International Journal of Chronic Obstructive Pulmonary Disease

Dovepress
Taylor & Francis Group

Open Access Full Text Article

ORIGINAL RESEARCH

An Interpretable AdaBoost Model for 1-Year Readmission Risk Prediction in AECOPD Patients with Hypertension

Xinxin Zhang[1], Maolang He[1], Jingyi Zhang[2], Luna Zhao[1], Dong Liu[1]

[1]Department of Pulmonary and Critical Care Medicine, The First Affiliated Hospital of Shihezi University, Shihezi, People's Republic of China; [2]Shihezi University School of Medicine, Shihezi, People's Republic of China

Correspondence: Dong Liu, Department of Pulmonary and Critical Care Medicine, The First Affiliated Hospital of Shihezi University, No. 107 North second Road, Hongshan Street, Shihezi, 832008, People's Republic of China, Email 2322800100@qq.com

Background: Chronic obstructive pulmonary disease (COPD) complicated by hypertension imposes a substantial global health burden, with acute exacerbation of chronic obstructive pulmonary disease (AECOPD) significantly increasing 1-year readmission risk. This study aimed to develop and validate an interpretable machine learning (ML) model that predicts 1-year readmission risk in AECOPD patients complicated by hypertension using real-world data.

Methods: This retrospective cohort study enrolled 2042 patients with AECOPD complicated by hypertension from the First Affiliated Hospital of Shihezi University between 2015 and 2024. The data were split into training and test sets at a 7:3 ratio. Feature selection was performed based on machine learning methods. Eight ML models were trained and tested to construct predictive models. Model performance was evaluated by the area under the receiver operating characteristic curve (AUC), accuracy, recall, specificity, and F1-score. The Shapley additive explanation method (SHAP) was used to rank the feature importance and explain the final model. An online risk prediction tool was developed based on the optimal model to facilitate clinical application.

Results: The 1-year readmission rate of patients with AECOPD complicated by hypertension was 37.5%. Seven independent predictors, including times of inhospitalization, procalcitonin, total protein, international normalized ratio (INR), prothrombin time, D-dimer, and hypoproteinemia, were identified as the most valuable features for establishing the models. The AdaBoost model showed optimal performance, with an AUC of 0.884 in the test set and an average AUC of 0.889 in 5-fold cross-validation. SHAP analysis confirmed that times of inhospitalization were the strongest predictor, followed by INR and total protein. An online calculator was deployed (https://fast.statsape.com/tool/detail?id=17) for clinical use.

Conclusion: This study developed an interpretable AdaBoost-based online calculator for 1-year readmission risk assessment in AECOPD patients by hypertension. The tool highlight the importance of addressing hypercoagulability and nutritional status to reduce readmission risk. Further external multi-center validation is needed to enhance its generalizability.

Keywords: acute exacerbation of chronic obstructive pulmonary disease, hypertension, 1-year readmission, machine learning, web calculator

Introduction

Chronic obstructive pulmonary disease (COPD) is primarily characterized by persistent and incompletely reversible airflow limitation.[1] COPD is the third leading cause of death globally, with approximately 2.9 million deaths attributed to the disease in 2023. It is projected that by 2050, the number of individuals aged 25 and above suffering from COPD worldwide will increase by 23%, approaching 600 million.[2] Acute exacerbation of chronic obstructive pulmonary disease (AECOPD) is a significant factor affecting the health and prognosis of COPD patients. One-year mortality has been reported as low as 5% for patients without acute exacerbation in 1 year and as high as 43% for patients following an acute exacerbation of severe COPD, posing a substantial economic and social burden on both patients and healthcare systems.[3]

https://doi.org/10.2147/COPD.S557298
Received: 31 July 2025
Accepted: 22 December 2025
Published: 8 January 2026

Cardiovascular disease is the most common comorbidity of COPD, with over 70% of COPD patients also suffering from primary hypertension.[4] In China, the prevalence of hypertension has experienced a significant increase over the past few decades and consistently ranks as the most prevalent chronic disease.[5] COPD is prone to being complicated by hypertension, which may be associated with risk factors such as obesity, smoking, and abnormal blood lipid levels.[6–8] Hypertension amplifies systemic inflammation, accelerates endothelial dysfunction, and increases left-ventricular afterload—all of which can precipitate acute exacerbations and early re-hospitalization. Studies have shown that the incidence of hypertension is higher among COPD patients, and this comorbidity may exacerbate the patient's condition and increase the risk of cardiovascular events.[9] The readmission rate within one year is an important indicator for assessing patient health status and the quality of healthcare services. The disease burden of AECOPD is largely attributable to the neglect of managing modifiable risk factors. Therefore, establishing a practical risk prediction model for the prognosis of AECOPD complicated by hypertension is crucial to enable personalized management and improve patient outcomes.

Recent years have witnessed a growing number of studies predicting readmission risk in COPD patients and developing corresponding prediction tools. Most existing models have focused on 30- or 90-day readmission windows and were built on general COPD populations without adequately accounting for the impact of specific comorbidities. For instance, one retrospective cohort study leveraged electronic health record data to develop a clinical prediction model for 30-day readmissions among AECOPD patients.[10] Another study enrolling 4327 COPD patients established a model for readmission within 90 days following acute exacerbations of COPD.[11] However, Long-term risk predictive model dedicated to AECOPD patients with concurrent hypertension are still lacking. Machine learning (ML) achieves efficient data processing through its unique rapid computation methods and has recently received considerable attention for forecasting patient prognosis.[12] The crucial aspect lies in addressing the intricate nonlinear relationships that exist between predictor variables and outcome metrics to generate more trustworthy predictions.[13,14]

This study leverages real-world data and machine learning algorithms to develop and validate an interpretable web-based calculator specifically designed for predicting 1-year readmission risk in AECOPD patients complicated by hypertension. This study provides evidence-based guidance for optimizing clinical management and improving survival prognosis in individuals with AECOPD complicated by hypertension.

Materials and Methods

Study Population

We conducted a retrospective cohort study at the First Affiliated Hospital of Shihezi University, selecting patients over 40 years old with AECOPD complicated by hypertension who were hospitalized in the Department of Respiratory and Critical Care Medicine from January 2015 to January 2024 as the study subjects. The inclusion criteria were (1) patients admitted with a main diagnosis of AECOPD (AECOPD was diagnosed according to International Classification of Diseases-10 codes J44.1, and hypertension was defined by I10, based on the primary discharge diagnoses documented in the medical records for all hospitalized patients); (2) patients had a history of hypertension; and (3) patients aged ≥ 40 years. We have established the following exclusion criteria: (1) patients with bronchial asthma, bronchiectasis, or interstitial lung disease; (2) patients with Cushing's syndrome, pheochromocytoma, or primary aldosteronism; (3) patients who are admitted to the hospital due to other diseases within one year after discharge; (4) patients with severe complications and malignant tumors; and (5) patients lacking complete clinical information. After screening, a total of 2042 patients with AECOPD complicated by hypertension were included in this study. This study followed the Declaration of Helsinki and was approved by the Ethics Committee of the First Affiliated Hospital of Shihezi University (KJ2024-522-02). Due to its retrospective design, informed consent was not required. Clinical trial number: not applicable.

Data Collection and Preprocessing

This study systematically gathered and documented data from electronic medical records, including demographics and laboratory data. The demographic data encompassed age, gender, body mass index (BMI), temperature, heart rate, diastolic pressure, systolic pressure, smoking and alcohol consumption history, number of hospitalizations, length of hospital stay, and medical histories of hypertension, diabetes, and hypoproteinemia. The hematological parameters included complete blood counts, liver and kidney function tests, glucose and lipid profiles, procalcitonin, erythrocyte sedimentation rate, C-reactive protein (CRP), cardiac enzymes, electrolytes, coagulation profile, and arterial blood gas analysis. Smoking and drinking statuses were documented as "yes" or "no", while information regarding hypertension and diabetes was obtained through self-reports from the participants. Hypoproteinemia was defined as a serum protein level $\leq$ 6 g/dL.[15] From January 2015 to January 2024, this study recorded re-hospitalization within one year due to AECOPD combined with hypertension as the outcome variable. Consequently, patients were stratified into two groups based on 1-year readmission status following hospitalization for AECOPD complicated by hypertension: the readmission group and the non-readmission group.

