Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

A combined model of monocyte percentage and albumin predicts arteriovenous fistula maturation failure in hemodialysis patients: a retrospective cohort study

Lina Yin
The Third People's Hospital of Chengdu (Affiliated Hospital of Southwest Jiaotong University, Southwest Jiaotong University

Xiaowei Song
The Third People's Hospital of Chengdu (Affiliated Hospital of Southwest Jiaotong University, Southwest Jiaotong University

Bo Yang
The Third People's Hospital of Chengdu (Affiliated Hospital of Southwest Jiaotong University, Southwest Jiaotong University

Lizhu Jin
The Third People's Hospital of Chengdu (Affiliated Hospital of Southwest Jiaotong University, Southwest Jiaotong University

Dan Wu
The Third People's Hospital of Chengdu (Affiliated Hospital of Southwest Jiaotong University, Southwest Jiaotong University

Ying Shu
138081858940163.com

The Third People's Hospital of Chengdu (Affiliated Hospital of Southwest Jiaotong University, Southwest Jiaotong University

---

Research Article

Keywords: arteriovenous fistula, maturation failure, monocyte percentage, albumin, prediction model, hemodialysis

Posted Date: April 10th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-9154443/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: No competing interests reported.

Abstract

Background

Autologous arteriovenous fistula (AVF) maturation failure remains a significant challenge in hemodialysis patients. This study aimed to develop and validate a prediction model for AVF maturation failure using monocyte percentage and albumin, two routine laboratory parameters reflecting inflammation and nutritional status.

Methods

We retrospectively analyzed 160 patients undergoing first-time AVF creation. Multivariable logistic regression identified independent predictors. Model performance was assessed by area under the curve (AUC), calibration plots, and decision curve analysis. Internal validation was performed using bootstrap resampling (1000 replicates).

Results

Monocyte percentage (OR = 1.61, 95%CI 1.31–1.98) and albumin (OR = 0.84, 95%CI 0.77–0.91) were independent predictors. The combined model showed good discrimination (AUC = 0.811, 95%CI 0.745–0.876) with excellent calibration (mean absolute error = 0.048). At the optimal cutoff of 0.328, the model achieved a sensitivity of 86.2% and specificity of 70.6%. Decision curve analysis demonstrated a positive net benefit (0.214), exceeding both "treat all" and "treat none" strategies. Bootstrap validation confirmed model stability (optimism bias = 0.0011).

Conclusions

The monocyte percentage-albumin combined model provides a simple, cost-effective tool for predicting AVF maturation failure, with good discrimination, calibration, and clinical utility. This two-biomarker approach may facilitate preoperative risk stratification and individualized management to improve AVF maturation outcomes.

1. Introduction

Chronic kidney disease (CKD) is a global health burden affecting more than 800 million individuals, marked by a progressive deterioration that often leads to end-stage kidney disease (ESKD)[1]. Renal replacement therapy is essential for the treatment of ESKD[2]. The global population receiving renal replacement therapy currently exceeds 2.5 million and is projected to reach approximately 5.4 million by 2030[3]. This imposes a substantial burden on both patients and healthcare systems. Hemodialysis remains the predominant form of renal replacement therapy [4], which relies on vascular access for its

maintenance. Drawing from recommendations in certain international guidelines, the expert panel suggests that autologous arteriovenous fistula (AVF) should be the preferred option for long-term vascular access due to its low risk of fatal infection, extended usability, and reduced incidence of clinical adverse events[5]. Establishing and preserving a reliable AVF serves as the "lifeline" for effective treatment in maintenance hemodialysis (MHD) patients. The reported maturation rate of AVFs at six months ranges between 33% and 62% [6].AVF failure to mature presents a substantial clinical burden, leading to compromised dialysis quality, elevated hospital readmission rates, and the frequent need for subsequent surgical or endovascular interventions [7]. Inflammation and nutritional status are increasingly recognized as key determinants of AVF maturation[8]. Monocytes, as central players in the inflammatory response, infiltrate the vessel wall and release pro-inflammatory cytokines that impair vascular remodeling [9]. Conversely, hypoalbuminemia reflects malnutrition and chronic inflammation, and low albumin levels have been consistently associated with AVF maturation failure [10]. Several prediction models for AVF maturation have been developed in recent years, but many rely on complex combinations of multiple variables or require specialized measurements, limiting their clinical applicability [11]. Simple, cost-effective prediction tools incorporating routine laboratory parameters are still lacking. Monocyte percentage and albumin are routinely measured laboratory parameters that reflect two fundamental biological pathways: systemic inflammation and nutritional status. The combination of these two biomarkers may capture the "inflammation-nutrition axis" that underlies AVF maturation. However, to date, no study has specifically investigated the combined predictive value of monocyte percentage and albumin for AVF maturation failure. Therefore, this study aimed to develop and validate a prediction model for AVF maturation failure using monocyte percentage and albumin in ESKD patients undergoing first-time AVF creation, and to evaluate its discriminative ability, calibration, and clinical utility.

2. Materials and methods

2.1. Study design

We consecutively screened patients who underwent AVF surgery for the first time from January to December 2024. The inclusion criteria were as follows: (1) patients aged ≥ 18 years diagnosed with ESKD requiring long-term hemodialysis; (2) patients undergoing primary radiocephalic AVF creation with end-to-side anastomosis performed by the same surgical team.; (3) Availability of complete clinical and follow-up data; and (4) preoperative ultrasonography confirming patent arterial and venous lumens, with no evidence of stenosis or thrombosis. Exclusion criteria were as follows: (1) Patients with a previous history of upper limb vascular access surgery;(2) Patients with combined malignant tumors or autoimmune diseases, severe heart failure or mental illness; (3) acute complications such as trauma, bleeding, infection or acute AVF occlusion; (4) loss to follow-up within 12 weeks postoperatively; (5) death before AVF maturation could be assessed; (6) acute AVF occlusion within one week postoperatively (classified as technical failure). During the study period, a total of 179 patients who met the inclusion criteria were initially enrolled. After applying the exclusion criteria, 19 patients (10.6%) were

excluded due to: death (n = 3), loss to follow-up (n = 6), acute thrombosis within 1 week (n = 2), missing laboratory data (n = 4), and severe heart failure (n = 4). The remaining 160 patients constituted the final study cohort (Fig. 1).

