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Case Report

Perioperative Management of Neuroanesthesia in Patients with Supratentorial Tumors Who Have Excised Tumors Using Neuroprotection Technique and Total Intravenous Anesthesia

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ABSTRACT

Background: Neuroanesthesia management presents a unique challenge for anesthesiologists. They must provide an optimal surgical condition without worsening the patient's neurological deficits. Therefore, we need drugs with neuroprotective abilities. This case report explains the perioperative management of neuroanesthesia in patients with supratentorial tumors who have excised tumors using a neuroprotection technique and total intravenous anesthesia (TIVA).
Case: A 43-year-old female patient with space-occupying process cerebri with the differential diagnosis of meningioma frontotemporal dextra, post trepanation frontal sinistra tumor excision, edema cerebri, and hydrocephalus on ventriculoperitoneal shunt. On physical examination, Glasgow coma scale E3M6$V_{aphasia}$, aphasia and left hemiparesis were found. She underwent a tumor excision procedure with total intravenous anesthesia modified with neuroprotection techniques and total intravenous anesthesia techniques using 300 mg thiopental, 2 mg midazolam, 150 µg fentanyl, 80 mg lidocaine, and 50 mg rocuronium. Intraoperative anesthesia management was carried out by administering propofol 50 mg/hour, fentanyl 50 µg/hour, and atracurium 15 mg/hour.
Conclusion: Total intravenous anesthesia is a complete general anesthesia method used in all intravenous agents, where the benefits of this method are used in neurosurgery, including accelerating the patient's return from the effects of anesthesia, faster recovery of cognitive function, as well as reducing intracranial pressure and the risk of ischemia.

Keywords: Supratentorial tumors, neuroanesthesia, craniotomy, neuroprotection

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INTRODUCTION

Anesthesia management in neurosurgical and neurocritical patients poses unique challenges for anesthesiologists. Along with the development of science and technology, anesthesia management has developed into a science that is comprehensively researched with the aim of providing a safe and comfortable procedures for patients during the perioperative period. Craniotomy is a commonly performed neurosurgical procedure in which a part of the skull is removed to gain access to the intracranial cavity. The objective of anesthesia in neurosurgery is to achieve a balance that ensures optimal cerebral hemodynamics and oxygenation. This involves maintaining adequate cerebral perfusion pressure (CPP), preventing intracranial hypertension, and creating the best possible surgical conditions to prevent the exacerbation of existing neurological conditions.[1,2] This case report explain the perioperative management of neuroanesthesia in patients with supratentorial tumors who have excised tumors using neuroprotection technique and total intravenous anesthesia.

CASE

In this case report, perioperative anesthesia management will be explained which focuses on neuroprotective techniques in a patient with supratentorial tumor undergoing tumor excision surgery, and also to describe the use of neuroprotective drugs aimed at cerebral protection starting from induction and maintenance agents.

A woman, 43 years old, was taken by her family to the hospital with complaints of difficulty speaking and unconnected speech for the past 2 months and dizziness that has been intermittent for the past 2 years. She has a history of frontal meningioma. She had a trepanation procedure for excision of the

left frontal tumor 4 years ago and the installation of a Kocher ventriculoperitoneal (VP shunt) 1 year ago. The patient came for further surgery. She has a cough but denies fever and shortness of breath.

On physical examination, Glasgow coma scale (GCS) $E3M6V_{aphasia}$ was obtained, blood pressure 125/72 mmHg, respiratory rate 22-26 times/minute, pulse 80 times/minute, temperature 36.8°C. Neurological examination revealed aphasia (+), lateralization (-), motor strength 5/5, sensory strength 2/2. Lung examination revealed symmetrical chest movements, auscultation of right and left vesicular breath sounds, wheezing (-), and ronchi (-). Cardiac examination revealed single S1 – S2 heart sounds, murmur/gallop (-). Abdominal examination revealed soft, no tenderness, bowel sounds were normal. There was no edema or cyanosis in the extremities but we found the left hemiparesis.

Supporting and additional examinations obtained the following data (**Table 1**).

