

Problem-Based Learning Discussion

Spinal Cord Protection during Descending Thoracic Aortic Surgery

Benjamin Drenger, M.D.

Associate Professor of Anesthesia
 Director, Cardiothoracic Anesthesia
 Department of Anesthesiology and CCM
 Hadassah University Hospital
 Jerusalem, Israel

Christopher J. O'Connor, M.D.

Associate Professor
 Director, Cardiothoracic Anesthesia
 Department of Anesthesiology
 Rush-Presbyterian-St. Lukes Medical Center
 Chicago, IL

OBJECTIVES

At the conclusion of this PBLD session, the participant will be able to:

1. Describe the blood supply of the spinal cord and the incidence of spinal cord injury after DTA surgery.
2. Understand the etiology and pathophysiology of spinal cord injury/ischemia during DTA surgery.
3. Recognize patients and aortic pathology associated with the greatest risk of spinal cord injury. Describe intraoperative techniques to monitor spinal cord function and discuss their utility.
4. Understand the available clinical methods of spinal cord protection and the scientific data supporting or refuting their efficacy.

CASE PRESENTATION

The patient is a 70-year-old man with a history of hypertension, heavy tobacco use, and CAD who presents for a thoracoabdominal aortic aneurysm resection. He presented 8 weeks prior to admission with cough; evaluation revealed a 5-cm descending thoracic aortic aneurysm just distal to the left subclavian artery and a second, separate 7-cm aneurysm distal to the first lesion ending above the celiac artery. Cardiac assessment revealed significant CAD and he underwent a CABG 6 weeks prior to admission, complicated only by postoperative atrial flutter. His medications include Rythmol, Digoxin, Albuterol inhaler, aspirin, and diltiazem. His blood pressure was 135/80 in both arms and his heart rate was 80 BPM (a-flutter). Labs included a hemoglobin of 13.6 g/dL and a creatinine of 1.3 mg/dL. The ECG revealed atrial flutter at a rate of 100.

QUESTIONS:

1. What are the potential implications of the patient's presenting symptoms?
2. Which specific aortic lesions are at highest risk for postoperative paraplegia/paresis? What is his expected risk of paraplegia?
3. What is the difference between immediate-onset and delayed-onset paraplegia?
4. What cardiovascular and neurologic monitors are available for intraoperative monitoring? Which are appropriate for this procedure?
5. Should a CSF catheter be placed, and if so, how much CSF should be removed? What is the target CSF pressure?

CASE

Right radial artery and 14-gauge intravenous catheters are placed and anesthesia is induced with 15 mcg/kg of fentanyl/ 0.1 mg/kg of midazolam. A 41-Fr right-sided DLT and TEE probe are placed and a pulmonary artery and 12-Fr triple lumen high-flow catheter are inserted in the right and left internal jugular veins, respectively. Bronchoscopy confirms proper DLT position; the left MSB appears pulsatile. Three gms of magnesium and 500 mg of Solu-medrol are given IV before clamping. The patient is positioned, a left thoracotomy performed, femoral-femoral CPB established, and the DTA clamped. The blood pressure fluctuates between 110-170/60-85 mmHg during aortic clamping, with PA pressures varying from 15-35/9-19 mmHg. Sevoflurane is used at

low doses throughout and no antihypertensives are required. The surgeon instructs the perfusionist to "keep the patient warm". A sequential clamping technique is used and all intercostal vessels are oversewn. The cross-clamp time is 116 min. and unclamping is uneventful with stable pressures. The patient awakens 1 hr later and is unable to move his lower extremities. He has no neurologic recovery.

QUESTIONS:

6. What is the "safe" duration of descending thoracic aortic clamping?
7. What are the advantages and disadvantages of left-atrial and femoral-femoral bypass? Do they reduce spinal cord ischemia and the incidence of paraplegia?
8. Should you keep the patient warm, or let the temperature drift to 33-34°C? Is deep hypothermic circulatory arrest a reasonable surgical option?
9. What medications can be administered to reduce spinal cord ischemia? Do they work?
10. Is nitroprusside, or nitroglycerin, dangerous? How should blood pressure be controlled?

