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Mini-Review Article

Why does acute pneumonia in its etiology not correspond to the category of infectious diseases?

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Abstract

The substantiation of erroneous ideas about the absolute role of pathogens in the occurrence and development of acute nonspecific inflammation in the lungs is given. The main causes of this phenomenon and ways to correct them are noted.

Keywords: *Acute pneumonia, Etiology, Pathogenesis, Antibacterial agents, Disease doctrine*

The etiology of acute inflammation of the lung tissue is a relatively new section of the disease. The causes and stimuli of acute pneumonia (AP) began to be intensively investigated against the background of more than two thousand years of history only with the advent of microbiology. To date, the ideas about the microbiological features of the etiology of AP are based on a huge amount of information, however, the perception of the accumulated materials, their assessment and subsequent conclusions, with the resulting practical solutions, may have fundamental differences depending on the conceptual view of the essence of the problem.

Since the first description of AP by Hippocrates and up to the 19th century, medicine had a very vague idea of the causes of the development of this disease, but at the same time, all the accumulated experience has convincingly shown that this nosology is not dangerous to others and does not belong to the category of infectious processes. The old postulate that people suffer from pneumonia and but do not get infected continues to be confirmed by the facts of current reality, but the last statement, in the light of the prevailing ideas today, requires details and explanations.

The main causative agent of AP, Streptococcus pneumoniae or Pneumococcus, was discovered only at the end of the 19th century and, due to its predominance in the etiology of the disease, received a specific name. The priority of this microorganism among the causes of AP remained undisputed until the middle of the 20th century, reaching a frequency of 90% to 95% on the eve of the use of antibiotics (1-3). Nevertheless, the disease could not relate to monoetiological processes and therefore was considered as a form of nonspecific inflammation.

The circumstances that have changed over the past decades in the section "Etiology of AP" provide abundant information for reflection, assessments and conclusions. So, by now, the former superiority of Pneumococcus among the pathogens of the disease has been completely lost. Among the positive results of microbiological studies in this category of patients, streptococcal pneumonia occurs in only 10.9%-22.5% (4). At the same time, it should be recalled that during the 1960s and 1970s of the last century, it actually completely, albeit temporarily, lost its leadership to Staphylococcus.

Such grandiose shifts in the etiology of AP, which during the previous period was characterized by a certain constancy, do not allow us to ignore the undeniable fact that the observed dynamics relates exclusively to the period of antibiotic use. The inevitable impact of this type of therapy on the microflora that accompanies the vital activity of our body, with a change in the previous proportions and a shift in the immune response system to maintain the necessary balance with a new set of symbionts, should, in my opinion, be the subject of increased attention and research.

Currently, the World Health Organization (WHO) has declared the development of microflora resistance to antibiotics one of the 10 main global threats to public health that humanity faces (5). It is quite natural that the main reason for the observed evolution of microbes is what WHO experts consider the abuse of antimicrobial drugs, but as a remedy for the dangerous consequences of this biological phenomenon, they consider the creation of more effective means of this action.

The paradoxical and illogical nature of such conclusions, from my point of view, is quite obvious. Simplistically, this means that it is proposed to further improve the factors that themselves caused the irreversible evolution of the microflora. The result of such work, even if it is possible to obtain a short-term therapeutic effect, will be the inevitable continu-

ation and deepening of the process of drug resistance. The inevitability of more severe side effects of such therapy is due to the fact that representatives of the microbial world are separate biological objects with the ability to resist external aggression and adapt to it, which they have successfully demonstrated over the past decades.

In this regard, it is necessary to pay attention to the excessive demonization of antibiotic-resistant strains. The fact of the appearance and subsequent spread of such microorganisms can create additional difficulties only in the treatment of the disease, does not contribute to an increase in the frequency of AP. For example, the possibility of neutral presence of such strains in the microflora of healthy people has long been known and described in the literature, and severe forms of AP are not at all due to the dominance of resistant pathogens.

Thus, Y. Lee and colleagues discovered that in severe forms of AP, the detection rate of methicillin-resistant Staphylococcus aureus (MRSA) in such patients decreased from 12.0% to 2.7% in recent years, while the detection rate of Corynebacterium striatum increased to 13.3% (6). Such statistics only emphasize that the severity of the disease is not determined by the quality of the pathogen, which has continued to demonstrate interchangeability in recent decades against the background of persistent antimicrobial therapy.

After a sufficiently long period of antibiotic use, a stereotype has developed according to which an adequately selected antibiotic is considered the key to success in the treatment of patients with AP. In this context, it is necessary to clarify that the appearance of this stereotype is the result of the action of antibiotics themselves and the consequence of the resistance of the banal microflora, which continues to grow. The initial success of the antibiotic era was achieved with only one drug, and the first striking results were obtained without preliminary bacteriological studies, right?

The initial effect of this therapy was lost after a short period of time, and there was a need for the modernization of drugs, which subsequently became inexorable. To date, the list of such drugs is so extensive that it is often published in the form of separate appendices and reference books. However, the reproduction of more advanced forms of etiotropic drugs, despite the experience and consequences of the entire previous period, continues to be the main hope for improving the results of AP treatment today (5), since the standard of ideas and actions formed as a result of the use of antibiotics considers inflammation of the lung tissue as an infectious process and etiotropic therapy as the main way to eliminate it.

