Can We Protect The Spinal Cord?

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LEARNING OBJECTIVES:


2. Recognize perioperative events that increase the risk of postoperative spinal cord ischemia after endovascular stent repair of descending thoracic aortic aneurysms.

3. Learn clinical strategies to prevent and detect spinal cord ischemia in patients undergoing endovascular stent repair of descending thoracic aortic aneurysms.

4. Learn and understand the physiologic rationale for interventions to treat acute spinal cord ischemia during and after endovascular stent repair of descending thoracic aortic aneurysms.

5. Understand the potential benefits and risks of lumbar cerebrospinal fluid drainage in patients undergoing thoracic aortic endovascular stent graft repair.

CASE:

A 72 year-old female was found to have an incidental abdominal aortic aneurysm (5.0 cm) extending beneath her renal arteries during her yearly exam. Further evaluation with CT angiogram revealed a large thoracic aneurysm of the ascending aorta (7.1 cm), the aortic arch (6.6 cm) and the descending thoracic aorta (5.4 cm) with sparing of the aortic valve and root. Past medical history was positive for hypertension, hyperlipidemia, asymptomatic coronary artery disease, carotid stenosis, and tobacco dependence.

The patient underwent Stage I of an elephant trunk procedure which involved total replacement of the ascending aorta and the aortic arch as well as coronary artery bypass grafting to the RCA. Four months later after adequate recovery, the patient underwent Stage II of the procedure to repair her thoracoabdominal aortic aneurysm. This was accomplished in two different operations. She underwent a 10-hour aortic debranching procedure first; this included aortobifemoral bypass, aortoiliac bypass, aorta to superior mesenteric artery bypass, aorta to left and right renal arteries bypass. Anesthesia included use of arterial line, central line and intraoperative TEE. The patient
did not require any pressor support as she maintained adequate perfusion pressures with average MAP > 80 mm Hg. Urine output was maintained with infusion of 3.5L of crystalloid and 2 units of PRBCs. She was extubated after this procedure and suffered no neurological deficits.

Two days later, she returned to the operating room for thoracoabdominal endovascular aortic endograft repair (TEVAR), which involved thoracic endograft placement of the descending thoracic and abdominal aorta using three Gore TAG grafts. Preoperatively to TEVAR, she underwent placement of a lumbar drain. Spinal cord perfusion pressure was augmented by drainage of CSF to achieve a lumbar CSF pressure > 10 mm HG. Prior to deployment of the grafts, an infusion of phenylephrine 80 mcg/min was started to achieve a MAP of > 95 mm Hg. Three grafts were deployed in sequence in thoracoabdominal aorta; the grafts covered proximal into the elephant trunk in the thoracic aorta to just proximal to the distal bypass grafts in the abdominal aorta that were constructed 2 days prior during the aortic debranching procedure (see illustration). Using phenylephrine to maintain a MAP > 95 mm Hg, she remained hemodynamically stable for the duration of the procedure. Total TEVAR procedure time was approximately 4 hours. The patient awakened immediately following emergence from general anesthesia and had normal neurologic function in all four extremities.

The patient remained on phenylephrine infusion for approximately 24 hours in the ICU. She was gradually weaned as arterial pressures improved and maintained a MAP of > 90 mm Hg. The CSF drain was removed without incident 72 hours postoperatively during which time the patient remained asymptomatic. Four days postoperatively, she complained of decreased right-sided vision. Neurological assessment revealed significant decreased visual acuity thought to be related to her significant right internal carotid occlusion. Motor strength was equal in all extremities and she complained of no sensory deficits. The patient was discharged POD 21 and did suffer permanent unilateral vision loss.

**KEY QUESTIONS:**

1. Did this patient’s previous thoracic aortic repair increase her risk for spinal cord ischemia? Explain what are risk factors for spinal cord ischemia during TEVAR.

2. Should somatosensory evoked potential monitoring have been used in this case? Is SEP monitoring the only means to detect intraoperative spinal cord ischemia? Are there other techniques to detect intraoperative spinal cord ischemia? Would it be better to perform these operations using regional anesthesia?

3. A lumbar CSF drain was inserted prior to the endovascular operation. Which patients justify this technique? What is the evidence to support the use of lumbar CSF drainage in this setting?

4. Describe two techniques to increase spinal cord perfusion for the treatment of spinal cord ischemia after repair of descending thoracic aortic aneurysms.

