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Letter to Editor

The reasons for some misconceptions in the modern concept of acute pneumonia

Igor Klepikov*

Department of MD, professor, retired, USA

***Corresponding author:** Igor Klepikov, Department of MD, professor, retired, Renton, WA, USA.

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Introduction

The successful result of scientific research and the effective solution of practical problems completely depend on the correct definition of their goals. This is an axiom of medical science and practice, without which it is impossible to count on the success of any endeavors. The reliability and verifiability of this rule only increases the feeling of deep disappointment and annoyance when it comes to widespread and persistent attempts to solve the problem of acute pneumonia (AP) in the same way that led it to a state of modern stagnation. The professional stereotype of ideas about the origin and development of AP, which has developed over the past few decades, continues to persistently consider only selective causes of this disease as the basis for a strategic solution to the problem.

A detailed description of the misconceptions and contradictions observed today in the interpretation of the fundamentals of AP, with objective evidence and the results of clinical testing of pathogenetic therapeutic approaches was presented in a special issue [1]. It is impossible to present this voluminous material within the framework of a journal article. Therefore, the text proposed below is devoted to only one, but at the same time the main reason for the existing misconceptions, without eliminating which it is impossible to imagine a consistent and constructive solution to the whole problem.

In this case, we are talking about the didactic effect of antibiotics, as a result of which there was a shift in priorities in assessing the causes and mechanisms of AP development with a loss of attention to a number of important specific factors. The urgent need to draw attention to this side of the problem lies in the fact that such a selective position in the interpretation of the basics of the disease is not perceived today among professionals as gaps in training, and the preservation of such an ideology will not allow us to justify logical and adequate goals and ways to solve the problem.

For many centuries, acute inflammation in the lungs has been considered and treated as an inflammatory process, which with the development of microbiology began to be defined as non-specific in contrast to infectious diseases caused by a specific pathogen. Back in the second half of the 19th century, one of the discoverers of AP pathogens, Gram C [2], presented evidence that this disease can be caused by more than one microorganism. In subsequent years, a significant list of AP pathogens was compiled, which by now has already exceeded a hundred possible variants [3].

By the time antibiotics appeared in medical practice, it was already known that these drugs act only and exclusively on the microbial factor of inflammation, without directly affecting the mechanisms of the process that has arisen. The initial unprecedented effectiveness of the use of antibiotics has demonstrated the ability to provide a rapid therapeutic effect to most patients with inflammatory processes without resorting to other means of assistance. This has allowed for many years to successfully use the principle of "antibiotics alone" in the treatment of many patients with AP.

Over time, antibiotics acquired the image of the main means of helping this category of patients, and the causative agent of the disease, despite the diversity and variability of this concept in this pathology, began to be perceived as the main cause of the occurrence and development of AP. The gradual change of generations in medicine with the emergence of new specialists, whose training was traditionally based on the experience and

professional worldviews of teachers, led to a gradual distortion of views on the essence of AP and the principles of its treatment.

A gradual change in views on the essence of the AP problem occurred in parallel with the inevitable and consistent decrease in the effectiveness of antibiotics. The latter circumstance required the introduction of additional means of assistance, the choice of which was determined by the emerging conceptual idea of the disease. The leading role assigned to the microbial factor in AP allowed the use of techniques by analogy with other inflammatory processes, where such methods justified themselves.

The features of lung inflammation, the localization of which is radically different from the location of the foci of inflammation in any other inflammatory diseases, dictate the need for completely different principles of diagnosis, assessment and correction of disorders observed in the body. The inflammatory transformation of the affected tissues is based on a vascular reaction with a classical sequence of stages. The resulting increase in pressure in the pulmonary vessels is much more dangerous for the body than a similar process on the periphery.

The pressure in the pulmonary vessels is about 5-8 times lower than in the large circle of blood circulation [4,5]. An increase of this indicator by 5 mmHg contributes to interstitial edema, and its increase by 10 mmHg is dangerous for the development of generalized pulmonary edema [6]. The elimination of unexpected pulmonary hypertension, as well as maintaining the necessary proportions and synchronicity in the work between the two circles of blood flow provides autonomous protection of the body in the form of the so-called discharge reflex, emanating from the baroreceptors of the small circle, which was described almost a century ago [7]. This protective reaction is accompanied by a reflex drop in pressure in systemic vessels with a delay in part of the blood circulating in them and a decrease in venous return.

