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Original Article

Assessment of the Antimicrobial Resistance Spectrum and Identification of Extended-spectrum ß-lactamase (ESBL) in *Acinetobacter Baumannii* From Clinical Samples in Erbil City

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ABSTRACT

Introduction: *Acinetobacter baumannii* is a dangerous opportunistic pathogen responsible for a wide range of various infections, particularly in healthcare settings, affecting immunocompromised individuals. It is a significant source of nosocomial infections due to its ability to produce extended-spectrum β-lactamases (ESBLs) and carbapenemase enzymes, making it a major concern for antibiotic resistance.

Materials & Methods: To investigate the significant occurrence of ESBL-producing *A. baumannii* in hospitalized individuals, a total of 250 clinical specimens were obtained cultured on blood agar and MacConkey agar media. Identification of isolates was conducted using both conventional microbiological techniques and the automated VITEK 2 system. Duplicate isolates and specimens from colonization-prone sites, including throat and perianal areaswere excluded. Antimicrobial susceptibility and ESBL production were evaluated according to Clinical and Laboratory Standards Institute (CLSI) standards.

Results: Out of 250 clinical specimens, 60(24%) out of 250 were culture-positive for *A. baumannii* infection. Thirty out of sixty isolates (50%) showed an *A. baumannii* infection that produced ESBL. 86.67% of the isolated bacteria exhibited multidrug resistance overall (52/60). Resistance profiling revealed that amikacin had the greatest resistance rate among the 60 isolates (100%), while tigecycline showed the lowest resistance rate among just 10 isolates (16.67%). Notably, colistin demonstrated complete efficacy, with a 0.00% resistance rate, making it the most effective antibiotic against *A. baumannii* in this study. The presence

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of ESBL genes were correlated with antibiotic resistance, particularly with cephalosporin medicines.

Conclusion: In conclusion, the present study highlights the prevalence of ESBL-producing *A. baumannii* strains, emphasizing the need for cautious antibiotic use and systematic monitoring of resistance mechanisms. The emergence of new ESBL strains necessitates continuous surveillance and further research on other *ESBL*-associated genes.

1. Introduction

Acinetobacter baumannii is a gram-negative (G^-), rod-shaped bacterium that typically appears as a small, nearly spherical coccobacillus. It causes blood, urinary tract, and lung infections (pneumonia), as well as sores in other areas of the body. In patients with open wounds or respiratory secretions (sputum), it may also "colonize" or remain there without producing illnesses or symptoms [1].

Recognized as an opportunistic pathogen, *A. baumanni* predominantly impacts individuals with compromised immune responses and is increasingly acknowledged as a significant nosocomial pathogen [2]. Unlike bacteria equipped with flagella, *Acinetobacter* species exhibit alternative motility mechanisms, such as twitching and swarming, likely facilitated by type IV pili—elongated structures capable of extension and retraction [3].

Infections caused by *A. baumannii* are more frequently observed in individuals with prolonged hospital stays, compromised immune systems, advanced age, underlying medical conditions, severe trauma or burns, prior antibiotic usage, or those requiring invasive procedures. Patients with indwelling medical devices, such as catheters or mechanical ventilators, are particularly at risk for such infections [4]. Finding an exact death rate for critically ill patients with *A. baumannii* infections is hard because their prognosis is already not good [5]. However, rough death rates have ranged from 23% to 68%. Currently, the cause of variations in clinical presentation between community-acquired and hospital-acquired infections, whether attributable to host or bacterial variables, remains unclear [6].

Hospitalized patients, especially those with prolonged hospital stays, prior exposure to broad-spectrum antibiotics or anticancer medications, are increasingly experiencing *A. baumannii* colonization. Spreading genes that are not sensitive to antibiotics have become a big problem in treating *A. baumannii* infections, leading to multiple drug resistance (MDR). Previous investigations have consistently demonstrated that *A. baumannii* displays resistance to a broad spectrum of antibiotics, encompassing fluoroquinolones, cephalosporins, carbapenems, tetracyclines, and aminoglycosides. The mechanisms of resistance entail both intrinsic and acquired approaches, including enzymatic inactivation, genetic mutations at the target sites, modifications in outer membrane permeability, and the excessive expression of efflux pumps. Notably, efflux pump systems contribute to MDR by enabling bacteria to expel antibiotics effectively [7]. This hospital-acquired disease is mostly resistant to antibiotics because of β-lactamases, changes to membrane porin channels, and mutations that change how cells work. The main way that bacteria become resistant is by making hydrolytic enzymes, attacking antibiotics, especially extended-spectrum β-lactamases (ESBLs) [8]. The predominant mechanism underpinning the resistance of *A. baumannii* to β-lactam antibiotics and various other antimicrobial agents in recent years has been elucidated as the production of ESBLs. These enzymes are responsible for conferring resistance to a broad range of antibiotics, including monobactams, cephalosporins, and penicillins. Hospitals worldwide are increasingly seeing MDR patterns brought on by pathogenic bacteria's development of ESBLs. Because it results in treatment failures, longer hospital stays, and increased mortality rates, this is a public health issue [9].

