

Is it possible to save on the treatment of acute pneumonia?

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Abstract

This message is intended primarily for specialists in the field of acute inflammatory lung diseases who are familiar with the problems of treatment of acute pneumonia (AP) today and who do not need to provide a review of the literature on this topic. That is why, the title of this letter, I have almost no doubt, will cause many readers at least extreme bewilderment. And such bewilderment in the light of the current situation in this field of medicine at first glance will be quite reasonable and logical. Indeed, what savings in the treatment of AP can be said if, according to the World Health Organization, the existing treatment potential is already extremely insufficient? Pneumonia caused by bacteria can be treated with antibiotics, but only one third of children with pneumonia receive the antibiotics they need. And it is known that "Pneumonia accounts for 16% of all deaths of children under 5 years old, killing 920 136 children in 2015".

Citation: Klepikov I (2018) Is it possible to save on the treatment of acute pneumonia? *Adv Pediatr Res* 5:20. doi:10.24105/apr.2018.5.20

Received: November 27, 2018; **Accepted:** November 28, 2018; **Published:** December 03, 2018

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Competing interests: The authors do not have any competing interests.

Sources of funding: No funding for this research.

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Letter to Editor

In the light of well-known facts, the issue of saving the cost of treatment of patients with AP may look strange and unethical. Although, on the other hand, huge amounts are spent on the treatment of this category of patients in the best health systems, but the current results are of concern to specialists who do not find this trend due explanation [1].

"Pneumonia is a leading cause of hospitalization among children in the United States, with medical costs estimated at almost \$1 billion in 2009. Despite this large burden of disease, critical gaps remain in our knowledge about pneumonia in children" [2].

"Pneumonia puts thousands of young children in the hospital each year at a cost in the U.S. of about \$1 billion, not to mention suffering of kids and hardship for their families" [3].

"Pediatric pleural empyema has increased substantially over the past 20 years and reasons for this rise remain not fully explained" [4].

"The rates of para pneumonic effusions have been increasing in the USA and Europe over recent years, and it is now encountered in approximately 40% of all patients with bacterial pneumonias" [5].

Even more impressive are the results of treatment of AP in adults. "Inpatient mortality rates are as high as 23% in North America. The associated costs of pneumonia in the United States exceed \$17 billion each year" [6].

The above quotes briefly summarize the work of huge health systems. At the same time, both the statistics and their comments show that the reasons for the poor results and the ways to improve them are very vague and leave no hope for the future. Such analysis and conclusions leave no doubt that the issue of AP needs to be critically examined and evaluated from the broadest possible point of view.

A critical analysis of any problem situation begins with an assessment of the relevance of the problem-solving strategy to the depth and range of scientific facts and knowledge in the field. This critical analysis is the first step in solving the problem, as

the strategy determines the nature of further tactics and other necessary amendments.

The modern strategy of AP is based on the leading and dominant role of microbial factor in the occurrence and development of inflammation in the lungs. The elementary penetration of microorganisms into the lung tissue is considered as the beginning of inflammation, and the characteristics of the microbial pathogen serve as arguments to explain the clinical manifestations of the disease. It is quite logical that the consequence of such a strategy is the concentration of the main therapeutic efforts on the choice of antimicrobial drugs. However, if the interested reader begins to analyze modern concepts of AP in the light of the known scientific facts, results of such analysis and comparison, I think, can cause bigger surprise and bewilderment, than the above-stated heading of my letter.

First, as you know, the causative agents of AP are representatives of the symbiotic micro flora of the body. At the same time, it is known that among healthy people there is a certain percentage of hidden carriers of microorganisms, which are considered the most dangerous pathogens of AP. To this well-known fact about the secretive carrier of opportunistic microorganisms should be added the absence of any data on the risk of infection with banal forms of AP or the presence of epidemics in the history of this disease. A certain caveat can only be made about a significant increase in the AP incident during influenza epidemics or other viral infections. However, in this context, pneumonia is rightly seen as a complication of the current epidemics, and not as an independent catastrophe.

Therefore, the presence of a conditionally pathogenic microbe in the body does not necessarily mean the development of AP. To start the inflammatory process, additional conditions are necessary and therefore the microbial factor cannot be the main and only reason to beginning.

