

# Development and External Validation of a Machine Learning Model for 90-Day Readmission in Hospitalized Older Patients with AECOPD: A Two-Center Study

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**Background:** Short-term readmission after hospitalization for acute exacerbation of chronic obstructive pulmonary disease (AECOPD) is common in older adults, yet early risk stratification remains limited. We aimed to develop and validate a 90-day readmission model using early admission data.

**Methods:** This retrospective two-center study used a development cohort from the Affiliated Hospital of North Sichuan Medical College and an external validation cohort from Dazhou Integrated Traditional Chinese and Western Medicine Hospital. Predictors were limited to early admission variables harmonized across sites. The neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and systemic immune-inflammation index (SII) were derived, with component blood counts removed to reduce collinearity. Feature selection used stability selection with Elastic Net regularization. Five models were trained and compared: multivariable logistic regression, naïve Bayes (NB), linear discriminant analysis (LDA), gradient boosting machine (GBM), and extreme gradient boosting (XGBoost). Discrimination was assessed by area under the receiver operating characteristic curve (AUC), with internal validation using bootstrap-derived optimism-corrected AUC and Brier score for overall error. Interpretability was examined with Shapley additive explanations (SHAP).

**Results:** A total of 692 patients were included (development, n=513; external validation, n=179). Six predictors were retained: NLR, SII, D-dimer, frequent exacerbations (FE), body mass index (BMI), and albumin (ALB). No strong multicollinearity was detected ( $|r| < 0.90$ ; variance inflation factors (VIFs)  $< 5$ ). XGBoost showed the best discrimination in the development cohort (AUC=0.892) and remained stable after internal validation (optimism corrected AUC=0.864). In the external cohort, XGBoost again achieved the highest AUC (0.847) with a lower Brier score than alternative models. SHAP analyses indicated D-dimer, NLR, and FE as major contributors and suggested non-linear effects.

**Conclusion:** Using early admission data, we developed and externally validated a 90-day readmission prediction model for older adults hospitalized with AECOPD. XGBoost showed stable performance and clinically interpretable risk patterns, supporting its potential for early risk stratification.

**Keywords:** acute exacerbation of chronic obstructive pulmonary disease, 90-day readmission, extreme gradient boosting, external validation, shapley additive explanations, multicohort study

## Introduction

Chronic obstructive pulmonary disease (COPD) remains one of the leading chronic respiratory diseases worldwide and continues to exert a heavy, long-term burden on public health and healthcare resources. The Global Burden of Disease (GBD) study estimated that approximately 212 million people were living with COPD in 2019, with the overall burden still increasing.<sup>1</sup> COPD demonstrates a strong age-related pattern: as individuals grow older, airway remodeling,



immunosenescence, and accumulating comorbidities make the condition both more prevalent and more challenging to manage. A systematic review suggests that COPD prevalence among adults aged  $\geq 65$  years is roughly 15%, far exceeding that seen in younger populations.<sup>2</sup> As populations age, the number of older adults with COPD is expected to rise further, and their higher comorbidity burden and reduced physiological reserve are likely to translate into greater hospitalization risk and healthcare utilization.<sup>1,2</sup>

Across the course of COPD, acute exacerbations (AECOPD) are key turning points. They can trigger sudden worsening of symptoms, drive emergency department visits and hospitalizations, and often make longer-term disease control more difficult.<sup>3</sup> Short-term readmission after hospitalization is among the most frequent and challenging problems in AECOPD management and is commonly regarded as a clinical signal of inadequate disease control and heightened patient vulnerability. Prior systematic reviews suggest that 30–90-day readmission following hospitalization for COPD exacerbation is not uncommon; although substantial heterogeneity exists across regions and studies, the overall readmission level remains high.<sup>4</sup> In older adults ( $\geq 65$  years), diminished physiological reserve, greater comorbidity burden, and more pronounced inflammatory/coagulation stress responses may delay recovery and increase the likelihood of recurrent decompensation after discharge, thereby elevating short-term readmission risk.<sup>5</sup> Therefore, developing a risk assessment tool that can be applied early in the hospitalization course—so as to identify patients at high risk of readmission within 90 days and to inform post-discharge management decisions—has clear clinical value.<sup>4</sup>

Inflammation is central to the onset and progression of COPD as well as to acute exacerbations, and it is closely associated with adverse outcomes.<sup>6</sup> During AECOPD, airway and systemic inflammation may worsen simultaneously, impairing gas exchange and exercise tolerance and hindering overall recovery, which may in turn increase the risk of short-term adverse events after discharge.<sup>6</sup> In practice, single inflammatory markers can be sensitive to sampling time and short-term fluctuations. Consequently, composite inflammatory indices derived from routine blood cell counts have attracted increasing attention in recent years, including the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and the systemic immune-inflammation index (SII).<sup>7–9</sup> These measures are readily available, low-cost, and obtainable early after admission, offering practical biological signals for early risk stratification in older patients hospitalized with AECOPD.<sup>7,9</sup>

As electronic health records and other clinical datasets have expanded, machine learning (ML) has become a common approach for building readmission risk models. Compared with conventional regression, ML can more readily combine many predictors and accommodate non-linear patterns and interactions, which may enhance the accuracy of patient-level risk stratification.<sup>10–12</sup> However, existing ML studies on COPD/AECOPD readmission still face barriers to clinical translation, including limited external validation, reliance on in-hospital course or post-discharge variables that hinder early implementation at admission, and insufficient interpretability of key risk drivers.<sup>12–14</sup>

These limitations reflect broader methodological concerns in the field. Recent systematic reviews have categorized artificial intelligence applications in COPD into three major modalities: imaging-based diagnosis, physiological signal monitoring where respiratory sound analysis has emerged as particularly effective, and multi-modal fusion frameworks that integrate clinical, imaging, and genomic data.<sup>15</sup> However, the task of short-term prognostic stratification after acute exacerbation remains comparatively underexplored. A recent meta-analysis of 71 prediction models found that 57.9% exhibited high risk of bias and none had undergone formal clinical utility evaluation.<sup>16</sup> Furthermore, while candidate predictors for AECOPD have expanded considerably, promising laboratory indicators such as the neutrophil-to-lymphocyte ratio and D-dimer have yet to be incorporated into a rigorously externally validated prediction framework.<sup>17</sup> This gap between the expanding predictor landscape and the scarcity of valid, clinically tested tools highlights the need for models designed from the outset for early bedside deployment and rigorous external evaluation.

