



Research Article

A predictive model for heart failure in patients with hypertension and persistent atrial fibrillation: A retrospective single-center study

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ABSTRACT

Objective: This study aimed to develop and validate a predictive nomogram for heart failure in patients with hypertension and persistent atrial fibrillation (HT-PeAF).

Methods: We retrospectively analyzed clinical data from patients with HT-PeAF hospitalized at Chinese People's Liberation Army General Hospital between 2010 and 2019. Predictive variables were selected based on multivariable logistic regression analysis and clinical relevance, which were then incorporated into a clinical nomogram. Model performance was evaluated using the area under the receiver operating characteristic curve (AUC) for discrimination, the Hosmer–Lemeshow test for calibration, and decision curve analysis for clinical utility.

Results: Of the 395 enrolled patients, 151 experienced heart failure. Nine predictive variables were identified: prior catheter ablation, low-density lipoprotein cholesterol, hemoglobin, uric acid, creatinine, total bilirubin, fasting blood glucose, red blood cell distribution width, and coronary artery disease. The nomogram demonstrated good discriminative ability (AUC = 0.77, 95% CI: 0.72–0.81), good calibration ($P = 0.44$), and favorable clinical utility.

Conclusion: This accessible tool facilitates individualized risk stratification for heart failure in HT-PeAF patients, though prospective validation in multicenter Asian cohorts is warranted.

1. Introduction

Hypertension (HT) is the most prevalent risk factor for atrial fibrillation (AF).^{1–3} The coexistence of HT and persistent atrial fibrillation (HT-PeAF) introduces complex therapeutic challenges and synergistically heightens the risk of adverse cardiovascular events, profoundly impacting patients' quality of life and long-term prognosis.^{4–7} Clinically, HT promotes the initiation and perpetuation of AF through fibro-inflammatory remodeling of the left atrium⁸ while PeAF itself further influences atrial remodeling, exacerbates ventricular dysregulation, and aggravates renal dysfunction.^{9–11} This reciprocal reinforcement establishes a deleterious cycle that ultimately culminates in overt heart failure.

Despite this well-established epidemiological and pathophysiological link, there remains a critical lack of convenient and accurate tools for assessing the probability of heart failure development specifically in patients with HT-PeAF. Existing AF risk scores, such as CHA₂DS₂-

VASc (designed primarily focused on thromboembolic events or initial AF detection)¹² and HATCH (predicting progression from paroxysmal to PeAF),¹³ were not designed for this high-risk subpopulation and fail to capture the combined electroanatomical and hemodynamic decline. Consequently, clinicians continue to rely on fragmented clinical judgment, potentially underestimating heart failure risk in seemingly stable HT-PeAF patients, particularly in primary care settings where advanced echocardiographic metrics are unavailable.

Identifying clinical risk factors within this specific population is crucial for implementing early prevention and personalized intervention strategies. Nomograms have emerged as highly pragmatic clinical instruments that translate complex statistical regressions into intuitive, individualized visual frameworks for rapid risk stratification. However, current understanding of these prospective relationships in the HT-PeAF cohort remains incomplete. Therefore, this study aims to comprehensively analyze clinical data of patients with HT-PeAF to identify critical factors associated with myocardial dysfunction and to develop a practi-

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cal predictive nomogram for heart failure in this specific patient cohort. The findings are anticipated to provide valuable references for improving the clinical management and long-term prognosis of patients with HT-PeAF.

2. Patients and methods

2.1. Study population

This study reviewed the medical records of patients hospitalized with HT-PeAF in the Department of Cardiology at the Chinese People's Liberation Army General Hospital between January 2010 and September 2019. Only patients with complete medical records were included. Exclusion criteria were: (1) congenital heart disease or valvular heart disease; (2) history of malignant tumors; and (3) recent serious infection.

The sample size was justified based on the events per variable (EPV) criterion, a widely accepted standard for developing robust clinical prediction models using multivariable logistic regression. To ensure model stability and minimize overfitting, an EPV of at least 10 is generally recommended.

