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Anatomical aberration of posterior part of circle of Willis with special reference to third nerve palsy

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ABSTRACT

Background: Due to the close anatomical proximity, aneurysm of posterior communicating artery may cause oculomotor nerve palsy, which is a common manifestation and it may present with pupil involving extra ocular muscle paresis. The onset may be acute due to sudden aneurysm dissection along with subarachnoid hemorrhage. This may warrant urgent lifesaving surgical intervention along with the options of interventional radiological procedures.

Normally, the occipital lobe is supplied by the posterior cerebral artery (PCA). When the pre-communicating portion of the PCA is larger than the posterior communicating artery (PCA), the occipital lobe is primarily supplied by the vertebrobasilar artery. Sometimes the pre-communicating portion of the PCA may be smaller than the PCoA in which the occipital lobe is primarily supplied by the internal carotid artery.

Aim & Objective: Understanding the posterior circulation anomalies of Circle of Willis.

Materials and Methods: This study involved 50 fetal brains belonging to perinatal age group and 30 adult brains of human cadavers by dissection.

Results: The PCoA was missing in 14% of the samples. The difference between the outer diameters of the P1 portion of the PCA and the PCoA was very significant.

Conclusion: So, a detailed anatomical understanding of Circle of Willis and manifestation of the third nerve palsy is sometimes equivocal for a clinician for better management and patient outcome.

1. Introduction

The circulatory system of the central nervous system holds a particular fascination due to the high energy needs of neural tissue.[1,2]

A substantial portion of the brain receives its blood supply from the branches of both the vertebral arteries and the internal carotid arteries. Notably, there is a significant connection known as the "circulus arteriosus" between the vertebral and carotid arterial networks.[3] This interconnected system was originally identified by Thomas Willis, an English anatomist and physician from the 17th century, who observed a circular arrangement of arteries at the base of the brain within the interpeduncular fossa. Consequently, this arterial circle has come to be known as the "Circle of Willis".[4,5]

The arterial circle, often referred to as the Circle of Willis, takes on a polygonal shape, specifically a nonagon with nine sides.[6,7] This intricate structure resides at the base of the brain, enveloping important components such as the optic chiasma and structures within the interpeduncular fossa. Positioned within the suprasellar system beneath the hypothalamus and the third ventricle, it encompasses the ventral surface of the diencephalon, running alongside the optic nerves and optic tracts. Its location is medial to both

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the tentorium and the temporal lobe, situated anteriorly to the midbrain.[8]

Comprising the Circle of Willis are the internal carotid artery, which connects through the anterior cerebral arteries on both the right and left sides, facilitated by an anterior communicating artery that links these right and left anterior cerebral arteries. Additionally, the carotid system establishes connections with the posterior cerebral arteries of the vertebral system through two posterior communicating arteries, one on each side.[9]

The conventional depiction of the Circle of Willis, in its classical form, presents a symmetrical and normally sized polygonal structure. However, there exist numerous variations in the typical arrangement of this arterial circle.[10]

The primary function of the arterial circle lies in its ability to serve as an alternate pathway in situations where one of the major arteries leading to it becomes blocked.[11] When an arterial blockage occurs due to an embolus or thrombus, it typically results in the deprivation of blood supply to a specific region, leading to tissue infarction.[12]

Normally, the occipital lobe is supplied by the posterior cerebral artery (PCA). When the pre-communicating portion of the PCA is larger than the posterior communicating artery (PCA), the occipital lobe is primarily supplied by the vertebrobasilar artery.[13,14] Sometimes the pre-communicating portion of the PCA may be smaller than the PCoA in which the occipital lobe is primarily supplied by the internal carotid artery.[15]

1.1. Early diagnosis and treatment

Anomalies in the posterior circle of Willis, such as hypoplastic or absent segments, can lead to reduced blood supply to critical brain structures. This reduced blood flow can affect the oculomotor nerve, resulting in third nerve palsy.[16] Recognizing this relationship is crucial because early diagnosis of posterior circle of Willis anomalies can help initiate timely treatment and interventions to improve blood flow and prevent further nerve damage.

1.2. Neurological implications

Third nerve palsy often presents with a range of neurological symptoms, including ptosis (drooping eyelid), diplopia (double vision), and impaired eye movements. Identifying the underlying vascular anomalies in the posterior circle of Willis can provide valuable insights into the cause of these neurological symptoms and guide appropriate management strategies.[17]

1.3. Risk of ocular complications

Third nerve palsy can lead to various ocular complications, such as strabismus (misalignment of the eyes), amblyopia (lazy eye), and visual impairment. Understanding the association with posterior circle of Willis anomalies is important for clinicians to predict and mitigate the risk of such complications, thereby preserving or improving visual function in affected patients.[18]

1.4. Wider clinical implications

The relationship between third nerve palsy and posterior circle of Willis anomalies may have broader clinical implications. It could serve as an indicator of potential vascular issues in the brain, which may extend beyond oculomotor nerve dysfunction.[19] Clinicians should consider the possibility of associated vascular abnormalities and conduct comprehensive neurological assessments in patients presenting with third nerve palsy.

