Article

Discovery Medicine 2026; 38(208): 1529–1536
https://doi.org/10.24976/Discov.Med.202638208.119

Association of HbA1c and cTnI Levels With Arteriovenous Fistula Thrombosis in Hemodialysis Patients

Chunya Liu[1], Li Wang[1], Linfeng Luo[1], Yinbing Zhu[1,*]

[1]Department of Nephrology, The Quzhou Affiliated Hospital of Wenzhou Medical University, Quzhou People's Hospital, 324000 Quzhou, Zhejiang, China

*Correspondence: zybqzy6569@163.com (Yinbing Zhu)

Submitted: 7 January 2026 Revised: 23 March 2026 Accepted: 3 April 2026 Published: 20 May 2026

Background: Observational and mechanistic studies have linked glycated hemoglobin (HbA1c) and cardiac troponin I (cTnI) to chronic inflammation and thrombosis. We explored how these two routinely available biomarkers relate to arteriovenous fistula (AVF) thrombosis in maintenance hemodialysis (MHD), and quantified their predictive value singly and in combination.

Methods: We reviewed 236 patients with consecutive end-stage renal disease (ESRD) who underwent autogenous AVF creation between January 2021 and June 2023. Baseline demographic, clinical and biochemical variables were recorded. Logistic regression (univariate then multivariate) identified independent correlates of thrombosis. Using the final model, we built a nomogram and evaluated its performance using receiver operating characteristic (ROC) analysis, calibration behavior and decision curve analysis (DCA).

Results: Within 12 months, 25 patients (10.6%) met criteria for AVF thrombosis. Four variables remained independently associated with events: C-reactive protein (CRP) ≥10 mg/L, intradialytic hypotension, HbA1c ≥6.5%, and cTnI ≥0.2 µg/L (all $p <$ 0.05). A nomogram combining these factors demonstrated good discriminative ability (area under the curve [AUC] 0.850; 95% CI 0.741–0.960) and showed acceptable calibration (Hosmer–Lemeshow p = 0.244).

Conclusion: HbA1c and cTnI may serve as sensitive markers for postoperative AVF thrombosis in hemodialysis patients, facilitating earlier risk identification, preventive measures, and timely intervention. Their predictive performance is further enhanced when combined with CRP and intradialytic hypotension.

Keywords: hemodialysis; arteriovenous fistula thrombosis; end-stage renal disease; risk prediction; nomogram; glycated hemoglobin; cardiac troponin I

Introduction

The population requiring chronic hemodialysis has grown progressively alongside increasing human longevity. For end-stage renal disease (ESRD), maintenance hemodialysis (MHD) is still the dominant renal replacement therapy [1]. Access choices include autogenous arteriovenous fistula (AVF), tunneled catheter and prosthetic graft; of these, AVF is typically favored because it tends to last longer, is associated with a lower infection rate, and is easier to maintain in routine practice [2]. Even so, guideline endorsement does not grant indefinite patency [3]. Early functional failure—largely attributable to thrombosis—has been quoted at roughly 20%–60% and threatens both dialysis adequacy and patient survival [4]. Clinicians, therefore, seek markers that flag risk before the vascular access failure occurs.

Multiple contributors to AVF dysfunction have been described—systemic inflammation, glycemic milieu, and blood-pressure instability among them—yet the pathways are not fully mapped [5]. Local shear irregularities and repeated cannulation stress the endothelium and can promote thrombosis [6]. In parallel, higher glycated hemoglobin (HbA1c) and cardiac troponin I (cTnI) levels have been associated with endothelial disturbance and vascular injury signals [7]. Endothelial cells preserve barrier integrity, regulate vascular tone, and coordinate inflammatory cell trafficking; perturbation of this system increases the likelihood of intimal thickening and clotting. Despite being easy to measure, HbA1c and cTnI have not been routinely used to predict AVF thrombosis. We therefore explored whether these markers—alone and in combination with clinical factors—predict AVF thrombosis over one year, and whether a simple point-based nomogram could provide practical utility at the chairside.

