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Prevalence of Deep Vein Thrombosis in Patients with COVID-19 Admitted to the Intensive Care Unit

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A B S T R A C T

Introduction: Critically ill patients with COVID-19 exhibit a pronounced hypercoagulable state, predisposing them to deep vein thrombosis during ICU admission. Inflammation-driven endothelial dysfunction, hypoxia, and immobilization contribute to this elevated risk. Determining the true prevalence of DVT in this population is essential for optimizing thromboprophylaxis strategies and improving clinical outcomes.

Material and methods: This retrospective observational study evaluated critically ill patients with concurrent COVID-19 and active malignancy admitted to a tertiary ICU. Clinical records, laboratory parameters, and imaging-confirmed thrombotic events were systematically analyzed. Multivariable logistic regression was applied to identify independent risk factors for vascular thrombosis, providing clinically relevant insights for risk stratification and management in this high-risk population.

Results: Thrombotic complications occurred in 28.3% of critically ill ICU patients with concurrent COVID-19 and active malignancy, while 71.7% remained free of thrombosis (P<0.001). Among affected patients, venous thromboembolism predominated, with lower-extremity deep vein thrombosis and pulmonary embolism being the most frequent manifestations, whereas arterial, cerebral, and coronary events were uncommon.

Conclusion: In conclusion, thrombotic complications remain a common and clinically significant problem among ICU patients with concurrent COVID-19 and active malignancy. Venous thromboembolism predominates, and its occurrence is associated with older age, higher BMI, specific cancer types, greater comorbidity burden, prolonged ICU stay, increased need for mechanical ventilation, and higher mortality.

Introduction

Coronavirus disease 2019 (COVID-19), caused by the severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2), has posed unprecedented challenges to healthcare systems worldwide since its emergence in late 2019. Although initially recognized as a primarily respiratory illness, accumulating evidence has demonstrated that COVID-19 is a complex multisystem disease with profound effects on the cardiovascular and hematologic systems. Among these complications, thromboembolic events particularly deep vein thrombosis (DVT) have emerged as a major contributor to morbidity and mortality, especially among critically ill patients admitted to intensive care units (ICUs) (1).

The hypercoagulable state observed in patients with severe COVID-19 appears to be multifactorial, involving endothelial dysfunction, excessive inflammatory response, platelet activation, and dysregulation of the coagulation cascade.

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SARS-CoV-2 infection is associated with a marked inflammatory surge, often referred to as a "cytokine storm," characterized by elevated levels of interleukin-6, tumor necrosis factor-α, and other proinflammatory mediators. These inflammatory pathways promote thrombin generation, impair fibrinolysis, and enhance platelet aggregation, collectively predisposing patients to venous and arterial thrombosis (2). In critically ill patients, these mechanisms are further amplified by immobilization, hypoxia, mechanical ventilation, and the frequent presence of multiple comorbidities. Deep vein thrombosis represents a clinically significant manifestation of COVID-19–associated coagulopathy. Undiagnosed or untreated DVT may progress to pulmonary embolism, which is a potentially fatal complication and has been reported at alarmingly high rates in ICU patients with COVID-19. Early observational studies from Europe and China suggested that standard thromboprophylaxis regimens might be insufficient to prevent venous thromboembolism in this population, raising concerns regarding the true prevalence of DVT and the adequacy of conventional preventive strategies (3).

Critically ill patients admitted to ICUs are inherently at high risk for venous thromboembolism due to prolonged immobilization, use of sedatives and neuromuscular blockers, invasive procedures, and systemic inflammation. In the context of COVID-19, this baseline risk is significantly exacerbated. Hypoxemia, a hallmark of severe COVID-19, promotes thrombosis through increased blood viscosity and hypoxia-inducible transcription factors that stimulate coagulation pathways. Moreover, direct viral invasion of endothelial cells via angiotensin-converting enzyme 2 receptors results in endothelial injury, which plays a pivotal role in initiating and propagating thrombus formation (4).

