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COVID-19 AND THROMBOSIS: HEMOSTATIC DISORDERS IN THE CARDIOVASCULAR SYSTEM

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Abstract. COVID-19 is now understood not only as a respiratory infection but also as a systemic vascular and thromboinflammatory disease. Since the early phases of the pandemic, clinical and pathological studies have shown that SARS-CoV-2 infection can disturb hemostatic balance, activate coagulation pathways, injure the endothelium, alter platelet function, impair fibrinolysis, and trigger both venous and arterial thrombosis.

Keywords: COVID-19, SARS-CoV-2, thrombosis, hemostasis, coagulopathy, endothelium, platelet activation, cardiovascular system, venous thromboembolism, pulmonary embolism, myocardial infarction, stroke, microthrombosis, inflammation, D-dimer.

INTRODUCTION

COVID-19-associated thrombosis remains highly relevant because the infection continues to affect high-risk groups, and the cardiovascular burden of the disease extends beyond the acute viral phase[1,2]. The Centers for Disease Control and Prevention notes that severe COVID-19 outcomes are more likely in older adults and in people with underlying conditions such as heart failure, coronary artery disease, cardiomyopathies, diabetes, chronic kidney disease, cerebrovascular disease, pulmonary hypertension, and prior pulmonary embolism. These same groups are also more vulnerable to thrombotic and cardiovascular complications once infected. At the same time, more recent cardiovascular literature indicates that the consequences of COVID-19 are not limited to pneumonia alone; patients may develop venous thromboembolism, myocardial injury, arrhythmias, ischemic events, and persistent vascular dysfunction even after clinical recovery. Therefore, the study of thrombosis and hemostatic disorders in COVID-19 has important implications not only for acute management but also for long-term cardiovascular prevention, rehabilitation, and risk stratification[3,4].

COVID-19-associated thrombosis represents one of the clearest examples of how viral infection can destabilize the delicate equilibrium between coagulation and anticoagulation. Under physiological conditions, normal hemostasis depends on the coordinated interaction of the vascular endothelium, circulating platelets, coagulation factors, fibrinolytic pathways, and regulatory proteins that prevent inappropriate clotting. In COVID-19, this balance is disrupted by intense inflammation, endothelial injury, immune activation, tissue factor expression, complement activation, platelet hyperreactivity, and impaired fibrinolysis. Instead of a simple bleeding disorder or a classic disseminated intravascular coagulation pattern, many patients develop a prothrombotic state characterized by elevated D-dimer, increased fibrin formation, endothelial activation, and thrombi in large vessels as well as in the microcirculation[5,6]. Reviews published in 2024 and 2025 emphasize that the COVID-19 thrombus has distinctive features, including a highly inflammatory signature built on endothelial inflammation and neutrophil extracellular traps, which help explain why thrombosis became such a central clinical problem in moderate and severe disease.

The endothelial layer occupies a central position in the pathogenesis of COVID-19-related thrombosis. The endothelium is not merely a passive lining of blood vessels; it regulates vascular tone, anticoagulant signaling, leukocyte trafficking, platelet adhesion, and fibrinolytic activity. When endothelial cells are activated or damaged, they shift from an anticoagulant to a procoagulant and proinflammatory phenotype. In COVID-19, endothelial dysfunction has been repeatedly linked to disease severity and vascular complications[7,8]. Reviews describe how SARS-CoV-2 infection, inflammatory mediators, oxidative stress, and immune dysregulation can promote endothelial activation, increase von Willebrand factor release, favor P-selectin-mediated adhesion, and reduce the normal antithrombotic properties of the vessel wall. This

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process is especially dangerous in the cardiovascular system because endothelial injury in coronary, pulmonary, cerebral, and peripheral vessels can facilitate both macrovascular and microvascular clot formation, leading to ischemic damage in vital organs.

Inflammation and thrombosis are tightly linked in COVID-19, which is why the term "thromboinflammation" is often used. Severe infection may trigger a cytokine-rich inflammatory state that amplifies coagulation cascades and suppresses endogenous anticoagulant mechanisms. Neutrophils release extracellular traps, monocytes express tissue factor, and activated platelets interact with leukocytes and endothelial cells in a manner that promotes fibrin-rich clot formation. Complement system activation adds another layer of vascular injury and coagulation amplification. As a result, COVID-19 does not simply increase the probability of ordinary clotting; it creates a biologically aggressive environment in which clot formation becomes part of the inflammatory response itself. This helps explain why thrombosis in COVID-19 can occur in venous, arterial, and microvascular beds simultaneously, and why even patients receiving standard prophylaxis have sometimes developed clinically significant thromboembolic events[9,10].