1.5. Improved patiënt management

Awareness of the link between third nerve palsy and posterior circle of Willis anomalies can enhance patient management. Clinicians can tailor treatment plans to address both the neurological deficits caused by the nerve palsy and the vascular issues contributing to the condition. Multidisciplinary approaches, involving neurologists, ophthalmologists, and vascular specialists, may be necessary to provide comprehensive care.

1.6. Research and advances in vascular neurology

Documenting and publishing cases where third nerve palsy is associated with posterior circle of Willis anomalies can contribute to the growing body of knowledge in vascular neurology. These cases may serve as valuable data points for further research, potentially leading to advancements in understanding the etiology and treatment of such conditions.

Research has shown ethnic variations in the occurrence of intracranial aneurysms.[20]

Due to the close anatomical proximity, aneurysm of posterior communicating artery may cause oculomotor nerve palsy, which is a common manifestation and it may present with pupil involving extra ocular muscle paresis. The onset may be acute due to sudden aneurysm dissection along with subarachnoid haemorrhage.[21,22] This may warrant urgent lifesaving surgical intervention along with the options of interventional radiological procedures.

So, a detailed anatomical understanding of Circle of Willis and manifestation of the third nerve palsy is sometimes equivocal for a clinician for better management and patient outcome. A minor clinical clue like pupil involving third nerve palsy sometimes indicate the underlying impending rupture of posterior communicating artery which urgently needs interventions like coiling and other thrombolytic procedures.[23,24] Aberrant posterior communicating artery is not common.

Our study also aimed at the novelty in understanding the posterior circulation anomalies among the paucity of the current literature in this region with a view of geographical

and other ethnical variations.

As the neurological signs depends on the site of the lesion, an understanding of the distribution of the arteries is very essential. A detailed knowledge on various configuration of the circle of Willis is also important for surgical interventions.

Given that neurological symptoms are contingent upon the location of the lesion, a comprehensive understanding of arterial distribution is crucial. Moreover, a thorough grasp of the diverse configurations of the Circle of Willis holds significant importance for surgical interventions.

Despite the remarkable progress in anatomical studies facilitated by technologies like computerized tomographical angiography and magnetic angiography, traditional dissection-based studies maintain their significance.[25,26]

Numerous scholars have dedicated their efforts to studying the Circle of Willis, although such research in the northeastern region of India has been relatively scarce. With this in mind, we decided to embark on this study.

2. Materials and Methods

2.1. Place of study

The present study, "Anatomical aberration of posterior part of circle of Willis with special reference to third nerve palsy" was done in the Department of Anatomy, Assam Medical College & Hospital, Dibrugarh.

2.2. Duration of study

One year.

2.3. Selection of specimens

The present study was conducted on 50 fetal brains belonging to perinatal age group and 30 adult female and male brain samples from human cadavers.(Table 1)

The perinatal period extends from the end of 22^{nd} weeks of gestation to 7 days after birth.[27]

Table 1: Distribution of specimens

Group	Number of brains	Percentage
Fetus	50	62.5%
Adults	30	37.5%
Total	80	100%

Specimens was collected from the dead fetuses of perinatal age group received from the Department of Obstetrics and Gynecology, the adult cadavers provided to the Department of Anatomy for dissection, and autopsies performed by the Department of Forensic Science & Medicine of the Assam Medical College & Hospital.(Table 2)

Table 2: Source of collection of specimens

Source	Number of brains
Obs & Gynae Deptt.	50
Forensic Science Deptt.	20
Anatomy Dissection Room	10
Total	80

2.4. Instruments and materials used

1. Scalpel.
2. Forceps- toothed, plane and pointed.
3. Scissors.
4. Hand gloves.
5. Metallic tray.
6. Dissecting microscope.
7. Ocular micrometer.
8. Stage micrometer.
9. Vernier calipers.
10. Buckets with preservative (10% formalin).
11. Gloss enamel paint (Red).
12. Painting brush (no. 0)
13. Chisel.
14. Hammer.
15. Bone cutting saw.
16. Thread.
17. Card board labels.

In the present work all, the specimens are collected from human cadavers. The particulars as per proforma are taken from Cadaver Report Form when the cadavers are brought to Department of Anatomy, and Autopsy Record of Department of Forensic Sciences, Assam Medical College & Hospital.

