

Extended aortic coverage in thoracic aortic endovascular repair is not associated with spinal cord ischemia



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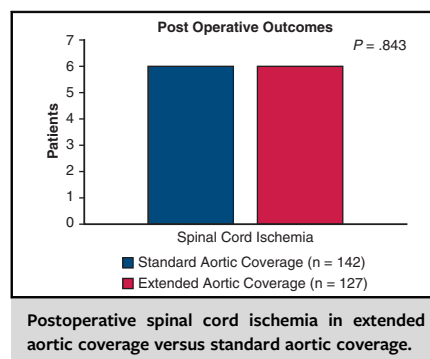
ABSTRACT

Objective: Spinal cord ischemia (SCI) after thoracic endovascular aortic repair (TEVAR) remains a debilitating complication, occurring in 10% of patients. Studies have shown that extended aortic coverage is a risk factor for SCI. This study evaluates whether extended aortic length coverage is a significant risk factor for SCI.

Methods: This study retrospectively reviewed 277 consecutive patients who underwent TEVAR successfully between 2006 and 2021 at a single institution. The patients were classified into 2 groups: ≥ 205 mm and < 205 mm of thoracic aortic coverage. Analysis of variance was used to compare these variables and associated aortic coverage between the 2 groups. Univariable logistical regression was used to compare SCI and associated factors.

Results: Of the 269 patients who underwent successful TEVAR, 127 (47.2%) had ≥ 205 mm and 142 (52.8%) had < 205 mm of aorta coverage. Patients with ≥ 205 mm of thoracic aorta coverage were more likely to be smokers ($P < .01$) and to have a history of previous stroke ($P < .05$). Patients with extended coverage were more likely to receive a preoperative lumbar drain (LD) ($P < .01$). Extended aortic coverage was not associated with a higher risk of SCI compared to standard aortic coverage (4.7% vs 4.2%; $P = .84$). Extended aortic coverage with or without a preoperative LD did not have an association with SCI ($P = .91$). Type II endoleaks were seen more in extended aortic coverage ($P < .01$).

Conclusions: Extended aortic coverage (compared with the standard approach) was not associated with a higher risk of SCI; however, this may have been mitigated by a higher prevalence of prophylactic lumbar drainage in this population. (JTCVS Open 2024;21:366-71)



CENTRAL MESSAGE

Extended aortic coverage (compared with the standard approach) was not associated with a higher risk of spinal cord ischemia; however, this may have been mitigated by this population's higher prevalence of prophylactic lumbar drainage.

PERSPECTIVE

For patients undergoing thoracic endovascular aortic repair, extended aortic coverage can be pursued to treat thoracic aortic pathology and is not associated with spinal cord ischemia. It is however necessary to address preoperative risk factors to minimize risk.

TEVAR has been shown to decrease operative mortality compared with open surgery in patients with complicated type B aortic dissection and thoracic aortic aneurysm.¹ However, certain aortic pathology may necessitate extended

coverage (≥ 205 mm). Extended coverage in type B dissections has shown to eliminate false lumen flow throughout the descending thoracic aorta compared to standard aortic coverage.² This may lead to fewer reinterventions in the

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Abbreviations and Acronyms

CI	= confidence interval
CSF	= cerebrospinal fluid
EVAR	= endovascular aneurysm repair
LD	= lumbar drain
LSA	= left subclavian artery
MAP	= mean arterial pressure
OR	= odds ratio
SCI	= spinal cord ischemia
TEVAR	= thoracic endovascular aortic repair

future, as antegrade or retrograde flow through the false lumen does not occur. The determination of ≥ 205 mm of thoracic aortic coverage was described by Amabile and colleagues,³ who identified the length of aortic coverage as the sole independent predictive factor for SCI after endovascular treatment in both dissections and aneurysms. Other authors, such as Feezor and colleagues,⁴ have suggested that the extent of aortic coverage and location relative to the celiac artery are associated with an increased risk of SCI. Therefore, the current literature is inconclusive on SCI risk and extended aortic coverage.

