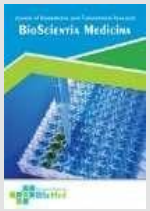
eISSN (Online): 2598-0580



Bioscientia Medicina: Journal of Biomedicine & Translational Research

Journal Homepage: www.bioscmed.com

Navigating Frontal Lobe Arteriovenous Malformation Resection: A Case Report on TIVA with Propofol-Remifentanil TCI for Hemodynamic Stability and ICP Control

I Gede Catur Wira Natanagara[1*], I Putu Pramana Suarjaya[2], Ida Bagus Krisna Jaya Sutawan[2]

[1]Student, Anesthesiology and Intensive Therapy Study Program, Faculty of Medicine, Universitas Udayana/Prof. Dr. I.G.N.G. Ngoerah General Hospital, Denpasar, Indonesia

[2]Lecturer, Anesthesiology and Intensive Therapy Study Program, Faculty of Medicine, Universitas Udayana/Prof. Dr. I.G.N.G. Ngoerah General Hospital, Denpasar, Indonesia

ARTICLE INFO

Keywords:
Brain arteriovenous malformations
Craniotomy
Intracranial pressure
Invasive monitoring
Propofol

***Corresponding author:**
I Gede Catur Wira Natanagara

E-mail address:
caturwira@hotmail.com

All authors have reviewed and approved the final version of the manuscript.

https://doi.org/10.37275/bsm.v9i8.1353

A B S T R A C T

Background: Arteriovenous malformations (AVMs) located within the frontal lobe present considerable anesthetic challenges. These challenges arise from the critical functions governed by this brain region and the inherent risks associated with intracranial surgery, notably hemodynamic instability and the potential for elevated intracranial pressure (ICP). The utilization of Total Intravenous Anesthesia (TIVA) through Target-Controlled Infusion (TCI) systems for propofol and remifentanil provides a sophisticated strategy for achieving precise control over anesthetic depth and maintaining physiological homeostasis. This report offers a detailed account of such a case. **Case presentation:** A 25-year-old male patient, classified as ASA III, presented with a right frontal lobe Spetzler-Martin Grade I AVM and was scheduled for elective microsurgical resection. The anesthetic management centered on a TIVA approach, employing propofol administered via an Eleveld TCI model (target concentration range: 2-5 µg/mL) and remifentanil via a Minto TCI model (target concentration range: 4-6 ng/mL). Comprehensive intraoperative monitoring included invasive arterial blood pressure and central venous pressure. Pharmacological adjuncts included mannitol, dexamethasone, and tranexamic acid. Throughout the procedure, stable intraoperative hemodynamics (target Mean Arterial Pressure [MAP] 70-90 mmHg) were successfully maintained, and intracranial pressure was effectively controlled, thereby facilitating the complete AVM resection. The patient was extubated in the post-operative period, demonstrated a stable neurological status, and was subsequently managed in the Intensive Care Unit (ICU). **Conclusion:** A meticulously planned and executed TIVA-TCI regimen, featuring propofol and remifentanil, when integrated with thorough invasive monitoring and proactive pharmacological interventions, demonstrated effectiveness in preserving crucial hemodynamic stability and fostering optimal intracranial conditions. This comprehensive anesthetic strategy was instrumental in the successful surgical resection of a frontal lobe AVM and contributed to a favorable neurological outcome for the patient.

1. Introduction

Cerebral arteriovenous malformations (AVMs) represent congenital vascular anomalies defined by an aberrant direct conduit between arteries and veins, critically bypassing the normal intervening capillary network. This structural anomaly culminates in the formation of a "nidus," a tangle of abnormal vessels where high-pressure arterial blood is shunted directly into thin-walled venous structures. Such exposure renders these venous components susceptible to

progressive dilatation, the formation of associated aneurysms, and, most critically, the risk of spontaneous hemorrhage. The clinical manifestations of AVMs are diverse; however, intracranial hemorrhage stands as the most devastating initial presentation in a substantial proportion of diagnosed individuals, reported to be between 38% and 68% of cases, with a predilection for occurring within the brain parenchyma (approximately 82% of hemorrhages). Other common presentations encompass seizures, the insidious development of progressive neurological deficits, and persistent or severe headaches. Although AVMs are inherently congenital, their clinical presence typically emerges in young adulthood, most frequently between the ages of 20 and 50 years. The reported incidence of AVMs ranges from 0.89 to 1.42 per 100,000 person-years, though the actual prevalence is considered to be higher owing to a significant number of asymptomatic cases that remain undiagnosed.[1,2]

AVMs situated within the frontal lobe, as exemplified in the present case report, introduce a distinct set of clinical and anesthetic considerations. The frontal lobe is responsible for orchestrating critical executive functions, shaping personality and behavior, facilitating language processing (particularly in the dominant hemisphere), and governing motor control. Surgical interventions within this eloquent and functionally vital area demand exceptionally meticulous anesthetic management. The primary goals of such management are to preserve these indispensable neurological functions while simultaneously establishing optimal surgical conditions for the neurosurgeon. Several factors have been identified as increasing the risk of AVM rupture, including a frontal lobe location, the presence of deep venous drainage patterns, a deeply situated nidus, and the coexistence of associated aneurysms.[3,4]

Surgical resection continues to be a cornerstone therapeutic modality for AVMs, especially for those deemed to carry a high risk of future rupture or those that have already become symptomatic. The fundamental objective of surgical intervention is the complete obliteration of the AVM nidus, thereby aiming to prevent subsequent hemorrhagic events and to alleviate or resolve existing neurological symptoms. Despite its therapeutic efficacy, the procedure is intrinsically associated with substantial potential risks. These include, but are not limited to, intraoperative hemorrhage, the development of cerebral ischemia, significant postoperative cerebral edema, and the challenging phenomenon known as normal perfusion pressure breakthrough (NPPB). NPPB describes a complex pathophysiological state characterized by cerebral hyperperfusion, leading to subsequent vasogenic edema or even new hemorrhage, which can manifest in brain tissue surrounding a resected AVM. This occurs when chronically hypoperfused tissue, due to vascular steal by the AVM, is suddenly exposed to normal physiological perfusion pressures following the AVM's removal and the restoration of normal circulatory pathways.[5,6]

