

Anesthetic Management of Mechanical Thrombectomy for Acute Ischemic Stroke with Severe Mitral Stenosis: A Case Report

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Abstract

Acute ischemic stroke from large vessel occlusion requires prompt reperfusion to preserve brain function. Mechanical thrombectomy is the treatment of choice in selected cases, especially cardioembolic strokes. Anesthetic management plays a critical role, particularly in patients with cardiac comorbidities. We report a 62-year-old woman presenting with left-sided weakness and reduced consciousness, six hours prior to admission. She had a history of rheumatic mitral stenosis and atrial fibrillation. CT angiography showed infarction in the right MCA territory with distal ICA occlusion. Intravenous thrombolysis was initiated but failed to show improvement. Mechanical thrombectomy was then performed under general anesthesia. Due to the patient's decreased consciousness and aspiration risk, general anesthesia was preferred to secure the airway and allow precise hemodynamic control. The anesthetic strategy focused on maintaining systemic vascular resistance while avoiding increased pulmonary vascular resistance to prevent right heart overload in mitral stenosis, also oxygenation and ventilation were carefully managed to avoid hypoxia and hypercapnia. Full vessel recanalization (mTICI 3) was achieved without complication. This case highlights the importance of tailoring anesthesia to individual risk profiles, particularly in stroke patients with valvular heart disease, where general anesthesia may provide optimal safety and procedural success.

Keywords: General Anesthesia, Ischemic Stroke, Mitral Stenosis, Neuroanesthesia, Thrombectomy

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Introduction

An acute stroke is a sudden loss of neurological function due to a vascular issue in the brain, spinal cord, or retina. In 2021, strokes caused one in six cardiovascular deaths in the U.S. Over 795,000 Americans have a stroke each year—610,000 are first-time events, and 185,000 (23%) occur in people with prior strokes. Ischemic strokes, caused by blocked brain blood flow, make up 87% of cases.¹ The TOAST classification divides ischemic strokes into five subtypes: cardioembolic (from heart-origin clots), small

vessel occlusion, large artery atherosclerosis (with >50% narrowing and infarcts >1.5 cm), strokes of undetermined cause (due to unclear or multiple factors), and strokes from other known but rare causes like clotting disorders. Diagnosis depends on clinical signs and imaging evidence.² Acute ischemic stroke caused by large vessel occlusion (LVO) is a leading cause of disability and mortality worldwide.³ Rapid diagnosis and timely reperfusion therapy are critical to improving neurological outcomes. Mechanical thrombectomy has emerged as the standard treatment for patients presenting with anterior

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circulation LVO, particularly when intravenous thrombolysis is contraindicated or fails to achieve reperfusion.⁴

A key aspect of mechanical thrombectomy is the choice of anesthesia.⁵ While conscious sedation (CS) is often favored for its shorter preparation time and fewer hemodynamic fluctuations, general anesthesia (GA) may provide better airway protection, patient immobility, and procedural safety—particularly in patients with decreased consciousness or high aspiration risk. The decision between GA and CS remains debated, with studies reporting mixed outcomes influenced by patient selection and procedural factors. We present a case of a 62-year-old woman with cardioembolic stroke secondary to RHD and atrial fibrillation who underwent emergency mechanical thrombectomy under general anesthesia after unsuccessful intravenous thrombolysis. This report highlights the clinical reasoning behind anesthetic choice in complex stroke patients and discusses the potential impact of anesthesia modality on procedural success and patient outcomes.

Case

History

A 62-year-old female patient was referred with a chief complaint of decreased consciousness that began approximately six hours before admission. The symptoms were first noted after she awoke and was found lying unresponsive in the living room. Prior to this episode, the patient had been in her usual state of health and able to carry out daily activities without limitation. On the morning of symptom onset, she experienced a sudden onset of left-sided body weakness, headache, nausea, vomiting, and slurred speech. There was no history of trauma, seizures, or fever. The patient had a known history of hypertension diagnosed in 2023, for which she was on regular follow-up. She also had a documented history of cardiac arrhythmia and rheumatic heart disease (RHD) since 2023, but had not been under regular cardiology care for the past year. Her regular medications included Candesartan 4 mg once daily, Spironolactone 25 mg once daily,

Bisoprolol 2.5 mg once daily, and Clopidogrel 75 mg once daily. She denied any history of diabetes mellitus, stroke, or other systemic illnesses.

