

Multiple Drug Resistance in Burn Patients

Abstract

Infections, the most common consequence and the main cause of mortality for burn patients, thrive in environments created by burn wounds. After severe burns, the immune system is suppressed, which makes invasive infections like pneumonia and urinary tract infections more likely to occur. Emerging infections that are resistant to many drugs provide a challenge to therapy, requiring the development of novel antimicrobial medications and rigorous procedures to limit infection. Because these infections have a high morbidity and death rate, the rise of multidrug-resistant organisms (MDROs) in burn victims is a serious issue. Because of their wide wound surfaces, weakened immune systems, and need for several invasive operations and extended hospital stays, burn patients are especially vulnerable to MDROs. The increasing prevalence of multidrug-resistant organisms (MDROs) in burn patients presents a serious risk to both patient outcomes and efficient treatment. Due to their weakened skin barrier, extended hospital stays, and frequent use of intrusive devices—all of which promote the spread of infections—burn victims are especially susceptible.

Keywords: Infections, burn, Multiple Drug Resistance

Introduction

Despite a global decline in the incidence of burn injuries and decreasing mortality rates due to advancements in burn care systems, burn injuries remain a significant public health issue, with an estimated 11 million cases annually(1). These injuries are particularly prevalent in low- and middle-income countries (LMICs), where socioeconomic factors, hazardous work conditions, and inadequate safety regulations exacerbate the risk(2)(3). Burn wounds create an optimal environment for infections, which are the most frequent complications and leading cause of death among burn patients(4)(5). The immune suppression following severe burns increases susceptibility to invasive infections, including pneumonias, urinary tract infections (UTIs), and bloodstream infections (BSIs)(6). Emerging multi-drug-resistant pathogens further complicate treatment, necessitating innovative antimicrobial therapies and stringent infection control measures(7). The burden of burns, measured in disability-adjusted life years (DALYs), remains substantial, with significant economic losses, particularly in LMICs where access to specialized burn care is limited(8)(9).

The emergence of multidrug-resistant organisms (MDROs) in burn patients is a significant concern due to the high morbidity and mortality associated with these infections. Burn victims are particularly susceptible to MDROs because of their compromised immune systems, extensive wound surfaces, and the need for prolonged hospital stays and multiple invasive procedures(10)(11)(12). Studies have shown that MDROs, including *Pseudomonas aeruginosa*, *Staphylococcus aureus*, and *Enterobacter* species, are frequently isolated from burn patients, with *Pseudomonas aeruginosa* being notably prevalent(13)(14)(15). The incidence of MDROs in burn units is influenced by factors such as antibiotic exposure, the use of invasive devices, and inadequate antimicrobial treatment(6)(16).

Epidemiology of MDROs

Burn wounds, initially sterile, become colonized by bacteria over time, with early colonization typically involving gram-positive bacteria from the skin, such as *Staphylococcus aureus*, within the first two days(16). As the wound environment evolves, gram-negative bacteria from the respiratory and gastrointestinal tracts, including *Pseudomonas aeruginosa* and *Acinetobacter baumannii*, become predominant(17). The incidence of multidrug-resistant organisms (MDROs) in burn patients is notably high, with studies indicating that 11.3% to 65.85% of burn patients acquire MDROs during their hospital stay. These infections significantly impact patient outcomes, leading to increased morbidity, mortality, and length of hospital stay(18)(19). Risk factors for MDRO acquisition include extensive total body surface area (TBSA) burns, prolonged hospital stays, and the use of invasive devices such as catheters and ventilators(20)(21)(11).

Burn patients are particularly susceptible to infections from multidrug-resistant organisms (MDROs), which significantly complicate their treatment and increase morbidity and mortality rates. Studies have consistently shown that infections in burn patients are predominantly caused by a few key pathogens. For instance, a study conducted in a U.S. military burn Intensive Care Unit(ICU) from 2003-2008 identified *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, *Klebsiella pneumoniae*, and *Staphylococcus aureus* as the most common bacteria, accounting for 76% of all infections. This aligns with findings from various other studies. For example, *Pseudomonas aeruginosa* and *Staphylococcus aureus* are frequently reported as prevalent pathogens in burn units, with *Pseudomonas aeruginosa* being particularly noted for its high resistance rates and association with increased mortality(11)(22)(23). Additionally, *Enterococcus* spp. and *Enterobacter* spp., though less common, are still significant contributors to the

infection burden in burn patients(12)(14)(20). The prevalence of these pathogens underscores the importance of continuous microbiological surveillance and the implementation of stringent infection control measures, such as hand hygiene and antimicrobial stewardship, to mitigate the spread of MDROs(16)(24). Moreover, the risk factors for MDRO acquisition in burn patients include prolonged hospital stays, the use of invasive devices, and inadequate antimicrobial treatment, which necessitate targeted interventions to improve patient outcomes(25).

Multi-Model Strategies for Prevention and Control

Infection prevention and control in burn centers is critical due to the high prevalence of multi-drug-resistant organisms(MDROs), which can originate from the patient's own microbes or the hospital environment. Effective cleaning and disinfection of hospital surfaces are essential in reducing the burden of MDROs. Enhanced cleaning methods, such as the use of ultraviolet (UV) devices and hydrogen peroxide vapor, have been shown to significantly lower infection rates, with some studies reporting reductions of up to 85% in certain MDROs(26)(27). Burn patients are particularly vulnerable to infections due to the loss of the skin barrier and induced immunosuppression, which increases their susceptibility to both localized and systemic infections(11)(14). The implementation of strict infection control measures, including hand hygiene, environmental disinfection, and the use of personal protective equipment, is crucial in managing MDRO outbreaks in burn units(25)(28). Studies have demonstrated that multi-model strategies, including ongoing staff education, cohorting or isolation of patients, and preemptive barrier precautions, can effectively reduce the incidence of healthcare-associated infections (HAIs) caused by MDROs(28)(29). Additionally, early excision of burn and wound grafting are essential to decrease the duration of hospitalization, infectious risk, and mortality. The use of novel antimicrobial therapies, such as cold plasma and topical antiseptics(15), along with rapid diagnostic tests and appropriate antimicrobial stewardship, further aids in combating MDRO infections(30).

Rubin et al.2023 demonstrated that universal patient decolonization effectively halted a Methicillin-resistant *Staphylococcus aureus* outbreak in a burn ICU, highlighting the critical role of decolonization in controlling nosocomial infections in high-risk settings(31). Similarly, Yahia et al. 2023 found that the application of nasal mupirocin significantly reduced MRSA infections, underscoring the efficacy of targeted decolonization protocols(32). However, universal contact precautions alone have shown limited effectiveness in curbing the spread of multidrug-resistant organisms (MDROs), possibly due to high rates of hand contamination after glove removal, which can undermine the

benefits of such precautions(33). This is supported by findings that emphasize the importance of comprehensive infection control measures, including rigorous hand hygiene practices(34). Additionally, antimicrobial stewardship programs, which aim to optimize the use of antibiotics, have been shown to improve patient outcomes and reduce MDRO infections, particularly when combined with other infection control strategies such as decolonization and environmental cleaning(35). For instance, a study on the implementation of a nasal antiseptic decolonization program in ICUs reported a reduction in healthcare-associated infections (HAIs), including MRSA bacteremia, further validating the effectiveness of decolonization measures(36).

