Iran J Burn Wound Res
ISSN: 3060-8414

2025, Volume 1, Number 4

Review Paper

Antibiotic resistance in bacterial infections of burn wounds: Challenges, mortality, and solutions

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ABSTRACT

Article info:
Received: 08 Sep 2025
Accepted: 25 Sep 2025

Keywords:
Burn wounds
Bacterial infections
Multidrug-resistant
Extensively drug-resistant

Burn wounds pose a significant clinical challenge due to their vulnerability to bacterial infections, compounded by the frequent emergence of multidrug-resistant (MDR) and extensively drug-resistant (XDR) pathogens. Factors such as immune dysregulation from burn injury, loss of the skin barrier, prolonged hospitalization, and invasive procedures contribute to increased susceptibility to colonization and infection by resistant bacteria, notably *Pseudomonas aeruginosa*, *Acinetobacter baumannii*, *Klebsiella pneumoniae*, and methicillin-resistant *Staphylococcus aureus* (MRSA). This review synthesizes current evidence on the microbiological landscape of burn wound infections, highlighting high rates of MDR/XDR isolates harboring diverse resistance mechanisms, including carbapenemases and extended-spectrum beta-lactamases. These resistant pathogens significantly impair healing by prolonging inflammation and biofilm formation and severely restrict effective antibiotic options, often necessitating last-resort therapies with associated toxicity. Risk factors predictive of MDR infections include extensive burns, invasive device use, prior antibiotic exposure, and longer intensive care unit (ICU) stays. Clinical management challenges extend from infection control and wound care to antimicrobial stewardship and the integration of emerging diagnostics. Future perspectives emphasize rapid molecular diagnostics, development of novel antimicrobials and topical agents, and multidisciplinary approaches involving surgeons, infectious disease specialists, and pharmacists to optimize outcomes. Prevention strategies targeting both hospital and community settings are critical for reducing burn incidence and infection rates. In conclusion, addressing antibiotic resistance in burn wounds requires comprehensive, evidence-based interventions and continued innovation to reduce morbidity and mortality and lower healthcare costs

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https://iranjburns.com/

https://doi.org/10.61882/ijbwr.1.4.41

1. Introduction

Patients with burns are uniquely vulnerable to colonization and infection by these resistant pathogens due to several factors, including immune dysregulation caused by the burn injury, the loss of the skin barrier, and prolonged hospital stays with frequent invasive procedures such as mechanical ventilation, catheterization, and multiple surgical interventions. Burn wounds represent a serious clinical challenge due to their vulnerability to bacterial infections that often complicate treatment and delay healing. One of the most critical problems in managing these infections is the emergence and spread of antibiotic-resistant bacteria, which significantly compromise therapeutic efficacy and increase morbidity and mortality among burn patients [1,2]. Burn wound infections involve a broad spectrum of bacterial organisms, complicating both diagnosis and management. Common pathogens include Gram-negative bacteria such as *Pseudomonas aeruginosa*, *Acinetobacter baumannii*, and *Klebsiella pneumoniae*, as well as Gram-positive organisms such as Staphylococcus aureus (including methicillin-resistant *S. aureus*, MRSA) and *Enterococcus* species. This microbial diversity poses challenges, as traditional culture methods are time-consuming and sometimes insensitive, delaying targeted therapy. Emerging molecular diagnostics and rapid susceptibility testing show promise but are not yet widely accessible in many burn centers. The presence of multidrug-resistant (MDR) and extensively drug-resistant (XDR) isolates further complicates management, as these pathogens evade many conventional antibiotics, forcing reliance on last-resort or combination therapies that may increase toxicity and cost. Persistent MDR and XDR infections prolong wound inflammation, hinder re-epithelialization, and promote secondary complications such as sepsis, leading to poorer healing outcomes [3-5]. Burn wounds infected with MDR and XDR bacterial isolates present highly limited treatment options both topically and systemically. The resistance mechanisms harbored by these bacteria restrict the efficacy of most conventional antibiotics, curtailing the range of antimicrobial agents effective against them. Therapeutic options are often limited to a few last-resort antibiotics such as polymyxins, tigecycline, and certain carbapenems, but resistance to these agents is increasingly reported, further complicating management. The high prevalence of MDR/XDR strains in burn centers demands regular susceptibility testing to tailor treatment, but lack of new antibiotic classes and rising resistance restrict options. Ultimately, the convergence of resistant pathogens, protective biofilms, and limited antibiotic options constitutes a major clinical challenge in burn wound infection management [6-8].

