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Review Article

AcuteInflammation Of The Lungs Problem Essence

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ABSTARCT

A systematic approach to the treatment of acute inflammatory processes of the lungs has been formed over the past decades under the influence of the use of antibiotics. Inspiration for practitioners and hope for patients came as a result of the phenomenal results of the first experience of penicillin therapy. This psychological effect and some euphoria were quite understandable and corresponded to the initial results, but the subsequent course of events with the growth of resistance of pathogens and a decrease in the effectiveness of antibiotics looks, to put it mildly, strange and illogical.

Keywords: Bacterial resistance; Microflora; Acute pneumonia

INTRODUCTION

After the widespread introduction of antibacterial therapy, there was a gradual decrease in its effectiveness, which continues throughout the entire period of its use. The process of adaptation of microflora to pharmacological aggression in comparison with the initial state has already achieved significant changes, resulting in a fairly representative group of antibiotic-resistant strains. The slow but steady loss of effectiveness of antibiotics required constant development and introduction of new forms of medicines. Today, the result of this continuous and intensive work is a long list of different antibiotics and their generation, which was the result of forced competition with growing bacterial resistance.

It is well known that microflora is one of the representatives of the living biological sphere and as a biological object is able to adapt to changes in environmental conditions. In this regard, the development of antibiotic resistance should be considered as its mandatory and inevitable mutation in response to an external threat. This interpretation of the consequences of antibacterial therapy does not allow us to consider its use as an infinitely harmless method of treatment, does it? This obvious circumstance is enough to cast doubt on the logic and validity of official medicine's perception of the role of antibiotics in the treatment of patients with acute pneumonia (AP), as the main, and often the only means.

However, the desire and intuitive desire to successfully continue using antibiotics prevailed over a rational scientific assessment of their role in the complex of General medical care for these patients. As a result, despite the reduced effectiveness of these drugs and an increase in the number of resistant strains of bacteria, the initial treatment of many patients was carried out on the principle of "antibiotics alone". Moreover, it has become quite common to use a single drug as the main treatment for a number of diseases that are not comparable in their clinical characteristics, but coincide in the identity and sensitivity of the suspected pathogens. No one in the medical scientific world has thoroughly questioned why antibiotics are gradually being elevated to the status of a panacea, while the number of patients for whom such treatment does not achieve its goal is becoming more and more every year.

This question is not only appropriate, but also necessary. After all, we are talking about drugs that initially have only one action antimicrobial. They do not show any other therapeutic effects. Therefore, the use of antibiotics as the main treatment can only have one goal to suppress the microbial pathogen, right? But nature has not changed its rules for the development of inflammation, has it? What result of antibacterial therapy can the patient's body get when the function of the affected organ, especially such an important one as the lungs, is disrupted as a result of the onset of an inflammatory reaction? The more severe and faster the inflammatory process develops, the more dramatic its functional consequences are and the more difficult it is for the body to cope with them without additional help. I think that this axiom does not require further explanation, since it reflects well-known biological features and patterns of development of inflammatory diseases.

Over the past few decades, persistent but unsuccessful attempts to restore the former effectiveness and universality of antibacterial therapy have continued. As a result of such efforts, the system of views on the nature of AP during this time has significantly shifted towards the leading role of its pathogens, and the disease itself has become treated as infectious, without any signs of the actual infectious process. Although most patients with this disease continued to be cured without an objective determination of its etiology, each failure was usually

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attributed to the lack of bacteriological tests for rapid diagnosis of the pathogen and the inability to conduct targeted antibacterial therapy.

Explaining the reasons for unsuccessful treatment is only a search for their interpretation, but patients with AP in such situations need real additional help. In accordance with the idea that the main cause of AP is its pathogen, the virulence of which determines the entire subsequent picture, the experience of additional assistance received in other inflammatory processes was automatically transferred to this group of patients. If we evaluate the complex of treatment methods used in patients with severe forms of AP, this list will differ little from the complex treatment, for example, in patients with diarrhea or peritonitis. The leading role among these additional methods is taken by standard infusion therapy for critical situations.

REVIEW AND LITERATURE

The General scheme of such complex treatment is widespread and generally accepted, but it does not significantly change the fate of severe patients with AP. The mortality rate in this group of patients admitted to intensive care units reaches 36%-50% [1-3]. But even in this case, explanations are given that contradict objective data. Septic shock is considered one of the main causes of death in AP, although positive bacteriological blood tests in such patients do not exceed 10% [4,5]. It is believed that the high percentage of sterile blood cultures is due to the pre-administration of antibiotics [6,7]. However, it remains completely unclear why such effective antibacterial therapy does not prevent the development of septic shock. Or maybe the AP-shock clinic has a different mechanism of development?

