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RESEARCH ARTICLE

VARIATIONS OF ANTERIOR CEREBRAL ARTERY AND ANTERIOR COMMUNICATING ARTERY: THEIR EMBRYOLOGICAL CORRELATION AND CLINICAL SIGNIFICANCE

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ABSTRACT

Background & Objectives: To study variations of the Anterior cerebral artery –anterior Communicating complex (ACA- ACoA complex). Anomalies of the ACA- ACoA like fenestrations and median trunk – Azygous ACA and Bihemispheric ACA are exceedingly very rare anomalies. Any variation in the vessels of anterior part of Circle of Willis influences the change in the hemodynamics of blood supply of frontal lobe. The awareness of this rare anatomical variant is important considering today advanced imaging modalities and give consideration to its potential serious complications with fellow clinicians.

Method: The study variations of the Anterior Cerebral Artery (ACA) and Anterior Communicating Artery (ACoA) of the circle of Willis at the base of the brain were studied in 50 human brain specimens.

Results: In the anterior part of the circle, complete absence, double ACoA and fenestrated ACoA was found in one case each 2%. The 'H' complex was seen more commonly in the ACoA - ACA complex found in 80%, 'V' shaped complex in 8%. Absence of A1 segment on the right side was seen in one case (2%) and hypoplasia of the A1 segment was seen in another case (2%). In one case (2%), both the A2 segments arose from contralateral ACA – Bihemispheric ACA. An Azygous anterior cerebral artery/ median trunk ACA was found in 6.66% of the cases.

INTRODUCTION

The Anterior Cerebral Artery (ACA): It is the smaller of the two terminal branches of the internal carotid artery and starts at the medial end of the lateral cerebral sulcus. It passes forwards and medially above the optic nerve to the commencement of the longitudinal fissure. Here it comes in close relationship with the opposite artery and is joined to it by a short transverse trunk which is the Anterior Communicating Artery (ACoA). The part of the anterior cerebral artery (ACA) before the communication is called the A1 segment (pre-communicating or horizontal), while the part of the anterior cerebral artery after this communication is called the A2 segment (post-communicating or vertical).

The Anterior Communicating Artery (ACoA): It is a small vessel that lies at the end of the inter-hemispheric fissure. It connects the A1 segments of the anterior cerebral arteries of both the sides in the midline across the commencement of the longitudinal fissure. Sometimes it is double, fenestrated or multi-channeled. This anastamosis forms the ACA-ACoA complex. This arrangement facilitates the potential cross circulation between the two carotids of both sides via Circulus Arteriosus. An illustration by Luzsa in 1974 describes the types of variations of ACoA which are, as follows: Type I: Normal; Type II: Complete absence of artery; Type III: A trunk formation; Type IV: Plexiform connection; Type V: A duplication; Type VI: A median artery of corpus callosum (Luzsa, 1974).

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MATERIALS AND METHODS

The present study was conducted in the Department of Anatomy, J. J. M. Medical College, Davangere and M V J Medical College & Research Hospital, Hoskote Bangalore. The study variations of the Anterior Cerebral Artery (ACA) and Anterior Communicating Artery (ACoA) of the circle of Willis at the base of the brain were studied in 50 human brain specimens. The measurement of the lengths of the components of the circle of Willis were taken: right and left A1 segment of ACA from the point of its origin to the point of the ACoA communication; anterior communicating artery from the point of communication between right A1 segment to the left A1segment of ACA. A detailed study of the arteries was undertaken *in situ* to avoid the errors which may have occurred. Photographs were taken on a Nikon 5.0 mega pixel camera, 3x magnification lens and digitally painted on Adobe Photoshop version: 9 software CS-3; color code: cb1616 with 50% opacity of the color used

RESULTS AND DISCUSSION

Name of the arterial segment	Length (mm)		Diameter (mm)	
	Right	Left	Right	Left
ACA-A1 Segment	14.5 ± 0.53	14.5 ± 0.18	1.94 ± 0.08	2.07 ± 0.08
ACoA	4.5 ± 0.23		2.05 ± 0.17	

Specimen 2: Anterior part: ACoA is fenestrated; all the components, A1 segments and ACoA present; normal in their origin & size.

