Evaluation of carotid arteries with colour doppler sonography in patients with leukoaraiosis at cranial MRI

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Purpose

Leukoaraiosis is characterized by white matter hyperintense lesions (WMHRL) on T2-weighted magnetic resonance imaging (MRI) as the frequent finding of brain aging (1). WMHRL indicate a strong relationship between age, arterial hypertension, diabetes and other vascular risk factors(2,3). However, the genetic predisposition is thought to be closely related in terms of leukoaraiosis. Histological underlying causes are decreased myelin, axonal loss and astrocytic gliyozis (4,5). Population-based epidemiological studies, suggests the link between cognitive performance, psychomotor deceleration and balance disorder of elderly people who have WMHRL. Healthy people of advanced age, put forward a clear association between decreased mental performance and WMHRL (6). The presence of WMHRL may be a risk factor for stroke(7,8).

Pathogenesis of WMHRL is not fully cleared. Small vessel disease appear to be the main etiological factor of WMHRL(9), also disruption of the blood brain barrier may have effect on the process (10). However, close association between the presence of atherosclerosis in the large arteries, the deep white matter infarcts and leukoaraiosis are well establised (8,11-15). Any large arterial disease wich cause white matter ischemia could be an indirect factor in the development of leukoaraiosis. Arterial tromboembolization leading to irreversible hemodynamic changes may play a secondary role in this mechanism.

In our study, in patients with patchy hyperintensities which tended to coalesce, compatible with leukoaraiosis on MRI, intima-media thickness, types of atherosclerotic plaque and stenosis of the internal carotid arteries were measured. We aim to analyse prospectively the relationship between the Leukoarios and extracerebral arterial system, using a control group; to investigate the pathophysiology and risk factors of leukoaraiosis.

Methods and Materials

Leukoaraiosis is characterized by white matter hyperintense lesions (WMHRL) on T2-weighted magnetic resonance imaging (MRI) as the frequent finding of brain aging (1). WMHRL indicate a strong relationship between age, arterial hypertension, diabetes and other vascular risk factors(2,3). However, the genetic predisposition is thought to be closely related in terms of leukoaraiosis. Histological underlying causes are decreased myelin, axonal loss and astrocytic gliyozis (4,5). Population-based epidemiological studies, suggests the link between cognitive performance, psychomotor deceleration and balance disorder of elderly people who have WMHRL. Healthy people of advanced age, put forward a clear association between decreased mental performance and WMHRL (6). The presence of WMHRL may be a risk factor for stroke(7,8).

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In our study, in patients with patchy hyperintensities which tended to coalesce, compatible with leukoaraiosis on MRI, intima-media thickness, types of atherosclerotic plaque and stenosis of the internal carotid arteries were measured. We aim to analyse prospectively the relationship between the Leukoarios and extracerebral arterial system, using a control group; to investigate the pathophysiology and risk factors of leukoaraiosis.

In this study between 2010-2011, 32 patients (Group A) who applied to Medeniyet University Goztepe Training and Research Hospital Radiology Department, between the ages of 55-90 patchy hyperintensities that tend to coalesce on T2-weighted MR sequences compatible with periventricular leukoaraiosis, (figure 14) were examined by color Doppler of the carotid arteries. Toshiba Medical Systems Corp. device Aplio 7.5 MHz linear probe was used. The control group (Group B) consist of 36 patients was in same age range as the Group A.

Examinations were performed in supine position, as head was slightly extended position. On gray scale examination bilateral carotid arteries were displayed. Intima-media thickness was measured. The ostium of ICA and ECA displayed. Characteristics of the existing plaques were classified on cervical ICA segment. With pulse wave, color wave and spectral doppler presence of stenosis were investigated in carotid arteries. Up to 50% Stenosis degrees are classified using NASCET and ACAS criteria as a method of directly visual grading system. More than 50% stenotic lesions were assessed by peak systolic velocity.

