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COVID-19-Associated Coagulopathy: Epidemiology, Pathophysiology, Biomarkers, and Antithrombotic Management

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ABSTRACT

The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) that causes coronavirus disease 2019 (COVID-19) is now known to be a systemic illness that affects more than only the respiratory system. New research has shown that the vascular and coagulation systems are widely involved, underscoring the important role that thrombotic complications play in the course of the disease and patient outcomes. A hypercoagulable state is often linked to COVID-19, which can result in both microvascular and macrovascular thrombosis. This illness, also known as COVID-19-associated coagulopathy (CAC), significantly increases mortality and organ failure. A significant percentage of COVID-19 patients have been found to have laboratory abnormalities suggestive of coagulation problems, such as higher D-dimer levels, prolonged prothrombin time, and thrombocytopenia. Among these markers, increased D-dimer levels are useful prognostic indicators since they are closely linked to mortality, thrombotic complications, and the severity of the disease. Coagulation disorders are strongly associated with poor clinical outcomes and are especially common in critically ill patients.The current knowledge on COVID-19-associated coagulopathy's epidemiology, clinical signs, pathophysiological mechanisms, diagnostic biomarkers, and treatment approaches is compiled in this narrative review. The significance of routine monitoring of coagulation parameters and the role of D-dimer in early detection and risk classification are highlighted. Low-molecular-weight heparin (LMWH) is a first-line treatment for venous thromboembolism (VTE) prevention in hospitalized patients, according to available data. The review also covers modern antithrombotic therapeutic strategies for managing and preventing thrombotic episodes in COVID-19.Improving patient prognosis requires rapid diagnosis and appropriate therapy of COVID-19-associated coagulopathy because to the significant correlation between coagulation dysfunction

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and poor clinical outcomes. A comprehensive understanding of this coagulation anomalies could lead to more successful therapeutic strategies and lower COVID-19-related morbidity and death.

Key Words: COVID-19, SARS-CoV-2, COVID-19-Associated Coagulopathy, Thrombosis, D-Dimer, Hypercoagulability, Venous Thromboembolism, Anticoagulation.

Introduction

The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) that caused coronavirus disease 2019 (COVID-19) first appeared in late 2019 and quickly developed into a worldwide public health emergency. A growing body of research has shown that COVID-19 is a complicated multisystem disease affecting the cardiovascular, neurological, renal, gastrointestinal, and hematological systems, despite being first identified as a respiratory ailment. Due to their high correlation with disease severity, thrombotic complications, and mortality, coagulation abnormalities have drawn a lot of attention among its extrapulmonary symptoms (Colling & Kanthi, 2020; Goswami et al., 2021).

The development of a hypercoagulable state, known as COVID-19-associated coagulopathy (CAC), is a characteristic of severe COVID-19. Elevated D-dimer levels, more fibrin breakdown products, a longer prothrombin time, and occasionally thrombocytopenia are the hallmarks of this illness. In contrast to traditional disseminated intravascular coagulation (DIC), CAC has distinct clinical and laboratory features and is more likely to cause thrombosis than hemorrhage (Garrahy et al., 2024; Connors & Levy, 2020).

The pathogenesis of CAC is multifactorial and involves a complex interplay among endothelial dysfunction, excessive inflammatory responses, platelet activation, complement system dysregulation, and activation of the coagulation cascade. The angiotensin-converting enzyme 2 (ACE2) receptor, which is found on endothelial cells and other bodily tissues, allows SARS-CoV-2 to enter host cells. Endothelial damage brought on by viral invasion and the ensuing inflammatory reaction can create a prothrombotic milieu with elevated thrombin production and decreased fibrinolysis. Microvascular and macrovascular thrombotic events are also influenced by immune-mediated thrombosis and cytokine-mediated inflammation (Colling & Kanthi, 2020; Goswami et al., 2021).

Clinical research has repeatedly shown that hospitalized COVID-19 patients, especially those in need of critical care, have a significant prevalence of venous and arterial thromboembolic consequences. In extreme situations, there has been an increased risk of myocardial infarction, deep vein thrombosis, ischemic stroke, and pulmonary embolism. Coagulation monitoring is a crucial part of patient care since elevated D-dimer levels have become one of the most accurate biomarkers for predicting disease progression, thrombotic risk, and death (Goswami et al., 2021; Garrahy et al., 2024).

