Variants of cerebral arteries – anterior circulation

Grzegorz Makowicz, Renata Poniatowska, Małgorzata Lusawa

Department of Neuroradiology, Institute of Psychiatry and Neurology, Warsaw, Poland

Author's address: Grzegorz Makowicz, Department of Neuroradiology, Institute of Psychiatry and Neurology, Warsaw, Poland, e-mail: gmakowicz@gmail.com

Summary

Advances in imaging techniques allow for *in vivo* identification of abnormalities and normal variants of cerebral arteries. These arterial variations can be asymptomatic and uncomplicated although, some of them increase the risk of aneurysm formation, acute intracranial hemorrhage, play a vital role in neurosurgical planning or can be misidentified as serious pathology and medical errors. The goal of this publication is to discuss arterial anomalies of anterior cerebral circulation, their prevalence and demonstrate radiological images of some of those variants. In this article we will discuss variants of internal carotid artery, anterior cerebral artery, anterior communicating artery, middle cerebral artery, persistent stapedial artery and fenestration.

Key words: arterial abnormalities • congenital absence of the ICA • duplication • fenestration

PDF file: http://www.polradiol.com/download/index/idArt/889403

Background

Nowadays, we are able to visualize vessels non-invasively with high precision using computed angiography with contrast (Angio-CT) or magnetic resonance with or without administration of contrast (Angio-MR). Estimated data from the United States show a significant increase in the number of performed computed tomography examinations from 3 million in 1980 to 63 million scans in 2006, and almost one third of those studies involving the head [1]. There are studies proving that application of 64-slice computed tomography apparatuses is superior to 4- and 16-slice CT scanners with regard to detection of small aneurysms and acquired results are comparable to those obtained in subtraction angiography or even 3-dimensional rotational angiography [2]. Currently, multi-slice CT scanners detect over 90% of small vessels compared to digital subtraction angiography and are able to reliably demonstrate arteries with diameter over 0.7 mm [3]. These results are better than those acquired with double-detector devices available at the end of the 90s [4]. We found no analyses in the literature regarding increase in frequency of detection of anatomical variants depending on the use of newer CT and MR scanners. However, in light of the above-mentioned facts, it seems that due to technological progress and availability of non-invasive imaging methods we are currently able to detect greater number of even subtle anatomical variants. Until now, they were not identified *in vivo* in asymptomatic patients. The goal of this work is to review anatomical variants of anterior cerebral circulation arteries, frequency of their occurrence and to demonstrate radiological picture of some of those variations. It is estimated that lack of knowledge of anatomical variants is the cause of about 10% of medical errors [5].

Short Description of Normal Anatomy of Arteries in Cerebral Circulation

Arterial circulation of the brain originates from common carotid arteries that arise from the arch of aorta on the left and from brachiocephalic trunk on the right side. Anterior cerebral circulation is formed by two internal carotid arteries (ICA) that emerge from common carotid arteries. In its extracranial portion ICA does not give lateral branches. Arterial circle, also known as the Circle of Willis, provides arterial supply to the brain. Clinically, it is divided into an anterior and posterior part.

Anterior portion of arterial circle consists of internal carotid arteries bilaterally. Two anterior cerebral arteries (ACA) connected by an unpaired anterior communicating artery (ACoA) extend anteriorly. Laterally, middle cerebral arteries (MCA) constitute morphological extensions of internal carotid arteries.

Posterior cerebral circulation originates from vertebral arteries (VA) that arise from subclavian arteries. Both

vertebral arteries merge intracranially, forming the basilar artery (BA). Posterior cerebral arteries (PCA) are the distal branches of basilar artery. Both parts of the arterial circle are connected only by posterior communicating arteries (PCoA). The role of cerebral arterial circle is to provide collateral circulation to the brain. There are numerous individual deviations from the described normal arterial blood supply to the brain.

