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A study of the clinical impact of different heart rate control management in patients with STEMI after percutaneous coronary intervention

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Abstract

Post-PCI STEMI patients may suffer from reduced perfusion capacity and myocardial injury, and few existing studies have focused on the role of heart rate control management in the clinical impact of post-PCI. In this paper, we first selected post-PCI STEMI patients as an example, given specific inclusion and exclusion criteria, and conducted controlled experiments through heart rate stability testing with different heart rate control administration programs. Secondly, linear regression combined with the SCAD penalty function was utilized for the screening of clinical impact variables in post-PCI STEMI patients. Finally, the clinical response, TIMI flow classification and myocardial injury of STEMI patients after PCI were analyzed, and the clinical influencing factors of STEMI patients were fully explored by combining ROC curve with linear regression. The results showed that the total effective rate of clinical efficacy in the MACE group was 95.65%, the significance test result of TIMI flow classification was 0.012, and myocardial injury indexes showed significant differences at 1% level after different stages of the beta-lactam dosing regimen. The predictive sensitivity in the MACE group was 95.29%, and its 95% confidence interval was 0.882~0.945. The number of high risks in the linear regression decreased almost 9-fold when the threshold of $LogP$ was increased from 0 to 0.4. The management of heart rate control in post-PCI STEMI patients can be effectively achieved by different stages of the beta-lactam dosing regimen, and the negative impact on STEMI patients after PCI can be reduced.

Keywords: Linear regression; SCAD penalty function; Sensitivity; PCI; STEMI patients. **AMS 2010 codes:** 90B06

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1 Introduction

Acute myocardial infarction has become one of the common cardiovascular diseases that seriously threaten the physical and mental health of human beings and the safety of life, and it is an acute necrosis of the myocardium caused by persistent and severe myocardial ischemia. According to whether the ST-segment elevation on the electrocardiogram is elevated or not, it can be divided into two categories: acute ST-segment elevation myocardial infarction and acute non-ST-segment elevation myocardial infarction [1]. ST-segment elevation myocardial infarction can undergo rapid changes in a short period of time, and it has a high mortality rate. Percutaneous coronary intervention (PCI) is the main method of clinical treatment of STEMI. PCI has the advantages of a high recanalization rate and low bleeding risk and is more and more widely used in the clinic at this stage [2].

PCI can rapidly open the infarcted vessels of STEMI patients, restore ischemic myocardial blood perfusion, and reduce myocardial injury, but the risk of postoperative adverse cardiovascular events is still high, and the reperfusion therapy is accompanied by a series of reperfusion injuries while improving the myocardial blood supply [3]. For example, reperfusion arrhythmias, cardiomyocyte metabolic disorders and microvascular structural damage and dysfunction significantly increase the risk of major adverse cardiovascular events such as recurrent myocardial infarction, heart failure, and sudden cardiac death [4]. Therefore, effective management of heart rate control in patients with myocardial infarction treated with PCI is necessary to help rehabilitate the care of STEMI patients after PCI.

At present, a number of studies have explored the association between the level of TGRLs and poor prognosis in patients with STEMI after PCI, but few studies have focused on its clinical impact on patients with STEMI undergoing percutaneous coronary intervention with different heart rate control. In this paper, we started with the selection of the study population and set up different scenarios of post-PCI STEMI patients as well as a control group, given the clinical diagnostic requirements for STEMI. Based on the description of different beta-lactam dosing regimens for heart rate stability testing, the evaluation criteria of heart rate stability were given, and linear regression analysis with SCAD penalty function was introduced to screen the clinical impact variables of post-PCI STEMI patients to further optimize the clinical response of patients under heart rate control. In order to verify the effectiveness of the methods given in this paper in practical application, the clinical efficacy, TIMI flow classification, and myocardial injury were analyzed for different bethanechol dosing regimens, and the clinical responses of patients under different heart rate control were also analyzed using ROC curves as well as linear regression. Different heart rate control management can help STEMI patients after PCI to reduce the occurrence of blood flow reperfusion, reduce myocardial injury, and promote the rapid recovery of STEMI patients.