Methods

Study Population

We retrospectively identified patients with ESRD who underwent autogenous AVF creation at The Quzhou Affiliated Hospital of Wenzhou Medical University (Quzhou People's Hospital), Quzhou, China, between January 2021 and June 2023. Inclusion criteria included (i) ESRD diagnosis; (ii) hemodialysis delivered via AVF in our unit; (iii) cephalic vein–radial artery end-to-side anastomosis; and (iv) availability of complete 12-month follow-up data in dialysis records. Exclusion criteria were (i) severe organ failure or active malignancy; (ii) pre-existing thrombotic disease; and (iii) psychiatric disorder limiting cooperation. The Quzhou Affiliated Hospital of Wenzhou Medical University Ethics Institution approved the protocol (Approval No. QSRMH2024-083), consistent with the Declaration of Helsinki. Because this was a retrospective study based on existing clinical records, and this study has obtained the informed consent of both the patients and their families. During the study period, protocols for AVF creation, perioperative care, dialysis procedures, and routine laboratory monitoring remained consistent at our center, with no major changes in clinical practice that would systematically affect AVF patency outcomes. All included patients were followed for up to 12 months after AVF creation. Follow-up information was obtained from routine dialysis records, and patients with incomplete follow-up data were excluded from analysis.

Data Collection

For each patient, we recorded sex, age, body mass index (BMI), hemoglobin (Hb), C-reactive protein (CRP), hypertension (HTN), diabetes mellitus (DM), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), triglycerides (TG), total cholesterol (TC), and intradialytic hypotension (defined as a fall in systolic blood pressure $\geq$20 mmHg, or mean arterial pressure $\geq$10 mmHg, during dialysis). Intradialytic hypotension was defined based on documented episodes in routine dialysis records during the baseline assessment period and analyzed as a binary variable to reflect clinically meaningful recurrent hypotension. We also documented the type and proportion of low-molecular-weight heparin (LMWH) used for anticoagulation. All laboratory parameters, including HbA1c, cTnI, and CRP, were obtained as part of routine baseline assessments before or at the time of AVF creation, and values measured after the occurrence of thrombotic events were not included in the analysis.

Specimen Collection and Technical Procedures

Hemodialysis used a Fresenius F60S dialyzer (Fresenius Medical Care, Bad Homburg, Germany; surface 1.3 m^2; single-use) and bicarbonate dialysate (dialysate flow 500 mL/min) with blood flow ~250 mL/min. LMWH provided anticoagulation for thrice-weekly sessions. Low-molecular-weight heparin dosing followed standard weight-based protocols in our dialysis unit and was adjusted according to routine clinical assessment; no major changes in anticoagulation strategy occurred during the study period. For laboratory testing, HbA1c was measured from 1.5 mL EDTA-anticoagulated whole blood using high-performance liquid chromatography (HPLC) on an MQ-2000PT HbA1c analyzer (Shanghai Huizhong Medical Science and Technology Co., Shanghai, China). Serum cTnI levels were determined from 2 mL venous blood samples after centrifugation at 3500 r/min for 5 min, using an acridinium ester chemiluminescence immunoassay on the ARCHITECT i2000SR analyzer (Abbott Laboratories, Abbott Park, IL, USA). All assays were performed according to the manufacturers' instructions. Internal quality controls were included in each batch, and laboratory personnel were blinded to clinical outcomes.

AVF Endpoint and Follow-up

Patients were followed for 12 months. AVF thrombosis was diagnosed if any of the following criteria were met: disappearance of thrill and bruit; markedly weakened thrill/bruit plus dialysis blood flow $<$160 mL/min; evidence of thrombus on ultrasound; or failure of contrast passage on angiography. AVF thrombosis was recorded as a binary outcome indicating whether thrombosis occurred within the 12-month follow-up period. Although the exact date of arteriovenous fistula thrombosis was recorded in clinical records, the present analysis focused on the occurrence of thrombosis within a fixed 12-month period. Therefore, logistic regression was used to evaluate the 12-month risk rather than a time-to-event outcome.

Statistical Analysis

Statistical analysis was performed using SPSS 22.0 [IBM (International Business Machines Corporation), Armonk, NY, USA]. After assessing normality and homogeneity of variance, normally distributed variables were expressed as mean $\pm$ SD and compared using independent-sample t tests; categorical variables were reported as percentages and compared using χ^2 test. Continuous variables were dichotomized based on clinically relevant cut-off values for univariate logistic regression analysis. Variables with $p < 0.2$ or strong clinical relevance proceeded to multivariate logistic regression to identify independent predictors. To reduce the risk of overfitting, given the limited number of outcome events, the number of variables entered into the multivariable model was carefully restricted based on clinical relevance and univariate screening results. Regression coefficients were used to construct a nomogram for individualized risk estimation. Model discrimination, calibration, and clinical net benefit were evaluated using the receiver operating characteristic (ROC) curve of the combined multivariable model, calibration plots, and decision

Table 1. Baseline clinical characteristics of MHD patients.

