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PATHOMORPHOLOGY OF THE LUNGS IN POST-COVID SYNDROME

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Abstract: This article examines pathological changes that develop in lung tissue in post-COVID syndrome. It focuses on predicting outcomes and complications, as well as the morphogenesis of possible secondary proliferative lesions. Additionally, the study analyzes sclerotic changes in lung tissue and highlights specific features in localized areas of morphofunctionally impaired bronchopulmonary segments. Based on these findings, the work aims to develop recommendations for improving the effectiveness of rehabilitation and tactical treatment using clearly defined criteria.

Keywords: disease, syndrome, muscle, pneumonia, diagnosis, lung, morphogenesis, symptom, tissue, pathophysiology, rehabilitation, intensive care.

Coronavirus infection 2019 (COVID-19) is an infectious disease caused by SARS-CoV-2, the virus responsible for severe acute respiratory syndrome. The disease was first identified in 2019 in Wuhan, China, and spread globally, leading to the 2019–2020 coronavirus pandemic. It is characterized by symptoms such as high fever, cough, and difficulty breathing. In some cases, muscle pain, sputum production, and sore throat are also observed. Although most infected individuals experience mild symptoms, in some patients the disease can lead to severe pneumonia and multiple organ failure. Among diagnosed cases, the average mortality rate is about 3.4%. This rate is approximately 0.2% among individuals under 20 years of age and about 15% among those over 80.

It has now been four years since the emergence of the novel coronavirus infection COVID-19 caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). COVID-19 has had a profound impact on public health systems and social structures, triggering a global economic crisis. The development of COVID-19 vaccines has significantly reduced the risk of SARS-CoV-2 infection and prevented deaths and severe forms of the disease following acute infection. However, approximately 10–30% of patients who recover from COVID-19 experience chronic conditions that persist for more than three months and develop within 28 days after the acute illness. These ongoing health issues are referred to as post-COVID syndrome or long COVID, also known as post-acute sequelae of SARS-CoV-2 infection (PASC). The symptoms reported by survivors vary widely, but common manifestations include fatigue, insomnia, shortness of breath, and cough.

A subset of PASC involves pulmonary manifestations, where persistent lung-related symptoms continue to trouble patients even after recovery from acute infection. Recent large cohort studies have shown that individuals who have had COVID-19 are at an increased risk of developing comorbid conditions, particularly diabetes, as well as cardiovascular and kidney diseases. Given the global prevalence of PASC and its long-term consequences in COVID-19

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survivors—affecting quality of life and contributing to years lived with disability—PASC remains a serious global challenge for healthcare systems today.

COVID-19 usually causes only mild symptoms, but some people can become severely ill. This is more common among older individuals or those with pre-existing conditions. Therefore, it is important for everyone to help slow the spread of infection by consistently following rules and regulations established at local and national levels. After being infected, it typically takes about 4–5 days before symptoms of COVID-19 appear. In most infected individuals, symptoms develop within 10 days. You are most infectious 1–2 days before symptoms appear and on the first day of symptoms. In post-COVID syndrome, recommendations are developed to improve the effectiveness of rehabilitation and tactical treatment based on clearly defined criteria. These include predicting the outcomes and complications of pathological changes developing in lung tissue, studying the morphogenesis of possible secondary proliferative lesions, and identifying specific features of sclerotic changes in lung tissue as well as localized areas in morphofunctionally impaired bronchopulmonary segments. The morphofunctional aspects of pulmonary-cardiac pathology developing as a result of hypertension in lung tissue are also elucidated. In addition, recommendations are provided to improve bronchial drainage indicators in lung tissue segments.

Globally, the sharp increase in secondary complications following the COVID-19 pandemic is characterized by the emergence of background and comorbid diseases in an associated form, especially in patients with chronic conditions. In the United States and European countries, since late 2020 and throughout 2021, there has been a rapid rise in pathological changes affecting the lungs, pancreas, heart, kidneys, and musculoskeletal system, collectively referred to as post-COVID syndrome. This trend is particularly explained by processes occurring in lung tissue, including carnification (consolidation of lung tissue), the development of hypertension in the pulmonary circulation, and, as a consequence, an increase in mortality associated with pulmonary heart failure from 3.4% to 31.4%. At the same time, in post-COVID syndromes, morphofunctional changes in the cardiopulmonary system lead to hemodynamic disturbances in all parenchymal organs. In particular, hypofunctional states in liver tissue have been observed. However, morphological aspects of membranous edema in lung tissue and pulmonary fibrosclerosis remain insufficiently studied. Furthermore, clear clinical and morphological criteria for treatment strategies have not yet been fully developed, highlighting the relevance and importance of this topic.