Table 1. Laboratory results

Parameters	Results	Parameters	Results
Hemoglobin Hematocrit	12.6 g/dl	PT	11.5 seconds
Leukocytes Platelets	38.6%	APTT	27.10 seconds
Sodium Potassium	5850/mm^3	Albumin	3.55 g/dl
Chloride	254,000//mm^3	GDS	87 mg/dl
Urea Creatinine SGOT	141 mEq/l	eGFR	129.6046
SGPT	3.07 mEq/l	BGA :	
	112 mEq/l	pH	7.46
	21.8 mg/dl	HCO_3	26.5
	0.35 mg/dl	$PaCO_2$	74.2
	11 u/L	HCO_3	19.1
	8 u/L	BE	-4.9
		SpO_2	95.4%

PT: prothrombin time; APTT: activated partial thromboplastin time; SGOT: serum glutamic oxaloacetic transaminase, SGPT: serum glutamate pyruvate transaminase; eGFR: estimated glomerular filtration rate; BGA: blood gas analysis; $PaCO_2$: partial pressure of carbon dioxide; HCO_3: bicarbonate; BE: base excess; SpO_2: peripheral oxygen saturation

An electrocardiogram showed a sinus rhythm of 65 beats/minute. The results of head magnetic resonance imaging (MRI) (**Figures 1** and **2**) showed multiple meningiomas in the right frontotemporal region and bilateral temporal region which caused subfalcine herniation to the left of 3.2 cm. Therefore, it could be interpreted as an increasing condition, trans-tentorial downward herniation at the level of the mesencephalon, so it can be stated as a condition of elevation, cerebral edema, and chronic infarction in the right-sided mesencephalon and left frontotemporal lobe shown in **Figure 1**.

