Predisposing factors in posterior circulation infarcts: A vascular morphological assessment

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Aims and objectives

The term dolichoectasia is used for dilated, tortuous, and elongated arteries. Dilatation is the most striking feature and therefore it is also called dilatative arteriopathy. Dilatative arteriopathy is most commonly observed in intracranial vertebral and basilar arteries (1-3). Previous studies have observed that risk of ischemic symptom development is higher in cases with basilar artery ectasia, and those with tortuous and elongated basilar arteries develop cranial nevre palsy (4, 5). Elongation and angulation of intracranial arteries cause reduced blood flow as a result of tension and distortion of arterial branches, especially penetrating branches of large arteries. Pontine infarcts at the territory of basilar artery are the best example (6, 7). It has been reported that posterior and anterior circulation infarcts do not differ from each other with regard to risk factors but they have distinct symptomatologies as a result of involvement of different anatomical regions (8). However, the most prominent difference is that arteries of the posterior circulation have a vertical course, while arteries of the anterior circulation have a horizontal course. This difference may explain why more than one infarct focus is observed in posterior circulation infarcts as compared to their anterior counterparts. In clinically important pathologies of posterior circulation timely detection and treatment of at-risk patients will be life-saving. This work will assess the effect of shape, diameter, elongation and deviation criteria of basilar artery (BA), convergence angle and diameter variations of vertebral arteries (VA), and concurrent chronic diseases on posterior circulation infarcts.
Methods and materials

Brain MRI images of 3120 patients who underwent brain and diffusion MRI examination with suspected cerebrovascular accident (CVA) between January 2010 and May 2013 were accessed from our hospital's picture archiving and communication system (PACS) and evaluated, and 418 patients found to have a posterior circulation infarct were selected for analysis. Patients with a cardiac pathology with a possible causal relationship with infarct (e.g. atrial fibrillation, heart failure, aortic or mitral stenosis etc.), and those with carotid, vertebral, or basilar artery stenosis or occlusion detected at digital substraction angiography (DSA), computerized tomography (CT) angiography, magnetic resonance (MR) angiography, or Doppler ultrasonography (USG) or with any infarct zone at the territory of internal carotid artery were excluded from the study. One hundred and eighty-six patients with an isolated posterior circulation infarct positive (PCIP) group and 120 age- and sex-matched control group having no similar posterior circulation infarct (PCIN) (having no anterior and posterior circulation infarcts) were included in the study. Vertebral artery (VA) and basilar artery (BA) diameter, right and left VA angles at the level of bifurcation, and elongation, deviation, and shape of BA were assessed in a total of 306 patients (120 infarct negative control group and 186 infarct positive patient group).

The brain MRI was performed in a routine supine natural position using a 1.5-Tesla scanner (Intera, Gyroscan, Philips Medical Systems, The Netherlands). All images were taken according to a standard protocol using axial T2-weighted turbo spin echo (repetition time/echo time (TR/TE): 4466/100, slice thickness (ST) 5 mm, number of excitation (NEX) 3), coronal and sagittal T2-weighted turbo spin echo (TR/TE: 4800/100, ST: 4 mm, NEX: 3), axial fluid-attenuated inversion recovery (TR/TE: 6000/100, ST: 5 mm, NEX: 3) and axial T1-weighted spin echo sequences (TR/TE: 462/11, ST: 5 mm, NEX: 3) covering the whole brain. Brain MR images were evaluated and measurements were taken by a radiologist who was experienced in neuroradiology. From axial T2-weighted brain MR images the diameter of the intracranial part of the vertebral arteries, right (R) and left (L) VA angles, diameter of BA, basilar bifurcation height (elongation), transverse position of BA (deviation), and shape of BA were evaluated. Shape of basilar artery was classified as normal (N), C, S, and J. Basilar and vertebral artery diameters were measured at their widest point from axial T2-weighted images. Based on the study by Smoker et al. (9, 10), the elongation of the BA is divided into three group as mild (1; within the suprasellar cistern), moderate (2; at level of third ventricle floor), ans severe (3; indenting and elevating the floor of the third ventricle). Also deviation criteria were classified as severe (3; in cerebellopontine angle cistern), moderate (2; lateral to lateral margin of clivus or dorsum sellae), and mild (1; medial to lateral margin of clivus or dorsum sellae).

