Original Article

Establishment and validation of a machine learning model to stratify malnutrition risk in hospitalized older patients with chronic heart failure

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Background and Objectives: Malnutrition among older hospitalized adults with chronic heart failure (CHF) is associated with adverse clinical outcomes, yet reliable early risk stratification tools remain lacking. This study aimed to develop and validate a machine learning (ML) model for malnutrition risk stratification in this population. **Methods and Study Design:** Malnutrition among older hospitalized adults with chronic heart failure (CHF) is associated with adverse clinical outcomes, yet reliable early risk stratification tools remain lacking. This study aimed to develop and validate a machine learning (ML) model for malnutrition risk stratification in this population. **Results:** Malnutrition prevalence was 44.1% (348/790). In the internal testing, CatBoost (CAT) achieved superior performance with an AUC of 0.901 (95% confidence interval [CI]: 0.858-0.943), accuracy of 0.840, recall of 0.753, and the lowest Brier score of 0.113. This model demonstrated strong calibration, clinical utility, and the highest composite score (62/64). External validation confirmed CAT's generalizability (AUC: 0.916, 95% CI: 0.887-0.945). SHAP analysis identified body mass index (BMI), calf circumference, New York Heart Association (NYHA) classification, age, and diabetes as significant contributors to malnutrition risk. **Conclusions:** The CAT-based model effectively stratifies malnutrition risk in older hospitalized CHF patients, offering a tool for early intervention to improve outcomes. Further multicenter prospective studies are needed to validate its real-world applicability.

Key Words: geriatric, malnutrition, chronic heart failure, machine learning, risk stratification

INTRODUCTION

Chronic heart failure (CHF) is a complex clinical syndrome characterized by structural and functional cardiac impairments that diminish the heart's ability to pump blood effectively, leading to insufficient perfusion to satisfy metabolic requirements.[1] As a leading cause of hospitalization and disability in older adults, CHF presents significant challenges to healthcare systems worldwide.[2] Epidemiological data indicate that there are more than 26 million cases globally, with escalating morbidity and mortality rates attributable to aging populations and the increasing prevalence of comorbidities.[3, 4] The prognosis for CHF remains poor, with a five-year mortality rate of approximately 50% following diagnosis.[5] Older adults are disproportionately susceptible to adverse outcomes associated with CHF due to age-related physiological decline, multimorbidity, and reduced physiological reserve.[6] These pathophysiological processes not only heighten mortality risks but also compromise physical and psychological well-being, resulting in substantially reduced quality of life, frequent hospitalizations, and greater caregiver dependency.[7]

From a metabolic perspective, adequate nutrition is crucial for preserving myocardial contractility and cardiac efficiency, acting as a protective mechanism against energy depletion in failing hearts.[8] However, as CHF progresses, malnutrition disrupts this balance, leading to immune dysfunction, prolonged hospitalization, increased readmission rates, and heightened mortality risk.[9] Clinically, malnutrition presents across a spectrum, ranging from reduced appetite and weight loss to sarcopenia, severe cardiac cachexia, chronic inflammation, and metabolic derangements.[10] Alarmingly, the prevalence of malnutrition increases with disease severity, affecting up to 90% of CHF patients. This increase is driven by factors such as aging, multiple comorbidities, and intestinal malabsorption.[11] Hospitalization further exacerbates nutritional decline, as patients face acute stressors, dietary inconsistencies, and physiological deterioration.[12] Despite its significant impact on patient outcomes, malnutrition remains underdiagnosed due to its complex etiology and

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the lack of robust screening methods. This highlights the need for innovative strategies to accurately predict malnutrition risk and enable early intervention, ultimately enhancing clinical care and patient quality of life.

Machine learning (ML) provides a transformative approach to addressing complex clinical challenges through its capacity to analyze multidimensional datasets, identify subtle predictive patterns, and generate individualized risk assessments.[13] In healthcare, ML has emerged as a powerful tool for prognostication in HF, demonstrating efficacy in predicting mortality, readmissions, and therapeutic strategy optimization.[14] For example, ML models have been employed to predict three-year all-cause mortality in HF patients using echocardiographic phenotypes[15] and to assess the in-hospital prognosis of acute HF patients through electronic health record (EHR) data.[16] These studies underscore ML's ability to detect nuanced associations that may not be apparent with conventional statistical methods. Despite these advancements, the application of ML to address malnutrition—a critical yet often underrecognized comorbidity in older adults with CHF—remains underexplored. Current screening tools (e.g., MUST, COUNT) exhibit limitations due to their insufficient specificity for HF pathophysiology and failure to incorporate disease-specific factors.[17, 18] This gap highlights the potential for ML-based approaches to improve the accuracy of nutritional risk assessments by integrating real-time clinical, nutritional, and biomarker data.

Therefore, this study aims to construct and validate an ML-based model for identifying and stratifying the risk of malnutrition in hospitalized older adults with CHF. By leveraging comprehensive clinical data, we developed and rigorously validated a screening model designed to assist healthcare professionals in identifying patients at increased risk of malnutrition. While further prospective studies are needed to confirm clinical utility, this model represents a preliminary tool that may support early risk stratification, enabling clinicians to consider tailored nutritional interventions as part of broader patient management strategies.

METHODS

Study design and patients

In this prospective study, data were collected from 1,128 older adults diagnosed with CHF at two medical centers (Heping and Hunnan) affiliated with the First Affiliated Hospital of China Medical University between January 2021 and December 2024. The model development cohort, consisting of participants enrolled at Heping Medical Center from January 2021 to June 2023, was randomly divided into a training set and an internal testing set at a 7:3 ratio. Participants recruited from Hunnan Medical Center between July 2023 and December 2024 constituted the external validation set. This study received approval from the Ethics Committee of the First Affiliated Hospital of China Medical University (Ethical Approval No. EC-2021-187-2) and was conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants prior to the study.

Inclusion criteria were as follows: (1) age ≥ 60 years; (2) confirmed diagnosis of CHF according to the established guidelines;1 (3) New York Heart Association (NYHA) functional class II-IV with clinical stability; (4) conscious and able to provide written informed consent; (5) incomplete data <10%. Exclusion criteria included: (1) newly diagnosed acute heart failure (HF) during hospitalization (e.g., acute myocardial infarction) or specific cardiac conditions (e.g., congenital heart disease, hyperthyroid heart disease, or cardiac transplantation); (2) comorbidities associated with increased short-term mortality risk or severe organ dysfunction (e.g., life-threatening thromboembolic events, severe infection, active malignancies, acute pancreatitis, hepatic/renal failure, or uncontrolled hyperthyroidism); (3) history of gastric bypass surgery, inability to tolerate oral intake, or diagnosed anorexia nervosa; (4) receipt of ongoing long-term nutritional support at admission; (5) cognitive impairment or psychiatric disorders; (6) treatment in intensive care or surgical units. Following comprehensive screening, 1,128 patients met the eligibility requirements and were enrolled in this study. The participant selection workflow is illustrated in Figure 1.

Data collection

Clinical data from all participants were prospectively collected and categorized as follows: (1) Demographic variables: age, gender (men/women), residence (rural/urban), education level (below high school or above), monthly household income (yuan), body mass index (BMI, kg/m2), living arrangement (living alone or not), dentition status (number of natural teeth), current smoking status, and alcohol consumption. (2) Functional capacity: handgrasp strength (kg), mid-upper arm circumference (cm), and calf circumference (CC, cm). (3) Clinical data: CHF duration (months), NYHA classification (II-IV), comorbidities (peripheral edema, hypertension, dyslipidemia, diabetes, chronic obstructive pulmonary disease [COPD], coronary heart disease [CHD], atrial fibrillation, valvular heart disease, anemia, chronic kidney disease [CKD], and gastrointestinal disease), and medication usage (diuretics, mineralocorticoid receptor antagonist [MRA], angiotensin-converting enzyme inhibitors/angiotensin II receptor blockers [ACE-I/ARB], β-blockers, and total number of prescribed medications). (4) Laboratory parameters assessed within 24 h of hospitalization included: total protein (TP, g/L), hemoglobin (HGB, g/L), creatinine (Cr, mg/dL), sodium (Na, mmol/L), potassium (K, mmol/L), fasting blood glucose (FBG, g/L), estimated glomerular filtration rate (eGFR, mL/min/1.73m^2), B-type natriuretic peptide (BNP, mg/L), N-terminal pro-B-type natriuretic peptide (NT-proBNP, ng/L), white blood cell count (WBC, 10^9/L), albumin (Alb, mg/L), neutrophils (Neu, 10^9/L), lymphocytes (Lym, 10^9/L), platelets (PLT, 10^9/L), monocytes (Mon, 10^9/L), neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), lymphocyte-to-monocyte ratio (LMR), and prognostic nutritional index (PNI).

