

Wake-Up Stroke Due to Middle Cerebral Artery Occlusion and the Decision Between Medical Management and Stenting: A Case Report

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Abstract

Intracranial arterial dissections (IAD) are rare but high-morbidity entities, especially in young adults. Optimal management remains controversial, ranging from conservative medical therapy to emergent endovascular intervention. A 27-year-old female presented with a "wake-up stroke" (National Institutes of Health Stroke Scale (NIHSS) 21). Initial angiography revealed a supraclinoid right internal carotid artery (ICA) dissection with 40% stenosis without hemodynamic compromise. Conservative management with dual antiplatelet therapy (DAPT) was initiated, given the patient's young age, leading to initial improvement (NIHSS 9), with a favorable clinical course; she was therefore discharged home five days after treatment. However, 48 hours after discharge, she suffered a neurological deterioration. Repeated angiography showed critical progression to 90% stenosis and M1 occlusion. An emergent rescue stenting with a LVIS EVO stent (4×21 mm) achieved total revascularization (Thrombolysis in Cerebral Infarction (TICI) 3). The patient experienced a remarkable recovery, with substantial improvement in motor strength (3/5 in the upper limb, 4/5 in the lower limb) and normalized speech. This case highlights the dynamic and potentially unstable nature of supraclinoid dissections. Although initial medical management is standard, close monitoring is vital, as rapid progression may require urgent endovascular reconstruction to prevent catastrophic outcomes.

Categories: Neurology, Radiology, Emergency Medicine

Keywords: internal carotid artery dissection, intracranial stenting, ischemic stroke, lvis evo, wake-up stroke, young adult

Introduction

Intracranial arterial dissections (IAD) are uncommon entities but carry high morbidity and mortality. Certain populations, including young adults and patients with underlying connective tissue disorders, may have a higher risk of developing IAD [1]. Compared with extracranial carotid and vertebral dissections, intracranial dissections are reported less frequently and remain less well characterized from an epidemiological standpoint. Extracranial dissections have a combined incidence of 3.6 to 4.4 per 100,000 persons per year [2], whereas epidemiological data regarding IAD remain limited, making the true incidence of this condition difficult to determine [3]. Although the clinical presentation of IAD is often nonspecific, the two most characteristic manifestations are subarachnoid hemorrhage and cerebral ischemia [1,2]. Approximately 50-60% of intracranial dissections present with subarachnoid hemorrhage, while 30-78% present with cerebral ischemia [4].

Ischemic events may result from progressive luminal stenosis, arterial occlusion, hemodynamic compromise, or thromboembolic phenomena arising from the dissected segment, highlighting the dynamic and potentially unstable nature of this disease process [5].

The optimal management of IAD remains controversial, as there is no evidence supporting a single treatment strategy. Pharmacological management with either anticoagulant or antiplatelet therapy has been described as an initial therapeutic approach [1]. However, treatment decisions are often individualized according to clinical presentation, radiological findings, and disease progression [6]. Additionally, endovascular therapy has been increasingly studied, particularly in non-hemorrhagic IAD, with the aim of restoring vessel patency, stabilizing the arterial wall, preventing recurrent ischemic events, and limiting progression of neurological deficits [7].

The present case illustrates the potentially unstable and rapidly evolving course of IAD. It involves a wake-up stroke (WUS), a subtype of ischemic stroke in which patients have no neurological deficits before sleep but awoken with new-onset neurological symptoms [2]. Despite appropriate initial medical management, the patient experienced progressive vascular deterioration with recurrent neurological compromise, ultimately

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requiring urgent endovascular intervention. This case highlights the importance of close clinical and imaging surveillance during the early stages of the disease in order to promptly identify neurological deterioration or radiological progression. Furthermore, it underscores the potential role of endovascular treatment in selected patients with evidence of failure of conservative management, where timely intervention may significantly modify the clinical course and improve functional outcomes [7].

