

Anesthesia Management for Patients with Unruptured Giant Thrombosed Saccular Aneurysms

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ABSTRACT

A giant aneurysm in the middle cerebral artery (MCA) is a rare condition but has a high risk of rupture, requiring optimal anesthesia planning. This report presents a case of a 33-year-old man with an unruptured giant thrombosed saccular aneurysm in a symptomatic MCA branch since 2018. The patient had undergone several DSA examinations before being planned for craniotomy, aneurysm trapping, and high-flow EC-IC bypass. This report aims to describe the anesthetic management of giant MCA aneurysms undergoing complex surgical procedures to maintain hemodynamic stability and prevent perioperative complications. Physical and neurological examinations were within normal limits, and hemodynamic and laboratory results were stable. Anesthesia management included invasive monitoring, induction using tiopental, remifentanyl TCI, and rocuronium, and maintenance of anesthesia with tiopental titration, remifentanyl, and low-dose sevoflurane. The surgery lasted 11 hours and 45 minutes, with stable hemodynamic conditions and no intraoperative complications. Postoperatively, the patient was admitted to the ICU with intravenous analgesics. No signs of re-bleeding, hemodynamic instability, or new neurological deficits were observed. The report confirms that anesthesia management in cases of giant MCA aneurysms requires strict control of blood pressure, intracranial pressure, and sympathetic responses during critical phases of induction and extubation. A comprehensive and collaborative approach is essential to prevent complications and support the success of surgery for complex intracranial aneurysms.

Keywords:

giant MCA aneurysm; anesthesia; intracranial aneurysm; craniotomy; EC-IC bypass.

INTRODUCTION

Unruptured aneurysms originating from the middle cerebral artery (MCA) account for about 29%–41.4% of all unruptured aneurysms, while ruptured MCA aneurysms account for about 14.1%–19% of all ruptured aneurysms. Large (10–25 mm diameter) and giant (≥ 25 mm diameter) MCA aneurysms are not uncommon, accounting for 9.8% of all MCA aneurysms (Atallah et al., 2025; Beneš et al., 2024; Gadzhiagaev et al., 2022; Inagawa, 2022). Patients with large or giant MCA aneurysms may exhibit several clinical symptoms, including intracranial hemorrhage, headaches, focal neurological deficits caused by mass effects, and cerebral seizures or ischemia (Al-Shalchy et al., 2026; Barbarawi et al., 2025; Battal & Castillo, 2024; Rothrock, 2024; Stacy, 2022).

Previous studies have shown high rupture rates in large and giant MCA aneurysms (Duan et al., 2023; Gadzhiagaev et al., 2022; Gupta et al., 2023; Mosteiro et al., 2024; Nagy et al., 2025; Zhou et al., 2023). The UCAS Japan (Unruptured Cerebral Aneurysm Study of Japan) reported that the annual rupture rate of large and giant MCA aneurysms was 4.11% and 16.87%, respectively. Additionally, the ISUIA (International Study of Unruptured Intracranial

Aneurysms) determined that the 5-year cumulative rupture rate for patients without a history of subarachnoid hemorrhage (SAH) who had an aneurysm located in the internal carotid artery, anterior communicating artery, anterior cerebral artery, or MCA was 14.5% for aneurysms with a diameter of 13–24 mm and 40% for aneurysms with a diameter of ≥ 25 mm. Therefore, treatment must be considered to prevent rupture or re-bleeding and relieve symptoms due to the mass effect (O'Brien et al., 2022; Seiffge et al., 2024; Zhang et al., 2026).

This gap in the literature is particularly significant given the unique considerations posed by giant thrombosed aneurysms. These lesions often require more extensive surgical dissection, prolonged operative times, and complex revascularization procedures, all of which demand meticulous anesthetic planning and execution. The presence of intraluminal thrombus adds further complexity, as manipulation may carry risks of thromboembolism. Despite the availability of general neuroanesthesia guidelines, there is a scarcity of detailed case reports documenting the real-world application of these principles in the context of giant thrombosed MCA aneurysms, particularly those managed with combined trapping and bypass procedures.

