MSI Journal of
Medicine and Medical Research (MSIJMMR)

Phenotypic And Molecular Characterization of Acinetobacter Species Isolated from Bloodstream Infections in Intensive Care Unit Patients

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The authors declare that no funding was received for this work.

Received: 11-October-2025
Accepted: 22-November-2025
Published: 26-November-2025

This article is published in the **MSI Journal of Medicine and Medical Research (MSIJMMR)**
ISSN 3049-1401 (Online)

The journal is managed and published by MSI Publishers.

Volume: 2, Issue: 11 (November-2025)

ABSTRACT: Background: In the demanding environment of intensive care units (ICUs), bloodstream infections (BSIs) attributed to Acinetobacter species represent a formidable clinical challenge. This is primarily driven by the pathogen's escalating multidrug-resistant (MDR) phenotypes, which severely restrict effective therapeutic avenues.

Objective: The primary aim of this investigation was to comprehensively characterize Acinetobacter strains recovered from the blood of ICU patients exhibiting clinical signs of septicemia, with a specific focus on determining their phenotypic antibiotic susceptibility profiles and genotypically identifying key resistance determinants.

Methods: A cross-sectional observational study was executed between July 2021 and June 2024 across the ICUs of two prominent tertiary care centers in Dhaka, Bangladesh: Combined Military Hospital and Kurmitola General Hospital.

Blood specimens from a cohort of 301 patients with suspected septicemia were cultured. The resulting Acinetobacter isolates were subjected to rigorous antimicrobial susceptibility testing and polymerase chain reaction (PCR) analysis to detect underlying resistance genes.

Results: Acinetobacter species were successfully isolated from 46 of the 301 patients, corresponding to a prevalence rate of 15.3%. Phenotypically, these isolates demonstrated alarmingly high resistance levels against ceftazidime (76.1%), followed closely by ceftriaxone and cotrimoxazole (both at 71.7%), cefixime (69.6%), and levofloxacin (63%). Moderate levels of resistance were noted for gentamicin (52.2%), amikacin (50%), netilmicin (47.8%), and piperacillin-tazobactam (43.5%). Notably, 28.3% of isolates were resistant to meropenem. Conversely, colistin and tigecycline retained high efficacy, with resistance rates of 0% and 2.2%, respectively. Molecular analysis of five representative isolates indicated a universal (100%) presence of blaNDM-1, blaCTX-M, and blaTEM-793 genes. Furthermore, blaOXA-48, blaCTX-M-15, blaSHV, and mphA were identified in 80% of these samples, while blaCMY-2 and blaCTX-M-2 were found in 40% and 20%, respectively. Genes such as blaCMY, blaKPC, and other CTX-M variants were absent.

Conclusion: The findings highlight a substantial burden of MDR Acinetobacter spp. in ICU-related bloodstream infections, underpinned by intricate genetic mechanisms that include both ESBL and carbapenemase genes. While colistin and tigecycline currently remain viable therapeutic options, their long-term utility is threatened. These results advocate for continuous resistance surveillance, rigorous infection prevention protocols, and the incorporation of routine molecular diagnostics to steer targeted treatment and effective containment measures.

Keywords: *Intensive Care Unit Sepsis, Acinetobacter Bacteremia, Multidrug Resistance Profiles, Carbapenemase Genes, Molecular Epidemiology.*

Introduction

Bloodstream infections (BSIs) persist as a critical healthcare dilemma for patients admitted to intensive care units (ICUs) due to their profound vulnerability. This heightened susceptibility typically stems from the frequent necessity for invasive medical instrumentation, compromised host immune defenses, and extended durations of hospitalization (Vincent et al., 2009; Allegranzi et al., 2011). Within this high-risk context, *Acinetobacter* species, and most notably *Acinetobacter baumannii*, have emerged as significant opportunistic pathogens. They are frequently implicated in a spectrum of severe nosocomial infections, ranging from ventilator-associated pneumonia and urinary tract infections to wound infections and life-threatening septicemia (Peleg, Seifert and Paterson, 2008; Howard et al., 2012).

