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(thromboembolism, fasciotomies, amputation, and surgical management of cannula bleeding) was required in 30 patients (41.7%). Same-side arterial and venous cannulas, cannula size, and absence of distal perfusion cannula did not increase risk of vascular complication. Multivariate analysis identified age (odds ratio, 0.948; 95% confidence interval, 0.909-0.988; $P = .0116$) and pre-existing peripheral arterial disease (odds ratio, 3.489; 95% confidence interval, 1.146-10.624; $P = .0278$) as independent predictors of need for vascular surgery interventions. The mortality rate of patients who developed vascular complications was not significantly different compared with the mortality rate of those who did not develop vascular complications (61% vs 64%; $P = .92$).

Conclusions: This study represents the largest series to date of lower extremity vascular outcomes in patients undergoing VA-ECMO. Our results confirm the high associated mortality rate; however, vascular complications are not a significant predictor of mortality as previously reported. Same-sided VA-ECMO cannulas, cannula size, and the presence or absence of distal perfusion cannula do not predict vascular complications. Increasing age and presence of peripheral arterial disease are independent predictors of need for vascular surgery intervention in patients on VA-ECMO.

Author disclosures: **C. Siems:** Nothing to Disclose; **R. Valentine:** Nothing to Disclose; **J. Duke:** Nothing to Disclose; **M. Brunsvold:** Nothing to Disclose; **A. B. Reed:** Nothing to Disclose.

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Re-vascularization Of Left Subclavian Artery May Not Influence The Incidence Of Spinal Cord Injury After Endovascular Repair Of Acute Type B Aortic Dissection

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Objective: The objective of this study was to analyze whether left subclavian artery (LSA) revascularization in patients undergoing thoracic endovascular aortic repair (TEVAR) for acute type B aortic dissection (TBAD) is associated with decreased spinal cord ischemia (SCI).

Methods: The national Vascular Quality Initiative TEVAR module was queried for all procedures performed between 2014 and 2021. Patients presenting with aortic aneurysms or aortic ruptures were excluded from the analysis. Patients with therapeutic spinal drains were excluded as well. Patients were divided into two groups according to whether their left subclavian artery (LSA) was revascularized (prior to or during TEVAR) or not. A propensity score matching approach was used to account for possible confounders and evaluate the effect of LSA revascularization on the primary outcome of SCI.

Results: Among patients who had TEVAR for acute TBAD, 852 patients had the LSA covered. The LSA was revascularized prior to or concomitant with TEVAR in 44% of these patients ($n = 378$). The incidence of LSA revascularization significantly increased over the study period (Fig) ($P < .001$). A total of 650

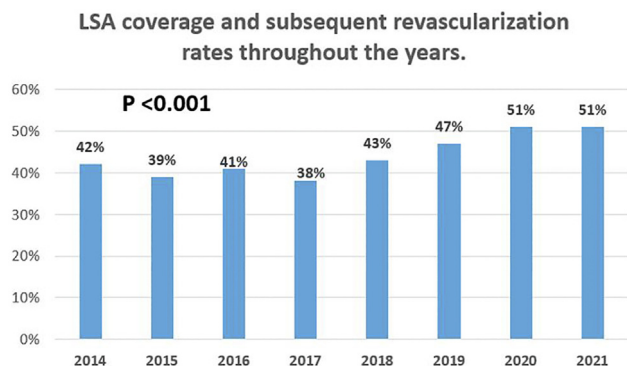


Fig. Left subclavian artery (LSA) revascularization rates following LSA coverage by thoracic endovascular aortic repair (TEVAR).

Table. Comparison of baseline characteristics, comorbidities, intraoperative variables, and postoperative outcomes between patients who had left subclavian artery (LSA) revascularization and those who did not

