

## THE CLINICAL COURSE OF COVID-19 PNEUMONIA

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**Annotation.** Plague and smallpox have been replaced by dangerous viral infections in the new century. The study of coronavirus allows to development of more effective methods of diagnostics, treatment, and vaccination, which contributes to public health.

**Key words:** coronavirus, COVID-19 pneumonia.

**Introduction.** The disease associated with novel coronavirus was originally called Novel coronavirus-infected pneumonia, NCIP. The World Health Organization (WHO) renamed it COVID-19, which is an acronym for coronavirus disease 2019. March 11, 2020. WHO declared COVID-2019 a pandemic caused by SARS-CoV-2 RNA virus and an international health emergency [1, 2, 3].

As of April 8, 2020, the epidemic has spread to more than 199 countries and more than 1 million people have contracted the virus worldwide with 81,478 deaths. [4, 6]. In mid-September 2020, more than 33 million people worldwide were affected by COVID-19, with more than 50 million cases in early November; more than 1.263 million people have died. However, these numbers are likely underestimates because not all patients are tested, especially those who are asymptomatic or have only symptoms and no comorbidities.

Pneumonia develops in about one in five people infected with coronavirus infection. The disease is most common in people with weak immune systems, those over 65 years of age, and those with diabetes.

“COVID-19-associated pneumonia” is interpreted as an interstitial lesion of the lung parenchyma of SARS-CoV-2. Traditionally, bacterial pneumonia is early fibrin exudation into the alveoli followed by a granulocyte infiltrate. Coronavirus infection results in interstitial pneumonitis with persistent presence of fibrin masses in the alveoli, often with microthrombosis [5,7,10]. In the work of L. Hariri et al. analytical comparison of changes in influenza and COVID-19. It is noted that both cases are characterized by the development of acute interstitial pneumonia, with thrombosis in COVID-19. On the other hand, the histopathology of COVID-19 reveals parallels with the previously described and related coronavirus diseases SARS and MERS [8,9].

When the virus enters the body by airborne droplets, the infection spreads through the blood. At this time, the infected person feels the usual cold manifestations. The initial stage is 2-14 days.

Then the virus affects the epithelium of the respiratory tract, causing inflammation. If pneumonia is not diagnosed at this stage, there is a high risk of complications such as acute respiratory distress syndrome, which requires artificial ventilation (AVP).

SARS-CoV-2-infected patients may not experience specific clinical manifestations for some time until critical condition, which depends primarily on the extent of the lesion and concomitant pathology. The replication phase of the virus is maximal before the onset of symptoms. In the pulmonary phase, there is no longer any live virus in the person, but there are circulating virus particles (viral debris). The immune response to these particles kills the patient. The live virus is not fatal.

A persistently high body temperature greater than  $38^{\circ}$ , rarely less than  $<36^{\circ}$ , is characteristic. It does not change for 5 days or more. Antipyretics help for a short time.

Dyspnea, present even at rest, is felt more on days 6-9 of the disease and is manifested by the development of hypoxia and decreased oxygenation. Breathing becomes easier if you take a position lying on the stomach. In this case, the lower parts of the lungs begin to work.

Dyspnea is accompanied by an excruciating attack-like dry cough. Sputum, as a rule, there is no. If it appears, then in a small amount.

In the lungs on both sides are heard crepitating and moist fine bubbly rales.

At an early stage of the disease, there is a decrease in the level of leukocytes and lymphocytes in the peripheral blood and an increase in ESR and C-reactive protein (CRP). In patients, all biochemical indicators are violated. A severe course of pneumonia may develop in 5% of patients and require immediate hospitalization.

If the course of pneumonia is severe, there may be:

- Acute respiratory failure;
- Increased risk of thrombosis;
- Cardiac complications;
- Organ failure.

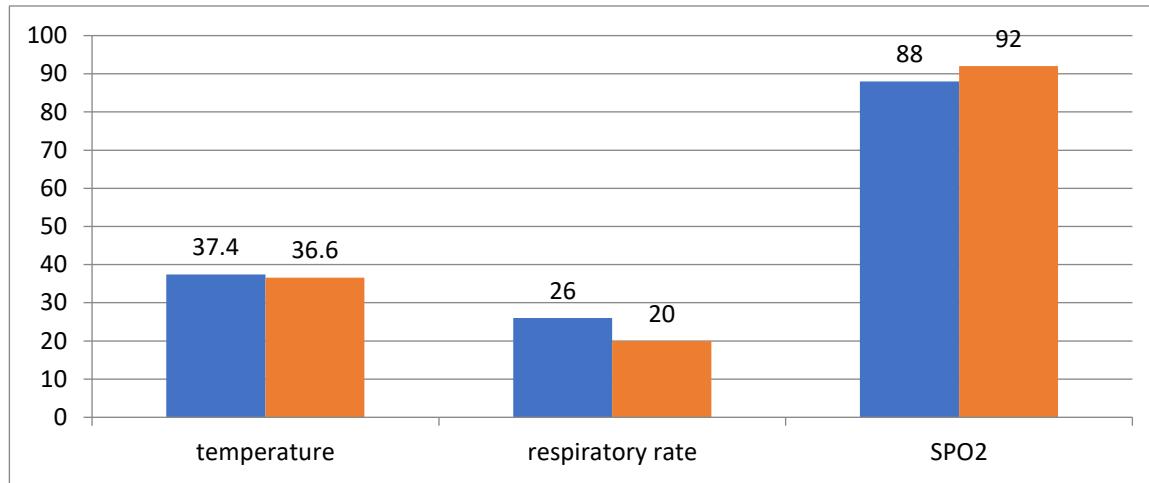
The recovery process in patients requires long rehabilitation.