The dominance of *A. baumannii* in ICU environments can be largely attributed to its remarkable ability to withstand harsh environmental conditions, its proficiency in forming resilient biofilms, and its rapid acquisition of resistance to a broad array of antimicrobial agents (Dijkshoorn, Nemec and Seifert, 2007; Jawad et al., 1998). These traits facilitate its prolonged survival on medical equipment, inanimate hospital surfaces, and the hands of healthcare personnel, thereby fueling transmission throughout hospital wards (Munoz-Price and Weinstein, 2008). Of particular concern is the escalating prevalence of multidrug-resistant (MDR), extensively drug-resistant (XDR), and pan-drug-resistant (PDR) strains, which drastically narrow the spectrum of viable therapeutic interventions available to clinicians (Magiorakos et al., 2012; CDC, 2019).

The resistance mechanisms employed by *Acinetobacter* spp. are multifaceted, including the synthesis of potent β-lactamases—especially OXA-type carbapenemases capable of hydrolyzing carbapenems—alongside modifications in penicillin-binding proteins, enhanced expression of efflux pump systems, decreased permeability of the outer bacterial membrane, and the integration of foreign resistance genes via mobile genetic elements like plasmids, transposons, and integrons (Poirel and Nordmann, 2006; Evans and Amyes, 2014). Resistance to carbapenems is a critical issue, often strongly associated with specific genes such as *bla*OXA-23, *bla*OXA-24/40, and the highly potent *bla*NDM-1 (Karah et al., 2012; Hamidian and Nigro, 2019). The frequent location of these genes on mobile genetic platforms facilitates their rapid horizontal dissemination within healthcare facilities (Zarrilli et al., 2013).

The accurate identification and detailed characterization of *A. baumannii* isolates require a dual diagnostic approach: phenotypic methods, such as standard culture and antimicrobial susceptibility testing (AST), combined with advanced molecular techniques designed to detect specific resistance genes and aid in epidemiological tracking (CLSI, 2022; Higgins et al., 2010). While phenotypic results are essential for guiding immediate clinical therapy, genotypic analysis provides deeper insights into the underlying molecular mechanisms of resistance and is invaluable for monitoring the spread of resistant clonal lineages (Nowak and Paluchowska, 2016).

Globally, the burden of MDR *A. baumannii* is on the rise, a trend that is especially acute in resource-constrained healthcare systems where infection control measures and antimicrobial stewardship initiatives may be inadequately implemented (Kumarasamy et al., 2010; Ayobami et al., 2019). Patterns of resistance exhibit significant geographical variability, often shaped by local antibiotic prescribing practices and the circulation of specific epidemic clones, such as International Clones I and II (IC1 and IC2) (Roca et al., 2012; Durante-Mangoni and Zarrilli, 2011).

This research was formulated to meticulously examine *Acinetobacter* strains recovered from blood cultures of ICU patients with a clinical diagnosis of septicemia. The study aims to systematically evaluate both their phenotypic antibiotic resistance profiles and the presence of critical resistance genes. It is anticipated that these findings will furnish essential data regarding local resistance trends, thereby informing more effective targeted therapeutic strategies and bolstering infection control efforts within critical care settings.

Methodology Study Setting:

This observational, cross-sectional research was carried out within the Intensive Care Units of two prominent healthcare institutions in Dhaka, Bangladesh—Combined Military Hospital and Kurmitola General Hospital—spanning the period from July 2021 through June 2024.

Study Population: The study enrolled a total of 301 hospitalized patients who presented with clinical signs suggestive of septicemia. For each participant, a comprehensive medical history was recorded, complemented by a thorough physical examination. Enrollment was strictly contingent upon obtaining informed consent from the patients' legal guardians. **Study**

Procedure: From each enrolled patient, approximately 8–10 mL of venous blood was collected under strict aseptic conditions and immediately inoculated into designated blood culture bottles. These bottles were subsequently incubated in an aerobic environment at 37°C utilizing the automated BD BACTEC FX Blood Culture System.

