(disorganization of the connective tissue, fibrinoid changes of basic substance of the connective tissue, generalized damage of the blood stream – vasculitis, lymphoid and plasma cell infiltrates, etc.); common character of individual clinical signs, especially at the initiatory stage of the disease (Raynaud's syndrome); systemic, multiple organ damage (joints, skin, muscles, kidneys, serous membranes, heart, lungs); general laboratory indicators of the inflammation activity; common group and immunological markers distinctive for each disease; general principlesof treatment (anti-inflammatory drugs, immunosuppression, extracorporeal cleansing methods and pulsed corticosteroid therapy in crisis situations).

Conclusions. Thus, the development mechanism of the systemic connective tissue diseases is not fully examined. However, the practical application of the diagnostic immunological markers of the disease and the determination of its activity will permit to improve the prognosis in these diseases.

THE PATHOPHYSIOLOGY OF 'HAPPY' HYPOXEMIA IN COVID-19

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Introduction. Despite all precautions, the spread of COVID-19 is getting worse every day. In addition, the lack of a proven, effective and safe method of treating the disease remains a key problem in the healthcare system. COVID-19 usually begins with damage to the epithelium of the upper respiratory tract and then spreads to the alveoli and lungs, leading to the development of pneumonia. Severe pneumonia, including those associated with COVID-19, is almost always accompanied by severe hypoxemia, which is a serious additional risk factor for an unfavorable course of the disease.

Aim. The aim of this review is to provide an overview of the current knowledge on the mechanisms of development of "silent" or "happy" hypoxemia in Covid-19.

Materials and methods. Data analysis of literature and Internet sources.

Results and discussion. "Happy hypoxia", or silent hypoxemia, is characterized by the fact that patients with low saturation levels do not have signs of suffocation, impaired consciousness and excessive work of breathing. The pathogenesis of "happy hypoxia" is a violation of the ventilation/perfusion ratio. It is known that the respiratory center first reacts to changes in pH and CO_2 levels and only after that to changes in PO_2 levels. Subsequently, as a result of the development of hypercapnia and respiratory alkalosis, the HbO2 dissociation curve shifts to the left, which leads to a discrepancy between PO_2 and SpO_2. The main cause of hypoxemia in most of both viral and bacterial pneumonias is shunting of blood in unventilated areas of the lungs. But with COVID-19 in the pathogenesis of hypoxemia, thickening of the alveolar-capillary membrane and disturbances in ventilation-perfusion ratios, associated with both numerous microthrombosis, and with a primary violation of hypoxic vasoconstriction. Severe shortness of breath almost always worsens violation of the mechanical properties of the lungs in severe pneumonia, whereas in COVID-19, its severity is far from always proportional to the degree of hypoxemia

Conclusions. Ventilation-perfusion mismatch, ranging from shunts to alveolar dead space ventilation, is the central hallmark and offers various therapeutic targets. A thorough understanding of the pathophysiological determinants of respiratory drive and hypoxemia may promote a more complete comprehension of a patient's clinical presentation and management.