Variations of Internal Carotid Artery

Aberrant Internal Carotid Artery (AbICA) is a rare congenital variation. It is considered a collateral pathway formed as a result of agenesis of the first cervical ICA segment. Abnormal vessel develops from fusion of the inferior tympanic branch of the ascending pharyngeal artery with the caroticotympanic artery [6,7]. AbICA is diagnosed in transverse CT slices based on the following criteria: 1) posteriorly, ICA borders on the jugular bulb and is reduced in diameter, 2) an enhancing mass is visible in the hypotympanum, 3) bony plate separating ICA from tympanic cavity is absent, 4) vertical segment of carotid canal is absent [6]. It is more common in women (90% of AbICA) and more frequently right-sided [8]; in 30% of cases it is accompanied by persistent stapedial artery [9]. This anomaly is usually asymptomatic. It sometimes causes tinnitus or conduction deafness. Clinically, it may resemble osteosclerosis, angioneuroma or other vascular malformations. A pathological mass may be visible behind the tympanic membrane in otolaryngological examination. This variation may be important when making a tympanic membrane incision or during middle ear surgery. Its presence is related to the risk of injury and severe intraoperative bleeding [6].

Persistent stapedial artery (PSA) is a remnant of fetal development. During fetal life this vessels connects branches of future facial artery with internal carotid artery. PSA is a rare variation observed in 0.48% of temporal bone sections [7] in post-mortem examinations. However, most authors report more rare occurrence of this variation – between 1:5000 and 1:10000 (0.02–0.01%) of cases identified during surgical procedures [7]. Its presence is associated with abnormal ICA course. Some authors suggest that abnormal course of internal carotid artery is the result of traction exerted by the stapedial artery, but not everyone supports this view [6]. Currently the hypothesis of secondary fusion ensuing from segmental ICA agenesis gains increasingly more acceptance [10]. Persistent stapedial artery arises from the petrous segment of ICA or AbICA, then runs anteriorly and superiorly through the tympanic cavity, supplying blood to the middle meningeal artery. PSA may be identified accidentally as a pulsatile mass during middle ear surgery. In angiographic studies absence of normal middle meningeal artery may suggest this variation. Under normal conditions middle meningeal artery and vein run through foramen spinosum, thus absence of the artery entails the absence of this foramen as well. CT changes encompass the following: absence of foramen spinosum on the side of PSA, soft tissue lesion in the proximal part of tympanic segment of the facial nerve. It is important to recognize this variant when differentiating with facial nerve tumors and the diagnosis influences planning of laryngological surgery in the middle ear region. Most of the identified PSAs are asymptomatic,

Figure 1. Congenital absence of the ICA. Lateral MIP projection of CT angiogram shows long narrowing of the cervical segment of the right internal carotid artery (ICA). The CCA and ECA indicate common carotid artery and external carotid artery respectively (own material).

although tinnitus and dizziness may occur. Surgical ligation of the artery or endovascular obliteration should be considered in order to reduce the symptoms [10,11].

Congenital absence of ICA is a rare congenital anomaly. Agenesis occurs in less than 0.01% of the population (estimated data) [12,13], while bilateral absence of the artery is seen in less than 10% of cases of agenesis [14,15]. Prevalence of hypoplasia is 0.079% [14,15]. In retrospective analysis of a series of MR and angiographic examinations either the absence or hypoplasia of ICA was diagnosed in 0.13% [15]. The term "absence" is more common and encompasses a spectrum of developmental abnormalities such as: agenesis, aplasia and hypoplasia. Agenesis and aplasia signify total absence of the vessel. Hypoplasia is characterized by ICA narrowing along its entire course as a reflection of incomplete development (Figure 1). Hypoplastic internal carotid artery may end as the ophthalmic artery. Diagnosis is stated by visualizing a narrowing or absence of bony carotid canal (Figure 2). It allows for differentiating between inborn and acquired absence of the artery due to: chronic dissection, fibromuscular dysplasia or atherosclerotic stenosis. Many patients may be asymptomatic. Internal carotid artery variants are often (24–34%) accompanied by aneurysms [12,16]. They are formed due to hemodynamic disturbances or genetic changes lying at the background of the anomaly. Congenital absence of ICA may be also associated with anencephaly or inborn Horner syndrome. It should be considered when planning an endarterectomy or gaining transsphenoidal surgical access, during which transsphenoidal collateral circulation may be damaged [12].

