

Evaluating the Efficacy of Interventional Approaches for Cardiac Arrhythmias in Acute Myocardial Infarction

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Abstract: *Objective:* To assess the effectiveness of interventional treatments for cardiac arrhythmias in acute myocardial infarction (AMI). *Methods:* Eighty AMI patients admitted between August 2022 and August 2023 were selected and randomly assigned into groups using the random number table method. The control group ($n = 40$) received conventional thrombolytic treatment, while the observation group ($n = 40$) underwent percutaneous coronary intervention (PCI). Clinical effects were compared between the two groups. *Results:* Before treatment, there were no significant differences in heart rate indicators, cardiac function indicators, and physiological indicators between the two groups ($P > 0.05$). After treatment, the observation group showed significantly improved heart rate indicators, cardiac function indicators, and physiological indicators compared to the control group ($P < 0.05$). The adverse reaction rates in the observation group were lower than in the control group ($P < 0.05$). *Conclusion:* PCI treatment demonstrated significant improvements in heart rate, cardiac function, and physiological indicators among AMI patients, leading to a reduced incidence of adverse reactions such as arrhythmia. The overall effect is deemed significant.

Keywords: Interventional therapy for acute myocardial infarction; Arrhythmia; Cardiac function; Adverse reactions

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1. Introduction

Acute myocardial infarction (AMI) is a critical and severe cardiovascular disease characterized by its sudden onset, rapid progression, and challenging curability. Patients are susceptible to adverse reactions such as arrhythmia, heart failure, and shock following the onset of the disease. The pathological mechanism of AMI involves the rupture of blood vessel intima due to atherosclerosis, leading to the formation of blood clots that embolize local coronary arteries, resulting in vessel obstruction, acute ischemia, and hypoxia, ultimately leading to necrosis^[1].

With the aging of the nation's population intensifying, the prevalence of AMI continues to rise, affecting progressively younger demographic groups. This trend significantly impacts patients' quality of life and health. Timely perfusion therapy is essential for AMI patients after disease onset, as it helps preserve normal cardiac

function and reduces the incidence of adverse cardiovascular events, such as arrhythmia.

Percutaneous coronary intervention (PCI) emerges as a crucial intervention, swiftly clearing infarction-related arteries in AMI patients. It improves blood flow in the infarcted myocardium, rescuing dying myocardial tissue and achieving notable results^[2,3]. This study observed 80 AMI patients to assess the impact of PCI on the treatment of cardiac arrhythmia, aiming to provide a reliable reference for subsequent AMI treatments.

2. Materials and methods

2.1. General information

Eighty patients undergoing interventional treatment for AMI admitted between August 2022 and August 2023 were selected and divided using the random number table method, with 40 cases in each group. In the observation group, there were 22 males and 18 females, constituting 55.00% and 45.00%, respectively. Their ages ranged from 50 to 78 years with an average of 57.78 ± 4.39 years. Onset time varied from 2 to 6 hours with an average of 3.87 ± 0.33 hours. Common comorbidities included 12 cases of hypertension (30.00%), 13 cases of diabetes (32.50%), and 15 cases of coronary heart disease (37.50%). The AMI locations were distributed as follows: 15 cases on the anterior wall (37.50%), 8 cases on the anteroseptal wall (20.00%), and 17 cases on the inferior wall (42.50%).

In the control group, there were 24 males and 16 females, accounting for 60.00% and 40.00%, respectively. The age range was 51 to 76 years with a mean of 57.82 ± 4.41 years. Onset time was between 3 to 6 hours with a mean of 3.89 ± 0.36 hours. Common comorbidities included 10 cases of hypertension (25.00%), 14 cases of diabetes (35.00%), and 16 cases of coronary heart disease (40.00%). The AMI locations were distributed as follows: 16 cases on the anterior wall (40.00%), 10 cases on the anteroseptal wall (25.00%), and 14 cases on the inferior wall (35.00%). There were no statistically significant differences in the relevant data between the two patient groups ($P > 0.05$).

2.2. Inclusion and exclusion standards

Inclusion criteria included all patients diagnosed with AMI requiring PCI surgery^[4], time of onset ≤ 6 hours, as well as patients and their families agreeing to participate in the study and signing the informed consent.

Exclusion criteria included individuals with organ dysfunction or malignant arrhythmia, those with malignant tumors or immune diseases, those who received thrombolytic therapy before PCI, and those with mental illness or cognitive impairment.

2.3. Methods

In the control group, patients received an initial intravenous infusion of recombinant human prourokinase 20 mg, followed by a continued infusion of 30 mg, with the total infusion time controlled within 30 minutes.

