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Original Article

Development of a predictive nomogram for perioperative lower extremity deep vein thrombosis in patients with breast cancer and the efficacy of dextran 40 prophylaxis

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Abstract: Deep vein thrombosis (DVT) is a significant postoperative complication in patients with breast cancer; however, validated risk-prediction models specific to this population remain scarce. This study aimed to develop a nomogram model to predict postoperative DVT and evaluate the preventive efficacy of dextran 40. Clinical data from 594 patients undergoing breast cancer surgery without prophylactic anticoagulation (May-November 2024) were retrospectively analyzed as a derivation cohort. Univariate and multivariate logistic regression analyses were used to identify independent risk factors, and a nomogram prediction model was constructed. Model performance was assessed using receiver operating characteristic curves, decision curve analysis, and calibration curves. A clinical validation cohort was prospectively collected (April-August 2025), where patients were stratified into high- and low-risk groups based on nomogram-derived scores. DVT rates between the two groups were compared to evaluate the model's discriminative ability. The prophylactic effect of dextran 40 was also analyzed. Multivariate regression analysis identified elevated D-dimer levels, age >50 years, body mass index (BMI) ≥28 kg/m^2, hypertension, and diabetes as independent risk factors for perioperative lower extremity DVT. The nomogram model demonstrated robust predictive performance in both the training (area under the receiver operating characteristic curve [AUC] = 0.797) and internal validation (AUC = 0.796) cohorts. In the clinical validation cohort, 228 patients (52.0%) were classified as high-risk, exhibiting a significantly higher DVT incidence than the low-risk group (17.5% vs. 1.9%, P<0.001). Within the high-risk subgroup, patients treated with dextran 40 had a significantly lower DVT incidence (11.4%, 15/132) than the control group (26.0%, 25/96). In summary, elevated D-dimer levels, age >50 years, BMI ≥28 kg/m^2, hypertension, and diabetes are risk factors for perioperative DVT in patients with breast cancer. Dextran 40 use was independently linked to a lower DVT rate (OR = 0.40, P = 0.011). Given the observational nature of this study, this finding is exploratory and awaits confirmation in a randomized controlled trial.

Keywords: Breast cancer, perioperative deep vein thrombosis (DVT), nomogram model, dextran 40

Introduction

According to the 2022 Global Cancer Statistics Report, breast cancer remains a significant global health burden with an incidence rate of 11.5%, ranking as the second most common cancer worldwide and the most prevalent malignancy among women [1]. Standard therapeutic interventions include surgery, chemotherapy, radiotherapy, and endocrine therapy. Throughout the treatment course, patients frequently experience severe symptom-related distress and a diminished quality of life [2]. While surgical intervention remains a primary treatment modality [3], these patients often present in a hypercoagulable state. Postoperative immobility further exacerbates this risk by inducing venous stasis, collectively increasing the incidence of deep vein thrombosis (DVT) [4]. Although the postoperative DVT risk in breast cancer is lower than that observed in orthopedic or major pelvic surgeries [5], the clinical impact remains substantial. DVT can lead to prolonged hospitalization, increased healthcare costs, and life-threatening pulmonary embolism [6]. Therefore, accu-

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rate risk identification and the implementation of effective preventive measures are essential for this population.

The reported incidence of perioperative lower extremity DVT in patients with breast cancer ranges from 2.2% to 19.96% [7-9], highlighting the urgent need for robust risk prediction. While established tools such as the Autar, Caprini, and Padua scales are available for assessment, they vary significantly in focus and scoring criteria [10-12]. These generalized models often fail to account for malignancy-specific risk factors, necessitating the development of personalized prediction tools tailored to the breast cancer population.

The nomogram serves as an intuitive statistical tool that integrates multivariate logistic regression (MLR) analysis into a graphical interface. By quantifying the contribution of individual risk factors, it calculates the specific probability of disease occurrence for a given patient [13]. While nomograms have demonstrated robust predictive capability for survival outcomes in various malignancies [14], specialized models for predicting postoperative DVT in Chinese patients with breast cancer, or within breast malignancy populations more broadly, are lacking. Furthermore, the clinical utility of a predictive model extends beyond risk identification; it should ideally guide intervention strategies. Research combining DVT risk prediction with specific pharmacological prophylaxis, such as dextran 40, remains sparse in real-world clinical decision-making scenarios.

Current clinical thromboprophylaxis typically encompasses mechanical methods, such as intermittent pneumatic compression devices, and pharmacological prophylaxis, including low molecular weight heparin [15, 16]. However, the use of conventional anticoagulants is often limited by elevated bleeding risks or specific contraindications [17, 18]. Dextran 40, a blood volume expander, has been proposed as a potential thromboprophylactic agent that enhances microcirculation, reduces blood viscosity, and inhibits platelet aggregation [19]. Nevertheless, its efficacy remains inconsistent across studies, and there are a lack of individualized, risk-stratified assessments regarding its use in patients undergoing surgery for malignant breast tumors.

This study aimed to identify independent risk factors for perioperative DVT through a retrospective analysis of clinical data from patients with breast cancer. We constructed and internally validated a nomogram to predict DVT risk and subsequently verified its stratification capability in a clinical validation cohort. Based on this model, we analyzed the efficacy of dextran 40 across different risk strata to provide evidence-based support for individualized and precise thrombosis prevention.

Materials and methods

Study population

In this retrospective study, clinical data were collected from 594 patients with breast cancer who underwent surgery without prophylactic anticoagulation at the Breast Center of the Fourth Hospital of Hebei Medical University between May and November 2024, forming the derivation cohort.

Between April and August 2025, 438 patients with breast cancer undergoing surgery were enrolled in a prospective, observational, non-randomized clinical validation cohort. Within this cohort, clinicians administered prophylactic dextran 40 (500 mL IV drip qd, from the day of surgery to 2 days postoperatively) to selected patients based on clinical judgment; the infusion rate was maintained at approximately 50 drops per minute, following an initial 5-minute rate of 30 drops per minute. This group was defined as the intervention group. The remaining patients received no prophylactic anticoagulant therapy and were defined as the control group. For patients with renal insufficiency, the attending physician determined the use and dosage of dextran 40 based on individual clinical status. This study did not intervene in clinical decision-making, solely observing and documenting outcomes.

