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Early and delayed rupture after endovascular abdominal aortic aneurysm repair in a 10-year multicenter registry

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Abstract

Objective—Rupture after abdominal endovascular aortic aneurysm repair (EVAR) is a function of graft maintenance of the seal and fixation. We describe our 10-year experience with rupture after EVAR.

Methods—From 2000 to 2010, 1736 patients with abdominal aortic aneurysm (AAA) from 17 medical centers underwent EVAR in a large, regional integrated health care system. Preoperative demographic and clinical data of interest were collected and stored in our registry. We retrospectively identified patients with postoperative rupture, characterized as “early” and “delayed” rupture (≤30 days and >30 days after the initial EVAR, respectively), and identified predictors associated with delayed rupture.

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Results—The overall follow-up rate was 92%, and the median follow-up was 2.7 years (interquartile range, 1.2–4.4 years) in these 1736 EVAR patients. We identified 20 patients with ruptures; 70% were male, the mean age was 79 years, and mean AAA size at the initial EVAR was 6.3 cm. Six patients underwent initial EVAR for rupture (n = 2) or symptomatic presentation (n = 4). Of the 20 post-EVAR ruptures, 25% (five of 20) were early, all occurring within 2 days after the initial EVAR. Of these five patients, four had intraoperative adverse events leading directly to rupture, with one type I and one type III endoleak. Of the five early ruptures, four patients underwent endovascular repair and one received repair with open surgery, resulting in two perioperative deaths. Among the remaining 15 patients, the median time from initial EVAR to rupture was 31.1 months (interquartile range, 13.8–57.3 months). Most of these delayed ruptures (10 of 15) were preceded by AAA sac increases, including three patients with known endoleaks who underwent reintervention. At the time of delayed rupture, nine of 15 patients had new endoleaks. Among all 20 patients, six patients did not undergo repair (all delayed patients) and died, nine underwent repeated EVAR, and five had open repair. For patients who underwent repair for delayed rupture, mortality at 30 days and 1 year were 44.4% and 66.7%, respectively. Multivariable Cox regression analysis identified age 80 to 89 (hazard ratio, 3.3; 95% confidence interval, 1.1–9.4; P =.03), and symptomatic or ruptured initial indication for EVAR (hazard ratio, 7.4; 95% confidence interval, 2.2–24.8; P < .01) as significant predictors of delayed rupture.

Conclusions—Rupture after EVAR is a rare but devastating event, and mortality after repair exceeds 60% at 1 year. Most delayed cases showed late AAA expansion, thereby implicating late loss of seal and increased endoleaks as the cause of rupture in these patients and mandating vigilant surveillance.

Endovascular aortic aneurysm repair (EVAR) was first described[1] in 1991 and has since become the standard of care for treatment of abdominal aortic aneurysm (AAA). Multiple trials have shown excellent short-term outcomes of EVAR compared with traditional open AAA repair,[2–7] but the superiority of long-term results has yet to be determined. Graft durability remains a chief concern, and lifelong radiographic surveillance has been considered mandatory to detect treatable complications such as endo-leak, device migration, and aneurysm expansion.

AAA rupture is a dreaded but known complication after EVAR that can occur in the immediate perioperative period or after a delay. Aneurysm rupture after EVAR might occur because of technical error or the inability of devices to accommodate changes in anatomy over time, or might be due to graft material fatigue leading to failure. Although rare, the incidence of aneurysm rupture does not appear to have changed significantly since EVAR was introduced, and AAA rupture after EVAR continues to carry substantial associated morbidity and mortality.[8–13]

We have previously described our experience with EVAR based on a large, multicenter registry over a 10-year period.[14] The purpose of the current study was to characterize early and delayed rupture after EVAR and to identify factors associated with delayed rupture after EVAR in the community setting.

METHODS

Kaiser Permanente Northern California (KPNC) is a large, integrated health care delivery system caring for more than 3 million people who are broadly representative of the local and statewide population. The KPNC Institutional Review Board approved a retrospective review of 1736 EVARs performed by clinicians from 17 KPNC medical centers from 2000 to 2010 with waiver of consent. Relevant clinical data were prospectively collected by trained research nurses, with December 31, 2010, as the last follow-up date. Baseline preoperative demographic and clinical characteristic data including sex, age, race and/or ethnicity, AAA sac size (hereafter termed aneurysm size), comorbidities, smoking status, and statin history were collected from digitized health records. Device type and operative details were collected from the operative report and device entry forms. Decisions regarding indications for surgery, suitability for endovascular repair, device type, and need for secondary intervention were made at the discretion of the operating surgeon. Data from the follow-up period such as rupture, aneurysm size, endoleak, reintervention, and mortality were also recorded in our registry.