Case Presentation

A 27-year-old female with no prior medical history presented to the emergency department at 09:15 AM following a "WUS" characterized by dysarthria, left central facial palsy, and severe left hemiparesis (National Institutes of Health Stroke Scale (NIHSS) 21). She reported a persistent holocranial headache over the preceding week. Initial neuroimaging (MRI) confirmed an evolving ischemic event in the right frontotemporal-parietal and insular territories (M1-M2 distribution) (Figures 1-3). Given the risk of hemorrhagic transformation, the patient was not a candidate for thrombolysis and was taken for urgent angiography.

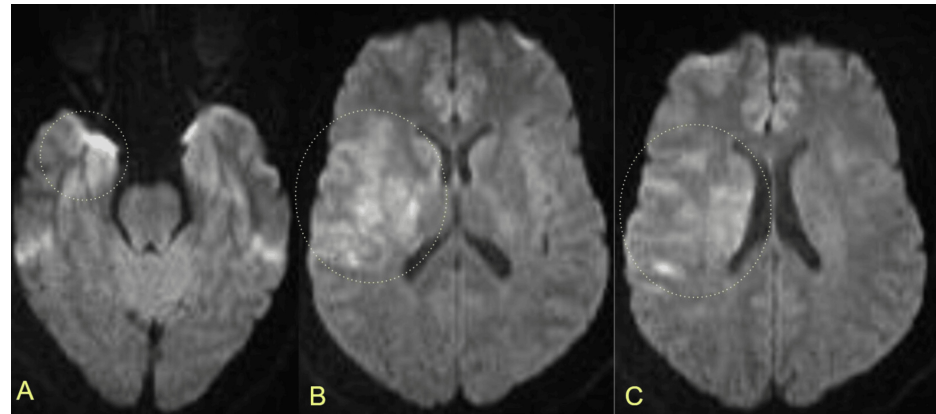


FIGURE 1: Diffusion-weighted MRI demonstrating acute right middle cerebral artery infarction

Axial diffusion-weighted imaging (DWI) at three levels. (A) Focal restriction in the right temporal cortex, consistent with early cortical infarction. (B) Marked restriction in the right insular cortex and basal ganglia, indicating an established infarct core. (C) Extension into the insular and fronto-parietal opercular regions.

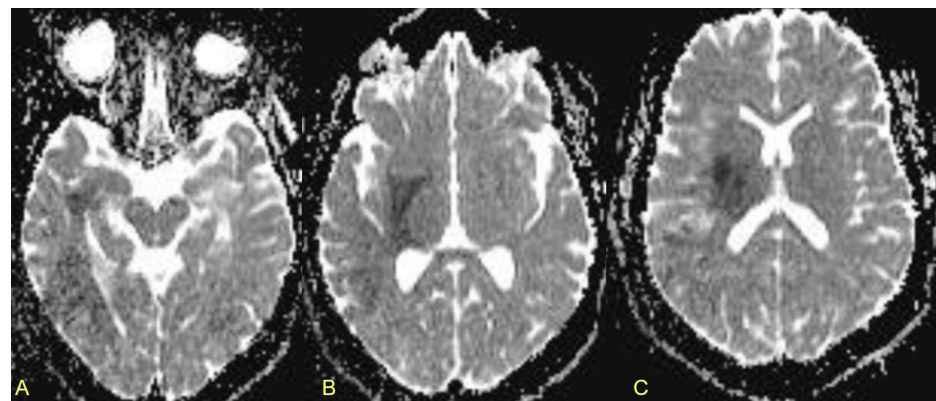


FIGURE 2: Apparent diffusion coefficient (ADC) mapping confirming true diffusion restriction in acute right MCA territory infarction

Axial apparent diffusion coefficient (ADC) maps at three levels (A–C). Hypointense signal in the right cerebral hemisphere (left side of the image) confirms true diffusion restriction. (A, B) Marked ADC reduction in the right insular cortex. (C) Persistent ADC hypointensity at the level of the basal ganglia.

