Clinical Interventions in Aging

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ORIGINAL RESEARCH

Developing a LASSO Regression-Based Nomogram to Predict Outcomes in Abdominal Aortic Aneurysm Patients Post-Endovascular Aneurysm Repair

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Introduction: This study aimed to develop a prognostic model to predict outcomes in patients undergoing endovascular aneurysm repair (EVAR) for abdominal aortic aneurysms (AAA).

Methods: 304 participants were divided into training and validation sets in a 7:3 ratio. Six risk factors were identified using LASSO regression, univariate, and multivariate Cox regression analyses: history of stroke, CIA atherosclerosis, age, hemoglobin levels, monocyte count, and large AAA. A nomogram was constructed to predict 1-year and 3-year all-cause mortality (ACM).

Results: A total of 304 AAA patients who underwent EVAR were included in this study (84.87% male; median age 72 [IQR: 65–77] years). The model showed good predictive performance, with area under the curve (AUC) values of 0.84 (95% CI: 0.79–0.89) and 0.81 (95% CI: 0.76–0.86) for 1-year and 3-year mortality in the training set, and 0.71 (95% CI: 0.62–0.80) and 0.80 (95% CI: 0.73–0.87) in the validation set.

Discussion: These results suggest the model's effectiveness in aiding clinicians with risk stratification and tailoring treatment strategies for post-EVAR patients.

Keywords: abdominal aortic aneurysm, prognosis, endovascular aneurysm repair, nomogram

Introduction

An abdominal aortic aneurysm (AAA) is a medical condition characterized by the progressive and irreversible expansion of the aorta. AAA is defined as an enlargement greater than 3 cm or 1.5 times the size of a normal aortic size, typically identified through computed tomography (CT) scans or intravascular ultrasonography. The prevalence of AAA is about 5.5 occurrences per 10,000 males and 1.1 per 10,000 females.[1–3] AAAs can grow over time and may eventually rupture, accounting for 1%–2% of the total mortality rates.[4] Surgical intervention is often necessary for large AAAs due to the significant risk of rupture. Recently, endovascular aneurysm repair (EVAR) has emerged as a preferred treatment option for AAA patients because it is less invasive and generally requires a shorter hospital stay.[5] However, the risk factors associated with mortality for patients undergoing EVAR have yet to be thoroughly examined.

Received: 31 August 2024
Accepted: 18 July 2025
Published: 28 November 2025

Certain features are associated with worse outcomes in EVAR patients, including large preoperative AAA diameter, infrarenal neck diameter, and aortic thrombus.[6,7] While previous studies have explored demographic and morphological risk models, focusing on factors such as gender and cardiovascular history,[8,9] there has been limited emphasis on laboratory data. To bridge this gap, we utilized LASSO regression, a method adept at simplifying models and avoiding multicollinearity, making it particularly suitable for clinical application. This approach established a more streamlined and interpretable model, which could assist clinicians in efficiently identifying high-risk EVAR patients. Consequently, in this article, we developed and validated a nomogram with LASSO Cox regression analysis to predict the all-cause mortality (ACM) of AAA in post-EVAR patients.

Materials and Methods

Study Population

AAA patients were enrolled from March 2015 to May 2023 at the First Affiliated Hospital of Zhengzhou University. The inclusion criteria were as follows: (1) AAA diagnosed with computed tomographic angiography (CTA) reports indicating an abdominal aortic diameter larger than 3 cm or 1.5 times the size of a normal aorta, (2) patients undergoing EVAR during hospitalization, and (3) patients ≥ 18 years old. The exclusion criteria were as follows: (1) known history of tumor and immune disease, (2) Marfan syndrome, (3) failed follow-up, (4) ruptured AAA, (5) receiving open surgery. A total of 304 AAA patients were included in this study and were randomly divided into training and validation sets in a 7:3 ratio. The training set was used to construct the nomogram to enhance model accuracy, while the testing set was used for validation. The flowchart of the study is illustrated in Figure 1.

The patients underwent standard preoperative evaluation and EVAR procedure with commercially available infrarenal aortic endograft according to clinical guidelines. All procedures included in this analysis were elective EVAR; emergency procedures for ruptured AAA were excluded due to their distinct clinical characteristics and outcomes.

Endpoint and Follow-Up

The primary endpoint of this study was ACM. The median follow-up time of the study was 24 (16–33) months, and the follow-up data on patients' conditions was obtained through clinic visits. All data involved in the study was collected and preserved in a manner ensuring that none of the subjects could be identified. The study protocol was approved by the ethics committee of the First Affiliated Hospital of Zhengzhou University.

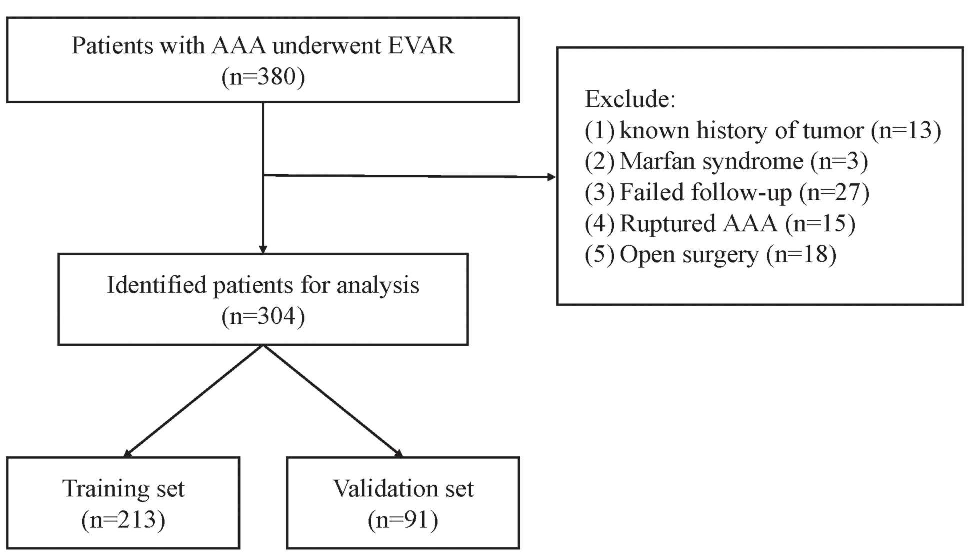

Figure 1 Flowchart of Patient Selection and Dataset Division for Analysis.

