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Hyperplastic Anterior Choroidal Artery Identified Using Magnetic Resonance Angiography: A Report of Two Cases

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Introduction

As magnetic resonance angiography (MRA) is increasingly utilized as the initial imaging examination for the cerebral vasculature, particularly in the setting of acute stroke and subarachnoid hemorrhage, noninterventional radiologists must become familiar with vascular anomalies that were classically described on conventional angiography. Indeed, these anomalies may impact the anatomical distribution of brain ischemia and the surgical or endovascular approach for the management of intracranial pathology.

A hyperplastic anterior choroidal artery (AChorA) supplying the temporal and/or occipital distribution of the posterior cerebral artery (PCA) territory is an uncommon anomaly [1–3]. Until now this anomaly has been described only in angiographic, surgical and postmortem studies [1–8].

We report the cases of 2 patients where anomalous hyperplastic AChorAs were depicted on 3-dimensional (3D) time of flight (TOF) MRA of the circle of Willis.

Case Reports

Case 1. A 24-year-old man with peripheral T cell lymphoma presented to our institution with neutropenic fever. Magnetic resonance imaging (MRI) and a 3D TOF MRA were performed to evaluate for a possible intracranial origin of fever and/or vasculitis. The MRA did not demonstrate evidence of vasculitis and showed a conventional vascular anatomy of the right PCA, bilateral middle (MCA) and anterior (ACA) cerebral arteries. No posterior communicating artery (PCOM) was identified on the right side (fig. 1).

On the left side a large PCOM was present, which appeared of similar size to the first segment of the left PCA (P1). The left P1 and PCOM united to form a left P2 artery, which subsequently

Fig. 1. Reformation of a 3D TOF MRA for the evaluation of a 24-year-old man with peripheral T cell lymphoma and neutropenic fever, demonstrates a hyperplastic AChorA giving rise to parieto-occipital and calcarine branches. The P2 segment of the PCA proper branches into posterior temporal arteries. ICA = Internal carotid artery; P1 = P1 segment of the PCA; P2 = P2 segment of the PCA. (The image was flipped horizontally to respect the convention in radiological image orientation.)

branched into left posterior temporal arteries. A second artery of similar caliber to the large PCOM was also identified arising from the supraclinoid internal carotid artery (ICA) distal to the origin of the PCOM, corresponding to a hyperplastic AChorA. This hyperplastic AChorA coursed posteriorly, parallel to the left PCOM and left P2, and gave rise to parieto-occipital and calcarine arteries.

Case 2. A 7-year-old boy with a family history of arteriovenous malformations presented with new-onset migraine-like headaches. An MRI including a 3D TOF MRA was performed to evaluate for vascular malformations.

The MRA (fig. 2) demonstrated no aneurysms or vascular malformations and showed a conventional anatomy of bilateral ACAs and MCAs. Bilateral large PCOMs were noted with codom-

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Fig. 2. Reformation of a 3D TOF MRA for the evaluation of a 7-year-old boy with a family history of arteriovenous malformations, demonstrates a hypoplastic P2 segment of the PCA proper and a hyperplastic AchorA giving rise to the remaining branch arteries of the PCA territory. (The image was flipped horizontally to respect the convention in radiological image orientation.)

inant P1 segments. The right P2 segment, and its subsequent branches, had a conventional anatomy, while the left P2 was hypoplastic. However, on the left side, an additional artery was identified arising from the supraclinoid ICA distal to the origin of the left PCOM, which coursed posteriorly, lateral to the PCOM. This hyperplastic AchorA gave rise to the temporal and occipital branches of the left PCA.

Discussion

In up to 1% of patients, the AchorA is hyperplastic and supplies all, or a portion of, the PCA territory [1–3]. Indeed, during normal development, the AchorA has potential anastomoses with its neighboring arteries, especially the PCOM and PCA. Hyperplasia of the AchorA may represent a scenario in which 1 (or more) of these anastomotic channels is hemodynamically favored and therefore enlarges, with resultant variable degrees of hypoplasia of the PCA proximal to the anastomosis [1, 4]. The conventional anatomy and relationship of the AchorA and PCA are illustrated in figure 3.

