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COVID AND CHANGES IN BLOOD CLOTTING

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Abstract. This article explores the relationship between COVID-19 infection and alterations in blood clotting mechanisms. Since the onset of the pandemic, clinical evidence has demonstrated that COVID-19 is not only a respiratory disease but also a systemic condition with significant hematological complications. Patients with severe forms of COVID-19 frequently exhibit abnormalities in coagulation, including increased risk of venous thromboembolism, disseminated intravascular coagulation (DIC), and microthrombi in vital organs. The article examines the underlying pathophysiological mechanisms, clinical manifestations, diagnostic challenges, and therapeutic strategies related to coagulation disorders in COVID-19. Particular attention is given to the role of endothelial dysfunction, hyperinflammatory response, and cytokine storm in promoting a hypercoagulable state.

Keywords: COVID-19, coagulation, thrombosis, blood clotting, D-dimer, endothelial dysfunction, hypercoagulability, cytokine storm.

INTRODUCTION

The COVID-19 pandemic, caused by the SARS-CoV-2 virus, has presented unprecedented challenges to global healthcare. While initial attention focused primarily on respiratory symptoms and complications, ongoing clinical observations revealed that COVID-19 also exerts profound effects on the hematological system. Altered blood clotting emerged as one of the most serious and life-threatening complications of the disease. Increased incidence of pulmonary embolism, deep vein thrombosis, and ischemic strokes among COVID-19 patients pointed to the existence of a hypercoagulable state triggered by viral infection. Understanding these changes in blood clotting is crucial for improving treatment outcomes, preventing fatalities, and developing long-term management strategies for survivors.

MATERIALS AND METHODS

COVID-19 is associated with a hypercoagulable state, in which the balance of the hemostatic system is shifted toward excessive clot formation. Clinical studies have shown elevated levels of D-dimer, fibrinogen, and other coagulation markers in many hospitalized patients. High D-dimer levels, in particular, are strongly correlated with poor prognosis and increased risk of mortality. These laboratory findings suggest that COVID-19 triggers widespread activation of the clotting cascade, resulting in the formation of thrombi in both large vessels and microcirculation [1].

The underlying mechanisms of these clotting abnormalities are multifactorial. One key factor is endothelial dysfunction caused by the direct invasion of SARS-CoV-2 into endothelial cells via ACE2 receptors. This damages the vascular lining, exposing procoagulant factors and triggering platelet aggregation. Simultaneously, the so-called cytokine storm, characterized by elevated levels of interleukins and tumor necrosis factor-alpha (TNF-α), amplifies the inflammatory response, further activating the coagulation cascade. The combined effects of endothelial injury, systemic inflammation, and platelet hyperactivation create conditions conducive to thrombosis.

RESULTS AND DISCUSSION

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Another important aspect of COVID-related coagulation changes is the occurrence of microvascular thrombosis. Autopsy studies have revealed the presence of widespread microthrombi in the lungs, kidneys, liver, and brain of deceased patients. These microclots impair organ perfusion, contributing to multi-organ dysfunction and increasing mortality. Unlike traditional thromboembolic diseases, COVID-19-related microthrombosis appears to be linked more closely to immune-mediated mechanisms, such as complement activation, than to conventional clotting disorders. This immune-thrombotic interaction is now recognized as a hallmark of severe COVID-19 pathology [2].

Clinical manifestations of altered blood clotting in COVID-19 are diverse. Patients may develop venous thromboembolism (VTE), including deep vein thrombosis and pulmonary embolism, often without classic symptoms. Some also experience arterial thrombotic events, such as myocardial infarction or ischemic stroke, even in the absence of pre-existing cardiovascular disease. Disseminated intravascular coagulation (DIC), though less common, has been reported in critically ill patients, characterized by simultaneous clotting and bleeding due to exhaustion of clotting factors. Such wide-ranging manifestations highlight the complexity of COVID-associated coagulopathy.

Diagnosis of coagulation changes in COVID-19 requires careful laboratory and imaging evaluation. Routine monitoring of D-dimer, prothrombin time (PT), activated partial thromboplastin time (aPTT), fibrinogen, and platelet count is essential in hospitalized patients. Doppler ultrasound and CT angiography may be used to detect thrombotic complications. However, the unpredictable nature of COVID-19-related thrombosis poses challenges, as clotting events can develop rapidly and unexpectedly, even in patients receiving anticoagulant therapy [3].

