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CASE REPORT

Rescue mechanical thrombectomy for large vessel occlusion during endovascular treatment of aneurysmal subarachnoid hemorrhage: A case report

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ABSTRACT

Aneurysmal subarachnoid hemorrhage (aSAH) is a life-threatening cerebrovascular emergency in which thromboembolic complications during endovascular treatment are rare but potentially devastating. This case report describes a 52-year-old male patient with aSAH caused by rupture of an anterior communicating artery aneurysm who developed an intra-procedural middle cerebral artery occlusion during coil embolization. Rescue mechanical thrombectomy achieved complete recanalization (thrombolysis in cerebral infarction Grade 3) followed by successful aneurysm coiling; however, the patient subsequently developed a large infarction, cerebral edema, and fatal brainstem compression. This case highlights that although rescue thrombectomy can provide technical success when thrombolysis is contraindicated, angiographic recanalization does not necessarily translate into favorable clinical outcomes and emphasizes the need for careful peri-procedural management in patients with combined ischemic and hemorrhagic pathology.

Keywords: Endovascular treatment, mechanical thrombectomy, neurointervention, stroke, subarachnoid hemorrhage.

Aneurysmal subarachnoid hemorrhage (aSAH) is a life-threatening cerebrovascular emergency requiring immediate intensive care management. It results from the rupture of a cerebral aneurysm, leading to hemorrhage in the subarachnoid space between the arachnoid mater and pia mater. Aneurysmal subarachnoid hemorrhage represents a distinct subtype of stroke, affecting approximately 9 per 100,000 individuals annually. Nearly half of these cases occur in individuals under 55 years of age, making it a leading cause of morbidity and mortality in younger populations. Due to its high fatality rate and long-term disability, the loss of productive life years resulting from aSAH is comparable to ischemic stroke, the most prevalent form of cerebrovascular disease.[1,2]

The primary objective of this report is to present a complex case of aSAH complicated by an intraoperative thromboembolic event during endovascular treatment (EVT), which required rescue mechanical thrombectomy (MT). While EVT has become the preferred intervention for many ruptured aneurysms due to its minimally invasive nature and favorable outcomes compared to surgical clipping, the procedure carries inherent risks, including aneurysmal perforation, coil migration, and thromboembolic complications. These adverse events, particularly thromboembolic complications,

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significantly impact clinical outcomes and may necessitate urgent rescue interventions.

Aneurysmal rupture is the most frequent cause of SAH, accounting for approximately 85% of cases.[2] The primary management goal in aSAH is to prevent rebleeding while minimizing secondary ischemic injury. However, the treatment of ruptured aneurysms remains complex, requiring individualized approaches based on aneurysm morphology, location, and patient factors. Endovascular treatment, particularly coil embolization, has revolutionized the management of intracranial aneurysms, offering a less invasive alternative to neurosurgical clipping. The International Subarachnoid Aneurysm Trial (ISAT) demonstrated that EVT is associated with a lower risk of post-procedural decline in the Glasgow-Liege Coma Scale (GLCS) and reduced post-treatment epilepsy, particularly in cases involving aneurysms in deep or surgically inaccessible locations. Despite these advantages, EVT is not without complications, with thromboembolic events occurring in approximately 14% of cases, predominantly in patients with ruptured aneurysms.[3-5]

Thromboembolic complications during EVT present significant challenges, as they can lead to complete or partial occlusion of arteries at the aneurysm site or within distal vascular territories. The risk of such events is heightened in the setting of aSAH due to the associated vasospasm and hypercoagulability.[4] Unlike patients with acute ischemic stroke, who are candidates for intravenous thrombolysis, patients with aSAH cannot safely receive systemic thrombolytic therapy due to the risk of catastrophic rebleeding. When thromboembolic complications occur intraoperatively, MT remains the only viable option, though its use in this setting is not yet standardized. Rescue thrombectomy is estimated to be required in approximately 2.37% of EVT-treated ruptured aneurysms.[6]

