PBLD Spinal Cord Protection

OBJECTIVES:

1. Learn to identify patients at risk for spinal cord ischemia after open or endovascular thoracic and thoracoabdominal aortic aneurysm (TAAA) repair.

2. Learn what techniques can be employed to decrease the risk of spinal cord ischemia for thoracic and thoracoabdominal aortic repair.

3. Learn what techniques can be used to detect spinal cord ischemia in patients under general anesthesia.

4. Understand the potential complications of lumbar cerebrospinal fluid drainage and how to avoid them

Case Presentation;

42 year old man presented to the emergency room with new onset of severe abdominal and back pain. Patient had history of Marfan’s syndrome, and known dilatation of thoracic abdominal aorta that was followed closely. His medications included Metoprolol 50 mg once a day and Lisinopril 10mg once a day. He had no other significant medical history.

At presentation to the emergency room, aortic dissection was suspected. CT scan was done and showed TAAA of 6 cm at the widest point with contained dissection from L subclavian to iliac bifurcation. He was hemodinamically stable but in a lot of pain. Esmolol infusion was started and he was treated with Morphine for pain. Blood pressure was controlled by infusion of nitroglycerin.
Questions:

1. What is the next step in treatment of this patient?
2. What are the available treatment options for TAAA?
3. Describe the Crawford’s classification of aortic aneurysms and the significance
4. What is the devastating complication of TAAA procedures?
5. What are the risk factors that this patient have going into surgery of developing serious complication – spinal cord ischemia, and do they differ in open versus endovascular repair.
6. Is there more chance of developing the complication with open repair or endovascular treatment of the aneurysm?

Case continuation:

Patient was emergently taken to the operating room. His BP was in his usual range of 120/75 and heart rate was 60. His pain was controlled with narcotics. Standard ASA Monitors were placed, A line and 2 large bore IVs and patient was prepped for the placement of spinal drain to enable drainage of CSF in order to control spinal perfusion pressure. Drain was placed using 14 G Touhy needle in the L3/L4 intervertabral space. Procedure went smoothly without complications.

After spinal drain placement, patient was induced with narcotic, propofol and Succinylcholine. Double lumen ETT was placed and L lung isolated successfully to ease surgical exposure. Anesthesia was maintained with Isoflurane, Propofol and Remifentanil infusion.

Spinal drain was placed at the level of the lumbar spine and CSF pressure was transduced. During the procedure CSF was drained as needed.

Monitoring of SSEP and MEP was started at this point.

Questions:

1. What are the techniques used to try to prevent SCI?
2. What is the rationale for using CSF pressure monitoring and drainage? Spinal cord perfusion pressure equation?

3. Are SSEP reliable monitor for avoiding motor neuron ischemia?

4. How does your choice of anesthetic technique influence EP monitoring?

5. What is the target BP for this patient? Do you need really tight control of BP in this young patient and why?

6. What can surgeon do to decrease the risk of SCI? What about EVAR?

Case continuation:
Aorta was exposed and left heart bypass started at flows of 2-3 L/min. Body temperature was allowed to fall and then maintained at 32°C.

Blood pressure was maintained in the 120-130 mmHg systolic range.

CSF was intermittently drained to maintain CSF P at 10-12 mmHg. Total of 250cc of CSF was drained during the procedure.

Dacron Hemashild graft was used to replace the diseased part of the aorta. Surgeon reimplanted T7-L1 intercostal arteries, as well as celiac, superior mesenteric and both renal arteries.

In the middle part of the procedure, BP suddenly dropped to the 80 systolic. SSEP and MEP tracings changed and ischemia was suspected. Phenylephrine was used to increase the pressure and patient was back to 120’s within few beats. As the pressure recovered, SSEP and MEP tracing came back to baseline.

Questions:

1. How will SSEP and MEP tracing show ischemia

2. What is the rationale for L heart bypass

3. How will cross clamp change perfusion of the spinal cord
4. How do surgeons address collaterals in open vs endovascular procedures?

Case continuation:

Procedure continued and ended uneventfully. There were no further changes in BP or SSEP/MEPs.

Patient was transported to the ICU where CSF pressure continued to be monitored and CSF drained to keep it at 10-12 mmHg. He woke up about 10 h after ICU arrival without any neurologic deficit.

During first 8 postoperative hours 80cc of CSF were drained. In the next 8 hours, 250cc of CSF was drained to keep the pressure at 10mmHg.

At this time patient suddenly became unresponsive with dilated pupils. CSF drainage was checked and noticed that it became blood tinged.

Questions:

1. What happened so suddenly to the patient that was fully awake and neurologically intact until then?

2. What is the next step? CT scan. Next? Is this an emergency?

3. What could we have had done to avoid this complication?

Case Continuation:

CT scan showed moderate hydrocephalus, subarachnoid hemorrhage, large midline cerebellar hemorrhage with extension to the fourth ventricle. Ventriculostomy tube was placed by a neurosurgeon.

