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Original Article

Age, psychotropic medication, hypertension, and D-dimer based nomogram predicts venous thromboembolism in hospitalized patients with depression

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Abstract: Objective: The development and validation of a nomogram to predict the risk of venous thromboembolism VTE in depressed patients requiring hospitalization. Methods: A retrospective cohort of 726 depressed inpatients who were treated between December 2023 to October 2025 was analyzed in this study. Computer-generated random numbers were used to randomly assign the patients (to training 70% and validation 30% cohorts). Analysis was performed as logistic regression with backward stepwise selection, which is based on Akaike information criterion. Variance inflation factor was used to test multicollinearity. ROC, calibration and decision curve analysis were used to assess model performance. Results: Predictors of age, male gender, hypertension, antidepressants, antipsychotics, D-dimer ≥ 0.5 μg/mL, and length of hospital stay were independent variables. Good discrimination (AUC 0.827 training; 0.822 validation), good calibration, and good clinical utility of the nomogram were observed. Conclusion: The tested nomogram allows stratifying risk of VTE in patients with depression and could be used to inform specific thromboprophylaxis in the psychiatric ward.

Keywords: Depression, venous thromboembolism risk factors, risk, prediction model, column charts

Introduction

Depression is a prevalent mental disorder characterized by persistent and significant low mood or loss of interest, leading to impaired psychosocial functioning and reduced quality of life, and it is a major global mental health challenge [1]. The World Health Organization (WHO) predicts that major depressive disorder will be the leading cause of the global burden of disease by 2030 [2]. Venous thromboembolism (VTE) is the formation of a blood clot within a vein, which includes Deep Vein Thrombosis (DVT) and Pulmonary Embolism (PE). It is a frequent and hazardous complication during hospital consultations. PE contributes to approximately 10% of hospital-related deaths, as well as being a common preventable cause of death in hospitals [3]. Both depression and VTE contribute to increasing morbidity and mortalities worldwide [2-4].

Studies have shown that depression increases the risk of VTE [5]; the prevalence of VTE in depressed patients is as high as 8.5% [6]. In patients with mental disorders, VTE is a major cause of sudden death in patients [7]. Depression complicated by VTE seriously affects patients' quality of life, prolongs hospitalization, and increases economic and psychological burden.

The VTE risk factors in patients with depression have not been fully explained. There is a lack of systematic data and evidence-based guidelines to assess the risk of VTE in hospitalized patients with depression [8]. The existing Padua Score developed to predict VTE in hospitalized medical patients [9], cannot reliably predict VTE risk in psychiatric patients because depressed patients usually do not undergo surgery and do not suffer from severe physical illness. Besides, the clinical specificity of concomitant VTE in depressed patients is not obvious, mostly asymptomatic type, and the symptoms of VTE in depressed patients are easily confounded by psychiatric symptoms and psychological fac-

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tors, which makes timely and accurate diagnosis and treatment very difficult [10].

This study aimed to evaluate potential risk factors for VTE in patients with depression and to develop a VTE risk prediction nomogram. Current VTE risk assessment instruments like the Padua Prediction Score have been designed to assess VTE risks in general medical patients and are not sensitive in the psychiatric population. The peculiar clinical characteristics of depressed inpatients such as psychomotor retardation, hyperprolactinemia caused by drugs, and poor mobility cannot be reflected using the traditional tools. Thus, the study assists in determining depression-specific VTE predictors and the creation of the first nomogram specific to the hospitalized depressed population to enhance the initial risk stratification and prevention policies in psychiatric units.

Materials and methods

Study population

This study was conducted at Sir Run Run Shaw Hospital, Zhejiang University School of Medicine. A retrospective study was conducted by retrieving data from the clinical database of inpatients in the mental health department. Originally, this study employed a retrospective cohort grouping approach where patients diagnosed with depression were divided into VTE and non-VTE groups on the basis of inclusion/exclusion criteria and diagnosis of VTE was confirmed during hospitalization.

