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**INNOVATIVE DEVELOPMENT
OF SCIENCE, TECHNOLOGY
AND EDUCATION**



**PROCEEDINGS OF VI INTERNATIONAL
SCIENTIFIC AND PRACTICAL CONFERENCE
MARCH 14-16, 2024**

**VANCOUVER
2024**

UDC 001.1

The 6th International scientific and practical conference “Innovative development of science, technology and education” (March 14-16, 2024) Perfect Publishing, Vancouver, Canada. 2024. 445 p.

ISBN 978-1-4879-3792-8

The recommended citation for this publication is:

Ivanov I. Analysis of the phaunistic composition of Ukraine // Innovative development of science, technology and education. Proceedings of the 6th International scientific and practical conference. Perfect Publishing. Vancouver, Canada. 2024. Pp. 21-27. URL: <https://sci-conf.com.ua/vi-mizhnarodna-naukovo-praktichna-konferentsiya-innovative-development-of-science-technology-and-education-14-16-03-2024-vankuver-kanada-arhiv/>.

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MEDICAL SCIENCES

PECULIARITIES OF INFLAMMATORY MARKERS IN PATIENTS WITH COMMUNITY-ACQUIRED PNEUMONIA ASSOCIATED WITH CORONAVIRUS INFECTION

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Introduction.

In 2003, a virus that caused severe acute respiratory syndrome (SARS-CoV) was detected in the Middle East, and nine years later, the Middle East respiratory syndrome coronavirus (MERS-CoV) caused a new epidemic. Currently, the main threat is the SARS-CoV-2 virus (severe acute respiratory syndrome, coronavirus-2), which appeared in December 2019. The first outbreak of this respiratory virus occurred in the Chinese city of Wuhan, from where the infection quickly spread to Southeast Asia, Europe, and other countries. In March 2020, the World Health Organization officially declared COVID-19 a pandemic, and many countries began to impose strict quarantine measures (Chen Y., 2020; N. Zhu, D. Zhang, W. Wang, 2019).

Respiratory viruses belong to different families and differ in how easily they spread (transmissibility) and how they spread. Understanding the relative contribution of different transmission routes is critical to evaluating the effectiveness of non-pharmacological interventions in a population. The primary mechanism of

transmission of SARS-CoV-2 is airborne, typically by airborne droplets and household transmission (Lipsitch M., Swerdlow D. L., Finelli L., 2020; Lamers M. M., Haagmans B. L., 2022).

Patients with COVID-19 with hypoxic respiratory failure show signs of systemic hyperinflammation, including the release of inflammatory cytokines such as interleukin-1 (IL), IL-6, tumor necrosis factor- α , and increased levels of the acute-phase inflammatory protein C-reactive protein (CRP). The release of inflammatory cytokines in combination with fibrin synthesis by fibroblasts and subsequent formation of hyaluronic membranes in the lung tissue, as well as respiratory failure caused by damage to type 2 alveolar cells, are considered to be the main pathological mechanisms leading to severe impairment of visceral function. Despite the fact that SARS-CoV-2 causes acute lung damage, the disease should be considered a systemic disease because the vascular endothelium is involved; COVID-19-associated pneumonia is characterized by pulmonary infiltration by macrophages and neutrophils, which causes diffuse alveolar damage. (Chen R., 2020; M. Ciotti, M. Ciccozzi, A. Terrinoni, 2020).

The aim of the study was to determine the peculiarities of inflammatory markers in patients with community-acquired pneumonia associated with coronavirus infection.

Materials and methods. An open, prospective, observational study was conducted to achieve the goal and solve the problems. In the period from January 2021 to February 2022, 256 patients with community-acquired pneumonia (CAP) aged 40 to 65 years were examined at the outpatient clinic of the Kherson City Clinical Hospital named after Athanasius and Olga Tropin of the Kherson City Council, of whom 177 were associated with SARS-CoV-2 and 79 tested negative for coronavirus infection. In addition, 35 healthy volunteers were examined on an outpatient basis. To participate in the study, patients signed the Voluntary Informed Consent to Participate in the Study form. All patients were carefully examined for compliance with the inclusion/exclusion criteria. The diagnosis of community-acquired pneumonia was verified based on the adapted evidence-based

clinical practice guideline Community-Acquired Pneumonia in Adults, 2019. COVID-19 was diagnosed according to Order No. 722 of the Ministry of Health of Ukraine dated 03/28/2020 as amended by Order No. 2122 of the Ministry of Health of Ukraine dated 09/17/2020.

