REVIEW

Anatomic variants of the anterior part of the cerebral arterial circle at multidetector computed tomography angiography

Variantes anatomiques de la partie antérieure du cercle artériel de la base du cerveau en angioscanner multidétecteur

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Summary Imaging of the cerebral arterial circle (CAC) is essential in neurovascular diseases such as ischemic stroke for detecting arterial occlusions and evaluating arterial supply, and in subarachnoid or intralobar hemorrhage for detecting intracranial malformations. Multidetector computed tomography angiography (MD-CTA) is increasingly being used for the detection and treatment planning of intracranial aneurysm. For optimal interpretation and treatment planning, this method requires suitable post-processing equipment, and extensive knowledge of the relevant anatomy and anatomical variants. Anatomical variants of the CAC are common, particularly in the anterior CAC, the most common site of intracranial aneurysm. The aim of this review is to illustrate the normal anatomy and most common anatomical variants of the anterior CAC detected by MD-CTA, and to discuss the relevant embryological and technical considerations.

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Introduction

Imaging of the cerebral arterial circle (CAC) is essential in neurovascular diseases such as ischemic stroke for detecting arterial occlusions and evaluating the arterial supply, and in non-traumatic, acute subarachnoid or intralobar hemorrhage for detecting intracranial malformations (aneurysm or arteriovenous malformation). For the latter, digital subtraction angiography (DSA) is still considered the imaging modality of choice for assessing suspected intracranial malformation, particularly aneurysm [1–6]. However,
the technique is invasive, time-consuming and a possible cause of neurological complications (1–2%) [4]. For this reason, multidetector computed tomography angiography (MD-CTA) is increasingly being used for the detection and treatment planning of intracranial arterial malformations [1,2,4,5,7]. This non-invasive imaging technique has high sensitivity and specificity for the diagnosis of even small intracranial aneurysms in patients with non-traumatic, acute subarachnoid hemorrhage, and achieves quality and accuracy approaching that of DSA [1,2,4,5,7]. In addition, MD-CTA offers the advantages of faster scanning and more complete anatomical information [1–6].

However, for optimal interpretation and treatment planning, the method requires suitable post-processing equipment, and an extensive knowledge of anatomy and anatomical variations. Anatomical variations of the CAC are commonly found, particularly in the anterior CAC [8–11]. In addition, the anterior CAC, which includes the anterior cerebral arteries (ACAs) and anterior communicating artery (ACoA), is the most common site of intracranial aneurysm (about 40%) [12]. These aneurysms are frequently associated with anatomical variations, some of which are considered risk factors for aneurysm development [8–10,12–14]. However, multiple anatomical variants are also observed with no aneurysm. Indeed, diagnostic efficiency is seen to improve in parallel with a thorough understanding of both normal cerebral vascular anatomy as well as the variants that can mimic pathology [11].

The purpose of the present review is to illustrate and discuss the normal anatomy and anatomical variations detected in the anterior CAC with MD-CTA.

Imaging technique

To illustrate the normal anatomy and anatomical variants of the anterior CAC detected by MD-CTA, we prospectively collected cases from December 2005 to June 2009. MD-CTA is a quick, thin-section, volumetric spiral CT examination performed with a time-optimized bolus of contrast medium to enhance the cerebral arteries. Using a 64-slice CT scanner (Lightspeed VCT, General Electric Medical Systems, Milwaukee, WI, USA), the examination included the region from the first vertebral body to the vertex, using the following parameters: 120 kV; 300 mAs; section thickness 0.6 mm.

For enhancement of the intracranial arteries, 70 mL of non-ionic contrast medium (libritrodil, Xenetix® 300, Guerbet, France) was routinely used, injected intravenously at a flow rate of 4 mL/s, followed by a 40-mL saline flush, using a power injector. A bolus-tracking method was also routinely used to achieve optimal synchronization of contrast medium flow and scanning.

Acquired data were transferred to a GE Advantage Workstation 4.2 (General Electric Healthcare, Milwaukee, WI, USA) for analysis and post-processing. After studying the axial source images, post-processing methods such as multiplanar reformation (MPR) in two other orientations were then used—specifically, in parallel and orthogonal to the main course of the respective vascular segment—along with thin-section (2–5 mm) maximum intensity projection (MIP) and volume rendering (VR), rotated interactively for three-dimensional (3D) visualization.

