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Opinion

The Myth of Significant Fluid Loss in Acute Pneumonia

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Introduction

Modern ideas about the nature of Acute Pneumonia (AP), formed under the influence of long-term beliefs in the exceptional role and therapeutic indispensability of antibiotics, have left many fundamental laws of medical science without the necessary attention. The search for a solution to the problem of this disease cannot be successful without a revision of the conceptual views that determine both the search directions and subsequent therapeutic efforts.

Currently, almost all urgently hospitalized patients immediately get access to the venous bed and begin to receive an infusion of solutions. This priority of this procedure is due not only to the need to have the most effective way of administering medications, but also to compensate for the loss of fluid, which in acute diseases has many reasons for this. Further recommendations for the correction of water-electrolyte and volume losses and the choice of the infusion rate are determined by the general criteria for their diagnosis in accordance with the parameters of the large circle of blood circulation. Considering AP, first of all, as a result of infection and not focusing on the localization of the process, modern medicine does not make exceptions in this therapeutic direction for patients with inflammation of the lung tissue.

For many years, fever and tachypnea were considered the main causes of fluid deficiency in patients with AP [1]. But the role of these factors in the occurrence of inconspicuous losses is hardly worthy of comparison with the consequences of homeostasis disorders that accompany such diseases as, for example, enterocolitis or peritonitis, when the body really loses large volumes of fluid, and these losses are quite noticeable and can be assessed both quantitatively and qualitatively.

Despite such a significant difference between demonstrative and hidden losses, the recommendations regarding the volume and speed of infusions in severe patients with inflammatory diseases are the same, regardless of the location of the primary focus [2-5]. From my point of view, the lack of liquid in the AP, which occurs in a short time as a result of evaporation, is clearly exaggerated. Practical medicine does not have precise methods for determining the losses expected as a result of perspiration. At the same time, one of the main reasons for the appointment of infusion therapy for AP is the tendency of these patients to hypotension. It is this sign that serves as a guideline for intravenous infusions, since the next recommendation after the start of bolus infusions, which often do not achieve the expected effect, is the introduction of vasopressors to these patients.

The idea of the causes of the severity of clinical manifestations of AP, which today is based primarily on the characteristics of the pathogen, changes significantly if we recall the fundamental foundations of inflammation in general and lung tissue in particular. In modern publications, the mechanism accompanying inflammation in the lungs is not given due attention, so the most severe cases of AP that require intensive treatment are not analyzed as a separate group. The general material for the analysis of such conditions usually includes information about various diseases, in which patients with lung tissue inflammation account for up to 40-50% [3]. The combination of diseases with diametrically opposite pathogenetic mechanisms is a very serious misconception in such analytical work.

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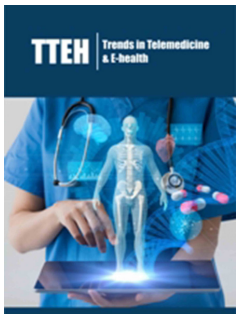

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In this regard, first of all, it is necessary to remember that the basis of the inflammatory transformation of tissues in the affected area is the indispensable development of a consistent reaction of blood vessels with impaired blood flow and increased permeability of their walls, as well as the mandatory accompaniment of these changes with five classic signs of inflammation (heat, pain, redness, swelling and loss of function). The last sign, a violation of the function of the affected organ, plays a leading role in the clinical manifestations of the disease.

But the main feature of the topic under discussion is the fact that AP is the only representative of inflammatory processes occurring in the small circle of blood circulation, unlike all other nosologies localized in the large circle. The inverse proportion of the functional state between the two circulatory circles with their inseparable anatomical and functional connection and interdependence underlies the differences in the pathological mechanisms accompanying the different localization of the primary focus of inflammation. In this regard, the interpretation of the pathogenesis of AP by analogy with other forms of acute inflammation can in no way have the same scenario and monitoring of emerging functional disorders should have a different understanding.

In healthy people, the blood pressure in the pulmonary vessels is always several times lower than in the arteries of the great circle [6,7]. This difference is maintained automatically, allowing the two halves of the heart to synchronously perform their functions and direct equal volumes of blood to completely incomparable vascular systems in anatomical parameters. Maintaining this balance is a vital condition for the body since possible shifts of these functional parallels lead to conditions incompatible with life.

The appearance of a focus of acute inflammation in the vessels of the small circle is a disaster for the body and a cause that disrupts the balance between the two halves of the circulatory system. The localization of this zone not only creates a physical obstacle to the main blood flow, which is ejected by the right half of the heart (Figure 1), but also is a source of reflex spasm of the pulmonary vessels [8-11]. The pressure in the vessels of the small circle increases, and its throughput decreases, creating an excess of venous return. To correct this situation and avoid asynchronous operation of the cardiovascular system, the body changes the parameters of the large circle of blood circulation, reducing the pressure in it and increasing its volume for a sudden "excess" of circulating blood [8].

