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Case Report

Successful Leptomeningeal Enhancement in a Patient with Tandem Occlusion of a Carotid and Middle Cerebral Artery Following Carotid Artery Stenting for Contralateral Carotid Artery Stenosis

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Objective: Although the presence of leptomeningeal anastomosis is known as a predictor of favorable outcome in patients with acute large vessel occlusion, the efficacy of enhancing leptomeningeal collateral flow has rarely been demonstrated.

Case Presentation: A 73-year-old man previously diagnosed with asymptomatic bilateral carotid stenosis was admitted to our emergency department 2 hours after the onset of fluctuating symptoms, including aphasia, left conjugate deviation, and right hemiparesis. CT demonstrated no hemorrhagic lesion. Considering the history of the patient, emergent angiography was performed and demonstrated tandem occlusion of the left cervical internal carotid artery (ICA) with left common carotid injection, leptomeningeal flow compensating for distal territory of occluded segment of left middle cerebral artery (MCA) via the left anterior cerebral artery through severe cervical ICA stenosis with right common carotid injection, and the proximal segment of the left MCA through the posterior communicating artery and occlusion of the M2 segment with left vertebral injection. Given the results of angiography and fluctuating symptoms, hemodynamic insufficiency was considered the underlying stroke mechanism for this case. Although recanalization of tandem lesions was initially considered, the risk of distal clot migration was a concern, so the patient underwent right carotid artery stenting (CAS) to enhance leptomeningeal collateral flow. This resulted in immediate resolution of symptoms after right CAS.

Conclusion: Stenting for carotid artery stenosis contralateral to tandem occlusive lesion may offer an effective alternative when both Willisian and leptomeningeal collaterals are robust.

Keywords leptomeningeal enhancement, emergent carotid artery stenting, contralateral carotid artery occlusion, contralateral middle cerebral artery occlusion

Introduction

Leptomeningeal anastomoses are widely recognized as an important factor contributing to the severity of acute ischemic stroke.[1–4] Since leptomeningeal collaterals seem to be therapeutic targets, many clinical studies have examined augmentation of collateral blood flow using pharmacological approaches via vasodilation, volume expansion, hemodilution[5] and induction of hypertension,[6] or non-pharmacological approaches such as partial aortic occlusion,[7] external counter pulsation,[8] or sphenopalatine ganglion stimulation.[9] Unfortunately, none of those approaches demonstrated efficacy in enhancing leptomeningeal collaterals. However, this could have been due to the inclusion of patients who were unlikely to prove responsive to the treatment. Here, we report the case of a patient with tandem occlusion of the carotid and middle cerebral arteries who was treated with leptomeningeal enhancement by emergent carotid artery stenting (CAS) for contralateral carotid artery stenosis.

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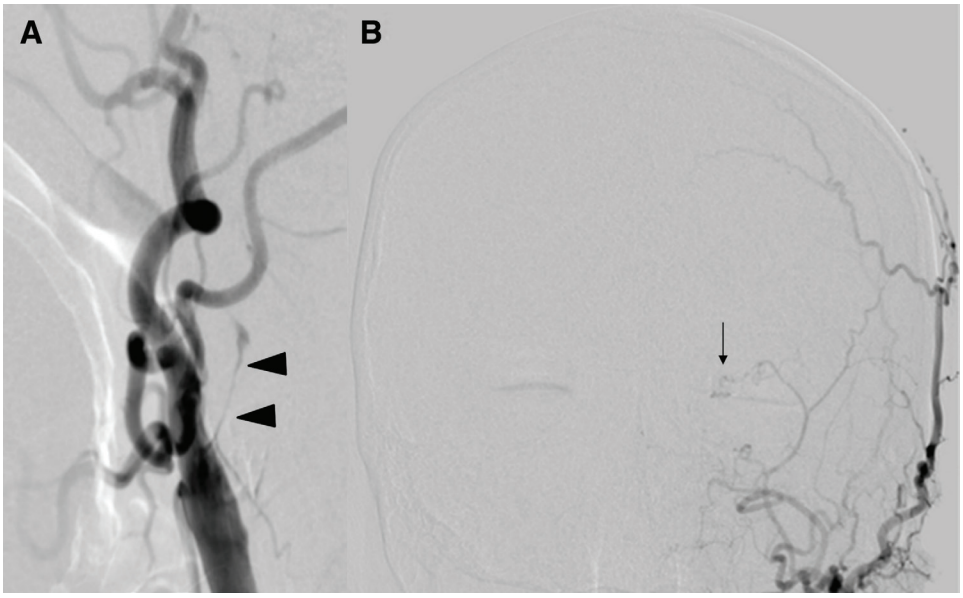

