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Original Article

Incidence and independent risk factors of lower extremity deep vein thrombosis in patients with acute carbon monoxide poisoning: a retrospective study

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Abstract: Objective: To investigate the incidence and independent risk factors of lower extremity deep vein thrombosis (DVT) in patients with acute carbon monoxide poisoning and to evaluate the predictive performance of a risk factor model to support early identification and individualized intervention for high-risk patients. Methods: This retrospective cohort study included 180 patients diagnosed with acute carbon monoxide poisoning from January 2021 to December 2023. Lower extremity venous ultrasound was performed within 48 hours of admission to detect DVT, and patients were categorized into DVT and non-DVT groups. Clinical data - including demographic characteristics, poisoning-related variables, and biochemical markers - were collected and analyzed. Variables with statistical significance were subjected to logistic regression analysis to identify independent risk factors for DVT. Spearman correlation analysis and receiver operating characteristic (ROC) curve analysis were further conducted to assess variable relationships and the predictive performance of the risk model. Results: Among the 180 patients, 23 (12.78%) developed DVT. Spearman correlation analysis showed that coma duration, carboxyhemoglobin concentration, C-reactive protein, procalcitonin, D-dimer, creatinine, blood urea nitrogen, lactate dehydrogenase, myoglobin, and creatine kinase were positively correlated with DVT (all $P < 0.001$), whereas earlier initiation of hyperbaric oxygen therapy and higher albumin levels were negatively correlated ($r = -0.397$, $P < 0.001$). Logistic regression identified coma duration, D-dimer level, and carboxyhemoglobin concentration as independent risk factors for DVT. The ROC curve demonstrated good predictive performance, with an area under the curve of 0.888 (95% CI: 0.827-0.948, $P < 0.05$). Conclusion: Lower extremity DVT is relatively common in ptients with acute carbon monoxide poisoning. Coma duration, D-dimer levels, and delayed initiation of hyperbaric oxygen therapy are significantly associated with increased risk. The proposed risk factor model demonstrates strong predictive value and may assist in early clinical detection and targeted prevention strategies.

Keywords: Carbon oxide poisoning, venous thrombosis of lower limbs, risk factors

Introduction

Carbon monoxide (CO) poisoning is one of the most common forms of acute intoxication, typically resulting from the accumulation of CO gas in enclosed environments [1]. Due to its colorless, odorless, and tasteless properties, and often low atmospheric concentrations, CO is difficult to detect, increasing the risk of unintentional inhalation and rapid intoxication. Clinical manifestations vary by severity, ranging from mild symptoms such as headache and fatigue to severe outcomes including coma, re-spiratory failure, and death. CO exerts its toxic effects by binding to hemoglobin to form carboxyhemoglobin (COHb), which impairs oxygen transport and leads to tissue hypoxia [2]. This hypoxia results in cellular injury and metabolic disturbances, particularly in oxygen-sensitive organs such as the brain, heart, and kidneys, potentially causing long-term sequelae. Although survival rates in acute CO poisoning have improved, studies have reported an increased incidence of complications - particularly coagulopathy and thrombosis - which significantly impact both mortality and long-term quality of life [3].

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Deep vein thrombosis (DVT), characterized by thrombus formation in the deep veins of the lower extremities (typically the thigh or calf), arises primarily from three interrelated factors - hypercoagulability, venous stasis, and endothelial injury - collectively known as Virchow's triad. Acute CO poisoning may exacerbate each of these mechanisms, thereby promoting thrombogenesis. CO-induced hypoxia can activate the coagulation cascade and increase coagulation factor activity. In addition, prolonged immobility during intoxication contributes to venous stasis, while systemic inflammatory responses to CO poisoning can damage the vascular endothelium, further increasing the risk of thrombosis [4, 5]. Clinically, DVT is not only associated with localized thrombotic events but also with serious complications such as pulmonary embolism (PE) and chronic venous insufficiency [6].

