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Case Report

Intraoperative Neurophysiological Monitoring in Ruptured-Unruptured Multiple Aneurysm Surgery: A Case Report

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ABSTRACT

Intraoperative Neurophysiological Monitoring (IONM) refers to the use of various electrophysiological methods to monitor the function of the brain, spinal cord, and related nerves during surgical procedures. IONM involves the use of neurophysiological recordings to detect changes in nervous system function during surgery, allowing doctors to identify potential nerve damage before it occurs. Reported is a 65-year-old male patient with a diagnosis of unruptured aneurysm of the Posterior Communicating Artery (PcomA) and left ophthalmic segment of C5 whose aneurysm was planned to be clipped. The patient was planned to have the aneurysm clipped under general anesthesia. The challenge in the anesthesia process for aneurysm cases is maintaining the pressure in the aneurysm and cerebral perfusion pressure (CPP), protecting the brain during periods of ischemia, and providing as wide an operating field as possible. Post-operatively, the patient showed stable neurological function with no new deficits, highlighting the importance of intraoperative neurophysiological monitoring in achieving a favorable outcome.

Introduction

Surgical repair of ruptured and unruptured intracranial aneurysms is a complex and high-risk neurosurgical procedure. To reduce the risk of neurological complications and enhance surgical outcomes, intraoperative neurophysiological monitoring (IONM) technology has been increasingly utilized. IONM involves real-time neurophysiological recordings to detect functional changes in the nervous system during surgery, enabling clinicians to identify potential nerve injury before it occurs [1]. In the management of intracranial aneurysms, IONM offers several advantages. Through multimodal techniques such as electroencephalography (EEG), somatosensory evoked potentials (SSEPs), motor evoked potentials (MEPs), and direct cortical stimulation (DCS), IONM facilitates the detection of alterations in brain and peripheral nerve function. This allows for early intervention to prevent neurological injuries, such as perioperative stroke or motor neuron impairment, associated with surgical procedures [2]. Evidence suggests that the use of IONM can lower the incidence of neurological complications during intracranial aneurysm surgery. For instance, a meta-analysis of 873 patients revealed a reduction in new neurological deficits among patients undergoing surgery with IONM compared to those without it [1]. Additionally, another study demonstrated that IONM may reduce the risk of perioperative stroke [2].

In the context of intracranial aneurysm surgery, IONM not only aids in preventing neurological damage but also optimizes surgical precision and effectiveness. By continuously monitoring nerve function, IONM enhances the ability of clinicians to achieve favorable outcomes, establishing it as a valuable component of modern management strategies for intracranial aneurysms.

Case Report

A 65-year-old male patient, weighing 60 kg and measuring 164 cm in height, presented with complaints

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of a persistent headache described as a heavy, constricting sensation around the entire head, ongoing for the past month and partially relieved by analgesics. Over the past week, he noted increasing difficulty in opening his left eye, limited movement of the left eyeball, progressive blurring of vision, and soreness in the left eye. The patient reported persistent headaches unrelieved by analgesics. He denied symptoms such as vertigo, limb weakness, facial drooping, dysarthria, paresthesia, numbness, nausea, vomiting, or changes in vision. He has a history of hypertension, managed with regular medication.

On examination, the patient was alert and oriented (compos mentis), with a blood pressure of 163/80 mmHg, pulse rate of 92 bpm, respiratory rate of 16 breaths per minute, temperature of 36.8°C, and SpO_2 of 99% on room air. Neurological examination revealed a Glasgow Coma Scale (GCS) score of E4V5M6, anisocoria with the right pupil 3 mm and the left pupil 5 mm, and limited movement of the left eye. Visual acuity was 3/60 in the right eye and 2/60 in the left eye, with ptosis present in the left eye. No meningeal signs or motor deficits were observed.

Laboratory results were within normal limits, and no abnormalities were found in chest X-ray findings. Multislice Spiral Computed Tomography (MSCT) angiography revealed a saccular aneurysm in the left internal carotid artery at segment C5 (medially directed, neck width approximately 0.47 cm, dome size approximately 0.68 x 0.54 cm), without additional aneurysms, stenosis, or occlusions of cerebral arteries. No infarctions, hemorrhage, or signs of raised intracranial pressure were identified (Figure 1). The preliminary diagnosis was an unruptured left ophthalmic artery aneurysm, segment C5, classified as Fisher Group 1 and Hunt and Hess Grade 1. The patient was scheduled for aneurysm clipping via craniotomy.