Antimicrobial stewardship

Antimicrobial stewardship (AMS) is a critical strategy in healthcare aimed at optimizing the use of antimicrobial agents to improve patient outcomes, enhance safety, and reduce the incidence of infections such as *Clostridium difficile*(37). The emergence of multidrug-resistant organisms (MDROs) is a significant concern, particularly in burn infections, which are highly susceptible to these resistant pathogens(38). AMS programs are designed to address this issue by implementing a range of interventions, including the prudent selection, dosage, and duration of antimicrobial therapy, as well as the de-escalation of empiric therapy based on microbiology results(39). These programs also emphasize the importance of infection control measures, such as hand hygiene and the use of computerized alert systems, to prevent the spread of drug-resistant bacteria within healthcare facilities(40). In the intensive care unit (ICU), where the prevalence of resistant pathogens and the complexity of pharmacology are particularly high, AMS interventions have been shown to improve the quality and quantity of antimicrobial prescribing without compromising patient outcomes(41). Additionally, AMS programs have demonstrated significant benefits in reducing hospital stay lengths, readmission rates, and mortality associated with infections, as well as lowering healthcare costs and the incidence of *Clostridium difficile* colitis(42).

Rapid identification and antimicrobial susceptibility testing

Rapid identification and antimicrobial susceptibility testing (AST) are essential for optimizing antibiotic use and improving patient outcomes, particularly in critical infections like bloodstream infections (BSI). The Accelerate PhenoTest BC Kit is a notable advancement, providing pathogen identification and resistance profiles in approximately 7 hours, significantly faster than the traditional 48-72 hours required by conventional methods(43). This rapid turnaround is crucial for timely adjustments in treatment, potentially reducing morbidity and mortality associated with infections. Similar rapid testing systems, such as the

BiofireFilmarray and Verigene, also offer the capability to detect multiple bacteria and antibiotic resistance genes, enhancing the speed and accuracy of diagnosis(44). The EUCAST-RAST method, for instance, can provide results within 4 to 8 hours, although its efficacy varies among different pathogens, with *Staphylococcus aureus* showing 100% categorical agreement at 4 hours(45). Other innovative approaches include the FAST™ System, which isolates and concentrates microorganisms from positive blood cultures within 30 minutes, allowing for direct downstream testing and rapid resistance detection(46). Additionally, methods like the microfluidic ladder-based system and automated platforms that use fluorescently labelled rRNA probes have reduced AST turnaround times to as little as 4-5 hours, maintaining high accuracy and agreement with traditional methods(47)(48)(49).

Ventilator-associated pneumonia (VAP) is a significant concern in burn patients, often leading to severe outcomes if not promptly and accurately treated. The initial use of inappropriate antibiotics can exacerbate these outcomes, particularly due to the prevalence of multidrug-resistant organisms (MDROs) in these settings. Rapid diagnostic methods have emerged as crucial tools in identifying the causative pathogens and their resistance profiles, thereby guiding more effective and targeted antibiotic therapy. Techniques such as multiplex PCR, which can identify a wide range of pathogens and resistance markers within hours, have shown promise in improving diagnostic accuracy and speed(50). Similarly, nanopore-based metagenomic next-generation sequencing (mNGS) can detect pathogens and antimicrobial resistance genes within approximately 5 hours, significantly faster than traditional cultures(51)(52). Other methods like GeneXpert Carba-R, which identifies carbapenem-resistant genes directly from clinical samples, have demonstrated high sensitivity and specificity, aiding in the rapid detection of resistant strains such as *Acinetobacter baumannii* and *Klebsiella pneumoniae*(53). These advancements are particularly beneficial in the ICU, where VAP is common and associated with high morbidity and mortality(54). The use of these rapid diagnostic tools can help in the early initiation of appropriate antimicrobial therapy, reducing the risk of MDROs and improving patient outcomes(55).

PNA-FISH (Peptide Nucleic Acid Fluorescence in Situ Hybridization) is a molecular technique that allows for the rapid identification of microorganisms directly from clinical samples without the need for traditional culturing methods. This method uses fluorescently labelled peptide nucleic acid probes that hybridize to specific ribosomal RNA sequences of target pathogens, enabling their visualization under a fluorescence microscope. PNA-FISH has been FDA-approved for use in blood cultures and has shown promise in detecting pathogens in burn

wounds, as demonstrated in animal studies. The application of PNA-FISH in burn wounds is particularly advantageous because it provides rapid results, which is crucial for timely and appropriate treatment. Traditional culture methods can take several days to yield results, whereas PNA-FISH can identify pathogens within hours, thus facilitating quicker clinical decisions. This rapid identification is essential in burn wound management, where infections can quickly progress to sepsis and other severe complications(56)(57)(58). Moreover, the use of PNA-FISH can help reduce the empirical use of broad-spectrum antibiotics, thereby minimizing the risk of developing multi-drug resistant organisms (MDROs)(59)(60). Studies have shown that culture-independent methods like PNA-FISH and FISHseq provide additional diagnostic information that can be missed by conventional culture techniques, including the detection of nonplanktonic bacterial life forms and microbial biofilms, which are often implicated in chronic and non-healing wounds(61)(62).

Treatment Options for MDROs

Methicillin-resistant *Staphylococcus aureus*

Vancomycin remains the first-line treatment for methicillin-resistant *Staphylococcus aureus* (MRSA) infections, but its use is challenging in burn patients due to variable renal function, necessitating careful dosage to achieve therapeutic trough concentrations of 15-20 mg/L. However, AUC-based dosage is preferred to minimize nephrotoxicity and ensure efficacy, especially when the minimum inhibitory concentration (MIC) exceeds 2 mcg/mL, at which point alternative therapies are recommended(63)(64). Daptomycin serves as a viable alternative for MRSA wound and bloodstream infections, offering simpler renal dosing and higher efficacy at doses of 8-10 mg/kg daily for critically ill patients, although it is ineffective for lung infections due to inactivation by pulmonary surfactant(65)(66).

