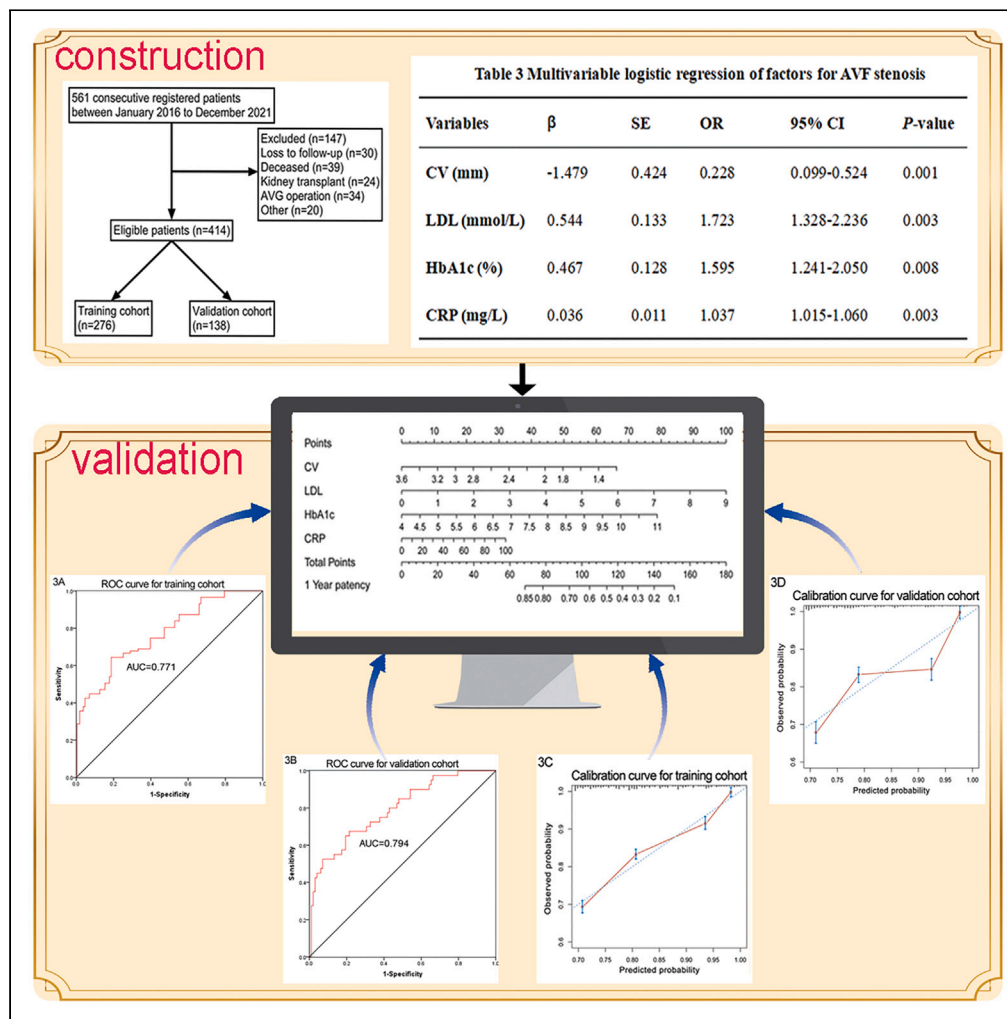**Article**

Prognostic nomogram for the patency of wrist autologous arteriovenous fistula in first year

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Highlights

Our study established a
practical model to predict
the stenosis probability of
AVF

Our study included as
much data as possible in
our institution over a 6-year
period

Nomogram model is a
relatively novel research
approach in AVF related
studies

Article

Prognostic nomogram for the patency of wrist autologous arteriovenous fistula in first year

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SUMMARY

Autologous arteriovenous fistula (AVF) is preferred in hemodialysis patients. Maintaining its patency is a critical problem. This study aimed to create a nomogram model for predicting 1-year primary patency of AVF. Consequently, a total of 414 patients were retrospectively enrolled and randomly allocated to training and validation cohorts. Risk factors were identified by multivariable logistic regression and used to create a nomogram model. Performance of the model was evaluated by receiver operating characteristic (ROC) curve, Hosmer-Lemeshow test, and calibration curve. The results suggested that diameter of cephalic vein, low-density lipoprotein, glycosylated hemoglobin (%), and C-reactive protein were risk factors which could predict the patency of AVF. Area under ROC curves for training and validation cohorts were 0.771 and 0.794, respectively. Calibration ability was satisfactory in both cohorts. Therefore, present nomogram model could predict the 1-year primary patency of AVF.

INTRODUCTION

The incidence of chronic kidney disease (CKD) is gradually increasing, and more patients require maintenance hemodialysis (MHD).[1,2] Autologous arteriovenous fistula (AVF) is the optimal vascular access for MHD.[3] However, AVF stenosis is a major challenge for these patients and vascular surgeons. It is the most important reason for AVF failure.[4,5] This also translates to higher treatment costs.[6] Although many risk factors for AVF stenosis have been reported,[7–9] predicting AVF stenosis or patency remains difficult after its first cannulation.

A nomogram is a graphical model in which certain risk factors work together to perform precise prediction, and there are several nomograms used as prediction tools for various diseases.[10–12] Concerning the topic of AVF, Tng et al. created a nomogram model to predict AVF maturation.[13] Ouyang et al. also created a model to predict the survival of patients with MHD.[14] However, no nomogram model has yet been developed for predicting (primary) AVF patency. Therefore, we constructed and validated a nomogram model based on relevant variables which may predict AVF stenosis.

