Case Report

A case of acute cerebral infarction associated with an accessory middle cerebral artery in a patient who underwent thrombectomy

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Background: The accessory middle cerebral artery (AMCA) is a middle cerebral artery (MCA) anomaly originating from the anterior cerebral artery. We report our experience of a case in which thrombectomy was undertaken for a patient with hemodynamics that were specific to the AMCA.

Case Presentation: An 84-year-old man with a history of atrial fibrillation developed paralysis of the left upper and lower extremities. Imaging examinations suggested tandem occlusion of the right internal carotid artery and the origin (M2 segment) of the right MCA. An extremely narrow MCA was visualized. Because there was concern regarding development of frontal lobe infarction, thrombectomy was carried out to restore anterograde blood flow, but an AMCA was found. Recanalization of the main MCA in the infarction zone resulted in hemorrhagic infarction, and the patient died of cerebral herniation.

Conclusion: When a vascular variation like AMCA is suspected, a careful evaluation of hemodynamics is necessary before undertaking endovascular intervention.

Key words: Accessory middle cerebral artery, acute ischemic stroke, cerebrovascular anomaly, endovascular therapy, hemodynamic

INTRODUCTION

A NACCESSORY MIDDLE cerebral artery (AMCA) is a variant vessel that branches from the anterior cerebral artery and provides blood flow to the frontal branch of the middle cerebral artery (MCA) territory.^{1,2} The AMCA is found during autopsy and cerebral angiography in approximately 0.3–4.0% of individuals.³ We report a rare case in which thrombectomy was carried out for a patient with hemodynamics that were specific to an AMCA.

CASE REPORT

A N 84 -year-old man with a history of hypertension, diabetes mellitus, and atrial fibrillation was brought to

Corresponding: Ichiro Deguchi, MD, PhD, Department of Neurology, Saitama Medical University International Medical Center, 1397-1 Yamane, Hidaka, Saitama 350-1298, Japan. 3E-mail: ideguchi@saitama-med.ac.jp. Received 12 Jun, 2019; accepted 30 Aug, 2019; online publication 27 Sep, 2019 Funding information None. our hospital after weakness of the left upper and lower extremities was observed on waking (time from detection of symptoms to arrival at the hospital, 67 min; time from last feeling well to arrival at the hospital, 11 h, 37 min). On arrival to our hospital, his blood pressure, pulse rate, and temperature were 146/83 mmHg, 85 b.p.m. (irregular), and 36.8°C, respectively. Neurological findings showed right conjugate deviation and incomplete paralysis of the left side. Pathological reflexes were present on the left side (Babinski and Chaddock). His National Institutes of Health Stroke Scale score⁴ was 15. Brain computed tomography (CT) (Fig. 1A,B) showed a hypodense area that extended from the right temporal lobe to the parietal lobe (Alberta Stroke Program Early CT score⁵ [ASPECTS], 6). Diffusionweighted magnetic resonance imaging (MRI) showed high signal intensity in an area of the right temporo-parieto-occipital lobes, part of the superior frontal lobe, the right posterior limb of the internal capsule, and the left medial occipital lobe (Fig. 1C,D; diffusion-weighted imaging [DWI]-ASPECTS⁶, 5/10). The area that extended from the right temporal lobe to the parietal lobe also appeared slightly hyperintense on fluid-attenuated inversion recovery images

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Fig. 1. A, B, On admission of an 84-year-old man with paralysis of the left upper and lower extremities, axial brain computed tomography shows a hypodense area extending from the right temporal lobe to the parietal lobe. C, D, On admission, axial brain diffusion-weighted imaging shows a high signal intensity area in the right temporo-parieto-occipital lobes, part of the superior frontal lobe, the right posterior limb of the internal capsule, and the left medial occipital lobe. E, F, On admission, axial brain fluid attenuated inversion recovery images show faint high signal intensity areas extending from the right temporal lobe to the parietal lobe. G, Magnetic resonance angiography does not show the right internal carotid artery from its origin, but faintly shows the right middle cerebral artery because of collateral blood flow from the contralateral side. H, On day 2, axial brain computed tomography shows extensive hemorrhagic cerebral infarction in the right middle cerebral artery territory and uncal herniation.

