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Original Article

Predictive value of cardiopulmonary exercise test parameters for atrial fibrillation risk in hypertrophic cardiomyopathy patients

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Abstract: Objective: To investigate the relationship between cardiopulmonary exercise test (CPET) parameters and the occurrence of atrial fibrillation (AF) in patients with hypertrophic cardiomyopathy (HCM). Methods: A retrospective study was conducted on 160 HCM patients hospitalized at Quanzhou First Hospital Affiliated to Fujian Medical University from June 2020 to December 2023. Patients were divided into AF (n=62) and non-AF (n=98) groups. Univariate analysis and logistic regression were used to compare demographic and CPET parameters between groups. A nomogram model was developed to predict AF risk. Results: AF prevalence was 38.75%. Peak Oxygen Uptake per kilogram (Peak VO_2/kg), Metabolic Equivalent of Task (METs), and Oxygen Uptake at Anaerobic threshold per kilogram (VO_2/kg AT) were lower in the AF group compared to the non-AF group, while Ventilation to Carbon Dioxide Production Slope (VE/VCO_2 slope) was higher (all P<0.05). Pearson correlation analysis showed a significant negative correlation between Peak VO_2/kg and New York Heart Association (NYHA) classification (r=-0.231, P=0.003). Logistic regression identified NYHA classification, smoking history, and VE/VCO_2 slope as risk factors for AF, while Peak VO_2/kg, METs, and VO_2/kg AT were protective factors (P<0.05). The nomogram model had an area under curve (AUC) of 0.867 (0.808-0.925), demonstrating good calibration and clinical utility. Conclusion: CPET parameters, combined with clinical indicators, can effectively predict AF risk in HCM patients. The nomogram model provides a valuable tool for clinical risk assessment.

Keywords: Hypertrophic cardiomyopathy, cardiopulmonary exercise test, peak oxygen consumption, atrial fibrillation

Introduction

Hypertrophic cardiomyopathy (HCM) is a common heterogeneous hereditary cardiomyopathy, classified into obstructive and non-obstructive types [1]. It is characterized by primary left ventricular hypertrophy, particularly at the base of the septum, with pathological features including irregular cardiomyocyte arrangement, fibrous scarring, and microvascular structural and functional abnormalities. These manifestations lead to heart failure and arrhythmias [1, 2]. HCM has an insidious onset, with some patients remaining asymptomatic for an extended period, making early clinical recognition challenging. Echocardiography represents the primary diagnostic modality for HCM. The global prevalence of HCM is approximately 1 in 500 adults, and it is the leading cause of exercise intolerance and sudden cardiac death [3]. It is estimated that over one million individuals in China are affected by HCM, with atrial fibrillation (AF) being a common arrhythmia observed in these patients [4]. Epidemiological studies have indicated that the prevalence of AF in patients with HCM is approximately 10-30%, making it a significant prognostic factor in HCM patients [5, 6]. The risk of AF in HCM patients is 4-6 times higher than in those without HCM [7]. This increased risk is associated with the progression of left ventricular diastolic disorder to myocardial diastolic dysfunction and left ventricular outflow tract obstruction in HCM patients [7]. Furthermore, HCM patients with AF exhibit an eightfold increased risk of stroke compared to those with sinus rhythm [8].

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Additionally, HCM patients over the age of 50 experience an increase in left atrial diameter and a decline in ventricular function with advancing age, which further raises their stroke risk and impacts prognosis. In AF patients, left ventricular filling time is shortened, exacerbating left ventricular diastolic dysfunction [9]. This not only reduces the quality of life but also results in frequent hospitalizations, and an increased risk of ischemic stroke and cardiovascular death. Liu L et al. [10] demonstrated that HCM patients with AF are at an elevated risk of thromboembolism, which correlates with increased mortality. It is therefore of particular importance to identify high-risk HCM patients who may develop AF at an early stage, to facilitate effective clinical management and to inform prognosis judgement.

