

Middle Cerebral Artery Anomalies Detected by Conventional Angiography and Magnetic Resonance Angiography

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Objective : Middle cerebral artery(MCA) anomalies are found incidentally on conventional cerebral angiography and magnetic resonance angiography(MRA). Our goal is to examine the incidence and types of MCA anomalies.

Methods : Cerebral angiography was performed in 448 patients and MRA in 743; the patients had or were suspected to have cerebrovascular disease. The images were retrospectively evaluated for arterial anatomic anomalies. We use Teal's classification for definition of accessory and duplicated MCAs.

Results : On cerebral angiography, the following anomalies of the MCA were found in seven patients : fenestration (n = 2, incidence = 0.45%); duplication (n = 2, incidence = 0.45%); accessory MCA (n = 2, incidence = 0.45%); aplasia (n = 1, incidence = 0.22%). On MRA, eight patients had anomalous MCAs : fenestration (n = 1, incidence = 0.14%); duplication (n = 6, incidence = 0.81%); accessory (n = 1, incidence = 0.14%).

Conclusion : Although the clinical significance is not great, we find a relatively high incidence of anomalous MCAs. Knowledge and recognition of these MCA anomalies are useful and important in the interpretation of cerebral images and during neurosurgical procedures.

KEY WORDS : Middle cerebral artery · Vascular anomalies · Cerebral angiography · Magnetic resonance angiography.

Introduction

It is of paramount importance that the vascular neurosurgeon be thoroughly conversant with the normal anatomy of the basal arteries of the brain and their cortical distributions, as well as with that of the perforating systems. Many common abnormalities of these vascular systems are recognized both angiographically and at surgery, and may take the form of aplasia, hypoplasia, and duplications. Anomalies of the MCA are seen less frequently than anomalies in the other major intracranial arteries²³. The purpose of our study was to examine the incidence and types of MCA anomalies detected by conventional angiography and MRA.

Materials and Methods

Conventional cerebral angiography (Philips V-5000, Philips Medical Systems, Eindhoven, Netherlands) was performed

in 448 patients (July 1997 and February 2004) and cranial MRA (1.5T, Signa MR/i, General Electric, Milwaukee, WI, USA.) in 743 (May 2001 and February 2004). The MRA and angiography were undertaken for a variety of clinical reasons, including symptoms of cerebral ischemia, cerebral infarction, hemorrhagic contusion, intracranial hemorrhage, and headache. For the MRA studies, a three-dimensional time-of-flight technique with a neurovascular phased array coil (MRI devices, Milwaukee, Wis., USA.) and a multiple overlapping thin slab acquisition technique were used. The following imaging parameters were selected : repetition time = 30ms; echo time = 6.9ms; field of view = 26×26cm; number of slices = 108-112; slice thickness = 1.6mm; slab thickness = 24-28mm; imaging matrix = 256×192; number of excitation = 1. No intravenous paramagnetic contrast agent was administered to any of the patients. In each patient, a total of 20 maximum-intensity projection(MIP) images in the frontal view (both from left lateral to right lateral, 180° and craniocaudally 180° were routinely displayed stereoscopically. Both cerebral angiography and MRA were performed in 53 of the patients. The images were obtained either from a routine diagnostic study or from the initial diagnostic part of an interventional procedure. All the angiograms and MRAs were evaluated retrospectively for cerebral arterial anatomic anomalies by one of the authors (MS Kim). In patients with anomalies in the MCA, special attention

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MCA Anomalies

was given to defining the origin and size of the anomalous vessel as well as its course. We also recorded the presence of associated vascular lesions, including cerebral aneurysm, and vascular stenosis or occlusion. Teal's²¹⁾ definitions of the accessory and duplicated MCA are widely accepted and were used in this study.

Results

Fifteen patients (five men, ten women, 9 to 74 years of age, median age = 60 years) had a MCA anomaly. Those patients that underwent both conventional cerebral angiography and cranial MRA showed no MCA anomalies.

Seven (1.56%) of the patients undergoing cerebral angiography had an anomalous MCA; two had an accessory MCA, two a fenestration of the MCA, two a duplicated MCAs, and one aplasia of the MCA. Four aneurysms, one probable case of moyamoya disease, and one stenosis of internal carotid artery were associated with the anomalous vessels (Table 1).

Eight patients (1.08%) among 743 patients that underwent MRA demonstrated anomalies of the MCA; six had duplicated MCAs, one an accessory MCA, and one a fenestrated MCA. Associated vascular anomalies included a primitive trigeminal artery and a duplication of an anterior cerebral artery (Table 2).

