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CASE REPORT

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Successful reversal of recurrent spinal cord ischemia following endovascular repair of a descending thoracic aortic aneurysm

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ABSTRACT

Despite recent advances in technique, spinal cord ischemia remains one of the most dreaded complications of thoracic aortic surgery. Recently, it has been suggested that thoracic endovascular aortic repair may decrease the risk of paraplegia. We present a case of delayed paraplegia following thoracic endovascular aortic repair that was successfully reversed on 3 separate occasions in the same patient. This highlights the importance of vigilant clinical assessments, efficient multidisciplinary teamwork, and maintenance of the determinants of spinal cord perfusion following endovascular thoracic aortic intervention.

Keywords: *vascular surgery, anesthesia, aneurysm, endovascular aortic repair, spinal cord ischemia, spinal cord paraplegia.*

A 77 year old male, with no previous thoracic or abdominal surgeries, presented with dysphagia. Barium swallow studies demonstrated external compression of the lower esophagus. A computed tomography (CT) scan revealed a 7 cm thoracoabdominal aortic aneurysm extending from the inferior half of the descending thoracic aorta to the renal arteries (*Figure 1*), with patency of the left subclavian, renal, mesenteric and iliac arteries.

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Rather than a traditional open thoracoabdominal repair, a 2-stage hybrid surgical approach was used. First, visceral vessel debranching of the celiac and superior mesenteric arteries was carried out with a bifurcated surgical graft from the left iliac artery. Three months later, the patient underwent the second stage with thoracic endovascular aortic repair (TEVAR) via femoral artery access. Given multiple risk factors for spinal cord ischemia (SCI) a pre-operative lumbar cerebrospinal fluid (CSF) drain was placed, transduced at the level of the right atrium, and set to drain at 10 cmH2O.
Using a combination of pharmacologic mean arterial pressure (MAP) augmentation and

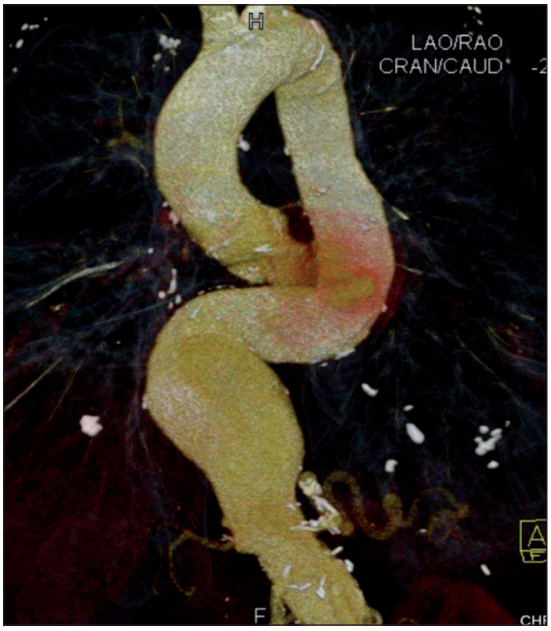

Figure 1 - *Pre-operative thoracoabdominal contrast enhanced computed tomography scan (computer 3D rendered) demonstrating oxbow shaped anuerysm extending from T8 to L2.*

CSF drainage (limited to < 10 mL/hr, for a total of 6 mL for the case), spinal cord perfusion pressure (SCPP) was maintained at 70-80 mmHg. Intra-operative neurophysiologic monitoring was not used. Two stent grafts were deployed with the distal landing zone immediately proximal to the renal arteries.

Emergence from anesthesia was expedited with extubation and confirmation of normal neurologic status in the operating room. The patient was then transferred to the Cardiovascular Intensive Care Unit (CVICU) and managed with our institution's standardized protocol as summarized below. On the first post-operative day (POD 1) the patient experienced sudden loss of function in his lower extremities with 1-2/5 motor power at a spinal level below L1. At this time MAP was 85 mmHg, central venous pressure (CVP) was 9 mmHg, and oxygen saturation was 98%. The attending intensivist and CV surgeon were immediately notified. The CSF drain was lowered to 10 cmH20 and 14 mL drained, MAP was increased to 110 mmHg with norepinephrine, a bolus of 1000 mL of Lactated Ringers was given, and red cells transfused to achieve a hemoglobin of > 10 g/dL. Complete neurologic resolution was obtained 58 minutes after onset of symptoms.