The inclusion criteria were as follows: (1) Female with breast cancer undergoing surgical treatment at this center; (2) Preoperative bilateral lower extremity venous ultrasound confirming the absence of DVT; and (3) Complete clinical data. The exclusion criteria were as follows: (1) Concurrent other malignancies; (2) Concurrent primary hematologic disorders; (3) Preoperative ultrasound indicating lower extremity DVT; and (4) Inability to confirm the

presence of postoperative lower extremity venous thrombosis during hospitalization.

This study adhered to the Declaration of Helsinki and was approved by the Ethics Committee of the Fourth Hospital of Hebei Medical University. All subjects provided informed consent forms.

Data collection

Clinicopathological data collected included laboratory parameters (D-dimer, prothrombin time [PT], activated partial thromboplastin time [APTT], fibrinogen, white blood cell count, and platelet [PLT] count); patient characteristics (age, body mass index [BMI], and comorbidities such as diabetes and hypertension); oncological factors (TNM stage, pathological classification, and vascular or perineural invasion); and treatment-related variables (surgery type, anti-tumor therapy, and central venous catheter [CVC] placement). Lower extremity deep vein ultrasound was used to diagnose DVT, including isolated distal DVT.

Development and validation of the nomogram

Participants were randomly assigned to training and validation cohorts in a 7:3 ratio. Within the training cohort, univariate logistic regression (ULR) was performed on all collected variables. Variables with $P<0.05$ in the ULR were included in a MLR analysis to identify independent risk factors for perioperative lower extremity DVT in patients with breast cancer.

Because D-dimer values were positively skewed, a natural logarithm transformation, ln(D-dimer), was applied for all regression analyses. D-dimer levels were measured via immunoturbidimetry using an automated coagulation analyzer with a manufacturer-specified reference range of <0.243 mg/L. The laboratory maintained strict adherence to internal and external quality control protocols in accordance with established clinical standards.

Based on the MLR results, the regression coefficients of each independent predictor were converted into integer scores to construct a visualizable nomogram. The area under the receiver operating characteristic (ROC) curve (AUC) was calculated for the model in both the training and internal validation cohorts. Model consistency between predicted probabilities and actual observed incidence rates was assessed using calibration curves and Brier scores. A Hosmer-Lemeshow goodness-of-fit test was performed, where $P>0.05$ indicated adequate calibration. Finally, decision curve analysis (DCA) quantified the clinical net benefit of the model across different threshold probabilities.

Establishment and validation of the clinical application cohort

By inputting data from 438 patients in the clinical validation cohort into the nomogram, an overall risk score was calculated for each participant. Based on the optimal cutoff value from the training cohort ROC curve, all patients were categorized into high- or low-risk groups.

The DVT incidence in the high-risk group was compared with that of the low-risk group to validate model stratification. Additionally, DVT incidence in patients receiving dextran 40 was compared with that of patients not receiving dextran 40 within these two groups to verify the preventive effect.

We ran a multivariable logistic regression to see whether Dextran 40 was independently associated with postoperative DVT. First, we did univariable analyses for all clinical variables we had collected. Any variable with a *P* value below 0.05 in the univariable step was then put into a multivariable model using forward stepwise selection.

A few points are worth keeping in mind. This was a prospective observational study, meaning Dextran 40 was given at the attending clinician's discretion, not randomly assigned. So any association we found reflects a statistical correlation rather than a cause and effect relationship. Throughout this paper, when we talk about "associated factors" or "correlations", we are referring to adjusted statistical associations only. We are not claiming causation.

Statistical analysis

Data analysis was performed using SPSS (version 27.0) and R (version 4.3.3). Continuous variables were assessed using Q-Q plots. Normally distributed variables were presented as mean ± standard deviation and compared

Table 1. Baseline characteristics of the training and validation cohorts

Characteristic	Cohort		P
	Training Cohort n = 416	Validation Cohort n = 178	
D-dimer (mg/L), Mean ± SD	0.14 ± 0.12	0.12 ± 0.09	0.126
PT (s), Mean ± SD	11.00 ± 0.70	11.00 ± 0.67	0.979
APTT (s), Mean ± SD	30.42 ± 3.79	30.72 ± 3.51	0.352
FIB (g/L), Mean ± SD	3.05 ± 0.62	3.02 ± 0.47	0.504
WBC (10^9/L), Mean ± SD	5.69 ± 1.93	5.58 ± 1.72	0.481
PLT (10^9/L), Mean ± SD	256 ± 141	251 ± 77	0.622
Age (years), n (%)			0.657
≤50	167 (40.1%)	68 (38.2%)	
>50	249 (59.9%)	110 (61.8%)	
BMI (kg/m^2), n (%)			0.123
<28	320 (76.9%)	142 (79.8%)	
≥28	96 (23.1%)	36 (20.2%)	
Anti-tumor treatment, n (%)			0.560
No	263 (63.2%)	117 (65.7%)	
Yes	153 (36.8%)	61 (34.3%)	
CVC placement, n (%)			0.247
No	284 (68.3%)	130 (73.0%)	
Yes	132 (31.7%)	48 (27.0%)	
T, n (%)			0.462
0-1	207 (49.8%)	97 (54.5%)	
2-4	202 (48.5%)	81 (45.5%)	
N, n (%)			0.142
0	197 (47.4%)	96 (53.9%)	
1-3	219 (52.6%)	82 (46.1%)	
M, n (%)			0.168
0	402 (96.6%)	176 (98.9%)	
1	14 (3.4%)	2 (1.1%)	
TNM Stage, n (%)			0.795
0-I	138 (33.2%)	61 (34.3%)	
II-IV	278 (66.8%)	117 (65.7%)	
Pathological classification, n (%)			0.088
DICS	29 (7.0%)	6 (3.4%)	
IBC	387 (93.0%)	172 (96.6%)	
Vascular tumour embolism, n (%)			0.384
No	366 (88.0%)	16 (90.4%)	
Yes	50 (12.0%)	17 (9.6%)	
Neuroinvasion, n (%)			0.965
No	393 (94.5%)	168 (94.4%)	
Yes	23 (5.5%)	10 (5.6%)	
Type of surgery, n (%)			0.556
Lumpectomy	49 (11.8%)	18 (10.1%)	
Mastectomy	367 (88.2%)	160 (89.9%)	
Diabetes, n (%)			0.825
No	369 (88.7%)	159 (89.3%)	
Yes	47 (11.3%)	19 (10.7%)	

