Published in final edited form as:

Surgery. 2017 October ; 162(4): 721–731. doi:10.1016/j.surg.2017.01.022.

Open versus Endovascular Abdominal Aortic Aneurysm Repair in Medicare Beneficiaries

Sarah E. Deery, MD and Marc L. Schermerhorn, MD

Beth Israel Deaconess Medical Center

Abstract

Abdominal aortic aneurysms (AAA) are relatively common and often life-threatening, with especially high mortality following aneurysm rupture. The introduction of endovascular aneurysm repair (EVAR), a minimally invasive alternative to traditional open repair, lead to decreased mortality and morbidity in randomized controlled trials, but these trials were conducted in highly selected patients and providers and were underpowered to detect differences in rare adverse events throughout follow-up. With observational studies of Medicare beneficiaries, we demonstrated that the randomized trial results were generalizable to the majority of patients undergoing AAA repair in the United States. Additionally, with the larger cohort, comparative analyses evaluating rare, previously unstudied late outcomes such as laparotomy-related complications, late reinterventions, mortality with reinterventions, and late rupture could be conducted. Furthermore, trends in management over time and relationships between surgeon- and hospital-volume and outcomes can be studied. The goal of this review is to summarize the existing literature regarding abdominal aortic aneurysms amongst Medicare beneficiaries and to evaluate the benefits and limitations of administrative claims data in comparative effectiveness research.

Introduction

Abdominal aortic aneurysms (AAA) are relatively common and often life-threatening, with over 15,000 domestic AAA ruptures annually and a mortality of >80% with aneurysm rupture.^{1–3} Abdominal aortic aneurysms were first described in the 16th century by the French anatomist Vesalius;⁴ however, over three centuries passed before the first successful repair was performed. While many surgeons attempted to treat AAA with aortic ligation, Rudolph Matas in 1923 was the first to do this successfully, and the patient subsequently lived 18 months.⁵ However, by 1940, only 23 ligations had been recorded and only five of these were successful.⁶ Later, cellophane was used to wrap the aorta in an attempt to restrict growth that, infamously, failed to prevent the death of Albert Einstein. Major advances were then rapidly made when Dubost et al. performed the first successful homograft replacement

Corresponding Author/Reprints: Marc L. Schermerhorn, MD, FACS, Beth Israel Deaconess Medical Center, 110 Francis Street, Suite 5B, Boston, MA 02215, *Telephone:* 617-632-9971, *Fax:* 617-632-7977, *mscherm@bidmc.harvard.edu*.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

Dr. Schermerhorn serves as a consultant for Endologix.

in 1951,⁷ Voorhees et al. implanted the first successful prosthetic aortic graft into a dog in 1952,⁸ and DeBakey and Cooley completed the first thoracic aortic replacement with Dacron in 1953.⁹ Finally, in 1966, Creech combined the prosthetic graft technique with the endoaneurysmorrhaphy described by Matas in 1903,¹⁰ leading to the modern open AAA repair.¹¹

With the success of this procedure, surgeons began to electively repair aortic aneurysms to prevent rupture and its accompanying mortality and morbidity. After early advances, however, little progress was made as rates of elective AAA repair, of ruptured AAA, and mortality with elective and ruptured AAA remained stable from the late 1970s to 1990s.¹² However, the introduction of endovascular aneurysm repair (EVAR), a minimally invasive alternative to traditional open repair, in 1991 by Dr. Juan Parodi, revolutionized aneurysm management. The Food and Drug Administration approved EVAR in 1999, and now 80% of infrarenal aneurysm repairs are performed endovascularly, allowing patients previously considered too high risk for open surgery to undergo repair. In the 2000s, three major randomized, controlled trials (RCTs), EVAR-1,¹³ DREAM,¹⁴ and OVER,¹⁵ showed that EVAR has lower perioperative mortality and morbidity compared to open repair. A meta-analysis of randomized trials showed a significant mortality benefit with 1.4% mortality following EVAR and 4.2% following open repair in 2,790 total patients. However, these randomized trials were limited by including only highly selected subjects amenable medically and anatomically to both open and endovascular repair, and were conducted only in highly selected institutions with experienced physicians. Therefore, the generalizability of these results to the average American population may be limited. Furthermore, with fewer than 3,000 patients combined, the studies were underpowered to detect differences in rare and remote outcomes, such as late reintervention, laparotomy-related complications, and rupture.

The Medicare Program from the Center for Medicare and Medicaid Services (CMS) is uniquely positioned to address the limitations of these randomized trials. Medicare is the United States' health insurance program for adults aged 65 and older, some persons under age 65 with disabilities, and persons with specified chronic medical conditions such as end-stage renal disease, with approximately 97% of persons aged 65 and older covered by Medicare. All claims files are generated annually and can be used to construct patient-level longitudinal histories of health care utilization for Medicare beneficiaries enrolled in traditional fee-for-service (FFS) Medicare. While CMS collects and maintains health care claims for all services billed to and paid for under Medicare's fee-for-service (FFS) payment system, they do not have claims data available for research for those enrolled in Medicare Advantage (MA), Medicare's managed care program, which made up 5–10% of Medicare beneficiaries between 2001 and 2008.¹⁶ As 70% of the AAA repairs performed in the United States are covered by FFS Medicare, these data are widely generalizable across the country. Additionally, with over 15,000 beneficiaries undergoing elective AAA repair per year, we are much better powered using observational Medicare data than the randomized trials to detect differences in late outcomes. Furthermore, with Medicare, we can better address questions about the durability of EVAR, differences by graft type, and account for prior laparotomy in propensity score models. The goal of this review is to summarize our existing

studies of abdominal aortic aneurysms amongst Medicare beneficiaries and to evaluate the benefits and limitations of administrative claims data in comparative effectiveness research.