2.2. The definition of AVF maturation and failure to mature

Maturation assessment followed a sequential protocol based on the Chinese Expert Consensus on Vascular Access for Hemodialysis (2nd Edition)[12]. Initial evaluation was conducted at six weeks postoperatively by a senior hemodialysis nurse with specialized expertise in vascular access. Clinical suitability was determined by the presence of a clearly identifiable venous segment that was straight, superficially located, and of sufficient length to accommodate two needles, accompanied by a well-defined palpable thrill along the venous outflow tract. Fistulas meeting the clinical criteria proceeded to color Doppler ultrasonography, performed by a radiologist specializing in vascular imaging. Ultrasound parameters for maturation, consistent with the Chinese consensus thresholds [1], included a draining vein diameter of at least 5 mm, a skin-to-vein distance not exceeding 6 mm, and brachial artery blood flow of 500 mL/min or greater. Successful maturation required fulfillment of both the physical examination and ultrasound parameters. Only fistulas satisfying all criteria were classified as mature and subsequently used for hemodialysis cannulation. Maturation failure was defined as either: (a) failure to satisfy any of the prespecified clinical or ultrasound criteria by the 12-week postoperative evaluation, or (b) the need for any procedural intervention—such as angioplasty, thrombectomy, or surgical revision—to enable fistula use.

2.3. Data Collection

Demographic and clinical data were collected from electronic medical records, including age, sex, and comorbidities (hypertension and diabetes). Preoperative vascular assessment was performed using duplex ultrasonography to measure the radial artery and cephalic vein diameters. Fasting blood samples were obtained within one week prior to surgery. The following laboratory parameters were measured: high-sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6), albumin, ferritin, transferrin, calcium, white blood cell count (WBC), platelet count (PLT), lymphocyte count (LYM), monocyte percentage, fibrinogen, D-dimer, parathyroid hormone (PTH), and inorganic phosphate (P). All measurements were performed in the clinical laboratory at the Third People's Hospital of Chengdu following standard protocols.

2.4. Statistical analysis

All statistical analyses were performed using IBM SPSS Statistics for Windows, Version 27.0 (IBM Corp., Armonk, NY, USA) and R software (version 4.4.0, R Foundation for Statistical Computing, Vienna, Austria). A two-sided P-value < 0.05 was considered statistically significant. Continuous variables were tested for normality using the Kolmogorov-Smirnov test. Normally distributed variables were expressed as mean ± standard deviation and compared using Student's t-test; non-normally distributed variables were expressed as median (interquartile range) and compared using the Mann-Whitney U test. Categorical variables were expressed as numbers (percentages) and compared using the Chi-square

test or Fisher's exact test. Univariable logistic regression was performed to identify potential predictors of AVF maturation failure.

Variables with $P < 0.05$ in univariable analysis were entered into multivariable logistic regression. Additionally, based on clinical relevance and established evidence, diabetes, radial artery diameter, and cephalic vein diameter were also included in the multivariable model to adjust for potential confounding. Multicollinearity was assessed using the variance inflation factor (VIF), with VIF > 5 indicating significant collinearity. Results were reported as odds ratios (OR) with 95% confidence intervals (CI). The performance of the final model was assessed by the area under the receiver operating characteristic curve (AUC). The optimal cutoff value was determined using the Youden index (sensitivity + specificity − 1). Calibration was assessed by the Hosmer-Lemeshow goodness-of-fit test and calibration plots. Clinical utility was evaluated using decision curve analysis. To assess model stability, internal validation was performed using bootstrap resampling with 1000 replicates. The bias-corrected AUC and its 95% CI were calculated. Sensitivity analysis comparing baseline characteristics between included and excluded patients was performed to assess potential selection bias.

3. Results

3.1. Study population

A total of 160 patients were included in the final analysis, as described in the Methods section (Fig. 1). Among these, 102 patients (63.8%) achieved successful AVF maturation within 12 weeks postoperatively, while 58 patients (36.2%) were classified as maturation failure.

3.2. Baseline characteristics

Baseline characteristics of the 160 included patients are summarized in Table 1, stratified by AVF maturation status. The maturation failure group (n = 58) had significantly lower albumin levels (32.3 ± 3.5 vs. 36.6 ± 5.8 g/L, $P < 0.001$) and higher monocyte percentages (8.4 ± 1.5% vs. 6.6 ± 2.1%, $P < 0.001$) compared to the maturation group (n = 102). No significant differences were observed between the two groups in age, sex, diabetes, hypertension, radial artery diameter, or cephalic vein diameter (all $P > 0.05$).

Table 1
Baseline characteristics stratified by AVF maturation status

Variables	Maturation group (n = 102)	Non-maturation group (n = 58)	*P*-value
Female, n (%)	53(51.9%)	22(55%)	0.359
Age (years)	58.77 ± 13.087	62.35 ± 12.345	0.163
Hypertension, n (%)	89(87.3%)	48(82.8%)	0.436
Diabetes, n (%)	62(60.8%)	32(55.2%)	0.488
Radial artery diameter (mm)	1.7 ± 0.2	1.8 ± 0.2	0.209
Cephalic vein diameter (mm)	1.8 ± 0.2	1.7 ± 0.2	0.181
Hs-CRP (mg/L)	2.32 (0.80, 10.54)	4.23 (1.10, 19.72)	0.501
LYM (×10⁹/L)	0.92 ± 0.36	1.02 ± 0.40	0.200
WBC (×10⁹/L)	6.39(4.86, 7.78)	6.07(4.61, 8.19)	0.962
PLT (×10⁹/L)	161 ± 15	153 ± 12	0.613
Albumin (g/L)	36.6 ± 5.8	32.3 ± 3.5	<0.001
Monocyte percentage (%)	6.6 ± 2.1	8.4 ± 1.5	<0.001
P (mmol/L)	1.69(1.38, 2.06)	1.55(1.08, 2.09)	0.310
Transferrin (g/L)	2.063(1.746, 2.332)	2.067(1.796, 2.274)	0.895
Calcium (mmol/L)	1.95 ± 0.24	2.03 ± 0.37	0.217
PTH (pg/mL)	253(131, 328)	281(151, 403)	0.082
Ferritin (μ g/L)	185(98, 503)	213(95, 428)	0.669
D-dimer (mg/)	1.37(0.69, 2.74)	1.17(0.66, 2.99)	0.990
Fibrinogen (g/L)	4.75 ± 1.39	4.86 ± 1.32	0.687
IL-6 (p g/mL)	6.27(3.56, 12.30)	7.15(4.02, 13.26)	0.595

Note: Categorical variables were presented as n (%) and compared using the Chi-square test or Fisher's exact test (when expected counts were < 5). Normally distributed continuous variables were compared using Student's t-test and were presented as mean ± standard deviation. Non-normally distributed continuous variables were compared using the Mann-Whitney U test and presented as median (25th-75th percentiles). Abbreviations for laboratory parameters are as defined in the Methods section. *P*-value < 0.05 was considered statistically significant.

