HSPI The Open Access Publisher

JCICM JOURNAL OF CLINICAL INTENSIVE CARE AND MEDICINE
ISSN 2639-6653

Research Article

Self-devaluation of Antibiotics as a Side Effect of Their Use

Igor Klepikov*

Retired, Renton, WA, USA

Abstract

Despite their long history of use, antibiotics remain the primary treatment for inflammatory diseases. However, it is often overlooked that these drugs are capable of killing only certain types of bacteria and do not directly affect inflammatory mechanisms. Moreover, long-term antimicrobial therapy leads to the development of side effects, including changes in the etiology of acute pneumonia. The prolonged and methodical eradication of common pneumonia pathogens forces nature to gradually replace them with agents unrelated to the spectrum of antimicrobial activity. It is this circumstance, and not microbial resistance, as is currently believed, that is the main reason for the decline in antibiotic effectiveness. This conclusion is supported by the prevalence of viral forms of pneumonia, while only a few resistant strains of pathogens are identified among the small number of bacterial infections. This latter statement indicates the need to reconsider the concept of disease and remove antibiotics from the list of miracle cures.

More Information

***Corresponding author:** Igor Klepikov, MD, Professor, Retired, Renton, WA, USA, Email: igor.klepikov@yahoo.com

Submitted: March 05, 2026
Accepted: March 11, 2026
Published: March 12, 2026

Citation: Klepikov I. Self-devaluation of Antibiotics as a Side Effect of Their Use. J Clin Intensive Care Med. 2026; 11(1): 001-005. Available from: https://dx.doi.org/10.29328/journal.jcicm.1001056

Keywords: Acute pneumonia; Etiology; Antibiotics; Side effects; Pathogenesis; Conception of disease

Abbreviations: AP: Acute Pneumonia; MRSA: Methicillin-Resistant Staphylococcus Aureus; SARS-CoV-2: Severe Acute Respiratory Syndrome Coronavirus 2; WHO: World Health Organization

Introduction

Antibiotics, rightfully considered one of the most remarkable discoveries of the 20th century and having saved millions of lives, continue to be the leading, and often the only, treatment for inflammatory diseases. The biological origin of these drugs and their selective action on associated but uncontrolled microorganisms naturally prompted nature to use its capabilities to correct the consequences of antibiotic use. The rapid development of microbial resistance to these drugs was noted even in the preclinical stage of their research [1,2], and the first side effects began to appear soon after the advent of antibiotics, the most striking example being acute pneumonia (AP).

Discussion

In the pre-antibiotic era, Streptococcus pneumoniae was consistently the predominant causative agent of AP, accounting for 95% or more of cases, regardless of region and time of study [3-7]. However, soon after the introduction of this therapy into widespread practice, new types of drugs were needed due to penicillin's ineffectiveness against a number of microorganisms, the proportion of which began to increase among the causative agents of the disease. Moreover, practice has shown that among host symbionts capable of causing AP, Staphylococcus aureus was distinguished by a more rapid ability to develop resistance [8], which created conditions for the displacement of pneumococcus. The dynamics of AP etiology changed so rapidly and energetically that within one and a half to two decades, Staphylococcus aureus actually became the leading causative agent of the most severe and complicated forms of pneumonia, especially in children [8].

New antimicrobials for the treatment of inflammatory diseases have generated excitement and a storm of emotion since their introduction. Therefore, the development of resistance, the potential of which was already known in the microflora, primarily raised concerns about the future effectiveness of these drugs. This latter circumstance outweighed the importance of the role and influence of resistant microorganisms on the development of new problems. Since the initial focus was solely on the development of protective properties in individual members of the microflora, and antimicrobial therapy continued to ensure empirical compliance of the list of AP pathogens with the spectrum of activity of the drugs used, this situation did not cause particular concern. Practical medicine, having initially mastered the maximum number of currently known antibiotics [9], continued and continues for many years to invest in the early diagnosis of AP pathogens to expedite targeted antimicrobial therapy [10-12].