Against this background, the present study focuses on hospitalized older adults ( $\geq 65$  years) with AECOPD. By integrating routinely available early-admission clinical information with inflammation-related indicators, we will compare multiple models, conduct external validation, and perform interpretability analyses. Our aim is to develop a generalizable, explainable, and clinically practical tool for predicting 90-day readmission risk.

## Methods

### Ethics Statement

This retrospective study used a development cohort from the Affiliated Hospital of North Sichuan Medical College and received approval from the hospital's Ethics Committee (Approval No. 2025ER707-1). Given the retrospective nature of the study and the use of de-identified data, the requirement for informed consent was waived by the hospital in accordance with local ethical oversight requirements and institutional policies. In line with these requirements and policies, the Dazhou Hospital of Integrated Traditional Chinese and Western Medicine (external validation cohort) conducted only secondary analyses of previously collected, de-identified clinical data. As the study met the criteria for an ethics exemption, no additional ethical review was required. All data were anonymized before statistical analysis. The study was conducted in accordance with the Declaration of Helsinki and applicable local laws and regulations.

### Study Population

This retrospective, multicenter study enrolled patients aged  $\geq 65$  years with COPD who were hospitalized in the Department of Respiratory and Critical Care Medicine at the Affiliated Hospital of North Sichuan Medical College between January 2023 and June 2024, as well as patients admitted to Dazhou Hospital of Integrated Traditional Chinese and Western Medicine between January and June 2024. COPD was defined as a post-bronchodilator FEV<sub>1</sub>/FVC ratio  $< 0.70$  confirmed by spirometry, or a previously documented diagnosis recorded in the medical chart.<sup>18</sup> Acute exacerbation was characterized by worsening respiratory symptoms requiring additional pharmacological treatment beyond the patient's regular maintenance therapy.

Exclusion criteria were: (1) coexisting asthma, active pulmonary tuberculosis, or any malignancy; (2) hematologic diseases (eg., acute myeloid leukemia); (3) autoimmune disorders; (4) other acute illnesses (eg., acute myocardial infarction); (5) severe psychiatric or psychological conditions that could affect data reliability or outcome ascertainment; (6) in-hospital death; and (7) missing key outcome data or essential covariates that could not be reasonably imputed.

### Outcome Definition and Ascertainment

The primary outcome was readmission for AECOPD within 90 days of discharge from the index hospitalization. A readmission was counted when a new AECOPD-related inpatient stay was registered during the 90-day period after the index discharge date. Events were identified by querying and confirming inpatient records in each center's hospital information system (HIS) and/or electronic medical record (EMR). Both centers followed the same extraction rules and used the same method to define the 90-day window to keep outcome ascertainment consistent. Because only in-house information systems were available, admissions to other hospitals could not be captured.

### Data Collection and Processing

Candidate predictors were drawn from information routinely available at admission: (1) demographics (age, sex, smoking history, and body mass index (BMI)); (2) comorbidities (hypertension, cardiovascular disease, and diabetes); (3) laboratory tests (complete blood count with differential, platelet count, hemoglobin (Hb), red cell distribution width (RDW), coagulation markers including D-dimer, and biochemistry such as albumin and creatinine); (4) respiratory failure (RF), defined as respiratory failure indicated by arterial blood gas analysis at admission; and (5) frequent exacerbations (FE), defined as  $\geq 2$  hospitalizations for acute exacerbations in the past year. To improve bedside usability and minimize information leakage, we restricted predictors to early admission data. Laboratory values were taken from the first results obtained within 24 hours of admission; if multiple measurements were available in that period, the earliest was used. Comorbidity data were derived from prior medical history and discharge diagnoses, and were cross-checked against supporting examinations when available.

Data from the development cohort and the external validation cohort were cleaned and preprocessed separately. Variable derivation was performed independently within each cohort using the same prespecified definitions to ensure consistency in variable meaning. For categorical variables, factor levels were harmonized across the two cohorts. To reduce multicollinearity introduced by component variables and to improve interpretability, inflammatory composite

indices were calculated and included when the relevant blood cell counts were available: the neutrophil-to-lymphocyte ratio (NLR; absolute neutrophil count/absolute lymphocyte count), the platelet-to-lymphocyte ratio (PLR; platelet count/absolute lymphocyte count), and the systemic immune-inflammation index (SII; platelet count  $\times$  absolute neutrophil count / absolute lymphocyte count).<sup>7–9</sup> In subsequent modeling, the component variables—absolute neutrophil count, absolute lymphocyte count, and platelet count—were excluded.

Missing data were handled separately in the development cohort and the external validation cohort to preserve each center's missingness patterns as much as possible and to avoid bias that could arise from cross-center imputation. To enhance the model's usability in multicenter external validation and to limit uncertainty introduced by variables with substantial missingness, we prespecified an inclusion rule before modeling: any variable with a missing rate  $>30\%$  in either cohort was excluded from the candidate predictor set. For the remaining variables, missing values were imputed within each cohort using multiple imputation by chained equations (MICE). Continuous variables were imputed using predictive mean matching, whereas binary variables were imputed using logistic regression.

