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Opinion

Pathogenesis is the Basis of the Doctrine of Acute Pneumonia and the Principles of its Treatment

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Modern medicine has achieved phenomenal success in many areas, turning into a visual and tangible reality the embodiment of some phenomena that in previous years could only be read in works of science fiction. However, in parallel with the amazing achievements in certain sections of medical science and practice, there continue to be problems to solve which no significant progress has yet been observed. One of these most relevant topics, which has been the subject of active research and lively discussions for many years, is the solution to the problem of acute pneumonia (AP) when the many years of efforts undertaken do not bring satisfaction with their results.

Scientists have long argued that the development of science and our ideas about the phenomenon or subject under study are developing in a spiral. This thesis implies a return to previous ideas and previously obtained materials, which at a certain stage of studying the problem began to be perceived as an anachronism, but as new and additional facts are obtained and accumulated, they begin to acquire scientific significance again, only now at a different, higher and more reliable level of research. The history of one of the oldest medical nosologies, which is AP, can serve as a reflection of this postulate about the development of our knowledge. For most of this historical period, which lasted almost two and a half millennia since the first descriptions of the disease appeared, medicine did not have enough information to scientifically substantiate and formulate the principles of its treatment. Nevertheless, the care of such patients was carried out based on empirically acquired knowledge, with the selection of certain tools and techniques that have been used for many centuries. The explanation for the unusually long period of preservation in the arsenal of medicine of many ancient methods of treatment can only be their noticeable and tangible effect, since in the absence of the latter, such methods of medical care would have long since sunk into oblivion.

The main and very significant drawback of the treatment, compiled based on exclusively practical experience, was the lack of clear ideas about the nature of the disease, the peculiarities of its development, the uncertainty of the mechanism of action of the prescribed treatment methods and the lack of reasoned indications and contraindications for their use. Therefore, such approaches to helping patients with AP did not differ in the clarity of the timing, priority, intensity, and sequence of various procedures, and the achievement of the expected results was rather seen as a hope for good luck than as a natural consequence of a purposeful and understandable impact on the mechanisms of the disease.

More than a hundred years ago, official medicine not only continued to intensively use methods that have been part of its therapeutic arsenal throughout history but also considered them as indispensable first aid tools in the treatment of patients with AP. Among the few publications of that time in medical periodicals were articles devoted to the successful application of such methods as cold wraps of the patient's body and cupping therapy for AP [1,2]. At the same time, it was about the treatment of patients with croup pneumonia as the most severe form of the disease, which currently forms a contingent of patients immediately referred to intensive care units. Such an authority of his time Sir William Osler strongly recommended cupping therapy as first aid for croup pneumonia [3]. However, it should be noted that such recommendations did not have objective arguments confirming the pathogenetic effectiveness of such assistance and were based mainly on clinical experience and observations. The pathophysiological aspects of the disease were still in their infancy.

Assessing the causes of severe croup pneumonia, Professor J. Winters of the Medical College of the University of New York in 1906 argued that nervous shock and the condition of the pulmonary vessels are of paramount importance in this process [4]. Justifying the causes of the severe development of the disease only on the basis of his observations and clinical experience, the author suggested starting treatment of such patients with the appointment of opiate drugs. As the future has shown, such drug treatment did not receive further justification and turned out to be unnecessary in this category of patients, but the insightful assessment of an experienced clinician who was able to consider the main causes of the severe manifestation of the disease was confirmed in subsequent pathophysiological studies, the results of which are included in modern interpretations of its pathogenesis.

Although in the 30s of the last century, one of the most significant discoveries in the pathophysiology of the lungs was made, which is the so-called unloading reflex [5], however, it was still very far from a more detailed understanding of the pathological processes accompanying AP. In the same period of the 30s of the last century, progress in the development of microbiology gave rise to the first attempts to influence the etiology of AP with the help of specific anti-pneumococcal drugs. So, in the USA, the use of anti-pneumococcal antiserum as a therapeutic method was started, and just a few years later, sulfapyridine, the first anti-pneumococcal sulfonamide [6], appeared in practical medicine. These antimicrobial innovations made it possible to slightly improve the results of AP treatment, which led to a shift in the focus in the general complex of medical care for these patients from pathogenetic methods to etiotropic agents, laying the psychological basis for future overestimated estimates of antibacterial therapy.

These etiotropic transformations in the treatment of patients with AP almost coincided with the discovery of penicillin and only slightly outstripped its clinical approbation. By the time the first results of the use of antibiotics were obtained, there were psychological prerequisites for the expectation of promising opportunities from a new therapeutic direction in the face of etiotropic therapy. The resulting enthusiasm for the successful use of antibiotics has for many years determined the strategic approach to the treatment of patients with acute nonspecific inflammation of the lungs (ANSIL), reducing it only to antimicrobials. The sense of far-reaching prospects that appeared after the first successes of antibacterial therapy were transformed for a long period into the therapeutic principle of “antibiotics alone”. However, interference with the standard state of the microflora accompanying us and a prolonged violation of its balance with the help of drug exposure could not continue without a trace and indefinitely. Therefore, over the years, the number of patients with AP began to grow, in other nonspecific agents began to perform the role of the leading pathogen in addition to pneumococcus, and the above principle of the etiotropic approach, despite the development and use of more advanced drugs, increasingly required the use of additional means of assistance.

