

The Factual Aspects of Acute Nonspecific Inflammation of Lung Tissue and their Role in Addressing this Problem

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Abstract

Acute nonspecific inflammation of lung tissue—or acute pneumonia (AP)—has remained a major, pressing issue in recent decades, characterized by a lack of treatments with proven efficacy and rising mortality rates. An analysis of the evolution of therapeutic approaches reveals the decisive impact that the advent of antibiotics had on prevailing concepts of the disease; this factor shaped the subsequent development of the professional understanding of AP's nature. Today, alongside the negative consequences of the “antibiotic era,” the primary obstacle to resolving this issue lies in the expert community's views regarding its fundamental nature.

Keywords: *Acute Inflammation; Nonspecific Etiology; Antibiotics; Pathogenesis; Changes in the Etiology of the Disease; The Exclusion of Antibiotics; Distortions in Professional Training*

Abbreviations

AP: Acute Pneumonia; CAP: Community-Acquired Pneumonia; COVID-19: Coronavirus Disease 2019; MRSA: Methicillin-Resistant *Staphylococcus aureus*; SARS-CoV-2: Coronavirus Pandemic

Introduction to the Core of the Problem

Acute nonspecific inflammation of lung tissue, better known as “acute pneumonia” (AP), is one of the oldest diseases known to medicine. Despite its long history, AP remains a formidable ailment to this day. Although the overall incidence of AP declined from 22.9 to 9.9 cases per 100,000 population over the quarter-century from 1999 to 2023 [1], the mortality rate among hospitalized patients rose to 22-30%, and the disease remains a leading cause of death worldwide across all age groups [2,3]. Many experts acknowledge the effectiveness of existing methods for preventing and treating AP, attributing the rise in fatal outcomes primarily to insufficient vaccination coverage, nutritional issues, and a lack of measures to improve environmental conditions, particularly in low- and middle-income countries [4].

Unfortunately, such expert explanations fail to reflect the obvious circumstances surrounding the onset and progression of AP in the modern era. Despite significant changes in the broader context, the conceptual approach to CAP has essentially remained at an early stage: it continues to rely on the premise that a bacterial pathogen is the primary cause of the disease and that antibiotics are the cornerstone of treatment [5]. Without delving into an analysis of fundamental shifts in AP etiology, the medical community has focused on preserving and maintaining the efficacy of antibacterial therapy, which has, of course, undergone constant adjustment, yet the intensity of these empirical efforts remains unchanged to this day [6-8].

In light of the obvious facts, current explanations for the high mortality rate associated with this disease appear to be a clear case of self-deception, steering research away from the true factors determining the severity of the illness. While noting that such figures are typical of developing countries, experts studiously avoid explaining why the United States, despite possessing a healthcare system that leads in key areas such as funding, innovation, and workforce training, suffered a catastrophic failure during the pandemic, becoming the undisputed leader in deaths from COVID-19-related pneumonia [9,10].

An analysis of the current situation in this field reveals a complex interplay of factors and long-established scientific axioms that are often overlooked in the treatment of patients with AP. In recent years, the author has repeatedly published the results of such studies and summarized them in monographs. These materials are publicly available. This article attempts to briefly outline the most important circumstances and natural mechanisms that must be taken into account, as failure to do so will leave the problem of AP unresolved.

Fundamental knowledge and obvious facts that do not receive due attention when considering this problem

Emergence of changes in the etiology of AP

In the pre-antibiotic era, which spanned several decades, the pneumococcus was the causative agent of pneumonia in 95% or more of cases, regardless of the time or location of the study [11-15]. Although the accuracy and reliability of data from that period may be open to question today, no other information is available to us. Therefore, these figures and their consistency should be accepted as a baseline, especially given that the same methodologies were employed in subsequent bacteriological studies.

Even during the early years of clinical antibiotic use, findings from preclinical studies were confirmed showing that pneumococci were less resistant to penicillin than staphylococci [16,17]. This characteristic of the new therapy led to an increase in the proportion of staphylococci among the causative agents of acute pneumonia; by the early 1960s, staphylococci had not only become the predominant pathogen (especially in children) but—according to some statistical data—were responsible for nearly 100% of cases of purulent-necrotic diseases, including complicated forms of acute pneumonia [18].