Methods

Study design and population

A total of 561 patients with end-stage renal disease (ESRD) who underwent preventative autologous wrist AVF creation between radial artery (RA) and cephalic vein (CV) were retrospectively enrolled in this study. All patients were treated in the Department of General Surgery of Xuanwu Hospital between January 1, 2016, and December 31, 2021. A series of measurements was evaluated, including medical history, physical examination, and laboratory tests. Vascular ultrasonography was the most important examination. AVF creation was considered when the arterial and venous diameters were greater than 1.5 and 2.0 mm, respectively.[15]

First, nephrologists and surgeons carefully evaluated the necessity of AVF creation. Afterward, the running and diameter of RA and CV for all patients were examined using ultrasound before the operation. Next, under local anesthesia, an end-to-side wrist fistula was constructed in patients without contraindications (such as central venous stenosis).

After AVF creation, patients who met the following criteria were included in the study: (1) age ≥18 years; (2) the AVF had matured successfully and functioned normally (Qa within brachial artery >500 mL/min, diameter of puncture segment ≥5 mm, distance between skin and AVF <6 mm); (3) patients who received a preventative AVF creation and underwent MHD in our institution; (4) patients who underwent surgery by the same group of surgeons.

Patients who met the inclusion criteria were randomly divided into training and validation cohorts at a ratio of 2:1 using an online random number generator (https://c.runoob.com/front-end/6680/). To evaluate the stability of the proposed model, both training and

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validation cohorts were repeated by 10 random resampling. All statistical analyses were performed using a fixed-effects meta-analysis of 10 resampling.

After the first cannulation of the AVF, vascular ultrasound was performed every three months by the same group of vascular ultrasound doctors.[16] The main focus point was the diameter of the anastomosis and the blood flow within the brachial artery at the cubital fossa. The surgical process and management of patients followed the recommendations of the relevant guidelines.[17]

The present study involving human participants was conducted following the ethical standards of our institution and the 1964 Helsinki Declaration and its later amendments. The Independent Ethics Committee (IEC) of Xuanwu Hospital, Capital Medical University (no. [2018] 030) approved this study. All patients provided written informed consent for participation in the present study and publication of relevant data.

Data collection

We retrospectively collected demographic data (age, gender, BMI), etiology of ESRD (diabetes mellitus, primary glomerular disease, hypertension, and others), vascular risk factors (diameter of RA and CV), laboratory tests around AVF creation [hemoglobin, white blood cells, platelets, calcium, phosphorus, albumin, serum creatinine, total cholesterol, low-density lipoprotein (LDL), glycosylated hemoglobin (HbA1c), and C-reactive protein (CRP)], and statin application rate. Laboratory tests around AVF creation referred to the mean value of the latest test results before and after surgery. Meanwhile, AVF usage and the latest vascular ultrasound results at 12 months after the first cannulation were collected by telephone by the first author. The period between AVF creation and the first cannulation was also recorded.

AVF stenosis was defined as follows: the results of vascular ultrasound showed a diameter reduction of >50% and a Qa within the brachial artery of <250 mL/min, which could not meet the requirement of MHD.[15]

Statistical analysis and model evaluation

Statistical analysis was performed using SPSS (version 26; IBM Corp., Armonk, NY, USA) and RStudio software. Statistical significance was set at $p < 0.05$ (two-sided). The power test was performed byPASS software. Categorical variables were presented as numbers and percentages, and continuous variables were presented as mean $\pm$ standard deviation. Differences between the two cohorts were explored using the chi-square test for categorical variables and the independent t-test for continuous variables. Additionally, the multicollinearity of included variables was examined to determine whether multicollinearity would significantly affect the nomogram model.

To construct a nomogram, the differences between patients with and without AVF stenosis in training cohort were compared, and multivariable logistic regression analysis was performed to determine independent factors of AVF stenosis. Finally, the odds ratios (ORs) and 95% confidence intervals (CIs) for each risk factor in the logistic regression model were calculated.

The performance of the nomogram model was assessed using training and validation cohorts. First, the discrimination of the nomogram was assessed using the area under the receiver operating characteristic curve (ROC-AUC). An AUC of 1.0 indicated that the nomogram had perfect discrimination ability. Second, the calibration of the nomogram was evaluated using the Hosmer-Lemeshow goodness-of-fit test ($p > 0.05$), which suggested no significant difference between predicted and observed values. In other words, this model exhibited good calibration ability.[18] Moreover, the calibration curve was plotted to analyze the relationship between the observed and predicted probabilities in training and validation cohorts.

RESULTS

In total, 561 patients were registered between January 2016 and December 2021. Among all patients, 30, 39, 24, 34, and 20 patients were excluded due to loss of follow-up, being deceased, receiving a kidney transplant, receiving an arteriovenous graft operation, and other reasons. Ultimately, 414 patients were included in the present study, with 276 and 138 patients in the training and validation cohorts, respectively (Figure 1). There were no significant differences in patient characteristics between the two cohorts (Table 1).

Univariate analyses of patients with or without AVF stenosis in training cohort

In the training cohort, 80 patients developed AVF stenosis, whereas 196 remained patency. AVF stenosis predominantly manifested at the anastomosis site ($n = 52$, 65%), followed by the puncture point ($n = 16$, 20%) and outflow route ($n = 12$, 15%). Univariate analyses of the training cohort showed that patients with stenotic AVF exhibited elevated LDL levels (3.68 $\pm$ 1.26 versus 2.77 $\pm$ 1.15 mmol/L, $p = 0.001$), heightened HbA1c (%) levels (6.22 $\pm$ 1.22 versus 5.45 $\pm$ 1.07, $p = 0.008$), and a higher prevalence of diabetes mellitus (50% versus 36.22%, $p = 0.034$). Furthermore, they demonstrated elevated CRP levels (13.04 $\pm$ 20.58 versus 7.17 $\pm$ 11.36 mg/L, $p = 0.035$) and a reduced CV diameter (2.39 $\pm$ 0.36 versus 2.56 $\pm$ 0.34 mm, $p = 0.012$) (Table 2). Moreover, relevant studies have indicated that multicollinearity among the included variables did not significantly impact the nomogram model (Figure S1).

