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ULTRASTRUCTURAL CHANGES IN THE VESSELS OF THE LUNG MICROCIRCULATORY BED IN CORONAVIRUS INFECTION

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Damage to the endothelium of pulmonary microcirculatory vessels and the development of coagulopathy are considered important elements of the pathogenesis of coronavirus infection. The purpose of the study was to determine the ultrastructural changes of the microcirculatory bed of the lungs of patients who died due to respiratory failure during the coronavirus infection, using transmission electron microscopy. In the vessels of the microcirculatory bed of the lungs of patients who died on the 14th, 20th, 22nd, and 40th days of the disease, dyscirculatory processes of varying severity were observed in the form of severe hyperemia, stasis, microthrombi, and alternative changes in the endothelium. Damage to the vessels of the microcirculatory bed was accompanied by pronounced coagulopathy and endotheliitis, which are key aspects of the pathogenesis and thanatogenesis of coronavirus infection.

Key words: coronavirus infection, COVID-19, transmission electron microscopy, pulmonary microcirculatory bed, endothelium, coagulopathy.

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УЛЬТРАСТРУКТУРНІ ЗМІНИ В СУДИНАХ МІКРОКРОЦИРКУЛЯТОРНОГО РУСЛА ЛЕГЕНЬ ПРИ КОРОНАВІРУСНІЙ ІНФЕКЦІЇ

Важливими елементами патогенезу коронавірусної інфекції вважають пошкодження ендотелію легеневих мікроциркуляторних судин і розвиток коагулопатії. Метою дослідження було визначення ультраструктурних змін мікроциркуляторного русла легенів пацієнтів, які померли внаслідок дихальної недостатності під час коронавірусної інфекції, за допомогою трансмісійної електронної мікроскопії. У судинах мікроциркуляторного русла легень хворих, які померли на 14, 20, 22 і 40-ту добу захворювання, спостерігалися дисциркуляторні процеси різного ступеня вираженості у вигляді важкої гіперемії, стазу, мікротромбів та альтернативних змін ендотелію. Ураження судин мікроциркуляторного русла супроводжувалося вираженою коагулопатією та ендотеліїтом, які є ключовими аспектами патогенезу та танатогенезу коронавірусної інфекції.

Ключові слова: коронавірусна інфекція, COVID-19, трансмісійна електронна мікроскопія, мікроциркуляторне русло легень, ендотелій, коагулопатія.

The study is a fragment of the research project "Study of pathogenetic mechanisms and pathomorphological features of diseases of the endocrine, cardiovascular, respiratory, nervous, digestive, urinary and reproductive systems with the aim of improving their morphological diagnosis", state registration No. 0123U201668.

At the end of 2019, the first reports of a new severe respiratory disease in Wuhan developed due to human infection with the SARS-CoV-2 coronavirus were made. Due to the rapid spread and large scale of the disease, in March 2020, the WHO Directorate-General declared COVID-19 a global pandemic [3, 15].

To date, the pathogenesis and pathomorphology of COVID-19 still need to be better understood. It is known that the SARS-CoV-2 virus uses angiotensin-converting enzyme 2 (ACE-2) receptors to infect cells. The ACE-2 protein is expressed in significant amounts on the cytoplasmic membrane of type 2 pneumocytes, type 1 pneumocytes, cardiomyocytes, cholangiocytes, vascular endothelium, epithelial cells of the esophagus, stomach, ileum, and rectum, and the epithelium of the proximal tubules of the kidneys and bladder [13].

The glycoprotein trimer, through which the coronavirus interacts with the cell, consists of two subunits: S1, the receptor subunit, and S2, responsible for the fusion of the coronavirus with the cell membrane [11, 8]. Cells infected with the virus undergo alternative changes, accompanied by activation of alveolar macrophages, production of proinflammatory cytokines and chemokines, and the development of a "cytokine storm" [11].

The scientific literature contains the results of electron microscopic studies of the lungs of patients who died due to respiratory failure in COVID-19, based on transmission and scanning electron microscopy [1, 6, 10, 13]. The results of these studies describe pathological changes in the parenchymal elements of the lungs, as well as stromal components, including the vessels of the lung interstitium. According to the results of scanning electron microscopy and microvascular corrosion, patients who died due to respiratory failure in COVID-19 develop pulmonary capillary deformation and invasive angiogenesis [1]. Transmission electron microscopic examination of lung biopsies taken by transbronchial "cryobiopsy" from patients who were on mechanical ventilation within 30 minutes after their death revealed ultrastructural correlates of different phases of diffuse alveolar damage, including alveolar epithelial detachment, type 2 alveolocyte hyperplasia, with preservation of endothelial integrity in areas where the alveolar epithelium had already been detached [6].

