyond the degree of device oversizing; these patients require aortic cuff placement. Migration patients without a loss of proximal fixation length have a benign natural history that does not warrant reintervention. Therefore, we suggest that increased infrarenal aortic neck diameter beyond oversizing, infrarenal aortic neck shortening, and loss of proximal fixation length are more clinically relevant predictors of proximal stent graft failure than simple migration distance alone.

AUTHOR CONTRIBUTIONS

Conception and design: RAL, CED, RAW
Analysis and interpretation: RAL, CED, RAW
Data collection: RAL, RAW
Writing the article: RAL, RAW
Critical revision of the article: RAL, CED, SLC, RAW
Final approval of the article: RAL, CED, RAW
Statistical analysis: SLC, RAW
Overall responsibility: RAL

REFERENCES

1. Zarins CK, White RA, Moll FL, Crabtree T, Bloch DA, Hodgson KJ, et al. The AneuRx stent graft: four-year results and worldwide experience 2000. J Vasc Surg 2001;33(2 Suppl):135-45.
2. Zarins CK. AneuRx Clinical Investigators. The US AneuRx clinical trial: 6-year clinical update 2002. J Vasc Surg 2003;37:904-8.
3. Lifeline Registry of EVAR Publications Committee. Lifeline registry of endovascular aneurysm repair: long-term primary outcome measures. J Vasc Surg 2005;42:1-10.
4. Curl GR, Faggioli GL, Stella A, D'Addato M, Ricotta JJ. Aneurysmal change at or above the proximal anastomosis after infrarenal aortic grafting. J Vasc Surg 1992;16:855-60.
5. Zarins CK, Bloch DA, Crabtree T, Matsumoto AH, White RA, Fogarty TJ. Stent graft migration after endovascular aneurysm repair: importance of proximal fixation. J Vasc Surg 2003;38:1264-72.
6. Cao P, Verzinin F, Zannetti S, De Rango P, Parlani G, Lupatteli L, et al. Device migration after endoluminal abdominal aortic aneurysm repair: analysis of 113 cases with a minimum follow-up period of 2 years. J Vasc Surg 2002;35:229-35.
7. Conners MS, Sternbergh WC, Carter G, Tonnessen BH, Yoselevitz M, Money SR. Endograft migration one to four years after endovascular abdominal aortic aneurysm repair with the AneuRx device: a cautionary note. J Vasc Surg 2002;36:476-84.
8. Matsumura JS, Chaikof EL. Continued expansion of aortic necks after endovascular repair of abdominal aortic aneurysms. J Vasc Surg 1998;28:422-31.

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