Specimen No: 7Circulus Arteriosus: Circle: Complete; **Shape**: Polygon; Symmetric. Its Anterior part: ACoA absent; A1 segments of both sides unite to form a median trunk (Azygous ACA) in the midline before entering the longitudinal fissure. Its length before entering the fissure is 4.3 mm and external diameter is 2.3mm.

Specimen No: 8Circulus Arteriosus: Circle: Complete; **Shape**: Polygon; Asymmetric. Its Anterior part: ACoA absent; A1 segments of both sides unite to form a median trunk (Azygous ACA) in the midline before entering the longitudinal fissure. Its length before entering the fissure from the point of union is 10.3 mm and external diameter is 3.3 mm.

Specimen No: 17 Circulus Arteriosus: Circle: Complete. **Shape**: Nonagon; Asymmetric. Its Anterior part: ACoA - ACA complex - 'H' shaped; double ACoA found - ACoA-1, ACoA-2, length 5.2 and 5.5mm respectively and their external diameter is 1.1 mm each; A1 segments present, normal in their origin & size.

Specimen No: 20 Circulus Arteriosus: Circle: Incomplete. A1 - R absent; A2 segments on both sides originate from ACA - L, A1 segment at the longitudinal fissure divides into three branches, two A2 segments which run in the longitudinal fissure and a branch which run into to the sylvian fissure.

Specimen No: 27 Circulus Arteriosus: Circle: Complete. **Shape**: Polygon; Symmetric. Its Anterior part: ACoA - ACA complex: ACoA absent; an Azygous ACA (median trunk) is formed by the union of A1 segments of both the sides in the midline. A1 segments present; normal in their origin & size.

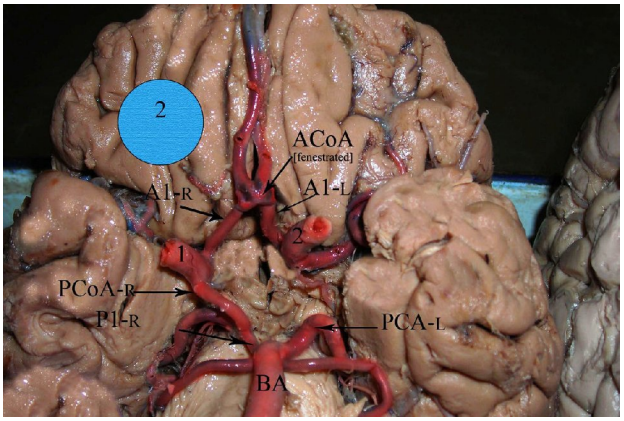

Figure 1. Circle of Willis – Fenestrated ACoA

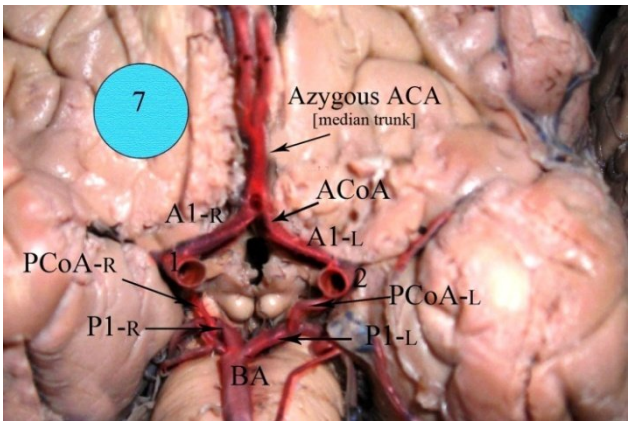

Figure 2. Circle of Willis – Azygous ACoA(Median Trunk)