The anesthesiologist's contribution to the multidisciplinary team managing AVM surgery is pivotal and encompasses several critical objectives. These include the stringent maintenance of hemodynamic stability to ensure adequate cerebral perfusion pressure (CPP) without inducing episodes of hypertension that could precipitate intraoperative bleeding from the AVM or surrounding vessels. Concurrently, meticulous control of intracranial pressure (ICP) is essential. Further objectives involve the optimization of cerebral oxygen delivery and consumption, the provision of a relaxed, or "slack," brain to facilitate optimal surgical exposure and minimize retraction injury, and the facilitation of a rapid and smooth emergence from anesthesia to enable prompt and reliable early neurological assessment.[7,8]

Total intravenous anesthesia (TIVA) has garnered increasing prominence and acceptance in the field of neuroanesthesia, largely due to its favorable pharmacological profile concerning cerebral hemodynamics and ICP. Specifically, the application of TIVA utilizing target-controlled infusion (TCI)

systems for the administration of anesthetic agents such as propofol and remifentanil permits a highly precise and rapidly titratable control over the depth of anesthesia. This level of precision is of paramount importance in these delicate and often lengthy neurosurgical procedures. Propofol, a cornerstone of TIVA, characteristically reduces the cerebral metabolic rate for oxygen ($CMRO_2$), which in turn leads to a coupled and beneficial decrease in cerebral blood flow (CBF) and, consequently, a reduction in ICP. Importantly, propofol generally preserves the cerebrovascular reactivity to carbon dioxide (CO_2), allowing for physiological manipulation of CBF when necessary. Remifentanil, an ultra-short-acting opioid agonist, provides potent intraoperative analgesia and effectively blunts sympathetic nervous system responses to intense surgical stimuli. This contributes significantly to maintaining hemodynamic stability. Its principal advantage lies in its rapid metabolic degradation by non-specific plasma and tissue esterases, ensuring a swift offset of clinical effect upon discontinuation of the infusion, thereby contributing to prompt postoperative neurological evaluation.[9,10]

The complexity of anesthetic management is further amplified by the need for judicious fluid administration to maintain euvolemia, the inherent potential for massive intraoperative blood loss requiring preparedness for transfusion, and the necessity for specific adjunctive pharmacological interventions. These interventions often include the use of osmotic diuretics like mannitol and corticosteroids such as dexamethasone to actively manage cerebral edema and control ICP. The employment of comprehensive invasive monitoring techniques, most notably intra-arterial blood pressure measurement and central venous pressure monitoring, is indispensable for providing real-time, continuous hemodynamic assessment and for guiding timely and appropriate therapeutic interventions.

This case report aims to describe in detail the comprehensive anesthetic management of a 25-year-old male patient who presented with a right frontal AVM, classified as Spetzler-Martin Grade I, and underwent successful microsurgical resection. The core anesthetic technique implemented was TIVA, with propofol and remifentanil delivered via advanced TCI systems, and this was supplemented by meticulous invasive hemodynamic monitoring and the proactive use of pharmacological adjuncts designed to optimize intraoperative physiological conditions and enhance overall patient safety. The novelty of this report is centered on the detailed, sequential exposition of a balanced and multimodal anesthetic strategy, specifically tailored to address the unique physiological and surgical challenges presented by the resection of a frontal lobe AVM. It particularly emphasizes the synergistic interplay between the TIVA-TCI technique, the achievement of targeted physiological control parameters, and the implementation of proactive measures to effectively mitigate known perioperative risks, such as significant ICP elevation and deleterious hemodynamic volatility. The aim of this report is, therefore, to present and thoroughly discuss the successful application of this contemporary anesthetic approach.

2. Case Presentation

A 25-year-old male, with a body weight of 83 kg and a height of 173 cm, resulting in a body mass index (BMI) of 27.7 kg/m^2, was diagnosed with a malformation of the arteries and veins (AVM) situated in the right frontal region of his brain. This AVM was graded as Spetzler-Martin Grade I. The planned surgical intervention was a craniotomy for the microsurgical resection of this AVM. The patient's medical history revealed that he had been experiencing intermittent headaches for the duration of one year prior to this admission. Notably, one year before this presentation, he had an acute episode characterized by a sudden decrease in his level of consciousness, which was associated with projectile vomiting and weakness affecting his left-sided extremities. A computed tomography (CT) scan performed at that time confirmed the presence of an intracranial hemorrhage, which necessitated an urgent craniectomy procedure for the evacuation of

the blood clot. Following this emergency neurosurgical intervention, his level of consciousness improved, and he was eventually discharged from the hospital in a stable condition.

During the pre-anesthetic evaluation conducted for the scheduled AVM resection, the patient was found to be fully conscious, alert, and cooperative, with a Glasgow coma scale (GCS) score of E4V5M6. He continued to report the intermittent headaches as his primary symptom. He specifically denied experiencing any nausea, vomiting, seizures, fever, or shortness of breath. While he did report a recent history of a cough and cold approximately two weeks earlier, he denied any personal history of chronic conditions such as hypertension, diabetes mellitus, asthma, or other significant systemic illnesses. The airway assessment revealed a Mallampati Class II, and there were no loose teeth, no signs of peripheral edema, and his capillary refill time was brisk at less than 2 seconds. The neurological examination was thorough: his pupils were equal in size (3mm bilaterally) and demonstrated a normal, brisk reaction to light; there were no meningeal signs elicited; and no lateralizing neurological deficits were observed. Motor system examination showed symmetrical power, graded at 5/5 in all four extremities, with normal muscle tone, trophism, and physiological reflexes; no pathological reflexes were present. His respiratory system examination was unremarkable; his respiratory rate was 16 breaths per minute, auscultation revealed clear vesicular breath sounds bilaterally, and his oxygen saturation (SpO_2) was 98% while breathing room air. Cardiovascular system assessment showed a blood pressure reading of 117/76 mmHg and a heart rate of 78 beats per minute with a regular rhythm; no cardiac murmurs or gallops were audible. Examination of the abdomen found it to be soft and non-tender, with normal bowel and bladder function reported by the patient. A summary of the patient's pertinent clinical findings is presented in Table 1.

