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Opinion

What does the Current Terminology of Acute Pneumonia Mean for Clinical Practice?

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Abstract

The modern concept of acute pneumonia is formed on the principle of the dominant role of the pathogen in the occurrence and development of the disease, and targeted etiotropic therapy continues to be considered as the only true path to success. The decrease in the effectiveness of antibiotics as a result of the development of microbial resistance, the constant dynamics of etiology, dissatisfaction with the results of emergency bacteriological diagnostics and the desire to increase the effectiveness of antimicrobial therapy have given rise to a tendency to classify the disease depending on the conditions of its occurrence, suggesting infection with various pathogens depending on the situation. The lack of convincing practical achievements in this case is another argument for revising such narrowly selective ideas about the nature of the disease, and the steady growth of viral forms in recent years is an incentive to accelerate this process. The essence of this classification may be of interest to statisticians, but does not have a significant impact on clinical results.

Keywords: *Acute Nonspecific Inflammation in the Lungs; Acute Pneumonia; Etiology; Classification; Pathogenesis; Conception of Disease*

What pneumonia is today is known not only to professional specialists, and the term "acute pneumonia" (AP) has long been used to refer to the most severe and dramatic manifestations of this disease. Already in the early stages of the origin and development of microbiology, it was established that acute inflammation of the lung tissue can be caused by more than one microorganism [1] and as knowledge in this direction expanded, this disease began to be designated by the additional term "acute nonspecific inflammation in the lungs" (ANSIL), which was more of a broader description and decoding of the term AP than an independent designation. The clinical essence of the disease and its features continued to be defined by the term AP.

The introduction of antibiotics into widespread clinical practice quickly gave rise to the idea that these drugs could serve as the basis for the treatment of this category of patients. For quite a long time, the optimal treatment for most patients with AP was "antibiotics alone." However, the duration and continuity of this approach have been hampered by the natural development of bacterial resistance and a marked slowdown in the pace of development of new, more effective drugs [2]. The conviction that had developed by this time in the primacy of the pathogen and the decisive importance of the targeted use of antibiotics to achieve success was an urgent requirement of that time to bring the process of targeted diagnosis and treatment to a new level.

Since the accuracy and speed of obtaining results of microbiological diagnosis of AP left much to be desired, an approximate method of etiological diagnosis was proposed. For this purpose, the most common pathogens of AP were identified depending on the conditions in

which the disease occurs. According to this principle, AP was divided into groups such as community-acquired (CAP), hospital-acquired (HAP), ventilatory-acquired (VAP) and others, where for each form a specific list of antimicrobial drugs was recommended for their rapid empirical selection. The use of this technique did not bring the expected results, and the effectiveness of the modern complex of medical care for patients with AP continues to slowly but steadily decline. However, the introduced terminology, against the backdrop of the persistent dominance of the concept of "microbe-antibiotic", continues to be widely used in everyday practice. This classification of the disease today has the character of a familiar stereotype, which is more important for statistics than for final results.

Despite the lack of positive clinical results from the application of the above classification of AP, in recent years one can observe an expansion of the original scale and further attempts to improve it. Thus, a new form has emerged, uniting patients with a disease that arose during treatment in intensive care units - "ICU-acquired pneumonia" [3,4]. In this case, we are talking not so much about a newly emerging disease, but about the addition of a new pathogen to an existing inflammation of the lung tissue in the form of a bacterial or viral infection [5-7]. The study of this issue in patients hospitalized in intensive care units is aimed primarily at confirming the role of specific types of microorganisms in the development of critical conditions, with an emphasis on resistant microflora and the aggressiveness of coronavirus. The authors of these studies openly acknowledge the lack of such convincing evidence, but express the hope that it will be obtained through further research.