Other variants of internal carotid artery include: duplication, ICA fenestration, high or low branching of carotid artery (from Th2 to C1 level). Rarely, ICA may originate directly from the aorta [14]. It may give lateral branches:
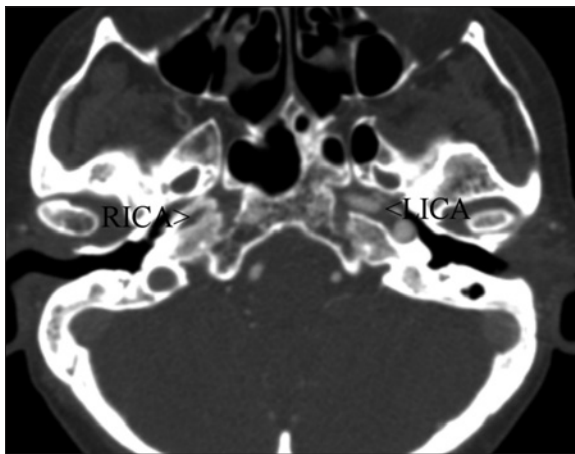
Figure 2. Congenital absence of the ICA. Axial CT image (the same patient) of the skull base shows hypoplastic right carotid canal (RICA). The LICA indicates the normal left carotid canal (own material).

ascending pharyngeal artery, superior thyroid artery, persistent stapedial artery or carotid-vertebrobasilar anastomoses. Kinking and looping of the extracranial part of internal carotid artery occur in 15% of angiograms [14].

Fenestration is also known as partial or segmental arterial duplication. It involves segmental division of a vessel to form two separate lumens with endothelial walls. These lumens may be surrounded by the same adventitia or have separate external laminae. Therefore, there are two morphological types of fenestrations: 1) small, slit-like fenestration or 2) large, lenticular fenestration. It is a developmental variant ensuing from disruption of fusion of a multi-channel network of vessels that develop into vessels during fetal life. Fenestration may occur in all cerebral arteries. However, it most commonly involves the Circle of Willis. Prevalence reported in the literature differs depending on imaging methods and location. In large post-mortem and surgical studies fenestrations in the area of anterior communicating artery are reported in about 40% of subjects [17] (Figure 3). Their prevalence in the ACA region as diagnosed in 3-dimensional rotational angiography is about 28% [18]. In Angio-CT imaging of entire cephalic circulation it is diagnosed in 11% of cases [19]. In large studies performed with classical angiography prevalence of fenestrations is 0.07–0.7% [18]. In a large study based on the analysis of Angio-CT studies in Polish population reported prevalence of fenestrations was 3.5%. They were most often visualized in the basilar artery (45%), anterior cerebral artery (40%) and anterior communicating artery (10%) [20]. These differences ensue from different ability to visualize small vessels. Many fenestrations are built by very thin vessels, about 0.1–0.3 mm in diameter. Coexistence of fenestrations with other vascular malformations, particularly with aneurysms, is well documented in literature [17,21]. Typically, aneurysms are associated with proximal fragment of the fenestration. It is most likely due to a so-called "medial defect." It is a focal smooth muscle defect in arterial intima media at a bifurcation site. Hemodynamic turbulences also appear at the place of branching. Association of fenestrations with aneurysms is suggested on the basis of coexistence of fenestrations with a rare type of aneurysm located in the inferior part of basilar artery. Descriptions of atypically located fenestrations accompanied by aneurysms seem to support this association [17,21].

