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Influence of early application of simvastatin on high-sensitivity C-reactive protein and fibrinolysis coagulation of unstable angina pectoris patients.

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Abstract

Purpose: To explore the influence of early application of Simvastatin on high-sensitivity C-reactive protein and fibrinolysis coagulation of unstable angina pectoris patients.

Method: 70 cases of unstable pectoris patients received and treated in our hospital during March 2011-2014 are taken as the research objects, and are divided into two groups randomly, 35 cases in each group. The patients of research group I are treated with simvastatin pills and that of research group II are treated with nitrate types and angiotensin invertase enzyme inhibitor; at the same time, 35 patients with no cardiac diseases are selected as the blank control group. The changes on blood lipid, high-sensitivity C-reactive protein and fibrinolysis coagulation of the three groups before and after treatment are observed.

Results: The difference on blood lipid between research group I and research group II has no statistical significance (P>0.05). Compared with that of research group II, the high-sensitivity C-reactive protein of research group I is significantly reduced, with difference with statistical significance (q=9.25, P=0.02<0.05). On the aspect of coagulation fibrinolysis, the plasma blood coagulation factor VII, the fibrinogen and the plasminogen activator inhibitor of research group I are significantly reduced, and they have differences with statistical significance (P<0.05) when compared with that of research group II. At the same time, it has lower tissue-type plasminogen activator than that of research group II (F, P<0.05).

Conclusion: Early application of simvastatin can effectively reduce the blood lipid content and decrease the high-inflammation level of unstable angina pectoris patients; in addition, it can improve the anti-coagulation level, helpful to the stability of plaques.

Keywords: Unstable angina pectoris, Simvastatin, High-sensitivity C-reactive protein, Fibrinolysis coagulation.

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Introduction

The unstable angina pectoris is the clinical manifestations between the angina pectoris of effort and acute myocardial infarction and sudden death. It has the following clinical manifestations: progressive exacerbation of manifestation of angina pectoris, longer lasting time for angina pectoris and increase in time of angina pectoris during night [1]. Due to unique physiopathological mechanism, unstable angina pectoris may lead to deterioration of disease if no timely clinical treatment is provided, endangering the life of patients. Generally speaking, plaque rupture of unstable angina pectoris patients will lead to artery occlusion; therefore, it is required to control the blood lipid level and reduce the inflammation level during treatment. At the same time, the fibrinolysis coagulation level reflects the stability of plaques [2]. In conventional therapies, hypogloeeis taking of nitroglycerin and calcium ion antagonist can be utilized to relieve the symptoms of angina pectoris, but they cannot play an effective role in control of inflammation level [3]. Based on the treatment to unstable angina pectoris patients in our hospital, this article explores the influence of early application of simvastatin on high-sensitivity C-reactive protein and fibrinolysis coagulation, with the following research findings.

Data and Method

Research data

Totally 70 unstable angina pectoris patients were received and treated in our hospital during March 2011-2014, and they were taken as the research objects and were divided into two groups randomly, 35 cases in each group. Research group I included 19 males and 16 females, with ages of 38-67 y old, 56.3 ± 8.6 on average. Research group II included 18 males and 17 females, with ages of 40-69 y old, 57.0 ± 9.2 on average. There was no statistical significance on illness degree between the two groups (P>0.05). At the same time, 35 health people were selected as the blank control group, including 17 males and 18 females, with ages of 40-67 y old, 57.2 ± 7.7 on average. There was no statistical significance on ages and genders among the three groups (P>0.05).

Admission criterion

(1) Change on the nature of the original stable angina pectoris; i.e., more frequent attacks, more severe condition and longer lasting time; (2) Attacks of angina pectoris during rest time; (3) Dynamic horizontal or adown and oblique depression of ST section ≥ 1 mm or elevation of ST section (limb lead ≥ 1 mm, chest lead ≥ 2 mm) was the most important for diagnosis. The patients with acute and chronic infection, damage, hepatic and renal function damage or those with rheumatic immunologic diseases, obsolete or acute myocardial infarction were not included into the research [4,5].