All of the figures in this article were obtained using the clinical Workstation software.

Embryology

The basic principles of cerebral artery embryology are well-known, although the development of some anomalies remains controversial [8,9,15–19]. Embryogenesis of the cerebral arteries begins at approximately 5 weeks of gestation. From the primitive intracranial arteries, many arterial branches develop and form Anastomoses among themselves; then, certain arterial segments regress [8,9,15]. During this stage, the adaptation process between the cerebral vessels, on the one hand, and the morphology and metabolic needs of the brain, on the other hand, is complex [9].

At 5 weeks of gestation, two principal arteries are present: the internal carotid arteries (ICAs); and the bilateral longitudinal neural arteries (BLNA). Normally, there are also transient anastomoses between these two principal trunks: the trigeminal arteries; otic arteries; hypoglossal arteries; and proatlantal arteries [8,9,15–17]. The anterior CAC originates from the ICA, while the BLNA gives rise to the vertebrobasilar system [8,9,15–17]. During the same period of time, the ICA bifurcates into cranial and caudal divisions. This is an important stage in cerebral artery embryogenesis. Division occurs at what will later become the posterior communicating artery (PCoA). The cranial division gives rise to the anterior CAC, which includes the ACA, anterior choroidal artery and middle cerebral artery (MCA). The caudal division terminates as the PCoA—a normally definitive carotid—vertebrobasilar communicating artery that ‘closes off’ the CAC posteriorly—the proximal segment of the posterior cerebral artery and the upper basilar system (distal to the origin of the trigeminal artery) [8,9,16,20].

To understand the genesis of the anterior CAC and its most common variations, we first need to consider Padget’s theory [8,16]. According to Padget [8,16], the embryogenesis of the anterior CAC is the result of two important stages: first, the development, from the cranial division of the ICA, of numerous arteries supplying the anterior part of the brain; and then, the regression of certain arterial segments in utero and, in some cases, during ex utero development into adult-hood [8,14,16]. The embryological CAC, which is more highly developed than the CAC in adults, has three ‘ACAs’, with an anterior communicating plexus (ACoP) connecting these three arteries (Fig. 1). The third ‘ACA’ follows the course of the two other ‘ACAs’ and is known as the median artery of corpus callusom (MACC). The MACC and ACoP should then regress [8,16], and failure to do so can lead to numerous anatomical variants, such as an accessory ACA due to a persistent MACC, or a double ACoA due to incomplete regression of the ACoP. Some segments that should persist may also abnormally regress (resulting in, for instance, aplasia or hypoplasia of the proximal ACA segment).

However, this theory has been partially refuted by Baptista [9,17], who maintain that an accessory ACA detected in the adult brain and considered an anatomical variant has a different course from the embryological MACC. Therefore, it may not be an abnormally persistent embryonic vessel, but an additional vessel, resulting from the fusion of pericallosal branches [9,17]. This theory could also explain other
anatomical variations, such as an azygous or bihemispherical ACA [9,17].

In addition, some anatomical variations of the MCA could be described as a variant of the anterior CAC, as the MCA is embryologically considered a collateral branch of the ACA [8,9,16,20].

Modal anatomy

Anterior cerebral arteries

The ACA arises from the termination of the ipsilateral ICA, and supplies blood to the medial regions of the frontal and parietal cortex, corpus callosum and falx cerebri. It curves around and over the corpus callosum up to the splenium. Both ACAs are connected to each other via the ACoA [8,9,21]. The ACA is usually divided into an A1 segment (between the carotid bifurcation and ACoA), an A2 segment (from the rostrum to the genu of the corpus callosum), an A3 segment (around the genu of the corpus callosum) and A4–A5 segments (in the horizontal trajectory over the corpus callosum, divided by a vertical line over the coronal suture) [9,21]. This terminology is used in neurosurgery and neuroradiology for diagnostic purposes, but is considered anatomically inadequate [9].

The A1, or precommunicating, segment courses anteriorly and medially to reach the longitudinal fissure, usually passing over the optic nerve (Fig. 2). It gives rise to anteromedial central arteries that supply the hypothalamus, pituitary gland and optic chiasm [9,21]. The anteromedial central arteries are not usually visualized by MD-CTA.