Isolation and Identification of Bacterial Isolates: Blood culture bottles that flagged positive for growth were subcultured onto both Blood agar and MacConkey agar plates, followed by aerobic incubation at 37°C. Initial identification of any resulting bacterial growth was based on colony morphological characteristics, Gram staining reactions, and standard biochemical assays. To guarantee maximum diagnostic accuracy, all pure isolates underwent definitive confirmation using the BD Phoenix M50 automated microbial identification platform.

Antimicrobial Susceptibility Testing: The susceptibility of isolates to various antibiotics was assessed employing the Kirby-Bauer disc diffusion technique on Mueller-Hinton agar, in strict adherence to the guidelines established by the Clinical and Laboratory Standards Institute (CLSI). Isolates were categorized as susceptible or resistant based on the measured zones of inhibition; for the purpose of this analysis, any isolates exhibiting intermediate susceptibility were reclassified as resistant. The panel of antibiotics tested included amikacin, gentamicin, netilmicin, ceftazidime, ceftriaxone, colistin, piperacillin-tazobactam, meropenem, tigecycline, cefixime, levofloxacin, and cotrimoxazole.

Molecular Characterization: A subset of five *Acinetobacter* isolates, identified as multidrug-resistant, was randomly selected for further molecular analysis via polymerase chain reaction (PCR).

1) **DNA Extraction:** Genomic DNA was successfully extracted from these five selected isolates utilizing the QIAamp DNA Mini Kit (QIAGEN, Germany), strictly following the manufacturer's operational protocols.

2) **PCR:** Amplification of specific target resistance genes was performed using GoTaq Green Master Mix (Promega, USA) within a C1000 Touch Thermal Cycler (Bio-Rad, USA). The resulting PCR products were resolved by electrophoresis on a 1.5% agarose gel (Biometra Agarose Gel Mini System, Germany) stained with ethidium bromide (1 µL/mL) for 30 minutes. The electrophoresis run was conducted at 100V using 1X TAE buffer. Visualization of the amplified DNA bands was achieved using a GelDoc Go Imaging System (Bio-Rad, USA), with amplicon sizes verified against a 100 bp DNA ladder (New England BioLabs, USA).

Table 1: PCR Primers Used in the Study (Data maintained as per original)

Gene	**Primer used**	**Amplicon size (bp)**	**References**
bla$_{TEM}$	F: CATTTCCGTGTCGCCCTTAT	793	(Randall *et al.*, 2004)
	R: TCCATAGTTGCCTGACTCCC		
blaCTX-M	F: ATGTGCAGYACCAGTAARGTKATGGC	592	(Gundran *et al.*, 2019)
	R: TGGGTRAARTARGTSACCAGAAYSAGCGG		
bla$_{CMY}$	F: TGGCCAGAACTGACAGGCAAA	462	(Mandakini *et al.*, 2020)
	R: TTTCTCCTGAACGTGGCTGGC		
bla$_{SHV}$	F: TCGCCTGTGTATTATCTCCC	768	(Van *et al.*, 2008)
	R: CGCAGATAAATCACCACAATG		
bla$_{KPC}$	F: CGTCTAGTTCTGCTGTCTTG	798	(Poirel *et al.*, 2011)
	R: CTTGTCATCCTTGTTAGGCG		
blaCTX-M-15	F: CACACGTGGAATTTAGGGACT	996	(Paterson *et al.*, 2003)
	R: GCCGTCTAAGGCGATAAACA		
blaCTX-M-2	F: CGGTGCTTAAACAGAGCGAG	624	(Thabit *et al.*, 2011)
	R: CCATGAATAAGCAGCTGATTGCCC		

