CLINICAL RESEARCH STUDIES

Comparative effectiveness of endovascular versus open repair of ruptured abdominal aortic aneurysm in the Medicare population

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Objective: **Endovascular aortic repair (EVAR) for abdominal aortic aneurysm (AAA) is increasingly used for emergent treatment of ruptured AAA (rAAA). We sought to compare the perioperative and long-term mortality, procedure-related complications, and rates of reintervention of EVAR vs open aortic repair of rAAA in Medicare beneficiaries.**

Methods: **We examined perioperative and long-term mortality and complications after EVAR or open aortic repair performed for rAAA in all traditional Medicare beneficiaries discharged from a United States hospital from 2001 to 2008. Patients were matched by propensity score on baseline demographics, coexisting conditions, admission source, and hospital volume of rAAA repair. Sensitivity analyses were performed to evaluate the effect of bias that might have resulted from unmeasured confounders.**

Results: **Of 10,998 patients with repaired rAAA, 1126 underwent EVAR and 9872 underwent open repair. Propensity score matching yielded 1099 patient pairs. The average age was 78 years, and 72.4% were male. Perioperative mortality was 33.8% for EVAR and 47.7% for open repair ($P < .001$), and this difference persisted for >4 years. At 36 months, EVAR patients had higher rates of AAA-related reinterventions than open repair patients (endovascular reintervention, 10.9% vs 1.5%; $P < .001$), whereas open patients had more laparotomy-related complications (incisional hernia repair, 1.8% vs 6.2%; $P < .001$; all surgical complications, 4.4% vs 9.1%; $P < .001$). Use of EVAR for rAAA increased from 6% of cases in 2001 to 31% in 2008, whereas during the same interval, overall 30-day mortality for admission for rAAA, regardless of treatment, decreased from 55.8% to 50.9%.**

Conclusions: **EVAR for rAAA is associated with lower perioperative and long-term mortality in Medicare beneficiaries. Increasing adoption of EVAR for rAAA is associated with an overall decrease in mortality of patients hospitalized for rAAA during the last decade. (J Vasc Surg 2014;59:575-82.)**

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Despite better preventive practices and increasing rates of repair of intact abdominal aortic aneurysms (AAA) in older and higher-risk populations,[1] ruptured AAA (rAAA) continues to cause >5000 deaths annually in the United States.[2,3] Autopsy data demonstrate that 50% to 70% of patients with rAAA do not survive to hospital presentation.[4] For those that do, the traditional treatment has been emergent open aortic repair, but mortality after open aortic repair remains >40%.[5-7] For intact aneurysms, endovascular aortic repair (EVAR) offers improved perioperative mortality and speedier recovery than open repair,[8-10] and EVAR has become the dominant treatment for intact AAA repair in the U.S.[11] Critically ill patients with rAAA also may benefit from EVAR, but necessary preoperative imaging and specific anatomic requirements can make EVAR less well suited for emergent use. As of 2008, only 31% of rAAA in the U.S. were treated with EVAR, whereas >85% of intact repairs were treated with EVAR.[1,12]

Successful use of EVAR for rAAA was first reported in 1994.[13,14] Subsequent case series and observational studies suggest that for selected patients, EVAR offers improved

mortality compared with open repair.[12,15-22] Conversely, small randomized controlled trials demonstrated no difference in perioperative mortality,[23,24] whereas other trials are ongoing.[25,26]

More than 76% of rAAAs occur in those aged >65 years enrolled in Medicare.[4] Thus, experiences in Medicare provide the most comprehensive data on rAAA available. We sought to compare the perioperative and long-term mortality and short-term and long-term complications in patients receiving EVAR vs open repair for rAAA in the Medicare population. We also examined trends in mortality for rAAA to estimate the overall effect of rising adoption of EVAR on survival after rAAA.

METHODS

The study was approved by the Harvard Medical School Institutional Review Board.

Patients. We identified all Medicare beneficiaries aged ≥67 years who were admitted to a U.S. hospital with a primary discharge diagnosis of rAAA (International Classification of Diseases, 9th Edition-Clinical Modification [ICD-9-CM] code 441.3) between 2001 and 2008. We excluded patients with concurrent diagnoses of thoracic aneurysm (441.1, 441.2), thoracoabdominal aneurysm (441.6 or 441.7), and aortic dissection (441.00-441.03), and those with procedural codes for repair of the thoracic aorta (38.35, 38.45, 39.73) and visceral or renal bypass (38.46, 39.24, 39.26).

To accurately identify rAAAs as distinct from intact AAAs, we analyzed hospital and physician claims and only included patients for whom the diagnosis was consistent in both (Supplementary Fig 1, online only). Overall mortality rates were consistent with those in published reports when both the hospital and physician code were required to indicate rupture. Treatment type was determined by examining ICD-9 and Current Procedural Terminology (CPT) codes (American Medical Association, Chicago, Ill) for open surgical repair (ICD-9 38.44, 39.25, CPT 35081, 35082, 35102, 35103, 35646) and for endovascular repair (ICD-9 39.71, CPT 34800, 34802, 34803, 34804, 34805). When hospital and physician claims conflicted regarding repair type (open vs endovascular), physician claims were given priority.[10] Patients who had EVAR and open repair during the same hospitalization were classified as EVAR patients who had conversion to open repair.

All patients had had at least 2 years of prior Medicare experience, which we used to control for coexisting conditions that might influence outcomes. We excluded beneficiaries enrolled in a Medicare Advantage health plan before or during the index hospitalization.

Creating matched cohorts. To control for the nonrandom assignment of patients to treatment groups, we created matched cohorts of patients after estimating logistic regression models predicting the likelihood of EVAR (propensity score). As explanatory variables, we used baseline demographic and coexisting conditions identified using inpatient and outpatient claims from the 2 years before but not including the index hospitalization.[10,27] We also included as covariates measures of hospital volume of AAA repair, admission through the emergency department, transfer from another hospital, and calendar year. We matched each beneficiary who underwent EVAR with one beneficiary who underwent open repair with the closest estimated propensity to undergo EVAR. We chose one-to-one matching, rather than many-to-one matching, to maximize balance between treatment groups. To ensure close matches, we required the estimated log-odds of endovascular repair between a patient who underwent endovascular repair and one who underwent open repair to be within 0.60 standard deviations. This value removes ~90% of the bias in estimates of effects due to differences in covariate distributions between treatment and comparison groups.[28]

These methods, however, are not able to account for the presence of unmeasured factors that might have influenced the treatment decision. For instance, if hemodynamically unstable patients were preferentially treated with open vs EVAR, our results could be biased in favor of EVAR. To address this issue, we performed an additional sensitivity analysis and an analysis of temporal trends in repair type and mortality.

Mortality outcomes. Perioperative mortality was defined as death during the index hospitalization or ≤30 days of the procedure. Long-term mortality included all deaths during the entire follow-up period. Dates of death were obtained from Medicare enrollment data through December 13, 2010. Claims-based outcomes were censored at December 31, 2008.

Perioperative outcomes and complications. Perioperative complications were assessed by examining diagnosis and procedure codes for the index hospitalization. Complications of interest included conversion of EVAR to open repair, reoperation for bleeding, tracheostomy, embolectomy, wound dehiscence, mesenteric ischemia, bowel resection, myocardial infarction, pneumonia, and renal failure. We also examined length of stay of patients who survived to hospital discharge and discharge disposition to home vs an institutional facility. A full listing of diagnosis codes used to identify complications is included in Supplementary Table II (online only).