The A2, or post-communicating, segment courses anteriorly to the rostrum and genu of the corpus callosum, and gives rise to three main branches: the recurrent artery of Heubner (RAH), or medial striate artery, which supplies the anterior part of the caudate nucleus, anterior third of the putamen, anterior limb of the internal capsule, olfactory trigone and tract, and caudomedial part of the orbitofrontal cortex; and the orbitofrontal artery and polar frontal artery, which supply the inferomedial and anteromedial parts, respectively, of the frontal cortex [9,21]. The RAH is not usually visualized by MD-CTA.

The A3–A5 segments surround the corpus callosum. There are usually two main divisions originating from the A3 segment: the pericallosal artery; and the callosomarginal artery. The pericallosal artery supplies the corpus callosum and anteromedial part of the parietal cortex via the precuneal artery. Its final branch, the posterior pericallosal artery, anastomoses with the posterior cerebral arterial system. The callosomarginal artery supplies the posteromedial frontal cortex and the cingular cortex via the paracentral artery [9,21].
Figure 3  Modal anatomy of the anterior communicating artery (ACoA): posterolateral volume rendering (VR) MD-CTA demonstrates the single ACoA connecting the anterior cerebral arteries (ACAs; arrow).

**Anterior communicating artery**

The ACoA is a single artery connecting the ACAs that ‘closes off’ the CAC anteriorly. It is located above and anterior to the optic chiasm (Fig. 3) [8,9,21]. It defines the A1 and A2 segments of the ACAs, and gives rise to small anteromedial central branches (same supply as the A1 segment), which are not usually visualized by either MD-CTA or DSA [1,5].

**Anatomical variations**

Anatomical variants of the CAC, segment by segment, are presented in Table 1 from the most common to the most rare, with an embryological explanation for each variation. However, these variations are frequently associated with each other.

**ACoA**

The most common variations are ‘multiple ACoA’ (frequency: 4—40%) [8,18,22,23] and ‘hypoplasia’ (frequency: 3—16%) [8,22], whereas aplasia is a rare occurrence (frequency: 0—0.3%) [8,22]. Multiple ACoA includes several variants, such as double, plexular, and Y- or V-shaped ACoA [8,22,23] (Fig. 4), and is described as incomplete regression of the embryological ACoP [8,13,22]. Hypoplasia or aplasia of the ACoA is considered an abnormal regression of the ACoP. Side-to-side connections between ACAs with no distinct vessel are also considered aplasia [8]. These variations do not appear to be significantly associated with aneurysm [8,12].

**A1 segment**

The most common variations of the A1 segment are hypoplasia (frequency: 10—35%) [8,19,23], fenestration (frequency: 0.1—8%; Fig. 5) [8] and an accessory/duplicate MCA (frequency: 0.2—4%; Fig. 6) [8,10,24]. Aplasia, and low origin and infraoptic course of the A1 segment are rare [8,18,19]. Hypoplasia and aplasia are easily explained by abnormal regression of the A1 segment, which should normally persist [8]. However, the genesis of fenestration of the A1 segment (or any other arterial branch) is controversial: incomplete regression of embryological vessels, and partial duplication or crossing with a non-vascular structure have been suggested [8,11]. Nevertheless, these two variants are frequently associated with ACoA aneurysm [8,12,13,18]. A second MCA that mostly originates at the ICA termination is referred to as a ‘duplicate’ MCA. It can also originate from the A1 or A2 segments, when it is then termed an ‘accessory’ MCA [8,9,24]. Several explanations have been proposed for the development of this variation, including anomalous early ramification of the early branch of the MCA, which originates from the distal end of the ICA (duplicate MCA) or from the A1 portion of the ACA (accessory MCA) [24]. As for the more unusual variations of the proximal ACA segment, such as low origin or an infraoptic course, Padget and Lasjaunias each have incriminated dysembryogenesis of the ophthalmic artery (OphA) [8,9,16,18,19]. Both have described the primitive dorsal and ventral OphAs that form a transient anastomotic loop around the optic nerve. Persistence of either of these primitive OphAs would explain the low origin or infraoptic course of the proximal ACA segment [8,9,16,18,19]. Another explanation points to a persistent embryological anastomosis between the primitive maxillary artery, which supplies the optic vesicle before 5 weeks of gestation, and the ACA [18,19].

