CASE REPORT

Anesthesia Management and Complications of Reperfusion Syndrome After Thrombectomy in Acute Ischemic Stroke: A Case Report

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ABSTRACT

Background: Acute ischemic stroke (AIS) caused by cerebral vascular occlusion requires rapid reperfusion to prevent irreversible neuronal damage. Mechanical thrombectomy is currently the standard of care alongside intravenous thrombolysis for appropriately selected patients with AIS, as supported by randomized clinical trials and stroke guidelines. However, despite its clinical benefits, thrombectomy presents significant anesthesiological challenges, particularly regarding the prevention of post-procedural neurological complications, such as reperfusion syndrome.

Case: A 64-year-old man with heart disease and diabetes mellitus underwent mechanical thrombectomy for AIS under general anesthesia with close monitoring of blood pressure, ventilation, and hemodynamic status. Following successful recanalization, the patient developed persistent aphasia with suspected cerebral edema. Reperfusion syndrome was considered based on the temporal relationship between restored cerebral blood flow and neurological deterioration, while other possible causes were evaluated clinically.

Discussion: Reperfusion syndrome is a recognized but uncommon complication after successful cerebral recanalization. Intensive care unit (ICU) management included close neurological observation, hemodynamic stabilization, blood pressure control, optimization of oxygenation and ventilation, targeted temperature management at 35–36°C for 24 hours, and antioxidant therapy with N-acetylcysteine 200 mg every 8 hours. These interventions were applied as individualized supportive strategies rather than established standard treatment.

Conclusion: This case highlights the importance of individualized anesthetic management, strict physiological control, early recognition of post-thrombectomy neurological deterioration, and multidisciplinary ICU care to minimize secondary brain injury.

Keywords: acute ischemic stroke; antioxidant; cerebral vascular occlusion; general anesthesia; mechanical thrombectomy; reperfusion syndrome

INTRODUCTION

Acute ischemic stroke (AIS) occurs when blood flow to the brain is blocked due to a blood vessel occlusion, leading to rapid and potentially irreversible neuronal damage. In AIS caused by cerebral vascular occlusion, rapid reperfusion is critical to salvage the ischemic penumbra and improve neurological outcomes. Mechanical thrombectomy has become the recommended standard reperfusion therapy in guidelines for appropriately selected patients with AIS. The 2019 American Heart Association/American Stroke Association guidelines recommend mechanical thrombectomy within 16 hours and consider it reasonable up to 24 hours in selected patients. Similarly, the European Stroke Organisation–European Society for Minimally Invasive Neurological Therapy guidelines state that mechanical thrombectomy is the standard of care for patients with acute stroke. Evidence from randomized clinical trials and meta-analyses of individual patient data shows that endovascular thrombectomy significantly reduces disability at 90 days compared with standard medical therapy.[1,2,3]

Despite its efficacy, the perioperative management of thrombectomy remains a complex challenge for anesthesiologists. Patients presenting with AIS often exhibit multi-organ dysfunction and significant comorbidities such as cardiovascular disease or diabetes mellitus. They are frequently on anticoagulant therapy, which heightens the risk of intraprocedural hemorrhage. Consequently, the anesthesiologist must navigate a narrow therapeutic window: maintaining precise blood pressure control to ensure collateral perfusion while preventing reperfusion injury, ensuring hemodynamic stability, and monitoring neurological status in real-time. Furthermore, meticulous regulation of ventilation and oxygenation is mandatory to prevent hypoxia or hypocapnia, both of which can exacerbate secondary brain injury.[4,5]

Although the clinical benefits of mechanical thrombectomy are well established, post-recanalization reperfusion injury remains an important clinical problem. The reported incidence varies depending on the definition used, ranging from radiological hyperperfusion, cerebral edema, hemorrhagic transformation, to clinically apparent cerebral hyperperfusion syndrome. A study evaluating post-ischemic hyperemia after endovascular therapy found hyperemia in 52% of patients early after EVT, 49% at 24 hours, and 40% at 5 days, suggesting that perfusion disturbance after successful recanalization is relatively common. In contrast, clinically apparent cerebral hyperperfusion syndrome appears less frequent; a recent randomized controlled trial reported a 7-day incidence of cerebral hyperperfusion syndrome of 14.1% in the placebo group after mechanical thrombectomy. Hemorrhagic manifestations of reperfusion-related injury are also clinically relevant, with meta-analytic data showing symptomatic intracranial hemorrhage after endovascular thrombectomy in approximately 6.72% of patients, while a large multicenter registry reported any intracranial hemorrhage in 24.2% and symptomatic intracranial hemorrhage in 4.5% of cases. Therefore, post-thrombectomy reperfusion injury represents a meaningful peri-procedural concern rather than an isolated phenomenon. This case report highlights the anesthetic management of a high-risk patient with multiple comorbidities who developed suspected post-thrombectomy reperfusion syndrome, emphasizing the importance of strict hemodynamic control, early neurological assessment, and intensive post-procedural monitoring.[6,7,8,9]

