THE PROBLEM OF MANAGING PATIENTS WITH COMMUNITY-ACQUIRED PNEUMONIA COMBINED WITH COVID-19 IN COMORBID CONDITIONS (LITERATURE REVIEW)

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ABSTRACT

Background. The unprecedented in the history of mankind problem of COronaVIrus Disease 2019 (COVID-19) caused by Severe Acute Respiratory Syndrome CoronaVirus 2 (SARS-CoV-2), having raised a huge number of fundamental questions about the pathogenesis of pneumonia.

Aim. To analyse the features of community-acquired pneumonia in COVID-19, markers of inflammation, the impact of comorbidities, and the implications for improving diagnosis and treatment in patients with comorbidities according to the professional literature.

Materials and Methods. We analyzed the literature on community-acquired pneumonia associated with COVID-19 in comorbid conditions. The search for scientific information was carried out using the scientific databases Scopus, Pub Med, Web of Science, Google Scholar.

Results. The analysis of the literature showed that the diagnosis of community-acquired pneumonia in COVID-19 requires the use of modern polymerase chain reaction platforms to verify the SARS-CoV-2 virus, atypical bacterial pathogens, fungal flora and determine drug resistance. SARS-CoV-2 pneumonia is characterized by the development of radiological patterns that can only be detected by Computed Tomography (CT) of the chest. Digital software processing of CT images allows to determine the dynamics and stage of development of pneumonia in COVID-19, to assess the effectiveness of treatment and the presence of residual changes. Community-acquired pneumonia provokes the development of hypercoagulability, but the likelihood of developing thrombosis in pneumonia with COVID-19 is much higher, which requires additional research and medical correction.

Conclusions. The role of the outpatient stage of medical care is important in increasing patient adherence to timely diagnosis, treatment and prevention of chronic diseases, which can contribute to the benign course of community-acquired pneumonia in COVID-19 and reduces the risk of death.

Keywords: *inflammatory markers, thrombosis, comorbidities, coronavirus disease.*

Introduction

The unprecedented in the history of mankind problem of COronaVIrus Disease 2019 (COVID-19) caused by Severe Acute Respiratory Syndrome CoronaVirus 2 (SARS-CoV-2), having raised a huge number of fundamental questions about the pathogenesis of pneumonia. The interaction of the virus with the lung microbiome and the human immune system, heterogeneity and unpredictable severity of the course, remains the main topic of our time [1].

Due to the atypical clinical and radiological picture of pneumonia and the lack of effect of available etiotropic therapy, experts periodically ask the question: is the process occurring in the lungs of a patient with COVID-19 really pneumonia? Wouldn't it be more appropriate, for example, to use the term "pneumonitis" to emphasis the immunological features of this process? Another argument in favor of interpreting the pathological process in the lungs of patients with coronavirus infection as community-acquired pneumonia is the current understanding of the pathogenesis of COVID-19. Another important aspect of the problem of COVID-19-associated pneumonia, which

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pulmonologists have recently been facing, is the likely short- and long-term consequences in the form of residual fibrosis and, in some cases, progressive pulmonary fibrosis. Experts are concerned about the presence of common features in the pathogenesis and pathomorphology of COVID-19-associated pneumonia and such a severe and rapidly developing lung disease as Idiopathic Pulmonary Fibrosis (IPF) [2].

COVID-19 with comorbidities leads to a vicious circle, huge morbidity and higher mortality. The impact of SARS-CoV-2 in comorbidities, such as diabetes (lung inflammation and increased ACE-2 expression), cardiovascular disease (impaired heart and immune system function) and Chronic Obstructive Pulmonary Disease (COPD), is damaging to the lungs, heart, kidneys and liver [3].

Although the negative role of comorbidities on COVID-19 morbidity and mortality has been scientifically proven, the impact of comorbidities on COVID-19-associated pneumonia remains unclear. According to Chaban I.V. & Marushchak M.I., the highest frequency of concomitant cardiac pathology in patients with community-acquired pneumonia associated with COVID-19 is essential hypertension (28.10%), coronary heart disease (20.00%) and dysmetabolic cardiomyopathy (12.38%) [4].