2 Review of relevant research work

Acute ST-segment elevation myocardial infarction (SETMI) is an important form of coronary atherosclerosis and is the primary cause of death in people with coronary heart disease. The emergency percutaneous coronary intervention has been developing in recent years, and breakthroughs have been made for the treatment of acute SETMI, which has largely reduced its disability and fatality rates [5-6]. Liang, Z. et al. analyzed a randomized trial of bivalirudin versus heparin in the treatment of STEMI patients with PCI and concluded that the acceptance level of patients with STEMI under different drug treatments was different. A new round of randomized controlled trials was conducted in 87 clinical centers in China, based on STEMI patients receiving PCI radial artery fibrinolytic therapy, random allocation of glycoprotein inhibitors, and heparin alone,

it can be clearly found that the degree of impact of different drug treatments on STEMI patients is also different [7]. Zahler, D., et al. used 2,345 first-time post-PCI treatment patients as an example to analyze the time delay correlation between symptom onset and balloon inflation in STEMI patients. The time to experience balloon pain in STEMI patients can be verified by post-PCI, which reduces the mortality rate of myocardial infarction to a certain extent [8]. Das, A. et al. discussed the role of cDTI in the in vivo characterization of myocardial microstructure and the restorative nature of the left ventricular ejection fraction in the course of myocardial infarction episodes. The application of cDTI biomarkers to post-STEMI patients promotes the remodeling of the patient's left ventricle, reduces the damage caused to STEMI patients after reperfusion, and, to some extent, improves their postoperative survival [9]. Hafshejani, N. J. et al. combined physiological techniques with diagnostic ECG databases to analyze the data on cardiac signals of 52 cases of healthy diseases. The lead ECG waves during heart failure consist of T and Q waves as well as ST segments, and acute changes in the ST segments lead to the onset of acute myocardial infarction disease [10].

Percutaneous coronary intervention is one of the effective methods of clinical treatment of myocardial infarction at present, but there is also the risk of coronary artery restenosis and ischemia after the operation. At present, after carrying out PCI, clinicians usually ask post-PCI patients to rest in bed for two days before they can start their activities as a way to better help the post-PCI patients recover their health [11-12]. Chimed, S. et al. analyzed the changes in right ventricular function and size in patients with the changes in right ventricular function and size in patients with STEMI analyzed, and there may be some prognostic implications of ventricular remodeling in patients undergoing PCI for the first time. The optimal threshold for ventricular remodeling in STEMI patients was defined as 240%, and validation was carried out using 2280 ming STEMI patients after initial PCI [13]. Marcos, G. V. et al. analyzed the importance of carrying out cardiac magnetic resonance prognostic value data in STEMI patients and quantitatively investigated this by using 247 ming STEMI patients aged 70 years as an example. Based on the cardiac MRI results of elderly STEMI patients after PCI, the prognostic value of PCI can be more clearly defined, and the role of PCI in the treatment of STEMI can be further expanded [14]. Park, S. et al. In order to analyze the clinical outcomes of primary PCI for STEMI and MVD with immediate hemodynamic revascularization, an example study was carried out using 248 patients. Immediate revascularization may have certain lesions during the initial procedure, while PCI for STEMI can effectively reduce the incidence of recurrent myocardial infarction and has superior therapeutic ability compared to revascularization [15]. Hu, M. et al. analyzed the non-cause-specific mortality rate of patients with right ventricular myocardial infarction of the upper ST-segment over two years, using 9,308 patients with STEMI as an example. Through the data, it was shown that the risk of death that mainly affects the patients with STEMI in the upper ST segment is a combination of hemodialysis, stroke, and hemorrhage, while there is no significant difference in the patients with STEMI in the lower ST segment [16].

3 Information and methodology

3.1 General information

A total of 203 STEMI patients admitted to the First People's Hospital of L City, S Province, who underwent PCI from April 2021 to April 2023 were selected as the study subjects and were categorized into the MACE group ($n=69$) and the non-MACE group ($n=134$) on the basis of whether or not MACE occurred. In addition, 60 cases were selected as the control group, who were examined in the hospital at the same time and confirmed to have no history of AMI and acute and chronic infections. A comparison of the general data of the three groups is shown in Table 1. The general data of the three groups were similar, and there were no significant differences or statistical significance. The Medical Ethics Committee gave its approval to this study.

