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Previous thoracic aortic repair is not associated with adverse outcomes after thoracic endovascular aortic repair

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Abstract

Background: As many as 20% of patients who have undergone previous thoracic aortic repair will require reintervention, which could entail thoracic endovascular aortic repair (TEVAR). A paucity of data is available on mortality and the incidence of spinal cord ischemia (SCI) and other postoperative complications associated with TEVAR after previous aortic repairs exclusive to the thoracic aorta. The aim of the present study was to assess the effect of previous thoracic aortic repair on the 30-day mortality and SCI outcomes for patients after TEVAR.

Methods: The Society for Vascular Surgery Vascular Quality Initiative database was queried for all cases of TEVAR from 2012 to 2018. Patients were excluded if they had undergone previous abdominal aortic repair, the TEVAR had extended beyond aortic zone 5, or SCI data were missing. The 3 cohorts compared were TEVAR with previous ascending aortic or aortic arch repair (group 1), TEVAR with previous descending thoracic aortic repair (group 2), and TEVAR without previous repair (group 3). The primary outcomes of interest were 30-day mortality and SCI. The secondary outcomes included stroke, myocardial infarction, cardiac complications, respiratory complications, postoperative length of stay, and reintervention. The patient variables were compared using χ^2 tests, analysis of variance, or Kruskal-Wallis tests, as appropriate. Logistic regression analysis was performed to identify the predictors of 30-day mortality and SCI.

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AUTHOR CONTRIBUTIONS

Conception and design: RK, MW, JR, EG, TB, RV

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Results: A total of 4010 patients met the inclusion criteria, with 470 in group 1, 132 in group 2, and 3408 in group 3. The 30-day mortality was 4% (19 of 470) in group 1, 6% (8 of 132) in group 2, and 6% (213 of 3408) in group 3 ($P = .17$). The incidence of SCI was 3% (14 of 470) in group 1, 3% (4 of 132) in group 2, and 3.8% (128 of 3408) in group 3 ($P = .65$). Stroke, reintervention, myocardial infarction, and cardiac complications were not significantly different among the 3 groups. The incidence of respiratory complications was greatest for group 3 (11%; 360 of 3408) compared with groups 1 (9%; 44 of 470) and 2 (4%; 5 of 132; $P = .034$). Similarly, the postoperative length of stay was longest for group 3 (9.6 ± 19.4 days vs 8.2 ± 18.3 days for group 1 and 5.9 ± 8.6 days for group 2; $P = .038$). The independent predictors of 30-day mortality for all TEVAR patients included units of packed red blood cells transfused intraoperatively, urgent or emergent repairs, older age, increasing serum creatinine level, inability to perform self-care, total procedure time, occlusion of the left subclavian artery intraoperatively, distal endograft landing zone 5, and diabetes. The predictors of SCI included the total procedure time, urgent and emergent repairs, and increasing serum creatinine level.

Conclusions: TEVAR after previous thoracic aortic repair was not associated with an increased risk of SCI or 30-day mortality compared with TEVAR without previous aortic repair.

Keywords

Mortality; Spinal cord ischemia; TEVAR

Thoracic endovascular aortic repair (TEVAR) has been increasingly used, and, in many cases, as first-line treatment of severe aortic pathologic entities such as dissections, aneurysms, and traumatic aortic injuries.^{1–4} Because aortic disease can be chronic and progressive, a subset of patients exist who will require TEVAR after previous thoracic aortic repair. As many as 20% of patients who have undergone previous thoracic aortic repair will require reintervention, which could entail TEVAR.^{5–9} Spinal cord ischemia (SCI) is a highly morbid postoperative complication related to TEVAR, occurring at rates of 3% to 15%.^{10–14} TEVAR after previous abdominal aortic repair has been expected to result in an increased risk of SCI.¹⁵ However, a paucity of data is available on mortality and the incidence of SCI and other postoperative complications associated with TEVAR after previous thoracic aortic repair. Our aim was to use the data from the Society for Vascular Surgery Vascular Quality Initiative (VQI) database to investigate the rates of, and risk factors, for 30-day mortality and SCI for patients who had undergone TEVAR with and without previous aortic repairs exclusive to the thoracic aorta.