The fetal cadavers are injected with 10% formalin through the anterior fontanelle and kept preserved for 72 hours.

2.5. Dissection of brain

A circular incision is made from supraorbital margin and a point above the auricle to external occipital protuberance to remove the calvaria, then the brain is removed by following steps:

1. A longitudinal incision is made about 1cm from either side of superior sagittal sinus. Then the falx cerebri is removed from its attachments to crista galli. Following this, the falx cerebri is pulled out of the longitudinal fissure of brain.
2. The head is hyperextended for better exposure of cranial nerves in the base of the brain. Then the brain is released from the base of brain by cutting the nerves serially.
3. The tentorium cerebelli is cut on both sides to release the cerebellum.

4. The spinal cord is cut above the cervical vertebra.
5. Now, the brain is finally removed from the cranial cavity, put in a tray and the morphology of the circle of Willis is studied. (Cunningham's Manual of Practical Anatomy 15th edition; Gray's Dissection Guide for Human Anatomy).

The damaged ones were discarded and only intact specimens were taken into account. 10% formalin was used to preserve the specimens that were obtained. At the time of preservation, the dissected specimens were cleaned and numbered.

Each brain's Circle of Willis was thoroughly dissected, and if necessary, a piece of the brain's base was cut and removed to clearly display the artery circle.

2.6. Study of circulus arteriosus

The interpeduncular fossa's meninges were carefully removed, and the circle of Willis was then seen in its natural setting. Each specimen's Willis circle was meticulously examined, and the results were reported and calculated in relation to:

Variations are seen depending on whether or not each circular segment differs from the others in terms of size, shape, form of anastomosis, etc.

Only after all necessary measurements of the arterial circle's constituent arteries had been taken was the arterial circle colored with premium gloss enamel red color paint using a "0" number brush. Photographs were taken on Canon 7.1 mega pixels camera with optical zoom.

3. Results and Observations

Table 3: Incomplete circulus arteriosus - in adults

Incomplete circle	No. of cases	Percentage
Anterior part	1	3.33%
Posterior part	4	13.33%

3.1. Circulus arteriosus completion

1. In adults, the anterior part of the circle was complete and normal in 25 out of 30 cases (83.33%), whereas the posterior part was complete in 14 out of 30 cases (46.67%).(Table 3)
2. In fetal specimens, the anterior part was complete and normal in 46 out of 50 cases (92%), and the posterior part was complete in 43 out of 50 cases (86%).
3. The posterior part of the circle was more commonly anomalous in adults (53.33%) compared to fetuses (14%).

3.2. Circulus arteriosus: Posterior part

3.2.1. Pre-communicating segment (P1) of the posterior cerebral artery

Table 4: Distributionof P1 segment of PCA

Brains	Present	Absent
Fetus	86.0%	14.0%
Adult	86.67%	13.33%

3.2.2. P1 segment of the posterior cerebral artery

1. In both fetal and adult brains, the P1 segment was present in the majority of cases (86% for fetuses and 86.67% for adults).(Table 4)
2. In fetal brains, it was absent in 14% of cases, with 6% on the left, 4% on the right, and 4% bilaterally. In adults, it was absent in 13.33% of cases, with 10% on the right and 3.33% on the left.

3.2.3. Posterior communicating artery (PCoA segment)

Table 5: PCoA segment in fetal brains

PCoA portion	No. of samples	Percentage
Present	43	86%
Absent	7	14%
Both	2	4%
$PCoA_R$	2	4%
$PCoA_L$	3	6%

Table 6: PCoA portion in adult brains

PCoA portion		No. of specimens	Percentage
Present		26	86.67%
Absent		4	13.33%
Both		-	-
$PCoA_R$		3	10%
$PCoA_L$		1	3.33%
	Both	5	16.66%
Hypoplasia	$PCoA_R$	7	23.33%
	$PCoA_L$	2	6.66%

3.2.4. Posterior communicating artery (PCoA) segment

1. In fetal brains, the PCoA segment was present in 86% of cases and absent in 14% of cases.(Table 5)
2. Among those with absent PCoA, 4% and 6% had anomalies in the form of
3. PCoA-R or PCoA-L respectively, with 4% showing both PCoA-R and PCoA-L anomalies.(Figure 1)
4. In adult brains, (Table 6) the PCoA segment was present in 86.67% of cases and absent in 13.33%. Hypoplasia of the PCoA was observed in some adult

cases (16.66% for both sides, 23.33% on the right, and 6.66% on the left).(Figure 2)