In the conditions of emerging compensatory and adaptive mechanisms, the administration of infusion therapy to patients with AP will be directed against the protective reactions of the body and will have the opposite effect compared to inflammatory processes of peripheral localization [1-10]. An additional factor in the misinterpretation of circulatory disorders in patients with AP is the assessment of such shifts in terms of systemic rather than pulmonary arterial pressure Singer M, et al. [11], which leads to overdiagnosis of so-called septic complications and an even greater intensification of intravenous infusions [1].

The results of such treatment are quite natural and expected, but their negative signs, which continue to grow statistically, are explained by the development of resistance of microorganisms to antibiotics. Currently, microflora resistance is declared by the World Health Organization (WHO) as one of the ten global threats to public health [12]. For the vast majority of the world's inhabitants, this information is a sign of WHO's concern about the health of the population. However, having made such a statement, the experts of this organization do not focus on the fact that for many decades, during which there was an increase in these effects of antibiotics, until recently there were no programs to reduce this burden and rational use of antibiotics.

It is known in the professional environment that the discoverer of penicillin, Fleming A [13], warned about the development of bacterial resistance to antibiotics at the beginning of the marathon of this therapy, and Abraham EP and Chain E [14], who were able to isolate penicillin for practical use in the process of this work, for the first time noted the development of this quality in microorganisms. On the other hand, the danger of antibiotic-resistant forms of bacteria looks like a clear exaggeration, since the same WHO document notes that such strains are increasingly found as symbionts in healthy and unsuspecting people [12].

All this information, representing the resistance of microflora to antibiotics as an unexpected and frightening phenomenon, defies logical and rational explanations, when during the SARS-CoV-2 pandemic in patients with COVID-19 pneumonia, in which the use of antibiotics loses its meaning, and microbial coinfection is detected on average only in 10% of observations, these drugs they are prescribed as the main treatment in 70-80 or more percent of cases [15-18]. But even more puzzling is the proposal of WHO experts to overcome the resistance of microorganisms by further developing more effective antimicrobial drugs [12]. Such recommendations authorize the continuation of competition between pharmaceuticals and microflora, in which the latter has a constant handicap, and the former has only chances of possible success and no chance of winning.

As for reducing the burden of resistant microorganisms with the help of new forms of antibiotics, a number of experts quite rightly and reasonably express doubts about the success of such attempts and consider them undesirable because of the possible deepening of the problem [19-21]. In general, the fixation of attention on the discussion of this section of the AP problem is perceived as a general concern about the decrease in the effectiveness of antibiotics and the desire to return the success of etiotropic treatment of these patients. The encouraging call of some authors for the need to rethink pneumonia actually refers only to a more detailed understanding of the physiology of acute pulmonary infection [19].

Thus, the general atmosphere of ideas about the nature of AP is characterized by the existing confidence in the infectious onset of the disease. This belief was formed under the prolonged didactic influence of antibiotics, which initially, in accordance with their purpose, could not perform the role of the main, and even more so the only means of treating patients with AP.

The occurrence of a focus of inflammation in the lung tissue puts forward a violation of the function of the affected organ, as one of the classic signs of these processes, as the main factor determining the features of clinical manifestations of the disease. That is why inflammatory processes of the same etiology, but of different localization differ in their clinic and the nature of the disorders that occur. That is why repeated attempts at differential diagnosis of pneumonia in accordance with their etiology without microbiological tests have not led and cannot lead to convincing evidence. The latter statement is particularly clearly confirmed by

the lack of clear differences between the inflammatory processes of the lungs of viral and bacterial etiology [22-24].

Etiotropic therapy of AP can bring the expected and noticeable effect only in cases when the disease has not reached the stage of manifest functional disorders. In the latter case, the method of choice should be pathogenetically justified methods of emergency care, which become more important than the desire to suppress pathogenic microorganisms. Such methods of care should be focused on the pathogenesis of the disease, which should be brought into line with the basics of medical science. For example, the modern standard of urgent oxygen supply to a patient with AP, designed to increase its diffusion into the bloodstream, as a rule, does not make decisive changes and does not eliminate respiratory failure. This is due to the fact that circulatory disorders in the respiratory cycle chain cannot be eliminated by oxygen insufflation.

The continuation of the principles of AP treatment, which are based on the priority of etiotropic treatment and remain widespread, will support the trend of further deterioration of results. Attempts to reduce the burden of microflora resistance will not provide a strategic solution to the problem, especially since this phenomenon is not the only biological consequence of antibiotics. Such a trend of recent decades as the constant change of leaders among the pathogens of AP and the extraordinary growth of viral forms of the disease has not yet received sufficient discussion. The first step and the only way out of this situation is not to find ways to preserve the usual stereotypes, but a radical revision of the system of existing views and the approval of a new doctrine of the disease.

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Conflict of interest

No conflict of interest.

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