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ORIGINAL RESEARCH

Interpretable Machine Learning Analysis of Inflammatory Biomarkers for Predicting Arteriovenous Fistula Stenosis in Hemodialysis Patients: A Retrospective Cohort Study

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Objective: To develop an interpretable machine learning model for predicting arteriovenous fistula (AVF) stenosis in hemodialysis patients using inflammatory biomarkers and identify key influencing factors.

Methods: A retrospective cohort study was conducted on 974 end-stage renal disease patients undergoing hemodialysis with AVF at The Central Hospital of Wuhan (2017–2024). Clinical data (demographics, comorbidities, inflammatory markers) were collected. After data preprocessing (imputation, normalization, feature selection), eight machine learning models including Logistic Regression (LR) were built and validated via 10-fold cross-validation. SHAP (SHapley Additive Explanations) was used to interpret model outputs.

Results: The LR model outperformed others with an AUC of 0.833 (95% confidence interval [CI]: 0.796–0.868), an accuracy of 0.782 (95% CI: 0.751–0.811), and an F1 score of 0.756 (95% CI: 0.718–0.791). Key factors associated with AVF stenosis included AVF surgical history, thrombosis history, comorbidities, smoking, alcohol consumption, monocyte-to-high-density lipoprotein cholesterol ratio (MHR), and platelet-to-high-density lipoprotein cholesterol ratio (PHR) ($p < 0.05$). SHAP visualization showed these factors significantly impacted model predictions, with MHR/PHR correlating with reduced stenosis risk when elevated.

Conclusion: The LR model based on inflammatory biomarkers effectively predicts AVF stenosis. Integrating SHAP (SHapley Additive Explanations) values enhances the interpretability of the model, thus providing a practical tool for clinical risk stratification and early intervention of AVF stenosis in hemodialysis patients.

Keywords: arteriovenous fistula stenosis, hemodialysis, metabolism-integrated inflammatory biomarkers, machine learning, SHAP value

Introduction

End-stage renal disease (ESRD) is a severe medical condition characterized by the complete loss of renal function, requiring either dialysis or kidney transplantation to sustain patient life. Kidney transplantation and renal replacement therapies are the principal treatment strategies for managing ESRD. Globally, 78% of individuals with chronic kidney disease (CKD) undergo dialysis, while 22% receive kidney transplants.[1] Approximately 90% of patients undergoing dialysis receive hemodialysis treatment, with the primary vascular access methods for this procedure being arteriovenous fistulas (AVFs) and central venous catheters.[2] An AVF is a surgically constructed vascular access point, established by anastomosing the radial artery near the wrist in the forearm to an adjacent vein. This procedure facilitates the diversion of arterial blood into the venous system, thereby inducing arterialization of the vein. Consequently, the venous walls undergo gradual thickening due to the increased arterial pressure, ensuring sufficient blood flow necessary for effective dialysis. The global number of hemodialysis patients is projected to reach 5.439 million by 2030. As of the end of 2022, the number of patients with ESRD undergoing dialysis in China has exceeded one million, of whom 844,000 receive

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hemodialysis, and this number is increasing annually.[3,4] According to the consensus of vascular access experts in our country's hemodialysis, the proportion of patients using AVFs as vascular access for hemodialysis in each dialysis center should be greater than 80%.[5] Therefore, the number of end-stage renal disease patients undergoing dialysis using AVF is a significant group, and vascular access for haemodialysis has long been recognized as an unresolved clinical challenge worldwide despite its widespread application.[6]

However, the use of arteriovenous fistulas faces many challenges. A meta-analysis examining the risk of AVF failure revealed that the incidence of AVF failure varied between 3.9% and 39%.[7] The primary factors contributing to the failure of arteriovenous fistulas are stenosis, thrombosis, and aneurysms. Notably, the incidence of cephalic arch stenosis varies widely, with reported rates ranging from 4% to 64%.[8,9] Studies have indicated that inflammation significantly contributes to the development and failure of arteriovenous fistulas Stenosis in these fistulas is primarily due to intimal hyperplasia, which is linked to inflammation. High systemic inflammatory markers, such as interleukin-6 (IL-6), are associated with fistula dysfunction. Inflammation impacts endothelial cell function, promotes immune cell infiltration, and causes abnormal vascular smooth muscle cell proliferation, worsening intimal hyperplasia and stenosis.[10–12]

The AVF serves as a critical lifeline for patients undergoing dialysis. Maintaining its patency is essential for extending patient survival. Early intervention plays a pivotal role in the outcomes of patients undergoing hemodialysis via AVF. Notably, the risk of re-catheterization due to fistula failure is 1.5 times higher in AVF patients who did not receive early follow-up than in those who did.[13] Stenosis in arteriovenous fistulas is hard to detect early, often delaying diagnosis and treatment, which raises the risk of thrombosis and access failure. The physical examination demonstrates a certain degree of sensitivity and specificity in the early detection of AVF stenosis; however, its application is constrained by various factors. Research indicates that although physical examination exhibits acceptable sensitivity and specificity for evaluating AVF maturity and stenosis, its widespread clinical application is hindered by insufficient training and knowledge of physical examination techniques among dialysis practitioners.[14] Recently, immune-inflammatory markers have gained attention for their role in AVF stenosis. Research indicates that immune-inflammatory responses are crucial in the onset and progression of AVF stenosis. Research has demonstrated a significant association between the neutrophil-to-lymphocyte ratio (NLR) and AVF stenosis, identifying NLR as a pertinent systemic inflammatory marker. One particular study reported a marked elevation of NLR levels in patients with AVF stenosis, with a positive correlation observed between NLR and the severity of stenosis. These findings suggest that NLR may serve as a valuable predictive marker for AVF stenosis.[7] Furthermore, other inflammatory markers, including the monocyte-to-lymphocyte ratio (MLR) and platelet-to-lymphocyte ratio (PLR), have also been recognized for their potential utility in predicting AVF stenosis[15] Consequently, the early utilization of inflammatory markers to predict and preemptively address arteriovenous fistula stenosis holds substantial significance in the prevention of this condition.

Among the diverse immune-inflammatory markers, we specifically selected the NHR, MHR, and PHR for this study, based on their strong biological relevance to the pathophysiology of AVF stenosis and clinical practicability. NHR reflects the synergy of neutrophil-mediated inflammation and anemia-related oxidative stress—both key drivers of intimal hyperplasia in AVFs.[10,12] MHR and PHR integrate the pro-inflammatory effects of monocytes/platelets with the anti-inflammatory, endothelial-protective properties of high-density lipoprotein cholesterol (HDL-C); these dual-component ratios are more sensitive to local vascular inflammation in AVFs than single-cell or lipid markers alone. Unlike NLR and PLR (the most commonly used inflammatory ratios in previous studies), NHR, MHR, and PHR account for metabolic and hematological abnormalities (eg, anemia, dyslipidemia) that are highly prevalent in hemodialysis patients, thus making them more tailored to the unique clinical characteristics of this population.[7,15] Additionally, these ratios are derived from routine blood test results, requiring no additional clinical examinations, which ensures their feasibility for widespread clinical application in dialysis centers.

Recently, machine learning has been increasingly used to predict arteriovenous fistula stenosis, highlighting the medical field's need for precision and its ability to manage complex data. A study employed an integrative methodology, combining tensor decomposition with machine learning techniques, to evaluate the feasibility of detecting arteriovenous fistula stenosis using B-mode ultrasound videos. The findings revealed that by restricting the video frames to regions depicting blood flow, the machine learning model achieved a marked improvement in the accuracy of stenosis detection. Despite the limited dataset size, the model demonstrated promising predictive performance.[16]

Research indicates that machine learning models are capable of accurately classifying the severity of arterial stenosis, thereby facilitating early intervention opportunities in treatment decision-making for this condition. This machine learning-based classification approach enhances diagnostic accuracy and minimizes errors associated with the manual review process.[17]

Several prediction models for AVF stenosis have been developed to date, including traditional clinical risk scores and machine learning-based models. The latest clinical core curriculum for hemodialysis vascular access has further emphasized the urgent need for accurate and interpretable predictive models to optimize AVF management and improve clinical outcomes.[18] Conventional clinical risk scores primarily rely on demographic and clinical factors (eg, age, diabetes mellitus, smoking, AVF surgical history),[13] but they overlook the contribution of immune-inflammatory pathways and have limited predictive accuracy due to their linear design. Existing ML-based models for AVF stenosis have focused on ultrasound imaging features (eg, B-mode ultrasound videos),[16] or a narrow panel of inflammatory markers (eg, NLR, PLR),[7,15] with two key limitations: first, imaging-based models require specialized equipment and trained personnel, making them inaccessible to low-resource dialysis centers; second, models using only conventional inflammatory markers fail to capture the complex interplay between inflammation, metabolism, and vascular function in hemodialysis patients. Furthermore, most existing ML models are "black boxes" with limited interpretability, which hinders their clinical adoption by nephrologists and dialysis practitioners.[17]