In summary, burn wound infections remain complicated by rapidly evolving antibiotic resistance among bacterial pathogens. This situation underscores the importance of stringent infection control measures, rational antibiotic use, and ongoing microbiological monitoring to improve clinical outcomes. Addressing these challenges is vital for reducing morbidity and mortality associated with infected burn wounds and for optimizing therapeutic strategies in burn care units [9-11]. Therefore, this study aims to provide a comprehensive overview of burn wounds, the antibiotic-resistant bacterial isolates commonly found in these wounds, the challenges posed by such resistance, and effective strategies to reduce the incidence of resistant infections in burn patients.

2. Overview of burn wound

Burn injuries result in complex wounds that present unique clinical challenges and require multidisciplinary management to improve patient outcomes. The skin, being the body's largest organ and first line of defense, when damaged by thermal, chemical, electrical, or radiation burns, triggers a cascade of physiological disruptions that complicate healing and often lead to severe systemic effects. Clinically, burn wounds range from superficial to full-thickness injuries, with deeper burns necessitating prompt surgical intervention to avoid mortality and long-term disability [12,13]. The depth and extent of skin and underlying tissue damage generally classify burn wounds. The main categories include superficial (first-degree), partial-thickness (second-degree), full-thickness (third-degree), and fourth-degree burns. Superficial burns affect only the epidermis, causing redness and pain but usually heal without scarring within days. Partial-thickness burns penetrate deeper into the dermis and are further divided into superficial and deep partial-thickness burns. These wounds are characterized by blistering, moisture, and varying degrees of pain depending on the extent of dermal involvement; healing time can range from days to weeks, with possible scarring [14,15]. Full-thickness burns involve destruction of the epidermis and dermis and often extend into the subcutaneous tissues; these burns appear white and leathery and are generally painless due to nerve damage, requiring surgical intervention. Fourth-degree burns extend beyond skin into muscle, bone, or other deeper structures and carry high morbidity and mortality risks. Each type presents distinct clinical challenges: superficial burns primarily require symptom management and infection prevention; partial-thickness burns pose risks of delayed healing and scar formation; full-thickness burns face increased infection risk and a need for grafting; and fourth-degree burns confront profound tissue loss and systemic complications [14,15]. One of the primary clinical challenges in burn treatment is managing the initial systemic response characterized by burn shock. This results from increased vascular permeability and extensive plasma leakage leading to hypovolemia, decreased cardiac output, and impaired perfusion of vital organs, which can culminate in multi-organ dysfunction if not promptly treated. Fluid resuscitation remains a cornerstone of early burn therapy, aiming to maintain adequate circulation and tissue oxygenation. However, precise fluid management is critical to prevent complications such as pulmonary edema and compartment syndromes. The burn size, generally quantified by the total body surface area (TBSA) affected, dictates the severity of burn shock and the intensity of required interventions; burns exceeding 20% TBSA in adults trigger systemic inflammatory responses that complicate clinical management [13,16,17]. Another formidable challenge is burn wound infections, which remains the leading cause of mortality in burn patients despite advances in care. The loss of the skin barrier, coupled with immunosuppression, facilitates bacterial colonization, resulting in local infections that may evolve into systemic sepsis, a life-threatening condition. Infection control is hindered by the presence of necrotic tissue or eschar, which not only serves as a nidus for microbial growth but also acts as a physical barrier to systemic antibiotics and topical agents. Severe infections may lead to prolonged inflammation, delayed healing, and extensive scarring, thereby increasing the risk of complications such as contractures and functional impairment [12,13]. Surgical management, including early excision of necrotic tissue and grafting, remains vital to decrease infection risk and promote wound closure. Early excision within the first 72 hours is associated with reduced mortality and length of hospital stay, and autologous split-thickness skin grafting is the current gold standard for wound coverage. However, this approach is not without its challenges. Donor site morbidity, limited availability of donor skin, especially in extensive burns, and the risk of graft rejection or failure complicate surgical care. These limitations have driven the development of dermal substitutes and bioengineered skin grafts, which aim to provide adequate temporary or permanent coverage, minimize scarring, and improve aesthetic and functional outcomes. However, widespread clinical adoption is still evolving [16,17].

