Mortality variability after endovascular versus open abdominal aortic aneurysm repair in a large tertiary vascular center using a Medicare-derived risk prediction model

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Objective: **Previous reports have documented better outcomes after open abdominal aortic aneurysm (AAA) repair in tertiary centers compared with lower-volume hospitals, but outcome variability for endovascular AAA repair (EVAR) vs open AAA repairs in a large tertiary center using a Medicare-derived mortality risk prediction model has not been previously reported. In the current study, we compared the observed vs predicted mortality after EVAR and open AAA repair in a single large tertiary vascular center.**

Methods: **We retrospectively analyzed all patients who underwent repair of a nonruptured infrarenal AAA in our center from 2003 to 2012. Univariable and multivariable logistic regression were used to evaluate 30-day mortality. Patients were stratified into low-risk, medium-risk, and high-risk groups, and mortality was predicted for each patient based on demographics and comorbidities according to the Medicare risk prediction model.**

Results: **We analyzed 297 patients (EVAR, 72%; open AAA repair, 28%; symptomatic, 25%). Most of our patients were of high and moderate risk (48% and 28%, respectively). The observed 30-day mortality was 1.9% after EVAR vs 2.4% after open repair (odds ratio [OR], 0.77; 95% confidence interval [CI], 0.14-4.29; $P = .67$). There was no difference in mortality with EVAR vs open repair after adjusting for predefined patient characteristics (OR, 0.92; 95% CI, 0.16-7.43; $P = .93$); only preoperative renal disease was predictive of 30-day mortality after AAA repair in our cohort (OR, 8.39; 95% CI, 1.41-67.0). The observed mortality within our study was significantly lower than the Medicare-derived expected mortality for each treatment group within patients stratified as high risk or medium risk ($P \leq .0002$ for all).**

Conclusions: **Despite treating patients with high preoperative risk status, we report a 10-fold decrease in operative mortality for EVAR and open AAA repair in a tertiary vascular center compared with national Medicare-derived predictions. High-risk patients should be considered for aneurysm management in dedicated aortic centers, regardless of approach. (J Vasc Surg 2015;61:291-7.)**

Abdominal aortic aneurysms (AAAs) affect between 4% and 8% of people in the United States and account for >7500 deaths annually.[1,2] Historically, the surgical standard of care for symptomatic or large (>5.0 cm) AAAs was open surgical repair. However, the advent of endovascular aneurysm repair (EVAR) in recent years has drastically changed current practice patterns, especially in the presence of data suggesting that perioperative mortality is reduced with EVAR.[3-6] EVAR has also been demonstrated to reduce perioperative complications and hospital length of stay compared with open AAA repair, often making this the favored approach among older and higher-risk patients.[7-12]

Despite this, long-term mortality after EVAR and open AAA repair appears to be similar, and EVAR is associated with higher costs, more intensive follow-up regimens, and an increased need for reintervention postoperatively.[6,9,11,13] As a result, the indications for performing one approach over another are not currently clear. Several algorithms to predict mortality have been developed in an attempt to risk-stratify patients considering surgical repair for AAA, including the Glasgow Aneurysm Score, Leiden Score, Society for Vascular Surgery/American Association for Vascular Surgery Comorbidity Scoring System, Hardman Index, Eagle Score, and Vascular Governance North-West model.[14-20] However, these models were developed based on data only from patients undergoing open AAA repair and have been shown to overpredict mortality in the EVAR population.[18,20] A more recent model proposed by Giles et al[15] used data from 45,660 Medicare beneficiaries to develop a scoring algorithm to predict perioperative (30-day) mortality after EVAR or open AAA repair. Patients are stratified as high risk, medium risk, and low risk for surgical repair based on their age, sex, comorbidities, and proposed surgical approach, generating an individualized risk assessment that can help guide clinical decision making.

