Acute middle cerebral artery stroke in a patient with a patent middle cerebral artery

Jamie Cooke, MBBS, Julian Maingard, MBBS, Ronil V. Chandra, MBBS, MMed, FRANZCR, CCINR, Lee-Anne Slater, MBBS, FRANZCR, CCINR, Mark Brooks, MBBS, FRANZCR, EBIR, CCINR, and Hamed Asadi, MD, PhD, FRANZCR, EBIR, CCINR

Correspondence

Dr. Cooke
jamiepaulcooke@gmail.com

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Abstract

Purpose of review

Knowledge of cerebrovascular anatomical variants is vital for clinicians working with patients presenting with signs and symptoms of cerebral infarction, particularly in the era of endovascular clot retrieval.

Recent findings

We provide an overview of a cerebrovascular anatomical variation and detail a patient presenting with cerebral infarction secondary to occlusion of their anomalous vessel who underwent successful endovascular clot retrieval with excellent functional outcome. We also include technical descriptions.

Summary

Given the clinical importance of the areas supplied by the accessory middle cerebral artery, knowledge of this vessel is not only important for diagnosis but also for neurosurgical or endovascular management of patients with this variant.

Knowledge of cerebrovascular anatomical variants plays an important role in both the diagnosis and management of patients presenting with signs and symptoms of cerebral infarction, particularly in the era of endovascular clot retrieval (ECR). Failure to recognize normal variants may result in a missed diagnosis or compromised management. In this review, we describe the successful ECR in a patient having acute ischemia in the left middle cerebral artery (MCA) territory with a patent MCA on cross-sectional angiographic imaging.

Case report

A 37-year-old right-hand-dominant woman, with a known atrial septal defect, presented to the emergency department after being found with an altered conscious state. On arrival, she was found to be alert and responsive but had a dense right-sided hemiplegia and an expressive dysphasia in keeping with a left MCA syndrome.

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[Page 185](#)

Case

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[Page 279](#)

From the Anatomy and Neuroscience (JC), School of Biomedical Sciences, University of Melbourne, Parkville; Department of Surgery (JC), Alfred Hospital, Melbourne, Victoria; Interventional Radiology Service (JM, MB, HA), Department of Radiology, Austin Hospital, Melbourne; School of Medicine (JM, MB, HA), Faculty of Health, Deakin University, Waurn Ponds; Stroke Division (JM, MB, HA), Florey Institute of Neuroscience and Mental Health, University of Melbourne, Heidelberg, Victoria; Interventional Neuroradiology Service (HA), Department of Radiology, St Vincent's Hospital; Interventional Neuroradiology Unit (RVC, L-AS, HA), Monash Imaging, Monash Health; and Faculty of Medicine (RVC, HA), Nursing and Health Sciences, Monash University, Melbourne, Australia.

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Initial noncontrast brain CT revealed a small volume of established ischemic changes within the left frontal lobe with a loss of gray-white differentiation (figure 1A) and hyperattenuating intravascular thrombus (figure 1B) suggesting a left-sided cerebral infarction secondary to occlusion of the left MCA. This was thought to have been confirmed on subsequent perfusion imaging (figure 1, C and D), which demonstrated an extensive ischemic penumbra within the territory of the left MCA with suggestion of ischemic mismatch.

The diagnosis of a proximal MCA occlusion was questioned, however, when CT angiography revealed normal contrast opacification in a horizontal middle cerebral arterial vessel and the distal cortical branches (figure 1, E and F). This finding was inconsistent with the clinical diagnosis and prompted a search for alternative causes to explain the discordant clinical and imaging findings.

The patient was subsequently transferred to the angiography suite where selective digital subtraction angiography (DSA) was performed. This confirmed patency of a horizontal middle cerebral arterial vessel and the distal cortical branches as identified on the CT angiography (figure 2A). However, there was absence of superior division cortical branches and the lenticulostriate vessels (figure 2, B and D). This prompted a detailed search and identification of a small stump arising from the proximal left anterior cerebral artery (ACA) (figure, 2 A and B, red arrows).

The stump was probed with a Synchro2 0.014-inch micro-wire (Stryker, Fremont, CA), which passed through the irregularity and into a sylvian cortical vessel. This confirmed the angiographic diagnosis of an occluded accessory MCA (aMCA), and the patient underwent ECR. A Trevo 0.021-inch microcatheter (Stryker, Fremont, CA) was then advanced beyond the occlusion into the distal branches of the aMCA with a subsequent DSA via the microcatheter confirming the suspected missing branches on the initial angiogram (figure 3, A and B). A single pass with a 4 × 40-mm Solitaire Stentriever (Medtronic, Irvine, CA) retrieved the occluding thrombus. Complete reperfusion (thrombolysis in cerebral infarction scale [TICI] 3) of the aMCA was achieved within 10 minutes of groin puncture (figure 3, C and D).