Despite the substantial advancements in our understanding of the relationship between COVID-19 and thrombosis, numerous questions remain regarding the underlying mechanisms, the most effective methods for diagnosing the illness, and evidence-

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based antithrombotic therapies. Therefore, a comprehensive review of the literature is necessary to incorporate current knowledge and guide treatment decision-making. With an emphasis on the disease's effects on patient outcomes and possible future treatment options, this narrative review aims to give an overview of the epidemiology, pathophysiology, laboratory abnormalities, clinical manifestations, diagnostic biomarkers, and current management approaches of COVID-19-associated coagulopathy.

Covid 19 Impact on patients:

We should treat COVID-19 as a systemic illness, according to numerous observations.

For individuals with inflammatory autoimmune systemic disorders who are immunocompromised, COVID-19 infection presents a significant risk. (Mohamed et al.,2021) Patients with autoimmune systemic disorders (ASD) had a higher frequency of COVID-19 than the general population, according to observational cohorts. ASD patients are currently thought to be among the patient categories most susceptible to SARS-CoV-2 infection and morbidity, presumably as a result of continuing immunosuppressive therapies and deep immune-system changes. (Ferri et al.,2021) The systemic nature of COVID-19 is indicated by COVID-19 virology, the functions of the ACE-2 receptor in COVID-19 pathogenesis, immunological features of the disease, endothelial dysfunction and coagulopathy, and autopsy investigations. (Mohamed et al.,2021) As a result, everyone involved in the pandemic's management should start viewing COVID-19 infection as a systemic illness with significant pulmonary involvement rather than only a lung disease with potential involvement of other organs. A better understanding of the clinical, pathophysiological, and therapeutic features of COVID-19 infection would likely be possible with this shift in viewpoint. The systemic and multi-organ localization of COVID-19 infection has been shown in an increasing number of reports. These results enable us to speculate that COVID-19 infection a systemic illness independent of the well-known, potentially fatal pulmonary consequences. We will likely have a better understanding of COVID-19 infection if we start thinking of it as a systemic illness. (Bongiovanni M &Marra.,2021) Numerous organ-specific and systemic phenotypes have been brought about by the Covid-19 epidemic; the mechanism or mechanisms driving the development of acquired thrombophilia and systemic coagulopathy. (Becker, 2020) Infectious, immunological, autoimmune, neurological, and hemodynamic environments are diverse and extremely dynamic. (Becker et al., 2025)

In COVID-19 infection, coagulation abnormalities and thrombosis are significant pathophysiological alterations. An increased D-dimer, a prolonged prothrombin time, a low platelet count, and other laboratory abnormalities are indicative of coagulation problems in up to 95% of COVID-19 patients (Teimury et al., 2022) Because SARS-CoV-2 infection prolongs a state of hypercoagulability, it is linked to frequent thrombotic events at the micro and macro-vascular levels. One important factor in the development of organ damage caused by SARS-CoV-2 is the so-called "COVID-19 associated coagulopathy" (CAC). (Lorinet al., 2021) In general, problems in blood coagulation might be regarded as a prognostic factor in COVID-19 patients. (Teimury et al., 2022) Therefore, coagulation dysfunction should be carefully addressed in

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clinical practice as it is a potential risk factor for unfavorable outcomes in COVID-19. Patients with COVID-19, particularly those with the more severe types, frequently experience coagulation malfunction, which reduces their chances of survival. Lazzaroni et al., 2021) It has been established that these patients have a high risk of thrombotic problems. (The Lancet Haematology, 2020)

SARS-CoV-2, the virus that causes COVID-19, affects the respiratory system and causes serious coagulation abnormalities and thrombotic problems, especially in severe instances. These hematological alterations and "their association with disease severity and outcomes" were assessed in this investigation (Teimury et al., 2022) The epidemiology, clinical characteristics, established and hypothesized pathophysiology-based models, and therapeutic recommendations pertaining to COVID-19 coagulopathy are all summarized in this narrative review. (Marchan dot et al., 2020) The vascular wall endothelium and the coagulation, fibrinolytic, anticoagulation, and complement systems are delicately balanced to provide proper hemostasis. (Sayyadi et al., 2023) Based on a number of investigations, we discovered that COVID-19 patients frequently experience coagulation issues such venous thromboembolism and pulmonary embolism. Early diagnosis of coagulation issues can be aided by measuring d-dimer levels upon admission and throughout hospital stays. monitoring of d-dimer and C-reactive protein. Those elements and actions may be useful in lowering coagulation problems and enhancing patient care. In order to treat patients promptly, it can be beneficial to regularly check coagulation markers and thrombotic problems. Reviewing the coagulation issues in COVID-19 patients is therefore beneficial. (Teimury et al., 2022)