Subsequently, in conjunction with clinical expertise, the following variables were incorporated into the final multivariable logistic regression: LDL (OR: 1.723; 95% CI: 1.328–2.236; $p = 0.003$), HbA1c (%) (OR: 1.595; 95% CI: 1.241–2.050; $p = 0.008$), CRP level (OR: 1.037; 95% CI: 1.015–1.060; $p = 0.003$), and CV diameter (OR, 0.228; 95% CI: 0.099–0.524; $p = 0.001$) (Table 3).

Construction and validation of the nomogram model

A nomogram model was developed based on LDL, HbA1c (%), CRP, and CV diameter (Figure 2). The discriminative ability of the nomogram model was good, with an AUC of 0.771 (95% CI: 0.711–0.830) in the training cohort (Figure 3A) and 0.794 (95% CI: 0.711–0.877) in the validation
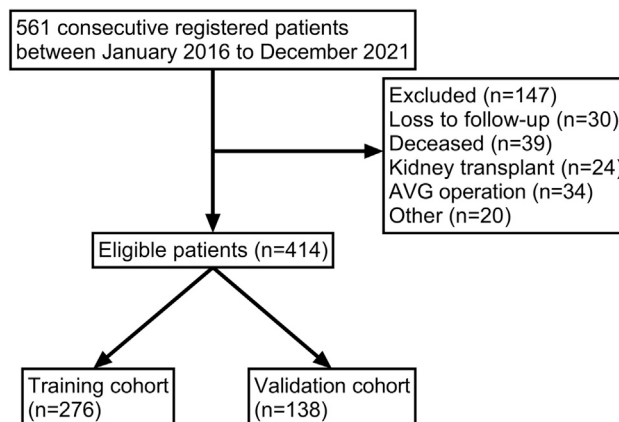
Figure 1. Flowchart of patient selection in present study

cohort (Figure 3B). Moreover, the calibration ability of the current nomogram model was considered satisfactory in both training cohort (Hosmer-Lemeshow test, p = 0.172) and the validation cohort (Hosmer-Lemeshow test, p = 0.679). The calibration curve of the nomogram model is shown in Figures 3C and 3D. The calibration curve closely approximated the standard line, indicating a good agreement between the observed and predicted probabilities in both the training and validation cohorts.

With regard to the application of present model. Figure 2 illustrates that specific values of each indicator can correspond to a particular score on the "Point" axis. Subsequently, by summing the scores of each indicator, the total score can be calculated and located on the "Total points" axis. Ultimately, the users can determine the patency probability of each AVF. Patients with a lower likelihood of AVF patency warrant increased attention during MHD.

DISCUSSION

The utility of a nomogram model for predicting AVF maturation has been previously reported,[13] and various factors have been identified. However, the mechanisms between stenosis of functional AVF and maturation failure may be different. It is widely believed that neointimal hyperplasia (NIH) may be one of the reasons of vascular stenosis, but it has recently been challenged.[19,20] Moreover, results from Lee T. et al. also indicated that AVF stenosis may result from NIH and fibrosis of the adventitia.[21] Conversely, maturation failure may be caused by preoperative vascular diameter and shear stress at the time of AVF creation.[22,23] In terms of predicting AVF patency, Ricardo Peralta et al. previously developed a machine learning model, with results that partially overlapped with those of the present study.[24] However, no nomogram model exists to predict the patency probability of AVF after initial cannulation. Thus, this study developed and validated a nomogram model based on LDL, HbA1c (%), CRP, and CV diameter, demonstrating favorable discrimination and calibration capabilities. The nomogram model can give surgeons a precise probability of AVF patency one year after initial cannulation. Based on various clinical findings, the nomogram model can be readily employed after initial cannulation. The likelihood of AVF patency was calculated by inputting the aforementioned indicators into the current model.

Diameter of cephalic vein

Traditionally, vasculature diameter has been considered a pivotal factor in AVF maturation and failure.[25] However, the diameter of the RA was not included in our nomogram model, which is consistent with the findings of several previous studies.[26,27] This observation is consistent with the distribution of AVF stenosis locations, as reported by Rajan DK et al.[28] These findings suggest that the diameter of the CV may be more significant than that of the RA in AVF stenosis, although further investigation of the underlying mechanism is warranted.

In addition to nomogram-based prediction methods, some researchers have explored alternative approaches for predicting the probability and severity of AVF stenosis. The results of study conducted by Park et al. elucidated the relationship between AVF stenosis and shunt sounds using deep learning techniques.[29,30] These findings suggest a potential correlation between AVF shunt sounds and venous outflow (CV) stenosis. These results partially align with those of current study. Therefore, after the initial cannulation of AVF, more attention should be paid to the diameter of CV.

Low-density lipoprotein

Our previous study indicated that LDL could be a risk factor for AVF stenosis.[4] Furthermore, results from a meta-analysis suggested a potential association between AVF failure, increased LDL levels, and decreased high-density lipoprotein levels.[31] Additionally, several studies have demonstrated that LDL may play a crucial role as a triggering agent for endothelial dysfunction.[32,33] Moreover, several studies have also demonstrated that LDL could increase the proliferation and migration of human umbilical vein endothelial cells (HUVECs),[34,35] which could

