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Pathogenetic and Pathomorphological Aspects of Pulmonary Tissue Damage in COVID-19 as a Manifestation of Vasculitis

Objective — to determine the features of pathogenesis in patients with the pulmonary form of SARS-CoV-2. To establish, based on clinical examination data and electron microscopy, that vasculitis of the respiratory system is the main pathophysiological and pathomorphological factor in lung tissue damage in COVID-19.

Materials and methods. A total of 692 patients aged 18 to 73 years with severe SARS-CoV-2 were examined. The control group consisted of 50 patients (25 women and 25 men) aged 25—70 years with a moderately severe clinical course of SARS-CoV-2. 58 patients underwent immunological tests, which included the study of O-lymphocytes and D-lymphocytes, circulating small and large immune complexes. A group of 30 people who died of COVID-19 pneumonia underwent a pathohistological examination of tissue samples. A group of 10 people who died of COVID-19 pneumonia underwent electron microscopic examination of tissue samples.

Results and discussion. Immunological examination revealed an increase in the number of O-lymphocytes (53.3 ± 1.3), an increase in the number of D-lymphocytes (4.9 ± 0.7), which highlights the impairment of T-cells of immunity; the predominance of the total mass of an increased level of small CICs (71.3 ± 2.5), which indicates a particularly «malignant» autoimmune component with damage to the connective tissue structure, primarily the vessels of the lungs, and therefore the vessels of epithelial organs, and an increase in the number of large CICs, which explains the material basis of allergic complications that accompany the severe course of SARS. Ultrastructural electron microscopic examination of the respiratory tract of patients who died from respiratory failure in COVID-19 revealed the development of degenerative changes in the capillaries and endothelium, characterised by the expansion of the tubules of the granular endoplasmic reticulum, the destruction of ribosomes, the appearance of vacuoles in the cytoplasm surrounded by a double-contour membrane and containing small virion-like bodies, the basement membrane of haemocapillaries was heterogeneously thickened. The development of venulitis was recorded, which is characterised by the accumulation of neutrophils in the vessel wall, pronounced degenerative changes in the endothelium, accompanied by the destruction of cytoplasmic organelles, vacuolisation of the cytoplasm and its heterogeneous osmophilia, the appearance of heterogeneous microvilli on the lumenal surface of the endothelium, and heterogeneous thickening of the cytoplasmic membrane. Ultrastructural changes in the vascular endothelium in COVID-19 indicate the primary occurrence of acute vasculitis as a pathogenetic dominant in COVID-19 pneumonia.

Conclusions. Clinical manifestations and ultrastructural pathomorphological studies conducted by us reliably indicate that the primary pathogenetic basis of atypical pneumonia in COVID-19 syndrome is the occurrence of acute vasculitis with predominant damage to the vessels of the pulmonary system. The «malignant» course of COVID-19 syndrome involves the transformation of acute vasculitis into systemic vasculitis with subsequent unpredictable damage to a number of systems (respiratory, cardiovascular, hepatobiliary, genitourinary).

Keywords

SARS-CoV-2, COVID-19, vasculitis, pneumonia, pulmonary system, coronavirus infection, pathomorphology, electron microscopic changes, fatalities, forensic medicine.

The severe and dangerous COVID-19 disease for citizens and patients has been declared a pandemic by the World Health Organization in countries around the world. It has caused certain public health problems [3, 11—13]. 776,696,616 people have been confirmed to have the disease, 13,642,098,070 citizens have been vaccinated, and the mortality rate for this pathology has reached 7,073,446 confirmed deaths. Due to the high infectiousness of COVID-19 and the lack of experience in performing autopsies in cases of death caused by infectious diseases, the pandemic has created certain difficulties for practising forensic doctors [17].

