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The Effect of Cardiopulmonary Exercise Ability to Clinical Outcomes of Patients with Coronary Artery Disease Undergoing Percutaneous Coronary Intervention

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Video Abstract

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Objective: To analyze the relationship between the cardiopulmonary function and prognosis of patients with coronary heart disease after percutaneous coronary intervention (PCI).

Methods: A total of 153 patients with coronary heart disease who underwent PCI from January 2018 to April 2020 were enrolled in this study. Through careful assessment, cardiopulmonary exercise test (CPX) was performed 5 to 7 days after PCI. Patients were followed up every 3 months by outpatient examination or telephone visiting for 3 years after discharge. Clinical outcomes were followed up, including cardiac death, rehospitalization, heart failure, atrial fibrillation, stroke and transient ischemic attack. A single clinical event was defined as a poor prognosis and divided into a good prognosis group and a poor prognosis group according to the prognosis. By comparing the cardiorespiratory fitness (CRF) variables and clinical parameters, the variables that may affect the prognosis of patients were determined.

Results: CRF decreased significantly in the poor prognosis group, and peak VO2, VO2/kg AT, PETCO2 and OUES decreased compared with the good prognosis group, and the differences were statistically significant. Heart rate reserve (HRR) increased in the poor prognosis group compared with the good prognosis group, and the difference was statistically significant. Among them, peak VO2 and acute myocardial infarction were independent risk factors for poor prognosis.

Conclusion: Peak VO2 is an independent risk factor for the prognosis of cardiovascular disease after PCI for coronary heart disease.

Keywords: coronary heart disease, cardiopulmonary exercise testing, percutaneous coronary intervention

Coronary artery disease (CAD), one of the most common cardiovascular diseases, severely poses a threat to public health. Generally, for patients suffering myocardial ischemia, PCI is considered as one of the most important medical choice; however, fewer improvements are found in some of them due to poor prognosis.[1] In previous studies, we have known that coronary flow reserve fractional,[2] SYNTAX score, CTO (≥2 artery occlusion),[3] and HALP score[4] are associated with adverse cardiovascular events in patients. These factors can partially indicate the long-term prognosis of patients, but they are scattered and lack of comprehensiveness.

Cardiopulmonary exercise testing (CPET) is an objective predictor of cardiopulmonary exercise ability and exercise capacity for patients with CAD and has proved to be a crucial approach to reflect myocardial ischemia and evaluate the effect of intervention therapy, which it can comprehensively indicate the long-term prognosis of patients.[5] We reviewed information on CAD patients and cardiopulmonary exercise ability results of patients who had undergone PCI before discharge to explore the association between cardiopulmonary exercise ability of CAD patients after PCI and their medium-term and long-term prognoses.

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Subjects and Methods

Subjects

A total of 153 patients who had undergone PCI in the Second Affiliated Hospital of Anhui Medical University from January 2018 to April 2020 were enrolled in this study. CPX was performed at 5–7 days after PCI during hospitalization. All patients will be followed up regularly, any follow-up clinical adverse events, including cardiac death, readmission, heart failure, atrial fibrillation, stroke, and transient ischemic attacks, were identified as the poor prognosis and on the contrary, the good prognosis was defined without events. Inclusion criteria for CPX: 1. All eligible patients with CAD should meet the diagnostic criteria of the European Heart Association[6] and the Chinese guidelines for percutaneous coronary intervention (2016);[7] 2. Patients who underwent PCI for the first time after a successful operation; 3. Postoperative shunt grade III, postoperative residual stenosis less than 20%. Exclusion criteria: 1. Uncontrolled heart failure (New York Heart Association Class III–IV); 2. Comorbidities such as moderate and severe anemia, tumor, and lung disease; 3. Combined with valvular heart disease; 4. Patients who cannot reach the anaerobic threshold during exercise. The First Affiliated Hospital of Anhui Medical University Ethics Committee approved this study (approval number: PJ 2023–14-75). All procedures involving human subjects adhered to the 1964 Declaration of Helsinki. All patients signed informed consent forms before the study. There is no interest conflict for authors. The data are available.

Methods

Cpet

In this study, patients received symptom-limited CPX with reference to the Italian COSMED system. The treadmill RAMP test protocol was used for patients on beta-blockers. After knowing about the exercise program and providing informed consent, the participants first underwent a static pulmonary function test and electrocardiogram examination and then tried a mask on a bicycle. After a 3-min rest, they began to warm up for 3 min at 0 W. After that, 10–15 W/min increase in workload. Patients maintained pedaling speed (60r/min) during the exercise test while their ECG, blood pressure, and finger oxygenation levels were monitored. The change in HR from rest to peak exercise is represented by HRR. Expiratory gas analysis was continuously monitored, with peak VO2 (mL/kg/min) defined as the highest VO2 attained during exercise. The anaerobic threshold (mL/kg/min) was calculated using the V-slope method. The system should be calibrated before every exercise test. Calibration of airflow, volume, and O2 and CO2 analyzers is included.