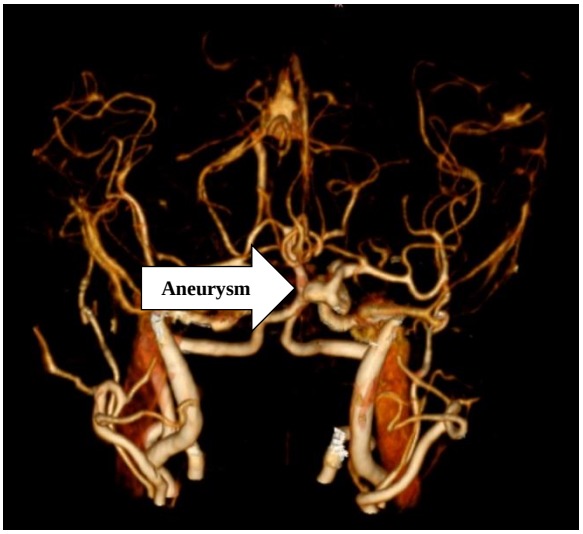

Figure 1- MSCT angiography.

DSA revealed an additional unruptured aneurysm in the Posterior Communicating Artery (PComA) (Figure 2). The final diagnosis was amended to include an unruptured aneurysm of the PComA and the left ophthalmic segment C5, Fisher Group 1, Hunt and Hess Grade 1, with a planned aneurysm clipping craniotomy incorporating intraoperative neurophysiological monitoring (IONM).

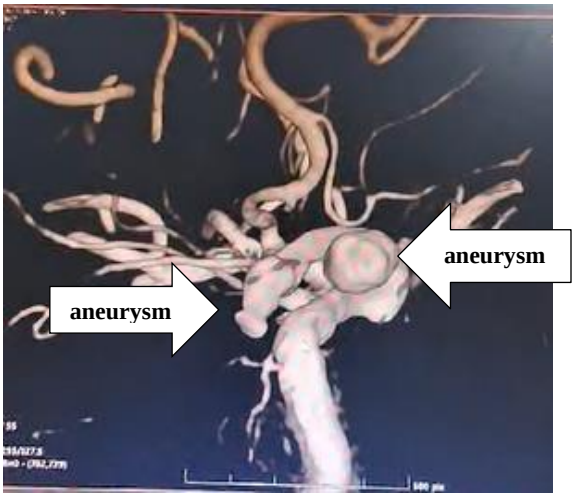

Figure 2- Other aneurysm in the posterior communicating artery (PcomA).

The patient was prepped for aneurysm clipping with a GCS of 15, blood pressure of 160/70 mmHg, pulse rate of 97 bpm, and SpO_2 of 100% on 2 L/min of oxygen via nasal cannula. Induction was achieved with propofol 2 mg/kg, fentanyl 1.5 mcg/kg, rocuronium 1 mg/kg, lidocaine 1.5 mg/kg, oxygen, and saline. Induction proceeded gradually with minute-by-minute blood pressure monitoring, during which the systolic pressure decreased to 100 mmHg, necessitating 5 mg ephedrine administration, raising the systolic pressure to 130 mmHg. A 7.5-mm non-kinking endotracheal tube was inserted, subsequently increasing the systolic pressure to 150 mmHg. Anesthesia was maintained with continuous propofol (50-150 mcg/kg/h) and dexmedetomidine (0.2-0.6 mcg/kg/h). Arterial line monitoring, a subclavian central venous catheter (CVC), and IONM were established following induction. Mannitol 125 cc was administered after the craniotomy.

The operation lasted 8 hours, with a total fluid intake of 2500 ml Ringer's solution and 480 ml packed red cells (PRC), blood loss of 600 ml, and urine output of 1200 ml, while hemodynamic parameters remained stable (Figure 3). The brain was relaxed (Figure 4), and IONM waveforms were well maintained (Figure 5). During clipping, the PComA segment ruptured due to operative manipulation; immediate hemorrhage control was achieved. For the left ophthalmic artery C5 segment, wall augmentation was performed in place of clipping due to its fragile, deep-seated position.