Variable	Thrombosis group (n = 25)	Control group (n = 211)	Statistic	*p* value
Age (years)	44.45 ± 7.22	46.81 ± 10.65	–1.078	0.282
Sex (Male/Female)	10 (40.00%)/15 (60.00%)	63 (29.86%)/148 (70.14%)	1.076	0.300
BMI (kg/m^2)	23.26 ± 2.94	22.55 ± 2.69	1.236	0.218
RBC (×10^{12}/L)	4.95 ± 1.07	5.12 ± 1.05	–0.764	0.446
Hemoglobin (g/L)	98.94 ± 12.20	100.06 ± 11.98	–0.441	0.660
WBC (×10^9/L)	10.45 ± 2.08	9.88 ± 1.83	1.451	0.148
Neutrophil count (×10^9/L)	7.35 ± 1.80	6.91 ± 2.07	1.018	0.310
Lymphocyte count (×10^9/L)	3.36 ± 1.20	3.02 ± 1.06	1.495	0.136
Platelet count (×10^9/L)	102.82 ± 42.13	102.58 ± 27.66	0.038	0.970
Prothrombin time (s)	12.00 ± 1.43	12.01 ± 1.88	–0.026	0.979
CRP (mg/L)	9.53 ± 3.79	7.45 ± 2.79	3.381	0.001
HbA1c (%)	6.81 ± 1.05	6.00 ± 0.76	4.819	<0.01
cTnI (µg/L)	0.30 ± 0.09	0.19 ± 0.09	5.778	<0.01
HDL-C (mmol/L)	1.22 ± 0.65	1.35 ± 0.72	–0.862	0.390
LDL-C (mmol/L)	2.25 ± 0.95	2.53 ± 0.99	–1.343	0.181
Triglycerides (mmol/L)	1.42 ± 0.63	1.55 ± 0.71	–0.875	0.382
Total cholesterol (mmol/L)	4.24 ± 1.22	4.58 ± 1.65	–0.998	0.319
Intradialytic hypotension (Yes/No)	19 (76.00%)/6 (24.00%)	95 (45.02%)/116 (54.98%)	8.589	<0.01
Diabetes (Yes/No)	10 (40.00%)/15 (60.00%)	77 (36.49%)/134 (63.51%)	0.118	0.731
Hypertension (Yes/No)	14 (56.00%)/11 (44.00%)	87 (41.23%)/124 (58.77%)	1.991	0.158
LMWH anticoagulation (Yes/No)	18 (72.00%)/7 (28.00%)	172 (80.9%)/39 (19.1%)	1.290	0.256

MHD, maintenance hemodialysis; BMI, body mass index; CRP, C-reactive protein; HbA1c, glycated hemoglobin; cTnI, cardiac troponin I; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; LMWH, low-molecular-weight heparin; RBC, Red Blood Cell; WBC, White Blood Cell.

curve analysis (DCA), respectively. To internally validate the model and quantify potential overfitting, bootstrap resampling with 500 repetitions was performed. Samples were drawn with replacement from the original dataset, the model was refitted in each bootstrap sample, and its performance was evaluated on the original sample. The average performance of these analyses provided an optimism-corrected estimate of the model's expected performance on new data. Variance inflation factors were assessed to detect multicollinearity. All variables included in the multivariable model exhibited VIF values <5, indicating no significant multicollinearity. Baseline missing data were minimal and handled using complete-case analysis. A two-sided $p < 0.05$ was considered statistically significant. Because the outcome was defined as the occurrence of AVF thrombosis within a fixed 12-month follow-up period, logistic regression was considered more appropriate than time-to-event analysis in this study.

Results

Baseline Clinical Characteristics

Altogether 236 MHD patients were included; 25 (10.59%) developed AVF thrombosis within one year. As shown in Table 1, the thrombosis and non-thrombosis groups were comparable with respect to sex, age, BMI, DM, HTN and most biochemistry (all $p > 0.05$). In contrast, intradialytic hypotension was more frequent in the thrombosis group (76.00% vs. 45.02%, $p < 0.01$), and CRP, HbA1c and cTnI were higher (CRP: 9.53 ± 3.79 vs. 7.45 ± 2.79 mg/L, $p = 0.001$; HbA1c: 6.81 ± 1.05% vs. 6.00 ± 0.76%, $p < 0.01$; cTnI: 0.30 ± 0.09 vs. 0.19 ± 0.09 µg/L, $p < 0.01$), suggesting additive inflammatory, metabolic and myocardial stress signals.

