Strategies to improve spinal cord ischemia in endovascular thoracic aortic repair: Outcomes of a prospective cerebrospinal fluid drainage protocol

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Purpose: **Although endovascular repair of thoracic aortic aneurysm has been shown to reduce the morbidity and mortality rates, spinal cord ischemia remains a persistent problem. We evaluated our experience with spinal cord protective measures using a standardized cerebrospinal fluid (CSF) drainage protocol in patients undergoing endovascular thoracic aortic repair.**

Methods: **From 2004 to 2006, 121 patients underwent elective (n = 52, 43%) and emergent (n = 69, 57%) endovascular thoracic aortic stent graft placement for thoracic aortic aneurysm (TAA) (n = 94, 78%), symptomatic penetrating ulceration (n = 11, 9%), pseudoaneurysms (n = 5, 4%) and traumatic aortic transactions (n = 11, 9%). In 2005, routine use of a CSF drainage protocol was established to minimize the risks of spinal cord ischemia. The CSF was actively drained to maintain pressures <15 mm Hg and the mean arterial blood pressures were maintained at ≥90 mm Hg. Data was prospectively collected in our vascular registry for elective and emergent endovascular thoracic aortic repair and the patients were divided into 2 groups (+CSF drainage protocol, −CSF drainage protocol). A χ^2 statistical analysis was performed and significance was assumed for $P < .05$.**

Results: **Of the 121 patients with thoracic stent graft placement, the mean age was 72 years, 62 (51%) were male, and 56 (46%) underwent preoperative placement of a CSF drain, while 65 (54%) did not. Both groups had similar comorbidities of coronary artery disease (24 [43%] vs 27 [41%]), hypertension (44 [79%] vs 50 [77%]), chronic obstructive pulmonary disease (18 [32%] vs 22 [34%]), and chronic renal insufficiency (10 [17%] vs 12 [18%]). None of the patients with CSF drainage developed spinal cord ischemia (SCI), and 5 (8%) of the patients without CSF drainage developed SCI within 24 hours of endovascular repair ($P < .05$). All patients with clinical symptoms of SCI had CSF drain placement and augmentation of systemic blood pressures to ≥90 mm Hg, and 60% (3 of 5 patients) demonstrated marked clinical improvement.**

Conclusion: **Perioperative CSF drainage with augmentation of systemic blood pressures may have a beneficial role in reducing the risk of paraplegia in patients undergoing endovascular thoracic aortic stent graft placement. However, selective CSF drainage may offer the same benefit as mandatory drainage. (J Vasc Surg 2008;48:836-40.)**

Thoracic endovascular aneurysm repair (TEVAR) has emerged as a less invasive alternative to the traditional open surgical repair and is generally associated with a significant reduction in morbidity and mortality.[1] Although the exact etiology of spinal cord ischemia (SCI) following TEVAR remains ill defined, most recent studies suggest that TEVAR is associated with a 3-6% incidence of SCI.[2-4] Several factors have been implicated in increasing the risk of SCI; including prior abdominal aortic repair, length of thoracic aortic coverage, hypogastric artery interruption, subclavian artery coverage, emergent repair, and sustained hypotension.[5-7]

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Findings from open surgical repair would suggest that cerebrospinal fluid (CSF) drainage, maintenance of normotension, lumbar reimplantation, and hypothermia may reduce the incidence of spinal cord ischemia.[4,8] Some of these adjuncts are applicable to TEVAR and have been used selectively in high risk patients.[7,9] In our early experience, high-risk patients underwent selective CSF drainage, and even with this strategy some low-risk TEVAR patients developed symptoms of spinal cord ischemia. In light of these findings, we established a protocol that included CSF drainage in all patients undergoing TEVAR and this study reports our findings.

METHODS

From 2004 to 2006, 121 consecutive patients underwent elective (n = 52, 43%) and emergent (n = 69, 57%) TEVAR. The pathologies treated were TAA (n = 94, 78%), symptomatic penetrating ulceration (n = 11, 9%), pseudoaneurysm (n = 5, 4%), and traumatic aortic transactions (n = 11, 9%).