Routine laboratory investigations were conducted, including a complete blood count (which showed normal hematocrit, hemoglobin, leukocyte, and platelet levels), serum electrolytes, liver function tests (SGOT, SGPT), and renal function tests (creatinine); all results were within their respective normal ranges. The coagulation profile, encompassing Prothrombin Time (PT), Activated Partial Thromboplastin Time (APTT), and International Normalized Ratio (INR), was also normal. Both a chest X-ray and an electrocardiogram (ECG) were performed and revealed no abnormalities. Advanced neuroimaging studies provided crucial details regarding the AVM. A CT angiography study visualized the AVM in the right frontal lobe, which was noted in this report as Grade II (this contrasts with the Spetzler-Martin Grade I mentioned at diagnosis), and also showed evidence of associated intraventricular hemorrhage (IVH) and surrounding cerebral edema. A subsequent magnetic resonance imaging (MRI) scan of the head confirmed the presence of the AVM nidus in the right frontal region and provided an estimated volume of the subacute intracerebral hemorrhage at approximately 6.5 cc. This MRI also clearly depicted perifocal edema surrounding the lesion and the existing defect from the previous craniectomy procedure. Further detailed findings from the MRI included the identification of feeding arteries originating from branches of the M4 segment of the right middle cerebral artery (MCA) and the anterior cerebral artery (ACA), with the draining veins identified as leading to the superficial cortical vein via an anterior anastomosis. Additional incidental findings noted on the MRI included areas of encephalomalacia, evidence of small vessel ischemic changes, chronic ethmoidal sinusitis, bilateral maxillary retention cysts, and a deviated nasal septum. The magnetic resonance angiography (MRA) findings were consistent with the MRI concerning the AVM's precise location and its vascular supply and drainage pathways. Based on the comprehensive assessment of his neurological pathology, the patient was classified as belonging to the American Society of Anesthesiologists (ASA) physical status III category. A detailed discussion regarding the proposed anesthetic plan and its associated risks was held with the patient during the pre-anesthetic visit. Written informed

consent was duly obtained, explicitly covering general anesthesia, the necessity for insertion of central venous and arterial catheters for monitoring, the potential requirement for blood product transfusion, and a comprehensive outline of the potential risks and complications associated with both the anesthetic procedure and the surgery itself, including perioperative morbidity and mortality.

The procedure of treatment and follow-up is summarized in Table 2. On the day of the scheduled surgery, prior to the induction of anesthesia, premedication was administered intravenously (IV). This consisted of dexamethasone 10 mg, midazolam 2 mg, and fentanyl 25 μg. An arterial line was meticulously inserted into the left radial artery, facilitated by local anesthesia with 2% lidocaine at the puncture site, after confirming adequate collateral circulation in the hand via a positive Allen's test. The induction of general anesthesia was achieved using a combination of propofol administered via TCI (utilizing the Eleveld pharmacokinetic model, with an initial plasma target concentration set to 3–5 μg/mL) and remifentanil also via TCI (utilizing the Minto pharmacokinetic model, with an initial effect-site target concentration of 4–6 ng/mL). Muscle relaxation to facilitate endotracheal intubation was achieved with rocuronium 0.6 mg/kg IV, and lidocaine 80 mg IV was also administered to blunt the cardiovascular and intracranial pressure responses to laryngoscopy and intubation. The patient's trachea was successfully intubated with a non-kinking endotracheal tube (ETT) of size No. 7.5; correct bilateral lung ventilation was confirmed by auscultation and capnography, and the ETT was then securely fixed in place. Following intubation, a central venous catheter was also inserted for central venous pressure monitoring and vascular access.

Anesthesia was subsequently maintained with a continuous infusion of propofol via TCI, with the target plasma concentration adjusted to a range of 2–3 μg/mL, and remifentanil via TCI, with the target effect-site concentration maintained at 4–6 μg/mL throughout the surgical procedure. Intermittent bolus doses of rocuronium, typically 0.1 mg/kg, were administered approximately every 45 minutes as required to maintain an adequate level of neuromuscular blockade. Mechanical ventilation was precisely controlled, with tidal volumes set at 6-8 ml/kg of ideal body weight, and the respiratory rate was adjusted dynamically to maintain an end-tidal CO_2 ($ETCO_2$) level between 30–35 mmHg. The inspired oxygen fraction (FiO_2) was maintained at 0.5–0.6. The patient was carefully positioned supine on the operating table, with his head slightly extended and turned towards the right, and securely immobilized using a Mayfield headrest.

Throughout the intraoperative period, the patient's hemodynamic parameters were closely and continuously monitored. Arterial blood pressure was vigilantly maintained within a target range of 98–127 mmHg systolic and 63–81 mmHg diastolic, which corresponded to a mean arterial pressure (MAP) of approximately 70–90 mmHg. The heart rate was kept between 61–89 beats per minute. Infusions of norepinephrine and nicardipine were prepared and readily available for immediate administration should rapid intervention be required for blood pressure management. To actively manage intracranial pressure and reduce brain bulk, thereby optimizing surgical exposure, mannitol 0.5 g/kg IV was administered as a slow infusion. Additionally, tranexamic acid 1 gram IV was given as an antifibrinolytic agent to minimize potential intraoperative blood loss. Fluid management was carefully conducted using balanced salt solutions; the total intravenous fluid input during the procedure was 1500 ml of crystalloids, against an estimated blood loss of 300 ml over the 5-hour duration of the surgery. Urine output was consistently maintained at approximately 1 ml/kg/hr.

During the surgical dissection, the AVM located in the right frontal region was clearly identified by the neurosurgical team. The observed feeding arteries and draining veins were found to be consistent with the findings from the preoperative digital subtraction angiography (DSA) (although DSA was not explicitly

mentioned in the initial imaging reports, its use is standard for detailed AVM angioarchitecture assessment and is implied by the phrase "sesuai hasil DSA pre-operatif" from the discussion in the source document). The microsurgical resection of the AVM was performed meticulously, following the standard neurosurgical principles of initial devascularization of the identified feeding arteries, followed by careful circumferential dissection of the nidus, and finally, management of the draining veins towards the completion of the nidus resection. After the AVM resection was deemed complete by the surgeon, the absence of any residual nidus components was confirmed both by direct visual inspection under the operating microscope and with the aid of intraoperative Doppler ultrasonography. Following this confirmation, the dura mater was closed meticulously to ensure a watertight seal. An autologous bone flap, which had been previously harvested and stored in a subgaleal pocket (presumably from the patient's prior craniectomy procedure), was then prepared and carefully re-implanted to repair the cranial defect; this bone flap was securely fixed in place using mini plates and screws.

