Case Report/Series

Perianesthetic management in a teenager with focal cortical dysplasia who underwent an epilepsy surgery

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ABSTRACT
Medically resistant epilepsy (MRE) is defined as the failure of adequate trials of 2 tolerated and appropriately used antiepileptic drugs to achieve sustained seizure freedom, where epilepsy surgery (ES) is indicated in patients with MRE due to complex epileptic syndromes, such as Lennox-Gastaut and Sturge-Weber syndromes, and focal cortical dysplasia (FCD), to disconnect the epileptogenic foci from other cerebral structures. ES demands excruciating perianesthetic considerations due to its complexity and various pharmacological interactions. Our patient is a 15-year-old male with MRE secondary to FCD. He successfully underwent ES in our neurosurgical center. We describe our perianesthetic challenges in addition to a brief overview of MRE and FCD.

KEYWORDS anesthesia, epilepsy, focal cortical dysplasia, surgery

Focal cortical dysplasia (FCD), a congenital malformation of cortical development and organization,[1] is a common cause of medically resistant epilepsy (MRE). Epilepsy surgery (ES) is a key intervention for patients with MRE, involving the identification and excision of epileptogenic foci.[2] Administering anesthesia for ES is particularly challenging due to the complexity of the procedure and the associated pharmacological interplay.

Adequate intraoperative electrophysiological monitoring and optimal anesthetic management are crucial for a successful outcome.[3,4] Target-controlled infusion (TCI) of propofol and remifentanil is preferred, as these agents exert minimal inhibitory effects on motor evoked potentials (MEPs) and somatosensory evoked potentials (SSEPs) during electrocorticography (ECoG).[4] Here, we present the perioperative anesthetic management of a patient with FCD who underwent successful ES. This case report highlights the critical anesthetic strategies employed to ensure patient safety, optimize surgical outcomes, and emphasize the necessity of a multidisciplinary approach in managing complex cases of MRE.

CASE REPORT

A 15-year-old male presented with a 3-year history of recurrent seizures, often preceded by an olfactory aura, blank stare, and automatism, including lip-smacking, chewing, and eye-blinking. Each seizure episode lasted approximately 5 min, occurred spontaneously without clear precipitating factors, and was followed by drowsiness, with full recovery within an hour. His seizures occurred frequently during the day and occasionally at night, as reported by his parents. According to his mother, he did not exhibit generalized convulsions, described as having jerky movements of the four limbs and upward eye deviation, cyanosis, tongue biting, or loss of bladder or bowel control. He also did not experience physical trauma during seizure episodes.

Seizure frequency remained stable at two to three monthly episodes over 3 years. However, it had increased in the past 6 months despite adherence to the maximum doses of three oral antiepileptic drugs (AEDs): phenytoin (400 mg three times daily), carbamazepine (600 mg twice daily), and levetiracetam (1 g twice daily).

During examination, he was alert and conscious. His baseline blood pressure was 107/62 mmHg, pulse rate was 75 beats per minute, respiratory rate was 15 breaths per minute, and oxygen saturation was 100% on room air. He did not appear syndromic, and his neurological examination was intact. Other systemic examinations, including those of the cardiorespiratory system, were unremarkable.

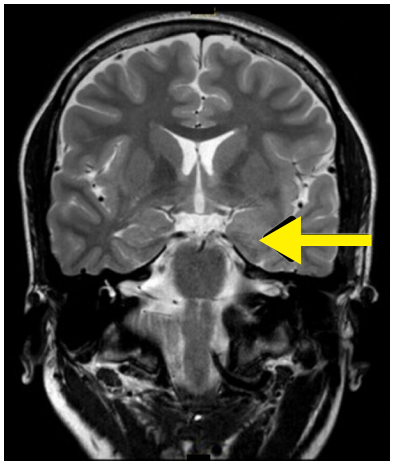

Figure 1. Preoperative coronal view of the patient's MRI brain showing an enlarged left amygdala. It had poor gray-white differentiation at this region, showing left amygdala FCD. FCD=focal cortical dysplasia; MRI=magnetic resonance imaging