The etiological factor that causes this disease is the SARS-CoV-2 coronavirus. The mechanism of infection and further replication of the virus occurs as follows [18, 19]:

- SARS-CoV-2 coronavirus enters the human body through the mucous membranes of the respiratory tract and enterocytes of the small intestine;
- the largest amount of virus during replication is localised in alveolar cells of type I and II, and this is explained by the fact that virus replication is carried out using angiotensin converting enzyme 2 (ACE2) receptors;
- at the same time, the largest amount of ACE2 is located on the surface of respiratory tract cells, especially on alveolocytes of type I and II;
- this explains the large percentage of lung damage in infected citizens, i. e. damage to the alveoli and capillaries leads to impaired gas exchange processes, due to which hypoxaemia and secondary damage to internal organs and systems develop.

The pathogen SARS-CoV-2 is facilitated by proteases that are intracellular. The activity of ACE2 is due to interferon (IFN). In turn, it has been established that the tropism of the coronavirus glycoprotein to endothelial cells that have the ACE2 receptor leads to the development of systemic vasculitis [9, 16].

Damage also occurs in other organs and systems. It is noted that the pathology develops in the heart and lungs. The kidneys, brain, and digestive tract are also affected. Necrotic cell death in coronavirus pathology leads to endothelial dysfunction, which is a causal factor in the systemic disruption of microcirculation of various organs and systems. This is an important factor that influences the development of vasculitis in COVID-19 pathology. There is also an autoimmune mechanism of damage in this pathology. Literature indicates that the interaction of the SARS-CoV-2 virus with the corresponding receptors on the surface of alveolar cells occurs [1, 4, 5, 10]. with the production of pro-inflammatory cytokines, the concentration of which can be extremely high in the form of the so-called «cytokine storm», which underlies acute respiratory distress syndrome (ARDS) and multiple organ failure syndrome (MOFS) [5].

Fatal outcomes among patients with COVID-19 are directly related to:

- high serum interleukin-6 (IL-6) levels [5, 10];
- comorbidities (cardiovascular disease, type 1 and type 2 diabetes, hypertension) [18, 19].

These disorders share a common pathophysiology involving the renin-angiotensin system (RAS) that may be of clinical relevance. In particular, angiotensin-converting enzyme 2 (ACE2) activity is impaired in cardiovascular disease and this enzyme is used by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) to initiate infection. Cardiovascular disease and pharmacological inhibition of RAS increase ACE2 levels, which may increase the virulence of SARS-CoV-2 in the lungs and heart [4, 5].

It is known that the vascular endothelium is a paracrine, endocrine, and autocrine organ that is essential for maintaining homeostasis. Endothelial dysfunction is a major determinant of microvascular dysfunction by shifting the vascular balance towards vasoconstriction with subsequent organ ischemia, as well as a pro-coagulant state of the blood.

In addition, the induction of apoptosis and pyroptosis (a type of programmed necrotic cell death) may play an important role in endothelial vascular damage in patients with COVID-19, which may explain [10, 15]:

- systemic impairment of microcirculation in the vascular bed of various organs, systems and clinical manifestations and consequences in patients with COVID-19;
- theoretical justification for therapy aimed at stabilising endothelial cells and their function in combating viral replication, especially with the help of anti-inflammatory anti-cytokine drugs, ACE inhibitors and statins;
- a strategy that may become relevant for a particularly vulnerable category of patients with pre-existing endothelial dysfunction, male gender, smoking, arterial hypertension, diabetes, obesity, cardiovascular diseases.

Objective — to determine the features of pathogenesis in patients with the pulmonary form of SARS-CoV-2. To establish, based on clinical examination data and electron microscopy, that vasculitis of the respiratory system is the main pathophysiological and pathomorphological factor in lung tissue damage in COVID-19.

Materials and methods

The studies were conducted from 2022 to October 2024. A total of 692 patients aged 18 to 73 years with severe SARS-CoV-2 were examined. The control group consisted of 50 patients (25 women and

Table. **Immunogram indicators of patients with SARS-CoV-2**

Immunity indicators	Patients with SARS-CoV-2 with autoaggression	Patients with SARS-CoV-2 without autoaggression
O-lymphocytes, %	53.3 ± 1.3 ($p < 0.001$)	43.4 ± 1.6 ($p < 0.001$)
D-lymphocytes, %	4.9 ± 0.7 ($p < 0.001$)	1.8 ± 0.6 ($p < 0.001$)
CIC-small, ODU	71.3 ± 2.5 ($p < 0.001$)	53.3 ± 1.9 ($p < 0.001$)
CIC-large, ODU	69.4 ± 0.86 ($p < 0.001$)	61.2 ± 0.8 ($p < 0.001$)
Number examined	n = 58	n = 50

Note. ODU — optical density units.