The staphylococcal infection outbreak took on the characteristics of a bacterial pandemic and became the first serious consequence of antibiotic use, an event whose underlying causes were, unfortunately, never fully elucidated. Subsequent developments revealed a periodic shift in the causative agents of AP, while the need to maintain therapeutic efficacy drove the development and release of new drugs, the majority of which had already been created by the early 1970s [19].

Having failed to achieve a sustained therapeutic effect through the development of new antibiotic formulations, medical specialists shifted their focus to the earliest possible diagnosis of the causative agents of AP. The goal was to enable the early initiation of targeted antimicrobial therapy; however, traditional methods of direct and indirect bacteriological diagnosis also failed to yield the expected results. In recent years, the efforts of researchers and developers have focused on creating advanced microbiological diagnostic systems. Despite optimistic conclusions regarding the use of these rapid diagnostic methods, there have been no fundamental changes in AP treatment outcomes [20,21]. There are reasons for this.

- First, the strategy for addressing this problem has retained its fundamental principle for years: the pathogen itself is considered the root cause of the disease, and antibiotics remain the treatment of choice [5].
- Second, recent decades have seen a significant rise in cases of viral pneumonia, which has lately become the predominant form within this disease classification [22-24].
- Third, despite substantial progress in microbiological diagnostics, the etiology of community-acquired pneumonia remains undetermined in 60% or more of patients [22,23].

- Finally, while continuing to focus heavily on identifying the causative agents of bacterial pneumonia, many specialists assert that **Streptococcus pneumoniae** has once again become the leading etiological agent [25-27]. Such conclusions lack rigorous critical analysis; rather, they are driven by a desire to promote pneumococcal vaccination. Indeed, can one speak of a leading role for **S. pneumoniae** when positive microbiological test results are obtained in only about 40 - 50% of cases (amid the increasingly frequent predominance of viruses), and this microorganism accounts for merely 20 - 40% of identified pathogens?

Changes in the etiology of AP as a cause of reduced antibiotic efficacy

In the pre-antibiotic era, AP was not classified as an infectious or contagious disease, despite the fact that the pneumococcus played a key role in its etiology, a fact that gave the bacterium its specific name. However, with the development of microbiology, acute inflammatory lesions of the lung tissue were, quite justifiably, reclassified as forms of nonspecific inflammation, highlighting the ability of a multitude of other bacteria to cause the identical disease. The use of antibiotics disrupted the established equilibrium, triggering continuous changes in the spectrum of the primary pathogens causing AP. Nature's response to this unexpected aggression was predictably aimed at creating conditions capable of neutralizing the cause of the resulting imbalance.

For a long time, the pharmaceutical industry has sought to adapt antimicrobial therapy methods, attempting to target the etiology of inflammatory processes and thereby assist physicians in the primary task of maintaining therapeutic efficacy. However, this "race" could not continue indefinitely; recent decades have seen the emergence of a new situation characterized by a rise in the incidence of viral pneumonias, which were previously considered rare [28,29]. Despite efforts to preserve the efficacy of antibacterial therapy, specialists have failed to fundamentally revise the strategy for addressing this issue. Above all, the fact that changes in disease etiology are a serious adverse consequence of antibiotic use is being overlooked.

Viruses and fungi have long been recognized as potential etiological factors in AP, yet for a long time, their role held little practical significance. In the past, such cases of inflammation were extremely rare. Today, however, viral pneumonias have become a leading cause of AP, ceasing to be the exception to the rule. Against this backdrop, other circumstances give rise to both concern and bewilderment. Above all, antibiotics remain the primary treatment method, a fact clearly demonstrated at the onset of the SARS-CoV-2 pandemic, when the vast majority of patients with coronavirus pneumonia (in the absence of bacterial coinfection) continued to be treated according to standard protocols [30-33]. And although the reasons for this shift in etiology might be attributed to global warming [34] or even suspected to be the result of deliberate provocation [35], the most plausible and logical explanation, a side effect of antibiotic use that unfolded right before the eyes of specialists and necessitated constant adjustments to treatment approaches, is not even being discussed.