Data Collection

Clinical data include gender, age, acute myocardial infarction condition, BMI, smoking, alcohol consumption, hypertension, diabetes, hyperlipidemia, low-density lipoprotein, color Doppler echocardiography left ventricular ejection fraction, and LVEF. The data of the CPET regimen include maximal power, peak oxygen uptake, peak oxygen consumption per kilogram, peak respiratory exchange rate (RER), end-expiratory partial pressure of oxygen (PET02), end-expiratory partial pressure of carbon dioxide (PETCO2), vital capacity (VC), oxygen pulse (VO2/HR), peak metabolic equivalent (MET), HRR, peak systolic pressure, diastolic pressure, AT-kg oxygen uptake, VE/VCO2 slope, oxygen uptake efficiency slope (OUES), vital capacity (VC), forced expiratory volume in one second (FEV1/FVC), maximal ventilation volume (MVV), and respiratory reserve (BR).

Follow-Up of Clinical Outcomes

Post-treatment results, including cardiac death, readmission, heart failure, atrial fibrillation, stroke, and transient cerebral ischemia, were followed up through routine clinical visits or telephone interviews for 3 years until death or April 2023. Any clinical outcomes that indicated poor prognosis.

Statistics

In the study, R4.2.1 software was applied for statistical analysis, while continuous variables were shown as mean ± SD. *T*-test or Mann–Whitney *U*-test comparisons were performed. Categorical variables were compared using $\chi 2$ test or Fisher's exact test. Variables with significant statistical differences were included in the multivariate logistic regression analysis. $P < 0.05$ was defined as a significant statistical difference.

Results

Comparison of the Clinical Data Between the Two Groups

The clinical characteristics of the eligible patients (N=153) are shown in Table 1, with 78.4% (N = 120) having a good prognosis and 21.6% (N=33) having a poor prognosis. Adverse prognostic events included cardiac death, readmission, heart failure, atrial fibrillation, stroke, and transient cerebral ischemia. As shown in the table, there was a statistically significant difference between 51.7% of patients with a good prognosis and 81.8% of those with a poor prognosis. No significant differences were observed between the two groups in terms of comorbidities and basic characteristics such as BMI, low-density lipoprotein (LDL), left ventricular end-diastolic diameter (LVDD), and ejection fraction (EF).

Table 1 Clinical Characteristics of the Subjects

	Good Prognosis (N=120)	**Poor Prognosis (N=33)**	**P**
Acute myocardial infarction			
N	58 (48.3%)	6 (18.2%)	0.004
Gender			
Male	85 (70.8%)	21 (63.6%)	0.561
Female	35 (29.2%)	12 (36.4%)	
Age			
Mean (SD)	60.1 (14.3)	65.2 (16.7)	0.114
Median [Min, Max]	61.5 [24.0, 91.0]	67.0 [19.0, 93.0]	
BMI			
Mean (SD)	25.0 (3.45)	24.1 (4.18)	0.451
Median [Min, Max]	24.2 [18.4, 33.0]	24.2 [17.2, 31.2]	
Hypertension			
N	47 (39.2%)	9 (27.3%)	0.293
Diabetes mellitus			
N	87 (72.5%)	26 (78.8%)	0.614
Hyperlipidemia			
N	97 (80.8%)	24 (72.7%)	0.44
Family history of CAD			
N	117 (97.5%)	33 (100%)	0.835
Smoking history			
N	66 (55.0%)	23 (69.7%)	0.173
Quit smoking	22 (18.3%)	2 (6.1%)	
Alcohol history			
N	82 (68.3%)	23 (69.7%)	0.977
Less	20 (16.7%)	6 (18.2%)	
Excessive	5 (4.2%)	1 (3.0%)	
Quit alcohol	13 (10.8%)	3 (9.1%)	
Low-density lipoprotein			
Mean (SD)	2.45 (0.854)	2.08 (0.738)	0.208
Median [Min, Max]	2.39 [0.890, 4.28]	2.05 [1.03, 3.37]	
LVEDD			
Mean (SD)	49.1 (5.78)	47.4 (6.91)	0.317
Median [Min, Max]	49.6 [33.7, 60.4]	48.1 [34.8, 56.9]	
EF			
Mean (SD)	65.1 (9.34)	61.9 (12.0)	0.206
Median [Min, Max]	65.9 [34.7, 97.2]	63.2 [21.4, 75.2]	
Follow-up (week)			
Mean (SD)	82.1 (32.3)	83.3 (34.5)	0.851
Median [Min, Max]	93.4 [2.70, 158]	95.1 [6.90, 137]	