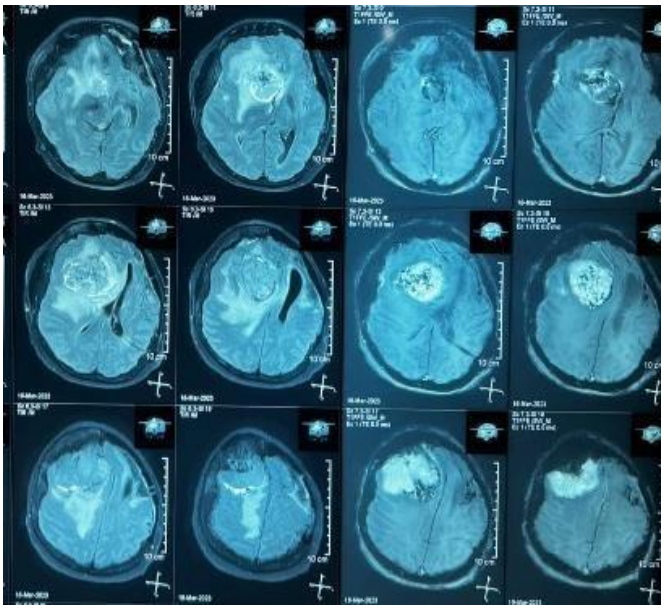

Figure 1. Head MRI of the patient

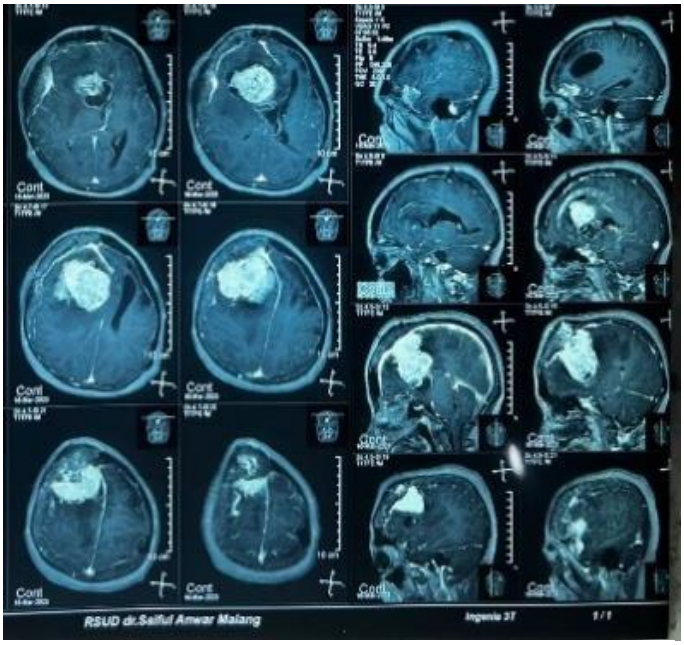

Figure 2. Head MRI of the patient

The patient was diagnosed with Space Occupying Process Cerebri, with differential diagnosis Frontotemporal Meningioma Dextra, Post trepanation excision of the left frontal tumor, Cerebral Edema, and Hydrocephalus on VP shunt. Initial assessment showed that she was included in American Society of Anesthesiologist (ASA) category 3. The patient was planned to undergo a tumor dissection craniotomy. Preoperative preparation of the patient was performed with informed consent, fasting on solid food 6 hours before surgery, clear drinks 2 hours before surgery, preparation of the intensive care room for post-surgery, and premedication by administering ranitidine 50 mg +

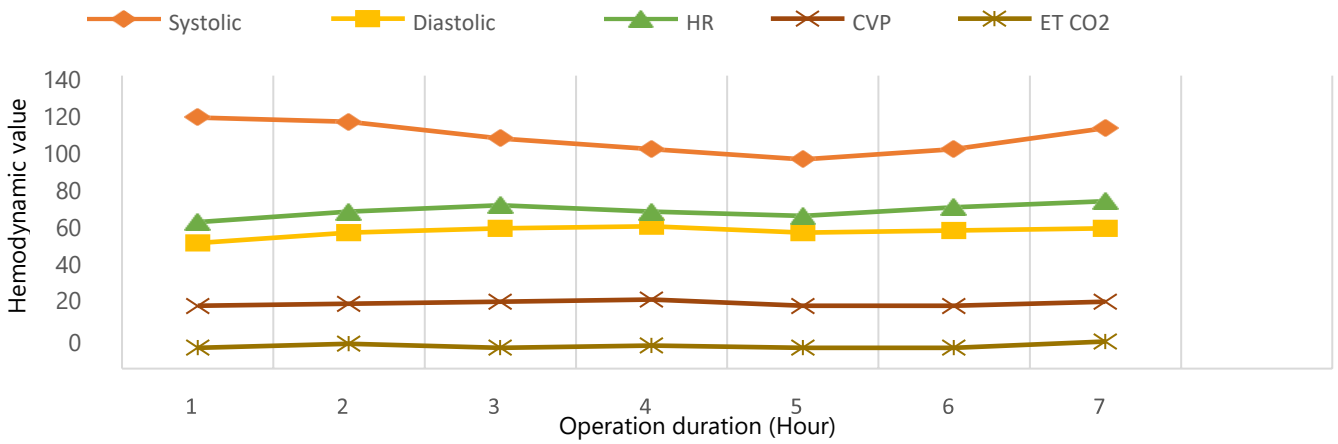

Figure 3. Hemodynamics state during surgery

metoclopramide 10 mg intravenous (IV) 1 hour before surgery.

In this patient, anesthesia was induced using a total intravenous procedure with the principle of neuroprotection through a combination of thiopental 300 mg IV, midazolam 2 mg, fentanyl 150 µg IV, lidocaine 80 mg IV, and rocuronium 50 mg. Previously, preoxygenation was carried out for 5 minutes with 100% O_2, monitor installation, 3-lead ECG, and SpO_2. Intubation was facilitated using 50 mg of rocuronium using a non-kinking endotracheal tube (ETT) no 7. Hemodynamics measured blood pressure 120/60 mmHg, pulse 70 times/minute, respiratory rate 22 times/minute, O_2 saturation 99%.