25 men) aged 25—70 years with a moderately severe clinical course of SARS-CoV-2.

All patients underwent general clinical and laboratory examination methods. A PCR test was mandatory. Radiological examination methods included chest radiography and computed tomography of the lungs. 58 patients underwent immunological tests, which included the study of O-lymphocytes and D-lymphocytes, circulating immune complexes of small and large.

Pathological and medical studies indicate that the autopsy of corpses due to nosocomial coronavirus disease (COVID-19) has enormous medical, legal and socio-economic significance, since the autopsy is an important and evidentiary element for the correct statistical accounting of deaths from COVID-19, presenting new, complete and objective data that are important for political information to the population, as well as for improving the work of the entire healthcare sector in general and a separate healthcare institution in particular [6].

A group of 30 people who died from COVID-19 pneumonia underwent a pathohistological examination of tissue samples.

A group of 10 people who died from COVID-19 pneumonia underwent an electron microscopic examination of tissue samples. These were patients aged 34 to 85 years, with a male to female ratio of 1 to 1.5. The duration of the disease was from 9 to 40 days (1 patient died on the 9th day, three patients on the 14th day, three patients on the 17th day, one patient on the 18th, 22nd and 40th day of the disease.

For electron microscopic examination, the material was taken at autopsy using a puncture needle, no later than 2 hours after the time of death of the patients.. The material was fixed in Millonig's fixative with a pH of 7.36. For this, 0.2 molar Millonig's phosphate buffer was combined with 1.4 % osmium tetroxide solution in a 1:1 ratio. As a result, a 2 % solution of osmium tetroxide in 0.1 molar Millonig's phosphate buffer was obtained. The material was dehydrated in ethanol of increasing strength with a concentration difference from 10 % to 70 % ethanol solution in distilled water. Further, the material was kept in 3 portions of absolute ethanol for 10 min each, transferred to 2 portions of propylene oxide for 5 min and stored for 24 h in a mixture of araldite. of the following composition: Araldite M, sealant HY964 1 : 1, thoroughly mixed. Ultrathin sections 60 nm thick were made using an LKB 2188 Ultrotome NOVA ultramicrotome. The resulting sections 60 nm thick were mounted on support grids through water, dried for 2 h at a temperature of 60 °C and contrasted with uranyl acetate and lead citrate according to Reynolds. They were washed in 0.02 M NaOH solution, and then in distilled water with subsequent drying.

The samples were viewed in a transmission electron microscope PEM 100-01, and photo fixation was carried out using a KAPPA Image Base digital camera.

Results and discussion

It is important to note that previous studies and monitoring of the dynamics of the spread of COVID-19 in Ukraine as of April 13, 2024 show that [2, 7, 8, 14]:

- 5,557,995 (13.5 %) citizens fell ill with COVID-19;
- 112,416 (2.0 %) of them were declared fatal cases;
- 32,603,808 tests were performed;
- 75,485 tests per 1 million citizens.

During our clinical examinations, it was noted that patients complained of: elevated body temperature, mixed shortness of breath, cough with a small amount of mucous sputum, loss of smell and taste.

Immunological examination revealed (Table):

- an increase in the number of O-lymphocytes (53.3 ± 1.3) %, which indicates autoaggression with subsequent systemic damage to vessels and immunocompetent organs;
- an increase in the number of D-lymphocytes (4.9 ± 0.7) %, which highlights the impairment of T-cells of the immune system;
- a predominance in the total mass of an increased level of small CICs (71.3 ± 2.5) ODU, which indicates a particularly «malignant» autoimmune component with damage to the connective tissue structure, primarily the vessels of the lungs, and therefore the vessels of epithelial organs;
- an increase in the number of large CICs, which explains the material basis of allergic complications that accompany the severe course of SARS.