Fig. 1 Left common carotid injection (lateral view) demonstrates pseudo-occlusion of the cervical ICA (arrowheads) (**A**). Left common carotid injection (anterior view) demonstrates faint filling of the C3 segment of the ICA via anastomosis between a middle meningeal artery and an ophthalmic artery (arrow) without antegrade flow in the ICA (**B**). ICA: internal carotid artery

Case Presentation

A 63-year-old man diagnosed with asymptomatic bilateral carotid stenosis, hypertension, and diabetes at another hospital was admitted to our emergency department 2 hours after the onset of mild aphasia and right upper limb weakness with a National Institutes of Health Stroke Scale (NIHSS) score of 2. Stroke code was initiated, and CT of the brain showed negative results for both large vessel occlusion and hemorrhagic lesions with an Alberta Stroke Program Early CT score (ASPECTs) of 10. However, the patient showed neurological deterioration immediately after CT, including total aphasia, left conjugate deviation, unilateral spatial neglect, and right hemiplegia with an NIHSS score of 24, and symptom severity continued to fluctuate dynamically with NIHSS scores ranging from 2 to 24. Considering the history of bilateral carotid stenosis and the need for angioplasty on antiplatelet agents, intravenous thrombolytic therapy was reserved, and emergent angiography was performed to evaluate the dynamics of cerebral blood flow. Left common carotid injection demonstrated pseudo-occlusion of the cervical internal carotid artery (ICA) without functional filling of the intracranial ICA (**Fig. 1A** and **1B**). Right common carotid injection revealed severe stenosis of the cervical ICA (**Fig. 2A**), and leptomeningeal collateral compensation from the left anterior cerebral artery (ACA) to the distal middle cerebral artery (MCA) via the anterior communicating artery (**Fig. 2B** and **2C**). Left vertebral injection demonstrated the proximal segment of the left MCA through the posterior communicating artery and occlusion of the M2 segment of the MCA supplying the central artery or anterior or posterior parietal artery (**Fig. 2D** and **2E**). Due to fluctuations in symptoms, hemodynamic insufficiency through leptomeningeal collaterals from the left ACA to the MCA was considered one pathological mechanism in the present case. Recanalization of the left tandem lesion was initially considered. However, since the distal extent of thrombus from the cervical ICA to the intracranial ICA was unknown, the risk of distal thromboembolism during angioplasty or stenting for carotid artery stenosis seemed overly high even with the use of a distal protection device. The decision was therefore made to attempt CAS of the stenosed right cervical ICA to supplement blood flow to the left distal MCA territory through leptomeningeal collaterals from the ACA. If symptoms had continued to fluctuate or deteriorate even after right CAS, endovascular reconstruction of the left tandem lesions was considered as a second-line treatment. Since the patient was on chronic therapy with clopidogrel at 75 mg/day, aspirin alone was administered through a gastric tube at a loading dose of 200 mg, followed by emergent CAS for the right cervical ICA stenosis. A 9-Fr balloon guiding catheter was advanced into the right distal common carotid artery (CCA) after systemic heparinization. Next, a distal embolic protection