## Statistical Analysis

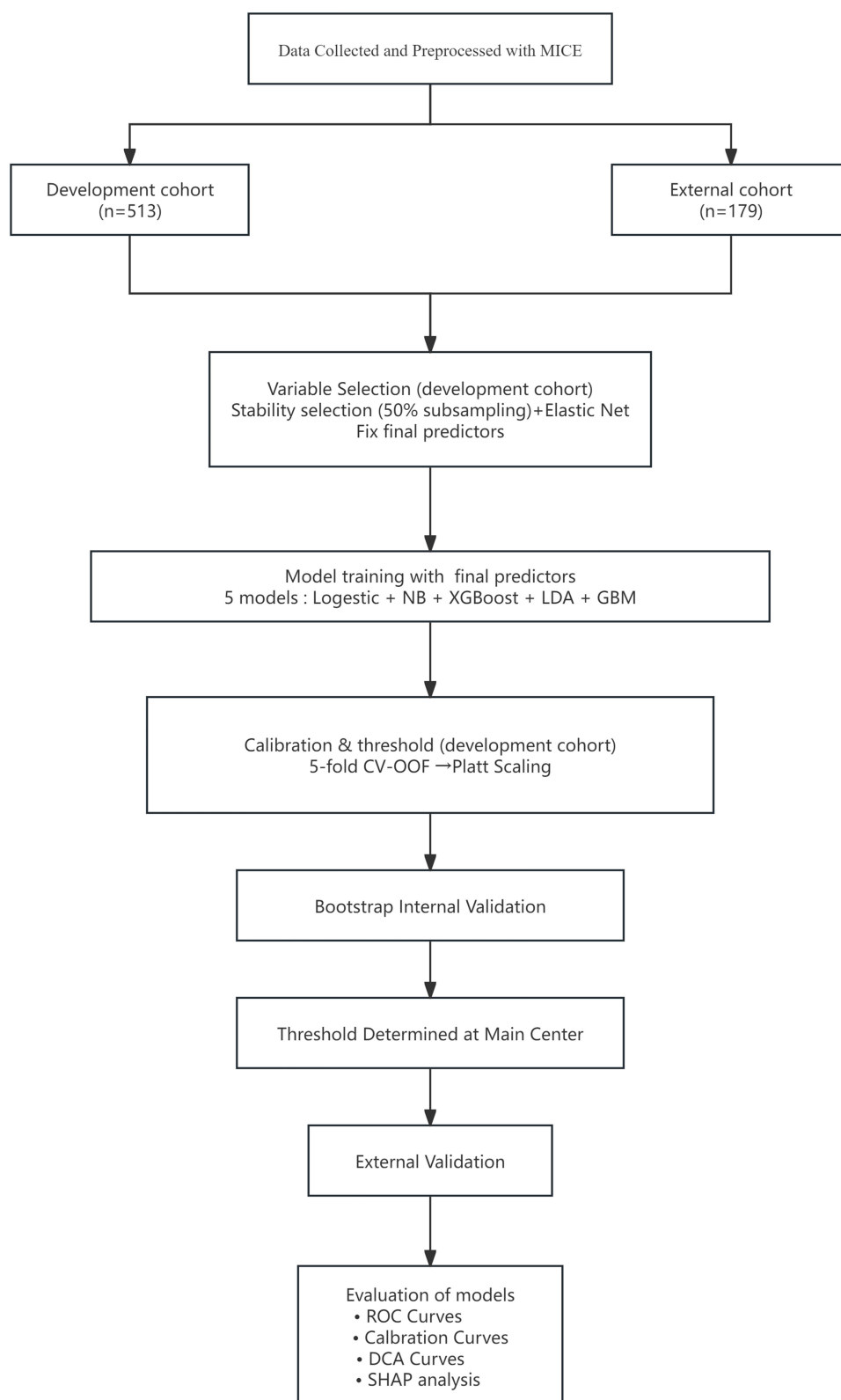
All statistical analyses were conducted using R (version 4.5.1). Data from the Affiliated Hospital of North Sichuan Medical College served as the development cohort, and data from Dazhou Hospital of Integrated Traditional Chinese and Western Medicine served as the external validation cohort. Baseline characteristics were summarized separately for the development and validation cohorts. Continuous variables are reported as mean  $\pm$  standard deviation, and categorical variables as number (percentage). The baseline table was intended to describe differences in case mix between cohorts and was not used for hypothesis testing, feature selection, or model-development decisions. Candidate predictors entered into modeling were based on the results of the data-processing stage (cleaning, harmonized coding, and variable construction). Variable definitions and factor levels for categorical variables were kept consistent across cohorts to ensure comparability between model development and external validation. The overall study workflow and the modeling–calibration–validation framework are shown in [Figure 1](#).

To mitigate multicollinearity and improve model stability, collinearity diagnostics were performed in the development cohort before model building. Variance inflation factors (VIFs) were calculated from a design matrix that included one-hot encoding for categorical variables; the maximum and mean VIF were summarized at the variable level. In addition, the condition number ( $\kappa$ ) of the standardized design matrix was computed, and pairwise correlation coefficients were examined to identify highly correlated terms ( $|r| \geq 0.9$  indicating strong correlation).<sup>19</sup> When substantial collinearity was suspected, variables were handled based on clinical relevance and data completeness to support robust downstream model fitting.

Next, predictor selection in the development cohort was performed using stability selection combined with Elastic Net. Under outcome stratification, 50% subsamples were randomly drawn, repeated 500 times. Within each subsample, an Elastic Net logistic regression model ( $\alpha = 0.5$ ) was fitted, and the penalty parameter was chosen by 10-fold cross-validation using  $\lambda_{1se}$ . Predictor stability was quantified as the proportion of resamples in which a variable was selected.<sup>20</sup> A prespecified selection-frequency threshold of  $\geq 0.90$  was applied, yielding a fixed set of predictors for subsequent model development.

The five candidate models were selected to represent both conventional statistical classifiers and machine-learning algorithms with different assumptions. Logistic regression was included as a clinically interpretable benchmark model. Naïve Bayes and linear discriminant analysis were included as simple probabilistic and linear classification methods, respectively, whereas GBM and XGBoost were selected to capture potential non-linear associations and feature interactions. Elastic Net was used for feature selection because it combines L1 and L2 penalties and is suitable for clinical datasets with potentially correlated predictors. The Elastic Net mixing parameter was set at  $\alpha = 0.5$  to balance variable sparsity and coefficient shrinkage, and the penalty parameter  $\lambda$  was determined by 10-fold cross-validation using the  $\lambda_{1se}$  criterion. Stability selection was performed with 500 repeated stratified 50% subsamples, and predictors with a selection frequency  $\geq 0.90$  were retained.

For tree-based models, hyperparameters were tuned in the development cohort using stratified cross-validation. For XGBoost, the tuned parameters included the number of boosting rounds, maximum tree depth, learning rate, minimum



**Figure 1** Study workflow. After preprocessing and multiple imputation, patients were divided into a development cohort (n=513) and an external validation cohort (n=179). Predictors were selected using stability selection with Elastic Net. Five models were developed, internally validated, and externally tested. Performance was evaluated by ROC, calibration, decision curve analysis, and SHAP.

child weight, subsampling ratio, and column subsampling ratio. For GBM, the tuned parameters included the number of trees, interaction depth, shrinkage, and minimum observations per terminal node. Early stopping or optimal tree-number selection was applied to reduce overfitting. The final hyperparameters were fixed after model development and were not re-estimated in the external validation cohort.

