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Case Report

Propofol–Dexmedetomidine Total Intravenous Anesthesia for STA–MCA Bypass in Moyamoya Syndrome

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ABSTRACT

Moyamoya syndrome (MMS) is a progressive cerebrovascular disorder characterized by stenosis of the internal carotid arteries and the formation of fragile collateral vessels, predisposing patients to cerebral ischemia. In post-stroke and post-craniectomy patients, impaired autoregulation and altered intracranial anatomy present significant anesthetic challenges. Maintaining hemodynamic stability and adequate cerebral perfusion is essential during revascularization procedures such as temporal artery–middle cerebral artery (STA–MCA bypass). We report a 60-year-old man with prior hemorrhagic stroke and previous decompressive craniectomy secondary to MMS who was scheduled for superficial temporal artery–middle cerebral artery (STA–MCA) bypass. Magnetic resonance angiography (MRA) demonstrated occlusion of the right M2 segment of the middle cerebral artery (MCA) and right frontal gliosis. Anesthesia was induced using propofol with a Schnider target-controlled infusion (TCI) model (target effect-site concentration 5 µg/mL), fentanyl 150 µg, intravenous lidocaine 90 mg, and rocuronium 0.8 mg/kg and maintained with propofol TCI combined with a dexmedetomidine infusion. Intraoperatively, systolic blood pressure (SBP) ranged from 96 to 115 mmHg, diastolic pressure from 56 - 72 mmHg, heart rate (HR) from 56 to 72 bpm, and $EtCO_2$ from 29 to 34 mmHg. Depth of anesthesia monitoring using CONOX showed qCON values between 45 and 57. After STA–MCA anastomosis, superficial temporal artery and middle cerebral artery pressures were 72 mmHg and 54 mmHg, respectively, with a pressure gradient of 18 mmHg and satisfactory Doppler flow. The patient was monitored in the intensive care unit for 24 hours postoperatively with stable hemodynamics and a Glasgow Coma Scale (GCS) score of E4M6Vett and was gradually extubated without neurological complications. This case highlights that propofol–dexmedetomidine TIVA can provide stable hemodynamics, controlled cerebral blood flow, and cerebral metabolic demand and may help maintain cerebral perfusion balance during STA–MCA bypass in patients with impaired cerebral autoregulation.

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Introduction

Moyamoya disease (MMD) is a chronic cerebrovascular disorder characterized by progressive stenosis or occlusion of intracranial arteries, particularly the terminal internal carotid artery, resulting in fragile collateral vessel formation and the characteristic angiographic "puff of smoke" appearance [1]. Clinically, MMD may present with ischemic stroke, transient ischemic attack, intracranial hemorrhage, seizures, or headache.

Cerebral revascularization is commonly performed to restore cerebral perfusion and prevent recurrent stroke in symptomatic patients. Anesthetic management of moyamoya syndrome (MMS) is challenging because impaired cerebrovascular autoregulation and fragile collateral circulation increase sensitivity to fluctuations in blood pressure, carbon dioxide levels, and cerebral perfusion. Accordingly, the anesthetic management for superficial temporal artery–middle cerebral artery (STA–MCA) bypass should maintain stable hemodynamics while carefully balancing cerebral oxygen delivery with metabolic demand. This case report describes the anesthetic management of a patient with MMS undergoing STA–MCA bypass, emphasizing strategies to maintain hemodynamic stability and minimize perioperative ischemic or hyperperfusion complications.

Case Report

History

A 60-year-old man with a history of hemorrhagic stroke treated with decompressive craniectomy 9 months earlier was diagnosed with MMS on follow-up angiography. He had no signs of raised intracranial pressure and no previous anesthetic complications. His medical history included hypertension treated with candesartan 8 mg daily and unstable angina 7 years earlier, with no diabetes, renal disease, smoking, or alcohol use.

Physical Examination

On examination, the patient was fully alert and hemodynamically stable (BP 124/72 mmHg, HR 64 bpm, RR 18/min, temperature 36.5°C, VAS 7). Neurological examination showed a GCS score of E4M6V5, equal and reactive pupils (3 mm/3 mm), intact cranial nerve function, and no motor or sensory deficits. Physical examination revealed no other abnormalities, except for a right frontal skull defect consistent with prior craniectomy.