The research protocol was approved by the Institutional Ethics Committee of the Sixth Medical Center of Chinese People's Liberation Army General Hospital (No. HZKY-PJ-2021–5), which granted a waiver of informed consent due to the retrospective nature of the study.

2.2. Data collection and definitions

Clinical characteristics were extracted from inpatient electronic medical records, including demographic characteristics, lifestyle, anthropometric measurements, laboratory tests, echocardiography, medical history, and medication use.

PeAF was defined as an episode lasting 7 days or more,⁶ documented by an electrocardiogram or electronic medical record. HT was defined as systolic blood pressure ≥ 140 mmHg (1 mmHg = 0.133 kPa) and/or diastolic blood pressure ≥ 90 mmHg, which is equivalent to a 24-hour ambulatory blood pressure monitoring average $\geq 130/80$ mmHg, or a home blood pressure monitoring average $\geq 135/85$ mmHg.¹⁴ Heart failure was defined following the Chinese Guidelines for the Diagnosis and Treatment of Heart Failure.¹⁵ Patients were identified as having heart failure if they presented with: (1) typical symptoms of heart failure (e.g., dyspnea, fatigue) or signs (e.g., edema, pulmonary rales); and (2) objective evidence of cardiac structural or functional abnormalities at rest, primarily determined by an ejection fraction (EF) $< 50\%$ or echocardiographic evidence of diastolic dysfunction. A history of stroke, prior catheter ablation, hyperlipidemia, diabetes mellitus, coronary artery disease (CAD), renal insufficiency, smoking, alcohol consumption, and medication use were self-reported by patients.

2.3. Statistical analyses

All analyses were performed using SPSS (version 22.0, IBM Corp., Armonk, NY, USA) and R (version 3.4.3, R Foundation for Statistical Computing, Vienna, Austria).

Categorical variables are expressed as n (%). Continuous variables with a normal distribution are expressed as mean $\pm$ standard deviation, while those without a normal distribution are expressed as median and interquartile range. The Kolmogorov–Smirnov test was used to assess normality. Differences between groups were compared using Chi-square test or Fisher's exact test for categorical variables, Student's t -test for normally distributed continuous variables, and Mann–Whitney U test for non-normally distributed continuous variables.

Multivariable logistic regression analysis was performed to identify significant predictors of heart failure in patients with HT-PeAF. These significant predictors, along with selected clinically relevant variables, were combined to construct a nomogram, which was calibrated using a

calibration plot. To evaluate the model's discrimination, receiver operating characteristic curves and the Kolmogorov–Smirnov (KS) curve were used, and to assess its calibration, the Hosmer–Lemeshow goodness-of-fit test was used. Additionally, decision curve analysis was applied to evaluate its clinical usefulness.

To adjust for overfitting and evaluate internal validity, internal validation was performed using the bootstrap resampling method with 1000 replications. This enabled the calculation of a bias-corrected C-index and the generation of a bias-corrected calibration curve.

3. Results

3.1. Clinical characteristics of HT-PeAF patients

A total of 395 patients were enrolled. In this study, the primary outcome (heart failure) occurred in 151 patients, and 9 predictive variables were included in the final multivariable model, yielding an EPV of 16.8 (151/9). This value well exceeds the recommended minimum threshold of 10, demonstrating that the sample size of 395 patients was statistically adequate for model development.

Patients with heart failure were older than those without ($P = 0.017$). They had higher rates of prior catheter ablation, renal insufficiency, and CAD (all $P < 0.001$). Regarding laboratory findings, patients with heart failure had lower hemoglobin levels ($P = 0.001$), and higher levels of red blood cell distribution width (RDW), fasting blood glucose, creatinine, and uric acid (all $P < 0.01$). In terms of lipid profile, patients with heart failure had lower total cholesterol and low-density lipoprotein cholesterol (LDL-C) concentrations ($P < 0.01$). Echocardiography showed that patients with heart failure had a lower EF ($P < 0.001$) and a larger left atrial diameter ($P = 0.016$). Regarding medication use, the proportion of patients receiving anticoagulation therapy was significantly higher in the heart failure group than in the non-heart failure group (59.60% vs. 13.11%, $P < 0.001$) (Table 1).