After the final predictor set was established, five types of predictive models were developed and compared in the development cohort: multivariable logistic regression, naïve Bayes, extreme gradient boosting (XGBoost), linear discriminant analysis (LDA), and gradient boosting machine (GBM). For XGBoost and GBM, models were trained on the one-hot-encoded design matrix, with complexity controlled through cross-validation and early stopping or optimal tree-number selection to reduce overfitting.<sup>21</sup> To obtain prediction probabilities that better reflect expected generalization performance and to support consistent calibration, we used 5-fold stratified cross-validation to generate out-of-fold (OOF) predicted probabilities for each model. Platt scaling (estimating an intercept and slope) was then applied to produce calibrated probabilities. Calibration parameters were estimated in the development cohort, fixed thereafter, and applied unchanged in the external validation cohort.<sup>22</sup>

Discrimination was assessed using the area under the receiver operating characteristic curve (AUC), alongside accuracy, sensitivity, specificity, and the Brier score. The classification threshold was determined in the development cohort based on calibrated OOF probabilities and was then carried forward to the external validation cohort. Internal validation was performed using stratified bootstrap resampling (500 iterations) to estimate optimism and compute an optimism-corrected AUC. We validated the model's generalizability in the validation cohort using the same metrics, visualizing results with ROC and calibration curves, and assessing clinical net benefit via decision curve analysis. To enhance interpretability, we applied SHAP to the final model, calculating SHAP values for each feature. We summarized feature importance by mean absolute SHAP values and used SHAP summary plots, heatmaps, and dependence plots to analyze feature contributions, individual patterns, and interactions, clarifying model behavior and identifying key risk drivers.

## Results

### Baseline Characteristics

**Table 1** summarizes the baseline characteristics of the two cohorts. A total of 692 patients were included in this study, comprising 513 in the development cohort and 179 in the external validation cohort. The two cohorts were broadly comparable in age and sex distribution (age:  $76.1 \pm 6.31$  vs.  $76.3 \pm 4.98$  years; female: 34.7% vs. 34.1%). Patients in the

**Table 1** Demographic and Clinical Characteristics of the Cohort

Characteristics	All N=692	Development Cohort N=513	External Cohort N=179
Age	76.1 (5.99)	76.1 (6.31)	76.3 (4.98)
Gender:			
Female	239 (34.5%)	178 (34.7%)	61 (34.1%)
Male	453 (65.5%)	335 (65.3%)	118 (65.9%)
BMI (kg/m <sup>2</sup> )	21.5 (2.76)	21.3 (2.86)	21.9 (2.41)
Smoke:			
No	340 (49.1%)	269 (52.4%)	71 (39.7%)
Yes	352 (50.9%)	244 (47.6%)	108 (60.3%)
NIV:			
No	267 (38.6%)	201 (39.2%)	66 (36.9%)
Yes	425 (61.4%)	312 (60.8%)	113 (63.1%)
RF:			
No	332 (48.0%)	253 (49.3%)	79 (44.1%)
Yes	360 (52.0%)	260 (50.7%)	100 (55.9%)

(Continued)

Table 1 (Continued).

Characteristics	All N=692	Development Cohort N=513	External Cohort N=179
Readmission:			
No	542 (78.3%)	407 (79.3%)	135 (75.4%)
Yes	150 (21.7%)	106 (20.7%)	44 (24.6%)
FE:			
No	460 (66.5%)	362 (70.6%)	98 (54.7%)
Yes	232 (33.5%)	151 (29.4%)	81 (45.3%)
Hypertension:			
No	475 (68.6%)	354 (69.0%)	121 (67.6%)
Yes	217 (31.4%)	159 (31.0%)	58 (32.4%)
CVD:			
No	351 (50.7%)	254 (49.5%)	97 (54.2%)
Yes	341 (49.3%)	259 (50.5%)	82 (45.8%)
Diabetes:			
No	576 (83.2%)	427 (83.2%)	149 (83.2%)
Yes	116 (16.8%)	86 (16.8%)	30 (16.8%)
PaO <sub>2</sub> (mmHg)	85.2 (27.0)	86.0 (24.5)	82.9 (33.2)
PaCO <sub>2</sub> (mmHg)	51.5 (13.8)	51.3 (14.1)	52.4 (13.1)
D (Ug/mL)	2.13 (2.37)	2.04 (2.22)	2.37 (2.75)
TT (s)	17.9 (1.53)	17.8 (1.56)	18.0 (1.44)
APTT (s)	36.2 (7.20)	35.9 (6.27)	36.9 (9.35)
PTA (%)	80.3 (19.9)	80.7 (21.3)	78.9 (15.2)
BNP (pg/mL)	2437 (3008)	2321 (3052)	2770 (2857)
ALB (g/L)	37.2 (3.88)	37.3 (3.77)	37.0 (4.19)
Creatinine (umol/L)	75.4 (24.3)	73.3 (22.7)	81.3 (27.6)
WBC (10E9/L)	7.95 (3.24)	7.89 (3.19)	8.13 (3.39)
RBC (10E12/L)	4.38 (0.67)	4.38 (0.69)	4.40 (0.63)
Hb (g/L)	129 (20.3)	130 (20.1)	129 (20.7)
HCT (%)	0.41 (0.07)	0.42 (0.07)	0.41 (0.06)
RDW (%)	14.7 (1.37)	14.7 (1.43)	14.6 (1.20)
MPV (fl)	10.5 (1.55)	10.5 (1.40)	10.4 (1.93)
Monocyte (10E9/L)	0.51 (0.24)	0.51 (0.25)	0.52 (0.24)
Eosinophils (10E9/L)	0.09 (0.11)	0.09 (0.11)	0.10 (0.12)
Basophil (10E9/L)	0.02 (0.02)	0.02 (0.02)	0.02 (0.02)
NLR	8.61 (7.53)	8.82 (7.87)	8.02 (6.45)
PLR	238 (166)	244 (169)	220 (157)
SII	1518 (1461)	1555 (1497)	1412 (1348)

**Notes:** Data are presented as mean (SD) for continuous variables and n (%) for categorical variables. Bold text indicates column headings and subgroup labels only; it does not denote statistical significance.

