Rupture Rate of Large Abdominal Aortic Aneurysms in Patients Refusing or Unfit for Elective Repair

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Rupture of abdominal aortic aneurysm (AAA) can be prevented by elective surgical repair, but because most AAA never rupture, elective repair is reserved for patients at high risk of rupture. The most commonly used predictor of rupture is the maximum diameter of the AAA. Two randomized trials found no reduction in mortality from repairing AAA smaller than 5.5 cm in patients at low operative risk. No randomized trials are available in patients with larger AAA, and decision making in these patients is often complicated by advanced age and serious comorbidities. Surgery is usually deferred in high-operative-risk patients until the AAA attains a diameter at which the risk of rupture is thought to outweigh the operative risk. However, few data are available on the rupture risk of large AAA, resulting in substantial disagreement among experts. We conducted a prospective observational Veterans Affairs Cooperative Study to determine the incidence of rupture in patients with large AAA for whom elective repair was not planned because of medical contraindication or patient refusal.

METHODS
Eligible patients were those evaluated at 47 Veterans Affairs medical centers who were diagnosed as having AAA of at least 5.5 cm in diameter by ultrasonography or computed tomography (CT) within 3 months prior to enrollment and for whom elective AAA repair was not planned because of medical contraindication or patient refusal.

Context Among patients with abdominal aortic aneurysm (AAA) who have high operative risk, repair is usually deferred until the AAA reaches a diameter at which rupture risk is thought to outweigh operative risk, but few data exist on rupture risk of large AAA.

Objective To determine the incidence of rupture in patients with large AAA.

Design and Setting Prospective cohort study in 47 Veterans Affairs medical centers.

Patients Veterans (n = 198) with AAA of at least 5.5 cm for whom elective AAA repair was not planned because of medical contraindication or patient refusal. Patients were enrolled between April 1995 and April 2000 and followed up through July 2000 (mean, 1.52 years).

Main Outcome Measure Incidence of AAA rupture by strata of initial and attained diameter.

Results Outcome ascertainment was complete for all patients. There were 112 deaths (57%) and the autopsy rate was 46%. Forty-five patients had probable AAA rupture. The 1-year incidence of probable rupture by initial AAA diameter was 9.4% for AAA of 5.5 to 5.9 cm, 10.2% for AAA of 6.0 to 6.9 cm (19.1% for the subgroup of 6.5-6.9 cm), and 32.5% for AAA of 7.0 cm or more. Much of the increased risk of rupture associated with initial AAA diameters of 6.5-7.9 cm was related to the likelihood that the AAA diameter would reach 8.0 cm during follow-up, after which 25.7% ruptured within 6 months.

Conclusion The rupture rate is substantial in high-operative-risk patients with AAA of at least 5.5 cm in diameter and increases with larger diameter.

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whom elective repair was not expected in the next 6 months because of medical contraindications to surgery or patient refusal. Patients with the following were excluded: symptoms or radiological evidence of rupture, previous aortic surgery, dissection of the thoracic aorta, known condition associated with secondary AAA (eg, Marfan disease), or death expected in the next 30 days. Informed consent and signature by the patient and next of kin of a nonbinding form indicating willingness to have autopsy were required. Patients were identified when they were referred to the vascular surgery service, a process facilitated by use of notices and reminders sent to other physicians. The protocol required the vascular surgery team at each center to offer entry to all eligible patients.

Follow-up began at enrollment by telephone call to the study’s central office confirming eligibility and consent, so the study was entirely prospective. Subsequent measurements of AAA were obtained by ultrasonography at 6-month intervals throughout the study. The maximum outside AAA diameter was used, as determined by the radiologist’s reading at the participating medical center, consistent with usual clinical practice. Follow-up ended at the time of elective AAA repair, following successful repair of rupture, at death, or at the end of the study.

The main outcome measure was incidence of rupture by initial and attained AAA diameter. Attained AAA diameter was determined by allocating patients to a diameter stratum based on the day that the AAA attained that diameter, either initially or later during follow-up, at which time they were censored out of any previous smaller AAA stratum (so events were only counted once). Patients were not moved from larger to smaller diameter strata. Measurements obtained within 7 days before a possible rupture (which could not be used for decisions regarding elective repair and could reflect acute prevalence of AAA expansion) were not used to assign new strata or to compute AAA enlargement rate as a possible predictor of rupture.