Anterior Arteries

Some degree of asymmetry of anterior arteries is present in 80% of patients [22]. Common variants of anterior cerebral arteries include aplasia or hypoplasia of A1 segment. Hypoplasia is found in 10% and aplasia in 1–2% of post-mortem examinations [23], while Angio-MR demonstrated hypoplasia of A1 segment in 3% and of A2 segment in 2% of cases [24]. In such cases the contralateral A1 segment of ACA is dilated and through dilated anterior communicating artery supplies blood to the entire area vascularized by anterior cerebral arteries. This variant increases the risk and extent of neural tissue ischemia in the frontal lobe region during intravascular procedures performed in the area of anterior communicating artery or during ischemic episodes. Anterior cerebral artery fenestration is another variant occurring in this area. Its prevalence in the A1 region is 0–4% as described in anatomical studies, 0.058% as described in angiographic studies [23,25], and 0.8% [25], 1.2% [24] in MR vascular sequences. A2 fenestration was found in 2% of fetal post-mortem examinations [23].

Azygos anterior cerebral artery (azygos-ACA) is a rare variant involving a common trunk in the A2 segment (above the anterior communicating artery) (Figure 4). Its prevalence is determined at 0.3–2% on the basis of post-mortem studies and angiography [27]. Such structure of anterior circulation is typical of primates. Baptista distinguished three types of this anatomical variant. Type 1 involves an "unpaired," single ACA. In Type 2 there is a "bihemispheric" anterior cerebral artery, which gives branches to both hemispheres (sometimes, a second, hypoplastic ACA or a second A2 ACA segment is present, which ends below the level of genu of corpus callosum). In Type 3 there is an additional vessel arising from anterior communicating artery accompanied by two hypoplastic ACA arteries in the A2 segment [26]. This type is most common [26, 27]. It is associated with a large number of cerebral anomalies such as: agenesis of corpus callosum, prosencephalic cysts, hydranencephaly, lobular holoprosencephaly, septo-optic dysplasia (blindness, optic nerve atrophy, absence of septum pellucidum) and aneurysms of the distal part of anterior cerebral artery. Azygos-ACA is considered an important predictor of bilateral frontal strokes [28]. Saccular azygos ACA aneurysms are relatively common – 13–71% [28]. There are publications describing the occurrence of atypical aneurysms in this place such as: fusiform aneurysms with wide base, complex branching or thrombi. They might cause complications during endovascular procedures or disqualify from this part of treatment [27].

Accessory/triplicated anterior cerebral artery is an additional vessel arising from the anterior communicating artery (Figure 5). It occurs with a frequency of 2–13% [23]. Its presence in Angio-MR studies is reported in about 3% of cases [24]. This anatomical variant most likely represents persistent median callosal artery [23]. It is important to describe this variant before clipping of anterior communicating artery aneurysms.
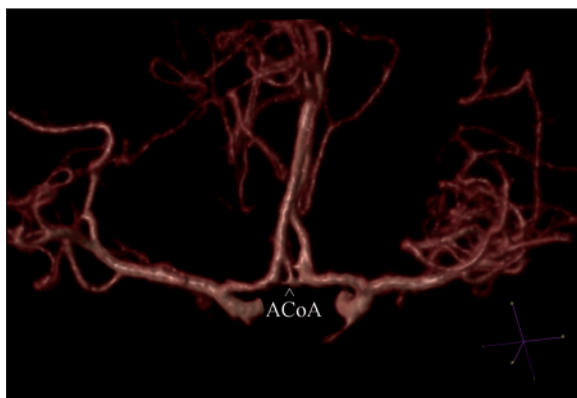
Figure 3. Fenestration. Posterior VR projection of CT angiogram shows dual fenestration of the anterior communicating artery (ACoA) (own material).
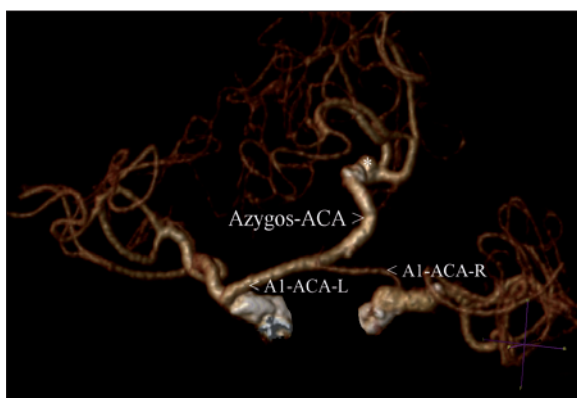

Figure 4. Azygos anterior cerebral artery. Posterior VR projection of CT angiogram shows an unpaired A2 segment of the anterior cerebral artery (Azygos-ACA) and saccular aneurysmal dilatation on the A2 and A3 junction (*). CT angiogram demonstrates a hypoplastic A1 segment of the right anterior cerebral artery (A1-ACA-R). The A1-ACA-L indicates a normal A1 segment of the left anterior cerebral artery (own material).

