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*Case Report*

# Direct carotid stenting with minimal predilation after embolic protection device crossing failure in cervical internal carotid artery dissection: A case report

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## ABSTRACT

**Background:** Cervical internal carotid artery dissection (ICAD) may cause acute ischemic stroke requiring endovascular reperfusion. When repeated early reocclusion occurs and a distal embolic protection device (EPD) cannot cross the dissected segment after intravenous thrombolysis (IVT), the optimal strategy is uncertain.

**Case Description:** A 70-year-old man with prior chronic subdural hematoma presented with right hemiparesis and aphasia (National Institutes of Health Stroke Scale score 18). Magnetic resonance angiography showed left cervical ICAD without intracranial large-vessel occlusion. IVT with alteplase (0.6 mg/kg) was followed by aspiration thrombectomy, which achieved only transient recanalization with repeated early reocclusion of the internal carotid artery. Although the guidewire of a distal filter-type EPD crossed the dissection, the filter system could not be delivered. Under proximal balloon occlusion, we therefore performed minimal low-pressure predilation with small balloons and directly deployed a closed-cell carotid stent, obtaining good expansion and durable reperfusion (final modified Thrombolysis in Cerebral Infarction grade 3). Because emergent stenting was unavoidable, dual antiplatelet therapy (DAPT) was loaded intraoperatively but withheld thereafter until 24-h imaging confirmed stability of a small post-procedural subarachnoid hemorrhage. No in-stent restenosis, reocclusion, or new infarcts occurred during follow-up; the modified Rankin Scale score was 3 at 3 months.

**Conclusion:** This single-case experience suggests that, in cervical ICAD with repeated early reocclusion and distal EPD delivery failure, switching to proximal flow arrest, performing minimal low-pressure predilation, and proceeding to direct closed-cell carotid stenting can be a pragmatic bailout to secure sustained reperfusion. After prior IVT, a stepwise antiplatelet strategy – intra-procedural DAPT loading when emergent stenting is unavoidable, followed by withholding maintenance therapy until 24-h imaging confirms hemorrhagic stability – may help balance thrombotic and hemorrhagic risks, particularly in patients with a history of intracranial hemorrhage.

**Keywords:** Acute ischemic stroke, Carotid artery stenting, Direct stenting, Internal carotid artery dissection, Thrombectomy

## INTRODUCTION

Cervical internal carotid artery dissection (ICAD) is a major cause of ischemic stroke in young- and middle-aged adults, responsible for about 15–25% of ischemic strokes in patients younger than 50 years.[8,19,31] Population-based data historically estimated an annual incidence of 2–3/100,000, but more recent cohorts, aided by advanced vascular imaging, report 4.7–8.9/100,000 when all cervical artery dissections (carotid and vertebral) are considered.[16] ICAD arises from intimal tears and/or intramural hematoma, producing true-lumen stenosis or occlusion and a false lumen that can cause ischemic stroke through artery-to-artery embolism or hemodynamic compromise.[8,31] These epidemiologic and pathophysiological features underpin imaging strategies, antithrombotic choices, and acute reperfusion decisions in the emergency setting.

For diagnosis, characteristic findings include intramural hematoma on fat-suppressed T1-weighted magnetic resonance imaging (MRI) (crescent sign) and tapering stenosis, double lumen, or dissecting aneurysm on magnetic resonance angiography (MRA) or computed tomography angiography (CTA).[38] In routine practice, MRA combined with high-resolution cervical vessel wall imaging and/or CTA is widely used, and a systematic review suggests that MRI/MRA and CTA have comparable diagnostic performance for cervical artery dissection.[29] Combining "wall" imaging with "lumen" imaging is especially important for assessing patency of the false lumen and dynamic stenosis caused by an intimal flap. In particular, axial time-of-flight (TOF) MRA images suggesting an intimal flap can directly influence treatment strategy, including the feasibility of embolic protection devices (EPDs) and stent selection.[29,38]

For secondary prevention, the relative merits of antiplatelet therapy versus anticoagulation have long been debated. The cervical artery dissection in stroke study (CADISS) trial showed no significant difference between these strategies,[7] and the TREAT-CAD trial failed to demonstrate non-inferiority of aspirin.[12] A 2024 individual patient data meta-analysis combining these two randomized controlled trials also found no significant difference in 90-day composite outcomes and concluded that either approach is acceptable.[18] Consistent with this, the 2021 European Stroke Organisation (ESO) guideline recommends individualised antithrombotic choice based on patient characteristics rather than a single universally optimal regimen.[9]

In the acute phase, ICAD may present as a tandem lesion with cervical internal carotid artery (ICA) occlusion plus an intracranial large-artery occlusion, necessitating salvage carotid artery stenting (CAS) of the cervical lesion in addition to endovascular thrombectomy (EVT). Multicenter registries suggest that, even in patients who receive intravenous thrombolysis (IVT) before EVT plus CAS, this combined approach can improve recanalization rates and functional outcomes without a clear increase in symptomatic intracranial hemorrhage (sICH).[3,11,14] However, randomized evidence is scarce, and key questions remain, particularly regarding antithrombotic management and the timing of dual antiplatelet therapy (DAPT).