This report describes the case of a 52-year-old patient with aSAH due to an anterior communicating artery (AComA) aneurysm, complicated by an intraoperative thromboembolic occlusion of the left middle cerebral artery (MCA) during coil embolization. The report aims to provide a detailed account of the clinical presentation, diagnostic evaluation, and therapeutic interventions employed. The discussion will highlight the procedural decision-making process, including the rationale for choosing MT over pharmacologic thrombolysis. Furthermore, the case underscores the challenges of optimizing perioperative and postoperative management in high-risk aSAH patients.

By detailing the clinical course, technical aspects of EVT, and subsequent rescue MT, this report aims to contribute to the ongoing efforts to refine management strategies for aSAH. The case emphasizes the need for continued research into intraoperative strategies, risk mitigation techniques, and standardized protocols for post-procedural care to improve outcomes in this complex and high-risk patient population.

CASE REPORT

A 52-year-old male patient was brought to the emergency department by ambulance after being found in a compromised state outdoors, exhibiting vomiting and loss of bladder and bowel control. The prehospital team recorded a blood pressure (BP) of 240/120 mmHg and administered intravenous clonidine. Upon arrival, the patient was disoriented regarding time and place and appeared restless. Physical examination revealed bilateral vesicular breath sounds, sinus tachycardia at approximately 100 beats per minute, and a BP of 240/118 mmHg. The initial neurological assessment revealed marked neck stiffness (suggestive of severe meningeal irritation), intact cranial nerve function, and mild quadriparesis (muscle strength graded as 4/5 on the Medical Research Council scale). Deep tendon reflexes were brisk (3+ bilaterally), and Babinski's sign was negative. The GLCS score was 16, and the National Institutes of Health Stroke Scale (NIHSS) score was 11.

The patient had no comorbidities other than poorly controlled hypertension and no history of antiplatelet or anticoagulant use. Initial management with loop diuretics, intravenous clonidine, and oral calcium channel blockers resulted in a modest reduction of BP to approximately 200/100 mmHg.

The initial laboratory tests, including complete blood count, C-reactive protein, renal and hepatic panels, electrolytes, and coagulation studies, were within normal limits, except for slightly elevated low-density lipoprotein and blood glucose levels. A non-contrast computed tomography (CT) scan of the head performed upon admission revealed diffuse SAH involving the falx cerebri, both Sylvian fissures, and the tentorium cerebelli, with small amounts of blood in the third and fourth ventricles. No ischemic injury was evident. Given the extensive hemorrhage and its diffuse distribution, a ruptured brain aneurysm was suspected. Computed tomography angiography, considered the gold standard for aneurysm detection, confirmed a small saccular aneurysm (approximately 3.3 × 3.2 × 3.3 mm) in the AComA without additional vascular pathology, as shown in Figure 1.

Based on clinical and radiological assessments, the patient was classified as GLCS 16, NIHSS 11, Hunt and Hess Grade 3–4, World Federation of Neurosurgical Societies Grade 2, and Fisher Scale 4. Intravenous nimodipine was initiated immediately to prevent vasospasm and secondary ischemic complications.

An interdisciplinary team, including a neurologist, a neurosurgeon, an anesthesiologist, and an interventional radiologist, determined that the patient was a suitable candidate for EVT via coil embolization to prevent rebleeding.

Within the first 24 hours, the patient was taken for EVT under general intubation anesthesia. After induction of general anesthesia and sterile preparation, femoral arterial access was obtained through the right femoral artery. Initial diagnostic digital subtraction angiography (DSA) was performed to evaluate the cerebral vessels and confirm the morphology of the AComA aneurysm.