In addition to the already observed global rise in viral diseases, recent epidemiological surveillance data from the United States indicate a sharp increase in the incidence of inflammatory conditions of fungal etiology [36]. These observations were made over a short two-year period and further attest to the increasing aggressiveness of these pathogens.

Thus, the process of adaptation to prolonged antibiotic exposure has altered the etiological profile of AP; specifically, the need for antimicrobial agents has significantly decreased. This illustrative example confirms the axiom that disrupting the balance of natural factors cannot lead to a stable state. In this instance, the observed changes clearly demonstrate how the long-term use of antibiotics ultimately resulted in their displacement from the ranks of primary therapeutic agents.

Why is antibiotic-resistant microflora considered a cause of treatment failure in acute pneumonia?

The emergence of antibiotic-resistant microbial strains became evident soon after these new treatments entered widespread clinical practice. Initially, however, the development of this side effect did not raise concerns among specialists regarding the potential consequences of its progression. The medical community was guided by the "microbial" concept of antibiotic therapy and was impressed

by the brilliant results achieved during the early stages of using these drugs. Consequently, subsequent efforts focused primarily on preserving and maintaining the therapeutic efficacy of antibiotic treatment.

The process of achieving this goal gradually became the primary focus of therapeutic efforts in treating patients with nonspecific pneumonia. Over time, as generations of physicians succeeded one another, these conceptual principles became increasingly rigid and entrenched. In recent decades, a strictly defined approach to selecting treatment strategies for AP has become established in clinical guidelines. Initially, a list of pathogens considered most likely at the time was provided. This list was periodically revised to reflect changes in the etiology of AP. Subsequently, antibiotics deemed most suitable for the given period were recommended. These recommendations formed the basis of AP treatment, while all other drugs and therapeutic measures were relegated to the category of adjunctive therapy.

Despite all efforts, it proved impossible to restore the initial efficacy of antibiotics; it continued to decline slowly but steadily. Nevertheless, the measures taken made it possible to maintain antimicrobial therapy for a long time as the primary, and, in effect, the only, treatment method. This approach shaped the mindset of subsequent generations of physicians, who became accustomed to relying exclusively on antibiotics from the outset when treating AP. Changes in the disease's etiology, accompanied by the emergence of rare pathogens, and the resulting implications for addressing the issue failed to receive adequate attention or analytical scrutiny.

When an increase in the incidence of viral pneumonia was observed at the turn of the millennium, a search began for antiviral drugs capable of replacing antibiotics. To date, these efforts have not yielded the desired results, and antibiotics remain the primary treatment method. The decline in therapeutic efficacy, observed against the backdrop of a radically altered etiology of AP, led to the conclusion that the presence of resistant microflora, along with other factors, a major cause of treatment failure. This view quickly gained widespread acceptance and became the standard explanation for the challenges encountered in treating AP.

Over the past two decades, there has been an increase in publications asserting, often without supporting data, that resistant microflora negatively affect treatment outcomes. The vast majority of such reports provide only general estimates, lacking specific figures characterizing the prevalence of these cases. Naturally, this discussion concerns bacterial pneumonia and antibacterial therapy. In the few studies presenting such data, the proportion of resistant microflora within the etiological structure of AP does not exceed 1 - 2% [37,38]. At the same time, established facts, such as the prevalence of resistant strains among the healthy population, often fail to receive due attention from specialists. For instance, MRSA (methicillin-resistant *Staphylococcus aureus**), the most active and widespread of the resistant strains, is detected as part of the commensal microflora in 2% of healthy individuals [39], whereas in certain occupational groups, this figure reaches 6 - 10% or higher [40-44]. This phenomenon has not yet been convincingly explained.