Clinic and Demographic Characteristics

Blood samples were collected from fasting patients at least 8 hours before EVAR. The collected demographic data included gender, age, smoking status, drinking status and medication history.

Coronary artery disease (CAD) was characterized by a documented history of angina, myocardial infarction, or procedures aimed at coronary revascularization. Similarly, diabetes, hypertension, a prior occurrence of stroke, and chronic obstructive pulmonary disease (COPD) were defined based on previous diagnoses and respective treatment.

The measured laboratory data, including white blood cell (WBC), hemoglobin, neutrophil, lymphocyte, monocyte, low-density lipoprotein cholesterol (LDL-C), fasting blood glucose (FBG), and estimated glomerular filtration rate (eGFR), were collected on the first day of hospitalization after 8-hour fasting.

CTAs were performed with scanners in the First Affiliated Hospital of Zhengzhou University. Data obtained from three-dimensional CTA images included common iliac artery (CIA) atherosclerosis, intraluminal thrombus (ILT) in the aneurysm, AAA diameter, AAA volume, and neck angulation based on the standard measurement method.[10] Large AAA was defined as 5.5 cm in males and 5 cm in females. We define a current smoker as an individual who has smoked more than five packs in their lifetime and currently smokes on some days or every day.

Model Construction

In this study, we employed the LASSO regression method to identify key predictors of ACM in post-EVAR patients. LASSO regression performs both variable selection and regularization, improving model accuracy and interpretability. The λ parameter, a critical component of the LASSO method that controls the strength of the penalty applied to the regression coefficients, was selected using a 10-fold cross-validation approach. This technique involves partitioning the dataset into 10 subsets and iteratively training the model on 9 subsets while validating it on the remaining subset to optimize predictive accuracy and prevent overfitting. The choice of λ directly influences the model's robustness: a higher λ results in greater regularization, potentially excluding more variables to create a simpler and more generalizable model. In contrast, a lower λ might include more variables, improving the model fit but at the risk of capturing noise and overfitting. The final λ value was chosen to achieve a balance between model complexity and predictive power, as demonstrated by the minimized mean squared error observed during cross-validation.

Cox regression analyzes the time to an event while accounting for various covariates. It was employed to determine which selected risk factors independently predict ACM in post-EVAR patients. Parameters with a P value less than 0.05 in the univariate Cox regression analysis were then included in the multivariate Cox regression analysis. To refine the multivariable Cox regression model, we employed the Akaike Information Criterion (AIC)-stepwise method for model selection. The AIC-stepwise approach involves iteratively adding or removing variables based on their contribution to the model's overall fit, as measured by the AIC score. The AIC balances model complexity and goodness-of-fit by penalizing the inclusion of additional variables that do not significantly enhance predictive power. Variables were sequentially added or removed from the model based on whether their inclusion or exclusion improved the AIC score, continuing until the model with the lowest AIC was identified. This stepwise process ensured that the final model was both parsimonious and robust, including only those variables that provided independent and significant contributions to the prediction of ACM.

Time-dependent receiver operating characteristic (ROC) curves and the area under the curve (AUC) assess the model's ability to discriminate between different outcomes. They were used to evaluate the predictive accuracy of the nomogram for 1-year and 3-year ACM in post-EVAR patients.

Youden's Index is a statistic used to determine the optimal cutoff point for a diagnostic test. It is defined as $J = \mathit{Sensitivity} + \mathit{Specificity} - 1$, where sensitivity is the true positive rate and specificity is the true negative rate. The index ranges from 0 to 1, with higher values indicating better test performance. The optimal cutoff maximizes Youden's Index, balancing sensitivity and specificity to distinguish between different patient outcomes effectively. We plotted calibration curves to analyze the concordance between predicted mortality and observed ACM events, using bootstrapping with 1000 resamples.

 https://doi.org/10.2147/CIA.S493841

Statistical Analysis

The Kolmogorov–Smirnov test was employed to assess normal distribution, all the continuous variables were not normally distributed, therefore Mann–Whitney *U*-tests were used for group comparison. Kaplan-Meier (KM) analysis was employed to demonstrate the risk stratification ability of the model. Decision curve analysis (DCA) was utilized to estimate clinical utility. Post hoc power analysis is used to evaluate the adequacy of sample size. All analyses and model development were performed using R Version 4.1.2, and the packages used for analysis are listed in Supplementary Table 1. A two-sided p-value < 0.05 was considered statistically significant.

Results

Patient Characteristics

A total of 304 patients were enrolled in the study based on the exclusion and inclusion criteria. The median age of the population was 72 (65–77), including 46 (15.13%) females and 258 (84.87%) males. 32 (10.53%) and 187 (61.51%) patients were diagnosed with type 2 diabetes (T2DM) and hypertension, respectively. 54 (17.76%) patients had a history of stroke. 159 (52.30%) and 113 (37.17%) patients were diagnosed with CIA atherosclerosis and large AAA based on CTA results. The overall dataset was divided into the training (n = 213) set and validation set (n = 91). There were no significant differences observed between the two datasets (Table 1), suggesting random and reasonable grouping.