At the conclusion of the surgical procedure, any residual neuromuscular blockade was reversed using a combination of neostigmine and glycopyrrolate (standard reversal doses assumed, as specific doses were not provided). The continuous infusions of propofol and remifentanil were then discontinued. The patient emerged smoothly and promptly from anesthesia. He was extubated in the operating room once protective airway reflexes had fully returned, he demonstrated adequate spontaneous ventilation, and he was able to follow commands, indicating satisfactory cognitive recovery. Post-extubation, he was noted to be conscious and alert, with a GCS of E4V5M6, and his hemodynamic parameters were stable.

Subsequently, the patient was transferred to the Intensive Care Unit (ICU) for close observation, continuous monitoring, and ongoing postoperative management. His postoperative analgesia regimen consisted of a continuous intravenous fentanyl infusion (prepared as 400 µg of fentanyl in 50 mL of 0.9% NaCl, infused at a rate of 2.1 mL/hour, equating to approximately 16.8 µg of fentanyl per hour) and intravenous paracetamol 1 gram administered every 6 hours. In the ICU, his neurovital signs, including GCS and pupillary responses, were meticulously monitored on an hourly basis. Prophylactic anticonvulsant therapy was administered to prevent the occurrence of postoperative seizures (the specific agent was not mentioned but is standard neurosurgical practice). The patient's course in the ICU was uneventful. He was subsequently transferred to the neurosurgery ward in a stable condition. His neurological status remained stable, with no new deficits noted upon subsequent follow-up assessments.

3. Discussion

The successful anesthetic management of the 25-year-old male patient undergoing microsurgical resection of a right frontal lobe arteriovenous malformation (AVM), as presented, hinged upon a meticulously orchestrated interplay of advanced pharmacological techniques, comprehensive physiological monitoring, and a profound understanding of both cerebrovascular pathophysiology and the specific demands of neurosurgery. Arteriovenous malformations represent a direct conduit between the arterial and venous systems, bypassing the high-resistance capillary bed. This abnormal connection, the nidus, exposes the thin-walled venous structures to abnormally high arterial pressures, leading to a cascade of potential pathological changes including venous hypertension, progressive dilatation of the draining veins, formation of intranidal or flow-related aneurysms, and, critically, an elevated risk of hemorrhage.

Table 1. Summary of patient's clinical findings.

Parameter	Finding
Demographics & vitals	
Age	25 years
Weight	83 kg
Height	173 cm
BMI	27.7 kg/m²
Blood pressure (Pre-op)	117/76 mmHg
Heart rate (Pre-op)	78 beats/minute, regular
Respiratory rate (Pre-op)	16 breaths/minute
SpO_2 (Room Air, Pre-op)	98%
Presenting complaints	
Primary symptom	Intermittent headaches for 1 year
Past neurological event	1 year prior: Decreased consciousness, projectile vomiting, left-sided weakness (ICH on CT)
Current symptoms (Denied)	Nausea, vomiting, seizures, fever, dyspnea
Past medical history	
Previous surgery	Craniectomy for clot evacuation (1 year prior)
Other conditions (Denied)	Hypertension, diabetes, asthma, other systemic illnesses
Recent illness	Cough and cold (2 weeks prior)
Neurological examination	
GCS	E4V5M6 (Compos Mentis)
Pupils	Isokor, 3mm/3mm, reactive to light
Meningeal signs	Negative
Lateralization	None
Motor strength	Symmetrical 5/5 in all extremities
Tone, trophism, reflexes	Normal physiological reflexes, no pathological reflexes
Airway assessment	
Mallampati score	Class II
Laboratory findings	
CBC, electrolytes, LFTs, RFTs	All within normal limits
Coagulation profile (PT, APTT, INR)	Normal
Chest X-ray, ECG	No abnormalities
Neuroimaging	
Diagnosis	Right Frontal AVM, Spetzler-Martin Grade I
CT angiography	Right frontal lobe AVM (Grade II reported here), IVH, cerebral edema
MRI head	Nidus AVM frontal kanan, subacute ICH (~6.5cc), perifocal edema, post-kraniektomi defect
MRI feeders/drainage	Feeders: Right MCA (M4) & ACA branches. Drainage: Superficial cortical vein (anterior anastomosis)
MRA head	Consistent with MRI on AVM location, feeders, drainage
ASA classification	ASA III

Table 2. Summary of treatment procedure and follow-up.

Aspect	Detail
Premedication (IV)	Dexamethasone 10 mg, Midazolam 2 mg, Fentanyl 25 µg
Invasive line placement	Left radial arterial line (Allen's test positive, 2% lidocaine), Central Venous Catheter
Anesthesia induction (TCI)	Propofol (Eleveld model, target 3–5 µg/mL), Remifentanil (Minto model, target 4–6 ng/mL)
Intubation adjuncts	Rocuronium 0.6 mg/kg IV, Lidocaine 80 mg IV
Airway management	ETT non-kinking No. 7.5, bilateral ventilation confirmed, ETT fixed
Anesthesia maintenance (TCI)	Propofol (target 2–3 µg/mL), Remifentanil (target 4–6 ng/mL)
Muscle relaxation (Maintenance)	Rocuronium intermittent 0.1 mg/kg approx. every 45 mins
Ventilation parameters	Controlled ventilation, FiO_2 0.5–0.6, $ETCO_2$ 30–35 mmHg
Patient positioning	Supine, head slightly extended, turned right, Mayfield headrest
Intraoperative hemodynamic targets	BP 98–127/63–81 mmHg (MAP ~70-90 mmHg), HR 61–89x/min
Vasoactive drug preparedness	Norepinephrine and Nicardipine prepared
ICP/Brain bulk management	Mannitol 0.5 g/kg IV slow
Antifibrinolytic	Tranexamic acid 1 gram IV
Fluid management	1500 ml crystalloids IV input
Estimated blood loss (EBL)	300 ml
Urine output	Approx. 1 ml/kg/hr
Surgical procedure	Microsurgical AVM resection: devascularization of feeders, nidus dissection, management of draining veins
Confirmation of resection	Visual (microscope) and intraoperative Doppler ultrasound: No residual nidus
Closure	Watertight dural closure, autologous bone flap re-implantation with mini plates/screws
Emergence & extubation	Neuromuscular blockade reversal (Neostigmine/Glycopyrrolate), Propofol/Remifentanil discontinued. Extubated in OR.
Immediate post-op status	Conscious (GCS E4V5M6), hemodynamically stable
Postoperative care unit	Intensive Care Unit (ICU)
Postoperative analgesia	Fentanyl infusion (400µg in 50mL 0.9% NaCl @ 2.1mL/hr), Paracetamol 1g IV Q6H
Postoperative monitoring	Hourly neurovital signs (GCS, pupils)
Seizure prophylaxis	Anticonvulsant administered
Follow-up outcome	ICU course uneventful, transferred to neurosurgery ward, neurological status stable, no new deficits