Despite the aforementioned facts, WHO experts, without providing further clarification, declared the issue of resistant microflora a global crisis. Notably, this declaration coincided with the peak of the SARS-CoV-2 pandemic, a time when the medical community's capacity to treat pneumonia patients was viewed with skepticism. A phenomenon that had existed throughout the antibiotic era, yet had failed to garner sufficient attention or elicit an adequate response, suddenly transformed into a major global crisis. A wide range of specialists readily embraced this concept, as many continue to pin their hopes on improving antimicrobial therapy and actively seek the most suitable drugs. However, an analysis of accumulated data regarding the emergence of resistant microflora and fundamental shifts in the etiology of AP reveals a direct link between these phenomena and reduced antibiotic efficacy; yet, this link is clinically significant only in cases where antimicrobial agents serve as the primary treatment modality. The narrow focus and artificial nature of this "global crisis" surrounding resistant microflora point to serious conceptual distortions in the professional understanding of the issue at hand.

Uniqueness of the pathogenesis of AP

AP is a unique inflammatory process occurring within the pulmonary circulation under specific pathophysiological conditions. Lung tissue is essentially formed by a network of pulmonary vessels, whereas the blood supply to other organs and tissues is provided by

the systemic circulation. It is well established that blood pressure in the pulmonary vessels is approximately eight times lower than in the systemic circulation [45]. Nevertheless, the body maintains this vital gradient even under extreme conditions, ensuring a balance of cardiac output and rhythm between the right and left sides of the heart [46]. Any disruption to the equilibrium of these parameters is incompatible with life.

The information presented is widely accepted and elucidates the functional characteristics of the circulatory system. However, a logical question arises: where and how are these crucial characteristics taken into account when determining treatment principles for AP? It should be noted that efforts to address this issue remain focused on the causative agent and rely on the microbial concept of the disease. Consequently, the severe condition of AP patients is still attributed to the same factors associated with any other inflammatory process. Nevertheless, the physiology of the pulmonary circulation leaves no doubt that the pathogenesis of AP differs fundamentally from that of inflammation in other locations and plays a pivotal role in addressing the problem of pneumonia [47].

An analysis of data on the topic under consideration indicates that insufficient attention to the aforementioned results has led to a number of misconceptions regarding the nature and dynamics of this pathological process. This applies primarily to the principles for assessing the condition of patients in this category. The mechanism of the inflammatory process in the lungs in this case can be described as follows.

Acute inflammation is primarily an active vascular response accompanied by a fundamental reorganization of blood flow within the affected section of the vasculature. These changes lead to increased pressure in the pulmonary vessels. Such shifts carry serious consequences: a rise in pulmonary circulation pressure of just 5 mmHg triggers interstitial pulmonary edema, whereas an increase of 10 mmHg leads to pronounced pulmonary edema [46]. Immediate correction of these abnormalities is essential, as a life-threatening condition develops.

In such situations, a natural mechanism is required to rapidly restore the balance of these ratios. Although this mechanism was discovered nearly a century ago [48], its role and significance in the pathogenesis of the disease long remained virtually overlooked. Given that AP initially causes a rise in pressure within the pulmonary circulation, while compensatory mechanisms in the systemic circulation (including the so-called unloading reflex [48]) are rapidly activated to protect the lungs and maintain vital central hemodynamic parameters, deviations in systemic arterial pressure (when assessed using standard diagnostic criteria for severity and sepsis in AP patients) lead to a distortion of statistical data regarding the clinical presentation of the disease.

Currently, the factors determining the critical condition of patients with AP are viewed in the same light as the mechanisms of inflammatory processes in other organs and tissues: the development of AP is generally not considered a process that follows a unique course [49,50]. This major misconception has led to unexpected conclusions in modern medicine. First, patients with AP account for at least half of all sepsis cases [22,51,52]. Second, according to generally accepted criteria, the abnormalities classified as sepsis are actually manifestations of a compensatory response. Although the lack of correlation between the severity of the pathological process and the type of causative agent has been repeatedly and comprehensively confirmed over the years, this factor continues to influence clinical thinking, largely due to its association with antimicrobial therapy [47]. Finally, equating the mechanisms of AP development with those of other inflammatory diseases dictates the use of standard supportive therapy.

