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Descending Thoracic Aortic Repair – Spinal Cord Protection Strategies
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Stem Case and Key Questions Content
Case Presentation: A 42-year old man with a Crawford type 2 TAAA presents for an open repair by left thoracoabdominal approach. Co-morbidities include Marfan's disease, hypertension, and chronic renal insufficiency. After arrival to the OR suite, a lumbar drain (L4-5 level) and a thoracic epidural (T8-9 level) are placed. Anesthesia is induced after placement of a right radial arterial line. Thereafter, a 9.0 F right internal jugular introducer is placed. An Arndt (Cook Inc) endobronchial blocker is positioned in the left mainstem bronchus, with the aid of fiberoptic bronchoscope.
Surgical repair of the aneurysm is performed after initiation of left heart bypass. The aneurysmal aorta is replaced with a PTFE graft from distal to the left subclavian artery to the aortic bifurcation. Bypasses are performed to the celiac, superior mesenteric and bilateral renal arteries.

1. What is the likelihood of paraplegia after an open TAAA repair?
2. What are the risk factors for spinal cord injury (paraplegia) following open TAAA repair?
3. Are any additional intraoperative monitors warranted to detect or decrease the risk of intraoperative spinal cord ischemia?
4. What are the determinants of spinal cord perfusion pressure?
5. What are the effects of clamping and unclamping of the aorta on CSF hydrodynamics during an open TAAA repair?
6. How strong is the evidence to support the use of lumbar CSF drainage in open TAAA repairs?
7. What other measures can be taken to minimize the risk of spinal cord ischemia during a TAAA repair?
8. Would you cancel or proceed with the surgical repair if you encountered a bloody tap during placement of the lumbar drain?

Case Continuation: CSF drainage is initiated prior to aortic clamping in order to maintain CSF pressure around 10 mm Hg. Upon termination of the surgery, patient is transferred to the ICU intubated. Post-operatively, patient is noted to have bilateral lower extremity weakness, which does not resolve despite optimization of spinal cord perfusion pressure.

1. To what reference point should the lumbar drain be zeroed?
2. What should be the goals for CSF drainage and how long should CSF drainage be maintained in this scenario?
3. How would you determine if this patient's symptoms are from an epidural hematoma versus spinal cord ischemia?

4. What complications related to lumbar drains would you be worried about?
5. What if CSF becomes bloody during drainage postoperatively?

Additional Clinical Scenario for Discussion: Imagine the same patient, described above, undergoing a 4-vessel fenestrated endovascular repair instead of an open repair.

1. Is a lumbar drain still indicated for the fenestrated endovascular repair?
2. How do you determine if a lumbar CSF drain is indicated?
3. Are the risk factors for spinal cord injury (paraplegia) following thoracic endovascular aortic aneurysm repair (TEVAR) any different?
4. What's the evidence to support the use of lumbar CSF drainage for TEVARs?
5. Is there a role for CSF drainage as a rescue measure for delayed paraplegia?

Model Discussion Content
Open thoraco-abdominal aortic aneurysm (TAAA) repairs are one of the most formidable operative procedures in vascular surgery. The risk of spinal cord injury (paraplegia) after open TAAA repairs has been reported to be as high as 40%, although more recent reports indicate an incidence of around 20%. Also, patients who develop paraparesis or paraplegia not only have to deal with the physical disability but have an increased risk of ventilator-dependent respiratory failure and a higher early and late mortality (1-4).

The risk factors for development of paraplegia after an open TAAA repair are more or less similar to the ones for endovascular repair:

• Emergency surgery (dissection, or rupture)
• Perioperative hypotension
• Aneurysm extent, specifically Crawford type I or II aneurysms
• Prolonged aortic cross-clamp
• Previous AAA repair or distal aortic procedures
• Ligation of spinal collateral vessels (including artery of Adamkiewicz)
• Severe occlusive peripheral vascular disease
• Renal insufficiency
• Advanced age

CSF production increases during ischemia (from the aortic clamping) causing increased pressure immediately after aortic cross-clamping. It is believed that spinal cord edema may accompany spinal cord ischemia and contribute to an increase in the CSF pressure. The combination of compromised collateral circulation, increased CSF pressure, inadequate arterial perfusion pressure, or systemic hypotension increases the risk of spinal cord ischemia. Aortic clamp release and reperfusion may also create conditions that increase the risk of spinal cord ischemia. Hypercarbia (increases CSF pressure), systemic hypotension (which decreases spinal cord perfusion pressure), metabolic acidosis (increases ICP and CSF pressure), low cardiac output (that increases venous pressure) and spinal cord edema are all factors that may contribute to spinal cord ischemia when collateral vessels to the spinal cord are sacrificed or compromised as a consequence of TAAA repair.

