ORIGINAL ARTICLE

Unravelling the Versatile Nature of *Pseudomonas aeruginosa*: Challenges and Innovations in Infection Management

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Abstract

This review delves into the intricate and multifaceted nature of *Pseudomonas aeruginosa (P. aeruginosa)*, a notorious pathogen known for its adaptability, virulence, and resistance mechanisms. *P. aeruginosa* presents formidable challenges in both healthcare and community settings due to its ability to thrive in diverse environments, form biofilms, and acquire antibiotic resistance. The range of infections it causes, varying from mild to severe, highlights the urgent need for effective management strategies. Key aspects of *P. aeruginosa* pathogenesis, transmission, and associated risk factors are discussed, underscoring the significance of infection control measures, particularly in healthcare settings. The emergence of multidrug-resistant strains further emphasizes the necessity for innovative treatment approaches. Alternative therapies, including phage therapy; antimicrobial peptides; and natural products offer promising avenues for combatting *P. aeruginosa* infections, especially those caused by multidrug-resistant strains. Additionally, antibiotic combination therapy, incorporating antivirulence compounds, demonstrates potential in both treating infections and curbing the spread of antibiotic resistance. Despite these promising alternatives, challenges persist in the development and implementation of these approaches, encompassing efficacy, safety, and regulatory considerations. Further research, experimentation, and clinical trials are imperative to refine these strategies and address the ongoing threat posed by *P. aeruginosa* and other multidrug-resistant organisms. In summary, this review provides valuable insights into the current challenges and advancements in managing *P. aeruginosa* infections, with a specific focus on exploring novel therapeutic options and enhancing patient outcomes. Continued efforts in research and development are paramount for effectively combating this formidable pathogen and mitigating its impact on public health.

Keywords

Pseudomonas aeruginosa, Multidrug resistance (MDR), Antimicrobial resistance, Alternative therapy, Phage therapy, Antimicrobial peptide, Combination therapy

INTRODUCTION

Pseudomonas aeruginosa is a Gram-negative bacilli shape, aerobic bacterium, oxidase-positive, and lactose non-fermenters known for its versatile metabolism and resistance to antibiotics[1,2]. It commonly inhabits soil, water, and plant surfaces but is also a notorious opportunistic pathogen causing a wide range of infections[3], particularly in immunocompromised individuals and those with cystic fibrosis[3]. Its ability to form biofilms and acquire resistance mechanisms poses significant challenges in clinical settings, making it a critical focus of research in antimicrobial therapy and infection control[3,4].

Pseudomonas aeruginosa poses a significant threat in both hospital and community settings, contributing to infections such as healthcare-associated and community-acquired bloodstream infections. Delays in administering appropriate antibiotics and elevated mortality rates are often associated with these infections[5].

The aim of this review is to highlight the virulence and resilience of *Pseudomonas aeruginosa* as a pathogen and to explore current and emerging strategies for combating infections caused by this formidable organism. We will assess the challenges posed by *P. aeruginosa* in healthcare settings and the community, examining its ability to develop resistance mechanisms and form biofilms. Furthermore, we will discuss the existing arsenal of treatment options and identify areas where gaps in therapy persist. Ultimately, this review aims to provide insights into the ongoing efforts to develop novel therapies and improve infection control measures to better manage *P. aeruginosa* infections.