Feature Selection

Given the complexity associated with multiple risk factors, achieving high diagnostic efficiency through traditional univariate approaches proves challenging. We therefore leveraged machine learning techniques to develop a prediction model for 1-year readmission risk in AECOPD complicated by hypertension. The Boruta algorithm was first employed to identify potentially influential variables on the target outcome. Subsequently, feature importance ranking was performed using ML techniques to refine the initially screened variables. Random forest and AdaBoost algorithms were implemented to determine the top 10 most critical risk features. Final feature variables were established by identifying intersections through Venn diagram analysis. This combined approach, documented in prior clinical prediction studies, serves to improve model generalizability and interpretability.[16]

Model Construction and Evaluation

We developed eight machine learning algorithms, including AdaBoost, decision tree, K-nearest neighbors, logistic regression, naïve Bayes, random forest, support vector machine, XGBoost. Subsequently, the study subjects were randomly divided into training and test sets in a 7:3 ratio. Based on this division, the aforementioned parametric models were fitted and tested. The performance of the models was compared using metrics such as the area under the curve (AUC), accuracy, recall, specificity, and F1 score, ultimately leading to the selection of the optimal predictive model. Detailed formulas and computational methods for all performance metrics are provided in the Supplementary Materials 1. Model robustness was evaluated via 5-fold cross-validation. The data was partitioned into ten subsets, with nine subsets training the model and one testing performance per iteration. Receiver operating characteristic (ROC) analysis is commonly employed to describe the identification accuracy of diagnostic models.[17] Hence, we quantitatively evaluated the model through ROC analysis. Decision curve analysis (DCA) was utilized to measure clinical utility and net benefit, providing significant advantages in assessing the clinical applicability of models.[18] Calibration curves were employed to determine the predictive ability of the models, and a comprehensive evaluation of the predictive models was conducted to verify their usability in decision support.[19] The precision-recall curve quantifies the average precision rate of the model across all possible threshold settings, and this curve is widely used to evaluate model performance.

Model Interpretation

Shapley additive explanation (SHAP) is a prominent machine learning interpretability tool grounded in Cooperative Game Theory, widely recognized for elucidating the predictive outcomes of various machine learning models.[20] The fundamental premise of SHAP is to assign importance values to each feature within the model, thereby clarifying the prediction process.[21] Its strengths are attributed to a robust theoretical foundation, extensive applicability, and the capacity to provide both global insights and localized details for complex models.[22] In this study, after training the machine learning model with the dataset, the SHAP values for each feature were computed using the SHAP library, and subsequently, these values were visualized to facilitate model interpretation.

Statistical Analysis

We conducted a systematic analysis of the collected clinical data using DecisionLinnc 1.0 software. This is an integrated, one-stop automated statistical platform that provides no-code visual programming and node-based task deployment. Variables with missing data exceeding 30% were excluded, as elaborated in Supplementary Table 1. Variables with missing data exceeding 30% were excluded from the analysis, as elaborated in Supplementary Materials. For remaining variables with missing rates below this threshold, multiple imputation was employed to construct a complete dataset. Normality tests were performed before analyzing the continuous variables. For conforming to a normal distribution, we expressed the results as mean ± standard deviation and utilized independent samples *t*-tests to compare differences between the two groups. Conversely, for continuous variables that did not conform to a normal distribution, we represented the data as median and interquartile range and employed the Mann–Whitney *U*-test for group comparisons. Additionally, binary variables were presented in terms of frequency and percentage, with chi-square tests used to evaluate differences between the two groups. We checked for multicollinearity using variance inflation factors (VIF), where a VIF > 10 was considered highly collinear. Spearman correlation analysis is used to analyze the correlation between variables. A statistical significance threshold was set at $P < 0.05$.

Result

Basic Characteristics of the Study Population

The study included data from the database of the First Affiliated Hospital of Shihezi University on AECOPD complicated by hypertension from 2015 to 2024. Initially, a total of 3562 patients were included. After applying the inclusion and exclusion criteria, 2042 patients were enrolled. Of these, 765 (37.5%) experienced readmissions within 1 year, while 1277 (62.5%) did not (Figure S1). Compared to the non-readmission group, the readmission group showed significantly higher levels of times of inhospitalization, white blood cell, procalcitonin, creatinine, creatine kinase-myocardial band, prothrombin time (PT), activated partial thromboplastin time (APTT), D-dimer, temperature, and hypoproteinemia ($P < 0.001$), while the levels of international normalized ratio (INR) and thrombin time (TT) were significantly lower ($P < 0.001$) (Table 1). The study adopted a 7:3 division ratio, with 1429 samples included in the training set, and the test set

Table 1 Baseline Characteristics of Participants with AECOPD Complicated by Hypertension From 2015 to 2024

Characteristic	All Patients	NR Group	R Group	*P* value
	N=2042	N=1277	N=765	
Age	76.23 (8.34)	76.30 (8.52)	76.13 (8.03)	0.650
Gender				0.648
Female	937 (45.89%)	581 (45.50%)	356 (46.54%)	
Male	1105 (54.11%)	696 (54.50%)	409 (53.46%)	
BMI (kg/m^2)	25.56 (16.57)	25.76 (20.39)	25.23 (6.23)	0.383
SBP (mmHg)	136.82 (29.13)	137.79 (33.99)	135.21 (18.23)	0.026
DBP (mmHg)	77.05 (11.81)	77.79 (12.29)	75.81 (10.86)	0.036
Heart rate (bpm)	89.23 (15.11)	88.54 (15.97)	90.38 (13.50)	0.005
Temperature (°C)	36.59 (0.43)	36.56 (0.43)	36.63 (0.43)	< 0.001
Times of inhospitalization	3.64 (2.72)	2.92 (2.36)	4.83 (2.86)	< 0.001
Inhospital length (day)	9.27 (3.47)	9.20 (3.50)	9.39 (3.42)	0.221
White blood cell (10^9/L)	7.84 (2.97)	7.63 (2.90)	8.20 (3.06)	< 0.001
Red blood cell (10^{12}/L)	4.54 (0.65)	4.55 (0.67)	4.52 (0.61)	0.273
Neutrophil (10^9/L)	5.03 (2.26)	5.02 (2.22)	5.05 (2.33)	0.737
Hematocrit (%)	0.42 (0.06)	0.42 (0.06)	0.41 (0.06)	0.440
Hemoglobin (g/L)	136.45 (17.84)	136.51 (18.11)	136.35 (17.40)	0.849
Platelet (10^9/L)	225.05 (74.46)	226.38 (73.83)	222.84 (75.48)	0.302
Procalcitonin (ng/mL)	0.18 (0.12)	0.18 (0.09)	0.20 (0.14)	< 0.001
ESR (mm/h)	23.14 (22.23)	23.65 (23.30)	22.28 (20.29)	0.165
CRP (mg/L)	28.65 (37.48)	27.31 (38.07)	30.88 (36.38)	0.035
ALT (U/L)	20.97 (15.73)	21.51 (16.75)	20.05 (13.83)	0.033
TBIL (μmol/L)	13.69 (6.36)	13.87 (5.98)	13.38 (6.94)	0.101
TP (g/L)	72.98 (6.49)	72.71 (7.01)	73.42 (5.50)	0.010
Creatinine (μmol/L)	73.24 (19.00)	71.84 (14.68)	75.59 (24.41)	< 0.001

(*Continued*)

Table 1 (Continued).