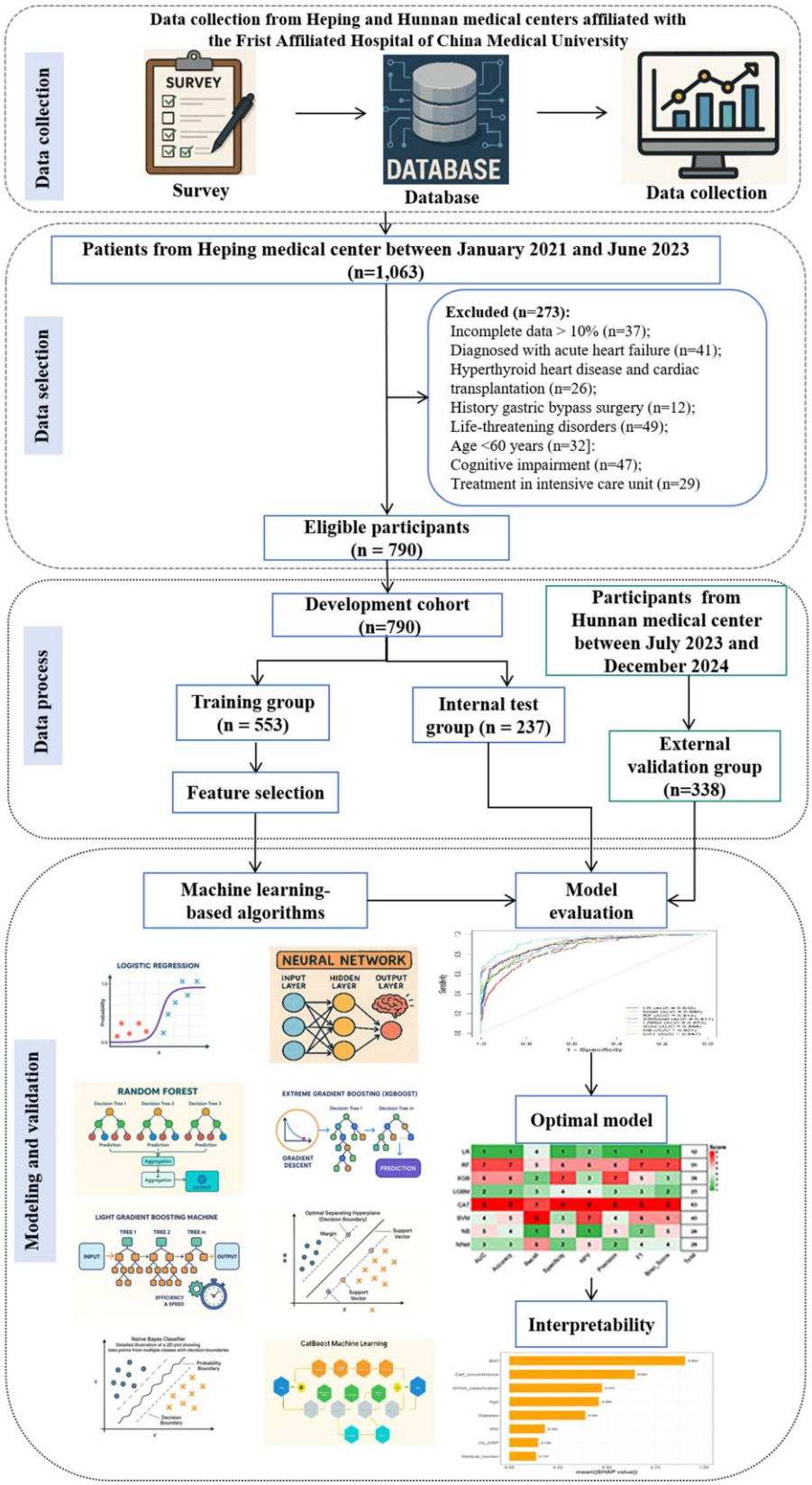

Figure 1. Flowchart of the participant enrolment and study design.

Diagnostic criteria of malnutrition

Malnutrition was diagnosed according to the Global Leadership Initiative on Malnutrition (GLIM) consensus framework,[19] employing a validated two-step approach specifically designed for older adults with CHF. All participants underwent a systematic nutritional evaluation within 24 h of hospitalization. The Nutritional Risk Screening 2002 (NRS-2002) tool was utilized to identify individuals at nutritional risk.[20] Patients with an NRS-2002 score ≥3 underwent a confirmatory evaluation for malnutrition, which required meeting at least one phenotypic criterion and one etiological criterion in accordance with GLIM guidelines. Phenotypic criteria included: weight loss >5% within six months or >10% beyond six

months; BMI <18.5 kg/m^2 for individuals aged ≤70 years or <20 kg/m2 for those >70 years; and reduced muscle mass, assessed via CC measurements (≤30 cm for men; ≤29.5 cm for women).[21] Etiological criteria included chronic inflammation, disease burden, reduced food intake, or impaired nutrient assimilation. In this cohort, all participants inherently satisfied the disease burden criterion owing to their CHF diagnosis.

Data processing and feature selection

Variables with >10% missing values were excluded to avoid unreliable imputation. Remaining missing values were imputed using MissForest, a non-parametric random forest-based algorithm, selected for its ability to model complex interactions in clinical data.[22] Missing data patterns in the model development cohort before and after imputation are compared in Supplementary Figure 1. The dataset was split into training (70%) and internal testing (30%) sets using stratified random sampling to preserve outcome distribution. Categorical variables were converted into factors with clinically meaningful labels to ensure proper handling in downstream analyses. Continuous features were standardized to zero mean and unit variance using preproc_pipeline.fit_transform, ensuring the transformation pipeline was fitted exclusively on the training set to prevent leakage.

In this study, feature selection was conducted using the Least Absolute Shrinkage and Selection Operator (LASSO) regression with an L1 penalty, followed by multivariable logistic regression. LASSO regression identified risk factors with non-zero coefficients, thereby optimizing model generalizability. The optimal regularization parameter (λ) was selected via ten-fold cross-validation using the 1-standard-error rule (λ-1se). This approach balances feature sparsity and predictive performance by selecting the largest λ value within one standard error of the minimum cross-validated mean squared error (MSE). This criterion prioritizes model simplicity to mitigate overfitting risks.[23] Variables retained by LASSO were subsequently incorporated into the multivariable logistic regression model, with coefficients estimated using maximum likelihood.

Modeling

This study employed a comprehensive range of ML algorithms to ensure robust analytical outcomes. The implemented models included Logistic Regression (LR), Neural Network (NNet), Support Vector Machine (SVM), Naïve Bayes (NB), Random Forest (RF), Extreme Gradient Boosting (XGB), Light Gradient Boosting Machine (LGBM), and CATBoost (CAT). Detailed descriptions of these algorithms, including their theoretical underpinnings, are provided in Supplementary Table 1. All eight models were trained using identical input features to ensure methodological consistency. Hyperparameter optimization was conducted using grid search and randomized search strategies, integrated with five-fold cross-validation on the training dataset. This approach facilitated the systematic identification of optimal hyperparameter configurations for each model, with the area under the receiver operating characteristic curve (AUC-ROC) serving as the primary optimization metric. Detailed hyperparameter tuning results are presented in Supplementary Table 2. The incorporation of cross-validation enhanced model robustness, mitigated the risk of overfitting, and improved the generalizability of predictive performance across heterogeneous datasets.

