Discussion on the Treatment Methods and Value of the No-Reflow Phenomenon During Percutaneous Coronary Intervention

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Abstract: Objective: To explore the treatment methods and value of the no-reflow phenomenon during percutaneous coronary intervention (PCI). Methods: 180 patients with acute myocardial infarction (AMI) who underwent PCI treatment at the First Hospital of Hebei Medical University from November 2020 to December 2022 were selected and divided into Group A, Group B, and Group C by random number table extraction method, each group included 60 cases. Group A received intracoronary injection of recombinant human urokinase and Group B received intracoronary injection of diltiazem. Group C adopted a targeted drug administration strategy of “front and back pinch method,” where targeted thrombolysis was performed in the target vessel before the stent was released to quickly dissolve the residual thrombus near the lesion and the small distal thrombus; recombinant human urokinase was subsequently injected in the targeted vessel. The incidence of cardiovascular events, cardiac function, and quality of life of the three groups were analyzed. Results: After the intervention, the incidence rates of angina pectoris, heart failure, and recurrent myocardial infarction in Group C were lower than those in Group B (P < 0.05); the overall incidence rate was the lowest in Group C, followed by Group A and Group B; after intervention, left ventricular end-diastolic diameter, end-systolic diameter, and ejection fraction in Groups A and C were all better than those of group B (P < 0.05), and Group C was better than Group A; after intervention, the scores of different dimensions of quality of life in Groups A and C were higher than those of Group B (P < 0.05). Conclusion: AMI patients are prone to the no-reflow phenomenon after PCI treatment. Using recombinant human urokinase to complete the “front and back pinch method” can effectively improve the no-reflow phenomenon, reduce the incidence of cardiovascular events, optimize cardiac function, and improve the patient’s quality of life and survival rate. Keywords: Percutaneous coronary intervention; Acute myocardial infarction; No-reflow phenomenon; Recombinant human urokinase

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1. Introduction

Acute myocardial infarction (AMI) is a disease caused by the sudden interruption of coronary blood flow leading to myocardial ischemia and necrosis. The common cause of the disease is thrombosis or vasospasm...
caused by coronary atherosclerosis, resulting in insufficient myocardial blood supply [1]. Symptoms such as severe chest pain, dyspnea, nausea, vomiting, and other related symptoms may occur after the onset of the illness. The occurrence of AMI can increase the incidence of complications such as heart failure and arrhythmia, increasing the risks faced by individuals [2]. Currently, percutaneous coronary intervention (PCI) is mostly used to treat AMI. The implementation of this method can help reduce patient mortality and disability rates. However, based on the clinical situation, slow blood flow, no-reflow, or other abnormal angiographic results may occur during stent implantation. No-reflow refers to a state of myocardial tissue hypoperfusion after the coronary blood vessels have been opened [3]. The existence of no-reflow or slow blood flow can directly affect myocardial horizontal perfusion, causing further damage to the myocardium and impairing cardiac function [4]. In response to this situation, certain clinical measures are taken to prevent and treat the no-reflow phenomenon. The use of suction catheters and distal protection devices, vasodilator drugs (nitroglycerin, sodium nitroprusside, adenosine, anisodamine, etc.), and antiplatelets drugs (tirofiban, etc.) can reduce the incidence of no-reflow, but the overall incidence remains high [5]. The no-reflow phenomenon can directly affect the patient’s prognosis, increase the economic burden on the family and the country, and threaten the recovery of the patient’s cardiac function. In order to reduce the incidence of no-reflow during PCI treatment, this study carried out a corresponding analysis, as detailed below.

2. Materials and methods

2.1. General information

180 patients with AMI who underwent PCI treatment at the First Hospital of Hebei Medical University from November 2020 to December 2022 were divided into 3 groups by random number table extraction method: Group A, Group B, and Group C; each group included 60 cases. Group A included 32 males and 28 females, aged 63 to 81, with an average age of 72.65 ± 6.88 years; Group B included 34 males and 26 females, aged 64 to 82 years old, with an average age of 73.11 ± 6.93 years; Group C included 35 males and 25 females, aged 65 to 83 years old, with an average age of 73.85 ± 7.01. A detailed analysis of the basic information of the patients in the three groups showed no significant differences (P > 0.05). Inclusion criteria: (1) All participants in the study have been diagnosed with AMI, meet the relevant indications for PCI treatment, and can tolerate PCI surgery; (2) All patients are aware of the relevant contents of the study, agree to participate in it, and sign an informed consent form. Exclusion criteria: (1) Patients with severe dysfunction of important organs; (2) Patients with mental illness.

2.2. Methods

All patients in the study were given nitroglycerin 100–200 μg 1 to 3 times according to their conditions during interventional treatment. Group A: A bolus injection consisting of 20 mg recombinant human prourokinase diluted with saline was given within 5 minutes. Group B: An intracoronary bolus injection consisting of diltiazem hydrochloride diluted with saline was given at 400 μg each time. Group C: 10 mg of recombinant human prourokinase diluted with saline was injected into the relevant blood vessels proximal to the lesion through a suction catheter or punctured balloon. After the stent was released, 10 mg of recombinant human prourokinase was administered again.

