JNI Jurnal Neuroanestesi Indonesia Case Report

Anaesthetic Management for Patient with Trigeminal Neuralgia underwent Microvascular Decompression (MVD)

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Received: May 19, 2024; Accepted: May 22, 2024; Publish: June 27, 2024
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Abstract

Trigeminal neuralgia (TN) is a chronic pain with repeated brief episodes of electric shock-like pain affecting the fifth cranial nerve. Microvascular decompression (MVD) is one of treatments for TN. Anesthetic management for MVD requires special consideration to reduce brain volume (slack brain) and optimise Mean Arterial Pressure (MAP). Female 29-yo, 40kgbw with chief complaint: throbbing pain and intermittent stiffness in right facial area since 1 year ago. Brain MRI examination showed crossing of right superior cerebellar artery (RSCA) branch with right trigeminal nerve near the root entry zone and underwent MVD. Anesthesia using smooth intubation technique and maintenance using a combination of inhalational anaesthetics (sevoflurane 1 vol%) and intravenously (propofol 100mcg/kg/minute, remifentanil 0.2mcg/kgbw/min, and rocuronium 10mcg/kgbw/min). Target for MAP (90mmHg) and $EtCO_2$ (30mmHg). We didn't use mannitol for slack brain. Early emergence with smooth extubation to prevent sudden haemodynamic changes and minimising coughing then for early neurological detection of intracranial complications.The combined use of sevoflurane < 1MAC and continuous propofol provides optimal visualisation of the operating area. This combination reduces cerebral blood flow which makes the brain slack and keeps MAP optimal to maintain cerebral perfusion pressure and reduce the risk of cerebral ischemia. The combination of these agents also makes early recovery for more rapid neurological assessments. Anaesthesia management for MVD uses neuroanesthesia principles, balanced anaesthesia, and strict haemodynamic monitoring. The combination of inhalation anaesthetic sevoflurane and intravenous propofol gave optimise visualisation in the operation area and the patient's recovery can be enhanced.

Keywords: Microvascular decompression, neuroanesthesia, trigeminal neuralgia

J. neuroanestesi Indones 2024;13(2): 77–85

I. Introduction

Trigeminal neuralgia (TN), also known as tic douloureux, is a chronic pain condition characterised by recurrent brief episodes of electric shock-like pain affecting the fifth cranial (trigeminal) nerve, which innervates the forehead, cheek and lower jaw. The condition is almost always unilateral and can involve one or more parts of the trigeminal nerve. TN is a syndrome characterised by paroxysmal facial pain.[1] There are two types of TN, Type 1 as intermittent pain and Type 2 as constant pain representing distinct clinical, pathological, and prognostic entities. Although several mechanisms involving peripheral pathology at the root (compression or traction), and dysfunction of the brainstem, basal ganglion, and cortical pain modulation mechanisms may play a role, neurovascular conflict is the most accepted theory.[2]

The lifetime prevalence of TN is estimated to be 0.16%-0.3%, while the annual incidence is 4–29 per 100,000 people/year. The prevalence of TN is found more in women than men (F:M ratio 3:2).

doi: https://doi.org/10.24244/jni.v13i1.565
ISSN (Print): 2088-9674 ISSN (Online): 2460-2302

JNI is accredited as Sinta 2 Journal: https://sinta.kemdikbud.go.id/journals/profile/796

How to cite: Hardian R, et al, "Anaesthetic Management for Patient with Trigeminal Neuralgia underwent Microvascular Decompression (MVD)".

The incidence increases with age, with a mean age of onset of 53–57 years and a range of 24–93 years in the adult series. Furthermore, a recent paediatric headache clinic of 1040 identified five children in the age range of 9.5–16.5 years with TN.[3] Microvascular decompression is the surgical treatment of choice in TN which is resistant to medical treatment.[2] Microvascular decompression is generally performed in younger and healthier patients, especially patients with isolated pain in the eye nerve or in all three branches of the trigeminal nerve, or patients with secondary trigeminal neuralgia. The prognosis of cure is 80% and the recurrence rate is among the lowest among all invasive procedures for pain intervention (recurrence rate 20% at 1 year, 25% at 5 years). [4]

Anaesthetic consideration is needed to prevent complication during microvascular decompression procedures especially when the location is in fossa posterior. Anaesthetic management for MVD requires special consideration to decrease brain volume (slack brain) and optimise Mean Arterial Pressure (MAP). Anaesthetists should keep in mind that the potential problems may arise during posterior fossa surgery, including possible injury to vital brainstem centres, pneumocephalus, as well as unusual patient positioning, C-spine injury and upper airway swelling or decubitus injury to dependent parts. Other considerations include the potential use of Intra Operative Neurophysiological Monitoring (IONM).[5] In this case report a 29 year old, female, with trigeminal neuralgia underwent microvascular decompression (MVD).

