

Pandemic and modern medicine: time to recognize and correct previous misconceptions

Igor Klepikov

Pediatric Surgeon, 2116 27th St. NE Renton, WA, USA.

***Corresponding Author:** Igor Klepikov, Pediatric Surgeon, 2116 27th St. NE Renton, WA, USA.

Received date: September 24, 2020; Accepted date: November 23, 2020; Published date: December 07, 2020

Citation: I Klepikov. (2020) Pandemic and modern medicine: time to recognize and correct previous misconceptions. General medicine and Clinical Practice. 3(4) DOI: [10.31579/2639-4162/039](https://doi.org/10.31579/2639-4162/039)

Copyright: ©2020 Igor Klepikov. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

A year ago, few people doubted the scientific validity and practical feasibility of existing approaches to the treatment of acute pneumonia (AP). Even the use of a single antibiotic as the main treatment for completely different and disparate diseases was a common situation, the contradiction of which to the basics of clinical medicine was not questioned. The activity of antibiotics only against bacterial pathogens without additional effects on other manifestations of the disease did not prevent them from achieving the image of an almost panacea.

The gradual decrease in the effectiveness of antibiotics with the biologically natural appearance of resistant bacterial strains was regularly compensated by the release of new, more active drugs. Decades of close attention to the suppression of pathogens have formed a persistent infectious ideology of the causes of AP, and the disease itself has become classified as infectious. However, this terminology did not correspond to the actual situation, since in practice patients with AP were not subject to mandatory isolation and other strict anti-epidemic measures. At the same time, during the period of antibacterial therapy, the number of patients in need of additional medical care steadily increased, but the infectious interpretation of the nature of the disease suggested the choice of methods tested for other inflammatory processes. The specifics of pulmonary inflammation faded into the background and no longer determined the specifics of treatment.

The hypnotic effect of infectious distortion of the AP ideology affected its diagnosis and control assessment. Various methods for determining the pathogen for the targeted use of antibiotics in AP did not bring the expected results. Throughout the entire period of use of antibiotics, their choice in AP remained empirical, that is, based on General recommendations, assumptions and experience of doctors. In the vast majority of cured patients, the causative agent of the disease remained unknown, and experts eventually concluded that attempts to make a microbiological diagnosis of AP did not affect the results of treatment (1). However, the uncertainty of the bacteriological diagnosis and the known probability of involving a whole galaxy of microorganisms in this inflammation led to a broad explanation of treatment failures (without convincing arguments) by the presence of particularly virulent strains.

The primary [1] diagnosis of AP is also not directly related to infectious disease assessments. Treating doctors do not wait for the response of bacteriological tests, but are guided by the results of x-ray studies, which allow you to determine the degree of inflammatory changes, but not the pathogen and its type, right? The bacterial pathogen of inflammation acts

like a burning match, but the "fire" that has started (the inflammatory process) develops further according to its biological canons, which have long been known to medical science and these materials are included in its classic Fund. Therefore, the course of treatment with "antibiotics alone" leaves the extinguishing of this "fire" to the body itself.

At the present time, when scientific developments and research have allowed us to reach the genetic, subcellular and molecular levels, it is unlikely that anyone will find the question of our individual uniqueness debatable. The ability of the body to adapt to sudden functional deviations is one of its unique features, along with many other personal characteristics that we habitually and automatically use in everyday life. Compensatory capabilities in inflammatory diseases depend on the rate of development of the local process and its localization.

For example, when a boil occurs, we lose some or all of the activity of the corresponding area of our body, although some people do not pay much attention to this abscess and irritation. Inflammation of the middle ear is accompanied by an infinite number of individual hearing disorders. Similar examples can be given for any tissue or organ of our body, since functional disorders are a classic sign of inflammation, and their manifestation depends on the violation of the function of a particular organ, and not on our assumptions and analogies with other inflammatory processes caused by a similar pathogen.

When acute inflammation occurs in the lungs, it is usually considered a respiratory disaster, which is quite logical and corresponds to the functional characteristics of the affected organ. However, this ignores the fact that the lungs are an integral part of the circulatory system and its regulation. Representing one of the halves of the entire circulatory system in the body, the vessels of the lungs are functionally the complete opposite of the vessels of organs and tissues on the periphery. Blood pressure in the lungs is several times lower than in the large circle of blood circulation, and this proportion is regulated autonomously, in particular, by the baroreceptors of the pulmonary vessels. A persistent violation of this ratio is incompatible with the synchronous operation of the cardiovascular system and the preservation of the body's viability, and direct analogies between AP and other inflammatory diseases based on identical blood circulation tests are obviously doomed to incorrect conclusions.

