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Outcomes after endovascular aneurysm repair conversion and primary aortic repair for urgent and emergency indications in the Society for Vascular Surgery Vascular Quality Initiative

Salvatore T. Scali, MD, **Sara J. Runge, MD**, **Robert J. Feezor, MD**, **Kristina A. Giles, MD**, **Javairiah Fatima, MD**, **Scott A. Berceli, MD, PhD**, **Thomas S. Huber, MD, PhD**, and **Adam W. Beck, MD**

Division of Vascular Surgery and Endovascular Therapy, University of Florida College of Medicine

Abstract

Objective—Open conversion after endovascular aortic aneurysm repair (EVAR-c) is performed nonelectively in up to 60% of cases. EVAR-c has been reported to have significantly greater risk of postoperative morbidity and mortality than primary aortic repair, but few data exist on outcomes for symptomatic or ruptured presentations. This study determined outcomes and identified predictors of postoperative major adverse cardiac events (MACEs) and mortality for patients undergoing nonelective EVAR-c compared with nonelective primary aortic repair (PAR) in the Vascular Quality Initiative (VQI).

Methods—All VQI patients undergoing urgent/emergency EVAR-c or urgent/emergency PAR from 2002 to 2014 were reviewed. Urgent presentation was defined by repair ≤24 hours of a nonelective admission, and emergency operations had clinical or radiographic evidence, or both, of rupture. End points included in-hospital MACE (myocardial infarction, dysrhythmia, congestive heart failure) and 30-day mortality. Possible covariates identified on univariate analysis ($P < .2$) were entered into a multivariable model, and stepwise elimination identified the best subset of predictors. Generalized estimating equations logistic regression analysis was used to determine the relative effect of EVAR-c compared with PAR on outcomes.

Results—During the study interval, we identified 277 EVAR-c, and 118 (43%) underwent urgent/emergency repair. nonelective PAR was performed in 1388 of 6152 total (23%). EVAR-c

Correspondence: Salvatore T. Scali, MD, FACS, RPVI, Division of Vascular Surgery and Endovascular Therapy, University of Florida School of Medicine, PO Box 100128, 1600 SW Archer Rd, Ste NG-45, Gainesville, FL 32610 salvatore.scali@surgery.ufl.edu.

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patients were older (75 ± 9 vs 71 ± 10 years; $P < .0001$), more likely to be male (84% vs 74%; $P = .02$), and had a higher prevalence of hypertension (88% vs 79%; $P = .02$) and coronary artery disease (38% vs 27%; $P = .01$). No differences in MACE (EVAR-c, 31% [n = 34] vs PAR, 30% [n =398]) or any major postoperative complication (EVAR-c, 57% [n =63] vs PAR, 55% [n =740]; $P = .8$) were found; however, 30-day mortality was significantly greater in EVAR-c (37% [n = 41]) than in (PAR, 24% [n = 291]; $P = .003$), with an odds ratio (OR) of 2.2 (95% confidence interval [CI], 1.04–4.77; $P = .04$) for EVAR-c. Predictors of any MACE included age (OR, ×1.03 for each additional year; 95% CI, 1.01–1.03; $P = .0002$), male gender (OR, 1.3; 95% CI, 1.03–1.67; $P = .03$), body mass index ≤20 kg/m^2 (OR, 1.8; 95% CI, 1.13–2.87; $P = .01$), chronic obstructive pulmonary disease (OR, 1.2; 95% CI, 0.86–1.80; $P = .25$), congestive heart failure (OR, 1.5; 95% CI, 0.98–2.34; $P = .06$), preoperative chronic β-blocker use (OR, 1.3; 95% CI, 0.97–1.63; $P = .09$), and emergency presentation (OR, 2.3; 95% CI, 1.8–3.01; area under the curve, 0.70; $P < .0001$). Significant predictors for 30-day mortality were age (OR × 1.07 for each additional year; 95% CI, 1.05–1.09; $P < .0001$), female gender (OR, 1.6; 95% CI, 1.01–2.46; $P = .04$), preoperative creatinine >1.8 mg/dL (OR, 1.6; 95% CI, 1.04–2.35; $P = .03$), an emergency presentation (OR, 4.8; 95% CI, 2.93–7.93; $P < .0001$), and renal/visceral ischemia (OR, × 1.1 for each unit increase log (time-minutes); 95% CI, 1.02–1.22; area under the curve, 0.84; $P = .01$).

Conclusions—Nonelective EVAR-c patients are older and have higher prevalence of cardiovascular risk factors than PAR patients. Similar rates of postoperative complications occur; however, urgent/emergency EVAR-c has a significantly higher risk of 30-day mortality than nonelective PAR. Several variables are identified that predict outcomes after these repairs and may help risk stratify patients to further inform clinical decision making when patients present nonelectively with EVAR failure.

