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Letter to Editor

Mortality in Acute Pneumonia: Fatal Inevitability?

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The formation of the so-called risk group among patients with acute pneumonia(AP) follows certain criteria and laws, and the principles of this process differ little from the similar division in other diseases. After the initial diagnosis, patients are isolated who have aggressive AP development and who are immediately sent to the hospital for treatment. This group also includes patients with AP with various aggravating factors and concomitant diseases. In the future, the number of hospitalized AP cases increases at the expense of patients, primary treatment of which was ineffective. However, the process of selection of patients with AP for hospitalization is not discussed in this case. In this context, it should be noted that it is in the group of hospitalized patients that fatal cases are recorded.

Further separation of patients with AP and concentration of groups with the most unfavorable prognosis occurs after hospitalization, and modern statistics give a good idea of these trends. Thus, from the number of hospitalized patients with AP from 10 to 18% are sent to intensive care unit, but even the most intensive therapy is powerless in 13 - 28% of patients in this group [1,2]. The situation becomes even more dramatic if AP is accompanied by the development of septic shock, which is observed in 30% of such hospitalizations [2]. When this complication, the mortality rate rises to tragic figures, reaching 36 - 50% [3-5]. In fact, these figures suggest that modern medicine is not able to save one out of every 2 - 3 patients with AP, if the course of the disease is accompanied by septic shock.

The statistics presented today reflect a very unfavourable situation in this area of health, and if we take into account the fact that these figures have not changed for many years and even have a tendency to increase, there is inevitably a sense of hopelessness. However, any situation should have an explanation of the reasons for its development, especially, that it is a question of human lives and this section is in the jurisdiction of medical science. Scientific medical literature considers shock in AP as septic, and the reasons of its development connects, first of all, with the virulence of pathogens of the process and inadequate antimicrobial therapy. Such ideas about the most severe manifestations of AP are not only widespread, but also form the basis of the whole complex of medical care for these patients by analogy with septic shock of another origin.

However, the unreasonableness of such interpretation of shock in AP and illogicality of automatic transfer of the General principles of anti-shock therapy to this group of patients become obvious, from my point of view, if we take into account the following facts. Thus, modern AP statistics provide data that are usually presented, but are not subject to explanation and discussion. For example, it is well known that septic shock develops as a result of the penetration of pathogens into the bloodstream. At the same time, it was found that the penetration of bacteria into the blood can occur in the form of sepsis or be recognized as bacteremia in various inflammatory and infectious processes. If bacteremia is an additional characteristic of the disease and does not have certain clinical manifestations, then septic shock with its bright clinic should, as a rule, be accompanied by the presence of bacteria in the bloodstream.

Based on these assumptions, the septic nature of the shock in AP is not consistent with the statistics. Pathogenic microorganisms in the blood of patients with severe AP are detected with a frequency of only 10 - 12% not only in septic shock, but also include the state of sepsis and bacteremia [1,6,7]. Moreover, sometimes statements about the nature of septic shock in AP in many patients are not based on the results of direct blood culture, and indirect studies, such as urine antigen test [8]. Such tests, which fix the traces of the previous stay

of a certain microbe in the body, from my point of view, do not correspond to the seriousness of the diagnosis and cannot be an absolute argument in favor of the leading role of this pathogen in the dramatic events that occur.

Paradoxes of statistics and illusory ideas about the septic nature of shock in patients with AP can be understood and explained, if we take into account the following indisputable facts. Of all the known acute inflammatory processes that occur in the human body, only AP has its localization in the pool of vessels of the small circle of blood circulation. All other foci of acute inflammation are located within the large circle of blood circulation. This fact is fundamentally important for understanding the mechanisms that cause subsequent changes in the body of patients, and makes us consider the pathogenesis of AP separately from the pathogenesis of other nosologies.

The development of acute inflammation of any localization is accompanied by five classic signs, which were described by Celsus and Galen many centuries ago and which today remain one of the axioms of medical science- heat, pain, redness, swelling and loss of function. In this context, the second and fifth signs of inflammation are of particular importance for understanding the processes that occur and develop in AP.

The pain is well known to every patient who has undergone acute inflammation. The reason for this sign is the irritation of pain receptors in the area of inflammation as a result of changes in chemical proportions and increase in interstitial pressure. It should be recalled that most patients with AP pain syndrome is absent, since the lung tissue has no pain receptors. Pain in AP is usually a sign of involvement of pleural leaves in the inflammatory process.

The absence of pain receptors in the pulmonary parenchyma is abundantly compensated by sensitive elements of a different orientation, which in this place are more important for the body. In particular, pulmonary vessels play a significant role in the regulation of blood flow and systemic pressure. Baroreceptors of pulmonary vessels are one of the most important mechanisms for the automatic preservation of the inverse proportions of blood pressure between the small and large circles of blood circulation (Schwiegk's reflex). This regulation allows to maintain the equality of blood volumes of the two halves of the heart and the synchronicity of their work.