According to Monique Freire Santana et al. [14], who conducted an transmission electron microscopic study of the lungs of patients with COVID-19, necrosis, and apoptosis of pneumocytes of types I and II, degenerative changes in the endothelium, dilatation of capillaries and exposure of their basement membrane, with platelet adhesion and the formation of fibrin thrombi develop. Transmission electron microscopic also detected lung endothelial damage and thrombosis during COVID-19 [10].

Thus, the study of ultrastructural changes in the vessels of the lung microcirculatory system (MCS) using transmission electron microscopic will allow for a more detailed and in-depth analysis of the development of discirculatory processes and description of structural changes in the vascular endothelium, blood cells, and thorough investigation of individual links in the pathogenesis of coronavirus infection.

The purpose of the study was to investigate the ultrastructural components of the lung microcirculatory bed of patients who died due to respiratory failure caused by COVID-19 to identify and describe the detected ultrastructural pathomorphological changes in detail.

Materials and methods. The material was taken by autopsy using a puncture needle no later than 2 hours after the patient's death. The study group consisted of ten people aged 34 to 85, with a male-to-female ratio of 1 to 1.5. The disease duration ranged from 9 to 40 days (1 patient died on day 9, three patients on day 14, three patients on day 17, and one patient on days 18, 22, and 40 of the disease). The material was fixed in Millonig's fixative with a pH of 7.36. Dehydration of the material was performed in ethanol of increasing strength with a concentration difference from 10 % to 70 % of ethanol solution in distilled water. Subsequently, the material was kept in 3 portions of absolute ethanol for 10 minutes each, transferred to 2 portions of propylene oxide for 5 minutes each and resized for 24 hours in a mixture of Araldite of the following composition: Araldite M, HY964 sealer 1:1, mixed thoroughly. Ultrathin sections with a thickness of 60 nμ were made using an LKB 2188 Ultrotome NOVA ultramicrotome. The obtained 60 nμ thick sections were mounted on support grids through water, dried for two hours at 60°C, and contrasted with uranyl acetate and lead citrate by Reynolds. The slides were washed in 0.02 M NaOH solution and then in distilled water, followed by drying.

The samples were viewed in a transmission electron microscope PEM 100-01, and photographic recordings were made using a KAPPA Image Base digital camera.

Results of the study and their discussion. Patients with respiratory failure due to COVID-19 were diagnosed with bilateral interstitial pneumonia, progressive diffuse alveolar damage accompanied by the appearance of hyaline membranes, hyperplasia of type II pneumocytes, severe dyscirculatory changes, and fibroblast activation.

In patients who died during the first 14 days of the disease (late exudation phase), pronounced dyscirculatory changes in the vessels of MCS of the lungs and alternative changes in endothelial cells were observed. In connection with the development of hyperemia, the lumens of arterioles, capillaries, and venules were expanded and filled with swollen, deformed red blood cells (RBCs). In hemocapillaries, RBCs are of different shapes and sizes. The cytoplasm of RBCs has a heterogeneous electron density. In particular, in some cells the cytoplasm had an increased electron density, while in others the plasma membrane acquired a heterogeneous electron density and underwent focal destruction. In hemocapillaries, the development of stasis was noted, which was accompanied by the adhesion of individual RBCs to the luminal surface of endothelial cells and the aggregation of neighboring RBCs (Fig. 1A). Ultrastructural changes in the endothelial cells of hemocapillaries are characterized by lightening of the cytoplasmic matrix, expansion of the tubular and granular endoplasmic reticulum (Fig.1B), with heterogeneity and swelling of their membranes, destruction of ribosomes. An increase in the content of condensed chromatin was observed in the nuclei of individual endothelial cells. The expansion of hemocapillaries, the adhesion of RBCs in their lumen, as well as the adhesion of RBCs to the luminal surface of the endothelium indicates the development of a pronounced dyscirculatory disorder in the hemocapillaries of the lung interstitium.