Laboratory abnormalities commonly observed in severe COVID-19, such as elevated D-dimer levels, prolonged prothrombin time, and increased fibrinogen concentrations, further support the presence of a distinct prothrombotic phenotype. Among these markers, D-dimer elevation has been consistently associated with disease severity, ICU admission, and poor clinical outcomes. Several studies have reported a strong correlation between markedly elevated D-dimer levels and the occurrence of DVT, underscoring the clinical relevance of systematic thrombotic risk assessment in critically ill COVID-19 patients (5).

Despite increasing recognition of thrombotic complications, the reported prevalence of DVT among ICU patients with COVID-19 has varied widely across studies, ranging from less than 10% to more than 40%. This heterogeneity may be attributed to differences in study design, patient populations, diagnostic strategies, timing of screening, and anticoagulation protocols. Some investigations relied solely on clinically suspected cases, while others employed routine duplex ultrasonography screening, which has been shown to detect a substantial proportion of asymptomatic DVT cases (6). Consequently, the true burden of DVT in this population remains incompletely defined.

The diagnosis of DVT in critically ill COVID-19 patients presents unique challenges. Clinical signs such as limb swelling or pain are often difficult to assess in sedated or mechanically ventilated patients, and laboratory markers lack specificity in the setting of systemic inflammation. Imaging studies, although essential for definitive diagnosis, may be limited by infection control concerns, hemodynamic instability, or resource constraints during pandemic surges. As a result, DVT may be underdiagnosed, potentially leading to preventable complications and worse outcomes (7).

Understanding the prevalence of DVT in ICU patients with COVID-19 is of paramount importance for several reasons. First, it provides insight into the magnitude of thrombotic risk and informs clinical vigilance. Second, accurate prevalence data are essential for evaluating the effectiveness of thromboprophylaxis strategies and guiding optimization of anticoagulation protocols. Third, identification of high-risk populations within the ICU setting may facilitate individualized preventive approaches, balancing thrombotic risk against the potential for bleeding complications (8).

Current international guidelines recommend pharmacologic thromboprophylaxis for hospitalized patients with COVID-19, including those admitted to the ICU, unless contraindicated. However, controversy persists regarding the optimal anticoagulant dose, with some studies suggesting potential benefits of intermediate or therapeutic dosing in selected patients. The lack of consensus partly reflects uncertainty regarding the actual incidence of thrombotic events such as DVT in critically ill patients, emphasizing the need for robust epidemiological data (9).

Moreover, the prevalence of DVT in ICU patients with COVID-19 may be influenced by patient-specific factors such as age, sex, obesity, diabetes mellitus, hypertension, and preexisting cardiovascular disease. These comorbidities are highly prevalent among patients with severe COVID-19 and are independently associated with increased thrombotic risk. In addition, ICU-related factors, including duration of mechanical ventilation, use of vasopressors, renal replacement therapy, and length of ICU stay, may further modulate the likelihood of DVT development (10).

From a public health and healthcare systems perspective, thrombotic complications in COVID-19 impose a substantial burden by increasing ICU length of stay, resource utilization,

and mortality. Preventing DVT and its sequelae may therefore contribute not only to improved patient outcomes but also to more efficient use of critical care resources during periods of high demand. Accurate estimation of DVT prevalence is a crucial step toward achieving these goals (11).

Despite extensive research on COVID-19, data from different regions and healthcare settings remain limited, particularly in developing countries and resource-constrained environments. Variations in patient demographics, ICU practices, availability of diagnostic tools, and anticoagulation strategies highlight the importance of region-specific studies to better characterize the epidemiology of DVT in critically ill COVID-19 patients. Such data may reveal unique patterns or risk factors that are not fully captured in studies from high-income countries (12).

In this context, investigating the prevalence of deep vein thrombosis among patients with COVID-19 admitted to the ICU is both clinically and scientifically relevant. A clear understanding of the frequency of DVT in this high-risk population can inform early detection strategies, guide prophylactic and therapeutic interventions, and ultimately reduce thromboembolic complications and mortality. Furthermore, these findings may contribute to the growing body of evidence on COVID-19–associated coagulopathy and support refinement of existing clinical guidelines (13).