There was no endoleak reported during hospitalization. During the follow-up period, 46 ACM events were reported (Table 2). Among them, 30 (65.22%) were related to aneurysm and 6 (13.04%) were linked to cardiac death. Notably, the

Table 1 Clinical Characteristics of Patients in Training and Validation Cohorts

Variables	Training Set n=213	Validation Set n=91	*P*
Demographic data (%)			
Female	36 (16.90)	10 (10.99)	0.223
Age (year)	71.00 (65.00, 77.00)	72.00 (65.50, 76.50)	0.992
Smoking	99 (46.48)	47 (51.65)	0.901
Drinking	63 (29.57)	24 (26.37)	0.678
ACM	33 (15.49)	13 (14.29)	0.863
Comorbidity and surgical intervention (%)			
Diabetes	25 (11.74)	7 (7.69)	0.684
Stroke	34 (15.96)	18 (19.78)	0.873
Hypertension	131 (61.50)	56 (61.54)	0.201
CAD	64 (30.05)	33 (36.26)	0.893
COPD	8 (3.76)	2 (2.20)	0.729
Hypogastric artery intervention	20 (9.39)	11 (12.09)	0.512
Medication (%)			
ACEI	43 (20.19)	19 (20.88)	0.878
β-blocker	57 (26.76)	29 (31.87)	0.749
CCB	90 (42.25)	42 (46.15)	0.380
Statin	80 (37.56)	45 (49.45)	0.309
Laboratory data			
WBC (10^9/L)	6.60 (5.59, 8.59)	7.00 (5.48, 9.09)	0.452
Hemoglobin (g/L)	124.00 (109.00, 137.00)	125.50 (116.40, 138.00)	0.767
Monocyte (10^9/L)	0.50 (0.37, 0.67)	0.53 (0.37, 0.66)	0.718
Neutrophil (10^9/L)	4.47 (3.51, 6.27)	4.56 (3.42, 6.12)	0.976
Lymphocyte (10^9/L)	1.37 (0.94, 1.72)	1.35 (1.01, 1.86)	0.347
FBG (mmol/L)	5.01 (4.55, 5.90)	5.01 (4.45, 5.80)	0.567
LDL-C (mmol/L)	2.29 (1.79, 2.94)	2.27 (1.81, 2.88)	0.645

(*Continued*)

Table 1 (Continued).

Variables	Training Set n=213	Validation Set n=91	*P*
eGFR (mL/min/1.73 m^2)	82.49 (64.00, 92.31)	81.89 (66.39, 91.60)	0.792
Imaging data			
ILT (%)	107 (50.23)	49 (53.85)	0.261
CIA Atherosclerosis (%)	113 (53.05)	46 (50.55)	0.170
Large AAA (%)	76 (35.68)	37 (40.66)	0.438
AAA neck angle (°)	40.10 (36.15, 55.30)	45.60 (31.60, 59.60)	0.568
AAA diameter (mm)	47.60 (40.50, 63.00)	51.70 (41.05, 64.55)	0.199
AAA volume (mm^3)	61.80 (38.84, 115.04)	63.20 (38.87, 110.40)	0.505

Abbreviations: ACM, all-cause mortality; ACEI, angiotensin-converting enzyme inhibitor; CAD, coronary artery disease; CCB, calcium entry blockers; COPD, chronic obstructive pulmonary disease; CIA, common iliac artery; eGFR, estimated glomerular filtration rate; WBC, white blood cell; FBG, fasting blood glucose; LDL-C, low density lipoprotein cholesterol; ILT, intraluminal thrombus; AAA, abdominal aortic aneurysm.

Table 2 Clinical Characteristics of Patients with or Without ACM Events

Variables	Total n=304	Non-ACM n=258	ACM n=46	*P*
Demographic data (%)				
Female	46 (15.13)	38 (14.73)	8 (17.39)	0.656
Age (year)	72.00 (65.00, 77.00)	70.00 (64.25, 76.00)	75.00 (71.00, 79.00)	0.001
Smoking	146 (48.03)	124 (48.06)	22 (47.83)	0.322
Drinking	87 (28.62)	73 (28.29)	14 (30.43)	0.86
Comorbidity and surgical intervention (%)				
Diabetes	32 (10.53)	28 (10.85)	4 (8.70)	0.799
Stroke	54 (17.76)	40 (15.50)	14 (30.43)	0.011
Hypertension	187 (61.51)	155 (60.08)	32 (69.57)	0.252
CAD	97 (31.91)	80 (31.01)	17 (36.96)	0.492
COPD	10 (3.29)	8 (3.10)	2 (4.35)	0.651
Hypogastric artery intervention	31 (10.20)	27 (10.47)	4 (8.70)	0.679
Medication (%)				
ACEI	62 (20.39)	54 (20.93)	8 (17.39)	0.693
β-blocker	86 (28.29)	72 (27.91)	14 (30.43)	0.725
CCB	132 (43.42)	111 (43.02)	21 (45.65)	0.749
Statin	125 (41.12)	109 (42.25)	16 (34.78)	0.417
Laboratory data				
WBC (10^9/L)	6.70 (5.50, 8.60)	6.70 (5.50, 8.45)	7.95 (6.23, 10.04)	0.013
Hemoglobin (g/L)	125.00 (110.15, 137.00)	126.80 (116.00, 138.00)	108.50 (90.65, 122.75)	<0.001
Monocyte (10^9/L)	0.51 (0.37, 0.67)	0.50 (0.37, 0.64)	0.61 (0.42, 0.84)	0.015
Neutrophil (10^9/L)	4.53 (3.50, 6.16)	4.36 (3.47, 5.90)	5.28 (3.62, 8.16)	0.021
Lymphocyte (10^9/L)	1.36 (1.00, 1.73)	1.38 (1.01, 1.73)	1.21 (0.89, 1.64)	0.28
FBG (mmol/L)	5.01 (4.51, 5.90)	4.99 (4.46, 5.89)	5.32 (4.75, 5.94)	0.091
LDL-C (mmol/L)	2.29 (1.80, 2.92)	2.29 (1.79, 2.92)	2.33 (1.84, 2.91)	0.624
eGFR (mL/min/1.73 m^2)	82.46 (64.73, 92.30)	82.67 (66.36, 92.30)	80.88 (56.47, 89.83)	0.387
Imaging data				
ILT (%)	156 (51.32)	121 (46.90)	35 (76.09)	<0.001
CIA Atherosclerosis (%)	159 (52.30)	122 (47.29)	37 (80.43)	<0.001
Large AAA (%)	113 (37.17)	87 (33.72)	26 (56.52)	0.005

(*Continued*)

Table 2 (Continued).