Based on the work by Voetsch et al. (11), ischemic lesions at the posterior circulation in the patient group were classified according to proximal, middle, and distal infarct zone. Combined effects of all possible risk factors for infarct were assessed with multivariate logistic regression analysis.
Results

A total of 186 PCIP patients having no occlusion or prominent stenosis in basilar and vertebral arteries (mean age 67.9 (SD±11) years, 78 females (41.9%), 108 males (58.1%)) were included. Posterior circulation infarct negative (PCIN) group included 120 cases with a mean age of 68.8 (SD±12.2) years, of whom 63 were female (52.5%) and 57 male (47.5%). No significant difference was noted between both groups with respect to mean age (p=0.749) and sex distribution (p=0.070).

Posterior circulation infarct positive group had a significantly higher rate of chronic diseases including DM, HT, and hypercholesterolemia (p<0.05). The demographic data of PCIN and PCIP groups are given on Table 1.

There were no significant differences between the groups with respect to median R VA diameter, L VA diameter, and R VA angle (p>0.05). Compared to the control group, the patient group had a significantly higher median L VA angle (p=0.018). Clinical characteristics of PCIN and PCIP groups are given in detail on Table 2.

Median basilar artery diameter was 2.75 mm in the PCIN group and 2.8 mm in the PCIP group. No significant difference was present between both groups with regard to median BA diameter (p=0.158).

The ratio of subjects with a N-shaped BA shape was significantly lower (p<0.001) whereas the ratio of those with a J and C-shaped BA was significantly higher (p<0.05) in the PCIP group. The 2 groups were not different with respect to the ratio of subjects with a S-shaped BA (p>0.05).

Classification according to elongation criteria in the PCIP group revealed that the ratio of subjects with a BA elongation of 1 was significantly lower (p<0.001), whereas the ratio of subjects with a BA elongation of 2 or 3 was significantly higher (p<0.001 ve p=0.002) (Table 3a). Classification based on the deviation criteria in the PCIP group showed that the ratio of subjects with a BA transverse location of 1 was significantly lower (p<0.001), while the ratio of subjects with a transverse location of 2 or 3 was significantly higher (p<0.001) (Table 3b).

Combined effects of all possible risk factors for infarct were assessed with multivariate logistic regression analysis. After correction for other risk factors, factors most effective on infarct development were a BA elongation of 2 or 3 followed by a BA transverse location of 2 or 3, an increased L VA angle, and history of HT, DM, and hypercholesterolemia. Odds ratios and 95% confidence intervals of all possible risk factors for infarct are given on Table 4.
In our study the distribution of subjects according to location of infarct zone at the posterior circulation was as follows: middle in 108 (58.1%) patients, proximal in 70 (37.6%) patients, and distal 54 (29%) in patients.

No significant difference existed between the subjects with a proximal infarct location and those with a middle or distal infarct location with respect to chronic disease, BA diameter, shape, elongation, and transverse location, R VA diameter, L VA diameter, R VA angle, and L VA angle (p>0.05).
Fig. 1: Table 1: Demographic characteristics of the study subjects according to infarct negative control group (PCIN) and infarct positive patient group (PCIP). HT: hypertension, DM: diabetes mellitus