The optimal treatment for a failing endovascular aortic aneurysm repair (EVAR) remains unresolved. There is a complex treatment algorithm for the management of a failing EVAR that includes rupture risk, treatment risk, life expectancy, patient preference, failure mode, available technology, and durability of the remedial strategy. The risk associated with nonelective open conversion of a failed EVAR (EVAR-c) is relevant to the treatment algorithm given that 30% to 60% of patients present with symptomatic or ruptured indications, or both.[1] However, the complication rates after nonelective EVAR-c are poorly defined. The published experience with nonelective EVAR-c includes <100 patients and predominantly consists of single-center retrospective reviews with heterogeneous populations of elective and nonelective patients.[1–7]

In contrast, outcomes after nonelective primary open abdominal aortic aneurysm (AAA) repair (PAR) are well established, with 30-day mortality rates of 1% to 20% after urgent intact open aneurysm repair, and 30% to 50% for emergency/ruptured presentations.[8–11] Multiple predictors have been identified to influence outcomes after symptomatic/ruptured PAR, including age, hemodynamic instability, gender, and aortic cross-clamp level.[12] Notably, several reportshighlight elevated rates of morbidity and mortality after EVAR-c and frequently cite increased procedural complexity and patient-level factors as the drivers of these outcomes.[1–7] To date, there are neither well-established descriptions of what the additive risk of EVAR-c is compared with native open AAA repair nor validated predictors of outcomes.

The purpose of this analysis was to determine outcomes of nonelective EVAR-c and benchmark them to nonelective PAR in the Vascular Quality Initiative (VQI). In addition, we sought to define the additive risk of EVAR-c and identify predictors of postoperative morbidity and mortality.

METHODS

This study was approved by the Society for Vascular Surgery VQI Research Advisory Committee and includes national data from all VQI regional quality groups. Details regarding this multicenter collaboration have been published and are available at www.vascularqualityinitiative.org.[13,14] The University of Florida Institutional Review Board also approved the study, and the requirement for patient consent was waived.

Patient cohort

All VQI patients undergoing nonelective EVAR-c and nonelective PAR from 2002 to 2014 were reviewed. Nonelective indications are categorized in the registry as urgent/symptomatic or emergency/ruptured. All elective EVAR-c and elective PAR procedures were intentionally excluded. Of note, the VQI does not record patients undergoing open aneurysm repair for traumatic, infectious, or anastomotic pseudoaneurysm indications and does not record aortic aneurysm repairs involving a major renal artery such that the proximal aortic anastomosis is above at least one major renal artery and reimplantation or bypass of a main renal artery is required. However, data are captured for infrarenal AAA repairs with concomitant renal bypass that is performed to treat renal artery occlusive disease. In addition, open AAA repair occurring below the main renal arteries that requires ligation, reimplantation, or bypass of accessory renal arteries is included. Finally, the registry excludes isolated open iliac aneurysm repair that does not involve an anastomosis to the aorta and revision of previous open AAA repairs.

The VQI does not record unique operative details specific to the conduct of EVAR-c such as the type of endoprosthesis removed, whether partial or total explant of the endograft occurred, or the specific indication for conversion. Information about the index EVAR procedure (eg, whether an endoleak was present, anatomic variables, device characteristics, date of the index EVAR, etc.) is not available. Similarly, no data on prior endovascular remedial interventions after the index EVAR are captured, and no information is available on use of intra-aortic balloon occlusion during nonelective PAR or EVAR-c cases. Nonetheless, variables for both cohorts are captured for procedure time, renal/visceral ischemia, blood loss, transfusion of crystalloid, colloid, or blood products, anastomotic sites, graft size and configuration, patency of the inferior mesenteric artery and hypogastric arteries, use of pharmacologic agents (eg, heparin, mannitol, renal perfusion, vasopressors), adjunctive procedures, exposure, and aortic cross-clamp position.

Definitions

Urgent repair was defined by operation ≤24 hours of a nonelective admission due to symptomatic presentation. Emergency operations had clinical or radiographic evidence, or both, of rupture. All patient demographic and clinical variables (>100 in total) are

prospectively aggregated in the VQI registry. The definitions for specific comorbidities, procedure-related variables, adjunctive procedures, and postoperative complications are available upon request on-line at www.vascularqualityinitiative.org/components/svs-pso.[13,14]

For the purposes of this analysis, all reported postoperative complications were in-hospital events. Major adverse cardiac events (MACEs) were defined as postoperative development of arrhythmia requiring treatment, congestive heart failure, or myocardial infarction. Myocardial infarction was defined as new ST or T wave electrocardiogram changes, troponin elevation, or documentation by echocardiogram or other imaging. Clinically significant arrhythmias included new atrial or ventricular rhythm disturbances requiring treatment with medication or cardio-version, or both. Congestive heart failure included new pulmonary edema documented by chest radiograph and requiring treatment or monitoring, or both, in the intensive care unit. All mortality events were verified by query of the Social Security Death Master File.

End points and statistics

The primary end point was 30-day mortality. Secondary end points included MACE, complications, and long-term survival. The primary goal of the analysis was to estimate the effect of nonelective EVAR-c relative to nonelective PAR on the likelihood of death ≤30 days or development of in-hospital MACE, or both. Because the data set contained a relatively small number of 30-day deaths (n = 332) and MACE (n = 436), inclusion of all possible covariates in multivariable models was not feasible.

