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# Effect of Sodium Tanshinone IIA Sulfonate on uric acid, sicam-1, ET-1 and FMD in patients with coronary heart disease complicated with hyperuricemia

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**ABSTRACT**

This study was to observe the effect of Sodium TanshinoneIIA Sulfonate (ST-IIAS) on blood uric acid (UA), human Soluble Intercellular Adhesion Molecule-1 (sICAM-1), Endothelin-1 (ET-1) and percentage of brachial artery Flow-Mediated Dilatation (FMD) in individuals with Hyperuricemia Complicated Coronary Heart Disease (HC-CHD). The study's participants were 108 patients with HC-CHD who attended our hospital between January 2020 and June 2022. In the trial, the patients were split into two groups with 54 instances each: the general group and the observation group. The observation group received ST-IIAS therapy, while the general group received standard care. The experiment chose to observe and compare the difference of uric acid, sICAM-1, ET-1, FMD, therapeutic effectiveness and negative effects between the two groups at various times. Results showed that on the 14th day, the observation group's amounts of UA, sICAM-1, and ET-1 were inferior to the general group ($P$<0.05); On the 7th and 14th days, the observation group's amount of ET-1 was lower than that of the general group ($P$<0.05); The observation group's FMD of patients on the 14th day was inferior to the general group after treatment ($P$<0.05); The observation group's overall effective rate was 94.44% higher than the general group's ($P$<0.05); The observation group experienced fewer negative responses than the general group did ($P$<0.05). In conclusion, ST-IIAS can be used for uric acid, vascular endothelial systolic and diastolic function in patients with HC-CHD, and has better clinical efficacy and lower risk of adverse reactions.

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## Introduction

CHD, as a common cardiovascular disease in China, can cause problems such as insufficient cardiac output and decreased renal blood perfusion. CHD, as a heart disease based on coronary atherosclerosis, mostly has abnormal lipid metabolism, and its clinical manifestations worsen with the aggravation of atherosclerosis. Hyperuricemia is closely related to coronary atherosclerosis (1). Serum uric acid and high-density lipoprotein cholesterol had positive and negative correlations with atherosclerosis, respectively (2). It can be seen that there are many patients with HC-CHD. At present, for patients with HC-CHD, the combination of hypouricemic and hypolipidemic drugs is mainly based on the conventional treatment of CHD. This method will lead to the recurrence of clinical symptoms in patients, and long-term continuous use of multiple drugs will also lead to a decline in drug compliance and safety. ST-IIAS has many effects, such as expanding coronary arteries, reducing blood lipids, inhibiting blood coagulation, and improving myocardial ischemia-reperfusion injury. However, ST-IIAS is not a drug that directly reduces uric acid. Thus, to further improve the current situation of poor efficacy of combined medication in patients with HC-CHD, our hospital took ST-IIAS as the main treatment drug to observe the blood uric acid, sICAM-1, ET-1 and FMD of patients with HC-CHD. The findings are listed below.

## Materials and Methods

### General information

Select 108 patients with HC-CHD who visited our hospital between January 2020 and June 2022, and split them into the general group and observation group, with 54 individuals in each group. Inclusion criteria: ① The clinical symptoms, electrocardiogram, cardiac ultrasound, coronary angiography and other examinations were in line with the relevant domestic and international guidelines for the diagnosis of CHD (3-5); ② With typical symptoms of angina pectoris, patients often feel chest compression, tightness and accompanied by dyspnea, fatigue, etc.; ③ At the time of symptom attack, the electrocardiogram has transient ST segment change and T wave abnormality; ④ Medical image data showed segmental wall motion abnormalities; ⑤ Coronary angiography with Judkins method at least one coronary artery ≥ 50%; ⑥ Under the condition of a normal diet and purine diet, the blood uric acid level of male patients with non-two-day fasting blood uric acid level is more than 420μmol/L, female non-two-day