Table 1. Patients included in training and validation cohorts

Variable	Training (n = 276)	Validation (n = 138)	p value
Demographics			
Age (years), mean (SD)	56.86 ± 11.74	57.10 ± 11.75	0.865
BMI, mean (SD)	24.80 ± 3.60	24.96 ± 3.80	0.716
Female, n (%)	108(39.13%)	60(43.48%)	0.398
Etiology			
Diabetes mellitus, n (%)	111(40.22%)	50(36.23%)	0.433
Primary glomerular disease, n (%)	97(35.14%)	47(34.06%)	0.101
Hypertension, n (%)	50(18.12%)	26(18.84%)	0.858
other, n (%)	18(6.52%)	15(10.87%)	0.124
Vascular Diameter			
CV (mm), mean (SD)	2.51 ± 0.37	2.50 ± 0.37	0.964
RA (mm), mean (SD)	2.68 ± 0.54	2.72 ± 0.56	0.585
Laboratory test, mean (SD)			
HB (g/L)	88.01 ± 21.66	87.43 ± 20.04	0.823
WBC (10^9/L)	6.76 ± 2.14	6.55 ± 2.09	0.415
PLT (10^9/L)	190.23 ± 72.11	186.27 ± 63.74	0.641
ALB (g/L)	33.35 ± 6.09	33.49 ± 5.72	0.851
Scr (μmol/L)	692.36 ± 292.71	673.23 ± 280.78	0.588
Serum calcium (mmol/L)	1.98 ± 0.31	1.99 ± 0.30	0.745
Serum phosphorus (mmol/L)	1.80 ± 0.54	1.79 ± 0.53	0.882
TC (mmol/L)	4.48 ± 1.34	4.41 ± 1.24	0.664
LDL (mmol/L)	2.90 ± 1.16	2.83 ± 1.05	0.613
HbA1c (%)	5.66 ± 1.12	5.63 ± 1.04	0.775
CRP (mg/L)	8.51 ± 14.43	8.08 ± 13.05	0.803
Statins, n (%)	192 (69.57%)	99 (71.74%)	0.732
Period between AVF creation and its first cannulation (Day)	87.9 ± 23.28	89.2 ± 18.98	0.747
AVF status of 12 months*	196 (70.01%)	101 (73.19%)	0.663

BMI, body mass index; CV, cephalic vein; RA, radial artery; HB, hemoglobin; WBC, white blood cell; PLT, platelet; ALB, albumin; Scr, Serum creatinine; TC, total cholesterol; LDL, low density lipoprotein; HbA1c, glycated hemoglobin A1c; CRP, C-reactive protein. AVF, autologous arteriovenous fistula. *: Primary patency rates of AVFs at 12 months after its first cannulation.

contribute to venous stenosis. A previous study has also shown that over 60% of AVF stenosis occurs on the venous side,[28] which may explain why LDL levels could predict the patency rate of AVF.

HbA1c (%)

Diabetes mellitus is one of the major causes of morbidity and mortality among old patients and remains the foremost cause of ESRD globally.[36,37] As an indicator of long-term glucose control and a predictive marker for risk, HbA1c constitutes an essential component of routine diabetes mellitus management.[38] Given the association between elevated HbA1c and AVF failure in peripheral arterial disease and endothelial dysfunction, increased HbA1c could potentially correlate with AVF failure. The findings of the present study indicate that HbA1c level could serve as an indicator of AVF stenosis. However, due to the inherent characteristics of the present study, the underlying mechanisms have not yet been thoroughly investigated. Nevertheless, the relationship between elevated HbA1c and vascular disease has been well demonstrated. In patients with diabetes, reductions in HbA1c levels have been shown to correspond to reductions in microvascular complications.[39,40] Moreover, high HbA1c level have been associated with NIH.[41] Furthermore, other studies have demonstrated that diabetes mellitus was linked to fibrotic remodeling of veins, which could also be the underlying mechanism of AVF stenosis.[42]

C-reactive protein (CRP)

CRP has emerged as a notable risk factor for AVF failure in patients undergoing MHD, as demonstrated in numerous studies.[43,44] The current study similarly identified CRP level as a predictor of AVF stenosis. Additionally, a study conducted by Feng Zhu et al. suggested that the

Table 2. Patients with and without AVF stenosis in training cohort

Variables	Patency (n = 196)	Stenosis (n = 80)	p-value
Demographics			
Age (years), mean (SD)	55.91 ± 10.92	58.08 ± 11.69	0.144
BMI, mean (SD)	24.69 ± 3.63	f24.65 ± 3.30	0.921
Female, n (%)	56(28.57%)	24(29.58%)	0.893
Etiology			
Diabetes mellitus, n (%)	71(36.22%)	40(50%)	0.034*
Primary glomerular disease, n (%)	71(36.22%)	26(32.5%)	0.557
Hypertension, n (%)	31(15.82%)	19(23.75%)	0.121
other, n (%)	11(5.61%)	7(8.75%)	0.311
Vascular Diameter			
CV (mm), mean (SD)	2.56 ± 0.34	2.39 ± 0.36	0.012*
RA (mm), mean (SD)	2.67 ± 0.46	2.72 ± 0.72	0.576
Laboratory test, mean (SD)			
HB (g/L)	87.89 ± 23.22	89.24 ± 18.86	0.646
WBC (10^9/L)	6.95 ± 2.16	6.56 ± 2.19	0.175
PLT (10^9/L)	190.21 ± 76.68	194.39 ± 65.65	0.67
ALB (g/L)	33.57 ± 6.16	33.09 ± 5f.98	0.477
Scr (μmol/L)	694.39 ± 291.18	670.62 ± 280.18	0.535
Serum calcium (mmol/L)	1.98 ± 0.31	2.01 ± 0.28	0.477
Serum phosphorus (mmol/L)	1.77 ± 0.52	1.80 ± 0.49	0.706
TC (mmol/L)	4.51 ± 1.32	4.80 ± 1.66	0.126
LDL (mmol/L)	2.77 ± 1.15	3.68 ± 1.26	0.001*
HbA1c (%)	5.45 ± 1.07	6.22 ± 1.22	0.008*
CRP (mg/L)	7.17 ± 11.36	13.04 ± 20.58	0.035*
Statins, n (%)	142 (72.37%)	55 (68.42%)	0.559
Period between AVF creation and first cannulation (Day)	89.7 ± 25.35	87.5 ± 20.06	0.566

BMI, body mass index; CV, cephalic vein; RA, radial artery; HB, hemoglobin; WBC, white blood cell; PLT, platelet; ALB, albumin; Scr, serum creatinine; TC, total cholesterol; LDL, low density lipoprotein; HbA1c, glycated hemoglobin A1c; CRP, C-reactive protein. AVF, autologous arteriovenous fistula. *, $p < 0.05$.

platelet-to-lymphocyte ratio (PLR), an indicator of systemic inflammation, may serve as a risk factor for developing early AVF restenosis.[45] However, the beta coefficient of CRP in present study was relatively minimal compared to that of other factors, which could be attributed to the wide range of CRP levels observed. However, regular monitoring of CRP levels is crucial. Meanwhile, further analysis is warranted to elucidate the underlying mechanism by which CRP level predicts AVF stenosis.