II. Case Report

Mrs MO, 29 years old, 40 kg in body weight, height 156 cm tall, BMI 16 kg/m2 (undernutrition) with chief complaint throbbing pain and stiffness in the right facial area since 1 year ago She had no history of weakness in facial muscles, opened mouth within normal limits, ate and drank well, history of seizures (-), nausea (-), vomiting (-). No history of past illness. Physical Examination: Patient compos mentis, GCS E4V5M6, pupil isokor, light reflex positive, Airway clear, RR 18x/min, vesicular sound, no rales, no wheezing, SpO_2 98% room air. Heart rate 82x/mnt, blood pressure 130/82 mmHg. Examination for abdomen and extremity within normal limit. Examination for cranial nerves within normal limit. Laboratory findings: Hb 12.8 g/dL, Hct 38,4%, WBC 8.700/mm3, Plt 345.000/mm3, PT 11", aPTT 24.4", INR 1.0, blood glucose 116 mg/dL, AST/ALT 19/43 U/L. Chest X-ray: cor pulmo within normal limit. ECG: sinus rhythm, HR 75x/mnt, hypertrophy (-), ischaemic (-).

MRI Head with contrast: MRI Brain parenchyme normal, No visible infarction/bleeding/tumour. From 3D Cube T2HR it's showed right N. V:

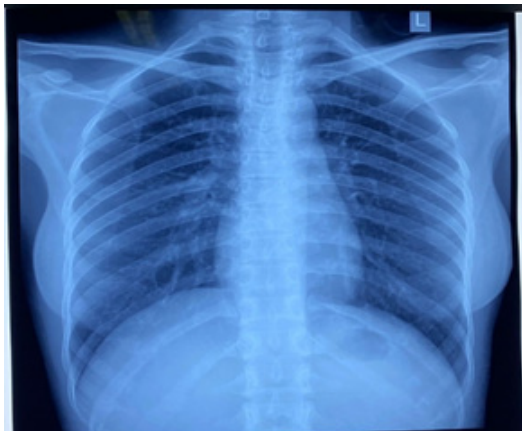

Figure 1. Chest x-ray

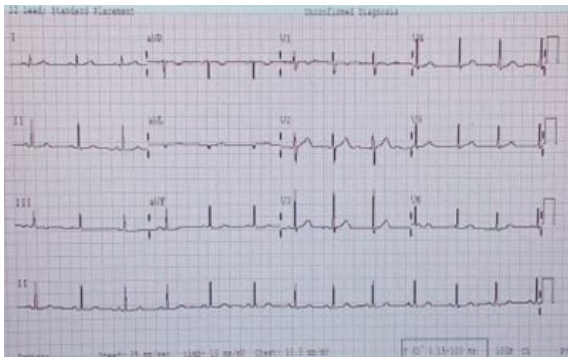

Figure 2. Electrocardiogram

RSCA branch crossing the right N. V near the root entry zone; Left N. V: No visible neuro-vascular conflict; N. VII, VIII right and left: No visible neuro-vascular conflict.

Impression: MRA within normal limits, no visible stenosis of intracranial major arteries. No visible