She had a 7 Franc central venous catheter (CVC) installed in the right subclavian vein and an arterial line in the right radial artery. IBP and CVP are continuously connected to the monitor. A urinary catheter was installed with a 200 ml urine rest. After checking that the hemodynamics state are stable, then she was positioned on the left side (true lateral). Maintenance of anesthesia was carried out by administering propofol 50 mg/hour, fentanyl 50 µg/hour, and atracurium 15 mg/hour with tidal volume (VT) control ventilation mode of 440 ml, frequency 12 times/minute, and oxygen saturation reading 99-100%.

The surgery lasted 7 hours. Trepanation and resection of approximately 80% of the tumor were performed. The hemodynamic state was stable during the surgery (**Figure 3)**. The brain image during surgery can be seen in **Figure 4.** Laboratory results during BGA duration (15.03), DL: 7.32/29.3/114.1/20.3/—9.5/96.5%. Fluid estimation during surgery can be seen in the **Table 2.**

Table 2. Fluid estimation during surgery

Outputs	Inputs	Balance
Urine 2000 ml/7 hours Bleeding 1300 ml	Crystalloid 2000 ml Colloid 500 ml Mannitol 200 ml PRC 800 ml	Deficit 200 ml

After surgery, delayed emergence was performed. The patient was taken to the Intensive Care Unit (ICU) in a state of intubation and controlled ventilation. A CXR examination showed minimal left pleural effusion, cardiomegaly, and new lesion pneumonia. An MRI head examination was also carried out after surgery (**Figure 5**). Post-operatively, the patient was given in. Ceftriaxon 2x1 g, inj. Dexamethasone 3x5mg, inj, Metamizole 3x1 g, inj. Ranitidine 2x50 mg, inj. Phenytoin 3x100mg, and inj. Tranexamic acid 3x500 mg. We also administered Midazolam 5 mg/hour and fentanyl 50 µg/hour via syringe pump.