The study received approval from Sir Run Run Shaw Hospital Medical Ethics Committee (Ethics No. 2024-Research-0001, approved by Sir Run Run Shaw Hospital). Informed consent from the patient was not required because only de-identifying data obtained during routine care was used. The medical records of 726 patients with depression who were admitted to psychiatric wards between December 01, 2023 to October 01, 2025 were systematically reviewed. The researchers conducted a systematic chart review of 726 patients with depression who were admitted to mental health wards for treatment, using a form to record information, including demographic characteristics, laboratory tests, past medical history, and medication use.

Inclusion criteria: (1) Meeting the diagnostic criteria for depression in the 10th edition of the International Classification of Diseases (ICD-10) [11]. (2) During hospitalization, DVT or PE was diagnosed through venous ultrasound of both lower limbs or CT angiography of the pulmonary arteries; the diagnostic criteria for VTE were based on the Guidelines for the Diagnosis and Treatment of VTE [12]. (3) Patients admitted to our hospital's psychiatric ward with complete medical records. Exclusion criteria: (1) Clearly diagnosed with VTE before admission. (2) Receiving anticoagulation therapy before admission. (3) Association with severe bleeding disorders.

VTE screening program

D-dimer is a sensitive indicator of venous thrombosis, and plasma D-dimer levels are routinely measured on admission in every depressed patient, and when plasma D-dimer levels exceed 0.5 μg/ml, patients receive lower extremity Doppler ultrasound to screen for venous thrombosis. The threshold is common in clinical settings and generally proposed in the global recommendations of VTE diagnosis owing to its strong negative predictive value to rule out thrombosis. Studies have shown that D-dimer levels have a negative predictive value of 99% for thrombosis [13]. Patients are considered to have no thrombosis when D-dimer levels are less than 0.5 micrograms per milliliter. Diagnostic confirmation was performed by CT angiography (CTPA) scan of the pulmonary artery in patients with suspected PE.

Data collection

Based on previous findings, we retrospectively collected data on demographics and risk factors that may influence concurrent VTE in patients with depression. The analysis focused on specific factors related to patients with depression including age, sex, duration of illness, BMI, history of smoking, history of alcohol consumption, use of antidepressants, use of antipsychotics, presence of comorbidities (hypertension, hyperlipidemia, diabetes mellitus, cerebrovascular disease, hyperprolactinemia, active tumor, history of orthopedic surgery, other surgery in 1 month), presence of catatonia, history of restraints; relevant monitoring parameters such as pituitary prolactin, homocysteine,

coagulation and fibrinolysis parameters including prothrombin time (PT), active partial thromboplastin time (APTT), international normalized ratio (INR).

The Medical Ethics Committee of Sir Run Run Shaw Hospital, Zhejiang University School of Medicine granted its approval of this study (Approval No: 2024-2014-01). A waiver on patient consent was made because of the use of retrospective anonymized data.

Statistical analysis

The distribution of variables was evaluated through Shapiro-Wilk tests. Continuous data with a normal distribution was expressed as mean with standard deviation, while non-normally distributed data was presented as median with interquartile ranges. Categorical data was reported as numbers with corresponding percentages. Normally and non-normally distributed continuous data were compared among groups using Student's t-test and Mann-Whitney U test, respectively. Differences in categorical data among groups were evaluated employing the chi-squared test.

Patients were randomly allocated into training and validation cohorts in a 7:3 ratio using computer-generated random numbers without replacement. Logistic regression analysis was applied to identify independent risk factors related to VTE in depressed inpatients. Variance Inflation Factor (VIF) was used to determine the multicollinearity between the variables and all the variables proved to have a VIF lower than 3, meaning that they were not highly multicollinear. Variables demonstrating an association with VTE in univariable analysis ($P < 0.05$) were incorporated into the subsequent multivariable logistic regression analysis. To mitigate overfitting, the backward stepwise process, grounded in the Akaike information criterion, was employed for variable selection. Subsequently, estimated odds ratio (OR) and the corresponding 95% confidence interval (95% CI) were computed.

