ORIGINAL ARTICLE

VARIATION OF POSTERIOR COMMUNICATING ARTERY IN HUMAN BRAIN: A MORPHOLOGICAL STUDY

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ABSTRACT

Background: The circle of Willis, present in the interpeduncular cistern at the base of the brain, is the major source of blood supply of the brain. Posterior communicating artery is its most important component which connects vertebro-basilar and carotid arterial system. Absence or hypoplasia of this artery will influence development of collateral channel in case of obstruction or narrowing of the major cerebral arteries, thus explaining the different neurological symptoms and the prognosis of the disease.

Material & Methods: We have studied 60 brain specimens which were collected from consecutive adult donated cadavers from the Department of Anatomy, Mamata Medical College, khammam, A.P, India and Department of Anatomy, Medical College, Kolkata, W.B, India. Posterior communicating artery was cleaned and detailes were recorded.

Results: 38.3% of those were found to have normal posterior communicating artery. In 38.2% cases it was found to be absent and in 23.3% cases hypoplastic. Details of the findings have been presented and compared with those of the previous workers on this topic. Clinical implication of the variation and embryological explanation has also been attempted. Conclusion: Exact knowledge of the variation of Posterior communicating artery is essential not only to explain various neurological symptoms but also for successful micro-vascular surgery in this region.

KEY WORDS: Posterior communicating artery, Circle of Willis, Aplasia, Hypoplasia.

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INTRODUCTION

Circle of Willis is a major source of blood supply of human brain. It is contributed by blood of both the internal carotid arteries (ICA) and the vertebro-basilar arterial system via anterior, middle and posterior cerebral arteries. The cerebral part of ICA terminates by dividing into anterior and middle cerebral arteries. The anterior cerebral arteries of the two hemispheres are joined together by the anterior communicating artery. Posteriorly, the basilar artery divides into two posterior cerebral arteries at the upper border of pons and each communicates with the ipsilateral ICA by the posterior communicating artery (PCA).[1] The PCA actually is the collateral channel between the two systems. When the carotid and basilar arterial system are normal, the pressure of both the streams will be equal in PCA resulting in almost no mixture of blood. However, if any of the system has a blockage of any degree, blood will flow from the normal arterial system to that with lower pressure via the communicating arteries. So circle of Willis is an alternate route of blood flow which acts as a safety mechanism, allowing adequate blood supply to brain areas even when blockage occurs in arterial system preventing a cerebro-vascular accident. Though classical circle described by Willis is complete and symmetrical, later on it was found to be extremely variable in nature. Don F Schomer et al[2] has found a definitive correlation between narrow or absent PCA and cerebral infarction in persons with internal carotid artery occlusion. V Papantchev et al[3] has established that if there is no blood flow through PCA, patient may suffer from visual, endocrine or vegetative disturbances, hemiballism, decrease level of consciousness, impaired coordination, hemiplagia, hemianasthesia etc in cases of disturbances of any of those arterial system. As per Defelice et al[4] non-

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functioning PCA and idiopathic sudden hearing loss has a strong correlation. So, study of the variability of the PCA is essential both for academic and clinical aspects since they have important influences on the mode of presentation, plan of investigation and plan of treatment of various neurological disorders.

MATERIAL AND METHODS

For present study a total number of 60 brains were obtained from March 2009 to May 2011 via routine dissection for 1st year MBBS class. The brains were dissected from consecutive adult donated cadavers from the Department of Anatomy of two medical colleges, Mamata Medical College, Khammam, A.P and Medical College, Kolkata, West Bengal, India and were examined. After opening the calvarias, dura was incised and brains were detached at spinomedullary junction and preserved in 10% formalin. The base of brain was cleaned by removing the arachnoid and the component of Circle of Willis was dissected out. The origin, course and the termination of the PCA were noted and recorded. The external diameter of the vessels was taken with the help of divider and by the thread and scale. Individual specimen was serially numbered and photographs taken. Collected data was analyzed in Microsoft excel software. Any specimen with evidence of pathology or trauma of the brain and its blood supplying vessels that may have affected the topography of the arteries was excluded from the study. The study was done by brain of male cadaver alone, in order to control gender as one possible confounding factor.