3.2. Associations between risk factors and heart failure in patients with HT-PeAF

Logistic regression analysis was performed to identify risk factors associated with heart failure in patients with HT-PeAF (Table 2). In unadjusted analyses, prior catheter ablation, LDL-C, hemoglobin, uric acid, creatinine, total bilirubin, fasting blood glucose, RDW, and CAD were associated with the occurrence of heart failure. After multivariable adjustment, CAD (adjusted OR = 2.31, 95% CI: 1.38–3.96), RDW (adjusted OR = 1.41, 95% CI: 1.15–1.76), total bilirubin (adjusted OR = 1.04, 95% CI: 1.01–1.07), and creatinine (adjusted OR = 1.01, 95% CI: 1.00–1.02) remained independently associated with an increased risk of heart failure. Conversely, higher LDL-C levels were independently associated with a decreased risk of heart failure (adjusted OR = 0.72, 95% CI: 0.52–0.98).

3.3. Establishing a prediction model for heart failure occurrence

To develop a clinically useful and robust prediction model, variables were selected based on both statistical significance and clinical relevance. The final model primarily included variables that achieved significance in the multivariable analysis ($P < 0.05$). However, we deliberately retained specific indicators from the univariate screening (prior catheter ablation, hemoglobin, uric acid, and fasting blood glucose; $P < 0.05$) whose statistical associations weakened after multivariable adjustment ($P > 0.05$). This approach was applied to preserve potentially critical clinical information and avoid omitting clinical relevant predictors through strict reliance on statistical thresholds alone.

Consequently, these clinically meaningful variables were integrated into the final nomogram prediction model. The total score was calculated based on prior catheter ablation, LDL-C, hemoglobin, uric acid, creatinine, total bilirubin, fasting blood glucose, RDW, and CAD. Each

Table 1
Baseline characteristics of HT-PeAF patients with or without heart failure.

Characteristics	Heart failure group (n = 151)	Non-heart failure group (n = 244)	P value
Age (yrs, mean ± SD)	71.11 ± 9.59	68.62 ± 10.30	0.017
Men [n (%)]	97 (64.24)	149 (61.07)	0.527
BMI (kg/m ² , mean ± SD)	25.93 ± 4.04	27.78 ± 3.16	0.298
Smoking [n (%)]	62 (41.06)	90 (36.89)	0.407
Alcohol consumption [n (%)]	59 (39.07)	91 (37.30)	0.724
Medical history [n (%)]			
History of stroke	58 (38.41)	71 (29.10)	0.055
Prior catheter ablation	29 (19.20)	15 (6.15)	<0.001
Hyperlipidemia	47 (31.13)	74 (30.33)	0.867
Diabetes mellitus	46 (30.46)	66 (27.05)	0.464
Coronary artery disease	118 (78.15)	147 (60.25)	<0.001
Renal insufficiency	29 (19.21)	15 (6.15)	<0.001
Laboratory findings			
Hemoglobin (g/L, mean ± SD)	134.89 ± 20.40	142.14 ± 19.96	0.001
WBC (× 10 ⁹ /L, mean ± SD)	6.37 ± 2.07	6.51 ± 2.58	0.584
RDW [% , median (IQR)]	13.40 (12.90, 14.50)	12.90 (12.43, 13.50)	<0.001
FBG [mmol/L, median (IQR)]	5.56 (4.99, 7.34)	5.37 (4.65, 6.57)	0.009
Creatinine [mmol/L, median (IQR)]	91.60 (73.00, 118.10)	81.45 (69.63, 95.75)	<0.001
Uric acid [μmol/L, median (IQR)]	410.50 (311.80, 505.50)	364.50 (301.23, 437.60)	0.003
CRP [mg/L, median (IQR)]	0.32 (0.10, 0.67)	0.32 (0.10, 0.43)	0.105
Total bilirubin [μmol/L, median (IQR)]	15.40 (10.20, 22.80)	14.65 (11.73, 18.40)	0.425
Total cholesterol [mmol/L, median (IQR)]	3.73 (3.04, 4.15)	3.83 (3.33, 4.47)	0.001
Triglyceride [mmol/L, median (IQR)]	1.25 (0.79, 1.38)	1.29 (0.94, 1.55)	0.102
HDL-C [mmol/L, median (IQR)]	1.07 (0.87, 1.25)	1.09 (0.92, 1.27)	0.218
LDL-C (mmol/L, mean ± SD)	2.10 ± 0.76	2.39 ± 0.82	<0.001
Echocardiography data			
Ejection fraction [% , median (IQR)]	50.00 (40.00, 58.00)	59.00 (56.00, 63.00)	<0.001
Left atrial diameter (mm, mean ± SD)	45.04 ± 6.66	43.50 ± 5.83	0.016
Left ventricular wall thickening* [n (%)]	38 (25.17)	49 (20.08)	0.236
Medication use [n (%)]			
Antiplatelet drugs	16 (10.60)	16 (6.56)	0.153
ACEI	14 (9.27)	15 (6.15)	0.247
ARB	26 (17.22)	56 (22.95)	0.172
CCB	48 (31.79)	78 (31.97)	0.970
Diuretic	5 (3.31)	12 (4.92)	0.444
Amiodarone	2 (1.32)	2 (0.82)	0.639
Anticoagulation	90 (59.60)	32 (13.11)	<0.001