Linezolid, an oxazolidinone antibiotic, is frequently employed to treat methicillin-resistant *Staphylococcus aureus* (MRSA) pneumonia and skin infections due to its efficacy against Gram-positive bacteria and its ability to achieve high concentrations in lung fluid and tissues(67)(68). Its mechanism involves binding to the 50S ribosome, inhibiting protein synthesis, which is effective against a range of Gram-positive organisms, including multi-resistant strains(69)(70). However, its bacteriostatic nature, which inhibits bacterial growth rather than killing the bacteria outright, makes it less suitable for bloodstream infections (BSI) where bactericidal (bacteria-killing) activity is often preferred(71). In critically ill patients, the pharmacokinetics and pharmacodynamics of linezolid can be significantly altered, necessitating careful consideration of dosing regimens to ensure therapeutic efficacy while minimizing the risk of

adverse effects(72)(73). Long-term use of linezolid is limited by potential severe side effects, including bone marrow suppression, peripheral neuropathy, and optic neuropathy, which can lead to acute multiorgan failure, especially in patients with preexisting comorbidities(74)(75). Despite these risks, linezolid has shown a high clinical success rate in treating Gram-positive infections in critically ill patients, with a reported success rate of 82.2% in a multi-center study(76).

Ceftaroline, a fifth-generation cephalosporin, is notable for its efficacy against methicillin-resistant *Staphylococcus aureus* (MRSA) due to its strong binding affinity to penicillin-binding protein 2a (PBP-2a)(77). It has been approved for treating community-acquired pneumonia (CAP) and complicated skin and soft tissue infections (cSSTIs) in both adults and children, demonstrating similar clinical and microbiological efficacy to existing treatments(78)(79). Ceftaroline has shown promising results in treating MRSA pneumonia, including in burn patients, with a common dosing regimen of 600 mg every 12 hours, although an eight-hour dosing schedule is also frequently used(80)(81). Resistance to ceftaroline among MRSA strains has been documented, with variations in resistance rates observed across different regions. For instance, a study found that 2.9% of pediatric MRSA isolates exhibited reduced susceptibility to ceftaroline, particularly among healthcare-associated infections(82). Another study identified that 7.69% of MRSA isolates had increased minimum inhibitory concentrations (MICs) for ceftaroline, indicating emerging resistance(83). The resistance is often linked to mutations in the *mecA* gene, which encodes PBP2a, although secondary chromosomal mutations may also contribute(84). Despite these challenges, ceftaroline remains a valuable option for treating severe infections caused by resistant pathogens, including MRSA, due to its broad-spectrum activity and favorable safety profile(85).

Eravacycline and omadacycline, both derived from tetracycline, have shown promising results against methicillin-resistant *Staphylococcus aureus* (MRSA) in laboratory settings. Omadacycline is approved for treating community-acquired bacterial pneumonia (CABP) and acute bacterial skin and skin structure infections (ABSSSI)(86)(87). It has demonstrated efficacy comparable to other antibiotics in clinical trials for these indications, with a favorable safety profile and lower risk of adverse events leading to discontinuation (88)(89). Additionally, omadacycline has shown significant activity against *Mycobacterium abscessus*, a challenging pathogen, in both in vitro and animal studies, suggesting its potential for treating difficult lung infections(90)(91). Eravacycline, on the other hand, is approved for complicated intra-abdominal infections and has shown high effectiveness against multidrug-resistant *Acinetobacter baumannii*, particularly when combined with other

antibiotics like amikacin(92). Both antibiotics exhibit broad-spectrum activity and have been evaluated for their pharmacokinetics in patients with various comorbidities, indicating no need for dose adjustments based on these factors(93). Omadacycline also possesses unique immunomodulatory properties, which may enhance its therapeutic potential in conditions where immune response modulation is beneficial(94).

Therapeutics for Vancomycin-Resistant Enterococcal

Vancomycin-resistant enterococci (VRE) are significant nosocomial pathogens, with *Enterococcus faecium* exhibiting higher rates of vancomycin resistance compared to *Enterococcus faecalis*(95). These bacteria are notorious for their ability to resist most anti-gram-positive agents, posing a substantial challenge in clinical settings (96). While VRE may occasionally be susceptible to β -lactams, such instances are rare, necessitating alternative treatment strategies(97). Linezolid and high-dose daptomycin are commonly used treatment options for VRE infections. Linezolid, an oxazolidinone, has been particularly effective, although resistance to this drug has also been reported in some strains(98). High-dose daptomycin, often combined with other antibiotics such as ampicillin, ceftriaxone, or ceftaroline, has shown efficacy in treating VRE infections, especially in cases of persistent bacteremia and infective endocarditis(99). Additionally, newer antibiotics like eravacycline and omadacycline have demonstrated activity against VRE, although eravacycline is less effective for urinary tract infections (UTIs)(100). The prevalence of VRE in clinical settings, particularly in intensive care units (ICUs), underscores the need for stringent infection control measures and antibiotic stewardship programs to mitigate the spread of these resistant pathogens(101).

Addressing Carbapenem Resistance in *Klebsiella pneumoniae*

Klebsiella pneumoniae carbapenemases (KPCs) are a significant contributor to carbapenem resistance in Enterobacteriaceae, posing a severe public health threat due to their rapid spread and high mortality rates, particularly in immunocompromised patients(102)(103). The introduction of new β -lactam/ β -lactamase inhibitors (BL/BLIs) like ceftazidime-avibactam has shown promise against KPC-producing strains, but resistance is emerging, often due to mutations in the KPC enzyme(104)(105)(106). These inhibitors are ineffective against class B metallo- β -lactamases (MBLs) and some class D β -lactamases, necessitating alternative treatments(107)(108). Combination therapies and novel drugs such as cefiderocol, which has shown high activity against MBL-producing isolates, are being explored to address these resistant strains(109)(110). Plazomicin and eravacycline are also effective against

carbapenem-resistant *K. pneumoniae* (CRKP), but their clinical data is limited, and resistance issues persist(109).

Effectiveness of Ceftolozane-Tazobactam Against Resistant *Pseudomonas aeruginosa*

Ceftolozane-tazobactam (C/T) is a potent combination drug used to treat serious infections caused by *Pseudomonas aeruginosa*, including multidrug-resistant (MDR) and carbapenem-resistant strains. Despite tazobactam's inability to inhibit carbapenemases, C/T remains effective against many resistant strains due to ceftolozane's robust activity against *P. aeruginosa*, including carbapenem-resistant isolates when resistance mechanisms other than carbapenemase production are involved(111)(112). Studies have shown that C/T is highly active against *P. aeruginosa*, with susceptibility rates exceeding 90% in various regions, although resistance can occur, particularly in strains harboring metallo-beta-lactamases (MBLs) like blaIMP and blaVIM(113)(114)(115). Comparative studies indicate that C/T and ceftazidime-avibactam (CAZ-AVI) have similar effectiveness and safety profiles for treating MDR *P. aeruginosa* infections, with no significant differences in clinical outcomes such as mortality and clinical cure rates(116). Additionally, imipenem-cilastatin-relebactam (IMI-REL) has shown efficacy against *P. aeruginosa*, including strains resistant to C/T, although resistance patterns vary geographically(117). Cefiderocol (CFD) is another promising agent, demonstrating high effectiveness against various resistant strains, including those resistant to C/T, and showing synergistic effects when combined with other antimicrobials like CAZ-AVI and fosfomycin(118).