Validation

Model performance was rigorously validated using a combination of quantitative metrics and graphical analyses. Eight evaluation metrics were calculated: AUC, accuracy, precision, recall, specificity, negative predictive value (NPV), F1 score, and Brier score.[24, 25] Graphical assessments included calibration curves to evaluate the agreement between predicted probabilities and observed outcomes and decision curve analysis (DCA) to assess clinical net benefit across probability thresholds. Additionally, a composite scoring system adapted from established methodologies was developed to provide a holistic ranking of model performance.[24, 26] This system integrated all eight metrics, assigning equal weights to each, to generate a cumulative score on a 0-64 scale, where higher scores indicate superior predictive performance.

Model explainability

Interpretability is essential in clinical ML to ensure model trustworthiness and provide clinically actionable insights. In this study, Shapley Additive exPlanations (SHAP), a game theory-based framework, was employed to elucidate the decision logic of the final model.[27] SHAP quantifies individual feature contributions to predictions by calculating Shapley values, which reflect the marginal impact of each feature relative to baseline expectations. These values enable dual interpretability: global interpretability that aggregates feature importance across the dataset and local interpretability that attributes feature-level contributions to individual predictions, thereby reconciling the complexity of "black-box" models with clinician-friendly explanations. To enhance transparency, SHAP-derived insights were visualized through summary plots, force plots, and waterfall plots. These visual tools support rigorous model auditing and facilitate the translation of predictions into context-specific clinical interventions.

Statistical analysis

All statistical analyses were performed using SPSS software (version 27.0, IBM Corp.) and R software (version 4.4.1, Foundation for Statistical Computing). Continuous variables, which were assessed for non-normal distribution, were reported as median with interquartile range (IQR). Categorical variables were presented as frequencies and proportions (%). Group differences in categorical variables were evaluated using the chi-square test, while non-parametric Wilcoxon rank-sum tests were applied to continuous variables. Potential risk variables were quantified as odds ratios (ORs) with corresponding 95% confidence intervals (CIs). A p-value (two-tailed) < 0.05, after adjustment for multiple comparisons using false discovery rate (FDR), was considered statistically significant.[28]

RESULTS

Patient's baseline characteristics

The model development cohort consisted of 790 hospitalized older CHF patients, with a median age of 74.0 years [IQR: 67.0, 80.0]. Among these participants, 56.2% (444/790) were women, and 85.7% (677/790) exhibited severe functional impairment (NYHA class III/IV). Malnutrition was identified in 44.1% of the cohort (348/790), whereas the remaining 55.9% (442/790) maintained a normal nutritional status. Comparative baseline characteristics between the malnutrition and non-malnutrition groups were detailed in Table 1. For model development, the cohort was divided into a training subset (n = 553, 70%) and an internal testing subset (n = 237, 30%), with malnutrition prevalence rates of 45.4% (n = 251) and 40.9% (n = 97), respectively. As shown in Table S3, the baseline characteristics between the training and internal testing cohorts showed no statistically significant differences (all $p > 0.05$).

External validation was conducted using an independent cohort consisting of 338 hospitalized older CHF adults. The prevalence of malnutrition in this validation cohort was 40.5% (137/338), closely aligning with the prevalence observed in the model development cohort (44.1%). Baseline clinical characteristics of both the development and external validation cohorts were compared and presented in Supplementary Table 4.

Variable selection via LASSO regression

We employed LASSO regression with an L1 penalty term to optimize feature selection by shrinking the coefficients of redundant predictors to zero (Figure 2A). The optimal regularization parameter ($\lambda = 0.043$) was selected via ten-fold cross-validation using the λ-1se method (dashed blue vertical line), effectively balancing predictive accuracy with model complexity reduction (Figure 2B). This approach yielded eight robust risk factors: age, BMI, CC, diabetes, hs-CRP, polypharmacy (≥ 5 medications), NYHA classification, and PNI (Figure 2C). These variables were subsequently analyzed using multivariable logistic regression, which confirmed significant associations with malnutrition risk (all $p < 0.05$; Figure 2D). Following correction for the FDR, all eight risk variables retained statistical significance (Supplementary Table 5). Consequently, these variables were selected for final model construction.

Modeling and performance evaluation

The model development process utilized a grid search strategy for hyperparameter optimization, in which configurations were systematically selected to maximize ROC-AUC performance. In the training set, the CAT algorithms achieved superior discriminative performance (AUC = 0.947, 95% CI: 0.930-0.964), followed by RF (AUC = 0.914, 95% CI: 0.891-0.937) and XGB (AUC = 0.911, 95% CI: 0.888-0.935) (Figure 3A and 3B, Supplementary Table 6). Furthermore, the CAT model exhibited robust performance across various metrics, achieving the highest composite score (63/64) in a multidimensional assessment framework (Figure 3C). Calibration analysis demonstrated favorable alignment of the CAT model's predicted probabilities with observed outcomes, as evidenced by its proximity to the ideal calibration curve and the lowest Brier score (0.099) (Figure 3D). DCA indicated strong clinical utility across all models, with CAT and SVM algorithms generating the highest net benefit across threshold probabilities (Figure 3E).

Internal and external validation

All eight ML models underwent rigorous internal and external validation analyses. As illustrated in Figure 4A and Supplementary Table 6, the CAT model demonstrated superior discriminative performance in the internal testing set, achieving an AUC of 0.901 (95% CI: 0.858-0.943), alongside an accuracy of 0.840, recall of 0.753, F1 score of 0.794, specificity of 0.900, and NPV of 0.840. In the external validation set, the CAT model maintained strong discriminative capabilities across various metrics (Figure 4A and Supplementary Table 6), though a marginal reduction in AUC (0.916, 95% CI: 0.887-0.945) was observed relative to the training set. When evaluated using a predefined composite scoring system, the CAT model attained the highest total scores of 62 and 57 in the internal and external validation sets, respectively (Figure 4B), indicating its consistent superiority over comparator algorithms. Calibration performance, assessed via calibration curves and Brier scores (Figure 5A and Supplementary Table 6), further underscored the CAT model's reliability, with predicted probabilities exhibiting close alignment to observed event rates in both internal and external cohorts. DCA reinforced the clinical applicability of the CAT model, revealing favorable net benefits across a broad range of threshold probabilities in internal and external datasets (Figure 5B). Collectively, these findings substantiated the CAT algorithm as the optimal model for malnutrition risk stratification in hospitalized older adults with CHF.

Feature importance and individual prediction

To elucidate the relationships between the top-performing CAT model and the underlying data, we employed SHAP to generate interpretable visualizations of feature contributions to malnutrition risk probabilities. The SHAP summary plot (Figure 6A) identified the most influential risk factors in the CAT model, including BMI, CC, NYHA classification, age, and diabetes, ranked in descending order of importance. Notably, the feature importance rankings exhibited consistency between internal and external validation cohorts (Supplementary Figure 2 and 3), underscoring the model's generalizability. Furthermore, the SHAP beeswarm plot (Figure 6B) delineated individualized feature contributions to the model's risk stratification, where positive SHAP values (depicted in black) corresponded to an elevated probability of malnutrition risk, whereas negative values (depicted in grey) were associated with reduced risk. This bidirectional influence highlights the nuanced interplay of variables in shaping patient-specific predictions.

