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Abstract: Objective: Willis polygon forms the basis of the arterial circulation of the cerebrum. Willis polygon is a vascular structure whom variations are not rare. Knowledge of the anatomy and preservation of its integrity is crucial for performing neurovascular surgery and intracranial tumour surgery. Because of the important vascular and neurological structures, approaches to this region are considered extremely risky. One of the main variations in-person basis is the diameter differences of the arteries, which forms Willis polygon, between the left and right hemispheres. About structure and variations, studies of Rhoton and Yasargil had formed the touchstone. Our aim is to contribute to the literature and clinical studies, to be done in the future, by comparing our results with previous studies about variations and morphometric features of Willis polygon. Methods: Arteries of 30 fresh cadaver brains were examined during autopsies in T.C. Ministry of Justice Istanbul Forensic Science Institute. Bilaterally anterior cerebral artery A1 segment lengths, distance between anterior communicating artery-callosomarginal artery outputs, posterior cerebral artery P1 segment lengths were measured using a digital calliper. After dissections and measures, photos of the region were taken and vascular anatomy and variations noted. From every single cerebrum samples were obtained from bilaterally A1, A2, callosomarginal artery, middle cerebral artery, posterior communicant artery, P1 and basilar artery. Samples were fixed by using 10% buffered-formalin. Taken samples were transported to Marmara University Faculty of Medicine, Department of Anatomy Laboratory. Samples were examined and interior diameters were measured under the microscope. Results: Our results with artery diameters and lengths were similar with literature. Different from literature, in anterior cerebral artery A1 segment, posterior cerebral artery P1 segment and posterior
communicant artery no aplasia were noted. In 50% of the samples, callosomarginal artery were originate from A2 segment. In one case, we observed left and right pericallosal arteries were joined together at the end of the A2 segment and continued as a single pericallosal artery. We could not find any information about this variation in the literature. Conclusion: Before surgical operations, detailed knowledge of Willis polygon and evaluation of the pre-op cerebral angiography considering possible variations, reduce mortality and morbidity ratios. In addition, because of the role of flow gradients of Willis polygon in aneurysm formation, and in terms of better understanding the collateral circulation which is important in vascular occlusive diseases and vascular surgery, we believe, more anatomic studies about this region needed.

Key words: Willis polygon, Circle of Willis, morphometric analysis

Introduction

Willis polygon forms the basis of the arterial circulation of the cerebrum (Figure 1). Willis polygon is a vascular structure whom variations are not rare (1, 3). Knowledge of the anatomy and preservation of its integrity is crucial for performing neurovascular surgery and intracranial tumour surgery. Because of the important vascular and neurological structures, approaches to this region are considered extremely risky (16). One of the main variations in-person basis is the diameter differences of the arteries, which forms Willis polygon, between the left and right hemispheres (5). About structures and variations, studies of Rhoton and Yasargil had formed the touchstone (10, 16).

Our aim is to contribute to the literature and to the clinical studies, to be done in the future, by comparing our results with previous studies about variations and morphometric features of Willis polygon.

Material and method

To evaluate the morphological characteristics of the Willis polygon and its constituent arteries in the Turkish population, arteries of 30 fresh cadaver brains (60 hemispheres) were examined during routine autopsies in T.C. Ministry of Justice Istanbul Forensic Science Institute. All the protocols were conducted with the consent of T.C. Ministry of Justice Istanbul Forensic Science Institute, Board of Science. For the study, cadavers aged between 25 – 50 years old, without head injury were selected. During routine autopsies, after calvarium removal, cerebrums were removed with caution by preserving vessels. On removed cerebrums, arteries of Willis polygon were dissected macroscopically and examined (Figure 2). Bilaterally anterior cerebral artery (ACA) A1 segment lengths, distance between anterior communicating artery (AComA)-callosomarginal artery (CMA) outputs, posterior communicating artery (PComA), posterior cerebral artery(PCA) P1 segment lengths were measured using digital calliper. After dissections and measurements, vascular anatomy and variations were documented by taking photos of the region.

From every single cerebrum, samples were obtained from bilaterally A1, A2, CMA, middle cerebral artery (MCA), PComA, P1 and basilar artery (BA). Samples were fixed by using 10%
buffered-formaldehyde. Taken samples were examined and interior diameters were measured under a microscope in Marmara University Faculty of Medicine, Department of Anatomy Laboratory (Figure 3).

Statistical evaluations were made using The Statistical Package for Social Sciences (SPSS) 22.0 (Chicago, IL). All continuous variables were reported as median±standart deviations (SD).

