Influence of rosuvastatin on blood lipid, inflammatory factor and oxidative stress index of patients with chronic heart failure

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ARTICLE INFO

Article history:
Received
Received in revised form
Accepted
Available online

Keywords:
Rui Shu Vatatin
Blood lipid
Inflammatory factor
Oxidative stress

ABSTRACT

Objective: To explore the effect of rosuvastatin on blood lipid and inflammatory factor and oxidative stress index of patients with chronic heart failure (CHF). Methods: 108 cases with CHF were selected as the study case, using the single blind method and were randomly divided into two groups, including 54 cases in the control group were treated with routine department of internal medicine treatment, while 54 patients in the observation group were treated with rosuvastatin on the basis of conventional department. The blood lipid, inflammatory factor and oxidative stress indicators of changes were detected and compared. Results: After treatment, LVEDD, LVESD of the observation group were (53.20±1.44) mm, (37.44 ±3.03) mm, and LVEF was (48.89±6.15)%, compared with the group before treatment and control group after treatment were improved significantly (P<0.05). After treatment, TC, TG, LDL-C of observation group were (5.42±0.38) mmol/L, (1.57 ±0.24) mmol/L, (3.10±0.43) mmol/L, and HDL-C was (1.74±0.35), compared with the group before treatment and control group after treatment were improved significantly (P<0.05). After treatment, IL-6, TNF-alpha, CRP of the observation group were (23.55±2.45) ng/L, (37.55±3.84) ng/L, (6.34 ±1.30) ng/L, compared with the group before treatment and control group after treatment were decreased significantly (P<0.05). After treatment, SOD, MDA of the observation group were (173.82±5.33) U/mL, (4.00±0.42) nmol/mL, compared with the group before treatment and control group after treatment were improved significantly (P<0.05). Conclusion: The curative effect of rosuvastatin on CHF is effective, can significantly improve the blood lipid, inflammatory factor and oxidative stress index, and has the higher the value of clinical application.

1. Introduction

Chronic heart failure (CHF) is the final destination and death cause of most cardiovascular disease patients, and it is also one of the clinical treatment problems at present. Previous treatment usually use strong heart, diuresis, expanding coronary artery and other drugs which can improve the clinical symptoms of patients to a certain extent, but cannot improve the prognosis of patients[1-3]. In recent years, with the increasing research of statins, it has been gradually applied in clinical treatment of CHF, which can significantly reduce the level of blood lipids, inflammatory factors and improve the prognosis of the patients[4-6]. The purpose of this study is to explore the clinical effect of conventional internal medicine combined rosuvastatin on the blood lipid, inflammatory factors and oxidative stress indexes of patients with CHF, detailed reports as follows.

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Fund: work was supported by the Science and Technology Innovation Fund Project Plan of Shanghai City (12456671403).
2. Materials and methods

2.1. Clinical information

108 cases with CHF admitted in our hospital from Aug. 2012 to Nov. 2013 were selected as the study case. All patients were diagnosed by clinical symptoms, electrocardiogram and ultrasound Heartbeat figure examinations. With male 65 cases, female 43 cases, age ranged from 40 to 77 years old, with an average age (55.54±4.34) years. According to NYHA (New York Heart Association), cardiac function grade II in 43 cases and 65 cases of grade III. The disease course was more than 6 months, and the left ventricular ejection fraction (LVEF) less than or equal to 40%. Excluded patients with acute heart failure, congenital heart disease, severe liver or kidney dysfunction and malignant tumor; patients used other lipid-lowering or antioxidant treatment. Used double blind method, patients were randomly divided into two groups, which showed no significant difference in the basic data and was comparable (P>0.05).

2.2. Treatment method

The control group patients were given conventional internal medicine treatment, including diuretics, beta blockers, digitalis, vasodilators and so on. The observation group patients were given rosvastatin on the basis of the conventional treatment, the dose was 20 mg/time, 1 times/day. Two groups had no difference on other medication; 3 month is 1 treatment course.

2.3. Evaluation indexes

Collected elbow venous blood after 12 h fasting, 3ml blood were analyzed by automatic biochemical analyzer to detect the blood lipid indexes, including triglyceride (TG), cholesterol (TC), high density lipoprotein cholesterol (HDLC-L) and low density lipoprotein cholesterol (LDLC-L). 5 mL blood were separated serum by centrifugal, to measure the inflammatory factors indexes which including C reaction protein (CRP), tumor necrosis factor alpha (TNF-α ) and interleukin-6 (IL-6), and the oxidative stress indexes which including superoxide dismutase (SOD) and malondialdehyde (MDA). Used color ultrasound diagnostic apparatus to determine the Left ventricular ejection fraction (LVEF), left ventricular end systolic diameter (LVESD) and left ventricular end diastolic diameter (LVEDD).