Characteristic	All Patients	NR Group	R Group	*P* value
	N=2042	N=1277	N=765	
BUN (mmol/L)	7.00 (4.94)	6.96 (6.05)	7.08 (2.03)	0.517
Uric acid (μmol/L)	276.62 (75.51)	275.09 (74.25)	279.18 (77.54)	0.241
Glucose (mmol/L)	6.76 (2.72)	6.86 (2.76)	6.61 (2.64)	0.043
TC (mmol/L)	3.30 (1.19)	3.28 (1.22)	3.34 (1.15)	0.259
TG (mmol/L)	1.31 (0.79)	1.30 (0.90)	1.32 (0.58)	0.651
HDL-C (mmol/L)	1.21 (0.24)	1.21 (0.24)	1.20 (0.24)	0.319
LDLC (mmol/L)	2.19 (0.61)	2.18 (0.60)	2.19 (0.61)	0.919
LDH (U/L)	240.44 (101.92)	240.64 (113.02)	240.10 (80.09)	0.899
K^+ (mmol/L)	3.90 (0.47)	3.89 (0.47)	3.91 (0.46)	0.407
Cl^- (mmol/L)	102.87 (5.23)	102.66 (5.34)	103.22 (5.02)	0.018
Na^+ (mmol/L)	139.75 (4.50)	139.60 (4.53)	140.00 (4.45)	0.052
Ca^{2+} (mmol/L)	2.19 (0.13)	2.18 (0.13)	2.20 (0.13)	0.011
P^- (mmol/L)	1.10 (0.23)	1.11 (0.25)	1.08 (0.21)	0.011
Mg^{2+} (mmol/L)	0.87 (0.10)	0.87 (0.10)	0.87 (0.09)	0.751
INR	1.18 (0.38)	1.16 (0.45)	1.21 (0.21)	< 0.001
PT (s)	12.41 (3.09)	11.75 (3.38)	13.51 (2.12)	< 0.001
TT (s)	19.51 (5.86)	20.06 (7.03)	18.59 (2.83)	< 0.001
APTT (s)	23.97 (9.60)	22.67 (11.30)	26.16 (5.02)	< 0.001
Fibrinogen (g/L)	3.56 (1.23)	3.52 (1.24)	3.64 (1.21)	0.025
D-dimer (mg/L)	0.59 (0.49)	0.52 (0.32)	0.71 (0.68)	< 0.001
CK (U/L)	116.99 (49.14)	116.24 (43.78)	118.25 (56.99)	0.402
CKMB (U/L)	13.61 (9.75)	12.74 (7.91)	15.06 (12.08)	< 0.001
pH	7.41 (0.04)	7.41 (0.05)	7.41 (0.04)	0.571
PaO_2 (mmHg)	72.04 (20.98)	71.90 (21.21)	72.29 (20.59)	0.677
$PaCO_2$ (mmHg)	40.96 (8.26)	41.16 (8.71)	40.64 (7.44)	0.152
AB (mmol/L)	25.34 (4.17)	25.41 (4.39)	25.20 (3.80)	0.255
SB (mmol/L)	24.93 (2.87)	24.99 (3.02)	24.84 (2.59)	0.232
SaO_2 (%)	93.28 (4.57)	93.19 (4.72)	93.43 (4.29)	0.237
AG (mmol/L)	8.03 (2.39)	8.06 (2.45)	7.99 (2.30)	0.539
Lactate (mmol/L)	0.84 (3.00)	0.70 (3.33)	1.06 (2.33)	0.005
Smoking				0.472
No	1251 (61.26%)	790 (61.86%)	461 (60.26%)	
Yes	791 (38.74%)	487 (38.14%)	304 (39.74%)	
Drinking				0.517
No	1724 (84.43%)	1073 (84.03%)	651 (85.10%)	
Yes	318 (15.57%)	204 (15.97%)	114 (14.90%)	
Diabetes				0.011
No	1637 (80.17%)	1046 (81.91%)	591 (77.25%)	
Yes	405 (19.83%)	231 (18.09%)	174 (22.75%)	
Hypoproteinemia				< 0.001
No	1769 (86.63%)	1143 (89.51%)	626 (81.83%)	
Yes	273 (13.37%)	134 (10.49%)	139 (18.17%)	

Abbreviations: BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; ESR, erythrocyte sedimentation rate; CRP, C-reactive protein; TBIL, total bilirubin; ALT, alanine aminotransferase; TP, total protein; BUN, blood urea nitrogen; TC, total cholesterol; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; LDH, lactate dehydrogenase; INR, international normalized ratio; PT, prothrombin time; TT, thrombin time; APTT, activated partial thromboplastin time; CK, creatine kinase; CKMB, creatine kinase-myocardial band; PaO_2, partial pressure of Oxygen; $PaCO_2$, partial pressure of Carbon Dioxide; AB, actual bicarbonate; SB, standard bicarbonate; SaO_2, Oxygen saturation; AG, anion gap.

comprised 613 subjects. Supplementary Table 2 compares baseline characteristics between the training and test sets. Statistical analyses demonstrated no significant differences in baseline variables between the two cohorts ($P > 0.05$).

Feature Selection

The initial assessment included 58 variables across demographic, clinical, and laboratory domains. Subsequently, 23 potential predictors were identified using the Boruta algorithm, with shaded features indicating their relevance (Figure 1A). These variables included total bilirubin, neutrophil, C-reactive protein, creatine kinase, triglyceride, lactate dehydrogenase, heart rate, lactate, creatinine, hypoproteinemia, white blood cell, fibrinogen, temperature, creatine kinase-myocardial band, blood urea nitrogen, TT, D-dimer, total protein, INR, APTT, procalcitonin, PT, and times of hospitalization. The identified shadow characteristics are then used for further training and the construction of ML models. To enhance the prediction accuracy of machine learning models, feature variables were optimized by applying machine learning algorithms, namely AdaBoost (Figure 1B) and Random forest (Figure 1C). Each algorithm was utilized to identify the top 10 most important feature variables for their respective models. Subsequently, a comprehensive analysis was conducted using Venn diagrams, ultimately identifying seven variables—times of inhospitalization, procalcitonin, total protein, INR, PT, D-dimer, and hypoproteinemia—for model construction to eliminate redundant variables (Figure 1D). VIF confirmed absence of significant multicollinearity in final models (all VIF < 1.2). (Supplementary Table S3).

The associations among various variables were assessed through the Spearman correlation test. As illustrated in Figure 2, the correlation heatmap visually demonstrated the degree of association among variables, where the correlation coefficient between PT and TP was 0.296, indicating only a weak correlation between them. Notably, remaining variables also demonstrated minimal correlation.

Model Evaluation

We conducted model training and test based on the selected features mentioned above, utilizing eight ML algorithms to build predictive models from the training set (Table 2). By comparing the AUC, we found that AdaBoost exhibited superior diagnostic performance among the test set, with AUC values of 0.884, surpassing those of the other models (Figure 3A). To compare the practical utility of different models, we employed DCA analysis to plot curves on the test set. The DCA analysis (Figure 3B) demonstrated that AdaBoost achieves the highest net benefit across most threshold ranges. In summary, these results indicated that AdaBoost is the most suitable model for the application. Furthermore, the 5-fold cross-validation of the AdaBoost model exhibits strong predictive performance, with average AUC values of 0.889, indicating robust predictive performance and confirming the reliability of our findings. Subsequently, the calibration plot and confusion matrix demonstrated excellent performance for the AdaBoost model (Figure 3C and D).

Model Interpretation

Subsequently, we conducted a SHAP analysis to evaluate the importance of each feature variable in the AdaBoost model and its contribution to model prediction. As shown in Figure 4A, the times of inhospitalization emerged as the most critical predictor in the AdaBoost model. INR was found to be positively correlated with the risk of readmission within one year for patients with AECOPD complicated by hypertension. Additionally, our analysis revealed that total protein was associated with a decreased risk of readmission within one year for these patients. Figure 4B demonstrated that its SHAP values rose significantly as the times of inhospitalization increased. Figure 4C and D provided detailed analyses for patients aged > 40 years with AECOPD complicated by hypertension, yielding a predicted value of 1. The SHAP heat force plot and waterfall plot provided independent risk predictions for each sample and helped to examine how each characteristic variable affects the target variable.