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To date, the Giles model has not been applied to assess outcomes after EVAR vs open AAA repair in tertiary centers specifically. Previous studies have demonstrated that early mortality after AAA repair is highly dependent on hospital volume and surgeon experience.[21-23] This effect is thought to be the result of better management of complications such as renal failure at high-volume institutions[24]; the higher mortality that is observed at lower-volume hospitals is thought to be the result of a "failure to rescue" phenomenon.[25] The experience-based outcome variability for EVAR has not yet been evaluated but would presumably follow a similar pattern, with better outcomes observed in more experienced, tertiary centers. In the current study, we compare the perioperative morbidity and mortality observed with EVAR vs open AAA repair at a single large tertiary vascular center with the predicted mortality as generated by application of the Giles risk stratification model.

METHODS

Approval from the Johns Hopkins Hospital Institutional Review Board was obtained before the study was initiated. All patients who underwent elective infrarenal AAA repair at our institution between November 28, 2003, and August 30, 2012, were identified for analysis. The study excluded patients with connective tissue disorders, inflammatory aneurysms, and ruptured aneurysms. The electronic medical records were retrospectively reviewed by two independent study team members to collect data on patient demographics, symptoms, comorbidities, surgical technique, postoperative outcomes, and mortality. Patient comorbidities were abstracted based on physician documentation within the electronic medical record. Disagreements on patient coding were settled by input from a third independent team member.

All patients with postoperative clinical visits <1 month old were contacted by telephone to confirm their mortality status. Our primary outcome was perioperative mortality (≤30 days of surgery) after open AAA repair vs EVAR. Our secondary outcome was the incidence of postoperative acute renal failure (ARF), defined according to Acute Kidney Injury Network guidelines.[26]

Descriptive statistics are described as mean ± standard error of the mean or count with percentage, as appropriate. Univariable statistics were performed using Student *t*-tests for continuous variables or the Fisher or Pearson χ^2 tests, or both, for categoric variables to compare morbidity and mortality between patients undergoing open repair vs EVAR. Multivariable logistic regression was used to evaluate adjusted observed perioperative mortality with open repair vs EVAR after accounting for age, sex, and pertinent patient comorbidities (congestive heart failure [CHF], chronic obstructive pulmonary disease, coronary artery disease, and chronic renal insufficiency [CRI]) chosen based on commonly included covariates from prior AAA risk mortality prediction studies.[15-19,27,28]

Patients were then stratified into low-risk, medium-risk, and high-risk groups using the risk prediction model for perioperative mortality of EVAR vs open AAA repair developed by Giles et al.[15] In this model, each patient's risk of mortality after AAA repair is calculated using a logistic regression equation that considers patient characteristics (age, gender), comorbidities (end-stage renal disease [ESRD], CRI, CHF, peripheral vascular disease, and cerebrovascular disease), and treatment (open vs endovascular approach) to derive an overall risk score: Risk score = 4I (female) + 1I (age 70-75) + 6I (age 76-80) + 11I (age >80) + 9I (ESRD)+ 7I (CRI) + 6I (CHF) + 3I (vascular disease) + 12I (open surgery).

Note that in this equation, "*I*" is event, with I = 1 if the event is true, 0 otherwise. Patient data were obtained from manual record review and input into the Medicare-derived equation to determine an individual overall mortality risk score. Scores >11 were designated as high risk for mortality, scores between 3 and 11 were designated as medium risk, and scores <3 were designated as low risk. For risk stratification calculations performed within treatment groups, the treatment effect coefficient of 12 (open surgery) was excluded, with respective mortality probabilities of <1%, 1% to 2%, and >2% for EVAR and <3%, 3% to 6%, and >6% for open AAA repair, as reported by Giles et al.[15]

In addition, a specific Medicare-derived predicted mortality probability (P) was calculated for each patient based on the same model: logit(P) = −5.02 + 0.42 × female + 0.15 × age(70 to 75) + 0.63 × age(76 to 80) + 1.14 × age(>80) + 0.71 × CRI + 0.95 ×ESRD + 0.55 × CHF + 0.30 × vascular disease + 1.17 × open repair.