The purpose of this case report is not to determine the optimal anesthetic technique or drug regimen for mechanical thrombectomy, but rather to describe the anesthetic management of a high-risk patient with AIS who underwent mechanical thrombectomy and developed reperfusion syndrome following successful recanalization. This report emphasizes the importance of individually tailored anesthetic planning, hemodynamic control, early detection of post-procedural neurological deterioration, and intensive monitoring to minimize secondary brain injury in patients with complex comorbidities.

CASE

A 64-year-old male (70 kg, 170 cm, body mass index 28.22 kg/m²) was referred to the hospital following a sudden onset of decreased consciousness six hours prior to admission. The patient's family reported that he was found unresponsive and somnolent in his room, despite having been ambulatory and communicative earlier that day. Clinical observations by the family noted decreased activity on the left side of his body, though symptoms such as facial drooping, slurred speech, visual disturbances, and projectile vomiting were denied. The patient had a significant medical history, including a previous stroke in 2021 (with full recovery), chronic heart disease, and type 2 diabetes mellitus. His daily medication regimen consisted of aspirin, bisoprolol, candesartan, furosemide, and glimepiride. Notably, he was a heavy smoker who had only ceased the habit three months prior.

Upon physical examination, the patient's glasgow coma scale (GCS) was E2V2M5. Neurological assessment revealed left-sided supranuclear facial nerve (CN VII) paresis and grade 1 flaccid lateralization of the left extremities. Vital signs recorded a blood pressure of 140/90 mmHg, a heart rate of 97 bpm, and an oxygen saturation of 100% on 2 lpm nasal cannula. Airway evaluation showed Mallampati class II with incomplete dentition. Supporting laboratory tests were largely within normal limits, with the exception of a slightly elevated creatinine level (1.38 mg/dL) and a reduced e-GFR (53.65 ml/min/1.73m²). Pre-procedural non-contrast head CT identified chronic cerebral infarctions in the right nucleus lentiformis and left corona radiata, while echocardiography revealed severe cardiac impairment, including left ventricular dilatation, concentric hypertrophy, and a severely reduced ejection fraction of 17.64% with Grade III diastolic dysfunction. (Figure 1)

The patient was classified as American Society of Anesthesiologists IV and scheduled to undergo mechanical thrombectomy under general anesthesia. Induction was performed with sufentanyl at a dose of 0.25 mcg/kg, Sevoflurane maintenance at 2-3 V% until sedation was achieved, and rocuronium at 0.5 mg/kgbb. The procedure lasted 60 minutes, during which intraoperative hemodynamics remained relatively stable, with blood pressure fluctuating between 130–145/75–91 mmHg, monitored via an arterial line inserted prior to anesthesia. Digital subtraction angiography (DSA) through left common coronary artery (LCCA) injection confirmed a total proximal occlusion of the left internal carotid artery (LICA). Aspiration thrombectomy was performed twice using Ballast and Sofia 6F catheters, successfully achieving maximal restoration of the middle cerebral artery (MCA) circulation. Additional vascular findings included 50% stenosis of the right internal carotid artery (RICA) and critical stenosis of the right vertebral artery (RVA) ostium.

Following the intervention, the patient was transferred to the intensive care unit (ICU) for close neurological and hemodynamic monitoring. Although his level of consciousness improved during the post-procedural period, he developed persistent aphasia accompanied by suspected cerebral edema, raising concern for reperfusion-related neurological deterioration. Other potential causes of post-thrombectomy neurological worsening, including hemorrhagic transformation, re-occlusion, seizure, metabolic disturbance, infarct progression, and residual anesthetic effects, were clinically considered.

Supportive neurocritical care was initiated in the ICU with the main goal of preventing secondary brain injury. Hemodynamic status, oxygenation, ventilation, blood glucose, and body temperature were closely monitored. As adjunctive neuroprotective measures, targeted temperature management was performed by maintaining the patient's core body temperature at 35–36°C for 24 hours, followed by gradual rewarming. Continuous monitoring was conducted to prevent shivering, hemodynamic instability, electrolyte disturbances, and other potential complications related to temperature modulation. Antioxidant therapy was also administered using N-acetylcysteine 200 mg every 8 hours to help mitigate oxidative stress associated with suspected reperfusion injury.