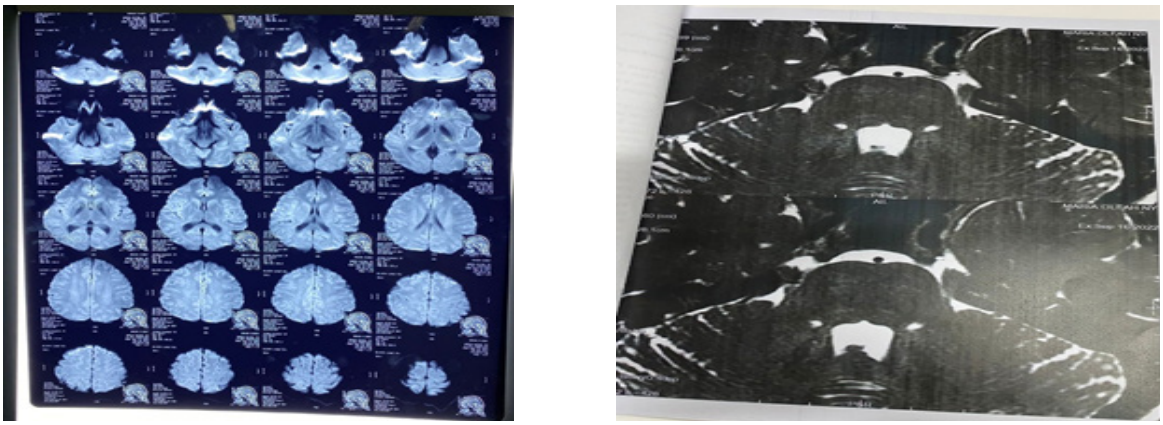

Figure 3: MRI and MRA Images of the Head Axial-Sagittal-Coronal Sections

aneurysma/vascular malformation.

The patient was concluded as physical status ASA I; no medical problems. with surgical problems were narrowed operating field, risk of bleeding and risk of prolong operation. And anaesthetic problem included how to make slack brain, risk of neuropraxia from park bench position, risk of increased ICP and risk of hipothermia.

Preoperative Anaesthesia

In the operating table, patient was in supine position. Vital signs: HR 85 x/mnt, BP 129/79 mmHg, SpO_2 99% room air. We applied non invasive blood pressure (NIBP), pulse oxymetri and End tidal CO_2 ($EtCO_2$) monitor. Arterial line for measuring Intraaretrial blood pressure (IABP) was inserted after the intubation.

Intraoperative

We performed smooth intubation with remifentanil as strong analgetic with loading dose 1 mcg/kgbw in one minute and continued 0.2–0.4 mcg/kgbw/minutes. For induction we were using propofol 2 mg/ kgbw intravenously followed by continued dose of 100 mcg/kgBB/minute. For intubation we used rocuronium 0.8 mg/kgbw followed by continuing dose of 10 mcg/ kgbw/ minute. We inserted endotracheal tube (ETT) size 7.0 and connected to breathing circuit in anaesthesia machine. We're used VC-CMV, FiO_2 50%, VT 280 ml, RR 14 times/minute, I:E ratio 1:2, PEEP 5 cmH_2O. After the intubation, patient was positioned in park bench position. Head Pin was inserted, after positioning the patient, all monitoring and ventilators were checked and the patient was in good condition so that surgery could begin. Intraoperative, we used combination remifentanil, propofol and sevoflurane 1vol%, rocuronium intermitent if needed.

