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Review Article

Features of First and Emergency Care for Acute Inflammation of Lung Tissue

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Abstract

Successful treatment of acute diseases largely depends on the adequacy of first aid. Analysis of such aid and its results in severe forms of acute pneumonia (AP) shows that the initial treatment of this disease does not correspond to the meaning of such a term. The reason for the existing discrepancy is the negative didactic effect of the long-term priority of antibiotics in the treatment of such patients. Such an important feature of this disease as the uniqueness of the functions of the lung tissue remains outside the theoretical and practical implementation. The existing interpretations of the manifestations of AP contribute to the unfounded diagnosis of sepsis, and the results of treatment cause deep concern among specialists. The long-overdue discrepancy between the doctrine of the disease and the observed facts of medical practice has now acquired a pronounced manifestation, which requires the need for a radical revision of views with subsequent correction of treatment principles.

Keywords: Acute Pneumonia, Etiology, Pathogenesis, First Aid, Disease Concept.

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Introduction

In modern medicine, priority in the treatment of acute inflammatory processes belongs to etiotropic therapy, the principles of which are based on the dominance of pathogens in the development of this pathology. The basis of such therapy continues to be antibiotics, the prescription of which plays the role of the first and most important aid. In situations where such treatment does not bring the expected effect, auxiliary care is used, which widely includes, regardless of the symptoms of the process, infusion therapy and symptomatic agents. Acute nonspecific inflammation in the lungs (ANSIL) is no exception to this rule, since the modern understanding of diseases of this localization completely repeats the current general doctrine of inflammatory processes. ANSIL is actually represented by acute pneumonia (AP), the term for which has been uniform throughout the history of this disease, but has recently begun to be divided according to the supposed etiological features.

For many decades, attention was focused on identifying and suppressing the ANSILS pathogen, while the peculiarity of the localization of such processes with characteristic functional disorders remained out of sight. By now, a huge amount of facts and objective evidence has accumulated that contradict the currently accepted and widespread concept of ANSIL and force us to take a closer look and evaluate the uniqueness and specificity of these diseases. This long-overdue need is an inevitable requirement of the time and its implementation will correct deeply rooted misconceptions, changing the principles and effectiveness of modern therapy.

Discussion

The picture of classic acute inflammation begins with the reaction of the vessels in the affected area and is accompanied by the development of five classic signs described at the beginning of our era by Celsus and Galen. Centuries of experience, which turned these signs into one of the fundamental canons of medicine, have shown that the loss of function is of primary importance for the specificity of the clinical manifestations of the inflammatory process. It is the damage to a certain organ that allows us to identify specific nosologies already during the examination of the patient, which reflects the specificity of functional disorders. At the same time, the etiology of the process

does not affect the clinical features of the disease. For example, pneumococcal pneumonia cannot be confused with pneumococcal meningitis, tonsillitis or otitis media, right?

The uniqueness of the development of AP is that the onset of the inflammatory process originates in the vessels of the pulmonary circulation. The cardinal difference in the parameters and functional features of the vessels of the pulmonary and systemic circulation is known to every graduate of a medical university and does not require additional evidence. Arterial pressure in the pulmonary vessels is approximately 8 times lower than on the periphery (1) and maintaining these proportions is important for the synchronous operation of the two halves of the heart and both circulatory systems. A sudden increase in pressure in the pulmonary vessels by 5 mm Hg leads to interstitial edema of the lung tissue, and by 10 mm Hg - to pulmonary edema (2).

As is known, in real conditions our organism has various protective mechanisms that act autonomously, independently of our consciousness. One of such reactions is the so-called unloading reflex, discovered by H. Schwiegk almost a century ago (3). An important feature of this reflex is a decrease in systemic pressure and deposition of part of the circulating blood on the periphery, which is accompanied by unloading of the vessels of the pulmonary circulation. More than forty years ago, the author of these lines carried out a large work, which included various studies, including an experimental model of AP and clinical testing of new therapeutic approaches to the treatment of aggressive forms of the disease, which from the time of primary diagnosis were purposefully concentrated in our department. However, after emigration, the opportunity to present the obtained results to a wide range of specialists appeared only in recent years (4,5).

The main objective of the work was to bring the concept of AP in line with the classical canons of medical science, which forced us to pay the most serious attention to pathogenetic methods of providing assistance. The use of such methods as cervical novocaine vagosympathetic blockade (CVSB), cupping therapy (CPT) or short-term general cooling (GC) of the patient in the initial period of severe forms of AP brought a quick positive result. Registration of comparative rheopulmonograms made it possible to obtain objective confirmation of the reflex effect of the inflammation focus on pulmonary blood flow, when literally a few minutes after the procedure the ratio between ventilation and blood flow leveled out, a tendency to normalize respiratory and circulatory parameters appeared (4,5).

