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Bacterial Isolates from Wound and Burn Infections in Three Iraqi Provinces (Baghdad, Sulaymaniyah and Misan): A Review of Epidemiology, Pathogenesis and Antimicrobial Resistance

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ABSTRACT: ***Background:*** *Burn and wound infections represent a significant public health burden in Iraq, contributing to high rates of morbidity and mortality. Decades of armed conflict, economic instability and deteriorating healthcare infrastructure have severely compromised the capacity of Iraqi hospitals to prevent and manage these infections effectively.* ***Objective:*** *This comprehensive review consolidates scientific literature to evaluate the epidemiology antimicrobial resistance patterns, pathogenic mechanisms and clinical outcomes associated with bacterial wound and burn infections across Iraqi hospitals and healthcare systems.* ***Methods:*** *A systematic search identified studies on bacterial isolates from wound and burn infections across Iraqi hospitals, with data drawn primarily from Sulaymaniyah, Baghdad and Misan provinces as geographically and demographically representative samples rather than a comprehensive national survey.* ***Results:*** *Pseudomonas aeruginosa, Staphylococcus aureus and Acinetobacter baumannii collectively account for the majority of wound and burn infections. Multidrug-resistant (MDR) pathogens are highly prevalent, including MRSA rates approaching 87%, ESBL-producing Enterobacteriaceae and carbapenem-resistant Gram-negative organisms. Nosocomial transmission is the predominant route of spread, driven by overcrowding, poor infection control and contaminated environments. Burn patient mortality ranges from 13–28%, with sepsis responsible for 44–65% of burn-related deaths. Virulence factors, biofilm formation and bacterial adaptation to wound environments contribute to treatment failure and infection persistence.* ***Conclusions:*** *Urgent improvements are required in antibiotic stewardship, infection prevention, epidemiologic surveillance and evidence-based treatment protocols across Iraqi healthcare facilities. Inter-hospital collaboration and investment in regional microbiological diagnostic capacity are essential to addressing this growing public health crisis.*

KEYWORDS: *Pseudomonas aeruginosa; Staphylococcus aureus; Nosocomial infection; Biofilm; Virulence factors*

INTRODUCTION

Burn injuries are one of the most substantial sources of trauma around the world and developing countries have much higher rates of incidence and mortality than developed nations [1]. The World Health Organization estimated that there were 180,000 deaths due to burns worldwide in 2018, with the majority of these deaths happening in developing countries [2]. Iraq has seen an increase in burn injury-related morbidity and mortality due to 30 years of protracted conflict, socioeconomic instability, infrastructure decay and continued limitations of the healthcare system [3]. According to Iraqi health surveillance data, an estimated 92,734 burn patients require medical treatment each year, which is an average of about 250 patients per day between all the healthcare facilities in Iraq [4]. Reports of

approximately 6,149 burn cases were reported in Iraq in 2015 and it was estimated by the WHO that approximately 6,000 fire-related deaths occur each year [5]. Wound infections, distinct from burns, are a major problem in Iraq. They include surgical wounds, traumatic injuries (lacerations, blast, crush) and chronic wounds (diabetic ulcers). Conflict-related wounds with foreign bodies and delayed debridement are common. Bacteriology overlaps with burns [*Staphylococcus aureus (S. aureus)*, *Pseudomonas aeruginosa (P. aeruginosa)*] but more often includes Enterobacteriaceae and *Acinetobacter baumannii (A. baumannii)*. Resistance patterns (MRSA, ESBL, carbapenem-resistance) are similarly severe [3]. Infection by bacteria complicates the clinical course of burn injuries with sepsis accounting for 51-65% of burn related deaths in Iraq [6]. Approximately 53.3% of the deaths of burn patients are due to multiple organ failure and 44.4% are due to septicemia, thus showing the importance of appropriate infection prevention and management strategies [7].

The increase and distribution of multidrug resistant organisms associated with wound and burn infections has made the treatment of burn injuries very difficult around the world [8]. The scenario is compounded in Iraq with the combination of multiple risk factors including the lack of available Antibiotics during the sanctions period, unfettered access and widespread empirical use of antibiotics after the sanctions period, transmission within hospitals due to overcrowding and contaminated environmental conditions have produced an environment conducive to the development and spread of resistance pathogens [9], [10]. A major limitation of this review is the absence of recent, published microbiological data from the majority of Iraqi provinces. While Iraq has 19 governorates, this review was only able to identify peer-reviewed studies reporting bacterial isolates from wound and burn infections in three provinces: Sulaymaniyah (north), Baghdad (center) and Misan (south). No eligible studies meeting our inclusion criteria were found for the remaining 16 provinces, including Anbar, Nineveh, Kirkuk, Najaf, Karbala, Wasit, Diyala, Babil and others.

This thorough literature review explores the bacterial epidemiology of burn and wound infections in Iraq and specifically addresses Antimicrobial Resistance in specific provinces. Patterns, pathogenic mechanisms, including virulence factors and biofilm development on burn and wound infections and how they relate to clinical management practices and public health interventions.

