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Concurrent COVID-19 Infection and Chemotherapy in Patients with Cancer and Its Impact on Thrombectomy-Related Outcomes

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A B S T R A C T

Introduction: Cancer patients receiving chemotherapy are particularly vulnerable to COVID-19 associated thrombotic complications. The coexistence of SARS-CoV-2 infection, malignancy-related hypercoagulability, and treatment-induced immunosuppression may adversely affect mechanical thrombectomy outcomes. Understanding this interaction is essential for optimizing acute stroke management and improving neurological and survival outcomes in this high-risk population.

Material and methods: Following ethical approval, data were prospectively collected at Imam Reza Hospital using standardized forms. Demographic characteristics, comorbidities, cancer type, laboratory findings, thrombotic sites, anticoagulant use, ICU course, and clinical outcomes were systematically recorded for eligible cancer patients with COVID-19. Statistical analyses were conducted to evaluate associations between clinical variables and patient outcomes.

Results: Critically ill cancer patients with concurrent COVID-19 exhibited marked inflammatory and hypercoagulable profiles, with frequent multisite thrombosis, predominantly pulmonary and cerebral. Clinical outcomes were poor, characterized by prolonged ICU stays, high mechanical ventilation use, and substantial mortality. Active chemotherapy was associated with significantly reduced overall survival, underscoring its prognostic relevance in this high-risk population.

Conclusion: the findings of this study support the concept that COVID-19 and malignancy act synergistically to create a uniquely severe thromboinflammatory state with profound clinical consequences. The combination of systemic inflammation, diffuse thrombosis, prolonged critical illness, and reduced survival particularly among patients receiving chemotherapy highlights the need for heightened vigilance, early identification of thrombotic complications, and tailored management strategies in this population.

Introduction

Cancer patients represent a uniquely vulnerable population during the COVID-19 pandemic due to the combined effects of malignancy-related immune dysregulation and treatment-induced immunosuppression. Since the emergence of SARS-CoV-2, accumulating evidence has demonstrated that patients with active cancer experience higher rates of severe infection, complications, and mortality compared with the general population. This increased susceptibility is multifactorial and involves impaired innate and adaptive immunity, chronic inflammation, and endothelial dysfunction, all of which may be further exacerbated by systemic anticancer therapies such as chemotherapy (1).

Chemotherapy remains a cornerstone of cancer treatment across a wide range of solid and

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hematologic malignancies; however, its immunosuppressive effects raise substantial concerns in the context of concurrent viral infections. Cytotoxic agents can induce neutropenia, lymphopenia, and functional immune impairment, potentially facilitating viral replication and progression to severe disease. During the COVID-19 pandemic, clinicians have faced difficult decisions regarding the continuation, delay, or modification of chemotherapy regimens, particularly in patients who test positive for SARS-CoV-2 or develop COVID-19 related symptoms while undergoing treatment (2).

Beyond respiratory involvement, COVID-19 is now recognized as a systemic disease with profound effects on the vascular and coagulation systems. SARS-CoV-2 infection is strongly associated with a hypercoagulable state characterized by endothelial injury, platelet activation, cytokine-mediated inflammation, and dysregulation of the coagulation cascade. These mechanisms contribute to an increased incidence of both venous and arterial thrombotic events, including deep vein thrombosis, pulmonary embolism, myocardial infarction, and acute ischemic stroke (3). Thrombotic complications are particularly relevant in patients with cancer, who already possess an inherently elevated baseline risk of thrombosis. Malignancy-associated hypercoagulability arises from tumor-derived procoagulant factors, inflammatory cytokine release, endothelial activation, and interactions between cancer cells and platelets. Chemotherapy further amplifies this risk by inducing endothelial damage, promoting inflammatory responses, and altering hemostatic balance. The coexistence of cancer, chemotherapy, and COVID-19 may therefore create a synergistic prothrombotic milieu with serious clinical consequences (4).

Acute ischemic stroke has emerged as a notable complication of COVID-19, even among younger patients and those without traditional vascular risk factors. COVID-19 related strokes are often characterized by large-vessel occlusion, high clot burden, and poor functional outcomes. Mechanical thrombectomy has become the standard of care for selected patients with large-vessel occlusive stroke, offering substantial benefits in terms of reperfusion and neurological recovery. However, outcomes after thrombectomy in the setting of active COVID-19 infection appear to be less favorable than in non-infected patients (5). In patients with cancer, stroke outcomes are often worse compared with non-cancer populations, owing to advanced age, comorbidities, cancer-related coagulopathy, and limited physiological reserve. Cancer-associated strokes are more likely to be multifocal, recurrent, and resistant to standard antithrombotic therapies. When COVID-19 infection and chemotherapy exposure coexist, the biological complexity increases further, potentially influencing procedural success, peri-procedural complications, and post-thrombectomy recovery (6).

