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Modern Concepts of Bronchopneumonia as a Complication of Coronavirus Infection

Nuriddinov Asliddin Mekhridinovich
Bukhara State Medical Institute

Abstract: The first cases of human infection with a new coronavirus infection, called "Corona Virus Disease 2019" (COVID-19), appeared in late 2019 - early 2020 in Wuhan (China). At the time of writing this review, more than 6 million cases and more than 400 thousand deaths have been registered worldwide. During this time, a large number of articles have appeared describing the diagnosis, pathogenesis, clinic and therapy of this disease. This review is devoted to the complication of coronavirus infection bronchopneumonia.

Keywords: coronavirus infection, bronchopneumonia, complications.

Relevance. According to a study conducted by a group of scientists [Samsonova M.V., et al., 2020]: With COVID-19, severe patients develop viral interstitial pneumonia with the development of diffuse alveolar damage, often with severe alveolar-hemorrhagic syndrome and widespread thrombosis of the microvasculature, less often - large vessels of the lungs, which causes a severe course of the disease, hypoxia and respiratory failure. The incomplete correspondence of morphological changes in the lungs (phases of diffuse alveolar damage) to the duration of the disease is probably due to the undetermined duration of the asymptomatic course in many patients. The course of a new coronavirus infection is characterized by a predominant lesion of the lungs, a decrease in SpO2, lymphopenia with a simultaneous increase in leukocytosis, signs of hypercoagulability with the risk of developing thrombosis of arteries and veins of various organs and, as a result, infarctions of the lungs, myocardium, brain in extremely severe and comorbid patients. We assume that the occurrence of viral-bacterial pneumonia is possible mainly in patients with tracheal intubation with tracheostomy and prolonged mechanical ventilation.

In the lungs with COVID-19, in all cases, diffuse alveolar damage was determined with varying degrees of severity of DIC, as in other viral lung lesions (caused by SARS-nCoV, MERS-CoV, influenza A / H1N1) [Tsinzerling A.V., 1977, Petrosillo N, Viceconte G, Ergonul O, Ippolito G, Petersen E., 2020]. However, we found that, in contrast to viral pneumonia caused by the A/H1N1 influenza virus, where the phase of diffuse alveolar damage correlated quite clearly with the duration of the disease, with a new coronavirus infection there is no clear relationship between the duration of the disease and the identified morphological changes. Most likely, this can be explained by the erased onset of the disease and the presence of a period of asymptomatic course of the disease in some patients [Wei Cao, Taisheng Li., 2020]. It has been established that in the so-called therapeutic window (days 7–10 of the disease), the described signs correspond to the exudative stage of diffuse alveolar damage, therefore, the use of therapy aimed at improving respiratory function and correction of hypercoagulation can have a positive effect [Ling Lin, Lianfeng Lu , Wei Cao, Taisheng Li., 2020].

As the disease progresses, patients in critical condition develop organizing pneumonia and focal fibrosis, which was also observed in influenza A/H1N1 [Chernyaev A.L., et al., 2010]. Data on

systemic hypercoagulability in the new coronavirus infection, which are actively discussed in the literature, are confirmed in our study by the presence of diapedetic hemorrhages in various organs. Basically, they appear in the lungs in the form of focal hemorrhages and heart attacks in the presence of blood clots, mainly in the small branches of the pulmonary arteries and veins. In viral pneumonia caused by the A/H1N1 influenza virus, we have previously observed the formation of blood clots mainly in the branches of the pulmonary veins. In addition, the difference from influenza pneumonia is the detection of predominantly fresh hemorrhages. However, these data are preliminary and require analysis of more extensive material. Identified megakaryocytes in the capillaries of the interalveolar septa were previously described in acute respiratory distress syndrome caused by various factors, including viral etiology. According to the authors, megakaryocytes can participate in thrombocytogenesis, as well as contribute to thrombosis in various organs [Kungurova V.V., Khasanyanova S.V., 2015]. However, we consider it premature to conclude that coagulation disorders are observed mainly in the lungs, although the lungs are undoubtedly the main target organ in coronavirus infection [Yan Zhang, et.al., 2020]. The question of the dominant damage of type II alveolocytes by the SARS-CoV-2 coronavirus is discussed. Thus, earlier in an experimental study it was demonstrated that the SARS-CoV virus mainly affects type II alveolocytes, and not type I [Qian Z, Travanty EA, Oko L, et al., 2013]. These data, however, require confirmation for COVID-19, as we observed desquamation of type I alveolocytes in the early phase of diffuse alveolar injury. In this regard, we assume that most likely there is damage to both those and other cells. The issue of viral damage to lymphocytes, mainly CD4+ T cells, is discussed in the literature. Despite the absence of ACE2 receptors on lymphocytes, there is an assumption that the virus can enter the cell by membrane fusion and endocytosis, as a result of which some of the lymphocytes can die, presumably by apoptosis or pyroptosis. The cell fragments found in the cytoplasm of macrophages may be an indirect evidence of apoptosis of lymphocytes, but this requires further study. The appearance of hemorrhages and thrombi in small blood vessels in other organs is probably associated with viremia in severe patients, which leads to direct viral damage to endotheliocytes with subsequent development of infectious-toxic shock (hemorrhagic shock), which aggravates the course of COVID-19 [Qian Z, Travanty EA, Oko L, et al., 2013]. In the presented observations, bacterial pneumonia was detected in only two of the deceased, while in one of them it most likely arose as a complication of a viral one. In another observation, there was aspiration pneumonia, which was later complicated by viral pneumonia. Bacterial complications are not likely to be a common complication of COVID-19, but may occur in individuals on invasive ventilation. At the same time, it should be remembered that high titers of bacterial pathogens do not always indicate the development of a viral-bacterial infection [Xu Zhe, Shi Lei, Yijin Wang, Jiyuan Zhang, Lei Huang, Chao Zhang, et al., 2020]. No matter how the covid affects the entire body, it is still primarily a respiratory infection. Complaints of cough, shortness of breath, post-covid syndrome remain in 10% of people 12 weeks after the illness. Shortness of breath in this case can be both true and false. The first is associated with respiratory failure and damage to the lung tissue, and the second - with the subjective perception of the patient, that is, the so-called hyperventilation syndrome. If we talk about shortness of breath as part of respiratory failure, then it is usually the most difficult for patients to exhale. If we are talking about hyperventilation syndrome, then more often, on the contrary, it is difficult to take a breath.

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