Strategies to decrease the risk of spinal cord ischemia require a multi-pronged approach

towards spinal cord protection that include distal aortic perfusion, hypothermia, reattachment of the intercostal arteries, CSF drainage, arterial blood pressure augmentation, administration of neuroprotective agents, and monitoring of somato-sensory or motor-evoked potentials. It is important to note that spinal cord ischemia often causes hypotension as a consequence of autonomic dysfunction. Early detection and treatment of spinal cord ischemia is essential to prevent evolution to permanent spinal cord infarction. In one study (Jex et al) the risk of spinal cord injury decreased from 44% to 8% with distal aortic perfusion (5). The techniques for distal aortic perfusion are passive shunts, partial left heart bypass or total cardiopulmonary bypass.

Active distal bypass perfusion has been shown to achieve significantly greater distal aortic pressure than either the clamp and sew technique or passive shunting. Cooling of the spinal cord either by regional methods (epidural catheters with cold saline perfusion) or by systemic methods (cardiopulmonary bypass) have both been reported as adjuncts to protect the spinal cord from ischemia. Several pharmacologic agents including free radical scavengers, barbiturates, corticosteroids, papaverine, cocaine-derived anesthetics, and opiate antagonists have been tried to increase the tolerance of the spinal cord to ischemia, but evidence supporting their clinical effectiveness is indeterminate at best.

Clinical evidence that proves the benefits of CSF drainage is limited given the paucity of randomized controlled trials, but the American Heart Association guidelines for the management of thoracic aortic diseases and a Cochrane analysis both recommend the use of lumbar CSF drainage as an adjunct for the treatment and prevention of paraplegia among patients undergoing TAAA repair who are at risk for spinal cord ischemia (6-14). Differing study designs (in the amount and timing of CSF drained) as well as the use of other adjuncts (such as distal aortic perfusion, arterial pressure augmentation, reattachment of collateral vessels, mild hypothermia, intrathecal papaverine, epidural cooling, moderate heparinization, neuroprotective agents such as naloxone) for spinal cord protection in the studies so far, make it difficult to interpret the specific incremental benefits of CSF drainage. In fact two extensive reviews concluded that CSF drainage in the absence of other spinal cord protective adjuncts may have limited benefit. In a relatively large recent RCT (Coselli et al, 2002) involving Crawford type I and II TAAA repairs, patients in the interventional arm (CSF drainage to maintain CSF pressure < 10 mm Hg) had an 80% reduction in postoperative neurologic deficits (13% vs. 2.6%, P = 0.03) compared to the control group (7). Both the groups had mild hypothermia, left heart bypass and reattachment of the intercostals.

Lumbar drains can be placed either in an awake patient or after induction of anesthesia. The advantage of placing it in an awake patient is that the patient is able to verbalize if there is pain or paresthesia during insertion to minimize the risk of nerve injury. There are institutions though, where drains are routinely placed after the induction of anesthesia (15). The catheter is usually threaded 10-15 cm into the subarachnoid space to ensure adequate drainage of CSF and reduce the chance of dislodgement. There is no clear consensus as to what course should be taken if there is a "bloody" tap during lumbar drain placement. Neuraxial hematomas are a concern, but there are no clinical studies to indicate a best approach to this situation. The decision to proceed with the surgery after a traumatic tap is usually based on the combined judgment of the anesthesiologist and surgeon and the risk of hemorrhagic complications may be decreased by ensuring delay in the administration of heparin for anticoagulation and maintaining a tight control of coagulation function.

The lumbar CSF pressure should always be measured continuously. When using an electronic pressure transducer, it is important that the pressure transducer be primed with either preservative-free saline or patient's CSF and that it is not connected a pressurized flushing system. The anatomic site used to provide the zero reference for measuring lumbar CSF pressure is also controversial. Some recommend that the lumbar drain transducer be zeroed at the level of the right atrium (phlebostatic axis) while others recommend zeroing at the level of the tragus. The tragus provides a better assessment of intracranial hemodynamics when there is a concern for intracranial hypotension or risk of subdural hematoma. Setting the zero at the level of the right atrium may provide a better measure for estimating the actual spinal cord perfusion pressure. Although there is no consensus, it is preferred that CSF pressure (and waveform) be monitored continuously and drained intermittently whenever the lumbar CSF pressure exceeds 10 mm Hg. In the absence of any neurologic deficits post-operatively, the lumbar CSF drain can be removed between 24 - 72 hours after operation. Lumbar drains can be left in place longer in the presence of neurologic deficit or coagulopathy although this may increase the infection risk.