DISCUSSION:

The incidence of spinal cord (SC) injury ranges from < 2% for coarctation repair to 43% after surgery for extensive thoracoabdominal aortic resection. Aneurysm rupture or acute dissection increases the risk of paraplegia. 50% of patients with immediate paralysis never recover neurologic function. A single anterior spinal artery supplies the anterior cord and receives blood supply from several intercostal arteries (among other vessels), the largest of which originates at T₉-L₃. The variable origin of these feeding vessels and the segmental supply of the anterior spinal cord account for the varied incidence of paraplegia after aortic surgery.

Two patterns of injury are seen: immediate-onset and delayed-onset paraplegia; the latter occurs in ~ 30% of cases and is seen from 1-30 days postoperatively. It is secondary to postoperative hypotension, SC edema, reperfusion injury, or thromboembolic occlusion of reattached intercostal vessels. The risk of SC injury is determined by 4 major events: (1) reduced SC blood flow during aortic clamping; (2) permanent loss of SC blood flow due to sacrificed or thrombosed critical intercostal vessels; (3) reperfusion injury; and (4) postoperative SC hypoperfusion.

The etiology of SC injury includes: (1) duration of aortic clamping, esp. > 30 minutes, has shown a linear correlation with the risk of paraplegia; (2) more extensive aneurysms, such as Crawford type I, II TAA, are associated with the highest incidence because of widespread intercostal artery involvement and the prolonged clamp time required; (3) failure to reimplant critical intercostal vessels, although this is somewhat controversial; (4) intra- and postoperative hypotension both reduce distal SC perfusion via collateral vessels (a possible mechanism of the theoretical nifedipine-associated risk); (5) presence of acute rupture or dissection increase the risk significantly; hence, part of the high risk associated with emergency procedures. (6) An acute increase in CSF pressure and significant decrease in spinal cord blood flow upon application of the AXC both decrease spinal cord perfusion pressure. The mechanism of increase in CSFP is a combination of proximal arterial hypertension, venous overload and sympathetic discharge, all of which contribute to spinal cord venous engorgement and consequently, a rise in CSFP.

Several methods of SC protection are advocated, but few have been prospectively investigated as isolated techniques to improve SC blood flow and reduce paraplegia. (1) Distal perfusion techniques may improve collateral flow to the distal aorta and SC and *appear* to reduce the incidence of SC injury, although some surgeons still advocate a simple clamp and sew approach. The decision on which additional methods of spinal cord protection to employ is largely dependent on whether partial bypass is used. Induced hypothermia and hemodynamic control are facilitated by the use of left-sided bypass. Several institutions try to equilibrate mean proximal and distal pressures by adjustment of pump flow and volume and vasopressor administration. Minimum mean distal arterial pressures of 50 torr are suggested to maintain adequate spinal cord blood flow, while maximal proximal MAP's are maintained at or above 100 mmHg, although limited human data are available regarding the impact of specific distal flow rates and pressures on outcome;(2) The use of left-sided heart or femoral venoarterial bypass enables controlled

induction of moderate hypothermia and adequate re-warming prior to clamp release. A team not using distal bypass techniques will have to either allow intentional passive hypothermia (32-34⁰), or apply selective cooling of the spinal cord by cold saline irrigation of either the isolated mid-thoracic aortic segment and renal arteries, or the epidural space itself; (3) CSF drainage theoretically improves SSCP, but may only be effective when used with other adjuvants; (4) Limitation of the duration of aortic clamping; (5) Pharmacotherapy: a variety of pharmacological agents have been investigated but their efficacy remains unproven. They include magnesium, steroids, mannitol, calcium and sodium channel antagonists, and others. Preliminary data suggest that endovascular repair may be associated with less paraplegia and reduced morbidity when compared to conventional surgery.

Evoked potential monitoring with SSEP may detect SC ischemia, but they only monitor the more ischemia-resistant posterior SC neurons, not the susceptible ischemia-sensitive anterior horn cells. Also, lack of sensitivity and specificity and technical problems (impact of cold, anesthetics, etc) have limited their utility and led to use of epidural evoked potentials or motor-evoked potentials; some investigators have shown that these techniques can effectively guide the reattachment of critical vessels. Of these, transcranial motor-evoked myogenic potentials have shown a very short delay in the appearance of ischemic changes, which makes them particularly useful in intraoperative real-time guidance of the surgical team.

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