IBEROAMERICAN JOURNAL OF MEDICINE
iberoamericanjm
Journal homepage: www.iberoamericanjm.tk

Editorial Comment

Commentary to article: Esteban-Zubero E, García-Muro C, Alatorre-Jiménez MA, Marín-Medina A, López-García CA, Youssef A, Villeda-González R. Management of Community acquired pneumonia in the Emergency Room. Iberoam J Med. 2019;1(1):3-15

Igor Klepikov[a,*]

[a]*Retired, Russia*

Dear colleagues,

The discovery of antibiotics and the initial triumph of their use started a trend in the treatment of acute pneumonia mainly by suppressing its pathogens. The prevalence of this doctrine and the long definition of therapeutic efforts as "antibiotics alone" have led to a gradual distortion of views on the nature of the disease, to the oblivion of its biological basis as an inflammatory process and the modern interpretation of its leading cause as infectious [1]. Reducing the effectiveness of antibiotics and the increasing number of antibiotic-resistant strains, the gradual deterioration of treatment outcomes and the persistence of high rates of complications and mortality are common characteristics of the current state of the problem. It would be logical to expect that such a reputation would automatically lead to a revision of the doctrine of disease. However, this long-overdue need is constantly being pushed back, and the appearance of new articles on this topic is more upsetting than encouraging.

The number of publications on acute pneumonia (community-acquired pneumonia-CAP) appears to have declined markedly in recent years. In my view, this trend is not the result of success in dealing with this problem, when the issues and topics for discussion are gradually exhausted. The whole situation in this section looks exactly the opposite: the number of unanswered questions is growing over the years, and the discussion of the problem remains focused mainly on the etiology of the disease. In the context of such a narrow aspect of the discussion, a significant part of the published articles, as a rule, is devoted to the identification and statistical analysis of pathogens, as well as a list of recommended antibiotics. In this regard, the publications on this problem are very similar in presentation and differ (depending on the time of research and geography) only by the statistics of pathogens.

The analyzed work is a review of articles published over the past one and a half to two decades. The authors of this review have done a great deal of analytical work, showing readers an alarming summary of CAP treatment outcomes and the direction of diagnostic and treatment efforts that are the main hopes. The main emphasis in the presentation of this review just repeats the above approach to the discussion, emphasizing the importance of determining the pathogen and ways to suppress it.

But in this situation, I would like to draw attention not to the style and principles of presentation of the results of the study, but to the material itself, which was taken as a basis. A distinctive feature of this review is that it included reports of the most severe group of patients with CAP who were referred to the emergency room. The latter fact serves as indirect evidence of the maximum possible examination of these patients. In this regard, a more thorough assessment of the reliability of the survey results and their significance for practical implementation is of interest, especially in such a priority test as the detection of the causative agent of the disease. This test is now seen as a basic prerequisite for determining further treatment

* *Corresponding author.*
E-mail address: igor.klepikov@yahoo.com

http://doi.org/10.5281/zenodo.3548152

outcomes. Of course, the main stake in this concept of the disease is calculated on the success of antibacterial therapy. Let's look at what criteria are currently based on specialists to assist patients with CAP.

First, according to summary statistics, the causative agent of the disease remains unknown in 40-60% of patients with CAP. These data mean that antibacterial therapy is conducted empirically and depends entirely on the experience and preferences of the attending physician. For such situations, clinical and epidemiological conditions are recommended (table 2), which are a possible assumption and cannot guarantee the accuracy of the bacteriological diagnosis. Thus, the microbiological diagnosis of CAP in most patients remains unidentified, and antibacterial therapy is carried out on the basis of assumptions. In this regard, it is appropriate to recall the historical beginning of the era of antibiotics, when 2-3 injections of penicillin (the only drug in that period) were often sufficient to obtain a noticeable clinical effect and no one complained about the shortcomings of bacteriology. Today, the use of drug combinations and long-term treatments are often ineffective.

Secondly, it is a pity that the authors of the review did not attach due importance to the nature of the material subjected to bacteriological research. Currently, material outside the area of inflammation is widely used, such as nose-mouth-pharynx smears or the detection of indirect signs of the presence of certain strains in the patient's body (the review mentions antigenuria). From the point of view of known facts, such methods of bacteriological diagnosis should also be considered approximate. For example, it is well known that healthy people can be carriers of opportunistic and antibiotic-resistant strains. However, the mere presence of a microbe in the body does not necessarily mean the development of a disease. Moreover, microorganisms from the list of the most frequent causative agents of CAP (including table.1) usually belong to representatives of the symbiotic microflora and any of them can be the causative agent, while others will be found outside the focus of inflammation.

The only way to accurately determine the causative agent of CAP can be attributed to the receipt of material directly from the zone of inflammation. But, this possibility appears only in the later stages of the disease in a relatively small group of patients in the case of purulent complications. However, the results of bacteriology in purulent complications cast doubt on the infectious concept of CAP, as in a number of patients, despite the development of pleural empyema, crops are sterile. In other words, inflammation progresses despite suppression of microflora [1].

If we now try to assess the practical benefits of the dominant desire by any means to determine the causative agent of CAP, then, from my point of view, it is purely declarative and does not have a decisive (as implied and asserted in modern publications) influence on the final results of treatment [2-5].