Postoperative surveillance varied across medical centers (no standardized post-EVAR protocol existed during the study period); however, patients generally received a computed tomography (CT) scan 1 month postoperatively and then at regular intervals (usually every 6 to 12 months depending on the clinical scenario). EVAR in patients who presented with ruptured or symptomatic aneurysms at the preoperative CT scan was characterized as “urgent.” All other EVAR was categorized as “elective.” Detailed medical record review was conducted by the study investigators to confirm the patients who had a rupture event after the initial EVAR and their clinical characteristics of interest, including history of endoleak, aneurysm size, intervention before rupture, types of repair for rupture, and complications (eg, cardiac, pulmonary, renal, gastrointestinal, infectious) after rupture.[15]

Our primary outcome measures were the incidence and timing of rupture after the initial EVAR. Rupture after initial EVAR was categorized into two groups: early and delayed. Early ruptures were those that occurred in the immediate perioperative period (within 30 days) after initial EVAR; other ruptures were categorized as delayed. Secondary outcomes included change in aneurysm size over time, presence and type of endoleak, and the need for additional intervention.

Statistical methods

Differences in age and aneurysm size at the initial EVAR were compared between the early and delayed rupture groups using the *t*-test. Comparisons of demographic and clinical variables including sex, treatment of rupture, aneurysm size expansion at the time of rupture, and overall mortality at 30 days and 1 year between the early and delayed rupture groups were evaluated using χ^2 tests or Fisher exact tests. Before determining the potential risk factors associated with delayed rupture, we performed a bivariate analysis comparing the delayed rupture group and patients without rupture (henceforth called “no rupture group”) using χ^2 tests or Fisher exact tests for categorical variables (demographic: sex; at the initial EVAR: age groups, urgent vs elective initial EVAR, comorbidities, history of statin treatment, and smoking status; intraoperative: bifurcated graft, adjunctive maneuver, and

endoleak) and nonparametric Wilcoxon Mann-Whitney tests for nonnormally distributed continuous variables (age and aneurysm size at the initial EVAR). Because of the small number of patients with delayed rupture, a stepwise Cox proportional hazards model was used to identify risk factors of delayed rupture in 15 patients compared with 1716 with no rupture. The significance level to enter and remain in the model was set at $P < .05$. All analyses were performed using SAS 9.3 (SAS Institute Inc, Cary, NC) with the threshold of significance set at $P < .05$.

RESULTS

During the study period, 1736 EVARs were performed. The overall follow-up rate was 92%, and the median follow-up was 2.7 years (interquartile range [IQR], 1.2–4.4 years). Twenty cases of rupture after EVAR (1.2%) were identified, including 5 that occurred within 30 days (“early” rupture) and 15 cases occurring after 30 days (“delayed” rupture).

Seventy percent of ruptures occurred in male patients. In patients with ruptures, mean age and aneurysm size at the time of initial EVAR were 79 ± 9.1 years and 6.3 ± 0.7 cm, respectively (Table I). Six patients underwent initial EVAR for urgent repair, including two for ruptured AAA and four for symptomatic AAA; the remaining 14 patients had elective repair (Table I).

Demographic characteristics and complications of patients with early ruptures

Three of the five patients with early rupture were male, the mean age of patients was 78.2 ± 10.8 years, and the mean aneurysm size was 6.0 ± 0.6 cm at the time of initial EVAR (Table I). All five cases were performed in the second half of the study period (2006–2010). All of the ruptures occurred within 2 days of the initial EVAR. Two of these patients were noted to have intraoperative endoleaks (one type I and one type III; Table II), which were successfully treated during the initial EVAR with additional angioplasty and cuff placement, respectively. Two patients died within 30 days of rupture, of which one patient had a known ruptured AAA at the time of EVAR repair; the other was taken back to the operating room on the day of EVAR and found to have a large type I endoleak which was treated with angioplasty. All five patients experienced significant postoperative complications (Table III).