Given the ongoing evolution of the COVID-19 pandemic and the emergence of new variants, critically ill patients continue to represent a vulnerable population with complex pathophysiology. Thrombotic complications remain a central concern in their management. Therefore, systematic evaluation of DVT prevalence in ICU patients with COVID-19 is essential to improving clinical decision-making and optimizing patient care in the critical care setting.

Material and methods

Study Design

This study was designed as a retrospective observational investigation. Following approval by the Ethics Committee of Tabriz University of Medical Sciences, the study was conducted on critically ill patients with confirmed COVID-19 and concurrent active malignancy who were admitted to the intensive care unit (ICU) of Imam Reza Hospital, Tabriz. All procedures were performed in accordance with institutional ethical standards and predefined inclusion and exclusion criteria.

Study Setting and Duration

The study was carried out at Imam Reza Hospital, a tertiary referral center affiliated with Tabriz University of Medical Sciences, located in Tabriz, Iran. Data collection covered a nine-month period from the beginning of the year 2025 to the end of December 2025. Medical records of patients admitted to the ICU during COVID-19 pandemic waves were systematically reviewed.

Study Population

The target population consisted of medical records of patients diagnosed with COVID-19 who were concurrently suffering from active cancer and required admission to the ICU at Imam Reza Hospital. Eligible patient records were identified through the hospital information system and ICU registries.

Study Samples

The study samples included clinical records of patients who were diagnosed with COVID-19 during the pandemic period and simultaneously hospitalized in the ICU due to active malignancy. Only patients meeting all predefined eligibility criteria were included in the final analysis.

Sample Size and Sampling Method

The minimum required sample size was calculated based on findings from a similar study, assuming a venous thromboembolism prevalence of 49% among cancer patients with COVID-19, a statistical power of 80%, and a type I error rate of 0.05. Accordingly, the minimum sample size was estimated at 185 patients. Given that a census sampling method was applied, additional records were reviewed to enhance the study's validity. Ultimately, 360 eligible patient records were included and analyzed. Patients were categorized into two groups based on the presence or absence of thrombotic complications, and relevant variables were compared between these groups.

Inclusion and Exclusion Criteria

Inclusion criteria comprised complete medical records, documented diagnostic imaging for thrombotic events, confirmed COVID-19 infection, ICU admission, presence of active malignancy, and availability of laboratory parameters related to thrombosis, including PT, PTT, platelet count, D-dimer levels, red blood cell count, hemoglobin levels, ICU length of stay, ICU survival status, administration of anticoagulation protocols, and receipt of COVID-19 treatment and anticancer therapies such as chemotherapy or radiotherapy.

Exclusion criteria included incomplete medical records, patients who had passed more than six months since their last chemotherapy session, individuals with known immunodeficiency disorders, patients with metastatic disease to other organs, preexisting coagulation disorders, morbid obesity, chronic kidney disease, chronic liver disease, and documented coagulation abnormalities.

Data Collection Procedure

After obtaining ethical approval and coordinating with Imam Reza Hospital, data collection was performed by a trained research assistant. A standardized data collection form was used for each patient. Recorded variables included age, body mass index, sex, underlying comorbidities (such as hypertension, diabetes mellitus, cardiovascular disease, and chronic pulmonary diseases including asthma and COPD), baseline laboratory values at ICU admission (platelet count, white blood cell count, red blood cell count, hematocrit, hemoglobin, PT, PTT, CRP, D-dimer, fibrinogen, and ferritin), duration of ICU stay, total hospital stay, need for mechanical ventilation, ICU mortality, ICU discharge status, use of anticoagulant therapy prior to ICU admission and during ICU stay, cancer type (including breast, brain and spinal cord, lung, gastrointestinal, reproductive system, and thyroid malignancies), and anatomical location of thrombotic involvement. Thrombotic sites were categorized as upper and lower extremity arteries, upper and lower extremity veins, carotid artery, aorta and its branches, pulmonary embolism, cerebral vessels, and coronary arteries.