PSEUDOMONAS AERUGINOSA PATHOGENESIS

Pseudomonas aeruginosa is a highly adaptable and opportunistic pathogen capable of causing a diverse array of infections, particularly in individuals with compromised immune systems or underlying health conditions[4,6]. Its pathogenesis is multifaceted and involves several key factors. Firstly, *Pseudomonas aeruginosa* utilizes various adhesins and surface structures to adhere to host tissues, facilitating colonization of mucosal surfaces such as the respiratory tract, urinary tract, and skin[7]. Additionally, it exhibits a remarkable ability to form biofilms, structured communities of bacteria encased within a self-produced matrix, enhancing bacterial persistence and resistance to host immune defenses and antimicrobial agents, thereby contributing to chronic and recurrent infections[8,9]. The secretion of virulence factors further exacerbates its pathogenicity, including exotoxins like exotoxin A, which disrupts host protein synthesis and immune evasion; and pyocyanin, a pigment with pro-inflammatory and cytotoxic effects. Enzymes such as elastase; proteases; phospholipases; and lipases degrade host proteins and disrupt cell membranes, promoting tissue invasion and destruction. *Pseudomonas aeruginosa* also produces siderophores like pyoverdine and pyochelin to scavenge iron, facilitating bacterial growth and survival in iron-limited environments like host tissues[10,11]. Moreover, its intrinsic and acquired antibiotic resistance mechanisms, including impermeable outer membranes and efflux pumps, pose significant challenges to treatment, complicating infections and contributing to their severity[12,13]. Overall, the pathogenesis of *Pseudomonas aeruginosa* infections involves a complex interplay of bacterial adherence; biofilm formation; virulence factor secretion; and antibiotic resistance, allowing it to establish and persist in diverse host environments and causing a broad spectrum of clinical manifestations, ranging from superficial to life-threatening infections[10,14,15].

Pseudomonas aeruginosa spreads primarily through environmental sources and direct contact with contaminated surfaces or infected individuals[16]. Environmental exposure *to Pseudomonas aeruginosa* is common, as the bacterium thrives in natural environments like soil, water, and vegetation[3]. Healthcare-associated infections (HAIs) also pose a significant risk, especially for immunocompromised individuals or those with indwelling medical devices like catheters or ventilators[17]. These infections can be transmitted via contaminated medical equipment, invasive procedures, or contact with healthcare personnel. While less common, person-to-person transmission of *Pseudomonas aeruginosa* can occur, particularly in settings where individuals have compromised skin integrity or are in close contact with infected individuals, such as households or long-term care facilities[11,18]. Various risk factors predispose individuals to *Pseudomonas aeruginosa* infections, including immunocompromised states; underlying health conditions like cystic fibrosis or COPD; hospitalization; and residence in long-term care facilities[2]. Infections caused by *Pseudomonas aeruginosa* can vary in severity, ranging from mild urinary tract or skin infections to life-threatening

conditions such as pneumonia or bloodstream infections, particularly in high-risk individuals[19]. Early recognition of risk factors and implementation of appropriate infection control measures are crucial for preventing and *managing Pseudomonas aeruginosa* infections, especially in vulnerable patient populations[20].

PSEUDOMONAS AERUGINOSA EPIDEMIOLOGY

According to surveillance data on antimicrobial resistance, a significant prevalence of carbapenem resistance in *Pseudomonas aeruginosa* isolates has been observed throughout Europe. This prevalence varies widely, with rates ranging from less than 5% in countries like the Netherlands and the United Kingdom to as high as 66% in Romania. On average, the rate of carbapenem resistance across Europe is nearly 18%[21]. A significant study conducted in the United States, utilizing data from a nationally representative sample collected by microbiological laboratories, revealed that approximately 22% of *Pseudomonas aeruginosa* strains causing pneumonia exhibited multidrug resistance[22]. A study in China focused on pneumonia patients revealed that *Pseudomonas aeruginosa* strains causing hospitalized pneumonia exhibited resistance rates of 35.7% to imipenem and 22.9% to meropenem[23].