Variables	Total n=304	Non-ACM n=258	ACM n=46	*P*
AAA neck angle	44.50 (33.62, 57.62)	43.90 (31.68, 57.68)	46.35 (37.23, 56.25)	0.478
AAA diameter	49.20 (40.65, 63.12)	48.05 (39.52, 59.93)	56.65 (45.98, 69.80)	0.006
AAA volume (mm^3)	62.15 (38.81, 114.95)	60.14 (38.04, 109.63)	98.90 (47.59, 157.26)	0.003

Abbreviations: ACM, all-cause mortality; ACEI, angiotensin-converting enzyme inhibitor; CAD, coronary artery disease; CCB, calcium entry blockers; COPD, chronic obstructive pulmonary disease; CIA, common iliac artery; eGFR, estimated glomerular filtration rate; WBC, white blood cell; LDL-C, low density lipoprotein cholesterol; ILT, intraluminal thrombus; AAA, abdominal aortic aneurysm.

rate of stroke in the ACM group (30.43%) was significantly higher than in the non-ACM group (15.50%). The WBC, and monocyte levels were significantly higher, while the hemoglobin level was lower. Moreover, ACM was inclined to occur in patients with advanced age, ILT, CIA atherosclerosis, larger AAA diameter and volume, which was consistent with our assumptions.

Variable Analysis and Selection

LASSO regression was used to select variables, effectively addressing multicollinearity and overfitting, and enhancing model interpretability. Figure 2a showed the variation of the parameter coefficients. The model variables were reduced to seven upon achieving the minimum mean squared error of log(λ), and further reduced to five when it reached a standard error (onefold SE) of the minimum distance, as determined by the 10-fold cross-validation (Figure 2b). We preferred to choose the optimal log(λ) and involve seven parameters in the Cox regression analysis.

Parameters with $P < 0.05$ in the univariable Cox analysis were entered into the multivariable Cox analysis (Table 3), and the six following variables were independent risk factors of ACM: history of stroke (HR=3.59, 95% CI: 1.67–7.70), CIA atherosclerosis (HR=4.48, 95% CI: 1.92–10.49), age (HR=1.54, 95% CI: 1.07–2.23), hemoglobin (HR=0.53, 95% CI: 0.37–0.76), monocyte (HR=1.28, 95% CI: 1.11–1.47), and large AAA (HR=3.09, 95% CI: 1.43–6.72).

Nomogram Construction

Based on the six variables, a prognostic nomogram predicting 1- and 3-year mortality for post-EVAR patients was developed with the rms package (Figure 3a). In the nomogram, distinct points on the scale were assigned to each level of

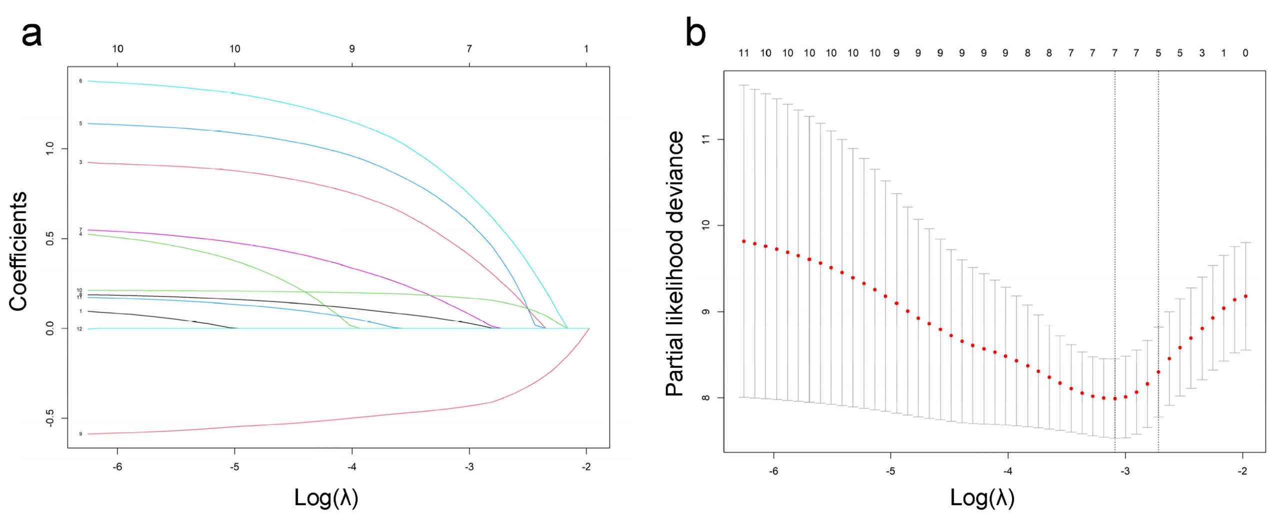

Figure 2 Regularization Path and Model Selection for LASSO Regression. (**a**) Parameter coefficient variation plot. (**b**) Ten-fold cross-validation identifying optimal log(λ) values.