Instead, we entered all univariate predictors ($P < .2$), except EVAR-c, into a logistic regression model with 30-day death or MACE as the outcome and used a stepwise elimination algorithm based on the Akaike information criterion[15] to reduce the number of covariates to a best subset of predictors. To assess the additive risk of EVAR-c on the 30-day mortality and MACE end points, we then included EVAR-c along with the chosen predictors in the reduced 30-day mortality and MACE prediction models. Model validation was completed by bootstrapping 1000 iterations. Generalized estimating equations logistic regression analysis was used to account for clustering of observations within medical centers and to estimate the additive risk of the EVAR-c variable. We used the Kaplan-Meier method with log-rank testing to compare long-term survival across the entire data set between patients receiving nonelective EVAR-c and nonelective PAR. Statistical analyses were performed using R 3.1.3 software (The R Foundation for Statistical Computing, Vienna, Austria). $P < .05$ was considered statistically significant.

RESULTS

Demographics and comorbidities

During the study interval, we identified 277 EVAR-c and 118 (43%) underwent urgent/emergent repair. Nonelective PAR was performed in 1388 of 6152 total (23%). Most patients in either nonelective cohort presented with evidence of rupture (Fig 1). Compared with PAR patients, EVAR-c patients were older (75 ± 9 vs 71 ± 10 years; $P < .0001$), more likely to be

male (84% vs 74%; $P = .02$), and had a higher prevalence of hypertension (88% vs 79%; $P = .02$) and coronary artery disease (38% vs 27%; $P = .01$). Nonelective PAR patients more frequently had a preoperative smoking history (83% vs 79%; $P < .0001$). Additional details regarding demographics, comorbidities, and preoperative medication use are reported in Table I.

Operative details

There were several significant differences in operative variables between EVAR-c and PAR patients. Compared with the nonelective PAR cohort, the nonelective VQI patients undergoing EVAR-c more often had retroperitoneal aortic exposure (27% vs 13%; $P = .0002$), were more often repaired with a bifurcated graft (62% vs 43%; $P = .0001$), experienced greater median (25th, 75th quartile) blood loss (3000 [1900, 5100] mL vs 2000 [1050, 4000] mL; $P < .0001$) and had longer procedure times (260 ± 140 minutes vs 220 ± 100 minutes; $P = .0002$). Although PAR patients more frequently received supramesenteric aortic cross-clamp placement (58% vs 41%; $P < .0001$), the renal/visceral ischemia cross-clamp times were significantly longer for nonelective EVAR-c patients (20 ± 29 vs 16 ± 28 minutes; $P = .007$). Additional information regarding intraoperative details is reported in Table II.

Postoperative outcomes

Postoperative outcomes after nonelective EVAR-c and nonelective PAR are summarized in Table III. The 30-day mortality rate was significantly higher for EVAR-c (37% [n = 41]) than for PAR (24% [n = 291]; $P = .003$). Notably, a different 30-day mortality risk was identified for symptomatic and ruptured presentations (Fig 2). No differences in length of stay, rates of MACE (EVAR-c: 31% [n = 34] vs PAR: 30% [n = 398]; odds ratio [OR], 0.97; 95% confidence interval [CI], 0.6–1.4; $P = .9$), or any major postoperative complications were found for EVAR-c (57% [n = 63]) vs PAR (55% [n = 740]; $P = .8$). To explore additive risk of EVAR-c compared with PAR, generalized estimating logistic regression analysis was used to further control for clustering of observations within and between centers. No additive risk of postoperative MACE for EVAR-c was noted compared with PAR (OR, 0.97; 95% CI, 0.6–1.4; $P = .9$). However, EVAR-c patients had a twofold higher risk of 30-day mortality (OR, 2.2; 95% CI, 1.04–4.8; $P = .04$).

MACE outcome predictors

A single prediction model for MACEs was generated using nonelective EVAR-c and nonelective PAR patients because there was no difference in MACE rates on univariate analysis or by generalized estimating logistic regression analysis. Not surprisingly, a ruptured presentation, age, and several other well-known demographic and cardiac risk factors were important predictors of the composite end point (area under the curve [AUC], 0.70; 95% CI, 0.67–0.87; Table IV).

Thirty-day mortality predictors

Among nonelective EVAR-c and PAR patients, there were 332 postoperative 30-day mortality events. In contrast to MACE prediction, EVAR-c was noted to significantly

increase risk of 30-day mortality on univariate analysis (AUC, 0.82; 95% CI, 0.67–0.87; Table IV). Additional variables that were identified as being independent predictors of postoperative death included age, preoperative creatinine >1.8 mg/dL, history of carotid endarterectomy or stenting, or both, and female gender. However, a history of prior lower extremity bypass, retroperitoneal aortic exposure, and chronic β-blocker exposure (on medication >30 days preoperatively) were associated with reduced risk of 30-day mortality. Fig 3 demonstrates how different covariate combinations can influence the risk of 30-day mortality after nonelective EVAR-c compared with PAR.

Because EVAR-c was identified as an independent predictor (OR, 2.2; 95% CI, 1.2–3.8; P = .005) of 30-day mortality, a separate risk model specific to EVAR-c (41 mortality events) was created to understand unique covariates that influence early mortality in these patients (1000 bootstrap iterations; AUC, 0.77; 95% CI, 0.67–0.87). Specifically, a ruptured presentation (OR, 6.1; 95% CI, 2.1–17.8; P = .0009) and suprarenal (OR, 4.1; 95% CI, 1.1–14.9; P = .03) and supramesenteric aortic cross-clamp (OR 4.3; 95% CI, 1.2–16.7; P =.02) position were independently associated with postoperative death. A preoperative history of chronic β-blocker exposure (>30 days) reduced the likelihood of 30-day mortality (OR, 0.6; 95% CI, 0.2–1.4; P = .2).

