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Morphological Characteristics of Post-Covid Pulmonary Fibrosis

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Abstract: As the pandemic progresses, effective vaccines and new treatments are developed, a deeper understanding of late complications in patients who experience SARS-CoV-2 infection is becoming increasingly important. Due to the prevalence of respiratory failure in critically ill patients and the need for mechanical ventilation, there is growing concern about long-term pulmonary complications, primarily pulmonary fibrosis (PF).

Key words: coronavirus, pandemic, lung, fibrosis, alveolus.

Relevance. The first reports of the novel coronavirus SARS-CoV-2 spread from Wuhan, China, in December 2019. The highly transmissible virus has spread rapidly across the planet, rapidly depleting medical resources and becoming a leading cause of disease and death worldwide. At the same time, lung cancer remains the leading cause of death worldwide, especially in Russia, from new products of poor quality [3]. In the emerging context, it is critical to ensure that oncology care is error-free and that the highest standards of OS diagnosis and treatment are maintained [16]. As the pandemic progresses, effective vaccines and new treatments are developed, a deeper understanding of late complications in patients who experience SARS-CoV-2 infection is becoming increasingly important. Due to the prevalence of respiratory failure in critically ill patients and the need for artificial lung ventilation, there is growing concern about long-term pulmonary complications, primarily pulmonary fibrosis (PF) [7].

Given that survivors of COVID-19 with persistent lung damage will require long-term specialized care, including oncology, physicians from various specialties are interested in understanding and addressing the various risk factors associated with postcoidal pneumofibrosis (PPF).

Interstitial lung disease (ILD) is a term that unites various diffuse parenchymatous diseases in the lungs with a certain spectrum of clinical, radiological and pathological features. The term IO'K is characterized by common signs of inflammation and fibrosis [9]. Pneumofibrosis (PF) is a morphological outcome of severe and chronic diseases that disrupt the normal control of tissue regeneration in the lungs [8]. PF is considered a subgroup of IO'K. It is characterized by impaired regeneration of damaged alveolar epithelium, proliferation of fibroblasts, excessive accumulation of extracellular matrix components, especially collagen, and disruption of normal structures in the lungs [7].

The morphological term "fibrosis" usually refers to the accumulation of collagen [9]. Studies in humans and animal models show that myofibroblast proliferation is involved in alveolar remodeling [19]. However, the morphological progression of postcoidal PF has not been sufficiently studied. To clarify some related terms, diffuse alveolar damage is not considered fibrosis per se, but fibrotic changes may form part of it. In the fibrosis phase of diffuse alveolar injury, there is insufficient clearance of alveolar collagen, which is degraded in the early stages of the lung injury process. "Gross glass" is a radiographic sign of inflammation that is more likely to be reversible in pneumonia than fibrosis [12].

Idiopathic pulmonary fibrosis (OIF) is a special type of fibrosis IO'K. Viral infections, in particular, are associated with the development of OIF, as a factor involved in the onset of the disease, but OIF does not have a known trigger for detection [5]. A meta-analysis showed that the presence of persistent or chronic viral infections significantly increases the risk of developing OIF, but the available data did not address the role of coronaviruses [18].

Clarification and introduction of special terminology is required to avoid confusion. The term "postcoid pulmonary fibrosis" (PCO'F) should be used interchangeably with terms such as "post-inflammatory PF", "PF after acute respiratory distress syndrome", "postviral PF" or "postviral IO'K" [9]. The etiology, progression, and prognosis of postviral PF are generally different from those of OIF. The term PKPF is used to designate the nonidiopathic form of PF associated with SARS-CoV-2 infection, where the COVID-19 pandemic has not yet been concluded and many aspects of this condition remain uncertain. PKPF diagnosis should be based on clinical, radiological and pathological data.

In patients who have experienced COVID-19 or are suspected of having SARS-CoV-2 infection, laboratory tests, respiratory function studies, and high-resolution computed tomography (CT) can provide evidence to support the diagnosis of PKPF. In pandemic conditions, invasive procedures such as bronchoscopy or surgery for lung biopsy cannot be justified, as their results would not have changed patient management tactics [6; 21].

To date, there are no reliable data on the frequency and severity of PF associated with COVID-19. A recent study described patients with residual radiographic changes consistent with pulmonary fibrosis [20]. Most of the 90 patients hospitalized with COVID-19 had mild or significant residual lung changes on CT performed on day 24 after symptom onset [27]. Some authors have radiographically diagnosed PKPF on the basis of extensive and persistent fibrotic changes, including parenchymatous cysts, mesh shadows, and tractional bronchiectasis with or without cellularity. Subsequent CT scans were noted to show extensive fibrosis [9]. As there is no single test that confirms the diagnosis, it is important that radiographic changes are accurately recorded over time, confirming the association with recently experienced COVID-19.

There are certain differences in the symptoms and severity of PKPF, but in most cases, a double-sided picture of frosted glass is observed, which later changes to fibrosis. In a study of 131 lung samples from patients with COVID-19, three histological patterns of lung damage, often hidden, were identified: epithelial, vascular, and fibrosis [16]. At autopsy, a fibrotic pattern of diffuse alveolar damage was often observed, which is usually manifested by either fibrosis of the alveolar stream or diffuse thickening of the alveolar wall. Potential causes of PKPF include viral pneumonia [7], acute respiratory distress syndrome (ARDS), and sepsis associated with COVID-19 [15; 11], thromboembolism, hyperoxia, impaired immune responses and lung damage due to long-term OSV. The listed causes are hidden, and some, for example, lung damage from OSV, are not considered a necessary condition for the development of PKPF.