Carbapenem-Resistant *Acinetobacter baumannii*

Acinetobacter baumannii, a significant nosocomial pathogen, often exhibits resistance to carbapenems, posing a substantial treatment challenge(119)(120). Polymyxins, such as colistin, are effective against carbapenem-resistant *A. baumannii* (CRAB) but are associated with severe nephrotoxicity and neurotoxicity, limiting their use(121)(122). Minocycline remains a viable option, although its efficacy can be compromised by biofilm formation, which necessitates higher antibiotic concentrations to eradicate biofilm-associated cells compared to planktonic cells(123). Tigecycline, while useful, has shown higher mortality rates when used as monotherapy compared to combination therapies, such as cefoperazone/sulbactam, which have demonstrated better clinical outcomes in CRAB bloodstream infections (BSI)(124)(125). Cefiderocol, a novel siderophore cephalosporin, has shown potent activity against multi-drug-resistant Gram-negative pathogens, including CRAB, and is particularly effective against strains with various β -lactamase enzymes(125)(126).

Conclusion

The increasing prevalence of multidrug-resistant organisms (MDROs) in burn patients poses a significant threat to effective treatment and patient outcomes. Burn patients are particularly vulnerable due to their compromised skin barrier, prolonged hospital stays, and frequent use of invasive devices, which facilitate the spread of infections. The emergence of MDROs, such as methicillin-resistant *Staphylococcus aureus* (MRSA) and resistant strains of *Pseudomonas*, complicates treatment further. Effective infection control measures, including stringent hygiene practices and antimicrobial stewardship, are essential to limit the spread of these organisms and reduce antibiotic pressure that is selected for resistant strains. Regular microbiological surveillance and sensitivity testing are crucial for guiding appropriate antibiotic use and preventing the escalation of resistance.

References

1. Gerstl JVE, Ehsan AN, Lassarén P, Yearley A, Raykar NP, Anderson GA, et al. The global macroeconomic burden of burn injuries. *Plast Reconstr Surg*. 2021;10–1097.
2. Yakupu A, Zhang J, Dong W, Song F, Dong J, Lu S. The epidemiological characteristic and trends of burns globally. *BMC Public Health*. 2022;22(1):1596.
3. Herndon DN, Lemaster J, Beard S, Bernstein N, Lewis SR, Rutan TC, et al. The quality of life after major thermal injury in children: an analysis of 12 survivors with 80% total body, 70% third-degree burns. *J Trauma Acute Care Surg*. 1986;26(7):609–19.
4. Collier ZJ, Bourcier AJ, Naidu P, Magee III WP, Potokar T, Gillenwater J. 57 Understanding the Burden of Burn Injury in Latin America & the Caribbean. *J Burn Care Res*. 2022;43(Supplement_1):S39–40.
5. Lu S, Yakupu A, Zhang J, Dong W, Song F, Dong J. The Epidemiology and Trends in the Burden of Burns throughout the World. 2022;
6. Kelly EJ, Oliver MA, Carney BC, Shupp JW. Infection and burn injury. *Eur Burn J*. 2022;3(1):165–79.
7. van Niekerk A. Burn-related injuries. In: *Oxford Research Encyclopedia of Global Public Health*. 2022.

8. Collier ZJ, McCool K, Magee III WP, Potokar T, Gillenwater J. 58 Burn injuries in Asia: a global burden of disease study. *J Burn Care Res.* 2022;43(Supplement_1):S40–1.
9. ANDRIADZE M, CHIKHLADZE N, KERESSELIDZE M. GENERAL EPIDEMIOLOGICAL CHARACTERISTICS OF BURN RELATED INJURIES. *Exp Clin Med Georg.* 2022;63–6.
10. Dunbar C, Santorelli JE, Marshall WA, Haines LN, Box K, Lee JG, et al. Cross-border antibiotic resistance patterns in burn patients. *Surg Infect (Larchmt).* 2023;24(4):327–34.
11. Cabral L, Rodrigues L, Tavares AH, Tomé G, Caetano M, Chaves C, et al. Analysis of Potential Risk Factors for Multidrug-Resistance at a Burn Unit. *Eur Burn J.* 2023;4(1):9–17.
12. Raza AA, Ibrahim M, Ishfaq R, Saleem I, Altaf MA, Asmat U. Incidence, Clinical Evaluation and Antibiogram of Bacterial Isolates Obtained from Burn Patients. *Pakistan J Med Heal Sci.* 2022;16(10):282.
13. Herbin SR, Barber KE, Isaacson AR, Dolman HS, McGee JD, Baylor III AE, et al. When more is still not enough: a case of ceftazidime-avibactam resistance in a burn patient. *J Burn Care Res.* 2022;43(2):474–8.
14. Buriro F, Ishaque S, Saeed A, Qamar MA, Batool A. Prevalence of Multidrug-Resistant Organism in ICU Burns Patients at Tertiary Care Hospital. *J Burn Care Res.* 2023;44(4):949–54.
15. Robben PM, Ayalew MD, Chung KK, Ressler RA. Multi-Drug-Resistant Organisms in Burn Infections. *Surg Infect (Larchmt).* 2021;22(1):103–12.
16. Cleland H, Tracy LM, Padiglione A, Stewardson AJ. Patterns of multidrug resistant organism acquisition in an adult specialist burns service: a retrospective review. *Antimicrob Resist Infect Control.* 2022;11(1):82.
17. ALfadli M, El-Sehsah EM, Ramadan MAM. Risk factors and distribution of MDROs among patients with healthcare associated burn wound infection. *Germes.* 2018;8(4):199.
18. van Langeveld I, Gagnon RC, Conrad PF, Gamelli RL, Martin B, Choudhry MA, et al. Multiple-drug resistance in burn patients: a retrospective study on the impact of antibiotic resistance on survival and length of stay. *J Burn Care Res.* 2017;38(2):99–105.