RESULTS

Out of 60 brain specimens, 23 (38.3%) of them were having normal PCA in respect to its course and diameter. In rest 37 specimens (61.6%) PCA was either absent (bilateral, Fig.1 or unilateral, Fig.2) or hypoplastic. The average diameter of the said artery varied between 1.0 -1.5 mm. But in some specimens the vessel was very narrow, diameter being less than 0.5 mm, considered as hypoplastic which might be bilateral (Fig.3) or unilateral (Fig.4). The details of those are presented in table 1.

In some unilateral aplastic and hypoplastic

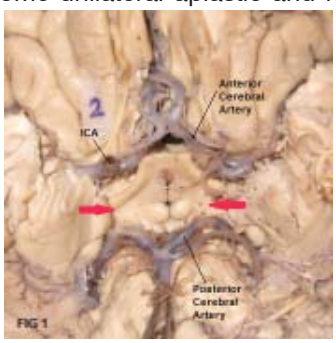

Fig. 1: Red arrows indicate bilateral aplasia of PCA.

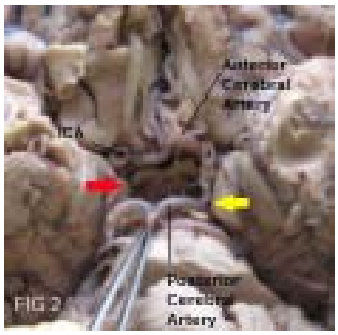

Fig. 2: Red arrow indicates unilateral aplasia of PCA, Yellow arrow indicates normal PCA.

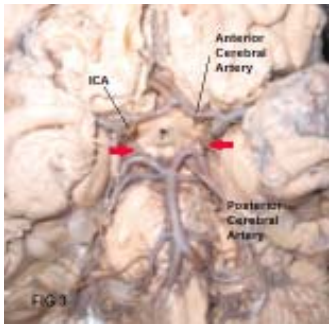

Fig. 3: Red arrows indicate bilateral hypoplasia of PCA.

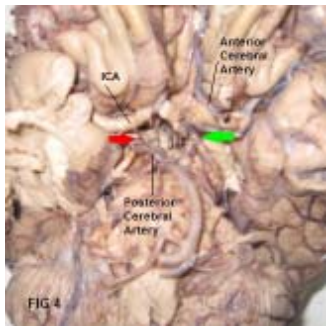

Fig.4: Red arrow indicates unilateral hypoplasia of PCA, Green arrow indicates dilated PCA.

Table 1: Incidences of variation of PCA in 60 cadavers brain specimens.

Nature of variation	Aplasia		Hypoplasia (<0.5mm)	
	Number	Percentage (n=60)	Number	Percentage (n=60)
Bilateral	10	16.6%	2	03.3%
Unilateral	13	21.6%	12	20.0%
Right Sided	8	13.3%	7	11.6%
Left Sided	5	08.3%	5	08.3%

PCA cases the other side was found to be dilated (diameter more than 1.5 mm). In one case where PCA was bilaterally absent, middle cerebral artery on the left side was having two roots while arising from the ipsilateral ICA.

DISCUSSION

Circle of Willis is formed at the base of the brain in order to preserve the cerebral perfusion well enough to avoid the symptoms of ischemia in case of blockage of any arterial system. Hence, aplasia or narrowing of PCA does not lead to any symptoms if other component vessels of circle are normally functioning and that explains its presence in otherwise normal persons. After occlusion (complete or partial) of a cerebral artery or its branches, it takes some hours to develop cerebral infarction. As cerebral blood flow declines, different neuronal dysfunctions appear at various thresholds. Symptoms of ischemia develop when blood flow decrease to less than half of its normal [5]. Normally infarction is avoided by the opening of the anastomotic channels within that time. When blood flow is quickly restored, brain tissue can recover fully and symptoms are only transient, called TIA (Transient Ischemic Attack).[6] Sometimes an adjustment time is required for collateral vessels to establish a proper circulation that can support the normal function of the supplying area. With increasing blood flow through them, these communicating arteries will gradually enlarge. Hence existence of previous vascular occlusion and anomalies of vascular arrangement must influence the outcome.[7] The occipital lobe, supplied mainly by the posterior cerebral artery naturally will suffer the most in aplasia or hypoplasia of PCA, as this will not take place when vertebro-basilar or carotid arterial system develops a blockage or a narrowing. Papantchev et al [3] had studied Willis circle amongst 112 cadavers and classified it into 6 types. They further established that the site of arterial obstruction varies in different types. According to them type Ia or hypoplastic PCA was 27.3% cases. In 2007, Kapoor et al [8] found aplasia of PCA in 1% and hypoplasia in 13.2% which is widely varied from our study report. Comparison of the findings of different workers with that of ours has been placed in table 2.