Following the decision to proceed with endovascular coil embolization, 5000 international units (IU) of heparin was administered intravenously to prevent intra-procedural thromboembolic events. The intervention was performed under continuous DSA guidance. A 6 Fr arterial introducer was advanced via the right femoral artery, and a HeadWay 17 microcatheter (MicroVention, Inc., Aliso Viejo, CA, USA) was carefully navigated through the left MCA to the AComA to achieve aneurysm access and enable subsequent treatment.

During the procedure and intra-arterial contrast injection, an occlusion of the proximal segment (M1) of the left MCA was detected, indicating an ipsilateral thromboembolic complication, as shown in Figure 2. Intravenous thrombolysis was contraindicated, and consequently, a rescue MT was performed using an SOFIA 5F/6F aspiration catheter (MicroVention, Inc., Aliso Viejo, CA, USA), a Traxcess-14 micro-guidewire, and a ERIC 3x20 mm retrieval device (MicroVention Europe, Amsterdam, The Netherlands). Subsequent angiography confirmed complete revascularization

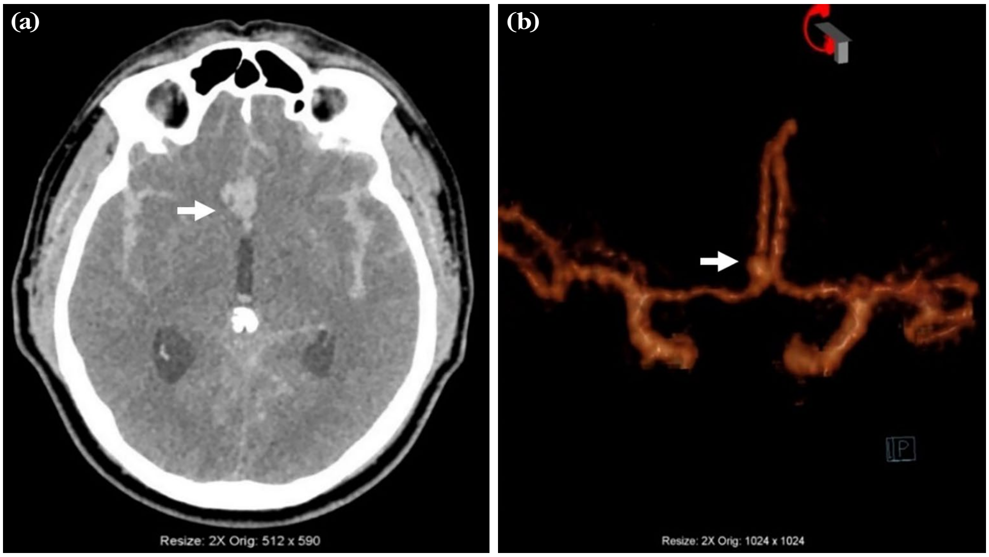

Figure 1. Computed tomography and CT angiography demonstrating diffuse subarachnoid hemorrhage and an AComA aneurysm. **(a)** Non-contrast CT of the head in the axial plane demonstrating diffuse subarachnoid hemorrhage involving the basal cisterns and cortical sulci. **(b)** CT angiography with volume rendering technique illustrating the cerebral arterial circulation and revealing a saccular aneurysm arising from the AComA (maximum diameter approximately 3.3 mm, arrow). No additional aneurysms, significant vasospasm, or other vascular abnormalities are identified. The imaging findings are consistent with the presumed source of diffuse aneurysmal subarachnoid hemorrhage.

CT, computed tomography; AComA, anterior communicating artery.

Figure 2. Intraoperative thromboembolic occlusion of the left MCA and rescue mechanical thrombectomy. Digital subtraction angiography of the left internal carotid artery was performed during endovascular treatment. **(a)** Digital subtraction angiography demonstrating acute thromboembolic occlusion of the proximal M1 segment of the left MCA (arrow). **(b)** Intraoperative rescue aspiration and mechanical thrombectomy of the occluded M1 segment of the left MCA. **(c)** Final post-thrombectomy angiography demonstrating complete recanalization of the left MCA with restoration of antegrade flow throughout the MCA territory (TICI grade 3), with re-establishment of the previously occluded M1 segment (arrow).