Univariate Logistic Regression Analysis

Variable encoding and univariate results are shown in Table 2. Several variables were associated with thrombosis in unadjusted analyses—namely, cTnI ≥0.2 µg/L, HbA1c ≥6.5%, CRP ≥10 mg/L, and intradialytic hypotension—while other covariates were not statistically significant.

Multivariate Logistic Regression and Nomogram Construction

After adjustment (Table 3), cTnI ≥0.2 µg/L (OR 3.69, 95% CI 1.33–10.21; $p = 0.012$), HbA1c ≥6.5% (OR 5.21, 95% CI 1.94–14.02; $p = 0.001$), CRP ≥10 mg/L (OR 6.65, 95% CI 2.44–18.10; $p < 0.01$) and intradialytic hypotension (OR 5.46, 95% CI 1.92–15.52; $p = 0.001$) remained significant. A nomogram integrating these predictors is shown in Fig. 1A. Predicted and observed risk demonstrated a good agreement (Fig. 1B; Hosmer–Lemeshow $p = 0.244$). The combined model showed strong discriminative performance with an area under the curve of 0.850 (95% CI

Table 2. Variable assignment and univariate logistic regression analysis.

Variable	Thrombosis (n = 25)	Control (n = 211)	OR (95% CI)	*p* value
cTnI $\geq$0.2 µg/L = 1	18 (72%)	100 (47.4%)	2.85 (1.14–7.12)	0.024
HbA1c $\geq$6.5% = 1	14 (56%)	59 (28%)	3.28 (1.41–7.63)	0.006
CRP $\geq$10 mg/L = 1	16 (64%)	60 (28.4%)	4.47 (1.87–10.68)	$<$0.01
Intradialytic hypotension = 1	19 (76%)	95 (45%)	3.87 (1.48–10.07)	0.006
Age $\geq$50 years = 1	12 (48%)	75 (35.5%)	1.67 (0.73–3.85)	0.226
Male = 1	10 (40%)	63 (29.9%)	1.57 (0.67–3.67)	0.302
BMI $\geq$24 kg/m^2 = 1	12 (48%)	70 (33.2%)	1.86 (0.81–4.29)	0.146
Hemoglobin $<$100 g/L = 1	14 (56%)	90 (42.7%)	1.71 (0.74–3.95)	0.208
WBC $\geq$10 × 10^9/L = 1	16 (64%)	104 (49.3%)	1.83 (0.77–4.32)	0.169
Platelet $<$100 × 10^9/L = 1	16 (64%)	109 (51.7%)	1.66 (0.70–3.93)	0.246
PT $>$12 s = 1	12 (48%)	94 (44.5%)	1.15 (0.50–2.64)	0.743
HDL-C $\leq$0.9 mmol/L = 1	8 (32%)	65 (30.8%)	1.06 (0.43–2.57)	0.903
LDL-C $\geq$3.1 mmol/L = 1	16 (64%)	147 (69.7%)	0.77 (0.32–1.84)	0.563
TG $\geq$1.7 mmol/L = 1	15 (60%)	133 (63%)	0.88 (0.38–2.05)	0.767
TC $\geq$5.7 mmol/L = 1	14 (56%)	134 (63.5%)	0.88 (0.37–2.05)	0.767
Diabetes = 1	10 (40%)	77 (36.5%)	1.16 (0.50–2.71)	0.731
Hypertension = 1	13 (52%)	97 (46%)	1.27 (0.56–2.92)	0.568
LMWH anticoagulation = 1	18 (72%)	172 (81.5%)	0.58 (0.23–1.49)	0.261

Continuous variables were dichotomized according to clinically relevant cut-off values based on routine laboratory reference ranges and commonly used criteria in clinical practice. All dichotomous variables were coded as 1 for the presence of the characteristic. TG, triglycerides; TC, total cholesterol; PT, Prothrombin Time.

Table 3. Multivariate logistic regression analysis of predictors of arteriovenous fistula (AVF) thrombosis.