These efforts and attempts to improve the results of etiotropic therapy reflected the established strategy for treating AP and the priority given to defining the basic principles of treatment. This approach could not ignore the professional perspective on the essence of the problems being addressed, consistently focusing on the causative agent of AP as the underlying cause of the disease, with antibiotics firmly established as the primary treatment method. Despite dynamic changes in the conditions for the development of AP, this professional understanding of the problem remained unchanged throughout the decades of the antibiotic era and has not been seriously revised, despite a number of contradictions and inconsistencies. This approach to the essence of the AP problem, which originated in the first half of the last century as the so-called microbial concept of disease [13], acquired a stable dominant position in subsequent years, defining modern strategy and tactics in this field of medicine.

Despite the strictly defined goals of early diagnosis of the etiology of AP, expert forums on this issue have deemed the obtained results irrelevant, and the principles of antibiotic prescription empirical [14,15]. However, the most significant omission that modern medicine has failed to address is the dramatic change in the composition of pathogens that, according to numerous statistical data, have become the leading causes of AP. Bacterial factors are no longer the primary cause, which, in turn, includes pathogens that are quite "exotic" for this disease [16,17]. Already a couple of decades ago, a significant increase in the incidence of viral forms of AP was noted, reaching almost half of all cases worldwide [18,19]. However, it was well known that in the early days of antibiotic therapy, such forms of the disease were rare.

In this situation, a radical rethinking of the leading role of the "microbe-antibiotic" tandem as the primary, but now irrelevant, goal of solving the problem seems justified and necessary, doesn't it? After all, nature has replaced the bacterial factor with viruses, and antibiotics in this situation cannot fulfill their intended purpose. However, as is already known, in recent decades, no obvious attempts have been made to bring the strategic basis for solving the problem closer to real circumstances. Ultimately, according to the logic of events in recent years, the emergence of the SARS-CoV-2 pandemic seemed a completely natural phenomenon. However, in the context of entrenched didactic misconceptions about the concept of an epidemic, this phenomenon was perceived as a catastrophe, accompanied by manifestations of professional depression [20-23], searches for the causes of so-called conspiracy theories [24], and attempts to shift professional responsibility to the shoulders of the government [25].

The current literature on this issue convincingly demonstrates that the lessons of the pandemic remain unexplored and unlearned. The etiologic profile of AP, transformed in the era of antibiotics, surprisingly has no impact on our current understanding of the problem. The "emergency," unjustifiably widespread use of antibiotics during the pandemic [26-28] also did not contribute to a critical analysis of these events. Currently, despite the changed realities, calls are increasingly being made for the development and release of new, more advanced generations of antibiotics [29-31]. This initiative is actively supported by the WHO and is developing against the backdrop of ongoing practical efforts to early diagnose the etiology of AP using the latest testing systems [10-12].

Given the actual results, recent studies are more promotional than practical. Added to this is the often-cited assertion that pneumococcus remains the most common pathogen causing AP [32-34], which, as shown above, not only lost its prevalence in the early years of antibiotic use but has not even approached its previous levels since then [13,16]. Its current presence in overall statistics is too low for pneumococcus to play a significant role. However, such publications are usually accompanied by a rationale for continuing pneumococcal vaccination. These statements, attempting to emphasize the importance of previous vaccination efforts that failed to reduce the risk of severe AP, appear during a period of increasing viral pneumonia and reflect an undeniable bias. In addition to the currently observed trends, numerous studies are ongoing to find the optimal antibacterial therapy options for patients with AP [35-37]. All this creates the almost complete impression that the biological conditions for the development of the disease are perceived today in the same way as in the early years of the development of this field, as if nothing has changed.

The prevailing climate of understanding the nature of AP continues to methodically and relentlessly dictate the rules of the so-called germ theory of disease, which has exerted a powerful didactic influence and dominated professional opinion on this issue throughout the long history of antibiotic therapy. Judging by a series of ongoing studies and the continuation of previous initiatives, freeing ourselves from the web of misconceptions that have already shaped this persistent worldview will not be easy, although it is urgently needed. The persistent influence of this concept on professional views and approaches to solving the problem of AP is emphasized by the identified explanation for the low effectiveness of treatment for this category of patients. Although the causative agent of AP remains unknown in half or more cases [30,38,39], and viruses are beginning to predominate among positive results, the main consequence of long-term antibiotic use should be considered the gradual creation of conditions in which their further use becomes unnecessary. This conclusion is consistent with modern realities, but it is not even taken into account in the current understanding of the problem of AP.