MCA, middle cerebral artery.

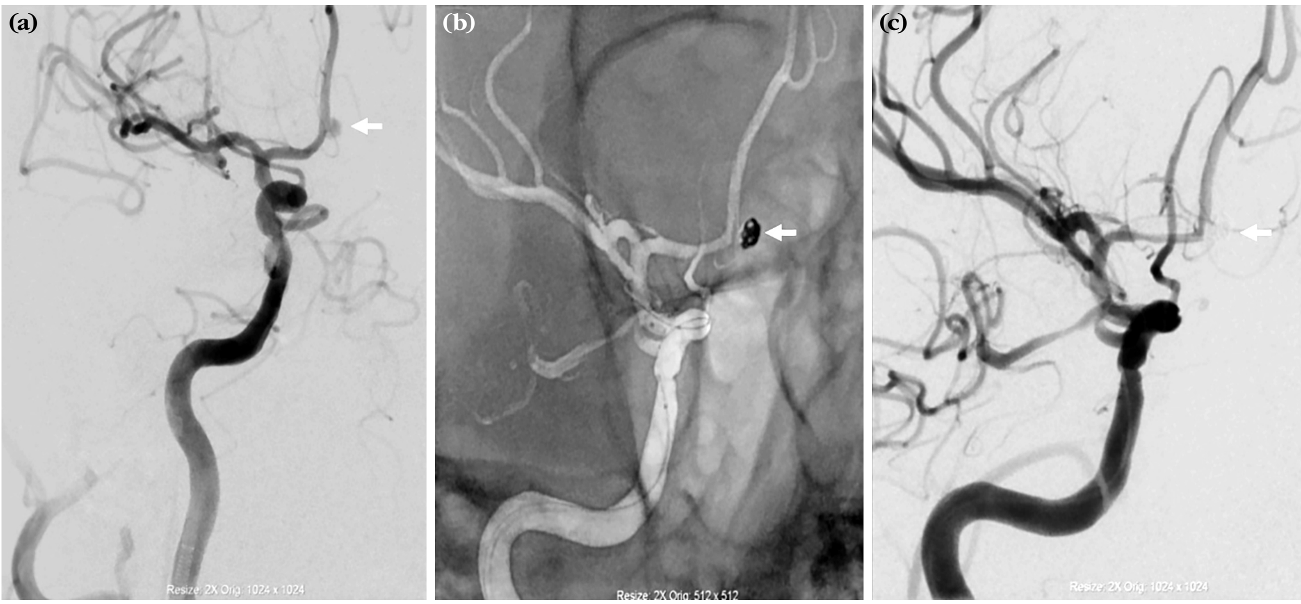

Figure 3. Endovascular coil embolization of the AComA aneurysm. Digital subtraction angiography (DSA) of the right internal carotid artery obtained during endovascular treatment. **(a)** Pre-embolization DSA demonstrating a saccular aneurysm arising from the AComA (arrow). **(b)** Intraoperative DSA showing coil deployment during embolization of the AComA aneurysm. **(c)** Post-embolization angiography demonstrating complete occlusion of the AComA aneurysm and preserved arterial patency, without distal flow impairment (arrow).

AComA, anterior communicating artery.

Figure 4. Early ischemic injury following rescue mechanical thrombectomy. Follow-up non-contrast computed tomography of the brain, obtained 24 h after endovascular treatment, demonstrated a newly developed hypodense area involving the left insular region and temporoparietal cortex, consistent with an acute ischemic infarction in the left middle cerebral artery territory. Residual subarachnoid hemorrhage is decreased compared with baseline imaging, without evidence of aneurysmal rebleeding.

of the occluded M1-M2 segment of the left MCA (Figure 2).