Spinal drain was removed and epidural blood patch performed. Patient stayed in ICU for several days and his neurological status recovered to the baseline. He was discharged home in good condition.
Discussion:

Aneurysm or dissection in the descending and thoracic abdominal aorta (TAA) occur in 9 in 100000 women and 16 in 100000 men. Left untreated, aneurysms above 6 cm have 14% annual risk of rupture, dissection or death. They increase with time and growth accelerates with increase in size. Rupture is a catastrophic complication and survival is very low. In a series of 1004 patients, 5 year mortality was 39% for treated versus 87% for untreated patients.

Elective surgery of these aneurysms has clear benefits but it is tied to serious complications. Here, complication rate exceeds the rate in other elective surgeries.

Open surgical procedures for TAAA and dissection have 8-28% of the complication rate while the occurrence is diminished but still significant at 4-7% in endovascular repair procedures.

Spinal cord ischemia (SCI) is a well recognized complication of both operative and endovascular procedures on the TAAA. It can lead to paraplegia and paraparesis both transient and permanent. Neurological complications are directly related to mortality. Patients with persistent paraplegia, stroke and spinal ischemia have significantly higher mortality rate compared to patients without these complications.

Multiple risk factors for SCI have been identified, attributed to both the patient and the procedure.

A simple-risk score was developed for predicting poor outcome in patients being considered for open DTA or TAA repair. One point is added for each for the following: age >60, history of cerebrovascular disease, Crawford extent 2 or 3 repair and emergent/acute rupture or dissection. Estimated risk of poor outcome was shown to increase from 7% with zero risk factors to 60% with three or more risk factors.
Other risk factors for poor outcome include:
- hypotension perioperative
- open repair
- acute rupture
- aortic dissection
- longer cross clamp time (OR)
- failure to reimplant segmental arteries (OR)
- prior distal aortic surgery (sacrificed collaterals)
- anemia
- diabetes
- injury to the external iliac artery (collaterals disrupted) ER
- occlusion of the LSC or hypogastric arteries (ER)

Extent of the aneurysm has been identified as a major risk of morbidity and mortality.
Descending aortic and TAAA are classified by Crawford’s classification depending on the extent of the aneurysm.

Type I, from below the left subclavian artery to above the celiac axis, or opposite the superior mesenteric and above the renal arteries.
Type II, from below the left subclavian and including the infrarenal abdominal aorta to the level of the aortic bifurcation. Type III, from the sixth intercostal space tapering to just above the infrarenal abdominal aorta to the iliac bifurcation.
Type IV, from the 12th intercostal space, tapering to above the iliac bifurcation.
Type V, from the sixth intercostal space, tapering to just above the renal arteries.
Highest risk for spinal cord ischemia (SCI) exists in Crawford II, then I and III, and it is lowest in Crawford IV.

In order to better understand the problem of SCI, it is useful to review blood supply to the spinal cord:
Main supply comes from one anterior spinal artery that is formed from both vertebral arteries, and two posterior spinal arteries also formed from vertebral arteries.
Segmental contribution comes from: internal iliac, middle sacral, mesenteric, lumbar, intercostal arteries and the biggest, Artery Of Adamkiewicz

Numerous techniques are used to decrease the risk of SCI during repair:
They are used with the rationale of minimizing SCI time, increasing tolerance to ischemia and augmenting spinal cord perfusion.

**To minimize SCI time**, area below the clamp can be perfused using left heart bypass or passive shunt (Gott). Selective visceral perfusion and segmental reconstruction (moving the clamp) are used as well.
Gott shunt is a heparin coated tube shunting blood from proximal aorta or LV to distal aorta,
Left heart bypass is used for active diversion of blood from left atrium to distal aorta. It unloads LV, and regulates the temperature as needed.

**To increase tolerance to ischemia** patients are kept at mild hypothermia of 32C unless circulatory arrest is needed when they are cooled to 18C.
Hypothermia consistently protects neuronal tissue from ischemia by decreasing metabolism and stabilizing the cell membrane.
Selective hypothermia by spinal cord cooling where epidural catheter delivers cold saline has also been described.
Pharmacologic protection of the spinal cord is sometimes used but no real efficacy has been proven. Methylprednisolone 1 g, Mannitol 12.5-25 g, Mg 1-2 g, Lidocaine 100-200 mg, thiopental 0.5-1.5 g iv are the drugs usually used.
To augment spinal cord perfusion following procedures are used:
- increasing systemic BP to mean pressure (MAP) of at least 80mmHg. If ischemia persists, increasing MAP by 5 mmHg increments.
- decreasing CSF pressure by draining CSF fluid to the pressure of 10 mmHg
- maintaining spinal cord perfusion pressure at least 70 mmHg (spinal cord perfusion pressure = MAP - lumbar CSF pressure or CVP)
- reimplanting intercostal and lumbar arteries
- preserving flow in left subclavian artery

Keeping adequate MAP is achieved with usual pressors and volume. Hypotension from bleeding needs to be treated promptly. Hypotension can also mean early sign of cord ischemia due to autonomic instability.

