Effects of atorvastatin calcium, amlodipine, in combined with nursing intervention on the carotid atherosclerosis in patients with primary hypertension

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1. Introduction

Hypertension is a disease mainly with vasculopathy involved, and is closely associated with atherosclerosis (AS). Long-term elevation of the blood pressure will cause the sclerosis, thickness, and stiffness of the arterial vascular wall, reduce the compliance, induce severe heart, brain, and kidney diseases, and damage the health[1]. With the quickening of the life rhythm, the hypertension patients are increasing day by day with more and more young individuals involved due to the living habits and high fat diets, and the morbidity is sharply increasing, which has been a severe worldwide public health issue and is an important risk factor for developing cervical AS[2]. The cervical IMT can specifically reflect the early AS and sclerosis degree, and is an independent risk factor for predicting...
the occurrence of cardiovascular and cerebrovascular events\cite{3}. The treatment for this disease is to reduce the blood pressure, and more importantly is to decrease the target organ damage due to hypertension in order to reduce the adverse cardiovascular events, improve the prognosis, and enhance the living qualities\cite{4}. The study is aimed to explore the effects of atorvastatin calcium, amiodipine, in combined with nursing intervention on the carotid atherosclerosis in patients with primary hypertension.

2. Materials and methods

2.1. General materials

A total of 70 patients with primary hypertension merged with carotid atherosclerosis who were admitted in our hospital from December, 2014 to December, 2015 were included in the study, among which 41 were male, and 29 were female; aged from 63 to 74 years old, with an average age of (64.5±5.6) years old. The patients were randomized into the observation group and the control group with 35 cases in each group. The comparison of the general materials between the two groups was not statistically significant ($P>0.05$).

2.2. Inclusion and exclusion criteria

All the patients were in accordance with the related diagnostic criteria of primary hypertension merged with carotid atherosclerosis\cite{5}. The cervical IMT was 0.9 mm confirmed by the color Doppler ultrasound. Those who had secondary hypertension, diabetes, coronary heart disease, hyperthyroidism, COPD, severe liver and renal dysfunction, severe anemia, and malignant tumors were excluded from the study.

2.3. Methods

The patients in the two groups were orally administered with atorvastatin calcium (Pfizer Pharmaceuticals Limited, Approval No. J20070061), 20 mg/time, 1 time/night, and amiodipine (Suzhou Dawnrays Pharmaceuticals Limited, Approval No. H20020390), 5 mg/time, and 1 time/d. For patients whose blood pressure not reducing to 140/90 mmHg after 2 weeks, the dosage was increased to 10 mg/time. Six-month treatment was regarded as one course. On this basis, the patients in the control group were given the routine nursing, while the patients in the observation group were given the comprehensive nursing interventions, i.e. on the basis of general nursing, given diet nursing (avoiding alcohol and cigarette, light diet, easy digestion, rich in vitamins, low-salt, low-fat, and low-sterol), medication nursing (controlling the dosage, slowly reducing the blood pressure, long-term sticking to the medication principles, abiding by the doctors’ advice for regular medications), mental nursing (communicating with the patients to know the disease change, patiently answering the questions, eliminating the nervous emotions, and actively cooperating with the treatment), and health education (performing the health education periodically to let the patients know the occurrence and development of the disease, the complications, and attention to the medications, work and rest, appropriate exercise, and controlling the weight, and altering the bad living habits). The levels of blood pressure, TG, TC, LDL-C, and HDL-C before and after treatment in the two groups were detected. The color ultrasonic diagnostic apparatus was used to measure IMT and plaque number.

2.4. Statistical analysis

SPSS 13.0 software was used for the statistical analysis. The measurement data were expressed as mean ± SD, and $t$ test was used. Chi-square test was used for the enumeration data. $P<0.05$ was regarded as statistically significant.

3. Results

3.1. Comparison of the blood pressure before and after treatment between the two groups

After treatment, the reduced degree of SBP and DBP in the observation group was significantly superior to that in the control group ($P<0.05$) (Table 1).

