Analysis between carotid atherosclerosis and related risk factors after ultrasonic testing.

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Abstract

Objective: To evaluate the relationship between carotid atherosclerosis and related risk factors.
Methods: Patients who were participant in neurology department of our hospital were selected for study. According to the ultrasound test results, 60 patients with carotid atherosclerosis were divided as the observation group, another 60 patients with normal carotid artery as the control group. All patients were subjected to following detection: bilirubin, Homocysteine (Hcy), C-reactive protein, Uric Acid (UA), blood lipids, blood pressure level. The test results were subjected to univariate and multivariate comparative analysis.
Results: After univariate comparative analysis, the history of diabetes, hypertension, alcohol consumption, smoking prevalence in the observation group was significantly higher, the difference was significant difference (P<0.005). After univariate comparative analysis, two DB, TB, Hcy, UA, High-Density Lipoprotein Cholesterol (HDL-C), Low-Density Lipoprotein Cholesterol (LDL-C), Triglycerides (TG), Diastolic Blood Pressure (DBP), Systolic Blood Pressure (SBP), BMI comparative analysis, the difference was significant difference (P<0.05). After multiple regression analysis, according to the size of the contribution rate, from low to high was Hcy, history of diabetes, TG, LDL-C, smoking, hypertension, above risk factors and carotid atherosclerosis was positively correlated the HDL-C is a protective factor.
Conclusion: Carotid atherosclerosis is closely related to traditional risk factors for cardiovascular disease, the new risk factors can be defined as the assessment of atherosclerosis effective index.

Keywords: Carotid atherosclerosis, Risk factors, Ultrasonic.
patients with carotid atherosclerosis were enrolled into the observation group, in which there were 36 males and 24 females with an average age of (66.5 ± 6.28 y old), and the other 60 patients whose carotid arteries were normal were enrolled as the control group, in which there were 33 males and 27 females with an average age of (65.2 ± 6.45 y old). Based on the consultation with the patients and the diagnosis, patients with the recent administration of folic acid and vitamin B, relative diseases such as severe hepatic or renal dysfunctions, malignant tumor, blood system-associated diseases or gout were excluded in this study. All patients underwent the ultrasonic detection of carotid atherosclerosis. In comparison of gender and age, we found that there is no statistically significant differences among all patients (p>0.05) (Table 1), suggesting that the general material of patients was comparable.

<table>
<thead>
<tr>
<th>Groups</th>
<th>n</th>
<th>Male/female (Cases)</th>
<th>Age (y)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Observed group</td>
<td>60</td>
<td>36/24</td>
<td>66.5 ± 6.28</td>
</tr>
<tr>
<td>Control group</td>
<td>60</td>
<td>33/27</td>
<td>65.2 ± 6.45</td>
</tr>
<tr>
<td>T value</td>
<td>0.3069</td>
<td></td>
<td>1.1186</td>
</tr>
<tr>
<td>P value</td>
<td>0.5796</td>
<td></td>
<td>0.2656</td>
</tr>
</tbody>
</table>

### Methods

**Ultrasonic examination**

Ultrasonic examination was carried out using the Philips IU-22 color ultrasonography scanner with a probe, in which the frequency was set as 7.5 to 10 MHz. The probe was moved from the proximal end of carotid artery in bottom-top orientation, and the real-time two-dimensional images of bilateral common carotid arteries, bifurcation of internal and external carotid arteries and the extracranial segment of internal carotid artery were used to perform segmental detection for quantitative measurement of the thickness between intima and media of vascular wall and the plaque, and calculation of stenosis rate of vascular lumen. Judgment criteria were stipulated in accordance with the definitions of target organ injuries in Guidelines for prophylaxis of hypertension in China, i.e. atherosclerosis refers to the thickness between the intima and media of vascular lumen in any segment of carotid artery system is higher than 0.9, or the formation of plaque (the thickness between the intima and media of vascular lumen in any segment of carotid artery system is higher than 1.3), or stenosis in lumen. Stenosis of vascular lumen=\(\frac{\text{Diameter of lumen in original vessel-Diameter of lumen in residual vessel}}{\text{Diameter of lumen in original vessel}}\times 100\%\), in which measurement ≥ 70% was for severe stenosis, ≥ 50% for moderate stenosis and <50% for mild stenosis.