Though percentage of aplasia of PCA varied widely as evident from table 2, but in all the incidence of unilateral aplasia is more than that of bilateral ones. To define hypoplasia of the said artery various authors used different measurement. Ves-

Table 2: Comparison of findings of variation of PCA in 60 cadavers brain specimens.

Researchers	No of total cadavers studied	Aplasia (%)		Hypoplasia (%)	
		Unilateral	Bilateral	Unilateral	Bilateral
Present study	60	21.6%	16.6%	20.0%	3.3%
Windle[9](1887)	200	11%	1.5%	21.5%	3.5%
B. Efterkhar[10] (2006)	102	7%	3%	27%	33%
Ardakani[11] (2008)	30	19.9%	-	43.3%	-

Table 3: Comparison of incidences of hypoplasia of PCA in 60 cadavers brain specimens.

	Year	No		Diameter of PCA		
			Method of study	0.5mm	0.8mm	1mm
Present study	2011	60	cadaveric	23.3%		
Fatter & Moran[16]	1941	…….	cadaveric	23%		
P. Merkkola et al[17]	2006	87	Cadaveric (angiography)	14%		
Sylvia Kamath[15]	1981	100	cadaveric	10%		
Krabbe-Hartkamp et al[12]	1998	150	3D-TOF MR Angiography		28%	
Hoksbergen et al[13]	2003	50	3D- TOF MR Angiography		29%	
Windle[9]	1987	200	cadaveric			25%
V. Papantchev et al[3]	2007	112	cadaveric			30%

sel with diameter <1mm has been classified as hypoplastic by various morphometric studies.[9-11] Radiological studies by 3DTFMR angiography has considered hypoplasia if the diameter of the vessel is less than 0.8mm.[12-13] Hoksbergen et al[14] have described that the threshold diameter for supplying collateral flow is even smaller, namely between 0.4mm - 0.6mm by their color –coded duplex ultrasonography**.** We have taken 0.5 mm as the cut off limit following that of Kamath.[15] Findings of different workers utilizing different measurements to define the hypoplastic vessel have been placed in table 3.

Table 3 shows that, if 1mm (vessel diameter) is taken as a cut off limit for hypoplasia almost one fourth of the brain specimens become hypoplastic. Researchers who utilized 0.5mm as the upper limit of hypoplasia have found almost similar incidences, though it varied in between them a bit. The difference might be due to technical or racial reasons. Aplasia and hypoplasia has been found on both the sides, more common on the right side as per all the previous studies which matches ours (Table 4).

Embryological explanation

Table 4: Comparison of findings of variation of PCA on right and left side in 60 cadavers brain specimens.

Name of researchers	Aplasia		Hypoplasia	
	Right	Left	Right	Left
B. Efterkhar[10]	4 %	3 %	16 %	11 %
S K Ardakani[11]	16.6 %	3.3 %	26 %	16.6 %
Present study	13.3 %	08.3 %	11.6 %	8.3 %

ICA first gives ophthalmic branch to eye vesicle after reaching forebrain and then divide into 3 branches- anterior cerebral artery, middle cerebral artery & a third branch which goes towards occipital pole representing the initial posterior cerebral artery. Afterwards during development of brain basilar artery divides into two, the definitive posterior cerebral artery which join with the primitive posterior cerebral artery (third branch of ICA). Due to gradual increase in size of definitive posterior cerebral artery, there will be narrowing of initial posterior cerebral artery which later represent as PCA.[18] Modification of vessels occurs during postnatal development of brain due to increased functional demands in connection with rapid growth of occipital lobes.[19] Complete or incomplete disappearance of the initial posterior cerebral artery might result in an aplastic or hypoplastic PCA. Genetic factors play an important role in it.[20]

CONCLUSION

Our study shows a wide variability in the posterior arterial component of the circle of Willis. Absence and significant hypoplasia in a considerable number of cases have various clinical importances. Exact knowledge of the variation of posterior communicating artery is essential not only to explain various neurological symptoms but also for successful micro-vascular surgery in this region.