Understanding the epidemiology of MDR *P. aeruginosa* is crucial for effectively managing infections. It has been shown that there are significant regional and interregional differences in the reported prevalence of MDR *P. aeruginosa* in general clinical samples. Egypt has reported the highest prevalence at 75.6%, while Morocco has reported the lowest prevalence at 0%. In Saudi Arabia and Qatar, the prevalence stands at 7.3% and 8.1%, respectively[24]. It's noteworthy that in the Kingdom of Bahrain, carbapenem-resistant MDR *P. aeruginosa* isolates predominantly carry blaVIM, similar to neighboring countries. However, they uniquely harbor blaNDM, a trait rare or absent in the region except for Saudi Arabia, Iraq, and Egyp[25,26]. Moreover, a study has shown that MDR *P. aeruginosa* prevalence in ICU samples across MENA region countries exhibited significant variation, showing an opposite trend compared to general clinical samples. Saudi Arabia (61%) and Syria (54%) had the highest rates, contrasting with Egypt (22.5%), Libya (36.4%), Lebanon (33.3%), and Morocco (28.5%). Moderate resistance to piperacillin-tazobactam was observed in Iraq (42.3%), Jordan (37.8%), Libya (37%), and Lebanon (22%), while lower rates were recorded in Oman (7%) and Saudi Arabia (17.2%)[24].

INFECTIONS CAUSED BY *PSEUDOMONAS AERUGINOSA*

Infections caused by *Pseudomonas aeruginosa* can range from relatively mild, superficial infections such as urinary tract infections and skin infections to severe, life-threatening conditions including pneumonia, bloodstream infections (septicemia), and soft tissue infections, particularly in high-risk individuals[19]. Early recognition of risk factors and implementation of appropriate infection control measures are essential for preventing and managing *Pseudomonas aeruginosa* infections, especially in vulnerable patient populations.

Pseudomonas aeruginosa is a formidable pathogen known for its ability to cause a spectrum of infections, particularly in individuals with compromised immune systems or underlying health conditions[20]. In hospital settings, it poses a significant threat as a cause of hospital-acquired pneumonia, especially among patients undergoing mechanical ventilation or those with pre-existing lung diseases[27]. Additionally, *Pseudomonas aeruginosa* can lead to urinary tract infections, often affecting individuals with indwelling catheters or structural abnormalities of the urinary tract. Skin and soft tissue infections[20], including hot tub folliculitis and cellulitis, are also common, particularly among burn patients[28]. Furthermore, invasive infections caused by *Pseudomonas aeruginosa* can result in bloodstream infections and sepsis, posing life-threatening risks, especially to immunocompromised individuals[29]. The bacterium can also manifest as otitis externa[30], eye infections[2], and chronic respiratory infections, notably in individuals with cystic fibrosis or chronic obstructive pulmonary disease[31,32]. Moreover, burn patients are at high risk of *Pseudomonas aeruginosa* infections, often leading to severe complications and delayed wound healing[2] (Table 1).

RESISTANCE MECHANISMS

Pseudomonas aeruginosa is renowned for its intrinsic and acquired antibiotic resistance mechanisms, bolstering its survival in diverse environments and facilitating persistent infections[33]. Among its arsenal of resistance strategies, *Pseudomonas aeruginosa* employs efflux pumps, actively expelling antibiotics from the bacterial cell to reduce their intracellular concentration and render them ineffective against a

Table 1. Infections caused by *Pseudomonas aeruginosa*

Disease Caused by *Pseudomonas aeruginosa*	Reference
Urinary tract infections	19
Skin infections	20
Pneumonia (including hospital-acquired pneumonia)	27
Bloodstream infections (septicemia)	29
Soft tissue infections (e.g., cellulitis)	20
Otitis externa	30
Eye infections	2
Chronic respiratory infections	31, 32
Burn-related infections	2
Sepsis	29
Risks to individuals with indwelling catheters or structural abnormalities of the urinary tract	20
Risks to individuals with pre-existing lung diseases	27
Risks to individuals undergoing mechanical ventilation	27
Risks to individuals with cystic fibrosis or chronic obstructive pulmonary disease	31, 32