Group	Number	Age	Sex (Male/Female)		
MACE	69	62.12 ± 11.13		50/19	
Non-MACE	134	63.05 ± 10.11		99/35	
Control group	60	62.37 ± 10.68	38/22		
F / χ^2		0.655	0.529		
P		0.523	0.761		
Group	Hypertension	Diabetes mellitus	Smoke	Drink	Baric index
MACE	29(42.03%)	20(28.99%)	42(60.87%)	$11(15.94\%)$	25.86 ± 2.71
Non-MACE	83(61.94%)	62(46.27%)	63(47.01%)	32(23.8%)	25.37 ± 2.62
Control group	18(30.00%)	12(20.00%)	$11(18.33\%)$	8(13.33%)	25.05 ± 2.69
F / χ^2	3.559	1.762	3.792	0.454	1.689
P	0.168	0.411	0.156	0.786	0.187

Table 1. Comparison of the three groups of general data

The inclusion criteria were as follows:

- 1) Compliance with the diagnostic criteria of the Diagnostic and Therapeutic Guidelines for Acute Myocardial Infarction and indication for PCI treatment.
- 2) Admitted to the hospital within 8 hours of AMI onset.
- 3) Age 20-85 years old, and all patients received follow-up for 1 year after surgery.
- 4) Subjects or family members voluntarily signed the informed consent.

The exclusion criteria were as follows:

- 1) Combination of severe arrhythmia, acute and chronic heart failure, recent infection and other serious diseases.
- 2) Old myocardial infarction or history of previous PCI treatment.
- 3) Recent application of glucocorticoids or immunosuppressants.

The clinical diagnosis of STEMI needs to fulfill at least 2 of the following 3 points, namely:

- 1) ST-segment elevation occurs in more than two consecutively appearing leads on the electrocardiogram.
- 2) Proportionate elevation of laboratory cardiac biomarkers, i.e., troponin, cardiac enzymatic markers.
- 3) Typical angina symptoms.

STEMI can be clinically diagnosed in patients with both clinically compatible troponin elevation and elevation of the ST leads on the ECG.

3.2 Heart rate stability testing

1) Detection of heart rate stability

The patient's postoperative ECG data were imported from the MUSE central monitoring system into the MARS ECG analysis system of General Electric GE, and after the interference was eliminated, at least two physicians from the Department of Critical Care Medicine of the First People's Hospital of L City, S Province, reviewed the generated ECG templates. Including sinus tachycardia, sinus bradycardia, atrial period contraction, ventricular period contraction, atrial fibrillation, ventricular fibrillation, and atrioventricular block, etc., and the electrocardiographic templates were used to make machine-autonomous judgments on the electrocardiographic data and finally summarized.

2) Evaluation criteria for heart rate stability

Heart rate stability includes the number of total heartbeats, the number of premature ventricular beats, the percentage of premature ventricular beats, single premature ventricular beats, and paired premature ventricular beats. The ECG interpretation criteria for premature ventricular beats were that the ECG could detect a cluster of QRS waves occurring early after the sinus P wave, the overall duration of the cluster was more than 0.15 seconds, and the shape of the cluster was a wide aberration that was completely different from the normal QRS waveform, and the direction of the T wave and the ST segment was the opposite of the direction of the main wave in front of it. The similarity of QRS waves was considered as a monomorphic premature ventricular beat, and the difference of QRS waves was considered as a polymorphic premature ventricular beat. The QRS waves are similar for monomorphic premature ventricular beats and different for polymorphic premature ventricular beats. Paired premature ventricular beats are two consecutive premature ventricular beats [17]. The machine interpreted all of the above results and then manually reviewed them, and the samples with possible abnormal results in the frequency of premature ventricular beats were reviewed again.

3) Dosing regimens in different phases

There were two phases in this study. The first phase was from April 2021 to April 2022, during which a single oral bethanechol dose of ≤80 mg and a cumulative total of ≤120 mg on the same day was administered to a total of 150 patients. Table 2 shows the bethanechol dosing regimen for the first phase based on basal heart rate.