It is easy to see that the principle underlying the appearance of this gradation remains unchanged for many years, despite the changes that have occurred during this time in the etiological characteristics of AP, and also despite the accumulation of new facts that contradict the prevailing concepts of views on this disease. Over the long period of use of antibiotics in the treatment of AP, the leading causative agents of the process have repeatedly changed, putting forward new microorganisms as leaders and expanding the share of viruses. Each segment of this period was characterized by attempts to neutralize certain types of pathogens, which were gradually replaced by others, but the general trend in the development of the disease and, most importantly, its classical manifestation remained the same, all the time returning us to fundamental ideas about the meaning of inflammation and its classical symptoms. However, modern medicine, while continuing to persistently declare the leading role of the pathogen in the occurrence and development of AP, has not been able to provide criteria for differential diagnosis depending on the etiology of not only bacterial forms, but also their difference from viral ones [8-10].

The phenomenon of heterogeneous manifestations of the same disease has been known for a long time, and AP in this case is no exception. At the same time, emerging problems in providing care to patients are most clearly and distinctly manifested in cases of aggressive and rapid development of the process, allowing in such situations to note the maximum severity of those deviations, the tendency to which is characteristic of all observations of this nosology. The essence of disease assessments on this scale has a wide range from barely noticeable signs to the occurrence of critical situations. The latter variant of the development of AP is characterized by the need for early intensive therapy, an increased tendency to develop various types of complications and high mortality. This feature of the individual development of AP has recently begun to attract closer attention from researchers due to the fact that the problem of this disease is most clearly and expressively manifested in patients with a severe onset of the process, and the results of their treatment serve as an indicator of its effectiveness.

On the one hand, the SARS-CoV-2 pandemic has clearly demonstrated the important role and determining influence of the body's individual response to contact with an identical pathogen. In conditions of equivalent mass infection of large populations and the absence of specific etiotropic means of assistance, the results of such contact had diametrically opposite consequences - from latent carriage of the pathogen (up to 40%) to the need for hospitalization (about 20%) due to COVID-19 pneumonia [11-13]. In this situation, it was no longer possible to ignore such an obvious fact, when the observed results of the spread of coronavirus in the context of the fear that arose in front of it did not fit into the prevailing theory of the predominant influence of the pathogen. Individual characteristics of the body's response to the occurrence and development of inflammatory processes in the lungs were discussed based on the materials of the pandemic at

last year's meeting of the American Thoracic Society, but such messages were purely informational in nature without any changes in the strategy and principles of treatment [14-16].

On the other hand, it seems that the panorama of professional ideas about the essence of the problem of AP is finally beginning to expand, creating the necessary springboard for revising the concept of the disease and the principles of its treatment. Unfortunately, such feelings and expectations so far look deceptive and do not correspond to the current situation. As is known, the main contingent of patients with AP is replenished due to the so-called CAP, however, in recent years, experts have begun to pay special attention to severe forms of this disease, highlighting a separate type in the above classification - "severe community-acquired pneumonia, sCAP" [17-19]. This identification of sCAP as a special group of observations is due to an attempt to purposefully analyze the results of their treatment, which actually concentrate all the negative variants of this disease. The main purpose of this separation of patients, as stated by the authors of such studies, is to search for signs and prerequisites that make it possible to identify patients with a high probability of severe development of the process in the early stages of the disease. At the same time, the main approaches to interpreting the causes of the problem and eliminating them remain the same, and a significant place in such analytical studies is still given to early diagnosis of the pathogen and the desire for targeted etiotropic therapy [20-23].

Thus, the process of dividing ANSIL into separate categories observed in recent decades is based on the desire to indirectly emphasize the features of the etiology of various variants of the disease. This trend arose against the backdrop of continuing difficulties in quickly diagnosing the causative agent of AP and a decrease in the effectiveness of etiotropic therapy, and the very principle of this classification emphasizes the inviolability of long-standing ideas about the predominance of the pathogen in the occurrence and development of this disease and the desire to revive the primary prestige of antimicrobial drugs. The clinical usefulness of such a classification remains, to put it mildly, more than questionable, especially in light of ongoing changes in the etiological characteristics of AP. Over the past decades, the share of viruses among the causative agents of AP reaches at least half of all observations [24-26] which is especially noticeable in recent years. Such transformations in the etiology of the disease reduce the feasibility of using antibiotics and the meaning of using this gradation. But, most importantly, over the entire period of application of this classification, it was not possible to obtain a significant improvement in treatment results.