The "vascular steal" phenomenon associated with AVMs is also of paramount importance; the low-resistance shunt of the AVM can divert blood flow from adjacent healthy brain tissue, potentially leading to chronic ischemia, gliosis, and neurological deficits even in the absence of overt hemorrhage. This chronic hypoperfusion can also impair local cerebrovascular autoregulation, rendering the surrounding brain tissue exquisitely sensitive to changes in systemic blood pressure, a factor that becomes particularly relevant during the post-resection phase with concerns for Normal Perfusion Pressure Breakthrough (NPPB).[11,12]

The location of the AVM in the right frontal lobe, as in this case, carries specific implications. The frontal lobes are integral to higher cognitive functions,

including executive planning, working memory, emotional regulation, social behavior, and, in the dominant hemisphere, language production (Broca's area). The right frontal lobe, while non-dominant for language in most individuals, is still crucial for visuospatial skills, attention, and aspects of emotional processing. Surgical manipulation in or near these eloquent areas requires an anesthetic technique that not only provides optimal surgical conditions but also minimizes the risk of postoperative neurological deficits. Anesthetic agents and techniques must be chosen to preserve cerebral perfusion, avoid exacerbating intracranial pressure, and allow for rapid emergence to facilitate early and accurate neurological assessment. The patient's history of a previous hemorrhage from the AVM, requiring a craniectomy for clot evacuation, underscored the fragile nature of this particular lesion and heightened the imperative for meticulous perioperative control of factors known to precipitate re-bleeding, such as hypertension or sudden increases in ICP. The Spetzler-Martin Grade I classification, typically indicating a smaller, more superficial lesion with superficial venous drainage and located in non-eloquent cortex, generally predicts a more favorable surgical risk profile. However, any intracranial surgery, particularly for a vascular lesion, carries inherent risks.[13,14]

A comprehensive preoperative evaluation is foundational to safe neuroanesthetic practice. In this patient, the ASA III classification accurately reflected the systemic impact of a significant neurological condition, despite otherwise normal organ function. The detailed neurological examination confirmed a stable baseline with no new focal deficits, which is crucial for interpreting any postoperative changes. The review of imaging (CT, MRI, MRA) provided the neuroanesthesiologist and neurosurgeon with a precise anatomical roadmap of the AVM, including its feeding arteries (branches of the right MCA and ACA) and draining veins (superficial cortical vein). This information is vital for anticipating the degree of surgical difficulty, potential for blood loss, and areas particularly at risk during dissection. The presence of perifocal edema and a previous hemorrhage suggested a degree of existing brain tissue compromise and an inflammatory response.[15,16]

The premedication regimen was thoughtfully chosen. Dexamethasone (10 mg IV) is a potent synthetic glucocorticoid with strong anti-inflammatory and anti-edema effects. Its administration aims to reduce vasogenic edema surrounding the AVM, thereby improving brain compliance, reducing baseline ICP, and potentially facilitating surgical exposure. While its efficacy in the context of AVMs without significant mass effect or edema can be debated, its use is common, particularly if imaging suggests perilesional edema. Midazolam (2 mg IV) provided necessary anxiolysis and sedation, reducing the patient's stress response prior to entering the operating room, which can help prevent undesirable catecholamine-driven hemodynamic fluctuations. The dose was modest, minimizing the risk of respiratory depression or excessive sedation that could delay emergence. Fentanyl (25 μg IV) served multiple purposes: it provided a degree of preemptive analgesia, blunted the hemodynamic response to painful stimuli such as arterial line insertion, and contributed to a smoother induction sequence by reducing the required dose of induction agents. This multimodal premedication strategy aimed to create a calm, hemodynamically stable patient primed for the induction of anesthesia.[17,18]

The decision to employ TIVA with propofol and remifentanil administered via TCI systems was central to the anesthetic management and reflects contemporary best practice in neuroanesthesia for several compelling reasons. Propofol is an intravenous anesthetic agent that acts primarily through potentiation of GABA-A receptor activity, leading to widespread neuronal inhibition. Its effects on cerebral physiology are generally favorable for neurosurgery. Propofol induces a dose-dependent reduction in the cerebral metabolic rate for oxygen ($CMRO_2$). Due to the tight coupling between cerebral metabolism and blood flow (flow-metabolism coupling), this reduction in $CMRO_2$ leads to a corresponding decrease in cerebral

blood flow (CBF). The combined reduction in CBF and cerebral blood volume contributes to a decrease in intracranial pressure (ICP), which is highly desirable in patients at risk for intracranial hypertension or those requiring a slack brain for surgical access. Furthermore, propofol can exert neuroprotective effects in certain ischemic conditions, potentially by reducing excitotoxicity, scavenging free radicals, and stabilizing cell membranes, although the clinical significance of this in AVM surgery is still under investigation. Importantly, propofol generally preserves cerebrovascular autoregulation and CO_2 reactivity, allowing for physiological manipulation of CBF when indicated. The Eleveld pharmacokinetic model used for propofol TCI is one of several validated models that predict plasma or effect-site concentrations, allowing for more stable and predictable anesthetic depth compared to manual infusion schemes. The target plasma concentrations of 3-5 µg/mL for induction and 2-3 µg/mL for maintenance are within standard ranges for achieving adequate hypnosis and suppression of awareness while minimizing excessive cardiorespiratory depression.[19,20]