**Objective of the study.** To study the peculiarities of the clinical course of COVID-19 pneumonia in a patient.

Here is a case history of patient D. with coronavirus pneumonia. A 76-year-old patient was admitted with complaints of shortness of breath at the slightest movements, weakness, and cough. The disease began 4 days ago with chills, joint pain, and sore throat. I tested positive for COVID-19. The doctor from the polyclinic prescribed azimac, paracetamol, and arbidol. The condition did not improve, shortness of breath, and cough increased and the patient was hospitalized in Zangiata Hospital No. 2. In the anamnesis she had IBS, type 2 diabetes mellitus. COVID-19 test for positive.

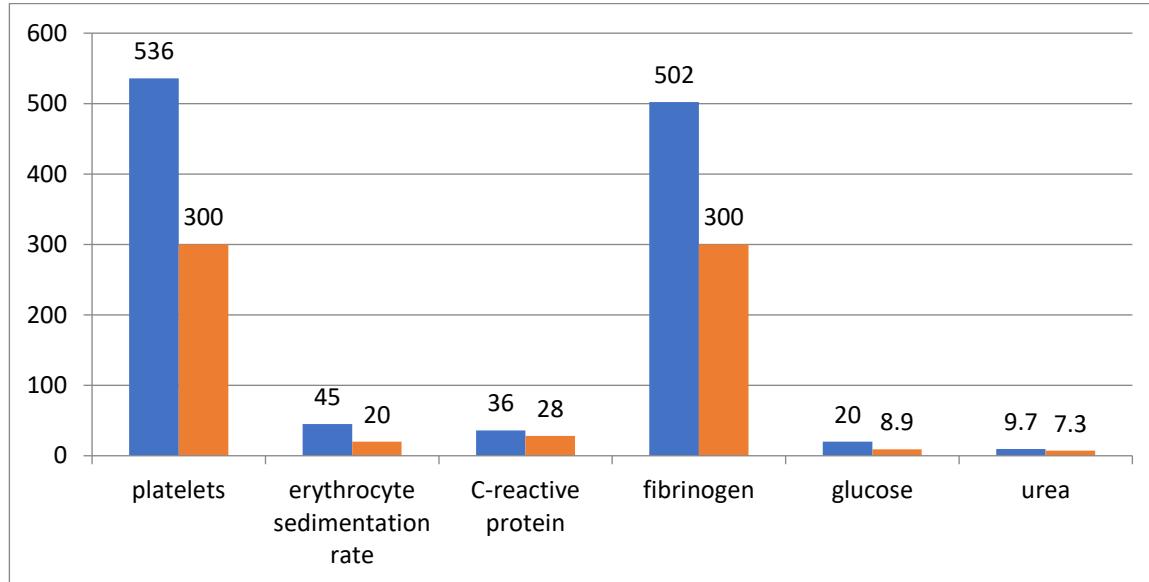
On admission, activity is limited due to dyspnea. Auscultated in the lungs: dry and moist rales. Heart tones are muffled. ECG shows chronic coronary insufficiency. The liver and spleen are not palpated.

Figures 1 and 2 show the clinical and laboratory parameters of patient D. on admission and 3 months after discharge.



*Figure 1: Clinical symptoms on admission and after 3 months*

On admission, the patient had elevated body temperature, dyspnea, and decreased oxygenation. After 3 months the indices normalized.



*Figure 2: Laboratory values on admission and after 3 months.*

Laboratory parameters are characteristic of an acute inflammatory process with symptoms of hypercoagulability and hyperglycemia. After 3 months there was improvement, but no normalization.

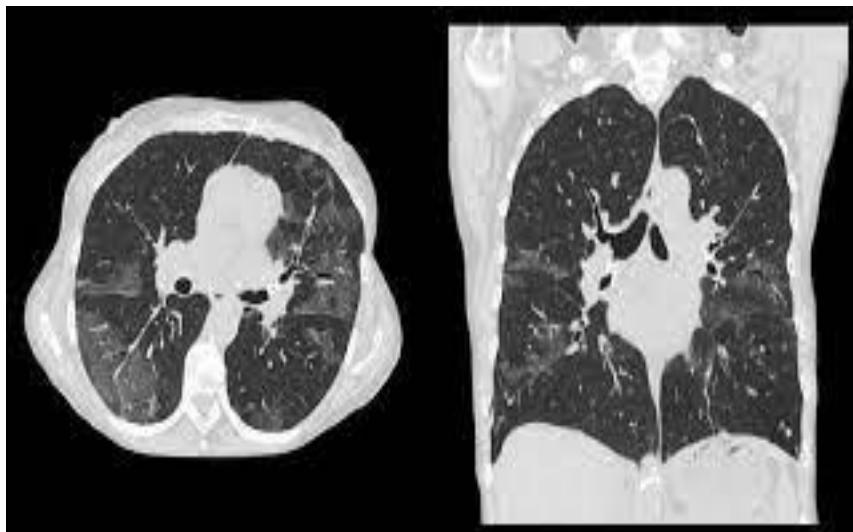


Figure 3: MRI image of the lungs.

