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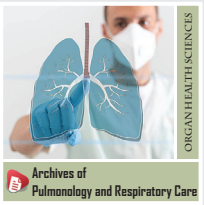

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Opinion

Principles of First Aid and Emergency Care and their Optimal Application in the Treatment of Community-Acquired Pneumonia

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Abstract

As recent experience, including unique data from the SARS-CoV-2 pandemic, has shown, treatment of patients with community-acquired pneumonia is focused on those with severe disease. The rapid development of inflammation in lung tissue clearly demonstrates the futility of etiotropic medications, which lack the properties of effective first aid in such conditions. The lack of correlation between pneumonia severity and the type of pathogen, as well as the persistence of underlying disease mechanisms regardless of the etiology, necessitate the earliest possible application of pathogenetic treatment to ensure positive results and prevent complications.

Abbreviations

ANSIL: Acute Nonspecific Inflammation in the Lung; AP: Acute Pneumonia; ARDS: Acute Respiratory Distress Syndrome; CAP: Community-Acquired Pneumonia; COVID-19: Coronavirus Pneumonia; ICU: Intensive Care Unit; SARS-CoV-2: Coronavirus Pandemic 2; sCAP: Severe Community-Acquired Pneumonia; WHO: World Health Organization

Introduction

Acute nonspecific inflammation in the lung (ANSIL) or acute pneumonia (AP) remains a serious disease, consistently ranking among the leading causes of hospitalization and mortality [1,2]. According to generally accepted classifications, this definition of AP primarily refers to community-acquired pneumonia (CAP), which encompasses the main and most numerous group of patients. However, this general assessment of CAP creates a rather strange and contradictory impression against the backdrop of statistics from recent decades. For example, it is noted that the overall incidence of CAP is decreasing [3], and most cases are easily treated on an outpatient basis with antibiotics alone [4]. At the same time, despite the decrease in the total number of cases, the number of deaths has increased [3], and mortality among such patients in intensive care units (ICUs) reaches 40% [5,6].

Some clarification of the reasons for these statistical discrepancies can be obtained by considering the fact that for several decades, severe community-acquired pneumonia (sCAP) has been singled out and analyzed as a separate group of observations [7-9]. At first glance, this approach seems justified and reasonable, since it is in this category of patients that the disease progresses with the development of critical complications and fatal outcomes, and the clinical manifestations of the severity of the patients' condition are quite obvious, which allows for observation and study of the identified deviations. However, from a methodological point of view, this approach cannot be considered correct, since both mild and severe forms of the disease constitute the category of CAP, which allows for manipulation of statistical indicators with such a division. Moreover, it is known that a certain percentage of patients with CAP are hospitalized at a certain stage of outpatient treatment due to worsening of their condition and follow the same course as patients with sCAP.

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Discussion

For many years, pathogen virulence was considered the most significant reason for differences in the development and course of community-acquired pneumonia, although during this long period, no one was able to present convincing and objective arguments in support of this assumption. In recent years, antibiotic resistance has been added to this explanation for treatment failure. Microbial resistance, which appeared soon after the onset of the antibiotic era, has only recently begun to be mentioned as an explanation for low treatment efficacy [10]. Moreover, such statements are primarily declarative in nature, devoid of objective argumentation, and the few materials with the results of bacteriological studies show that resistant microflora is detected in patients with community-acquired pneumonia in only 1-2% of cases, while the overall frequency of positive cultures does not exceed half of observations [11-13]. Numerous attempts to develop diagnostic criteria for community-acquired pneumonia depending on its etiology have been unsuccessful, and the inability to differentiate bacterial and viral forms of inflammation put an end to this issue, demonstrating the inconsistency of this idea [14-16].