broad spectrum of antibiotics, including β-lactams, fluoroquinolones, and aminoglycosides[34,35]. Moreover, its outer membrane exhibits reduced permeability compared to other Gram-negative bacteria, limiting antibiotic entry and diminishing their efficacy[36]. The bacterium can also modify antibiotic targets, such as penicillin-binding proteins and DNA gyrase, impairing the affinity of antibiotics for their targets and reducing their bactericidal effects[37]. Additionally, *Pseudomonas aeruginosa* produces antibiotic-degrading enzymes, such as β-lactamases, to hydrolyze and inactivate antibiotics, conferring resistance to penicillins, cephalosporins, and carbapenems[38,39]. Furthermore, its remarkable ability to form biofilms provides a protective niche, shielding bacterial cells from antibiotics and host immune defenses, thereby fostering chronic infections[40]. Through horizontal gene transfer mechanisms, *Pseudomonas aeruginosa* can acquire antibiotic-resistance genes rapidly, facilitating the emergence of multidrug-resistant strains and complicating treatment efforts[21]. The rise of extensively drug-resistant and pandrug-resistant strains underscores the urgent need for innovative antimicrobial strategies and robust infection control measures to combat *Pseudomonas aeruginosa* infections effectively[41].

Pseudomonas aeruginosa employs a multifaceted approach to antibiotic resistance, utilizing various genes and mechanisms to evade the effects of antimicrobial agents. Among these, AmpC β-lactamase stands out as an inducible enzyme capable of hydrolyzing a broad spectrum of β-lactam antibiotics, rendering them ineffective against the bacterium[21,42]. Additionally, the production of Extended-Spectrum β-lactamases (ESBLs) further contributes to resistance, conferring the ability to hydrolyze extended-spectrum cephalosporins and monobactams. Carbapenemases, including metallo-β-lactamases (MBLs) and serine carbapenemases, pose a significant challenge by hydrolyzing carbapenem antibiotics, often considered the last resort for treating multidrug-resistant infections[4]. Furthermore, efflux pumps (MexA) actively expel antibiotics from the bacterial cell, while mutations in quinolone resistance-determining regions and the production of aminoglycoside-modifying enzymes diminish the efficacy of fluoroquinolones and aminoglycosides, respectively[34]. Changes in outer membrane porins (OprM) and the formation of biofilms further bolster resistance by impeding antibiotic entry and providing a protective environment for bacterial cells[34]. These mechanisms highlight the adaptability of

Table 2. Different resistance mechanisms against different antibiotics by *Pseudomonas aeruginosa*

Antibiotics	Resistance Mechanism	Reference
Beta lactam such as 1st, 2nd, 3rd cephalosporins, aztreonam	AmpC β-lactamase, Extended-Spectrum β-lactamases (ESBLs)	18, 32, 33
penicillins, cephalosporins, carbapenems	Carbapenemases (including metallo-β-lactamases and serine carbapenemases)	34, 35
Carumonam, ceftriaxone, and cefotaxime	The reduced permeability of the outer membrane	36
Fluoroquinolones	Efflux pumps (MexA), mutations in quinolone resistance-determining regions	37
Aminoglycosides	Efflux pumps (MexA), production of aminoglycoside-modifying enzymes	37
Ciprofloxacin	Biofilm formation	23

Pseudomonas aeruginosa in combating antimicrobial agents, necessitating vigilant surveillance and the development of novel therapeutic strategies to address infections caused by multidrug-resistant strains. (Table 2).