Despite extensive research into the diagnosis and treatment of acute CO poisoning, studies exploring its associated complications - particularly the development and risk factors of DVT - remain scarce. Existing literature largely centers on acute management and neurological recovery, with limited attention paid to thrombotic events. With advances in hyperbaric oxygen therapy and prolonged post-poisoning rehabilitation, patient survival has markedly improved; however, this has also led to increased recognition of secondary complications, such as thrombosis. Therefore, understanding the incidence and independent risk factors of DVT in patients with acute CO poisoning is of significant clinical importance.

Materials and methods

Sample size calculation

Based on a DVT prevalence of 14.3% reported by Feng et al. [10], the required sample size was calculated using the formula: $N = Z^2 \times [P \times (1-P)]/E^2$, where $Z = 1.96$ (corresponding to a 95% confidence level), $E = 0.05$ (margin of error), and $P = 0.143$ (estimated prevalence). The calculated minimum sample size was 188 patients. The final sample size was determined based on available clinical data.

Study subjects

A retrospective analysis was performed on 180 patients diagnosed with acute CO poisoning who were admitted to the Emergency Department of Hanzhong Central Hospital between January 2021 and December 2023. All patients underwent lower extremity venous Doppler ultrasound within 48 hours of admission and were categorized into two groups: DVT group (n = 23) and non-DVT group (n = 157). This study was approved by the Ethics Committee of Hanzhong Central Hospital.

Inclusion and exclusion criteria

Inclusion criteria: (1) Diagnosis of acute CO poisoning confirmed by blood gas analysis and meeting established clinical diagnostic criteria [7]; (2) First-time admission, age ≥ 18 years; (3) Lower extremity venous ultrasound performed within 48 hours of admission; (4) Availability of complete medical history and laboratory records.

Exclusion criteria: (1) History of chronic venous insufficiency, venous thromboembolism, or previous DVT; (2) Severe multi-organ failure (e.g., heart, liver, or kidney dysfunction); (3) Prior use of anticoagulant or thrombolytic therapy; (4) End-stage malignancy or life expectancy < 3 months; (5) Major trauma, surgery, or prolonged bed rest within 3 months prior to admission; (6) Current use of anticoagulants, antiplatelet agents, or oral contraceptives.

Standard treatment protocol

Initial management of acute CO poisoning includes prompt administration of 100% oxygen via a non-rebreather mask or mechanical ventilation based on severity. In severe cases, hyperbaric oxygen therapy (HBOT) is initiated promptly to accelerate CO elimination and improve tissue oxygenation. Supportive care includes hemodynamic stabilization, correction of electrolyte and acid-base imbalances, and protection of vital organs (brain, heart, and kidneys). Patients are closely monitored for COHb levels, oxygen saturation, and vital signs.

For patients with DVT, anticoagulation therapy (e.g., low-molecular-weight heparin or direct oral anticoagulants) is initiated to prevent thrombus extension. Coagulation parameters such as prothrombin time (PT) and activated partial thromboplastin time (aPTT) are monitored regularly. Compression therapy using graduated compression stockings is recommended to reduce edema and promote ven-

ous return. Early mobilization and physical therapy are encouraged to prevent further thrombus formation. In cases of massive thrombosis or complications such as pulmonary embolism, thrombolytic therapy or surgical intervention may be considered.

Data collection

Patient Demographics: Age, sex, height, weight, body mass index (BMI), smoking history, and alcohol consumption history were extracted from the hospital's electronic medical record (EMR) system.

CO Poisoning-Related Variables: Duration of coma (from CO exposure to recovery of consciousness, as reported by the patient or family), time from diagnosis to initiation of HBOT, and COHb concentration.