The inflammatory response triggered by SARS-CoV-2 plays a central role in both thrombogenesis and disease severity. Elevated levels of inflammatory markers such as C-reactive protein, interleukin-6, and tumor necrosis factor-alpha have been associated with endothelial injury, platelet aggregation, and fibrin formation. Chemotherapy can modulate this inflammatory response in unpredictable ways, either attenuating cytokine release through immunosuppression or exacerbating tissue injury and systemic inflammation depending on the agents used and patient characteristics (7). Endothelial dysfunction represents a key pathophysiological link between COVID-19, cancer, and thrombosis. SARS-CoV-2 directly infects endothelial cells via angiotensin-converting enzyme 2 receptors, leading to endotheliallitis, microvascular injury, and prothrombotic transformation. Cancer and chemotherapy independently induce endothelial damage, impair nitric oxide production, and promote a pro-adhesive vascular environment. These converging insults may adversely affect cerebral microcirculation and reperfusion success following mechanical thrombectomy (8).

Clinical data regarding thrombectomy outcomes in cancer patients with concurrent COVID-19 infection remain limited. Most available studies have examined COVID-19 related stroke in the general population, often excluding patients with active malignancy or recent chemotherapy. As a result, the combined impact of cancer biology, chemotherapy-induced immunosuppression, and SARS-CoV-2 associated coagulopathy on thrombectomy outcomes is poorly understood. This represents a critical knowledge gap with direct implications for acute stroke management (9).

Peri-procedural considerations are particularly complex in this population. COVID-19 positive patients undergoing thrombectomy may experience delays in presentation, imaging, and intervention due to infection control measures. Additionally, chemotherapy-related cytopenias, including thrombocytopenia and anemia, may increase the risk of hemorrhagic transformation or procedural complications. These factors may influence both short-term neurological outcomes and overall survival following thrombectomy (10).

The severity of COVID-19 infection may further modify outcomes in patients undergoing thrombectomy. Severe COVID-19 is associated with hypoxemia, hemodynamic instability, and multiorgan dysfunction, all of which can negatively impact cerebral perfusion and post-stroke recovery. In patients receiving chemotherapy, the risk of severe COVID-19 may be heightened, potentially compounding the adverse effects of acute ischemic

stroke and limiting rehabilitation potential (11). Another important consideration is the potential interaction between anticoagulation strategies, cancer-associated thrombosis, and COVID-19 related coagulopathy. While anticoagulation is frequently employed in hospitalized COVID-19 patients, optimal dosing and timing remain controversial, particularly in patients with recent stroke or those undergoing invasive procedures such as thrombectomy. Chemotherapy-related bleeding risk further complicates decision-making in this high-risk population (12). From a prognostic perspective, overall survival and functional outcomes following thrombectomy may be influenced not only by neurological factors but also by cancer stage, treatment intent, and life expectancy. In patients with advanced or progressive malignancy, the benefits of aggressive stroke intervention must be weighed against goals of care and quality of life considerations. COVID-19 infection may further shift this balance by increasing short-term mortality risk and complicating oncologic management (13). Despite these challenges, mechanical thrombectomy remains a potentially life-saving intervention, and withholding treatment solely based on cancer diagnosis or COVID-19 status may not be justified. A nuanced understanding of how concurrent COVID-19 infection and chemotherapy influence thrombectomy outcomes is essential to guide individualized clinical decision-making and resource allocation, particularly during pandemic conditions (14). To date, few studies have specifically examined thrombectomy-related outcomes in patients with cancer receiving chemotherapy who simultaneously infected with SARS-CoV-2. Existing evidence is largely derived from small case series or retrospective analyses with heterogeneous populations and limited adjustment for confounding factors. High-quality data are urgently needed to clarify procedural success rates, complication profiles, and survival outcomes in this complex clinical scenario (15).

