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Case Report

Unusual hemodynamic stroke related to an accessory middle cerebral artery: The usefulness of fusion images from three-dimensional angiography

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Abstract

Background: Ischemic stroke associated with an anomaly of the middle cerebral artery (MCA) is a rare occurrence. The diagnosis is very difficult when there are steno-occlusive lesions associated with an accessory middle cerebral artery (AMCA).

Case Description: A 77-year-old female with hypertension and hyperlipidemia experienced repeated transient ischemic attacks (TIAs) of motor aphasia and dysarthria. Although angiography showed only left intracranial occlusion, the fusion images of three-dimensional digital subtraction angiography (3-D DSA) showed complex steno-occlusive lesions and an AMCA related with the TIA. The cerebral blood flow (CBF) to the left frontal lobe was supplied by the AMCA, via the anterior communicating artery from the right internal carotid artery. The left temporal and parietal lobes were supplied by the stenotic MCA, via the left posterior communicating artery from the left posterior cerebral artery. Single-photon emission computed tomography showed a marked decrease in CBF to both the left frontal and temporal lobes. A left superficial temporal artery (STA)-to-left MCA double anastomosis was performed, in which each branch of the STA supplied branches of the AMCA and MCA.

Conclusion: This is the first reported case of ischemic stroke in a patient with an AMCA. The exact diagnosis could be made only by using fusion images of 3-D DSA, which were useful for understanding the complicated CBF pattern and for the choice of recipient artery in bypass surgery.

Key Words: Accessory middle cerebral artery, revascularisation, transient ischemic attack, 3D-DSA

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INTRODUCTION

Intracranial vascular anomalies involving the middle cerebral artery (MCA) are rare. Among this group, an accessory middle cerebral artery (AMCA) has been reported to have an incidence of approximately 0.3-4%.[3,7,12,17,18] This anomalous vessel originates from the anterior cerebral artery (ACA)[14] and runs through

the Sylvian fissure along with the MCA.[6] There are numerous reports of AMCAs that focus on the association of the AMCA with cerebral aneurysm, but only a few cases have involved an ischemic event.[2,5,9,16,19] This report describes the first case of surgical intervention for transient ischemic attack (TIA) due to a hemodynamic mechanism associated with an AMCA. Fusion images of three-dimensional angiography were the only way of assessing the complex CBF pattern and choosing the method of surgical treatment in this case.

CASE REPORT

A 77-year-old female experienced repeated TIAs of motor aphasia and dysarthria. On admission, she was alert and well oriented. She had no cranial nerve deficits or focal neurological signs. Magnetic resonance (MR) imaging revealed no evidence of acute cerebral infarction [Figure 1a]. MR angiography showed an occlusion of the left internal carotid artery (ICA) and stenosis of the right MCA [Figure 1b and c]. Some anomalous arteries were also identified faintly on MR angiography and ordinary angiography, but the detailed anatomy was not clear. Fusion images of three-dimensional digital subtraction angiography (3D-DSA) demonstrated an occlusion of the cervical portion of the left ICA and stenosis of both the proximal segments (M1) of the MCA [Figure 2]. In addition, a left AMCA, originating from near the anterior communicating artery (ACoA), was identified [Figures 2 and 3].

Three-dimensional DSA revealed the characteristic cerebral blood flow (CBF) supply pattern. The AMCA supplied CBF to the frontal lobe alone. The AMCA was perfused by the right ICA via the ACoA. In addition, the left MCA, which was perfused by the left posterior cerebral artery via the left posterior communicating artery, supplied CBF to the temporal and parietal lobes [Figures 2-4]. Because the proximal segment (A1) of the left ACA was occluded, the flows of the AMCA and MCA were clearly separate [Figure 4]. Single-photon emission computed tomography (SPECT) showed a marked decline in the CBF to the left fronto-temporo-parietal lesion at rest, while cerebral vascular reactivity was impaired by acetazolamide loading [Figure 5]. After diagnosis, we started antiplatelet medication (ASA 100 mg/day) and it continued until bypass surgery.