Patients in the observation group underwent PCI treatment. They were administered aspirin 300 mg + clopidogrel 300 mg or chewed ticagrelor 180 mg. Coronary angiography precisely located the site of vascular obstruction, and standard percutaneous transluminal coronary angioplasty was performed to swiftly establish a venous circulation pathway. During the procedure, the patient received 100 U/kg heparin four times. A suitable stent was accurately inserted based on the patient's vascular condition. After restoring blood flow, the clinician administered 4000 μ g of low molecular weight heparin subcutaneously every 12 hours, along with oral aspirin 100 mg and clopidogrel 300 mg once daily.

2.4. Observation indicators

Comparison of heart rate indicators [corrected QT interval (QTc), QT interval dispersion (QTd), and heart rate (HR)] before and after treatment, cardiac function indicators [using color Doppler ultrasound to detect left ventricular ejection fraction (LVEF), left ventricular end-diastolic and end-systolic diameters (LVEDD, LVESD)] before and after treatment, physiological indicators [detection of brain natriuretic peptide (BNP) by radioimmunoassay, detection of C-reactive protein (CRP) level by immunoturbidimetry] before and after treatment, and the occurrence of adverse reactions (heart failure, ventricular arrhythmia, cardiogenic shock) post-medication were examined.

2.5. Statistical analysis

SPSS 27.0 was employed as the data analysis software. Measurement data were expressed as mean \pm standard deviation (SD), and a *t*-test was conducted. Count data were expressed as *n* (%), and a χ^2 test was performed. A significance level of $P < 0.05$ indicated a significant difference between the data.

3. Results

3.1. Comparison of heart rate indicators between the two groups

Prior to treatment, there was no statistically significant difference in QTc, QTd, and HR between the groups ($P > 0.05$). However, after treatment, the QTc, QTd, and HR indicators of the observation group demonstrated improvement compared to the control group ($P < 0.05$), as shown in **Table 1**.

Table 1. Comparison of heart rate indicators before and after treatment (mean \pm SD)

Group	QTc (ms)		QTd (ms)		HR (times/min)	
	Before	After	Before	After	Before	After
Observation group (<i>n</i> = 40)	452.78 \pm 40.37	433.42 \pm 48.64	67.14 \pm 5.38	32.63 \pm 3.39	98.55 \pm 8.81	65.69 \pm 6.88
Control group (<i>n</i> = 40)	452.81 \pm 40.43	400.15 \pm 53.18	67.48 \pm 5.42	34.79 \pm 3.72	98.12 \pm 8.75	60.62 \pm 7.53
<i>t</i>	0.003	2.920	0.282	2.714	0.219	3.144
<i>P</i>	0.997	0.005	0.779	0.008	0.827	0.002

3.2. Comparison of cardiac function indicators between the two groups

Table 2 shows that the evaluation of LVEDD, LVEF, and LVESD between the groups showed no statistical significance before treatment ($P > 0.05$). Nevertheless, post-treatment, the LVEDD, LVEF, and LVESD levels in the observation group exhibited improvement compared to the control group ($P < 0.05$).

Table 2. Comparison of cardiac function indicators before and after treatment (mean \pm SD)

Group	LVEDD (mm)		LVEF (%)		LVESD (mm)	
	Before	After	Before	After	Before	After
Observation group (<i>n</i> = 40)	66.02 \pm 6.15	51.39 \pm 4.35	35.22 \pm 2.81	46.37 \pm 3.57	56.05 \pm 6.29	42.35 \pm 4.02
Control group (<i>n</i> = 40)	66.04 \pm 6.27	60.21 \pm 5.42	35.26 \pm 2.85	40.56 \pm 2.45	56.09 \pm 6.27	50.25 \pm 5.12
<i>t</i>	0.014	8.027	0.063	8.487	0.028	7.675
<i>P</i>	0.989	< 0.001	0.950	< 0.001	0.977	< 0.001

3.3. Comparison of physiological indicators between the two groups

The evaluation of BNP and CRP levels between groups did not reveal any statistical significance before treatment ($P > 0.05$). However, after treatment, the BNP and CRP levels in the observation group were lower than those in the control group ($P < 0.05$), as shown in **Table 3**.

Table 3. Comparison of physiological indicators before and after treatment (mean \pm SD)

Group	BNP (pg/ml)		CRP (mg/L)	
	Before	After	Before	After
Observation group ($n = 40$)	464.27 \pm 57.26	340.08 \pm 50.26	12.62 \pm 2.59	5.21 \pm 1.57
Control group ($n = 40$)	465.32 \pm 57.39	432.05 \pm 54.49	12.64 \pm 2.58	8.18 \pm 2.55
<i>t</i>	0.082	7.847	0.035	6.273
<i>P</i>	0.935	< 0.001	0.973	< 0.001

3.4. Comparison of adverse reaction rates between the two groups

Table 4 shows that the adverse reaction rate in the observation group was significantly lower than in the control group ($P < 0.05$).