Lumbar drains although a useful adjunct in protecting the spinal cord are not without risks as shown by a recent study (Wynn et al, 2009) of 486 patients undergoing thoracoabdominal aneurysm repair (15-18). Neurological deficits attributable to the spinal drain itself including intracranial hemorrhage (ICH) occurred in 1% of patients. Complications can be classified into three broad groups:

• Catheter placement- or removal-related complications
o Nerve root injury
o Neuraxial hematoma
o Catheter fracture
o CSF leak
• Indwelling catheter
o Local infection
o Meningitis

• Drainage-related complications
o Intracranial hypotension (headache, abducens nerve palsy)
o Intracranial hemorrhage
o Subdural hematoma

ICH is thought to be from excessive drainage of CSF over a short period of time. Patients with cerebral atrophy, AV malformations, cerebral aneurysms or previous subdural hematomas are predisposed to developing ICH. Patients treated with antiplatelet agents or warfarin may also be at increased risk of early or delayed subdural hematoma. It has been suggested that no more than 10 to 20 ml per hour of CSF be drained unless drainage is being performed to treat acute spinal cord ischemia manifested by paraparesis or paraplegia in which case more aggressive drainage (greater than 20 ml/h and maintenance of lumbar CSF pressure in the range of 8-10 mm Hg) may be warranted.

Presently, endovascular repair with fenestrated grafts are an alternative to open TAAA repair among patients with Crawford extent II and III TAAA. Existing clinical data suggests that

endovascular repair may be associated with a decreased risk of spinal cord ischemia and with lower perioperative morbidity and mortality rates when compared with standard open aortic repair (19-29). The advantages of endovascular repair include avoidance of

• aortic clamping and unclamping
• partial (left heart) bypass
• large thoraco-abdominal incision
• one-lung ventilation
• large fluid shifts

Although the incidence of paraplegia is lower with endovascular procedures, it is still reported to be between 5 and 8%. The cause of paraplegia in endovascular procedures is likely different from open surgery since no aortic cross-clamping is required. It is most likely because of permanent coverage of important intercostal arteries or from disruption of other important collateral arteries from the pelvic, lumbar or hypogastric vessels.
Hypoperfusion seems to be the most likely reason for spinal cord injury as suggested by the following risk factors that predispose to paraplegia after TEVAR:

• Previous distal aortic operations or AAA repair (previously damaged collateral circulation)
• Extensive coverage of the thoracic aorta by the graft
• Prolonged hypotension
• Severe atherosclerosis of the thoracic aorta
• Injury to the external iliac artery
• Occlusion of the left subclavian or the hypogastric arteries
• Renal insufficiency

Hnath et al (2008) in a prospective observational study reported significantly lower incidence of paraplegia, compared to historical controls, in 121 patients who underwent elective or emergent TEVAR along with prophylactic lumbar CSF drainage (19). They were also able to show that the benefit was greatest in patients at a higher risk for spinal cord injury such as previous AAA repair, more extensive graft coverage and occlusion of the left subclavian, hypogastric and iliac arteries.

There are several case reports in the literature that highlight the role of CSF drainage as a rescue therapy in postoperative patients where lumbar CSF drainage was not performed prophylactically (30-31). The existing published clinical series and case reports suggest that immediate treatment with arterial pressure augmentation and lumbar CSF drainage upon the onset of spinal cord ischemia is necessary to achieve a favorable clinical recovery. Delayed treatment may be less successful because of the onset of irreversible infarction. Bajwa et al (2008) reported an interesting case where the patient developed spinal cord injury after an endovascular repair of an infrarenal aneurysm (31). This patient had ectatic and calcified common iliac arteries along with tortuous calcified external iliac arteries. Due to the iliac vessels involvement endovascular stenting was performed in two stages. First, the left internal iliac artery was embolized and a right to left femoro-femoral crossover grafting was performed. In the second stage, a right aorto uniliac endovascular stent was deployed via the right ilio-femoral conduit. Post-operative paraplegia responded to CSF drainage and optimization of spinal cord perfusion pressure. The authors of the case report point out that the spinal cord of their patient

was most likely dependent on the pelvic circulation and hence the ischemia (following embolization of left internal iliac and stenting of the right iliac).

In summary, despite all of the advances in the treatment of thoracoabdominal aortic repairs, spinal cord injury is still a major complication. Fortunately, advances in the understanding of the etiology of spinal cord ischemia among patients undergoing TAAA repair have increased our ability to prevent, detect, and treat this serious complication. A multimodal approach is necessary since no single technique has been shown to be completely effective for the prevention and treatment of spinal cord ischemia.

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