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Importance of Multimodal Spinal Cord Monitoring and Hemodynamic Augmentation during High Thoracic Ventral Dural Tear Repair Using the Posterior Approach

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Abstract

Cerebrospinal fluid (CSF) leakage due to large ventral dural tears (VDT) often requires surgical intervention. Surgical closure of a high thoracic VDT is challenging and associated with high morbidity, especially if it is performed after multiple epidural blood patch (EBP)/fibrin glue injections. A 44-year-old woman was diagnosed with spontaneous intracranial hypotension due to VDT at T1-T2, causing CSF leakage. Multiple EBP and fibrin glue injections failed to treat her symptoms; hence, the patient underwent surgical closure using the posterior approach. The patient was anesthetized using standard anesthetic drugs and was maintained under total intravenous anesthesia to facilitate continuous motor-evoked potential (MEP) monitoring. The surgical course was complicated by bleeding, hypotension, and MEP loss. Continuous MEP monitoring, effective team communication, quick restoration of blood pressure(BP) and BP augmentation with fluid, blood, and vasopressor helped to restore the MEP back to baseline. Hence, the patient recovered without neurological morbidity. This case report highlights the importance of adequate vascular access, multimodal spinal cord monitoring, and BP augmentation during a high thoracic VDT repair.

Keywords

- cerebrospinal fluid leakage
- hemodynamic management
- intracranial hypotension
- multimodal spinal cord monitoring
- ventral dural tear

Introduction

Intracranial hypotension due to spontaneous cerebrospinal fluid (CSF) leak is common in middle-aged females with a female: male ratio of 1.9:1. It is often managed initially with adequate hydration, analgesics, and caffeine.[1–3] Those who fail to respond are treated with a targeted epidural blood patch (EBP)[4]/fibrin glue injection.[5] CSF leaks due to large ventral dural tears (VDT) often require surgery. Surgical closure of high thoracic VDT is technically challenging, especially after multiple EBP/fibrin glue injections, due to increased adhesion and scar formation between the dura and overlying structures,[6–8] which increases the risk of bleeding. This report highlights the importance of continuous intraoperative multimodal spinal cord monitoring and blood pressure (BP) augmentation in patients undergoing high thoracic VDT repair.

Case Report

A 44-year-old American Society of Anesthesiologists-1 woman (height—155 cm; weight—68 kg) presented with

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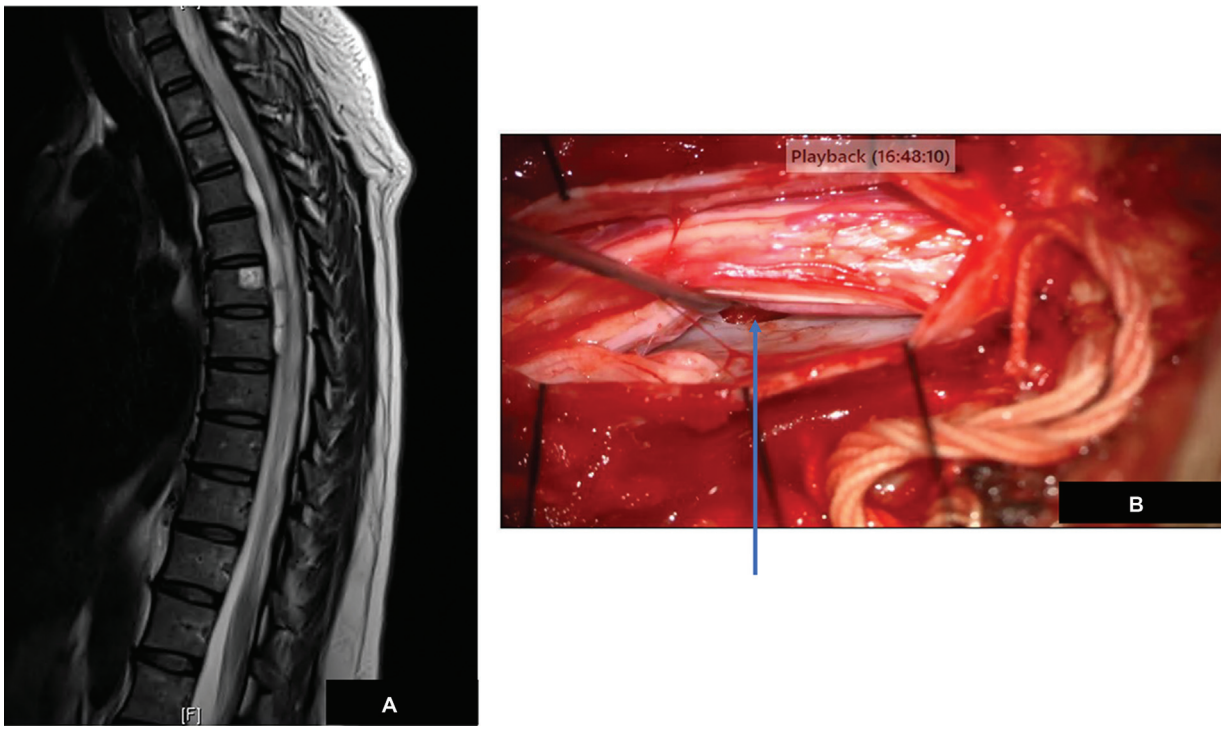