**Figure 1** - Illustration of arteries that form Willis Polygon. ACA: Anterior cerebral artery, ACoA: Anterior communicating artery, MCA: Middle cerebral artery, PCoA: Posterior communicating artery, PCA: Posterior cerebral artery, BA: Basilar artery

**Figure 2** - Arteries of Willis Polygon. ACA: Anterior cerebral artery, ACoA: Anterior communicating artery, MCA: Middle cerebral artery, PCoA: Posterior communicating artery, PCA: Posterior cerebral artery, BA: Basilar artery

**Figure 3** - Microscopic image of an artery sample

**Results**

In our study, we used 30 cadaver cerebrums with no head trauma (14 female, 16 male).

**A1 Segment**

A1 segment diameters on the right were measured as maximum 2104.593 µ, minimum 1039.152 µ and median was 1540.092±366.9 µ. On the left, maximum 2699.584 µ, minimum 1247.152 µ and median was 1841.398±355.0 µ (table 1).

A1 segment lengths on the right were measured as maximum 18mm, minimum 10mm and median was 13.56±2.25mm. On the left, maximum 17mm, minimum 10mm and median was 13.76±1.87mm (table 1) (Figure 4).

**A2 Segment**

A2 segment diameters on the right were measured as maximum 2185.284 µ, minimum 1627.145 µ and median was 1860.174±129.5 µ. On the left, maximum 2148.649 µ, minimum 1699.004 µ and median was 1841.061±14.6 µ (table 1).

A2 segment lengths on both right and left were measured as maximum 25mm,
minimum 14mm. Median on the right was 18.83±3.81mm and on the left was 18.73±3.02mm (table 1) (Figure 4).

**Posterior Communicating Artery**

PComA diameters on the right were measured maximum 1394.94µ, minimum 638.17µ and median was 881.37±170.9µ. On the left, maximum 1410.156µ, minimum 648.95µ and median was 892.247±201.4µ.

Posterior communicant artery lengths on the right were measured as maximum 26mm, minimum 11mm and median was 15.63±3.39mm. On the left, maximum 24mm, minimum 11mm and median was 15.9±3.24mm (table 1).

**P1 Segment**

P1 segment diameters on the right were measured as maximum 2529.639 µ, minimum 902.703µ and median was 1807.549±402.42µ. On the left, maximum 2548.47µ, minimum 948.29µ and median was 1809.082±350.05µ (table 1).

P1 segment lengths on the right were measured as maximum 11mm, minimum 5mm and median was 7.033±1.44mm. On the left, maximum 10mm, minimum 5mm and median was 6.933±1.2mm (table 1) (Figure 5).

**Middle Cerebral Artery**

MCA diameters on the right were measured maximum 3017.994µ, minimum 2645.399µ and median was 2819.351±101.01µ. On the left, maximum 2992.678µ, minimum 2649.495µ and median was 2819.284±94.18µ.

**Basilar Artery**

Basilar artery diameters were maximum 3040.072µ, minimum 1939.17µ and median was 2722.276±247.57µ (Figure 5).

**Callosomarginal Artery**

CMA diameters on the right were measured as maximum 1489.14µ, minimum 836.447µ and median was 1232,402±361,99µ (Figure 6).

CMA was observed in 3 cases only on the right side and in 1 case, only on the left side. In two case bilaterally CMA was absent.

The origins of CMA were, left A3 in 10 cases, left A2 in 15 cases and right A3 in 12 cases, right A2 in 15 cases. In 11 cases, CMA were originated from different segments.