In 2005, a CSF drainage protocol was initiated which included placement of a spinal drain preoperatively on all patients and maintenance of CSF pressures less than 15 mm

Hg. Eleven patients during this period did not receive drains secondary to either hemodynamic instability or technical issues secondary to spinal anatomy and were placed in the −CSF group. Preoperatively, a lumbar drain was placed in the subarachnoid space between approximately L2 and L4. Intraoperatively, the open CSF drainage system with a pressure transducer (Integra, Plainsboro, NJ) was monitored to maintain CSF pressures of less than 15 mm Hg. Once the stent graft was deployed, the mean arterial pressures (MAP) were maintained at ≥90 mm Hg using either intravenous fluid infusions and/or intravenous vasopressors as necessary. Following completion of TEVAR, all patients were kept on bed rest in the intensive care unit. The MAPs were recorded every hour and maintained ≥90 mm Hg for 24 hours by using fluid boluses and intravenous vasopressors. The CSF pressures were recorded every hour; for pressures greater than 10 mm Hg, the stopcock in the drainage system was opened and CSF was allowed to passively drain in the collection bag in 20 mL increments and pressure was re-evaluated. If the patient developed symptoms of spinal cord ischemia, CSF was actively drained and maintained at a pressure of <10 mm Hg. There was no set limit for volume of CSF drainage. After 24 hours, the MAP was allowed to drift below 90 mm Hg and the vasopressors are weaned off. If there were no signs of spinal cord ischemia after 12 hours, the spinal drain was clamped and the patients were mobilized out of bed, then the drain was subsequently removed. If there were any signs or symptoms of SCI during the trial of lowered MAPs or drain clamping, the drain was unclamped and CSF was drawn off to keep the intrathecal pressure of less than 10 mm Hg, and the MAPs are maintained at ≥90 mm Hg using vasopressors as needed.

Patients without spinal drainage that developed SCI underwent emergent placement of a spinal drain and standard CSF drainage protocol was followed. CSF was actively drained to <10 mm Hg and MAPs were elevated to >90 mm Hg.

The patients were divided into two groups, Group 1 (+CSF drainage) and Group 2 (−CSF drainage). Data was prospectively collected in a vascular registry after chart review and analyzed for demographics, operative indications, intra-operative blood loss (EBL), length of aortic coverage, and subclavian artery coverage. The length of thoracic aortic coverage was analyzed based on 1-month post procedure CT angiogram.

Statistical analysis was performed using Minitab 14. The two groups were compared using a χ^2 square analysis, two sample *t* tests, and a Mann-Whitney Test and the *P* values < .05 were considered statistically significant. IRB approval was obtained in accordance with hospital policy prior to study initiation.

RESULTS

Of 121 patients that underwent TEVAR between 2004 and 2006, 56 (46%) had preoperative spinal drain placement (+CSF drainage) and 65 (54%) did not (−CSF drainage).

Table I. Patient demographics

	+CSF drainage	*−CSF drainage*	P-*value*
N	56 (46%)	65 (54%)	NS
Age	73	75	NS
Male	50%	52%	NS
CAD	43%	41%	NS
HTN	79%	77%	NS
COPD	32%	34%	NS
CRI	17%	18%	NS

CSF, Cerebrospinal fluid; *N,* number; *CAD,* coronary artery disease; *HTN,* hypertension; *COPD,* chronic obstructive pulmonary disease; *CRI,* chronic renal insufficiency; *NS,* not significant.

Table II. TEVAR indications

Indication	*+CSF drainage*	*−CSF drainage*	P-*value*
N	56 (46%)	65 (54%)	NS
Asymptomatic TAA	32 (57%)	28 (43%)	NS
Symptomatic TAA	13 (23%)	21 (32%)	NS
Symptomatic ulceration	3 (5%)	8 (12%)	NS
Pseudoaneurysm	2 (4%)	3 (5%)	NS
Traumatic transection	6 (9%)	5 (9%)	NS

N, xxx; *CSF,* cerebrospinal fluid; *TAA,* thoracic aortic aneurysm; *NS,* not significant; *TEVAR,* thoracic endovascular aneurysm repair.

The +CSF drainage and −CSF drainage groups were similar in respect to age, demographics, and comorbidities such as coronary artery disease (CAD), hypertension (HTN), chronic obstructive pulmonary disease (COPD), and chronic renal insufficiency (CRI) (Table I). The indications for TEVAR were also similar between the two groups (Table II).