Cardiopulmonary exercise testing (CPET) is based on the principles of cardiopulmonary coupling theory. The test entails lung ventilation and the exchange of oxygen and carbon dioxide between the lungs and blood. Oxygen and carbon dioxide are transported and exchanged between blood and capillaries, as well as surrounding muscle tissues, through four distinct processes [11]. The indexes of cardiopulmonary function were collated to evaluate the overall functionality and reserve capacity of the cardiopulmonary system under specific exercise conditions. CPET is a non-invasive, objective, and quantitative assessment of cardiopulmonary function. The core index of CPET is Peak Oxygen Uptake (Peak VO_2), which represents the maximum oxygen uptake capacity of the human body during extreme exercise and serves as the limit of the human body's oxygen supply capacity [12]. Furthermore, previous studies have demonstrated that peak VO_2 is an independent risk factor for mortality in HCM patients, and certain CPET indices correlate with the degree of coronary artery stenosis [13, 14]. While numerous studies have validated the use of CPET in the diagnosis and treatment of HCM, the relationship between CPET indices and the incidence of AF in HCM patients remains unclear. Furthermore, there is a paucity of research exploring the potential of CPET in predicting the risk of AF occurrence in HCM patients.

The nomogram model is a statistical tool that quantifies multiple risk factors and visually display the probability of patients developing related diseases in the form of a total score. This model is helpful for identifying high-risk groups and developing appropriate intervention strategies [15]. This study aims to explore the relationship between core CPET indicators and AF in HCM patients and to develop a nomogram risk model based on CPET results. The resulting tool will provide a new and effective method for clinical risk assessment, guiding the treatment and management of HCM patients and reducing the incidence of AF and its associated complications.

Materials and methods

Research subjects

A retrospective study was conducted on 160 patients with HCM hospitalized at Quanzhou First Hospital Affiliated to Fujian Medical University between June 2020 and December 2023.

Inclusion criteria: (1) Left ventricular wall thickness ≥15 mm on echocardiography or ≥13 mm with a family history, meeting the diagnostic criteria for HCM [16]; (2) Age between 18-85 years; (3) Consciousness with normal communication ability; (4) Stable vital signs, allowing for CPET; (5) Complete clinical data.

Exclusion criteria: (1) Left ventricular wall thickening due to hypertension or aortic stenosis; (2) Patients with acute myocardial infarction; (3) Intolerance to CPET; (4) Previous cardiac surgery; (5) Resting blood pressure ≥180/100 mmHg, with poor drug control. This study was approved by the Ethics Committee of Quanzhou First Hospital Affiliated to Fujian Medical University.

Grouping basis

Patients were classified into two groups based on the occurrence of AF. The diagnostic criteria for AF included documentation of typical f-waves on 24-hour ambulatory electrocardiogram (ECG) monitoring, combined with clinical history and confirmation by a qualified physician during hospital admission.

Data collection

Data were collected from the electronic medical record system, including gender, age, Body Mass Index (BMI), the New York Heart Association (NYHA) Heart function grade, family history of HCM, history of hypertension, diabetes, coronary heart disease, hyperlipidemia, smok-

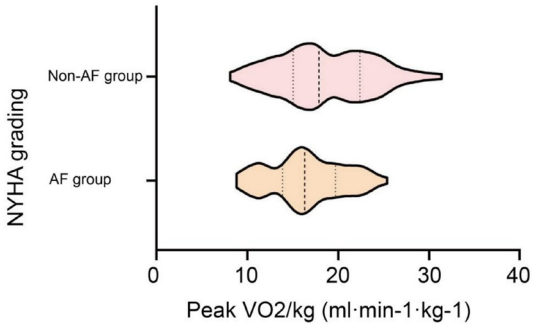

Figure 1. Comparison of Peak VO_2/kg between the AF group and non-AF group. Abbreviations: NYHA, New York Heart Association; AF, atrial fibrillation; Peak VO_2/kg, Peak Oxygen Uptake per kilogram.

ing and alcohol consumption. A history of smoking was defined as smoking ≥1 cigarette per day for 1 year, or long-term smoking with cessation for less than 6 months; A history of alcohol consumption was defined as consumption times ≥2 times a week for males and ≥1 times a week for females, including liquor, beer, and wine.