Table 3 Univariable and Multivariable Cox Analysis Results

Variable	Univariable Analysis		Multivariable Analysis	
	HR (95% CI)	*P* value	HR (95% CI)	*P* value
Large AAA	3.43(1.73–6.8)	<0.001	3.09(1.43–6.72)	0.004
Stroke	3.44(1.65–7.18)	0.001	3.59(1.67–7.7)	0.001
CIA Atherosclerosis	5.05(2.16–11.81)	<0.001	4.48(1.92–10.49)	<0.001
Age	1.81(1.23–2.66)	0.003	1.54(1.07–2.23)	0.021
WBC	1.42(1.14–1.77)	0.002		
Hemoglobin	0.42(0.3–0.59)	<0.001	0.53(0.37–0.76)	<0.001
Monocyte	1.27(1.14–1.42)	<0.001	1.28(1.11–1.47)	<0.001

Abbreviations: AAA, abdominal aortic aneurysm; ACM, all-cause mortality; CIA, common iliac artery; WBC, white blood cell.

the variables. To use the nomogram, the variable of interest was initially located on the horizontal axis of the nomogram, and a straight line was drawn vertically from the variable's value to the corresponding point on the vertical axis of the nomogram. This process was repeated for each variable included in the model. Once the points for all variables were determined, a cumulative score was derived for each patient by summing the assigned points, and the estimated probabilities of ACM events within the 1- and 3-year timeframes were determined.

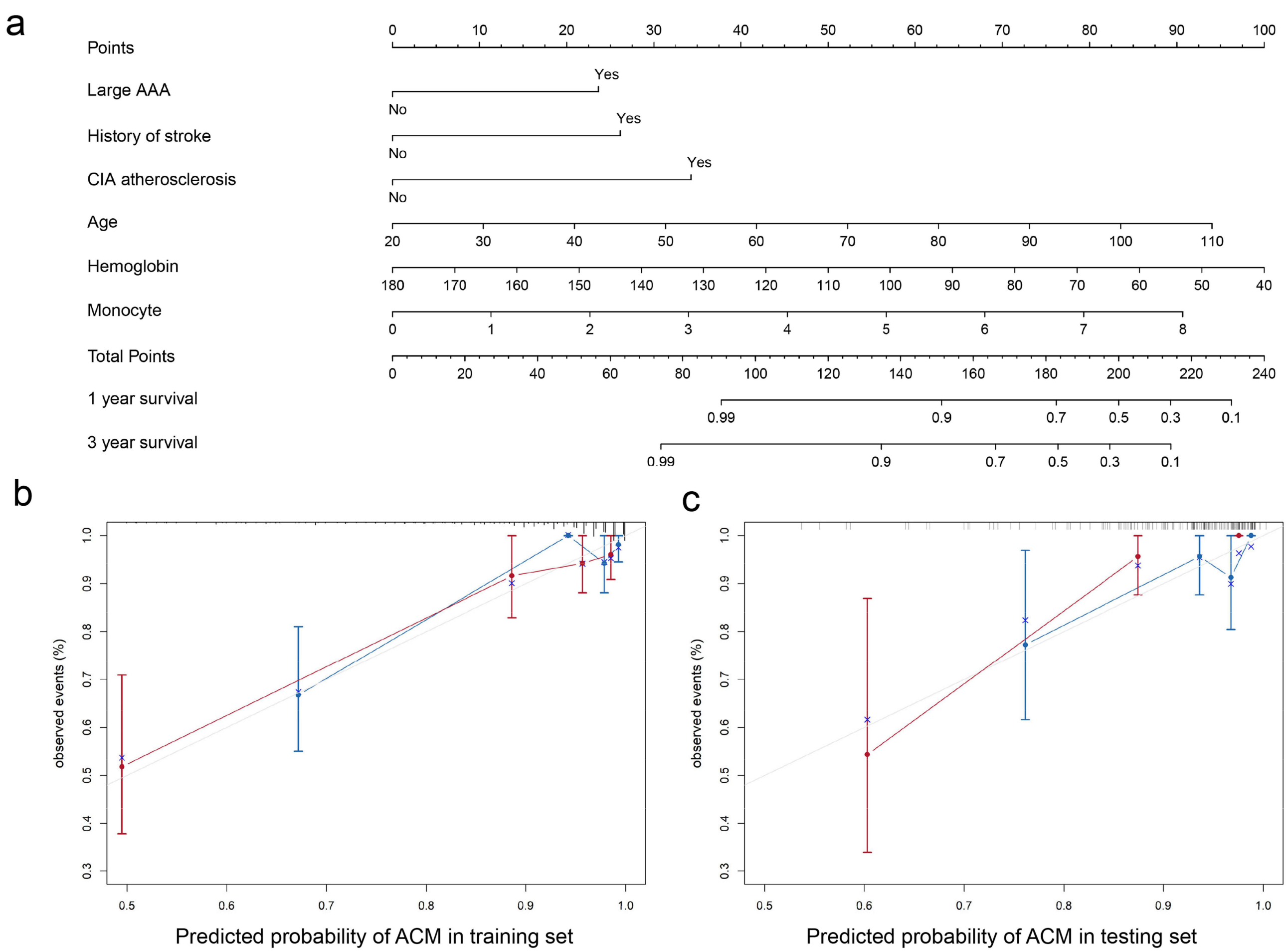

Figure 3 Nomogram for Predicting Survival (**a**) and Calibration Plots for Training (**b**) and Validation Cohorts (**c**).

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Model Performance and Clinical Utility

In the internal validation cohort, 13 ACM events (14.39%) were observed during the follow-up period. Calibration curves demonstrated strong concordance between predicted and observed ACM probabilities in both training and validation cohorts (Figure 3b and c), confirming the nomogram's reliability. Time-dependent ROC curves revealed excellent discriminative performance with AUCs at years 1 and 3 of 0.84 (95% CI: 0.79–0.89) and 0.81 (95% CI: 0.76–0.86) in the training set (Figure 4a), and 0.71 (95% CI: 0.62–0.80) and 0.80 (95% CI: 0.73–0.87) in the validation set (Figure 4b).