In patients who died on days 17–22 of the disease (proliferation phase), severe alternative endothelial changes were detected, as well as discirculatory changes with severe coagulopathy and the development of thrombosis of the vessels of the MCS. As a result of a sharp hemorrhage, the vessels of the MCS were significantly dilated and crowded with RBCs. The lumens of many hemocapillaries were completely blocked by clusters of swollen RBCs, and RBCs adhesion of the sludge syndrome type was recorded.

Alternative changes progressed in the endothelium in the form of pronounced dilation of smooth and granular tubules of the endoplasmic reticulum, destruction of a significant number of ribosomes, swelling of mitochondria, lightening and vacuolization of their matrix, destruction of cristae. The development of necrotic processes was recorded in some endothelial cells. In the arterioles and venules, there was a marked hemorrhage with pronounced aggregation of RBCs. Deep fibrin masses were visualized next to RBCs. In some hemocapillaries, in addition to RBCs, clawed masses of blood plasma and fibrin were detected. In the cytoplasm of some endothelial cells, vacuoles of medium electron density lipoproteins were detected. Due to the increase in vascular permeability, diapedesis of RBCs outside the vessels of the MCS and their accumulation in the lung interstitium were noted (Fig. 2).

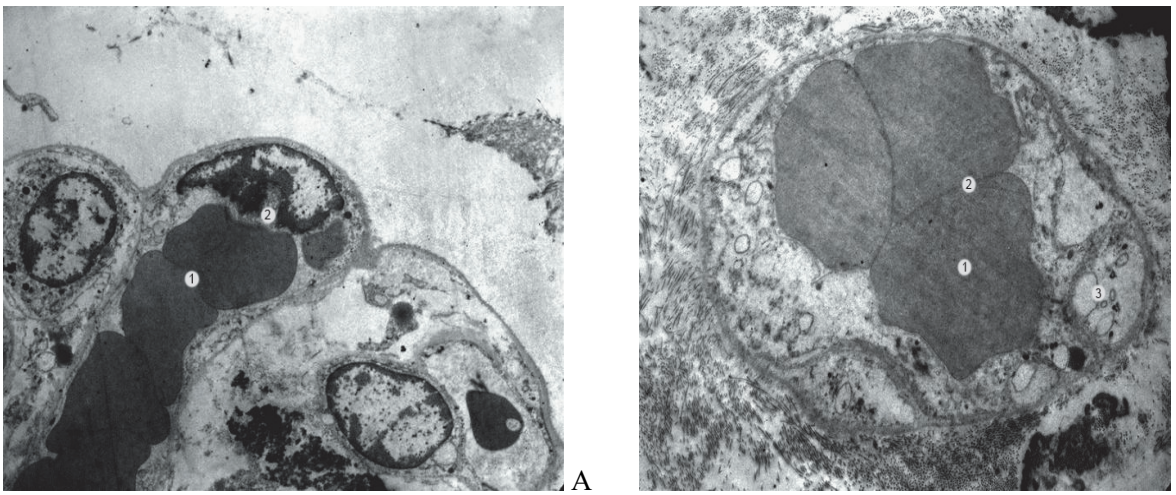

Fig. 1. Ultrastructural changes in RBCs in the hemocapillary lumen. Electrogram. x 2200 (fragment A) and x3800 (fragment B). A: 1 – aggregation of RBCs, 2 – adhesion of RBCs the luminal surface of the endothelial cytoplasm; B: 1 – swollen RBCs, 2 – aggregations of RBCs, 3 – enlightenment of the cytoplasmic matrix, expansion of the tubules of the granular endoplasmic reticulum of the endothelial cytoplasm.