Fig. 1 Magnetic resonance imaging spine sagittal plane (T2-weighted) shows a localized cerebrospinal fluid collection along the ventral aspect of the thecal sac between the T2 and T7 vertebrae (**A**). The intraoperative image shows a ventral dural tear with an osteophyte complex (blue arrow; (**B**).

occipital headache and postural variation associated with nausea and vomiting. There was no history of fever, altered sensorium, posturing, or loss of consciousness. She was investigated elsewhere and diagnosed with spontaneous intracranial hypotension (SIH) due to VDT at T1 to T2 causing a CSF leak. The patient was initially managed conservatively, followed by targeted EBP twice, at 3 months intervals that failed to improve her symptoms. The third time, she was administered a fibrin glue injection, which also failed to seal the leak. Magnetic resonance imaging(MRI) of the spine (T2-weighted-sequence) showed localized CSF collection along the ventral aspect of the thecal sac between T2 and T7, causing a compromise in the anterior thecal sac without any signal change (►**Fig. 1A**). Although the exact site of the dural leak could not be identified, the T1 to T2 level was considered a potential leak site, considering the posterior disc osteophyte complex (►**Fig. 1B**). MRI brain revealed decreased mamillopontine distance(<4 mm), pontomesencephalic angle (42 degrees), slit-like lateral ventricles, and a plump pituitary gland, consistent with SIH. On examination, her vital signs were stable, with normal higher mental function, cranial nerve function, and sensory and motor functions.

She was scheduled for a T1 to T2 laminectomy and VDT repair under continuous motor evoked potential (MEP) monitoring. After placing the standard monitors, she was induced with propofol (3mg/kg), fentanyl (2µg/kg), paralyzed using atracurium (0.5mg/kg), and intubated with 7.0 mm endotracheal tube. A bite block was placed to prevent tongue bite and lip injuries during motor cortex stimulation. A peripherally inserted central line was placed to administer fluid and vasopressors. The patient was turned prone and the head was fixed using a Mayfield head clamp. Anesthesia was maintained with total intravenous anesthesia using propofol (100–150 µg/kg/min) and fentanyl infusion (1–2 µg/kg/hr) and was titrated to maintain a bispectral index between 40 and 50. Baseline MEP responses were recorded (►**Fig. 2A**) using transcranial electrical stimulation of the motor cortex with the help of cork screws placed at C1/C2, according to the international 10 to 20 system. A train of four pulse stimulations with an intensity of 90 to 120V, pulse width of 300 to 400 ms, and interstimulus interval of 3ms was used to obtain the baselines. Five sets of lower limb muscles were monitored bilaterally, with the right and left upper limb thenar muscles as controls. A T1 to T2 wide laminectomy was performed, the dura and arachnoid layers were opened, and the denticulate ligaments were cut, following which one cm VDT was identified (►**Fig. 1B**). Using a posterolateral approach with minimal retraction of the cord, the T1 to T2 disc was emptied extradurally and the central osteophyte was drilled. During this period, brisk bleeding was observed in the dilated epidural venous plexus. Another wide-bore peripheral intravenous cannula was placed on the lower limb. Because the blood loss was acute and massive, the systolic BP dropped to 70 mm Hg for less than 3 minutes. Blood loss was replaced with fluids, packed red blood cells (PRBC), and vasopressors (noradrenaline). Systolic BP was maintained at 90 to 95 mm Hg for approximately 10 minutes because of brisk bleeding. At this point, the lower-limb MEP potentials decreased with a normal control response from the thenar muscle (►**Fig. 2B**). The fractional inspiratory concentration of oxygen was increased to 100%, the second unit of PRBC was rushed, and BP was raised to 140/80