THE EPIDEMIOLOGY OF BURN AND WOUND INFECTIONS IN IRAQ

Demographic Characteristics

Epidemiological studies conducted in numerous Iraq's provinces have demonstrated an observable relationship between demographics and the pattern of acute burn injuries [11], [12]. In Sulaymaniyah Province, an extensive study indicated that in the course of the year, there were 947 recorded cases of acute burn injuries, with women making up 53.5% of the population and men 46.5% [13]. Children ages zero to fourteen represented 41% of admissions. A similarly high incidence was reported from Misan, with a 2:1 male-to-female ratio and close to one-third of all patients being children ages 0-5 [14]. There are two age peaks of instability, early childhood and young adulthood. A report from the Baghdad Burn Hospital estimated that 37% of all patients treated were between the ages of twenty-one and thirty and the next largest grouping being under five years of age. An overwhelming majority, 67.1%, of those treated at this urban tertiary facility were males and it is expected that many of these are work-related injuries.15 Of note, 34.8% are military personnel demonstrating the effect of the ongoing challenges from security issues on the epidemiology of burn injuries in Iraq [15]. Socioeconomic status significantly influences the patterns of burn injuries in Iraq. The overwhelming majority of burn patients (60.7 percent) had a maximum level of education equal to primary school and 53 percent of the patients said they came from a poor socioeconomic background. Approximately 84 percent of those injured sustained burns as a result of fires that occurred in their home and 91 percent of the patients that sustained burn injuries were alone within their home at the time of injury. This indicates that burn injuries are preventable and provides opportunities for public health intervention.

Mechanisms and Severity of Injury

According to studies conducted in Iraq regarding the mechanisms of burn injuries, the primary method of injury is through either flame or scalds. For example, 59 percent of the total burn injury cases in the Sulaymaniyah Province were flame injuries, while 37 percent were scald injuries.13 Misan reported a similar trend: flame burns accounted for 51 percent and scalds accounted for 41.7 percent of the reported burn injuries. A report published by the Burn Hospital in Baghdad indicated that 71.6 percent of patients received flame burns while 23.4 percent received scald injuries.15 The other

means of injury for patients containing either contact, electrical (12 percent), chemical (10 percent) and radiated (5 percent) burns demonstrate the differences in the mechanisms of burn injuries among Iraqi patients [16]. Causative agents were also reported to be different for patients based upon their age and location. For example, pediatric patients had tea pots as the most significantly identified source of burn injuries, at 42 percent, followed by Kerosene stoves at 36 percent and burned most commonly in either sitting rooms (53 percent) or kitchens (36 percent) [4]. Patients with intentional self-injuries used kerosene in 82 percent of burn cases [17]. The cause of burn injuries in approximately 40 percent of domestic situations was hot water and hot liquids (including steam); hot tools represented 18 percent of the total and open flame represented 15 percent.16 Variations in the total body surface area (TBSA) indicated a large difference in incidence among different patient populations. The average TBSA burned in Misan was 30% (range 18 - 45%), with some patients having a 0% TBSA while others had 100% TBSA [14]. The median TBSA reported for Sulaymaniyah was 19%, with about one-half of patients treated in Baghdad suffering TBSA burn injuries of 11-20% [13], [15]. The majority of severe burn injuries suffered by Iraqi civilian burn victims were second-degree (50%), with fewer cases of both third-degree and first-degree burns [15]. The anatomical distribution of these severe burn injuries demonstrated that 48% of applicants had burns to their hands, 16% to their legs, 12% to their abdomen and 10% to their chest [18].

Mortality Rates and Predictive Factors

The in-hospital mortality rates for burn patients within Iraq show a troubling trend. The reported ranges of in-hospital fatalities among the burn patient population ranged from 13%-29.4% based upon the patient population being treated and the facilities providing care [9], [15], [18]. The documentation from Misan showed a mortality rate of 22%, whereas the rate reported for Sulaymaniyah hospitals was between 27–28% for admitted burn patients [14], [18]. These figures directly exceed the reported mortality rates of burn patients in high-income countries where advanced resources for treating burn patients are available. The most significant predictor of burn-patient mortality identified in multiple studies is the total body surface area burned. Patients with 26–50% of their body burned had a death risk 16.6 times that of patients with 0–25% of their body burnt, whilst patients with 51–75% of their body burned had an almost 87 times greater risk than the same category, all according to their odds ratio or odds of survival [19]. Additionally, in one study examining over 75% TBSA burnt patients showed a 63% risk of dying, as opposed to just 0.6% risk of dying in patients with less than 25% TBSA burned. The almost universal risk of dying from a burn covering 75–100% of TBSA should include awareness of this level of burn coverage and highlight the severity of injury to the individual [20]. Furthermore, additional mortality risk factors exist for all patients. One of these factors is the presence of suicidal intent with primary burns resulting in a risk of death that was seven times greater than those with unintentional burns. Even age is a deciding factor in mortality rates, as patients who are 60 years or older had a mortality (OR) risk ratio of 4.8 compared to children younger than five years of age (95% CI 1.3–20) [2]. Another risk factor for increased mortality for both genders was the diagnosis of a smoke inhalation injury. Furthermore, seasonal variation in burn injuries also exists, with burn injuries occurring during the autumn months rated significantly higher for mortality risk (OR 4.6).

The patient's length of stay in the hospital varies between 5-8 days depending on the degree of the burn and the capability of the hospital to accommodate patients with a significant burn. In the same study, suicidal burn patients treated for an extensive percentage of TBSA (total burn spreading) indicated a median length of stay of only 5 days (interquartile range of 1–12 days), in addition to extending or increasing the amount of time they were allowed to remain in the hospital, resulting in fewer patients who received successful treatment [20]. The trend for patients who committed self-harm by burning is seen in a high percentage of patients treated in hospitals, particularly among young women. Females of reproductive age are primarily affected by self-inflicted burn injuries, which are typically associated with domestic violence, socioeconomic circumstances and depression. These individuals generally have a large total body surface area (TBSA) burned (median 80%, interquartile range 60-95%) and are at an extremely high risk of dying from their injuries (72.6% mortality rate). The majority of patients were not educated (92%), married (55%) and considered themselves economically disadvantaged (53%). This demonstrates the intersection of gender-based violence, mental health issues and a lack of access to psychosocial services in the Iraqi community [21].

