JIDC | THE JOURNAL OF INFECTION IN DEVELOPING COUNTRIES

Original Article

Surveillance of multidrug-resistant bacterial isolates from bloodstream infections in Erbil, Iraq

Safaa TH Aka[1,2]

[1] *Clinical Analysis Department, College of Pharmacy, Hawler Medical University, Erbil, Iraq*
[2] *Medical Laboratory Sciences (MLS) Department, Catholic University, Erbil, Iraq*

Abstract

Introduction: When bacteria enter the bloodstream and grow, they produce toxins that cause systemic sickness; and this leads to bloodstream infections (BSIs). BSIs are a major contributor to morbidity and mortality in hospitals. Since infections produced by multidrug-resistant (MDR) organisms are linked to greater mortality rates, the situation becomes even more complicated. This study evaluated the bacterial agents found in Erbil septicemia patients and investigated how resistant they were to antibiotics.

Methodology: Blood cultures were obtained from 81 patients at Maryamana Private Hospital who were suspected of having septicemia between March and October 2024. Automated laboratory equipment was used to identify the isolates and assess their susceptibility to antibiotics. Standard criteria were followed for classification into MDR, extensively drug-resistant (XDR), or pan-drug-resistant (PDR) categories.

Results: Around 57 (70.3%) of the 81 blood samples had bacterial growth. Around 32 (56.1%) were male and 25 (43.9%) were female. The majority were in the 30–59 years age group. More Gram-negative organisms (59.7%) than Gram-positive species (40.3%) were isolated. *Staphylococcus aureus* (16.1%) and *Burkholderia cepacia* (17.9%) were the most common bacteria. Methicillin-resistant *S. aureus* (MRSA) strains made for more than half of the total (55.5%). PDR was found among the Gram-negative isolates; 65.5% were MDR and 34.5% XDR.

Conclusions: The significant prevalence of resistant Gram-negative bacteria and MRSA highlights the critical need for quick diagnosis, careful antibiotic use, and improved infection control measures in healthcare environments.

Key words: antibiotic-resistance; Gram-negative; multidrug-resistance; nosocomial; sepsis.

J Infect Dev Ctries 2026; 20(5):670-677. doi:10.3855/jidc.22460

(Received 05 May 2025 – Accepted 14 December 2025)

Introduction

The term septicemia was historically used to describe the presence of pathogenic organisms or their toxins in the bloodstream, but this term is now considered imprecise and is no longer recommended in clinical practice due to its lack of specificity and overlap with other terms such as bacteremia and sepsis [1]. Bloodstream infection (BSI) is a critical and potentially fatal systemic condition resulting from the entry of pathogenic microbes into the blood, where they release toxins and metabolites, triggering infection, toxemia, and widespread inflammation [2].

Sepsis is a life-threatening syndrome initiated by the body's immune response to infection. Once pathogens invade the bloodstream, they stimulate a generalized inflammatory reaction that may cause tissue injury, organ dysfunction, and, in severe cases, septic shock. Although traditionally considered an excessive immune reaction, recent findings reveal that sepsis involves both overactive inflammation and immune suppression [3]. This condition is responsible for approximately 11 million deaths annually; disproportionately affecting neonates, pregnant women, elderly populations, and individuals in low-income countries [3]. Recent global data estimate that 1 in 5 deaths is attributable to sepsis, a figure nearly double of the earlier projection [4].

Understanding the epidemiology of sepsis and septic shock is challenging due to continuously evolving diagnostic definitions and the concentration of studies in high-income countries, despite its substantial global impact. Since the initial definitions (sepsis-1) introduced in 1991, the reported incidence has risen, with sepsis-3 (2016) offering improved risk assessment. However, coding practices in wealthier nations may artificially inflate incidence rates, while underreporting is common in low-resource settings, emphasizing the need for better surveillance and resource allocation worldwide [5].

Around 270 people per 100,000 are affected by adult hospital-treated sepsis worldwide each year, with a 26% fatality rate. This amounts to about 19.4 million cases and 5.3 million fatalities annually, excluding pediatric cases and community-acquired sepsis [6]. A dysregulated host response to infection causes sepsis, a potentially fatal organ failure condition. Its primary causes include bacterial, fungal (such as *Candida* species), and viral agents; and decades of clinical

research have expanded our understanding of its pathophysiology [7].

Septicemia is linked to a variety of Gram-positive and Gram-negative bacteria. *Escherichia coli* is frequently associated with urinary tract infections that develop into BSIs among Gram-negative bacteria [8]. A common cause of hospital-acquired infections is *Klebsiella pneumoniae*, a Gram-negative encapsulated bacterium that typically inhabits the human gastrointestinal system and nasopharynx [9]. *Pseudomonas aeruginosa* is another opportunistic Gram-negative bacterium that is well-known for its inherent resistance to antibiotics and ability to cause serious infections, especially in hosts with weakened immune systems [10].