Implementation of a Web Calculator

Following model selection, an online calculator was developed using DecisionLinnc software for clinical applications, with its web service accessible at https://fast.statsape.com/tool/detail?id=17. The online calculator, based on the AdaBoost model, exhibited excellent diagnostic performance in evaluating the risk of readmission within one year for

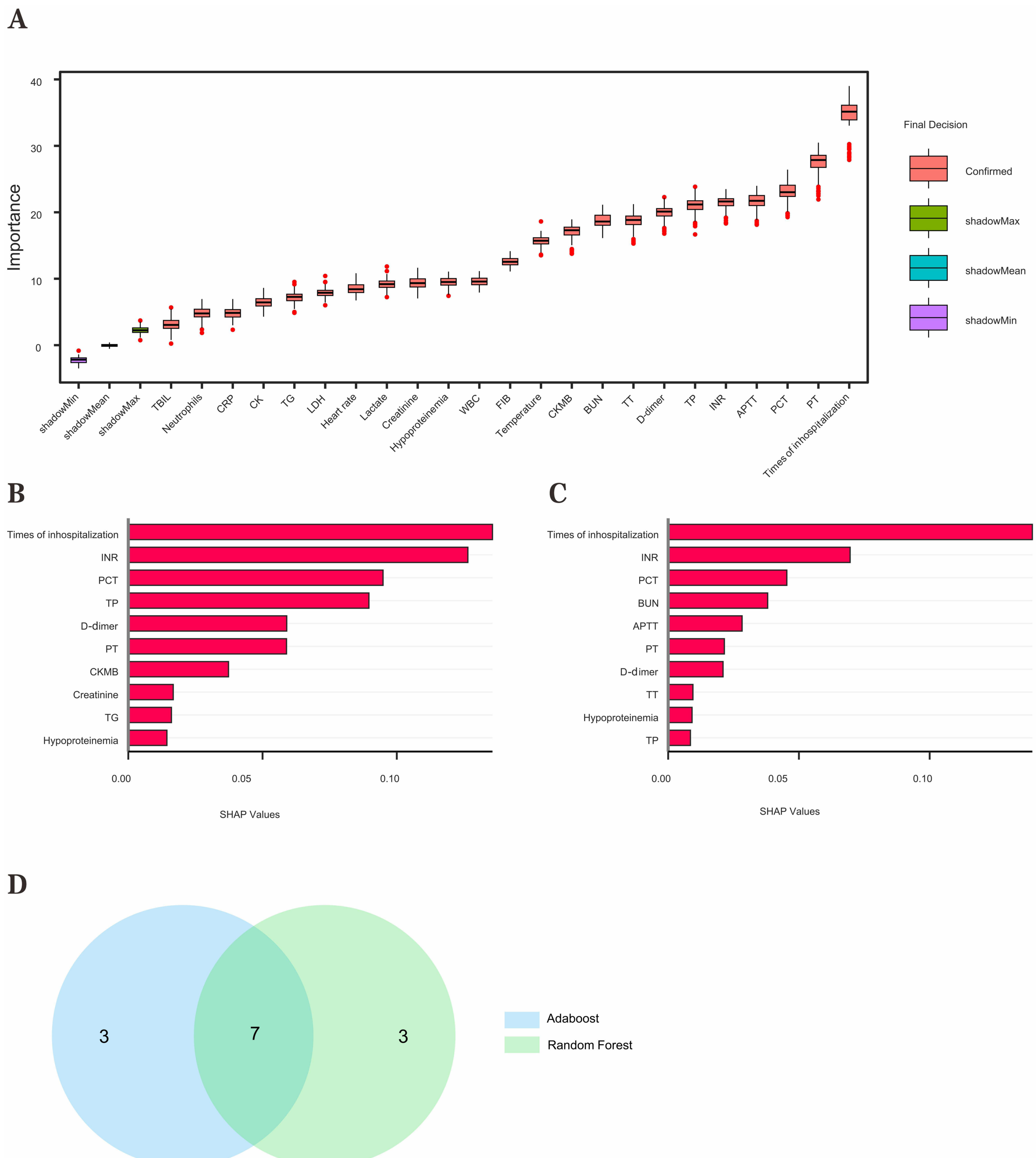

Figure 1 Selection of important variables. (**A**) Image of Boruta method for selecting ML model potentially influential variables. (**B** and **C**) The results of Adaboost and Random forest machine learning algorithms filter the top 10 important variables. (**D**) Venn analysis of the results of the above two machine algorithms.

Abbreviations: TBIL, total bilirubin; CRP, C-reactive protein; CK, creatine kinase; TG, triglyceride; LDH, lactate dehydrogenase; FIB, fibrinogen; CKMB, creatine kinase-myocardial band; BUN, blood urea nitrogen; TT, thrombin time; TP, total protein; INR, international normalized ratio; APTT, activated partial thromboplastin time; PCT, procalcitonin; PT, prothrombin time.

AECOPD complicated by hypertension. By entering blood test results and times of inhospitalization, clinicians can receive personalized risk scores in real-time. An intuitive user guide with clinical examples demonstrates the tool's clinical utilities. For instance, a patient with AECOPD complicated by hypertension can utilize the online tool to input his basic information and blood test results, thereby receiving a personalized one-year readmission risk score.

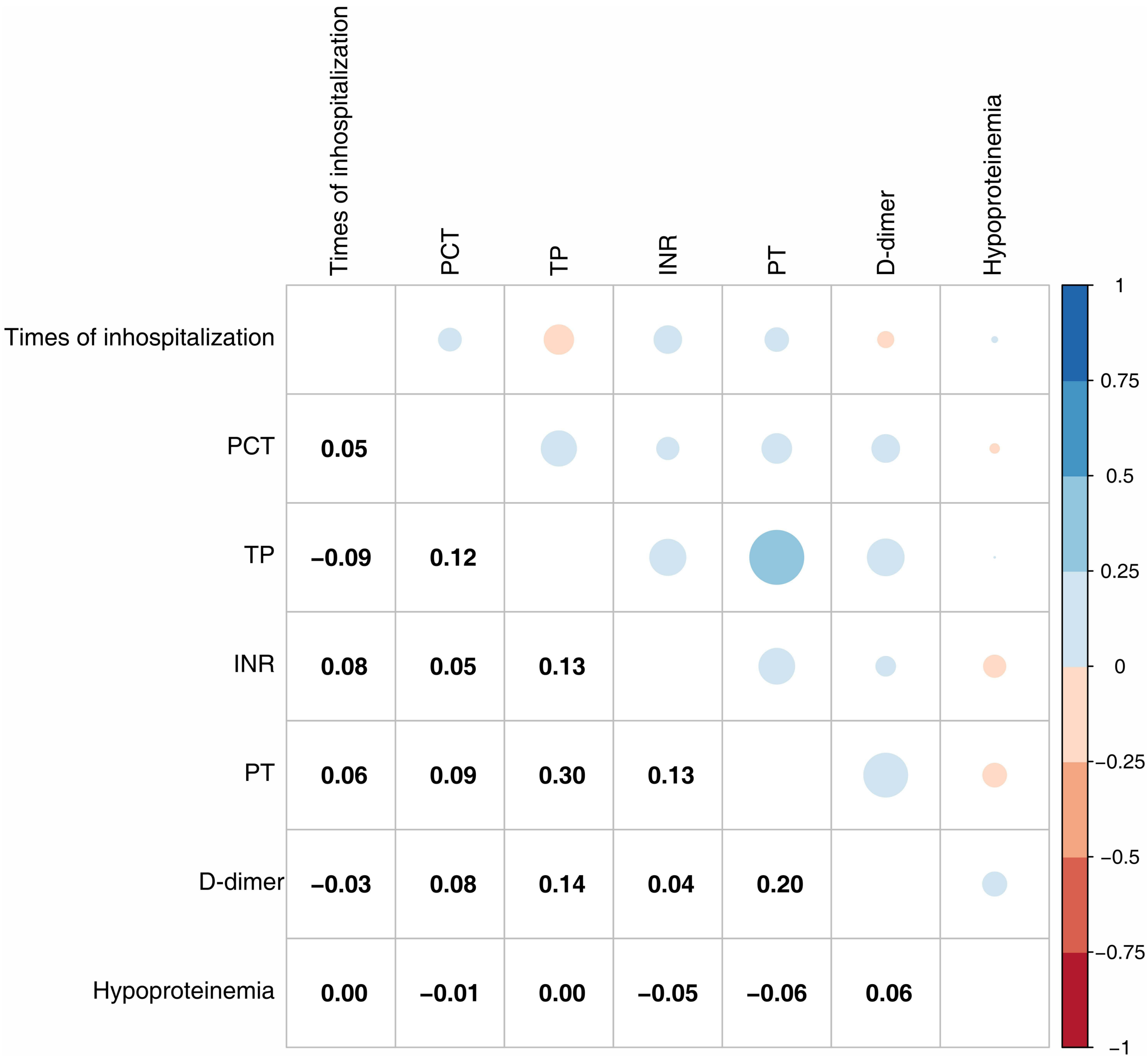

Figure 2 Results of the correlation heatmap between predictive variables.
Abbreviations: PCT, procalcitonin; TP, total protein; INR, international normalized ratio; PT, prothrombin time.