Definition of Thrombotic Events

Thrombotic events were defined as deep vein thrombosis of the upper or lower extremities, pulmonary embolism, and arterial events including ischemic stroke or peripheral arterial embolism. All thrombotic events required confirmation by appropriate imaging modalities and were considered major thrombotic outcomes.

Statistical Analysis

Collected data were entered into SPSS software version 26 for statistical analysis. Descriptive statistics were presented as mean ± standard deviation for continuous variables and as frequency and percentage for categorical variables. Between-group comparisons were performed using independent t-tests, while within-group comparisons were conducted using analysis of variance (ANOVA) and the Mann Whitney U test as appropriate. A P-value of less than 0.05 was considered statistically significant, with a confidence level set at 90%.

Univariate logistic regression analysis was initially performed to assess the predictive ability of baseline risk factors for vascular thrombosis. Variables with a P-value < 0.10 in univariate analysis were entered into a multivariable logistic regression model. After excluding variables with very low prevalence, clinically relevant variables that demonstrated statistical significance in bivariate analyses were retained in the final multivariable model to identify independent risk factors for vascular thrombosis. Continuous variables were reported as median with interquartile range, and categorical variables as number and percentage. Fisher's exact test or the chi-square test was applied when appropriate. Following construction of the final multivariable logistic regression model, weighted scores proportional to the beta regression coefficients were assigned to significant predictors.

Ethical Considerations

This study was approved by the Ethics Committee of Tabriz University of Medical Sciences (IR.TBZMED.REC.1403.021, approved on April 16, 2024). Due to the retrospective nature of the study, informed consent was waived, and ethical approval was considered sufficient in accordance with institutional and national research guidelines.

Results

Patients who developed thrombotic complications were significantly older and had a higher body mass index compared with those without thrombosis (both P<0.01). The prevalence of lung cancer, hypertension, diabetes mellitus, and underlying cardiovascular disease was also significantly greater in the thrombosis group (P<0.05 for all). In addition, patients with thrombosis experienced longer ICU stays, required mechanical ventilation more frequently, and had a markedly higher ICU mortality rate (all P≤0.01). Conversely, the use of anticoagulation therapy during ICU admission was significantly lower among patients who developed thrombosis (P=0.015) (table 1).

Table 1. Comparison of Baseline Clinical Characteristics and Outcomes Between ICU Patients with and Without Thrombotic Complications

Variable	Thrombosis (+) (n=109)	Thrombosis (–) (n=251)	P value
Age, years (mean ± SD)	66.4 ± 11.2	61.8 ± 12.5	0.002
Male sex, n (%)	72 (66.1)	142 (56.6)	0.098
Body mass index, kg/m² (mean ± SD)	27.9 ± 4.6	26.3 ± 4.2	0.004
Cancer type, n (%)	-	-	-
Lung	41 (37.6)	69 (27.5)	0.041
Gastrointestinal	34 (31.2)	88 (35.1)	0.48
Breast	18 (16.5)	52 (20.7)	0.37
Other malignancies	16 (14.7)	42 (16.7)	0.65
Comorbidities, n (%)			
Hypertension	63 (57.8)	112 (44.6)	0.019

Diabetes mellitus	48 (44.0)	79 (31.5)	0.021
Cardiovascular disease	39 (35.8)	61 (24.3)	0.028
ICU length of stay, days (median [IQR])	14 [9-21]	9 [6-15]	<0.001
Mechanical ventilation, n (%)	81 (74.3)	141 (56.2)	0.001
ICU mortality, n (%)	47 (43.1)	71 (28.3)	0.006
Anticoagulation therapy during ICU stay, n (%)	68 (62.4)	189 (75.3)	0.015

Patients who developed thrombotic events had a significantly higher burden of venous thromboembolism, with lower-extremity deep vein thrombosis being the most prevalent manifestation, followed by pulmonary embolism. Upper-extremity deep vein thrombosis occurred less frequently, while arterial, cerebral, and coronary thromboses were relatively rare. The overall distribution of thrombotic event types differed significantly between patients with and without thrombosis (P<0.001), indicating a predominance of venous over arterial thrombotic complications among affected ICU patients (table 2).