Overview of Hemostasis

Hemostasis, which means to stop bleeding, is derived from the Greek word heme, which means blood, and stasis, which means to stop. (Palta et al., 2014) The coagulation system and platelets are both necessary for hemostasis. A hemostatic plug made of platelets and fibrin forms at the locations of vessel damage, reducing bleeding. (Mack man et al., 2007) In a statement, the International Society on Thrombosis and Hemostasis (ISTH) recommended low molecular weight heparin (LMWH) as a preventative measure. However, because little is known about how COVID-19 impacts hemostasis, the best prophylactic approach is still debatable. (Peyvandi et al.,2020) According to the conventional theory of blood coagulation control, the intrinsic pathway is necessary for amplification whereas the extrinsic pathway initiates the phase. (Mack man et al., 2007) In 1955, the idea of intrinsic and extrinsic coagulation pathways was presented, and in 1957, it was mentioned in the literature. Blood coagulation is triggered by both extrinsic (tissue factor) and intrinsic (contact activation) routes. (Park & Park, 2024)

Extrinsic pathway

The transmembrane receptors tissue factor (TF) and plasma factor VII/VIIA (FVII/FVIIA) make up the extrinsic route. (Mack man et al., 2007) It is regarded as the initial stage of plasma-mediated hemostasis. TF, which is expressed in the subendothelial tissue, activates it. Normal vascular endothelium minimizes contact between TF and plasma procoagulants under physiological settings, but vascular injury exposes TF, which interacts with calcium and factor VIIA to encourage the

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conversion of factor X to XA (Palta et al., 2014) Using normal plasma protein concentrations, the extrinsic pathway's predicted clotting time was roughly 8.6 seconds. (Zhu, 2007; Bilal et al., 2021) Thrombosis requires the extrinsic pathway of blood coagulation. On the other hand, thrombosis results from TF expression in the vasculature under pathologic circumstances. A deeper comprehension of the function of TF and FVIIA in hemostasis and thrombosis is necessary for the development of safe and efficient hemostatic agents and antithrombotic medications. (Mack man et al., 2007)

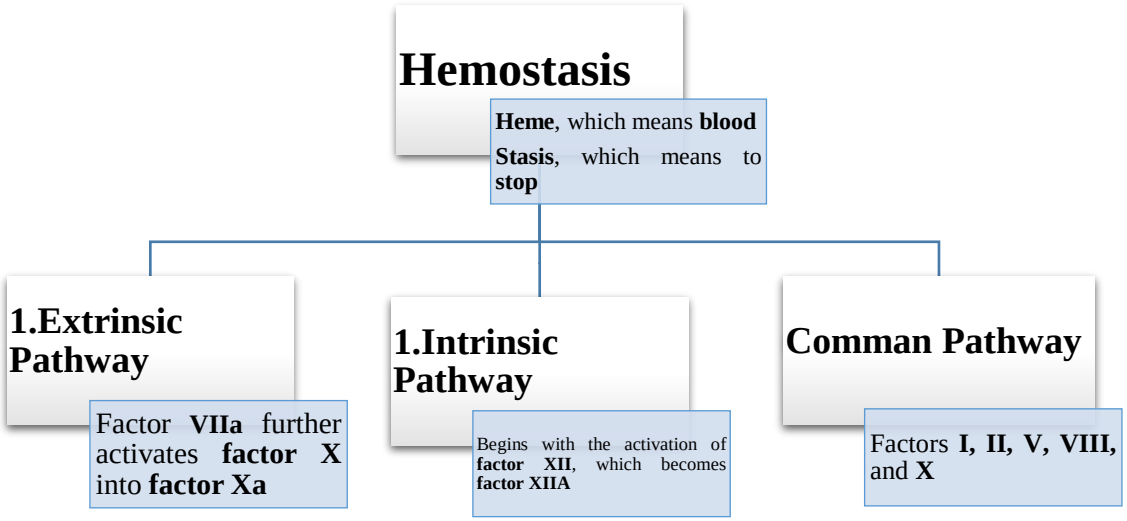
Intrinsic pathway

The intrinsic pathway's estimated clotting time was 44 seconds. (Zhu, 2007). Plasma FXI, FIX, and FVIII make up the intrinsic route. (Mack man et al., 2007) It is a parallel route by which factor XII activates thrombin. Factor XII, HMW kininogen, *Prekallikrein*, and factor XI are the first to activate factor XI. Factor IX is further activated by activated factor XI. Factor IX then forms a tense complex on a phospholipid surface with its cofactor, factor VIII, to activate factor X. (Palta et al., 2014) The hemorrhagic morphologies of patients lacking certain components of this pathway, however, point to more intricate connections. Hemophilia B and hemophilia A are caused by deficiencies in factor IX or its cofactor, factor VIII. (Gailani & Renné, 2007)