It was concluded that bypass surgery was needed for the territory of both the AMCA and MCA. A double anastomosis was performed that consisted of: (1) the frontal branch of the left superficial temporal artery (STA) to the cortical artery of the left AMCA; and (2) the parietal branch of the STA to the cortical branch of the left MCA. Prior to anastomosis, intraoperative indocyanine green video angiography was performed and demonstrated bidirectional flow in the left frontal cortical artery [Figure 6]. After anastomosis, the flow in the frontal cortical artery improved. A remarkable improvement in CBF in all lesions was confirmed postoperatively. The patient was discharged with no neurological deficits and remained free from ischemic attacks without antiplatelet medication for at least 1 year.

DISCUSSION

Intracranial vascular anomalies involving the MCA are relatively rare. There are two known types of double-M1

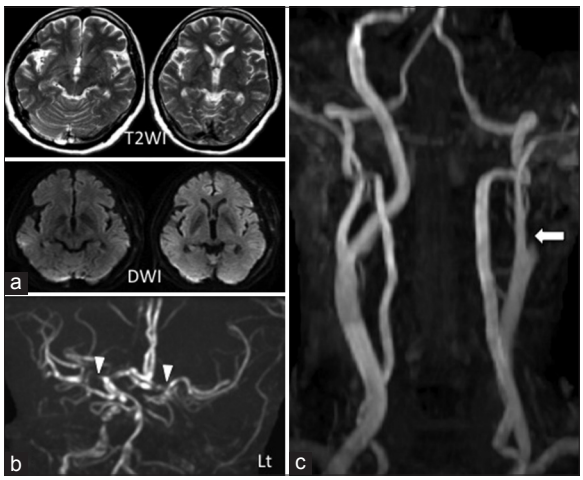

Figure 1: Magnetic resonance (MR) imaging on admission showed no acute infarction (a) T2WI, T2-weighted images; DWI, diffusion weighted images. MR angiography showed an occlusion of the left internal carotid artery (b and c, arrow) and stenosis of the bilateral middle cerebral artery (arrowhead). Some anomalous arteries were also suspected (b), but the detailed anatomy was unclear

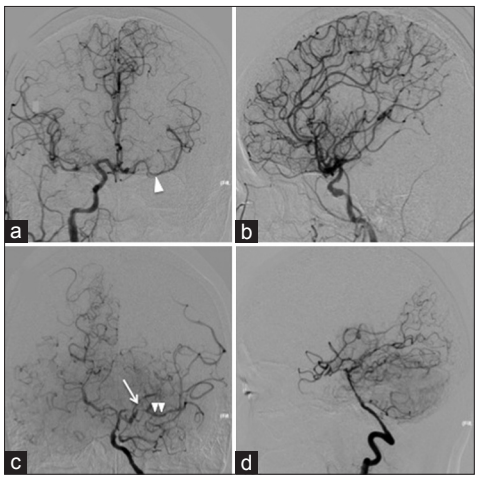

Figure 2: Conventional angiography of the right internal carotid artery (a and b) and left vertebral artery (c and d). Anterior-to-posterior view (a and c) and lateral view (b and d). The arrow (c) indicates stenosis of the left proximal segment (M1) of the middle cerebral artery (MCA), which is supplied via the left posterior communicating artery. Note that the left accessory MCA supplied only the left frontal lobe (arrowhead in a and b), while the left MCA supplied the area beneath the Sylvian fissure (arrowheads in c and d)