In this study, we aim to develop a clinically applicable, interpretable ML model for predicting AVF stenosis in hemodialysis patients by integrating a panel of metabolism-integrated inflammatory markers (NHR, MHR, PHR, etc.) with routine clinical factors. Our study's novel contributions to the existing literature are threefold: (1) we systematically evaluate the predictive value of NHR, MHR, and PHR—metabolism-coupled inflammatory ratios—for AVF stenosis for the first time, filling the gap in existing models that overlook the synergy of inflammation and metabolic abnormalities in hemodialysis patients; (2) we compare eight ML models to identify the optimal one, and use SHAP values to achieve full interpretability of the model outputs, addressing the "black box" problem of most existing ML-based AVF stenosis prediction models; (3) we validate the performance of a simple, interpretable LR model alongside complex ML models, providing a practical tool that can be easily implemented in routine clinical practice without specialized computational support. By addressing these gaps, our study aims to provide a precise, accessible, and interpretable risk stratification tool for AVF stenosis, thereby facilitating early intervention and improving vascular access outcomes in hemodialysis patients.

Methods

Study Population

This research constitutes a retrospective cohort study, wherein data were gathered from patients diagnosed with end-stage renal disease who received hemodialysis through an arteriovenous fistula. These patients were treated at the Department of Nephrology, The Central Hospital of Wuhan, over the period from January 1, 2017, to December 31, 2024. Throughout the 8-year study period (2017–2024), the clinical diagnostic criteria for AVF stenosis, hemodialysis treatment protocols, and standardized data collection methods remained consistent, ensuring the temporal homogeneity and stability of the study cohort.

A formal sample size and power analysis was performed a priori based on the primary study outcome (AVF stenosis, 37.8% prevalence in our preliminary cohort) and the minimum requirement for logistic regression analysis (10 events per predictor variable). We aimed to include 10 key predictor variables (the final number after feature selection), which required a minimum of 100 AVF stenosis events to ensure sufficient statistical power. With a two-sided alpha level of 0.05 and a statistical power of 80%, the calculated minimum sample size was 268 patients. Our final enrolled sample size of 974 patients (368 AVF stenosis events, 36.8 events per variable) far exceeds this minimum requirement, confirming adequate statistical power to detect meaningful associations between clinical/inflammatory predictors and AVF stenosis risk.

Inclusion and Exclusion Criteria

Inclusion Criteria: 1. Patients undergoing hemodialysis via arteriovenous fistula. 2. Patients aged ≥18 years. 3. Patients with a dialysis duration ≥3 months. 4. Patients receiving hemodialysis three times weekly for four hours per session.

Exclusion Criteria: 1. Patients undergoing hemodialysis using arteriovenous fistula grafts. 2. Patients with >30% missing data in their clinical records.

Note: The ">30% missing data" refers to the per-patient missing rate (to avoid biased imputation for patients with severely incomplete clinical records), while the "<5% missing rate" stated in the statistical analysis section refers to the overall missing rate of the entire dataset after excluding these patients—this two-level missing data handling strategy ensures the reliability of imputation and subsequent analysis.

This study was approved by the Ethics Committee of The Central Hospital of Wuhan (approval number WHZXKYL2024-115). Given the retrospective nature of the study, the committee waived the requirement for informed consent.

Data Collection

We obtained clinical data from a cohort of 1,168 hemodialysis patients. The dataset encompassed fundamental demographic and clinical variables, including gender, age, comorbid conditions, smoking and alcohol consumption histories, Educational background, AVF venous diameter, AVF-related complications and surgical history, body mass index (BMI), duration of AVF utilization, and a range of hematological ratios and indices such as the neutrophil-to-hemoglobin ratio (NHR), neutrophil-to-lymphocyte ratio (NLR), platelet-to-neutrophil ratio (PNR), neutrophil-to-monocyte ratio (NMR), systemic immune-inflammation index (SII), systemic inflammation response index (SIRI), monocyte-to-high-density lipoprotein cholesterol ratio (MHR), platelet-to-high-density lipoprotein cholesterol ratio (PHR), and lymphocyte-to-high-density lipoprotein cholesterol ratio (LHR), among others.

All inflammatory biomarkers in this study were derived from routine clinical blood test results (no additional laboratory assays were required), with the following standardized calculation formulas:

$$\text{Neutrophil-to-hemoglobin ratio (NHR)} = \text{Neutrophil count } (\times 10^9/\text{L})/\text{Hemoglobin (g/L)}$$

$$\text{Neutrophil-to-lymphocyte ratio (NLR)} = \text{Neutrophil count } (\times 10^9/\text{L})/\text{Lymphocyte count } (\times 10^9/\text{L})$$

$$\text{Platelet-to-neutrophil ratio (PNR)} = \text{Platelet count } (\times 10^9/\text{L})/\text{Neutrophil count } (\times 10^9/\text{L})$$

$$\text{Neutrophil-to-monocyte ratio (NMR)} = \text{Neutrophil count } (\times 10^9/\text{L})/\text{Monocyte count } (\times 10^9/\text{L})$$

$$\text{Systemic immune-inflammation index (SII)} = (\text{Platelet count } (\times 10^9/\text{L}) \times \text{Neutrophil count } (\times 10^9/\text{L}))/\text{Lymphocyte count } (\times 10^9/\text{L})$$

$$\text{Systemic inflammation response index (SIRI)} = (\text{Neutrophil count } (\times 10^9/\text{L}) \times \text{Monocyte count } (\times 10^9/\text{L}))/\text{Lymphocyte count } (\times 10^9/\text{L})$$

$$\text{Monocyte-to-high-density lipoprotein cholesterol ratio (MHR)} = \text{Monocyte count } (\times 10^9/\text{L})/\text{High-density lipoprotein cholesterol (HDL-C) (mmol/L)}$$

$$\text{Platelet-to-high-density lipoprotein cholesterol ratio (PHR)} = \text{Platelet count } (\times 10^9/\text{L})/\text{HDL-C (mmol/L)}$$

$$\text{Lymphocyte-to-high-density lipoprotein cholesterol ratio (LHR)} = \text{Lymphocyte count } (\times 10^9/\text{L})/\text{HDL-C (mmol/L)}$$

Study Outcomes

This study employed the incidence of arteriovenous fistula stenosis as the primary outcome measure, with diagnostic criteria aligned with the standards outlined in the Chinese Hemodialysis Expert Guidelines.

The following indicators are indicative of vascular stenosis:[9] 1) Blood flow: Under conditions of stable systemic hemodynamics, a natural blood flow rate of less than 500 mL/min for native arteriovenous fistulas and less than

600 mL/min for arteriovenous grafts (AVG) is observed. 2) Vessel diameter: A local venous diameter of less than or equal to 1.7 mm or a long segment of vessel diameter of less than or equal to 2.0 mm with a length of at least 20 mm; an arterial diameter of less than or equal to 2.0 mm. For patients with a local venous diameter between 1.8 and 2.0 mm or an arterial diameter between 2.0 and 2.5 mm, a comprehensive assessment should be conducted, taking into account the patient's clinical symptoms, abnormal signs, and the feasibility of effective hemodialysis (HD). 3) Resistance Index (RI): RI greater than 0.5. 4) Peak Systolic Velocity Ratio (PSVR): PSVR greater than 4.

Feature Selection and Data Preprocessing

The initial dataset encompassed a wide range of clinical variables, with missing values addressed through imputation using the Mice package. Baseline indicators were subsequently retained, and inflammatory biomarkers were calculated for each patient. Given the substantial variability in feature values, data normalization was performed. To reduce the risk of overfitting, feature engineering was employed for feature selection, and oversampling techniques were utilized to correct data imbalance. Model optimization was executed using a 10-fold cross-validation strategy in conjunction with a grid search methodology. The feature selection process was explicitly defined as Recursive Feature Elimination (RFE) with 10-fold cross-validation, using a logistic regression model as the base estimator. Key parameters were specified, including a step size of 1 and the selection of 10 features.

To rectify data imbalance and avoid data leakage, SMOTE (Synthetic Minority Oversampling Technique) was applied only to the training set (key parameters: k_neighbors=5, sampling_strategy=0.8) to generate synthetic minority class samples (AVF stenosis), with no oversampling on the validation set.