Biochemical Parameters: Laboratory tests at admission included inflammatory markers (C-reactive protein [CRP], procalcitonin [PCT]), coagulation marker (D-dimer), nutritional marker (albumin [Alb]), and biochemical markers (creatinine [Cr], blood urea nitrogen [BUN], lactate dehydrogenase [LDH], myoglobin [Mb], and creatine kinase [CK]). All tests were performed in a single standardized laboratory to minimize inter-assay variability. Hemolyzed specimens were excluded. Data accuracy was verified by two independent researchers.

Diagnosis and evaluation of lower extremity DVT

All patients underwent bilateral lower extremity venous color Doppler ultrasound within 48 hours of admission. Diagnoses were confirmed independently by two radiologists, each with over five years of experience.

Ultrasound criteria for DVT diagnosis [8]: Presence of hypoechoic or anechoic areas in the venous lumen; Partial or complete occlusion; Absence or restriction of blood flow on Doppler imaging; Incomplete vein compression under probe pressure.

Clinical features (supportive, not diagnostic): Limb swelling, pain, skin discoloration, and increased local temperature.

Statistical analysis

Data were analyzed using SPSS version 26.0. Continuous variables were expressed as mean ± standard deviation ($\bar{x} \pm sd$) and compared using independent samples t-tests. Categorical variables were expressed as frequency and percentage (n, %) and compared using the chi-square test or Fisher's exact test. Spearman correlation analysis was used to explore associations between clinical variables and DVT occurrence. Multivariate logistic regression was employed to identify independent DVT risk factors. Receiver operating characteristic (ROC) curves were constructed to evaluate model performance. A two-tailed *p*-value < 0.05 was considered statistically significant.

Results

Incidence of DVT

A total of 180 patients with acute CO poisoning were included in this study, conducted in the Emergency Department from January 2021 to December 2023. Among them, 23 patients (12.78%) developed lower extremity DVT. The distribution of DVT cases is shown in **Figure 1** and **Table 1**. Muscular venous thrombosis: 18 cases (78.26%), involving the left lower limb (n = 7), right lower limb (n = 5), and both lower limbs (n = 6). Other deep venous thrombosis: 5 cases (21.74%), involving the popliteal vein (n = 2), peroneal vein (n = 1), and posterior tibial vein (n = 2); localization included the left lower limb (n = 2), right lower limb (n = 2), and both limbs (n = 1).

Comparison of basic characteristics

No statistically significant differences were found between the DVT and non-DVT groups regarding age, sex, BMI, smoking history, or alcohol use (all P > 0.05; **Table 2**).

Comparison of poisoning-related indicators

The DVT group had a significantly longer coma duration and a longer delay in initiating HBOT compared to the non-DVT group (both P < 0.01). Additionally, the COHb concentration was significantly higher in the DVT group (P < 0.05; **Table 3**).

Comparison of biochemical indicators

Levels of CRP, PCT, and D-dimer were significantly elevated in the DVT group (all P < 0.01), while Alb levels were significantly lower (P < 0.05). Moreover, Cr, BUN, LDH, Mb, and CK levels were also significantly higher in the DVT group (all P < 0.01; **Table 4**).

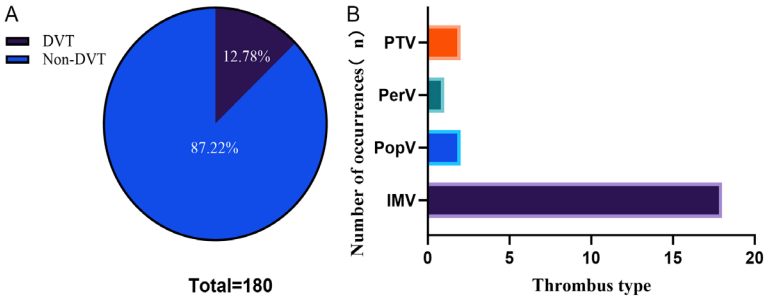

Figure 1. Distribution of lower extremity DVT. Note: DVT: Deep vein thrombosis.