HT-PeAF: comorbidity of hypertension and persistent atrial fibrillation; BMI: body mass index; SD: standard deviation; WBC: white blood cell; RDW: red blood cell distribution width; FBG: fasting blood glucose; IQR: interquartile range; CRP: C-reactive protein; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; ACEI: angiotensin-converting enzyme inhibitor; ARB: angiotensin receptor antagonist; CCB: calcium channel blocker. *Defined as left ventricular wall thickness greater than 12 mm.

Table 2
Associated risk factors for heart failure in patients with HT-PeAF.

Factors	Crude OR (95% CI)	P value	Adjusted OR (95% CI)	P value
Prior catheter ablation	0.10 (0.01–0.78)	0.028	0.14 (0.01–0.76)	0.065
LDL-C	0.63 (0.47–0.83)	0.001	0.72 (0.52–0.98)	0.038
Hemoglobin	0.98 (0.97–0.99)	0.001	0.99 (0.98–1.00)	0.113
Uric acid	1.00 (1.00–1.01)	0.001	1.00 (0.99–1.00)	0.085
Creatinine	1.01 (1.01–1.02)	<0.001	1.01 (1.00–1.02)	0.015
Total bilirubin	1.03 (1.01–1.06)	0.014	1.04 (1.01–1.07)	0.012
FBG	1.11 (1.01–1.22)	0.040	1.09 (0.98–1.21)	0.099
RDW	1.60 (1.32–1.95)	<0.001	1.41 (1.15–1.76)	0.001
Coronary artery disease	2.36 (1.49–3.75)	<0.001	2.31 (1.38–3.96)	0.002

OR: odds ratio; CI: confidence interval.

predictor corresponds to a specific score on the points scale axis, and the overall risk of heart failure predicted by this nomogram ranged from 0.10 to 0.99 (Fig. 1).

3.4. Validation and clinical value of the nomogram model

Calibration curve showed that the bias-corrected curve lies close to the ideal curve, indicating an outstanding degree of concordance between predicted and observed heart failure probabilities (Fig. 2). The Hosmer-Lemeshow goodness-of-fit test further supported good model fit ($\chi^2 = 7.98$, $df = 8$, $P = 0.44$).

The ROC curve for predicting heart failure in patients with HT-PeAF is shown in Fig. 3A, with an area under the curve (AUC) of 0.77 (95% CI: 0.72–0.81). The optimal cutoff value determined by maximizing the Youden index was 0.433, yielding a sensitivity of 63.6%, a specificity of 82.0%, and a Youden index of 0.455. The positive and the negative likelihood ratios were 3.526 and 0.444, respectively. The KS value was 0.46, and the corresponding KS curve indicated good discrimination of the nomogram (Fig. 3B).

Decision curve analysis showed that the nomogram model accurately predicted the incidence of heart failure and provided a net clinical benefit (Fig. 4).