Demographic characteristics and complications of patients with delayed ruptures

Among the remaining 15 patients, the median time from initial EVAR to rupture was 31.1 months (IQR, 13.8–57.3 months). The mean age of these patients at the initial EVAR was 79.2 ±8.9 years, initial mean AAA size was 6.4 ± 0.7 cm, and 73% were male (Table I). One patient had a known ruptured AAA, and three patients underwent initial EVAR for symptomatic indications. Three patients had unplanned adjunctive maneuvers performed at the time of initial EVAR, including a renal snorkel/cuff placement for a type I leak, an iliac stent for an arterial dissection, and an aortic cuff for a type I leak.

Known previous endoleak and/or reintervention

As shown in Table IV, three of the 15 patients with delayed rupture had known persistent endoleaks before rupture (one patient with a presumed type I endoleak, two patients with presumed type II endoleaks). All three patients underwent prerupture interventions. The first patient had a possible type I vs type II leak seen on CT scan and underwent a diagnostic arteriogram that confirmed a type Ia leak with type II leak outflow. The patient declined further intervention and shortly after presented with a fatal, untreated rupture. The second patient had a persistent type II leak with sac growth despite coil embolization of lumbar vessels and subsequent direct sac injection of cyanoacrylate glue and embolization coils. On open exploration after rupture, she was found to have a type III leak and died intraoperatively. The third patient presented with a stable rupture from a presumed type II leak. On open exploration, slight suture-hole graft bleeding was noted, and a large inferior mesenteric artery was ligated, because this was presumed to be the causative agent. The patient was subsequently re-explored for persistent hemodynamic instability, and the endograft was relined to treat a presumed type III leak. The patient had satisfactory results from a surgical standpoint but subsequently died 40 days after initial rupture because of multiorgan failure.

Endoleak and aneurysm size at the time of rupture

At the time of delayed rupture, 12 of 15 patients were found to have endoleaks (six type I leaks, two type I/II, one type I/III, one type II/III, two type III; Table IV). Of the 12 patients with endoleaks at the time of rupture, nine leaks occurred in patients in whom endoleaks had not been identified before rupture. Three of these newly diagnosed leaks occurred in the setting of graft migration.

The remaining three patients did not have confirmed endoleaks at the time of rupture. Of these three, one patient was demonstrably free of any endoleak during follow-up, but the available electronic medical records and imaging studies did not specify the perirupture endo-leak status of the remaining two patients in the delayed group.

Of the 15 patients who presented with delayed rupture, 10 patients (66.7%) experienced aneurysm size expansion before rupture (Table I). The remaining five patients had discernible sac shrinkage after the initial EVAR.

Treatment of rupture and mortality

Of the 20 patients who presented with rupture after EVAR, six patients (all with delayed ruptures) did not undergo repair because of prohibitive risk and/or family decision, and all of these patients died. The remaining 14 (70%) patients underwent repair (nine endovascular revisions and five open repairs). Of the five early ruptures, four underwent endovascular repair and one was repaired with open surgery, resulting in two perioperative deaths. For patients who underwent repair, overall mortality at 30 days and 1 year were 42.9% and 64.3%, respectively (Table I). Mortality rates were similar between the patients with early and delayed rupture who underwent repair (40% vs 44.4%, respectively, at 30 days, and 60% vs 66.7% at 1 year).

Complications after repair

Following repair after rupture, all but one of the 14 patients had major complications, including cardiovascular complications (4 of 14), respiratory complications (7 of 14), renal complications (6 of 14), infectious complications (7 of 14), gastrointestinal complications (6 of 14), and other significant complications (8 of 14; Table III).

Risk factors for delayed rupture

In Table V, the results of the bivariate analysis comparing the delayed rupture and no rupture groups are summarized. Of the variables included in the bivariate analysis, the stepwise Cox regression analysis identified age 80 to 89 years at the initial EVAR (hazard ratio, 3.3; 95% confidence interval, 1.1–9.4; $P = .03$), and symptomatic or ruptured AAA operative indication at the initial EVAR (hazard ratio, 7.4; 95% confidence interval, 2.2–24.8; $P < .01$) as significant predictors of delayed rupture.

