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Editorial

Take Antibiotics Off the Pedestal

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Currently, medical professionals worldwide are expressing growing concern regarding the observed trend of declining treatment efficacy for inflammatory processes of nonspecific etiology. Lower respiratory tract infections occupy a distinct position within the comparative spectrum of such diseases, characterized by the lowest survival rate, 83.3%, relative to all other patient categories [1]. The explanation for this phenomenon, which has gained widespread acceptance within the medical community and remains entirely uncontroversial, lies in the increasing resistance of microbial flora: a problem that the WHO declared a global crisis several years ago [2].

Given the prevailing circumstances and the reasons behind the deterioration of treatment outcomes, the increasingly frequent proposals and calls for the development and release of new, more advanced generations of antimicrobial agents appear, at first glance, to be entirely logical [3-7]. However, this holds true only at first glance, as a critical analysis of publicly available information reveals this problem in a completely different light. Therefore, let us not rush to definitive conclusions, and instead, let us evaluate the role and significance of the materials comprising the vast body of data accumulated throughout the entire "antibiotic era".

When A. Fleming discovered the antimicrobial activity of penicillin in 1929 [8], nearly a decade and a half elapsed before the drug was refined, studied, and introduced into clinical practice [9]. Nevertheless, during this period, forms of etiotropic therapy were already being employed in patients with acute pneumonia, specifically, antipneumococcal serum and sulfonamides [10]. The use of these etiotropic agents demonstrated improved treatment outcomes, thereby giving rise to the so-called "microbial concept" of acute pneumonia [10]. On the one hand, this conceptual model of the disease identified the microbial factor as the primary cause of inflammation; on the other, it positioned antimicrobial agents as the principal means of providing care to such patients.

This limited yet positive experience with the etiotropic treatment of AP, accumulated immediately prior to the introduction of antibiotics into clinical practice, exerted a peculiar "sensitizing" influence on professional medical thinking. Consequently, when the first truly phenomenal results emerged, demonstrating the efficacy of penicillin against a "pristine" microbial flora, the aforementioned concept gradually evolved into a guiding ideology. Thus, at the very dawn of the antibiotic era, a fundamental error was committed in interpreting the capabilities of this new therapeutic modality, an error that has not only persisted to this day but remains an immutable postulate of modern strategies for treating AP.

The essence of this misconception lies in overlooking the fact that any form of acute inflammation manifests through the five classic signs described by Celsus and Galen, signs validated by two millennia of clinical experience. These signs constitute the foundation for diagnosing inflammatory diseases, as it is precisely their specific manifestations, varying according to the location and type of the

affected structures, that primarily determine the severity and nature of the accompanying functional impairments. However, antibiotics, capable only of suppressing specific strains of pathogens, lack the capacity to exert other effects, including the direct correction of these functional impairments. This inherent limitation leads to a protracted waiting period for clinical results, a delay that, from the perspective of emergency medical care, is simply unacceptable.

The negative and relatively rapid impact of antibiotics on bacterial flora, manifesting as the development of microbial resistance (MR), was established as early as the preclinical stage of their study [11,12]. In his Nobel lecture, A. Fleming specifically underscored the danger inherent in the emergence and spread of MR resulting from the unjustifiably widespread use of these drugs [13]. However, as the history of antibiotic therapy demonstrates, these warnings regarding adverse consequences failed to influence the clinical strategies of medical professionals, whose primary focus remained exclusively on preserving and maintaining initial therapeutic outcomes. Moreover, it was precisely during this period that antibiotics began to be employed not for their direct indications in treating diseases, but rather for the prophylaxis of inflammatory processes in an attempt to eliminate the most active strains [14,15].

Confronted with the continuous impact of antimicrobial agents, nature began to mobilize its own resources in an effort to mitigate the consequences of aggressive external human interference in the established ecological equilibrium. This adaptation manifested itself in shifts within the etiological structure of diseases, specifically, in the replacement of bacteria exhibiting reduced sensitivity to antibiotics by microorganisms demonstrating higher levels of resistance. In clinical practice, this phenomenon became evident with the advent of widespread antibiotic therapy, when the pneumococcus, previously the invariably dominant pathogen in the etiology of AP, was displaced by the *Staphylococcus*. Indeed, in the span of just a decade and a half, by the early 1960s, the latter had assumed a dominant position among the causative agents of AP, particularly in children [16].