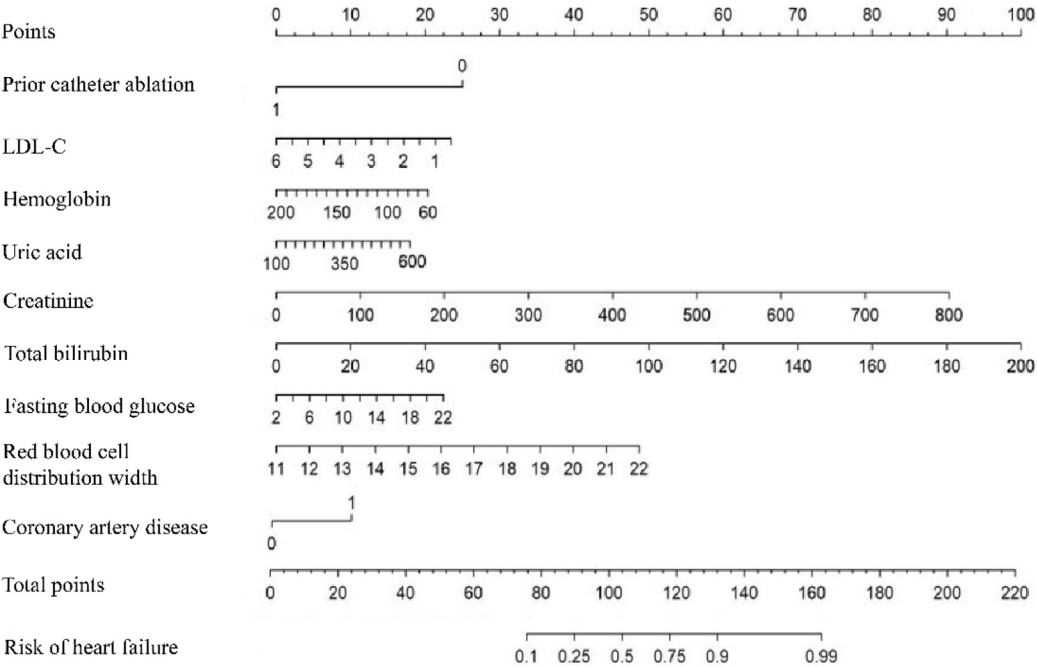


Fig. 1. Nomogram for predicting the risk of heart failure in patients with HT-PeAF. Each predictor is assigned a score on the point scale. The total score is calculated by summing the individual scores. By projecting the total score onto the lower total point scale, the corresponding probability of heart failure is estimated.

