Short Term and Long Term Outcomes After Endovascular or Open Repair for Ruptured Infrarenal Abdominal Aortic Aneurysms in the Vascular Quality Initiative

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WHAT THIS PAPER ADDS

Within the Vascular Quality Initiative, endovascular aortic repair (EVAR) for ruptured infrarenal abdominal aortic aneurysm (rAAA) has been adopted increasingly with favourable short term outcomes in terms of morbidity and mortality as compared with open aortic repair (OAR). Unlike elective AAA repair, a convergence of survival rates between EVAR and OAR in long term follow up for patients who survived the index hospitalisation was not observed, suggesting that the early significant benefits of EVAR are sustained over time and an endovascular-first strategy in anatomically feasible candidates with rAAA may be associated with long term benefits.

Objective: Repair of ruptured infrarenal abdominal aortic aneurysms (rAAA) has shifted from open surgical (OAR) to endovascular (EVAR) over the last decade. However, the long term impact of EVAR *vs.* OAR for rAAA has not been well described.

Methods: Prospectively collected registry data (Vascular Quality Initiative [VQI]) were analysed retrospectively to identify patients who underwent EVAR or OAR for rAAA (2004–2018). The primary outcome was death (in hospital and overall post-discharge). Inverse probability weighting (IPW) was used to adjust for treatment selection. Poisson regression assessed the number of one year post-discharge re-interventions.

Results: In total, 4257 patients receiving EVAR ($n = 2389$ [56%]) or OAR ($n = 1868$ [44%]) for rAAA were identified. Patients were predominantly male ($n = 3310$ [77.8%]) with a mean ± standard deviation age of 72.7 ± 9.6 years; most ($n = 2449$ [59.4%]) presented with haemodynamic instability. Use of EVAR for rAAA increased from 7.8% in 2004 to 67.2% in 2018. After IPW, OAR was associated with a higher odds of in hospital mortality (odds ratio [OR] 1.76, 95% confidence interval [CI] 1.54–2.01; $p < .001$), which was confirmed after multivariable logistic regression (OR 2.08, 95% CI 1.76–2.45; $p < .001$). Multivariable Cox proportional hazards showed that OAR was also associated with increased overall post-discharge mortality among all patients (hazard ratio 1.36, 95% CI 1.23–1.51; $p < .001$). Within weighted treatment groups, five year survival was significantly different (55% for EVAR *vs.* 46% for OAR; $p < .001$). OAR showed a significantly higher risk of one year post-discharge re-interventions (incidence rate ratio 2.10, 95% CI 1.52–2.89; $p < .001$).

Conclusion: Within the VQI, EVAR for rAAA repair has been increasingly adopted with favourable short term outcomes in terms of morbidity and mortality, as compared with OAR. Unlike elective AAA repair, survival rates between EVAR and OAR do not converge in long term follow up for patients who survived the index hospitalisation.

INTRODUCTION

Repair of ruptured infrarenal abdominal aortic aneurysms (rAAA) has shifted from open surgical (OAR) to endovascular aneurysm repair (EVAR) over the last decade, mainly driven by reduced rates of early mortality and morbidity with endovascular treatment.[1,2] A variety of study designs

and databases have been used to compare EVAR and OAR for rAAA, and studies of various designs from different databases have reached vastly different conclusions.[3] Therefore it remains controversial whether EVAR improves outcomes after rAAA *vs.* OAR. Indeed, the decision to use EVAR instead of OAR needs to incorporate both early and potential late outcomes, in addition to anatomical restrictions, but the long term outcomes of emergency EVAR for rAAA are not well established. Additionally, the identification of optimal patient subgroups that could benefit from either treatment is poorly defined. Thus, the aim of this study was to contrast the short and long term outcomes after EVAR or OAR for rAAA within a large contemporary national registry.

MATERIALS AND METHODS

Data sources and study design

Prospectively collected data from the Society for Vascular Surgery's Vascular Quality Initiative (VQI) were used to identify patients who had undergone EVAR or OAR for rAAA at participating centres between January 2004, and October 2018. The inclusion criterion for cases was any infrarenal AAA that underwent attempted repair. Patients who were selected to not receive AAA repair (for any reason) or who died prior to incision are not included in the registries. The VQI is a national network of regional quality groups made up of >370 North American academic and community hospitals, with data on > 100 distinct variables for each specific modules (https://www.vqi.org/data-analysis/). Patients having symptomatic or elective AAA repair were excluded.

Exposures, definitions, and outcomes

The primary exposure of interest was procedure type, EVAR or OAR. Patients who underwent both OAR and EVAR in the same month and year were assumed to have had the procedures on the same day, assigned as EVAR converted to OAR, and included as EVAR ($n = 20$). Patients who underwent two OAR or two EVAR procedures in the same month and year were excluded ($n = 6$), as were patients who underwent EVAR converted to OAR who were recorded only in the open repair registry without a corresponding record in the EVAR registry ($n = 38$). Patients who underwent repeated aortic procedures during the study period were assigned according to their index operation ($n = 5$).

Pre-operative haemodynamic instability was defined as any of the following: lowest blood pressure <90 mmHg, cardiac arrest, or altered mental status. Average annual centre volume of all elective, urgent, and emergency AAA cases was calculated across all years for each institution that contributed at least one record to the VQI registries, excluding the first year of participation and 2018 (as these years were unlikely to include a full year of data). Average annual centre volume was categorised by quartiles.

The primary outcomes were in hospital mortality and overall post-discharge mortality. Mortality was assessed for all patients by multiple mechanisms, including assessment at clinical follow up in VQI (9—18 months) and matching of patients to the Social Security Death Index and to eligible Medicare claims.

Secondary outcomes included cardiovascular (myocardial infarction [MI]; new onset congestive heart failure [CHF]; and new onset dysrhythmia) and respiratory complications (pneumonia and re-intubation), post-operative hospital and intensive care unit (ICU) length of stay (LOS), adverse discharge status (any other than home), rates of packed red blood cells (PRBC) transfusions and one year post-discharge aortic re-interventions (captured at one year follow up assessment only).

Statistical analysis

Inverse probability weighting (IPW) was used to adjust for treatment selection. This method allows use of the entire data set, giving greater weight to patients with similar characteristics between groups and less weight to those less likely to receive either treatment. To accomplish this, a logistic regression model of undergoing EVAR was developed by including factors with statistical significance or of clinical significance. The IPW was calculated for each patient as the marginal probability of the treatment received divided by the predicted probability of the treatment received derived from the logistic regression model. Patients missing variables used to develop the logistic regression model were excluded ($n = 3$). Standardised differences were used to assess patient factors between repair types before and after weighting. The standardised difference compares the difference in means in units of the pooled standard deviation (SD) and is not influenced by sample size.[4] Furthermore, it allows for the comparison of the relative balance of variables measured in different units (e.g., years *vs.* mmHg). A standardised difference < .10 denotes balance in the studied variable.

Categorical variables were assessed as frequencies. Continuous variables were evaluated as mean ± SD or median (interquartile range) for parametric and non-parametric data, respectively. In the unweighted cohort, differences between groups were calculated using Fisher's exact, Kruskal—Wallis, or chi square tests as appropriate.

