

The effect of phase I cardiac rehabilitation exercise training combined with respiratory training on the Improvement of Cardiopulmonary Function: A Randomized Controlled Trial

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Abstract

Background

Few investigators have tested the effect of phase I cardiac rehabilitation (CR) exercise training and respiratory training in acute myocardial infarction (AMI) patients after percutaneous coronary intervention (PCI).

Objective

To explore the effect of phase I CR combined with respiratory training in AMI patients after PCI.

Methods

A total of 110 AMI patients who had undergone PCI were screened for inclusion and randomly divided into experimental and control groups at a ratio of 1:1. The experimental group received phase I CR exercise training combined with respiratory training after PCI, while the control group did not receive any treatment. We compared cardiopulmonary exercise test (CPET) results, echocardiographic parameter changes and laboratory parameters between the experimental and control groups during hospitalization and after 3 months.

Results

A total of 106 patients (91 males, 85.8%) with an average age of 57.3 ± 9.0 years. After three months, the oxygen uptake peak (VO_2 peak), VO_2 anaerobic threshold (AT), metabolic equivalents (METs) and O_2 pulse in the experimental group increased by 2.446, 1.541, 0.762 and 1.162 units, respectively (all $p < 0.05$). There were no significant changes in other echocardiographic indices, ventilatory equivalents for carbon dioxide (VE/VCO_2) or ventilatory equivalents (VEs) between the two groups (all $p > 0.05$).

Conclusions

Phase I CR combined with respiratory training can improves VO_2 AT, VO_2 peak and O_2 pulse of AMI patients after PCI. Thus, it had a positive effect on the cardiopulmonary function.

INTRODUCTION

Acute myocardial infarction (AMI) is a common cause of hospitalization for heart disease^[1]. It is characterized by abrupt onset, high severity, and elevated mortality^[2]. AMI remains a leading global health burden, with an estimated 800 – 100 million annual cases and AMI mortality rate is as high as

15%^[3]. Percutaneous coronary intervention (PCI) is an effective treatment for AMI^[4]. While primary PCI reduces acute mortality, but survivors experience persistent ventricular dysfunction or functional limitations. However, the exercise capacity of AMI patients after PCI has not improved significantly and even decreased in some of the patients. The PEACE prospective cohort study shows that nearly 40% of AMI patients in China are unable to return to work one year after onset due to decreased activity tolerance^[5]. Most patients with AMI have significantly reduced cardiovascular endurance and mental health problems which seriously affect their quality of life^[6].

Specifically, many AMI patients face a significant decline in exercise capacity post-PCI. This is linked to factors like reduced cardiac output, muscle deconditioning from prolonged inactivity. Cardiac rehabilitation (CR) not only helps patients regain independence in daily living but also contributes to long-term cardiovascular health. CR includes five major prescriptions: medication, exercise, nutrition, psychology and smoking cessation. It includes 4 phases: phase 1 involves the hospitalization, phase 2 is the immediate discharge, phase 3 is the structured exercise plan, and phase 4 is the maintenance period. CR has become an important component of cardiovascular disease treatment, as it improves patients' exercise tolerance and cardiopulmonary function and enhances their psychological health and quality of life^[7]. Early CR training has great benefits for patients with cardiovascular disease. It can help patients with cardiovascular disease gradually restore a normal state of life^[8,9]. Cardiopulmonary endurance is closely related to the prognosis and all-cause mortality rate of cardiovascular disease patients^[10]. Although CR has been widely proven to have significant clinical benefits in improving the functional status of AMI patients after PCI, there remains uncertainty in the academic community regarding how to further enhance its intervention effects. Heart and lung are interdependent and closely related. Cardiopulmonary endurance is closely related to the occurrence, prognosis and all-cause mortality of cardiovascular diseases^[11]. Respiratory training generally uses pursed lip breathing and abdominal exercises, which can increase tidal volume and alveolar ventilation, thereby improving cardiovascular endurance and quality of life^[12].

Long-term bed rest in patients with AMI can lead to complications such as decreased exercise endurance, reduced blood volume and thromboembolism. It is not conducive to the recovery of patients with heart disease^[13]. Early bedside phase I CR exercise and breathing training can shorten hospitalization time and promote the recovery of daily living and exercise abilities^[14,15]. Early CR also can significantly increase the oxygen uptake of patients, reduce the incidence of recurrent angina pectoris and arrhythmia^[16]. At the same time, the guidelines recommend CR for secondary prevention for patients with CVD^[17]. A prospective observational study conducted at the Cardiology Department of Khyber Teaching Hospital in Pakistan showed that the incidence of recurrent myocardial infarction and arrhythmia in patients with AMI decreased after CR^[18]. In our clinical research, we have observed that patients undergoing CR treatment showed a reduction in major adverse cardiovascular events (MACE) and an average reduction of 1.8 days in hospital stay during the follow-up period.

Moreover, the mechanisms underlying the effects of CR on cardiopulmonary function remain unclear. Given the uncertainty of the role of CR in AMI patients after PCI, our study aimed to compare the differences of 3-month changes in cardiopulmonary exercise test (CPET) and cardiac function indices between experimental and control groups. Therefore, to explore the impact of phase I cardiac rehabilitation exercise training combined with respiratory training on AMI patients after PCI.

For patients with AMI, although cardiac rehabilitation is a guideline-recognized and evidence-based approach worldwide, the mechanism by which it affects cardiopulmonary function remains unclear. At present, there are relatively few studies at home and abroad on whether the prognosis of Phase I CR exercise training combined with respiratory training after PCI for acute myocardial infarction is better than that of cardiac rehabilitation exercise alone for patients after PCI for acute myocardial infarction. There is a lack of sufficient literature to confirm this. This study aims to explore the effect of phase I CR exercise training combined with respiratory training on patients after PCI for acute myocardial infarction. It is evaluated through changes in CPET and cardiac function indicators. Thus, it provides certain clinical evidence for the clinical treatment plan of acute myocardial infarction.