19. Chen YY, Wu PF, Chen CS, Chen IH, Huang WT, Wang FD. Trends in microbial profile of burn patients following an event of dust explosion at a tertiary medical center. *BMC Infect Dis.* 2020;20:1–11.
20. Ruegsegger L, Xiao J, Naziripour A, Kanumuambidi T, Brown D, Williams F, et al. Multidrug-resistant gram-negative bacteria in burn patients. *Antimicrob Agents Chemother.* 2022;66(9):e00688-22.
21. Xu Y, Li T, Qi S, Shen R, Chen D, Ben X, et al. An investigation of bacterial epidemiology and an analysis of bacterial resistance to antibiotics in a burn unit from 1993 to 1999. *Zhonghua Shao Shang za zhi= Zhonghua Shaoshang Zazhi= Chinese J Burn.* 2002;18(3):159–62.
22. Khudhair MK, AlAubydi MA. Determination the prevalence and antimicrobial susceptibility of bacteria isolated from burns and wounds. *Iraqi J Agric Sci.* 2023;54(1):93–9.
23. Haque ME, Bhuiyan MAT, Sultana R, Rahman A, Das MK, Siddique NEA, et al. Different Infection Profiles and Antimicrobial Resistance Patterns between Burn Intensive Care Unit (ICU) and Common Wards. *Sch J App Med Sci.* 2022;11:2019–25.
24. Golubkova AA, Kutlaeva YY, Bagin VA. Features of nosocomial infections in patients with severe burn injury. *Epidemiol Infect Dis.* 2021;26(5):214–23.
25. D'Abbondanza JA, Shahrokhi S. Burn infection and burn sepsis. *Surg Infect (Larchmt).* 2021;22(1):58–64.
26. Li S, Lin J, Tao S, Guo L, Huang W, Li J, et al. Multi-Model Strategies for Prevention of Infection Caused by Certain Multi-Drug Resistant Organisms in A Rehabilitation Unit: A Semi-Experimental Study. *Antibiotics.* 2023;12(7):1199.
27. Leybold T, Schäfer B, Beier JP. Measures for Preventing Infection in Burn Surgery. *Surg Technol Int.* 2022;41:sti41-1604.
28. Wang C, Zhang F, Breland A, Lineaweaver WC. Efficacy of infection control measures in managing outbreaks of multidrug-resistant organisms in burn units. *Ann Plast Surg.* 2021;86(4S):S454–7.
29. Tejiram S, Shupp JW. Innovations in infection prevention and treatment. *Surg Infect (Larchmt).* 2021;22(1):12–9.

30. LAZARESCU AL, GROSU-BULARDA A, ANDREI MC, FRUNZA A, GRAMA S, STOIAN A, et al. Burn infections characteristics: A review. *Rom J Med Pract.* 2021;16(1).
31. Rubin LG, Balamohan A, Kohn N. The continued effect of routine surveillance and targeted decolonization on the rate of *Staphylococcus aureus* infection in a level IV neonatal intensive care unit. *Infect Control Hosp Epidemiol.* 2023;44(11):1894–5.
32. Yahia A, Barber K, Herbin S, White M, Faris J, Laddaran L, et al. 700 Implementation of an Intranasal Decolonization Protocol and Line Changing Policy in Adult Burn Patients. *J Burn Care Res.* 2023;44(Supplement_2):S125–6.
33. Hockenberry T, Waterfield J, Richey K, Foster K. 739 A Review of Current Methicillin-Resistant *Staphylococcus Aureus* Decolonization Practices in a Pediatric Burn Population. *J Burn Care Res.* 2023;44(Supplement_2):S150–S150.
34. Kreiling S, Watson R, Perez G, Carr A, Wolfe R. 1199. Implementation of a Nasal Antiseptic Decolonization Program Reduces the Occurrence of Healthcare-Associated Infections in the Adult Intensive Care Unit Setting. In: *Open Forum Infectious Diseases.* Oxford University Press US; 2022. p. ofac492-1032.
35. Xun H, Modica A, Payne R, Seetharaman S, Reilly L, Bertuzzi R, et al. A multi-modal environmental bundle to reduce nosocomial methicillin-resistant *Staphylococcus aureus* transmission in a high volume burn intensive care unit: A prospective study. *J Plast Reconstr Aesthetic Surg.* 2023;77:397–9.
36. Stern RA, Harris BD, DeVault M, Talbot TR. Identifying barriers to compliance with a universal inpatient protocol for *Staphylococcus aureus* nasal decolonization with povidone-iodine. *Infect Control Hosp Epidemiol.* 2023;44(7):1167–70.
37. Creel JP, Maves RC. The microbiome and antimicrobial stewardship in surgical patients. *Surg Infect (Larchmt).* 2023;24(3):220–5.
38. Dyar OJ, Huttner B, Schouten J, Pulcini C. What is antimicrobial stewardship? *Clin Microbiol Infect.* 2017;23(11):793–8.
39. Rather MY, Waza AA, Hassan Y, Majid S, Farhat S, Bhat MH. Antimicrobial Stewardship Programme: Why Is It Needed? In: *Non-traditional Approaches to Combat Antimicrobial Drug*

Resistance. Springer; 2023. p. 309–20.

40. Albano GD, Midiri M, Zerbo S, Matteini E, Passavanti G, Curcio R, et al. Implementation of a year-long antimicrobial stewardship program in a 227-bed community hospital in Southern Italy. *Int J Environ Res Public Health*. 2023;20(2):996.
41. Parija SC. Antimicrobial Therapy BT - Textbook of Microbiology and Immunology. In: Parija SC, editor. Singapore: Springer Nature Singapore; 2023. p. 305–17.
42. Lanckohr C, Bracht H. Antimicrobial stewardship. *Curr Opin Crit Care*. 2022;28(5).
43. Park JM, Kwon M, Hong KH, Lee H, Yong D. European Committee on Antimicrobial Susceptibility Testing-Recommended Rapid Antimicrobial Susceptibility Testing of *Escherichia coli*, *Klebsiella pneumoniae*, and *Staphylococcus aureus* From Positive Blood Culture Bottles. *Ann Lab Med*. 2023;43(5):443.
44. Brosh-Nissimov T, Tzur A, Grupel D, Cahan A, Ma'aravi N, Heled-Akiva M, et al. Clinical impact of the accelerate PhenoTest® BC system on patients with gram-negative bacteremia and high risk of antimicrobial resistance: a prospective before-after implementation study. *Ann Clin Microbiol Antimicrob*. 2023;22(1):62.
45. Burg L, Crewe G, DiMeo J, Guo X, Li CG, Mayol M, et al. Rapid pathogen identification and phenotypic antimicrobial susceptibility directly from urine specimens. *Sci Rep*. 2022;12(1):18315.
46. Verroken A, Hajji C, Bressant F, Couvreur J, Anantharajah A, Rodriguez-Villalobos H. Performance evaluation of the FAST™ System and the FAST-PBC Prep™ cartridges for speeded-up positive blood culture testing. *Front Microbiol*. 2022;13:982650.
47. Zhang X, Wang X, Shen Z, Xu Y, Wang B, Zhang H, et al. Rapid antibiotic susceptibility testing of bacteria by single-field tracking centrifugation of bacteria solution. In: *Optics in Health Care and Biomedical Optics XII*. SPIE; 2022. p. 127–33.
48. Nguyen A V, Yaghoobi M, Azizi M, Davaritouchaee M, Abbaspourrad A. Ladder shaped microfluidic system enabling rapid antibiotic susceptibility testing with standardized concentration panel. *bioRxiv*. 2022;2008–22.
49. Gerhalter M, Kofler L, Zisser G, Merl-Pham J, Hauck SM, Bergler