ANTIBIOTIC THERAPY OF *PSEUDOMONAS AERUGINOSA*

Antibiotic therapy for *Pseudomonas aeruginosa* infections necessitates a thorough understanding of the bacterium's resistance mechanisms and the specific context of the infection. For mild to moderate cases, such as urinary tract or skin and soft tissue infections, oral antibiotics may suffice[43]. Conversely, severe or systemic infections, particularly those involving multidrug-resistant strains, often require intravenous antibiotics[44]. Commonly employed first-line therapies for susceptible strains include antipseudomonal β-lactam antibiotics like piperacillin-tazobactam, ceftazidime, and cefepime[20,39,45]. In severe infections or those involving multidrug-resistant strains, combination therapy may be necessary to enhance efficacy and mitigate further resistance development, often involving adjunctive agents such as aminoglycosides or fluoroquinolones[20,31]. In cases of carbapenem-resistant *Pseudomonas aeruginosa*, alternative agents like polymyxins, tigecycline, or newer cephalosporins may be considered, albeit cautiously due to their associated toxicities and limited efficacy[46,47]. Moreover, treatment decisions should be informed by local susceptibility patterns, patient-specific factors, and guided by culture and susceptibility results whenever feasible. Duration of therapy varies based on infection site and severity, typically ranging from 7 to 14 days, with vigilant monitoring for clinical response and resistance emergence throughout treatment[44].

ALTERNATIVE APPROACHES IN TREATING *PSEUDOMONAS AERUGINOSA*

Phage therapy, a form of targeted antimicrobial therapy utilizing bacteriophages (viruses that infect bacteria), has emerged as a potential alternative or adjunctive treatment for *Pseudomonas aeruginosa* infections, particularly in cases of multidrug-resistant strains where conventional antibiotics are ineffective[30,48,49]. Phages are highly specific to their bacterial hosts, targeting and lysing *Pseudomonas aeruginosa* cells while leaving beneficial bacteria unharmed[50].

In phage therapy for *Pseudomonas aeruginosa* infections, specific phages that target the infecting strain are isolated, purified, and formulated into therapeutic preparations[49]. These phage preparations can be administered topically, intravenously, or via inhalation, depending on the site and severity of the infection[51,52].

Studies have demonstrated the efficacy of phage therapy in treating *Pseudomonas aeruginosa* infections, including wound infections, burn infections, cystic fibrosis-associated lung infections, and urinary tract infections[32,49]. Phage therapy has shown promise in reducing bacterial load, improving clinical outcomes, and even eradicating multidrug-resistant strains[53].

However, challenges remain in the widespread implementation of phage therapy, including the need for rigorous characterization and quality control of phage preparations, potential development of phage resistance by the bacteria, limited understanding of phage pharmacokinetics and pharmacodynamics, and regulatory hurdles in some regions[30,54,55].

Despite these challenges, ongoing research and clinical trials are exploring the potential of phage therapy as a valuable tool in the armamentarium against *Pseudomonas aeruginosa* infections, offering hope for patients with limited treatment options due to multidrug-resistant strains[56]. Further studies are needed to elucidate the optimal strategies for phage selection, dosing, administration routes, and combination therapies to maximize efficacy and safety in clinical settings.

Antimicrobial peptides (AMPs) represent a promising class of molecules for treating *Pseudomonas aeruginosa* infections due to their broad-spectrum antimicrobial activity, rapid killing kinetics, and low propensity for inducing bacterial resistance[31,57,58]. These peptides are naturally occurring components of the innate immune system found in various organisms[59] including humans, plants, and animals[60]. AMPs exert their antimicrobial effects through diverse mechanisms, including disruption of bacterial cell membranes, inhibition of cell wall synthesis, and modulation of intracellular processes[61]. Due to their multifaceted mode of action, AMPs are less prone to bacterial resistance compared to conventional antibiotics[62].

Several AMPs have demonstrated efficacy against *Pseudomonas aeruginosa* both in vitro and in animal models of infection[40]. For example, peptides such as LL-37, magainin, and cecropins have shown potent activity against *Pseudomonas aeruginosa* by disrupting bacterial membranes and inducing cell lysis[63,64]. Additionally, synthetic AMPs, designed to mimic the structure and function of naturally occurring peptides, have been developed and tested for their efficacy against *Pseudomonas aeruginosa*[65].