Survival

A Kaplan-Meier curve demonstrated a greater survival advantage in the PAR group. However, the dissociation in survival occurred primarily during the immediate postoperative interval (Fig 4, *A*). The estimated 1-, 3-, and 5-year survival for PAR was 71% ± 1%, 63% ± 2%, and 52% ± 2%, and for EVAR-c was 59% ± 5%, 45% ± 6%, 45% ± 6%, respectively (log-rank P = .0006). Long-term survival remained significantly different even at 3 years postoperatively according to symptom presentation and whether patients underwent nonelective EVAR-c or nonelective PAR (Fig 4, *B*).

DISCUSSION

This study represents the largest published experience in the literature evaluating outcomes after nonelective EVAR-c. These results are derived from VQI data that are prospectively collected from multiple surgeons, institutions, and regions and provide a broad perspective of outcomes nationally. Notably, the number of observations in this analysis exceeds the sum of the published cases in the literature describing outcomes of nonelective EVAR-c. This facilitated development of a mortality prediction tool that could be used to determine the risk of postoperative mortality for patients with symptomatic or ruptured infrarenal AAAs, or both, after prior EVAR.

As anticipated, significant morbidity and mortality occur after both urgent/emergency EVAR-c and PAR in the VQI; however, this analysis identified important differences between the two groups. First, nonelective EVAR-c patients are older and have higher prevalence of cardiovascular risk factors than nonelective PAR patients. Also, operative complexity is greater for EVAR-c relative to PAR, which is reflected by longer procedure times and greater blood loss. Despite these differences, similar morbidity rates were noted after nonelective EVAR-c and PAR. Notwithstanding similar morbidity rates, the 30-day

mortality was twofold greater after EVAR-c compared with PAR. Several important preoperative predictors were identified that can help risk-stratify patients to further inform clinical decision making and patient and family discussions when urgent or emergency conversion is indicated for a failed EVAR.

Incident rates, morbidity, and mortality of symptomatic and ruptured native AAA repair are well established. The estimated rate of AAA ruptures presenting to the hospital decreased from 33.4 to 16.8 per 100,000 Medicare beneficiaries between 1995 and 2008.[11] Of this cohort, 65% to 75% of patients undergo some form of repair.[11] Historical outcomes of nonelective PAR are associated with surgical mortality rates of 45% to 50%[8,9,16] and overall morbidity of 65% to 90%,[16,17] whereas intact symptomatic AAA repair has reported operative mortality of 1% to 30%,[10] similar to the findings of this study. Interestingly, the 30-day mortality of native ruptured AAA in this analysis was 35.1%, reflecting a continued trend toward improved survival in contemporary series.[10,11]

In contrast to nonelective native AAA repair, the risk of symptomatic or ruptured EVAR-c is poorly characterized. The current literature describing outcomes of EVAR-c is predominantly characterized by single-center, retrospective series that include heterogeneous cohorts of elective and nonelective patients.[1–7] These studies usually lack a comparison group to understand what the additive risk of the endoprosthesis explantation has on the open aortic aneurysm repair. A recent systematic review by Kouvelos et al[1] identified a 30-day mortality rate of 29.2% after nonelective EVAR-c compared with 9.1% for elective presentations. Importantly, these data comprise all indications of EVAR-c, including infectious etiologies, which are not recorded in the VQI.

A recent meta-analysis by Antoniou et al,[18] based on 11 articles with 16,974 patients, reported 190 post-EVAR ruptures, for an incidence of 0.9% (95% CI, 0.77%–1.05%) at a mean of 37 months (range, 16–50 months). The corresponding pooled estimated 30-day mortality was 32% (95% CI, 24%–41%).[18] Notably, the risk of nonelective EVAR-c has been debated since some reports suggest that presence of an endograft confers a survival advantage for a ruptured presentation compared with native aneurysm repair.

For example, May et al[19] reported a significantly lower mortality in ruptured AAA patients with previous EVAR compared with native aneurysm patients (17% vs 54%; $P < .01$). However, significant differences were not demonstrated in other series describing outcome of ruptured EVAR-c vs native ruptured AAA repair (29% vs 39%).[20] The explanation for the mechanism responsible for this protective effect could that 60% of the EVAR-c patients were hemodynamically stable. Alternatively, authors have speculated that the presence of the endograft may limit the quantity and rate of blood loss with the rupture thereby ameliorating hemodynamic changes.[18–20]

Importantly, this analysis refutes the concept that presence of the endograft is protective, because nonelective EVAR-c was associated with a twofold higher risk of 30-day mortality compared with native nonelective AAA repair (absolute difference: urgent presentation, 9.7% [$P = .02$]; ruptured, 16.4% [$P = .009$]; Fig 2). Potential mechanisms responsible for the increased morbidity and mortality of EVAR-c relative to PAR may be related to differences

in comorbidities and overall operative complexity. For example, nonelective EVAR-c patients in this study were typically older and had higher rates of coronary artery disease than the PAR patients. These factors are known to be independent predictors of 30-day mortality for nonelective native AAA repair.[21–23]

