

## To Restore the Principles of Providing Emergency Medical Care for Acute Pneumonia

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To prevent possible objections and disagreements from readers regarding the topic indicated in the title, it is necessary first of all to assess the compliance of modern methods of providing primary medical care for acute pneumonia (AP) with the concept of first and emergency aid. After all, today the problem of providing adequate and effective emergency care for AP is focused on improving the methods that already perform this function. Currently, first-line therapy for this disease consists of etiotropic drugs, which are mainly represented by antibiotics. This group of drugs has been the undisputed leader in the treatment of AP for more than 80 years, but despite a significant increase in viral forms of inflammation, no adequate replacement for these drugs has been found, and antibiotics continue to play the role of the main therapeutic agent.

Antibiotics can neutralize or destroy certain types of bacteria, but they do not act against viruses and do not directly affect the mechanisms of the inflammatory process. Nevertheless, many specialists continue to believe that most patients with mild forms of pneumonia are successfully treated with short courses of "empirical" antibiotic therapy on an outpatient basis [1,2]. The successful elimination of the causative agent of pneumonia, as one of the factors of inflammation, allows the body to cope with the process independently and eliminate it. This scenario of the indirect benefit of antibiotics outwardly retains its original effect to a certain extent, however, the widespread belief in their absolute value and therapeutic indispensability in the professional community is completely refuted by the results of the SARS-CoV-2 pandemic, when 80 percent of the population infected with the coronavirus and receiving outpatient treatment recovered without specific treatment [3,4].

However, all the main problems of this disease are most clearly manifested in the group of patients with severe pneumonia, who have already been repeatedly studied and analyzed as a separate category. Such patients require mandatory hospitalization, and their initial treatment is not limited to the prescription of etiotropic drugs. The severe course of the inflammatory process in the lungs does not allow relying on the effectiveness of antimicrobial drugs, the effect of which can only be assessed after 48 - 72 hours [5-7]. The aggressive development of the lesion is accompanied by a deterioration of vital bodily functions and the patient's condition. This situation does not allow for passive observation of the development of complications and dictates the need for additional treatment methods. Currently, the second main method of initial medical care, starting with the empirical selection of antibiotics, is infusion therapy [8,9].

Intravenous administration of solutions in acute inflammatory diseases is currently considered a mandatory treatment rule, the implementation of which is necessary due to the risk of developing general circulatory disorders. Therefore, "prophylactic" intravenous fluid administration upon hospitalization of such patients is an integral standard, and if there is a tendency towards circulatory disorders, especially hypotension, the volume of such infusions is increased and administered as a bolus [9,10]. However, the validity of this treatment strategy completely loses its force in patients with AP, given the inextricable link and complete opposition of blood flow parameters while maintaining parity of cardiac output and autonomous protective regulation between pulmonary and systemic circulation [11].

In this context, the fundamental difference in vascular response to the onset of inflammation depending on its location becomes quite evident. In inflammation in areas of systemic circulation, overall blood flow initially remains without significant deviations, and the appearance of signs of its disruption, which were absent in the initial stage of the disease, indicates a high probability of generalization of the process, implying the development of sepsis and septic shock. The mechanism of inflammation in lung tissue develops quite differently. Pulmonary vessels contain baroreceptors that regulate fluctuations in arterial pressure in the pulmonary circulation, thereby preventing a life-threatening imbalance between the two halves of the circulatory system. In such situations, the so-called unloading reflex is activated, discovered almost a century ago [12].

The vascular reaction that causes the five classic signs of inflammation instantly and inevitably triggers a reflex response of the pulmonary vessels in the affected area. The fastest and most powerful reaction, extending beyond the inflamed areas of the lung, is observed in the aggressive development of acute pneumonia, when the body does not have time to adapt to the resulting disturbances at the same rate. This reaction manifests itself as a generalized reflex spasm of the vessels of the pulmonary circulation, which in such cases is an uncoordinated protective reaction of the lung tissue against progressive edema and infiltration [13,14]. The reduction in the volume of blood flowing through the pulmonary circulation creates a relative excess of venous return, followed by an overload of the right side of the heart. As a result, a picture emerges that corresponds to the concept of shock, which is of a pulmonogenic, rather than septic, nature, as it is interpreted today [13].