3.2. Comparison of the serum lipid before and after treatment between the two groups

After treatment, the reduced degree of TC and LDL-C in the observation group was significantly superior to that in the control group ($P<0.05$), while TG and HDL-C levels were not significantly different from those before treatment, and the comparison between the two groups was not statistically significant ($P>0.05$) (Table 2).

3.3. Comparison of the cervical IMT and plaque number before and after treatment between the two groups

After treatment, the cervical IMT and plaque number in the observation group were significantly lower than those in the control group ($P<0.05$) (Table 3).

Table 1

<table>
<thead>
<tr>
<th>Groups</th>
<th>$n$</th>
<th>SBP Before treatment</th>
<th>SBP After treatment</th>
<th>DBP Before treatment</th>
<th>DBP After treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Observation</td>
<td>35</td>
<td>171.3±10.54</td>
<td>126.7±11.37*</td>
<td>98.71±9.45</td>
<td>76.82±8.45*</td>
</tr>
<tr>
<td>Control</td>
<td>35</td>
<td>168.9±12.55</td>
<td>139.8±13.45*</td>
<td>97.56±9.68</td>
<td>88.36±10.41*</td>
</tr>
</tbody>
</table>

* $P<0.05$, when compared with before treatment; # $P<0.05$, when compared with the control group.
extension of the exposure time of lipoprotein with the vascular wall, an important factor for developing AS. Hypertension can cause the occurrence of cerebrovascular events, and the development of AS is closely associated with hypertension, and is an important risk factor for developing coronary heart disease, heart failure, and cerebral stroke[6]. The elevation of SBP will increase the blood flow rate, bring a certain impact damage on the vascular wall, trigger inflammation reaction and lipid infiltration, thicken the cervical IMT, increase the vascular hardness, and reduce the compliance, resulting in arterial endothelial cell damage, and promoting the occurrence and development of AS; therefore, it is deemed that the blood pressure instability and high SBP can promote the vascular endothelial cell damage[7,8]. Researches demonstrate that the vascular endothelial dysfunction is closely associated with hypertension, and is an important factor for developing vascular endothelial injury, which can promote the development of AS, thicken the cervical IMT, and cause coronary artery and peripheral vascular diseases[9].

The formation of AS is a gradual process, and hypertension is its main independent risk factor. The long-time elevation of blood pressure will damage the normal vascular endothelium, and destroy the barrier function, resulting in the detachment of the damaged endothelial cells, exposure to the subintimal tissues, and increased blood platelet adhesion and aggregation, thus the mural thrombus is formed. Moreover, the blood platelet can release various growth factors to stimulate the proliferation of fibroblasts and smooth muscle cells to form the connective tissues, finally, the plaque is formed[10,11]. It is reported that every a standard deviation of cerebral IMT is increased, the occurrence rate of cerebral stroke and myocardial infarction is increased by 1.36 times; therefore, the anti-hypertension treatment to alleviate the carotid atherosclerosis is one of the key factors to prevent the occurrence of cardiovascular and cerebrovascular events[12].

The evidence-based medicine shows that dyslipidemia is another important factor for developing AS. Hypertension can cause the extension of the exposure time of lipoprotein with the vascular wall, which is closely associated with the reduced vascular endothelium dependent vasodilation performance, and the increased endothelial permeability and LDL penetrability[13]. The clinical researches verify that the clinical effect of statins is far exceeding the blood lipid regulating role. Atorvastatin calcium has a strong lipid-lowering effect through inhibiting the synthesis of endogenous cholesterol, promoting the liver to uptake LDL-C and VLDL in the blood circulation, increasing HDL-C level, and decreasing LDL-C level in the blood to reduce the plaque surface tension and shrink the lipid nucleus inside the plaque in order to improve the vascular endothelial cell function and stabilize the plaque fibrous cap to promote its fading[14,15]. It is reported that[16] atorvastatin calcium can significantly improve LDL-C and TG levels, reduce the lipid nucleus size, and stabilize or reverse the plaque, with a pleiotropia effect, showing that for patients with hypertension, the anti-hypertension in combined with lipid-lowering treatment can significantly improve the vascular endothelial function and prevent the occurrence of AS. Amlodipine can block the inflow of transmembrane ions and voltage dependent L type channel, and weaken the vascular smooth muscle contraction to slowly reduce the blood pressure. Amlodipine can also protect the vascular endothelial cells, maintain the vascular endothelial function, and inhibit the proliferation of smooth muscle cells in order to slow down the thickening of cervical IMT and effective restrain the development of AS[17].