Figure 5. Triplication of the anterior cerebral artery. Postero-lateral VR projection of CT angiogram shows triplicated ACA. There are three A2 segments (<) that arise from the anterior communicating artery. Note saccular aneurysmal dilatation of the anterior communicating artery (*). The A1-ACA indicates normal A1 segments of the anterior cerebral arteries (own material).

Figure 6. Recurrent artery of Heubner. Superior VR projection of CT angiogram shows the anterior cerebral artery with a smaller-caliber accessory cerebral artery (RAH) with cortical branches that arises from the junction of the A1-A2 segment. This accessory artery can be named as a recurrent artery of Heubner or also as accessory middle cerebral artery (type-3 according to Manelfe). The < indicates cortical branches of the left middle cerebral artery. Note contralateral occlusion of the internal carotid artery and a proximal part of the M1 segment of the right middle cerebral artery (MCA-R). The A1-ACA indicates A1 segments of the anterior cerebral arteries (own material).

Anterior communicating artery hypoplasia/aplasia. Noninvasive imaging studies often fail to visualize the anterior communicating artery, but it does not mean that this artery is absent. Based on examination during surgical procedures reported incidence of this variant is estimated at 5% [23].

Anterior communicating artery fenestrations are frequent. In large post-mortem studies fenestrations have been reported with a frequency of 40%. As these vessels are very small, it is rarely possible to visualize fenestrations with the same frequency in imaging studies [17]. Their incidence in CT angiography is about 10% [20].

Infraoptic course of anterior cerebral artery is a rare variant. It is an anastomosis between internal carotid artery and arterial cerebral circulation. In this type of anomaly anterior cerebral artery arises from the internal carotid artery at a level of exit of ophthalmic artery. Horizontal position of the vessel below ophthalmic nerve is characteristic. It subsequently runs upward to the typical place of confluence with anterior communicating artery.
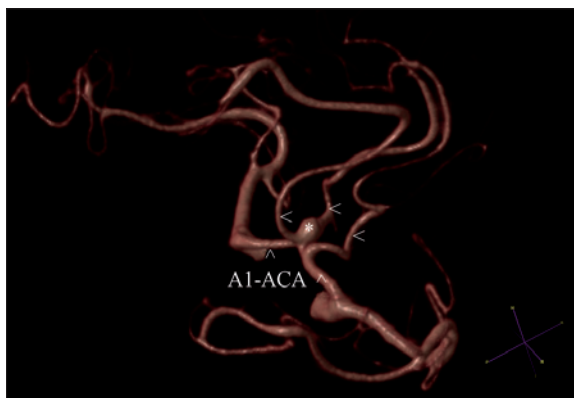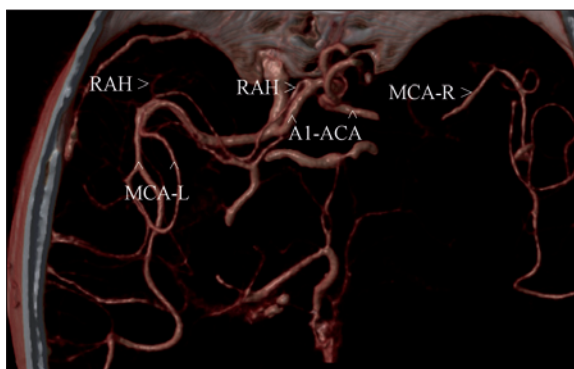
Embryological origin of this artery is unclear. It is most frequently identified on the right side, but there were described cases of its bilateral occurrence as well as coexistence with normal ipsilateral A1 ACA segment. It is often associated with anterior communicating artery aneurysms. Presence of this variant may be important for planning of aneurysm surgery in the ACA-ACoA region. Such procedure may require different surgical access or may be associated with impairment of vision [22].