The use of an EPD is generally recommended during CAS to reduce distal embolization, yet in cases of severe stenosis or complex dissection, technical problems such as failure to cross the lesion or difficulty retrieving the device occur in roughly 10–20% of procedures.[13,20] The latest Cardiovascular and Interventional Radiological Society of Europe standards of practice (2024) support the use of EPDs but also outline alternative strategies – proximal balloon occlusion using a balloon guide catheter (BGC) or dedicated proximal protection device, reversed flow, or "direct stenting" after limited predilation – for anatomies in which distal protection is not feasible.[33] Thus, the practical scenario of "the EPD will not cross" is central to real-world decision-making about salvage CAS.

In the present case, a cervical ICAD with an intimal flap suspected on axial TOF-MRA showed only transient recanalization after thrombectomy, with repeated early reocclusions. When the filter component of a distal EPD could not be advanced across the lesion, we performed direct CAS with a closed-cell stent after minimal predilation under proximal protection, achieving stable, durable perfusion while tailoring the timing of DAPT initiation in the setting of a small post-procedural subarachnoid hemorrhage (SAH). We discuss the rationale and safety of direct CAS in this gray zone of EPD-crossing failure, as well as antithrombotic strategies after IVT, in the light of current evidence.

Of note, the patient had a history of surgically treated chronic subdural hematoma (CSDH). Prior intracranial hemorrhage (ICH) has traditionally been considered a relative contraindication for IVT; however, recent systematic reviews and observational studies suggest that IVT in patients with remote ICH does not necessarily confer a substantially higher risk of sICH, underscoring the importance of confirming stability of any residual hematoma, assessing coagulation status, and making individualized decisions.[10,15,39]

## CASE DESCRIPTION

### Patient

A 70-year-old man with a pre-stroke modified Rankin Scale (mRS) score of 1.

### Past medical history

He had hypertension, benign prostatic hyperplasia, and chronic kidney disease. He had undergone surgery for a left CSDH; the present stroke occurred 1 year and 10 months later, and follow-up at the previous hospital had been completed 1 year and 8 months earlier without recurrence.

### Risk factors and predisposing conditions

There was no history of neck manipulation, no family history suggestive of arterial dissection, and no use of antithrombotic agents. His history of head trauma was unclear.

### History of present illness and findings on arrival

On the morning of onset, before 8:30, he noticed weakness of the right upper and lower limbs and aphasia (exact onset unwitnessed). Emergency medical services were called at 8:30, and he arrived at our hospital at 9:06. On arrival, his level of consciousness was Japan Coma Scale (JCS) I-2. Vital signs were temperature 36.5°C, blood pressure 167/95 mmHg, heart rate 104 beats/min (sinus rhythm), respiratory rate 18 breaths/min, and oxygen saturation 98% on room air. Neurologically, he had right-sided hemiparesis and aphasia. After MRI at 9:40, his language function deteriorated to global aphasia. The National Institutes of Health Stroke Scale score on arrival was 18.

### Laboratory data on admission

Blood tests showed a platelet count of 24.4 × $10^4$/μL, a prothrombin time of 10.5 s, and an activated partial thromboplastin time of 22.4 s, without prolongation. D-dimer was 1.4 μg/mL. Serum creatinine was 1.93 mg/dL, consistent with chronic kidney disease. Serum potassium was mildly decreased at 3.3 mmol/L, and blood glucose was 164 mg/dL. Other parameters showed no marked abnormalities.

### Imaging findings

Non-contrast computed tomography (CT) on arrival demonstrated a thin residual left CSDH without any new hemorrhage [Figure 1]. MRI at 9:40 (diffusion-weighted imaging [DWI] with b = 1,000 s/mm$^2$) showed a DWI–Alberta stroke program early CT score of 10 with a small hyperintense lesion in the left parietal cortex [Figure 2, upper panel]. MRA revealed poor visualization of the left cervical ICA from just distal to the carotid bifurcation. Axial TOF-MRA suggested an intimal flap extending from the common carotid artery (CCA) to the ICA [Figure 2, lower panel]. The dissection extended from the upper margin of the C3 vertebral body (just above the carotid bifurcation) to the upper margin of C2, with an approximate length of 15 mm.

Acute treatment and antithrombotic timeline [Figure 3 and Table 1].