(Fig. 1E,F). Magnetic resonance angiography (MRA) (Fig. 1G) did not show the right internal carotid artery (ICA) from the level of its origin, but faintly showed the right MCA because of collateral circulation from the left side. The MRI and MRA findings suggested tandem occlusions of the right ICA and the origin (M2 segment) of the right MCA (inferior trunk). These imaging methods showed an established right temporoparietal infarction. Despite the absence on DWI lesion, the patient had impaired consciousness and his eyes showed conjugate deviation to the right, suggesting the functional impairment of the right frontal lobe. We considered that the right frontal area was at high risk for irreversible ischemic damage as only collateral flow from the contralateral side through the anterior communicating artery perfused it. After consulting with physicians at the Department of Endovascular Therapy, we decided to undertake thrombectomy to relieve occlusion of the ICA only and to ensure anterograde blood flow. A 9-Fr sheath was inserted from the right groin. Right ICA angiography showed occlusion of the cervical portion of the right ICA. The Penumbra aspiration system ACE 68 (Penumbra, Alameda, CA, USA) was guided up to the thrombus, and a dark red thrombus was retrieved by the direct aspiration first-pass technique. Because repeated angiography showed occlusion at the top of the ICA (Fig. 2A), the Solitaire FR device (6×40 mm; Covidien, Irvine, CA, USA) was guided to the occlusion site. The thrombus was retrieved using the Penumbra system and a stent retriever, and a dark red thrombus was found in the stent. Thrombectomy achieved complete recanalization, which was defined as a thrombolysis in cerebral infarction score⁸ of 3 (door to puncture, 50 min; door to recanalization, 128 min). Right carotid angiography identified an AMCA, which arose from the proximal A1 segment of the right anterior cerebral artery, and was slightly thinner than

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Fig. 2. Angiography images from an 84-year-old man with an accessory middle cerebral artery (AMCA) who underwent thrombectomy. A, Digital subtraction angiography shows occlusion at the top of the internal carotid artery (ICA; arrow). B, Right carotid angiography (anteroposterior view), which was obtained after thrombectomy, shows an AMCA arising from the proximal A1 segment of the right anterior cerebral artery, which is slightly thinner than the main middle cerebral artery (MCA). C, The MCA which was observed on preprocedural magnetic resonance angiography, was actually the AMCA (black arrowhead) as visualized by collateral blood flow (dotted arrow).



Fig. 3. Photomicrographs of a thrombus aspirated by endovascular therapy from an 84-year-old man with an accessory middle cerebral artery who underwent thrombectomy. The thrombus was composed of platelets, fibrin, and red blood cells. A, White blood cells were also present. B, In particular, numerous red blood cells were present in the central part of the thrombus. Neither calcified materials nor lipid deposits were seen. Hematoxylin–eosin stain. Scale bar = $100 \mu m$.

the main MCA (Fig. 2B). A pathological examination identified the retrieved thrombi as fresh thrombi, which were mainly composed of fibrin and erythrocytes. This was consistent with embolization resulting from atrial fibrillation (Fig. 3A,B). Because brain CT that was carried out immediately after the procedure showed increased edema, antiedema therapy and strict antihypertensive management were initiated. However, on the day following the procedure, the patient's level of consciousness deteriorated and anisocoria occurred. Brain CT (Fig. 1H) showed extensive hemorrhagic cerebral infarction in the right MCA territory, subfalcine herniation, and uncal herniation. Because his family did not request cerebral decompression, he was conservatively treated and pronounced dead on hospital day 4.

DISCUSSION

I N THE PRESENT case, the patient appeared to have tandem occlusion of the right ICA and the origin (M2 segment) of the right MCA (a branch perfusing the temporal and parietal lobes) on MRI and MRA images. Although the MCA was visualized because of collateral blood flow from the contralateral side, the MCA was extremely narrow. There was concern that there was a risk of infarction in the frontal lobe, which includes the motor cortex. Thrombectomy was carried out to restore anterograde blood flow. After thrombi were retrieved from the ICA, an AMCA was found, and recanalization of the main MCA in the infarction zone resulted in hemorrhagic infarction. Comparison of preprocedural and postprocedural MRA findings showed that the thin MCA that was observed on preprocedural MRA was actually the AMCA as visualized by collateral blood flow (Fig. 2C).

To reduce the time of recanalization of the occluded blood vessel, blood flow from the contralateral side was not examined. If angiography by the contralateral approach had been undertaken in this case, we might have identified the presence of a variant vessel (AMCA) and avoided endovascular surgery.

Because thrombectomy is effective for acute cerebral infarction,9 the number of patients who have this procedure could increase in the future. Consequently, opportunities to treat patients with concomitant vascular variations are also expected to increase. A previous report described a patient with discordant imaging findings and clinical symptoms, in whom an AMCA was observed after mechanical thrombectomy, which is similar to the present case.¹⁰ In cases of vascular variation, such as the present case, endovascular therapy could lead to undesirable consequences. We should consider the detailed evaluation of hemodynamics before endovascular intervention, especially when the patient had both infarcted and normal-appearing regions by marked contrast in the same MCA area, despite the occlusion of ICA (unusual presentation of cerebral infarction). Like our case, these patients could possess AMCA and the occlusion of one artery might have caused an infarction in only a partial area of the MCA.

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DISCLOSURE

Approval of the research protocol: The Institutional Review Board of Saitama Medical University International Medical Center approved the case study (No. 19-092).

Informed consent: N/A.

Registry and the registration no. of the study/trial: N/A. Animal studies: N/A.

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