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Letter to Editor

Is Acute Pneumonia an Infectious Disease?

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Throughout the centuries-old history of the AP, there has been no evidence of the contagiousness of this disease and the danger of its transmission from a sick person to a healthy one. People who became ill with AP were not subjected to isolation and other precautions necessary for infectious processes. There was only a certain seasonal difference in the frequency of this disease, but no epidemics of AP were observed.

The development of microbiology, which originated much earlier than the appearance of antibiotics, did not affect the image of the AP in any way. This disease was still regarded as acute inflammation. With the discovery of *Streptococcus pneumoniae* more than 140 years ago, it was found that it is the most frequent causative agent of AP, as a result of which it received the meaningful name "*pneumococcus*" [1]. However, along with *pneumococcus*, inflammatory processes of the lungs could occur with the participation of other microorganisms, so for a long time the microbiological and pathological essence of the disease was precisely concentrated in the term "acute nonspecific inflammation of the lungs". In this name, the word "nonspecific" emphasizes the absence of a pathogen specific only to AP, as well as the fact that strains of microbes involved in inflammation of lung tissue can be found among symbionts in healthy people [2].

The nonspecific nature and inconstancy of the etiology of AP complicate the scientific explanation of the fact that this inflammatory process, at least over the past couple of decades, has become regarded as an infectious disease. As an example, we can give a couple of brief quotations typical of scientific articles of recent years.

"Community-acquired pneumonia (CAP) is the infectious disease with the highest number of deaths worldwide" [3].

"Among infectious diseases, CAP is the most frequent cause of hospitalization and mortality in industrialized countries" [4].

This interpretation is all the more illogical because during this period there were no drastic changes in the etiology and epidemiology of AP. After *Staphylococcus* lost its leading position among pathogens (after the "staphylococcal catastrophe" in the 60 - 70 years of the last century), pneumococcus returned to its usual place, although its advantage was significantly reduced, accounting for a third of observations among positive bacteriological tests [5-7]. In addition, it should be added that in 62% of patients with AP, the pathogen remains unknown at all [8,9], and, according to some data, this indicator can range from 55 to 74% [10]. How justified are the claims about the infectious nature of the disease, if in most cases there is no accurate information about the etiology of AP?

Epidemiological situation around patients with AP (before the coronavirus pandemic) it also did not change, and they were still in free mode. The only exceptions were patients who had an antibiotic-resistant flora. However, apart from the fact of this diagnosis and additional difficulties in choosing antimicrobials, no other special manifestations were observed in the treated patients. In addition, the probability of the presence of antibiotic-resistant strains among the symbiotic microflora in healthy people has already become a familiar fact, although such findings were not equivalent to a mandatory disease [11-13].

The distortion of views on the nature of AP occurred gradually along with a decrease in the effectiveness of antibiotics and an increase in the resistance of microflora. Acute inflammation of the lung tissue for a long time after the start of the use of antibiotics continued to be considered as a nonspecific inflammatory process, but not as an infectious disease. However, a further decrease in the effectiveness of antibiotics, instead of a critical reassessment of their place in the surrounding reality and the development of side effects, was accompanied by a growing desire to preserve their effectiveness. The tendency to solve the problem of AP with the help of etiotropic therapy has grown with each new generation of doctors, accustoming them to the idea of the leading role of the infectious principle in this disease.

The attribution of bacterial forms of AP to the category of infectious diseases is the result of a long and excessive perception of the role of antibiotics as the main means in the treatment process. The introduction of this term (infectious) in the description of the AP has no reasoned scientific basis and only further narrows the view of the problem, creating additional difficulties in solving it.

In this context, it is useful to recall the viral forms of AP, the pathogens of which have the ability to spread rapidly, which requires compliance with epidemiological measures. The frequency of viral lung lesions in recent decades has been characterized by an unprecedented increase in the entire history of AP [14-16], which not without reason allows us to consider this phenomenon as a natural consequence of prolonged exposure to antibacterial drugs. If earlier banal bacterial inflammations were often the result of respiratory viral diseases, during which the number of such patients increased, then in recent years the viral expansion has grown so much that the growth of these forms of AP in the autumn-winter period is already becoming an annual ritual, which periodically takes on the character of epidemics [17] and even pandemics.

With the viral etiology of AP, the term "infectious" may correspond to such characteristics of the disease as the ease of transmission of the pathogen and the need to comply with anti-epidemic measures. However, even the dangerous ease of infection does not mean a mandatory disease. According to the statistics of the current SARS-COV-2 pandemic, the causative agent of which experts assess as particularly virulent, the overwhelming number of infected people (up to 80%) overcome this contact without resorting to medical care, and in a fifth of them the infection usually remains asymptomatic [18-21].