THE BACTERIAL EPIDEMIOLOGY OF WOUNDS AND BURNS IN IRAQ

Predominant Bacterial Isolates

In Iraq, different geographic regions have demonstrated consistent trends in the types of bacteria that colonize burn wounds and surgical wounds. The data supporting this conclusion come from multiple studies performed on patients with burn injuries at sites throughout Iraq and represent a wide variety of lesion sites and research design [22]. One study conducted at Al-Sadr Teaching Hospital in Misan evaluated 105 burn patients and provided a broad overview of the type of bacteria identified in these patients. Overall, researchers identified 9 different species of bacteria from the 105 patients and noted that *P. aeruginosa* was the most commonly identified pathogen (20% of all isolates), followed by *S. aureus* (17.14%), *Enterobacter* spp. (16.19%), *Proteus vulgaris* (13.33%), Proteus mirabilis (10.47%), Escherichia coli (7.6%), *Klebsiella pneumoniae (K. pneumoniae)* (6.6%) and two other bacteria (Staphylococcus lentus and Aeromonas sobria) that were isolated at much lower rates (4.7% each) (16,23). Another recent paper published from research done at Sulaimani Burn Hospital analyzed 1402 bacterial isolates from 760 patients admitted with burn injuries and found that *S. aureus* was the most common pathogen (34% of the isolates). The second most common pathogen was *P. aeruginosa* [4]. There are a number of different infections associated with burns, with the most common infection being *S. aureus* (25%) and other organisms, such as *A. baumannii* (20%) and *P. aeruginosa* (16.7%), found amongst the top three pathogens, followed closely by *K. pneumoniae* (13.3%), *Escherichia coli (E. coli)* (11.7%) and Proteus mirabilis (10%) as well as Burkholderia cepacia (3.3%), a bacterium common in burn infections in the area, than in the other areas of the world [23], [24]. Multi-hospital studies conducted in Baghdad to identify the most common bacteria associated with burns have corroborated the data found in the Sulaimaniyah study, but have included data from 200 burn samples identified as containing *S. aureus* (31.3%), *Staphylococcus epidermidis* (9.1%), *P. aeruginosa* (16%), the Enterobacteriaceae family and other members of the genus *Enterobacter* [4].

Studies analyzing the results of previous studies to compare bacterial colonization of burn wounds in Iraqi burn units from the Sulaimaniyah study also support these findings, as *S. aureus* is often present early in the colonizing phase of burn wound infections [25], [26]. Over the past few decades, the bacterial ecology associated with burn wounds has shifted toward a predominance of aerobic gram-negative organisms, largely due to selective pressure from broad-spectrum antibiotics. While anaerobic bacteria may contribute to burn wound infections, their role is less significant compared to aerobic gram-negative pathogens such as *P. aeruginosa* and *A. baumannii.* During both the pre-antibiotic and early antibiotic eras, *S. aureus* was responsible for the majority of death associated with septic complications due to invasive burn wounds [27]. The introduction of broad-spectrum antibiotics has significantly changed the epidemiology of burn wound infections, with a clear shift toward an increasing prevalence of gram-negative bacteria in the invasive burn wound setting [28]. As illustrated by this transition in the epidemiology of burn wound infections, gram-negative organisms possess biological properties that enhance their ability to thrive within a damaged burn wound environment and the antibiotics used to kill the susceptible gram-positive organisms provide a substantial selective pressure toward the gram-negative organisms. For example, *P. aeruginosa* has considerable adaptability to environmental stresses, intrinsic resistance to a large number of antibiotics and complex virulence factors that promote successful colonization of the damaged tissue within the burn wound environment [29]. *P. aeruginosa*'s ability to form biofilm, utilize multiple nutrient sources, rapidly acquire and disseminate resistance determinants through horizontal gene transfer make it highly successful from an evolutionary standpoint within the burn unit setting [30], [31].

A. baumannii is of particular concern as it has emerged as a powerful pathogen in both Iraq and the wounded service members in particular. Extensive infection with *A. baumannii*i was documented by military medical personnel on service in Iraq during Operation Iraqi Freedom and this resulted in the widespread adoption of the term "Hulk-a-baumannii" by the media to describe the phenomenon [32]. Historically, *A. baumannii* was considered low-virulence species with little clinical importance but it has changed significantly and has become an important nosocomial pathogen with extensive antibiotic resistance and persistence under most healthcare conditions [33], [34]. The unique combination of traumatic injuries associated with wartime injuries, an extensive system of long-distance medical evacuations, the extended period of antibiotic therapy and environmental contamination of healthcare facilities in Iraq created optimal conditions for the selection and dissemination of *A. baumannii* [11].

PATTERNS OF ANTIMICROBIAL RESISTANCE

Resistance in Gram-positive Organisms

1 MRSA (Methicillin-resistant *Staphylococcus aureus*)

The prevalence of MRSA in Iraqi burn units is of concern because MRSA has been reported to occur in much higher proportions than in most developed countries. The results of the comprehensive Sulaimani study showed that 87.1% (176 of 202) of all *S. aureus* isolates were MRSA, reflecting the near-universal presence of beta-lactam resistance in staphylococcal infections of burn wounds [8]. The exceptionally high proportion of MRSA isolates severely limits the first-line choices for therapy and requires the use of the glycopeptide antibiotics such as vancomycin as a primary empirical therapy. In addition to methicillin resistance, Iraqi patients with burn injuries have shown other notable resistance patterns with their *S. aureus* isolates. For instance, a study from Baghdad found that 86.6% of *S. aureus* isolates were resistant to amikacin and therefore the effectiveness of aminoglycosides was also compromised [23]. In cases of burn-associated infections due to bacteremia occurring in Northern Iraq, all *S. aureus* isolates were shown to exhibit methicillin resistance and therefore the authors recommended that while vancomycin and teicoplanin would be valuable components of empirical therapy for sepsis [35]. Infections due to *S. aureus* in burn patients are caused by a variety of virulence factors that promote tissue colonization and invasion. A fibrinogen/fibrin binding protein is responsible for the attachment and invasion of *S. aureus* into blood clots and destroyed tissue; thus, *S. aureus* is an ideal pathogen for creating infections in both surgical and burn wounds [36]. Other bacterial characteristics that contribute to virulence include the production of toxins (Toxic Shock Syndrome Toxin and Enterotoxins), the ability to produce biofilms and the means by which *S. aureus* avoids the immune response through the use of protein A (an immunoglobulin binding protein) [37].

