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Editorial

The Solution to the Problem of Acute Pneumonia can be Realized Today

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After centuries of empirical experience in treating acute nonspecific inflammation in the lung (ANSIL), or, to use the more common term, acute pneumonia (AP), significant progress in the treatment of inflammatory processes was achieved by the middle of the last century. Rapid advances in microbiology and the initial results of antimicrobial therapy, especially after the discovery of antibiotics, were perceived by specialists as establishing the cause of the disease and a decisive victory in the treatment of such patients. However, this narrow understanding of the essence of the problem and the subsequent adherence to this ideology, which persisted despite mounting conflicting data without significant correction, led to a cascade of subsequent incorrect conclusions and misconceptions.

Pneumonia was not previously considered a dangerous infection or contagious disease, but antibiotics, with only a specific etiotropic effect, soon became the primary, and then the only, treatment. The inclusion of this topic in university curricula and the requirements of documents regulating medical practice contributed to the strengthening of the so-called microbial concept of pneumonia [1]. The goal and objectives of solving this problem have remained virtually unchanged since their inception nearly a century ago, becoming increasingly ingrained in the worldview of each new generation of physicians. The development and consolidation of professional ideas over many decades has naturally led to results in this field that to this day stand in stark contrast to medical advances in other fields, and pneumonia remains a leading cause of death worldwide [2-4].

The decline in antibiotic effectiveness and attempts to restore it throughout the history of their use have failed to halt this natural process. Currently, the failure to achieve expected results compels a relentless search for ways to improve their effectiveness. Such efforts are motivated by the prevailing understanding of the disease, devoid of a comprehensive critical analysis of the actual circumstances. Existing approaches and methods for finding solutions to the problem under discussion are determined and shaped by a dubious and palliative formulation of the tasks before us. For example, without analyzing the consequences already observed over the long era of antibiotics, proposals have emerged to develop and release new, more advanced generations of antimicrobial drugs [5-7]. Seeking a purely therapeutic effect, the authors of such proposals fail to consider that the percentage of bacterial forms of AP for which antibiotics could retain their essential status has sharply declined, and that the nature of even more serious consequences, if such ideas are implemented, could significantly outweigh the benefits of such therapy.

The unique experience of the SARS-CoV-2 pandemic has exposed the vulnerability of medical care for COVID-19 pneumonia [8,9]. However, this awareness has not led to a radical revision of established principles and approaches to solving this problem. The current situation has returned to the conditions that defined medical care for this group of patients before the pandemic. Specialists in this field are once again faced with familiar challenges, the search for solutions for which has been ongoing for several decades. At the same time, the need to align professional views on the nature of the disease with the fundamental principles of biomedical science and accumulated

objective data is absolutely clear. We are talking about natural phenomena and mechanisms that will continue to operate regardless of our preferences and assumptions. Available materials on this topic allow us to formulate a general plan for the pathogenesis of AP, but this work should be performed by specialists, not by artificial intelligence, which many experts place great hopes on its important assistance in developing such solutions [10,11]. It is important to understand that artificial intelligence will not be able to produce precise results, since it will rely on the information we provide, right?

The development of antibiotic-resistant microflora (ARM) as a side effect of antibiotics was known even before their clinical use [12,13] and has been observed throughout the history of this therapy. ARM began to be perceived as a threatening and frightening factor when the ineffectiveness of antibiotics became absolutely clear. It is no coincidence that the World Health Organization (WHO) loudly declared ARM a global disaster precisely at the peak of the SARS-CoV-2 pandemic [14]. ARM is currently presented as the main reason for the low effectiveness of treatment in patients with AP, which is consistent with the microbial concept of the disease [1] but does not provide a comprehensive explanation.

Resistant strains, having acquired protective properties against external aggression, have not themselves become more aggressive. The only new difficulty is that antibiotics, which form the basis of traditional treatment, are ineffective in the presence of such pathogens. However, in real-world conditions, ARMs among the pathogens causing AP occur in only 1 - 2% of cases [15-17], while latent carriage of such strains is quite common. For example, MRSA alone was detected in 6 - 10% of certain categories of workers 15 - 20 years ago [18-20]. Therefore, ARMs cannot be considered the primary cause of treatment failure in patients with AP, especially since the pathogens causing this disease are often representatives of the commensal microflora and merely contribute to the inflammatory process.

Among the false hypotheses, it's worth noting the persistent belief that the pathogen's qualitative characteristics are factors determining the severity and progression of AP. This thesis hasn't been supported by long-term comparisons of bacterial forms of the disease or even attempts to differentiate bacterial from viral pneumonia [21-23]. Despite the obvious lack of evidence, similar studies continue without previous success [24-28]. The spread of a single pathogen during the SARS-CoV-2 pandemic, with its kaleidoscope of possible reactions in the event of contamination, has forced experts to pay attention to this feature [29,30], but so far this looks more like a "happy discovery" of a long-awaited characteristic than specific solutions and effective recommendations. Some specialists have also begun to pay attention to the long-obvious phenomenon of dynamic changes in the etiology of the disease, noting that viruses increasingly play a leading role [26]. In particular, natural adaptation to the aggressiveness of antibiotics has gradually led to their spontaneous displacement from the list of suitable treatments, which is the main reason for the decrease in the effectiveness of this therapy.

