Anatomic variations of cerebral circulation on 3D TOF MRA in Montenegrin population

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Authors: M. Abramovic, S. Cejovic; Podgorica, Montenegro/ME  
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Aims and objectives

The gold standard for detection and differentiation of vascular anomalies remains digital subtraction angiography, despite its invasive nature (1-3). Nowadays 3D time of flight (TOF) magnetic resonance angiography (MRA) is proven to be sensitive and non-invasive method in evaluating normal and variant cerebral vessels, which uses the blood flow pattern or velocity of moving blood to detect cerebral arteries, and thus reduces need for invasive DSA to minimum (4-6).

Knowledge of the vascular anatomic variations is important in neurology, neurosurgery and interventional neuroradiology and has different impact on each, depending on the type of variation (7-9).

The aim of our study was to investigate the incidence of cerebral arterial variations in Montenegrin population using 3D TOF MRA technique, compare the acquired data with available anatomical, microsurgical, CT and MR angiographic studies, and analyze differences in their outcomes.
Methods and materials

The study was performed in Clinical Center of Montenegro, from September 2016. to September 2017. We performed MR angiographies on 143 patients, who underwent MRI for different reasons and had no previous history of vascular anomalies or manifestations of cerebrovascular disease. Patients were informed about the study and their consent was obtained. Patients were subjected to MRI scanning on 1.5 T Siemens Avanto scanner with a 3D time of flight protocol. Axial source images, multi direction volume rendering (VRT) and maximum intensity projection (MIP) images were evaluated by experienced neuroradiologist and radiology resident.
Results

The circle of Willis represents an arterial cycle, connecting carotid and vertebrobasilar system\cite{10,11}.

The carotid system, or anterior cerebral circulation, consists of two internal carotid arteries (ICA), and their terminal branches - anterior and middle cerebral arteries (ACA and MCA). ACAs are connected by a single anterior communicating branch (AcoA). \cite{12} Posterior circulation is consisted of two vertebral arteries (VA) which unite to form basilar arteries (BA) and give off posterior inferior cerebellar arteries (PICA). BA gives two pair of side branches - anterior inferior cerebellar arteries (AICA) and superior cerebellar arteries (SCA), and two final branches - posterior cerebral arteries (PCA) \cite{2,13}

The complete circle of Willis was present in 28%, and only 9,2% patients had all branches of basilar and vertebral arteries present with textbook described origin. Comparing it to other studies we found that results show large range of values: from 16,6% to 46,7% \cite{1,14,15} in angiographic studies, and from 45,2% \cite{16} to 91,4% \cite{11} in anatomical studies. Difference between studies can be due to fact that certain studies were performed on healthy patients, other on patients with different neurovascular diseases. Hartkamp et al, who analyzed MRA results on patients with ICA obstruction and healthy patients, report significantly higher percentage of complete circle in the first group \cite{17} while Kwak et al did a similar study, and had different results, where complete circle was more present in young, healthy persons \cite{18}. We presented, in table No2, results of few other MR angiographic studies in order to compare it with ours.

The variants of individual arteries include aplasia or hypoplasia, absence of vessel, variable origin, course or termination, duplication, fenestrations, presence of accessory or persistent primitive vessels.

Azygos ACA (unpaired ACA) is presented as a single trunk or common A2. Incidence range 0,2 - 4%, depending on the type of study, where higher incidence is reported on angiographic studies \cite{19,20}. We found only one case of azygos ACA (0,7%). The significance is related to the fact it supplies both hemisphere, which can result in enormous ischemic area in cases of its occlusion. Some authors relate azygos variant with increased risk of aneurysm \cite{21}, while others report high percentages of saccular aneurysms up to 41,1% \cite{22} and even 71% \cite{12}.

Median artery of corpus callosum (MACC) (figures No1, No3, No11) is a branch of ACoA which runs parallel to and behind pericallosal artery. Its incidence varies from 3-22% \cite{23,24}. In our study it was present in 8,4% of cases, while in other MRA studies its percentage is lower. The greatest significance lays in the fact it may become the draining artery of ACoA aneurism, which can lead to its oversight on the angiography. \cite{19}
Aplasia or hypoplasia of A1 segment (figures No2, No4, No5) usually leads to dilatations of contralateral A1 segment and therefore promote the formation of aneurysm (14, 25). In our study there were only 4.9% cases. Anatomical studies range from 0% (26) to 10% (3,27), while on MRA this percentage is 2.6% (18) up to 10% (15). Krzyzewski et al (28) did a CT angiographic study on patients with proven AcoA aneurysm, and 36% of them had hypo or aplastic segment of A1 segment.