Hypertension, n (%)			0.592
No	302 (72.6%)	133 (74.7%)	
Yes	114 (27.4%)	45 (25.3%)	

PT: prothrombin time; APTT: activated partial thromboplastin time; FIB: fibrinogen; WBC: white blood cell count; PLT: platelet count; BMI: body mass index; CVC: central venous catheter; DICS: Ductal carcinoma in situ; IBC: invasive breast cancer.

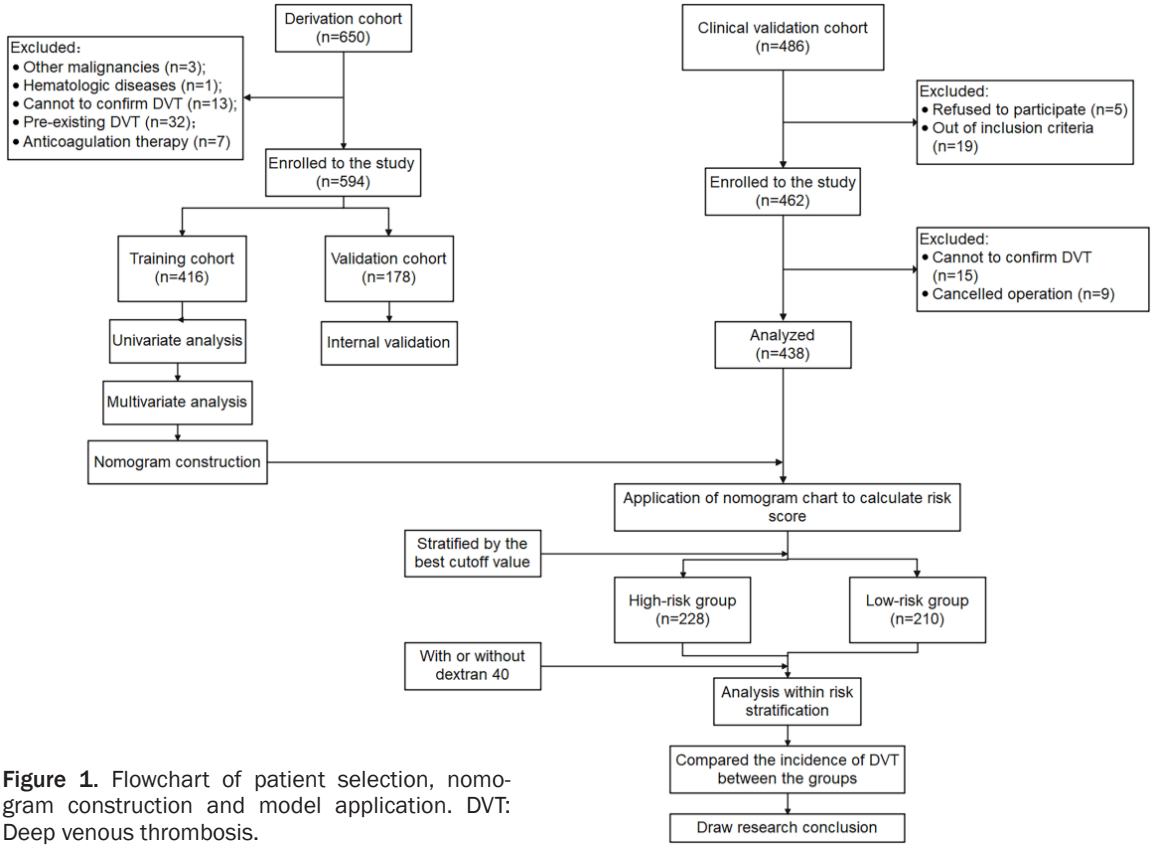

Figure 1. Flowchart of patient selection, nomogram construction and model application. DVT: Deep venous thrombosis.

using the independent samples t-test. For skewed variables (e.g., D-dimer), natural log transformation was applied, and the transformed data were confirmed to be approximately normal by Q-Q plots; the transformed data were then presented as mean ± standard deviation and compared using the independent samples t-test. Categorical variables were presented as counts (percentages) and compared using the χ^2 test, or Fisher's exact test when any expected cell count was less than 5. The retrospective cohort was randomly divided into a training set and a validation set at a 7:3 ratio. ULR was used to screen variables; those with P<0.05 were entered into a multivariable logistic regression using forward stepwise selection to identify independent factors. Discrimination of the nomogram was assessed using ROC curves and AUC, calibration using calibration curves, and clinical utility using decision curve analysis (A two-sided *P* value <0.05 was considered statistically significant).

Results

Nomogram construction and validation

Clinical and pathological characteristics: The derivation cohort included 594 patients with breast cancer who were randomly assigned in a 7:3 ratio to the training (n = 416) and validation (n = 178) sets. Among the 594 patients who were enrolled, 95 (15.9%) developed perioperative DVT. Detailed baseline information is provided in **Table 1**, and the patient selection flowchart is depicted in **Figure 1**.