Subsequently, clinicians further guide screening or preventive actions. For detailed information on the online calculator, please refer to the webpage above.

Discussion

In this retrospective cohort study, the readmission rate within one year after discharge for patients with AECOPD complicated by hypertension was 37.5%. Based on real-world data, we constructed an interactive web-based clinical prediction model. The Boruta algorithm, combined with two machine learning algorithms, was employed for feature selection in this study, ultimately identifying seven feature variables, including times of inhospitalization, procalcitonin, total protein, INR, PT, D-dimer, and hypoproteinemia. Among eight machine learning algorithms evaluated, AdaBoost demonstrated superior predictive performance, establishing it as the optimal model. SHAP analysis elucidated the optimal model's decision logic, quantifying each feature's contribution to predictions. Our study demonstrated that the

Table 2 Comparative Analysis of the Performance Outcomes Across Various Machine Learning Models

Model	F1 Score	Accuracy	Precision	AUC	Recall	Specificity
AdaBoost	0.855(0.822–0.888)	0.887(0.859–0.915)	0.840(0.804–0.876)	0.884(0.856–0.912)	0.872(0.840–0.904)	0.840(0.808–0.872)
DT	0.662(0.618–0.706)	0.773(0.740–0.806)	0.768(0.725–0.811)	0.817(0.781–0.853)	0.581(0.532–0.630)	0.892(0.864–0.920)
KNN	0.806(0.770–0.842)	0.855(0.824–0.886)	0.822(0.787–0.857)	0.843(0.812–0.874)	0.791(0.750–0.832)	0.894(0.867–0.921)
LR	0.617(0.570–0.664)	0.741(0.707–0775)	0.707(0.662–0.752)	0.789(0.750–0.828)	0.547(0.498–0.596)	0.860(0.829–0.891)
NB:	0.633(0.587–0.679)	0.706(0.671–0.741)	0.605(0.557–0.653)	0.777(0.737–0.817)	0.662(0.615–0.709)	0.734(0.697–0.771)
RF	0.627(0.580–0.674)	0.777(0.744–0.810)	0.865(0.832–0.898)	0.893(0.867–0.919)	0.491(0.442–0.540)	0.953(0.934–0.972)
SVM	0.305(0.253–0.357)	0.671(0.636–0.706)	0.710(0.663–0.757)	0.789(0.750–0.828)	0.194(0.151–0.237)	0.953(0.934–0.972)
XGBoost	0.602(0.554–0.650)	0.739(0.705–0.773)	0.720(0.675–0.765)	0.791(0.752–0.830)	0.517(0.468–0.566)	0.876(0.845–0.907)

Abbreviations: AdaBoost, adaptive boosting; DT, decision tree; KNN, K-Nearest neighbors; LR, logistic regression; NB, naive bayes; RF, random forest; SVM, support vector machine; XGBoost, extreme gradient boosting; AUC, area under the curve.

identified biomarkers exhibit significant prognostic utility, enabling early intervention and personalized treatment for high-risk patients.

Previous studies have explored predictive models for AECOPD readmission. A retrospective cohort study from the University of Chicago enrolled 3238 adult patients and developed a random forest model based on demographic data, comorbidities, and EHR data. Results showed that 61% of the 1103 patients readmitted within 90 days had respiratory-

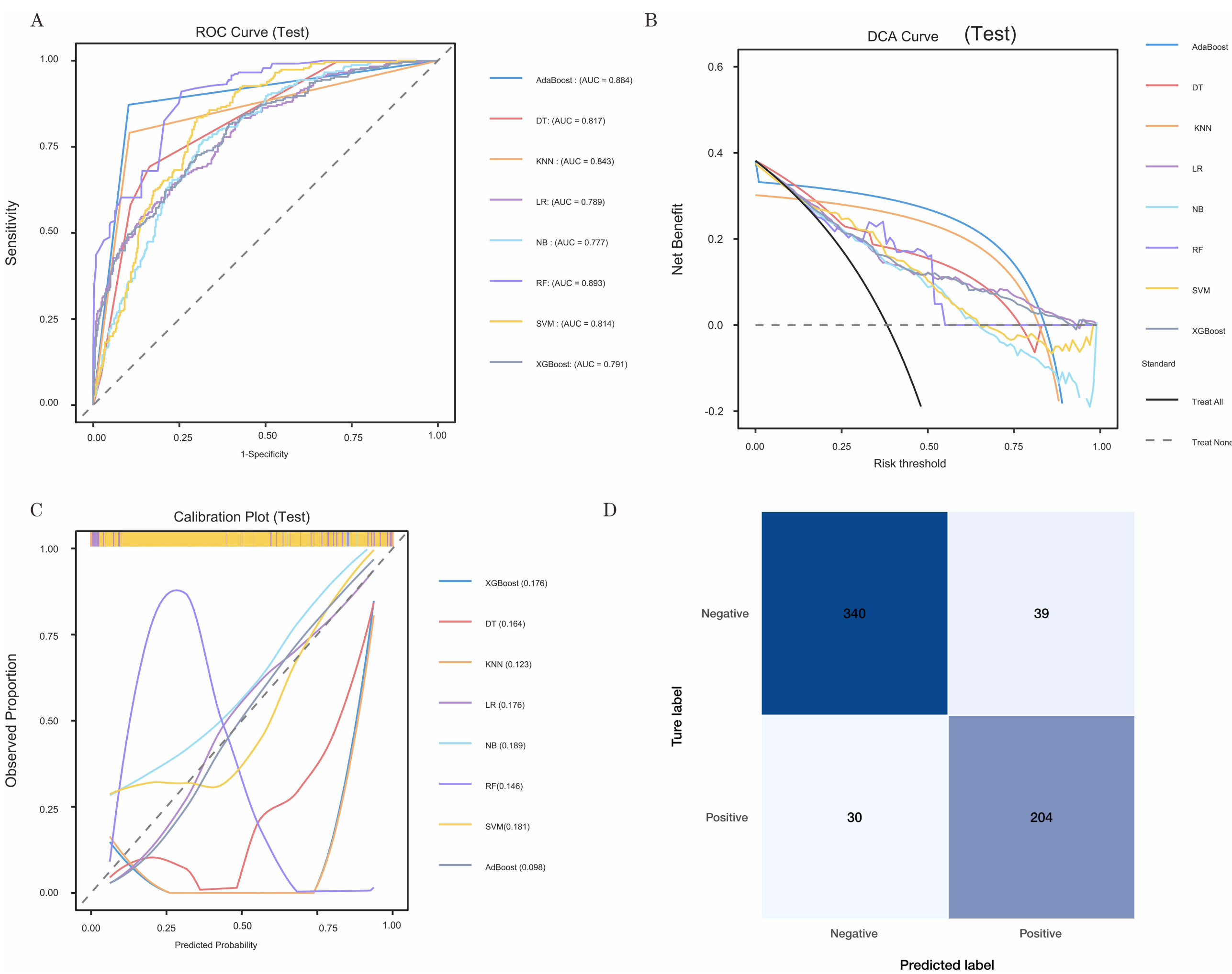

Figure 3 The construction and evaluation of the machine learning model. (**A**) Receiver operating characteristic (ROC) curves of the test sets of 8 ML models. (**B**) Decision curves of the test sets of 8 ML models. (**C**) Calibration curve of the test sets of 8 ML models. (**D**) Confusion matrix of the AdaBoost model on the test set.
Abbreviations: AdaBoost, adaptive boosting; DT, decision tree; KNN, K-Nearest neighbors; LR, logistic regression; NB, naive bayes; RF, random forest; SVM, support vector machine; XGBoost, extreme gradient boosting.

