

Professional Passion for Antibiotics in The Age of Viral Pneumonia

Igor Klepikov*

Professor, Retired, Renton, WA, USA.

*Corresponding Author: Igor Klepikov., Professor, Retired, Renton, WA, USA.

Received Date: 27 October 2025 | Accepted Date: 10 November 2025 | Published Date: 26 November 2025

Citation: Igor Klepikov, (2025), Professional Passion for Antibiotics in The Age of Viral Pneumonia, *Journal of Clinical and Laboratory Research*, 8(5); DOI:10.31579/2768-0487/188

Copyright: © 2025, 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.

Abstract

These facts, from the point of view of logic and the cause-and-effect relationship of the observed events, are a striking example of an inexplicable selective approach to assessing one's own professional activity. Is it necessary to further explain that such an understanding of the problem crosses out any hopes for progress in this direction? Finally, current efforts to move the solution of the AP problem from a dead point lack a comprehensive connection with the real situation in this area and do not take into account the facts without which one cannot count on the expected success

Key words: drugs; disease; microbiology

Introduction

The concept of acute inflammation of the lung tissue or acute pneumonia (AP) has changed throughout the history of this disease depending on the discovery of new information about the essence of the process. Thus, for many centuries this disease was considered a severe inflammation that did not pose a danger to others. The development of microbiology made it possible to identify the causative agents of these processes, showing that AP does not belong to the category of specific inflammations and can be caused by various types of bacteria, including ordinary symbionts of the body [1,2]. These circumstances confirmed the old postulate that pneumonia is something you get sick with, not something you catch. Since the discovery and study of microbiological factors in the etiology of AP, *Streptococcus pneumoniae* has emerged among its pathogens, which, due to its absolute prevalence, has received a very specific term. The frequency of pneumococcus in the etiology of AP was stable in the list of pathogens at a level of 95% or more until the middle of the last century [3-7]. Beginning from this period, after the discovery and widespread use of antibiotics, the hegemony of this pathogen began to disappear irrevocably and never again even approached its original indicators [8]. However, looking ahead, it is necessary to note a very characteristic and indicative fact. By now, the etiology of pneumonia has changed dramatically compared to the initial period of antibiotic use. However, this obvious phenomenon does not affect the treatment strategy. The main goal for many years was the early detection of the causative agent of the process to accelerate targeted antimicrobial therapy. But despite the constant improvement of microbiological diagnostic methods, in more than half of the cases the causative agent of AP remains unidentified [8]. Among the remaining observations of the disease in which

it was possible to establish the etiology with a certain degree of probability, the prevalence of viral forms of inflammation is increasingly noted [9-11]. Thus, in reality, bacterial pneumonia accounts for only a small number of cases. Therefore, the fairly widespread assessment of the role of pneumococcus in the etiology of AP quite reasonably causes surprise and concern. Thus, despite such meager statistics of bacterial forms of AP, a number of specialists continued to assert until recently that the main causative agent of the disease remains pneumococcus, noting its leadership in the list of established microbial variants [12,13]. Even greater controversy is caused by the recommendations often given on the basis of such conclusions regarding the further improvement of pneumococcal vaccines [12,13]. If we consider such statements from the position of even theoretical unprovenness of the fact that a vaccine against one pathogen can ensure success with such a polyvalent etiology, which is characteristic of AP, then we can understand the basis of those paradoxes that are observed today in this section of medicine. For a long period, the etiology of AP was associated exclusively with bacterial strains, which corresponded to the real state of affairs. For example, fungal forms of the disease were largely considered a consequence of intensive antibacterial treatment and were described in the literature as rare. In addition, they were not particularly acute in development. Currently, fungal forms are gradually ceasing to be a rarity in clinical practice and the experience of their treatment in the literature is already based on group observations [14,15]. Viral pneumonia, first described in the first half of the last century [16], was also a rare variant of acute inflammation of the lung tissue for a long time. However, by the early 2000s, the situation had changed significantly and viral forms of the disease