Physical Examination

On examination, the patient's axillary temperature was 36.8°C, and her body mass index (BMI) was calculated at 27.55 kg/m² based on a weight of 65 kg and height of 155 cm. Neurological examination revealed a Glasgow Coma Scale (GCS) score of E3V5M6. Pupils were equal, round, and reactive to light, measuring 3 mm bilaterally. There was supranuclear paresis of cranial nerves VII and XII on the left side, accompanied by clear signs of left-sided motor deficit, indicating neurological lateralization. No signs of meningeal irritation or neck stiffness were noted. Cardiovascular assessment showed a blood pressure of 185/99 mmHg and a heart rate of 77 beats per minute with strong peripheral pulses. Heart sounds were regular without murmurs or gallop. Respiratory examination revealed symmetrical chest expansion, a respiratory rate of 20 breaths per minute, and clear vesicular breath sounds without rhonchi or wheezing. Oxygen saturation was 98% on nasal cannula at 3 liters per minute. Abdominal and genitourinary examinations were unremarkable; bowel sounds were normal, and there was no abdominal tenderness or distension. Nevertheless, a nasogastric tube was still inserted. The patient had a Foley catheter in place with a urine output of 700 mL over six hours.

Supporting Examination

Laboratory evaluation revealed a hemoglobin level of 11.4 g/dL, indicating mild anemia, with blood sugar of 155 mg/dL, and a serum potassium level of 3.03 mmol/L, consistent with mild hypokalemia, with otherwise normal renal and liver function profiles. Coagulation tests were within normal limits. The chest radiograph demonstrated cardiomegaly with a cardiothoracic ratio of 69% (Figure 1A). Electrocardiography revealed atrial fibrillation with rapid ventricular response at a rate of 110 bpm, normal axis, and no ST-T changes (Figure 1B). Echocardiography showed left atrial dilatation without left ventricular hypertrophy, preserved systolic and diastolic function, and