The urgency of addressing this gap is underscored by several factors. First, the high rupture rates of giant aneurysms create pressure for timely surgical intervention, yet the complexity of these procedures increases perioperative risks. Second, the prolonged duration of such surgeries (often exceeding 10 hours) presents unique challenges for maintaining hemodynamic stability, fluid balance, and anesthetic depth. Third, the need for intraoperative neurophysiological monitoring and the potential for temporary arterial occlusion require sophisticated anesthetic techniques that preserve cerebral function while providing optimal surgical conditions. Fourth, the postoperative period carries risks of re-bleeding, cerebral edema, and new neurological deficits that may be influenced by intraoperative anesthetic management.

This case report addresses the identified gap by providing a detailed account of the anesthetic management of a 33-year-old male with an unruptured giant thrombosed saccular aneurysm at the MCA bifurcation, who underwent craniotomy for aneurysm trapping and high-flow EC-IC bypass. The novelty of this report lies in its comprehensive documentation of the entire perioperative anesthetic journey, from preoperative optimization through intraoperative management of a prolonged 11-hour 45-minute procedure to postoperative intensive care and follow-up. By detailing the specific anesthetic agents used, hemodynamic targets achieved, monitoring techniques employed, and management of critical phases including induction, temporary clipping, and extubation, this report provides practical insights that complement existing guidelines and general principles.

The primary purpose of this case report is to contribute to the body of knowledge on anesthetic management for complex intracranial aneurysm surgery by sharing a successful clinical experience. The specific objectives are: (1) to describe the comprehensive preoperative assessment and optimization of a patient with a giant thrombosed MCA aneurysm; (2) to detail the intraoperative anesthetic management, including induction, maintenance, monitoring, and hemodynamic control strategies; (3) to document the postoperative care and short-term outcomes; and (4) to discuss the key anesthetic principles and lessons learned from this case in the context of existing literature. The benefit of this report is to provide practicing anesthesiologists with a practical example of how theoretical principles can be applied in a

challenging clinical scenario, potentially improving outcomes for future patients undergoing similar complex neurosurgical procedures.

METHOD

Anamnesis

A 33-year-old male patient, with a diagnosis of Unruptured Giant Thrombosed Saccular Aneurysm MCA Bifurcation D, Post DSA Diagnostic (8/01/20), Post DSA Diagnostic (13/12/24), Post DSA Diagnostic (18/08/25) came for a Craniotomy Trapping Aneurysm action plan. The patient previously had a history of right-sided headaches (since 2018), headaches said to have disappeared. The patient had a history of hospitalization at Bhakti Rahayu Hospital and a CT scan was said to have a lump in the blood vessel. The patient was then referred to Siloam Hospital and DSA was carried out (January 2020), advised to take action but the patient refused because it was not covered by BPJS. The patient after that never again controls to a neurologist. In the past year, the patient felt a headache reappear. The headache is said to be throbbing on the right side of the head, disappeared and aggravated 1 month ago. Headaches are said to be approximately 1-2 times a week, it is said that they cannot do activities at all when they have headaches. Headaches are said to improve with rest and taking paracetamol. Other complaints such as weakness in half of the body, squinting lips, slurred speech are denied. Treatment history: Ibuprofen 400mg every 12 hours IO, Lisinopril 5 mg every 24 hours IO, Klobazam 10 mg every 24 hours IO, Diazepam 2 mg every 24 hours IO. History of hypertension (+) since 2018 since headache with Lisinopril 5 mg treatment every 24 hours IO. History of DM, stroke, heart disease is denied. A history of drug and food allergies is currently denied. The history of smoking is approximately since 10 years (1 pack per day) last smoked 1 day ago, drank alcohol (+), Currently the patient is no longer working, and is only able to do moderate light physical activity without complaints of tightness and chest pain.



Figure 1. Patient Conditions Before Surgery

Physical Examination

On physical examination, it was found that patients were in general state of moderate pain with awareness of GCS E4V5M6 compos mentis, blood pressure 120/180 mmHg, pulse 79 times/minute, regular, sufficient content, respiratory frequency 16-18 times/minute vesicular without rhonki and wheezing, body temperature 36.7 °C, oxygen saturation (SpO2) 98% room water, numeric rating scale (NRS) 1/10 both in a stationary and moving state. Patients weighing 78 kg and BMI 27.3 kg/m2. Neurological examination, Glasgow Coma Scale (GCS) E4V5M6. The pupil is rounded isocor, right and left diameter 3 mm, the right and left pupils are reactive to direct light and indirect light. Examination of meningeal stimulation did not find any stiffness of the knee. There are no neurological deficits. Motor power 55555/55555 // 55555/55555.