The clinical symptoms of such severe forms of AP corresponded to the picture of a shock reaction, which disappeared after the above procedures. We interpreted and described this clinical phenomenon as pulmonogenic shock, and considered the mechanism of this reaction as a reflex effect of the focus of pulmonary inflammation on the vessels of the pulmonary circulation with a violation of blood flow in them (4,5). This explanation is quite justified, since in this sector of the vascular system there are baroreceptors that react to the slightest changes in arterial pressure and are one of the main drivers of the so-called unloading reflex. The severity of these manifestations increases as the expansiveness of the inflammatory reaction increases.

The reflex effect of the lesion on the overall pulmonary blood flow has received additional confirmation in recent years using the example of patients with so-called COVID-19 pneumonia. The authors of such studies, studying the results of computed tomography, found a significant decrease in the blood filling of the network of small pulmonary vessels with a diameter of less than 2 mm (6,7). Moreover, the authors of these studies noted that the more pronounced such deviations are, the more patients need oxygen therapy. The latter circumstance does not depend on the size of the lesion, but such symptoms increase as the aggressiveness of the development of AP increases.

Unfortunately, at present the main cause of AP in general and its severe forms in particular remains the inflammatory agent, the suppression of which is the main focus of efforts. The severity of the condition of patients with AP for the diagnosis of possible septic complications is assessed according to generally accepted schemes, in which respiratory and peripheral blood flow disorders are decisive and which are used for all other inflammatory diseases (8,9). However, if signs of respiratory and circulatory disorders in inflammatory processes of peripheral localization can be a manifestation of septic complications, then for patients with AP such disorders are typical reflections of the onset of the disease, aren't they? Moreover, a decrease in systemic arterial pressure in AP, which in aggressive forms of the process serves as a sign of a compensatory reaction, is considered in modern conditions as a critical factor requiring immediate correction with the help of infusion therapy.

The indicated approach to assessing the condition of patients with AP allows sepsis to be diagnosed at the very beginning of the disease (10), and among the sources of septic complications, this disease already exceeds half of all cases of sepsis and septic shock, reaching, according to some data, almost two-thirds of all cases of generalized infection (11-13). Quite indicative is the fact that the complete uncertainty in the success of infusions in such patients is constantly emphasized by recommendations for

the further use of vasopressors. It is no coincidence, in my opinion, that a number of specialists openly report that AP often continues to progress after hospitalization, despite intensive treatment with the development of sepsis and even septic shock (10,14-16).

Due to the particular severity of the course and high mortality, many specialists have recently begun to consider and analyze patients with aggressive forms of AP separately. This approach is very subjective and approximate, although to a certain extent justified, since all the difficulties in solving the problem are concentrated in such patients (17,18). However, primary and emergency care continue to be based on etiotropic therapy. At the same time, the empirical prescription of antibiotics is accompanied by a waiting period of up to 48-72 hours for the first results (19,20). In the conditions of a rapidly progressing process, classifying such care as emergency measures does not seem justified even with a big stretch. In such situations, the prerogative of the pathogen and the choice of antimicrobial agents continues to operate, although, as is known, specific signs and disorders are due to the localization of the inflammatory process. In modern discussions, the fact that an antibiotic acts only on certain types of pathogens, without directly affecting the mechanisms of inflammation is not even discussed. Having only an indirect effect on the underlying disease, antibiotics can in no way claim to be an emergency treatment they actually are today.

The second method, widely used in severe forms of AP development as a first-priority measure, is infusion therapy, which also cannot provide the necessary emergency care to such patients. Moreover, especially in the initial period of the disease, when the phenomena of edema and infiltration in the focus of pulmonary inflammation increase especially intensively and the irritant of the baroreceptors includes the unloading reflex, infusion therapy acts as an antagonist of those compensatory reactions that are aimed at unloading the vessels of the pulmonary circulation. In addition, infusions in this period of the disease can stimulate the above-mentioned phenomena of edema and tissue infiltration, which we have proven in the course of our studies (4,5). That is why we abandoned this method in AP, which only contributed to the rapid achievement of successful treatment.

Another method of providing first aid to patients with AP is oxygen therapy. This procedure deserves attention only because it is usually used first and often at the prehospital stage. It is more in line with symptomatic and supportive therapy, but it is expected that its use will bring at least some relief to patients. For a long time, it was believed that the main cause of gas exchange disorders in AP are infiltrative changes in the lung tissue that block the diffusion of gases, but it was not possible to establish a proportionality between the volume of pulmonary inflammation and a decrease in blood oxygenation. This procedure involves increasing the concentration of oxygen in the inhaled air, which should contribute to an increase in blood oxygenation.

An intense discussion on this topic arose during the SARS-CoV-2 pandemic, when the traditionally familiar hope for antibiotics disappeared and specialists began to pay attention to auxiliary and symptomatic methods. An endless stream of publications in the medical literature on this topic was devoted to various aspects of increasing the efficiency of oxygen supply, the discussion of which extended from the position of patients during the procedure to the comparative use of various technical options for connecting and directing the oxygen flow to the nasopharynx of patients. It is quite obvious that such efforts to improve the symptomatic procedure could not have a significant impact on the overall treatment results of patients with viral pneumonia. However, in this case, another circumstance attracts attention. Among the abundance of publications concerning methods of gas exchange correction, the author did not come across a single work where the analysis of the causes of respiratory failure would go beyond ventilation-diffusion disorders.