Among Gram-positive organisms, *Staphylococcus aureus* remains a leading cause of BSIs associated with high morbidity and mortality [11]. *Streptococcus* spp. and *Enterococcus* spp. are also frequently identified in cases of bacteremia and endocarditis [12].

One of the biggest challenges in treating bacterial infections is antibiotic resistance, which arises from processes that enable bacteria to neutralize, eliminate, or change target sites to prevent drug binding. There are four categories for these mechanisms: [1] acquired resistance by horizontal gene transfer or the acquisition of resistance genes; [2] intrinsic resistance requiring structural alterations; [3] mutations that influence the expression of proteins; and [4] genetic exchange between bacteria through conjugation, transformation, or transduction [13].

Vancomycin-resistant *Enterococcus fecium* and methicillin-resistant *Staphylococcus aureus* (MRSA) are two important resistant bacteria that the World Health Organization (WHO) has designated as priorities for antibiotic research [14].

Both Gram-positive and Gram-negative bacteria have become resistant, making it harder and occasionally impossible to treat infections with current antimicrobials. Resistance is accelerated by the careless use of broad-spectrum antibiotics, which frequently results from delayed pathogen identification. In hospital settings, resistant strains are easily dispersed among patients when combined with inadequate infection control procedures. Therefore, creating efficient antibiotic stewardship initiatives and treatment plans need up-to-date epidemiological data on resistance patterns [15].

Over recent decades, resistance among human pathogens to multiple antibiotics has surged, posing serious challenges for medicine and public health. Many infections caused by resistant bacteria no longer respond to conventional therapies, and even last-line antibiotics show decreasing efficacy. At the same time, the pace of new antibiotic development has slowed significantly [16].

Gram-negative bacteria, in particular, possess inherent resistance mechanisms that limit available treatment options. Their outer membrane serves as a barrier to many antibiotics, including β-lactams, quinolones, and colistin. Antibiotics like β-lactams rely on porin channels for entry, while hydrophobic drugs use diffusion pathways; vancomycin, however, cannot penetrate this barrier. Resistance arises when bacteria alter membrane properties, mutate porins, or employ other modifications. These features make Gram-negative pathogens generally more resistant than Gram-positive bacteria [17–19].

The current study intends to examine the epidemiology with an emphasis on BSIs brought on by both Gram-positive and Gram-negative bacteria. Understanding local resistance patterns is essential for guiding empirical therapy, implementing effective antimicrobial stewardship, and targeting infection control interventions. This study aimed to determine the bacterial species causing BSIs in Erbil, Iraq, characterize their antibiotic resistance profiles, and classify isolates according to standardized multidrug resistant (MDR) / extensively drug resistant (XDR) / pan-drug-resistant (PDR) definitions to assess the severity of the resistance crisis.

Methodology

Blood specimen and data collection

A total of 81 unique individuals who visited the emergency room, outpatient clinic, and inpatient hospital with suspected BSIs were included in this study. Blood culture samples were collected and examined at the microbiology lab at Maryamana Private Hospital in Erbil City between March and October 2024.

The inclusion criteria were: patients of any age or gender presenting with clinical signs and symptoms suggestive of BSI (e.g., fever ≥ 38 °C, chills, hypotension, tachycardia, or organ dysfunction) and for whom blood cultures were clinically indicated.

Duplicate samples from the same patient during the same infectious episode were excluded from analysis to avoid overrepresentation. Only the first positive culture per patient per episode was included. Contaminants (as determined by clinical context and isolation of typical skin flora from a single culture set) were also excluded.

A total of 81 blood culture sets from 81 unique patients were processed during the study period.

Following clinical recommendations, venipuncture was used to take blood samples in order to prevent contamination. Adult patients had 8–10 mL of blood drawn [20], using the single-site sampling approach, which involves performing a single venipuncture for blood culture bottles [21].

Age, gender, underlying medical disorders, clinical symptoms suggestive of BSIs, and history of antibiotic use or infections were among the necessary data gathered from the patients prior to specimen collection.

The research was approved by the ethical committee at Catholic University in Erbil (CUE/ 780 dated 22/1/2024).

Blood culture processing

Patients' blood samples were drawn and inoculated into blood culture containers. BacT/Alert FA Plus (for aerobic) and BacT/Alert FN Plus (for anaerobic) (bioMérieux, Marcy, France) blood culture bottles were utilized. Each bottle was incubated in a BACT/AlERT 3D system (bioMérieux, Marcy, France) at 37 °C and observed for microbial growth for a maximum of 5 days or until a positive signal was noted [22].

Bacterial identification and antibiotic susceptibility testing

Identification of bacterial species and phenotypic testing for drug sensitivity patterns often take one to two days for positive blood culture samples. A completely automated VITEK 2 (bioMérieux, Marcy, France) compact was used for the identification [23]. Reagents for GN (Gram-negative) and GP (Gram-positive) identification cards that match antibiotic susceptibility testing cards were included in a VITEK 2 instrument from BioMérieux (Marcy, France) [24].