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<table>
<thead>
<tr>
<th>Variables</th>
<th>PCIN group (n=120)</th>
<th>PCIP group (n=186)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>BA diameter</td>
<td>2.75 (1.2-4.0)</td>
<td>2.8 (0.9-6.0)</td>
<td>0.158</td>
</tr>
<tr>
<td>BA shape</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>100 (%83.3)</td>
<td>109 (%58.6)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>C</td>
<td>3 (%2.5)</td>
<td>16 (%8.6)</td>
<td>0.031</td>
</tr>
<tr>
<td>J</td>
<td>16 (%13.3)</td>
<td>52 (%28.0)</td>
<td>0.003</td>
</tr>
<tr>
<td>S</td>
<td>1 (%0.8)</td>
<td>9 (%4.8)</td>
<td>0.095</td>
</tr>
<tr>
<td>BA elongation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>“1”</td>
<td>111 (%92.5)</td>
<td>123 (%66.1)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>“2”</td>
<td>9 (%7.5)</td>
<td>49 (%26.3)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>“3”</td>
<td>-</td>
<td>14 (%7.5)</td>
<td>0.002</td>
</tr>
<tr>
<td>BA deviation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>“1”</td>
<td>107 (%89.2)</td>
<td>108 (%58.4)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>“2”</td>
<td>13 (%10.8)</td>
<td>52 (%28.1)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>“3”</td>
<td>-</td>
<td>25 (%13.5)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>R VA diameter</td>
<td>2.0 (0.9-4.5)</td>
<td>1.7 (0.5-5.8)</td>
<td>0.649</td>
</tr>
<tr>
<td>L VA diameter</td>
<td>2.3 (1.0-3.5)</td>
<td>2.3 (0.5-5.1)</td>
<td>0.852</td>
</tr>
<tr>
<td>R VA angle</td>
<td>36.0 (4.0-98.0)</td>
<td>32.0 (6.0-79.0)</td>
<td>0.255</td>
</tr>
<tr>
<td>L VA angle</td>
<td>29.5 (4.0-63.0)</td>
<td>30.5 (5.0-123.0)</td>
<td>0.018</td>
</tr>
</tbody>
</table>

**Fig. 2:** Table 2. Clinical characteristics of the study subjects according to infarct negative control group (PCIN) and infarct positive patient group (PCIP). BA: basilar artery, VA: vertebral artery.

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**Fig. 3:** Table 3 a-b: The ratio of subjects with the BA elongation (right) and BA deviation (left) according to infarct negative control group (PCIN) and infarct positive patient group (PCIP).

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Fig. 4: Table 4. Evaluation of combined effects of possible risk factors that may determine distinguishing the infarct negative control group (PCIN) and infarct positive patient group (PCIP) according to multivariate logistic regression analysis. DM: diabetes mellitus, HT: hypertension, BA: basilar artery, VA: vertebral artery.

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Fig. 5: Table 5. Right and Left VA angles according to infarct negative control group (PCIN) and infarct positive patient group (PCIP).

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Conclusion

Our study demonstrated that factors effective on posterior infarct development were, in descending order, a BA height (elongation) of 2 or 3, a BA transverse location (deviation) of 2 or 3, and an increase in left VA angle. In addition, it was re-established that chronic diseases such as HT, hypercholesterolemia, and DM are the risk factors for posterior circulation infarcts. Evaluation of segments of basilar artery in 186 patients with posterior circulation infarct did not yield any signs of occlusion or significant stenosis in the entire patient group. One patient who has a fenestration in the proximal basilar trunk of the BA is excluded from the study.

The term basilar artery dolichoectasia defines elongation, tortuosity, and dilatation of basilar arteries. Thus, it is also called dilatative arteriopathy. A basilar arterial diameter more than 4.5 cm is accepted as ectasia. Smoker et al. defined dolichoectasia criteria with semi-quantitative assessment with CT (9, 10). No standard and widely accepted dolichoectasia criteria are present with MRI and MRA examinations and some difficulties exist with routine MRI reporting. A cohort study by Ubogu et al. showed that a vertebrobasilar artery diameter more than 4.5 mm, a deviation more than 10 mm, and a length more than 29.5 mm were independent risk factors for transient or permanent posterior circulation deficits (12).

In our study the median basilar artery diameter was 2.8 mm in the infarct positive group and it was not different from the control group. This size is similar to that found in the study by Keyik et al. (13) but smaller than those found by many other studies (12, 14). Elongation and angulation of intracranial arteries cause reduced blood flow as a result of tension and distortion of arterial branches, especially penetrating branches of large arteries. Pontine infarcts at the territory of basilar artery are the best example (6, 7). Blood flow in dilated arteries is in a to-and-fro fashion as a result of reduced antegrade blood flow (15, 16). Reduced antegrade blood flow leads to blood stagnation in the dilated arterial segment and thrombus formation. Luminal thrombosis occludes arterial branches and causes distal embolism (17, 18). These events culminate in persistent or transient brain ischemia.

