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Assessment of Anatomical Variations and Cerebral Vessel Diameters in Ischemic Stroke Patients Using CT Angiography Examination

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ABSTRACT

Background: Willis Polygon (WP), cerebral circulation, and brain function are closely linked. Anatomical variations may contribute to cerebrovascular diseases, making their understanding vital for assessing stroke risk. Exploring the relationship between WP variations and vessel diameters holds clinical significance. The aim of this study was to investigate different anatomic variations and dimensions of WP in patients with ischemic stroke.

Methods: This observational, descriptive, and retrospective study evaluated CW anatomy in 132 ischemic stroke patients and 130 controls using CT angiography. WP arterial diameters were measured, and variations recorded.

Results: In the ischemic stroke patient group, anterior system variation was 48.9% and posterior system variation was 51%, with no significant difference compared to controls (p=0.5).

Diameters of right and left internal carotid arteries (ICA), A1 segment, and middle cerebral artery (MCA) were significantly lower in the study group. However, no significant differences were found in diameters of

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the basilar artery and the P1 segment of the right and left posterior cerebral arteries between groups.

Conclusion: In our study, no significant differences were found in WP variation and basilar artery and P1 segment of posterior cerebral arteries between ischemic stroke patients and the normal population.

However, arterial diameters (bilateral ICA, A1, MCA) were significantly lower in the ischemic stroke group compared to controls. In conclusion, arterial calibrations forming the anterior circulation of the Willis polygon were notably lower in patients with anterior circulation infarction, suggesting a significant role of decreased arterial diameters in ischemic stroke pathophysiology.

KEY WORDS

Willis Polygon, ischemic stroke, variations, arterial diameter, CT angiography, anterior system variation

Introduction:

Willis Polygon, situated at the base of the skull within the interpeduncular fossa, serves as a pivotal vascular network regulating cerebral circulation. It comprises various arteries and connections, with the anterior segment primarily constituted by the anterior cerebral artery (ACA), while the anterior communicating artery (AcomA) serves to connect the right and left ACAs. The posterior division involves the bifurcation of the basilar artery into the right and left posterior cerebral arteries (PCAs), each establishing connections with the bilateral internal carotid arteries (ICAs) via the posterior communicating arteries (PcomAs) [1].

Functionally, the WP plays a crucial role in safeguarding the brain against ischemia by ensuring a consistent and regulated supply of arterial blood [2]. Despite its relatively small mass, the brain commands a significant share of resources, necessitating a substantial portion of the cardiac output and oxygen supply [3]. Arteriogenesis, representing a multifaceted embryological process, can lead to various anatomical variations within the WP [4].

In situations of significant occlusion in cerebral arteries, collateral vessels, including the WP, assume a critical role in preserving essential blood circulation [5]. Ischemic stroke, comprising various subtypes, accounts for over eighty-seven percent of all stroke cases [6]. Structural variations within the WP, influenced by genetic and hemodynamic factors, often do not substantially affect brain function due to collateral circulations [7].

Nevertheless, WP variations may disrupt cerebral hemodynamic, potentially leading to diverse cerebrovascular diseases, such as cerebral aneurysms and ischemic stroke [8]. Individuals with efficient collateral circulations demonstrate a reduced risk of developing ischemic stroke compared to those with less effective collateral circulations [9]. Understanding intracranial artery variations is crucial, as they may increase susceptibility to aneurysms and impact cerebral blood flow [10].

WP variations hold significant clinical relevance, influencing the risk of ischemic stroke [11]. While most normal variations typically have minimal clinical impact, they are crucial considerations for surgical and interventional procedures [12]. Arterial diameters serve as vital predictors of vascular health, with larger diameters potentially associated with vascular events [13]. Clinicians routinely rely on arterial diameters for assessing vascular health, utilizing observations such as focal luminal narrowing in coronary arteries and lumen reductions at the carotid bifurcation and focal points within intracranial arteries to stratify stroke risk [14,15].

Therefore, the objective of our study was to assess the relationship between Circle of Willis variations and vessel diameters concerning ischemic stroke.

