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RESEARCH

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Anatomical variations in posterior part of the circle of Willis and their associations with brain infarct in different vascular territories

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Abstract

Background: The relationship between anatomical variations in circle of Willis and brain infarction is controversial. The purpose of this study was to evaluate the relationship between anatomical variations in posterior portion of the circle of Willis assessed by MR angiography (MRA) and ischemic infarction in different brain territories.

Methods: This cross-sectional study was conducted on consecutive patients who underwent brain MRI and MRA for suspected cerebrovascular accident. The frequency of anatomical variations including persistent fetal origin of posterior cerebral artery (fPCA) and hypoplastic/aplastic posterior communicating artery (PCoA) and their association with infarction in different intracranial vascular territories was assessed.

Results: In total, 298 patients (155 male/143 female with mean age ± SD of 57 ± 15) were enrolled in the study and categorized into two groups with infarction ($n = 142$) and without infarction ($n = 156$). Sixty-three patients (21/1%) had fPCA and 231 (77.5%) had PCoA hypoplasia/aplasia. No significant correlation was identified between fPCA or PCoA hypoplasia/aplasia and presence of infarction. However, regarding the territories involved by infarction, the frequency of thalamus infarction was higher in subgroup with PCoA hypoplasia/aplasia, 17/101 (16.8%) compared to 1/41(2.4%) in the subgroup without ipsilateral PCoA hypoplasia/aplasia ($p = 0.024$). In two subgroups with and without ipsilateral fPCA variation, frequency of infarction in brain territories was not different significantly.

Conclusions: In patients with brain infarction, aplastic/hypoplastic ipsilateral PCoA is associated with higher incidence of thalamic territory infarction.

Keywords: Circle of Willis, MR angiogram, Posterior cerebral artery, Posterior communicating artery, Brain infarction

Background

Stroke is a major cause of disability and the second leading cause of death worldwide [1]. Seventy-five percent of all stroke deaths and 81% of the total disability adjusted life years lost due to stroke occur in developing countries [2, 3]. Based on an Iranian systematic review in 2010, the annual stroke incidence among various ages ranged from 23 to 103 per 100,000 people [4]. Stroke risk factors include age, sex, race, hypertension, diabetes, hyperlipidemia, diet, smoking, and alcohol [5].

Arteriogenesis is a complex embryologic process and can lead to numerous anatomical variations [6, 7]. In case of a major cerebral arterial occlusion, collateral vessels play an important role in maintaining essential blood flow. Circle of Willis is the most important collateral system in the brain with multiple potential anatomical variations [8–10]. Some of the most common variations in the circle of Willis include hypoplasia or aplasia of one or both posterior communicating arteries (PCoA) (34 to 68%), hypoplasia or aplasia of the A1 segment of anterior cerebral artery (ACA) (4 to 10%), absence or

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fenestration of anterior communicating artery (ACoA) (12 to 21%), persistent fetal origin of posterior cerebral artery (fPCA) (4% to 26%), and infundibular dilatation or widening of PCoA (7% to 15%) [11–17].

Although there are numerous studies on anatomical variations of the circle of Willis, presence of any association between anatomical variations of circle of Willis and incidence of ischemic stroke is still unclear [18–24]. Previous studies have suggested that PCoA hypoplasia is associated with higher incidence of ischemic stroke, even in the absence of ICA occlusion and the most common ischemic event in patients with PCoA hypoplasia was ipsilateral thalamic lacunar infarct with or without occipital lobe involvement [19]. In case of fPCA, Shahan et al. reported that an enlarged PCoA (persistent fetal-type circulation) may improve collateralization between the anterior (internal carotid) and posterior (vertebrobasilar) circulations and may decrease the risk of stroke [22]. On the other hand, an incomplete anterior circle of Willis and a one-sided or two-sided incomplete posterior circle of Willis are associated with increased incidence of anterior circulation stroke [23]. A recent meta-analysis showed that patients with any anatomical variation in circle of Willis were 1.38 times more likely to develop ischemic stroke compared to those with intact complete circle of Willis and concluded that there was a positive association trend between circle of Willis variations and ischemic stroke [24].