	Overall (N = 650)	LSA non-revascularized (n = 325)	LSA revascularized (n = 325)	P value
Baseline characteristics				
Age, years	57 (13)	57 (14)	58 (12)	.45
Gender		.23		
Male	458 (71)	236 (73)	222 (68)	
Female	192 (30)	89 (27)	103 (32)	
Race		.62		
African American	235 (36)	111 (34)	124 (38)	
White	318 (49)	167 (51)	151 (47)	
Others	97 (15)	47 (15)	50 (15)	
BMI, kg/m ²	30 (7)	31 (7)	30 (7)	.26
Smoking status		.31		
Never	278 (43)	139 (43)	139 (43)	
Prior	126 (19)	70 (22)	56 (17)	
Current	246 (38)	116 (36)	130 (40)	
Medical history				
CVD	42 (7)	25 (8)	17 (5)	.20
CAD	67 (10)	32 (10)	35 (11)	.70
CHF	52 (8)	27 (8)	25 (8)	.77
COPD	105 (16)	58 (18)	47 (15)	.24
Hypertension	553 (87)	272 (85)	281 (87)	.21
Diabetes	69 (11)	35 (11)	34 (11)	.90
Prior aortic surgery	72 (11)	40 (12)	32 (10)	.32
Prior aneurysm repair	64 (10)	36 (11)	28 (9)	.29
Intraoperative variables				
Contrast volume, mL	116 (66)	121 (74)	111 (58)	.19
EBL, mL	194 (444)	149 (194)	237 (592)	<.01
Fluoro time, minutes	19 (15)	19 (17)	19 (12)	<.01
Procedure time, minutes	162 (99)	138 (87)	186 (104)	<.01
Spinal drain placement		.10		
None	308 (47)	166 (51)	142 (44)	
Preoperative	323 (50)	148 (46)	175 (54)	
Postoperative - prophylactic	19 (3)	11 (3)	8 (3)	
Outcomes				
SCI	26 (4)	15 (5)	11 (3)	.42
Cerebral stroke	46 (7)	30 (9)	16 (5)	.03
30-day mortality	32 (5)	17 (5)	15 (5)	.72
1-year mortality	57 (9)	30 (9)	27 (8)	.68

CHF, Congestive heart failure; COPD, chronic obstructive pulmonary disease; CVD, cerebrovascular disease; EBL, estimated blood loss; LE, lower extremity; PCI, percutaneous coronary interventions; SCI, spinal cord ischemia. Data are presented as number (%) or mean (standard deviation).

patients were split equally and matched between the two groups. Average age was 57 years, and 71% (n = 458) were male (Table). Spinal cord ischemia developed in 26 patients (4%), and cerebral stroke in 46 patients (7%). On univariate analysis, patients who had their LSA revascularized were significantly less likely to develop cerebral stroke (5% vs 9%; $P = .03$). However, this association dropped after accounting for preoperative and intraoperative variables ($P = .14$). No significant difference was seen when comparing SCI, 30-day mortality, or 1-year mortality between patients who had LSA revascularization and those who did not (Table). The average follow-up was 24 months (range, 0-99 months). Long-term survival did not differ between the two groups on Kaplan-Meier analysis.

Conclusions: In patients with acute TBAD undergoing TEVAR requiring LSA coverage, an increasing percentage of patients underwent preoperative or concomitant LSA revascularization over the course of the study: 51% in 2021. In this study, LSA revascularization did not affect the incidence of postoperative SCI, cerebral stroke, or short or long-term mortality. LSA revascularization may carry its own morbidity in TEVAR requiring LSA coverage.

Author disclosures: **A. Natour:** Nothing to Disclose; **A. D. Shepard:** Nothing to Disclose; **M. Weaver:** Nothing to Disclose; **A. Peshkepja:** Nothing to Disclose; **T. Nypaver:** Nothing to Disclose; **L. Kabbani:** Nothing to Disclose.

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Incidence And Risk Factors For Interval Aortic Events During Staged Fenestrated-Branched Endovascular Aortic Repair

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Objective: Staged endovascular repair of complex aneurysms with thoracic endovascular aneurysm repair and later fenestrated-branched endovascular repair (FB-EVAR) may decrease the risk of spinal cord injury. However, it lengthens time for final repair with potential for interval aortic events (IAEs). Thus, the objective of this study was to define the risk of IAEs during staged complex EVAR.

Methods: This was a single-center retrospective review of patients undergoing planned staged FB-EVAR from 2013 to 2021. Clinical and procedural details were reviewed. Endpoints were the incidence of IAE, factors associated with IAE (rupture, symptoms, unexplained death), and outcomes in patients with or without IAE. Rupture was: (1) confirmed - imaging or operative findings; (2) likely - based on clinical details/events; or (3) possible - unexplained out of hospital death.

Results: Of 591 FB-EVAR candidates, 142 were planned for staged repairs. Only 120 were planned for completion repair and reviewed (22 excluded where completion was deferred for frailty/complications). The

mean age was 73 ± 6 years, and 51% were female. The incidence of IAE was 13% (16/120). This included confirmed rupture in six, likely rupture in four, symptomatic presentation in four, and early unexplained interval death with possible rupture in two. The median time to IAE was 17 days (range, 2-101 days), and median time to completion repair for non-IAE repairs was 82 days (interquartile range, 30-147 days). Age, sex, and comorbidities were similar between groups. There were no difference in familial aortic disease, genetically triggered aneurysms, aneurysm extent, or presence of chronic dissection. Patients with IAE had significantly larger aneurysm diameters than those without IAE (76.6 vs 66.5 mm; $P \leq .001$). This difference persisted with indexing for body surface area (aortic size index: 3.9 vs 3.5 cm/m²; $P = .04$), and height (aortic height index: 4.5 vs 3.9 cm/m; $P \leq .001$). IAE mortality was 69% (11/16) compared with no perioperative deaths for non-IAE cases.