METHODS

Study Selection

This was a prospective, randomized controlled trial assessing the pivotal role of phase I cardiac rehabilitation exercise training combined with respiratory training in improving cardiopulmonary function in AMI patients after PCI. This study was approved by Yulin First People's Hospital, China (YLSY-IRB-SR-2022019) and was conducted in accordance with the Declaration of Helsinki. All patients provided written informed consent. The study adhered to the CONSORT guidelines for reporting randomized trials.

Patients

A total of 110 AMI patients who underwent PCI at our center between May 2022 and May 2023 were screened for inclusion. Patients who met the following criteria were included: aged 18 years or older, without new or recurrent chest pain within the past 8 hours, with no further increase in the level of myocardial injury markers, no obvious signs of decompensation in heart failure, and no new arrhythmias or changes in the electrocardiogram. The study exclusion criteria included an abnormal blood pressure response, aortic dissection, severe aortic valve stenosis, liver and kidney dysfunction, rheumatic heart disease, pulmonary heart disease, cardiomyopathy, myocarditis, severe arrhythmia, and severe heart failure. Block randomization was adopted for participant allocation, with a block size of 1 to 200 and random numbers generated using SPSS software. The patients were randomly divided into an experimental group (receiving phase I CR exercise training combined with respiratory training) and control group (standard care without this intervention) at a 1:1 ratio according to the computer-generated randomization table.

Intervention

The standard care for all participants is as follows: All participants strictly follow medical advice and medication throughout the entire process. Closely monitor complications such as malignant arrhythmia, heart failure, and cardiogenic shock, and simultaneously take care of diet and bowel movements. The control group received routine treatment. The experimental group underwent one week of rehabilitation exercise and respiratory training simultaneously with routine treatment.

The one-week rehabilitation plan was as follows (Table 1). The breathing training method are as follows:

- ☒ Abdominal pursed lip breathing: using a lying or standing position, placing one hand on the abdomen and slowly inhaling through the nose. When inhaling, the abdominal wall rises, and then slowly exhaling through the mouth. When exhaling, the lips are pursed into a whistling shape, and slight pressure can be applied to the abdomen to make it retract as much as possible. The exhalation time should be 1–2 times longer than the inhalation time, 5 to 10 minutes each time, 2 times a day.
- ☒ Breathing exercises: Breathing exercises: The patient stands with their feet shoulder width apart. Perform 4–8 breaths with hands on hips, hands on shoulders, alternating one leg lifting, pursed lips and abdomen, and alternating leg abduction. After discharge, both the experimental group and the control group only received standard home rehabilitation recommendations (including home-based walking exercise, daily respiratory training reminders, and dietary advice) and did not receive supervised phase 2 rehabilitation. The home rehabilitation recommendations were provided in written form and explained to patients and their family members before discharge.

Table 1
Daily Rehabilitation Schedule

Day	Core Rehabilitation Tasks	Activity Intensity/Scope	Degree of Self-Care
Day 1	1. Strict bed rest 2. Passive joint exercises (dorsiflexion and plantar flexion of the ankle once per hour when awake)	Only passive bed-bound activities; no independent getting up	Fully dependent on caregivers
Day 2	1. Sitting at the bedside 2. Active/passive movement of all joints	Confined to sitting at the bedside; no standing or walking	Requires assistance from caregivers
Day 3	1. Getting out of bed to stand + warm-up exercises 2. Slow walking 15–25 meters in the ward, twice a day 3. Rest for 15–30 minutes after activity	Short-distance slow walking; mandatory rest after activity	Mostly self-sufficient
Day 4	1. Activities within the room + performing cardiac rehabilitation exercises 2. Moderate-pace walking 25–50 meters, twice a day	Moderate-pace walking; activity scope extended to the entire room	Fully self-sufficient (able to walk to the bathroom and treatment room)
Day 5	1. Moderate-pace walking 100–150 meters, twice a day 2. Able to climb up and down 1 flight of stairs 3. Permitted to walk freely in the ward corridor at any time	Extended walking distance; adding stair-climbing movement	Fully self-sufficient
Days 6–7	1. Moderate-pace walking 200–400 meters, twice a day 2. Able to climb up and down 2 flights of stairs	Further extended walking distance; increased number of stair flights	Fully self-sufficient; daily activity intensity may be slightly higher than pre-illness level

Endpoints

The primary endpoints in our study were the changes in cardiopulmonary exercise test indices [oxygen uptake peak (VO₂ peak), VO₂ anaerobic threshold (AT), metabolic equivalents (METs), oxygen pulse (O₂ pulse), Ventilatory equivalents for carbon dioxide (VE/VCO₂) and VE] and echocardiographic parameters [left ventricular ejection fraction (LVEF), left atrial diameter (LA) and left ventricular end-diastolic

diameter (LVD)] after 3 months. Specifically, the primary endpoints of CPET indices include VO_2 AT, VO_2 peak, METs, O_2 pulse; the echocardiographic parameters include LVEF.

Secondary Endpoints: These include two subcategories: secondary outcome indicators and baseline characteristics. Secondary outcome indicators mainly cover the incidence of adverse cardiovascular events. Baseline characteristics include demographic data (age, gender), clinical history (history of hypertension, diabetes mellitus etc.), anthropometric parameters (body mass index, BMI), and laboratory test results (fasting blood glucose, total cholesterol, triglycerides, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, creatinine, etc.). These parameters are used to analyze potential confounding factors and explore the correlation between baseline status and intervention outcomes.