Cardiopulmonary exercise testing (CPET)

The symptom-limited maximum CPET was conducted in accordance with the standard continuous increasing power protocol prescribed by the UCLA Medical Center. Prior to the commencement of CPET, the instrument underwent airflow calibration using a two-point system, with high and low concentrations of oxygen and carbon dioxide calibrated independently. Following a series of rigorous flow rate calibrations, the metabolic simulator was evaluated. Basic lung function and blood pressure were measured two hours after lunch, ensuring that any abnormalities in blood pressure, blood sugar, and heart rate were excluded. A treadmill exercise test with standard incremental power was conducted utilizing the Quark PFT4 ergo cardiopulmonary exercise system (COSMED, Italy). The protocol comprises three minutes of rest, a three-minute no-load warm-up at a speed of (60±5) r/min, followed by an incremental load exercise at 20 w/min until volitional exhaustion. The recovery phase was then initiated, with subjects monitored for a minimum of five minutes.

The following CPET parameters were recorded: Peak Oxygen Uptake per kilogram (Peak VO_2/kg), Metabolic Equivalent of Task (METs), Heart Rate Reserve (HRR), Peak Heart Rate (Peak HR), Resting Systolic Blood Pressure (Rest SBP), Resting Diastolic Blood Pressure (Rest DBP), Peak Systolic Blood Pressure (Peak SBP), Peak Diastolic Blood Pressure (Peak DBP), Peak Power (Power peak), Oxygen Uptake Anaerobic threshold per kilogram (VO_2/kg AT), and Ventilation to Carbon Dioxide Production Slope (V_E/VCO_2 Slope).

The CPET was terminated if any of the following occurred: (1) the patient was unable to maintain the prescribed rotational speed; (2) the patient experienced severe dyspnea, chest tightness, dizziness, etc.; (3) the patient reported significant chest pain; (4) the patient's blood pressure exceeded 180/120 mmHg; (5) the patient became pale and had a blood pressure of less than 90/60 mmHg; (6) the patient's ECG during exercise showed significant ST-T changes or frequent heart rate abnormalities.

Statistical analysis

Continuous variables were described as mean ± standard deviation, and comparisons between groups were made using the t-test or Mann-Whitney U-test. Categorical variables were described as frequencies and percentages, and between-group differences were analyzed using the chi-square test and Fisher's exact test. Correlations were analyzed using Pearson correlation analysis, and logistic regression was used to identify factors influencing the occurrence of AF in HCM patients. A predictive model was developed using a nomogram, and the area under the Receiver operator characteristic (ROC) curve (AUC) was plotted to evaluate the model's discriminatory ability. The calibration curve was plotted to assess model calibration, and the Hosmer-Lemeshow test was performed. Decision Curve Analysis (DCA) was used to evaluate the clinical benefit of the model. All statistical analyses were conducted using SPSS 23.0. Two-sided tests were performed, and a *P*-value <0.05 was considered statistically significant.

Results

AF occurrence and peak oxygen consumption in CM patients

Of the 160 patients included, 62 had AF, resulting in an AF incidence of 38.75%. The average Peak VO_2/kg in the AF group was (16.63±4.09) $ml{\cdot}min^{-1}{\cdot}kg^{-1}$, while in the non-AF group it was (18.50±5.09) $ml{\cdot}min^{-1}{\cdot}kg^{-1}$ (**Figure 1**).

Comparison of general data between the AF and non-AF groups

The proportion of NYHA Grade III patients and smokers was significantly higher in AF group compared to the non-AF group (P<0.05). Peak VO_2/kg, METs and VO_2/kg AT were significantly lower in the AF group than those in the non-AF group, while VE/VCO_2 Slope was significantly higher (all P<0.05, **Table 1**). There was no significant difference in the distribution of other parameters (P>0.05).

Comparison of peak oxygen consumption among different subgroups

There were no statistically significant differences in Peak VO_2/kg between male and female subgroups, the subgroups aged <50 years and ≥50 years, or between BMI ≥24 kg/m^2 and <24 kg/m^2 subgroups (all P>0.05). However, within the NYHA classification subgroups, Peak VO_2/kg in class III subgroup (17.94±3.96 ml·min^{-1}·kg^{-1}) and class IV subgroup (16.23±4.30 ml·min^{-1}·kg^{-1}) were significantly lower than class II subgroup (18.98±5.67 ml·min^{-1}·kg^{-1}) (P= 0.012) (**Table 2**). Pearson's correlation analysis show-ed a significant negative correlation between Peak VO_2/kg and NYHA classification (r=-0.231, P=0.003).