Long-term complications. We identified subsequent AAA-related hospitalizations with diagnosis and procedure codes related to AAA, including open surgical reinteventions (open repair of the aneurysm, conversion from endovascular to open repair, repair of a graft-enteric fistula or graft infection, axillobifemoral bypass), endovascular reinterventions (repeat endovascular repair, stent graft extension, embolization, aortic or iliac angioplasty, graft thrombectomy), and laparotomy-related complications, including nonsurgical bowel obstruction, surgical bowel obstruction (obstruction-related lysis of adhesions or bowel resection), and repair of an incisional hernia.

Statistical analysis. We compared the clinical characteristics of the treatment cohorts using χ^2 tests for categoric variables and *t*-tests for continuous variables other than time-to-event variables. Survival time, freedom from

Table I. Patient characteristics

	Unmatched			*Matched*		
Variables	*EVAR (n = 1126)*	*Open (n = 9872)*	P	*EVAR (n = 1099)*	*Open (n = 1099)*	P
Demographics						
Age, mean (SD) years	78.2 (6.6)	77.2 (6.3)	<.001	78.2 (6.6)	78.2 (6.6)	.75
Age, %						
67-69 years	10.39	11.97	<.001	10.65	10.1	.98
70-74 years	23.18	25.32		23.29	22.84	
75-79 years	27.35	31.98		27.48	27.48	
80-84 years	21.31	16.68		21.11	22.11	
≥85 years	17.76	14.04		17.47	17.47	
Male, %	74.2	74.2	.97	74.8	75.1	.88
Race, %						
Black	6.3	3.8	<.001	6.1	6.6	.60
Other	3.1	2.0	.01	2.8	2.3	.42
Source of admission, %						
Emergency department	63.2	80.5	<.001	64.2	63.4	.72
Outside hospital transfer	6.0	2.4	<.001	5.3	5.4	.92
Coexisting conditions, %						
Myocardial infarction						
In past 6 months	1.1	0.9	.66	0.9	0.9	1
In past 7-24 months	7.1	3.8	<.001	7.1	7.1	1
Congestive heart failure	19.0	12.2	<.001	18.4	16.7	.31
Cardiac arrhythmias	24.0	15.6	<.001	23.4	19.3	.02
Valvular disease	11.0	6.9	<.001	10.6	10.7	.94
Peripheral vascular disease	16.7	11.4	<.001	16.3	13.1	.04
Hypertension	61.1	54.4	<.001	60.7	60.2	.83
COPD	31.0	25.8	<.001	30.8	30.2	.78
Diabetes mellitus						
Uncomplicated	16.2	11.8	<.001	15.6	15.8	.86
Complicated	3.9	2.2	<.001	3.6	2.6	.14
Renal disease	8.2	5.4	.02	8.2	8.7	.65
Metastatic cancer	2.7	0.9	<.001	2.6	1.8	.24
Solid tumor	16.0	11.9	<.001	15.9	15.3	.68
Obesity	2.0	1.6	.29	2.0	1.9	.88
ESRD	1.6	0.6	<.001	1.5	1	.33
Cerebrovascular disease	12.7	10.6	.03	12.4	12.0	.79
Intact AAA diagnosis	25.6	16.4	<.001	25.1	22.3	.12

AAA, Abdominal aortic aneurysm; *COPD,* chronic obstructive pulmonary disease; *ESRD,* end-stage renal disease; *EVAR,* endovascular aortic repair; *SD,* standard deviation.

rupture, and time to early and late complications were examined using Kaplan-Meier life-table methods. Differences between groups were tested with the log-rank test. All analyses were performed in SAS 9.3 software (SAS Institute, Cary, NC).

Temporal trends. To further explore if selection contributed to differences in observed mortality for EVAR vs open repair, we examined temporal trends in overall repair rates and 30-day mortality of EVAR and open repair. We used hospital claims to identify all hospitalizations with a discharge diagnosis code of rAAA (441.3). EVAR patients were defined as those who had a concomitant code for an endovascular repair (ICD-9-CM 38.44, 39.25), whereas open repair patients were defined as those who had a claim for open aortic repair (ICD-9-CM 39.71, 39.90). Patients with a claim for neither repair were considered to be unrepaired. If more hemodynamically stable patients were offered EVAR, and more unstable patients were preferentially offered open repair as EVAR was adopted over time, we would expect to see increasing 30-day mortality rates in those treated with open repair.

Sensitivity analysis. To examine the effect of unmeasured confounders, we jointly modeled perioperative mortality (conditional on EVAR) and treatment choice (EVAR vs open repair) according to the methods described by O'Malley et al.[29] In this approach, any unmeasured confounders are absorbed in the errors terms of the patient's underlying propensity to (1) undergo EVAR and (2) suffer perioperative death. By examining the correlation of the error terms in (1) and (2), we can estimate whether a relationship exists between unmeasured factors that make EVAR more likely and unmeasured factors that make mortality more likely. More details on this approach and the results of the sensitivity analysis can be found in the Supplementary Statistical Analysis (online only).

RESULTS

We identified 10,998 patients who underwent repair for rAAA from January 2001 to December 2008.

Of these, 9872 patients underwent open repair and 1,126 underwent EVAR. Baseline characteristics of the patients and their coexisting conditions are listed in Table I. Before matching, patients receiving EVAR tended to be older (78.2 vs 77.2 years; $P < .001$), have more medical diagnoses (eg, 19.0% with congestive heart failure vs 12.2%; $P < .001$), and were more likely to have a pre-existing diagnosis of AAA (25.6% vs 16.4%; $P < .001$). In contrast, patients receiving open repair were more likely to have been admitted through the emergency department (80.5% vs 63.2%; $P < .001$) and were less likely to have been transferred from another hospital (2.4% vs 6.0%; $P < .001$).

Propensity score matching yielded 1099 patient pairs. With the exception of cardiac arrhythmias (16.3% vs 13.1%; $P = .02$) and history of peripheral vascular disease (16.3% vs 13.1%; $P = .04$), there were no remaining statistically significant differences between the populations.

Perioperative outcomes. Perioperative outcomes are summarized in Table II. Perioperative mortality was 33.8% for patients who underwent EVAR vs 47.7% for patients who underwent open repair ($P < .001$). Patients treated with EVAR experienced a lower rate of most postoperative complications, including pneumonia (28.5% vs 35.9% for open repair; $P < .001$), acute renal failure (33.4% vs 45.4%; $P < .001$), respiratory failure requiring tracheostomy (4.6% vs 9.9%; $P < .001$), and gastrointestinal complications, including colon resection (4.4% vs 8.5%; $P < .001$) and mesenteric ischemia (7.6% vs 14.7% $P < .001$). Several procedural complications also were less common in EVAR than in open repair, including embolectomy (3.6% vs 6.3%; $P = .003$) and wound dehiscence (2.5% vs 4.6%; $P = .008$). In contrast, procedure-related hematoma was more common in EVAR patients (8.0% vs 4.5%; $P < .001$), and conversion from EVAR to open repair occurred in 4.9% of patients within the index hospitalization or by 30 days.

Among patients who survived to discharge, the median length of stay for patients undergoing EVAR was 7 days (interquartile range, 4-24 days) compared with 14 days (interquartile range, 9-23 days) for patients undergoing open repair. Discharge was to home for 62.8% of EVAR patients compared with 40.7% of patients who underwent open repair ($P < .001$).

Late outcomes. Long-term survival for the entire cohort and stratified by age is shown in Fig 1. EVAR was associated with a survival benefit that persisted for >4 years after the intervention ($P < .001$) in all age groups.