Adverse cardiovascular events include ① all-cause mortality: defined as any death occurring from any cause (including cardiovascular and non-cardiovascular causes) during the study follow-up period, regardless of the underlying mechanism or pathology; ② recurrent myocardial infarction: defined as a new episode of myocardial necrosis in patients with a prior history of myocardial infarction, excluding cases of myocardial injury caused by non-ischemic factors (e.g., myocarditis, heart trauma); ③ malignant arrhythmia: defined as life-threatening arrhythmias that can lead to hemodynamic instability, cardiac arrest, or sudden death, mainly including ventricular fibrillation, ventricular tachycardia, and high-degree atrioventricular block (third-degree atrioventricular block) accompanied by syncope or hypotension; ④ stroke: defined as a focal or global disturbance of cerebral function lasting more than 24 hours (or leading to death within 24 hours), caused by either cerebral ischemia or cerebral hemorrhage, excluding transient ischemic attacks (TIAs). All participants were assessed for cardiopulmonary function, cardiac structural changes, and adverse cardiovascular events at baseline (one week after PCI when the patient's condition was stable) and 3 months after randomization. Regarding blinding, all outcome assessors (including CPET operators and echocardiographers) will be strictly blinded to the patient's group allocation. **Follow-up Methods:** A combination of on-site follow-up and telephone follow-up will be adopted.

CPET

The CPET was performed by a Master Screen CPX tester from German Yeger (Model, manufacturer: Master Screen, Careu Fusion Germany234GmbH). The test consists of three consecutive stages: a 5-minute rest stage, a 3-minute warm-up stage, and a 3-minute exercise stage. The speed and level increase every 3 minutes. Blood pressure, heart rate, and power were monitored throughout the entire exercise process. Then, experienced medical staff calculated indices such as the VO_2 AT, VO_2 peak, MET, O_2 Pulse, VE/VCO_2 , and VE based on the experiments. The trial was terminated based on the following criteria: (1) The subject reached their predicted maximum heart rate ($\geq 90\%$ of the predicted maximum heart rate for age, calculated as $220 - \text{age}$); (2) Obvious electrocardiogram abnormalities occur (such as ST segment depression $\geq 1\text{mm}$, ventricular tachycardia); (3) Severe symptoms, including chest pain, dizziness, severe shortness of breath (Modified Borg Scale score ≥ 9) or leg fatigue, making it impossible

to move further; (4) Despite the increase in workload, systolic blood pressure still decreased by ≥ 20 mmHg; (5) The participant requests to stop. All CPET tests were conducted by well-trained medical staff who were unaware of the allocation of participant groups. To ensure quality control and reproducibility.

Statistical Analysis

Statistical analyses were performed using SPSS 23.0 software. The normality of continuous data distribution was assessed using the Shapiro-Wilk test. Continuous variables with a normal distribution are expressed as mean $\pm$ standard deviation (SD), and comparisons between two groups were conducted using the independent samples t test. For continuous variables that did not conform to a normal distribution, non-parametric tests (Mann-Whitney U test) were applied. The count data are expressed as the number of patients and percentage (%). A chi-square test was used for comparisons between groups. The relationships between CPET and the experimental groups were analyzed by multiple linear regression. The proportion of missing data for all key variables was $< 5\%$, and listwise deletion was used. A value of $P < 0.05$ in a two-sided test was considered to indicate statistical significance.

RESULTS

Patient enrollment process and follow-up status

Among the 125 ACS patients receiving PCI treatment who were assessed for eligibility, 110 patients were randomly divided into an experimental group and a control group at a ratio of 1:1. Fifteen patients were excluded. Of these patients, 6 did not meet the inclusion criteria, 4 did not provide consent, 3 had poor exercise capacity, and 2 died. A total of 4 patients were lost to follow-up, including 3 in the experimental group and 1 in the control group. One patient in the control group was lost to follow-up due to absence. Two patients in the experimental group were lost to follow-up due to their refusal to follow up, and one patient was lost to follow-up due to other health problems. The final 106 participants completed the 3-month follow-up. Among the participants who completed follow-up, 49.0% were in the experimental group. A total of 4 patients were lost to follow-up, with a loss to follow-up rate of 3.6% (following the CONSORT 2010 reporting standard in the flow diagram: Fig. 1).

Baseline Characteristics of the Study Participants

This study included a total of 106 patients (91 males, 85.8%) with an average age of 57.3 ± 9.0 years. Patients with hypertension and diabetes accounted for 45.3% and 20.8%. There was no statistically significant difference between the experimental group and the control group in terms of age, sex, smoking history, BMI, medical history, blood cholesterol, triglycerides, creatinine, uric acid, myocardial enzymes or blood pressure ($p > 0.05$). No participants experienced any adverse events during the training process. The heart rate of the experimental group was greater than that of the control group ($p <$

0.05). There was no statistically significant difference in the number of lesions or lesions in the left main trunk during surgery (Table 2).