As is known, the respiratory function is gas exchange between the outside air and the body's cells. This cycle consists of successive links and if any of them is disrupted, respiratory failure may occur. If methods of improving ventilation and diffusion are used in case of inflammatory damage to the lung tissue, this does not mean that respiratory failure is eliminated. During the discussion, the question of the role of the vascular factor in the development of respiratory failure in patients with AP remained open. But the pulmonary vessels, which are the first in the circulatory system to react to a sudden irritant, in the most severe cases of the disease exhibit their reflex reaction in the form of a generalized spasm, which leads to a change in the proportions between blood flow and ventilation, stimulating an increase in the latter. In this regard, the appearance of dyspnea is an attempt by the body to compensate for disruptions in pulmonary blood flow and use the massaging effect of breathing on blood circulation.

The latter statement may cause skepticism and objections, but it has very convincing objective evidence. Thus, after conducting CVSB, on the control comparative rheopulmonogram, within a few minutes (with the appearance of Horner's syndrome), signs of normalization of ventilation-perfusion ratios with a decrease in the frequency of respiratory excursions and heart contractions, as well as a decrease in the amplitude of the systolic wave were noted (4,5). In other words, such a rapid positive

effect after blocking the autonomic innervation of the lung on the affected side can only be explained by the elimination of the reflex effect of the inflammation focus on the vascular network. These studies were performed at the peak of our capabilities at that time, and today the study of this phenomenon can be significantly expanded. At the same time, the results of our previous studies, as well as the materials of other researchers (6,7) show that oxygen insufflation in patients with AP has a serious obstacle to rapid and complete oxygenation of the blood in the form of generalized spasm of the pulmonary vessels. Removal of this obstruction by one of the above methods (in the most severe cases, CVSB and GC are best) makes the use of oxygen unnecessary in most cases.

Thus, the procedures for the primary treatment of patients with AP, which currently perform the functions of first and emergency care, do not meet such criteria and are not able to immediately provide a noticeable positive result with its reflection in the control functional indicators. Ultimately, the existing approaches to the primary treatment of AP lead to the loss of precious time and do not prevent the development of the process, which, against the background of continuing such therapy, can lead to severe complications. The reason for this failure is the continued concentration of efforts on achieving the effect of etiotropic care. The futility of this direction has been repeatedly confirmed not only by the lack of differential diagnostic criteria for bacterial forms of the disease, but also by the impossibility of such a separation of bacterial and viral pneumonia (21-23). Such results have repeatedly emphasized that the nature of the pathogen is not a factor determining the characteristics of the disease development, and the desire to clarify the microbiological diagnosis of AP by any means and use targeted etiotropic therapy continue to dominate the strategy for solving the problem.

The issue of a detailed revision and rethinking of the existing concept of the disease still remains open and this long-overdue need is constantly being replaced by attempts to use tactical methods. In recent years, the search for criteria in the form of biomarkers, tests and other factors for predicting severe and complicated course of AP and the probability of a fatal outcome has become very popular (24-27). The very principle of this approach and the goals set cause deep disappointment, since they reflect and testify to the recognition of the hopelessness and prophetic nature of the expected results embedded in them. In other words, instead of an active and radical solution to the problem, it is proposed to define criteria that can only predict further individual consequences of the process.

Of the above-mentioned first aid methods, two of them, CPT and GC, have been used for many centuries, although they did not have a clear scientific basis and were determined by trial and error. However, the duration of their popularity suggests that they brought relief to patients. Otherwise, they could not have enjoyed such a long-term recognition. Studying this experience at the current level of knowledge is an important step in solving the problem under discussion, and the first results in this direction have already been obtained (4,5). At the same time, the practice of recent years confirms an indecisive wait-and-see attitude, without targeted attempts to influence the mechanisms of the inflammatory process. For example, when treating patients with COVID-19 pneumonia, many specialists adhered to monitoring patients and choosing the time of their intubation, describing the chosen tactics as a strategy (28,29). However, such a strategy cannot lead to any solutions and successes, since this term hides conventional supportive therapy. Such information is additional evidence that when solving the problem of AP, decisive volitional efforts are needed to change ideas about its essence.

Conclusion

A brief analysis of the current state of the problem of AP shows that the main obstacle to achieving successful results are mental distortions of professional ideas about the essence of the disease under discussion. The formed worldview is the result of a long-term negative didactic influence of the role of antibiotics. The functional uniqueness of the lungs with their complete opposite to other localizations of inflammation remains unaccounted for in the general concept of the disease, but the influence of these features on the development and course of AP will act regardless of our desires and priorities. It is unrealistic to bypass or exclude these factors from the pathogenesis of the disease. They can only be influenced by appropriate methods. The inevitable role of such factors in the pathogenesis of the disease and the importance of their correction when providing first aid for AP is a priority task today, and a quick solution to the latter will help to avoid many human disasters. However, the beginning of this process lies in bringing the concept of AP in line with the canons of medical science.

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Conflict of interest

The author states that he has no conflict of interest.

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