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fasting blood uric acid level, blood uric acid>357μmol/L; ⑦ The age is 40-75 years old. Exclusion criteria: routine ① those with experimental drug allergy; ② Those with aphasia, hemiplegia or inability to communicate verbally; ③ Previous history of epilepsy or brain hypoplasia; ④ Patients with tumor, anemia, immune deficiency or coagulation dysfunction; ⑤ Those who have undergone cardiac surgery; ⑥ Complicated with liver and kidney dysfunction, accompanied by gout, lactic acid poisoning, malignant tumor and other systemic diseases; ⑦ other diseases of the blood system lead to abnormal metabolism of uric acid; ⑧ Pregnant or lactating women; ⑨ Patients with incomplete or missing hospitalization information. The medical ethics council of the institution examined and authorized this research, and the study's participants gave their full permission and consented to its terms. 36 males and 18 females were in the general group; Age (62.79 ± 5.76) years; 38 cases of dyslipidemia; There were 15 cases of left main coronary artery disease, 23 cases of single branch disease and 16 cases of double branch disease; 32 cases were complicated with hypertension; 29 cases were treated with atorvastatin calcium tablets and 25 cases with simvastatin. 38 males and 16 females were in the observation group; Age: (62.88 ± 5.43) years old; 39 cases of abnormal blood lipid; There were 16 cases of left main coronary artery disease, 21 cases of single branch disease and 17 cases of double branch disease; 33 cases were complicated with hypertension; Atorvastatin calcium tablets were used in 30 cases and simvastatin in 24 cases. Sex, age, the location of a coronary artery disease, hyperlipidemia, hypertension, and between the two groups, there were no appreciable differences in the use of lipid-lowering drugs (P>0.05).

## Treatment

### *General Group*

Use routine treatment. Routine treatment of anti-CHD: the combination of antiplatelet, β receptor blocker, nitrate and statin lipid-regulating drugs. Aspirin enteric-coated tablets (Yunnan Baiyao Group Co., Ltd., GYZZ H53021845, specification: 25mg) 50mg, once a day, oral; Metoprolol succinate sustained-release tablets (Guangdong Dongguang Pharmaceutical Co., Ltd., GYZZ H20213975, specification 47.5mg) 95mg, once a day, oral; Isosorbide mononitrate sustained-release tablets (produced by Lunambat Pharmaceutical Co., Ltd., GYZZ H19991039, specification: 40mg) 80mg, once a day, oral in the morning; Atorvastatin calcium tablets (produced by Tiandi Hengyi Pharmaceutical Co., Ltd., GYZZ H20203358, specification: 20mg) 10mg, once a day, oral; Or simvastatin (produced by Anhui Yongshengtang Pharmaceutical Co., Ltd., GYZZ H20083382, specification: 10mg) 10mg, once a day, oral treatment. In addition to the conventional treatment of anti-CHD, the treatment of anti-hyperuricemia should be added. Benzbromarone (produced by Chengdu Beite Pharmaceutical Co., Ltd., national drug approval number H20223418, specification: 50mg) 50mg should be taken once a day at breakfast. The course of treatment was 14 days.

### *Observation group*

The patients were treated with ST-IIAS, and the statins in routine treatment were retained. That is to say, on the basis of atorvastatin or simvastatin, tanshinone II A sulfonate sodium treatment was performed. Atorvastatin or simvastatin are still treated in the observation group, and tanshinone IIA sodium sulfonate injection (produced by Shanghai Shangyao First Biochemical Pharmaceutical Co., Ltd., GYZZ H3102558, specification: 2ml: 10mg), in 250ml of 5% glucose shot or 0.9 sodium chloride injection, 40–80mg are dissolved., once a day, for 14 days.