Fenestration of the MCA

Two fenestrated MCAs were detected in conventional angiographic study group and one in MRA group : the incidence was 0.45% (2/448) and 0.14% (1/743), respectively. In each patient, the origin of the fenestration was at the proximal portion of the main trunk of the MCA. Owing to poor spatial resolution of MRA, temporo-polar artery(TPA) was not visualized in the MRA of one patient with a fenestrated MCA. In the other two cases, the TPA was visualized with conventional angiography; the distances between the origin of the TPA and the internal carotid artery bifurcation were 5.8 and 7.8mm. Associated

Table 2. Summary of eight patients with MCA anomalies detected by magnetic resonance angiography

Case	Age/Sex	MCA anomaly	Associated vessel anomaly	Symptom
1	F/74	MCA fenestration	Trigeminal artery	TIA
2	M/9	Accessory MCA	No	Hemorrhagic confusion
3	F/60	Duplicated MCA	No	Headache
4	M/60	Duplicated MCA	No	TIA
5	F/70	Duplicated MCA	No	TIA
6	F/49	Duplicated MCA	No	TIA
7	M/57	Duplicated MCA	A1 duplication	Infarction
8	M/17	Duplicated MCA	No	TIA

sMCA : middle cerebral artery, TIA : transient ischemic attack, A1 : proximal anterior cerebral artery

anomalies included an aneurysm, a primitive trigeminal artery, and a stenosis of the internal carotid artery. In one patient underwent cerebral angiography, an early branching TPA was seen; it arose from the inferior limb of the fenestrated segment (Fig. 1).

MCA duplication

Two MCA duplication were detected in conventional angiographic group and six in MRA group; the incidence was 0.45% (2/448) angiographic study and 0.81% (6/743) on MRA study. Five of the duplicated MCAs were right sided and three were left sided. The duplicated MCAs had a smaller diameter than the main MCAs in three patients; however, the main MCAs and the duplicated MCAs were of similar diameter in five patients. The duplicated MCA coursed in the Sylvian fissure with an anterior sharp curve to the temporal lobe in six patients, or coursed parallel to the horizontal portion of the MCA in two patients (Fig. 2). Associated vascular anomalies were an aneurysm of the contralateral MCA bifurcation and a duplication of A₁.

Accessory MCA

Two accessory MCA were detected in conventional angiographic group and one in MRA group; the incidence was 0.45% (2/448) on cerebral angiography and 0.14% (1/743) on MRA.

The accessory MCAs originated from the proximal A₁ portion of the anterior cerebral artery in one patient and from the distal A₁ segment (near the anterior communicating artery) in two patients. In each patient, the diameter of the accessory MCA was narrower than that of the anterior cerebral artery and the main MCA. Each accessory MCA originated from the left anterior

Table 1. Summary of seven patients with middle cerebral artery anomalies detected by transfemoral cerebral angiography

Case	Age/Sex	MCA anomaly	Associated vessel anomaly	Symptom
1	F/46	left Accessory MCA	no	headache
2	F/18	left Accessory MCA	Probable moyamoya disease	TIA
3	M/62	left MCA fenestration	ICA stenosis	infarction
4	F/62	right MCA fenestration	A-com aneurysm	SAH
5	F/38	left MCA duplication	right MCA aneurysm	SAH
6	F/60	left MCA duplication	no	SAH
7	F/64	left MCA aplasia	Proximal ACA aneurysm, A-com aneurysm	SAH

MCA : middle cerebral artery, ICA : internal carotid artery, TIA : transient ischemic attack, SAH : subarachnoid hemorrhage, A-com : anterior communicating artery, ACA : anterior cerebral artery

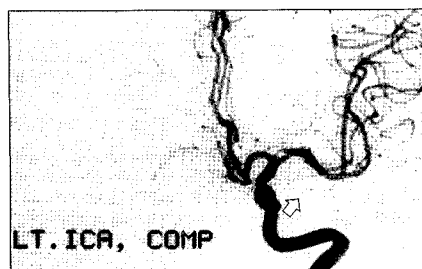


Fig. 1. Left carotid angiogram showing fenestration of the proximal middle cerebral artery with an early branching temporo-polar artery (arrow) arising from the inferior segment of the fenestrated segment.



Fig. 2. Magnetic resonance angiography showing a right duplicated middle cerebral artery running parallel to the M1 segment.

unruptured proximal anterior cerebral artery aneurysm. In this case, a MCA aplasia was found during surgery. And the cord-like rudimentary MCA had no internal blood flow (Fig. 4).