To enhance model interpretability and visualize individualized risk profiles, SHAP waterfall plots were constructed for representative true-positive (Figure 6C) and true-negative cases (Figure 6D). Arrow directions represented the magnitude and directionality of each risk factor's influence on malnutrition risk, with black hues

Table 1. Baseline characteristics of the participants

Parameters	Total (n = 790)	Malnutrition (n = 348)	Non-malnutrition (n = 442)	Statistics	*p* value
Age	74.0 [67.0, 80.0]	76.00 [70.3, 81.0]	71.00 [65.0, 79.0]	-6.41	<0.001***
Gender					
Men	346 (43.8%)	157 (45.1%)	189 (42.8%)	0.439	0.508
Women	444 (56.2%)	191 (54.9%)	253 (57.2%)		
Residence					
Rural (%)	349 (44.2%)	152 (43.7%)	197 (44.6%)	0.063	0.802
Urban (%)	441 (55.8%)	196 (56.3%)	245 (55.4%)		
Education level					
< high school	518 (65.6%)	228 (65.5%)	290 (65.6%)	0.001	0.978
≥high school	272 (34.4%)	120 (34.5%)	152 (34.4%)		
Monthly household income (Yuan)					
<3000	353 (44.7%)	163 (46.8%)	190 (43.0%)	1.21	0.547
3000-5000	293 (37.1%)	125 (35.9%)	168 (38.0%)		
>5000	144 (18.2%)	60 (17.2%)	84 (19.0%)		
BMI (kg/m^2)	24.2 [20.6, 27.4]	22.6 [18.1, 27.0]	24.9 [22.3, 27.8]	-7.54	<0.001***
Current smoking (%)	298 (37.7%)	144 (41.4%)	154 (34.8%)	3.54	0.060
Current drinking (%)	294 (37.2%)	132 (37.9%)	162 (36.7%)	0.136	0.712
Disease duration (months)					
≤6	594 (75.2%)	261 (75.0%)	333 (75.3%)	0.012	0.913
>6	196 (24.8%)	87 (25.0%)	109 (24.7%)		
NYHA classification					
II	113 (14.3%)	22 (6.3%)	91 (20.6%)	66.5	<0.001***
III	403 (51.0%)	157 (45.1%)	246 (55.7%)		
IV	274 (34.7%)	169 (48.6%)	105 (23.8%)		
Teeth number					
≥20	321 (40.6%)	129 (37.1%)	192 (43.4%)	3.28	0.070
<20	469 (59.4%)	219 (62.9%)	250 (56.6%)		
Living alone (%)	71 (9.0%)	38 (10.9%)	33 (7.5%)	2.84	0.092
Comorbidities					
Peripheral edema (%)	459 (58.1%)	201 (57.8%)	258 (58.4%)	0.030	0.862
Hypertension (%)	511 (64.7%)	223 (64.1%)	288 (65.2%)	0.099	0.753
Dyslipidemia (%)	366 (46.3%)	164 (47.1%)	202 (45.7%)	0.159	0.690

BMI, Body Mass Index; NYHA, New York Heart Association; COPD, Chronic Obstructive Pulmonary Disease; CHD, Coronary Heart Disease; CKD, Chronic Kidney Disease; MRA, Mineralocorticoid Receptor Antagonist; ACEI/ARB, Angiotensin-Converting Enzyme Inhibitor/Angiotensin II Receptor Blocker; TP, Total Protein; HGB, Hemoglobin; Cr, Creatinine; Hs-CRP, High-sensitivity C-Reactive Protein; Na, Sodium; K, Potassium; FBG, Fasting Blood Glucose; NT-proBNP, N-Terminal pro B-type Natriuretic Peptide; WBC, White Blood Cell; Alb, Albumin; Neu, Neutrophil; Lym, Lymphocyte; PLT, Platelet; Mon, Monocyte; NLR, Neutrophil-to-Lymphocyte Ratio; PLR, Platelet-to-Lymphocyte Ratio; PNI, Prognostic Nutritional Index.
Continuous values are presented as median [IQR] and category values are presented as frequency (%).
**$p < 0.01$, ***$p < 0.001$.

Table 1. Baseline characteristics of the participants (n = 790)

Parameters	Total (n = 790)	Malnutrition (n = 348)	Non-malnutrition (n = 442)	Statistics	*p* value
Comorbidities					
COPD (%)	156 (19.7%)	73 (21.0%)	83 (18.8%)	0.594	0.441
CHD (%)	479 (60.6%)	213 (61.2%)	266 (60.2%)	0.086	0.770
Atrial fibrillation (%)	272 (34.4%)	125 (35.9%)	147 (33.3%)	0.611	0.434
Valvular heart disease (%)	211 (26.7%)	90 (25.9%)	121 (27.4%)	0.228	0.633
Diabetes (%)	227 (28.7%)	147 (42.2%)	80 (18.1%)	55.4	<0.001***
Anemia (%)	358 (45.3%)	167 (48.0%)	191 (43.2%)	1.79	0.181
CKD (%)	281 (35.6%)	135 (38.8%)	146 (33.0%)	2.82	0.093
Gastrointestinal disease (%)	301 (38.1%)	160 (46.0%)	141 (31.9%)	16.4	<0.001***
Functional capacity					
Hand grasp (kg)	17.0 [13.0, 21.0]	17.0 [14.0, 21.0]	18.0 [13.0, 22.0]	-0.801	0.423
Upper arm circumference (cm)	24.0 [20.0, 30.0]	25.0 [20.0, 30.0]	24.0 [19.0, 29.0]	-1.75	0.081
Calf circumference (cm)	34.0 [31.0, 37.0]	33.0 [30.0, 36.0]	35.0 [32.0, 37.0]	-6.76	<0.001***
Medication					
Diuretics (%)	536 (67.8%)	233 (67.0%)	303 (68.6%)	0.228	0.633
MRA (%)	678 (85.8%)	302 (86.8%)	376 (85.1%)	0.470	0.493
β-blocker (%)	438 (55.4%)	188 (54.0%)	250 (56.6%)	0.508	0.476
ACEI/ARB (%)	587 (74.3%)	261 (75.0%)	326 (73.8%)	0.158	0.691
Medical number (≥5)	306 (38.7%)	169 (48.6%)	137 (31.0%)	25.3	<0.001***
Laboratory tests					
TP (g/L)	54.0 [46.0, 63.0]	53.5 [46.0, 63.0]	54.0 [46.0, 63.0]	-0.331	0.740
HGB (g/L)	133 [121, 147]	134 [123, 147]	132 [120, 148]	-0.810	0.418
Cr (mg/dL)	1.50 [1.10, 1.90]	1.50 [1.10, 1.90]	1.50 [1.10, 1.80]	-1.12	0.264
Hs-CRP (<0.5mg/L)	503 (63.7%)	251 (72.1%)	252 (57.0%)	19.2	<0.001***
Na (mmol/L)	135 [129, 141]	136 [130, 141]	135 [129, 141]	-0.584	0.559
K (mmol/L)	4.10 [3.80, 4.50]	4.14 [3.80, 4.50]	4.10 [3.80, 4.50]	-0.072	0.943
FBG (g/L)	5.30 [4.70, 5.90]	5.30 [4.70, 5.90]	5.30 [4.60, 5.93]	-0.036	0.971
eGFR (ml/min/1.73m^2)	70.0 [56.0, 87.0]	68.0 [55.3, 84.0]	72.0 [56.0, 88.0]	-1.72	0.085
BNP (mg/L)	372 [222, 491]	379 [244, 496]	351 [210, 484]	-1.85	0.064
NT-proBNP (ng/L)	2989 [2032, 3938]	3059 [2096, 4077]	2950 [1974, 3852]	-1.64	0.102
Alb (mg/L)	36.0 [28.0, 41.0]	35.0 [28.0, 40.0]	37.0 [29.0, 42.0]	-3.25	0.001**
WBC (10^9/L)	10.1 [8.38, 12.5]	10.0 [8.30, 12.6]	10.2 [8.40, 12.4]	-0.005	0.996

BMI, Body Mass Index; NYHA, New York Heart Association; COPD, Chronic Obstructive Pulmonary Disease; CHD, Coronary Heart Disease; CKD, Chronic Kidney Disease; MRA, Mineralocorticoid Receptor Antagonist; ACEI/ARB, Angiotensin-Converting Enzyme Inhibitor/Angiotensin II Receptor Blocker; TP, Total Protein; HGB, Hemoglobin; Cr, Creatinine; Hs-CRP, High-sensitivity C-Reactive Protein; Na, Sodium; K, Potassium; FBG, Fasting Blood Glucose; NT-proBNP, N-Terminal pro B-type Natriuretic Peptide; WBC, White Blood Cell; Alb, Albumin; Neu, Neutrophil; Lym, Lymphocyte; PLT, Platelet; Mon, Monocyte; NLR, Neutrophil-to-Lymphocyte Ratio; PLR, Platelet-to-Lymphocyte Ratio; PNI, Prognostic Nutritional Index.
Continuous values are presented as median [IQR] and category values are presented as frequency (%).
**$p < 0.01$, ***$p < 0.001$.