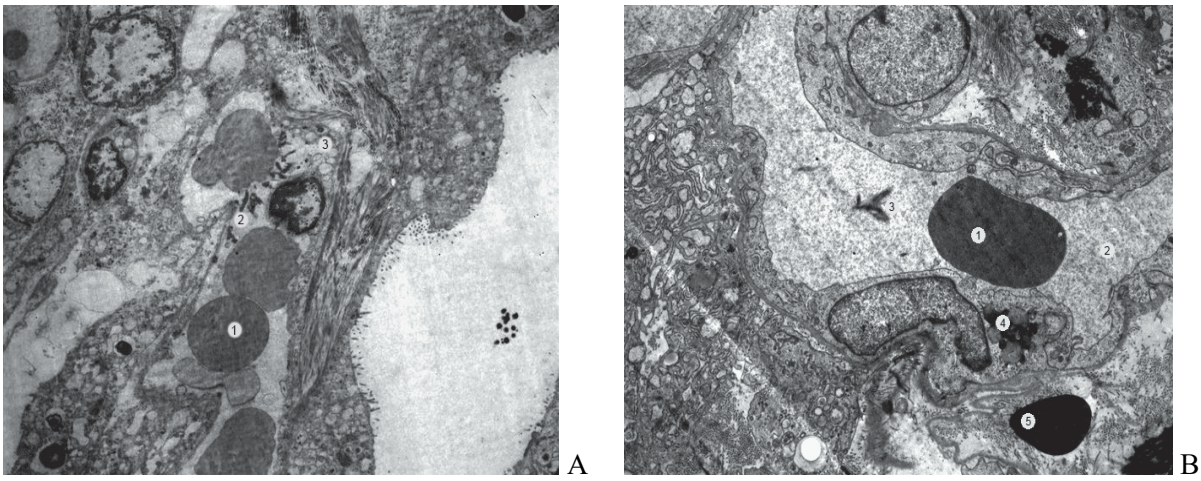

Fig. 2. Ultrastructural changes of lungs hemocapillars. Electrogram. x 1500 (fragment A) and x3000 (fragment B). A: 1 – swollen RBCs, 2 – fibrin masses in the venous lumen, 3 – expansion of granular endoplasmic reticulum tubules of the endothelial cytoplasm; B: 1 – RBC, 2 – clawed plasma masses, 3 – fibrin in the lumen of a hemocapillary, 4 – lipoproteins in the cytoplasm of an endothelial cell, 5 – RBC in the lung interstitium.

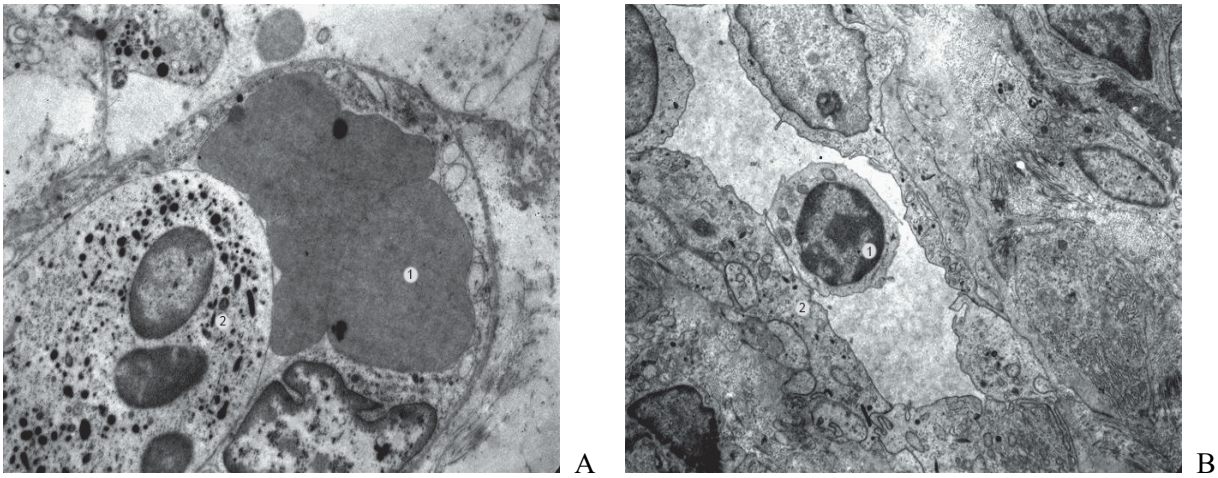

Fig. 3. Ultrastructural changes of different blood cells. Electrogram. x 3800 (fragment A) and x3000 (fragment B). A. Aggregation of blood cells in the hemocapillary lumen: 1 – swollen RBCs, 2 – segmented neutrophil in the hemocapillary lumen; B: 1 – lymphocyte in contact with the luminal surface of the endothelial cytoplasm (2).