Additional Test

Routine laboratory tests were within normal limits. (Table 1). Chest radiography demonstrated no cardiopulmonary abnormalities.

MRA/MRV showed a right frontal skull defect with frontal gliosis. The right M2 segment of the middle cerebral artery was not seen suggesting vasospasm or occlusion. There was no intracranial aneurysm, arteriovenous fistula, or arteriovenous malformation was identified, and the dural venous sinuses were normal (Figure 1). SPECT (HMPAO) cerebral perfusion imaging showed reduced uptake in the lateral and medial right prefrontal regions consistent with focal hypoperfusion while the left hemisphere demonstrated relatively preserved perfusion (Figure 2).

Table 1- Laboratory Results

Examination	Value	Unit
HEMATOLOGY		
Hemoglobin	15.4	g/dL
Hematocrite	44.0	%
Leukocyte count	10,900	/uL
Platelet count	192,000	/uL
HEMOSTASIS		
Prothrombin time (PT)	9.7 (11)	seconds
Activated Partial Thromboplastin Time (aPTT)	29.7 (31.2)	
International Normalized Ratio (INR)	0.89	
BLOOD CHEMISTRY		
Blood Urea Nitrogen (BUN)	14.0	mg/dL
Creatinine	0.90	mg/dL
Albumin	4.23	g/dL
SGOT/SGPT	13/16	U/L
Random Blood Glucose	84	mg/dL
ELECTROLYTES		
Natrium	136	mmol/L
Potassium	4.2	mmol/L
Chloride	104	mmol/L

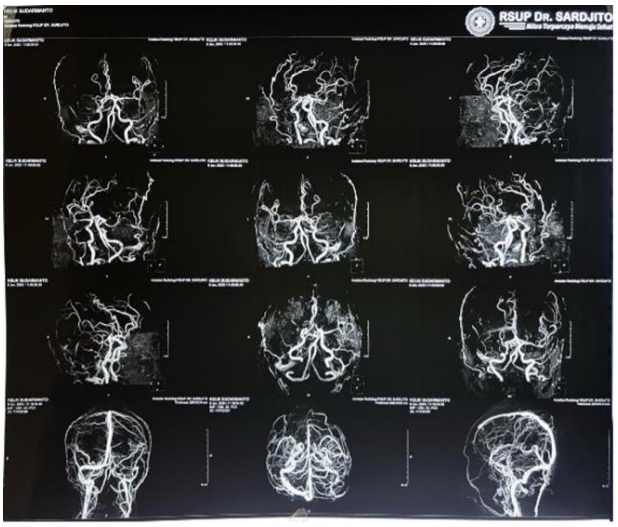

Figure 1- Preoperative MRA of the brain

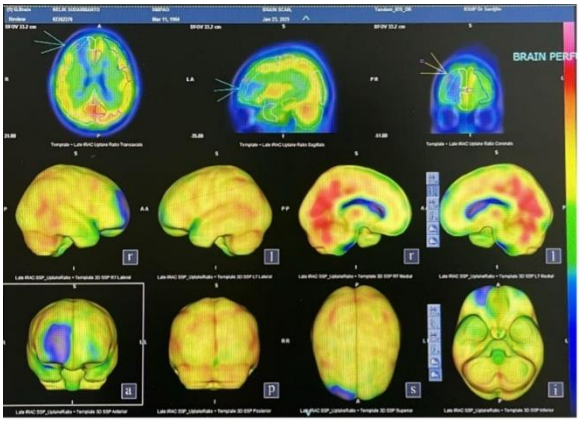

Figure 2- Contrast-enhanced brain MRI

Anesthetic Management

The patient was classified as ASA physical status III with MMD and a history of spontaneous intracerebral hemorrhage (SICH). Major perioperative concerns included bleeding, secondary brain injury, and intracranial pressure fluctuations.