At 10:30, IVT with alteplase 0.6 mg/kg, the approved standard dose in Japan, was initiated. At 11:35, arterial puncture was performed. An 8-Fr long sheath and an 8 Fr BGC (BGC; Branchor XS 8 Fr × 90 cm) were advanced to the left CCA. Using a direct aspiration first pass technique (ADAPT) with a REACT 71 aspiration catheter, a Phenom

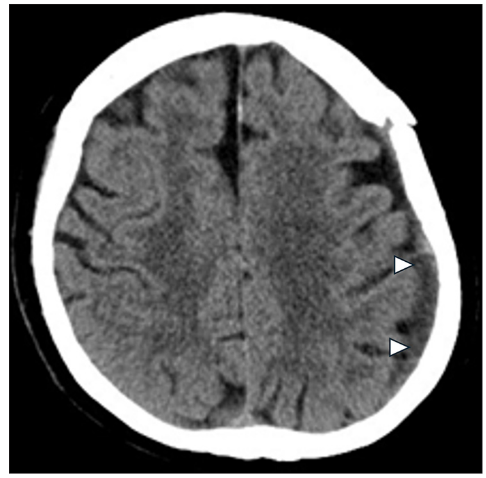

**Figure 1:** Axial noncontrast computed tomography (CT) on admission. Axial CT on admission showed a thin residual chronic subdural hematoma along the left frontoparietal convexity (arrowheads) without new intracranial hemorrhage or mass effect.

21 microcatheter, and a CHIKAI 14 200-cm microwire, we reached the proximal cervical lesion [Figure 3]. The first recanalization was achieved at 11:50, but elastic recoil led to reocclusion by 12:00. Given this early reocclusion with elastic recoil and the anticipated need for lesion stabilization by emergent CAS, we administered an intra-procedural DAPT loading dose of aspirin 300 mg plus clopidogrel 300 mg to prevent acute in-stent thrombosis. In line with guideline recommendations, maintenance antiplatelet therapy was withheld for the first 24 h after IVT, with a plan to restart after imaging confirmed the absence of worsening hemorrhage.[23,27,28] The time-stamped antithrombotic strategy and its rationale are summarized in Table 1.

The second recanalization was achieved at 12:30, but reocclusion recurred at 12:45. After the second early reocclusion with persistent elastic recoil, we judged that stent deployment to scaffold the flap would be required to prevent further reocclusion. Between 12:58 and 13:50, we sequentially attempted two distal filter-type EPDs (FilterWire EZ 190 cm and SpiderFX 6.0 × 320/190 mm). Although the guidewire could be advanced across the lesion, the filter portion could not be passed, and EPD use was abandoned. Given distal EPD delivery failure, we adopted proximal flow arrest with the BGC as the primary embolic protection strategy for the subsequent predilation and stenting steps. The third recanalization was obtained at 14:00. At 14:05, we inflated the BGC to achieve proximal flow arrest and performed the first predilation (SHIDEN 2.5 × 40-mm balloon, 6 atm).

Another elastic recoil occurred at 14:10, during which the BGC slipped back into the aortic arch and had to be repositioned. The fourth recanalization was achieved at 14:32. At 14:38, we performed a second predilation under proximal

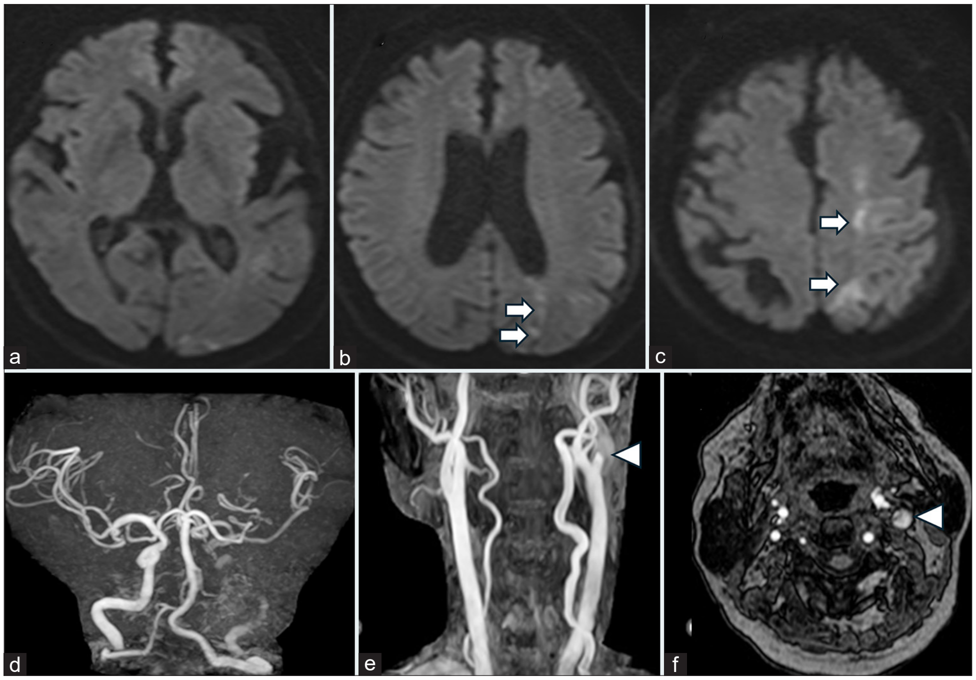


**Figure 2:** Magnetic resonance imaging and magnetic resonance angiography (MRA) on admission. (a-c) Axial diffusion-weighted images (b = 1,000 s/mm$^2$) showing a small hyperintense lesion in the left parietal cortex (b and c, arrow), with a diffusionweighted imaging – alberta stroke program early computed tomography score of 10. (d) Three-dimensional time-of-flight (TOF) MRA of the intracranial arteries showing no large-vessel occlusion. (e) Coronal TOF-MRA of the neck demonstrating poor visualization and severe stenosis of the left cervical internal carotid artery just beyond the carotid bifurcation (arrowheads). (f) Axial TOF-MRA at the level of the carotid bifurcation suggesting an intimal flap extending from the common carotid artery to the internal carotid artery (arrowheads), consistent with cervical internal carotid artery dissection.