Surveillance patterns in delayed rupture patients

Of the 15 patients with delayed rupture, the median time from the last imaging study to rupture was 4.1 months (IQR, 1.4–8 months) (individual values can be seen in Table IV). Imaging study follow-up averaged 1.5 studies per person-year from the time of the initial EVAR to rupture.

DISCUSSION

EVAR is associated with a shorter hospital stay and decreased short-term morbidity than traditional open repair.[2–6,16,17] Long-term graft durability, however, remains a concern, and problems such as endoleak, device migration, and device failure might require adjunctive procedures to prevent aneurysm expansion. Of even greater concern is the potential for AAA rupture, which might occur in the setting of one of these other complications or might occur without warning in an aneurysm that appears to have been adequately excluded. Our study reveals that age 80 to 89 years at the initial EVAR and a symptomatic or ruptured aneurysm as the initial indication for EVAR are significant predictors of delayed rupture.

Only a few reported case series of ruptures after EVAR have been published, most of which are clinical trial summary reports or data from international registries.[8–12,18–20] The EVAR trial data from Wyss et al[13] have provided the most comprehensive summary to date. Our rate of delayed rupture is lower than the published rates from the EVAR trials.[13] The EVAR trials and the current study showed excellent follow-up (98% EVAR 1, 97% EVAR 2, 92% our study), and the increased incidence of delayed rupture noted in the EVAR trials might be because of their longer duration of follow-up (mean 4.8 years compared with 2.7 years in the current study). In addition, the trial summary report excluded nonelective cases of EVAR, and in our series there were six nonelective cases of EVAR that resulted in rupture: two patients who originally received surgery for ruptured AAA and four patients who had presented with symptomatic AAA. Two smaller European studies have shown even lower rates of rupture after EVAR.[19,20]

Five of our patients experienced rupture in the immediate perioperative period; four of these patients underwent endovascular repair and one underwent open repair. Further review of these cases suggested that technical difficulties and significant intraoperative events preceded these ruptures, implying that we should be able to reduce the rate of these rare events further with more meticulous pre-operative planning, improved technical performance, and the introduction of new graft designs.[19]

Delayed ruptures can be separated into two groups: patients who had known endoleak before rupture and those who did not. Three of our 15 patients with delayed ruptures had known persistent endoleaks before rupture, and all had undergone intervention for these endoleaks. Nine additional patients had newly detected endoleaks at the time of rupture. Three patients had neither an endoleak detected before rupture nor an endoleak identified at the time of rupture. However, two of these patients had known aneurysm expansion in the absence of detectable endoleak, and it could be presumed that there was a contributory but undetected endoleak present, although the presence of these leaks was not confirmed using imaging after rupture. It is interesting to note that there were only two patients who presented with aneurysm rupture with apparent pre-operative evidence of isolated type II endoleak. On repair however, one patient was found to have a previously undiagnosed type I leak and the other patient had a previously unknown type III leak. Thus, no ruptures in this series could be attributed to an isolated type II leak.

Our multivariable analysis demonstrated that age 80 to 89 years at the initial EVAR and urgent initial repair were independently associated with delayed rupture. Age has previously been shown to be associated with rupture after EVAR.[13] Our initial urgent repairs (107 of 1736 EVARs) were all done for symptomatic or ruptured aneurysms, and 78% were ≥5.5 cm, supporting the generally agreed-on threshold of 5.5 cm as an indication for elective repair. If more aneurysms are electively rather than urgently or emergently repaired, the delayed rupture rate might potentially decrease. Unlike Wyss et al,[13] we did not find previous complications or interventions to be associated with rupture after EVAR. This might be because of the small proportion of post-EVAR ruptures or the need for a lengthier follow-up period.

Short-term mortality remained high even in patients who were deemed stable enough to undergo emergency repair. Overall 30-day and 1-year mortality were 42.9% and 64.3%, respectively, and among those who underwent repair after delayed rupture, 30-day and 1-year mortality were 44.4% and 66.7%, respectively. Wyss et al[13] found an overall 30-day and 1-year survival of 33.3% and 14.8% in the EVAR trials, and 30-day survival of 75% among those who underwent intervention after rupture. Our lower 30-day survival in patients who underwent repair compared with that in the EVAR trials might be because of the proportionally larger percentage of patients in our study who elected intervention after delayed rupture rather than selecting comfort measures only (60% vs 44.4%).