Influencing factors of AF occurrence in HCM patients

Using the occurrence of AF in HCM patients as the dependent variable Y (yes =1, no =0) and the statistically significant variables from **Table 1** as independent variables (X), a binary Logistic regression model was established. The assignment of dependent variables is shown in **Table 3**. The results indicated that NYHA grade, smoking history and VE/VCO_2 Slope were risk factors for AF in HCM patients, while Peak VO_2/kg, METs, and VO_2/kg AT were protective factors for AF in HCM patients (P<0.05) (**Table 4**).

Construction of nomogram model

NYHA classification, smoking history, Peak VO_2/kg, METs, VO_2/kg AT, VE/VCO_2 Slope obtained by Logistic regression analysis were used to construct a Nomogram prediction model. The total score corresponds to the risk of AF in HCM patients (**Figure 2**).

Evaluation and verification of nomogram model

The constructed nomogram was evaluated and verified using the ROC curve. The results showed an AUC of 0.867 (0.808-0.925), with sensitivity and for predicting AF occurrence of 80.60% and 83.90%, respectively, demonstrating good discrimination (**Figure 3A**). A calibration curve was generated, showing only a slight deviation between the predicted results of the nomogram and the ideal model, indicating that the predictions are in good agreement with the actual outcomes (**Figure 3B**). The Hosmer-Lemeshow test was performed on the constructed nomogram model (χ^2=14.464, P=0.070, P>0.05), indicating that the model had good calibration.

Clinical benefits of the nomogram

A nomogram decision curve analysis was conducted, where the X-axis represents threshold probability, and the Y-axis represents clinical net benefit. The results showed that the nomogram model curve was above the two extreme lines in the threshold probability interval of >0.1. This indicates that applying the nomogram model when the threshold probability is >10% can provide clinical benefit and has high practical application value (**Figure 4**).

Discussion

Patients with hypertrophic cardiomyopathy (HCM) often present symptoms such as chest tightness, shortness of breath, and activity limitation due to impaired cardiac function. These symptoms can further increase the risk of sudden death when combined with atrial fibrillation (AF). However, a significant proportion of patients with paroxysmal AF remain undiagnosed due to limited awareness of AF [17]. It has been proposed that effective management of AF in HCM patients may reduce mortality and hospitalizations due to heart failure, and the risk of thromboembolic events and severe bleeding, among other benefits [18]. The present study aimed to compare the CPET results between HCM patients and those with both HCM and AF, with a focus on the key index Peak VO_2/kg, in order to provide clinical guidance for the early identification and management of AF in HCM patients.