Open surgical and endovascular AAA-related reinterventions were more common after EVAR than after open repair (Table III). Among EVAR patients, 1.9% underwent open reintervention by 12 months and 3.9% by 36 months vs 0.5% and 0.9% of open repair patients, respectively ($P = .002$ by log-rank test). In addition, an endovascular reintervention had occurred in 4.6% of EVAR patients by 12 months and in 10.9% by 36 months vs 0.6% and 1.5%, respectively, for patients who had open repair ($P < .001$ by log-rank test).

Table II. Perioperative outcomes

Outcome	*EVAR (n = 1099)*	*Open (n = 1099)*	P
Perioperative mortality, %			
All ages	33.8	47.7	<.001
67-69 years	18.8	38.7	<.001
70-74 years	27.7	41.0	<.001
75-79 years	30.1	43.0	<.001
80-84 years	39.2	53.5	<.001
≥85 years	50.0	61.5	.02
Medical complications, %			
Myocardial infarction	16.7	19.2	.13
Pneumonia	28.5	35.9	<.001
Acute renal failure	33.4	45.4	<.001
Hemodialysis	0.7	0.6	.80
Respiratory failure/ tracheostomy	4.6	9.9	<.001
Venous thromboembolism	8.0	6.8	.29
Surgical complications, %			
Conversion to open repair	4.9		
Reoperation for bleeding	2.3	3.0	.24
Embolectomy	3.6	6.4	.003
Wound dehiscence	2.5	4.6	.008
Operative site hematoma	8.0	4.5	<.001
Gastrointestinal bleeding	10.3	13.8	.01
Mesenteric ischemia	7.6	14.7	<.001
Bowel obstruction or ileus	12.7	17.0	.005
Colon resection	4.4	8.5	<.001
Ostomy	2.64	5.28	.002
Discharge to home, %	62.8	40.7	<.001
LOS of survivors to hospital discharge, median (IQR) days	7 (4-24)	14 (9-23)	<.001

EVAR, endovascular aortic repair; *IQR*, interquartile range; *LOS*, length of stay.

Laparotomy-related complications at 36 months were more common in patients who had had open aortic repair, including incisional hernia repair (6.2% vs 1.8% for EVAR; $P < .001$), nonsurgical bowel obstruction (35.8% vs 18.7% for EVAR), and any obstruction-related surgical intervention (9.1% vs 4.4% for EVAR).

Trends in rAAA mortality. Trends of EVAR use and 30-day mortality by treatment type are presented in Fig 2. Use of EVAR for rAAAs increased from 6% of repairs in 2001 (220 cases, 4.2% of total rAAA admissions) to 31% of repairs in 2008 (828 cases, 20% of total rAAA admissions). The number of untreated rAAAs remained nearly constant, but the proportion of untreated rAAAs increased from 27% in 2000 to 38.7% in 2004 and then decreased to 34.5% in 2008. Perioperative mortality for rAAA treated by EVAR decreased from 46% in 2001 to 27% in 2008, and mortality for rAAA treated by open repair decreased from 44.7% to 40%. The 30-day mortality of rAAA without treatment remained ~80%. Overall mortality for rAAA admissions, whether treated or not, decreased from 55.8% to 50.9% between 2001 and 2008.

Sensitivity analysis. As described in the Supplementary Statistical Analysis (online only), our analyses of unmeasured selection suggest that patients whose unobserved factors make them more likely to be treated with EVAR have a higher predicted mortality than other

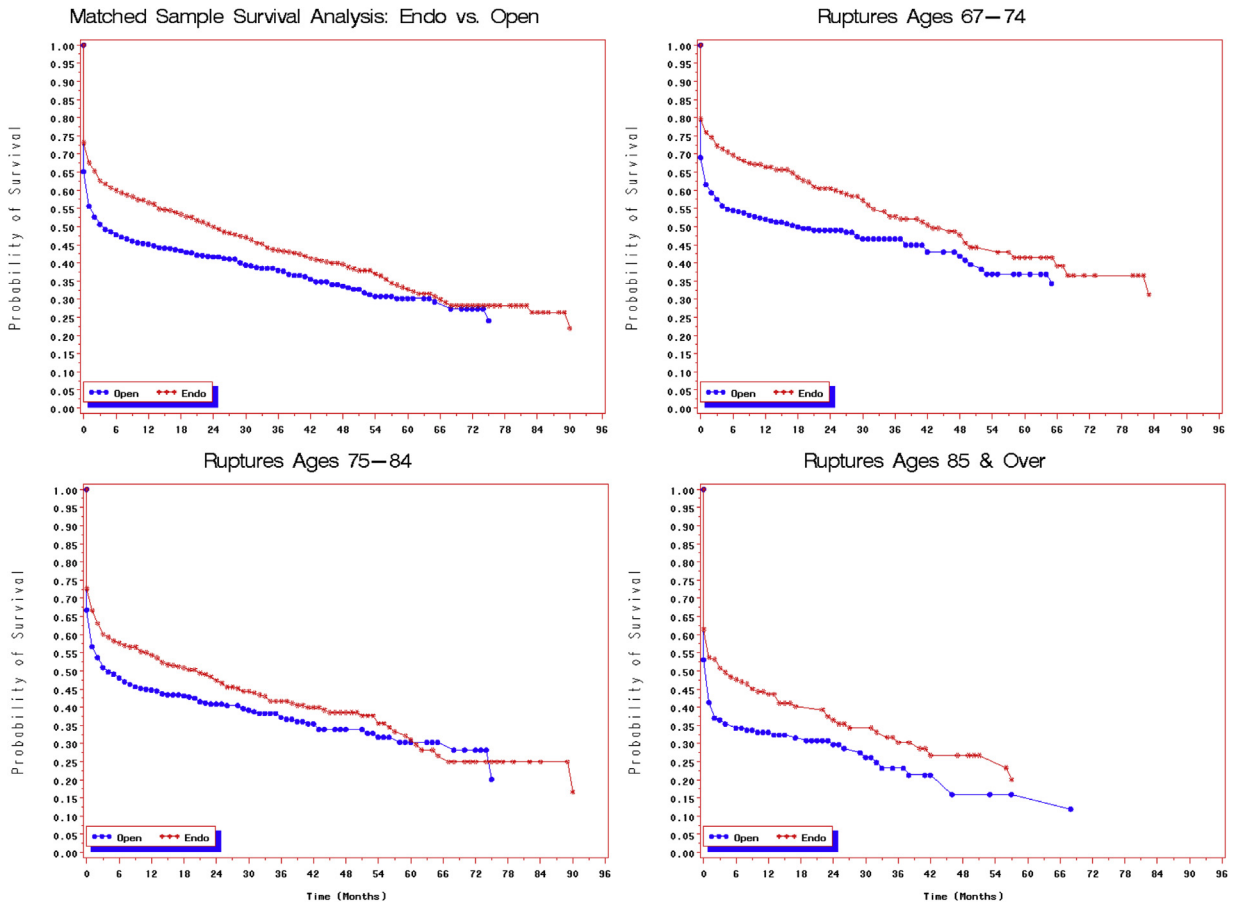

Fig 1. Mortality of propensity-matched patients undergoing endovascular (*Endo*) and open repair of ruptured aortic aneurysm (rAAA) is shown overall and stratified by age.

patients with the same observed predictors. This suggests that any unmeasured confounders were associated with an underestimation rather than an overestimation of treatment effect and would bias our findings against (rather than for) EVAR (Supplementary Statistical Analysis, online only).

