Successful Management of Both Early and Delayed-Onset Neurological Deficit Following Extent II Thoracoabdominal Aneurysm Repair

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Introduction. Delayed-onset paraplegia is an uncommon but devastating complication of thoracoabdominal aneurysm repair.

Report. We report the successful use of repeat cerebrospinal fluid drainage in the management of both immediate- and delayed-onset (21 days) paraplegia in the same patient undergoing open Type II thoracoabdominal aneurysm repair.

Discussion. Few studies have looked specifically at preventing delayed onset of symptoms. We advocate continued attention to blood pressure management and hydration for the duration of hospital stay and recommend repeat CSF drainage if symptoms occur.

Keywords: Thoracoabdominal aneurysm; Paraplegia; CSF drainage.
It is our practice to wake patients at the end of the operation. They remain sedated but rousable during further management on the Intensive Care Unit enabling continual neurological assessment of spinal cord function. Immediately following the operation it was apparent that the patient was unable to move his lower limbs. Active CSF drainage (−2 mmHg) was commenced. In order to optimise spinal cord perfusion pressure, the epidural infusion was discontinued and a norepinephrine infusion was commenced (target MAP >110 mmHg). Profound paraplegia was evident for 72 hours but by the fourth day power returned to normal. The CSF drain was left in place for a further 48 hours before it was clamped for 24 hours and removed.

There were no other major complications of the operation and the patient began rehabilitation. On two occasions leg weakness on walking was documented by the physiotherapists but this was not clearly distinguished from general weakness following major surgery. During this time CSF continued to leak from the drain site until day 18. He had poor oral intake of food and fluids and his anti-hypertensive agents were frequently withheld to maintain BP within normal limits.

Twenty-one days following the procedure, the patient experienced sudden loss of bladder control and lower limb power (Medical Research Council power scale grade 2). Magnetic resonance imaging of the spinal cord was performed and was normal. The CSF drain was re-inserted and a phenylephrine infusion was used to maintain the MAP >80 mmHg with invasive monitoring. There was a rapid improvement (<1 hour) in neurological function. These measures were continued for 6 days and resulted in further gradual improvement in motor power (MRC grade 4). The patient was discharged on day 38 to the regional spinal injuries unit for further rehabilitation and eight months later was able to walk into the outpatient clinic.

Discussion

Delayed neurological deficit, accounting for 36% of neurological complications in patients undergoing TAAA repair, has been reported up to 91 days postoperatively. The current literature describes a number of peri-operative neuroprotective adjuncts to thoracoabdominal surgery but the spinal cord remains vulnerable. A further insult, such as a period of relative hypotension, can jeopardize the precarious spinal cord perfusion and result in neurological dysfunction. Little attention has been given to preventing delayed complications.

The most important strategy in preventing delayed neurological deficit is limitation of the initial ischaemic injury. This is minimized through re-implantation of spinal arteries with or without motor evoked potentials, reduction of aortic cross clamp time, serial segmental clamping, left heart bypass enabling distal aortic perfusion and reduction of the metabolic demands of the spinal cord using moderate hypothermia. Mean arterial
pressure is maintained >80 mmHg and hypotensive agents, particularly nitrates, are avoided. Despite these measures, a degree of spinal cord ischaemia is almost inevitable so CSF monitoring and drainage, particularly during cross-clamping, are instituted to allow for the resulting cord oedema and optimize perfusion. In combination, these measures have reduced the incidence of neurological complications from extensive aortic surgery from 40% to 3.3%. These recommendations for management relate mainly to the peri-operative period and there is little guidance beyond this point.

This patient had early profound paraplegia suggesting ischaemic or ischaemia-reperfusion injury of the spinal cord and consequent oedematous swelling. In retrospect it would appear that spinal cord perfusion remained precarious but was compensated for by continued leak of CSF from the drain site once the drain had been removed. The subsequent late onset of paraplegia on day 21 was temporally related to healing of the drain site on day 18 which prevented further spontaneous decompression of the CSF. Once the drain site had healed up (after day 18), CSF could no longer be displaced resulting in a rise in CSF pressure compromising spinal cord perfusion. In addition, the patient became slightly dehydrated due to inadequate oral intake of fluids and was intermittently administered his anti-hypertensive medications.

Recommendations aimed at reducing neurological complications focus on intra-operative adjuncts and measures employed for the first 48–72 hours after surgery. The duration of prophylactic CSF monitoring and drainage has not been determined and no studies have assessed beyond 100 hours. This case demonstrates that repeated CSF drainage can be effective in reversing delayed neurological deficits, even 21 days after surgery.

We suggest that close monitoring of hydration and blood pressure control is maintained for the duration of hospital stay with omission of vasodilating antihypertensives where necessary. We would argue that attention to spinal cord perfusion and the factors affecting it should be continued beyond the early post-operative period if the incidence of delayed onset paraplegia is to be reduced further.

References


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