Aim. To analyze, according to the professional literature, the features of community-acquired pneumonia in COVID-19, markers of inflammation, the impact of comorbidities, and the implications for improving diagnosis and treatment in patients with comorbidities.

Materials and Methods

We analyzed the literature on community-acquired pneumonia associated with COVID-19 in comorbid conditions. Using Internet resources, we searched for scientific information in the Scopus, PubMed, Web of Science, and Google Scholar databases. After reviewing the abstracts of the articles and reading their full text, 47 sources were selected for analysis.

Results and Discussion

The main problem in the diagnosis of COVID-associated pneumonia is the need to distinguish between viral lung disease and the development of secondary bacterial pneumonia. Viral pneumonia can be of varying severity, but does not require antibiotic therapy. At the same time, the addition of bacterial flora in the setting of viral lung disease requires immediate administration of antibacterial drugs [5].

The dominant microflora of the respiratory tract in patients with COVID-19 and pneumonia is mycotic infection, among which fungi of the genus Candida prevailed. The isolation of moldy fungi Aspergillus spp. and such highly virulent bacterial pathogens as P. aereginosa, E. coli and E. faecium only in patients over 60 years of age, corresponds to the severe and most severe degree of disease in these individuals, requiring invasive respiratory support. The majority of microorganisms isolated from sputum of patients with COVID-19 were found to be resistant in vitro to most groups of antibiotics and antimycotics. The use of antibacterial and antifungal agents should be based on local microbiological monitoring data with the determination of resistance [6].

Melnyk V.P. et al. identified the features of infection in the focus of infection, clinical course of the disease, the scope and frequency of examination of patients, communication with a family doctor and treatment of patients with pneumonia caused by the SARS-CoV-2 virus. Based on the study results, the authors conclude that routine prescription of chest CT scans for patients with suspected SARS-CoV-2 pneumonia, as well as for patients with confirmed pneumonia of this etiology by polymerase chain reaction, antibacterial drugs (especially levofloxacin moxifloxacin, meropenem, linezolid, amikacin) without a confirmed need is not only unnecessary, but even dangerous due to the potential increase in resistance to them (they are the main ones in the treatment of resistant tuberculosis) [7].

Previously, radiography was sufficient to diagnose community-acquired pneumonia. Pneumonia resulting from SARS-CoV-2 infection is characterized by the development of certain radiological patterns, such as frosted glass opacity and others, which can only be detected by Computed Tomography (CT) of the chest. Digital software processing of CT images makes it possible to determine the dynamics and stage of development of pneumonia in COVID-19, to assess the effectiveness and necessity of treatment measures [8].

In severe COVID-19, Community-Acquired Pneumonia (CAP) in the 1st week of illness is manifested by dyspnea, decreased saturation, and a small area of lung damage; verification of pneumonia should be based on the presence of severe dyspnea and decreased saturation. At 2–3 weeks of illness, the area of lung involvement does not exceed 50% on CT and 18 points on the Lung Ultrasound Scoring System (LUSS). Critical course of COVID-19 with signs of pneumonia is characterized by [60–90]% lung involvement on CT and ≤15 LUSS score. The CT pattern of "frosted glass"

corresponds to the ultrasound patterns of IP, pleural thickening, and absence of A-lines; the CT pattern of consolidation corresponds to the ultrasound pattern of consolidation [9].

High-Resolution Computed Tomography (HRCT) was used to diagnose lung parenchymal area involvement of 25% to 80% (mean 58.85%) in the group of patients with severe COVID-19 who died, depending on the day of illness on the day of HRCT. The following radiological patterns were noted during the HRCT examination: bilateral, predominantly diffuse, lung parenchymal lesions with a frosted glass pattern, "crazypaving" pattern, consolidation pattern, organizing pneumonia, pneumothorax, pneumomediastinum with subcutaneous emphysema [10].

Patients with bullous-emphysematous complications (vanishing lung syndrome) account for 30% of all patients with severe community-acquired viral pneumonia (COVID-19) who required oxygen support. To diagnose complications and predict the course of severe community-acquired viral pneumonia COVID-19, it is necessary to perform CT of the chest in the dynamics with densitometric studies of the lung parenchyma [11].

