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ROLE OF CORONAVIRUS INFECTION IN THE DEVELOPMENT OF CARDIOVASCULAR PATHOLOGY

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Coronavirus disease 2019 (COVID-19), caused by Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), has been strongly associated with both acute and chronic cardiovascular complications. Emerging evidence indicates that COVID-19 not only exacerbates pre-existing cardiovascular disease (CVD) but also contributes directly to the development and progression of new cardiovascular pathology through multiple interconnected mechanisms.

COVID-19-related cardiovascular involvement includes myocarditis, myocardial infarction, arrhythmias, heart failure, and thromboembolic events. The incidence of these complications is significant, with heart failure reported in up to 33% of cases and myocardial infarction in up to 11%. Importantly, patients with concurrent COVID-19 and cardiovascular conditions exhibit higher mortality rates, highlighting the clinical relevance of this interaction.

A central mechanism underlying cardiovascular injury in COVID-19 is the interaction between SARS-CoV-2 and angiotensin-converting enzyme 2 (ACE2). ACE2 serves as the viral entry receptor and is widely expressed in cardiovascular tissues. Viral binding leads to downregulation of ACE2, disrupting the protective ACE2/Ang1-7 pathway and enhancing the deleterious effects of the ACE/Ang II axis. This imbalance promotes vasoconstriction, inflammation, fibrosis, and myocardial injury, thereby accelerating cardiovascular disease progression.

Endothelial dysfunction is another key contributor. SARS-CoV-2 directly infects endothelial cells and induces inflammatory and oxidative stress responses, resulting in impaired vascular integrity and function. This dysfunction promotes thrombosis, increased vascular permeability, and inflammatory cascades, all of which contribute to complications such as pulmonary embolism, myocardial infarction, and microvascular injury.

At the cellular level, immune dysregulation plays a significant role. COVID-19 induces the formation of neutrophil extracellular traps (NETs), which contribute to inflammation, thrombosis, and atherosclerosis progression. Additionally, cellular senescence and the release of pro-inflammatory mediators further amplify tissue damage and sustain chronic cardiovascular complications, particularly in long COVID.

Metabolic disturbances also link COVID-19 to cardiovascular pathology. Through mechanisms including interferon regulatory factors and transcriptional modifications, the infection can cause insulin resistance and metabolic dysregulation. These alterations contribute to myocardial injury, fibrosis, and increased susceptibility to cardiovascular events. Furthermore, disruptions in iron homeostasis and increased oxidative stress promote cardiomyocyte damage and arrhythmogenesis.

Psychosocial factors associated with the pandemic, including stress, reduced physical activity, and social isolation, further exacerbate cardiovascular risk. These factors contribute indirectly by increasing the prevalence of hypertension, metabolic syndrome, and other CVD risk factors.

In conclusion, COVID-19 plays a multifaceted role in the development and progression of cardiovascular pathology through direct viral effects, immune-mediated injury, endothelial dysfunction, and metabolic and psychosocial factors. Understanding these mechanisms is essential for improving risk stratification, prevention, and management of cardiovascular complications in both acute and post-COVID settings.