## Observation indicators

(I) Serological indicators: The normal range of 3mL of peripheral blood from the two patient groups was collected before treatment, 7 days after treatment and 14 days after treatment. The blood was sent in 2 hours to the hospital's medical laboratory. Two professional laboratory physicians were selected to measure blood UA, SICAM-1and ET-1. The patient's peripheral blood samples were centrifuged at 2500r/min to obtain serum for standby. Among them, the UA test was evaluated by the uric acid oxidase peroxide coupling method. For SICAM - 1 and ET - 1, the automatic biochemical analyzer is used to determine them by nephelometry and ELISA. (II) Cardiac ultrasound examination: collect the percentage of brachial artery blood flow-mediated dilatation (FMD) under quiet state before and 14 days after treatment. The target artery at 2 cm above the elbow fossa of the right upper arm was seen by cardiac ultrasound, the brachial artery's proximal and posterior sides' intimal diameters (D0), and the measured value of the probe were adjusted in the longitudinal section. The cuff of the medical sphygmomanometer was used to pressurize the brachial artery of the upper arm, and the systolic pressure was more than 30mmHg (1mmHg=0.133kPa); Ultrasonic examination showed that there was no positive blood flow in the brachial artery and the gas in the cuff was rapidly released after 5 minutes, and the ultrasonic probe could be fixed at the original measurement position; The internal diameter of the brachial artery (D1) shall be recorded within 9s after deflation, using FMD=(D1-D0)/D0×100%; Take the average of three measurements. (III) Clinical efficacy and adverse reactions: The efficacy was evaluated by the clinician, and the obvious effect was that the chest pain of the patient disappeared obviously and the blood uric acid returned to normal; Effectively, the patient's chest pain was significantly reduced, and the level of blood uric acid decreased, sometimes exceeding 470 μmol/L but no persistence; Ineffective: there is still obvious chest pain or blood uric acid is still persistent (>3 days) more than 470 μmol/L. Adverse reactions were collected by the doctor and the patient through two channels of self-complaint, and the incidence of adverse reaction symptoms such as cardiac discomfort, diarrhea, stomach discomfort, and liver dysfunction was collected for comparison. (IV) Correlation analysis: coronary Gensini score was selected to reflect. Gensini score is mainly applied to quantitatively assess the degree of stenosis of each coronary artery lesion. If the diameter of stenosis is less than 25%, 1 point will be counted; 25% ≤ diameter<50%, 2 points; 4 points for 50% ≤ diameter<75%; 8 points for 75% ≤ diameter<90%; 90% ≤ diameter<99%, 16 points; diameter≥ 99%, 32 points. The sum of the scores of each diseased branch is the total score of the degree of coronary artery stenosis of the patient. The severity of CHD increases with the Gensini number.

### Statistical methods

SPSS 25.0 is applied for the data procedure. If the distribution interval of the data conforms to the normal distribution, it is expressed as $\bar{x}\pm s$. The comparison of the general group and the observation group passed the independent sample *t*-test; Randomized block univariate analysis of variance or paired sample t-tests were applied for comparison at different times within the group. Frequency or component ratios are used to describe the enumeration statistics and are statistically analyzed by $\chi^2$ test or Fisher's exact probability method, with the test level of $\alpha$=0.05. When $P<0.05$, it is possible to say that the data contrast is statistically important; When $P<0.01$, it can be considered that the data comparison has significant statistical significance.

## Results

### Comparison of general data of two groups of patients

None of the subjects selected in the experiment were exfoliated, and their baseline data are shown in Table 1. When it came to gender, age, cholesterol, and other basic statistics, there was no discernible variation involving the two organizations($P>0.05$). Therefore, the results show that the two patient groups' follow-up information is similar.

### Comparison of UA levels between two groups of patients

The two patient groups' UA values have changed over time, as shown in Table 2. From the data in the table, it is not difficult to find that, prior to therapy and on day 7 of treatment, there is no statistically meaningful variation between the two groups UA levels. On the 14th day of treatment, the UA level of the observation group was inferior to the general group, and a statistically meaningful difference was present($P<0.05$). The results of one-way ANOVA of randomized blocks showed that, despite the fact that there was a remarkable variation in duration, the groups or connections showed no statistically meaningful variation ($P<0.05$).