### Assay of biochemical indicators

Before blood collection, 120 patients were required to withdraw from the alcohol for 24 h, and the venous blood was collected in the morning after patients were fasted for 12 h for detection of Direct Bilirubin (DB), Total Bilirubin (TB), Homocysteine (Hcy), Uric Acid (UA), High Density Lipoprotein Cholesterol (HDL-C), Low Density Lipoprotein Cholesterol (LDL-C) and Triglyceride (TG). All biochemical indicators were measured using the Hitachi-7060 Automatic Biochemical Analyzer, in which TG and UA levels were detected by enzymatic method, HDL-C and LDL-C by immunoturbidimetry (kits provided by Shanghai Yulan Biotechnology Co., Ltd), Hcy by ADVIA centaur automatic immunochemiluminescence system and the corollary reagent that were provided by Bayer (Germany), bilirubin by end-point method of diazonium (kits provided by Shanghai Kehua Dongling Biotechnology Co., Ltd.).

### Statistical analysis

The single factor and multiple factors leading to the risk of carotid atherosclerosis were identified through comparison and analysis. SPSS 14.0 was adopted for statistics. Measurement data were presented as mean ± Standard Deviation (SD), and t test was performed for comparison between groups. Chi-square test was carried out for the comparison of enumeration data, and ranked sum test (Wilcoxon test) for ranked data. p<0.05 suggested that the difference had statistical significance.

### Results

**Comparison and analysis of the enumeration data of single factor for carotid atherosclerosis of patients in the two groups**

Through the single-factor comparison and analysis, we found that the incidence rates of history of diabetes mellitus, hypertension, alcohol intake and smoking were significantly higher than those in the control group, and the differences between the two groups had statistical significance (p<0.005; Table 2).

<table>
<thead>
<tr>
<th>Factors</th>
<th>Observed group (n=60)</th>
<th>Control group (n=60)</th>
<th>(\chi^2) value</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes history (cases)</td>
<td>35 (58.33)</td>
<td>1525.0</td>
<td>13.7143</td>
<td>0.0002</td>
</tr>
<tr>
<td>Hypertension (cases)</td>
<td>42 (70.0)</td>
<td>2135.0</td>
<td>14.7368</td>
<td>0.0001</td>
</tr>
<tr>
<td>Alcoholism (cases)</td>
<td>38 (63.33)</td>
<td>17 (28.33)</td>
<td>14.8028</td>
<td>0.0001</td>
</tr>
</tbody>
</table>
Discussion

As a systemic diffusion disease, atherosclerosis is characterized with the pathological changes of formation of atherosclerotic plaque that is a kind of hyperplastic, degenerative and non-inflammatory lesions in arteries; those lesions refer that after the damages to the intima in an early stage, plaque will be formed through the continuous accumulation of lipid in blood followed by sclerosis and local stenosis, which results in the blockage of vessels and affect the vascular patency, thereby leading to the ischemia and dysfunction of relevant organs [4,5]. The onset of atheromatous plaque may be from the youth of patients, and without any obvious clinical symptoms in the following decades. With the accumulation of the plaque, several organs, including kidney, brain and heart, will form atheromatous plaque. Thus, detecting or monitoring the progression of atherosclerosis and identifying the relevant risk factors of atherosclerosis in asymptotic phase are of great significance for prophylaxis and treatment of cerebrovascular and cardiovascular diseases [6,7].

In this study, a total of 120 patients who went for outpatient service or were admitted to the department of neurology of this hospital between February 2013 and June 2014 were enrolled. The study showed that atherosclerotic lesions are categorized as a kind of very complicated, multifactorial diseases, and metabolic disorder, hyperglycemia and hypertension are considered as the major risk factors followed by obesity and smoking. However, new risk factors have also been discovered; for example, Hcy is correlated with the carotid atherosclerosis. The results of this study are similar to those of clinical studies [8,9]. Multiple studies showed that metabolic disorder, hyperglycemia, hypertension, obesity, smoking and Hcy as the risk factors are correlated with the carotid atherosclerosis.

Some unhealthy life styles are also one of the key risk factors contributing to carotid atherosclerosis, and the correlations of the onset of atherosclerosis with hypertension, dyslipidemia, obesity and smoking have been ascertained [10,11]. In serum, TG exists in the very low density chylomicron and the lipoprotein containing TG that not only can cause the onset of atherosclerosis with hypertension, dyslipidemia and toxicity to vascular endothelial cells. In addition, changes in function and damage of endothelial cells also play an important role in atherosclerosis pathogenesis.

