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Original article

DYNAMICS OF INFLAMMATORY MARKERS OVER SIX MONTHS IN PATIENTS WITH ASTHMA WHO UNDERWENT COVID-19

T.G. Lobova. T.I. Vitkina

Abstract

Background. Asthma is a chronic progressive heterogeneous respiratory disease accompanied by the development of a systemic inflammatory response. Due to the widespread prevalence of the novel coronavirus infection, there is concern about the impact of COVID-19 on patients with asthma. Current evidence suggests that SARS-CoV-2 activates the inflammasome response, triggering a hyperinflammatory reaction in patients with asthma, but the long-term consequences are still insufficiently studied.

Purpose. To study the features of the systemic inflammatory process over six months after SARS-COV-2 infection in individuals with asthma.

Materials and methods. In patients with mild, partially controlled asthma after mild COVID-19 infection (n = 52) and individuals with asthma (n = 34) who did not contract COVID-19, an assessment of inflammatory markers in peripheral blood was performed. Patients were examined immediately after recovery and confirmation of a negative SARS-COV-2 test result; one month, three months, and six months after the infection. The control group included conditionally healthy volunteers (n=15) who did not contract COVID-19. The cellular composition of peripheral blood, ESR, CRP, D-dimer levels were determined by classical methods; interleukin concentrations: (IL)-1β, IL-6, IL-18, NOD-like receptor protein 3 (NLRP3) inflammasome, gasdermin D (GSDMD) were measured by ELISA methods.

Results. It was revealed that for all studied parameters, there is a slow decrease in the level of indicators by six months. The levels of GSDMD, platelets, IL-1β, D-dimer, ESR, IL-6, NLRP3 after six months do not reach the values of the control group, which indicates a persistent hyperinflammatory response of the immune system.

Conclusion. Activation of NLRP3 and gasdermin D by the SARS-CoV-2 virus directly or indirectly can lead to dysregulation of inflammasome-mediated mecha-

nisms, triggering and maintaining a hyperinflammatory reaction for six months, prolonging the recovery process in patients with asthma. The study of these processes will expand the understanding of the pathological mechanism. This will allow the development of new therapeutic strategies for asthma patients who have suffered from the novel coronavirus infection.

Keywords: asthma; COVID-19; long-term consequences; inflammatory markers; NLRP3; gasdermin D

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Научная статья

ДИНАМИКА МАРКЕРОВ ВОСПАЛЕНИЯ В ТЕЧЕНИЕ ШЕСТИ МЕСЯЦЕВ У ПАЦИЕНТОВ С БРОНХИАЛЬНОЙ АСТМОЙ, ПЕРЕНЕСШИХ COVID-19

Т.Г. Лобова, Т.И. Виткина

Аннотация

Обоснование. Бронхиальная астма – хроническое прогрессирующее гетерогенное респираторное заболевание, сопровождающееся развитием системной воспалительной реакции. В связи с широкой распространенностью новой коронавирусной инфекции существует обеспокоенность по поводу воздействия COVID-19 на пациентов с бронхиальной астмой. Современные данные свидетельствуют о том, что SARS-CoV-2 активирует ответ инфламмасом, вызывая гипервоспалительную реакцию у пациентов с бронхиальной астмой, но долгосрочные последствия все еще недостаточно изучены.

Цель работы. Изучить особенности системного воспалительного процесса в течение шести месяцев после заражения SARS-COV-2 у лиц с бронхиальной астмой.

Материалы и методы. У пациентов с легкой, частично контролируемой бронхиальной астмой после легкой формы заражения COVID-19 (n = 52) и лиц с бронхиальной астмой (n = 34), которые не были инфицированы COVID-19, была проведена оценка маркеров воспаления в периферической крови. Пациенты были обследованы сразу после выздоровления и подтверждения отрица-

тельного результата теста на SARS-COV-2; через месяц, три месяца и шесть месяцев после заражения. В контрольную группу вошли условно здоровые добровольцы (n=15), которые не заразились COVID-19. Клеточный состав периферической крови, СОЭ, уровень СРБ, D-димера определяли классическими методами; концентрацию интерлейкинов: (IL)-1β, IL-6, IL-18, инфламмасомы NOD-подобного рецептора белка 3 (NLRP3), газдермина D (GSDMD) определяли методами иммуноферментного анализа.

