Original Article
Efficacy and safety of coronary stent intervention for coronary heart disease and its impact on short-term and long-term prognosis

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Abstract: Objective: To investigate the effect of coronary stent intervention on the efficacy, safety, and short-term and long-term prognosis of patients with coronary heart disease (CHD). Methods: From March 2016 to November 2017, 60 cases of CHD patients receiving coronary stent interventiontherapy (research group) and 42 cases of patients receiving conventional drug therapy (control group) in our hospital were recruited as research objects. The clinical efficacy, safety and prognosis in the two groups were compared, and the levels of matrix metalloproteinase-9 (MMP-9) and interleukin-33 (IL-33) in the two groups were measured before and after treatment. Results: The clinical efficacy and prognostic quality of life of patients in the research group were higher than those in the control group (*P*<0.050), and the serum levels of MMP-9 and IL-33 in the research group were higher than those in the control group after treatment (*P*<0.050). Conclusion: Compared with drug therapy alone, coronary stent intervention therapy has better clinical efficacy and short-term and long-term prognosis in treating CHD, but it is easy to promote inflammatory reaction after surgery. Therefore, attention should be paid to the control of inflammation before and after treatment.

Keywords: Coronary stent, coronary heart disease, efficacy, safety, prognosis

Introduction

Coronary atherosclerotic heart disease is referred to as coronary heart disease (CHD), which is a common clinical cardiovascular disease caused by coronary atherosclerosis, which has the characteristics of acute onset and severe illness. In recent years, due to changes in people's lifestyle and eating habits, the incidence of CHD has increased year by year. In Britain, an estimated 2.3 million people suffer from CHD [1]. CHD not only has an extremely high incidence rate, but also a high mortality rate. According to the team of Richards SH [2], over one seventh of men and one tenth of women in Britain die of CHD every year, with approximately 70,000 deaths. In addition, the World Health Organization reported in 2015 that cardiovascular diseases account for 31% of all deaths in the world, of which an estimated 7.4 million died of CHD.

CHD is the main cause of death in the world. Although smoking, diabetes, hypertension and other major risk factors have been confirmed, its specific pathogenesis is still not completely clear [4, 5]. The incidence of CHD is mostly in middle-aged and elderly people, and in recent years, there has been a trend towards younger age [6]. Its main clinical manifestations include chest pain, chest tightness, syncope, etc. Without timely and effective treatment, myocardial infarction or sudden cardiac death may occur. At present, the main treatment methods for CHD include drug therapy and surgical treatment [7, 8]. With the development and maturity of minimally invasive surgery technology, coronary stent has gradually become the main method for the treatment of CHD [9]. Coronary stent intervention is to provide mechanical support by implanting stents in narrow coronary arteries to expand the initial diameter of a patient's blood vessel, thereby reducing and

avoiding elastic recoil of the vascular wall. It has been reported that coronary stent intervention is not only minimally invasive, but also effective and safe [10]. However, some studies also suggest that the probability of sudden cardiac death after coronary stent intervention is increasing, which may lead to the occurrence of cardiac-related adverse events [11]. The matrix metalloproteinase-9 (MMP-9) gene is one of the important members of the MMP family, with a vital role in degrading and reshaping the dynamic balance of the extracellular matrix [12]. Interleukin-33 (IL-33), a new member of the IL family, is one of the important regulators of inflammatory response and immune bias, mainly inducing Th2-type immunity; it is present in the nucleus under normal conditions and functions as a transcription factor, and when cells are damaged, it can be secreted into the extracellular area and then bind to the receptor ST2, thus acting as a cytokine [13]. Both MMP-9 and IL-33 are recognized as crucial indicators for the formation of vascular endothelial injury, inflammatory response, vascular plaque and obstruction, and have important significance in CHD [14, 15]. Therefore, for further understanding of the efficacy and safety of coronary stenting intervention in CHD, we also measured levels of MMP-9 and IL-33 in patients' serum before and after treatment to clarify their specific application value, so as to provide effective and reliable references for future clinical treatment of such patients.

Data and methods

General data

A total of 102 patients with CHD admitted to Zhuji Affiliated Hospital of Shaoxing University from March 2016 to November 2017 were recruited as the research objects. Among them, 60 patients who received coronary stent interventional therapy was enrolled in the research group, and another 42 patients treated with conventional drugs were enrolled in the control group. This study was approved by the ethical committee of our hospital, and all study subjects above signed the informed consent.

Inclusion and exclusion criteria

Inclusion criteria: patients met the clinical diagnostic criteria of CHD and had not received relevant treatment before the experiment [16]. Exclusion criteria: patients could not tolerate surgery; Patients were complicated with malignant tumors, other autoimmune diseases or systemic infection, end-stage disease, or drug allergy; patients had mental diseases and were unable to cooperate with the treatment; Patients had incomplete clinical and pathological data; Patients transferred to other hospitals.