Table 2. Univariate and multivariate analysis of the characteristics in training cohort

Characteristic	Training cohort		Univariable			Multivariable		
	DVT(-) *n* = 349	DVT(+) *n* = 67	OR	95% CI	*P*	OR	95% CI	*P*
D-dimer (mg/L)	0.13 ± 0.11	0.20 ± 0.15	-	-	-	-	-	-
Ln(D-dimer)	-2.32 ± 0.72	-1.84 ± 0.68	2.64	1.77, 3.95	<0.001	2.34	1.52, 3.6	<0.001
PT (s)	11.01 ± 0.68	10.93 ± 0.79	0.84	0.57, 1.25	0.401			
APTT (s)	30.51 ± 3.59	29.94 ± 4.70	0.97	0.91, 1.03	0.266			
FIB (g/L)	3.04 ± 0.63	3.10 ± 0.59	1.18	0.79, 1.78	0.423			
WBC (10^9/L)	5.60 ± 1.90	6.15 ± 2.08	1.15	1.01, 1.30	0.035	1.04	0.89, 1.20	0.643
PLT (10^9/L)	255 ± 150	260 ± 78	1.00	1.00, 1.00	0.777			
Age (years)								
≤50	159 (45.6%)	8 (11.9%)	-	-		-	-	
>50	190 (54.4%)	59 (88.1%)	4.77	2.36, 9.65	<0.001	3.19	1.38, 7.38	0.007
BMI (kg/m^2)								
<28	278 (79.7%)	42 (62.7%)	-	-		-	-	
≥28	71 (20.3%)	25 (37.3%)	2.33	1.33, 4.08	0.003	2.07	1.09, 3.93	0.026
Anti-tumor treatment								
No	221 (63.3%)	42 (62.7%)	-	-				
Yes	128 (36.7%)	25 (37.3%)	1.03	0.60, 1.77	0.921			
CVC placement								
No	239 (68.5%)	45 (67.2%)	-	-				
Yes	110 (31.5%)	22 (32.8%)	1.06	0.61, 1.86	0.832			
T								
0-1	182 (52.1%)	31 (46.3%)	-	-				
2-4	167 (47.9%)	36 (53.7%)	1.18	0.70, 1.99	0.539			
N								
0	166 (47.6%)	31 (46.3%)	-	-				
1-3	183 (52.4%)	36 (53.7%)	1.05	0.62, 1.78	0.846			
M								
0	339 (97.1%)	63 (94.0%)	-	-				
1	10 (2.9%)	4 (6.0%)	2.15	0.65, 7.08	0.207			
TNM Stage								
0-I	117 (33.5%)	21 (31.3%)	-	-				
II-IV	232 (66.5%)	46 (68.7%)	1.20	0.68, 2.12	0.529			
Pathological classification								
DICS	23 (6.6%)	6 (9.0%)	-	-				
IBC	326 (93.4%)	61 (91.0%)	0.72	0.28, 1.83	0.488			
Vascular tumor embolism								
No	304 (87.1%)	62 (92.5%)	-	-				
Yes	45 (12.9%)	5 (7.5%)	0.54	0.21, 1.43	0.217			
Neuroinvasion								
No	329 (94.3%)	64 (95.5%)	-	-				
Yes	20 (5.7%)	3 (4.5%)	0.77	0.22, 2.67	0.682			
Type of surgery								
Lumpectomy	40 (11.5%)	9 (13.4%)	-	-				
Mastectomy	309 (88.5%)	58 (86.6%)	0.83	0.38, 1.81	0.647			
Diabetes								
No	320 (91.7%)	50 (74.6%)	-	-		-	-	
Yes	29 (8.3%)	17 (25.4%)	4.05	2.09, 7.85	<0.001	2.48	1.16, 5.31	0.020

Hypertension								
No	272 (77.9%)	30 (44.8%)	-	-		-	-	
Yes	77 (22.1%)	37 (55.2%)	4.36	2.53, 7.51	<0.001	1.88	1.00, 3.55	0.041

PT: prothrombin time; APTT: activated partial thromboplastin time; FIB: fibrinogen; WBC: white blood cell count; PLT: platelet count; BMI: body mass index; CVC: central venous catheter; DICS: Ductal carcinoma in situ; IBC: invasive breast cancer. Abbreviations: ln(D-dimer), natural logarithm of D-dimer. Note: D-dimer levels were log-transformed (ln) due to a right-skewed distribution. The transformation improved model fit and met the linearity assumption of logistic regression.

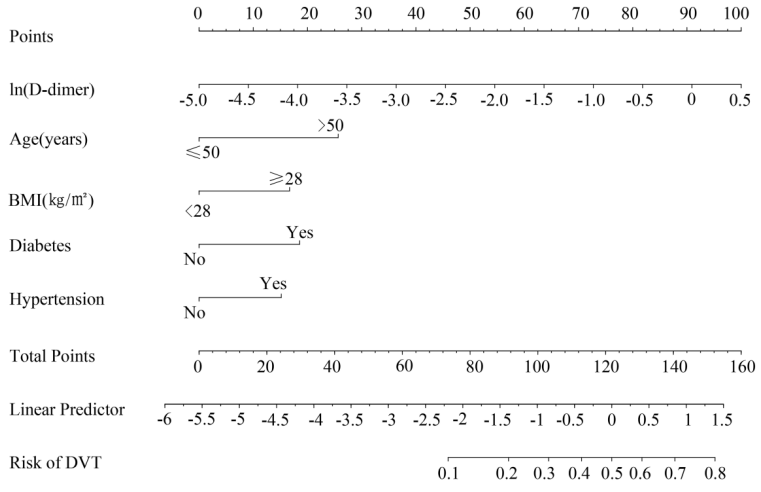

Figure 2. Nomogram prediction model for the risk of DVT in breast cancer patients perioperatively. Assign points for each risk factor level, sum the points, and find the corresponding total points on the 'Total Points' scale to read the predicted DVT risk on the 'Risk' scale. D-dimer was ln-transformed for regression analysis. Original values (mg/L) are shown in the nomogram to aid clinical interpretation.

