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Opinion

COVID-19 Pneumonia: Finding Clinical Solutions

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The SARS-CoV-2 pandemic, despite the severity and tragedy of its manifestations, plays, oddly enough, a positive role for modern medicine. Some aspects of medical care have long required radical revision, and with the activation of the coronavirus, the need for new approaches and solutions has become more obvious, although old treatment stereotypes continue to determine the search strategy. Therefore, it is necessary to note the new facts and circumstances that have accumulated during the current pandemic, as well as to assess them impartially and properly in order to make the necessary adjustments.

The main and characteristic pathology in coronavirus infection is lung damage [1]. Although some experts consider this form of acute inflammation to be a new disease with clinical uncertainty [2], the vast majority of researchers and clinicians define it as COVID-19 pneumonia. This term continues the tradition of recent years to classify lung inflammation with an emphasis on the probable etiology of the process [3]. In this regard, it should be noted that the experience of such a gradation did not bring practical benefits, since we are talking about one nosology "acute pneumonia" (AP), and the reliability of the etiology of the process from such a definition has not improved [4]. In addition, the unity and generality of bacterial forms of AR and COVID-19 pneumonia is further confirmed by the complexity of their differential diagnosis [5].

Although the definition of the nosological affiliation of the disease is one of the most important issues of discussion, in this situation it is only the tip of the iceberg that sinks the ships of researchers and testers.

Thus, the development of the pandemic showed the unpreparedness of the official medical service for such a turn of events. Experts ' concern about the growing share of viral pneumonia in recent years continues to be expressed in the form of a statement of this fact, rather than in new methods of treatment [6]. The experience of at least two major coronavirus epidemics over the past couple of decades (SARS, MERS) has not allowed us to develop effective ways to help in the event of its recurrence. The questionable effectiveness of medical care, the problematic and hasty search for rational methods of treatment without reliable results over the past year and a half clearly demonstrate the confusion and indecision of specialists at all levels.

The familiar traditions and stereotypes of recent decades, which emphasize the exceptional importance of the causative agent of the disease, continue to determine the search for solutions in the face of new circumstances. During the current pandemic, clinical trials of various antiviral drugs are constantly being conducted, and ongoing research is constantly being duplicated in different parts of the world. However, the efforts made have not yielded any encouraging results, as eloquently evidenced by the final report of the World Health Organization, which summarizes the materials of 405 hospitals from 30 countries [7].

For many recent years, failures in the treatment of patients with AP have been attributed to a mismatch between the pathogens of the disease and current antibiotic therapy. This reason was considered as the main one in the case of complicated course of the process and fatal outcomes. This point of view, which had no con-

vincing objective arguments, continues to dominate at the present time, although current events completely break it. For example, it is no secret that under the conditions of the participation of a single coronavirus, the spectrum of infection manifestations is infinitely wide and repeats the variants of the course of bacterial forms of AP. The death rate, which peaks in intensive care units, for COVID-19 pneumonia does not differ significantly from the percentage of deaths from so-called community-acquired pneumonia in recent years [8-10]. At the same time, it is known that patients with viral lesions do not receive therapy, which is still considered life-saving.

Over many years of active antibacterial therapy, the belief that all the troubles in patients with AP are caused by the pathogen has paradoxically grown and strengthened, despite many contradictory facts. Today, this opinion is so ingrained in the professional worldview that the fundamental materials of medical knowledge that are most directly related to this disease have actually ceased to be taken into account when interpreting it. Currently, all research and the search for therapeutic solutions are carried out taking into account the etiology of the process. Many researchers, given the lack of effective etiotropic drugs and the possibility of direct suppression of the pathogen in SARS-CoV-2, are trying, if not to destroy it, then at least to reduce the viral activity. Such efforts are aimed at possible ways to protect cells from the penetration of the virus into them, or at eliminating various factors and shifts in the body that are considered as a result of viral aggression.

Since the main object of study in this search is the coronavirus, such studies are conducted at the microscopic level, focusing on individual cell types, subcellular structures and chemical compounds. However, it is not only the virtual nature of the results of such studies that initially creates difficulties in their application in patients with COVID-19 pneumonia. The goals pursued by such studies do not always imply obtaining ways to actually help patients with SARS-CoV-2. For example, discussing options for protecting the body's cells from being invaded by the coronavirus may actually provide additional options for preventing the disease in the event of infection [11-14]. Such methods of protecting the body, if they are found and proposed, may enhance the effect of the current vaccination, but will clearly provide belated help to those who are already ill.