3.2.5. Part of the posterior cerebral artery before the communication (P1 segment) vs posterior communicating artery (PCoA)

Table 7: The configuration in fetal brains

		No. of brains	Percentage
Fetal Type	Right side	5	10%
	Left side	5	10%
	Bilateral	13	26%
	Total	23	46%
Adult type	Right side	7	14%
	Left side	3	6%
	Bilateral	21	42%
	Total	31	62%

Table 8: The configuration in adult brains

		No. of brains	Percentage
Fetal Type	Right side	3	10%
	Left side	-	-
	Bilateral	1	3.33%
	Total	4	13.33%
Adult type	Right side	1	3.33%
	Left side	7	23.33%
	Bilateral	21	70%
	Total	29	96.66%

3.2.6. Comparison of P1 Segment and PCoA

1. In fetal brains, (Table 7) the external diameter of P1 segment (P1R and P1L) was compared to PCoA (PCoAR and PCoAL). On the right side, P1R was more prominent than PCoAR in 56% of cases, while on the left side, P1L was more prominent than PCoAL in 54% of cases.
2. In adult brains, (Table 8) P1R was more prominent than PCoAR in 66.66% of cases on the right side and 86.66% of cases on the left side.
3. A statistical analysis indicated that the external diameter of PCoA-R and P1-R significantly differed (p=0.0002, t=3.942) in adult brains but not in fetal brains (p=0.3472, t=0.9446).

4. Conclusion

Comparing all of the arterial circle's segments, the posterior communicating artery was the most abnormal. The front half of the circle was less abnormal than the posterior part.

Overall, the observations suggest differences in the circulus arteriosus, P1 segment, and PCoA between fetal and adult brains. Fetal brains appear to have a more complete circulus arteriosus, with fewer cases of anomalies and hypoplasia. In adults, anomalies and hypoplasia are more common, and there are significant differences in the diameters of the P1 segment and PCoA. These findings may have implications for our understanding of brain development and vascular anatomy.

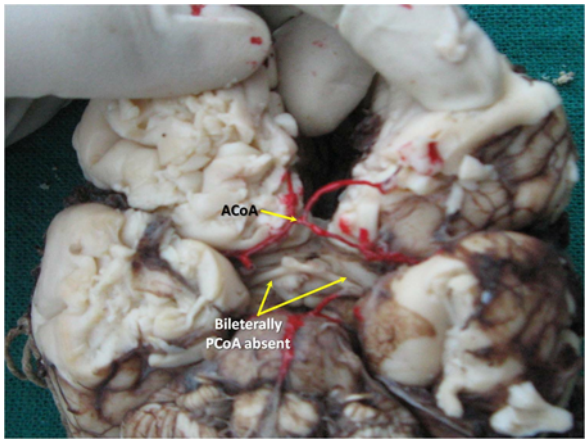

Figure 1: Bilaterally PCoA absent

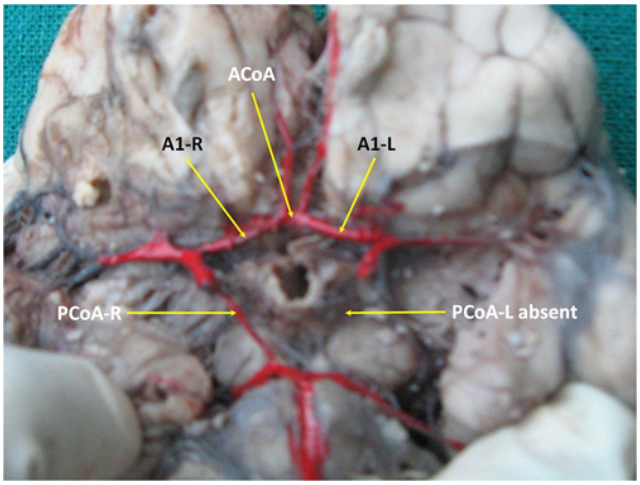

Figure 2: Unilaterally PCoA absent

Also, understanding the clinical importance of third nerve palsy in relation to anomalies in the posterior circle of Willis is essential for early diagnosis, effective management, and the prevention of associated complications.

Due to the close anatomical proximity, aneurysm of posterior communicating artery may cause oculomotor nerve palsy, which is a common manifestation and it may present with pupil involving extra ocular muscle paresis. The onset may be acute due to sudden aneurysm dissection along with subarachnoid haemorrhage. This may warrant urgent lifesaving surgical intervention along with the options of interventional radiological procedures.

So, a detailed anatomical understanding of Circle of Willis and manifestation of the third nerve palsy is sometimes equivocal for a clinician for better management and patient outcome.

5. Source of Funding

None.

6. Conflict of Interest

None.

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