Additional medical therapy included intravenous citicoline 500 mg every 12 hours, oral acetylsalicylic acid 80 mg once daily, oral simvastatin 40 mg every 24 hours, and intravenous paracetamol 1000 mg every 8 hours. After 48 hours of intensive monitoring and stabilization of his hemodynamic and neurological status, the patient's condition was deemed stable enough for transfer to the general ward for further neurological follow-up and rehabilitation.



Figure 1. CT Angiography identified chronic cerebral infarctions in the right nucleus lentiformis and left corona radiata

DISCUSSION

The management of AIS through mechanical thrombectomy presents a high-stakes challenge for anesthesiologists, particularly when complicated by severe cardiac comorbidities and post-procedural reperfusion syndrome. The novelty of this case lies in the successful anesthetic navigation of a patient with a critically low ejection fraction (17.64%) and multi-vessel disease who developed reperfusion-induced aphasia despite successful recanalization. While mechanical thrombectomy is the gold standard for large vessel occlusion, this case highlights that the restoration of blood flow is not merely a mechanical success but a physiological transition that carries significant risks of secondary brain injury.[4,5,10,11] In this case, general anesthesia was selected over conscious sedation. Existing literature often debates the superiority of these techniques; however, general anesthesia provides definitive airway protection and total immobility, which reduces the risk of iatrogenic vascular injury and precise control over arterial carbon dioxide levels.[12–17] The trade-offs between these approaches are summarized in Table 1.

A critical aspect of this patient's management was physiological stabilization and early prevention of secondary brain injury after successful recanalization. In this case, general anesthesia was selected due to the patient's reduced level of consciousness, the need for airway protection, and the requirement for immobility during the endovascular procedure. Continuous arterial blood pressure monitoring was used, and blood pressure was maintained within 130–145/75–91 mmHg to preserve cerebral perfusion while avoiding excessive hypertensive surges after recanalization.[18,19,20]

Following the procedure, the patient developed persistent aphasia with suspected cerebral edema, raising concern for reperfusion-related neurological deterioration. Post-procedural management was performed in the ICU and focused on neurocritical supportive care, including close neurological observation, continuous hemodynamic monitoring, blood pressure control, optimization of oxygenation and ventilation, glucose monitoring, and surveillance for hemorrhagic transformation, seizure, metabolic disturbance, and possible re-occlusion.[21,22,23,24]

As adjunctive neuroprotective measures, targeted temperature management and antioxidant therapy were administered after suspected reperfusion-related neurological deterioration. Targeted temperature management was initiated in the ICU by maintaining the patient's core body temperature at 35–36°C for 24 hours, followed by gradual rewarming. Continuous temperature monitoring was performed, with attention to the prevention of shivering, hemodynamic instability, electrolyte disturbances, and other potential complications. Antioxidant therapy consisted of N-acetylcysteine 200 mg every 8 hours to help mitigate oxidative stress associated with suspected reperfusion injury. These interventions were applied as individualized supportive strategies rather than established standard treatment, considering that current evidence for routine hypothermia and antioxidant therapy after post-thrombectomy reperfusion injury remains limited.[23–28]

Table 1. Comparison of general anesthesia and conscious sedation in thrombectomy (12-17)

Anesthetic technique	Advantages	Disadvantages
General anesthesia	Airway protection; reduced aspiration risk; patient immobility; controlled ventilation and $PaCO^2$	Potential for induction hypotension; delayed neurological assessment; longer "door-to-needle" time
Conscious sedation	Faster procedure initiation; better hemodynamic stability; continuous neurological monitoring	Risk of patient movement; airway obstruction or hypoxia; potential conversion to general anesthesia

CONCLUSION

This case highlights that successful mechanical recanalization does not eliminate the risk of post-procedural neurological deterioration. In high-risk patients with severe cardiac dysfunction and multiple comorbidities, anesthetic management should prioritize physiological stability, including strict blood pressure control, avoidance of induction-related hypotension, prevention of excessive post-recanalization hypertension, and optimization of oxygenation and ventilation. Persistent aphasia after thrombectomy should prompt consideration of reperfusion syndrome, while other causes, such as hemorrhagic transformation, re-occlusion, seizure, metabolic disturbance, and infarct progression, must be evaluated. The key lesson from this case is that anesthesiologists have an important role not only in supporting successful recanalization but also in early recognition and management of secondary brain injury through intensive post-thrombectomy monitoring and multidisciplinary neurocritical care.

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