Limitations of the study

The present study has several limitations. First, it was a retrospective study predominantly involving wrist AVF, potentially introducing selection and detection biases. Second, for individuals applying this model, inherent characteristics such as confirming the score for each variable

Table 3. Multivariable logistic regression of factors for AVF stenosis

Variables	β	SE	OR	95% CI	p-value
CV (mm)	−1.479	0.424	0.228	0.099–0.524	0.001
LDL (mmol/L)	0.544	0.133	1.723	1.328–2.236	0.003
HbA1c (%)	0.467	0.128	1.595	1.241–2.050	0.008
CRP (mg/L)	0.036	0.011	1.037	1.015–1.060	0.003

CV, cephalic vein; LDL, low density lipoprotein; HbA1c, glycated hemoglobin A1c; CRP, C-reactive protein; SE, standard error; OR, odds ratio; CI, confidence interval.
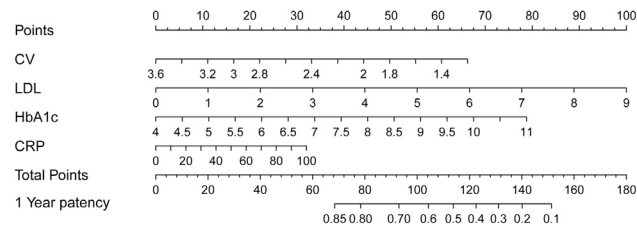
Figure 2. Nomogram model for the prediction of AVF patency

To estimate the probability of AVF patency. We could mark each value at corresponding axis, draw a straight line perpendicular to the "Point" axis and find corresponding score of each item. Then, scores of all items were added together. Next, we mark the sum on "Total Points" axis and draw a straight line perpendicular to "1 Year Patency" axis. Finally, the corresponding value is the patency probability of each AVF. CV, cephalic vein; LDL, low-density lipoprotein; HbA1c, glycated hemoglobin A1c; CRP, C-reactive protein.

may lead to a small range of errors in the predicted patency probability. Third, present study is a pilot study; prospective study with a larger sample size may be required in the future.

Conclusion

Present study developed a nomogram model for predicting the one-year patency of AVF after its initial cannulation. The nomogram incorporated variables, including CV diameter, LDL, HbA1c (%), and CRP. This model can serve as a valuable tool for informing patients about their prognosis and can help optimize preoperative decision-making.

RESOURCE AVAILABILITY

Lead contact

Further information and requests for resources and reagents should be directed to and will be fulfilled by the lead contact, Tao Luo (taoluo35@126.com).

Materials availability

Methods in this study will be made available by the lead contact upon request.

Data and code availability

- All the data are available from the corresponding authors upon reasonable request.
- This paper does not report original code.
- Any additional information related to data analysis in this paper is available from the lead contact upon reasonable request.

AUTHOR CONTRIBUTIONS

Data curation: Y.C., W.C., J.W., C.Z., and L.Z.; Funding acquisition: T.L.; Supervision: C.B.; Validation: C.B. and T.L.; Writing – review and editing: Y.L. and J.Y.

DECLARATION OF INTERESTS

The authors declare no competing interests.

STAR★METHODS

Detailed methods are provided in the online version of this paper and include the following:

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- EXPERIMENTAL MODEL AND STUDY PARTICIPANT DETAILS
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  - Ethical approval
- METHOD DETAILS
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- QUANTIFICATION AND STATISTICAL ANALYSIS

SUPPLEMENTAL INFORMATION

Supplemental information can be found online at https://doi.org/10.1016/j.isci.2024.110727.
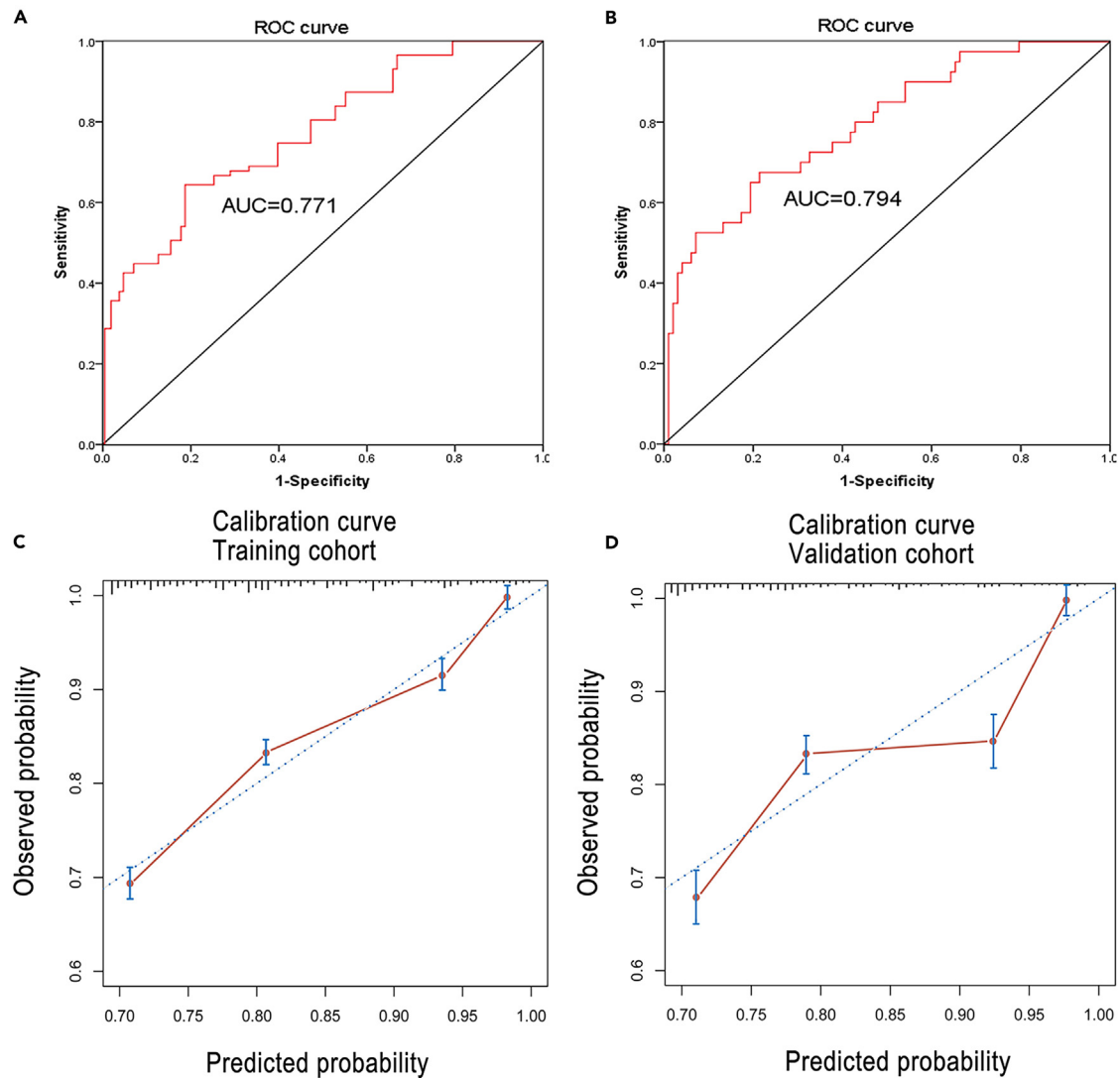
Figure 3. Receiver operating characteristics (ROCs) curves and calibration curves of the nomogram model
(A and B) ROC curve of the training cohort (A) and validation cohort (B); (C and D) Calibration curve of training cohort (C) and validation cohort (D). AUC, Area Under Curve.