Medicare Claims Data

We utilized data from several CMS sources: 1) The Beneficiary Summary File (formerly Denominator File) contains demographic, vital status, and enrollment data for each beneficiary; 2) The Medicare Provider Analysis and Review (MEDPAR) File contains inpatient discharges from acute care hospitals and skilled nursing facilities; 3) The Physician/Supplier (Carrier) File contains one claim for each physician service covered by Medicare Part B; 4) The Outpatient File contains claims for Part B services provided by outpatient institutional providers, which often includes follow-up imaging data. Additionally, the Inpatient Standard Analytic File contains more detailed claims, to more accurately capture hospital costs (in contrast to payments) associated with the initial procedure. To improve the coding accuracy, we also examined physician claims occurring during the hospitalizations and, in cases of conflict, assigned patients to the appropriate treatment group based on the physician's diagnosis and/or procedure codes. Thus, our approach allowed us to validate assignment based on hospital discharge codes as well as to reassign the small percentage that might have been misclassified if relying only on hospital claims.

A comprehensive list of all AAA repairs performed under Medicare can be identified by querying the appropriate *International Classification of Diseases, Ninth Revision, Clinical Modification* (ICD-9-CM) codes. We searched hospital claims for the ICD-9-CM discharge diagnosis code of AAA with (441.3) or without rupture (441.4) that also had a procedure code indicating open (38.44 or 39.25) or endovascular (39.71) repair during that admission. To limit the analysis to infrarenal AAA, we excluded patients with diagnosis codes indicating thoracic aneurysm (441.1 or 441.2), thoracoabdominal aortic aneurysm (441.6 or 441.7), or aortic dissection (441.0–441.03), or with procedure codes for repair of the thoracic aorta (38.35, 38.45, 39.73), fenestration of dissection (39.54), or visceral/renal bypass (38.46, 39.24, or 39.26). We then further analyzed physician billing data to categorize repairs as open or endovascular using the CPT codes (open: 35081 and 35082, EVAR: 34800–34805), since physicians should more accurately categorize the procedure they performed. For the years 2001–2005, the CPT codes correlated with ICD-9 codes in >95%.

For studies involving comparative analyses or risk prediction modeling, we limited our analysis to patients aged 67 or older at the time of repair, as this allowed us to obtain two years of data prior to the procedure for purposes of controlling for baseline health status and clinical conditions. We utilized inpatient and outpatient ICD-9 diagnosis codes throughout those two years, not including the index admission, to determine preoperative comorbidities for each patient. Then, using a method adapted from the Elixhauser algorithm to include outpatient diagnoses,^{17, 18} we matched each beneficiary who underwent endovascular repair to the beneficiary who underwent open repair with the closest estimated propensity score. In the unmatched cohort, EVAR patients were older and sicker, with higher rates of essentially all comorbid conditions; however, after propensity-score matching, there were no statistical

differences between EVAR and open repair patients.^{19, 20} By propensity score matching on demographics and comorbid baseline conditions, we could control for the nonrandom assignment of patients to one of the two procedures.

Perioperative Mortality

In our initial comparative effectiveness studies amongst Medicare beneficiaries, we utilized the above methodology to identify all patients aged 67 or older with at least two years of claims data that underwent open or endovascular repair of intact AAA between 2001 and 2004. We identified 45,660 propensity-score matched beneficiaries, with half of these undergoing open and the other half endovascular repair.²⁰ We found that combined 30-day and in-hospital mortality was 5.3% after open repair but only 1.7% after endovascular repair (relative risk for the open repair group 3.2, 95% confidence interval 2.8–3.5, $P < .001$).^{20, 21} These results are comparable to those found in the meta-analysis of RCTs, despite including a more generalized cohort.²² The most significant mortality discrepancy was in patients older than 85, with an absolute 30-day and in-hospital mortality difference of 8.5%, suggesting that these older patients most benefited by the introduction of EVAR.²⁰ We performed a subsequent analysis with Medicare beneficiaries from 2001–2008, with 79,932 propensity-matched patients. The perioperative mortality remained significantly higher following open repair (5.2% vs. 1.6%, $P < .001$). Notably, however, the rates of both decreased steadily over the eight-year period, with an 11% reduction in mortality following open repair (5.7% to 5.1%, $P = .001$), but a 36% mortality reduction following EVAR (2.2% to 1.4%, $P = .01$).¹⁹ Notably, the rate of death following open repair did not stabilize until three months postoperatively, at which point interval death rates approached that of EVAR (Figure 1).²¹ Additionally, the rate of early conversion to open repair dropped dramatically over the same time period, from 2.2% to 0.3% ($P < .001$).

