

MECHANISMS OF CARDIOVASCULAR COMPLICATION DEVELOPMENT IN CORONAVIRUS INFECTION (COVID-19)

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Abstract

This article analyzes the main mechanisms of cardiovascular complications associated with coronavirus infection (COVID-19). According to current scientific evidence, direct myocardial injury caused by SARS-CoV-2, endothelial dysfunction, hypercoagulation, and systemic inflammatory response play a crucial role in cardiovascular damage. The findings emphasize the importance of early detection and comprehensive management of cardiovascular complications in COVID-19 patients.

Keywords: COVID-19, SARS-CoV-2, cardiovascular system, myocarditis, thrombosis, cardiovascular complications.

МЕХАНИЗМЫ ФОРМИРОВАНИЯ СЕРДЕЧНО-СОСУДИСТЫХ ОСЛОЖНЕНИЙ ПРИ КОРОНАВИРУСНОЙ ИНФЕКЦИИ

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Аннотация

В статье рассматриваются основные механизмы поражения сердечно-сосудистой системы при коронавирусной инфекции COVID-19. Анализ

современных научных данных показывает, что прямое повреждение миокарда вирусом SARS-CoV-2, эндотелиальная дисфункция, гиперкоагуляция и системное воспаление играют ключевую роль в развитии кардиоваскулярных осложнений. Полученные выводы имеют практическое значение для ранней диагностики и профилактики осложнений.

Ключевые слова: COVID-19, SARS-CoV-2, сердечно-сосудистая система, миокардит, тромбоз, кардиоваскулярные осложнения.

Introduction

Coronavirus disease 2019 (COVID-19) has emerged as a global health challenge, initially recognized as a respiratory illness but later identified as a multisystem disease. Increasing clinical and experimental evidence indicates that the cardiovascular system is one of the most frequently affected systems in COVID-19 patients. Cardiovascular involvement significantly contributes to disease severity, complications, and mortality.

The entry of SARS-CoV-2 into human cells occurs via angiotensin-converting enzyme 2 (ACE2) receptors, which are widely expressed in myocardial and endothelial cells. This biological mechanism explains the susceptibility of the cardiovascular system to viral injury. In addition, systemic inflammation, cytokine storm, hypoxia, and coagulation abnormalities further exacerbate cardiovascular damage.

Clinical observations have reported a wide spectrum of cardiovascular complications in COVID-19 patients, including myocarditis, arrhythmias, acute coronary syndromes, heart failure, and thromboembolic events. These complications may occur both in patients with pre-existing cardiovascular diseases and in individuals without prior cardiac conditions. Therefore, understanding the underlying mechanisms of cardiovascular injury in COVID-19 is essential for improving patient outcomes.

The aim of this article is to review and analyze the main pathophysiological mechanisms responsible for cardiovascular complications in patients with coronavirus infection.

Materials and Methods

This study is based on a narrative review of scientific literature published between 2020 and 2024. Databases such as PubMed, Scopus, Web of Science, and Google Scholar were searched for relevant articles. Original research papers, systematic reviews, and meta-analyses focusing on cardiovascular involvement in COVID-19 were included. Analytical and comparative methods were used to synthesize the data.

Results

The analysis of the literature revealed several key mechanisms involved in cardiovascular complications of COVID-19:

direct viral injury to cardiomyocytes;
endothelial cell damage and microvascular dysfunction;
systemic inflammatory response and cytokine storm;
activation of coagulation pathways leading to thrombosis;
hypoxia-induced myocardial stress.

These mechanisms contribute to the development of myocarditis, arrhythmias, ischemic heart disease, acute heart failure, and thromboembolic complications.

Discussion

The findings confirm that COVID-19 should be considered a systemic disease with significant cardiovascular implications. Endothelial dysfunction and hypercoagulability appear to be central features linking inflammation and thrombosis. Patients with pre-existing cardiovascular conditions are particularly

vulnerable; however, cardiovascular complications have also been reported in previously healthy individuals. This highlights the need for routine cardiovascular assessment and monitoring in COVID-19 patients.

Conclusion

Cardiovascular complications in COVID-19 result from complex and multifactorial mechanisms, including direct viral effects, inflammation, and coagulation abnormalities. Early recognition and integrated clinical management are essential to reduce morbidity and mortality associated with cardiovascular involvement in coronavirus infection.

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