Variable	β Coefficient	SE	Wald χ^2	OR (95% CI)	*p* value
cTnI $\geq$0.2 µg/L = 1	1.306	0.519	6.325	3.69 (1.33–10.21)	0.012
HbA1c $\geq$6.5% = 1	1.651	0.505	10.684	5.21 (1.94–14.02)	0.001
CRP $\geq$10 mg/L = 1	1.894	0.511	13.741	6.65 (2.44–18.10)	$<$0.01
Intradialytic hypotension = 1	1.698	0.533	10.165	5.46 (1.92–15.52)	0.001

0.741–0.960, Fig. 1C). DCA indicated a net clinical benefit across a wide range of threshold probabilities (Fig. 1D).

Bootstrapping Results

After performing 500 bootstrap resamples for internal validation, the optimism-corrected area under the curve was calculated to be 0.804 (95% CI 0.785–0.823, Fig. 2A). Furthermore, the bootstrap-corrected calibration curve demonstrated an acceptable agreement between predicted and observed outcomes (Fig. 2B). The bootstrap-corrected DCA also indicated a net clinical benefit across a wide range of threshold probabilities (Fig. 2C).

Discussion

Kidney disease remains a substantial public-health burden [1]. Each year, the number of individuals treated for kidney failure grows faster than the general population growth, and approximately 2.4 million deaths are attributed to kidney disease, making it the 11th leading cause of death globally [8]. In China, there were 632,653 patients receiving renal replacement therapy by 2019, with approximately 57% on hemodialysis [9]. Hemodialysis prolongs life [10], but long-term treatment hinges on a durable access. The AVF is generally considered the most reliable option [11]. Nonetheless, puncture problems, progressive luminal narrowing and vascular wall stiffening hasten thrombus formation and shorten access longevity, increasing clinical risk [12]. Standard diagnostic approaches—physical examination, duplex, angiography—identify failure after it has occurred; in contrast, a forward-looking tool is more valuable for prevention.

Neointimal hyperplasia is the lesion primarily implicated in AVF thrombosis [13]. Endothelial stress and disordered shear promote smooth-muscle proliferation and matrix deposition, leading to luminal encroachment. ESRD promotes systemic inflammation via acid-base imbalance, uremic toxins and immune dysregulation, while monocyte–macrophage infiltration and inflammatory mediators at the anastomosis accelerate hyperplasia [14]. CRP—a widely measured biomarker—reflects atherosclerosis across vascular beds [15], and platelet–complement crosstalk can further amplify thrombus growth [16]. Prior studies have identified CRP as an independent predictor of AVF stenosis/thrombosis (AUCs $>$0.70) [17]. Consistent with these findings, CRP remained an independent factor in our multivariable analysis.

