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IMMUNOHISTOCHEMICAL EXAMINATION OF LUNG TISSUE CHANGES IN POST-COVID SYNDROME

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Abstract:
As material for the article, a number of cases were taken in the Department of Adult Pathology and Biopsy Diagnosis of the Republican Center for Pathological Anatomy during 2020-2022, and the diagnosis of coronavirus infection was confirmed on the basis of clinical and morphological data. An analysis of clinical and laboratory data of patients who died of COVID-19 was obtained.

Keywords: dysfunction, pulmonary, histopathological, indicator, factors, epithelial, immune, histological analysis, cytotoxic results, histological, microscopic,

As a result of the development of life-threatening complications of COVID-19, endothelial cell dysfunction, blood microcirculatory dysfunction, which contributes to the development of complications such as venous thromboembolic disease and multiorgan damage. Modern methods of determining pulmonary fibrosis include histological examination of tissue samples and radiological examination with the help of computer tomography. According to the results of histological analysis, the appearance of fibrosis, collagen area fraction and cell localization can be observed. Collagen zone fractions assessed on histopathological images are strongly correlated with histological indices of fibrosis and provide a reliable index to quantify fibrosis [36]. Immunohistochemical studies show that T-cell immune responses predominate in COVID-19 interstitial pneumonia. A sharp decrease in the total number of T-lymphocytes, the absence of CD20+ V-lymphocytes, CD 57+ NK-cells in the fibrotic stage is an indicator of the progressive suppression of immunological reactivity, while the increased expression of CD68 and CD 31 (PECAM-1) in macrophages indicates an unfavorable prognosis. One of the important factors

contributing to the development of fibrosis is the activation of latent TGF-β due to tissue damage.

Getz M, Wang Y, An G (2021) Developed a multifunctional lung tissue simulator that can be used to study intracellular viral replication mechanisms, epithelial cell infection, stem cell immune response, and tissue injury. The authors focused on the mechanisms of the fibrotic process in lung tissue during SARS-CoV-2 infection. In silico experiments were performed to determine the effects of the chaotic behavior of TGF-β sources (stationary and mobile), the rate of TGF-β activation from the sources, and the duration of activation of TGF-β sources on development and progression. The scientists used collagen sphere fractions from histological analysis to compare the simulation results of the developed model. An open source platform was used for multidimensional spatio-temporal modeling of epithelial tissue, viral infection, cellular immune response, and tissue damage, specifically designed to be modular and scalable to support continuous innovation and parallel development. Basic modeling of a simplified field of epithelial tissue and the immune response shows clear patterns of infection dynamics, ranging from widespread infection to reduction and clearance. Slower internalization of the virus and faster recruitment of immune cells slows down the infection and helps contain it. Because antiviral drugs can have side effects and reduce clinical efficacy when administered later in the course of infection, we examined the effect of treatment efficacy on progression and time to first treatment after infection. In simulations, even when low-potency therapy with a drug that reduces the rate of viral RNA replication was given early in infection, gross tissue damage significantly reduced this viral load. Many combinations of dosage and treatment time produced cytotoxic results, with some simulation replicates showing clearance or control (treatment success) and others showing rapid infection of all epithelial cells (treatment failure). Thus, although high-potency therapy is generally ineffective when administered later, later treatment is sometimes effective.

Zarubin E.A. and all (2023) noted that the manifestations of lung injury can be independent and develop separately from each other. Viral proteins are found in the same cells at different frequencies and intensities in different forms of lung injury, which may indicate possible common mechanisms of lung injury in COVID-19 and

the different ways the virus enters lung tissue. Histological-microscopic examination of lung tissue revealed the following pathological processes: diffuse alveolar damage, lymphocytic alveolitis, focal or croupous pneumonia with the addition of secondary flora.

Alveolar type II pneumocytes, macrophages and endothelial cells play the main role in the pathogenesis of coronavirus infection. Staining of these cells in different forms of infection with viral particles can reveal mechanisms of mutual pathogenesis. The detection of cellular proteins in the cells described above in the late stages of the disease can be a potential cause of the development of post-covid syndrome. Taking into account the dynamics of tissue differentiation and infection, using a stepwise approach that integrates various experimental data, the authors developed a cell model adapted to study the dynamics of infection and regeneration in a pseudostratified NAO culture. In addition, a new method for determining tissue integrity based on image data combined with standard transepithelial electrical resistance measurements is presented. This analysis represents the first goal of elucidating cell type-specific infection kinetics and shows how changes in tissue composition and regenerative capacity may affect disease progression and pathology in, for example, smokers. Furthermore, we identified key indicators that still need to be evaluated to improve inferences about cell type-specific infection kinetics and disease progression. This approach, combined with additional experimental data, provides a method that can be used to dissect the complex dynamics of viral infection and immunity in human airway epithelial systems.