Supporting Examinations

The laboratory results can be seen in Table 1. Thoracic imaging showed a 49% cor and no abnormalities (Figure 2). DSA (18/08/2025) Visible saccular anourysma in MCA bifurcation with domo to neck ratio 7.9, Aspocrt ratio 8.5.

The patient was diagnosed with Unruptured Giant Thrombosed Saccular Aneurysm, MCA Bifurcation, D, Post DSA Diagnostics (8/01/20), Post DSA Diagnostics (13/12/24), Post DSA Diagnostics H1 (18/08/25). The patient is planned to undergo Craniotomy Trapping Aneurysm + High Flow EC-IC Bypass.

Table 1. Laboratory results

Complete Blood (22/08/2025)	WBC 11,97 x 103/μL (4.1 - 11.0); HGB 15,3 g/dL (13.5 - 17.5); HCT 46,6 % (41.0 - 53.0); PLT 268 x 103/μL (150 - 440); MCV 81,8 fL (80.0 - 100.0); MCH 26,8 pg (26.0 - 34.0); MCHC 32,8 g/dL (31 - 36).
Fail Hemostasis (22/08/2025)	PPT 9.7 seconds (10 - 12.7); INR 0.84 (0.9 - 1.1).
Clinical Chemistry (22/08/2025)	SGOT 36 U/L (5.00 - 34.00); SGPT 88 U/L (< 55); BUN 12.3 mg/dL (8.9 - 20.6); Creatinine 0.97 mg/dL (0.72 - 1.25); e-LFG 102.14 ml/min/1.73m ² (>= 90); K 4 mmol/L (3.50 - 5.10); 140 mmol/L (136 - 145); Cl 105.1 mmol/L (94 - 110); GDS 65 mg/dL (70 - 140)
EKG (12/09/2025)	Sinus aritmia, 75 bpm, Normoaxis, No ST-T Change
Photo: Thorax AP (22/08/2025)	Cast 49% and pulmo no abnormalities