Resistance in Gram-negative Organisms

1 *Pseudomonas aeruginosa*

P. aeruginosa is a multidrug-resistant (MDR) organism with a current presence in all healthcare facilities across Iraq, which will create significant challenges for treatment. The resistance rates were as high at the 60% level for colistin and tobramycin. These results are concerning because colistin is used as a "last resort" for treating carbapenem-resistant Gram-negative infections [38]. Furthermore, the resistance to other antibiotics was also found to be at a high rate, with ciprofloxacin having a resistance of 93.7%, aztreonam 86.8%, piperacillin 85.4%, ceftazidime 82.6%, amikacin 82% and imipenem 79.2% from burn unit studies [39]. Carbapenem-resistance in Iraqi *P. aeruginosa* clinical isolates reached alarming proportions. Reports indicate a 45% imipenem resistance rate and 40% meropenem resistance in the Iraqi burn unit population, which greatly limits treatment options for severe Pseudomonas infections. The data from Iraq indicate an increase in carbapenem resistance, mirroring the same pattern observed globally, but at a much higher percentage than that observed in other areas and regions [40]. The intrinsic resistance mechanisms of *P. aeruginosa* include low outer membrane permeability, constitutively expressed MexAB-OprM and MexXY-OprM and MexCD-OprJ efflux pumps and chrDNA, that encodes an AmpC beta-lactamase [41]. The acquired resistance mechanisms are further complicating the prevention and treatment of serious Pseudomonas infections. Resistance genes for metallo-beta-lactamases have been transferred through horizontal gene transfer of the VIM, IMP and NDM genotype; extended-spectrum beta-lactamases (PER, VEB); and aminoglycoside-modifying enzymes. Mismanagement of antibiotics has resulted in mutations occurring in the genes that encode the outer membrane protein as a result of OprD loss; and mutations in the regulatory genes responsible for the expression of the efflux pumps and in the DNA gyrase/topoisomerase alter the genes to confer quinolone resistance, resulting in the multidrug-resistant phenotype that continues to be found in isolates in Iraq [42].

2 *Acinetobacter baumannii*

Based on the data in hospitals in Baghdad, *A. baumannii* has been shown to have exceptionally high rates of resistance in all classes of antibiotics within the burn units. *A. baumannii* appears to be resistant to many other beta-lactams [23]. A study conducted on the effects of OPERATION ENDURING FREEDOM (OEF)/OPERATION IRAQI FREEDOM (OIF) found that only colistin, polymyxin B and minocycline were still active against 95% or more of the *A. baumannii* isolates in the military hospitals in Iraq. Colistin resistance developed during therapy and was detected in a number

of cases [43]. Resistance patterns in *A. baumannii* isolated from patients in Iraq have been shown to be increasing over time, as evidenced by the decreased susceptibility of *A. baumannii* to imipenem from 87% to 56% during the 2 year period from October 2003 - November 2005. Isolates from deployed patients exhibited a significantly higher incidence of resistance compared with isolates from non-deployed patients, indicating that the context of the Iraqi medical environment and combat injury may have favoured the selection of extremely resistant strains [44]. The mechanisms of resistance of *A. baumannii* are varied and most often occur in combination with one another. The mechanism of resistance to carbapenems is generally by the production of carbapenemases (OXA-type enzymes and metallo-beta-lactamases) through the loss or alteration of the outer membrane porins and the upregulation of the efflux pumps [44]. In addition, *A. baumannii* has the unique ability to acquire resistance genes through horizontal transfer and the ability to survive on the environmental surfaces for significant periods of time, providing it with the opportunity to persist in the hospital setting [45].

3 Enterobacteriaceae and ESBL Production

Among the organisms isolated from the burn centres in Iraq, Enterobacteriaceae including organisms that produce extended-spectrum beta-lactamases (ESBLs), are widespread, making therapy for the infections caused by these organisms very limited. Data from Mosul showed that 68.5% of the Gram-negative bacilli isolated from burn wounds showed extensive drug-resistant (XDR) patterns. As seen, 30.71% showed pandrug resistance (PDR), which will not respond to any currently available antibiotics [46]. High rates of colistin resistance have also been reported among Enterobacteriaceae in Iraq. For instance, a study from Baghdad reported that 100% of *K. pneumoniae* and P. mirabilis isolates were resistant to colistin and tobramycin [23]. However, these exceptionally high rates are not consistently reported across all Iraqi studies. A more recent multinational review found that while Iraq has high levels of cephalosporin and colistin resistance, colistin resistance in Enterobacteriaceae was not universal [11]. Furthermore, a study from Karbala reported a colistin resistance prevalence of only 6% among *E. coli* isolates [10]. The discrepancy highlights the need for large-scale, standardized surveillance to accurately determine the true burden of colistin resistance in Iraq [23]. ESBL producing Enterobacteriaceae has been well established as a cause of burn-related bacteremia in Iraq. A high proportion of Enterobacteriaceae strains made extended-spectrum beta-lactamases and required the use of the carbapenem class of antibiotics [35]. The emergence of carbapenemase production within this group of ESBL producing Enterobacteriaceae has created a new group of extensively resistant organisms with no treatment options [47]. *Enterobacter* organisms are increasingly associated with nosocomial infections in immunocompromised burn patients. Enterobacteriaceae, such as *Enterobacter* aerogenes and *Enterobacter* cloacae, are capable of acquiring mobile genetic elements which confer resistance and virulence factors, thus strongly contributing to their enhanced pathogenesis [4]. The percentage of *Enterobacter* species identified as isolates in the Misan study was 16.19%, ranking them as the third most commonly found pathogens after *P. aeruginosa* and *S. aureus* [22].

