Designation as "Unfit for Open Repair" Is Associated With Poor Outcomes After Endovascular Aortic Aneurysm Repair

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Background—Endovascular aortic aneurysm repair (EVAR) is often offered to patients with abdominal aortic aneurysms (AAAs) considered preoperatively to be unfit for open AAA repair (oAAA). This study describes the short- and long-term outcomes of patients undergoing EVAR with AAAs <6.5 cm who are considered unfit for oAAA.

Methods and Results—We analyzed elective EVARs for AAAs <6.5 cm diameter in the Vascular Study Group of New England (2003–2011). Patients were designated as fit or unfit for oAAA by the treating surgeon. End points included in-hospital major adverse events and long-term mortality. We identified patient characteristics associated with being unfit for open repair and predictors of survival using multivariable analyses. Of 1653 EVARs, 309 (18.7%) patients were deemed unfit for oAAA. These patients were more likely to have advanced age, cardiac disease, chronic obstructive pulmonary disease, and larger aneurysms at the time of repair (54 versus 56 mm, P=0.001). Patients unfit for oAAA had higher rates of cardiac (7.8% versus 3.1%, P<0.01) and pulmonary (3.6 versus 1.6, P<0.01) complications and worse survival rates at 5 years (61% versus 80%; log rank P<0.01) compared with those deemed fit for oAAA. Finally, patients designated as unfit for oAAA had worse survival, even adjusting for patient characteristics and aneurysm size (hazard ratio, 1.6; 95% confidence interval, 1.2–2.2; P<0.01).

Conclusions—In patients with AAAs <6.5 cm, designation by the operating surgeon as unfit for oAAA provides insight into both short- and long-term efficacy of EVAR. Patients unable to tolerate oAAA may not benefit from EVAR unless their risk of AAA rupture is very high. **(*Circ Cardiovasc Qual Outcomes*. 2013;6:575-581.)**

Key Words: aneurysm ■ complications ■ mortality

The introduction of endovascular aneurysm repair (EVAR) for the treatment of abdominal aortic aneurysms (AAAs) has reduced short-term morbidity and perioperative mortality compared with open AAA repair (oAAA).[1–4] Because periprocedural morbidity can be reduced with the use of EVAR, patients with advanced age,[5] as well as cardiac and pulmonary comorbidities,[6] are often offered EVAR when oAAA would have been judged to be of prohibitive risk. Despite success in limiting the short-term risks associated with AAA repair, EVAR has not been associated with better long-term survival as compared with oAAA.[2,3]

Further, many have questioned whether patients deemed too high risk for oAAA will gain any survival benefit when compared with medical treatment of their aneurysms. In the best known trial addressing this question, the EVAR-2 investigators randomized 338 patients to either EVAR or no intervention from 1999 to 2003.[7] These patients included those with active cardiac disease, poor respiratory function, uncompensated congestive heart failure, severe valvular disease, or poor renal function.[8] Although there was no survival benefit of EVAR in these high-risk patients, the trial was criticized because of multiple crossovers between groups. Further, this trial was performed during an earlier era of experience in EVAR and represents data that are now nearly a decade old.

It remains unknown to many patients and physicians how the results of the EVAR-2 trial have translated into real-world practice. To investigate this question, we used the Vascular Study Group of New England (VSGNE) cohort, which is a regional consortium of physicians and hospitals dedicated to tracking outcomes after vascular surgery procedures and quality improvement efforts. We sought to identify patients undergoing EVAR to see whether being designated as unfit for open repair in the VSGNE database by the operating surgeon was associated with greater short- and long-term morbidity and mortality compared with patients considered fit for oAAA in patients with AAAs <6.5 cm.

Received January 14, 2013; accepted July 1, 2013.

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This manuscript was handled independently by Frederick L. Grover, MD as Guest Editor. The Editors had no role in the evaluation of the manuscript or the decision about its acceptance.