We employed Youden's Index to determine the optimal cutoff score (155.1) in the total dataset and stratified patients into high- and low-risk groups. Kaplan-Meier curves demonstrated satisfactory discrimination in both training (Figure 5a) and testing sets (Figure 5b) with statistically significant differences between risk groups (log-rank $P<0.05$).

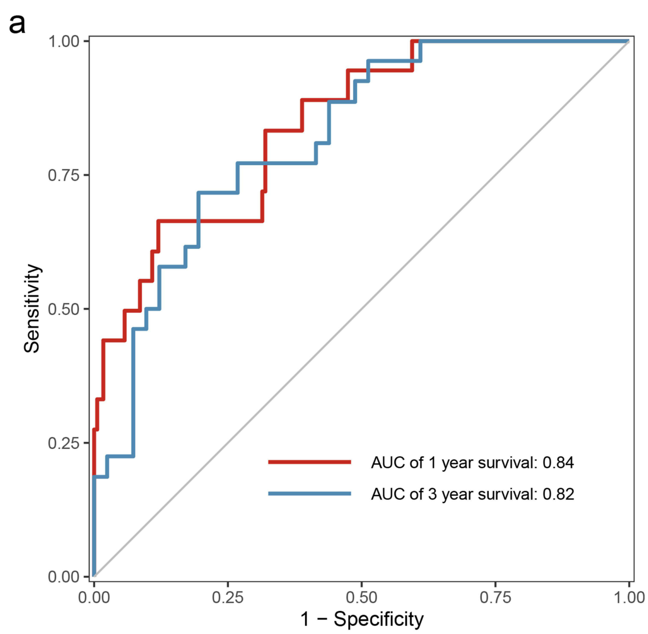

Figure 4 ROC Curves for 1-Year (**a**) and 3-Year (**b**) Survival Predictions in Training and Testing Sets.

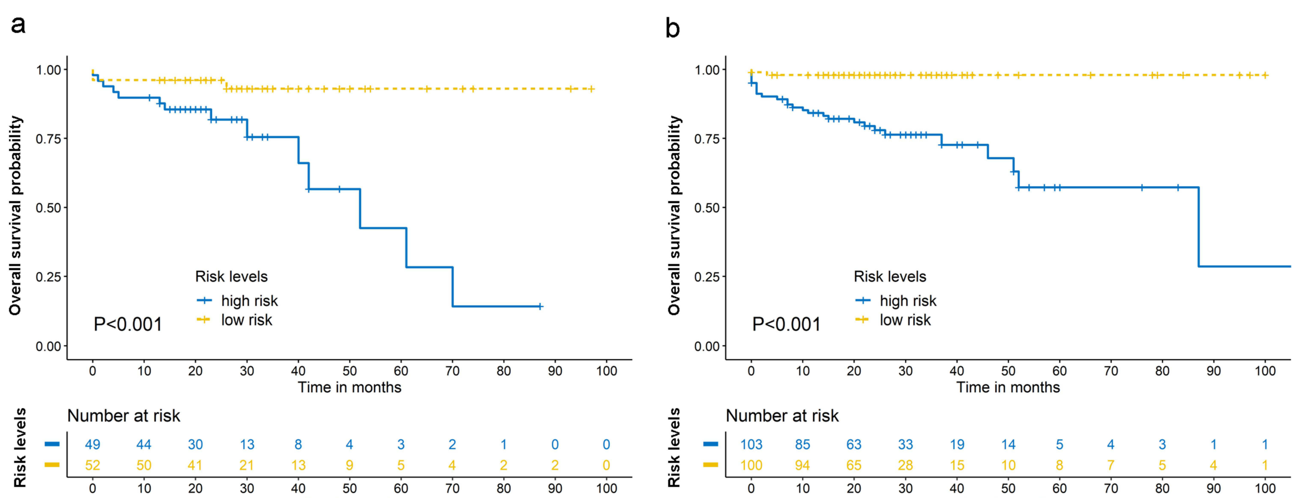

Figure 5 Kaplan-Meier Survival Curves for High and Low Risk Groups in Training (**a**) and Testing (**b**) Sets.

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Decision curve analysis (DCA) confirmed the clinical utility of our model, demonstrating effective prediction of 1- and 3-year survival (Figure 6). The model's calibration was assessed using time-dependent Brier scores, which were 0.0691 at 1 year and 0.1136 at 3 years—both lower than the Kaplan-Meier reference model (0.0759 and 0.1203, respectively). These results indicate superior predictive accuracy with good calibration across both short-term and long-term follow-up periods.

A post hoc power analysis based on CIA atherosclerosis—one of the most significant predictors in the final multivariable Cox model (HR = 4.48)—confirmed adequate statistical power. Given the observed event rates (80.43% in the ACM group vs 47.29% in the non-ACM group), the calculated power exceeded 99% using a two-sided test with $\alpha = 0.05$, confirming sufficient sample size for reliable conclusions.

Discussion

With the increasing population of patients undergoing EVAR, there is a growing need for predictive tools as well. This study employed LASSO-Cox regression analysis to develop a predictive model based on data from our center. Six parameters were associated with ACM events, and these included a history of stroke, CIA atherosclerosis, age, hemoglobin, monocyte, and large AAA. A nomogram was constructed based on those factors to predict outcomes for AAA patients after EVAR, which was validated in internal validation cohorts.