Existing plaques in carotid arteries were characterized by ultrasonography (Bock-Lusby Classification). Type 1 plaque is characterised by hypoechoic plaques, Type 2 plaques are more hypoechoic component, but also containing echogenic component, Type 3 plaques have predominant echogenic component of the more calcified ones are characterized as Type 4.

Degree of stenosis in the carotid artery were identified in 4 categories: Group 1 patients had no stenosis, group 2, 3 and 4, carotid arteries have % 0 - 29%, 30-60% and 60 - 99% stenosis respectively. Occlusions were not included in the study. As a complementary factor, atherosclerotic irregularities, and the mean intima-media thickness on carotid artery were graded. No apparent irregularity in the atherosclerotic intima-media wall characterized as (-), 0.8mm-1mm wall thickness and atherosclerotic irregularity cases graded as (+), and atherosclerotic irregularity with a thickness more than 1mm were categorized as (+ +). Plaques detected in the carotid arteries classified into 4 categories according to their gray-scale ultrasound examination based on Bock-Lusby Classification. Statistical data were analyzed by Mann-Whitney U test, Fisher's exact chi-square test, Pearson's test.
Results

A total of 32 Leukoaraiosis patients between the ages of 55-90, 11 males, 21 females, and in the same age range 36 people who had no leukoaraiosis a total of 15 men and 21 women were examined with color Doppler ultrasound examination for atherosclerotic intima-media thickness, degree of stenosis and plaque types and statistically analyzed using Mann Whitney U test, Fisher's exact chi-square test, the data were Pearson's test.

<table>
<thead>
<tr>
<th>AGE</th>
<th>N</th>
<th>Mean ± std. deviation</th>
<th>Median</th>
<th>Minimum</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients</td>
<td>32</td>
<td>77,69 ± 7,79</td>
<td>81,00</td>
<td>60</td>
<td>88</td>
</tr>
<tr>
<td>Control</td>
<td>36</td>
<td>68,81 ± 7,05</td>
<td>68,00</td>
<td>58</td>
<td>85</td>
</tr>
<tr>
<td>Total</td>
<td>68</td>
<td>72,99 ± 8,60</td>
<td>73,50</td>
<td>58</td>
<td>88</td>
</tr>
</tbody>
</table>

There are statistically significant differences between the groups in terms of age (p = 0.001 *). Leukoaraiosis patients are older than controls. Between the two groups Intima-media thickness values shows statistically significant difference (p = 0.001 *). Intima-media thickness in patient group is higher than in the control group.

<table>
<thead>
<tr>
<th>Intima media thickness</th>
<th>N</th>
<th>Mean ± Std. deviation</th>
<th>Median</th>
<th>Minimum</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient</td>
<td>32</td>
<td>1,08 ± 0,11</td>
<td>1,1</td>
<td>0,8</td>
<td>1,3</td>
</tr>
<tr>
<td>Control</td>
<td>36</td>
<td>0,88 ± 0,09</td>
<td>0,8</td>
<td>0,8</td>
<td>1,1</td>
</tr>
<tr>
<td>Total</td>
<td>68</td>
<td>0,98 ± 0,14</td>
<td>1,0</td>
<td>0,8</td>
<td>1,3</td>
</tr>
</tbody>
</table>

When grouped according to the stenosis values, there is statistically significant difference between the control group, patients (leukoaraiosis +) (p = 0.001 *).

<table>
<thead>
<tr>
<th>Group</th>
<th>Patient</th>
<th>Control</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>2</td>
<td>26</td>
<td>28</td>
</tr>
<tr>
<td></td>
<td>(%7,1)</td>
<td>(%92,9)</td>
<td></td>
</tr>
<tr>
<td>Group 2</td>
<td>3</td>
<td>5</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>(%37,5)</td>
<td>(%62,5)</td>
<td></td>
</tr>
<tr>
<td>Group 3</td>
<td>18</td>
<td>5</td>
<td>23</td>
</tr>
</tbody>
</table>
There are differences depending on the type of plaque when leukoaraiosis patients compared to controls (p = 0.001 *). The incidence of Type 1 and Type 2 plaque detection was significantly more frequent in the control group.