Persistent Primitive Olfactory Artery (PPOA) – this variant involves an abnormal course of proximal anterior cerebral artery. It is rare. Persistent olfactory artery replaces the proximal fragment of ACA and joins with the distal segment of anterior cerebral artery. Such vessel runs

a characteristic course, takes a sharp 180 degree turn called a "hairpin turn." In this variant, the proximal fragment of anterior artery runs anteriorly along the olfactory tract, takes a turn upward and returns to the proper location of anterior cerebral artery [29]. It is associated with the absence of anterior communicating artery. There is an increased frequency of aneurysms in the region of the turn.

Persistent dorsal ophthalmic artery is a rare and rarely diagnosed variant. Ophthalmic artery arises from cavernous segment of internal carotid artery. This artery enters the orbit through superior orbital fissure instead of a normal course through the optic canal. There are two primary ophthalmic arteries during embryonic development – dorsal and ventral. Ventral ophthalmic artery departs from the future anterior cerebral artery and joins with the ICA in the course of development, forming an ophthalmic artery. On the other hand, dorsal artery undergoes regression, forming the inferolateral trunk (ILT) – a minute branch of ICA. This variant forms as a result of opposite to usual development of an artery supplying the eyeball from dorsal ophthalmic artery. Normal ventral artery undergoes regression in such cases [29,30].

Recurrent Artery of Heubner (RAH) is also called a median striatal artery. It is one of the largest perforators. It arises from a fusion of small perforators into one artery during fetal life. This vessel departs from the anterior cerebral artery in the A1 or A2 segment, most often in the area of anterior communicating artery. In 62.3% of post-mortem examinations the artery exits from the site of confluence of anterior artery and anterior communicating artery (Figure 6), in 23.3% from proximal A2 segment and in 14.3% from A1 segment [31]. The vessel is about 0.8mm in diameter and is often present in cerebral circulation under normal conditions [31]. The artery returns laterally along the A1 segment of anterior cerebral artery and supplies blood to the head of caudate nucleus, anterior part of lentilform nucleus and anterior horn of internal capsule. It is important for clinical practice, as ischemia of internal structures may occur during procedures in the region of anterior and anterior communicating arteries. Sometimes, this artery is a large vessel with cortical branches. In the opinion of some authors accessory MCA and duplicated MCA are considered RAH or RAH-like vessels with cortical branches. However, there were also descriptions of accessory middle arteries with perforators that coexist with Heubner arteries [32].

Middle Arteries

Vascular variants of middle cerebral arteries include doubled MCA, accessory MCA, early branching of middle cerebral arteries and fenestrations. According to the Teal classification surplus middle cerebral arteries are categorized into: accessory middle cerebral artery and duplicated middle cerebral artery [33]. Accessory middle cerebral artery begins at the A1 segment of anterior cerebral artery and runs parallel to MCA. Incidence is about 0.4% in angiography series [32]. In post-mortem examinations it is identified more frequently – about 3% [34]. Incidence cited by authors varies between 0.3 and 4.0% [32,35]. Duplicated MCAs begin in the distal part of internal carotid artery above the origin of anterior choroidal artery before the middle cerebral artery. Its prevalence is estimated at 0.2–2.9% based on angiographic and post-mortem studies [32,35]. Manelfe divided accessory middle cerebral arteries into 3 types: Type 1 corresponds to duplicated MCA, Type 2 – accessory MCA arising from proximal A1 ACA segment, and Type 3 – corresponding to accessory MCA arising from the distal A1 ACA segment near the anterior communicating artery [32]. Origin of those vessels is not entirely clear. Phylogenetically, MCA develops later than anterior communicating artery. ACA is considered a continuation of internal carotid artery. Thus, MCA may be considered a branch of anterior cerebral artery. Duplicated middle cerebral artery may be therefore treated as an abnormal, early branching of middle cerebral artery that arises from internal carotid artery, while the accessory artery as a true, anomalous middle cerebral artery arising from ACA [32]. Some authors identify an accessory middle cerebral artery as an enlarged recurrent artery of Heubner or RAH-like artery with cortical branches. There are reports of association between those lesions and formation of brain aneurysms, particularly in a branch in the A1 segment. Presence of accessory arteries is important for planning of neurosurgical treatment [32,35].