Despite a satisfactory angiographic outcome (thrombolysis in cerebral infarction [TICI] grade 3), the duration of the occlusion and the collateral vessels may have influenced infarct volume. Perfusion imaging post-thrombectomy was not available to assess tissue viability, which limits the interpretation of the functional outcome.

Thereafter, the microcatheter and guidewire were re-navigated to the ruptured aneurysm. A single HydroSoft 3D coil (3×8 mm) (MicroVention, Inc., Aliso Viejo, CA, USA) was deployed using a V-Grip device (MicroVention, Inc., Aliso Viejo, CA, USA) to embolize the aneurysm. Immediate postprocedural CT imaging confirmed optimal coil placement without additional hemorrhage or hypodensities, as shown in Figure 3.

The patient was transferred to the neurological intensive care unit and remained intubated. Shortly after the procedure, the patient was able to follow simple commands.

The next day, the patient's neurological status deteriorated, with progression from mild quadriparesis to severe quadriparesis, more pronounced on the right side. Due to adequate spontaneous breathing, the patient was extubated.

A follow-up CT scan performed 24 h after the procedure revealed a hypodensity in the left insular cortex, consistent with an acute temporo-parietal ischemic stroke, along with a slight reduction in SAH, as shown in Figure 4.

Following multidisciplinary consultation, a low dose of low-molecular-weight heparin (Nadroparin 0.4 mL) was incorporated into the treatment regimen. One day later, the patient suddenly became comatose and quadriplegic, with anisocoria indicating brainstem dysfunction-GLCS: 7, NIHSS: 28. The patient was reintubated and placed on invasive ventilation using synchronized intermittent mandatory ventilation mode, and a nasogastric tube was inserted. During this period, a markedly elevated BP (up to 220/100 mmHg) was recorded. Due to the patient's clinical deterioration into coma with quadriplegia, another CT scan was performed to rule out aneurysmal rebleeding. The neuroimaging revealed an enlargement of the ischemic stroke area with diffuse cerebral edema, and cerebellar tonsillar herniation (16-17 mm into the foramen magnum) compressing the brainstem, as shown in Figure 5.

Shortly thereafter, hyperpyrexia and a fixed gaze with dilated pupils were observed. The patient's BP subsequently dropped abruptly. Despite administration of high-dose dopamine (up to 10 mcg/kg/min), his clinical condition deteriorated markedly, ultimately culminating in death several hours later. A written informed consent was obtained from the patient.

DISCUSSION

Aneurysmal subarachnoid hemorrhage remains a devastating cerebrovascular emergency with high rates of morbidity and mortality, despite advances in neurocritical care and EVT. Coil embolization is currently a mainstay for aneurysm occlusion, but periprocedural thromboembolic complications continue to represent one of the most frequent adverse events.