Mortality Risk Prediction Model

Using Medicare beneficiaries from 2001–2004, we also developed a risk prediction model for perioperative mortality following AAA repair.²³ We again used propensity-score matched cohorts to conduct this analysis, with half the sample used to develop the prediction model, and the other half used to validate the model. We found the strongest predictors of in-hospital or 30-day mortality were open repair (odds ratio 3.2, 95% CI 2.7–3.8), age > 80 years (3.1, 2.4–4.2), and end-stage renal disease (2.6, 1.5–4.6). Other predictors included age 76–80 (1.9, 1.4–2.5), female sex (1.5, 1.3–1.8), chronic renal insufficiency (2.0, 1.6–2.6), congestive heart failure (1.7, 1.5–2.1), and peripheral/cerebral vascular disease (1.3, 1.2–1.6). There were no differential interactions between these variables and repair type. From these results, we developed a scoring system that places patients into high, medium, or low risk categories, with differential mortality rates following open vs. endovascular repair (Table I).²³ This scoring system was validated within the second half of our cohort, with the area under the receiver operative curve (ROC) of 71.8 in the validation cohort. Most notably, with increased preoperative comorbidity burden, the absolute mortality benefit from EVAR compared to open repair increases, with significantly more favorable outcomes following EVAR.

Perioperative Morbidity

We also found that perioperative morbidity is significantly lower after EVAR compared to open repair. In our above series of patients from 2001–2004, we showed lower rates of myocardial infarction (7.0% vs. 9.4%, $P < .001$), pneumonia (9.3% vs. 17%, $P < .001$), acute renal failure (5.5% vs. 11%, $P < .001$), and dialysis (0.4% vs. 0.6%, $P < .001$) in EVAR patients compared to open repair.²⁰ Other surgical complications were also slightly more frequent in patients undergoing open repair, although the rates were low in both groups, including: acute mesenteric ischemia (2.1% vs. 1.0%, $P < .001$), reintervention for bleeding (1.2% vs. 0.8%, $P < .001$), embolectomy (1.7% vs. 1.3%, $P < .001$), and tracheostomy (1.6% vs. 0.2%, $P < .001$).²⁰ Abdominal complications were higher in the open repair group: bowel resection (1.3% vs. 0.6%, $P < .001$) and obstruction or ileus without intervention (17% vs. 5.1%, $P < .001$). The hospital length of stay (LOS) was also shorter in the EVAR group, with a mean LOS 3.4 days vs. 9.3 days ($P < .001$) and median LOS 2 days vs. 7 days ($P < .001$), and fewer patients were discharged to a skilled nursing facility (5.6% vs. 18%, $P < .001$).^{19, 20}

Late Survival

The mid- and long-term outcomes from the three major RCTs show loss of the initial survival benefit of EVAR 1–2 (DREAM and EVAR-1) or 5 years (OVER) following repair.^{24–26} In our substantially larger cohort that was not restricted to patients who would qualify medically for a clinical trial, we still identified an early survival advantage amongst all age groups, which lasted for approximately 3 years. We showed rates of overall survival similar to the rates found in the RCTs, with 8-year survival of 45.1% following EVAR and 45.3% following open repair ($P = .76$). Notably, the risk (hazard) of death was not proportional over time (Table II).¹⁹ Although EVAR was associated with a substantial early survival benefit, rates of death were higher following EVAR between 90 days and 4 years postoperatively among those who had survived to this period, after which the rates remained higher but only slightly (HR 1.05, 95% Confidence Interval 1.00 – 1.09). The reason for the increased hazard of death following EVAR after 90 days is likely multifold, and chance alone is unlikely given that these findings have been repeated in many series.^{15, 19, 24, 26} We believe that the main reason is that the older, sicker patients can survive EVAR, but then die sooner than the younger, fitter patients who undergo open repair. However, late rupture and reintervention likely also play a role.

Late Outcomes

Reintervention

One of the goals of using Medicare data was to establish a large enough cohort of AAA patients to be powered to detect differences in rare although clinically significant late outcomes. One of the early criticisms of EVAR was the concern for graft durability, with high rates of AAA-related complications. The EVAR-1 study indeed reported significantly lower rates of reintervention-free survival following EVAR at 8 years (72% vs. 90%, $P < .001$).²⁶ However, this only accounts for AAA-related complications, and the majority of vascular reinterventions following EVAR are relatively minor endovascular procedures. Not

all late complications are graft-related. In particular, patients undergoing EVAR will not have a transabdominal or retroperitoneal incision; therefore, laparotomy-related complications should be accounted for, including abdominal wall hernias (with repair) and bowel obstructions (those managed with nonoperative admission as well as those that underwent lysis of adhesion or bowel resection).

