 doi: 10.2176/jns-nmc.2021-0403

NMC Case Report Journal 9, 49-53, 2022

New Cortical Spot Cerebral Infarction Out of Border Zone in ICA Occlusion Suggests Recanalization: A Case Report

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Abstract

Internal carotid artery occlusion rarely recanalizes spontaneously. Awareness of signs of recanalization is important, as it may necessitate changing the treatment strategy. We report a case of new cortical infarction outside the border zone, which led to the realization of internal carotid artery recanalization and revascularization.

A 76-year-old woman presented with mild dysarthria. Magnetic resonance imaging showed cerebral infarction in the left-hemispheric border zone and occlusion of the internal carotid artery origin. Cerebral angiography performed showed complete occlusion of the internal carotid artery origin and intracranial collateral blood flow from the external carotid artery through the ophthalmic artery. She was diagnosed with infarction due to a hemodynamic mechanism caused by internal carotid artery occlusion and was treated with supplemental fluids and antithrombotic drugs. Four days after hospitalization, the right paralysis worsened and a new cerebral infarction was observed in the cortex, outside the border zone. This infarction appeared to be embolic rather than hemodynamic; thus, we suspected recanalization of the internal carotid artery. The patient underwent emergency cerebral angiography again, which revealed slight recanalization. Thus, emergency revascularization and carotid artery stenting were performed. New cortical infarcts outside the border zone in patients with complete internal carotid artery occlusion is an important finding, suggesting spontaneous recanalization of the occluded internal carotid artery.

Keywords: ICA occlusion, spontaneous recanalization, carotid artery stenting, border zone infarction, hemodynamic infarction

Introduction

Carotid artery stenting (CAS) is indicated for internal carotid artery (ICA) stenosis. CAS is not a common treatment for complete occlusion of the ICA, and ICA occlusion sometimes results in spontaneous recanalization.

We report a case of complete occlusion of the ICA, which caused a hemodynamic cerebral infarction. The present case showed a late spontaneous recanalization in the subacute phase, causing a cerebral embolus in the cortex outside of the border zone. A new cortical infarction in a case of complete occlusion of the ICA is an important finding because it highlights the possibility of spontaneous recanalization of the occlusion.

Case Report

A 76-year-old woman was admitted to our hospital for dysarthria. She had developed dysarthria 3 days before the admission. She had a medical history of smoking, hypertension, and dyslipidemia. On admission, her blood pressure was 168/98 mm/Hg and her pulse was 60/min with a regular rhythm. No atrial fibrillation was observed on monitoring ECG or Holter ECG later. She had mild dysarthria and a National Institutes of Health Stroke Scale (NIHSS) score of 1. Other neurological deficits, including paresis, sensory impairment, and impairments of higher function, were not observed.

