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Construction of a nomogram prediction model for deep vein thrombosis in epithelial ovarian cancer

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Abstract

Objective: To develop and validate a nomogram prediction model for deep vein thrombosis (DVT) in epithelial ovarian cancer (EOC).

Methods: Between May 2021 and May 2024, 429 EOC patients admitted to our hospital were retrospectively identified. The patients were randomly divided into a modeling group and a validation group. Based on whether DVT occurred, the modeling group was classified into a DVT group and a non-DVT group. The influencing factors associated with DVT in EOC were analyzed using multivariable logistic regression. R software was used to construct the nomogram model. The receiver operating characteristic (ROC) curve was used to evaluate the discrimination of the nomogram. Moreover, the decision curve analysis (DCA) was used to evaluate the clinical utility of the model.

Results: Of 429 patients, 116 developed DVT, with an incidence rate of 27.04%. In the modeling group of 300 patients, 81 developed DVT, with an incidence rate of 27.00%. Multivariate logistic regression showed that age, BMI, hypertriglyceridemia, tumor staging, tumor grade, CA125 level, platelet count (PLT), and fibrinogen level (FIB) were independent risk factors for developing DVT in EOC (P < 0.05). The area under the ROC curve (AUC) for the modeling group was 0.893, and the AUC of the validation group was 0.973. The Hosmer-Lemeshow (H-L) test of the modeling group showed $\chi^2 = 7.324$ (P = 0.722), and the H-L test of the validation group showed $\chi^2 = 7.043$ (P = 0.711), suggesting good calibration. DCA curve showed that the threshold probability was between 0.08 and 0.97, the clinical value of the DVT nomogram model provided a net clinical benefit.

Conclusion: Age, BMI, hypertriglyceridemia, tumor stage, tumor grade, CA125 level, platelet count (PLT), and fibrinogen level (FIB) are significant independent risk factors for EOC patients

Received for publication March 20, 2025; accepted April 25, 2025.

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- Funding/support: None reported.
- Financial disclosures/Conflicts of interest: None reported.
- This study involving human participants was in accordance with the ethical standards of the Medical Ethics Committee of Wenzhou Central Hospital(No.202502241026000138822) and with the 1964 Helsinki Declaration. Written informed consent was obtained from all individual participants included in the study (or their legal guardians).
- The datasets used during the present study are available from the corresponding author upon reasonable request.
- C.P.: project development, data collection, data analysis, manuscript

developing DVT. The nomogram constructed with these factors demonstrates good predictive performance and clinical utility in predicting the risk of DVT in EOC patients.

Key Words: Deep vein thrombosis, Epithelial ovarian cancer, Influencing factors, Nomogram.

(Menopause 2025;32:000-000)

varian cancer was diagnosed in nearly 300,000 individuals in 2018, leading to ~180,000 deaths, underscoring its high mortality rate.¹ Epithelial ovarian cancer (EOC), which accounts for more than 90% of ovarian cancer cases, is the most common type. The insidious onset of the disease means early symptoms are often inconspicuous or absent. As the disease progresses, symptoms like abdominal bloating and reduced appetite may appear. Due to the lack of specificity in early presentation, most patients are diagnosed at an advanced stage, significantly limiting treatment options and contributing to poor prognoses.² Currently, standard treatment for EOC involves surgery and chemotherapy, but extensive surgery procedures can increase the risk of postoperative complications, potentially compromising patient survival rates.³ Venous thromboembolism, a condition where blood in the veins abnormally clots, primarily includes pulmonary embolism and deep vein thrombosis (DVT), and is a major cause of postoperative mortality.⁴ DVT can lead to swelling and thickening in the lower limbs. If left untreated, the clot may detach and travel through the bloodstream to the lungs, causing pulmonary embolism, which leads to respiratory difficulty, shock,

writing. S.X.: data collection, data analysis. A.L.: data collection, data analysis. L.L.: project development, data collection, data analysis, manuscript editing.