Early detection of SCI is crucial in attempt to avoid spinal cord injury. Ischemia at normothermia is tolerated for about 5 min, when neuronal injury occurs. Despite hypothermia and efforts to preserve oxygen delivery by preserving high hemoglobin level, O2 level, perfusion pressure, ischemia during and after procedure is still a constant threat. The earlier the ischemia is recognized, the sooner the therapy can be implemented and injury to the cord is reduced.

When patient is awake, motor strength and sensation can be examined directly. Any neurologic deficit is considered ischemia until proven otherwise. There is no time for imaging studies if delayed ischemia is suspected.

Under general anesthesia, somatosensory evoked potentials (SSEP) and motor evoked potentials (MEP) are used to detect changes in spinal cord function following acute ischemia as well as to assess efficacy of the treatment.

Following changes in the evoked potentials, adequate early intervention is possible if ischemia is shown. Interventions include increase in perfusion pressure (MAP, CSF drainage), reimplantation of critical vessels, increase in oxygen carrying and delivery and decrease in consumption. Transient intraop SSEP/MEP changes might also identify patients with risk of late SC injury and delayed paraplegia.

Sensitivity and specificity of these techniques is variable. SSEP and MEP are recorded before, during and after cross clamp and amplitude, latency, and disappearance-before, during and after cross clamp are compared.
For SSEP monitoring electrodes are placed on the skin above peripheral nerves. Electrical stimulation through the electrodes generates action potential. Recording electrodes are at lumbar plexus, brachial plexus, spine, brainstem, cortex and thalamus.

For MEP monitoring, electrodes are placed over motor cortex, transcortical electric stimulation with multipulse electrical stimulation is delivered and myogenic potentials are recorded in extremity muscle groups.

Findings that point to increased risk of ischemia are MEP that disappear sooner or are absent longer.

Anesthetic has to be adjusted during evoked potentials monitoring since high dose of gasses, thiopental or propofol attenuate SSEP signal and muscle relaxants disable MEP.

Advantage of SSEP monitoring over MEP is that is easy to do and interpret. SSEP monitoring is not enough to recognize spinal ischemia in anterior portion since motor deficit can exist without sensory deficit.

Changes in the response to stimulation such as attenuation or flat line are used as a signal of SCI. It is also possible that the change is induced by local nerve ischemia from any other cause, isolated limb malperfusion due to cannulation, vessel obstruction by emboli, dissection and low perfusion after cross clamping. In acute stroke there is selective loss of cortical signal, and changes in both upper and lower extremities.

CSF drainage is used to keep CSF pressure at 10-12 mmHg in order to provide adequate perfusion pressure to the spinal cord. Efficacy of this procedure is supported by multiple data. Catheter is placed in the subarachnoid space in lumbar area, CSF pressure is measured and CSF drained continuously or intermittently. Daily production of CSF is 400-600cc. Circulating volume is 150 cc in the brain and spinal cord.

Complications of CSF drainage occur in 1.5-5% of cases. They include infection (meningitis), bleeding, intracranial hypotension followed by intracranial bleeding, spinal headache, persistent CSF leak, and direct cord injury. Intracranial hypotension causes displacement of the brain caudally, stretching of the sensory receptors in the dural sinuses and headache.
Most serious complication is intracranial hemorrhage with death rate of 40-50%. Blood tinged/bloody CSF drainage is very sensitive indication of intracranial bleeding with or without neurological signs. Bleeding is located in cerebellar, cranial and subdural areas. Most common location is cerebellum due to caudal displacement of the brain. Hemorrhage may be due to venous engorgement, stretching and tearing of venous sinuses.

Risk factors for cranial hypotension and bleeding are multiple: traumatic drain insertion, volume and speed of CSF drainage, size of the veins that rupture, venous engorgement, preexisting intracranial pathology (old subdural hematoma, brain atrophy, old trauma, alcoholism), coagulopathy, possible hypertension, high CVP.

Precautions such as continuous CSF pressure measurement, controlled drainage (15cc/h), occluding drain for 24 h prior to removal, assessment of coagulation status, placing the drain prior to surgery, maintaining closed system are taken to decrease complications. Subdural hematoma can occur with a delay from hours up to 5 months postoperatively.
Further reading:


Greenberg RK et al. Contemporary Analysis of Descending Thoracic and Thoracoabdominal Aneurysm Repair A Comparison of Endovascular and Open Techniques: Circulation 2008; 118: 808-817