Groups	Single dose of drug	Drug reduction	Addressing situation	
$<$ 50 b pm			1. First 5 cases: $12.0mg$	
	0 _{mg}		$2.HRV \ge 5pm$: 12.0mg	
		1. HR s38bpm: 12.0mg	1.HRV \geq 5bpm: 12.0~24.5mg	
$50-$	30mg	2. $HRV \leq 4bpm$: 12.0mg	2. People who have anxiety and fear of emotions:	
65bpm		3. Self-service: 12.0mg	24.5mg	
$65-$ 75bpm		1.HR \leq 67bpm: 12.0mg	1.HRV \geq 5bpm: 12.0~24.5mg	
	55mg	$2.HRV \leq 3$ bpm: 12.0mg		
		3. Self-service: 24.5×50 mg	2. People who have anxiety and fear of emotions: 24.5mg	
		4. Hypopiesia: 24.5mg		
		1. Self-service: $24.5 \sim 50$ mg	1.HRV \geq 5bpm: 24.5mg	
$75-$ 85bpm	70mg		$2.HR \geq 80$ bpm: 24.5mg	
		2.Hypopiesia: 24.5mg	3. People who have anxiety and fear of emotions:	
			24.5mg	
\geq 85bpm		1. Self-service: $24.5 \sim 50$ mg		
	120mg	2.Hypopiesia: 24.5mg		

Table 2. Phase 1 betalk dosing regimen

The second phase was from May 2022 to April 2023, and Table 3 shows the second phase bethanechol dosing regimen, with a single oral bethanechol dose of ≥80 mg when the patient's HR was ≥60 bpm, and a cumulative total of \leq 180 mg on the same day, for a total of 200 patients.

Groups	Single dose of drug	Drug reduction	Addressing situation		
$<$ 50bpm	0 _{mg}		HRV≥5bpm, 12.0~24.5mg		
$50-$ 65bpm		1. HR <58bpm: 12.0mg	1.HRV \geq 5bpm: 24.5mg		
	55mg	2. Self-service: 24.5 mg	2. People who have anxiety and fear of emotions: 24.5mg		
		3. Hypopiesia: 24.5 mg			
$65 -$ 75bpm	110mg	1. Self-service: 24.5 mg	1.HRV \geq 5bpm: 12.0~24.5mg		
		2.Hypopiesia: 24.5mg	2. People who have anxiety and fear of emotions: $24.5 - 50$ mg		
		3. Bealk sensitive: 24.5×50 mg	3. Betak tolerant: 24.5 mg		
$75-$	140mg 85bpm	1. Self-service: $24.5 \sim 50$ mg	1.HRV \geq 5bpm, 24.5~50mg		
		2.Hypopiesia: 24.5mg	2. When patients have anxiety and fear, 24.5-50mg		
\geq 85bpm		1. Self-service: $24.5 \sim 50$ mg	--		
	180mg	2.Hypopiesia: 24.5mg			

Table 3. Phase 2 betalk dosing regimen

3.3 Linear regression analysis

1) Fundamentals of linear regression

The LS algorithm is commonly applied to solve unconstrained optimization problems and can also be used to fit curves. The main core idea of the LS algorithm is to utilize the definition of the unknown parameter θ , while the final frequency estimate is generated after several iterations, then:

$$
E = \sum_{i=0}^{n} e_i^2 = \sum_{i=0}^{n} (y_i - \hat{y}_i)
$$
 (1)

Where y_i denotes the actual value and \hat{y}_i denotes the corresponding estimated value. An example of the LS algorithm is shown in Fig. 1, where the red color denotes the sample points, the blue straight line denotes the best solution matched by the LS algorithm, and the green color denotes the error interval, and there are four data points in the figure [18].

Figure 1. Example of the LS algorithm

Linear regression can effectively characterize the clinical reflections of STEMI patients after PCI under different heart rate control management and provide a basis for further analysis of different heart rate control management to accelerate the health recovery of STEMI patients after PCI.