**Table 1:** Time-stamped antithrombotic strategy and rationale.

| Time | Clinical/angiographic event | Antithrombotic action | Rationale |
|---|---|---|---|
| 09:06 | Arrival; CT showed thin residual CSDH without new hemorrhage [Figure 1] | No antithrombotics before imaging | Establish baseline intracranial bleeding status before reperfusion therapy |
| 10:30 | IVT started (alteplase 0.6 mg/kg) | Alteplase IV (0.6 mg/kg) | Bridging therapy; remote, stable CSDH and normal coagulation parameters |
| 11:35 | Arterial puncture; MT initiated | No systemic heparinization (ACT 101 s) | Avoid additive hemorrhagic risk after alteplase and prior CSDH |
| 12:00 | 1st early reocclusion with elastic recoil | Intra-procedural DAPT loading: aspirin 300 mg+clopidogrel 300 mg | High likelihood of emergent CAS; prevent acute in-stent thrombosis while planning to withhold maintenance for 24 h after IVT |
| 14:45 | Direct carotid stenting under proximal flow arrest | No additional antithrombotics; maintenance antiplatelets withheld | Avoid routine antithrombotics within 24 h after IVT; assess for procedure-related hemorrhage |
| Post-procedure | CT showed small sulcal SAH (Fisher group 1) [Figure 4] | Continue to withhold maintenance antiplatelets | Minimize risk of hemorrhagic expansion; strict blood pressure control |
| +24 h | Follow-up CT confirmed no hemorrhagic worsening [Figure 4b] | Start maintenance DAPT: Aspirin 100 mg/day+clopidogrel 75 mg/day | Balance stent patency and hemorrhagic risk; initiate after imaging confirmation per guidelines |

ACT: Activated clotting time, CAS: Carotid artery stenting, CSDH: Chronic subdural hematoma, CT: Computed tomography, DAPT: Dual antiplatelet therapy, IVT: Intravenous thrombolysis, MT: Mechanical thrombectomy, SAH: Subarachnoid hemorrhage