Currently, intravenous infusions serve as the primary method for preventing and correcting circulatory disturbances associated with septic complications of inflammatory processes; the intensity of such infusion therapy increases significantly as the observed disturbances worsen [49-51]. Although this therapeutic approach may be effective in cases of localized peripheral inflammation, in patients with progression of the underlying disease [47]. Recent attempts to study the impact of intravenous infusions without regard for the nature of

the primary pathology have failed, and inevitably will fail, to yield AP, such therapy has the opposite effect, contributing to the concrete results [53]. However, if these specific aspects of AP pathogenesis are ignored, attempts to treat the disease will continue to be made “blindly,” resulting in severe consequences for many patients.

Although the analysis presented here does not yet allow its findings to serve as direct recommendations for treating critically ill patients with AP (as such recommendations currently lack official status), the published data help explain why the treatment methods currently employed for this disease have not yielded successful results.

The modern didactic climate for addressing the AP problem

In the modern world, physicians increasingly encounter situations where antibiotics, traditionally regarded as the cornerstone of treatment, prove unnecessary. However, calls and attempts to restore the former efficacy of antimicrobial agents [54,55] are a source of far greater concern. Such statements and publications indicate that this group of specialists has not fully grasped the lessons of the antibiotic era, including all the associated consequences and side effects. Moving to a new stage of therapeutic intervention in natural processes and interrelationships without first studying and evaluating the experience gained constitutes a serious error.

A modern understanding of the nature of AP requires a fundamental shift in perspective, one that takes into account both the “forgotten” classical axioms of medical science and the vast body of data accumulated during the antibiotic era. The challenge lies in the fact that the prevailing viewpoint, which is in need of revision, not only dominates the field but is also enshrined in various documents adopted at a wide range of levels. Nevertheless, it is crucial to grasp the scale of this task from the outset; otherwise, a successful solution to the problem will remain out of reach.

Conclusion

In recent decades, AP has remained a disease characterized by a high incidence of severe complications, suboptimal treatment efficacy, and high mortality rates. An analysis of available data clearly reveals the existence of deeply rooted and persistent misconceptions regarding the nature of AP, views that emerged with the advent of antibiotics and have endured despite contradictory clinical evidence. Successfully addressing this issue today depends primarily on overcoming these long-standing dogmatic distortions.

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Conflict of Interest

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Bibliography

1. Obianyo CM., *et al.* “Trends and disparities in pneumonia-related mortality in the U.S. population: A nationwide analysis using the CDC WONDER data”. *Cureus* 17.5 (2025): e83371.
2. Karim R., *et al.* “Clinical findings and radiological evaluation of WHO-defined severe pneumonia among hospitalized children”. *Cureus* 15.1 (2023): e33804.
3. Dion CF and Ashurst JV. “*Streptococcus pneumoniae*”. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing (2026).
4. CDC. Pneumonia. Prevention and Control. Pneumonia. CDC (2023).