AAA has long posed a significant global health burden due to its progressive nature and the high mortality rate associated with ruptured AAA (rAAA). Numerous studies have investigated the short-term outcomes of patients undergoing endovascular aneurysm repair (EVAR), particularly in emergency settings. For instance, Ye et al analyzed 1124 AAA patients and 6524 healthy controls using a multi-locus genetic risk score linked to AAA incidence and expansion.[11] In a study by Tambyraja et al, clinical indicators such as hemoglobin < 9 g/dL, shock (blood pressure < 90 mmHg), and a Glasgow Coma Scale score < 15 were identified as sensitive markers for assessing the health status of rAAA patients and were associated with perioperative mortality, though this model has yet to be validated.[12]

The choice of surgical intervention for rAAA also significantly impacts patient outcomes. Dittman et al found that patients with a history of aortic repair exhibited higher mortality and morbidity when treated with open repair,

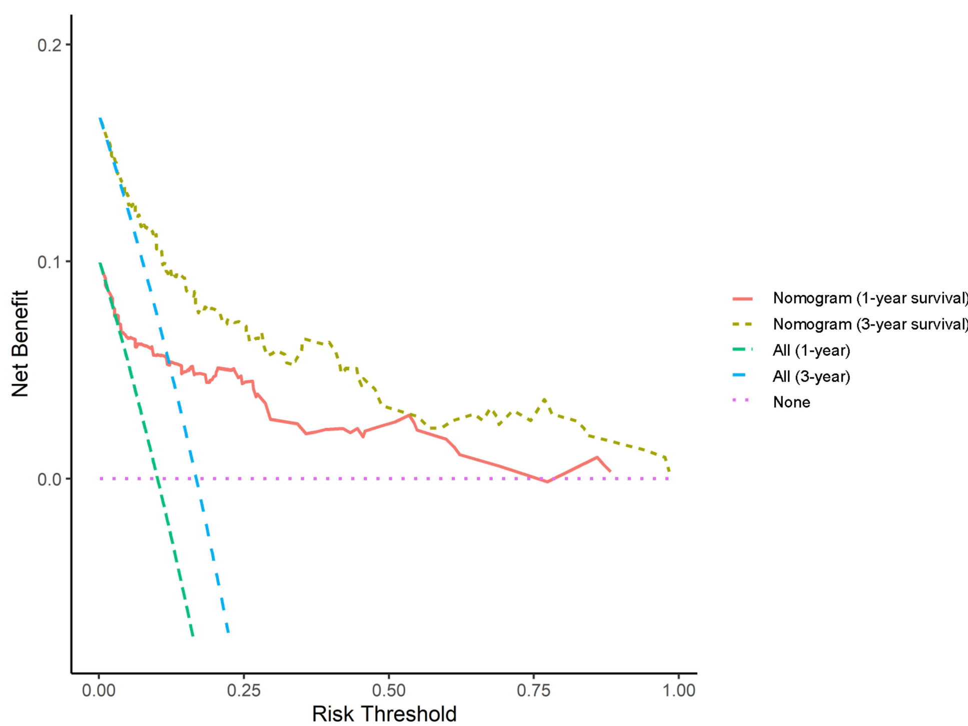

Figure 6 Decision Curve Analysis for 1-Year and 3-Year Survival Predictions Using Nomogram.

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recommending EVAR as the preferred approach in rupture cases.[13] A meta-analysis conducted by Krisna Pertiwi et al demonstrated that EVAR, especially fenestrated-EVAR, improved short-term outcomes compared to open surgery, though it did not show superiority in long-term outcomes.[14] Another meta-analysis reported similar findings, highlighting EVAR's advantage in reducing peri-operative mortality.[15]

In addition to surgical strategies, the development of predictive tools and models is critical in improving AAA management. Zhou et al generated a nomogram based on morphological features, including hostile aneurysm neck, conical neck, and angulated neck, with moderate discrimination ability (AUC: 0.81, 95% CI: 0.73–0.89).[16] Another study, which involved 205 patients with highly angulated necks, analyzed risk factors for endograft-related complications, such as increasing proximal neck diameter and decreasing seal zone inner curve length.[17] The case-control study conducted by Memon et al identified myeloperoxidase as a promising prognostic factor, with an AUC of 0.71.[18] Liu et al developed a nomogram to predict type II endoleak after EVAR,[19] while Zhao et al assessed risk factors for progression of sub-aneurysmal aorta.[20] Another model proposed by Liu et al incorporated both morphological and biomechanical indices to predict AAA rupture risk.[21]

However, current nomograms and predictive models mostly rely on demographic, anatomical, or procedural variables, often overlooking laboratory biomarkers that may reflect real-time physiological and inflammatory states. Some models also lack interpretability or integration into clinical workflows, reducing their utility in clinical decision-making.

In our study, the new predictive model incorporates laboratory data, which has been relatively underutilized. This inclusion allows for a more comprehensive assessment of patient health and the risk of ACM, potentially capturing nuances in patient conditions that anatomical data alone might miss. The model displayed a great discrimination ability for 1- and 3-year survival predictions with AUCs of 0.88 (95% CI: 0.78–0.98) and 0.87 (95% CI: 0.76–0.98), respectively, in the training cohort. Such insights could assist physicians in identifying high-risk patients and taking proactive measures to prevent adverse events. Overall, utilizing predictive models in AAA patients is promising to enhance patient outcomes and reduce healthcare costs by facilitating early intervention and tailored treatment plans.

Machine learning enhances healthcare by improving diagnostics, personalizing treatments, predicting patient outcomes, and optimizing operations. It aids in analyzing complex medical data for accurate diagnoses, tailoring treatments to individual patients, and predicting risks such as disease progression or post-surgery complications.[22,23] In this article, we reported six variables as independent risk factors for ACM. Among them, the diameter of an aneurysm has been the gold standard for evaluating AAA progression and rupture prediction. Therefore, patients with large AAA should receive surgical treatment to prevent adverse outcomes.[24] Previous studies have reported that the growth rate of aneurysms in males with diameters of approximately 3 and 5 cm were 1.28 and 3.61 mm per year, respectively. Additionally, the growth rate accelerated by 0.59 mm per year for every 0.5 cm increment in aneurysm diameter. The relationship between diameter and the probability of rupture was consistent across genders, with females carrying a higher risk of rupture across the entire diameter range.[25,26] Choi et al found that an aneurysm diameter > 65 mm was a risk factor for type Ib endoleak.[27] A recent meta-analysis involving 1514 patients from 11 studies reported that the rupture rate in AAAs 5.5–6, 6.1–7, and >7 cm were 3.5%, 4.1%, and 6.3%, respectively. The study further revealed that the risk of death from other causes surpassed the risk of death from rupture, thereby underscoring the significance of investigating ACM in AAA patients.[28]