ULR and MLR analyses of DVT risk factors: As shown in **Table 2**, five independent risk factors for DVT were identified through ULR and MLR analyses: ln(D-dimer) level (OR = 2.34; 95% CI 1.52-3.6; P<0.001), age >50 years (OR = 3.19; 95% CI 1.38-7.38; P = 0.007), BMI ≥28 kg/m^2 (OR = 2.07; 95% CI 1.09-3.93; P = 0.026), diabetes mellitus (adjusted OR = 2.84; 95% CI 1.16-5.31; P = 0.020), and hypertension (OR = 1.88; 95% CI 1.00-3.55; P = 0.041).

Nomogram construction and model validation: A nomogram was developed based on these five independent risk factors to predict the probability of postoperative DVT in patients with breast cancer (**Figure 2**). In the nomogram, each variable contributed specific points to a total score, which was converted to a linear predictor and an estimated DVT probability. For example, a ln(D-dimer) level of 0 (equivalent to a D-dimer level of 1.00 mg/L) corresponded to approximately 90 points, while a BMI of 29 kg/m^2 corresponded to approximately 17.5 points. Total scores were mapped to a scale with estimated DVT risk ranging from 10% to 80%. D-dimer and age were the strongest predictors, as reflected by their wider point ranges.

Model discrimination was evaluated using ROC curves. As shown in **Figure 3A**, **3D**, the AUC was 0.797 (95% CI: 0.737-0.856) for the training set and 0.796 (95% CI: 0.722-0.869) for the validation set, indicating stable discrimination. Calibration curves (**Figure 3B**, **3E**) demonstrated a high degree of consistency between predicted and actual values in the training cohort. While the validation cohort showed a slight deviation in the high-probability range of >0.6, overall calibration remained acceptable. DCA confirmed clinical utility (**Figure 3C**, **3F**); across threshold probabilities of approximately 10%-70%, the nomogram provided greater net benefit than strategies of intervening in all patients or none.

Cutoff value selection: ROC curve analysis identified an optimal cutoff of 16% (maximum Youden index), providing 77.3% sensitivity and 72.3% specificity in the training cohort. To prioritize the identification of high-risk patients, the clinical decision threshold was set at 10%, which increased sensitivity to 87.9% and resulted in a specificity of 55.0%. **Table 3** presents the sensitivity, specificity, false negative rate, false positive rate, and predictive values across various cutoffs.

Clinical application and the preventive effect of dextran 40

Prospective cohort characteristics: For the clinical validation cohort, 486 patients were

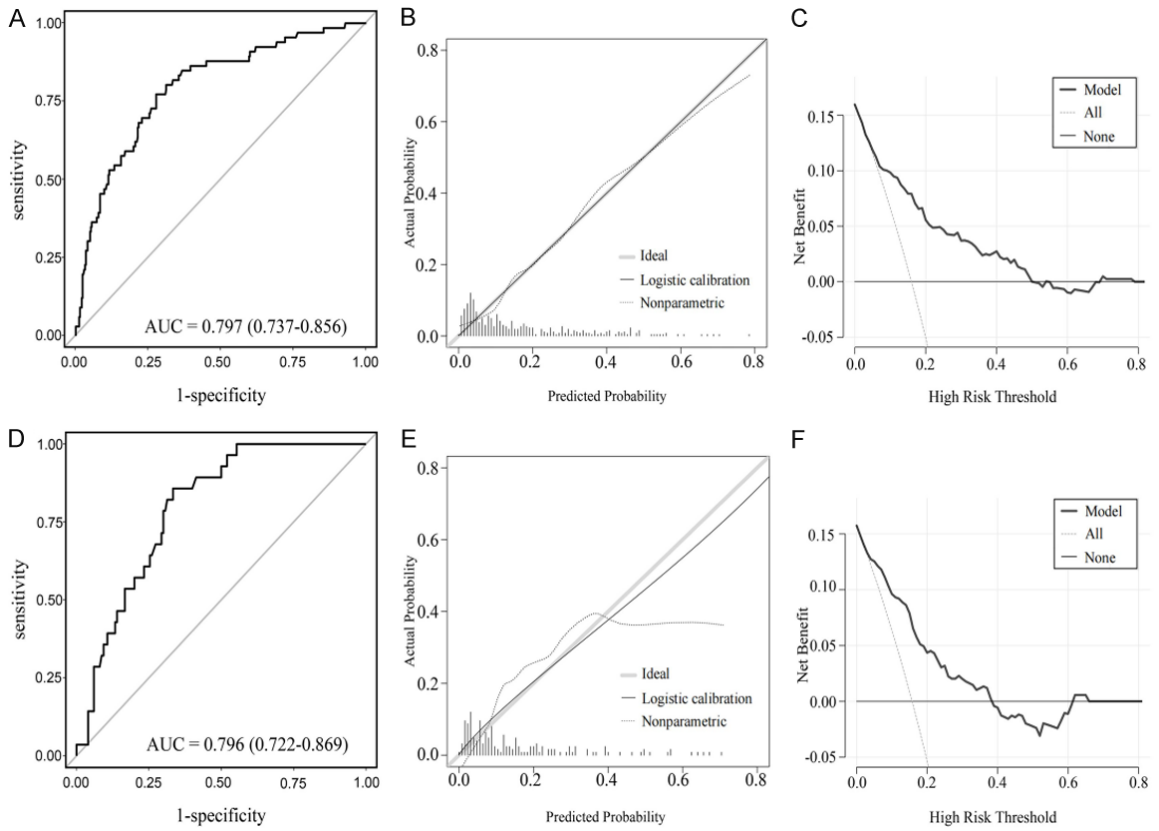

Figure 3. Nomogram verifcation for training cohort (A-C), validation cohort (D-F). ROC curves for the nomogram model in the (A) training cohort (AUC = 0.797) and (D) validation cohort (AUC = 0.796). Calibration curves for the nomogram model in the (B) training cohort and (E) validation cohort. The dashed 45-degree line represents perfect calibration. The solid line represents the performance of the nomogram; closeness to the dashed line indicates good calibration. Decision curve analysis (DCA) for the nomogram model in the (C) training cohort and (F) validation cohort. The y-axis represents net benefit. The "All" line assumes all patients receive prophylaxis. The "None" line assumes no patients receive prophylaxis. The solid line representing the nomogram shows the net benefit achieved by using the model to guide prophylaxis decisions across different threshold probabilities (x-axis). The grey line represents the number of high-risk patients identified.