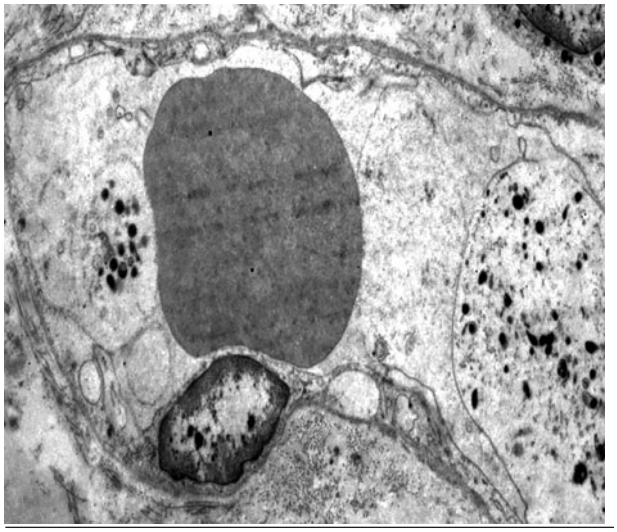

Fig. 1. **Endotheliitis due to COVID-19**

Erythrocyte and neutrophils in the lumen of the hemocapillary. Disintegration of the cytoplasm of the neutrophil. Degenerative changes in the endothelium. Vacuoles in the cytoplasm of the endothelium. Electronogram × 3800.

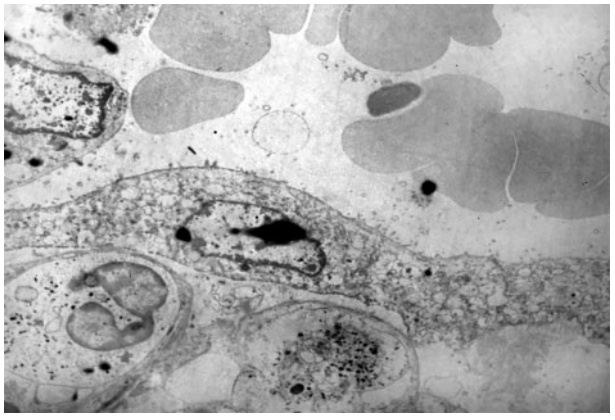

Fig. 2. **Venulitis due to COVID-19**

Aggregated erythrocytes in the dilated lumen of the venule. Degenerative changes in the cytoplasm of the endothelium. Neutrophils in the wall of the venule. Electronogram × 1900.

Need to note that the dynamics of the study of the obtained indicators among the patients of the examined groups indisputably proved the growth of the hypoimmune and autoaggressive component in patients with severe SARS-CoV-2, which, in our opinion, was the basis for the fatal outcome.

In addition, during the autopsy study of those who died from coronavirus disease, the lungs were enlarged, sharply hyperemic, and the tissue was mottled. Pronounced edema, hemorrhages, and local thrombosis of the microcirculatory bloodstream were observed. The macroscopic «picture» of the lungs resembled atypical pneumonia. Depending on the duration of the disease (on what day the patient died), the density of the lung tissue differed. For example, in the case of long COVID, the lungs were sharply compacted and had reduced airiness.

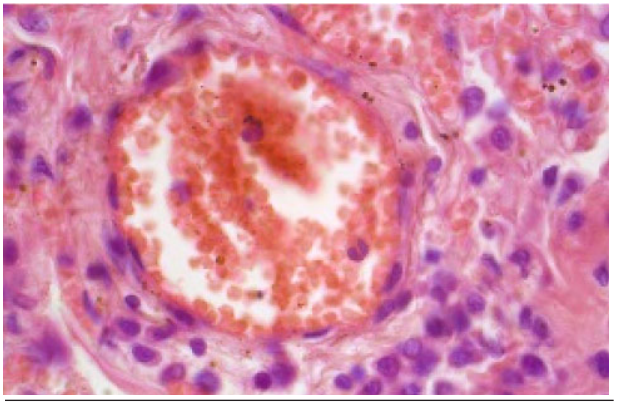

Fig. 3. **The lumens of the haemocapillaries were dilated, overflowing with glued swollen erythrocytes and neutrophils**

Hematoxylin & Eosin × 100.