<table>
<thead>
<tr>
<th>TABLE 1</th>
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<tbody>
<tr>
<td>Diameter and length measurements of arteries of the Willis Polygon as mean, minimum and maximum</td>
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<table>
<thead>
<tr>
<th>Blood vessels</th>
<th>Mean</th>
<th>Min.</th>
<th>Max.</th>
</tr>
</thead>
<tbody>
<tr>
<td>A1 Diameter (µ) - Right</td>
<td>1540.092±366.9</td>
<td>1039.153</td>
<td>2104.593</td>
</tr>
<tr>
<td>Diameter (µ) - Left</td>
<td>1841.398±355.0</td>
<td>1247.152</td>
<td>2699.584</td>
</tr>
<tr>
<td>Length (mm) - Right</td>
<td>13.56±2.25</td>
<td>10</td>
<td>18</td>
</tr>
<tr>
<td>Length (mm) - Left</td>
<td>13.76±1.87</td>
<td>10</td>
<td>17</td>
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<tr>
<td>Artery</td>
<td>Diameter (µ)</td>
<td>Length (mm)</td>
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<tr>
<td><strong>A2</strong></td>
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<tr>
<td>Diameter - Right</td>
<td>1860.17±129.5</td>
<td>16.83±1.18</td>
<td></td>
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<tr>
<td>Diameter - Left</td>
<td>1841.06±14.6</td>
<td>18.73±3.02</td>
<td></td>
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<tr>
<td><strong>PComA</strong></td>
<td></td>
<td></td>
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<tr>
<td>Diameter - Right</td>
<td>881.37±170.9</td>
<td>15.63±3.39</td>
<td></td>
</tr>
<tr>
<td>Diameter - Left</td>
<td>892.24±201.4</td>
<td>15.9±3.24</td>
<td></td>
</tr>
<tr>
<td><strong>P1</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diameter - Right</td>
<td>1807.54±402.42</td>
<td>7.03±1.44</td>
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<tr>
<td>Diameter - Left</td>
<td>1809.08±350.05</td>
<td>6.93±1.2</td>
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<tr>
<td><strong>MCA</strong></td>
<td></td>
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<tr>
<td>Diameter - Right</td>
<td>2819.35±101.01</td>
<td>5.03±1.44</td>
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<tr>
<td>Diameter - Left</td>
<td>2819.28±94.18</td>
<td>5.93±1.2</td>
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<tr>
<td><strong>BA</strong></td>
<td></td>
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<tr>
<td>Diameter - Right</td>
<td>1232.40±152.51</td>
<td>1267.75±361.99</td>
<td></td>
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<tr>
<td>Diameter - Left</td>
<td>1267.75±361.99</td>
<td>1267.75±361.99</td>
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</table>

**Figure 4** - A1 and A2 segments of ACA

**Figure 5** - P1 segment of PCA and BA
Discussion

The blood circulation of the cerebrum is formed by 4 arteries as two internal carotid arteries and two vertebral arteries. Internal carotid arteries form the anterior system and vertebral arteries form the posterior system. At the base of the cerebrum, the vascular web that creates the connection between these two systems is named as the “circle of Willis”, or “Willis polygon” (1, 10).

Vessels participating the structure of the polygon are two ICA’s, two ACA’s, AComA, two PComA’s, two PCA’s, BA and two MCA’s (1, 9). Willis polygon provides collateral circulation between vertebrobasilar system and carotid system, and in-between cerebral hemispheres. Direction of flow inside the polygon is determined by vessel diameters and pressure gradient. The efficiency of this collateral circulation is the main determinant of the degree of neurological deficits, in case of stenosis or occlusion in one of the main feeder arteries (2, 6).

Many variations in the anatomy of the Willis polygon had been shown and the configuration which could be described as normal is seen in only 50% of the population (6, 9, 11). Approaches to this region are frequently caused by tumours, endovascular interventions and vascular pathologies such as aneurysms and arteriovenous malformations. With better understanding of the region anatomy, lower complication and rates better outcomes would be achieved. Besides differences in configuration, variations in Willis polygon are also caused by differences in vessel diameters and lengths (11).

In our study, in 19 (63%) cadavers left A1 inner diameters were measured wider and in 11 (37%) cadavers right A1 inner diameters were wider. Differences were significant in 15 (50%) cadavers for the left A1 and 8 (26.6%) cadavers for the right A1. In 13 cases (43%), left A1 were longer than right A1 and in 9 cases (30%) vice versa. In 8 cases (27%) bilaterally A1’s were the same length.

Diameter differences between A1 segments have great value on formation mechanisms of AComA aneurysms. As a general rule, in case of diameter difference between A1 segments, AComA aneurysms were originated from the wider side. If the left and right A1 diameters were equal then ACom aneurysms more likely to originate from the middle portion (14, 15). It is reported that, in 10%, A1 segment could be one sided hypoplastic or aplastic (12). A1 hypoplasia is associated with anterior communicating artery aneurysms in 85% of cases. This is the only known variant which is correlated with cerebral aneurysm localization and formation (9). In our study, we did not observe any hypoplastic or aplastic A1 segment. A1 segment aneurysms forms 1.4% of all intracranial aneurysms (15). Ipsilateral fronto-temporo-sphenoidal craniotomy were
suggested as most suitable approach for aneurysms of this area (15).

In 19 cadavers (63%) right A2 inner diameter was wider than left A2. In 10 cadavers (33%) left A2 was wider but the difference was not significant. In one case we observed that after AComA, left ACA was ended with branching on the medial surface of left hemisphere. The right ACA was continuing as the only pericallosal artery, and then in the beginning of A3 divided and continued as two pericallosal arteries. In literature it is reported that pericallosal arteries might origin from single log, but no ratio was reported (12). In our study, occurrence rate of this variation were 3.3%. In addition, in one case we observed that left and right pericallosal arteries were joined together at the end of A2 and continued as one single pericallosal artery. We could not find any information about this variation in the literature.