Diabetes and cardiovascular disease pose a significant risk for the progression and death of coronavirus disease. One of the main pathophysiological basis of this burden is chronic heart failure, various types of myocardial dysfunction, endothelial dysfunction and metabolic disorders in the setting of diabetes mellitus. COVID-19 causes a multiorgan distress disease, creating an imbalance between the cellular and cytokine immune systems, which leads to a hyperinflammatory cytokine storm that affects systemic homeostasis. The presence of COVID-19 in patients with diabetes mellitus who already have immune disorders worsens their general condition [4; 12–14].

There is reason to believe that the mucous membrane of the distal small intestine, along with the respiratory system, is the entry gate for the pathogen in severe COVID-19. The detected morphological manifestations of severe endothelial dysfunction with endothelial damage are more indicative of endotoxin damage by the mechanism of a paraallergic reaction rather than direct viral damage [15].

Potential risk factors for death in hospitalized patients with COVID-19 include age over 57 years, presence of sternum pain and neuromuscular symptoms on admission, more than 2 comorbidities, primarily hypertension, diabetes mellitus, cerebrovascular disease, coronary heart disease, heart failure and arrhythmia, as well as a number of laboratory parameters: lymphocyte count ≤0.66; AST>50.2 U/l; total protein ≤66.1 g/l; creatinine >102.7 μmol/l; urea >7.54 mmol/l; C-Reactive Protein (C-RP) >46.8 mg/l [16].

In oxygen-dependent patients with COVID-19, the patient's age and comorbidity are associated with the outcome of the disease. If the patients' age was >66 years (Area Under the Curve (AUC)= =0.636, p=0.002) and the Charlson comorbidity index was >5, the probability of death was significant (AUC=0.652, p<0.001) [17].

In patients with critical acute course of COVID-19, dyspnea in the post-acute phase is consistently caused by both residual morphological lesions of the respiratory system and cardiovascular pathology. The presence of comorbid cardiovascular disease requires collaboration between pulmonologists and cardiologists, especially when dyspnea does not correlate with morphological lesions of the respiratory system on chest CT [18].

In the context of the COVID-19 pandemic, there was a risk that inhaled corticosteroids used by patients with COPD could adversely affect the course of coronavirus disease, in particular, prolong the period of virus replication, lead to severe complications, and increase mortality. Although research in this area is still ongoing, there is no evidence that inhaled corticosteroids increase the risk of SARS-CoV-2 infection, contribute to complications or worsen the course of COVID-19, increasing the need for hospitalization, non-invasive and artificial ventilation [19].

According to the US Centers for Disease Control and Prevention (CDC), patients with asthma and allergies are at particular risk during the current COVID-19 pandemic. The aggressive SARS-CoV-2 virus mainly affects the lungs, and most patients with asthma are at increased risk of infection and are likely to have a potentially more severe course of COVID-19 [20].

The proportion of patients with asthma among those hospitalized for community-acquired viral pneumonia (COVID-19) requiring oxygen support is 2.9%. In patients with asthma undergoing basic therapy with inhaled corticosteroids, severe community-acquired viral pneumonia (COVID-19) occurs in cases of uncontrolled asthma, comorbidities or long-term use of systemic glucocorticosteroids. The peculiarities of community-acquired pneumonia of viral etiology (COVID-19) in patients with concomitant asthma requiring oxygen support are almost complete resolution of

pathological changes in the lungs within the first 3 weeks according to CT scan of the chest on the background of adequate therapy, absence of additional emphysematous changes in the lungs and symptoms of exacerbation of asthma [21].

The analysis of the presence/absence of obesity, diabetes mellitus, coronary heart disease, and dyslipidemia as markers of cardiovascular risk in patients with COVID-19-associated pneumonia showed that both among patients with and without comorbid hypertension, the percentage of patients with obesity was lower. The study of cardiometabolic risk markers such as chronic kidney disease and smoking did not show a significant association with the severity of COVID-19-associated CAP and did not affect the comorbid course with hypertension. The comorbidity of COVID-19-associated community-acquired pneumonia and hypertension is accompanied by almost equally high and very high cardiovascular risk, regardless of the severity of pneumonia, largely due to age, heart failure, and peripheral vascular disease, while patients without concomitant hypertension are diagnosed with moderate cardiovascular risk [22].