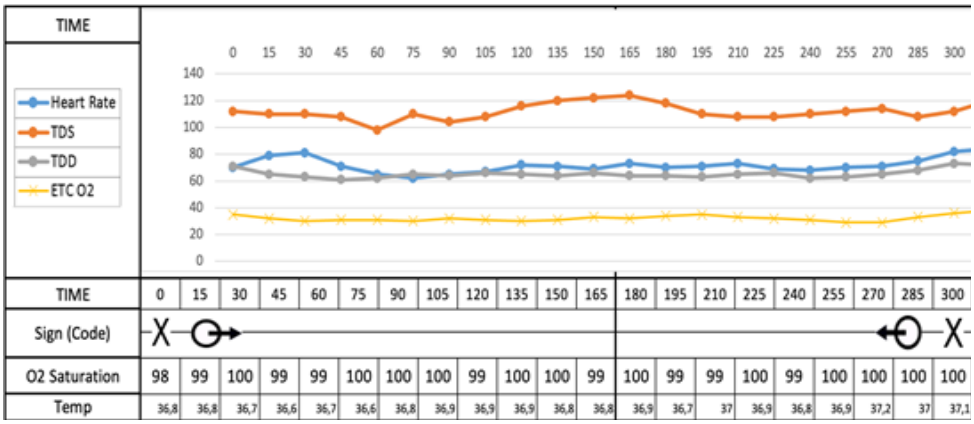

Figure 3. Intraoperative Hemodynamic Monitoring

Table 3. Follow Up Condition in ICU

Parameters	POD 1	POD 2
S	Shortness (-), swallowing (+), speech (+), dizziness (<), good consciousness (+), pain (<), nausea (-), vomiting (-), eating (+), drinking (+), chills (-) fever (-)	Shortness (-), swallowing (+), speech (+), dizziness (-), good consciousness (+), pain (<<), nausea (-), vomiting (-),eating (+), drinking (+), chills (-) fever (-)
O	B1: airway clear, spontaneous breathing, RR 18 x/ min Rh -/-, Wh -/-, no retraction SpO_2 100% on NC 3 lpm B2: BP 111/79 (89) mmHg, HR 75x/m regular, strong lifting. B3: E4V5M6 3mm/3mm isochor pupil R. Light +/+ NRS still 1/10 motion 2/10 B4: BAK (+) via DC, UO 586cc/15 hours, (39cc/ hour), BC +515cc/15 hours B5 : distension (-), Mrs (+), supple (+), tenderness (-), tympanic (+) B6 : T 36.6^{o}C, acral warmth Edema (-/-)	B1: airway clear, spontaneous breathing, RR 18 x/min Rh -/-, Wh -/-, no retraction SpO2 100% on room air B2: BP 100/58 (71) mmHg, HR 71x/ min regular, strong lift. B3: E4V5M6 3mm/3mm isochor pupil R. Light +/+ NRS still 0/10 motion 1/10 B4: BAK (+) via DC, UO 1056cc/24 hours, (44cc/hour), BC +321cc/24 hours B5: distension (-), Bu (+), supple (+), tenderness (-), tympanic (+) B6: T 36.7^{o}C, warm acral Edema (-/-)
A	Post op MVD ec TN	Post op MVD ec TN
P	F: soft rice diet TKTP 3x1 inf. NaCL 0.9% 1000cc/24hours inf. RL 500cc/24hours A: drip tramadol 100mg in 100cc NS discharged in 5 min /12h, Inf. Paracetamol 3x1gram, PO. Carbamazepine 2x200mg S : - T : - H : 30° head-up, mobilisation, seated U : - G : Periodic GDS/day (142 mg/dl) Injection. Inj. Ceftriaxon 2x1 gr H1	F: high calory and protein, rice diet 3x1 inf. NaCL 0.9% 1000cc/24hours inf. RL 500cc/24hours A: drip tramadol 100mg in 100cc NS discharged in 5 min /12h, PO. Paracetamol 3x500mg, Carbamazepine 2x200mg S : - T : - H : Head-up 30°, mobilisation, sitting, standing U : - G : Periodic GDS/day (157 mg/dl) Injection. Inj. Ceftriaxon 2x1 gr H2

Emergence

Once the surgery was confirmed to be completed and the patient had been returned to the supine position and the head pin removed, a re-evaluation of vital signs and the effect of muscle relaxants was performed. Reversal agents were given, namely neostigmin 1.5 mg and sulfas atropine 0.5 mg. Smooth extubation process with remifentanil analgesic continues to run 0.05mcg/kgBB/ minute, ensure adequate breathing, oxygenation was achieved, and the patient could open her eyes and respond cooperatively.

Postoperative Anesthesia

During postoperative analgesics we gave tramadol 100mg and paracetamol 3x1 gram intravenously. Oral administration of carbamazepine 2x200mg The patient was transferred to the ICU for further observation. The MVD procedure took 5 hours long with minimal bleeding.

III. Discussion

Trigeminal neuralgia (TN) is defined as a condition of sudden, acute, severe, stabbing, and recurrent pain distributed over the branching of the trigeminal nerve. Diagnosis is based on history of illness and pain characteristics. Pain occurs paroximally, where each pain may last for a few seconds to minutes. The frequency of pain varies with attacks occurring hundreds of times or prolonged in a single day. The condition can be triggered by mild sensitisation of the trigeminal nerve region, such as eating, talking, washing the face, or cleaning the teeth.[6] Based on population studies, the lifetime prevalence of trigeminal neuralgia is 0.16–0.3% and the patient incidence is 12.6–27.0 per 100000 people/year. Trigeminal neuralgia is also seen in more women (60%) than men (40%). Onset generally occurs at the age of 53–55 years.[7] Based on the patient's history and physical examination, the patient's symptoms lead the diagnosis to trigeminal neuralgia. Based on the International Classification of Headache Disorders (IHCD) and International Association for the Study of Pain (IASP) subclassifications, trigeminal neuralgia can be divided based on causes such as idiopathic (unknown), classical (vascular compression causing changes in nerve root morphology), and secondary (comorbid conditions such as tumours).[7]