In the weighted cohort, univariable and multivariable models were used to identify factors independently associated with outcomes. Logistic regression was used for in hospital mortality, and Cox proportional hazards for overall post-discharge mortality, along with Kaplan—Meier estimates with log rank test to assess differences. Linear regression assessed hospital and ICU LOS, Poisson regression assessed rates of re-interventions, and PRBC; logistic regression assessed MI, new onset dysrhythmia, new onset CHF, respiratory complications, and unfavourable discharge. Statistical significance was defined as a p value < .05. All statistical analyses were performed using SAS version 9.4 (SAS Institute, Cary, NC, USA).

Table 1. Baseline demographics, clinical characteristics, and centre variables before and after inverse probability weighting for endovascular aneurysm repair (EVAR) versus open aneurysm repair (OAR) in patients registered in the Vascular Quality Initiative for treatment of ruptured infrarenal abdominal aortic aneurysms (2004–2018)

	Unweighted statistics			Weighted statistics		
	EVAR (n = 2 389)	OAR (n = 1 868)	Std Diff.	EVAR (n = 2 389)	OAR (n = 1 853)	Std Diff.
Male sex	1 861 (77.9)	1 449 (77.6)	.008	1 849 (77.3)	1 434 (77.4)	.002
Age − y	73 (66, 81)	72 (66, 79)	.105	73 (66, 80)	73 (66, 79)	.004
Race						
White	2 059 (86.2)	1 710 (91.5)	.171	2 116 (88.4)	1 639 (88.4)	<.001
Black or African American	188 (7.9)	72 (3.9)	.172	146 (6.1)	111 (6.0)	.004
Other/unknown	142 (5.9)	86 (4.6)	.060	132 (5.5)	103 (5.6)	.004
BMI − kg/m^2	27.5 (24.0, 31.8)	27.5 (24.2, 31.5)	.030	27.7 (24.2, 31.8)	27.4 (24.0, 31.2)	.060
Hypertension (≥140/90 mmHg or history)	1 855 (78.3)	1 445 (78.5)	.006	1 858 (78.6%)	1 446 (79.2)	.016
History of CAD						
None	1 837 (77.9)	1 415 (77.9)	.001	1 832 (77.8)	1 423 (79.0)	.029
History MI but no symptoms	379 (16.1)	291 (16.0)	.002	375 (15.9)	281 (15.6)	.009
Stable angina	88 (3.7)	82 (4.5)	.039	94 (4.0)	69 (3.8)	.009
Unstable angina or MI < 6 mo	7 (0.3)	7 (0.4)	.006	10 (0.4)	4 (0.2)	.011
MI < 6 mo	35 (1.5)	14 (0.8)	.046	33 (1.4)	16 (0.9)	.033
Unstable angina	12 (0.5)	8 (0.4)	.004	11 (0.5)	8 (0.4)	.002
Prior CABG/PCI						
None	1 842 (78.2)	1 413 (77.6)	.012	1 852 (78.7)	1 401 (77.7)	.023
PTCA/CABG < 5 y ago	153 (6.5)	134 (7.4)	.034	159 (6.8)	132 (7.3)	.022
PTCA/CABG ≥ 5 y ago	362 (15.4)	273 (15.0)	.010	343 (14.6)	269 (14.9)	.011
History of CHF						
None	2 063 (87.6)	1 650 (91.0)	.108	2 080 (89.0)	1 619 (89.8)	.027
Asymp., hx CHF	169 (7.2)	98 (5.4)	.073	152 (6.5)	111 (6.2)	.013
Mild	71 (3.0)	42 (2.3)	.044	64 (2.7)	43 (2.4)	.022
Moderate	34 (1.4)	16 (0.9)	.036	28 (1.2)	19 (1.1)	.007
Severe	17 (0.7)	8 (0.4)	.018	14 (0.6)	10 (0.6)	.003
Pre-operative EF − %						
< 30	48 (2.0)	33 (1.8)	.016	43 (1.8)	37 (2.0)	.013
30–50	121 (5.1)	79 (4.2)	.040	126 (5.3)	77 (4.1)	.054
> 50	279 (11.7)	219 (11.7)	.001	257 (10.8)	224 (12.1)	.041
Not done	1 289 (54.0)	1 008 (54.0)	<.001	1 330 (55.6)	975 (52.6)	.060
Unknown	652 (27.3)	529 (28.3)	.023	636 (26.6)	541 (29.2)	.058
Prior CEA/CAS	29 (1.4)	50 (3.6)	.141	41 (2.1)	35 (2.3)	.014
Prior bypass	48 (2.0)	43 (2.4)	.020	46 (2.0)	42 (2.3)	.024
Prior PVI (PTA/stent)	74 (3.1)	65 (3.6)	.023	73 (3.1)	67 (3.7)	.035
Prior major amputation	16 (0.7)	7 (0.4)	.019	14 (0.6)	8 (0.4)	.008
Prior aneurysm repair	67 (2.9)	171 (9.4)	.271	120 (5.2)	102 (5.7)	0.024
Diabetes mellitus						
None	1 980 (83.9)	1 557 (85.1)	.034	1 986 (84.2)	1 551 (85.6)	.038
Diet	91 (3.9)	83 (4.5)	.034	91 (3.9)	77 (4.2)	.020
Non-insulin meds	202 (8.6)	136 (7.4)	.041	201 (8.5)	130 (7.2)	.051
Insulin	87 (3.7)	53 (2.9)	.044	79 (3.3)	54 (3.0)	.022
Smoking						
Never	549 (23.4)	312 (17.3)	.153	481 (20.6)	372 (20.6)	<.001
Prior	839 (35.8)	601 (33.2)	.053	792 (33.9)	612 (33.9)	<.001
Current	957 (40.8)	895 (49.5)	.175	1063 (45.5)	822 (45.5)	<.001
COPD						
None	1 663 (70.7)	1 236 (67.7)	.065	1 614 (68.6)	1 251 (69.1)	.012
Not treated	235 (10.0)	238 (13.0)	.095	278 (11.8)	204 (11.3)	.016
On meds	314 (13.4)	281 (15.4)	.058	348 (14.8)	266 (14.7)	.002
On home oxygen	140 (6.0)	71 (3.9)	.096	114 (4.8)	88 (4.9)	.002
Pre-operative creatinine	1.2 (1.0, 1.6)	1.2 (1.0, 1.6)	.002	1.2 (1.0, 1.6)	1.2 (1.0, 1.6)	.007
Pre-operative dialysis						
No	2 331 (98.1)	1 832 (99.2)	.070	2 340 (98.7)	1 816 (98.9)	.015
Functioning transplant	3 (0.1)	6 (0.3)	.013	4 (0.2)	4 (0.2)	.002