Presented at the American Heart Association Quality of Care and Outcomes Research, Atlanta, GA, May 2012.

The online-only Data Supplement is available at http://circoutcomes.ahajournals.org/lookup/suppl/doi:10.1161/CIRCOUTCOMES.113.000303/-/DC1.

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WHAT IS KNOWN

- Endovascular aneurysm repair has reduced operative mortality and morbidity compared with open abdominal aortic aneurysm repair (oAAA).
- Data suggest that repair of AAA in morbidly sick patients does not improve overall mortality.

WHAT THE STUDY ADDS

- Chronic obstructive pulmonary disease, advanced coronary disease, and congestive heart failure are major contributors to patents being deemed unable to tolerate oAAA.
- Physician's assessment of a patient's ability to tolerate oAAA (the eye-ball test) has discriminatory and prognostic value for postoperative events and survival in addition to standard patient risk factors.

Methods

Subjects and Database

This is a retrospective analysis of data collected by the VSGNE, a regional cooperative quality improvement initiative developed in 2002 to study regional outcomes in vascular surgery. Further details about this registry have been published previously.[9] The VSGNE collects preoperative, operative, and postoperative data as well as information on long-term outcomes such as sac size, need for revision or reintervention, and other pertinent outcomes within the first 2 years after surgery. Registry data are compared with participating hospital claims in regular audits, and missing cases are retrieved to ensure complete capture of all operative procedures at participating centers.[9] Mortality is audited using the Social Security Death Index (SSDI).

Definitions

For this analysis, we limited our cohort to those patients who underwent elective EVAR between 2003 and 2011. Patients undergoing oAAA or emergent or urgent repair were excluded. In addition, we excluded patients with AAAs ≥6.5 cm in maximum AP diameter because the alternative of medical treatment is seldom applicable in large AAAs.[10] Those with AAAs <5.5 cm, typically the size threshold for repair, were included in this analysis because these patients had synchronous iliac artery aneurysms, saccular aneurysms, or rapid expansion indicative of repair.

Our main outcome measure was mortality. We studied both perioperative mortality, defined as in-hospital death, and long-term survival, calculated using both 1-year follow-up in the VSGNE database and by matching patient information with the Social Security Death Index. If discrepancies were encountered, the Social Security Death Index date was considered the correct date for use.

Our exposure variable was the designation of a patient being unfit for oAAA. This variable is determined preoperatively by the referring physician or surgeon for all patients before undergoing EVAR. All patients undergoing EVAR within the VSGNE must be designated as fit versus unfit for oAAA during the data collection process. This variable is included with the other pre, intra-, and postprocedural data that are tracked. This variable is, by intent, subjective and is meant to indicate that if EVAR could not be performed, the patient would not be an appropriate candidate for oAAA.

Cardiovascular complications were defined as clinical myocardial infarction (MI) with EKG changes, enzyme positive only MI, dysrhythmias requiring medication or cardioversion, or acute CHF. Respiratory complications were defined as a new pneumonia or need for ventilator support after initial extubation postoperatively. Renal dysfunction was defined as elevation of creatinine by 0.5 mg/dL. Leg ischemia was defined as a change in the preoperative vascular examination or drop of >0.15 in the ankle brachial index. Bowel ischemia was diagnosed by colonoscopy, bloody bowel movement, or clinically.

Data Collection and Statistical Analysis

Physicians, nurses, or clinical data abstractors entered data prospectively on clinical and demographic variables. Research analysts were blinded to patient, surgeon, and hospital identity. The Committee for the Protection of Human Subjects at Dartmouth Medical School (IRB) has approved the use of deidentified data from VSGNE for research purposes.