The heterogeneity of burn wounds (in terms of depth, size, and tissue involvement) adds complexity to clinical assessment and treatment planning. Precise evaluation often requires advanced imaging techniques combined with predictive models to enhance accuracy in diagnosing burn depth and viability, which directly influences surgical decisions and prognosis. Standard research models that rely on homogeneous burns fail to capture this heterogeneity, sometimes limiting the translational value of experimental treatments for clinical practice [18,19].

Another clinical dimension involves managing the long-term sequelae of burns. Besides physical complications like scarring, contractures, and chronic wounds, psychological impacts, including post-traumatic stress disorder (PTSD) and depression, are common in burn survivors, requiring integrated psychiatric and rehabilitative care. Ensuring rehabilitation and quality-of-life improvements remains a critical focus that extends well beyond wound closure [8,13]. Finally, challenges in antimicrobial treatment have escalated due to the increasing prevalence of MDR organisms colonizing burn wounds. Resistance limits therapeutic choices and mandates robust infection surveillance and antibiotic stewardship programs in burn units. Adjunctive therapies, including topical antimicrobials and emerging nanotechnology-based drug delivery systems, are being explored to enhance efficacy and reduce systemic toxicity [7,20]. Among these, full-thickness and deep partial-thickness burns are more prone to colonization and infection with antibiotic-resistant bacteria. In contrast, superficial burns, which retain intact protective skin layers and heal rapidly, have a much lower risk of colonization by resistant bacteria. Thus, the severity and depth of the burn directly correlate with the likelihood of infection by antibiotic-resistant strains, making the management of full-thickness and deep partial-thickness wounds particularly challenging in burn care (Figure 1) [7,8,12].

3. Antibiotics resistance isolates in burn wounds

Burn wounds provide an ideal environment for bacterial proliferation, mainly when necrotic tissue and protein-rich exudates serve as nutrient-rich niches. MDR and XDR bacteria, commonly including *P. aeruginosa*, *A. baumannii*, and *K. pneumoniae*, often dominate burn wound flora in many burn centers worldwide. Studies have shown alarming rates of MDR and XDR isolates among these species; for example, a report indicated that up to 100% of *P. aeruginosa* isolates are MDR and 67% XDR, with similar figures for *A. baumannii* and other Gram-negative bacilli. These bacteria frequently harbor resistance mechanisms such as carbapenemase production and extended-spectrum beta-lactamases (ESBLs), severely limiting the effectiveness of antibiotics [5, 21, 22]. Among these pathogens, *P. aeruginosa* stands out as a significant cause of infection, exhibiting troubling levels of MDR. Studies in burn centers have reported that nearly half of *P. aeruginosa* isolates demonstrate resistance to commonly used antibiotics such as cloxacillin, cotrimoxazole, and ceftazidime. Genetic elements such as class 1 integrons facilitate the spread of antibiotic resistance genes among burn wound pathogens, exacerbating the problem [5]. The clinical consequences of infections with MDR and XDR bacteria in burn patients are severe. These infections are associated with higher rates of sepsis, systemic inflammatory response syndrome (SIRS), and multiple organ dysfunction syndrome (MODS). Mortality attributable to resistant infections is significantly elevated, while general burn mortality ranges widely depending on severity. Studies have found infection-related mortality rates as high as 40-75% in patients infected with MDR/XDR pathogens. The limited antibiotic arsenal complicates the treatment of such infections.