The patient recovered well after the procedure with only a mild persistent expressive dysphasia resulting in an NIHSS of 1 and was successfully discharged home. She later returned to the hospital electively and underwent successful endovascular closure of her atrial septal defect, without complication. Follow-up MRI demonstrated minor left-sided basal ganglia infarction (figure 4A) but no cortical infarction (figure 4B). Her three-month modified Rankin Scale was 0.

Discussion

The aMCA is an uncommon but clinically important variant of cerebral vascular anatomy with prevalence between 0.14%

Figure 1 Imaging on presentation

(A) Noncontrast brain CT showing established ischemic changes within the frontal lobe in the left middle cerebral artery territory (red arrow). (B) Noncontrast brain CT with a hyperattenuating intravascular thrombus (green arrow) on the left correlating with the expected position of a left middle cerebral artery thrombus. (C) Tmax and (D) cerebral blood volume from a CT perfusion demonstrating extensive ischemic penumbra of the left MCA territory with suggestion of ischemic mismatch. (E and F) Maximum intensity projection from CT angiography demonstrates normal contrast opacification in a horizontal middle cerebral arterial vessel and the distal cortical branches of the middle cerebral artery.

There has been some debate regarding the embryological origins of the accessory middle cerebral artery.

and 4% in angiographic and autopsy studies.^{1–8} It is a distinct and separate variation from a duplicated MCA (dMCA), which has a similar origin and path.

There has been some debate regarding the embryological origins of the aMCA. It was initially suggested that an aMCA may represent hypertrophy of the recurrent artery Heubner (RAH) as they originate from a similar area of the ACA.⁹ The primary arguments against this are that the aMCA and RAH may coexist and that they may be differentiated by the fact that the RAH enters the anterior perforated substance medial to the lenticulostriate arteries, whereas the aMCA travels laterally to areas normally supplied by the MCA.^{10–12}

As an aMCA runs laterally through the Sylvian fissure, it may supply orbitofrontal, precentral, and central areas making its presence, and occlusion, clinically important.^{4,11,13} Most of the time the aMCA has a smaller diameter than the MCA, but when the diameter is larger than the MCA, the territory supplied is also larger.⁴

The use of the terms accessory MCA and dMCA has had some overlap in the literature, leading to confusion regarding the exact definition.^{14,15} The initial description of the aMCA was by Crompton¹ in his 1962 article, which described an artery arising from the internal carotid artery (ICA), or near the anterior communicating artery (ACOM), which ran with the MCA through the Sylvian fissure supplying a portion of the MCA territory. Of the 11 examples of the aMCA found by Crompton¹ in his 347 specimens, 10 of the 11 arose from the ICA between the anterior choroidal artery (AChA) and the bifurcation of the ICA, with only 1 arising from the region of the anterior communicating artery.¹

The definition of what constitutes an aMCA has changed over time with Teal et al.¹⁰ giving us the modern definition based on the origin of the artery. Teal et al. defined duplication of the MCA to be present when 2 vessels originate from the distal ICA, whereas an accessory MCA is present when a vessel originating from the proximal ACA goes on to supply a distribution normally supplied by the MCA.¹⁰ This has subsequently become widely accepted within the medical literature and will be the definition used within this article.^{11,16,17}

To further understand the aMCA and its likely clinical implications, Manelfe et al. proposed a more detailed classification for the different anatomic subgroups of the aMCA based on the origin of the vessel and territory supplied, which has subsequently been modified by Lasjaunias.^{3,18} The goal

Figure 2 Digital subtraction angiography via left internal carotid artery injection

Digital subtraction angiography via left internal carotid artery injection. (A–D) Irregularity (red arrows) of the proximal A1 segment of the anterior cerebral artery was noted, as well as a lack of filling within the superior division cortical branches, and lenticulostriate vessels in the middle cerebral artery territory (green arrow) correlating with findings on CT angiography. This also confirmed patency of a horizontal middle cerebral arterial vessel and the distal cortical branches as identified on the CT angiography.