Preoperative variables were compared for each end point using χ^2 for categorical variables with Fisher's exact as indicated. A 2-sample *t* test or Wilcoxon rank sum test was used to compare normally or nonnormally distributed continuous variables, respectively. For survival analysis, comparisons were made with univariate log-rank or Cox proportional hazards for categorical and continuous variables, respectively. Variables of clinical significance and those with a probability value of <0.1 by univariate survival analysis were included in a backwards stepwise multivariable Cox proportional hazards model to identify significant predictors of long-term mortality. Variables were removed using the likelihood ratio test by assessing their clinical and statistical association with long-term survival. Additionally, a similarly developed backwards stepwise multiple logistic regression was performed to identify patient factors associated with being designated as unfit for open repair. As a separate analysis, we derived our multivariable analysis for unfit on a randomly split dataset. Both the overall analysis as well as spilt dataset analysis model discrimination was assessed using the c-statistic as well as the Hosmer-Lemeshow goodness of fit test. Both logistic and Cox models were clustered by surgeon for analysis. This compares the model's expected risk of a patient being classified as unfit based on his or her preoperative risk factors with the actual observed designation of unfit. A $P>0.05$ suggests that the observed and predicted are not statistically different and that the model fits the data well. Continuous variables with nonlinear risk were categorized for analysis. Age was categorized by quartiles for analysis. Probability values <0.05 were considered significant.

Results

Patient Characteristics

Over the study interval, 87 physicians in 21 different centers performed 1653 elective EVARs for AAAs between 2.1 cm and 6.5 cm. Those <4 cm constituted only 4% of the cohort, the majority of which (69%) included iliac artery aneurysms. Of the total EVAR cohort, 309 (19%) were performed in patients deemed unfit for oAAA by the operating surgeon, whereas the remaining 81% were deemed eligible to undergo either oAAA or EVAR.

Patients deemed unfit for oAAA were older (median 73 versus 77 years, $P<0.01$), had slightly larger aneurysms (median 54 mm versus 56 mm, $P<0.001$), and were more likely to be women (26% versus 19.3%, $P=0.004$; Table 1). Patients deemed unfit for oAAA also had higher rates of coronary artery disease, congestive heart failure, and chronic obstructive pulmonary disease (COPD). Finally, they also were more likely to have abnormal stress tests, have a depressed ejection fraction (EF), and have worse renal function, and less likely to be on statin medications or β-blockers (Table 1).

Patient Factors Associated with the Designation Unfit for oAAA

To identify factors associated with the designation as unfit for oAAA, a multivariable model was created based on the

Table 1. Patient Characteristics

	Fit for oAAA (n=1344)	Unfit for oAAA (n=309)	*P* Value
Age (y), median (IQR)	73 (67–79)	77 (70–82)	<0.001*
Max AAA Diameter (mm), median (IQR)	53.5 (6.2)	56 (52–60)	<0.001*
Male, %	80.7	73.5	0.004
Smoking history, %	86.0	87.7	0.427
Hypertension, %	84.3	84.8	0.823
Diabetes mellitus, %	20.6	19.7	0.733
Coronary artery disease, %			
None	68.5	50.8	<0.001
Previous MI	21.9	29.4	
Stable angina	8.6	19.1	
Recent MI/unstable angina	1.0	0.7	
Previous CABG/PTCA, %	29.9	32.7	0.33
CHF, %			
No	92.9	77.9	<0.001
Asymptomatic	4.1	11.4	
Mild	2.5	9.1	
Severe	0.4	1.6	
Ejection fraction, %			
<30%	1.5	6.2	<0.001
30% to 50%	9.7	15.5	
≥50%	40.9	36.9	
Not Done	44.9	37.5	
Unknown	3.1	3.9	
COPD, %			
No	71.5	40.5	<0.001
Yes, not treated	12.3	16.8	
Yes, on medication	14.1	26.2	
Home O_2	2.1	16.5	
Stress tests, %			
Not done	56.4	51.6	0.012
Normal	31.1	29.4	
Abnormal	12.5	19.0	
eGFR, %			
>60	66.5	57.1	0.007
40–60	24.5	31.1	
30–39	6.7	9.1	
<30	2.3	2.7	
Aspirin use, %	72.9	71.8	0.316
Plavix use, %	6.0	9.4	0.066
Statin, %	70.8	66.7	0.314
β-blocker use, %	77.3	71.8	0.043

