

## Anatomical variations of a1 and a2 segments of anterior cerebral artery

### ABSTRACT

Imaging of the cerebral arterial circulation is key in neurological diseases and knowledge of anatomical variants is very important in both radiologic interpretation and interventional procedures. We present a case of A1 segment hypoplasia and azygos A2 anterior cerebral artery anomalies with the aim of discussing the normal anatomy, embryological perspectives and the most common variants of anterior cerebral artery.

**KEY WORDS:** Anterior cerebral artery; azygos; hypoplasia,

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## INTRODUCTION

*Anterior cerebral artery (ACA) is the smaller of the two terminal branches of internal carotid artery (ICA). Along with the anterior communicating artery (ACoA), it forms the anterior component of circle of Willis. It runs and occupies mostly in the frontal aspect of the interhemispheric fissure supplying the orbitofrontal and medial hemispheric aspects of the brain. Parts of it curve around and over the corpus callosum, from the genu down to the splenium with central and cortical branches. The cortical branches form the vascular territory supplying the anterior two third of the medial hemispheres, corpus callosum, inferomedial surface of the frontal lobe and the anterior two third of the cerebral convexity adjacent to the interhemispheric fissure.<sup>1</sup> The anterior cerebral artery has three segments; The horizontal (A1) which courses medially over the optic chiasma and nerve to the midline to join its opposite or contralateral counterpart via the anterior communicating*

*artery.<sup>2</sup> This segment has two branches; proximal medial lenticulostriate that supplies the medial basal ganglia and the more distal recurrent artery of Heubner that supplies the inferomedial basal ganglia and the anterior limb of the internal capsule. The second segment which is known as the vertical (A2) segment extends superiorly in the interhemispheric fissure extending from the A1-Anterior communicating junction to the rostrum of the corpus callosum. It has two cortical branches; orbitofrontal and frontopolar arteries that supply the undersurface and inferomedial aspect of the frontal lobe. The third segment which is known as the callosal (A3) also has two branches, the pericallosal and callosomarginal.<sup>3</sup>*

## CASE REPORT

A 38 year old female, presented with one week history of right sided throbbing headache. There was no associated history of fever, vomiting, vertigo, trauma, seizure, or loss of consciousness and no history suggestive of any chronic illness or addiction. General physical and neurological examinations yielded no gross abnormal findings. Laboratory investigations revealed normal hematological and biochemical parameters. Contrast enhanced magnetic resonance imaging (MRI) showed no areas of restricted diffusion or susceptibility to suggest the presence of infarct or hemorrhage in diffusion weighted imaging (DWI) and gradient recall echo (GRE) sequences respectively (Figure 1 and 2). There was no evidence of micro bleed or calcifications on susceptibility weighted angiogram (SWAN) study (Figure 2). Magnetic resonance angiogram (MRA) showed narrowing of the transverse (A1) segment as well as a single vertical (A2) segment of the right anterior cerebral artery (Figure 3).

## RESULTS AND DISCUSSION

The development of some anatomical variants still remains controversial even though the basic principles of cerebral artery embryology are well-known.<sup>4,5</sup> Embryogenesis of the cerebral arteries begins at about five weeks of gestation. At this time of the gestational age, two chief primitive intracranial arteries develop: the internal carotid arteries (ICA) and the bilateral longitudinal neural arteries (BLNA). At the same time, many arterial branches develop and form transient anastomoses between these two main primitive trunks which are; trigeminal arteries, otic arteries, hypoglossal arteries, and proatlantal arteries.<sup>6,7</sup> The anterior cerebral arterial circulation (CAC) originates from the ICA, while the BLNA gives rise to the vertebrobasilar system.<sup>5</sup> Approximately this same

period of time, the ICA bifurcates into cranial and caudal divisions which is an important stage in cerebral artery embryogenesis. The cranial division later becomes the anterior CAC, which includes the ACA, anterior choroidal artery and middle cerebral artery (MCA) while the caudal division will later become the posterior communicating artery (PCoA) that forms definitive caroti-vertebrobasilar communicating artery that 'closes off' the cerebral arterial circulation posteriorly.<sup>8</sup>

Padgett's<sup>9</sup> theory explains the genesis of the anterior CAC and its most common variations. He explains that the embryogenesis of the anterior CAC is the result of two important stages: firstly, the development

of CAC from the cranial division of the ICA which has several branches supplying the anterior part of the brain and secondly, the regression of certain arterial segments and or persistence of certain anastomoses in utero and, in some cases, during ex utero development into adulthood. 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. The third 'ACA' follows the course of the two other 'ACAs' and is known as the median artery of corpus callosum (MACC). The MACC and ACoP should ordinarily regress and failure to do so for whatever reason (s) can lead to numerous anatomical variants, such as an accessory ACA as a result of persistence in MACC, or a double ACoA due to incomplete regression of the ACoP.<sup>6</sup> In the other hand, some segments that should persist may also regress abnormally giving rise to aplasia or hypoplasia of the proximal ACA segment. The complexity of adaptation process between the cerebral vessels, on the one hand, and the morphology and metabolic needs of the brain, on the other hand may be attributed to the dynamism of regression or persistence of certain segments resulting to anatomical variations.<sup>10</sup>