SCI after TEVAR remains a debilitating complication, occurring in 10% of patients.¹ Established risk factors for SCI include extended length of aortic coverage, previous abdominal aortic surgery, perioperative hypotension, and left subclavian artery (LSA) coverage.⁵ The blood supply to the spinal cord is quite complex and has evolved beyond the artery of Adamkiewicz.^{6,7} This collateral network is an interconnected network of blood vessels between the anterior spinal artery and the blood supply to the adjacent muscles of the back. TEVAR avoids the adverse effects of aortic cross-clamping or associated reperfusion injury to the spinal cord and is associated with a significantly lower rate of paralysis. However, it does permanently exclude many of the segmental arteries that are occluded by a covered thoracic stent. Therefore, delayed paralysis is a more common presentation of SCI after TEVAR compared to immediate paralysis following open repair, which could be transient or permanent depending on patient's collateral blood flow.

Spinal cord protection during TEVAR requires a team approach during the perioperative period, including input from the anesthesiologist and surgeon. To prevent or minimize SCI, implementing widely used prophylactic measures, including LSA revascularization, cerebrospinal fluid (CSF) drainage, and arterial pressure augmentation, as well as identifying both patient and surgical risk factors and applying appropriate mitigation, are important. Surgical risk factors include urgency of the procedure, ≥ 205 cm of thoracic aortic coverage, use of ≥ 3 stents, extended duration of the procedure, and increased blood loss.⁸ Patient risk factors include advanced age (>70 years), perioperative hypotension

(mean arterial pressure [MAP] <70 mm Hg), renal insufficiency, chronic obstructive pulmonary disease, and hypertension. This study evaluated whether extended thoracic aortic length coverage is a significant risk factor for SCI.

METHODS**Patient Population and Study Design**

This observational study retrospectively reviewed 277 patients who underwent successful TEVAR between January 2006 and December 2021 at a single institution. Institutional Review Board approval and need for consent were waived (STUDY18120143). Exclusion criteria included use of frozen elephant trunk, type A aortic dissection, conversion to open procedure, and connective tissue disorder. A total of 269 patients were evaluated for baseline characteristics (diabetes, hypertension, smoking, ischemic heart disease) (Table 1). These patients were stratified to standard aortic coverage (<205 mm) or extended aortic coverage (≥ 205 mm). Definitions and terminology were consistent with the Society of Thoracic Surgeons database.⁹ Postoperative outcomes included excessive blood loss (≥ 1200 mL), endoleak, malperfusion, and subclavian revascularization (Table 2).

One hundred forty-one patients had type B dissection and were further evaluated by chronicity of symptom onset (acute [<14 days] or chronic [>14 days]), previous optimized medical therapy before presentation, and complexity (eg, impending rupture, refractory pain, malperfusion) (Table 3). The other 128 patients had a descending thoracic aneurysm and were further evaluated by rapid growth (≥ 10 mm/year), urgency (within 24–48 hours of presentation) and previous endovascular abdominal aortic aneurysm repair (Table 4). Optimized medical therapy consisted of beta-blockers, diuretics, calcium blockers, angiotensin-converting enzyme inhibitors, and/or alpha-blockers, as described by Estrera and colleagues.^{7,10}

Operative Proceedings

Indications for repair of thoracic aortic aneurysm followed Society for Vascular Surgery guidelines, which recommend endovascular intervention for asymptomatic, degenerative, or traumatic aneurysms exceeding 5.5 cm, as well as all saccular aneurysms due to risk of rupture.⁹ The classification of type B dissection was based on initiation of symptoms, with acute defined as <14 days and chronic/subacute defined as >14 days. In the acute phase, patients were mitigated with anti-impulse medical regimens and pain control in an intensive care setting. Failure to achieve medical optimization and pain control was an indication for repair. The indication for repair of chronic/subacute type B dissection was to prevent aortic false lumen growth and aneurysm degeneration via exclusion.