2.4. Statistical analysis

The dates were analyzed by statistics software SPSS 18.00, measurement dates showed by (mean±SD), used t test, P<0.05 was considered statistically significant difference.

3. Results

3.1. The changes of heart function indexes of the patients before and after treatment

After treatment, LVEDD, LVESD of the observation group were (53.20±1.44) mm, (37.44±3.03) mm, which were significantly decreased compared with the same group before treatment and the control group after treatment; LVEF was (48.89±6.15)%, which was significantly increased compared with the same group before treatment and the control group after treatment. The differences were all statistically significant (P<0.05). Detailed results are shown in Table 1.

<table>
<thead>
<tr>
<th>Group</th>
<th>Time</th>
<th>LVEDD (mm)</th>
<th>LVESD (mm)</th>
<th>LVEF (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>Before treatment</td>
<td>65.44±2.23</td>
<td>48.84±3.32</td>
<td>36.56±5.33</td>
</tr>
<tr>
<td></td>
<td>After treatment</td>
<td>60.01±1.73</td>
<td>43.52±3.54</td>
<td>42.61±5.03</td>
</tr>
<tr>
<td>Observation</td>
<td>Before treatment</td>
<td>66.00±2.04</td>
<td>49.03±2.83</td>
<td>35.10±4.85</td>
</tr>
<tr>
<td></td>
<td>After treatment</td>
<td>53.20±1.44</td>
<td>37.44±3.03</td>
<td>48.89±6.15</td>
</tr>
</tbody>
</table>

Ps compared with the control group, P<0.05; compared with the same group before treatment, P<0.05.

3.2. The changes of the blood lipid indexes of the patients before and after treatment

After treatment, TC, TG and LDL-C of the observation group were (5.42±0.38) mmol/L, (1.57±0.24) mmol/L and (3.10±0.43) mmol/L, which were significantly decreased compared with the same group before treatment and the control group after treatment; HDL-C was (1.74±0.35) mmol/L, which was significantly increased compared with the same group before treatment and the control group after treatment. The differences were all statistically significant (P<0.05). Detailed results are shown in Table 2.

<table>
<thead>
<tr>
<th>Group</th>
<th>Time</th>
<th>TC (mmol/L)</th>
<th>TG (mmol/L)</th>
<th>HDL-C (mmol/L)</th>
<th>LDL-C (mmol/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>Before treatment</td>
<td>6.23±0.44</td>
<td>2.10±0.26</td>
<td>1.35±0.22</td>
<td>3.60±0.55</td>
</tr>
<tr>
<td></td>
<td>After treatment</td>
<td>5.90±0.37</td>
<td>1.99±0.21</td>
<td>1.27±0.16</td>
<td>3.50±0.38</td>
</tr>
<tr>
<td>Observation</td>
<td>Before treatment</td>
<td>6.34±0.51</td>
<td>2.01±0.23</td>
<td>1.40±0.24</td>
<td>3.54±0.45</td>
</tr>
<tr>
<td></td>
<td>After treatment</td>
<td>5.42±0.38</td>
<td>1.57±0.24</td>
<td>1.74±0.35</td>
<td>3.10±0.43</td>
</tr>
</tbody>
</table>

Ps compared with the control group, P<0.05; compared with the same group before treatment, P<0.05.
3.3. The changes of the inflammatory factor indexes of the patients before and after treatment

After treatment, IL-6, TNF-α and CRP of the observation group were (23.55±2.45) ng/L, (37.55±3.84) ng/L and (6.34±1.30) ng/L, which were significantly decreased compared with the same group before treatment and the control group after treatment, and the differences were all statistically significant (P<0.05). Detailed results are shown in Table 3.

<table>
<thead>
<tr>
<th>Group</th>
<th>Time</th>
<th>IL-6 (ng/L)</th>
<th>TNF-α (ng/L)</th>
<th>CRP (g/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>Before treatment</td>
<td>39.54±3.55</td>
<td>65.44±5.34</td>
<td>14.38±2.12</td>
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<tr>
<td></td>
<td>After treatment</td>
<td>37.22±3.43</td>
<td>60.31±3.75</td>
<td>13.25±1.82</td>
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<tr>
<td>Observation</td>
<td>Before treatment</td>
<td>38.43±3.04</td>
<td>66.34±4.03</td>
<td>14.72±2.00</td>
</tr>
<tr>
<td></td>
<td>After treatment</td>
<td>23.55±2.45b</td>
<td>37.55±3.84ab</td>
<td>6.34±1.30b</td>
</tr>
</tbody>
</table>

Ps: compared with the control group, *P<0.05; compared with the same group before treatment, †P<0.05.