Without long-term anatomic information for the entire 1736-patient EVAR registry, it is difficult to speculate about the durability of any particular stent graft and its ability to provide adequate outflow and continued aneurysm exclusion. It is reasonable to assume that as the duration of follow-up continues to lengthen in this cohort, we will see an increasing

number of late ruptures. At present, open repair has a negligible risk of late aneurysm development and rupture, and from our series of >1700 patients, much the same can be concluded about EVAR for most—but not all—patients. However, despite coordinated follow-up in our integrated system, many of the patients with delayed rupture presented with new issues related to their repair, which led to rupture. This would suggest that more rigorous surveillance of “at-risk” patients is warranted, but “at-risk” is as yet poorly defined. In addition, despite best practices, two patients had more than 1 year elapse between imaging studies, implying an interruption in provider-patient communication. Both of these patients had an increase in aneurysm size and new endoleaks that might have been detected in a more timely fashion. Because our long-term follow-up rate of 92% is high compared with historical case series, we speculate that instances of missed follow-up likely occur more often than documented and can lead to undetected adverse outcomes.

Limitations of our study fall into four areas: variation in practice, lack of initial morphologic data for the entire EVAR cohort, self-reporting of adverse events, and small sample size. Despite regionalized surveillance after EVAR in our health care system, treatment thresholds are not uniform among providers and individual surgeons’ decision-making and practice patterns vary widely. Furthermore, incomplete initial morphologic data impair our ability to comment on compliance with instructions for use in our overall cohort and how it might compare with the ruptured cohort. Although the data were prospectively collected, adverse events were self-reported, and underreporting is a possibility. In addition, it is difficult to account for ruptures possibly occurring outside of our health care system or to know whether any of the 22 patients (of the entire 1736-patient cohort) who died after EVAR with no known cause of death might have experienced ruptures. These represent possible sources of underreporting in this cohort. Finally, despite our sizable EVAR registry, rupture after EVAR is a rare event and the small sample size limits our ability to draw significant conclusions about event occurrence at this time. Our future work will use device-specific details from our large national dataset to identify more sensitive predictors of rupture and graft durability after EVAR.

CONCLUSIONS

Rupture after EVAR is a rare event but is associated with significant morbidity and mortality. In our large contemporary series of community-based practice encompassing elective and urgent EVAR, most patients with delayed ruptures showed late aneurysm size expansion with concomitant undetected endoleak, suggesting device-related failure as the most likely etiology. Vigilant and continued radiographic surveillance is crucial postoperatively, especially in troublesome or suboptimal cases, and further studies are needed to identify more sensitive predictors of rupture after EVAR.