In the presence of concomitant endocrine and cardiac pathologies against the background of a more severe clinical course of COVID-19, immunoinflammatory changes were increased due to absolute and relative leukopenia, neutrophilic granulocytopenia, neutropenia, in the presence of relative lymphocytosis and monocytosis, normal erythrocyte sedimentation rate, which confirms the inflammatory process of viral etiology, which, if left untreated, may tend to form post-COVID and/or long-term consequences of COVID-19 with multisystemic damage [23].

It is known that community-acquired pneumonia of any etiology provokes the development of hypercoagulability, but the likelihood of developing thrombosis in pneumonia with coronavirus disease (COVID-19) is much higher. The risk of thrombosis in the post-COVID period is higher in severe acute COVID-19 in the absence of anticoagulation in the post-COVID period. Instead, when anticoagulants are taken for at least two months after the first symptoms of COVID-19 appear, the risk of post-COVID thrombosis is low not only in patients with severe but also with critical COVID-19 acute course [24].

In patients with moderate severity, hemostatic disorders in the form of hypercoagulation predominated. In patients with a prolonged course of the disease for more than 21 days or in patients with severe or extremely severe hypercoagulability, hypercoagulation changed to hypocoagulation and Disseminated Intravascular Coagulation (DIC) occurred. Hemostatic parameters were directly correlated with the severity of coronavirus infection [25].

Early lung damage in COVID-19 in the deceased was determined by a distinct interstitial-alveolar edema, blood microclots and leukocyte stasis in microvessels, and less often by the presence of hyaline membranes. Bilateral polysegmental subtotal pneumonia with edema and lymphocytic infiltration of the pulmonary interstitium, inflammatory peribronchial and perivascular focal polymorphonuclear cell infiltrates was detected in 90.2% of the deceased, foci of atelectasis and dyslexia, presence of erythrocytes, hamosiderophages, macrophages, dysplastic and exfoliated alveolar epithelium in the alveoli. In 9.7% of patients, bilateral subtotal viral and bacterial fibrinous-purulent bronchopneumonia occurred. In those who died on the 22–27th day of the disease, large-focal pneumofibrosis was recorded. Pathological findings confirmed thrombotic complications in 22.0% of the deceased, which could not be diagnosed in all patients during their lifetime. The majority of those who died as a result of COVID-19 had morphological signs of chronic cardiovascular disease [26].

Increased levels of interleukin-6 (IL) observed in patients with COVID-19 correlate with the severity of the disease and may also be associated with age (especially in the 70–79 age group) and a number of comorbidities and clinical conditions, including coronary heart disease, obesity, fever, high blood pressure (systolic), and decreased saturation. Higher levels of interleukin-6 are observed with an increase in the content of C-reactive protein, residual nitrogen, erythrocyte sedimentation rate, and the number of rods of neutrophils. Taking these factors into account in combination can be useful for building a prognostic model of the course of coronavirus disease [27].

The level of IL-10 in early COVID-19 increases in accordance with the increase in IL-6, indicating its possible proinflammatory effect in the pathogenesis of the acute phase of the disease. An increase in IL-10 concentration reflects the severity of the disease, the risk of death and may be associated with a number of comorbidities, clinical conditions and changes in general laboratory parameters [28].

In patients with COVID-19 and pneumonia, the level of serum nitrotyrosine elevation depends

on the development of oxygen dependence and the outcome of the disease. The highest nitrotyrosine levels were found in patients with COVID-19 and severe pneumonia. The degree of increase in this indicator has a diagnostic value in predicting the likelihood of an unfavourable course of the disease [29].

Serum ferritin levels during hospitalisation increase with the severity of COVID-19 coronavirus disease in patients with hypertension. Serum ferritin levels are a predictor of in-hospital mortality in patients with hypertension. However, its predictive properties for severe/extremely severe course and the need for oxygen therapy are weak. A ferritin level of 438.0 ng/ml can be considered a threshold value for predicting in-hospital mortality [30].