Результаты. Было выявлено, что по всем изучаемым параметрам наблюдается медленное снижение уровня показателей к шести месяцам. Уровни GSDMD, тромбоцитов, IL-1β, D-димера, СОЭ, IL-6, NLRP3 через полгода не достигают значений контрольной группы, что свидетельствует о стойкой гиперинфекционной реакции иммунной системы.

Заключение. Активация NLRP3 и gasdermin D вирусом SARS-CoV-2 прямо или косвенно может привести к нарушению регуляции механизмов, опосредованных инфламмасомами, вызывая и поддерживая гипервоспаление реакцию в течение шести месяцев, продлевая процесс выздоровления у пациентов с бронхиальной астмой. Изучение этих процессов расширит понимание патологического механизма. Это позволит разработать новые терапевтические стратегии для пациентов с астмой, которые пострадали от новой коронавирусной инфекции.

Ключевые слова: бронхиальная астма; COVID-19; отдаленные последствия; маркеры воспаления; NLRP3; газдермин D

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Introduction

Asthma represents a global health problem that affects from 1% to 18% of the population. Asthma is a chronic lung disease characterized by systemic inflammation, mucus hyperproduction, hyperreactivity, bronchial remodeling, and resistance to applied treatment methods [5].

During the widespread prevalence of the novel coronavirus infection caused by the SARS-CoV-2 virus, patients with asthma require special attention, as asthma is associated with reduced antiviral protection and disturbances in the functioning of the immune system. Due to the fact that COVID-19 also affects the respiratory organs, it is logical to assume that patients with asthma are most vulnerable to COVID-19. The proven role of respiratory viral infections as both

triggers and inducers of the disease raises concerns about the increased susceptibility of asthma patients to coronavirus infection. The question of how the recovery process after COVID-19 proceeds in patients with asthma remains open. It has been shown that some patients suffer from long-term persistent shortness of breath and weakness, which greatly reduces the quality of life and requires prolonged rehabilitation.

SARS-CoV-2 enters host cells through angiotensin-converting enzyme-2 (ACE-2) receptors. ACE2 is a protein that performs various roles, such as a catalyst, amino acid transporter, or viral receptor. ACE2 receptors are ubiquitously expressed on cells of the gastrointestinal tract, blood vessels, and in large numbers in the lungs [6]. This increases the percentage of virus penetration into lung tissue and the circulatory system, causing the activation of immunocompetent cells and polyprotein complexes, such as inflammasomes, which in turn trigger a cascade of reactions via caspase-1 with the formation of proinflammatory cytokines.

The novel coronavirus infection caused by SARS-CoV-2 is characterized by a systemic hyperinflammatory response, elevated levels of circulating inflammatory markers, including C-reactive protein (CRP), D-dimer, and a wide range of inflammatory cytokines associated with an unfavorable disease outcome. The proinflammatory cytokines IL-1β and IL-18 play a significant role in lung damage and respiratory distress caused by SARS-CoV-2 (Conti et al., 2020; Satis et al., 2021). IL-1β plays an important role in attracting immune cells to the site of infection, as well as in stimulating cells to produce and secrete a number of inflammation mediators, including IL-6 and C-reactive protein (Slaats et al., 2016). IL-18 is associated with cytopenia, disease severity, being one of the leading cytokines of macrophage activation syndrome [7].

The production of IL-1β and IL-18 depends on the activation of inflammasomes upon perception of molecular patterns associated with PAMPs/DAMPs by intracellular sensors of innate immunity (Martinon et al., 2002; Sharma and Kanneganti, 2016). Inflammasomes activate a class of caspases known as inflammatory caspases [8; 9]. Their main substrates are cytokines (IL-1β, IL-18), which are important mediators of the hyperinflammatory reaction. There is evidence of both the triggering of the inflammasome response in the acute course of SARS-CoV-2 [10; 11] and the significance of this mechanism in the aggravation of BA. However, at present, the dynamics of the attenuation of the hyperinflammatory process in patients with asthma who have suffered from COVID-19 remains unclear.

Purpose. To study the features of the systemic inflammatory process over six months after SARS-COV-2 infection in individuals with BA.