Table 1. Comparison of baseline information between the two groups

Variable	AF group (n=62)	Non-AF group (n=98)	χ^2/t	P
Age	42.85±11.28	43.16±12.07	0.162	0.871
Gender [n (%)]				
Male	38 (61.29)	56 (57.14)	0.270	0.604
Female	24 (38.71)	42 (42.86)		
BMI ($\bar{x}$±s, kg/m^2)	23.25±2.74	23.38±2.51	0.308	0.759
NYHA classification [n (%)]				
II	14 (22.58)	46 (46.94)	11.655	0.003
III	21 (33.87)	30 (30.61)		
IV	27 (43.55)	22 (22.45)		
HCM Family History [n (%)]				
Yes	11 (17.74)	13 (13.27)	0.597	0.440
No	51 (82.26)	85 (86.73)		
Hypertension [n (%)]				
Yes	38 (61.29)	56 (57.14)	0.270	0.604
No	24 (38.71)	42 (42.86)		
Diabetes [n (%)]				
Yes	21 (33.87)	37 (37.76)	0.248	0.619
No	41 (66.13)	61 (62.24)		
Coronary heart disease [n (%)]				
Yes	18 (29.03)	23 (23.47)	0.617	0.432
No	44 (70.97)	75 (76.53)		
Hyperlipidemia [n (%)]				
Yes	15 (24.19)	19 (19.39)	0.524	0.469
No	47 (75.81)	79 (80.61)		
Smoking [n (%)]				
Yes	40 (64.52)	41 (41.84)	7.814	0.005
No	22 (35.48)	57 (58.16)		
Alcohol consumption [n (%)]				
Yes	27 (43.55)	34 (34.69)	1.262	0.261
No	35 (56.45)	64 (65.31)		
Peak VO_2/kg ($\bar{x}$±s, ml·min^{-1}·kg^{-1})	16.63±4.09	18.50±5.09	2.437	0.016
METs ($\bar{x}$±s)	4.33±0.69	5.14±0.94	5.857	<0.001
HRR ($\bar{x}$±s, n/min)	22.00 (9.00-36.00)	25.00 (10.00-40.00)	0.523	0.602
Peak HR ($\bar{x}$±s, time/min)	132.56±25.28	135.78±24.38	0.802	0.424
Rest SBP ($\bar{x}$±s, mmHg)	112.84±12.38	113.58±11.51	0.385	0.701
Rest DBP ($\bar{x}$±s, mmHg)	75.23±8.54	76.54±8.62	0.940	0.349
Peak SBP ($\bar{x}$±s, mmHg)	164.25±21.54	158.76±23.57	1.783	0.140
Peak DBP ($\bar{x}$±s, mmHg)	85.36±12.45	83.23±11.56	1.102	0.272
Power peak ($\bar{x}$±s, W)	112.54±21.84	110.62±23.54	0.517	0.606
VO_2/kg AT ($\bar{x}$±s, ml·min^{-1}·kg^{-1})	13.41±2.84	14.67±3.25	2.506	0.013
V_E/VCO_2 Slope ($\bar{x}$±s)	30.54±4.28	27.79±3.63	4.352	<0.001

Abbreviations: AF, atrial fibrillation; BMI, body mass index; NYHA, New York Heart Association; HCM, hypertrophic cardiomyopathy; Peak VO_2/kg, Peak Oxygen Uptake per kilogram; METs, Metabolic Equivalent of Task; HRR, Heart Rate Reserve; Peak HR, Peak Heart Rate; Rest SBP, Resting Systolic Blood Pressure; Rest DBP, Resting Diastolic Blood Pressure; Peak SBP, Peak Systolic Blood Pressure; Peak DBP, Peak Diastolic Blood Pressure; VO_2/kg AT, Oxygen Uptake at Anaerobic threshold per kilogram; V_E/VCO_2 Slope, Ventilation to Carbon Dioxide Production Slope.

The results of this study demonstrated that the prevalence of AF in 160 patients with HCM was 38.75% (62/160), which is comparable to the 38.8% AF incidence reported by Fauchier L et

Table 2. Comparison of Peak VO_2/kg among different subgroups

Grouping	Peak VO_2/kg (ml·min^{-1}·kg^{-1})	t	P
Gender			
Male (n=94)	17.29±5.19	1.606	0.110
Female (n=66)	18.54±4.31		
Age			
<50 (n=119)	17.62±4.96	0.827	0.410
≥50 (n=41)	18.35±4.62		
BMI			
≥24 kg/m^2 (n=57)	17.63±5.06	0.549	0.584
<24 kg/m^2 (n=103)	18.12±4.54		
NYHA classification			
II (n=60)	18.98±5.67	4.517	0.012
III (n=51)	17.94±3.96		
IV (n=49)	16.23±4.30		

Abbreviations: Peak VO_2/kg, Peak Oxygen Uptake per kilogram; NYHA, New York Heart Association.