REFERENCES

1. Zhang, L., Wang, F., Wang, L., Wang, W., Liu, B., Liu, J., Chen, M., He, Q., Liao, Y., Yu, X., et al. (2012). Prevalence of chronic kidney disease in China: a cross-sectional survey. Lancet *379*, 815–822.

2. Coresh, J., Astor, B.C., Greene, T., Eknoyan, G., and Levey, A.S. (2003). Prevalence of chronic kidney disease and decreased kidney function in the adult US population: Third National Health and Nutrition Examination Survey. Am. J. Kidney Dis. *41*, 1–12.

3. Schmidli, J., Widmer, M.K., Basile, C., de Donato, G., Gallieni, M., Gibbons, C.P., Haage, P., Hamilton, G., Hedin, U., Kamper, L., et al. (2018). Editor's Choice - Vascular Access: 2018 Clinical Practice Guidelines of the European Society for Vascular Surgery (ESVS). Eur. J. Vasc. Endovasc. Surg. *55*, 757–818.

4. Campos, R.P., Do Nascimento, M.M., Chula, D.C., Do Nascimento, D.E., and Riella, M.C. (2006). Stenosis in hemodialysis arteriovenous fistula: evaluation and treatment. Hemodial. Int. *10*, 152–161.

5. He, X., Feng, Z.P., Liu, Z.Y., Jiang, X.Y., Li, B., and Peng, H.Y. (2021). Investigation on the mechanism of p38MAPK in the failure of autogenous arteriovenous fistula caused by stenosis. J. Physiol. Pharmacol. *72*.

6. Dolmatch, B., Hogan, A., and Ferko, N. (2018). An Economic Analysis of Stent Grafts for Treatment of Vascular Access Stenosis: Point-of-Care and Medicare Perspectives in the United States. J. Vasc. Interv. Radiol. *29*, 765–773.e2.

7. Li, Y., Cui, W., Wang, J., Zhang, C., and Luo, T. (2021). Factors associated with dysfunction of autogenous arteriovenous fistula in patients with maintenance hemodialysis: a retrospective study. Ann. Palliat. Med. *10*, 4047–4054.

8. Moon, J.Y., Lee, H.M., Lee, S.H., Lee, T.W., Ihm, C.G., Jo, Y.I., Han, S.W., and Shin, S.G. (2015). Hyperphosphatemia is associated with patency loss of arteriovenous fistula after 1 year of hemodialysis. Kidney Res. Clin. Pract. *34*, 41–46.

9. Smith, G.E., Gohil, R., and Chetter, I.C. (2012). Factors affecting the patency of arteriovenous fistulas for dialysis access. J. Vasc. Surg. *55*, 849–855.

10. Hoshino, N., Hida, K., Sakai, Y., Osada, S., Idani, H., Sato, T., Takii, Y., Bando, H., Shiomi, A., and Saito, N. (2018). Nomogram for predicting anastomotic leakage after low anterior resection for rectal cancer. Int. J. Colorectal Dis. *33*, 411–418.

11. Ge, J.C., Tao, M., Li, L., Ma, Z.L., Jiang, B., Yuan, C.H., Wang, H.Y., Peng, Y., and Xiu, D.R. (2021). Nomogram and competing risk model to predict recurrence after curative surgical resection of PDAC. Pancreatology *21*, 1102.

12. Xu, L., Li, L., Wang, P., Zhang, M., Zhang, Y., Hao, X., Yan, L., Li, B., Wen, T., and Xu, M. (2019). Novel Prognostic Nomograms for Hepatocellular Carcinoma Patients with Microvascular Invasion: Experience from a Single Center. Gut Liver *13*, 669–682.

13. Tng, A.R.K., Lee, K.G., Tan, R.Y., Pang, S.C., Foo, M.W.Y., Choong, H.L., Li, H.H., Goh, N., Shaam, A., Tan, Y.L., et al. (2020). Validation of the failure to maturation equation and proposal for a novel scoring system for arteriovenous fistula maturation in multiethnic Asian haemodialysis patients. J. Vasc. Access *21*, 169–175.