Several authors have reported techniques to reduce the morbidity and mortality associated with EVAR conversion, including Nabi et al,[6] who advocate partial endograft removal, when possible, to minimize pararenal and iliac dissection, operative time, and renal/visceral ischemia. Indeed, Marone at al[4] demonstrated 30-day mortality rates of 1.9% and morbidity of 31% in appropriately selected patients (n = 54) with device-specific technical considerations, but only 13% were for nonelective indications. Truly determining how much the existing EVAR device itself contributes to the complexity or the increased mortality is not possible with the available VQI data. However, differences were noted between EVAR-c and PAR with regard to operative conduct, such as exposure, renal/visceral ischemia time, blood loss, and operative times, which suggest that the EVAR device itself may complicate the repair. Each of these variables has previously been reported to be an important predictor of adverse postoperative outcomes for elective and nonelective native AAA repair.[24–26]

To mitigate the additive risk of EVAR-c, our group has adopted a practice of avoiding attempts to explant the device in its entirety unless clinically indicated such as with infected endografts. This often allows a lower cross-clamp position, which can be placed on the device itself, and an anastomosis created to the residual graft, which is frequently en bloc with the aortic wall. Also, the iliac limbs of the existing EVAR device can be clamped directly upon opening the aneurysm sac and can often be left in place and used as the site of the distal anastomoses. This technique avoids unnecessary additional pelvic dissection, which may increase the operative time and blood loss.

If the iliac limbs are left in situ, we attempt to incorporate the terminal aorta or common iliac vessels into the anastomosis such that the anastomosis is really to the native vessel and residual endoprosthesis. In this manner, we attempt to avoid any potential problems from a graft/limb anastomosis such as migration or endoleak. Using these methods, we have been able to obtain similar outcomes in elective EVAR-c compared with open repair.[27] Unfortunately, the VQI does not collect data that allow exact determination of the conduct of the EVAR-c, the type of device explanted, or whether any endograft was left in situ to corroborate these results.

The relevance of the current findings to clinical decision making is underscored by the fact that EVAR has largely supplanted open AAA repair during the last decade due to patient preferences, increasingly widespread availability of devices, and the minimally invasive nature of the procedure that results in early morbidity and mortality benefits compared with PAR.[28,29] As EVAR experience has matured, the technology is being extended to more anatomically and physiologically complex patients such as those with ruptured aneurysms, marginal iliac access/landing zones, or with short angulated proximal seal zones.[30,31] An important consequence of older-generation devices accruing follow-up time and treatment of more difficult anatomies with modern devices may be higher rates of failure.[5,30]

The current data in the literature and this analysis do not support a specific choice between open or endovascular approaches to elective or nonelective reintervention for failing EVAR. However, it is evident that EVAR-c confers higher risk in the nonelective situation and may justify our bias to attempt endovascular salvage techniques in selected patients to reduce mortality similarly to how EVAR has begun to supplant open repair of native ruptured AAA.[32,33] Further, preoperative identification of the clinical and procedure-specific predictors may provide important risk stratification for clinicians that can inform operative decision making and patient and family counseling. Decisions regarding aortic cross-clamp placement and the effect on renal/visceral ischemia time can be factored into the operative plan when possible to reduce postoperative morbidity and mortality.

This study has some important limitations, including modest patient numbers and events for the development of the models. This is a retrospective analysis, and data entry errors are a possibility, although the VQI centers undergo auditing for procedure capture and receive frequent communication and iterative training on maintaining data entry fidelity.

In theory, outcomes for PAR and EVAR-c procedures performed in higher-volume centers may not be applicable across the country. However, a volume-outcome analysis was not fully explored in the VQI data set for this analysis because <10% of the centers performed >20 to 25 cases/year to be identified as “high-volume,” as defined by prior reports. In addition, the relatively small number of nonelective EVAR-c patients (n = 118) limits the ability to perform subgroup analysis.

No information is available about the factors that influenced the choice to pursue open repair of a patient presenting nonelectively with a failed EVAR or native AAA. Importantly, we do not have specific details on the indications for the procedure such as whether the EVAR-c occurred due to endoleak to some other graft-related complication (eg, graft thrombosis). Information about prior remedial endovascular attempts is not available in the VQI, which would be helpful in understanding anatomic or graft-related features, or both, that led to a nonelective presentation and the subsequent decision to explant the device. In addition, we do not have information about the index EVAR procedure may have provided important insights about other risk factors that affect the outcome of nonelective EVAR-c.

The choice to analyze symptomatic and ruptured EVAR-c patients together could be criticized because the outcomes are different between symptomatic and ruptured native AAA repair. However, the risk models we created accounted for symptom status, and an emergency presentation was an important independent predictor for PAR and EVAR-c patients. Although this potentially justifies performing a separate analysis of the different symptom status groups, the primary focus of the study was to determine the additive risk of EVAR-c relative to native AAA repair when patients present nonelectively. Further dichotomization of the groups would decrease the number of events and reduce ability to explore all of the preoperative and intraoperative factors that drive outcomes after nonelective EVAR-c.

Lastly, we concede that the optimal “control” group may not be PAR but could be argued to be a nonelective endovascular remediation of the failing EVAR cohort. Unfortunately, the

VQI does not capture all of these types of procedures, and we cannot define the most appropriate remedial treatment for a failing endograft.