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eISSN: 1530-0374

DOI: 10.1097/GME.00000000002603

and, in severe cases, respiratory failure.⁵ Furthermore, studies suggest that DVT and VTE can promote tumor progression and metastasis, further adversely affecting patient prognosis.⁶ The pathogenesis of DVT complicating EOC remains unclear, and identifying factors that influence DVT onset is crucial for early prevention, which can improve patient prognosis. Nomograms, as predictive models, simplify statistical prediction models into clear graphics, generating numerical probabilities of clinical events tailored to individual patients. This helps provide personalized treatment for patients and assists clinicians in formulating preventive measures.^{7,8} Despite their utility, limited research exists regarding the use of nomograms for predicting DVT in EOC patients. Therefore, this study aims to develop and validate a nomogram prediction model for DVT complicating EOC.

METHODS

General information

We retrospectively identified 429 EOC patients admitted to Wenzhou Central Hospital between May 2021 and May 2024. The patients were randomly divided into two groups in a 7:3 ratio to either the modeling group (n = 300) or the validation group (n = 129). Patients in the modeling group were further categorized into a DVT group or a non-DVT group based on whether they developed postoperative DVT. A case flow diagram is shown in Figure 1. Inclusion criteria were: (1) pathologically confirmed diagnosis of EOC9; (2) confirmed diagnosis of postoperative DVT based on the criteria below (see the DVT diagnosis criteria section); (3) no prior anti-cancer treatment (eg, surgery, chemotherapy) or anticoagulant therapy before admission; (4) complete clinical data available for analysis. Exclusion criteria were: (1) presence of other concurrent or previous malignant tumors; (2) known underlying coagulation disorders; (3) severe hepatic or renal dysfunction; (4) previous history of venous thrombosis (VTE); (5) known primary hematological diseases; (6) psychiatric disorders precluding cooperation with the study. This study was approved by the Ethics Committee of our hospital.

DVT diagnosis criteria¹⁰

Postoperative DVT was diagnosed based on established diagnostic guidelines,¹⁰ clinical assessment, and confirmatory imaging examinations. Clinically, patients were suspected of having DVT if they presented with symptoms such as sudden swelling and pain in the affected limb, increased tension in the soft tissues, worsening pain



FIG. 1. Case flow collection diagram. DCA, decision curve analysis; DVT, deep vein thrombosis; EOC, epithelial ovarian cancer; ROC; receiver operating characteristic.

TABLE 1. Comparison of	clinical	data	between	the	modeling
group and the validation	group				

	Modeling	Validation		
	group	group		
Factor	(n = 300)	(n = 129)	χ^2	Р
Age (%)			0.041	0.840
≥ 60 y old	141 (47.00)	62 (48.06)		
< 60 y old	159 (53.00)	67 (51.94)		
BMI (%)	· /	× /	0.376	0.540
$\geq 25 \text{ kg/m}^2$	123 (41.00)	557 (44.19)		
$< 25 \text{ kg/m}^2$	177 (59.00)	72 (55.81)		
Menopausal (%)	· /	· · · ·	0.314	0.575
Yes	160 (53.33)	65 (50.39)		
No	140 (46.67)	64 (49.61)		
Hypertension (%)			0.078	0.780
Yes	83 (27.67)	34 (26.36)		
No	217 (71.60)	95 (73.64)		
Diabetes (%)	· /	× /	0.977	0.323
Yes	86 (28.67)	31 (24.03)		
No	214 (71.33)	98 (75.97)		
Hypertriglyceridemia		· · · ·	0.040	0.842
(%)				
Yes	110 (36.67)	46 (35.66)		
No	190 (63.33)	83 (64.34)		
Coronary heart disease			0.312	0.577
(%)				
Yes	10 (3.33)	3 (2.33)		
No	290 (96.67)	126 (97.67)		
Surgical history (%)			0.275	0.600
Yes	115 (38.33)	46 (35.66)		
No	185 (61.67)	83 (64.34)		
Pathological type (%)	100 (01107)	00 (0 110 1)	0.080	0 778
Slurry type	163 (54 33)	72 (55 81)	0.000	0.770
Nonslurry type	137 (45.67)	57 (44.19)		
Tumor staging (%)		. ()	0.013	0 909
I-II stage	154 (51 33)	67 (51 94)		
III-IV stage	146 (48 67)	62 (48 06)		
Tumor classification (%)	110 (10107)	02 (10100)	0.014	0 906
G1+G2	160 (53 33)	68 (52.71)	0.01.	0.000
G3	140 (46 67)	61 (47.29)		
CA125 (%)	110 (10107)	01 (1,12))	0.019	0 890
>785 U/L	81 (27.00)	34 (26 36)	0.017	0.090
< 785 U/L	219(73.00)	95 (73.64)		
PLT (%)	21) (/5.00)	<i>y</i> (<i>y</i> :, <i>y</i>)	0.092	0 761
$>400\times10^{9}/L$	128 (42.67)	53 (41 09)	0.072	0.701
$\leq 400 \times 10^{9}/L$	172(57,33)	76 (58 91)		
PT (%)	172 (37.33)	/0 (00.01)	0 107	0 744
> 12 s	154 (51 33)	64 (49 61)	0.107	0.711
< 12 s	146 (48 67)	65 (50 39)		
APTT (%)	140 (40.07)	05 (50.57)	0 266	0.606
> 33 s	150 (50.00)	61 (47 29)	0.200	0.000
< 33 s	150(50.00) 150(50.00)	68(5271)		
TT (%)	150 (50.00)	00 (02.71)	0 727	0 394
>17 s	153 (51.00)	60 (46 51)	0.727	0.574
< 17 s	147 (49 00)	69 (53 49)		
FIB (%)	117 (00.00)	07 (33.47)	0.058	0.810
>4 g/L	134 (44 67)	56 (43 41)	0.050	0.010
< 4 o/L	166 (55 83)	73 (56 59)		
· 5′		(30.37)		