blaCTX-M-8	F: ACGCTCAACACCGCGATC	490	(Thabit *et al.*, 2011)
	R: CGTGGGTTCTCGGGGATAA		
blaCTX-M-9	F: GATTGACCGTATTGGGAGTTT	947	(Thabit *et al.*, 2011)
	R: CGGCTGGGTAAAATAGGTCA		
blaNDM1	F: GGTTTGGCGATCTGGTTTTC	621	(Poirel *et al.*, 2011)
	R: CGGAATGGCTCATCACGATC		
blaOXA-48	F: GCGTGGTTAAGGATGAACAC	438	(Poirel *et al.*, 2011)
	R: CATCAAGTTCAACCCAACCG		
blaCMY-2	F: TGGCCGTTGCCGTTATCTAC	870	(Torkan, Khamesipour and Anyanwu, 2015)
	R: CCCGTTTTATGCACCCATGA		
mphA	F: GTGAGGAGGAGCTTCGCGAG	403	(Nguyen *et al.*, 2009)
	R: TGCCGCAGGACTCGGAGGTC		

Statistical Analysis: All pertinent data were prospectively gathered and documented in standardized data collection forms. Subsequent processing and analysis were conducted utilizing the Statistical Package for the Social Sciences (SPSS) software, version 27. Descriptive statistical methods were employed to summarize the findings, which are presented in appropriate tables and graphical charts.

Ethical Clearance: The study protocol received formal ethical approval from the Ethical Review Committee of the Directorate General of Medical Services (DGMS) as well as the institutional authorities of Kurmitola General Hospital (KGH), Dhaka.

Results Among the 301 patients admitted to the Intensive Care Unit with suspected septicemia, *Acinetobacter* species were successfully isolated from 46 individuals, representing an isolation rate of 15.3% of the total cohort.

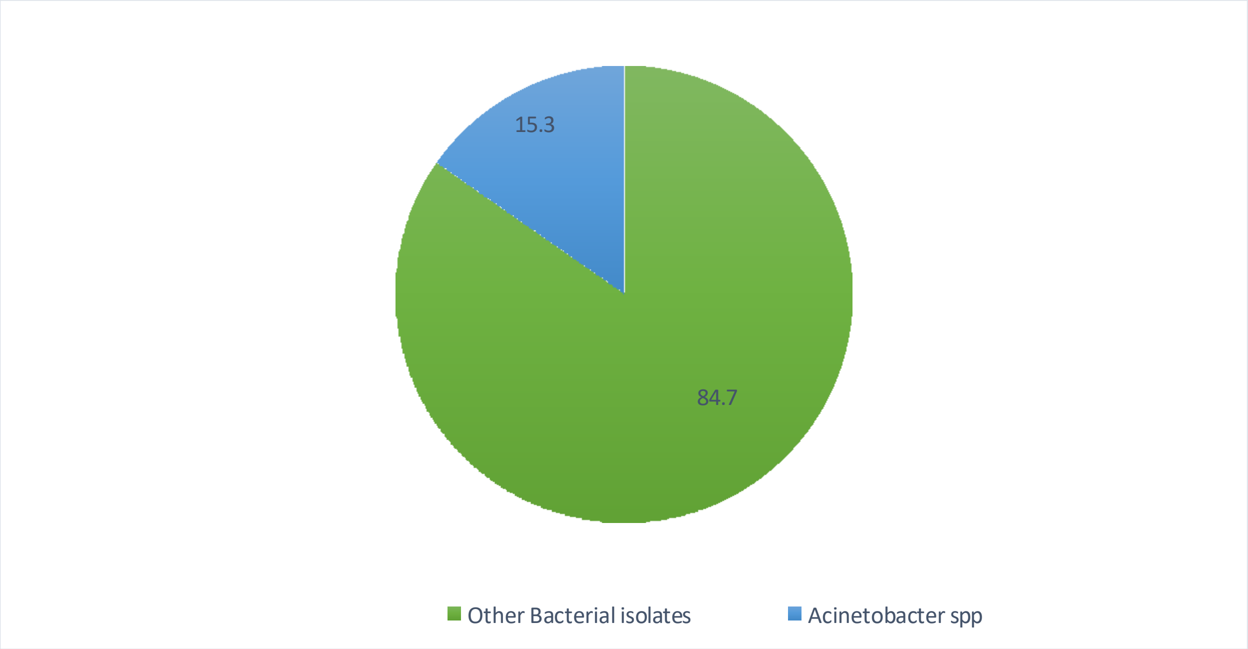

Figure 1: This pie chart illustrates the percentage of Acinetobacter species responsible for septicemia among ICU admitted patients (n=301), showing 15.3% Acinetobacter spp. and 84.7% other bacterial isolates.