In linear regression, the loss function can generally be chosen as the mean square error, and in the LS algorithm, the mean square error can be thought of as dividing by *m* . Therefore, the optimal solution obtained by the LS algorithm is derived using the mean square error as the loss function. For the feature samples in the figure, the fitting function can be written as $h_\theta(x) = \theta_0 + \theta_1 x$, and the loss function is:

$$
J(\theta_0, \theta_1) = \sum_{i=0}^{m} (y_i - h_{\theta}(x_i))^2 = \sum_{i=0}^{m} (y_i - \theta_0 - \theta_1 x_i)^2
$$
 (2)

Where i is the i nd sample point. In order to minimize the loss function, the loss function can be considered as a multivariate function, which is calculated by the partial derivative of the multivariate function. $J(\theta_0, \theta_1)$ separately for θ_0, θ_1 and make its partial derivative equal to 0 to get:

$$
\frac{\partial J(\theta_0, \theta_1)}{\partial \theta_0} = -2 \sum_{i=1}^m (y_i - \theta_0 - \theta_1 x_i) = 0
$$
\n(3)

$$
\frac{\partial J(\theta_0, \theta_1)}{\partial \theta_1} = -2 \sum_{i=1}^m (y_i - \theta_0 - \theta_1 x_i) x_i = 0
$$
\n(4)

Joining the two equations and solving them gives:

$$
\theta_0 = \frac{\sum_{i=1}^{m} x_i^2 \sum_{i=1}^{m} y_i - \sum_{i=1}^{m} x_i \sum_{i=1}^{m} x_i y_i}{m \sum_{i=1}^{m} x_i^2 - \sum_{i=1}^{m} x_i \left(\sum_{i=1}^{m} x_i\right)^2}
$$
(5)

$$
\theta_{1} = \frac{m \sum_{i=1}^{m} x_{i} y_{i} - \sum_{i=1}^{m} x_{i} \sum_{i=1}^{m} y_{i}}{m \sum_{i=1}^{m} x_{i}^{2} - \sum_{i=1}^{m} x_{i} \left(\sum_{i=1}^{m} x_{i}\right)^{2}}
$$
(6)

For the example in Fig. 1, the values of θ_0 and θ_1 are obtained by substituting equations (5) and (6) for the calculation. This solution is still applicable to samples with *ⁿ* -dimensional features. For feature dimension (x_1, x_2, \dots, x_n) , it is necessary to add a 0th dimension $x_0 = 1$, at which point the augmented feature vector can be expressed as $x = (x_0, x_1, \dots, x_n)$ and the augmented weight vector as $\theta = (\theta_0, \theta_1, \dots, \theta_n)$. The final fit function becomes:

$$
h_{\theta}(x) = \sum_{i=0}^{n} \theta_i x_i = \theta_0 + \theta_1 x_1 + \dots + \theta_n x_n
$$
\n⁽⁷⁾

The loss function becomes:

$$
J(\theta) = \sum_{j=1}^{m} (h_{\theta}(x_j) - y_j)^2 = \sum_{j=1}^{m} \left(\sum_{i=0}^{n} \theta_i x_{ij} - y_j \right)^2
$$
 (8)

The loss function $J(\theta)$ is obtained by taking the partial derivative of θ_i ($i = 0, 1, \dots, n$), respectively:

$$
\frac{\partial J(\theta)}{\partial \theta_i} = 2 \sum_{j=1}^m \left(h_\theta \left(x_j \right) - y_j \right) x_j = 2 \sum_{j=1}^m \left(\sum_{i=0}^n \theta_i x_{ij} - y_j \right) x_j, i = 0, 1, \cdots, n \tag{9}
$$

Making the partial derivative equal to 0 gives:

$$
2\sum_{j=1}^{m} \left(\sum_{i=0}^{n} \theta_i x_{ij} - y_j \right) x_j = 0, i = 0, 1, \cdots, n
$$
 (10)

The final result obtained is a system of linear equations containing $n+1$ unknown quantity and $n+1$ equations, which can be solved by using the Gaussian elimination method $\theta_i(i=0,1,\dots,n)$. For the problem of linear regression, the eigenspace can be transformed according to the form of the fitted function, i.e. generalized linear regression.

2) Selection of Penalty Function

The SCAD method is the first penalty function to satisfy the Oracle property, and it is also a method for variable selection based on compression [19]. Unlike previous methods, its penalty function is not based on penalization for the following estimation:

$$
\hat{\beta} = \arg\min_{\beta} \left\| y - \sum_{j=1}^{p} x_j \beta_j \right\|_2^2 + \sum_{j=1}^{p} p_{\lambda, \gamma} \left(\left| \beta_j \right| \right) \tag{11}
$$

The first order derivative of its penalty function $J_{\lambda}(\theta)$ is defined as follows:

$$
p'_{\lambda,\gamma}(\beta_j;\gamma) = \lambda \left\{ I\left(\beta_j \leq \lambda\right) + \frac{\left(\gamma \lambda - \beta_j\right)_+}{\left(\gamma - 1\right)\lambda} \left(\beta_j > \lambda\right) \right\} \tag{12}
$$

where $\gamma > 2$.

