Cerebral Infarction Associated with Accessory Middle Cerebral Arteries: Two Case Reports

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Abstract

We herein present two cases of cerebral infarction in the middle cerebral artery (MCA) territory associated with an accessory middle cerebral artery (AMCA), which is a rare anomalous vessel arising from the anterior cerebral artery that coexists with the main trunk of the MCA. Cerebral infarction occurred in both patients: due to occlusion of the MCA main trunk in one patient and occlusion of the AMCA in the other patient. These cases suggest the importance of recognizing an AMCA when interpreting neuroradiological findings in patients with MCA ischemic stroke, especially in the hyperacute phase.

Key words: accessory middle cerebral artery, cerebral angiography, cerebral infarction, cerebrovascular anomaly, magnetic resonance angiography

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Introduction

An accessory middle cerebral artery (AMCA) is a relatively uncommon anomaly of the middle cerebral artery (MCA) that arises from the anterior cerebral artery (ACA) and runs in the Sylvian fissure along with the main trunk of the MCA to supply part of the MCA territory. Its prevalence ranges from 0.3% to 4.0% in autopsy and angiographic series (1-6). Although there is a well-documented association between the incidence of AMCA and intracranial aneurysms, there have been few reports regarding an association with anomalous vessels in relation to acute ischemic stroke (7-10). We herein describe two cases of infarction in the MCA territory associated with an ipsilateral AMCA in which stroke was caused by occlusion of the MCA main trunk and AMCA, respectively.

Case Reports

Case 1

A 60-year-old right-handed male smoker with type 2 diabetes mellitus and hyperlipidemia presented to our emergency department due to the sudden onset of difficulty in speaking. His symptoms began to improve in the ambulance and he arrived at our hospital approximately 30 minutes after onset. One day before this event, he had experienced transient slurring of speech lasting for one hour.

Upon arrival, the patient was alert and cooperative. His blood pressure was 122/64 mmHg, and his pulse was regular at 85 beats/min. A neurologic examination revealed non-fluent aphasia without focal weakness and sensory changes. His National Institute of Health Stroke Scale (NIHSS) score was 3. Initial magnetic resonance imaging (MRI) performed on admission revealed small acute infarcts in the left centrum semiovale (Fig. 1A), although no fresh infarcts responsible for the aphasia were detected. Magnetic resonance angiography (MRA) showed clubbed protrusion from the distal part of the left internal carotid artery (ICA). In addition, a vessel arose adjacent to the left ICA bifurcation that supplied the cortical territory of the orbitofrontal and prefrontal arteries (Fig. 1B). The truncated area of protrusion from the ICA was found to have caused occlusion of the main MCA trunk, and the feeding artery off the left MCA territory was thought to be an AMCA.
Figure 1. Case 1: (A) On admission, axial cerebral DWI reveals acute infarction in the left centrum semiovale. (B) Initial magnetic resonance angiography (MRA) shows a left AMCA with occlusion of the main MCA. (C) On day 3, axial cerebral DWI reveals acute infarction in the left anterior temporal lobe. (D) Left carotid angiography (anteroposterior view), shows an AMCA arising from the proximal region of the ACA with stenosis of the proximal region of the recanalized main MCA.

Intravenous thrombolytic therapy was not initiated due to the patient’s rapid neurological improvement, instead he was treated with a continuous infusion of heparin sodium. MRI repeated three days after admission showed a new infarct restricted to the left anterior temporal lobe, while a large part of the left MCA territory was spared from infarction (Fig. 1C). MRA suggested recanalization of the main MCA, with persistent stenosis at the proximal site. Left carotid angiography performed four days after admission identified an AMCA arising from the proximal A1 segment of the left ACA that was slightly thinner than the main MCA. No aneurysms were noted. Severe stenosis of the proximal main MCA trunk was confirmed (Fig. 1D), which remained unchanged on follow-up MRA performed six months later.

A cardiac evaluation, including echocardiography and 24-hour Holter monitoring, revealed no source of embolism. Carotid ultrasonography demonstrated hyperechoic plaque in the origin of the bilateral internal carotid artery; however, no significant stenosis was observed. We suspected that thrombi arising in the stenotic MCA had induced artery-to-artery embolism affecting the distal cortical branches, although the exact etiology was not determined. The patient was treated conservatively with acetylsalicylic acid (100 mg/day) and exhibited a good outcome with the complete resolution of aphasia within one week. No further ischemic episodes have occurred during nine months of follow-up.

Case 2

An 81-year-old woman with a history of hypertension and hyperlipidemia presented to our hospital with the sudden onset of right-sided weakness and slurred speech. On admission, she was alert and oriented, although dysarthria, right facial palsy and dense right-sided hemiparesis were noted. The remainder of the neurological examination was normal; the NIHSS score was 8.