The 46 *Acinetobacter* strains isolated from blood cultures exhibited well-defined antibiotic resistance profiles. The most profound resistance was noted against ceftazidime (76.1%), with high rates also seen for cotrimoxazole and ceftriaxone (both at 71.7%), cefixime (69.6%), and levofloxacin (63.0%). Moderate levels of resistance were recorded for the aminoglycosides: gentamicin (52.2%), amikacin (50.0%), and netilmicin (47.8%). Comparatively lower resistance was observed against piperacillin-tazobactam (43.5%) and meropenem (28.3%). Most notably, tigecycline demonstrated high efficacy with a minimal resistance rate of 2.2%, while colistin remained completely effective, showing 0% resistance across all isolates tested.

Table 2: Resistance pattern of Acinetobacter species isolated from blood culture in the study (n=46)

Antibiotic	**Resistance pattern Frequency (n)**	**Percentage (%)**
Cotrimoxazole	33	71.7
Levofloxacin	29	63
Ceftriaxone	33	71.7
Gentamicin	24	52.2

Ceftazidime	35	76.1
Amikacin	23	50
Meropenem	13	28.3
Netilmicin	22	47.8
Cefixime	32	69.6
Tazobactam + Piperacillin	20	43.5
Colistin	0	0
Tigecycline	1	2.2

Genotypic analysis of the five randomly selected *Acinetobacter* spp. isolates revealed a widespread distribution of various resistance genes. The genes *bla*CTX-M, *bla*NDM-1, and *bla*TEM-793 were universally present, detected in 100% of these isolates. Other genes, specifically *bla*CTX-M-15, *bla*OXA-48, *bla*SHV, and *mph*A, were each identified in 80% of the samples. The *bla*CMY-2 gene was found in 40% of the isolates, whereas *bla*CTX-M-2 was present in 20%. In contrast, genes such as *bla*CMY, *bla*CTX-M-8, *bla*CTX-M-9, and *bla*KPC were not detected in any of the tested isolates (0%).