The medical community responded to such etiological shifts not through a critical analysis of their root causes, but rather by seeking new approaches aimed at preserving the original efficacy of existing therapeutic methods. These challenges and needs served as a powerful impetus for the development and introduction of new, more advanced antimicrobial agents, the vast majority of which had already been discovered by the early 1970s [17]. While the process of pathogen succession continued unabated, the development of new therapeutic agents slowed significantly. Remaining true to its established principles and objectives, medicine shifted its focus toward identifying methods for the early diagnosis of the microbiological factors underlying acute inflammatory lung diseases. This line of research remains highly relevant to this day, striving to pave the way for the prompt and targeted administration of antibiotics [6,7,18,19].

Throughout the entire "antibiotic era," the emergence and spread of MR have been observed; nevertheless, awareness of this phenomenon was not accompanied by any systemic measures aimed at mitigating the severity of this problem. This circumstance was attributable to the fact that, over the many decades of antibiotic use, pneumonia was predominantly bacterial in nature. During this period, numerous generations of physicians, professionals whose training was oriented toward viewing this specific therapeutic approach, antibiotic therapy, as the primary and most logically sound method of treatment, came and went. Concurrently, such training reinforced and entrenched a conception of pneumonia rooted in the "microbial concept" of disease.

Consequently, when a significant rise in the incidence of viral forms of pneumonia began to be noted at the turn of the millennium, specialists proved unable to adequately adapt their conceptual frameworks to the changing epidemiological landscape of these diseases. The established dominance of an exclusively antibacterial approach, based on relying solely on antibiotics to resolve this problem, served as a serious impediment to a critical re-evaluation and radical revision of the fundamental ideology surrounding this disease. Thus, for instance, the lessons learned from two major coronavirus epidemics (SARS and MERS) exerted absolutely no influence on either professional perspectives or treatment protocols for pneumonia. As a result, when the medical community confronted the SARS-CoV-2 pandemic, its therapeutic arsenal contained no means of combating this viral aggression other than antibiotics, the utilization rate of which ultimately reached nearly 100 percent [20-22].

During the pandemic, relying predominantly on auxiliary and supportive treatment methods while observing their low efficacy, the WHO resorted to an explanation for these failures that, from the standpoint of the prevailing paradigm, appeared entirely logical: after eight decades of observation, it declared MR a global calamity [23]. This interpretation of treatment failures was enthusiastically embraced by specialists as a working hypothesis, which subsequently came to be actively disseminated in publications on the subject. However, no one draws attention to the utter untenability of such an explanation.

First, there is no evidence that resistant microorganisms have acquired increased virulence. On the contrary, their recently acquired ability to successfully withstand antimicrobial agents poses difficulties for antibiotic therapy when the latter is employed as the primary method of treatment.

Secondly, the prevalence of MR has reached a level at which such strains are becoming habitual symbionts of the human body. For instance, MRSA has become endemic within the U.S. population and is detected in healthy individuals in 2% of cases [24]. Among workers in certain professions, this strain was detected in a latent form in 6-10% of cases as recently as a couple of decades ago [25-27]. In this regard, it is highly telling that the vast majority of assertions regarding the dangers of MR and its negative impact on treatment outcomes for AP are not substantiated by specific statistical data reflecting the contemporary etiology of this disease. At the same time, this MRSA strain is isolated in patients with AP in no more than 2% of cases [28,29].

Finally, modern statistical data regarding the etiology of AP indicate that in more than half of all cases, the causative agent remains unidentified; however, among cases with positive test results, viruses predominate [30-34]. In light of these figures, bacterial forms of inflammation account for only a negligible fraction of cases and among these, an even smaller number are associated with resistant microflora. If, in this context, it is indeed appropriate to speak of a "global catastrophe," then today this characterization applies far more to viruses than to bacteria.

One could continue the critical analysis of the observed distortions in professional conceptions regarding the fundamental nature of AP, that represent further links in this unified and pressing problem domain. Illustrative examples include the interpretation of the causes of systemic disturbances in this category of patients, the pathogenesis of which, despite being unique, is erroneously conflated with the mechanisms underlying all other inflammatory processes, as well as attempts to apply general therapeutic approaches, such as fluid resuscitation, which, in the context of AP, produces the exact opposite effect, or the classification of the disease's complications as distinct nosological entities. However, within the scope of the present context, the issue under discussion is of a more specific nature, and the material presented above is entirely sufficient to draw substantive conclusions.

