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Clinical and Morphological Indicators of the Algorithm of Respiratory Damage in Covid – 19

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Resume. Coronavirus disease pandemic 2019 (COVID-19) is spreading rapidly. The cause of the disease is the new coronavirus SARS-CoV-2, which has a high infectivity in the absence of effective antiviral drugs and vaccines. SARS-CoV-2 is a single-stranded RNA-containing virus belonging to the Coronaviridae family. According to genome sequencing, it was shown that there is a genetic affinity of the new virus with the previously known coronaviruses SARS-CoV (similarity ~79%) and MERSCoV (similarity ~50%).

Keywords: pandemic, coronavirus, respiratory organs, COVID - 19

Relevance. At the end of 2019, an outbreak of a new coronavirus infection occurred in the People's Republic of China (PRC) with an epicenter in the city of Wuhan (Hubei Province), the causative agent of which was given the temporary name 2019-ncov[6,8].

On February 11, 2020, the World Health Organization (WHO) assigned the official name of the infection caused by the new coronavirus - COVID-19 ("coronavirus diseases 2019")[4,7]. January 11, 2020. assigned the official name of the causative agent of infection - Atypical pneumonia-COV-2[3,5].

The appearance of COVID-19 has set tasks for healthcare professionals related to rapid diagnosis and provision of medical care to patients[2,6]. Currently, information about the epidemiology, clinical features, prevention and treatment of this disease is limited [4,7].

It is known that the most common clinical manifestation of a new variant of coronavirus infection is bilateral pneumonia, in 3-4% of patients the development of acute respiratory distress syndrome (ARDS) was registered[1,5].

According to the results of serological and phylogenetic analysis, coronaviruses are divided into four genera: Alpha-coronavirus, Betacoronavirus, Gammacoronavirus and Deltacoronavirus. The natural hosts of most of the currently known coronaviruses are mammals[3].

Until 2002, coronaviruses were considered as agents causing mild upper respiratory tract diseases (with extremely rare deaths). 2002 2002 coronavirus (Atypical pneumonia) appeared, the causative agent of atypical pneumonia, which caused SARS in humans. This virus belongs to the genus Betacoronavirus. The natural reservoir of SARS is bats, intermediate hosts are camels and Himalayan civets[6]. In total, during the epidemic period, more than 8000 cases were registered in 37 countries around the world, of which 774 were fatal. Since 2004 no new cases of atypical pneumonia caused by SARSKoV have been registered.

2012 2012 the world is faced with a new MERS coronavirus (MERS-COV), the causative agent of the Middle East respiratory syndrome, also belonging to the genus Betacoronavirus. The main natural reservoir of MERS-Cov coronaviruses are single-humped camels (dromedaries). From 2012 to January 31, 2020, 2,519 cases of coronavirus infection caused by the MERS-CoV virus were registered, of which 866 were fatal. All cases of the disease are geographically associated with the Arabian Peninsula (82% of cases are registered in Saudi Arabia). "MERS-COV"[2,6].

The new coronavirus SARS-coronavirus-2 is a single-stranded RNA-containing virus, belongs to the Coronaviridae family, belongs to the beta-cov line B. The virus is assigned to the second group of pathogenicity, as are some other representatives of this family (SARS virus-MERS-CoV coronavirus).

The SARS-COV-2 coronavirus is presumably a recombinant virus between a bat coronavirus and an unknown coronavirus by origin[1,4]. The genetic sequence of SARSCoV-2 is similar to the sequence of SARS-COV by at least 79%.



The entrance gate of the pathogen is the epithelium of the upper respiratory tract and epithelial cells of the stomach and intestines[3]. The initial stage of infection is the penetration of SARS-CoV-2 into target cells having type II angiotensin converting enzyme (ACE2) receptors. ACE2 receptors and are present on the cells of the respiratory tract, kidneys, esophagus, bladder, ileum, heart, central nervous system. However, the main and quickly achievable target is alveolar cells of type II (AT2) of the lungs, which determines the development of pneumonia[5,7]. The role of CD147 in the invasion of SARS-COV-2 cells is also discussed.

It has been established that the dissemination of SARS-CoV-2 from the systemic bloodstream or through the plate of the latticed bone (cribrosa plate) can lead to brain damage. A change in the sense of smell (hyposmia) in a patient at an early stage of the disease may indicate damage to the central nervous system, as well as swelling of the nasopharyngeal mucosa.

The purpose of the study. Study of the morphology of lung lesions in COVID-19 based on the analysis of autopsy data.

Materials and methods of research. Results 200 autopsies (121 deceased men and 79 women; average age 68.5 ± 15.63 years) were examined, for a unique COVID-19 number of pathoanatomic autopsies conducted in Uzbekistan from March 20 to May 22, 2020.

The results of the study. The pathological changes of the lungs characteristic of COVID-19, varying in their prevalence, were detected in all the deceased and consisted in the development of diffuse alveolar damage (DAP) in combination with damage to the vascular bed of the lungs (microangiopathy, thrombosis, in some cases destructive-productive vasculitis) and alveolar-hemorrhagic syndrome, mainly in the first, exudative, phase of DAP. Such viral interstitial pneumonia with a vascular and hemorrhagic component was the morphological substrate of ARDS.

Clinical manifestations of acute respiratory infection (body temperature above 37.5°C and one or more signs: cough, dry or with scanty sputum, shortness of breath, chest congestion, blood oxygen saturation according to pulse oximetry, sore throat, runny nose and other catarrhal symptoms, weakness, headache, anosmia, diarrhea) in the presence of at least one of the epidemiological signs:

- return from a foreign trip 14 days before the onset of symptoms;
- the presence of close contacts over the past 14 days with a person under surveillance for COVID-19, who subsequently became ill;
- the presence of close contacts over the past 14 days with a person who has a laboratory confirmed diagnosis of COVID-19;
- work with patients with confirmed and suspected cases of COVID-19.

The presence of clinical manifestations of severe pneumonia, with characteristic changes in the lungs according to computed tomography or chest X-ray (see paragraph 3.1 and appendix 1 of these recommendations) regardless of the results of a single laboratory test for the presence of SARS-coronavirus-2 RNA and an epidemiological history.

A case suspected of COVID-19 when it is impossible to conduct a laboratory test for the presence of RNA is Atypical pneumonia-CoV-2.

Conclusion. The revealed pathomorphological features of the inflammatory process of COVID-19 in patients (priority of endothelial damage with micro- and macrothrombosis, relatively late development of the exudative phase of inflammation and a tendency to develop pneumofibrosis) determine the long duration of therapy and the need for respiratory rehabilitation, mainly aimed at pulmonary recruitment.

At the same time, the obtained results raise a number of urgent questions about the expediency and duration of the use of a number of medicines.

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