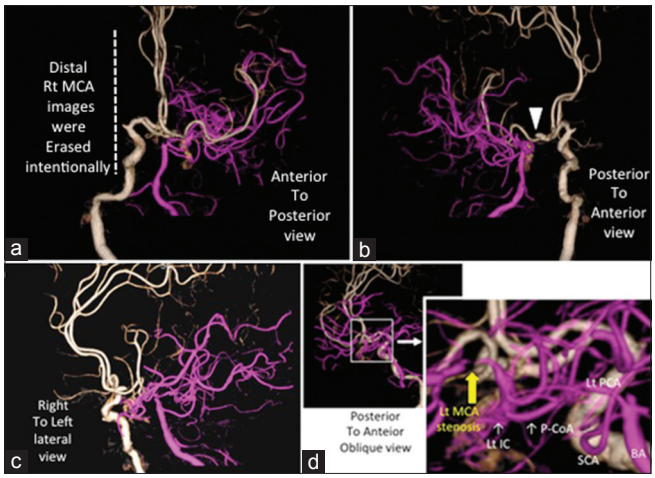

Figure 3: Fusion images of three-dimensional angiography of the right internal carotid artery (brown) and left vertebral artery (red). To clearly illustrate the distal part of the middle cerebral artery (MCA), the right side of all images was intentionally eliminated. The left accessory MCA and left MCA run independently, and the areas they supply are clearly distinguished. The arrowhead (b) indicates the site of occlusion of the left anterior cerebral artery. The stenosis of the left MCA is indicated by the arrow (d)

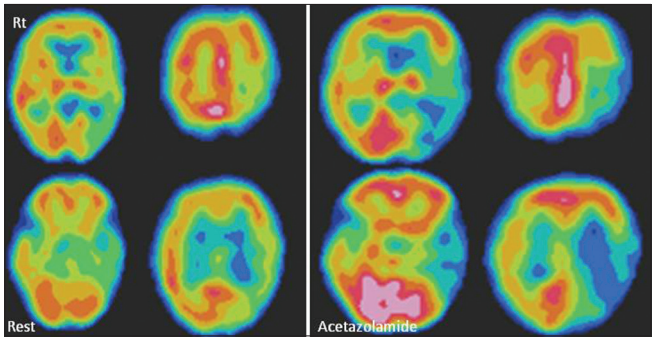

Figure 5: Single-photon emission computed tomography showed a marked decline in cerebral blood flow at the left fronto-temporo-parietal lesion, and cerebral vascular reactivity was impaired by acetazolamide loading

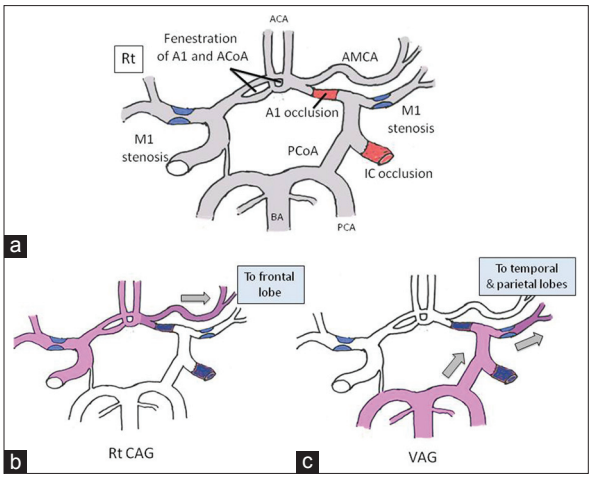

Figure 4: Schematic drawing of the anomalous arteries and steno-occlusive changes found in this patient, showing the blood flow in the right internal carotid artery angiography (b) and the left vertebral artery angiography (c) as seen on Figures 2 and 3

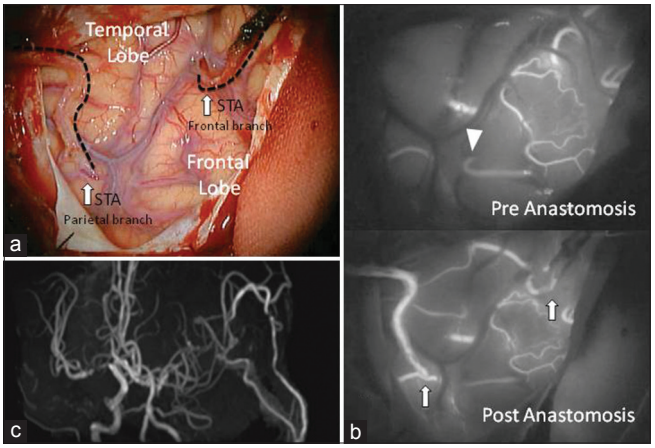

Figure 6: The intraoperative findings (a and b) of the left superficial temporal artery-to-middle cerebral artery double anastomosis and magnetic resonance angiography after operation (c). Intraoperative indocyanine green video angiography showed the time delay of blood flow and bidirectional flow. The arrows indicate the anastomosis sites (a and b)