https://doi.org/10.2147/COPD.S557298

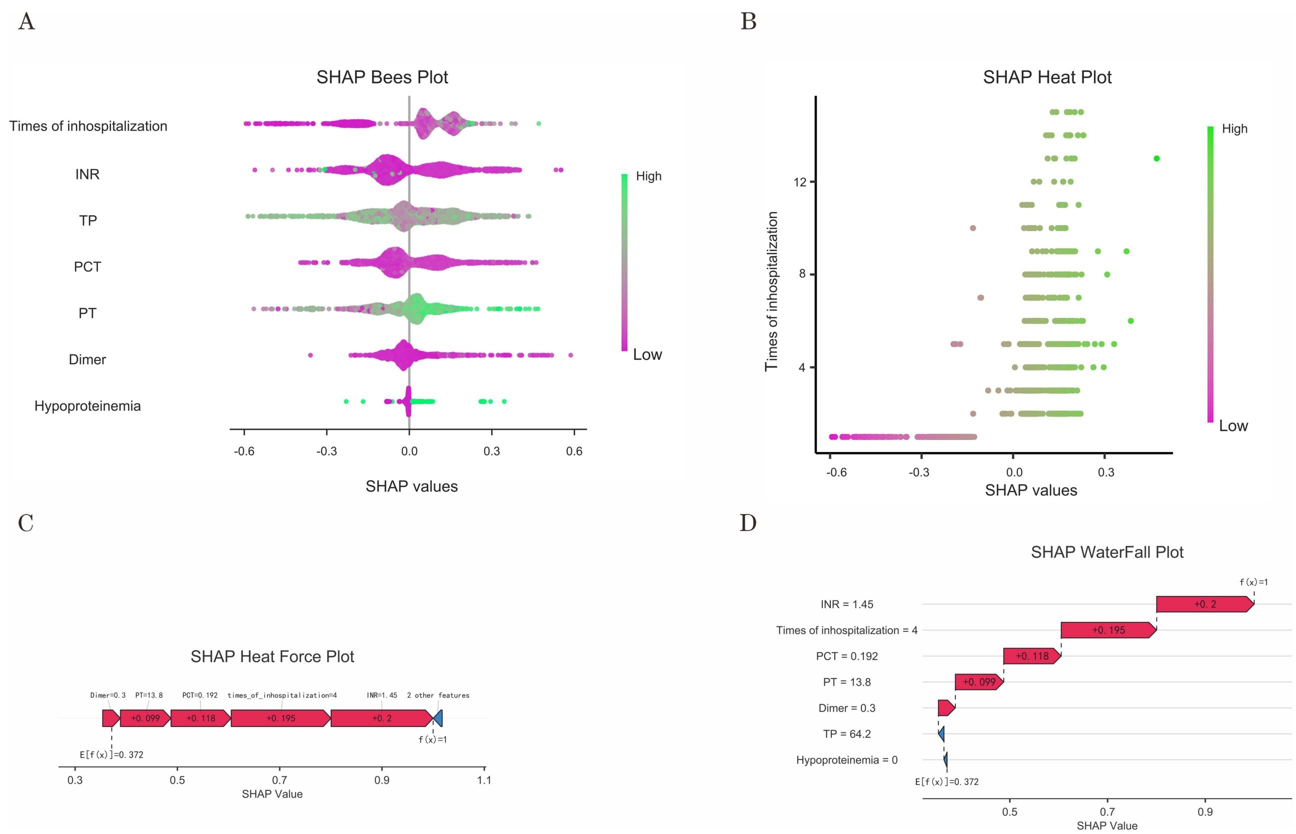

Figure 4 SHAP diagram of AdaBoost model. (**A**) SHAP honeycomb diagram of the AdaBoost model. (**B**) SHAP heat scatter plot of readmission times. (**C**) SHAP force plot. (**D**) SHAP waterfall plot.

Abbreviations: PCT, procalcitonin; TP, total protein; INR, international normalized ratio; PT, prothrombin time.

related causes, with the model achieving an AUC of 0.73 for predicting respiratory-related readmission.[23] Another study analyzed 636 patients and constructed an XGBoost model, finding that a history of acute exacerbation in the past year, long-acting beta$_2$ agonist use, inhaled corticosteroid use, alanine aminotransferase level, and total COPD assessment test score were associated with readmission risk; the XGBoost model exhibited superior predictive performance, with an AUC of 0.722 in the test set.[24] Additionally, a study analyzed electronic medical record data from two Taiwanese hospitals and developed an XGBoost model, identifying that prior AECOPD episodes, mMRC score, CAT score, respiratory rate, and inhaled glucocorticoid use were associated with the risk of AECOPD occurrence at 3 and 6 months. The XGBoost model demonstrated excellent predictive value, with an AUC of 0.795 for 3-month prediction and 0.813 for 6-month prediction.[25] Numerous studies have confirmed the favorable performance of machine learning in AECOPD readmission prediction. However, predictive models specifically targeting the high-risk subgroup of AECOPD patients with hypertension comorbidity remain lacking. This study focuses on this population to fill the research gap and provide evidence-based support for the accurate identification of high-risk individuals.

Among patients with AECOPD complicated by hypertension, a history of frequent hospitalizations was the strongest predictor of readmission, indicating that a higher number of prior hospitalizations may lead to subsequent readmission due to disease exacerbation within 1 year. Prior studies have similarly demonstrated that patients with frequent AECOPD-related hospitalizations exhibit a significantly increased likelihood of readmission within 90 days,[26] underscoring the need for targeted interventions to mitigate this risk. Additionally, one study identified that patients with more than six AECOPD-related hospitalizations annually have higher readmission rates.[27] These findings emphasize the importance of incorporating hospitalization history into risk assessment models to identify high-risk patients who may benefit from intensified care strategies. The relationship between prior hospitalization and readmission is further modulated by comorbidities and disease severity. Patients with chronic conditions such as congestive heart failure or hypertension are more prone to recurrent exacerbations and readmissions, especially if they have a history of multiple hospitalizations.[28] Therefore, for patients with prior hospitalizations ≥ 4 times, intensified follow-up management should be implemented after discharge: Clinicians should closely monitor the patient's condition, optimize medication regimens, intervene in modifiable risk factors, and guide patients in self-monitoring of symptoms. This enables early identification of signs of acute exacerbation and timely intervention to prevent progression to the point requiring readmission.

Procalcitonin, a peptide precursor of calcitonin, is typically produced by the thyroid gland under normal physiological conditions. However, its production is upregulated across various tissues during systemic bacterial infections, making it a sensitive biomarker for bacterial sepsis and other severe infections.[29] The association between procalcitonin levels and readmission risk in AECOPD is complex.[30] Evidence suggested that elevated procalcitonin levels significantly predicted higher 30-day readmission risk in AECOPD.[31] Another study similarly indicated that elevated procalcitonin levels at admission correlate with poorer clinical outcomes, including longer hospital stays and higher ICU utilization rates, which may indirectly influence readmission rates.[32] A retrospective study involving 359 COPD patients revealed that during a 5-year follow-up period, elevated procalcitonin levels, along with comorbidities such as hypertension and diabetes, were identified as independent predictive factors for readmission.[33] Our analysis confirms procalcitonin as a significant independent predictor of 1-year readmission in hypertension-complicated AECOPD patients. Collectively, these findings establish procalcitonin not only as a biomarker of acute infection but as a stratifying predictor for long-term readmission risk, particularly in patients with AECOPD complicated by hypertension. Clinically, the rational use of PCT levels to guide antibiotic therapy can reduce readmissions resulting from drug resistance or inadequate infection control; for patients with elevated PCT at discharge, intensified follow-up should be conducted to monitor for residual infections.

Hypoproteinemia, characterized by abnormally low total protein levels in the blood, serves as a critical clinical marker of poor prognosis across various medical conditions, including AECOPD.[34] As a composite indicator of albumin and globulin, total protein is essential for assessing nutritional status and prognostic management in AECOPD patients.[35] Study demonstrated that total protein and albumin levels were significantly lower in AECOPD patients compared to healthy individuals or those with mild COPD, suggesting that protein metabolic dysregulation may exacerbate acute-phase inflammation and respiratory failure.[36] Notably, another study confirmed hypoalbuminemia as an independent predictor of early COPD readmission (adjusted OR 2.02, 95% CI 1.03–3.95), doubling readmission risk and correlating with increased mortality.[37] While hypoproteinemia does not directly cause AECOPD, its role as a marker of systemic inflammation and impaired immune function is linked to lung function decline and 1-year readmission risk, underscoring the necessity of nutritional interventions.[38] For patients with hypoproteinemia, individualized nutritional plans should be formulated by dietitians. For severe cases, high-protein preparations or intravenous albumin should be administered during hospitalization, followed by switching to oral supplementation after discharge. Long-term monitoring of serum protein levels is required with dynamic adjustments to the nutritional plan, so as to break the vicious cycle of malnutrition and readmission. Additionally, prognostic nutritional index reflecting total protein and albumin levels is independently associated with increased 30-day readmission rates and worse clinical outcomes in AECOPD patients.[39] Our study further demonstrated that hypoproteinemia and total protein level served as independent predictors of 1-year readmission in patients with AECOPD complicated by hypertension.