**A2—A5 segments**

Anatomical variants of the A2—A5 segments are numerous and sometimes difficult to distinguish. The most common

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Figure 4  Multiple anterior communicating artery (ACoA): (A) axial MIP and (B) posterolateral VR MD-CTA demonstrate a plexular ACoA (arrow).

Figure 5  Fenestration of the anterior cerebral artery (ACA): (A) coronal MIP and (B) anterior VR MD-CTA demonstrate fenestration of the right A1—A2 segment junction (large arrow). Fenestration in the right middle cerebral artery (MCA) is also visualized (thin arrow).

Figure 6  Duplicate middle cerebral artery (MCA): (A) coronal MIP and (B) posterior VR MD-CTA demonstrate a right duplicate MCA (large arrow) associated with hypoplasia of the A1 segment of the right anterior cerebral artery (ACA) and an aneurysm of the anterior communicating artery (ACoA; thin arrow).
variations are triple or accessory ACAs (frequency: 0.2—13%; Fig. 7) [8,13], single ACA trunk (frequency: 1.7—4%; Fig. 8) [8] and azygous ACA (frequency: 0—10%) [8,10,13]. A bihemi-spherical ACA (Fig. 9) and early A2 segment bifurcation are rare [8]. A simplified representation of the normal anatomy and common variations of the A2—A5 segments is shown in Fig. 10.

A triple or accessory ACA is defined as the presence of a third artery supplying the anterior cerebral territory and, in particular, the pericallosal artery territory (generally bilaterally). As already mentioned above, its embryogenesis is still controversial. An 'azygous' ACA is defined as a single, unpaired ACA arising as a solitary midline trunk and supplying both ACA territories, and can be explained by abnormal fusion of ACA arterial segments [8,9,13]. When fusion is incomplete, with the first division of the trunk located above the genu of the corpus callosum, it is referred to as a ‘single ACA trunk’, which is a more common finding [8]. The length of the trunk varies. Azygous ACA and single ACA trunk may be associated with aneurysms of the pericallosal arteries [8,10]. Azygous ACA may also be seen in cases with holoprosencephaly and neuronal migration abnormalities [8,13]. A ‘bihemispherial’ ACA is one that supplies blood to both cerebral hemispheres, while the contralateral ACA is usually hypoplastic. This includes many variations — from a single contralateral branch to an azygous-like artery [8,9,13]. Although a full description of all these variants is not possible, it is important to be able to recognize the presence of a bihemispherial ACA supplying a large contralateral territory. An early bifurcation of the A2 segment is one that is located above the genu of the corpus callosum [8], and usually gives rise to a callosomarginal-like artery. These last two variations may also be associated with aneurysm of the pericallosal arteries and can be explained by abnormal persistence of segments of the embryological ACoP [8,10].

Figure 7 Accessory anterior cerebral artery (ACA): (A) sagittal MIP and (B) anterior VR MD-CTA demonstrate an accessory ACA (arrowhead). Fenestration of the A1 segment of the right ACA is also visualized (thin arrow).

Figure 8 Examples of a single anterior cerebral artery (ACA) trunk from two different patients: (A) anterior VR MD-CTA demonstrates a short single ACA trunk (thin arrow); (B) lateral VR MD-CTA demonstrates a long single ACA trunk (large arrow). An aneurysm at the bifurcation of the single ACA trunk is also visualized (arrowhead).
### MD-CTA limitations