Informed consent was obtained and standard ASA monitoring was established including invasive arterial blood pressure, $EtCO_2$, and depth-of-anesthesia monitoring (CONOX) and blood products were prepared. A peripheral 22G IV was inserted and maintenance of crystalloid initiated.

Baseline vital signs on operating room admission were stable (blood pressure 138/74 mmHg, HR 84 bpm, oxygen saturation 98% on room air, GCS E4M6V5). After preoxygenation, anesthesia was induced with fentanyl 150 µg (2.5 µg/kg), TCI propofol (Schnider model, Ce 5 µg/mL), lidocaine 90 mg, and rocuronium 0.8 mg/kg.

Mechanical ventilation (VCV) with a 50% oxygen–air mixture was used to maintain normocapnia. A scalp block was performed using 0.25% bupivacaine (24 mL). Anesthesia was maintained with propofol-based TIVA using TCI combined with dexmedetomidine infusion (0.3–0.5 µg/kg/h).

A total of 250 mL of medications and 1500 mL of crystalloid were administered during the 6-hour procedure. Urine output was 1,300 mL and estimated blood loss was 400 mL. Intraoperative hemodynamics remained stable (systolic blood pressure 96–115 mmHg, HR 56–72 beats/min, $EtCO_2$ 29–34 mmHg, qCON 45–57) (Figure 3). Following STA–MCA anastomosis, invasive measurements demonstrated mean pressures of 72 mmHg in the STA and 54 mmHg in the MCA, with Doppler confirmation of adequate graft flow. The brain appeared relaxed with preserved pulsatility. The patient was transferred to the intensive care unit with the endotracheal tube in situ for continued postoperative monitoring.

Postoperative Care

The patient was maintained in the intensive care unit for 24 hours postoperatively and was hemodynamically stable. Postoperative laboratory tests showed hemoglobin level of 12.5 g/dL and a hematocrit of 37% with all other parameters within normal limits. Sedation was stopped on postoperative day 1 and the patient regained consciousness within about 20 minutes. Neurological assessment demonstrated a GCS score of E4M6Vett, with the ability to follow commands and intact cough and swallowing reflexes. The patient was subsequently extubated uneventfully and transferred to the general ward later the same day.