Microbial resistance has become the primary and only explanation for treatment failure. This side effect of antibiotics has been observed relatively unnoticed throughout the history

of their use, without any significant discussions or major decisions. Increased interest in this consequence of antibiotic use has emerged in the last couple of decades, reflected in a significant increase in the number of publications and special WHO documents on this topic, which, incidentally, coincided with the rise of viral forms of AP. However, the recognition of this side effect as a global scourge was somewhat unexpected and "surprisingly" announced at the peak of the SARS-CoV-2 pandemic [40]. Given all the known and aforementioned facts, the timing of declaring microbial resistance a global scourge in the midst of a viral pandemic (!) seems symbolic, consistent with the prevailing understanding of the nature of AP today.

The WHO's recognition of resistant microflora as a global catastrophe logically fits into the understanding of the leading role of the pathogen and the lost hope for the effective use of antibiotics in the treatment of patients with AP. This assertion has found active support among the professional community, creating the image of a scientific explanation for the growing treatment failures. However, as an analysis of the actual state of microbial resistance shows, the overwhelming majority of opinions about the important and significant role of such microflora in solving the entire discussed problem of AP are declarative in nature and give rise to a new level of misconceptions and self-deception. Such publications are not supported by specific results or convincing evidence.

The stated danger of antibiotic-resistant microbes appears completely different when presented with statistics. It has been established that the etiologic role of such pathogens in AP is no more than 2% [41-43]. A logical question arises: how significant is the impact of resistant microorganisms on the outcome of the disease in the entire cohort of patients? This estimate is more the result of an adjustment based on the modern concept of AP than a critical analysis of the facts, isn't it? Several obvious factors should be added to this. First, no one has presented evidence for such an important argument in this case as the increased virulence and aggressiveness of resistant bacteria. By developing their own defenses, such microorganisms do not become more dangerous from a microbiological perspective. They can become a serious obstacle only if antibiotics remain the primary treatment.

Secondly, no researchers or clinicians currently compare the prevalence of resistant microflora in patients with AP with the percentage of asymptomatic carriers of similar strains in the healthy population. For example, the prevalence of MRSA as a commensal pathogen in healthy individuals in the general population is higher than in patients with AP, reaching 3%, while screening results in some groups of healthy individuals have shown this strain to be prevalent at a frequency of 6%–10% [44–46]. Attempts to explain the fact that the most medically dangerous microbial strains are found many times more frequently among healthy individuals, while posing little risk to them, would be futile, as there are no comprehensive explanations for such comparisons. Moreover, doing so would undermine the entire framework that explains current treatment failures.

Finally, the danger of misconceptions arising from exaggerating the role of microbial resistance in reducing the effectiveness of therapy lies in the fact that, while maintaining adherence to the old concept of AP, many call for the development and release of new-generation antibiotics [29,47,48]. It is difficult to imagine what even more significant consequences the implementation of such ideas could lead to without a clear and consistent analysis of past events. Although expert reviews of the rationale for the use of antibiotics have been conducted in the recent past, the results of which showed excessive enthusiasm for this therapy, these materials in no way influenced the events of the coronavirus pandemic, when antimicrobials began to be prescribed to almost all hospitalized patients [26-28]. Currently, it is necessary to carefully analyze the number of patients with AP for whom the indications for the use of these drugs remain due to the changes in the etiologic list of pathogens that have occurred in the antibiotic era, and it is clear that the number of such cases remains small.

Conclusion

Thus, if we rely on objective facts and statistical data, rather than manipulation of generally accepted concepts, the most important biological effect of antibiotic use is the steady change in the etiology of AP, with a gradual devaluation of the role of these drugs as therapeutic agents. The importance of resistant strains is significantly exaggerated due to the interpretation of this side effect, which is based on an outdated understanding of the disease. Antimicrobials cannot serve as the primary treatment for inflammatory processes, since their action is aimed solely at suppressing the microbial factor but does not directly affect the mechanisms of inflammation. In aggressive inflammatory processes, the frequency of which is gradually increasing, even effective neutralization of the pathogen cannot significantly affect the emergence of inflammatory triggers and their mechanisms of action.