Severity of Multidrug Resistance

Multidrug-resistant organisms appear at a much higher frequency in Iraqi burn units than in most developed countries.The longitudinal research performed on burn victims in Sulaimani, Iraq demonstrated the emergence of multidrug resistant organisms and their associated virulence factors within the *P. aeruginosa* species. Of the 1,402 isolates, 715 (51%) had multi-drug resistance (MDR) defined as the presence of MDR in three or more classes of antibiotics [4]. Non-fermenting bacteria, including *P. aeruginosa*, had a significant percentage of the isolates expressing MDR at 28.8% which creates a significant challenge for physicians selecting empirical antibiotic therapy and leads to a higher failure rate and therefore a greater incidence of death in the Iraqi burn patients. A clear relationship exists between the development of antibiotic resistance and the presence of antibiotic selection pressure and horizontal gene transfer within the burn wounds of the infected patients [24]. There are numerous virulent mechanisms employed by *P. aeruginosa* that contribute to the organism's ability to cause serious tissue damage and systemic illness in patients with severe *P. aeruginosa* burn infections. High prevalence rates of genes that control the production of the virulence factor exotoxin A (toxA) have been documented in the literature related to *P. aeruginosa* burn wound isolates. The presence of this gene has been demonstrated in at least 90% of the burn wound isolates studied. The role of Exotoxin A is to inhibit the eukaryotic protein synthesis by ADP-ribosylation of the eukaryotic elongation factor two (eEF2) resulting in host cell death and tissue necrosis [26]. The expression of elastase (lasB), another virulence factor, is similarly prevalent among the burn wound isolates (>85%). LasB elastase is capable of destructing elastin, collagen, immunoglobulins and complement proteins.

This clear example of a virulence factor that aids in the invasion of tissue and provides the bacteria with a source of nutrients while lowering the host's immunity [48]. Both genes responsible for the production of phospholipase C (plcH), a virulence factor that acts by damaging the membrane of the host's epithelial cells, were present in over 80% of burn isolates. Through hydrolysis of the membrane phospholipids, the phospholipase C enzyme creates a pathway for the bacteria to migrate into areas otherwise considered to be sterile [49]. Neuraminidase (nan1) exhibited high prevalence rates (60-70%) among burn unit isolates. The neuraminidase cleaves the Sialic acid residues from host glycoproteins and glycolipids creating new attachment sites for the bacteria and enabling the bacteria to avoid the host immune system [50]. Iron-related virulence factors such as iron acquisition systems by *P. aeruginosa* represent critical virulence factors for the pathogenesis of *P. aeruginosa* burn wound infections. *P. aeruginosa* has developed two methods of acquiring iron for growth; these are ruled by two distinct siderophore systems, pyoverdine and pyochelin, both of which are capable of removing iron from the environment or other iron-containing proteins. The gene pvdS that encodes a sigma factor for the regulation of the synthesis of the siderophore pyoverdine demonstrates a ten-fold increase in the level of the expression of this gene when compared to the levels found in planktonic cultures [51]. The production of pyocyanin, the blue-green phenazine pigment produced by *P. aeruginosa*, results in the release of reactive oxygen species (ROS) and damage to the host cells. Pyocyanin is produced to a greater degree in burn wound exudates than standard laboratory media demonstrating that the unique microenvironment of a burn wound enhances the production of this virulence factor [52]. A third virulence factor produced by *P. aeruginosa* is rhamnolipids. Rhamnolipids are polysaccharide glycosides that can function both as biosurfactants and as aids for swarming motility and biofilm formation. Rhamnolipids also have increased expression (rhlA gene) within the burn wound environment leading to increased colonization and development of biofilm structure within the burn wounds [53].

Biofilm Formation by Isolates from Burns

There is evidence that biofilm formation is an effective pathogenic mechanism employed by bacteria to persistently colonies burn wounds even when antimicrobial therapies and host immune responses are being applied, thus preventing clearance of infection by these treatments. Biofilms are three-dimensional structures created by microbial cells that form communities encased in an extraneous polymeric matrix (EPMS) [43]. The ability to produce biofilm is directly related to the number of antimicrobial agents to which a strain is resistant. The level of biofilm production was greater for multidrug-resistant *P. aeruginosa* isolated from burn wounds than for Non-MDR isolates ($P<0.001$) [38]. The fact that biofilm formation and resistance have a close relationship indicates that they may share similar regulatory mechanisms or may be co-selected because *P. aeruginosa* is commonly present in burn units. The use of animal models demonstrated that once infected, burn wounds were rapidly colonised by *P. aeruginosa* and that the bacterial load stabilized at about 1×10^9 CFU/g of burn tissue by 7 days post-inoculum, regardless of whether the initial inoculum is low or high [26], [52].