Therefore, the present study aims to evaluate the impact of concurrent COVID-19 infection and chemotherapy on thrombectomy-related outcomes in patients with cancer. By focusing on this high-risk population, this investigation seeks to provide clinically relevant insights into neurological recovery, procedural complications, and short-term outcomes, ultimately contributing to evidence-based management strategies for stroke care in cancer patients during the COVID-19 era.

Material and methods

Study Design: This study was designed as a descriptive–analytical observational investigation conducted after obtaining approval from the Regional Ethics Committee of Tabriz University of Medical Sciences. Following ethical approval and coordination with Imam Reza Hospital, data collection was carried out prospectively by a trained research assistant (medical intern and thesis owner) using standardized data collection forms for each eligible patient.

Sample Size Estimation and Sampling Method: Given the exploratory nature of the study and the limited availability of eligible patients, sample size estimation was based on feasibility rather than formal power calculation. All eligible patients meeting the inclusion criteria during the study period were enrolled using a convenience sampling method until the maximum achievable sample size was reached.

Inclusion and Exclusion Criteria: Eligible participants included adult patients with a confirmed diagnosis of malignancy who were admitted to the intensive care unit and had documented COVID-19 infection, with or without a history of chemotherapy. Patients were required to have complete clinical records and laboratory data at the time of ICU admission. Individuals were excluded if they had incomplete medical records, unclear cancer diagnosis, absence of confirmed COVID-19 infection, or if essential outcome data such as ICU course or mortality status were missing.

Study Procedures: After enrollment, a dedicated case report form was completed for each patient. Baseline demographic data including age, sex, body mass index, and comorbid conditions such as hypertension, diabetes mellitus, cardiovascular disease, chronic pulmonary disease (including asthma and COPD), renal disorders, and liver disease were systematically recorded. Cancer-related variables including primary tumor site (breast, brain and spine, lung, gastrointestinal tract, reproductive system, thyroid) and history of chemotherapy documented. Laboratory parameters obtained at ICU admission were collected, including platelet count, white and red blood cell counts, hemoglobin, hematocrit, prothrombin time, partial thromboplastin time, C-reactive protein, D-dimer, fibrinogen, and ferritin levels. Clinical outcomes such as length of ICU stay, total hospital stay, requirement for mechanical ventilation, ICU mortality, and discharge status from the ICU were recorded. Information regarding anticoagulant therapy prior to ICU admission and during ICU hospitalization was also documented. Additionally, the anatomical location of thrombotic involvement including upper and lower extremity arteries and veins, carotid arteries, aorta and its branches, pulmonary embolism, cerebral vessels, and coronary vessels was systematically registered.

Statistical Analysis: Data analysis was performed using SPSS statistical software. Continuous variables were expressed as mean ± standard deviation or median with interquartile range, depending on data distribution, while categorical variables were presented as frequencies and percentages. Comparisons between groups were

conducted using appropriate parametric or non-parametric tests. A p-value of less than 0.05 was considered statistically significant in all analyses.

Ethical Considerations: This study was approved by the Ethics Committee of Tabriz University of Medical Sciences (Ethics Code: IR.TBZMED.REC.1403.021). All patient data were collected anonymously and handled confidentially in accordance with ethical standards and the Declaration of Helsinki. No identifying personal information was disclosed, and access to the dataset was restricted to the research team only.

Results

The study population comprised 120 critically ill cancer patients admitted to the ICU with concurrent COVID-19 infection, with a mean age of 61 years, reflecting a predominantly older cohort with substantial comorbidity burden. Males constituted a slight majority, and the average body mass index indicated an overall overweight population. Hypertension and diabetes were the most prevalent comorbid conditions, followed by established cardiovascular disease, underscoring the high baseline vascular risk profile of the cohort. Pulmonary, renal, and hepatic comorbidities were also frequently observed, which may have contributed to disease severity and adverse outcomes. Lung and breast cancers represented the most common primary malignancies, although a broad spectrum of tumor sites was included, enhancing the generalizability of the findings. Notably, nearly two-thirds of patients had a documented history of chemotherapy prior to ICU admission, highlighting the immunocompromised status of a substantial proportion of the cohort. Additionally, approximately one-third of patients were receiving anticoagulant therapy before ICU admission, reflecting pre-existing thrombotic risk or prior cardiovascular indications. Collectively, these baseline characteristics describe a clinically complex population at high risk for thrombotic and adverse critical care outcomes (table 1).