In our 8-year follow-up of Medicare beneficiaries, we confirmed that aneurysm-related interventions were much more common following EVAR (19% vs. 3.7%, $P < .001$), including major reinterventions (2.3% vs. 0.8%, $P < .001$) such as conversion to open and graft infection; and minor reinterventions (18% vs. 3.1%, $P < .001$) such as coil embolization or extension (Table III). However, laparotomy-related reinterventions, especially incisional hernia repairs, were more common amongst patients undergoing open repair (18% vs. 8.2%, $P < .001$), as were nonoperative admissions for bowel obstructions (22% vs. 17%, $P < .001$). After combining aneurysm-related interventions with laparotomy-related interventions, patients undergoing endovascular repair did still have higher rates of reinterventions (25% vs. 21% at 8 years, $P < .001$).¹⁹ Additionally, a large percentage of the reinterventions following EVAR were only “minor” vascular reinterventions. Conversely, hospitalizations related to the aneurysm or laparotomy-related complications without intervention were lower following EVAR (18% vs. 22%, $P < .001$). Interestingly, after stratifying our cohort by age, we found that only patients age 75 or older were more likely to have reinterventions following EVAR, with patients < 75 more likely to have reinterventions following open repair (Table III). This likely reflects improved patient selection in younger, healthier patients with little perioperative survival difference for whom a more durable repair is desired, and an acceptance of higher late re-intervention and rupture risk in older, more frail patients in whom the up-front mortality difference is substantial.

Rupture

Another concern with the durability of EVAR is that it will lead to higher rates of late rupture than traditional open repair. However, as the rates of late rupture are quite low, this hypothesis had not previously been demonstrated. In our study of Medicare beneficiaries from 2001–2008, we identified significantly higher rates of rupture following EVAR, with rates of 5.4% following EVAR and 1.4% following open repair through eight years of follow-up ($P < .001$).¹⁹ Overall, we found that patients undergoing EVAR had lower freedom from rupture, aneurysm-related reintervention, and laparotomy-related reintervention compared to open repair patients (Figure 2). These curves increasingly separate over time, suggesting that, in patients with long life expectancy, we need to identify those patients at risk for late reintervention and rupture, and consider this in the surgical approach.

Mortality with Reintervention and Rupture

The occurrence of rupture, reintervention, and readmission in follow-up are not insignificant complications. In a separate analysis of Medicare beneficiaries from 2001–2004, we showed that overall rates of readmission or reintervention were slightly higher following EVAR (7.6 vs. 7.0/100 person-years, relative risk 1.1, $P < .001$), with a 30-day mortality of 9.1% associated with reinterventions, notably higher than the mortality associated with the index

AAA repairs.²⁷ Patients undergoing EVAR were more likely to undergo AAA-related interventions (major intervention and minor open and endovascular intervention), but open repair patients were more likely to undergo laparotomy-related intervention (major and minor). These secondary interventions and hospitalizations accounted for 9.6% of all EVAR deaths and 7.6% of all open repair deaths in the follow-up period. It is again important to note that the greatest number of reinterventions after EVAR fell into the minor endovascular AAA-related category, which carried a lower overall 30-day mortality of 4% compared to 13.7% following major AAA-related reinterventions. Major laparotomy-related reinterventions also were associated with a relatively high 30-day mortality of 12.2%, and were more than twice as common following open repair. Late AAA rupture, although rare, was again 6 times more likely following EVAR, and was associated with a 28% mortality.²⁷ After exclusion of deaths within 90 days of the original AAA intervention, patients with a reintervention or readmission after EVAR had a decreased survival compared with those without. Similarly, after open AAA repair, patients who needed a reintervention or readmission had a lower long-term survival. Furthermore, survival among patients who underwent open repair with reintervention was lower than with those who underwent EVAR without reintervention (Figure 3). Amongst patients who never experience rupture, reintervention, or readmission for laparotomy-related complications, the survival curves meet one year later at 4 years, after which they are overlapping (Figure 4). Given these findings, differences in rates of rupture and reintervention alone do not explain the higher hazard of late mortality in patients undergoing EVAR. The patients who survive EVAR are older and sicker and therefore more likely to die early than the healthier patients who survive open repair.

Trends in AAA Presentation, Repair, and Mortality

Medicare claims data are uniquely situated to be able to show near-complete population-level data for patients over the age of 65 in the United States. While unable to capture patients who die from AAA rupture prior to hospital presentation, Medicare claims data allow for trend analysis of AAA-related mortality, rates of rupture, and type of repair. We analyzed all Medicare beneficiaries who presented with AAA during the period from 1995 to 2008, and found that while the use of EVAR increased from 1.5% to 77%, more intact AAA repairs were performed (79.9 to 85.0 repairs per 100,000 Medicare beneficiaries) and fewer patients presented with rupture (33.4 to 16.8 ruptures per 100,000 beneficiaries).²⁸ Those patients 80 years and older seemed to derive the most benefit from the introduction of EVAR, with rates of intact AAA repair increasing from 57.7 to 92.3 per 100,000 Medicare beneficiaries, with 83% of these repairs done via EVAR in 2008. Over this time period, overall short-term aneurysm-related mortality, which included perioperative mortality after intact and ruptured repair as well as in-hospital mortality from ruptured AAA without repair, decreased from 26.1 to 12.1 per 100,000 Medicare beneficiaries (Figure 5). Therefore, we have shown decreased aneurysm-related mortality with time as EVAR has become more frequently utilized, with the largest benefit in those patients > 80 years old, who were likely to have been considered too high risk for open surgical repair. Outcomes following EVAR are also improving, as 2-year rates of aneurysm-related reintervention following EVAR has

decreased from 10.4% to 9.1% ($P < .001$), due primarily to lower rates of coil embolization for type II endoleak (4.2% to 2.5%, $P < .001$).