The aim of our study was to evaluate the relationship between anatomical variations in the posterior aspect of circle of Willis as identified on MRA, and occurrence of ischemic infarct in different vascular territories.

Methods

Patients

This retrospective cross-sectional study was approved by our institutional review board and all methods were performed in compliance with the Helsinki Declaration. The need for informed consent was waived by Shahid Beheshti Medical University ethics committee (IR.SBMU.MSP.REC.1398.420). All consecutive patients who underwent brain MRI and MRA to evaluate for suspected cerebrovascular accident at Loghman Hakim hospital between March 20, 2017, and April 10, 2018, were included in this study. Those with a history of head trauma, craniotomy or craniectomy, vasculitis, pregnancy, vascular malformation (including aneurysm or arteriovenous malformation), hemorrhagic infarction, significant stenosis or occlusion of internal carotid artery, significant stenosis or occlusion of basilar artery or its major branches were excluded from the study. Demographic data, patient's presenting symptoms, and past medical history including hypertension, diabetes, hyperlipidemia, heart disease, and smoking were reviewed from the patients' electronic medical records.

Imaging protocol

Scans were performed using a 1.5-T Vantage Elan magnet (Canon Medical Systems, Otawara, Tochigi, Japan). The examination was performed without intravenous contrast and included echo-planar T1-weighted images (TR: 591 ms, TE:15 ms, Spatial Resolution: 6.2 mm, FoV: 230 mm*230 mm), T2-weighted images (TR: 4048 ms, TE:90 ms, Spatial Resolution:6.2 mm, FoV: 230 mm*230 mm), FLAIR, and diffusion-weighted images (DWI).

For evaluation of circle of Willis, images were obtained in 3 slabs each containing 30 slides with 3-dimensional time-of-flight MRA technique (TR: 22 ms, TE:7 ms, number of acquisitions: 2; flip angle: 17°; 1 mm slice thickness with a 0.5 mm overlap; matrix size: 256*160, FOV: 200*200 mm). MRA images were reconstructed in transverse oblique planes using a maximum intensity projection algorithm.

All images were reviewed by a national board-certified radiologist with 5 years of experience in neuroimaging (M.H.) and a fourth-year radiology resident (E.S.) using a consensus approach.

Definitions

The following variations in posterior aspect of the circle of Willis on MRA were recorded: Partial or complete fetal origin of posterior cerebral artery (pfPCA and cfPCA) and aplasia or hypoplasia of PCoA.

CfPCA was defined as the P1 segment being absent and PCA originating completely from the internal carotid artery (ICA). PfPCA was defined as the existing P1 segment with a caliber equal to or smaller than PCoA [12].

Due to the limited resolution of MRA, it was difficult to differentiate PCoA hypoplasia (< 1 mm in diameter) from aplasia (absence of PCoA). Therefore, we considered hypoplasia/aplasia together, defined as PCoA diameter of < 1 mm or non-visualization of PCoA [20].

In cases of infarction, the affected side, vascular territory, and the age of infarct (acute/subacute versus chronic) were recorded based on the MRI findings and clinical history. The areas of restricted diffusion (bright signal on DWI and corresponding low ADC values) were qualitatively determined based upon consensus by the reviewing radiologists and were considered acute/subacute infarct. The areas of encephalomalacia or gliosis without associated restricted diffusion and with volume loss were considered chronic infarct. In cases with simultaneous acute and chronic infarcts, the territory with acute infarct was considered.

The vascular distribution of infarct was divided into anterior, posterior, thalamus, and watershed area similar to previous studies [19].

In the presence of unilateral anatomical variation, if it was on the same side as the infarct, it was considered positive, so we described it as "ipsilateral anatomic variation." When the patient had bilateral anatomical variation, the side with infarction was counted and reclassified as "ipsilateral."