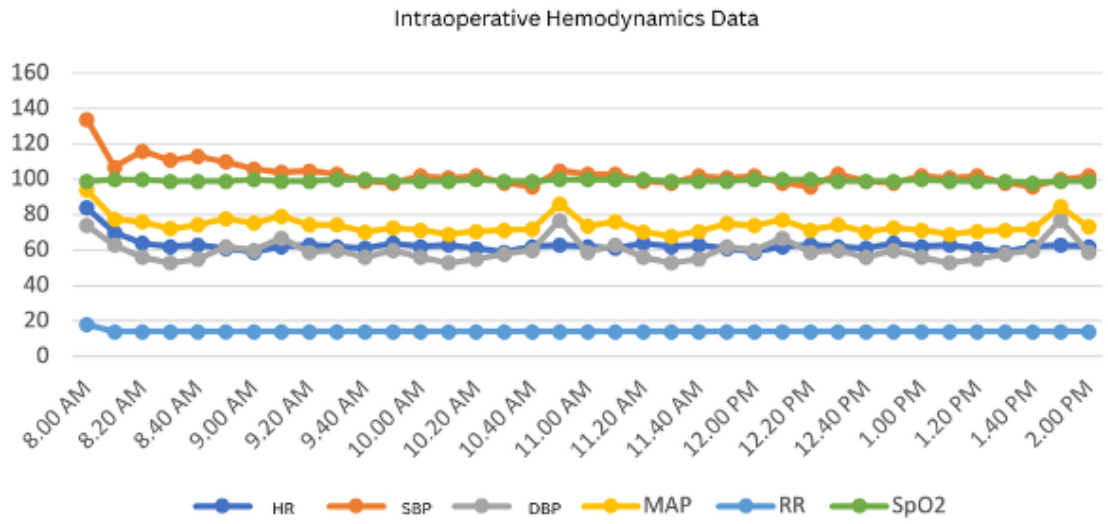

Figure 3- Intraoperative hemodynamic monitoring

Discussion

MMD is a rare, progressive, and chronic cerebrovascular disorder characterized by stenosis or occlusion of the terminal portions of the internal carotid artery and its major branches. The term MMS refers to secondary moyamoya-like vasculopathy associated with underlying conditions such as autoimmune diseases (e.g., systemic lupus erythematosus), hematologic disorders (e.g., sickle cell disease), prior cerebrovascular injury, or other pathological states [2]. In the present case, the history of hemorrhagic stroke followed by decompressive craniectomy is consistent with a secondary form of moyamoya vasculopathy, fulfilling the definition of MMS. Both MMD and MMS are characterized by the formation of fragile collateral vessels as a compensatory response to reduced CBF. However, these vessels are structurally weak and prone to rupture.

Cerebral angiography remains the gold standard for the diagnosis of moyamoya vasculopathy, although MRA is widely used as a noninvasive diagnostic adjunct [3]. On digital subtraction angiography (DSA), the abnormal collateral network produces the classic "puff of smoke" appearance [4]. In this patient, despite the absence of clinical signs of raised ICP, MRA demonstrated non-visualization of the right M2 segment of the middle cerebral artery, indicating reduced or absent distal flow consistent with MMS. Associated right frontal gliosis was identified, most likely representing a sequela of a prior hemorrhagic stroke and a decompressive craniectomy. Although MMS may cause chronic ischemia and gliosis in hypoperfused brain regions, the gliotic changes in this case were localized to the right frontal lobe at the site of the previous bone flap, supporting a post-surgical or post-stroke etiology. Importantly, MRA excluded other causes of cerebral flow disturbance including aneurysm, arteriovenous fistula, and arteriovenous malformation, in favor of a non-atherosclerotic and idiopathic vasculopathy consistent with MMS. Cerebral perfusion imaging with SPECT showed focal hypoperfusion of the right frontal region with preserved perfusion of the contralateral hemisphere, confirming the angiographic findings and supporting the indication for revascularization with STA-MCA bypass to restore cerebral perfusion.

The neuroanesthetic care of MMS patients with a history of stroke and decompressive craniectomy has distinct anatomic and physiologic problems. In MMS, progressive arterial stenosis and aberrant collateralization are related to small, brittle and weakly elastic arteries, contributing to cerebral hypoperfusion and increased susceptibility to blood pressure variations. Decompressive craniectomy has been shown to alter CSF circulation dynamics, including reduced intracranial pulsatility and altered CSF circulation. These alterations can result in diminished local perfusion and buildup of CSF collections such hygroma or hydrocephalus [5]. Post-operative scarring and gliosis add further to impairment of CSF circulation and interstitial fluid drainage. Previous studies have demonstrated that intracranial pulsatility can approach zero after craniectomy, reflecting a significant disturbance of CSF flow and local cerebral perfusion [6]. In patients with MMS, the combination of unstable collateral circulation, impaired cerebral autoregulation, and altered CSF dynamics increases vulnerability to perfusion imbalance, impaired metabolic waste clearance, focal edema, and progression of chronic hypoperfusion. Consequently, these patients are particularly sensitive to perioperative fluctuations in blood pressure, arterial carbon dioxide tension, and intravascular volume, all of which are critical considerations during STA–MCA bypass surgery.

The goal of anesthetic care of patients with MMS with a history of decompressive craniotomy is to preserve the optimum balance between the cerebral oxygen supply and demand. The brain is very sensitive to alterations in cerebral perfusion secondary to frail collateral arteries and abnormal CSF dynamics. This is accomplished by avoiding hypotension, preserving normocapnia, and giving enough depth of anesthesia and analgesia to reduce the rise in CMRO2 during unpleasant stimuli such as pin fixation, laryngoscopy, intubation, and surgical manipulation [7].

