Post–Endovascular Aneurysm Repair Rupture Rate Can Be as Low as 0.5%; Is It the Result of Treating More Small Abdominal Aortic Aneurysms? Maybe They Need No Treatment at All

Dieter Raithel, MD, PhD, Nuernberg, Germany; Lefeng Qu, MD, PhD, Nuernberg, Germany; Gudrun Hetzel, MD, PhD, Nuernberg, Germany

Background

The natural history of abdominal aortic aneurysms (AAAs) is marked by progressive expansion and final rupture, which results in considerable morbidity and mortality as well as significant costs to family and society.[1] This is the reason why AAAs should be treated and, accordingly, the postoperative rupture rate should be the most important end point for evaluation of treatment effectiveness. We researched 10 years of experience with endovascular aneurysm repair (EVAR)—1,064 consecutive infrarenal AAAs with a post-EVAR rupture rate of only 0.5%, which is much lower than the reported post-EVAR rupture rate of 1.6 to 10%.[2,3]

Frank Veith asked, "Post-EVAR rupture rate can be as low as 0.5%; is that the result of treating more small AAAs? Maybe they need no treatment at all?" To answer the above question, we reviewed our data and the related references.

Methods

Data for 1,064 consecutive infrarenal patients with AAA with EVAR performed within 10 years, ending May 2005, were analyzed retrospectively. Mean follow-up was 33 months (range 0.2 to 88 months). All patients received commercially available, CE-approved devices. The 11 different grafts included Ancure® (Guidant-EVT, Menlo Park, CA), AneuRX® (Medtronic Sunrise, FL), Chuter® (Meadox, NJ), Endofit® (Le Maitre, Burlington, MA), Powerlink (Endologix®, Irvine, CA), Excluder® (W.L. Gore & Associates, Flagstaff, AZ), Fortron® (Cordis, Miami, FL), Lifepath® (Edwards, Irvine, CA), Talent® (Medtronic, Sunrise, FL), Vanguard/Mintec® (Boston Scientific, Natick, MA), and Zenith® (Cook, Bloomington, IN).

Nine hundred sixteen (86.1%) patients received an endograft of bifurcated configuration, and 148 (13.9%) patients received a tube endograft. To evaluate the influence of size on the outcome of EVAR, the study cohort was subdivided into two groups according to the preoperative aneurysm diameter: small AAA group (EVARsmall), with an aneurysm diameter < 5 cm; and big AAA group (EVARbig), with an aneurysm diameter ? 5 cm.

Inclusion criteria included all the consecutive 1,064 AAAs treated with EVAR, including ruptured AAAs managed by EVAR. Baseline data including age, gender, comorbidities, and smoking history; anatomic aspects; and operative details such as bleeding amount, operative time length, primary endoleak, and conversion were recorded (Table 1). Findings at the follow-up visits, which involved clinical examination and computed tomography (CT), angiography, or magnetic resonance angiography, were recorded on data forms and kept in the data room. Follow-up visits were scheduled at 14 days, 12 months, and annually thereafter. Deaths that occurred within 30 days of the initial procedure were recorded as a perioperative death, and those after 30 days as a late death.

Table 1. Baseline Characteristics in the Study Cohort

Total Cohort	EVARsmall	EVARbig
Mean age (yr)	70.7	69.7	65.9
Gender ratio (male:female)	983:81	496:53	487:28
Has smoked at some time	260	149	111
History of cardiac symptoms	195	120	75
Hypertension	176	97	79
COPD	73	39	34
Diabetes	69	39	30
Renal insufficiency	23	14	9
Mean AAA diameter (cm)	4.9	4.3	5.8

AAA = abdominal aortic aneurysm; COPD = chronic obstructive pulmonary disease.

Other outcome events observed during the follow-up included endoleak, device migration, limb occlusion, and severe device kinking. Post-EVAR rupture was documented in detail on the data forms. Results were reported as mean, range, and percentage of patients with discrete variables, unless otherwise specified.