APTT, activated partial thromboplastin time; BMI, body mass index; CA125, cancer antigen 125; FIB, fibrinogen; PLT, platelet count; PT, prothrombin time; TT, thrombin time.

with activity (potentially relieved by elevation), and localized tenderness over the affected vein(s). Other possible signs included visible superficial veins and (potentially) a positive Homan's sign. In severe cases, patients could develop "phlegmasia cerulea dolens." The primary imaging **TABLE 2.** Comparison of clinical data between DVT and non-DVT groups

		Non-DVT			
	DVT group	group	oup		
Factor	(n = 81)	(n = 219)	χ^2	Р	
Age (%)			13 174	< 0.001	
> 60 v old	52 (64 20)	89 (40 64)	15.174	• 0.001	
≤ 60 y old	20 (35 80)	130(50.36)			
	29 (33.80)	150 (59.50)	10 700	- 0.001	
$\sum_{n=1}^{n} \frac{1}{2} k a/m^2$	50 (61 73)	73 (33 33)	19.709	\0.001	
$\geq 25 \text{ kg/m}^2$	21(28,27)	13(55.55)			
► 2.5 Kg/III Manapausal (%)	51 (56.27)	140 (00.07)	0.008	0.754	
Vos	12 (51.85)	119 (52.99)	0.098	0.754	
I CS	42(51.05)	110(33.00) 101(46.12)			
Hypertension (%)	39 (40.13)	101 (40.12)	0.020	0.864	
Non	22 (28 40)	60 (27.40)	0.029	0.804	
Yes	23 (28.40)	150(27.40)			
	38 (71.00)	159 (72.00)	0.050	0.000	
Diabetes (%)	24 (20 (2)	(2, (20, 21))	0.050	0.823	
Yes	24 (29.63)	62 (28.31)			
No	57 (70.37)	157 (71.69)	11 010	0.001	
Hypertriglyceridemia (%)			11.018	0.001	
Yes	42 (51.85)	68 (31.05)			
No	39 (48.15)	151 (68.95)			
Coronary heart disease			0.887	0.346	
(%)					
Yes	4 (4.94)	6 (2.74)			
No	77 (95.06)	213 (97.26)			
Surgical history (%)	· /	· /	0.065	0.799	
Yes	32 (39.51)	83 (37.90)			
No	49 (60.49)	136 (62.10)			
Pathological type (%)			0.067	0.796	
Slurry type	45 (55.56)	118 (53.88)			
Nonslurry type	36 (44.44)	101(46.12)			
Tumor staging (%))	12 484	< 0.001	
I-II stage	28 (34 57)	126 (57 53)			
III-IV stage	53 (65 43)	93 (42.47)			
Tumor classification (%)	00 (00110)	<i>y</i> ((1 (1))	11 840	0.001	
G1+G2	30 (37 04)	130 (59 36)	11.010	0.001	
63	51 (62.96)	89 (40 64)			
CA125 (%)	51 (02.90)	0) (10.01)	13 378	< 0.001	
>785 U/I	51 (62.96)	30 (39 27)	15.570	• 0.001	
< 785 U/I	30(37.04)	133(60.73)			
PI T (%)	50 (57.04)	155 (00.75)	14 416	< 0.001	
$>400\times10^{9}/I$	49 (60 49)	79 (36.07)	14.410	< 0.001	
$\leq 400 \times 10^{9}/L$	32(3051)	140(63.03)			
PT (%)	52 (57.51)	140 (05.75)	1 3 2 3	0.250	
×12 a	46 (56 70)	108 (40.22)	1.323	0.230	
≥ 12.8	40(30.79)	108(49.32)			
< 12 S	35 (43.21)	111 (30.08)	0 422	0.516	
AP11 (%)	42 (52.00)	107 (40.00)	0.423	0.516	
≥ 33 S	43 (33.09)	107 (48.80)			
< 33 S	38 (46.91)	112 (51.14)	21 720	. 0. 001	
11 (%)	41 (50 (3))	110 (51.14)	21.728	< 0.001	
$\geq 1/s$	41 (50.62)	112 (51.14)			
< 1 / s	40 (49.38)	107 (48.86)	1		
FIB (%)	50 (64.00)	00 (05 / 1	17.125	< 0.001	
$\geq 4 \text{ g/L}$	52 (64.20)	82 (37.44)			
<4 g/L	29 (35.80)	137 (62.56)			

