OPINION

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Concepts and reliability of the etiology of acute pneumonia

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Acute pneumonia (AP) remains one of the most pressing health problems in all regions of the world for many years, occupying a leading position among the causes of morbidity and mortality [1-4]. Moreover, the reason for reasonable concern of specialists is a clear trend of reducing the effectiveness of treatment of the disease. This concern is related to the increase in the number of cases requiring hos pitalization and intensive care, as well as to the gradual increase in mortality and morbidity due to purulent complications even in the best health systems of developed countries [5-11]. However, the lack of reasoned scientific explanations of these phenomena and, most importantly, the acute lack of proposals and strategic plans to address this problem, and not only the specific results of treatment of AP and their adverse dynamics, should be of greater concern.

In such a situation, it is logical to expect a deep scientific analysis of the basics of the disease and the principles of its treatment. Meanwhile, contrary to the classical logic of finding solutions, the attention of experts remains stubbornly focused only on ways to suppress the pathogens of the process, and the AP itself, as a result of such persistent narrowly focused efforts, is increasingly considered in the literature as an infectious disease. In this regard, it is extremely surprising and puzzling that many of the provisions of the modern doctrine of the AP are very illusory, do not have proper objective arguments or approved contrary to known scientific data. It is easy to verify the doubtfulness and illogicality of many ideas about the nature and mechanisms of the development of AP, but this must be done in order to understand the essence of traditional misconceptions and to carry out the necessary correction of medical prescriptions. To make the above assessment of the existing concept of AP not look like another Declaration, let's consider the most important sections of the modern doctrine of the disease and pay attention to their objective justification and practical significance.

Agents AP. Over the years, the listing and description of microbes, which are considered to be the most common etiological factor of the disease, occupies a significant place and volume in publications on AP. Such statements, which are usually presented in the form of extended tables, are an integral part of not only textbooks and monographs, but also review and analytical articles on this topic. Microbiological characteristics of AP pathogens are provided by a parallel list of recommended antibiotics, which are the most optimal measure to counter a certain microorganism. All these descriptions and recommendations look quite convincing and scientifically justified, but only on paper. The practical implementation of such projects is very far from such literary declarations, which is explained and conditioned by the following well-known facts.

First, the choice of antibiotic and subsequent treatment is carried out empirically without establishing the actual pathogen in the area of inflammation. The ability to investigate the material, which is taken directly in the lesion, occurs only in a small group of patients at the stage of purulent complications of AP. It cannot be denied that the etiology of the disease remains unproven in the vast majority of patients treated for AP.

Secondly, it is well known that the causative agents of AP are not only microorganisms, but also viruses, fungi and factors such as aspiration or parasites [1-3]. Approximately one third of all AP cases worldwide are associated with viral infection [4]. When the viral nature of the disease antibiotics can no longer be recommended as a targeted etiotropic treatment, and can only be considered as a preventive measure of possible subsequent bacterial stratification. Therefore, in this category of patients, the usual initial appointment in the form of "antibiotics alone" is not actually therapeutic, but only a preventive measure of care.

Third, statistical data on the pathogens of AP, which can be found in the literature, are based mainly on the results of studies of the microflora of the nasopharynx and oropharynx. Such statistics on the etiology of AP is presented even in a press release of such a solid and responsible organization as the Center for disease control and prevention, where the information is based on the materials of a journal article [12,13]. Such an approach to attempts to define the aetiology of AP is widespread and is also discussed in special manuals on this topic [14]. However, there are no convincing counter-arguments against the practical significance of such data in the description and interpretation of these studies. Meanwhile, it is well known that many healthy people have in the symbiotic microflora of the nasopharynx and oropharynx opportunistic microorganisms, which are described as predominant among the pathogens of AP [15]. But the presence of these bacteria in the body does not necessarily mean the development of acute inflammation in the lungs. In addition, to date, the list of identified are pathogens has already exceeded 100 [2] and significantly exceeds the small lists provided in the practical guidelines.