Table 1. Distribution of lower extremity DVT

Patient distribution	n	Proportion (%)
Total number of patients	180	100
DVT	23	12.78
Intermuscular vein thrombosis	18	78.26
Left lower limb	7	30.43
Right lower limb	5	21.74
Both lower limbs	6	26.09
Other deep vein thrombosis	5	21.74
Left lower limb	2	8.7
Right lower limb	2	8.7
Both lower limbs	1	4.35

DVT: Deep vein thrombosis.

Correlation between clinical indicators and DVT

Spearman correlation analysis revealed positive correlations between DVT occurrence and the following variables: coma duration, COHb concentration, CRP, PCT, D-dimer, Cr, BUN, LDH, Mb, and CK (all P< 0.001). In contrast, time to initiation of HBOT and Alb levels were negatively correlated with DVT occurrence (both P < 0.001; **Table 5**).

Risk factors for DVT in acute CO poisoning

Univariate logistic regression identified delayed HBOT and lower Alb levels as potential protective factors, while prolonged coma duration, higher COHb, CRP, PCT, D-dimer, Cr, BUN, LDH, Mb, and CK were potential risk factors for DVT (**Table 6**). Multivariate logistic regression further confirmed that coma duration and D-dimer level were independent risk factors for DVT in patients with acute CO poisoning (P < 0.05; **Table 7**).

Predictive value of the multivariate model (ROC analysis)

ROC analysis demonstrated that the combined model using coma duration and D-dimer levels to predict DVT yielded an AUC of 0.888 (95% CI: 0.827-0.948, P < 0.05), with a sensitivity of 79.3% and specificity of 80.1% (**Figure 2**).

Ultrasound findings of lower extremity deep veins

Ultrasound evaluation was performed in all patients to detect DVT. Representative images are shown in **Figure 3**. **Figure 3A** illustrates a normal ultrasound of the right lower limb, showing clearly visualized common femoral, superficial femoral, deep femoral, popliteal, posterior tibial, and peroneal veins. The vessel walls are smooth and continuous, with no intraluminal echogenicity. Color Doppler displays normal bidirectional flow signals, and spectral Doppler shows continuous, phasic waveforms indicating unobstructed venous return.

Figure 3B displays an abnormal scan with partial hypoechoic filling of the superficial femoral vein lumen, consistent with thrombus formation. The affected vein shows loss of compressibility, and spectral Doppler indicates absent flow in the occluded segment. The adjacent saphenous vein appears dilated (maximum diameter: 0.36 cm), likely reflecting compensatory dilation due to proximal outflow obstruction.

Discussion

CO poisoning is a common and potentially life-threatening toxicological emergency that can result in neurological impairment, metabolic disturbances, multi-organ dysfunction, and death. Emerging clinical evidence has highlighted the increasing occurrence of DVT in patients with acute CO poisoning, drawing growing attention from the medical community [9]. Although the management of CO poisoning is well-established, thrombotic complications and their pathophysiological mechanisms remain underexplored. This study investigated the incidence and independent risk factors of lower extremity DVT in patients with acute CO poisoning, aiming to inform early identification of high-

Table 2. Comparison of basic characteristics between DVT and Non-DVT groups

Index	DVT group (n = 23)	Non-DVT group (n = 157)	t/x^2	*P*
Age (years)	49.56±10.84	47.84±12.11	0.699	0.490
Gender (male/female, n)	16/7	101/56	0.242	0.623
BMI (kg/m^2)	25.57±4.05	24.74±2.70	0.956	0.348
Smoking history (yes/no, n)	12/11	71/86	0.390	0.532
Drinking history (yes/no, n)	10/13	64/93	0.061	0.805

Note: BMI: Body Mass Index, DVT: Deep vein thrombosis.