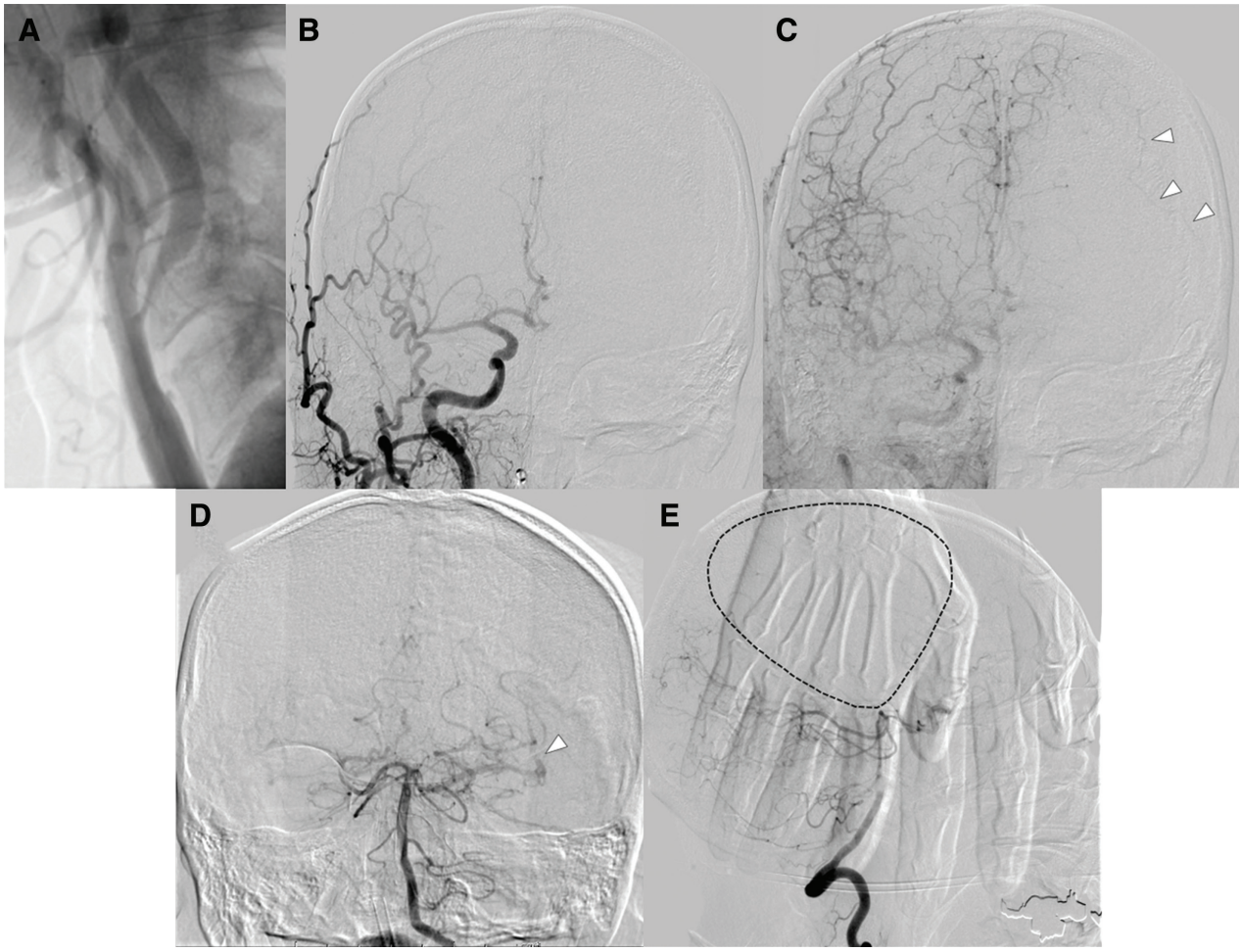

Fig. 2 Right common carotid injection (lateral view) showing severe stenosis of the cervical ICA (**A**), and demonstrating patency of the contralateral ACA through the anterior communicating artery in the early phase (**B**) and flow compensation via leptomeningeal anastomoses between distal ACAs to distal middle cerebral arteries in the late phase (**C**, arrowheads). Left vertebral injection demonstrating patency of the M1 segment of the left MCA via the posterior communicating artery (arrowhead) (**D**), and occlusion of the M2 segment of the MCA supplying the frontoparietal cortex of the left hemisphere (encircled by black dotted line) (**E**). ACA: anterior cerebral artery; ICA: internal carotid artery; MCA: middle cerebral artery

device (SpiderFX; Covidien, Dublin, Ireland) was navigated over a wire through a guide catheter, and a closed-cell stent (Carotid Wallstent; Stryker, Kalamazoo, MI, USA) was deployed to cover the stenotic cervical ICA (**Fig. 3A**), followed by postdilation with a 5 mm × 30 mm non-compliant balloon. Right carotid injection immediately after CAS showed robust leptomeningeal flow compensation from the left ACAs to the left distal MCAs, and antegrade flow in the left MCA (**Fig. 3B**). All symptoms resolved within about several minutes after completing treatment. Diffusion-weighted MRI 1 day after treatment demonstrated no ischemic lesion in the left frontal cortex, which was considered to represent a terminal territory supplied by leptomeningeal flow from the left ACAs without any ischemic lesions in the right hemisphere, but revealed spotty high-intensity lesions in the left insular and temporal cortex, suggesting that thrombus fragment had migrated into the inferior division of the MCA (**Fig. 4A**). MRA demonstrated recanalization of the occluded M2 branches (**Fig. 4B**). The patient was discharged home 6 days after treatment, with an NIHSS score of 0.

