

## Shock in Acute Pneumonia and its Mechanism

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The discovery of antibiotics was one of the greatest achievements of medical science. The introduction of these drugs into medical practice has saved and continues to save millions of lives. At the same time, every year it becomes more and more obvious that antibiotics are not a universal remedy for inflammatory diseases, and their long-term use entails the emergence of new problems. The decrease in the effectiveness of antibiotic therapy, the constant increase in the number of antibiotic-resistant forms of microorganisms, the increase in the frequency of purulent complications of acute pneumonia (AP) make us look for a way out of this situation. AP treatment is almost indistinguishable from medical care in other non-specific inflammatory processes that occur within a large circle of blood circulation. Even in the conditions of coinciding etiology, a significant difference of AP is its localization in the area of the small circle of blood circulation, as a special form of inflammation. It is well known that small and large circles of blood circulation have not only a direct anatomical connection, but also a reverse functional interdependence. In this case, the initial treatment ("antibiotics alone") has an individual effectiveness, and all the failures of subsequent care are explained exclusively by the presence of super-aggressive pathogens. This view continues to dominate despite the growing number of so-called sterile pleural empyema.

The work, the results of which open the possibility of guaranteed prevention of complicated course of AP, was performed in Siberia more than 30 years ago and tested in the clinic [1]. In total, a detailed analysis of the results of treatment of 994 patients with AP and its various complications was carried out. The results of the work were originally published only in Russian. Over the years, the problem of treating AP in the world has only deepened, but other proposals for its strategic solution have not appeared.

The main advantage of this work was the creation of a new doctrine of AP with a reassessment of the role of various factors and mechanisms in its development. A new view on the nature of the disease was based on the classical and axiomatic provisions of medical science and biology with the addition of the results of their own research and experiments.

The set of materials allowed to present the pathogenesis of AP as follows. The banal entry of microbes into the lung tissue is an insufficient fact for the beginning of the inflammatory process. Such microbial penetration becomes fatal in the presence of pre-sensitization of the body and bronchial obstruction. The occurrence of inflammatory infiltration of even a small area of pulmonary tissue leads to irritation of vascular receptors and subsequent widespread spasm of the vessels of the small circle of blood circulation. A sharp restructuring of perfusion in the small circle of blood circulation immediately causes reciprocal changes in circulation in the large circle. Clinical manifestations of such circulatory changes depend, on the one hand, on the rate of development of the inflammatory reaction and on the other hand, on the adaptive capabilities of the body. Individual variants of the confrontation between these two factors give an endless range of clinical manifestations of the disease. The faster and more aggressive the inflammatory process develops, the less time and opportunities the body has for adaptation. In hyperergic type of inflammatory response, the emerging clinic is fully consistent with the picture of shock.

This type of shock was described by us earlier in the scientific literature in Russian [1-4] and was called "pulmonary shock". Pulmonary shock has a fundamental difference in the sequence of its development from other described shock States. All known variants of shock

have the root cause of its appearance within a large circle of blood circulation. The subsequent progression of pathological changes leads to secondary lesions of the microvessels of the small circle of blood circulation, which is known as “shock lung”.

The main changes in OP, on the contrary, first occur at the level of the microcirculatory bed of the small circle of blood circulation. Aggressive development of the process and the inability of the body to adequate and timely adaptation lead to serious violations in the blood supply system of a large circle.

Such fundamental differences in the mechanisms of shock require diametrically opposed measures of medical care. However, the fact of localization of the process in the respiratory system in combination with reflex shortness of breath gives a false idea of the respiratory catastrophe. In this case, the shock observed in AP is regarded exclusively as septic despite the absence of microbiological confirmation of sepsis in many patients.

False ideas about the pathogenetic mechanisms of the disease give rise to the use of inadequate therapeutic efforts. The consequences of such efforts are very evident in the conditions of the only and unique localization of the main cause of the disease in the pool of the small circle of blood circulation. Therefore, when conducting intensive shock therapy AP on the protocols of other shock options, local manifestations of the disease begin to progress catastrophically quickly. It is in this plane that the explanation for the steady growth of complicated forms of AP in the world lies. We saw this in the initial period of the work presented here.

For example, such a common anti-shock measure as infusion therapy increases blood flow to the area of inflammation and stimulates edema and tissue infiltration. The rejection of this technique in the initial period of the disease and efforts to reduce the reflex effect of the focus on pulmonary blood flow gives the opposite result.

A new view on the leading mechanisms of AP development has allowed to radically revise the principles and priorities of medical care to this contingent of patients. The new treatment strategy was used in a total of 203 patients at the time of their hospitalization. The results achieved were irrefutable proof of the importance of the new doctrine of AP for the effectiveness of the treatment of this disease. Current opportunities for research and monitoring of patients allow to expand and deepen the understanding of the pathogenesis of AP on the basis of existing materials.

A more detailed description of targeted research and scientific justification of the new doctrine of acute pneumonia, as well as the results of clinical testing of pathogenetic approaches to the treatment of this disease can be found in the published book [5].

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