DISCUSSION

In this comprehensive analysis of Medicare patients, we found that EVAR of rAAA was associated with lower perioperative and long-term mortality, fewer in-hospital complications, and shorter length of stay. The long-term survival benefit of EVAR persisted for >4 years. Consistent with our prior work on intact AAA repair, patients undergoing EVAR had a higher likelihood of reintervention for AAA, but this was balanced by lower rates of laparotomy-related interventions. During this interval, overall mortality of patients admitted to a hospital with an rAAA, regardless of intervention, declined nearly 5%, whereas the proportion of repairs performed using EVAR increased from 6% to 31%.

Because our study is observational, unmeasured selection remains of significant concern. Most important, it is possible that hemodynamically unstable patients are preferentially offered open repair, which would result in higher mortality for patients treated with open repair. We used three approaches to mitigate this concern:

First, we created matched cohorts using propensity score models that used all available patient factors, including age, sex, race, ethnicity, coexisting conditions, and hospital-level factors such as AAA repair volume. Because patients transferred from other hospitals rather than those admitted from the emergency department are more likely to be hemodynamically stable and have a contained rupture, we also included in our propensity model an indicator variable for transfer from another hospital as well as admission through the emergency department. We observed that more patients who received EVAR had a prior diagnosis of intact AAA, were less commonly admitted through the emergency department, and were more commonly transferred between hospitals before treatment. In our final propensity-matched analysis, these variables were well balanced between groups (Table I), which should eliminate bias associated with these conditions.

Table III. Late outcomes

Outcome	12 months		36 months		
	EVAR, %	*Open*, %	*EVAR*, %	*Open*, %	P[a]
Reintervention					
Open surgical[b]	1.9	0.5	3.9	0.9	.002
Endovascular[c]	4.6	0.6	10.9	1.5	<.001
Laparotomy-related complications					
Bowel obstruction					
Nonsurgical admission[d]	7.3	15.4	18.7	35.8	<.001
Surgical[e]	0.7	1.8	2.5	3.0	.157
Incisional hernia	0.3	1.2	1.8	6.2	<.001
Any surgical intervention[f]	1.0	3.0	4.4	9.1	<.001

EVAR, Endovascular aortic repair.
[a]Late outcomes were analyzed using survival analysis, and *P* values are those generated by the log-rank test.
[b]Conversion to open repair, open aneurysm repair, aortobifemoral bypass, axillofemoral or axillobifemoral bypass, repair of infected graft or graft-enteric fistula, thrombectomy, femoral-femoral bypass.
[c]Repeat EVAR, embolization, angioplasty (aortic or iliac), extension cuff.
[d]Hospital admission with diagnosis of bowel obstruction without lysis of adhesions or bowel resection.
[e]Lysis of adhesions or bowel resection performed for diagnosis of obstruction.
[f]Surgical intervention for bowel obstruction or incisional hernia.

Second, we performed a sensitivity analysis that simultaneously modeled the selection effect and outcomes.[29] The results of this sensitivity analysis suggest that unobserved features that make EVAR more likely are modestly associated with higher mortality. Hence, this suggests that *sicker* patients may be selected for EVAR, thus biasing our results in favor of open repair.

Finally, we examined overall trends in rupture repair and mortality. In the decades preceding the introduction of EVAR, the rate of repair and mortality associated with rAAA repair was stable.[6] Since the introduction of EVAR, however, overall mortality for rAAA hospitalizations, with or without treatment, has decreased.[1] During the same period, the proportion of rAAA repairs performed by EVAR increased, whereas the mortality for EVAR and open repair for rAAA decreased. If improved mortality in EVAR patients were due to selection of healthier patients, we would expect to see an increase in mortality for patients undergoing open repair as EVAR was adopted. Instead, we see a decrease in mortality with open rAAA repair, consistent with sicker patients being offered EVAR. This finding contrasts with previous work where we showed that mortality after open intact AAA repair remained stable during this same interval.[10] Thus, it is unlikely that our results could be explained by more unstable patients being preferentially treated with open repair rather than with EVAR.

We also examined trends in unrepaired rAAAs. The improved outcomes with EVAR and open repair over time could be explained by an increasing proportion of patients being turned down for surgical intervention. Although we saw an increase in the proportion of unrepaired cases of all rAAA admissions with a concomitant decrease in the proportion of open repairs from 2001 to 2003 (Fig 2), this trend reversed after 2003, and a steady decline occurred in the percentage of unrepaired rAAAs. Also during this period, the proportion of EVAR increased, and the proportion of open repair continued to decline. Hence the observed improved mortality for all ruptures, regardless of treatment, may be driven by improved outcomes in EVAR vs open repair and by the adoption of EVAR being associated with an increasing proportion of patients with rAAA receiving any treatment.

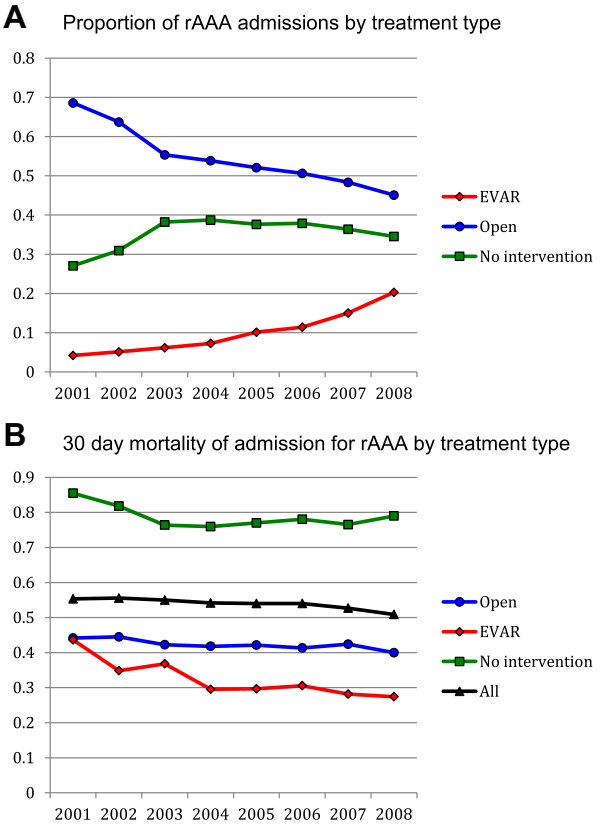

Fig 2. Trends in ruptured abdominal aortic aneurysm (*rAAA*) treatment and 30-day mortality by treatment type from 2001 to 2008. **A,** Proportion of rAAA admissions by treatment type. **B,** 30-day mortality of admission for rAAA by treatment type. *EVAR*, Endovascular aortic repair.

Our results also are consistent with clinical reports of the use of EVAR for rupture patients. Some centers perform EVAR on patients in shock without preoperative imaging[17] and have optimized protocols to expedite the use of EVAR in critically ill patients[18,19] Some have also argued that EVAR may be of most benefit to the most critically ill because they are least likely to survive an open procedure.[30] These authors describe a protocol of rapid transport to the operating room with percutaneous femoral access with an awake patient through which an aortic occlusion balloon may be inserted for rapid supraceliac

aortic occlusion without opening the abdomen; thus avoiding the commonly observed loss of hemodynamic stability associated with the release of the hemoperitoneum. At this point, the surgeon may proceed with EVAR if appropriate or open repair if EVAR is not possible based on anatomy. The adoption of such protocols and the multispecialty coordination and training may also help improve mortality with open repair as well.