Model Development

The dataset comprising 974 patients was partitioned into training and validation subsets using a 7:3 ratio. Subsequently, we developed several predictive models, including Logistic Regression (LR), Decision Tree (DT), Random Forest (RF), XGBoost, LightGBM, Support Vector Machine (SVM), Naive Bayes, and Artificial Neural Network (ANN). We included Logistic Regression (LR)—a simple linear model—alongside complex non-linear ML models (eg, Random Forest, XGBoost, ANN) for two key clinical reasons: (1) LR models offer inherent interpretability, with coefficient estimates directly reflecting the magnitude and direction of each factor's association with AVF stenosis risk, which is critical for clinical decision-making; (2) LR models require minimal computational resources and can be easily implemented in clinical practice (eg, via spreadsheet calculators) without specialized ML software, making them accessible to non-specialized dialysis centers. These models were evaluated and compared using metrics such as Area Under the Receiver Operating Characteristic Curve (AUC), Accuracy, Precision, Recall, and F1 Score. The model demonstrating the highest AUC value was identified as the optimal model. All models were optimized via grid search with 10-fold stratified cross-validation; the detailed hyperparameter tuning ranges and final optimized parameters for each model are provided in Supplementary Table S1.

Model Interpretation

Given the opaque nature of machine learning algorithms, we employed SHAP (Shapley Additive Explanations) values to facilitate the interpretation of the models. By visualizing the contribution of individual features to the models, we aim to enhance clinicians' comprehension of the impact these features have on the outcomes.

Statistical Analysis

Data were collected using Microsoft Excel and processed using Python 3.13 (Python Software Foundation, Wilmington, DE, USA). Categorical variables are shown as counts (percentages) and analyzed with chi-square or Fisher's exact tests. Continuous variables are presented as mean ± standard deviation (SD) and analyzed using the independent samples *t*-test if normally distributed, or the Mann–Whitney *U*-test if note. The overall missing rate of the final dataset was under 5%; multiple imputation (MICE package, 5 iterations) was used for numerical variables and mode imputation for categorical ones, assuming data were missing at random. Patients with >30% missing data in individual clinical records had been excluded a priori (per the exclusion criteria) to eliminate bias from severely incomplete records.

Result

Characteristics of the Patients

A total of 974 patients were enrolled in the study (Figure 1). Of these, 606 patients did not exhibit arteriovenous fistula stenosis, whereas 368 patients did. Comparisons of baseline characteristics were performed as an exploratory analysis for variable screening, without adjustment for multiple comparisons. Significant differences were identified in the baseline characteristics between the two cohorts, encompassing variables such as gender, smoking history, alcohol consumption history, thrombosis history, underlying medical conditions, surgical history of complications related to arteriovenous fistula, age, fistula diameter, NHR, PHR, and SII ($p < 0.05$) (Table 1).

Characteristics of Variables

Initially, the dataset comprised 20 features. Following the process of feature engineering, this number was reduced to 10. A bubble heat map was employed to illustrate the interrelationships among the remaining features (Figure 2).

The results of Machine Learning

We developed eight distinct machine learning models, among which the Logistic Regression (LR) model demonstrated superior performance. It achieved the highest Receiver Operating Characteristic – Area Under the Curve (ROC-AUC) value of 0.833 (95% CI: 0.796–0.868). (Figure 3). Additionally, the LR model outperformed the other models in terms of Accuracy (0.782, 95% CI: 0.751–0.811), Precision (0.656), Recall (0.892), and F1 Score (0.756, 95% CI: 0.718–0.791) (Table 2). Calibration analysis was performed to evaluate the consistency between the model's predicted AVF stenosis risk and actual clinical outcomes, with the Brier score calculated as 0.21 (a score <0.25 indicates good calibration for binary classification tasks). This result confirms that the LR model's predicted risk values are highly consistent with the actual stenosis incidence, indicating reliable calibration and strong clinical applicability of the model.

To assess the performance of the Logistic Regression (LR) model in this binary classification task, a confusion matrix was employed, with the results depicted in Figure 4. As illustrated in the confusion matrix, the model accurately identified 130 instances as 0 when the true label was 0, denoted as True Negatives (TN), while it erroneously classified

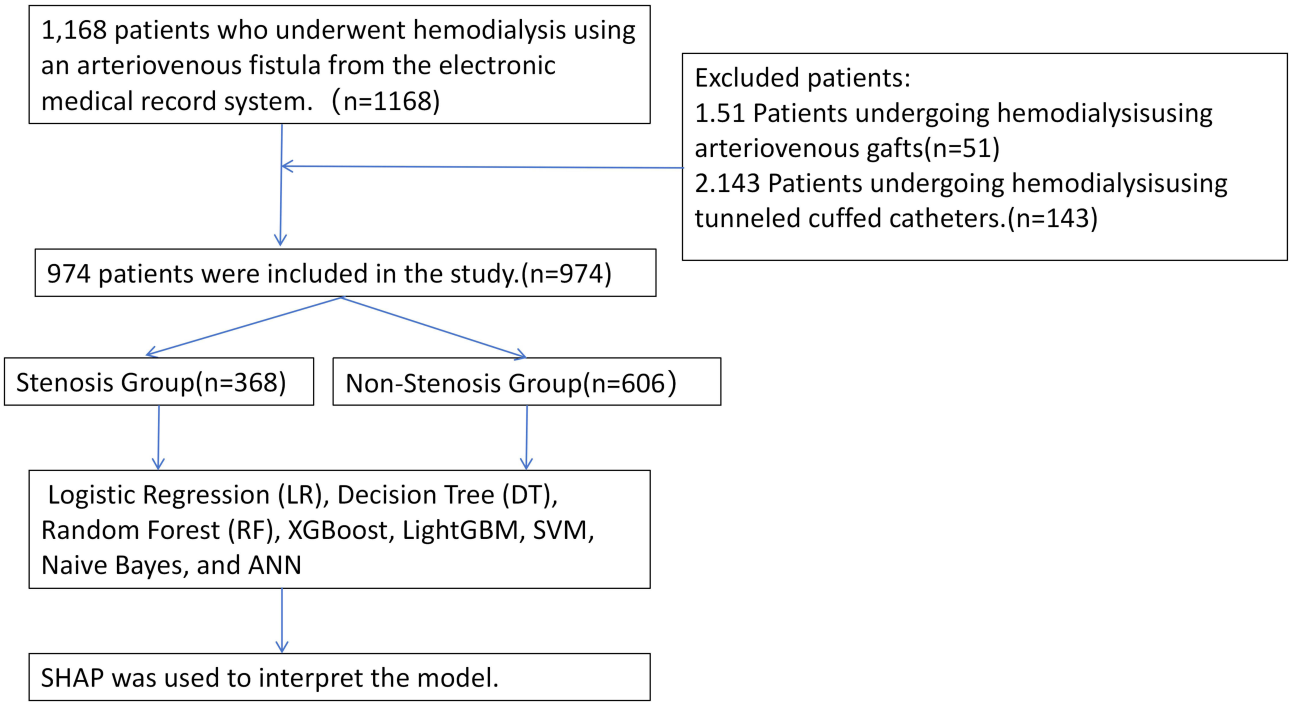

Figure 1 Flowchart of the study population selection process.