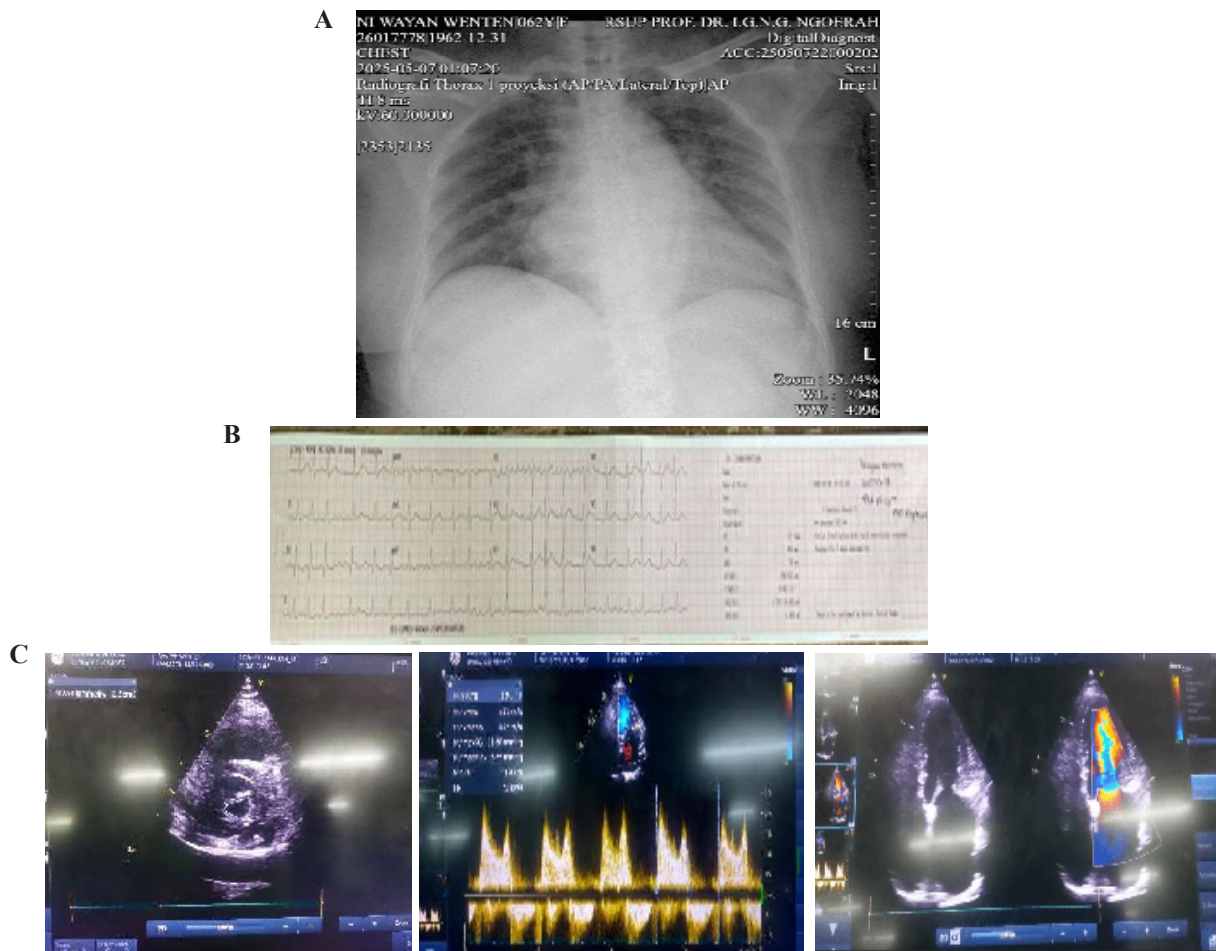


Figure 1. Cardiology assessment. (A) Chest X-ray showed cardiomegaly (CTR 69%). (B) ECG demonstrated atrial fibrillation with rapid ventricular response. (C) Echocardiography revealed left atrial dilatation, preserved biventricular function (EF 63%, TAPSE 2.8 cm), and normokinetic wall motion. Valve assessment showed posterior mitral leaflet calcification with moderate mitral stenosis, mild mitral regurgitation, mild pulmonary regurgitation, and an estimated right atrial pressure of 8 mmHg

an ejection fraction of 63%. Right ventricular function was normal, with a TAPSE of 2.8 cm and normokinetic wall motion. Valve assessment indicated posterior mitral leaflet calcification and thickening, consistent with moderate mitral stenosis and mild mitral regurgitation, along with mild pulmonary regurgitation. The estimated right atrial pressure was 8 mmHg (Figure 1C). Based on her cardiac assessment, the patient was then assessed with atrial fibrillation with rapid ventricular response (AF RVR). Based on the CHA₂DS₂-VASc score, the patient was recommended for anticoagulation therapy. However, the HAS-BLED score indicated a moderate risk of bleeding. There was a suspicion of congestive heart failure (CHF) secondary

to suspected valvular rheumatic heart disease (RHD) based on echocardiographic findings suggesting moderate mitral stenosis (MS) and mild mitral regurgitation (MR). The patient was subsequently treated with Ramipril 5 mg once every 24 hours, Bisoprolol 1.25 mg once daily, Spironolactone 25 mg once daily, Furosemide 40 mg IV as needed, and Digoxin 0.5 mg IV if the heart rate in atrial fibrillation reached or exceeded 110 bpm.

A CT scan of the head with angiography was performed in axial slices with coronal and sagittal reformats, both pre- and post-contrast, including CT angiography (CTA) and CT venography (CTV) sequences (Figure 2). The imaging revealed a

characteristic of an acute ischemic infarct in the territory of the right MCA. Additionally, multiple well-defined hypodense lesions with densities approaching that of cerebrospinal fluid (CSF) were noted in the right external capsule and right lentiform nucleus, consistent with chronic infarction in these areas. The arterial phase of the CT angiography demonstrated that both internal carotid arteries (ICA) had normal calibers without evidence of plaques, stenosis, or aneurysmal changes. Notably, the right MCA demonstrated segmental luminal narrowing at the M4 segment, although contrast opacification was still visualized distally, indicating partial stenosis. The left MCA, anterior cerebral arteries (ACA), posterior cerebral arteries (PCA), anterior and posterior communicating arteries (AcomA and PcomA), basilar artery, and both vertebral arteries were all of normal caliber and free of any vascular pathology. Venous phase imaging showed preserved flow and normal appearance of all major intracranial venous structures, including the transverse and sigmoid sinuses bilaterally, superior and inferior sagittal sinuses, internal jugular veins, cavernous sinuses, internal cerebral veins, vein of Galen, straight sinus, and torcular Herophili. In summary, the radiological demonstrated occlusion of the distal segment (D) of the right internal carotid artery (ICA), suggestive of a cardioembolic or thromboembolic ischemic stroke, likely secondary to the patient's underlying rheumatic heart disease and cardiac arrhythmia, consistent with an acute infarction in the right frontotemporal cortical-subcortical

region, corresponding to the right MCA territory (ASPECT score 7), with chronic infarcts observed in the right external capsule and lentiform nucleus. There is partial stenosis of the right MCA at the M4 segment, which may be contributing to the acute ischemic event.