MD-CTA is a valuable tool for diagnosing intracranial aneurysms and anatomical variants, especially since the development of the 64-section MD-CTA, which can improve the detection of aneurysms less than 4 mm compared with 4- or 16-section MD-CTA [4]. The technology also offers other advantages over DSA: it is faster; non-invasive; has less radiation exposure; requires less contrast medium [3]; is less expensive; and provides more complete anatomical information (unlimited viewing angles, 3D views and the ability to detect atheroma, mural calcification and thrombosis) [2]. However, it also has some limitations compared with DSA: it has lower spatial resolution; is unable to visualize small arteries less than 1 mm (RAH, anterior choroidal artery and perforating arteries) [1,5]; and has difficulty analyzing structures adjacent to bone, or enhancing the cavernous sinus (periporphthalmic ICA, cavernous ICA, posterior inferior cerebellar artery, ACoP) [2,4], or in the presence of other venous contrast [4]. However, the advent of 3D rotational angiography appears to offer increased aneurysm detection, with improved visualization of aneurysm configuration and contour compared with DSA alone [4]; for example, A1 segment fenestration may be missed with MD-CTA, but is correctly visualized with 3D rotational angiography (Fig. 11).

However, there are currently no standardized methods available for data analysis, especially for 3D imaging, although several authors have recommended a procedure to follow [1,25,26]. First, study of the axial source images is essential, as these images on their own can provide complete information and do not include artifacts introduced by various post-processing techniques [1,25,26]. However, it is important to note any discordance between the reconstructed and source images; these images are invaluable for detecting calcifications, which can be misinterpreted as aneurysms on VR views, and thrombosis, which can be missed on MIP or VR views [1,26]. Second, MPR is useful for further examination of MD-CTA data, as any oblique plane can easily be achieved. MPR is advantageous compared with MIP and VR techniques for luminal and intraluminal information, as it is akin to taking thin sequential slices through the vessel. However, MPR views are required in at least two planes to improve information accuracy [1,26]. MIP is a projection technique in which only the brightest voxels of a volume are collected and used to create an image. It is widely known that MIP improves aneurysm detection sensitivity. It is also important to note that MIP post-processing reduces image information by about 10% of the full source image. A further limitation of MIP is that it is not a 3D technique per se, but a projection of 3D volume data onto a two-dimensional plane. This means that MIP images do not offer depth information, and the relationships among the intracranial arteries cannot be directly extracted from MIP images. Furthermore, with MD-CTA, the MIP algorithm is limited by the higher density of bone compared with opacified vessels. This means that whenever vessels and bone are lying in the same projection beam, the vessel will be masked by the bone [25]. Finally, the VR technique uses all of the pixels in the dataset and, thus, provides a true 3D view. For this reason, it offers a clearer perception of the courses and relationships of the intracranial arteries than does the MIP algorithm, and that is often a deciding factor in treatment planning [1,25].

Three-dimensional time-of-flight magnetic resonance angiography (3D TOF MRA) is also an effective modality for exploring the CAC (Fig. 12), and 3D TOF MRA at 3T...
Figure 11  MD-CTA vs 3D rotational angiography: (A) lateral 3D rotational angiography demonstrates fenestration (large arrow) at the origin of the right pericallosal artery (early bifurcation of the right A2 segment); (B) lateral VR MD-CTA does not show the fenestration (large arrow). Aneurysm of the anterior communicating artery (ACoA) is also visualized (thin arrow).

Figure 12  Infraoptic course of the right A1 segment, demonstrated by 1.5-Tesla magnetic resonance imaging (MRI): (A, B) axial T2-weighted image shows the right A1 segment (large arrow) under the optic nerve (thin arrow); (C) anterior VR view on 3D TOF MRA also demonstrates the infraoptic right A1 segment (large arrow). Aplasia of the left A1 segment and a plexular anterior communicating artery (ACoA; thin arrow) are also visualized.

appears to have no significant differences in diagnostic performance compared with 64-section MD-CTA [6]. The modality also reduces the risks associated with contrast medium, X-ray exposures, and confusion between bony and venous structures [6]. However, due to its lower spatial resolution compared with MD-CTA, 3D TOF MRA is not routinely used for the detection of intracranial aneurysm in acute subarachnoid hemorrhage but is, instead, useful in high-risk asymptomatic patients, in the follow-up of treated aneurysms and in the study of ischemic stroke.

Conclusion

MD-CTA is an essential tool for the evaluation of intracranial vascular pathology. Anatomical variants in the anterior CAC are especially common and some are associated with aneurysm. However, diagnostic efficiency can be improved with a thorough understanding of post-processing methods and normal CAC anatomy, as well as of the anatomical variants that can mimic pathology and which need to be taken into account in surgery planning.

Conflicts of interest

None.

References