**Abbreviations:** BMI, body mass index; NIV, non-invasive ventilation; RF, respiratory failure; readmission, 90-day readmission; FE, frequent exacerbations; hypertension, hypertension; CVD, cardiovascular disease; PaO<sub>2</sub>, arterial partial pressure of oxygen; PaCO<sub>2</sub>, arterial partial pressure of carbon dioxide; D, D-dimer; TT, thrombin time; APTT, activated partial thromboplastin time; PTA, prothrombin activity; BNP, B-type natriuretic peptide; ALB, albumin; WBC, white blood cell count; RBC, red blood cell count; Hb, hemoglobin; HCT, hematocrit; RDW, red cell distribution width; MPV, mean platelet volume; NLR, neutrophil-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio; SII, systemic immune-inflammation index.

external validation cohort had a slightly higher BMI ( $21.9 \pm 2.41$  vs.  $21.3 \pm 2.86$  kg/m<sup>2</sup>), a higher proportion of smokers (60.3% vs. 47.6%), and a higher prevalence of frequent exacerbations (FE) (45.3% vs. 29.4%). The 90-day readmission rate was also modestly higher in the external validation cohort (24.6% vs. 20.7%). The distribution of comorbidities was largely similar between cohorts (hypertension: 32.4% vs. 31.0%; cardiovascular disease: 45.8% vs. 50.5%; diabetes: 16.8% vs. 16.8%). Likewise, the proportions of non-invasive ventilation (NIV) use and respiratory failure (RF) were

comparable (NIV: 63.1% vs. 60.8%; RF: 55.9% vs. 50.7%). Laboratory and arterial blood gas measures were generally consistent across cohorts, although the external validation cohort showed slightly lower PaO<sub>2</sub> and higher PaCO<sub>2</sub>, along with modestly higher D-dimer and creatinine levels. The composite inflammatory indices (NLR, PLR, and SII) were overall similar between the two cohorts.

## Variable Screening

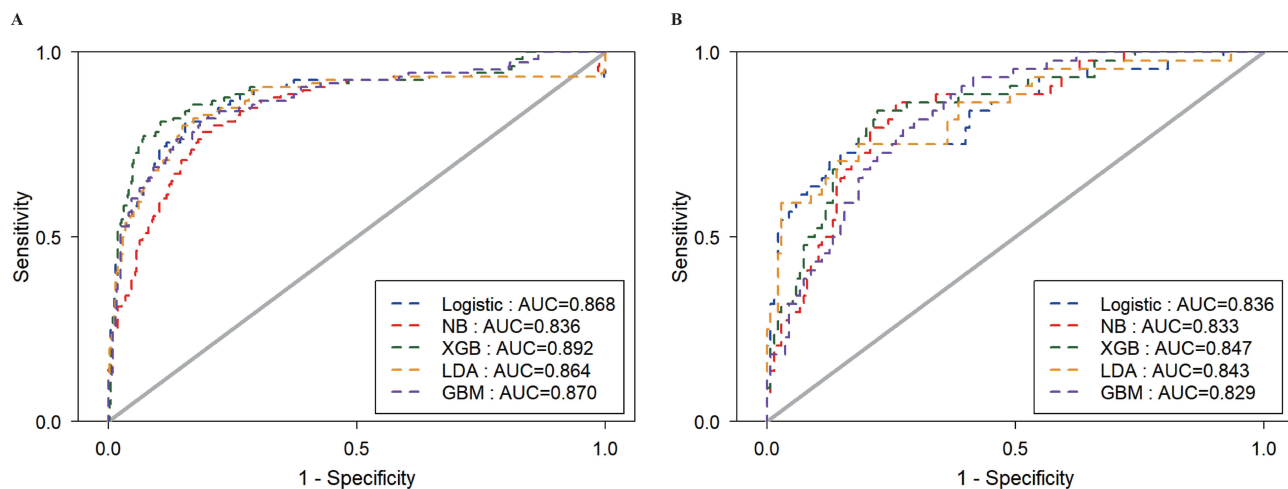
In the development cohort, predictor selection was performed using stability selection (stratified subsampling) combined with Elastic Net. This process resulted in a fixed set of six predictors: NLR, SII, D, FE, BMI, and ALB. After the predictor set was finalized, we further examined multicollinearity. The absolute values of pairwise correlation coefficients among all continuous variables were below 0.90, and variance inflation factors (VIFs) for all predictors in the multi-variable logistic regression framework were <5. These findings indicate no meaningful multicollinearity, and all six predictors were therefore retained for subsequent model development and validation.

## Model Performance Comparison

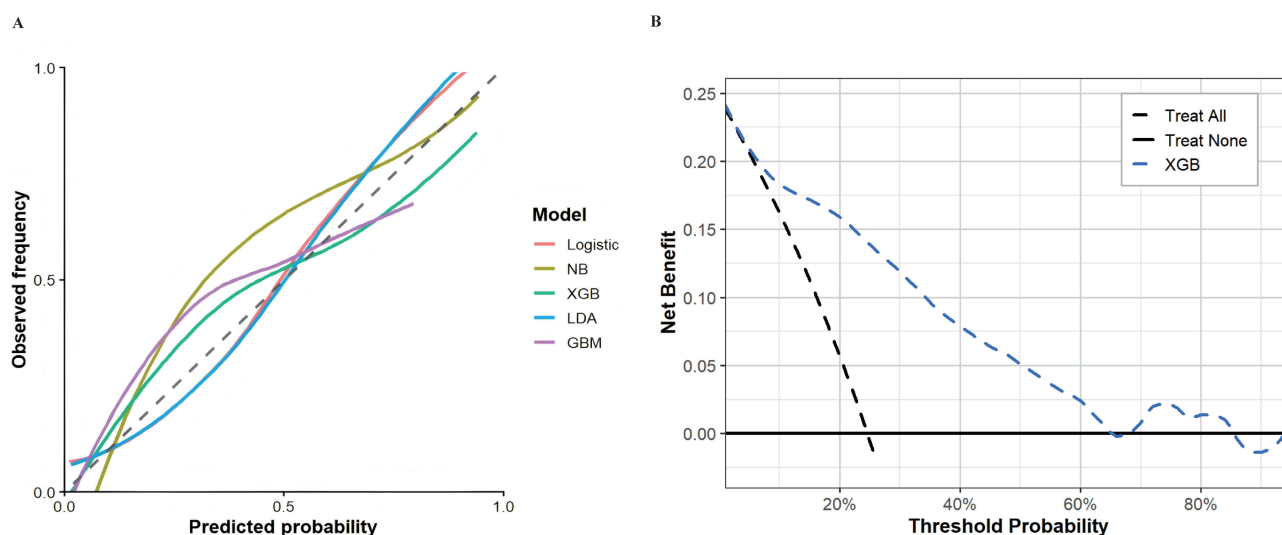
After fixing the final predictor set and completing probability calibration, we compared the discriminative performance, internal robustness, and external transportability of five models in both the development and external validation cohorts (Figures 2 and 3; Table 2). In the development cohort, the ROC curves of all five models lay clearly above the diagonal reference line (Figure 2A), indicating good overall discrimination. Among them, XGBoost achieved the best performance (AUC = 0.892). GBM, logistic regression, and LDA showed similar discrimination (AUC approximately 0.86–0.87), whereas naïve Bayes yielded a comparatively lower AUC (0.836). In addition to discrimination, we assessed overall probabilistic error using the Brier score. The results suggested meaningful differences across models, with XGBoost showing a lower overall prediction error and a more balanced performance profile (Table 2).