Given the acute presentation of left-sided neurological deficits, imaging-confirmed large vessel occlusion, and the fact that the patient remained within the therapeutic window for intervention, with onset of less than 4,5 hours, the patient was classified with NIHSS Scoring 15 (moderate), and underwent thrombolytic using alteplase administered intravenously at a dose of 0.6 mg/kg body weight, divided into two phases: the first 10% of the total dose was given as an intravenous bolus over 1 minute, and the remaining 90% of the total dose was administered via continuous infusion over 60 minutes. After 1.5 hours of observation, the patient still showed no clinical improvement based on neurological evaluation. Since the onset was still within 6 hours and the condition was suspected to involve a major cerebral vessel, an emergency mechanical thrombectomy was indicated.

Anesthesia Management

The procedure was performed under general anesthesia to provide optimal airway protection, patient immobility, and procedural control, especially in a patient with impaired consciousness. The patient was then positioned in supine, and vital sign was measured with pre-

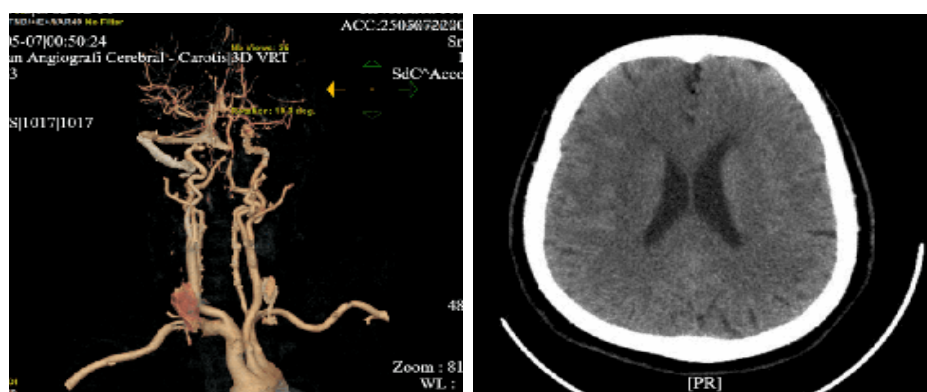


Figure 2. CT scan angiography revealed occlusion of the distal segment (D) of the right internal carotid artery (ICA), suggestive of a ischemic stroke