Continued

Table 1-*continued*

	Unweighted statistics			Weighted statistics		
	EVAR (n = 2 389)	OAR (n = 1 868)	Std Diff.	EVAR (n = 2 389)	OAR (n = 1 853)	Std Diff.
On dialysis	41 (1.7)	8 (0.4)	.083	27 (1.1)	16 (0.9)	.017
Pre-operative beta blocker						
No	1 264 (54.4)	951 (52.6)	.036	1 239 (53.2)	961 (53.4)	.005
Pre-operative 1−30 d	64 (2.8)	85 (4.7)	.103	90 (3.9)	66 (3.7)	.012
Chronic > 30 d	842 (36.2)	637 (35.2)	.021	829 (35.6)	638 (35.5)	.003
No, for medical reason	32 (1.4)	32 (1.8)	.025	43 (1.9)	32 (1.8)	.007
Operation day only	114 (4.9)	90 (5.0)	.003	114 (4.9)	91 (5.1)	.008
Non-compliant	9 (0.4)	14 (0.8)	.025	12 (0.5)	10 (0.5)	.003
Pre-operative ACE-I/ARB						
No	1 347 (65.5)	918 (66.8)	.027	1 261 (65.4)	982 (66.0)	.012
Yes	642 (31.2)	408 (29.7)	.034	599 (31.1)	463 (31.1)	.001
No for medical reason	52 (2.5)	35 (2.5)	.001	56 (2.9)	33 (2.2)	.045
Non-compliant	14 (0.7)	13 (0.9)	.017	13 (0.7)	11 (0.7)	.004
Pre-operative statin						
No	1 272 (54.6)	987 (54.7)	.001	1 294 (55.5)	958 (53.4)	.042
Yes	1 000 (42.9)	755 (41.8)	.023	974 (41.8)	771 (43.0)	.025
No for medical reason	38 (1.6)	40 (2.2)	.037	45 (1.9)	42 (2.3)	.029
Non-compliant	20 (0.9)	24 (1.3)	.030	19 (0.8)	22 (1.2)	.026
Pre-operative antiplatelets	980 (42.1)	779 (43.1)	.021	953 (40.9)	768 (42.8)	.039
Pre-operative chronic anticoagulant						
None	1 755 (85.1)	1 203 (87.4)	.068	1 655 (85.5)	1 289 (86.5)	.028
Warfarin	206 (10.0)	102 (7.4)	.091	182 (9.4)	130 (8.7)	.024
Other	67 (3.2)	44 (3.2)	.003	58 (3.0)	49 (3.3)	.017
No, for medical reason	30 (1.5)	25 (1.8)	.023	36 (1.9)	21 (1.4)	.027
Non-compliant	5 (0.2)	2 (0.1)	.006	5 (0.2)	2 (0.1)	.009
Ambulatory status						
Fully ambulatory	522 (84.1)	1 266 (90.8)	.206	528 (85.2)	1 353 (90.4)	.160
Ambulatory with assistance	65 (10.5)	96 (6.9)	.127	60 (9.7)	108 (7.2)	.086
Wheelchair	21 (3.4)	13 (0.9)	.157	18 (2.9)	15 (1.0)	.124
Bedridden	13 (2.1)	19 (1.4)	.047	14 (2.3)	20 (1.3)	.058
Living status						
At home	2 298 (96.9)	1 806 (97.7)	.052	2 307 (97.2)	1 786 (97.6)	.027
Nursing home	61 (2.6)	38 (2.1)	.033	52 (2.2)	40 (2.2)	<.001
Homeless	13 (0.5)	4 (0.2)	.021	15 (0.6)	4 (0.2)	.028
Lowest pre-intubation BP − mmHg	91.0 (70.0, 116.0)	84.0 (64.0, 110.0)	.230	90.0 (70.0, 113.0)	89.0 (66.0, 112.0)	.052
Lowest pre-intubation BP < 90 mmHg	1 046 (45.9)	993 (55.4)	.190	1 144 (49.9)	897 (50.7)	.016
Mental status						
Normal	1 780 (75.9)	1 304 (70.6)	.119	1 754 (74.4)	1 337 (73.5)	.021
Disoriented	332 (14.2)	269 (14.6)	.012	329 (13.9)	261 (14.4)	.012
Unconscious	233 (9.9)	273 (14.8)	.148	274 (11.6)	220 (12.1)	.015
Cardiac arrest	195 (8.3)	253 (13.7)	.173	240 (10.2)	192 (10.5)	.012
Pre-operative haemodynamic instability	1 274 (55.5)	1 175 (64.2)	.178	1 374 (59.4)	1 076 (59.8)	.009
Pre-operative haemoglobin − g/dL	11.5 (9.8, 13.2)	11.6 (10.0, 13.3)	.007	11.5 (9.8, 13.2)	11.6 (10.0, 13.3)	.014
Year of procedure						
2004	4 (0.2)	47 (2.5)	.150	29 (1.2)	23 (1.2)	.001
2005	4 (0.2)	39 (2.1)	.123	36 (1.5)	19 (1.0)	.031
2006	9 (0.4)	42 (2.2)	.120	21 (0.9)	22 (1.2)	.018
2007	10 (0.4)	40 (2.1)	.110	26 (1.1)	22 (1.2)	.008
2008	9 (0.4)	38 (2.0)	.106	24 (1.0)	20 (1.1)	.007
2009	21 (0.9)	43 (2.3)	.091	36 (1.5)	33 (1.8)	.017
2010	47 (2.0)	65 (3.5)	.093	64 (2.7)	49 (2.6)	.002
2011	109 (4.6)	98 (5.2)	.032	115 (4.8)	89 (4.8)	.001
2012	190 (8.0)	144 (7.7)	.009	185 (7.7)	146 (7.9)	.004
2013	269 (11.3)	209 (11.2)	.002	258 (10.8)	198 (10.7)	.004
2014	300 (12.6)	263 (14.1)	.045	322 (13.4)	244 (13.2)	.008
2015	399 (16.7)	252 (13.5)	.090	373 (15.6)	286 (15.4)	.005
2016	377 (15.8)	246 (13.2)	.074	341 (14.2)	263 (14.2)	.002
2017	375 (15.7)	212 (11.3)	.127	339 (14.2)	259 (14.0)	.006
2018	266 (11.1)	130 (7.0)	.146	223 (9.3)	181 (9.8)	.016

Table 1-*continued*

	Unweighted statistics			Weighted statistics		
	EVAR (n = 2 389)	OAR (n = 1 868)	Std Diff.	EVAR (n = 2 389)	OAR (n = 1 853)	Std Diff.
Surgery on weekend						
Monday—Friday	1 697 (71.0)	1 362 (72.9)	.042	1 704 (71.2)	1 361 (73.4)	.050
Saturday—Sunday	692 (29.0)	506 (27.1)		689 (28.8)	492 (26.6)	
AAA cases/centre/y						
≤ 15	73 (3.1)	118 (6.3)	.155	70 (2.9)	110 (5.9)	.145
> 15—29	263 (11.0)	235 (12.6)	.049	245 (10.2)	254 (13.7)	.107
> 29—53	634 (26.5)	412 (22.1)	.105	598 (25.0)	401 (21.6)	.079
>53	1 353 (56.6)	1 090 (58.4)	.035	1 421 (59.4)	1 072 (57.8)	.031
Unable to calculate	66 (2.8)	13 (0.7)	.132	59 (2.5)	16 (0.9)	.101