Preoperative optimization of brain perfusion and oxygenation is necessary prevent abrupt hemodynamic alterations. Careful selection of anesthetic agents and techniques is important to minimize the risk of ischemic or hypoxic brain injury. Blood products were prepared in case of possible intra-operative bleeding, especially during vascular manipulation for the STA–MCA anastomosis. Continuous arterial blood pressure monitoring was used because changes in MAP directly affect CPP. MAP was kept at an appropriate level (50-150 mmHg) to ensure adequate cerebral perfusion and to prevent disruption of autoregulation or damage to the blood-brain barrier (BBB) [8]. Impaired venous drainage, Trendelenburg position, increased intra-abdominal pressure, high positive end-expiratory pressure (PEEP >15 cmH2O) or jugular venous cannulation may impair CPP [9]. Ventilation was titrated using $EtCO_2$ monitoring to maintain normocapnia ($PaCO_2$ 35–40 mmHg), which is critical in MMS. In this patient, impaired autoregulation limits the brain's ability to compensate for CO_2 fluctuations; hypocapnia may induce vasoconstriction of collateral vessels and worsen ischemia, whereas hypercapnia may cause excessive vasodilation in normal territories, resulting in a steal phenomenon that further compromises perfusion of ischemic regions [10-11]. Fentanyl was selected for induction due to its ready availability, good hemodynamic profile, and possible neuroprotective

effects by increasing regional cerebral blood flow in the prefrontal cortex and caudate nucleus [12]. Although this effect may be beneficial in patients with frontal hypoperfusion, fentanyl was used only during the induction phase to prevent excessive increases in cerebral blood flow during maintenance. Propofol delivered by TCI (Schnider model) enabled accurate titration of depth of anaesthesia, proportional decreases in CBF and CMRO2 and effective control of ICP, thus conferring neuroprotective effects whilst minimising the risk of hypoperfusion in vulnerable areas [13]. Intravenous lidocaine was used to blunt airway reflexes and sympathetic responses during intubation, thus contributing to hemodynamic stability and attenuation of transient increases in MAP, HR and ICP [14]. Furthermore, a scalp block with bupivacaine was performed to reduce nociceptive input due to the scalp incision and Mayfield pin fixation [15].

The choice of propofol based TIVA with dexmedetomidine was based on the choice of hemodynamic stability and cerebral physiology. Dexmedetomidine is a highly selective α2-adrenergic agonist that provides sedation and analgesia with minimal respiratory depression and stable sympathetic suppression [16]. In the setting of impaired autoregulation, however, volatile anesthetic agents such as sevoflurane may increase cerebral blood flow and intracranial pressure in a dose-dependent manner, especially at concentrations >1 MAC, with the potential for increased cerebral edema [17]. High doses of opioids were also avoided due to risk of vasoconstriction and reduced blood flow to ischemic areas [18]. The combined use of propofol and dexmedetomidine in TIVA has synergistic effects, in that propofol causes proportional decreases in CBF and CMRO2 and that dexmedetomidine preserves blood pressure stability, so that intraoperative hemodynamic fluctuations are minimized during STA–MCA bypass [13]. STA–MCA bypass is a low-flow extracranial–intracranial revascularization procedure, which usually provides blood flow in the range of 15–50 mL/min. This is sufficient to improve cerebral perfusion in ischemic territories [1]. However, patients with MMS have a reduced cerebral oxygen reserve due to a pre-existing maximal vasodilation at the level of the microcirculation, resulting in an increased oxygen extraction ratio (OER) and increased susceptibility to ischemia. Hence, intraoperative blood pressure was controlled within 10–20% of baseline values to maintain cerebral perfusion [7]. In this example, steady systolic and diastolic blood pressure and HR throughout the surgery suggested effective hemodynamic management. This finding is compatible with sympatholytic effects of propofol–dexmedetomidine TIVA, which offers hemodynamic stability without substantial hypotension and bradycardia. Cheng et al. (2025) found considerably larger intraoperative blood pressure variability with sevoflurane than propofol (ARV systolic 6.4 vs 5.2; diastolic 3.9 vs 3.2; MAP 4.5 vs 3.8; all $p < 0.002$) indicating improved hemodynamic stability with intravenous propofol during MMD revascularization. [19]. Chinnarasan et al. (2024) reported steady hemodynamics with no bouts of hypotension or bradycardia during TIVA for craniotomy. The dexmedetomidine group demonstrated putative neuroprotective effects and needed less propofol and opioids [20]. Oh et al. (2024) reported that propofol-based TIVA was not associated with increased 90-day mortality compared with inhalational anesthesia but was associated with fewer postoperative complications (47.1% vs. 50.3%; OR 0.88; 95% CI 0.86–0.90; $p < 0.001$) in a nationwide cohort of >140,000 cranial surgery patients [21]. Taken together these data suggest that propofol–dexmedetomidine TIVA may be used in patients with MMS, providing better hemodynamic stability, better control of cerebral blood flow and possible neuroprotective benefits. This physiologically adapted approach seems to be especially beneficial in situations with impaired cerebral autoregulation.