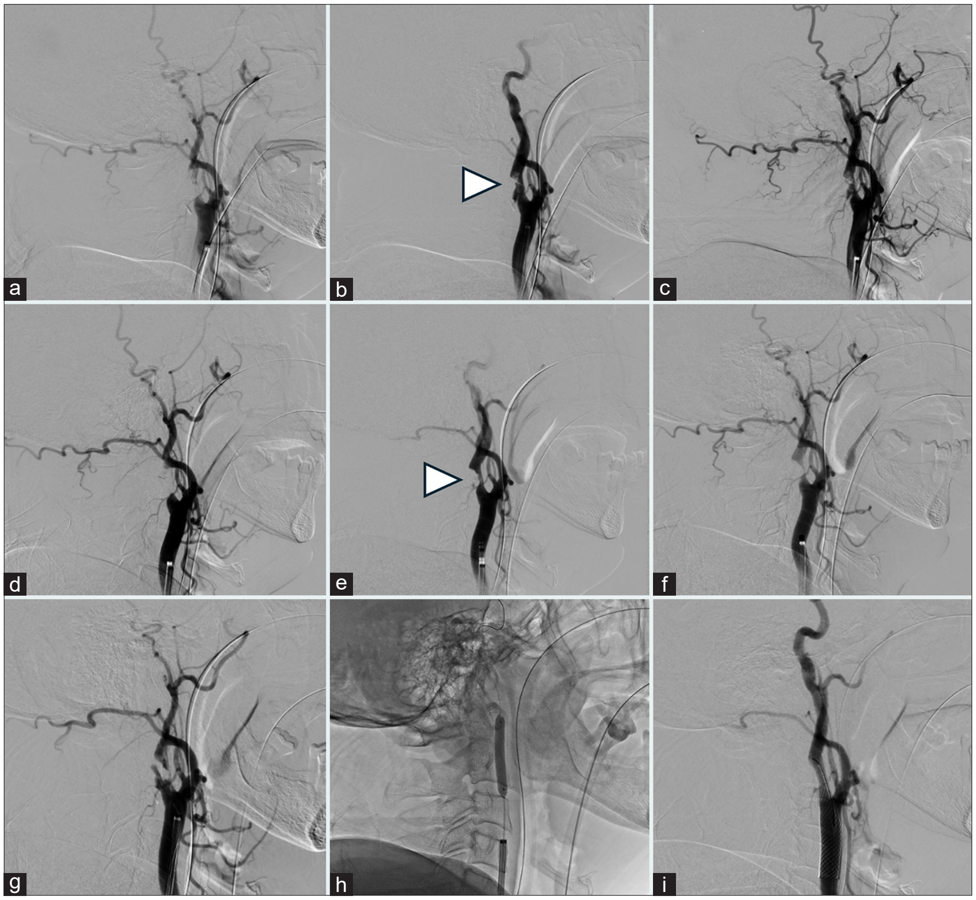


**Figure 3:** Angiographic course during mechanical thrombectomy and emergent carotid artery stenting for left cervical internal carotid artery dissection. (a-g) Sequential lateral digital subtraction angiograms from left common carotid artery injection showing a dissecting, tapering high-grade stenosis of the cervical internal carotid artery. Repeated aspiration thrombectomy, a direct aspiration first pass technique (ADAPT) temporarily reopened the true lumen with restoration of intracranial flow, but early reocclusions with loss of distal opacification recurred (examples in b and e, arrowheads), consistent with a mobile intimal flap. (h) Lateral fluoroscopic image during low-pressure balloon predilation of the dissected segment under proximal balloon occlusion with an 8-Fr balloon-guide catheter. (i) Final angiogram after direct implantation of a 10 × 31-mm closed-cell carotid stent, showing good stent expansion and smooth antegrade intracranial perfusion without embolization into new territory.

occlusion using a SHIDEN 4.0 × 40-mm balloon at 6 atm. At 14:45, with proximal occlusion maintained, a 10 × 31-mm Carotid Wallstent (closed-cell design) was deployed directly, achieving good expansion. Final angiography showed satisfactory dilation of the cervical ICA with good antegrade intracranial flow. Reperfusion of the anterior circulation was graded as modified thrombolysis in cerebral infarction 3, and the cervical ICA was completely patent (arterial occlusive lesion score 3), with no embolization into new territory (ENT). The total contrast volume was 150 mL, and the radiation dose was 1,157 mGy. The activated clotting time during the procedure was 101 s, and no intra-procedural heparin was administered. Systemic heparinization was withheld because of preceding IV alteplase and the history of CSDH, to minimize hemorrhagic risk.

## Peri-procedural management and complications

Post-procedural CT revealed a small SAH along the left convexity sulci, corresponding to Fisher group 1 [Figure 4]. Dual-energy CT was not used; however, follow-up CT on the next day showed persistence of the small sulcal SAH without any new hyperdense areas, making contrast extravasation unlikely. Blood pressure was strictly controlled at a systolic range of 110–140 mmHg for the first 3 days after the procedure, and subsequently maintained below 180 mmHg. Sedation consisted of pentazocine 30 mg and midazolam 2 mg, without continued postoperative sedation. As noted above, because repeated reocclusions made emergent CAS unavoidable, we loaded DAPT intraoperatively (aspirin 300 mg and clopidogrel 300 mg) to prevent acute stent thrombosis. Maintenance therapy was withheld for the first 24 h after IVT; after confirming on repeat imaging that hemorrhage had not worsened, we initiated aspirin 100 mg plus clopidogrel 75 mg.[23,27,28]

### Follow-up and clinical outcome

Digital subtraction angiography performed 1 week after the procedure showed no in-stent thrombus or restenosis [Figure 5a].

Carotid duplex ultrasound demonstrated preserved flow in the stented segment at both 1 week and 3 months. The peak systolic velocity (PSV) and end-diastolic velocity (EDV) were 71.3 and 23.6 cm/s, respectively, at 1 week (contralateral EDV 22.2 cm/s), and 81.0 and 13.7 cm/s at 3 months (contralateral EDV 13.6 cm/s); a representative ultrasound image at 3 months is shown in Figure 5b. Post-operative diffusion-weighted MRI on the following day showed scattered small ischemic lesions in the left middle cerebral artery territory without large territorial progression [Figure 6a and b].

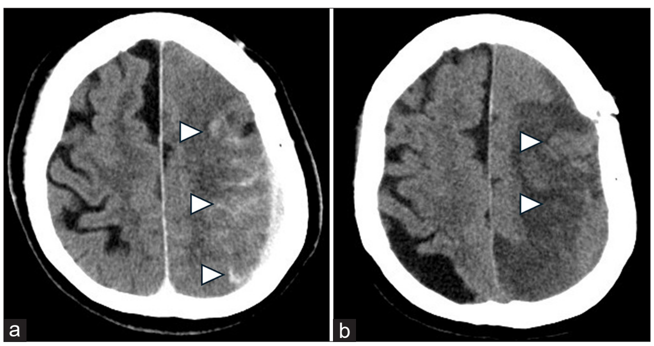

**Figure 4:** Post-procedural computed tomography (CT) showing a small, stable subarachnoid hemorrhage. (a) Axial non-contrast CT immediately after the procedure demonstrating a small subarachnoid hemorrhage along the left convexity sulci (arrowheads). (b) Follow-up CT on the next day showing persistence of the small hemorrhage without enlargement or any new intracranial hyperdense lesions (arrowheads).

