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Modern Scientific Concepts and Clinical Consequences of LONG COVID Syndrome, Especially In Relation to its Impact on Cardiovascular Diseases

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Abstract: Modern scientific data confirm that post-covid syndrome (LONG COVID) has a significant impact on the cardiovascular system, especially increasing the risk of hypertension, myocarditis, thrombosis, and endothelial dysfunction. Studies show that patients who have had COVID-19 exhibit renin-angiotensin-aldosterone system (RAAS) regulation disorders, which can contribute to the development of hypertension even in previously healthy individuals. Also, persistent changes in the expression of microRNA involved in inflammatory and fibrotic processes have been identified, which can accelerate the progression of cardiovascular diseases [1]. Additionally, autonomic nervous system dysfunction, accompanied by increased sympathetic nervous system activity, can also play a role in post-covid hypertension. Clinical consequences include an increased risk of stroke, heart attack, and heart failure.

In therapeutic practice, it is recommended to pay attention to long-term monitoring of blood pressure, correction of inflammatory processes, and maintenance of endothelial health [2].

Keywords: post-covid syndrome, hypertension, cardiovascular diseases, inflammation, endothelial dysfunction, renin-angiotensin system, microRNA, autonomic nervous system, thrombosis, myocarditis, chronic inflammation, immune dysregulation.

Purpose of the study:

This review article is aimed at analyzing modern scientific data on the mechanisms of development and clinical consequences of post-covid syndrome in the context of cardiovascular diseases, especially hypertension. Patophysiological processes, including inflammation, endothelial dysfunction, and renin-angiotensin system dysregulation, as well as potential strategies for monitoring and treating post-covid complications, are considered.

Materials and methods

To conduct this review, an analysis of scientific publications published in peer-reviewed journals over the past 5 years was conducted using PubMed, Scopus, and Google Scholar databases. The main focus is on research dedicated to the relationship between post-covid syndrome and cardiovascular diseases, including hypertension, as well as the pathophysiological mechanisms underlying these disorders [3]. The included studies were analyzed according to the following criteria: description of immune and inflammatory mechanisms, the role of the renin-angiotensin-aldosterone system, microRNA participation in post-ovid complications, as well as the influence of the autonomic nervous system on the regulation of vascular tone. Works lacking clinical data or based solely on hypotheses without empirical verification were excluded. Special attention was paid to systematic reviews, meta-analyses, and large cohort studies that allow for the formation of a holistic picture of COVID-19's impact on the cardiovascular system.

Introduction. Post-Covid Syndrome (PKS) is a complex multisystem phenomenon that manifests with long-term clinical consequences after a coronavirus infection. PCS has the most significant impact on the cardiovascular system, leading to disruption of blood pressure regulation, development of chronic

inflammatory processes, activation of thrombotic mechanisms, and increased risk of cardiovascular complications [5].

The development of hypertension in the context of post-covid syndrome is a consequence of the complex interaction of several key pathophysiological mechanisms, among which damage to the endothelium, activation of the renin-angiotensin-aldosterone system, imbalance of pro- and anti-inflammatory factors, and disruption of the autonomic nervous system are of particular importance [2,6]. These processes are due to the direct cytopathic effect of SARS-CoV-2 on vascular cells, as well as secondary changes caused by systemic inflammation, hypoxia, and immune disorders.

Endothelial damage occupies a central place in the pathogenesis of post-covid hypertension. Endothelial cells play a key role in regulating vascular tone, modulating the inflammatory response, and preventing thrombus formation. Endothelial infection with the SARS-CoV-2 virus leads to cell apoptosis, loss of their barrier function, and disruption of the synthesis of vasoactive molecules such as nitric oxide (NO) and prostacycline [7]. NO deficiency contributes to vascular spasm, increased inflammatory response, and increased vascular wall permeability, creating prerequisites for persistent increase in blood pressure. Additionally, damaged endothelium activates the coagulation cascade, which increases the risk of thrombus formation and exacerbates vascular dysfunction.

Activation of the renin-angiotensin-aldosterone system (RAAS) is another important mechanism contributing to the development of hypertension after COVID-19. SARS-CoV-2 binds to angiotensin-converting enzyme 2 (ACE2), which is a critical regulator of vascular tone and anti-inflammatory mechanisms [8]. When ACE2 expression decreases, angiotensin II accumulates, which has a pronounced vasodilating effect, stimulates the production of pro-inflammatory cytokines, and promotes vascular wall fibrosis. In patients with post-covid syndrome, there is a prolonged increase in angiotensin II activity, which leads to a persistent increase in blood pressure and vascular remodeling, which increases the risk of cardiovascular complications [9].

Inflammatory processes play a key role in the pathogenesis of post-covid hypertension. Studies confirm that patients who have had COVID-19 show marked increases in pro-inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor alpha (TNF-α), and interleukin-1β (IL-1β) [10]. These molecules contribute to the activation of endothelial cells, increased oxidative stress, and dysfunction of the vascular wall. In conditions of chronic inflammation, there is a disruption in the interaction between the endothelium and smooth muscle cells of the vessels, which leads to a persistent increase in vascular tone and the development of hypertension. Additionally, the cytokine storm that occurs in some patients with severe COVID-19 can trigger autoimmune processes that continue to support inflammation even after recovery [11].