MCA: Middle cerebral artery

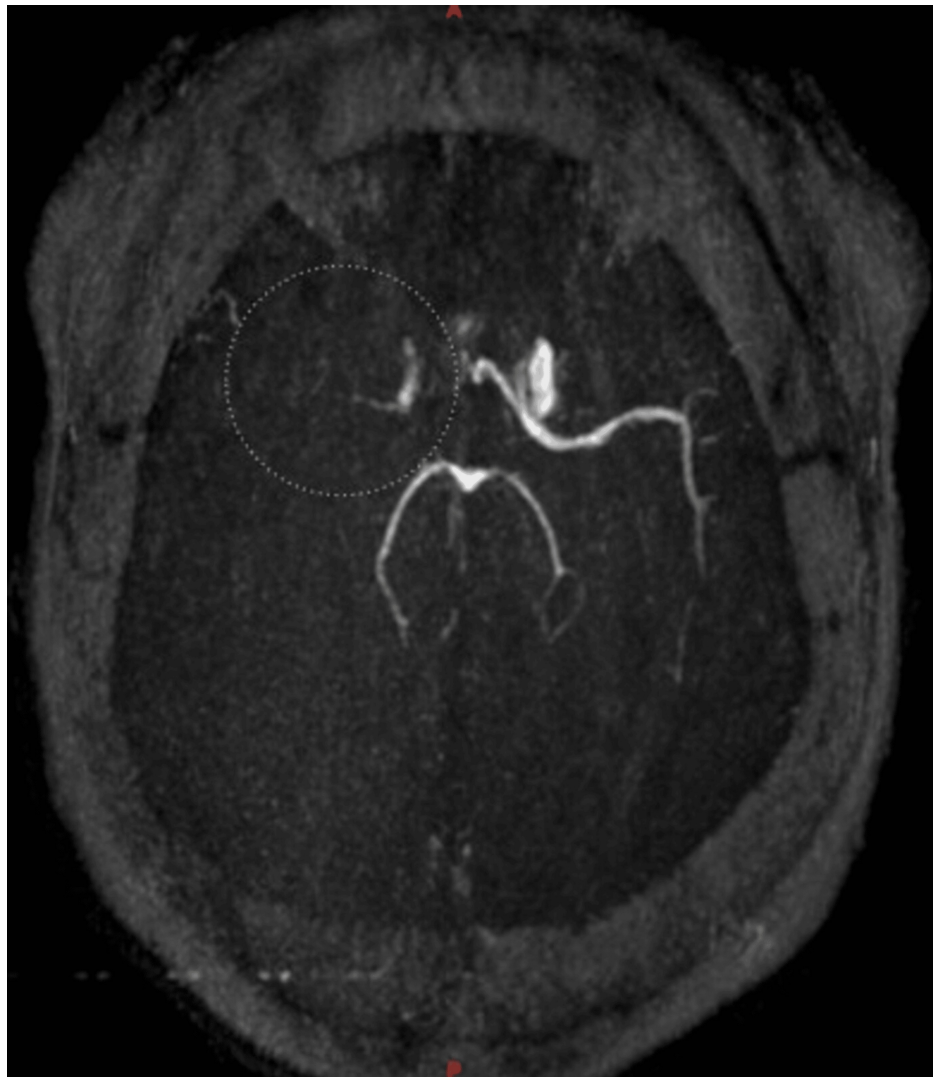


FIGURE 3: Time-of-flight MR angiography showing right internal carotid artery (ICA) occlusion

Axial TOF MR angiography shows the absence of flow-related signal in the right ICA (circled), consistent with occlusion or critical stenosis, with reduced distal opacification.

During the initial angiography, a 40% stenosis was identified in the supraclinoid segment of the right internal carotid artery (ICA) due to an arterial dissection (Figure 4). Because intracranial hemodynamics remained stable at that time, and because of her young age, a conservative strategy was adopted using dual antiplatelet therapy (DAPT) (clopidogrel and aspirin). The patient showed initial clinical improvement, reaching an NIHSS of 9, and was discharged five days later.



FIGURE 4: Digital subtraction angiography (DSA): reduced flow and 40% stenosis in the supraclinoid segment of the internal carotid artery (ICA)

(A) Projection demonstrating a focal defect in the supraclinoid segment of the ICA, with absent distal flow. (B) Lateral DSA projection confirming a supraclinoid defect corresponding to approximately 40% stenosis.

(1) Cervical ICA segment; (2) Petrous ICA segment; (3) Cavernous ICA segment.