14. Ouyang, H., Shi, Q., Zhu, J., Shen, H., Jiang, S., and Song, K. (2021). Nomogram for predicting 1-5-and 10-year survival in hemodialysis (HD) patients: a single center retrospective study. Ren. Fail. *43*, 1508–1519.

15. Jin, Q., Wang, Y., Ye, C., and Shi, Y. (2019). Expert consensus on vascular access for hemodialysis in China (2nd Edition). Chin J Blood Purif *18*, 365–381.

16. Guedes Marques, M., Ibeas, J., Botelho, C., Maia, P., and Ponce, P. (2015). Doppler ultrasound: a powerful tool for vascular access surveillance. Semin. Dial. *28*, 206–210.

17. Lok, C.E., Huber, T.S., Lee, T., Shenoy, S., Yevzlin, A.S., Abreo, K., Allon, M., Asif, A., Astor, B.C., Glickman, M.H., et al. (2020). KDOQI Clinical Practice Guideline for Vascular Access: 2019 Update. Am. J. Kidney Dis. *75*, S1–S164.

18. Kramer, A.A., and Zimmerman, J.E. (2007). Assessing the calibration of mortality benchmarks in critical care: The Hosmer-Lemeshow test revisited. Crit. Care Med. *35*, 2052–2056.

19. Ma, S., Duan, S., Liu, Y., and Wang, H. (2022). Intimal Hyperplasia of Arteriovenous Fistula. Ann. Vasc. Surg. *85*, 444–453.

20. Vazquez-Padron, R.I., Duque, J.C., Tabbara, M., Salman, L.H., and Martinez, L. (2021). Intimal Hyperplasia and Arteriovenous Fistula Failure: Looking Beyond Size Differences. Kidney360 *2*, 1360–1372.

21. Lee, T., Somarathna, M., Hura, A., Wang, Y., Campos, B., Arend, L., Munda, R., and Roy-Chaudhury, P. (2014). Natural history of venous morphologic changes in dialysis access stenosis. J. Vasc. Access *15*, 298–305.

22. Kaller, R., Arbănași, E.M., Mureșan, A.V., Voidăzan, S., Arbănași, E.M., Horváth, E., Suciu, B.A., Hosu, I., Halmaciu, I., Brinzaniuc, K., and Russu, E. (2022). The Predictive Value of Systemic Inflammatory Markers, the Prognostic Nutritional Index, and Measured Vessels' Diameters in Arteriovenous Fistula Maturation Failure. Life *12*, 1447.

23. Colley, E., Carroll, J., Anne, S., Shannon, T., Ramon, V., and Tracie, B. (2022). A longitudinal study of the arterio-venous fistula maturation of a single patient over 15 weeks. Biomech. Model. Mechanobiol. *21*, 1217–1232.

24. Peralta, R., Garbelli, M., Bellocchio, F., Ponce, P., Stuard, S., Lodigiani, M., Fazendeiro Matos, J., Ribeiro, R., Nikam, M., Botler, M., et al. (2021). Development and Validation of a Machine Learning Model Predicting Arteriovenous Fistula Failure in a Large Network of Dialysis Clinics. Int. J. Environ. Res. Public Health *18*, 12355.

25. Jemcov, T.K. (2013). Morphologic and functional vessels characteristics assessed by ultrasonography for prediction of radiocephalic fistula maturation. J. Vasc. Access *14*, 356–363.

26. Bojakowski, K., Gorczyca-Wiśniewska, E., Szatkowski, M., Walecki, J., and Andziak, P. (2010). Preoperative ultrasonographic examination of the radial artery and the cephalic vein and risks of dialysis arterio-venous fistula dysfunction. Pol. J. Radiol. *75*, 7–12.

27. Jeon, E.Y., Cho, Y.K., Cho, S.B., Yoon, D.Y., and Suh, S.O. (2016). Predicting Factors for Successful Maturation of Autogenous Haemodialysis Fistulas After Salvage Percutaneous Transluminal Angioplasty in Diabetic Nephropathy: A Study on Follow-Up Doppler Ultrasonography. Iran. J. Radiol. *13*, e32559.

28. Rajan, D.K., Bunston, S., Misra, S., Pinto, R., and Lok, C.E. (2004). Dysfunctional autogenous hemodialysis fistulas: outcomes after angioplasty–are there clinical predictors of patency? Radiology *232*, 508–515.

29. Park, J.H., Yoon, J., Park, I., Sim, Y., Kim, S.J., Won, J.Y., and Han, K. (2023). A deep learning algorithm to quantify AVF stenosis and predict 6-month primary patency: a pilot study. Clin. Kidney J. *16*, 560–570.

30. Park, J.H., Park, I., Han, K., Yoon, J., Sim, Y., Kim, S.J., Won, J.Y., Lee, S., Kwon, J.H., Moon, S., et al. (2022). Feasibility of Deep Learning-Based Analysis of Auscultation for Screening Significant Stenosis of Native Arteriovenous Fistula for Hemodialysis Requiring Angioplasty. Korean J. Radiol. *23*, 949–958.

31. Wei, S., Liu, N., Fu, Y., and Sun, M. (2023). Novel insights into modifiable risk factors for arteriovenous fistula failure and the importance of CKD lipid profile: A meta-analysis. J. Vas. Acc. *1*, 11297298221115557.

32. Dias, M.T.S., Aguilar, E.C., Campos, G.P., do Couto, N.F., Capettini, L.D.S.A., Braga, W.F., Andrade, L.d.O., and Alvarez-Leite, J. (2023). Butyrate inhibits LPC-induced endothelial dysfunction by regulating nNOS-produced NO and ROS production. Nitric Oxide *138–139*, 42–50.

33. Chiu, T.H., Ku, C.W., Ho, T.J., Tsai, K.L., Yang, Y.D., Ou, H.C., and Chen, H.I. (2023). Schisanhenol ameliorates oxLDL-caused endothelial dysfunction by inhibiting LOX-1 signaling. Environ. Toxicol. *38*, 1589–1596.