METHODS

The Society for Vascular Surgery VQI database was queried for all cases of TEVAR from 2012 to 2018 and for aortic pathologic entities, including aneurysms, dissections, traumatic aortic injuries, penetrating aortic ulcers, and intramural hematomas. Data were obtained on patient demographics, comorbidities, aortic pathologic entities, perioperative and operative details, and postoperative outcomes. Patients with pathologic lesions extending beyond aortic zone 5 (Fig 1), those with data missing for postoperative SCI, and patients with previous abdominal aortic repairs were excluded (Fig 2). Three groups were compared: group 1, TEVAR with previous ascending aortic or aortic arch repair; group 2, TEVAR with

previous descending thoracic aortic repair; and group 3, TEVAR without previous repair. The primary outcomes were 30-day mortality and the incidence of postoperative SCI. The secondary end points included reintervention, stroke, myocardial infarction, cardiac complications, respiratory complications, and postoperative length of stay. SCI was defined as all events of transient or permanent lower extremity neurologic deficits. Stroke included any transient ischemic attack or permanent neurologic deficit. Myocardial infarction was defined by troponin elevation, electrocardiographic findings, or clinical assessment findings. Cardiac complications included postoperative congestive heart failure, dysrhythmia, and myocardial infarction. Respiratory complications included pneumonia and reintubation.

The categorical variables were compared using $2 \times 3 \chi^2$ tests and are reported as frequencies and proportions. Continuous variables were analyzed using analysis of variance or Kruskal-Wallis tests, as determined by variable normality, and are presented as the mean $\pm$ standard deviation or median and interquartile range (IQR). Normality was assessed using the Shapiro-Wilk tests and histograms. Univariable analyses were conducted for the primary and secondary outcomes using the same statistical tests to compare all 3 TEVAR groups. Multiple logistic regression models were created to identify the predictors of 30-day mortality and SCI for the entire cohort. Variables with $P < .10$ were considered for inclusion in the multiple logistic regression models. Variable multicollinearity was investigated using Pearson's correlation coefficient and linear regression models. A Pearson correlation coefficient of 0.6 and a variance inflation factor of 2 were used as the threshold values. The results of the multivariable analysis are presented as odds ratios (ORs) and 95% confidence intervals (CIs). All statistics were analyzed using SPSS Statistics, version 24.0 (IBM Corp, Armonk, NY). The present study was exempted from review by the institutional review board of the Medical University of South Carolina because we used de-identified data, and written informed consent from the patients not required. A two-tailed P value of $<.05$ was considered to indicate statistical significance.

RESULTS

A total of 4010 patients were identified who met the selection criteria. Group 1 (TEVAR with previous ascending aortic or aortic arch repair) included 470 patients, group 2 (TEVAR with previous descending thoracic aortic repair) included 132 patients, and group 3 (TEVAR without previous repair) included 3408 patients. TEVAR had been performed for various aortic pathologic entities. The most common were aneurysm repair and dissection (Table I). The overall 30-day mortality after TEVAR for the entire cohort was 6.0% (240 of 4010), and the incidence of SCI was 3.6% (146 of 4010).

Multiple demographic, anatomic, medication-related, and procedural variables differed among the three groups (Table I). Group 3 had the greatest rates of transfers from outside institutions, diabetes, intramural hematoma, penetrating aortic ulcers, trauma, American Society of Anesthesiologists class 5, symptomatic presentation, rupture, urgent and emergent repairs, left subclavian artery (LSCA) coverage, and packed red blood cells (pRBCs) transfused intraoperatively. The primary end points are summarized in Table II. Mortality at 30 days was 4% for group 1 (19 of 470), 6% for group 2 (8 of 132), and 6% for group 3 (213 of 3408; $P = .167$ across all 3 groups). The incidence of SCI was 3% for group

1 (14 of 470), 3% for group 2 (4 of 132), and 3.8% for group 3 (128 of 3408; $P = .652$ across all 3 groups). The incidence of postoperative stroke, reintervention, postoperative myocardial infarction, and cardiac complications was not significantly different among the 3 groups. The incidence of respiratory complications was greater for group 3 (11%; 360 of 3408) and group 1 (9%; 44 of 470) compared with group 2 (4%; 5 of 132; $P = .034$). Similarly, the postoperative length of stay was longer for group 3 (9.6 ± 19.4 days) and group 1 (8.2 ± 18.3 days) compared with group 2 (5.9 ± 8.6 days; $P = .038$). The primary and secondary end points across all three groups are listed in Table III, excluding high-risk cases (ie, trauma, urgent and emergent procedure, and rupture). Most trends observed for the primary and secondary end points were similar, even after exclusion of high-risk cases, with the exception that the postoperative length of stay for group 3 decreased from 9.6 days to a mean of only 6 days, and the length of stay increased for groups 1 and 2. The outcomes after excluding the high-risk patients were as follows: 30-day mortality, 1% for group 1, 5% for group 2, and 3% for group 3 ($P = .06$); SCI, 3% for group 1, 2% for group 2, and 3% for group 3 ($P = .68$); and postoperative length of stay, 6.3 ± 7 for group 1, 9.4 ± 36 for group 2, and 6.0 ± 11 for group 3 ($P = .03$). No statistically significant differences were found for retreatment, stroke, myocardial infarction, cardiac complications, or respiratory complications.