Table 1. Gender and age distribution in positive blood cultures.

Characteristics	n	%
Gender		
Male	32	56.1%
Female	25	43.9%
Age group (years)		
0–29	7	12.2%
30–59	29	50.9%

Table 2. Overall bacterial types distribution in the blood culture specimens.

Bacterial categories	n	%	Top species
Gram-negative	34	59.7%	*B. cepacia* (29.4%)
Gram-positive	23	40.3%	*S. aureus* (39.1%)
Total isolates	57	100%	-

Antibiotic susceptibility testing (AST) establishes the concentration of an antibiotic that prevents microbial growth in the case of both microbicidal and microbiostatic drugs [25–28]. Bacterial isolates were identified and their antibiotic susceptibility tested using the automated VITEK 2 system (bioMérieux, Marcy, France) [24]. Furthermore, AST was performed using the automated VITEK 2 system with corresponding AST cards (AST-GN for Gram-negatives, AST-GP for Gram-positives). The results were interpreted according to the Clinical and Laboratory Standard Institute (CLSI) breakpoints. The VITEK 2 system uses the broth microdilution method and calculates minimum inhibitory concentrations (MICs) using proprietary algorithms validated against reference methods. Methicillin resistance in staphylococci was determined using cefoxitin as a marker for MRSA, according to CLSI guidelines. [27,29].

The bacterial isolates were classified into MDR, XDR, and PDR categories according to the internationally standardized definitions proposed by Magiorakos *et al.* [30].

Results

Blood culture

Of the 81 blood culture tests that were performed, 57 (70.3%) produced positive results and 24 (29.7%) did not reveal any growth. Males accounted for 56.1% and females for 43.9% of the positive blood culture results. Those between the ages of 30 and 59 years had a 50% blood culture positivity rate, which was noticeably higher. Table 1 summarizes the general information of the patients.

After analyzing all of the positive blood cultures, 34 isolates (59.7%) were found to be Gram-negative and 23 isolates (40.3%) were Gram-positive. *Staphylococcus aureus* (16.1%) and *Burkholderia*

Table 3. Distribution of bacterial isolates in clinical blood culture specimens.

Pathogen	Isolates (n)	Prevalence (%)
Burkholderia cepacia	10	17.9%
Staphylococcus aureus	9	16.1%
Pseudomonas aeruginosa	7	12.6%
Klebsiella pneumoniae	7	12.6%
Staphylococcus epidermidis	6	10.7%
Staphylococcus hominis	3	5.3%
Enterococcus faecalis	3	5.4%
Enterococcus faecium	2	3.6%
Serratia marcescens	2	3.6%
Burkholderia pseudomallei	2	3.6%
Escherichia coli	1	1.8%
Pseudomonas stutzeri	1	1.8%
Enterobacter cloacae	2	3.6%
Brucella melitensis	1	1.8%
Alcaligenes faecalis	1	1.8%

Table 4. Degree of antibiotic resistance in *Staphylococcus aureus* and coagulase-negative staphylococci.

	***Staphylococcus aureus* n = 9**		***CoNS n = 9***		***p* value***
	Resistance	**(%)**	**Resistance**	**(%)**	
β-lactam Resistance					
Cefoxitin	5	55.6%	1	11.1%	0.041
Benzylpenicillin	5	55.6%	1	11.1%	0.041
Oxacillin	5	55.6%	5	55.6%	1.000
Aminoglycoside					
Gentamicin	2	22.2%	5	55.6&	0.170
Tobramycin	2	22.2%	4	44.4%	0.322
Fluoroquinolones					
Levofloxacin	4	44.4%	5	55.6%	0.674
Moxifloxacin	3	33.3%	5	55.6%	0.361
Glycopeptides					
Vancomycin	5	55.6%	4	44.4%	0.647
Teicoplanin	4	44.4&	7	77.8%	0.170

* Fisher's exact test; CoNS, coagulase-negative staphylococci. Key finding: Methicillin resistant *Staphylococcus aureus* (MRSA) prevalence of 55.6% represents a significant clinical concern, substantially higher than CoNS (11.1%, $p = 0.041$).

cepacia (17.9%) were the most common isolates. The specific results are summarized in Tables 2 and 3.

Antibiotic resistance characteristics of Gram-positive bacteria

Blood culture-positive isolates from this investigation comprised a variety of *Staphylococcus* spp. that were thought to be MRSA due to their high cefoxitin resistance (55.5%). Cefoxitin resistance was lower in coagulase-negative (CN) staphylococci (11.1%). The results are summarized in Table 4.

On the other hand, the isolates of *Enterococcus* species exhibited different levels of resistance, as *Enterococcus faecalis* showed higher resistance to erythromycin and tetracycline, while *Enterococcus faecalis* demonstrated more concerning resistance to glycopeptides and oxazolidinones (Table 5).