Early branching of middle cerebral artery is defined as cortical branches originating from middle cerebral artery segment before the bi- or trifurcation. The more proximal is the origin of such vessel, the wider the diameter. Knowledge of such variation may be important for planning of surgical procedures [35].

Conclusions

Variations of anterior cerebral circulation may be asymptomatic and may not produce complications, although some of them increase the risk of aneurysm formation and acute intracranial hemorrhages, play an important role in planning of neurosurgical procedures or may be mistaken for serious pathologies. Actual prevalence of those anomalies in general population remains unknown. Rapid technological progress in the field of imaging and availability of non-invasive angiographic studies will certainly lead to increased number of diagnosed anatomical variants among examined patients. Most common congenital variants are worth knowing in order to avoid complications they are related to.

References:

1. Brenner DJ, Hall EJ: Computed tomography – an increasing source of radiation exposure. N Engl J Med, 2007; 357: 2277–84

2. McKinney AM, Palmer CS, Truwit CL et al: Detection of aneurysms by 64-section multidetector CT angiography in patients acutely suspected of having an intracranial aneurysm and comparison with digital subtraction and 3D rotational angiography. Am J Neuroradiol, 2008; 29: 594–602

3. Villablanca JP, Rodriguez FJ, Stockman T et al: MDCT angiography for detection and quantification of small intracranial arteries: comparison with conventional catheter angiography. Am J Roentgenol, 2007; 188: 593–602

4. Skutta B, Fürst G, Eilers J et al: Intracranial stenoocclusive disease: double-detector helical CT angiography *versus* digital subtraction angiography. Am J Neuroradiol, 1999; 20: 791–99

5. Sanudo J, Vazquez R, Puerta J: Meaning and clinical interest of the anatomical variations in the 21st century. Eur J Anat, 2003; 7(Suppl.1): 1–3

6. Sauvaget E, Paris J, Kici S et al: Aberrant Internal Carotid Artery in the Temporal Bone. Imaging Findings and Management. Arch Otolaryngol Head Neck Surg, 2006; 132: 86–91

7. Roll JD, Urban MA, Larson TC III et al: Bilateral aberrant internal carotid arteries with bilateral persistent stapedial arteries and bilateral duplicated internal carotid arteries. Am J Neuroradiol, 2003; 24: 762–65

8. Caldemeyer KS, Carrico JB, Mathews VP: The radiology and embryology of anomalous arteries of the head and neck. Am J Roentgenol, 1998; 170: 197–203

9. Vattoth S, Shah R, Curé JK: A compartment-based approach for the imaging evaluation of tinnitus. Am J Neuroradiol, 2010; 31: 211–18

10. Silbergleit R, Quint DJ, Mehta BA et al: The persistent stapedial artery. Am J Neuroradiol, 2000; 21: 572–77

11. Wysocki J, Szymański I, Janiuk I: Anatomia topograficzna tętnicy szyjnej wewnętrznej w odcinku wewnątrzskroniowym Otorynolaryngologia, 2003; 2: 161–66 [in Polish]

12. Luk YS, Man EM, Sy AN: Bilateral Congenital Hypoplasia of the Internal Carotid Arteries. Singapore Med J, 2010; 51: 163–65

13. Nicoletti G, Sanguigni S, Bruno F et al: Hypoplasia of the internal carotid artery: collateral circulation and ultrasonographic findings. A case report. Ultrasound, 2009; 12: 41–44