Long-term attention to the antimicrobial treatment of patients with AP gradually formed the idea of its causative agent as the main factor in the occurrence and development of the disease. This concept of the disease began to dominate professional views on the problem, and many generations of future medical professionals were trained on its basis. The successes of physiology and pathophysiology in lung research did not coincide and even contradicted the dominant etiological interpretations of AP, remaining unclaimed in solving this problem. Therefore, when the need for detailed diagnosis and comprehensive treatment of patients with complications of AP began to grow, the main guideline remained the causative agent of the disease and not a violation of the functional uniqueness of the affected organ. As a result of the application of a single principle when choosing additional means of medical care, inflammation in the lungs ceased to differ from other localizations of acute inflammatory processes. The inflammatory reaction of the pulmonary vessels, which, according to their circulatory indicators, are the complete opposite and mirror image of the vessels of the large circulatory circle, did not entail logical differences during intensive and anti-shock measures, depending on the localization of the lesion.

The vessels of the small circulatory circle are equipped with baroreceptors that regulate the parameters of the systemic blood flow, automatically equalizing the necessary proportions between the two halves of the vascular system of the body in case of critical situations [5]. With the aggressive development of inflammation in the lung tissue, the vessels of the small circulatory circle react to this powerful stimulus with an uncoordinated generalized spasm, creating a problem for the general blood flow at this level, which begins to manifest itself as a picture of a peculiar form of pulmonogenic shock [7-10]. The use of pathogenetically justified procedures during this period, such as general cooling of the patient’s body, cupping therapy, or cervical vagosympathetic blockade, eliminates this uncoordinated spasm and relieves blockage of pulmonary vessels. The immediate effect of such effects was proved by a comparative recording of rheopulmonograms [7,8].

If we now imagine what kind of medical care a patient with AP receives in case of hospitalization in a hospital, then we will be able to understand and explain the gradual deterioration of those statistics that reflect the results of treatment of this category of patients. Such patients are provided with access to the venous bed during hospitalization as a prerequisite in case of acute need for intravenous administration of medications. However, often this procedure continues with the drip introduction of liquids (to maintain water balance?), even in the apparent absence of indications for this. The presence

of a focus of acute inflammation in the path of intravenously administered solutions, in which intensive processes of edema and infiltration occur, causes their stimulation regardless of the speed and volume of the injected solutions. At the same time, the earlier intravenous infusions are started, the more negative consequences they can lead to [7,8]. The latter circumstance is associated with the phase of inflammation when the phenomena of tissue edema initially progress especially intensively.

Acute inflammation triggers a cascade of its own mechanisms, which in the case of aggressive development are characterized by poorly compensated indomitable character. The clinical manifestation of the disease during this period is determined by a violation of the functional features of the affected organ, while the causative agent of inflammation does not play a decisive role. The latter circumstance explains the fact that long-term efforts to differentiate the diagnosis of AP depending on its etiology have not brought decent results not only in bacterial forms of processes but also in attempts to separate bacterial inflammation from viral [11-13].

Despite the obvious inconsistencies between assumptions and real facts, the etiology of AP continues to be considered the main cause of the progression of the inflammatory process in the lung tissue, and the beginning of modern treatment is still focused on the expectation of an effect after-the introduction of etiotropic agents [14]. At this time, the more severe the patient's condition is assessed, the more intensive intravenous infusions are carried out, the volume of which increases to bolus [15,16]. An increase in venous return and additional overload of the pulmonary vessels leads to the development of pulmonogenic [7,8], and not septic, as it is considered today, shock. The development of this type of shock in AP is significantly accelerated by such intensive therapy. The presented mechanism is an explanation of the negative results of microbiological blood tests in patients with AP in comparison with septic complications in other localities of inflammation [17,18], as well as published revelations about the deterioration of patients' condition during intensive treatment [19-21] and the reason for pre-prepared recommendations on the use of vasopressors [15,16]. The fact of the negative effect of intravenous infusions on the dynamics of AP was proved not only by clinical observations but also by the results of experimental studies [7,8].

The unpredictability of the results of modern medical care for patients with AP has led to frequent statements about a high risk of death during their hospitalization compared with other nosologies and a low possibility of such a prognosis [22-24]. On the other hand, in recent years there has been a whole series of publications about the psychological impact on medical personnel of those ineffective efforts that are made during the treatment of patients with AP. The feeling of disappointment in the existing therapeutic possibilities, confusion, and despondency that has become observed among professionals turn into a kind of confession when a psychologist working in intensive care wards describes the occurrence of hopeless situations [25].

However, there is undoubtedly a way out of this general and seemingly hopeless situation, but for this it is necessary, first of all, to critically rethink and adjust views on the essence of the problem under discussion. Attempts to find any justification for the collapse of hopes placed on modern medicine in the treatment of patients with ANSIL look more like repentance. Such grateful statements of facts, devoid of detailed scientific analysis, will not bring us one iota closer to getting rid of those misconceptions in assessing the causes of the problem under discussion that have formed and been cultivated over the past decades. Every year there is less and less time for unhurried discussions of this problem, and the next surprise that nature is ready to present to us in this area is able, according to the observed patterns, to surpass the consequences of the SARS-CoV-2 pandemic that has just passed [26]. And has this threat passed if the population in different parts of the globe continues to suffer from pneumonia caused by COVID-19 [27]?

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