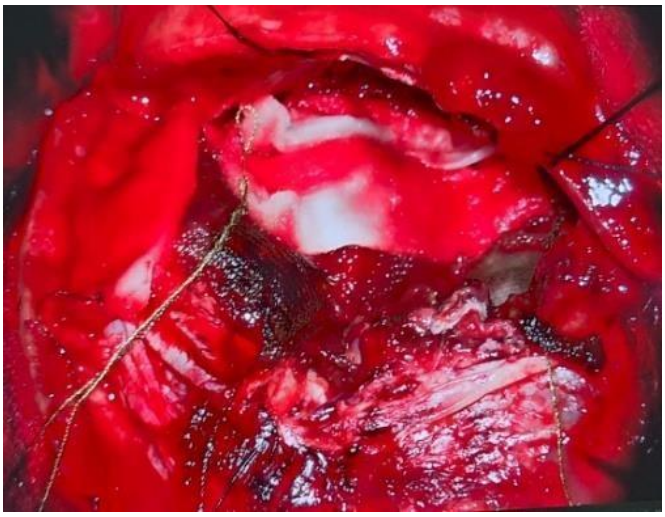

Figure 4. Image of the brain during surgery

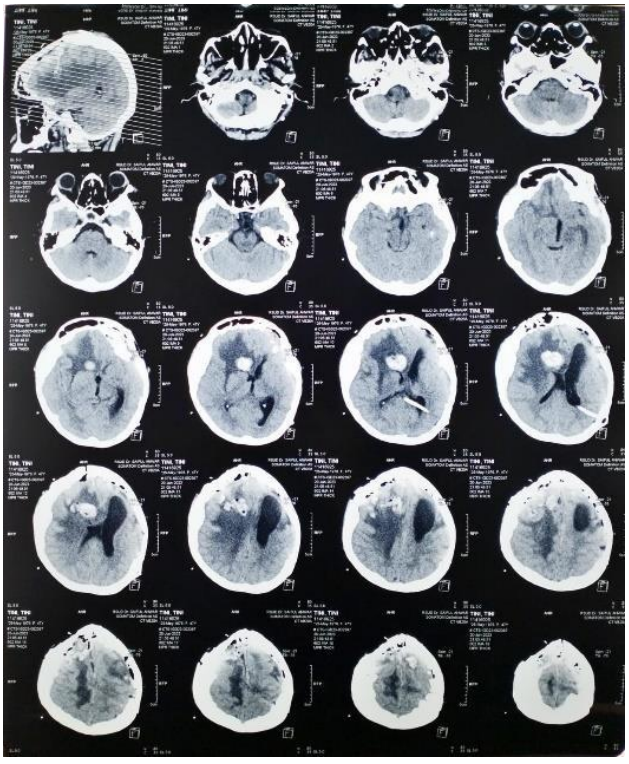

Figure 5. MRI examination 06/20/2023

First-day post-operative monitoring: desaturation (-), Bradycardia (-), Hypotension (-), Hypertension (-), Tachycardia (-). On physical examination, it was found that she was conscious under sedation, blood pressure 118/83 mmHg, breathing installed ETT on ventilator PSIMV mode SPO2 100%, Ves +/+, Rh -/-, wheezing -/-, Retraction (-), pulse 69x/ minute. Aphasia, lateralization (-), motor strength is difficult to evaluate, and sensory strength is difficult to evaluate. Assessment of post-extubation neurological deficits returned to preoperative conditions and even reduced complaints of headaches and weakness in the extremities. After 2 days of ICU treatment, the patient returned to the inpatient room.

DISCUSSION

General anesthesia (GA) is usually used in major operations. To achieve the depth of anesthesia during surgery, the patient is given various general anesthetics, either single drugs or in combination with other agents. GA has long been theorized to have long-term effects on the brain. In animal studies, GA has been found to induce cytosolic calcium accumulation and impair the function of autophagosomes and autolysosomes to reduce cytoprotective autophagy, which would bias cells toward apoptosis.[2] In this case, the anesthetic technique used in this patient was total intravenous anesthesia with the principle neuroprotective.

Neuroanesthesia techniques use the ABCDE principle, which consists of Airway, Breathing, Circulation, Drugs, and Environment. Airway refers to an airway that must be free by intubation with a non-kinking endotracheal tube of the largest possible size and an oropharyngeal airway of the right size to prevent sore throat/coughing, which can increase intracranial pressure. Next, fixation is carried out, and its position in the airway is monitored. Breathing refers to controlled ventilation to obtain adequate oxygenation by avoiding spontaneous breathing in patients taking the muscle relaxant drug rocuronium. Ventilate with a goal of PaO_2 < 200 mmHg and $PaCO_2$ normocapnia/ slight hypocapnia and subsequent monitoring. Circulation prioritizes stable blood pressure, where fluctuations can exacerbate cerebral edema and increase intracranial pressure. This is performed using systematic induction drug administration techniques, head-up position, and fluid administration targeting normovolemic, normoglycemic, and iso-osmolar fluids. Drugs refer to giving brain protection drugs and avoiding drugs that can increase intracranial pressure, ranging from induction anesthesia, maintenance, and muscle relaxants to narcotic analgesics. *Environment*, namely maintaining a mild hypothermic temperature of 35ºC, which is measured in the pharynx and tympanic membrane, by regulating the temperature of the operating room and the temperature of the infusion fluid.[3] In this case, the ABCDE principles of neuroanesthesia techniques have been applied: installation of an endotracheal tube, use of a mechanical ventilator, administration of fluids and drugs to maintain hemodynamic stability, and environmental regulation.

The brain is highly susceptible to ischemic injury due to its relatively elevated oxygen consumption and substantial reliance on aerobic glucose metabolism. Compromised cerebral perfusion, deficiency in metabolic substrates (such as glucose), or severe hypoxemia can result in rapid functional impairment. Furthermore, reduced perfusion diminishes the clearance of potentially toxic metabolites. If normal oxygen tension, blood flow, and glucose supply are not promptly restored, then most of the ATP stores will be depleted, and irreversible nerve injury will begin.[4]