It was found that the prognosis of severe COVID-19 depends on the time of timely treatment and hospitalisation before the period of severe respiratory failure, as 85.7% of patients who died were transferred in a serious condition from other medical institutions, and the average blood saturation on admission in patients with severe COVID-19 who died was 78.0% compared to 85.4% in patients who recovered. An important demographic factor that had a positive impact on recovery was the younger age and female gender of patients. Lupus Anticoagulant (LA) was detected in 40% of patients with severe COVID-19, which was associated with severe respiratory failure, stroke and vascular thrombosis, which in turn allows us to consider the presence of Secondary Antiphospholipid Syndrome (APS) and thrombotic complications associated with APS. Among the laboratory parameters, leukemia, elevated creatinine as a manifestation of renal failure, hypoproteinemia, and high serum glucose had the most negative impact on recovery [10].

In COVID-19, a polysyndromic course of the disease with inflammatory heart damage is possible, which can last for several months and worsen the prognosis of such patients. The mechanisms of inflammatory cardiac damage in COVID-19 include direct damage by the SARS-CoV-2 virus, hyperinflammatory cytokine release syndrome, and dysregulation of the renin-angiotensin system. All of this can be compounded by right heart overload in patients with multifocal pneumonia, thrombotic coronary artery disease, and myocardial ischemia. Cardiac inflammation is manifested not only by symptoms of typical myocarditis and pericarditis, but also by a clinic of heart failure with rapid decompensation, cardiac arrhythmia, acute coronary syndrome, or sudden death. In case of inflammatory damage to cardiomyocytes, laboratory tests show an increase in the level of C-Reactive Protein (C-RP), Brain Natriuretic Peptide (BNP) and NT-proBNP (N-Terminal pro-Brain Natriuretic Peptide), D-dimers. Transthoracic echocardiography allows to assess left ventricular dysfunction and detect fluid accumulation in the pericardium. Magnetic resonance imaging of the heart using the Lake-Louise Criteria is an informative diagnostic method in case of acute myocardial inflammation. Imaging studies are performed only when the results can affect the tactics of managing the patient according to the shortest possible protocol during the infectious period [31].

Chronic viral hepatitis in patients with COVID-19 significantly worsens the prognosis, as liver damage, coagulation disorders, and exacerbation of the inflammatory process can lead to dangerous complications, including the development of DIC and even death. The presence of seropositive HCV infection in patients with COVID-19 increases the virulence of SARS-CoV-2 and is a strong predictor of in-hospital mortality, regardless of comorbidities, laboratory changes during hospitalization, or SARS-CoV-2-induced liver damage. The mechanisms involved may be related to extrahepatic effects that enhance Angiotensin-Converting Enzyme 2/Transmembrane Serine Protease (2 ACE-2/TMPRSS) penetration of SARS-CoV-2 and are associated with baseline cytokine-mediated inflammation and endothelial dysfunction [32; 33].

Symptoms of COVID-19 can vary greatly from very severe to asymptomatic. COVID-19 presents a wide range of manifestations with negative clinical outcomes or even death in vulnerable older people with comorbidities not only hypertension, diabetes, obesity, cardiovascular disease, but also chronic kidney disease and cancer [34].

The incidence of kidney damage among patients with COVID-19 can range from 1 to 13%, and the development of acute kidney injury is a risk predictor associated with high in-hospital mortality. IL-6 cytokines can cause intrarenal inflammation and lead to the development of kidney damage. Patients who were hospitalized with elevated serum creatinine levels have a long-term increase in the number of white blood cells and lower levels of lymphocytes and platelets [35].

COVID-19 has significant negative clinical, laboratory, socioeconomic, and psychological consequences among patients with malignant tumors. This is more noticeable in those with hema-

tologic diseases. People with lung cancer have the highest mortality rate among cancers from COVID-19. COVID-19 and cancer overlap at the molecular level, which may have therapeutic implications for cancer patients. The COVID-19 pandemic has caused serious disruptions in the provision of clinical services to cancer patients around the world. Delayed referrals and diagnosis due to stressed healthcare systems and patients' reluctance to seek care during the pandemic have had a significant impact on the deterioration of detection and confirm that cancer is a risk factor for mortality from coronavirus disease [36].