The yellow arrow indicates decreased opacification at the supraclinoid segment. Red lines delineate areas of absent distal flow.

MCA: Middle cerebral artery; ACA: Anterior cerebral artery; OA: Ophthalmic artery; ICA: Internal carotid artery

However, 48 hours after discharge, the patient was readmitted with a seven-hour history of neurological deterioration and intensified hemiparesis. Repeat angiography revealed a critical progression of the supraclinoid dissection, now causing a 90% stenosis and marked intracranial hypoperfusion in the right middle cerebral artery (Figure 5). An emergent endovascular rescue was performed with the successful deployment of a self-expanding stent, achieving complete revascularization and a TICI (Thrombolysis in Cerebral Infarction) 3 flow (Figure 6).

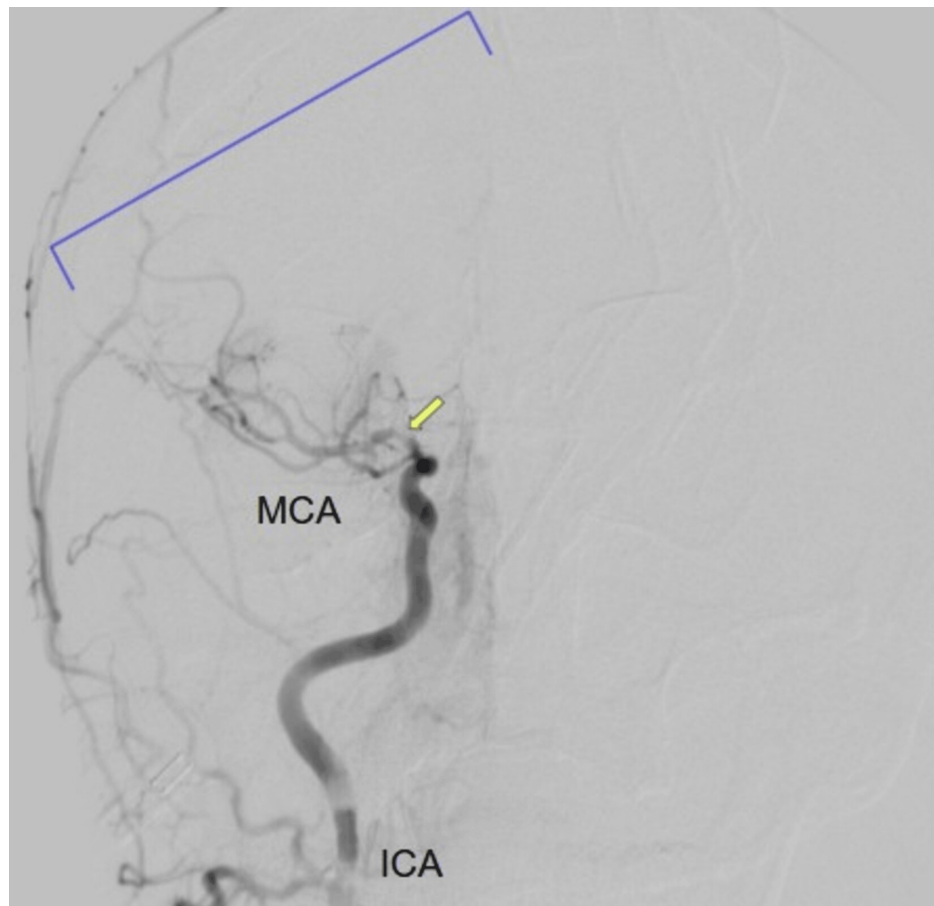


FIGURE 5: Digital subtraction angiography (DSA): reduced flow by 90% in the supraclinoid segment of the internal carotid artery (ICA)

Blue lines delineate the ischemic hemisphere.