More than 300 unique ESBL variants have been delineated among G^- bacterial populations. Globally, the *blaTEM* and *blaSHV* genes have emerged as the predominant genetic determinants associated with ESBL production. In recent years, there has been a notable increase in the prevalence of the *blaCTX-M* gene family among clinical isolates. This gene family encompasses more than 130 β-lactamase variants, which are systematically classified into five distinct categories: *blaCTX-M-1*, *blaCTX-M-2*, *blaCTX-M-8*, *blaCTX-M-9*, and *blaCTX-M-25* [10].

Recent trends across neighboring regions indicate a comparable rise in antibiotic resistance, especially among ESBL-producing bacteria. Researchers in Saudi Arabia have found that *blaCTX-M*, *blaSHV*, and blaTEM genes are commonly found in clinical isolates,indicating a regional pattern of resistance that poses a significant a public health concern [11]. Furthermore, a research conducted in Iran demonstrates a high prevalenceof ESBL-encoding genes in *A. baumannii* isolates [12]. In order to develop evidence-based strategies to address antibiotic resistance in clinical settings, it is essential to characterize the resistance genes encoding ESBL-producing *A. baumannii* [2, 11].

1.1. Objectives

This study aims to investigate the antimicrobial resistance profiles of clinical strains and evaluate the prevalence of *blaSHV*, *blaTEM*, and *blaCTX* genes in *A. baumannii* isolates collected from various hospitals in Erbil city. The study underscores the critical role of ESBLs in antibiotic resistance and their implications for treatment failures.

2. Materials and Methods

2.1. Collection and identification of isolates

Patients were selected based on clinical indicators of infection, including symptoms such as fever, inflammation, and purulent discharge, alongside laboratory findings suggestive of bacterial infection. Samples were collected from patients across different departments, including intensive care units (ICUs), surgical wards, and outpatient clinics to capture a broad spectrum of *A. baumannii* infections. Between March 20, 2024, to June 19, 2024, a total of 250 infected samples were obtained from various clinical specimens: sputum (n=87), wound swab (n=64), stool (n=51), and burn (n=48). All samples were moved right away to the laboratory in an ice-pack-equipped cooler. Clinical samples were cultured on Blood and MacConkey agar (Merck, Germany) and incubated aerobically at 37 ˚C for 24 hours. To isolate pure colonies, non-lactose fermenting colonies on MacConkey agar, as well as non-hemolytic, creamy, and opaque colonies on blood agar, underwent further sub-culturing on MacConkey agar with an additional incubation period of 24 hours under the same conditions. Identification of *A. baumannii* isolates was performed through morphological examination of colonies and Gram staining. Verification of *A. baumannii* was achieved using the VITEK 2 system and standard biochemical tests, including oxidase, catalase, citrate utilization, urease activity, and indole production.

Further molecular confirmation was carried out using polymerase chain reaction (PCR) targeting the *16S rRNA* gene. Amplification was performed using the Alpha PCRmax system (UK) with specific primers (forward: CACCTTCCGATACGGCTACC; reverse: GTTGACTGCCGGTGACAAAC). The PCR mixture consisted of 12.5 µL of master mix (AMPLIQON, Denmark), 1.0 µL of each primer, 1.5 µL of genomic DNA, and PCR-grade water to achieve a final volume of 25 µL. Amplification conditions included 40 cycles of denaturation at 95 ˚C for 30 seconds, annealing at 59 ˚C for 45 seconds, and extension at 72 ˚C for 60 seconds, with a final elongation at 72 ˚C for 10 minutes. The resulting amplicons were analyzed on a 1.2% agarose gel, revealing an expected size of 372 base pairs for the amplified *16S rRNA* gene [13].