The +CSF group had a significantly higher percentage of patients with previous abdominal aortic aneurysm (AAA) repair (26, 46% vs 15, 23%; $P < .05$), subclavian artery coverage (22, 39% vs 12, 18%; $P < .05$), the need for vasopressors (36, 64% vs 22, 34%; $P < .05$), and a greater length of aortic coverage (28 cm vs 20 cm; $P < .05$). The −CSF drainage group had significantly greater median EBL (500 mL vs 200 mL; $P < .05$) (Table III). In the +CSF drainage group, one patient (1.8%) had malfunction of the CSF drain, and developed SCI 24 hours following TEVAR. After placement of a new CSF drain and resumption of CSF drainage protocol, he had full recovery. In the −CSF drainage group, 4 patients (6.2%) developed symptoms of SCI and underwent placement of CSF drainage catheters; 2 patients had full recovery and 2 had persistent SCI.

To identify patient risk factors for developing SCI, we took a closer look at all patients that developed any spinal ischemia complications.

Patient 1. A 73-year-old male with prior endovascular AAA repair presented with 5.8 cm thoracic aortic aneurysm (TAA) and underwent TEVAR along with CSF drainage per protocol. The total length of thoracic aortic coverage was 19 cm, and the EBL was 200 mL. Within 24 hours

Table III. Spinal cord risk factors

Spinal cord risk factor	*+CSF drainage*	*−CSF drainage*	P-*value*
Prior AAA repair	26 (46%)	15 (23%)	<.05
Left subclavian artery coverage	22 (39%)	12 (18%)	<.05
Median blood loss (mL)	200 (range, 30-1200)	500 (range, 30-3000)	<.05
Mean aortic coverage (cm)	28 (range, 10-39)	20 (range, 10-32)	<.05
Perioperative vasopressors	36 (64%)	22 (34%)	<.05

CSF, cerebrospinal fluid; *AAA*, abdominal aortic aneurysm.

postoperatively, the CSF drain malfunctioned and was removed having not drained any CSF, several hours later when the patient got out of bed, he developed bilateral lower extremity weakness, and had a MAP of 83 mm Hg. Another CSF drain was placed and MAPs were augmented to 90 mm Hg. Several hours later, the patient had full neurological recovery. The drain was eventually removed 6 days later, and the patient discharged home in postoperative day 9.

Patient 2. An 84-year-old female with prior open surgical AAA repair, presented with hypotension and a ruptured 6.2 cm TAA, and underwent emergent TEVAR without CSF drainage. The total length of thoracic aortic coverage was 22 cm, and the EBL was 1000 mL. Approximately 29 hours postoperatively, the patient developed bilateral lower extremity paralysis; the MAP at the time was 105 mm Hg. A spinal drain was placed and the CSF protocol was instituted; the patient had a full neurological recovery. The drain was removed 4 days later, and the patient was discharged home on postoperative day 8.

Patient 3. An 84-year-old female with prior endovascular AAA repair, presented with hypotension and a ruptured 5.7 cm TAA, and underwent emergent TEVAR without CSF drainage. The total length of thoracic aortic coverage was 18 cm, and the EBL was 1000 mL. Approximately 12 hours postoperatively, the patient developed bilateral lower extremity paralysis; the MAP at the time was 60 mm Hg. A spinal drain was placed and CSF protocol instituted; the patient had no neurologic recovery, remained hypotensive, and underwent exploratory laparotomy and resection of gangrenous bowel. The patient never recovered neurological function and the drain was removed 3 days later. The patient eventually died of multisystem organ failure on postoperative day 38.

Patient 4. A 68-year-old female with significant CAD, and prior open AAA repair, underwent TEVAR for an asymptomatic 5.5 cm TAA. Preoperative attempts of spinal drain placement were unsuccessful. The total length of thoracic aortic coverage was 20 cm, and the EBL was 2000 mL. Approximately 17 hours postoperatively, the patient developed bilateral lower extremity paralysis; the MAP at the time was 71 mm Hg. A spinal drain was placed and CSF protocol instituted; the patient had partial recovery in one leg only. Four days later, the patient had a massive myocardial infarction, and sustained anoxic brain injury. Ventilatory support was subsequently withdrawn and he died on postoperative day 9.