Statistical analysis

The results were presented as mean ± standard deviation (SD) for quantitative variables and were summarized by absolute frequencies and percentages for categorical variables. Categorical variables were compared using Chi-square test or Fisher's exact test when more than 20% of cells with expected count of less than 5 were observed. Quantitative variables were compared using the student T test and Mann Whitney U test, for parametric and nonparametric variables, respectively. For the statistical analysis, the statistical software SPSS version 21.0 for windows (SPSS Inc., Chicago, IL) was used. p values of 0.05 or less were considered statistically significant.

Results

A total of 298 cases were enrolled in the study and categorized into two groups: with infarction ($n=142$) and without infarction ($n=156$). The group with infarction had higher mean age, higher rate of hypertension, diabetes mellitus, and ischemic heart disease, as detailed in Table 1.

Among the group with infarction, 46.5% had right-sided and 53.5% had left-sided infarcts. Acute/subacute infarcts were noted in 90.1% and chronic infarcts, as the only type of infarct, were present in 9.9% of patients.

Table 1 Distribution of cerebrovascular risk factors in patients with and without infarction

Parameter	With infarction ($n=142$)	Without infarction ($n=156$)	p value
Male gender	80 (56.3)	75 (48.1)	0.154
Mean age, year	63.36 ± 12.82	52.61 ± 16.30	< 0.001
Hypertension	103 (72.5)	76 (48.7)	< 0.001
Diabetes mellitus	57 (40.1)	35 (22.4)	0.001
Hyperlipidemia	14 (9.9)	13 (8.3)	0.647
Smoking	23 (16.2)	13 (8.3)	0.038
Ischemic heart disease	41 (28.9)	25 (16.0)	0.008

Territorial distribution of ischemic infarct in patients with fPCA

Sixty-three patients (21.1%) had fPCA including 48 (16.1%) with cfPCA and 16 (5.3%) with pfPCA. One patient had cfPCA on one side and pfPCA on the other side. CfPCA and pfPCA were right-sided in 27 (56.2%) and 9 (56.2%), left-sided in 16 (33.3%) and 4 (31.2%) and bilateral in 4 (8.3%) and 2 (12.5%) patients, respectively. Seven cases with bilateral fPCA were excluded. In remaining cases with unilateral fPCA, ipsilateral infarct was observed in 11 patients. There was no significant association between fPCA and infarcts.

Among the patients with infarction, subgroup analysis of infarcted territory between those with and without fPCA revealed no significant difference as detailed in Table 2. When comparing the patients with versus without ipsilateral fPCA, the anterior circulation infarcts [ACA and middle cerebral artery (MCA)] were seen in 6/11 (54.4%) versus 77/127 (60.6%) patients; posterior circulation territory infarcts [basilar artery, superior cerebellar artery (SCA), posterior cerebral artery (PCA), and posterior inferior cerebellar artery (PICA)] were seen in 3/11 (27.2%) versus 23/127 (18.1%) patients; thalamic infarcts were seen in 1/11 (9.1%) versus 17/127 (13.4%) patients, and watershed territory infarcts were seen in 1/11 (9.1%) versus 10/127 (7.9%) patients.

No significant relationship was found between fPCA and clinical risk factors of cerebrovascular disease (including male gender, diabetes, hypertension, hyperlipidemia, smoking, and ischemic heart disease) in patients with infarction.

Territorial distribution of ischemic infarction in patients with PCoA hypoplasia/aplasia

There were 231 cases with PCoA hypoplasia/aplasia (77.5%), including 35 (11.7%) on the right side, 38 (12.7%) on the left side and 158 (53.2%) on both sides. In cases with infarction and bilateral PCoA hypoplasia/aplasia, we recorded the vascular anatomical variation that was ipsilateral to the infarct and used it for comparison to those

Table 2 Involvement of different territories in groups with and without fPCA variation

Infarcted vascular territory	With ipsilateral fPCA ($n=11$)	Without ipsilateral fPCA ($n=127$)
Hemispheric stroke		
Anterior	6 (54.4)	77 (60.6)
Posterior	3 (27.2)	23 (18.1)
Thalamus	1 (19.1)	17 (13.4)
Watershed	1 (9.1)	10 (7.9)

without infarction. Overall, the frequency of ipsilateral PCoA hypoplasia/aplasia in patients with infarction was 101/142 (71.1%) compared to 116/156 (74.4%) in those without infarction and the difference was not statistically significant ($p=0.531$) (Fig. 1).