Table 2
Demographics and characteristics of the patients

Clinical data	Total (n = 106)	Experimental group (n = 52)	Control group (n = 54)	p
Age (years)	57.3 ± 9.0	56.5 ± 10.1	58.1 ± 7.7	0.359
Male [n (%)]	91(85.8)	46(88.5)	45(83.3)	0.580
Smoking history [n (%)]	58(57.4)	30(57.7)	28(51.9)	0.565
BMI (kg/m ²)	24.59 ± 3.38	24.75 ± 3.13	24.44 ± 3.63	0.649
Hypertension [n (%)]	48(45.3)	23(44.2)	25(46.3)	0.848
Diabetes [n (%)]	22(20.8)	9(17.3)	13(24.1)	0.475
TC (mmol/L)	4.69 ± 0.90	4.75 ± 1.00	4.64 ± 0.80	0.518
TG (mmol/L)	1.97 ± 1.53	2.14 ± 1.90	1.80 ± 1.06	0.257
HDL(mmol/L)	1.02 ± 0.31	1.04 ± 0.33	1.00 ± 0.29	0.542
LDL(mmol/L)	3.00 ± 0.84	2.96 ± 0.98	3.05 ± 0.68	0.615
CR (μmol/L)	95.87 ± 31.91	90.0 ± 23.8	101.6 ± 37.5	0.060
UA (μmol/L)	358.2 ± 103.2	352.3 ± 107.2	363.9 ± 99.8	0.565
CTnT	3564.9 ± 3136.9	3242.9 ± 2979.6	3874.9 ± 3278.9	0.302
CK	190.4 ± 214.0	168.6 ± 211.7	211.5 ± 216.1	0.305
CK-MB	132.2 ± 163.5	110.9 ± 154.8	152.7 ± 170.4	0.190
SBP (mmHg)	133.76 ± 24.07	132.9 ± 23.3	134.6 ± 25.0	0.726
DBP (mmHg)	85.33 ± 15.60	83.5 ± 16.1	87.1 ± 15.1	0.243
HR (times/min)	79.10 ± 15.59	82.5 ± 17.4	75.8 ± 12.9	0.025
Left main coronary artery disease	6(5.7)	1(1.9)	5(9.3)	0.206
Number of diseased vessels				0.278
One	26(24.5)	13(25.0)	13(24.1)	
BMI: body mass index, CTnT: Cardiac troponin T, CK: creatine kinase, CK-MB: creatine kinase isoenzyme, TG: triglycerides, TC: total cholesterol, HDL: High density lipoprotein, LDL: Low density lipoprotein, UA: uric acid, Cr: serum creatinine, SBP: systolic blood pressure, DBP: diastolic blood pressure, HR: heart rate				

Clinical data	Total (n = 106)	Experimental group (n = 52)	Control group (n = 54)	p
Two	36(34.0)	14(26.9)	22(40.7)	
Three	44(41.5)	25(48.1)	19(35.2)	
BMI: body mass index, CTnT: Cardiac troponin T, CK: creatine kinase, CK-MB: creatine kinase isoenzyme, TG: triglycerides, TC: total cholesterol, HDL: High density lipoprotein, LDL: Low density lipoprotein, UA: uric acid, Cr: serum creatinine, SBP: systolic blood pressure, DBP: diastolic blood pressure, HR: heart rate				

CPET and echocardiographic parameter outcomes in the study population

All study participants underwent a CPET and cardiac ultrasound evaluation after PCI and 3 months postoperatively. The EF of the population after PCI was 50.8 ± 9.6 , the VO_2 AT was 13.07 ± 3.13 , and the VO_2 peak was 16.50 ± 3.65 . After 3 months, the EF was 54.8 ± 10.8 , the VO_2 AT was 14.48 ± 3.25 , and the VO_2 peak was 18.42 ± 3.96 . The EF, LVD, VO_2 AT, VO_2 peak, METs, VE and O_2 Pulse all increased compared with baseline, while VE/ VCO_2 slope decreased compared with the baseline ($p < 0.05$) (Table 3).

Table 3
CPET and echocardiographic parameters outcomes in the study population

Total(n=106)	after PCI	after 3 months	p
LVEF [(%)]	50.8 ± 9.6	54.8 ± 10.8	<0.001
LA	32.5 ± 4.3	33.4 ± 4.4	0.071
LVD	46.7 ± 5.4	49.3 ± 5.9	<0.001
VO_2 AT(ml/min/kg)	13.07 ± 3.13	14.48 ± 3.25	<0.001
VO_2 peak(ml/min/kg)	16.50 ± 3.65	18.42 ± 3.96	<0.001
METs	4.72 ± 1.05	5.28 ± 1.17	<0.001
O_2 Pulse (mL/beat)	9.48 ± 1.98	10.17 ± 2.08	<0.001
VE/ VCO_2 slope	35.112 ± 5.730	32.986 ± 4.608	<0.001
VE(L/min)	45.4 ± 11.1	50.4 ± 9.9	<0.001
LVEF: left ventricular ejection fraction, LA: Left atrial diameter, LVD: Left ventricular end diastolic diameter, AT: anaerobic threshold, VO_2: oxygen uptake, VO_2 peak: oxygen uptake peak, METs: metabolic equivalents, O_2 Pulse: oxygen pulse, VE/VCO_2: Ventilatory equivalents for carbon dioxide, VE: ventilation			

CPET and echocardiographic parameter outcomes in the experimental and control groups

After PCI, there was no significant difference in EF, LA, LVD, VO_2 AT, VO_2 peak, MET, oxygen pulse, VE/VCO_2 slope, or VE between the experimental group and the control group. At 3 months, follow-up cardiac ultrasound and CPET results showed that the experimental group had significantly greater levels of VO_2 AT, VO_2 peak, MET, and O_2 pulse than did the control group ($p < 0.05$). There was no significant difference between the two groups in terms of cardiac ultrasound. (Table 4).