Potential mechanisms by which prolonged viral persistence contributes to COVID include virus-related pathophysiological changes (eg, microcoagulation), immunological and inflammatory dysregulation resulting from acute infection, and certain disease sequelae in critically ill patients (post-intensive care syndrome). The Lancet Respiratory Medicine issue on acute outcomes of COVID-19 discusses the diagnosis, treatment, and management of post-covid syndrome, focusing on the respiratory, neurocognitive, psychological, and systemic consequences of severe acute COVID-19.

Known risk factors for post-Covid syndrome include severe acute illness, co-morbidities (such as diabetes and chronic heart failure), and critical life-saving

treatments such as invasive mechanical ventilation. It has been reported that 30% of survivors of COVID-19 experience fatigue after hospitalization. The authors describe the complex interrelationship between pre-existing comorbidities, frailty, socioeconomic factors, intensive care, and post-Covid syndrome outcomes. This interaction is important for models of care that include careful assessment of physical and psychological function after hospital discharge, as well as consideration of nutrition, medication, and social support. Coordinated multi-profile medical care for people at risk of post-Covid syndrome senile asthenia, as well as further research on vulnerable groups is needed. The protective effect of the COVID-19 vaccine against post-covid syndrome was analyzed by Japanese scientists.

In the pre- and post-Covid period, a rehabilitation method using controlled hypo-hyperoxic training of the lungs was proposed. It has been proven that the oxygen distribution function of the blood and the efficient functioning of the mitochondrial apparatus of the cells during the performance of the maximum intensity load with the low pulse tension of the lungs and systemic hemodynamics. The method of breathing exercises based on compensating the oxygen demand of the body cells to activate the recovery functions has been effective for the rehabilitation of patients with advanced lung failure after pneumonia of various causes, including this method is effective in the case of COVID-19. In this case, due to the elimination of bronchospasms, the permeability of the bronchi improves, the production of surfactants by alveolar cells increases, which prevents the occurrence of atelectasis in the lungs and improves the activity of the ciliated epithelium of the bronchi. Increasing the oxygen capacity of the blood optimizes the activity of the endothelium of pulmonary vessels, processes of oxygen utilization in mitochondria, improves the contractile function of the myocardium, and extends the threshold for the occurrence of arrhythmia. Specific pulmonary complications, including pulmonary fibrosis and thromboembolic disease, require careful evaluation and may require specialized investigations and treatment. Complications of COVID-19 in people with pre-existing respiratory conditions vary depending on the nature, severity, and control of the respiratory disease. Extrapulmonary complications, such as decreased exercise capacity and weakness, can cause shortness of breath after COVID-19. Nonpharmacologic treatments, including tailored pulmonary rehabilitation

programs and respiratory management techniques through physical therapy, can help relieve respiratory distress in patients after COVID-19. The study of the pathogenesis and morphogenesis of the post-covid syndrome of the COVID-19 coronavirus infection is currently an urgent issue, which requires the collection of factual data, their processing and analysis. Understanding the mechanisms of disease development and their characteristics increases the quality of diagnosis and the effectiveness of treatment, as well as reduces mortality. The spread of cases following COVID-19 is significant, and the health effects of COVID-19 are likely to be long-lasting and pose a variety of challenges to the health care system.

In 2020-2022, the Department of Adult Pathology and Biopsy Diagnostics of the Republican Center for Pathological Anatomy received 63 cases where the diagnosis of coronavirus infection was confirmed on the basis of clinical and morphological data. The following methods were used to carry out the research: analysis of clinical and laboratory data of patients who died of COVID-19; conducting a histological examination of lung tissue using hematoxylin-eosin dye; examination of lung tissue by immunohistochemical method; calculation of relative sizes of all morphofunctional areas of lung tissue by histometry method. The aim of this article was to determine the morphological and morphometric changes that develop in the main morphofunctional areas of the lung tissue during coronavirus infection. In this study, the main cause of death was pulmonary edema, septic shock, polyorgan failure. In all cases, the background disease is characterized by the morphofunctional impairment of immunocompetent cells developed against the background of COVID-19.

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