Materials and Methods:

Study Design and Patient Selection

In our study, a total of 428 patients aged 45 and over who presented to the emergency department or neurology outpatient clinic with symptoms of acute ischemic stroke (AIS) between February 01, 2019, and February 01, 2020, were included. After admission,

these patients underwent diffusion-weighted magnetic resonance imaging (DW-MRI) and cerebral CT angiography (CTA) at the radiology department. Patients who consented to participate in the study were further evaluated based on their radiological findings, clinical characteristics (including symptoms and the presence of concomitant diseases such as hypertension, diabetes, and coronary artery disease), and laboratory data (including total cholesterol, triglycerides, HDL, and LDL cholesterol). The data were retrospectively collected from our hospital's PACS system. Ethical approval for the study was obtained from the BAIBU Ethics Committee (ethics committee number:2020/114). Patients with posterior circulation infarctions (n=138), cerebral hemorrhage (n=16), and those with missing laboratory data (n=12) identified during the DW-MRI examination were excluded from the study. Consequently, a total of 132 patients with complete clinical, radiological, and laboratory data, who had anterior circulation infarctions, were included in the study. In order to detect a difference between the groups with an anticipated small to medium effect size of d=0.35, we needed at least 260 participants in total (alpha = 0.05 and power = 80%). Therefore, along with the 132 patients in the study group, we included 130 age and gender-matched control subjects. The control group comprised individuals who presented with suspected acute infarction but had normal findings on non-contrast brain CT and diffusion-weighted MRI examinations. The flowchart of our study is shown in Figure 1.

Radiological Examination: The patients underwent diffusion-weighted MRI examination using a 1.5 Tesla MRI machine (Symphony; Siemens, Erlangen, Germany) located in our radiology department. The diffusion-weighted MRI examination was performed to assess the presence or absence of infarction and, if present, to determine its localization. For the cerebral CT angiography (CTA) examination, a 64-slice CT angiography machine was employed (General Electric Revolution EVO, 64 slices).

The CTA examination was utilized to evaluate WP anterior system variations in both the patient and control groups. The classification system for WP variations, as used in previous similar studies, was adopted as a reference for categorizing these variations [16]. According to this classification system, the anterior system was categorized (figure 2) as follows: (a) complete anterior system, (b) two or more AcomA, (c) the origin of the corpus callosum median artery from AcomA, (d) short-segment fusion of the ACA, (e) division of the ACA into two branches distally after a common trunk, (f) the MCA originating from two separate vessels from the ICA, (g) hypoplasia or absence of AcomA, (h) unilateral hypoplasia or aplasia of the A1 segment with the other A2 segment originating from the existing A1 segment, (i) hypoplasia or absence of one ICA, and (j) hypoplasia/aplasia of AcomA with accompanying MCA arising as two separate branches.

In the study of anatomical variations of the posterior part of the WP, various configurations were observed