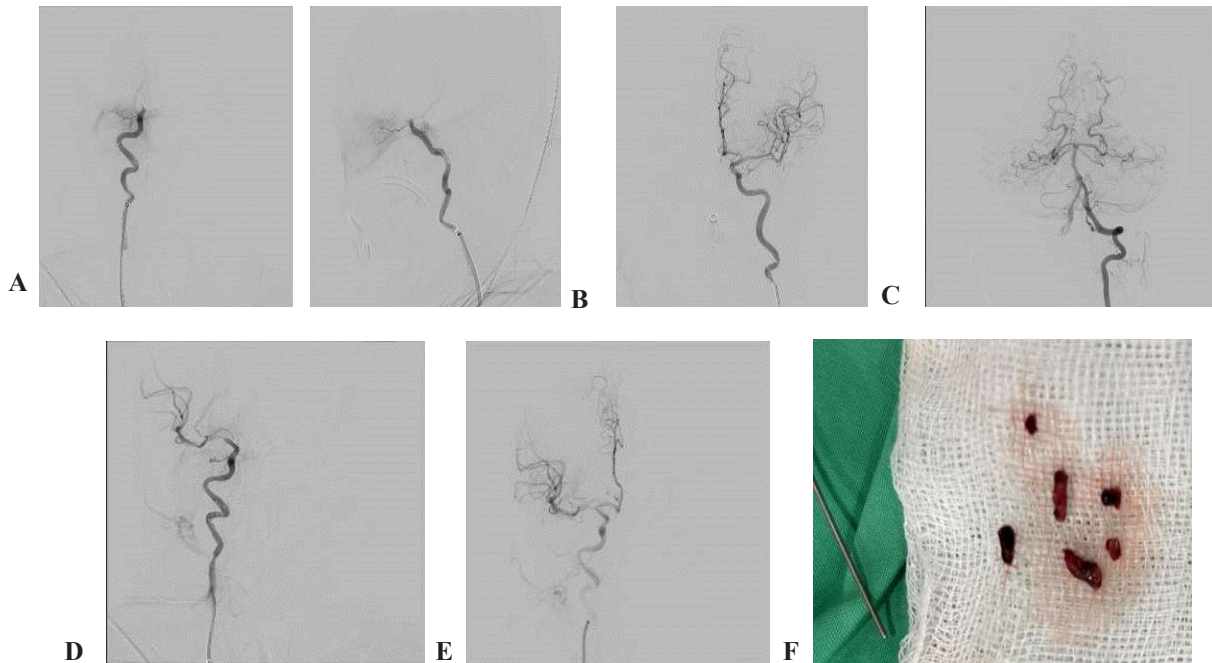


Figure 3. In the pre-thrombectomy DSA, the right internal carotid artery (ICA) is visualized up to the C6 segment, with an occlusion observed at C6 (A) and left middle cerebral artery and anterior cerebral artery are visualized up to their distal segments. Cortical venous drainage is directed toward the sinus system. No stenosis, aneurysm, or vascular malformation is observed. (B); The branches of the vertebral artery and basilar artery as well as the posterior circulation, are well visualized. The posterior communicating artery (Pcomm) is visible. The contralateral posterior circulation is also well visualized. (D) The internal carotid artery (ICA) is visualized up to the C6 segment, with an occlusion observed at C6. (E) After aspiration was done, there was migration of occlusion to ICA up to the C7 segment. (F) Multiple fragments of thrombus retrieved following mechanical thrombectomy.

induced blood pressure of 185/99 mmHg; heart rate 77 times per minute, and respiration rate times per minute; with peripheral oxygen saturation of 99%. Local infiltration with 2% lidocaine was performed prior to right radial artery line placement. Preoxygenation was carried out with 100% oxygen 6 liters per minute for 5 minutes, meanwhile, ensuring that all drugs for induction had reached their onset of action. The induction was performed by administering fentanyl at a dose of 4 mcg/kgBW and propofol at 2 mg/kgBW, along with the use of the muscle relaxant rocuronium at 1 mg/kgBW, continued with intubation with a 7.5 cuffed endotracheal tube. With the patient's blood pressure after intubation was 160/78 mmHg with the heart rate of 68 times per minute. Throughout the procedure, volume-controlled ventilation was carried out with low flow FiO_2 90%, FG Flow 0.25, P max 25, tidal volume 350 ml, RR 10 x/min, Peep 4 and T Ins 2.0, with the target of PCO_2 30 – 35 cmH₂O and

peripheral oxygen saturation of 96-99%. The patient was maintained during the procedure by giving combining anesthesia gas Sevoflurane 0.3–0.5 MAC and intermittent Fentanyl 0.25 mcg/kgBW and Rocuronium 0.15 mg/kgBW every 30-45 minutes.

Procedure Management

A digital subtraction angiography (DSA) procedure was performed using a femoral approach, with access established via an 8 French femoral sheath. The procedure utilized a 5 Fr diagnostic catheter (Davis), a Terumo 0.035" guidewire, an 8 Fr BMX guiding catheter, a 6 Fr Sofia aspiration catheter, a Vasco 25 microcatheter, and a 0.014" Traxcess microwire. Angiographic imaging of the left internal carotid artery (LICA) demonstrated good visualization of the middle cerebral artery (MCA) and anterior cerebral artery (ACA) up to the distal segments, with visible anterior and posterior communicating

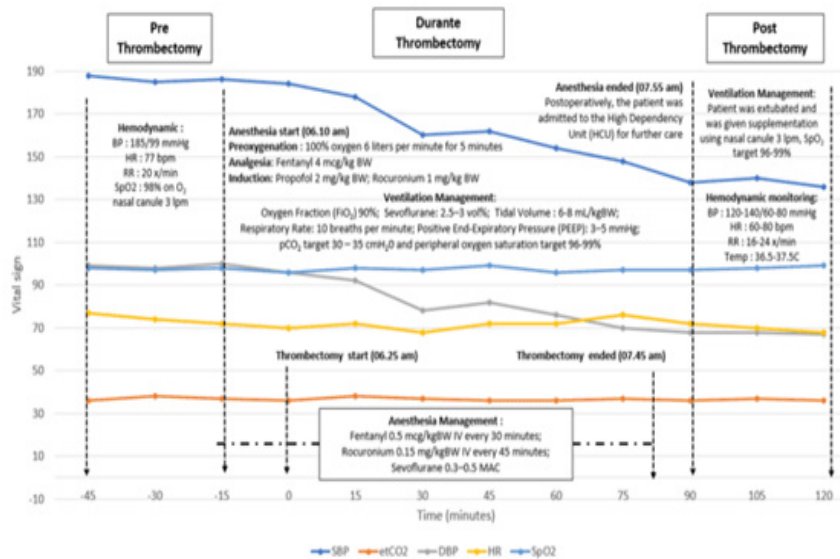


Figure 4. Perioperative Hemodynamic and Ventilation Strategy during the Procedure