Multivariable analyses were performed to determine whether previous thoracic aortic repairs were independently predictive of poor outcomes after controlling for other baseline characteristics and operative variables. The significant predictors of 30-day mortality after TEVAR were intraoperative transfusion (OR, 1.15; 95% CI, 1.08–1.23; $P < .001$), urgent or emergent repair (OR, 2.22; 95% CI, 1.50–3.30; $P < .001$), increasing age (OR, 1.02; 95% CI, 1.01–1.04), preoperative serum creatinine level (OR, 1.34; 95% CI, 1.14–1.57; $P < .001$), independent functional status with the ability to perform self-care (OR, 0.28; 95% CI, 0.14–0.57; $P < .001$), total procedure time (OR, 1.17; 95% CI, .07–1.28; $P = .001$), occlusion of the LSCA intraoperatively (OR, 1.80; 95% CI, 1.23–2.65; $P = .003$), distal landing aortic zone 5 (OR, 3.50; 95% CI, 1.21–10.12; $P = .021$), and diabetes (OR, 1.56; 95% CI, 1.02–2.39; $P = .040$; Table IV). The independent predictors of SCI were the total procedure time (OR, 1.19; 95% CI, 1.08–1.32; $P = .001$), urgent or emergent repair (OR, 1.65; 95% CI, 1.09–2.49; $P = .017$), and increasing preoperative serum creatinine level (OR, 1.22; 95% CI, 1.02–1.46; $P = .033$; Table V).

The association of LSCA status with 30-day mortality, SCI, rupture, urgent or emergent cases, and trauma cases is presented in Table VI. Thirty-day mortality was associated with LSCA occlusion intraoperatively (10% vs 6%; $P = .002$) and nonpatent and nonrevascularized LSCA (9% vs 6%; $P = .04$). Aortic rupture, urgent or emergent repair, and trauma had all had greater rates of LSCA occlusion intraoperatively and nonrevascularization of the LSCA. LSCA status and the associated 30-day mortality and SCI is shown in Table VII, with the exclusion of high-risk patients. No statistically significant differences in 30-day mortality and SCI when stratified by LSCA status were found when the high-risk patients had been excluded.

DISCUSSION

With the high rate of reintervention among a subset of patients who have undergone previous thoracic aortic repair and TEVAR frequently used as first-line treatment of most thoracic aortic pathologic entities, describing the perioperative outcomes and risks with TEVAR after previous thoracic aortic repair has become imperative for the contemporary management of aortic pathologic entities.^{1,2} In the present patient cohort, the mortality after TEVAR without previous repair was comparable to the reported rates. In addition, this rate did not differ for those who had undergone TEVAR after previous aortic repair. TEVAR after open ascending aortic and aortic arch repair was previously associated with 30-day or in-hospital mortality of 4% to 9%, which was also consistent with the rates for these patients in the VQI database.^{16–19} The group without previous thoracic aortic repair contained most of the patients with trauma-related aortic injuries, ruptures, and symptomatic repairs. Such patients have a high risk of perioperative mortality owing to the nature of the aortic injury and concomitant injuries. Thus, the heterogeneity among the study groups could have masked differences in mortality between those with and without previous repair. Removing the high-risk patients (ie, cases of trauma, rupture, urgent and emergent repair) did not change the primary and secondary outcomes across the 3 groups. These data suggest that perioperative mortality is not associated with a history of thoracic aortic repair.

These VQI data provided similar risk factors for mortality after TEVAR, including older age, increasing serum creatinine level, pRBCs transfused intraoperatively, and urgent and emergent repair. Additionally, the VQI data demonstrated that an inability to perform self-care, increasing total procedure time, coverage of the LSCA intraoperatively, landing the endograft in distal aortic zone 5, and diabetes were predictors of 30-day mortality. A complete frailty assessment was not conducted in the present study. However, the VQI data suggested that an inability to perform self-care was predictive of mortality. Schechter et al²⁰ were not able to demonstrate an association with total psoas muscle volume and mortality related to TEVAR. However, using the National Surgical Quality Improvement Program database, Harris et al²¹ showed that a frailty-based risk score that included with age, gender, functional status, chronic obstructive pulmonary disease, aortic zone 2 involvement, and access exposure was able to accurately predict for 30-day major adverse events, including mortality. More studies on the effect of frailty on postoperative mortality after TEVAR might be necessary.

