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Cardiovascular Implications of Long COVID: What We Know So Far

Spread Faint*

Department of Cardiovascular Radiology, University of Belgrade, Belgrade, Serbia

Introduction

The COVID-19 pandemic, caused by the SARS-CoV-2 virus, has led to a global health crisis with wide-ranging impacts on various body systems. While acute COVID-19 is well-documented, the long-term effects, often referred to as "Long COVID," are only beginning to be understood. Among the most concerning are the cardiovascular implications, which pose significant challenges for public health and clinical management. This article explores the current understanding of the cardiovascular consequences of Long COVID, examining potential mechanisms, clinical manifestations and management strategies. As the COVID-19 pandemic progresses, attention has shifted from the acute phase of the disease to its long-term effects, often termed Long COVID or post-acute squealed of SARS-CoV-2. Long COVID is characterized by a range of persistent symptoms that extend beyond the typical recovery period, affecting various organ systems, including the cardiovascular system. Understanding the cardiovascular implications of Long COVID is crucial for managing the long-term health of millions of individuals worldwide. One of the most concerning cardiovascular implications of Long COVID is myocarditis, an inflammation of the heart muscle and pericarditis, inflammation of the lining around the heart. Both conditions have been observed in patients who have recovered from acute COVID-19. Myocarditis can lead to impaired heart function, arrhythmias and in severe cases, heart failure. Imaging studies, such as cardiac MRI, have revealed persistent myocardial inflammation in some individual's months after the initial infection [1].

Description

Thromboembolism, including Deep Vein Thrombosis (DVT) and Pulmonary Embolism (PE), has been documented in Long COVID patients. The hypercoagulable state induced by the SARS-CoV-2 virus may persist even after the acute phase, leading to an increased risk of clot formation. This risk is particularly concerning because thromboembolic events can result in significant morbidity and mortality, especially in those with pre-existing cardiovascular conditions. Autonomic dysfunction, particularly Postural Orthostatic Tachycardia Syndrome (POTS), is another cardiovascular manifestation reported in Long COVID patients. POTS are characterized by an abnormal increase in heart rate upon standing, accompanied by symptoms such as dizziness, palpitations and fatigue. This condition is thought to be related to autonomic nervous system deregulations, which may be triggered or exacerbated by SARS-CoV-2 infection. Emerging evidence suggests that micro vascular dysfunction, affecting the small blood vessels, may play a role in the cardiovascular symptoms of Long COVID. The underlying mechanisms may involve endothelial cell damage, inflammation and immune system deregulations. Understanding the mechanisms underlying the cardiovascular implications of Long COVID is essential for developing effective treatments. Several potential mechanisms have been proposed. Chronic inflammation is a hallmark of Long COVID and is thought to contribute to on-going cardiovascular issues. Elevated levels of inflammatory markers, such as C-reactive protein and interleukin-6, have been observed in Long COVID patients, indicating a state of persistent immune activation. This chronic inflammation can damage the cardiovascular system, leading to conditions like myocarditis and endothelial dysfunction [2,3].

The endothelium, the inner lining of blood vessels, plays a crucial role in vascular health. SARS-CoV-2 can infect endothelial cells, leading to endothelial dysfunction, which in turn can result in micro vascular damage, impaired blood flow and an increased risk of thromboembolism. Endothelial dysfunction is also linked to the development of atherosclerosis, further increasing the risk of cardiovascular events in Long COVID patients. The autonomic nervous system, which controls involuntary bodily functions, may be disrupted by SARS-CoV-2 infection, leading to conditions like POTS. Long COVID is associated with immune system deregulations, which may contribute to on-going cardiovascular issues. These deregulations could lead to autoimmune responses, where the body's immune system mistakenly attacks its tissues, including the heart and blood vessels. Autoantibodies targeting cardiovascular tissues have been detected in some Long COVID patients, suggesting a potential role for autoimmune mechanisms. Managing the cardiovascular implications of Long COVID presents unique challenges, as the condition is still not fully understood and there are no standardized treatment protocols. However, several approaches have been suggested. Regular cardiovascular monitoring is essential for Long COVID patients, especially those with a history of cardiovascular disease or who present with new cardiovascular symptoms. This may include routine imaging, such as echocardiography or cardiac MRI and laboratory tests to assess inflammatory markers and coagulation status [4].

Given the role of inflammation in the cardiovascular implications of Long COVID, anti-inflammatory therapies may be beneficial. Corticosteroids, No Steroidal Anti-Inflammatory Drugs (NSAIDs) and more targeted immunomodulatory therapies, such as monoclonal antibodies, are being explored as potential treatments. However, the long-term use of these therapies requires careful consideration due to potential side effects. For patients at high risk of thromboembolic events, anticoagulation therapy may be necessary. The duration and intensity of anticoagulation therapy should be individualized based on the patient's risk factors and clinical presentation. It is essential to balance the risk of thrombosis with the potential for bleeding complications. For patients with POTS or other forms of autonomic dysfunction, a multidisciplinary approach is often required. This may include lifestyle modifications, such as increased fluid and salt intake, physical therapy and medications like beta-blockers or fludrocortisone to manage symptoms. The exact mechanism is not fully understood, but it may involve direct viral damage to autonomic pathways or an immune-mediated response that disrupts normal autonomic function. This dysfunction can lead to impaired blood flow, contributing to symptoms such as chest pain, fatigue and shortness of breath [5].

Conclusion

The cardiovascular implications of Long COVID represent a significant and growing concern as the number of individuals affected by the condition increases. While much remains to be understood about the mechanisms and optimal management strategies, it is clear that Long COVID can have lasting effects on cardiovascular health. Continued research and clinical vigilance

***Address for Correspondence:** Spread Faint, Department of Cardiovascular Radiology, University of Belgrade, Belgrade, Serbia; E-mail: sfaint@gmail.com*

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are essential to address these challenges and improve outcomes for those affected by Long COVID. As our understanding evolves, so too will our ability to effectively manage and treat this complex condition, ultimately improving the quality of life for millions of people worldwide.

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Conflict of Interest

None.

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