AAA indicates abdominal aortic aneurysm; CABG/PTCA, coronary artery bypass graft/percutaneous transluminal coronary angioplasty; CHF, congestive heart failure; COPD, chronic obstructive pulmonary disease; eGFR, estimated glomerular filtration rate in mL/min per 1.73 m^2; IQR, interquartile range; MI, myocardial infarction; and oAAA, open AAA repair.

*Comparison by Wilcoxon Rank Sum test.

univariate patient characteristics associated with this designation. As shown in Table 2, COPD was associated with designation as unfit for oAAA, increasing with the extent of COPD, from medication-controlled COPD (odds ratio [OR], 3.7; 95% confidence interval [CI], 2.7–5.1) to requiring home oxygen therapy (OR, 16.4; 95% CI, 9.2–29.3). Other factors, including age >80 years (OR, 2.7; 95% CI, 1.6–4.3), women (OR, 1.7; 95% CI, 1.2–2.4), stable angina (OR, 2.8; 95% CI, 1.9–4.2), history of myocardial infarction (OR, 1.6; 95% CI, 1.1–2.4), and EF <30% (OR, 4.9; 95% CI 2.1–11.0) were also associated with the designation as unfit for oAAA. Finally, increasing aneurysm size (>5.5 cm versus <5.5 cm) was associated with being unfit for oAAA. Our model demonstrated good discrimination with an area under the curve of 0.79 and "fit" the data well (Hosmer-Lemeshow goodness of fit *P*=0.42). These findings were similar to our derivation and validation analysis with an area under the curve of 0.77 and Hosmer-Lemeshow *P*=0.37 on the validation cohort (see online-only Data Supplement).

Table 2. Multivariate Predictors of Being Deemed Unfit for Open AAA Repair

	OR	95% CI	*P* Value
Age, y			
<65	1.0 (Ref)		
65–74	0.9	0.6–1.5	0.787
75–80	1.2	0.7–2.0	0.529
≥80	2.7	1.6–4.3	<0.001
Female	1.7	1.2–2.4	0.003
Coronary artery disease			
None	1.0 (Ref)		
Prior MI	1.6	1.1–2.4	0.007
Stable angina	2.8	1.8–4.2	<0.001
Recent MI/unstable angina	0.7	0.1–5.0	0.735
Ejection fraction			
>50%	1.0 (Ref)		
30% to 50%	1.8	1.1–2.9	0.015
<30%	4.9	2.1–11.0	<0.001
Not evaluated	1.1	0.7–17	0.692
COPD			
None	1.0 (Ref)		
Not treated	2.5	1.8–3.6	<0.001
On medications	3.7	2.7–5.1	<0.001
Home O_2	16.4	9.2–29.3	<0.001
Aneurysm size >5.5 cm	1.9	1.5–2.5	<0.001

AAA indicates abdominal aortic aneurysm; CI, confidence interval; COPD, chronic obstructive pulmonary disease; eGFR, estimated glomerular filtration rate in mL/min per 1.73 m^2; MI, myocardial infarction; and OR, odds ratio.

Perioperative Outcomes

Overall, patients deemed unfit for oAAA had worse perioperative outcomes when undergoing EVAR when compared with patients designated as fit for oAAA. The risk of any major complication (death, respiratory, or cardiovascular) was higher for unfit patients compared with patients fit for oAAA (12.5% versus 3.7%, *P*<0.001). These differences were determined largely by higher rates of cardiovascular complications (7.8% versus 3.1%, *P*<0.001) and respiratory complications (3.6% versus 1.6%, *P*=0.029; Table 3). Cardiac complications were a result of higher rates of MI in unfit patients. Higher respiratory