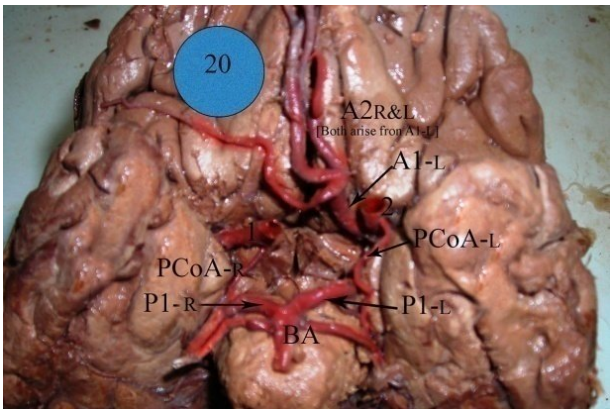

Figure 3. Circle of Willis – Bihemispheric ACA

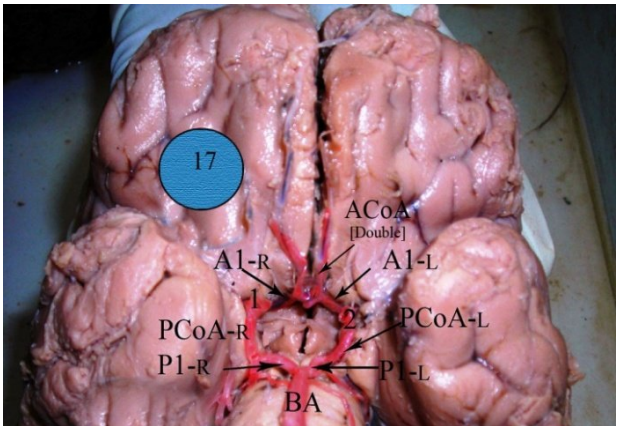

Figure 4. Circle of Willis – Double ACoA

Table 1. Comparisons of the studies on Azygous ACA

S.No.	Authors	Azygous ACoA
1.	Fawcett& blackford[2]	0.14%
2.	Luzsa[1]	1.2%
3.	Bergmann[5]	1.16%
4.	Serizawa[12]	3%
5.	Osborn[13]	0.2-4%
6.	Kanchan K[11]	0.9%
7.	Present study	6.66%

DISCUSSION

In the present study, among the total ACoA-ACA complexes, ACoA was present and single in 92%. The most common was a 'H' shaped complex found in 80%, 'V' shaped complex in 8% and fenestrated in 2%, plexiform or double in 2% of the cases each, Azygous ACA was found in 6.66% of the cases Windle reported ACoA absent in 3% and double in 3% of the cases (Windle and Bertram 1887). Fawcett &Blackford's report indicated that the artery was single in 92.1%, absent in 0.14% and double in 7.2% of the cases (Fawcett and Blackford 1905). Vare&Bansal studies showed that ACoA was absent in 1.14%; double in 10.28% and ACoA-ACA complex was 'H' shaped in 1.14% (Vare and Bansal 1970). Luzsa illustrated that the ACoA was absent in 0.3% of the cases (Luzsa 1974). From an unpublished paper by seven investigators Bergmann et.al, it was illustrated that the artery was absent in 0.22%; ACoA was single in 81% (Bergman *et al.,* 1988).Orlandini *et al.* reported 7% of the cases not having an ACoA (Giovanni *et al.,* 1985). RadomirVuccetic in his report states that segmental duplications and fenestrations ACoA- ACA complex are not only uncommon but these segmental duplication of the ACA is a rare embryonic malformation, arising from incomplete fusion of the small branches which are the precursors of the artery at about 35 day post-conception, and further states that the frequency of fenestrated ACA's with aneurysm formation is unknown, but is not low (Radomir 1998). Gast& Von Rooji in their studied stated a presences of fenestration of the ACoA in 5.3% and out of 5.3% fenestrations, 83% were associated with 1 or more aneurysms of the ACoA. On the wholefenestration, which accounts for an incidence of ACoA with aneurysms on presentation, was about 7.9% (Gast ?). Another radiologic study showsprevalence ofduplication of the ACoA is about 18%, whereas fenestration of the ACoA is present in 12%–21% of the population.

A1 segment: The present study A1 Segment was present in 96%, Absent A1 segment on right side in 2% of the cases , Hypoplasia of A1 segment (right) 2% , A2 Segment arising contralateral anterior cerebral is about 2%.Luzsa states the absence of the A1 in 0.7 - 11% and hypoplasia in 8 – 15% (Luzsa 1974). Windle reports the absence of the A1segment anterior cerebral artery in 1% (Windle and Bertram 1887). Macchiand Stephen & Johnalso reports 2% hypoplasia of the A1 segment (Macchi *et al.,* 1996; Stephen *et al.,* 1991). Arthur reports 9.61% each of aplasia & hypoplasia of A1 segment (Arthur *et al.,* 1996). Osborn states the absence of the A1 segment in 1 - 2% (Anne Osborn 1999). Kanchan K also states A1 segment absent in 0.4%, hypoplasia 1.7% of cases (KanchanKapoor *et al.,* 2002).