Table 4
CPET and echocardiographic parameters outcomes in the experimental and control group

After PCI	Experimental group (n = 52)	Control group (n = 54)	<i>p</i>
LVEF [(%)]	50.5 ± 10.9	51.2 ± 8.0	0.693
LA	32.8 ± 4.4	32.2 ± 4.1	0.527
LVD	46.9 ± 6.4	46.5 ± 4.2	0.689
VO ₂ AT(ml/min/kg)	12.91 ± 3.07	13.22 ± 3.22	0.607
VO ₂ peak(ml/min/kg)	16.12 ± 3.47	16.86 ± 3.81	0.297
METs	4.61 ± 0.99	4.82 ± 1.10	0.305
O ₂ Pulse (mL/beat)	9.51 ± 1.81	9.44 ± 2.15	0.847
VE/VCO ₂ slope	35.199 ± 6.108	35.028 ± 5.398	0.878
VE(L/min)	45.2 ± 11.8	45.6 ± 10.5	0.873
3 moths			
LVEF [(%)]	55.9 ± 11.4	53.7 ± 10.2	0.312
LA	34.1 ± 4.6	32.8 ± 4.0	0.117
LVD	49.4 ± 7.0	49.2 ± 4.8	0.889
VO ₂ AT(ml/min/kg)	15.35 ± 3.42	13.63 ± 2.87	0.006
VO ₂ peak(ml/min/kg)	19.44 ± 3.84	17.45 ± 3.85	0.009
METs	5.60 ± 1.12	4.97 ± 1.15	0.005
O ₂ Pulse (mL/beat)	10.78 ± 1.91	9.59 ± 2.09	0.003
VE/VCO ₂ slope	32.466 ± 4.537	33.486 ± 4.663	0.256
VE(L/min)	51.5 ± 10.7	49.3 ± 9.0	0.264
LVEF: left ventricular ejection fraction, LA: Left atrial diameter, LVD: Left ventricular end diastolic diameter, AT: anaerobic threshold, VO₂: oxygen uptake, VO₂ peak: oxygen uptake peak, METs: metabolic equivalents, O₂ Pulse: oxygen pulse, VE/VCO₂: Ventilatory equivalents for carbon dioxide, VE: ventilation			

Changes in CPET between the experimental and control groups

After 3 months, the changes in the VO_2 AT, VO_2 peak, METs, and O_2 pulse differed significantly between the experimental group and the control group. The experimental group was significantly higher than the control group ($p < 0.05$). However, there was no significant difference in the VE/VCO_2 slope, VE , or EF between the two groups (Table 5 and Fig. 2).

Table 5
Changes in CPET between the experimental and control groups

Changes(3 months-After PCI)	Experimental group (n = 52)	Control group (n = 54)	p
LVEF [(%)]	4.50(0.00, 9.75)	3.00(-2.25, 8.00)	0.132
LA	1.00(-2.00, 4.00)	0.00(-2.00, 2.25)	0.356
LVD	3.00(-1.00, 6.00)	3.00(0.00, 5.00)	0.797
VO_2 AT(ml/min/kg)	2.50(0.60, 3.50)	0.80(-0.65, 1.43)	<0.001
VO_2 peak(ml/min/kg)	3.35(1.80, 4.65)	0.70(-0.60, 2.00)	<0.001
METs	0.95(0.60, 1.38)	0.20(-0.33, 0.60)	<0.001
O_2 Pulse (mL/beat)	1.10(0.43, 2.00)	0.20(-0.50, 0.80)	<0.001
VE/VCO_2 slope	-2.275(-4.700, 0.088)	-1.385(-3.013, 0.095)	0.173
VE (L/min)	4.5(-0.8, 14.0)	3.0(0.0, 7.0)	0.237
LVEF: left ventricular ejection fraction, LA: Left atrial diameter, LVD: Left ventricular end diastolic diameter, AT: anaerobic threshold, VO_2: oxygen uptake, VO_2 peak: oxygen uptake peak, METs: metabolic equivalents, O_2 Pulse: oxygen pulse, VE/VCO_2: Ventilatory equivalents for carbon dioxide, VE: ventilation			

Multiple linear regression of the CPET and the experimental group

Multiple linear regression analysis was performed, including the changes in the VO_2 AT, VO_2 peak, METs, and O_2 pulse as the dependent variables and the group (experimental group, control group) as the independent variable. Model 1 was not adjusted for any factors; Model 2 was adjusted for age, sex, and BMI; Model 3 was adjusted for age, sex, BMI, heart rate (HR), smoking history, hypertension, diabetes, creatinine, uric acid, total cholesterol, triglycerides, high-density lipoprotein, low-density lipoprotein, and left main artery disease. After further adjustment for confounding factors, the change in the VO_2 AT in the experimental group was greater than that in the control group ($\beta = 1.541$, 95% CI 0.605–2.477, $p = 0.002$), the change in the VO_2 peak in the experimental group was greater than that in the control group

($\beta = 2.446$, 95% *CI* 1.473–3.418, $p < 0.001$), the change in the METs in the experimental group was greater than that in the control group ($\beta = 0.762$, 95% *CI* 0.471–1.053, $p < 0.001$), and the change in the O₂ pulse in the experimental group was greater than that in the control group ($\beta = 1.162$, 95% *CI* 0.622–1.701, $p < 0.001$). This result further suggests that after 3 months of follow-up, the VO₂ AT, VO₂ peak, METs, and O₂ pulse increased by 1.541, 2.446, 0.762, and 1.162 units, respectively, compared with the control group (Table 6 and Fig. 3).

Table 6
Multiple linear regression of CPET and experimental group

CPET	Multiple linear regression		
	Model 1 (β)	Model 2 (β)	Model 3 (β)
VO ₂ AT(ml/min/kg)	2.035**	1.984**	1.541**
VO ₂ peak(ml/min/kg)	2.730**	2.677**	2.446**
METs	0.840**	0.821**	0.762**
O ₂ Pulse (mL/beat)	1.114**	1.152**	1.162**
AT: anaerobic threshold, VO₂: oxygen uptake, VO₂ peak: oxygen uptake peak, METs: metabolic equivalents, O₂ Pulse: oxygen pulse; Model 1 no factors were adjusted; Model 2 adjusts for age, sex, BMI; Model 3 adjusts for age, sex, BMI, HR, Smoking history, hypertension, diabetes, creatinine, uric acid, total cholesterol, triglycerides, high-density lipoprotein, low-density lipoprotein, left main artery disease			
# $p < 0.1$; * $p < 0.05$ ** $p < 0.01$			