CONCLUSIONS

Nonelective EVAR-c and PAR in the VQI results in significant morbidity and mortality. Nonelective EVAR-c patients are older and have a higher prevalence of cardiovascular risk factors than PAR patients. Similar rates of postoperative complications occur; however, the risk of 30-day mortality is significantly greater after nonelective EVAR-c than after PAR. Several predictors have been identified that may help risk-stratify patients to further inform clinical decision making and patient and family counseling.

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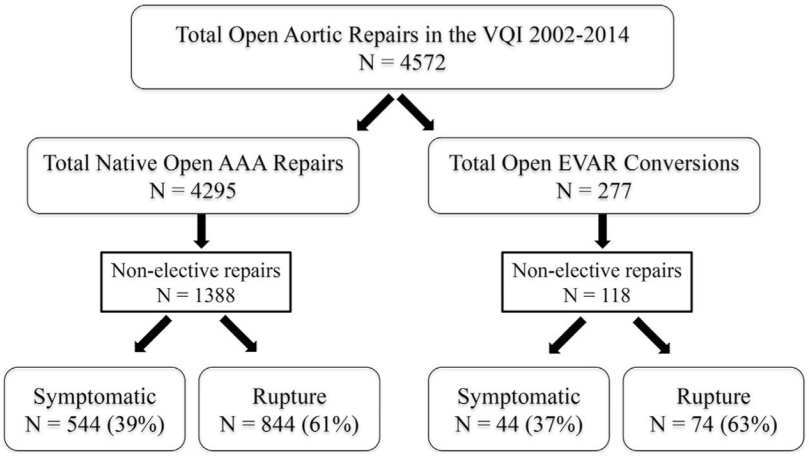


Fig. 1.

This flow chart shows the different inclusion and exclusion groups in the analysis. Of all native abdominal aortic aneurysm (*AAA*) repairs in the Vascular Quality Initiative (*VQI*), 32% were nonelective. There were 277 endovascular aneurysm repair (*EVAR*) conversion (EVAR-c) procedures (6% of total infrarenal aortic operations). Estimating the true incidence of EVAR-c from the VQI is not possible because patients undergoing an index EVAR at a VQI institution can undergo EVAR-c at non-VQI facilities. The mode of presentation for nonelective primary aortic repair and nonelective EVAR-c was similar (~60% for a ruptured indication).

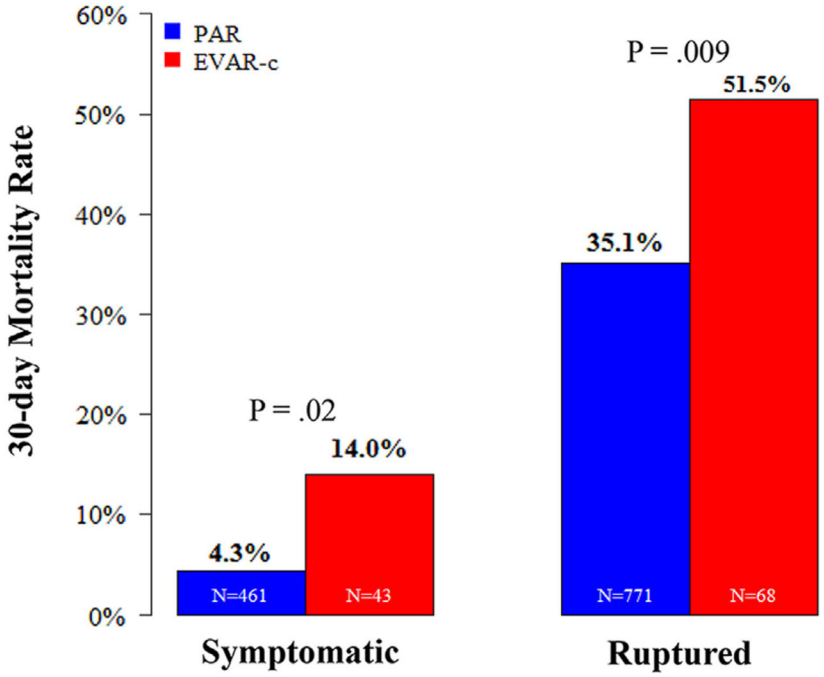


Fig. 2.

This bar graph highlights the expected differences that symptomatic and ruptured presentations have on 30-day postoperative mortality. Not surprisingly, a ruptured presentation for native abdominal aortic aneurysm (AAA) and endovascular aneurysm repair conversion (*EVAR-c*) patients had significantly worse outcome. Interestingly, EVAR-c was not associated with higher rates of postoperative complications; however, mortality was significantly different. EVAR-c patients undergoing nonelective repair had significantly greater risk of 30-day mortality compared with nonelective primary aortic repair (*PAR*) irrespective of presenting with a symptomatic or ruptured indication.