Comparison of the CPET Between the Two Groups

CPET results vary among patients with different prognoses, as shown in Table 2. Compared with patients in the poor prognosis group, the peak power level was notably higher in the good prognosis group, with a statistically significant difference between them. The same results were found in terms of Peak_VO2/kg level, PetCO2 level, HRR, and OUE.

Table 2 Differences in CPET Between Patients with Good and Poor Prognoses

	Good Prognosis (N=120)	Poor Prognosis (N=33)	P
Peak power level			
Mean (SD)	92.0 (35.7)	58.5 (27.6)	<0.001
Median [Min, Max]	88.5 [10.0, 185]	54.0 [14.0, 140]	
Peak VO2			
Mean (SD)	1370 (401)	994 (342)	<0.001
Median [Min, Max]	1320 [565, 2610]	940 [382, 1680]	
Peak VO2/kg			
Mean (SD)	19.9 (4.45)	15.3 (4.42)	<0.001
Median [Min, Max]	19.5 [9.60, 31.7]	14.9 [6.50, 28.1]	
RER			
Mean (SD)	1.08 (0.134)	0.994 (0.0704)	<0.001
Median [Min, Max]	1.10 [0.900, 1.80]	1.00 [0.900, 1.10]	
Peak PetO2			
Mean (SD)	115 (10.5)	113 (6.31)	0.331
Median [Min, Max]	113 [100, 200]	112 [100, 125]	
Peak PetCO2			
Mean (SD)	39.6 (9.58)	36.3 (6.32)	0.021
Median [Min, Max]	38.0 [24.0, 100]	37.0 [23.0, 49.0]	
VE			
Mean (SD)	48.0 (15.2)	35.4 (13.5)	<0.001
Median [Min, Max]	47.3 [15.9, 104]	32.8 [8.30, 62.9]	
Peak VO2/HR			
Mean (SD)	10.7 (2.60)	8.82 (2.31)	<0.001
Median [Min, Max]	10.8 [4.10, 18.1]	9.20 [3.90, 12.7]	
Peak MET			
Mean (SD)	5.73 (1.32)	4.41 (1.24)	<0.001
Median [Min, Max]	5.60 [2.80, 11.0]	4.30 [1.90, 8.00]	
Peak HR			
Mean (SD)	128 (21.4)	111 (18.7)	<0.001
Median [Min, Max]	130 [84.0, 177]	109 [71.0, 150]	
HRR			
Mean (SD)	33.2 (19.2)	43.6 (21.7)	0.016
Median [Min, Max]	35.0 [−2.00, 85.0]	42.0 [10.0, 93.0]	
Peak systolic pressure			
Mean (SD)	168 (27.3)	157 (29.6)	0.069
Median [Min, Max]	164 [108, 233]	160 [89.0, 210]	
Peak diastolic pressure			
Mean (SD)	82.3 (16.6)	76.7 (18.4)	0.13
Median [Min, Max]	81.5 [36.0, 131]	76.5 [31.0, 131]	
VO2/kg AT			
Mean (SD)	15.0 (3.03)	12.6 (4.04)	0.008
Median [Min, Max]	14.9 [7.10, 25.3]	13.0 [2.50, 21.2]	

(*Continued*)

Table 2 (Continued).

	Good Prognosis (N=120)	Poor Prognosis (N=33)	P
VE/VCO2			
Mean (SD)	28.1 (7.22)	29.9 (10.5)	0.37
Median [Min, Max]	28.7 [−18.7, 49.3]	28.8 [0, 62.6]	
OUES			
Mean (SD)	2020 (526)	1710 (466)	0.002
Median [Min, Max]	1940 [1090, 4140]	1790 [753, 2740]	
VC			
Mean (SD)	3.23 (0.891)	2.87 (0.911)	0.061
Median [Min, Max]	3.21 [1.39, 5.30]	2.75 [1.22, 4.50]	
FEV1			
Mean (SD)	2.48 (0.779)	2.23 (0.862)	0.164
Median [Min, Max]	2.47 [0.720, 4.54]	2.03 [0.640, 4.13]	
FEV1/FVC			
Mean (SD)	77.7 (10.0)	76.8 (16.3)	0.779
Median [Min, Max]	79.1 [27.1, 95.8]	80.7 [8.30, 94.7]	
MVV			
Mean (SD)	92.5 (31.8)	74.7 (32.2)	0.011
Median [Min, Max]	92.0 [30.7, 192]	73.5 [22.8, 143]	
RF			
Mean (SD)	33.6 (8.00)	32.8 (9.28)	0.671
Median [Min, Max]	33.3 [15.4, 67.4]	31.8 [18.6, 65.9]	
BR			
Mean (SD)	44.5 (20.3)	44.2 (24.6)	0.952
Median [Min, Max]	46.9 [−49.4, 78.1]	48.7 [−6.80, 88.6]	