The dominant system of views on the problem of AP today, focusing the efforts of medicine on the search for new effective etiotropic agents, leaves without due attention a rather limited limit of the possibilities of such therapy. As is known, etiotropic drugs are directed against inflammatory pathogens, but they do not have a direct effect on the mechanisms of the disease and regenerative processes in tissues. The destruction of the pathogen with the help of antimicrobial therapy can lead to a state of sterilization of the focus, but further elimination of tissue transformation remains the task of the body.

As you know, damaged tissues lose their protective properties and are a favorable ground for infection. Consequently, the area of inflammation, even after effective drug suppression of the pathogen, remains vulnerable until the morphology and function of tissues are restored. At the same time, the "sterilization" of the focus of inflammation, excluding the participation of the pathogen in it, does not block other neglected mechanisms of the disease. The most obvious example of such a situation can be the observations of the last decades of complicated forms of the disease, in which the so-called sterile empyema of the pleura is formed.

Modern medicine, considering AP as an infectious process, continues to make efforts to determine the specifics of differential diagnostic criteria characteristic of individual pathogens of the disease. The lack of real results from this long-term work with bacterial forms of inflammation of the lung tissue over time began to be recognized by leading experts on this problem (7,8). Similar, but inconclusive, studies are currently being conducted in the hopes of identifying differences between bacterial and viral inflammation as soon as possible (9-11).

From my point of view, the negative results of the above studies should be considered as an important pointer for further action, allowing us to note that the pathogen of AP does not have a specific effect on the mechanisms and dynamics of the disease. The peculiarity of the clinical manifestation and the severity of the disease remain dependent on the localization of the process and the classical specifics of functional disorders of the affected organ. The etiology of AP, as evidenced by the above materials, has no specific features inherent in infectious diseases, and misunderstandings in this matter that arose under the didactic influence of antibacterial therapy require a radical revision

It should be added to the above that during the current SARS-CoV-2 pandemic (Severe acute respiratory syndrome coronavirus 2), the appearance of a new form of AP in the form of COVID-19 pneumonia (coronavirus disease 2019) and the rapid spread of coronavirus are not identical phenomena. While the infection of the population with coronavirus occurs easily and quickly, requiring anti epidemiological measures, the development of the inflammatory process in the lungs retains its selectivity, independent of the presence of the pathogen and uncharacteristic for severe infections. That is, in this situation, we are talking about the intensive spread of a virus unusual for humanity, the most severe and frequent consequence of which is lung damage, but for the development of this complication, simple contact with the pathogen is not enough.

COVID-19 pneumonia statistics show that inflammation in the lung occurs only in 20% of cases of confirmed coronavirus infection. This percentage may actually be even lower, since this indicator is determined in relation to the contingent that passed the test. The number of infected who were not examined and excluded from statistical processing remains unknown, but among those examined, it was found

that 20% of them learn about the presence of coronavirus only on the basis of positive tests in the absence of any clinical symptoms (12-14).

To date, WHO considers the increase in microflora resistant to antimicrobials to be one of the global threats to public health and the cause of the growth of infectious diseases. The reasons for such significant changes in the microflora of the body, including symbionts, are considered to be the abuse of antibiotics (?), unsanitary conditions, poor infection prevention (?) and lack of knowledge (5). Against the background of the description of changes in non-specific forms of inflammation, information about the dynamics of infectious diseases that previously belonged to the category of dangerous infections is of interest.

For example, a leading specialist in infectious diseases in the USA, Dr. Anthony S. Fauci (15), draws attention to the fact that the relative stability in the list of infectious diseases that practical medicine has faced in previous years has begun to change dramatically in recent decades, when new infections arise and some old ones return. Noting these shifts, the author sees their cause in the negative impact of human activity on the environment and, in particular, as a result of climate change. At the same time, antibiotics, as the most aggressive factor in relation to the microcosm surrounding us, are not considered among the possible causes of such dynamics by the author.

Saving millions of lives as a result of the appearance of antibiotics in medical practice is an indisputable fact. However, over a long period of use, drastic changes have occurred not only in its therapeutic effectiveness. The persistent and unconditional introduction of etiotropic approaches to the treatment of AP has led to a distortion of professional views on the nature of the disease, which contradict the canons of medical and biological science and the real facts of the surrounding reality. The irreversible consequences of long-term use of antibiotics observed today make us think about the question: what results, mostly positive or negative, has the possibility of using antimicrobials brought to humanity and is it possible to correct or neutralize the side effects of this therapy?

If the task of eliminating orat least weakening the irreversible effects of antibiotics, such as widespread microbe resistance, necessitates the pursuit of additional solutions and actions, then a radical revision of modern ideas about AP should begin immediately. This step is connected with bringing the existing system of views on the nature of the disease in line with the classical provisions of science and requires our individual assessment of the problem from a different angle. The beginning of this process, with the results of additional studies and clinical testing of other treatment approaches, was successfully laid out a representative work (16).

The dominant utopia today with AP being classified as an infectious disease, which it is not, is the main obstacle to finding a solution to the problem of treating this disease, concentrating the process of research and analysis on the etiology of the process. This direction, which supports the illusion of the absolute role of etiotropic agents in the treatment of AP, already convincingly demonstrates to us the results of these misconceptions over the previous period, and in order to avoid deepening the inconsistencies that have arisen, they must be corrected first.

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