2.2. Antimicrobial susceptibility screening

In accordance with the guidelines established by the Clinical and Laboratory Standards Institute (CLSI) [14], antimicrobial susceptibility was assessed using the disk diffusion method. The antibiotic susceptibility tests involved a panel of antibiotics, including amikacin (AK, 30 µg), cefepime (CFP, 30 µg), ceftazidime (CAZ, 30 µg), ciprofloxacin (CIP, 5 µg), colistin (CST, 5 µg), gentamicin (G, 10 µg), imipenem (IMP, 30 µg), levofloxacin (LEV, 5 µg), meropenem (MEM, 10 µg), netilmicin (NET, 30 µg), piperacillin (PIP, 30 µg), tigecycline (TGC, 30 µg), and tobramycin (TOB, 10 µg), all procured from Bioanalyse, Turkey. Using a sterile brush, 100 µL of inoculum (1.5×10^8 CFU/mL) was evenly distributed across the whole surface of a Mueller Hinton Agar plate (Himedia, India), following the 0.5 McFarland standard to create a lawn of *A. baumannii*. Within fifteen minutes of inoculation, the disks were firmly placed on the surface of the agar plate [15].

2.3. Extraction of genomic DNA

Genomic DNA was extracted using the BETA BAYRN Genomic DNA Extraction Kit (BETA BAYRN, Germany), following the manufacturer's guidelines. DNA was eluted with 50 µL of elution buffer and stored at -20 ˚C for subsequent PCR analysis. DNA concentration and purity were assessed using a NanoDrop 1000 spectrophotometer (ThermoFisher Scientific, USA).

Table 1. List of primers used for multiplex PCR amplification

Target Genes	Primer	Sequence (5'-3')	Amplicon Size
blaTEM	Forward	TCG CCG CAT ACA CTA TTC TCA GAA TGA	445-bp
	Reverse	ACG CTC ACC GGC TCC AGA TTT AT	
blasHV	Forward	ATG CGT TATATT CGC CTG TG	747-bp
	Reverse	TGC TTT GTT ATT CGG GCC AA	
blacrx	Forward	ATG TGC AGY ACC AGT AAR GTK ATG GC	593-bp
	Reverse	TGG GTR AAR TAR GTS ACC AGA AYC AGC GG	

2.4. Identification of ESBL-related genes through PCR analysis

Conventional multiplex PCR was employed to examine the molecular profile of genes associated with ESBLs (*blaSHV*, *blaTEM*, and *blaCTX*) in *A. baumannii* strains producing ESBLs. Primer sequences and their respective sizes are revealed in Table 1 [15].

For detecting ESBL genes, PCR reactions were conductedin a 25 µL reaction volume containing 7 µL of PCR-grade water, 10 µL of master mix, 1 µL of primer mix, and 2 µL of genomic DNA. The cycling parameters included an initial denaturation at 95 ˚C for 10 minutes, followed by 40 cycles of denaturation at 95 ˚C for 30 seconds, annealing at 60 ˚C for 90 seconds, and extension at 72 ˚C for 120 seconds, with a final extension step at 72 ˚C for 10 minutes. PCR Amplicons were separated by electrophoresis on a 1.2% agarose gel containing Red Safe dye and visualized under UV light.

2.5. Data analysis

GraphPad Prism software, version 9.3 was employed for statistical analysis. Qualitative data were evaluated using the chi-square test. A $P<0.05$ was considered statistically significant for all analyses.

3. Results

3.1. Detection of *A. baumannii* isolates

The study analyzed 250 clinical samples comprising sputum (n=87), wound swabs (n=64), stool samples (n=51), and burn samples (n=48). Of these, 67 isolates (26.8%) were identified as *A. baumannii* using biochemical tests and the VITEK 2 system with GN cards. The isolates, collected at the Biotechnology Laboratory, Salahaddin University-Erbil, Iraq, were characterized as Gram-negative coccobacilli. They exhibited oxidase-negative, catalase-positive, urease-negative, citrate-negative, and indole-negative profiles. Verification of the 67 isolates through PCR confirmed the presence of the *16S rRNA* gene (Figure 1). Among the isolates, 25(37.31%) were from females, and 42(62.69%) were from males, with a mean age of 51.43±0.8 years.

3.2. Detection of ESBL genes

Among the 67 *A. baumannii* isolates examined, 53(83.58%) contained the *blaSHV* gene, while 34(50.74%) harbored the *blaCTX* gene. The *blaTEM* gene was the most prevalent, detected in all analyzed isolates, as illustrated in Figure 2.