Methods

Patients in the control group received hypoglycemic lipid and other medications: 300 mg of aspirin (Sichuan Pacific Pharmaceutical Co., Ltd., State Food and Drug Administration: H51021475) orally, 300-600 mg of clopidogrel (Shenzhen Salubris Pharmaceutical Co., Ltd.; State Food and Drug Administration: H200-00542) supplemented, and whether other anti-anginal drugs or calcium channel blocker were needed according to the patient's condition. Patients in the research group received coronary stent interventional therapy on the basis above: all stents were introduced via the femoral or radial artery, and after completion of local puncture, a 6-7 F sheath was placed and inserted into the left coronary artery contrast tube and hooked to the left coronary artery orifice, and contrast agent was injected. Then, the patient was illuminated in multiple projection positions. When the operation in the left side was completed, the right coronary artery orifice was hooked with the right coronary angiography tube, and the contrast agent was injected and the patient was illuminated in multiple projection positions. After coronary angiography, 5000-7000 U of heparin (Shenzhen Saibaoer Bio-Pharmaceutical Co., Ltd., State Food and Drug Administration: H20052319) was added through the lateral wall of the arterial sheath, and the additional dose of heparin was 1000-2000 U/h. When a lesion site was found, a micro-guide wire was placed into it along the catheter position for the patient requiring coronary stent, and a balloon was implanted along the micro-guide wire according to the diameter of the vessel to achieve a pre-dilatation effect. After that, the balloon was withdrawn and a balloon-expandable stent was applied, and a stent (1.1:1) was selected to put into the lesion site along the micro-guide wire stent. Then, the stent was placed under through 12-14 atm high pressure, and angiography was performed for the patients again to determine the stent implantation.

Outcome measures

Clinical efficacy of the two groups of patients: Marked response: patients with good cardiac function score, and the clinical symptoms disappeared. Effective response: patients with good cardiac function score, and the clinical symptoms relieved. No response: patients' symptoms did not improve or worsen. Total effective rate = (marked response + effective response)/total number of cases ×100%. The incidence of complications between the two groups was compared. A questionnaire in a 100-point scale was applied to score the quality of life of patients three months after discharge, mainly including physical function score, cognitive function score, social activity function score, anxiety and other adverse emotion score, disease recurrence score, and pain score. The higher the score, the better the quality of life. Patients were followed up for two years to compare their survival. The related indexes were also compared: levels of MMP-9 and IL-33 and their correlation: fasting venous blood (5 ml) was drawn from patients before treatment (T1), 3 days after treatment (T2) and 7 days after treatment (T3), respectively. The venous blood was stored in a refrigerator at low temperature. After coagulation for 60 min, the blood was centrifuged at 1000×g at 4°C for 20 min, and the serum was frozen. The levels of MMP-9 and IL-33 in serum of the two groups of patients were determined by enzyme-linked immunosorbent assay (ELISA). The MMP-9 and IL-33 kits were purchased from Zhen Shanghai Industrial Co., Ltd., with item numbers of Hz-MP9-C, HZ-IL33-Mu. All operations were carried out in strict accordance with the kit instructions.

Statistical methods

SPSS24.0 statistical software (Shanghai Yuchuang Network Technology Co., Ltd.) was used to process all data results. All graph results were plotted by Graphpad8 (SOFTHEAD Inc., Shenzhen). Enumeration data were expressed in the form of rate, and chi-square test was used for inter-group comparison. Measurement data were expressed in the form of (mean ± standard deviation), and t test was used for inter-group comparison. Expression at multiple time points were compared using ANOVA with repeated measurements. Pearsom

correlation analysis was used for correlation analysis. $P<0.050$ was considered as statistically significant.

Results

General data

There was no significant difference in age, gender, cardiac function classification, previous history (hypertension, diabetes), educational level, place of residence, smoking, and drinking between the two groups ($P>0.050$), proving the comparability of the two groups, as shown in **Table 1**.

Comparison of clinical efficacy

The comparison of clinical efficacy between the two groups showed that the total effective rate of the research group was 96.67%, which was notably higher than 69.05% of the control group ($P<0.001$). In addition, most of patients in the research group had marked response, while most of the patients in the control group had effective response, as shown in **Table 2**.

Comparison of incidence of complications

The incidence of complications in the two groups was analyzed. It was found that there was less incidence of complications in the two groups and no remarkable difference between the two groups ($P>0.050$), as shown in **Table 3**.

Quality of life evaluation

The quality of life score 3 months after discharge showed that the average score of the research group was (81.19±6.05), which was notably higher than that of the control group (77.49±5.64) ($P=0.002$), and all the scores were better in the research group than in the control group ($P<0.05$) (**Table 4**).