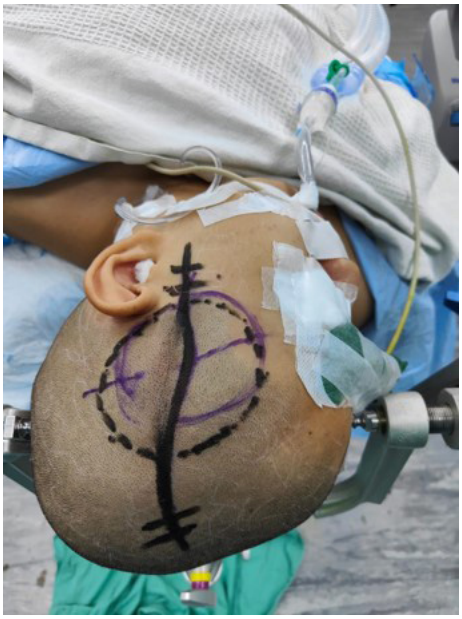

Figure 2. Patient in left lateral position during surgery to expose the surgical site

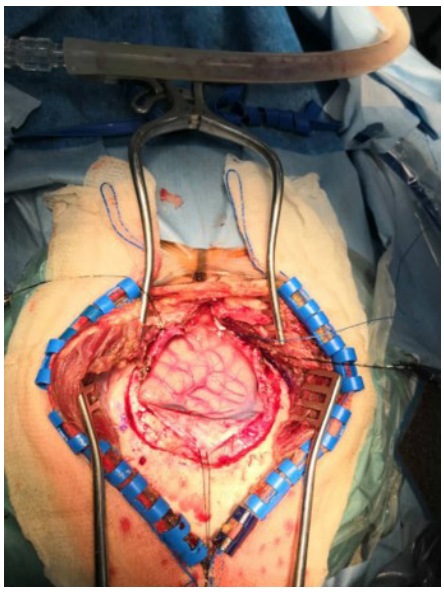

Figure 3. A linear incision exposing the left temporal lobe

Hematological investigations showed a hemoglobin level of 13.2 g/dl (normal: 11–14 g/dl), total white blood cells of 7.4 × 10^9/l (normal: 7–12 × 10^9/l), and a platelet count of 265 × 10^9/l (normal: 150–450 × 10^9/l). Biochemical investigations were within normal ranges, and therapeutic drug monitoring confirmed that phenytoin and carbamazepine levels were within the therapeutic range.

Electroencephalography (EEG) indicated left temporal cortical irritability and focal dysfunction. Cerebral magnetic resonance imaging (MRI) revealed focal cortical thickening in the left temporal lobe and an enlarged left amygdala compared to the right, with poor gray-white matter differentiation and high signal intensity (Figure 1). Bilateral hippocampi were slightly asymmetrical, with the right hippocampus measuring 0.6 cm and the left 0.7 cm in maximum thickness. These findings were consistent with the FCD of the left amygdala.

The provisional diagnosis was MRE secondary to left amygdala FCD, confirmed by EEG and MRI. Other differential diagnoses, including generalized epilepsy syndromes such as Lennox-Gastaut syndrome, Dravet syndrome, or tuberous sclerosis, were excluded based on clinical and neuroimaging findings. Given the diagnosis of MRE secondary to left amygdala FCD, the patient and his parents were thoroughly counseled and scheduled for elective left amygdalohippocampectomy (AH). Informed consent was obtained from the patient's parents for surgery, general anesthesia (GA), and manuscript publication. The parents were informed of AH's potential benefits, including reduced seizure frequency and improved quality of life (QoL), as well as possible risks, such as bleeding, neurological deficits, infection, aspiration, and seizure recurrence.

He fasted for 8 hours preoperatively and received 0.9% intravenous (IV) normal saline for fluid maintenance during fasting. All oral AEDs were administered on the morning of surgery. GA was induced with TCI of remifentanil and propofol, along with IV rocuronium (1 mg/kg) for neuromuscular blockade. A 6.5-mm endotracheal tube was inserted to secure the airway. For hemodynamic monitoring and resuscitation, a right radial artery catheter and subclavian vein catheter were placed. GA was maintained with TCI remifentanil (2.4–4.7 ng/ml) and propofol (3.1–4.8 mcg/ml), with a bispectral index (BIS) monitor placed on the right forehead to assess anesthetic depth.