APTT, activated partial thromboplastin time; BMI, body mass index; CA125, cancer antigen 125; FIB, fibrinogen; PLT, platelet count; PT, prothrombin time; TT, thrombin time.

modality was venous compression ultrasonography (CUS), with diagnostic criteria including impaired venous blood flow (eg, lack of Doppler signal augmentation), visualization of intraluminal thrombus, venous distension at the affected site, and non-compressibility of the vein segment.

Variable	Assignment method
Age	≥ 60 y old = 1, < 60 y old = 0
BMI	$\geq 25 \text{ kg/m}^2 = 1, < 25 \text{ kg/m}^2 = 0$
Hypertriglyceridemia	Yes = 1, $no = 0$
Tumor staging	III-IV stage = 1, I-II stage = 0
Tumor classification	$G_3 = 1, G_1 + G_2 = 0$
CA125	\geq 785 U/L = 1, < 785 U/L = 0
PLT	$\geq 400 \times 10^9 / L = 1$, $< 400 \times 10^9 / L = 1$
FIB	$\geq 4 \text{ g/L} = 1, < 4 \text{ g/L} = 0$
BMI, body mass index: CA	125. cancer antigen 125: FIB. fibrinogen: PLT.

TABLE 3.	Assignment	methods of	of argument	variables

BMI, body mass index; CA125, cancer antigen 125; FIB, fibrinogen; PLT platelet count.

Collection of clinical data and laboratory indicators

Patient data were extracted retrospectively through the electronic medical record (EMR) database. Data extraction was performed by trained personnel using a standardized data collection form to ensure accuracy and validity. Data integrity was verified through cross-checks. The collected baseline clinical data included: age, body mass index (BMI), menopausal status, history of hypertension (defined as systolic blood pressure \geq 140 mm Hg or diastolic blood pressure ≥ 90 mm Hg, or current use of antihypertensive medication), diabetes mellitus, hyperlipidemia (defined as fasting total cholesterol > 5.7 mmol/L or triglycerides > 1.7 mmol/L, or current use of lipid-lowering medication), coronary heart disease, previous surgical history, EOC histological type, FIGO tumor stage, and tumor grade. Baseline laboratory indicators measured on admission included CA-125, platelet count (PLT), prothrombin time (PT), activated partial thromboplastin time (APTT), thrombin time (TT), and fibrinogen (FIB).

Statistical analysis

All statistical analyses were performed using SPSS version 25.0. Count data were analyzed with the χ^2 test and presented as cases (%). Normally distributed continuous data are analyzed using the *t* test and presented as $\bar{x} \pm s$. Logistic regression analysis is used to identify the factors influencing the occurrence of DVT in EOC patients. A nomogram model is constructed using R software. The receiver operating characteristic (ROC) curve is used to evaluate the discriminative ability of the nomogram. The clinical decision curve analysis (DCA) is employed to assess

the clinical application value of the model. A P-value of < 0.05 is considered statistically significant.