The data are n (%) or median (Q1, Q3) unless stated otherwise. Patients without a known value were excluded from the denominator of that variable. Number of missing values for each variable: body mass index (BMI), n = 308; hypertension, n = 48; history of coronary artery disease (CAD), n = 82; prior coronary artery bypass grafting (CABG)/percutaneous coronary intervention (PCI), n = 80; history (hx) of congestive heart failure (CHF), n = 89; prior carotid endarterectomy (CEA)/carotid artery stenting (CAS), n = 802; prior bypass, n = 75; prior peripheral vascular intervention (PVI) (percutaneous transluminal angioplasty [PTA]/stent), n = 78; prior major amputation, n = 73; prior aneurysm repair, n = 128; diabetes mellitus, n = 68; smoking, n = 104; chronic obstructive pulmonary angioplasty (COPD), n = 79; pre-operative dialysis, n = 36; pre-operative beta blocker, n = 123; pre-operative angiotensin converting enzyme inhibitor (ACE-I)/angiotensin receptor blocker (ARB), n = 828; pre-operative statin, n = 121; pre-operative antiplatelets, n = 121; pre-operative chronic anticoagulant, n = 818; ambulatory status, n = 2 242; living status, n = 37; lowest pre-intubation blood pressure (BP), n = 186; mental status, n = 66; cardiac arrest, n = 58; pre-operative haemodynamic instability, n = 133; pre-operative haemoglobin, n = 287. Std Diff. = standard difference; MI = myocardial infarction; PTCA = percutaneous transluminal coronary angioplasty; Asymp. = asymptomatic; EF = ejection fractionACE-I = angiotensin converting enzyme inhibitor; AAA = abdominal aortic aneurysm.

Ethical approval

This study was approved by the VQI research advisory council and deemed exempt from approval by the Mayo Clinic Institutional Review Board. All patients' personal health information was protected, records and outcomes were de-identified, and no testing or procedures were required for this study. Thus, the need for informed consent was waived.

RESULTS

Cohort characteristics

A total of 4257 patients was identified who received EVAR (n = 2389 [56.1%]) or OAR (n = 1868 [43.9%]) for rAAA during the study period (baseline demographics, clinical characteristics, and centre variables in non-weighted treatment groups are reported in Table S1 [Supplementary Material]; procedural details in non-weighted treatment groups are reported in Table S2 [Supplementary Material]). The use of EVAR for rAAA increased from 7.8% in 2004 to 67.2% in 2018 among all rAAA repairs. Prior to weighting, patients who underwent EVAR were older, with smaller AAA, and lower prevalence of haemodynamic instability at presentation. However, no significant differences were found in pre-operative haemoglobin levels. Average centre volume varied across the two approaches; however, similar proportions of EVAR and OAR patients were at the highest quartile volume centres. Patients who had EVAR had a longer median time from symptoms to repair and from admission to repair. Total procedure time was shorter for EVAR. IPW adjustments resulted in similar weighted treatment groups for comparison of outcomes: the two groups were well balanced in terms of baseline demographics and clinical characteristics (Table 1), as well as procedural features (Table 2).

Primary outcomes

Primary and secondary outcomes in non-weighted treatment groups are reported in Table S3 (Supplementary Material). During the study period, overall annual in hospital mortality in the unweighted cohort declined from 29.4% in 2004 to 24.7% in 2018 (p = .034; Table S4 [Supplementary Material]). In weighted treatment groups, OAR was associated with higher odds of in hospital mortality (odds ratio [OR] 1.76, 95% confidence interval [CI] 1.54—2.01; p < .001) (Table 3). Multivariable logistic regression demonstrated that OAR was strongly associated with increased odds of in hospital death compared with EVAR (OR 2.08, 95% CI 1.76—2.45; p < .001) (Table 4). The presence of haemodynamic instability was also significantly predictive of in hospital mortality, with pre-operative cardiac arrest carrying the highest risk (OR 7.07, 95% CI 5.33—9.38; p < .001). Annual AAA volume was associated with reduced risk of in hospital mortality (third quartile volume OR 0.62, 95% CI 0.41—0.93 [p = .021]; highest quartile volume OR 0.69, 95% CI 0.47—1.01 [p = .053]).

As shown in Fig. 1A, in weighted treatment groups, overall survival including in hospital deaths was 55% (95% CI 53—58) for EVAR *vs.* 46% (95% CI 44—49) for OAR (p < .001) five years after surgery. After multivariable adjustment, Cox proportional hazards assessment demonstrated that OAR was associated with increased overall mortality among all patients (hazard ratio [HR] 1.36, 95% CI 1.23—1.51; p < .001) (Table 5). However, when the analysis was limited to patients who were alive at discharge (Fig. 1B), overall mortality in weighted treatment groups was not significantly different at

Table 2. Procedural details before and after inverse probability weighting for endovascular aneurysm repair (EVAR) *versus* open aneurysm repair (OAR) in patients registered in the Vascular Quality Initiative for treatment of ruptured infrarenal abdominal aortic aneurysms (2004–2018)

Variables	Unweighted statistics			Weighted statistics		
	EVAR (n = 2 389)	OAR (n = 1 868)	Std Diff.	EVAR (n = 2 393)	OAR (n = 1 853)	Std. Diff.
Time from symptoms to repair − h	8.0 (4.0, 24.0)	6.5 (4.0, 18.0)	.089	7.5 (4.0, 21.5)	7.0 (4.0, 20.5)	.053
Time from admission to repair − hours	1.5 (1.0, 3.5)	1.3 (0.8, 3.0)	.072	1.5 (0.9, 3.5)	1.4 (0.8, 3.0)	.053
Pre-operative max. AAA diameter − mm	72.0 (60.0, 85.0)	78.0 (65.0, 90.0)	.22	74.0 (61.0, 90.0)	75.0 (61.0, 90.0)	.036
Iliac aneurysm						
No	1 693 (76.1)	1 412 (78.0)	.044	1 743 (76.7)	1 349 (76.6)	.001
Unilateral	300 (13.5)	189 (10.4)	.094	274 (12.0)	215 (12.2)	.005
Bilateral	231 (10.4)	210 (11.6)	.039	257 (11.3)	196 (11.1)	.005
Hypogastric ligated/occluded						
None	1 260 (88.8)	1 547 (89.4)	.020	1 163 (89.1)	1 516 (89.7)	.017
Single	131 (9.2)	115 (6.6)	.096	116 (8.9)	114 (6.7)	.081
Both	28 (2.0)	68 (3.9)	.12	26 (2.0)	61 (3.6)	.099
Anaesthesia						
Local	299 (12.6)	0 (0)	.54	285 (12.0)	0 (0)	.52
Regional	36 (1.5)	0 (0)	.097	41 (1.7)	0 (0)	.11
General	2 038 (85.9)	1 866 (100)	.57	2 045 (86.3)	1 848 (100)	.56
Total procedure time − min	130.0 (92.0, 180.0)	200.0 (150.0, 268.0)	.75	135.0 (95.0, 185.0)	202.0 (152.0, 270.0)	.73

Data are n (%) or median (Q1, Q3). AAA = abdominal aortic aneurysm; Std Diff. = standard difference.

75% (95% CI 72–78) for EVAR and 75% (95% CI 71–78) for OAR five years after discharge. After multivariable adjustment, Cox proportional hazards assessment demonstrated that survival was similar between treatment groups (HR 0.99, 95% CI 0.79–1.22; p = .90) (Table 6).