Fenestrations are defined as the division of the arterial lumen into separate channels, where each of them has its own endothelial and muscular layer, and common or separate adventitia (3, 4, 12, 29,30,31). Fenestration of ACA range from 0, through 4% to 7.2% (32,33,19) in anatomic studies, and in angiographic studies 0.058%, 0.36% -0.62% (CTA and MRA), 0.8% (MRA) up to 1.2% (MRA) (7,31,34,23). Our two cases (1.4%) were found on A1 segment (figure No3).

Duplications of ACoA are seen in 18% (27,30) to 40% (35) in anatomical studies. Fenestrations of ACoA are seen in 12-21% (36), 22% (37) to 40% (19) in anatomical studies and on angiographies in 5,3% (36)- 10% (12). We found only 3 cases of fenestrations - 2,1% (figures No4 and No5). In large 3DRA study fenestration of ACoA was related to aneurysm in 83% of cases (36). In our study more frequent was the absence of the ACoA - 23% (figure No6). Krzyzewski et al (28) report absence of ACoA in 7% on CT angiographies, while on certain microsurgical cadaveric study there was none (26). MRA studies also differ in results 11,64 (14) - 21,6% (15). Small percentage of fenestration and higher percentage of absent arterie, can be explained by the fact that bridging arteries can be too thin, with very slow blood flow and thus undetectable on 1,5T MRI machine. What we found that could be of importance is the statistically significant occurrence of absent ACoA with absent at least one PCoA (p<0.05), which can certainly compromise collateral circulation in cases of cerebrovascular diseases.

We found no variants of MCA. Anatomic variants of MCA include doubled MCA, accessory MCA, early branching of MCA and MCA fenestrations and are extremely rare (38). Accessory MCA was found only in 0.31% cases in 6000 participants (39), with another reported incidence 0,3% to 3% (19,41,40). MCA duplications are found in 0,2-2,9% (19,38,42,43) and fenestrations in 0,17% on angiographies and 1% in anatomic studies (44).

The most common variation in our study was the absence of PCoA, which was present in 45,5%, where 22,4% was unilateral (figures No1,No5,No7,No8), and 23,1% bilateral (figures No3,No4,No6,No11). Anatomical studies showed only 1% cases of missing PCoA (16), and MRA, 41%-69,2% (14,15). Stock et al (5) did a study by analyzing DSA and MRA images, and got the result of 26,85% absent PCoA on DSA and 38,9% on MRA images. Their absence can be important in development of collateral blood flow in cerebrovascular diseases, or when performing surgical procedures. We found only one case of double PCoA and no cases of PCoA fenestration, which corelates with published literature with only isolated report for PCoA fenestration (45), with 0% on large DSA and angiographic studies (7,46,47).
Fetal PCA (figure No2,No5,No7) arises from the terminal ICA, without communication with BA (full fetal PCA). If P1 segment is less in diameter than PCoA its partial fetal PCA (4,11,48) (figure No8). Sometimes the shift from the carotid to the basilar system during embryogenesis doesn't occur, embryonic PCoA fails to regress and so blood supply comes from ICA for occipital lobe (19,49). Incidence for fetal range from 11-46% (48). We found it present in only 14%, and differ from other compared MRA studies who got higher percentages. Clinical significance is related to treatment of ICA-PCoA aneurysm, where is important not to occlude fetal PCA and to avoid infarction of its territory (19). Also, atherosclerotic lesions in terminal ICA can be dislodged into PCA via PCoA, and lead to paradoxical PCA territory infarction (11,48). Only one case of PCA duplication was found (0,7%), which doesn't differ from published studies - 0,2% on CTA study and 1% in anatomical study (50,51). We found no cases of PCA fenestrations (in literature from 0%, 0,4% on DSA (7,46), 0% on CTA (47,50) and 1% in anatomical study) (51).

Common origin of PCA and SCA was present in 7,7% (figure No6), while variant origin of SCA from PCA was present in 4,9% (figure No9). In 8,4 % there was double SCA, of which in one case (0,7%) one was arising from BA and other from PCA (figure No9), and in one both arose from PCA (figure No9). Cases of common origin have high range incidence value in available literature 2-22% (19,52,53), as well as for its origin from PCA - from 4% (13,52), to 25,3% (53). Also, as for duplication of SCA, incidence in anatomic studies is higher than in our angiographic study, ranging from 11% (55) to 36% (54).

As for the inferior cerebellar arteries, most common variations were absence of AICA (37,8%) (figures No 2, No4, No5, No7, No8, No11) and of PICA (29,4%) (figure No8), which correlates with literature (2). We found statistically significant occurrence of co-absence of at least one of PICA and one AICA (p<0,05) which can probably affect the cerebellar vascularization, affected area in cases of infarction, any kind on neurosurgical or neurovascular procedures in posterior fossa. There was only one case of double PICA (0,7%) and double AICA was present in 5,6% (figure No10).