Materials and methods

The study included 101 people (age 42±5 years), of which 85 were patients with asthma, and 15 people were included in the control group. The inclusion criteria were the presence of mild, partially controlled asthma, without exacerbation; a previous novel coronavirus infection confirmed by a PCR test. The exclusion criteria were the presence of chronic diseases of internal organs in the acute stage, diabetes mellitus, oncological diseases, and bad habits in the subjects.

The main group (52 people) included individuals with mild, partially controlled asthma who received personalized basic therapy with glucocorticoids (ICS) and symptomatic (for symptom relief) therapy, and who had a positive PCR result for SARS-CoV-2. The novel coronavirus infection was mild in severity.

The comparison group consisted of 34 people with BA who did not contract SARS-CoV-2; at the time of the examination, a negative PCR result was detected. The group was matched by sex, age, and received therapy. The control group was represented by 15 conditionally healthy volunteers who did not contract COVID-19. The examination was performed at different time periods: 0 - early (after recovery and confirmation of a negative SARS-CoV-2 test result), then after 1, 3, and 6 months. At the first visit, participants answered questions from a questionnaire about the course and state of the main disease - asthma, as well as questions including the timing of infection with the novel coronavirus infection, a description of the condition (complaints), prescribed medications, and allergic reactions. Informed consent was obtained from all participants in accordance with the requirements of the Declaration of Helsinki (2013) according to a protocol approved by the Biomedical Ethics Committee (Protocol No. 15/2021 dated 27.12.2021).

Throughout the study period, testing for IgM to SARS-CoV-2 was performed by the enzyme immunoassay method using Vector-BEST kits and a VERP 2000 analyzer (SIEMENS, Germany). Control sera that did not contain IgM to SARS-CoV-2 were used in the work.

Biochemical, hematological, and coagulological studies were performed directly on the day of biomaterial collection. For enzyme immunoassay studies, the selected blood serum was stored at a temperature of minus 80°C for no more than 3 months. Samples with hemolysis and high lipid content were excluded. The content of hematological parameters of peripheral blood was determined using a Mindray 6800 analyzer (China). Morphological changes in blood cells were determined by the microscopic method using an Axioscope

5 microscope (Carl Zeiss, Germany) at magnifications of x10 and x100. The content of C-reactive protein (CRP) was determined using Rendox FN3452 and CP 7950 test systems on a Sapfir 500 biochemical analyzer (Japan). D-dimer was determined using a TS-190 analyzer (China) with reagents from Technologia-Standard (Russia). The content of the NOD-like receptor protein 3 (NLRP3) inflammasome (BIOMATIK ELISA Kit, Israel), gasdermin D (GSDMD) (Human GSDMD Simple Step ELISA Kit Abcam, UK), and interleukins (IL) 1β, 6, and 18 in peripheral blood serum (Vector-BEST, Russia) was determined by ELISA on a VERP 2000 analyzer (SIEMENS, Germany).

Statistical analysis

Statistical processing was performed using the Statistica 10.0 program (Statsoft). Data were presented as median and quartiles. The statistical significance of differences between groups was assessed using the Kruskal-Wallis test. The critical level of significance (p) when testing statistical hypotheses was accepted at $p < 0.05$.

Results

In the group of patients with BA, compared to the control group, an increase in a number of inflammatory markers was observed: gasdermin D by 1076% ($p < 0.001$), CRP by 215% ($p < 0.05$), D-dimer by 214% ($p < 0.05$), ESR by 167% ($p < 0.05$), and monocytes by 114% ($p < 0.05$). The cytokine status was also characterized by high values: IL-18 increased by 141% ($p < 0.05$), IL-6 by 124% ($p < 0.05$), and IL-1β by 116% ($p < 0.05$) (Fig. 1).

