CASE REPORT

# Incidental bilateral accessory middle cerebral arteries on MR imaging and MR angiography

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### **ABSTRACT**

Intracranial vascular variations involving the middle cerebral artery are rarely seen. Herein, we report an 11-year-old girl with incidental bilateral accessory middle cerebral arteries, an extremely rare variation, which is the first case in the related literature demonstrated by cranial magnetic resonance imaging and magnetic resonance angiography.

Key words: • magnetic resonance imaging • magnetic resonance angiography • middle cerebral artery

he 3 most common intracranial vascular variations involving the middle cerebral artery (MCA) are a duplicated MCA, an accessory MCA, and a fenestrated MCA (1). According to the classification of Teal et al. (2), when the 2 vessels originate from the distal end of the internal carotid artery (ICA), the condition is called a duplicated MCA. The term accessory MCA is used when the anomalous vessel originates directly from the anterior cerebral artery (ACA).

Although they are very rare in autopsy- and angiography-based studies, one can come across MCA variations relatively frequently with a careful examination as magnetic resonance angiography (MRA) is used as a screening technique (1). Unilateral cases with MCA anomalies appear in the literature not uncommonly; however, a bilateral accessory MCA is an extremely rare entity. Herein, we report a case of bilateral accessory MCA, which was an incidental finding, with cranial magnetic resonance imaging (MRI) and MRA findings. To the best of our knowledge, this is the first case of bilateral incidental accessory MCA diagnosed by MRI and MRA.

# Case report

An 11-year-old girl was referred to our center for further assessment of mental and motor retardation. The positive findings in physical and neurological examinations were lower extremity spasticity and inability to construct sentences containing 3 or more words. All blood laboratory studies were within normal ranges.

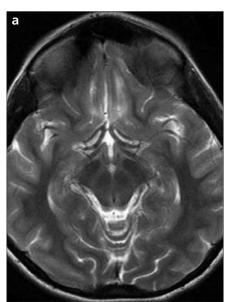
Cranial MRI was performed on a 3T MRI system (Siemens, Allegra, Erlangen, Germany). Cranial MRI was unremarkable, except for a vascular variation. On axial T2-weighted turbo spin echo (TSE) images (TR/TE, 4000/93 ms; turbo factor, 11), bilateral anomalous vessels with a similar course to that of the MCA was observed (Fig. 1). Three-dimensional time of flight (TOF) MRA (TR/TE, 39/4.4 ms; flip angle, 18°; slab thickness, 1.1 mm) showed the anomalous arteries were thinner than the main MCA and originated from the A1 segment of the ACA. Laterally, they had a parallel course with the main MCA (Fig. 2).

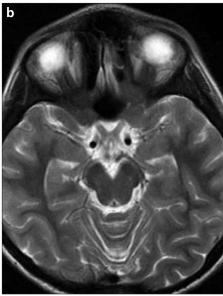
## Discussion

The embryological origin of an MCA is the ACA. There is no consensus on the embryological development of MCA variations. Handa et al. proposed that variant arteries are identical to the hypertrophied recurrent artery of Heubner (RAH) (3). Teal et al. disagreed with this idea since the RAH coexisted with an accessory MCA, perforating arteries did not always originate from an accessory MCA, and moreover, an accessory MCA had a different course than the RAH. On the other hand, an accessory MCA frequently has perforating arteries and the RAH can be multiple. Komiyama et al. proposed that both an acces-

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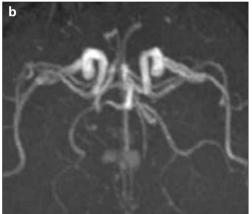
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**Figure 1. a, b.** Axial T2-weighted turbo spin echo images show the bilateral accessory arteries arising from the proximal anterior cerebral arteries (a) with a course parallel to middle cerebral arteries (b).





**Figure 2.** *a,* **b.** Maximum intensity projection (MIP) images of three-dimensional time of flight (TOF) magnetic resonance angiography (MRA) show the origin of the proximal anterior cerebral artery (a) with a course similar to that of middle cerebral artery (b).

sory MCA and a duplicated MCA are the anomalous early branches of the main MCA (4).

An accessory MCA and a duplicated MCA are also different in terms of cortical territories. An accessory MCA supplies the anterior frontal lobe, while a duplicated MCA supplies the anterior temporal lobes (1).

There are different studies on the frequency of accessory MCAs. The reported frequencies of duplicated MCAs and accessory MCAs are 0.2%–2.9%, and 0.3%–4.0%, respectively, in anatomy- and angiography-based studies (5, 6). Uchino et al. reported accessory and duplicated MCA frequencies of 1.2% and 2.1%, respectively, in their

MRA series. MCA variations can be detected relatively frequently on MRA examinations (1). Cerebral aneurysms are encountered more frequently in cases with anatomic variations, such as an accessory MCA (7); however, the reason for this association is still vague.

Teal et al. defined duplicated MCAs and accessory MCAs according to the origins of the anomalous vessels (2). There are 2 types of accessory MCAs based on the origin of variant vessels, which can be proximal (type 1) or distal (type 2) segments of the ACA. Our case could be defined as a type 1 accessory MCA according to this classification, which is widely accepted (4).

Another classification by Manelfe includes 3 types of accessory MCAs based on the origin of the variant vessels (4, 8): The anomalous vessel originates from the proximal segment of the ICA bifurcation in type 1 (Teal's classification called this variation a duplicated MCA), from the proximal portion of A1 in type 2, and from the distal portion of A1 in type 3. Frequently, the caliber of an accessory MCA is thinner than that of the main MCA and ACA, as was in the presented case. The differentiation between a duplicated MCA and an accessory MCA arising from the proximal segment of the ACA (types 1 and 2 accessory MCA, respectively, in Manelfe's classification) is difficult when the caliber of the variant artery is equal to the main artery. In Teal's classification, a type 1 accessory MCA can also be regarded as a variant of a duplicated MCA (4).

In our case, there were bilateral accessory MCAs, which were of thinner caliber than the main MCA, both originating from the proximal segment of A1 with a course parallel to the main MCA. The vessels could be regarded as type 1 accessory MCAs, according to Teal's classification, and type 2 according to Manelfe's. Recognition of these variations gains importance in 2 conditions: The first is a planned surgical dissection for associated aneurysms in which awareness of those variations may be vital, and the second is a possible MCA occlusion when the accessory MCA may supply the collateral blood flow of the frontal lobe and the basal ganglia. Each case should be carefully evaluated and managed regarding these variations.

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