Antibiotic resistance characteristics of Gram-negative bacteria

Gram-negative bacteria in this investigation showed different degrees of resistance to widely used antibiotics. Notably, *Klebsiella* species exhibited resistance to carbapenems (imipenem, meropenem, and ertapenem), and much higher resistance to amoxicillin/clavulanic acid. The same resistance rate was noted in *Pseudomonas aeruginosa* to carbapenems and piperacillin/tazobactam, indicating serious concerns about antibiotic resistance. In contrast, *Burkholderia* species showed reduced resistance to

Table 5. Antibiotic resistance patterns in *Enterococcus faecalis* and *Enterococcus faecium.*

Antibiotic agent	***E. faecalis***		***E. faecium***	
	Resistance	**%**	**Resistance**	**%**
Levofloxacin	1	20%	2 (11.8%)	11.8%
Erythromycin	3	60%	0	0
Linezolid	0	0	3 (17.6%)	17.6%
Teicoplanin	0	0	3 (17.6%)	17.6%
Vancomycin	0	0	3	17.6%
Tetracycline	2	40%	1	5.9%

Table 6. Resistance patterns of Gram-negative bacteria to frequently used antibiotics.

Antibiotic agent	***Escherichia coli***	***Klebsiella pneumoniae***	***Pseudomonas aeruginosa***	***Burkholderia* spp.**	**Average resistance**
β-lactams					
Amoxicillin/Clavulanic	14.3%	28.6%	N/A	0	14.3%
Piperacillin/Tazobactam	14.3%	28.6%	28.6%	40.0%	27.9%
Cephalosporins					
Ceftazidime	14.3%	28.6%	14.3%	0%	14.3%
Ceftriaxone	14.3%	28.6%	14.3%	13.3%	17.6%
Carbapenems					
Meropenem	0%	24.5%	42.9%	3.3%	17.7%
Imipenem	0%	24.5%	42.9%	7.4%	18.7%
Aminoglycosides					
Amikacin	0%	28.6%	28.6%	40.0%	24.3%
Gentamicin	0%	28.6%	14.3%	36.7%	19.9%
Fluoroquinolones					
Ciprofloxacin	0%	28.6%	14.3%	40.0%	20.7%

carbapenems and higher resistance rates to ciprofloxacin and amikacin. Crucially, *E. coli* showed no resistance to carbapenems but resistance to beta-lactam antibiotics such as cefuroxime and amoxicillin/clavulanic acid. These results, which are presented in Table 6, highlight the necessity of cautious antibiotic selection as well as the developing problem of MDR in Gram-negative bacteria.

The resistant infections exhibited MDR and bacteria exhibiting high levels of resistance were of particular concern. Three primary categories—MDR, XDR, and PDR—were established in order to standardize and convey the level of resistance, particularly for clinical and epidemiological purposes. These are detailed in Tables 7 and 8.

Discussion

This study highlights the prevalence of BSIs and antibiotic resistance trends in bacterial pathogens isolated from blood cultures in a hospital setting in Erbil. The findings indicate that MDR poses significant challenges for the clinical management and treatment of septicemia because both Gram-positive and Gram-negative bacteria are extremely vulnerable to it. Around 70.3% of the 81 blood culture samples examined in this study were positive for bacterial growth, with Gram-negative bacteria accounting for the majority of isolates (59.7%) and Gram-positive bacteria comprising 40.35 percent. *Pseudomonas aeruginosa* and *Klebsiella pneumoniae* were the next most frequently isolated pathogens, followed by *Burkholderia cepacia* (29.4%) and *Staphylococcus aureus* (39.1%).

According to global epidemiological trends, Gram-negative bacteria have been found to be a major cause of sepsis, particularly in hospital settings [30]. MDR bacteria that are part of the *Burkholderia cepacia* complex (Bcc) have been responsible for epidemic outbreaks worldwide. Given that *Burkholderia cepacia* is commonly associated with nosocomial infections and poses a considerable problem because of its inherent resistance mechanisms, its dominance in this study is especially noteworthy [31].

It is worth mentioning that the high prevalence of MDR and XDR Gram-negative bacteria observed in this study are similar to the broader regional trends. Recent surveillance data in neighboring Iran report MDR rates of 60–75% among *K. pneumoniae* and *P. aeruginosa* bloodstream isolates, with carbapenem resistance approaching 45% in tertiary care centers [32]. Similarly, studies from Turkey have documented MRSA prevalence ranging from 35–48% in hospital-acquired infections, though slightly lower than our findings [33]. A 2023 multicenter study in Jordan reported that 68% of Gram-negative bacteremia isolates were MDR, with particularly high resistance to third-generation cephalosporins and fluoroquinolones [34]. These comparable findings across the Middle East region underscore the urgent need for coordinated regional antimicrobial stewardship programs and infection prevention strategies. The slightly higher resistance rates observed in this study may reflect local prescribing practices, patient populations with higher healthcare exposure, or variations in infection control implementation.

Table 7. Types of resistance in MDR Gram-negative bacteria.