Comparison of survival conditions

Patients were followed up for two years. Through telephone, hospital reexamination and on-site follow-up, a total of 101 patients were successfully followed up, with a success rate of 99.02%. By comparing the survival conditions of the two groups, we found no significant difference in one-year survival rate ($P>0.050$), while the comparison of two-year follow-up

Table 1. Comparison of general data [n (%)]

	Research group (n=60)	Control group (n=42)	t or χ^2	P
Age (years)	59.43±6.41	58.67±6.12	0.600	0.550
Gender			0.755	0.385
Male	38 (63.33)	23 (54.76)		
Female	22 (36.67)	19 (45.24)		
Cardiac function classification			0.011	0.918
Grade II	18 (30.00)	13 (30.95)		
Grade III and above	42 (70.00)	29 (69.05)		
Previous history			0.083	0.774
With	26 (43.33)	17 (40.48)		
Without	34 (56.67)	25 (59.52)		
Education level			0.062	0.803
Below high school	20 (33.33)	15 (35.71)		
High school and above	40 (66.67)	27 (64.29)		
Place of residence			1.833	0.176
City	35 (58.33)	30 (71.43)		
Countryside	25 (41.67)	12 (28.57)		
Smoking			2.090	0.148
Yes	24 (40.00)	11 (26.19)		
No	36 (60.00)	31 (73.81)		
Drinking			0.223	0.637
Yes	16 (26.67)	13 (30.95)		
No	44 (73.33)	29 (69.05)		

Table 2. Comparison of clinical efficacy between the two groups [n (%)]

	Research group (n=60)	Control group (n=42)	χ^2	P
Marked response	46 (76.67)	11 (26.19)		
Effective response	12 (20.00)	18 (42.86)		
No response	2 (3.33)	13 (30.95)		
Total effective rate (%)	96.67	69.05	15.020	<0.001

revealed a higher survival rate in the research group than in the control group (P<0.050), as shown in **Table 5**.

Comparison of serum MMP-9 and IL-33 levels

There was no significant difference in serum MMP-9 and IL-33 levels between the two groups at T1 (P>0.050). The levels of MMP-9 and IL-33 in the research group were significantly higher than those in the control group at T2 and T3 (P<0.010), and there was no remarkable difference in the two levels in the research group at T1, T2 and T3 (P>0.050). The levels of MMP-9 and IL-33 in the control group were the highest at T1, lower at T2 than T1, and the lowest at T3 (P<0.050). As shown in **Figure 1**.

Correlation analysis between serum MMP-9 and IL-33

Through Pearson correlation analysis, it was found that the expression of MMP-9 and IL-33 was positively correlated (r=0.6139, P< 0.0001), i.e. IL-33 expression increased with the elevated expression of MMP-9. More details are shown in **Figure 2**.

Discussion

CHD is called ischemic heart disease, which is the most common type of organ diseases caused by atherosclerosis. The incidence of CHD has increased markedly in recent years and CHD is one of the major causes of death worldwide. It is reported that 7.3 million deaths occurred in 2013, accounting for one third of the total deaths, and has become one of the major diseases threatening human health and life [17, 18]. The main cause of CHD is vascular stenosis caused by coronary atherosclerosis, which gives rise to myocardial ischemia and hypoxia, thus causing organic injury endangering the life of patients. The predisposing factors include changeable and unchangeable risk factors, including hypertension and dyslipidemia, etc. The unchangeable risk factors include age and heredity, etc. [19]. Due to the acute onset and severe condition of CHD, once a patient was confirmed, effective treatment measures should be taken immediately. Patients with CHD often require continuous drug therapy, but the effect of drug therapy is limited. In recent years, with the continuous development and update of medical technology and equipment, coronary stent has become the main treatment

Table 3. Comparison of incidence of complications in the two groups [n (%)]

	Research group (n=60)	Control group (n=42)	χ^2	P
Coronary artery dissection	2 (3.33)	0 (0.00)		
Hypotension	4 (6.67)	1 (2.38)		
Arrhythmia	2 (3.33)	2 (4.76)		
Nausea and vomiting	1 (1.67)	4 (9.52)		
Incidence rate (%)	15.00	16.67	0.052	0.820

Table 4. Comparison of quality of life between the two groups

	Research group (n=60)	Control group (n=42)	t	P
Physical function	82.15±6.07	78.31±5.49	3.269	0.002
Cognitive function	83.42±5.58	81.01±5.04	2.233	0.028
Social activity function	78.33±6.34	75.25±6.03	2.463	0.016
Negative emotions	79.25±6.69	76.03±6.28	2.453	0.016
Recurrence of disease	83.47±5.36	78.31±5.19	4.847	<0.001
Pain	80.52±6.24	76.05±5.80	3.664	<0.001
Average score	81.19±6.05	77.49±5.64	3.125	0.002