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References

1. Parodi JC, Palmaz JC, Barone HD. Transfemoral intraluminal graft implantation for abdominal aortic aneurysms. Ann Vasc Surg. 1991; 5:491–9. [PubMed: 1837729]
2. Prinssen M, Verhoeven EL, Buth J, Cuypers PW, van Sambeek MR, Balm R, et al. A randomized trial comparing conventional and endovascular repair of abdominal aortic aneurysms. N Engl J Med. 2004; 351:1607–18. [PubMed: 15483279]
3. Greenhalgh RM, Brown LC, Kwong GP, Powell JT, Thompson SG. Comparison of endovascular aneurysm repair with open repair in patients with abdominal aortic aneurysm (EVAR trial 1), 30-day operative mortality results: randomised controlled trial. Lancet. 2004; 364:843–8. [PubMed: 15351191]
4. Blankensteijn JD, de Jong SE, Prinssen M, van der Ham AC, Buth J, van Sterkenburg SM, et al. Two-year outcomes after conventional or endovascular repair of abdominal aortic aneurysms. N Engl J Med. 2005; 352:2398–405. [PubMed: 15944424]
5. Lederle FA, Freischlag JA, Kyriakides TC, Padberg FT Jr, Matsumura JS, Kohler TR, et al. Outcomes following endovascular vs open repair of abdominal aortic aneurysm: a randomized trial. JAMA. 2009; 302:1535–42. [PubMed: 19826022]
6. Schermerhorn ML, O'Malley AJ, Jhaveri A, Cotterill P, Pomposelli F, Landon BE. Endovascular vs. open repair of abdominal aortic aneurysms in the Medicare population. N Engl J Med. 2008; 358:464–74. [PubMed: 18234751]
7. Ren S, Fan X, Ye Z, Liu P. Long-term outcomes of endovascular repair versus open repair of abdominal aortic aneurysm. Ann Thorac Cardiovasc Surg. 2012; 18:222–7. [PubMed: 22790994]
8. Lumsden AB, Allen RC, Chaikof EL, Resnikoff M, Moritz MW, Gerhard H, et al. Delayed rupture of aortic aneurysms following endovascular stent grafting. Am J Surg. 1995; 170:174–8. [PubMed: 7631925]
9. Alimi YS, Chakfe N, Rivoal E, Slimane KK, Valerio N, Riepe G, et al. Rupture of an abdominal aortic aneurysm after endovascular graft placement and aneurysm size reduction. J Vasc Surg. 1998; 28:178–83. [PubMed: 9685144]
10. Torsello GB, Klenk E, Kasprzak B, Umscheid T. Rupture of abdominal aortic aneurysm previously treated by endovascular stentgraft. J Vasc Surg. 1998; 28:184–7. [PubMed: 9685145]
11. Bernhard VM, Mitchell RS, Matsumura JS, Brewster DC, Decker M, Lamparello P, et al. Ruptured abdominal aortic aneurysm after endovascular repair. J Vasc Surg. 2002; 35:1155–62. [PubMed: 12042725]
12. Fransen GA, Vallabhaneni SR Sr, van Marrewijk CJ, Laheij RJ, Harris PL, Buth J. Rupture of infra-renal aortic aneurysm after endovascular repair: a series from EUROSTAR registry. Eur J Vasc Endovasc Surg. 2003; 26:487–93. [PubMed: 14532875]
13. Wyss TR, Brown LC, Powell JT, Greenhalgh RM. Rate and predictability of graft rupture after endovascular and open abdominal aortic aneurysm repair: data from the EVAR Trials. Ann Surg. 2010; 252:805–12. [PubMed: 21037436]
14. Chang RW, Goodney P, Tucker LY, Okuhn S, Hua H, Rhoades A, et al. Ten-year results of endovascular abdominal aortic aneurysm repair from a large multicenter registry. J Vasc Surg. 2013; 58:324–32. [PubMed: 23683376]
15. Chaikof EL, Blankensteijn JD, Harris PL, White GH, Zarins CK, Bernhard VM, et al. Reporting standards for endovascular aortic aneurysm repair. J Vasc Surg. 2002; 35:1048–60. [PubMed: 12021727]
16. EVAR trial participants. Endovascular aneurysm repair versus open repair in patients with abdominal aortic aneurysm (EVAR trial 1): randomised controlled trial. Lancet. 2005; 365:2179–86. [PubMed: 15978925]
17. Greenhalgh RM, Brown LC, Powell JT, Thompson SG, Epstein D, Sculpher MJ. Endovascular versus open repair of abdominal aortic aneurysm. N Engl J Med. 2010; 362:1863–71. [PubMed: 20382983]
18. Schlosser FJ, Muhs BE. Endoleaks after endovascular abdominal aortic aneurysm repair: what one needs to know. Curr Opin Cardiol. 2012; 27:598–603. [PubMed: 23032712]

19. Holt PJ, Karthikesalingam A, Patterson BO, Ghatwary T, Hinchliffe RJ, Loftus IM, et al. Aortic rupture and sac expansion after endovascular repair of abdominal aortic aneurysm. Br J Surg. 2012; 99:1657–64. [PubMed: 23023521]

20. Szmidt J, Galazka Z, Rowinski O, Nazarewski S, Jakimowicz T, Pietrasik K, et al. Late aneurysm rupture after endovascular abdominal aneurysm repair. Interact Cardiovasc Thorac Surg. 2007; 6:490–4. [PubMed: 17669913]

Table I

Demographic and clinical characteristics of 20 patients with early and delayed rupture after initial endovascular aortic aneurysm repair (*EVAR*)