The emergence of the SARS-CoV-2 pandemic has been a very instructive and revealing lesson for modern medicine. For at least two decades, nature had been demonstrating the prerequisites and subtle warnings of a future viral invasion, during which the principles and approaches to treating CAP did not undergo fundamental changes. The unpreparedness to provide adequate and effective care to a large number of patients with coronavirus pneumonia became evident not only to specialists but also to the general public. Even in such a tense situation, nothing more effective than antibiotics was offered [17-19]. However, the most revealing result of the pandemic for specialists was the observation of the spread of the same strain of coronavirus with completely different consequences [20-22]. It would seem that such a "natural experiment" should have significantly changed the understanding of the etiology and pathogenesis of the disease, altering the direction and focus of medical care.

Unfortunately, as reality has shown, no significant changes have occurred in the strategy for managing the disease and the tactics of its treatment that could influence the development and dynamics of CAP. Yes, many researchers and clinicians are now paying attention to the reasons for differences in individual responses to an equivalent stimulus [23,24] and are studying them. But this feature has been known for a long time and is one of the inherent conditions of various types of acute non-specific inflammation, isn't it? While variations in the body's protective response are being studied, the treatment of patients with CAP has not changed at all, repeating the principles that were applied before the pandemic. Only now has the chorus of voices calling for the development and release of new antibiotics intensified. Numerous analytical and research reports are devoted to new panels for bacteriological diagnostics [25-27], as if their introduction and application will change the natural patterns of development and course of CAP.

The modern understanding of community-acquired pneumonia, according to which, since the development of microbiology, the main causative agent of the disease is considered to be a pathogenic microorganism, and antibiotics have remained the main method of treatment for more than 80 years, demonstrates how firmly and unshakably the old and long-refuted concept of this problem has taken root [28]. The priority given to antibiotics as the main method of treatment hinders the understanding that antimicrobial therapy cannot replace pathogenetically justified first-line methods and emergency care for patients with severe community-acquired pneumonia. Numerous attempts to prove differences in the development and course of acute pneumonia depending on the type of pathogen have not yielded the expected results. Acute inflammation of the lung tissue, regardless of etiology, continues to demonstrate a stable and specific clinical picture that is difficult to confuse with any other disease, emphasizing the leading importance of pathogenetic mechanisms for external manifestations. And it is these signs that form the basis of diagnosis and subsequent monitoring of patients.

Currently, the main method of treating patients with severe community-acquired pneumonia remains the administration of antibiotics. The significant increase in viral forms of the disease over the past decades has not been reflected in the quality of medical care or the development of effective antiviral drugs. This is partly due to the fact that such medications need to be administered in the first couple of days of the illness, before the viruses penetrate the cells. At the same time, the appearance of the first symptoms indicates that the viruses have already reached their target. This situation was confirmed during the SARS-CoV-2 pandemic, when a large-scale collaborative study conducted under the auspices of the WHO did not reveal any noticeable benefit from any of the tested drugs [29]. At the same time, this period showed that medicine, left without its usual etiotropic treatment methods in the face of a large number of coronavirus pneumonias, demonstrated its confusion and adherence to stereotypes, continuing to use antibiotics in almost all cases [17-19].

A significant drawback of the widespread use of antibiotics in community-acquired pneumonia is not only their prescription without indication, but also the fact that this therapy continues to be used as a first-line and emergency treatment. Numerous clinical studies have been conducted with the earliest possible, hourly administration of these drugs, but it has not been possible to prove that earlier administration of these medications yields better results. Currently, when prescribing antibiotics, treating physicians continue to wait 48-72 hours to assess their effect [30,31]. That is, after diagnosis and empirical initiation of antibiotic therapy, the doctor must wait 2-3 days for the treatment results. In mild cases of the disease, it has already been established that antimicrobial drugs are not necessary for a favorable outcome. This last statement is supported by the recovery of many patients from pneumonia caused by COVID-19 without any specific therapy [20-22], as well as the experience of outpatient treatment of patients with community-acquired pneumonia without antibiotics [32,33]. In such cases, the disease does not have a rapid onset and

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progression, and the body has enough time and resources to cope with the problem.