Table 2. Distribution and Types of Thrombotic Events Among ICU Patients with Thrombosis

Type of Thrombotic Event	Number (n)	Percentage (%)
Deep vein thrombosis – lower extremity	47	43.1
Deep vein thrombosis – upper extremity	21	19.3
Pulmonary embolism	26	23.9
Arterial thrombosis	9	8.3
Cerebral thrombosis (ischemic stroke)	4	3.7
Coronary thrombosis	2	1.8
Total	109	100

The prevalence of thrombotic complications among critically ill ICU patients with concurrent COVID-19 and active malignancy was significantly lower than the proportion of patients without thrombosis (28.3% vs. 71.7%, P<0.001). This marked difference indicates that although thrombotic events affected a substantial minority of patients, the majority of the cohort did not experience clinically confirmed thrombosis during ICU admission (figure 1).

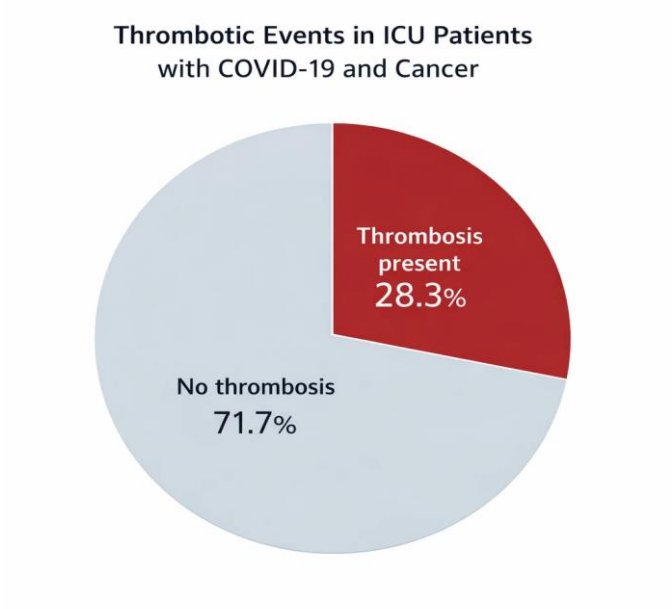

Figure 1. Prevalence of Thrombotic Events Among ICU Patients with COVID-19 and Active Malignancy