Secondary outcomes

Univariable comparison of outcomes in weighted treatment groups showed that OAR was associated with significantly higher odds of MI (OR 1.51, 95% CI 1.25–1.82; p < .001) and respiratory complications (OR 2.08, 95% CI 1.81–2.38;

Table 3. Univariate comparison of outcomes for endovascular aneurysm repair (EVAR; n = 2 389) *versus* open aneurysm repair (n = 1 868), in weighted treatment groups, of patients registered in the Vascular Quality Initiative for treatment of ruptured infrarenal abdominal aortic aneurysms

Outcomes	OR (reference: EVAR)	95% CI	p value
Primary			
In-hospital mortality	1.76	1.54–2.01	<.001
Overall post-discharge mortality, excluding in-hospital deaths − HR	1.32	1.20–1.45	<.001
Overall post-discharge mortality, limited to patients alive at discharge − HR	0.93	0.77–1.12	.44
Secondary			
Hospital LOS (difference in days) − IRR	3.1	SE: 0.5	<.001
ICU LOS (difference in days) − IRR	3.4	SE: 0.3	<.001
MI (troponin or clinical/ECG)	1.51	1.25–1.82	<.001
New onset dysrhythmia	1.76	1.50–2.07	<.001
New onset CHF	1.48	1.15–1.90	.002
Respiratory complications (pneumonia/re-intubation)	2.08	1.81–2.38	<.001
PRBC transfusions − IRR	1.60	1.57–1.64	<.001
Unfavourable discharge (any other than to home)	2.25	1.98–2.56	<.001
Re-interventions − IRR	2.10	1.52–2.89	<.001

Values are reported as odds ratio (OR) unless indicated otherwise. CI = confidence interval; HR = hazard ratio; LOS = length of stay; IRR = incidence rate ratio; ICU = intensive care unit; SE = standard error; MI = myocardial infarction; ECG = electrocardiogram; CHF = congestive heart failure; PRBC = packed red blood cells.

Table 4. Multivariable logistic regression for in-hospital mortality of patients registered in the Vascular Quality Initiative for the treatment of ruptured infrarenal abdominal aortic aneurysms

Parameter	Class level	In-hospital mortality (OR)	95% CI	*p* value
Treatment group (EVAR vs OAR)	EVAR	(Ref.)		
	OAR	2.08	1.76–2.45	<.001
Average number of cases at centre within 1 y	≤15	(Ref.)		
	>15–29	0.90	0.59–1.38	.62
	>29–53	0.62	0.41–0.93	.021
	>53	0.69	0.47–1.01	.053
	Unable to calculate	0.42	0.20–0.89	.024
Year of surgery	2006/2007	0.90	0.09–9.44	.93
	2008	0.70	0.25–2.00	.51
	2009	1.79		
	2010	(Ref.)	0.81–3.93	.15
	2011	0.80	0.44–1.47	.48
	2012	0.97	0.55–1.71	.93
	2013	0.71	0.41–1.23	.22
	2014	0.87	0.51–1.49	.62
	2015	0.94	0.56–1.58	.81
	2016	1.13	0.67–1.92	.64
	2017	1.31	0.77–2.23	.31
	2018	1.12	0.65–1.95	.68
Sex	Male	(Ref.)		
	Female	1.19	0.98–1.44	.08
Age (per increase of 1 y)		1.05	1.04–1.06	<.001
Prior CHF	None	(Ref.)		
	Asymp., hx CHF	1.16	0.84–1.62	.38
	Mild	1.28	0.76–2.15	.36
	Moderate	1.48	0.74–2.93	.27
	Severe	1.68	0.59–4.75	.33
	Unknown	2.60	0.82–0.32	.11
Pre-operative ejection fraction – %	<30	(Ref.)		
	30–50	0.51	0.26–1.00	.051
	>50	0.41	0.22–0.77	.005
	Not done	0.48	0.27–0.88	.017
	Unknown	0.65	0.35–0.18	.15
Prior bypass	No	(Ref.)		
	Yes	1.36	0.80–2.34	.26
		0.04	0.002–0.83	.037
Prior PVI (PTA/stent)	No	(Ref.)		
	Yes	2.17	1.39–3.36	<.001
	Unknown	22.77	1.17–443.66	.039
Smoking	Never	(Ref.)		
	Prior	0.68	0.55–0.85	<.001
	Current	0.76	0.61–0.95	.016
	Unknown	2.40	1.13–5.10	.023
Pre-operative creatinine – per increase of 1 mg/dL		1.12	1.03–1.22	.012
Pre-operative BB	No	(Ref.)		
	Pre-operatively 1–30 d	0.67	0.37–1.22	.19
	Chronic > 30 d	1.03	0.85–1.24	.79
	No, for medical reasons	1.26	0.65–2.42	.49
	Operation day only	0.50	0.31–0.81	.004
	Non-compliant	0.58	0.15–2.17	.42
	Unknown	12.41	2.25–68.39	.004
Pre-operative statin	No	(Ref.)		
	Yes	0.83	0.69–0.99	.041
	No for medical reason	1.13	0.61–2.09	.69
	Non-compliant	1.37	0.59–3.16	.46
	Unknown	0.24	0.04–1.36	.11
Lowest pre-intubation BP < 90 mmHg	≥90	(Ref.)		
	<90	1.96	1.65–2.33	<.001
	Unknown	1.85	1.19–2.88	.007
Mental status	Normal	(Ref.)		
	Disoriented	1.34	1.07–1.68	.011
	Unconscious	1.78	1.36–2.33	<.001
	Unknown	1.47	0.72–2.99	.29

Continued

Table 4-*continued*				
Parameter	**Class level**	**In-hospital mortality (OR)**	**95% CI**	***p* value**
Cardiac arrest	No	(Ref.)		
	Yes	7.07	5.33–9.38	<.001
	Unknown	1.70	0.75–3.85	.20
Pre-operative haemoglobin — per increase of 1 g/dL		0.95	0.92–0.98	.004

OR = odds ratio; CI = confidence interval; EVAR = endovascular aneurysm repair; OAR = open aneurysm repair; Ref. = reference; CHF = congestive heart failure; Asymp. = asymptomatic; hx = history; PVI = peripheral vascular intervention; PTA = percutaneous transluminal angioplasty; BB = beta blocker; BP = blood pressure.