Characteristic	Total	Early (n = 5)	Delayed (n = 15)	*P*
Preoperative				
Age at initial EVAR, years				.84[a]
Mean ± SD	79.0 ± 9.1	78.2 ± 10.8	79.2 ± 8.9	
Median (IQR)	81 (72–87)	81 (73–87)	81 (72–87)	
Male sex	14 (70)	3 (60)	11 (73)	.61
AAA size, cm				.29[a]
Mean ± SD	6.3 ± 0.7	6.0 ± 0.6	6.4 ± 0.7	
Median (IQR)	6.2 (5.9–6.8)	6.0 (5.8–6.2)	6.2 (5.9–7.0)	
Urgent initial EVAR	6 (30)	2 (40)	4 (27)	.61
Treatment and/or repair of rupture				.15
None	6 (30)	0 (0)	6 (40)	
Open	5 (25)	1 (20)	4 (27)	
Endovascular	9 (45)	4 (80)	5 (33)	
Aneurysm size expansion at time of rupture	10 (50)	0 (0)	10 (67)	.03
Mortality				
Overall				
30 days	11 (55)	2 (40)	9 (60)	.62
One year	15 (70)	3 (60)	12 (80)	.56
If underwent repair				
30 days	6 (43)	2 (40)	4 (44)	>.99
One year	9 (64)	3 (60)	6 (67)	>.99

AAA, Abdominal aortic aneurysm; *IQR*, interquartile range; *SD*, standard deviation.

Data are presented as number (%), except where otherwise stated.

[a]Comparison result using *t*-test.

Table II

Demographic and clinical characteristics and mortality in patients with early (perioperative) aneurysm ruptures

Patient No.	Operative indication[a]	EVAR device[a]	AAA Diameter, cm[a]	Endoleak type[b]	Repair type[b]	Mortality at end of follow-up
1	Elective	Medtronic	5.8	I	EV	Died within 30 days
2	Elective	Zenith	5.2	None	EV	Died within 1 year
3	Symptomatic AAA	Gore	6.2	None	EV	Died within 30 days
4	Elective	Gore	6.0	None	OR	Alive
5	Ruptured AAA	Medtronic	6.8	III	EV	Alive

AAA, Abdominal aortic aneurysm; *EV*, endovascular; *EVAR*, endovascular aortic aneurysm repair; *OR*, open repair.

[a] At initial EVAR. Devices from Medtronic Vascular, Santa Rosa, Calif; Cook Zenith, Bloomington, Ind; and W. L. Gore & Associates, Inc, Flagstaff, Ariz.

[b] At time of AAA rupture.

Table III

Complications after repair within 30 days in 20 patients who had rupture after initial endovascular aortic aneurysm repair (EVAR)

Complication, No.	Early group (n = 5)	Delayed group (n = 10)[a]
Cardiac	1	2
Cardiac arrest (not attributable to another complication)	1	0
Unknown cardiac complication	0	2
Pulmonary	2	4
Respiratory failure requiring prolonged mechanical ventilation	2	2
Unknown pulmonary complication	0	2
Renal	3	2
Acute renal failure	3	1
Unknown renal complication	0	1
GI: ischemic colitis	1	0
Infectious	3	3
Pneumonia	1	1
Clostridium difficile	1	0
Sepsis	1	0
Unknown infectious complication	0	2
Other	2	3
Paraplegia	1	0
Graft limb thrombosis requiring fem-fem bypass	1	0
Hematoma requiring evacuation	0	2
Multiorgan dysfunction	0	1

GI, Gastrointestinal.

[a] Five patients in the delayed group did not undergo repair and were not included in this table: one patient did not have complication data available for analysis; three patients had incomplete data available; and one patient died from exsanguination during surgery.

Table IV

Demographic and clinical characteristics and mortality in patients with delayed aneurysm ruptures after surgery