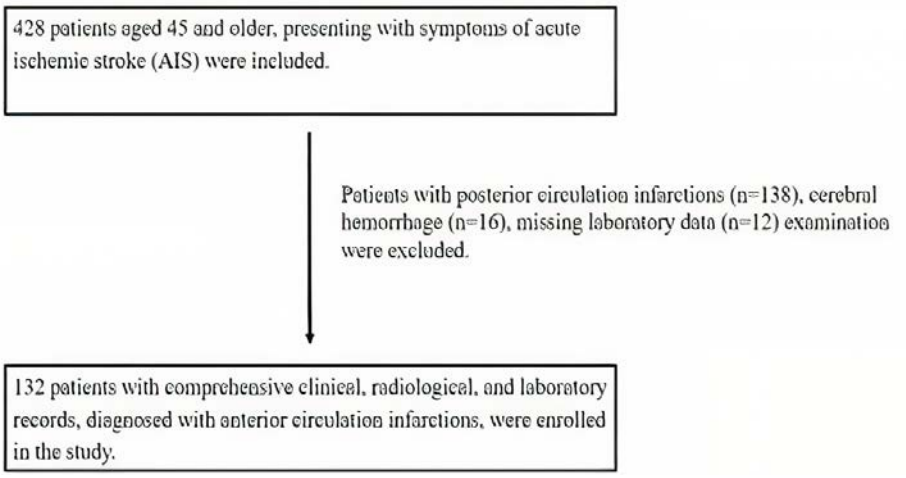

Figure 1: *The flowchart of our study is shown in Figure 1.*

(figure 3). (a) Bilateral PcomAs are present, (b) PCA originates predominantly from the ICA. This variant is known as a unilateral fetal type PCA; the PcomA on the other side is patent. Bilateral fetal PCA variation was not observed in our population (c) Bilateral fetal type PCAs with both pre-communicating segments of the PCAs patent. (d) Unilateral PcomA present. (e) Hypoplasia or absence of both PcomAs and isolation of the anterior and posterior parts of the circle at this level. (f) Unilateral fetal type PCA and hypoplasia or absence of the pre-communicating segment of the PCA. (g) Unilateral fetal type PCA and hypoplasia or absence of the contralateral PcomA. (h) Unilateral fetal type PCA and hypoplasia or absence of both pre-communicating segment of the PCA and the PcomA. (i) Bilateral fetal type PCAs with hypoplasia or absence of bothpre-communicating segments of the PCAs. (j) Bilateral fetal type PCAs with hypoplasia or absence of the pre-communicating segment of either PCA.

The maximum arterial diameter was measured bilaterally in the supraclinoid internal carotid artery (ICA), anterior cerebral artery (ACA) A1 segment, middle cerebral artery (MCA) M1 segment, basilar artery, posterior cerebral artery (PCA) P1 and P2 segments from the proximal 5 mm portion. Vessel diameters were meas-

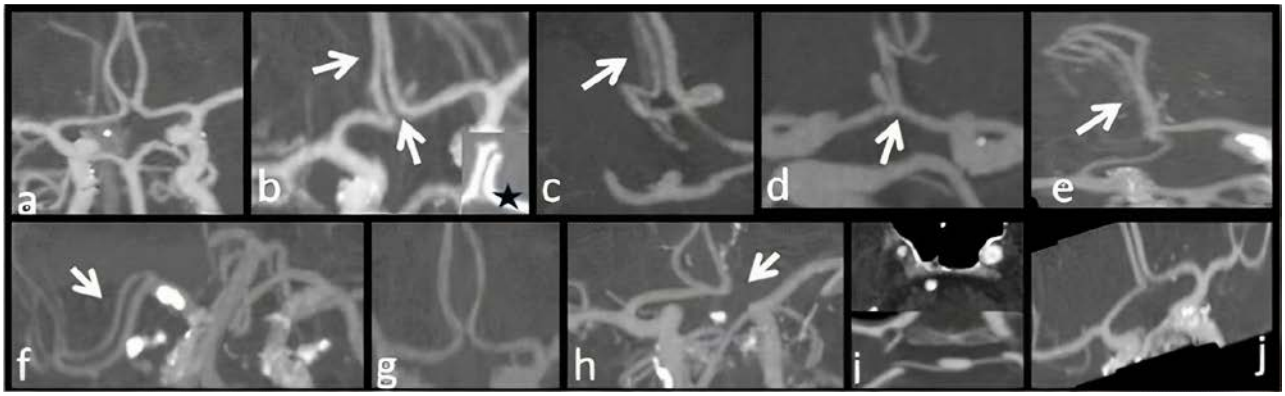

Figure 2: *Complete anterior system (a), two or more AcomA (b, arrow and star), the origin of the corpus callosum median artery from AcomA(c), short-segment fusion of the ACA(d), division of the ACA into two branches distally after a common trunk(e), the MCA originating from two separate vessels from the ICA(f), hypoplasia or absence of AcomA(g), unilateral hypoplasia or aplasia of the A1 segment with the other A2 segment originating from the existing A1 segment (h), hypoplasia or absence of one ICA(i), and hypoplasia/aplasia of AcomA with accompanying MCA arising as two separate branches(j).*