Figure 1 shows the average trend of the change in UA level in the two groups of individuals. From the figure, the UA level in both groups indicated a gradual downward trend. During the period from before treatment to 7 days of treatment, the UA level of the general group decreased more than the observation group. During this time, there was no statistically meaningful variation between the categories. Subsequently, on the 14th day of treatment, the UA level of patients in the observation group was 342.88 ± 60.07 μ Mol/L, UA level in the general group was 368.94 ± 63.60 μ mol/L. There was a noticeable disparity between the groups during this time. Therefore, during the period from 7 days to 14 days of therapy, the patients' UA level in the observation group was inferior to the general group.

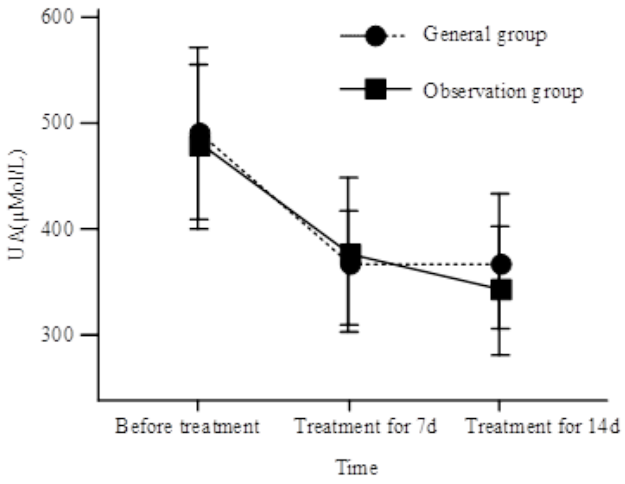


**Figure 1.** UA level change curve of two groups of patients.

**Table 1.** Comparison of general data of two groups of patients.

| Group (*n*) | Gender (example) | | Age (years old) | Dyslipidemia (case) | Hypertension (case) |
|---|---|---|---|---|---|
| | Male | Female | | | |
| General (54) | 36 | 18 | 63.46±6.33 | 38 | 32 |
| Observation (54) | 38 | 16 | 63.89±4.88 | 39 | 33 |
| $\chi^2/t$ | 0.172 | | -0.391 | 0.045 | 0.039 |
| *P* | 0.679 | | 0.696 | 0.832 | 0.844 |

| Group | Coronary artery lesion site (case) | | | Lipid-lowering drugs (case) | |
|---|---|---|---|---|---|
| | Left trunk | Single branch | Double branch | Atorvastatin calcium tablets | Simvastatin |
| General | 15 | 23 | 16 | 29 | 25 |
| Observation | 16 | 21 | 17 | 30 | 24 |
| $^2$ | | 0.153 | | 0.037 | |
| *P* | | 0.926 | | 0.847 | |

**Table 2.** Comparison of UA changes between two groups of patients (μmol/L, $\bar{x}\pm s$).

| Group (*n*) | Before treatment | Treatment for 7d | Treatment for 7d | *F* | *P* |
|---|---|---|---|---|---|
| General (54) | 490.34±80.47 | 363.60±53.06 | 368.94±63.60 | $F_{interblock}$=1.396 | $P_{interblock}$=0.240 |
| Observation (54) | 477.46±76.49 | 376.33±71.90 | 342.88±60.07 | $F_{time}$=162.057 | $P_{time}$=0.000 |
| *t* | 0.852 | -1.046 | 2.189 | $F_{mutual}$=0.430 | $P_{mutual}$=0.514 |
| *P* | 0.396 | 0.298 | 0.031 | | |