Patient 5. An 84-year-old female underwent TEVAR for an asymptomatic 6.0 cm TAA. The total length of thoracic aortic coverage was 15 cm, and the EBL was 300 mL. Unlike in all other patients with SCI, she had no history of a prior AAA. Approximately 7 hours postoperatively, the patient experienced right leg weakness; the MAP at the time was 80 mm Hg. A spinal drain was placed and CSF protocol instituted; the patient had full neurological recovery. The drain was removed 4 days later and the patient discharged home on postoperative day 6.

Of the 5 patients with SCI, none had effective CSF drainage at the time of symptom onset and the rate of SCI was significantly higher in the undrained group (0, 0% vs 5, 7.7%). The SCI patients were at increased risk compared to all other patients in terms of prior AAA repair (4, 80% vs 37, 32%; $P < .05$), emergent TEVAR (n = 40% vs n = 10-15%), and postoperative use of intravenous vasopressors to maintain MAP of ≥90 mm Hg (5, 100% vs 53, 46%; $P < .05$). The length of aortic coverage was significantly longer in patients without SCI (19 cm, range, 15-22) vs 23 cm, range, 10-39; $P < .05$) and the median EBL was not significantly different between the two groups (458 mL, range, 30-3000 vs 900 mL, range, 300-2000; P = NS) (Table IV).

None of the patients were noted to have any of the complications of spinal drain placement including epidural hematoma, meningitis, infections, or intracranial hemorrhage.

DISCUSSION

TEVAR has shown to reduce morbidity and mortality, such as complications of SCI, when compared to open surgical repair.[2] To date, there have been no prospective evaluations comparing outcomes of mandatory CSF drainage during TEVAR, and selective use of CSF drainage has usually been recommended for high-risk patients. Earlier reports have suggested prior AAA repair increased thoracic aortic coverage and subclavian artery interruption to increase the risks of SCI during TEVAR.[1,3,4]

In our experience, although the +CSF drainage group had a higher incidence of prior AAA repair (+CSF: 46%, vs −CSF 23%), greater length of thoracic aortic coverage (+CSF: mean 28 cm, vs −CSF: mean 20 cm), and a higher incidence of left subclavian artery coverage without revascularization (+CSF: 21%, vs −CSF 8%), complications of SCI were significantly lower in this group when compared to the −CSF drainage group (0% vs 8%, $P < .05$). The

Table IV. Subset analysis of spinal cord ischemia patients

	+Spinal cord ischemia	*−Spinal cord ischemia*	P-*value*
N	5	116	
+CSF drainage	0 (0%)	56 (48%)	<.05
Prior AAA repair	4 (80%)	37 (32%)	<.05
AAA coverage (cm)	19 (range, 15-22)	23 (range, 10-39)	<.05
EBL (mL)	900 (range, 200-2000)	458 (range, 30-3000)	NS
Post op vasopressors	5 (100%)	53 (46%)	<.05

N, number; *CSF,* cerebrospinal fluid; *AAA,* abdominal aortic aneyrysm; *EBL,* estimated blood loss.

patients in −CSF drainage group did have a significantly higher median intraoperative estimated blood loss (+CSF: 200 mL vs −CSF: 500 mL, $P < .05$), and a non-significant trend towards a higher incidence of patients presenting with symptomatic and ruptured TAA requiring emergent repair (+CSF: 23% vs −CSF: 32%, P = NS). Patients without CSF drainage that developed complications of SCI following TEVAR did have a significantly increased incidence of prior AAA repair (80%, 4 of 5), and all required intravenous vasopressors in the perioperative period. The 2 patients that presented with ruptured TAA required vasopressors to treat hypotension, the remainder used vasopressors for artificially elevating the MAPs. In this series, of the 5 patients with SCI without CSF drainage, 4 would have been considered high risk secondary to prior AAA repair. Of these, 2 patients presented with ruptured TAA and underwent emergent TEVAR without spinal drainage, 1 patient had spinal drain malfunction, and 1 patient had unsuccessful attempts in spinal drain placement. Only 1 patient with spinal cord ischemia would have been considered low risk with only a short segment TAA, no prior history of AAA repair, and no hemodynamic instability. Furthermore, this patient recovered completely with blood pressure augmentation and postoperative spinal drain placement.