Platelet dysfunction is another major feature of COVID-19-associated coagulopathy. Normally, platelets prevent bleeding by adhering to injured vascular surfaces and promoting clot formation when necessary. In COVID-19, however, platelet activation may become excessive and maladaptive. Activated platelets can increase thrombin generation, support leukocyte recruitment, enhance endothelial injury, and accelerate the development of both macrothrombi and microthrombi. This has special relevance for the cardiovascular system because platelet-driven thrombosis is closely related to acute coronary syndromes, arterial occlusion, and myocardial ischemia. When combined with inflammation, endothelial dysfunction, and circulating procoagulant factors, platelet hyperreactivity contributes to a state in which patients are vulnerable not only to venous thromboembolism but also to myocardial infarction and stroke.

One of the most clinically important manifestations of these hemostatic disturbances is venous thromboembolism, especially deep vein thrombosis and pulmonary embolism. Pulmonary embolism is particularly significant in COVID-19 because it worsens hypoxemia, increases right ventricular afterload, impairs pulmonary circulation, and may contribute to sudden hemodynamic deterioration[11]. A 2024 observational study published in *Heart & Lung* reported that thromboembolic events occurred in 5.5% of hospitalized COVID-19 patients in its cohort, while cardiovascular complications overall affected 14.3% of patients. The same study found that older age, pre-existing cardiovascular disease, hypertension, diabetes, smoking history, longer hospitalization, and higher D-dimer levels were associated with greater risk. These findings are consistent with the broader literature showing that thromboembolic complications are especially pronounced in patients with severe disease, prolonged immobilization, systemic inflammation, and multiple comorbidities.

Arterial thrombosis is less common than venous thrombosis in many settings, but when it occurs its consequences are often devastating. COVID-19-associated arterial thrombosis may present as acute myocardial infarction, ischemic stroke, limb ischemia, or other ischemic vascular syndromes. Several mechanisms may contribute: plaque destabilization from systemic inflammation, endothelial dysfunction, platelet activation, coronary thrombosis, vasoconstriction, and impaired microcirculatory perfusion. In the same 2024 *Heart & Lung* study, myocardial infarction and/or stroke occurred in 2.39% of hospitalized patients, and the odds were particularly increased in patients with dementia, hemiplegia, smoking history, and higher C-reactive protein concentrations[12,13]. These findings underline an important clinical point: COVID-19 is not only a disease of venous clots but also a condition capable of precipitating arterial cardiovascular emergencies, particularly in biologically vulnerable patients.

Microvascular thrombosis deserves special attention because it bridges the fields of hematology, cardiology, pulmonology, and critical care. In many patients with severe COVID-19, thrombotic injury does not occur only in large vessels visible on routine imaging. Instead,

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microthrombi may form in the pulmonary circulation, coronary microvasculature, renal capillaries, and other small vessels, contributing to tissue hypoxia, myocardial dysfunction, multiorgan injury, and persistent inflammation. In the heart, microvascular thrombosis may worsen myocardial oxygen mismatch even in the absence of classic epicardial coronary occlusion. This helps explain why some patients develop elevated cardiac biomarkers, ischemic ECG changes, ventricular dysfunction, or heart failure without a typical obstructive coronary pattern. The cardiovascular impact of COVID-19, therefore, is often mediated not only by large clots but also by diffuse microcirculatory injury[14,15].

The effects of COVID-19-related hemostatic abnormalities on the heart are broad and clinically important. Pulmonary embolism can produce acute right ventricular strain and right-sided heart failure. Coronary thrombosis and platelet-rich arterial clots can trigger myocardial infarction. Microvascular obstruction may contribute to myocardial injury and worsening left ventricular function. Systemic inflammation and ischemia may provoke arrhythmias, destabilize pre-existing heart failure, and intensify the burden on patients with underlying coronary artery disease or cardiomyopathy. More recent reviews of the long-term cardiovascular impact of COVID-19 have also raised concern about persistent vascular dysfunction and increased risk of cardiovascular events after the acute phase, suggesting that the thromboinflammatory consequences of infection may have a prolonged clinical tail in at least some patients[16,17].

Biomarkers play a crucial role in recognizing COVID-19-associated coagulopathy. Among them, D-dimer has become one of the most widely used and clinically meaningful markers. Elevated D-dimer reflects active fibrin formation and breakdown and has repeatedly been associated with more severe disease and greater thrombotic risk. The 2024 *Heart & Lung* study found that higher D-dimer levels independently increased the odds of thromboembolic events. Other laboratory abnormalities may include increased fibrinogen, elevated inflammatory markers such as C-reactive protein, altered platelet counts, prolonged clotting times in some cases, and evidence of endothelial injury. However, no single biomarker should be interpreted in isolation[18,19]. A comprehensive assessment combining laboratory results, imaging findings, oxygenation status, cardiac markers, and clinical context remains essential.