One surprising finding is that in our cohort, patients with unrepaired rAAA had a 30-day mortality of ~80%, in contrast to previous findings that >98% of patients with unrepaired rAAA die ≤30 days.[31] There are several possible explanations for this. One possibility is that some patients are coded as rAAA as a "rule out" diagnosis and that they do not actually have an rAAA. Another possibility is that some "unoperated-on" patients identified using hospital claims for our trends analysis received repair but were not correctly coded. Importantly for our analysis, we have no reason to suspect that coding practices changed over time, and hence, our analyses of trends in repair type and mortality should be unaffected. Given the concern of diagnostic accuracy of hospital claims alone, for our primary analysis of mortality, early and late complications of EVAR and open repair, we used both physician and hospital claims to ensure a more homogenous cohort of rAAA patients (Supplementary Fig 1, online only).

Although our findings are consistent with prior studies of rAAA in Medicare patients,[20] our results extend earlier work in several ways:

First, we include more recent data, with procedures performed up to 2008. The period 2005 to 2008 demonstrated the fastest growth of EVAR for rAAA, and these more recent data better reflect current approaches.

Second, we report rates of perioperative and postoperative complications, rates of reintervention, discharge disposition, and length of stay, which have not been reported in the Medicare population.

Third, we incorporate longer-term data on outcomes and show that the benefits of EVAR are durable for >4 years. This is longer than has been seen in trials for EVAR for intact AAA, which may indicate a larger survival advantage for rAAA than for intact AAA using endovascular approaches.

Fourth, our approach incorporates physician diagnoses in addition to hospital discharge data. When hospital coding is compared with physician coding, we find significant discrepancies (Supplementary Fig 1, online only), suggesting substantial potential for misclassification of intact AAAs as rAAAs. Although our approach likely excluded some true rupture cases from our analyses, by requiring consistent coding of rAAA across physician and hospital claims, we generated a cohort that is likely less contaminated by these incorrectly coded cases. Because 80% of intact AAAs are now repaired using EVAR and given the substantially lower perioperative mortality seen with EVAR for intact AAA, inappropriately including intact AAAs in our cohort likely would bias our results in favor of EVAR. Our approach attempted to minimize the likelihood of this occurrence by using the strictest criteria possible to identify cases of rAAA from the administrative data.

Our results are in contrast to two randomized controlled trials that showed no mortality benefit for EVAR vs open repair for rAAA.[23,24] In general, these trials were small (<150 patients) and, hence, had limited power to detect the mortality differences seen in our work and less power to detect the early and late complications we report. These trials also required preoperative imaging to assess eligibility for EVAR, thus excluding patients too unstable to undergo a computed tomography scan and limiting generalizability of their results. Larger, more inclusive clinical trials are ongoing.[25]

Our work has several limitations. Administrative data are subject to coding errors and variability, and the lack of clinical data made it impossible to validate the coding of rAAA using other sources such as imaging results. We did make efforts to minimize errors in the initial diagnosis by requiring consistent physician and hospital claims, although this did exclude many potential cases (Supplementary Fig 1, online only). We also did not have data regarding aneurysm anatomy, which is important in determining whether a patient can receive EVAR, but we attempted to account for this by eliminating patients with renal or visceral bypass or involvement of the thoracic aorta.

CONCLUSIONS

EVAR is associated with improved perioperative and long-term mortality for rAAA, shorter length of stay, and decreased laparotomy-related complications. The use of EVAR for rAAA increased from 2001 to 2008, and the overall mortality for patients hospitalized for rAAA decreased from 55.8% to 50.9%. As centers continue to adopt EVAR and current centers refine protocols for its emergent use for rAAA, there will be further opportunities to study the effects of EVAR on rAAA outcomes.

AUTHOR CONTRIBUTIONS

Conception and design: SE, MS, AO, RB, RH, PC, BL
Analysis and interpretation: SE, MS, AO, RB, RH, PC, BL
Data collection: PC
Writing the article: SE, MS, AO, BL
Critical revision of the article: SE, MS, AO, RB, RH, PC, BL
Final approval of the article: SE, MS, AO, RB, RH, PC, BL
Statistical analysis: AO, PC
Obtained funding: MS, BL
Overall responsibility: BL

REFERENCES

1. Schermerhorn ML, Bensley RP, Giles KA, Hurks R, O'malley AJ, Cotterill P, et al. Changes in abdominal aortic aneurysm rupture and short-term mortality, 1995-2008: a retrospective observational study. Ann Surg 2012;256:651-8.
2. Giles KA, Pomposelli F, Hamdan A, Wyers M, Jhaveri A, Schermerhorn ML. Decrease in total aneurysm-related deaths in the era

of endovascular aneurysm repair. J Vasc Surg 2009;49:543-50; discussion: 550-1.
3. Kochanek KD, Xu J, Murphy SL, Miniño AM, Kung HC. Deaths: final data for 2009. Centers for Disease Control. Nat Vital Stat Rep 2011;59:1-51.
4. Bengtsson H, Bergqvist D. Ruptured abdominal aortic aneurysm: a population-based study. J Vasc Surg 1993;8:74-80.
5. Bown MJ, Sutton AJ, Bell PR, Sayers RD. A meta-analysis of 50 years of ruptured abdominal aortic aneurysm repair. Br J Surg England 2002;89:714-30.
6. Heller JA, Weinberg A, Arons R, Krishnasastry KV, Lyon RT, Deitch JS, et al. Two decades of abdominal aortic aneurysm repair: have we made any progress? J Vasc Surg 2000;32:1091-100.
7. Visser P, Akkersdijk GJ, Blankensteijn JD. In-hospital operative mortality of ruptured abdominal aortic aneurysm: a population-based analysis of 5593 patients in The Netherlands over a 10-year period. Eur J Vasc Endovasc Surg 2005;30:359-64.
8. 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.
9. 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.
10. 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.
11. Landon BE, O'Malley AJ, Giles K, Cotterill P, Schermerhorn ML. Volume-outcome relationships and abdominal aortic aneurysm repair. Circulation 2010;122:1290-7.
12. Mcphee J, Eslami MH, Arous EJ, Messina LM, Schanzer A. Endovascular treatment of ruptured abdominal aortic aneurysms in the United States (2001-2006): a significant survival benefit over open repair is independently associated with increased institutional volume. J Vasc Surg 2010;49:817-26.
13. Marin ML, Veith FJ, Cynamon J, Sanchez LA, Lyon RT, Levine BA, et al. Initial experience with transluminally placed endovascular grafts for the treatment of complex vascular lesions. Ann Surg 1995;222:449-65; discussion: 465-9.
14. Yusuf SW, Whitaker SC, Chuter TA, Wenham PW, Hopkinson BR. Emergency endovascular repair of leaking aortic aneurysm. Lancet 1994;344:1645.
15. Alsac JM, Desgranges P, Kobeiter H, Becquemin JP. Emergency endovascular repair for ruptured abdominal aortic aneurysms: feasibility and comparison of early results with conventional open repair. Eur J Vasc Endovasc Surg 2005;30:632-9.
16. Najjar SF, Mueller KH, Ujiki MB, Morasch MD, Matsumura JS, Eskandari MK. Percutaneous endovascular repair of ruptured abdominal aortic aneurysms. Arch Surg 2007;142:1049-52.
17. Veith FJ, Lachat M, Mayer D, Malina M, Holst J, Mehta M, et al. Collected world and single center experience with endovascular treatment of ruptured abdominal aortic aneurysms. Ann Surg 2009;250:818-24.
18. Mehta M, Taggert J, Darling RC 3rd, Chang BB, Kreienberg PB, Paty PS, et al. Establishing a protocol for endovascular treatment of ruptured abdominal aortic aneurysms: Outcomes of a prospective analysis. J Vasc Surg 2006;44:1-8; discussion: 8.
19. Nedeau AE, Pomposelli FB, Hamdan AD, Wyers MC, Hsu R, Sachs T, et al. Endovascular vs open repair for ruptured abdominal aortic aneurysm. J Vasc Surg 2012;55:1001-7.
20. Egorova N, Giacovelli J, Greco G, Gelijns A, Kent CK, McKinsey JF. National outcomes for the treatment of ruptured abdominal aortic aneurysm: comparison of open versus endovascular repairs. J Vasc Surg 2008;48:1092-100. 1100.e1-2.
21. Holt PJ, Karthikesalingam A, Poloniecki JD, Hinchliffe RJ, Loftus IM, Thompson MM. Propensity scored analysis of outcomes after ruptured abdominal aortic aneurysm. Br J Surg 2010;97:496-503.
22. Giles KA, Hamdan AD, Pomposelli FB, Wyers MC, Dahlberg SE, Schermerhorn ML. Population-based outcomes following endovascular and open repair of ruptured abdominal aortic aneurysms. J Endovasc Ther 2009;16:554-64.
23. Hinchliffe RJ, Bruijstens L, MacSweeney ST, Braithwaite BD. A randomised trial of endovascular and open surgery for ruptured abdominal aortic aneurysm—results of a pilot study and lessons learned for future studies. Eur J Vasc Endovasc Surg 2006;32:506-13; discussion: 514-5.
24. Reimerink JJ, Hoornweg LL, Vahl AC, Wisselink W, van den Broek TA, Legemate DA, et al. Endovascular repair versus open repair of ruptured abdominal aortic aneurysms: a multicenter randomized controlled trial. Ann Surg 2013;258:248-56.
25. Powell JT. Time to IMPROVE the management of ruptured abdominal aortic aneurysm: IMPROVE trialists. Eur J Vasc Endovasc Surg 2009;38:237-8.
26. Desgranges P, Kobeiter H, Castier Y, Sénéchal M, Majewski M, Krimi A. The Endovasculaire vs Chirurgie dans les Anévrysmes Rompus PROTOCOL trial update. J Vasc Surg 2010;51:267-70.
27. Elixhauser A, Steiner C, Harris DR, Coffey RM. Comorbidity measures for use with administrative data. Med Care 1998;36:8-27.
28. Cochran W. Controlling bias in observational studies: a review. Sankhya Ser A 1973;35:427-9.
29. O'Malley AJ, Cotterill P, Schermerhorn ML, Landon BE. Improving observational study estimates of treatment effects using joint modeling of selection effects and outcomes: the case of AAA repair. Med Care 2011;49:1126-32.
30. Veith FJ, Powell JT, Hinchliffe RJ. Is a randomized trial necessary to determine whether endovascular repair is the preferred management strategy in patients with ruptured abdominal aortic aneurysms? J Vasc Surg 2010;52:1087-93.
31. Lindholt JS, Søgaard R, Laustsen J. Prognosis of ruptured abdominal aortic aneurysms in Denmark from 1994-2008. Clin Epidemiol 2012;4:111-3.