3.3. Multivariable analysis

Based on univariable analysis results and clinical relevance, five variables were entered into multivariable logistic regression: monocyte percentage, albumin, diabetes, radial artery diameter, and cephalic vein diameter. After adjusting for all five variables simultaneously, monocyte percentage remained a significant risk factor (OR = 1.61, 95% CI 1.31–1.98, P < 0.001), while albumin remained a significant protective factor (OR = 0.84, 95% CI 0.77–0.91, P < 0.001) for AVF maturation failure (Table 2). Diabetes, radial artery diameter, and cephalic vein diameter were not significant in the multivariable model (all P > 0.05).

Table 2
Multivariable logistic regression analysis for AVF maturation failure

Variables	*P*-value	OR value	95% CI
Monocyte percentage	< 0.001	1.61	(1.31–1.98)
Albumin	< 0.001	0.84	(0.77–0.91)
Radial artery diameter	0.638	0.66	(0.12–3.73)
Cephalic vein diameter	0.250	0.22	(0.02–2.89)
Diabetes	0.271	1.55	(0.71–3.39)
Note: OR, odds ratio; CI, confidence interval			

3.4. Predictive performance of individual biomarkers and the combined model

The predictive performance of albumin, monocyte percentage, and their combination was assessed using receiver operating characteristic (ROC) curve analysis and is summarized in Table 3.

Table 3
Predictive performance of albumin, monocyte percentage, and the combined model

Biomarker	AUC (95% CI)	Optimal cutoff	Sensitivity (%)	Specificity (%)
Albumin	0.718 (0.641–0.795)	34.70 g/L	69.0	60.8
Monocyte percentage	0.736 (0.661–0.812)	6.80%	81.0	53.9
Combined model	0.811 (0.745–0.876)	0.328	86.2	70.6
Note: AUC, area under the receiver operating characteristic curve; CI, confidence interval				

Albumin alone achieved an AUC of 0.718 (95% CI 0.641–0.795), with an optimal cutoff of 34.70 g/L yielding a sensitivity of 69.0% and specificity of 60.8%. Monocyte percentage alone showed an AUC of 0.736 (95% CI 0.661–0.812), with an optimal cutoff of 6.80% giving a sensitivity of 81.0% and specificity of 53.9%.

As shown in Fig. 2, the combined model incorporating both biomarkers achieved a superior AUC of 0.811 (95% CI 0.745–0.876). Using the Youden index, the optimal probability cutoff for predicting AVF maturation failure was 0.328. At this threshold, the model demonstrated a sensitivity of 86.2% and a specificity of 70.6% (Table 3).

These findings indicate that the combined model provides better discrimination than either biomarker alone, with a well-balanced sensitivity and specificity profile suitable for clinical application.

3.5. Internal validation

To assess the stability and generalizability of the model, internal validation was performed using bootstrap resampling with 1000 replicates. The bootstrap-corrected AUC was 0.812 (95% CI 0.739–0.877), which was nearly identical to the original model's AUC of 0.811. The optimism bias was minimal at 0.0011, with a standard error of 0.0345(Supplementary Figure S1).

3.6. Calibration

The calibration of the combined model was assessed using a calibration plot with 200 bootstrap resamples (Fig. 3). The calibration curve demonstrated good agreement between predicted probabilities and observed outcomes, with the fitted line closely following the ideal diagonal line. The mean absolute error was 0.048, and the 90th percentile of absolute error was 0.08, indicating that 90% of predictions were within 8% of the true probability. The Hosmer-Lemeshow test yielded a chi-square value of 24.65 (df = 8, P = 0.002).

3.7. Decision curve analysis

Decision curve analysis was performed to evaluate the clinical value of the combined model (Fig. 4). The model yielded a positive net benefit for threshold probabilities below 0.64. At the selected cutoff of 0.328, the combined model provided a net benefit of 0.214, surpassing both the "treat all" (net benefit = -0.308) and "treat none" (net benefit = 0) strategies. The combined model curve crossed the "treat all" curve at a threshold of approximately 0.64, indicating superior net benefit across the clinically relevant range (< 0.64).

4. Discussion

In this single-center retrospective cohort study of 160 patients undergoing first-time AVF creation, we developed and validated a prediction model for AVF maturation failure based on two routine laboratory parameters: monocyte percentage and albumin. The combined model demonstrated good discriminative ability with an AUC of 0.811 (95% CI 0.745–0.876), and was well-calibrated with a mean absolute error of 0.048. Decision curve analysis confirmed its clinical utility, showing a net benefit of 0.214 at the optimal cutoff of 0.328, which exceeded both the "treat all" (net benefit = -0.308) and "treat none" (net benefit = 0) strategies[13]. The performance of our model (AUC = 0.811) compares favorably with recently published prediction models for AVF maturation. A systematic review of 14 prediction models reported that risk

score approaches achieved C-statistics ranging from 0.70 to 0.886, while machine learning methods achieved 0.80 to 0.85[11]. Our model falls within the upper range of these performance metrics, despite using only two routinely available biomarkers.

Several recent studies have developed predictive models incorporating inflammatory and nutritional markers. Zhao et al. developed a nomogram based on SIRI, TyG-BMI, and HRR, achieving strong predictive accuracy in a cohort of 249 patients, with an AUC of 0.79 [14]. Zeng et al. identified arterial diameter, cholesterol levels, lean tissue index, and history of coronary artery disease as predictors, with an AUC of 0.79 [15]. Gao et al. developed a HALP score (hemoglobin, albumin, lymphocyte, platelet) model in 509 patients, achieving an AUC of 0.78 (95% CI 0.73–0.83) for predicting AVF maturation failure [16]. Compared to these models, our two-biomarker approach offers comparable or superior predictive performance with greater simplicity and clinical applicability.