Therapeutic strategies for managing coagulation disorders in COVID-19 include anticoagulant therapy, most commonly with low-molecular-weight heparin (LMWH). Prophylactic anticoagulation has become standard practice for hospitalized COVID-19 patients to prevent thromboembolic complications. In severe cases with significantly elevated D-dimer levels, intermediate or therapeutic doses may be considered. Additionally, anti-inflammatory treatments, such as corticosteroids and immunomodulators, indirectly reduce thrombotic risk by dampening the cytokine storm. Nevertheless, balancing anticoagulation with bleeding risk remains a major challenge, requiring individualized treatment plans.

Long-term consequences of COVID-related clotting abnormalities are still being studied. Some survivors report persistent symptoms linked to post-COVID syndrome, including fatigue, chest pain, and cognitive impairment, which may be related to microvascular damage from previous thrombotic events. The recognition of COVID-19 as a systemic disease with significant hematological implications emphasizes the need for long-term follow-up and rehabilitation of patients [4].

One important area of discussion in COVID-associated clotting abnormalities is the influence of genetic and individual susceptibility. Not all patients infected with SARS-CoV-2 develop thrombotic complications, suggesting that inherited differences in coagulation pathways may play a role. Variations in genes related to clotting factors, such as Factor V Leiden mutation or prothrombin G20210A, may predispose individuals to thrombosis when combined with the pro-inflammatory environment induced by COVID-19. Similarly, polymorphisms affecting ACE2 receptor expression could influence viral entry and vascular injury, thereby modifying the risk of coagulopathy. These observations highlight the importance of personalized medicine approaches in predicting and managing clotting risks in COVID-19 patients.

Another key factor is the impact of comorbidities, particularly diabetes mellitus, obesity, and hypertension, which are highly prevalent among patients with severe COVID-19. These conditions

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are already associated with endothelial dysfunction and chronic low-grade inflammation, both of which enhance the risk of clot formation. For instance, obese individuals often exhibit elevated levels of procoagulant factors such as fibrinogen and plasminogen activator inhibitor-1 (PAI-1), predisposing them to thrombosis. When combined with the acute inflammatory surge of COVID-19, this baseline vulnerability intensifies the likelihood of venous and arterial thromboembolic events. Thus, patients with metabolic and cardiovascular comorbidities represent a particularly high-risk group requiring vigilant monitoring of coagulation parameters.

Although most reports have focused on adults, pediatric and young adult patients also exhibit unique clotting challenges in the context of COVID-19. While children typically present with milder respiratory symptoms, some develop multisystem inflammatory syndrome in children (MIS-C), a rare but serious condition characterized by hyperinflammation and cardiovascular involvement. MIS-C has been linked to thrombotic events, myocardial injury, and coagulopathy despite the absence of traditional risk factors. In adolescents and young adults, thrombotic complications such as stroke have been documented, underscoring that age alone does not provide immunity against COVID-associated coagulopathies. These findings reinforce the need for clinicians to remain alert to clotting abnormalities even in younger populations [5].

Gender and hormonal influences also play a role in shaping clotting risks during COVID-19. Men are generally observed to have higher rates of severe disease and thrombotic complications, potentially due to differences in immune response, hormonal regulation, and prevalence of comorbidities. At the same time, women, particularly those using hormonal contraceptives or undergoing hormone replacement therapy, may also face increased thrombotic risk. Estrogen is known to enhance coagulability, and in the context of COVID-19, it may act as an additional trigger for thrombotic events. Understanding these gender-specific differences is essential for tailoring prevention and treatment strategies, especially in populations with overlapping risk factors.

Finally, an emerging area of research concerns the impact of vaccination and antiviral therapies on clotting dynamics. While rare cases of vaccine-induced immune thrombotic thrombocytopenia (VITT) have been reported with certain adenovirus-vector vaccines, the overall benefits of vaccination in reducing COVID-related clotting far outweigh the risks. Vaccinated individuals who contract COVID-19 generally experience milder disease, with reduced incidence of hypercoagulable complications. Similarly, early administration of antiviral agents such as remdesivir or nirmatrelvir/ritonavir may limit viral replication, attenuate systemic inflammation, and indirectly reduce thrombotic burden. These developments illustrate how preventive and therapeutic strategies can reshape the landscape of COVID-associated coagulation disorders, offering hope for improved outcomes in future waves of the pandemic.

CONCLUSION

COVID-19 has redefined the understanding of viral infections by demonstrating how a respiratory illness can profoundly affect the blood clotting system. The disease induces a hypercoagulable state through mechanisms involving endothelial dysfunction, systemic inflammation, and immune-mediated thrombosis. Clinical outcomes are heavily influenced by the presence of coagulation abnormalities, which significantly increase morbidity and mortality. Effective management requires early recognition, continuous monitoring, and appropriate anticoagulant therapy. Future research should focus on refining treatment protocols, understanding long-term consequences, and developing targeted therapies to address the unique coagulopathies associated with COVID-19.

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