We planned to enroll 120 patients in each of 5 strata of AAA diameter: 5.5 to 5.9 cm, 6.0 to 6.4 cm, 6.5 to 6.9 cm, 7.0 to 7.9 cm, and 8.0 cm or larger. Because fewer patients were actually enrolled, resulting in low numbers in some strata, we collapsed the data into 3 strata, 5.5 to 5.9 cm, 6.0 to 6.9 cm, and 7.0 cm or larger, selected because they had been used in previous reports and resulting in at least 50 patients per stratum.

Medical records, including hospital records, nursing home notes, and imaging and autopsy reports, were requested for deaths or AAA repair procedures. Eyewitness accounts were obtained by telephone for deaths that occurred outside of health care facilities. Death certificates were not used to assign cause of death.

Rupture rates were generated by product-limit estimates (SAS PROC LIFETEST, SAS Institute Inc, Cary, NC) for definite, probable, and possible ruptures. Definite ruptures were those confirmed at surgery or autopsy or by CT. Probable ruptures included, in addition to definite ruptures, cases in which patients died with symptoms consistent with rupture (severe abdominal, flank, or back pain) but without objective confirmation of rupture, and also patients who had urgent repair of AAA that had not ruptured but had developed symptoms consistent with rupture, a presentation that often heralds imminent rupture. Possible ruptures included probable ruptures and cases in which patients had sudden unexplained or un-witnessed deaths without autopsy. Many patients in the latter group would be expected to have died of cardiac or pulmonary disease, but AAA is also a leading cause of sudden death. We expected that the true rupture rate should be between the definite rupture rate and the possible rupture rate, and we consider the probable rupture rate to be the best estimate of the true rate.

We used Cox regression models (SAS PROC PHREG) to assess baseline variables as predictors of rupture and logistic regression models (SAS PROC LOGIST) that included last measured AAA diameter to assess AAA enlargement rate as a predictor of rupture. Enlargement rate was calculated using the first and last imaging test for each patient after excluding measurements within 7 days of possible rupture and patients with less than 120 days between the first and last measurements (because of the greater distortion due to measurement variation). The number of ruptures observed was not large enough to generate valid predictive models using all collected variables simultaneously, so models using AAA diameter plus 1 other variable were used.

**RESULTS**

Among 266 eligible patients, 68 refused enrollment. The remaining 198 patients were enrolled between April 1995 and April 2000 and followed up through July 2000. All patients but 1 were men, nearly all had a history of smoking (TABLE 1), reflecting both the veteran population and the population at risk for AAA, and most were...
elderly and had high rates of comorbidities, especially coronary artery disease and chronic obstructive pulmonary disease.

Vital and operative status as of July 2000 was confirmed for all patients. Hospital records were obtained for all cases of AAA repair, and firsthand information (including hospital and nursing home records, autopsy reports, and eyewitness reports, as applicable in each case) was obtained for all deaths.

Table 2. Cumulative Incidence of Rupture by Initial AAA Diameter

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Patients With AAA 5.5-5.9 cm (n = 61)

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Patients With AAA 6.0-6.9 cm (n = 85)

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Patients With AAA ≥7.0 cm (n = 52)

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*Data are given as percentages. AAA indicates abdominal aortic aneurysm; ellipses, data not shown (for instances in which <10 patients remained in observation at the beginning of the interval). Definite ruptures were confirmed by autopsy, surgery, or computed tomographic scan. Probable ruptures were defined as all definite ruptures plus cases of death with symptoms consistent with AAA rupture and cases of repair of symptomatic unruptured AAA. Possible ruptures were defined as all probable ruptures plus cases of sudden unexplained/unwitnessed deaths.

Figure 1. Cumulative Incidence of Probable Rupture by Initial Abdominal Aortic Aneurysm (AAA) Diameter

The ≥7.0-cm stratum was significantly different from the other 2 strata (both P<.01), which did not differ significantly from each other (P=.65).

During a mean follow-up of 1.52 years, 112 patients (57%) died. All-cause mortality was 29.8% at 1 year, 55.5% at 2 years, and 75.4% at 3 years. Autopsy was performed on 52 (46%) of the 112 deaths. Rupture was confirmed by surgery or autopsy in 35 patients (17.7%), of whom 17 had attempted aneurysm repair (2 endovascular repairs) and 6 survived. Four patients had urgent repair of symptomatic unruptured AAA, 3 of whom survived. Six patients (3.0%) died with symptoms consistent with AAA rupture. Seven patients (3.5%) had sudden unexplained or unwitnessed deaths with no autopsy. Follow-up was terminated because of elective repair of symptomatic AAA in 21 patients (10.6%), 2 of whom had endovascular repair, with 3 postoperative deaths (all in patients who had open repair). Sixty-nine patients (34.8%) died from a known cause other than AAA (21 cardiac, 20 respiratory, 6 stroke, 14 cancer, and 8 miscellaneous deaths). Fifty-six patients (28.3%) were alive without AAA rupture at the end of the study.