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Supplementary Statistical Analysis (online only).

Our primary sensitivity analysis was designed to test for the extent to which unmeasured differences in health status might be biasing our results. Although propensity score methods account for confounding by observed predictors, they offer no reassurance against unobserved confounders. One approach to account for unmeasured confounding is to jointly model perioperative mortality conditional on endovascular aortic repair (EVAR) and use of EVAR vs open repair. Because perioperative mortality and EVAR are dichotomous variables, the bivariate probit model[1-5] may be used to account for unmeasured confounding while modeling the effects of treatment (EVAR vs open) and other observed predictors of perioperative mortality.

This model is defined by a simultaneous equation system, wherein an equation for treatment selection is linked to an equation for the outcome from treatment through their shared dependence on unmeasured variables. The direction and magnitude of the net effect of the unmeasured confounders is quantified by the extent to which the relationship between perioperative mortality and EVAR deviates from that predicted based on the observed predictors. The size and magnitude of this deviation is quantified in terms of a correlation coefficient. The further the correlation is from 0, the more that unmeasured selection effects are expected to yield biased estimates under a propensity score or other method reliant on the nonexistence of unmeasured confounders. For a complete description of the bivariate probit model in the context of a comparison between EVAR and open repair, please refer to O'Malley et al.[6]

The results of the bivariate probit analysis are presented in Table A; estimates (together with t values and P values) for the equation predicting the likelihood of perioperative mortality are presented in the first three columns to the right of the list of predictors, whereas those for the equation predicting the likelihood of undergoing endovascular surgery are presented in the right-most three columns. Those predictors that are particular to a given model (eg, the effect of EVAR on mortality) only have estimates for that model.

The effect of EVAR (the effect of primary interest) is significant and negative (-0.630; $P = .0292$) implying that the risk of perioperative mortality is lower for EVAR than for open, all else equal. Secondly, the selection effect correlation parameter is estimated to be 0.093 ($P = .250$), suggesting that individuals who are more likely to receive EVAR in unmeasured ways are more likely to suffer perioperative mortality. Therefore, the propensity score analysis that does not adjust for unmeasured confounding is more likely biased against finding a significant effect (as EVAR patients essentially have unmeasured risk factors that place them at greater risk for perioperative mortality) than finding a significant effect when none exists.

Owing to the relative scarcity of EVAR observations, effects estimated for it are less precise than those for open. For example, the effect of institutional EVAR volume has a larger negative coefficient than that for institutional open volume -0.170 vs -0.042 but is less significant ($P = .227$ vs $.011$). Both volume effects are consistent with past findings that the risk of perioperative mortality is lower at institutions with higher annual volumes of the same procedures. Further face validity in the bivariate probit model is seen from the fact that perioperative mortality is much higher for urgent cases (0.088; $P = .004$) and that the relative use of EVAR has increased rapidly over time (effect of 0.008 per day; $P < .0001$). The coherency of these findings with past observations by others offers further evidence that the estimated effects under this model are reliable.

REFERENCES

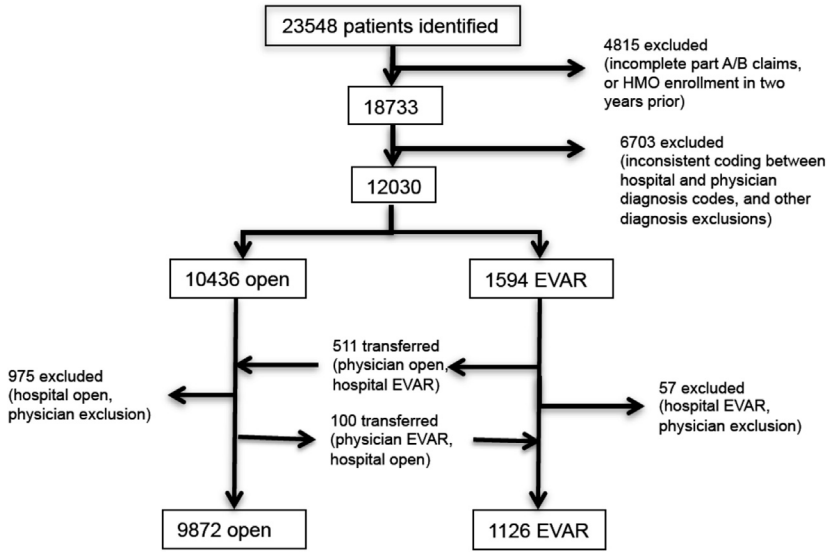
1. Amemiya T. Bivariate probit analysis: minimum chi-square methods. J Amer Statist Assoc 1974;69:940-4.
2. Gitto L. Dialysis modality selection according to the medical care provided at dialysis units: An econometric analysis. Health Serv Res 2004;5:227-41.
3. Zeng F, O'Leary JF, Sloss EM, Lopez MS, Dhanani N, Melnick G. The effect of medicare health maintenance organizations on hospitalization rates for ambulatory care-sensitive conditions. Med Care 2006;44:900-7.
4. Bhattacharya J, Goldman D, McCaffrey D. Estimating probit models with self-selected treatments. Stat Med 2006;25:389-413.
5. Freedman DA, Sekhon JS. Endogeneity in probit response models. Polit Anal 2010;18:138.
6. O'Malley AJ, Cotterill P, Schermerhorn ML, Landon BE. Improving observational study estimates of treatment effects using joint modeling of selection effects and outcomes: the case of AAA repair. Med Care 2011;49:1126-32.
7. Box G, Cox D. An analysis of transformations. J Royal Statist Soc B (Methodological) 1964;26:211-52.