When comparing the patients with versus without ipsilateral PCoA hypoplasia/aplasia (Table 3), the anterior circulation territory infarcts (ACA and MCA) were seen in 57/101 (56.4%) versus 30/41 (73.2%) patients; posterior circulation territory infarcts (basilar artery, SCA, PCA, and PICA) were seen in 17/101 (16.8%) versus 9/41 (22.0%) patients; thalamic infarcts were seen in 17/101 (16.8%) versus 1/41 (2.4%) patients; and watershed territory infarcts were seen in 10/101 (9.9%) versus 1/41 (2.4%) patients. A statistically significant difference in frequency of infarcts in patients with versus without PCoA hypoplasia/aplasia was present only for thalamic infarcts ($p=0.024$) (Fig. 2). In the subgroup with ipsilateral PCoA hypoplasia/aplasia and simultaneous ACA involvement, ipsilateral A1 hypoplasia was present in 2 patients and ipsilateral A2 hypoplasia was seen in one patient.

To account for the potentially confounding clinical risk factors in patients with anatomical variants, the relationship between PCoA hypoplasia/aplasia with clinical risk factors was analyzed and no significant correlation was depicted.

Discussion

The relationship between the anatomical variations in circle of Willis and occurrence of infarcts in different vascular territories has been controversial. Many previous studies have proposed that such variations (anatomical or pathological) may be associated with a higher rate of brain infarction [19–21, 25]. In our study, we assessed the anatomical variations in the posterior aspect of circle of Willis based on MRI and MRA findings and investigated the relationship between these variations and infarcts in different vascular territories.

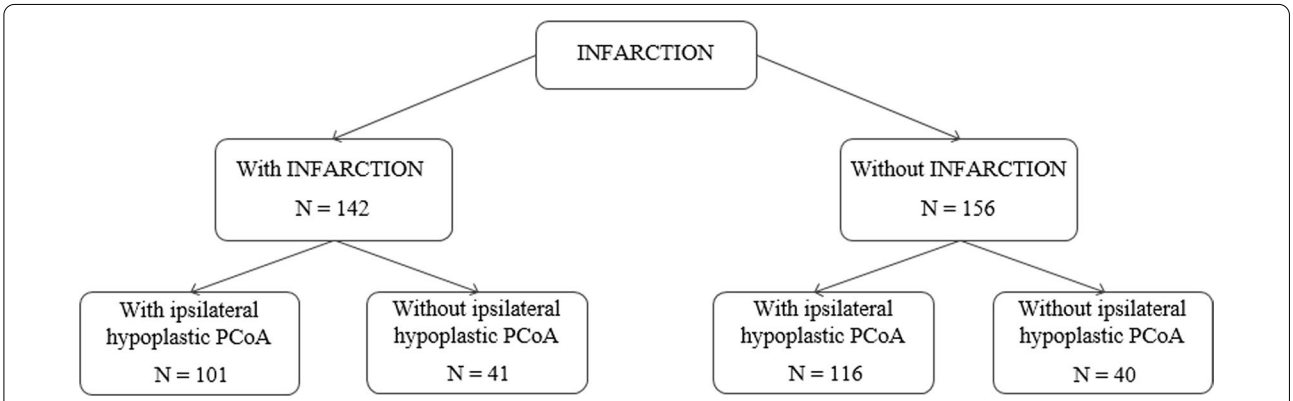

Fig. 1 Number of cases with ipsilateral PCoA hypoplasia/aplasia in groups with and without infarction