COPD is a chronic inflammatory disorder characterized by a hypercoagulable state.[40] Growing evidence indicates that hypercoagulability in COPD involves changes in multiple coagulation factors.[41] AECOPD originates from localized pulmonary hypoxia, which initiates inflammatory cascades that drive airway remodeling, parenchymal destruction, and vascular injury. This self-amplifying cycle exacerbates inflammation and damages pulmonary endothelium, culminating in systemic coagulation activation.[42] D-dimer, a molecular marker of hypercoagulability and excessive fibrinolysis in vivo, effectively reflects thrombin generation and fibrinolytic activity.[43] Elevated D-dimer levels are associated with increased risks of readmission and 1-year mortality in AECOPD patients.[44] In this study, the average D-dimer level in AECOPD patients exceeded the normal range, indicating a hypercoagulable state, particularly in those with 1-year readmission. INR and PT are also commonly used clinical indicators for assessing coagulation function. Study reported that compared to stable COPD patients and controls, the AECOPD group demonstrated significantly lower INR and prolonged PT, indicating hypercoagulability during exacerbations.[45] Our study further demonstrated that among patients with AECOPD complicated by hypertension, those with 1-year readmission had lower INR values and longer PT compared with non-readmitted patients. The close association between D-dimer, INR, PT, and 1-year readmission in AECOPD complicated by hypertension highlights their role as sensitive markers of disease severity and deterioration risk, and reflects early abnormalities in coagulation function. Clinically, for patients with AECOPD complicated by hypertension who present with significant prolongation of PT or marked elevation of D-dimer during hospitalization, early initiation of thromboprophylaxis and optimization of respiratory support are recommended to block the hypoxia-induced hypercoagulable state. Meanwhile, regular monitoring of coagulation indices is required after discharge, with timely adjustment of anticoagulant regimens.

Limitations

This study has several limitations. Firstly, as a retrospective single-center study, its results are susceptible to selection bias and confounding factors and lack the support of a prospective design for causal inference. The single-center nature further limits the generalizability of the findings to multi-institutional populations, necessitating future studies with diverse regional samples to enhance the universality of the results. Secondly, although baseline covariates were adjusted in the statistical analysis, key confounding factors—such as dynamic adjustments in post-discharge treatment regimens including respiratory rehabilitation and nutritional interventions—were not included in the model. Thirdly, some variables contained missing data, and while this issue has been addressed through multiple imputation, it may still introduce bias and thereby compromise the model's accuracy. Finally, this study focused on predictive correlations without delving into the pathophysiological mechanisms underlying patients with AECOPD complicated by hypertension, warranting further mechanistic investigation.

Conclusion

In summary, this study preliminarily explored the application of machine learning algorithms, ultimately adopting the AdaBoost model to develop a predictive tool for 1-year readmission risk in patients with AECOPD complicated by hypertension. Notably, this study is limited by its single-center, retrospective design, and the model's performance is currently supported only by internal validation. Therefore, the model's generalizability and long-term utility require further verification through multi-center, prospective external validation.

Data Sharing Statement

The datasets used and analyzed during the current study are available from the corresponding author on reasonable request.

Ethics Approval and Consent to Participate

This study was approved by the Ethics Review Committee of the First Affiliated Hospital of Shihezi University, China (approval number: KJ2024-522-02). As this was a retrospective study analyzing anonymized clinical data, the requirement for informed consent was waived by the Ethics Committee. Clinical trial number: not applicable.

Acknowledgments

We acknowledge the DecisionLinnc1.0 software (https://www.statsape.com/) for its professional technical support in data analysis.

Author Contributions

XZ was responsible for the concept and design of the study. DL obtained funding. XZ, JZ, and MH participated in the pre-processing of the datasets. XZ, JZ, MH, and LZ performed the statistical analyses.

All authors made substantial contributions to conception and design, acquisition of data, or analysis and interpretation of data; took part in drafting the article or revising it critically for important intellectual content; 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.

Funding

This work was supported by the Science and Technology Program of Xinjiang Production and Construction Corps (grant number: 2023ZD019).

Disclosure

The authors declare no competing interests.

References

1. Ramakrishnan S. Chronic obstructive pulmonary disease: 10 years of precision-guided success. *Lancet Respir Med*. 2023;11(3):227–228. doi:10.1016/S2213-2600(23)00013-9
2. Chen S, Kuhn M, Prettner K, et al. The global economic burden of chronic obstructive pulmonary disease for 204 countries and territories in 2020-50: a health-augmented macroeconomic modelling study. *Lancet Glob Health*. 2023;11(8):e1183–e1193. doi:10.1016/S2214-109X(23)00217-6
3. Owusuaa C, Van der Leest C, Helfrich G, et al. The development of the ADO-SQ model to predict 1-year mortality in patients with COPD. *Palliat Med*. 2022;36(5):821–829. doi:10.1177/02692163221080662
4. Greulich T, Weist BJD, Koczulla AR, et al. Prevalence of comorbidities in COPD patients by disease severity in a German population. *Respir Med*. 2017;132:132–138. doi:10.1016/j.rmed.2017.10.007
5. Wang JG, Zhang W, Li Y, Liu L. Hypertension in China: epidemiology and treatment initiatives. *Nat Rev Cardiol*. 2023;20(8):531–545. doi:10.1038/s41569-022-00829-z
6. Chen Q, Zhou H, Tang J, et al. An analysis of exogenous harmful substance exposure as risk factors for COPD and hypertension co-morbidity using PSM. *Front Public Health*. 2024;12:1414768. doi:10.3389/fpubh.2024.1414768
7. Li T, Chen L, Xu H, et al. The association between cardiovascular diseases and their subcategories with the severity of chronic obstructive pulmonary disease: a large cross-sectional study based on a Chinese hospital population cohort. *Front Cardiovasc Med*. 2025;12:1502205.
8. Yang HY, Hu LY, Chen HJ, Chen RY, Hu CK, Shen CC. Increased risk of chronic obstructive pulmonary disease in patients with hyperlipidemia: a nationwide population-based cohort study. *Int J Environ Res Public Health*. 2022;19(19):12331.
9. Su TH, Chang SH, Chen PC, Chan YL. Temporal trends in treatment and outcomes of acute myocardial infarction in patients with chronic obstructive pulmonary disease: a nationwide population-based observational study. *J Am Heart Assoc*. 2017;6(3). doi:10.1161/JAHA.116.004525
10. Fakhraei R, Matelski J, Gershon A, et al. Development of multivariable prediction models for the identification of patients admitted to hospital with an exacerbation of COPD and the prediction of risk of readmission: a retrospective cohort study using electronic medical record data. *COPD*. 2023;20(1):274–283. doi:10.1080/15412555.2023.2242493
11. Chokkara S, Hermsen MG, Bonomo M, et al. Comparison of chart review and administrative data in developing predictive models for readmissions in chronic obstructive pulmonary disease. *Chronic Obstr Pulm Dis*. 2025;12(2):175–183. doi:10.15326/jcopdf.2024.0542
12. Sajjadian M, Lam RW, Milev R, et al. Machine learning in the prediction of depression treatment outcomes: a systematic review and meta-analysis. *Psychol Med*. 2021;51(16):2742–2751. doi:10.1017/S0033291721003871
13. Tseng PY, Chen YT, Wang CH, et al. Prediction of the development of acute kidney injury following cardiac surgery by machine learning. *Crit Care*. 2020;24(1):478. doi:10.1186/s13054-020-03179-9
14. Huang S, Yang J, Fong S, Zhao Q. Mining prognosis index of brain metastases using artificial intelligence. *Cancers*. 2019;11(8):1140. doi:10.3390/cancers11081140
15. Elkadri A, Thoeni C, Deharvengt SJ, et al. Mutations in plasmalemma vesicle associated protein result in sieving protein-losing enteropathy characterized by hypoproteinemia, hypoalbuminemia, and hypertriglyceridemia. *Cell Mol Gastroenterol Hepatol*. 2015;1(4):381–394.e387. doi:10.1016/j.jcmgh.2015.05.001
16. Guo QH, Xie FC, Zhong FM, et al. Application of interpretable machine learning algorithms to predict distant metastasis in ovarian clear cell carcinoma. *Cancer Med*. 2024;13(7):e7161. doi:10.1002/cam4.7161
17. Habibzadeh F. On the use of receiver operating characteristic curve analysis to determine the most appropriate p value significance threshold. *J Transl Med*. 2024;22(1):16. doi:10.1186/s12967-023-04827-8
18. Van Calster B, Wynants L, Verbeek JFM, et al. Reporting and interpreting decision curve analysis: a guide for investigators. *Eur Urol*. 2018;74(6):796–804. doi:10.1016/j.eururo.2018.08.038
19. Vickers AJ, Van Calster B, Steyerberg E. Decision curves, calibration, and subgroups. *J Clin Oncol*. 2017;35(4):472–473. doi:10.1200/JCO.2016.69.1576
20. Qi X, Wang S, Fang C, Jia J, Lin L, Yuan T. Machine learning and SHAP value interpretation for predicting comorbidity of cardiovascular disease and cancer with dietary antioxidants. *Redox Biol*. 2025;79:103470. doi:10.1016/j.redox.2024.103470
21. Fu Q, Wu Y, Zhu M, et al. Identifying cardiovascular disease risk in the U.S. population using environmental volatile organic compounds exposure: a machine learning predictive model based on the SHAP methodology. *Ecotoxicol Environ Saf*. 2024;286:117210. doi:10.1016/j.ecoenv.2024.117210
22. Li J, Liu S, Hu Y, Zhu L, Mao Y, Liu J. Predicting mortality in intensive care unit patients with heart failure using an interpretable machine learning model: retrospective cohort study. *J Med Internet Res*. 2022;24(8):e38082. doi:10.2196/38082
23. Bonomo M, Hermsen MG, Kaskovich S, et al. Using machine learning to predict likelihood and cause of readmission after hospitalization for chronic obstructive pulmonary disease exacerbation. *Int J Chron Obstruct Pulmon Dis*. 2022;17:2701–2709. doi:10.2147/COPD.S379700
24. Chen L, Chen S. Prediction of readmission in patients with acute exacerbation of chronic obstructive pulmonary disease within one year after treatment and discharge. *BMC Pulm Med*. 2021;21(1):320. doi:10.1186/s12890-021-01692-3
25. Liao KM, Cheng KC, Sung MI, et al. Machine learning approaches for practical predicting outpatient near-future AECOPD based on nationwide electronic medical records. *iScience*. 2024;27(4):109542. doi:10.1016/j.isci.2024.109542

