

Acute Pneumonia: Infection or Inflammation in the Lung?

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The question in the title of this letter is not a comparison of terms. The answer to this question is fundamental to substantiate the principles of care and further results of treatment of the disease. The choice of the answer to this dilemma determines the strategy for further directions in solving the problem of acute pneumonia (AP). The validity and correctness of this choice will affect not only the effectiveness of further actions and efforts, but also the fate of many patients. To come to a meaningful and reasoned view of the essence of AP, it is enough to analyze the arguments and facts accumulated by medical science and practice.

Over the past decades, the whole strategy in solving the problem of AP is based on the infectious nature of the disease. Modern medicine considers the microbial factor as the main cause of the emergence and subsequent development of this nosology. It is logical that such an interpretation of the nature of AP dictates the concentration of efforts primarily on antimicrobial therapy. From the standpoint of the infectious onset of the disease, differences in the severity of clinical manifestations of AP are explained by the virulence of a specific pathogen, and the lack of effectiveness of modern antibiotic therapy is associated

with the lack of methods for rapid and accurate bacteriological testing. These explanations of the features of AP from the point of view of infection look at first glance reasoned, and an attempt to revise this point of view can be regarded as ignorance and incompetence. The possibility of such a revision of views contradicts the principles of modern medical education in the section AP and job requirements that determine the volume and sequence of medical care in the treatment of this category of patients. However, the need for such an audit is inevitable, as the modern understanding of the nature of AP has many contradictions and declarative statements without a sufficient base of objective arguments.

The formation of views on AP as an infectious process was put with the beginning of the use of antibiotics and this transformation, from my point of view, for many years was paradoxical. On the one hand, each new generation of doctors was brought up in the spirit of the increasing role of antibiotics in everyday practice, which ultimately led to the creation of the brand of initial AP treatment as "antibiotics alone". But, on the other hand, the decline in the effectiveness of antibiotic therapy, the constant search for new drugs

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Received: Apr 08, 2019

Accepted: Apr 24, 2019

Published: May 17, 2019

Editor: Maria Therese, Mother Theresa PG & Research Institute of health Science, Puducherry, India.

and the emergence of antibiotic-resistant strains are increasingly focused on the microbial factor, highlighting it as the main cause of the disease. This transformation of views has been particularly active over the past two decades, and AP is now increasingly referred to and described as an infection.

First, AP has been known to medicine since ancient times, but this disease has never been classified as a dangerous infection that occurs after contact with the patient. True epidemics of this process are unknown in the history of medicine. In other words, the probability of receiving the pathogen AP in the process of contact with the patient does not mean the development of the disease and does not pose a direct danger to the contacting person. However, strangely enough, in the latest documents of the World Health Organization, such probability of direct infection of AP is already declared, without providing proofs and any recommendations [1]. If this transmission pathway is characteristic of viral infections, such a statement is surprising and puzzling about the banal microflora.

Secondly, this disease does not depend only on one specific pathogen, which is one of the main characteristics for typical infectious processes. And although publications usually indicate several varieties of possible pathogens, to date, more than 100 microorganisms have been implicated in the development of AP [2].

Thirdly, even among the most mentioned bacteria there is no stable frequency of their participation in the development of AP. Over the past decades, there has been a change of leaders among the pathogens, which is easily confirmed by comparing the dynamics of the literature data on this indicator [3].

Fourth, the main paradox of modern ideas about the nature of AP is as follows. Despite the fact that the microbial factor is considered to be the main cause of the disease, the etiology of AP in the vast majority of patients remains unknown. In the initial period of the disease, the possibility of taking material for bacteriological examination directly from the inflammation zone is practically absent. This probability occurs only in a small group of patients with pleural effusion in the late stages of the process. The lack of accurate information about the pathogen leads to an empirical choice of antibacterial drugs [4, 5, 6]. In this

regard, the exaggeration of the role of the microbial factor in AP becomes more obvious if we return to the history of antibiotic use. In the initial period of antibiotic therapy AP no one complained about the lack of microbiological diagnosis. The choice of antimicrobial drugs was small and usually consisted in the introduction of penicillin, which did not interfere with the rapid recovery of patients. Now that the resistance of the microflora is increasing and the effectiveness of the latest antibiotics is falling, statements about, that the lack of reliable bacteriological tests in AP is one of the main reasons for the insufficient effectiveness of treatment, every year it is becoming more and more popular explanation.

Fifth, the long-term perception of banal microflora as a fatal cause of AP and the complexity of establishing the etiology of the disease are the basis of the trend of indirect determination of the pathogen by the results of bacteriological studies from the upper respiratory tract [7, 8, 9]. However, the results of such studies from the nasopharynx and oropharynx can not serve as an objective confirmation of the etiology of AP. It is now well known that in these parts of the human body, a whole set of opportunistic microorganisms can be found in healthy people, which are classified as the most dangerous AP pathogens [10, 11, 12, 13]. In other words, the detection of a certain strain in the microbiome does not necessarily mean the development of inflammation in the lungs, and the detection of a certain bacterium in the nasopharynx or oropharynx in a patient with AP can not reliably indicate its participation in the inflammatory process.

