Brown-Séquard Syndrome Following Removal of a Cerebrospinal Fluid Drainage Catheter After Thoracic Aortic Surgery

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Neurological deficit remains a devastating complication of thoracic aortic surgery despite advances in methods to protect the spinal cord from ischemia. Various techniques have been used, including the combination of cerebrospinal fluid (CSF) drainage and distal aortic perfusion to decrease the incidence of postoperative neurological deficit. These deficits are usually bilateral and result in paraplegia. In this case report we present a patient with Type B aortic dissection and thoracoabdominal aortic aneurysm repair with insertion of a lumbar CSF drainage catheter. Postoperatively, the patient developed unilateral neurological features consistent with Brown-Séquard syndrome after removal of the CSF catheter. The lumbar cerebrospinal fluid catheter was reinserted and the CSF was drained. Medullary T6-7 signal abnormalities were seen on spinal cord magnetic resonance imaging, and we suggest that the spinal cord suffered a direct injury during catheter removal. The patient had an uneventful recovery.

Case Report

A 65-yr-old 91-kg male patient presented for elective repair of a chronic aortic dissection (Stanford type B) and thoracoabdominal aortic aneurysm. The superior aspect of the aneurysm began just distal to the arch of aorta (maximal aneurysmal dilation was 61 mm just distal to the left subclavian artery). It tapered to 45 mm at the diaphragmatic cross, 40 mm as it entered the abdomen, and 34 mm at the celiac artery. The type B dissection extended from just distal to the origin of the left subclavian artery to both common iliac arteries. After general anesthesia was induced, a lumbar intrathecal catheter was inserted after the first attempt at the L3-4 interspace via a 14-gauge Tuohy needle. Correct positioning was confirmed by the flow of clear CSF. The catheter was advanced up to 17 cm into the intrathecal space, secured with adhesive dressing, and adjusted to CSF overflow at 10 mm Hg. Intraoperatively the intercostal vessels were ligated because the aorta was dissected and could not be reimplemented. A size 28 Hemashield graft was anastomosed to the aorta (from the left subclavian artery to the celiac artery).

In the intensive care unit, tracheal extubation was successful within 8 h of admission. The oxygen saturations (S\textsubscript{a}O\textsubscript{2}) after extubation were between 92% and 97% on oxygen via nasal cannulae at 6 L/min. The pre-extubation P\textsubscript{a}O\textsubscript{2} was 81.3 mm Hg and S\textsubscript{a}O\textsubscript{2} was 97% on F\textsubscript{io}2 of 0.4. The CSF catheter was kept to overflow at 10 mm Hg in addition to hourly 10-mL aspirations of CSF if the pressure exceeded 12 mm Hg. The spinal perfusion pressure (mean arterial blood pressure minus CSF pressure [CSFP]) was maintained at 70–80 mm Hg. Postoperatively, after 16 h the CSF drainage catheter was clamped for 6 h. During the 6 h the CSFP increased to a maximum of 15 mm Hg and the patient could move both lower limbs. After excluding coagulation abnormalities (prothrombin time, 11.7 s; international normalized

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ratio, 1.1; partial thromboplastin time, 36.9 s; and platelet count, 205/mm³), the CSF drainage catheter was removed uneventfully.

Within 1 h of catheter removal, the patient complained of loss of motor function in the left lower limb. Neurological examination revealed motor power of 1/5 in the hip, knee, and ankle joints of the left side. There was no sensory loss on the left side and no motor loss on the right side. However, there was definite moderate loss of temperature (ice) and pain (pinprick) on the right side compared with the left side from the hip down. The CSF drainage catheter was reinserted in the L3-4 space. The CSFP at this time was 15 mm Hg. The CSF catheter was then kept to overflow at 10 mm Hg in addition to hourly aspirations of CSF if the pressure exceeded 12 mm Hg. A spinal cord magnetic resonance imaging (MRI) scan detected left-sided T6-7 intramedullary signal abnormalities and excluded epidural/spinal hematoma (Fig. 1). A formal neurology consultation was obtained and clinical findings were confirmed. Over the next 4 days motor strength improved and the patient regained full neurological function. The CSFP was maintained <10 mm Hg. The CSF drainage was 145, 238, and 102 mL, respectively, over the next 3 days. The CSF drainage catheter was successfully removed on the fourth day after temporarily clamping for 6 h. The patient subsequently had an uneventful recovery.