Finally, another section of the review deserves attention, which also follows from the infectious concept of the disease and is more indicative and recommendatory than a guide to the practical effects on the course of the inflammatory process in the lungs. We are talking not so much about prognostic factors, the value of which is mainly cognitive-consultative in nature, as about the criteria for the development of sepsis and shock states. These criteria are presented as a result of exposure to aggressive microflora and this presumed cause of disease severity is not questioned in the absence of objective evidence. In connection with the last remark, it is necessary to recall the fundamental principles of the origin and development of CAP, which can hardly be revised and rejected, and their impact on the dynamics of the disease will act regardless of our attitude to them.

CAP is a classic inflammatory process based on a vascular response with a regular sequence of stages. The intensity of such a reaction is an individual feature of the body and depends on its sensitization and the variant of the immune response. Among the long list of acute inflammatory diseases, CAP is the only process that is localized in the vessels of the small circle of blood circulation. The inseparable anatomical connection and the inverse dependence of blood flow in the small and large circulatory circles is the leading cause of the difference in the pathogenesis of CAP from other inflammatory processes, even in conditions of coinciding etiology. Therefore, shock, which is observed in aggressive forms of CAP, has its own unique mechanism, and its interpretation as septic is not confirmed by objective criteria in the examination of this group of patients [6-10].

The lack of direct confirmation of the cause of this severe complication leads researchers to look for suitable explanations. In terms of the prevailing ideas about the infectious nature of the disease, the criteria for sepsis (table. 3) there are such assumptions. At the same time, the frequency of the main confirmation of the septic nature of complications (positive blood culture) is not even given. The latter fact is not surprising, since in publications on this topic only rarely can be found mention of a low percentage of detection of bacteremia in the most severe patients with CAP [10].

Just as the position and angle of view change our ideas about the object in question, so the interpretation of the results of one study will differ depending on the ideas about the phenomenon being studied. I think it makes no sense to analyze the minor details of this article, which are a natural consequence of the modern CAP doctrine.

Despite the above comments, this article has one undoubtedly important quality. Having done a complex analytical work, the authors presented the final result of the main efforts of specialists in different parts of the world in recent years. These generalized comparisons allow us to note that the main reason for the unsuccessful treatment of patients with cap were before and remain at the present stage distorted ideas about the nature of the disease, which do not have a clear informative and objective confirmation. The discrepancy between the dominant assumptions and

the actual facts is becoming more pronounced every year. Therefore, the main conclusion of this review should be made first of all about the need to revise the doctrine of CAP [11-13].

I hope that the authors of the analyzed article will be able to correctly understand the meaning of my comments and in case of questions will be able to count on the answer and additional explanations.

1. REFERENCES

1. Esteban-Zubero E, García-Muro C, Alatorre-Jiménez MA, Marín-Medina A, López-García CA, Youssef A, Villeda-González R. Management of Community acquired pneumonia in the Emergency Room. Iberoam J Med. 2019;1(1):3-15.

2. Klepikov I . Acute Pneumonia in Children – Illness or Infection? - Open Letter to the Editorial Staff. J Infect Non Infect Dis 2017;3:22. doi: 10.24966/INID-8654/100022.

3. Klepikov I. Acute Pneumonia: What Infection We're Treating?. Res Pediatr Neonatol. 2018;3(2):226-7. 2018. doi: 10.31031/RPN.2018.03.000558.

4. Klepikov I. Associations and reality in the etiology of acute pneumonia. J Clin Rev Case Rep. 2019;4(2):1-5.

5. Klepikov I. Inflammation in the Lung Today-Hyperbolic Perceptions of Infection. J Neo Res Pedia Care 2019,2(1):180012.

6. Klepikov I. The Meaning of Pulmonary Reflexes in the Pathogenesis of Acute Pneumonia. Intern Med 7:232. doi: 10.4172/2165-8048.1000232.

7. Klepikov I . First Aid for Aggressive Forms of Acute Pneumonia. EC Pulmonology and Respiratory Medicine. 2018;7(2): 34-7.

8. Klepikov I. Shock in Acute Pneumonia and its Mechanism.EC Emergency Medicine and Critical Care. 2018;2(2):52-3.

9. Klepikov I. Acute Pneumonia is More Cardiovascular than Respiratory Disaster. J Emerg Med Care. 2018; 1(1):105.

10. Klepikov I. Mortality in Acute Pneumonia: Fatal Inevitlitabiy?.EC Anesthesia. 2019;5(4): 106-9.

11. Klepikov I. The Imperative of Modern Concepts of Acute Pneumonia in Comparison with Basic Medical Science. J Pulmon Respir Sci. 2018;3(2): 000122.

12. Klepikov I. Acute Pneumonia: Biological Rules and Laws require Attention and Respect. Journal of Respiratory Diseases. 2019;1(1):25-9. doi:10.14302/issn.2642-9241.jrd-19-2667.

13. Klepikov I. Do you really want to improve the results of treatment for acute pneumonia? J Clin Intensive Care Med. 2019; 4:023-027. doi: 10.29328/journal.jcicm.1001021.