Acute Pneumonia is More Cardiovascular than a Respiratory Disaster

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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. OP 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 superaggressive pathogens. This view continues to dominate despite the growing number of so-called sterile pleural empyema.

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. This type of shock was described by us earlier in the scientific literature in Russian and was called "pulmonary shock" [1-4]. 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".

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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 revising 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 expanding and deepening 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].

References