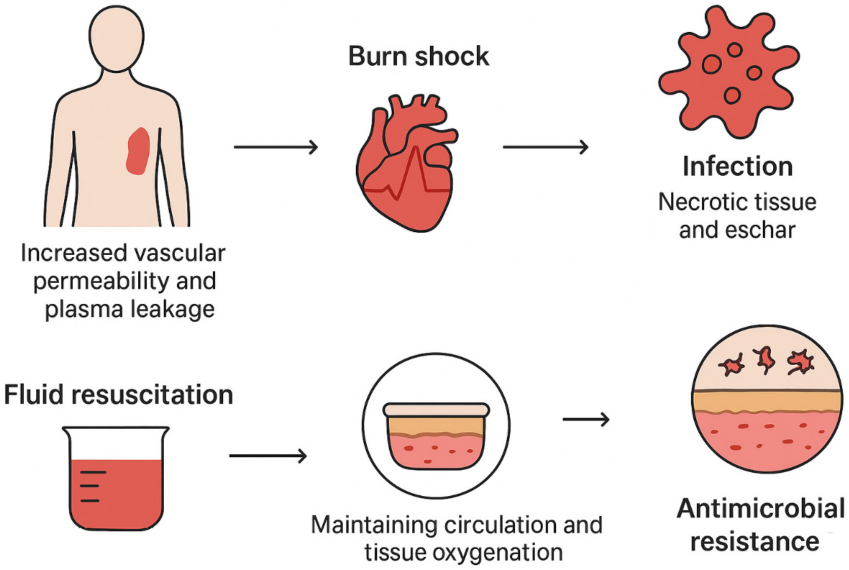

Figure 1. The cohesive visual summarizes how burn shock, infection, resuscitative efforts, and antimicrobial resistance intertwine to influence burn patient outcomes. The figure depicts a simplified, stepwise flowchart of key processes and clinical challenges following a burn injury. On the left, a human figure shows a localized burn area, labeled "Increased vascular permeability and plasma leakage," which is the initial physiological response. An arrow points to a stylized heart icon labeled "Burn shock," representing the systemic cardiovascular effects resulting from fluid shifts and hypovolemia. Another arrow points to a depiction of an infectious agent labeled "Infection," with emphasis on the presence of necrotic tissue and eschar, which serve as breeding grounds for pathogens. Below this sequence, the image highlights "Fluid resuscitation" with a beaker containing fluid, signifying efforts to restore blood volume and maintain tissue perfusion. An arrow points to a circular wound representation, symbolizing oxygenated, circulating blood. The final arrow points to a magnified wound surface showing bacteria, labeled "Antimicrobial resistance," illustrating the challenge of treating infections caused by resistant microorganisms in burn wounds.

Although colistin and tigecycline are sometimes employed as last-resort options, issues of toxicity and emerging resistance even to these agents pose therapeutic dilemmas [1, 4].

Research highlighted the evolving and adaptable nature of *P. aeruginosa*, stressing the importance of rigorous infection prevention protocols in burn units to control the dissemination of these highly resistant bacterial strains. The study conducted detailed genomic and molecular analyses of MDR and XDR *P. aeruginosa* isolates obtained from burn patients. Molecular testing via polymerase chain reaction (PCR) identified beta-lactamase genes spanning all four Ambler classes, with significant prevalence of *blaNDM*-1 (16.48%) and *blaVIM*-2 (31.87%), and a subset (3.30%) carrying both genes concurrently. Other detected resistance genes included *blaPER*-1 (15.38%), *blaCTX*-M (4.40%), *blaOXA*-1 (84.62%), and *blaOXA*-48 (51.65%). Additionally, class I integrons were found in 84 isolates. Biofilm formation was notably strong in 21% of the bacteria, supported by the widespread detection of biofilm-related genes such as *pelB*, *pilT*, and *rhlB*. Whole-genome sequencing of two selected XDR isolates revealed diverse beta-lactamase genes, including *blaPDC*-98, *blaPDC*-374, *blaOXA*-50, *blaOXA*-677, and *blaOXA*-847 [5]. Another investigation highlights the increasing prevalence of MDR bacteria in burn wound infections, underscoring the urgent need for robust infection control strategies and practical therapeutic approaches to tackle these biofilm-forming MDR pathogens. In the study, 56.8% of the bacterial isolates formed biofilms, with nearly half (48%) of these biofilm producers exhibiting multidrug resistance. Considerable resistance was observed to commonly prescribed antibiotics, including quinolones, cephalosporins, and cotrimoxazole. Specifically, *S. aureus* isolates showed resistance to ofloxacin, penicillin G, and amikacin; *Klebsiella* species were highly resistant to antibiotics, including ampicillin, ceftazidime, trimethoprim-sulfamethoxazole, tetracycline, and chloramphenicol; *P. aeruginosa* isolates displayed high resistance to trimethoprim-sulfamethoxazole; while *Acinetobacter* species exhibited resistance to cefotaxime, ceftriaxone, cefixime, and trimethoprim-sulfamethoxazole. Among Gram-positive bacteria, *S. aureus* retained full susceptibility to linezolid, vancomycin, and netilmicin, and coagulase-negative Staphylococci were sensitive to all tested antibiotics. Carbapenem antibiotics displayed the highest effectiveness among Gram-negative bacteria, including *Klebsiella*, *Proteus*, *P. aeruginosa*, and *Acinetobacter* species [23]. In one investigation conducted at a Burn and Plastic Surgery Hospital, 27 *A. baumannii* isolates were collected and analyzed. Antimicrobial susceptibility testing revealed that 63% (17 of 27) of these isolates exhibited XDR, with resistance to 8 of 9 tested antibiotic classes. Meanwhile, 37% (10 out of 27) were identified as MDR. All isolates carried the *blaTEM*-1 gene, and one isolate harbored two copies of *blaCTX*-1. Additionally, several isolates harbored the *blaAmpC* gene. The study emphasized the critical need to identify potential sources of infection and implement strict control measures to prevent the spread of these highly resistant strains [24]. A cross-sectional investigation was conducted to evaluate the distribution of MDR *K. pneumoniae* isolates across two burn hospitals and the antibiotic resistance profiles at different burn sites within individual patients. In this study, 55% (11 out of 20) of the *K. pneumoniae* isolates were identified as MDR, and 35% exhibited ESBL production, a primary mechanism of antibiotic resistance. Importantly, bacterial isolates obtained from different burned areas of the same patient showed varied antibiotic susceptibility patterns. The findings highlight that most *K. pneumoniae* strains contaminating burn wards in Iraqi hospitals are MDR, and that tigecycline is the most effective antibiotic against these resistant isolates [25].