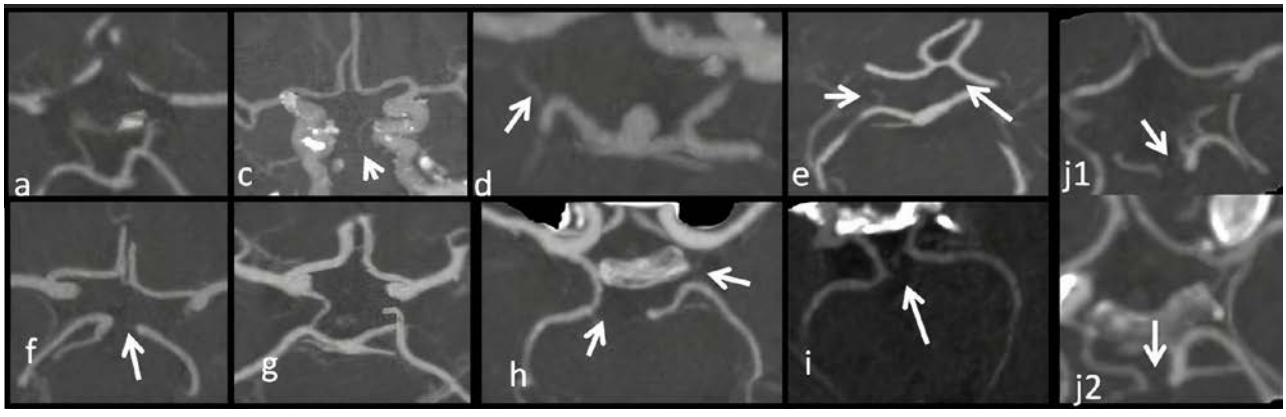

Figure 3: *a) Bilateral PcomAs present,(c) Bilateral fetal type PCAs, both pre-communicating segments patent, (d) Unilateral PcomA present(e) Hypoplasia or absence of both PcomAs, isolation of anterior and posterior parts of circle, (f) Unilateral fetal type PCA, hypoplasia or absence of pre-communicating segment, (g) Unilateral fetal type PCA, hypoplasia or absence of contralateral PcomA, (h) Unilateral fetal type PCA, hypoplasia or absence of both pre-communicating segment and PcomA, (i) Bilateral fetal type PCAs, hypoplasia or absence of both pre-communicating segments, (j) Bilateral fetal type PCAs, hypoplasia or absence of pre-communicating segment in either PCA*

ured by non-contrast enhanced CT and CT angiography in stroke patients.

Arterial diameter measurements were performed axial section, from the anterior wall to the posterior wall, with measurements taken from more proximal areas where luminal plaques were present as well as from normal segments.

The presence of accompanying aneurysms or vascular malformations was also noted. Radiological data were jointly evaluated by two radiologists with 14 and 16 years of experience in a consensus manner, ensuring the reliability of the findings.

Statistical Analysis: Descriptive data are presented as number (percentages) or median and interquartile ranges (25th - 75th percentiles) or mean ± standard deviation. Independent samples t-tests or non-parametric Mann-Whitney U-tests were used for the comparison of continuous variables between two groups. Pearson's chi-square or Fisher's exact tests were used for the categorical variables. Receiver operating characteristic (ROC) curve analysis was used to determine the optimal cutoff for points for measurements distinguishing between patients with and without infarct, and the area under the ROC curve (AUC), sensitivity and specificity were calculated. Multivariate logistic regression analyses were carried out to identify the predictors associated with infarct, with calculation of odds ratios (ORs) and 95% confidence intervals (95%CI). The analyses were performed using the Statistical Package for Social Sciences 25.0 for Windows (SPSS Inc., Chicago, Illinois, USA). The results were considered to be significant at a level of $p < 0.05$.

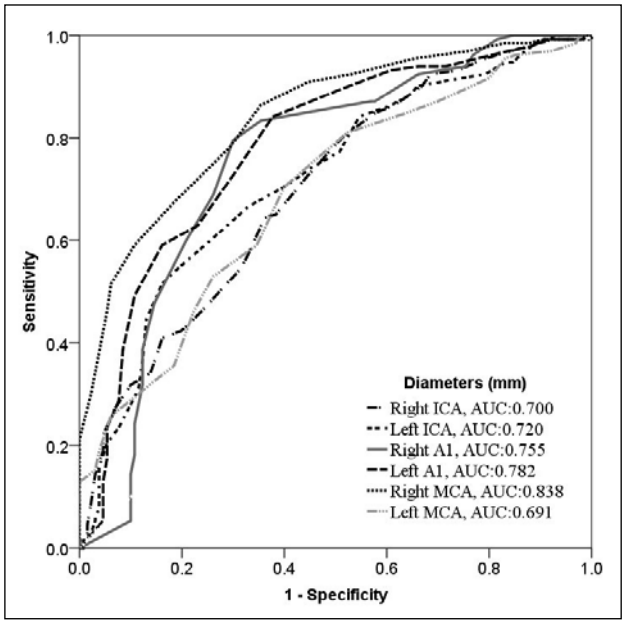

Figure 4: ROC curve analysis for ICA, A1 and MCA diameters for classifying infarct

Results:

A total of 262 patients were included in the study, comprising 132 patients in the study group and 130 patients in the control group. The mean age of patients in the study group was 70.54±12.24, while in the control group, it was 68.69±12.01, with no significant difference observed between the two groups. In the study group, 72 patients (54.5%) were male, and in the control group, 83 patients (63.8%) were male, with no significant difference in gender distribution between the two groups. When evaluating the study and control groups for cardiovascular risk factors such as hypertension, coronary artery disease, diabetes, and hyperlipidemia, no significant differences were found between the two groups. The univariate analysis of the characteristics for the patients in control and study groups were presented in Table 1.