5. Provide some possible explanations for hypotension associated with delayed-onset paraplegia or paraparesis after thoracic or thoracoabdominal aortic operations. What diagnostic tests are useful to determine the cause of hypotension in this setting?
6. What are the risks of lumbar CSF drainage? What can be done to decrease the complications associated with lumbar CSF drainage?

DISCUSSION:

Due to improvements in medical and surgical therapies, more patients are presenting for surgical repair of complex aortic aneurysms. A tragic complication of thoracoabdominal aortic aneurysm repair is paraplegia, the incidence of which is thought to be equal to or less than 20%. The cause of neurological deficits is thought to be multifactorial; patients with vascular disease often have significant comorbidities, decreased collateral circulation, and decreased perfusion pressure. Ensuring adequate spinal perfusion intraoperatively can be difficult during both open and endograft repairs of the aorta. Thoracic endovascular aortic repair (TEVAR) may offer some decrease in morbidity and mortality in the vascular patient population compared to open repair; however, the existing clinical experience with endovascular stent graft repair of isolated descending thoracic aortic aneurysms indicates that spinal cord ischemia remains a serious complication of this procedure. The reported incidence of spinal cord ischemia after endovascular stent repair of thoracic aortic aneurysms has ranged from 3.6% to 12.0%. The incidence of permanent neurologic deficit as a consequence of spinal cord ischemia after endovascular stent repair was comparable to the 2.6% to 2.7% incidence of neurologic deficits observed after open repair of isolated descending thoracic aneurysms.

Studies suggesting modalities for organ protection during open and endovascular thoracoabdominal aortic repairs are limited and often difficult to conduct prospectively. However, recent guidelines published in Circulation in 2010 by multidisciplinary medical societies on thoracoabdominal aortic disease help provide recommendations for the detection, diagnosis and management of these processes. Strategies include deep hypothermic arrest, antegrade and retrograde cerebral perfusion, CSF drainage, re-implantation of intercostal arteries, distal aortic perfusion, motor and somatosensory evoked potential monitoring (MEP and SEP) to detect ischemia, and hemodynamic optimization to increase spinal cord perfusion pressure (SCPP).

Although stent grafting (TEVAR) avoids the period of aortic cross clamp and may be associated with fewer episodes of intraoperative hypotension from hemodynamic perturbations or blood loss, the risk of spinal cord ischemia has not been eliminated. Possible explanations for the persistent risk of spinal cord ischemia after stent grafting include coverage of a greater extent of aorta to accommodate ideal proximal and distal landing zones for the graft that are further away from the true aneurysm. In the open repair technique, intercostal arteries that would normally be sacrificed after stent grafting could potentially be reattached. Stent grafting was also associated with risk of injury to iliofemoral vessels that may be important for collateral circulation to the spinal cord through the hypogastric and pelvic vascular plexus. Finally, candidates for endovascular stent graft repair were often older patients, patients with a greater number and severity of co-morbidities, and patients who would not normally be considered candidates for open surgical repair. As a consequence, a management strategy using intraoperative neurophysiologic monitoring, interventions to improve spinal cord perfusion pressure in patients at risk, and early detection and treatment of spinal cord ischemia are important considering the
risk of permanent paraplegia or paraparesis during endovascular stent repair of descending thoracic aortic aneurysms.

Factors believed to cause or contribute to the development of spinal cord ischemia after endovascular stent repair of thoracic aortic aneurysm are previous abdominal aortic aneurysm repair, hypotension associated with an occult retroperitoneal bleed, severe atherosclerosis of the thoracic aorta, injury to the external iliac artery, and the extent of the descending thoracic aorta covered by graft (see Table 1 below). The risk of spinal cord ischemia in patients with extensive coverage of the descending thoracic aorta may be explained by the number of intercostal arteries excluded or exclusion of critical intercostal arteries at the T6 to T12 vertebral levels that supply the anterior spinal artery. The risk of spinal cord ischemia in patients with prior abdominal aortic aneurysm repair may be explained by compromised collateral vascular supply to the spinal cord from the pelvic and hypogastric circulation as a consequence of prior sacrifice of the inferior mesenteric artery or sacrifice of the median sacral artery. Endovascular stent grafting of a descending thoracic aortic aneurysm in a patient with a prior abdominal aortic aneurysm repair may be considered an anatomic or physiologic equivalent to an open extent II or III thoracoabdominal aneurysm repair. Similarly, injury to the external iliac artery from intravascular delivery of the stent or severe pre-existing occlusive disease of the femoral or iliac arteries may contribute to spinal cord ischemia because spinal cord collaterals originating from the iliac arteries may be compromised.