3.3. Antimicrobial resistance

All isolates shown total resistance to AK. CST exhibited the greatest effectiveness against *A. baumannii* isolates, with all isolates showing sensitivity to CST. Sixty-one isolates (91.04%) exhibited resistance to CFP, CIP, IMP, and LEV, marking the greatest resistance rate after that of the AK antibiotic (Figure 3).

4. Discussion

4.1. Detection of ESBL genes

The results revealed a significant proportion of *A. baumannii* isolates producing ESBLs. Studies from Iran have reported alarming levels of drug and multidrug resistance in *A. baumannii*, including resistance to highly effective antibiotics such as IMP and MEM [16]. Ting et al. identified resistance genes—including *blaTEM*, *blaSHV*, *blaCTX*, *blaDHA*, *blaCIT*, *blaIMP*, *blaVIM*, *blaKPC*, and *blaOXA*-23—in seven IMP-resistant *A. baumannii* strains. The isolates exhibited the presence of *blaTEM* (100%) and *blaOXA*-23 (100%) genes. While, the other genes, including *blaSHV*, *blaCTX*, *blaDHA*, *blaCIT*, *blaIMP*, *blaVIM*, and *blaKPC*, were undetectable in seven strains of IMP-resistant *A. baumannii*.

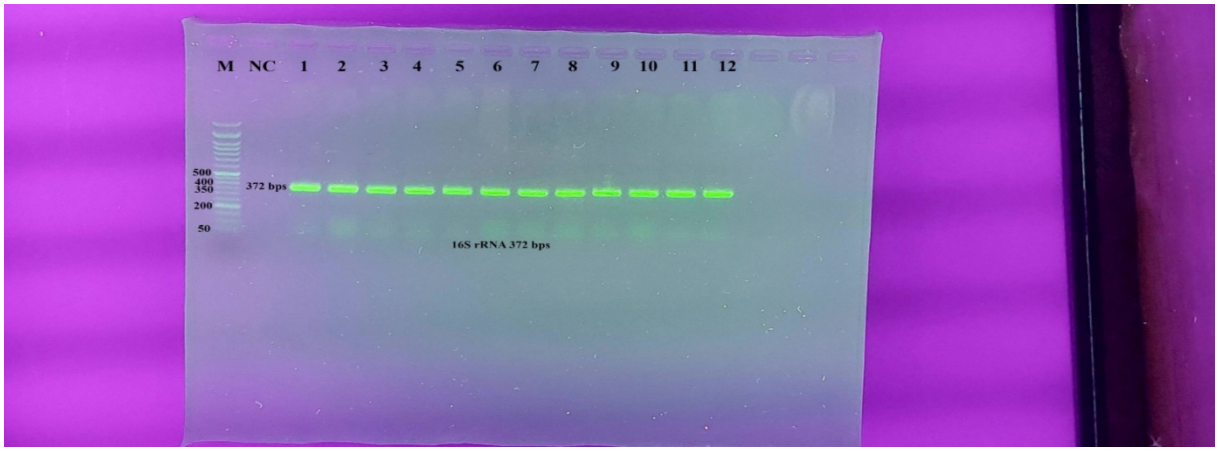

Figure 1. *16S rRNA* gene amplification via agarose gel electrophoresis

Note: Lanes 1–12 show a positive amplicon for the *16S rRNA* gene at 372 bps, lane M is a 50 bps DNA ladder, and lane NC is a negative control.

In the current investigation, consistent with previous findings [17], many resistence genes were identified, including *blaSHV* (58%), *blaTEM* (20%), and *blaVIM* (30%). Shahcheraghi et al. conducted a research in Tehran, Iran, demonstrating that the MBL expressing genes identified among 203 *A. baumannii* isolates were *blaVIM-2*, *blaSPM-1*, *blaIMP-2*, *blaGES-1*, *blaOXA-51*, and *blaOXA-23*. Six isolates were discovered to generate MBLs, while 94 produced OXA-type carbapenemases. Their research suggests that the prevalence of MBL-producing *A. baumannii* strains in Tehran is lower than what was observed in the present study from Hamadan City. They detected *blaSPM-1*, *blaGES-1*, *blaOXA-51*, and *blaOXA-23* genes in 6, 2, 94, and 84 bacterial isolates, respectively [18].

A previous investigation by Rezaee et al. identified genes encoding for 76 Acinetobacter species, including *blaIMP*, *blaSPM-1*, *blaVIM*, *blaPER-1*, *blaVEB-1*, *blaTEM*, *blaSHV*, *blaGES*-1, and *blaCTX-M*. Moreover, they noted that 13.15% of their analyzed isolates carried the *blaTEM-1* gene, which is similar to the 20% found in the current study, and 37% of isolates harbored at least one of the *blaPER-1* or *blaTEM-1* genes. Furthermore, none of the *A. baumannii* isolates they examined contained *blaVEB-1*, *blaSHV, blaCTX-M2*, or *blaGES-1* [19].