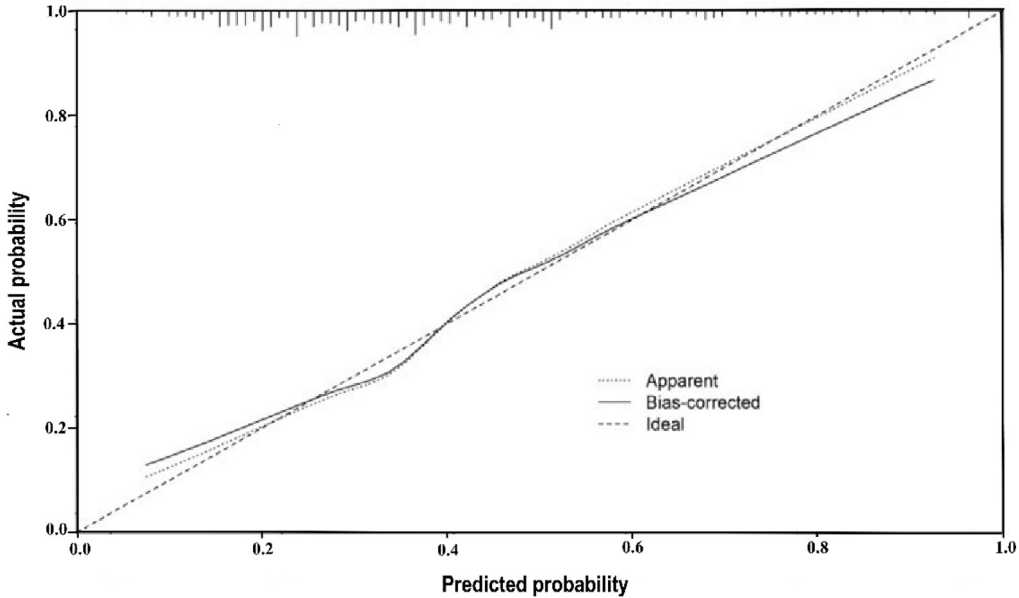


Fig. 2. Calibration curves of the nomogram for predicting the probability of heart failure in patients with HT-PeAF.

4. Discussion

4.1. Study design and model development

We prospectively developed a multivariable nomogram integrating nine clinical variables—CAD, fasting blood glucose, hemoglobin, LDL-C, RDW, total bilirubin, uric acid, creatinine, and prior catheter ablation—to establish a risk prediction framework for heart failure in patients with HT-PeAF.

4.2. Key predictors and pathophysiological insights

The strong association between CAD and heart failure risk (adjusted OR = 2.31) reflects the well-documented mechanisms through which ischemic cardiomyopathy promotes ventricular remodeling.¹⁶ HT and AF act synergistically to worsen coronary microvascular dysfunction, which promotes myocardial energy imbalance through calcium overload and mitochondrial damage, ultimately accelerating metabolic exhaustion in cardiac tissue.^{17–19} This finding aligns with clinical observa-

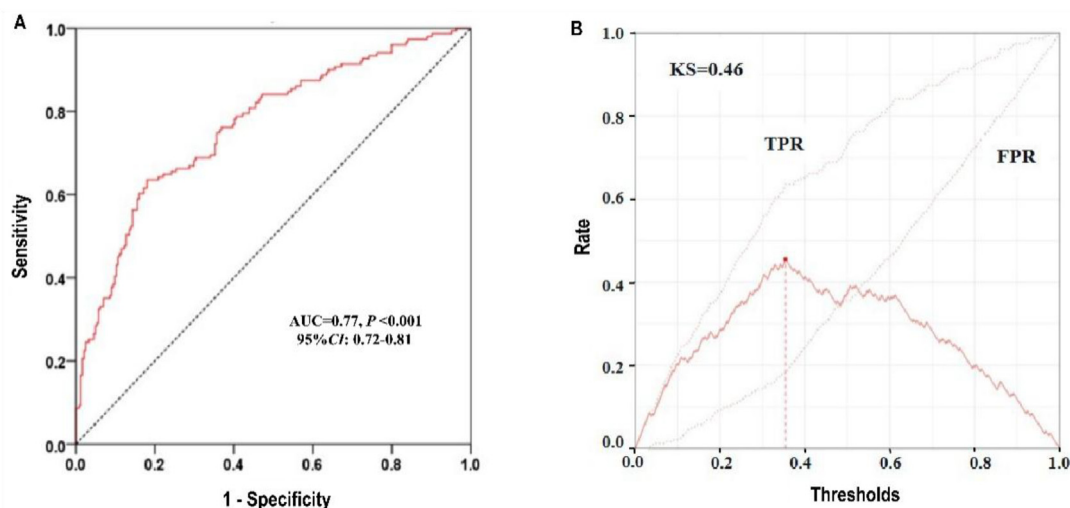


Fig. 3. Discriminative performance of the nomogram for predicting heart failure in patients with HT-PeAF. (A) receiver operating characteristic curve; (B) KS curve. KS: Kolmogorov-Smirnov; TPR: true positive rate; FPR: false positive rate.