Based on the outcomes of the preceding multivariable logistic regression analysis, a nomogram was developed. The performance of the nomogram was evaluated through assessments of discrimination, calibration, and clinical net benefits. The model's discrimination capacity was assessed using the receiver operating characteristic curve (ROC), derived from 1000 bootstrap resamples, and the area under the curve was subsequently calculated. The calibration curve scrutinized the concordance between the predicted values and the actual observed values. Additionally, decision curve analysis quantified standardized net benefits across various threshold probabilities to evaluate the clinical usefulness. The aforementioned performance metrics of the nomogram were assessed in both the training and validation cohorts.

A significance level of < 0.05 for a two-tailed *p*-value was deemed statistically significant in this study. The statistical analyses were performed using SPSS software (version 25.0) and R software (version 3.6.3). Internal validation was performed using an independent validation cohort derived from a 7:3 training - validation split, with model performance evaluated through receiver operating characteristic analysis, calibration plots, and decision curve analysis.

Results

Baseline characteristics

A total of 726 depressed inpatients were included in this study and were randomly divided into a training cohort (n = 508) and a validation cohort (n = 218). In the training cohort, 53 patients (10.4%) were diagnosed with VTE, while 455 patients did not develop VTE. In the validation cohort, VTE was identified in 26 patients (11.9%), whereas 192 patients were VTE-free. The prevalence of VTE was comparable between the training and validation cohorts, supporting the consistency and representativeness of the dataset for subsequent analyses.

Table 1 presents the baseline categorical data of VTE and non-VTE. No significant differences were observed between groups, indicating good baseline comparability. Data are expressed as percentages of patients with or without each characteristic. Statistical comparisons between groups were performed using Pearson's chi-square test. The results indicate that there were no significant differences between the thrombotic and non-thrombotic groups for any of the categorical variables (all $P > 0.05$), suggesting that the groups were well

Table 1. Baseline categorical characteristics of depressed inpatients in the training and validation cohorts

Variable	Training (n = 508) n (%)		Validation (n = 218) n (%)		*P*-value
	VTE (n = 53)	Non-VTE (n = 455)	VTE (n = 26)	Non-VTE (n = 192)	
Smoking	52 (10.3)	456 (89.7)	192 (87.9)	26 (12.1)	0.464
Antidepressant use	83 (16.4)	425 (83.6)	179 (82.3)	39 (17.7)	0.642
Hypertension	92 (18.2)	416 (81.8)	177 (81.0)	41 (19.0)	0.779
Hyperlipidemia	7 (1.4)	501 (98.6)	217 (99.6)	1 (0.4)	0.238
Diabetes	33 (6.5)	475 (93.5)	204 (93.5)	14 (6.5)	0.988
Antipsychotic use	46 (9.1)	462 (90.9)	200 (91.8)	18 (8.2)	0.702
Cerebrovascular disease	8 (1.6)	500 (98.4)	211 (97.0)	7 (3.0)	0.212
Physical restraint	5 (1.0)	503 (99.0)	218 (100.0)	0 (0.0)	0.125
Stupor	1 (0.2)	507 (99.8)	218 (100.0)	0 (0.0)	0.494
History of surgery	205 (40.4)	303 (59.6)	123 (56.3)	95 (43.7)	0.398
Malignant tumor	26 (5.1)	482 (94.9)	210 (96.1)	8 (3.9)	0.493
Sex (Male)	379 (74.7)	129 (25.3)	67 (30.7)	151 (69.3)	0.121
Orthopedic surgery	30 (5.9)	478 (94.1)	199 (91.3)	19 (8.7)	0.161
Alcohol consumption	52 (10.3)	456 (89.7)	189 (86.6)	29 (13.4)	0.217

Table 2. Comparison of continuous clinical and laboratory variables between depressed inpatients with and without venous thromboembolism