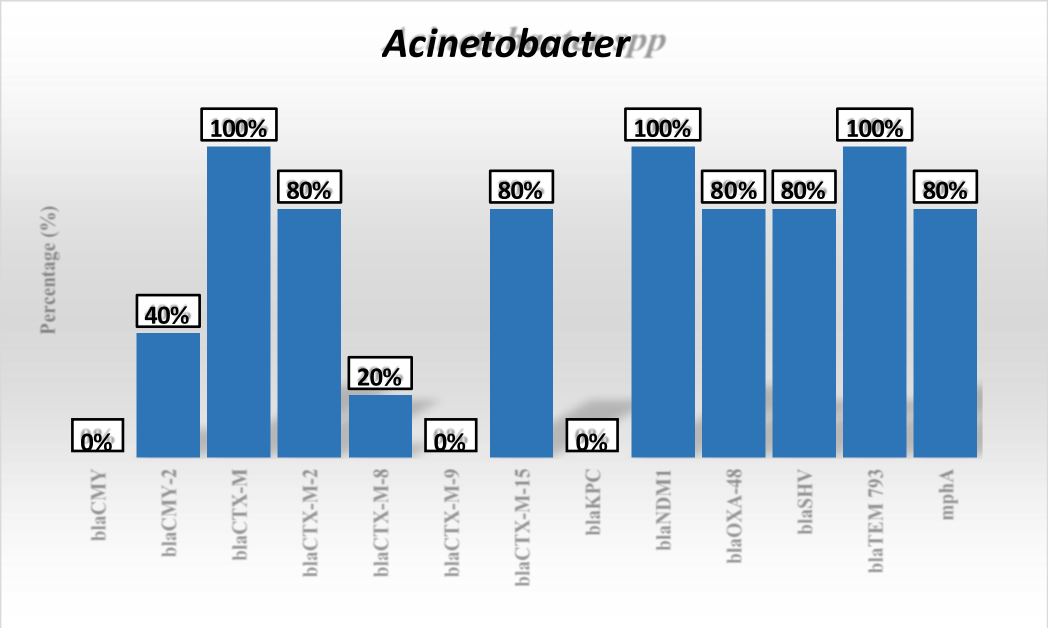

Figure 2: Distribution of different antibiotic resistance genes for randomly selected Acinetobacter spp (n=5).

This figure illustrates the distribution of various antibiotic resistance genes among five randomly selected Acinetobacter spp. isolates. The blaCTX-M, blaNDM-1, and blaTEM-793 genes were detected in all isolates (100%). Other frequently identified genes included blaCTX-M-15, blaOXA-48, blaSHV, and mphA, each found in 80% of *the isolates. The blaCMY-2 gene was present in 40% of the isolates, while blaCTX-M-2 was detected in 20%. In contrast, blaCMY, blaCTX-M-8, blaCTX-M-9, and blaKPC genes were not detected in any of the isolates (0%).]*

Discussion

The rise of *Acinetobacter baumannii* as a dominant pathogen responsible for bloodstream infections (BSIs), particularly within critical care environments, has escalated into a major global health concern, largely driven by its profound resistance to numerous antibiotic classes (Peleg et al., 2008). In the current investigation, *Acinetobacter* species were recovered from 15.3% of the 301 ICU patients with suspected septicemia. This prevalence aligns with previous reports from comparable hospital settings, which typically document isolation rates between 10% and 20% (Howard et al., 2012). Such findings reinforce the significant status of *Acinetobacter baumannii* as a persistent nosocomial pathogen, especially threatening to patients who are immunocompromised or require mechanical ventilation.

The antimicrobial susceptibility profiles documented in this study reveal a disconcerting trend toward high resistance. The isolates showed peak resistance to ceftazidime (76.1%), cotrimoxazole (71.7%), ceftriaxone (71.7%), cefixime (69.6%), and levofloxacin (63%). These results corroborate existing literature that indicates widespread resistance among *Acinetobacter* spp. to third-generation cephalosporins and fluoroquinolones, often mediated by mechanisms such as β-lactamase production and the overexpression of efflux pumps (Roca et al., 2012). Furthermore, the substantial resistance to cotrimoxazole points towards the likely acquisition of sulfonamide resistance genes, potentially facilitated through plasmid-mediated horizontal transfer (Lee et al., 2017).

Resistance rates were notably lower for aminoglycosides, specifically gentamicin (52.2%), amikacin (50%), and netilmicin (47.8%), as well as for meropenem (28.3%). While these rates are not negligible, they suggest that aminoglycosides might still offer some therapeutic value, particularly when utilized as part of combination regimens. However, the gradual upward trend in aminoglycoside resistance, often attributed to aminoglycoside-modifying enzymes and 16S rRNA methyltransferases, is a growing concern (Zarrilli et al., 2013). While these agents retain some utility, their slowly diminishing efficacy is alarming, particularly regarding meropenem, which is frequently the drug of last resort for severe MDR infections (Munoz-Price & Weinstein, 2008). The fact that nearly one-third of isolates exhibited carbapenem resistance highlights the emerging threat of Carbapenem-Resistant *Acinetobacter baumannii* (CRAB), a pathogen designated as a critical priority by the World Health Organization (WHO, 2017).