There are proposals for the treatment of patients with COVID-19 pneumonia of a more specific nature. For example, neutralization and elimination of cytokines or correction of disorders in the renin-angiotensin-aldosterone system [RAAS] are considered as possible prospects for successful treatment of such patients [15-18]. Promising results of such efforts have not yet been presented, and the likelihood of achievements in this direction is very doubtful for the following reasons.

First, the release of cytokines and changes in the RAAS are not specific features of the pathology discussed here. These deviations can be characteristic not only for AP, but also for many other diseases that are not accompanied by inflammation [19]. Such therapy is comparable to reducing the patient's high temperature, which does not stop the development of the disease.

Secondly, it is necessary to recall that any deviations in the body are initially protective and adaptive in nature. It is the initial shifts in homeostasis that allow the body to adapt to new unexpected conditions. Only if the active reasons for the violation of such parameters are preserved, there is a need to correct them. In this regard, a more important and logical search would be to return to the old concept of "norm in pathology" and clarify the limits of permissible deviations.

Finally, we must have a clear idea of what place in the chain of successive mechanisms of the development of the disease occupies a particular reaction and what are the causes of its occurrence. Taking into account all the above-mentioned features of the development of the inflammatory process in the lung, the proposed options for medical care for patients with COVID-19 pneumonia resemble the reasoning of one of the heroes of O'Henry's novels. The young man asks the interlocutor: "Where is the wind from?" and, without waiting for an answer, answers himself: "Because the trees are swaying?"

At the same time, during the year and a half of the pandemic, there were no proposals to provide truly adequate and effective therapy for patients with viral pneumonia. According to the results of the conducted studies and tests, only the use of dexamethasone, which is also not a specific remedy and is usually prescribed in desperate situations, gives some benefit to already ill patients [20,21]. In this regard, it is no coincidence that out of the huge amount of work on this problem, on which about $ 2 billion was spent in a short period of time, only 8% of completed or discontinued studies are sent for reporting publication [22].

Thus, one of the leading areas of medical science in the study of AP remains the search for effective correction of disorders in the body, which are considered as a result of the aggression of the pathogen. The choice of the purpose of such studies is initially based on erroneous ideas about the causes, sequence and significance of the studied parameters, so the expected results can not radically improve the treatment of this group of patients. The basis for this conclusion is the following arguments and facts.

First, during the current pandemic, when it comes to infecting large populations with a single pathogen, 80% of those infected carry this event on their own, without resorting to medical care, and 20% of such contacts are not accompanied by the disease at all [23,24]. The presented data contradict the point of view that the properties of the pathogen determine the development and features of the course of AP, since in most cases of infection there are no questions and problems that invariably arise with the development of inflammation in the lung tissue, right?

Secondly, for many years there has been a clear contradiction between the theory and practice of AP. In the process of diagnosis and monitoring, the theoretical dominance of the pathogen imperceptibly loses its leadership, giving way to more important and informative tests. For example, in the process of treating patients with AP, the attending physicians are more interested in the data of X-ray examinations of the lungs and the parameters reflecting the functions of the vital systems of the body. Microbiological studies and other cell-molecular tests are relegated to the background and are not subject to such frequent re-checks, do you agree?

If we take into account the fact that the lungs are responsible not only for respiratory function, but also are an important sector of the vascular system that is involved in the regulation of blood flow, then the following diagram can give a clear idea of the influence of the focus of inflammation on the overall blood circulation (see figure 1). This representation is more mechanical in nature, since the totality of the processes occurring in the small circle of blood circulation cannot be depicted in an ordinary drawing. However, even such a simple image allows us to note that, unlike inflammation of other internal organs, AP creates an obstacle to the general blood flow. This circumstance underlies the severity of the manifestations of the disease, which increases in parallel with the increase in the volume of the lesion.

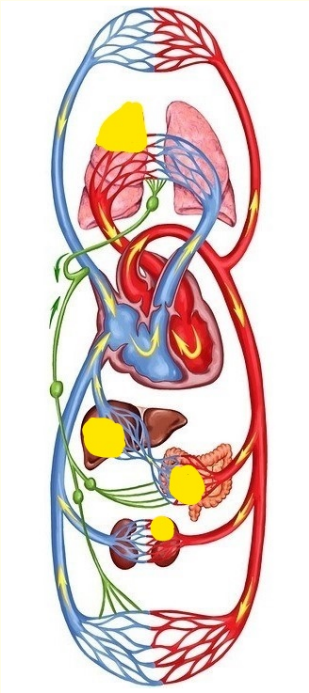

Figure 1: Schematic representation of the human circulatory system. Comparative value of foci of acute inflammation (yellow fields) for different departments and volumes of blood flow, depending on the possible localization.