Results

One thousand sixty-four consecutive infrarenal patients with AAA, 983 men and 81 women, ranged in age from 42 to 98 years. The average diameter of the AAAs was 4.9 cm (range 2.8 to 10 cm). The EVARsmall group included 549 patients (51.6%) with diameters < 5 cm (range 2.8 to 4.9 cm); the EVARbig group included 515 patients (48.4%) with diameters ? 5 cm (range 5.0 to 10.0 cm). The average ages in the EVARbig and EVARsmall groups were 65.9 and 69.7 years, respectively. Dilatation of the common iliac arteries was more frequent in the EVARbig group. The incidence of primary endoleaks in the whole cohort was 13.8%. There was a slightly higher incidence of primary endoleaks in the EVARbig group compared with the EVARsmall group (15.5% vs 12.2%), but there was no significant deviation (Table 2). The incidence of secondary endoleaks for the whole cohort was 11.6% in total (11.6% and 11.4% in the EVARsmall and EVARbig groups, respectively). Primary or first-month conversion to open repair was performed in 39 patients (3.7%), 25 (4.9%) in the EVARbig group and 14 (2.6%) in the EVARsmall group. Limb occlusion occurred in 59 cases (5.5%): 35 cases (6.4%) in the EVARsmall group and 24 cases (4.7%) in the EVARbig group. Renal infarction at the follow-up with CT was found in 97 cases (9.1%): 46 cases (8.9%) in the EVARbig group and 51 cases (9.3%) in the EVARsmall group.

The overall perioperative mortality was 16 patients (1.5%) in total, 10 patients (1.9%) in the EVARbig group and 6 (1.1%) in the EVARsmall group. Rupture post-EVAR occurred in 6 patients in the entire study cohort, with 6 (0.5%) ruptures in the EVARbig group and no ruptures in the EVARsmall group.

Multivariate analysis indicated that in patients with large aneurysms, age, pulmonary comorbidity, and smoking are factors with an independent correlation in post-EVAR rupture.

Table 2. Post-EVAR Results

Total Cohort	EVARsmall	EVARbig
Primary endoleak		
Type I distal	29	13	16
Type I proximal	36	16	20
Type II	65	31	34
Type III	9	4	5
Type IV	5	1	4
Types I + II	3	2	1
Secondary endoleak		
Type I distal	22	15	7
Type I proximal	41	22	19
Type II	42	21	21
Type III	6	1	5
Type IV	4	1	3
Types I + II	4	2	2
Types II + III	2	2	0
Types I + III	2	0	2
Renal infarction	97	51	46
Conversion ( )	85	31	54
Early conversion (30 d)	39	14	25
Occlusion of limb	59	35	24
Post-EVAR rupture	6	0	6
Total mortality	67	23	44
Early mortality (30 d)	16	6	10

Discussion

From the above data, everyone can answer the first question from Frank Veith: Yes, the post-EVAR rupture rate in total can be as low as 0.5% as a result of treating smaller AAAs. We are treating approximately 50% of small AAAs, and that could be the reason for a good therapeutic result. But the response to Frank Veith's second question of whether treatment is needed is quite controversial. The annual risk of rupture exceeds the elective operative mortality in the absence of significant coexisting comorbidity. Therefore, in large AAAs with the diameter of 5.0 cm (or 5.5 cm) or more, most vascular surgeons would recommend intervention (open or endovascular). This fact can lead to the debate focus: why should we treat AAAs? The answer is simple: to prevent rupture and death.

If the AAA will not rupture in the patient's remaining lifetime, it certainly needs no treatment at all. So then, how do you know whether the AAA is going to rupture in a specific patient? The current standard for estimating rupture risk is a maximum AAA diameter, but this standard is clearly not ideal. Small AAAs can and do rupture even with frequent and reliable surveillance.[4–9] Assuming that most vascular surgeons have the experience (skills) to treat ruptured small AAAs, the hot topic arises again: should small AAAs be treated?

As with larger AAAs, the risks between natural rupture rate and elective operative mortality should be considered. At the recent international vascular surgery meetings, this has been one of the most debated controversies. There are always two sides: for or against the timely treatment of small AAAs.

Those in favor of treating small AAAs hold the following opinions:

- It is a fact that small AAAs can and do rupture in the hospital. Cronenwett and colleagues reported that patients with 3 cm AAAs and the presence of significant pulmonary disease as well as arterial hypertension had a rupture risk of 54% within 3 years of the diagnosis.[6] Katz and colleagues estimated a 3.3% per annum rupture risk of aneurysms < 4 cm in diameter.[10] In a postmortem examination of 265 aneurysm patients, Darling and colleagues found that 12.8% of ruptured aneurysms were smaller than 5 cm.[4]
- Small AAAs continue to expand over time. Watson and colleagues assessed the median annual growth rate of very small AAAs by ultrasonography at intervals of 6 to 12 months under long-term surveillance.[11] The results showed that during the follow-up period, the median annual growth rate for aneurysms < 3.0 cm was 1 mm. The growth rate increased to 2 mm when the aneurysm was between 3.0 and 3.9 cm. It continued to rise to a 3 mm growth rate when the aneurysm was between 4.0 and 4.9 cm in diameter.
- As small aneurysms continue to grow, if we follow up these patients conservatively, we may risk missing the time for an elective operation owing to unexpected rupture. However, this can also occur with close follow-ups. The effectiveness of surveillance in preventing aneurysm rupture has not been established. Despite close monitoring and early surgery when indicated, ruptures occurred in the surveillance group of both prospective clinical trials.[8,9,12] Federal estimates indicate that more than 15,000 deaths owing to aneurysm rupture occur each year in the United States.[13] At least 62% of patients who experience the rupture of an AAA have been estimated to die prior to reaching the hospital.[14]
- While waiting for aneurysms to grow larger, the patients also grow older and get additional or more severe comorbidities, such as coronary artery disease or respiratory disease. A multivariate analysis from EUROSTAR demonstrated that aneurysm size, patient age, renal insufficiency, and pulmonary comorbidity are factors with an independent correlation with increased risk for aneurysm-related death.[3] Undoubtedly, higher ages of patients with larger aneurysms will result in higher mortality.
- During surveillance some AAAs have changed in morphology and become unsuited for EVAR, resulting in only one or no choice for open surgery.[3,15]
- The perioperative mortality of small ruptured AAAs is not different from that of large ruptured AAAs.
- At a given AAA diameter, women are at a higher risk of AAA rupture than are men.[2,3] Fillinger and colleagues demonstrated that the average diameter of ruptured AAAs in women was 5 mm smaller than in men.[16]

Those who are against the treatment of small AAAs consider that the surgical risk is higher than the natural rupture rate. Although debated fiercely, a definite agreement has not been reached; however, all parties agreed to wait for the results of the United Kingdom Small Aneurysm Trial.[8,9] These two prospective, randomized clinical trials of good-risk patients with AAA diameters of < 5.5 cm found no difference in the overall survival rates between patients treated with early elective surgical repair and those followed up with ultrasonographic surveillance, but these results are comparing elective open surgical repair and ultrasonographic surveillance.[8,9,12] The total rupture rate of the surveillance group in the UK Small Aneurysm Trial is 5.1%.[9] But the total post-EVAR rupture rate in our group is only 0.5%, and even 0% in EVARsmall group. What is the reason for that great difference? The answer lies in EVAR.

It is a fact that EVAR has gained in acceptance in the clinical management of infrarenal AAA since its introduction in the early 1990s.[17] Endovascular repair compares favorably to open surgical repair in short and mid-term analyses,[18–21] and it may have long-term benefits with reduced aneurysm-related death.[22] Two recent prospective, randomized clinical trials comparing EVAR

NOTES

to open surgical repair found a three- to fourfold reduction in the 30-day operative mortality in patients undergoing EVAR.[23,24] Therefore, the concept of EVAR as a method of choice not only in patients with high-risk AAAs but also in other, carefully selected patients has been gradually accepted. Favorable results with EVAR have also been demonstrated by other authors in patients with small aneurysms.[2,3,25] Zarins and colleagues compared the outcome of patients with small AAAs treated in a prospective trial of EVAR with patients randomized to the surveillance arm of the UK Small Aneurysm Trial.[2] The patients in Zarins and colleagues' group had a higher age, more comorbidities, and a higher risk than did those in the UK group, but after statistic adjustment, the former group still achieved a better result, with a total rupture rate and total mortality of 1.6% versus 5.1% and 18% versus 48%, respectively.

Our results are even better. Therefore, the answer to Professor Frank Veith's second question should be addressed in two steps: small AAAs need treatment, and when they are treated, they should be treated with endovascular techniques.

Conclusion

The answers to Frank Veith's questions are quite clear now:

Yes, we are treating more small AAAs with a good result and small AAAs should be treated in time with EVAR as the first therapeutic choice.