Table 3. Comparison of acute poisoning-related indicators between DVT and Non-DVT group

Index	DVT group (n = 23)	Non-DVT group (n = 157)	*t*	P
Coma time (h)	7.17±3.15	4.22±1.80	4.387	< 0.001
Time to start hyperbaric oxygen therapy (h)	2.01±0.85	3.05±1.30	-5.061	< 0.001
Carboxyhemoglobin concentration (%)	22.30±5.73	15.54±4.92	5.370	< 0.001

Note: DVT: Deep vein thrombosis.

Table 4. Comparison of biochemical indicators between DVT and Non-DVT groups

Index	DVT group (n = 23)	Non-DVT group (n = 157)	t/x^2	*P*
CRP (mg/L)	18.59±9.98	8.47±3.37	4.823	< 0.001
PCT (ng/mL)	1.98±0.78	1.18±0.69	4.651	< 0.001
D-Dimer (mg/L)	2.88±0.73	1.42±0.69	9.035	< 0.001
Alb (g/L)	30.96±3.05	35.50±3.27	-6.608	< 0.001
Cr (μmol/L)	97.98±15.86	80.00±13.02	5.188	< 0.001
BUN (mmol/L)	7.86±1.69	5.86±1.74	5.278	< 0.001
LDH (U/L)	336.63±53.52	279.90±47.50	4.813	< 0.001
Mb (μg/L)	136.88±26.71	93.94±25.12	7.255	< 0.001
CK (U/L)	498.81±129.20	388.48±87.26	3.965	< 0.001

Note: CRP: C-Reactive Protein, PCT: Procalcitonin, Alb: Plasma Albumin, Cr: Creatinine, BUN: Blood Urea Nitrogen, LDH: Lactate Dehydrogenase, Mb: Myoglobin, CK: Creatine Kinase, DVT: Deep vein thrombosis.

Table 5. Correlation analysis of clinical indicators with DVT occurrence in acute CO poisoning patients

Index	r_s	*P*
Coma time	0.334	< 0.001
Time to start hyperbaric oxygen therapy	-0.281	< 0.001
Carboxyhemoglobin concentration	0.367	< 0.001
CRP	0.415	< 0.001
PCT	0.310	< 0.001
D-Dimer	0.488	< 0.001
Alb	-0.397	< 0.001
Cr	0.351	< 0.001
BUN	0.333	< 0.001
LDH	0.349	< 0.001
Mb	0.442	< 0.001
CK	0.286	< 0.001

Note: CRP: C-Reactive Protein, PCT: Procalcitonin, Alb: Plasma Albumin, Cr: Creatinine, BUN: Blood Urea Nitrogen, LDH: Lactate Dehydrogenase, Mb: Myoglobin, CK: Creatine Kinase, DVT: Deep vein thrombosis, CO: carbon monoxide.

risk individuals and enable personalized preventive strategies.

In this study, the incidence of DVT among acute CO poisoning patients was 12.78%, closely aligning with the previously reported rate of 14.3% [10]. This relatively high incidence may be attributed to systemic inflammatory responses, hemodynamic disturbances, and activation of the coagulation cascade triggered by CO poisoning. CO exerts its toxic effects by binding with hemoglobin to form CO-Hb, thereby impairing oxygen transport and inducing tissue

Table 6. Univariate logistic regression analysis of risk factors for DVT occurrence in acute CO poisoning patients

Index	β	S.E.	Wald	*P*	OR	95% CI for Exp (β)
Coma time	0.610	0.130	21.897	0.000	1.840	1.425-2.375
Time to start hyperbaric oxygen therapy	-0.715	0.209	11.753	0.001	0.489	0.325-0.736
Carboxyhemoglobin concentration	0.254	0.053	22.564	0.000	1.289	1.161-1.431
CRP	0.351	0.07	24.927	0.000	1.421	1.238-1.631
PCT	1.492	0.348	18.406	0.000	4.446	2.249-8.791
D-Dimer	3.148	0.607	26.858	0.000	23.29	7.081-76.597
Alb	-0.438	0.09	23.562	0.000	0.645	0.541-0.770
Cr	0.088	0.018	22.971	0.000	1.092	1.053-1.132
BUN	0.686	0.157	19.116	0.000	1.986	1.460-2.702
LDH	0.024	0.005	19.513	0.000	1.024	1.013-1.035
Mb	0.069	0.014	24.852	0.000	1.071	1.042-1.100
CK	0.011	0.003	18.817	0.000	1.011	1.006-1.016