### Risk Factors for Paraplegia after TAAA

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<tr>
<th>Risk factors for paraplegia after open TAAA repair</th>
<th>Mechanism of injury</th>
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<tr>
<td>Emergency presentation (aortic dissection or rupture)</td>
<td>Decreased perfusion pressure</td>
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<td>Postoperative hypotension</td>
<td>Acute disruption of collateral circulation</td>
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<td>More extensive aneurysms (Crawford type I or II)</td>
<td>Worsen ischemia-reperfusion injury</td>
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<td>Ligation of spinal collateral vessels</td>
<td>Decreased collateral circulation</td>
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<td>Prolonged aortic cross-clamp time</td>
<td>More medical comorbidities</td>
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<td>Previous abdominal aortic aneurysm repair</td>
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<td>Severe atherosclerotic disease</td>
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<td>Diabetes</td>
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<td>Advanced age</td>
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### Risk factors for paraplegia after endovascular TAAA repair (TEVAR)

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<tr>
<td>Previous abdominal aortic aneurysm repair</td>
<td>Decreased collateral circulation</td>
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<tr>
<td>Severe atherosclerosis of the thoracic aorta</td>
<td>Decreased perfusion pressure</td>
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<tr>
<td>Hypotension</td>
<td>Acute disruption of collateral circulation</td>
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<tr>
<td>Injury to the external iliac artery</td>
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<tr>
<td>Occlusion of left subclavian artery or hypogastric arteries</td>
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<td>More extensive coverage of the thoracic aorta by graft</td>
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Hypotension has been associated with the development of spinal cord ischemia after repair of thoracoabdominal aortic aneurysms. Hypotension may be caused by hemorrhage, drug therapy, or even neurogenic shock from spinal cord ischemia. Hypotension preceding the onset of two distinct episodes of postoperative paraparesis may have been caused by autonomic dysfunction and represented an early manifestation of spinal cord ischemia.
Arterial blood pressure augmentation to improve spinal cord perfusion pressure is often effective for the treatment of spinal cord ischemia after endovascular stent repair of descending thoracic aortic aneurysms. Restoration of arterial blood pressure by volume expansion and vasopressor therapy should be considered for the acute treatment of paraplegia associated with hypotension. Tests should be performed to identify sites of occult hemorrhage. Phenylephrine and norepinephrine infusion to treat hypotension associated with spinal cord ischemia has also been effective for the treatment of neurogenic shock caused by autonomic dysfunction as a consequence of spinal cord ischemia. The immediate administration of epinephrine and norepinephrine infusions to augment the arterial pressure in the patient with intraoperative spinal cord ischemia detected by SEP monitoring restored lower extremity SEP signals and may have prevented postoperative paraplegia or paraparesis. In general, arterial pressure should be augmented to maintain a spinal cord perfusion pressure (MAP-lumbar CSF pressure) of at least 70 mm Hg at all times and vasopressors specifically administered to increase the mean arterial pressure to a range of 85 mm Hg to 100 mm Hg in patients with clinical evidence of spinal cord ischemia. The risk of arterial hemorrhage as a consequence of arterial pressure augmentation may be less after endovascular compared to open repairs because of the decreased or absence of major vascular anastomoses.

Lumbar CSF drainage has been advocated as an important intervention to prevent and treat spinal cord ischemia after open TAAA repair. The primary objective of lumbar CSF drainage is to decrease the lumbar CSF pressure in order to increase the net spinal cord perfusion pressure. Meta-analysis performed on 372 publications that include 3 randomized controlled trials involving 289 patients and 5 cohort studies involving 505 patients showed that lumbar CSF drainage was effective in decreasing the risk of paraplegia with a pooled odds ratio of 0.30 (p=0.05, 95% CI, 0.17-0.54). A meta-analysis performed by the Cochrane Collaborative on the three published randomized controlled trials of lumbar CSF drainage demonstrated a significant benefit of lumbar CSF drainage with a pooled odds ratio of 0.48 (95% CI, 0.25-0.92). However, if the randomized controlled trial that tested lumbar CSF drainage in combination with intrathecal papaverine was eliminated from the meta-analysis, there was insufficient evidence to support the efficacy of lumbar CSF drainage in the remaining two randomized controlled trials. Based on this reasoning, the Cochrane report concluded that lumbar CSF drainage is recommended as a component of the multi-modality approach for prevention of neurological injury and that use of lumbar CSF drainage alone as protection has not been established from the available evidence.