Invasion of tissues below the surface of the burn was shown histologically and by scanning electron microscopy to occur between 500 and 600 µm below the burn's surface. This deep invasion of tissues may allow *P. aeruginosa* to enter the bloodstream and lead to the development of septic complications [26]. Molecular mechanisms regulating biofilm formation involve co-regulation of several genetic systems. A quantitative gene expression study of *P. aeruginosa* obtained from burn wounds indicated a significant increase in alginate biosynthesis gene expression (algD, alg8, algE), Pel polysaccharide production gene expression (pelB, pelC, pelD) and virulence factor gene expression (lasA, lasB, pvdS) when compared to *P. aeruginosa* grown in planktonic culture [26]. The amount of alginate biosynthetic exopolysaccharide extracted from burn tissues was found to be 8-12 times greater compared to that found in planktonic cultures. The substantial 4-10-fold increase in the total number of genes responsible for pel polysaccharide synthesis has now been confirmed. However, no observed increases in abundance of the pslA and pslB genes have been found in the psl polysaccharide system. Therefore, it can be speculated that there are differential types of regulation for these two polysaccharide systems based on the environmental cues they receive from their surroundings and therefore support the finding that each polysaccharide system is likely to respond differently to the same environmental cues [50]. The primary means by which bacteria regulate biofilm formation and virulence factors in the burn environment is through a form of cell density-dependent regulation known as "quorum sensing". As previously stated, there are a number of different quorum-sensing systems in *P. aeruginosa*; each of these has multiple quorum-sensing circuits and most of these circuits regulate hundreds of downstream genes [52]. The degree of activation of quorum-sensing genes in the fluid collected

from a burn wound is moderate, which also correlates with great activation of downstream genes. This supports the concept that the burn environment provides an environment that encourages the development of pathogenicity through quorum-sensing mechanisms [54]. Creation of a biofilm allows *P. aeruginosa* to create a significant increase (potentially 100-1000 times greater than treatment of planktonic bacteria with antibiotics) in the number of bacteria that are affected by the antibiotics used to try to kill them. There are many ways to achieve this type of resistance. For example, there may be a physical barrier to the penetration of antibiotics through the biofilm matrix; this barrier could be due to decreased metabolic activity in biofilm cells which exist within the deeper layers of the biofilm; this decreased metabolic activity would provide an environment in which bacteria could more readily activate a stress response; persistent cells that exist in a dormant state may provide an additional level of resistance. Thus, there are a number of reasons why it is generally believed to be difficult to treat established infections in burn wounds with antibiotics that are effective against the same organisms in vitro in the planktonic state [46], [55]. *P. aeruginosa* is not the only biofilm-forming pathogen in burn wounds. *S. aureus* (including MRSA) produces biofilms via the ica operon and Bap protein, promoting glycopeptide resistance. *A. baumannii* forms robust biofilms on burn eschar and devices through pili, vesicles and BapAb, enhancing persistence and MDR. K. pneumoniae and Enterobacteriaceae use type 3 fimbriae (MrkA) and exopolysaccharides for biofilm-mediated colonization and carbapenem resistance. Overall, biofilm formation across multiple genera universally compromises antibiotic efficacy and immune clearance.

Risk Factors and Transmission Dynamics

Nosocomial transmission is the predominant route by which infections are acquired in burn units in Iraq. Community-acquired infections are substantially less common than nosocomial infections in burn units in Iraq.There are several things happening at once that allow for the easier spread of germs to be spread in hospitals: 1) the number of patients in burn units far exceeds the amount of space in those units; 2) persistent contaminated environments with resistant bacteria can remain alive for long periods of time on surfaces; 3) a lack of compliance with hand hygiene standards; 4) and inadequate isolation capability for colonized or infected patients [22], [56]. Bacterial samples taken from burn wounds (via swabs) indicate that 83 percent of all samples will grow bacteria and only 16 percent of those samples remain sterile throughout the duration of hospitalization.8 This means that virtually all hospital patients will have been in contact with something that could potentially make them sick due to an exposure to a pathogenic organism while in the hospital. Also, the average number of organisms per sample was 1.5, meaning that people are often infected with more than one organism at a time. This makes it more difficult for physicians to determine which treatment to reatment to allow horizontal gene transfer of any resistance mechanisms developed by one species against another [8]. An example of a problem that hospitals in Iraq face with nosocomial pathogens is *A. baumannii*. This organism has been known to survive on dry surfaces like beds, door knobs and medical devices for weeks, so it will remain as an environmental reservoir for an extended period of time [57]. An incident at a military medical facility showed that a thorough reinforcement of standard infection control measures (such as washing hands before and after patient care, strict adherence to isolation practices and thorough cleaning of the environment) was crucial in preventing further nosocomial transmission [58].

Sources of Bacterial Contamination

The colonization of a burn wound with bacteria can come from multiple points, all of which add to the overall burden of infection. Endogenous flora is one source, as it comes within hours after burn injury, surviving commensal organisms from adjacent unburned skin and intestinal flora colonize the denuded tissue [59], [60]. Environmental contamination is another major source of hospital-acquired pathogens; surfaces in hospitals, equipment used for patient care, water sources (e.g., tap and hydrotherapy tanks) and fomites (non-living objects), can all potentially harbor infectious microorganisms [61]. Although the risks of contamination via the air are less pronounced than via contact transmission, the establishment of colonization on exposed/denuded tissue by Aspergillus and other environmental fungi in facilities with poorly designed ventilation systems may become an increasingly important problem during the healing process of tissues [62]. At present, compliance with healthcare personnel hand "hygiene" practices is the most pivotal determiner of rates of cross-transmission of bacteria within burn units. Multiple studies have demonstrated that healthcare personnel with contaminated hands are often the primary route by which patients acquire hospital-acquired pathogens [63]. Non-compliance with appropriate hand hygiene protocols in

between patient contacts in addition to inadequate glove use practices has been shown to promote the transmission of multi-resistant organisms throughout burn units. Healthcare workers' hands may harbor MRSA and multir-esistant *P. aeruginosa* and are typically contagious for prolonged periods, unless these pathogens are properly removed through hand washing or the use of an alcohol-based hand sanitizer [64].