Note: CRP: C-Reactive Protein, PCT: Procalcitonin, Alb: Plasma Albumin, Cr: Creatinine, BUN: Blood Urea Nitrogen, LDH: Lactate Dehydrogenase, Mb: Myoglobin, CK: Creatine Kinase, OR: odds ratio, DVT: Deep vein thrombosis, CO: carbon monoxide.

Table 7. Multivariate logistic regression analysis of risk factors for DVT occurrence in acute CO poisoning patients

Index	β	S.E.	Wald	*P*	OR	95% CI for Exp (β)
Coma time	0.618	0.197	9.857	0.002	1.855	1.261-2.728
D-Dimer	3.228	0.686	22.117	0.000	25.219	6.570-96.807

Note: OR: odds ratio, DVT: Deep vein thrombosis, CO: carbon monoxide.

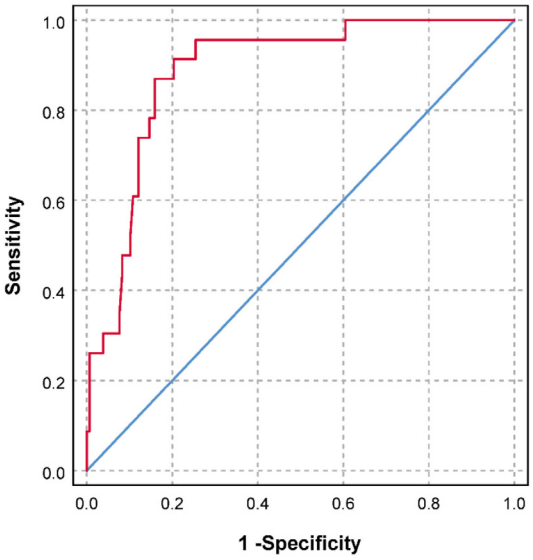

Figure 2. ROC curve analysis of the predictive performance of the multivariate regression model.

hypoxia. Hypoxia not only impairs cellular metabolism but also activates the coagulation system, promoting thrombus formation [11, 12]. Inflammatory responses further exacerbate thrombotic risk. In addition, the growing recognition of thrombotic events may be partly due to improved survival during the acute phase, which has shifted clinical focus toward long-term complications [13].

Compared with the non-DVT group, patients in the DVT group showed significantly longer coma durations, higher COHb levels, and elevated concentrations of CRP, PCT, D-dimer, Cr, BUN, LDH, Mb, and CK, alongside delayed initiation of HBOT. Albumin levels were notably lower in the DVT group. These findings suggest that hypoxia, inflammation, renal dysfunction, and tissue injury contribute to thrombogenesis. Spearman correlation analysis confirmed significant positive associations between DVT and coma duration, CRP, PCT, D-dimer, and Cr levels, while HBO initiation time and albumin levels were negatively correlated with DVT risk. Key insights include:

Coma duration: Prolonged unconsciousness is strongly associated with immobility and venous stasis, which are known contributors to thrombosis [14, 15]. It may also reflect the severity of poisoning and systemic deterioration.