However, the number of severe cases of CAP is increasing every year, characterized by a persistent progression of the pathological process. Since the causative agent is still considered the reason for the severe course of the disease, antimicrobial drugs are often changed or added, but this does not always yield the desired result. The natural and rapid sequence of inflammatory process development, which antibiotics do not directly affect, requires additional support, the modern methods of which are of an auxiliary or palliative nature. Oxygen administration to the patient, which is given so much importance, usually does not lead to significant changes in the development of the disease and the patient's condition. In addition to antibiotics, the second first-line treatment in such situations is general medical procedures, such as fluid therapy, especially when signs of complications appear [34-36]. Moreover, the more severe the patient's condition and the more significant the shifts in functional indicators, the more intensive administration of solutions is recommended. This period of the disease is literally critical for many patients with CAP.

One of the fatal misconceptions of modern medicine in the field of pulmonology is the obvious, yet unexplained, fact of ignoring the fundamental pathogenetic differences in the development of acute inflammatory processes in the periphery (more precisely, in the systemic circulation) and in the lung tissue. The vascular system of the lungs, which constitutes half of the total blood circulation in the body and is designed to maintain parity between systemic and pulmonary circulation, thanks to its autonomous regulatory system [37,38], has parameters directly opposite to those of the systemic circulation [39,40]. The inflammatory process in the periphery affects individual branches of the systemic bloodstream, which does not affect the overall circulation until serious disturbances occur, caused by fluid loss or complications such as generalization of the infection.

Classic acute inflammation, regardless of its location, begins with a vascular reaction, which is accompanied by increased blood flow in the affected area, a sharp increase in the permeability of the vascular walls, edema, and infiltration of the surrounding tissues. However, in the case of an inflammatory process in the lung tissue, the mechanisms of its development follow a different, unique scenario of subsequent events. The reaction of baroreceptors in the pulmonary vessels to this change in blood flow occurs immediately and in a standard manner, since the vital balance between the function of the two halves of the heart and the two circulatory systems depends on it.

In patients with a mild form of the disease, the body reacts relatively slowly to changes in the pulmonary vessels, giving it enough time to adequately adapt to the unexpected new conditions. In such cases, obvious signs of respiratory failure may be absent, and the result of the delayed action of the so-called unloading reflex [37] is, for example, a change in skin color. This category includes patients who are treated on an outpatient basis, using "only antibiotics", and recently even without them [32,33].

The situation takes on a completely different character in the case of aggressive development of the inflammatory process in the lungs, turning into an uncontrolled chain of events with rapid deterioration of the patients' condition. The reason for this transformation, strangely enough, is the autonomous protective reaction of the pulmonary vessels to a powerful irritant, which is the rapidly progressing focus of inflammation. Arbitrary suppression of this reflex is impossible, but, on the other hand, the body receives a constant alarm signal about the danger of increasing edema and infiltration of lung tissue. In this situation, the protective reflex, developing autonomously and continuing to perform its role, goes beyond reasonable adaptation, causing generalized spasm of the vessels of the pulmonary circulation [41-43]. This latter mechanism completely changes the conditions and parameters of central hemodynamics. Blood transit through the pulmonary vessels is sharply reduced, disrupting the ratio between blood flow and ventilation and creating a relative excess of venous return.

Impaired central hemodynamics leads to generalized microcirculatory dysfunction, impairing gas exchange at all levels, from the lungs to peripheral tissues. This latter factor, along with decreased gas exchange at the pulmonary level (changes in the ventilation-to-blood flow ratio), plays a significant role in the development of hypoxemia. At this stage, the direction and quality of first aid determine the further course of the disease and, quite literally, the patient's fate. Simple oxygen inhalation, administered to all patients without exception, has no significant impact on the severity of the disease, as this procedure does not address the underlying cause of the disorder and is purely palliative. However, eliminating the reflex action of the inflammatory focus in the lungs and promoting the redistribution of circulating blood through such ancient methods as cupping therapy or short-term whole-body cooling provides an almost immediate positive effect, as confirmed by objective tests [41].