Remifentanil is an ultra-short-acting synthetic opioid agonist with high potency at µ-opioid receptors. Its primary advantage in neuroanesthesia is its unique pharmacokinetic profile: it is rapidly metabolized by non-specific plasma and tissue esterases, resulting in a very short context-sensitive half-time (approximately 3-5 minutes) irrespective of the duration of infusion. This allows for profound intraoperative analgesia and blunting of sympathetic responses to noxious stimuli (such as Mayfield pin application, skin incision, craniotomy, and dural manipulation) with rapid dissipation of effect upon discontinuation of the infusion. This rapid offset is invaluable for facilitating prompt emergence and neurological assessment at the end of the procedure. The Minto pharmacokinetic model used for remifentanil TCI targets the effect-site concentration, which more closely correlates with the clinical effect than plasma concentration, allowing for precise titration of opioid effect. The target effect-site concentrations of 4-6 ng/mL for induction and maintenance provided potent analgesia, contributing significantly to the observed intraoperative hemodynamic stability (BP 98–127/63–81 mmHg, HR 61–89x/min). By preventing wide swings in blood pressure and heart rate, remifentanil helps maintain stable CPP and minimizes stress on the AVM. The combination of propofol and remifentanil TCI offers a synergistic effect. Propofol provides hypnosis and reduces $CMRO_2$/CBF/ICP, while remifentanil provides potent analgesia and sympatholysis. This combination often allows for lower doses of each agent than if used alone, potentially reducing side effects. TIVA avoids the use of volatile anesthetic agents, which, while also reducing $CMRO_2$, can cause dose-dependent cerebral vasodilation, potentially increasing CBF and ICP, particularly in patients with impaired intracranial compliance. This vasodilatory effect of volatile agents can be particularly problematic during periods of dural closure before the bone flap is replaced, or in patients with large lesions causing significant mass effect. TIVA is also associated with a lower incidence of postoperative nausea and vomiting (PONV), which is beneficial as vomiting can cause straining and increase ICP in the postoperative period. The smooth and rapid emergence typically seen with propofol-remifentanil TIVA is critical for allowing immediate postoperative neurological evaluation, which can detect early signs of complications such as hematoma or stroke.

Secure airway management is paramount. The administration of rocuronium 0.6 mg/kg IV provided rapid and reliable neuromuscular blockade for endotracheal intubation. Rocuronium is a non-depolarizing muscle relaxant with a relatively fast onset, suitable for neurosurgical cases. The co-administration of lidocaine 80 mg IV prior to laryngoscopy and intubation is a common practice in neuroanesthesia. Lidocaine is thought to suppress the cough reflex and attenuate the hypertensive and ICP-elevating responses to airway manipulation by reducing afferent neural traffic from the airway and potentially through central effects. Intubation with a

non-kinking endotracheal tube (ETT No. 7.5) is standard to prevent airway obstruction, especially when the patient's head is manipulated or fixed in position for prolonged periods. Confirmation of bilateral ventilation is a critical safety check. The maintenance of MAP within the target range of 70-90 mmHg was crucial. This range was chosen to ensure adequate CPP while avoiding hypertension that could increase the risk of AVM rupture or excessive surgical bleeding. CPP is calculated as MAP minus ICP (or CVP, if higher than ICP). Assuming a normal ICP of 5-15 mmHg, a MAP of 70-90 mmHg would generally yield a CPP well above the critical threshold of 50-60 mmHg required to prevent ischemia in the healthy brain. However, in the context of an AVM with potential vascular steal and impaired autoregulation in surrounding tissues, slightly higher MAP values within the target range might be preferred by some practitioners to ensure perfusion to these vulnerable areas, while still avoiding excessive pressure on the AVM itself. The readiness of norepinephrine (an alpha- and beta-agonist vasopressor) and nicardipine (a dihydropyridine calcium channel blocker with arterial vasodilating properties) allowed for swift pharmacological correction of any deviations from the target blood pressure range, reflecting a proactive approach to hemodynamic management. The choice of nicardipine for hypertension is often favored in neuroanesthesia because it tends to preserve or even improve cerebral blood flow due to its cerebral vasodilatory effects, unlike some other antihypertensives that might reduce CBF.

Several strategies were employed to optimize intracranial conditions. The patient was positioned supine with the head slightly extended, turned to the right, and secured in a Mayfield headrest. Slight head-up tilt (15-20 degrees) promotes cerebral venous drainage, which can lower ICP. Care must be taken to ensure that the head rotation and fixation do not cause jugular venous compression, which would paradoxically increase ICP by impeding venous outflow. Mechanical ventilation was targeted to achieve an $ETCO_2$ of 30-35 mmHg, inducing a state of mild hypocapnia. $PaCO_2$ is a potent regulator of cerebrovascular tone; hypocapnia causes cerebral vasoconstriction, leading to a reduction in CBF, cerebral blood volume, and consequently ICP. This can result in a "slacker" brain, improving surgical exposure and reducing the need for aggressive brain retraction. However, excessive hypocapnia ($PaCO_2$ < 25-30 mmHg) must be avoided, as it can lead to critical reductions in CBF and cerebral ischemia. The chosen $ETCO_2$ range represents a balance between effective ICP reduction and the maintenance of adequate cerebral perfusion. The FiO_2 was maintained at 0.5-0.6 to ensure adequate arterial oxygenation (PaO_2 > 100 mmHg) while avoiding excessively high oxygen levels, as hyperoxia can also have complex effects on cerebral vasculature, potentially causing vasoconstriction in some circumstances or increasing oxidative stress. Mannitol (0.5 g/kg IV administered slowly) was used to reduce brain water content and ICP. Mannitol is an osmotic diuretic that, when administered intravenously, increases serum osmolality. This creates an osmotic gradient across the blood-brain barrier (BBB), drawing water from the brain parenchyma (where osmolality is lower) into the intravascular compartment (where osmolality is higher), thereby reducing brain volume and ICP. The effect typically begins within 15-30 minutes and can last for several hours. Slow administration is important to prevent an initial transient increase in intravascular volume that could elevate blood pressure and potentially ICP before the osmotic effect predominates. Mannitol also has rheological benefits, reducing blood viscosity and potentially improving microcirculatory flow. Repeated doses or continuous infusion would require monitoring of serum osmolality (target < 320 mOsm/L) and electrolytes (particularly sodium and potassium) to avoid complications like hypernatremia, hypokalemia, and renal dysfunction. Dexamethasone administered preoperatively would have contributed to reducing perilesional vasogenic edema, further aiding in brain relaxation. Its effects are genomic, taking several hours to become clinically apparent.