The anterior cerebral artery shows a considerable number of morphologic and angiographic anomalies. It is of importance to know such anomalies since they could have serious clinical consequences.<sup>11</sup> The most common anatomical variations of the A1 segment as previously reported, are hypoplasia with an incidence of 10–35%, fenestration 0.1–8% and an accessory MCA (0.2–4%). Kwak et al., 1980; Hillen 1987.<sup>12</sup> Aplasia, and low origin and infraoptic course of the A1 segment are quite rare in incidence Compton, 1962.<sup>13</sup>

Explanation for hypoplasia and aplasia is quite clear which is attributed to abnormal regression of the A1 segment, which should normally persist while genesis of fenestration of the A1 segment (or any other arterial branch) is controversial.<sup>14</sup> However, incomplete regression of embryological vessels as well as partial duplication or crossing with a non-vascular structure have been put forward for a possible explanation.<sup>5</sup> Agenesis and hypoplasia resulting in defective circulation has been reported in literature. That is to say, if the artery on one side is hypoplastic or even

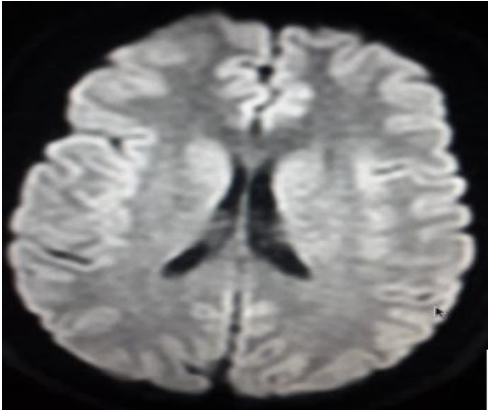
aplastic, the vascular insufficiency is usually compensated by arteries of the opposite side crossing over or giving branches to the defective side.<sup>15</sup>

Accessory middle cerebral artery (MCA) which originates from the A1 or A2 segments is said to be due to anomalous early ramification of the early branch of the MCA, which originates from the A1 or A2 portion of the ACA. As for infraoptic variant of A1 is due to persistence of the primitive ophthalmic arteries. Another explanation points to a persistence of embryological anastomosis between the primitive maxillary artery, that supplies the optic vesicle before the fifth week of gestation and that of the ACA.<sup>16</sup> This anomaly is one of the two unusual and important variation. It is associated with high prevalence (40%) of cerebral aneurysm.<sup>17</sup> Azygos or a single midline anterior cerebral artery is the second uncommon but important anterior cerebral artery anomalies that arises in A2 segment. It is seen in the holoprocencephaly spectrum.<sup>17</sup>

Niederberger *et al*<sup>18</sup> in their study, grouped azygos variant into five subtypes; Type I is the classical type of azygos subtype which is also the type seen in this study. This subtype is seen in the anterior interhemispheric fissure of brain with an incidence of 23% in relation to all the subtypes. Type II is a short median stem of azygos anterior cerebral artery. This variant further divides into two branches to supply both hemispheres separately with incidence of 1.8%. Type III shows 2 separate A2 segments, out of which one remained very short and end earlier and this variant hardly gives off branches. It is the most common subvariant of azygos ACA (30.1%). Type IV shows normal appearance of A2 segment except for the pericallosal arteries. The pericallosal arteries on both sides are the terminal branches of one. This was present in 23% of all subtypes. Type V shows a third azygos median A2 artery and this is the least in prevalence accounting for 0.9% of all subtypes.

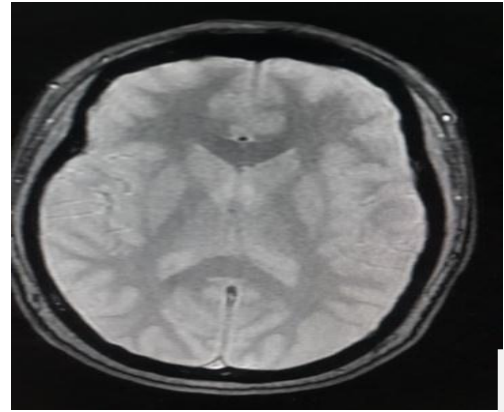
All these variations are usually associated with one another. That is to say if one vessel is thin or hypoplastic, the opposite vessel becomes large and dominant to compensate for the diminution of blood supply, however, this is the not case in this present study.