Table 3. Postoperative Outcomes After Endovascular Aneurysm Repair

Variable	Fit for oAAA (n=1344) % (n=2009)	Unfit for oAAA (n=309) % (n=1406)	*P* Value
ICU stay (days, median, IQR)	1 (1–2)	1 (1–3)	0.0008*
Any major complication, %	3.7	12.5	<0.001
Mortality, %	0.3	0.7	0.313†
Any cardiovascular complication, %	3.1	7.8	<0.001
Any MI, %	1.3	4.2	<0.001
Troponin only	0.8	2.3	
EKG or clinical	0.5	1.9	
Dysrhythmia, %	1.9	4.5	0.005
Congestive heart failure, %	0.9	1.9	0.11
Any MI, %	1.3	4.2	<0.001
Troponin only	0.8	2.3	
EKG or clinical	0.5	1.9	
Any respiratory complication, %	1.6	3.6	0.029
Pneumonia	0.6	1.3	
Ventilation >24 h	1.0	2.3	
Not extubated in OR, %	98.1	95.3	0.009
Vasopressors requirement, %	2.91	4.5	<0.001
Change renal, %			
No	97.3	95.4	0.011†
Creatinine elevation >0.5 mg/dL	2.5	3.6	
Temporary dialysis	0.2	0.0	
Permanent dialysis	0.0	1.0	
Leg ischemia, %	1.0	1.0	0.998†
Bowel ischemia, %	0.3	1.6	0.015†
Wound complication, %	0.8	1.6	0.196

Any major complication: any cardiovascular or respiratory complication or postop death; any cardiovascular complication, MI, congestive heart failure, or dysrhythmia; any respiratory complication, pneumonia or need for reintubation. ICU indicates intensive care unit; IQR, interquartile range; MI, myocardial infarction; and oAAA, open abdominal aortic aneurysm repair.

*Rank Sum; †Fisher exact.

complications were a result of increased rates of both pneumonia and reintubation in unfit patients. unfit patients also had longer ICU stays, were less likely to be extubated in the operating room after surgery, and had higher rates of bowel ischemia and renal dysfunction. However, there was no difference in postoperative mortality between unfit patients and patients deemed fit for oAAA repair (0.65% versus 0.3%, P=0.3).

Long-Term Survival

Long-term survival was worse in patients deemed unfit for oAAA. The 1-, 3-, and 5-year survival for those deemed fit for oAAA was 96%, 89%, and 80% compared with 93%, 73%, and 61% for those unfit for oAAA (log rank<0.001; Figure 1).

Among all patients in our cohort, a multivariable Cox proportional hazards model was developed to identify independent factors associated with long-term mortality (Table 4). The patient characteristics most closely associated with poor long-term survival were unstable angina or recent MI (hazard ratio [HR], 3.9; 95% CI, 1.9–8.1), home oxygen use (HR, 2.3; 95% CI, 1.5–3.6), and poor renal function (eGFR<30; HR, 2.5; 95% CI, 1.2–5.4), whereas aspirin use was protective (HR, 0.7; 95% CI, 0.6–0.9). As expected, increasing age reduced the likelihood for long-term survival. Even when accounting for patient baseline factors, designation as unfit for oAAA was a significant predictor of worse 5-year survival (HR, 1.6; 95% CI, 1.2–2.2). Although females were more likely to be deemed unfit for oAAA, they were not associated with a difference in survival.

Discussion

Asymptomatic AAA repair is a prophylactic operation intended to prevent death from ruptured AAA. Thus, the treating physician must carefully weigh the expected benefit from repair (prevention of rupture, which is almost universally fatal) versus the risk of operative repair, in the context of the patient's comorbidity burden and life expectancy. Patients with advanced comorbid conditions, which may prompt their treating physician to designate them as unfit for oAAA, also have reduced life expectancy compared with patients without comorbidities. One may infer, therefore, that patients with the most significant comorbidity burdens gain the least survival benefit from AAA repair because of the competing risk of death from their nonaneurysm-related comorbidities.