In our study, which was conducted three decades ago, a comparison of the microflora found in the area of inflammation with the microflora isolated from the oropharynx and nasopharynx was started. Such a parallel analysis was possible only in patients with purulent complications of AP. The initial results showed a mismatch in the microbial composition of these sites, so further work in this direction was discontinued, and the data of the first results were not preserved. At present, one can only regret that such an important and illustrative study has not been completed.

Fourthly, the long-term concentration of attention and therapeutic efforts only on the suppression of microflora in AP and the lack of reasoned explanations for the gradual decrease in the effectiveness of such measures led to the interpretation of the onset of the disease as contagious. For example, World Health Organization documents on the subject read:" *Pneumonia* ***can be spread*** *in several ways. Viruses and bacteria, which are usually present in the nose or throat of a child, can infect the lungs when they are inhaled. They may also* ***be spread*** *by air droplets from coughing or sneezing"* [2] [emphasis added]. However, this statement is not supported by any evidence. With regard to viral epidemics, this statement is well known and is unlikely to raise doubts and objections. However, microbial agents of pneumonia are mainly representatives of symbiotic microflora and such a serious conclusion about the possibility of contagion of AP cannot be declarative and requires objective confirmation. and most importantly, there is no further guidance and recommendations for the isolation of patients, which is so necessary in this method of transmission. By the way, it should be recalled that the AP never belonged to the category of infectious diseases and did not require the isolation of such patients, but today already known recommendations for quarantine for carriers of antibiotic-resistant strains.

Fifth, the desire to solve topical issues of AP only through the prism of its pathogen is clearly manifested in the modern results of pneumococcal vaccination. Already the first results of this large company led researchers to confusion [16,17]. If the results of such vaccination had reached their goal, the AP would have repeated the fate of the plague or smallpox. However, contrary to the expected success after many years of vaccination, the number of empyema of the pleura has increased significantly. And the most depressing fact is not so much the end result as the lack of reasoned explanations of this phenomenon. But one explanation for these results is the fact that AP does not refer to diseases with strictly defined pathogens. Therefore, specific protection against pneumococcus cannot give the same effect against other bacteria. In this regard, it should be noted that the same analytical reviews provide, for example, data on a significant reduction in the number of cases of pneumococcal meningitis, for which the microbe is actually a specific pathogen.

Sixth, the dominance of the concept of AP, based on the fatal role of infection in the nature of the disease, and the gradual decrease in the effectiveness of antibiotics as the main means of treatment, logically led to the emergence of another hypothesis, more explaining the difficulties of hospital treatment than reflecting the real situation. In recent years, all cases of AP are divided into non-hospital and intra-hospital. At the same time, nosocomial diseases are considered to be a more dangerous and severe form of damage, which is explained by the concentration of more virulent and antibiotic-resistant microflora in hospitals. Such assumptions are based on the results of sanitary inspection of premises and medical equipment inside such institutions. However, the obvious logic of these fears requires reasoned objective evidence, and there is no such confirmation in the literature on this issue. Ideas about the causative agents of hospital pneumonia are based, as a rule, on the results of a study of the microflora of the oropharynx and nasopharynx, as if we are talking about sinusitis or tonsillitis, and not pneumonia. In addition, most publications on the development of AP in hospitalization of patients consider the onset of the disease as a contact transfer of certain microorganisms.

The last conclusion logically assumes separate isolation of all hospitalized patients and similar decisions at preservation of the existing representations about AP it is quite possible to expect in the foreseeable future, and for now quarantine conditions use only for carriers of antibiotic-resistant strains. The idea of hospital pneumonia as a result of infection of patients with aggressive microflora distracts the attention of researchers from assessing the role of their initial state, which caused hospitalization. People who are sent to the hospital are mostly patients with reduced resistance of the body and other disorders of various organs and systems. In such circumstances, AP will have more severe manifestations and is more likely to lead to serious complications. This direction requires separate monitoring and analysis depending on the nosology of the causes of hospitalization.