arteries. No evidence of stenosis, aneurysm, or vascular malformations was noted, and the venous drainage from the cortical regions toward the sinus system appeared normal. Evaluation of the left vertebral and basilar arteries (LVA-BA) showed good visualization of their respective branches, with intact posterior circulation and well-opacified posterior communicating arteries. The contralateral posterior-lateral circulation was also well maintained. In contrast, imaging of the right internal carotid artery (RICA) revealed opacification up to the C6 segment, with an occlusion seen just distal to this level. Based on these findings, the decision was made to proceed with a mechanical thrombectomy.

The thrombectomy process began with a catheter exchange using an 8 Fr BMX catheter, followed by the insertion of a Sofia 6 Fr aspiration catheter up to the thrombus location (Figure 3). The aspiration catheter was then connected to the aspiration system, which was activated and allowed to run for three minutes. After this, the catheter was carefully withdrawn into the guiding catheter while maintaining negative pressure. This aspiration maneuver was repeated twice through the guiding catheter. Subsequent angiographic imaging showed that part of the thrombus had migrated distally to the C7 segment of the ICA. Contact aspiration was

used to remove this migrated clot. A follow-up DSA demonstrated further clot migration to the proximal segment of the ICA. A microcatheter and microwire were then advanced just proximal to the thrombus, followed by advancement of the aspiration catheter. Once the position was secured, the microcatheter and microwire were withdrawn, and aspiration was again performed using the contact aspiration technique. Final angiographic evaluation demonstrated successful recanalization, with complete opacification of the distal branches of the right MCA and ACA. In total, 60 cc of iopamidol contrast was used during the procedure. There were no complications, and bleeding was minimal and limited to the access site. Intraoperative monitoring demonstrated stable hemodynamics, oxygenation, and ventilation throughout the peri-thrombectomy period (Figure 4). The procedure was deemed successful, achieving a modified Thrombolysis in Cerebral Infarction (mTICI) score of 3 after three aspiration passes, indicating complete reperfusion. The patient was then monitored in high dependency unit by maintaining systolic blood pressure below 140 mmHg and performing a follow-up CT scan within 24 hours to monitor for any complications.

Discussion

Cerebral blood flow is mainly regulated by vessel resistance, which depends on vessel diameter, and by cerebral perfusion pressure.⁶ Autoregulation keeps blood flow stable despite changes in pressure, typically within a MAP range of 60–150 mm Hg. This involves smooth muscle responses and possibly vasoactive substances like nitric oxide, though mechanisms are not fully known. Outside this range, or during conditions like ischemic stroke, autoregulation fails.² The middle cerebral artery (MCA) is the most commonly affected vessel. It supplies the lateral brain, including areas responsible for motor and sensory function in the face and upper limbs. MCA strokes often cause contralateral facial and arm weakness, sensory loss, aphasia (if the dominant hemisphere is involved), visual field defects, neglect (with nondominant strokes), and dysarthria. In acute ischemic stroke, the infarct core forms in brain tissue with no blood supply, while the surrounding ischemic penumbra remains viable briefly through collateral circulation.⁷ Without prompt treatment, the infarct expands. This supports the urgency behind the phrase “time is brain.” The primary goal in treating acute ischemic stroke is to preserve brain tissue in areas of reduced perfusion (ischemic penumbra) by rapidly restoring blood flow. This is achieved through intravenous thrombolysis—using agents like alteplase within 3–4.5 hours or tenecteplase as an alternative—and mechanical thrombectomy, ideally within 6 hours, but extendable to 24 hours in select patients based on imaging criteria (DAWN, DEFUSE 3 trials).⁸

MRI-guided thrombolysis is also an option for wake-up strokes using DWI/FLAIR mismatch. Recent trials (ANGEL-ASPECT, SELECT-2) show that patients with large infarcts (ASPECTS 3–5) may still benefit from thrombectomy, as can patients with basilar artery occlusion (up to 24 hours, per ATTENTION and BAOCHÉ trials).^{9–12} In this patient, since the onset of stroke symptoms was still under 4.5 hours, it was initially decided to perform thrombolysis using alteplase. However, after observation and the absence of clinical improvement based on neurological evaluation — with the onset still under 6 hours and a suspected involvement of a major cerebral

artery — it was then decided to proceed with mechanical thrombectomy.