Whenever possible, preoperative imaging was performed to evaluate size and landing zones and mitigate the use of contrast material. During the study period, patients underwent TEVAR placement with a standard right groin cutdown and percutaneous access on the left side. For elective TEVAR in which coverage of the LSA was necessary for adequate stent graft seal, preoperative or concomitant LSA revascularization was performed.^{9,11} Routine neurocerebral monitoring was performed via somatosensory evoked potentials and electroencephalography. Postoperative neurovascular checks were done to identify delayed presentation of any neurologic deficit. LD placement was selectively done according to such patient risk factors as urgency of the procedure, hypogastric patency, LSA coverage, use of ≥ 3 stents, extended duration of the procedure, perioperative hypotension (MAP <70 mm Hg), previous endovascular aneurysm repair (EVAR), and surgeon preference.¹²

Statistical Analysis

Continuous variables are presented as mean \pm SD for normally distributed data and as median and interquartile range (IQR) for non-normally

TABLE 1. Preoperative baseline characteristics

Variable	Standard coverage	Extended coverage	P value
Age, y, median (IQR)	70.00 (57.00-78.00)	71.00 (62.00-79.00)	.4662
Female sex, n (%)	59 (41.55)	58 (45.67)	.4962
Diabetes mellitus, n (%)	10 (7.04)	17 (13.39)	.0839
Hypertension, n (%)	104 (73.24)	97 (76.38)	.5543
Ischemic heart disease, n (%)	28 (19.72)	20 (15.75)	.3959
Smoking, n (%)	56 (39.44)	74 (58.27)	.0020
Chronic kidney disease, n (%)	12 (8.45)	16 (12.60)	.2661
Previous stroke, n (%)	5 (3.52)	13 (10.24)	.0278
Preoperative lumbar drain, n (%)	28 (19.72)	45 (35.43)	.0038
Previous cardiac surgery, n (%)	49 (34.51)	38 (29.92)	.4222
Aortic diameter, mm, median (IQR)	49.00 (39.00-61.00)	52.50 (42.50-65.50)	.0743

IQR, Interquartile range.

distributed data. Categorical data are summarized using frequency and percentage. The Student *t* test was used to compare normally distributed continuous variables between groups, and the nonparametric Mann-Whitney *U* test was used for non-normally distributed continuous variables. The χ^2 or Fisher exact test was used to compare categorical variables between groups, as appropriate. Univariable logistic regression results are reported as odds ratio (OR) with Wald 95% confidence interval (CI). All tests were 2-sided, with an α level of 0.05 considered to indicate statistical significance. All statistical analyses were performed using SAS/STAT version 15.2 (SAS Institute).

RESULTS

Baseline Characteristics

After exclusion of 8 patients, a total of 269 patients underwent successful TEVAR placement. Of those, 127 (47.2%) had ≥ 205 mm of thoracic aorta coverage, and the other 142 (52.8%) had < 205 mm of coverage. Patients who had greater aortic coverage were more likely to be

smokers (58.2% vs 36.2%; $P < .01$) and to have experienced previous stroke (10.2% vs 3.5%; $P < .05$). However, other preoperative conditions, such as diabetes, previous cardiac surgery, chronic lung disease, age, and sex, were statistically similar between both groups. Patients with ≥ 205 mm of aortic coverage were more likely to present with a rapid growth in aortic diameter (> 10 mm/year) (23.6% vs 9.3%; $P < .01$); however, urgency of TEVAR did not show a significant statistical difference in terms of aortic coverage.