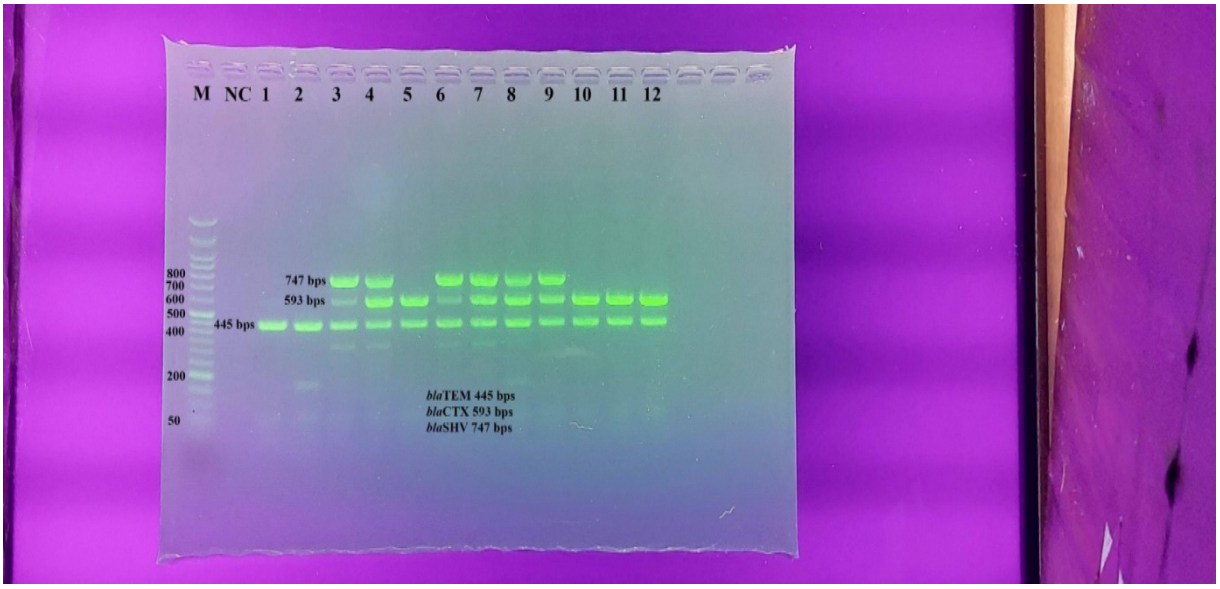

Figure 2. Multiplex PCR of ESBL genes (*blaCTX*, *blaTEM*, and *blaSHV*) using agarose gel electrophoresis

Note: Lane M: 100 bps DNA ladder, lane NC: Negative control, lanes 1–12 with 445 bps *blaTEM* gene, lanes 3–12 with 593 bps *blaCTX* gene, and lanes 3, 4, 6, 7, 8, and 9 with 747 bps *blaSHV* gene.

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Figure 3. The heat map elucidates the levels of sensitivity (resistant, intermediate, and sensitive) of *A. baumannii* to several tested antimicrobial drugs

A. baumannii is a bacterial pathogen known for causing nosocomial infections, primarily due to its high level of drug resistance [20, 21]. Its ability to adhere to a variety of surfaces and medical devices significantly enhances its potential for colonization and transmission among hospitalized individuals [22]. This bacterium can withstand a wide range of existing drugs by acquiring resistance factors and enhancing innate resistance mechanisms [23]. *A. baumannii* exhibiting multidrug resistance causes severe infections and high fatality rates, especially in immunocompromised individuals [24, 25]. According to our investigation, every isolate of *A. baumannii* showed resistance to a number of antibiotics. All isolates were sensitivity to CST and resistant to AK, aligning with results from previous Iranian investigations [26, 27]. Zarifi et al. found that *A. baumannii* had significant resistance to all antibiotics except CST. The resistance rates for IMP, MEM, CAZ, cefotaxime, cefuroxime, ceftriaxone, CFP, ertapenem, and ampicillin/sulbactam were 97.9%, 98.1%, 96.4%, 97.9%, 99.3%, 97.9%, 97.9%, 98.6%, and 97.1%, respectively. CST showed the highest efficacy as an antibiotic against *A. baumannii*, with a susceptibility rate of 97.9%, while AK exhibited a sensitivity of 27.1% [28].