Logistic Regression Analysis

To further analyze the risk factors that affect the prognosis of the patients, multivariate logistic regression analysis was adopted, including the variables of acute myocardial infarction, gender, Peak_VO2/kg, VO2/kg AT, PetCO2, HRR, and OUES, according to the results of univariate analysis and the characteristics of the disease. The results are shown in Table 3 and Figure 1. Multiple regression analysis suggested that MI and peak VO2/kg were independent risk factors for prognosis. The incidence of adverse events was significantly higher in patients with MI than in those without MI. The peak VO2/kg level of patients with a good prognosis was significantly higher than that of patients with a poor prognosis.

Table 3 The Logistic Regression Analysis

		OR (univariable)	OR (multivariable)
Acute myocardial infarction	**N**		
	Y	4.21 (1.62–10.93, p=0.003)	5.17 (1.62–16.56, p=0.006)
Gender	**Male**		
	Female	1.39 (0.62–3.12, p=0.428)	1.67 (0.57–4.89, p=0.351)
Peak VO2/kg	**Mean ± SD**	0.78 (0.70–0.87, p<0.001)	0.83 (0.70–0.99, p=0.042)
VO2/kg AT	**Mean ± SD**	0.78 (0.68–0.90, p<0.001)	0.92 (0.76–1.12, p=0.396)
Pet CO2	**Mean ± SD**	0.94 (0.87–1.00, p=0.051)	0.98 (0.92–1.05, p=0.596)
HRR	**Mean ± SD**	1.03 (1.01–1.05, p=0.011)	1.01 (0.98–1.03, p=0.579)
OUES	**Mean ± SD**	1.00 (1.00–1.00, p=0.004)	1.00 (1.00–1.00, p=0.981)

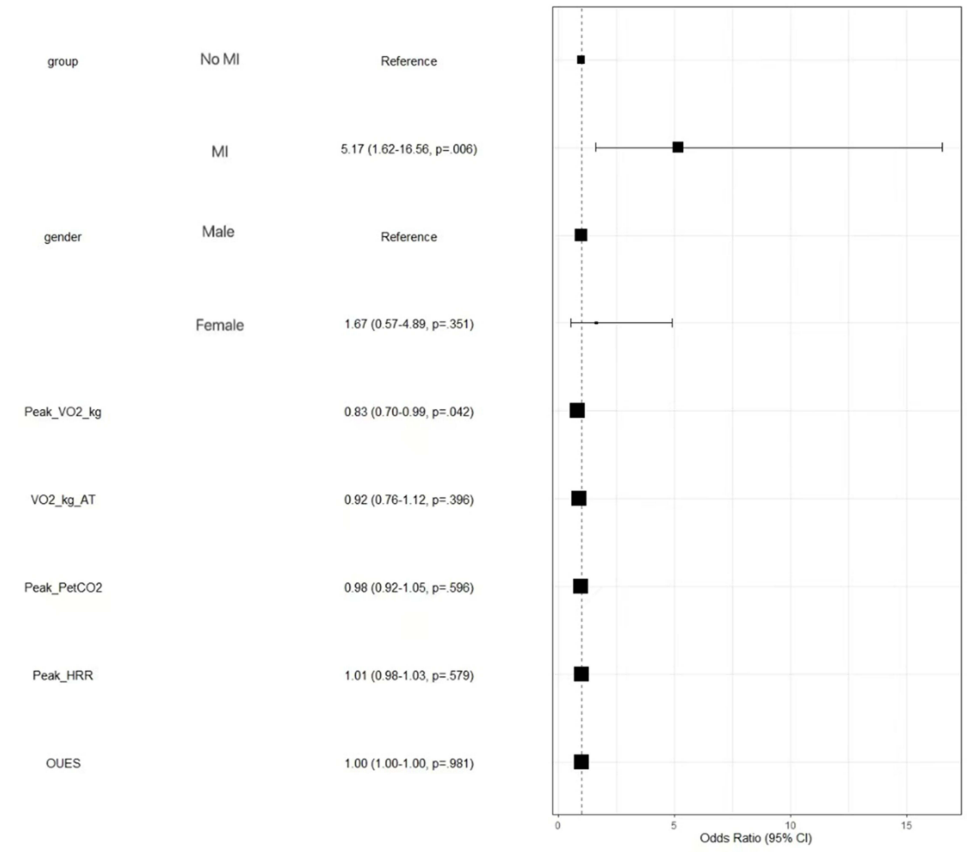

Figure 1 The forest plot of Logistic regression.