Table 1. Baseline characteristics of the participants (cont.)

Parameters	Total (n = 790)	Malnutrition (n = 348)	Non-malnutrition (n = 442)	Statistics	*p* value
Neu (10^9/L)	8.00 [6.00, 11.0]	9.00 [6.00, 11.8]	8.00 [5.00, 10.0]	-2.85	0.004**
Lym (10^9/L)	1.70 [1.40, 2.00]	1.60 [1.40, 1.90]	1.70 [1.30, 2.10]	-1.31	0.189
PLT (10^9/L)	190 [145, 246]	190 [145, 244]	190 [145, 247]	-0.496	0.620
Mon (10^9/L)	0.52 [0.28, 0.73]	0.50 [0.25, 0.72]	0.53 [0.30, 0.73]	-1.26	0.208
NLR	4.71 [3.16, 6.67]	5.00 [3.46, 6.87]	4.44 [2.94, 6.36]	-3.06	0.002**
PLR	111 [81.8, 155]	113 [85.2, 150]	108 [77.0, 156]	-0.704	0.482
PNI	44.5 [38.0, 50.0]	44.0 [37.0, 48.5]	45.5 [38.5, 51.0]	-3.52	<0.001***

BMI, Body Mass Index; NYHA, New York Heart Association; COPD, Chronic Obstructive Pulmonary Disease; CHD, Coronary Heart Disease; CKD, Chronic Kidney Disease; MRA, Mineralocorticoid Receptor Antagonist; ACEI/ARB, Angiotensin-Converting Enzyme Inhibitor/Angiotensin II Receptor Blocker; TP, Total Protein; HGB, Hemoglobin; Cr, Creatinine; Hs-CRP, High-sensitivity C-Reactive Protein; Na, Sodium; K, Potassium; FBG, Fasting Blood Glucose; NT-proBNP, N-Terminal pro B-type Natriuretic Peptide; WBC, White Blood Cell; Alb, Albumin; Neu, Neutrophil; Lym, Lymphocyte; PLT, Platelet; Mon, Monocyte; NLR, Neutrophil-to-Lymphocyte Ratio; PLR, Platelet-to-Lymphocyte Ratio; PNI, Prognostic Nutritional Index.
Continuous values are presented as median [IQR] and category values are presented as frequency (%).
**$p < 0.01$, ***$p < 0.001$.

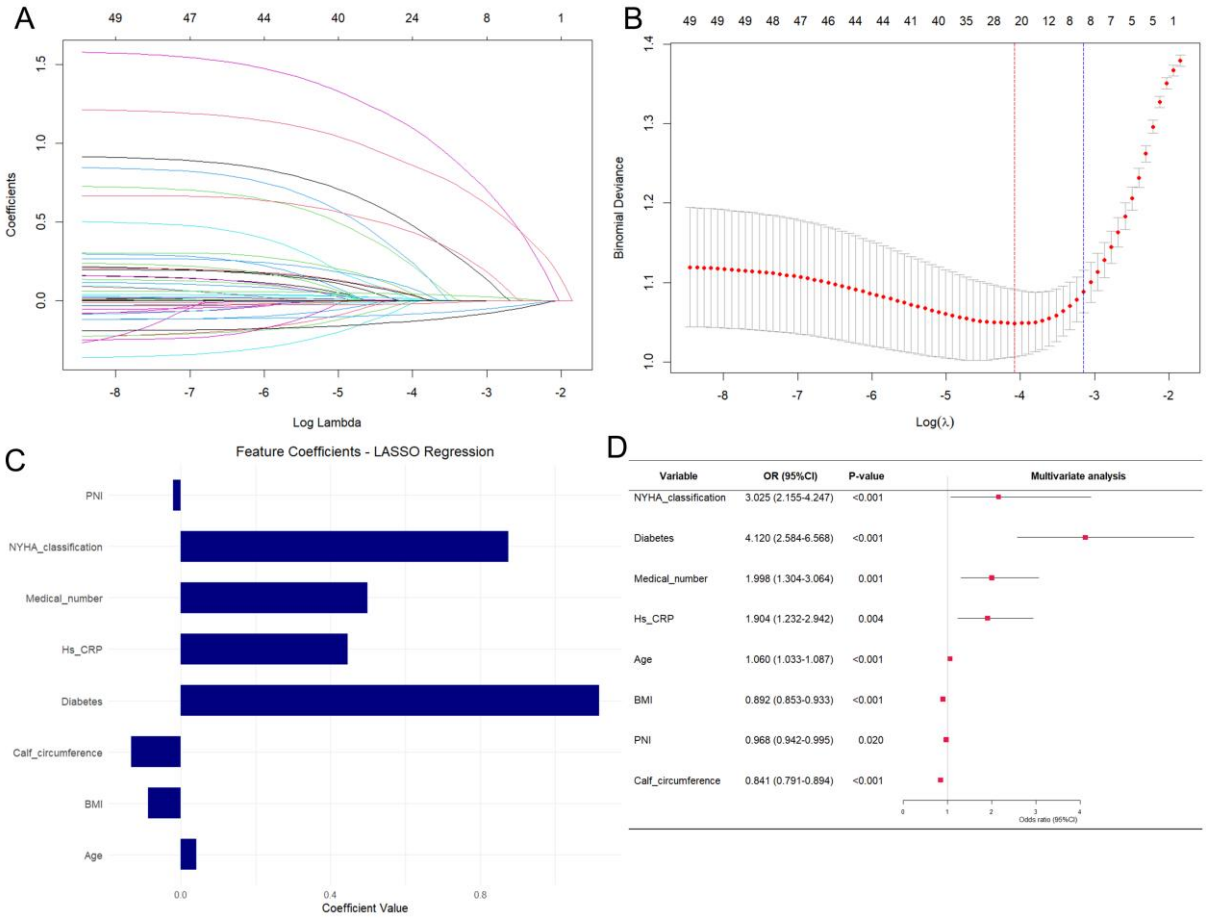

Figure 2. Variable selection using LASSO regression and multivariable regression analysis. (A) LASSO coefficient path plots illustrating variables shrinkage across increasing penalty parameters. (B) Ten-fold cross-validation curve for selecting the optimal penalty parameter (λ) in the LASSO regression model. The dashed red vertical line indicates λ-min (the λ value yielding the minimum mean squared error, MSE). The dashed blue vertical line denotes λ-1se (the largest λ within one standard error of the minimum MSE). (C) LASSO regression at the λ-1se value retained eight variables with non-zero coefficients, forming the final feature subset. (D) Independent predictors of malnutrition identified through multivariable regression analysis

denoting risk-elevating factors and grey hues signifying protective effects. The summation of individual risk factor contributions yielded the final SHAP value, which quantifies the net deviation from the baseline value. For instance, the representative true-positive case displayed a high SHAP value (3.9), indicative of a pronounced malnutrition risk, whereas the true-negative case exhibited a strongly negative SHAP value (-1.94), reflecting robust protective determinants.