2.3. Observation indicators

(1) Incidence rate of cardiovascular events: The occurrence of angina pectoris, heart failure, and recurrent myocardial infarction in the three groups after the intervention were observed.
(2) Cardiac function indicators: The left ventricular end-diastolic diameter (LVEDD), end-systolic diameter (LVESD), and ejection fraction (LVEF) indicators were collected from the three groups after the intervention.

(3) Quality of life: The SF-36 (36-Item Short Form) health survey was used to measure the quality of life of all patients. The survey includes psychological, physiological, physical, and social functions. The individual items are on a hundred-point scale and are positively correlated.

2.4. Statistical processing and analysis
The statistical software SPSS 22.0 was used to analyze the data of this study. The counting data were expressed as \[n (\%)\] and the \(c^2\) test was used; the measurement data were expressed as mean ± standard deviation (SD) and the \(t\)-test was used. \(P < 0.05\) indicated that the difference was statistically significant.

3. Results

3.1. Comparison of incidence rates of cardiovascular events between the groups
After intervention, the incidence rates of angina pectoris, heart failure, and recurrent myocardial infarction in Group C were lower than those in Group B \((P < 0.05)\); the overall incidence rate was the lowest in Group C, followed by Group A and Group B, as shown in Table 1.

**Table 1.** Comparison of incidence rates of cardiovascular events between the groups \([n (\%)]\)

<table>
<thead>
<tr>
<th>Group</th>
<th>Angina pectoris</th>
<th>Heart failure</th>
<th>Recurrent myocardial infarction</th>
<th>Overall incidence rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A ((n = 60))</td>
<td>3 (8.33)</td>
<td>4 (6.67)</td>
<td>3 (6.67)</td>
<td>10 (21.67)</td>
</tr>
<tr>
<td>Group B ((n = 60))</td>
<td>7 (10.00)</td>
<td>5 (10.00)</td>
<td>6 (13.33)</td>
<td>18 (38.33)</td>
</tr>
<tr>
<td>Group C ((n = 60))</td>
<td>2 (3.33)</td>
<td>2 (3.33)</td>
<td>1 (1.67)</td>
<td>5 (8.33)*</td>
</tr>
</tbody>
</table>

*\(P < 0.05\) compared with Group B

3.2. Comparison of cardiac function indicators between the groups after intervention
After intervention, LVEDD, LVESD, and LVEF in Group A and Group C were all better than those in Group B \((P < 0.05)\). The cardiac function indicators of Group C were better than those of Group A, as presented in Table 2.

**Table 2.** Comparison of cardiac function indicators between the groups after intervention (mean ± SD)

<table>
<thead>
<tr>
<th>Group</th>
<th>LVEDD (mm)</th>
<th>LVESD (mm)</th>
<th>LVEF (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A ((n = 60))</td>
<td>51.69 ± 4.32*</td>
<td>40.27 ± 2.34*</td>
<td>49.97 ± 4.54*</td>
</tr>
<tr>
<td>Group B ((n = 60))</td>
<td>53.33 ± 4.26</td>
<td>42.36 ± 2.39</td>
<td>47.36 ± 4.39</td>
</tr>
<tr>
<td>Group C ((n = 60))</td>
<td>47.36 ± 3.21*</td>
<td>36.54 ± 2.65*</td>
<td>52.69 ± 5.65*</td>
</tr>
</tbody>
</table>

*\(P < 0.05\) compared with Group A, *\(P < 0.05\) compared with Group B

3.3. Comparison of quality of life between the groups after intervention
After the intervention, the scores of different dimensions of quality of life in Group C were higher than those in Groups A and B, and the quality of life indicators in Group A were higher than those in Group B \((P < 0.05)\), as displayed in Table 3.
Table 3. Comparison of the quality of life between the groups after intervention (mean ± SD)

<table>
<thead>
<tr>
<th>Group</th>
<th>Psychological functions (points)</th>
<th>Physiological functions (points)</th>
<th>Physical functions (minutes)</th>
<th>Social functions (points)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A (n = 60)</td>
<td>73.14 ± 5.32*</td>
<td>72.46 ± 6.64*</td>
<td>74.97 ± 5.54*</td>
<td>74.37 ± 4.77*</td>
</tr>
<tr>
<td>Group B (n = 60)</td>
<td>70.38 ± 5.34</td>
<td>70.15 ± 5.36</td>
<td>72.28 ± 4.65</td>
<td>72.85 ± 4.26</td>
</tr>
<tr>
<td>Group C (n = 60)</td>
<td>80.28 ± 5.07*#</td>
<td>79.33 ± 6.11*#</td>
<td>80.27 ± 5.37*#</td>
<td>79.55 ± 4.69*#</td>
</tr>
</tbody>
</table>