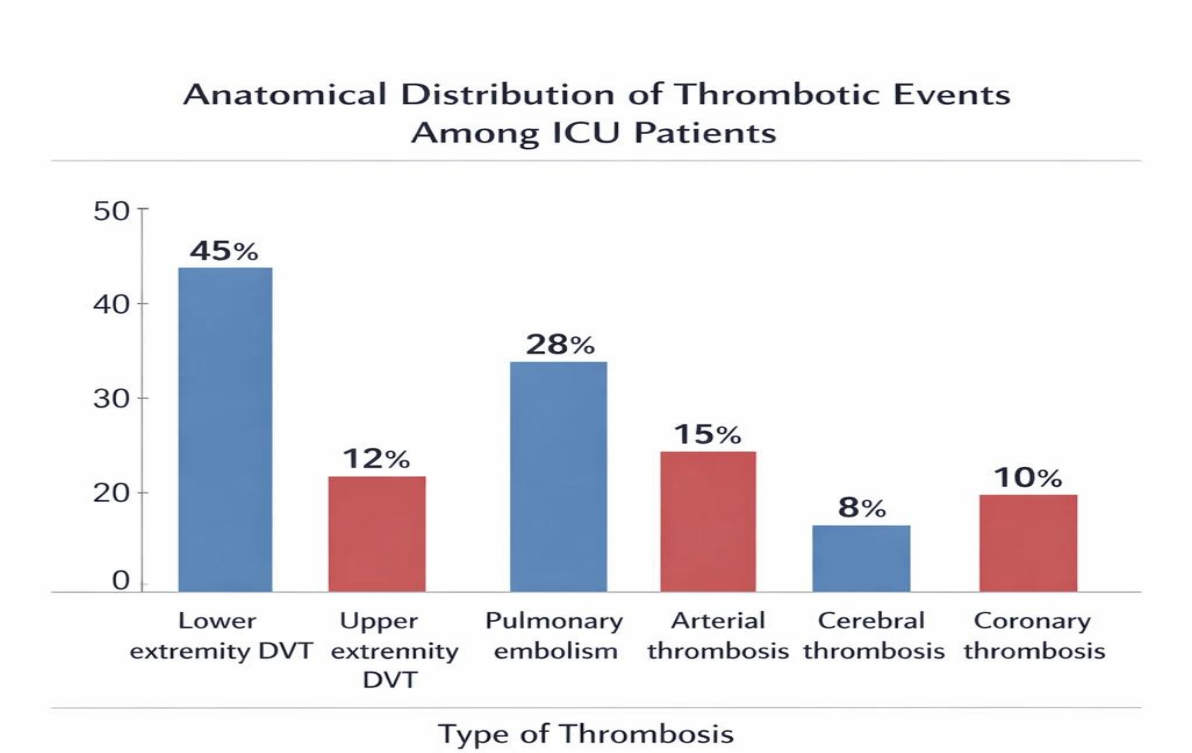

Figure 2. Distribution of Thrombotic Events by Anatomical Location Among ICU Patients with Thrombosis

Discussion

The present study demonstrates a substantial burden of thrombotic complications among critically ill ICU patients with concurrent COVID-19 infection and active malignancy, with more than one-quarter of the cohort developing at least one objectively confirmed thrombotic event. This finding reinforces the concept that the coexistence of COVID-19 related coagulopathy and cancer-associated thrombosis creates a uniquely high-risk clinical milieu, even though the majority of patients did not experience overt thrombosis during ICU admission. The observed prevalence of 28.3% aligns with prior reports describing markedly elevated thrombotic rates in critically ill COVID-19 populations, particularly when compounded by malignancy-related prothrombotic mechanisms.

Patients who developed thrombotic complications were significantly older and had higher body mass index values, suggesting that age-related endothelial dysfunction and obesity-associated chronic inflammation may amplify the already dysregulated coagulation cascade seen in COVID-19 and cancer. Aging is known to impair endothelial nitric oxide production and promote platelet hyperreactivity, while obesity contributes to increased levels of procoagulant factors such as fibrinogen and plasminogen activator inhibitor-1. In the context of severe SARS-CoV-2 infection, these baseline vulnerabilities likely predispose older and overweight patients to thrombus formation under conditions of systemic inflammation, hypoxia, and immobilization (14,15).

The higher prevalence of lung cancer observed among patients with thrombosis deserves particular attention. Lung malignancies are strongly associated with hypercoagulability due to tumor-derived procoagulant factors, endothelial injury from both tumor burden and anticancer therapies, and frequent coexistence of hypoxia and pulmonary inflammation. When combined with COVID-19 related endothelialitis and microvascular injury, these mechanisms may synergistically increase the risk of venous thromboembolism, particularly pulmonary embolism and lower-extremity deep vein thrombosis (16,17). By contrast, gastrointestinal and breast cancers did not show significant differences between groups, suggesting that cancer type-specific biology may modulate thrombotic risk in critically ill patients with COVID-19.

Comorbid conditions such as hypertension, diabetes mellitus, and underlying cardiovascular disease were significantly more common in the thrombosis group, further underscoring the role of chronic vascular dysfunction in thrombus formation. These conditions are characterized by baseline endothelial injury, impaired fibrinolysis, and heightened inflammatory signaling, all of which may potentiate COVID-19 induced coagulopathy. The convergence of these comorbidities with cancer-associated thrombosis likely explains the markedly elevated thrombotic risk observed in this subgroup (18,19).

Patients with thrombotic complications experienced significantly longer ICU stays and required mechanical ventilation more frequently, reflecting both greater disease severity and the bidirectional

relationship between thrombosis and critical illness. Prolonged immobilization, invasive procedures, central venous catheter use, and mechanical ventilation are well-established risk factors for venous thromboembolism. Conversely, the development of thrombosis itself may worsen respiratory failure, hemodynamic instability, and multiorgan dysfunction, thereby prolonging ICU stay and increasing the need for ventilator support (20,21). This vicious cycle likely contributes to the significantly higher ICU mortality observed among patients with thrombosis in the present cohort.