Ethical approval

All procedures in studies involving human participants were performed in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards.

Informed consent

Informed consent was obtained from the patient.

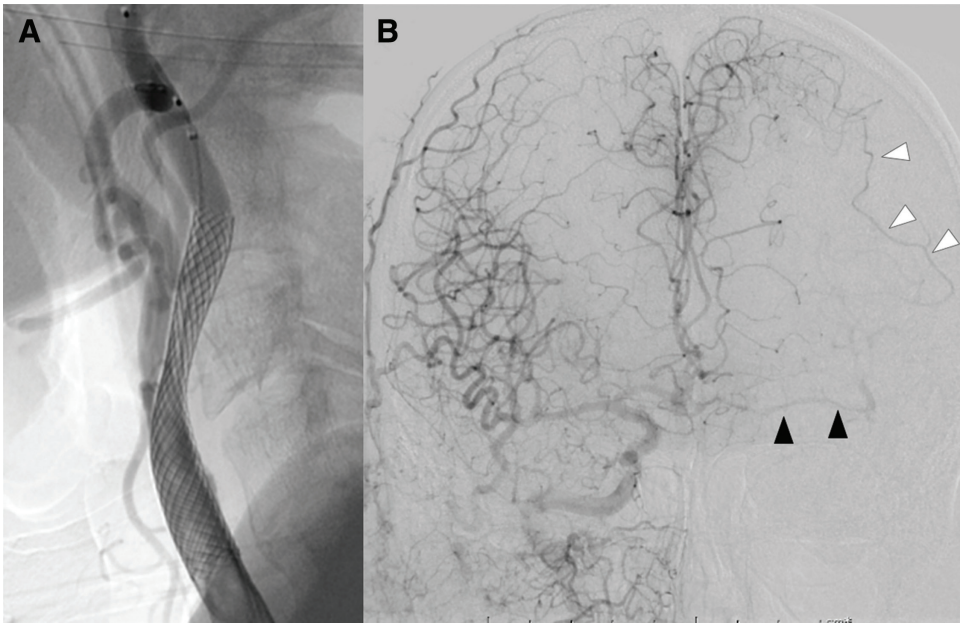

Fig. 3 Postoperative right common carotid injection shows resolution of the cervical ICA stenosis (**A**), and increased patency of the cortical arteries via leptomeningeal collaterals (white arrowheads) and the M1 segment via Willisian collaterals (black arrowheads) (**B**). ICA: internal carotid artery

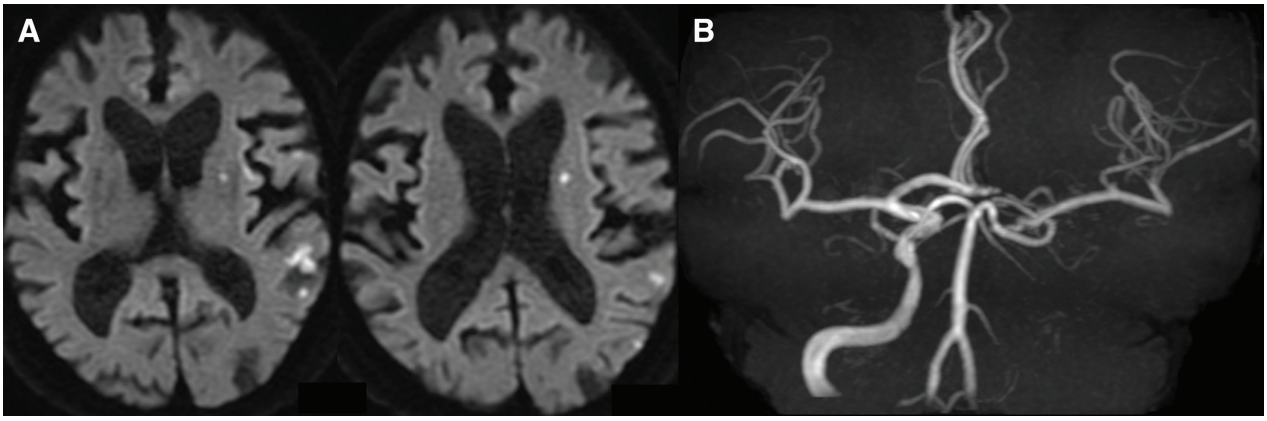

Fig. 4 Diffusion-weighted MRI on postoperative day 1 reveals only spotty high-intensity lesions in the left insular and temporal lobes, with no infarction in the right hemisphere (**A**), and MRA demonstrates recanalization of the distal segment of the left MCA (**B**). MCA: middle cerebral artery