The cumulative incidence of possible, probable, or definite rupture for 3 strata of initial AAA diameter is shown in Table 2 and of probable rupture for 3 strata of initial AAA diameter in Figure 1. Analysis of the data using the originally planned 5 strata revealed that AAA of 6.5 to 6.9 cm had an intermediate rupture risk between AAA of 5.5 to 6.4 cm and AAA of 7.0 cm or more. The rates of probable rupture for AAA with initial diameter of 6.5 to 6.9 cm at 6, 12, and 18 months (after which <10 patients remained in observation) were 10.3%, 19.1%, and 19.1%, respectively.

Assessment of rupture rate by attained AAA diameter required follow-up imaging measurements. The percentage of ultrasonography follow-up visits completed within 1 month of the due date was 72%. For all patients who had possible rupture, imaging measurements were available for all but 4 patients within 6 months before the event and for all but 1 patient within 12 months before the event. Cumulative incidence of rupture by 3 strata of attained AAA diameter is shown in Table 3. Figure 2 shows the probable rupture rate by attained AAA diameter using the originally planned 5 strata. The risk of rupture increased significantly when the AAA reached at least 8.0 cm (Figure 2), indicating that much of the increased risk associated with initial diameters of 6.5 to 7.9 cm was related to an increased likelihood that the aneurysm would reach 8.0 cm during follow-up. The rates of probable rupture for attained AAA diameter of at least 8.0 cm at 6, 12, 18, and
24 months (after which <10 patients remained in observation) were 25.7%, 36.4%, 39.5%, and 54.7%, respectively.

Diameter of the AAA was the strongest predictor of rupture in terms of variance explained (relative risk [RR], 1.39 per 1 cm; 95% confidence interval [CI], 1.11-1.73). After adjustment for AAA diameter at entry, the following other factors were also significant predictors of probable rupture: renal artery involvement of the AAA (RR, 2.36; 95% CI, 1.12-4.97), lower weight (RR, 0.75 per 10 kg; 95% CI, 0.61-0.91), and, paradoxically, no history of smoking (RR, 0.30; 95% CI, 0.11-0.84), no myocardial infarction (RR, 0.46; 95% CI, 0.24-0.88), and no coronary artery bypass graft surgery (RR, 0.30; 95% CI, 0.12-0.71). Age (RR, 1.00 per year; 95% CI, 0.96-1.05), family history of AAA (RR, 0.79; 95% CI, 0.27-2.31), systolic blood pressure (RR, 1.00 per mm Hg; 95% CI, 0.98-1.01), diastolic blood pressure (RR, 1.01; 95% CI, 0.98-1.04), chronic obstructive pulmonary disease (RR, 0.76; 95% CI, 0.41-1.38), use of β-blockers (RR, 0.53; 95% CI, 0.24-1.21), and poor medical condition (compared with refusal alone as the reason for no planned repair, RR, 0.81; 95% CI, 0.40-1.65) were not significant predictors.

The median rate of change in AAA diameter was an increase of 0.43 cm per year (interquartile range, 0.08-0.79 cm per year). Unadjusted for AAA diameter, patients with probable rupture had a significantly greater mean AAA enlargement rate than patients without rupture (0.75 cm per year vs 0.41 cm per year; \( P = .003 \)). In models that included both enlargement rate and the last measurement of AAA diameter as independent variables, enlargement rate was not a significant predictor of probable rupture (RR per 0.1 cm per year, 1.07; 95% CI, 0.99-1.15; \( P = .09 \)).