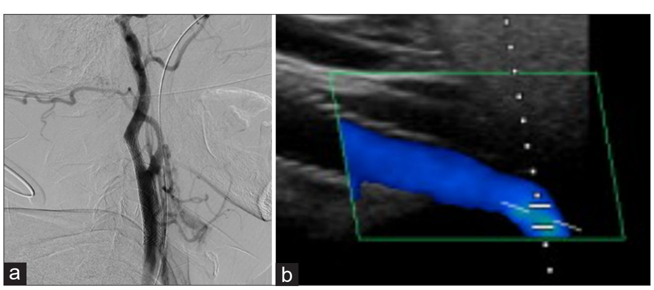

**Figure 5:** Follow-up angiography and carotid ultrasound. (a) Digital subtraction angiography at 1 week demonstrating a well-expanded carotid Wallstent without in-stent thrombus or restenosis. (b) Carotid duplex ultrasound at 3 months showing preserved laminar flow in the stented segment, with peak systolic and end-diastolic velocities within the normal range.

Follow-up DWI at 3 months [Figure 6c] demonstrated the chronic stage of these lesions without any new diffusion-restricted areas. At discharge (approximately 2.5 months after onset), his level of consciousness was JCS 2; he had residual right-sided weakness (manual muscle testing grade 2 in both upper and lower limbs) and expressive aphasia. He was able to take oral nutrition and walk with assistance using handrails. The mRS score at 3 months was 3.

### Key time metrics (minutes)

Because the exact onset time was unwitnessed, the last-known-well time was defined as before 8:30. Onset-to-door (OTD): 36 min (8:30 → 9:06). Door-to-imaging: 34 min (9:06 → 9:40). Door-to-needle: 84 min (9:06 → 10:30). Onset-to-needle: 120 min (8:30 → 10:30). Door-to-puncture: 149 min (9:06 → 11:35). Puncture-to-reperfusion: 190 min (11:35 → 14:45). Onset-to-reperfusion: 375 min (approximately 6 h 15 min).

## DISCUSSION

### Pathophysiologic context and significance of this case

Although the present case involved an isolated cervical ICA lesion rather than a true tandem lesion, much of the available evidence on acute CAS and DAPT timing comes from registries and subgroup analyses of tandem occlusions, which are therefore referenced below. Acute reperfusion in carotid artery dissection, including tandem presentations, is particularly challenging: dynamic stenosis due to a dissection flap and/or a patent false lumen can promote recurrent occlusion and distal embolization, and technical difficulties with EPD passage are frequent. In this case, IVT was administered first, followed by mechanical thrombectomy (MT). Once EPD passage failed, we proceeded promptly to direct CAS after reopening the artery, to cover and stabilise the false-lumen entry. This approach achieved durable reperfusion and an acceptable clinical outcome. In multicenter registries and pooled analyses, such as thrombectomy in TANdem occlusions registry and the Endovascular Treatment in Ischemic Stroke registry, acute CAS in tandem occlusions has been associated with higher recanalization rates and better functional outcomes overall,[21,37] although a dedicated subanalysis of dissection-related tandem occlusions found no clear association between day-1 carotid patency and clinical outcome.[26]

### Strategy when an EPD cannot be advanced: how to maintain "protection"

Embolic protection is a fundamental principle in CAS, yet in clinical practice a distal filter may fail to cross a severely stenotic, highly tortuous, or dissected segment with a

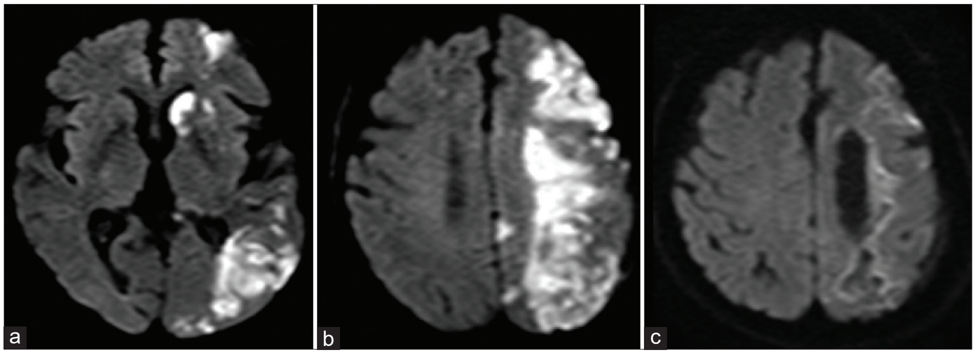

**Figure 6:** Postoperative diffusion-weighted magnetic resonance imaging. (a and b) diffusionweighted imaging (DWI) on the day after the procedure showing multiple small hyperintense ischemic lesions in the left middle cerebral artery territory without large territorial infarction. (c) DWI at 3 months demonstrating the chronic stage of these lesions without any new diffusion-restricted lesions.