Variable	VTE (n = 53)	Non-VTE (n = 455)	*p*-value	Test
Age (years)	46.74 ± 19.91	46.28 ± 19.79	0.86	t-test
Activated partial thromboplastin time (APTT, s)	35.61 ± 3.78	36.35 ± 4.24	0.041	t-test
Length of hospital stay (days)	9.26 ± 3.32	9.20 ± 2.95	0.851	t-test
Body Mass Index (BMI, kg/m^2)	22.15 ± 3.86	21.92 ± 3.21	0.057	t-test
Pituitary prolactin (ng/mL)	20.19 (13.0)	20.14 (14.75)	0.002	Mann-Whitney U
Homocysteine (< 15 μmol/L)	14.03 (4.6)	13.78 (4.6)	0.296	Mann-Whitney U
International Normalized Ratio (INR)	1.01 (0.09)	1.03 (0.08)	0.575	Mann-Whitney U
D-dimer (DDI, μg/mL)	0.56 (0.32)	0.54 (0.31)	0.86	Mann-Whitney U
Prothrombin time (PT, s)	13.37 (0.9)	13.43 (0.9)	0.174	Mann-Whitney U

balanced with respect to smoking status, medication use, comorbidities, sex, history of surgery, and alcohol consumption.

Table 2 summarizes the continuous variables in depressed inpatients stratified by VTE status. Normally distributed variables (Age, APTT, length of hospital stay, BMI) were compared using independent samples t-test, while non-normally distributed variables (Pituitary prolactin, Homocysteine, INR, D-dimer, PT) were compared using the Mann-Whitney U test. Variables showing significant differences included Age, APTT, Pituitary prolactin, and D-dimer, indicating these factors may contribute to VTE risk in this population.

Multivariable logistic regression analysis identified several independent predictors of VTE (**Table 3**). Increasing age was associated with a higher risk of VTE (OR 1.09 per year; 95% CI 1.05-1.13; P < 0.001). Antidepressant use (OR 12.55; 95% CI 4.55-34.64; P < 0.001) and antipsychotic use (OR 13.99; 95% CI 4.70-41.70; P < 0.001) showed strong associations with VTE. Male sex (OR 3.28; 95% CI 1.02-10.53; P = 0.046) and hypertension (OR 3.05; 95% CI 1.19-7.78; P = 0.020) were also independently associated with VTE occurrence. Elevated D-dimer levels (≥ 0.5 mg/mL) were associated with a four-fold increase in VTE risk (OR 4.09; 95% CI 2.62-6.39; P < 0.001). Longer hospital stay was inversely associated with VTE

Table 3. Multivariable logistic regression analysis for predictors of venous thromboembolism

Variable	β (SE)	Odds Ratio (OR)	95% CI for OR	*p*-value
Age (per year)	0.083 (0.019)	1.09	1.05-1.13	< 0.001
Length of hospital stay (per day)	-0.180 (0.075)	0.84	0.72-0.97	0.016
Antidepressant use	2.530 (0.518)	12.55	4.55-34.64	< 0.001
Antipsychotic use	2.639 (0.557)	13.99	4.70-41.70	< 0.001
Male sex	1.189 (0.595)	3.28	1.02-10.53	0.046
Hypertension	1.113 (0.478)	3.05	1.19-7.78	0.020
D-dimer ≥ 0.5 mg/mL	1.409 (0.228)	4.09	2.62-6.39	< 0.001

risk (OR 0.84 per day; 95% CI 0.72-0.97; P = 0.016).

Development of a nomogram for predicting venous thromboembolism

A nomogram for predicting VTE was formulated by incorporating seven risk factors identified through multivariable logistic regression analysis (**Figure 1A**).

In the nomogram, weighted points were assigned to age, hypertension, antidepressant use, antipsychotic use, and pituitary prolactin level. The total score, calculated by summing the points for each predictor, ranged up to 280 and corresponded to an estimated VTE risk between 0.1 and 0.9. A higher total score indicated a greater likelihood of developing VTE.

Evaluation of the predictive nomogram's performance

The discriminative performance of the predictive nomogram was evaluated using receiver operating characteristic (ROC) curve analysis. In the training cohort, the nomogram demonstrated good discrimination with an area under the ROC curve (AUC) of 0.827 (95% CI: 0.766-0.888). Consistent performance was observed in the validation cohort, with an AUC of 0.822 (95% CI: 0.726-0.918), indicating stable and robust predictive accuracy across datasets (**Figure 1B**).