The association of occlusion of the LSCA intraoperatively and distal landing zone 5 with mortality could have been related to the extent of aortic disease. Presumably, patients with TEVAR landing in zone 5 had had more extensive aortic disease than those with more proximal distal landing zones. Similarly, coverage of the LSCA was likely related to extensive proximal aortic disease. Reported data have shown that coverage of the LSCA is not associated with increased mortality.^{22–24} However, in a large review, LSCA coverage was associated with an increased risk of stroke, which might play a role in the observed increased mortality associated with LSCA occlusion, because stroke is a known risk factor for postoperative mortality.²⁵ However, the data are conflicting. Teixeira et al,²⁶ using VQI data from 2011 to 2014, showed that LSCA coverage was not predictive of stroke after adjustment for confounding variables. It is also conceivable that those who had undergone

urgent and emergent TEVAR were more likely to have had their LSCA occluded, which could also explain its association with increased 30-day mortality and a potential reason that group 3 had similar mortality rates compared with those who had undergone TEVAR after previous repair. Studying the current VQI data, 15% of patients with rupture had had their LSCA occluded intraoperatively. In contrast, of those without aortic rupture, only 7% had had their LSCA occluded intraoperatively. Additionally, 20% of those who had had their LSCA occluded were trauma cases compared with only 13% of nontrauma cases. The incidence of stroke and transient ischemic attack was significantly greater for those with subclavian occlusion (7% vs 4%). In our study, stroke was not included in the multivariate analysis for 30-day mortality because it was a secondary end point. Regardless, these data suggest that coverage of the LSCA intraoperatively is associated with increased 30-day mortality.

Our assessment of VQI-derived data has shown that previous thoracic aortic repairs are not associated with an increased incidence of SCI. The association of previous abdominal aortic repair with SCI after TEVAR is consistent with the hypothesis that more extensive aortic coverage will compromise circulation to the spinal cord via the artery of Adamkiewicz, the intercostal arteries, and lumbar arteries.^{27,28} No study, to the best of our knowledge, has investigated the effect of previous aortic repairs isolated to the thoracic aorta on the incidence of SCI after TEVAR. The SCI rate was 3% after TEVAR with previous repair and 4% after TEVAR without previous repair, which are within the lower range found in the reported data. One potential explanation for the low observed rates of SCI within the groups with previous repair is that the interval between the previous repair and TEVAR might have allowed for the development of collateralized perfusion to the spinal cord.^{29,30} Significant improvements in SCI outcomes have been described for staged procedures in cases of thoracoabdominal aortic repairs; thus, the same might hold true for repairs isolated to the thoracic aorta.³¹ In addition, multiple studies have reported on the potential utility of spinal drain protocols for preventing SCI. However, our analysis failed to show an association between preoperative spinal drain usage and SCI. The SCI rate without a preoperative spinal drain was 3.5% compared with 3.8% with a spinal drain.^{32,33} In addition, even after the exclusion of trauma cases, ruptures, and urgent/emergent cases, previous thoracic repair did not predispose patients to develop SCI.

The risk factors for SCI reported in studies of TEVAR have included chronic renal insufficiency, peripheral artery disease, older age, aortic coverage length, chronic obstructive pulmonary disease, hypertension, intraoperative hypotension, simultaneous coverage of 2 vascular beds, and a history of abdominal aortic aneurysm (repaired or unrepaired).^{10,11,14,34–36} However, most of these were not replicated in the present contemporary evaluation of TEVAR for isolated thoracic aortic pathologic entities. Limiting the study to repairs in the thoracic aorta and excluding previous abdominal aortic repairs enabled us to identify unique risk factors for SCI pertaining to those had undergone isolated thoracic repairs. Three variables were predictive of SCI after TEVAR: the total procedure time, urgent or emergent repair, and increasing preoperative serum creatinine level. Renal insufficiency can be considered analogous to an increasing preoperative serum creatinine. The total procedure time and urgent or emergent procedures were found to be predictive, not only of SCI, but also of 30-day mortality, highlighting the severity of illness in this subset of

patients. The association between the development of SCI and mortality has been previously reported and, again, supports the acuity of patients with thoracic aortic pathologic entities.³⁷

The present study had several limitations that warrant discussion. The data were acquired from a large national database, which inherently predisposes it to reporting biases, coding errors, and missing data. Some data fields changed variable coding during the period of data collection; thus, some reconciliation was required using our best clinical judgment. For missing data, we attempted to be conservative in our multivariable analyses by excluding variables with >40% missing data points. Some of our end points had low event rates, which might have predisposed our results to a type II error. We chose to include many TEVAR cases at the expense of significant heterogeneity in our groups. We then attempted to account for the heterogeneity in our multivariable regression models. Most variables were recoded dichotomously, with all nonpositive variables defaulting to 0, leading to a loss of ordinal disease severity. We included all thoracic aortic pathologic entities included in the VQI, which provided a less conservative approach to identifying the specific risk factors according to the aortic pathologic type. However, we appropriately defined the pathologic types and included them within our multivariate analyses. Data on the interval between the index TEVAR and any previous repairs was not reported; therefore, it was unclear whether the timing between procedures could have influenced the outcomes. Although it is a known risk factor for SCI, the aortic length covered was not reported consistently from 2012 to 2018 and could not be effectively evaluated in our study. However, the proximal and distal aortic landing zones were used as a surrogate. Specific data on the previous repairs were limited to whether a repair had been “open” or “endovascular.” Data to determine whether the previous repair had been an arch reconstruction, debranching, frozen elephant trunk, fenestrated endograft, or other type of aortic repair were unavailable. Other patient management variables, such as blood pressure augmentation and the use of management protocols, were not available from the VQI, which prohibited an assessment of how these critical care protocols might optimize the outcomes.