Albumin, as a nutritional indicator, has been shown by current research to be related to the outcome of AVF[14]. Kordzadeh et al. reported that preoperative hypoalbuminemia (< 35 mg/dL) was independently associated with a 40% reduction in functional maturation of radiocephalic AVF[17].

A recent systematic review and meta-analysis including 38 studies confirmed that low serum albumin level is significantly associated with early arteriovenous fistula failure(SMD = -0.423, 95% CI -0.733 to -0.113, P = 0.007) [10], providing the highest level of evidence for this association. Okuhata et al. followed 57 patients after initial vascular access intervention therapy and found that low serum albumin was the most significant predictor of short-term shunt stenosis (p = 0.031)[18]. Several studies have also demonstrated the predictive value of albumin-based composite indices. Hu et al. reported that the C-reactive protein to albumin ratio (CAR) was independently associated with AVF dysfunction in 726 patients (HR = 1.31 per unit increase, 95% CI 1.08–1.58)[19]. Similarly, Zhao et al. identified the high-sensitivity C-reactive protein to albumin ratio (HRR) as an independent predictor of AVF maturation failure (HR = 1.44, 95% CI 1.28–1.61, P < 0.001) in a prospective cohort of 249 patients[14]. These composite indices highlight the synergistic impact of inflammation and malnutrition on vascular remodeling.

The protective role of albumin in vascular health extends beyond its function as a nutritional marker. Aldecoa et al. comprehensively reviewed the role of albumin in preserving endothelial glycocalyx integrity—a critical structure that regulates vascular permeability, mechanotransduction, and inflammation. Albumin plays a vital role in maintaining vascular integrity and capillary permeability, and its deficiency is often a marker of ongoing inflammation [20]. A key mechanism involves albumin's transport of sphingosine-1-phosphate (S1P). Recent studies have demonstrated that albumin-bound S1P plays an essential role in maintaining vascular resistance and endothelial barrier function [21]. Furthermore, albumin acts as a free radical scavenger through its Cys34 thiol group, reducing oxidative stress and modulating nitric oxide (NO) signaling, thereby protecting endothelial function [22]. These mechanisms provide a biological basis for our finding that low albumin levels are associated with

increased risk of AVF maturation failure, as endothelial dysfunction and impaired vascular remodeling are key pathological processes in AVF non-maturation.[23–25]

The role of monocyte percentage in AVF maturation has been increasingly recognized. Satam et al. made a particularly important contribution by demonstrating that monocyte percentage is a sex-dependent predictor of early AVF maturation, specifically in female patients[26]. This finding supports the biological plausibility of monocyte percentage as a predictor and aligns with our result that each 1% increase in monocyte percentage was associated with a 61% increase in the risk of maturation failure (OR = 1.61). Monocyte-derived composite indices have also shown predictive value. Hu et al. demonstrated in a large cohort of 769 patients that the monocyte-to-lymphocyte ratio (MLR) was independently associated with AVF dysfunction[27]. Similarly, Tanaka et al. reported that a monocyte count ≥ 400/μL was an independent risk factor for vascular access failure in hemodialysis patients[28]. These clinical observations are supported by emerging mechanistic evidence. Recent single-cell RNA sequencing of human AVFs revealed that early remodeling is characterized by increased monocyte infiltration, and failed AVFs display persistent inflammation with abundant proinflammatory macrophages (derived from circulating monocytes)[9]. These findings provide a cellular-level explanation for our observation that elevated monocyte percentage predicts maturation failure, establishing a biological basis for incorporating this routine laboratory marker into clinical prediction models.

Monocyte percentage and albumin together capture two fundamental physiological processes—inflammation and nutrition—that critically influence AVF maturation [9, 20]. Monocyte percentage reflects the inflammatory burden that drives maladaptive vascular changes, while albumin represents the nutritional reserve and anti-inflammatory capacity essential for adaptive healing [14, 20]. The Malnutrition-Inflammation Complex Syndrome, frequently observed in ESRD patients, establishes a self-reinforcing cycle: inflammation suppresses albumin production while hypoalbuminemia compromises inflammation resolution [10, 29]. This reciprocal relationship likely accounts for the superior predictive performance of their combination (AUC = 0.811) compared to either marker alone. The Hemodialysis Fistula Maturation study has fundamentally shifted our perspective on AVF failure, identifying impaired outward remodeling from wall fibrosis as the dominant mechanism rather than isolated intimal hyperplasia [9, 30]. This fibrotic process may be amplified by concomitant inflammation and malnutrition, providing mechanistic support for our findings.

The identification of monocyte percentage and albumin as independent predictors of AVF maturation failure has several practical implications for clinical management. Using the established cutoff of 0.328, clinicians can identify high-risk patients (predicted probability ≥ 0.328). In high-risk patients, preoperative interventions aimed at reducing systemic inflammation and improving nutritional status may be beneficial. This includes screening for occult infections, optimizing glycemic control in diabetic patients, and correcting hypoalbuminemia through nutritional support, as low albumin levels have been consistently associated with AVF failure[10, 27]. High-risk patients should undergo more frequent postoperative monitoring, including serial ultrasound assessments at 2, 4, and 6 weeks after surgery [31]. Early detection of impaired flow (< 500 mL/min) may prompt timely intervention, such as

angioplasty or surgical revision, before complete failure occurs[15]. This risk-stratified approach, guided by two simple and inexpensive laboratory tests, has the potential to improve AVF maturation rates while avoiding unnecessary interventions in low-risk patients, ultimately reducing the burden of vascular access complications in hemodialysis patients.

Several limitations of this study should be acknowledged. First, this was a single-center retrospective study with a relatively modest sample size (n = 160), which may limit the generalizability of our findings. However, bootstrap validation (optimism bias = 0.0011) confirmed model stability, and our sample size meets the recommended events per variable criterion of at least 10 (EPV = 11.6). Second, the retrospective design may introduce selection bias, although sensitivity analysis showed no significant differences between included and excluded patients (all $P > 0.05$). Despite these limitations, the model demonstrated robust performance through internal validation, and the use of two routinely available laboratory tests enhances its potential clinical applicability. Future large-scale prospective studies are warranted to validate our findings and further evaluate the clinical utility of this prediction model in diverse populations.