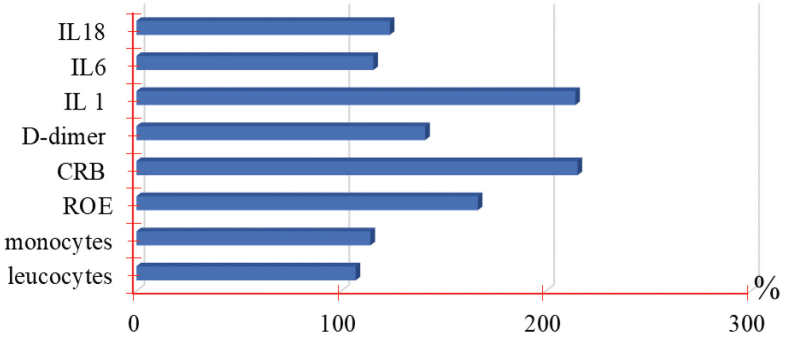

Fig. 1. Markers of inflammation in patients with asthma.

Note: the indicators are presented as a percentage relative to the control group, the parameters of which are taken as 100%.

In patients with asthma who suffered from the novel coronavirus infection (COVID-19), in the early period (after recovery and confirmation of a negative test result for the presence of IgM SARS-CoV-2), compared to patients with asthma who did not contract the novel coronavirus infection, an increase in the following indicators was detected: D-dimer by 300% ($p < 0.001$), ESR by 183% ($p < 0.05$), and CRP by 139% ($p < 0.05$). An increase in the level of interleukin IL-1β by 234% ($p < 0.001$) was shown in Fig. 2.

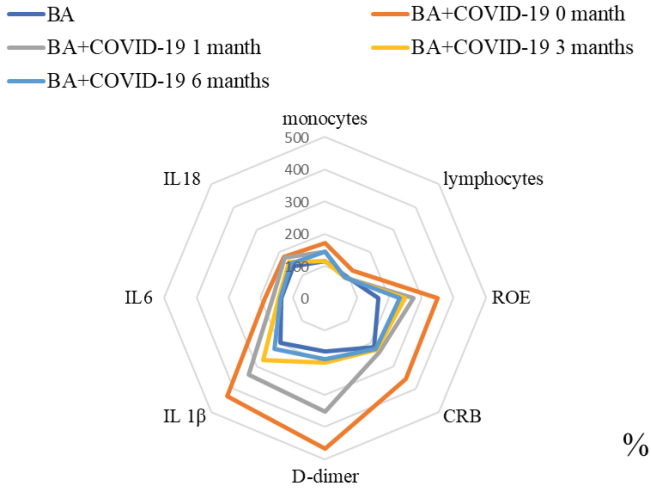

Fig. 2. Dynamics of inflammation markers in 0, 1, 3, 6 months patients with asthma who underwent coronavirus infection.

Note: the indicators are presented as a percentage relative to the control group, the parameters of which are taken as 100%.

One month after the recovery of patients with asthma who suffered from COVID-19, in comparison with BA + COVID-19 at 0 months of recovery from the infection, a slow decrease in indicators was observed: D-dimer by 115% ($p < 0.05$), ESR by 75% ($p < 0.05$), and CRP by 116% ($p < 0.05$). A decrease in interleukin levels was noted: IL-1β by 234% ($p < 0.001$), IL-6 by 49% ($p < 0.05$), and IL-18 by 39% ($p < 0.05$).

Three months after recovery, in patients with BA who suffered from COVID-19, compared to the previous period (BA+COVID-19 1 month), a gradual decrease in parameters was also observed: D-dimer by 152% ($p < 0.05$), ESR by 20% ($p < 0.05$), and CRP by 16% ($p < 0.05$). A decrease in interleukin

levels was noted: IL-1β by 64% ($p < 0.05$), IL-6 by 20% ($p < 0.05$), and IL-18 by 16% ($p < 0.05$).

In patients with BA who suffered from coronavirus infection, six months after the infection, compared to the previous period (BA+COVID-19 3 months), inflammatory markers continued to decrease: D-dimer by 10% ($p < 0.05$), ESR by 17% ($p < 0.05$), and CRP by 5% ($p < 0.05$). A decrease in interleukin levels was detected: IL-1β by 48% ($p < 0.05$), IL-6 by 3% ($p < 0.05$), and IL-18 by 9% ($p < 0.05$).

The decrease in a number of parameters, such as D-dimer, ESR, CRP, IL-1β, IL-6, and IL-18, over six months occurred slowly, not reaching the values of the group of patients with BA who did not contract coronavirus infection by six months.