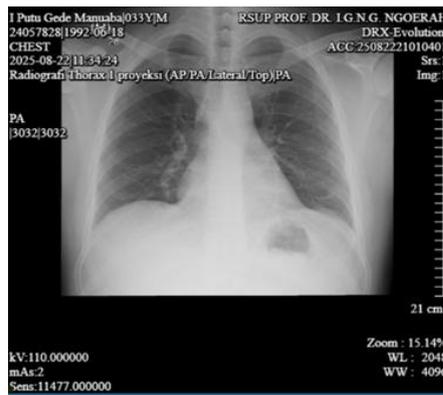


Figure 2. Photo Thorax

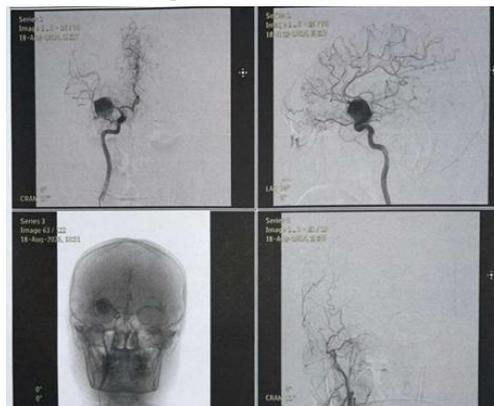


Figure 3. DSA

Anesthesia Management

While in the reception room, the patient has installed intravenous access to the manus dextra. While in the operating room, electrocardiogram (ECG) monitoring of 5 leads, SpO₂, was carried out, then continued with the installation of arterial lines and CVCs. Patients were given continuous drip premedication of dexmedetomidine at a dose of 0.4 mcg/kgBB/hour. Patients were induced with Tiopental 200 mg (3 mg/kgBB) IV and Remifentanyl TCI Mode Minto analgetic with a Target Effect of 4 – 6 ng/m, as well as with 40 mg IV rocuronium muscle paralysis. Post-intubation hemodynamics were obtained with blood pressure of 110/68 mmHg, pulse rate of 68 times/minute. After intubation, the patient is positioned supine and head up 30 degrees. During maintenance operations, anesthesia was performed by administration of Tiopental titration of 1-3 mg/kgBB/hour, continuous remifentanyl drip with a Target Effect of 2 – 4 ng/mL, sevoflurane 0.3 – 0.6 Minimum Alveolar Concentration (MAC) using a Persus ventilator, with minimal flow. During the operation, a ventilator is used with volume control mode, with a fresh gas flow of 0.8 liters per minute, with an oxygen fraction of 80%, with sevoflurane of 0.3 – 0.4 MAC. After intubation, etCO₂ monitoring is also carried out. Hemodynamics during surgery were relatively stable, with blood pressure in the range of 92 – 122 / 58 – 75 mmHg, so pulse was 57 – 68 times/minute, SpO₂ 98 – 99%, PaCO₂ 31 – 35 mmHg. The operation lasted for 11 hours and 45 minutes, with 400 ml of bleeding, urine output of 1700 ml, with 3000 ml of crystalloid incoming fluid. 25 mcg fentanyl is given 5 minutes before extubation and dsmedetomidine infusion is maintained at 0.3 mcg/kg/min to prevent coughing. Once the anesthesia is stopped, we extubage it when it is easy to wake up in the operating room.

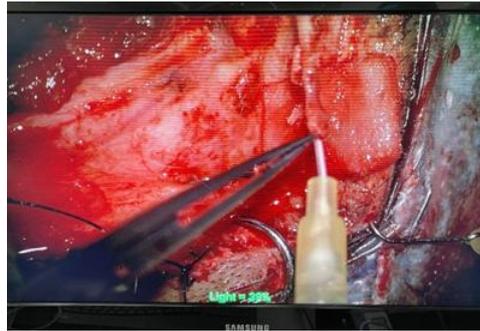


Figure 3.

Post-Surgical Care

Postoperatively, patients are treated in intensive therapy rooms, with DPO awareness, with stable hemodynamics. Patients were given 400 mcg of Fentanyl in 50 mL of 0.9% NaCl at a rate of 2.1 mL/hour and Paracetamol 1000 mg every 8 hours intravenously to control pain.

RESULT AND DISCUSSION

Background: Preoperative evaluation should focus on the pathophysiological consequences of intracranial aneurysms that need to be anticipated. Neurological complications include increased intracranial pressure, decreased brain blood flow, brain autoregulatory disorders, and cerebral ischemia. Ruptured aneurysms, inflammation, and direct irritation of the brainstem stimulate the sympathetic system, which can lead to neurogenic pulmonary edema. Sympathetic hyperactivity also causes myocardial dysfunction. Cardiac arrhythmias and electrocardiographic changes such as sinus bradycardia, sinus tachycardia, ST-segment depression, T-wave inversion, U-waves, and elongated QT intervals are often observed. Intracranial aneurysms are also associated with fluid and electrolyte imbalances such as hypokalemia, hyperglycemia (which may require insulin administration), and hyponatremia caused by cerebral salt wasting due to increased secretion of brain natriuretic peptides, followed by suppression of aldosterone synthesis. Immediate correction of this fluid, glucose, and electrolyte imbalance needs to be performed by an anesthesiologist before surgery. Serial monitoring of blood and urine electrolytes and osmolality is also required when mannitol or hypertonic saline is administered. Patients with poorer neurological status are associated with increased intracranial pressure, intraoperative brain edema, cerebral autoregulatory disorders, and impaired cerebrovascular reactivity to carbon dioxide.