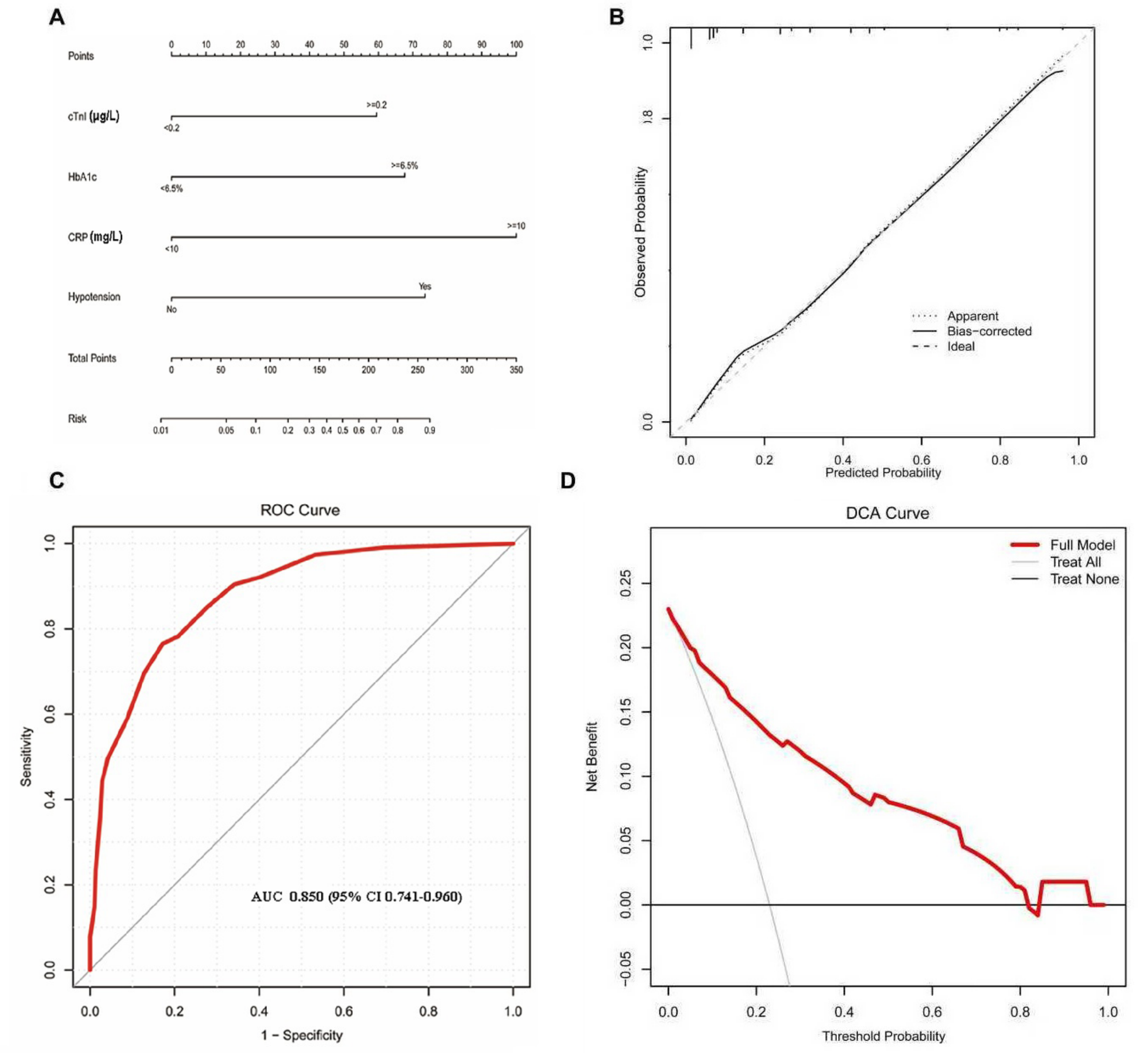

Fig. 1. Multivariate logistic regression and nomogram construction. (A) Nomogram integrating the four independent predictors. (B) Calibration curve. (C) ROC curve. (D) Decision curve analysis for the AVF thrombosis prediction model. ROC, receiver operating characteristic; AUC, area under the curve; DCA, decision curve analysis.

Intradialytic hypotension lowers arterial driving force and reduces access flow—conditions favoring stasis and clotting. Lower pre- and post-dialysis pressures have been associated with a higher thrombosis rate [18] and low flow presages occlusion [19]. In accesses without structural defects, hypotension alone has been estimated to account for 20%–40% of events [20]. Analyses with detailed comorbidity and event capture similarly link frequent hypotension or low predialysis pressure to adverse outcomes [21]. Intradialytic hypotension was significantly associated with AVF thrombosis in regression analysis. Pressure variability and post-dialysis hemoconcentration may further increase risk, both being mechanistically linked to endothelial dysfunction and intimal thickening [19].

HbA1c reflects non-enzymatic glycation of hemoglobin and scales with both the level and duration of hyperglycemia [22,23]. Beyond glucose control, HbA1c conveys cardiovascular hazard: in a large European cohort (~10,232 adults; ~6 years), each 1% increment is linked to 20%–30% higher cardiovascular events and all-cause mortality [24,25]. HbA1c relates to endothelial dysfunction, while thrombosis is closely associated with oxidative/endothelial injury [26]. However, in hemodialysis patients, HbA1c levels may be influenced by anemia, erythropoiesis-stimulating agent use, and shortened red blood cell lifespan, potentially leading to underestimation of true glycemic exposure. In addition, detailed data on short-term glycemic patterns, such as fasting or post-