Patients with preoperative placement of an LD were significantly more likely to undergo TEVAR with ≥ 205 mm of coverage (35.4% vs 19.7%; $P < .01$). The extended coverage group ($n = 127$) included 45 patients with a preoperative LD and 82 without a preoperative LD. Among the 45 patients with a preoperative LD, 4 had SCI, and among the 82 patients without an LD, 2 had SCI. These results show no statistical difference in SCI between those with and without a preoperative LD in the extended aortic coverage group ($P = .91$).

Postoperative Outcomes

Twelve patients in the cohort developed postoperative SCI, including 6 dissections and 6 aneurysms. Four of these patients had a preoperative LD. Salvage LDs were successful in resolving neurologic deficits in 35% of the patients. There was no significant difference in the incidence of SCI between the 2 aortic coverage groups (4.7% in the standard coverage group vs 4.2% in the standard coverage group; $P = .84$). Postoperative stroke, mesenteric ischemia, acute renal failure, and acute limb ischemia also were comparable in the 2 groups ($P > .05$). Unsurprisingly, type II endoleaks were more frequent in the extended aortic coverage group compared to standard aortic coverage group (12.6% vs 3.5%; $P < .01$).

Further analysis of univariable logistical regression results showed no association of preoperative LD with SCI

TABLE 2. Postoperative outcomes

Outcomes	Standard coverage, n (%)	Extended coverage, n (%)	P value
LSA revascularization	54 (59.65)	59 (53.35)	.1621
Excessive blood loss	5 (3.52)	5 (3.94)	1.000
Extremity ischemia	4 (2.82)	9 (7.09)	.1031
Spinal cord ischemia	6 (4.23)	6 (4.72)	.8430
Mesenteric ischemia	0 (0.00)	3 (2.36)	.0655
Disabling stroke	1 (0.70)	2 (1.57)	.4973
Endoleak			
Type 1	14 (9.86)	19 (14.96)	.2029
Type 2	5 (3.52)	16 (12.60)	.0056
Type 3	1 (0.70)	4 (3.15)	.1382
Type 4	—	—	—
Type 5	—	—	—

LSA, Left subclavian artery.

TABLE 3. Type B dissections

Parameter	Standard coverage, n (%)	Extended coverage, n (%)	P value
Type B dissection (n = 141)	73 (73.38)	68 (67.62)	.8894
Previous OMT	25 (17.61)	28 (22.05)	.3605
Chronicity			
Acute	39 (17.02)	45 (21.27)	>.05
Chronic	28 (16.31)	29 (15.60)	>.05
Complications (eg, malperfusion, rupture)	51 (35.92)	56 (44.09)	.4188

OMT, Optimized medical treatment.

(OR, 1.985; 95% CI, 0.610-6.454; $P = .255$). Dissections yielded an OR of 0.261 (95% CI, 0.069-0.987; $P = .048$) (Table 5). Extended aortic coverage yielded an OR of 1.124 (95% CI, 0.353-3.577), which was not statistically significant ($P > .05$). Previous EVAR returned an OR of 8.433 (95% CI, 0.804-88.402), with was near statistical significance ($P = .07$). Of note, the sample size was small for this cohort.

DISCUSSION

Numerous studies have debated the repercussions of SCI after TEVAR; however, few have evaluated the implication of extended aortic coverage. The permanent coverage of the thoracic segmental arteries has been identified as a significant risk factor for SCI and has led to judicious placement of stents ≥ 205 mm. This has led to propagation or inadequate coverage of dissection or aneurysm, necessitating a subsequent procedure.¹³ The appropriate aortic coverage is based on each patient’s pathology and risk factors that can be mitigated perioperatively.