The following day the CSF drain was returned to 15 cmH2O and the MAP was reduced in a stepwise fashion. Once the MAP decreased to 85-90 mmHg (with a CVP of 10 mmHg) a the patient's neurologic deficits returned to a lesser extent but were eliminated solely by raising the MAP back to 95-100 mmHg with norepinephrine where it remained for the next 2 days with decreasing levels of pharmacologic support. By POD 4 the patient was maintaining an unsupported MAP of 90 mmHg and had no further neurologic events. Given that no CSF had drained in the previous 36 hours the decision was made to remove the patient's drain following administration of fresh frozen plasma to correct an International Normalized Ratio (INR) of 1.5. Unfortunately the patient suffered transfusion related anaphylactic shock dropping his MAP to 40 mmHg and return of his lower extremity paralysis. The patient was emergently intubated, epinephrine infusion started, bolused over 3000 mL of Lactated Ringers and 20 mL of CSF drained. Fortunately the patient had full neurologic recovery within 35 minutes and suffered no further events as all pharmacologic support and CSF drain were discontinued the next day.

While in hospital oral antihypertensive agents were introduced cautiously at low doses to maintain MAP at 85-90 mmHg. At 6 month follow-up, a CT scan demonstrated a well-positioned stent graft (*Figure 2*). The patient was active, his blood pressure was stable at his discharge values, and he had experienced no recurrence of any lower extremity weakness.

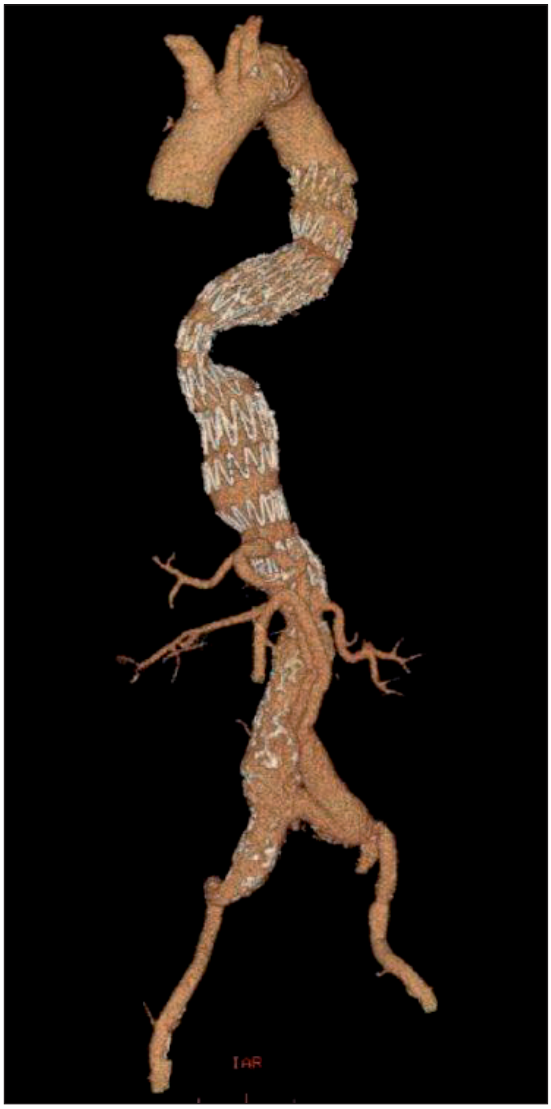

Figure 2 - *Post-op 3D-computed tomography scan image illustrating good stent graft coverage & patent visceral bypass grafts from left iliac artery.*

DISCUSSION

Paraplegia due to spinal cord ischemia is a devastating complication of thoracoabdominal aortic surgery (1, 2). Rates of neurologic deficits following open surgery are between 5% and 16% while those following TEVAR appear to be lower, between 2.5% and 7% (2-6). Previously identified risk factors for paraplegia following TEVAR, which include prior abdominal aortic surgery, increased stent graft length, coverage of the lower thoracic aorta (below T6), left subclavian arterial coverage, trauma to the internal iliac artery, perioperative hypotension, female gender and combined hybrid vs. staged procedure(1, 3, 5). Magnetic resonance angiography (MRA) may also aid in detecting higher risk patients with a poor collateral network (7).