Takahashi et al. [1] described 4 subtypes of hyperplastic AchorAs, according to the course and distribution of the vessel. Type 1 consists of a hypertrophic uncal branch that supplies the anterior temporal branch of the PCA. Type 2, also known as ‘the anomalous temporal artery’, gives rise to most of the anterior and posterior temporal branches of the PCA. In type 3 an anomalous

Fig. 3. Illustration of normal vascular anatomy of the right AchorA and the left PCA. A1 = Segment of the anterior cerebral artery; AT = anterior temporal; MT = middle temporal; PT = posterior temporal; LPCh = lateral posterior choroidal; MPCh = medial posterior choroidal; PO = parieto-occipital; Calc = calcarine; SC = short circumflex; LC = long circumflex; PP = peduncular perforators; TGP = thalamogeniculate perforators; TP = thalamoperforators; Hippo = hippocampal branch; Unc = uncal branch; OT = branch supplying optic tract; Ped = peduncular branch; LG = lateral geniculate body; Chor = choroidal branches.

artery supplies the parieto-occipital and calcarine arteries. In type 4, also known as ‘anomalous temporo-occipito-parietal artery’, the original PCA is almost completely replaced by the anomalous hyperplastic AchorA.

In the first case presented, the parieto-occipital and calcarine arteries arise directly from the ICA via a hyperplastic AchorA, and the temporal branches are supplied by the PCA proper, supported by the basilar artery and a well-developed PCOM. This pattern is consistent with Takahashi’s type 3 hyperplastic AchorA (fig. 4a). A persistent anastomosis of the AchorA with the PCA distal to the branching of its temporal arteries is thought to explain the appearance of this anomalous parieto-occipital artery [1, 4].

In the second case presented, the temporal, parieto-occipital and calcarine branches are supplied by a hyperplastic AchorA, directly from the supraclinoid ICA. A well-developed ipsilateral PCOM and P1 segment are present, with hypoplasia of the P2 segment of the PCA proper. This case is an example of Takahashi’s type 4 hyperplastic AchorA (fig. 4b), in which an anastomosis may have remained at the level of the PCOM or anterior ambient segment of the PCA, leading to almost complete replacement of the PCA by the hyperplastic AchorA [1, 4].

In addition to hyperplasia, other developmental abnormalities of the AchorA have been reported. Anomalous origin of the AchorA has been described [3], including origin from the MCA,

Fig. 4. a Type 3 Takahashi anomaly. A hyperplastic AChorA supplies the parieto-occipital and calcarine branches, while the PCA proper gives rise to the temporal branches. **b** Type 4 Takahashi anomaly. The PCA territory is almost completely fed by a hyperplastic AChorA arising from the ICA.

the PCOM and rarely from the terminal ICA bifurcation or the proximal ICA before the take-off of the PCOM. Duplication of the AChorA has also been reported, with either separate or a common origin from the ICA [3]. On the other hand, absence of the AChorA is exceedingly rare [8]. Finally AChorA aneurysms represent 2–4% of all intracranial aneurysms [9]. Although associations between other vascular anomalies and aneurysms have been reported, the literature has not yet revealed an increased incidence of aneurysm formation in connection with a hyperplastic AChorA.

Conclusion

With the increasing utilization of MRA for the initial evaluation of the intracranial vasculature, noninterventional radiologists must become familiar with anatomic variants that may alter the anatomical distribution of brain ischemia and the surgical or endovascular approach for the management of intracranial pathology. We report 2 cases of anatomic variants of the anterior choroidal artery identified on 3D TOF MRA, which represent examples of type 3 and 4 Takahashi hyperplastic anterior choroidal arteries.

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Subarachnoid Hemorrhage from Spontaneous Dissection of the Anterior Cerebral Artery

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Dissection of intracranial arteries usually concerns the posterior circulation [1, 2]. We report 2 cases of subarachnoid hemorrhage (SAH) related to a spontaneous dissection of the anterior cerebral artery (ACA). We discuss the pathophysiology and management of this rare entity.

Case 1

A 65-year-old woman experienced severe acute headache without associated neurological signs except a slight aphasia. She had a history of arterial hypertension and had not suffered any