The cytoplasmic matrix of endothelial cells appeared swollen and lightened. Numerous elements with an electron-dense core and sharp outgrowths on the periphery, bounded by a double-ring membrane, were visualized in the cytoplasm of individual endothelial cells, which could probably be virion-like particles. In connection with the development of necrotic changes, the cytoplasm of the endothelium brightened sharply, and the nucleus underwent pyknotic changes: it decreased in volume, acquired an

irregular shape, was filled with intensively condensed chromatin and the cariolemma lost clear contours. In areas of pronounced alteration of the endothelium, areas of exposure of the basal membrane of hemocapillaries were observed. In addition, the basement membrane of some hemocapillaries has lost electron density. Platelets of different sizes and fluffy masses of blood plasma were also visualized in the lumen of individual capillaries. In addition to swollen RBCs, neutrophils were also visualized in the lumens of hemocapillaries. Adhesion of neutrophils to the luminal surface of endothelial cells was noted. There were also lymphocytes adherent to the luminal surface of endothelial cells (Fig. 3). The matrix of mitochondria localized in the cytoplasm of lymphocytes was enlightened, and cristae were destroyed.

Due to alternative changes in the endothelium, vascular permeability was recorded, and coagulopathy and endotheliitis developed with the induction of neutrophil infiltration. In addition, RBCs diapedesis, migration of neutrophils, macrophages, and single lymphocytes outside the vessels, accumulation in the interstitium and alveoli, and severe inflammatory changes were observed. An increase in the number of active fibroblasts was noted in the lung stroma, indicating the initiation of fibrosis processes.

On day 40 of COVID-19 (fibrosis phase), in addition to hyperemia, stasis, RBC sludge, thrombosis of capillaries, venules, and arterioles, massive perivascular fibrin deposition was recorded (Fig. 4A). In addition to RBCs, neutrophils were also visualized in the capillaries. Adhesion of RBCs and neutrophils to the luminal surface of endothelial cells was noted. In the nuclei of neutrophils, there was an increase in the content and heterogeneous arrangement of condensed chromatin, as well as swelling and focal increase in the electron density of the cariolemma. Aggregation of blood cells was also observed in the lumen of hemocapillaries. Structural changes of endothelial cells were characterized by the expansion of granular tubules of the endoplasmic reticulum and their focal degranulation due to the destruction of ribosomes. The destruction of their cristae and pronounced lightening of the matrix was observed in the mitochondria. Some endothelial cells underwent necrotic changes. A rather characteristic phenomenon in patients who died on the 40th day after the onset of the disease is the activation of fibrosis processes. This is evidenced by the accumulation in the interstitium of many fibroblastic dipheron cells, including active fibroblasts (Fig. 4B), myofibroblasts, and multidirectional bundles of collagen fibres fibrils.

Thus, the ultrastructural examination of the respiratory lungs in patients with COVID-19 revealed the development of endotheliitis and severe discirculatory processes (hyperemia, stasis, and microthrombosis), which were accompanied by the development of interstitial fibrosis on days 17-22, and especially on day 40 of the disease.