In the present study, we have demonstrated that patients designated by the operating surgeon as unfit for oAAA most commonly receive this designation because of advanced cardiac, respiratory, and renal comorbidities. Interestingly, female gender was also associated with being deemed unfit, chiefly as a result of advanced age and higher rates of COPD among females in our cohort. Further, although designation as unfit for oAAA does not result in higher perioperative mortality as compared with other patients undergoing EVAR, this designation is associated with more perioperative complications and worse long-term survival even when adjusting for other competing comorbidities. In other words, although objective quantification of comorbidites is important in perioperative risk stratification, subjective physician assessment (ie, the eyeball test) among providers in our region remains an important element in identifying patients who are likely to have a complicated postoperative course and unlikely to maximize the long-term survival benefit of AAA repair.

It is important to note that our study design is observational in nature and has no control arm wherein patients judged unfit for oAAA did not undergo AAA repair. In contrast, the randomized EVAR-2 study published in 2004 did address this concern. In EVAR-2, 338 patients >60 years of age who were deemed unfit for oAAA were randomized to either EVAR (n=166) or no AAA repair (n=172).[7] During this earlier time period, the 30-day mortality rate after EVAR was much higher than in our present study (9% versus <1%). Despite high perioperative mortality in EVAR-2, there were no differences noted in crude or adjusted aneurysm-related death or all-cause mortality at 4 years. The treatment arm of EVAR-2 demonstrated a 34% survival at 5 years. In comparison, our cohort of patients unfit for oAAA had an intermediate survival of 61% at 5 years. This was higher than the EVAR-2 cohort but lower than those able to tolerate oAAA (Figure 1) in the VSGNE.

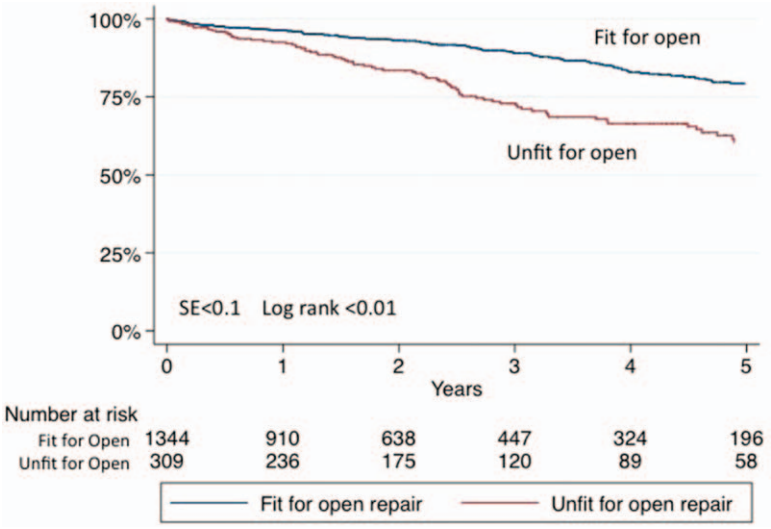

Figure 1. Survival after endovascular aneurysm repair within the Vascular Study Group of New England.

Why might these differences exist between our work and the EVAR-2 trial? First, EVAR-2 operations took place from 1999 to 2003, early in the EVAR experience and dissemination, and centers only needed 20 prior EVAR procedures to enroll patients. In comparison, the VSGNE studied patients who underwent surgery from 2003 to 2011. Providers in this later time frame may have had more experience in preoperative planning, execution, and patient selection for successful EVAR with reduced morbidity.

