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Commentary

Why does Severe Acute Pneumonia Remain a Stumbling Block for Modern Medicine?

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Abstract

Taking into account the results and actions performed during the previous COVID-19 pandemic waves, it seems that in the last wave in our Region, the decision-making process that concerned patients, both surgical and those who required intensive or critical cares, have been adapted and changed, based on modifications suffered in care indicators, both in terms of available resources and in the volume of activity demanded by both COVID-19 infected or non infected patients operated on.

This article aims to offer some new perspectives on this issue.

Keywords: *COVID-19 Pandemic; SARS-CoV-2 Infection; Safety Management; Surgery*

Acute pneumonia (AP) is a nosological entity that has always encompassed all cases of acute nonspecific inflammation of the lung tissue, regardless of the causative agents. In recent decades, one attempt to improve targeted antimicrobial therapy has been to divide these patients into separate categories depending on the place and circumstances of the disease's onset, which implied differences in the causative agents of the inflammatory process. Although this classification reflected the declining effectiveness of antibiotics and did not bring the expected success, the proposed terminology has become widespread, continuing to largely fulfill a symbolic and psychological role. The largest, in fact almost complete, group consists of patients with so-called community-acquired pneumonia (CAP).

Recently, the introductory sections of many analytical reports and reviews consistently emphasize the heavy burden that CAP place on healthcare systems worldwide, remaining one of the leading causes of hospitalization and mortality [1-3]. Almost invariably, these reports include caveats indicating that this situation has remained unchanged for many years, despite significant progress in the development and application of new diagnostic and treatment methods. It is unlikely that any specialist reading these prefaces pays much attention to them, as they contain contradictions with reality, highlighting the questionable nature of the claimed successes and achievements. The main criterion for success is the results of patient treatment, isn't it? But if it has not been possible to achieve better results and reduce the number of deaths for many years, then what kind of progress and success can we talk about?

The approach to treating AP is still based on an idea that originated about a century ago. According to this concept, known as the microbial theory [4], the main cause of the disease is a microbial factor, and the main method of treatment is antimicrobial drugs. With the advent of antibiotics, this concept became an undeniable dogma. The changes in the conditions and prerequisites for the development of AP, which began shortly after the widespread clinical use of antibiotics, were not properly taken into account as a side effect of these

drugs. Such serious changes as the steady increase in viral forms of AP, including the gradually (initially these were the SARS and MERS epidemics), but steadily approaching SARS-CoV-2 pandemic, have brought medicine to the brink of disaster. The unique experience gained during the pandemic, with difficulty, but nevertheless, has begun to yield its first results.

The didactic uniqueness of the SARS-CoV-2 pandemic lies in the fact that, against the backdrop of established ideas about the dependence of the severity of AP on the type and virulence of the pathogen, nature demonstrated to us its principles of the development of this disease. In the context of the spread of a single type of pathogen, with a pronounced tendency to primarily affect the lungs (COVID-19 pneumonia), the range of variations in this interaction in a huge number of people turned out to be infinitely wide, from asymptomatic carriage to critical cases of the disease [5,6].

And now, practically after the pandemic, it suddenly became clear that, along with the etiology of the disease, the individual reaction of the organism plays an important role in its onset and development, requiring assessment and appropriate correction [7,8]. These are still isolated statements and the first attempts to find verifiable differences in this characteristic. A striking "discovery"! One might think that the uniqueness of our organisms, the specificity and uniqueness of their reaction to equivalent stimuli, were unknown to us? In this case, this timid and somewhat belated reaction to the obvious and impressive results of the pandemic speaks to the depth and strength of those narrow and distorted ideas about the essence of the problem of AP that have formed over a long period of antibiotic use. The simplified representation of the essence of AP through the "antibiotic versus microbe" paradigm has a very strong influence on maintaining the old worldview.

At first glance, it might seem that a new period of fundamental changes and expanded understanding of the problem is beginning. However, one should not rush to hasty conclusions, since examples of research on the body's response and attempts at practical application of the results show that the ideology of the disease is still based on its microbiological characteristics. If, in this context, the inflammatory process is considered as involving the interaction of two factors - the pathogen and the body's defense mechanisms, which should be considered and studied equally - then in reality, the undeniable dependence of the disease symptoms is still attributed to its etiology [9,10]. Consequently, there is a deepening of the study of the nuances of the body's response depending on the causative agent of acute pneumonia.

The research results presented above, demonstrating previously unknown changes at the cellular and molecular levels, can quite rightly be considered successes and achievements. The problem is that this deepening of knowledge at the micro-level does not lead to the expected clinical results, but in such cases, assurances are usually given about the need to continue the work with confidence in future success. After such conclusions, the purpose of continuing the work, which initially did not yield the expected breakthrough, remains unclear. Skepticism regarding the achievement of the stated goals naturally intensifies when considering the current conditions and priorities in shaping the principles of medical care in this area.

On the one hand, many experts point to the predominance of viral forms of pneumonia over bacterial ones [2,9,11]. On the other hand, the advantages of diagnostic panels for identifying bacterial pathogens of pneumonia are actively promoted and advertised, while antibiotics remain the main method of treatment [12,13]. The lack of effective antiviral therapy and the uncertainty regarding the possibility of early use of currently available antiviral drugs, as well as the timing of their effectiveness [14], do not justify the continued widespread but unwarranted prescription of antibiotics. And the main misconception in this tangle of contradictions remains the unwavering belief in the exceptional capabilities of these drugs as the main and only effective method of treatment.