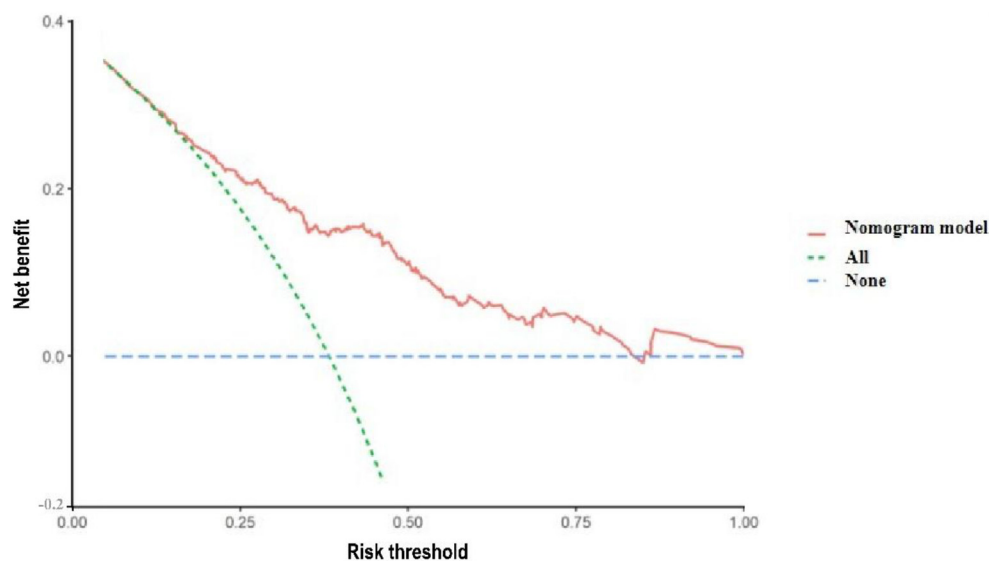


Fig. 4. Decision curve analysis for the nomogram model. The blue line assumes that no patients have heart failure, and the green line assumes that all patients have heart failure. The red line represents the nomogram.

tions of increased heart failure incidence in AF patients with comorbid CAD.²⁰

RDW demonstrated predictive utility beyond conventional anemia indices (adjusted OR = 1.41). As a biomarker of erythrocyte dysfunction linked to systemic inflammation^{21,22} and myocardial fibrosis,^{23,24} RDW was incorporated into AF-related heart failure prediction for the first time, independent of hemoglobin levels. Although hemoglobin and RDW are routinely measured hematological parameters, RDW reflects more than anemia alone. Elevated RDW has been associated with iron deficiency, systemic inflammation, oxidative stress, and impaired erythropoiesis, all of which are closely linked to adverse cardiac remodeling and heart failure progression. In the present study, RDW remained independently associated with heart failure even after adjustment for hemoglobin levels, suggesting that RDW captures pathophysiological information beyond simple anemia status.

Total bilirubin emerged as a novel oxidative stress marker (adjusted OR = 1.04). Although bilirubin's role in heart failure remains controversial—with some studies emphasizing its antioxidant and antifibrotic properties^{25,26} while others report no inverse correlation with cardiovascular disease risk²⁷ or even positive associations with coronary cal-

cification²⁸—our findings indicate a possible paradoxical pro-oxidant effect in HT-PeAF patients.

4.3. Clinical implications of the nomogram

From a clinical perspective, the inclusion of prior catheter ablation in the nomogram highlights the potential importance of rhythm control strategies in selected patients with HT-PeAF. Patients with a history of catheter ablation exhibited a lower risk of heart failure,²⁹ which may reflect reduced AF burden, improved ventricular rate control, or favorable baseline clinical characteristics rather than a direct causal effect of ablation itself.^{30,31}

Importantly, the present model is not intended to define indications for catheter ablation or to guide treatment selection. Instead, it provides a pragmatic tool for identifying HT-PeAF patients at higher risk of developing heart failure who may benefit from closer surveillance and comprehensive rhythm and rate control assessment. A higher predicted risk may prompt timely referral for specialist evaluation, optimization of medical therapy, or consideration of rhythm control strategies in accordance with contemporary clinical guidelines.