Observed vs expected mortality (calculated as observed incidence of mortality/the expected incidence of mortality as calculated based on the mean mortality probability within a given group) was compared within risk groups to assess the applicability of the model to predict mortality within our institution's patient population. Note that within the mortality prediction equation, risk with open AAA repair is weighted 1.17 times more than EVAR. Because we noted a particularly high predicted mortality within our patient cohort, we also performed sensitivity analyses with and without the weighted treatment coefficient for open AAA repair (1.17) to determine whether including it significantly affected the calculated predicted mortality risk.

RESULTS

Observed experience. Overall, 297 patients (79.4% male), with a mean age of 72.8 ± 0.47 years, were identified for inclusion in the study. Of these, 214 (72.1%) underwent EVAR and 83 (27.9%) underwent open AAA repair. Patients undergoing open repair were younger (69.2 ± 0.86 vs 74.3 ± 0.54 years; $P < .0001$) and had a higher prevalence of smoking (71.1% vs 54.7%; $P = .01$) compared with patients undergoing EVAR. There were no significant differences in patient gender, race, comorbidities, or symptomatology between groups (Table I).

Death ≤30 days of surgery occurred in 2.02% (n = 6) of patients overall. The observed perioperative mortality was 1.9% (n = 4) after EVAR vs 2.4% (n = 2) after open repair (odds ratio [OR], 0.77; 95% confidence interval [CI], 0.14-4.29; $P = .67$). There was no difference in

Table I. Patient characteristics by repair type

Characteristic[a]	*Open AAA repair (n = 83)*	*EVAR (n = 214)*	P *value*
Sex			.345
Male	63 (75.9)	173 (80.8)	
Female	20 (24.1)	41 (19.2)	
Age, years	69.2 ± 0.86	74.3 ± 0.54	<.0001
Symptomatic	25 (30.1)	50 (23.4)	.229
Diabetes	12 (14.5)	38 (17.8)	.495
Hypertension	75 (90.4)	180 (84.1)	.165
Dyslipidemia	63 (75.9)	166 (77.6)	.759
CHF	4 (4.8)	24 (11.2)	.091
COPD	25 (30.1)	55 (25.7)	.441
CAD	47 (56.6)	106 (49.5)	.272
Vascular disease	11 (13.3)	39 (18.2)	.304
CRI	19 (22.9)	45 (21.0)	.726
ESRD	3 (3.6)	5 (2.3)	.542
Cancer	18 (21.7)	54 (25.2)	.522
Smoking	59 (71.1)	117 (54.7)	.01

AAA, Abdominal aortic aneurysm; *CAD,* coronary artery disease; *CHF,* congestive heart failure; *COPD,* chronic obstructive pulmonary disease; *CRI,* chronic renal insufficiency; *ESRD,* end stage renal disease; *EVAR,* endovascular AAA repair.

[a]Age is expressed as mean ± standard error of the mean, and categoric variables are shown as number (%).

Table II. Multivariable analysis of mortality after abdominal aortic aneurysm (*AAA*) repair

Covariate	*OR (95% CI)*	P *value*
Age	1.03 (0.92-1.15)	.60
Male gender	0.93 (0.05-7.11)	.95
CHF	0.82 (0.04-7.71)	.88
COPD	2.78 (0.48-16.3)	.24
CAD	0.84 (0.13-5.11)	.85
CRI	8.39 (1.41-67.0)	.02
EVAR vs open AAA repair	0.92 (0.16-7.43)	.93

CAD, Coronary artery disease; *CHF,* congestive heart failure; *CI,* confidence interval; *COPD,* chronic obstructive pulmonary disease; *CRI,* chronic renal insufficiency; *OR,* odds ratio.

mortality with EVAR vs open repair after adjusting for predefined patient characteristics (OR, 0.92; 95% CI, 0.16-7.43; $P = .93$). Only preoperative renal disease was predictive of 30-day mortality after AAA repair in our cohort (OR, 8.39; 95% CI, 1.41-67.0; Table II).

Postoperative ARF occurred in 2.4% (n = 6) of patients overall. ARF was more common after open repair (6.0% [n = 5]) compared with EVAR (0.5% [n = 1]; OR, 14.3; 95% CI, 1.64-125; $P = .006$). However, no patients who developed ARF died secondary to this complication.