Among the 262 patients included in the study, 134 (51.1%) had a complete WP anterior system without any variations. In the study group, 67 patients (50.8%) had WP variations, while in the control group, 61 patients (46.9%) had WP variations, resulting in a total of 128 patients (48.9%) with WP variations in the combined dataset. There was no statistically significant difference in the frequency of WP variations between the study and control groups (p=0.5). Among the 128 patients with WP variations, the following distribution of variation types was observed: 6 patients (4.6%) had Type B, 1 patient (0.8%) had Type C, 48 patients (37.7%) had Type D, 3 patients (2.3%) had Type E, 1 patient (0.8%) had Type F, 13 patients (10.0%) had Type G, 47 patients (36.9%) had Type H, 2 patients (1.5%) had Type I, and 7 patients (5.4%) had Type J variations (Table 2). The types of variations in the patient and control groups are detailed in Table 2.

Among the 262 patients included in the study, 19 (7.3%) had a complete WP posterior system without any variations. In the study group, 124 patients (93.9%) had WP variations, while in the control group, 119 patients (91.5%) had WP variations, resulting in a total of 243 pa-

Table 1. Demographics, cardiovascular risk factors and degrees of control and study groups.

	Total (n=262)	Study Group (n=132)	Control Group (n=130)	P
Demographics				
Age (years)	69.19±12.15	70.54±12.24	68.69±12.01	0.219
Gender, male	155 (59.2%)	72 (54.5%)	83 (63.8%)	0.126
Cardiovascular risk factors				
Hypertension	129 (49.2%)	66 (50.0%)	63 (48.5%)	0.803
Coronary artery disease	51 (19.5%)	26 (19.7%)	25 (19.2%)	0.924
Diabetes	77 (29.4%)	38 (28.8%)	39 (30.0%)	0.830
Hyperlipidemia	102 (38.9%)	53 (40.2%)	49 (37.7%)	0.683

Values are expressed as n (%) or means ± SD. For categorical variables Pearson's chi-square or Fisher's exact test are used. For continuous variables, p-values are calculated using independent samples t-test. Bold p-values indicate statistical significance at a<0.05.

Table 2. Comparisons of diameters of right and left ICA, A1 and MCA between control and study groups.

	Total (n=262)	Study Group (n=132)	Control Group (n=130)	P
Right ICA (mm)	4.23±0.77	3.97±0.70	4.50±0.74	**<0.001**
Left ICA (mm)	4.27±0.86	3.97±0.75	4.56±0.86	**<0.001**
Right A1 (mm)	2.0 (1.6-2.4)	2.2 (2.0-2.5)	2.8 (2.5-3.0)	**<0.001**
Left A1 (mm)	2.1 (1.8-2.4)	2.6 (2.3-2.9)	2.9 (2.6-3.2)	**<0.001**
Right MCA (mm)	2.53±0.54	2.23±0.46	2.83±0.43	**<0.001**
Left MCA (mm)	2.77±0.47	2.62±0.46	2.93±0.43	**<0.001**
Aneurism	17 (6.5%)	6 (4.5%)	11 (8.5%)	0.198
Anterior WP variation	128 (48.9%)	67 (50.8%)	61 (46.9%)	0.535
Anterior WP variation type				0.911
1 (type B variation)	6 (4.6%)	3 (4.2%)	3 (5.1%)	
2 (type C)	1 (0.8%)	1 (1.4%)	0 (0.0%)	
3 (type D)	48 (37.7%)	28 (42.3%)	20 (32.2%)	
4 (type E)	3 (2.3%)	2 (2.8%)	1 (1.7%)	
5 (type F)	1 (0.8%)	0 (0.0%)	1 (1.7%)	
6 (type G)	13 (10.0%)	7 (9.9%)	6 (10.2%)	
7 (type H)	47 (36.9%)	22 (32.4%)	25 (42.3%)	
8 (type I)	2 (1.5%)	1 (1.4%)	1 (1.7%)	
9 (type J)	7 (5.4%)	4 (5.6%)	3 (5.1%)	