Magnetic resonance (MR) imaging showed acute

Received December 14, 2021; Accepted January 31, 2022

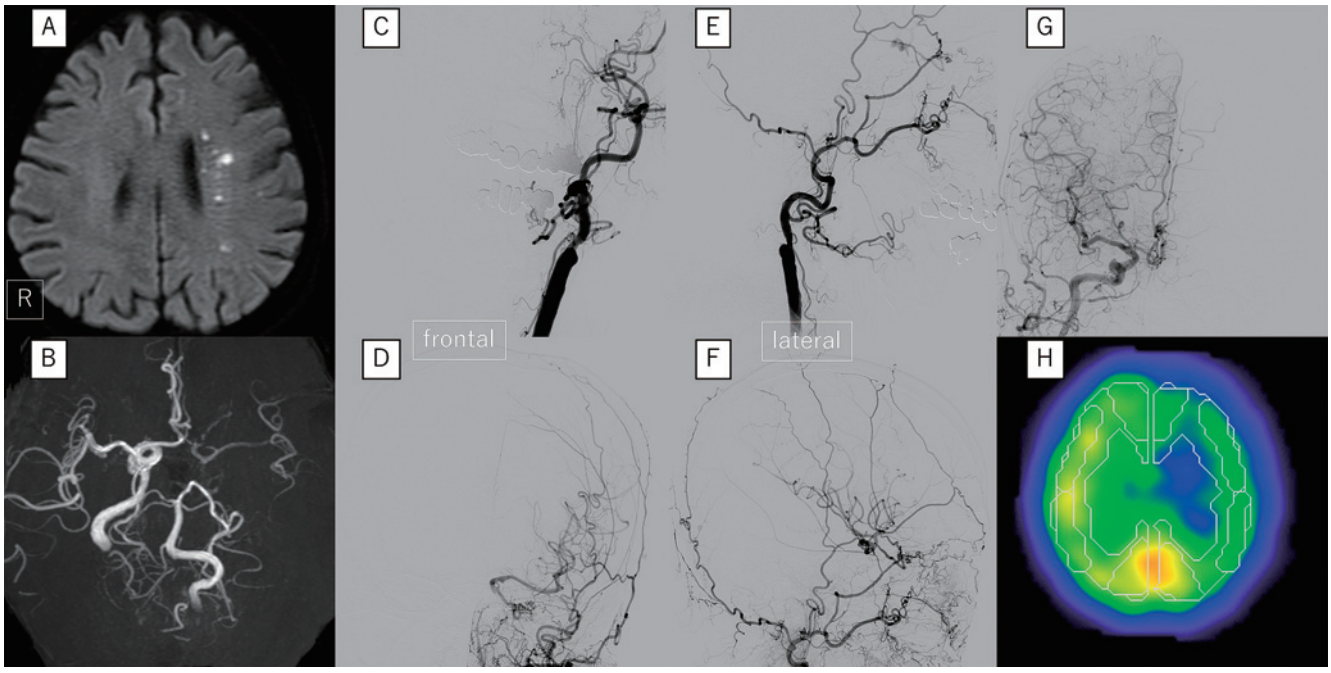

Fig. 1 Imaging findings on admission.
MR images (DWI: A, MRA: B), left CCA angiography (early phase; C, E, late phase; D, F), and right ICA angiography on admission. Resting single-photon emission tomography (SPECT) of ^{123}I-IMP on the day after admission (H).
Diffusion-weighted MR image showing acute ischemic lesions distributed in the linear border zone (A). The left ICA is not delineated (B). The left ICA is occluded at its origin and collateral blood flow from the ipsilateral external carotid artery through the ophthalmic artery to the ICA was observed (C-F). Right ICA angiography showed no crossflow into the left MCA via the Acom (G). ^{123}I-IMP resting SPECT showed decreased blood flow mainly in the border zone of the left hemisphere (H).
Abbreviations: MR, magnetic resonance; MRA, magnetic resonance angiography; DWI, diffusion-weighted image; CCA, common carotid artery; ICA, internal carotid artery; SPECT, single-photon emission tomography; MCA, middle cerebral artery; Acom, anterior communicating artery.

ischemic lesions in the border zone of the middle cerebral artery (MCA) and anterior cerebral artery (ACA) of the left hemisphere (Fig. 1A). MR angiography did not delineate the left internal carotid artery (Fig. 1B). On the day of admission, cerebral angiography revealed that the left ICA was completely occluded from its origin (Fig. 1C, D). Collateral blood flow from the ipsilateral external carotid artery through the ophthalmic artery to the ICA was observed (Fig. 1D, F). Right ICA angiography showed no crossflow into the left MCA via the anterior communicating artery (Fig. 1G). The left MCA, which was faintly visible on MRA, was believed to reflect collateral blood circulation coming mainly from the external carotid artery.

The patient was diagnosed with hemodynamic cerebral infarction associated with occlusion of the left ICA. Three days since the onset of the disease, the patient's symptoms were stable with only mild dysarthria, and collateral circulation from the external carotid artery was well developed; we decided not to administer recombinant tissue-type plasminogen activator or perform acute revascularization. Resting single-photon emission tomography (SPECT) of ^{123}I-IMP on the day after admission showed decreased blood flow mainly in the border zone of the left hemisphere (Fig. 1H). She was treated with antithrombotic therapy comprising aspirin, clopidogrel, and argatroban. Four days after admission, the patient developed right-sided hemiparesis especially strong paralysis in the upper limbs, and her NIHSS score was 6. Diffusion-weighted MR image revealed a mildly enlarged infarct zone in the centrum semiovale and new spot infarcts in the left precentral and postcentral gyri (Fig. 2A). The enlargement of the infarcts located in the centrum semiovale seemed to be consistent with a hemodynamic mechanism. Nevertheless, as cortical new spot infarcts were not considered to be due to hemodynamic mechanism but instead an embolic mechanism, we suspected recanalization of the occluded left ICA and performed emergent cerebral angiography. A left carotid angiogram revealed recanalization of the left ICA, which had severe stenosis and slow antegrade blood flow (Fig. 2 B, C). An Optimo 9F balloon catheter (Tokai Medical Products, Kasugai, Aichi, Japan) was placed in the left common carotid artery via the coaxial technique. Then, a Shouryu HR 4 × 7 mm balloon catheter (Kaneka, Osaka, Osaka, Japan) along with a CHIKAI micro guidewire (Asahi Intecc, Nagoya, Aichi, Japan) was placed in the left external carotid artery. By occlusion of both the external and com-