DISCUSSION

Our primary finding was that early exercise improved the VO₂ AT, VO₂ peak, METs, and O₂ pulse in the experimental group without increasing the incidence of adverse clinical events. After 3 months of follow-up, the change in the VO₂ AT in the experimental group was greater than that in the control group ($\beta = 1.541$, 95% *CI* 0.605–2.477, $p = 0.002$). similarly, the change in the VO₂ peak ($\beta = 2.446$, 95% *CI* 1.473–3.418, $p < 0.001$), METs ($\beta = 0.762$, 95% *CI* 0.471–1.053, $p < 0.001$), and O₂ pulse ($\beta = 1.162$, 95% *CI* 0.622–1.701, $p < 0.001$) were all more pronounced in the experimental group. Our research focused mainly on early bedside cardiac rehabilitation after PCI. These results align with previous studies that have emphasized the benefits of early CR for AMI patients after PCI. At the same time, phase I cardiac rehabilitation exercise training combined with respiratory training has been shown to enhance exercise endurance and quality of life in this population^[19,20]. A study conducted in the community also showed that performing CR can improve quality of life, which includes increasing cardiac ejection fraction, exercise tolerance, and physical status in AMI patients after PCI^[21].

The physiological mechanisms underlying the observed CPET improvements may be attributed to the effects of early moderate in-bed exercise. Postoperative active movement can promote systemic blood circulation, increase cardiac preload and optimize myocardial perfusion. Additionally, early exercise may improve respiratory function by increasing tidal volume and lung compliance, thereby boosting oxygen exchange capacity. These effects collectively contribute to higher VO_2 AT, VO_2 peak. However, the VE/VCO_2 slope or VE in the experimental group did not significantly improve in the short term. The VE/VCO_2 and VE are mainly related to lung function, which may require longer intervention durations or targeted respiratory training to modify. Collectively, these results confirm the efficacy and safety of early bedside exercise for AMI patients after PCI, reinforcing the role of early CR as a critical secondary prevention strategy to reduce cardiovascular readmission and sudden death risk^[22,23].

There is a significant correlation between HR and adverse outcomes of AMI. The higher the HR, the higher the risk of cardiovascular death in patients with AMI^[24]. A study in the United States showed a close correlation between heart rate and 3-year mortality rate in AMI patients after discharge^[25]. In our study, the HR of the experimental group was greater than that of the control group. Therefore, HR was included as a confounding variable in subsequent analyses. This adjustment was critical to ensure that the observed improvements in CPET indicators were not erroneously attributed to HR fluctuations, but rather to the early exercise intervention itself.

Notably, our study did not detect significant improvements in cardiac structure or function (assessed via EF, LA, and LVD) at the 3-month follow-up. This finding can be contextualized by the pathophysiological timeline of AMI: ventricular remodeling begins within hours of infarction, progressing from localized myocardial damage to global left ventricular structural changes, and is accompanied by myocardial hypertrophy and systolic dysfunction. The lack of functional improvement may be explained by two key factors. First, the 3-month intervention duration may be insufficient to reverse established remodeling processes, as cardiac structural adaptations typically require longer-term interventions to become detectable. Second, heart failure is a common complication in AMI patients. However, in patients with heart failure, CR can significantly improve the LVEF^[26–28]. However, in our study, patients with heart failure were excluded. Therefore, the absence of an effect of early CR training on the cardiac function of AMI patients after PCI may be associated with the short duration of the intervention and the exclusion of heart failure patients.

Importantly, there were no adverse events in our study. In addition, with the advancement of PCI technology, postoperative complications have significantly decreased. However, intrastent thrombosis and malignant arrhythmia were still identified as the strongest predictors of mortality. In the present study, exercise training did not increase the incidence of recurrent myocardial infarction among patients after PCI, indicating that early CR training was safe for such patients, which was consistent with previous literature reports^[21,29].

This study has several notable strengths. First, it focused on early bedside CR. This is a practical, hospital-based intervention that is easily implementable in clinical settings. Second, we employed

objective CPET indicators (e.g., VO_2 AT, O_2 pulse) to assess intervention effects, which are more sensitive to early cardiopulmonary adaptations than standard cardiac function measures (e.g., EF). Third, we adjusted for HR as a confounding variable, enhancing the robustness of our causal inferences regarding the intervention's effects on CPET outcomes. Despite these strengths, our study has several limitations that should be considered when interpreting the results. First, the study included only patients who could perform and agreed to engage in exercise training. Second, the study only included 3 months of follow-up. There is no research on the long-term effects on cardiopulmonary function and cardiac structure. Finally, the study was a single-center trial with a small sample size. In the future, we should conduct multi-center and larger-scale studies to enhance applicability. In addition, we need to extend the follow-up period to assess the long-term effects on cardiac structure and function. Furthermore, researching the potential mechanisms for improving cardiac function (for example, assessing myocardial fibrosis through cardiac MRI) can inform the design of targeted intervention measures to enhance long-term cardiac remodeling outcomes.

CONCLUSION

This study demonstrated that early phase I CR exercise training combined with respiratory training improved cardiopulmonary fitness (VO_2 AT, VO_2 peak, O_2 pulse) and exercise capacity without increasing the incidence of cardiovascular events or prosthesis dysfunction among AMI patients with PCI. However, no significant changes in cardiac function were observed in patients with short-term intervention, which emphasizes the necessity of conducting long-term follow-up studies to determine long-term effects. Based on this study, early CR can be used as a routine intervention plan for AMI patients after PCI. But this study is a single-center study and has limitations. Future research should expand the sample size and include patient groups from different regions and with different disease severity levels to improve the generalizability of the findings.