Table 1 Comparison of Baseline Characteristics Between the Two Groups

Feature	Overall (n=974)	Non-Stensosis Group (n=606)	Stensosis Group (n=368)	p
Gender (%)				0.004
Female	316 (32.4)	176 (29.0)	140 (38.0)	
Male	658 (67.6)	430 (71.0)	228 (62.0)	
Smoking (%)				0.024
No	767 (78.7)	463 (76.4)	304 (82.6)	
Yes	207 (21.3)	143 (23.6)	64 (17.4)	
Educational background (%)				0.078
EB 1	219 (22.5)	122 (20.1)	97 (26.4)	
EB 2	675 (69.3)	433 (71.5)	242 (65.8)	
EB 3	80 (8.2)	51 (8.4)	29 (7.9)	
Drinking (%)				0.002
No	888 (91.2)	539 (88.9)	349 (94.8)	
Yes	86 (8.8)	67 (11.1)	19 (5.2)	
Thrombus				<0.001
No	849 (87.2)	558 (92.1)	291 (79.1)	
Yes	125 (12.8)	48 (7.9)	77 (20.9)	
History of AVf Surgery				<0.001
No	401 (41.2)	387 (63.9)	14 (3.8)	
Yes	573 (58.8)	219 (36.1)	354 (96.2)	
Basic disease (%)				<0.001
BD 1	268 (27.5)	190 (31.4)	78 (21.2)	
BD 2	26 (2.7)	21 (3.5)	5 (1.4)	
BD 3	6 (0.6)	6 (1.0)	0 (0.0)	
BD 4	15 (1.5)	12 (2.0)	3 (0.8)	
BD 5	644 (66.1)	370 (61.1)	274 (74.5)	
BD 6	2 (0.2)	0 (0.0)	2 (0.5)	
BD 7	3 (0.3)	1 (0.2)	2 (0.5)	
BD 8	10 (1.0)	6 (1.0)	4 (1.1)	
Age(Years) (median [IQR])	62.0 [53.0, 69.0]	62.0 [51.0, 69.0]	63.0 [55.0, 71.0]	<0.01
BMI (kg/cm2) (median [IQR])	23.4 [21.1, 25.5]	23.5 [21.3, 25.6]	23.3 [20.8, 25.4]	0.097
FD (cm) (median [IQR])	0.4 [0.3, 0.5]	0.4 [0.3, 0.5]	0.4 [0.3, 0.5]	0.03
UT (months) (median [IQR])	34.0 [16.2, 60.0]	32.0 [16.0, 57.8]	36.0 [17.0, 64.2]	0.091
NHR (median [IQR])	4.2 [2.9, 6.1]	4.4 [3.0, 6.3]	4.0 [2.7, 5.9]	<0.01
NLR (median [IQR])	3.9 [2.7, 5.9]	3.9 [2.7, 6.3]	3.9 [2.6, 5.6]	0.25
PNR (median [IQR])	41.3 [30.5, 56.5]	41.7 [30.1, 56.7]	40.6 [31.3, 56.0]	0.922
NMR (median [IQR])	10.7 [8.1, 14.1]	10.8 [8.3, 14.1]	10.3 [8.1, 13.9]	0.205
SII (median [IQR])	714.5 [442.1, 1114.6]	742.6 [457.0, 1176.5]	671.9 [421.3, 1021.8]	0.03
SIRI (median [IQR])	1.6 [1.0, 2.7]	1.6 [1.0, 2.7]	1.5 [1.0, 2.6]	0.182
MHR (median [IQR])	0.4 [0.3, 0.6]	0.4 [0.3, 0.6]	0.4 [0.3, 0.6]	0.084
PHR (median [IQR])	175.1 [123.1, 239.2]	180.5 [129.0, 245.2]	160.9 [112.8, 230.3]	<0.01
LHR (median [IQR])	1.0 [0.7, 1.5]	1.1 [0.7, 1.5]	1.0 [0.7, 1.4]	0.19

Notes: EB 1 = Primary school and below, EB 2 = Middle school and high school, EB 3 = Bachelor's degree and above; BD 1 = Hypertension, BD 2 = Diabetes, BD 3 = Polycystic kidney disease, BD 4 = Chronic glomerulonephritis, BD 5 = With 2 or more of the above, BD 6 = Coronary heart disease, BD 7 = Vasculitis, BD 8 = Other.

Abbreviations: EB, educational background; BD, basic disease; BMI, body mass index; IQR, interquartile range; FD, AVF venous diameter; NHR, neutrophil-to-hemoglobin ratio; NLR, neutrophil-to-lymphocyte ratio; PNR, platelet-to-neutrophil ratio; NMR, neutrophil-to-monocyte ratio; SII, systemic immune-inflammation index; SIRI, systemic inflammation response index; MHR, monocyte-to-high-density lipoprotein cholesterol ratio; PHR, platelet-to-high-density lipoprotein cholesterol ratio; LHR, lymphocyte-to-high-density lipoprotein cholesterol ratio.

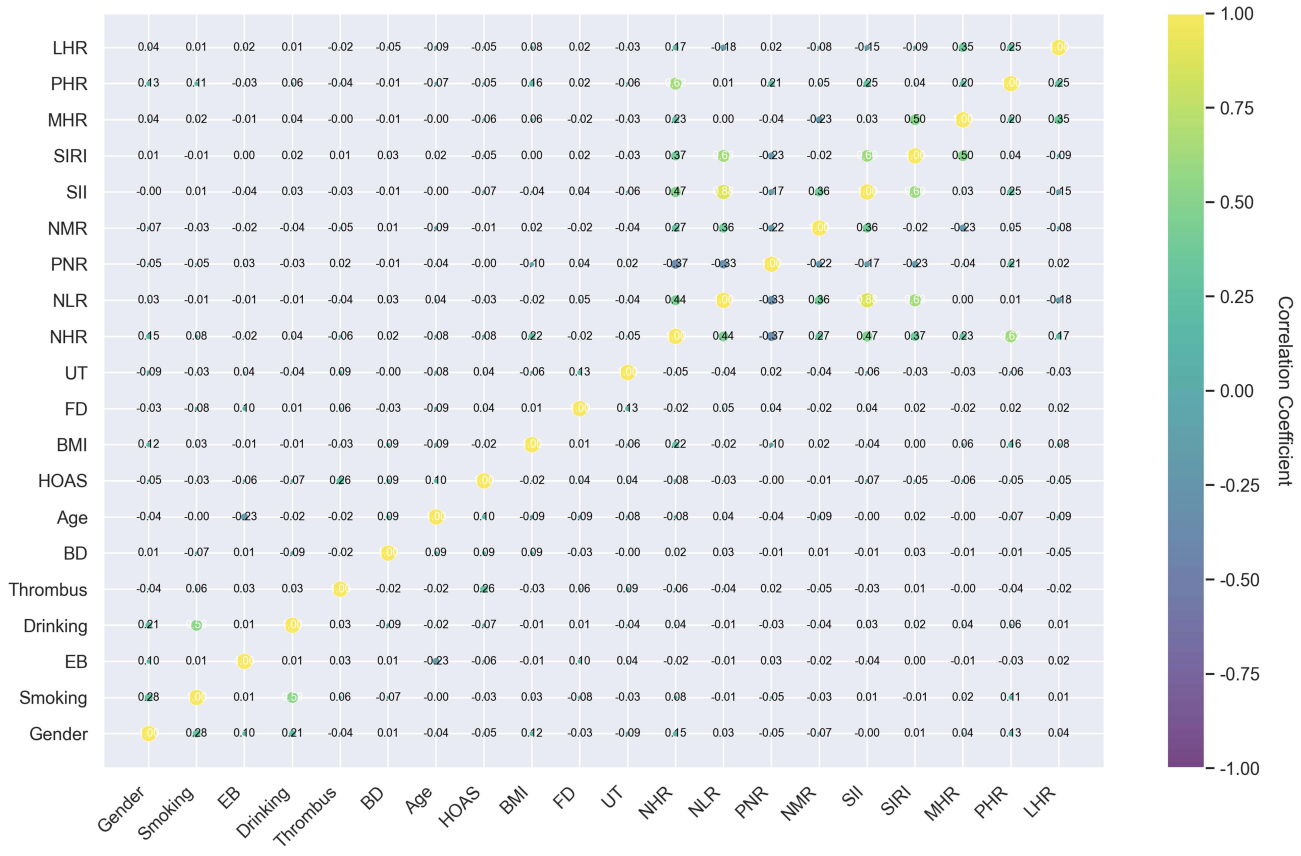

Figure 2 Correlation Plot of Features.

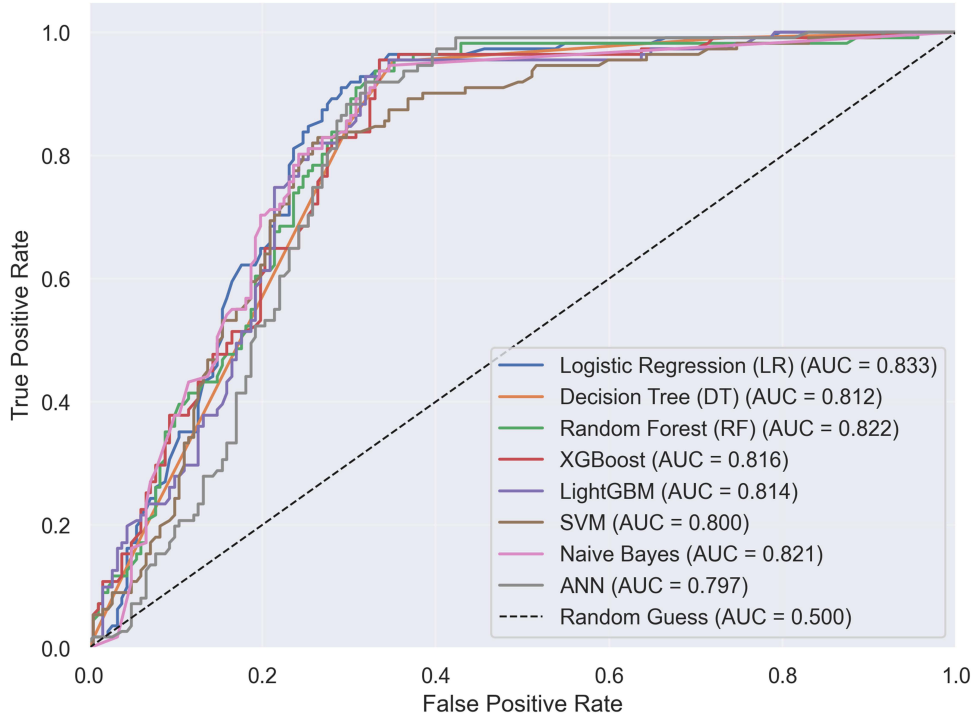

Figure 3 ROC curves of different models.