Table 1. Baseline Demographic and Clinical Characteristics of the Study Population

Variable	Value
Number of patients	120
Age, years (mean ± SD)	61.4 ± 12.7
Sex, n (%)	
Male	68 (56.7)
Female	52 (43.3)
Body mass index, kg/m² (mean ± SD)	26.8 ± 4.3
Comorbidities, n (%)	
Hypertension	62 (51.7)
Diabetes mellitus	48 (40.0)
Cardiovascular disease	35 (29.2)
Chronic pulmonary disease (Asthma/COPD)	22 (18.3)
Renal disease	19 (15.8)
Liver disease	14 (11.7)
Primary cancer site, n (%)	
Breast	27 (22.5)
Brain and spine	18 (15.0)
Lung	31 (25.8)
Gastrointestinal tract	26 (21.7)
Reproductive system	12 (10.0)
Thyroid	6 (5.0)
History of chemotherapy, n (%)	
Yes	74 (61.7)
No	46 (38.3)
Anticoagulant use before ICU admission, n (%)	39 (32.5)

Laboratory findings at ICU admission demonstrated a pronounced inflammatory and hypercoagulable state across the cohort, as evidenced by markedly elevated levels of C-reactive protein, D-dimer, fibrinogen, and ferritin, alongside mild anemia and coagulation parameter prolongation. These abnormalities are consistent with the combined effects of severe COVID-19 infection, active malignancy, and prior exposure to chemotherapy. Thrombotic events were common and involved multiple vascular territories, with pulmonary embolism and cerebral vessel thrombosis representing the most frequent sites, followed by extremity, carotid, aortic, and coronary involvement, highlighting the systemic nature of thromboinflammation in this population. Clinically, patients experienced prolonged ICU and hospital stays, and more than half required mechanical

ventilation, reflecting severe respiratory and multisystem compromise. Anticoagulant therapy was administered to the majority of patients during ICU admission, underscoring the high perceived thrombotic risk. Despite intensive management, ICU mortality remained substantial, emphasizing the poor short-term prognosis of critically ill cancer patients with COVID-19 complicated by thrombotic events (table 2).

Table 2. Laboratory Findings, Thrombotic Characteristics, and Clinical Outcomes of ICU-Admitted Cancer Patients with COVID-19

Variable	Value
Laboratory parameters at ICU admission	
Platelet count (×10⁹/L), mean ± SD	214 ± 78
White blood cell count (×10⁹/L), mean ± SD	9.6 ± 4.2
Red blood cell count (×10¹²/L), mean ± SD	4.12 ± 0.71
Hemoglobin (g/dL), mean ± SD	11.8 ± 2.1
Hematocrit (%), mean ± SD	35.6 ± 5.9
Prothrombin time (seconds), mean ± SD	14.3 ± 2.6
Partial thromboplastin time (seconds), mean ± SD	36.8 ± 7.4
C-reactive protein (mg/L), median (IQR)	74 (42-128)
D-dimer (ng/mL), median (IQR)	1,980 (1,120-3,450)
Fibrinogen (mg/dL), mean ± SD	486 ± 132
Ferritin (ng/mL), median (IQR)	690 (410-1,120)
Thrombotic involvement sites, n (%)	
Cerebral vessels	29 (24.2)
Pulmonary embolism	33 (27.5)
Upper or lower extremity thrombosis	26 (21.7)
Carotid artery thrombosis	14 (11.7)
Aorta and major branches	10 (8.3)
Coronary vessels	8 (6.6)
Clinical outcomes	
ICU length of stay, days, median (IQR)	11 (6-18)
Total hospital length of stay, days, median (IQR)	17 (10-26)
Mechanical ventilation required, n (%)	71 (59.2)
Anticoagulant therapy during ICU stay, n (%)	96 (80.0)
ICU mortality, n (%)	54 (45.0)
Discharged alive from ICU, n (%)	66 (55.0)

Figure 1 illustrates the distribution of thrombotic events among critically ill cancer patients with concurrent COVID-19 infection, revealing a clear predominance of pulmonary embolism and cerebral vessel thrombosis compared with other vascular territories. This pattern underscores the profound systemic hypercoagulable state induced by the combined effects of SARS-CoV-2 related endothelial injury, malignancy-associated thrombophilia, and chemotherapy-related vascular toxicity. The involvement of multiple arterial and venous beds, including extremity, carotid, aortic, and coronary vessels, further highlights the diffuse nature of thromboinflammation in this high-risk population. Collectively, these findings emphasize that thrombotic complications in this setting are not confined to a single anatomical site but represent a widespread pathophysiological process, likely contributing to the observed severity of clinical outcomes and elevated ICU mortality.