Our extended aortic coverage cohort had higher prevalences of smokers ($P = .0067$) and patients with previous stroke ($P = .0381$). An association between smoking and aortic disease has been well documented as far back as Hammond and Horn in 1958.¹⁴ More recently, Lederle and colleagues¹⁵ noted a 6-fold increase in aortic aneurysm–related events. This increase in relative risk did not improve after cessation. Therefore, we hypothesize

TABLE 4. Thoracic aortic aneurysms

Parameter	Standard coverage, n (%)	Extended coverage, n (%)	P value
Descending thoracic aneurysm (n = 128)	67 (66.62)	61 (61.38)	.3552
Urgency	34 (23.94)	45 (35.43)	.0389
Previous EVAR	3 (2.11)	1 (0.79)	.6245
Rapid growth (>10 mm/y)	13 (9.15)	30 (23.62)	.0012

EVAR, Endovascular aneurysm repair.

TABLE 5. Univariable logistic regression for spinal cord ischemia

Effect	OR	95% CI	P value
Extended aortic coverage (≥ 205 mm)	1.124	0.353-3.577	.843
Females	0.925	0.286-2.991	.896
Diabetes mellitus	0.858	0.106-6.972	.886
Hypertension	1.383	0.291-6.579	.684
Ischemic heart disease	1.700	0.434-6.661	.446
Smoking	1.073	0.337-3.414	.906
Chronic kidney disease	0.826	0.102-6.705	.858
Previous stroke	3.013	0.608-14.925	.177
Previous cardiac surgery	0.386	0.083-1.801	.226
Previous EVAR	8.433	0.804-88.402	.075
Left subclavian revascularization	0.442	0.117-1.673	.229
Lumbar drain	1.985	0.610-6.454	.255
Dissection	0.261	0.069-0.987	.048
Previous OMT	0.338	0.042-2.729	.309
Acute dissection	0.729	0.150-3.554	.696
Chronic dissection	0.547	0.066-4.529	.576
Complicated dissection (eg, malperfusion, rupture)	0.994	0.307-3.222	.993
Aneurysm	3.119	0.825-11.783	.094
Urgency*	1.766	0.543-5.743	.344
Rapid growth (>10 mm/y)	0.465	0.059-3.701	.470

OR, Odds ratio; CI, confidence interval; EVAR, endovascular aneurysm repair; OMT, optimized medical treatment. *Urgency is defined as within 24 to 48 hours of presentation.

this cohort was likely at elevated risk for aortic intervention and possibly extended coverage.

In our cohort, extended aortic coverage was not associated with an elevated risk of SCI compared to standard aortic coverage (4.7% vs 4.2%; $P = .84$). Further analysis of extended aortic coverage showed an OR of 1.124 (95% CI, 0.353-3.577; $P = .843$) for SCI. Although we did not observe any difference in terms of SCI, we should consider the possibility that it exists, owing to the more liberal use of lumbar drainage in the population with extended aortic coverage ($P < .0038$). Further investigation of SCI in the extended coverage cohort with and without preoperative LD showed no statistical difference ($P = .91$). A univariable logistical regression model verified no correlation between SCI and LD placement ($P = .255$); therefore, extended coverage and SCI were not confounded by preoperative LDs, because they were not associated on analysis.

For patients in whom extended aortic coverage is warranted, preoperative mitigation in a multidisciplinary approach is of paramount importance. While preoperative LDs in open repairs have been robustly studied by Coselli and colleagues,¹⁶ TEVAR has lacked a randomized control trial. Currently, the European Association for Cardiothoracic Surgeons recommends LDs for prevention of SCI

in the context of TEVAR (class of indication Iia; level of evidence C).^{17,18} In patients at significant risk for SCI, a preoperative LD can improve spinal perfusion in concert with blood pressure augmentation and neurophysiology monitoring.¹⁹ Aucoin and colleagues²⁰ have shown that rescue LDs have increased complications and inferior outcomes to preoperative LDs. Studies have reported rates of LD-associated complications as high as 11%, ranging from headache to subdural hematoma and infection.²¹ Zipfel and colleagues^{3,22} reported that 66% of their TEVAR patients did not require ICU care or required ICU care for <24 hours, which they were able to achieve because of the absence of CSF drains in their patients. This can allow for early mobilization and improved utilization of hospital resources. While most surgeons still would prefer postoperative drain placement in a patient with delayed SCI, reversal of deficits can be achieved only within the first 1 to 2 hours of symptom onset. Therefore, our cohort received selective preoperative LDs to maximize benefits with high-risk patients and simultaneously decrease drain complications. The decision to place a preoperative LD was based on surgeon preference.