Table 3 Involvement of different territories in groups with and without ipsilateral hypoplastic PCoA variation

Infarcted vascular territory	With ipsilateral PCoA hypoplasia/aplasia ($n=101$)	Without ipsilateral PCoA hypoplasia/aplasia ($n=41$)
Hemispheric stroke		
Anterior	57 (56.4)	30 (73.2)
ACA	3 (2.9)	4 (9.8)
MCA	54 (53.5)	26 (63.4)
Posterior	17 (16.8)	9 (21.9)
PCA	6 (5.94)	2 (4.9)
SCA/Basilar/PICA	11 (10.9)	7 (17.0)
Thalamus*	17(16.8)	1 (2.4)
Watershed	10 (9.9)	1 (2.4)

Values in parenthesis are percentages

ACA, anterior cerebral artery; MCA, middle cerebral artery; PCA, posterior cerebral artery; SCA, superior cerebellar artery; PICA, posterior inferior cerebellar artery

* Significant ($p=0.024$)

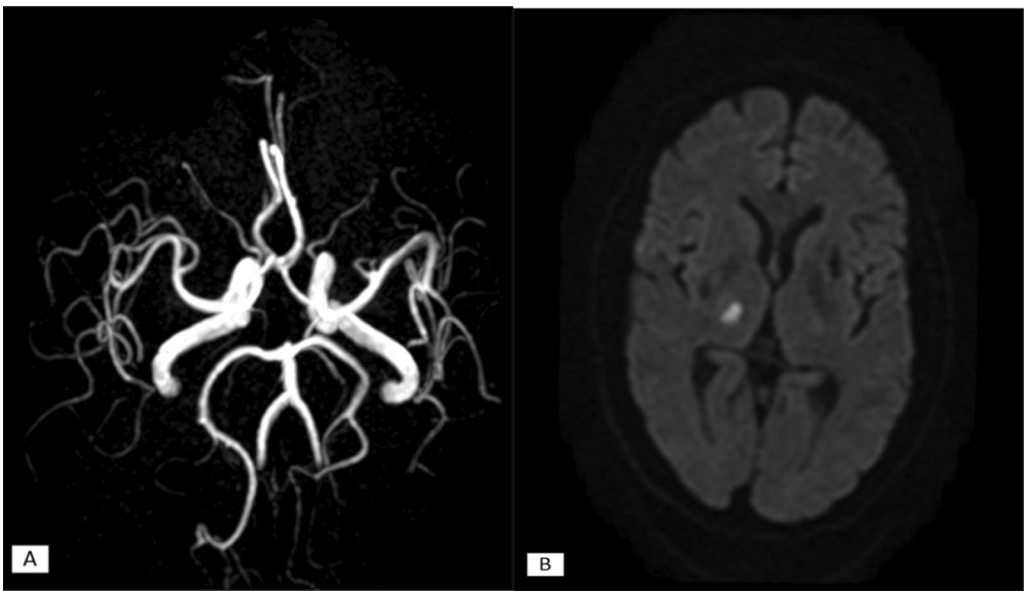

Fig. 2 Axial Maximal Intensity Projection (MIP) reconstructed MRA image (**A**) demonstrates right PCoA hypoplasia/aplasia. Corresponding diffusion-weighted image (**B**) depicts an acute right thalamic infarct

Our results showed that patients with infarcts expectedly had higher mean age and higher prevalence of hypertension, diabetes, smoking, and heart diseases.

FPCA and PCoA hypoplasia/aplasia were identified in 21.1% and 77.5% of our studied patients, respectively. The reported prevalence of fPCA and PCoA hypoplasia/aplasia in literature is variable. In a review published by Eftekhar et al. [13] the prevalence of unilateral fPCA was reported 7%-42%, that of bilateral fPCA 0.14%-11.3%, that of unilateral PCoA hypoplasia 13.5%-51.6%, and that of bilateral hypoplasia 0.7%-49% in autopsy samples. No definite ethnic difference was reported. Different size criteria for definition of hypoplasia (luminal diameter less than 0.5 mm versus 1 mm) might be partly responsible for the wide variability of reported numbers.