A separate study was conducted to explore the factors linked to the development of multi-drug-resistant organisms (MDROs) in critically ill burn patients admitted to burn intensive care units (BICUs), as well as to assess how common these MDROs are within this group. The study involved 173 patients in total, of whom 168 were matched into two groups: 84 in the MDRO group (cases) and 84 in the non-MDRO group (controls). Compared to the control group, patients with MDRO infections exhibited lower baseline scores on the Glasgow Coma Scale (GCS), higher baseline scores on the Sequential Organ Failure Assessment (SOFA), elevated APACHE II scores, were more likely to be on invasive mechanical ventilation (MV) upon admission, and had a larger percentage of their total body surface area (TBSA) burned. Nevertheless, regression analysis identified that a lower baseline GCS score, a higher percentage of TBSA affected, and the presence of urinary tract infections were significant predictors of MDRO infection risk. Among the MDRO group, *A. baumannii* was the predominant pathogen isolated (57%), with pneumonia being the most frequent infection observed (52.4%). Additionally, both the duration of mechanical ventilation and the length of stay in the ICU were notably longer for patients in the MDRO group than for those without MDROs [26].

One study found that 55% of burn patients developed at least one infection during their hospital stay. Factors such as more extended hospital stay, occurrence of sepsis, wound dressings performed under anesthesia, receiving blood transfusions, and female gender were independently associated with an increased risk of infection in these patients. The infections were mainly caused by *S. aureus* (48.7%), followed by *P. aeruginosa* (22.6%) and *A. baumannii* (15.7%). Most bacterial isolates were multidrug-resistant or extensively drug-resistant, exhibiting diverse patterns of antibiotic susceptibility. Among burn patients with infections, 44.1% experienced recurrent infections, and more extended hospital stay, sepsis, surgical procedures including burn excision and skin grafting, and central line insertion were independent predictors of these recurrent infections [27]. Patient data, pathogen types, sources, and antimicrobial resistance patterns were retrospectively analyzed from burn patients in both the burn intensive care unit (BICU) and the burn care ward (BCW). Wound secretions represented 86.6% of samples in the BCW and 44.9% in the BICU. Compared to BCW samples, those from the BICU showed a higher proportion of fungi (11.8% vs. 8.1%), increased Gram-negative bacteria (60.0% vs. 50.8%), and fewer Gram-positive bacteria (28.2% vs. 41.1%).