Comman Pathway

The prothrombinase complex, which transforms prothrombin into thrombin, is formed by activated factor X, its cofactor (factor V), tissue phospholipids, platelet phospholipids, and calcium. In addition to activating factor XIII, which covalently crosslinks fibrin polymers included in the platelet plug, this thrombin further cleaves circulating fibrinogen to insoluble fibrin. As a result, a fibrin network is formed, stabilizing the clot and creating a permanent secondary hemostatic plug. (Palta et al., 2014)

Balance between clotting and fibrinolysis



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Blood clotting is an international phenomenon that is known to provide protection against external cellular and tissue damage. The entrance of SARS-CoV-2, which causes COVID-19 infection, is mediated via the interaction of the spike glycoprotein with the ACE2 receptor on the surface of the host cell. The production of enigmatic blood clots in the numerous tissues and organs of COVID-19 patients is one such riddle that requires urgent research. New clinical findings of SARS-CoV-2 pathogenesis are emerging daily. The spike protein of SARS-CoV-2 may bind to the ACE2 receptor expressed in blood vessel endothelial cells, causing vasoconstriction and activating the intrinsic pathway of coagulation, which ultimately leads to the formation of blood clots. This could be the cause of the mysterious clots observed in COVID-19 patients.

By converting Ag II to Ag (1–7), ACE2 protects endothelial cell function and prevents early atherosclerosis, which can be brought on by a blood vessel clog. (Biswas et al., 2021)

An urokinase-driven route in SARS-CoV-1 infection-stimulated fibrinolysis is suggested by animal models. Prolong prothrombin times (PT) were observed in COVID-19 patients in comparison to healthy controls, despite more noticeable changes in fibrinogen and D-dimers. This suggests that fibrinogen monitoring may be useful as a prognostic indicator for COVID-19 patients who are hospitalized. Thrombocytopenia, prolonged PT, and elevated D-dimers all point to subsequent fibrinolysis after coagulation activation in COVID-19 infection. It is clear that alterations in the hemostatic balance in COVID-19 patients are strongly disturbed and contribute to a high prothrombotic status, justifying the use of anticoagulant therapy. (Devreese, 2021) There are still topics for debate and conjecture regarding the modifications to fibrinolysis and blood coagulation mechanisms. (Coccheri,2020).

Pathophysiology

The primary goal of the research is to comprehend the pathophysiology of COVID-19 in relation to the endothelium's involvement so that the management protocol can include more pertinent treatments. (Otifi & Adiga, 2022) One potentially fatal consequence of SARS-CoV-2 infection is COVID-19-associated coagulopathy (CAC). Our present understanding of CAC can be divided into three primary pathogenic mechanisms: hypercoagulability, a hyper-inflammatory immune response, and vascular endothelial cell failure. (Conway et al., 2022) (Otifi & Adiga, 2022) . The pathophysiology of SARS CoV-2 infection is highlighted in the current study on endothelial dysfunction through the effects of direct viral-induced endothelial injury, uncontrolled immune and inflammatory response, imbalanced coagulation homeostasis, and their interactions, which create a vicious cycle that exacerbates the disease process. (Otifi & Adiga, 2022) It has been discovered that cytokine storm is a clinical predictor of morbidity and mortality in a number of viral diseases, including influenza. (Prasad et al., 2021) In COVID-19, coagulopathy and thrombotic consequences are common. However, the features of COVID-19-associated coagulopathy (CAC) differ from those of bacterial sepsis-induced coagulopathy (SIC) and disseminated intravascular coagulation (DIC), with CAC typically exhibiting elevated levels of fibrinogen and D-dimer but initially mild abnormalities in platelet count and prothrombin time. Compared to SIC/DIC, venous thromboembolism and

