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Letter to Editor

Acute pneumonia: why is fundamental science resting?

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- Specialists in the field of inflammatory diseases of the lungs are undoubtedly familiar with modern views on the nature of acute pneumonia (AP), the principles of their treatment and the tendency to reduce the effectiveness of this aid. Therefore, I hope that the problem, the essence of which I want to draw attention, will be understood by readers without a detailed review of the literature.

Introduction

Successful treatment of any disease depends primarily on the depth of our understanding of the nature and causes of the disease and the mechanisms of its development. This definition reflects the quality of patient care at all stages of the historical development of medical science and practice and is not debatable. Specialists in the field of inflammatory lung diseases are undoubtedly familiar with modern ideas about the nature of AP, the principles of its treatment and the tendency to reduce the effectiveness of this assistance. Therefore, I hope that the problem, the essence of which I want to draw attention to, will be clear to readers without a detailed review of the literature. The reason for my present appeal is to draw the attention of a wide range of experts to long-known and proven scientific facts, which, despite their importance, remain unclaimed.

AP has been known to medicine for more than two and a half millennia and unlike many infectious diseases has never been considered as a form that requires patient isolation and compliance with special epidemiological requirements. The only exception may be patients with a clear viral etiology of the onset of the disease, especially during periods of widespread epidemics. Therefore, until the middle of the last century, it was reasonable to assume that pneumonia is a disease, not an infection.

The persistent and historically confirmed notion of AP as an inflammatory process of nonspecific etiology began to change with the advent of antibiotics in the therapeutic Arsenal. The first experience of using antibiotics showed that the rapid suppression of one of the main trigger factors of the disease (microbial pathogen) is enough to interrupt the working mechanism of inflammation. Subsequently, despite the gradual decrease in the effectiveness of antibacterial therapy, the opinion that the main and actually the only cause of AP is the microbial factor has increased and

strengthened. Continuing this course of understanding the nature of the disease has led to AP being described and classified exclusively as an infection in recent years.

In this regard, I have no explanation for the paradox and amazing oddities that continue to hypnotize the medical scientific world. Advertising the importance of microbial factors in the AP and fear of its danger is not justified by objective data. There is no need to revise publications on this topic to confirm this fact. It is enough to look at the summary statistics[1] of such a serious and responsible organizations as the National Center for Health Statistics, Centers for Disease Control and Prevention (USA). You can find in these reports any characteristics of patients who were treated with a diagnosis of AP, but you will not find any information about the causative agents of the reported cases. This is logical since this characteristic can be determined only by taking the material for bacteriological examination directly from the focus of inflammation. It is no secret that the vast majority of such patients worldwide are treated empirically without verification of the pathogen. This diagnosis is possible only in a small group of patients in the later stages of the process when there are purulent complications.

In this regard, it is appropriate to make the following comments and reminders. Fear of a certain type of non-specific pathogen is purely declarative, which is further confirmed by the absence of the expected triumph of vaccination against AP. To date, there are already more than a hundred AP agents [2]. In this case, the main role among the causative agents of this disease is played by representatives of the symbiotic microflora, and additional facts only refute the modern "infectious" concepts of AP. For example, it is well known that among us live a certain number of healthy people who are carriers of various potentially dangerous microorganisms. Antibiotic-resistant strains have been added to these microbes in recent years. However, it is also well known that such a latent community can last indefinitely and the presence of one microbe is not enough to start the disease.

The reasons for the beginning of AP are more complex than the banal penetration of microorganisms into the pulmonary tissue. But it is this simplified concept of the onset of the disease is presented in many modern scientific publications. If just inhaling a microbe was enough to start pneumonia, we would be talking about dangerous infections on an epidemic scale. But we know that in real life there is no such danger. An increase in the number of AP patients is usually observed in certain situations, for example, during viral epidemics. The number of cases of AP is much lower than the total number of viral diseases and is rightly considered a complication.

The list of discrepancies between the existing ideas and facts about the role of a microbial factor in the AP is much larger than above. However, in this context we are not talking about details, but about the General system of views on the nature of AP, on which the choice of treatment priorities depends. Analysis of modern concepts of AP indicates the gradual development of phobias before conditionally pathogenic microflora of our body with a clearly overestimated assessment of its role in the nature of the disease.

Such a narrow view of the nature of AP leaves without attention the important factors of the disease pathogenesis. It is significant that all these materials are part of the information for basic medical education, and their mention should not be an unexpected discovery for graduates. The enumeration of these factors shows that the development of the inflammatory process in the lungs cannot avoid their influence on the dynamics and characteristics of the disease. Therefore, it is necessary to have a clear understanding of the role and importance of known biological laws and phenomena in the dynamics of AP to justify adequate approaches to treatment. It is necessary to note only the most important and well-known moments of this information.

So, on the one hand, at the heart of the inflammatory transformation of the tissues of the body is a vascular reaction, which proceeds

according to the standard scenario with a consistent change of stages. Such stages of inflammatory changes are well known on the basis of the study of morphogenesis of lobar pneumonia as one of its most aggressive forms [3]. In other words, acute inflammation of nonspecific etiology, regardless of the localization of the process, develops due to a radical restructuring of the blood flow in combination with an increase in vascular permeability in the affected area.

However, on the other hand, there is one fundamental difference in the results of this reaction for the body when it comes to AP. Of the entire list of currently known inflammatory non-specific diseases, AP is the only process occurring in the region of the small circle of blood circulation. As you know, any inflammatory process is inevitably accompanied by five classical signs, which were described several centuries ago by Celsus and Galen (heat, pain, redness, swelling, 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.

In this regard, if we recall that 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), a new understanding of the pathogenesis of the disease is found.

The beginning of inflammation leads to irritation of the receptors located in the affected area. If during this period of the disease there is no involvement in the process of pleural tissue, then, as a rule, there is no second classic sign (pain), due to the lack of pain receptors inside the lung tissue. However, vascular receptors react to this irritation by generalized spasm of the vessels of the small circle. The sudden change in blood flow conditions now extends not only to the small but also to the large circle of blood circulation. Individual consequences of such a generalized restructuring of blood circulation in the body have an innumerable range, which is due to a variety of combinations between the intensity of the body's reaction to inflammation (its reactivity) and its protective and adaptive ability. Therefore, clinical manifestations of the same disease can range from unclear symptoms to shock.

A new view of the mechanisms of AP development leaves no doubt about the need to radically change the existing approaches in the treatment of this group of patients. This conclusion does not detract from the role and importance of antibiotic therapy in the overall treatment complex, but gives a clear idea of its partial possibility and explains the reasons for the lack of effectiveness of "antibiotics alone". The sequence of links in the pathogenesis of AP is the key to prioritize the use of different methods of treatment, depending on the stage of the process and the Directive for the selection of such methods.

The revision of ideas about the nature of the AP was supplemented by special studies of the author. As a result of this work, a scheme of the pathogenesis of the disease was created and the principles of medical care for these patients were revised. The validity of the new point of view on the solution to this problem is confirmed by the results of clinical studies, which suggest the possibility of guaranteed prevention of complicated course of AP. The results of this work have been described in more detail in the main publications [4, 5]. The main result of this work was the justification and formation of a new doctrine of acute nonspecific inflammation in the lungs. The inevitability of participation and influence of known biological laws and phenomena on the dynamics of AP obliges, first of all, to conduct an appropriate correlation of views on the nature of the disease, as this is the first and inevitable step in solving this problem.

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