

Signal recapture in transcranial motor evoked potentials can herald early spinal cord reperfusion

Ectopic calcification can result in ossification of the posterior longitudinal ligament (OPLL), a condition prevalent among males in the fifth and sixth decades of life.^[1] Familial inheritance in OPLL can be linked to bone morphogenetic proteins.^[2,3] Surgical decompression with laminectomy, laminoplasty or instrumentation, and fusion offers symptomatic improvement in OPLL. Intraoperative neuromonitoring (IONM) aids in diagnosing neurologic injury during spinal instrumentation.^[4]

A 40-year-old female patient presented to us with gradually progressing paresthesia and weakness of bilateral lower limbs of 6-month duration. Clinical examination revealed signs of myelopathy with bilateral spasticity of lower limbs and grade-3 motor power. All sensations were diminished below the D8 dermatomal level. Magnetic resonance imaging of the spine revealed OPLL of D5–D9 level causing severe spinal canal compromise and compression of dorsal spinal cord [Figure 1]. A clinical diagnosis of nonfamilial premature localized OPLL of D5–D9 was made. Surgical decompression with laminectomy under general anesthesia (GA) and IONM was planned.

GA was induced with intravenous (IV) fentanyl 3 mcgs/kg; propofol 2 mg/kg and endotracheal intubation was facilitated with IV vecuronium 0.1 mg/kg. Total IV anesthesia was used for GA maintenance with IV continuous infusions of fentanyl and propofol targeted

to a bispectral index of 40–60. No further skeletal muscle relaxants were used during the maintenance of GA to facilitate IONM. Transcranial motor-evoked potentials (TcMEP) induced compound muscle action potentials (CMAP) were recorded from bilateral adductor pollicis (AP) of upper limbs, tibialis anterior (TA) and extensor hallucis longus (EHL) of lower limbs. Baseline TcMEPs were obtained with monophasic 100–800 V current at a pulse width of 75 μ s and stimulation rate of 250–500 Hz. CMAPs were recorded from bilateral APs, whereas no recordings were obtained from bilateral TA and EHL. The patient underwent decompressive laminectomy from D5 to D9 level in prone operative position under GA. TcMEP recorded after laminectomy consistently showed CMAPs of 600–700 μ V from right TA [Figure 2]. No potentials were recorded from left TA and bilateral EHL. The surgical procedure was otherwise uneventful. Postoperatively, the patient had spasticity of bilateral lower limbs. She could be ambulated on a wheelchair by the 5th postoperative day and could walk with support by the 30th postoperative day.

The loss of CMAPs during spinal instrumentation and distraction are classic alterations that predict early neurologic injury.^[5,6] An intraoperative uniform loss of preoperatively recorded CMAPs in bilateral upper and lower limbs accounts for hemodynamic alterations, hypothermia, or similar systemic insult. An isolated loss of CMAP triggers surgical factors affecting neuronal integrity. Our case highlights a novel advantage of IONM with TcMEPs, i.e. an intraoperative signal recovery of a previously absent CMAP, predicting recovery of neuronal perfusion and thereby function. Our patient had only a partial recovery of CMAP recorded only in right TA. CMAPs to TcMEPs were absent in left TA and bilateral

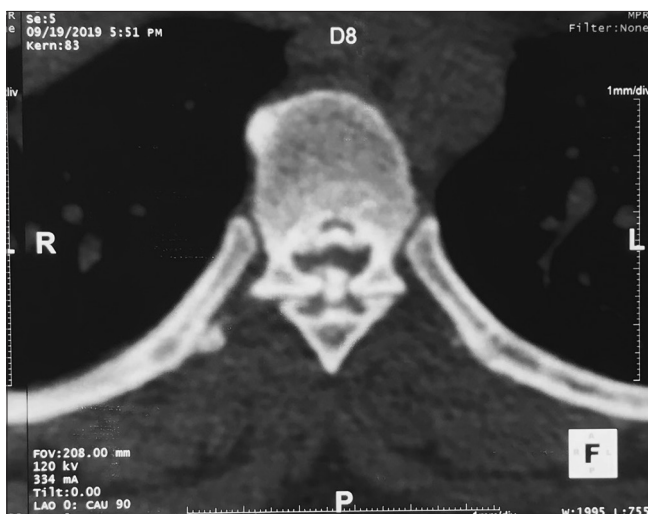


Figure 1: MRI image showing ossification of posterior longitudinal ligament at D8 level with spinal cord compression (space available for cord = 4.5mm)

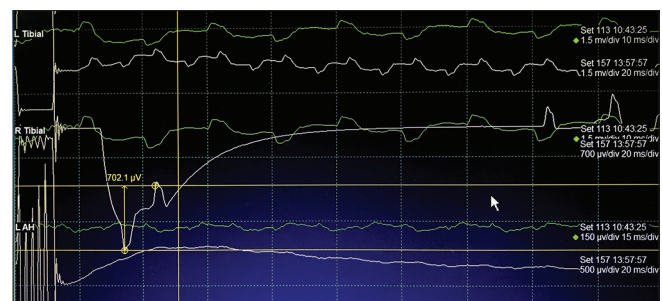


Figure 2: Waveform 4 demonstrates Transcranial Motor Evoked Potential induced Compound Muscle Action Potential (CMAP) of 702.1 μ V from right Tibialis Anterior after Laminectomy (Waveform 3 shows no CMAP pre-laminectomy)

EHL postlaminectomy. This could be explained by a possible ischemic reperfusion injury.^[7] The slow recovery of motor power by the 30th postoperative day supports this fact. Case series of CMAP recovery with TcMEPs in a larger number of patients with symptomatic myelopathy undergoing spinal decompression/instrumentation can consolidate our findings.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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