Therapeutic method

Research group I was treated with simvastatin, nitroglycerin, calcium ion antagonist, angiotensin converting enzyme inhibitor. Research group II was treated with nitroglycerin, calcium ion antagonist and angiotensin converting enzyme inhibitor. The control group was treated for blank control.

Measurement index

Blood lipid: The patients were taken with venous blood of 5ml for blood lipid detection with the full-automatic biochemical analyzer, with detection indexes of serum Total Cholesterol (TC), Triglyceride (TC), Low Density Lipoprotein (LDL) and High Density Lipoprotein (HDL) contents.

High-sensitivity C-reactive protein: The patients were taken with venous blood of 5 ml before and after the treatment, respectively, and the rate nephelometry method was adopted for measurement of hs-CRP concentration, with operation method strictly according to the description for the reagent [6].

Anti-coagulation and fibrinolysis indexes: The patients were taken with venous blood of 5 ml before and after treatment, respectively, and the one-stage method was adopted for detection on the plasma coagulation factor VII level. The developing substrate method was adopted for tissue-type plasminogen activating agent (tPA) and tissue-type plasminogen activation of zymogens (PAI). The semi-quantitative method was adopted for fibrinogen (FG).

Statistical treatment

The SPSS 20.0 was adopted for treatment of the database. The mean ± standard deviation was adopted for measurement data. The F-test was adopted for comparison among groups. The q-test was adopted for pair-wise comparison among three groups; the difference had statistical significance if $P<0.05$.

Results

Blood lipid level

The difference on blood lipid between the two research groups had no statistical significance ($P>0.05$). The serum total cholesterol, triglyceride and low density lipoprotein of research group I were significantly reduced and the high density lipoprotein was increased.

The changes on related indexes of research group II were the same as that of research group I, but the differences between the two groups had no statistical significance (q=0.56-0.90, P average>0.05) (Table 1).

Table 1. Comparisons on blood lipid levels between the two research groups (unit: mmol/L).

	n	TG		TC		LDL		HDL	
		Before	After	Before	After	Before	After	Before	After
Research group I	35	1.56 ± 0.26	1.37 ± 0.16	5.90 ± 0.56	3.83 ± 0.32	2.64 ± 0.53	2.60 ± 0.39	1.02 ± 0.18	1.04 ± 0.20
Research group II	35	1.57 ± 0.30	1.38 ± 0.28	5.82 ± 0.50	3.84 ± 0.31	2.65 ± 0.50	2.62 ± 0.43	1.01 ± 0.12	1.03 ± 0.19
q		0.67	0.84	0.9	0.87	0.74	0.56	0.78	0.8
P		0.76	0.73	0.83	0.86	0.79	0.82	0.84	0.83

Inflammation level

The hs-CRP level of research group I before treatment was 9.02 ± 0.37 mg/L; that of research group II was 9.01 ± 0.38 mg/L; that of the blank control group was 6.24 ± 0.42 mg/L; the difference among the three groups had statistical significance ($F=8.36$, $P=0.02<0.05$); i.e., the levels of the two research groups were significantly higher than that of the control group. However, there was no statistical significance ($q=0.49$, $P=0.82>0.05$) on the difference between the two research groups. After the treatment, the hs-CRP level of research group I was 6.42 ± 0.30 mg/L; that of research group II was 7.40 ± 0.40 mg/L; that of the blank control group was 6.24 ± 0.44 mg/L; the differences among the three groups had statistical significance ($F=12.25$, $P=0.01<0.05$). At the same time, the differences between the two research groups had statistical significance ($q=9.25$, $P=0.02<0.05$).

Anti-coagulation level

On the aspect of fibrinolysis coagulation, the levels of plasma coagulation factor Ⅶ, the fibrinogen and the plasminogen activation of zymogens were significantly reduced, and the difference with that of research group II had statistical

significance (q=5.90-8.43, P<0.05). At the same time, the level of the tissue-type plasminogen activating agent was increased, but lower than that of the two research groups (q=5.40, P<0.05) (Table 2).