RESULTS

Comparison of clinical data between the modeling and validation groups

There were no significant differences in baseline clinical characteristics or laboratory indicators between the modeling group (n = 300) and the validation group (n = 129) (all P > 0.05), indicating successful randomization (Table 1).

Comparison of clinical data between the DVT and non-DVT groups

Of 429 patients, 116 developed DVT, with an incidence rate of 27.04%. In the modeling group of 300 patients, 81 developed DVT, with an incidence rate of 27.00%. Significant differences were observed between the two groups in terms of age, BMI, hyperlipidemia, tumor stage, tumor grade, CA125, PLT, and FIB (P < 0.05). No significant differences were found in other clinical data (P > 0.05) (Table 2).

Multivariate analysis of the factors influencing DVT in EOC patients

Using the occurrence of DVT (yes = 1, no = 0) as the dependent variable, the factors identified as potentially significant (P < 0.10) in the univariable analysis (from Table 2) were included as independent variables in the multivariable logistic regression model. Variable assignment and coding are shown in Table 3. The final multivariable logistic regression analysis revealed that age, BMI, hyperlipidemia, tumor stage, tumor grade, CA125 level, PLT count, and FIB level were independent risk factors for developing postoperative DVT in EOC patients (all P < 0.05). Odds ratios (ORs), 95% CIs, and *P*-values are presented in Table 4.

Construction of a nomogram for DVT in EOC patients

The nomogram model was constructed as follows: $P = e^{x}/(1+e^{x})$, where $x = 7.183 \times age + 4.872 \times BMI + 4.099 \times$ hyperlipidemia+3.325×tumor stage+2.529×tumor grade +2.175×CA125+3.015×PLT+3.324×FIB. The factors, based on their scores, are as follows: age, BMI, hyperlipidemia, tumor stage, FIB, tumor grade, CA125, and PLT.

Variable	β value	SE variable	Wald χ^2 variable	P variable	OR variable (95% CI)
Age	1.972	0.401	24.139	< 0.001	7.183 (3.271-15.772)
BMI	1.583	0.399	15.788	< 0.001	4.872 (2.231-10.640)
Hypertriglyceridemia	1.411	0.388	13.240	< 0.001	4.099 (1.917-8.764)
Tumor staging	1.204	0.376	10.286	0.001	3.325 (1.597-6.961)
Tumor classification	0.928	0.400	5.393	0.020	2.529 (1.156-5.536)
CA125	0.777	0.362	4.607	0.032	2.175 (1.070-4.422)
PLT	1.104	0.304	13.179	< 0.001	3.015 (1.662-5.471)
FIB	1.201	0.326	13.576	< 0.001	3.324 (1.755-6.297)
Constant	-3.956	0.420	88.676	< 0.001	0.019 (-)

4 |



FIG. 2. Construction of a nomogram for DVT in patients with EOC. DVT, deep vein thrombosis; EOC, epithelial ovarian cancer; FIB, fibrinogen; PLT, platelet count.

For example, a patient aged below 65 years (0 points), with a BMI <25 kg/m² (0 points), hyperlipidemia (71.5 points), FIGO tumor stage II-IV (62.0 points), tumor grade G3 (48.5 points), CA125 \geq 785 U/L (33.5 points), PLT \geq 400×10⁹/L (14.5 points), and FIB \geq 4 g/L (46.5 points), would have the total score of 276.5 points. Drawing a vertical line at this total score would give a prediction value of 76% (Fig. 2).

Validation of the nomogram model for DVT in EOC patients

The performance of the nomogram was evaluated in both the modeling and validation datasets. The ROC

curves were plotted (Fig. 3). The AUC for the modeling group was 0.893 (95% CI = 0.845-0.940), and the AUC for the validation group was 0.973 (95% CI = 0.950-0.995), as shown in Figure 3. The calibration curve slopes for both groups were close to 1. The Hosmer-Lemeshow (H-L) test for the modeling group was $\chi^2 = 7.324$ (P = 0.722), and for the validation group, it was $\chi^2 = 7.043$ (P = 0.711), indicating that the model has good predictive ability, as shown in Figure 4.