The calibration plot demonstrates good agreement between predicted and observed VTE probabilities, with the bias-corrected curve closely following the ideal reference line across the prediction range (**Figure 1C**). Bootstrap internal validation (1,000 resamples) shows minimal calibration error (mean absolute error = 0.008), indicating stable and well-calibrated model performance.

Decision curve analysis shows that the nomogram provides a higher net benefit than the treat-all and treat-none strategies across a broad range of threshold probabilities (≈0.05-0.60), indicating good clinical utility for individualized VTE risk prediction (**Figure 1D**).

To further explore the functional relationship between D-dimer levels and the predicted risk of VTE a partial dependence plot was generated based on the nomogram model (**Figure 1B**). The partial dependence plot showed that there was a nonlinear association between D-dimer and VTE probability in which the risk is more likely to rise as the concentration exceeds 0.5 mg/mL. This then reinforces the clinical significance of this threshold in depressed inpatients. The plot demonstrates a clear positive association between increasing D-dimer levels and the predicted probability of VTE after adjusting for other covariates in the model.

Notably, the predicted VTE risk increases gradually at lower D-dimer concentrations and rises more sharply as D-dimer levels exceed the clinical threshold of 0.5 mg/mL, indicating a nonlinear relationship. This finding suggests that elevated D-dimer levels contribute disproportionately to VTE risk in depressed inpatients and supports its role as a strong and clinically meaningful predictor in the nomogram. **Figure 2** has demonstrated the nonlinear relationship between the D-dimer levels and the probability of predicted VTE.

Discussion

In this sample of over 700 individuals with depression, 10.9% of patients were found to have VTE. This is comparable to an earlier study that reported VTE prevalence of 8.5% in depressed patients [6]. In this retrospective cohort study, we developed and internally vali-