prominent flap. Classic reports have highlighted technical limitations of EPDs – device profile, torque transmission, and retrieval difficulties – and more recent series have described EPD-related complications and bailout techniques.[13,20] Registry data indicate that unprotected CAS is associated with approximately a four-fold increase in perioperative stroke risk,[24] and should therefore be avoided whenever possible. When a distal filter cannot be used, alternatives include (1) switching to proximal protection (using a BGC or a device such as the Mo.Ma), (2) facilitating filter passage with minimal predilation using a small balloon, and (3) performing direct stenting under proximal flow arrest. The randomized PROFI trial showed that proximal balloon occlusion significantly reduced cerebral microembolization compared with distal filter protection,[5,22,32] and BGC use has been associated with fewer distal emboli during stroke thrombectomy.[32] In our patient, because EPD passage failed, we chose proximal flow arrest with low-pressure, short-duration predilation followed by rapid stent deployment. In retrospect, earlier conversion to direct CAS after the second early reocclusion might have shortened the overall procedure, but we initially prioritised attempts to obtain distal protection, given the well-established association between unprotected CAS and higher perioperative stroke risk. Ultimately, this approach stabilised the lesion without provoking additional distal emboli and is consistent with the principle of "never giving up protection," instead using procedural planning to mitigate risk.[2,33]

### Choice of stent device: Providing both "scaffolding" and a "sieve" for dissection

In dissecting lesions, adequate scaffolding is required to cover the flap and occlude the false lumen, making closed-cell or fine micromesh (double-layer) stents a rational choice. Observational studies and analyses of filter-captured debris and new DWI lesions suggest that stent design influences microembolic burden, with open-cell stents associated with more new DWI lesions.[25,30] Double-layer micromesh stents such as CASPER and ROADSAVER have been reported to reduce plaque protrusion and new ischemic lesions,[17,30] and are increasingly used in contemporary CAS, including acute settings.[33] Conversely, excessive post-dilation may increase microembolization. Experimental and clinical data support using only the minimal balloon size, inflation pressure, and number of inflations required to achieve sufficient scaffolding, while ensuring that the stent is long enough to cover the entire flap from its origin.[33,34]

### Acute CAS after recombinant tissue-type plasminogen activator (rt-PA): Revisiting safety and antithrombotic therapy

Bridging therapy with IVT followed by EVT is generally considered safe even in tandem lesions and has been linked to improved recanalization and functional outcomes without clear increases in hemorrhagic complications.[3,13,38] The optimal timing of antiplatelet initiation in this context remains controversial. The American Heart Association/American Stroke Association (AHA/ASA) guideline recommends avoiding antithrombotic agents during the first 24 h after IVT and starting them only after follow-up imaging has excluded hemorrhage (Class I), while acknowledging that carefully selected exceptions may be reasonable in patients requiring emergent stent placement (Class IIb).[23,28] In observational studies of emergent CAS for tandem occlusions, DAPT started after 24 h and continued for 4–12 weeks is commonly used.[6,27] In our case, the combination of EPD-crossing failure and repeated reocclusion made emergent CAS unavoidable. We therefore used a stepwise approach: Intra-procedural DAPT loading only, avoidance of maintenance antiplatelets for the first 24 h after IVT, and re-initiation of DAPT once imaging confirmed hemorrhagic stability. This strategy is consistent with the principles outlined in the 2019 AHA/ASA guideline and the 2023 European Society for Vascular Surgery guideline, as well as real-world data on DAPT timing after emergent CAS.[23,27,28]

### Appropriateness of IV rt-PA in a patient with prior CSDH

A history of CSDH surgery poses a particular challenge when assessing IVT safety. Historically, AHA/ASA guidelines treated "prior intracranial hemorrhage" as a potentially harmful condition (Class III: Harm), whereas a 2015 revision of the United States Food and Drug Administration label for alteplase removed prior ICH from the list of absolute contraindications, allowing more selective use in practice.[28,36] The 2021 ESO guideline on IVT likewise acknowledges the limited evidence base but conditionally permits IVT in carefully selected patients with remote ICH.[4] An accumulating body of observational studies and meta-analyses suggests that a history of ICH does not clearly increase sICH risk after IVT.[10,15,39] In the present case, we judged IVT to be acceptable because the residual CSDH was thin and stable, coagulation parameters were normal, and blood pressure could be strictly controlled. After confirming no radiologic worsening at 24 h, we transitioned to maintenance antiplatelet therapy, which we consider reasonable in light of current evidence.