Hospital management includes maintaining blood pressure below 180/105 mmHg after thrombolysis and under 185/110 mmHg before thrombectomy.⁸ In patients undergoing treatment for stroke, oxygen saturation should be kept above 94%, but excessive oxygen (hyperoxia) offers no benefit. Low carbon dioxide levels (hypocapnia) have been linked to poorer outcomes, so arterial CO₂ (PaCO₂) should be maintained within 4.5–5.0 kPa. Glucose levels should be kept between 140–180 mg/dL, and hyperthermia (>38°C) should be avoided.² Elevated blood sugar is common after stroke and correlates with worse prognosis; therefore, blood glucose should be kept between 7.8–10 mmol/L for the first 48 hours, with regular monitoring to prevent hypoglycemia.¹³

Additionally, high body temperature can harm brain recovery and must be actively managed, although therapeutic hypothermia has not shown benefit in this setting.¹⁴ Early enteral feeding is encouraged, with nasogastric or PEG tubes used if dysphagia persists. Deep Vein Thrombosis (DVT) prophylaxis involves pneumatic compression, and anticoagulation with low-dose heparin may be used when bleeding risk is low.² Seizures should be treated if they occur, but routine prophylaxis is not recommended. Depression screening is important due to high post-stroke incidence.² Cerebral and cerebellar edema must be monitored closely, with early neurosurgical consultation for signs of increased intracranial pressure.² For secondary prevention, aspirin should be given within 24–48 hours, and anticoagulation for atrial fibrillation may be started within 4–14 days, depending on stroke size and risk of hemorrhagic transformation.¹⁵ High-intensity statins are indicated for patients under 75 with atherosclerotic disease, and can be continued if already prescribed pre-stroke.¹⁶ In this patient, the blood pressure was maintained with the target of 140–160 / 80 – 90 mmHg with the medication of Ramipril 5 mg once every 24 hours, Bisoprolol 1.25 mg once daily, Spironolactone 25 mg once daily, and Furosemide 40 mg IV as needed. No

statins were given to this patient because there was no prior atherosclerotic disease known. The patient was also given oxygen supplementation by using a nasal cannula at 3 liters per minute to maintain peripheral oxygen saturation >94%. The patient was monitored closely in the intensive care unit 48 hours after the thrombectomy for signs of increased intracranial pressure and glucose level. Before the thrombectomy, the patient's glucose level was found to be 155 mg/dL, and was evaluated daily post-thrombectomy. The HAS-BLED score in this patient indicates a moderate risk of bleeding, also considering the risk of hemorrhagic transformation, so no anticoagulation was given continuously post-thrombectomy.

General anaesthesia, compared to conscious sedation, offers several advantages during thrombectomy, including airway protection with controlled ventilation, an immobile patient that may reduce procedural complications and recanalisation time, and the presence of dedicated anaesthetic staff who can closely monitor and manipulate physiological parameters.¹⁴ However, it also carries disadvantages such as longer door-to-groin puncture time, increased risk of hypotension—particularly during induction—and the inability to assess neurological changes intra-procedure. Additionally, GA requires more manpower, including postoperative care, and is associated with higher risks of postoperative delirium (POD) and cognitive dysfunction (POCD). Conscious sedation, on the other hand, allows for faster procedural start, more stable haemodynamics, and real-time neurological assessment. Yet, it can lead to patient discomfort or distress, and the potential for movement that may complicate the procedure. It also carries risks of aspiration, hypoxia, and airway obstruction if sedation is not carefully managed. The link between general anaesthesia (GA) and poor neurological outcomes remains debated, as anaesthetic drugs may be both neuroprotective and neurotoxic. Hypotension and abnormal CO₂ levels during anaesthesia can worsen perfusion to the ischaemic brain. However, delays from imaging to reperfusion is also critical. Overall, factors like team efficiency and haemodynamic

control likely matter more than anaesthetic choice. Current AHA/ASA guidelines recommend selecting anaesthesia based on individual patient risks and procedural needs.¹³ In this patient, the procedure was done through general anesthesia for several reasons. First, general anesthesia ensured complete immobility and eliminates patient motion during the delicate procedure, reducing the risk of catheter dislodgment, vessel injury, or prolonged procedure times. Stillness achieved under GA improves the quality of imaging and roadmap guidance during thrombectomy. By facilitating accurate device navigation and clot retrieval, particularly in complex anatomy, it was expected that the time needed from door to needle could be compensated for by the time from needle to retrieval of the clot. Besides better control of blood pressure, CO₂ levels, and oxygenation, general anesthesia also secures the airway with endotracheal intubation, preventing aspiration, especially in this patient who had a high risk of vomiting.