Information about the nature of AP for many centuries was very limited, but this did not prevent doctors of that time from quite rightly considering inflammation as the main and integral sign of this disease. The five signs of inflammation, described about two thousand years ago by Celsus and Galen, have stood the test of time, becoming classic manifestations of these processes. A special place in this list is occupied by the loss of function of those body structures that are in the zone of inflammation. It is this sign of the inflammatory process that forms its clinical manifestations depending on the location of the lesion. According to modern ideas about the leading role of the pathogen in the development and manifestations of AP, in recent years, repeated attempts have been made to determine differential diagnostic criteria for various types of AP depending on the etiology of the process. The absence of such signs, the specificity and identity of the clinical picture of the disease make us recall functional disorders of the affected organ, however, attempts to find differences in AP depending on the pathogen continue to this day [8-10,20].

Along with fundamental ideas about the development and manifestation of inflammation, it is necessary to remember the purpose and capabilities of etiotropic drugs in general and antibiotics in particular. The current tendency to rely primarily on the action of etiotropic drugs does not take into account their narrow antimicrobial specificity of action and the inability to have a direct effect on the mechanisms of inflammation. In the case of aggressive development of AP, immediate elimination of disease stimuli emanating from the primary focus is necessary through targeted influence on the mechanisms of the process. The desire to neutralize the pathogen and rely on an indirect effect, which cannot manifest itself instantly and requires waiting, is a waste of time to provide timely pathogenetic assistance.

In addition, it is time to pay attention and give an appropriate critical assessment to such a fact as the use of one antimicrobial drug as the main treatment for completely different and incomparable diseases. The only factor that unites them is the causative agent of inflammation, while the pathogenesis and clinical picture of such diseases have nothing in common. If for pneumonia, meningitis and tonsillitis of pneumococcal etiology, one antibiotic is prescribed as the leading remedy, then what result do you want to get in case of aggressive development of AP?

Conclusion

Thus, the classification of AP according to etiological principles has not brought and cannot bring the expected benefit in everyday clinical practice, creating a false impression of targeted treatment selection. The gradations of the disease that are widely used today may be of interest for statistical processing of the material, but do not bring the expected practical benefit. The generator of such classifications is the modern concept of AP, formed on the basis of the narrow principle of the dominant role of the pathogen and the guarantee of success in the event of its suppression, and the leading role of such ideas today makes it difficult to solve the problem under discussion.

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Conflict of Interest

The author states that he has no conflict of interest.

Bibliography

1. Gram C. "Über die isolierte Färbung der Schizomyceten in Schnitt- und Trocken-präparaten". *Fortschritte der Medizin* 2.6 (1884): 185-189.
2. Aminov RI. "A brief history of the antibiotic era: lessons learned and challenges for the future". *Frontiers in Microbiology* 1 (2010): 134.
3. Llitjos JF., *et al*. "Increased susceptibility to intensive care unit-acquired pneumonia in severe COVID-19 patients: a multicentre retrospective cohort study". *Annals of Intensive Care* 11.1 (2021): 20.
4. J Johnstone., *et al*. "Definitions, rates and associated mortality of ICU-acquired pneumonia: A multicenter cohort study". *Journal of Critical Care* 75 (2023): 154284.
5. Kristin R Wise., *et al*. "Increased long-term mortality in patients admitted to the intensive care unit with health-care associated pneumonia". medRxiv (2021): 2021.12.23.21267010.
6. Lakbar I., *et al*. "Association between mortality and highly antimicrobial-resistant bacteria in intensive care unit-acquired pneumonia". *Scientific Reports* 11 (2021): 16497.
7. Lee YH., *et al*. "Risk factors for mortality in intensive care unit patients with *Stenotrophomonas maltophilia* pneumonia in South Korea". *Acute and Critical Care* 38.4 (2023): 442-451.
8. C Heneghan., *et al*. "Differentiating viral from bacterial pneumonia". The Centre for Evidence-Based Medicine. Evidence Service to support the COVID-19 response. University of Oxford (2020).
9. Kamat IS., *et al*. "Procalcitonin to distinguish viral from bacterial pneumonia: a systematic review and meta-analysis". *Clinical Infectious Diseases* 70.3 (2020): 538-542.