Table 3. Variable assignment

Variable	Assignment
NYHA classification	II=0, III/IV=1
Smoking	Yes =1, No =0
Peak VO_2/kg	Actual value
METs	Actual value
VO_2/kg AT	Actual value
V_E/VCO_2 Slope	Actual value

Abbreviations: NYHA, New York Heart Association; Peak VO_2/kg, Peak Oxygen Uptake per kilogram; METs, Metabolic Equivalent of Task; VO_2/kg AT, Oxygen Uptake at Anaerobic threshold per kilogram; V_E/VCO_2 Slope, Ventilation to Carbon Dioxide Production Slope.

al. [19]. This further supports the assertion that HCM patients are at an elevated risk of developing AF. The underlying mechanism for this elevated risk is attributed to the aberrant structural organization of myocardial tissue resulting from disrupted cardiomyocyte alignment in HCM. This leads to heterogeneous electrical signal conduction between cardiomyocytes, thereby increasing the electrophysiological instability of the atrial myocardium and facilitating the formation of re-entry circuits, which provide a pathological foundation for the onset of AF [20]. In this study, a comparison of the general data between the AF and non-AF groups revealed that the AF group exhibited a more severe NYHA classification, a higher proportion of smokers, lower Peak VO_2/kg, METs, and VO_2/kg AT, and higher VE/VCO_2 Slope. These findings suggest that patients in the AF group had greater cardiac function impairment and poorer lifestyle habits. Additionally, the occurrence of AF further reduced the patients' exercise tolerance. A comparison of Peak VO_2/kg in different subgroups showed no statistically significant differences between genders, age groups, or BMI categories. However, significant differences in Peak VO_2/kg were observed between different NYHA classifications. Pearson correlation analysis revealed a negative correlation between Peak VO_2/kg and NYHA classification, indicating that cardiac insufficiency is closely related to the decline in exercise tolerance.

This study identified, through Logistic regression, that the risk factors for AF in HCM patients included NYHA grade, smoking history, and VE/VCO_2 Slope, while the protective factors included Peak VO_2/kg, METs and VO_2/kg AT. This indicates that a higher NYHA grade and a smoking history, along with lower VE/VCO_2 Slope, Peak VO_2/kg, METs and VO_2/kg AT, are associated with an increased risk of AF in HCM patients. Conversely, a higher VE/VCO_2 Slope is linked to a higher risk of AF. The underlying associations may include the following: As the NYHA grade increases, the patient's cardiac function worsens, with more severe myocardial hypertrophy and fibrosis, leading to structural remodeling of the atrium. This remodeling alters the number and distribution of gap junctions between myocytes, affecting electrical signal transmission and increasing conduction heterogeneity in the atrium, ultimately increasing the probability of AF [21]. Sandeep B et al. [22] emphasized that structural remodeling increased. Tobacco smoke contains various harmful chemicals, including tar, nicotine and carbon monoxide, which generate a large number of free radicals during metabolic processes. These free radicals seize electrons from other molecules in the process of seeking stability, resulting in lipid peroxidation of the cardiac cell membrane and disrupting its integrity and function. This leads to

Table 4. Logistic regression analysis of independent factors influencing AF occurrence in HCM patients

Variable	β	SE	Wald	P	OR (95% CI)
NYHA classification	0.963	0.451	2.136	0.033	2.621 (1.083-6.344)
Smoking	0.984	0.422	2.329	0.020	2.674 (1.169-6.118)
Peak VO_2/kg	-0.097	0.045	-2.175	0.030	0.907 (0.831-0.990)
METs	-1.281	0.274	-4.680	<0.001	0.278 (0.163-0.475)
VO_2/kg AT	-0.164	0.070	-2.336	0.019	0.849 (0.740-0.974)
VE/VCO_2 Slope	0.163	0.056	2.907	0.004	1.177 (1.054-1.313)
Constant	3.663	2.382	1.538	0.124	38.971

Abbreviations: SE, Standard Error; OR, Odds Ratio; NYHA, New York Heart Association; Peak VO_2/kg, Peak Oxygen Uptake per kilogram; METs, Metabolic Equivalent of Task; VO_2/kg AT, Oxygen Uptake at Anaerobic threshold per kilogram; V_E/VCO_2 Slope, Ventilation to Carbon Dioxide Production Slope.

