ORIGINAL RESEARCH PAPER
Volume - 10 | Issue - 10 | October - 2021 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

CLINICAL AND EMBRYOLOGICAL STUDY OF A CASE OF BILATERAL ABSENT POSTERIOR COMMUNICATING ARTERIES

Anatomy

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ABSTRACT

Background: The circle of Willis represents a vascular network located at the base of the skull in the interpeduncular fossa. In the posterior part, the basilar artery separates into the right and left posterior cerebral arteries, and their connection to the ipsilateral internal carotid artery is provided via a posterior communicating artery. The posterior communicating artery, which represents a significant element of the circle of Willis, functions as a collateral channel for maintaining the blood flow.

Objectives:

1. To discuss a case of bilateral absent posterior communicating arteries
2. To review various theories regarding its embryological basis,

Methodology & Case presentation: A sixty seven year old male patient presented to the casualty of a tertiary care hospital with a history of giddiness and weakness in left upper and lower extremities. Patient is a known diabetic under treatment since 20 years. Neurological examination revealed left hemiparesis.

Conclusion: PCoAs absence is a congenital variant of Willis circle that occurs in 16.6% of the general population, and it is known that it becomes symptomatic when associated with internal carotid artery stenosis. However, some evidences suggest that PCoA hypoplasia per se predisposes to thalamic lacunar stroke because of the critical role of PCoA in the collateral supply of proximal PCA territory. Thus, it is suggested that the occurrence of the anatomic variant might have led to a hemodynamic infarction due to a poor regional collateral flow.

KEYWORDS

Bilateral absent posterior communicating arteries, Circle of Willis

INTRODUCTION:

The posterior communicating artery (PCoA) is a smaller branch of the internal carotid artery (ICA). It runs backward to anastomoses with the posterior cerebral artery (PCA) to contribute in the formation of circle of Willis (CW). It forms the main communicating channel between the internal carotid arterial system and the vertebro-basilar arterial system.[1,2] The size of PCoA is usually very small but it varies frequently to form fetal PCoA.[2]

Case discussion:

A sixty seven year old male patient presented with a history of mild headache, giddiness & weakness in left upper & lower extremities. Patient is a known diabetic under treatment since 20 years. General examination showed that the vitals were stable. Neurological examination revealed left hemiparesis, power on the left side was 3/5, left plantar reflex was exaggerated, the power on the right side was 5/5. Contrast MRI brain revealed right thalamo-capsular infarction and bilateral absent posterior communicating arteries. The patient was managed as a case of ischemic stroke.

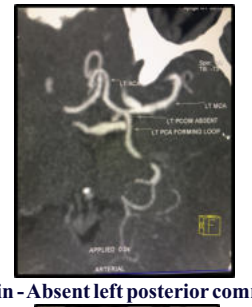

Image 1: MRI Brain - Absent left posterior communicating artery

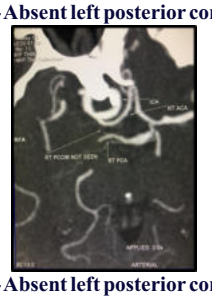

Image 2: MRI Brain - Absent left posterior communicating artery

Embryology:

The blood vessels of the brain develop from a primitive plexus of vascular channels. Certain vascular channels develop and persist and certain channels disappear in accordance with contemporary needs and relations of the areas to be supplied.

The CW starts developing around 4th week (24th day) of IUL and by 7th week (CRL 40mm), fully formed, closed CW can be visualised[3].The standard textbook of Embryology[4] describe that initially the Internal carotid artery (ICA) divides into *Anterior, Middle, & Posterior cerebral arteries*. The latter extends backwards and divides into two branches- the medial branch joins the *Basilar* artery while the lateral ex- tends to supply the occipital & temporal lobes. The growth of occipital lobes and brain stem provides the initial directive for the development of vertebro-basilar system. In due course of time blood from the basilar artery starts circulating in the medial branch & from there in the lateral branch sup- plying the temporal & occipital lobes. On account of hemodynamic factors, the proximal part of the embryonic PCA up to its division becomes smaller in calibre & is now called the posterior communicating artery (PCoA).The medial & lateral branches of the original PCA are now named as proximal and distal segments of PCA arising from the basilar artery. The anterior communicating artery (ACoA) appears in the human embryo at (CRL 18 mm) as a reticulated anastomosis between the two ACAs. At 24mm CR length, this network fuses to form a single ACoA[3].

Functional demands of the growing brain tissue govern the development of cerebral vasculature. Soon with the regression of some earlier vessels and appearance of new channels, a fully developed CW becomes functional at the end of 6-7weeks. As the cerebral vascular tree reaches its typical configuration, the multiple events that occur during the embryological stage might lead to a diverse spectrum of vascular anatomical variants[5]. Duplications and fenestrations of an artery can occur due to an incomplete fusion of arteries[6].

DISCUSSION:

Anatomical variations of the posterior circulation are frequent but commonly asymptomatic. Familiarity with these variants is important as not to mistake them as pathological findings. Although typically discovered incidentally, they are infrequently related to intracranial vascular pathology.[7]

Asymmetric vascular accidents occur in over two-thirds of individuals [8,9] and an incomplete circle of Willis (COW) may be seen in over half

[10]. A persistent trigeminal artery is the most common carotid–vertebrobasilar anastomosis [11]. Other intracranial anomalies include fenestration of the vertebrobasilar junction (0.3–0.6%) which may predispose to PC aneurysm formation[12], persistent hypoglossal artery, the artery of Percheron, fetal origin of one or both PCAs (fPCA) and absence of one or both posterior communicating arteries (PCoAs)

In aplasia of PCoA or P1, blood supply to the occipital pole retains from a single source. In such cases, collateral circulation lacks. Blood supply may get hampered due to hemodynamic factors affecting vessel wall to change in the development of aneurysm or occlusion. Collateral circulation through Circle of Willis becomes important in occlusive vascular diseases of the brain. Compensatory circulation will be more effective in the presence of a normal and complete Circle of Willis. In addition, it has been reported that the incomplete circle of Willis predisposes about one-sixth of individuals to cerebral ischemia during the transient closure of carotid artery, but the risk is more than three times in case of contralateral internal carotid artery occlusion. [13].

However, in cases such as the one reported here, due to the absence of communicating arteries, the alternative routes may not be available. From the study of literature it can be seen that PCoA aplasia/ hypoplasia predisposes to small vessel infarcts of the thalamus, perhaps via a different mechanism than found in typical lacunar infarctions.

CONCLUSION:

PCoAs absence is a congenital variant of Willis circle and it is known that it becomes symptomatic when associated with internal carotid artery stenosis. However, some evidences suggest that PCoA hypoplasia per se predisposes to thalamic lacunar stroke because of the critical role of PCoA in the collateral supply of proximal PCA territory. Thus, it is suggested that the occurrence of the anatomic variant in this case might have led to a hemodynamic infarction due to a poor regional collateral flow. This is one of the rare anomalies and knowledge regarding this anomaly is important to clinicians.

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