CONCLUSIONS

TEVAR after previous thoracic aortic repair was not associated with an increased risk of 30-day mortality or SCI compared with TEVAR as the first intervention. The incidence of postoperative stroke, myocardial infarction, cardiac complications, and retreatment were not affected by previous thoracic aortic repair. The total procedure time, urgent or emergent repair, and increasing preoperative serum creatinine were associated with both increased 30-day mortality and SCI after TEVAR. However, the inability to perform self-care and occlusion of the LSCA might also be associated with greater perioperative mortality after TEVAR. The results from our analysis have shown that TEVAR can be performed safely after previous thoracic aortic repair with perioperative outcomes comparable to those with TEVAR without previous aortic repair.

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ARTICLE HIGHLIGHTS

- **Type of Research:** A retrospective analysis of prospectively collected registry data from the Vascular Quality Initiative
- **Key Findings:** Of 4010 thoracic endovascular aortic repair (TEVAR) cases, the 30-day mortality was 4% for TEVAR with previous ascending aortic or aortic arch repair (group 1), 6% for TEVAR with previous descending thoracic aortic repair (group 2), and 6% for TEVAR without previous repair (group 3; $P = .17$). The incidence of spinal cord ischemia was 3% for group 1, 3% for group 2, and 3.8% for group 3 ($P = .65$).
- **Take Home Message:** TEVAR after previous thoracic aortic repair was not associated with an increased risk of spinal cord ischemia or 30-day mortality compared with TEVAR without previous aortic repair.

Fig 1. Aortic zones described by the Society for Vascular Surgery reporting standards for thoracic endovascular aortic repair (TEVAR). Aortic zones 0 to 5 included. Zone 6 begins at the celiac origin and extends to the superior mesenteric artery.²⁰

Fig 2.

STROBE (strengthening the reporting of observational studies in epidemiology) diagram of study design.³⁸ AAA, Abdominal aortic aneurysm; SCI, spinal cord ischemia; TEVAR, thoracic endovascular aortic repair; VQI, Vascular Quality Initiative.

Table I.
Comparison of results of thoracic endovascular aortic repair (TEVAR) with and without previous aortic repair

Variable	TEVAR with previous ascending aorta or aortic arch repair (n = 470)	TEVAR with previous descending thoracic aortic repair (n = 132)	TEVAR without previous aortic repair (n = 3408)	P value
Categorical				
Male gender	288/470 (61)	81/132 (61)	2055/3408 (60)	.90
African American	103/467 (22)	35/131 (27)	796/3397 (23)	.53
Transfer from hospital or rehabilitation	130/470 (28)	22/132 (17)	1435/3407 (42)	<.001
Lives at home	465/470 (99)	131/132 (99)	3347/3406 (98)	.41
Able to perform self-care	457/469 (97)	129/132 (98)	3314/3394 (98)	.96
Cerebrovascular disease	56/470 (12)	18/132 (14)	298/3394 (9)	.02
Coronary artery disease	69/470 (15)	20/132 (15)	474/3398 (14)	.86
Congestive heart failure	69/470 (15)	13/132 (10)	301/3399 (9)	<.001
COPD	110/470 (23)	39/132 (30)	716/3398 (21)	.04
Diabetes	54/470 (12)	16/132 (12)	538/3398 (14)	.03
Dialysis	10/470 (2)	2/132 (2)	91/3403 (35)	.58
Hypertension	423/470 (90)	119/132 (90)	2694/3399 (80)	<.001
Smoking history	307/469 (66)	87/132 (66)	2150/3381 (64)	.65
Previous cardiac intervention	99/470 (21)	16/132 (12)	421/3398 (12)	<.001
Previous carotid intervention	6/470 (1)	5/132 (4)	53/3398 (2)	.11
Previous bypass	46/464 (10)	9/131 (7)	132/3342 (4)	<.001
Previous peripheral vascular intervention	13/470 (3)	6/132 (5)	100/3398 (3)	.55
Positive stress test	10/470 (2)	4/132 (3)	77/3403 (2)	.82
Ejection fraction >50%	312/374 (83)	76/89 (85)	1721/1985 (87)	.24
Preoperative medication				
Aspirin	292/469 (62)	77/132 (58)	1354/3394 (40)	<.001
P2Y12 antagonist	22/469 (5)	6/132 (5)	208/3393 (6)	.37
Statin	273/469 (58)	77/132 (58)	1398/3392 (41)	<.001
β-Blocker	390/469 (83)	96/132 (73)	2091/3393 (62)	<.001
ACE-I/ARB	185/469 (39)	50/132 (38)	1272/3392 (38)	.72
Anticoagulation	100/469 (21)	16/132 (12)	299/3392 (9)	<.001
Indication for procedure				<.001