**COMMENT**

We report a prospective, multicenter, observational study of the rupture rate of large AAA in mostly high-operative-risk male patients for whom outcome ascertainment was complete and based on firsthand reports. Rupture risk was substantial for all AAA greater than 5.5 cm and increased markedly when initial diameter exceeded 6.5 or 7.0 cm or attained diameter exceeded 8.0 cm. These diameters could serve as useful thresholds for decision making in high-operative-risk male patients but may not apply to women or to patients who have low operative risk. The 9.4% 1-year probable rupture rate we observed for initial AAA of 5.5 to 5.9 cm is 10-fold higher than the rates observed in 2 recent randomized trials for AAA of 4.0 to 5.4 cm. While this large difference likely results in part from the difference in AAA diameters, it may also reflect a difference in rupture rates between our mostly high-operative-risk patients with severe comorbidities and the healthier patients randomized into the trials. Consistent with this possibility, ineligible patients followed up outside the United Kingdom Small Aneurysm Trial had a higher rupture rate than

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<td>Patients With AAA 5.5-5.9 cm (n = 61)</td>
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<td>Definite (n = 4)</td>
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| Patients With AAA 6.0-6.9 cm (n = 113) |
| Definite (n = 6)       | 2.0  | 3.8  | 6.5  | 13.5 | 13.5 |
| Probable (n = 8)       | 3.0  | 6.1  | 8.8  | 15.6 | 15.6 |
| Possible (n = 11)      | 3.0  | 7.4  | 10.0 | 20.2 | 20.2 |

| Patients With AAA ≥7.0 cm (n = 107) |
| Definite (n = 25)       | 11.0 | 23.4 | 28.7 | 31.8 | 37.1 |
| Probable (n = 31)       | 11.9 | 29.2 | 34.1 | 37.0 | 47.1 |
| Possible (n = 34)       | 14.0 | 30.9 | 35.7 | 41.0 | 50.5 |

*Data are given as percentages. Patient could be evaluated in more than 1 stratum in this analysis, but events are counted only once. AAA indicates abdominal aortic aneurysm; ellipses, data not shown (for instances in which <10 patients remained in observation at the beginning of the interval). Definite ruptures were confirmed by autopsy, surgery, or computed tomographic scan. See Table 2 footnote for rupture definitions.

**Figure 2. Cumulative Incidence of Probable Rupture by Attained AAA Diameter**

Patients could be evaluated in more than 1 stratum, but events are counted only once. The 6.0-6.4-cm and ≥8.0-cm strata each differed significantly from all other strata (all \( P < .01 \)). The other 3 strata did not differ significantly from each other (all \( P > .20 \)).

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randomized with comparable AAA diameters.12

Because most large AAA are surgically repaired, natural history data are difficult to obtain, even in patients at high operative risk, as evidenced by our 47 medical centers requiring 5 years to enroll 198 patients. For this reason, few data exist in the literature, and most predate the introduction of accurate AAA measurement techniques. In 1966, Szilagyi et al11 reported rupture in 61 of 141 AAA of at least 7 cm among patients who were followed up for a mean of 17 months, a crude rate of 30.5% per year. Recently, Powell et al12 observed 0.28 ruptures per patient-year in 100 patients with AAA greater than 5.5 cm. Several other studies have also reported rupture rates that are generally consistent with our findings.5,6,14

As expected, AAA diameter was the strongest predictor of rupture in our study. The unexpected protective effects of smoking, myocardial infarction, and coronary artery bypass graft surgery may be artifacts of the high mortality rate. Enlargement rate of AAA was a significant univariate predictor of rupture, but the effect was not significant after adjustment for last measured AAA diameter. An enlargement rate of at least 1.0 cm/year has been used as an independent indication for repair of small AAA in 3 large randomized trials.2,3,15 However, we are aware of no previous studies that measured the impact of enlargement rate on rupture risk. Our data suggest but do not confirm an independent effect, and further studies are needed to resolve this question.

Outcome ascertainment is problematic in studies of AAA rupture because causes of sudden death are difficult to distinguish. Our requirement of written intent to have autopsy, consigned by next of kin, resulted in a rate 4 times the national rate16 but still less than 50%. Hospital and nursing home records and eyewitness accounts were therefore important for optimizing outcome assessment in our study.

We conclude that the rupture rate is substantial in patients with high operative risk and AAA greater than 5.5 cm, and increases markedly with diameter.

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Critical revision of the manuscript for important intellectual content: Ledere, Johnson, Wilson, Ballard, Jordan, Blebea, Littooy, Freischlag, Bandky, Rapp, Salam. Statistical expertise: Johnson, Ballard.

Obtained funding: Ledere, Johnson.

Administrative, technical, or material support: Ledere, Johnson, Wilson, Blebea, Freischlag. Study supervision: Ledere, Johnson, Blebea, Rapp, Salam.

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REFERENCES