Conclusions

Monocyte percentage and albumin are independent predictors of AVF maturation failure. Their combination provides a simple, cost-effective prediction model with good discrimination (AUC = 0.811), excellent calibration (MAE = 0.048), and positive clinical utility (net benefit = 0.214). This two-biomarker approach may facilitate preoperative risk stratification and individualized management to improve AVF maturation outcomes.

Declarations

Ethics Approval declaration

This study was approved by the Ethics Committee at the Third People's Hospital of Chengdu (Approval No. 2025-S-202). All procedures performed in this study involving human participants were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. The requirement for informed consent was waived due to the retrospective nature of the study.

Authors contributions

Y.S. designed the research and revised the manuscript. L.Y. collected and analyzed data and wrote the manuscript. X.S. and B.Y. participated in the surgical operation. L.J. and D.W. assisted in data collection. All authors read and approved the final manuscript.

Disclosure statement

No potential conflict of interest was reported by the authors. Patients or the public WERE NOT involved in the design, conduct, or reporting or dissemination plans of our research.

Data availability statement

The original contributions presented in the study are included in the article Material. Further inquiries can be directed to the corresponding author.

Funding

This work was supported by the Chengdu Municipal Health Commission under Grant 2022305.

References

1. Sun J, Hu W, Ye S, Xu M, Deng D, Chen M. Global, regional and country-specific burden of chronic kidney disease due to type 1 diabetes mellitus: A systematic analysis of the 2021 global disease burden study. Diabetes Obes Metab. 2025;27(6):3397–409.
2. Gondal M. Overview of, and Preparations for, Dialysis. Med Clin North Am. 2023;107(4):681–7.
3. Global regional, national burden of chronic kidney disease. 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet (London England). 2020;395(10225):709–33.
4. Bello AK, Okpechi IG, Osman MA, Cho Y, Htay H, Jha V, Wainstein M, Johnson DW. Epidemiology of haemodialysis outcomes. Nat Rev Nephrol. 2022;18(6):378–95.
5. Poushpas S, Normahani P, Kisil I, Szubert B, Mandic DP, Jaffer U. Tensor decomposition and machine learning for the detection of arteriovenous fistula stenosis: An initial evaluation. PLoS ONE. 2023;18(7):e0286952.
6. Lok CE, Huber TS, Orchanian-Cheff A, Rajan DK. Arteriovenous Access for Hemodialysis: A Review. JAMA. 2024;331(15):1307–17.
7. Allon M, Young CJ, Lee T. Optimizing Dialysis Vascular Access: Moving beyond Fistula First. 9900:10.2215/CJN.0000000880
8. Akkaya Ö, Arslan Ü. Sex-Specific Impact of Inflammation and Nutritional Indices on AVF Blood Flow and Maturation: A Retrospective Analysis. Diagnostics (Basel Switzerland) 2025, 15(10).
9. Martinez L, Stoyell-Conti FF, Tabbara M, Rojas MG, Pereira-Simon S, Santos Falcon N, Hernandez Lopez R, Galtes D, Kosanovic C, Griswold AJ, et al. The single-cell landscape of the human vein after arteriovenous fistula creation and implications for maturation failure. Kidney Int. 2026;109(1):160–77.
10. Salih SSM, Mohamed KO, Mohamedali AOO, Mahmoud AAO, Ibrahim DAS, Abdallah KF, Salih MSK, Abdhameed AEB, Salih NSA, Salih KSK, et al. Predictors of early arteriovenous fistula failure in patients with end-stage renal disease on hemodialysis: a systematic review and meta-analysis. Patient Saf Surg. 2025;19(1):24.

11. Meng L, Ho P. A systematic review of prediction models on arteriovenous fistula: Risk scores and machine learning approaches. J Vasc Access. 2025;26(3):735–46.
12. Association VAWGotBPCBotCH: Chinese Expert Consensus on Vascular Access for Hemodialysis (2nd Edition). Chinese Journal of Blood Purification. 2019, 18(6):365–381.
13. Quintana AN, Schmid CH, Qu K, Gainey M, Nasrin S, Monjory M, Nelson EJ, Alam NH, Levine AC. Evaluating clinical utility of multi-category outcome risk prediction models. *medRxiv: the preprint server for health sciences* 2025.
14. Zhao B, Fu G, Zhan S, Zhang L, Cui R, Guo S, Li J, Lu H, Wang Y. Development and validation of an inflammatory-based nomogram for predicting arteriovenous fistula maturation failure in end-stage renal disease patients. Langenbeck's archives Surg. 2025;410(1):231.
15. Zeng W, Zhang B, Wang X, Wang R, Niu Y, Yue X, Liang X, Wang P. Prediction model for radial-cephalic arteriovenous fistula failure to mature. Ann Med. 2025;57(1):2559124.
16. Gao X, Liang D, Jia X, Liu Y, Guo K, Li X. The hemoglobin, albumin, lymphocyte, and platelet (HALP) score serves as a predictive indicator for the autogenous arteriovenous fistula maturation failure. Sci Rep 2026.
17. Kordzadeh A, Tokidis E, Askari A, Hoff M, Panayiotopoulos Y. The independent association of preoperative serum albumin on the functional maturation of radiocephalic arteriovenous fistulae. J Vasc Access. 2017;18(2):148–52.
18. Okuhata Y, Sakai Y, Ikenouchi A, Kashiwagi T, Iwabu M. Low Serum Albumin Levels are Associated with Short-Term Recurrence of Arteriovenous Fistula Failure. J Nippon Med School = Nippon Ika Daigaku zasshi. 2024;91(4):383–90.
19. Hu S, Wang R, Ma T, Lei Q, Yuan F, Zhang Y, Wang D, Cheng J. Association between preoperative C-reactive protein to albumin ratio and late arteriovenous fistula dysfunction in hemodialysis patients: a cohort study. Sci Rep. 2023;13(1):11184.
20. Aldecoa C, Llau JV, Nuvials X, Artigas A. Role of albumin in the preservation of endothelial glycocalyx integrity and the microcirculation: a review. Ann Intensiv Care. 2020;10(1):85.
21. Del Gaudio I, Bonnin P, Vessiéres E, Robidel E, Garcia MC, Proux C, Boutigny A, Baudrie V, Ha HTT, Couty L, et al. Blood-borne sphingosine 1-phosphate maintains vascular resistance, blood pressure, and cardiac function in mice. Proc Natl Acad Sci USA. 2026;123(2):e2512853123.
22. Belinskaia DA, Voronina PA, Goncharov NV. Integrative Role of Albumin: Evolutionary, Biochemical and Pathophysiological Aspects. J Evol Biochem Physiol. 2021;57(6):1419–48.
23. González I, Maldonado-Agurto R. The role of cellular senescence in endothelial dysfunction and vascular remodelling in arteriovenous fistula maturation. J Physiol 2025.
24. Bao J, Tian G, Tu Y, Liao Q, Yao L. Vascular Smooth Muscle Cell Metabolic Reprogramming in Arteriovenous Fistula Failure. *Biomedicines* 2025, 13(10).
25. Shiu YT, Northrup H, Huang Y, Cho ME, Bunsawat K. Cellular and molecular mechanisms underlying hemodialysis arteriovenous fistula dysfunction and approaches to promote maturation: a vascular perspective. Am J Physiol Heart Circ Physiol. 2025;329(1):H241–57.

