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Sodikova Dilrabokhon Tozhidinovna.,

Department of GP 2

Andijan State Medical Institute

CLINICAL JUSTIFICATION OF ACUTE RESPIRATORY DISTRESS SYNDROME AND OTHER PULMONARY COMPLICATIONS IN COVID-19

Resume: Acute respiratory distress syndrome —ARDS) is an acute diffuse inflammatory lesion of the lung parenchyma that develops as a non-specific reaction to various damaging factors and leads to the formation of acute respiratory failure (ODN) (as a component of multiple organ failure) due to a violation of the structure of lung tissue and a decrease in the mass of aerated lung tissue

This article concludes that severe acute respiratory distress syndrome caused by the novel coronavirus-2 (COVID-19) is considered an emergency pandemic of respiratory diseases, the clinical picture of this infection often meets the criteria of acute respiratory distress syndrome (ARDS) with progressive stress leading to death.

Key words: COVID-19, acute respiratory distress syndrome,pulmonary complications.

Содиқова Дилрабохон Тожидиновна.,

Кафедра ВОП 2

Андижанский государственный медицинский институт

КЛИНИЧЕСКОЕ ОБОСНОВАНИЕ ОСТРОГО РЕСПИРАТОРНОГО ДИСТРЕСС-СИНДРОМА И ДРУГИХ ЛЕГОЧНЫХ ОСЛОЖНЕНИЙ ПРИ COVID-19

Резюме,:стрый респираторный дистресс-синдром (ОРДС) — остро возникающее диффузное воспалительное поражение паренхимы легких, развивающееся как неспецифическая реакция на различные повреждающие факторы и приводящее к формированию острой дыхательной недостаточности (ОДН) (как компонента полиорганной недостаточности) вследствие нарушения структуры легочной ткани и уменьшения массы аэрированной легочной ткани

В этой статье делается вывод о том, что тяжелый острый респираторный дистресс-синдром, вызванный новым коронавирусом-2 (COVID-19), считается чрезвычайной пандемией респираторных заболеваний, клиническая картина этой инфекции часто соответствует критериям острого респираторного дистресс-синдрома (ОРДС) с прогрессирующей нагрузкой, приводящей к смерти.

Ключевые слова: COVID-19, острый респиратор дистресс-синдром, легочная осложнения.

Relevance. New coronavirus infection (COVID-19) is a potentially dangerous acute respiratory disease caused by a new coronavirus (SARS-CoV-2), mainly with an aspiration transmission mechanism. COVID-19 can occur not only as a mild acute respiratory viral infection, but also in severe forms, which are characterized by the development of a clinical picture of acute respiratory distress syndrome (ARDS) and multiple organ failure with high mortality [1]. During the passing period of the pandemic, scientific information on the etiology, epidemiology, pathogenesis and morphological changes, clinical features, treatment and prevention of a new coronavirus infection is being updated [3].

The spread of COVID-19 is particularly dangerous in relation to the decompensation of chronic diseases. The most common severe forms of COVID-19 are observed in patients with chronic obstructive pulmonary disease, obesity, diabetes mellitus, hypertension, coronary heart disease, chronic kidney

disease, malignant neoplasms [6]. The pathogenesis of CID-19 is in the process of active study. In the domestic and foreign literature, it has been stated that the main cell receptor to which the S-protein (Spike with protein) of the SARS-coronavirus-2 shell binds is angiotensin converting enzyme 2 (angiotensin converting enzyme 2, ACE2 and). Infection occurs with the participation of transmembrane serine protease 2 (transmembrane protease serine 2, TMPRSS2), which is necessary for the activation of S-protein [4].

The main target of the SARS-2 virus is the respiratory tract. There is a lesion of alveolocytes of the 1st and 2nd types, vascular endothelial cells, which leads to disruption of the functioning of the aerogematic barrier and the surfactant alveolar complex [15, 16]. One of the most relevant pathogenetic concepts of COVID-19 is immune dysfunction (dysregulation), which is based on macrophage activation syndrome (macrophage activation syndrome, MAS) [7].