The modern concept of AP, which defines the strategy for combating this disease, emerged and took its final shape in the mid-20th century. Antibiotics played a decisive role in solidifying this concept and ensuring its universal acceptance. However, in the time that has elapsed since then, the fundamental underpinnings and original logic of this doctrine have never undergone revision, necessitated both by the changing etiology of pneumonia and by the emergence of irrefutable evidence contradicting the initial worldview. By now, this virtually unchanged concept has become ossified, transforming into an indisputable dogma focused exclusively on the efficacy of antibiotic use. Remarkably, not a single specialist or expert working in this field has expressed even the slightest bewilderment regarding the fact that only now, approximately eight decades into the "antibiotic era", has microbial resistance been declared a global crisis, despite the fact that the efficacy of antimicrobial agents has been steadily and consistently declining throughout this entire period. Now, having failed to establish the early identification of AP pathogens while simultaneously continuing the empirical use of antibiotics, specialists have suddenly turned to a new "trendy" practice, dubbed "de-escalation", under which treatment courses may be significantly shortened or completely discontinued in cases where the disease follows a favorable clinical trajectory [35-37].

In light of the experience gained in treating patients with COVID-19-associated pneumonia, where positive outcomes were achieved even in the absence of specific etiotropic agents, the currently widespread practice of "de-escalation" should be regarded as a fiction and a form of self-deception. Etiotropic drugs, which exert no influence on the mechanisms of inflammation, fundamentally cannot be considered a primary or life-sustaining therapeutic modality, particularly in cases where the inflammatory process manifests in an aggressive form. This assertion holds all the more true given that it is precisely these mechanisms that determine both the dynamics of clinical manifestations and the overall severity of the disease. And although attempts to identify effective antiviral agents have thus far proven fruitless, this serves as no justification either for the indiscriminate use of antibiotics or, even less so, for the introduction of new drugs without a comprehensive assessment of the consequences derived from the clinical experience already accumulated.

A detailed analysis of over 80 years of antibiotic therapy reveals that viewing microbial resistance as the sole negative consequence of using these drugs is a clear misconception, and claims regarding the extreme danger posed by such strains are vastly exaggerated and lack an evidentiary basis. In reality, the most serious consequence of antimicrobial therapy is its negative "didactic" impact, specifically, the shaping of a deeply ingrained professional mindset that regards antibiotics as the primary and indispensable therapeutic tool. While rethinking the issue of antibiotic resistance is essential for developing effective solutions, the shift in disease etiology, accompanied by a significant narrowing of indications for antibiotic use, is often overlooked as a consequence of this therapy. Yet, it is precisely this phenomenon that has led to these drugs being displaced from the first-line treatment arsenal. The changing microbiological factors underlying pulmonary inflammatory processes must be recognized as a real and pivotal factor, one that underscores the merely adjunctive role of etiotropic agents.

MR, currently cited as the primary cause of treatment failure in patients with AP, is a natural consequence of antibiotic use, reflecting the ability of microflora to develop their own protective mechanisms. The emergence of such protective traits does not imply that these strains have become more virulent or aggressive. Claims regarding the extreme danger of MR stem solely from the medical community's deeply ingrained reverence for antibiotics, that overlooks clear inconsistencies and contradictions concerning biological principles and objective facts. A comprehensive analysis of management strategies for AP, as necessitated by the current situation, inevitably leads us back to the aforementioned "didactic" side effect of antimicrobial therapy. Ultimately, it is high time to define the true role of antibiotics in treating acute pneumonia and to strip them of their status as indispensable, life-saving agents. Moreover, attempting to find a genuine solution to the problem by focusing exclusively on the disease's etiology amounts to outright self-deception.

Over time, as the etiology of the disease has shifted, nature itself has naturally limited the role of antibiotics in treating AP, relegating them to a supportive function. Unfortunately, the medical community, having pinned high hopes on this treatment method during its initial success, yet failing to fully grasp the drugs' underlying mechanisms of action and long-term consequences, continues to view antibiotics as the primary means of combating AP without subjecting this practice to comprehensive critical analysis. The changing clinical course of AP clearly demonstrates the inadequacy of this approach, and recent events underscore the urgent need for a radical reassessment of established views on the disease. It is time to move away from the prevailing theory of AP pathogenesis and treatment, which relies excessively on antibiotic efficacy, and assign antibiotics their proper supportive role, leaving behind unfounded expectations regarding the outcomes of such therapy.

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Conflict of Interest

The author states that he has no conflict of interest.

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