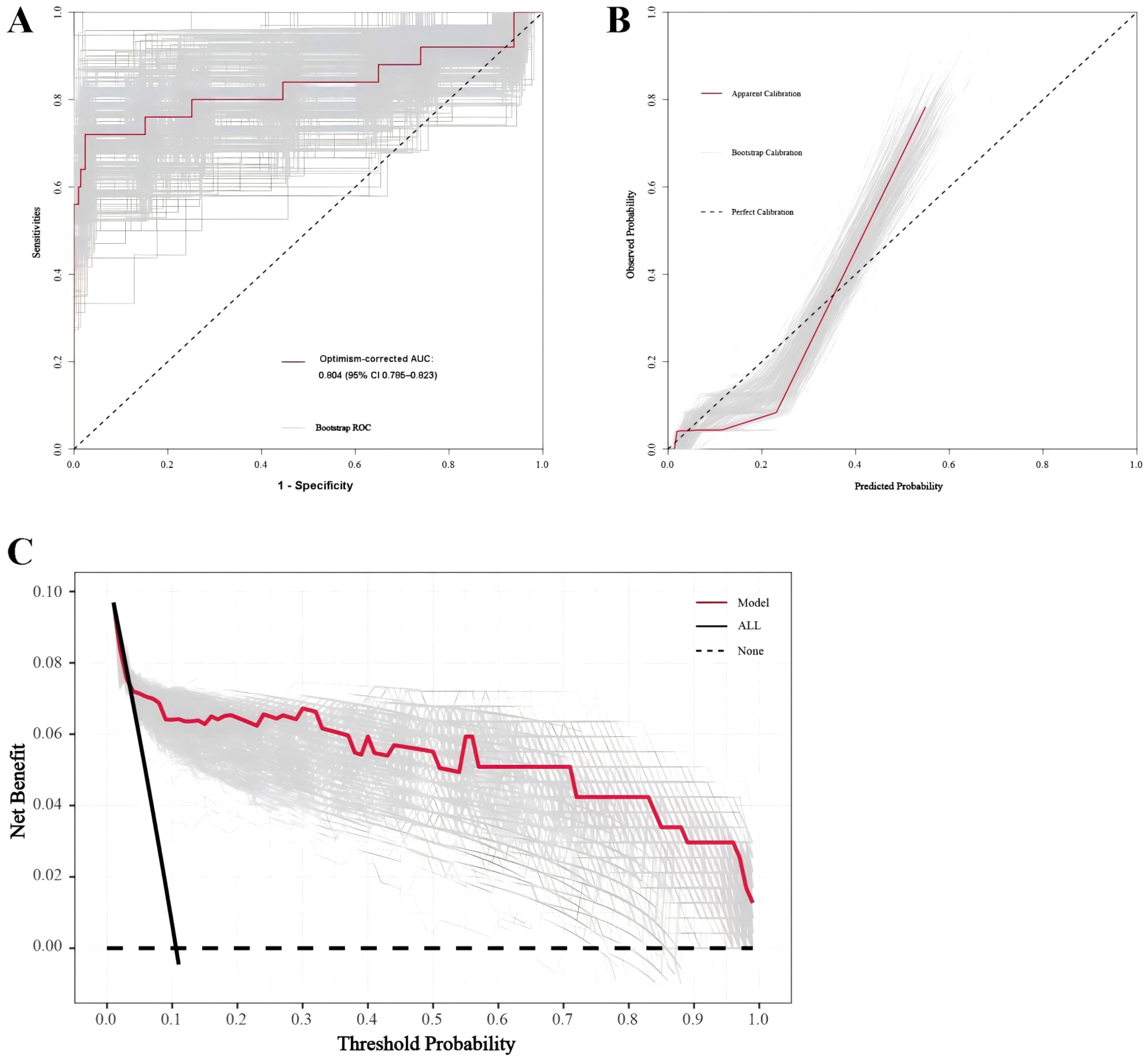

Fig. 2. Internal validation using bootstrap resampling (500 repetitions). (A) Time-dependent receiver-operating characteristic curves showing the discriminative performance of the combined predictor. (B) Calibration curves demonstrating an agreement between predicted and observed probabilities; the apparent and bootstrap-corrected curves are shown alongside the perfect calibration line. (C) Decision curve analysis plotting the net benefit across a range of threshold probabilities. The black solid line represents the "treat all" strategy, the black dashed lines represent the "treat none" strategy, and the red lines represent the combined prediction model.