**Table 3.** Comparison of changes in SICAM-1 and ET-1 between two groups of patients ($\bar{x}$±s).

| Group (*n*) | SICAM-1 (ng/L) | | | *F* | *P* |
|---|---|---|---|---|---|
| | Before treatment | Treatment for 7d | Treatment for 7d | | |
| General (54) | 981.56±136.92 | 971.68±151.52 | 952.13±127.65 | $F_{interblock}$=4.898 | $P_{interblock}$=0.029 |
| Observation (54) | 979.17±146.41 | 961.63±80.52 | 863.73±110.07 | $F_{time}$=17.700 | $P_{time}$=0.000 |
| *t* | 0.089 | 0.431 | 3.854 | $F_{mutual}$=6.231 | $P_{mutual}$=0.014 |
| *P* | 0.929 | 0.667 | <0.001 | | |
| **Group (*n*)** | **ET-1 (pg/ml)** | | | ***F*** | ***P*** |
| | **Before treatment** | **Treatment for 7d** | **Treatment for 7d** | | |
| General (54) | 76.62±7.56 | 71.48±7.23 | 64.11±6.70 | $F_{interblock}$=83.717 | $P_{interblock}$<0.001 |
| Observation (54) | 75.70±8.25 | 65.32±7.43 | 51.65±4.21 | $F_{time}$=170.471 | $P_{time}$<0.001 |
| *t* | 0.602 | 4.364 | 11.575 | $F_{mutual}$=16.929 | $P_{mutual}$<0.001 |
| *P* | 0.548 | <0.001 | <0.001 | | |

## Comparison of sICAM-1 and ET-1 levels between the two groups

Figure 2 and Table 3 display the variations in sICAM-1 and ET-1 levels over the course of therapy for the two patient groups. From the data in the table, it is not difficult to find that before therapy and on day 7 of treatment, there is no statistically meaningful variation in sICAM-1 level between the two groups. On the 14th day of treatment, the SICAM-1's level in the observation group was remarkably inferior to the general group ($P$<0.05). The results of one-way ANOVA indicated that there were statistically significant variations in groups, time and interaction ($P$<0.05). Before therapy, there was no discernible change in the levels of ET-1 involving the two groups. On the 7th and 14th day of treatment, the degree of ET-1 in the observation group was much inferior to the general group($P$<0.05). The results of one-way ANOVA indicated that there were statistically remarkable variations among groups, time and interaction ($P$<0.05). The levels of SICAM-1 and ET-1 in both groups showed a gradual downward trend, as shown in Figure 2.

Figure 2 shows the average trend of changes in SICAM-1 and ET-1 levels in two groups of individuals. It can be seen from the figure that the levels of SICAM-1 and ET-1 of patients in both groups showed a gradual downward trend. In Figure 2 (a), the patients' SICAM-1 level in the observation group decreased more than the general group from the time before treatment to the 14th day of treatment. On the 14th day of therapy, the patients' SICAM-1 level in the observation group was 863.73±110.07μmol/L. The patients' SICAM-1 level in the general group was 952.13±127.65μmol/L. Therefore, during the period from 7 days to 14 days of therapy, the observation group's amount of SICAM-1 was inferior to the general group. In Figure 2 (b), during the course of the therapy, the ET-1 in the observation group dropped more noticeably than in the general group. On the 14th day, the ET-1 level in the observation group was 51.65±4.21μmol/L, and the ET-1 level in the general group was 64.11±6.70μmol/L. Therefore, during the period from 7 days to 14 days of treatment, the observation group's amount of ET-1 was superior to the general group's. The variations mentioned above were all numerically meaningful.

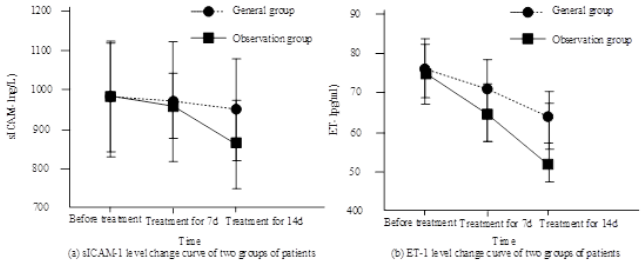


**Figure 2.** Changes of SICAM-1 and ET-1 levels in two groups of patients.

## FMD changes in two groups of patients

Table 4 shows the change in FMD levels during treatment in two groups of individuals. The data in the table indicates that before and after the 7-day treatment session, there is no notable difference in the UA readings between the two groups. Patients in the observation group had reduced FMD after 14 days of therapy than the general group, and this difference was statistically significant ($P$<0.05).