Table A. Parameter estimates of regression coefficients for bivariate probit model adjusting for clinical and nonclinical adjusters, reason, and source of admission, and institutional volume for the same procedure that was performed on the patient.

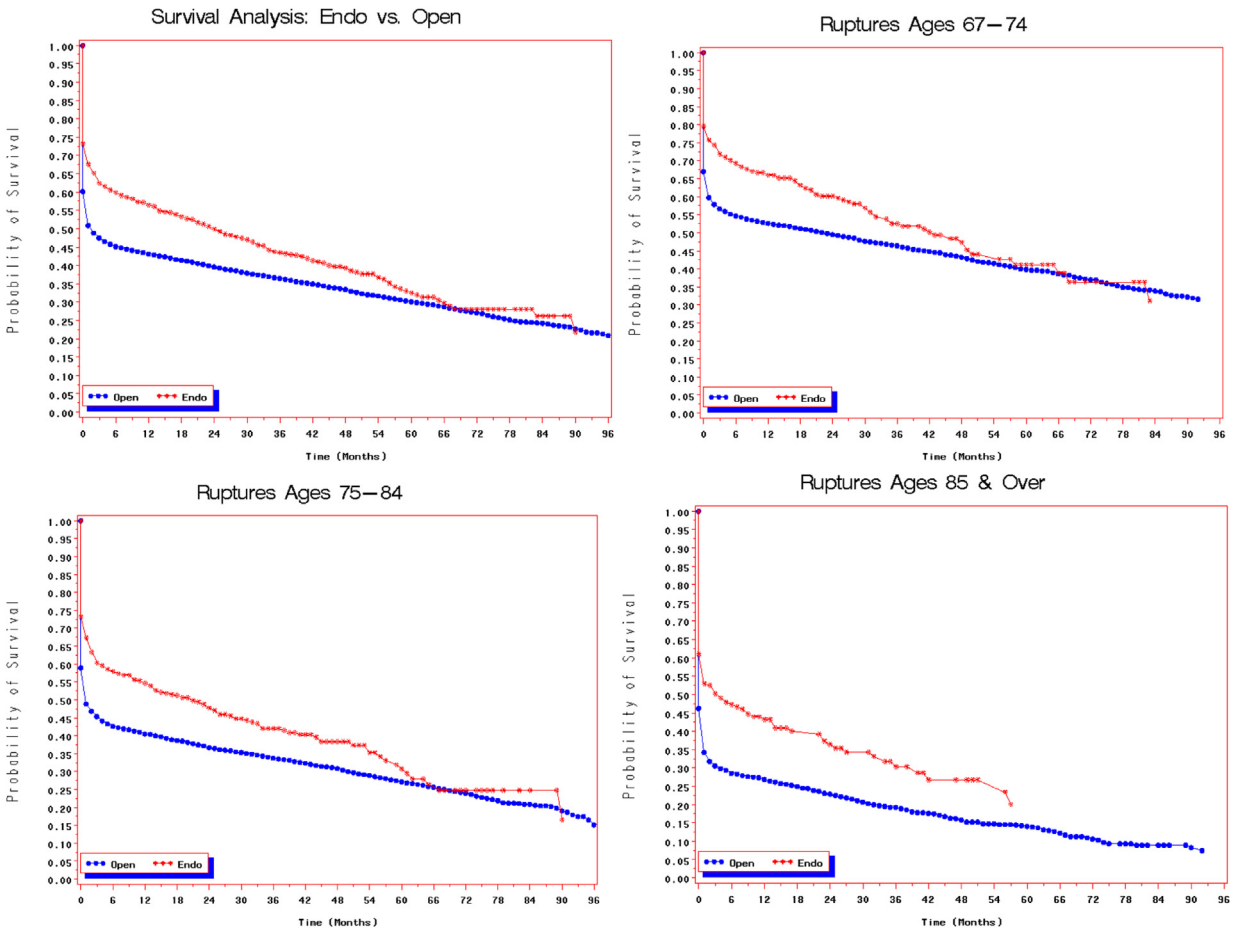
	Perioperative equation			*Endovascular equation*		
Term	*Estimate*	t *value*	P *value*	*Estimate*	t *value*	P *value*
Key predictors						
Endovascular repair	−0.630	−2.18	.0292			
Proportion endo				2.064	21.040	<.0001
BC(total volume)#				0.058	1.890	.059
λ(total volume)				0.352	2.560	.010
BC(endo volume, endo pats)#	−0.170	−1.210	.227			
BC(open volume, open pats)#	−0.042	−2.540	.011			
λ(endo volume, endo pats)	−0.294	−0.680	.493			
λ(open volume, open pats)	0.274	1.870	.061			
Nonclinical adjusters						
Procedure date				0.008	10.040	<.0001
Procedure date (endo pats)	0.002	1.210	.227			
Procedure date (open pats)	−0.001	−1.470	.142			
Urgent admission	0.088	2.910	.004	−0.331	−8.020	<.0001
Transfer	−0.062	−0.870	.384	0.259	2.860	.004
Patient characteristics						
Intercept	−0.066	−1.220	.224	−3.132	−24.860	<.0001
Age 70-74 (baseline 67-69)	0.158	4.340	<.0001	0.029	0.490	.625
Age 75-79 (baseline 67-69)	0.509	12.730	<.0001	0.185	2.900	.004
Age 80 & over (baseline 67-69)	0.766	10.300	<.0001	0.369	3.530	.000
Male	−0.124	−4.660	<.0001	0.100	2.350	.019
Black	−0.299	−4.990	<.0001	0.291	3.510	.000
ESRD	0.342	2.680	.007	0.254	1.540	.123
Chronic renal insufficiency	0.203	3.740	.000	−0.059	−0.760	.446
CABG	−0.378	−3.360	.001	−0.352	−1.500	.132
PCI	−0.254	−2.540	.011	0.127	0.980	.328
CAD without procedure	0.038	1.050	.296	0.153	3.000	.003
CHF	0.155	4.020	<.0001	0.173	3.090	.002
COPD	0.076	2.760	.006	0.044	1.030	.302
Vascular disease	0.102	3.250	.001	−0.028	−0.580	.564
Prior AAA diagnosis	−0.002	−0.070	.942	0.117	2.400	.016

AAA, Abdominal aortic aneurysm; *CABG*, coronary bypass surgery; *CAD*, coronary artery disease; *CHF*, chronic heart failure; *COPD*, chronic obstructive pulmonary disease; *ESRD*, end-stage renal disease; *PCI*, percutaneous coronary intervention.

The estimated selection effect (correlation between the unmeasured latent variables affecting perioperative mortality and likelihood of receiving EVAR) is 0.093 ($t = 1.15$; $P = .250$). #The Box-Cox transformation[7] with parameter λ of x is given by $x^{(\lambda)} = (x^{\lambda} - 1)/\lambda$ if $\lambda \neq 0$ or otherwise $x^{(\lambda)} = \log(x)$.



