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PATHOMORPHOLOGICAL CHANGES IN THE LYMPH NODES OF PATIENTS WHO DIED FROM COVID-19 DISEASE.

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Annotation. In Covid-19, specific morphological changes in immune organs are accompanied by varying degrees of morphological changes depending on the nature of the pathogen and the general reactivity of the body. In any disease of the same viral nature, different subpopulations of T-lymphocytes (CD 3+,4+,carrying antigen) or B-lymphocytes (CD 20+, 23+) on the part of immune cells of varying degrees, a change in antigen-bearing populations, manifested by quantitative and qualitative changes. Changes in the immune organs are characterized by the accumulation of lymphocytes at different levels or the transition with metaplastic changes in parenchymal elements.

Key words: spleen, thymus, lymphocytes, lymphoid follicle, immune organs, immunohistochemistry.

In COVID-19 (SARS-CoV-2), the disease was mainly studied as presenting in the form of severe respiratory distress syndrome. Interaction of SARS-CoV-2 with target cells leads to dysfunction of immune cells [1]. As a result, the disease manifests with rapid progression. Consequently, widespread cell necrosis is observed, and excessive production of cytokines by immune cells (cytokine storm) leads to a systemic response involving all vascular components [2,3,4]. Clinically, these changes are manifested by severe vasodilation, a drop in blood pressure, development of shock of varying degrees, and ultimately sepsis, which leads to death in 28% of cases [5,6]. These vasoparalytic changes are characterized by severe impairment of perfusion in the liver, heart, lungs, and kidneys, as well as immune organs, accompanied by a deficiency of immune cells.

The SARS-CoV-2 pathogen mainly damages angiotensin-converting enzyme 2 (ACE-2) receptors, increases viral replicative activity, and induces pyroptosis in host cells (involving leukotrienes, CRP, AAB, MRSA, and others), which is manifested by an intensification of the inflammatory process. During pyroptosis, intermediate products stimulate neighboring cells—epithelial cells, endothelial cells, alveolar macrophages, mast cells—to massively release pro-inflammatory cytokines (IL-6, IL-8, MIP1α, MIP1β, MCP1). This, in turn, attracts white blood cells within the vascular lumen, including monocytes, T-lymphocytes, macrophages, and plasma cells, leading to a predominantly hyperergic type of inflammatory reactivity [7,8].

As a result, the disease has a very severe clinical course and is manifested by a marked disruption of blood circulation at the capillary level in vital organs such as the lungs, heart, kidneys, pancreas, adrenal glands, and immune organs, leading to multiple organ failure. The high mortality rate and the need to further improve clinical effectiveness in restoring circulation during the peak stages of the disease make the study of these changes highly relevant at present. In this context, our work focuses on examining the specific general and local morphological

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changes in the spleen tissue, which is considered one of the immune organs [9]. The study aimed to investigate morphological changes in the spleen tissue of patients who died from COVID-19 based on autopsy findings and to develop practical recommendations for their application. The spleen tissues of patients who died with a diagnosis of COVID-19 during 2020–2021 in the practice of the Republican Pathoanatomical Medical Center of the Ministry of Health of the Republic of Uzbekistan were studied using macroscopic, microscopic, and immunohistochemical methods. Clinical and anamnestic data were analyzed based on medical records and autopsy reports. Histological sections were prepared from paraffin-embedded blocks of spleen tissue obtained during autopsy and stained with hematoxylin and eosin. The histological preparations were examined under a trinocular light microscope, and microphotographs were taken from selected areas.

The results of the study showed that when lymph node changes were examined using hematoxylin and eosin staining, no specific or characteristic alterations were significantly observed in the lymph nodes; instead, changes typical of general viral diseases were identified. In particular, the lymph node capsule appeared congested, while the subcapsular spaces demonstrated mainly cystic dilated foci and marked proliferation of reticulocytes (see Figure 1). The cortical region was characterized by pronounced proliferation of B-lymphocytes, hypermutation of B-lymphocytes under the influence of the SARS-CoV-2 pathogen, and the presence of foci of monocellular cytolysis. Around these cytolyzed plasma cells, cellular debris was observed, along with prominent macrophage infiltration foci in the surrounding areas. In our study, accumulation of interdigitating cells around B-lymphocytes was noted, as well as marked antigen-dependent lymphocyte proliferation. The accumulation of CD4+ and CD5+ a ntigen-positive lymphocytes in the marginal zones of lymphoid follicles was also identified.

In the paracortical zones, most T-lymphocytes following a crown-like trajectory showed a relatively reduced antigen-dependent proliferation. Widening of the interstitial spaces indicated damage to the cellular immune system and a decrease in immature CD20+ and CD25+ marker-positive lymphocytes. These areas were also characterized by the accumulation of numerous macrophages and reticulocytes, as well as the proliferation of loose fibrous connective tissue. Variable degrees of congestion in postcapillary venules within the paracortical region, along with a defective appearance of the highly membranous endothelial cell walls, indicate impaired transendothelial migration of T-lymphocytes. The proliferation of pericytes around the endothelium of most postcapillary venules, along with a marked increase in loose fibrous connective tissue components, may serve as evidence of a significant reduction in cellular immune response due to inhibition of vascular wall permeability and impaired migration of antigen-dependent lymphocytes. At the same time, pronounced denudation of the perimedullary reticular processes in the medullary cords of the lymph node, together with a decrease in lymphocyte proliferation within the parenchyma of the lymph node, was found to be associated with disrupted feedback regulation and the development of a reticulosis-like process (see Figure 2). As a result, accumulation of numerous metabolically altered macrophages was observed in the medullary cords. Reticulocytosis and interstitial edema led to macroscopic deformation of the lymph nodes and, from a clinical perspective, made them non-palpable in some cases, while in others resulting in lymph node enlargement. Reticulosis in the medullary cords of the lymph node causes displacement of the vascular network toward the periphery and hinders lymph flow, leading to congestion in the medullary and hilar regions of the lymph node. Histological examination revealed the formation of cystically dilated foci.

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When measured morphometrically, these foci were found to be mostly 45–75 μm in size. Normally, cystic dilatations of this size are not observed in the medullary region of lymph nodes (see Figures 3 and 4). This, in turn, provides a basis for concluding that impaired lymphatic dynamics may clinically lead to pulmonary edema based on these morphological changes. Thus, in SARS-CoV-2 infection, changes in the lymph nodes are mainly characterized by varying degrees of functional responses in the active zones of the lymph node, namely paralysis of the T-cell zone, marked proliferation of the B-cell zone, varying degrees of congestion and sclerosis of postcapillary venules, reticulocytosis in the medullary cords, formation of cystically dilated foci, inhibition of T- and B-lymphocyte migration in the medullary cords, and overall dysfunction of the cellular immune system. A marked proliferation of macrophages and reticulocytes around T-zone lymphocytes, increased proliferative activity of connective cells within the stromal–vascular structures of the lymph node, thickening of the vascular wall, and impaired migration of T-lymphocytes from postcapillary venules (microangiosclerosis) were observed. Subsequently, disruption of lymphatic fluid dynamics through the vessels was also detected, leading to the formation of numerous cystically dilated foci.

Clinically, these changes are manifested by a severe deficiency of cellular components of the immune system and the involvement of secondary infectious factors, as well as paralysis of the lymphatic drainage function in vital organs. This is primarily expressed in lung tissue by the development of mixed-type pulmonary edema (non-cardiogenic and lymphostatic).

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