To evaluate overfitting and internal reproducibility, we used stratified bootstrap resampling for internal validation and calculated optimism-corrected AUCs. The corrected AUCs closely matched the apparent AUCs, indicating minimal overfitting. XGB was the best performer (corrected AUC = 0.864), followed by logistic regression and LDA, with NB and GBM having lower values.

In the external validation cohort, all models showed acceptable AUC values, demonstrating good generalizability (Figure 2B). XGB had the highest AUC at 0.847, followed by LDA and logistic regression with AUCs of 0.843 and 0.836, respectively. NB and GBM had slightly lower AUCs at 0.833 and 0.829. While AUCs were slightly lower than in the development cohort, the models' relative rankings remained consistent, indicating stable performance.



**Figure 2** Receiver operating characteristic (ROC) curves of the five prediction models. (A) Development cohort. (B) External validation cohort. The diagonal dashed line indicates no discrimination. XGBoost achieved the highest area under the curve (AUC) in both cohorts.



**Figure 3** Calibration and clinical utility in the external validation cohort. **(A)** Calibration curves for logistic regression, naive Bayes, XGBoost, linear discriminant analysis, and gradient boosting machine; the dashed line indicates ideal calibration. **(B)** Decision curve analysis of the XGBoost model compared with the treat-all and treat-none strategies.

In summary, the calibration plots indicated that all models demonstrated satisfactory concordance between predicted and observed risks within the external cohort (Figure 3A). Regarding clinical utility, decision curve analysis revealed that the XGB model offered a greater net benefit compared to the “treat-all” and “treat-none” strategies across various threshold probabilities in the external cohort (Figure 3B), underscoring its potential clinical applicability. Consequently, we selected the XGB model as the primary model for subsequent interpretability analyses based on SHAP values.

## Model Interpretation

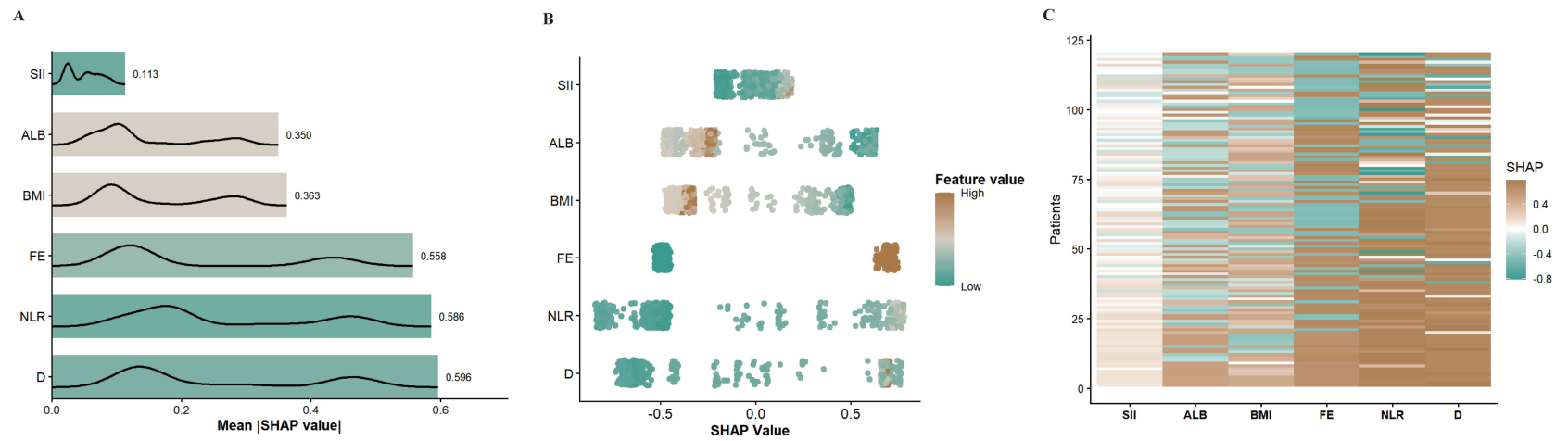
After identifying XGB as the primary model, we used SHAP to clarify how each predictor contributed to the model output and to examine possible non-linear patterns (Figures 4 and 5). In the global importance ranking, all six predictors

**Table 2** Performance of Five Models in Development and External Cohorts

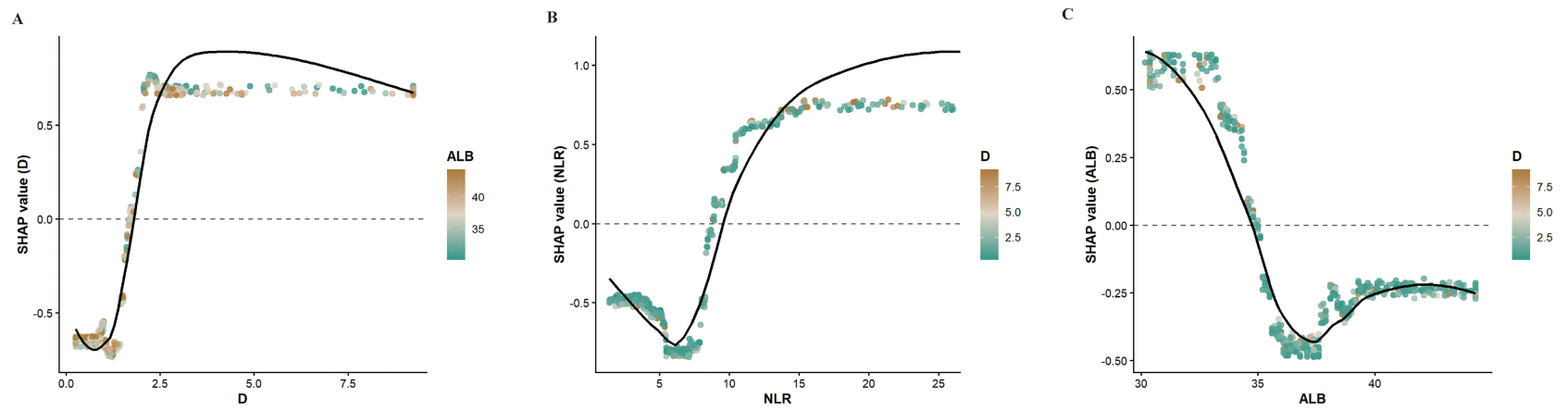
Model	AUC	95% CI	Accuracy	Sensitivity	Specificity	Brier Score
<b>Development cohort</b>						
LR	0.868	(0.818, 0.917)	0.864	0.670	0.914	0.096
NB	0.836	(0.785, 0.886)	0.797	0.783	0.801	0.119
XGB	0.892	(0.850, 0.934)	0.901	0.755	0.939	0.086
LDA	0.864	(0.814, 0.914)	0.865	0.679	0.914	0.098
GBM	0.870	(0.828, 0.913)	0.850	0.698	0.890	0.100
<b>External cohort</b>						
LR	0.836	(0.760, 0.912)	0.827	0.705	0.867	0.117
NB	0.833	(0.767, 0.900)	0.765	0.795	0.756	0.142
XGB	0.847	(0.781, 0.912)	0.804	0.591	0.874	0.131
LDA	0.843	(0.770, 0.915)	0.816	0.682	0.859	0.118
GBM	0.829	(0.767, 0.891)	0.777	0.477	0.874	0.147