Overall mortality following ruptured AAA has decreased for the first time in decades to 36.3%, due primarily to the markedly lower mortality of 27.5% following EVAR for ruptured AAA (Figure 6). If the observed lower mortality with EVAR for ruptured AAA was simply a matter of selection of the more hemodynamically stable patients who would do well with either approach, then one would expect a concomitant increase in open repair mortality over time with a stable overall mortality rate. Instead, the mortality rate with open repair remains stable; thus, these data strongly support the concept that rAAA mortality is in fact lower since the introduction of EVAR.

As EVAR is more frequently utilized, and perioperative outcomes of intact AAA repair improve, increased screening may identify more aneurysms and allow for timely repair. However, currently, U.S. Preventative Services Task Force (USPSTF) guidelines only recommend a one-time screening ultrasound in men aged 65–75 with a history of smoking, and these patients, as well as those with a family history of AAA, are the only ones reimbursed by CMS for a screening ultrasound. Unfortunately, we previously identified that compliance with screening ultrasound attendance is low. We previously contacted 30,000 randomly selected Medicare beneficiaries, not restricted by age or sex, from the referral region of three university-affiliated hospitals, and found that only 7% of patients contacted actually presented for free screening ultrasound. Amongst the screened patients, 2.8% of men and 0.2% of women were found to have previously undetected aneurysms, although these estimates are likely lower than the general population as they reflect the more-well patients who were willing to attend screening. Now that this is a service provided by Medicare for a subgroup of beneficiaries, further studies are necessary to determine the role of expanded screening to additional groups, and to consider targets for quality improvement to improve compliance with screening attendance.

Volume-Outcomes Relationship

An additional benefit of using Medicare claims data is the ability to perform volume-outcomes analyses. Multiple series using the Nationwide Inpatient Sample and Medicare have identified relationships between surgeon volume and outcomes following AAA repair.^{29–34} Using Medicare data, we initially evaluated the influence of institutional volume on outcomes using cases from 2001–2004.³² During that time period, there was a marked drop in mortality following EVAR between the first and second lowest quintiles of institutional volume, with steady decreases in each subsequent quintile, suggesting that only a very low institutional threshold is necessary with little improvement beyond that low level. With open repair, however, there were steady decreases in mortality across each increased quintile of volume. However, these data reflect the relatively early experience with EVAR, which has now become much more widely utilized. Furthermore, this study did not account for surgeon volume. Therefore, we conducted an additional analysis of Medicare data from 2001–2008, and found that while perioperative mortality following EVAR was not associated with surgeon volume, it did decrease slightly with greater hospital volume (1.9% in quintile 1 vs. 1.4% in quintile 5).³⁴ Following open repair, perioperative mortality

decreased independently with both higher surgeon volume (6.4% in quintile 1 vs. 3.8% in quintile 5) and hospital volume (6.3% in quintile 1 vs. 3.8% in quintile 5). While the specific volumes necessary to achieve acceptable perioperative outcomes are not agreed on, these data suggest that EVAR and open repair should be performed in high-volume centers, and that open repair should be performed by high-volume surgeons.

Discussion

Using Medicare claims data, we have previously shown that endovascular AAA repair is associated with lower perioperative mortality and morbidity, with increased rates of long-term reinterventions and rupture, and comparable long-term mortality.^{19–21, 23, 27, 28, 35} Perhaps the most important benefit of using Medicare claims data is that we are able to capture a nearly comprehensive sample of patients undergoing AAA intervention in this country, as greater than 70% of AAA repairs are conducted under the Medicare program. Our analyses demonstrated the results of the landmark randomized, controlled trials conducted in centers of excellence are generalizable to the broad U.S. population.^{15, 36, 37} Notably, this is not always the case, as Wennberg et al. identified higher rates of perioperative mortality following carotid endarterectomy in a cohort of all Medicare beneficiaries than was seen in either the NASCET or ACAS trials.^{38–40} Additionally, we have been able to evaluate much larger cohorts of patients, with adequate power to detect differences in rare late complications, such as rupture and reintervention by type of procedure. Medicare also provides the unique opportunity to perform analyses of outcomes by hospital and surgeon volume. An additional benefit of Medicare data for future analyses is the ability to link hospital claims and diagnosis codes with costs, including all costs throughout follow-up.

Despite these advantages, Medicare claims data are limited to diagnosis and billing codes. While some clinical granularity can be deduced by the types of procedure codes used, most vascular-specific data, including aneurysm size and other anatomic characteristics, are not available. Furthermore, only patients over the age of 65 are included, so any analyses on optimal management in patients < 65 cannot be performed, although this is a small minority of AAA patients. However, the large numbers of patients included in Medicare studies, and the capability of analyzing comprehensive long-term follow-up data without loss to follow-up to other institutions, still makes Medicare uniquely positioned for comparative effectiveness studies. In the future, some of the limitations related to lack of anatomic and clinical granularity will be addressed by linkage of patients in national vascular registries to their Medicare claims data. This will allow for further patient-centered evaluation of the decision to treat with an endovascular or open approach.