Despite extensive and compelling evidence supporting the presented assessment of antibiotic side effects, changing prevailing opinions on this issue depends on the didactic consequences of this therapy. Statements that developed over decades and were based on the belief in the exceptional therapeutic effect of antibiotics have lost their original meaning over time, and changes in the etiology of AP have created a complex problem that is becoming increasingly apparent with each passing year. To realistically assess the consequences of long-term antibiotic use, it is necessary, first and foremost, to align professional views on the problem of AP with the canons of medical science and the accumulated data on this issue. Such steps, requiring a fundamental rethinking of the disease concept, will allow us to understand the causes of existing misconceptions and pave the way for successful solutions.

References

1. Abraham EP, Chain E. An enzyme from bacteria can destroy penicillin. Rev Infect Dis. 1988;10(4):677–678. Available from: https://pubmed.ncbi.nlm.nih.gov/3055168/
2. Rammelkamp T. Resistance of Staphylococcus aureus to the action of penicillin. Exp Biol Med. 1942;51:386–389. Available from: https://doi.org/10.3181/00379727-51-13986
3. Avery OT, Chickering HT, Cole RI, Dochez AR. Acute lobar pneumonia: prevention and serum treatment. Monographs of the Rockefeller Institute for Medical Research. New York: Rockefeller Institute for Medical Research; 1917. Available from: https://digitalcommons.rockefeller.edu/cgi/viewcontent.cgi?article=1004&context=monographs-rockefeller-institute
4. Cole RI. Acute pulmonary infections. De La Mar Lectures. Baltimore: Williams & Wilkins; 1927–1928.
5. Sutliff WD, Finland M. The significance of the newly classified types of pneumococci in disease: types IV to XX inclusive. JAMA. 1933;101:1289–1295. Available from: https://jamanetwork.com/journals/jama/article-abstract/245616
6. Heffron R. Pneumonia, with special reference to pneumococcus lobar pneumonia. Cambridge: Harvard University Press; 1939.
7. Small JT. A short history of the pneumococcus with special reference to lobar pneumonia. Edinb Med J. 1948;55(3):129–141.
8. MRSA history timeline: the first half-century, 1959–2009. Chicago: The University of Chicago Medical Center; 2010. Available from: https://doi.org/10.1093/jac/dkr437
9. Aminov RI. A brief history of the antibiotic era: lessons learned and challenges for the future. Front Microbiol. 2010;1:134. Available from: https://doi.org/10.3389/fmicb.2010.00134
10. Kyriazopoulou E, Karageorgos A, Liaskou-Antoniou L, et al. BioFire FilmArray pneumonia panel for severe lower respiratory tract infections: subgroup analysis of a randomized clinical trial. Infect Dis Ther. 2021;10:1437–1449. Available from: https://doi.org/10.1007/s40121-021-00459-x
11. Pickens CI, Gao CA, Morales-Nebreda L, Wunderink RG. Microbiology of severe community-acquired pneumonia and the role of rapid molecular techniques. Semin Respir Crit Care Med. 2024;45(2):158–168. Available from: https://doi.org/10.1055/s-0043-1777770
12. Ling L, Lai CKC, Rhee C. Bacterial multiplex polymerase chain reaction tests for the diagnosis and management of pneumonia: ready for prime time? Thorax. 2025;80:862–872. Available from: https://doi.org/10.1136/thorax-2024-222297
13. Podolsky SH. The changing fate of pneumonia as a public health concern in 20th-century America and beyond. Am J Public Health. 2005 Dec;95(12):2144–2154. Available from: https://doi.org/10.2105/ajph.2004.048397
14. Metlay JP, Waterer GW, Long AC, Anzueto A, Brozek J, Crothers K, et al. Diagnosis and treatment of adults with community-acquired pneumonia: an official clinical practice guideline of the American Thoracic Society and Infectious Diseases Society of America. Am J Respir Crit Care Med. 2019 Oct 1;200(7):e45–e67. Available from: https://doi.org/10.1164/rccm.201908-1581st
15. Martin-Loeches I, Torres A, Nagavci B, Aliberti S, Antonelli M, Bassetti M, et al. ERS/ESICM/ESCMID/ALAT guidelines for the management of severe community-acquired pneumonia. Intensive Care Med. 2023;49:615–632. Available from: https://doi.org/10.1007/s00134-023-07033-8