Table 3. Diagnostic performance of the predictive nomogram at different cutoff values in the training and validation cohorts

	Cutoff	Sensitivity	Specificity	FNR	FPR	NPV
Training cohort	8%	87.9%	46.1%	12.1%	53.9%	95.2%
	10%	87.9%	55%	12.1%	45%	96%
	12%	84.8%	63.4%	15.2%	36.6%	95.7%
	14%	80.3%	67.4%	19.7%	32.6%	94.7%
	16%	77.3%	72.3%	22.7%	27.7%	94.4%
Validation cohort	8%	89.3%	50%	10.7%	50%	96.2%
	10%	85.7%	60.7%	14.3%	39.3%	95.8%
	12%	85.7%	64%	14.3%	36%	96%
	14%	78.6%	70%	21.4%	30%	94.6%
	16%	64.3%	74%	35.7%	26%	91.7%

FNR: False-negative rate; FPR: False positive rate; NPV: Negative predictive value. Cutoff: When the risk of postoperative thrombosis in cancer patients is higher than this value, it is recommended to take appropriate preventive measures.

Table 4. Baseline characteristics of the intervention group and control group

Characteristic	High-risk Group		Low-risk Group	
	Control Group n = 96	Intervention Group n = 132	Control Group n = 98	Intervention Group n = 112
D-dimer (mg/L), Mean ± SD	0.22 ± 0.22	0.20 ± 0.42	0.09 ± 0.06	0.10 ± 0.07
PT (s), Mean ± SD	10.78 ± 0.76	11.05 ± 0.74	10.92 ± 0.73	11.10 ± 0.70
APTT (s), Mean ± SD	29.96 ± 2.59	30.87 ± 3.22	30.56 ± 2.77	30.49 ± 2.44
FIB (g/L), Mean ± SD	3.22 ± 0.69	3.22 ± 0.54	2.95 ± 0.56	3.05 ± 1.23
WBC (10^9/L), Mean ± SD	5.82 ± 1.63	6.29 ± 2.43	5.49 ± 2.01	5.98 ± 2.74
PLT (10^9/L), Mean ± SD	241 ± 77	270 ± 71	230 ± 72	266 ± 74
Age (years), n (%)				
≤50	6 (6.3%)	7 (5.3%)	76 (77.6%)	85 (75.9%)
>50	90 (93.8%)	125 (94.7%)	22 (22.4%)	27 (24.1%)
BMI (kg/m^2), n (%)				
<28	70 (72.9%)	85 (64.4%)	90 (91.8%)	104 (92.9%)
≥28	26 (27.1%)	47 (35.6%)	8 (8.2%)	8 (7.1%)
Anti-tumor treatment, n (%)				
No	56 (58.3%)	90 (68.2%)	59 (60.2%)	71 (63.4%)
Yes	40 (41.7%)	42 (31.8%)	39 (39.8%)	41 (36.6%)
CVC placement, n (%)				
No	59 (61.5%)	91 (68.9%)	61 (62.2%)	73 (65.2%)
Yes	37 (38.5%)	41 (31.1%)	37 (37.8%)	39 (34.8%)
T, n (%)				
0-1	47 (49.0%)	54 (41.2%)	48 (49.0%)	56 (50.0%)
2-4	49 (51.0%)	78 (58.8%)	50 (51.0%)	56 (50.0%)
N, n (%)				
0	43 (44.8%)	69 (51.9%)	49 (50.0%)	54 (48.2%)
1-3	53 (55.2%)	63 (48.1%)	49 (50.0%)	58 (51.8%)
M, n (%)				
0	94 (97.9%)	130 (98.5%)	96 (98.0%)	108 (96.4%)
1	2 (2.1%)	2 (1.5%)	2 (2.0%)	4 (3.6%)
TNM Stage, n (%)				
0-I	24 (25.0%)	45 (33.8%)	35 (35.7%)	41 (36.6%)
II-IV	72 (75.0%)	87 (66.2%)	63 (64.3%)	71 (63.4%)
Pathological classification, n (%)				
DICS	8 (8.3%)	7 (5.3%)	6 (6.1%)	9 (8.0%)
IBC	88 (91.7%)	125 (94.7%)	92 (93.9%)	103 (92.0%)
Vascular tumour embolism, n (%)				
No	87 (90.6%)	120 (90.8%)	87 (88.8%)	102 (91.1%)
Yes	9 (9.4%)	12 (9.2%)	11 (11.2%)	10 (8.9%)
Neuroinvasion, n (%)				
No	87 (90.6%)	124 (93.9%)	96 (98.0%)	106 (94.6%)
Yes	9 (9.4%)	8 (6.1%)	2 (2.0%)	6 (5.4%)
Type of surgery, n (%)				
Lumpectomy	9 (9.4%)	15 (11.4%)	15 (15.3%)	22 (19.6%)
Mastectomy	87 (90.6%)	117 (88.6%)	83 (84.7%)	90 (80.4%)
Diabetes, n (%)				
No	79 (82.3%)	112 (84.8%)	95 (96.9%)	112 (100.0%)
Yes	17 (17.7%)	20 (15.2%)	3 (3.1%)	0 (0.0%)

Hypertension, n (%)				
No	60 (62.5%)	68 (51.5%)	96 (98.0%)	107 (95.5%)
Yes	36 (37.5%)	64 (48.5%)	2 (2.0%)	5 (4.5%)

PT: prothrombin time; APTT: activated partial thromboplastin time; FIB: fibrinogen; WBC: white blood cell count; PLT: platelet count; BMI: body mass index; CVC: central venous catheter; DICS: Ductal carcinoma in situ; IBC: invasive breast cancer.

screened. Following exclusions for declining participation (*n* = 5), failing the inclusion criteria (*n* = 19), inability to confirm DVT status (*n* = 15), and surgery cancellation (*n* = 9), 438 patients were included in the final analysis. Baseline characteristics are summarized in **Table 4**, and the selection flowchart is depicted in **Figure 1**.