From a diagnostic perspective, the challenge lies in distinguishing ordinary clinical deterioration from thrombotic progression. Worsening hypoxemia, pleuritic chest pain, new tachycardia, unexplained right ventricular dysfunction, sudden hypotension, asymmetrical limb swelling, acute neurologic deficits, or rising D-dimer levels should raise suspicion for thrombotic events. In cardiovascular practice, ECG, echocardiography, cardiac biomarkers, venous ultrasound, CT pulmonary angiography, and brain imaging may all be relevant depending on the presentation. Importantly, the overlap between pulmonary inflammation, myocardial injury, and thrombosis means that clinicians must think integratively rather than organ by organ. A patient with COVID-19 and chest pain may have myocarditis, pulmonary embolism, myocardial infarction, or a mixed picture involving several mechanisms at once.

Risk stratification remains central to prevention. The CDC identifies older age as the strongest risk factor for severe COVID-19 outcomes and notes higher risk among individuals with cardiovascular disease, diabetes, chronic kidney disease, cerebrovascular disease, pulmonary hypertension, and other major comorbidities. In the 2024 cardiovascular observational study, cardiovascular complications were more frequent in older patients and in those with pre-existing cardiovascular disease, hypertension, diabetes mellitus, smoking history, and more severe pneumonia at presentation. The practical implication is clear: thrombosis prevention and cardiovascular surveillance should be most vigilant in patients who are older, multimorbid, immobilized, severely inflamed, or previously affected by vascular disease.

Treatment principles for COVID-19-associated thrombosis center on prevention, early recognition, and individualized antithrombotic management. Standard thromboprophylaxis became a cornerstone of inpatient care early in the pandemic because of the high burden of venous thromboembolism. Yet subsequent experience showed that not every patient benefits

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from the same intensity of anticoagulation, and the risk of bleeding must always be balanced against thrombotic risk. Patients with confirmed pulmonary embolism, deep vein thrombosis, myocardial infarction, or cardioembolic stroke require condition-specific evidence-based treatment, while those with high D-dimer, immobilization, severe pneumonia, or critical illness may require closer monitoring and prophylactic strategies according to evolving institutional or specialty guidance. In broader terms, management is most effective when it addresses both the clot itself and the inflammatory-endothelial environment that produced it.

Long-term follow-up is increasingly important because the cardiovascular consequences of COVID-19 may persist beyond the acute infection. Recent reviews describe prolonged cardiovascular risk, ongoing endothelial dysfunction, and continued concern for thrombotic and ischemic complications after recovery in some patient populations. This does not mean that every person with COVID-19 will develop chronic cardiovascular disease, but it does mean that clinicians should not automatically regard hospital discharge or viral recovery as the end of risk. Patients who experienced severe disease, thromboembolic events, myocardial injury, or substantial cardiovascular symptoms may benefit from continued assessment, risk factor modification, and rehabilitation-oriented follow-up.

From a broader scientific perspective, COVID-19 changed the way medicine understands the intersection between infection, coagulation, and cardiovascular disease. It demonstrated with unusual clarity that the cardiovascular system can become a major target of systemic viral inflammation through endothelial damage, platelet activation, coagulation cascade dysregulation, and microvascular injury. It also showed that hemostatic disruption is not merely a laboratory abnormality but a clinical process with direct consequences for survival, organ function, and long-term health. For this reason, the study of COVID-19 and thrombosis remains relevant not only for pandemic medicine but also for future approaches to viral vascular syndromes more generally.

Conclusion. In conclusion, COVID-19-associated thrombosis is one of the most significant cardiovascular expressions of SARS-CoV-2 infection. The disease disrupts normal hemostasis through a complex interaction of endothelial injury, inflammatory activation, platelet hyperreactivity, coagulation cascade amplification, neutrophil extracellular traps, and impaired fibrinolysis. As a result, patients may develop venous thrombosis, pulmonary embolism, myocardial infarction, ischemic stroke, and diffuse microvascular thrombosis, all of which can severely affect cardiovascular function. Older age, pre-existing cardiovascular disease, diabetes, kidney disease, smoking history, and severe inflammatory illness substantially increase the risk of these complications. Elevated D-dimer and related markers help identify patients at risk, but clinical judgment and integrated cardiovascular assessment remain essential. The overall evidence indicates that COVID-19 should be understood as a thromboinflammatory vascular disease as much as a respiratory one, and that its effects on the cardiovascular system may continue beyond the acute phase in a subset of patients. Therefore, the prevention, diagnosis, and treatment of thrombosis must remain central to both acute COVID-19 management and long-term cardiovascular care.

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