REFERENCES

1. Crossman AR. Vascular supply of the brain. In: Standring S, editors. Gray's Anatomy: The Anatomical Basis of Clinical Practice.39th ed. Edinburg: Elsevier Churchill Livingstone; 2005. P. 297-301.
2. Schomer DF, Marks MP, Steinberg GK, Johnstone IM, Boothroyd DB, Ross MR, et al. The Anatomy of the Posterior Communicating Artery as a Risk Factor for Ischemic Cerebral Infarction. N Engl J Med 1994; 330:1565-70.
3. Papantchev V, Hristov S, Todorova D, Naydenov E, Paloff A, Nikolov D, et al. Some variations of the circle of Willis, important for cerebral protection in aortic surgery: a study in eastern Europeans. Eur J Cardiothorac Surg 2007; 31: 982-9.
4. Felice CD, Capua BD, Tassi R, Mencattini G, Passali D. Non-functioning posterior communicating arteries of circle of Willis in idiopathic sudden hearing loss. The Lancet 2000; 356: 1237-8.
5. Allen CMC, Lueck CJ, Dennis M. Neurological disease. In: Colledge NR, Walker BR, Ralston SH editors. Devidson's Principles & Practice of Medicine. 21th ed. Edinburgh: Elsevier Churchill Livingstone; 2010. P. 1181-2.
6. Smith WS, English JD, Johnston SC. Cerebrovascular Diseases. In: Fauci AS, Braunwald E, Kasper DL, Hauser SL, Longo DL, Jameson JL, et al. Eds. Harrison's Principles of Internal Medicine 17th ed, vol II , McGraw-Hill Medical, New York, 2008: 2513.
7. Ropper AH, Brown RH. Cerebrovascular Diseases. Adams and Victor's Principles of Neurology 8th ed, McGraw-Hill Medical, New York, 2005:664.
8. Kapoor k, Singh B, Dewan LI. Variations in the configuration of the circle of Willis. Anat Sci Int 2008; 83(: 96-106.
9. Windle B. On the arteries forming the circle of Willis. J Anat Physiol 1887; 22: 289-93.
10. EfterkharB, Dadmehr M, Ansari S, Ghodsi M, Nazparvar B, Ketabchi E. Are the distributions of variations of circle of Willis different in different populations? Results of an anatomical study and review of literature. BMC Neurology 2006; 6:22.
11. Ardakani SK, Dadmehr M, Nejat F, Ansari S, Tajik BE, Khashab ME, et al. The cerebral arterial circle

(Circulus Arteriosus Cerebri): an anatomical study in fetus and infant samples. Pediatr Neurosurg 2008; 44:388-92.

12. Krabbe-hartkamp MJ, Van der grond J, De leeuw FE, De groot JC, Algra A, Hillen B, et al. Circle of Willis: morphologic variation on three-dimensional time-of-flight MR angiograms. Radiology 1998; 207:103-11.
13. Hoksbergen AWJ, Mojoie CBL, Hulsmans FJH, Legemate DA. Assesment of the collateral function of the circle of Willis: 3D-TOF MR angiography compared with TCCD sonography. Am J Neuroradiol 2003; 24: 456-62.
14. Hoksbergen AWJ, Fülesdi B, Legemate DA, Csiba L. The collateral configuration of the circle of Willis: transcranial color-coded duplex ultrasonography and comparison with postmortem anatomy. Stroke 2000; 31:1346-51.
15. Kamath S. Observations on the length and diameter of vessels forming the circle of Willis. J Anat 1981; 133:419–23.
16. Fetterman GH, Moran JJ. Anomalies of the circle of Willis in relation to cerebral softening. Arch Pathol 1941; 32: 251-7.
17. Merkkola P, Tulla H, Ronkainen A, Soppi V, Oksala A, Koivisto T, et al. Incomplete circle of Willis and right axillary artery perfusion. Ann Thorac Surg 2006; 82:74-9.
18. Hamilton WJ, Mossman HW. Human Embryology. 4thed. London: Macmillan Publishers Ltd; 1976.
19. Van overbeeke JJ, Hillen B, Tulleken CA. 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.
20. Milenkovic Z, Vucetic R, Puzic M. Asymmetry and anomalies of the circle of Willis in fetal brain. Microsurgical study and functional remarks. Surg Neurol 1985; 24:563-70.

CONFLICT OF INTEREST
Authors declare no conflict of interest.
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None declared.