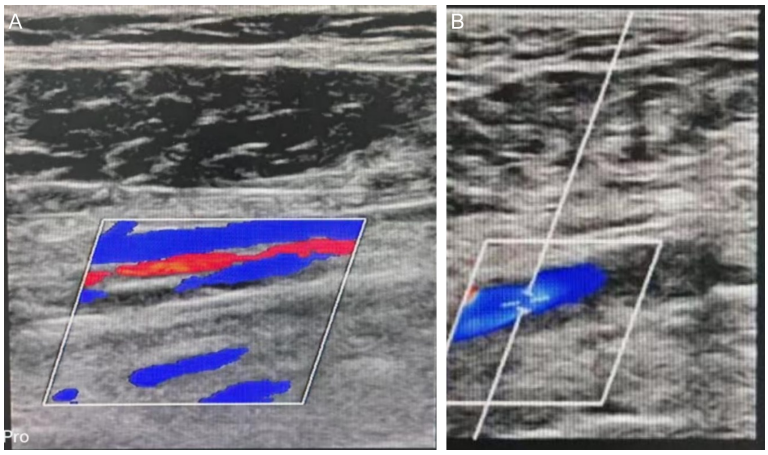

Figure 3. Results of deep vein ultrasound of the lower limbs. A. This image showed normal blood vessels and blood flow. B. This image showed varices and hypoechoic tissues.

Delayed HBO therapy: HBO improves oxygen delivery, reduces COHb levels, and mitigates hypoxic injury [16]. Delayed initiation may prolong hypoxia and elevate thrombotic risk. While some studies support the protective role of early HBO [17], discrepancies may be due to variations in study design, patient populations, and treatment timing.

COHb concentration: Higher COHb levels indicate greater severity of poisoning [18], contributing to systemic hypoxia, endothelial activation, platelet aggregation, and release of procoagulant factors. Hypoxia-induced oxidative stress may further disrupt endothelial integrity [19].

CRP and PCT: These inflammatory markers are indicative of endothelial damage and immune dysregulation [20], both of which promote a pro-thrombotic environment. Inflammation also increases blood viscosity and coagulation activity, as reflected by elevated D-dimer levels.

D-dimer: A well-established marker of active coagulation and fibrinolysis, D-dimer elevation reinforces the presence of ongoing thrombogenic processes in CO poisoning [21].

Cr and BUN: These renal markers may reflect kidney impairment, which has been associated with altered hemostas-is and increased thrombotic risk.

LDH, Mb, and CK: These markers of tissue injury indicate hypoxia-induced muscle and endothelial damage, supporting their association with thrombus formation.

Alb: Hypoalbuminemia may reflect malnutrition or acute-phase inflammation [23], both of which can disrupt vascular homeostasis and reduce endogenous anticoagulant capacity.

Multivariate logistic regression identified coma duration and D-dimer level as independent predictors of DVT in patients with acute CO poisoning. These findings are consistent with previous reports linking prolonged immobility, altered blood rheology, and coagulation activation to thrombosis in this context [24]. The established predictive value of D-dimer for DVT [25-27] further supports its inclusion in the risk model. ROC curve analysis revealed strong diagnostic performance for the model, with an area under the curve (AUC) of 0.888, sensitivity of 79.3%, and specificity of 80.1%, indicating the model's potential clinical utility for early risk stratification and targeted intervention.

Despite these findings, this study has limitations. As a single-center retrospective study, it may be subject to selection bias and limited generalizability. Prospective multicenter studies are warranted to validate the predictive model. Moreover, the molecular mechanisms underlying CO-induced thrombosis were not investigated in this study. Future research should explore the molecular and cellular pathways involved in coagulation dysregulation and vascular injury following CO exposure to improve our understanding of DVT pathophysiology in this setting.

In conclusion, DVT in patients with acute CO poisoning is closely linked to alterations in blood rheology, coagulation activation, endothelial injury, systemic inflammation, renal dysfunction, and impaired oxygenation. These factors interact synergistically to elevate thrombosis risk. Early identification of high-risk patients based on clinical and biochemical markers is critical for timely intervention and preven-

tion of thrombotic complications in acute CO poisoning.

Disclosure of conflict of interest

None.

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