26. Echevarria C, Steer J, Heslop-Marshall K, et al. The PEARL score predicts 90-day readmission or death after hospitalisation for acute exacerbation of COPD. *Thorax*. 2017;72(8):686–693. doi:10.1136/thoraxjnl-2016-209298
27. Bell J, Lim S, Mikami T, Bahk J, Argiro S, Steiger D. The impact on thirty day readmissions for patients hospitalized for acute exacerbations of chronic obstructive pulmonary disease admitted to an observation unit versus an inpatient medical unit: a retrospective observational study. *Chronic Respir Dis*. 2024;21:14799731241242490. doi:10.1177/14799731241242490
28. Cavalot G, Dounaevskaia V, Vieira F, et al. One-year readmission following undifferentiated acute hypercapnic respiratory failure. *COPD*. 2021;18 (6):602–611. doi:10.1080/15412555.2021.1990240
29. Pei Y, Chen L, Zhao Y, et al. Advances of immunosensors based on noble metal composite materials for detecting procalcitonin. *Mikrochimica acta*. 2025;192(2):72. doi:10.1007/s00604-025-06953-0
30. Li Z, Yuan X, Yu L, Wang B, Gao F, Ma J. Procalcitonin-guided antibiotic therapy in acute exacerbation of chronic obstructive pulmonary disease: an updated meta-analysis. *Medicine*. 2019;98(32):e16775. doi:10.1097/MD.0000000000016775
31. Ulrich RJ, McClung D, Wang BR, Winters S, Flanders SA, Rao K. Introduction of procalcitonin testing and antibiotic utilization for acute exacerbations of chronic obstructive pulmonary disease. *Infect Dis*. 2019;12(1178633719852626). doi:10.1177/1178633719852626
32. Davies AJ, Blessing PW, Eilbert WP. Measurement of procalcitonin as an indicator of severity in patients with chronic obstructive pulmonary disease admitted with respiratory illness. *Cureus*. 2022;14(8):e28511. doi:10.7759/cureus.28511
33. Flattet Y, Garin N, Serratrice J, Perrier A, Stirnemann J, Carballo S. Determining prognosis in acute exacerbation of COPD. *Int J Chron Obstruct Pulmon Dis*. 2017;12:467–475. doi:10.2147/COPD.S122382
34. Ding CW, Huang SS, Xu YH, et al. Lactate dehydrogenase to albumin ratio and prognosis in patients with acute exacerbation of chronic obstructive pulmonary disease: a retrospective cohort study. *BMC Pulm Med*. 2025;25(1):154. doi:10.1186/s12890-025-03622-z
35. Dai L, Liang BM, Ou XM. Predictive value of neutrophil-to-lymphocyte ratio and bilirubin levels in the readmission of acute exacerbation of chronic obstructive pulmonary disease. *Am J Med Sci*. 2023;365(2):169–175. doi:10.1016/j.amjms.2022.05.026
36. Luo L, Zheng D, Da L, Cheng J, Cao Y, Wang N. Thrombin generation indices and wells score predict pulmonary embolism in patients with acute exacerbation of chronic obstructive pulmonary disease. *Clinics*. 2025;80:100582. doi:10.1016/j.clinsp.2025.100582
37. Lin J, Xu Y, Wu X, et al. Risk factors associated with chronic obstructive pulmonary disease early readmission. *Curr Med Res Opin*. 2014;30 (2):315–320. doi:10.1185/03007995.2013.858623
38. Shi G, Yue L, Tang Z, Wang Y, Hu X, Tong Y. Serum growth differentiation factor 15 as a biomarker for malnutrition in patients with acute exacerbation of chronic obstructive pulmonary disease. *Front Nutr*. 2024;11:1404063. doi:10.3389/fnut.2024.1404063
39. Yuan FZ, Xing YL, Xie LJ, et al. The relationship between prognostic nutritional indexes and the clinical outcomes of patients with acute exacerbation of chronic obstructive pulmonary disease. *Int J Chron Obstruct Pulmon Dis*. 2023;18:1155–1167. doi:10.2147/COPD.S402717
40. Kyriakopoulos C, Gogali A, Kostikas K, Konstantinidis A. Hypercoagulable state in COPD-A comprehensive literature review. *Diagnostics*. 2021;11(8):1447. doi:10.3390/diagnostics11081447
41. Rahaghi FN, Pistenmaa CL. Hypercoagulation in COPD: the clot thickens. *ERJ Open Res*. 2021;7(4):00534–2021. doi:10.1183/23120541.00534-2021
42. Zheng LL, Wang S, Li ZG, et al. Correlation of coagulation dysfunction with infection and hypercapnia in acute exacerbation of COPD patients. *Infect Drug Resist*. 2023;16:5387–5394. doi:10.2147/IDR.S421925
43. Li L, Feng Q, Yang C. The D-dimer to albumin ratio could predict hospital readmission within one year in patients with acute exacerbation of chronic obstructive pulmonary disease. *Int J Chron Obstruct Pulmon Dis*. 2024;19:2587–2597. doi:10.2147/COPD.S481483
44. Hu G, Wu Y, Zhou Y, et al. Prognostic role of D-dimer for in-hospital and 1-year mortality in exacerbations of COPD. *Int J Chron Obstruct Pulmon Dis*. 2016;11:2729–2736. doi:10.2147/COPD.S112882
45. Liu M, Hu R, Jiang X, Mei X. Coagulation dysfunction in patients with AECOPD and its relation to infection and hypercapnia. *J Clin Lab Anal*. 2021;35(4):e23733. doi:10.1002/jcla.23733