5. Podolsky SH. "The changing fate of pneumonia as a public health concern in 20th-century America and beyond". *American Journal of Public Health* 95.12 (2005): 2144-2154.
6. A Putot., *et al.* "Comprehensive management of pneumonia in older patients". *European Journal of Internal Medicine* 135 (2025): 14-24.
7. R Cavallazzi and JA Ramirez. "Definition, epidemiology, and pathogenesis of severe community-acquired pneumonia". *Seminars in Respiratory and Critical Care Medicine* 45.2 (2024): 143-157.
8. Pickens CI., *et al.* "Microbiology of severe community-acquired pneumonia and the role of rapid molecular techniques". *Seminars in Respiratory and Critical Care Medicine* 45.2 (2024): 158-168.
9. Nuzzo JB and Ledesma JR. "Why did the best prepared country in the world fare so poorly during COVID?" *Journal of Economic Perspectives* 37 (2023): 3-22.
10. Mathieu E., *et al.* "Coronavirus Pandemic (COVID-19)". Our World in Data (2025).
11. Avery OT., *et al.* "Acute lobar pneumonia: prevention and serum treatment". Monographs of the Rockefeller Institute for Medical Research. Rockefeller Institute for Medical Research, New York (1917).
12. Cole RI. "Acute pulmonary infections". De La Mar Lectures. Williams & Wilkins, Baltimore (1927-1928).
13. Sutliff WD and Finland M. "The significance of the newly classified types of pneumococci in disease: types IV to XX inclusive". *Journal of the American Medical Association* 101.17 (1933): 1289-1295.
14. Heffron R. "Pneumonia, with special reference to pneumococcus lobar pneumonia". Cambridge: Harvard University Press (1939).
15. Small JT. "A short history of the pneumococcus with special reference to lobar pneumonia". *Edinburgh Medical Journal* 55.3 (1948): 129-141.
16. Rammelkamp T. "Resistance of *Staphylococcus aureus* to the action of penicillin". *Experimental Biology and Medicine* 51.3 (1942): 386-389.
17. Kirby WM. "Extraction of a highly potent penicillin inactivator from penicillin resistant staphylococci". *Science* 99.2579 (1944): 452-453.
18. "MRSA History Timeline: The First Half-Century, 1959-2009". The University of Chicago Medical Center (2010).
19. Aminov RI. "A brief history of the antibiotic era: lessons learned and challenges for the future". *Frontiers in Microbiology* 1 (2010): 134.
20. Kyriazopoulou E., *et al.* "BioFire® FilmArray® pneumonia panel for severe lower respiratory tract infections: subgroup analysis of a randomized clinical trial". *Infectious Diseases and Therapy* 10.3 (2021): 1437-1449.
21. Enne VI., *et al.* "Multicentre evaluation of two multiplex PCR platforms for the rapid microbiological investigation of nosocomial pneumonia in UK ICUs: the INHALE WP1 study". *Thorax* 77.12 (2022): 1220-1228.
22. Cilloniz C., *et al.* "Pure viral sepsis secondary to community-acquired pneumonia in adults: risk and prognostic factors". *Journal of Infectious Diseases* 220.7 (2019): 1166-1171.
23. B Ponnuthurai., *et al.* "Etiological and clinical profile of virus-associated community-acquired pneumonia in adults". *Chest Infections* 157.6 (2020): A55.