Observed vs expected experience. Most patients in our study were categorized as moderate-risk (27.6% [n = 82]) or high-risk (48.1% [n = 143]) for AAA repair according to the mortality risk scoring equation developed by Giles et al[15] (Fig 1). The expected mean operative mortality within our study cohort was 20.9% (95% CI, 17.1%-24.9%) overall, including 23.4% (95% CI, 15.5%-31.3%) after open repair and 20.1% (95% CI, 15.6%-24.6%) after EVAR. Among low-risk open AAA repair patients, the observed vs expected mortality of 3.01% vs 2.1% was similar ($P = .77$). However, the observed mortality was significantly lower than the expected mortality for medium-risk and high-risk open AAA and all EVAR patients regardless of risk stratification ($P \leq .0002$ for all; Fig 2; Table III).

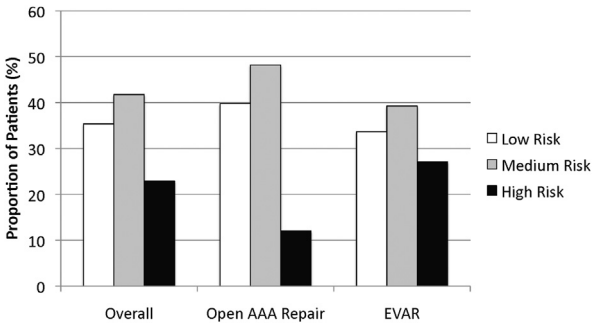

Fig 1. Risk stratification of patients undergoing abdominal aortic aneurysm (*AAA*) repair. Most patients in our study were categorized as moderate risk or high risk for AAA repair surgery according to the mortality risk scoring equation developed by Giles et al.[15] *EVAR,* Endovascular aneurysm repair.

To address the possibility that our predicted mortality might be exaggerated, we performed sensitivity analyses with and without the weighted treatment coefficient for open AAA repair. Results of this analysis demonstrated that the predicted mortality risk for open AAA and EVAR were unchanged, regardless of whether the treatment coefficient was included (P = NS):

Expected mortality for open repair:
- With treatment effect: 23.4%
- Without treatment effect: 17.7%

Expected mortality for EVAR:
- With treatment effect: 20.1%
- Without treatment effect: 20.1%

DISCUSSION

Previous reports have documented better outcomes after open AAA repair in tertiary centers compared with lower-volume hospitals,[29] but outcome variability for EVAR vs open AAA repairs in a large tertiary center using a Medicare-derived mortality risk prediction model has not been previously reported. In the current study, we compared the observed vs predicted mortality after EVAR and open AAA repair in a single large tertiary vascular center. We found that our 30-day mortality rates are much lower than expected with both operative approaches, particularly in patients who were stratified as medium-risk and high-risk. ARF was more common after open repair but did not affect mortality outcomes.

AAA repair is a high-risk operation, with 30-day mortality rates estimated to range between 2% and 5%, depending on the operative approach.[30-33] As a result, predictive