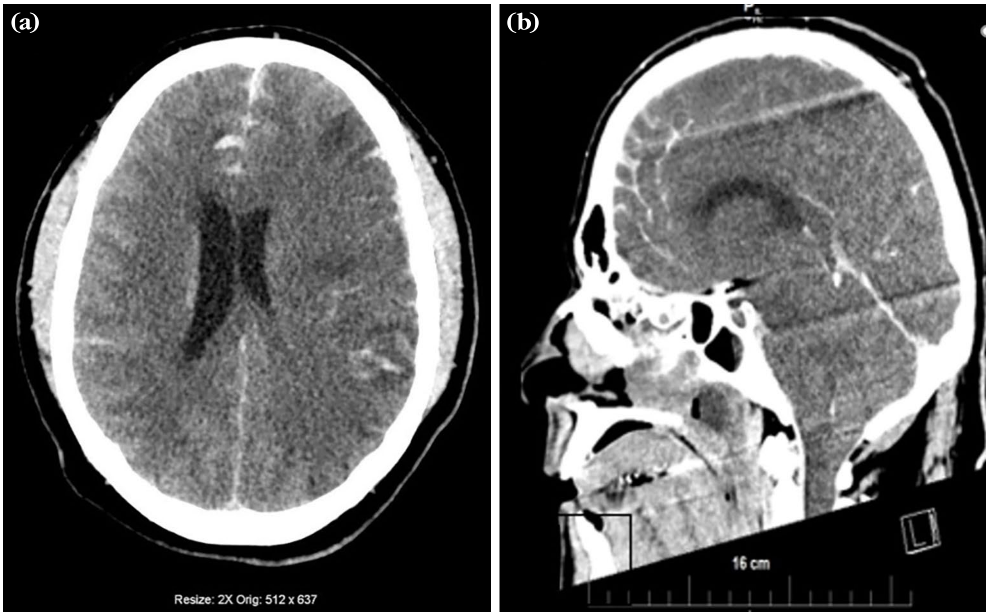

Figure 5. Malignant cerebral edema with transtentorial and tonsillar herniation. Follow-up non-contrast CT of the brain was obtained after the clinical deterioratioh. **(a)** Axial CT image demonstrating extensive infarction of the left cerebral hemisphere with diffuse hypodensity, severe cerebral edema, effacement of cortical sulci, and marked mass effect with significant midline shift. **(b)** Sagittal CT reconstruction illustrating inferior displacement of the cerebellar tonsils by approximately 16-17 mm through the foramen magnum, resulting in compression of the brainstem, consistent with malignant hemispheric infarction and fatal secondary brain injury.

CT, computed tomography.

The primary objective in managing SAH is to prevent secondary complications. In this case, secondary ischemic injury superimposed on the initial subarachnoid hemorrhage created a dual pathology, amplifying cerebral edema and contributing to rapid neurological deterioration. The early phase of injury, often referred to as "early brain injury," is characterized by a sudden increase in intracranial pressure and a subsequent decrease in cerebral perfusion pressure, leading to global ischemia. In addition, vasospasm triggered by hemorrhagic extravasation, disruption of the blood-brain barrier, and impaired cerebrovascular autoregulation further exacerbate the injury.[3]

Thromboembolic events during aSAH may occur spontaneously or as periprocedural complications identified incidentally during EVT. Iatrogenic factors, such as the manipulation of a micro-guidewire during EVT, can increase the risk of thrombus formation. Although thromboembolic complications are the most common adverse events during aneurysm coiling, their intraoperative occurrence remains relatively infrequent. Data regarding the optimal treatment and prophylaxis of these complications are contradictory. Many centers administer heparin in doses ranging from 3,000 to 5,000 IU as prophylaxis following the deployment of the first platinum coil. In this case, 5,000 IU of heparin was initiated prior to commencing the coiling procedure. Despite the use of anticoagulants, thromboembolic events may still occur. Some reports describe the administration of aspirin (100–200 mg) and clopidogrel (75 mg) via a nasogastric tube after initial heparin-induced anticoagulation. However, this strategy has achieved incomplete recanalization in approximately 20% of cases with thromboembolic complications. When recanalization fails, intra-arterial thrombolysis with urokinase (240,000–300,000 IU) has been employed, resulting in complete or near-complete recanalization in the remaining patients.[6] A retrospective study of 19 patients who developed thromboembolic complications during endovascular coiling and were subsequently treated with intra-arterial thrombolysis reported favorable outcomes in 14 cases, three patients experienced persistent neurological deficits, and two died- one due to pre-existing subarachnoid hemorrhage and the other from a massive intracranial hematoma secondary to thrombolytic therapy.[7]

Although intra-arterial thrombolysis is a viable treatment option for thromboembolic complications during aneurysm coiling, its widespread use is limited by the significant risk of irreversible intracranial hemorrhage, particularly in acute aSAH.[8,9] Alternative strategies, such as MT, have been proposed, with several studies demonstrating favorable outcomes.[10,11] It is also important to note that SAH may occur in patients who have undergone intravenous thrombolysis followed by endovascular thrombectomy, though this does not appear to significantly impact clinical outcomes.[12]