The SCAD penalty improves the properties of the Lass penalty function by allowing large β 's to not be over-penalized and by ensuring continuity in the set of solutions. When the design matrix is orthogonal, the solution of Eq. (11) is:

$$
\hat{\beta} = \begin{cases}\n\operatorname{sgn}(z)(|z| - \lambda)_+, & |z| \le 2\lambda \\
\{(\gamma - 1)z - \operatorname{sgn}(z) \gamma \lambda \} / (\gamma - 2), & 2\lambda < |z| \le \gamma \lambda. \\
z, & |z| > \gamma \lambda\n\end{cases}
$$
\n(13)

It can be seen that the penalty function has two unknown parameters λ and γ , which can generally be chosen appropriately based on certain criteria, such as cross-testing.

Let $\beta_0 = (\beta_1, \dots, \beta_p)^T = (\beta_{10}^T, \beta_{20}^T)$, without loss of generality, assume $\beta_{20} = 0$. $I(\beta_0)$ denotes the fisher information matrix, and $I_1(\beta_{10},0)$ denotes the fisher information matrix for which $\beta_{20} = 0$ is known. It can be shown that when $\lambda_n \to 0$, the parameter estimates under the SCAD penalty converge at a 1 O_p $\left\lfloor n \right\rfloor^2 + a_n$ $\begin{pmatrix} 1 & \frac{1}{2} & \frac{$ $\left| n^{2}+a_{n} \right|$ $\begin{pmatrix} 1 & 1 \\ 1 & 1 \end{pmatrix}$ rate. Moreover, a \sqrt{n} consistent estimator can be obtained when $n^{1/2}$ $n^{1/2}\lambda_n \to \infty$ the asymptotic normal distribution is satisfied β_2 $\hat{\beta}_2 = 0$ as well as $\hat{\beta}_1$ $\hat{\beta}_1$ when the asymptotic normal distribution is satisfied.

4 Results and analysis

Based on the methods of different heart rate control management and linear regression analysis given in the previous article, the clinical efficacy of post-PCI STEMI patients under different heart rate control management was statistically analyzed by SPSS software, which provided the basis for further clarifying the clinical influencing factors of post-PCI STEMI patients.

4.1 Clinical response of patients after PCI

1) Clinical efficacy

Based on the MACE group, non-MACE group and control group given in the previous section, the clinical efficacy of different heart rate control management methods in different groups for STEMI patients after PCI was investigated during a 2-year follow-up visit. The results of the clinical efficacy comparison among the three groups are shown in Table 4.

From the MACE group, non-MACE group and the control group, different heart rate control and different stages of drug administration will have a greater impact on the PCI patients, and its clinical efficacy in the MACE group is 95.65%, while the non-MACE group's effective rate is only 79.10%, and the control group's effective rate is only 50%. The use of different stages of drug delivery for post-PCI STEMI patients can effectively inhibit the effects of post-PCI on STEMI patients and enhance the recovery ability of STEMI patients.

Table 4. Results of comparative clinical outcomes

2) TIMI blood flow grading

Myocardial perfusion was evaluated in patients in the MACE and non-MACE groups immediately after PCI. Grade 0 indicated the absence of antegrade flow beyond the occlusion, grade I the presence of weak antegrade flow, grade II the presence of slow or delayed antegrade flow with complete filling of the distal vascular bed, and grade III normal antegrade flow. The results of TIMI flow grading in

STEMI patients after PCI in both groups after different heart rate control management are shown in Table 5.

The TIMI flow grading in the MACE group was better than that in the non-MACE group after different stages of drug administration in STEMI patients after PCI, with a Z-value of 2.968 and a significance test of 0.012<0.05. By controlling STEMI patients after PCI with drug administration, myocardial perfusion can be realized and grasped. The control of post-PCI STEMI patients through the administration of a drug regimen can realize the control of myocardial perfusion so as to better manage heart rate, and the clinical efficacy is accurate.