ICA: Internal carotid artery; MCA: Middle cerebral artery

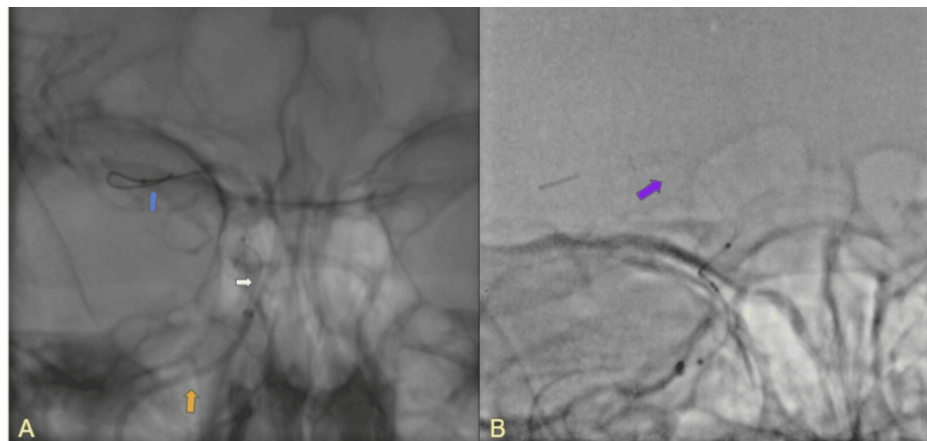


FIGURE 6: Digital subtraction angiography (DSA): device positioning for intracranial stent deployment

(A) The SOFIA catheter (orange) is positioned in the ICA, and the microcatheter (white) is advanced over a J-shaped microguidewire (blue) to the M1 segment. (B) The purple arrow indicates deployment of a 4 × 21 mm LVIS EVO stent, extending from the proximal M1 segment to the ophthalmic segment of the ICA.

ICA: Internal carotid artery

Following the intervention, the antiplatelet regimen was adjusted to ticagrelor and aspirin. The patient demonstrated a remarkable clinical recovery, with an NIHSS score of 3. At the time of her second discharge, five days after stenting, her speech had fully normalized, and her motor strength had improved significantly to 3/5 in the right upper limb and 4/5 in the right lower limb on the Medical Research Council (MRC) scale. This case highlights the high-risk nature of supraclinoid dissections and the necessity of rapid endovascular reconstruction when medical management fails to maintain hemodynamic stability.

Endovascular technique

First Intervention and Initial Management

The patient's initial evaluation was performed with color Doppler ultrasound. Right common femoral artery access was obtained using a 5 Fr micropuncture kit (MAK-NV™, Merit Medical Systems, Inc., South Jordan, UT), and a guidewire was advanced using the Seldinger technique. A 5 Fr femoral introducer sheath was then placed. A Simmons 2 diagnostic catheter (GLIDECATH®, Terumo, Tokyo, Japan) was advanced over a 0.035-inch × 150-cm hydrophilic guidewire. Selective catheterization of the supra-aortic vessels revealed a dissection in the supraclinoid segment of the right ICA. At this stage, the lesion caused approximately 40% stenosis without compromising intracranial blood flow (Figure 4).

The contralateral carotid axis and the vertebrobasilar system appeared normal, with no evidence of aneurysms or malformations. Given the hemodynamic stability observed, the medical team prioritized a cautious, patient-centered approach, initiating DAPT and close monitoring. The procedure was completed without neurological, vascular, or procedural complications.

Second Endovascular Intervention

Following a change in clinical status, a second intervention was urgently performed. Repeat angiography revealed a critical progression of the previously identified lesion: the supraclinoid dissection now caused a 90% stenosis, resulting in marked intracranial hypoperfusion (Figure 5).

To restore cerebral flow, we transitioned to an endovascular rescue strategy. Under general anesthesia, the right common femoral artery was accessed using a micropuncture set (Merit Medical Systems, Inc., South Jordan, UT). A 6 Fr femoral introducer sheath was advanced over a 0.035 × 150 cm hydrophilic guidewire (Terumo, Tokyo, Japan). A hemostatic valve connected to a continuous heparinized saline flush system was used. A 6 Fr chaperon guiding catheter (MicroVention, Terumo, Tokyo, Japan) was advanced, and selective catheterization of the supra-aortic vessels was performed with contrast injection, confirming a dissection of the right supraclinoid ICA segment.