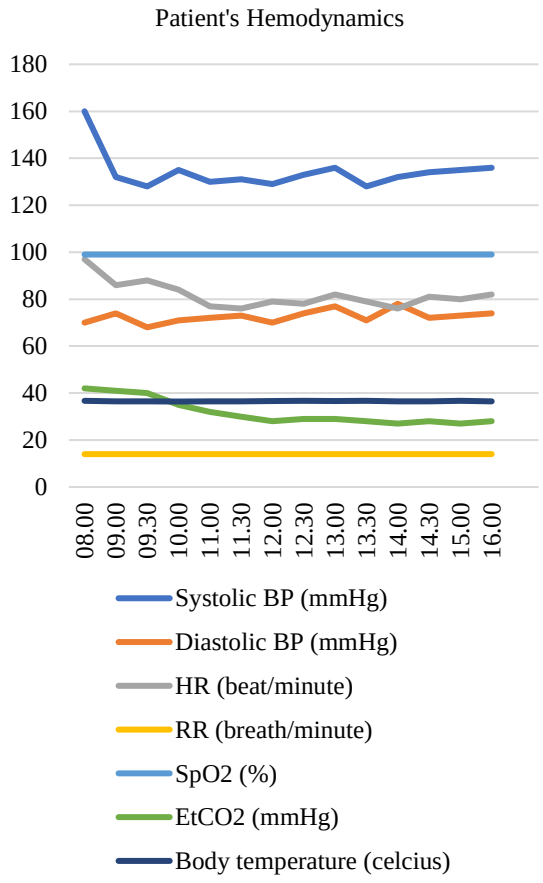

Figure 3- Patient's hemodynamics during surgery.

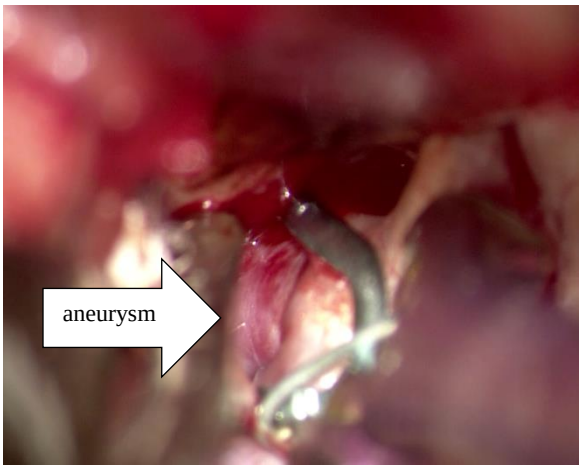

Figure 4- Image of the aneurysm prior to clipping during surgery.

The patient was transferred to the Intensive Care Unit (ICU) on mechanical ventilation and was extubated after 12 hours. Postoperative management included dexmedetomidine 0.2 mcg/kg/h and paracetamol 1 g every 6 hours. No additional neurological deficits were observed post-extubation, and pain was minimal. On postoperative day 3, the patient was transferred to the general ward.

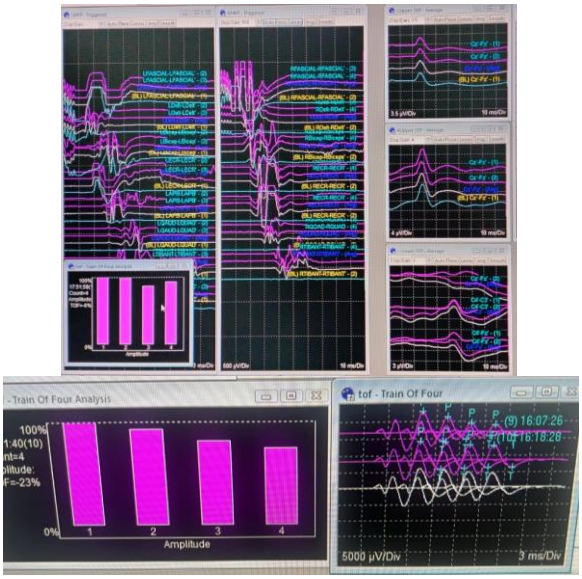

Figure 5- IONM and train of four monitoring during surgery.