accounted for almost half of all AP observations in the world [17,18]. Surprisingly, such statistics did not affect treatment approaches, which continued to rely on antibiotics as the main treatment. The relatively sudden events associated with the SARS-CoV-2 pandemic should have become very illustrative and instructive examples for modern official medicine. On the one hand, a dilemma arose regarding the rapid spread of infection and the introduction of quarantine measures. At the same time, the flow of patients with inflammatory processes in the lung tissue unexpectedly increased. Such unexpected conditions and circumstances represented a genuine and impartial assessment of the quality and effectiveness of medical care, as well as its readiness for such cataclysms. On the other hand, the influx of patients with viral pneumonia COVID-19 into hospitals made it possible to analyze the professional understanding of the dynamics in the etiology of AP, which is observed throughout the entire period of antibiotic use, and to note the ability of expert thinking to adapt to new non-standard challenges. Now, as past and current events and phenomena of reality show, the reasons for the fiasco that medicine suffered during the pandemic remain largely misunderstood and unanalyzed. The tense, anxious and even panicky atmosphere that arose in society with the onset of the SARS-CoV-2 pandemic was not due to the super-aggressiveness of the unknown pathogen. The claim that the coronavirus was highly dangerous was largely far-fetched and arose as a result of the inability of official medicine to provide effective assistance in cases where such a need arose. We have enough counterarguments not to accept such a claim on faith without assessing the real circumstances. Firstly, not only the significant increase in viral pneumonia in recent decades required a revision of the principles of medical care, which continued to rely on antimicrobial drugs. By the time the pandemic emerged, medicine had already faced two major epidemics of this pathogen, characterized by a predominant tendency towards pulmonary processes (SARS and MERS), and coronavirus pneumonia continued to be registered in everyday practice in the post-epidemic period, accounting for up to 5% of cases among all observations of AP [19,20]. Despite these facts, there were no significant changes in the previous therapy, and this is also a fact. Secondly, the very fact of the rapid spread of the new strain of coronavirus and the introduction of quarantine measures did not have a significant impact on the results of morbidity and mortality. For example, the "Swedish experiment" in abandoning such measures yielded very impressive results [21,22]. The spread of coronavirus among the population of different continents was variable, reaching large scales in a number of countries. However, the ratio of the range of consequences of such infection remained the same regardless of the observation zone. On average, 20-40% of infected people demonstrated latent carriage of the pathogen, learning about it only after testing [23,24]. In other observations, contact with this pathogen was accompanied by symptoms of the disease, which in most cases was mild, did not require hospitalization, and recovery was achieved without any specific means of providing assistance. Only about 15% of infected people required hospitalization in general departments and another 5% were sent to intensive care units [23,24]. The overall mortality rate did not exceed 1-2% [25]. Thirdly, despite the rather favourable statistics of the pandemic, this invasion clearly demonstrated that the level of modern medicine does not have reliable levers of influence in case of illness and cannot guarantee a cure regardless of the initial condition of the patient. In the context of the increased number of patients with COVID-19 pneumonia, nothing more possible was proposed than to continue the widespread use of antibiotics [26-28]. In general, all medical care turned out to be at the level of symptomatic and auxiliary means. In such a situation, the prospect of getting infected and getting sick naturally provoked uncertainty in medical care and caused a feeling of fear not so much among the population as among specialists working with this