The level of NLRP3 was higher by 135% ($p < 0.001$) and gasdermin D by 976% ($p < 0.001$) in patients with BA compared to the indicators of patients in the control group. In patients with BA+COVID-19 at 0 months, relative to patients with BA, the level of NLRP3 was increased by 503% ($p < 0.001$) and gasdermin D by 892% ($p < 0.001$). Over time, in patients with BA+COVID-19 at 1 month compared to the previous period, NLRP3 decreased by 142% ($p < 0.001$) and gasdermin D by 394% ($p < 0.001$). Three months after recovery, compared to patients with BA+COVID-19 at 1 month, NLRP3 decreased by 78% ($p < 0.05$) and gasdermin D by 121% ($p < 0.05$). After six months, NLRP3 decreased by 78% ($p < 0.05$) and gasdermin D by 121% ($p < 0.05$) compared to patients with BA+COVID-19 at 3 months (Fig. 3).

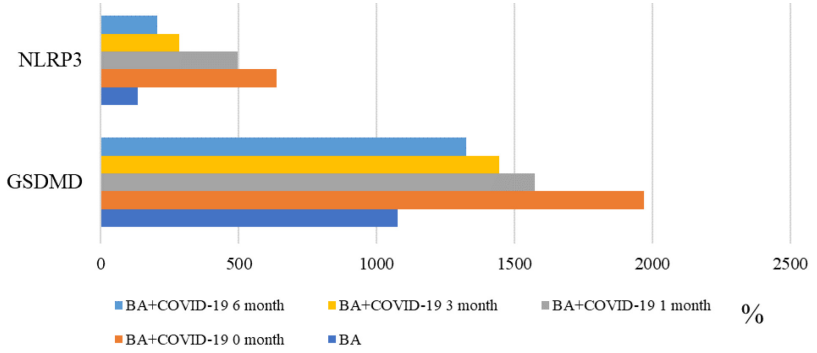

Fig. 3. Dynamics of NLRP3 and gasdermine D in patients with Asthma who suffered a new coronavirus infection.

Note: the indicators are presented as a percentage relative to the control group, the parameters of which are taken as 100%.

There was a tendency towards a decrease in NLRP3 and gasdermin D at the decreed time points (one month, three months, six months) in patients with BA who suffered from the novel coronavirus infection, but the values after six months exceeded the level of these parameters in the group of patients with BA who did not contract COVID-19.

Discussion

BA, being a chronic, progressive lung disease, requires special attention when various respiratory infections are attached [12]. In the case of the novel coronavirus infection in individuals with BA, such important systems as the respiratory, cardiovascular, nervous system, and general metabolic processes are affected, and a hyperinflammatory response may develop [13]. The multifactorial inflammatory profile of BA is superimposed by the reaction of the immune system to the novel coronavirus infection, creating conditions for the mass death of immunocompetent cells [14]. In the presence of an elevated CRP concentration, the activity of immune system cells with cytotoxic properties increases, phagocytosis accelerates, the complement system comes into play, and metabolic disorders increase. This forms the prerequisites for a long recovery and, in some cases, the development of post-COVID syndrome.

The conducted study showed that in patients with asthma who have suffered from a novel coronavirus infection, the manifestations of a hyperinflammatory reaction are more active for 6 months than in individuals with asthma who have not contracted COVID-19. The process of recovery of indicators is slow and does not reach the values in patients with BA. The issue of risks and prognosis for patients with bronchial asthma in the long-term period after COVID-19 infection requires further study.

Conclusion

In patients with BA who have suffered from COVID-19, the inflammatory process proceeds much more actively than in patients with BA who have not contracted coronavirus infection. The revealed violations of the pool of immunocompetent cells that provide protective mechanisms of innate and acquired immunity demonstrate the presence of a hyperinflammatory process, indicating a high level of hematopoietic activity of the bone marrow. The hyperinflammatory process is largely supported by the activity of NLRP3 and gasdermin D, providing an active inflammatory profile in this category of patients after six months.

Ethics Committee Conclusion. The clinical study was approved by the Ethics Committee (Protocol No. 15/2021 dated 27.12.2021).

Informed Consent. Informed consent was obtained from all subjects who participated in the study.

Conflict of Interest Information. The authors declare that the presented article, its topic, subject, and content do not affect competing interests.

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