Parameters	Reference Range	DIC	Coagulopathy in covid -19

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arterial thrombosis are more common in CAC. (Iba et al., 2020) Coronavirus disease 2019 (COVID-19) is caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). According to recent evidence, pulmonary immuno-thrombosis plays a crucial role in the pathophysiology of severe COVID-19. (O'Donnell et al., 2021) In addition to elevated cytokines and activation of platelets, endothelium, and complement, COVID-19 causes a unique coagulopathy that is increasingly common as the severity of the illness worsens. This proinflammatory environment may lead to immune thrombosis, a dysregulated host defense mechanism that causes an excess of immunologically induced thrombi to develop, mostly affecting the microvasculature. (Loo et al., 2021) In COVID-19, pulmonary thrombi exhibited varying phases of organization and were hetero synchronous in character. This observation implies that they are not embolic in origin, but rather have evolved in situ. It's interesting to note that megakaryocytes were linked to microvascular thrombi in alveolar capillaries and that platelet formation was still occurring. These findings imply that platelet activation plays a significant role in the development of COVID-19 pulmonary microangiopathy since the microthrombi are rich in platelets. (O'Donnell et al., 2021)

Laboratory Findings and Biomarkers

The traditional laboratory results include elevated PT, APTT, and D-dimer levels, as well as decreased platelet counts and fibrinogen levels. The PT and APTT times are either slightly raised or stay normal. When infected with COVID-19, the level of fibrinogen is increased.

D-dimer deterioration is a sign of serious illness and a dismal prognosis. (Sarkar et al., 2021)

Prolonged prothrombin time, shortened or prolonged activated partial thromboplastin time (APTT), or aberrant thrombin time are common anomalies of regular coagulation testing. These alterations may be connected to fibrinogen, an acute-phase protein, and D-dimers, a marker of cross-linked fibrin and clot turnover. Pulmonary vascular thrombi appear to be predicted by raised D-dimers, elevated fibrinogen, and normal platelet counts (Chowdary, 2022). Prominent elevation of fibrinogen and D-dimer. The degree of D-dimer elevation positively correlates with mortality in COVID-19 patients, COVID-19 patients often have mild thrombocytopenia (Wool & Miller, 2021) The majority of patients had normal platelet counts, elevated D-dimer, PT, and APTT values in the upper reference interval. Increased D-dimer concentration, mildly prolonged prothrombin time (PT), mild thrombocytopenia, and decreased fibrinogen in late disease seemed to be associated with poor outcomes. (Lee et al., 2021) An increase in D-dimer in peripheral blood indicates that fibrin polymers are breaking down. (Becker, 2020) The majority of patients had normal platelet counts, elevated D-dimer, PT, and APTT values in the upper reference interval. Increased D-dimer concentration, mildly prolonged prothrombin time (PT), mild thrombocytopenia, and decreased fibrinogen in late-stage disease seemed to be associated with poor outcomes. (Lee et al., 2021)

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D-Dimer	<500 (ng mL-1)	Increased	Increased
PT	9.4-12.5 (s)	Frequently increased	Normal or increased
Appt	25.4-36.9 (s)	Frequently increased	Normal or increased
Platelet count	130-400 (GL-1)	Frequently low	Normal, thrombocytopenia is rare
Fibrinogen	200-393 (mgdL-1)	Decreased	Increased initially, may decreased in later stage
Fibrin degradation product (FDPs)	<10 mcg/mL	Decreased	Increased
Consumption coagulopathy		Seen	Rare
VWF		Increased	Increased
Antithrombin (AT)		Decreased	Increased
Outcome		Bleeding	Thrombosis

Czempik, P. F. (2019). Basilar artery thrombosis in a 20-year-old pregnant patient–difficulties in e<stablishing the aetiology. *Anaesthesiology Intensive Therapy*, *51*(4), 330-332.