Abbreviations

AMI: acute myocardial infarction; PCI: percutaneous coronary intervention; CPET: cardiopulmonary exercise testing; CR: Cardiac rehabilitation; AT: anaerobic threshold; METs: metabolic equivalents; O_2 Pulse: oxygen pulse; VE/VCO_2 : Ventilatory equivalents for carbon dioxide; VE:ventilation; VO_2 :oxygen uptake; VO_2 peak: peak oxygen uptake; LVEF: left ventricular ejection fraction; LA: Left atrial diameter, LVD: Left ventricular end diastolic diameter, BMI: body mass index; TG: triglycerides; TC: total cholesterol; LDL-C: low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol; UA: uric acid; Cr: serum creatinine; SBP: systolic blood pressure; DBP: diastolic blood pressure; HR: heart rate; OSAS: obstructive sleep apnea syndrome.

Declarations

Acknowledgements

Not Applicable.

Ethic approval and consent to participate

This study was approved by Yulin First People's Hospital (YLSY-IRB-SR-2022019). All procedures were performed in accordance with ethical standards. Written consent was obtained from all participants after they had been informed of the objectives, benefits, medical items and confidentiality agreement regarding their personal information.

Availability of data and materials

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

Competing interests

The authors declare that they have no competing interests, and all authors confirm its accuracy.

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Authors' contributions

Jun-Ting Luo and Ying-Feng conceived the study. Jun-Ting Luo analysis and interpretation. Jun-Ting Luo and Ying-Feng contributed equally to this work. Kai-Fang Pang and Jian-Ping Tan contributed to data collection. Chun-Mei Zeng and Yan-Mei Zhao contributed to the study conceptions and design. All authors read and approved the final version of the manuscript.

Consent for publication

Not applicable.