26. Satam K, Setia O, Moore MS, Schneider E, Chaar CIO, Dardik A. Arterial Diameter and Percentage of Monocytes are Sex-Dependent Predictors of Early Arteriovenous Fistula Maturation. Ann Vasc Surg. 2023;93:128–36.
27. Hu S, Wang D, Ma T, Yuan F, Zhang Y, Gao X, Lei Q, Cheng J. Association between Preoperative Monocyte-to-Lymphocyte Ratio and Late Arteriovenous Fistula Dysfunction in Hemodialysis Patients: A Cohort Study. Am J Nephrol. 2021;52(10–11):854–60.
28. Tanaka A, Ito Y, Tanaka T, Satozaki S, Hayashi F, Tsuda I. Blood monocyte count may be a predictor of vascular access failure in hemodialysis patients. Therapeutic apheresis dialysis: official peer-reviewed J Int Soc Apheresis Japanese Soc Apheresis Japanese Soc Dialysis Therapy. 2013;17(6):620–4.
29. Tian R, Chang L, Cheng L, Yang R, Zhang H. Malnutrition-inflammation-fluid overload complex syndrome and all-cause mortality in patients undergoing hemodialysis. Ren Fail. 2025;47(1):2512405.
30. Anderson EM, Huber TS, Neal D, Berceli SA, Shah SK, Stone DH, Scali ST. The Impact of Reintervention on Arteriovenous Fistula Maturation and Functional Patency in the Hemodialysis Fistula Maturation Study. Kidney Med. 2025;7(8):101036.
31. Veneziano A, Franchin M, Cervarolo MC, Monteleone S, Ros L, Tozzi M. Optimizing the life of vascular access during follow-up. J Cardiovasc Surg. 2025;66(1):26–9.