Conclusion

Amongst Medicare beneficiaries, endovascular aneurysm repair is associated with decreased perioperative mortality and morbidity compared to open repair. Late complications such as rupture and reintervention are more frequent in patients undergoing EVAR, and are associated with high rates of peri-procedure mortality. However, the majority of reinterventions following EVAR are minor AAA-related procedures; whereas patients

undergoing open repair have higher rates of laparotomy-related complications. In the future, studies of Medicare beneficiaries would be enhanced by linkage to registries with more detailed clinical and anatomic data, to predict who is at risk for late rupture and reintervention in order to best understand which patients would most benefit from endovascular versus open repair.

Acknowledgments

This work is supported by the Harvard-Longwood Research Training in Vascular Surgery NIH T32 Grant 5T32HL007734-23.

References

1. Sachs T, Schermerhorn M. Ruptured abdominal aortic aneurysm. *Minerva Chir.* 2010; 65:303–17. [PubMed: 20668419]
2. Schermerhorn, ML., Cronenwett, JL. *Vascular Surgery*. Philadelphia, PA: WB Saunders Company; 2005. *Abdominal Aortic Aneurysms*.
3. Bengtsson H, Bergqvist D. Ruptured abdominal aortic aneurysm: a population-based study. *J Vasc Surg.* 1993; 18:74–80. [PubMed: 8326662]
4. Cooley, DA. *Surgical treatment of aortic aneurysms*. Philadelphia: WB Saunders; 1986.
5. Matas R. Ligation of the Abdominal Aorta: Report of the Ultimate Result, One Year, Five Months and Nine Days after Ligation of the Abdominal Aorta for Aneurism at the Bifurcation. *Ann Surg.* 1925; 81:457–64. [PubMed: 17865196]
6. Elkin DC. Aneurysm of the Abdominal Aorta: Treatment by Ligation. *Ann Surg.* 1940; 112:895–908. [PubMed: 17857685]
7. Dubost C, Allary M, Oeconomos N. Resection of an aneurysm of the abdominal aorta: reestablishment of the continuity by a preserved human arterial graft, with result after five months. *AMA Arch Surg.* 1952; 64:405–8. [PubMed: 14894065]
8. Voorhees AB Jr, Jaretzki A 3rd, Blakemore AH. The use of tubes constructed from vinyon “N” cloth in bridging arterial defects. *Ann Surg.* 1952; 135:332–6. [PubMed: 14903863]
9. De Bakey ME, Cooley DA. Successful resection of aneurysm of thoracic aorta and replacement by graft. *J Am Med Assoc.* 1953; 152:673–6. [PubMed: 13044606]
10. Matas RI. An Operation for the Radical Cure of Aneurism based upon Arteriorrhaphy. *Ann Surg.* 1903; 37:161–96. [PubMed: 17861243]
11. Creech O Jr. Endo-aneurysmorrhaphy and treatment of aortic aneurysm. *Ann Surg.* 1966; 164:935–46. [PubMed: 5332747]
12. Heller JA, Weinberg A, Arons R, Krishnasastri 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. [PubMed: 11107080]
13. Greenhalgh RM, Brown LC, Kwong GP, Powell JT, Thompson SG, participants Et. Comparison of endovascular aneurysm repair with open repair in patients with abdominal aortic aneurysm (EVAR trial 1), 30-day operative mortality results: randomised controlled trial. *Lancet.* 2004; 364:843–8. [PubMed: 15351191]
14. Prinssen M, Verhoeven EL, Buth J, Cuypers PW, van Sambeek MR, Balm R, et al. A randomized trial comparing conventional and endovascular repair of abdominal aortic aneurysms. *N Engl J Med.* 2004; 351:1607–18. [PubMed: 15483279]
15. Lederle FA, Freischlag JA, Kyriakides TC, Padberg FT Jr, Matsumura JS, Kohler TR, et al. Outcomes following endovascular vs open repair of abdominal aortic aneurysm: a randomized trial. *JAMA.* 2009; 302:1535–42. [PubMed: 19826022]
16. Medicare Payment Advisory Commission (U.S.). *Healthcare spending and the Medicare program: a data book*. Washington, DC: Medicare Payment Advisory Commission. p. v.;