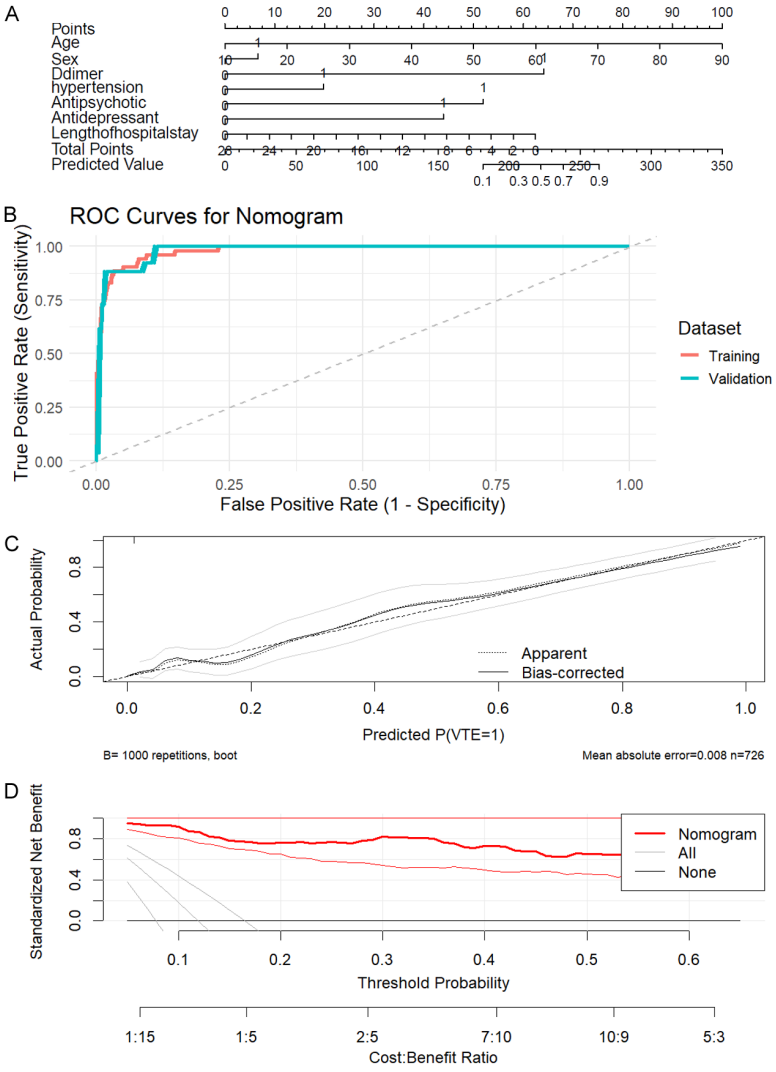

Figure 1. Development of a nomogram for predicting venous thromboembolism. A. Nomogram for predicting the risk of venous thromboembolism VTE in hospitalized patients with depression. The nomogram incorporates age, sex, D-dimer level, hypertension, antipsychotic use, antidepressant use, and length of hospital stay. Each predictor is assigned a point value according to its relative contribution to VTE risk. The total points, obtained by summing the individual scores, correspond to an estimated probability of VTE. B. Receiver operating characteristic (ROC) curves showing the discriminative performance of the nomogram for venous thromboembolism prediction in the training (red) and validation (blue) cohorts. The dashed diagonal line represents random prediction. The areas under the curve indicate good and consistent predictive accuracy across both datasets. C. Calibration plot of the nomogram for prediction of venous thromboembolism VTE. D. Decision curve analysis for the nomogram predicting venous thromboembolism VTE. The x-axis represents threshold probabilities, while the y-axis shows the net clinical benefit. The solid line represents the nomogram model.

dated a nomogram to predict the risk of VTE in hospitalized patients with depression. The model incorporated seven readily available clinical variables and demonstrated good discrimination, calibration, and clinical utility. To our knowledge, this is the first study to establish a nomogram-based VTE risk prediction tool specifically for depressed inpatients in a general hospital psychiatric setting. Ward et al. suggested that higher polygenic risk for major depressive disorder was associated with an increased risk of VTE [14]. Enga et al. found that patients who often felt depressed were 1.59 times more likely to have VTE than those who did not feel depressed [15]. Subsequent studies have also confirmed the conclusion that depression is significantly linked to a higher risk of VTE [16]. A prospective study from the Institute of Forensic Medicine showed that 31% of patients who died of pulmonary thromboembolism had a history of mental disorder [17], so we cannot underestimate the serious consequences of VTE. However, there is a lack of evidence-based thrombosis guidelines for patients with psychiatric disorders, and VTE prophylaxis is not included as a core measure in psychiatric VTE quality assessment. This highlights the lack of clinical attention to VTE in patients with mental illness. The currently used Padua Score, although proven effective in assessing thrombotic risk in non-surgical patients, does not reliably predict venous thrombosis in depressed patients [18]. In fact, in our sample, patients with depression complicating VTE had low-risk Padua scale scores at admission, suggesting that the Padua scale is not effective in predicting the occurrence of VTE in depressed patients due to its low sensitivity and poor