### Prevention of distal embolization (ENT) and microembolization

Intra-procedural embolization into new territory (ENT) during MT or CAS can adversely impact clinical outcome. The introduction of proximal flow arrest and aspiration techniques such as ADAPT has been associated with reductions in ENT.[2,22,32] This is particularly important when an EPD cannot be used, and meticulous proximal flow control becomes the main tool for limiting distal emboli.[25,30]

### Interpretation and management of hemorrhagic complications

In our patient, a small SAH was detected early after the procedure but remained stable under strict blood pressure management and temporary withholding of antithrombotic therapy. Guidelines recommend maintaining blood pressure below 180/105 mmHg during the first 24 h after IVT,[28] and in the setting of acute CAS, it is reasonable to employ even stricter control, as in this case.[23]

### Prevention and monitoring of reocclusion and restenosis

Day-1 stent patency has been reported to correlate with clinical outcome, and acute stent occlusion can occur in roughly 15–20% of cases, underscoring the importance of peri-procedural antiplatelet therapy and blood pressure control.[27] In our patient, carotid duplex ultrasound showed PSV values of 71–81 cm/s within the stent, consistent with the absence of significant restenosis. Commonly used thresholds for in-stent restenosis are a PSV ≳240 cm/s for ≥50–60% stenosis and ≳300–450 cm/s for ≥70% stenosis, although centers must calibrate their own criteria and should combine PSV with the ICA/CCA ratio and EDV.[1,35] Based on current recommendations, ultrasound surveillance at 1, 6, and 12 months after CAS is appropriate.[1,35]

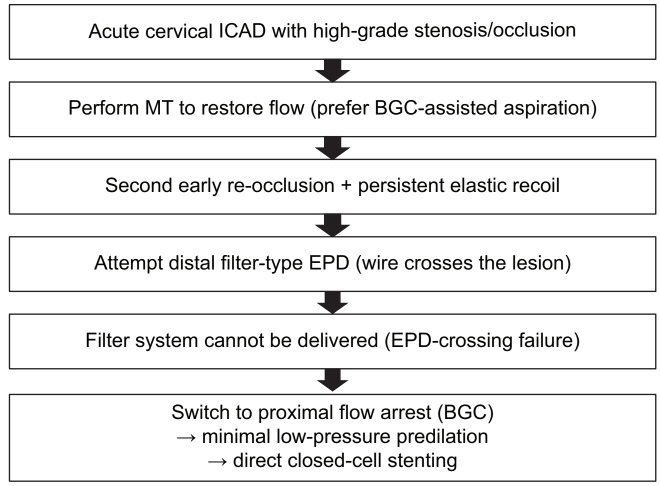


**Figure 7:** Bailout algorithm for cervical internal carotid artery dissection with distal embolic protection device delivery failure. The pathway highlights a pragmatic sequence—after a second early reocclusion with persistent elastic recoil, attempt distal filter protection; if the filter system cannot be delivered despite wire crossing, convert to proximal flow arrest, perform minimal low-pressure predilation, and proceed to direct closed-cell stenting. ICAD: Internal carotid artery dissection, MT: Mechanical thrombectomy, EPD: Embolic protection device, BGC: Balloon guide catheter.

### A pragmatic algorithm based on this case

Figure 7 summarizes a simple bailout algorithm derived from this case for acute cervical ICAD when distal EPD delivery fails. In brief, after MT to restore flow, a second early reocclusion with persistent elastic recoil suggests that emergent stenting may be required to stabilize a mobile dissection flap. If a distal filter cannot be delivered despite wire crossing, conversion to proximal flow arrest (BGC), minimal low-pressure predilation only as needed, and direct deployment of a closed-cell (or micromesh) stent long enough to cover the dissected segment may help maintain protection and achieve durable patency.[5,13,20,22,24,33] After prior IVT, maintenance antiplatelets should ideally be withheld for the first 24 h and started after follow-up imaging confirms hemorrhagic stability, although intra-procedural loading can be considered when emergent stenting is unavoidable.[23,27,28]

### Availability of data and materials

All data generated or analyzed during this case report are included in this published article.

## CONCLUSION

In this single-case report of cervical ICA occlusion due to ICAD, repeated early reocclusion after MT and failure to deliver a distal filter-type EPD prompted a bailout strategy of proximal flow arrest, minimal low-pressure predilation, and direct deployment of a closed-cell carotid stent, achieving sustained reperfusion without apparent excess distal embolization. A small post-procedural sulcal SAH remained stable under strict blood pressure control, allowing DAPT maintenance to be initiated after 24-h imaging confirmed no deterioration. No restenosis, reocclusion, or new infarcts were observed over 3 months. While this experience should not be generalized, it provides a practical technical lesson for maintaining embolic protection when distal EPD delivery is not feasible.

**Authors' contributions:** TN: Was the on-call stroke physician and primary operator for mechanical thrombectomy and CAS: He led the acute management, collected the clinical and imaging data, prepared the figures, and drafted the manuscript. YA and KG: Assisted with the endovascular procedures and acute clinical management and contributed to imaging interpretation and data collection. KM: Contributed to the clinical management and follow-up, participated in data collection and literature review, and helped refine the Introduction and Discussion. NK: Conceived the report, supervised the overall clinical decision-making and management strategy, co-drafted the manuscript with TN, and contributed to figure preparation, and provided critical revision and final approval of the manuscript. All authors read and approved the final manuscript.

**Ethics approval:** The Institutional Review Board approval is not required. The case was managed in accordance with the principles of the Declaration of Helsinki.

**Declaration of patient consent:** The authors certify that they have obtained all appropriate patient consent forms. In the form, the patients have given their consent for their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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