Seventh, in recent years, various publications on the topic of AP often contain the authors ' regret about the lack of available rapid diagnosis of pathogens, as well as calls and proposals for the development of such tests. This is another illusory direction, which is based on the idea of the infectious nature of AP. Such calls are aimed at compliance and improvement of the existing principles of treatment of patients with AP, but do not imply a broader and radical solution to the problem. In this regard, it is appropriate to recall that the first experience of antibiotics was not accompanied by a mandatory bacteriological examination of patients before treatment. However, at the time, the blind use of antibiotics was not an obstacle to their triumph. Why today the shortcomings of rapid microflora studies in patients with AP suddenly became one of the main reasons for failures in their treatment?

The answer to this question, as well as an explanation of phenomena such as the slow but steady decline in the effectiveness of antimicrobials and an increase in the number of antibiotic-resistant strains, should be sought in another direction, which is associated with the essence of antibiotic therapy and for anyone today is not a secret. For our body, one of the most important conditions for normal life is its stable balance with symbiotic microflora. The wide and long-term use of antibiotics not only affects the dynamics of the composition of this microflora, which in the example of AP can be considered as a change of leaders among pathogens in different periods of time. Microbes, as biological subjects, demonstrate an enviable ability to adapt to new conditions, which is reflected in a gradual increase in the number of resistant strains. Today it is difficult to

say, how many generations of antibiotics have been changed as a forced measure over the historical period of their use. However, it is necessary to really imagine that such a confrontation between pharmaceuticals and the microbial world has its limit and cannot be endless. Therefore, the continued concentration of efforts only on the suppression of microflora has a temporary tactical success and leads to a dead end [18].

The interpretation of AP only as a process of lung tissue infection not only narrows the format of medical care to antimicrobial measures, but also diverts attention from the important features of the disease that require special methods of care. In situations, when "antibiotics alone" do not give the expected result and the disease is actively progressing, there is a need for hospitalization of the patient and the use of additional methods of assistance. Such observations reveal the problem of modern approaches to the treatment of this group of patients and allow us to note the replacement of specific methods of treatment with General Therapeutic principles. The arguments for the use of such generally accepted methods in the AP are largely based on assumptions, and their actual impact on the dynamics of the process is not objectively verified. The effectiveness of General Therapeutic techniques, which was noted in the treatment of patients with other nosologies, cannot be considered a sufficient argument for the automatic application of the same effort in the presence of inflammation in the lungs. The latter circumstance radically changes the mechanism of action on the focus of inflammation, for example, such a method as intravenous infusion of solutions.

Intravenous infusion. During emergency hospitalization, the patient usually undergoes a procedure to ensure constant access to the vein to control blood tests and intravenous infusions. In such emergency situations, intravenous infusions begin immediately after the installation of the venflon, and the rate of administration of solutions depends on the severity of the patient's condition and an approximate assessment of the volume and quality of losses. However, if, for example, in the case of indomitable diarrhea or peritonitis, the need for fluid return is dictated by visible and significant losses, intravenous infusion in patients with AP does not look so logical and justified.

The purpose of intravenous infusions in AP is usually explained in modern publications by the need to compensate for losses through perspiration and for the purpose of so-called detoxification. In this regard, it is appropriate to ask a few questions. Why patients with acute pneumonia in the first hours of the disease should receive intravenous injections? What catastrophic loss of fluid can occur during this period of the disease in this category of patients? What is actually hidden behind the so-called term "intoxication"? To answer these questions, we need to turn to the scientific facts that form the basis of medical knowledge.

First, AP is an inflammatory process, not only in terminology, but above all in well-studied morphology.

Secondly, inflammatory transformation of tissues occurs due to the vascular reaction, which is based on successive stages of changes in blood flow, blood filling, permeability of the vascular wall. In the case of AP, the anatomical picture of these stages has long been described and is well known [19].

Thirdly, AP is a classic inflammatory process and has all five signs of inflammation, which were described by Celsus and Galen centuries ago- heat, pain, redness, swelling and loss of function. Depending on the localization of the process, the fifth sign (loss of function) is of the greatest practical importance, which determines the features and severity of clinical manifestations of the disease.