Median / Azygous ACA: Table I shows the percentages of Median truck / Azygous ACoA other researchers and the present study.Majorielemay in his paper explaining the phylogeny of ACA states in lower animals & mammals there is absences of anterior communicating artery instead ACA'sunties in the mid line form an Median trunk-Azygous ACA also further explains the three types of anomalies of the Azygous ACA Fig (5) a) True Azygous – median trunk ACA. b) 2ACA's but only one supply both hemispheric ACA –A Bihemispheric ACA. c) Triple ACA – accessory median artery arising from the median artery (Majorielemay 1966).

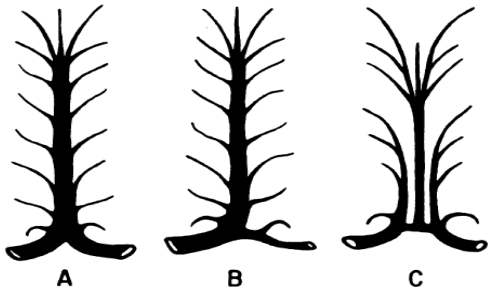

Figure 5. ***Courtesy: Majorielemay et al.*** **a) True Azygous – median trunk ACA**

b) Bihemispheric ACA – major arteries arising from single ACA
c) Triple ACA – accessory median artery arising from the median artery (Majorielemay 1966)

ErgutOzakul states that anterior cerebral arteries arise at about the fortieth day of gestation. The fusion of two anterior cerebral arteries arising from the medial branch of the prirnitive olfactory artery, which is known as Azygous ACA and the persistence of this primitive fusion in adult life, presents itself as median trunk ACA to short distance then artery splits to two A2 segments of the respective hemisphere. He further states that such azygous arteries are often accompanied by aneurysms and midline anomalies (Ertuğ ÖZKAL *et al.,* 1990). Scott Jennings in his case report on Azygous ACA discusses the clinical significance as the presences of Azygous ACA causes the alteration of arterial hemodynamics of the frontal lobe and the increased incidence of malformations like agenesis of the corpus callosum, hydrocephaly, saccular aneurysms and Arterio-venous malformations (Scott Jennings 2011).

In our study, Only one case of both A2 segments arising from contralateral A1 segment was seen, findings given in the results. There is hardly any literature available on such variant; a nother case was reported by Murai which describes that both A1 segments arising from one side only whether this type of anomaly is same as the Bihemispheric ACA described by Marjorie is debatable and for further research (Yasuo Y Murai *et al.,* 2005; Majorielemay 1966). Simon in his article on normal variants of cerebral circulations explains the variant similar to the present case as Bihemispheric ACA with the contralateral A2 segment providing the major arterial supply bilaterally to the anterior cerebral artery territory and also states that prevalence of Bihemispheric ACA anomaly is2%–7% (Simon *et al.,* 2009). These anatomical variations like Azygous ACA and Bihemispheric ACA are very important; and are prone for aneurysms affecting the circulatory dynamics in their territory of blood supply.

Conclusion

This study was undertaken to through light on the variations of the ACA and ACoA. The Anomalies of the ACoA –ACA complex like fenestrations, duplications, median trunk – Azygous ACA and Bihemispheric ACA discovered at time of

imaging or at the time of routinedissection are not uncommon but also havemuch significant clinical implication. There is need for further study on these exceedingly rare anomalies; many case reports have been reported and very less literature available on anatomic studies as most of these variations are discovered at the time of imaging. It is hoped the sharing of the present study on anatomical variants of ACoA –ACA Complex with underlying embryological correlations will alert fellow clinicians; as these above variantshave a considerable alteration in the arterial hemodynamics of the frontal lobe and the increased incidence of malformations like agenesis of the corpus callosum, potential neurological and neurosurgical conditions like occlusion will result in bilateral ACA territory stroke.An awareness of these malformations will allow more informed decisions in preserving the vessel from injury during various neurosurgical processes. It also important for the radiologist to be aware of such multiple variations of ACoA – ACA complex for pin pointing the exact anomalies by using high –end diagnostic imaging technologies like Multi-detector CT cerebral angiography and 3 D cerebral Angiographic imaging.

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