DCA curve for the nomogram model

The DCA curve shows that the clinical value of the nomogram model for predicting DVT in EOC patients is



FIG. 3. ROC curves. (A) ROC curve of modeling group. (B) ROC curve of validation group. ROC, receiver operating characteristic.



FIG. 4. Calibration curve. (A) Calibration curve modeling group. (B) Calibration curve of validation group. DVT, deep vein thrombosis.

high when the probability ranges from 0.08 to 0.97, as shown in Figure 5.

DISCUSSION

The etiology of ovarian cancer is complex and multifactorial, involving genetic predisposition, hormonal influences, and environmental factors that are thought to contribute to the malignant transformation of ovarian epithelial cells. The clinical treatment of this disease mainly involves cytoreductive surgery followed by a



FIG. 5. DCA curve for the nomogram. DCA, decision curve analysis.

combination of therapies (typically chemotherapy) that aim to suppress cancer cell proliferation and improve patient prognosis.11 However, patients undergoing treatment are prone to developing complications, including deep vein thrombosis (DVT). Furthermore, once a thrombus forms, it can also promote the proliferation and spread of tumor cells, leading to poor prognosis in malignant cancer patients.12 Currently, the exact pathophysiology of DVT in EOC patients is not well defined, but it is generally believed to be related to factors such as the pelvic location of the tumor, potential hormonal effects, and the systemic prothrombotic state induced by the cancer. In addition, surgery and chemotherapy can cause damage to vascular endothelium and induce inflammation, thereby increasing the risk of DVT.¹³ The results of this study show that 116 of 429 patients developed DVT, with an incidence rate of 27.04%. In the modeling group, 81 of 300 patients developed DVT, with an incidence rate of 27.00%. The high incidence rate highlights the importance of establishing a risk prediction model for prevention in clinical practice.

This study used multivariate logistic regression analysis to identify 8 independent risk factors (namely age, BMI, hyperlipidemia, tumor stage, tumor grade, CA125, PLT, and FIB) for postoperative DVT in EOC patients. The reasons for these influencing factors are as follows: (1) advanced age is a well-established risk factor for VTE. Elderly individuals often exhibit age-related vascular changes, including vascular stiffness and potential dysfunction of venous valves. As age increases, the body's vascular endothelial cells may produce more pro-coagulant compounds, while the production of anti-coagulant substances may decrease. In addition, reduced physical activity in elderly patients leads to venous stasis. Coupled with a higher prevalence of chronic comorbidities, the body can remain in a hypercoagulable state, potentially increasing blood viscosity. These factors directly or indirectly increase the risk of DVT.14,15 Strategies such as encouraging early mobilization and appropriate thromboprophylaxis are particularly important in older EOC patients. (2) Obesity is another risk factor for DVT. Obese patients often experience reduced mobility and increased intra-abdominal pressure, which slows down blood flow, affecting venous circulation. In obese patients, the inhibitor of plasminogen activator 1 (PAI-1) increases, which inhibits the activation of tissue plasminogen, thus reducing fibrinolysis. As a result, plasminogen is not converted to plasmin, ultimately increasing the risk of DVT.^{16,17} (3) In patients with hyperlipidemia, disordered lipid metabolism can increase blood viscosity and platelet aggregation, promote endothelial dysfunction, and ultimately leading to DVT.¹⁸ For patients with high blood lipids, it is recommended to adjust their diet by increasing soluble fiber intake and balancing energy consumption and intake. Proper exercise should also be conducted to burn off excess fat. (4 and 5) The presence of malignant tumors slows blood flow in the pelvic region, causing blood stagnation and a higher risk of thrombosis, particularly in patients with advanced tumor stages and grades. The tumor may invade venous vessels, damage the vascular endothelium, and accelerate the spread of cancer cells, which disrupts venous blood flow and can also involve other organs in the pelvic region. When lymph node metastasis occurs, it may compress blood vessels, altering pelvic hemodynamics and increasing the risk of DVT.^{19,20} (6) Elevated CA125 levels, often correlated with tumor burden and inflammation in EOC, were associated with increased DVT risk, potentially reflecting a more advanced or inflammatory disease state conducive to thrombosis.²¹ (7) Elevated platelet (PLT) counts (thrombocytosis), frequently observed in cancer patients, contribute to hypercoagulability. Treatment for ovarian cancer can damage blood vessel walls and surrounding tissues, activating the thrombotic mechanism and accelerating clot formation, thus increasing the risk of DVT.²² (8) Fibrinogen (FIB) plays a key role in hemostasis and coagulation. Elevated FIB levels (often seen as an acutephase reactant in cancer) indicate a hypercoagulable state in the body, which increases blood viscosity and slows blood flow, thereby increasing the risk of DVT.²³ Therefore, in clinical practice, the aforementioned factors should be carefully monitored, and corresponding treatments can be applied to reduce the incidence of DVT.