contingent of patients [29-32]. Fourthly, instead of critically assessing the existing approaches and principles of providing medical care to patients with inflammatory processes of the lungs in general and coronavirus in particular, leading specialists began to look for the reasons for failures in the political plane, thus trying to find an explanation for the ongoing professional stagnation in this area [33,34]. Against this background, the final assessment of the results of the pandemic, presented by some of the above-mentioned authors, who have now begun to prove the success of medicine in the fight against coronavirus by vaccinating the population, looks paradoxical [35]. Such an assessment, with an emphasis on disease prevention, the success of which is highly questionable due to the need for unusually frequent repetition of more advanced drugs and taking into account the results of the above-mentioned "Swedish experiment", does not contain a critical analysis of treatment measures. In other words, the most important aspect of the pandemic results is ignored, according to which the United States turned out to be the leader in the number of fatalities [36]. These facts, from the point of view of logic and the cause-and-effect relationship of the observed events, are a striking example of an inexplicable selective approach to assessing one's own professional activity. Is it necessary to further explain that such an understanding of the problem crosses out any hopes for progress in this direction? Finally, current efforts to move the solution of the AP problem from a dead point lack a comprehensive connection with the real situation in this area and do not take into account the facts without which one cannot count on the expected success. Thus, throughout the entire period of antibiotic use, there has been a radical change in the list of pathogens of this disease and its constant dynamics, which indicates the influence of this therapy on this phenomenon. Many years of attempts to learn to recognize the constant change of pathogens of AP have not been successful and the use of antibiotics continues as an empirical choice, and not a targeted appointment. The broadest stake on these drugs, regardless of the etiology of inflammation (see the pandemic experience above), has long led to the fact that their justification for prescribing to this category of patients has significantly decreased, and their effectiveness in the absence of indications raises legitimate doubts. In the present period, when viral forms of inflammation begin to prevail in diagnostics of AP, real bewilderment arises after affirmative assurances about the expected prospects of success of improved systems of bacteriological testing and further development of new forms of antibiotics [37-40]. Such statements can have, in my opinion, two possible reasons. On the one hand, this is the result of strictly automatic adherence to those unshakable stereotypes that have dominated in this section of medicine for many years without due analysis and comprehensive interpretation of the constantly occurring changes. On the other hand, such statements may well reflect lobbying of the interests of those companies that are engaged in the development and production of diagnostic systems and new generations of antibiotics. In any case and regardless of the presented assumptions, from a professional point of view, medicine has already crossed the line beyond which it becomes obvious that the nature of the pathogen does not have a decisive influence on the clinical picture of the disease and its leading signs remain regardless of the change in etiology. At the same time, without a critical analysis of the changes that have already occurred under the influence of antibiotics, it is very reckless to continue further development of this therapeutic direction. The mention of possible lobbying of the interests of various companies may well cause objections and even indignation at such suspicions. However, before being indignant at the assumption of such professional priorities, let us calmly look again at the real circumstances and changed conditions. If there is a professional understanding of what happened to the etiology of AP as a result of long-term antimicrobial exposure and how viruses turned into the leading group