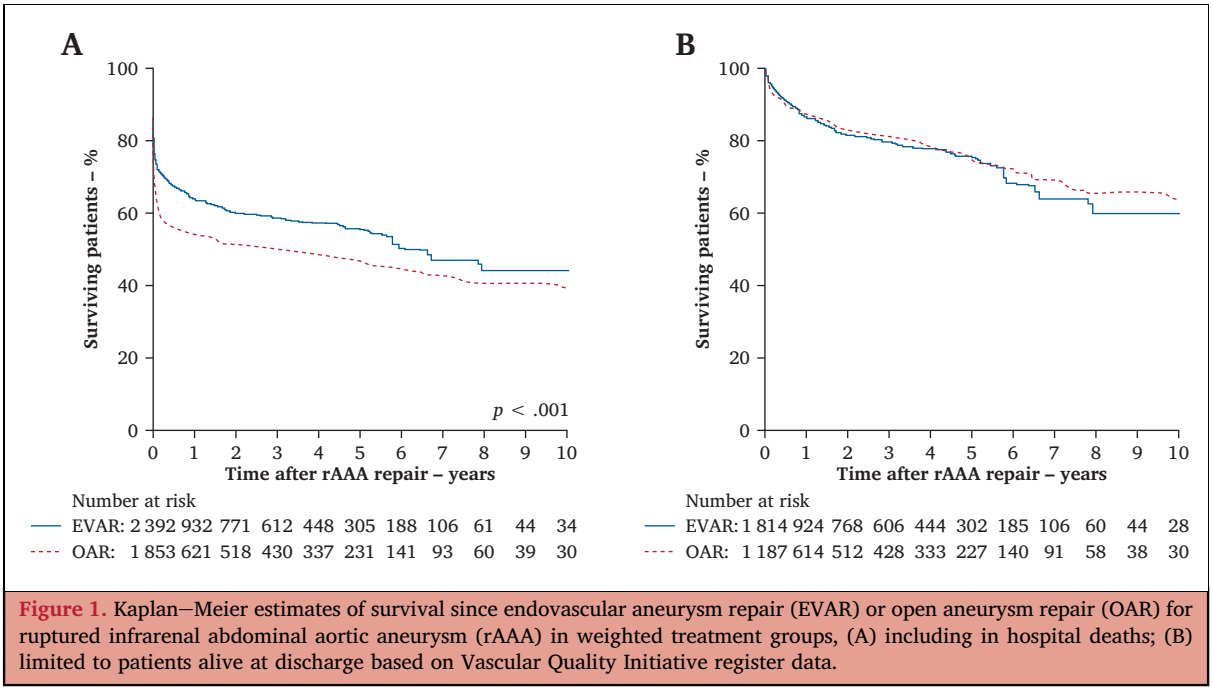

Figure 1. Kaplan–Meier estimates of survival since endovascular aneurysm repair (EVAR) or open aneurysm repair (OAR) for ruptured infrarenal abdominal aortic aneurysm (rAAA) in weighted treatment groups, (A) including in hospital deaths; (B) limited to patients alive at discharge based on Vascular Quality Initiative register data.

$p < .001$), higher rates of blood transfusion (incidence rate ratio [IRR] 1.60, 95% CI 1.57–1.64; $p < .001$), and longer hospital and ICU LOS (IRR 3.1 [$p < .001$] and IRR 3.4 [$p < .001$], respectively). Additionally, OAR showed a significantly higher risk of one year post-discharge re-interventions (IRR 2.10, 95% CI 1.52–2.89 [$p < .001$]; Table 3).

DISCUSSION

AAA rupture remains a dramatically morbid event and vascular surgeons must balance the immediate life saving procedure with the potential for long term efficacy. Current clinical practice guidelines from the European Society for Vascular Surgery strongly recommend EVAR as the first line option for treatment of rAAA when anatomically feasible (Recommendation 74, Class I, Level of Evidence B),[5] mainly owing to the immediate survival advantage. Indeed, the adjusted in hospital mortality from rAAA favoured EVAR with a nearly twofold increased risk of in hospital death with OAR. In the present study, using a large contemporary real world national registry, the use of EVAR has steadily increased over a 15 year period for rAAA repair, which was coupled with a concomitant reduction of the in hospital mortality rate (Fig. 2). Additionally, EVAR was associated with lower rates of peri-operative major adverse events and re-interventions during follow up.

These findings are consistent with previous observational studies and systematic reviews,[6–10] which have demonstrated significant peri-operative benefits to patients undergoing emergency EVAR for rAAA compared with OAR, especially in those with haemodynamic instability and/or higher pre-operative risk scores.[11,12] However, an individual patient meta-analysis based on data from three randomised controlled trials (RCTs) concluded that the use of EVAR does not reduce the acute mortality (0–90 days) from rAAA,[13] and this was confirmed in a subsequent Cochrane review.[14] Why RCTs and "real world" studies appear to demonstrate conflicting results on the potential benefit of EVAR for rAAA may be related to several factors.[15] Firstly, most providers are probably maximising the benefit of EVAR by appropriate patient selection and therefore relegating EVAR to more unstable patients, those presenting to low volume centres without readily available endovascular expertise and devices, or hostile anatomy for EVAR. Alternatively, RCTs may have focused on limited patient subgroups that are not representative of the full

Table 5. Multivariable Cox proportional hazards for overall postdischarge mortality (including in-hospital deaths) of patients registered in the Vascular Quality Initiative for treatment of ruptured infrarenal abdominal aortic aneurysm

Parameter	Class level	Overall mortality (HR)	95% CI	*p*-value
Treatment group (EVAR vs OAR)	EVAR	(Ref.)		
	Open AAA	1.36	1.23–1.51	<.001
Average number of cases at centre within 1 y	≤15	(Ref.)		
	>15–29	0.95	0.72–1.25	.69
	>29–53	0.69	0.53–0.89	.004
	>53	0.76	0.59–0.96	.023
	Unable to calculate	0.57	0.34–0.95	.032
Surgery year	2006	2.66	0.15–47.09	.51
	2007	0.52	0.10–2.74	.44
	2008	0.94	0.56–1.59	.82
	2009	1.34	0.89–2.02	.16
	2010	(Ref.)		
	2011	0.86	0.62–1.19	.36
	2012	0.89	0.65–1.21	.44
	2013	0.72	0.53–0.98	.038
	2014	0.79	0.59–1.07	.13
	2015	0.85	0.63–1.14	.28
	2016	0.97	0.72–1.31	.84
	2017	1.03	0.76–1.40	.85
	2018	1.08	0.78–1.50	.65
Sex	Male	(Ref.)		
	Female	1.17	1.03–1.32	.012
Age (per increase of 1 year)		1.04	1.03–1.05	<.001
Prior CHF	None	(Ref.)		
	Asymp., hx CHF	1.18	0.96–1.45	.11
	Mild	1.15	0.83–1.60	.41
	Moderate	1.35	0.85–2.12	.20
	Severe	0.99	0.48–2.03	.98
	Unknown	0.92	0.51–1.66	.79
Pre-operative ejection fraction − %	<30	(Ref.)		
	30–50	0.69	0.45–1.06	.09
	>50	0.53	0.36–0.80	.002
	Not done	0.64	0.44–0.93	.020
	Unknown	0.71	0.48–1.04	.08
Prior bypass	No	(Ref.)		
	Yes	1.44	1.01–2.05	.043
Prior PVI (PTA/stent)	No	(Ref.)		
	Yes	1.30	0.98–1.73	.07
	Unknown	1.93	0.43–8.57	.39
Smoking	Never	(Ref.)		
	Prior	0.78	0.67–0.89	<.001
	Current	0.85	0.74–0.98	.027
	Unknown	1.05	0.70–1.55	.83
Preop creatinine − per increase of 1 mg/dL		1.03	0.99–1.09	.18
Pre-operative BB	No	(Ref.)		
	Pre-operative 1–30 d	0.94	0.66–1.35	.75
	Chronic > 30 d	1.06	0.93–1.20	.38
	No, for medical reason	1.63	1.09–2.41	.016
	Operation day only	0.67	0.49–0.91	.011
	Non-compliant	1.18	0.50–2.80	.71
	Unknown	2.35	1.05–5.25	.037
Pre-operative statin	No	(Ref.)		
	Yes	0.90	0.80–1.01	.07
	No for medical reason	0.93	0.62–1.40	.74
	Non-compliant	1.15	0.63–2.10	.65
	Unknown	0.88	0.40–1.95	.76
Lowest pre-intubation BP < 90 mmHg	<90	1.36	1.22–1.53	<.001
	≥90	(Ref.)		
	Unknown	1.36	1.03–1.79	.031
Mental status	Normal	(Ref.)		
	Disoriented	1.23	1.06–1.42	.005
	Unconscious	1.32	1.11–1.55	.001
	Unknown	1.28	0.79–2.07	.32