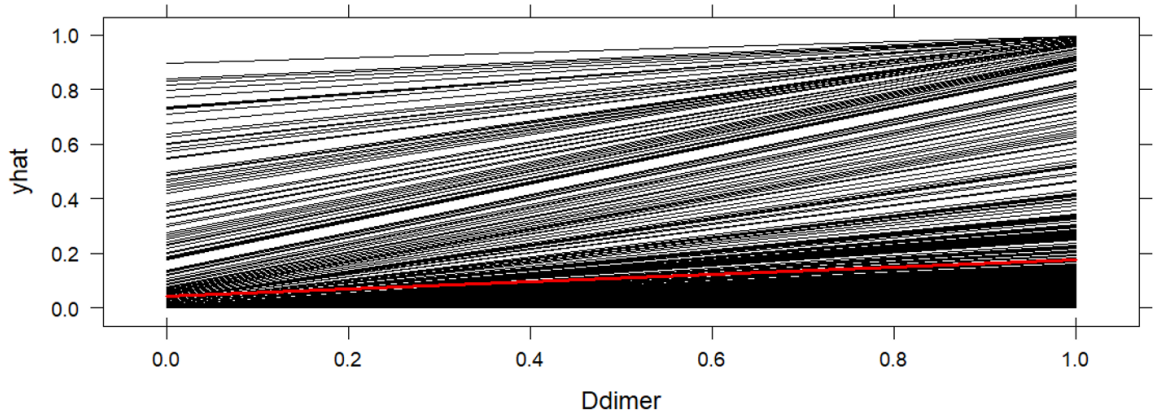

Figure 2. Partial dependence plot showing the effect of D-dimer (DDI) on predicted probability of venous thromboembolism VTE. The x-axis represents D-dimer levels, and the y-axis shows the predicted probability of VTE based on the nomogram model. The plot demonstrates the adjusted relationship between D-dimer concentration and VTE risk while holding other predictors constant.

applicability. This may be related to the specificity of the depressed group. Therefore, for early identification of patients at high risk for VTE, development of preventive measures, and appropriate implementation of intervention strategies, identification of specific risk factors in patients with depression is very important and helpful. Further studies should concentrate on the pathogenesis of depression complicated by VTE and establish a VTE assessment program to further improve the clinical utility of predictive models, guiding the implementation of thromboprophylaxis, and improving the quality and safety of nursing care.

Antidepressants and antipsychotics are commonly prescribed to treat depression. Studies have shown a significant correlation between the use of these drugs and VTE. Antidepressants and antipsychotics are common medications used in the treatment of depressed patients, and the medications have been reported to be strongly associated with VTE [19, 20], and our study found that the use of antidepressant and antipsychotic drugs were independent risk factors and strong predictors of VTE. Data from a study suggests that women taking antidepressants had a 40% higher risk of VTE compared to those not taking antidepressants, regardless of the type of antidepressant used [21, 22]. Comprehensive evidence indicates that there is an increased risk of VTE associated with the use of antidepressants compared to not using them, and this association is more consistent in women [10]. However, it is controversial whether the link between antidepressants and VTE is due to the medication, the depression itself, or a combination of both. The use of antidepressants may increase the VTE risk through decreased platelet synthesis and activity, and the increased risk of venous thrombosis with tricyclic antidepressants (TCAs) is the most pronounced of all classes of antidepressants. Studies have confirmed that the use of conventional antipsychotics also substantially increases the risk of idiopathic VTE [23, 24]. The evidence shows that the use of antipsychotic medication is significantly linked to a higher risk of VTE. Specifically, antipsychotic medication use is associated with a 1.5-fold increased risk of VTE and a 3.7-fold increased risk of pulmonary thrombosis when compared to those not using antipsychotic medication [25]. The use of antipsychotics for over 24 months was found to increase the VTE risk by 32% and atypical antipsychotics by 73% [26]. The possible mechanism by which first-generation antipsychotics increase the risk of thrombosis is related to increased platelet aggregation, while the possible mechanism for second-generation antipsychotics is related to the presence of antibodies against cardiolipin [27]. Growing evidence suggests that second-generation antipsychotics, such as clozapine and olanzapine, carry a higher risk of VTE [28]. Antipsychotics are known to act as dopamine D2 receptor antagonists, which can cause hyperprolactinemia, prolactin is a hormone produced by the pituitary gland that is suppressed by dopamine.

Its role as a platelet aggregation coactivator, potentially increases the risk of thrombosis [29]. It has been suggested that treatment with 5-hydroxytryptamine reuptake inhibitors or antipsychotic medications may lead to increased serum prolactin levels [30]. Our study also found that hyperprolactinemia is prevalent in patients with depression and is a significant risk factor for developing VTE. However, medication is still one of the most common treatments for patients with depression. Therefore, it is important to consider the risk of VTE related to antidepressant and antipsychotic medications, as VTE is a potentially fatal but treatable condition. Clinicians should exercise caution when administering medications and thoroughly assess the VTE risk in patients exposed to medications and other potential VTE risks. Medications with a lower risk of causing the disease should be selected, and patients should be closely monitored for any signs of VTE such as changes in temperature, heart rate, oxygen saturation, lower extremity edema, and D-dimer levels. Preventive care should be initiated early in patients at high risk of thrombosis to prevent potentially fatal embolic events and medical disputes.