In the described situation, a protective mechanism such as the unloading reflex is not always able to provide adequate adaptation, putting the organism in a critical state. In these circumstances, the fate of a particular organism literally depends on external assistance. According to available data, such assistance can direct the course of the disease in two mutually exclusive directions. The most common treatment method currently used in such situations is the intravenous administration of fluids to the patient. The result that intravenous fluid administration can lead to in the described mechanism should be clear without further explanation [13]. It is because of this approach to treatment during the SARS-CoV-2 pandemic, when the number of patients with pneumonia increased, that many specialists expressed concern about timely intubation and sufficient provision of hospitals with artificial lung ventilation devices, thereby acknowledging the ineffectiveness of existing treatment [15,16]. The results of this strategy have been evident for many years and are quite predictable, regardless of how much concern they continue to cause.

The second approach to providing emergency care to patients with AP involves the earliest possible application of methods capable of eliminating the cause of central hemodynamic disturbances. Relieving generalized vascular spasm and restoring vital proportions in the circulatory system, as well as returning to the necessary balance between circulation and ventilation, are crucial for the further course of events. Methods such as cervical vagosympathetic blockade, cupping therapy, and short-term general body cooling have been successfully tested and objectively proven effective [13]. It should be noted that the latter two methods have a centuries-long history of empirical use, but now they have received scientific justification and have taken their place in comprehensive treatment. These methods are the method of choice for providing first and emergency aid in the initial stages of AP, allowing for the immediate observation of their positive effect [13].

The limited antimicrobial potential of etiotropic drugs and the notion that the severity of AP depends on the type of pathogen, which, despite numerous studies, remains illusory and unproven, convincingly demonstrate the auxiliary role of antibiotics in the treatment of this disease. The indirect effect of these drugs on the inflammatory process does not correspond to the role of a fast-acting agent in the context of an aggressively progressing disease and the concept of emergency care. The pathogenesis of AP and the speed of its development are the main factors determining the urgency of applying pathogenetically justified methods of first and emergency aid to such patients. In the most severe cases of the disease, edema and inflammatory infiltration of lung tissue can progress literally by the hour, which must be taken into account when providing care to such patients. Delays in providing emergency care contribute to the deepening of pathological changes in tissue structures, which complicates and prolongs subsequent recovery.

Despite the fact that the described pathogenetic principles of emergency care for patients with AP were substantiated and successfully tested in the last century [13], for most specialists, the etiotropic approach to treating such patients remains the determining factor. Therefore, today, even though the first quarter of the 21<sup>st</sup> century has already passed, modern medicine, instead of providing guaranteed prevention of serious complications of pneumonia, such as sepsis, shock, ARDS, and multiple organ failure, spends a lot of effort and resources on developing prognostic criteria for these conditions and identifying patients for transfer to the ICU. The expectant management approach after prescribing antibiotics as the main and primary treatment has given rise to a tradition of unhurried and phased medical care. As a result, up to 20 - 25% of patients with AP are transferred to the ICU within the first two days of hospital treatment with suspected sepsis or septic shock [17,18].

The diagnosis of so-called sepsis is an illusory consequence of the microbial concept of AP, and its manifestations disappear after timely intervention aimed at the pathogenetic trigger of the underlying process. Unlike pseudoseptic complications, conditions such as ARDS or multiple organ failure represent manifestations of later stages of the disease with pronounced microcirculatory disturbances. Attempts to correct the situation at the time of diagnosis of these complications are extremely late, therefore, mortality in these groups of patients can exceed half of the cases [19,20].

In conclusion, it should be noted that the long-overdue need to assess and understand the nature of AP based on all accumulated information on this problem, rather than narrow, established dogmas, will allow us to understand why improving etiological approaches does not solve the problem of this disease. The specificity and stability of the clinical picture, corresponding to the dysfunction of the affected organ regardless of the nature of the etiological factor, emphasize the importance and crucial role of pathogenetic factors in this process. This first step will allow us to understand, in the context of rapidly progressing inflammation, the pessimism and futility of expecting an etiotropic effect instead of neutralizing pathogenetic mechanisms. Understanding the priority of pathogenetic methods of treating AP and the importance of their earliest possible application indicates the need to shift the main intensity of treatment from the final stage (ICU) to the initial period, including outpatient treatment. The practical implementation of such a system of medical care will significantly reduce the number of so-called septic complications and the number of patients referred to the ICU, and, most importantly, reduce mortality.

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### Conflict of Interest

The author states that he has no conflict of interest.

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