All acute inflammatory processes of non-specific etiology develop within the systemic circulation and only AP is the only disease with localization in the pool of the small circle of blood circulation. This feature of AP is fundamentally important for understanding the pathogenesis of the disease, and even the basics of medical education contain fundamental information about the close relationship and interdependence of two blood circles, as well as about the fundamental differences between the blood flow in these systems and the mechanisms for preserving such features.

Acute processes in the lungs change the blood flow conditions in them and affect the vascular baroreceptors. The result of this action may be changes in the parameters of General blood flow and heart activity, which are aimed at unloading and preserving the lungs. These mechanisms are mainly studied in pulmonary embolism, since in this case, critical situations up to sudden death occur very quickly [8,9].

The inflammatory process in AP, even in the case of a rapid and aggressive onset, does not have the suddenness that is observed in embolisms, so such sudden deaths do not occur at the very beginning of the disease. However, the constant involvement of small circle vessels in this process makes it inevitable to include the same unloading mechanisms, only the speed of such restructuring will not have such drastic changes. In this situation, the need for a thorough and objective study of the effect of infusion therapy on the hemodynamic parameters of such patients is clearly visible, the justification of which is currently based solely on assumptions and theoretical expectations. From my point of view, if this method of medical care, openly directed against the mechanisms of unloading the small circle of blood circulation, was first subjected to reasoned and objective scientific justification, it would not receive recommendations for its wide clinical application, especially in the initial stages of AP.

All of the above information does not have a novelty label. Moreover, more than 30 years ago, it was used as the basis for revising the concept of AP and found additional confirmation in the results of special studies and clinical trials, which allowed us to talk about the possibility of guaranteed prevention of complications of the disease [10]. However, the results of this work were initially presented only in Russian [11]. In addition, at that time, the situation in this field of medicine was more favorable and compared favorably with the current conditions. The gradual decline in the effectiveness of treatment of patients with AP and the absence of noticeable abrupt changes made it difficult for many researchers to see the causes of this problem from a different angle.

The situation has changed dramatically in recent months due to the onset of the pandemic. The search for the source of this universal tragedy occupies a prominent place in its research. However, regardless of the origin of the coronavirus, the ground for the manifestation of its character has been prepared over the past decades. And here it is necessary to return to bacterial forms of inflammation and antibiotics, despite the fact that we are talking about seemingly different problems.

Antimicrobial activity of antibiotics can be directed not only against pathogens of the disease. These drugs will surely have an impact on the symbiotic microflora. This action can change the accompanying microbial landscape. One example of such consequences can be the identification of atypical representatives and, in particular, antibiotic-resistant strains in healthy people. However, this is only the visible part of the iceberg.

The steady decline in the effectiveness of antibiotics has led to the need for their combined and long-term use. This type of antibacterial therapy cannot bypass the bacterial part of the body's microbiome. As we know, there are no voids in the biological world, and the partial loss of the accompanying microcosm must be filled in. In antimicrobial conditions, this role can be performed by viruses that make up a significant part of the microbiome. The features of this gradual transformation may eventually affect not only the composition of the microbiome, but also the immunological state of the body.

The last remark does not yet have direct and reasoned confirmations and is a postulate of the author. However, the events of the past two decades indirectly indicate the likelihood of such changes. Thus, if in previous years respiratory viral infections preceded bacterial inflammation in the lungs and created the necessary conditions for its development in recent years the role of true viral pneumonia has begun to increase [11-13]. A significant demonstration of the ongoing changes in

the etiology of AP was the SARS and MERS epidemics, which, unfortunately, did not lead to a revision of the strategy for the development of the disease.

Both during previous epidemics and during the current pandemic, official medicine does not have a reasonable and effective treatment package [14,15]. Currently, real care for patients with viral pneumonia is considered only in critical situations in the form of auxiliary resuscitation techniques, such as lung ventilation, positive end-expiratory pressure, extracorporeal membrane oxygenation [16]. The uncertainty of the situation and forecasts makes it necessary to calculate the availability of respiratory equipment and call for an increase in its production [17, 18]. However, current statistics do not allow us to expect significant improvement in results with increased availability of resuscitation equipment. While the overall mortality rate among hospitalized patients with coronavirus disease reaches 26% [16], it increases to almost 90% among patients who were on artificial ventilation [19]. And hopes for targeted assistance to such patients, continuing the etiotropic concept of AP, are postponed until the development and use of antiviral drugs [20, 21].

DISCUSSION

There is no doubt that the development and implementation of drugs to suppress viral infection will be an important part of helping such patients. But we can also say in advance that the successful neutralization of viruses will not help all patients equally, since the inflammatory changes in the tissues that have occurred by this time will continue to act and require additional help. And this is already a well-known fact on the example of many years of experience in the use of antibiotics, is not it?

