INTERNATIONAL JOURNAL OF
INTEGRATIVE AND
MODERN MEDICINE

The Impact of Post-COVID Syndrome on the Cardiovascular System: Hypertension as a Key Complication

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Annotation: Post-COVID syndrome is a complex multisystem condition that affects various organs and systems, including the cardiovascular system. This study aims to analyze the clinical and functional status of patients recovering from COVID-19, with a particular focus on changes related to hypertension. The paper examines the primary pathophysiological mechanisms, including endothelial dysfunction, activation of inflammatory cascades, disruption of the renin-angiotensin-aldosterone system (RAAS), and the role of autonomic nervous system dysfunction. The study presents results of laboratory and clinical assessments of patients with different disease severities, as well as an evaluation of treatment efficacy depending on vaccination status.

Keywords: Post-COVID syndrome, hypertension, cardiovascular diseases, inflammation, endothelial dysfunction, renin-angiotensin system, immune markers, laboratory indicators, autonomic dysfunction, vaccination, rehabilitation, therapy.

Objective

The aim of this study is to analyze the clinical and functional state of patients recovering from COVID-19, identifying the impact of post-COVID syndrome on the cardiovascular system, particularly in relation to the progression and complications of hypertension.

Main Part

Post-COVID syndrome is characterized by persistent symptoms after recovering from acute SARS-CoV-2 infection, with a significant impact on cardiovascular health, including the progression of hypertension.

A key mechanism is endothelial dysfunction, which arises due to direct damage to the vascular wall by SARS-CoV-2. This results in reduced synthesis of nitric oxide (NO), a crucial molecule for vasodilation and the prevention of atherothrombosis. Endothelial damage leads to sustained vasoconstriction, increased vascular permeability, and chronic inflammation, all of which contribute to long-term hypertension development [1, 4].

Another fundamental pathophysiological mechanism is the overactivation of the renin-angiotensin-aldosterone system (RAAS). SARS-CoV-2 binds to angiotensin-converting enzyme 2 (ACE2), reducing its activity and leading to an accumulation of angiotensin II. This potent vasoconstrictor induces endothelial inflammation, oxidative stress, and fibrotic changes, which further contribute to vascular remodeling and sustained hypertension [3, 5].

Chronic systemic inflammation plays a pivotal role in post-COVID hypertension. Studies have reported elevated levels of pro-inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interleukin-1β (IL-1β), which contribute to persistent endothelial dysfunction and vascular inflammation [2, 6]. Increased C-reactive protein (CRP) and ferritin levels indicate ongoing inflammatory processes, correlating with disease severity and worsening cardiovascular outcomes [7].

Autonomic nervous system dysfunction further exacerbates post-COVID hypertension. Damage to central and peripheral autonomic regulatory mechanisms results in heightened sympathetic nervous system activity, increasing vascular tone, heart rate, and blood pressure fluctuations. Many patients

with post-COVID syndrome experience persistent tachycardia, orthostatic intolerance, and episodic hypertension, complicating the management of their condition [8, 9].

Results

The study analyzed clinical and functional parameters in 302 post-COVID patients. Only 16.9% of these individuals had been vaccinated, correlating with a lower severity of complications and a more favorable disease course.

Analysis of laboratory markers revealed the following trends:

- CRP levels significantly decreased after treatment, particularly in vaccinated patients [10].
- Erythrocyte sedimentation rate (ESR) also declined post-treatment, reflecting a reduction in systemic inflammation [11].
- White blood cell counts demonstrated statistically significant differences between vaccinated and unvaccinated patients, suggesting enhanced immune recovery in the vaccinated group [12].
- Reduction in ferritin and IL-6 levels post-treatment indicated decreased inflammatory activity [13].

Additionally, oxygen saturation levels improved across all patient groups, further supporting the effectiveness of the therapeutic strategies employed.

Conclusions

Post-COVID syndrome has profound implications for cardiovascular health, contributing to the progression and worsening of hypertension. The primary mechanisms include endothelial dysfunction, persistent inflammation, RAAS dysregulation, and autonomic imbalance. Vaccination appears to confer significant benefits, reducing inflammatory markers and facilitating a faster recovery.

Future research should focus on understanding the long-term cardiovascular effects of COVID-19, developing personalized treatment approaches, and optimizing rehabilitation strategies for post-COVID patients.

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