16. Gadsby NJ, Musher DM. The microbial etiology of community-acquired pneumonia in adults: from classical bacteriology to host transcriptional signatures. Clin Microbiol Rev. 2022;35:e00015-22. Available from: https://doi.org/10.1128/cmr.00015-22
17. Sirota SB, Bender RG, Villanueva Dominguez RM, Movo A, Swetschinski LR, Araki DT, et al. Global burden of lower respiratory infections and aetiologies, 1990–2023: a systematic analysis for the Global Burden of Disease Study 2023. Lancet Infect Dis. 2025. Available from: https://doi.org/10.1016/s1473-3099(25)00689-9
18. World Health Organization. Revised global burden of disease 2002 estimates. Geneva: World Health Organization; 2004. Available from: http://www.who.int/healthinfo/global_burden_disease/estimates_regional_2002_revised/en/
19. Ruuskanen O, Lahti E, Jennings LC, Murdoch DR. Viral pneumonia. Lancet. 2011;377(9773):1264–1275. Available from: https://doi.org/10.1016/s0140-6736(10)61459-6
20. Leiter RE. Reentry. N Engl J Med. 2020 Oct 14. Available from: https://doi.org/10.1056/nejmp2027447
21. Rosenquist JN. The stress of Bayesian medicine—uncomfortable uncertainty in the face of COVID-19. N Engl J Med. 2021;384:7–9. Available from: https://doi.org/10.1056/nejmp2018857
22. Salisbury H. Helen Salisbury: what might we learn from the COVID-19 pandemic? BMJ. 2020;368:m1087. Available from: https://doi.org/10.1136/bmj.m1087
23. Oliver D. David Oliver: conveyor belt medicine. BMJ. 2020;368:m162. Available from: https://doi.org/10.1136/bmj.m162
24. Gostin LO, Gronvall GK. The origins of COVID-19—why it matters (and why it doesn't). N Engl J Med. 2023;388:2305–2308. Available from: https://doi.org/10.1056/nejmp2305081
25. Editors. Dying in a leadership vacuum. N Engl J Med. 2020 Oct 8;383(15):1479–1480. Available from: https://doi.org/10.1056/nejme2029812
26. Huttner BD, Catho G, Pano-Pardo JR, Pulcini C, Schouten J. COVID-19: don't neglect antimicrobial stewardship principles! Clin Microbiol Infect. 2020;26(7):808–810. Available from: https://doi.org/10.1016/j.cmi.2020.04.024
27. Beovic B, Doušak M, Ferreira-Coimbra J, Nadrah K, Rubulotta F, Belliato M, et al. Antibiotic use in patients with COVID-19: a 'snapshot' Infectious Diseases International Research Initiative (ID-IRI) survey. J Antimicrob Chemother. 2020;dkaa326. Available from: https://doi.org/10.1093/jac/dkaa326
28. Rawson TM, Moore LSP, Zhu N, Ranganathan N, Skolimowska K, Gilchrist M, et al. Bacterial and fungal co-infection in individuals with coronavirus: a rapid review to support COVID-19 antimicrobial prescribing. Clin Infect Dis. 2020;ciaa530. Available from: https://doi.org/10.1093/cid/ciaa530
29. Hsu J. AI discovers a new class of antibiotics to kill resistant bacteria. New Sci. 2024 Jan 6;261(3472):12. Available from: https://doi.org/10.1016/S0262-4079(24)00011-3
30. Malinis M, Abbo L, Vazquez JA, Ostrosky-Zeichner L. Community-acquired pneumonia: a US perspective on the guideline gap. J Antimicrob Chemother. 2024;79(5):959–961. Available from: https://doi.org/10.1093/jac/dkae050
31. Khan MA, Bajwa A, Hussain ST. Pneumonia: recent updates on diagnosis and treatment. Microorganisms. 2025;13(3):522. Available from: https://doi.org/10.3390/microorganisms13030522
32. Besteman SB, Bogaert D, Bont L, Mejias A, Ramilo O, Weinberger DM, et al. Interactions between respiratory syncytial virus and Streptococcus pneumoniae in the pathogenesis of childhood respiratory infections: a systematic review. Lancet Respir Med. 2024;12(11):915–932. Available from: https://doi.org/10.1016/s2213-2600(24)00148-6
33. Lansbury L, McKeever TM, Lawrence H, Pick H, Baskaran V, Edwards-Pritchard R, et al. Pneumococcal pneumonia trends in adults hospitalised with community-acquired pneumonia over 10 years (2013–2023) and the role of serotype 3. Thorax. 2025;80:86–96. Available from: https://doi.org/10.1136/thorax-2024-221976
34. Olarte L, Musher DM, Rodriguez-Barradas MC. Higher-valent

pneumococcal conjugate vaccines—perspective for 2026. JAMA Intern Med. 2025;185(12):1493–1494. Available from: https://doi.org/10.1001/jamainternmed.2025.5436