Figures

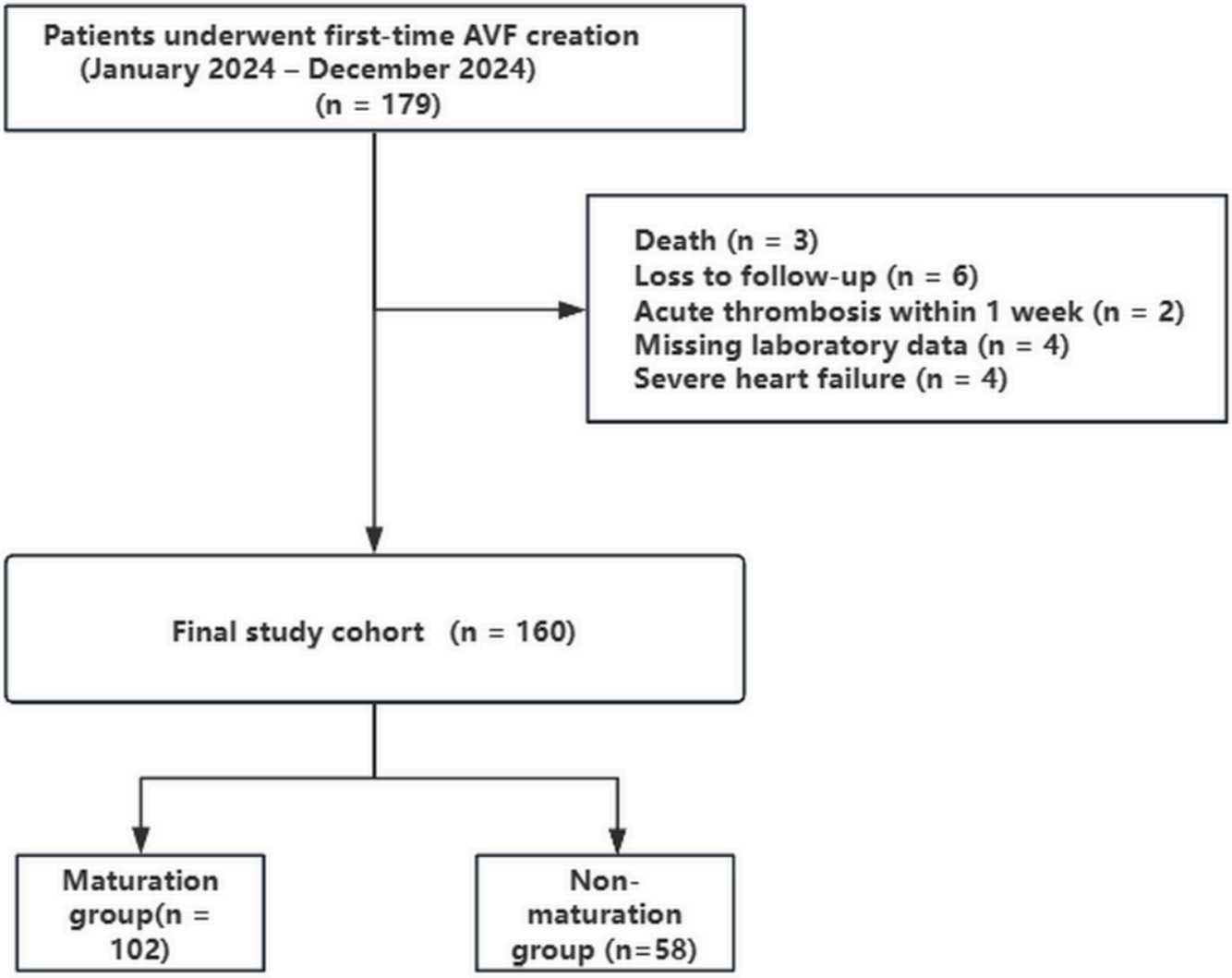


Figure 1

Flowchart of patient selection

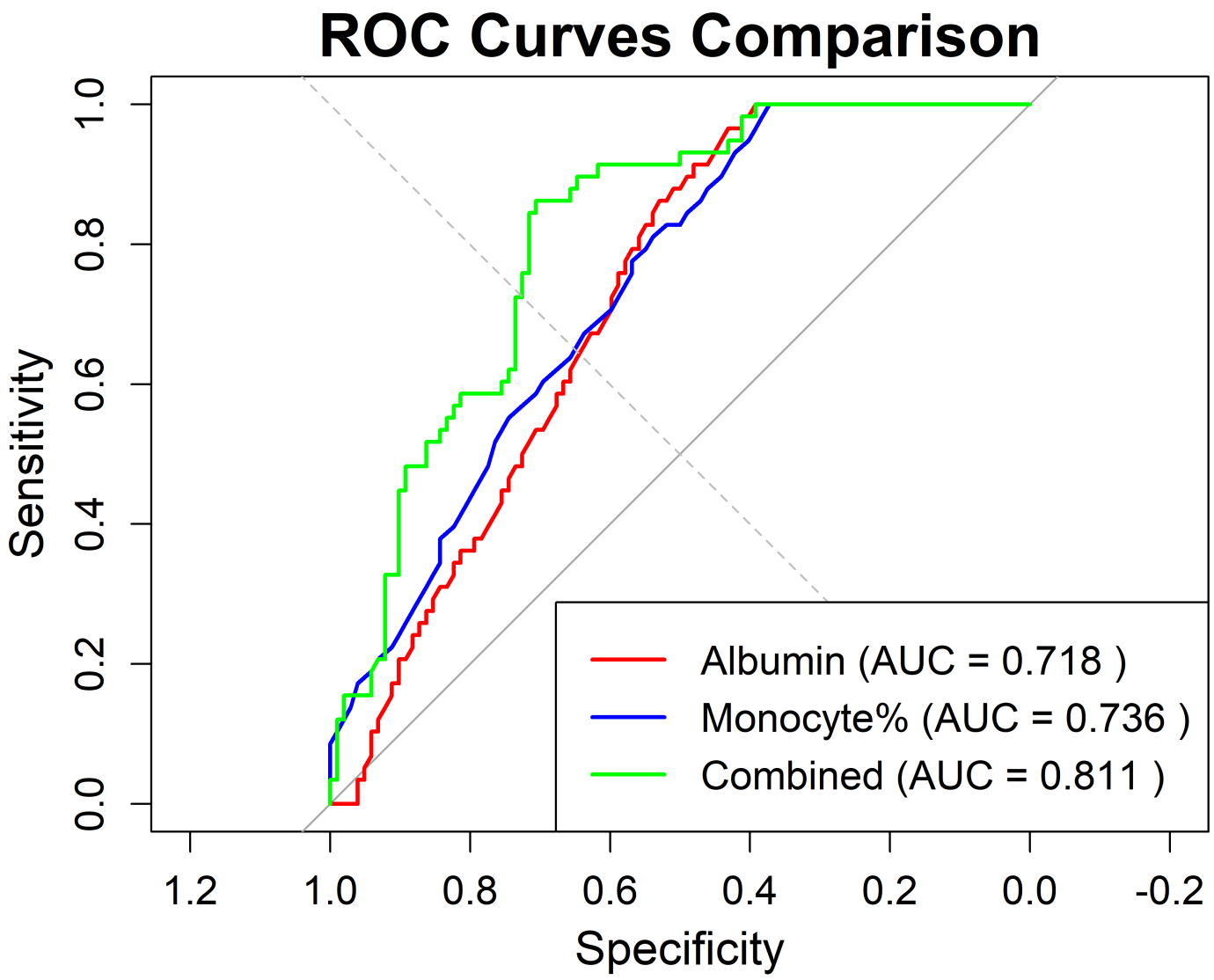


Figure 2

ROC curves for albumin, monocyte percentage, and their combination in predicting AVF maturation failure.