Our results did not reveal statistically significant association between cfPCA or pfPCA and infarcts in different vascular territories. As previously indicated, there are conflicting reports about the association between fPCA and increased risk of infarcts. In a study by de Monye et al. [16], risk of ischemic infarct or transient ischemic attack was not increased with fPCA which is consistent with our findings. On the other hand, Arjal et al. [20] noticed higher rates of ischemic infarct in partial fPCA compared to complete fPCA, despite the intuitive assumption that complete fetal PCA would impose higher risk of stroke. Shaban et al. [17] also showed that among patients with acute ischemic stroke, the frequency of pfPCA and cfPCA was not higher than in the general population; however, their conclusion needs to be confirmed with larger sample sizes.

We found no significant association between PCoA variation and increased risk of brain infarct; however, in the subgroup analysis, the rate of infarction in thalamic territory was significantly higher in patients with absent or hypoplastic ipsilateral PCoA compared to the group without PCoA hypoplasia/aplasia. Cheung et al. [19] have reported that PCoA hypoplasia is a contributor to the risk of ischemic stroke, particularly for strokes involving the arteries that penetrate the thalamus. Zelante et al. [26] have reported a case of Artery of Percheron infarct in a patient with absence of bilateral PCoA.

Thalamus has a complex arterial network. Some arterial branches such as thalamic, subthalamic, polar, and posterior/lateral choroidal all arise from proximal PCA. Thus, in patients with normal PCoA, obstruction in one of these branches cannot lead to infarcts due to the presence of supportive collateral; however, in those with PCoA hypoplasia/aplasia, occlusion of one of these branches may lead to tissue ischemia and death [27]. PCoA hypoplasia/aplasia through weakness in segmental collaterals may lead to thalamic infarcts. In patients presenting with major intracranial vascular occlusion, however presence of PCoA hypoplasia/aplasia is not associated with higher risk of ischemic infarct in ipsilateral hemisphere, because the hemodynamic effects of involvement of great vessels on brain circulation are more drastic than that of PCoA hypoplasia/aplasia (28).

Our study has inherent limitations. Limited resolution of our TOF MRA with 1.5 T MRI can potentially lead to non-visualization of PCoA when it is very narrow, however we put hypoplasia/aplasia together as vessel diameter of less than 1 mm or absence, to eliminate this error. In spite of these criteria, a potential thromboembolic occlusion/narrowing of the vessel can lead to misperception of such cases as PCoA hypoplasia/aplasia, however this is claimed to be of very low incidence. Other limitations include retrospective design, small sample size, and limited number of patients with simultaneous A1 and PCoA hypoplasia/aplasia limiting our ability to draw any conclusion about their combined effect.

Conclusions

Based on our results, absent/hypoplastic ipsilateral PCoA may be associated with higher frequency of thalamic infarcts.

Abbreviations

fPCA: Fetal origin of the posterior cerebral artery; cfPCA: Complete fetal origin of the posterior cerebral artery; pfPCA: Partial fetal origin of the posterior cerebral artery; PCoA: Posterior communicating artery; ACoA: Anterior communicating artery; ACA: Anterior cerebral artery; MCA: Middle cerebral artery; SCA: Superior cerebellar artery; PICA: Posterior inferior cerebellar artery; ICA: Internal carotid artery.

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Authors' contributions

MH contributed to conceptualization, methodology, data collection, reviewing, and editing; HBM contributed to data analysis and writing—reviewing and editing; ES contributed to data collection and writing—original draft preparation; AR contributed to reviewing and editing. All authors read and approved the final manuscript.

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Availability of data and materials

The data that support the findings of this study are not publicly available due to institutional restrictions but are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

This retrospective cross-sectional study was approved by our institutional review board and informed consent was waived.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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