As a result of widespread pulmonary vasospasm, pressure in the pulmonary circulation increases, triggering an unloading reflex that should influence systemic circulation through hypotension and blood deposition, reducing the relative volume of venous return. However, in the context of raging inflammation, this reflex fails to realize its potential. Furthermore, a tendency toward hypotension is now recognized as one of the main manifestations of sepsis, although in patients with CAP, this symptom reflects excessive compensation. Treatment of sepsis is now standardized for all patients, regardless of the location of the primary lesion. Bolus fluid therapy [34-36] plays a key role in this approach, becoming essential in patients with sCAP from the earliest stages of the disease.

At this stage of the analytical description of pathogenesis, many can already understand the reasons for the rapid deterioration of patients with severe community-acquired

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pneumonia during and despite treatment. Treatment outcomes for hospitalized patients with community-acquired pneumonia show that presumed sepsis or septic shock develops within the first two days in 20%-25% of cases [35,44]. Intravenous bolus fluid administration increases venous outflow, disrupting the body's compensatory attempts to slow or stop this process. Therefore, it is not surprising that treatment outcomes in this group of patients, given current care principles, leave much to be desired.

In conclusion, it is important to note several key features necessary for the successful treatment of CAP. First, sCAP is caused not so much by a respiratory catastrophe as by a circulatory catastrophe [41]. Significant changes in pulmonary blood flow inevitably trigger a restructuring of the systemic circulation, which is necessary to maintain vital balance and synchrony between the two halves of the circulatory system. A significant transformation of the peripheral circulation is entirely consistent with shock. However, this shock reaction is pulmonogenic, not septic in origin, as is currently believed.

Secondly, the functional impairments accompanying CAP have a completely different mechanism of occurrence and development compared to similar disorders associated with peripheral inflammatory processes. In this regard, it is important to consider the fact that changes in systemic blood flow parameters in patients with CAP are secondary, compensatory in nature. However, they are currently assessed using the same framework as other diseases with fundamentally different pathogenesis [45]. Given that CAP is statistically responsible for approximately half of sepsis and septic shock cases [46], one can imagine the number of patients with pseudosepsis and the amount of work and resources wasted on false diagnoses and inappropriate treatment.

Thirdly, the degree of functional impairment in CAP does not depend on the etiology of the disease, nor do the main clinical signs, which remain unchanged regardless of the pathogen. Therefore, etiotropic drugs in general and antibiotics in particular, which can neutralize the microbiological factor but do not directly affect the mechanisms of the process, cannot serve as the primary, much less the only, treatment.

Fourth, the reflex spasm of the entire pulmonary vascular system, which underlies systemic circulatory disorders, is a reflection of an uncontrolled and autonomously acting compensatory response. The absence or prolonged delay in applying methods that eliminate the obstruction to general pulmonary blood flow and divert some blood to the periphery increases the risk of complications such as ARDS and multiple organ failure.

Fifth, routine emergency care, such as intravenous fluid resuscitation in patients with CAP, has a profoundly negative impact, stimulating local and systemic dysfunction. These fluids, especially in the early stages and in bolus form, increase the area of inflammation in the lung tissue, contribute to the development of pleural effusion, and require the use of vasopressors.

Conclusion

Therefore, to achieve real progress in the treatment of acute inflammatory lung diseases, we must radically change our understanding of their nature, taking into account the unique characteristics and key role of their pathogenesis, which is fundamentally different from the mechanisms of inflammatory processes in other locations. Early and prompt application of treatments based on a pathogenetic approach can slow disease progression, help the body overcome sudden catastrophe, and prevent the development of potential complications.

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