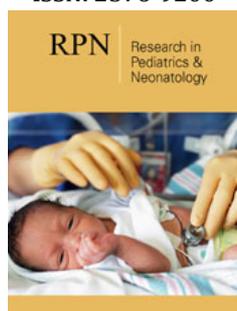


# Acute Lung Inflammation: The Crisis of Etiotropic Treatment and The Logic of Its Solutions

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## Opinion

Modern ideas about the nature of acute inflammatory processes in the lungs are focused on the leading significance of the etiology of the disease. The predominance of this concept defines etiotropic therapy as the basis for providing medical care to this category of patients. In fact, the causative agent of acute pneumonia in most patients remains unknown or the accuracy of its diagnosis is questionable. At the same time, the targeted use of etiotropic drugs is always delayed due to the delay in the results of microbiological diagnostics. Recently, these difficulties of etiotropic therapy have been compounded by an increase in the number of patients with viral lung inflammation and the lack of effective antiviral drugs. The paradoxical and illogical nature of the situation is supplemented by a discrepancy between the intended and actual goals of diagnosis and treatment. Declaring the leading role of pathogens in the development and severity of AP, the assessment of the condition of patients and their further observation within the existing concept of views are based on signs and tests that are due to the pathogenesis of the disease and do not depend directly on its etiology. For example, a cardinal diagnostic test such as an x-ray reflects inflammatory changes in the lung tissue and their dynamics, without having absolute etiological signs. Changes in the parameters of respiration, gas exchange and hemodynamics are the result of impaired lung function as a result of inflammation, regardless of its pathogen.

In other words, the AP pathogen plays the role of a burning match that ignites the hearth, and then the fire continues to spread. But the most important mistake arising from the existing system of views on the nature of the disease is that by focusing on the fight against infection, we lose sight of the unique features of the lesion of the lung tissue in contrast to all other localities of inflammation. The concentration of efforts on the infectious origin does not exclude the use of General Therapeutic measures instead of strictly specific ones. It is well known that the lungs provide not only respiration and gas exchange, but also perform a number of non-respiratory functions, among which one of the most important is participation in blood circulation and its regulation. Having an indissoluble anatomical connection with the system of the great circle of blood circulation, the vessels of the lungs are functionally their complete opposite. Maintaining equal volumes of blood entering each circulatory circle, and simultaneously maintaining the inverse proportions of their blood pressure are the basis of compensatory and adaptive mechanisms in the case of pathological abnormalities. Taking into account these features of pulmonary circulation in the dynamics of AP development is extremely important, especially since it is a scientifically proven and indisputable fact. Acute inflammation of the lung tissue primarily affects the blood flow in the vessels of this organ. However, in practice, correction of circulatory disorders in the body of patients with AP is carried out on the basis of evaluating the parameters of systemic circulation.

In other words, therapeutic efforts are directed not at the cause of pathological deviations, but at their consequence, which has other, directly opposite norms, is a secondary link in the mechanism of the disease and reflects an extreme compensatory reaction. The desire to normalize the indicators of peripheral blood flow without eliminating the root cause (for example, by intravenous infusions, vasopressors, hormones) contradicts the pathogenesis of AP and is not just a paradox, but one of the serious misconceptions in the interpretation of the mechanisms of disease development. At a time when bacterial forms of inflammation prevailed, the attention and main efforts of doctors were focused on etiotropic therapy of AP for a long period. Treatment of this group of patients was considered impossible without the widespread use of antibiotics. The decrease in the effectiveness of antibacterial therapy and the increase in the number of resistant strains during this time had a gradual development without sharp jumps and changes, which did not require accelerated solutions. This dynamic is largely stimulated the development of new antimicrobial drugs than the study of pathogenetic methods of treatment. The sudden increase in the number of severe patients with viral forms of pneumonia and the drop from the General medical list of ways to suppress the pathogen destroyed the usual stereotypes and clearly showed the weaknesses of providing assistance for AP.