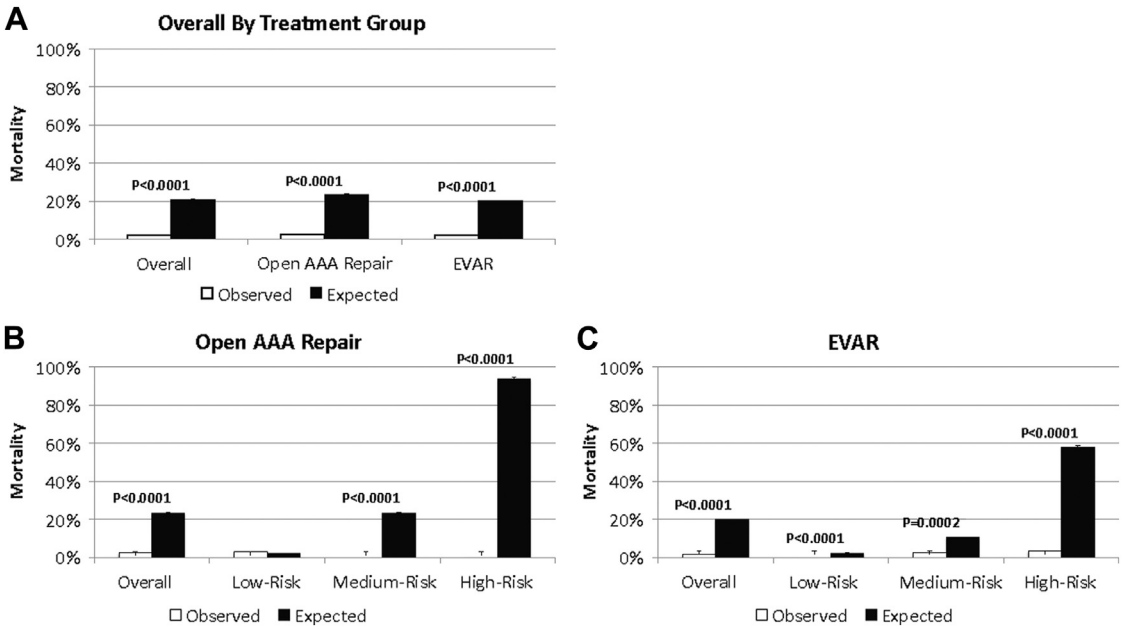

Fig 2. Observed vs expected mortality. **A,** The observed mortality was significantly lower than the expected mortality for each treatment group overall. When stratified by operative risk, low-risk open abdominal aortic aneurysm (*AAA*) patients had similar observed and predicted mortality rates, whereas medium-risk and high-risk **(B)** open AAA and all **(C)** endovascular aneurysm repair (*EVAR*) patients had significantly lower rates.

Table III. Observed vs expected mortality rates after abdominal aortic aneurysm (*AAA*) repair

		30-day mortality		
Treatment group	*No.*	*Observed, % (95% CI)*	*Expected, % (95% CI)*	P *value*
Overall	297	2.0 (0.4-3.6)	20.9 (17.1-24.9)	<.0001
Open AAA repair	83	2.4 (−0.96 to 5.8)	23.4 (15.5-31.3)	<.0001
Low-risk	33	3.0 (−3.1 to 9.2)	2.1 (2.1-2.2)	.77
Medium-risk	40	0	23.3 (12.7-33.8)	<.0001
High-risk	10	0.1 (−12.6 to 32.6)	93.8 (90.0-97.6)	<.0001
EVAR	214	1.9 (0.04-3.6)	20.1 (15.6-24.6)	<.0001
Low-risk	72	0.0	0.7 (0.7-0.7)	<.0001
Medium-risk	84	2.4 (−0.9 to 5.7)	10.6 (6.0-15.1)	.0002
High-risk	58	3.4 (−1.4 to 8.3)	57.9 (47.7-68.0)	<.0001

CI, Confidence interval; *EVAR,* endovascular AAA repair.

risk modeling for AAA repair is becoming increasingly popular for clinical decision making. The idea is that patients will be able to go into surgery being fully informed of their surgical risk with EVAR or open AAA repair, thus enabling them to make a truly informed decision about whether they are willing to accept the higher risks of an open repair vs potential longer-term complications and the need for lifelong surveillance after EVAR.[6] In an effort to facilitate this individualization of care, a number of risk prediction models for AAA have been developed.[14-20,27,34] Most of these models include patient age and comorbidities (specifically, preoperative renal and cardiac disease) to generate a predicted risk of mortality after surgery (Table IV).

In our study, the observed vs predicted mortality rates with EVAR and open AAA repair were drastically different. Of note, the 20.9% predicted mortality of our patient cohort is markedly higher than the standardly reported mortality rates after AAA repair that tend to range between 1% and 5%.[15,35] This likely reflects that many patients are referred to our tertiary institution after being deemed too high-risk for surgery at other centers. Although open repair is generally reserved for younger, healthier patients, many patients who undergo open repair at our institution do so because they have unsuitable anatomy for EVAR even though they have a high clinical risk with open repair. Consistent with this notion, 50 of 83 patients (60%) undergoing open AAA repair were classified as medium-risk or high-risk in our study.