We found no cases of fenestrations of vertebral or basilar artery. Basilar artery fenestration incidence range 0,6%-1,9% (3,19) on angiographic examination and 5% on autopsies, and for vertebral artery 0,3-2% (3,19). Cooke et al reported that 52,6% of all fenestrations in their study was involving basilar and 19,7% vertebral artery. There are reports suggesting higher incidence of aneurysms in patients with BA or VA fenestrations (19). There was one case (figure No11) which couldn't be explained otherwise than presence of communication between vertebral arteries, since it didn't have adequate shape to be pronounced as fenestration of vertebral artery. Similar case was described by Kovac et al, (50).
Table 1: Display of all detected variants

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<table>
<thead>
<tr>
<th>Display of all detected variants</th>
<th>Left</th>
<th>Right</th>
<th>Bilateral</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complete circle of Willis</td>
<td></td>
<td></td>
<td></td>
<td>28%</td>
</tr>
<tr>
<td>Complete anterior and posterior circulation - with PICA, AICA and SCA</td>
<td></td>
<td></td>
<td></td>
<td>9,2%</td>
</tr>
<tr>
<td>Azygos ACA</td>
<td></td>
<td></td>
<td></td>
<td>0,7%</td>
</tr>
<tr>
<td>Median artery of corpus callosum</td>
<td></td>
<td></td>
<td></td>
<td>8,4%</td>
</tr>
<tr>
<td>Hypo/aplastic A1 segment of ACA</td>
<td>2,8%</td>
<td>2,1%</td>
<td>-</td>
<td>4,9%</td>
</tr>
<tr>
<td>Fenestration of ACA (A1 segment)</td>
<td>0,7%</td>
<td>0,7%</td>
<td>-</td>
<td>1,4%</td>
</tr>
<tr>
<td>Absent AcoA</td>
<td></td>
<td></td>
<td></td>
<td>23,8%</td>
</tr>
<tr>
<td>Fenestration of AcoA</td>
<td></td>
<td></td>
<td></td>
<td>2,1%</td>
</tr>
<tr>
<td>Absent PcoA</td>
<td>9,1%</td>
<td>13,3%</td>
<td>23,1%</td>
<td>45,5%</td>
</tr>
<tr>
<td>Double PCoA</td>
<td></td>
<td>0,7%</td>
<td>-</td>
<td>0,7%</td>
</tr>
<tr>
<td>Fetal PCA</td>
<td>4,9%</td>
<td>7,7%</td>
<td>1,4%</td>
<td>14%</td>
</tr>
<tr>
<td>Double PCA</td>
<td></td>
<td>0,7%</td>
<td>-</td>
<td>0,7%</td>
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<tr>
<td>Common origin of SCA and PCA</td>
<td>5,6%</td>
<td>2,1%</td>
<td>-</td>
<td>7,7%</td>
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<tr>
<td>SCA branch of PCA</td>
<td>2,8%</td>
<td>2,1%</td>
<td>-</td>
<td>4,9%</td>
</tr>
<tr>
<td>Absent PICA</td>
<td>15,4%</td>
<td>12,6%</td>
<td>1,4%</td>
<td>29,4%</td>
</tr>
<tr>
<td>Absent AICA</td>
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<td>10,5%</td>
<td>7,7%</td>
<td>37,8%</td>
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<tr>
<td>Absent SCA</td>
<td></td>
<td></td>
<td>0,7%</td>
<td>0,7%</td>
</tr>
<tr>
<td>Double PICA</td>
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<td>-</td>
<td>-</td>
<td>0,7%</td>
</tr>
<tr>
<td>Double AICA</td>
<td>2,1%</td>
<td>3,5%</td>
<td>-</td>
<td>5,6%</td>
</tr>
<tr>
<td>Double SCA</td>
<td>4,2%</td>
<td>4,2%</td>
<td>-</td>
<td>8,4%</td>
</tr>
</tbody>
</table>

Table 2: Comparison of our results with other MRA studies, performed on 1,5T scanner