17. Baldwin LM, Klabunde CN, Green P, Barlow W, Wright G. In search of the perfect comorbidity measure for use with administrative claims data: does it exist? *Med Care*. 2006; 44:745–53. [PubMed: 16862036]
18. Elixhauser A, Steiner C, Harris DR, Coffey RM. Comorbidity measures for use with administrative data. *Med Care*. 1998; 36:8–27. [PubMed: 9431328]
19. Schermerhorn ML, Buck DB, O'Malley AJ, Curran T, McCallum JC, Darling J, et al. Long-Term Outcomes of Abdominal Aortic Aneurysm in the Medicare Population. *N Engl J Med*. 2015; 373:328–38. [PubMed: 26200979]
20. Schermerhorn ML, O'Malley AJ, Jhaveri A, Cotterill P, Pomposelli F, Landon BE. Endovascular vs. open repair of abdominal aortic aneurysms in the Medicare population. *N Engl J Med*. 2008; 358:464–74. [PubMed: 18234751]
21. Schermerhorn ML, Giles KA, Sachs T, Bensley RP, O'Malley AJ, Cotterill P, et al. Defining perioperative mortality after open and endovascular aortic aneurysm repair in the US Medicare population. *J Am Coll Surg*. 2011; 212:349–55. [PubMed: 21296011]
22. Dangas G, O'Connor D, Firwana B, Brar S, Ellozy S, Vouyouka A, et al. Open versus endovascular stent graft repair of abdominal aortic aneurysms: a meta-analysis of randomized trials. *JACC Cardiovasc Interv*. 2012; 5:1071–80. [PubMed: 23078738]
23. Giles KA, Schermerhorn ML, O'Malley AJ, Cotterill P, Jhaveri A, Pomposelli FB, et al. Risk prediction for perioperative mortality of endovascular vs open repair of abdominal aortic aneurysms using the Medicare population. *J Vasc Surg*. 2009; 50:256–62. [PubMed: 19249184]
24. De Bruin JL, Baas AF, Buth J, Prinssen M, Verhoeven EL, Cuypers PW, et al. Long-term outcome of open or endovascular repair of abdominal aortic aneurysm. *N Engl J Med*. 2010; 362:1881–9. [PubMed: 20484396]
25. Lederle FA, Freischlag JA, Kyriakides TC, Matsumura JS, Padberg FT Jr, Kohler TR, et al. Long-term comparison of endovascular and open repair of abdominal aortic aneurysm. *N Engl J Med*. 2012; 367:1988–97. [PubMed: 23171095]
26. United Kingdom ETI, Greenhalgh RM, Brown LC, Powell JT, Thompson SG, Epstein D, et al. Endovascular versus open repair of abdominal aortic aneurysm. *N Engl J Med*. 2010; 362:1863–71. [PubMed: 20382983]
27. Giles KA, Landon BE, Cotterill P, O'Malley AJ, Pomposelli FB, Schermerhorn ML. Thirty-day mortality and late survival with reinterventions and readmissions after open and endovascular aortic aneurysm repair in Medicare beneficiaries. *J Vasc Surg*. 2011; 53:6–12. 3 e1. [PubMed: 21030195]
28. 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. [PubMed: 22964737]
29. Birkmeyer JD, Stukel TA, Siewers AE, Goodney PP, Wennberg DE, Lucas FL. Surgeon volume and operative mortality in the United States. *N Engl J Med*. 2003; 349:2117–27. [PubMed: 14645640]
30. Dimick JB, Cowan JA Jr, Stanley JC, Henke PK, Pronovost PJ, Upchurch GR Jr. Surgeon specialty and provider volumes are related to outcome of intact abdominal aortic aneurysm repair in the United States. *J Vasc Surg*. 2003; 38:739–44. [PubMed: 14560223]
31. Holt PJ, Poloniecki JD, Gerrard D, Loftus IM, Thompson MM. Meta-analysis and systematic review of the relationship between volume and outcome in abdominal aortic aneurysm surgery. *Br J Surg*. 2007; 94:395–403. [PubMed: 17380547]
32. Landon BE, O'Malley AJ, Giles K, Cotterill P, Schermerhorn ML. Volume-outcome relationships and abdominal aortic aneurysm repair. *Circulation*. 2010; 122:1290–7. [PubMed: 20837892]
33. McPhee JT, Robinson WP 3rd, Eslami MH, Arous EJ, Messina LM, Schanzer A. Surgeon case volume, not institution case volume, is the primary determinant of in-hospital mortality after elective open abdominal aortic aneurysm repair. *J Vasc Surg*. 2011; 53:591–9 e2. [PubMed: 21144692]
34. Zettervall SL, Schermerhorn M, Soden PA, McCallum JC, Shean KE, Deery SE, et al. Effect of surgeon and hospital volume on mortality following open and endovascular repair of abdominal aortic aneurysms. *J Vasc Surg*. 2016; 63 In Press.

35. Edwards ST, Schermerhorn ML, O'Malley AJ, Bensley RP, Hurks R, Cotterill P, et al. Comparative effectiveness of endovascular versus open repair of ruptured abdominal aortic aneurysm in the Medicare population. *J Vasc Surg.* 2014; 59:575–82. [PubMed: 24342064]
36. Evar trial participants. Endovascular aneurysm repair versus open repair in patients with abdominal aortic aneurysm (EVAR trial 1): randomised controlled trial. *Lancet.* 2005; 365:2179–86. [PubMed: 15978925]
37. Prinssen M, Buskens E, Blankensteijn JD. The Dutch Randomised Endovascular Aneurysm Management (DREAM) trial. Background, design and methods. *J Cardiovasc Surg (Torino).* 2002; 43:379–84.
38. Endarterectomy for asymptomatic carotid artery stenosis. Executive Committee for the Asymptomatic Carotid Atherosclerosis Study. *JAMA.* 1995; 273:1421–8. [PubMed: 7723155]
39. North American Symptomatic Carotid Endarterectomy Trial C. Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis. *N Engl J Med.* 1991; 325:445–53. [PubMed: 1852179]
40. Wennberg DE, Lucas FL, Birkmeyer JD, Bredenberg CE, Fisher ES. Variation in carotid endarterectomy mortality in the Medicare population: trial hospitals, volume, and patient characteristics. *JAMA.* 1998; 279:1278–81. [PubMed: 9565008]

Figure 1.