It is necessary to pay attention to one undoubted, but very important fact of the current pandemic, which is noted in the modern literature. For example, in the conditions of large groups isolated from the outside world, it was found that not all were subjected to viral aggression, and among the infected, the disease in most cases had an asymptomatic course [1,2]. The authors of these publications are quite rightly concerned about the large number of carriers of the virus with an asymptomatic course and, consequently, the risk of spreading infection. There is no doubt that this data is a serious problem for epidemiologists, but the statistics presented here allow us to draw another important conclusion. In bacterial forms of lung inflammation, the variety of possible pathogens and the difficulty of determining them allowed us to use these reasons to explain failures in the etiotropic treatment of AP. At the same time, the microbial factor did not pose such a threat to the spread of the disease as is observed in coronavirus. Yes, the current pandemic creates conditions for strict compliance with sanitary and anti-epidemic measures. Yes, the number of patients with severe lung damage has increased significantly. But, at the same time, we are talking about monoinfection, which, despite the identity of the pathogen, differs in an infinite variety of clinical manifestations and these individual differences cannot be explained by the features of the etiology, right? These circumstances clearly indicate a distortion of ideas about the absolute significance of pathogens in the problem of AP and the need for a detailed study of the pathogenesis of the disease.

However, the most serious and at the same time delicate result of long-term use of antibiotics is their influence on the formation

of a stable worldview about the leading role of pathogens. This didactic feature is crucial for further study of the problem solution. The very desire to help patients is commendable, but the difficulty of overcoming existing stereotypes is confirmed by modern analysis of the features of medical care for patients with coronavirus disease. In the absence of effective antiviral drugs, contrary to logic and professional prescriptions, most of these patients receive antibiotics without clear direct indications [3-5]. In such conditions, the urgent need for a clear understanding of the classical mechanisms of the development of the inflammatory process in the lung tissue is the basis for providing adequate and effective medical care. Therefore, to get a successful result, it is necessary first of all to conduct a cardinal revision of the pathogenesis of the disease. This revision does not require intensive efforts, because to start this process, it is enough to remember and use the materials of fundamental medical science, which allow us to understand the unique features of the pathogenesis of AP. Moreover, this information has already been systematized and brought into the necessary sequence in accordance with the dynamics of the disease in acute inflammation of the lung tissue. The new system of views on the essence of the problem allowed us to justify a specific approach to the treatment of this particular pathology, and the results of subsequent clinical studies fully confirmed the correctness and effectiveness of pathogenetic methods of treatment.

At present, in the context of the current pandemic with a large number of viral pneumonia, the lack of effective means of suppressing the pathogen and the lack of pathogenetically based treatment methods, it is very important and promising to pay attention to the already proven direction of care for this category of patients. A detailed description of the materials of this work is currently quite available and is presented in the recently published monograph of Igor Klepikov "Acute pneumonia. New doctrine and first results of treatment"

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In this case, we are talking not so much about the publication of a book, but about the results of the work already done, the direction of which requires continuation, as the current situation shows. Classic materials of medical science, which cannot be ignored in the AP, are the basis for a successful solution of the problem, and our attitude to these biological rules and laws will not change their role and significance in the pathogenesis of the disease and will not eliminate the need to develop and apply special methods of treatment. The first task to be solved today is to understand the nature and direction of the body's protective and adaptive responses when an acute inflammatory process occurs in the lungs. This is exactly the way of research that will allow you to form a specific and permanent complex of care for such patients, regardless of the etiology of the disease.

## References

1. Keeley AJ, Evans CM, de Silva TI (2020) Asymptomatic SARS-CoV-2 infection: The tip or the iceberg? *Thorax* 75(8): 621-622.
2. Ing AJ, Cocks C, Green JP (2020) COVID-19: In the footsteps of Ernest Shackleton. *Thorax* 75(8): 693-694.
3. Rawson TM, Moore LSP, Zhu N, Raganathan N, Skolimowska K, et al. (2020) Bacterial and fungal co-infection in individuals with coronavirus: A rapid review to support COVID-19 antimicrobial prescribing. *Clin Infect Dis* pp. 1-10.
4. Kim D, Quinn J, Pinsky B, Shah NH, Brown I (2020) Rates of co-infection between SARS-CoV-2 and other respiratory pathogens. *JAMA* 323(20): 2085-2086.
5. Beović B, Doušak M, Coimbra JF, Nadrah K, Rubulotta F, et al. (2020) Antibiotic use in patients with COVID-19: A snapshot infectious diseases international research initiative (ID-IRI) survey. *J Antimicrob Chemother*, pp. 1-5.

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