Variable	TEVAR with previous ascending aorta or aortic arch repair (n = 470)	TEVAR with previous descending thoracic aortic repair (n = 132)	TEVAR without previous aortic repair (n = 3408)	P value
Aneurysm	263/470 (56)	84/132 (64)	1162/3404 (34)	
Dissection	267/470 (57)	45/132 (34)	1362/3404 (40)	
Trauma	1/470 (0.2)	2/132 (2)	529/3404 (16)	
Penetrating ulcer	23/470 (5)	11/132 (8)	400/3404 (12)	
Intramural hematoma	14/470 (3)	2/132 (2)	230/3404 (7)	
ASA class				<.001
1	0/470 (0)	2/132 (2)	21/3401 (1)	
2	13/470 (3)	9/132 (7)	78/3401 (2)	
3	182/470 (39)	41/132 (31)	1116/3401 (33)	
4	263/470 (56)	78/132 (59)	1959/3401 (58)	
5	12/470 (3)	2/132 (2)	227/3401 (7)	
Previous type of repair				<.001
Open surgery	402/467 (86)	62/132 (47)	0/3408 (0)	
TEVAR	65/467 (14)	70/132 (53)	0/3408 (0)	
None	0/467	0/132	3408/3408 (100)	
Symptomatic indication	205/470 (44)	45/132 (34)	1889/3397 (56)	<.001
Rupture	20/470 (4)	4/132 (3)	328/3397 (10)	<.001
Urgent/emergent presentation	129/470 (27)	28/132 (21)	1637/3408 (48)	<.001
General anesthesia	458/470 (97)	125/132 (95)	3301/3404 (97)	.26
Endoleak				
Type I	15/261 (6)	5/84 (6)	37/1193 (3)	.07
Type II	13/261 (5)	1/84 (1)	38/1193 (3)	.18
Type III	0/261	0/84	2/1193 (0.2)	.75
Proximal zone covered				<.001
0	65/470 (14)	6/132 (5)	101/3408 (3)	
1	40/470 (9)	7/31 (5)	175/3408 (5)	
2	157/470 (33)	31/132 (24)	1292/3408 (38)	
3	157/470 (33)	44/132 (33)	1374/3408 (40)	
4	45/470 (10)	34/132 (26)	401/3408 (12)	
5	6/470 (1)	10/132 (8)	65/3408 (2)	
Distal landing zone				<.001

Variable	TEVAR with previous ascending aorta or aortic arch repair (n = 470)	TEVAR with previous descending thoracic aortic repair (n = 132)	TEVAR without previous aortic repair (n = 3408)	P value
3	11/470 (2)	8/132 (6)	163/2060 (5)	
4	88/470 (19)	36/132 (27)	1185/2060 (35)	
5	371/470 (79)	88/132 (67)	2060/3408 (60)	
LSCA occluded				
Preoperatively	32/265 (12)	6/66 (9)	73/1630 (5)	<.001
Intraoperatively	73/425 (17)	9/100 (9)	561/2979 (19)	.04
LSCA patent postoperatively or revascularized intraoperatively	170/267 (64)	40/66 (61)	935/1645 (57)	.100
Preoperative spinal drain	261/470 (56)	73/132 (55)	1260/3405 (37)	<.001
No blood transfusions intraoperatively	357/464 (77)	115/132 (87)	2669/3398 (79)	.04
Continuous				
Age, years	65.1 ± 13	64.4 ± 14	62.6 ± 16	.02
Body mass index, kg/m ²	28.2 ± 8	27.6 ± 6	28.7 ± 7	.10
Preoperative hemoglobin, g/dL	11.6 ± 2	12.5 ± 2	11.9 ± 2	.21
Preoperative creatinine, mg/dL	1.1 ± 1	1.2 ± 1	1.2 ± 1	.28
Estimated blood loss, mL	100 [250]	110 [250]	100 [150]	<.001
Crystalloid intraoperatively, L	2.0 ± 1.3	1.8 ± 1.1	1.7 ± 1.2	<.001
Contrast volume used, mL	110 ± 70	95 ± 60	105 ± 70	.02
pRBCs transfused intraoperatively for patients transfused, U	3.23 ± 4	2.75 ± 2	4.62 ± 19	.68
Total procedure time, hours	2.78 ± 1.8	2.62 ± 1.7	2.24 ± 1.6	<.001

ACE-I, Angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; ASA, American Society of Anesthesiologists; COPD, chronic obstructive pulmonary disease; LSCA, left subclavian artery; pRBCs, packed red blood cell.