Recently, publications have begun to appear, the authors of which Express concern about the possible consequences of a long period of antibiotic use. The very fact that such publications have appeared is an important beginning of the necessary discussion on this topic, although this area has required close attention for many years. In this context, it is truly surprising that these publications mainly appear in the media, and their authors were able to understand the growing problem, despite the lack of special medical education. At the same time, official medicine avoids radical discussions on this topic, and expert opinions until recently continue to consider antibacterial therapy as the "cornerstone" of the treatment of AP [15-23].

In the light of current events, the following important points should be noted. First, official medicine, unexpectedly accepting a huge number of patients with acute inflammation in the lungs, also unexpectedly lost the usual approaches to their treatment. It is not surprising that in this situation, many specialists in the care of patients with AP are seriously puzzled by the problem, since during the entire period of their work, attention was focused on the suspected pathogens of the disease. During the entire period of use of antibiotics in AP, the determination of the microbial factor in most patients remained an unsolved problem, and the choice of drugs was still conducted empirically [15-24]. However, a long-term focus on a narrow antimicrobial focus continues to dominate the search for solutions today. For example, an analysis of viral pneumonia treatment during the current pandemic conducted by T. M. Rawson at al. [25].Showed that bacterial or fungal co-infection was detected in only 8% of cases, and antibiotics were given to 72% of patients.

Secondly, the basis of the disease remains the same. We continue to talk about acute inflammation in the lung tissue, and the General nosological formulation retains its name "acute pneumonia". Undoubtedly, there are differences in the elements of inflammatory transformation of tissue structures between bacterial and viral lesions, but the localization of the inflammatory process captures the same layers and elements of the organ, which indicates the identity of functional disorders in these variants of inflammation [26-28]. Therefore, the pathogenesis of the disease can not have fundamental differences depending on its etiology.

Finally, understanding the etiology and pathogenesis of any disease makes it possible to apply both etiotropic and pathogenetic approaches in their treatment. Currently, the lack of drugs to suppress coronavirus does not allow the use of etiotropic treatment in patients with a new form of AP. However, even if such drugs were available to modern medicine, their therapeutic effect could not be sufficient and equivalent for the entire category of these patients. Running mechanisms of the inflammatory response require additional pathogenetically based assistance, which can both speed up and slow down this response [10,11]. Such assistance is currently extremely important for coronavirus lung lesions, since morphological studies of the lung tissue show severe damage, especially in the vascular sector up to the development of thromboembolism [29,30].

CONCLUSION

Thus, we must pay due attention to a number of facts that reflect the impact of prolonged exposure to antimicrobials on the proportions and balance of the biological environment around us. For expert assessment and understanding of the role of these facts in the search for a solution to the problem of AP, it is necessary first of all to review the existing system of views, which is in contradiction with a number of scientific truths and axioms. The current situation, which has revealed the weaknesses of modern medicine in helping patients with viral pneumonia, is a clear indication of the crisis in this section of health care and a call for radical reform of the AP doctrine. In an era of advanced technology and the ability to objectively track the evaluation of various hypothetical initiatives and innovations, such work is doomed to success.

REFERENCES

1. Liapikou A, Rosales-Mayor E, Torres A. The management of severe community acquired pneumonia in the ICU. Expert Rev Respir Med.2014;8:293-303.
2. Vidal A, Santos L. Comorbidities impact on the prognosis of severe acute community-acquired pneumonia. Porto Biomed. 2017;2:247-346.
3. Kim JW,Kim JJ, Yang HJ, Lim YS, Cho JS, Hwang IC, et al. The Prognostic Factors of Pneumonia with septic shock in patients