**Notes:** No bold formatting is used to denote statistical significance or model performance.

**Abbreviations:** AUC, area under the curve; CI, confidence interval; LR, logistic regression; NB, naive Bayes; XGB, extreme gradient boosting; LDA, linear discriminant analysis; GBM, gradient boosting machine.



**Figure 4** SHAP-based interpretation of the XGBoost model. **(A)** Global feature importance ranked by mean absolute SHAP value. **(B)** SHAP beeswarm plot showing the direction and magnitude of feature effects across patients. **(C)** SHAP heatmap showing patient-level contribution patterns. D-dimer, neutrophil-to-lymphocyte ratio, and frequent exacerbations were the main contributors.



**Figure 5** SHAP dependence plots for key predictors in the XGBoost model. **(A)** D-dimer, colored by albumin. **(B)** Neutrophil-to-lymphocyte ratio, colored by D-dimer. **(C)** Albumin, colored by D-dimer. Positive SHAP values indicate higher predicted risk of 90-day readmission, and negative values indicate lower predicted risk.

contributed consistently, with D-dimer, NLR, and frequent exacerbations (FE) showing the highest mean absolute SHAP values, indicating that they were the main drivers of prediction. BMI and ALB also had notable influence, whereas SII contributed less overall (Figure 4A).

The SHAP beeswarm plot further showed clear between-patient variation in how features affected risk. In general, higher D-dimer and NLR values were more likely to push SHAP values upward and increase predicted risk, while lower values tended to pull risk downward. FE (yes) was associated with a positive contribution in most patients, consistent with a higher readmission risk among those with frequent prior exacerbations. For ALB and BMI, the direction of contribution changed across value ranges, suggesting non-linear effects (Figure 4B). Patient-level SHAP heatmaps also indicated that contribution patterns were not uniform across individuals, supporting patient-specific explanation pathways for risk stratification (Figure 4C).

To explore non-linear effects and interactions among key variables, we created SHAP dependence plots (Figure 5). D-dimer showed a threshold effect: its risk contribution was negative or near zero at low levels but increased rapidly before plateauing as levels rose, with variations potentially influenced by ALB levels (Figure 5A). NLR also had a non-linear association, with minimal or negative impact at low values, a sharp increase beyond a certain point, and stabilization at higher levels, possibly interacting with D-dimer (Figure 5B). ALB generally had an inverse relationship with SHAP values, where lower ALB increased risk contribution and higher ALB reduced it, suggesting higher albumin might lower readmission risk, with this pattern possibly affected by D-dimer levels (Figure 5C).

In summary, the SHAP analysis reveals that the predictions generated by the XGB model are collectively influenced by markers related to inflammation, history of prior exacerbations, and indicators of coagulation and nutrition. Furthermore, the findings underscore the presence of non-linear effects and potential interactions among principal predictors, thereby offering a comprehensible foundation for clinical interpretation and risk stratification.

## Discussion

We developed and externally validated a 90-day readmission risk prediction model using two hospital cohorts of patients aged  $\geq 65$  years who were admitted for AECOPD. Predictor selection was carried out with stability selection (stratified subsampling) combined with Elastic Net, yielding a fixed set of six key variables (NLR, SII, D-dimer, FE, BMI, and ALB). We then compared the performance of five modeling approaches: logistic regression, naïve Bayes, LDA, GBM, and XGBoost. Overall, XGBoost delivered the best performance in the development cohort, remained robust after bootstrap internal validation, and showed acceptable discrimination and calibration in the external cohort. It also demonstrated lower overall prediction error (Brier score) and potential clinical utility. SHAP analyses further indicated that model predictions were mainly driven by D-dimer, NLR, and FE, with evidence of non-linear effects and possible interactions. Together, these findings support the use of the model for early, admission-stage identification of patients at high risk of 90-day readmission.

Building on the interpretability findings, we propose that the three leading predictors map onto three key pathophysiological dimensions underlying 90-day readmission risk in older patients with AECOPD: inflammatory burden (NLR), an exacerbation-prone phenotype (FE), and coagulation/endothelial stress related to injury (D-dimer).

First, NLR, derived from routine complete blood counts, reflects both an intensified neutrophil-driven pro-inflammatory response and the immunoregulatory suppression suggested by lymphopenia. As such, it is often regarded as a simple and practical biomarker capturing the balance between inflammatory burden and immune status.<sup>23,24</sup> In the context of AECOPD, an elevated NLR typically corresponds to more pronounced systemic inflammatory activation and a less favorable acute clinical course, and has been linked to a higher risk of short-term adverse outcomes. This provides a physiologically plausible explanation for delayed post-discharge recovery and a greater likelihood of subsequent decompensation.<sup>24,25</sup> Moreover, in cohorts of older adults ( $\geq 65$  years) hospitalized with AECOPD, NLR has shown a positive association with 90-day readmission risk (multivariable OR  $\approx 1.57$ ; reaching  $\approx 1.75$  in higher-risk subgroups). Systematic reviews have likewise reported a markedly increased readmission risk when NLR exceeds 7 (pooled OR  $\approx 1.93$ ).<sup>26,27</sup> These findings align with the dominant risk-stratifying direction of NLR observed in our model.

