A Case of Acute Embolism of the Accessory Middle Cerebral Artery Treated Using ADAPT Thrombectomy without Lesion Passing

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Abstract

An accessory middle cerebral artery (AMCA) is a variant vessel that branches from the anterior cerebral artery (ACA) and runs through the Sylvian fissure along the middle cerebral artery (MCA). We report a case of acute embolic occlusion of the AMCA that was treated with thrombectomy using direct aspiration first pass technique (ADAPT). An 88-year-old woman with a history of atrial fibrillation, loss of consciousness, and right hemiparesis was referred to our hospital. Diffusion-weighted magnetic resonance imaging (MRI) showed high signal intensity in the left frontal lobe, insular cortex, and deep white matter, and magnetic resonance angiography (MRA) demonstrated left internal carotid artery (ICA) occlusion. Mechanical thrombectomy using the ADAPT technique was performed with complete recanalization. Final angiography revealed left ACA and AMCA because of the thrombus located at the origin of the left ACA and AMCA. In the case of an acute ischemic stroke associated with AMCA, it is difficult to understand and recognize the anatomy of the vessel before thrombectomy. Therefore, the ADAPT technique, which can treat acute embolic occlusion without lesion passing, is recommended due to its safety. If there is a mismatch between the perfusion area of the occluded artery and the ischemic area or the neurological findings before thrombectomy, it is extremely important to keep in mind the presence of vessel variation in the MCA.

Keywords: thrombectomy, accessory middle cerebral artery, ADAPT

Introduction

Ischemic stroke due to occlusion of the horizontal portion of the middle cerebral artery (MCA, M1) is very common. However, occlusion of the accessory MCA (AMCA) or duplication of MCA (DMCA) in acute ischemic stroke is extremely rare.

We report a case of acute embolic occlusion of the AMCA that was managed with thrombectomy using direct aspiration first pass technique (ADAPT). We also present the relevant review of the literature.

Case Report

An 88-year-old woman with a history of hypertension, and atrial fibrillation was referred to our hospital, presenting with acute onset of unconsciousness and right hemiparesis. Time from detection of symptoms to arrival at the hospital was 41 minutes; time from the last stable patient condition to arrival at the hospital was 77 minutes. On examination, she had left conjugate deviation, right severe hemiparesis, and impaired sensation of the right side. Her National Institutes of Health Stroke Scale (NIHSS) score was 19. Computed tomography (CT)
showed no hemorrhage and early ischemic changes in the left frontal lobe. Diffusion-weighted imaging (DWI) showed high signal intensity in an area of the left frontal lobe, insular cortex, and deep white matter (Alberta Stroke Program Early CT score [ASPECTS]-DWI, 6/11 points) (Fig. 1A and 1B).

Fig. 1 (A, B) DWI showing high signal intensity areas in the left frontal lobe, insular ribbon cortex, and deep white matter. (C, D) FLAIR images that showed high signal intensity at the DWI no longer showing high signal intensity areas. (E, F) (Stereo vision) MRA showing the left ICA occlusion. DWI: diffusion-weighted imaging, FLAIR: fluid-attenuated inversion recovery, ICA: internal carotid artery, MRA: magnetic resonance angiography.
These areas did not appear high signal intensity on fluid-attenuated inversion recovery (FLAIR) image (Fig. 1C and 1D). Magnetic resonance angiography (MRA) did not show the left internal carotid artery (ICA), and the left MCA, but showed the left anterior cerebral artery (ACA) because of collateral flow via the anterior communicating artery (Fig. 1E and 1F). We diagnosed acute cerebral embolism due to left ICA occlusion, and recognized the mismatch between high signal intensity of DWI and FLAIR. Therefore, we decided to perform endovascular thrombectomy to relieve the occlusion of the ICA. Taking into consideration the risk of cerebral hemorrhage, we skipped t-PA intravenous injection because of the borderline score of ASPECTS-DWI. Informed consent for the procedure was obtained from the patient’s family.

The first left ICA angiogram (before proceeding with the thrombectomy) revealed spontaneous recanalization of the left ICA and MCA, but no cerebral flow to the frontal lobe from a part of the superior trunk of MCA was noted (Fig. 2A and 2B). Moreover, there was a filling defect sign at the anterior superior wall of the left MCA near its origin (Fig. 2A and 2C). We considered that lesion passing was a high-risk factor for vascular perforation because we did not understand the vascular anatomy and we suspected an anomaly of the MCA. We attempted to perform a thrombectomy using the ADAPT technique at the filling defect sign without lesion passing. We introduced a balloon guiding catheter FlowGate (Stryker, Kalamazoo, MI, USA) into the left ICA (this was time consuming owing to sclerotic changes in the aorta). An aspiration catheter, the AXS Catalyst6 (Cat6, Stryker), was introduced to the nearby filling defect, using a stent-anchoring technique with Solitaire FR stent 4 × 40 mm (Medtronic, Dublin, Ireland) and expanded it from the left MCA horizontal portion to the distal ICA inside the filling defect (Fig. 2C and 2D). After recognizing the Cat6 contact at the filling defect, the Solitaire FR stent was withdrawn from the Cat6 followed by an aspiration from the Cat6 (ADAPT); thereafter, we pulled the Cat6 into the guiding catheter. A dark red thrombus was retrieved from Cat6 with ADAPT. The thrombectomy achieved complete recanalization (Fig. 3A and 3B), which was defined as a thrombolysis with a cerebral infarction score of 3 (TICI 3) (door to puncture, 124 minutes; puncture to recanalization, 94 minutes). Post-thrombectomy carotid angiogram identified AMCA, which arose from the proximal A1 segment of the left ACA (Fig. 3C). After the procedure, head CT showed a mild hemorrhagic infarction, which was caused by reperfusion after thrombectomy (Fig. 4A and 4B). We initiated antihypertensive control therapy. After the thrombectomy, the state of unconsciousness, left conjugate deviation, and paralysis were improved. However, the aphasia did not improve. The NIHSS score on the day after thrombectomy was 13. She was transferred to another hospital for rehabilitation on hospital day 19. Unfortunately, the modified Rankin Scale at 3 months after thrombectomy was four.