Infection of Wounds Resulting from War

The Iraq War has presented a unique series of wartime injuries over the duration of the conflict that display an array of differing types of bacteriology. As is the pattern of injuries sustained, those injuries sustained due to the blast from an improvised explosive device, penetrating injuries from firearms and/or shrapnel and crush injuries from collapse of dirty or dangerous buildings create multiple wounds causing extensive damage to tissue structures, an extensive reduction in the amount of blood flow to tissue and the introduction of foreign bodies into the wound—creating an ideal environment to sustain the establishment and growth of bacteria and ultimately lead to tissue invasive infection [65]. Studies conducted on the Iraqi civilian population, to date, have identified specific and unique patterns of types of bacteria that have caused wound infections in Iraqi civilian patients suffering from war injury. *K. pneumoniae* (13%) followed by *A. baumannii* (11%) and *P. aeruginosa* (10%) were the Predominant organisms isolated from infected war wounds [66]. The tendency of these gram-positive and gram-negative isolates to be resistant to many antibiotics demonstrated broad resistance to antibiotic drugs. In contrast, there was an early identification of gram-positive organisms that were skin commensals in wounds of military personnel, as reported in surveillance data.74 Early surveillance data indicated that only 5% of the wound cultures were identified as gram-negative organisms. However, by 9 months after injury, approximately 27% of the military patients presented with wound infections primarily due to gram-negative bacteria in comparison to the initial patterns of gram-positive organisms in damaged tissue [67]. The presence of metal fragments, contaminated soil and decomposing organic matter in a traumatic wound presents additional challenges to the body and healing. These foreign materials can be a source for bacterial colonization, form biofilm and impede the body's immune defense mechanisms [68]. The time delay between the initial injury and surgical intervention, a result of war dynamics and limited surgical resources, allows bacteria to proliferate after surgical management. The combination of the severity of wounds along with the delay in definitive surgical management have resulted in the selection of multi-drug resistant pathogens adapted for life in damaged tissue [69].

CLINICAL IMPLICATIONS AND TREATMENT STRATEGIES

Empirical Antibiotic Selection

Due to the high rate of multidrug resistance organisms in burn wounds and wound infections from Iraq, local resistance patterns must be followed carefully when selecting empirical therapy antibiotics. Data from studies of burnassociated bacteremia illustrate that a combination of a glycopeptide such as vancomycin or teicoplanin, as well as a carbapenem agent with anti-pseudomonal effects (i.e. imipenem, meropenem, doripenem) are required for good empirical antimicrobial coverage for gram-positive organisms (especially MRSA) [35]. The best empirically effective antibiotics for infected/colonized gram-negative bacteria were imipenem and amikacin; however, the prevalence of resistance has increased for both imipenem and amikacin [35]. The progressive increase in the number of carbapenem-resistant Enterobacteriaceae and Pseudomonas spp. has drastically reduced the number of viable therapeutic options. When colistin fails (resistance rates 60–100% in Iraq), alternatives are limited: tigecycline (not for *P. aeruginosa*), Fosfomycin, high-dose meropenem (if MIC low), or combination therapy (e.g., colistin + meropenem + rifampicin). If no agent is susceptible, management relies on source control (surgical debridement, drainage) and supportive care. Novel agents (cefiderocol, phage therapy) are not available in Iraq. Combination Antibiotic Therapy (CAT) instead of a monotherapy for Burn Injury Infections (BIIs) caused by multidrug-resistant organisms is an alternative strategy to improve patient outcomes. Synergisms between antibiotics with different mechanisms of action (e.g., beta-lactam and aminoglycoside or fluoroquinolone) may lead to increased bacterial kill rates and limit the emergence of resistance to both agents during the treatment of BIIs. However, in vitro studies of Colistin combined with either Carbapenems, Rifampicin or Fosfomycin had some clinically beneficial outcomes for patients infected with Carbapenem-Resistant *P. aeruginosa* or *A. baumannii* [70].

Microbiology diagnostic laboratories facilitate optimal therapy through clinical support and guid-

ance. The establishment of dedicated microbiology services (on-site) at Burn Hospitals in Iraq enabled pathogen identification and susceptibility testing along with tailoring empirical therapy based on culture results. Modern diagnostic testing has been improved with the VITEK 2 Compact automated instrument resulting in more accurate results, as well as reduced turnaround times for specimens submitted for testing, compared to traditional methods [71].

Surveillance culture tests taken on admission and at predetermined intervals throughout a hospital stay give an understanding of the colonizing organisms and their changing resistance profiles during that same time period.The predictive ability of surveillance culture for invasive infections is under review, as its use may assist healthcare personnel in making decisions regarding empirical treatment of infection in burn patients. Blood culture is the primary approach for diagnosing bacteremia and sepsis in burn patients and all positive cultures should require immediate antibiotic modifications based on the susceptibility information from the laboratory [72].

Burn units can utilize cumulative drug resistance data based on WHONET software to track the historical patterns of drug resistance for the purpose of implementing or updating the empirical guidelines for antibiotic therapy based on this information. Continuous monitoring of resistance will enhance the success of antimicrobials stewardship programs. In addition, each burn unit or service must communicate the antibiogram summarizing the local (unit/service) patterns of drug resistance to all healthcare providers involved with the care of burned patients [73]. Preventing and controlling infection in burn units relies on a comprehensive infection prevention and control program. Healthcare providers must comply with the hand hygiene requirements, including performing hand hygiene before and after every transaction with patients, after touching contaminated surfaces and after removing gloves. Hand hygiene supplies (alcohol-based hand antiseptics) must be located at every point of care; handwashing with soap and water is recommended for all personnel after handling visibly soiled hands or for providing care to patients with spore-forming bacteria [74].