Table 4. Multivariate Predictors of Mortality Among Patients Undergoing Endovascular Aortic Aneurysm Repair

Variable	HR	95% CI	*P* Value
Age, y			
<65	1.0 (ref)		
65–74	1.3	0.8–2.1	0.364
75–80	1.8	1.1–3.0	0.014
≥80	2.1	1.2–3.8	0.013
Coronary artery disease			
None	1.0 (ref)		
Previous MI	1.4	1.0–1.8	0.028
Stable angina	1.3	1.0–1.8	0.047
Recent MI/unstable angina	3.9	1.9–8.1	<0.001
COPD			
None	1.0 (ref)		
Not treated	0.7	0.5–1.0	0.07
On Meds	0.9	0.6–1.4	0.641
Home O2	2.3	1.5–3.6	<0.001
eGFR			
>60	1.0 (ref)		
40–60	1.2	0.9–1.5	0.131
30–39	1.5	0.9–2.5	0.098
<30	2.5	1.2–5.4	0.015
Aspirin	0.7	0.6–0.9	0.017
Statin	0.8	0.6–1.0	0.045
Unfit for open	1.6	1.2–2.2	<0.001

CI indicates confidence interval; COPD, chronic obstructive pulmonary disease; eGFR, estimated glomerular filtration rate in mL/min per 1.73 m^2; HR, hazard ratio; and MI, myocardial infarction.

Second, although our study cohort is similar to EVAR-1, it may not be identical. We noted a similar proportion of patients designated as unfit for oAAA (22% of eligible EVAR-1 patients placed in EVAR 2 versus 19% of VSGNE EVAR patients deemed unfit). However, designation as unfit for oAAA in our series is, by definition, subjective, and physicians did not have to define these criteria to make this designation as in EVAR-2. Thus, the EVAR-2 patient cohort may represent a more morbid patient group. When our present cohort is stratified by EVAR-2 criteria, survival more closely resembles the EVAR-2 trial results (46% survival; 95% CI, 34% to 58%) at 5 years (Figure 2). This highlights the effect of major patient morbidities on their long-term survival and that other nonmeasured risk factors exist that may be detected by physician assessment.

In an effort to understand the outcomes of EVAR in patients unfit for oAAA, Sobocinski et al[11] report the outcomes of a cohort of 469 patients undergoing EVAR, of whom 191 (41%) meet the EVAR-2 criteria for unfit for oAAA. In their report, they describe a 30-day mortality rate of 1.6%, similar to our current work (1%). Additionally, they reported a 2-year survival rate of 84% at 2 years,[11] which was identical to our 2-year survival estimate of 84%. In another study, the Society for Vascular Surgery performed an analysis of patients meeting the EVAR-2 criteria for high risk from composite data of 5 investigational device exemption device trials. Thirty-day mortality in high-risk patients was 2.9% and 4-year survival after EVAR was only 56%. Overall, these data suggest that the perioperative mortality for high-risk EVAR patients ranges from 1% to 10% by risk strata, and long-term (5 year) survival is poor[12]—both findings reflected in our current work.

In addition to reinforcing the findings from these prior studies, our study also indicates that subjective physician assessment adds accuracy to risk prediction, beyond objective comorbidity profiles. We suspect that if all risk factors were precisely determined in a dataset such as VSGNE, physician subjective designation as unfit for open repair might not remain an independent predictor. However, given currently