Although diffusion-weighted MRI performed on admission revealed no acute abnormalities, MRA demonstrated a segment of signal loss in a vessel arising from the proximal A1 portion of the left ACA. This vessel ran parallel to the ipsilateral MCA, although it was slightly thinner in diameter. A diagnosis of ischemic stroke due to poor perfusion caused by an AMCA was made, and the patient received intravenous thrombolysis with tissue plasminogen activator 120 minutes after the onset of the stroke. Follow-up MRI performed the next day showed left striatocapsular, centrum semiovale and frontal cortical infarcts (Fig. 2A), as well as occlusion of the AMCA (Fig. 2B). MRA was performed on day 5 of admission, which demonstrated a recanalized AMCA supplying the territory of the orbitofrontal, prefrontal, precentral and central arteries (Fig. 2C).
The levels of biochemical parameters and blood cell counts were unremarkable. Despite conducting an extensive cardiac evaluation, no potential source of embolism was documented. Carotid ultrasonography found atherosclerotic plaque in the proximal left internal carotid artery without significant stenosis. Although the cause of the patient’s stroke was unknown, she received anticoagulation with heparin and warfarin. Her condition gradually improved over the next four weeks until she was able to walk independently, with an NIHSS score of 4.

**Discussion**

In 1962, Crompton first used the term “AMCA” to refer to an anomalous artery arising from the ICA that followed a parallel course to the MCA and supplied part of the MCA territory (1). Subsequently, the term has been applied to similar arteries arising from either the ICA or ACA. In 1973, Teal et al. proposed using the term “AMCA” to describe an anomalous artery arising from the ACA and the term “duplication of the MCA” to describe a vessel arising from the distal ICA between the anterior choroidal artery and the terminal bifurcation of the ICA (2). Teal’s terminology is now widely accepted in the current literature on MCA variations. Although AMCA s are usually thinner than the main MCA, it can be difficult to determine whether an anomalous artery is an AMCA or represents duplication of the MCA in cases in which the artery originates from the neighborhood of the ICA terminal bifurcation and the caliber of both arteries is approximately equal.

Concerning the developmental origin of AMCA, Handa et al. (11) proposed that such vessels are variants of the recurrent artery of Heubner (RAH); however, other authors disagree, as a) the AMCA and RAH usually coexist independently of each other and b) the AMCA has a different course and supplies a different territory than the RAH. Owing to the similar cortical supply, Komiyama et al. (5) suggested that an AMCA represents an anomalous early ramification of the early branch of the MCA that reaches the anterior frontal lobe. The brain regions supplied by AMCA s usually include the territory of the orbitofrontal and/or prefrontal arteries. It has been reported that additional areas supplied by AMCA s include the territory of the precentral, central and/or anterior parietal arteries (4, 6, 9).

The clinical significance of AMCA s in the setting of acute MCA territory infarction remains obscure. Jain reported that there is abundant anastomosis on the cortical surface between the branches of the AMCA and the main MCA (3). Therefore, there is considerable potential for AMCA s to supply a collateral flow to the MCA territory. In fact, Mueller et al. reported a case of MCA occlusion in which an AMCA served as a well-developed collateral pathway (7). Similarly, a large portion of the MCA territory was rescued from ischemia in the present case 1 because blood was supplied by the AMCA, likely with some assistance of leptomeningeal collaterals originating from the ACA and/or the posterior circulation. On the other hand, Komiyama et al. reported two cases of MCA territory infarction due to concomitant embolic occlusion of the main MCA and distal ICA in patients with ipsilateral AMCA s that could not provide an adequate blood flow to rescue the MCA territory (8).

The occurrence of cerebral infarction due to AMCA occlusion has rarely been described (9, 10). In past reports, it has been suggested that the interruption of an AMCA may result in serious neurological deficits, particularly in the dominant hemisphere (4, 9). In the present case 2, the AMCA supplied a relatively large part of the MCA territory, and dense hemiparesis was noted on admission. On the other hand, the patient regained independence in walking, although it is unclear to what extent intravenous thrombolysis influenced this functional outcome. We consider that the severity of neurological symptoms depends on how much of the MCA territory is supplied by the anomalous vessel. To date, there is little information available concerning functional outcomes after cerebral ischemia due to AMCA occlusion.

Recently, Menon et al. described a case of MCA territory infarction due to ipsilateral AMCA occlusion, in which the proximal stump of the occluded AMCA was misdiagnosed as an area of aneurysmal dilatation on CT angiography (10). The authors suggested that the presence of AMCA occlusion is likely to be overlooked if the arterial stump is indistinct or exhibits the characteristics of an aneurysm.
In conclusion, the present two cases emphasize that knowledge of this rare anatomic variation is important when interpreting neuroimaging data during the hyperacute phase in patients with MCA territory stroke. However, further investigations are required to verify the clinical significance of AMCAs in patients with ischemic stroke.

The authors state that they have no Conflict of Interest (COI).

References


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