The persistence of the basic clinical picture of AP, regardless of its etiology, convincingly supports the idea that the process's signs are the result of pathogenetic mechanisms, not the consequence of etiological changes. Based on these disease manifestations, it is possible to diagnose or suspect the localization of the process, which cannot be confused with other types of acute inflammation, already during the initial examination of the patient. This also suggests that emergency care for such patients should be pathogenetically justified, and etiotropic therapy, if used, can play a supportive role. Furthermore, the disease symptoms that serve as the basis for diagnosis and patient monitoring are based on classic signs of inflammation, among which "dysfunction of the affected organ" is crucial. This latter circumstance requires a complete revision of the principles of AP treatment in general and, in particular, first aid.

The aggressive development of AP requires immediate and adequate treatment, so a clear understanding of the nature and sequence of pathogenetic mechanisms is crucial. First and foremost, attention should be paid to the characteristics of pulmonary circulation. AP is the only acute inflammation that occurs in the pulmonary circulation. The antagonism between pulmonary and systemic blood flow parameters, as well as the spontaneous regulation of these parameters by the pulmonary vessels, and especially their baroreceptors,

automatically maintain these opposing, yet vital, proportions. This natural compensatory mechanism indicates that assessing and correcting circulatory disorders in patients with AP using the same principles as in other inflammatory processes is a mistake. Ignoring this fact leads to an additional cascade of misconceptions.

With rapid development of the inflammatory process, the inflammation focus is accompanied by a reflex generalized spasm of the pulmonary vessels, which leads to difficulty and reduction of pulmonary circulation [31-33]. The subsequent compensatory restructuring of peripheral blood flow corresponds to the modern understanding of sepsis and shock. The latter, according to the germ theory, is considered septic, but in fact, its development is of pulmonary origin, unrelated to the generalization of infection [31]. Sepsis is currently the leading cause of death worldwide and is considered an independent nosological entity [34,35]. Considering that half or more cases of this formidable complication begin with AP [36], the most severe cases of the disease are reclassified as sepsis. One can imagine the true statistics of AP and the burden of diagnosing pseudosepsis.

The current lack of widespread practice for rapid resolution of reflex pulmonary vasospasm, the primary cause of these generalized disorders, perpetuates the risk of developing conditions such as pulmonogenic

shock, ARDS, and multiple organ failure. Oxygen insufflation in such patients is palliative and provides no significant benefit, as dyspnea, as well as subsequent hypoxemia, is a natural compensatory response to significant shifts in the relationship between blood flow and ventilation, and not, as is commonly believed, a consequence of gas diffusion blockage in the area of inflammation [31]. Etiotropic therapy is considered first-line treatment and requires waiting 48 - 72 hours for results [2,37,38]. Given the rapid spread of inflammation and the time constraints, such a loss of time and chances for success is irreparable. With regard to progressive circulatory disorders allegedly due to infection and the use of general therapeutic principles for its correction, modern medicine widely resorts to intravenous infusions, which, by increasing venous return, hinder the adaptation mechanism and stimulate the development of pathological changes [31].

Addressing the aforementioned shortcomings and incorporating key fundamental principles of biomedical science into the new disease concept will enable us to understand the underlying pathogenesis and treatment principles. Methods such as cervical vagosympathetic block, cupping therapy, or short-term whole-body cooling can be used as first aid. These methods have already been tested using objective control methods, demonstrating their immediate clinical effectiveness [31], and can be recommended for widespread use. A revised version of the reform aimed at combating AP will also help us understand the crucial importance of first and urgent medical care and shift the primary focus of treatment efforts to the earliest stages of the disease, eliminating various options for watchful waiting. Effective early treatment of pulmonary vasospasm addresses the underlying cause of potential mechanisms for subsequent complications, moving from routine attempts at delayed treatment to their effective prevention [31].

All of the aforementioned inconsistencies and contradictions have already been corrected, and the proposed ideas and approaches to solving the problem of AP have successfully undergone clinical trials on a large, representative sample, demonstrating not only excellent treatment outcomes and objective evidence from additional studies and experiments, but also the potential to prevent serious complications [31]. Current approaches to considering pulmonary shock, ARDS, and multiple organ failure in patients with AP as separate pathological conditions and emphasizing their delayed treatment reflect a combination of the aforementioned misconceptions and ignoring favorable opportunities for success. In the first half of the last century, the dominant principle in the United States was to provide the earliest possible care to patients with AP, which in this regard was compared to the diagnosis of acute appendicitis and lost with the advent of antibiotics [1]. Today, the current situation compels us to restore this principle to everyday practice and recognize patients with AP as the most urgent category, based on the location of the inflammation and the high risk of potential complications.

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