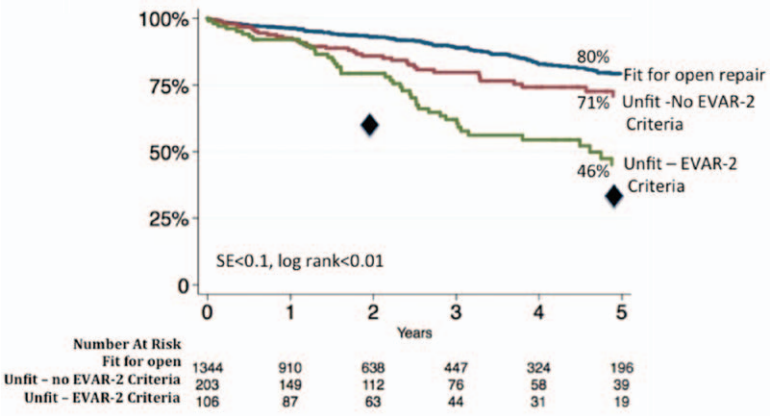

Figure 2. Survival after endovascular aneurysm repair (EVAR) within the Vascular Study Group of New England by EVAR-2 Trial Criteria designation. Diamond indicates survival in treated cohort of EVAR 2, 60% at 2 years and 34% at 5 years. EVAR-2 Criteria are recent myocardial infarction or angina, unstable angina, severe valvular disease, significant arrhythmia, uncontrolled congestive heart failure, severe chronic obstructive pulmonary disease, or poor renal function.

available data, subjective surgeon assessment of fitness for oAAA does have independent prognostic value. This is supported by our multivariable model predicting unfit for open repair, which had an area under the curve of 0.79. Thus, clinical variables alone cannot fully account for a patient's fitness for oAAA, leaving unaccounted for factors to be assessed by the operating surgeon.

How should we determine whether patients who are unfit for open repair should undergo any repair at all? Although a challenging question, several options exist to address this gap. First, one could repeat the EVAR-2 trial and attempt to ensure better adherence to randomization and treatment schemes. However, given the current broad acceptance of EVAR, especially in the United States, such a trial would be difficult to perform. Similarly, thoracic aortic aneurysm repair represents a similar disease process with distinct risks and benefits, and there is unlikely to be a trial of open repair and TEVAR. However, our group has seen similar trends in the use of TEVAR for thoracic aortic aneurysm repair, suggesting that many medically unfit patients may receive treatment with minimal survival benefit.[13] A more realistic approach would be to evolve our current procedural-based registries[14] into disease-based registries. This would allow us to follow a cohort of patients with aneurysms (rather than a cohort of patients with aneurysm repairs) over time. Although observational in nature, the clinical details in such a registry would reveal a group of high-risk patients who obviously would never benefit from prophylactic repair. Simply by appropriate patient selection, some high-risk patients would undergo EVAR, whereas others would not. This would allow examination of survival differences between treated and untreated patients, all of whom have some element of being unfit for open repair. These natural experiments, combined with analytic strategies aimed at accounting for measurable and unmeasurable selection bias (such as propensity scores and instrumental variables),[15] may address the limitations in our current study within a framework that engenders less expense, time, and complexity when compared with a randomized trial.

A potential criticism of our analysis is that we have excluded patients with aneurysms ≥6.5 cm in maximum diameter. This was chosen because larger aneurysms are at high risk for rupture.[10] Patients with large aneurysms are also often at high risk for complications, have high comorbid disease burdens, and have shorter survival. Thus, the risk/benefit ratio is altered for these patients. We have attempted to homogenize our analysis to those with similar risk/benefit ratios to aid in treatment decisions. Of note, of our total EVAR population (n=2940), 573 (19%) were >6.4 cm. Inclusion of these patients in our analysis did not change the factors associated with being deemed unfit for open repair or for long-term survival. However, those with larger AAAs did have higher rates of cardiac complications (8% versus 13%, $P<0.01$), perioperative death (1% versus 2%, $P<0.01$), and worse 5-year survival (61% versus 28%, logrank, $P<0.001$).

In conclusion, in patients with AAAs <6.5 cm, the overall assessment of patient's status as being unfit for oAAA is correlated with higher postoperative complications as well as with reduced long-term survival, even after adjusting for other comorbidities. Patient comorbidities, such as advanced age, severe COPD, active cardiac disease, and chronic renal disease, are more likely to be associated with being unfit for open repair. These factors, in addition to being classified as unfit for open repair, are each independently associated with poor long-term survival. Our future work will continue to explore methods to identify which patients are most likely to obtain long-term survival benefit from oAAA.

Disclosures

None.

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