Discussion

Fenestration of MCA

A fenestrated MCA is a rare anatomic anomalies, with an incidence of 1% on anatomic dissection and 0.17% on cerebral angiography^{17,23}. It is probable that the morphology of a cerebral artery fenestration would be detected more reliably by direct anatomic dissection than by a clinical imaging system or by direct observation of a limited surgical field. Therefore, the frequency of 1% reported by Umansky et al²³ is likely to represent the real incidence of MCA fenestration. In another large angiographic study (5190 patients; Sanders et al¹⁷), nine cases were detected, an incidence of 0.17%, compared with 0.45% in this study. In a previous MRA investigation of 425 patients (Uchino et al²²), the incidence of MCA fenestration was 0.47%, compared with 0.14% in our study. Okudera et al¹⁵ classified fenestrations of the M₁ portion into three types : the 'proximal type' at the proximal portion of M₁, the 'intermediate

cerebral artery. A recurrent artery of Heubner was observed in one patient (Fig. 3), and, in another patient, the accessory MCA was associated with probable moyamoya disease.

MCA aplasia

On cerebral angiography, aplasia of the MCA was detected in one patient; an incidence of 0.22% (1/448) on cerebral angiography. The patient received surgical treatment of associated ruptured anterior communicating artery aneurysm and

type' at the center portion of the M₁, and the 'distal type' at the portion just before the division of M₂. They reported that the proximal type was most common. And our three cases were of the proximal type.

On the 35th day of embryonic development, the primitive MCA forms a plexus with the internal carotid artery. The fenestration of the MCA is thought to be a persistence of the plexus¹³, and thus fenestration of the MCA occurs more frequently proximally than distally. However, Gailloud et al⁴ have suggested that early branching of the TPA may lead to the formation of the fenestration by interfering with the normal fetal development of the MCA. The distance between the origins of the TPA and the internal carotid artery bifurcation has been evaluated by Umansky et al²⁴; the mean values were 7.5mm for the right side and 7.4mm for the left side. In another study (Gailloud et al⁴), the distances between the internal carotid artery bifurcation and the origin of the TPA were evaluated by using the caliber of the cisternal segment of the internal carotid artery as a reference (4.1mm according to the study presented by Gibo et al in 1981⁵). Gailloud et al⁴ reported that, in their five cases of MCA fenestration, the TPA arose from the fenestrated segment itself and the distance between its origin and the internal carotid artery bifurcation had a mean value of 5.0mm; in our two patients, the

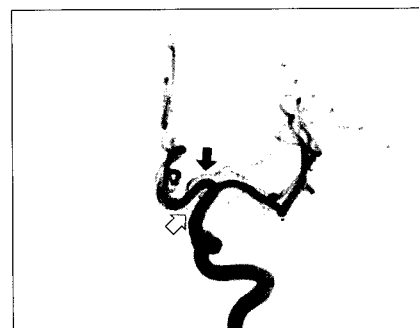


Fig. 3. Left carotid angiogram showing an accessory middle cerebral artery (black arrow) originating from the distal part of the horizontal portion of the anterior cerebral artery. Note also the recurrent artery of Heubner (white arrow).

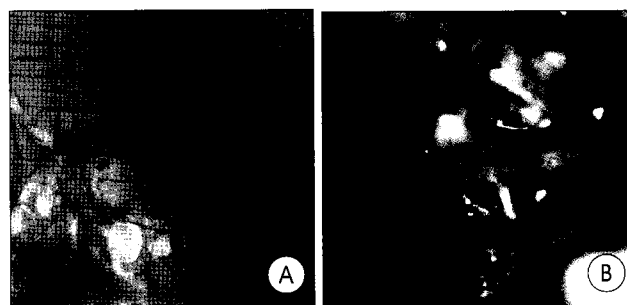


Fig. 4. A : Left carotid angiogram demonstrating absence of a typical T-shape at the internal carotid artery bifurcation, two aneurysms located at the proximal anterior cerebral artery (open arrow) and an anterior communicating artery (black arrow). B : Intra-operative findings showing a cord-like rudimentary structure at the site of internal carotid artery bifurcation (arrow).

distances were 5.8 and 7.8mm. In one case, the early branching TPA was demonstrated (Fig. 1).

MCA fenestration has no clinical significance, but a rare aneurysm can be seen at the proximal end of the fenestration²⁾. Actually, there does not seem to exist any clinical significance of arterial fenestrations other than the frequent association with various abnormalities. The discovery of a fenestrated MCA is usually an incidental finding, either during an angiography or during an operation, performed for another pathology²⁾.