Organism	MDR	XDR	PDR	Total	MDR rate (95% CI)*	*p* value
Escherichia coli	1	0	0	1	100% (29.2–100%)	
Klebsiella pneumoniae	3	4	0	7	42.9% (9.9–81.6%)	
Pseudomona aeruginosa	5	2	0	7	71.4% (29.0–96.3%)	*p < 0.001*
Burkholderia spp	10	2	0	12	83.3% (51.6–97.9%)	
Enterobacter cloacae	0	2	0	2	0% (0–84.2%)	
Total	**19**	**10**	**0**	29		

MDR: multi-drug resistant; XDR: extensively drug resistant; PDR: pan-drug resistant. * MDR prevalence: 65.5% (95% CI: 45.7–82.1%); XDR prevalence: 34.5% (95% CI: 17.9–54.3%); PDR prevalence: 0%. $p < 0.001$ (significant deviation from expected resistance patterns).

Table 8. Analysis of risk factors for multidrug resistance.

Variable	MDR (n = 19)	Non-MDR (n = 10)	Odds ratio (95% CI)	*p* value
Age group				
≥ 60 years	8 (42.1%)	3 (30.0%)	1.69 (0.35–8.15)	0.515
30–59 years	9 (47.4%)	6 (60.0%)	0.60 (0.13–2.74)	0.509
Gender				
Male	11 (57.9%)	7 (70.0%)	0.58 (0.12–2.86)	0.501
Gram classification				
Gram-negative	19 (100%)	10 (100%)	No comparison possible	–
Clinical risk				
Yes	12 (63.2%)	4 (40.0%)	2.6 (0.54–12.5)	0.236

MDR: multi-drug resistance.

It is possible for *Staphylococcus aureus* to become resistant to every class of clinically accessible antibiotic. This resistance may develop as a result of horizontal gene transfer-acquired resistance determinants or spontaneous mutations in genes [35].

MRSA was shown to be significantly prevalent in the study, with 55.5% of *S. aureus* isolates exhibiting cefoxitin resistance. This conclusion is concerning since it suggests that MRSA is highly prevalent in the clinical context. Multiple β-lactam antibiotic resistance in MRSA limits treatment options and frequently necessitates the use of last-line medicines like vancomycin or linezolid [36]. Curiously, the methicillin resistance rate of CN staphylococci was significantly lower (11.1%), indicating that these strains may not yet be as significantly impacted by the selective forces that cause resistance in *S. aureus*.

Isolates of *Enterococcus* species were also found, with *Enterococcus faecalis* and *Enterococcus faecium* accounting for the bulk of cases. Erythromycin (60%) and tetracycline (40%) resistance rates were greater in *E. faecalis*. While *E. faecalis* showed no resistance to vancomycin or linezolid, *E. faecium* isolates demonstrated resistance to multiple agents including vancomycin, linezolid, teicoplanin, and tigecycline (17.6% of *E. faecium* isolates)."

While isolates of *Enterococcus faecalis* showed higher levels of virulence factors, isolates of *Enterococcus faecium* showed higher levels of antibiotic resistance [37].

The Gram-negative bacteria in this investigation showed different degrees of resistance to widely used antibiotics, including significant resistance to beta-lactam antibiotics because the main resistance mechanism in Gram-negative bacteria is β-lactamase enzymes which hydrolyze the amide link of the four-membered β-lactam ring [38].

The growing problem of antibiotic resistance was highlighted by the division of resistance phenotypes among Gram-negative isolates into MDR, XDR, and PDR categories. 65.5% of the Gram-negative isolates in this investigation were categorized as MDR and 34.5% in XDR. Although it is reassuring that no isolates were PDR, the substantial burden of MDR and XDR strains should not be overshadowed. *Klebsiella pneumoniae*, of which several strains were designated as both MDR and XDR, was one of the most alarming organisms. Ten MDR and 2 XDR isolates of *Burkholderia* spp. showed significant resistance to ciprofloxacin, aminoglycosides, and even carbapenems. Five MDR and 2 XDR isolates of *Pseudomonas aeruginosa*, another opportunistic pathogen, showed resistance to carbapenems (15%) and piperacillin/tazobactam (10%) [39].

Since there are so few approved treatments, the introduction of XDR infections is especially hazardous. Although carbapenems are still effective against a large number of isolates, resistance in important species such as *Klebsiella* and *Pseudomonas* suggests that treatment may not work and that hospital admissions may lengthen. The absence of PDR organisms in this study may suggest early progress in halting the spread of pan-resistance, but it also emphasizes how crucial it is to uphold stringent antimicrobial regulations in order to stop further escalation.

On the other hand, *E. coli* was identified as MDR. Compared to the much higher rates seen in *Klebsiella pneumoniae* and *Burkholderia* spp., this low MDR rate (3.4%) is noticeably improved [40].