In post-COVID syndrome, the morphological landscape of lung tissue presents in a wide variety of forms, which are often not fully reflected in the patient's medical history. As a result, the pathology may progress in a latent manner, with clinical and morphological signs remaining masked by other underlying diseases. In many cases, this condition develops among the working-age population and may lead to unexpectedly high mortality rates. In post - COVID syndrome, damage is primarily manifested by secondary sclerotic changes in parenchymal organs, which plays an important role in determining the mechanisms of sanogenesis and thanatogenesis, as confirmed by the data presented above. In the CIS countries, the concept of post-COVID syndrome and the associated secondary changes were first described by scientists of the Russian Federation starting from May 2020 as "a sharp increase in sparse and coarse fibrous structures between tissues and blood vessels of vital organs, accompanied by a

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significant decline in the morphofunctional indicators of organs…" After COVID-19, physicians set the following tasks:

to study the morphological features of the lungs in post-COVID syndrome;
to investigate the morphometric parameters of lung tissue in post-COVID syndrome;
to examine and evaluate changes in lung tissue using immunohistochemical analysis in post-COVID syndrome;
to assess the lungs based on morphological, immunohistochemical, and morphometric characteristics in post-COVID syndrome and to develop a clinical–morphological diagnostic algorithm.

By developing a clinical–morphological diagnostic algorithm, it becomes possible to characterize patient rehabilitation in post-COVID syndrome and to provide recommendations based on clearly defined morphofunctional criteria. This also enables the prospective assessment of sanogenesis and thanatogenesis mechanisms that may arise in post-COVID syndrome, as well as the formulation of recommendations for appropriate therapeutic interventions to counteract them.

Potential mechanisms contributing to long-term persistence of COVID-19 include virus-related pathophysiological changes (such as microcoagulation), immune and inflammatory dysregulation resulting from acute infection, and specific consequences observed in patients with severe disease (post-intensive care syndrome). An issue of The Lancet Respiratory Medicine dedicated to the long-term effects of COVID-19 discusses data on the diagnosis, treatment, and management of post-COVID syndrome, with a focus on the respiratory, neurocognitive, psychological, and systemic consequences of severe acute COVID-19. Known risk factors for post-COVID syndrome include severe acute illness, comorbidities (such as diabetes mellitus and chronic heart failure), and life-saving interventions such as invasive mechanical ventilation of the lungs. It has been reported that about 30% of COVID-19 survivors experience weakness after hospitalization. One of the latent features of post-COVID syndrome is that it can affect individuals regardless of the severity of the initial illness. Studies have shown that post-COVID syndrome can also occur in patients with mild to moderate disease, including younger individuals who did not require respiratory support, hospitalization, or intensive care.

Post-COVID syndrome may also develop in patients who tested negative for SARS-CoV-2 but had previously been discharged from the hospital, as well as in those receiving outpatient treatment. Another concerning aspect is that post-COVID syndrome can affect children, including those who had asymptomatic COVID-19. This condition may manifest with symptoms such as shortness of breath, fatigue, myalgia, cognitive impairment, headache, palpitations, and chest pain, which can persist for at least six months. In lung tissue, various types of focal and diffuse fibroproliferative processes (determined by the extent of involvement during the exudative phase) can lead to the development of focal pneumofibrosis. Exudative pneumonia or pneumonitis, as a rule, includes the frequent formation of erythrocyte thrombi, especially in the interalveolar capillaries. This process is accompanied by proliferation of myofibroblasts, hyperplasia and metaplasia of the bronchoalveolar epithelium, fibro-cystic remodeling, and the formation of secondary foci of emphysema. In patients with post-COVID interstitial lung injury, the development of pulmonary fibrosis is observed, along with respiratory failure and symptoms such as shortness of breath, cough, and sputum production. These manifestations may also be accompanied by dysfunction of other organs.

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Myroshnychenko M.S. and Pasiyeshvili N.M. (2023) examined the morphological features of the lungs in post-COVID syndrome. Autopsy materials from 96 cadavers (59 males and 37 females) were used, specifically lung tissue samples. According to medical history, all patients had varying degrees of COVID-19 infection prior to death, and after treatment of the infection they exhibited different degrees of respiratory failure. The average duration of the post-COVID period was 148.6 ± 9.5 days. Based on the severity of COVID-19 in the medical history, all cases were divided into three groups. Group 1 included 39 patients with mild COVID-19. Group 2 included 24 patients with moderate COVID-19. Group 3 included 33 patients with severe COVID-19. Histological, histochemical, morphometric, and statistical research methods were used in the study. It was concluded that morphological changes in the lungs in post-COVID syndrome may include the development of pneumosclerosis, local and diffuse immune cell infiltration, emphysematous and atelectatic changes, alterative changes in the alveolar epithelium, metaplastic changes in connective tissue, dystrophic calcification, dystrophic, metaplastic, and dysplastic changes in the epithelial layer of the bronchial tree, as well as hemodynamic disturbances.