4.4. Model performance and advantages

The nomogram achieved superior discriminative ability (AUC = 0.77, 95% CI: 0.72–0.81, $P < 0.001$), significantly outperforming isolated metrics (e.g., EF: AUC = 0.60, 95% CI: 0.55–0.65, $P = 0.031$)³² and established risk scores (e.g., CHA₂DS₂-VASC for heart failure prediction: AUC = 0.653, HR = 1.21, 95% CI: 1.17–1.26; $P < 0.05$).³³ This marked improvement underscores the significant incremental predictive value of our model over traditional frameworks. While the CHA₂DS₂-VASC score remains a cornerstone clinical tool, it was fundamentally developed and optimized for thromboembolic stroke stratification, thereby failing to capture the complex, compounding hemodynamic and metabolic decline inherent to concurrent HT and PeAF. By contrast, our model addresses these critical limitations of prior tools (e.g., the modest heart failure predictive gain of the modified MAGGIC score in AF cohorts)³⁴ by expanding indicators beyond routine demographics to encompass parameters of progressive anatomical strain and metabolic stress. Consequently, this targeted integration enhances risk-assessment specificity for AF populations.

Key strengths include:

1. High accessibility: reliance on routine clinical parameters without specialized assays.
2. Individualized risk quantification: visual nomogram enabling patient-specific risk stratification (Fig. 1).
3. Multidimensional assessment: integration of inflammatory, oxidative, and structural injury pathways.

4.5. Limitations

Several limitations should be acknowledged. First, the single-center, retrospective design introduces an inherent risk of selection bias. Second, crucial biomarkers and highly specific echocardiographic parameters—such as N-terminal pro-B-type natriuretic peptide/B-type natriuretic peptide (NT-proBNP/BNP) levels, early transmitral flow velocity to early diastolic mitral annular velocity (E/e') ratio, and precise quantitative pulmonary artery pressures—had high rates of missing values because routine screening was not standardized in stable HT-PeAF patients during the study period (2010–2019). To prevent severe sample size reduction and selection bias, these parameters were excluded, which may restrict the model's comprehensive predictive power. Third, important clinical variables reflecting ventricular rate control and AF burden (e.g., continuous average heart rate derived from long-term ambulatory Holter monitoring or implantable devices) were not systematically captured in our routine inpatient records, precluding the evaluation of their potential impacts. Fourth, although baseline comparisons revealed an association between anticoagulation therapy and heart failure, this must be interpreted with caution as a marker of disease severity and confounding by indication rather than a causal risk factor; patients with comorbid PeAF and heart failure inherently present with higher thromboembolic risks and are thus more likely to receive anticoagulation. Fifth, specific pharmacological records were historically constrained; sodium-glucose cotransporter 2 inhibitors were not systematically captured as the study period largely preceded their widespread adoption in Asian clinical practice, and subclasses like beta-blockers could not be reliably binarized without introducing substantial misclassification bias. Finally, while this preliminary tool assists in initial risk stratification, external multi-center validation is warranted. Future prospective studies incorporating comprehensive pharmacological data, detailed echocardiographic parameters, continuous heart rate metrics, and contemporary heart failure therapies are strictly required to further refine and validate this risk prediction model.

5. Conclusion

This novel nomogram integrates nine routine variables to predict heart failure risk in HT-PeAF patients. While it addresses an unmet need

for early risk stratification in this specific population, its current clinical applicability is limited due to the single-center retrospective design. It serves best as a preliminary screening tool to prompt closer surveillance. Extensive prospective validation in multi-center cohorts, incorporating contemporary heart failure biomarkers and advanced echocardiographic parameters, is strictly required before broad clinical application.

Author's contributions

Li ZB contributed to conceptualization, writing-review & editing, project administration, and funding acquisition. Wang QY contributed to formal analysis, data curation, and writing original draft. Pan CP, Bai JQ, and Zhang ML contributed to data curation. Zou YT contributed to writing original draft. Liu YN contributed to formal analysis. Jin YF and Li RZ contributed to validation. All authors have read and approved the final manuscript.

Declaration of generative AI and AI-assisted technologies use

During the preparation of this work, the authors used large language models to assist with language editing, translation, and figure formatting. After using this tool/service, the authors closely reviewed and edited the content as needed and take full responsibility for the final content of the publication.

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Conflict of interest disclosure

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

The datasets generated and analyzed during the current study are not publicly available due to patient privacy and confidentiality.

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