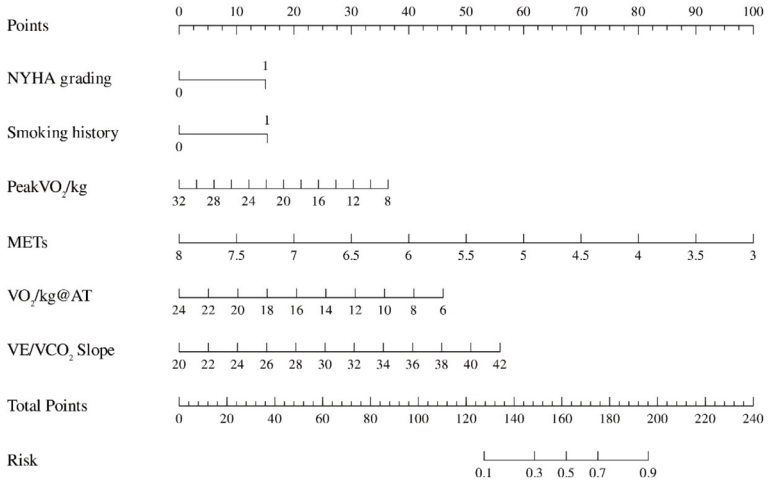

Figure 2. Nomogram prediction model. Abbreviations: NYHA, New York Heart Association; Peak VO_2/kg, Peak Oxygen Uptake per kilogram; METs, Metabolic Equivalent of Task; VO_2/kg AT, Oxygen Uptake at Anaerobic threshold per kilogram; V_E/VCO_2 Slope, Ventilation to Carbon Dioxide Production Slope.

changes in atrial electrophysiological characteristics and atrial remodeling, providing a pathological basis for AF development [23]. Therefore, HCM patients with a history of smoking are at a higher risk of developing AF.

CPET aligns with the concept of the new theoretical system of holistic integrated physiology and medicine. It provides an objective, quantitative measurement of the function of multiple systems in the human body and is suitable for both healthy individuals and patients with various diseases [24]. Peak VO_2/kg is a key indicator of cardiopulmonary function, reflecting cardiac performance during exercise. METs measure exercise intensity, representing a multiple oxygen consumption compared to resting levels. VO_2/kg AT represents the critical point at which the body transitions from aerobic to anaerobic metabolism, thus evaluating an individual's aerobic capacity. VE/VCO_2 Slope indicates the change in the ratio of ventilation to carbon dioxide production over time during exercise, serving as an indicator of ventilation efficiency. A low VE/VCO_2 Slope indicates more efficient carbon dioxide removal during exercise [25]. In patients with AF, atrial systolic function is impaired, leading to insufficient ventricular filling and decreased heart pumping efficiency. This results in decreased oxygen transport capacity to systemic tissues and a corresponding reduction in Peak VO_2/kg. Consequently, muscles are unable to sustain effective aerobic metabolism, which limits exercise tolerance and promotes the transition from aerobic metabolism to anaerobic metabolism at a lower exercise intensity, resulting in lower METs and VO_2/kg AT [26]. Additionally, the irregular pumping function of the heart in AF patients induces hemodynamic changes in the lungs, leading to an imbalance between ventilation and perfusion. This imbalance reduces the efficiency of oxygen and carbon dioxide exchange. To maintain proper gas exchange, the body increases ventilation (i.e., respiratory rate and/or depth), which, in turn, decreases ventilation efficiency, reflected by an increase in VE/VCO_2 Slope [27]. In a study of Baldi C et al. [28], low Peak VO_2 was associated with an increased risk of cardiac death during follow-up in patients with functional mitral regurgitation after MitraClip implantation. Li N et al. [29] found that Peak VO_2 can assess the severity of cardiac function damage and evaluate the cardiac status of patients, proving useful in the clinical diagnosis of coronary heart disease. These studies confirm the application value of CPET indices in clinical diagnosis and prognosis evaluation of cardiovascular diseases.