Recording blood pressure in patients with AP is one of the simplest and most widely available methods for monitoring their condition. At the same time, when deciding on corrective therapy, one should take into

account the fact that the cause of the disease is in the pulmonary vessels and the decrease in peripheral pressure may be no more than a reflection of compensatory shifts.

The above information is necessary to understand why modern approaches to the treatment of patients with AP suddenly stopped working with the onset of the coronavirus pandemic. Yes, this infection, unlike bacterial pneumonia, is characterized by its contagiousness and the ability to spread quickly, repeating the scenario of annual viral epidemics. Yes, COVID-19 has led to the need for strict compliance with anti-epidemic measures and the introduction of quarantine regimes. Yes, since the beginning of the pandemic, the number of patients with viral pneumonia has increased significantly, and practical medicine has lost antibiotics as a treatment, but lung damage in patients with coronavirus infection is one of the options for AP, and inflammatory changes affect the same lung structures as bacterial processes [2, 3]. A new term "COVID-19 pneumonia" has already appeared in the literature [4].

In the previous period, the annual number of cases of AP in the world was estimated at about 450 million, with the number of deaths being about 4 million [5,6]. If we compare these statistics with current estimates of COVID-19, the impartiality of the figures shows that the pandemic is significantly inferior to previous indicators. In the recent past, the negative dynamics of AP against the background of antibacterial therapy had its own explanations, and the existing treatment principles were supported and approved by specialists. The usual traditions were destroyed almost suddenly, and the reason for this fiasco of all complex AP therapy was the loss of only one type of medication.

The invasion of the coronavirus clearly showed that the scientifically based complex of AP treatment turned out to be an illusion and was, in fact, represented by a single group of drugs that do not have a specific effect on this disease. Simply, the pandemic has made its own adjustments to this problem and made it easier to check and evaluate the real therapeutic potential of modern medicine in patients with AP. Many specialists have already realized that there is no balanced medical care specific to AP, but the long-instilled and dominant image of the leading role of the pathogen prevents a panoramic assessment of the problem, justification and use of other directions in treatment [7, 8].

The lack of reliable and effective treatment methods for AP subconsciously supports the use of antibiotics, which are meaningless in viral infection, but which are currently received by 71 to 82.9% of patients with coronavirus pneumonia [9-11]. Modern specialized literature and mass media today are filled with expectations of the release of a vaccine for the prevention and reduction of morbidity and hopes for the development of effective antiviral drugs. All these hopes and expectations for the future cannot really help those who are already ill at the moment, and professional discussions are mainly focused on replacement and supportive methods, such as oxygen supply and auxiliary breathing options [7, 8, 12].

For many years, textbooks and manuals identified bacteria and viruses as the leading pathogens of AP, and the mention of the latter was more declarative than practical, since the proportion of such patients was small, and the proposed therapy was not focused on this option. Today, the importance of viral pneumonia has increased significantly, which requires a review of approaches and solutions. The explanation of the severity of the disease by the special virulence of its pathogen can no longer be considered as a serious argument. For example, observation and analysis of morbidity in the unique conditions of large isolated groups shows that infection with coronavirus as one identical pathogen is asymptomatic in more than 80% of cases and does not lead to disease [10, 11]. These indicators reflect not only the features of the epidemiology and spread of infection, but also the importance of individual characteristics of patients in the development of the disease.

The long period of use of antibiotics was marked not only by their undoubted benefits, but also by negative phenomena. The biological consequences of using these drugs in the form of transformation of the microcosm around us are not yet fully evaluated, but the growth of viral diseases observed in recent years can be considered as one of the long-term results of such therapy. Viral pneumonia has become a growing problem over the past couple of decades. The same coronavirus has already manifested itself in at least two epidemics (SARS, MERS). In addition, an exaggerated perception of the therapeutic properties of antibiotics played a negative didactic role and led to the forgetting of the scientific foundations of the unique nature of AP.