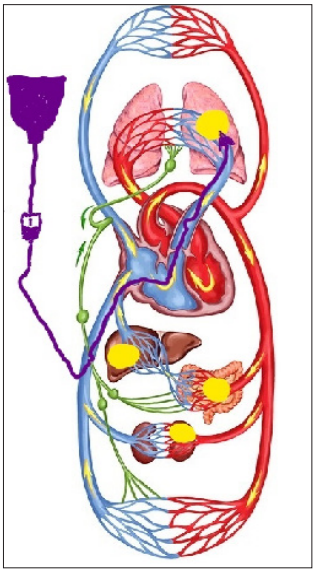

Figure 1: Schematic representation of the human circulatory system.

Explanation 1

a) The comparative value of foci of acute inflammation (yellow fields) for different departments and volumes of blood flow, depending on the possible localization.

b) The initial route of intravenous administration of solutions (dark purple arrow).

Explanation 2

The mechanism of hypotension in the large circle of blood circulation in AP as a result of damage to the pulmonary vessels is especially manifested in the aggressive development of the process. In this situation, it is not difficult to imagine the role played by infusions that increase venous return and additional blood flow to the focus of inflammation.

However, the effect of infusion therapy on the development of AP is a much more complex process than its visual version shown in the figure. The need to clarify the role of infusion therapy in the dynamics of the development of lung inflammation arose in our work many years ago, when the most aggressive bacterial forms of AP began to be purposefully hospitalized in our department during the initial period of the disease. The concentration of a large number of such patients was accompanied by the rapid development of pleural complications and high mortality in them and resembled

the current situation with the isolation of COVID-19 pneumonia. One of our typical examples of the dynamics of the disease is the following observation.

A 2-year-old girl was taken to the clinic with abdominal pain and shortness of breath 12 hours after their appearance. According to her medical history, the child was healthy, but in the last few days she had a mild respiratory syndrome with a runny nose and a cough without fever. Upon admission to the clinic, the patient was diagnosed with AP (Figure 2). Intensive treatment was immediately started, including intravenous administration of two antibiotics and intravenous fluids up to 30ml/kg/hour for 2 hours, followed by a decrease in the infusion rate to 10ml/kg/hour. Despite the treatment, the child's condition did not improve, and a control radiograph was diagnosed with pyopneumothorax 36 hours after hospitalization (Figure 3). The pus obtained from the pleural cavity during drainage was subjected to bacteriological and microscopic examination, but no microflora was found in it.

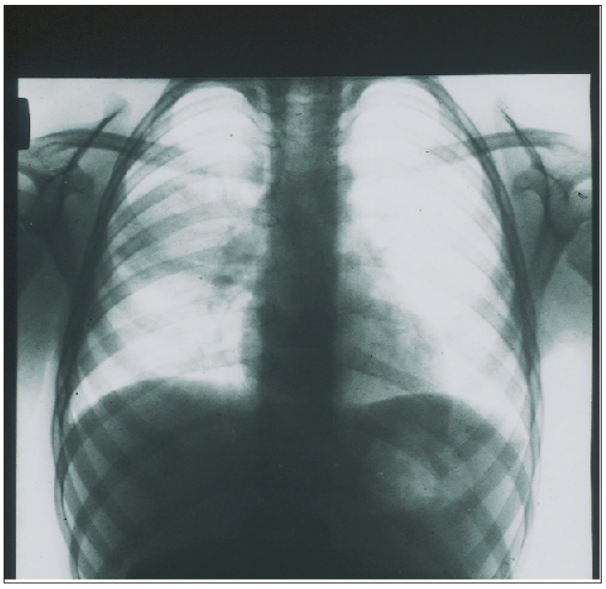

Figure 2: X-ray photograph of 2 years girl 12 hours after the first signs of AP with abdominal pain syndrome were discovered. There is homogeneous shading in a middle-right pulmonary field.

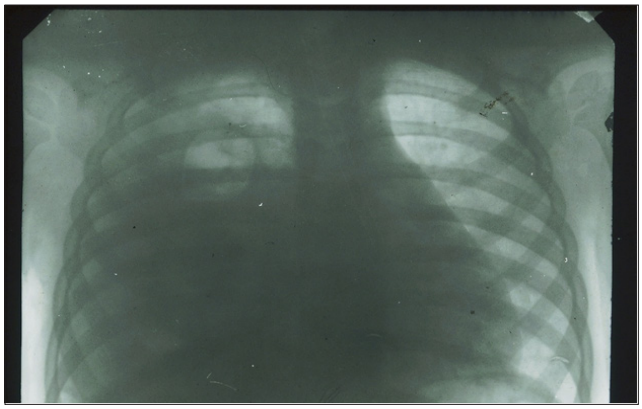

Figure 3: X-ray of the same patient, 36 hours after the start of inpatient treatment. There is an intense uniform darkening of almost the entire right hemithorax with a displacement of the mediastinum to the left, as well as a cavity with a fluid level in the upper pulmonary field.