- H. The novel pre-rRNA detection workflow “Riboprobing” allows simple identification of undescribed RNA species. *RNA*. 2024;rna-079912.
50. Alnimr A. Antimicrobial resistance in ventilator-associated pneumonia: predictive microbiology and evidence-based therapy. *Infect Dis Ther*. 2023;12(6):1527–52.
 51. Hori K, Shafiee R, Yenikomshian H, Newman D, Gillenwater J. 502 The Most Common Pathogens Isolated From Mechanically Ventilated Burn Patients With and Without Inhalation Injury. *J Burn Care Res*. 2023;44(Supplement_2):S77–S77.
 52. Owen RM, Chung KW. 894C358 Ventilator-Associated Pneumonia. Abd-Elseyed A, Abd-Elseyed A, editors. *Advanced Anesthesia Review*. Oxford University Press; 2023. p. 0.
 53. Chen T, Zhang L, Huang W, Zong H, Li Q, Zheng Y, et al. Detection of pathogens and antimicrobial resistance genes in ventilator-associated pneumonia by metagenomic next-generation sequencing approach. *Infect Drug Resist*. 2023;9:23–36.
 54. Briones-Rugama T, Marenco-Avilés S, Castillo-Cano MA, Porrás-Cortés GD. 2178. Microbiological Diagnosis of Ventilator Associated Pneumonia Caused by Gram Negative Bacterias Resistant to Carbapenems Using a Fast Molecular Method. In: *Open Forum Infectious Diseases*. Oxford University Press US; 2022. p. ofac492-1798.
 55. Fanning J, Panigada M, Bassi GL. Nosocomial Pneumonia in the Mechanically Ventilated Patient. In: *Seminars in Respiratory and Critical Care Medicine*. Thieme Medical Publishers, Inc.; 2022. p. 426–39.
 56. Scheuermann-Poley C, Wiessner A, Kikhney J, Gatzler R, Müller M, Stichling M, et al. Fluorescence In Situ Hybridization as Diagnostic Tool for Implant-associated Infections: A Pilot Study on Added Value. *Plast Reconstr Surgery–Global Open*. 2023;11(5):e4994.
 57. Kordestani SS, Mohammadi FS, Noordadi M, Rezaee F, Fayyazbakhsh F. Wound Infection Detection Using a Rapid Biomarker. *Adv Skin Wound Care*. 2023;36(1):35–40.
 58. Kotkot A, Ghabisha S, Ahmed F, Al-wageeh S, Al-shami E, Al-hajri A, et al. Fish skin as a biological dressing for burn injuries. *J*

Emerg Med Trauma Acute Care. 2022;2022(4):18.

59. Jaimes SL, Ramírez CE, Viviescas AF, Abril AF, Flórez DF, Sosa CD. Evaluation of burn wound infection in a referral center in Colombia. *Indian J Plast Surg.* 2022;55(01):75–80.
60. Zheng-Li C, Yu P, Guo-Sheng W, Xu-Dong H, Hao F, Xu-Dong Z, et al. Characterization of bacterial community structure dynamics in a rat burn wound model using 16S rRNA gene sequencing. *J Burn Care Res.* 2022;43(5):1086–94.
61. Wahyunitisari MR, Mustikasari MI, Hariani L. Mrsa Colonitacion Detection in Object Near Patients in Burn Unit RSUD Dr. Soetomo-Indonesia. *J Vocat Heal Stud.* 2021;5(1):22–5.
62. Thet NT, Jenkins ATA, Mercer-Chalmers JD, Coy K, Booth S, Collins D, et al. Laboratory study to investigate the sensitivity and specificity of a diagnostic dressing to detect burn wound infection. *medRxiv.* 2021;2007–21.
63. Menon V, van Hal SJ. Therapeutic Options for Resistant Gram Positives. *Curr Treat Options Infect Dis.* 2014;6:439–55.
64. Alhifany AA, Bifari N, Alatawi Y, Ullah Malik S, Almangour T. 465. Comparative efficacy of double vs. single antibiotic regimens for the empiric treatment of MRSA-induced acute bacterial skin and skin structure infection. In: *Open Forum Infectious Diseases.* Oxford University Press US; 2019. p. S227–8.
65. Barlow A, Heil EL, Claeys KC. Using an ordinal approach to compare outcomes between vancomycin versus ceftaroline or daptomycin in MRSA bloodstream infection. *Infect Dis Ther.* 2021;10:605–12.
66. Morrisette T, Alosaimy S, Abdul-Mutakabbir JC, Kebriaei R, Rybak MJ. The evolving reduction of vancomycin and daptomycin susceptibility in MRSA—salvaging the gold standards with combination therapy. *Antibiotics.* 2020;9(11):762.
67. Ma A, Dong M, Cheng J, Liao X, Dong W, Liu C, et al. Clinical efficacy and safety of linezolid in intensive care unit patients. *J Intensive Med.* 2023;3(1):65–72.
68. Hui LA, Bodolea C, Vlase L, Hiriscau EI, Popa A. Linezolid administration to critically ill patients: intermittent or continuous infusion? A systematic literature search and review. *Antibiotics.* 2022;11(4):436.

69. Bal AM. 7.11 - Oxazolidinone: Linezolid. In: Kenakin TBTCP, editor. Oxford: Elsevier; 2022. p. 201–12.
70. Wu W, Li L, Duan S, Wang Y. Clinical effectiveness and reliability of linezolid in the treatment of pulmonary tuberculosis complicated with severe pneumonia: a meta-analysis. *Am J Transl Res.* 2022;14(11):7622.
71. Zhang P, Tan J, Lin Y, Zhang H, Deng G, Chen X. Linezolid for patients with multidrug-resistant tuberculosis/extensively drug-resistant tuberculosis in China. *Drug Discov Ther.* 2022;16(2):96–8.
72. Nazarchuk OA, Vitkovskiy VL, Babina YM. Use of Linezolid in the treatment of surgical infectious complications under antibiotic resistance. *Perioperaciina Med.* 2020;3(2):34–9.
73. Shaikh A, McHugh J. Linezolid use and drug-induced liver injury. In: *Baylor University Medical Center Proceedings.* Taylor & Francis; 2021. p. 316–7.
74. Simon P, Busse D, Petroff D, Dorn C, Ehmann L, Hochstädt S, et al. Linezolid concentrations in plasma and subcutaneous tissue are reduced in obese patients, resulting in a higher risk of underdosing in critically ill patients: a controlled clinical pharmacokinetic study. *J Clin Med.* 2020;9(4):1067.
75. Fermeli DD, Marantos TD, Liarakos ALD, Panayiotakopoulos GD, Dedes VK, Panoutsopoulos GI. Linezolid: a promising agent for the treatment of multiple and extensively drug-resistant tuberculosis. *Folia Med (Plovdiv).* 2020;62:444.
76. Chen H, Du Y, Xia Q, Li Y, Song S, Huang X. Role of linezolid combination therapy for serious infections: review of the current evidence. *Eur J Clin Microbiol Infect Dis.* 2020;39:1043–52.
77. Lodise TP, Low DE. Ceftaroline fosamil in the treatment of community-acquired bacterial pneumonia and acute bacterial skin and skin structure infections. *Drugs.* 2012;72:1473–93.
78. McNeil JC, Sommer LM, Vallejo JG, Hulten KG, Kaplan SL, Flores AR. Reduced ceftaroline susceptibility among invasive MRSA infections in children: a clinical and genomic investigation. *Antimicrob Agents Chemother.* 2022;66(10):e00745-22.
79. Rosanova MT, Aguilar PS, Sberna N, Lede R. Efficacy and safety of ceftaroline: systematic review and meta-analysis. *Ther Adv Infect Dis.* 2019;6:2049936118808655.