Antibiotic resistance to monobactams, carbapenems, cephalosporins, and penicillin is mediatedby class A β-lactamases. These lactamases may have a restricted spectrum of activity or acquire an expanded range of antibiotic efficacy via point mutations. β-lactamase enzymes, particularly class A, contribute to resistance against antibiotics such as monobactams, cephalosporins, carbapenems, and penicillins. These enzymes are often

inhibited by agents like clavulanic acid [1]. Additionally, the dissemination of ESBL genes among Gram-negative bacteria is facilitated by mobile genetic elements, such as plasmids [2]. Regular monitoring of bacterial strains that produce ESBLs, coupled with the detection of associated genes such as *blaTEM-92*, *blaSHV*, *blaGES-11*, *blaGES-14*, *blaPER-1*, *blaPER-7*, and *blaVEB-1*, is vital for effective clinical management. Additionally, other significant enzymes in this group include the cefotaxime-expanding β-lactamase (CTX-M) family and the *Klebsiella pneumoniae* carbapenemase (KPC) enzymes [3].

Therefore, resistance to third-generation cephalosporins is closely linked to the production of genotypic ESBLs. The epidemiological variety of ESBL-encoding genes in *A. baumannii* may indicate the continual emergence of novel ESBL strains. Upcoming research highlights the investigation of other ESBL-encoded genes. This research emphasized the need for more prudence in antibiotic use and the concerning rate of resistance.

As mentioned in the book, *A. baumannii* can acquire antibiotic resistance by altering the specific location where antibiotics are targeted, controlling the movement of medications across its cell membranes, and enzymatically changing antibiotics to render them ineffective. *A. baumannii* may augment antibiotic resistance not only via innate genetic pathways but also via other virulence-associated mechanisms. Mechanisms contributing to the resistance of *A. baumannii* include structural and functional adaptations, such as alterations in outer membrane proteins (e.g. porins) and components of the cell envelope (e.g. lipopolysaccharides and bacterial capsules). The development of resistance is further enhanced by various specialized mechanisms, including the action of enzymes such as phospholipases C and D, glycan-specific adamalysin-like protease CpaA, as well as processes like quorum sensing and biofilm production.

5. Conclusion

In conclusion, this study underscores the significant prevalence of ESBL-producing *A. baumannii* in clinical samples from Erbil City, contributing to high resistance rates against critical antibiotics. The presence of *blaSHV*, *blaTEM*, and *blaCTX* genes highlights the genetic basis for this resistance, with the *blaTEM* gene being most prevalent. The findings call for careful antibiotic stewardship and enhanced surveillance to track resistance patterns and emerging ESBL strains. Future research should explore additional ESBL-associated genes to develop comprehensive strategies for managing and mitigating antibiotic resistance in *A. baumannii*.

One limitation of this study was the lack of control strains in the antimicrobial susceptibility testing. Additionally, the findings are specific to clinical settings in Erbil City, which may limit their generalizability to other regions with different antimicrobial resistance patterns. Future research should aim to incorporate a broader range of control strains and expand the geographical scope to enhance the validity and applicability of the results. Additionally, given the increasing challenge posed by antibiotic-resistant bacteria, research into alternative treatment strategies, such as bacteriophage therapy, may offer promising solutions

Ethical Considerations

Compliance with ethical guidelines

Ethical considerations

The research received ethical clearance from the Ethics Committee of International University of Erbil, Erbil, Iraq (Code: IUE-8). The study adhered to the ethical guidelines outlined by the committee, ensuring that all patient data were anonymized and handled with confidentiality. Informed consent was obtained from all participants or their legal guardians prior to sample collection. The current study was conducted in compliance with the Declaration of Helsinki and other local rules, emphasizing the reduction of possible hazards to participants while enhancing the scientific value of the research.

Data availability

The authors confirm that the data supporting the findings of this study are available within the article.

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Authors' contributions

Conceptualization and study design: Sayran Sabr Qadr and Pishtiwan Ahmed Hamad; Data acquisition: Sayran Sabr Qadr, Sozan Hamashrif Aziz, and Rebwar Muhammad Hamasalih; Writing the original draft: Mohsin Ali Ahmed and Pishtiwan Ahmed Hamad; Review and editing: Sayran Sabr Qadr, Mohsin Ali Ahmed, and Pishtiwan Ahmed Hamad.

Conflict of interest

The authors declared no conflict of interest.

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