of pathogens, then a logical question arises: why, realizing the causes and consequences of such a transformation, is the previous strategy so passionately supported, which led to the observed shifts, and itself no longer corresponds to the new conditions? I am deeply convinced that anyone who honestly and impartially tries to answer such a question will come to the same two conclusions as indicated above. However, supporters of further development of new generations of antibiotics have what they consider an irrefutable argument. This argument and basis is the global problem of resistant microflora. This problem has been "raised on a shield" by the WHO in recent years [39,41], declaring resistant bacteria one of the global health problems. In this regard, the question immediately arises: why has this phenomenon, the danger of which was warned about and objectively confirmed by the founders of this therapy [42,43], been passively observed for decades, without taking decisive measures to prevent it? It is unlikely that one can expect to receive an unambiguous and logical answer to this question, if already in the recent period, when all the above problems have become maximally aggravated, many specialists, noting the danger of further development of resistant microflora, at the same time unjustifiably widely prescribe antibiotics for viral pneumonia [26-28]. Today, resistant microflora is not only considered a compelling justification for developing new forms of antibiotics, but is also actively used as a reason to explain unsuccessful treatment results and fatal outcomes. And yet, if you carefully study the conclusions about the danger of resistant microflora, which have become so widespread recently, you can see that in the vast majority of cases such conclusions are unfounded declarations. In the rare publications that provide statistics on AP, when resistant strains are indicated as the causative agent, the frequency of such cases does not exceed 1-2% [44,45]. In recent publications, the authors provide indicators in relation to all observations of the disease, and not only in relation to a small group of exclusively bacterial processes. Thus, statistically, there is no particular reason to fear such microflora, since the frequency of processes with its participation is extremely low. In addition, the indicated frequency of resistant pathogens in AP is many times lower than, for example, the latent carriage of MRSA among some population groups [46-48]. Many strains of bacteria, having become resistant, have long been identified as symbionts of the body, causing no harm to it. The need to sanitize such observations with a course of antibiotics continues to be discussed to this day [49], although there is no need for this, except for the unreasonably excited fear of this microflora. No one has presented evidence that bacteria, developing their resistance to antibiotics, really become more aggressive and dangerous. So, what is the problem? This circumstance can be found only one logical explanation. While maintaining a commitment to the use of antimicrobial drugs as the main treatment, medicine suddenly clearly understood that at this stage, with the prevailing version of the essence of the disease, there are no other solutions except for the development of more advanced antibiotics. At the same time, there is no understanding that such a race to suppress bacterial pathogens does not provide a way out of the impasse. But what about the treatment of already developed viral processes? It turns out that we are observing a vicious circle of a race in which even a theoretical possibility of success is not visible. Thus, the presented analysis of the currently existing prerequisites and factors for the occurrence of AP shows that such a transformation of the general disease environment did not have a significant effect on its clinical picture, emphasizing the importance of localization of the inflammatory process and the leading role of morpho functional disorders of the affected organ, and not the type of pathogen. The so-called microbial theory of AP, finally formed under the didactic influence of antibiotics, requires immediate revision with the bringing of professional ideas into line with the basic rules and laws of medical and biological science. The

dominant role of the etiology of the disease in modern medicine should give way to pathogenetic approaches to solving this problem. Excessive professional passion for the narrowly specific action of antibiotics should be adapted to a meaningful and realistic understanding of their auxiliary role in comprehensive care for patients with AP.

Conflict of interest: the author states that he has no conflict of interest.

Reference

1. Gram C. (1884). "Über die isolierte Färbung der Schizomyceten in Schnitt- und Trockenpräparaten". *Fortschr. Med.* 2 (6): 185–89.
2. Jaccoud (1887). *Scientific American. Munn & Company.* p. 196.
3. Avery OT, Chickering HT, Cole RI, Dochez AR. (1917). Acute lobar pneumonia: prevention and serum treatment. Monographs of the Rockefeller Institute for Medical Research. Rockefeller Institute for Medical Research, New York
4. Cole RI. (1927–1928). Acute pulmonary infections. De La Mar Lectures. Williams & Wilkins, Baltimore
5. Sutliff WD, Finland M. (1933). The significance of the newly classified types of pneumococci in disease: types IV to XX inclusive. *JAMA* 101:1289–1295.
6. Heffron R. (1939). Pneumonia, with special reference to pneumococcus lobar pneumonia. Cambridge: *Harvard University Press.*
7. Small JT. (1948) A short history of the pneumococcus with special reference to lobar pneumonia. *Edinb Med J.*;55(3):129–41.
8. Shoar, S., Musher, D.M. (2020). Etiology of community-acquired pneumonia in adults: a systematic review. *Pneumonia* 12, 11.
9. Jain S, Self WH, Wunderink RG, Fakhran S, Balk R, Bramley AM, et al. (2015). CDC EPIC Study Team. Community-acquired pneumonia requiring hospitalization among U.S. adults. *N Engl J Med.*; 373:415–427.
10. Shorr AF, Zilberberg MD, Micek ST, Kollef MH. (2017). Viruses are prevalent in non-ventilated hospital-acquired pneumonia. *Respir Med.*; 122:76–80.
11. Palomeque, A., Cilloniz, C., Soler-Comas, A., Canseco-Ribas, J., Rovira-Ribalta, N., et al. (2024). A review of the value of point-of-care testing for community-acquired pneumonia. *Expert Review of Molecular Diagnostics*, 1–14.
12. Pick H, Daniel P, Rodrigo C, Bewick T, Ashton D, et al. (2020). Pneumococcal serotype trends, surveillance and risk factors in UK adult pneumonia, 2013-18. *Thorax.*;75(1):38-49.
13. R. Isturiz, L. Grant, S. Gray et al. (2021). Expanded Analysis of 20 Pneumococcal Serotypes Associated With Radiographically Confirmed Community-Acquired Pneumonia in Hospitalized US Adults, *Clinical Infectious Diseases*, ciab375,
14. Thompson GR3rd, Le T, Chindamporn A, et al (2021). Global guideline for the diagnosis and management of the endemic mycoses: an initiative of the European Confederation of Medical Mycology in cooperation with the International Society for Human and Animal Mycology. *Lancet Infect Dis.* 21: e364–e374.