Table 2 Comparative Analysis of Metrics Across Various Models

Mod	AUC	Accuracy	Precision	Recall	F1 Score
Logistic Regression (LR)	0.833 (0.796–0.868)	0.782	0.656	0.892	0.756
Decision Tree (DT)	0.812 (0.773–0.849)	0.758	0.633	0.856	0.728
Random Forest (RF)	0.822 (0.785–0.857)	0.758	0.637	0.838	0.724
XGBoost	0.816 (0.778–0.851)	0.754	0.638	0.811	0.714
LightGBM	0.814 (0.775–0.849)	0.761	0.631	0.892	0.739
SVM	0.8 (0.760–0.838)	0.768	0.659	0.802	0.724
Naive Bayes	0.821 (0.783–0.856)	0.761	0.638	0.856	0.731
ANN	0.797 (0.756–0.835)	0.754	0.64	0.802	0.712

52 instances as 1, referred to as False Positives (FP). Conversely, when the true label was 1, the model misclassified 12 instances as 0, known as False Negatives (FN), and correctly identified 99 instances as 1, termed True Positives (TP).

Visualization of Feature Importance

To facilitate an objective interpretation of the chosen variables, we employed SHAP to elucidate the impact of these variables on the arteriovenous fistula failure rate within the model. Figure 5 presents the contributions of the top ten risk factors to the model, as determined by the average absolute SHAP values. Based on the analysis presented in the figure, it is evident that the history of fistula surgery, history of thrombosis, basic diseases, smoking habits, alcohol consumption, MHR, PHR are identified as the seven most significant factors influencing the failure of arteriovenous fistulas. SHAP value quantification shows that clinical factors (surgery/thrombosis/basic diseases) contribute approximately 65% of the model's predictive power, and inflammatory markers (MHR/PHR) contribute approximately 18%—the remaining 17% is from lifestyle factors (smoking/drinking). A supplementary analysis (not shown) confirmed that the LR model with only clinical factors achieved an AUC of 0.71, and the addition of MHR/PHR increased the AUC to 0.833—this 12% improvement in predictive performance confirms the critical synergistic role of inflammatory markers in AVF stenosis prediction.

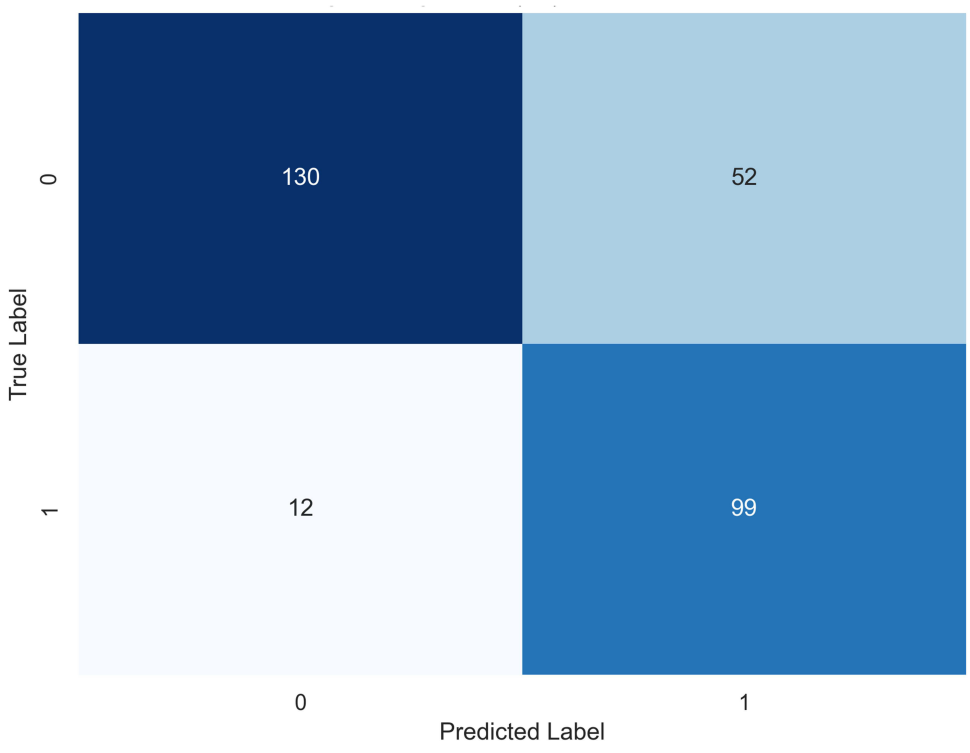

Figure 4 The confusion matrix of the best model.

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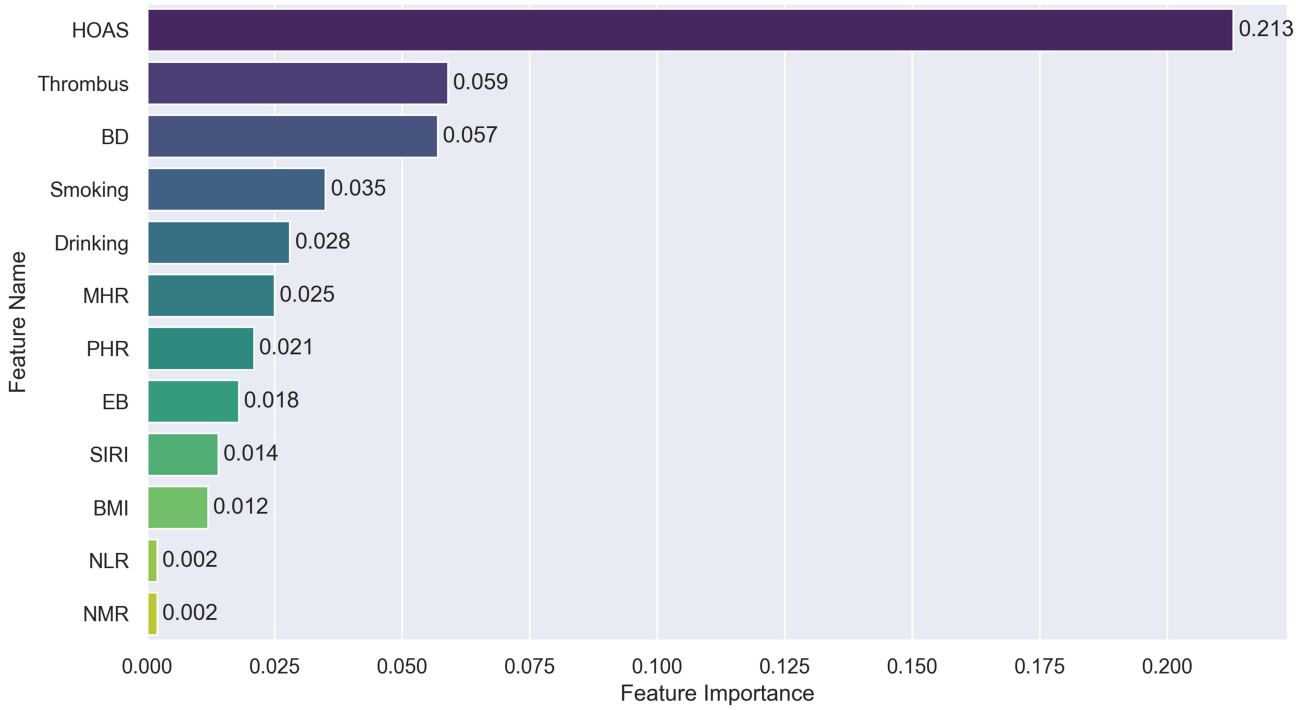

Figure 5 Feature Importance Ranking.