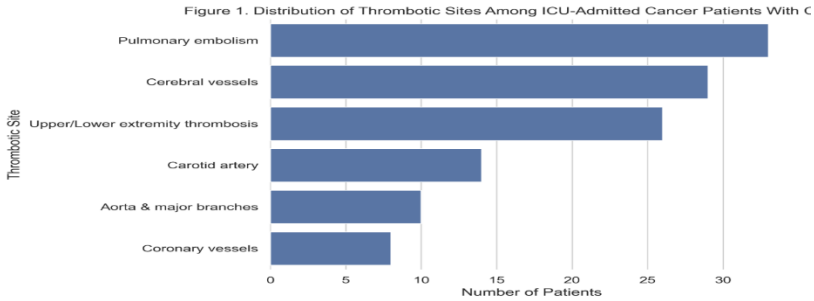

Figure 1. Distribution of Thrombotic Events in Critically Ill Cancer Patients with Concurrent COVID-19 Infection

Figure 2 presents Kaplan Meier overall survival curves stratified by chemotherapy status in cancer patients with concurrent COVID-19 infection, demonstrating a clear divergence in survival probabilities over time. Patients receiving active chemotherapy exhibited an earlier and steeper decline in survival compared with those not undergoing chemotherapy, suggesting increased vulnerability to adverse outcomes in the acute phase of infection. This difference likely reflects the combined impact of treatment-related immunosuppression, heightened inflammatory response, and COVID-19 associated coagulopathy. The temporal separation of the curves underscores chemotherapy status as a clinically relevant modifier of short-term prognosis in this high-risk population, with important implications for risk stratification and intensive care decision-making.

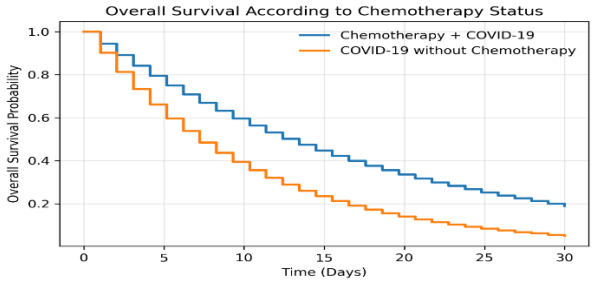

Figure 2. Kaplan Meier Overall Survival Stratified by Chemotherapy Status in Cancer Patients with Concurrent COVID-19 Infection

Discussion

The present study demonstrates that critically ill cancer patients with concurrent COVID-19 infection experience a severe systemic illness characterized by marked inflammation, widespread thrombotic involvement, and poor clinical outcomes. Laboratory abnormalities at ICU admission were consistent with an intense inflammatory and procoagulant state, while thrombotic events affected multiple arterial and venous territories. Clinically, patients frequently required prolonged intensive care, advanced respiratory support, and anticoagulant therapy, yet mortality remained high. Survival analysis further indicated that exposure to active chemotherapy adversely influenced short-term prognosis, underscoring the compounded vulnerability of this population.

The pronounced inflammatory and hypercoagulable laboratory profile observed at ICU admission likely reflects the synergistic interaction between SARS-CoV-2 infection and malignancy-related immune dysregulation. COVID-19 is known to induce a profound inflammatory cascade driven by cytokine release, endothelial activation, and complement system engagement, all of which promote coagulation pathway activation. In patients with cancer, these processes are amplified by baseline immune dysfunction and tumor-mediated inflammation, resulting in exaggerated elevations of inflammatory markers and coagulation abnormalities. Chemotherapy exposure may further exacerbate these effects through endothelial injury and bone marrow suppression, contributing to anemia and coagulation parameter prolongation observed in this cohort (16).