Risk Factors and Disease Severity

Globally, extensive COVID-19 cohort studies have demonstrated that male sex and advanced age are significant non-modifiable risk variables that lead to unfavorable outcomes. (Navaratnam et al., 2021) . Multivariate analysis revealed that COVID-19 severity was correlated with age > 60, male, obesity, comorbidity, abnormal D-dimer upon hospital admission, and abnormal peak hospitalization PT, APTT, FDP, and D-dimer. Significantly greater mortality was linked to extreme coagulation parameter abnormalities (PT > 16s, FDP > 50 ug/ml, and D-dimer > 5 ug/ml). (Xu et al., 2021) Moreover, a higher load of comorbidities and greater deprivation in these groups were only partially responsible for this elevated risk.7 Other research conducted in the UK supports the link between Black and Asian ethnicities and higher COVID-19 in-hospital death rates. In contrast, a study conducted in Louisiana, USA, revealed that after controlling for variables such as age, sex, Charlson Comorbidity Index score, living in a low-income neighborhood, obesity, and indicators for baseline vital signs and laboratory measurements, hospitalized There was no difference in the COVID-19 mortality risk between Black and White patients. (Navaratnam et al., 2021) Abnormal coagulation parameters were observed in patients with COVID-19, both at hospital admission (INR 2.3%, PT 7.9%, APTT 15.4%, TT 0.9%, FDP 2.3%, D-dimer 19.7%) and peak hospitalization (INR 4.8%, PT 13.4%, APTT 25.6%, TT 2.7%, FDP 10.4%,

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D-dimer 31.5%). Compared with non-severe patients with COVID-19, severe patients had a slightly higher INR, PT, APTT, whereas remarkably higher FDP and D-dimer ($p < 0.05$) (Xu et al., 2021) Additionally, a higher mortality risk has been linked to pre-existing comorbidities such cancer, severe obesity, diabetes, liver disease, cardiovascular disease, hypertension, and respiratory disorders. (Navaratnam et al., 2021

COVID-19 can alter coagulation parameters; the degree of these changes and their relationship to illness severity and death vary widely. Activate partial thromboplastin time (APTT), prothrombin time (PT), fibrinogen, fibrin split products (FSP), D-dimer, and platelet count are some of these markers. Additionally, patients with severe COVID-19 may develop macrovascular thrombosis and disseminated intravascular coagulation (DIC), which can result in significant morbidity and mortality. (Hadid et al., 2021)

Venous thromboembolism (VTE) is known to occur frequently in critically ill patients—up to 10%. However, both ICU and non-ICU COVID-19 patients have significantly greater incidence of VTE. (Devreese, 2021) While changes were found on other coagulation laboratory tests, APTT, PT, and INR did not change during the course of the trial (Ali et al., 2021). Both groups had higher amounts of fibrinogen. Lastly, patients in group SOFA > 10 had lower plasminogen levels and Protein C than patients in group SOFA <10, which may indicate less fibrinolysis activity during a state of hypercoagulability (Jawad et al., 2024). D-dimer plasma levels were higher than the normal reference range in both groups, and free protein S plasma levels were low in both groups at baseline and increased over time. (Correa et al., 2020) According to a recent meta-analysis and systematic review, different studies had different rates of VTE, including pulmonary embolism (PE) and deep vein thrombosis (DVT).9 The incidence of VTE was 14.1% overall, with a higher frequency of 22.7% among ICU patients. PE was found in 3.5% of non-ICU patients and 13.7% of ICU patients. It's interesting to note that neither age nor comorbidities varied across COVID-19 participants with and without VTE. However, the risk of developing VTE was 1.5 times higher in men. (Devreese, 2021; Basharat et al., 2024) While PT, D-D, and FIB levels were higher in severe cases than in mild instances, PLT levels were lower in severe cases ($P < 0.05$). There was no statistically significant difference in the two groups' APTT levels ($P > 0.05$). Compared to non-ICU patients, the PT of ICU patients was substantially longer ($P < 0.05$). PT and D-D were higher in non-survivors than in survivors, whereas PLT was lower ($P < 0.05$). APTT did not significantly differ between survivors and non-survivors ($P > 0.05$). (Zhang et al., 2020)

Diagnostic Approaches

In COVID-19 patients, elevated D-dimer levels were revealed to be an important laboratory marker in the risk assessment of thrombosis. (Devreese, 2021) Regression models revealed a direct correlation between the variation in PT and fibrinogen and C-reactive protein levels. (Di Minno et al., 2020) COVID-19 can impact all coagulation parameters. (Hadid et al., 2021) Activated partial thromboplastin time (APTT), prothrombin time (PT), fibrinogen, fibrin split products (FSP), D-dimer, and platelet count are some of these markers. (Hadid et al., 2021) Severe patients had