15. Tavleen Kaur Jaggi, Ritesh Agarwal, Pei Yee Tiew et al (2024). Fungal lung disease. *European Respiratory Journal* ;64(5): 2400803;
16. John H, Hodges MD (1989) Wagner, MD, Frederick B (ed.). "Thomas Jefferson University: Tradition and Heritage". Jefferson Digital Commons. Part III, Chapter 9: Department of Medicine. p. 253
17. (2004). WHO Revised global burden of disease 2002 estimates.
18. Ruuskanen O, Lahti E, Jennings LC, Murdoch DR (2011). *Viral pneumonia. Lancet* 377 (9773):1264-75
19. Visseaux B et al (2017). Prevalence of respiratory viruses among adults, by season, age, respiratory tract region and type of medical unit in Paris, France, from 2011 to 2016. *PLoS One.*;12(7): e0180888.
20. Shah MM et al (2022). Seasonality of common human coronaviruses, United States, 2014–2021. *Emerg Infect Dis.*;28(10):1970-6.
21. Björkman A, Gisslén M, Gullberg M, Ludvigsson J (2023). The Swedish COVID-19 approach: a scientific dialogue on mitigation policies. *Front Public Health.*; 11:1206732.
22. Andersson, F. N. G., & Jonung, L. (2024). The Covid-19 lesson from Sweden: Don't lock down. *Economic Affairs*, 44(1), 3–16.
23. Oran DP, Topol EJ (2020). Prevalence of Asymptomatic SARS-CoV-2 Infection: A Narrative Review. *Ann Intern Med.*;173(5):362-367.
24. Wu Z, McGoogan JM (2020). Characteristics of and Important Lessons From the Coronavirus Disease 2019 (COVID-19) Outbreak in China: Summary of a Report of 72 314 Cases From the Chinese Center for Disease Control and Prevention. *JAMA.*;323(13):1239–1242.
25. Mathieu E, Ritchie H, Rodés-Guirao L, Appel C, Giattino C, Hasell J, et al. (2020–2024). "Coronavirus Pandemic (COVID-19)". Our World in Data.
26. B.D. Huttner, G. Catho, J.R. Pano-Pardo et al. (2020). COVID-19: don't neglect antimicrobial stewardship principles! *Clinical Microbiology and Infection*, Vol 26, Issue 7, P808-810.
27. B.Beovic, 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*, dkaa326,
28. 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 [published online ahead of print, 2020 May 2]. *Clin Infect Dis*; ciaa 530.
29. R. E. Leiter (2020). Reentry. *NEJM*.
30. J. N. Rosenquist (2020). The Stress of Bayesian Medicine — Uncomfortable Uncertainty in the Face of Covid-19. *NEJM, N Engl J Med*; 384:7-9.
31. Salisbury H (2020) Helen Salisbury: What might we learn from the covid-19 pandemic? *BMJ* 368:m1087.
32. Oliver D (2020) David Oliver: Conveyor belt medicine. *BMJ* 368:m162.
33. Editors (2020). Dying in a Leadership Vacuum. *N Engl J Med.*;383(15):1479-1480.
34. Mareiniss DP (2021). The Emperor Has No Clothes - Medical Journals and Experts Must Stand Up and Condemn the Federal Pandemic Response. *Am J Emerg Med.*; 45:666.
35. Lessons for a Pandemic (Audio Interview: H. Fineberg and E. Rubin) (2023). *N Engl J Med*; 388: e67