Even after successful coil embolization and subsequent rescue MT, optimal postprocedural patient management remains a therapeutic dilemma. Given the lack of standardized guidelines, some centers initiate delayed antiplatelet therapy post-coiling, while others avoid it entirely in high-bleed-risk scenarios. Some studies advocate initiating low doses of unfractionated or low-molecular-weight heparin within the first 24 h postoperatively, while antiplatelet agents are generally contraindicated due to the increased bleeding risk.[11]

Although full recanalization (TICI grade 3) was achieved, the patient's poor clinical outcome illustrates that angiographic success represents only one aspect of effective reperfusion therapy. Neuronal recovery and neurological improvement ultimately depend on collateral circulation, infarct dynamics, and postprocedural cerebral hemodynamics. As highlighted by recent analyses, the extent of collateral circulation is a critical determinant of post-thrombectomy outcome, influencing both infarct volume and cerebral edema formation.[13] Moreover, in hemorrhagic scenarios such as aSAH, the absolute contraindication to systemic thrombolysis poses a therapeutic dilemma- balancing the need to prevent vessel re-occlusion against the risk of re-bleeding, as emphasized in prior analyses of reperfusion therapies and their hemorrhagic sequelae.[14]

This case highlights the complex and multifactorial challenges of managing aSAH complicated by intraoperative large vessel occlusion (LVO). Long-term outpatient care should be carefully individualized, with personalized patient management strategies according to the severity of the subarachnoid hemorrhage, the extent of secondary ischemic injury, and the presence of concurrent cardiovascular or thromboembolic risk factors. Optimal outcomes require a coordinated multidisciplinary approach involving neurologists, neurosurgeons, interventional neuroradiologists, intensive care specialists, rehabilitation physicians, and cardiovascular consultants. Such collaboration enables comprehensive surveillance for delayed ischemic deficits, prevention of thromboembolic recurrence, regulation of BP and coagulation profiles, and structured rehabilitation aimed at maximizing neurological recovery and quality of life. This integrated care model is particularly important in patients with dual ischemic and hemorrhagic pathology, where therapeutic decisions must balance the competing risks of rebleeding and recurrent thrombosis.

In conclusion, this case highlights the complex challenges of managing aSAH complicated by intraoperative LVO. Although rescue MT achieved complete recanalization and allowed successful aneurysm coiling, the clinical outcome was fatal due to delayed ischemic injury and cerebral edema. These findings emphasize that even technically optimal angiographic results do not necessarily translate into favorable neurological outcomes in patients with combined hemorrhagic and ischemic pathology. Rescue thrombectomy remains a technically feasible and potentially life-saving intervention in selected aSAH cases complicated by thromboembolic events. However, the absence of standardized postoperative management protocols continues to hinder outcome improvement. A structured, multidisciplinary approach, integrating neurology, neurosurgery, neuroradiology, intensive care, and rehabilitation, is essential to optimize postoperative care, prevent secondary ischemic injury, and improve long-term functional recovery in this high-risk patient group.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions: M.T.M., M.V.: Idea/concept; M.T.M., M.V., N.I., V.D.K.: Design; M.T.M., T.D., G.T., C.B.: Control/supervision; M.V., N.I., T.D., V.D.K., G.T., M.T.M.: Data collection and/or processing; M.T.M., V.D.K., G.T., C.B.: Analysis and/or interpretation; M.V., N.I., V.D.K., M.T.M.: Literature review; M.V., M.T.M.: Writing the article; M.T.M., G.T., C.B.: Critical review.

