Cerebrospinal Fluid Drainage During TEVAR

When and how we employ CSF drainage to prevent spinal cord ischemia.

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Over the past decade, thoracic endovascular aneurysm repair (TEVAR) has emerged as a less invasive alternative to the traditional open surgical repair and has been shown to have a significant reduction in the morbidity and mortality.[1] Most of the recent studies suggest that TEVAR is associated with a 3% to 6% incidence of spinal cord ischemia (SCI) and have implicated several factors that can increase the risk of SCI. These factors include prior abdominal aortic repair, length of thoracic aortic coverage, hypogastric artery interruption, subclavian artery coverage, emergent repair, and sustained hypotension.[2-7] During surgical repair of thoracic aortic aneurysms, several factors have been suggested to reduce the incidence of SCI and include cerebrospinal fluid (CSF) drainage, intercostal artery reimplantation, maintenance of normotension, and hypothermia.[4,8] During TEVAR, the use of CSF drainage to improve on the incidence of SCI is evolving and has been used in selective high-risk patients.[7,9]

THE ALBANY VASCULAR INSTITUTE EXPERIENCE

Since 2004, at the Albany Vascular Institute, approximately 200 patients have undergone TEVAR for a variety of emergent and elective thoracic aortic pathologies. Early in our experience, like other vascular centers, we also had many questions regarding the indications for CSF drainage during TEVAR. Our initial strategy was to employ CSF drainage selectively—only in patients with prior abdominal aortic reconstruction. Even with this strategy, some low-risk TEVAR patients developed symptoms of spinal cord ischemia, and in light of these findings, we established a protocol that included CSF drainage in all patients undergoing TEVAR. We share our experience in this report.

FINDINGS

Over a 3-year period, from 2004 to 2006, 121 patients underwent elective (n=52; 43%) and emergent (n=69; 57%) endovascular thoracic aortic stent graft placement for thoracoabdominal aneurysm (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 SCI. The CSF was actively drained to maintain pressures <15 mm Hg, and the mean arterial blood pressures were maintained at ≥90 mm Hg. After completion of TEVAR, all patients were kept on bed rest in the ICU. The mean arterial pressures (MAPs) were recorded every hour and maintained at >90 mm Hg for 24 hours by using fluid boluses and intravenous vasopressors. The CSF pressures were recorded every hour; for pressures >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 SCI, CSF was actively drained and maintained at a pressure of <10 mm Hg.

After 24 hours, the MAP was allowed to drift below 90 mm Hg, and the vasopressors were weaned off. If there were no signs of SCI after 12 hours, the spinal drain was clamped,

TABLE 1. 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

TABLE 2. 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, 30–1,200)	500 (range, 30–3,000)	<.05
Mean aortic coverage (cm)	28 (range, 10–39)	20 (range, 10–32)	<.05
Perioperative vasopressors	36 (64%)	22 (34%)	<.05

the patients were mobilized out of bed, and 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, the CSF was drawn off to keep the intrathecal pressure of <10 mm Hg, and the MAPs were maintained at >90 mm Hg using vasopressors as needed. Patients without spinal drainage who 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. Data were prospectively collected in our vascular registry for elective and emergent endovascular thoracic aortic repair, and the patients were divided into two groups (+ CSF drainage protocol, – CSF drainage protocol). Chi square statistical analysis was performed, and significance was assumed for *P*<.05.

The + CSF drainage and – CSF drainage groups were similar with respect to age, demographics, comorbidities, and the indications for TEVAR (Table 1). The + CSF group had a significantly higher percentage of patients with previous 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 estimated blood loss (EBL) (500 mL vs 200 mL; *P*<0.05) (Table 2).

In the + CSF drainage group, one patient (1.8%) had malfunction of the CSF drain and developed SCI 24 hours after TEVAR. After placement of a new CSF drain and resumption of CSF drainage protocol, he had a full recovery. In the – CSF drainage group, four patients (6.2%) developed symptoms of SCI and underwent placement of CSF drainage catheters; two patients had full recovery, and two had persistent SCI.

To identify patient risk factors for developing SCI, we took a closer look at all patients who developed any SCI complications. Of the five patients with SCI, none had effective CSF drainage at the time of symptom onset. The SCI patients were at increased risk compared to all other patients in terms of prior AAA repair (four of five [80%] vs 37 of 116 [32%], and postoperative use of intravenous vasopressors to maintain MAP of 90 mm Hg (five [100%] vs 53 [46%]; *P*<.05). Surprisingly, of the patients without CSF drainage, the ones that developed symptoms of SCI had significantly less mean aortic coverage (19 cm; range, 15–22 cm) when compared to those without any SCI symptoms (23 cm; range, 10–39 cm) (Table 3).

TABLE 3. 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
Mean aortic coverage (cm)	19 (range, 15–22)	23 (range, 10–39)	<.05
EBL (mL)	900 (range, 200–2,000)	458 (range, 30–3,000)	NS
Postoperative vasopressors	5 (100%)	53 (46%)	<.05

DISCUSSION

When compared to open surgical repair, TEVAR has shown to decrease mortality and morbidity, including complications of SCI.[2] Earlier reports have suggested previous abdominal aortic aneurysm (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 previous AAA repair (46% vs 23%), greater length of thoracic aortic coverage (mean 28 cm vs mean 20 cm), and a higher incidence of left subclavian artery coverage without revascularization (21% vs 8%), complications of SCI were significantly lower in this group when compared to the – CSF drainage group (0% vs 8%, *P*<.05). The patients in the – CSF drainage group did have a significantly higher mean intraoperative estimated blood loss (+ CSF: 200 mL vs – CSF: 500 mL, *P*<.05) and a nonsignificant trend toward a higher incidence of patients presenting with symptomatic and ruptured thoracic aortic aneurysms (TAA) requiring emergent repair (+ CSF: 23% vs – CSF: 32%, *P*=NS). Patients without CSF drainage who developed complications of SCI after TEVAR did have a significantly increased incidence of previous AAA repair (80%, 4/5), and all required intravenous vasopressors to maintain a mean arterial blood pressure of >80 mm Hg.

In this series, of the five patients with SCI without CSF drainage, four would have been considered high risk secondary to previous AAA repair. Of these, two patients presented with ruptured TAA and underwent emergent TEVAR without spinal drainage, one patient had spinal drain malfunction, and one patient had unsuccessful attempts in spinal drain placement. Only one patient with spinal cord ischemia would have been considered low risk with only a short segment TAA, no previous history of AAA repair, and no hemodynamic instability. Furthermore, this patient recovered completely with blood pressure augmentation and postoperative spinal drain placement.

CONCLUSION

Although our findings would suggest that a mandatory CSF drainage protocol might have a protective role in preventing complications of SCI, a subset analysis indicates that selective CSF drainage in all patients with previous AAA repair, extensive thoracic aortic coverage, and subclavian artery coverage without revascularization would be beneficial in decreasing SCI complications and may offer similar benefits as mandatory CSF drainage in all patients. Furthermore, patients without CSF drainage who develop SCI might benefit from adjunctive maneuvers of active CSF drainage with augmentation of mean arterial blood pressures. ■

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