Figure 3 shows the average trend of FMD level changes in two groups of patients. From the figure, the patients' FMD levels in both groups showed a gradual downward trend. The FMD level in the general group decreased more than in the observation group from the time before treatment to 7 days after treatment. At this point, there was no statistically meaningful variation between the groups. Then, on the 14th day of treatment, the FMD level in the observation group was 33.34±0.19μmol/L, FMD level of patients in the general group is 3.59 ± 0.51μmol/L. The

**Table 4.** Comparison of FMD changes between two groups of patients (%, $\bar{x}$±s)

| Group (*n*) | Before treatment | Treatment for 7d | Treatment for 14d | *t* | *P* |
|---|---|---|---|---|---|
| General (54) | 4.71±0.54 | 4.56±0.36 | 3.59±0.51 | -10.503 | <0.001 |
| Observation (54) | 4.78±0.49 | 4.12±0.23 | 3.34±0.19 | -19.236 | <0.001 |
| *t* | -0.683 | -1.076 | 3.385 | / | / |
| *P* | 0.496 | 0.274 | 0.001 | / | / |

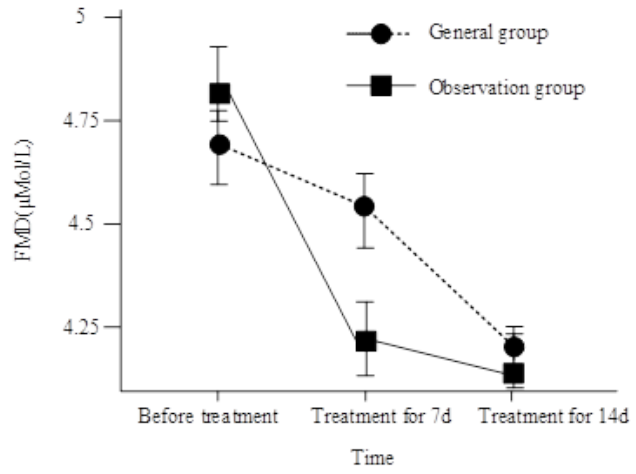


**Figure 3.** FMD level change curve of two groups of patients.

variation of groups during this period was significant. Therefore, during the period from 7 days to 14 days of therapy, the FMD level in the observation group was superior to the general group.

### Comparison of clinical efficacy and adverse reactions between the two groups

The observation group's overall effective rate was 94.44% (35 cases were highly effective, 16 cases were effective, and 3 cases were ineffectual), compared to the general group's overall effective rate of 79.63%(22 cases were highly effective, 21 cases were effective, and 11 cases were ineffectual). In comparison to the general group, the observation group's overall effective rate was greater, and the difference was remarkably significant ($\chi^2$=8.212, P=0.017). There was no obvious adverse reaction in the observation group; There were 2 cases of pit discomfort, 3 cases of diarrhea and 4 cases of stomach discomfort in the general group, and between the two categories, there was a statistically remarkable variation. (*P*=0.003) (Table 5).

### Correlation analysis of blood uric acid level with the number and severity of coronary artery disease

Figure 4 shows the correlation between the general group's blood uric acid level, heart plaques, and disease severity. It is not difficult to find that the quantity of heart plaques and blood uric acid levels are correlated favorably; There was also a positive correlation between serum uric acid level and Gensini score. Blood uric acid levels and the quantity of myocardial plaques had a 0.613 correlation coefficient; serum uric acid levels and the Gensini score had a 0.876 correlation coefficient.

Table 6 indicates the comparative findings of the Gensini score and the amount of coronary artery defects between the two groups. From the displayed data, the Gensini score in the general group was superior to the observation group, and the P value is less than 0.05. In addition, the number of coronary artery lesions in the observation group was also superior to the general group, and there was a statistically remarkable variation between the two categories.