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reduced PLT counts (SMD: -0.74; 95%CI: -1.01, -0.47) and higher PT (SMD: 0.41; 95%CI: 0.1, 0.60), D-Dimer (SMD: 0.67; 95%CI: 0.52, 0.82), and fibrinogen values (SMD: 1.84; 95%CI: 1.21, 2.47. (Di Minno et al., 2020) Additionally, patients with severe COVID-19 may develop macrovascular thrombosis and disseminated intravascular coagulation (DIC), which can result in significant morbidity and mortality. (Hadid et al., 2021) It is evident that changes in the hemostatic balance in COVID-19 patients are severely disrupted and lead to a high prothrombotic status, which justifies the prescription of anticoagulant medication. In addition to hemostatic abnormalities, high rates of VTE have been noted in COVID-19 patients (Devreese, 2021; Noor et al., 2024)

Imaging frequently reveals coagulopathy symptoms in COVID-19 hospitalized patients. All imaging tests that could detect thrombosis, embolism, or end-organ ischemia or infarction (Doppler ultrasound [US]; contrast-enhanced CT; and MRI of the head, neck, chest, abdomen, and upper and lower extremities) may be appropriate in COVID-19 patients with suspected systemic coagulopathy if there are no imaging contraindications. Excluded were radiography, nuclear medicine scans, non-Doppler US, and unenhanced CT (O'Shea et al., 2021; Ahmad et al., 2023; Bilal, 2021)

Patients with severe COVID-19 had prolonged baseline PT. The prognosis was likewise projected to deteriorate with extended baseline PT. A worse prognosis was linked to an increase from the baseline PT. Consequently, PT can be utilized in COVID-19 patients as a risk stratification and prognosis indicator (Tekle et al., 2022; Bilal et al., 2021)

Management and Treatment Strategies

The best methods for preventing thrombotic events and the selection of antithrombotic medications for the treatment of new or pre-existing thrombotic events in COVID-19 patients are crucial. (Hashemi et al., 2020) For hospitalized COVID-19 patients, LMWH is advised as first-line treatment to prevent VTE. (Hardy et al., 2020) The most widely used parenteral anticoagulant medications for the prevention and treatment of thrombotic disorders are unfractionated heparin and low-molecular-weight heparin (LMWH). (Hashemi et al., 2020) For COVID-19 patients who are not severely ill, routine use of therapeutic doses of anticoagulants over the recommended preventive dose is not advised. (Duo et al., 2022) Compared to preventative dosage, therapeutic anticoagulation decreased the risk of thrombosis but increased hemorrhage. (Zhou et al., 2026) Prophylactic anticoagulation in hospitalized patients without clinical contraindications, at the very least (Mondal et al., 2020) Due to the substantial inter- and intra-individual variability, unfractionated heparin may need to be adjusted. (Hardy et al., 2020) The most popular parenteral anticoagulant medications for the prevention and treatment of thrombotic disorders are unfractionated heparin and low-molecular-weight heparin (LMWH) (Hashemi et al., 2020) Escalated-dose anticoagulation, particularly therapeutic-dose heparin, has been investigated for thromboprophylaxis in hospitalized COVID-19 patients due to its non-anticoagulant properties. (Rahi et al., 2023) Due to potential drug interactions with concurrent antiviral (particularly anti-HIV protease inhibitors like ritonavir) and antibacterial (like azithromycin) treatment, low molecular weight heparins (LMWH) or unfractionated heparin (UFH) should be chosen over direct oral anticoagulants

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(DOACs). Treatment that disrupt the P-GP and/or CYP3A4 pathways may lessen the antithrombotic benefit of DOAC usage or raise the risk of bleeding.

The management of COVID-19 patients involves four key components:

(i) early diagnosis and follow-up of DIC using the ISTH score (platelet count, PT, fibrinogen, D-dimer, antithrombin, and protein C activity monitoring), which can determine prognosis and direct more appropriate critical care support;

(ii) identifying high-risk individuals, whether they are ambulatory or hospitalized;

(iii) (LMWH is the first-line medication and the thromboprophylaxis regimen should be optimized;

(iv) COVID-19 patients may benefit from LMWH's anti-inflammatory qualities;

(v) it may be necessary to incorporate other antithrombotic treatments, such as antithrombin and recombinant thrombomodulin, which may also be useful in this intricate "immune thrombosis" process.