To assess the generalizability of these findings, the SHAP framework was extended to evaluate additional ML models. As illustrated in Supplementary Figure 4, BMI, CC, age, NYHA classification, and diabetes consistently emerged as significant risk factors of malnutrition across all the other models. This recurrent prominence underscores their pivotal role in shaping risk stratification outcomes, irrespective of the algorithmic approach employed.

DISCUSSION

Principal findings

Malnutrition in older adults with CHF is a multifactorial condition driven by metabolic imbalances, reduced dietary intake, and systemic inflammation, contributing to adverse outcomes such as prolonged hospitalization, post-acute facility admission, and increased mortality.[29] Despite its clinical significance, timely identification of high-risk patients remains challenging due to heterogeneous risk profiles and dynamic disease trajectories. Our ML-based model was developed to address this gap by providing a screening tool tailored for clinically actionable risk stratification. The model's design emphasized clinical interpretability and feasibility, with key risk factors—including BMI, CC, age, NYHA classification, polypharmacy, hs-CRP, and PNI—consistent with established biological pathways linking CHF and malnutrition. These variables were rigorously validated through internal and external cohorts. Integration of this tool into EHR systems could enable automated risk alerts during patient admission, potentially prompting clinicians to prioritize nutritional screening or initiate early interventions. While preliminary findings suggest that the model may streamline risk stratification, further prospective studies are required to evaluate its impact on reducing diagnostic delays or optimizing resource allocation in clinical practice.

Comparison with prior work

Our findings revealed a malnutrition prevalence of 44.1% (348/790) among hospitalized older adults with CHF, which was consistent with the previously reported range of 6-60% in the literature.[11] However, Hersberger et al., observed a universal malnutrition risk of 100% (n = 645)

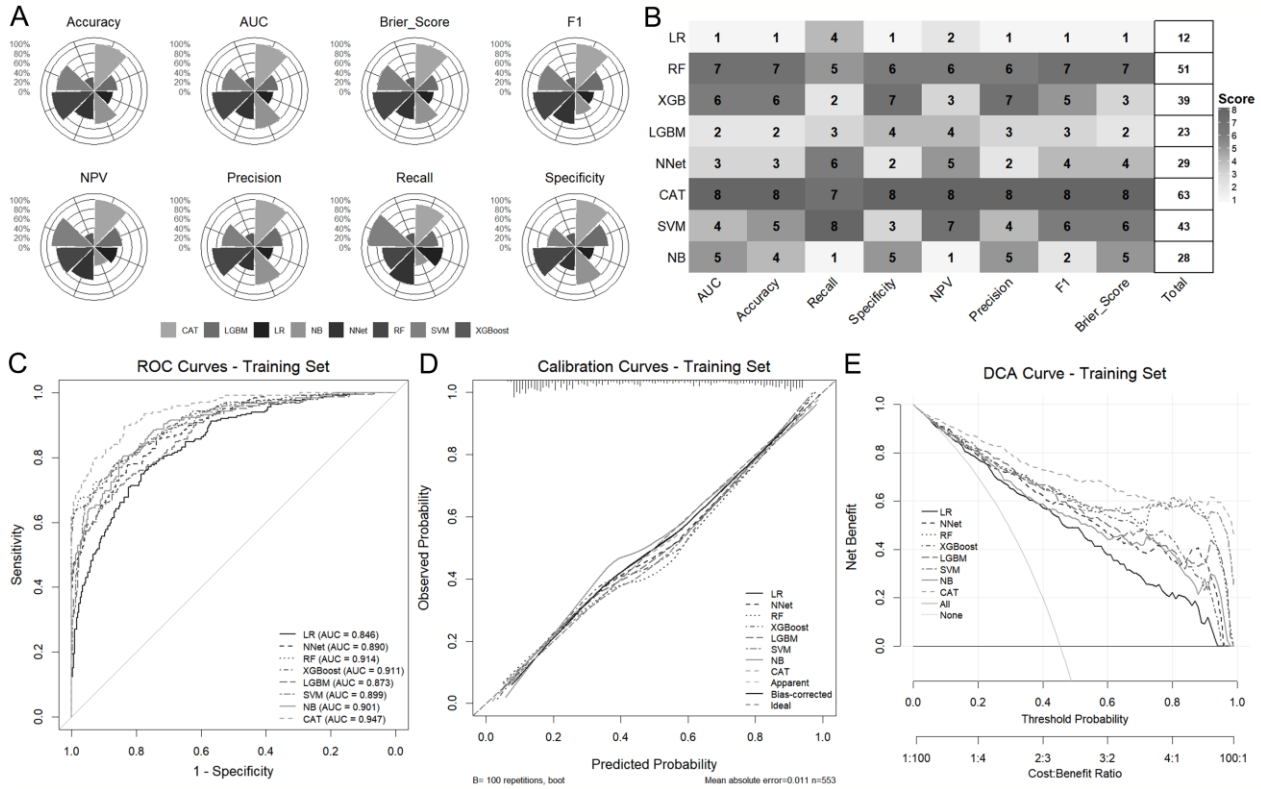

Figure 3. Comparative performance of eight ML models in the training set. (A) Comparison of model performance metrics across all models. (B) ROC curves comparing discrimination performance across models. (C) Heatmap scoring system (total score: 64 points) assessing model performance. Higher scores (red) indicate better performance; lower scores (green) denote poorer performance. (D) Calibration plots comparing predicted probabilities against observed outcomes. Closer alignment to the diagonal (ideal calibration line) reflects improve accuracy. (E) DCA evaluating net clinical benefit across probability thresholds. kindly note that manuscript will be printed in greyscale.