Tuble of the third close how choshipation results							
Group	Level 0	Level I	Level II	Level III			
MACE	$0(0.00\%)$	$6(8.70\%)$	14(20.29%)	49(71.01%)			
Non-NACE	5(3.73%)	26(19.40%)	39(29.10%)	64(47.77%)			
	$- -$	$- -$	$- -$	2.968			
	$- -$	--	$- -$	0.012			

Table 5. The TIMI blood flow classification results

3) Analysis of myocardial injury indicators

10mL of venous blood was collected from STEMI patients before and 36h after PCI, serum was separated after centrifugation, and the levels of patients' creatine kinase isoenzyme (CK-MB), cardiac troponin Ⅰ (cTnⅠ), and B-type brain natriuretic peptide (BNP) were measured by immunoassay analyzer, and the reagent kits were all purchased from DRG, and the operation was carried out in strict accordance with the instructions. The comparative results of myocardial injury in the two groups are shown in Table 6.

Before treatment, there was no significant difference in the levels of serum CK-MB, cTnI and BNP between the two groups (P>0.05), and after treatment, the STEMI patients in the MACE group were lower than those in the non-MACE group, with P values less than 0.01, which indicated that there was a very significant difference in myocardial injury between the two groups of patients before and after treatment. Betalucel can significantly reduce the myocardial injury of STEMI patients after PCI, and the reason may be related to the fact that betalucel has strong specificity, acts only on the localization of thrombus, effectively avoids activation of blood fibrinogen, and has little effect on the function of the body's fibrinolytic system. The different stages of the bethanechol administration program can enhance the myocardial injury of STEMI patients after PCI and effectively control their heart rate.

Group	$CK-MB$ (U/L)		cTnI $(\mu g/L)$		BNP (pg/ml)	
	Before	After	Before	After	Before	After
MACE	50.21 ± 8.13	$20.19 + 5.76$	6.92 ± 1.33	$1.62+0.43$	2391.38 + 386.52	572.54 ± 128.05
Non-NACE	51.16 ± 8.02	29.86 ± 6.02	7.01 ± 1.36	2.41 ± 0.54	$2435.62 + 361.34$	998.35 ± 149.41
т	0.557	6.912	0.892	7.359	0.578	14.132
D	0.568	0.003	0.384	0.001	0.593	0.002

Table 6. Comparison of myocardial injury

4.2 Post-PCI ROC curve analysis

Utilizing the ROC curve to target the predictive value of bethanechol on MACE in post-PCI STEMI patients can better help clinicians understand the specific role of bethanechol on heart rate control in patients so as to better carry out postoperative heart rate management in post-PCI-STEMI patients. Serum CK-MB, cTnI and BNP levels were also introduced to analyze the sensitivity and specificity of bethanechol on MACE in post-PCI STEMI patients, and the results are shown in Figure 2.

The ROC curve analysis showed that the areas under the curve of serum CK-MB, cTnI, and BNP were 0.781, 0.826, and 0.734, respectively. The difference between the three indicators was not statistically significant (p $>$ 0.05), indicating that the heart rate control effect of STEMI patients after PCI was more average under the three indicators. The three indicators would also respond to the heart rate control situation of STEMI patients to a certain extent. The three indexes will have a certain effect on the heart rate control of STEMI patients. The sensitivity and specificity of the combined diagnosis of MACE was 95.29%, the specificity was 83.64%, the AUC area was 0.913, and the 95% confidence interval was 0.882-0.945. The sensitivity and specificity of the three indexes in predicting STEMI patients can provide more evidence-based evidence for the application of bethanechol in post-PCI STEMI patients.

Figure 2. ROC trace analysis

4.3 Post-PCI linear regression analysis

Linear regression was applied to screen the variables related to clinical influencing factors in patients with STEMI after PCI under different heart rate control management, and multifactorial logistic regression was performed to determine the final predictor variables and the specific regression results are shown in Table 7.