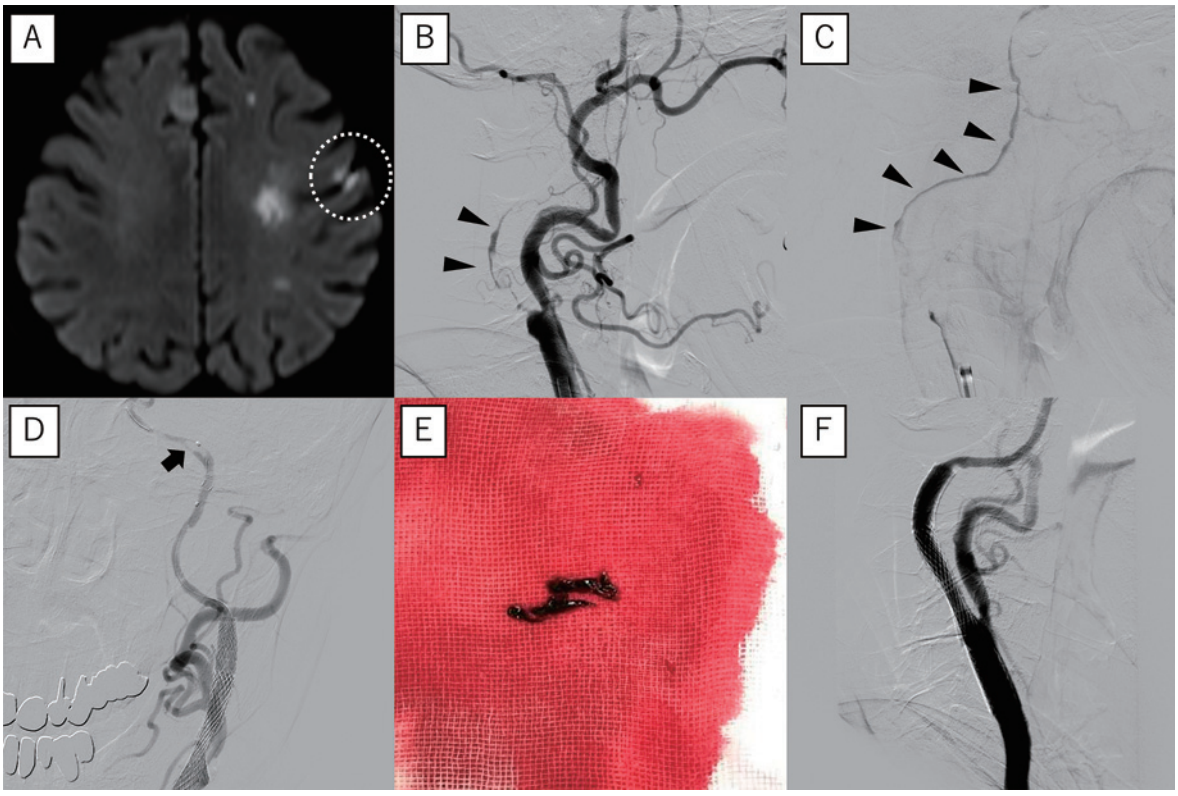

Fig. 2 Imaging when symptoms worsen and endovascular treatment.
MR image on day 4 when symptoms worsened (A). Angiography (B, C, D, F) and gross appearance of a retrieved thrombus (E). New infarcts are observed in the center of the MCA supply region (A, circle). The already existing infarcts in the centrum semiovale enlarge. The occluded ICA is recanalized (early phase; B, late phase; C, arrowheads). There is a thrombus after stenting (D, arrow). Red thrombi are retrieved (E). Finally, complete recanalization is performed (F).
Abbreviations: MR, magnetic resonance; MCA, middle cerebral artery; ICA, internal carotid artery.

mon carotid arteries, the blood flow was reversed and returned to the femoral vein. Under reverse flow, the lesion was passed using SpiderFX 4 mm (Medtronic, Irvine, CA, USA) and CHIKAI. SpiderFX was deployed distal to the stenosis. Predilation using the Sterling 3.5 × 20 mm (Boston Scientific, Marlborough, MA, USA) was performed under reverse flow as well as filter protection, followed by the deployment of the PRECISE 9 × 40 mm (Cordis, Santa Clara, CA, USA). Since there was residual stenosis proximal to the PRECISE, a WALLSTENT 10 × 24 mm (Boston Scientific, Marlborough, MA, USA) was overlapped. After retrieval of the SpiderFX, there was a thrombus located in the petrous segment (Fig. 2D). The thrombus was retrieved using React71 (Medtronic, Irvine, CA, USA) (Fig. 2E). Finally, complete revascularization was achieved (Fig. 2F). Right hemiparesis improved after the procedure, and a good outcome was obtained (the modified Rankin Scale score at 3 months was 1). Although carotid plaque imaging by MRI was planned, it could not be performed because of the rapid worsening of the patient's condition.