Discussion

BSS is a rare neurological condition characterized by a lesion in the spinal cord that results in hemiparaplegia on one side of the body and hemianesthesia on the opposite side (4). Complete hemisection causing classic clinical features of pure BSS is rare. However, incomplete hemisection causing variations on BSS is more common.

Our patient had neurological features consistent with BSS after removal of the CSF drainage catheter. This syndrome has not been reported with the use of intrathecal catheters (1,3,5,6). The possible causes of this delayed neurological deficit are spinal cord ischemia with increased CSFP secondary to hypotension, intercostal artery ligation, or embolization, pulmonary complications and hypoxia, and direct injury to the spinal cord.

For the spinal cord to become ischemic there must be a decrease in spinal perfusion pressure. In this case, the mean arterial blood pressure was more than 80 mm Hg postoperatively and the CSFP was maintained <10 mm Hg. Given that there was no neurological deficit even after the catheter had been clamped for 6 hours, it is unlikely that there was significantly decreased perfusion pressure before CSF catheter removal. The maximum CSFP before catheter removal was 15 mm Hg and similar to the CSFP after reinsertion of the catheter. However, some CSF volume would have been lost during catheter insertion. In addition, if spinal cord ischemia had been the case, the patient would probably have presented with paraplegia.

Intercostal artery embolization, although unlikely because of the absence of reimplantation, cannot be completely excluded. The patient had no pulmonary complications and the SaO₂ was between 92% and 97% on nasal oxygen.

We postulate that, during insertion, the catheter may have become partly coiled in the intrathecal space. During removal, the catheter uncoiled, striking and injuring the spinal cord. This may also explain why the injury was limited to one side, as confirmed by the left-sided T6-7 intramedullary signal abnormalities on MRI. The CSFP of 15 mm Hg was then probably significant enough to cause neurological deficit only to the injured segment, sparing the rest of the spinal cord.

The fact that the neurologic status improved even though the CSFP was not significantly increased suggests that the outcome might not have changed had
the CSF catheter not been reinserted. However, unilaterial neurological deficits have not been previously described in this situation. Thus, CSF catheter reinsertion and CSF drainage was performed immediately, as this is our standard protocol for treatment of any delayed neurologic deficit.

Because the proposed mechanism of T6-7 spinal cord injury is CSF catheter-related and not needle trauma, the site of needle insertion is irrelevant, especially if it is below the L2 vertebra. In addition, our patient did not show the usual features of spinal hematoma, which include pain and tenderness at the affected site with both sensory and motor weakness.

The routine use of CSF drainage is a simple technique to decompress the spinal cord. Placement of the CSF catheters in anesthetized patients has been safely demonstrated, even in patients requiring subsequent heparinization and cardiopulmonary bypass (6,7). There was no risk of hemorrhagic complications, neurologic injury was infrequent, and CSF catheter-related complications resulted in no permanent sequelae. In addition, using a protocol of CSF catheter reinsertion and CSF drainage for delayed neurologic deficit led to a more than 50% improvement in neurologic deficit (1,8) with approximately 40% full recovery. Thus, we do not recommend limiting the use of CSF catheters in thoracic and thoracoabdominal aortic surgery for delayed neurologic deficits.

In conclusion, BSS is a rare neurological disorder that occurred in our patient after removal of a CSF drainage catheter. The most probable cause is direct spinal cord injury during CSF catheter removal. Whether catheter reinsertion and CSF drainage prevented permanent damage remains debatable, but the patient had full neurologic recovery. The use of CSF catheters should not be limited in thoracic aortic surgery and the treatment of postoperative delayed neurologic deficits.

**References**