Fourth, AP is the only inflammatory process of nonspecific etiology, which develops in the pool of vessels of the small circle of blood circulation. This fact explains the fundamental differences in the causes of homeostasis disorders in AP compared to other inflammatory processes.

Fifth, the vessels of the small circle are a highly sensitive reflexogenic zone, which provides regulation of blood flow and blood pressure between the two circles of blood circulation and has a strong feedback (Schwiegk's reflex).

Sixth, the first barrier, which solutions reach regardless of the place of their intravenous injection, is the pulmonary tissue with a zone of increasing edema and infiltration.

Seventh, one of the main causes of lung function disorders in AP (the fifth sign of inflammation, described by Galen), is the reflex effect of the inflammation zone on the pulmonary blood flow and the subsequent restructuring of the systemic blood supply.

It was on the basis of the above facts that special studies and clinical trials of pathogenetic principles of treatment of AP were conducted, which confirmed the correctness of this approach and radically improved the final results. All these materials have already been published, so there is no reason to cover them again [20-26]. It should only be noted that the direction of therapeutic efforts and their results directly depend on the understanding of the causes of the severity of AP patients. If intravenous infusions are considered as a method of detoxification, the heavier the patient's condition, the more intense the infusion and the faster the x-ray picture of the process in the lung deteriorates. This stimulating quality of intravenous infusions on the dynamics of AP has been noted and documented in our studies. If medical care is aimed at eliminating the reflex effect of AP on pulmonary blood flow, the reduction in the signs of so-called intoxication can be observed without "washing the body". The latter circumstance serves as an additional refutation of the existing opinion on the role of the supervirulent microflora in such negative dynamics of the process. Moreover, such conclusions are not accompanied by objective arguments of microbiological verification of the diagnosis.

Oxygen therapy. Modern recommendations for the treatment of patients with AP necessarily include a section on oxygen inhalation. This recommendation is based on the known results of reducing blood oxygenation in these patients and mainly refers to cases that require hospitalization. But the General

explanation of the causes and mechanism of hypoxemia is, in my opinion, another misconception. Inflammatory changes in the tissues in the gas exchange zone are considered to be the reason for the reduction of blood oxygen saturation. The logic of such reasoning is based on well-known facts. To do this, just look at the results of anatomical studies. Pulmonary tissue in the area of inflammation completely loses its airiness, and pronounced infiltration eliminates any diffusion of gases. However, despite the logic of such reasoning, naturally, the following questions arise.

Why a small focus of acute inflammation in the lung can cause severe violations of gas exchange and we must give for patients the insufflation of oxygen? Why atelectasis of the lobe and even the entire lung usually does not give such heavy changes of respiration?

The answer to these questions we received in patients with AP after procedures that eliminate the reflex effect of the focus of inflammation on the pulmonary blood flow. For example, total initial violations between lung ventilation and perfusion had virtually returned to normal proportions (according to comparative rheopulmonography) after cervical Vago-sympathetic blockade. In parallel, there was a decrease in shortness of breath and an increase in blood oxygenation. The results objectively indicate another mechanism of gas exchange violation in the AP, which was described in previous publications [22-26].

The above considerations and comments are not merely the author's preferences. The presented estimates and arguments are based on long-proven and generally accepted materials of medical science. There is absolutely no doubt that every reader, analyzing modern ideas about the nature of AP in comparison with fundamental scientific facts, will come to this conclusion. Another fact causes reflection and bewilderment. The proposed examination of views on the nature of the AP was carried out 30 years ago and formed the basis of the revised doctrine of the disease. The transformation of views has led to a change in treatment principles and a radical improvement in results [20]. However, over the past three decades, previous ideas about the leading role of microflora in the development of AP and its complications have not changed, but, on the contrary, have focused even more on this factor.

Biological laws exist and operate independently of our worldviews and preferences. Phenomena that are insufficiently investigated and have no scientific explanation are constantly under the scrutiny and study of researchers. It's logical and reasonable. But when the scientific facts that refute the existing idea are already known, and the previous system of views, despite the absence of positive changes in the solution of the problem, remains unshakable, it is impossible to find a reasonable explanation for such intransigence.

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