One advantage of AMPs is their ability to synergize with conventional antibiotics, enhancing their antimicrobial activity and overcoming resistance mechanisms[66]. Furthermore, AMPs have been shown to possess immunomodulatory properties, including the ability to stimulate host immune responses and promote wound healing[67], which can aid in the clearance of *Pseudomonas aeruginosa* infections[68]. While the therapeutic potential of AMPs for treating *Pseudomonas aeruginosa* infections is promising, challenges remain, including issues related to peptide stability, formulation, and delivery[69]. Nevertheless, ongoing research efforts are focused on optimizing the design and development of AMP-based therapeutics, with the goal of providing effective and alternative treatment options for *Pseudomonas aeruginosa* infections, particularly those caused by multidrug-resistant strains.

The use of natural products in treating *Pseudomonas aeruginosa* infections has gained attention due to their potential therapeutic benefits and relatively low risk of inducing bacterial resistance. Natural products encompass a diverse array of compounds derived from plants, animals, fungi, and microorganisms, many of which possess antimicrobial properties[70,71].

Several natural products have demonstrated activity against *Pseudomonas aeruginosa*, either through direct antimicrobial effects or by modulating host immune responses to combat infection[72]. For example, plant-derived compounds such as flavonoids, alkaloids, tannins[73], and essential oils have exhibited antimicrobial activity against *Pseudomonas aeruginosa* by disrupting bacterial cell membranes, inhibiting bacterial enzymes, or interfering with essential cellular processes[74,75].

Propolis, a resinous substance collected by bees from plant buds and sap, has been investigated for its antimicrobial properties against Pseudomonas aeruginosa. Studies have shown that propolis extracts exhibit inhibitory effects against Pseudomonas aeruginosa growth and biofilm formation, making it a potential therapeutic agent for preventing and treating infections[15,76].

Another natural product with antimicrobial activity against *Pseudomonas aeruginosa* is honey. Honey contains various compounds including hydrogen peroxide, bee-derived peptides, and phytochemicals, which possess antimicrobial properties[77]. Additionally, honey exhibits anti-inflammatory and wound-healing effects, making it beneficial for managing *Pseudomonas aeruginosa* infections, particularly in wounds and burns[78].

Marine-derived natural products have also shown promise as potential therapeutics against *Pseudomonas aeruginosa*[79]. Compounds isolated from marine organisms, such as sponges, algae, and corals, have demonstrated antimicrobial activity against *Pseudomonas aeruginosa* through various mechanisms, including disruption of bacterial membranes and inhibition of bacterial enzymes[14,80,81].

While natural products offer potential benefits in treating *Pseudomonas aeruginosa* infections, challenges remain, including issues related to standardization, quality control, and pharmacokinetics[82[. Additionally, further research is needed to elucidate the mechanisms of action of natural products against *Pseudomonas aeruginosa* and optimize their therapeutic use. Nevertheless, the exploration of natural products as alternative or adjunctive therapies for *Pseudomonas aeruginosa* infections represents a promising avenue for developing novel antimicrobial agents with potential clinical utility[77].

Studies have demonstrated that a combination of antivirulence compounds, such as gallium (a siderophore quencher) and furanone C-30 (a quorum sensing inhibitor), along with four clinically relevant antibiotics (colistin, colistin, ciprofloxacin, meropenem, tobramycin), holds promise in not only treating infections but also curbing the proliferation of antibiotic resistance[83]. Antibiotics have been demonstrated as a good alternative approach to tackling multi-drug-resistant bacteria. They can attack different targets simultaneously, reduce the probability of developing resistance by bacteria and attenuate the virulence

of bacteria[84,85]. In clinical practice, the utilization of novel β-lactam combination antibiotic regimens, such as ceftazidime/avibactam, ceftolozane/tazobactam, imipenem/cilastatin/relebactam, among others, has emerged as a strategy for addressing infections caused by multidrug-resistant (MDR) or extensively drug-resistant (XDR) *Pseudomonas aeruginosa* strains. These combinations represent innovative therapeutic approaches that offer alternative treatment options in situations where traditional antibiotics may not be effective due to the development of resistance mechanisms. While these novel regimens are not typically used as first-line treatments, they serve as valuable adjunctive therapies in managing infections caused by particularly challenging strains of *P. aeruginosa* that exhibit resistance to conventional antibiotics[19]. There is emerging evidence suggesting that combinations of β-lactams, such as meropenem with aztreonam or meropenem with ceftazidime, exhibit augmented efficacy in combating infections induced by multidrug-resistant (MDR) *Pseudomonas aeruginosa* in an invertebrate model of systemic infection. These findings represent novel insights into therapeutic strategies that have not been documented previously[86].