Discussion

Reduced exercise capacity is a critical indicator of poor prognosis and disability in patients with cardiovascular disease and chronic heart failure.[8] To date, the effect of decreased exercise capacity on the clinical outcome of coronary heart disease has not been fully illustrated. Keteyian et al suggested[9] that regardless of gender, the VO2 peak is one of the strongest predictors of all-cause mortality, with an increase of 1 mL/min/kg in the VO2 peak and an approximately 15% reduction in the risk of death. Several studies have reported that decreased exercise capacity as assessed by CPX is an important indicator of poor outcomes in patients with coronary artery disease, including AMI.[10,11] Our study also showed that CRF in the group with poor prognosis was significantly lower than that in the group with good prognosis. Reduction was also observed in the poor prognosis group in terms of peak VO2, VO2/kg AT, PETCO2, and OUES. HRR in the poor prognosis group was significantly higher than that in the good prognosis group. Peak VO2 and a history of myocardial infarction were independent risk factors for poor prognosis. Peak VO2 was defined as the highest oxygen uptake obtained during exercise. A number of studies[12,13] have demonstrated that non-invasively determined peak cardiac output is considered a separate predictor of improved outcomes with prognostic benefit from peak VO2. Previous studies[14] showed a linear association between HRR, VO2/kgAT, and peak VO2/kg. In this study, we also found that there were statistical differences for these three parameters in two groups. Studies have suggested an association between PETCO2 and cardiac systolic function, a cardiac biomarker.[15] Decreased PETCO2 is an independent risk factor for readmission in patients with myocardial infarction,[16] and it can also predict vascular reactivity in patients with pulmonary hypertension. Our data are somewhat different from the previous ones, it has suggested that PETCO2 is lower in the poor prognosis group than in the good prognosis group, but PETCO2 is not an independent predictor of poor prognosis according to multivariate logistic analysis. The OUES Index was originally used to assess cardiorespiratory reserve function in children with heart disease,[17] which is proportional to CRF, with higher values indicating better CRF. The value of OUES decreased linearly with age,[18] which was also confirmed in this study. Multivariate logistic analysis showed that

peak VO2/kg and intervention therapy due to acute myocardial infarction (AMI) were independent predictors of poor outcomes, suggesting that a decrease in peak VO2 in patients with AMI after PCI may result in poor clinical outcome in the CPX test early after the operation. This may be because PCI procedures can lead to coronary artery spasm, endothelial cell injury, or even restenosis or thrombosis, especially in patients with acute myocardial infarction who have a poor prognosis after PCI.[19] Liu et al[20] also pointed out that the decrease in cardiorespiratory reserve function in patients with acute myocardial infarction is characterized by an obvious decrease in VO2. Sato et al[21] reported a significant decrease in cardiopulmonary exercise capacity in patients with acute myocardial infarction after PCI. VO2, a clinically sensitive indicator of cardiovascular reserve function,[22] is an accurate measure of exercise tolerance in patients. The study reported[23] that acute myocardial infarction results in exercise capacity decrease, with peak VO2≤12 in 28% of patients, which is consistent with our study.

This study has some limitations. First, this was a single-center study with a small sample size. Second, patients who could not tolerate the CPX test were excluded from this study, and patients with more severe diseases (such as shock requiring mechanical support and severe heart failure) could not be enrolled. Third, we did not assess preoperative exercise capacity in some patients with myocardial infarction before the onset of the disease. Fourth, the duration of follow-up was relatively short.

Conclusion

The study illustrated that the cardiopulmonary function of patients with poor prognosis after PCI was lower than that of patients with good prognosis before discharge. VO2 at the peak of the key CPX variable was identified as an independent risk factor for cardiovascular outcomes. Therefore, in the future work, we need to focus more on the effect of Peak VO2 to the clinical outcomes of patients after PCI. Future evidence with a larger sample size is required to validate these results.

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Disclosure

The authors declare that they have no competing interests.

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