<table>
<thead>
<tr>
<th></th>
<th>Leukoaraiosis (-)</th>
<th>Leukoaraiosis (+)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Typ 1-2 plaque</td>
<td>1</td>
<td>26</td>
<td>27</td>
</tr>
<tr>
<td></td>
<td>(%3,7)</td>
<td>(%96,3)</td>
<td></td>
</tr>
<tr>
<td>Typ 3-4 plaque</td>
<td>6</td>
<td>5</td>
<td>11</td>
</tr>
<tr>
<td></td>
<td>(%55,5)</td>
<td>(%45,5)</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>7</td>
<td>31</td>
<td>38</td>
</tr>
</tbody>
</table>

Images for this section:
Fig. 1: Mean age

Fig. 2: Individual value plot of age and groups
Fig. 3: Intima media thickness
Conclusion

Pathophysiological processes in the brain has been the subject of many research. The aim of our study, was to assess the contribution of pathological changes in the carotid arteries in advanced age patients with patchy periventricular hyperintensities on MR compatible with leukoaraiosis.

Failure of the vascular system is believed to cause leukoaraiosis in the white matter of the brain (2), but the pathogenesis is not fully cleared. Recent studies show that brain vascular structures significant differences between, leukoaraiosis observed patients and healthy people. leukoaraiosis patients, are more prone to ischemic and hemorrhagic stroke. Also patients with leukoaraiosis show weakness, depression, fecal incontinence, mental disorders and an increased incidence of dementia (5,6,7,8,16). For this reason it's important to determine the risk factors associated with leukoaraiosis.

Cerebral blood flow is thought to diminish by Extracranial carotid artery stenosis. This reduction in blood flow may be a factor in the development of leukoaraiosis (18). Our data suggest that the atherosclerotic stenosis and arterial system disorders in carotid vessels were found to be statistically more frequent in leukoaraiosis of patients compared to the control group. Diffuse white matter demyelination associated with atherosclerosis is detected in patients with leukoaraiosis. Brain hyperintensities consistent with periventricular leukoaraiosis detected in advanced stage of atherosclerotic changes. Many studies support leukoaraiosis is associated with atherosclerotic changes in cardiac, peripheral arteries and carotid arteries.

According to the results in our study the presence of leukoaraiosis there are atherosclerotic carotid artery intima-media layer thickening and stenosis. These results suggest the hypothesis (Fazekes et al) that the development of the leukoaraiosis is secondary to ischemic process (21,22,23). This hypothesis assume that, Leukoaraiosis occur as a result of reactive gliosis secondary to chronic ischemia, induced by occlusion of small arterioles supplying the white matter.

Correlation between the Carotid artery stenosis, atherosclerotic intima-media thickness and plaque types in patients with leukoaraiosis were examined. Leukoaraiosis is more commonly observed in elderly. This also suggests that a significant correlation between age and leukoaraiosis. Previous studies also support this data; age-related brain atrophy and Wallerian degeneration contribute to the process of developing leukoaraiosis (23,24).

Several studies are available demonstrating the connection between leukoaraiosis and cerebrovascular diseases (hypertension, diabetes, dyslipidemia, smoking) (4,25,26,27). Our data suggest a statistically significant correlation between leukoaraiosis and the age distribution, atherosclerosis, carotid artery stenosis and plaque types (Figure 17). Previous studies submit data that carotid arteries with fatty plaques (Type 1, Type 2) contribute to leukoaraiosis, the data in our study also supports this.
In summary, recent studies reveal structural changes in the white matter the parenchyma of the aging brain. Addition to T2 signal changes on MR imaging, further investigation is needed in the functional sense. However, we believe that these factors may be linked to each other. Greater number of participants will contribute to a clearer understanding of the pathophysiology of leukoaraiosis.

References


Personal Information