In addition, we failed to demonstrate the short-term mortality benefit with EVAR compared with open AAA

Table IV. Existing abdominal aortic aneurysm (AAA) mortality risk prediction algorithms

Risk prediction model	*Scoring algorithm*
Glasgow Aneurysm Score[19]	Risk score = age in years + (7 for myocardial disease) + (10 for cerebrovascular disease) + (14 for renal disease)
Leiden Risk Model[18]	Risk score = (−4 if age <60; +0 if age = 70; +4 if age >80) + (4 for female gender) + (3 for history of MI) + (8 for CHF) + (8 for electrocardiographic evidence of ischemia) + (12 for renal disease) + (7 for pulmonary disease)
SVS/AAVS Comorbidity Scoring System[16]	Grades (absent = 0; mild = 1; moderate = 2; severe = 3) given for cardiac, pulmonary, renal, and hypertensive disease as well as for age (0 if age <55; 1 if age 55-69; 2 if age 70-79; 3 if age >80). Risk score = (age) + (4 × cardiac) + (2 × pulmonary) + (2 × renal) + (hypertension)
Hardman Index[17]	Risk score = (+1 if age >76) + (1 if creatinine >176) + (1 if hemoglobin <9) + (1 if electrocardiographic evidence of ischemia) + (1 if loss-of-consciousness on arrival)
Eagle Score[27]	Risk score = 0.077 × (age − 10) + (1 if history of angina) + (1.4 if Q wave on electrocardiogram) + (1.2 if history of ventricular ectopic activity) + (1 if history of diabetes) + (1.3 if ischemic electrocardiographic changes) + (2.3 if redistribution of thallium)
Giles Medicare Score[15]	Risk score = (+1 if age 70-75; +6 if age 76-80; +11 if age >80) + (4 if female) + (9 if ESRD) + (7 if CRI + (6 if CHF) + (3 if vascular disease) + (12 if open repair)
VGNW Model[28]	Odds = exp (−9.3431 + [0.0486 × age (continuous in years)] + [0.7322 × female sex] + [0.6620 × diabetes] + [0.0073 × creatinine (continuous in μmol/L)] + [0.4718 × respiratory disease] + [0.7762 × antiplatelet medication] + [1.3130 × open surgery]). Risk = [odds/(1 + odds)] × 100

AAVS, American Association for Vascular Surgery; *CHF,* congestive heart failure; *CRI,* chronic renal insufficiency; *ESRD,* end-stage renal disease; *MI,* myocardial infarction; *SVS,* Society for Vascular Surgery; *VGNW,* Vascular Governance NorthWest.

repair that has been reported in prior studies.[6] The differences in our data compared with previously published data are also most likely a reflection of institutional practice patterns, whereby many high-risk patients regularly undergo extensive surgical interventions.

Among centers that routinely treat patients who would likely not be offered surgery at smaller institutions, multidisciplinary approaches to patient care and a strict adherence to hospital quality guidelines are common.[36-38] For example, the 6% rate of postoperative ARF after open AAA repair in our study was similar to that previously reported in the literature[14] and significantly higher than the ARF rate after EVAR. However, all patients undergoing AAA repair at our institution are monitored by a multidisciplinary intensive care team that includes physicians from a variety of training backgrounds, as well as intensivists, pharmacists, physical and occupational therapists, and nutritionists. As a result, the generally recognized postoperative risks for patient mortality after AAA repair may be minimized by early recognition and involvement of expert clinicians to help optimize each patient's individual postoperative course.

A large study by Ghaferi et al,[24] found that hospital mortality after inpatient surgery was dependent on the early recognition and management of major postoperative complications rather than an actual reduction in complication rates. More recently, Waits et al[39] demonstrated that a large proportion of the variation reported after AAA repair is driven by "failure to rescue" or death by a major postoperative complication. These findings support the concept that hospital-level factors and safety culture likely play a large role in postoperative outcomes within specific patient populations and may partially explain why tertiary institutions such as our own routinely report better outcomes after AAA repair than are reported in national databases.