DISCUSSION

The multifaceted nature of *Pseudomonas aeruginosa,* as a formidable pathogen, is known for its adaptability, virulence, and resistance mechanisms. The bacterium's ability to thrive in diverse environments, form biofilms, and acquire antibiotic resistance poses significant challenges in both healthcare and community settings[3,4,10,11]. *Pseudomonas aeruginosa* infections range from mild to severe, impacting various organ systems and often resulting in high morbidity and mortality rates, particularly among immunocompromised individuals[5]. The review delves into the pathogenesis of *Pseudomonas aeruginosa* infections, highlighting its adherence mechanisms, biofilm formation, and secretion of virulence factors, all of which contribute to its pathogenicity and persistence. Moreover, the review emphasizes the modes of transmission and risk factors associated with *Pseudomonas aeruginosa* infections, underscoring the importance of infection control measures, particularly in healthcare settings. The emergence of multidrug-resistant strains further complicates treatment strategies, necessitating innovative approaches such as phage therapy, antimicrobial peptides, and natural products[3,70]. Additionally, the review explores the potential of antibiotic combination therapy, including antivirulence compounds, in addressing *Pseudomonas aeruginosa* infections while mitigating the spread of antibiotic resistance[83]. Overall, the review provides insights into the ongoing challenges and advancements in managing *Pseudomonas aeruginosa* infections, aiming to improve patient outcomes and public health. Moreover, highlights the different alternative approaches and the challenges in tackling *Pseudomonas aeruginosa*. Further research, experiments, and trials are needed to develop novel and different agents for providing a potential therapy against MDR organisms.

CONCLUSION

In conclusion, the review underscores the complex and multifaceted nature of *Pseudomonas aeruginosa* as a formidable pathogen, renowned for its adaptability, virulence, and resistance mechanisms. From its ability to thrive in diverse environments to its capacity for biofilm formation and acquisition of antibiotic resistance, *P. aeruginosa* poses significant challenges in healthcare and community settings alike. The spectrum of infections it causes, ranging from mild to severe, underscores the critical need for effective management strategies. This review emphasizes the importance of infection control measures, particularly in healthcare settings. The emergence of multidrug-resistant strains further underscores the urgency for innovative treatment approaches. Alternative therapies such as phage therapy, antimicrobial peptides, and natural products offer promising avenues for combating *P. aeruginosa* infections, particularly those caused by multidrug-resistant strains. Additionally, antibiotic combination therapy, including antivirulence compounds, shows potential in both treating infections and curbing the proliferation of antibiotic resistance. However, challenges remain in the development and implementation of these alternative approaches, including issues related to efficacy, safety, and regulatory hurdles. Further research, experimentation, and clinical trials are essential to optimize these strategies and address the ongoing threat posed by *P. aeruginosa* and other multidrug-resistant organisms. Overall, the review provides valuable insights into the current challenges and advancements in managing *P. aeruginosa* infections, with a focus on exploring novel therapeutic options and improving patient outcomes. Further efforts in research and development are crucial for the continued progress in combating this formidable pathogen.

CONFLICT OF INTEREST

The author declared that there is no conflict of interest that is related to this study and this article.

DISCLOSURE

The author did not receive any form of commercial support, including compensation or financial assistance, for this case report. Additionally, the author has no financial interest in any of the products, devices, or drugs mentioned in this article.

ETHICAL APPROVAL

Not applicable.

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