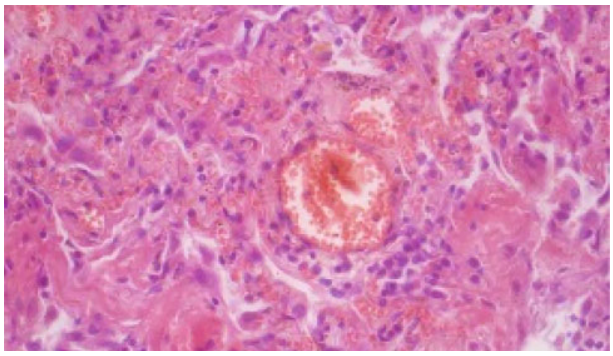

Fig. 4. **Venulitis with inflammatory infiltration within vascular wall and dilation of the lumen, overflowing erythrocytes and neutrophils**

Hematoxylin & Eosin × 40.

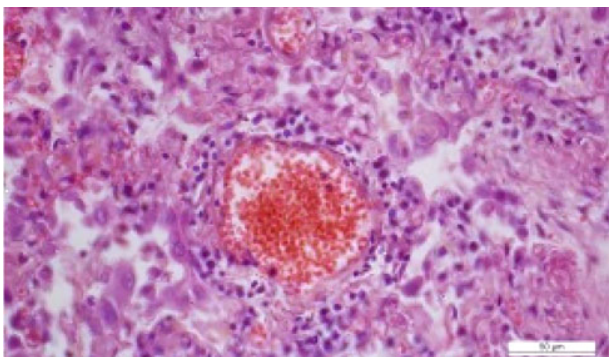

Fig. 5. **Endotheliitis with dilation and hyperaemia of the lumen and dense inflammatory infiltration with predominance of neutrophils within the wall**

Hematoxylin & Eosin × 40.

For in-depth study, we conducted a study of the respiratory departments of the lungs of patients who died from respiratory failure in COVID-19, and found the following (Fig. 1—5):

- development of endotheliitis was detected in the capillaries;
- signs of venulitis were recorded in the venules;
- the lumens of the haemocapillaries were dilated, overflowing with glued swollen erythrocytes and neutrophils;

- neutrophils were in contact with the luminal surface of endothelial cells;
- the development of degenerative changes was recorded in the endothelium, characterised by the expansion of the tubules of the granular endoplasmic reticulum, the destruction of ribosomes, the appearance of vacuoles in the cytoplasm that were surrounded by a double-contour membrane and contained small virion-like bodies;
- the basement membrane of haemocapillaries thickened heterogeneously;
- erythrocytes and neutrophils localized in haemocapillaries contacted the luminal surface of endothelial cells;
- the development of venulitis was recorded, which is characterised by the accumulation of neutrophils in the vessel wall, pronounced degenerative changes in the endothelium, accompanied by the destruction of cytoplasmic organelles, vacuolisation of the cytoplasm and its heterogeneous osmophilia, the appearance of heterogeneous microvilli on the luminal surface of the endothelium, and heterogeneous thickening of the cytoplasmic membrane.

Ultrastructural changes in the vascular endothelium in COVID-19 and pathomorphological changes indicate the primary occurrence of acute vasculitis as a pathogenetic dominant in COVID-19 pneumonia. Subsequently, the pathogenetic basis of acute vasculitis acquires the properties of systemic damage to tissues and organs, which determines the development of the clinical picture of post-COVID syndrome.

Conclusions

1. Clinical manifestations and ultrastructural pathomorphological studies conducted by us reliably indicate that the primary pathogenetic basis of atypical pneumonia in COVID-19 syndrome is the occurrence of acute vasculitis with a predominant lesion of the vessels of the pulmonary system.