Our patient had several risk factors for SCI: previous interventions to the abdominal aorta and left iliac artery as well as extensive graft coverage, including below T6 spinal level. This case report illustrates the precarious nature of spinal cord perfusion following surgery on the thoracic aorta, especially in high risk individuals. It also emphasizes the importance of optimizing the determinants of spinal cord perfusion and oxygen delivery to prevent or reverse neurologic deficits. These include MAP augmentation with pharmacologic agents, CSF drainage, fluid administration, and avoidance of high CVP, anemia, or hypoxia (4, 8-10). In our case the augmentation of MAP was primary in reversing all 3 episodes of SCI, highlighting its significant contribution to spinal cord blood flow. Finally, the importance of vigilant monitoring and effective multidisciplinary communication in detecting SCI and initiating therapies promptly cannot be overlooked.

The exclusion of the use of pre-operative MRA or intra-operative neurophysiologic monitoring could be seen as limitations to our management. Intra-operative evoked potential monitoring has shown promise in several studies (4, 11, 12). Unfortunately this was unavailable at our institution, therefore our strategy was to maintain SCPP at 70-80 mmHg following stent graft deployment and minimizing any delays to emergence from anesthesia. Given that our pa-

tient was being treated as high risk for SCI and that the territory of stent graft coverage was determined by the aneurysm, we felt that MRA would not provide any new information that would alter management.

Allowing MAP to be sequentially decreased from 110 mmHg towards 90 mmHg following the first episode of SCI could be questioned. There is evidence that the percentage of mean arterial pressure that is transmitted to the collateral network increases linearly beginning 24 hours after radicular artery sacrifice (13). Furthermore, since artificial augmentation to a high MAP can be detrimental, for example resulting in myocardial or bowel ischemia, we felt it was reasonable and safe to begin weaning towards a goal of 90 mmHg after the patient had been neurologically stable for several hours.

A final point of debate would be the decision to transfuse our patient with fresh frozen plasma to correct his INR of 1.5 prior to removing the CSF drain. Currently there are no evidence based guidelines for management of coagulopathy in patients with CSF drains. The current American Society of Regional Anesthesia and Pain Medicine guidelines would suggest removing an indwelling catheter only if the INR is less than 1.5 (14).

However these guidelines are based on the pharmacodynamics of Coumadin and case reports which generally involve spinal techniques. It is arguable whether these same principles can be safely applied to the aortic surgery population. INR's are elevated secondary to medical illness or dilutional coagulopathy, there may be co-existing coagulation abnormalities such as platelet dysfunction, and large intrathecal catheters are in place. Clearly attempting to correct coagulopathy has its own inherent risks, therefore clinicians must weigh the risks and benefits carefully in each case.

Based on existing literature and our local experience (including this case) our institution's current strategy for TEVAR includes (2, 4, 8-10):

- CSF drain insertion preoperatively for all patients considered to be high risk, though medical teams need to be aware that CSF drains may be associated with complications such as prolonged CSF leak, catheter fracture, infection, neuraxial hematoma and intracranial hemorrhage (15).
- Intra-operatively: CSF drain set at 10 cmH2O, maximum drainage of 10 cc/hr and MAP > 90 mmHg (or SCPP > 70 mmHg if drain transduced).
- Fast-track anesthesia with expedited emergence and early neurologic assessment.
- Admission to Cardiovascular Intensive Care Unit (CVICU) with one-to-one nursing care and hourly neurologic assessments.
- Post-operatively if neurologically intact: CSF drain is set at 15 cmH2O and MAP maintained at 90 mmHg for 48 hours. CSF drain capped on POD # 1 (Post operative day number 1) and left in situ for 24 hours prior to removal.
- Post-operatively if neurologically compromised: Intensivists, CV surgeon and CV anesthesiologist informed immediately. CSF drain is set at 10 cmH2O, correction of hypoxia and anemia, volume expansion (unless CVP elevated) and MAP pharmacologically augmented until neurologic status improves.
- Refractory neurologic deficit despite above interventions: further investigations (CT head, Computed Tomography Angiography, Magnetic Resonance Imaging, Electromyography, etc) and neurology consultation if appropriate.
- CSF drainage limited to 10 cc/hr at all times to minimize the risk of subdural hematoma. Rate may be increased temporarily if SCI refractory to all therapies

and alternative causes for neurologic deficit are eliminated.

Our knowledge of the pathophysiology, detection and treatment of SCI is constantly evolving. This case of multiple episodes of reversible paraplegia highlights the importance of vigilant clinical assessments, efficient multidisciplinary teamwork, and maintenance of the determinants of spinal cord perfusion following endovascular thoracic aortic intervention.

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