Table 2. *Comparisons on anti-coagulation indexes between the two research groups.*

	n	F-VII (%)		tPA (ku/L)		PAI (ku/L)		FG (g/L)	
		Before	After	Before	After	Before	After	Before	After
Research group I	35	154.3 ± 12.37	149.4 ± 10.29	0.15 ± 0.03	0.28 ± 0.07	0.89 ± 0.11	0.85 ± 0.09	3.75 ± 0.07	3.48 ± 0.10
Research group II	35	153.9 ± 12.93	130.2 ± 10.03	0.15 ± 0.02	0.17 ± 0.03	0.88 ± 0.12	0.76 ± 0.08	3.75 ± 0.05	3.70 ± 0.08
q		1.35	8.43	0.01	7.92	1	5.4	0.03	5.9
P		0.73	0.32	1	0.3	0.8	0.4	1	0.42

Discussion

According to above results, the q-test method is adopted for the pair-wise comparisons on related research index averages between the two research groups. The two research groups do not have significant differences on blood lipid levels; i.e., both simvastatin and other medicines for unstable angina pectoris such as nitrate can effectively relieve the temporary symptoms of angina pectoris and control the growth speed of plaques. On the aspect of anti-coagulation fibrinolysis index, the plasma coagulation factor Ⅶ, the fibrinogen and the plasminogen activation of zymogens levels are all reduced, and the tissue-type plasminogen activation of zymogens levels are increased; it illustrates the roles played by both of them on plaque stability. However, research group I has stronger stability, which can reduce the occurrence frequency of angina pectoris. Both of the two groups have declined levels on the aspect of hs-CRP. Considering that the blood lipid levels of angina pectoris patients are significantly higher than that of healthy people, they often have higher hs-CRP levels than healthy people based on large numbers of researches; therefore, related indexes of the blank control group are not specifically listed in the research result of this article.

Based on related literatures of the Branch of Cardiovascular Diseases of Chinese Medical Association, unstable angina pectoris refers to a group of clinical angina syndromes between stable angina pectoris and Acute Myocardial Infarction (AMI), with large numbers of subtypes, including initial angina pectoris of effort, exacerbated angina pectoris of effort, angina pectoris after infarction, rest angina pectoris and variant angina pectoris, the grades of which are generally conducted as per the Canadian Heart Disease Branch and there is no uniform standard for the levels on risk factors [7]. Some researchers in our country classify as per Braunwald UAP classification methods of 1989 in combined with our national conditions [8].

hs-CRP is the important research factor in the research on related risk factors of unstable angina pectoris patients. It belongs to the same protein as CRP, but it has more sensitive detection. As an inflammation landmark, hs-CRP is related to coronary artery events, stroke and peripheral vascular diseases, existing as the independent factor for the above diseases. Therefore, the detection on hs-CRP is an important index for evaluation on inflammatory reaction. At the same time, according to large numbers of literature researches, the probability of the people on rise of the index or occurrence of brain stroke is higher than 2 times of that of healthy people, and the probability on myocardial infarction is 3 times of that of healthy people [9]. Therefore, it is especially important to conduct related detection. In this article, the hs-CRP levels of the two research groups are significantly higher than that of the control group before treatment, illustrating the angina pectoris patients have relatively high body inflammation levels, which is reduced after treatment, especially research group I.

The formation and cracking of unstable plaques can be predicted through anti-coagulation fibrinolysis indexes, in which the tissue-type plasminogen activating agent, the tissue-type plasminogen activation of zymogens and the fibrinogen can be taken as related landmarks for thrombus formation and explanation [10,11]. The blood lipid level of research group I changes with small extent at early stage of the angina pectoris, but there are significant changes on related anti-coagulation fibrinolytic system indexes such as tPA, FG, PIA and F-Ⅶ.

There are large numbers of researches on unstable angina pectoris, but most of them are emphasized on the blood lipid level for long term angina pectoris patients with simvastatin, so as to judge the plaque levels of the patients. The angina pectoris patients have higher blood lipid level for long term and enlarged plaques. It may lead to thrombus due to further falling off of plaques, which results in myocardial infarction, endangering the life of patients.

Conclusion

The early application of simvastatin for unstable angina pectoris can reduce the blood lipid contents to small extent, which reduces the hs-CRP, i.e., the body high inflammation level, and increases the anti-coagulation level. It is helpful to the stability of plaques, which shall be reasonably utilized in clinic.

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