We developed a nomogram incorporating these eight factors to provide an individualized DVT risk prediction. The model demonstrated excellent discrimination, with AUCs for the modeling group and validation group of 0.893 and 0.973, respectively. Good calibration was confirmed by calibration plots and the Hosmer-Lemeshow (H-L) tests (P > 0.05 for both groups), indicating strong predictive accuracy. The DCA curve shows that the clinical value of the nomogram for assessing DVT risk in EOC patients is high when the probability is between 0.08 and 0.93. This model can assist clinicians in the prevention and early identification of high-risk groups with poor prognosis, reducing the risk of DVT.

This study, being a retrospective analysis, has some limitations. Potential selection bias may exist due to the exclusion of patients with incomplete clinical data, which might affect the actual DVT incidence and the generalizability of the findings. Furthermore, this study is a single-center study conducted in China, which limits generalizability to other populations and health care settings. The sample size, while sufficient for model development, may not fully represent the entire EOC population. Future studies should involve larger, multicenter prospective research to externally validate the nomogram and assess its clinical impact.

CONCLUSION

In summary, age, BMI, hyperlipidemia, tumor stage, tumor grade, CA125, PLT, and FIB are significant independent risk factors for postoperative DVT in EOC patients. The nomogram based on these factors provides a reliable prediction of DVT risk.

REFERENCES

- Gaona-Luviano P, Medina-Gaona LA, Magaña-Pérez K. Epidemiology of ovarian cancer. *Chin Clin Oncol* 2020;9:47. doi:10. 21037/cco-20-34
- Chen Z, Guo X, Sun S, Lu C, Wang L. Serum miR-125b levels associated with epithelial ovarian cancer (EOC) development and treatment responses. *Bioengineered* 2020;11:311-317. doi:10.1080/ 21655979.2020.1736755
- 3. Xu Z, Becerra AZ, Justiniano CF, et al. Complications and survivorship trends after primary debulking surgery for ovarian cancer. J Surg Res 2020;246:34-41. doi:10.1016/j.jss.2019.08.027
- Zhu C, Xu Z, Zhang T, et al. Updates of pathogenesis, diagnostic and therapeutic perspectives for ovarian clear cell carcinoma. *J Cancer* 2021;12:2295-2316. doi:10.7150/jca.53395
- Basaran D, Boerner T, Suhner J, et al. Risk of venous thromboembolism in ovarian cancer patients receiving neoadjuvant chemotherapy. *Gynecol Oncol* 2021;163:36-40. doi:10.1016/j.ygyno. 2021.07.030
- Miyake R, Yamada Y, Yamanaka S, et al. Tissue factor pathway inhibitor 2 as a serum marker for diagnosing asymptomatic venous thromboembolism in patients with epithelial ovarian cancer and positive D-dimer results. *Mol Clin Oncol* 2022;16:46. doi:10.3892/ mco.2021.2479
- Wang Y, Zhou H, Zhong G, Fu Z, Peng Y, Yao T. Development and validation of a nomogram to predict the probability of venous thromboembolism in patients with epithelial ovarian cancer. *Clin Appl Thromb Hemost* 2022;28:10760296221095558. doi:10.1177/ 10760296221095558
- Zheng H, Chen J, Huang J, Yi H, Zhang S, Zheng X. A novel clinical nomogram for predicting cancer-specific survival in patients with non-serous epithelial ovarian cancer: a real-world analysis based on the Surveillance, Epidemiology, and End Results database and external validation in a tertiary center. *Transl Oncol* 2024;42:101898. doi:10.1016/j.tranon.2024.101898
- Gorodnova TV, Sokolenko AP, Kuligina E, Berlev IV, Imyanitov EN. Principles of clinical management of ovarian cancer. *Chin Clin Oncol* 2018;7:56. doi:10.21037/cco.2018.10.06
- Modi S, Deisler R, Gozel K, et al. Wells criteria for DVT is a reliable clinical tool to assess the risk of deep venous thrombosis in trauma patients. *World J Emerg Surg* 2016;11:24. doi:10.1186/s13017-016-0078-1
- 11. Fahmi MN, Pradjatmo H, Astuti I, Nindrea RD. Cytokines as prognostic biomarkers of epithelial ovarian cancer (EOC): a