34. Luo, L., Liang, H., and Liu, L. (2022). Myristicin regulates proliferation and apoptosis in oxidized low-density lipoprotein-stimulated human vascular smooth muscle cells and human umbilical vein endothelial cells by regulating the PI3K/Akt/NF-κB signalling pathway. Pharm. Biol. *60*, 56–64.

35. Takahashi, A., Taniguchi, T., Fujioka, Y., Ishikawa, Y., and Yokoyama, M. (1996). Effects of lipoprotein(a) and low density lipoprotein on growth of mitogen-stimulated human umbilical vein endothelial cells. Atherosclerosis *120*, 93–99.

36. Lin, Y.C., Chang, Y.H., Yang, S.Y., Wu, K.D., and Chu, T.S. (2018). Update of pathophysiology and management of diabetic kidney disease. J. Formosan Med. Assoc. *117*, 662–675.

37. Thipsawat, S. (2021). Early detection of diabetic nephropathy in patient with type 2 diabetes mellitus: A review of the literature. Diab. Vasc. Dis. Res. *18*, 14791641211058856.

38. Weykamp, C. (2013). HbA1c: a review of analytical and clinical aspects. Ann. Lab. Med. *33*, 393–400.

39. (1998). Intensive blood-glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). UK Prospective Diabetes Study (UKPDS) Group. Lancet *352*, 837–853.

40. Nathan, D.M., Cleary, P.A., Backlund, J.Y.C., Genuth, S.M., Lachin, J.M., Orchard, T.J., Raskin, P., and Zinman, B.; Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications DCCT/EDIC Study Research Group (2005). Intensive diabetes treatment and cardiovascular disease in patients with type 1 diabetes. N. Engl. J. Med. *353*, 2643–2653.

41. Willfort-Ehringer, A., Ahmadi, R., Gessl, A., Gschwandtner, M.E., Haumer, A., Lang, W., Minar, E., Zehetmayer, S., and Ehringer, H. (2004). Neointimal proliferation within carotid stents is more pronounced in diabetic patients with initial poor glycaemic state. Diabetologia *47*, 400–406.

42. Labissiere, X., Zigmond, Z.M., Challa, A., Montoya, C., Manzur-Pineda, K., Abraham, A., Tabbara, M., Salama, A., Pan, Y., Salman, L.H., et al. (2022). Vein morphometry in end-stage kidney disease: Teasing out the contribution of age, comorbidities, and vintage to chronic wall remodeling. Front. Cardiovasc. Med. *9*, 1005030.

43. Stirbu, O., Gadalean, F., Pitea, I.V., Ciobanu, G., Schiller, A., Grosu, I., Nes, A., Bratescu, R., Olariu, N., Timar, B., and Tandrau, M.C. (2019). C-reactive protein as a prognostic risk factor for loss of arteriovenous fistula patency in hemodialyzed patients. J. Vasc. Surg. *70*, 208–215.

44. Kaygin, M.A., Halici, U., Aydin, A., Dag, O., Binici, D.N., Limandal, H.K., Arslan, Ü., Kiymaz, A., Kahraman, N., Calik, E.S., et al. (2013). The relationship between arteriovenous fistula success and inflammation. Ren. Fail. *35*, 1085–1088.

45. Zhu, F., Yao, Y., Ci, H., and Shawuti, A. (2022). Predictive value of neutrophil-to-lymphocyte ratio and platelet-to-lymphocyte ratio for primary patency of percutaneous transluminal angioplasty in hemodialysis arteriovenous fistula stenosis. Vascular *30*, 920–927.

STAR★METHODS

KEY RESOURCES TABLE

REAGENT or RESOURCE	SOURCE	IDENTIFIER
Software and algorithms		
SPSS software (version 26.0)	International Business Machines	https://www.ibm.com/cn-zh/spss
RStudio software (version 1.1.383)	R Core Team	https://posit.co/download/rstudio-desktop/
PASS software (version 11.0)	N/A	N/A
GraphPad Prism (version 8.0.2)	GraphPad software Inc	www.graphpad.com

EXPERIMENTAL MODEL AND STUDY PARTICIPANT DETAILS

Human work

The human work was performed by collecting the data of patients with AVF creation (n=414) in Xuanwu hospital, Beijing, China. All patients were of East Asian ethnicity. Among all patients, there were 168 females (40.58%) and 246 males (59.42%). Average age was 56.92 ± 11.74 years. All patients were diagnosed as chronic kidney disease which required AVF creation. According to the status of AVF, these patients were split into patency and stenosis groups.

Ethical approval

The present study involving human participants was conducted following the ethical standards of Xuanwu hospital and the 1964 Helsinki Declaration and its later amendments. The Independent Ethics Committee (IEC) of Xuanwu Hospital, Capital Medical University (no. [2018]030) approved this study. All patients provided written informed consent for participation in the present study and publication of relevant data.

METHOD DETAILS

Model construction

The patients were randomly divided into training and validation cohorts at a ratio of 2:1 using an online random number generator (https://c.runoob.com/front-end/6680/). We used the rms R package to create the model.

Model evaluation

Discrimination of the model was assessed using the area under the receiver operating characteristic curve (ROC-AUC). Calibration of the model was evaluated using the Hosmer-Lemeshow goodness-of-fit test. The relationships between observed and predicted probabilities in training and validation cohorts were analyzed by calibration curve.

QUANTIFICATION AND STATISTICAL ANALYSIS

Statistical analysis was performed using SPSS (version 26; IBM Corp., Armonk, NY, USA) and RStudio software. Statistical significance was set at *: $P<0.05$ (two-sided). The power test was performed by PASS software. Differences between the two cohorts were explored using the chi-square test for categorical variables and the independent t-test for continuous variables. Additionally, the multicollinearity of included variables was examined to determine whether multicollinearity would significantly affect the nomogram model.