Age is a well-known and important independent risk factor for VTE, and the incidence of VTE increases with age [31]. A study on the prevalence of VTE in hospitalized psychiatric patients taking antipsychotic medications found that VTE was most commonly diagnosed in patients with mood disorders over the age of 65, with an incidence of approximately 0.034% [32]. As individuals age, the likelihood of developing underlying conditions such as cerebrovascular, neurological, and cardiovascular diseases increases, which in turn raises the risk of VTE [33, 34]. Therefore, assessing and screening for VTE risk in elderly patients with depression is important.

Our study found that comorbid hypertension was an independent risk factor for the developing VTE, which has been less frequently investigated in existing studies related to risk factors for depression complicating VTE. Study shows that hypertension is a noteworthy risk factor for cardiovascular diseases, including heart failure and cerebrovascular disease, and hypertension is an independent risk factor for VTE compared to individuals with normal blood pressure [23]. VTE is a frequent complication of hypertension and results in abnormal platelet activation [35]. Therefore, controlling blood pressure and reducing multifactorial cardiovascular risks is beneficial in preventing thrombosis-related complications in hypertensive patients.

There are several limitations of this study. First, the sample size is limited, with only 726 patients selected, which may not provide sufficient relevant data. Additionally, the patients had multiple risk factors, and there is insufficient evidence to determine the most influential risk factors. Second, the psychiatric ward in our hospital primarily admits patients with psychosomatic diseases, which may limit our ability to identify other possible risk factors that are related to VTE in patients with depression. Studies have reported risk factors for certain treatments, including the use of restraining devices and non-convulsive electroconvulsive therapy (MECT) [36]; we were unable to collect data on these factors due to treatment criteria not being met or the treatment not being carried out in our ward. This may result in an underestimation of VTE risk. Third, depression is characterized by reduced volitional activity, and decreased activity is a known and established risk factor for VTE. However, depressed patients are typically not completely bedridden and inactive, and it may be difficult to quantify the amount of patient activity without proper data. This may lead to overlooking important risk factors for VTE in depressed patients. Although this study was conducted between July 2021 and July 2023, the data reflect recent real-world clinical practice, including contemporary psychiatric treatment strategies and current VTE screening protocols. Therefore, the findings remain timely and clinically relevant for present-day risk stratification in hospitalized patients with depression. Future studies could investigate the correlation between the level of specific activity in depressed patients and VTE. Finally, the data in this study came from a single center, and we only internally validated the model. Future studies could conduct multicenter, prospective studies to externally validate the data, thus further improving the performance and stability of the model. Although this single-center retrospective study has limitations, it revealed the increasing prevalence of VTE in patients with depression in the

clinic. Furthermore, this study identifies risk factors for VTE in patients with depression and presents a more reliable risk prediction model. Interestingly, the risk of VTE was negatively correlated to longer hospital stay. This can be attributed to the fact that high levels of D-dimer were detected at an early stage and diagnostic screening was followed by early anticoagulation therapy administration, hence reducing the length of stay of patients diagnosed with VTE. Regardless of the cause of VTE in patients with depression, medical personnel must be aware of the existence of VTE in patients with mental disorders and implement risk assessment. Thrombosis risk assessment is a key factor in preventing VTE.

Conclusion

The VTE risk prediction model for depression is effective in identifying patients with depression who are at high risk of thrombosis. It can be used in clinical practice to guide early and effective prevention. Future directions include assessing more comprehensive risk factors for VTE in depression, defining intervention strategies for individualized VTE prevention during treatment of depressed hospitalized patients, and ultimately reducing the incidence of VTE and its serious life-threatening consequences.

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Disclosure of conflict of interest

None.

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