Continued

Table 5-*continued*				
Parameter	**Class level**	**Overall mortality (HR)**	**95% CI**	***p*-value**
Cardiac arrest	No	(Ref.)		
	Yes	3.12	2.67−3.65	<.001
	Unknown	1.39	0.82−2.35	.22
Pre-operative haemoglobin − per increase of 1 g/dL		0.96	0.94−0.98	<.001

HR = hazard ratio; CI = confidence interval; EVAR = endovascular aneurysm repair; OAR = open aneurysm repair; Ref. = reference; CHF = congestive heart failure; Asymp. = asymptomatic; hx = history; PVI = peripheral vascular intervention; PTA = percutaneous transluminal angioplasty; BB = beta blocker; BP = blood pressure.

range of patients that vascular surgeons treat in clinical practice, thus limiting the external validity of findings to real world practice. Furthermore, OAR for rAAA repair probably results in cumulative adverse peri-operative events, while it is reasonable to assume that the less physiological stress and the faster recovery associated with EVAR are all major factors contributing to better early outcomes.

In the present study, a net reduction in hospital and ICU LOS, as well as decreased use of blood transfusions, have been demonstrated to be associated with EVAR for rAAA. These findings resemble those of a recent study, which demonstrated a significant reduction in the frequency of prolonged ICU LOS after both elective and non-elective EVAR procedures in the modern endovascular era.[16] The clinical importance of transfusions in patients undergoing major vascular surgery is also well documented and still debated, given that many patients have significant underlying cardiovascular morbidity.[17] Whether increased blood loss and more transfusions are causally associated with increased cardiovascular morbidity or are simply a marker of increased cardiovascular risk is unclear.[18,19] Nevertheless, they remain important considerations, and even more so under urgent circumstances and an EVAR-first policy for rAAA (i.e., always EVAR when anatomically suitable) might also result in improved healthcare resource use.

The likely shortcomings of most available series are the lack of long term follow up, as well as the presence of significant confounders to the analysis. Using robust IPW methodology to account for selection bias, long term post-discharge survival after emergency rAAA repair in the present cohort remained significantly higher during follow up for EVAR compared with OAR. This is in contrast to elective AAA repair where survival after two − four years is similar, with a net loss of the initial survival advantage from EVAR.[20,21] Nevertheless, the long term findings compare favourably with the three year results of the IMPROVE (Immediate Management of the Patient With Rupture: Open *vs.* Endovascular Repair) trial where, despite the absence of significant differences between EVAR and OAR in the emergency phase, there were other secondary advantages to EVAR (including shortening the length of hospitalisation and greater likelihood of favourable discharge disposition), which at three years had transformed into a true survival benefit.[22] This confirms previous observations that more patients are able to be discharged home after EVAR and suggests that emergency EVAR may confer significant benefits well beyond the index hospitalisation for rAAA repair.[23] Although in the current analysis EVAR did not seem to confer an independent survival benefit in the long run, the early survival gain after EVAR was maintained consistently over time. The cause for this discrepancy is unclear, but some explanations might be offered. Surviving a rAAA may be a physiological stress test, so those surviving are the healthiest and may have the best long term survival.[24] By contrast, elective EVAR may have a mix of less healthy patients that survive the procedure but have higher mid term mortality to match those of OAR in the long term. Furthermore, it is acknowledged that the risk of late death after initial rAAA repair is mainly driven by the underlying medical comorbidities and the initial clinical presentation rather than the repair modality itself.[25,26] However, given the immediate threat to life posed by rAAA, even non-inferiority of long term survival after EVAR would make a valid argument in favour of one treatment over the other.

A recent report from the IMPROVE trial has reported that although the mid term re-intervention rate after emergency AAA repair (by EVAR or OAR) appears to be twice as high as after elective AAA repair, severe complications were more common after OAR.[27] This further suggests that the advantages associated with an endovascular first strategy extend beyond the peri-operative period. In the present study, it was found that EVAR was associated with a lower one year re-intervention rate after the index hospitalisation *vs.* OAR. This was somewhat unexpected as OAR has traditionally been favoured for the potential for fewer secondary interventions in the long run, and EVAR placed in hostile aortic anatomy would be expected to have increased rates of adverse outcomes and device failure, as the emergency setting may offer placement of a device in marginal anatomy.[28] Indeed, the IMPROVE Trial Investigators have conducted an observational study of the treatment received, and not based on the original randomisation, with the aim of identifying morphological parameters that may influence outcomes, and found an inverse relationship between aneurysm neck length and 30 day mortality.[29] Yet, the opposite effect was found in the present study, where EVAR was associated with significantly lower rates of post-discharge secondary interventions in early follow up. This may be due to the overall paradigm

Table 6. Multivariable Cox proportional hazards for overall postdischarge mortality (limited to patients alive at discharge) of patients registered in the Vascular Quality Initiative for treatment of ruptured infrarenal abdominal aortic aneurysm

Parameter	Class level	Overall mortality after discharge (HR)	95% CI	*p* value
Treatment group (EVAR vs OAR)	EVAR	(Ref.)		
	Open AAA	0.99	0.79–1.22	.90
Average number of cases at centre within 1 year	≤15	(Ref.)		
	>15–29/unable to calculate	0.65	0.33–1.28	.21
	>29–53	0.81	0.44–1.49	.49
	>53	0.77	0.43–1.39	.39
Surgery year	2007	0.18	0.01–3.46	.26
	2008	1.40	0.65–3.00	.39
	2009	1.80	0.94–3.44	.07
	2010	(Ref.)		
	2011	1.15	0.68–1.93	.60
	2012	0.92	0.56–1.51	.74
	2013	0.71	0.42–1.18	.18
	2014	0.71	0.42–1.18	.18
	2015	0.83	0.50–1.38	.47
	2016	0.78	0.46–1.33	.36
	2017	0.37	0.18–0.76	.007
	2018	0.12	0.01–1.10	.06
Sex	Male	(Ref.)		
	Female	1.33	1.04–1.70	.025
Age – per increase of 1 y		1.06	1.05–1.08	<.001
Prior CHF	None	(Ref.)		
	Asymp., hx CHF	1.31	0.91–1.90	.15
	Mild	1.05	0.53–2.06	.89
	Moderate/severe	1.39	0.48–4.01	.55
	Unknown	2.12	0.24–18.97	.50
Pre-operative ejection fraction – %	<30	(Ref.)		
	30–50	1.14	0.40–3.22	.81
	>50	0.85	0.31–2.33	.76
	Not done	0.93	0.36–2.44	.89
	Unknown	0.94	0.36–2.50	.91
Prior bypass	No/unknown	(Ref.)		
	Yes	1.70	0.75–3.86	.20
Prior PVI (PTA/stent)	No/unknown	(Ref.)		
	Yes	0.80	0.41–1.57	0.52
Smoking	Never	(Ref.)		
	Prior	0.91	0.68–1.21	.50
	Current	1.12	0.83–1.53	.46
	Unknown	0.20	0.03–1.52	.12
Pre-operative creatinine – per increase of 1 mg/dL		1.05	0.94–1.18	.38
Pre-operative BB	No	(Ref.)		
	Pre-operative 1–30 d	1.39	0.79–2.44	.25
	Chronic > 30 d	1.16	0.91–1.49	.23
	No, for medical reason	3.01	1.36–6.67	.007
	Operation day only	1.06	0.65–1.73	.82
	Non-compliant	8.38	1.35–52.21	.023
	Unknown	0.66	0.07–6.13	.72
Pre-operative statin	No/unknown	(Ref.)		
	Yes	1.08	0.86–1.35	.53
	No, for medical reason	0.24	0.06–1.04	.06
	Non-compliant	0.33	0.03–3.40	.35
Lowest pre-intubation BP < 90 mmHg	<90	0.85	0.68–1.06	.15
	≥90	(Ref.)		
	Unknown	0.73	0.37–1.42	.35
Mental status	Normal	(Ref.)		
	Disoriented	1.21	0.90–1.62	.21
	Unconscious	1.05	0.67–1.64	.84
	Unknown	1.35	0.31–5.93	.69