Mitral stenosis (MS) is a significant risk factor for complications during and after surgery.¹⁷ The narrowed mitral valve restricts blood flow into the left ventricle, leading to elevated left atrial pressure, pulmonary congestion, increased pulmonary vascular resistance, and strain on the right heart. For anesthesiologists, managing these patients is complex due to unstable hemodynamics and large fluid shifts. Proper ventricular filling in MS is highly reliant on effective atrial contraction, making the maintenance of sinus rhythm essential. Both bradycardia and tachycardia can impair cardiac output—bradycardia by limiting output despite preserved stroke volume, and tachycardia by reducing diastolic filling time, thus exacerbating pulmonary congestion.¹⁷ Fluid therapy must be approached cautiously; excessive administration can worsen pulmonary edema due to already high left atrial pressure. Furthermore, the heart's pumping efficiency depends on the functional state of both ventricles. Chronic underfilling of the left ventricle can lead to impaired contractility, contributing to heart failure. Hemodynamic goals include maintaining normal systemic vascular resistance (SVR), as reducing afterload does not necessarily improve

output in MS. In addition, avoiding factors that increase pulmonary vascular resistance (such as hypoxia, hypercapnia, acidosis, or inadequate anesthesia) is critical, as these can worsen right heart strain and compromise overall perfusion.¹⁷ In the absence of clear evidence favoring a specific anesthetic agent, drug, and or technique selection during thrombectomy should be tailored to each patient's condition.¹⁸ Rapid decisions are often necessary amidst ongoing preparation, such as monitoring setup and patient positioning. Stroke patients are typically elderly with multiple comorbidities, and communication is frequently impaired—aphasia affects nearly half, making detailed history-taking challenging.² Most patients undergo initial diagnostics upon admission, and there's limited time for further testing. Standard institutional protocols can help streamline and ensure safe anesthesia management. Essential monitoring during thrombectomy includes ECG, pulse oximetry, capnography, and blood pressure measurement.¹⁴ General anesthesia is more prone to cause hemodynamic instability, particularly hypotension.

Arterial lines are advised and can be placed via the same femoral access used by the interventional team, although frequent non-invasive measurements (every 3 minutes) are generally acceptable. General anesthesia is better suited for patients with reduced consciousness, agitation, vomiting, or those with complex stroke presentations (e.g., dominant hemisphere or posterior circulation strokes, or tandem cervical-carotid lesions). While no specific anesthetic drug has demonstrated superiority, commonly used agents include IV drugs like propofol and remifentanyl or volatile anesthetics like sevoflurane. In this case, general anesthesia was delivered by using an intravenous drug combined with a low MAC of gas inhalant, which was meant for maintaining a more stable hemodynamic. Monitoring was done by continuous end tidal CO₂, three-lead electrocardiogram, peripheral oxygen saturation monitoring, and non-invasive blood pressure was measured every three minutes. All stroke patients undergoing thrombectomy require neurological and hemodynamic monitoring for at least 24 hours.¹⁹ The appropriate level and

duration of care depend on individual risk factors and procedural complexity. Those managed under general anaesthesia may require initial monitoring in recovery, high-dependency units, or critical care based on institutional resources.²⁰ While no universal criteria exist for ICU admission, some centers—like the authors'—reserve it for patients with low preprocedural consciousness, pre-existing intubation, or procedural complications. Decisions about escalation should involve collaboration between anaesthetists, stroke physicians, and interventional neuroradiologists. Regardless of demographics or stroke severity, early multidisciplinary care in a dedicated stroke unit significantly improves the chance of regaining independence. Ideally, patients should be assessed within 24–36 hours. Early interventions should include swallow screening, nutritional support, DVT prevention, and initiation of rehabilitation. Aspirin is recommended within 24–48 hours of stroke onset, though its use is delayed in those who received thrombolysis.¹⁴ For patients receiving stents, dual antiplatelet therapy may be necessary. In this case, there is no use of any stents, and post-procedurally, the patient was then monitored in a high-dependency unit for the next 3 days before discharge.

Conclusion

This case underscores the importance of individualized anesthetic decision-making in complex stroke patients, particularly those with valvular heart disease. General anesthesia was selected to ensure airway protection, hemodynamic stability, and procedural safety, highlighting how anesthesia modality may directly influence thrombectomy success and patient outcomes.

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