It is interesting to note that adherence to previous principles of providing medical care to patients with community-acquired pneumonia persists during a period when the identification of causative agents does not exceed 30 - 40% [9]. Moreover, many clinicians continue to assert the leading role of pneumococcus in the development of this disease, calling for further improvement of pneumococcal

vaccines [15,16]. The actual percentage of pneumococcal pneumonia among the total number of patients is distorted by considering only those cases where positive results of microbiological diagnostics were obtained, which creates a highly skewed picture. Such statements are more like lobbying for the release of vaccines than the result of a balanced expert assessment.

The data obtained during the SARS-CoV-2 pandemic are of interest not only from the point of view of observing the diversity of responses to contact with a single type of pathogen. With the onset of the pandemic, the number of patients with viral pneumonia increased sharply, putting practical medicine, accustomed to relying on antibiotics and unprepared for such a turn of events, in an extremely difficult position. In this situation, it became possible to observe what truly served as the determining factor in finding the necessary solutions. On the one hand, the treatment of patients with antibiotics continued as before, with their prescription reaching almost 100%, despite the insignificant number of cases of bacterial co-infection [17-19]. The realization of the loss of familiar treatment principles served as a powerful impetus for the search for an antiviral equivalent to antibiotics. The results of these emergency studies proved fruitless [14]. A comparison of the effectiveness of these drugs showed their passive role in the treatment of such patients. Moreover, as is known, antiviral drugs can have a direct effect on the pathogen at the very beginning of the disease, before the viruses penetrate the cells. This last condition is difficult to fulfill in real practice.

On the other hand, numerous studies have emerged focusing on the specifics of viral aggression, with an emphasis on the mechanisms of the observed disorders and methods for correcting them at the cellular and molecular levels. In the latter case, many examples of such research can be cited, including clinical trials and testing of methods for correcting the identified disorders, each of which was considered and presented as a separate and very important cause of the severity of the condition in patients with COVID-19 pneumonia. For example, how much effort was spent on research and heated discussions about the so-called cytokine storm, immune shifts and immunomodulatory therapy, or deviations in the renin-angiotensin-aldosterone system [20-24]. However, after a very short time, in parallel with the decrease in the intensity of the pandemic, interest in such research began to fade, as its hasty conduct in the midst of the crisis did not yield the expected results.

These are far from all the approaches that have been used in the search for an effective way to provide assistance. Moreover, no one describes the clinical equivalent of the specific symptoms of these disorders or a specific target that could be directly observed after eliminating the cause of these disorders. Thus, although a cytokine storm can still be linked to the development of shock, and it is expected that neutralizing cytokines will stabilize the patients' condition, the external manifestations of immune shifts do not even possess this degree of certainty. This approach to solving the problem, where an attempt to correct one of the many micro-links in the disease mechanism should lead to an overall effect, is initially unpromising. The same applies to continuing the search for a successful solution depending on the type of pathogen in general. The answer to the question of why this is so lies in a different plane.

Despite the fact that the causative agent of pneumonia is sometimes attributed characteristics that are not inherent to it, modern medicine continues to assess the severity of the condition in such patients based on functional changes in the lungs and cardiovascular system, which are inextricably linked through the pulmonary vessels and the process of gas exchange. The degree of observed functional disorders in such cases is determined using various diagnostic schemes (APACHE II, SOFA, CURB-65, etc.), in which objective indicators of respiration and circulation are of significant importance. These same methods are used to monitor the further dynamics of the disease and the effectiveness of treatment. It is well known that these diagnostic methods are applicable to all forms of pneumonia, regardless of its etiology. This circumstance unequivocally reflects the constancy of the pathogenesis of pneumonia regardless of the variety of its causative agents, which emphasizes the need for a treatment approach based on pathogenetically justified principles, leaving etiotropic agents as auxiliary therapy.

Such a radical change in the treatment regimen is extremely important, but it will not be sufficient to consistently achieve positive results. First and foremost, attention should be paid to cases of severe AP, where supportive therapy in the form of intravenous infusions

and vasopressors is necessary. Patients with severe AP have been considered and analyzed as a separate category of patients for several decades due to the large number of complications and high mortality rate. However, the principles of their treatment do not differ from the complex of measures for the care of critically ill patients with inflammatory processes of other locations, while the pathogenesis of AP is the complete opposite of the mechanism of development of diseases arising in the systemic circulation. Therefore, the uniformity of these treatment procedures, standard for most inflammatory foci, yields the exact opposite result in patients with AP [25].

A new era is currently dawning, in which artificial intelligence (AI) is playing a priority role in solving many tasks and problems. In light of these developments, many specialists hope to find a solution to the problem under discussion using AI [26,27], thus predicting the integration of molecular research with a shift towards more general characteristics, theories, and understanding [28,29]. However, given that AI operates on the basis of pre-programmed information, from which it generates the final result, but cannot extract missing, yet crucial information "out of thin air," the correctness of achieving the stated goal in this way seems highly questionable. If you want to cook meat, but put dough in the oven, no miracle will happen at the end of the cooking cycle, and a steak will not appear, will it? Therefore, we should not place excessive hopes on AI and delegate to it the part of the work that we are obligated to perform ourselves.

Thus, it is quite clear that the causes and mechanisms of AP are being misinterpreted, which necessitates an immediate revision of the entire concept of this disease, taking into account the unique features of its pathogenesis that have previously been ignored. The concept of AP, formed almost a century ago and remaining unchanged as long as antibiotics provided at least some effect, is clearly outdated, and the side effects of these drugs have long signaled the growing need for a radical revision of the professional understanding of the problem. Without the aforementioned steps and the elimination of contradictions between existing assumptions and real facts, achieving the desired results in the treatment of this group of patients will remain an unattainable dream.

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Conflict of Interest

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