Fluid therapy was managed with 0.9% normal saline, guided by the Holliday-Segar formula. Additional intraoperative measures included the placement of a mouth gag, nasopharyngeal temperature probe, and Ryle's tube to ensure airway management, normothermia, and gastric decompression. He was positioned supine with his head turned to the right, with the left zygoma elevated to facilitate gravitational retraction of the left temporal lobe (Figure 2).

A left craniotomy was performed, and the superior temporal sulcus was identified and gently opened (Figure 3). Neurosurgeons were guided by an intraoperative image-guided surgery (IGS) system. Dissection proceeded through the left subcortical region to expose the temporal horn. The left hippocampus, including its head, body, and amygdala, was resected piecemeal using an ultrasonic aspirator. During the resection, extreme care was taken to preserve the anterior and posterior cerebral arteries under IGS guidance.

The total duration was 3.5 hours, with an estimated blood loss of 300 ml and stable intraoperative hemodynamics. Before extubation, a bilateral scalp block was administered by a neuroanesthesiologist using levobupivacaine (0.375%) for postoperative analgesia, in addition to IV morphine and paracetamol. He was extubated uneventfully in the operating theatre with sugammadex, demonstrating a Glasgow Coma Scale score of 15 and full motor function (5/5 in all limbs). He was transferred to the neurocritical care unit for overnight monitoring, with oral AEDs resumed immediately postoperatively. No postoperative seizures were noted. Regular doses of paracetamol and parecoxib were administered for 2 days. He was discharged 3 days postoperatively without complications.

At 2-week follow-up, he reported no seizures or aura episodes and exhibited no surgical site infections or neurological deficits. He remained compliant with AED therapy. One month postoperatively, carbamazepine was reduced to 600 mg once daily, whereas the dosage of levetiracetam and phenytoin remained unchanged.

Three months postoperatively, he complained of a single, self-aborted seizure episode with an accompanying aura. By 6 months postoperatively, he had remained seizure-free, allowing for discontinuation of carbamazepine. Levetiracetam and phenytoin were continued, though the phenytoin dosage was reduced to twice daily. No frequent seizures or episodes of auras were noted following the new prescriptions.

One year after surgery, he remained well and seizure-free, with no frequent aura episodes. His AED regimen was further reduced to a daily dose of levetiracetam and phenytoin.

DISCUSSION

We report a case of a 15-year-old male with MRE secondary to FCD who successfully underwent ES at our neurosurgical center in Malaysia. This case highlights the perioperative anesthetic challenges and provides an overview of MRE and FCD. The International League Against Epilepsy defines a seizure as a transient occurrence of signs and symptoms due to abnormal, excessive neuronal activity in the brain.[2] Seizures contributed to over 0.5% of the global disease burden, accounting for over 13.5 million disability-adjusted life years worldwide.[1,3] In Malaysia, the lifetime prevalence of epilepsy is 7.8 per 1,000 people.[5]

MRE is defined as the failure of two adequately tolerated and appropriately used AEDs, either as monotherapy or in combination, to achieve sustained seizure freedom.[6,7] It is a primary indication for ES.[8] Patients with complex epileptic syndromes that are resistant to AEDs, such as Lennox-Gastaut, Sturge-Weber syndromes, and FCD, are ideal candidates for ES.[9–12]

FCD is a cortical developmental malformation highly associated with MRE and epileptogenic. First described histopathologically in 1971, FCD is characterized by cortical disorganization, bizarre neurons, and balloon cells.[13] It is considered the most common histopathological finding in epilepsy surgery cases.[14] ES is recommended when the seizure foci can be clearly identified.

ES is typically performed in specialized neurosurgical centers with experienced surgeons, neuroanesthesiologists, and adequate neurocritical care. It is a highly stimulatory procedure that requires careful perioperative planning. ES can be performed under GA, as in our patient, or during awake craniotomy. The latter was not considered in this case, as the seizure focus was located in the hippocampus.