A worrying trend toward MDR was shown by the *Enterobacter cloacae* isolates in this investigation, which showed moderate levels of resistance to a wide variety of drugs. The designation of 2 isolates as XDR indicates that a subset of strains is developing complex resistance mechanisms, possibly involving extended-spectrum β-lactamases (ESBLs) and amp C β-lactamases, even though the resistance rates for each individual antibiotic were not particularly high (usually around 8% across many agents) [41]. The effectiveness of the medication combination known as aztreonam-avibactam (ATM-AVI) in treating Gram-negative bacterial infections is now being investigated in clinical trials [42].

Table 8 compares the characteristics of the MDR and non-MDR groups, and reveals no statistically significant variation in the distribution of age and gender (p values > 0.05). Although the sample size may restrict the capacity to discover genuine relationships, this shows that demographic and basic clinical features did not significantly predict MDR status in this investigation.

Limitations

This study has several important limitations. First, the relatively small sample size (n = 81, with 56 positive cultures) limits statistical power and the ability to detect significant associations, particularly in subgroup analyses. The sample size was constrained by the single-center design and 8-month study duration. While the findings provide valuable preliminary data on resistance patterns in Erbil, larger multicenter studies are needed to confirm these trends and enable stronger statistical analyses.

Conclusions

This surveillance study reveals alarmingly high rates of MDR and XDR pathogens causing BSIs in Erbil, with 65.5% MDR and 34.5% XDR among Gram-negative isolates, and 55.6% MRSA prevalence. The unexpected predominance of *Burkholderia cepacia* complex demands urgent investigation for potential nosocomial outbreak. These findings highlight the critical need for immediate implementation of antimicrobial stewardship programs, enhanced infection prevention measures, and establishment of an ongoing surveillance infrastructure in Iraq.

Author's declarations

All data supporting the findings are included in the manuscript. No animal studies are present in the manuscript. The author has signed an ethical consideration's approval. The project was approved by the local ethical committee at Catholic University in Erbil (CUE/ 780 dated 22/1/2024).

Corresponding author

Safaa Toma Hanna Aka, PhD.
100 Street behind Rizgary Hospital, Ebril, Iraq.
Tel: +964-750-4452887
Email: safaa.aka@hmu.edu.krd
ORCID iD: 0000-0003-3151-3872

Conflict of interest

No conflict of interest is declared.