prandial glucose levels and glycemic variability indices, were not available in this retrospective cohort. Glycemic fluctuations may play an important role in vascular injury and thrombosis; therefore, the use of HbA1c alone may not fully capture the complexity of glycemic effects on AVF outcomes. Cardiac troponins (cTnC, cTnI, cTnT) are key regulators of contraction; circulating troponin is normally minimal but rises in response to myocardial injury [27]. Owing to unique epitopes and high analytical performance, cTnI/cTnT outperform older markers in diagnosing myocardial injury and prognosis assessment [28]. In ESRD patients, cardiovascular events are frequent, and even in asymptomatic MHD patients, elevated cTnI levels can reflect dialysis-related myocardial stress [29]. In addition, elevated cTnI levels in hemodialysis patients may also be influenced by non-thrombotic factors, including chronic myocardial injury, volume overload, and reduced renal clearance, which were not systematically excluded in this retrospective cohort. Therefore, cTnI in this study should be interpreted as a marker of overall cardiovascular and systemic stress rather than a specific indicator of thrombotic activity, which may affect its predictive specificity.

In our cohort, HbA1c and cTnI remained independent predictors in the multivariable analysis. When combined with CRP and intradialytic hypotension, the overall model showed good discriminative performance, underscoring the complementary roles of inflammatory, hemodynamic, metabolic, and myocardial stress pathways. Because all four predictors are routinely measured in dialysis practice, the nomogram can be integrated with minimal disruption. A monthly audit could (i) calculate risk at chairside; (ii) trigger earlier duplex in high-risk patients; (iii) review anticoagulation protocols; (iv) stabilize intradialytic pressures (dry-weight assessment, ultrafiltration profiling); and (v) tighten glycemic management with feedback to diabetology and to assess whether risk-guided interventions translate into fewer thrombotic events.

The model relies on simple, inexpensive measures and shows encouraging calibration and net benefit in this single-center study. However, these findings require validation in larger, externally validated cohorts before consideration for clinical use. It is a single-center study with modest size and a 12-month follow-up; long-term patency and external generalizability remain to be tested. In addition, only patients with a single AVF configuration (cephalic vein–radial artery end-to-side anastomosis) were included, which may limit the applicability of the findings to other AVF types or surgical techniques. Residual confounding (e.g., variation in cannulation technique) cannot be excluded. Several established determinants of vascular access outcomes–particularly detailed vascular anatomical characteristics and cannulation techniques–were not available in this retrospective dataset and may have influenced AVF outcomes, potentially introducing unmeasured confounding. In addition, although clinical protocols were generally stable during the enrollment period, unmeasured temporal variations in patient management over time may have introduced potential heterogeneity. Intradialytic hypotension was treated as a binary baseline variable, which may not fully capture its temporal variability or cumulative exposure over time, and this simplification may have led to residual misclassification. Furthermore, the relatively small number of thrombotic events may limit statistical power and increase the risk of model overfitting, which warrants cautious interpretation of the predictive performance. This may also have led to inflated effect-size estimates and wider confidence intervals for some predictors, and therefore, the magnitude of the reported odds ratios should be interpreted with caution. Future investigations should validate the tool across centers, explore time-updated biomarkers, and prospectively evaluate whether risk-guided strategies lower thrombosis and extend access longevity.

Conclusion

CRP, intradialytic hypotension, HbA1c, and cTnI are independently associated with AVF thrombosis in patients undergoing maintenance hemodialysis. A nomogram based on these routinely available variables shows moderate predictive performance in this single-center cohort. However, as this was an observational study, the findings reflect risk association rather than causal inference or proven clinical benefit. Further prospective and multicenter studies are needed before clinical implementation can be considered.

Availability of Data and Materials

Data are available from the corresponding author upon reasonable request.

Author Contributions

CYL and YBZ designed the study, performed analyses and drafted the manuscript. LW and LFL collected data and contributed to statistics. LW and LFL coordinated follow-up and aided interpretation. LW and LFL participated in the drafting or key revisions of the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work to take public responsibility for appropriate portions of the content and agreed to be accountable for all aspects of the work in ensuring that questions related to its accuracy or integrity.

Ethics Approval and Consent to Participate

Approved by the Ethics Committee of The Quzhou Affiliated Hospital of Wenzhou Medical University (Quzhou, China; Approval No. QSRMH2024-083). All procedures adhered to the Declaration of Helsinki. Written informed consent was obtained from all participants before enrollment.

Acknowledgment

We thank all participating patients and the nursing/technical staff of the Department of Nephrology, Quzhou People's Hospital, for their cooperation.

Funding

This work was supported by the Guiding Science and Technology Project of Quzhou City (Project No. 2023ZD051).

Conflict of Interest

The authors declare no conflict of interest.

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