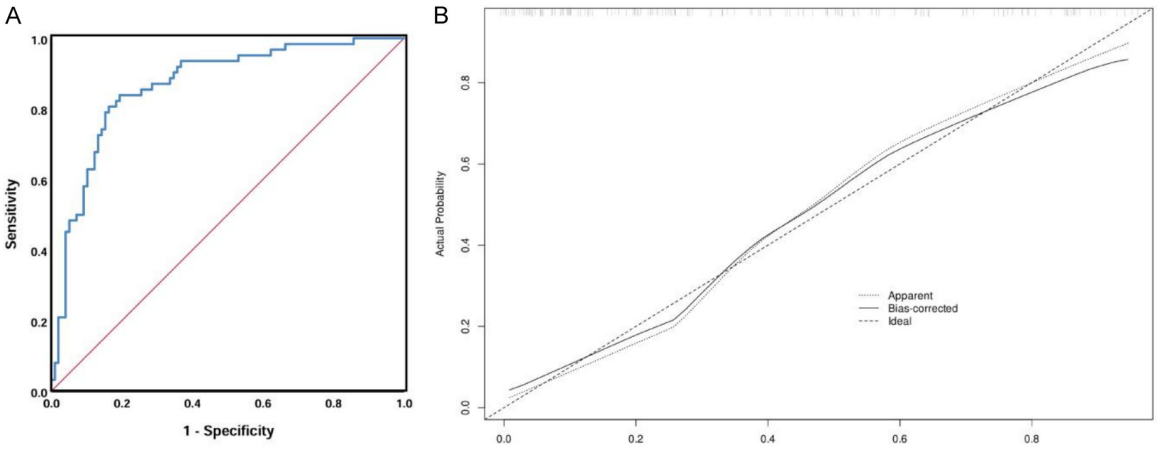

Figure 3. ROC curve and calibration curve analysis of predictive model. A. ROC curve; B. Calibration curve. Abbreviation: ROC, Receiver Operating Characteristic.

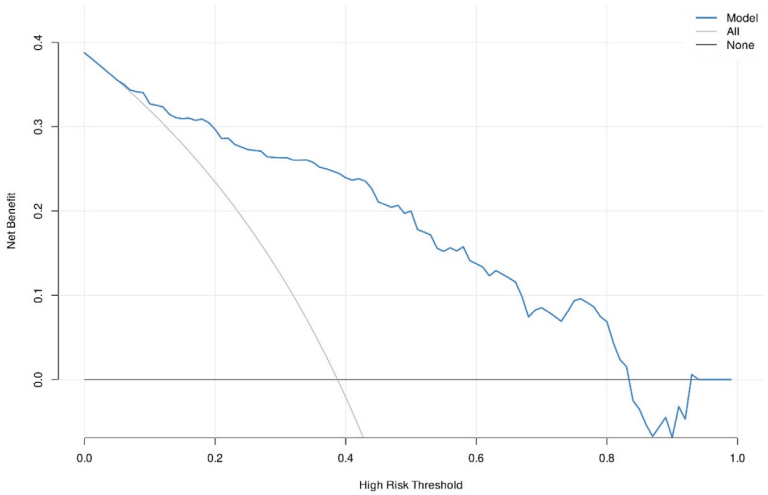

Figure 4. Decision curve analysis of prediction model.

In this study, influencing factors for AF occurrence in HCM patients were identified through Logistic regression analysis, and a nomogram prediction model was further constructed. The results showed that the AUC for predicting AF occurrence using the nomogram was 0.867 (0.808-0.925), and the calibration curve demonstrated good agreement between the predicted and actual incidence of AF in HCM patients. Additionally, the decision analysis curve indicated that the nomogram model had a high clinical benefit. This model can be utilized in future clinical practice to assess the risk of AF in HCM patients.

This study has some limitations. It was based on retrospective analysis with limited clinical information, which may introduce bias in the selection of indicators. Furthermore, as all patients were from the same center, there may be selection bias. Future studies with large sample sizes, multi-center designs, prospective approaches, and long-term follow-up are needed to explore the relationship between AF and other related factors, providing stronger scientific evidence for improving patient care practices.

Conclusion

By comparing the general data characteristics and CPET results of HCM patients and HCM patients with AF, this study found that the influencing factors for the occurrence of AF were NYHA grade, smoking history, Peak VO_2/kg, METs, VO_2/kg AT and VE/VCO_2 Slope. The constructed nomogram has a good degree of differentiation and calibration, which is helpful to identify the risk of AF in early clinical stages.

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Disclosure of conflict of interest

None.

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