In AP, along with the involvement of a part of the pulmonary vascular bed in the area of inflammation, there is a reflex spasm of intact vessels of the small circulatory circle, which, having a protective and adaptive nature, affects the clinical picture of the disease and in certain situations requires immediate correction [25]. This mechanism of rearrangement of blood circulation in the case of irritation of the baroreceptors of the pulmonary vessels was first described by Schwiegk [26] and is now known as the unloading reflex. In addition to the reflex response, humoral factors and, in particular, RAAS also take part in the adaptation process.

Among the large number of clinical studies devoted to SARS-CoV-2, there are some publications whose materials confirm the mechanisms of AP described above. For example, the use of tomography in patients with COVID-19 pneumonia to determine the

volume of blood in the lungs allowed us to establish a decrease in this indicator in small vessels with a diameter of no more than 2 mm [27]. The authors suggest that this fact is caused by spasm and thrombosis of the vessels of the small circle as a result of their viral damage. Generalized thrombosis of this localization can hardly be compatible with life, and the spasm is quite consistent with the already known and studied reactions.

Given the shock nature of peripheral disorders, the cause of the shock itself is considered to be viral sepsis, and not the shockogenic role of the focus of inflammation in the lungs. This interpretation of the mechanisms of AP is based on the ideology of complete dependence of the disease on its pathogen. The absence of microbiological evidence of sepsis does not prevent claims of septic shock in AP. These unconfirmed assumptions prevailed in bacterial forms of the disease and currently continue to be declared a viral variant of shock [28]. Shock in AP is diagnosed according to clinical and laboratory indicators, while theoretically its nature and mechanisms of development remain focused on the etiology of the process.

Modern principles of studying the problem of AP in general and coronavirus pneumonia in particular involuntarily and logically raise a number of questions. Why do cytokine release or changes in the RAAS correlate with the etiological factor of AP, although they are not a specific manifestation of this disease? At what stage of the disease do such disorders occur and what is their cause? What is the place of each indicator and its deviations in the general chain of pathogenesis? What is the limit of such shifts when they do not require special assistance? Why does the severity of AP not depend on its etiology, but shows a connection with the speed of development of the process and the volume of the lesion?

The list of questions, as well as various tests and parameters of the disease, can be continued, but the results of the search for solutions will remain at the same level, unless a radical revision of the AP doctrine is carried out. Taking into account the objective facts and fundamental materials of medical science, bringing the ideology of the disease in line with biological rules and laws will create a systematic approach to finding solutions, in which each mechanism of the process will take its place in this puzzle, helping to present the overall picture of the disease and the chain of its pathogenesis.

The need for a new AP doctrine is dictated by the discrepancy between the actual data and their interpretation. Theoretical misconceptions were formed under the impression of the initial use of antibiotic therapy and continue to deepen every year. It is time to look at the essence of the problem from the standpoint of the fundamental materials of medical science and successfully implement the possibilities of pathogenetically based medical care. The first steps in this direction have already been taken and have received excellent confirmation in clinical conditions [29,30]. In the current situation, there is an opportunity to develop success in this area of medical care, which will save many lives and bring a huge economic effect.

Bibliography

1. Caramaschi S., *et al*. "Histopathological findings and clinicopathologic correlation in COVID-19: a systematic review". *Modern Pathology* (2021).

2. V E Schaye., *et al*. "Collaborating Across Private, Public, Community, and Federal Hospital Systems: Lessons Learned from the Covid-19 Pandemic Response in NYC". *NEJM Catalyst Innovations in Care Delivery* 1.6 (2020).

3. Pneumonia.

4. J P Metlay., *et al*. "Diagnosis and Treatment of Adults with Community-acquired Pneumonia. An Official Clinical Practice Guideline of the American Thoracic Society and Infectious Diseases Society of America". *American Journal of Respiratory and Critical Care Medicine* 200.7 (2019): e45-e67.

5. Kamat IS., *et al*. "Procalcitonin to Distinguish Viral From Bacterial Pneumonia: A Systematic Review and Meta-analysis". *Clinical Infectious Diseases* 70.3 (2020): 538-542.

