

Morphological Changes in Regional Lymph Nodes in Pulmonary Pneumosclerosis

Khaidarova Nargiza Akhtamovna
Bukhara State Medical Institute

Summary: Pulmonary fibrosis is a pathological process in the lungs characterized by refractoriness to therapy and high mortality rates, it represents a heterogeneous group of disorders with progressive and irreversible destruction of lung architectonics due to scarring, which ultimately leads to organ dysfunction, gas exchange disorders and death due to respiratory failure. [D.V. Bestaev, 2014]. But changes in the lymph nodes during the fibrous process of the lung tissue are poorly understood. The article presents a review of the literature on structural changes in regional lymph nodes that occur in experimental pulmonary fibrosis.

Keywords: pulmonary fibrosis, regional lymph nodes, experiment.

Relevance. The novel coronavirus disease 2019 (COVID-19) pandemic, caused by the SARS-CoV-2 coronavirus, has generated serious concern among the global community due to its rapid spread, high mortality rate and lack of specific and effective treatment (in the first 4 months after the report of outbreak of the disease, about 3 million people became infected with SARS-CoV2 and 200,000 died) [WHO Bull, 2020].

Like previous strains (SARS-CoV and MERS-CoV) of the coronavirus family, SARS-CoV2 primarily affects the respiratory system, causing lower respiratory tract infection and serious complications in the lungs, including acute respiratory distress syndrome (ARDS). [Aitbaev K. A., 2021], which is the main cause of death in patients with coronavirus. As for patients who survive ARDS, a significant proportion will have long-term impairment of pulmonary function due to the development of fibrosis. It is important to note that pulmonary fibrosis is a well-recognized consequence of ARDS and, as has been shown in several studies, the so-called “protective ventilation” of the lungs reduced radiological abnormalities after ARDS [Burnham E. L., et.all., 2014].

The pathological correlate of ARDS is diffuse alveolar injury (DAI), which is characterized by an initial (acute inflammatory) exudative phase with edema, hyaline membranes and acute interstitial inflammation, followed by an organizing phase with loose organizing fibrosis, mainly within the alveolar septa and type II pneumocyte hyperplasia [Cardinal-Fernández P., et.all., 2017]. The powerful third and final stage of ARDS may be the fibrotic phase. Thille et al described a cohort of 159 autopsies of patients with ARDS, showing that these pathological findings can either resolve to normal lung parenchyma or progress to fibrosis [Thille A.W., et.all., 2013]. In this study, 4% of patients with disease duration of less than 1 week, 24% of patients with disease duration of 1 to 3 weeks, and 61% of patients with disease duration of more than 3 weeks developed fibrosis. This description, together with additional data, confirms that pulmonary fibrosis begins early in the development of ARDS [Burnham E. L., et.all., 2014]. It is important to note that there is evidence that progressive pulmonary fibrosis may be the cause of mortality in many patients with ARDS, while a significant proportion of survivors will have long-term pulmonary dysfunction and radiological abnormalities suggestive of pulmonary fibrosis. At the same time, the degree of reticulation on computed tomography clearly correlates with indicators of quality of life and pulmonary function, which indicates a deterioration in indicators such as forced vital capacity and lung diffusion capacity [Burnham E. L., 2013].

Pneumosclerosis (pneumosclerosis; Greek pneumon lung + sklerosis compaction; synonyms: pneumofibrosis, pulmonary sclerosis) is the proliferation of connective tissue of the lungs due to an inflammatory or dystrophic process, leading to impaired elasticity and gas exchange function of the

affected areas. Traditionally, it is indicated that a constant sign of diffuse pneumosclerosis is shortness of breath, which often tends to progress. Changes in ventilation are characterized by restrictive breathing disorders.

I.V. Davydovsky pointed out that with pneumosclerosis, scar fields in the lung often undergo smooth muscle transformations (“muscular cirrhosis”) due to constant respiratory excursions of the organ.

A number of scientists, under “pneumosclerosis,” describe chronic cicatricial and degenerative changes that develop both in the alveoli and in the interstitial tissue between the lobules, along the bronchi, in the walls of the bronchi themselves [S.O. Neville, 1963]. In this case, the blood vessels of the lungs, nerve trunks and lymphatic vessels are involved in the process. Moreover, pneumosclerosis does not represent an independent nosological form.

A serious consequence of the acute course of COVID-19 infection, which has acquired pandemic status, is the development of interstitial pneumonia, which can result in fibrosis and pneumosclerosis of the lungs. Analysis of patients with COVID-19 at their discharge from the hospital indicates a high incidence of pulmonary dysfunction due to fibrosis and pneumosclerosis. According to the literature data of Russian scientists, 47% of patients have disturbances in the gas exchange mechanism, 25% have a decrease in the function of the total vital capacity of the lungs, which disrupts the vital functions and viability of the body.

These facts express the relevance of the problem of fibrosis and pulmonary pneumosclerosis. During the pandemic, the problem of pneumosclerosis has become the most pressing, and its pharmacotherapy has taken center stage in the field of view of scientists around the world.

The lymphatic bed transports tissue fluid from the lesion into the blood and can be considered as an organ that ensures the constancy of the volume of plasma and interstitial fluid [Kozlov V.I., 2005; Sapin M.R. et al., 2007; Banin V.V., 2014]. The lymphatic system is structured as a chain of intervalvular segments with different wall structures; it organizes a special outflow path from the organs (collateral drainage to the veins) of tissue fluid in the form of lymph, and in its composition - antigens. The lymphoid system looks like a special attachment to the cardiovascular system: lymphoid couplings of varying structural complexity surround tissue channels and vessels as their biofilter attachments, regulating the cellular and protein composition of the internal environment of the body. At the heart of the lymphoid system are blood vessels closed in a circle through which lymphocytes (re)circulate. The lymphatic bed drains lymphoid formations and brings lymph to some of them for cleansing. The lymphoid and lymphatic systems are united at the periphery into an immunoprotective complex: the lymphatic bed and lymphoid tissue around the blood microvessels cooperate to ensure the genotypic homeostasis of the body and constitute the lymphoid-lymphatic apparatus as part of the cardiovascular system. The lymphatic system is involved in the organization of immunity in humans and animals, since the lymphatic bed carries out the influx of antigens into the lymph nodes and simpler lymphoid formations (lymphoid nodules and plaques) with afferent lymphatic pathways.

Each inflammatory process in the lungs is reflected in the lymph nodes, sometimes only in the regional ones, sometimes in the extrathoracic and distant ones. Their reaction to lung infection in the form of enlargement in children is much stronger and more frequent than in adults, and any inflammatory process in the lungs may be followed by an increase in regional nodes.

A study of the available literature data revealed a lack of information about the effect of pulmonary fibrosis on the structural and morphological state of the tissue of regional lymph nodes.

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