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REFERENCES

1. de Rooij NK, Linn FH, van der Plas JA, Algra A, Rinkel GJ. Incidence of subarachnoid haemorrhage: A systematic review with emphasis on region, age, gender and time trends. J Neurol Neurosurg Psychiatry 2007;78:1365-72. doi: 10.1136/jnnp.2007.117655.
2. Lindgren A, Vergouwen MD, van der Schaaf I, Algra A, Wermer M, Clarke MJ, et al. Endovascular coiling versus neurosurgical clipping for people with aneurysmal subarachnoid haemorrhage. Cochrane Database Syst Rev 2018;8:CD003085. doi: 10.1002/14651858.CD003085.pub3.
3. Wirtz R, Woischneck D, Claasen H, Scheuerle A, Röhrer S, Schebesch KM, et al. Aneurysmal subarachnoid haemorrhage-pathophysiology and clinical management. In: Kapapa T, König R, editors. Spontaneous Subarachnoid Haemorrhage: Well-known and New Approaches. New York: Nova Science Publishers; 2016. p. 1-22.
4. Ihn YK, Shin SH, Baik SK, Choi IS. Complications of endovascular treatment for intracranial aneurysms: Management and prevention. Interv Neuroradiol 2018;24:237-45. doi: 10.1177/1591019918758493.
5. Altay T, Kang HI, Woo HH, Masaryk TJ, Rasmussen PA, Fiorella DJ, et al. Thromboembolic events associated with endovascular treatment of cerebral aneurysms. J Neurointerv Surg 2011;3:147-50. doi: 10.1136/jnis.2010.003616.
6. Abdalkader M, Piotin M, Chen M, Ortega-Gutierrez S, Samaniego E, Weill A, et al. Coil migration during or after endovascular coiling of cerebral aneurysms. J Neurointerv Surg 2020;12:505-11. doi: 10.1136/neurintsurg-2019-015278.
7. Ahn JM, Oh JS, Yoon SM, Shim JH, Oh HJ, Bae HG. Procedure-related complications during endovascular treatment of intracranial saccular aneurysms. J Cerebrovasc Endovasc Neurosurg 2017;19:162-70. doi: 10.7461/jcen.2017.19.3.162.
8. Nomura M, Mori K, Tamase A, Kamide T, Seki S, Iida Y, et al. Thromboembolic complications during endovascular treatment of ruptured cerebral aneurysms. Interv Neuroradiol 2018;24:29-39. doi: 10.1177/1591019917739448.
9. Cronqvist M, Pierot L, Boulin A, Cognard C, Castaings L, Moret J. Local intraarterial fibrinolysis of thromboemboli occurring during endovascular treatment of intracerebral aneurysm: A comparison of anatomic results and clinical outcome. AJNR Am J Neuroradiol 1998;19:157-65.
10. Briganti F, Leone G, Marseglia M, Chiaramonte C, Solari D, Caranci F, et al. Mechanical thrombectomy in the treatment of distal occlusions during coil embolization of ruptured intracranial aneurysms. NMC Case Rep J 2016;3:115-7. doi: 10.2176/nmccrj.cr.2016-0022.
11. Yoon W, Jung MY, Jung SH, Park MS, Kim JT, Kang HK. Subarachnoid hemorrhage in a multimodal approach heavily weighted toward mechanical thrombectomy with solitaire stent in acute stroke. Stroke 2013;44:414-9. doi: 10.1161/STROKEAHA.112.675546.
12. Ahn JH, Jun HS, Song JH, Cho BM, Lee HK, Kim BC, et al. Rescue mechanical thrombectomy using a retrievable stent for thromboembolic occlusion occurring during coil embolization of ruptured intracranial aneurysms. J Neurointerv Surg 2017;9:244-9. doi: 10.1136/neurintsurg-2016-012457.
13. Topcuoglu M, Arsava EM, Özdemir A, Gurkas E, Dilek N, Ozturk S. Intravenous thrombolytic therapy in acute stroke: Problems and solutions. Turk J Neurol 2017;23:162-75.
14. Fatma A, Özlem A, Hayri D, Atilla Ö. Does having knowledge about collateral circulation affect patient selection in mechanical thrombectomy? Turk J Neurol 2021;27:322-6.