Conclusions: The incidence of IAE was 13% in patients planned for staged FB-EVAR. This represented a notable morbidity, including rupture, which must be balanced with spinal cord ischemic risk when planning repair. Larger aneurysms, especially when adjusted for body surface area are associated with IAE. These data support minimal time between stages vs single-stage repairs for larger (>7 cm) complex aortic aneurysms.

Author disclosures: **N. C. Cirillo-Penn:** Nothing to Disclose; **E. R. Tenorio:** Nothing to Disclose; **L. C. Cajas:** Nothing to Disclose; **M. D'Oria:** Nothing to Disclose; **B. C. Mendes:** Nothing to Disclose; **G. S. Oderich:** Consulting Fee; Cook Medical, W.L. Gore, Centerline Biomedical, GE Healthcare. Consulting Fee; Consulting fees/grants paid to institution, no personal income. Consulting Fee; Consultant. **R. R. DeMartino:** Nothing to Disclose.

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Genetic Risk Score Can Supplement Current Guidelines To Identify Patients At Elevated Risk For Abdominal Aortic Aneurysms

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Objective: Genetic risk scores (GRS) based on multiple disease-specific risk-associated single nucleotide polymorphism (SNPs) have been shown to be effective tools for stratifying risks of various diseases. We tested associations of GRS of AAA (GRS_{AAA}) and coronary artery disease (GRS_{CAD}) with risk and severity of AAA. We also explored their clinical utility in identifying high-risk subjects for AAA screening by comparing with the current screening guidelines of the United States Preventive Services Task Force.

Methods: Within the population-based United Kingdom Biobank, we identified an incident cohort for AAA (subjects without a diagnosis of AAA at time of recruitment) and followed their diagnosis of AAA. GRS_{AAA} and GRS_{CAD} were calculated based on 24 and 205 previously established risk-associated SNPs for AAA and CAD, respectively. Associations of GRS_{AAA} and GRS_{CAD} with the risk and severity of AAA were

Table. Risk factors for incident abdominal aortic aneurysm (AAA) in the United Kingdom Biobank, white (N = 447,498)

	Incident AAA				Incident severe AAA (case-case)			
	No. subjects (%)				No. subjects (%)			
	No. n = 445,076	Yes, n = 2422	OR (95% CI) ^a	P ^a	No. n = 1614	Yes, n = 808	OR (95% CI) ^a	P ^a
Gender of male	202,342 (45.5)	2002 (82.7)	4.61 (4.15-5.13)	3.24E-173	1267 (78.5)	735 (91)	2.54 (1.94-3.36)	3.53E-11
By age at recruitment, years								
≤55	167063 (37.54)	172 (7.1)	Ref		135 (8.36)	37 (4.58)	Ref	
55-65	191798 (43.09)	1085 (44.8)	4.86 (4.15-5.73)	4.15E-82	742 (45.97)	343 (42.45)	1.45 (0.99-2.18)	0.06
65-75	86215 (19.37)	1165 (48.1)	10.43 (8.91-12.30)	6.44E-179	737 (45.66)	428 (52.97)	1.77 (1.21-2.66)	4.27E-03
Ever smoking	204,581 (46)	1877 (77.5)	2.94 (2.67-3.24)	3.64E-106	1191 (73.8)	686 (84.9)	1.82 (1.45-2.29)	3.22E-07
Body mass index ≥25 kg/m ²	296,649 (66.7)	1924 (79.4)	1.30 (1.18-1.44)	3.17E-07	1258 (77.9)	666 (82.4)	1.12 (0.90-1.41)	0.32
GRS _{AAA} ≥1.5	43,918 (9.9)	390 (16.1)	1.73 (1.54-1.93)	7.71E-22	242 (15)	148 (18.3)	1.25 (0.98-1.57)	0.07
GRS _{CAD} ≥1.5	44,187 (9.9)	332 (13.7)	1.32 (1.17-1.49)	4.67E-06	215 (13.3)	117 (14.5)	1.08 (0.84-1.39)	0.55

CAD, Coronary artery disease; CI, confidence interval; GRS, genetic risk scores; OR, odds ratio.

^aMultivariable analysis and adjusted for genetic background.