2. The «malignant» course of COVID-19 syndrome involves the transformation of acute vasculitis into systemic vasculitis with subsequent unpredictable damage to a number of systems (respiratory, cardiovascular, hepatobiliary, genitourinary).

There is no conflict of interest.
Participation of authors: concept development and study design — A.A. Nevzghoda, O.A. Nevzghoda; data collection and processing, statistical analysis, manuscript writing — A.A. Nevzghoda, O.A. Nevzghoda, M.R. Semko; editing — O.A. Nevzghoda.

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Патогенетичні та патоморфологічні аспекти ушкодження легеневої тканини при COVID-19 як прояву васкуліту

Мета роботи — визначити особливості патогенезу в пацієнтів із легеневою формою SARS-CoV-2. Установити на підставі даних клінічних обстежень та електронної мікроскопії, що васкуліт дихальної системи є основним патофізіологічним та патоморфологічним чинником ураження легеневої тканини при COVID-19.

Матеріали та методи. Обстежено 692 хворих віком від 18 до 73 років із тяжким перебігом COVID-19. Контрольну групу утворено з 50 пацієнтів (25 жінок та 25 чоловіків) віком від 25 до 70 років із клінічним перебігом COVID-19 середньої тяжкості. У 58 хворих проведено імунологічні тести, зокрема досліджено О-лімфоцити й D-лімфоцити, великі та малі циркулюючі імунні комплекси. У групі осіб, які померли внаслідок пневмонії при COVID-19, у 30 випадках проведено патогістологічне дослідження зразків, у 10 — електронно-мікроскопічне дослідження.

Результати та обговорення. При імунологічному обстеженні виявлено збільшення кількості О-лімфоцитів (53,3 ± 1,3) і D-лімфоцитів (4,9 ± 0,7), що свідчить про ураження Т-клітинного імунітету. Підвищення вмісту малих циркулюючих імунних комплексів (71,3 ± 2,5) вказує на особливо «злоякісний» автоімунний компонент з ураженням сполучнотканинної структури, насамперед судин легень, а відтак і судин епітеліальних органів, а збільшення рівня великих ЦІК пояснює основу алергійних ускладнень, що супроводжують тяжкий перебіг SARS. При ультраструктурному електронно-мікроскопічному дослідженні респіраторних відділів легень пацієнтів, які померли внаслідок респіраторної недостатності при COVID-19, виявлено в капілярах та ендотелії дегенеративні зміни, зокрема розширення канальців гранулярного ендоплазматичного ретикулуму, деструкцію рибосом, появу в цитоплазмі вакуолей, оточених двохконтурною мембраною, які містили дрібні віріоноподібні тільця, неоднорідне потовщення базальної мембрани гемокапілярів. Реєстрували розвиток венуліту, який характеризувався накопиченням у стінці судин нейтрофілів, виразними дегенеративними змінами ендотелію, що супроводжувались деструкцією цитоплазматичних органел, вакуолізацією цитоплазми та її неоднорідною осміофільністю, появою на люменальній поверхні ендотелію гетерогенних мікроворсинок, неоднорідним потовщенням цитоплазматичної мембрани. Ультраструктурні зміни ендотелію судин при COVID-19 свідчать про первинне виникнення гострого васкуліту як патогенетичної домінанти при пневмонії, спричиненої COVID-19.

Висновки. Клінічні вияви та результати ультраструктурних патоморфологічних досліджень свідчать про те, що первинною патогенетичною основою атипової пневмонії при COVID-19 є виникнення гострого васкуліту з переважним ураженням судин легеневої системи. «Злоякісний» перебіг COVID-19 передбачає трансформацію гострого васкуліту в системний васкуліт із подальшим ураженням низки систем (дихальної, серцево-судинної, гепатобіліарної, сечостатевої).

Ключові слова: SARS-CoV-2, COVID-19, васкуліт, пневмонія, легенева система, коронавірусна інфекція, патоморфологія, електронно-мікроскопічні зміни, летальний наслідок, судова медицина.

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