For treatment, the chaperon catheter (MicroVention, Tokyo, Japan) was used as a support platform. A Neuroslider 21 microcatheter (Acandis GmbH, Pforzheim, Germany) was advanced over a Synchro microguidewire (Stryker, Kalamazoo, MI), both connected to a hemostatic valve system. Subsequently, a 5 Fr

SOFIA distal access catheter (MicroVention, Tustin, CA, USA; manufactured by Terumo Corporation, Tokyo, Japan) was navigated to the petrous segment of the ICA. The Neuroslider 21 microcatheter (Acandis, Pforzheim, Germany) was then advanced to the M1 segment of the middle cerebral artery (Figure 6A). An LVIS EVO stent (4x21 mm) (MicroVention, Terumo, Tokyo, Japan) was successfully deployed from the proximal M1 to the ophthalmic segment of the ICA (Figure 6B). A final angiographic check 10 minutes after deployment confirmed excellent stent patency without any signs of acute platelet aggregation (TICI 3; Figure 7).



FIGURE 7: Control digital subtraction angiography (DSA) after stent placement: restored caliber of the treated segment

Post-stenting DSA demonstrating the previously affected segment with adequate vessel caliber and restored flow. The yellow arrow indicates the site of the former stenosis.

Despite the technical complexity and a fluoroscopy time of 40.8 minutes, the procedure was successful and complication-free. Post-stenting control showed complete recanalization of the right ICA with a significant restoration of intracranial circulation (Figure 8). This case highlights how rapid endovascular intervention can effectively reverse the course of a symptomatic, high-grade supraclinoid dissection.



FIGURE 8: Follow-up computed tomography angiography (CTA)

A follow-up CTA showing the LVIS EVO stent correctly positioned across the previously dissected supraclinoid ICA, with restored luminal patency and no evidence of migration or acute complications.

ICA: Internal carotid artery

Discussion

The management of IAD remains one of the most debated frontiers in stroke neurology, primarily due to the lack of randomized controlled trials specifically addressing the supraclinoid segment [3]. As evidenced by the AHA/ASA (American Heart Association / American Stroke Association) 2019 guidelines, medical management often serves as the initial cornerstone for symptomatic dissections without immediate hemodynamic failure [4]. However, our patient's rapid progression from a 40% to a 90% stenosis and subsequent M1 occlusion demonstrates that "medical stability" in the setting of IAD can be a deceptive clinical state, especially in the supraclinoid ICA [7].

The initial therapeutic choice of DAPT with clopidogrel and aspirin aligns with the findings of the POINT and CHANCE trials, which established the superiority of DAPT in reducing early recurrent stroke risk for high-risk TIA [7]. DAPT was preferred over anticoagulation for three reasons. First, unlike extracranial cervical dissections, intracranial dissections carry a risk of subarachnoid extension and hemorrhage, making systemic anticoagulation relatively contraindicated [6]. Second, in the immediate post-thrombectomy setting with an established infarct, full anticoagulation would have increased the risk of hemorrhagic transformation of the reperfused territory [6]. Third, available randomized evidence (CADISS, TREAT-CAD), although derived from cervical artery dissection, showed no superiority of anticoagulation over antiplatelet therapy, and no trial data support anticoagulation for iatrogenic intracranial dissection [8,9]. Furthermore, the subsequent transition to ticagrelor and aspirin aligns with the THALES trial data, suggesting that more

potent antiplatelet inhibition may be necessary in high-risk patients to prevent secondary thrombotic events [10]. Yet, these pharmacological strategies primarily address the prevention of thrombus formation rather than the mechanical instability inherent to the dissected vessel wall, which, in this case, led to hemodynamic collapse [11].

From a diagnostic perspective, the management of this "WUS" was heavily influenced by the protocols established in the WAKE-UP and DAWN/DEFUSE 3 trials. By utilizing MRI-DWI/FLAIR mismatch, the clinical team identified salvageable brain tissue despite the unknown time of onset, a strategy that the European Stroke Organisation (ESO) and European Society of Minimally Invasive Neurological Therapy (ESMINT) have expedited as a standard of care [11]. This neuroimaging-guided approach allowed for the initial "watchful waiting" period, though it ultimately served to detect the catastrophic failure of conservative therapy during the second event when the patient presented with a new M1 occlusion [4].