Some evidence indicates the involvement of neurovascular conflict in changes in trigeminal nerve morphology, such as distortion, dislocation, distension, indentation, flattening or atrophy being one of the main causes. In addition, the aetiology of TN may resulted from hyperactivity of primary afferents that induce secondary central sensitisation of neurons in the trigeminal nucleus or central changes. There is also a theory that one of the causes of TN is focal demyelination of the trigeminal primary afferent near the trigeminal root towards the pons. This demyelination is resulted in a phase of hyperexcitability so that the nerve experiences continuous impulses. In secondary TN, the pathophysiological mechanism is the same as classic TN, but the underlying conditions are different. Secondary TN is caused by the presence of lesions such as tumours, meniongiomas, arteriovenous malformations, and aneurysms pressing on the trigeminal nerve region.[7]

Based on the supporting examination and radiographic images, the patient showed a branch of the right subclavian artery (RSCA) crossing the right trigeminal nerve near the root entry zone (REZ). This crossing can be classified as classic TN. According to the American Academy of Neurology and European Federation of Neurological Societies (EFNS) guidelines, pharmacological therapy is the first-line treatment for TN cases.[2,8] Carbamazepine (CBZ) can be the first-line pharmacological therapy for TN. In addition, the combination of lidocaine and magnesium has been effective in some patients. In addition to medical intervention, operative management can be performed if the patient shows resistance to the use of pharmacological therapy. One of the operative management options is Microvascular Decompression. The MVD technique is indicated in patients with classic TN or secondary TN, with neurovascular compression.[2]

The aim of MVD therapy is to relieve pressure on the cranial nerve.[9] With a relaxed brain (slack brain), the cerebellum is retracted. If there are blood vessels around the cranial nerves, they should be freed. Once free from compression, the dura is closed, other layers are also closed. The operating position of the patient can be performed differently such as lateral, prone, supine, or sitting.[10] Based on systematic reviews and meta-analyses, MVD management in TN can relieve pain in 76% of patients with an average follow-up of 1.7 years.[11] In this case, the patient was positioned in park bench which is a modification of the lateral position for MVD procedures. The use of this position is generally good for exploration of posterior fossa lesions. In the park-bench position, the head is rotated towards the shoulder contralateral to the lesion, without exceeding 30 degrees of lateral rotation. The following steps for the position: The roll is placed under the contralateral upper part of the chest, avoiding placing it directly under the axilla as the brachial plexus and axillary vessels will be compressed; the entire bony end should be protected; the

depressed arm hangs over the end of the table on an arm support or suspended with padded straps;

The ipsilateral hand is placed across the chest with the elbow in slight flexion; the ipsilateral knee is positioned in extension and the depressed knee is positioned in flexion; a pillow should be placed between the knees; lateral support is placed at the sternal region and at the interscapular region and thereafter the patient is secured across the pelvic region with padded straps. Several complications can be seen in the use of the park-bench position such as pressure sores, upper limb paralysis, tongue swelling or delayed airway obstruction.[12] However, no complications were found in this patient after the operation. Three things that must be considered in the anaesthetic management of posterior fossa surgery are the effect of intravenous vs inhaled drugs to keep air from entering the venous circulation, maintain adequate cerebral perfusion pressure (CPP), and maintain cardiovascular response when brainstem manipulation is performed. Intravenous access can withstand a better threshold than inhaled administration, reducing the severity of the impact in the event of air embolism.[13]

In this case, the patient was induced and maintained anaesthesia using the principle of anaesthesia balance. The patient was given a loading dose of remifentanil at 48 cc/hour for 1 minute, propofol at 80mg and rocuronium at 30mg. Then, remifentanil was given at 4.8 cc/hour, propofol at 24 cc/hour and rocuronium was given at 2.4 cc/hour continuously. The principle of balancing anaesthesia is the combination of drugs used to weaken the central nervous system. The combination of all drugs increases the positive aspects of all drugs. The goal of anaesthesia maintenance in neurosurgery is to control cerebral pressure through cerebral metabolic oxygen consumption rate ($CMRO_2$) and cerebral blood flow (CBF). Specific anaesthetic preparations are drug combinations that benefit cerebral haemodynamics, $CMRO_2$, and intracranial pressure (ICP) to provide good operating conditions and increase the likelihood of a quality outcome.[14]