10. Lhommet C., *et al*. "Predicting the microbial cause of community-acquired pneumonia: can physicians or a data-driven method differentiate viral from bacterial pneumonia at patient presentation?" *BMC Pulmonary Medicine* 20.1 (2020): 62.

11. Zheng YY., *et al*. "COVID-19 and the cardiovascular system". *Nature Reviews Cardiology* 17.5 (2020): 259-260.

12. Oran DP and Topol EJ. "Prevalence of asymptomatic SARS-CoV-2 infection". *Annals of Internal Medicine* 173.5 (2020): 362-367.

13. Wu Z and McGoogan JM. "Characteristics of and important lessons from the coronavirus disease 2019 (COVID-19) outbreak in China: summary of a report of 72314 cases from the Chinese Center for Disease Control and Prevention". *Journal of the American Medical Association* 323.13 (2020): 1239-1242.

14. The American Thoracic Society (ATS) 2023 International Conference". Washington, D.C., USA, in May 2023. *Respiratory AMJ* 1.1 (2023): 26-35.

15. Pratik Sinha. "Severe viral lower respiratory tract infections pose a significant burden on patients and healthcare systems". *Respiratory AMJ* 1.1 (2023): 26-35.

16. Nuala Meyer. "Dysregulated host immune response is the driver of disease progression and severe patient outcomes". *Respiratory AMJ* 1.1 (2023): 26-35.

17. Cao L., *et al*. "Epidemiology and mortality predictors for severe childhood community-acquired pneumonia in ICUs: A retrospective observational study". *Frontiers in Pediatrics* 11 (2023): 1031423.

18. Pierre-François Daquan., *et al*. "Hydrocortisone in severe community-acquired pneumonia". *New England Journal of Medicine* 388.21 (2023): 1931-1941.

19. Martin-Loeches I., *et al*. "ERS/ESICM/ESCMID/ALAT guidelines for the management of severe community-acquired pneumonia". *Intensive Care Medicine* 49.6 (2023): 615-632.

20. Alex R Schuurman., *et al*. "The host response in different aetiologies of community-acquired pneumonia". *The Lancet, Discovery Science* 81 (2022): 104082.

21. Jeremy Hsu. "AI discovers new class of antibiotics to kill resistant bacteria". *New Scientist* 261.3472 (2024): 12.

22. De Corte T., *et al*. "Characteristics of co-infection and secondary infection amongst critically ill COVID-19 patients in the first two waves of the pandemic". *Intensive Care Medicine* 50.6 (2024): 989-993.

23. Kling K Qi., *et al*. "The impact of next-generation sequencing added to multiplex PCR on antibiotic stewardship in critically ill patients with suspected pneumonia". *Diagnostics* 14.13 (2024): 1388.

24. Rudan I., *et al*. "Epidemiology and etiology of childhood pneumonia". *Bulletin of the World Health Organization* 86.5 (2008): 408-416.

25. WHO Revised global burden of disease 2002 estimates (2004).

26. Ruuskanen O., *et al*. "Viral pneumonia". *Lancet* 377.9773 (2011): 1264-1275.

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