of this classification is to guide clinicians not only to the vessel's origins but also its cortical and central territories of vascular supply if an aMCA is present.³ Three subtypes have been proposed in the modified Manelfe classification. Type 1 and type 2 vessels are proximal types with the vessels being classified as striate arteries, which arise from the A1 segment of the ACA.¹⁸ They will supply the medial and lateral striate territories.¹⁸ The true MCA in these 2 subtypes is the proximal vessel and is purely cortical.¹⁸ The only differentiating factor between these proximal types is the cortical territory supplied by the aMCA with a type 1 having a more hypoplastic true MCA, meaning more cortical territory is supplied by the aMCA.¹⁸ A type 3 aMCA is a distal subtype arising from the distal A1 or proximal A2 segments of the ACA at or near the junction of the anterior communicating artery, and it has an extensive cortical supply.^{3,18} In this subtype, the true MCA will supply lateral striate and cortical territories.¹⁸

Despite reports that there may be effective collateral flow between an aMCA and MCA^{19–21} occurring through the leptomeningeal and perforating arteries,²⁰ there are numerous cases in which occlusion of the aMCA results in considerable neurologic deficits, particularly if the dominant hemisphere is involved.^{15,22,23} This was also the case with our patient who had a patent MCA demonstrated on angiography, however, failed to have sufficient collateral supply to prevent an ischemic penumbra (figures 1 and 2). This demonstrates the importance of being able to identify an

Knowledge of cerebral vascular variants is important for clinicians working in neurointervention, radiology, neurology, and emergency medicine fields.

occluded aMCA to ensure optimal patient outcomes. If a patient presents with an MCA syndrome but is found to have a patent horizontal trunk, a search must be undertaken for lenticulostriate vessels, which, if missing, suggests an occluded aMCA as likely etiology for the patient's symptoms.

Technical descriptions for catheterizing an aMCA are scant because of the small number of cases reported and with most aneurysms being managed with surgical clipping. Catheterizing of the aMCA may be very difficult because of both the small caliber and the acute angle the vessel takes from its origin.²⁴ Because of the variability in morphology when dealing with an aMCA, the clinician must have contemplated a number of technical considerations before attempting to catheterize this vessel. Either an ipsilateral or contralateral approach may be taken, which will be largely dictated by the

Figure 3 Digital subtraction angiography during/after procedure

(A and B) After gentle advancement of a Synchro2 microwire, the abnormality was felt to relate to an accessory middle cerebral artery. A Trevo microcatheter was advanced beyond the occlusion into distal branches of the accessory middle cerebral artery correlating with suspected missing branches on the initial angiogram. (C and D) After a single pass with a 4 × 40-mm Solitaire Stentriever, all middle cerebral artery branches opacified with mTICI 3 recanalization. Note increased opacification of perforating lenticulostriate arteries to the deep white and gray matter structures.

Figure 4 Follow-up MRI 3 months after ECR

(A) T2-weighted image showing minor left-sided basal ganglia infarction. (B) FLAIR image showing no cortical infarction. FLAIR = fluid attenuation inversion recovery.

origin of the vessel with a more distal vessel favoring a contralateral approach and a proximal vessel favoring an ipsilateral approach. Because of the variability in vessel caliber, with a tendency to be smaller than that of the MCA, smaller stent retrievers such as a 3-mm stent may be required to successfully perform ECR. Smaller microcatheters such as the SL-10 (Stryker, Fremont, CA), the Catch + mini (Balt Extrusion, Montmorency, France), or a tapered tip such as the Headway Duo (MicroVention, Aliso Viejo, CA) may more easily allow catheterization. Intra-arterial thrombolysis should also be considered and was used in our case.

In Parthasarathy et al's.²⁴ technical description of performing glue embolization for a pediatric patient with a dissecting type 3 aMCA aneurysm, they were able to access the aMCA using a contralateral approach with a Magic 1.2FM (Balt Extrusion, Montmorency, France) microcatheter over a Hybrid 0.007 (Balt Extrusion, Montmorency, France) wire across the ACOM. This followed an unsuccessful attempt using a coiling microcatheter (Headway Duo 1.6F; MicroVention, Aliso Viejo, CA) over a Traxcess (MicroVention, Aliso Viejo, CA) microguidewire with nimodipine priming.²⁴ The contralateral approach was found to be useful when attempting to catheterize the vessel due to the acute angle it forms with the ACA.²⁴ In our adult patient with an occlusion in a proximal aMCA, we were able to successfully pass the Trevo microcatheter (Stryker) and Solitaire Stentriever (Medtronic) into the aMCA and beyond the occlusion using an ipsilateral approach. An ipsilateral approach was used in our case as the vessel was a more proximally located aMCA compared with type 3 described earlier.