Values are expressed as n (%), means ± SD or median (25^{th} – 75^{th} percentile). For categorical variables Pearson's chi-square or Fisher's exact test are used. For continuous variables, if values are reported in means, p-values are calculated using independent samples t-test; if values are given in medians, p-values are calculated using Mann Whitney U test. Bold p-values indicate statistical significance at a<0.05. ICA = Internal carotid artery CCA: Common carotid artery

tients (92.7%) with WP variations in the combined dataset. There was no statistically significant difference in the frequency of WP variations between the study and control groups (p=0.5). Among the 243 patients with WP variations, the following distribution of variation types was observed: 14 patient (5.3%) had Type C, 57 patients (21.8%) had Type D, 128 patients (48.9%) had Type E, 4 patient (1.5%) had Type F, 2 patients (0,8%) had Type G, 24 patients (9.2%) had Type H, 7 patients (2.7%) had Type I, and 7 patients (2.7%) had Type J variations.

The presence of accompanying aneurysms was noted in 6 patients in the study group and 11 patients in the control group, with no statistically significant difference observed between the two groups (p=0.1) (Table 2).

The diameters of right and left ICA, A1 and MCA were significantly higher in the control group, compared to the study group (p<0.001) (Table 2).

ROC curve analysis (Figure 3) revealed that the diameters right and left of ICA, Anterior cerebral artery A1 segment and middle cerebral artery M1 segment have the ability to detect infarct with high accuracy rates. Table 3 juxtaposes characteristics of the predictive power of the ROC curve analysis. For ICA, an accuracy of 0.700 (95%CI: 0.637-0.762, p<0.001) and 0.720 (95%CI: 0.659-0.782, p<0.001) for right and left ICAs were obtained, respectively. Optimum cut-off values for the right and left ICA diameters of ≤4.5 mm and ≤3.95 mm were found as the optimal cutoffs for infarct, respectively. When A1 diameters considered, we found that a right diameter of ≤2.05 mm and a left diameter of ≤2.25 mm were optimal cutoffs for identifying patients with infarct (area under the curve of 0.755 (95%CI: 0.694-0.816, p<0.001) and 0.782 (95%CI: 0.726-0.839, p<0.001), respectively). Calculation of the MCA revealed that the optimal cutoff values for right diameter is ≤2.65 mm

Table 3. Performance of ICA, A1 and MCA diameters for classifying infarct.

Diameters	Optimum cut-off	AUC	95% CI	p[a]	Sensitivity	Specificity
Right ICA (mm)	4.55	0.700	0.637-0.762	<0.001	48.5%	80.3%
Left ICA (mm)	3.95	0.720	0.659-0.782	<0.001	51.5%	83.8%
Right A1 (mm)	2.05	0.755	0.694-0.816	<0.001	79.5%	70.0%
Left A1 (mm)	2.25	0.782	0.726-0.839	<0.001	62.3%	84.1%
Right MCA (mm)	2.65	0.838	0.791-0.886	<0.001	86.4%	64.6%
Left MCA (mm)	2.85	0.691	0.627-0.754	<0.001	70.5%	60.0%

ICA: Internal carotid artery, Anterior cerebral arteryy A1 segment, Middle cerebral artery M1 segment, AUC: Area under the curve, CI: Confidence Interval, [a]Hypothesis test for H_0:AUC=0.5

Table 4. Comparisons of diameters of basilar artery and the posterior cerebral artery's P1 and P2 segment between control and study groups.

	Study Group (n=132)	Control Group (n=130)	p
Basilary artery (mm)	3.36±0.81	3.27±0.74	0.345
Left P1 (mm)	1.96±0.75	1.94±0.75	0.864
Right P1 (mm)	1.88±0.78	1.96±0.63	0.363
Left P2 (mm)	1.98±0.36	1.93±0.35	0.113
Right P2 (mm)	2.00±0.34	1.94±0.38	0.476

Values are expressed as n(%), means ± SD or median (25^{th} – 75^{th} percentile).

and a left diameter is ≤2.85 mm with the accuracies of 0.838 (95%CI: 0.791-0.886, p<0.001) and 0.691 (95%CI: 0.627-0.754, p<0.001), respectively.