The use of opioid-type drugs in anaesthesia balances shows various positive effects, such as a strong analgesic effect that can reduce perioperative pain, reduce anxiety, reduce somatic and autonomic responses such as respiratory tract manipulation, and produce haemodynamic stability when there is a noxious stimulus associated with surgery.[14] In addition, strong analgesia can aid in better postoperative recovery and its synergistic properties help to reduce the need for propofol and other hypnotic agents. The use of remifentanil, as one of the opioid drugs, produces little change in CBF and Vmca which essentially has minimal effect on intracranial pressure. Remifentanil is originally designed as a fast-acting opioid, but has a fast elimination rate.[13] The time it takes to reach blood-brain balance is only about 1–2 minutes. Infusion of 0.05–0.1 mcg/kgb/min provides analgesia with a concentration of 1–3 ng/mL in the blood. The rapid elimination of remfitanil avoids drug accumulation.[15] The MVD technique requires space for the surgeon to identify and relieve the problematic nerve pressure without excessive brain retraction in the process.[10] Anaesthetic management for MVD requires special consideration to decrease brain volume (slack brain) and optimise mean arterial pressure (MAP) to maintain cerebral perfusion pressure (CPP). One of the best anaesthetic induction agents in reducing brain volume is propofol (1–2 mg/kgBW). Propofol has a direct activity effect of cerebral vasoconstriction which can help avoid cerebral steal. ICP is also reported to be lower in patients using propofol as anaesthesia than isoflurane or sevoflurane, so it can be given as a therapy for intracranial hypertension. However, mean arterial blood pressure (MABP) monitoring should still be performed. CO_2 reactivity$_2$ is also maintained using propofol, so hyperventilation may lower ICP when propofol is used. In addition, propofol is also known to have neuroprotective effects. Some of the neuroprotective mechanisms include CMR reduction, antioxidant activity, GABA receptor activation, prevention of mitochondrial swelling, and endocannaboid system interaction. These benefits have led to propofol being widely used in neurological surgery.[13]

The muscle paralyser used is rocuronium. Rocuronium has a rapid onset, making it an appropriate drug as a muscle paralyser for intubation. Rocuronium is the best alternative because of its rapid onset and little effect on intracranial dynamics. Rocuronium also has the fewest side effects compared to drugs of the same class due to less histamine release.[13] Sevoflurane was used as a volatile agent in the MVD procedure for this TN case patient. Sevoflurane is a methyl isoprophylether derivative that exhibits anaesthetic effects of enhancing postsynaptic inhibition of GABA and glycine channel activity and inhibiting excitatory synaptic channel activity of NMDA, nicotinic acetylcholine, serotonin, and glutamate in the CNS. It is a halogenated anaesthetic delivered by a vaporiser to the lungs. The low blood/gas coefficient of sevoflurane coupled with the following properties suggest advantages in the neurosurgical setting: rapid onset, rapid offset, and nondistinctive disruption of cerebral haemodynamics. Indeed, in the case of long-duration neurosurgery (mean minimum alveolar concentration (MAC) time: 4.7 hours), intracranial surgery patients anaesthetised with sevoflurane (40% O_2) reported a shorter time to emergence and postoperative neurological assessment than patients anaesthetised with isoflurane (40% O_2) (n=60).[16] Sevoflurane was used in this case due to its cerebral vasodilating effect and the lowest CBF increase of all anaesthetic gases. Sevoflurane can decrease CBF due to its effect of decreasing cerebral metabolism at concentrations less than 1 MAC. This effect is unique given that sevoflurane is a volatile anaesthetic, as in-vitro and in vivo experiments have previously shown that other volatiles such as halothane, desflurane and isoflurane are potent vasodilators that increase CBF even though they decrease cerebral metabolism. Sevoflurane also has neuroprotective effects in the form of antiapoptosis. The decrease in cardiac output by sevoflurane is also lower than isoflurane or halothane, thus avoiding excessive fluid administration or the use of vasoconstrictors. [10,17] Fluid requirements in the MVD technique generally use standard rumoured fluids. Normal saline can be administered at 2–4 ml/kg/hour. The administration of mannitol as much as 0.25–0.5 gr/kgbb is sometimes also done to help reduce adequate brain volume (slack brain) for a better surgical field. In addition, hyperventilation to achieve a $PaCO_2$ of 25–30 mmHg can help lower ICP so that the working space can be increased.18. The use of cerebrospinal fluid drainage during dura opening can help increase the working space so as to accelerate the decompression process. In this patient to maintain slack brain by hyperventilating with a target $EtCO_2$ of 30mmHg to achieve $PaCO_2$ 25–30mmHg, without using mannitol, as well as the use of 1000cc of NS rumour fluid during surgery and 500cc for preloading. Haemodynamic monitoring is very important in patients undergoing neurosurgery. This includes close monitoring of arterial blood pressure and use of ECG to detect myocardial infarction. Pulse oximetry and $EtCO_2$ are also used to screen for venous air embolism which is one of the complications of MVD techniques in various positions.