When cerebral blood flow (CBF) falls below 10 mL/100 g/minute, cellular function is disrupted, and ion pumps fail to sustain cell vitality. This reduction in CBF leads to an increase in the lactate-to-pyruvate ratio as a result of anaerobic metabolism. During ischemia, intracellular potassium (K^+) levels decrease while intracellular sodium (Na^+) levels rise. Intracellular calcium (Ca^{2+}) also increases due to the failure of ATP-dependent pumps to expel ions, the elevated intracellular sodium concentration, and the release of the neurotransmitter glutamate. Glutamate activates NMDA receptors, further increasing calcium influx into cells and highlighting the potential neuroprotective benefits of NMDA inhibitors.[4]

The continuous rise in intracellular Ca^{2+} activates lipases and proteases, leading to the propagation of structural damage in neurons. Elevated concentrations of free fatty acids, along with increased cyclooxygenase and lipoxygenase activity, result in the production of prostaglandins and leukotrienes, some of which are potential mediators of cellular injury. The accumulation of toxic metabolites further impairs cellular function and disrupts repair mechanisms. Lastly, reperfusion of ischemic tissue can inflict additional damage due to the generation of oxygen-derived free radicals. The ensuing inflammation and edema can exacerbate nerve damage, ultimately leading to cellular apoptosis.[4]

Brain relaxation status is an important factor in anesthesia for intracranial surgery. A relaxed brain improves surgical conditions, minimizes the severity of retraction injuries, and prevents compression-induced ischemia; These factors contribute to improved patient outcomes. Various maneuvers can be employed to achieve brain relaxation during craniotomy. The anesthetic technique selected for craniotomy should aim to minimize cerebral blood volume (CBV), cerebral metabolic rate of oxygen ($CMRO_2$), and cerebral blood flow (CBF) without compromising neural function. Additionally, the technique should preserve cerebrovascular autoregulation and its responsiveness to carbon dioxide (CO_2). The technique should also allow the anesthesiologist to change the depth of anesthesia quickly and safely, thereby ensuring rapid and predictable recovery from the effects of anesthesia.[5-7]

Controlled hypotension is a commonly employed technique in patients undergoing craniotomy for supratentorial intracranial surgery. This technique is defined as a reduction in systolic blood pressure (SBP) to 80-90 mmHg (a 30% decrease from baseline SBP) or a decrease in mean arterial pressure (MAP) to 50-65 mmHg in normotensive patients.[5–7] In this case, the patient has relatively stable systolic and diastolic blood pressure and did not experience periods of hypotension.

Medications used to induce controlled hypotension can be administered alone or in combination. Agents such as sodium nitroprusside, nitroglycerin, and trimethaphan can be used individually, while calcium channel blockers (CCBs), β-blockers, and fenoldopam can be used either alone or in combination. Angiotensin-converting enzyme inhibitors (ACE-Is) and clonidine are primarily utilized as adjuncts. Inhalation agents like isoflurane and sevoflurane offer several advantages in neurosurgery, particularly at low cerebral perfusion pressures (< 30 mmHg). $CMRO_2$ is better maintained and affects the global cerebral oxygen supply/demand ratio. The use of controlled hypotensive anesthesia techniques is based on the belief that inducing hypotension will result in brain relaxation, decreased blood loss, reduced blood transfusion rates, and shorter duration of surgery.[7,8] In this case, the patient did not receive the drugs used to control hypotension because they did

not experienced hypotension during surgery.

The anesthesia technique in this case uses the total intravenous anesthesia (TIVA) method, a general anesthesia technique that uses a combination of agents administered via a syringe pump exclusively by the intravenous route without using inhalation agents (anesthetic gas). TIVA given together with propofol and remifentanil was found to provide benefits in neuroanesthesia procedures, especially in the return of patients from the effects of anesthesia and faster recovery of cognitive function. A rapid and coherent emergence without vomiting is crucial for neurosurgical patients to facilitate the prompt assessment of any changes in neurological status caused by the surgery or the progression of underlying pathology. TIVA also causes a decrease in intracranial pressure (ICP), and $CMRO_2$, reduces ischemia, and maintains cerebral autoregulation. One of the major indications for TIVA is when intraoperative neurophysiological monitoring is required, with the main modality being somatosensory-evoked and motor-evoked potentials.[9,10] In this case, the patient underwent total intravenous anesthesia with neuroprotective principles. The anesthesia method in the case presented consisted of induction of anesthesia using a combination of midazolam 2 mg, fentanyl 150 µg IV, lidocaine 80 mg IV, thiopental 300 mg IV, and rocuronium 50 mg IV. These medications were continued with maintenance using fentanyl 50 µg/hour, propofol 50 mg/hour, and atracurium 15 mg/hour.