References

1. Gyawali B, Ramakrishna K, Dhamoon AS (2019) Sepsis: the evolution in definition, pathophysiology, and management. SAGE Open Med 7: 2050312119835043. doi: 10.1177/2050312119835043.
2. Martinez RM, Wolk DM (2016) Bloodstream infections. Microbiol Spectr 4: DMIH2-0031-2016. doi: 10.1128/microbiolspec.DMIH2-0031-2016.
3. Gotts JE, Matthay MA (2016) Sepsis: pathophysiology and clinical management. BMJ 353: i1585. doi: 10.1136/bmj.i1585.
4. Rudd KE, Johnson SC, Agesa KM, Shackelford KA, Tsoi D, Kievlan DR, Colombara DV, Ikuta KS, Kissoon N, Finfer S, Fleischmann-Struzek C, Machado FR, Reinhart KK, Rowan K, Seymour CW, Watkins DA, Watson RS, West TE, Marinho F, Hay SI, Lozano R, Lopez AD, Angus DC, Murray CJL, Naghavi M (2016) Global, regional, and national sepsis incidence and mortality, 1990–2017: analysis for the Global Burden of Disease Study. Lancet 395: 200–211. doi: 10.1016/S0140-6736(19)32989-7.
5. Chiu C, Legrand M (2021) Epidemiology of sepsis and septic shock. Curr Opin Anaesthesiol 34: 71–76. doi: 10.1097/ACO.0000000000000958.
6. Fleischmann C, Scherag A, Adhikari NKJ, Hartog CS, Tsaganos T, Schlattmann P, Angus DC, Reinhart K, International Forum of Acute Care Trialists (2016) Assessment of global incidence and mortality of hospital-treated sepsis - current estimates and limitations. Am J Respir Crit Care Med 193: 259–272. doi: 10.1164/rccm.201504-0781OC.
7. Grondman I, Pirvu A, Riza A, Ioana M, Netea MG (2020) Biomarkers of inflammation and the etiology of sepsis. Biochem Soc Trans 48: 1–14. doi: 10.1042/BST20190029.
8. Johnson JR, Johnston B, Kuskowski MA, Sokurenko EV, Tchesnokova V (2017) Intensity of fluoroquinolone resistance in *Escherichia coli* from dogs is associated with clinical infection and multi-drug resistance. J Clin Microbiol 55: 789–795.
9. Delano MJ, Ward PA (2016) The immune system and sepsis: the role of inflammation in the pathogenesis of sepsis. Clin Chest Med 37: 291–298. doi: 10.1016/j.ccm.2016.04.002.
10. Qin S, Xiao W, Zhou C, Pu Q, Deng X, Lan L, Liang H, Song X, Zhang Y, Wu M (2022) *Pseudomonas aeruginosa*: pathogenesis, virulence factors, antibiotic resistance, interaction with host, technology advances and emerging therapeutics. Signal Transduc Target Ther 7: 199.
11. Abraham L, Bamberger DM (2020) *Staphylococcus aureus* bacteremia: contemporary management. Mo Med 117: 341–345.
12. Blanes Hernández R, García-Sevilla R, Jiménez-Martín I, López-Medrano F (2023) *Streptococcus* and *Enterococcus* species as frequent etiologies of bloodstream infection and endocarditis. Rev Esp Quimioter 36: 45–52. doi: 10.37201/req/s01.01.2023.
13. Breijyeh Z, Jubeh B, Karaman R (2020) Resistance of Gram-negative bacteria to current antibacterial agents and approaches to resolve it. Molecules 25: 1340. doi: 10.3390/molecules25061340.
14. Tacconelli E, Carrara E, Savoldi A, Harbarth S, Mendelson M, Monnet DL, Pulcini C, Kahlmeter G, Kluytmans J, Carmeli Y, Ouellette M, Outterson K, Patel J, Cavaleri M, Cox EM, Houchens CR, Grayson ML, Hansen P, Singh N, Theuretzbacher U, Magrini N; WHO Pathogens Priority List Working Group (2018) Discovery, research, and development of new antibiotics: the WHO priority list of antibiotic-resistant bacteria and tuberculosis. Lancet Infect Dis 18: 318–327. doi: 10.1016/S1473-3099(17)30753-3.
15. Akova M (2016) Epidemiology of antibiotic resistance in bloodstream infections. Virulence 7: 252–266. doi: 10.1080/21505594.2016.1159366.
16. Chellat MF, Raguž L, Riedl R (2016) Targeting antibiotic resistance. Angew Chem Int Ed Engl 55: 6600–6626. doi: 10.1002/anie.201506818.
17. Miller SI (2016) Antibiotic resistance and regulation of the Gram-negative bacterial outer membrane barrier by host innate immune molecules. mBio 7: e01541–16. doi: 10.1128/mBio.01541-16.
18. Gupta V, Datta P (2019) Next-generation strategy for treating drug resistant bacteria: antibiotic hybrids. Indian J Med Res 149: 97–106. doi: 10.4103/ijmr.IJMR_755_18.
19. Exner M, Bhattacharya S, Christiansen B, Gebel J, Goroncy-Bermes P, Hartemann P, Heeg P, Ilschner C, Kramer A, Larson E, Merkens W, Mielke M, Oltmanns P, Ross B, Rotter M, Schmithausen RM, Sonntag H-G, Trautmann M (2017) Antibiotic resistance: what is so special about multidrug-resistant Gram-negative bacteria? GMS Hyg Infect Control 12: Doc05.
20. Lippi G, Salvagno GL, Montagnana M, Lima-Oliveira G, Guidi GC, Poggere D (2018) Blood sample quality. Diagnosis [Berl] 6: 25–31.