The patient is also at risk of hypothermia, which requires temperature management. In addition, a catheter may be used to check and evaluate urine output after the administration of intravenous fluids. Sometimes, the use of EMG and SSEP are also performed to monitor the fascial nerve.[18] Hyperventilation can lower ICP and relax the brain. These two phenomena are related: ICP is measured with the cranium closed and is determined by all the contents of the cranium, whereas brain relaxation refers to the size and firmness of the brain in relation to the capacity of the cranium which is generally assessed by surgeons during craniotomy. Hyperventilation causes a reduction in CBV related to cerebral vasoconstriction, one of the components of intracranial contents; thus, reducing ICP and relaxing the brain. In healthy volunteers, hyperventilation causes a decrease of approximately 30% in CBF but only a 7% decrease in CBV when the arterial partial pressure of blood CO_2 ($PaCO_2$) decreases from normocapnia (41 mmHg) to hypocapnia (25 mmHg). Hypocapnia induced by hyperventilation can increase brain metabolic activity through several different mechanisms. Hypocapnia increases neural excitability and seizure duration, leading to increased oxygen and glucose

consumption, production of excitatory amino acids, and a switch to anaerobic metabolism and a switch to anaerobic metabolism causing a leftward shift of the oxygen-hemoglobin dissociation curve and inhibition of the usual negative feedback reaction by which low pH limits endogenous organic acid production.[19] The goals of anaesthesia during the emergence process are to prevent a sudden increase in blood pressure, rapid awakening, return of motor strength, and minimise coughing and pressure on the ETT.

Hypertensive conditioning or haemodynamic stabilisation can be done by administering esmolol or SNP by infusion. An antiemetic 30 minutes prior to extubation using metoclopramide 20 mg or ondansentron 4–8 mg can be given. Administration of acetaminophen or paracetamol 1 g IV or local anaesthesia to the scalp head may also be considered.[18] The conditions for faster extubation are determined by the condition and extent of the surgery (post-operative brain oedema). If there is extension to the medullary structures and the presence of oedema, the airway should be maintained until the patient wakes up. Additional anaesthesia may be given until the patient can return to normal. A complication to watch out for is postoperative nausea vomiting (PONV) so multimodal therapy according to the above indications and directions can be performed.[9] Carbamazepine administration of 2x200mg was given to postoperative patients. Several case studies have shown that additional pharmacological therapy is needed to relieve pain in patients after MVD therapy in TN patients. The first line for this therapy is anticonvulsants such as carbamazepine and oxcarbazepine. The use of carbamazepine can cure or relieve pain in patients by 58-100%. However, some things must be considered such as the use of carbamazepine in Asians with HLA-B*1502 genetics can show hypersensitivity reactions such as Steven-Jhonsons Syndrome.[20] Post-operative care may be performed in the intensive care unit due to some feared complications such as cerebral oedema, cerebral haemorrhage, LCS leakage and vascular rupture. During monitoring, the patient showed haemodynamic stability with a compos mentis consciousness score. The patient complained of pain at the surgical site with a scale of 3. Tramadol 100 mg in 100 cc NS was administered within 5 minutes/12 hours, carbamazepine 2x200mg, and paracetamol 3x1 gram were given as postoperative analgesics. In addition, additional drugs such as ondansentron 3x8mg and omeprazole 1x40 mg as antiemetic and gastric care were given. Ceftriaxone 2x1gram was administered through IV as infection prophylaxis. After 5 days, the patient was discharged

IV. Conclusion

Anaesthetic management in a trigeminal neuralgia patient undergoing microvascular decompression (MVD) using combination TIVA and inhalational agent sevoflurane give good brain relaxation and provide optimal visualization in the operative area. In this case, we used the principles of neuroanesthesia, anaesthetic balancing, and close haemodynamic monitoring to optimize visualization in the operating field, minimize the bleeding and early extubation after the operation.

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