The diameter measurements of left A1 (OR: 0.306; 95%CI: 0.160-0.586; p<0.001), right MCA (OR: 0.027; 95%CI: 0.007-0.098; p<0.001) and left MCA (OR: 4.575; 95%CI: 1.340-15.613; p=0.015) were also identified as statistically significant predictors of infarct. Although the diameters of Right and Left ICA, and Right A1 found statistically significantly difference between the two groups in the univariate analysis, these measurements lost their significant in the multivariate analysis.

Measurements of diameter from the basilar artery and the posterior cerebral artery's P1 and P2 segment revealed no significant difference between the two groups (p > 0.05) (Table 4).

Discussion:

The most significant finding of our study, which indicates the lack of a significant difference in WP variations between the ischemic stroke patient group and the comparison group. The results of studies examining the relationship between WP variations and infarcts are conflicting in the literature. In numerous studies, it has also been demonstrated that there is no significant association between WP variations and the risk of ischemic stroke, consistent with our findings [17-19]. However, in another studies were observed that some Willis polygon variations were more prevalent in ischemic stroke patients compared to the control group [20-22]. These contradictory results emphasize the need for further research to better understand the relationship between Willis polygon variations and the risk of infarcts.

Another result of our study is that the most frequently observed variation in the anterior system among the infarct group was Type D (short-segment fusion of the ACA) in 28 patients, followed by Type H in 22 patients. Conversely, the most common posterior system variation in both the infarct and control groups was Type E (Hypoplasia or absence of both PcomAs and isolation of the anterior and posterior parts of the circle at this level.

Another important finding of our study is the decrease in diameter observed in the ICA, A1, and M1 segments in the ischemic stroke patient group. In our study, only anterior circulation infarctions were included, and while arterial diameters in the anterior system were found to be significantly lower, no significant difference was found in the arterial diameters forming the posterior system. Arterial diameter measurements were performed axially, from the anterior wall to the posterior wall, with measurements taken from more proximal areas where luminal plaques were present as well as from normal segments. Based on this, we believe that the arteries being of a smaller caliber than normal may be a contributing factor to the development of infarction. The scarcity of arterial calibrations in the Willis polygon can be pathophysiologically linked to atherosclerosis and susceptibility to infarction. For instance, Jebarı-benslaıman, Shifa et al. [23] demonstrated that atherosclerosis is associated with vascular wall damage and plaque accumulation, which can lead to a reduction in arterial calibrations. Additionally, Wijesinghe et al. [24] suggested that the decrease in arterial diameters in the Willis polygon may increase the risk of infarction due to flow restrictions caused by atherosclerotic plaques. These findings indicate that the scarcity of arterial calibrations in the Willis polygon may contribute to the development of atherosclerosis and subsequently increase the risk of infarction. Therefore, understanding the relationship between the scarcity of arterial calibrations and susceptibility to atherosclerosis and infarction plays a crucial role in elucidating the underlying pathophysiological mechanisms.

Various studies suggest that the reduction in arterial diameters forming the Willis polygon is associated with cerebrovascular diseases. For instance, studies concerning white matter changes indicate that these reductions are linked with hyperintensities in the white matter, emphasizing the long-term effects of inadequate cerebral blood flow [25]. Therefore, the reduction in arterial diameters may play a significant role in the pathophysiology of ischemic stroke, consistent with our study findings, underscoring the need for further research in this area.

One of the primary limitations of our study is its single-center, retrospective design, and the relatively small sample size. A larger-scale, multicenter investigation would be needed to provide a more comprehensive understanding of the frequency of variations and vascular diameters in this patient population.

In conclusion, as a result of increased awareness regarding the relationship between decreased arterial

diameters, Willis polygon variations, and stroke, we believe that incorporating detailed information such as arterial diameters and variations related to the Willis polygon in the reports of patients undergoing imaging due to stroke risk will provide additional contributions to patient management. Additionally, we are of the opinion that detailed mapping of the Willis polygon (including variations and arterial diameters) before endovascular thrombectomy, which is an important treatment option for this patient group, could provide additional information facilitating the interventional procedure prior to the endovascular thrombectomy, we believe that detailed mapping of the Willis polygon (including variations and arterial diameters) before endovascular thrombectomy, which is an important treatment option for this patient group, could provide additional information facilitating the interventional procedure. **R**

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