Complications and Prognosis

Due to increased inflammation, hypoxia, immobilization, and diffuse intravascular coagulation (DIC), COVID-19 may increase the risk of both venous and arterial thromboembolic illness. Surprisingly, there aren't many reports of thrombotic problems (Bilal et al., 2021). Making decisions about the level of thromboprophylaxis, particularly for patients admitted to the intensive care unit (ICU) who are at the highest risk of thrombosis, requires precise understanding of the incidence of thrombotic events in COVID-19 patients. (Klok et al., 2020) Coagulopathy that results in venous thrombosis may worsen the clinical course of COVID-19 patients. (Hajra et al., 2020) An increasing concern is the possibility of venous thromboembolism (VTE) in hospitalized COVID-19 patients. (Terpos et al., 2020) According to reports, the COVID-19 illness spectrum's multi-system involvement is significantly influenced by the thrombotic problems brought on by the viral infection. In the context of COVID-19 infection, endothelial damage and proinflammatory cytokines initiate a coagulation cascade that results in thromboembolic events. It may be difficult to diagnose VTE in COVID-19 individuals. (Hajra et al., 2020) Serious side effects that some COVID-19 patients experience included coagulopathy and thrombotic events. It is unknown, though, if coagulopathy and the severity of COVID-19 sickness are related. Mild thrombocytopenia, a small lengthening of the prothrombin time, high DD, raised fibrinogen levels, factor VIII, and Von Willebrand factor are the characteristics of the coagulopathy linked to COVID-19. DD, a breakdown product of cross-linked fibrin, is correlated with the severity of the disease and predicts mortality, the need for ventilator support, and the risk of thrombosis. There have also been reports of increased mortality with coagulopathy and advanced age. Similar to this, deaths from new coronavirus pneumonia have been linked to aberrant coagulation, increased DD, and fibrin degradation product. (Al Nafea et al., 2023) Long COVID appears to be caused by a variety of pathophysiological processes, including viral factors (SARS CoV-2 persistence, reactivation, and bacteriophagic action); host factors (immune dysregulation, autoimmunity, metabolic and endocrine dysregulation, and chronic inflammation); and downstream effects (tissue damage from the initial infection, tissue hypoxia, host dysbiosis, and autonomic nervous system dysfunction). The long-

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term persistence of the condition, which is marked by endothelial thrombosis, endothelial inflammation, hyperactivated platelets, and micro clots of fibrin, is the result of these mechanisms. Every organ system is impacted by these blood vessel and coagulation problems, which serve as a common pathway for the many Long COVID symptoms. Turner et al., 2023) Activated partial thromboplastin time (APTT) and fibrinogen levels did not differ substantially between deceased COVID-19 patients and survivors, however the mean platelet count was significantly lower (20.58). Patients who survived had significantly decreased pooled mean differences for D-Dimer, International Normalized Ratio (INR), and prothrombin time (PT) (-2.45, -0.10, and -0.84, respectively). These results imply that platelet count, D-Dimer, INR, and PT could be useful markers of COVID-19 patients' mortality. (Abdollahi et al., 2024)

Current Challenges and Future Directions

In coronavirus disease 2019 (COVID-19), coagulopathy is a complex process that affects the majority of the hemostasis system rather than just one hemostatic component. (Sayyadi et al., 2023) While there are still a lot of unsolved questions, thoroughly carried out clinical trials have not yet determined the best course of action. (Becker, 2020) Different receptor binding, cytokine storm, and direct viral endothelium injury are some of the explanations that try to explain the pro-coagulant condition observed in COVID-19 patients. (Sayyadi et al., 2023) Prophylactic anticoagulation should be administered to all hospitalized COVID-19 patients. Research assessing COVID patients' therapeutic anticoagulation is required. (Salabei et al., 2021). There are some subjects for further research and consideration. Since COVID-19 patients are more likely to experience thrombotic problems, there are now several consensus guidelines and suggestions that can help with clinical decision-making. Nevertheless, the absence of data from randomized controlled clinical trials limits these recommendations. (Flaczyk et al., 2020)

Conclusion

Thrombosis and coagulation abnormalities are key pathophysiological changes caused by COVID-19 infection. The so-called "COVID-19 associated coagulopathy" (CAC) is a significant contributor to the development of organ damage induced by SARS-CoV-2. Blood coagulation issues may generally be considered a prognostic factor in COVID-19 patients. As a consequence, coagulation dysfunction should be addressed carefully in clinical practice because it is a possible risk factor for poor outcomes in COVID-19 patients. Persons with COVID-19, especially those with the more severe forms, typically encounter coagulation problems, which diminishes their chances of survival. COVID-19 may raise the risk of venous and arterial thromboembolic disease. Coagulopathy, which causes venous thrombosis, may exacerbate the clinical course of COVID-19 patients.

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