The pivotal question remains: should primary stenting be favored over conservative management in supraclinoid IAD? While DeBette and Leys highlight the unique structural vulnerabilities of intracranial arteries, which lack the robust external elastic lamina found in extracranial vessels, the risk of stenting a fragile, dissected vessel is significant [1]. Nonetheless, the rapid progression observed in this young patient suggests that the "wait-and-see" approach may be insufficient for high-flow supraclinoid lesions where intramural hematoma expansion can lead to sudden occlusion despite optimal medical management [12].

When the patient was re-presented with 90% stenosis, the intervention shifted to a rescue strategy. Studies by Zhou et al. and Baek et al. have documented that emergency stenting is a highly effective salvage therapy when traditional mechanical thrombectomy fails or when there is an underlying high-grade stenotic lesion [12]. Technically, the use of a triaxial system was crucial; the 5 Fr SOFIA intermediate catheter provided distal support in the petrous segment, allowing the Neuroslider 21 microcatheter to safely cross the dissected segment into the M1 territory over a Synchro 0.014" microwire, minimizing the risk of entering a "false lumen" during navigation [13].

The decision to proceed with intracranial stenting in this patient was not without difficulty. In a young patient, stent placement commits the patient to prolonged, potentially lifelong antiplatelet therapy, with the attendant long-term bleeding risk and adherence considerations that this entails [14]. This trade-off weighed heavily against intervention. Ultimately, however, the decision was guided by the severity of the dissection and the demonstrated failure of medical management, together with a favorable anatomy that rendered the lesion technically accessible and suitable for reconstruction. In the setting of an evolving occlusion, where "time is brain," emergent stenting represented a viable rescue option, and prompt revascularization offered the greatest opportunity to preserve salvageable tissue and improve the functional prognosis [15].

The choice of the LVIS EVO stent (4x21 mm) was particularly relevant for this supraclinoid reconstruction. As a low-profile, braided nitinol stent, it offers a balance of high radial strength and increased metal coverage (up to 28%), which helps in redirecting flow away from the dissection flap and promoting vessel wall remodeling [16]. In the context of a dissection, this "flow-diverting" effect, as discussed by Papanagiotou and Haussen, may be superior to traditional laser-cut stents, as it addresses the structural narrowing while potentially preventing the formation of dissecting pseudoaneurysms [13].

Furthermore, the procedural success was defined not only by the deployment but also by the immediate hemodynamic response. The achievement of total recanalization (TICI 3 flow) and the 10-minute delayed angiographic control were vital to ensure the absence of acute platelet aggregation within the device, a known risk in the acute phase of stroke stenting [13]. This technical vigilance, combined with the transition to more potent anti-aggregation, reflects the complexity of balancing vessel patency with the risk of hemorrhagic transformation in a previously infarcted territory [16,17].

In conclusion, while medical management with DAPT remains the current standard, the dynamic nature of supraclinoid lesions demands high vigilance. This case contributes to the evidence suggesting that early endovascular reconstruction may be justified in young patients with high-grade intracranial dissections [13,15,16]. Ultimately, the successful resolution through stent-assisted revascularization highlights the need for adaptive protocols that can transition rapidly from pharmacological prevention to mechanical rescue, as the boundary between stability and hemodynamic collapse in the supraclinoid ICA is remarkably thin [17,18].

Conclusions

This case illustrates the dynamic nature of IAD as a cause of ischemic stroke in young patients, particularly in the setting of WUS. It shows that a non-flow-limiting dissection can progress rapidly to critical stenosis and occlusion, underscoring the importance of close clinical and imaging follow-up. In this patient, rescue stenting achieved complete recanalization with a favorable functional outcome.

As these observations derive from a single case, they cannot be generalized. Still, they suggest that endovascular treatment may be considered in selected high-risk patients when there are signs of instability

or failure of medical therapy. Further comparative studies are needed to define optimal management of IAD.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Supervision: Juliana Salcedo-Mesa, Oscar F. Vargas, Paula Valera

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. Comité de Ética en Investigación del Hospital Departamental de Villavicencio issued approval DSI-2026-013. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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