CHARACTERISTICS STUDY OF THE SYSTEMIC CONNECTIVE TISSUE DISEASES DEVELOPMENT IN CHILDREN

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Introduction. Systemic connective tissue diseases (systemic lupus erythematosus, systemic scleroderma, diffuse fasciitis, idiopathic dermatomyositis, Sjogren's disease, mixed connective tissue disease (Sharp's syndrome), polymyalgia rheumatica, recurrent polychondritis, recurrent panniculitis (Weber-Christian disease) are classified as complex and urgent problems of medicine.

Detection frequency of this pathology in children is less significant in comparison with other diseases, their severity and severity cannot be underestimated. Systemic connective tissue diseases are characterized by a wide range of clinical manifestations and variability of the course, damage of many systems of the child's body, including the musculoskeletal system, which often leads to early disability and even death.

Aim – to study the features of the development and diagnosis of the systemic connective tissue diseases in children.

Materials and methods. Diagnosis of the systemic connective tissue diseases is quite difficult, whereby the laboratory diagnostic methods take the general place. It were performed the screening studies using an indirect immunofluorescence reaction (determination of antinuclear antibodies and antineutrophilic cytoplasmic antibodies).

These tests are characterized by high sensitivity to the systemic connective tissue diseases. Positive result of screening tests in patients requires further examination, and negative result confirm that the probability of the systemic connective tissue disease in this patient is very low and this diagnosis is highly improbable.

Results and discussion. Etiology of the systemic connective tissue diseases is considered from the autoimmunity multifactorial concept position, according to which the development of these diseases is stipulated by the infectious, genetic, endocrine and environmental factors interaction (that is, genetic predisposition and environmental factors such as stress, infection, hypothermia, insolation, trauma, and action of sex hormones, mainly female, pregnancy, abortion – systemic diseases of the connective tissue).

In systemic diseases of the connective tissue, there is the profound disorders of the immune homeostasis, expressed in the autoimmune processes development. The autoimmune process is based on the immunoregulatory imbalance, expressed in suppressive inhibition and increase of "helper" activity of T-lymphocytes, followed by the B-lymphocytes activity and autoantibodies overproduction of various specificities. In this case, the pathogenetic activity of autoantibodies is realized through the complement-dependent cytolysis, circulating and fixed immune complexes, interaction with cellular receptors and, as a result, it leads to the systemic inflammation development.

Thus, the common character of the systemic connective tissue diseases pathogenesis is violation of the immune homeostasis in form of the uncontrolled synthesis of the autoantibodies and formation of the immune antigen-antibody complexes circulating in blood and fixed in tissues, with the development of the severe inflammatory reaction (especially in the microcirculatory bloodstream, in joints, kidneys and other organs).

In addition to the common pathogenesis, all systemic connective tissue diseases are characterized by the following features: multifactorial type of predisposition with a certain role of the immunogenetic factors associated with the sixth chromosome; single morphological changes

(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.