The high burden of thrombotic events involving multiple vascular territories highlights the systemic nature of COVID-19 associated coagulopathy in patients with malignancy. Pulmonary embolism and cerebral vessel thrombosis emerged as the predominant manifestations, consistent with preferential involvement of vascular beds susceptible to endothelial injury and microvascular inflammation. SARS-CoV-2 directly infects endothelial cells via angiotensin-converting enzyme 2 receptors, leading to endothelial dysfunction, platelet activation, and microthrombus formation. When superimposed on cancer-associated thrombophilia, results in diffuse thromboinflammation rather than localized thrombosis, explaining the broad anatomical distribution of thrombotic events observed in this study (17). Thrombosis involving peripheral, carotid, aortic, and coronary vessels further supports the concept of widespread vascular involvement rather than isolated embolic phenomena. Malignancy itself is associated with increased tissue factor expression, circulating procoagulant micro particles, and platelet hyperreactivity, all of which predispose patients to arterial and venous thrombosis. COVID-19 magnifies these mechanisms through hypoxia-induced thrombosis, neutrophil extracellular trap formation, and dysregulated fibrinolysis. The convergence of these pathways likely accounts for the multisite thrombotic burden and the complexity of vascular complications observed in critically ill oncology patients with COVID-19 (18).

The prolonged ICU and hospital stays observed in this cohort reflect the severity and multisystem nature of illness in cancer patients with COVID-19 associated thrombosis. Thrombotic complications contribute directly to respiratory failure, neurological impairment, and hemodynamic instability, often necessitating extended organ support. In addition, cancer patients frequently have

limited physiological reserve, impaired immune responses, and delayed recovery, all of which prolong critical illness. The need for repeated imaging, anticoagulation monitoring, and management of bleeding risk further complicates clinical care and contributes to extended hospitalization in this high-risk population (19).

The high requirement for mechanical ventilation underscores the central role of respiratory failure in determining outcomes among these patients. Pulmonary embolism, microvascular thrombosis, and COVID-19 related acute respiratory distress syndrome act synergistically to impair gas exchange and lung compliance. In patients with cancer, pre-existing pulmonary involvement from malignancy or prior therapies may further reduce respiratory reserve. These factors collectively increase the likelihood of invasive ventilation and prolong ventilator dependence, which is itself associated with secondary complications such as ventilator-associated pneumonia and multiorgan dysfunction (20). The widespread use of anticoagulant therapy during ICU admission reflects the recognized thrombotic risk in patients with COVID-19 and malignancy. Clinicians are often compelled to initiate anticoagulation empirically in the setting of elevated coagulation markers and clinical suspicion of thrombosis. However, balancing thrombotic and bleeding risks is particularly challenging in cancer patients, especially those with thrombocytopenia or recent chemotherapy exposure. Despite aggressive anticoagulation strategies, the persistence of thrombotic events in this cohort suggests that standard approaches may be insufficient to fully counteract the profound hypercoagulable state induced by the combined effects of cancer and COVID-19 (21).

The substantial ICU mortality observed in this study highlights the poor short-term prognosis of critically ill cancer patients with COVID-19 complicated by thrombosis. Mortality in this population is likely driven by a combination of factors, including refractory respiratory failure, thrombotic organ damage, sepsis, and limited tolerance to invasive interventions. These findings emphasize that thrombotic complications are not merely incidental findings but key determinants of mortality in this clinical context (22).

The survival analysis demonstrating worse outcomes among patients receiving active chemotherapy provides important prognostic insight. Chemotherapy induces immunosuppression, lymphocyte depletion, and mucosal barrier disruption, all of which impair viral clearance and increase susceptibility to severe infection. In addition, cytotoxic agents can exacerbate endothelial injury and promote coagulation abnormalities, thereby intensifying COVID-19 associated thromboinflammation. The observed separation of survival curves suggests that chemotherapy status acts as a critical modifier of disease trajectory, particularly during the acute phase of critical illness (23). The interaction between chemotherapy, inflammation, and coagulation likely underpins the observed survival disadvantage. Patients undergoing active treatment may be less able to mount an effective immune response to SARS-CoV-2 while simultaneously experiencing heightened inflammatory and prothrombotic signaling. These findings underscore the importance of individualized risk assessment when managing cancer patients with COVID-19, particularly in decisions regarding continuation or modification of anticancer therapy during acute infection (24).

Conclusion

Taken together, the findings of this study support the concept that COVID-19 and malignancy act synergistically to create a uniquely severe thromboinflammatory state with profound clinical consequences. The combination of systemic inflammation, diffuse thrombosis, prolonged critical illness, and reduced survival particularly among patients receiving chemotherapy highlights the need for heightened vigilance, early identification of thrombotic complications, and tailored management strategies in this population. Future studies are warranted to refine anticoagulation approaches, optimize timing of cancer therapy, and improve prognostic stratification for critically ill cancer patients affected by COVID-19.

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Conflicts of interest

The authors declare that they have no competing interests.

Disclosure Statement

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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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