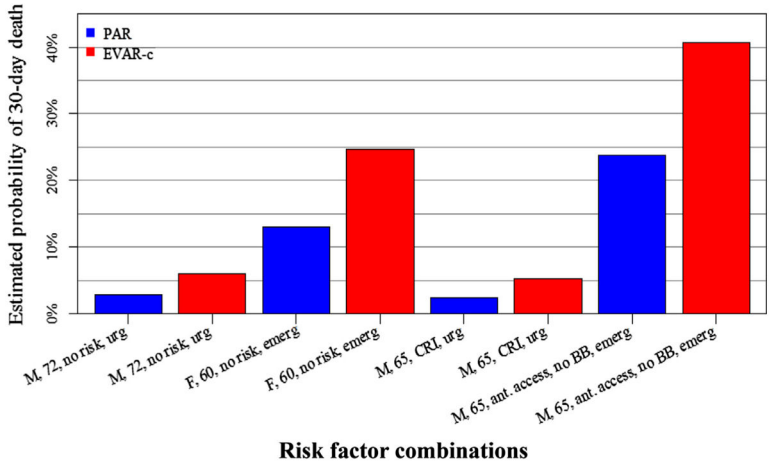
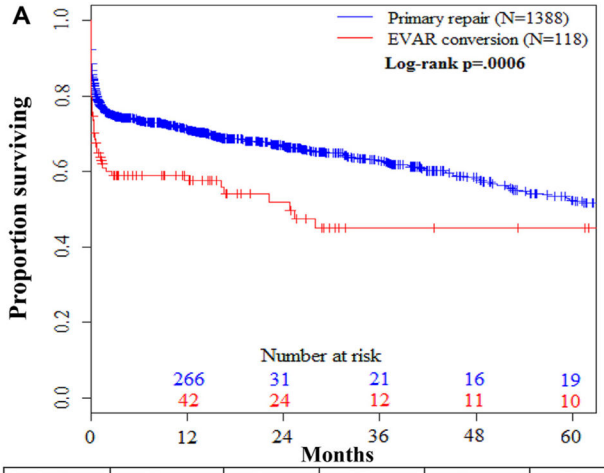
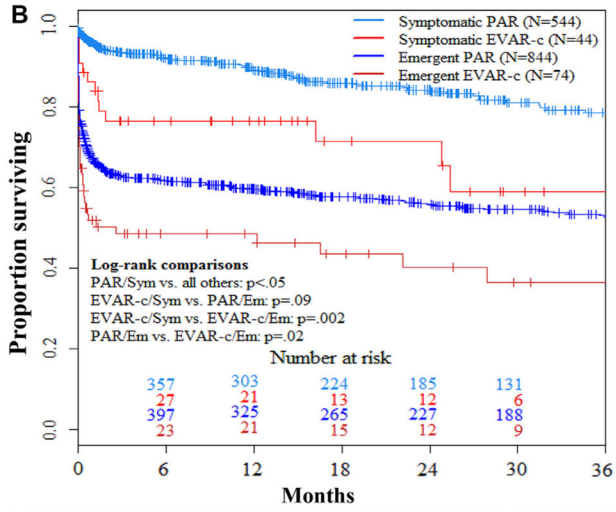


Fig. 3.

The additive risk that endovascular aneurysm repair conversion (*EVAR-c*) has to nonelective abdominal aortic aneurysm (AAA) repair is highlighted. Similar risk profiles are compared among hypothetical nonelective infrarenal aortic aneurysm repair patients, with or without endograft explantation. Various random combinations of different 30-day mortality risk factors (Table IV) are depicted. The 30-day mortality risk is twofold greater for nonelective patients undergoing EVAR-c compared with native AAA repair. *Ant,* Antegrade; *BB,* β-blockers; *CRI,* chronic renal insufficiency; *F,* female; *M,* male; *PAR,* primary aortic repair.



Standard error	12mo	24mo	36mo	48mo	60mo
Primary repair	.01	.01	.01	.02	.02
EVAR-c	.05	.05	.06	.06	.06



Standard error	12mo	24mo	36mo
Symptomatic PAR	.01	.02	.02
Symptomatic EVAR-c	.07	.08	.10
Emergent PAR	.02	.02	.02
Emergent EVAR-c	.06	.06	.07

Fig. 4.

A, The two survival curves highlight the overall survival comparison between nonelective endovascular aneurysm repair conversion (*EVAR-c*) and primary aortic repair (*PAR*) patients in the Vascular Quality Initiative (VQI). PAR patients have a significant survival advantage that is maintained out to at least 3 years (log-rank $P = .0006$). All displayed intervals have <10% standard error of the mean. **B,** The significant differences in survival among the different modes of presentation are demonstrated. Specifically, patients undergoing symptomatic or ruptured aortic repair with or without need for EVAR-c are compared. Among all intergroup comparisons, EVAR-c confers a significantly higher risk of all-cause mortality compared with PAR patients. Notably, EVAR-c patients are frequently older and

have an increased incidence of cardiovascular risk factors that affect long-term survival. All displayed intervals have <10% standard error of the mean.

Table I

Demographics and comorbidities for nonelective primary aortic repair (*PAR*) and nonelective endovascular aneurysm repair conversion (*EVAR-c*) patients in the Vascular Quality Initiative (VQI)

Feature	PAR (n = 1388)	EVAR-c (n = 118)	*P* value[a]
Age, mean ± SD, years	71 ± 10	75 ± 9	<.0001
Male gender, %	74	84	.02
BMI, mean ± SD, kg/m^2	27 ± 6	28 ± 6	.3
Comorbidities, %			
Hypertension	79	88	.02
Coronary artery disease	27	38	.01
Prior CABG/PCI	23	41	<.0001
Chronic obstructive lung disease	37	36	1
Prior/current smoker	83	79	<.0001
Diabetes mellitus	14	18	.2
Creatinine >1.8 mg/dL	13	17	.2
Congestive heart failure	9	12	.3
Medication history, %			
Aspirin	49	58	.05
Statin	46	56	.05
β-blocker	54	62	.09

BMI, Body mass index; *CABG,* coronary artery bypass graft; *PCI,* percutaneous coronary intervention; *SD,* standard deviation.

