CLINICAL AND FUNCTIONAL STATUS OF THE CARDIOVASCULAR SYSTEM IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE WITH COVID-19

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Annotation *In December 2019, the first cases of a new coronavirus infection were recorded, which was subsequently declared a pandemic. It has been established that COVID-19 is much more difficult for patients with chronic obstructive pulmonary disease. Despite its increasing prevalence, COPD remains a pathology that is poorly diagnosed and treated. There are good reasons to believe that treatment of COPD does not generally meet current guidelines. That is why the potentially dangerous combination of a new infectious disease and chronic obstructive pulmonary disease has become an important medical problem.*

Key words: *COVID-19, pandemic, cardiovascular system, COPD.*

On December 31, 2019, the first report of cases of pneumonia of unknown etiology appeared in Wuhan (PRC). On January 9, 2020, the Chinese Center for Disease Control and Prevention announced the discovery of the agent causing these pneumonias. It turned out to be a new severe acute respiratory syndrome coronavirus type 2 (SARS-CoV-2). The disease that this virus causes is called COVID-19. On March 11, 2020, the World Health Organization declared the beginning of the COVID-19 pandemic.

Patients with COPD are at highest risk of adverse outcomes from COVID-19. There is no scientific evidence that ICS should be discontinued in patients with COPD during the COVID-19 pandemic. Patients with COPD should maintain regular therapy. Oxygen therapy should be provided as needed, according to standard recommendations.

Published data from a meta-analysis, which included six studies (n-1558), two of which assessed the chance of admission to the intensive care unit (ICU), and four assessed the severity of clinical symptoms, revealed that the presence of COPD in patients with covid infection increases the risk of progression and severe course of COVID-19 is 6 (5.97) times; Arterial hypertension is 2.29, diabetes mellitus is 2.47 [2]. Another European study showed that among those hospitalized in the clinic, half of the patients with severe cases of the new coronavirus infection developed ARDS and 20% of them had concomitant COPD [3]. However, COPD itself is not an independent factor in SARS-Cov2 infection.

There is no reason to change the basic treatment provided, much less to de-escalate therapy during the COVID-19 pandemic. Moreover, a study that analyzed the treatment of patients with bronchial asthma (BA) and COPD (77% and 23%, respectively, n -7578) showed an increase in adherence to control therapy during the COVID-19 pandemic by

14.5% [4]. (l.Kayl, et al. J. Allergy Clin Immunol Pract 2020) This indicates increased patient discipline and a desire to prevent possible complications.

There are no grounds for restricting and canceling ICS if they are used (according to justified 15Journal "Interscience" No. 20 (196), part 2, 2021 indications) in the basic therapy of COPD. If during an exacerbation there is a need to use SGCS, then their prescription is carried out in accordance with clinical recommendations. In the case of a new coronavirus infection in a patient with COPD, drug interactions must be taken into account.

According to the literature, the incidence of thromboembolism of various locations in patients with COVID-19 hospitalized in intensive care units is 20-30%, venous thromboembolism - 47%, including pulmonary embolism, deep vein thrombosis of the lower and upper extremities [5].

Coagulopathy in COVID-19 is always accompanied by an increase in D-dimer; its high level is associated with an unfavorable prognosis of the disease, and D-dimer above 2.0 mg/l may be a predictor of an unfavorable outcome [6].

Data from pathological studies have shown that with COVID-19, various types of thrombosis are observed: arterial, venous, at the level of the microvasculature (microthrombosis) [7]. The pathogenesis of hemostasis disorders in patients with COVID-19 infection includes both local damage to the vascular endothelium (including in the lungs) with platelet activation and aggregation, increased platelet consumption (thrombocytopenia), and systemic hyperfibrinogenemia. Lung damage from COVID-19 is accompanied by the release of large amounts of proinflammatory cytokines, causing endothelial damage and activating the coagulation system.

Factors that accelerate blood clot formation in COVID-19 include:

1) induction of a “cytokine storm”:

• interleukins IL-1b and IL-6 stimulate the expression of tissue thromboplastin on immune cells,

• activation of platelets, binding them to damaged endothelium;

2) dysfunction of endothelial cells (accelerates the thrombotic reaction);

3) suppression of fibrinolysis by suppressing the activity of urokinase-type plasminogen activator, increasing the release of plasminogen activator inhibitor-1.

Thus, the data obtained allow us to conclude:

1) SARS-COV-2 infection is associated with hypercoagulation against the background of systemic inflammation;

2) the main characteristics of COVID-19-associated coagulopathy are increased levels of D-dimer and fibrinogen, slight changes in the levels of activated partial thromboplastin time, prothrombin time and platelet count;

3) high D-dimer levels are associated with increased mortality;

4) the severity of coagulopathy correlates with the severity of the disease;

5) prevention of thromboembolic complications is indicated for all patients with COVID-19-associated pneumonia.

Of course, patients with COPD during the COVID -19 pandemic are under the close attention of medical workers; they need to be fully provided with basic therapy medications,

draw up an action plan in case of deterioration of the condition, but the patients themselves must carefully adhere to basic therapy and follow all recommendations for prevention of SARS-Cov-2 infection.

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