Supplementary Fig 1 (online only). Cohort formation and determination of treatment type. *EVAR*, Endovascular aortic repair; *HMO*, health maintenance organization.



Supplementary Fig 2 (online only). Mortality of unmatched patients undergoing endovascular (*Endo*) and open repair of ruptured aortic aneurysm (rAAA) is shown overall and stratified by age.

Supplementary Table I (online only). Physician and hospital coding of ruptured (*rAAA*) and intact abdominal aortic aneurysm (*AAA*) from 2005 to 2008

Hospital diagnosis	*Physician diagnosis*	*No.*	*30-day mortality, %*	*Open repairs, %*	*Open repairs coded as ruptures, %*
Rupture	Intact	190	20	62	19
Intact	Rupture	2519	7	47	15
Rupture	Rupture	5351	45	82	87

Patients with consistent coding for rAAA in hospital and physician codes have a substantially higher 30-day mortality of 45%, which is consistent with prior reports regarding mortality of rAAA. Also, a substantially higher proportion of repairs were performed as open repairs (82%), which is also consistent with prior reports regarding trends in rAAA repair. Because separate codes exist for open repair of rAAA vs open repair of intact AAA, we examined the percentage of open rAAA repairs that had the procedure code for rupture repair. Among patients with consistent rupture diagnosis, 87% of open repairs were coded as rupture repairs, whereas 15% to 19% of patients with inconsistent codes were coded as rupture repairs. Taken together, it appears that patients with inconsistent coding often have intact AAA, as EVAR is the dominant treatment for intact AAA, and the mortality is much lower than for rAAA, and their inclusion into the cohort would bias the results to suggest a survival benefit for EVAR.

Supplementary Table II (online only). Outcome definitions

Outcomes	*Complication*
Perioperative outcomes	
Medical complications	
410.00	Acute myocardial infarction[a]
481.00, 482.00	Pneumonia[a]
584.5, 584.9, 584.0	Acute renal failure[a]
90935, 90937, 90940, 36800, 36810, 36815, 36818, 36819, 36820, 36821, 36825, 36830, 36831, 36832, 36833, 36834	Hemodialysis[b]
415.1, 453.4	Venous thromboembolism[a]
Surgical complications	
34830, 34831, 34832	Conversion to open[b]
35860, 35840	Reoperation for bleeding[b]
31603, 31605, 31600	Tracheostomy[b]
34201, 34203	Embolectomy[b]
97602, 97597, 97598	Wound dehiscence[b]
99812	Hematoma[b]
578	Gastrointestinal bleeding[a]
55700, 55790	Mesenteric ischemia[b]
560	Bowel obstruction (intestinal obstruction without mention of hernia)[a]
44140, 44141, 44143, 44144, 44145, 44146, 44147, 44150, 44151, 44155, 44160, 44204, 44205, 44206, 44207, 44208, 44210, 44211, 44212, 44213, 44110	Colon resection[b]
Late outcomes	
Open surgical reintervention[b]	
34830, 34831, 34832	Conversion to open repair
35082, 35082,35092,35103	Open AAA repair
35907, 35870	Graft infection
35654, 35621	Aortobifemoral, axillofemoral or axillobifemoral bypass
35875, 35876	Thrombectomy
35131, 35132	Iliac aneurysm repair
35661	Femoral-femoral bypass
Endovascular reintervention[b]	
34800, 34802, 34803, 34804, 34805, 0078T, 0080T, 0001T, 0002T	Endovascular AAA repair (redo)
35472, 35473,	Angioplasty
34825, 34826	Cuff extension
37204	Embolization
34900, 75954	Endovascular iliac aneurysm repair
Laparotomy-related complications	
Bowel obstruction (nonsurgical admission)[a]	
560.1	Paralytic ileus
560.8	Intestinal obstruction without mention of hernia
560.81	Intestinal or peritoneal adhesions with obstruction
560.89	Pseudo-obstruction or mural thickening causing obstruction
560.9	Unspecified intestinal obstruction
552.21	Incisional hernia with obstruction
Bowel obstruction (surgical)[b]	
44005	Enterolysis (freeing of intestinal adhesion)
44180	Laparoscopy, surgical, enterolysis (freeing of intestinal adhesion)
44202	Laparoscopy, surgical; enterectomy; resection of small intestine
44203	…each additional small intestine resection
44120	Enterectomy, resection of small intestine; single resection
44130	Enteroenterostomy, anastomosis of intestine
44186	Laparoscopy, surgical; jejunostomy (eg, for decompression or feeding)
44187	…ileostomy or jejunostomy, nontube
44140	Colectomy, partial; with anastomosis
44141	…with skin level cecostomy or colostomy
44143	…with end colostomy and closure of distal segment
44144	…with resection, with colostomy or ileostomy and creation of mucofistula
44160	Colectomy, partial, with removal of terminal ileum with ileocolostomy
44204	Laparoscopy, surgical; colectomy, partial, with anastomosis
44205	…with removal of terminal ileum with ileocolostomy
44206	…with end colostomy and closure of distal segment
44213	Laparoscopy, surgical; mobilization of splenic flexure with partial colectomy

(*Continued on next page*)

Supplementary Table II (online only). Continued.

Outcomes	*Complication*
44188	Laparoscopy, surgical, colostomy or skin level cecostomy
Incisional hernia[b]	
49560	Repair initial incisional or ventral hernia; reducible
49561	…incarcerated or strangulated
49565	Repair recurrent incisional or ventral hernia; reducible
49566	…incarcerated or strangulated
49568	Implantation of mesh for incisional or ventral hernia
49654	Laparoscopy, surgical, repair, incisional hernia; reducible
49655	…incarcerated or strangulated
49656	Laparoscopy, surgical, repair, recurrent incisional hernia; reducible
49657	…incarcerated or strangulated

AAA, Abdominal aortic aneurysm.
[a]International Classification of Diseases-9th edition (ICD-9) codes.
[b]Current Procedural Terminology (CPT) codes.

Supplementary Table III (online only). Perioperative outcomes in unmatched sample, by treatment

Outcome	*EVAR (n = 1126)*	*Open (n = 9872)*	P
Perioperative mortality, %			
All ages	33.8	50.2	<.0001
67-69 years	18.8	39.6	<.0001
70-74 years	27.6	42.2	<.0001
75-79 years	30.2	49.2	<.0001
80-84 years	38.8	58.0	<.0001
≥85 years	50.5	67.0	<.0001
Medical complications, %			
Myocardial infarction	16.9	15.7	.3199
Pneumonia	28.7	31.7	.0405
Acute renal failure	33.7	39.4	.0002
Hemodialysis	0.7	0.6	.7956
Respiratory failure/ tracheostomy	4.5	8.5	<.0001
Venous thromboembolism	8.1	4.4	<.0001
Surgical complications, %			
Conversion to open repair	4.8		
Reoperation for bleeding	2.3	2.5	.7082
Embolectomy	3.6	6.6	<.0001
Wound dehiscence	2.5	3.8	.0285
Operative site hematoma	8.2	3.9	<.0001
Gastrointestinal bleeding	10.4	11.2	.4169
Mesenteric ischemia	6.4	7.5	.192
Bowel obstruction or ileus	13.1	15.7	.0182
Colon resection	4.4	6.7	<.0001
Ostomy	2.8	4.2	.018
Discharge to home, %	62.3	45.3	<.0001
Length of stay, median (IQR) days	7 (4-13)	13 (9-21)	<.0001

EVAR, Endovascular aortic repair; *IQR,* interquartile range.