Evidence to support the efficacy of lumbar CSF drainage to decrease the incidence of spinal cord ischemia after endovascular stent graft procedures is limited, but successful use of lumbar CSF drainage for the prevention or treatment of spinal cord ischemia in this setting have been reported. Prophylactic use of lumbar CSF drainage may have contributed to recovery after evidence of intraoperative spinal cord ischemia detected by SEP monitoring. Prophylactic application of lumbar CSF drainage may be considered for patients with prior abdominal aortic aneurysm repair or who require extensive coverage of the descending thoracic aorta when the risk of spinal cord ischemia is perceived to be greater. Lumbar CSF pressures may not increase after endovascular stent deployment, but differences in CSF hemodynamics in response to endovascular compared to open repair of thoracic aortic aneurysms remain to be studied. In some reported cases of spinal cord ischemia that recovered after lumbar CSF drainage, elevated lumbar
CSF pressures may have contributed to the onset of spinal cord ischemia and may explain the efficacy of lumbar CSF drainage.

Although lumbar CSF drainage appears to be safe, even in patients subjected to full systemic anticoagulation for operations performed using extracorporeal circulation, subdural hematoma, intracranial hypotension, spinal hematoma, meningitis, and catheter fracture have been reported as complications of lumbar CSF drainage. Complications associated with lumbar CSF drainage may potentially be minimized or prevented with appropriate attention to the proper management of patients undergoing lumbar CSF drainage. The volume of CSF drained should be strictly controlled and the lumbar CSF pressure should be monitored continuously to maintain it at no less than 10 mm Hg to prevent intracranial hypotension. Draining large volumes of CSF or allowing the lumbar CSF pressure to decrease below 10 mm Hg may increase the risk of subdural hematoma caused by intracranial hypotension. Coagulation function should be assessed at both the time of lumbar CSF catheter placement and catheter removal to decrease the risk of hemorrhagic complications. Intracranial subdural hematoma, remote intracerebellar hemorrhage, or meningitis should be considered as potential complications of lumbar CSF drainage in patients with unexplained neurologic deterioration.

It is also important to consider and prevent complications related to the use of lumbar CSF drainage. Complications associated with lumbar CSF drainage include intracranial hypotension, subdural hematoma, intradural spinal hematoma, catheter fracture, and infection. Patients with lumbar CSF drainage also require a longer intensive care unit length of stay for management and removal of the lumbar CSF catheter.

Intraoperative neurophysiologic monitoring has also been a recognized technique for the detection of spinal cord ischemia during open repair of thoracoabdominal aortic aneurysm, but the clinical experience of intraoperative neurophysiologic monitoring for endovascular stent repair is limited. Intraoperative SEP monitoring may be considered when available for patients with prior abdominal aortic aneurysm repair or if there is a perceived risk of spinal cord ischemia. When using intraoperative monitoring of lower extremity SEP during endovascular stenting, it is important to distinguish patterns of lower extremity ischemia caused by vascular insufficiency as a consequence of vascular access complication from patterns associated with spinal cord ischemia. Prompt intervention to augment spinal cord perfusion pressure in response to spinal cord ischemia detected by SEP monitoring was associated with recovery of lower extremity SEP and the prevention of postoperative paraplegia or paraparesis. Although additional clinical experience will be necessary to justify the routine use of neurophysiologic monitoring in patients undergoing endovascular stent procedures, preliminary experience supports the ability of intraoperative SEP monitoring to detect spinal cord ischemia. Although not performed, monitoring intraoperative motor evoked potentials may be more sensitive than SEP monitoring for the detection of isolated motor deficits. Intraoperative motor evoked potential monitoring may be less demanding during endovascular stent procedures because the requirements for neuromuscular blockade are less compared to open repairs.

Repair of isolated descending thoracic aortic aneurysms with endovascular stent grafts may compromise the intercostal arterial supply to the spinal cord and causing spinal cord ischemia or infarction in susceptible patients. Patients requiring extensive graft coverage of the descending thoracic aorta, with a compromised pelvic hypogastric collateral supply to the spinal cord from
prior abdominal aortic aneurysm repair or external iliac artery injury appear to be at increased risk of spinal cord ischemia after endovascular stent graft repair. Events such as hemorrhage or autonomic dysfunction causing hypotension may also trigger spinal cord ischemia after endovascular stent repair. Immediate detection of spinal cord ischemia by intraoperative SEP monitoring or neurologic examination combined with interventions that increased spinal cord perfusion may be effective in treating paraplegia or paraparesis during and after endovascular stent repair.

REFERENCES:


Figure 1. Aortic CT reconstruction post aortic debranching procedure and endograft placement.