A. baumannii was the predominant pathogen in the BICU, whereas *S. aureus* was most common in the BCW. *S. aureus* was frequently found in wound secretions and tissue samples from both units, but *A. baumannii* was the leading pathogen in blood, sputum, and catheter samples from the BICU. Overall, MDR rates were higher in the BICU compared to the BCW. There was a notable rise in MDR *A. baumannii* and *K. pneumoniae*, particularly in the BCW. Additionally, carbapenem resistance in *K. pneumoniae* significantly increased from 4.5% to 40% in the BICU and from 0% to 40% in the BCW between 2011 and 2019. Conversely, the MDR rate for *P. aeruginosa* in the BICU dropped sharply from 85.7% to 24.5%. MRSA incidence was significantly higher in the BICU (94.2%) than in the BCW (71.0%) and consistently remained elevated in the BICU (ranging from 89.5% to 96.3%) [28].

However, risk factors predictive of MDR and XDR infections in burn patients include prolonged hospitalization, invasive device use (ventilation, central venous lines, urinary catheters), the extent and depth of burns (total body surface area and full-thickness injuries), prior antibiotic exposure, and malnutrition. These factors highlight the importance of preventive care and antimicrobial stewardship to restrict the spread and emergence of resistant bacteria in this vulnerable population [1,21].

Treating these infections is further complicated by the inherent challenges of burn wound healing. Effective management requires prompt wound decontamination, early excision of necrotic tissue (eschar), and skin grafting to reduce microbial load and promote repair. However, persistent infection with resistant organisms prolongs the inflammatory phase of healing, delays tissue regeneration, and promotes scarring. While topical antimicrobial agents such as silver-based creams and mafenide acetate remain integral to burn care, their effectiveness is increasingly threatened by resistance patterns and secondary fungal infections arising from prolonged antibiotic use. Systemic antibiotics often have limited penetration into the avascular burn eschar, reducing treatment efficacy [29,30].

The clinical and economic burden of antibiotic resistance in burn wound infections is substantial. Burn patients with infections exhibit over twice the mortality rate compared to their non-infected counterparts. Additionally, these infections lead to prolonged hospitalization due to difficulties in infection control, more extensive surgical interventions, and the requirement for specialized wound care. These factors collectively increase direct medical costs for antibiotics, dressings, and supportive therapies, as well as indirect costs such as loss of productivity and long-term disability. The heavy toll of antibiotic resistance on patient outcomes and healthcare systems underscores the critical need for preventive and control measures (Figure 2) [4,11,29].

4. Future Perspectives

Future advancements are likely to emerge from several promising avenues, starting with improvements to diagnostic tools. Rapid molecular and genomic techniques enabling real-time identification of pathogens and resistance genes could revolutionize targeted therapy, minimizing the reliance on empirical broad-spectrum antibiotics that foster resistance development. Alongside diagnostics, the development of novel antimicrobial agents and strategies is urgent given the scarcity of new antibiotic classes effective against resistant strains [31,32].

To combat antibiotic resistance in burn wound infections, several evidence-based strategies have been proposed and successfully implemented: 1) Structured programs optimizing antibiotic use by guiding empirical therapy based on local antibiograms and susceptibility profiles reduce unnecessary antibiotic exposure and subsequent resistance development [29]. 2) Early excision of burn eschar limits bacterial reservoirs. Meticulous wound inspection and adherence to hygiene protocols reduce the nosocomial spread of resistant pathogens [29,30]. 3) Topical agents such as mafenide acetate and silver-based dressings reduce microbial load. Alternative agents such as acetic acid and sodium hypochlorite solutions offer broad-spectrum antibacterial activity and may prevent biofilm formation and fungal colonization without promoting significant resistance [29]. 4) Continuous wound culture surveillance guides timely adjustment of systemic and topical antibiotics. This enables targeted treatment of emerging resistance patterns and promotes judicious use of narrow-spectrum agents where appropriate [10,11]. 5) Coordinated management by burn surgeons, infectious disease specialists, pharmacists, and wound care experts increases therapeutic success and reduces infection-related mortality by ensuring comprehensive, patient-centered care plans [4].