Study inclusion criteria: male and female patients aged 40 to 65 years; community-acquired pneumonia; informed consent of the patient to participate in the study.

Exclusion criteria for the study: Pregnant women; uncontrolled arterial hypertension; hypertension of the third stage; decompensated diabetes mellitus; congenital and acquired hemodynamically significant heart defects; chronic heart failure of the second and third stages; oncological diseases; lung damage of more than 75% according to CT scan; contraindications to the administration of drugs and their components; alcohol dependence, drug addiction, mental disorders; patient refusal to participate in the study.

Determination of interleukin-6, interleukin-10, and hsCRP was performed in blood plasma by enzyme-linked immunosorbent assay using standardized kits: "HF CRP-ELISA-Best", "IL-6-ELISA-Best", "IL-10-ELISA-Best" according to the attached instructions, in the certified laboratory of the Kherson City Clinical Hospital named after Athanasius and Olga Tropin of the Kherson City Council.

Statistical processing of the data obtained during the study began with descriptive statistics, including the calculation of the median and interquartile range (Me [Q25; Q75]), and the size of the analyzed subgroup (n). When testing statistical hypotheses, the null hypothesis was rejected at a statistical significance level of $p < 0.05$, which corresponds to the values accepted in biomedical research.

Results. During the screening, the main clinical characteristics of the examined persons were determined by taking anamnesis. The groups of subjects were comparable in age and social status.

The level of HF-CRP between the groups of patients was significantly higher by 27.5% in the group of patients with NHF with COVID-19 than in patients with NHF without COVID-19 - 17.20 [12.60; 19.70] mg/L vs. 13.50 [9.70; 19.00] mg/L,

respectively ($p < 0.05$). The median values of this indicator were significantly higher in both groups of patients compared to the value of 1.20 [0.70; 1.40] mg/l in the group of healthy volunteers ($p < 0.05$). IL-6 was the highest in the group of patients with UC with COVID-19 - 9.00 [7.78; 9.94] pg/mL and was significantly higher by 9.5% compared to the group of patients with UC without COVID-19 - 8.22 [6.94; 9.27] pg/mL and 3.8 times higher compared to the value of 2.40 [1.30; 3.10] pg/mL in practically healthy volunteers ($p < 0.05$). The value of IL-6 was also 22.7% higher in the group of patients with UC without COVID-19, 8.22 [6.94; 9.27] pg/ml versus 2.40 [1.30; 3.10] pg/ml in practically healthy volunteers ($p < 0.05$). Correlation analysis revealed the following relationships between the parameters: a direct relationship between IL-6 and hsCRP ($R = +0.43$, $p < 0.05$) and a direct relationship between IL-6/IL-10 and hsCRP ($R = +0.35$, $p < 0.05$). Whereas there was no significant correlation between IL-10 and hsCRP levels.

Our data are in line with the results of other studies showing that C-RB is the biomarker that correlates most strongly with the progression of COVID-19 and is significantly increased at an early stage of inflammation. According to L. Wang, C-RB is an independent discriminator of disease severity, which indicates its diagnostic value for patients with COVID-19, and its determination may be useful in clinical practice (Ali N., 2020; Wang L., 2020).

Thus, the immune system responds to the development of pneumonia, and changes in the cytokine profile depend on the pathogen, as the levels of inflammatory response markers in patients with community-acquired pneumonia associated with COVID-19 are significantly higher. It is promising to study the possibility of using inflammatory mediators to determine the course of community-acquired pneumonia associated with coronavirus infection.