80. Cilloniz C, Pericàs JM, Rojas J. Ceftaroline in severe community-acquired pneumonia. *Rev Española Quimioter.* 2022;35(Suppl 1):28.
81. Abate G, Wang G, Frisby J. Ceftaroline: systematic review of clinical uses and emerging drug resistance. *Ann Pharmacother.* 2022;56(12):1339–48.
82. Abdizadeh N, Haeili M, Kafil HS, Ahmadi A, Feizabadi MM. Evaluation of in vitro activity of ceftaroline on methicillin resistant *Staphylococcus aureus* blood isolates from Iran. *Iran J Microbiol.* 2021;13(4):442.
83. Lan SH, Chang SP, Lai CC, Lu LC, Chao CM. Ceftaroline efficacy and safety in treatment of complicated skin and soft tissue infection: a systemic review and meta-analysis of randomized controlled trials. *J Clin Med.* 2019;8(6):776.
84. Lan SH, Chang SP, Lai CC, Lu LC, Chao CM. Efficacy and safety of ceftaroline for the treatment of community-acquired pneumonia: a systemic review and meta-analysis of randomized controlled trials. *J Clin Med.* 2019;8(6):824.
85. Chen CW, Chang SP, Huang HT, Tang HJ, Lai CC. The efficacy and safety of ceftaroline in the treatment of acute bacterial infection in pediatric patients—a systemic review and meta-analysis of randomized controlled trials. *Infect Drug Resist.* 2019;1303–10.
86. Lodise TP, Gunter K, Mu F, Gao E, Yang D, Yim E, et al. Real-world effectiveness of omadacycline and impact of unapproved omadacycline prescription claims among adult outpatients with community-acquired bacterial pneumonia or acute bacterial skin and skin structure infections. *J Manag Care Spec Pharm.* 2023;29(8):952–64.
87. Li A, He S, Li J, Zhang Z, Li B, Chu H. Omadacycline, eravacycline, and tigecycline express anti-mycobacterium abscessus activity in vitro. *Microbiol Spectr.* 2023;11(3):e00718-23.
88. Lin F, He R, Yu B, Deng B, Ling B, Yuan M. Omadacycline for treatment of acute bacterial infections: a meta-analysis of phase II/III trials. *BMC Infect Dis.* 2023;23(1):232.
89. Rimal B, Nicklas DA, Panthi CM, Lippincott CK, Belz DC, Ignatius EH, et al. Efficacy of Omadacycline-Containing Regimen in a Mouse Model of Pulmonary *Mycobacteroides abscessus*

Disease. *Mosphere*. 2023;8(2):e00665-22.

90. Sakoulas G, Nowak M, Geriak M. Omadacycline in treating community-based infections: a review and expert perspective. *Expert Rev Anti Infect Ther*. 2023;21(3):255–65.
91. Trang M, Lakota EA, Safir MC, Bhavnani SM, Friedrich L, Steenbergen JN, et al. Evaluation of the Impact of Comorbidities on Omadacycline Pharmacokinetics. *Antimicrob Agents Chemother*. 2023;67(4):e02397-21.
92. Bryant AE, Stevens DL. Investigating the immunomodulatory activities of omadacycline. *J Antimicrob Chemother*. 2023;78(1):78–83.
93. Li JJ. Eravacycline (Xerava), A Novel and Completely Synthetic Fluorocycline Antibiotic. *Curr Drug Synth*. 2022;85–100.
94. Deolankar MS, Carr RA, Fliorent R, Roh S, Fraimow H, Carabetta VJ. Evaluating the efficacy of eravacycline and omadacycline against extensively drug-resistant *Acinetobacter baumannii* patient isolates. *Antibiotics*. 2022;11(10):1298.
95. Cairns KA, Udy AA, Peel TN, Abbott IJ, Dooley MJ, Peleg AY. Therapeutics for vancomycin-resistant enterococcal bloodstream infections. *Clin Microbiol Rev*. 2023;36(2):e00059-22.
96. Li G, Walker MJ, De Oliveira DMP. Vancomycin resistance in *Enterococcus* and *Staphylococcus aureus*. *Microorganisms*. 2022;11(1):24.
97. Nandini MS, Santharam P, Madhusadhan K, Puhazhendi T. Prevalence And Antimicrobial Susceptibility Pattern Of enterococccs Species With Special Reference To Vancomycin Resistance enterococcus In Various Clinical Samples. *NVEO-NATURAL VOLATILES Essent OILS Journal| NVEO*. 2021;6895–901.
98. Miller WR, Murray BE, Rice LB, Arias CA. Resistance in vancomycin-resistant enterococci. *Infect Dis Clin*. 2020;34(4):751–71.
99. Riccardi N, Monticelli J, Antonello RM, Di Lallo G, Frezza D, Luzzati R, et al. Therapeutic options for infections due to vanB genotype vancomycin-resistant enterococci. *Microb Drug Resist*. 2021;27(4):536–45.