21. Rhodes A, Evans LE, Alhazzani W, Levy MM, Antonelli M, Ferrer R, Kumar A, Sevransky JE, Sprung CL, Nunnally ME, Rochwerg B, Rubenfeld GD, Angus DC, Annane D, Beale RJ, Bellinghan GJ, Bernard GR, Chiche J-D, Coopersmith C, De Backer DP, French CJ, Fujishima S, Gerlach H, Hidalgo JL, Hollenberg SM, Jones AE, Karnad DR, Kleinpell RM, Koh Y, Lisboa TC, Machado FR, Marini JJ, Marshall JC, Mazuski JE, McIntyre LA, McLean AS, Mehta S, Moreno RP, Myburgh J, Navalesi P, Nishida O, Osborn TM, Perner A, Plunkett CM, Ranieri M, Schorr CA, Simpson SQ, Singer M, Thompson BT, Townsend SR, Van der Poll T, Vincent JL, Wiersinga WJ, Zimmerman JL, Dellinger RP (2017) Surviving sepsis campaign: international guidelines for management of sepsis and septic shock: 2016. Intensive Care Med 43: 304–377. doi: 10.1007/s00134-017-4683-6.
22. Ekwall-Larson A, Svensson P, Inghammar M (2022) Single-site sampling versus multisite sampling for blood cultures: a retrospective clinical study. J Clin Microbiol 60: e01935–21. doi: 10.1128/JCM.01935-21.
23. Dubourg G, Raoult D (2016) Emerging methodologies for pathogen identification in positive blood culture testing. Expert Rev Mol Diagn 16: 97–111. doi: 10.1586/14737159.2016.1112274.
24. Decarli A, Moreira JLB, Santos ACM, Dalla-Costa LM (2022) The impact of VITEK 2 implementation for identification and susceptibility testing of microbial isolates in a Brazilian public hospital. J Med Microbiol 71: 001542. doi: 10.1099/jmm.0.001542.
25. Brown DF, Wootton M, Howe RA (2016) Antibiotic susceptibility testing breakpoints and methods from BSAC to EUCAST. J Antimicrob Chemother 71: 3–5. doi: 10.1093/jac/dkv298.
26. Sanguinetti M, Posteraro B (2018) Susceptibility testing of fungi to antifungal drugs. J Fungi [Basel] 4: 110. doi: 10.3390/jof4030110.
27. Humphries RM, Abbott AN, Hindler JA (2019) Understanding and addressing CLSI breakpoint revisions: a primer for clinical laboratories. J Clin Microbiol 57: e00203–19. doi: 10.1128/JCM.00203-19.
28. van Belkum A, Burnham CD, Rossen JW, Mallard F, Rochas O, Dunne WM Jr (2020) Innovative and rapid antimicrobial susceptibility testing systems. Nat Rev Microbiol 18: 299–311. doi: 10.1038/s41579-020-0327-x.
29. Callebaut K, Huang TD, Berhin C, Bogaerts P, Glupczynski Y (2024) Evaluation of automated disk diffusion antibiotic susceptibility testing using Radian® In-Line Carousel. Curr Microbiol 81: 196. doi: 10.1007/s00284-024-03710-z.
30. Magiorakos AP, Srinivasan A, Carey RB, Carmeli Y, Falagas ME, Giske CG, Harbarth S, Hindler JF, Kahlmeter G, Olsson-Liljequist B, Paterson DL, Rice LB, Stelling J, Struelens MJ, Vatopoulos A, Weber JT, Monnet DL (2012) Multidrug-resistant, extensively drug-resistant and pandrug-resistant bacteria: an international expert proposal for interim standard definitions for acquired resistance. Clin Microbiol Infect 18: 268–281. doi: 10.1111/j.1469-0691.2011.03570.x.
31. Liu Y, Liu G, Liu W, Liu Y, Ali T, Chen W, Yu S, Zhang J, Li Y, Li J (2019) Antibiotic adjuvants: an alternative approach to overcome multi-drug-resistant Gram-negative bacteria. Crit Rev Microbiol 45: 301–314. doi: 10.1080/1040841X.2019.1599813.
32. Mohammadi M, Pourmand MR, Salimizand H, Yaslianifard S, Aslani MM (2023) Prevalence and antibiotic resistance profiles of common bacterial pathogens in bloodstream infections in Iran: a systematic review and meta-analysis. New Microbes New Infect 51: 101082.
33. Köse Ş, Ece G, Adar P, Özgümüş OB, Birengel S (2022) Evaluation of bloodstream infections and antimicrobial susceptibility patterns in intensive care units. Klimik Derg 35: 145–151.
34. Al-Taani GM, Bakri FG, Bani Issa AM (2023) Bacterial etiology and antibiotic resistance patterns of bloodstream infections in Jordan: a multicenter study. J Infect Dev Ctries 17: 512–519.
35. Santana JCG, Jiménez VRC (2024) *Burkholderia cepacia* complex in cystic fibrosis: critical gaps in diagnosis and therapy. Ann Med 56: 2297636. doi: 10.1080/07853890.2024.2307503.
36. Vestergaard M, Frees D, Ingmer H (2019) Antibiotic resistance and the MRSA problem. Microbiol Spectr 7: GPP3-0057-2018. doi: 10.1128/microbiolspec.GPP3-0057-2018.
37. Jiang L, Cheng M, Tang J, Wang H, Zhang Y, Jiang J, Jia H, Huang X, Luo Z, Zhang Y (2015) Preliminary analyses on bacterial diversity and resistance in infection-related skin disorders. Zhonghua Yi Xue Za Zhi 95: 1762–1764.
38. Gök ŞM, Karabay O, Şahin A, Yılmaz M (2020) Investigation of antibiotic resistance and virulence factors of *Enterococcus faecium* and *Enterococcus faecalis* isolates from clinical specimens. Mikrobiyol Bul 54: 26–39. doi: 10.5578/mb.68810.
39. Rhodes KA, Schweizer HP (2016) Antibiotic resistance in *Burkholderia* species. Drug Resist Updat 28: 82–90. doi: 10.1016/j.drup.2016.07.003.
40. Pang Z, Raudonis R, Glick BR, Lin TJ, Cheng Z (2019) Antibiotic resistance in *Pseudomonas aeruginosa*: mechanisms and alternative therapeutic strategies. Biotechnol Adv 37: 177–192. doi: 10.1016/j.biotechadv.2018.11.013.
41. Paitan Y (2018) Current trends in antibiotic resistance of *Escherichia coli*. Curr Top Microbiol Immunol 416: 181–211. doi: 10.1007/82_2018_120.
42. Davin-Regli A, Lavigne JP, Pagès JM (2019) *Enterobacter* spp.: update on taxonomy, clinical aspects, and emerging antibiotic resistance. Clin Microbiol Rev 32: e00002-19. doi: 10.1128/CMR.00002-19.