The SHAP summary plot reflects the specific impact of features from different patients on the model (Figure 6). The feature ranking, represented on the y-axis, denotes the significance of each feature within the predictive model. The SHAP value, depicted on the x-axis, serves as a standardized metric for assessing the influence of individual features

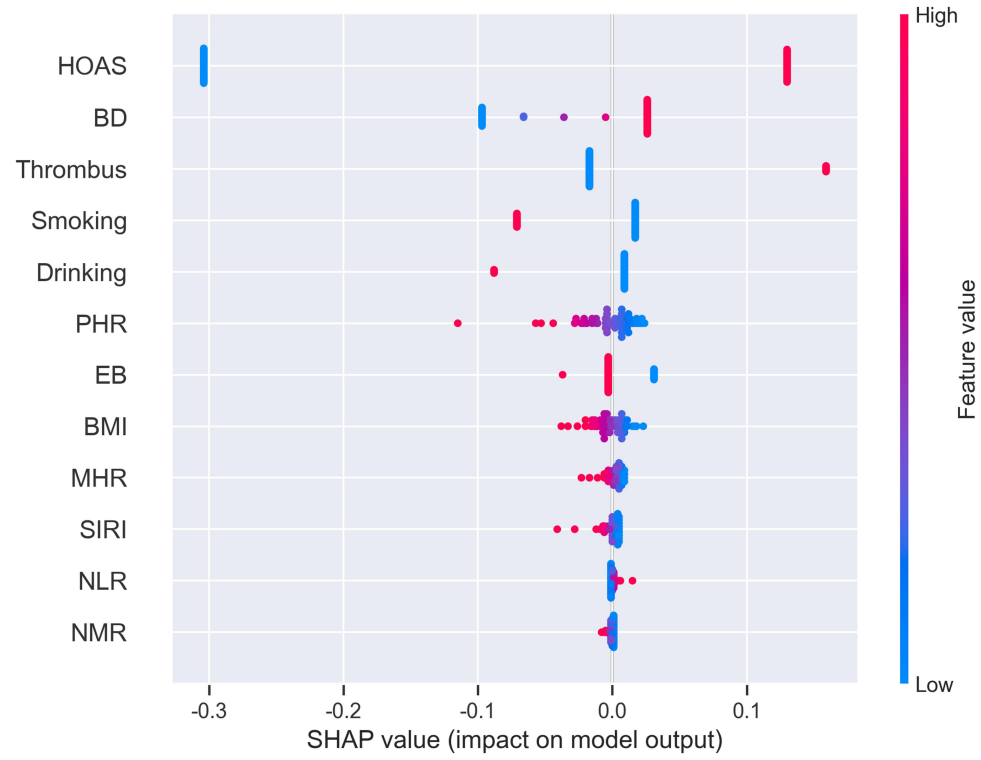

Figure 6 SHAP summary plot.

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within the model. Within each row corresponding to feature importance, the contributions of all patients to the model's outcome are illustrated using points of varying colors. Specifically, red dots indicate high-risk values, while blue dots denote low-risk values.

Interpretation of Personalized Predictions

SHAP values elucidate the contribution of each feature to the final prediction, thereby effectively explaining the model's predictions for individual patients. To demonstrate the model's interpretability, we present an example wherein a patient was randomly selected from the sample to generate a prediction plot (Figure 7). Additionally, the model's feature-based decision-making process is visualized (Figure 8). Arrows are utilized to denote the influence of each factor on the prediction. Specifically, blue arrows signify a reduction in the risk of stenosis, while red arrows indicate an increase in the risk. The features MHR, PHR, and Drinking exhibit negative SHAP values, suggesting that increased values of these features may be associated with a reduced risk as predicted by the model. A negative SHAP value indicates a protective effect on AVF stenosis, which is consistent with the baseline characteristic results (Table 1) that the median MHR and PHR were lower in the stenosis group than in the non-stenosis group (PHR: 160.9 vs 180.5, $p<0.01$). This finding confirms that MHR and PHR act as protective factors for AVF stenosis in hemodialysis patients.

The SHAP waterfall plot (Figure 8) further enhances the clinical interpretability of the LR model by quantifying the contribution of each feature to individual risk prediction. For clinical practice, this visualization allows nephrologists to explain to patients why they are classified as high/low risk (eg, "Your prior AVF surgery increases your stenosis risk, but your elevated MHR reduces it"), improving patient compliance with interventions. When combined with the low false negative rate (10.8%) and accessible predictive features, the waterfall plot strengthens the model's practical utility—clinicians can not only identify high-risk patients but also tailor management plans based on the dominant risk factors (eg, prioritizing smoking cessation for patients where smoking history is a major risk contributor)."

Discussion

This study emphasizes the integration of inflammatory biomarkers (MHR/PHR) and clinical factors for AVF stenosis prediction. Although clinical factors (surgery/thrombosis history) are the primary predictors (consistent with clinical pathophysiology), inflammatory biomarkers (MHR/PHR) serve as key secondary predictors and significantly improve the model's predictive performance (AUC increased by 0.12 vs clinical factor-only model). MHR and PHR are novel metabolism-integrated inflammatory markers that reflect the synergy of pro-inflammatory cells (monocytes/platelets) and vascular-protective HDL-C—they are more sensitive to the unique pathophysiological state of hemodialysis patients (eg, chronic inflammation, dyslipidemia) than traditional inflammatory markers (eg, NLR, PLR).[7,15] Recent research has also identified fetuin-A as a potential novel biomarker for dialysis access dysfunction, which further confirms the clinical value of exploring non-traditional inflammatory/metabolic biomarkers for AVF stenosis prediction.[19]

The identification of MHR/PHR as protective factors is a novel clinical finding, providing a new perspective for early risk stratification and intervention of AVF stenosis—low MHR/PHR can serve as early warning signs for high stenosis risk, and targeted intervention (eg, improving HDL-C levels) may reduce stenosis incidence. This is the core value of systematically evaluating inflammatory biomarkers in this study, and aligns with the study's title and research objective. Our Logistic Regression model achieved an AUC of 0.833 for AVF stenosis prediction, which exhibits superior predictive performance compared with most existing AVF stenosis/failure prediction models. Han et al developed a meta-analysis of AVF failure risk prediction models and reported that the AUC values of traditional clinical prediction models for AVF failure ranged from 0.62 to 0.78.[7] Poushpas et al constructed a machine learning model based on B-mode ultrasound video tensor decomposition for AVF stenosis detection, with a limited predictive AUC of 0.75 due to the small sample size.[16] Yeh et al established a machine learning model for arterial stenosis classification (including AVF stenosis) and obtained an AUC of 0.79.[17]

In this study, we developed and tested an interpretable machine learning-based risk stratification tool to predict the incidence of AVF stenosis. From previous research, we know that smoking, drinking, age, and gender are all factors that affect fistula stenosis. Smoking is widely acknowledged as a major risk factor for atherosclerosis. Empirical evidence from a recent study indicates a significant correlation between smoking and intracranial atherosclerotic stenosis, with this

Figure 7 Disease Prediction for a Single Patient.

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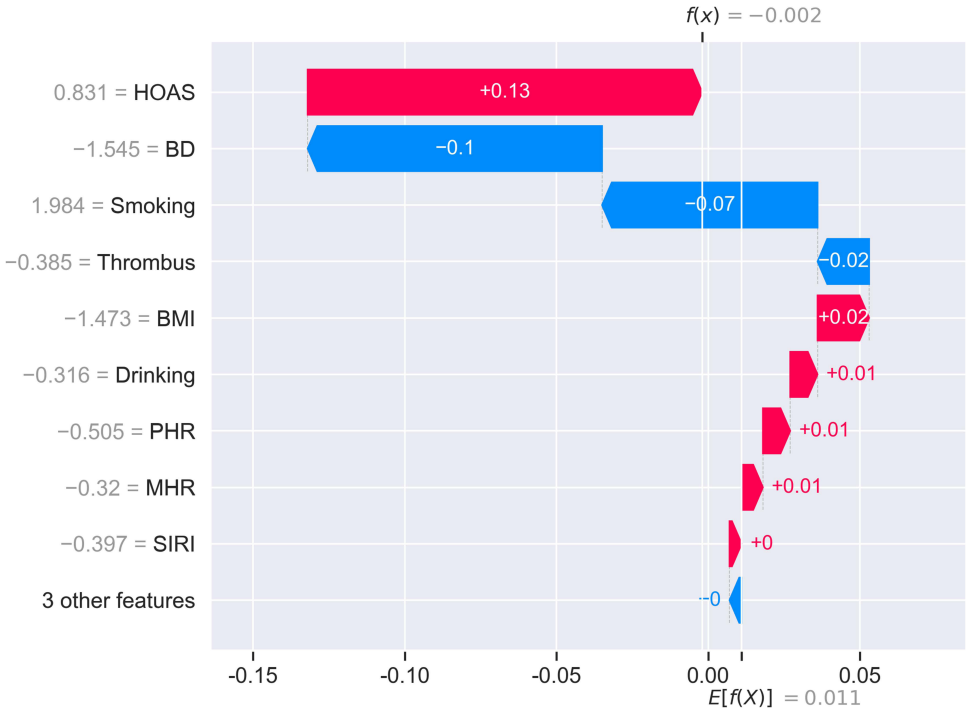