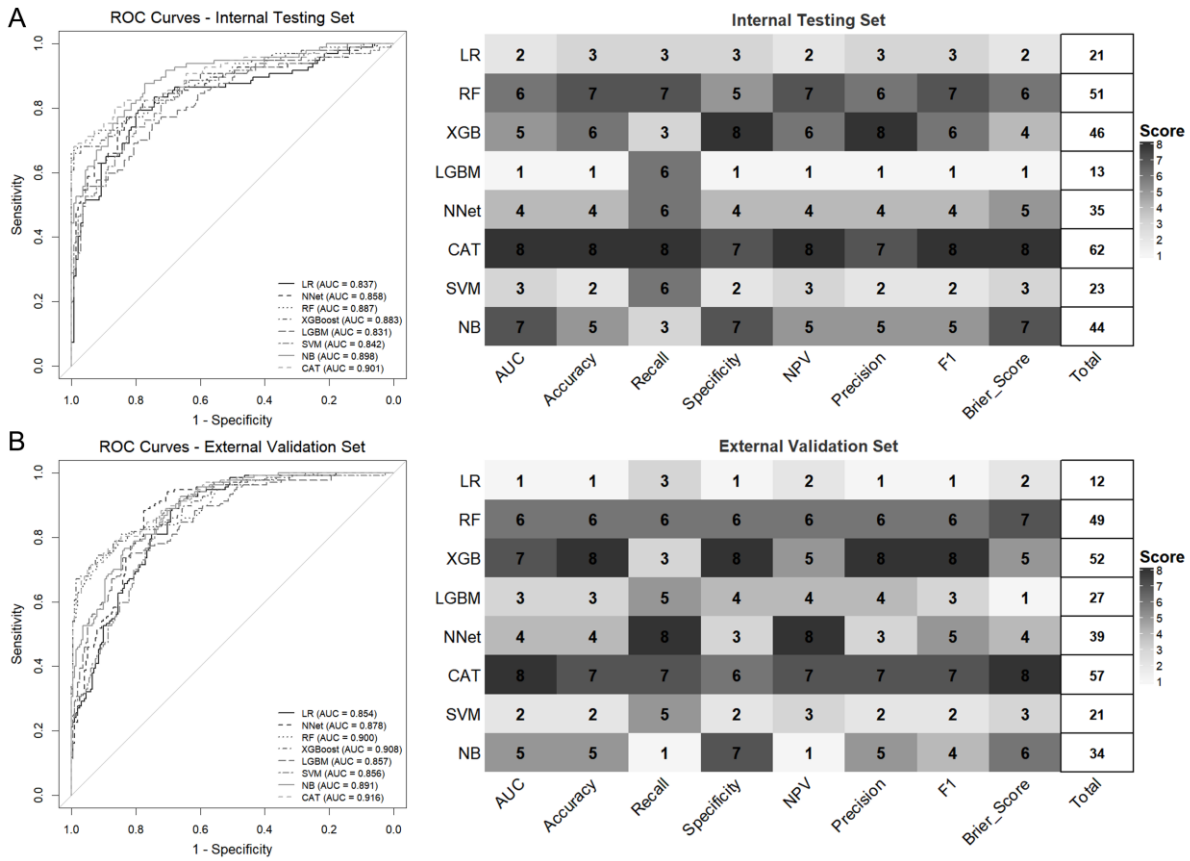

Figure 4. Internal and validation of eight ML models for malnutrition risk stratification. (A) Comparison of performance metrics across all ML models in both cohorts. (B) ROC curves comparing discrimination performance across models in both cohorts

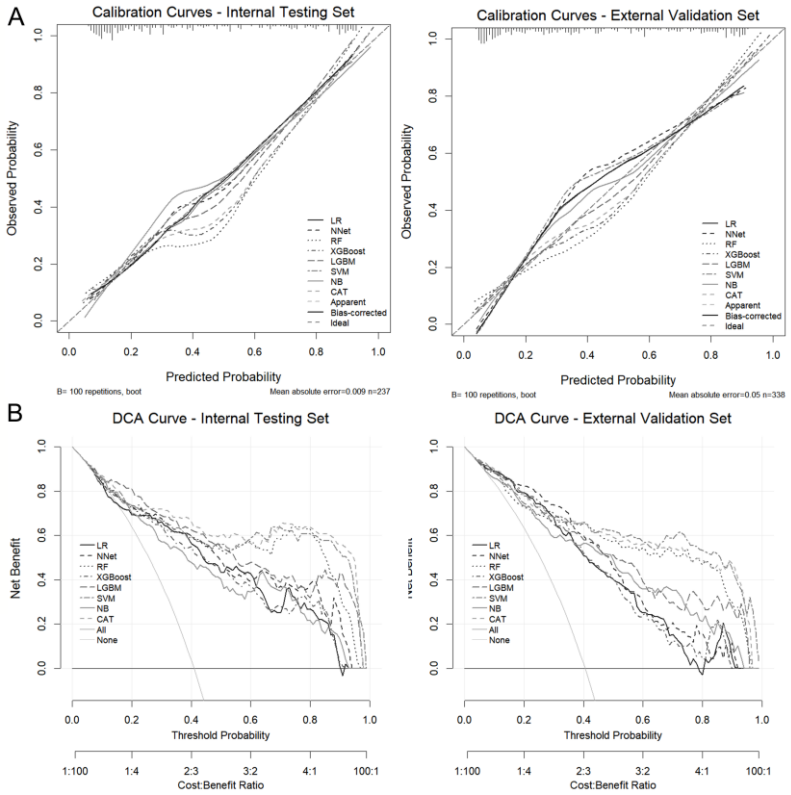

Figure 5. Calibration and clinical utility of the eight ML models in internal and external datasets. (A) Calibration curve evaluation. (B) DCA curve.

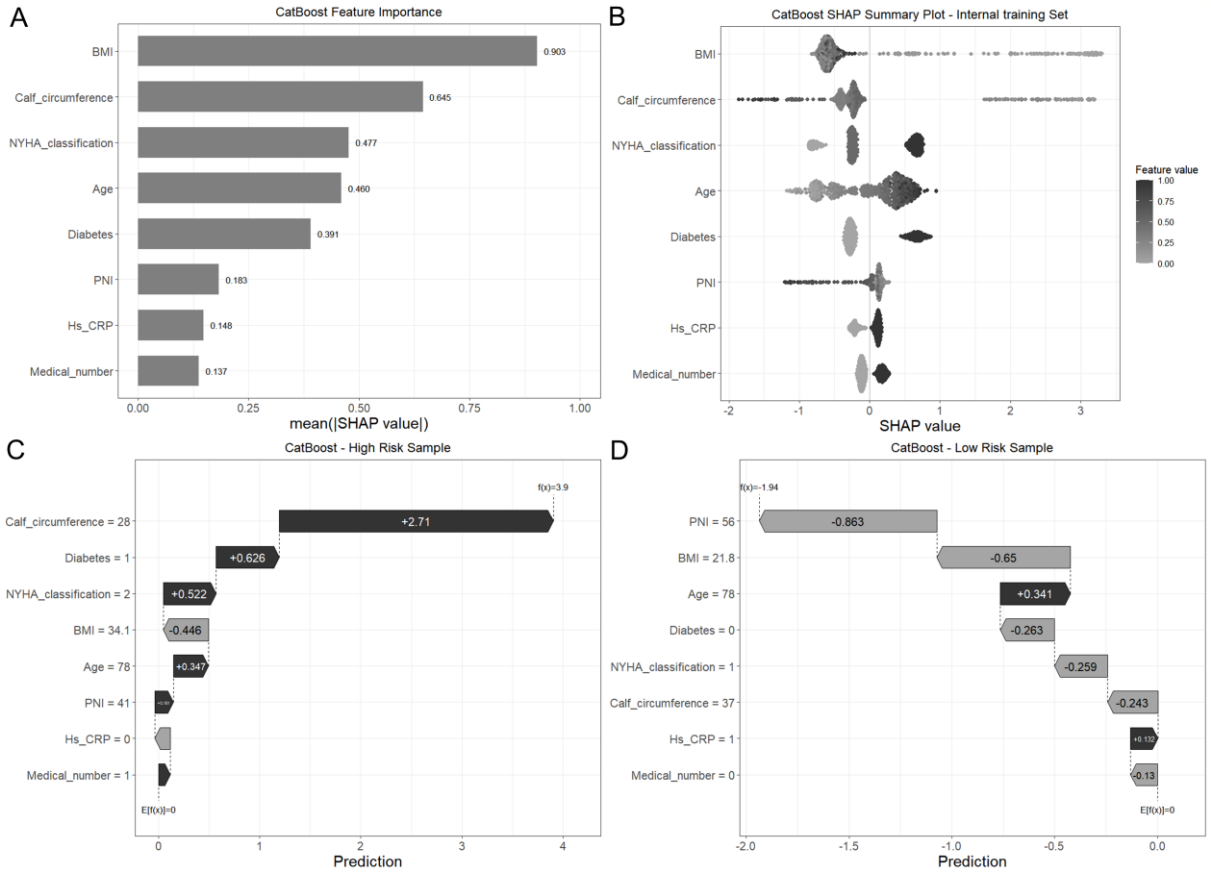

Figure 6. Interpretability of the CatBoost model using SHAP analysis. (A) SHAP summary plot ranking feature importance based on mean absolute SHAP values. (B) Beeswarm plot illustrating feature contributions across the cohort. (C-D) Waterfall plots demonstrating SHAP value contributions for a representative true-positive case (C) and a true-negative case (D). Positive SHAP value (black) increase malnutrition risk; negative values (grey) decrease risk

in CHF patients assessed using the NRS-2002 tool.[30] This disparity likely reflects differences in study populations, diagnostic criteria (e.g., NRS-2002 vs. GLIM criteria), and methodological heterogeneity in nutritional assessment. Recent efforts to predict malnutrition in cardiac populations have resulted in a variety of approaches. Shi et al. developed an XGBoost model to predict postoperative malnutrition in pediatric patients with congenital heart disease, achieving a superior AUC across all outcomes.[31] Similarly, Liu et al. constructed a nomogram for early malnutrition risk stratification in HF patients; however, its reliance on LR limited its adaptability to non-linear relationships.[32] Tang et al. advanced this field by integrating GLIM with LR model, demonstrating strong discriminative power in older HF cohorts.[33] While these studies underscore the value of risk stratification modeling, their scope remains limited to specific subpopulations or conventional statistical methods. Our work addressed a critical unmet need by introducing a ML-based model specifically optimized for older adults with CHF. Distinct from prior efforts, our methodology systematically evaluated eight ML algorithms, identifying the CAT model as superior in balancing interpretability and predictive accuracy. Rigorous hyperparameter tuning and five-fold cross-validation mitigated overfitting risks, while SHAP analysis elucidated clinically plausible risk factors. Therefore, these findings suggest that ML-driven tools, particularly the CAT framework, have the potential to provide a clinically actionable strategy for early malnutrition identification. This approach may facilitate timely and targeted nutritional interventions to mitigate downstream complications in this vulnerable population.