Nomogram model risk stratification capability: Data from the clinical validation cohort (n = 438) were entered into the nomogram model and stratified using the 10% risk cutoff (total score: 72.00). This yielded a high-risk group (*n* = 228; DVT incidence 17.5%, 42/228) and a low-risk group (*n* = 210; DVT incidence 1.9%, 4/210). The incidence in the high-risk group was significantly higher than that of the low-risk group (P<0.001), validating the model's stratification effectiveness.

Stratified evaluation of the preventive effect of dextran 40: Stratified analysis revealed that in the high-risk group (n = 228), the DVT incidence in the dextran 40 intervention group (11.4%, 15/132) was significantly lower than that of the control group (26.0%, 25/96; P = 0.004). In the low-risk group (n = 210), the DVT incidence was 1.8% (2/112) in the intervention group (n = 112) compared with 2.0% (2/98) in the control group (*P*>0.999).

To adjust for potential confounders, we performed MLR analyse. Variables with P<0.05 in the ULR (ln(D-Dimer), APTT, age, BMI, diabetes, hypertension, and Dextran 40 use) were entered into the MLR model using forward stepwise selection. As shown in **Table 5**, Dextran 40 use was independently associated with a lower DVT incidence (OR = 0.40, 95% CI 0.19-0.81, P = 0.011). Other independent factors included ln(D-Dimer) (OR = 2.70, 95% CI 1.65-4.44, P<0.001) and age >50 years (OR = 9.07, 95% CI 2.45-33.56, P<0.001). APTT, BMI, diabetes, and hypertension were not statistically significant in the MLR model. Of note, this was an observational study; Dextran 40 was given at the clinician's discretion rather than by randomization. Therefore, the results reflect a statistical correlation rather than a causal relationship. These findings are exploratory and await confirmation in randomized controlled trials.

Discussion

Through ULR and MLR analyses, we identified five independent risk factors for lower extremity DVT: elevated D-dimer levels, age >50 years, BMI ≥28 kg/m^2, hypertension, and diabetes. These risk factors align with the pathophysiological mechanisms of thrombosis. Elevated D-dimer levels directly reflect secondary hyperfibrinolysis and underlying hypercoagulability, serving as a key biomarker for thrombus formation [20]. To address the skewed distribution of this variable, we applied a natural logarithmic transformation and rebuilt the model. The resulting OR for ln(D-dimer) was 2.34 (95% CI: 1.52-3.6), which falls within the range of 2-5 that is commonly reported in the literature [21, 22]. Advanced age is typically associated with vascular sclerosis, increased blood viscosity, and impaired venous valve function, all of which are believed to raise DVT risk [23]. Patients with obesity often exhibit chronic inflammation and increased procoagulant substances secreted by adipose tissue, leading to a hypercoagulable state [24]. Hypertension and diabetes are highly prevalent chronic conditions, affecting over 100 million people worldwide [25]. Hypertension exposes the circulatory system to sustained high pressure, causing vascular narrowing and sclerosis, which promote thrombus formation [26]. In patients with diabetes, hyperglycemia and insulin resistance can damage vascular endothelial cells and enhance platelet aggregation. Abnormalities in both the coagulation and fibrinolytic systems are commonly present, placing these patients in a hypercoagulable state [27]. The five risk factors identified in this study are consistent with most previous reports. Studies in patients undergoing orthopedic and abdominal

Table 5. Univariate and multivariate analysis of the clinical validation cohort

Characteristic	Univariable					Multivariable				
	N	Event N	OR	95% CI	*P*	N	Event N	OR	95% CI	*P*
Ln(D-dimer)	438	44	2.94	1.85, 4.68	<0.001	438	44	2.70	1.65, 4.44	<0.001
PT (s)	438	44	0.63	0.39, 1.03	0.064					
APTT (s)	438	44	0.88	0.78, 0.99	0.032	438	44	0.90	0.79, 1.03	0.133
FIB (g/L)	438	44	1.00	0.96, 1.03	0.847					
WBC (10^9/L)	438	44	0.90	0.76, 1.06	0.200					
PLT (10^9/L)	438	44	1.00	1.00, 1.00	0.781					
Age (years)										
≤50	174	3	-	-		174	3	-	-	
>50	264	41	10.48	3.19, 34.41	<0.001	264	41	9.07	2.45, 33.56	<0.001
BMI (kg/m^2)										
<28	349	30	-	-		348	30	-	-	
≥28	89	14	1.98	1.00, 3.93	0.049	89	14	1.48	0.69, 3.17	0.313
Anti-tumor treatment										
No	276	26	-	-						
Yes	162	18	1.20	0.64, 2.27	0.570					
CVC placement										
No	284	26	-	-						
Yes	154	18	1.31	0.70, 2.48	0.401					
T										
0-1	205	18	-	-						
2-4	233	26	1.31	0.70, 2.47	0.401					
N										
0	215	22	-	-						
1-3	223	22	0.96	0.51, 1.78	0.885					
M										
0	428	43	-	-						
1	10	1	0.99	0.12, 8.02	0.994					
TNM Stage										
0-I	145	13	-	-						
II-IV	293	31	1.20	0.61, 2.36	0.605					
Pathological classification										
DICS	30	2	-	-						
IBC	408	42	1.61	0.37, 7.00	0.525					
Vascular tumor embolism										
No	396	41	-	-						
Yes	42	3	0.66	0.20, 2.25	0.510					
Neuroinvasion										
No	413	42	-	-						
Yes	25	2	0.77	0.17, 3.36	0.724					
Type of surgery										
Lumpectomy	61	3	-	-						
Mastectomy	377	41	2.36	0.71, 7.87	0.163					
Diabetes										
No	398	35	-	-		397	35	-	-	
Yes	40	9	3.01	1.33, 6.83	0.008	40	9	1.86	0.74, 4.67	0.184