A small number of cases of occlusion of the aMCA have also been reported, with 2 cases resulting in cerebral infarction and 1 case of suspected transient ischemic attack treated with thrombolysis and dual antiplatelets, respectively.^{15,22,23} A single case of ischemia relating to occlusion of the ICA and ACA in a patient with an ipsilateral aMCA has also been reported and managed with bypass surgery.²⁵ Although rare,

ECR has previously been reported for the management of aMCA occlusion.²⁶

It is also important to consider other vascular mimics, which may have a similar presentation to an apparent MCA occlusion. An occlusion of either the posterior cerebral artery (PCA) or the AChA may also present with motor and sensory deficits.^{27–30} Although proximal PCA occlusion is associated with the classic Webers syndrome of crossed hemiplegia with a third nerve palsy, a patient may also present with hemiplegia without a third nerve palsy if the occlusion occurs distal to the origin of the PCA at the end of P1 or start of P2.^{27,28} Occlusion in this area would spare the proximal branches to the oculomotor nucleus but leads to a lesion in the lateral midbrain involving the pyramidal tract.^{27,28} Occlusion of the AChA may result in a combination of contralateral hemiparesis, contralateral hemisensory deficit, and ipsilateral visual field deficit, although not all may be present.^{29,30} The motor deficits with AChA occlusion are thought to be through its supply to the cerebral peduncle and the posterior limb of the internal capsule with the hemisensory loss attributed to a lesion of the superior thalamic radiations in the posterior limb of the internal capsule.³⁰

Knowledge of cerebral vascular variants is important for clinicians working in neurointervention, radiology, neurology, and emergency medicine fields. Although uncommon, given the clinical importance of the areas supplied by the aMCA, knowledge of this vessel is not only important for diagnosis but also for neurosurgical or endovascular management of patients with this variant. If a patient is presenting with an MCA syndrome, but their horizontal trunk is patent, close examination on the angiogram for the lenticulostriate vessels and associated blush must be performed. If they are missing, one must consider the presence of an aMCA as a causative agent for the patient's symptoms. If ECR is performed on this vessel, the origin and caliber of the aMCA will

TAKE-HOME POINTS

- If a patient is presenting with an MCA syndrome, but their horizontal trunk is patent, close examination on the angiogram for the lenticulostriate vessels and associated blush must be performed. If they are missing, one must consider the presence of an aMCA as a causative agent for the patient's symptoms.
- If endovascular clot retrieval is performed on an aMCA, the origin and caliber of the vessel will largely dictate which technique and equipment should be used.
- An aMCA is a distinct and separate variation from a dMCA, which has a similar origin and path.

largely dictate which technique and equipment should be used.

Author contributions

J. Cooke: drafting/revising the manuscript. J. Maingard: drafting/revising the manuscript, data acquisition, study concept or design, acquisition of data, and study supervision. R. V. Chandra: drafting/revising the manuscript, data acquisition, study concept or design, analysis or interpretation of data, acquisition of data, and study supervision. L.-A. Slater: drafting/revising the manuscript. M. Brooks: study concept or design. H. Asadi: drafting/revising the manuscript, study concept or design, contribution of vital reagents/tools/patients, and acquisition of data.