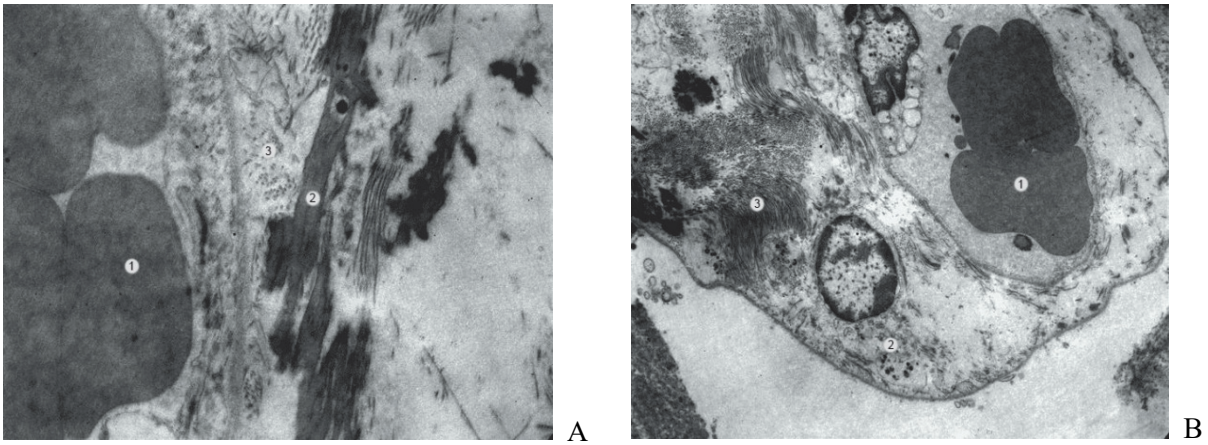

Fig. 4. Ultrastructural changes of lungs hemocapillars and interstitium. Electrogram. x 7500 (fragment A) and x2200 (fragment B). A: 1 – swollen and aggregated RBCs in the venous lumen, 2 – perivascular fibrin accumulation, 3 – collagen fibrils in the lung interstitium; B: 1 – aggregation of RBCs in the hemocapillary lumen, 2 – fibroblast, 3 – collagen fibrils in the interstitium.

It is known that the SARS-CoV-2 coronavirus has a cytopathic effect on type 2 pneumocytes, cardiomyocytes, epithelial cells of the respiratory tract, urinary system, neurons, and most importantly, endotheliotropic properties [5]. Damage to endothelial cells occurs due to viral penetration with damage to cell membranes, further developing microangiopathy and thrombosis. Along with the direct cytopathic effect of the virus, essential elements of the pathogenesis of the COVID-19 coronavirus infection are the development of uncontrolled immune and inflammatory responses and disruption of vascular homeostasis [3, 8].

With COVID-19, an increased risk of developing coagulopathies has been proven [9]. The development of hypercoagulation in COVID-19 is facilitated by endothelial dysfunction due to damage to the endothelium, platelets, and the blood coagulation system is activated. Dysregulation of the renin-angiotensin system occurs through the capture of angiotensin-converting enzyme two by SARS-CoV-2, leading to a pronounced immune response that can exacerbate endothelial changes. The occurrence of blood clots in the vessels of the pulmonary microcirculation in patients with COVID-19 is an important factor in determining the severity of the clinical picture of the disease [2].

According to Kanjakshi Ghosh [7], with COVID-19, the virus directly triggers the fibrinolytic system, a cytokine storm develops, exudate accumulates in the alveoli, intra- and extravascular blood coagulation, fibrinolysis, coagulation, and thrombus formation due to damage to the endothelium were recorded. The main coagulation catastrophe occurs due to immunocoagulation and the contribution of specific and non-specific cells (lymphocytes, monocytes, and neutrophils) that enhance the process [7]. Our data confirm severe dyscirculatory changes with active sludge syndrome alternative changes in the endothelium, which may indicate thrombotic microangiopathy.

Thrombi during SARS-CoV-2 coronavirus infection can form in vessels of various calibers, both arterial and venous [10], which are associated with the active release of inflammatory infiltrate by cellular mediators, activation of the complement system and fibrinolytic system. This leads to changes in the endothelium, increased vascular permeability, and thrombus formation by releasing many protein fragments into the blood, including fibrin breakdown elements [9].

The transmission electron microscopy of the lungs conducted by us also revealed the development of endotheliitis, which is an important link in the pathogenesis of lung damage during the COVID-19 coronavirus infection. According to Mosleh W. et al. [12], vascular endotheliitis may be the underlying pathological process leading to multiple organ failure and even death in patients with COVID-19. Endotheliitis caused by the SARS-CoV-2 coronavirus infection has a systemic nature and develops both as a result of direct exposure to the virus and as a result of the development of an inflammatory response in the host [3]. Given that with significant damage, the endothelium loses its physiological property of regulating homeostasis, fibrinolysis, and antiplatelet activity. Instead, a complex of reactions is triggered with the formation of numerous thrombi, which leads to coagulopathy in all cases of COVID-19 [4].

Damage to the endothelium of vessels with the development of alternative changes leads to a change in the properties of the surface of the endothelium from antithrombotic to prothrombotic. As a result of exposure to the proadhesive surface of the subendothelial layer, adhesive proteins, including fibrinogen, are drawn into the thrombosis. The alternative changes in the endothelium that we have identified lead to hypercoagulation and occlusion of hemocapillaries, small venules, and arterioles with a pronounced violation of blood circulation in the vessels of pulmonary MCS, which, in our opinion, is an important element of the pathogenesis and thanatogenesis of COVID-19.