## Discussion

Hyperuricemia is related to a greater danger of all-cause death 2 and 5 years after PCI in patients with CHD and myocardial infarction (6). At the same time, hyperuricemia and dyslipidemia will aggravate left ventricular hypertrophy, and there is a risk of high serum UA levels in patients with CHD and without the ApoEε4 gene (7). Research shows that the risk of CHD in patients with hyperuricemia has increased and is mainly mediated by metabolic factors, especially hypertension, hypercholesterolemia, low high-density lipoprotein cholesterol and low-density lipoprotein cholesterol (8). It can be seen that for patients with HC-CHD, their clinical medication should pay more attention to their abnormal lipid metabolism. Lipid-lowering and hypouricemia are common diagnoses and treatment ideas for such patients. Although CHD combined with hyperuricemia has not formed an authoritative diagnosis and treatment guide document, it still follows the principle of a combination of five drugs at this stage.

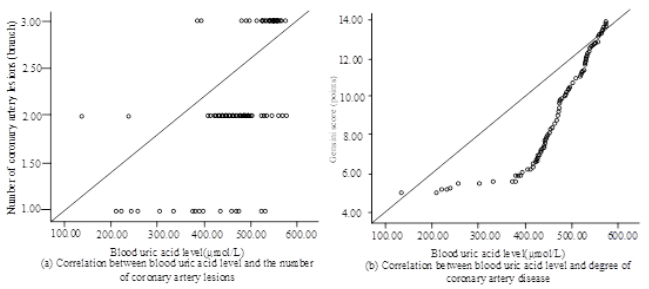


**Figure 4.** Correlation results between the blood uric acid level and the number of coronary artery lesions and the degree of disease in the general group.

**Table 5.** Comparison of adverse reactions between the two groups.

| Group (*n*) | Cardiac pit discomfort/case | Diarrhea/case | Gastric discomfort/case | Adverse reaction rate (%) | Total efficiency (%) |
|---|---|---|---|---|---|
| General (54) | 2 | 3 | 4 | 16.67% | 79.63% |
| Observation (54) | 0 | 0 | 0 | 0.00% | 94.44% |
| *t* | / | / | / | / | 8.212 |
| *P* | 0.001 | 0.023 | 0.031 | 0.003 | 0.017 |

**Table 6.** Comparison of Gensini score and number of coronary artery lesions between the two groups.

| Group (*n*) | Gensini score (points) | Number of diseased branches (branch) |
|---|---|---|
| General (54) | 54.78±8.14 | 2.13±0.31 |
| Observation (54) | 42.32±5.21 | 1.89±0.42 |
| *t* | 15.231 | 11.023 |
| *P* | 0.001 | 0.001 |

The drugs for reducing uric acid are mainly febuxostat and benzbromarone. Febuprista can cause sinus bradycardia, atrial fibrillation and other adverse cardiac reactions, and is usually not used as the clinical first choice for patients with CHD (9). Although benzbromarone has mild adverse reactions such as heart pit discomfort and edema, it has become a typical therapy for CHD patients due to the absence of earlier uric acid reduction and blood cholesterol improvement methods (10). However, there are still some individuals with HC-CHD from a therapeutic perspective.

ST-IIAS can antagonize the activity of purine oxidase, lower the rate of UA production noticeably, and also enhance the renal blood flow, promote the excretion of uric acid, and will not cause damage to the kidney. Meanwhile, ST-IIAS can dilate the coronary artery, reduce the left ventricular wall tension, reduce blood lipids, inhibit blood coagulation, and play the role of reducing myocardial oxygen consumption and protecting myocardium (11). The study demonstrates that ST-IIAS and enalapril coupled can lower hematocrit, whole blood viscosity, and plasma viscosity, demonstrating acceptable safety (12). ST-IIAS is mainly composed of tanshinone IIA, which is a part of the fat-soluble elements of the *Salvia miltiorrhiza* used in traditional Chinese medicine. After sulfonation, it becomes ST-IIAS (13). *S. miltiorrhiza* has the effect of cooling blood and eliminating carbuncle. Tanshinones have antibacterial, anti-inflammatory and sex hormone-like effects, and have been used in clinical diagnosis for many years.