Finally, it should be remembered that the pathogens of AP are not only microorganisms, but also viruses, fungi and other factors. Particularly noteworthy is the viral etiology of the disease, which is estimated to account for about a third of all AP cases in the world [2,4]. Such materials cast doubt on the hope of achieving optimal results of AP treatment by improving bacteriological studies and improving the effectiveness of antibiotic therapy. Therapy with "antibiotics alone" of viral processes will not be therapeutic, but a purely preventive measure. In addition, it is appropriate to remember that antibiotics are only a means of suppressing bacteria and do not have a direct impact on the dynamics of the inflammatory process. Elimination of local and systemic manifestations of inflammatory

transformation even under ideal antimicrobial action, it depends entirely on the protective and adaptive abilities of the body.

The general analysis of the above facts shows that the modern ideology of AP is largely based on distorted ideas about the leading role of the microbial factor in the development of the inflammatory process in the lungs and frankly ignores a number of important scientific arguments. At the same time, the existing concept of the disease is rather a constellation of impressions and declarations that emerged in the first years of antibiotic therapy. Despite the changes in this section of treatment compared with its first results, a logical revision of views on the place and role of antibiotic therapy in the overall complex of treatment was not carried out, and ideas about the value of this therapy, on the contrary, even more absolutized.

The current AP policy on the dominant role of some pathogens in the nature of the disease has long been compromised by conflicting facts and a negative trend in treatment outcomes. Such an ideology of disease cannot continue to exist only on the basis of assumptions and guesses. The concept of disease defines approaches to solving the problem, and the stakes are too high to be based on impressions rather than objective criteria and facts. If you start to analyze the modern concept of AP on the basis of known facts and statistics, you can get answers to questions that remain open for many years. For example, why has a long-term widespread "vaccination against pneumonia" not led to triumphant results, similar to the prevention of many infectious diseases? [14,15]. Is it possible to prevent the development of a disease that has dozens of pathogens, creating protection against only one of them? Moreover, if the correct assessment of the real facts, not impressions, was made before vaccination, initially, in my opinion, it was impossible to expect radical changes in the solution of the problem.

The tendency of recent years to present AP as an infection without the lack of evidence of its contact transmission makes it necessary to assess the true role and place of the microbial factor in the development of the disease. One of the areas of such assessment may be the results of experimental studies. And although the results of the experiments can not be an absolute repetition of clinical situations, animal experiments are designed to reproduce the features of the development

of AP and obtain information about the mechanisms of the disease that can not be studied in patients.

In this regard, it should be noted that author's attempts to obtain an AP model in the experiment by banal infection of pulmonary tissue, even in combination with a violation of bronchial patency, were not successful. Violation of bronchial drainage was accompanied only by the development of atelectasis. At the same time, the presence of bacterial culture in the alveolar parts of the lungs did not lead to the process of inflammation.

Completely different results were obtained after preliminary sensitization of animals, and acute inflammation in the lung tissue occurred after the introduction of the permissive dose of the allergen as an immune response [3,16,17]. These data do not correspond to modern ideas about the leading role of microflora in the Genesis of AP and show that bacterial pathogen is only one of the elements of its etiology. The accumulated clinical experience of using antibiotics suggests that this early suppression of the symbiont rebellion is enough for the body of most patients to cope with an outbreak of inflammation.

However, in the case of the development of the inflammatory process of hyperergic type, such narrowly focused medical care is not enough to stop the inflammatory process. In such situations, additional measures are necessary, but the result of such treatment depends entirely on the direction of our efforts, which can both slow down the cascade of pathological transformations, and accelerate this process [17,19-27]. When determining the principles of AP treatment, it is necessary to focus on the inflammatory nature of the disease, which develops in accordance with the biological laws and stereotypes of inflammation. At the same time, we should not forget about the radical difference between AP and all other inflammatory diseases, which consists in the peculiarities of localization and the polar influence on the homeostasis of the body. The last circumstance, from my point of view, is a contraindication to the automatic use of General methods of intensive care for patients with AP. Features of the pathogenesis of AP require the use of special methods of influence on the process, which do not coincide with the action of conventional first aid measures.

Currently, the main obstacle in the successful solution of the problem of AP is a false, in my opinion, the idea of the nature of the disease. The dominant perception of microbial factor as the main cause of this nosology leads away from understanding the unique features of acute inflammation in the lungs. I am very sorry to have to draw the attention of specialists to the obvious inconsistencies between objective facts and modern conceptual provisions. However, any attempt to improve the situation through tactical adjustments could not address the root causes of the problem. The results of AP treatment will continue to show a stable number of failures, as long as medical care for these patients will be carried out by analogy with many other diseases. Moreover, as the resistance of microflora to antimicrobial agents grows, we can expect only the deterioration of General statistical indicators. It's time, when it is necessary to realistically assess the true place of antibiotics in the overall treatment of various diseases and to reconsider the false idea of this undoubtedly important form of care as a "panacea for all diseases".

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