There are several medications that have cerebral protective effects. The administration of thiopental has been noted as an effective approach in preventing postoperative neurologic deficits due to its potent cerebral vasoconstrictive properties. Various mechanisms have been proposed to explain the cerebral protective effects of thiopental, including the augmentation of cerebral oxygenation and the reduction of the cerebral metabolic rate of oxygen, thereby diminishing oxygen consumption in ischemic regions, redistributing cerebral blood flow from hyperperfusion areas to hypoperfusion areas, and having anti-oxidative effects. The primary mechanism for brain protection is to reduce $CMRO_2$ to 55-60%, where the EEG becomes isoelectric.[9,11,12]

Propofol is an induction agent commonly used in TIVA, which can reduce postoperative nausea and vomiting and has antioxidant effects. Propofol also has neuroprotective effects in reducing cerebral metabolic rate, intracranial pressure, and increasing CPP. Additionally, although not a general anesthetic agent, lidocaine is often administered during induction and can also decrease CMR, CBF, and ICP. A moderate bolus (0.5 mg/kg) of lidocaine will increase regional CBF, whereas a large dose (5 mg/mg) results in a moderate decrease in CBF and $CMRO_2$. The main advantage is that it lowers CBF (by increasing cerebral vascular resistance) without causing other significant hemodynamic effects. Lidocaine may also have neuroprotective effects. Lidocaine infusion is used in some centers as an adjunct to general anesthesia to reduce the need for opioids.[9,11,12] Optimal analgesia is critical in the neurosurgical population because uncontrolled pain leads to increased ICP and unstable hemodynamics, both of which can affect recovery and patient outcomes. Adequate analgesia also reduces the incidence of postoperative nausea, vomiting and respiratory complications.[13–15]

Hypothermia is the recommended method for brain protection during focal and global ischemia. Hypothermia is typically employed within the first hour following complete circulatory arrest. It exerts its effects by reducing basal metabolic and electrical demands across the entire brain. Notably, metabolic requirements decline even after the cessation of electrical activity, a phenomenon not observed with anesthetic agents. Furthermore, hypothermia has the additional benefit of attenuating the production of free radicals and other mediators involved in ischemic injury. Next are anesthetic agents such as barbiturates, etomidate, propofol, isoflurane, desflurane, and sevoflurane, which can produce burst suppression. All agents except desflurane and sevoflurane can produce a complete loss of electrical activity in the brain and eliminate the metabolic 'cost' of electrical activity. Unfortunately, these agents have no effect on basal energy requirements. However, with the exception of barbiturates, the effects are not similar in that the agents affect different parts of the brain to varying degrees. Maintenance of CPP is essential as hypotension, increased venous pressure, and increased ICP must be avoided. Hyperglycemia potentiates neurological injury after focal or global ischemia, so blood glucose should be maintained below 180 mg/dl. Normocarbia should be maintained because hypercarbia and hypocarbia do not have a beneficial effect on cerebral ischemia; hypocarbia induces cerebral vasoconstriction and can worsen ischemia, while hypercarbia can cause cerebral vasodilation phenomena and worsen intracellular acidosis.[4] In this case, the patient did not undergo special hypothermia prevention procedures.

CONCLUSION

Good perioperative neuro anesthesia management is needed to maintain the patient's hemodynamic stability and prevent conditions that could worsen the condition of patients undergoing craniotomy procedures. Total intravenous anesthesia is a complete general anesthesia method used in all intravenous agents, where the benefits of this method are used in neurosurgery, including accelerating the patient's return from the effects of anesthesia, faster recovery of cognitive function, as well as reducing intracranial pressure and the risk of ischemia.

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CONFLICT OF INTEREST

The author declares there is no conflict of interest.

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