Morphological signs of COVID-19 at the present stage are mainly reduced to the description of changes in the early (exudative) and late (proliferative) stages of ARDS. Damage to endotheliocytes of the microcirculatory bed with disorders in the blood coagulation system, the development of DIC syndrome with multifocal microthrombosis and subsequent multi-organ dysfunction with a predominance of acute renal failure is also verified [5]. Some experts believe that with regard to the definition of lung damage in COVID-19, the term "pneumonia" does not reflect the morphology, clinical and radiological signs of the pathological process observed in lung damage by the SARS-coronavirus-2 virus at all.

The term "viral lung lesion" (viral pneumonitis, viral interstitiopathy) is proposed for use [3]. A number of authors suggest the term "microvascular obstructive thromboinflammatory lung syndrome" as a new name for severe COVID-19 [2].

The purpose of the study. To study the pathomorphogenesis of COVID-19 on the basis of autopsy studies with the formation of a working hypothesis of the conceptual scheme of clinical and morphological phases of the development of the disease.

Materials and methods of research. Retrospective, single-center, controlled, non-randomized. 25 patients met the inclusion criteria, 10 underwent artificial lung ventilation, three of them died. We compared the ARDS clinic for nonspecific severe community-acquired pneumonia and new coronavirus infection COVID-19.

The results of the study. The revealed features of diffuse alveolar damage in a new coronavirus infection (COVID-19) allowed us to present a working hypothesis of the pathomorphogenesis of COVID-19 interstitial pneumonia. We propose three phases — fulminant, persistent and fibrotic, each of which is conditionally limited by certain time parameters and is characterized by certain morphological features. Dysregulatory activation of monocytic phagocytes, the development of generalized microcirculatory thrombosis, pathological repair, progressive interstitial and intraalveolar fibrosis are the main links in the pathomorphogenesis of COVID-19-interstitial pneumonia.

In response to the introduction of the ATYPICAL pneumonia-2 virus, T-cell immunity reactions prevail in the exudative and proliferative stages. In the fibrotic stage, the total number of T-lymphocytes is sharply reduced, humoral immunity cells are not detected. The prevalence of CD8+ T-suppressor lymphocytes over the content of CD4+ T-helper lymphocytes may be associated with the mechanisms of autoimmune damage.

Lung damage with the development of COVID-19-interstitial pneumonia is the main cause of the severe course of the disease and fatal outcomes. The revealed features of the pathomorphogenesis of the clinical and morphological phases of COVID-19-interstitial pneumonia will improve the quality of diagnosis and treatment of a new coronavirus infection (COVID-19).

Conclusion. The basis of lung damage in the new coronavirus infection (COVID-19) is the development of ARDS (diffuse alveolar injury) with an atypical course, causing the development of COVID-19 - interstitial pneumonia with synchronous damage to the respiratory tract and microcirculatory bed.

Morphological signs of the fulminant phase of the progressive severe course of COVID-19 interstitial pneumonia, leading to a rapid fatal outcome (up to 10 days), correspond to the exudative stage of ARDS in combination with monocyte-macrophage hyperimmune reaction and the development of obstructive thromboinflammatory processes in the microcirculatory bed of the lungs, or are generalized.

Morphological signs of the persistent phase of the progressive severe course of COVID-19-interstitial pneumonia, leading to a fatal outcome (up to 20 days), correspond to the proliferative stage of ARDS. In this phase, there is a persistence of changes in the exudative stage in combination with monocyte-macrophage hyperimmune reaction, the development of generalized obstructive thromboinflammatory processes not only in the microcirculatory bed, but also in larger vessels, as well as common thrombosis and thromboembolic complications.

Morphological signs of the fibrotic phase of the progressive severe course of COVID-19-interstitial pneumonia leading to death (from 21 to 45 days) correspond to the fibrotic stage of ARDS with dysregenerative metaplastic and dysplastic changes, multiplicative sharply accelerated fibrosis effect and fibrotic remodeling of the pulmonary parenchyma.

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