Discussion

Neurosurgery, particularly procedures involving the brain, necessitates deep sedation to keep the patient calm and avoid any increase in intracranial pressure during both induction and the surgical intervention itself. The purpose of anesthesia in nearly all surgical procedures is to suppress motor and sensory functions, ensuring patient immobility and comfort. However, in neurosurgery, this suppression limits the ability to detect potential nerve damage during surgery, as it hinders the real-time clinical feedback that could alert the surgeon to neurological issues [3].

Intraoperative Neurophysiological Monitoring (IONM) is designed to mitigate this limitation by utilizing a range of electrophysiological techniques to monitor the functional integrity of the brain, spinal cord, and associated nerves throughout surgery. IONM methods include brainstem auditory evoked potentials (BAEP), visual evoked potentials (VEP), electroencephalography (EEG), electromyography (EMG), motor evoked potentials (MEP), and somatosensory evoked potentials (SSEP). Each technique involves stimulation of specific pathways in the nervous system, with the response recorded to confirm functional stability. Although IONM can encompass multiple modalities, not all procedures require the full spectrum of monitoring [4].

In this case, dexmedetomidine was employed as an alpha-2 adrenergic agonist with hypnotic, sedative, and analgesic effects. Studies have shown that dexmedetomidine reduces postoperative opioid requirements by approximately 60% [5]. Its administration blunts the adrenergic response to surgical

stimuli by decreasing catecholamine levels. Additionally, dexmedetomidine's analgesic effects are achieved without significant respiratory depression, making it useful for perioperative hemodynamic stability and reduced intraoperative opioid consumption [6]. Continuous infusion of dexmedetomidine also reduces the minimum alveolar concentration (MAC) of inhaled anesthetics and demonstrates opioid-sparing properties [7-8].

Propofol is often chosen for IONM, owing to its favorable pharmacokinetics and titratability. Although propofol dose-dependently depresses SSEP and MEP, the extent is limited at commonly used doses, allowing reliable IONM recordings. Thus, propofol is the agent of choice, particularly when MEP monitoring is required. High-dose opioids, such as short-acting fentanyl infusions, are also commonly utilized during procedures involving evoked potential monitoring to further reduce the need for deep anesthesia while allowing effective pain control [4, 9].

Lidocaine is administered as a neuroprotective adjunct, reducing cerebral metabolic rate of oxygen ($CMRO_2$) by 15-20%, which contributes to brain protection. The recommended dose is 1.5 mg/kg, administered intravenously during induction to minimize hemodynamic responses that could elevate mean arterial pressure [10]. Lidocaine's neuroprotective effects are attributed to its ability to decrease ion transference across membranes, reduce metabolic demand in the brain, modulate leukocyte activity, and limit excitotoxic release during ischemic events [11].

For muscle relaxation, rocuronium was administered at an induction dose of 1 mg/kg. Rocuronium's rapid onset and moderate duration of action provide reliable neuromuscular control, critical in surgeries such as aneurysm clipping, where patient immobility is essential to prevent any inadvertent nerve injury [12].

The primary anesthetic goals in aneurysm surgery are as follows: (1) Achieving brain relaxation to minimize the need for retraction maneuvers, (2) maintaining optimal Cerebral Perfusion Pressure (CPP) during any ischemic intervals, (3) reducing Transmural Pressure (TMP), particularly during and after vessel occlusion, (4) avoiding abrupt fluctuations in intracranial pressure (ICP), and (5) facilitating rapid postoperative awakening to allow prompt neurological assessment [13-14].

Conclusion

Successful anesthesia management for brain aneurysm patients requires a comprehensive understanding of the patient's medical history, the underlying pathophysiology, and the specifics of the surgical procedure. Key anesthetic objectives during aneurysm clipping include controlling transmural pressure in the aneurysm region, maintaining adequate cerebral oxygenation and perfusion, and minimizing factors that could alter intracranial pressure. Effective anesthetic strategies should also enhance surgical visibility and reduce brain retraction. Proper preventive measures and timely intervention can significantly improve patient outcomes.

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