In Yasargil’s series, pericallosal artery aneurysms are reported as 5.6% of ACoA-ACA aneurysms, and as 2.3% of the all intracranial aneurysms (15). Pericallosal artery aneurysms are mostly seen on A2 segment (4). In our literature research, we noted that on proximal side of CMA and frontopolar artery, aneurysms were more frequent (1, 15).

Interhemispheric approach, following paramedian frontal craniotomy, has been been using for pericallosal artery aneurysms (15). Distal ACA aneurysm surgeries have its own difficulties. Because of interhemispheric fissure and callosal cistern are narrow areas, lumbar drainage would be helpful to widen the workspace. Falx depth could be low and consequently there might be adhesions between both cingulate gyruses. Aneurysms of this area are often large based and sclerotic, and frequently involve the vessels which branch from here. Sclerosis could occur on both aneurysm fundus and contralateral pericallosal artery, as a result adhesions between these two structures may occur. This can cause difficulties during dissection. Some of the time, it is hard to tell, from which ACA, the aneurysm was originated and angiography would not be clear. Dome of the aneurysm can be adhered to piamater over cingulate gyrus or it could be buried inside the cingulate gyrus. For all of these, retraction of frontal lobe should be done minimal and with extreme caution. (15).

ACoA is the artery, where the most of the variations observed in Willis polygon (7). It can be as a single line or lines or as a vascular web (10). In our study, in 21 cadavers (70%) ACoA was as a single line; in 2 cadavers (6.6%) it was as a conjoint of both ACA’s; in 4 cadavers (13.3%) it was formed by 3 or 4 thin vessels; in 3 cadavers (10%) it was web-formed. Because of the large number of studies about ACoA and it is a detailed, deep subject; morphological analysis of this artery was not conducted.

In 12 of the cadavers (40%) left PCA inner diameter was wider than right PCA. In 18 cadavers (60%) right PCA was wider. There were significant differences on 7 cases (4 left PCA (13.3%), 3 right PCA (10%)). In 18 cadavers (60%) we measured the left PCA longer than right, and in 12 cadavers (40) right PCA was longer.

Basilar artery starts with two vertebral arteries joining together in pontomedullary
sulcus area and continues through prepontine cistern. With age, basilar artery may become more twisted, longer and the bifurcation may be positioned at a higher level (16). 15% of the saccular aneurysms occur in vertebrobasilar system, and 63% of them occur on basilar bifurcation. The concomitance of anomalies such as hypoplastic PCA or PCA, with basilar bifurcation aneurysms, is significantly high (8).

In 3 of the cadavers (10%), only left callosomarginal artery were missing. Right callosomarginal artery was missing in only one cadaver (3,3%). In 2 cadavers (6,6%) bilaterally callosomarginal arteries were absent. In Rhoton’s series, it is reported that callosomarginal artery were missing in 20% of the examined hemispheres (8)(66). In our study, among 60 hemispheres, the callosomarginal artery was absent in total 8 hemispheres.

Callosomarginal artery is the largest branch of the pericallosal artery and mostly takes origin from the A3 segment, but it can also take origin from A2 or A4 (9). In our study, callosomarginal artery was taking origin from left A3 in 10 cases, left A2 in 15 cases, right A3 in 12 cases and right A2 in 15 cases. In 11 cases, callosomarginal arteries origins were different segments. In total 60 hemispheres, 22(36.6%) of the callosomarginal artery were originated from A3 segments and 30 of the arteries (50%) were originated from A2 segments.

We saw no significant relationship between artery lengths and diameters.

In our study, our results were consistent with the literature most of the time. It is reported that A1, P1 and PCoA segments could be aplastic in 10%; but we did not encounter this variation in any of our cases.

**Conclusion**

Flow strength, intravascular pressure, turbulent flow and artery wall disorders in bifurcation areas takes part in aneurysm pathophysiology (13). Willis polygon, which provides a collateral circulation between carotid and vertebrobasilar system, is a place that intracerebral aneurysms frequently take place. The gold standard treatment of the intracranial aneurysm is still the clipping. Mastering the anatomy of Willis polygon and pre-operative analyses of angiography, considering possible variations, is crucial to decrease mortality and morbidity of this serious operations. In terms of better understanding the collateral circulation which is important in vascular occlusive diseases and vascular surgery, we believe, more anatomic studies about this region needed.

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**References**

1Alpers BJ, Berry RG, Paddison RM. Anatomical studies of the circle of Willis in normal brain. AMA archives of neurology and psychiatry 1959;81:409-418.