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References

1. Crompton MR. The pathology of ruptured middle-cerebral aneurysms with special reference to the differences between the sexes. *Lancet* 1962;2:421–425.
2. Komiyama M, Nakajima H, Nishikawa M, Yasui T. Middle cerebral artery variations: duplicated and accessory arteries. *AJNR Am J Neuroradiol* 1998;19:45–49.
3. Abanou A, Lasjaunias P, Manelfe C, Lopez-Ibor L. The accessory middle cerebral artery (AMCA): diagnostic and therapeutic consequences. *Anat Clin* 1984;6:305–309.
4. Kim MS, Lee HK. The angiographic feature and clinical implication of accessory middle cerebral artery. *J Korean Neurosurg Soc* 2009;45:289–292.
5. Marinkovic S, Milisavljevic M, Kovacevic M. Anatomical bases for surgical approach to the initial segment of the anterior cerebral artery: microanatomy of Heubner's artery and perforating branches of the anterior cerebral artery. *Surg Radiol Anat* 1986; 8:7–18.
6. Hamidi C, Bükte Y, Hattapoğlu S, et al. Display with 64-detector MDCT angiography of cerebral vascular variations. *Surg Radiol Anat* 2013;35:729–736.
7. Kim MS, Hur JW, Lee JW, Lee HK. Middle cerebral artery anomalies detected by conventional angiography and magnetic resonance angiography. *J Korean Neurosurg Soc* 2005;37:263–267.
8. Uchino A, Kato A, Takase Y, Kudo S. Middle cerebral artery variations detected by magnetic resonance angiography. *Eur Radiol* 2000;10:560–563.
9. Handa J, Shimizu Y, Matsuda M, Handa H. The accessory middle cerebral artery: report of further two cases. *Clin Radiol* 1970;21:415–416.
10. Teal JS, Rumbaugh CL, Bergeron RT, Segall HD. Anomalies of the middle cerebral artery: accessory artery, duplication, and early bifurcation. *Am J Roentgenol* 1973; 118:567–575.
11. Gibo H, Carver CC, Rhoton AL Jr, Lenkey C, Mitchell RJ. Microsurgical anatomy of the middle cerebral artery. *J Neurosurg* 1981;54:151–169.
12. Kuwabara S, Naitoh H. Ruptured aneurysm at the origin of the accessory middle cerebral artery: case report. *Neurosurgery* 1990;26:320–322.
13. Tacconi L, Johnston FG, Symon L. Accessory middle cerebral artery: case report. *J Neurosurg* 1995;83:916–918.
14. Hakim A, Gralla J, Rozeik C, et al. Anomalies and normal variants of the cerebral arterial supply: a comprehensive pictorial review with a proposed workflow for classification and significance. *J Neuroimaging* 2018;28:14–35.
15. Hiramatsu Y, Wakita M, Matsuoka H, Kasuya J, Hamada R, Takashima H. Cerebral infarction associated with accessory middle cerebral arteries: two case reports. *Intern Med* 2014;53:1381–1384.
16. Okahara M, Kiyosue H, Mori H, Tanoue S, Sainou M, Nagatomi H. Anatomic variations of the cerebral arteries and their embryology: a pictorial review. *Eur Radiol* 2002;12:2548–2561.
17. Takahashi S, Hoshino F, Uemura K, Takahashi A, Sakamoto K. Accessory middle cerebral artery: is it a variant form of the recurrent artery of Heubner? *AJNR Am J Neuroradiol* 1989;10:563–568.
18. Lasjaunias P, Berenstein A, Ter Brugge K. *Surgical Neuroangiography, Clinical Vascular Anatomy and Variations*. 2nd ed. Berlin: Springer; 2001.
19. Mueller DP, Sato Y, Yuh WT. Accessory middle cerebral artery as a source of collateral blood flow. *AJNR Am J Neuroradiol* 1991;12:1223–1224.
20. Liu Z-S, Zhou L-J, Sun Y, Kuang X-W, Wang W, Li C. Sufficient collateral blood supply from accessory middle cerebral artery in a patient with acute ischemic stroke. *Interv Neuroradiol* 2015;21:215–217.
21. Komiyama M, Nishikawa M, Yasui T. The accessory middle cerebral artery as a collateral blood supply. *AJNR Am J Neuroradiol* 1997;18:587–590.
22. Gao F, Jiang W-J. Transient ischemic attack associated with stenosis of accessory middle cerebral artery: a case report. *Clin Neurol Neurosurg* 2009;111:588–590.
23. Kuwahara H, Matsumura K, Watanabe M, Fujigasaki H. Intravenous t-PA for the occlusion of an accessory MCA. *Intern Med* 2013;52:163.
24. Parthasarathy R, Goel G, Gupta V, Narang KS, Anand S, Jha AN. Endovascular glue embolization of dissecting aneurysm of type-3 accessory middle cerebral artery: a contralateral approach. *Interv Neuroradiol* 2015;21:664–668.
25. Noguchi K, Aoki T, Komaki S, Takeuchi Y, Hirohata M, Morioka M. Unusual hemodynamic stroke related to an accessory middle cerebral artery: the usefulness of fusion images from three-dimensional angiography. *Surg Neurol Int* 2014;5:26.
26. Bayer-Karpinska A, Lutz J, Birnbaum T, Dieterich M, Wollenweber FA. Severe MCA stroke without MCA occlusion? Thrombectomy uncovers accessory middle cerebral artery. *Neurology* 2015;85:831–832.
27. North K, Kan A, de Silva M, Ouvrier R. Hemiplegia due to posterior cerebral artery occlusion. *Stroke* 1993;24:1757–1760.
28. Hommel M, Besson G, Pollak P, Kahane P, Le Bas JF, Perret J. Hemiplegia in posterior cerebral artery occlusion. *Neurology* 1990;40:1496–1499.
29. Hamoir XL, Grandin CB, Peeters A, Robert A, Cosnard G, Duprez T. MRI of hyperacute stroke in the AChA territory. *Eur Radiol* 2004;14:417–424.
30. Decroix JP, Graveleau P, Masson M, Cambier J. Infarction in the territory of the anterior choroidal artery: a clinical and computerized tomographic study of 16 cases. *Brain* 1986;109(pt 6):1071–1085.

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