100. M Alatrouny AM, A Amin M, S Shabana H. Prevalence of vancomycin resistant enterococci among patients with nosocomial infections in intensive care unit. *Al-Azhar Med J.* 2020;49(4):1955–64.
101. Goić-Barišić I, Radić M, Novak A, Rubić Ž, Boban N, Lukšić B, et al. Vancomycin-resistant *Enterococcus faecium* COLONIZATION and *Clostridium difficile* infection in a HEMATOLOGIC patient. *Acta Clin Croat.* 2020;59(3.):523–8.
102. Findlay J, Poirel L, Bouvier M, Gaia V, Nordmann P. Resistance to ceftazidime-avibactam in a KPC-2–producing *Klebsiella pneumoniae* caused by the extended-spectrum beta-lactamase VEB-25. *Eur J Clin Microbiol Infect Dis.* 2023;42(5):639–44.
103. Karampatakis T, Tsergouli K, Behzadi P. Carbapenem-resistant *Klebsiella pneumoniae*: virulence factors, molecular epidemiology and latest updates in treatment options. *Antibiotics.* 2023;12(2):234.
104. Maraki S, Mavromanolaki VE, Stafylaki D, Scoulica E. In vitro activity of newer β -lactam/ β -lactamase inhibitor combinations, cefiderocol, plazomicin and comparators against carbapenemase-producing *Klebsiella pneumoniae* isolates. *J Chemother.* 2023;35(7):596–600.
105. Liu Y. Advances in carbapenem resistance and hypervirulence of *Klebsiella pneumoniae*. In: *Second International Conference on Biological Engineering and Medical Science (ICBioMed 2022)*. SPIE; 2023. p. 1480–3.
106. Shen S, Tang C, Ding L, Han R, Yin D, Yang W, et al. Identification of KPC-112 from an ST15 *Klebsiella pneumoniae* strain conferring resistance to ceftazidime-avibactam. *Msphere.* 2022;7(6):e00487-22.
107. Hobson CA, Pierrat G, Tenaillon O, Bonacorsi S, Bercot B, Jaouen E, et al. *Klebsiella pneumoniae* carbapenemase variants resistant to ceftazidime-avibactam: an evolutionary overview. *Antimicrob Agents Chemother.* 2022;66(9):e00447-22.
108. Gaibani P, Amadesi S, Lazzarotto T, Ambretti S. Complete genome sequence of a multidrug-resistant *Klebsiella pneumoniae* strain carrying bla OXA181 and bla KPC-125 carbapenemase. *Microb Drug Resist.* 2022;28(9):916–20.
109. Íñigo M, Del Pozo JL. Treatment of infections caused by

carbapenemase-producing Enterobacterales. *Rev Española Quimioter.* 2022;35(Suppl 3):46.

110. Tamma PD, Bergman Y, Jacobs EB, Lee JH, Lewis S, Cosgrove SE, et al. Comparing the activity of novel antibiotic agents against carbapenem-resistant Enterobacterales clinical isolates. *Infect Control Hosp Epidemiol.* 2023;44(5):762–7.
111. Bassetti M, Vena A, Giacobbe DR. The safety of ceftolozane/tazobactam for the treatment of complicated urinary tract infections. *Expert Opin Drug Saf.* 2023;22(7):533–40.
112. Almangour TA, Ghonem L, Alassiri D, Aljurbua A, Al Musawa M, Alharbi A, et al. Ceftolozane-Tazobactam Versus Ceftazidime-Avibactam for the Treatment of Infections Caused by Multidrug-Resistant *Pseudomonas aeruginosa*: a Multicenter Cohort Study. *Antimicrob Agents Chemother.* 2023;67(8):e00405-23.
113. Karlowsky JA, Wise MG, Hsieh TC, Lu HC, Chen WT, Cheng MH, et al. Temporal and geographical prevalence of carbapenem-resistant *Pseudomonas aeruginosa* and the in vitro activity of ceftolozane/tazobactam and comparators in Taiwan—SMART 2012–2021. *J Glob Antimicrob Resist.* 2023;34:106–12.
114. Kang Y, Xie L, Yang J, Cui J. Optimal treatment of ceftazidime-avibactam and aztreonam-avibactam against bloodstream infections or lower respiratory tract infections caused by extensively drug-resistant or pan drug-resistant (XDR/PDR) *Pseudomonas aeruginosa*. *Front Cell Infect Microbiol.* 2023;13.
115. Hazirolan G, Özkul C. Evaluation of In Vitro Activity of Ceftolozane/Tazobactam and Ceftazidime/Avibactam Against Carbapenem-Resistant *Pseudomonas aeruginosa* Strains and Mechanisms of Carbapenem Resistance: Data from Tertiary Care Hospital. *Jundishapur J Microbiol.* 2023;16(3).
116. Kakehi A, Hagiya H, Iio K, Fujimori T, Okura M, Minabe H, et al. Susceptibility of ceftolozane/tazobactam against multidrug-resistant and carbapenem-resistant *Pseudomonas aeruginosa*. *New Microbiol.* 2023;46(2):213–5.
117. Karlowsky JA, Lob SH, Estabrook MA, Siddiqui F, DeRyke CA, Young K, et al. Susceptibility profile and β -lactamase content of global *Pseudomonas aeruginosa* isolates resistant to ceftolozane/tazobactam and/or imipenem/relebactam—SMART 2016–21. *JAC-Antimicrobial Resist.* 2023;5(3):dlad080.

118. Palombo M, Bovo F, Amadesi S, Gaibani P. Synergistic activity of cefiderocol in combination with piperacillin-tazobactam, fosfomycin, ampicillin-sulbactam, imipenem-relebactam and ceftazidime-avibactam against carbapenem-resistant Gram-negative bacteria. *Antibiotics*. 2023;12(5):858.
119. Seifert H, Müller C, Stefanik D, Higgins PG, Wohlfarth E, Kresken M. In vitro activity of cefiderocol against a global collection of carbapenem-resistant *Acinetobacter baumannii* isolates. *Antibiotics*. 2023;12(7):1172.
120. Desmoulin A, Sababadichetty L, Kamus L, Daniel M, Feletti L, Allou N, et al. Adaptive resistance to cefiderocol in carbapenem-resistant *Acinetobacter baumannii* (CRAB): microbiological and clinical issues. *Heliyon*. 2024;10(9).
121. Qader SS, Ganjo AR. Detection of carbapenemase in *Acinetobacter baumannii* enrolled in the relationship between biofilm formation and antibiotic resistance. *Zanco J Med Sci (Zanco J Med Sci)*. 2023;27(1):74–84.
122. Shields RK, Paterson DL, Tamma PD. Navigating available treatment options for carbapenem-resistant *Acinetobacter baumannii*-calcoaceticus complex infections. *Clin Infect Dis*. 2023;76(Supplement_2):S179–93.
123. Tavasol A, Khademolhosseini S, Noormohamad M, Ghasemi M, Mahram H, Salimi M, et al. Worldwide Prevalence of Carbapenem Resistance in *Acinetobacter baumannii*: A Systematic Review and Meta-analysis. *Infect Dis Clin Pract*. 2023;31(2):e1236.
124. Giannella M, Viale P. Treating carbapenem-resistant *Acinetobacter baumannii* infections. *Lancet Infect Dis*. 2023;23(9):994–5.
125. Gu S, Xiong J, Peng S, Hu L, Zhu H, Xiao Y, et al. Assessment of effective antimicrobial regimens and mortality-related risk factors for bloodstream infections caused by carbapenem-resistant *Acinetobacter baumannii*. *Infect Drug Resist*. 2023;2589–600.
126. Calò F, Onorato L, De Luca I, Macera M, Monari C, Durante-Mangoni E, et al. Outcome of patients with carbapenem-resistant *Acinetobacter baumannii* infections treated with cefiderocol: A multicenter observational study. *J Infect Public Health*. 2023;16(9):1485–91.