Table 5. Comparison of survival conditions between the two groups [n (%)]

Group	Number of cases	One-year follow-up	Two-year follow-up
Research group	60	1 (1.67)	4 (6.67)
Control group	41	4 (9.76)	9 (21.95)
t		3.387	5.074
P		0.066	0.024

method for CHD. However, there is still some controversy on the efficacy and prognosis of this method. Coronary stent can open infarct-related arteries, reduce infarct size and residual stenosis, and preserve and improve myocardial function [20]. The team of Luo ei al [21], however, suggests that coronary stent intervention will lead to higher incidence and mortality of major adverse cardiovascular events. Another report by the team of Zhang [22] also pointed out that after pathological changes of myocardial ischemia or necrosis, coronary artery stenosis or occlusion before coronary stent intervention may seriously affect cardiac function, and postoperative restenosis and myocardial ischemia may further affect the long-term efficacy of the operation. Therefore, this study investigated the treatment and prognosis effects of drug and coronary stent intervention on patients with CHD. We have found

that the clinical efficacy of patients treated with coronary stent intervention was remarkably better than those in the control group who were treated with drugs only, suggesting that coronary stent intervention has higher efficacy for patients with CHD. In addition, by comparing the occurrence of complications between the two groups, the occurrence rate of complications was low in both groups, and there was no obvious difference between the two groups, which proves that stent is as safe as of drug therapy. The quality of life of patients was assessed three months after discharge, and the short-term and long-term prognosis was investigated after follow-up for two years. It was found that the quality of life score in the research group was notably higher than that in the control group, and the one-year survival rate of the prognosis was not remarkably different. According to our follow-up, the survival rate of patients in the research group was significantly better than that of the control group, indicating that coronary stent intervention therapy can notably improve the short-term and long-term prognosis of patients with CHD, as well as the survival. It is suggested that patients with multiple CHD can have better quality of life through coronary stent intervention therapy [23], which is consistent with the results of our study. Finally, the changes of serum MMP-9 and IL-33 levels before and after treatment in the two groups were compared. There was no significant difference between the two groups before treatment, while the levels of MMP-9 and IL-33 in the research group were considerably higher than those in the control group three days and seven days after treatment. Besides, there was no remarkable difference between the levels of MMP-9 and IL-33 in the research group before and after treatment, and the levels of the two in the control group were the highest before treatment and the lowest after seven days of treatment. After treatment, MMP-9 and IL-33 levels were found to be positively correlated, and IL-33 levels increased with the increase of MMP-9. A large number of bioactive proteins, including matrix metallopro-
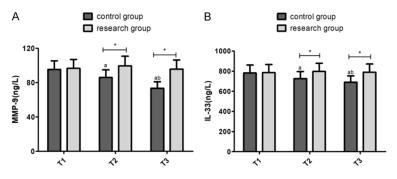
Figure 1. Comparison of MMP-9 and IL-33 levels at different time points. A. Comparison of serum MMP-9 levels. B. Comparison of serum IL-33 level. a denotes comparison with T1, P<0.050, b denotes comparison with T2, P<0.050; *denotes P<0.050.

Figure 2. Correlation analysis of serum MMP-9 and IL-33 levels in research group after treatment.

teinases (MMPs) and inflammatory factors, were found in vulnerable and unstable plaques [24]. MMPs are endopeptidase that are known to play key roles in the process of extracellular matrix decomposition. MMP-9 can affect plaque stability and increase the recurrence rate of cardiovascular events such as left ventricular enlargement [25]. It is reported that inflammatory factors can promote the formation of lipid plaques, and CHD is actually a chronic inflammatory response [26]. Among inflammatory factors, IL-33 is a member of IL-1 superfamily [27]. Early studies show that [28] IL-33 can cause and regulate a variety of inflammatory reactions and may be effective in chronic inflammation and autoimmune diseases. In the report of Gungor O et al. [29], it is also believed that the existence of IL-33 is related to the increased risk of vascular injury and cardiovascular events. In this experiment, both MMP-9 and inflammatory factor IL-33 increased

to different degrees after coronary stent intervention therapy, suggesting that the release of inflammatory factor is promoted due to injury of vascular endothelium and plaque extrusion during the operation process.

There are still some deficiencies in this study due to limited sample size within designated population which need to be further investigated in different races in the future.

To sum up, coronary stent interventional therapy has better clinical efficacy and short-term and long-term prognosis for CHD patients than drug therapy alone, but it is easy to induce inflammatory reaction after surgery. Therefore, attention should be paid to the control of inflammation before and after treatment.
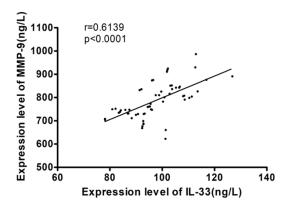
Disclosure of conflict of interest

None.

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