Patients with FCD, brain tumors, and meningitis have reported postoperative seizure-freedom rates of 78%, 61%, and 52%, respectively.[15–17] In this case, the patient met the overall indications and benefits of ES. Given that he was actively attending school, early intervention in MRE was crucial for his academic progress. Additionally, his participation in competitive sports and representing his school in football tournaments emphasized the need for effective seizure management to support his physical and cognitive development.

Patients undergoing ES require special perioperative anesthetic considerations to minimize potential triggers and ensure intraoperative stability.[18–21] Chronic AED use, particularly phenytoin, may cause gingival hypertrophy, increasing the risk of oral bleeding during laryngoscopy and intubation.[16,19,20] AEDs such as phenytoin, carbamazepine, and lamotrigine are well-known hepatic enzyme inducers, leading to increased metabolism of opioids and non-depolarizing muscle relaxants. Consequently, higher doses and more frequent administration of these drugs are required.[19–22]

Intraoperative ECoG assists neurosurgeons in identifying epileptiform activity by placing EEG leads directly on the cerebral cortex.[12,17] This technique guides surgeons in defining safe resection margins. During ECoG, potent opioids such as fentanyl, remifentanil, and alfentanil are often required to suppress epileptiform waveforms.[8,12] However, reducing the depth of anesthesia during ECoG increases the risk of accidental awareness.[8,14,16]

Optimal intraoperative management aims to maintain normotension, normoxia, normocapnia, normothermia, and normoglycemia while avoiding metabolic acidosis, hypocapnia, and dehydration to reduce the seizure threshold.[16,22] Anesthesia can be maintained using either TCI of remifentanil and propofol, as used in this case, or inhalational sevoflurane.[8,19] TCI with remifentanil and propofol is preferred, as it preserves cerebral autoregulation, maintains cerebral blood flow (CBF), and reduces intracranial pressure.[6,7] Propofol also has potent antiepileptic properties, making it beneficial for seizure prophylaxis and intraoperative seizure management.[6,19–21] Both propofol and remifentanil can be rapidly titrated to respond to neurosurgical stimulation, such as head pinning, which may trigger hypertensive episodes.[6,21] These agents also have minimal effects on SSEP, MEP, and ECoG compared to sevoflurane.[14,19] Sevoflurane, at a minimum alveolar concentration of 1.0, has no significant effect on CBF and protects against cerebral edema.[6,8,14] If intraoperative cerebral edema occurs, transient hyperventilation can be used to maintain a partial carbon dioxide pressure of 28–30 mmHg, along with IV mannitol of 0.25–1 g/kg administered over 30 min.

Seizures are a common complication of AH.[6,8] Thus, anesthesiologists must remain vigilant for clinical signs of seizures, including sudden limb jerking, eyelid

flickering, unexplained tachycardia, and hypertension. Immediate management includes increasing the fraction of inspired oxygen to 100% and administering IV propofol (0.5–1 mg/kg), midazolam (1–5 mg), or AEDs such as phenytoin.

Although ES is an effective treatment for MRE in patients with FCD, some limitations persist. First, ES is not widely available in Malaysia and is performed only at at selected centers, including Hospital Universiti Sains Malaysia, Hospital Kuala Lumpur, Hospital Sungai Buloh, and Hospital Sultanah Aminah. This limitation is due to the need for highly skilled neurosurgeons specializing in ES. Second, the diagnosis of FCD relies on advanced imaging techniques, including MRI and positron emission tomography scans, to detect subtle structural abnormalities, requiring well-trained neuroradiologists. Moreover, successful ES execution necessitates an operating theatre equipped with specialized neurocritical care services, including neuroanesthesiologists, trained nurses, and neuroanesthetia equipment, to ensure optimal perioperative monitoring and patient management.

In conclusion, ES should be considered for patients with MRE, particularly those with FCD, as it significantly improves seizure control, QoL, cognitive development, and neurological outcomes. Perianesthetic management during ES aims to prevent seizures by maintaining normoxia, normotension, normocapnia, normothermia, and normoglycemia. TCI with remifentanil and propofol is highly recommended as a safer and more effective anesthetic approach due to its minimal impact on the SSEP, MEP, and ECoG, making it an optimal choice for neurosurgical interventions.

Conflict of Interest

The authors affirm no conflict of interest in this study.

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