Informed consent was obtained from the patient’s family about publishing the case report, and it was passed through the ethics committee at our hospital.

Discussion

In 1962, Crompton described the AMCA, which included DMCA, and the anomalous vessel originating from the A1 portion of the ACA, which coursed parallel to the MCA. In 1973, Teals et al. proposed two types of AMCA: one originating from the ipsilateral ACA proximal to anterior communicating artery (the true AMCA) and one originating from the distal ICA between the anterior choroidal artery and the terminal bifurcation of the ICA (DMCA). Additionally, in 1998, Komiyama et al. postulated as follows: AMCA is the anomalously early ramification of the early branch of the MCA supplying the anterior frontal lobe and that DMCA is the anomalously early ramification of the early branch of the MCA supplying the anterior temporal lobe. Knowledge of anomalous early ramification of the MCA is important in understanding the collateral blood supply in ischemic stroke associated with DMCA or AMCA. In angiographic and anatomical studies, the AMCA and DMCA have been found to have an incidence of 0.3–4.0% and 0.2–2.9%, respectively. Consequently, it is very difficult to accurately diagnose acute ischemic stroke associated with the DMCA and AMCA, and further to treat as soon as possible. If we do not recognize the vessel anatomy in the case of an ICA occlusion proximal to the DMCA or AMCA, then after revascularization, we might misjudge recanalization of ICA and MCA despite occlusion of the DMCA or AMCA.

Several thrombectomy cases associated with the AMCA or DMCA had been reported. Koge et al. described endothelial injury by a stent retriever device in a case of ICA occlusion with concomitant MCA and DMCA occlusion and supported aspiration thrombectomy in similar situations. Ray N. et al. also supported the notion that a tandem occlusion case involving the AMCA could be treated by ADAPT. Moreover, Hayashi et al. reported cases of AMCA associated with unruptured aneurysm at its origin. Therefore, the risk of a lesion cross...
passing with a stent retriever, when the vessel anatomy is not understood, or especially suspected of the vessel anomaly of MCA, is considered to be high. However, the ADAPT technique does not require lesion passing. From a safety point of view, the ADAPT technique is better than a stent retriever in a thrombectomy in a case of acute embolism associated with the AMCA and DMCA. Additionally, we should attempt to recognize the anatomical variation of the AMCA or DMCA during thrombectomy as far as possible. If there is a mismatch between the occluded artery territory and the ischemic area on a DWI of the MRI or the neurological findings, a vessel variation like the AMCA or DMCA is a possibility. In the present case, we first diagnosed acute cerebral infarction due to left ICA occlusion; however, it was an AMCA that was not detected. A left ICA angiogram revealed spontaneous recanalization of the left ICA and MCA, not cerebral flow to the frontal lobe from ACA and a part of the superior trunk of MCA, and filling defect sign at the superior wall of the left MCA near its origin (white arrow). (D) Frontal view, enlarged. Aspiration catheter (AXS Catalyst6, bold white line) was introduced to nearby the filling defect. Thrombectomy using a ADAPT was performed. ACA: anterior cerebral artery, ADAPT: direct aspiration first pass technique, ICA: internal carotid artery, MCA: middle cerebral artery.
recanalization of the left ICA and MCA, but no cerebral flow to the frontal lobe from a part of the superior trunk of the MCA. A mismatch between vessel territory and ischemic lesion of MRI (DWI) or neurological findings was still observed. We suspected some variation of the MCA and could not diagnose it; therefore, we performed the ADAPT technique and thrombectomy without lesion passing. As a result, left ICA angiogram revealed recanalization of ACA and AMCA, that supplied the frontal lobe. In retrospect, we suggest that thrombectomy with a stent retriever for the AMCA or ACA, which is thinner than normal MCA, is not recommended because it may cause endothelial injury and dissection.

In conclusion, we report a case of an acute embolic occlusion of the AMCA who underwent a thrombectomy where we used the ADAPT technique. If there is a mismatch between the occluded artery territory and the ischemic area or the neurological findings before a thrombectomy, it is necessary to keep in mind the presence of an AMCA or DMCA. The ADAPT technique, which can treat acute embolic occlusion without lesion passing is highly
recommended rather than a stent retriever from the view of safety in the suspected case of occlusion of the AMCA or DMCA.

**Conflict of Interest Disclosure**

All authors have no conflict of interest.

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