*P < 0.05 compared with Group A, *P < 0.05 compared with Group B

4. Discussion and conclusion

AMI is one of the most common cardiovascular diseases. The occurrence of AMI is closely related to coronary artery stenosis or occlusion, emboli formation, and coronary artery spasm. Under the influence of the above factors, the coronary arteries can be blocked, resulting in stenosis or occlusion, blockage of coronary blood flow, inadequate oxygenation, and myocardial ischemia [6]. The occurrence of AMI can bring serious negative effects to the body. AMI can cause myocardial ischemia and necrosis, leading to myocardial cells being unable to work normally, reducing cardiac contractility, weakening cardiac function, and, in severe cases, causing heart failure [7]; at the same time, AMI can cause cardiac electrophysiological abnormalities, leading to arrhythmias, such as ventricular arrhythmias, ventricular tachycardia, and atrial fibrillation. If AMI is not effectively treated, it can cause myocardial damage, impair the heart’s pumping function, reduce cardiac output, and ultimately lead to heart failure, causing symptoms such as shortness of breath, edema, and fatigue [8].

Since AMI can result in significant harm, it is crucial to diagnose AMI as early as possible and take effective treatment measures. PCI is currently a common treatment method for AMI. It is an interventional therapy performed through catheters, which can re-establish the blood supply of the coronary arteries. PCI can restore blood flow as early as possible, limit the expansion of myocardial necrosis, and reduce subsequent complications. Restoration of coronary blood flow can minimize the area of myocardial necrosis, lower mortality rate, alleviate symptoms such as chest pain and dyspnea, reduce myocardial cell necrosis and myocardial damage, and preserve overall myocardial function [9]. However, it is vital to acknowledge that the no-reflow phenomenon is prone to occur during PCI. Poor blood flow recovery can directly reduce cardiac function and quality of life, affect the disease prognosis, and increase the risks of complications.

No-reflow phenomenon refers to the failure of blood flow to recover to the expected level after PCI, resulting in poor myocardial perfusion. The occurrence of this phenomenon is closely related to coronary embolism, ischemia-reperfusion injury, platelet aggregation, and inflammatory response [10]. Following the occurrence of no-reflow due to insufficient blood supply, the myocardium can remain in a state of ischemia, increasing the risk of serious complications such as myocardial infarction. No-flow can also lead to myocardial damage and fibrosis, affect cardiac systolic function, and aggravate the symptoms of heart failure [11]. During emergency PCI surgery, residual thrombi and fragmented atherosclerotic plaques can protrude from the stent mesh into the lumen and rush to the distal microvascular network due to the improved blood flow, causing slow blood flow, reperfusion injury, and reduced cardiac function. Effective clinical measures should be taken for prevention and remediation in response to this phenomenon. In this study, patients with AMI who underwent PCI were selected, and corresponding interventions were given during the implementation of PCI. The results of the study showed that after intervention in Group C, the incidence of angina pectoris, heart failure, and recurrent myocardial infarction was lower than that of Group A and Group B (P < 0.05); after the intervention, LVEDD, LVESD, and LVEF in Group C were all better than those of Group A and Group B (P < 0.05); after
the intervention, the quality of life scores in different dimensions of Group C were higher than those of Group A and Group B ($P < 0.05$).

It can be seen that the method adopted by Group C can effectively reduce the incidence of no-reflow, optimize patient prognosis, improve cardiac function, enhance quality of life, and reduce the incidence of adverse cardiovascular events. Recombinant human urokinase refers to human urokinase that is produced through genetic engineering technology. The human urokinase gene is introduced into bacteria or other organisms to express and secrete active urokinase, which is then applied to patients undergoing PCI [12]. It can dissolve blood clots, restore blood vessel patency, and reduce or prevent complications such as myocardial ischemia and necrosis [13]. As recombinant human urokinase exhibits strong thrombolytic activity, it can quickly dissolve thrombus in coronary arteries, restore smooth blood flow, and protect myocardium from ischemia and necrosis. Additionally, it can also improve coronary endothelial cell function, promote vasodilation and expansion, and restore normal blood vessel function [14]. Before the stent is released, recombinant human prourokinase is injected in the coronary artery for targeted thrombolytic therapy, which aids in quickly dissolving tiny thrombi; after the stent is released, the angiography time is delayed, and recombinant human urokinase is again injected into the targeted blood vessel. Prourokinase can effectively eliminate new distal microthrombus, fully improve the circulation of distal coronary microvessels, ultimately enhance post-stent blood flow, reduce the incidence of no-reflow or slow blood flow, and maximize cardiac protection function [15].

In summary, the targeted drug delivery strategy of the “front and back pinch method” in PCI treatment can effectively improve patient prognosis.

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**Disclosure statement**

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**References**


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