The goal of intraoperative fluid management in neurosurgery is to maintain euvolemia and electrolyte balance, typically using isotonic crystalloid solutions such as 0.9% saline or balanced salt solutions (PlasmaLyte, Hartmann's). In this case, 1500 ml of crystalloids were administered for an EBL of 300 ml during the 5-hour surgery, suggesting a relatively restrictive approach aimed at avoiding fluid overload which could exacerbate cerebral edema. Urine output was maintained at approximately 1 ml/kg/hr, indicating adequate renal perfusion. Tranexamic acid (1 gram IV) was administered as an antifibrinolytic. Tranexamic acid competitively inhibits the activation of plasminogen to plasmin, a key enzyme responsible for fibrin degradation. By preventing premature clot lysis, tranexamic acid can reduce intraoperative blood loss and the need for blood transfusion, particularly in surgeries with a high risk of bleeding like AVM resection. Its use in intracranial surgery is increasingly supported by evidence, provided contraindications are absent.

The surgical technique for AVM resection, involving meticulous microsurgical dissection with initial devascularization of feeding arteries followed by circumferential dissection of the nidus and final interruption of draining veins, is a standard and critical sequence. Early control of arterial feeders reduces blood flow into the AVM nidus, decreasing its turgor and minimizing bleeding during subsequent dissection. The draining veins are typically preserved until the very end of the resection to prevent premature venous occlusion, which could lead to acute swelling and hemorrhage of the AVM. The use of intraoperative Doppler ultrasonography to confirm complete nidus obliteration and the absence of residual AVM components is an important adjunct to visual confirmation under the microscope, as any residual nidus carries a high risk of future hemorrhage. The watertight dural closure and cranioplasty with the autologous bone flap are essential for preventing CSF leakage and protecting the brain. A significant concern following the resection of large or high-flow AVMs is the phenomenon of NPPB. Chronic shunting of blood through the low-resistance AVM can lead to chronic hypoperfusion and maximal vasodilation in the surrounding normal brain tissue due to the vascular steal effect. Autoregulation in these chronically ischemic capillary beds may become impaired. When the AVM is resected, blood flow is redirected from the shunt into these previously hypoperfused, maximally dilated, and poorly autoregulating vessels. This sudden exposure to normal or even slightly elevated perfusion pressures can lead to capillary leakage, vasogenic edema, and potentially intracerebral hemorrhage in the surrounding brain tissue. While this patient's AVM was Grade I, suggesting a lower flow state than higher-grade AVMs, the risk of NPPB, though perhaps lower, is never zero. Meticulous blood pressure control in the immediate post-resection period and extending into the postoperative phase is the cornerstone of NPPB prevention. This often involves maintaining MAP at the lower end of the normal range or even inducing mild hypotension, provided CPP remains adequate. The anesthesiologist plays a key role in this by ensuring a smooth transition to the postoperative period with tightly controlled hemodynamics.

The smooth and rapid emergence from anesthesia, leading to extubation in the operating room, is a testament to the favorable recovery characteristics of the propofol-remifentanil TIVA technique. Early extubation allows for immediate neurological assessment, which is crucial for detecting any new or evolving neurological deficits that might indicate a postoperative complication such as hematoma, ischemia, or significant edema. The patient's GCS of E4V5M6 post-extubation indicated an excellent immediate neurological outcome. Transfer to the ICU for close monitoring is standard practice after AVM resection. The postoperative management focused on several key areas. Hourly neurovital signs, GCS, and pupillary checks are essential for the early detection of any deterioration. Continued vigilance over blood pressure is critical to prevent both hypertension (which could precipitate re-bleeding or NPPB) and hypotension (which could cause ischemia). Adequate

pain control is vital for patient comfort and to prevent adverse physiological responses (hypertension, tachycardia) that can be triggered by pain. The multimodal approach using a continuous low-dose fentanyl infusion and regular intravenous paracetamol aimed to provide effective analgesia while minimizing opioid-related side effects such as respiratory depression or excessive sedation that could obscure neurological assessment. The administration of prophylactic anticonvulsants is common after craniotomy for AVMs. Seizures can be a presenting symptom of AVMs or can occur as a complication of surgery due to cortical irritation. Prophylaxis aims to reduce the incidence of early postoperative seizures, which can increase metabolic demand, elevate ICP, and potentially compromise neurological outcome. The choice of anticonvulsant would depend on institutional protocols and patient factors. The uneventful ICU course and subsequent stable neurological status confirm the success of the perioperative management strategy.

The anesthetic care provided in this case of frontal lobe AVM resection was comprehensive and aligned with modern neuroanesthetic principles. The use of TIVA with propofol and remifentanil TCI, coupled with meticulous physiological monitoring and proactive management of hemodynamics and ICP, created optimal conditions for surgery and facilitated a safe and rapid recovery. The successful outcome underscores the importance of a tailored anesthetic plan that addresses the specific pathophysiological challenges posed by cerebral AVMs and the intricacies of intracranial neurosurgery. The coordination within the multidisciplinary team, including neurosurgeons, anesthesiologists, and ICU staff, was undoubtedly pivotal in achieving this positive result. The detailed management of this case serves as a valuable illustration of how advanced anesthetic techniques can contribute significantly to patient safety and favorable neurological outcomes in high-risk neurovascular procedures. The careful titration of anesthetic agents to meet the dynamic needs of the surgery, the proactive measures to control brain bulk and bleeding, and the seamless transition to postoperative care collectively exemplify a high standard of neuroanesthetic practice.