<table>
<thead>
<tr>
<th>Study</th>
<th>Entire circle</th>
<th>Duplication of AcoA</th>
<th>Absent AcoA</th>
<th>MvCC</th>
<th>Azygos ACA</th>
<th>Hypo/aplasis of A1 segment</th>
<th>Unilateral fetal PCA</th>
<th>Bilateral fetal PCA</th>
<th>Unilateral absence of PcoA</th>
<th>Bilateral absence of PcoA</th>
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</thead>
<tbody>
<tr>
<td>Chen, 2004,</td>
<td>21,30</td>
<td>1,18</td>
<td>11,64</td>
<td>2,96</td>
<td>7,3</td>
<td>9,86</td>
<td>20,71</td>
<td>10,06</td>
<td>26,43</td>
<td>42,80</td>
</tr>
<tr>
<td>Bahadur, 2015</td>
<td>16,6</td>
<td>0,66</td>
<td>11,67</td>
<td>2,0</td>
<td>8,6</td>
<td>9,33</td>
<td>19,33</td>
<td>7,0</td>
<td>19,33</td>
<td>32,66</td>
</tr>
<tr>
<td>Maaly, 2011</td>
<td>46,7</td>
<td>11,6</td>
<td>21,6</td>
<td>0,5</td>
<td>9,4</td>
<td>10,0</td>
<td>37,8</td>
<td>25,0</td>
<td>37,2</td>
<td>3,8</td>
</tr>
<tr>
<td>Our study</td>
<td>28</td>
<td>0</td>
<td>23,8</td>
<td>8,4</td>
<td>0,7</td>
<td>4,9</td>
<td>12,6</td>
<td>1,4</td>
<td>22,4</td>
<td>23,1</td>
</tr>
</tbody>
</table>
Fig. 1: VRT reconstruction image showing presence of median artery of corpus callosum
Fig. 2: VRT reconstruction image showing: a) aplasia of A1 segment of right anterior cerebral artery, b) right fetal posterior cerebral artery, absence of left posterior communicating artery and anterior inferior cerebellar arteries, and early branching of left superior cerebellar artery. Both internal carotid arteries have been removed during post-processing in order of better visualization of present variants.

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Fig. 3: VRT reconstruction image and MIP thin transaxial image showing large ovoid shaped fenestration of A1 segment of right anterior cerebral artery (a). Other present variants in this case include median artery of corpus callosum (b) and absence of both posterior communicating arteries. Both internal carotid arteries have been removed during post-processing in order of better visualization of present variants.

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**Fig. 4:** VRT reconstruction images showing large ovoid shaped fenestration of anterior communicating artery (a), hypoplasia of A1 segment of the left anterior cerebral artery (b) and absence of both posterior communicating arteries and left anterior inferior cerebellar artery. Both internal carotid arteries have been removed during post-processing in order of better visualization of present variants

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![VRT reconstruction images](image1)

**Fig. 5:** VRT reconstruction image and MIP thin transaxial image showing fenestration of anterior communicating artery (a), hypoplastic A1 segment of right anterior cerebral artery (b) and right fetal posterior cerebral artery (c), as well as absence of both anterior inferior cerebellar arteries. Both internal carotid arteries have been removed during post-processing on the first image in order of better visualization of present variants

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Fig. 6: VRT reconstruction image, zoomed in, showing absence of every communicating artery. Common trunk of posterior cerebral and superior cerebellar artery is present on the left side (a). Both internal carotid arteries have been removed during post-processing in order of better visualization of present variants.

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Fig. 7: VRT reconstruction image showing presence of fetal posterior cerebral artery on the right (a). Left posterior communicating artery is not detectable. Both internal carotid arteries have been removed during post-processing in order of better visualization of present variants.

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Fig. 8: VRT reconstruction image showing partial fetal posterior cerebral artery on the right (a), with hypoplastic P1 segment (b). Other present variants are absence of left anterior and posterior cerebellar arteries. Both internal carotid arteries have been removed during post-processing in order of better visualization of present variants

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**Fig. 9:** MIP thin reconstruction coronal plane images showing double superior cerebellar artery with variant origin: a) both from left posterior cerebral artery and b) one from basilar artery and other from right posterior cerebral artery.

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Fig. 10: VRT reconstruction image, zoomed in, showing duplication of anterior inferior cerebellar artery on the left (a), and absence on the right.

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**Fig. 11:** VRT reconstruction images showing communication between vertebral arteries (a), presence of median artery of corpus callosum (b), duplication of superior cerebellar artery on the right (c), with one of them originating from posterior cerebral artery and absence of posterior communicating arteries and both anterior superior cerebellar arteries. Both internal carotid arteries have been removed during post-processing in order of better visualization of present variants

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Conclusion

Our study has shown similar results to other angiographic, but differs from anatomic studies, especially in percentages of communicating arteries’ aplasia, which can be due to limitations of 1.5T MR machine in detecting slow circulation in small caliber vessels. It is important to know limitations of each method we use for evaluation of variations while interpreting results.
References