3.4. The changes of oxidative stress of the patients before and after treatment

After treatment, SOD of the observation group was (173.82±5.33) U/mL, which was significantly increased compared with the same group before treatment and the control group after treatment; MDA was (4.00±0.42) nmol/mL, which was significantly decreased compared with the same group before treatment and the control group after treatment, and the differences were all statistically significant (P<0.05). Detailed results are shown in Table 4.

<table>
<thead>
<tr>
<th>Group</th>
<th>Time</th>
<th>SOD (U/mL)</th>
<th>MDA (nmol/mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>Before treatment</td>
<td>93.44±3.94</td>
<td>7.89±0.55</td>
</tr>
<tr>
<td></td>
<td>After treatment</td>
<td>100.30±4.75</td>
<td>7.24±0.63</td>
</tr>
<tr>
<td>Observation</td>
<td>Before treatment</td>
<td>95.29±5.24</td>
<td>8.20±0.56</td>
</tr>
<tr>
<td></td>
<td>After treatment</td>
<td>173.82±5.33b</td>
<td>4.00±0.42b</td>
</tr>
</tbody>
</table>

Ps: compared with the control group, *P<0.05; compared with the same group before treatment, †P<0.05.

4. Discussion

Chronic heart failure is a clinically common malignant syndrome and the death cause of variety organic heart diseases. In recent years, with the further research of CHF pathogenesis, people have confirmed and gradually pay attention to myocardial remodeling, which play an important role in the occurrence and development of the disease[7]. Studies suggest that myocardial remodeling is a complex pathological process[8]. In patients with early myocardial injury, many secondary factors affect myocardial cells directly or indirectly and improve myocardial remodeling. Clinical research has confirmed that cytokines and oxidative stress are the mainly factor mediate myocardial remodeling, which can mediate left ventricular remodeling, decrease myocardial contraction to trigger or worsen the occurrence and development of heart failure[9-11].

Normal myocardial cells do not produce TNF-α, but a variety of factors will stimulate the cell to produce large amounts of TNF-α, which participate in the ventricular remodeling process, induce the generation of IL-6 and promote the development of heart failure[12]. IL-6 can not only regulate the immune and inflammatory reaction, but also may affect myocardial hypertrophy and involve in myocardial remodeling[13]. SOD is a kind of important antioxidant enzymes in vivo, which can reflect the oxygen free radical scavenging capacity. MAD can cause metabolic products of peroxidation; its content changes can indirectly reflect the degree of tissue damage. The combination of SOD and MAD can be used as an index to judge the degree of oxidative stress[14,15]. Our study aims to explore the effect of rosuvastatin on the inflammatory factors and oxidative stress indexes of patients with CHF and we found that after treatment, inflammatory factor IL-6, TNF-α and CRP levels were significantly decreased compared with the conventional internal medicine group and the same group before treatment (P<0.05). Oxidative stress indexes SOD and MDA were significantly improved compared with the conventional internal medicine group and the same group before treatment (P<0.05). Our study indicated that rosuvastatin can decrease the level of inflammatory factors and improve the ability of antioxidation in patients with CHF.

Statins are clinical commonly used drugs in treatment of cardiovascular disease. In addition to a lipid-lowering effect, it also can improve the myocardial cell contraction and reverse ventricular remodeling in a certain extent[16]. Our study showed that after rosuvastatin combined with conventional internal medicine treatment, patients heart function indexes LVEDD, LVESD and LVEF were (53.20±1.44) mm, (37.44±3.03) mm and (48.89±6.15)% that were significantly improved compared with the conventional internal medicine group and the same group before treatment (P<0.05). The blood lipid indexes TC, TG and LDL-C levels were significantly decreased while HDL-C level was significantly increased (P<0.05), which indicated that rosuvastatin can well regulate blood lipids, change myocardial energy metabolism, repair damaged myocardial cells and improve heart function in patients with HCF.
In summary, the curative effect of rosuvastatin on CHF is effective, it is not only can significantly improve the heart function and blood lipid levels but also can significantly reduce the level of serum inflammatory factor and oxidative stress, so as to play a better role of anti-heart failure and has the high value of clinical application.

References