Hypertension										
No	331	22	-	-		330	22	-	-	
Yes	107	22	3.64	1.92, 6.88	<0.001	107	22	1.95	0.95, 4.03	0.070
Use of dextran 40										
No	194	27	-	-		194	27	-	-	
Yes	244	17	0.46	0.24, 0.88	0.018	243	17	0.40	0.19, 0.81	0.011

PT: prothrombin time; APTT: activated partial thromboplastin time; FIB: fibrinogen; WBC: white blood cell count; PLT: platelet count; BMI: body mass index; CVC: central venous catheter; DICS: Ductal carcinoma in situ; IBC: invasive breast cancer. Abbreviations: ln(D-dimer), natural logarithm of D-dimer. Note: D-dimer levels were log-transformed (ln) due to a right-skewed distribution. The transformation improved model fit and met the linearity assumption of logistic regression.

surgery have also found that D-dimer levels, age, BMI, hypertension, and diabetes are important predictors of postoperative DVT [28-31]. However, the relative importance ascribed to these factors varies across studies. For example, some research has reported that diabetes is not an independent risk factor for DVT [32]. These discrepancies may result from differences in baseline comorbidities, sample sizes, or statistical methods. This study integrates these five factors into a predictive model with a strong pathophysiological basis, suggesting that patients with breast cancer with these characteristics should receive proactive preventive interventions.

Existing clinical models, such as the Caprini or Khorana scores, exhibit significant limitations in predictive accuracy for patients undergoing breast cancer surgery. While some studies suggest that these models may over-identify low-risk patients who may not require prophylaxis [33], others highlight their overall diminished efficacy in patients with breast cancer [34]. For example, comparative analyses in hospitalized patients with cancer demonstrated that the Caprini score achieved a significantly higher AUC (0.705) than the Khorana score (0.581; P<0.001), identifying 82.4% of venous thromboembolism (VTE) cases compared with only 35.3% for the latter [35]. Similarly, in patients undergoing DIEP breast reconstruction, the Caprini score yielded an AUC of 0.70, with VTE incidence reaching 13% among those with scores >8 [36]. While these results affirm the relative clinical value of the Caprini score over the Khorana score, an AUC of approximately 0.70 represents only moderate predictive performance. In contrast, our proposed model demonstrated superior accuracy with an AUC of 0.797. Currently, most studies regarding postoperative thrombosis risk in patients with breast cancer rely on broad risk factor analyses, lacking accurate predictive models tailored to individual risk profiles [37]. The nomogram prediction model utilizes MLR analysis to integrate multiple independent predictors, facilitating personalized and precise risk stratification. Consequently, the current study retrospectively analyzed clinical data from 594 patients undergoing breast cancer surgery to develop a new nomogram model for predicting perioperative DVT risk. By incorporating common comorbidities and objective laboratory indicators, the model demonstrated robust discrimination, calibration, and clinical utility during internal validation.

This study is the first to demonstrate that the nomogram can effectively stratify risk within a prospective observational cohort. MLR analysis showed that Dextran 40 use was independently associated with a lower DVT incidence. It is important to emphasize that this was an observational study, dextran 40 was given at the clinician's discretion rather than by randomization. Therefore, this finding suggests a potential protective association but does not imply causation. This is consistent with previous research, including a meta-analysis of 6755 patients undergoing free-flap surgery where postoperative use of dextran 40 significantly reduced partial flap failure (RR = 0.535), indicating its utility in preventing thrombotic complications [38]. The mechanism of dextran 40, encompassing plasma volume expansion, reduced blood viscosity, and inhibition of platelet aggregation, differs from traditional anticoagulants [39]. This makes it a potential alternative for patients with contraindications to anticoagulation or an elevated bleeding risk.

However, the same meta-analysis noted that dextran 40 can cause severe pulmonary dis-

tress; therefore, its associated risks must be carefully weighed in clinical practice [38]. As current literature lacks a consensus on the optimal timing, duration, and dosage, our use of a short postoperative course requires further investigation. In the low-risk group, the extremely low event rate and limited sample size precluded reliable conclusions regarding efficacy. Crucially, our findings in the high-risk group are exploratory and should be considered hypothesis-generating rather than conclusive; prospective randomized controlled trials are essential to confirm these results.

This study has several limitations. First, the retrospective part was single-center, and selection bias cannot be completely ruled out. Second, the prospective part was observational - Dextran 40 was given at the clinician's discretion rather than by randomization, introducing indication bias. We did not record specific decision-making criteria, so residual confounding may remain. Third, the medication protocol was relatively fixed, and the optimal dose and duration are still unknown. Fourth, the nomogram model was validated only internally, lacking external validation. The calibration curve showed a deviation in the high-risk region (predicted probability >0.6), possibly due to the limited validation set (only 178 cases) and the small number of events in that region. In line with the TRIPOD statement, we did not perform post - hoc correction to avoid overfitting. Therefore, caution is needed when applying this model to high-risk patients, and future multicenter external validation is required. Importantly, due to the observational design, our findings are exploratory and do not imply causation. Randomized controlled trials are needed to validate the true efficacy and safety of Dextran 40.

Conclusion

This study identified independent risk factors for perioperative DVT in patients with breast cancer, including elevated D-dimer levels, age >50 years, BMI ≥28 kg/m^2, and comorbid hypertension and diabetes. We successfully developed and validated a robust nomogram with clinical applicability; in a prospective cohort, this model demonstrated strong discriminative power in stratifying patients into high- and low-risk categories. Dextran 40 use was independently linked to a lower DVT rate after breast cancer surgery after confounders were adjusted. But given the observational design, causation cannot be inferred. This provides exploratory evidence that Dextran 40 may help prevent DVT in these patients. Real answers on efficacy and safety will have to come from randomized controlled trials.

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Disclosure of conflict of interest

None.

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