6. Ruuskanen O., *et al*. "Viral pneumonia". *Lancet* 377 (9773): 1264-1275.

7. WHO Solidarity Trial Consortium. "Repurposed Antiviral Drugs for Covid-19 — Interim WHO Solidarity Trial Results". *The New England Journal of Medicine* 384 (2021): 497-511.

8. A Ceccato and A Torres. "Sepsis and community-acquired pneumonia". *Annals of Research Hospitals* 2 (2018): 7.

9. Grasselli G., *et al*. "Baseline characteristics and outcomes of 1591 patients infected with SARS-CoV-2 admitted to ICUs of the Lombardy region, Italy". *JAMA* (2020).

10. S Richardson., *et al*. "Presenting Characteristics, Comorbidities, and Outcomes Among 5700 Patients Hospitalized With COVID-19 in the New York City Area". *JAMA* (2020).

11. Attaway A H., *et al*. "Severe covid-19 pneumonia: pathogenesis and clinical management". *BMJ* 372 (2021): n436.

12. Zhang J., *et al*. "The interaction of RAAS inhibitors with COVID-19: Current progress, perspective and future". *Life Science* 257 (2020): 118142.

13. EJ Rubin., *et al*. "Interleukin-6 Receptor Inhibition in Covid-19 — Cooling the Inflammatory Soup". *The New England Journal of Medicine* 384 (2021): 1564-1565.

14. Cytokine Storm-to the Editor. *The New England Journal of Medicine* 384 (2021): e59.

15. The REMAP-CAP Investigators. "Interleukin-6 Receptor Antagonists in Critically Ill Patients with Covid-19". *The New England Journal of Medicine* 384 (2021): 1491-1502.

16. Christiansen CF., *et al*. "SARS-CoV-2 infection and adverse outcomes in users of ACE inhibitors and angiotensin-receptor blockers: a nationwide case-control and cohort analysis". *Thorax* 76 (2021): 370-379.

17. Albashir AAD. "Renin-Angiotensin-Aldosterone System (RAAS) Inhibitors and Coronavirus Disease 2019 (COVID-19)". *South Medical Journal* 114.1 (2021): 51-56.

18. H Clifford Lane and Anthony S Fauci. "Research in the Context of a Pandemic". *The New England Journal of Medicine* 384 (2020): 755-757.

19. Abeldaño Zuñiga RA., *et al*. "Clinical effectiveness of drugs in hospitalized patients with COVID-19: a systematic review and meta-analysis". *Therapeutic Advances in Respiratory Disease* 15 (2021): 17534666211007214.

20. Zhou B., *et al*. "COVID-19 pathogenesis, prognostic factors, and treatment strategy: Urgent recommendations". *Journal of Medical Virology* (2021): 1-11.

21. Z Wu., *et al*. "Characteristics of and Important Lessons From the Coronavirus Disease 2019 (COVID-19) Outbreak in China. Summary of a Report of 72 314 Cases From the Chinese Center for Disease Control and Prevention". *JAMA* 323.13 (2020): 1239-1242.

22. K T Kadakia., *et al*. "Leveraging Open Science to Accelerate Research". *The New England Journal of Medicine* (2021).

23. Keeley AJ., *et al*. "Asymptomatic SARS-CoV-2 infection: the tip or the iceberg?". *Thorax* 75 (2020): 621-622.

24. Ra SH., *et al*. "Upper respiratory viral load in asymptomatic individuals and mildly symptomatic patients with SARS-CoV-2 infection". *Thorax* 76 (2021): 61-63.

25. Klepikov I. "The Meaning of Pulmonary Reflexes in the Pathogenesis of Acute Pneumonia". *Internal Medicine* 7 (2017): 232.

26. Schwiegk, H. "Der Lungenentlastungsreflex". *Pflügers Archiv: European Journal of Physiology* 236 (1935): 206-219.

27. Thillai M., *et al*. "Functional respiratory imaging identifies redistribution of pulmonary blood flow in patients with COVID-19". *Thorax* 76 (2021): 182-184.

28. Prescott HC and Girard TD. "Recovery From Severe COVID-19: Leveraging the Lessons of Survival From Sepsis". *JAMA* 324.8 (2020): 739-740.

29. Klepikov I. "Acute pneumonia. New doctrine and first treatment results". Lambert Academic (2020).

30. Igor Klepikov. "How many Pneumonias exist in nature?" Generis Publishing (2021).

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