Another block of accumulated information concerning AP is most directly related to the term "infectious". It is well known and proven by time that a revolutionary step in the prevention of many infectious diseases was made thanks to the development and implementation of vaccination. The modern interpretation of AP as an infectious disease suggested the idea of such vaccinations to prevent acute inflammation of the lung tissue. The rationale for such a company was based mainly on analogies and even assumptions.

In this regard, it is necessary to recall once again that true infectious diseases are distinguished by the strict specificity of their pathogen. The use of a specific vaccine in such situations guarantees almost absolute success. The cardinal distinguishing feature of AP is the absence of one specific pathogen. In the event of conditions for the development of AP, any of the potentially known pathogens can play the role of a trigger factor. To date, more than a hundred microorganisms involved in acute inflammatory processes in the lungs have been identified [22].

The latest information on the spectrum of etiological variants of AP should have raised the question of which specific pathogen a vaccine should be created against in order for its use to bring success. After all, AP didn't have such a separate pathogen before, and the list of the most active strains has been constantly changing lately. In this situation, the lot fell on the creation of an anti-pneumococcal vaccine. And although at the beginning of vaccination against pneumonia, according to available data, *Streptococcus pneumoniae* prevailed among the pathogens, but initially such a campaign could not count on success similar to the prevention of other infections. The pneumococcal vaccine cannot cover most of the list of the most common pathogens of AP.

The first results of vaccination against pneumococcus raised a question that was not given a reasoned answer. Despite extensive and long-term vaccination, the number of patients with severe forms of AP did not decrease, and the number of pleural empyema among them increased statistically significantly [23,24]. The analysis of the results of vaccination of the population against pneumonia according to

the dynamics in the etiology of the disease is very indicative. For example, *Pneumococci* and *Haemophilus influenzae* decreased their presence among bacterial AP factors, but at the same time the participation of pathogens such as *Pseudomonas spp., Staphylococcus aureus* and *Influenza virus* increased [25].

These materials indicate that pneumonia has its own menu of pathogens and the suppression of one of them, even the most active, does not affect the entire process of acute inflammation in the lungs. Such statistics, which often appear in the literature, are only additional evidence of groundless efforts to combat the selected pathogen of AP. One of the manifestations of the consequences of such a company was the so-called staphylococcal catastrophe, which was mentioned above and which arose after a decade of active use of antibiotics.

By choosing *Streptococcus pneumoniae* as the main target and using a specific vaccine against it for preventive purposes for more than 20 years, the medical scientific world has been convinced during this time that the effectiveness of the original version of this vaccine has significantly decreased. To increase its protective properties, a significant correction of this drug was required [26,27]. Moreover, the proportion of *pneumococcus* in the etiology of AP has decreased significantly over the past period, and the latest versions of the vaccine require additional expansion of its range due to the appearance of new modifications of this microorganism [5-7]. In other words, attempts to exterminate one of the bacterial pathogens of AP are accompanied by an increase in its resistance to external aggression factors and an increase in the role of other varieties from the list of microbiological causes of the disease.

The decrease in the overall incidence of AP, which is observed after the start of pneumococcal vaccination of the population, is not the result of a specific action of this procedure. Probably, some strengthening of the protective functions of the body after receiving vaccination should be investigated in the direction of possible shifts in immunity indicators, which may be a general reaction. After all, in general, after vaccination there is no guarantee to avoid the disease, and in the case of the development of the latter, the risk of severe complications has continued to grow in recent years. For example, in England and France, where vaccination of the population against pneumonia has been actively carried out for a long time, pleural empyema as a complication of AP continues to grow, and there are no reasoned explanations for this fact [28,29].

The presented point of view regarding "vaccination against pneumonia" finds unexpected and rather strong confirmation during the current SARS-CoV-2 pandemic. The presence of one causative agent of a new form of COVID-19 pneumonia gave hope in advance that the start of vaccination would radically change the dramatic development of this phenomenon. However, now that in many countries the majority of the population has been vaccinated, it has become quite obvious that vaccination reduces the risk of disease, but does not provide any guarantees of full protection. Moreover, to date, the list of newly ill and deceased among vaccinated people continues to be replenished, although their percentage, according to news reports, is lower than among unvaccinated citizens, but the analysis of generalizing statistics has not yet been given. Meanwhile, the lack of the expected effect of vaccination has caused booster injections, which have been started in many countries.

The above information confirms the old rule that says that people do not get infected with pneumonia, but get sick. Infection of the body means the transmission of a pathogen that does not belong to the representatives of our microbiota, but this fact is not equivalent to the transmission of the disease, since additional conditions are necessary for the development of AP. In this regard, the emphasis on the word "infection" has a double meaning. Such terminology is relatively important only for the epidemiology of viral diseases. At the same time, the general meaning of this term for all variants of AP plays only a negative role. Even without this terminology, AP pathogens are currently considered the only cause of the disease, which is incorrect. Therefore, additional emphasis on the etiology of the process will support and deepen this misconception.

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