Pneumosclerosis, focal–diffuse infiltration of immune cells, alterative changes in the alveolar epithelium, emphysematous and atelectatic changes, as well as hemodynamic disturbances, tend to accelerate with the progression of COVID-19. Metaplastic changes in connective tissue, dystrophic calcinosis, and dystrophic, metaplastic, and dysplastic changes in the epithelial layer of the bronchial tree are not dependent on the severity of the infection. The changes identified by the authors help to explain the pulmonary manifestations of post-COVID syndrome. They should form the basis for raising clinical suspicion of neoplastic processes among physicians and for developing rehabilitation and therapeutic measures for this category of patients. Pulmonary fibrosis is an interstitial lung disease characterized by progressive scarring of lung tissue, which affects lung function and leads to impaired gas exchange and breathing difficulties. Currently, the incidence of pulmonary fibrosis is significantly increasing. The development of pulmonary fibrosis is associated with multiple risk factors, such as aging, smoking, genetic predisposition, and occupational exposure to dust and asbestos. Due to the lack of effective treatment methods capable of halting disease progression, patients with pulmonary fibrosis face an increased risk of mortality.

COVID-19 can lead to atypical pneumonia, which may result in acute lung injury and acute respiratory distress syndrome (ARDS). Clinical manifestations associated with COVID-19 can range from mild upper respiratory tract involvement to severe ARDS requiring long-term oxygen therapy due to pulmonary fibrosis. In COVID-19 patients who develop pulmonary fibrosis, the risk of death increases, as pulmonary fibrosis is a progressive disease that leads to respiratory failure and is associated with a poor prognosis. Lung transplantation remains the only treatment option shown to improve outcomes. Life-threatening complications of COVID-19 are associated with endothelial cell dysfunction, which contributes to microcirculatory disorders and the development of complications such as venous thromboembolic disease and multi-organ damage. Modern methods for detecting pulmonary fibrosis include histological examination of tissue samples and radiological imaging using computed tomography. Based on histological analysis, features such as fibrotic patterns, collagen area fraction, and cellular localization can be observed. Collagen zone fractions assessed in histopathological images show a strong correlation with histological indicators of fibrosis and provide a reliable index for quantifying the degree of fibrosis. Researchers used collagen area fractions obtained from histological analysis to compare

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the simulation results of the developed model. An open-source platform was used for multidimensional spatiotemporal modeling of epithelial tissue, viral infection, cellular immune response, and tissue damage, specifically designed to be modular and extensible to support continuous updates and parallel development. Simplified modeling of epithelial tissue regions and immune responses demonstrates realistic patterns of infection dynamics, from widespread infection to clearance. Slower viral internalization combined with faster accumulation of immune cells slows down the infection and helps contain it. Since antiviral drugs may have adverse effects and may reduce clinical efficacy when administered later during infection, we examined how treatment effectiveness depends on disease progression and the timing of the first therapeutic intervention after infection.

In simulations, even low-intensity therapy using drugs that reduce the rate of viral RNA replication significantly decreased overall tissue damage and viral load when administered early in infection. Various combinations of dosage and treatment timing resulted in cytotoxic outcomes: some simulation runs showed viral clearance or control (treatment success), while others demonstrated rapid infection of all epithelial cells (treatment failure). Thus, although high-intensity therapy is generally ineffective when applied late, delayed treatment can still be effective in certain cases. Life-threatening complications of COVID-19 are associated with endothelial cell dysfunction, which contributes to microcirculatory disturbances and the development of complications such as venous thromboembolic disease and multi-organ damage. Modern methods for detecting pulmonary fibrosis include histological examination of tissue samples and radiological imaging using computed tomography. Based on histological analysis, features such as fibrotic patterns, collagen area fraction, and cellular localization can be observed. Collagen zone fractions assessed in histopathological images show a strong correlation with histological indicators of fibrosis and provide a reliable index for quantifying the extent of fibrosis. Known risk factors for post-COVID syndrome include severe acute illness, comorbidities (such as diabetes mellitus and chronic heart failure), and life-saving interventions such as invasive mechanical ventilation of the lungs.

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