Second, FE reflects a relatively stable frequent-exacerbator phenotype, indicating poorer long-term disease control and greater baseline vulnerability. This phenotype likely arises from multiple, overlapping factors, including persistent

airway inflammation and structural remodeling, bacterial colonization and a propensity for recurrent infections, a heavier burden of comorbidities with reduced functional reserve, and differences in adherence and self-management capacity after discharge. Accordingly, the consistently positive risk contribution of FE observed in our model is clinically plausible.<sup>28–30</sup> Prior work also supports a clear “dose–response” pattern in effect size: patients with  $\geq 2$  hospitalizations for AECOPD in the previous year have a markedly higher risk of subsequent readmission (crude OR  $\approx 4.10$ ), and the risk increases further among those with  $\geq 3$  hospitalizations (crude OR  $\approx 7.51$ ).<sup>31</sup> These findings suggest that a history of exacerbation-related admissions is among the most informative clinical markers for predicting short-term readmission.

Finally, D-dimer, a marker of increased fibrin formation and breakdown, may reflect joint activation of the inflammation–endothelial activation–coagulation axis during AECOPD.<sup>32,33</sup> Prior studies have reported higher coagulation/fibrinolysis markers during exacerbations, supporting D-dimer as a signal of systemic stress and possible micro-circulatory impairment.<sup>32,33</sup> Most evidence links D-dimer to acute severity or longer-term outcomes; for example, Hu et al found that higher D-dimer was associated with increased in-hospital events and 1-year adverse outcomes.<sup>34</sup> In our study, we interpret D-dimer as a measurable indicator of acute systemic stress and disease-severity burden: elevated levels may imply slower recovery and greater susceptibility to post-discharge decompensation, aligning with its SHAP-derived risk contribution to 90-day readmission.

It should be emphasized that the incremental AUC improvement of XGBoost over logistic regression was modest, especially in the external validation cohort. Therefore, the value of XGBoost in this study should not be interpreted solely as a large gain in discrimination. Rather, its selection as the primary model was based on a more comprehensive performance profile, including discrimination, calibration, Brier score, decision-curve-derived net benefit, and interpretability. The relatively similar AUCs across models also suggest that part of the predictive information was captured by a small set of clinically meaningful variables, rather than by algorithmic complexity alone. This finding supports the robustness of the selected predictor panel and indicates that the model may be practically usable in clinical settings. The potential clinical application of this model lies in early risk stratification during hospitalization. Because all predictors were derived from routinely available admission data, the model could be embedded into the hospital information system or electronic medical record to automatically estimate 90-day readmission risk after the first laboratory results are available. Patients classified as high risk could be considered for intensified discharge planning, medication and inhaler-technique optimization, nutritional and rehabilitation assessment, earlier outpatient follow-up, and closer post-discharge monitoring. Nevertheless, the model should be used as a clinical decision-support tool rather than a substitute for physician judgment. Future prospective implementation studies are needed to determine whether model-guided management can reduce readmission rates and improve patient outcomes.

Compared with prior work, the practical value of this study lies in its closer alignment with the real-world need for short-term post-discharge risk control in older patients hospitalized for AECOPD. Many existing scoring systems and prediction studies in COPD/AECOPD focus on in-hospital severe outcomes (eg., in-hospital mortality, need for invasive ventilation, or ICU care) or on longer-term endpoints. For example, Zhang et al developed an explainable machine-learning model to predict hospitalization death in hospitalized patients with AECOPD.<sup>35</sup> In contrast, our study targets 90-day readmission after discharge in older ( $\geq 65$  years) patients and emphasizes routinely available early-admission variables with two-center development and external validation. These targets are useful for assessing acute severity, but they do not fully match the clinical meaning of 90-day readmission, a short-term outcome that more directly reflects inadequate disease control and patient vulnerability after discharge. For example, DECAF is primarily designed for stratifying the risk of in-hospital mortality, whereas BAP-65 places greater emphasis on acute severity and adverse in-hospital outcomes.<sup>36–38</sup> In addition, a systematic review of COPD readmission prediction models and AI-based studies has highlighted recurring limitations—such as reliance on single-center data, insufficient external validation, and substantial methodological heterogeneity—which can undermine model transportability and bedside usability.<sup>16</sup> Against this background, we prespecified 90-day readmission as the prediction target and deliberately restricted predictors to routine information available early after admission, aiming to minimize information leakage while improving clinical deployability.

Several limitations should be acknowledged. First, as a retrospective observational study, selection bias and residual confounding cannot be fully eliminated despite both internal and external validation. Moreover, readmissions were

identified only through rehospitalization records within each hospital's own information system; readmissions occurring at other institutions were not captured, which may underestimate the true readmission rate and limit generalizability. Accordingly, our findings should be interpreted as predictive associations rather than causal effects. Second, because participants were drawn from two hospitals within the same region, broader validation in larger, multicenter prospective cohorts across different areas is still needed. Third, key laboratory measurements were taken from the first test within 24 hours of admission, but differences in laboratory platforms and pre-analytical procedures across centers may introduce measurement variability. Fourth, some post-discharge management factors (eg., adherence, rehabilitation, and access to follow-up) were incompletely documented in retrospective records, potentially limiting the model's coverage of modifiable risks. Finally, SHAP provides contribution-based explanations under the current data distribution and should not be interpreted as effect sizes or evidence of causal mechanisms; prospective impact studies are required before routine clinical implementation.

In summary, we developed and externally validated a 90-day readmission prediction model in two cohorts of hospitalized patients aged  $\geq 65$  years with AECOPD, using routinely available clinical indicators obtained early after admission. The model maintained acceptable discrimination and calibration in the external cohort and showed a low overall prediction error. Interpretability analyses suggested that readmission risk was primarily driven by coagulation- and inflammation-related burden, together with a frequent-exacerbator phenotype. This tool may support early in-hospital risk stratification and guide post-discharge management decisions, although larger, multicenter prospective studies across diverse regions are still needed to confirm its clinical impact.

## Data Sharing Statement

The datasets used and/or analyzed in this study were derived from multiple cohorts and are currently being used for ongoing analyses. Due to institutional policies and ethics-related restrictions, the datasets are not publicly available. De-identified data may be provided by the corresponding author upon reasonable request, subject to appropriate institutional/ethics approvals and applicable regulations.

## Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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## Disclosure

The authors declare no conflicts of interest in this study.

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