Hcy in blood is also a risk factor of atherosclerosis. Ever since some scholars reported the correlation between Hcy in blood and atherosclerosis in youth, more and more studies have confirmed that the continuous increased level of Hcy in blood may contribute to dyslipidemia, hypertension and damages to vascular wall, thereby increasing the incidence of cardiovascular and cerebrovascular diseases [12-14]. Among the patients with carotid atherosclerosis, the level of Hcy in blood is significantly higher than that in the patients with

### Table 3. Single factor analysis of atherosclerosis between the two groups (x ± s).

<table>
<thead>
<tr>
<th>Factors</th>
<th>Observed group (n=60)</th>
<th>Control group (n=60)</th>
<th>T value</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking (cases)</td>
<td>43 (71.67)</td>
<td>19 (31.67)</td>
<td>19.2214</td>
<td>0.0000</td>
</tr>
<tr>
<td>Hypertension history</td>
<td>0.615</td>
<td>0.013</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Table 4. Multiple factor analysis of atherosclerosis between the two groups.

<table>
<thead>
<tr>
<th>Factors</th>
<th>Standard β</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>1.595</td>
<td></td>
</tr>
<tr>
<td>HDL-C (nmol/L)</td>
<td>-0.465</td>
<td>0.016</td>
</tr>
<tr>
<td>Hcy (μmol/L)</td>
<td>0.032</td>
<td>0.005</td>
</tr>
<tr>
<td>Diabetes history</td>
<td>0.118</td>
<td>0.043</td>
</tr>
<tr>
<td>TG (mmol/L)</td>
<td>0.178</td>
<td>0.004</td>
</tr>
<tr>
<td>LDL-C (nmol/L)</td>
<td>0.259</td>
<td>0.006</td>
</tr>
<tr>
<td>Smoking</td>
<td>0.342</td>
<td>0.035</td>
</tr>
</tbody>
</table>

### Multivariate regression analysis of the related risk factors of carotid atherosclerosis

Through multivariate regression analysis, Hcy, history of diabetes mellitus, TG, LDL-C, history of smoking and history of hypertension were identified as the risk factors, which were in positive correlation with the onset of carotid atherosclerosis.

Among these factors, the contribution rate of Hcy was the lowest, followed by history of diabetes mellitus, TG, LDL-C, history of smoking and history of hypertension. However, HDL-C was identified as the protective factor (Table 4).
normal carotid arteries. Hcy in blood may cause atherosclerosis through the following mechanism: a high level of Hcy in blood can facilitate the generation of oxygen radicals, which then increase the damages to vascular endothelium; those damages result in the gradual hyperplasia in aorta smooth muscle, and accelerate the formation of foamy cells and cholesterol oxidation in LDL, which will thicken vascular wall; thickened vascular wall can promote the blood coagulation through activating the platelet aggregation and affecting the functions of blood coagulation factor, thereby decreasing the fibrinolytic activity. In different forms of combination, these factors can change and influence the normal structure and functions of arteries, thus causing atherosclerosis under the involvement of different factors.

High UA is one of the key risk factors of atherosclerosis. UA at a high level can induce some disorders of blood lipid, activate the platelet, promote the release of vessel-related active substance, and damage the vascular endothelium, thus accelerating the lipid accumulation. Besides, the crystalized urate accumulates on vascular wall, which can directly damage the vessel and cause atherosclerosis [15-17].

Research has shown that the capability of bilirubin to capture the oxygen radicals can prevent the oxidative modification of LDL, which can decrease chemotactic and cytotoxic effect and the influence of oxidative LDL on plasmiraogen, thus reducing the incidence rate of atherosclerosis. In addition, it can increase the negative feedback on activity of hemeoxygenase, thereby ameliorating the damages of metabolite on the cells, and protect the cells through inhibiting the activity of protein kinase C. Research has also shown that among the patients with atherosclerosis, the bilirubin level was significantly lower than that in those with normal carotid arteries, suggesting that bilirubin in a low level is important to the occurrence of atherosclerosis [18-20].

In conclusion, carotid atherosclerosis is correlated with multiple factors. Despite of those conventional risk factors (smoking, diabetes mellitus, dyslipidemia and hypertension) that are still focused on nowadays, some new risk factors (high-level UA and Hcy) also requires more attention. All these factors are correlated with the occurrence of carotid atherosclerosis; thus, we should take comprehensive intervention measures in an early stage to reduce the effect of these risk factors, thus decreasing the occurrence of carotid atherosclerosis.

References


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