systematic review and meta-analysis. Asian Pac J Cancer Prev 2021; 22:315-323. doi:10.31557/APJCP.2021.22.2.315

- Gervaso L, Dave H, Khorana AA. Venous and arterial thromboembolism in patients with cancer: JACC: cardiooncology state-ofthe-art review. *JACC CardioOncol* 2021;3:173-190. doi:10.1016/j. jaccao.2021.03.001
- Cheng S, Gao H, Li Y, et al. Analysis of risk factors of postoperative lower extremity deep venous thrombosis in patients with cervical cancer. *Clin Appl Thromb Hemost* 2024;30:10760296241240747. doi:10.1177/10760296241240747
- Kim J, Kim H-J, Park S, Kim DK, Kim TH. Predictive factors of deep vein thrombosis in gynecologic cancer survivors with lower extremity edema: a single-center and retrospective study. *Healthcare* (*Basel*) 2020;8:48. doi:10.3390/healthcare8010048
- Lorchaivej S, Suprasert P, Srisuwan T, Rujiwetpongstorn J. Prevalence and risk factor of post-operative lower extremities deep vein thrombosis in patients undergoing gynecologic surgery: a singleinstitute cross-sectional study. *Thromb J* 2022;20:14. doi:10.1186/ s12959-022-00376-0
- Frischmuth T, Hindberg K, Aukrust P, et al. Elevated plasma levels of plasminogen activator inhibitor-1 are associated with risk of future incident venous thromboembolism. *J Thromb Haemost* 2022;20:1618-1626. doi:10. 1111/jth.15701
- Lilja L, Bygdell M, Martikainen J, Rosengren A, Kindblom JM, Ohlsson C. Overweight in childhood and young adulthood increases the risk for adult thromboembolic events. *J Intern Med* 2023;293:615-623. doi:10. 1111/joim.13617

- Panova-Noeva M, Wagner B, Nagler M, et al. Variation of platelet function in clinical phenotypes of acute venous thromboembolism results from the GMP-VTE project. *J Thromb Haemost* 2022;20: 705-715. doi:10.1111/jth.15595
- Roopkumar J, Poudel SK, Gervaso L, et al. Risk of thromboembolism in patients with ALK- and EGFR-mutant lung cancer: a cohort study. *J Thromb Haemost* 2021;19:822-829. doi:10.1111/jth. 15215
- 20. Yao J, Lang Y, Su H, Dai S, Ying K. Construction of risk assessment model for venous thromboembolism after colorectal cancer surgery: a Chinese single-center study. *Clin Appl Thromb Hemost* 2022;28:10760296211073748. doi:10.1177/ 10760296211073748
- Shim H, Lee YJ, Kim JH, et al. Preoperative laboratory parameters associated with deep vein thrombosis in patients with ovarian cancer: retrospective analysis of 3,147 patients in a single institute. J Gynecol Oncol 2024;35:e38. doi:10.3802/jgo.2024.35.e38
- 22. Huang G, Han F, Wu H, Fan T, Guo W. Risk Factors of lower extremity deep vein thrombosis after artificial femoral head replacement for elderly femoral neck fractures and a nomogram model construction. *Altern Ther Health Med* 2024;30: 325-331.
- 23. Fang X, Shen Y, Wang M, et al. Predictive value of Caprini risk assessment model, D-dimer, and fibrinogen levels on lower extremity deep vein thrombosis in patients with spontaneous intracerebral hemorrhage. *Front Neurol* 2024;15:1370029. doi:10.3389/fneur.2024. 1370029