35. Pan D, Nielsen E, Chung S, Niederman MS. Management of pneumonia in the critically ill. Minerva Med. 2023;114:667–682. Available from: https://doi.org/10.23736/s0026-4806.22.08467-1

36. Oliveira E Silva PG, Cerqueira Batista Filho LA, Ismael PF, Victoria VES, Alexandre TM, Larissa SM. Community-acquired pneumonia: epidemiology, diagnosis, prognostic severity scales, and new therapeutic options. Medwave. 2023;23(11):e2719. Available from: https://doi.org/10.5867/medwave.2023.11.2719

37. Palomeque A, Cilloniz C, Soler-Comas A, Canseco-Ribas J, Rovira-Ribalta N, Motos A, et al. A review of the value of point-of-care testing for community-acquired pneumonia. Expert Rev Mol Diagn. 2024;1–14. Available from: https://doi.org/10.1080/14737159.2024.2391027

38. Long ME, Mallampalli RK, Horowitz JC. Pathogenesis of pneumonia and acute lung injury. Clin Sci (Lond). 2022 May 27;136(10):747–769. Available from: https://doi.org/10.1042/cs20210879

39. Rabin EE, Walter JM, Wunderink RG, Qi C, Pickens CI. Clinical significance of culture-negative, PCR-positive bronchoalveolar lavage results in severe pneumonia. ERJ Open Res. 2023 Nov 13;9(6):00343-2023. Available from: https://doi.org/10.1183/23120541.00343-2023

40. World Health Organization. Antimicrobial resistance. 2021 Nov 17. Available from: https://www.who.int/news-room/fact-sheets/detail/antimicrobial-resistance

41. Sakamoto Y, Yamauchi Y, Jo T, Michihata N, Hasegawa W, Takeshima H, et al. In-hospital mortality associated with community-acquired pneumonia due to methicillin-resistant Staphylococcus aureus: a matched-pair cohort study. BMC Pulm Med. 2021;21:345. Available from: https://doi.org/10.1186/s12890-021-01713-1

42. Ding H, Mang NS, Loomis J, Ortwine JK, Wei W, O'Connell EJ, Shah NJ, Prokesch BC. Incidence of drug-resistant pathogens in community-acquired pneumonia at a safety net hospital. Microbiol Spectr. 2024;12:e00792-24. Available from: https://doi.org/10.1128/spectrum.00792-24

43. Gohil SK, Septimus E, Kleinman K, Avery TR, Varma N, Sands KE, et al. Initial antibiotic selection strategy and subsequent antibiotic use—insights from the INSPIRE trials. JAMA. 2025;334(12):1107–1109. Available from: https://doi.org/10.1001/jama.2025.11256

44. Aubry-Damon H, Grenet K, Ndiaye-Sall P, Che D, Corderio E, Bougnoux M, Rigaud E, Le Strat Y, Lemanissier V, Andremont A, et al. Antimicrobial resistance in commensal flora of pig farmers. Emerg Infect Dis. 2004;10:873–879. Available from: https://doi.org/10.3201/eid1005.030735

45. Albrich WC, Harbarth S. Health-care workers: source, vector, or victim of MRSA? Lancet Infect Dis. 2008;8:289–301. Available from: https://doi.org/10.1016/s1473-3099(08)70097-5

46. Graveland H, Wagenaar JA, Heesterbeek H, Mevius D, van Duijkeren E, Heederik D. 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. Available from: https://doi.org/10.1371/journal.pone.0010990

47. Serwecińska L. Antimicrobials and antibiotic-resistant bacteria: a risk to the environment and to public health. Water. 2020;12(12):3313. Available from: https://doi.org/10.3390/w12123313

48. Thara M. Antibiotic stewardship. Medicon Med Sci. 2024;6(3):1–2. Available from: https://doi.org/10.55162/MCMS.06.196