[a]The χ^2 or Fisher exact tests were used to compare the groups on nominal categoric variables and Mann-Whitney tests were used to compare them on continuous and ordered categoric variables, when appropriate.

Table II

Operative details for patients undergoing nonelective primary aortic repair (*PAR*) and nonelective endovascular aneurysm repair conversion (*EVAR-c*) in the Vascular Quality Initiative (VQI)

Feature[a]	PAR (n = 1388)	EVAR-c (n = 118)	*P* value[b]
Retroperitoneal aortic exposure, %	13	27	.0002
Any concomitant procedure,[c] %	27	23	.4
Bifurcation tube graft, %	43	62	.0001
Estimated blood loss, mL	2000 (1050, 4000)	3000 (1900, 5100)	<.0001
Crystalloid, mL	5000 (3000, 7000)	5000 (2650, 7500)	.5
Intraoperative red blood cell transfusion, U	3 (0, 7)	5 (2, 8)	<.0001
Cross-clamp position, %			
Supramesenteric	58	41	
Suprarenal	10	5	
Intrarenal/infrarenal	32	54	<.0001
Renal/visceral ischemia time, minutes	16 ± 28	20 ± 29	.007
Total procedure time, minutes	220 ± 100	260 ± 140	.0002

[a] Continuous data are shown as the median (25^{th} quartile, 75^{th} quartile) or as the mean ± standard deviation.

[b] The χ^2 or Fisher exact tests were used to compare the groups on nominal categoric variables, and Mann-Whitney tests were used to compare them on continuous and ordered categoric variables, when appropriate.

[c] Concomitant procedure includes renal/visceral bypass or reimplantation, or both, thromboembolectomy, or infrainguinal bypass, or both.

Table III

Postoperative outcomes after nonelective primary aortic repair (*PAR*) and nonelective endovascular aneurysm repair conversion (*EVAR-c*) in the Vascular Quality Initiative (VQI)

Outcome	PAR (n = 1388)	EVAR-c (n = 118)	*P* value[a]
Mortality, %			
Thirty-day	24	37	.003
In-hospital	22	36	.001
Length of stay, median (IQR) days	9 (6, 17)	9 (4, 18)	.7
Discharge to home, %	45	32	.001
Any postoperative complication, %	55	57	.7
MACE[b]	30	31	.8
Bleeding, return to OR	28	35	.5
Any renal injury, %	31	37	
Creatinine increase >0.5 mg/dL	21	22	
Temporary dialysis	7	11	
Permanent dialysis	3	4	.3
Wound complication, %	7	9	.5
Pneumonia, %	6	5	.2
Stroke, %	3	5	.1

IQR, Interquartile range (25^{th} quartile, 75^{th} quartile); *MACE,* major adverse cardiac event; *OR,* operating room.

[a]The χ^2 or Fisher exact tests were used to compare the groups on nominal categoric variables and Mann-Whitney tests were used to compare them on continuous and ordered categoric variables when appropriate.

[b]Includes myocardial infarction, congestive heart failure, arrhythmia requiring medication, and cardioversion.

Table IV

Preoperative predictors of major adverse cardiac event (*MACE*) and 30-day mortality after nonelective primary aortic repair (PAR) and nonelective endovascular aneurysm repair conversion (EVAR-c) in the Vascular Quality Initiative (VQI)

Predictor[a]	OR	95% CI	*P* value
MACE[b] (n = 436 events)			
Rupture presentation	2.5	1.9–3.2	<.0001
Age (per year)	1.03	1.01–1.04	.0001
BMI (<20 kg/m^2)	1.8	1.2–2.8	.008
Congestive heart failure	1.5	0.9–2.3	.05
Male gender	1.3	0.98–1.78	.06
Preoperative chronic β-blocker use	1.2	0.9–1.6	.08
Chronic obstructive pulmonary disease	1.2	0.9–1.6	.1
History of peripheral vascular intervention	0.7	0.4–1.4	.3
Retroperitoneal aortic exposure	0.7	0.5–1.1	.1
Thirty-day mortality[c] (n = 332 events)			
Rupture presentation	8.6	5.5–13.5	<.0001
EVAR-c	2.2	1.2–3.8	.005
Age (per year)	1.06	1.04–1.08	<.0001
Creatinine >1.8 mg/dL	1.3	0.9–1.9	.1
History of CEA/CAS	1.3	0.6–2.5	.5
Female gender	1.3	0.9–1.7	.2
History of lower extremity bypass	0.6	0.2–1.6	.4
Retroperitoneal aortic exposure	0.7	0.4–1.2	.2
Preoperative β-blocker[d]	0.7	0.5–0.9	.03

BMI, Body mass index; *CAS,* carotid artery stent; *CEA,* carotid endarterectomy; *CI,* confidence interval; *OR,* odds ratio.

[a] 1000 bootstrap iterations.

[b] MACE area under the curve (AUC), 0.77 (95% CI, 0.67–0.87).

[c] Thirty-day mortality AUC, 0.82 (95% CI, 0.67–0.87).

[d] Defined as medication >30 days preoperatively.