Our study demonstrated that in the PCIP group a BA elongation or deviation grade of 2 or 3 according to elongation criteria had the strongest effect on infarct development. They were followed by an increased L VA angle. In the PCIP group 154 (82.8%) patients were diagnosed to have a codominance in VA, 5 (2.7%) had a R VA dominance, and 27 (14.5%) had a L VA dominance (Table 5). These results may explain the role of the increased left vertebral angle in infarct development.

Voetsch et al (11) found that the infarct was located in middle perfusion area in 75%, in proximal perfusion area in 30, and in distal perfusion area in 50% of patients. In our study the distribution according to infarct zone by posterior circulation territory was 108 (58.1%)
middle, 70 (37.6%) proximal, and 54 (29%) distal perfusion areas. This variation was attributed to exclusion of patients with BA or VA stenosis-occlusion or cardiac pathology.

Comparison of the groups with respect to shape of basilar artery revealed that the ratio of patients in the PCIP group with a N-shaped BA was significantly lower (p<0.001), while the ratio of those with a J and C-shaped BA was significantly higher (p<0.05). Evaluation of data about elongation, deviation, and shape altogether suggests that dolichoectasia criteria other than diameter had an undeniable effect on infarcts originating from the middle segment of basilar artery.

It has been suggested that dolichoectasia develops as a result of atherosclerotic degeneration of arterial wall, and arterial hypertension is a pathogenetic risk factor acting alone or in combination. Some authors have shown that it is a congenital anomaly with findings of smooth muscle atrophy and defects in internal elastic membrane on histological sections. Its association with dilatation of other cerebral vessels and aortic aneurysm supports the theory of diffuse arterial defect. Hypertension and atheromatous process play an important role for ischemia development in dolichoectatic patients. The relationship between vertebrobasilar artery dolichoectasia and ischemic symptoms is not completely understood. Literature data mostly comes from case reports. Some studies have tried to explain its etiology and mechanism. Infarcts at distal sites have been linked to artery-to-artery embolism, while brain stem and some cerebellar infarcts to atherothrombotic occlusion of basilar artery (19, 20).

In line with previous studies (21), we showed in our study that chronic disorders such as DM, HT, and hypercholesterolemia lead to posterior circulation infarcts.

Acute posterior circulation infarcts are diagnosed more rapidly and earlier with DAG. The examination time is on the order of seconds. It is substantially effective in ischemic stroke despite artefacts and limitations in posterior fossa (22). Routine MRI can detect acute infarcts at 24-48 hours at the earliest, while the corresponding figure associated with DAG ranges between half an hour and 6 hours (23). A retrospective analysis of our study showed that the posterior circulation infarct was detected at chronic period in 120 (64.9%) patients, at acute period in 65 (35.1%) patients, and at subacute period in 8 (4.3%) patients.

In posterior circulation ischemia patients usually present with primary stroke without a previous history of TIA (8). The affected area of posterior circulation is brainstem in 60%, cerebellum in 50%; the infarcts are of basilar and/or vertebral artery origin in more than 50% of patients (24). The most common abnormality is basilar artery occlusion or stenosis. The causes of posterior circulation infarcts are small vessel diseases in 15%, cardiac embolism in 13%, and multifactorial (arterial stenosis and/or occlusion, lacunar lesions, and cardiac embolism) in 13% while a potential cause cannot be detected in 10% (25).
Tortuous and elongated arteries particularly cause medullary and pontine compression and distortion. In our study, 10 patients had vertigo, 2 had tinnitus, and 2 had fascial paralysis secondary to vascular compression as additional symptoms.

In posterior circulation infarcts BA deviation and elongation grades 2 or 3 and a widened L VA angle were significantly different from the control group. However, there were no significant differences between the groups with respect to "diameter" measurements that have been suggested to be the most important criteria for dolichoectasia. Reporting marked morphological variations in BA and VA detected at routine brain MR examinations will aid in selection of at-risk patients with chronic diseases to apply timely treatment options.
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References