In the study, in the observation group, atorvastatin calcium and amlodipine, in combined with the comprehensive nursing interventions could significantly enhance the efficacy in patients with primary hypertension merged with carotid atherosclerosis[18]. The results in the study showed that after treatment, the reduced degree of SBP and DBP in the observation group was significantly superior to that in the control group (P<0.05); after treatment, the reduced degree of TC and LDL-C in the observation group was significantly superior to that in the control group (P<0.05), while TG and HDL-C levels were not significantly different from those before treatment, and the comparison between the two groups was not statistically significant (P>0.05); after treatment, IMT and plaque number in the

### Table 2
Comparison of the serum lipid before and after treatment between the two groups (mmol/L).

<table>
<thead>
<tr>
<th>Groups</th>
<th>n</th>
<th>Before treatment</th>
<th>After treatment</th>
<th>Before treatment</th>
<th>After treatment</th>
<th>Before treatment</th>
<th>After treatment</th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>TC</td>
<td>LDL-C</td>
<td>HDL-C</td>
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<td>Before treatment</td>
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<td>After treatment</td>
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<td>Before treatment</td>
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<td>After treatment</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Observation</td>
<td>35</td>
<td>5.82±1.33</td>
<td>4.12±0.32</td>
<td>3.55±0.36</td>
<td>2.35±0.42</td>
<td>1.43±0.47</td>
<td>1.35±0.71</td>
</tr>
<tr>
<td>Control</td>
<td>35</td>
<td>5.91±1.25</td>
<td>4.84±0.57</td>
<td>3.48±0.73</td>
<td>2.93±0.36</td>
<td>1.42±0.36</td>
<td>1.34±0.82</td>
</tr>
</tbody>
</table>

*P<0.05, when compared with the control group; #P>0.05.

### Table 3
Comparison of the cervical IMT and plaque number before and after treatment between the two groups.

<table>
<thead>
<tr>
<th>Groups</th>
<th>n</th>
<th>IMT (mm)</th>
<th>Plaque number (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Before treatment</td>
<td>After treatment</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Observation</td>
<td>35</td>
<td>1.45±0.31</td>
<td>1.10±0.31</td>
</tr>
<tr>
<td>Control</td>
<td>35</td>
<td>1.43±0.26</td>
<td>1.21±0.24</td>
</tr>
</tbody>
</table>

*P<0.05, when compared with the control group.

### 4. Discussion

Hypertension is a common cardiovascular disease, with no obvious clinical symptoms in the early onset stage, and is an important risk factor for developing coronary heart disease, heart failure, and cerebral stroke[6]. The elevation of SBP will increase the blood flow rate, bring a certain impact damage on the vascular wall, trigger inflammation reaction and lipid infiltration, thicken the cervical IMT, increase the vascular hardness, and reduce the compliance, resulting in arterial endothelial cell damage, and promoting the occurrence and development of AS; therefore, it is deemed that the blood pressure instability and high SBP can promote the vascular endothelial cell damage[7,8]. Researches demonstrate that the vascular endothelial dysfunction is closely associated with hypertension, and is an important factor for developing vascular endothelial injury, which can promote the development of AS, thicken the cervical IMT, and cause coronary artery and peripheral vascular diseases[9].

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observation group were significantly lower than those in the control group ($P<0.05$), indicating that reasonable treatment in combined with effective nursing interventions can significantly enhance the efficacy in patients with primary hypertension merged with carotid atherosclerosis.

In conclusion, the combined application of atorvastatin calcium and amlodipine can play a synergistic effect, effectively regulate the serum lipid, protect the vascular endothelial cell function, and shrink the cervical IMT, which is combined with nursing intervention can effectively enhance the clinical therapeutic effect in patients with primary hypertension merged with carotid atherosclerosis.

References