The study's results revealed that the content of UA, sICAM-1 and ET-1 of patients treated with ST-IIAS at the end of treatment were lower in the general group, and the level of ET-1 was inferior to the general group at the beginning of 7 days of treatment. It can be seen that the treatment of ST-IIAS can reduce blood uric acid and improve vascular endothelial function. The occurrence of CHD is related to atherosclerosis, endothelial cell mitochondrial damage and scorch death (14). The general group was treated with benzbromarone. Although benzbromarone had an inhibitory effect on uric acid anion transporter 1 and reduced the reabsorption rate of uric acid in renal proximal convoluted tubules, compared with ST-IIAS, it did not reduce the activity of purine oxide enzyme in the body and still produced a certain amount of UA, which was still harmful to patients with CHD. ST-IIAS has the effect of reducing uric acid by antagonizing the activity of purine oxidase and promoting the excretion of uric acid, which is different from that of ST-IIAS. As a cell adhesion factor, the decrease of sICAM-1 marks the decrease of white blood cells adhered to the arterial endothelial cells, prevents the damage of vascular endothelial cell function and reduces vascular permeability. ET-1 is a vasoconstrictive factor, and its level is related to the decline of vascular endothelial regulatory function in patients. Patients with CHD have the problem with endothelial dysfunction, vascular contraction and relaxation dysfunction, leading to the increase of ET-1 level. Treatment with ST-IIAS can reduce ET-1 faster and improve the vascular endothelial contraction function of patients. FMD is used as an ultrasound indicator of vascular endothelial diastolic function damage. The treatment of ST-IIAS can also effectively reduce the FMD level after treatment and improve the vascular endothelial relaxation function. At the same time, the observation group's overall effective rate was 94.44% higher than the general group's ($P$<0.05); Research shows that ST-IIAS has a potential therapeutic effect on ischemic cerebral infarction, can improve the proteomics of the cerebral infarction site, and help improve the neurological function (15,16). Based on the similarity of cardio-cerebrovascular diseases, we can speculate that ST-IIAS can have beneficial effects on cardiovascular proteomics. Research and analysis found that the treatment of ST-IIAS can effectively reduce adverse cardiovascular events, blood lipids and total bilirubin levels in patients with CHD (17,18). Yang Liwang scholar (19) once found that ST-IIAS can induce autophagy and improve cardiac insufficiency in septic mice. The observation group experienced fewer unpleasant responses than the general group did, and this variation was numerically noteworthy ($P$<0.05). This result is similar to that of Li S et al. (20). However, this study is only limited to short-term clinical efficacy, and the clinical indicators of patients after the course of treatment have not been followed up. In the future, further consideration will be given to improving the way of administration of tanshinone IIA sulfonate sodium or finding a more convenient and convenient treatment for simultaneous anti-CHD and hyperuricemia. In addition, analysis was done on the link between blood uric acid levels, heart plaques, and the severity of the illness. The relationship between the blood uric acid level and the number of coronary lesions and the severity of the disease showed that the amount of heart plaques and the Gensini score were both favorably associated with the blood uric acid level, and the correlation coefficient between the blood uric acid level and the number of coronary lesions was 0.613; The Gensini score and blood uric acid content had a 0.876 association value. Statistics showed a disparity between the two groups, with more myocardial plaques and a higher Gensini score in the general group than in the observation group. There are many genetic, biochemical, physiological, and epigenetic findings related to heart diseases (21-26).

To sum up, ST-IIAS is used for uric acid, vascular endothelial contraction and relaxation function in patients with HC-CHD, and has better clinical efficacy and lower risk of adverse reactions.

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