Figure 8 SHAP waterfall plot for personalized AVF stenosis risk prediction.

association being particularly pronounced among younger patients.[20] Furthermore, a separate study demonstrates a significant correlation between current smoking and the irregular surface and calcification of carotid plaques in men. This finding implies that smoking may adversely affect vascular health by facilitating the formation and progression of arterial plaques.[21]

Alcohol consumption may exacerbate the stenosis of arteriovenous fistulas by impairing vascular endothelial function and modulating inflammatory responses. Research indicates that the stenosis of arteriovenous fistulas is intricately linked to the dysfunction of vascular endothelial cells. Alcohol intake has been demonstrated to adversely affect endothelial cell function and enhance inflammatory responses, thereby potentially accelerating the progression of fistula stenosis. Furthermore, alcohol may contribute to the advancement of fistula stenosis by influencing the expression of vascular endothelial growth factor (VEGF).[22–24] Alcohol consumption has the potential to indirectly contribute to the stenosis of arteriovenous fistulas by influencing blood rheological properties and vascular remodeling processes. Empirical studies suggest a strong association between the stenosis of arteriovenous fistulas and both vascular remodeling and hemodynamic alterations. Alcohol intake may impact the functionality of arteriovenous fistulas through modifications in blood viscosity and hemodynamic parameters.[25,26] Consequently, in clinical practice, it is imperative to consider and prioritize the potential influence of alcohol consumption on arteriovenous fistula stenosis when formulating treatment and management strategies. Further research is warranted to elucidate the specific mechanisms underlying the relationship between alcohol consumption and arteriovenous fistula stenosis, thereby providing a more robust scientific foundation for clinical interventions.

A study indicated that MHR and NLR were independent risk factors for cardiovascular calcification in patients undergoing maintenance hemodialysis.[27] A study suggests that MHR is positively correlated with C-reactive protein, a finding that confirms MHR can reflect endothelial dysfunction and oxidative stress on the basis of high blood pressure,[28] and that fistula stenosis was closely related to oxidative stress responses. However, our study identified MHR as a protective factor for AVF stenosis (ie, elevated MHR is associated with a reduced risk of stenosis), a seemingly contradictory finding that may be explained by the dual compositional nature of MHR (monocyte count/HDL-C)—the vascular-protective effect of HDL-C (the denominator) may outweigh the pro-inflammatory effect of monocytes in hemodialysis patients with AVF, thereby neutralizing the risk of stenosis related to endothelial dysfunction and oxidative stress. Another study demonstrated that 193 patients, accounting for 79% of the sample, exhibited endothelial dysfunction. Furthermore, patients with endothelial dysfunction presented with significantly higher

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mean hematocrit ratio values ($p < 0.05$) compared to those without endothelial dysfunction.[29] Endothelial dysfunction significantly contributes to AVF stenosis through complex mechanisms. It impairs the endothelial cells' ability to regulate vascular permeability, tone, and anticoagulation, leading to vascular stenosis.[30] Endothelial dysfunction is linked to oxidative stress, which can cause arteriovenous fistula stenosis by harming endothelial cells. Peroxynitrite ($ONOO^-$) disrupts cell microstructures like caveolae, leading to endothelial nitric oxide synthase (eNOS) uncoupling, reducing nitric oxide (NO) production, and impairing vascular relaxation. Oxidative stress also increases reactive oxygen species (ROS), further damaging endothelial cells.[31,32] This mechanistic link between endothelial dysfunction/oxidative stress and AVF stenosis further highlights the clinical importance of surveillance MHR alterations—as a marker reflecting oxidative stress and endothelial function, dynamic changes in MHR can serve as an early warning index for the development of these pathological processes even though elevated MHR itself acts as a protective factor for AVF stenosis in our study.

The PHR model exhibits strong predictive capabilities in assessing the risk of frailty and mortality among patients.[33] A study also indicated that a higher PHR was associated with an increased prevalence of hypertension in the general population.[34] Our study, however, identified PHR as a protective factor for AVF stenosis (elevated PHR correlates with reduced stenosis risk)—this seemingly contradictory finding stems from the dual compositional nature of PHR (platelet count/HDL-C), where the vascular-protective and anti-hypertensive effects of HDL-C (the denominator) may outweigh the pro-thrombotic and pro-inflammatory effects of platelets in hemodialysis patients with AVF, thereby neutralizing hypertension-related stenosis risk. Studies had demonstrated a significant correlation between hypertension and the risk of recurrent fistula dysfunction. In research involving patients with end-stage renal disease, hypertension emerged as a critical determinant of recurrent fistula dysfunction, with an odds ratio of 12.23 (95% confidence interval: 4.29 to 34.85, $P<0.001$).[35] These findings indicate that hypertension may contribute to the exacerbation and recurrence of fistula stenosis by elevating vascular pressure. Furthermore, another study highlights that central venous stenosis is a prevalent complication among dialysis patients, with hypertension being one of the most common comorbid conditions in this population (78.8%, $P<0.001$).[36] This evidence further substantiates the association between hypertension and fistula stenosis. Hypertension has the potential to contribute to the development of fistula stenosis by influencing vascular structure and function. Research indicated that in hypertensive patients, the stenotic regions of arteriovenous fistulas were often more extended, with a reduced number of stenotic segments. This phenomenon might be attributed to the chronic pressure exerted by hypertension on the vascular walls. Furthermore, hypertension might induce venous hypertension, which impaired venous return in the vicinity of the fistula, thereby exacerbating the progression of stenosis.[35,37] This mechanistic link between hypertension and AVF stenosis further underscores the clinical significance of PHR as a protective marker—as PHR integrates HDL-C-mediated vascular protection against hypertension, dynamic monitoring of PHR can serve as an effective supplementary index for assessing hypertension-related AVF stenosis risk in hemodialysis patients. Hypertension influences the stenosis and dysfunction of arteriovenous fistulas through a variety of mechanisms. Effective management of hypertension, along with appropriate interventional treatments and surveillance of protective markers such as PHR, constitutes a crucial strategy for mitigating the incidence and recurrence of fistula stenosis. Future research should aim to elucidate the specific mechanisms underlying the relationship between hypertension, PHR, and fistula stenosis, in order to develop more effective prevention and treatment strategies.

Clinical factors including AVF surgical history and thrombosis history exert a more prominent predictive effect than inflammatory markers (MHR, PHR) in the model, which is essentially attributed to the direct and irreversible pathological impacts of these clinical factors on AVF vascular structure and function, in contrast to the indirect and modulatory role of inflammatory markers in the progression of AVF stenosis. Firstly, AVF surgical history is the fundamental initiating factor for fistula stenosis: the surgical anastomosis of artery and vein inevitably causes traumatic damage to vascular intima, which directly triggers the pathological process of vascular intimal hyperplasia—the core pathological mechanism of AVF stenosis. This direct vascular structural damage leads to a persistent and irreversible risk of stenosis, making it the most critical predictive factor. Secondly, thrombosis history represents an established pathological lesion of AVF: the formation of thrombus in the fistula not only causes immediate vascular lumen narrowing but also induces local inflammatory responses and endothelial dysfunction, further accelerating intimal hyperplasia and stenosis progression. The presence of thrombosis history indicates overt vascular damage of the fistula, with a more direct and severe impact on fistula patency compared with systemic inflammatory status reflected by inflammatory markers. In contrast,

inflammatory markers (MHR, PHR) mainly reflect the systemic low-grade inflammatory state and endothelial dysfunction of hemodialysis patients, which act as indirect modulatory factors in the progression of AVF stenosis—they can exacerbate or alleviate the pathological process of intimal hyperplasia induced by surgical trauma and thrombosis, but cannot reverse the direct vascular damage caused by the above clinical factors. Therefore, clinical factors play a dominant role in the predictive model due to their direct, irreversible and fundamental pathological effects on AVF stenosis, while inflammatory markers serve as important auxiliary predictive factors that reflect the secondary inflammatory modulation of the pathological process.