6) Prevention must extend beyond the hospital setting, emphasizing public education and cultural change to reduce burn incidence. Campaigns aimed at children and adults with high-risk occupations—such as agricultural workers, industrial laborers, and kitchen staff—can promote safe handling of heat sources and chemicals. Early recognition and prompt first aid, including cooling burns with running water and avoiding harmful traditional remedies, can significantly reduce infection risk and improve outcomes. Collaboration among schools, workplaces, and healthcare providers is essential to fostering awareness and equipping communities to respond appropriately to burn injuries [30].

7) Interventions that support immune function, metabolic balance, and psychological well-being post-burn may reduce infection susceptibility and accelerate rehabilitation. 8) Digital health tools and telemedicine could play an increasingly important role in long-term follow-up, patient education, and early detection of complications, thereby improving the quality of life for survivors [33]. Overall, the future of burn wound infection management is likely to be shaped by integrated technological innovations, novel therapeutics, personalized medicine, and collaborative care models. These advances aim not only to overcome the daunting challenges of antibiotic resistance but also to improve healing, reduce morbidity and mortality, and enhance the holistic recovery of burn patients.

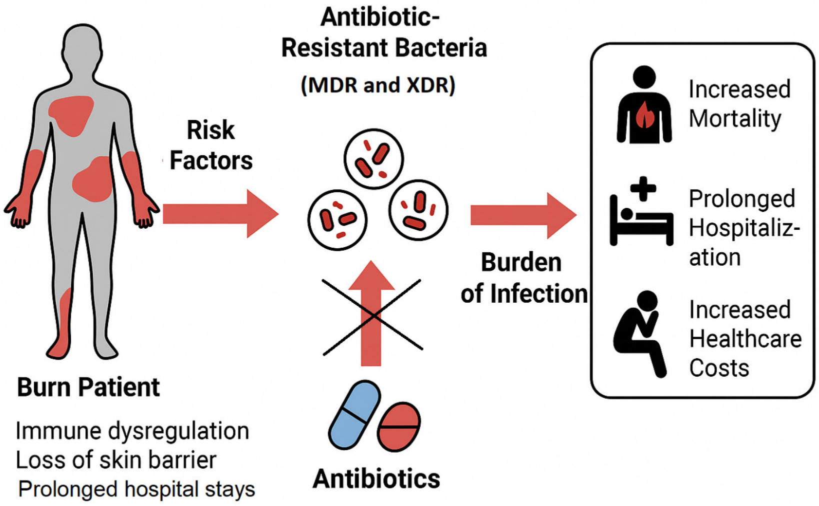

Figure 2. Burn patients are at high risk of infection due to immune dysregulation, loss of skin barrier, and prolonged hospital stays, leading to increased exposure to antibiotic-resistant bacteria (MDR and XDR). This figure illustrates how these risk factors contribute to a greater burden of infection, which is not easily controlled by antibiotics, resulting in increased mortality, longer hospitalizations, and higher healthcare costs.

5. Conclusion

Burn wound infections remain a significant challenge in clinical care due to the high prevalence of multidrug- and extensively drug-resistant bacterial pathogens. Factors such as immune dysregulation, loss of the protective skin barrier, prolonged hospitalization, and invasive procedures significantly contribute to the risk of colonization and infection with resistant organisms. These infections severely impede wound healing, increase inflammation, and elevate mortality and healthcare costs.

Effective management requires a multidisciplinary approach, including early wound excision and grafting, stringent infection control measures, regular antimicrobial susceptibility monitoring, and prudent antibiotic use guided by local resistance patterns. Emerging diagnostic technologies and novel therapeutic agents offer hope for overcoming current limitations. Prevention efforts that extend beyond clinical settings into public education and safety awareness are essential to reduce burn incidence and subsequent infection risk. Continued research and integrated care models are imperative to address the evolving problem of antibiotic resistance in burn wounds, ultimately improving patient outcomes and quality of life.

Authors' contributions

MTM: Conceptualization, Writing original draft, and editing. AB: Writing original draft. SSV: Writing original draft. ZTM: Writing original draft. MFK: Writing original draft, and editing. MM: Project administration and Supervision. All authors read and approved the final version of manuscript.

Conflict of interest

No potential conflict of interest was reported by the authors.

Ethical declarations

Not applicable.

Financial support

Self-funded.

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