Twelve patients developed SCI, including 4 with preoperative LDs and 8 with salvage LDs. Six patients had dissections, including 4 with hyperacute ruptures transferred from a referring hospital complicated by mortality. One patient with dissection presented with a hyperacute malperfusion with preoperative LD and achieved resolution of neurologic deficits. Six patients had aneurysms, including 2 who developed permanent neurologic deficits and 4 whose deficits were resolved with LD placement. Patients with salvage LDs were treated immediately on presentation of deficits; however, only 37% recovered from such deficits.

Of note, all patients with SCI had blood pressure augmentation to achieve a MAP >100 mm Hg. Patients who present with rupture and malperfusion may not recover from SCI even if LDs are used. Decreasing the physiologic CSF pressure by 5 mm Hg is unlikely to optimize spinal cord perfusion pressure, compared to increasing MAP by 20 to 40 mm Hg.²³ While spinal cord perfusion is attenuated with extended aortic coverage, Colman and colleagues²⁴ suggested that perfused nonoccluded segmental arteries can maintain blood flow to the ischemic spinal cord segment by reversing the direction of blood flow within the anterior spinal artery, as well as collateral intraspinal or paraspinal networks. Etz and colleagues^{17,18} described spinal blood flow remodeling within 2 to 5 days after graft deployment in animal models.^{24,25} Therefore, close postoperative neurovascular monitoring is key for preventing SCI, as TEVAR patients may experience delayed effects.²⁶

Our extended aortic coverage group had a higher incidence of type II endoleaks ($P < .006$). This is predictable as more segmental arteries are covered in patients with

extended aortic coverage compared to those with standard aortic coverage. Type II endoleaks are the second most frequent type, accounting for 10% to 25% of all endoleaks.²⁷ Almost all patients had spontaneous resolution on surveillance imaging. Only 1 patient had subsequent embolization of a type II endoleak at follow-up. Daye and Walker²⁸ reported that all-cause mortality and aneurysm-related mortality did not differ between patients with and those without a type II endoleak. While these procedures were not urgent, treatment of type II endoleaks is necessary to avoid pressurization of the aortic sac, which can lead to rupture or malperfusion. All patients with a type I or type III endoleak were repaired immediately intraoperatively after confirmation on aortography.

Limitations

A limitation of the study is its retrospective nature, with its inherent treatment and hospital center-specific bias. The comfort level of surgeons and indications for LDs may have varied and influenced the decision to pursue extended aortic coverage. The small sample size limited our statistical analyses of SCI. The extended aortic coverage group had significantly more smokers than the standard coverage group, making the 2 groups inherently different. It should be acknowledged that our study data are based on electronic medical record review and a single-center database. Patient presentation to hospitals outside of our health system could have delayed care and impacted outcomes.

CONCLUSIONS

Aortic coverage ≥ 205 mm previously has been identified as a significant risk factor for SCI. In our study cohort, extended aortic coverage (compared with the standard approach) was not associated with an elevated risk of SCI; however, this may have been mitigated by this population's higher prevalence of prophylactic lumbar drainage. Therefore, SCI is likely multifactorial.

Conflict of Interest Statement

Dr Sultan receives institutional research support from Abbott, Artivion, Atricure, Edwards, Medtronic, and Terumo Aortic. All other authors reported no conflicts of interest.

The *Journal* policy requires editors and reviewers to disclose conflicts of interest and to decline handling or reviewing manuscripts for which they may have a conflict of interest. The editors and reviewers of this article have no conflicts of interest.

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