During anesthesia, anesthetic depth was monitored using CONOX electrodes and qCON values were kept between 45 and 57. A qCON ≥80 suggests an awake or lightly sedated state, whereas values between 60 and 40 suggest an adequate level of general anesthesia [22]. Inhalational anaesthesia permits objective assessment of anaesthetic depth by measuring end-tidal concentrations of the agent, but TIVA does not allow direct measurement of depth. Thus, processed EEG monitoring such as CONOX is an essential part of correct assessment of the depth of anesthesia during neurosurgical procedures. This allows accurate titration of propofol and dexmedetomidine with preservation of haemodynamic stability and avoidance of reductions in CPP.

The patient received 1,500 mL crystalloid and a total of 250 mL of medications throughout the surgery. Urine output was adequate at 1,300 mL over 6 hours and estimated blood loss was approximately 400 mL. Fluid balance was relatively neutral, suggesting good maintenance of normovolemia, which is critical to CPP preservation in MMS patients. End-tidal CO2 levels were between 29 and 34 mmHg. This is explained by the physiological gradient between EtCO2 and PaCO2. Arterial PaCO2 was calculated to be ~34-39 mmHg, equal to mild hypocapnia. Roba et al. (2024) observed that mild hypocapnia (32–35 mmHg) did not adversely affect clinical outcomes in individuals with acute brain damage, whereas severe hypocapnia (26–31 mmHg) and hypercapnia (>45 mmHg) are related with higher mortality [23]. In the setting of MMS, the PaCO2 should ideally be maintained between 35 and 40 mmHg as hypocapnia can cause cerebral vasoconstriction and worsen ischemia and hypercapnia can increase ICP and

cause a steal phenomenon. The patient was well oxygenated as evidenced by consistent oxygen saturation of 99-100% during the surgery under regulated breathing. Invasive pressure measurements after STA-MCA anastomosis demonstrated mean pressures of 72 mmHg in the STA and 54 mmHg in the MCA, creating an effective pressure gradient of around 18 mmHg to promote donor-to-recipient flow. Doppler ultrasound showed adequate flow and patency of graft. Intraoperative hemodynamic stability was maintained to provide an optimal pressure gradient, allowing adequate bypass patency with no evidence of hyperperfusion. The brain was relaxed with preserved pulsatility suggesting low intracranial pressure and adequate cerebral perfusion. The effectiveness of the propofol–dexmedetomidine TIVA strategy was supported by stable perioperative hemodynamics. Postoperative strict control of ventilation, PaCO2 and blood pressure was achieved through the maintenance of the endotracheal tube, thus reducing the risk of hyperperfusion syndrome or secondary ischemia.

The patient was monitored in ICU for 24 hours postoperatively for the detection of any possible complications of hemodynamic instability like cerebral hyperperfusion syndrome, infarction, vasogenic edema or intracranial hemorrhage [24-25]. Stable laboratory parameters, rapid neurologic recovery and uncomplicated extubation demonstrated adequate cerebral oxygen delivery and successful perioperative management.

Conclusion

TIVA with propofol-dexmedetomidine is associated with stable hemodynamics and possible neuroprotection. In STA-MCA bypass, stable blood pressure and controlled ventilation maintain an optimal STA-MCA pressure gradient for graft patency and prevent hyperperfusion or ischemia. Postoperative blood pressure, PaCO2, and neurological status should be closely monitored to avoid hyperperfusion syndrome.

Ethics approval and consent to participate

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

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