Anesthesia Induction: The main purpose of anesthesia induction is to prevent hypertension caused by the laryngoscope and tracheal intubation. Airway manipulation stimulates the sympathetic system, resulting in hypertension. Hypertension can cause re-bleeding and increase the transmural pressure of the aneurysm, leading to sudden rupture. Therefore, blood pressure should be monitored after anesthesia induction, before laryngoscopy, and immediately after tracheal intubation. In this case, the patient's blood pressure after anesthesia induction was 120/63 mmHg, before laryngoscopy was 101/54 mmHg, and after tracheal intubation was 120/58 mmHg. The anesthesiologist should also be vigilant for signs of possible rebleeding, such as persistent hypertension, bradycardia, and sudden anisocoria. The installation of an arterial line can also cause pain and anxiety, leading to hypertension. Insertion of a pre-induced arterial line to monitor blood pressure is not necessary unless the

patient has cardiac dysfunction, including increased troponin levels and hemodynamic instability. In this case, the insertion of the arterial line was carried out before anesthesia induction. The Allen test was performed, followed by the installation of an arterial line and the administration of 2% Lidocaine as a local anesthetic. The patient's blood pressure was within normal limits (121/68 mmHg) after the insertion of the arterial line. Bag-mask ventilation can cause hypocarbia or hypercarbia. Hypercarbia causes vasodilation, which results in increased intracranial pressure and interferes with cerebral perfusion, while hypocarbia caused by hyperventilation leads to a decrease in intracranial pressure, which increases transmural pressure of the aneurysm.

Hemodynamic Goals: The hemodynamic goal is a 20% decrease in blood pressure from baseline or a systolic blood pressure of less than 160 mmHg. However, aggressive treatment in lowering blood pressure can lead to secondary ischemia. In this case, the patient's systolic blood pressure was maintained at 120-130 mmHg. Measures to prevent a hemodynamic response are required, such as increasing the depth of anesthesia, administering analgesic agents like bolus fentanyl or remifentanyl, and using short-acting antihypertensive agents such as esmolol and nicardipine. Temporary clip placement before permanent clip placement can decrease brain oxygenation. Therefore, an increase in blood pressure of 10-20% of pre-induction baseline blood pressure for a short period of time is required to increase blood flow to areas at risk of ischemia. Blood pressure may return to normal after the procedure is completed.

Postoperative Considerations: Herniation and rapid increases in intracranial pressure can lead to pupil dilation, arrhythmias, and ischemia. The surgeon may also notice an increase in blood flow at the site of the incision or sudden protrusion of the brain. Lowering intracranial pressure and neuroprotection are the main goals before opening the dura mater. The use of bolus propofol, short-acting opioid continuous infusion, or short-acting opioid bolus, or sodium thiopental, is beneficial for maintaining intracranial pressure, cerebral perfusion pressure, and average arterial pressure. Intravenous anesthesia is also beneficial in lowering the rate of cerebral metabolism. In this case, anesthesia was maintained by administering TCI Tiopental titration of 1-3 mg/kg/hour, Remifentanyl TCI Minto Mode with a target effect of 4-6 ng/mL, and Rocuronium 0.15 mg/kg every 45-60 minutes IV. Other strategies, such as moderate hyperventilation and short-term hypothermia, can also lower intracranial pressure. Some studies have found that mild hypothermia (32-35°C) shows benefits in lowering intracranial pressure in patients with good prognosis.

Hypertension and Extubation: Hypertension can also occur during extubation due to sympathetic nerve stimulation. In this case, the patient's blood pressure after extubation was 125/68 mmHg. Clinical manifestations of intraoperative aneurysm rupture can be seen postoperatively, such as delayed return to consciousness, rapid onset of neurological damage, hemodynamic instability, focal neurological deficits, or seizures. In these cases, after surgery, the patient was admitted to the intensive care unit and given mechanical ventilation. Neurological examination showed that the patient remained with aphasia without any other neurological deficits. Symptoms of recurrent headaches were absent, and there were no episodes of hemodynamic instability. The patient was discharged from the hospital on the 5th day postoperatively. Follow-up at 1 month postoperatively showed no complaints, with improvement in headache and ataxia.

CONCLUSION

Intracranial aneurysm rupture is life-threatening and requires immediate treatment, either through clipping craniotomy or endovascular coil insertion. The anesthesiologist should recognize the symptoms of residual bleeding during a thorough pre-anesthesia evaluation and ensure optimal perioperative management. This includes preventing re-bleeding, maintaining systolic blood pressure according to guidelines, neurophysiological monitoring, intracranial pressure control, providing neuroprotection, and preventing postoperative pain and complications.

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