Secondly, the generation of fears about a certain type of conditionally pathogenic micro flora is not confirmed by objective facts. The etiology of the vast majority of cured pneumonia remains unknown. In addition, the true causative agent of AP often remains unrecognized even in the case of purulent complications, when it becomes possible to obtain material for bacteriological examination directly from the affected area.

Third, AP is the only inflammatory process of non-specific etiology, which develops in the pool of blood vessels of the small circle of blood circulation. This fact is the key to understanding the pathogenesis of AP and a source of arguments to explain many of the nuances of the disease dynamics. In this regard, it is only necessary to recall the pathophysiology of inflammation and especially the regulation of blood circulation.

Inflammatory transformation of tissues occurs due to the vascular reaction, which is based on successive stages of changes in blood flow, blood filling, permeability of the vascular wall. In the case of AP, the anatomical picture of these stages has long been described and is well known [7].

AP, like any acute inflammatory process, is accompanied by 5 classical signs (heat, pain, redness, edema, loss of function), which were described several centuries ago by Celsus and Galen. Depending on the localization of the process, the fifth sign (loss of function) is of the greatest practical importance, which determines the features and severity of clinical manifestations of the disease.

The vessels of the small circle are a highly sensitive reflexogenic zone, which provides regulation of blood flow and blood pressure between the two circles of blood circulation and has a strong feedback (Schwiegk's reflex).

The beginning of inflammation leads to irritation of the receptors located in the affected area. If during this period of the disease there is no involvement in the process of pleural tissue, then, as a rule, there is no second classic sign (pain), due to the lack of pain receptors inside the lung tissue. However, vascular receptors react to this irritation by generalized spasm of the vessels of the small circle. The sudden change in blood flow conditions now extends not only to the small, but also to the large circle of blood circulation. Individual consequences of such a generalized restructuring of blood circulation in the body have an innumerable range, which is due to a variety of combinations between the intensity of the body's reaction to inflammation (its reactivity) and its protective and adaptive ability. Therefore, clinical manifestations of the same disease can range from unclear symptoms to shock.

Fourth, intensity of development of inflammatory reaction and a chain of the subsequent General violations in an organism of the patient are individual and are caused, on the one hand, by a condition of so-called reactivity of an organism. On the other hand, all these processes are opposed by the protective and adaptive capabilities of a particular person. The combination and counteraction of these two factors gives an infinite number of observed variants of the development of the same disease. Therefore, inflammatory changes in the tissues in the affected area can range from minor edema and infiltration to necrosis, and General changes in the body are manifested from subtle signs to the development of shock conditions.

The above-mentioned scientific facts should not be a revelation for certified specialists, as they belong to the category of basic knowledge acquired during the period of study at the University. However, in this situation, the question arises, what is their role in achieving the goals in the treatment of patients with AP. All these medical and biological principles continue to exist and operate independently of our perception. The fact that modern principles of treatment of AP are focused primarily on the suppression of pathogens without a detailed assessment of the mechanisms of development of the inflammatory process is nothing more than an illusion and self-deception. Antimicrobial therapy can not eliminate the General biological patterns that continue to operate and affect the dynamics of the disease.

A completely different course of events can be achieved if we correctly assess the nature and significance of the existing mechanisms of the disease, as well as the direction of this dynamics and its consequences. First of all, we will face a fundamentally different concept of the disease. A new idea of the driving forces in the dynamics of the development of AP no longer assumes, but forces to reconsider the principles of treatment of this pathology and is the basis for an objective assessment of the impact of individual techniques on the links of the current process.

Experience of such work already exists in the world, and its value and effectiveness have been

tested on representative clinical material. The details of the studies and their results were published first in Russian [8] and then in English [9]. The results suggest the possibility of guaranteed prevention of complications during AP. In addition, the analysis of the results showed that the revision of the doctrine of disease and principles of medical care for patients with AP can significantly reduce the length of stay of patients in hospital and reduce the cost of their treatment at least three times.

The above-mentioned initiatives to audit and transform understanding of the nature of AP and approaches to treating the disease are an untapped reserve for real improvement in outcomes. I hope that the arguments presented on the need for such a test will be sufficient to understand the validity of the question posed in the title of the text and the reality of its implementation.

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