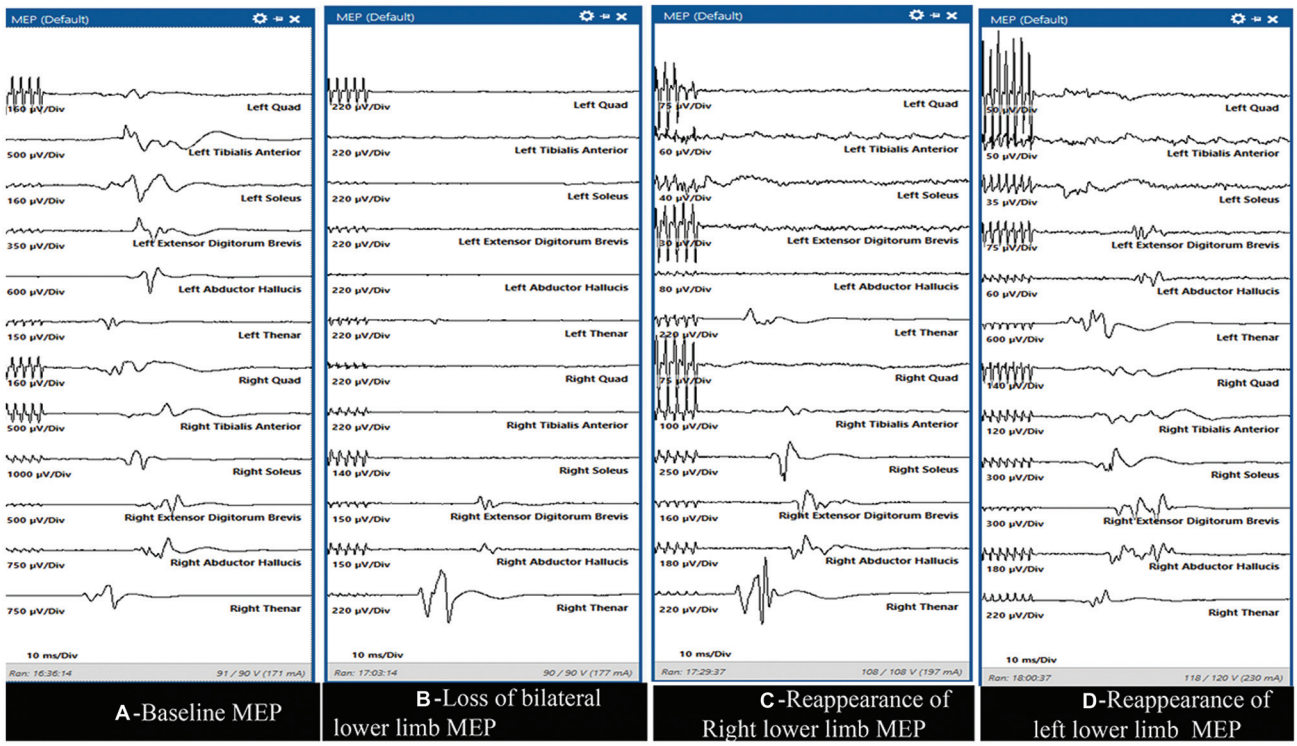

Fig. 2 The baseline motor-evoked potential (MEP; **A**), drop in lower limb MEP potentials with normal upper limb response at the time of hypotension (**B**), improvement in right lower limb MEP (26 min after the initial drop) with a persistent drop on the left lower limb (**C**) with blood pressure augmentation, and further improvement in bilateral lower limb MEP response (57 min after the initial drop) (**D**).