References

1. Reilly JM, Tilson MD. Incidence and etiology of abdominal aortic aneurysms. Surg Clin North Am 1989;69:705–11.
2. Zarins CK, Crabtree T, Arko FR, et al. Endovascular repair or surveillance of patients with small AAA. Eur J Vasc Endovasc Surg 2005;29:496–503.
3. Peppelenbosch N, Buth J, Harris PL, et al. Diameter of abdominal aortic aneurysm and outcome of endovascular aneurysm repair: does size matter? A report from EUROSTAR. J Vasc Surg 2004; 39:288–97.
4. Darling RC, Messina CR, Brewster DC, Ottinger LW. Autopsy study of unoperated abdominal aortic aneurysms: the case for early resection. Circulation 1977;56 Suppl 3:161–4.
5. Brown LC, Powell JT. Risk factors for aneurysm rupture in patients kept under ultrasound surveillance. UK Small Aneurysm Trial participants. Ann Surg 1999;230:289–96; discussion 296–7.
6. Cronenwett JL, Murphy TF, Zelenock GB, et al. Actuarial analysis of variables associated with rupture of small abdominal aortic aneurysms. Surgery 1985;98:472–83.
7. Nicholls SC, Gardner JB, Meissner MH, Johansen HK. Rupture in small abdominal aortic aneurysms. J Vasc Surg 1998;28:884–8.
8. United Kingdom Small Aneurysm Trial participants. Mortality results for randomized controlled trial of early elective surgery or ultrasonographic surveillance for small abdominal aortic aneurysms. The UK Small Aneurysm Trial participants. Lancet 1998;352:1649–55.
9. Lederle FA, Wilson SE, Johnson GR, et al. Immediate repair compared with surveillance of small abdominal aortic aneurysms. N Engl J Med 2002;346:1437–44.
10. Katz DA, Littenberg B, Cronenwett JL. Management of small abdominal aortic aneurysms. Early surgery vs watchful waiting. JAMA 1992;268:2678–86.
11. Watson CJE, Walton J, Shaw E, et al. What is the long-term outcome for patients with very small abdominal aortic aneurysms? Eur J Vasc Endovasc Surg 1997;14:299–304.
12. United Kingdom Small Aneurysm Trial participants. Long-term outcomes of immediate repair compared with surveillance of small abdominal aortic aneurysms. N Engl J Med 2002;346:1445–52.
13. Faries PL, Dayal R, Lin S, et al. Endovascular stent graft selection for the treatment of abdominal aortic aneurysms. J Cardiovasc Surg 2005;46:9–17.
14. Johansson G, Swedenborg J. Ruptured abdominal aortic aneurysms: a study of incidence and mortality. Br J Surg 1986;73:101–3.
15. Kapma MR, Verhoeven ELG, Tielliu IFJ, et al. Endovascular treatment of acute abdominal aortic aneurysm with a bifurcated stentgraft. Eur J Vasc Endovasc Surg 2005;29:510–5.
16. Fillinger MF, Rasusin J, Baker RK, et al. Anatomic characteristics of ruptured abdominal aortic aneurysm on conventional CT scans: implications for rupture risk. J Vasc Surg 2004;39:1243–52.
17. Parodi JC, Palmaz JC, Barone HD. Transfemoral intraluminal graft implantation for abdominal aortic aneurysms. Ann Vasc Surg 1991;5:491–9.
18. Moore WS, Kashyap VS, Vescera CL, Quinones--Baldrich WJ. Abdominal aortic aneurysm—a 6-year comparison of endovascular versus transabdominal repair. Ann Surg 1999;230:298–306.
19. Matsumura JS, Brewster DC, Makaroun MS, Naftel DC, for the Excluder Bifurcated Endoprosthesis Investigators. A multi-center controlled clinical trial of open versus endovascular treatment of abdominal aortic aneurysm. J Vasc Surg 2003;37:262–71.
20. Zarins CK, White RA, Moll FL, et al. The Aneur stent graft: four-year results and worldwide experience 2000. J Vasc Surg 2001;33 Suppl 2:S135–45.
21. Zarins CK, Heikkinen MA, Lee ES, et al. Short and long-term outcome following endovascular aneurysm repair. How does it compare to open surgery? J Cardiovasc Surg 2004;45:321–33.
22. Arko FR, Lee WA, Hill BB, et al. Aneurysm-related death: primary endpoint analysis for comparison of open and endovascular repair. J Vasc Surg 2002;36:297–304.
23. Greenhalgh RM, Brown LC, Kwong GP, et al. Comparison of endovascular aneurysm repair with open repair in patients with abdominal aortic aneurysm (EVAR trial 1), 30-day operative mortality results: randomized controlled trial. Lancet 2004;364:843–8.
24. Prinssen M, Verhoeven EL, Buth J, et al. A randomized trial comparing conventional and endovascular repair of abdominal aortic aneurysms. N Engl J Med 2004;351:1607–18.
25. Ouriel K, Srivastava SD, Sarac TP, et al. Disparate outcome after endovascular treatment of small versus large abdominal aortic aneurysm. J Vasc Surg 2003;37:1206–12.

NOTES