From the results of linear regression analysis, the risk factors for the occurrence of early ventricular rhythm market in STEMI patients after PCI were Killip classification, blood potassium level, white blood cell count, random blood glucose level, and creatinine level, and there was no multicollinearity among the risk factors. The above variables were assigned values in order, i.e., Killip classification $(X1)$, blood potassium level > 3.5 mmo/L $(X2)$, white blood cell count $> 10.0*109/L$ (X3), random blood glucose > 8.0 mmol/LX4), and blood creatinine > 80.0 µmol/L (X5). The resulting regression equation was:

$$
Log P = -0.694 + 0.714 * X1 - 2.596 * X2 + 0.738 * X3 + 0.751 * X4 + 0.618 * X5
$$
 (14)

Based on the results of the regression equation and linear regression analysis, the significance test results of Killip classification, blood potassium level, white blood cell count, random blood glucose level and creatinine level were all less than 0.05, of which the significance of Killip classification and

blood potassium level showed a very significant difference at the 1% level. The analysis of ventricular arrhythmia in early infarction can be effectively carried out by log-linear regression, which provides effective technical support for further mastering the development of heart rate in STEMI patients after PCI.

Table 7. Kisk factors of ventricular armythminas						
Variables	Ventricular arrhythmias					
	B	OR	95%CI	P		
Killip Grade	0.731	2.075	[1.385, 3.116]	0.001		
Serum potassium (mmol/L) >4.0	-2.589	0.074	[0.031, 0.168]	0.002		
WBC count $(10^9/L) > 12.0$	0.732	2.101	[1.112, 3.925]	0.018		
Glucose ($mmol/L$)>9.0	0.756	2.125	[1.168, 3.897]	0.012		
Creatinine (μ mol/L $>$ 90.0	0.627	1.856	[0.872, 1.029]	0.043		
Constant term	-0.695	0.503		0.451		

Table 7. Risk factors of ventricular arrhythmias

R software and Free Statistics software were applied to the risk factor model derived from the above regression, and the clinical impact curve was further plotted based on the model, as shown in Figure 3.

Based on the results of the clinical impact curves from the risk model, the predicted curve of the regression equation *LogP* coincides with the actual curve as the high-risk threshold is increased, which can assist clinicians in providing a net benefit to STEMI patients after PCI, and thus better control the heart rate of STEMI patients. In particular, when the threshold value of *LogP* was increased from 0 to 0.4, the number of high-risk decreased by nearly 9 times. The use of linear regression can fully explore the clinical influence of heart rate-related factors in STEMI patients after PCI, and provide effective help to further control the heart rate of STEMI patients.

Figure 3. Clinical impact curve of the risk model

5 Conclusion

STEMI is myocardial necrosis caused by severe and persistent acute ischemia of the corresponding myocardium due to the occurrence of sharp reduction or interruption of coronary blood supply on the basis of coronary artery lesions. And percutaneous coronary intervention needle therapy can effectively inhibit the myocardial blood flow situation of STEMI patients and reduce the lethality of STEMI patients. In this paper, taking the STEMI patients in the First People's Hospital of L City, S Province, as a research example, regression analysis was conducted to analyze the factors influencing the clinical risk of STEMI patients after PCI under the management of affecting different heart rate control by heart rate stability test combined with different stages of the drug administration program. The conclusions were drawn as follows:

- 1) The clinical efficacy under different bethanechol dosing regimens was 95.65% in the MACE group, while it was only 79.10% in the non-MACE group and even 50% in the control group. The control of STEMI patients after PCI through the drug administration program can realize the grasp of myocardial blood flow perfusion so as to better manage the heart rate and ensure the clinical efficacy is accurate.
- 2) ROC curve analysis showed that the areas under the curve of serum CK-MB, cTnI, and BNP were 0.781, 0.826, and 0.734, respectively, and the differences between the three indicators were not statistically significant. It indicates that the heart rate control effect of STEMI patients after PCI under the three indicators is more average, and the three indicators will also reflect the heart rate control of STEMI patients to a certain extent.
- 3) Based on the results of the clinical impact curves obtained from the risk model, with the increase of the high-risk threshold, the predicted curve of the above regression equation *LogP* basically coincides with the trend of the actual curve, which can assist clinicians in bringing a net benefit to patients with STEMI after PCI, so as to better grasp the control of heart rate in patients with STEMI.

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