A reminder of the role of pulmonary vessels in the regulation of systemic blood flow belongs to the category of fundamental materials of medical science and cannot be unknown information for specialists with medical education. In this situation, this reminder is necessary in order to understand what mechanisms begin to work in the patient's body in the presence of a focus of acute inflammation in the pulmonary tissue. Irritation of baroreceptors in the focus of AP is an inevitable consequence of inflammatory tissue transformation. Excessive blood filling of this zone causes a protective reaction in the form of spasm of blood vessels to reduce blood flow and inhibit further edema and infiltration. This is one of the mechanisms of self-regulation of our body as a biological system. The principal difference in the manifestation of the results of such a reflex reaction is determined by our individual response to irritation.

The diversity of individual reactions to inflammation, in particular, is well known to immunologists, and the results of research in this area are one of the fundamental foundations of this discipline. The reaction of the body to inflammation and the manifestation of its results are determined by the rate of change. In the case of the so-called hyperergic response, the body has less time and opportunities for alignment and stabilization of functional parameters. Therefore, when observing the aggressive development of the disease to reduce blood flow to the area of inflammation and unloading of pulmonary vessels in our body has no choice but to increase the volume of the vascular bed on the periphery and reduce venous return. In such situations, the reflection of this reaction is the registration of the tendency to hypotension. Another sign of compensatory changes in the AP clinic is shortness of breath, the cause of which is, first of all, a change in the proportions between ventilation and blood flow in the lungs with a predominance of the latter.

A brief description of one of the links of AP pathogenesis is based on already proven and generally accepted facts. Therefore, the presented scenario of functional shifts in the AP will exist and operate independently of our oblivion of these mechanisms. This scheme shows that the basis of the fifth sign of inflammation (loss of function) in AP is primarily a violation of blood circulation, not breathing. It should be added that the proposed concept of disease dynamics has been confirmed by additional studies and clinical results [9-12]. The

causal relationships in the series describes the mechanisms assumed as the real help for these patients, primarily performing procedures to eliminate the reflex influence of inflammation on the cardiovascular system. The effect of such methods of assistance can be objectively recorded immediately after their implementation [13,14].

Unfortunately, modern ideas about the causes and mechanisms of AP development are very simplified and hypnotically tied only to the causative agent of the process. At the same time, the etiology of AP in a huge number of cured patients remains unknown, and antimicrobial therapy has been and is conducted empirically [1,6,15]. Existing views on the nature of the disease and the lack of other ways to help in General practice give grounds for the treatment of AP viral etiology with the help of "antibiotics alone". Partial effectiveness of such treatment is maintained, despite the "blind" use of narrowly focused efforts. This fact suggests that for many patients with AP suppression of a possible rebellion of symbionts is sufficient for the body to cope with the disease on its own.

The essence of the problem, from my point of view, is fully revealed in the aggressive development of the disease. Illusory understanding of the septic nature of shock in AP and automatic transfer of the experience of treating shocks of this origin to this group of patients give depressing results. The principles of shock therapy in patients with AP, which are now common, have the opposite direction from pathogenetic approaches. The body, trying to unload the vessels of the small circle, changes the conditions of blood flow in the periphery. This signal of the body for help is regarded as a lack of fluid and such patients begin to receive intravenous infusion solutions. The area of inflammation in the lungs is the first barrier to these infusions, and an increase in blood flow to this area only exacerbates the mechanisms of the disease. In response to additional loads, the body of many patients does not give the expected effect, so the treatment complex includes vasopressors and hormones [1-3,5-7,16,17].

The above mortality rates are, in my opinion, a natural result of such an impact on the dynamics of the pathological process. In addition, at least 60% of patients with AP treated in hospitals have various pleural complications [1,18]. AP, like many serious diseases, can reach the stage of its development, when the changes in the body become irreversible. Such situations are possible in case of untimely treatment or wrong direction of treatment efforts. Timely pathogenetically based treatment can significantly improve results. This confidence and deep conviction in the possibility of preventing various complications of AP is based on our own experience of treating 994 children with various severe forms of the disease. In the first period of this work, total mortality among patients with AP, admitted to the hospital in the early stages of the disease, exceeded 10%.However, after the revision of the principles of medical care in a comparable group of patients, there were no deaths, and the treatment time decreased sharply [9,19].

More than 3 decades have passed since this work, during which the audit and revision of the concept of microbial factor as the main cause of AP and its complications have not been carried out. On the contrary, this concept has become even more dominant, and the disease itself increasingly belongs to the category of infectious. Such illusory representations do not have conclusive evidence, but contrary to numerous facts. The continuing development of AP of the process in the background and despite the treatment, as well as high mortality rates indicate serious shortcomings in the choice of therapeutic principles. This trend can be reversed only by a radical revision of the doctrine of disease.

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