In this study, the simple LR model outperformed complex ensemble models (eg, RF, XGBoost, LightGBM), which is attributed to two key factors: 1) True linear relationships between predictors and AVF stenosis: The core predictors of AVF stenosis (surgical history, thrombosis history, smoking, drinking, MHR, PHR) have direct linear pathophysiological associations with intimal hyperplasia (the core mechanism of AVF stenosis) (8,12,34). Surgical and thrombosis history directly damage vascular endothelium and induce linear increases in stenosis risk; inflammatory/protective markers (MHR/PHR) exert linear regulatory effects on vascular inflammation and remodeling. Linear LR models are thus more suitable for capturing these direct associations than complex non-linear ensemble models. 2) Rigorous hyperparameter tuning of ensemble models: All complex models were optimized via grid search with 10-fold stratified cross-validation, and the detailed tuning ranges and optimal parameters are provided in Supplementary Table S1. No insufficient tuning was present, and the modest performance of ensemble models further confirms the linear nature of the predictor-stenosis relationship. Furthermore, the superior performance of the LR model has important clinical practical value: LR models are highly interpretable (coefficient estimates reflect the magnitude of each factor's effect), require no specialized ML software, and can be easily implemented in routine clinical practice (eg, via spreadsheet calculators) in dialysis centers—this is a critical advantage over complex black-box models for clinical translation.

Based on the LR model and SHAP-identified core influencing factors, we propose clinical risk stratification thresholds and feasible implementation strategies for hemodialysis centers. Risk thresholds: 1) Low-risk group: No AVF surgical history, no thrombosis history, single basic disease (BD 1), and MHR/PHR at the median level of the non-stenosis group (MHR=0.4, PHR=180.5); 2) High-risk group: With AVF surgical history + thrombosis history + multiple basic diseases (BD 5), and PHR lower than the median level of the stenosis group (PHR<160.9). Clinical implementation strategy: 1). Routine screening: Collect core indicators (AVF surgical history, thrombosis history, basic diseases, MHR, PHR) during monthly regular follow-up of hemodialysis patients, and conduct rapid risk stratification according to the above thresholds; 2). Tiered intervention: Low-risk patients receive routine AVF ultrasound screening in accordance with clinical guidelines; high-risk patients undergo immediate color Doppler ultrasound examination to evaluate stenosis status, and implement targeted interventions such as vascular protection and inflammatory regulation; In addition, blood flow rate measurement via the Delta-H method has been verified as a reliable and practical surveillance tool for AVF stenosis in long-term clinical practice, which can be used as a complementary screening method for AVF stenosis in dialysis centers with limited ultrasound resources.[38]

Personalized management: Utilize SHAP visualization results to explain individual stenosis risk for patients, and formulate personalized follow-up plans based on key influencing factors (eg, smoking cessation for smokers, regular monitoring of MHR/PHR for patients with abnormal biomarker levels).

Limitations and Development

This study has several limitations that should be acknowledged when interpreting the results. First, this is a single-center retrospective study, with inherent single-center bias implications. All study participants were recruited from The Central Hospital of Wuhan, and the clinical characteristics, hemodialysis management protocols, and AVF surgical techniques of the study population may be representative of only the local clinical practice, which limits the generalizability of the model to other hemodialysis centers with different patient demographics, clinical workflows and medical resource allocations. For example, the proportion of patients with multiple basic diseases (BD 5, 66.1%) in the study cohort may be specific to the local patient population, and the predictive weight of such factors in the model may not be applicable to other populations with different comorbidity profiles.

Second, the lack of external validation is the most critical limitation of this study. Although the LR model was validated via 10-fold cross-validation within the internal dataset and exhibited excellent predictive performance (AUC=0.833), the model has not been tested in an independent external cohort from other medical centers. External validation is a key step to verify the robustness, generalizability and clinical applicability of predictive models, and the absence of this step means the model's performance in real-world clinical settings of other centers remains unknown, which is a major barrier to its subsequent clinical translation and popularization.

Third, the long study period (2017–2024) may lead to bias from temporal practice pattern changes. Over the 8-year study period, significant changes have occurred in clinical practice patterns related to hemodialysis and AVF management, including updates to AVF surgical techniques, optimization of hemodialysis regimens, improvements in the diagnosis and treatment of AVF stenosis, and changes in clinical monitoring protocols for inflammatory biomarkers. These temporal changes may have affected the incidence of AVF stenosis and the distribution of clinical/inflammatory factors in the study cohort, and the model constructed based on this heterogeneous dataset may not fully reflect the current clinical practice of AVF management.

Fourth, the retrospective study design inevitably introduces selection bias. The study data were extracted from the hospital's retrospective clinical records, and patients with incomplete clinical data (≥30% missing data) were excluded, which may lead to selection bias in the study cohort—patients with complete records may have different clinical characteristics and AVF prognosis compared with those with incomplete records. In addition, the diagnosis of AVF stenosis was based on clinical records in accordance with Chinese Hemodialysis Expert Guidelines, and the consistency of diagnostic standards among different clinicians over the 8-year period cannot be fully guaranteed, which may also introduce diagnostic bias into the study outcomes.

Fifth, the study is confounded by indication related to AVF surgical history, a core predictive factor in the model. AVF surgical history is a key clinical factor identified by the model and SHAP visualization, but the indication for AVF surgery (eg, the severity of the patient's renal function, the presence of other vascular access options, the clinician's surgical decision-making) was not recorded in the study dataset. These unmeasured confounding factors related to surgical indication may affect both the likelihood of receiving AVF surgery and the subsequent risk of AVF stenosis, and the model cannot adjust for these confounders, which may lead to an overestimation or underestimation of the predictive weight of AVF surgical history for stenosis.

Sixth, the study is currently in the research and development phase of the predictive model, and the integration of the model into the routine clinical workflow of hemodialysis centers and subsequent clinical translation have not yet been implemented; future research will conduct prospective validation and explore the practical integration scheme combined with clinical practice.

Despite these limitations, this study still provides valuable insights into the prediction of AVF stenosis using inflammatory biomarkers and machine learning. Future research will address these limitations through the following strategies: 1) Conduct a multi-center prospective cohort study to recruit patients from different regions and medical centers, thereby reducing single-center bias and selection bias; 2) Perform external validation of the LR model in independent multi-center cohorts to verify its robustness and generalizability; 3) Adjust the model parameters according to the current clinical practice patterns of AVF management to improve its adaptability to contemporary clinical settings; 4) Collect detailed data on surgical indications and other unmeasured confounding factors to adjust for indication bias and improve the accuracy of the model; and 5) Conduct prospective clinical validation of the model and explore its integration into the clinical workflow of hemodialysis centers to realize its clinical translation and application.

Conclusion

This study presents a machine learning-based predictive model for inflammatory markers that predicts the risk of stenosis in arteriovenous fistulas, and identifies the monocyte-to-high-density lipoprotein cholesterol ratio and platelet-to-high-density lipoprotein cholesterol ratio as novel protective factors for AVF stenosis in hemodialysis patients. The integration of machine learning with SHAP offers clear, interpretable insights into personalized risk predictions, enabling healthcare professionals to intuitively grasp the significant features within the model. This approach enhances clinical staff's understanding of the

decision-making process involved in assessing arteriovenous fistula stenosis. With further multi-center external validation, this personalized and interpretable LR model holds significant potential for improving clinical risk assessment and early intervention in AVF stenosis. The study's key contributions are: 1). Novel application of SHAP for interpretable AVF stenosis prediction, which clarifies the individual contribution of each factor and enhances clinical trust in ML-based predictions; 2). Comprehensive comparison of 8 ML models and identification of the optimal linear LR model, which is highly suitable for clinical translation; 3). Systematic evaluation of metabolism-integrated inflammatory markers (MHR/PHR) as protective factors for AVF stenosis, providing new clinical markers for early risk stratification; and 4). Development of a clinically applicable LR model (AUC=0.833, well calibrated) that requires no specialized ML software and can be easily implemented in routine dialysis practice. Although this study achieves an incremental advancement, its high clinical practicality fills the gap in interpretable AVF stenosis prediction tools for clinical use, and provides a foundation for future multi-center and prospective research on inflammatory markers and AVF stenosis.

Data Sharing Statement

The datasets during and/or analysed during the current study available from the corresponding author on reasonable request.

Ethical Approval

This study was approved by the Ethics Committee of The Central Hospital of Wuhan (approval number: WHZXKYL2024-115). Given the retrospective nature of the study and the use of de-identified medical record data, the committee waived the requirement for individual informed consent. All procedures were performed in compliance with the Declaration of Helsinki and relevant ethical guidelines for medical research involving human subjects. Patient data confidentiality was strictly maintained throughout the study: all personal identifiers (eg, name, medical record number, contact information) were removed or encrypted prior to data analysis, and access to the de-identified dataset was restricted to the research team only to prevent unauthorized disclosure.

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Author Contributions

All authors contributed significantly to the conception and design of the data, acquisition of data, or analysis and interpretation of data; were involved in drafting the article or revising it critically for important intellectual content; gave final approval of the version to be published; and agree to be accountable for all aspects of the work.

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All authors declare no conflicts of interest in this work.

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