14. Harrigan MR, Deveikis JP: Essential Neurovascular Anatomy. In: Harrigan MR, Deveikis JP (eds.), Handbook of Cerebrovascular Disease and Neurointerventional Technique Series: Contemporary Medical Imaging. Humana Press, New York, 2009; 1–87

15. Taşar M, Yetişer S, Taşar A et al: Congenital absence or hypoplasia of the carotid artery: radioclinical issues. Am J Otolaryngol, 2004; 25: 339–49

16. Lath N, Taneja M: Bilateral Congenital Hypoplasia of the Internal Carotid Arteries. J HK Coll Radiol, 2008; 11: 129–33

17. de Gast AN, van Rooij WJ, Sluzewski M: Fenestrations of the anterior communicating artery: incidence on 3D angiography and relationship to aneurysms. Am J Neuroradiol, 2008; 29: 296–98

18. van Rooij SB, van Rooij WJ, Sluzewski M et al: Fenestrations of intracranial arteries detected with 3D rotational angiography. Am J Neuroradiol, 2009; 30: 1347–50

19. Bharatha A, Aviv RI, White J et al: Intracranial arterial fenestrations: frequency on CT angiography and association with other vascular lesions. Surg Radiol Anat, 2008; 30: 397–401

20. Bożek P, Pilch-Kowalczyk J, Kluczewska E et al: Detection of cerebral artery fenestrations by computed tomography angiography. Neurologia i Neurochirurgia Polska, 2012; 3: 239–44

21. Kowada M, Yamaguchi K, Takahashi H: Fenestration of the vertebral artery with a review of 23 cases in Japan. Radiology, 1972; 103: 343–46

22. Given CA II, Morris PP: Recognition and importance of an infraoptic anterior cerebral artery: case report. Am J Neuroradiol, 2002; 23: 452–54

23. Dimmick SJ, Faulder KC: Normal variants of the cerebral circulation at multidetector CT angiography. Radiographics, 2009; 29: 1027–43

24. Uchino A, Nomiyama K, Takase Y et al: Anterior cerebral artery variations detected by MR angiography. Neuroradiology, 2006; 48: 647–52

25. Zhao H, Fu J, Lu Z et al: Fenestration of the anterior cerebral artery detected by magnetic. Chin Med J, 2009; 122: 1139–42

26. LeMay M, Gooding CA: The clinical significance of the azygos anterior cerebral artery (A.C.A.). Am J Roentgenol Radium Ther Nucl Med, 1966; 98: 602–10

27. Auguste KI, Ware ML, Lawton MT: Nonsaccular aneurysms of the azygos anterior cerebral artery. Neurosurg Focus, 2004; 17: E12

28. Huh JS, Park SK, Shin JJ et al: Saccular aneurysm of the azygos anterior cerebral artery: three case reports. J Korean Neurosurg Soc, 2007; 42: 342–45

29. Uchino A, Sawada A, Takase Y et al: MR angiography of anomalous branches of the internal carotid artery. Am J Roentgenol, 2003; 181: 1409–14

30. Tanaka M: Persistent primitive dorsal ophthalmic artery associated with paraclinoid internal carotid artery aneurysm. JNET, 2009; 3: 39–41

31. Loukas M, Louis RG Jr, Childs RS: Anatomical examination of the recurrent artery of Heubner. Clin Anat, 2006; 19: 25–31

32. Komiyama M, Nakajima H, Nishikawa M et al: Middle cerebral artery variations: duplicated and accessory arteries. Am J Neuroradiol, 1998; 19: 45–49

33. Teal JS, Rumbaugh CL, Bergeron RT et al: Anomalies of the middle cerebral artery: accessory artery, duplication, and early bifurcation. Am J Roentgenol, 1973; 118: 567–75

34. Jain KK: Some observations on the anatomy of the middle cerebral artery. Can J Surg, 1964; 7: 134–39

35. Otani N, Nawashiro H, Tsuzuki N et al: A ruptured internal carotid artery aneurysm located at the origin of the duplicated middle cerebral artery associated with accessory middle cerebral artery and middle cerebral artery aplasia. Surg Neurol Int, 2010; 1: 51