Discussion

The feasibility of emergent CAS in patients with acute ischemic stroke has been reported.[1,2] Nevertheless, the safety and feasibility of emergent CAS for an acutely occluded ICA have yet to be elucidated. Although there have been some reports of CAS for occluded ICA in the subacute or chronic phase,[3] it has potential hazardous risks such as dissection and extravasation for the neurointervention of occluded vessels, making it still quite challenging. Hence, recognition of the change from occlusion to stenosis is crucial in terms of a neurointerventional strategy. In the present case, the observation of a new cortical infarction was a key factor in terms of the treatment strategy.

Brain tissues in the border zones of different arteries are vulnerable to ischemia.[4] In patients with chronic ICA occlusion, infarcts are commonly observed in the border zones between the MCA and ACA because of reduced perfusion in the border zone, which is known as an infarct of hemodynamic mechanism.[5] Recent understandings have advocated that the pathogenesis of the border zones infarction or water-shed infarction in patients with steno-occlusive lesions is considered to be attributable mainly to

a decrease in washout of emboli.[6,7] In a circumstances that subtotal or complete occlusion of an artery develops, the blood flow diminishes, resulting in a decrease in antegrade perfusion. It brought a reduced washout of emboli especially in cortical border-zone portions of the brain circulation.[6,7] Conversely, infarcts of an embolic mechanism within the anterior circulation are commonly located in the center of the supply of the MCA territory.[8] Emboli have a predilection site for the destination of embolic particles. Therefore, if a recanalization occurs, the antegrade perfusion becomes more prominent, which should improve the washout of emboli, making it difficult for water-shed infarction to develop. By contrast, infarcts located at the junction between deep perforating arteries are described as subcortical border zone infarcts, the mechanism of which is considered to be hemodynamic compromise.[5]

In the present case, when the angiography revealed occlusion of the left ICA, infarcts were observed to be distributed in the cortical border zone at the junction of the MCA and ACA territories. Nevertheless, when recanalization of the occluded left ICA was observed on day 4, the patient had new infarcts not in the border zone but in the center of the supply of the left MCA territory. The observation that new infarcts developed in a territory other than a border zone in a patient with ICA occlusion suggest recanalization of the occluded ICA. Thus, recanalization of the occluded ICA must be considered when new infarcts are observed in a territory other than a border zone of the MCA and ACA territory, which should induce a change in the treatment strategy. Additionally, an enlargement of already existing infarcts in the left centrum semiovale was noted on day 4. This notification was considered to represent a progression of subcortical border zone infarcts by hemodynamic compromise.

Atheromatous occlusion of the ICA generally remains occlusive; nevertheless, spontaneous recanalization has been observed in 2.3%-10.3% of patients in long-term follow-up reports.[9-11] Most patients were asymptomatic at the time of recanalization; however, recanalization was accompanied by ischemic symptoms in 12.5% of the patients, and spontaneous recanalization itself can trigger a transient ischemic attack or cerebral infarction.[9] Hence, recognizing the recanalization of occlusive ICA is crucial. In our case, noting the development of new infarcts located in areas other than the border zone prompted us to perform emergent angiography.

Conclusion

We report a case of ICA occlusion showing a late spontaneous recanalization in an occlusive lesion, which was treated with CAS. The development of new cortical ischemic lesions in locations other than the border zone in patients with ICA occlusion should be considered a suggestive sign of change from occlusion to stenosis. Further analysis of additional cases is required to elucidate the validity of this sign in terms of emergent neurointervention for occlusive ICA.

Acknowledgments

None.

Author Contributions

H.I. analyzed the data and drafted the manuscript. M.O. and Y.N. reviewed and gave feedback on potential problems of the draft. M.M. and N.M. contributed to the organization of the conclusion and checked the final manuscript.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author, M.O., upon reasonable request.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Patient Consent

Informed consent was obtained from the patient for the publication of this case report and the accompanying images.

Conflicts of Interest Disclosure

The authors declare that they have no conflicts of interest.

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