In the 21st century, three global outbreaks of viral pneumonia have been reported before COVID-19: the SARS (SARS-CoV) coronavirus in 2002, influenza A H1N1 in 2009, and MERS-CoV in recent 2012. From 2002 to 2004, SARS-CoV led to more than 8,000 hospitalizations, with one in five developing fatal ARDS, an increase of over 9%. In 2009, the influenza A H1N1 pandemic resulted in 31% of intensive care unit admissions in the adult population and 14–27% of critically ill patients dying, with rates particularly high among patients requiring intensive care [17]. In the case of MERS-CoV, ICU mortality was reported to be 58–60% and 72–75% among those requiring OSV [19]. The death rates from both MERS-CoV and COVID-19 are alarming. But the consequences of COVID-19 are serious, given the dramatic difference in the spread of the disease.

Fibrosis was found in 33% of patients with MERS-CoV who were screened for residual lung radiographic changes after recovery. Fibrous changes have been associated with severe radiographic findings on chest radiographs, longer intensive care unit stays, longer OSV duration, older patient age, and higher levels of lactate dehydrogenase [14].

A lack of data on the problem of pulmonary complications after recovery from COVID-19 is evident. In one of the studies that examined surviving patients 8-12 weeks after the diagnosis of COVID-19, objective deviations in the state of health were found in 35% of patients. Symptomatics were noted mainly in patients undergoing oxygen therapy. At the same time, most patients suffer from complaints such as shortness of breath and cough before the targeted examination [30].

Today, the number of people infected with the new coronavirus infection (NWI), which spread from China (Wuhan) in December 2019, is more than 20 million worldwide, and the number of fatal outcomes is > 4 million.

In patients with a severe form of IAKI, 6 months after the end of inpatient treatment, it was determined that the lung functional state remained impaired, and high-quality computed tomography data showed pathological changes in the lungs, some of them with fibrotic changes. The pathomorphological characteristics of the development of AKI, as well as the ability of the virus to activate the connective tissue growth factor and increase the signaling of the transforming growth factor β, can lead to the formation of fibrosis. An increase in the titer of antinuclear and specialized autoantibodies indicates a violation of the control of the immune response in AKI, which leads to the acceleration of the emerging pneumonia and the development of fibrotic changes in the lung tissue. There is still no consensus among researchers about the clinical significance and further prognosis of AKI, which is the reason for its further study [18].

Analysis of scientific works on the results of research conducted during the COVID-19 pandemic indicates the risk of lung tissue fibrosis or the risk of exacerbation of existing interstitial disease with the development of pulmonary fibrosis in patients infected with the SARS-CoV-2 virus.

A fibrotic histological pattern was noted in approximately 22% of COVID-19 cases, beginning at week 3 of illness. The molecular basis of accelerated pulmonary fibrosis after SARS-CoV-2 infection remains unclear, but it is believed to be multifactorial, involving direct viral effects, immune dysregulation, cytokine storm, and increased oxidative stress. Questions such as why fibroblasts and myofibroblasts accumulate in a certain proportion of patients, transition to uncontrolled cell proliferation with excessive accumulation of collagen and extracellular matrix, what leads to rapid fibrosis of the lungs, severe course of the disease, and what increases the risk of severe complications and death are answered. requires. An important area of research is the search for biomarkers for the early identification of patients at high risk of severe COVID-19, complicated by the development of interstitial fibrosis lung damage [16].

Pulmonary fibrosis is a pathological process characterized by the replacement of normal lung tissue by fibrous (scar) tissue. As a result of such changes, serious changes occur in the respiratory system. The development of fibrosis leads to a decrease in the volume of functional lung tissue, as a result of which the efficiency of ventilation and breathing decreases. Gastrointestinal fibrosis is associated with mucosal thickening and motility disorders and includes meconium obstruction, constipation, distal bowel obstruction syndrome, gastroesophageal reflux disease, and small intestinal bacterial overgrowth. Distal intestinal obstruction syndrome is caused by dense intestinal contents that completely or partially block the passage of the small intestine, often at the ileocecal junction. This is thought to be related to an intestinal inflammatory cascade when a defect in transmembrane permeability control occurs [18].

Fibrosis is a widespread pathological process that damages almost every organ and has poor therapeutic efficacy. It leads to end-stage organ failure and aggravates dysfunction, and is the leading cause of death worldwide [4]. Fibrosis is a pathological overgrowth of extracellular matrix (ECM) with several molecular components, particularly collagens, glycoproteins, and proteoglycans. HTM maintains a homeostatic balance of synthesis and degradation through physiologically complex regulatory pathways, leading to wound healing under healthy conditions and tissue regeneration after injury or inflammation [1]. One of the signs of fibrosis is excessive shedding of the extracellular matrix [18]. However, excessive inflammation or overstimulation by pathological environmental factors activates mesenchymal cells, such as myofibroblasts and smooth muscle cells, to proliferate continuously. These cells are characterized by the secretion of HTM, and their abnormal increase leads to fibrosis.

In addition, nonmesenchymal cells such as fibrocytes, endothelial cells, and epithelial cells can transform into fibroblasts when stimulated [16]. Innate and adaptive immune responses are central to overcoming causative agents and their effects on fibrosis. The immune system represents a very

complex network of different cells in which molecules such as cytokines, chemokines, growth factors, angiogenic factors, and reactive oxygen species (ROS) play a role in intercellular communication. Activation of the immune system by heterogeneous stimuli can directly or indirectly activate mesenchymal and non-mesenchymal cells by modulating inflammation [13].

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