Age is another classic risk factor for AAA patients. Surgical treatment for older patients has always been a problem, even with the improvement of medical technology. Alberga et al identified age as an independent risk factor for adverse outcome, where octogenarians hold a higher risk of peri-operative mortality (1.4% for EVAR and 9.3% for open surgery).[29] A predictive model developed by a retrospective study also demonstrated age as an important risk factor (<70 for 0 points, 70–74.9 for 9 points, 75–79.9 for 10 points, and ≥80 for 17 points).[30] These analyses could assist clinicians with risk-benefit assessments when deciding on treatment strategies for elderly patients.

Stroke has been identified as a comorbidity among AAA patients. In a study involving 1136 individuals, the prevalence of a prior stroke was 12.3%,[31] which was relatively close to our present study (16.9%). However, in another cohort with 206 individuals, the prevalence could reach 38%,[32] highlighting a huge variation in the comorbidity prevalence and the need for further research focusing on the history of cerebrovascular diseases.

More interestingly, inflammatory cells, including WBC, neutrophils, and monocytes, significantly differed in patients with adverse outcomes. Among them, monocytes were considered an independent risk factor for ACM, which aligned with previous studies, thereby suggesting the association between monocytosis and cardiovascular disease.[33,34] Studies also discovered significant alterations in peripheral blood monocyte subsets and phenotypes, suggesting an active involvement of monocytes in AAA development. Consequently, its role in AAA prognosis requires further investigation.[35,36]

The findings from this study, through the development of a more comprehensive and accurate predictive model for ACM in post-EVAR patients, have the potential to transform clinical practice by enhancing patient stratification, personalizing treatment plans, supporting informed decision-making, and optimizing resource allocation.

Limitation

Firstly, this study was conducted at a single center, which may limit the generalizability of our findings to other healthcare settings or geographical regions. Patient demographics, treatment protocols, and the prevalence of comorbid conditions can vary significantly across different regions, potentially influencing the model's predictive accuracy. Secondly, the retrospective and observational design of the study inherently carries the risk of selection bias and residual confounding, despite our efforts to adjust for known confounders. The relatively small sample size and number of ACM events may also limit the robustness of the model, particularly in populations with different baseline risk profiles. Another limitation of our study is the lack of comprehensive data regarding specific endograft device types used in the procedures. Device characteristics and design variations could potentially influence long-term outcomes after EVAR, as different devices may have varying rates of durability, conformability, and fixation mechanisms. Future studies with prospective data collection protocols should aim to systematically document and analyze the impact of different device types on post-EVAR outcomes, which could further refine risk prediction models. Lastly, our model has yet to undergo external validation in independent cohorts. Without external validation, it is difficult to confirm its performance and utility across varied clinical environments. Future studies should aim to validate this nomogram in multi-center cohorts that include diverse populations from different geographical regions and with varying comorbidity profiles. Such efforts would provide a more comprehensive evaluation of the model's generalizability and ensure its broader clinical applicability.

Conclusion

In this study, we developed a nomogram to predict ACM in patients undergoing EVAR for AAA. Key independent risk factors identified include a history of stroke, CIA atherosclerosis, advanced age, low hemoglobin levels, elevated monocyte count, and the presence of a large AAA. The nomogram demonstrated good predictive accuracy and can enhance clinical decision-making by enabling personalized risk stratification and tailored treatment strategies. Future research should focus on validating the model in diverse cohorts and incorporating additional biomarkers to improve its predictive power.

Abbreviations

AAA, Abdominal aortic aneurysm; ACEI, angiotensin-converting enzyme inhibitor; ACM, all-cause mortality; AUC, area under the curve; CAD, coronary artery disease; CCB, calcium entry blockers; CIA, common iliac artery; COPD, chronic obstructive pulmonary disease; CTA, computed tomographic angiography; DCA, decision curve analysis; eGFR, estimated glomerular filtration rate; EVAR, endovascular aneurysm repair; FBG, fasting blood glucose; ILT, intraluminal thrombus; KM, Kaplan-Meier; LASSO, least absolute shrinkage and selection operator; LDL-C, low-density lipoprotein cholesterol; rAAA, ruptured AAA; ROC, receiver operating characteristic; WBC, white blood cell.

Data Sharing Statement

The data that support the findings of this study are available on request from the corresponding author, upon reasonable request.

 https://doi.org/10.2147/CIA.S493841

Ethics Approval

This study has been approved by the Ethics Committee of the First Affiliated Hospital of Zhengzhou University. The details of the study design are documented at http://www.chictr.org.cn (Identifier: ChiCTR2200063122). The data are anonymous, and the requirement for informed consent was therefore waived. Our research conformed to the Declaration of Helsinki. Personal information and data remained confidential and anonymous.

Acknowledgment

The authors wish to acknowledge the assistance of the First Affiliated Hospital of Zhengzhou University with the data extraction.

Author Contributions

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

Funding

This work was supported by the National Natural Science Foundation of China (No. 82222007 and 82170281 to J.T., U2004203 to J.Z.), Henan Province Key R&D Program (No.241111313300) to J.T., Henan Province Medical Science and Technology Key Joint Project (SBGJ202101012) to J.Z., Henan Province Science and Technology Research Joint Project (No.222301420054) to J.T., Henan Thousand Talents Program (No. ZYQR201912131) to J.Z., Funding for Scientific Research and InnovationTeam of The First Affiliated Hospital of Zhengzhou University (QNCXTD2023001 to J.T and ZYCXTD2023008 to J.Z.).

Disclosure

The authors report no conflicts of interest in this work.

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