The need to reduce the functional load on the affected organ has long been known to medicine and is used as an important condition for helping patients with various diseases. Previous empirical experience of AP treatment has shown an adaptive effect of such procedures as, for example, cupping therapy or short-term cooling of the patient's body. Today, modern technical capabilities of medicine allow us to conduct objective testing of such methods and evaluate their therapeutic qualities at a new level. However, currently such methods are used everywhere, including in fitness clubs and beauty salons, but not in emergency pulmonology, where they are most needed.

In order to make a real step in solving the problem of AP, it is necessary first of all to change the system of views, which should be based on facts and classical scientific materials, and not on assumptions and declarations. This step has already been taken, and it is expected that its impressive results will continue and be further developed. A more detailed description of this material and the first results of its successful clinical testing can be found in Igor Klepikov's recently published monograph "Acute pneumonia. New doctrine and first treatment results" - ISBN (978-620-2-67917-6) > {<https://www.cheapesttextbooks.com/IM/?keyval=ISBN+%28978-620-2-67917-6%29>}.

Many materials and facts accumulated on the topic of acute pneumonia do not correspond to or even contradict existing ideas about the nature of the disease, which has long required a thorough and comprehensive analysis of the entire set of problems. The beginning of the pandemic has left medicine with a choice - to continue the fight against the new pathogen of AP, repeating the already passed scenario with antibacterial therapy and hoping for possible success in the future, or, recognizing the mistakes made, to start a new stage of treatment of patients today. Such an audit is necessary not only to avoid repeating mistakes and improve previous achievements at a qualitatively new level, but also to determine the reasons for the complication of the situation, assess the actual level of danger and future prospects, as well as to choose the rational way to further solve the problem.

References

1. J. P. Metlay, G. W. Waterer, A. C. Long at al. (2019) 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". American Journal of Respiratory and Critical Care Medicine. 200(7);45-67.
2. Zhe Xu, Lei Shi, Yijin Wang at al. (2020). Pathological findings of COVID-19 associated with acute respiratory distress syndrome. The Lancet Respiratory Medicine. 8(4); 420-422.
3. M. Ackermann, S. E. Verleden, M. Kuehnel at al. (2020). Pulmonary Vascular Endothelialitis, Thrombosis, and Angiogenesis in Covid-19.

4. Lipman M, Chambers RC, Singer M, et al. (2020). SARS-CoV-2 pandemic: clinical picture of COVID-19 and implications for research. *Thorax* 2020; 75:614-616.
5. Ruuskanen O, Lahti E, Jennings LC, Murdoch DR (April 2011). "Viral pneumonia". *Lancet*. 377 (9773): 1264–75. doi:10.1016/S0140-6736(10)61459-1466. PMC 7138033. PMID 21435708.
6. Lodha R, Kabra SK, Pandey RM (June 2013). "Antibiotics for community-acquired pneumonia in children". *The Cochrane Database of Systematic Reviews*. 6 (6): CD004874. doi:10.1002/14651858.CD004874.pub4. PMC 7017636. PMID 23733365.
7. Mayank Vats., et al. "Second Wave of COVID-19 - Could it be More Lethal!!! - Novel 6 T's Strategies May Help". *EC Pulmonology and Respiratory Medicine* 9.8 (2020): 17-21.
8. D. A. Berlin, R. M. Gulick, F. J. Martinez (2020). Severe Covid-19. *NEJM*.
9. Rawson TM, Moore LSP, Zhu N, et al. (2020). Bacterial and fungal co-infection in individuals with coronavirus: A rapid review to support COVID-19 antimicrobial prescribing.
10. Beović, M. Doušak, J. Ferreira-Coimbra et al. (2020). Antibiotic use in patients with COVID-19: a 'snapshot' Infectious Diseases International Research Initiative (ID-IRI) survey. *Journal of Antimicrobial Chemotherapy*.
11. Kim D, Quinn J, Pinsky B et al. (2020). Rates of co-infection between SARS-CoV-2 and other respiratory pathogens. *JAMA* 2020; 323: 2085–2086.
12. Winearls S, Swingwood EL, Hardaker CL, et al. (2020). Early conscious prone positioning in patients with COVID-19 receiving continuous positive airway pressure: a retrospective analysis. *BMJ, Open Respiratory Research*.
13. Keeley AJ, Evans CM, de Silva TI (2020). Asymptomatic SARS-CoV-2 infection: the tip or the iceberg? *Thorax*. 75:621-622.
14. Ing AJ, Cocks C, Green JP (2020). COVID-19: in the footsteps of Ernest Shackleton. *Thorax*. 75:693-694.