It was found that patients with COVID-19 often have multiple neurological and mental disorders, such as agitation (69%), signs of corticospinal tract damage (67%), confusion (65%) and neuropsychological disorders (33%). Among the mental and psychological disorders observed after coronavirus disease, the most common are insomnia (42%), decreased concentration and attention (38%), anxiety (36%), memory disorders (34%), depression (33%), confusion (28%), and other disorders of consciousness (21%). In the group of middle-aged patients with metabolic syndrome, the incidence of depression was three times higher. Elderly patients with metabolic syndrome after COVID-19 were more likely to be diagnosed with anxiety - almost every second. These manifestations of anxiety and depression were accompanied by disorganization of bioelectrical activity in the brain [37].

COVID-19 in pregnant women is accompanied by more significant signs of intoxication and coagulation disorders with increased levels of D-dimers, but pregnancy does not pose a significant risk for the course of the disease. COVID-19 does not have a more severe course in pregnant women than in non-pregnant women in the same age groups. Pregnant women with COVID-19 who have obesity, hypertension, chronic pyelonephritis, or a combination of two or more comorbidities are at risk of a more severe course of the disease and longer hospitalization [38; 39].

According to the generalized data provided in the review published in 2022, the incidence of COVID-19 among HIV-infected people does not differ from the incidence in the general population The pandemic caused by COVID-19 has affected the provision of diagnostic, preventive and treatment services to HIV-infected people. There is an inverse proportional trend between the number of confirmed HIV cases and COVID-19 cases. The COVID-19 pandemic has reduced the number of rapid HIV tests, new HIV diagnoses, and AIDS diagnoses, which is likely to significantly increase HIV incidence in Ukraine in the near future and increase the number of new AIDS cases [40; 41].

The severity of dyspnea in the subacute period of COVID-19 depends on the extent of lung damage and the severity of the disease in the acute period. In the moderate acute course of COVID-19, the presence of dyspnea in the subacute period is mainly due to residual changes in the respiratory system (in 20.0% of patients) and mild obstructive ventilatory disorders (in 42.9% of patients). Cardiovascular disorders in these patients are detected mainly by elevated serum NT-proBNP levels (in 22.9% of patients), which may be a marker of the onset of heart failure in this cohort and require additional consultation with a cardiologist. In the severe course of the acute period of COVID-19, the presence of dyspnea in the subacute period is due to both residual morphological changes in the respiratory system according to CT of the upper respiratory tract (in 54.1% of patients) and pathology of the cardiovascular system (in 45.9% of patients) [42].

As a result of the analysis of possible ways of transformation of typical radiological changes in community-acquired pneumonia of viral etiology (COVID-19), three main pathways were identified. In 71 (64.0%) patients, according to CT scan of the chest, there was a gradual resorption of pathological changes and restoration of lung parenchyma in the dynamics. In 35 (31.2%) patients, signs of the "disappearing lung" syndrome were detected. In 5 (4.5%) patients, according to the CT scan of the chest and pathological examination, the diagnosis of Bipolar Affective Disorder (BAR) was established. Digital software processing of CT scan data in the dynamics allows to track the process of transformation of the lung parenchyma structure in patients with complicated community-acquired pneumonia of viral etiology (COVID-19) into BAR and in some cases confirm the secondary nature of the oncological process [43].

Persistent respiratory manifestations in the form of dyspnea (26.1%) and chronic cough (13.1%) are among the main symptoms of post-COVID-19 syndrome, which in 9.4% manifests as post-COVID-19 IPF. In 76% of post-COVID-19 patients, COPD manifests as a separate chronic respiratory pathology after severe COVID-19, in 16% it is the debut of SST after COVID-19, and in 8% it is a manifestation of other chronic COPD that was not diagnosed in time before the onset of

coronavirus infection. In patients with post-COVID-19 IPF, the radiologic pattern of fibrosis-like changes was noted in 64 %, and the formation of a radiologic pattern of pulmonary function associated with SST or other chronic IPF – in 36 %. It was found that the use of nintedanib for 3 months in the prolonged course of severe COVID-19 did not significantly affect the radiological pattern of pulmonary function and clinical progression post-COVID [11].