Encouragingly, resistance to tigecycline was exceptionally rare (2.2%), and no isolates demonstrated resistance to colistin (0%). This indicates that these two agents remain viable therapeutic alternatives, although the known nephrotoxicity of colistin and the potential for heteroresistance warrant its cautious administration (Nation et al., 2015). The low incidence of tigecycline resistance observed here mirrors global reports, supporting its continued role as a salvage therapy for complex MDR *Acinetobacter* infections (Castanheira et al., 2014).

From a genotypic standpoint, all five representative isolates carried *bla*CTX-M, *bla*NDM-1, and *bla*TEM-793 genes, indicative of extensive β-lactamase-driven resistance. The presence of the *bla*NDM-1 gene, which encodes the New Delhi metallo-β-lactamase, is particularly worrisome given its strong association with carbapenem resistance and its high potential for plasmid-mediated dissemination across different bacterial species (Kumarasamy et al., 2010). The universal detection of *bla*CTX-M, with *bla*CTX-M-15 found specifically in 80% of the isolates, further underscores the significant burden of extended-spectrum β-lactamases (ESBLs) in these strains, a finding consistent with global resistance trends (Cantón et al., 2012).

The identification of additional resistance genes, including *bla*OXA-48 (80%), *bla*SHV (80%), and *mph*A (80%), points to a complex and robust resistome capable of ensuring survival under varied antimicrobial pressures. While *bla*OXA-48, a class D carbapenemase, is typically associated with carbapenem-resistant *Enterobacteriaceae*, its increasing detection in *Acinetobacter baumannii* suggests likely acquisition via horizontal gene transfer (Poirel et al., 2012). Similarly, the *mph*A gene, which confers resistance to macrolides, while not clinically pivotal for treating *Acinetobacter* infections, serves as an indicator of co-selection pressure and the overall genetic diversity present within these isolates.

Conversely, the absence of genes such as *bla*CMY, *bla*CTX-M-8, *bla*CTX-M-9, and *bla*KPC may reflect distinct local epidemiological patterns regarding plasmid circulation and specific antibiotic usage. The presence of *bla*CMY-2 in 40% of isolates and *bla*CTX-M-2 in 20% suggests a heterogeneous distribution of AmpC β-lactamases within the studied bacterial population.

Collectively, these findings emphasize the critical necessity for sustained surveillance, rigorous antimicrobial stewardship programs, and stringent infection control protocols within ICU settings. The high prevalence of multidrug resistance, coupled with the co-harboring of multiple resistance genes in *A. baumannii* isolates, presents a formidable obstacle to effective patient treatment and hospital-wide infection control initiatives. Furthermore, the molecular characterization of resistance genes, as implemented in this study, is indispensable for grasping the mechanisms that fuel resistance and for tailoring empirical therapeutic approaches. Future research endeavors should prioritize whole-genome sequencing to uncover novel resistance determinants and to track clonal transmission pathways within healthcare facilities more precisely.

Conclusion

This investigation underscores the substantial clinical burden imposed by *Acinetobacter* species bloodstream infections in ICU patients and highlights the escalating global challenge of antimicrobial resistance. The isolated strains demonstrated high levels of resistance to several widely utilized antibiotic classes—most notably third-generation cephalosporins, fluoroquinolones, and sulfonamides—while fortunately retaining susceptibility to colistin and tigecycline. The identification of diverse resistance genes, including *bla*NDM-1, *bla*CTX-M, *bla*TEM, and *bla*OXA-48, illuminates the complex genetic architecture driving multidrug resistance in these pathogens. The alarming coexistence of both extended-spectrum β-lactamase (ESBL) and carbapenemase-encoding genes within bloodstream isolates signals a high potential for treatment failure and severely restricts available therapeutic choices. These critical findings necessitate urgent and persistent surveillance of antimicrobial resistance, the implementation of strict infection control measures, and judicious antibiotic prescribing practices, particularly in high-risk critical care environments. Integrating the molecular characterization of resistance determinants into routine microbiological diagnostic workflows is essential for informing effective empirical therapy and fortifying infection prevention strategies.

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