segments of the MCA, including a duplicated middle cerebral artery (DMCA) and an AMCA. Teal *et al.*[15] classified the DMCA and AMCA according to the point of origin: a DMCA arises from the ICA at the proximal point of bifurcation, whereas an AMCA arises from the proximal portion of the ACA or the distal portion of the A1 segment of the ACA near the ACoA. Gibo *et al.*[4] and Komiyama *et al.*[8] analysed the cortical territories of the DMCAs and AMCAs. A DMCA feeds the temporopolar territory and the anterior and middle temporal areas, while an AMCA feeds the orbitofrontal and prefrontal areas. However, the AMCA described in this case perfused a broader area, including the orbitofrontal, prefrontal, and precentral areas. In some cases, it is difficult to distinguish a DMCA from an AMCA, and it is important to clarify the relationship between A1 of the ACA and the abnormal MCA. However, in this case, A1 of the ACA was occluded, making it difficult to clarify whether this anomaly represented an AMCA or A1 of the ACA. Because the occluded artery had a similar appearance to A1 of the ACA and because the anomalous artery supplied CBF only to the frontal area, it was considered to be an AMCA.

The clinical presentation in this case likely represents a TIA due to a hemodynamic mechanism. There were three stenotic lesions contributing to cerebral ischemia: (1) occlusion of the left ICA, (2) occlusion of the ipsilateral A1 of the ACA, and (3) severe stenosis of the ipsilateral MCA. The stenosis in the left MCA caused the decrease in blood flow, while the occlusion of A1 of the ACA reduced the blood flow through the AMCA. Together, these obstructions resulted in a decrease in blood flow through the MCA [Figure 4], which corresponded to

the SPECT findings that revealed a marked decline of CBF in the left fronto-temporo-occipital region. Because there was no stenotic lesion at AMCA, which contributed in ischemic attack and the CBF of AMCA territory decreased, we concluded that TIA attack were occurred due to a hemodynamic mechanism.

AMCA is well-established anomalous artery originates from the ACA.[14] In general it has no other vascular anomaly and is sometimes found in healthy human. Embryologically, the MCA develops after the ACA, and the ACA is considered a continuation of the primitive ICA.[1,7,10] An AMCA could provide collateral blood supply.[8] However, in this case, the AMCA could not supply sufficient collateral blood flow, because of the occlusion of the A1 of the ACA. We can see Moyamoya artery like collaterals around the stenotic lesion on the right side, but cannot see on the left side [Figure 2]. First we have diagnosed these stenotic lesions were atherosclerotic lesions, because she has hypertension and dyslipidemia, and right ACA and AMCA has no stenosis. However, it is possible that this case has Moyamoya disease like pathology, because she has a rare vascular anomaly and has many stenotic lesisons.

Because the frontal and temporal areas clearly had separate blood supplies in this case and only CBF decrease could explain this patient's symptoms, an STA–MCA double bypass was performed to the frontal and temporal areas. Furthermore this patient is free from ischemic attack without antiplatelet medication. Now EC-IC bypass surgery was not accepted in worldwide evidence, however, there are some studies supporting the effectiveness of EC-IC bypass in Japan,[11,13] and Japanese neurosurgeons consider that EC-IC bypass is very effective and safety. Furthermore, this is a special case with complicated anomalies and many stenotic lesions. Then we performed double bypass and obtained excellent postoperative course without antiplatelet medication. To our knowledge, this is the first reported case of ischemic disease with an AMCA. Due to the rare presentation, it was difficult to clearly appreciate the relationship between the anomalous arteries and the ischemic lesions by CT angiogram or MR angiogram. When detailed vascular anatomy is needed, conventional angiography is superior to the CT and MR angiograms in some cases. In fact, we did not notice these abnormalities at first by simple observation of each 3D-DSA image. Fusion images from 3D-DSA were of significant value in clarifying these relationships.

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