Data are presented as n/N (%), median [interquartile range], or mean ± standard deviation; categorical variables were analyzed using $2 \times 3 \chi^2$ tests and continuous variables using analysis of variance or Kruskal-Wallis tests after normality had been assessed.

Table II.

Primary and secondary end points^a stratified by thoracic endovascular aortic repair (TEVAR) with and without previous aortic repair

Variable	TEVAR with previous ascending aorta or arch repair	TEVAR with previous descending thoracic aortic repair	TEVAR without previous aortic repair	P value
Categorical				
30-Day mortality ^b	19/470 (4)	8/132 (6)	213/3408 (6)	.17
SCI ^b	14/470 (3)	4/132 (3)	128/3408 (4)	.65
Postoperative retreatment	59/469 (13)	8/132 (6)	417/3401 (12)	.10
Postoperative stroke ^c	17/470 (4)	3/132 (2)	142/3407 (4)	.49
Postoperative myocardial infarction ^d	5/470 (1)	2/132 (2)	81/3408 (2)	.16
Cardiac complications ^e	41/470 (9)	10/132 (8)	363/3408 (11)	.25
Respiratory complications ^f	44/470 (9)	5/132 (4)	360/3408 (11)	.03
Continuous				
Postoperative length of stay, days	8.2 ± 18.3	5.9 ± 8.6	9.6 ± 19.4	.04

SCI: Spinal cord ischemia.

Data are presented as n/N (%) or mean ± standard deviation.

^a All end points were postoperative outcomes.

^b Primary end point.

^c Stroke was defined as any transient ischemic attack or permanent neurologic deficit.

^d Myocardial infarction was diagnosed by troponin elevation, echocardiographic findings, or clinical assessment findings.

^e Cardiac complications included postoperative congestive heart failure, myocardial infarction, and dysrhythmia.

^f Respiratory complications included pneumonia and reintubation.

Table III.

Primary and secondary end points stratified by thoracic endovascular aortic repair (TEVAR) with and without previous aortic repair^a

Variable	TEVAR with previous ascending aorta or aortic arch repair (n = 340)	TEVAR with previous descending thoracic aortic repair (n = 101)	TEVAR without previous aortic repair (n = 1728)	P value
Categorical				
30-Day mortality ^b	4/340 (1)	5/101 (5)	56/1728 (3)	.06
SCI ^b	9/340 (3)	2/101 (2)	56/1728 (3)	.68
Postoperative retreatment	28/340 (8)	8/100 (8)	108/1725 (6)	.35
Postoperative stroke ^c	11/340 (3)	2/101 (2)	56/1727 (3)	.78
Postoperative myocardial infarction ^d	1/340 (0.3)	2/101 (2)	28/1728 (2)	.15
Cardiac complications ^e	21/340 (6)	7/101 (7)	151/1728 (9)	.26
Respiratory complications ^f	25/340 (7)	5/101 (5)	106/1728 (6)	.57
Continuous				
Postoperative length of stay, days	6.3 ± 7	9.4 ± 36	6.0 ± 11	.03

SCI, Spinal cord ischemia.

Data are presented as n/N (%) or mean ± standard deviation.

^aRupture, trauma, and urgent and emergent procedures were excluded; all end points were postoperative outcomes.

^bPrimary end point.

^cStroke was defined as any transient ischemic attack or permanent neurologic deficit.

^dMyocardial infarction was diagnosed by troponin elevation, echocardiographic findings, or clinical assessment findings.

^eCardiac complications included postoperative congestive heart failure, myocardial infarction, and dysrhythmia.

^fRespiratory complications included pneumonia and reintubation.

Table IV.

Multiple logistic regression analysis of predictors of 30-day mortality after thoracic endovascular aortic repair (TEVAR)