The finding of a markedly higher ICU mortality rate among patients with thrombotic complications highlights the prognostic significance of thrombosis in critically ill COVID-19 patients with cancer. Thrombotic events may directly lead to fatal outcomes through massive pulmonary embolism, ischemic stroke, or myocardial infarction, but they may also serve as markers of severe systemic inflammation and advanced disease. The association between thrombosis and mortality observed in this study is consistent with previous reports identifying venous thromboembolism as an independent predictor of death in ICU patients with COVID-19 (22,23).

An important and clinically relevant observation in this study is the significantly lower use of anticoagulation therapy during ICU admission among patients who developed thrombosis. This finding suggests that insufficient or delayed anticoagulation may have contributed to thrombus formation in a subset of patients. While anticoagulation strategies in critically ill COVID-19 patients have been the subject of ongoing debate, accumulating evidence supports at least prophylactic-dose anticoagulation in high-risk populations, including those with active malignancy. Variability in anticoagulation use may reflect concerns regarding bleeding risk, thrombocytopenia, or coagulopathy, yet the present findings emphasize the potential consequences of underutilization in a population already predisposed to thrombosis (24,25).

The pattern of thrombotic events observed in this study was dominated by venous thromboembolism, with lower-extremity deep vein thrombosis representing the most common manifestation, followed by pulmonary embolism. Upper-extremity deep vein thrombosis occurred less frequently, likely related to central venous catheter use, while arterial, cerebral, and coronary thromboses were relatively rare. This distribution supports the hypothesis that venous thrombosis remains the predominant thrombotic phenotype in critically ill COVID-19 patients with cancer, despite growing recognition of arterial thrombotic complications in severe SARS-CoV-2 infection (26,27).

The relative rarity of arterial and cerebral thromboses in the present cohort may be explained by several factors, including aggressive supportive care, early recognition of neurologic symptoms, and potential selection bias inherent to ICU populations with high competing risks of mortality. Additionally, venous thrombosis may be more readily detected through routine ultrasound surveillance and clinical suspicion, whereas arterial events may be underdiagnosed in deeply sedated or mechanically ventilated patients (28).

Taken together, these findings highlight the multifactorial nature of thrombosis in critically ill patients with COVID-19 and active malignancy. The interplay between patient-related factors (age, obesity, comorbidities), disease-related mechanisms (cancer biology and COVID-19 induced coagulopathy), and treatment-related factors (immobilization, mechanical ventilation, anticoagulation practices) appears to drive the observed thrombotic burden. Importantly, while the majority of patients did not develop thrombosis, those who did experienced significantly worse clinical outcomes, underscoring the need for vigilant risk stratification and preventive strategies.

This study has important clinical implications. Early identification of high-risk patients, particularly older individuals with lung cancer and cardiovascular comorbidities, may allow for more tailored thromboprophylaxis and closer monitoring. Furthermore, the association between reduced anticoagulation use and thrombotic events suggests that standardized anticoagulation protocols, balanced against bleeding risk, may improve outcomes in this vulnerable population. Future prospective studies are warranted to define optimal anticoagulation strategies and to clarify whether intensified prophylaxis can reduce thrombotic complications without increasing hemorrhagic events (29-31).

Conclusion

In conclusion, thrombotic complications remain a common and clinically significant problem among ICU patients with concurrent COVID-19 and active malignancy. Venous thromboembolism predominates, and its occurrence is associated with older age, higher BMI, specific cancer types, greater comorbidity burden, prolonged ICU stay, increased need for mechanical ventilation, and higher mortality. These findings emphasize the critical importance of integrated thrombosis prevention and management strategies in this high-risk population.

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Conflicts of interest

The authors declare that they have no competing interests.

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Authors' Contributions
All authors contributed to data analysis, drafting, and revising of the paper and agreed to be responsible for all the aspects of this work.

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