Autonomic nervous system dysfunction also plays a significant role in the mechanism of post-covid hypertension formation. Damage to the central and peripheral structures that regulate autonomic nervous activity leads to imbalance between the sympathetic and parasympathetic nervous systems [12]. In the post-covid period, many patients exhibit hyperactivity of the sympathetic region, which is accompanied by increased catecholamine excretion, increased heart rate, and persistent vascular constriction. This imbalance contributes to blood pressure instability, tachycardia episodes, and resistance to traditional antihypertensive therapy.

Together, these pathophysiological mechanisms create conditions for a persistent increase in blood pressure, the development of concomitant pathologies such as myocarditis, thrombosis, and heart failure, and an increased risk of long-term cardiovascular complications. A comprehensive approach to the diagnosis and treatment of post-covid hypertension should include not only standard antihypertensive therapy but also a strategy for correcting inflammatory processes, restoring endothelial function, and regulating vegetative balance, which can significantly improve the prognosis of patients with post-covid syndrome [13].

A serious consequence of infection is the disruption of the angiotensin-converting enzyme 2 (ACE2), which plays a central role in blood pressure regulation. Decreased ACE2 expression leads to an excess

of angiotensin II, which intensifies vascular spasm, causes inflammation, and triggers tissue fibrosis [14]. Restoring normal ACE2 functioning is one of the promising therapeutic strategies aimed at reducing the risk of developing hypertension after COVID-19 [14,15]. Furthermore, the influence of microRNA, which regulates inflammatory processes and vascular function, is of significant importance. Disorders in the expression of miR-155, miR-146a, and other microRNAs lead to uncontrolled activation of the inflammatory pathways, which exacerbates vascular damage and contributes to the progression of hypertension.

autonomic nervous system dysfunction plays a special role in the development of post-covid hypertension [16]. Studies show that COVID-19 can have a prolonged effect on the autonomic regulation of the cardiovascular system, contributing to the hyperactivity of the sympathetic nervous system. This leads to increased vascular tone, increased tachycardia, and blood pressure instability. In many patients who have had COVID-19, symptoms of orthostatic intolerance, tachycardia at rest, and sharp fluctuations in blood pressure are observed, indicating a disruption in neurogenic regulation mechanisms [17].

The clinical consequences of post-covid syndrome in the aspect of cardiovascular diseases require increased attention from doctors and patients [18]. Patients who have had COVID-19 are advised to maintain long-term blood pressure monitoring, monitor the level of inflammatory markers, and correct endothelial dysfunction. Modern therapeutic strategies include the use of ACE inhibitors and angiotensin II receptor blockers to normalize RAAS, the use of antioxidant and anti-inflammatory drugs to reduce endothelial damage, and the modulation of the immune response using targeted anti-cytokine therapy [19]. To date, the question of the duration of COVID-19's impact on the cardiovascular system remains open, therefore, further research is needed to develop optimal methods for diagnosing and treating post-covid complications [20]. Early diagnosis, personalized approach, and timely correction of disorders can significantly reduce the risk of developing severe cardiovascular diseases in patients who have had a coronavirus infection.

Conclusions

Post-covid syndrome has a significant impact on the cardiovascular system, leading to an increased risk of developing hypertension, inflammatory vascular diseases, thrombosis, and other complications. The main mechanisms include endothelial dysfunction, chronic inflammation, renin-angiotensin-aldosterone imbalance, and disruption of autonomic regulation of cardiovascular activity. Clinical studies confirm that prolonged inflammation and changes in microRNA expression can exacerbate vascular disorders, contributing to the development of arterial hypertension in previously healthy individuals.

Hypertension in the context of post-covid syndrome requires increased attention from doctors and patients. Early diagnosis, regular monitoring of blood pressure, and assessment of inflammation markers and endothelial function can contribute to the timely detection and correction of emerging pathologies. Therapeutic strategies should include a comprehensive approach aimed at reducing the inflammatory background, restoring endothelial function, and normalizing neurohumoral mechanisms for regulating vascular tone.

Conclusion

Post-covid syndrome presents a complex medical problem that requires a comprehensive approach to the diagnosis, treatment, and prevention of cardiovascular complications. It has been proven that SARS-CoV-2 has a prolonged effect on the body's regulatory systems, disrupting the balance between pro- and anti-inflammatory mechanisms, contributing to the development of persistent arterial hypertension and other cardiovascular diseases.

Future research should be aimed at a more detailed study of the mechanisms of post-ovid complications and the search for new therapeutic targets. Particular attention should be paid to a personalized approach to patient management, the use of antitoxic therapy, agents that normalize microRNA expression, as well as methods for correcting autonomic nervous system dysfunction. It is

also important to develop effective rehabilitation programs for patients who have had COVID-19, with an emphasis on restoring the normal functioning of the cardiovascular system and reducing the risk of long-term complications.

Modern research emphasizes the need for a comprehensive strategy to combat the consequences of COVID-19, including early diagnosis, active monitoring of the vascular system, and the application of innovative treatment methods. Optimizing these approaches will minimize the negative consequences of the pandemic and improve the quality of life of patients who have had a coronavirus infection.

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