Continued

Table 6-*continued*				
Parameter	**Class level**	**Overall mortality after discharge (HR)**	**95% CI**	***p* value**
Cardiac arrest	No	(Ref.)		
	Yes	1.27	0.73–2.21	.39
	Unknown	1.39	0.33–5.86	.65
Pre-operative haemoglobin – per increase of 1 g/dL		0.95	0.91–1.00	.036

HR = hazard ratio; CI = confidence interval; EVAR = endovascular aneurysm repair; OAR = open aneurysm repair; Ref. = reference; AAA = abdominal aortic aneurysm; CHF = congestive heart failure; Asymp. = asymptomatic; hx = history; PVI = peripheral vascular intervention; PTA = percutaneous transluminal angioplasty; BB = beta blocker; BP = blood pressure.

shift towards endovascular techniques during the last decade and less experience with OAR in the emergency setting.[30] However, as short aneurysm necks increase the technical difficulty and complication rates in OAR, and impede conventional EVAR, this may partly explain the benefit of EVAR shown in observational studies but not in RCTs. It is also possible that as VQI tracking for re-interventions is limited to one year with varying follow up by region(s) and centre(s), this may be too early to detect failures needing revisions and this finding may be partly due to a time related bias, which cannot be ascertained. Alternatively, lower re-intervention rates with EVAR may be the result of proper patient selection and increased operator experience. Thus, further work to investigate the causes and types of re-interventions, methods to identify potential "high risk of re-intervention" patients, and more accurate adjustment of results based on baseline aortic anatomy are needed in future studies.

A recurrent finding of the literature is that the annual hospital volume of AAA procedures is a crucial factor to be considered,[31–33] as an inverse relationship with early mortality and morbidity rates seems to exist. In this study, multivariable analysis showed that high annual hospital volume of AAA repairs was associated with a trend towards decreased in hospital mortality. This, combined with the observed benefit from EVAR, would suggest that rAAA would be better centralised to tertiary care centres where protocols, expertise, and equipment for first line EVAR (i.e., EVAR first when feasible) exist but with the possibility of performing OAR if needed. Although it might be argued that relative EVAR and OAR volumes could represent a potential confounder to the analyses, in the present study it was elected to use a pragmatic approach and only include the total AAA volume. In reality, using either EVAR or OAR volumes actually measure the same phenomenon (i.e., a low volume EVAR centre is more likely to be a high volume OAR centre and vice versa), so they are collinear variables. For simplicity, total AAA volume as the metric was selected deliberately. However, broad adoption of a centralised EVAR first approach for the treatment of rAAA requires massive systemic changes, and the question of which patients with rAAA may wait to be transported to a centre that can offer both open and endovascular repair remains unclear. Indeed, despite the increasing evidence that regionalisation of emergency vascular surgery for patients with rAAA improves outcomes,[34] many rAAA repairs are still being performed in low volume centres and/or those offering a primary OAR strategy.[35] Therefore, future work is needed to improve regionalisation and standardisation of acute aortic care.

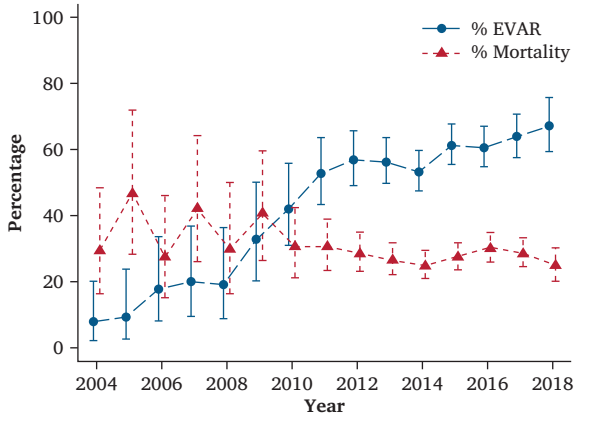

Figure 2. Graphical representation of in hospital mortality (percentage of in hospital deaths/year of study) *vs.* endovascular aneurysm repair (EVAR) use for ruptured infrarenal abdominal aortic aneurysm (percentage of EVAR cases/year of study) during the study period (2004–2018).

Limitations

This study has limitations. Firstly, it was a retrospective analysis of real world data and cannot replace a well designed RCT. However, to the extent possible, comparable groups were created using IPW techniques to adjust for selection bias and multivariable analysis to account for known residual confounders. Owing to limitations with Social Security Death Index data and patient follow up, mortality assessment may be under reported; however, it is not expected that this should differ by group and therefore is unlikely to alter the findings. It is acknowledged that there are no data from patients who died before surgery, and it is also not known if all patients were eligible for EVAR (on/off instructions for use) as certain anatomical data needed to determine this are missing in the open AAA data set. Indeed, neck diameter, length, and angulation are not collected in the open AAA forms for VQI; they are only in the EVAR forms. Also, tracking this information within EVAR forms was initiated around 2014, so it is not uniformly available. Thus, it was decided to leave this information out

of the analysis. The data do not capture how parallel grafting or other advanced endovascular techniques may augment treatment of juxtarenal/pararenal rAAA. However, all patients in both groups, by the VQI inclusion definition, have an infrarenal AAA. Thus, the clamp position in rAAA is not a surrogate for aneurysm extent in this setting, but probably a means of the most effective immediate aortic clamping and haemorrhage control. Lastly, indications for re-interventions in VQI are broad for both EVAR (sac growth, endoleak, migration, occlusion, stenosis, rupture, graft infection) and OAR (incision, graft, intestine, leg ischaemia). However, they may not be able to capture the entire spectrum of possible complications requiring secondary treatment.

CONCLUSION

Within the VQI, EVAR for rAAA repair has been increasingly adopted with favourable short term outcomes in terms of morbidity and mortality *vs.* OAR. Unlike elective AAA repair, survival rates between EVAR and OAR were not observed to converge in long term follow up for patients who survived the index hospitalisation. This suggests that the early significant benefits of EVAR are sustained over time and promotes an endovascular first strategy in anatomically feasible candidates with rAAA.

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CONFLICT OF INTEREST

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