The presented observation cannot be an absolute proof of the negative effect of infusion therapy on the dynamics of the inflammatory process in the lung. The results of the observed transformation in the area of inflammation only allow us to assume such a dependence and draw appropriate conclusions on an empirical basis. Therefore, in order to find additional arguments in favor of such an assumption, which cannot be obtained in clinical conditions, animal experiments were conducted. The volume of the description of experimental studies does not allow us to present them in the framework of a journal article. However, if it is necessary to obtain this information, it can be found in available sources [12,13]. Only the section of the study that is directly relevant to the issue under discussion is given here.

First of all, a model of the bronchogenic form of AP was created. At the same time, in order to reduce the charismatic etiology of the disease and to assess the significance of other factors, cultures of microbes that are usually not considered as pathogens of AP were used. The choice was made in favor of E Escherichia coli and Staphylococcus epidermidis. When a statistically reliable stable production of the AP model was obtained, in the final series of experiments, intravenous infusions of solutions were administered to rabbits during the occurrence of inflammation in the lungs. The volume of infusions was 30ml/kg/hour and was performed once a day for 3 days. In addition, in 6 cases, the addition of a methylene blue dye to the infusion solution was used. This technique was borrowed from the experiments of V. Menkin, who discovered the permeability factor [14].

The results obtained after euthanasia of animals on the fourth day of the experiment showed the following. Reproduction of the AP model without subsequent intravenous infusions was accompanied by the development of local inflammation of the lung tissue with a slight pleural reaction in some cases. Intravenous infusions in all cases were accompanied by the development of parapneumonic pleurisy. In two cases, pyopneumothorax was detected, the cause of which was small foci of destruction in the lung tissue. After infusions with the addition of dye, weakly colored lung tissues were found along the periphery of the inflammatory focus (Figure 4).

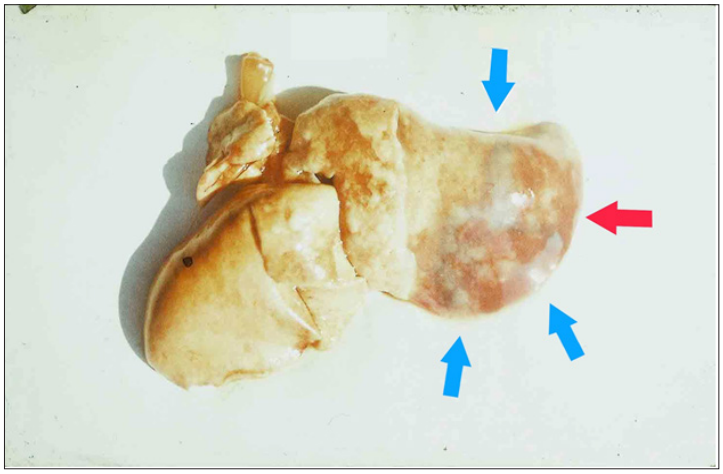

Figure 4: Macro-preparation of the lung (experiment, series 4b). Massive focus of the inflammation in a pulmonary surface (red arrow), surrounded by the additional sections of infiltration with blue shading (blue arrows). Explanations in the text.

Thus, the results of the experiments allowed us to obtain additional and undoubted confirmation of the negative role of intravenous infusions in AP. The use of the dye demonstrated a visual effect of the spread of inflammatory infiltration in the lung tissue, which is a consequence of increased blood flow to the area with increased vascular permeability. In addition, it should be emphasized that, despite the strict repetition of the experimental conditions in each specific case, the final results represented a number of different variants of pathology.

The presented information allows us to analyze the reasons for the continued growth of pleural empyema in patients with AP from a different angle, even in regions with advanced healthcare systems [15,16]. Such an analysis will allow us to evaluate one of the pathogenetic mechanisms of AP and understand why community-acquired pneumonia occurs with parapneumonic effusions in 20-50% of cases, and pleural empyema often turns out to be sterile in microbiological studies [17].

Today, the search for effective methods of treating AP is based on the concept of the disease, which has developed under the influence of long-term and insufficiently critical use of antibiotics. The dominant idea of the leading role of pathogens in the development of the disease and the oblivion of the fundamental laws of life of biological objects form the narrowly focused efforts of this search work and lead to problematic conclusions. This message is intended to draw attention to only one of the existing conceptual misconceptions, without correcting which the further success of treatment of this category of patients looks, to put it mildly, very doubtful.

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