Table 4. Comparison of adverse reaction rates after treatment [n (%)]

Group	Heart failure	Ventricular arrhythmias	Cardiogenic shock	Overall incidence
Observation group ($n = 40$)	1 (2.50)	1 (2.50)	0 (0.00)	2 (5.00)
Control group ($n = 40$)	3 (7.50)	5 (12.50)	1 (2.50)	9 (22.50)
χ^2	-	-	-	5.165
<i>P</i>	-	-	-	0.023

4. Discussion

Patients with AMI face challenges in maintaining stable vital signs post-onset, increasing the risk of complications such as heart failure and arrhythmia. Early clinical intervention to rapidly reduce myocardial infarction size and ensure blood flow recanalization holds significant importance^[5]. Clinical treatment of AMI predominantly centers on the timely opening of blocked blood vessels to restore and reperfuse blood in the infarcted myocardium. This approach proves beneficial in salvaging damaged myocardial cells and fostering positive improvements in cardiac function and symptoms. Intravenous thrombolysis, another common treatment for AMI, faces challenges influenced by factors like contraindications and time windows. The clinical efficacy of intravenous thrombolytic therapy varies due to individual differences among patients, leading to inherent limitations and rendering it unsuitable for every AMI patient^[6].

In contrast, early PCI treatment swiftly opens obstructed blood vessels, facilitating the restoration of coronary reperfusion. This method boasts advantages such as simplicity, minimal trauma, rapid recovery, and high safety^[7]. Prior to PCI treatment, a comprehensive evaluation of the AMI patient's condition is necessary, considering factors for complete revascularization. Notably, patients often present with complications such as arrhythmia, and PCI treatment plays a pivotal role in stabilizing hemodynamics. It significantly reduces cardiac afterload and myocardial oxygen consumption while simultaneously enhancing diastolic coronary blood flow. This dual effect significantly improves blood flow and perfusion, thereby safeguarding cardiac function and lowering the incidence of adverse reactions such as arrhythmia^[8].

This study reveals that post-treatment, the observation group exhibited higher QTc and HR levels, a lower QTd level, and a significantly better heart rate index compared to the control group ($P < 0.05$). These findings suggest that PCI treatment can enhance the heart rate of AMI patients and contribute to their recovery. Additionally, the observation group demonstrated lower levels of LVEDD and LVESD, along with higher LVEF levels after treatment, all of which were superior to those of the control group ($P < 0.05$). This implies that, in comparison to conventional thrombolytic treatment, PCI treatment is more effective in improving cardiac function and clinical prognosis.

The analysis attributes this efficacy to the use of aspirin, an anti-platelet aggregation drug that clinically inhibits cyclooxygenase, impedes thromboxane A₂ formation, and achieves anticoagulant and antithrombotic effects. Clopidogrel, another antiplatelet drug, effectively inhibits glycoprotein complex activation mediated by adenosine diphosphate, positively influences adenylyl cyclase activity, and inhibits platelet aggregation. It proves significant in treating AMI, promoting cardiac blood circulation during PCI surgery, reducing arterial restenosis, and aiding myocardial blood perfusion. Ticagrelor, with its inhibitory effect on arterial intimal hyperplasia and protective effect on cardiomyocytes, avoids damage to the vascular endothelium and promotes positive improvements in coronary blood flow. It binds organically to the adenosine diphosphate receptor P2Y₁₂, inhibiting platelet membrane glycoprotein complex production and platelet aggregation. Importantly, ticagrelor does not require liver metabolism activation, exerting its effect 2 hours after ingestion and maintaining a long half-life for stable drug effects. While inhibiting the antithrombotic mechanism, ticagrelor dilates blood vessels, significantly reducing cardiac load and myocardial oxygen consumption in AMI patients^[9,10].

In this study, the combination of aspirin + clopidogrel or chewable ticagrelor in treating AMI achieved noteworthy results. PCI treatment consistently enhanced coronary blood flow in AMI patients, demonstrating robust stability that maximized myocardium rescue and improved cardiac function. Post-treatment, the observation group exhibited lower levels of BNP and CRP compared to the control group ($P < 0.05$). This suggests that PCI treatment has a positive impact on the patient's coronary stenosis, quickly restoring blood supply to the myocardium to effectively relieve cardiac load. Simultaneously, it significantly reduced BNP secretion by ventricular myocytes, improving ventricular function and clinical prognosis. Moreover, PCI treatment reopened blocked infarction-related blood vessels, enhancing myocardial ischemia, collateral circulation, and cardiac function compensatory ability to prevent diseased artery re-occlusion. The study demonstrated that the adverse reaction rate in the observation group was 5.00%, significantly lower than the control group's 22.50% ($P < 0.05$). This suggests that PCI treatment can effectively reduce adverse reactions such as arrhythmia and enhance clinical safety in AMI patients.

In conclusion, PCI treatment shows promise in positively affecting heart rate, cardiac function, BNP, and CRP indicators in AMI patients. Its clinical efficacy, disease prognosis, and reduced adverse reaction rates, especially in arrhythmia, highlight its clinical significance.

Disclosure statement

The authors declare no conflict of interest.

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