4. Conclusion

The comprehensive anesthetic management detailed in this report for a 25-year-old male patient undergoing microsurgical resection of a right frontal lobe arteriovenous malformation proved to be highly effective and conducive to a successful surgical outcome and an uncomplicated neurological recovery. The chosen strategy, centered on Total Intravenous Anesthesia (TIVA) delivered via Target-Controlled Infusion (TCI) systems for propofol and remifentanil, in conjunction with meticulous invasive hemodynamic monitoring and proactive pharmacological interventions, demonstrably facilitated the maintenance of critical hemodynamic stability and the establishment of optimal intracranial conditions throughout the demanding neurosurgical procedure. This case compellingly illustrates that a well-structured, multimodal anesthetic approach, tailored to the specific pathophysiological challenges of a frontal AVM and the intricacies of intracranial surgery, is paramount. The successful navigation of this case was underpinned by several key factors: a thorough and insightful preoperative evaluation; the precise titration of anesthetic agents to achieve desired physiological endpoints while minimizing adverse effects; vigilant real-time monitoring of cardiovascular and neurological parameters; active and effective management of intracranial pressure using a combination of pharmacological and ventilatory strategies; and a seamless transition to the postoperative period ensuring continued stability and early detection of any potential complications. The TIVA-TCI technique, in particular, offered superior control over anesthetic depth and facilitated a rapid, high-quality emergence, which is invaluable for immediate postoperative neurological assessment. Ultimately, the favorable outcome achieved in this patient underscores the significant contribution of a sophisticated and individualized anesthetic plan to the

overall success of complex neurovascular surgery, reinforcing the importance of collaborative, evidence-based practice in this specialized field.

5. References

1. Hao Q, Zhang H, Han H, Jin H, Ma L, Li R, et al. Recurrence of cerebral arteriovenous malformation following complete obliteration through endovascular embolization. Transl Stroke Res. 2025; 16(2): 339–49.
2. Li Z, Han H, Ma L, Li R, Li A, Zhang H, et al. Venous aneurysms in unruptured supratentorial brain arteriovenous malformations: a protective factor against hemorrhagic stroke and insights into hemodynamic mechanisms. Eur Radiol. 2025; 35(5): 2660–9.
3. Chen Y, Wang C, Han H, Ma L, Li R, Li Z, et al. Long-term outcomes of endovascular embolization and stereotactic radiosurgery as the first-line treatment for ruptured arteriovenous malformations: a propensity score matched analysis using nationwide multicenter prospective registry data. Int J Surg. 2025.
4. Halim AX, Singh V, Johnston SC, Higashida RT, Dowd CF, Halbach VV, et al. Characteristics of brain arteriovenous malformations with coexisting aneurysms: a comparison of two referral centers. Stroke. 2002; 33(3): 675–9.
5. Lawton MT, UCSF Brain Arteriovenous Malformation Study Project. Spetzler-Martin Grade III arteriovenous malformations: surgical results and a modification of the grading scale. Neurosurgery. 2003; 52(4): 740–8; 748-9.
6. Ghosh I, Haldar R, Paul M, Agarwal A. Pregnancy with large arteriovenous malformation of tongue: Anesthetic challenges and conduct. A A Pract. 2021; 15(6): e01481.
7. Hiyoshi T, Shimizu K, Kimura S, Naritani T, Morimatsu H. Anesthetic management of a patient with Osler-Weber-Rendu syndrome with multiple pulmonary arteriovenous malformations and pheochromocytoma for femoral artificial bone replacement: a case report. JA Clin Rep. 2023; 9(1): 6.
8. Sundararaj R, Arulprakasam S, Tenzing E, Senthilnathan M. Abernethy malformation with pulmonary arteriovenous malformations in a pediatric patient: an anesthetic challenge. SBV J Basic Clin Appl Health Sci. 2025; 8(1): 27–9.
9. Neeta S, Rao R, Upadya M, Keerthi P. Arteriovenous malformation of face: a challenge to anesthesiologists. Anesth Essays Res. 2017; 11(3): 784–6.
10. Sim J-H, Lee J-H, Lee C-H, Ryu S-A, Choi S-S. Anesthetic management during cesarean delivery in a pregnant woman with ruptured cerebral arteriovenous malformation -A case report-. Anesth Pain Med. 2017; 12(3): 220–3.
11. Santos CDSE, Joyner DA, Tuma Santos CA, Grayson BE, Calimaran A, Bacon DR. Alternative to general anesthesia for a stat cesarean delivery in a patient with a large arteriovenous malformation involving the cervicomedullary junction in active labor. Case Rep Anesthesiol. 2020; 2020: 6893587.
12. Zanfini BA, De Martino S, Frassanito L, Catarci S, Vitale di Maio F, Giuri PP, et al. “Please mind the gap”: successful use of ultrasound-assisted spinal anesthesia for urgent cesarean section in a patient with implanted spinal cord stimulation system for giant chest wall arteriovenous malformation - a case report. BMC Anesthesiol. 2020; 20(1): 122.
13. Nedunchezhian AS, Praveen R, Sethuraman M, Varma S. Anesthetic challenges in a child with hereditary hemorrhagic telangiectasia with coexisting pulmonary and cerebral arteriovenous malformations for intracerebral

hematoma evacuation: a case report. J Clin Anesth. 2021; 74(110430): 110430.
14. Teig MK. Anesthetic management of patients undergoing intravascular treatment of cerebral aneurysms and arteriovenous malformations. Anesthesiol Clin. 2021; 39(1): 151–62.
15. Atallah. Cerebral arteriovenous malformation, cerebral cavernoma, and cerebral aneurysm: Management and outcome during pregnancy and puerperium with focus on neurosurgical side. J Anes Surg Res. 2022.
16. Süzer MA, Özhan MÖ, Çaparlar CÖ, Eşkin MB, Atik B. Airway management in general anesthesia for endovascular treatment of cerebral arteriovenous malformation: a retrospective observational study. Braz J Anesthesiol. 2022; 72(3): 359–64.
17. Agarwal R, Dhar M, Banerjee A, Vathulya M. Anesthesia and hemodynamic management in a pediatric arteriovenous malformation. Res Opin Anesth Intensive Care. 2022; 9(3): 252–3.
18. Aysel A, Aysan E, Tuncer B, Turan M, Erkılıç E. A rare case: Multiple mandibular arteriovenous malformation with high output: May a tooth extraction alone turn to a catastrophic condition? Open J Anesthesiol. 2023; 13(11): 221–5.
19. Khan MF, Khan MK, Nazir S, Shamim F. Surviving the nightmare: Massive bleeding from large intraoral arteriovenous malformation during airway management for angioembolization procedure. Case Rep Anesthesiol. 2024; 2024:6311200.
20. Zhao Z, Wang H, Min X, Li Z, Feng F. Controlled hypotension under rapid ventricular pacing technique in patients with cerebral arteriovenous malformation -a case report. Korean J Anesthesiol. 2025; 78(1): 79–84.