Although our findings would suggest that a mandatory CSF drainage protocol might have a protective role in preventing complications of SCI in that none of the patients in the CSF drainage group developed SCI, a subset analysis indicates that selective CSF drainage in all patients with prior AAA repair, extensive thoracic aortic coverage, and subclavian artery coverage without revascularization would be beneficial in decreasing SCI complications. Furthermore, patients without CSF drainage that develop SCI might benefit from adjunctive maneuvers of active CSF drainage with augmentation of mean arterial blood pressures.

There have been several large TEVAR series that have described SCI complications. Chiesa et al[7] reported their experience of elective and non-emergent TEVAR in 103 patients with selective CSF drainage in patients considered to be high risk (ie, prior AAA repair, aneurysm involving T8-L2, long aortic coverage length). Four (4%) patients without CSF drainage developed complications of SCI, all had unifying risk factor of MAP <70 mm Hg, and they all recovered with active CSF drainage. Cheung et al[8] have reported their findings of 75 patients that underwent TEVAR with selective CSF drainage; 5 (6.7%) patients developed complications of SCI, 3 of whom did not have previous AAA repair and were considered low risk for developing SCI. In our experience, of the 5 patients with SCI following TEVAR, only 1 (20%) patient had MAP <70 mm Hg at the onset of neurological changes, only 1 (20%) did not have a prior AAA repair, and all required perioperative intravenous vasopressors.

There have been several reports that used CSF drainage and MAP augmentation as treatment for SCI when mandatory drainage was not employed.[9-12] This treatment paradigm of immediate CSF drainage and MAP augmentation was employed in our SCI patients with 4 of 5 patients recovering function. The 2 asymptomatic TAA patients in this group each had SCI risk factor of previous AAA repair. The MAPs of these patients were 83 and 71 mm Hg at the time of the neurological changes. Neurological function returned fully in 1 patient and partially in the second. Two SCI patients in our series underwent TEVAR for ruptured TAA and were too unstable to undergo preoperative drainage. Incidentally, both had prior AAA repairs and perioperative hypotension. The MAPs at the time of neurological change were 105 and 60 mm Hg. Only one of these patients recovered function after CSF drainage and MAP augmentation. The final SCI patient in our series did not appear to have any risk factors that would be considered high risk for developing complications of SCI during TEVAR. This patient had full recovery with CSF drainage and MAP augmentation. The unifying factor in treatment of our SCI patients was rapid CSF drainage to a pressure less than 10 mm Hg and MAP augmentation to greater than 90 mm Hg.

The cause of SCI after TEVAR remains ill defined, the currently proposed etiologies are direct coverage of intercostal arteries, atherosclerotic embolization, and interruption of important collateral vessels.[13] The importance of collateral spinal cord blood supply from pelvic arteries and less emphasis on the artery of Adamkiewicz is beginning to challenge current principles in thoracic aortic surgery.[14-16]

Although in our series there were no complications resulting from placement of CSF drainage catheters, CSF drainage can lead to complications of spinal headache, epidural and subdural hematoma, with and without infection/abscess that can range from 1-3%.[17-18]

This is the first prospective TEVAR study that evaluates a standardized approach of mandatory CSF drainage and compares it to selective CSF drainage, and we recognize that there are several limitations of our study. This is a non-randomized study, and we were not able to enroll all consecutive patients into the mandatory CSF drainage protocol; several emergent cases of TAA rupture and traumatic thoracic aortic transections underwent emergent endovascular repair without CSF drainage. Furthermore, spinal cord blood supply via intercostal arteries, lumbar arteries, and cervical and pelvic collaterals was not evaluated.

This is the first TEVAR series to implement a mandatory CSF drainage protocol for all patients regardless of SCI risk factors. The use of our CSF drainage protocol appeared to be protective, resulting in no incidents of SCI in those with effective CSF drainage. Further analysis of our SCI patients in the undrained CSF group demonstrated that CSF drains would have been selectively placed in all of those with previous AAA repair. Our data also suggests that immediate CSF drainage and blood pressure augmentation for symptomatic patients is also an effective strategy for treatment of SCI.

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AUTHOR CONTRIBUTIONS

Conception and design: JH, MM, JT, YS, SR, RD
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Critical revision of the article: JH, MM, JT, YS, SR, RD
Final approval of the article: JH, MM, JT, YS, SR, RD
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