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Pathogenesis of the Development of Acute Purulent-Destructive Lung Diseases in Patients with SARS-CoV-2

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ABSTRACT

The problem of diagnosis and treatment of acute purulent-destructive lung diseases is still relevant due to the continuing high frequency of unsatisfactory outcomes. To date, the high effectiveness of such methods as increasing the effectiveness of antibiotic therapy with the use of intravascular (into the pulmonary artery, into the bronchial artery) administration of drugs and intracavitary (transthoracic, endobronchial) ways of sanitation of purulent-destructive foci in the lungs has already been proven. The main problems that need to be solved are the identification of patients at risk of developing acute purulent-destructive lung diseases and the development of more effective approaches to their treatment. One of these risks today is ventilator-associated pneumonia against the background of SARS-CoV-2. This manuscript is devoted to a review of the literature on the pathogenesis of the development of acute purulent-destructive lung diseases in patients with SARS-CoV-2

Keywords: *acute lung abscess, lung gangrene, SARS-CoV-2*

Based on the results of clinical observations, most publications note that 2/3 of patients with SARS-CoV-2 developed acute respiratory distress syndrome at the dawn of the pandemic. It was these patients who had an increased risk of developing ventilator-associated pneumonia.

V. Beaucote et al., in their observations, noted a characteristic trend in the development of purulent-destructive lung diseases in patients with SARS-CoV-2 who were under mechanical ventilation in the intensive care unit.

Indeed, pulmonary endotheliitis, which provokes the development of microthromboembolism in the peripheral parts of the lung tissue [1], has been widely reported among critical patients with COVID-19.

Wicky P.N. and co-authors [2] noted that it is in such patients, due to the insufficiency of the concentration of

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antibiotics in the lesion, that the risk of developing purulent-destructive diseases becomes very high.

Libby L.S. and others argue that purulent-destructive lung diseases in seriously ill patients with SARS-CoV-2 can also develop as a result of the addition of superinfection against the background of pulmonary microthromboembolism and endotheliitis [3]. The prerequisites for the development of purulent-destructive lung diseases in such cases are also the development of infarction of lung tissue areas [4].

Kalenchits T.I. et al. [5] described a case of polysegmental destructive viral-bacterial pneumonia complicated by an acute lung abscess pleural empyema in a 50-year-old patient who was treated in a hospital for SARS-CoV-2. A month was diagnosed with an abscess in the lower lobe of the right lung, which subsequently spontaneously drained into the pleural cavity. The authors believe that one of the factors in the formation of a lung abscess in SARS-CoV-2 may be a violation of the blood coagulation system with the formation of microthrombi in small pulmonary vessels.

The frequency of acute purulent-destructive lung diseases in patients with SARS-CoV-2 during the pandemic was ambiguous [6].

Thus, according to several hospitals in Europe, if during the first wave (from March to June 2020), the incidence of acute purulent-destructive lung diseases was noted in the range of 35-46%, then during the period of other waves (from August 2020 to April 2021) there was an increase in the number of cases of patients with acute purulent-destructive lung diseases in the range of 52-65% [7].

Based on the above, we can state that any study regarding the features of the clinical, radiological and microbiological manifestations of purulent-destructive lung diseases in patients with SARS-CoV-2 deserves close attention.

Literature sources cite many clinical cases of the development of acute purulent-destructive lung diseases in patients with severe SARS-CoV-2 [8].

At the same time, all patients were on mechanical ventilation as a result of the development of acute respiratory distress syndrome - episodic or recurrent [9].

The presence of SARS-CoV-2 is usually confirmed by reverse transcriptase polymerase chain reaction from nasopharyngeal swabs.

According to the 2016 Clinical Guidelines of the Surgical Infection Society of America and the American Society of Thoracic Surgeons, the main clinical and laboratory signs of ventilator-associated pneumonia correspond to those of systemic inflammatory response syndrome [10]. In particular, in patients with SARS-CoV-2, ventilator-associated pneumonia usually begins with fever, hyperthermia (≥38.3°C) or hypothermia (≤35°C), leukocytosis (total leukocyte count ≥10000x10^9/L) or leukopenia (total leukocyte count ≤4500x10^9/L), increased immature neutrophil count of more than 15%, presence of purulent sputum, need to change the ventilatory support system to enhance oxygenation as new areas of lung infiltration are identified within the next 48 hours of monitoring.

According to Kalil A.C. [11], ventilator-associated pneumonia is characterised by a microbiological landscape of the respiratory system of more than 10^3 CFU/ml in the distal bronchial sample, more than 10^5 CFU/ml in the tracheal aspirate, and more than 10^4 CFU/ml in the study of bronchoalveolar lavage.

Computed tomography of the chest is used to diagnose acute purulent-destructive lung diseases in patients with ventilator-associated pneumonia against the background of SARS-CoV-2 [12].

In the northwestern district of Paris (Argenteuil, France), with a population of 101300, 161 patients with SARS-CoV-2 underwent mechanical ventilation between March 6, 2020 and April 4, 2021. Of these, 73% (119 patients) developed ventilator-associated pneumonia. 68 patients (57%) of 119 patients with ventilator-associated pneumonia underwent computed tomography of the chest cavity. The presence of acute purulent-destructive lung diseases was revealed, which amounted to 14% of the total number of patients with ventilator-associated pneumonia [13].

It is noted that patients with acute purulent-destructive lung diseases against the background of ventilator-associated pneumonia were older. The consistent assessment of organ failure ranged from 3 to 5 organs of the system (p=0.05). The simplified assessment of acute functional disorders ranged from 26 to 35 points (p=0.045).