36. Nuzzo JB, Ledesma JR. (2023). Why did the best prepared country in the world fare so poorly during COVID? *J Econ Perspect*; 37:3-22
37. Kyriazopoulou E, Karageorgos A, Liaskou-Antoniou L, et al. (2021). BioFire® FilmArray® pneumonia panel for severe lower respiratory tract infections: subgroup analysis of a randomized clinical trial. *Infect Dis Ther*; 10:1437-1449.
38. Enne VI, Aydin A, Baldan R INHALE WP1 Study Group, et al (2022). Multicentre evaluation of two multiplex PCR platforms for the rapid microbiological investigation of nosocomial pneumonia in UK ICUs: the INHALE WP1 study. *Thorax*; 77:1220-1228.
39. World Health Organization (2023). *Antimicrobial resistance*.
40. Madhavi Thara (2024). "Antibiotic Stewardship". *Medicon Medical Sciences* 6.3: 01-02.
41. (2021). WHO. Antimicrobial resistance.
42. Abraham EP, Chain E (1940). An enzyme from bacteria able to destroy penicillin. 1940. *Rev Infect Dis*. 1988;10(4):677–678.
43. Fleming, A. (1945). "The Nobel Prize in Physiology or Medicine 1945 - Penicillin: Nobel Lecture". NobelPrize.org. Retrieved 17 October 2020.
44. Ding H, Mang NS, Loomis J, Ortwine JK, Wei W, et al. (2024). Incidence of drug-resistant pathogens in community-acquired pneumonia at a safety net hospital. *Microbiol Spectr* 12: e00792-824.
45. Sakamoto, Y., Yamauchi, Y., Jo, T. et al. (2021). In-hospital mortality associated with community-acquired pneumonia due to methicillin-resistant *Staphylococcus aureus*: a matched-pair cohort study. *BMC Pulm Med* 21, 345.
46. Albrich W.C., Harbarth S (2008). Health-care workers: Source, vector, or victim of MRSA? *Lancet Infect. Dis*; 8:289–301.
47. Aubry-Damon H., Grenet K., Ndiaye-Sall P., Che D., Corderio E., et al. (2004). Antimicrobial resistance in commensal flora of pig farmers. *Emerg. Infect. Dis.*; 10:873–879.
48. Graveland H., Wagenaar J.A., Heesterbeek H., Mevius D., van Duikeren E., et al. (2010). Methicillin Resistant *Staphylococcus aureus* ST398 in Veal Calf Farming: Human MRSA Carriage Related with Animal Antimicrobial Usage and Farm Hygiene. *PLoS ONE*. 2010;5: e10990.
49. C. Liu, and M. Holubar (2022). Should a MRSA Nasal Swab Guide Empiric Antibiotic Treatment? *NEJM Evid*;1(12)



This work is licensed under Creative Commons Attribution 4.0 License

To Submit Your Article Click Here:

Submit Manuscript

DOI:10.31579/2768-0487/188

Ready to submit your research? Choose Auctores and benefit from:

- fast, convenient online submission
- rigorous peer review by experienced research in your field
- rapid publication on acceptance
- authors retain copyrights
- unique DOI for all articles
- immediate, unrestricted online access

At Auctores, research is always in progress.

Learn more <https://auctoresonline.org/journals/journal-of-clinical-and-laboratory-research>