MCA duplication

In 1973, Teal et al²¹⁾ proposed using the term 'MCA duplication' to characterize the two vessels originating from the distal end of the internal carotid artery, and the term 'accessory MCA' to describe the anomalous vessel originating from the anterior cerebral artery. Their frequency of MCA duplication was 0.2% to 2.9%, compared with our frequencies of 0.45% in cerebral angiograms and 0.81% on MRA. An association between aneurysms and MCA duplication has been reported^{3,9,18,22)}. Some investigators have considered the etiology to be related to congenital factors¹¹⁾, and others regard the association as purely coincidental^{3,16)}. In our study, only one aneurysm (located at the contralateral MCA bifurcation) was associated with an MCA duplication.

Komiyama et al¹⁰⁾ reported that a duplicated MCA is embryologically an anomalous early ramification of the early branch of the MCA, which originates from the distal end of the internal carotid artery. The duplicated MCA consistently supplies the anterior temporal lobe. It may have branches (perforating arteries) and there may also be an associated recurrent artery of Heubner. Similarly, Komiyama et al¹⁰⁾ proposed that the accessory MCA is an anomalous early ramification of the early branch of the MCA, which originates from the A₁ portion of the anterior cerebral artery. The accessory MCA consistently supplies the anterior frontal lobe. Knowledge of the anomalous ramification of the MCA is important for the surgical treatment of cerebral aneurysm and for understanding the collateral blood supply in patients with cerebral ischemia.

Accessory MCA

Since it was first proposed by Teal et al²¹⁾ in 1973, the term accessory MCA has generally been restricted to an anomalous artery that arises from the anterior cerebral artery to supply the cortex (the region normally supplied by the MCA); a branch arising from the internal carotid artery has been called a duplication of MCA^{8,21)}. In previous angiographic and anatomic observations, the frequency of the accessory MCA was reported to be 0.3% to 4.0%¹⁰⁾, and in MRA studies of 425 patients, the

incidence was 1.25% (Uchino et al²²⁾); in our study, the incidences were 0.45% in conventional angiograms and 0.14% on MRA.

Handa et al⁷⁾ suggested that the accessory MCA is a variant form of the recurrent artery of Heubner. However, this hypothesis is disputed for the following reasons^{18,21)}: the perforating arteries only occasionally originate from the accessory MCA; the recurrent artery of Heubner coexists with the accessory MCA; and the recurrent artery of Heubner enters more medially to the anterior perforated substance than the accessory MCA. Takahashi et al²⁰⁾ proposed that both the recurrent artery of Heubner and the accessory MCA represent persistent anastomoses between the anterior cerebral artery and the MCA over the tuberculum olfactorium. In our study, one patient had recurrent artery of Heubner and an accessory MCA on the same side. Umansky et al²³⁾ reported that the cortical distribution of accessory MCA was in the region of the orbito-frontal, central and precentral arteries. Thus, interruption of an accessory MCA will lead to a severe neurological deficit, particularly in the dominant hemisphere¹⁹⁾. Despite its small size, the importance of collateral flow in the accessory MCA was shown by Mueller et al¹²⁾; in a 15-month-old patient, surgical treatment of a left MCA aneurysm resulted in complete occlusion of the MCA trunk. Subsequent angiography demonstrated that, in addition to anterior and posterior cerebral arterial cortical anastomoses, an accessory MCA contributed significantly to perfusion in the region supplied by the MCA. On a follow-up examination, the patient was neurologically normal but had a small lenticular infarct.

MCA aplasia

Previous anatomical studies have identified various anomalies and variations of the MCA, but aplasia of the vessel has only been previously described in three patients^{1,6,14)}. These patients were similar to our patient and had an associated aneurysm of the proximal anterior cerebral artery. Hemodynamic stress is well recognized as a causative factor in the initiation and growth of intracranial saccular aneurysms. Amagasaki et al¹⁾ reported that, to maintain an adequate blood flow, aplasia of the MCA results in widening of the vessel at the level of its branching from the proximal anterior cerebral artery, and the resultant hemodynamic stress leads to the growth of the aneurysm. In such patients, cerebral perfusion could be evaluated by single photon emission computed tomography. However, in two patients, investigation did not demonstrate hypoperfusion in the region supplied by a MCA aplasia^{1,6)}. Further studies of patients with aplasia of the MCA may increase our understanding of cerebral vascular anatomy and cerebral hemodynamics.

Conclusion

MCA anomalies are observed relatively rarely on conventional cerebral angiograms and cranial MRA. Although they hold little clinical significance, knowledge and recognition of these MCA anomalies is important in the interpretation of cerebral images and during neurosurgical procedures.

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