Patient No.	EVAR time periods[a]	EVAR device[a]	Time until rupture, months[b]	Time from last study to rupture, months	Aneurysm size expansion[b]	Previous endoleak	Previous reintervention	Endoleak type[c]	Repair type[c]	Reintervention successful	Mortality at end of follow-up
6	2000–2005	Medtronic	31.1	4.1	No	None	No	None	EV	N/A	Died within 30 days
7	2006–2010	Gore	2.9	2.5	No	I or II	Yes	I and II	None	No	Died within 30 days
8	2000–2005	Medtronic	86.5	20.4	Yes	None	No	I and III[c]	EV	N/A	Died within 1 year
9	2000–2005	Medtronic	87.0	1.3	No	None	No	III	EV	N/A	Alive
10	2000–2005	Zenith	18.2	2.1	Yes	II	Yes	I and II	OR	No	Died within 30 days
11	2006–2010	Medtronic	49.1	5.9	Yes	None	No	I[b]	EV	N/A	Alive
12	2006–2010	Gore	20.3	8.0	No	None	No	I	None	N/A	Died within 30 days
13	2000–2005	Zenith	57.3	9.7	Yes	None	No	I	None	N/A	Died within 1 year
14	2006–2010	Zenith	30.7	0.1	Yes	II	Yes	II and III	OR	No	Died within 1 year
15	2000–2005	Zenith	13.8	3.5	No	None	No	I	None	N/A	Died within 30 days
16	2000–2005	Medtronic	60.4	4.1	Yes	None	No	N/A	None	N/A	Died within 30 days
17	2000–2005	Medtronic	39.7	4.5	Yes	None	No	I	EV	N/A	Alive
18	2006–2010	Zenith	2.4	0.6	Yes	None	No	N/A	None	N/A	Died within 30 days
19	2000–2005	Zenith	6.9	1.4	Yes	None	No	III	OR	N/A	Died within 30 days
20	2000–2005	Medtronic	42.6	20.7	Yes	None	No	I[b]	OR	N/A	Died within 30 days

AAA, Abdominal aortic aneurysm; *EV*, endovascular; *EVAR*, endovascular aortic aneurysm repair; *N/A*, not applicable; *OR*, open repair.

Most EVARs (93.2%) in the entire cohort were performed with Cook Zenith (48.4%; Cook Inc, Bloomington, Ind), Medtronic Aneurx (19.5%; Medtronic Vascular, Santa Rosa, Calif), or low-permeability Gore Excluder (25.2%; W. L. Gore & Associates, Inc, Flagstaff, Ariz).

[a] At initial EVAR.

[b] From initial EVAR to AAA rupture.

[c] At time of AAA rupture.

Table V

Baseline preoperative demographic and clinical characteristics of interest of 1731 endovascular aortic aneurysm repair (*EVAR*) patients, stratified according to delayed rupture status (rupture >30 days after initial EVAR) during the follow-up period

Characteristics	Total	No rupture (n = 1716)	Delayed rupture (n = 15)	*P*[a]
Male sex	1498 (86.5)	1487 (86.7)	11 (73)	.13
Age, years				.10[b]
Mean ± SD	74.9 ± 7.9	74.9 ± 7.9	79.2 ± 8.9	
Median (IQR)	76.0 (70.0–81.0)	76.0 (70.0–81.0)	81.0 (72.0–87.0)	
Age group, years				.06
≤79	1205 (69.6)	1198 (69.8)	7 (47)	
80–89	499 (28.8)	492 (28.7)	7 (47)	
≥90	27 (1.6)	26 (1.5)	1 (7)	
Preoperative AAA size, cm				<.01[b]
Mean ± SD	5.8 ± 1.1	5.8 ± 1.1	6.4 ± 0.7	
Median (IQR)	5.6 (5.2–6.2)	5.6 (5.2–6.2)	6.2 (5.9–7.0)	
Urgent initial EVAR	105 (6.1)	101 (5.9)	4 (27)	.01
Treated with statin	579 (33.5)	573 (33.4)	6 (40)	.59
Preoperative embolization	116 (6.7)	115 (6.7)	1 (7)	>.99
Coronary artery disease	808 (46.7)	799 (46.6)	9 (60)	.30
Diabetes	441 (25.5)	435 (25.4)	6 (40)	.23
Hyperlipidemia	1321 (76.3)	1309 (76.3)	12 (80)	>.99
Hypertension	1450 (83.8)	1437 (83.7)	13 (87)	>.99
Peripheral vascular disease	366 (21.1)	362 (21.1)	4 (27)	.54
Current smoker	309 (17.9)	308 (18.0)	1 (7)	.49
Bifurcated graft	1481 (85.6)	1466 (85.4)	15 (100)	.15
Operative adjunctive maneuver	76 (4.4)	74 (4.3)	2 (13)	.13
Intraoperative endoleak	201 (11.6)	199 (11.6)	2 (14)	.69

AAA, Abdominal aortic aneurysm; *IQR*, interquartile range; *SD*, standard deviation.

Data are presented as number (%), except where otherwise stated. Group percentages might not total 100 due to rounding.

[a] For comparisons between the no rupture and delayed rupture groups.

[b] Comparison result using Wilcoxon Mann-Whitney test.