In the development of acute purulent-destructive lung diseases in patients with ventilator-associated pneumonia against the background of SARS-CoV-2, an important role is given to the presence of concomitant diseases. For example, obesity with a body mass index of more than 30 kg/m^2 was found in 53% of cases, chronic respiratory diseases in 24% of cases, diabetes mellitus in 18% of cases, immunosuppression in 12% of cases, and chronic kidney diseases in 6% of cases [14].

Patients with acute purulent-destructive lung diseases against the background of SARS-CoV-2 are characterised by higher fibrinogen values and relatively low values of d-dimers and platelet counts [15].

The evidence of the severity of the condition of this category of patients should be attributed to the results of their treatment. In 88% of cases, patients with acute purulent-destructive lung diseases against the background of ventilator-associated pneumonia after SARS-CoV-2 require support with vasopressins, and in 38% of cases, renal replacement therapy is required [16].

Statistics show that in 35% of cases, patients with acute purulent-destructive lung diseases against the background of ventilator-associated pneumonia after SARS-CoV-2 have thrombotic events during their stay in the intensive care unit. At the same time, the duration of stay in the intensive care unit ranges from 19 to 47 bed days, and the mortality rate is up to 65% [17].

According to several clinical observations, acute purulent-destructive lung diseases in patients with ventilator-associated pneumonia against the background of SARS-CoV-2 are diagnosed within 11 to 27 days after tracheal intubation [18].

Clinical signs of acute purulent-destructive lung diseases before computed tomography are detected in an average of 71% of patients. When diagnosing acute purulent-destructive lung diseases in patients with ventilator-associated pneumonia against the background of SARS-CoV-2, the average number of leukocytes and procalcitonin is 25 g/l (20-30 g/l) and 12 μg/l (7-22 μg/l), respectively. In most cases (71%), acute purulent-destructive lung diseases affect the right lungs, mainly the lower lobes (76%). The zone of lung damage is characterised by a peripheral part, often (94%) located subpleurally [20].

The problem of treating acute purulent-destructive lung diseases against the background of SARS-CoV-2 requires solving several problems, including the choice of antibiotic therapy, taking into account the clinical and pathological features of the purulent-inflammatory process. It is necessary to ensure the availability of the zone of purulent-necrotic destruction in the lung tissue for drugs and the evacuation of purulent contents from the foci of destruction in the lungs and pleural cavity [21].

The choice of methods of conservative and surgical treatment of acute purulent-destructive lung diseases against the background of SARS-CoV-2 should depend on the course of the process itself since effective methods of treating respiratory, multiple organ failure, and septic shock are required [22].

Taking into account the fact that the prevention of the development of acute purulent-destructive lung diseases in patients with SARS-CoV-2 depends on the effectiveness of treatment for ventilator-associated pneumonia, studies have been carried out to develop more effective methods for its treatment [23]. In the course of the study, clarifications were made on the complications of ventilator-associated pneumonia in patients with SARS-CoV-2 [24]. In the initial phase of acute purulent-destructive lung diseases, focal infectious purulent-necrotic destruction develops [25]. It is manifested by multiple purulent-necrotic foci of bacterial or autolytic proteolysis without a clear demarcation from viable lung tissue, which already complicates the course of ventilator-associated pneumonia against the background of SARS-CoV-2 [26].

With the prevalence of local clinical symptoms of the disease, according to the type of local infection, it is considered that such a course of ventilator-associated pneumonia is mild [27]. The moderate course of ventilator-associated pneumonia is characterised by a combination of both local and general symptoms of an inflammatory reaction. However, ventilator-associated pneumonia against the background of SARS-CoV-2 is characterised by a severe and extremely severe course with all the signs of a generalised infectious process [28]. At the same time, the severe course of the disease is manifested by correctable or non-correctable respiratory failure, shock pneumonia, with a predominance of hemodynamic and hemorheological disorders, early shock, functional sequence, or simultaneous multiple organ failure [29]. An extremely severe form of ventilator-associated pneumonia in the context of SARS-CoV-2 is characterised by refractory hypotension, also known as tardive shock, and multiple organ failure with organic changes in organs and tissues [30]. This course of the pathological process is similar to the course of abscess pneumonia, which indicates the similarity of the morphofunctional basis of the pathological process in the lung [31].

CONCLUSION

Taking into account that, according to the literature, the mortality rate in acute purulent-destructive lung diseases ranges from 11.7% to 28.5%, we can talk about a certain effectiveness of the developed methods of treatment. However, it is impossible to make such a statement regarding patients in whom acute purulent-destructive lung diseases have developed against the background of SARS-CoV-2. Mortality among this category of patients is still at a high level6, sometimes reaching up to 46.2%. With the development of generalisation of the purulent-inflammatory process, mortality can reach 80% or more. Only the mechanism of the development of acute purulent-destructive lung diseases against the background of SARS-CoV-2 can be to blame. The similarity of the pathogenetic mechanisms

of the formation and course of acute purulent-destructive lung diseases should allow for achieving positive results of treatment. However, unfortunately, patients who have had SARS-CoV-2 also have other pathomorphological changes in the lung tissue and in the hemostasis system, which reduce the effectiveness of the known methods of treating acute purulent-destructive lung diseases available in the arsenal of clinicians. In this aspect, it seems to us that it may be necessary, along with a comprehensive assessment of the course of acute purulent-destructive lung diseases, to assess the state of the endothelial system of the lungs, which, according to several researchers, plays an important role in the rapid formation of foci of lung destruction.

At the same time, further study of the pathogenesis of the development of acute purulent-destructive lung diseases will improve treatment methods and improve disease outcomes.

Conflict of Interest – None

Ethical aspect – the article is reviewed, and the information presented has a cited reference to primary sources.

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