According to MRI data on the first lung image on admission: bilateral polisegmental pneumonia with predominantly basal lesions of 2-3 degrees of severity.

2 years later on the second lung MRI image, the patient had bilateral segmental interstitial pneumonia.

In the lungs according to MRI data, there is bilateral interstitial pneumonia, which has not completely resolved even after 2 years.

Therapy: constant oxygen therapy, antiviral drugs intravenously, antibiotics intravenously, anticoagulants intravenously, glucocorticosteroids intravenously, glucocorticosteroids intravenously, bronchodilators, insulin simple and prolonged intravenously under blood glucose control and endocrinologist consultation.

After the therapy, the condition improved, the test for COVID-19 became negative, oxygenation increased, dyspnea decreased, and the patient was transferred for further rehabilitation to the Republican Medical Center of Therapy in 17 days.

Diagnosis: COVID-19 severe course. Bilateral polisegmental basal pneumonia of 2-3 degrees of severity Viral etiology (IO7.1, ICD-10). Diabetes mellitus type 2 severe course.

Thus COVID-19 pneumonia has interstitial character associated with vascular lesions and accompanied by hypercoagulation (increased platelets and fibrinogen). Interstitial lung lesion, ESR, CRP, and MRI data persist for a long time (from 3 months to 2 years), which suggests the need for long-term follow-up and appropriate correction.

## List of references used:

1. Opening remarks by the WHO Director-General at the media briefing on COVID-19 (11.03.2020). <https://www.who.int/dg/speeches/detail/who-director-general-s-opening-remarks-at-the-media-briefing-on-covid-19>, March 11, 2020 (as of March 22, 2020).
2. COVID-19 Pathologic Anatomy. ATLAS. Edited by O.V. Ayratyants, 2020.116 p.
3. Baimukhamedov C, Mirakhmedova K, Dossybayeva G. Long COVID: the time has come for globally acceptable definitions. *Rheumatol Int*. 2023 Nov;43(11):2155-2156. doi: 10.1007/s00296-023-05414-2. Epub 2023 Aug 5. PMID: 37543536.



4. Baimukhamedov C, Togizbayev G, Mirakhmedova K, Mamasaidov A, Shukurova S. Increasing autoimmune rheumatic diseases as a consequence of the COVID-19 pandemic: A hypothesis or fact? *Int J Rheum Dis.* 2024 Jan;27(1):e15011. doi: 10.1111/1756-185X.15011. Epub 2023 Dec 22. PMID: 38140787.
5. Bradley BT, Maioli H, Johnston R, et al. Histopathology and ultrastructural findings of fatal COVID-19 infections in Washington State: a case series. *Lancet.* 2020 Aug 1;396(10247):320-332. doi: 10.1016/S0140-6736(20)31305-2. Epub 2020 Jul 16. Erratum in: *Lancet.* 2020 Aug 1;396(10247):312. PMID: 32682491; PMCID: PMC7365650.
6. Coronavirus Update (Live): 629,450 cases and 28,963 deaths from the COVID-19 outbreak – Worldometer nd <https://www.worldometers.info/coronavirus/> (as of 28 March 2020).
7. Hariri LP, North CM, Shih AR, et al. Lung Histopathology in COVID-19 as Compared to SARS and H1N1 Influenza: A Systematic Review. *Chest.* 2020 Oct 7: S0012-3692(20)34868-6. doi: 10.1016/j.chest.2020.09.259. Epub ahead of print. PMID: 33038391; PMCID: PMC7538870.
8. Tabary M, Khanmohammadi S, Araghi F, Dadkhahfar S, Tavangar SM. Pathologic features of COVID-19: A concise review. *Pathol Res Pract.* 2020 Sep;216(9):153097. doi: 10.1016/j.prp.2020.153097. Epub 2020 Jul 4. PMID: 32825963; PMCID: PMC7334952.
9. Abdullaev U. S., Sayfullaevich A. U. EVALUATION OF LABORATORY-IMMUNOLOGIC METHODS OF CARDIOVASCULAR PATHOLOGY IN PSORIATIC ARTHRITIS PATIENTS //World Bulletin of Public Health. – 2023. – T. 25. – C. 24-28.
10. Abdullaev, U. S., Mirakhmedova, H. T., & Tursunova, M. U. (2021). *Research of clinical and biochemical changes of lipid metabolism for assessment of cardiovascular risk in patients with psoriatic arthritis* (Doctoral dissertation, Turkey).