24. 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.
25. Musher DM., *et al.* "Evolving understanding of the causes of pneumonia in adults, with special attention to the role of pneumococcus". *Clinical Infectious Diseases* 65.10 (2017): 1736-1744.
26. Olarte L., *et al.* "Higher-valent pneumococcal conjugate vaccines—perspective for 2026". *JAMA Internal Medicine* 185.12 (2025): 1493-1494.
27. Lansbury L., *et al.* "Pneumococcal pneumonia trends in adults hospitalised with community-acquired pneumonia over 10 years (2013-2023) and the role of serotype 3". *Thorax* 80.2 (2025): 86-96.
28. WHO Revised global burden of disease 2002 estimates (2004).
29. Ruuskanen O., *et al.* "Viral pneumonia". *Lancet* 377.9773 (2011): 1264-1275.
30. BD Huttner., *et al.* "COVID-19: don't neglect antimicrobial stewardship principles!" *Clinical Microbiology and Infection* 26.7 (2020): 808-810.
31. B Beovic., *et al.* "Antibiotic use in patients with COVID-19: a 'snapshot' Infectious Diseases International Research Initiative (ID-IRI) survey". *Journal of Antimicrobial Chemotherapy* 75.11 (2020): 3386-3390.
32. Rawson TM., *et al.* "Bacterial and fungal co-infection in individuals with coronavirus: A rapid review to support COVID-19 antimicrobial prescribing". *Clinical Infectious Diseases* 71.9 (2020): 2459-2468.
33. Lipman M., *et al.* "SARS-CoV-2 pandemic: clinical picture of COVID-19 and implications for research". *Thorax* 75.8 (2020): 614-616.
34. CDC. National Center for Emerging and Zoonotic Infectious Diseases (March 21, 2024). Climate and Infectious Diseases.
35. LO Gostin and GK Gronvall. "The origins of covid-19 - why it matters (and why it doesn't)". *New England Journal of Medicine* 388.35 (2023): 2305-2308.
36. Anderer S. "CDC reports increase in *Candida auris* infections". *Journal of the American Medical Association* 336.9 (2026): 728.
37. Ding H., *et al.* "Incidence of drug-resistant pathogens in community-acquired pneumonia at a safety net hospital". *Microbiology Spectrum* 12.8 (2024): e00792-24.
38. Sakamoto Y., *et al.* "In-hospital mortality associated with community-acquired pneumonia due to methicillin-resistant *Staphylococcus aureus*: a matched-pair cohort study". *BMC Pulmonary Medicine* 21.1 (2021): 345.
39. CDC, Saving Lives. Protecting People. Methicillin-resistant *Staphylococcus aureus* (MRSA). Healthcare Settings (2023).
40. Albrich WC and Harbarth S. "Health-care workers: Source, vector, or victim of MRSA?" *Lancet Infectious Diseases* 8.5 (2008): 289-301.
41. Aubry-Damon H., *et al.* "Antimicrobial resistance in commensal flora of pig farmers". *Emerging Infectious Diseases* 10.5 (2004): 873-879.
42. Graveland H., *et al.* "Methicillin resistant *Staphylococcus aureus* ST398 in veal calf farming: human MRSA carriage related with animal antimicrobial usage and farm hygiene". *PLoS ONE* 5.6 (2010): e10990.
43. Giri S., *et al.* "Prevalence of methicillin-resistant *Staphylococcus aureus* carriage among healthcare workers in South Asia in non-outbreak settings: A systematic review and meta-analysis". *American Journal of Infection Control* 51.2 (2022): 184-193.

44. Azzam A., *et al.* "Prevalence, antibiogram, and risk factors of methicillin-resistant *Staphylococcus aureus* (MRSA) asymptomatic carriage in Africa: a systematic review and meta-analysis". *BMC Infectious Diseases* 25.1 (2025): 505.
45. Blood pressure.
46. Olivia Vynn. *Cardiology secrets*. Chapter 41. Adair Edition: 2, illustrated Published by Elsevier Health Sciences, ISBN 1-56053-420-6, 978-1-56053-420 (2001): 210.
47. I Klepikov. *Myths, legends and real facts about acute lung inflammation*. Cambridge Scholars Publishing. ISBN: 1-0364-0293-2, ISBN13: 978-1-0364-0293-8 (2024): 338.
48. Schwiegk H. "Der Lungenentlastungsreflex". *Pflügers Archiv – European Journal of Physiology* 236 (1935): 206-219.
49. Singer M., *et al.* "The third international consensus definitions for sepsis and septic shock (Sepsis-3)". *Journal of the American Medical Association* 15.8 (2016): 801-810.
50. Jungian Chen., *et al.* "Performance of CURB-65, PSI, and APACHE-II for predicting COVID-19 pneumonia severity and mortality". *European Journal of Inflammation* 19 (2021).
51. Guarino M., *et al.* "2023 update on sepsis and septic shock in adult patients: Management in the emergency department". *Journal of Clinical Medicine* 12.9 (2023): 3188.
52. Torres LK., *et al.* "Attributable mortality of acute respiratory distress syndrome: a systematic review, meta-analysis and survival analysis using targeted minimum loss-based estimation". *Thorax* 76.12 (2021): 1176-1185.
53. The National Heart, Lung, and Blood Institute Prevention and Early Treatment of Acute Lung Injury Clinical Trials Network., *et al.* "Early restrictive or liberal fluid management for sepsis-induced hypotension". *New England Journal of Medicine* 388.6 (2023): 499-510.
54. World Health Organization. *Antimicrobial resistance* (2023).
55. Madhavi Thara. "Antibiotic Stewardship". *Medicon Medical Sciences* 6.3 (2024): 01-02.

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