The aim of the study by Homeliuk T.M. & Marushchak M.I. was to investigate and analyze the total white blood cell count and leukogram parameters in patients with community-acquired pneumonia caused by SARS-CoV-2 virus, to establish their relationship with subjective health assessment using the SF-36 questionnaire 1 year after hospital discharge. Researchers have found that in patients with community-acquired pneumonia caused by SARS-CoV-2 virus, an increase in white blood cell count during hospitalization is associated with a decrease in physical and psychological health components 1 year after hospital discharge [44].

Patients with COVID-19 are at higher risk of developing coronary heart disease, myocarditis, pericarditis, heart rhythm disturbances, heart failure, thromboembolic complications, and hypertension both in the acute phase and in the long-term, even in the absence of a history of heart disease, low cardiovascular risk, and mild disease, and require careful medical supervision to prevent the development of life-threatening complications. Given the prevalence of cardiovascular disease and its impact on mortality, such processes in the context of the COVID-19 pandemic pose a significant threat to the global health system [45; 46].

Thus, in more than 30% of patients with signs of post-COVID syndrome, a more detailed examination reveals a significant decrease in the diffusion capacity of the lungs ((Diffusion Capacity of the Lungs for Carbon Monoxide, DLCO) and associated damage to the pulmonary interstitium. About 10% of these patients are prone to severe organic changes in the lung parenchyma, namely, lung interstitial damage and subsequent pulmonary fibrosis. To improve the quality of life and prognosis of this group of patients, early detection and verification of respiratory system pathology and timely administration of appropriate therapy are necessary [47]. Despite numerous data and recommendations from international organizations on the need for close monitoring and management of patients with comorbidities, unfortunately, only half of patients received basic therapy for their underlying diseases, mostly antihypertensive, hypoglycemic, neurotropic, and anticoagulant therapy before admission to the hospital. The role of outpatient care is important in increasing patients' adherence to timely treatment and prevention of chronic diseases, which significantly reduces the risk of death from all causes, including alleviating the course of COVID-19 [16].

Conclusions

1. The diagnosis of community-acquired pneumonia in COVID-19 requires the use of modern polymerase chain reaction platforms to verify the SARS-COV-2 virus, atypical bacterial pathogens, fungal flora, and determine drug resistance.

2. Pneumonia in SARS-CoV-2 is characterized by the development of radiological patterns that can only be determined by computed tomography of the chest. Digital software processing of computed tomography images makes it possible to determine the dynamics and stage of development of pneumonia in COVID-19, to assess the effectiveness of treatment and the presence of residual changes.

3. Community-acquired pneumonia provokes the development of hypercoagulability, but the likelihood of developing thrombosis in pneumonia with coronavirus disease (COVID-19) is much higher, which requires additional research and medical correction.

4. Community-acquired pneumonia in COVID-19 has a severe course and high mortality in vulnerable elderly people with a combination of comorbidities - hypertension, diabetes mellitus, obesity, cardiovascular disease, chronic kidney disease, chronic liver disease, malignant tumors. Only half of patients receive basic therapy for their underlying diseases before admission to the hospital.

5. Respiratory manifestations in the form of shortness of breath and chronic cough are among the main symptoms of post-COVID syndrome. In more than 30% of patients with signs of post-COVID syndrome, a more detailed examination reveals a significant decrease in lung diffusion capacity and damage to the pulmonary interstitium. About 10% of these patients are prone to severe organic changes in the lung parenchyma.

6. The important role of the outpatient stage of medical care in increasing patient adherence to timely diagnosis, treatment and prevention of chronic diseases, which can contribute to the be-

nign course of community-acquired pneumonia in COVID-19 and reduces the risk of death.

DECLARATIONS:

Disclosure Statement

The authors have no potential conflicts of interest to disclosure, including specific financial interests, relationships, and/or affiliations rele-vant to the subject matter or materials included.

Statement of Ethics

The authors have no ethical conflicts to disclosure.

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