Discussion

Although a rare occurrence, we encountered a case of right cervical carotid artery stenosis concurrent with tandem occlusion of the left cervical ICA and the M2 segment of the left MCA. Several endovascular management strategies for tandem lesions of the anterior circulation have been trialed without reaching any clear consensus.[10,11] In the setting of carotid pseudo-occlusion, crossing the carotid lesion with an aspiration catheter is sometimes challenging, and the distal extent of the thrombus in the carotid artery is uncertain. Since the distal extent of thrombus from the cervical ICA to the intracranial ICA was unknown, appropriate positioning of distal protection devices is unclear in such settings. Therefore, prevention of distal thromboembolism may not be guaranteed after crossing the lesion by angioplasty or stenting. In the present case, the right common carotid injection showed patency of the anterior communicating artery via severe stenosis of the cervical ICA and leptomeningeal collateral flow from the left distal ACA to the occluded M2 segment. Moreover, patient symptoms such as aphasia and motor weakness showed fluctuation, suggesting hemodynamic ischemia. Considering the risk of thromboembolic complications

during recanalization of the left tandem lesions, leptomeningeal enhancement by stenting for right carotid artery stenosis was first attempted, successfully resolving the fluctuating neurological symptoms without complications.

A few reports have already demonstrated successful results from trans-anterior or -posterior communicating artery approaches for the treatment of patients with challenging direct routes to the occluded lesions.[12,13] However, due to anatomical variations in the anterior and posterior communicating arteries, those approaches would increase risks of events including arterial dissection, perforation, and propagation of thrombus to patent arterial territories, resulting in potentially catastrophic complications.[14]

Since the effect of leptomeningeal enhancement is to supplement blood flow via leptomeningeal collaterals to the territory showing hemodynamic insufficiency, clinical symptoms and angiographic patency of Willisian and leptomeningeal collaterals should be carefully evaluated. In the present case, the patient presented with fluctuating aphasia and motor weakness of the affected limbs, suggesting hemodynamic insufficiency. Diagnostic angiography showed right cervical ICA stenosis and tandem occlusion of the left cervical ICA and M2 segment of the ipsilateral MCA. In addition, preoperative right carotid injection demonstrated faint but longitudinal filling of the contralateral ACA into the frontoparietal cortex via the distal MCA through the anterior communicating artery in the late arterial phase, and left vertebral injection showed occlusion of the M2 segment of the left MCA, suggesting insufficient leptomeningeal compensation in the frontoparietal cortex. Based on these findings, we decided to perform CAS for the stenoses right cervical ICA. The right carotid injection obtained after stenting did show robust filling of leptomeningeal anastomoses via the left ACA and demonstrate antegrade flow in the proximal segment of the left MCA. Therefore, it seemed that leptomeningeal blood flow from the left ACA to the ipsilateral MCA and also from the recanalized distal MCA to the occluded MCA branches might have contributed to the improvement of neurological symptoms. Furthermore, although the increase in leptomeningeal flow indicated persistent occlusion of the left MCA branches on the postoperative angiogram, since the MRA obtained 1 day after the CAS revealed the good patency of the left MCA and the recanalization of the occluded MCA branch, the increase in antegrade MCA flow through the anterior communicating artery might also have contributed to the more improvement of blood flow.

Despite the successful results in the present case, the efficacy of enhancing leptomeningeal collaterals may vary depending on the volume of the territory showing insufficient hemodynamics or the time from onset to treatment. The symptoms and angioarchitecture in patients should therefore be carefully analyzed to clarify whether leptomeningeal enhancement would be efficacious.

Conclusion

A patient with tandem occlusion of the carotid and middle cerebral arteries was successfully treated with enhancement of the leptomeningeal and the antegrade flow through the Willisian collateral by emergent CAS for contralateral carotid artery stenosis. Stenting of the contralateral carotid artery may provide an effective alternative when antegrade recanalization of the tandem occluded arteries appears overly challenging.

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Disclosure Statement

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