Conclusion

Transmission electron microscopy of the lungs of patients who died as a result of respiratory failure caused by COVID-19 revealed pronounced dyscirculatory changes in the vessels of MCS with the development of hyperemia, stasis and microthrombosis, pronounced alterative-necrotic changes in the endothelium. The identified dyscirculatory processes were accompanied by coagulopathy and endotheliitis, which are key aspects of the pathogenesis and thanatogenesis of the COVID-19.

Prospects for further research are related to determining the mechanism of development of coagulopathies and the role of endothelial dysfunction in the pathogenesis of the COVID-19 coronavirus infection using adequate morphological methods.

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SOME INDICES OF METABOLISM IN CHILDREN WITH IRRITABLE BOWEL SYNDROME AND CO-EXISTING OVERWEIGHT

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In this article, we demonstrate the data on clinical course features of irritable bowel syndrome in children of school age with normal weight as well as co-existing overweight. Also, we conducted an assessment of certain indices of lipid and carbohydrate metabolism in these conditions. Clinical features of irritable bowel syndrome with overweight include duration of the disease (6.5±0.5 months), female sex (30 vs. 5 children, χ^2=5.1429, p<0.05), abdominal pain syndrome (85 % vs. 65 %, p<0.05), intermittent diarrhoea and constipation (62.5 % vs. 90 %, p<0.05), and bloating (77.5 % vs. 50 %, p<0.05). Initial signs of impaired carbohydrate metabolism were detected: an increase in the level of C-peptide (4.5±0.01 and 2.5±0.03, p<0.05) and NOMA index (3.41±0.02 and 2.86±0.01, p<0.05). The given data indicate deviations from the norm of the evaluated indicators of certain types of metabolism in school-age children with irritable bowel syndrome and overweight that will be reversible in case of adequate therapy.

Key words: children, irritable bowel syndrome, overweight, faecal calprotectin.

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СТАН ОКРЕМИХ ПОКАЗНИКІВ ОБМІНУ У ДІТЕЙ ІЗ СИНДРОМОМ ПОДРАЗНЕНОГО КИШЕЧНИКА ТА НАДЛИШКОВОЮ ВАГОЮ

В статті наведені дані про особливості перебігу синдрому подразненої кишки у дітей шкільного віку, на тлі нормальної та надлишкової маси тіла, а також проведено оцінку окремих показників ліпідного та вуглеводного обмінів при цій патології. Встановлено, що до клінічних особливостей обстежуваних дітей належать: тривалість (6,5±0,5 місяців), жіноча стать (30 і 5 дітей, χ^2=5,1429, p<0.05), больовий абдомінальний синдром (85 % і 65 %, p<0,05), чергування діареї та закрепу (62,5 % і 90 %, p<0,05) та метеоризм (77,5 % і 50 %, p<0,05). Інтенсивність больового синдрому була вищою у групі дітей із надлишковою масою тіла (6,5±0,5 і 4,3±0,1 балів, p<0.05). Виявлені початкові ознаки порушення вуглеводного обміну: зростання рівня С-пептиду (4,5±0,01 і 2,5±0,03, p<0.05) та індексу HOMA (3,41±0,02 і 2,86±0,01, p<0.05). Наведені дані свідчать про відхилення від норми показників окремих видів обміну в дітей із синдромом подразненої кишки на фоні надмірної маси тіла, які матимуть зворотній характер за умови адекватної терапії через рік від моменту спостереження.

Ключові слова: діти, синдром подразненої кишки, надлишкова маса тіла, фекальний кальпротектин.

The study is a fragment of the research project “Health state and features of adaptation in children in the Prykarpattya region with somatic disease, and its prevention”, state registration No. 0121U111129.

Functional gastrointestinal (GI) disorders and in particular irritable bowel syndrome (IBS) are one of the biggest concerns in paediatric gastroenterology globally taking into account the prevalence among children of school age (7 till 30 % according to the data of different authors), and its medical and psychosocial burden [1, 2, 7, 8].

At the same time, considering a comprehensive approach to the assessment of some chains of the pathogenesis, and major clinical symptoms of functional gastrointestinal disorders, we would like to note the presence of multiple blank spots in research of this field, especially when concomitant overweight or obesity is present [6, 8].

Based on data by WHO experts and novel research Global Burden of Disease Study (Imperial College London) [6, 7] the overall incidence of overweight in the world during the last three decades has increased by 27.5 % among adults and 47.1 % in children [7, 8]. In experts’ opinion, if the current trend