Risk factors for predicting malnutrition among older CHF adults

The SHAP analysis conducted in this study identified advanced age, lower BMI, reduced CC, severe cardiac dysfunction (NYHA class III-IV), reduced PNI, increased hs-CRP, diabetes, and polypharmacy (≥5 medications) as the most influential risk factors for malnutrition in older adults with CHF. The association between advanced age and malnutrition risk likely reflects the interplay of age-related sarcopenia, diminished appetite, and metabolic alterations exacerbated by the progression of CHF. Reduced skeletal muscle mass and fat reserves—evidenced by lower BMI and CC—serve as critical biomarkers of nutritional depletion, which were consistent with studies demonstrating their values in malnutrition screening.[34, 35] For example, an ML-based predictive model developed by Wang et al substantiated that older adults with lower BMI and CC are at an elevated risk of malnutrition.[36] Furthermore, a nomogram model developed by Duan et al. highlighted age and BMI as crucial predictors of malnutrition among hospitalized cancer patients.[37] Concurrently, severe cardiac dysfunction (NYHA class III-IV) intensifies metabolic stress and energy demands, thereby creating a persistent catabolic state that accelerates nutritional depletion.[38] This finding corroborated previous evidence linking advanced functional class in congestive HF patients to a higher prevalence of malnutrition.[39] Additionally, polypharmacy (≥5 medications)—common in the management of advanced CHF—was associated with an increased risk of malnutrition, consistent with studies linking polypharmacy and advanced cardiac disease to nutritional deficiencies.[40] The adverse effects of polypharmacy are multifaceted, encompassing drug-nutrient interactions, gastrointestinal disturbances, and appetite suppression, all of which contribute to the risk of malnutrition.[41]

Furthermore, the role of systemic inflammation in exacerbating malnutrition risk was prominently highlighted in our study. The ML-based model identified diabetes as a significant risk factor for malnutrition, underscoring the complex interplay between metabolic dysregulation and nutritional deterioration in older adults with CHF. These findings corroborate emerging evidence regarding the synergistic mechanisms linking comorbidity burden, chronic inflammation, and cardiac cachexia in this vulnerable population.[42] In patients with concurrent diabetes and CHF, persistent hyperglycemia, insulin resistance, and chronic inflammation—evidenced by elevated hs-CRP levels—are pivotal pathophysiological mechanisms contributing to malnutrition. Hyperglycemia disrupts gastrointestinal motility and mucosal integrity, impairing the bioavailability of essential nutrients, while insulin resistance exacerbates skeletal muscle catabolism, further depleting protein reserves.[43] Compounding these effects, stringent dietary restrictions—particularly carbohydrate limitations imposed on diabetic patients—may inadvertently precipitate micronutrient deficiencies and energy deficits.[44] Notably, in our cohort, reduced PNI levels and elevated hs-CRP levels—reflecting compromised nutritional status and systemic inflammation—were associated with accelerated muscle catabolism and anorexia, likely mediated by cytokine-driven pathways.[45, 46] This observation aligns with recent studies demonstrating the utility of hs-CRP and PNI as predictive biomarkers for malnutrition in patients with chronic conditions.[47, 48] Our results extend this paradigm to CHF, emphasizing that subclinical nutritional and inflammatory biomarkers—often overlooked in routine clinical assessments—may serve as early indicators of nutritional decline.

Clinical implications

These findings of this study hold potential implications for improving the management of hospitalized older patients with CHF. The SHAP analysis identified key risk factors of malnutrition (e.g., BMI, CC, NYHA classification), which may guide clinicians in systematically evaluating high-risk patients and addressing the multifactorial drivers of nutritional decline. Early recognition of these factors could enable targeted interventions to mitigate malnutrition risk, potentially improving clinical outcomes such as reduced hospital readmission rates. For example, incorporating routine assessments of anthropometric parameters (e.g., BMI, CC), cardiac function (NYHA class), and inflammatory and nutritional biomarkers (hs-CRP, PNI) at admission may facilitate timely risk stratification and nutritional support. Furthermore, the integration of this ML-based malnutrition risk stratification model with established nutrition screening tools, such as MUST or NRS-2002, could enhance clinical decision-making for hospitalized older adults with CHF. This hybrid approach may improve early risk detection, particularly in complex

patients with overlapping comorbidities that confound conventional assessments. While these strategies appear promising for optimizing quality of life and long-term outcomes, their clinical efficacy remains to be empirically tested in real-world settings.

Limitations

This study has several limitations that warrant consideration. First, the model was developed and validated using a single-institute cohort, which may introduce selection bias and limit generalizability to populations with divergent demographics, clinical practices, or healthcare ecosystems. Although external validation was performed, the external cohort derived from a geographically adjacent affiliated agency, potentially insufficient to capture broader population heterogeneity. Multicenter studies across diverse healthcare settings are needed to confirm the model's generalizability. Second, while the model demonstrated accuracy for stratifying malnutrition risk at admission, its cross-sectional design precludes insights into dynamic changes in risk during hospitalization or post-discharge. Factors such as treatment responses, dietary interventions, or new comorbidities acquired during hospitalization—which may modulate malnutrition risk—were not accounted for. Future longitudinal studies are required to validate the model's ability to predict evolving risk trajectories. Third, the dataset lacked granular details on psychological status, dietary habits, and caregiver support, all of which are established contributors to malnutrition risk. Incorporating these variables in prospective longitudinal studies could enhance risk stratification accuracy. Fourth, although the CAT algorithm outperformed other models in this study, its integration into clinical workflows necessitates further validation in real-world settings to assess usability and tangible impacts on patient outcomes. Finally, the model's potential to guiding nutritional interventions and improve clinical outcomes remains hypothetical. Randomized controlled trials are required to empirically quantify its clinical utility in patient management. Addressing these limitations will strengthen the translational potential of this tool and support its integration into evidence-based clinical practice for older adults with CHF.

Conclusion

This study developed and validated a ML-based model for stratifying malnutrition risk in hospitalized older adults with CHF. The CAT algorithm outperformed other models regarding discrimination, calibration, and clinical utility in both internal and external validations. By utilizing routinely collected clinical parameters—such as BMI, NYHA class, and hs-CRP—this tool offers a clinically feasible method for the early identification of high-risk patients during admission. While the model's screening accuracy supports its potential utility in guiding nutritional interventions, its efficacy in mitigating adverse outcomes remains to be empirically validated. Future studies should address existing limitations and explore the long-term implications of the risk stratification model.

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DISCLOSURE ON THE USE OF AI AND AI-ASSISTED TECHNOLOGIES

The authors declare that no generative AI or AI-assisted technologies were used in the writing, data collection, or image creation of this manuscript. The machine learning model mentioned in the title refers strictly to the statistical algorithms used for risk stratification, which are fully described in the Methods section. The authors reviewed and edited the content and take full responsibility for the content of the publication.

CONFLICT OF INTEREST AND FUNDING DISCLOSURES

The authors declare that they have no competing interests.

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