Bi-weekly death rates through one year in US Medicare beneficiaries undergoing open and endovascular abdominal aortic aneurysm repair from 2001 to 2004 (from Schermerhorn et al., *JACS* 2011; with permission from Elsevier).²¹

Figure 2. Freedom from rupture, aneurysm-related reintervention, or laparotomy-related reintervention (from Schermerhorn *et al.*, *NEJM* 2015; with permission from the Massachusetts Medical Society).¹⁹

Figure 3. Long-term survival in Medicare beneficiaries with and without reinterventions after initial open or endovascular aneurysm repair (from Giles et al., *J Vasc Surg* 2011; with permission from Elsevier).²⁷

Figure 4. Late survival amongst Medicare beneficiaries undergoing EVAR versus open repair, after excluding those patients who experienced late rupture or reintervention.

Figure 5. Short-term AAA-related deaths per 100,000 US Medicare beneficiaries (age and gender adjusted), 1995–2008 (from Schermerhorn et al., *Ann Surg* 2012; with permission from Wolters Kluwer Health, Inc.).²⁸

Figure 6.

Operative mortality for EVAR, open repair, and total AAA repairs for US Medicare beneficiaries, 1995–2008, in A) intact AAA, and B) ruptured AAA (from Schermerhorn et al., *Ann Surg* 2012; with permission from Wolters Kluwer Health, Inc.).²⁸

Table I

Scoring method for prediction of mortality after endovascular or open abdominal aortic aneurysm repair (from Giles et al., *J Vasc Surg* 2009; with permission from Elsevier).²³

Risk factor	Score
Age	
> 80	+ 11
76–80	+ 6
71–75	+ 1
Female	+ 4
Renal failure	
Dialysis dependent	+ 9
No dialysis	+ 7
Congestive heart failure	+ 6
Peripheral or cerebro-basilar vascular disease	+ 3

Total score =

Risk	Score range	Open	EVAR
		Predicted mortality	Predicted Mortality
High	> 11	> 6.3%	> 2%
Medium	3–11	2.8–6.3%	0.9–2.0%
Low	< 3	< 2.8%	< 1%

Table II

Hazard ratio of survival following EVAR compared to open AAA repair at different periods of time following intervention (HR < 1 favors EVAR) (from Schermerhorn et al., *NEJM* 2015; *with permission from the Massachusetts Medical Society*).¹⁹

Time segment	Hazard ratio	95% CI	P-Value
0–30 days	0.32	0.29 – 0.35	< .001
31–90 days	0.64	0.58 – 0.71	< .001
91 days - 1 year	1.14	1.06 – 1.21	< .001
1–4 years	1.17	1.13 – 1.21	< .001
4–8 years	1.05	1.00 – 1.09	.031

Table III

Eight-year outcomes after endovascular and open repair of AAA (from Schermerhorn et al., *NEJM* 2015; *with permission from the Massachusetts Medical Society*).¹⁹

Outcome	EVAR (N = 39,966)	Open (N = 39,966)	P-value
Death	14,548 (55%)	14,681 (55%)	.76
Rupture of aneurysm	962 (5.4%)	353 (1.4%)	< .001
Any aneurysm-related reintervention	4,165 (19%)	754 (3.7%)	< .001
Major reintervention	392 (2.3%)	186 (0.8%)	< .001
Minor reintervention	3,924 (18%)	597 (3.1%)	< .001
Minor reintervention for embolization	1,857 (8.0%)	161 (1.0%)	< .001
Hospitalization for AAA without reintervention	233 (1.2%)	55 (0.3%)	< .001
Reintervention for laparotomy-related complication	1,695 (8.2%)	4,427 (18%)	< .001
Repair of abdominal wall hernia	610 (2.7%)	3,070 (11%)	< .001
Lysis of adhesions without bowel resection	238 (1.4%)	654 (3.1%)	< .001
Bowel resection	1,035 (5.2%)	1,199 (6.0%)	.008
Admission for bowel obstruction, nonoperative	3,510 (17%)	4,805 (22%)	< .001
Aneurysm-related intervention or intervention for complications related to laparotomy	5,614 (25%)	5,034 (21%)	< .001
Hospitalization related to aneurysm or for complications related to laparotomy, without intervention	3,710 (18%)	4,846 (22%)	< .001
Reintervention or hospitalization without intervention for rupture, aneurysm, or complications related to laparotomy	6,279 (28%)	5,355 (22%)	< .001