Covariate	OR	95% CI	P value
Intraoperative pRBCs transfused, U	1.152	1.078–1.231	<.001
Urgent or emergent repair	2.223	1.498–3.299	<.001
Age, years	1.024	1.011–1.036	<.001
Preoperative serum creatinine, mg/dL	1.339	1.141–1.573	<.001
Able to perform self-care	0.282	0.139–0.574	<.001
Total procedure time, hours	1.170	1.068–1.281	.001
LSCA occluded intraoperatively	1.802	1.228–2.645	.003
Distal landing zones 3	1	1–1	NA
Distal landing zone 4	1.983	0.670–5.865	.22
Distal landing zone 5	3.502	1.211–10.124	.02
Diabetes	1.561	1.021–2.385	.04
Intramural hematoma	0.445	0.185–1.068	.07
Previous carotid intervention	2.250	0.867–5.843	.10
Coronary artery disease	1.430	0.918–2.225	.11
Preoperative b-blocker	0.766	0.536–1.094	.14
Crystalloid used, L	1.093	0.963–1.241	.17
African American	0.785	0.511–1.207	.27
Aneurysm pathology	0.820	0.550–1.223	.33
Patient lives at home	0.649	0.235–1.797	.41
Ruptured aneurysm	1.215	0.731–2.018	.45
Preoperative hemoglobin, g/dL	0.972	0.897–1.052	.48
Smoking (history or current)	0.927	0.657–1.309	.67
Estimated blood loss, mL	1.000	1.000–1.000	.78
Contrast volume used, L	0.919	0.108–7.835	.94

CI, Confidence interval; LSCA, left subclavian artery; NA, not applicable; OR, odds ratio; pRBCs, packed red blood cells.

Table V.

Multiple logistic regression analysis of predictors of spinal cord ischemia after thoracic endovascular aortic repair (TEVAR)

Covariate	OR	95% CI	P value
Total procedure time, hours	1.190	1.077–1.315	.001
Urgent or emergent repair	1.649	1.092–2.489	.02
Preoperative serum creatinine, mg/dL	1.218	1.016–1.461	.03
Hypertension	1.926	0.939–3.954	.07
Ruptured aneurysm	1.706	0.931–3.128	.08
Preoperative aspirin	1.350	0.909–2.004	.14
Coronary artery disease	1.397	0.871–2.241	.17
Penetrating aortic ulcer	0.566	0.251–1.277	.17
Distal landing zones	1	1–1	NA
Distal landing zone 4	0.529	0.192–1.456	.22
Distal landing zone 5	1.305	0.513–3.322	.58
Dissection pathology	1.217	0.800–1.850	.36
Age, years	1.006	0.992–1.022	.40
Crystalloid used intraoperatively, L	1.061	0.912–1.235	.44
Contrast volume used, L	0.810	0.073–9.012	.86
Intraoperative pRBCs transfused, U	0.997	0.968–1.028	.87
Estimated blood loss, mL	1.000	1.000–1.000	.96

CI, Confidence interval; NA, not applicable; OR, odds ratio; pRBCs, packed red blood cells.

Table VI.

Association of left subclavian artery (*LSCA*) status and management with primary end points and high-risk cases

Variable	LSCA occluded intraoperatively		P value
	Yes ^a	No	
30-Day mortality ^b	65/645 (10)	154/2870 (5)	<.001
SCI ^b	30/645 (5)	105/2870 (4)	.24
Rupture	94/642 (15)	211/2863 (7)	<.001
Urgent/emergent repair	405/645 (63)	1200/2870 (42)	<.001
Trauma	129/645 (20)	368/2868 (13)	<.001
	LSCA patent or revascularized		
	Yes ^c	No	
30-Day mortality ^b	74/1151 (6)	74/835 (9)	.04
SCI ^b	41/1151 (4)	39/835 (5)	.22
Rupture	49/1148 (4)	101/832 (12)	<.001
Urgent/emergent repair	412/1151 (36)	451/835 (54)	<.001
Trauma	85/1151 (7)	137/835 (16)	<.001

SCI, Spinal cord ischemia.

Data are presented as n/N (%).

^aIncluded all cases in which the LSCA was covered, coiled, or surgically occluded during thoracic endovascular aortic repair (TEVAR).

^bIndicates a primary end point.

^cIncluded all cases in which the LSCA was patent at the completion of the procedure, either by avoiding coverage or coverage and revascularization.

Table VII.

Association of left subclavian artery (LSCA) status and management with primary end points, excluding high-risk patients^a

Variable	LSCA occluded intraoperatively		P value
	Yes ^b	No	
30-Day mortality	31/870 (3.6)	10/232 (4.3)	.59
SCI	28/870 (3.2)	6/232 (2.6)	.62
LSCA patent or revascularized			
	Yes ^c	No	
30-Day mortality	14/376 (3.7)	27/699 (3.7)	1.00
SCI	13/376 (3.5)	21/726 (2.9)	.61

SCI, Spinal cord ischemia.

Data are presented as n/N (%).

^aHigh-risk patients included cases of trauma, rupture, and urgent and emergent procedures.

^bIncluded all cases in which the LSCA was covered, coiled, or surgically occluded during thoracic endovascular aortic repair (TEVAR).

^cIncluded all cases in which the LSCA was patent at the completion of the procedure, either by avoiding coverage or coverage and revascularization.