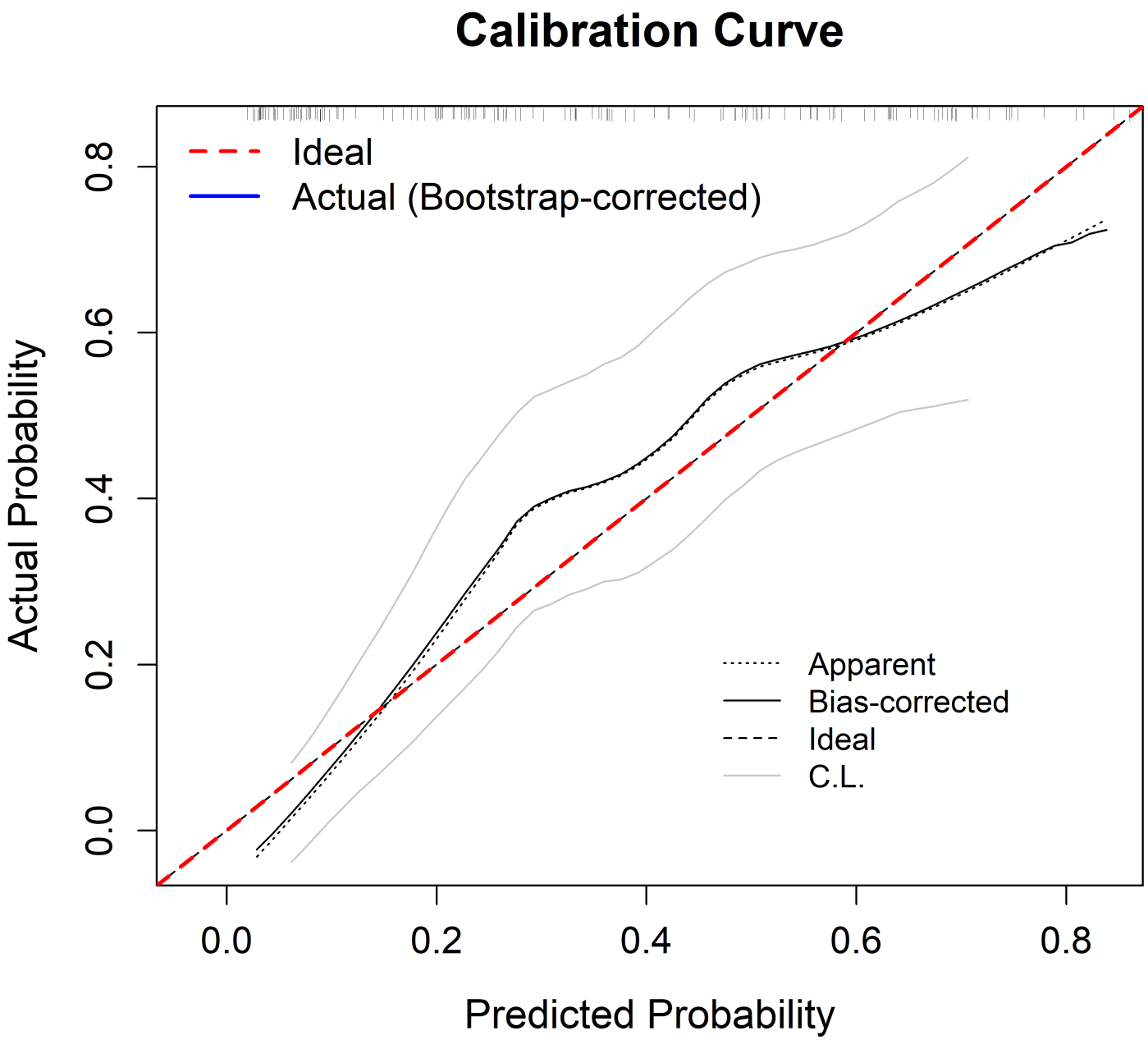


Figure 3

Calibration curve for the combined model

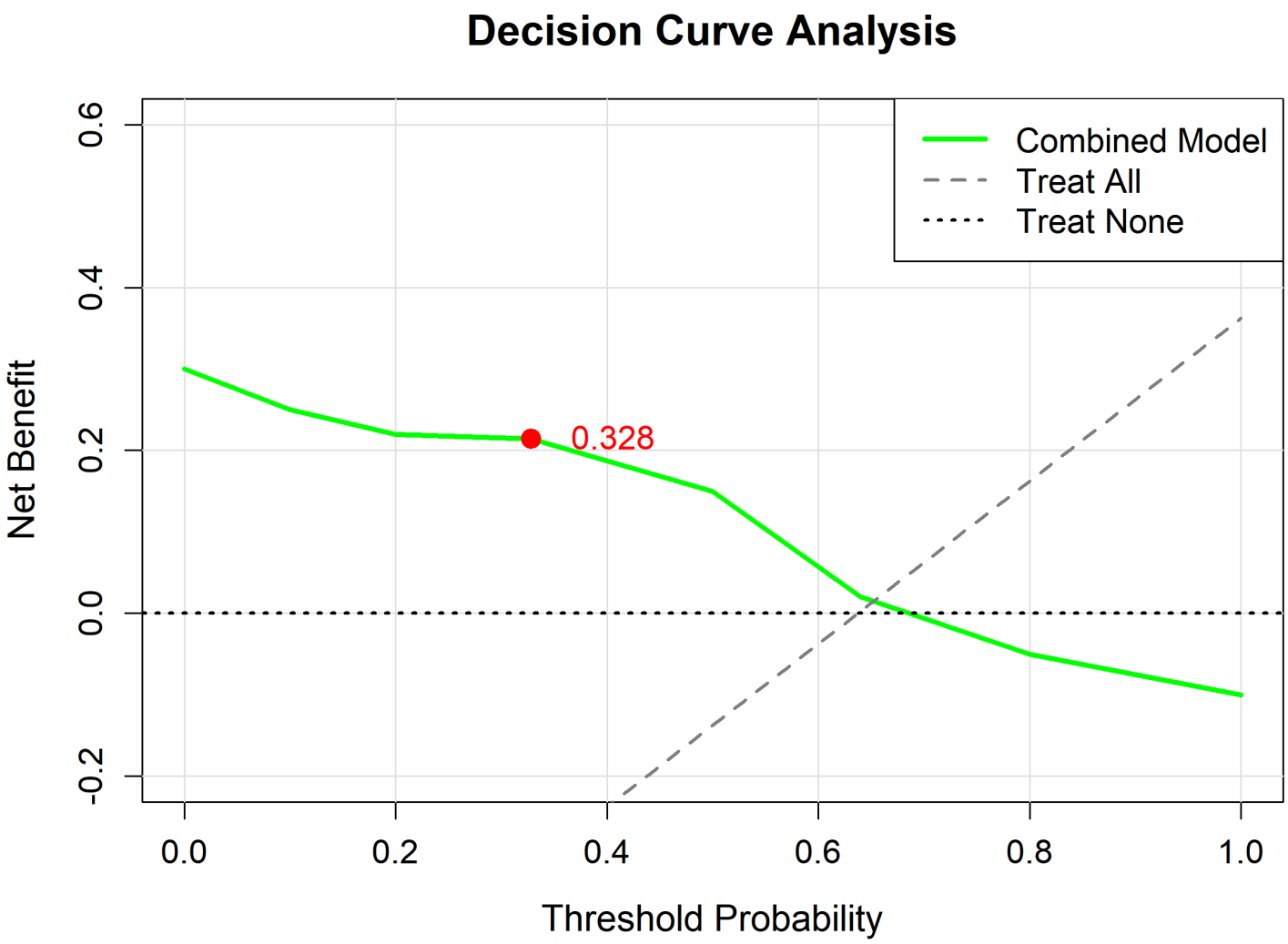


Figure 4

Decision curve analysis for the combined model. The green line represents the net benefit of the combined model, the gray dashed line represents the "treat all" strategy, and the black dotted line represents the "treat none" strategy. The vertical red line indicates the optimal cutoff of 0.328.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- FigureS1bootstrapdistribution.png