(100) mm Hg and maintained in that range. Blood gas analysis results were within normal limits, with a hematocrit of 27%. The VDT was repaired using 7-0 polypropylene intradurally, and the DuraGen (artificial dura) was placed extradurally ventral to the dural tear. Repeat MEP stimulation after 26 minutes of initial MEP loss revealed an improved MEP response from the right lower limb muscles (►**Fig. 2C**) with no improvement on the left. This could be due to slight retraction of the cord on the left side while closing the VDT. BP was maintained at 140 to 150/80 to 90 (100–105) mm Hg with noradrenaline for the next hour to improve cord perfusion. At the start of skin closure (57 min after the initial MEP loss), a 50% improvement in potential was noted in the left lower limb muscles (►**Fig. 2D**). After extubation, her muscle power was 3/5 in both the lower limbs and 5/5 in the upper limbs. The mean arterial pressure (MAP) was maintained at a high level (85–95 mm Hg) overnight with noradrenaline. Her lower limb power improved to 5/5 after 6 hours. The patient was ambulated after 3 days and discharged on the 6th postoperative day.

Discussion

Repairing high thoracic VDT is challenging because it is associated with increased morbidity. Posterior, anterior, lateral, and endoscopic approaches[3,9,10] have been described to repair VDT. Each approach has its own risks and benefits. With the posterior approach, the risk of cord injury is high because the surgeon must retract the cord to repair VDT. With the anterior approach, sternotomy and its associated morbidities, and the lateral approach, the need for thoracotomy, lung isolation, and their associated morbidities must be kept in mind. The posterolateral approach, with minimal retraction of the cord, is commonly used to repair VDT.

We could not perform MEP stimulation while the surgeon achieved hemostasis by slightly retracting the cord, as stimulation can cause gross movement under the microscope, which hampers hemostasis and aggravates cord injury caused by the retractor. Retrospectively, we felt the use of somatosensory evoked potential (SSEP) monitoring along with MEP could have helped us to identify the drop in potential early as SSEP monitoring can be done continuously without having to stop surgery. Studies have shown that continuous multimodal spinal cord monitoring with MEP and SSEP helps detect impending neurological injury earlier than MEP monitoring alone in high-risk spinal cord surgeries[11] however, there is no recommendation regarding preemptive augmentation of MAP during the critical stage of surgery in patients undergoing high-risk spinal cord surgeries. Fletcher et al recommended various MAP ranges for children with different age groups undergoing adolescent idiopathic scoliosis surgery during the critical stage of spine surgery (spine instrumentation and deformity correction stage) and during the postoperative period.[12] Various studies[13,14] and the American Association of Neurological Surgeons/Congress of Neurological Surgeons recommend a MAP of 85 to 90 mm Hg for 7 days following acute spinal cord injury (level III recommendation).[15] Studies have shown that spinal cord perfusion pressure (SCPP)-guided hemodynamic management improves neurological recovery following

acute spinal cord injury, and SCPP less than 50 mm Hg is associated with poor neurological recovery.[16,17] Subtracting the intraspinal pressure (by placing the catheter in the subarachnoid space) from MAP gives SCPP. We did not consider this option because the MEP recovered by the end of the case.

Quick restoration of BP, maintenance of high MAP, adequate oxygenation, maintaining hemoglobin at 10g/dL, and avoidance of hyperthermia, hypothermia, and acidosis are some of the measures taken to restore the lost MEP response during surgery that prevented morbidity in this case.[18] Preparedness to manage blood loss in the prone position, multimodal spinal cord monitoring with SSEP and MEP, and MAP augmentation to improve the spinal cord perfusion are some measures that can help to reduce morbidity during this surgery.

Conclusion

Thorough preoperative evaluation and preparation, vigilant intraoperative spinal cord monitoring, aggressive perioperative hemodynamic management, and effective team communication are key to successful outcomes in patients undergoing high thoracic VDT repair.

Conflict of Interest
None declared.

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