The problem with using existing AAA repair risk prediction models is that no adjustment is made for surgeon-specific or institution-specific factors that may affect patient outcomes after surgery. Previous studies have demonstrated significant hospital-level effects on outcomes after surgery in many specialties.[22,40-43] With respect to AAA repairs specifically, a large meta-analysis of >100,000 surgeries performed in the United Kingdom between 2001 and 2005 demonstrated an odds of death of 0.67 for procedures performed in high-volume (≥32 cases/y) vs low-volume centers.[23] On the basis of this data, the authors estimate that performing AAA repairs at low-volume centers results in 15 extra deaths per 1000 cases. Similar findings have been reported for EVAR repairs as well,[22] and surgeon experience has also been shown to play a role.[21,44,45]

Despite these data, no current risk prediction models account for surgeon-level or hospital-level effects. One reason for this is likely that most models were developed from single-institution experiences. Although Giles et al[15] developed their model from a broader selection of patients (ie, Medicare beneficiaries), institutional effects were not considered in the analysis. Choke et al[46] recently demonstrated that an "in-house" risk model for elective AAA repair is more accurate in predicting perioperative mortality than existing single-institution or national models, presumably because the model is specific for local demographics, case selection, and practice patterns. The dichotomous observed vs predicted outcomes that we report are likely reflective of the differences in our patient population compared with the standard Medicare beneficiary from which the Giles score was derived. Similar to how the American College of Surgeons National Surgical Quality Improvement Program database reports outcomes using risk-adjusted and case-mix-adjusted modeling,[47] a risk adjustment that accounts

for hospital experience with EVAR and open AAA repair is necessary to truly evaluate a patient's risk with AAA surgery.

The findings that we report are limited by a number of factors, including the retrospective design of our study, relatively small sample size, and lack of long-term outcome data. As with any retrospective record review, there is a risk of inaccurate data reporting. We attempted to minimize this possibility by having two independent researchers collect patient data individually and confer with a third independent party at any point when there was a disagreement. A larger sample size may reduce the differences that we report in our observed vs expect mortality. However, although only 83 patients were in the open AAA repair group, a power calculation based on our sample size (EVAR, 214; open AAA repair, 83) with $\alpha = .05$ demonstrated that our study had 99.9% power to detect a difference in mortality between groups, and it is unlikely that the 10-fold difference in outcomes that we report would have equalized even with a significantly larger cohort.

This is one of the first studies that we know of to apply the Medicare-based prediction modeling for AAA repair to a cohort of patients treated at a tertiary vascular center. With the increasing movement for AAA surgery to be performed at centers of excellence, delineating postoperative outcomes in high-risk patients treated at experienced institutions is essential.[29] Minimal data are available comparing perioperative mortality with EVAR vs open AAA surgery at high-volume vs low-volume institutions. It will be interesting to note whether future long-term studies at practiced vascular centers replicate the long-term differences in outcomes with the different operative approaches that have been previously reported.[6]

CONCLUSIONS

Despite treating patients with high preoperative risk status, we report a 10-fold decrease in operative mortality for EVAR and also open AAA repair in a tertiary vascular center compared with national Medicare-derived predictions. High-risk patients should be considered for aneurysm management in dedicated aortic centers, regardless of approach. Future studies are needed to determine to true extent of surgeon-level and hospital-level effects on operative morbidity and mortality after AAA repair.

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AUTHOR CONTRIBUTIONS

Conception and design: CH, JB, IA, UQ, BP, JF, MM
Analysis and interpretation: CH, JB, IA, LA, UQ, BP, JF, MM
Data collection: CH, IA, LA, UQ
Writing the article: CH, MM
Critical revision of the article: CH, JB, IA, LA, UQ, BP, JF, MM
Final approval of the article: CH, JB, IA, LA, UQ, BP, JF, MM
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