presenting to the emergency department. Korean J Crit Care Med 2015; 30: 258-264

4. Morgan AJ, Glossop AJ. Severe community-acquired pneumonia. BJA Edu 2016; 16: 167-172.
5. Waterer GW M,Quasney W, Cantor RM, Wunderink RG. Septic Shock and Respiratory Failure in Community-acquired Pneumonia Have Different TNF Polymorphism Associations.Am J RespirCrit Care Med. 2001;163: 1599-1604.
6. Schlapbach LJ, Straney L, Alexander J,MacLaren G, Festa M, Schibler A, et al. Mortality related to invasive infections, sepsis, and septic shock in critically ill children in Australia and New Zealand, 2002-13: Amulticentre retrospective cohort study. Lancet Infect Dis. 2015; 15:46–54.
7. Lin GL, McGinley JP, Drysdale SB. Epidemiology and immune pathogenesis of viral sepsis. Front Immunol 2018; 9:2147
8. Elliott C G. Pulmonary physiology during pulmonary embolism. Chest. 1992;101:163S–171S.
9. Turetz M, Sideris AT, Friedman OA, Triphathi N, Horowitz JM .Epidemiology, Pathophysiology, and Natural History ofPulmonary Embolism. SeminIntervent Radiol. 2018;35:92–98.
10. Igor Klepikov.The role and importance of biological stereotypes in the pathogenesis of acute pneumonia. EC pulmonology and respiratory medicine. 2019; 83: 239-246.
11. Klepikov I. Acute pneumonia and its purulent and destructive complications in children in the midst of a major industrial centre of Western Siberia. Dissertation for the degree of doctor of medical science. Leningrad. 1989
12. Nair GB, Niederman MS. Community-acquired pneumonia: An unfinished battle. Med Clin North Am. 2011; 95 : 1143–61.
13. Ruuskanen O, Lahti E, Jennings LC, Murdoch DR.Viral pneumonia. Lancet. 2011; 377: 1264–1275.
14. Groneberg DA, Hilgenfeld R, ZabelP.Molecular mechanisms of severe acute respiratory syndrome (SARS). Respir Res.2005; 6: 8
15. Peyrani P, Mandell L, Torres A, Tillotson GS. The burden of community-acquired bacterial pneumonia in the era of antibiotic resistance.Expert Rev Respir Med. 2019;13 :139-152,
16. Grasselli G, Zangrillo A, Zanella A, Carbini L,Castelli A, Castelli A, et al. (2020). Baseline characteristics and outcomes of 1591 patients infected with SARS-CoV-2 admitted to ICUs of the Lombardy region, Italy. JAMA.2020 ;323:1574-1581.
17. Johns Hopkins Center for Health Security. Ventilator stockpiling and availability in the US. (2020).
18. Ranney ML, Griffeth V, JhaA K. Critical Supply Shortages The Need for Ventilators and Personal Protective Equipment during the Covid-19 Pandemic. N Engl J Med. 2020; 382:41.
19. Richardson S, Hirsch JS, Narasimhan M. Presenting Characteristics, Comorbidities, and Outcomes Among 5700 PatientsHospitalized With COVID-19 in the New York City Area. JAMA. 2020; 323: 2052–2059.
20. Rubin EJ, Baden LR, Morrissey S. Audio Interview: Approaches to Covid-19 Vaccines and Antivirals. N Engl J Med 2020; 382:58.
21. Bhimraj A, Morgan RL, Shumaker AH. Infectious Diseases Society of America Guidelines on the Treatment and Management ofPatients with COVID-19. 2020.
22. Peyrani P, Mandell L, Torres A, Tillotson GS. The burden of community-acquired bacterial pneumonia in the era of antibiotic resistance.Expert Rev Respir Med. 2019;13:139-152.
23. Weiss SL, Peters MJ, Alhazzani W, Agus MSD, Flori HR, et al.Surviving Sepsis Campaign International Guidelines for theManagement of Septic Shock and Sepsis-Associated Organ Dysfunction in Children.PediatrCrit Care Med. 2020;21: 52-106.
24. Metlay JP, Waterer GW, Long AC, Anzueto A, Brozek J, et al. On behalf of the American Thoracic Society and Infectious Diseases Society of America. "Diagnosis and Treatment of Adults with Community-acquired Pneumonia. An Official Clinical Practice Guideline of the American Thoracic Society and Infectious Diseases Society of America". Am J RespirCrit Care Med. 2019; 200:45-67.
25. Rawson TM, Moore LSP, Zhu N,Ranganathan N, Skolimowska K, Gilchrist M, et al. Bacterial and fungal co-infection in individuals with coronavirus: A rapid review to support COVID-19 antimicrobial prescribing. Clin Infect Dis. 2020; 530.
26. Hwang DM, Chamberlain DW, Poutanen SM, Low DE, AsaSL,et al. Pulmonary pathology of severe acute respiratory syndrome in Toronto. Mod Pathol.2005;18:1–10.
27. Xu Z, Shi L, Wang Y, Zhang J, Huang L, Zhang c, et al. Pathological findings of COVID-19 associated with acute respiratory distress syndrome. Lancet Respir Med 2020; 8:420-422.
28. Luo W, Yu H, Gou J, Li X, Sun Y, et al. Clinical pathology of critical patient with novel coronavirus pneumonia (COVID-19). PatholPathobiol 2020:2020020407.
29. Ackermann XM, Verleden SE, Kuehnel M, Haverich A, Welte T,Laenger F, et al. Pulmonary Vascular Endothelialitis, Thrombosis, and Angiogenesis in Covid-19. N Engl J Med. 2020; 383:120-128.
30. Abernethy K, Sivakumar P, Patrick T, Robbie H, Periselneris J. Coexistent COVID-19 pneumonia and pulmonary embolism: challenges in identifying dual pathology.Thorax. 2020;75.