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Figures

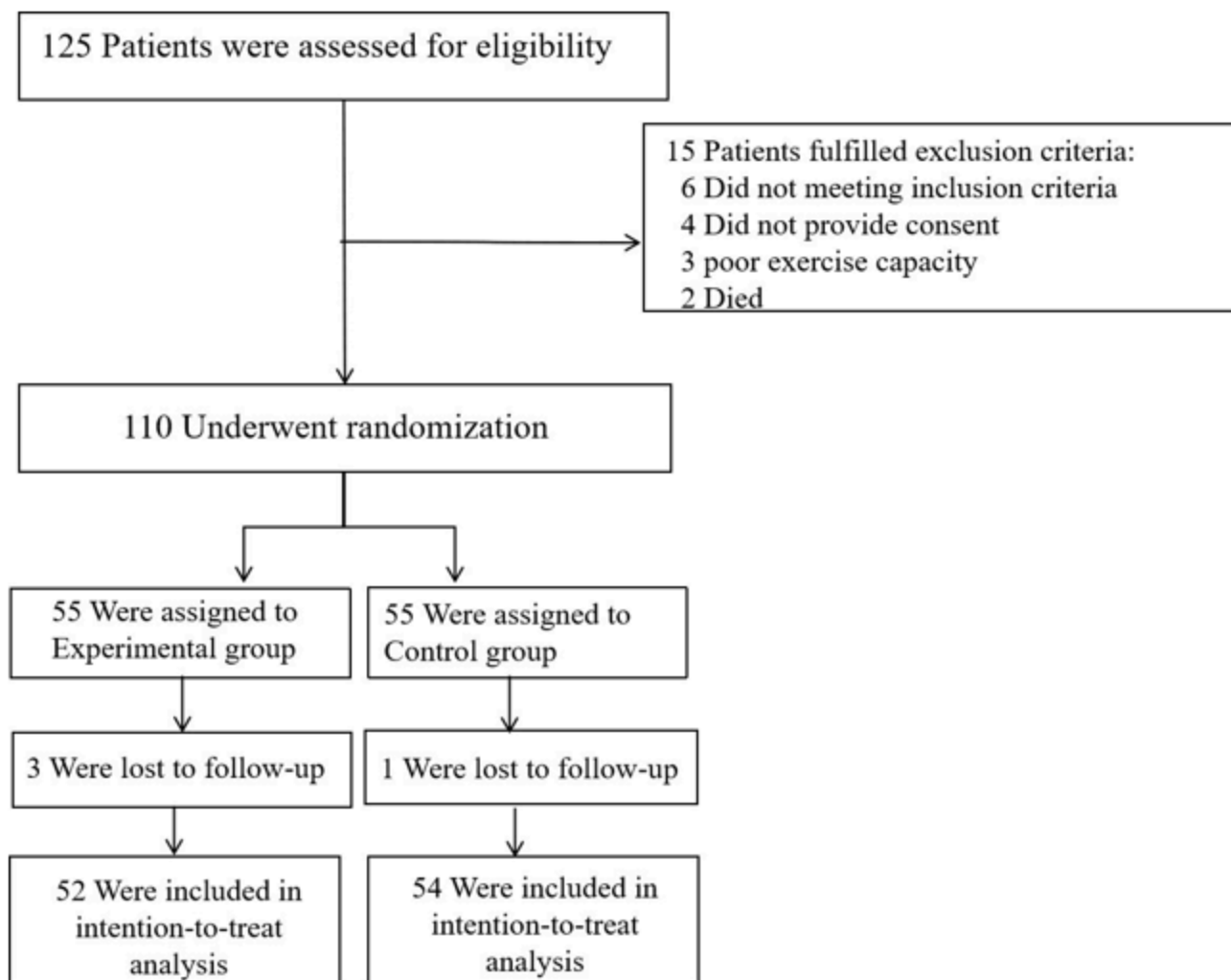


Figure 1

Flow diagram of our study☐CONSORT 2010☐

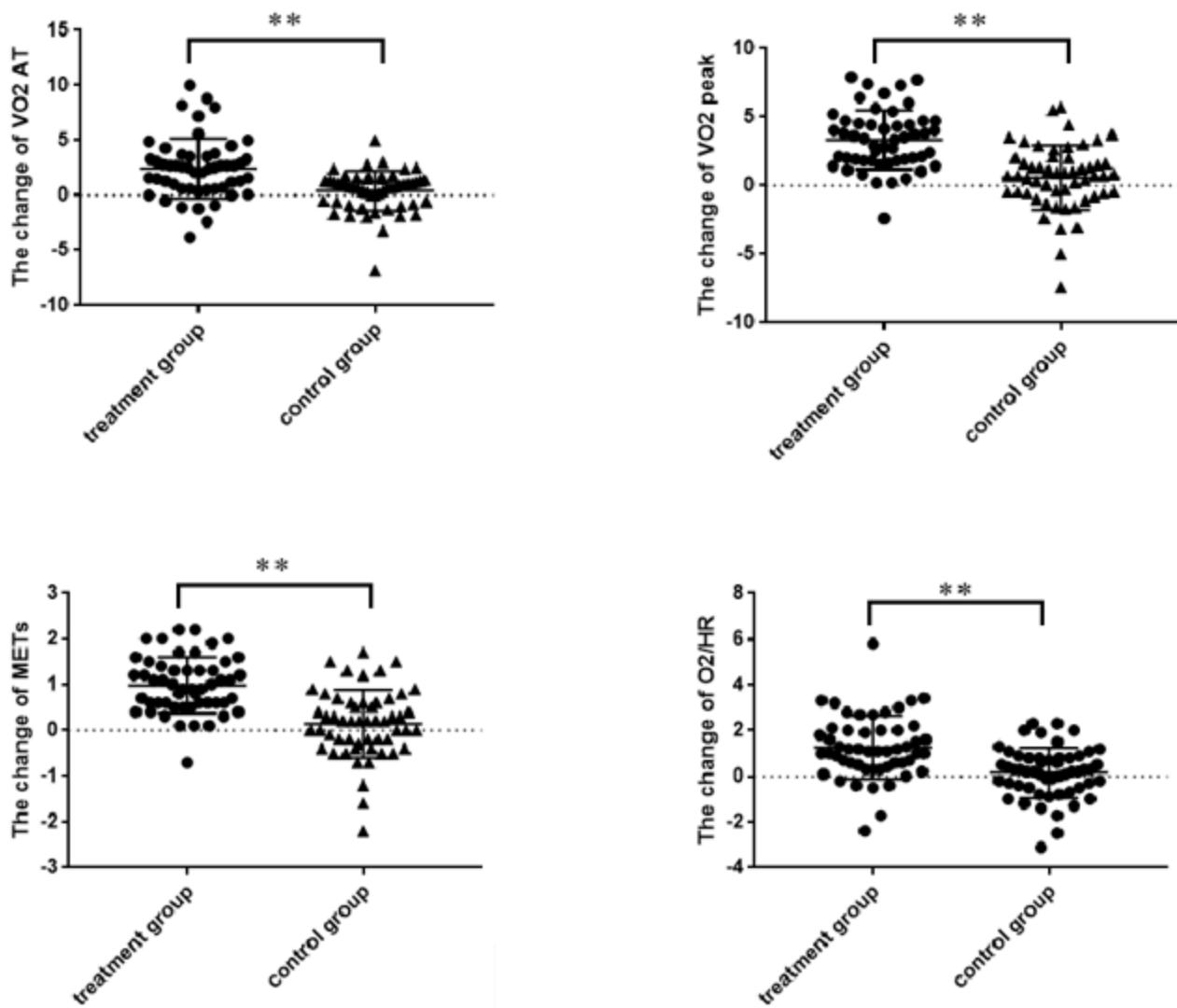


Figure 2

Changes in CPET between the experimental and control groups. A: Changes in VO_2 AT; B: Changes in VO_2 peak; C: Changes in METs; D: Changes in O_2 pulse

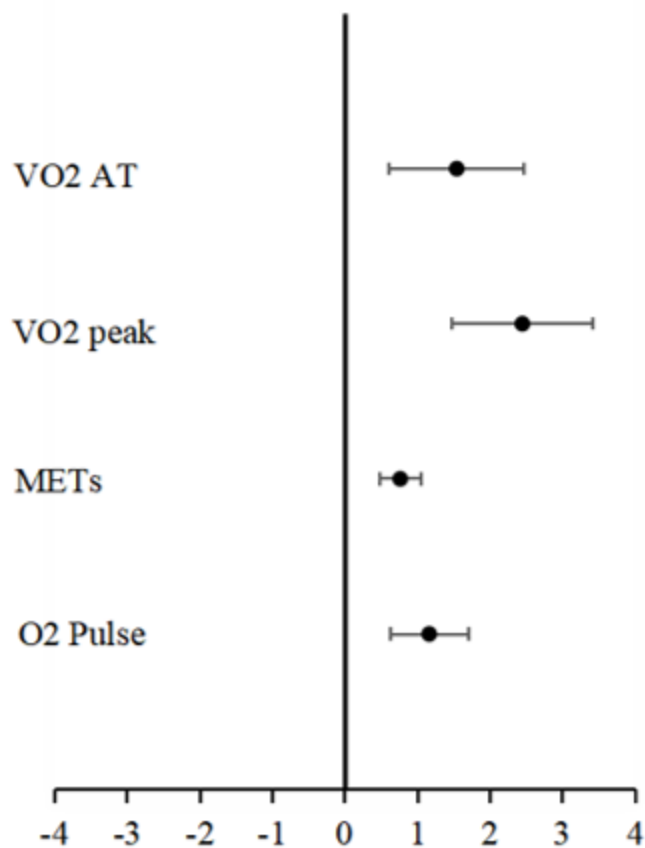


Figure 3

Multiple linear regression of CPET and experimental group

Supplementary Files

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- [Supplementary1CONSORT2010Checklist.doc](#)