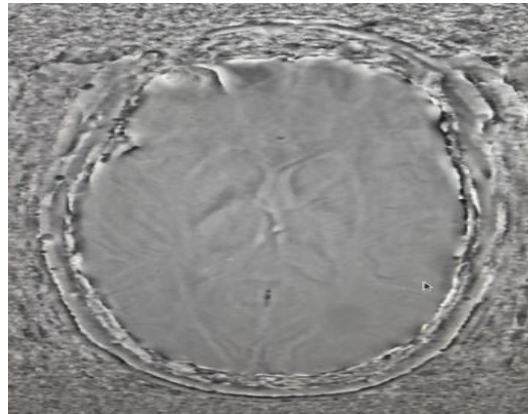
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Fig. 1 DWI showing no areas of diffusion restriction



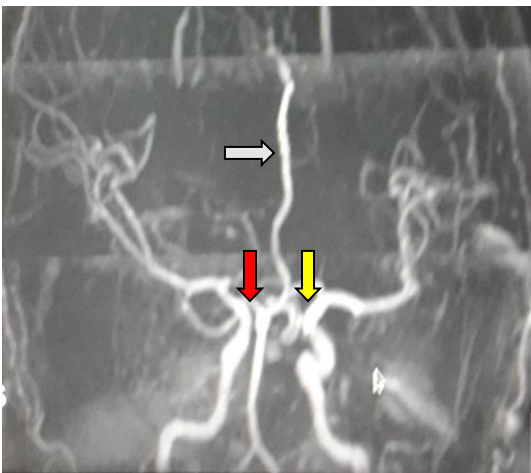
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Fig.2 GRE showing no areas of susceptibility.



3

Fig. 3. 3D SWAN showing no micro bleeds or calcifications



4a



4b

Fig. 4 a and b MRA showing hypoplastic A1 (red arrow), normal A1 segment (yellow arrow) azygos A2 segment (white arrow)

**CONCLUSION**

The incidence of anatomical variations of anterior cerebral artery is quite high as reported in literature. Knowledge of these anatomical variations is particularly important in the neurovascular

imaging for proper interpretation and interventional procedures.

**REFERENCES**

1. Chuang YM, Liu CY, Pan PJ, Lin CP. Anterior cerebral artery A1 segment hypoplasia may contribute to A1 hypoplasia syndrome. *Eur Neurol*. 2007; 57: 208-11.
2. Serizawa T, Saeki N, Yamaura A. Microsurgical anatomy and clinical significance of the anterior communicating artery and its perforating branches. *Neurosurgery* 1997; 40:1211-8.
3. Gurdal E, Cakmak U, Yalcinkaya M, Uzun I, Cavdar S. Two variants of the anterior communicating artery: a clinical reminder. *Neuroanatomy* 2004; 3:32-4
4. Riggs HE, Rupp C. Variations in form of Circle of Willis. *Arch Neurol* 1963; 8(1):8-14.
5. Pai SB, Kulkarni RN, Varma RG. Microsurgical anatomy of the anterior cerebral artery—Anterior communicating artery complex: An Indian study. *Neurol Asia* 2005; 10:21-
6. Goddard AJ, Annesley WD, Guthrie JA, Weston M. Duplication of the vertebral artery: report of two cases and review of the literature. *Neuroradiology* 2001; 43:477-80.
7. Ionete C, Omojola MF. MR angiographic demonstration of bilateral duplication of the extracranial vertebral artery: unusual course and review of the literature. *Am J Neuroradiol* 2006; 27:1304-6.
8. Lasjaunias P., Berenstein A., Ter Brugge K.G. Surgical neuroangiography clinical vascular anatomy and variations Berlin: Springer 2001:479-631
9. Ljiljana V, Ivan J, Slađana U, Milena, T. Remnants of embryonic arteries on the brain base: A case report. *World Journal of Neuroscience*, 2012; **2**; 133-137
10. Stefani MA, Schneider FL., Marrone AC, Severino AG, Jackowski AP, Wallace MC. Anatomic variations of anterior cerebral artery cortical branches *Clin Anat* 2000 ; 13 : 231-236
11. Kravenbuhl HA, Yasargil MG. Cerebral angiography. 2nd Ed. New York: Thieme Medical Publishers Inc, 1982:91-3.
12. Kwak R, Niizuma H, Hatanaka M, Suzuki J. Anterior communicating artery aneurysms with associated anomalies. *J. Neurosurg* 1980; 52:162-4.
13. Compton MR. The pathology of ruptured MCA aneurysms. *Lancet* 1962; 2:421-38.
14. Van Overbeeke JJ, Hillen B, Tulleken CAF. A comparative study of the circle of Willis in fetal and adult life. The configuration of the posterior bifurcation of the posterior communicating artery *J.Anat.* 1991:176; 45-54.
15. Uchino A, Nomiyama K, Takase Y, Kudo S. Anterior cerebral artery variations detected by MR angiography. *Neuroradiology* 2006; 48:647-52.
16. Saidi H, Kitunguu P, Ogengo JA. Variant anatomy of the anterior cerebral artery in Adult Kenyans. *Afr J Neurol Sci* 2008; 27:97-105.
17. Anne G. Osborn Osborn's Brain: Imaging, Pathology, and Anatomy (1st edition) Amirsys, Salt Lake City, UT, 2012. 1300 Pages
18. Niederberger E, Gauvrit JY, Morandi X, Carsin-Nicol B, Gauthier T, Ferré JC. Anatomic variants of the anterior part of the cerebral arterial circle at multidetector computed tomography angiography *Journal of Neuroradiology* 2010; 37(3):139-147.