Given high MDR/XDR rates in Iraqi burn units, alternative therapies are urgently needed. Bacteriophage-loaded functional nanofibers offer localized, sustained release and effective wound healing. Phage-loaded electro spun mats maintain activity against *P. aeruginosa* and *S. aureus* for up to four weeks, targeting biofilm infections. Other strategies include antimicrobial peptides, nanoparticle-based systems and photodynamic therapy. Key advantages: reduced systemic toxicity, lower resistance risk and enhanced biofilm penetration. Nanofiber dressings represent a paradigm shift from systemic to targeted topical treatment for burn injuries. In a murine burn wound model infected with *K. pneumoniae*, bacteriophage therapy demonstrated significant efficacy. A single intraperitoneal dose of phage Kpn5 reduced mortality, decreased bacterial counts in organs within 72 hours and led to complete skin recovery on histopathology. Even when treatment was delayed until 18 hours post-infection, approximately 27% of mice survived, highlighting the therapeutic potential of phages against K. pneumoniae burn infections [75]. When clinicians provide wound care, personal protective equipment (gloves, gowns, masks, etc.) should be worn in accordance with good practice standards. Patients who are colonized or infected with multidrug-resistant organisms should be managed using contact precautions, ideally in private patient rooms. If patients are housed together in the same room, it is suggested that they house patients who carry the same organism to further prevent cross-contamination. Cleaning and disinfecting high-touch surfaces should be routinely performed using appropriate disinfectants known to be effective against the most common pathogens. If performed regularly, the successful implementation of these fundamental infection control procedures will significantly minimize the risk of cross-transmission, even in resource-poor settings [76]. The comparative data in Table 1 reveal substantial inter-provincial heterogeneity. Sulaymaniyah (north) showed exceptionally high MRSA and carbapenem resistance rates, while Misan (south) had a different pathogen hierarchy (*P. aeruginosa* predominating). Baghdad (center) exhibited unique patterns of colistin resistance among Enterobacteriaceae. These differences may reflect regional variation in antibiotic prescribing practices, infection control resources, or local outbreak strains. However, the many empty cells in the table also demonstrate a critical limitation: the absence of standardized, multi-province surveillance. Without concurrent data from the same pathogen and same antibiotic class across all provinces, true comparative analysis remains incomplete.

Table 1. Comparison of Antimicrobial Resistance Patterns Across Three Iraqi Provinces (Sulaymaniyah, Baghdad and Misan)

Bacterial Pathogen	Resistance Pattern	Sulaymaniyah	Baghdad	Misan
S. aureus	MRSA (% of S. aureus)	87.1% (176/202)	86.6% (resistant to amikacin)	Not reported
P. aeruginosa	Imipenem resistance	79.2%	Not specified separately	Data not available
	Ciprofloxacin resistance	93.7%	Not specified	Data not available
	Colistin resistance	Up to 60%	Not specified	Data not available
	MDR prevalence	28.8% (of non-fermenters)	Not specified	Data not available
A. baumannii	Colistin resistance	Not reported	High (colistin resistance developing during therapy)	Not reported
	Imipenem resistance (2003-2005 trend)	Not reported	Decreased from 87% to 56% over 2 years	Not reported
Enterobacteriaceae	ESBL-producing	Present in high proportion	Not specified	Present
	Colistin resistance (K. pneumoniae)	Not reported	100%	Not reported
	Colistin resistance (*E. coli*)	Not reported	100%	Not reported
Overall MDR among all isolates	MDR prevalence	51% (715/1402 isolates)	Not specified	Not reported
Most common pathogen (rank)	1st	*S. aureus* (34%)	*S. aureus* (31.3%)	*P. aeruginosa* (20%)
	2nd	*P. aeruginosa*	*P. aeruginosa* (16%)	*S. aureus* (17.14%)
	3rd	*A. baumannii* (20%)	*Enterobacter* spp.	*Enterobacter* spp. (16.19%)

Note: Empty cells indicate that the original studies did not report data for that specific resistance pattern in that province. MDR = multidrug-resistant; MRSA = methicillin-resistant *S. aureus*; ESBL = extended-spectrum beta-lactamase.

CONCLUSION

In Iraq, the challenges associated with burn and wound infections are highly complex and multifactorial. A number of clinical and Public Health challenges must be dealt with including a high incidence of multidrug-resistant pathogens. High mortality rates associated with these infections as well as numerous underlying issues contribute to the overall burden on the Iraqi health care system. The predominant pathogens associated with the majority of burn/wound infections are *P. aeruginosa*, *S. aureus* and *A. baumannii*. The widespread nature of multi-drug resistance of these organisms presents a significant challenge to effectively managing the majority of patients with these types of infections, often leading to therapeutic failure. Poor overall infection control measures, high levels of overcrowding and an environment that is contaminated or free from contamination (or both) are all factors that contribute to the continued transmission and development of multiresistance. Many of the microorganisms associated with burn injuries utilize complex virulence mechanisms to avoid detection by the host defense mechanism(s), to evade antibacterial treatment and to establish chronic infection in the presence of host protective mechanisms. The implementation of an integrated approach focused on robust infection prevention and control, development of Antimicrobial Stewardship Programmes and development of robust, evidence-based treatment protocols, in conjunction with promotion of burn injury prevention, will ultimately result in improved outcomes for patients suffering from burn and other wound infections. Continuing Regional Partnership Development and continued International

Technical Assistance are essential for effectively addressing ongoing public health issues associated with these infections. The pursuit of further research in the area of treatment modalities aimed specifically at preventing biofilm formation and developing new Anti-Inflammatory Agents or alternatives to existing Antimicrobials will continue to provide new opportunities for future effective management of these infections.

SUPPLEMENTARY MATERIAL

None.

AUTHOR CONTRIBUTIONS

Zainab Agab Altaee: Data Collection, Writing – original draft. Nabaa Qais Jameel: Investigation, Writing – review & editing. Raad N. Hasan: Methodology, Supervision.

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CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

DECLARATION OF GENERATIVE AI USE

During the preparation of this manuscript, the authors used Grammarly for grammar checking and language polishing. After using this tool, the authors thoroughly reviewed and revised the generated content, taking full responsibility for the accuracy and integrity of the final manuscript.

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