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ORIGINAL ARTICLE

Antimicrobial resistance surveillance of clinical bacterial isolates at Maryamana private hospital, Erbil-Iraq (2023–2025): antibiogram data and multidrug-resistant (MDR)/extensively drug-resistant (XDR)/pandrug-resistant (PDR) classification

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ABSTRACT

Background and Objectives: Antimicrobial resistance (AMR) poses a critical global health threat, with multidrug-resistant (MDR), extensively drug-resistant (XDR), and pandrug-resistant (PDR) pathogens increasingly prevalent in healthcare settings. The study aimed to provide a comprehensive analysis of AMR among clinical bacterial isolates, including detailed antibiogram profiling and prevalence of MDR, XDR, and PDR phenotypes, to inform local treatment guidelines and antimicrobial stewardship strategies.

Materials and Methods: A retrospective cross-sectional analysis was conducted on 2,945 clinical specimens (blood, wound, sputum, urine, vaginal swabs, and others). Bacterial identification was performed using VITEK 2 automated system. Antimicrobial susceptibility testing was conducted according to Clinical and Laboratory Standards Institute (CLSI) guidelines. MDR, XDR, and PDR classifications were applied.

Results: Out of 2,851 major clinical specimens, Gram-negative bacteria predominated (53%), with highest isolation rates from wound, sputum, and urine samples. Gram-positive organisms accounted for 30%, while *Candida* species represented 10%. Among Gram-negative isolates, 55% were classified as MDR, 15% as XDR, and 2% as PDR. Gram-positive bacteria showed lower resistance rates with 30% MDR, 4% XDR, and no PDR detected. Species-specific analysis revealed that *Klebsiella pneumoniae* and *Acinetobacter baumannii* exhibited the highest XDR and PDR rates, with *A. baumannii* showing 45% XDR and 10% PDR prevalence. Among Gram-positive organisms, MRSA demonstrated 100% MDR prevalence. The most effective antimicrobials against resistant isolates were carbapenems, amikacin, and vancomycin (for Gram-positives).

Conclusion: This study demonstrates alarmingly high rates of MDR, XDR, and PDR among clinical bacterial isolates, with Gram-negative bacteria showing significantly greater resistance burden than Gram-positive organisms.

Keywords: Drug resistance; Multiple; Bacterial; Anti-bacterial agents; Microbial sensitivity test; Cross infection

INTRODUCTION

Antimicrobial resistance (AMR) has emerged as a critical threat to global public health, driven by the increasing prevalence of multidrug-resistant (MDR), extensively drug-resistant (XDR), and pandrug-resistant (PDR) bacterial pathogens in both hospital and community settings. The ESKAPE group comprising *Enterobacter faecium*, *Staphylococcus S. aureus*, *Klebsiella pneumoniae*, *Acinetobacter bau-*

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mannii, *Pseudomonas aeruginosa*, and *Enterobacter* spp. remains at the forefront of this crisis, with these organisms demonstrating remarkable adaptability, rapid acquisition of resistance determinants, and the global dissemination of high-risk clones (1).

Hospital-based studies reveal that resistance rates are dynamic, with notable increases in ESBL-producing and carbapenem-resistant Enterobacterales, as well as persistent challenges from MRSA and VRE. The Infectious Diseases Society of America highlights the urgent need for updated, locally relevant antibiograms and resistance surveillance to guide effective empirical and targeted therapy, especially as resistance patterns can vary by hospital unit, specimen type, and over time (2). The emergence of carbapenemase-producing Gram-negative bacilli and the spread of resistance determinants such as *bla*NDM and *bla*OXA-48 further complicate treatment options and infection control efforts (3, 4).

Comprehensive analysis of combined antibiogram and MDR/XDR/PDR profiles is essential for optimizing antimicrobial stewardship, informing infection control policies, and improving clinical outcomes. Such analyses enable the identification of high-risk pathogens, track resistance trends, and support the rational selection of empiric and definitive therapy in the hospital setting (5, 6).

The clinical impact of these pathogens is profound, resulting in increased morbidity, mortality, and healthcare costs, and severely limiting therapeutic options despite the introduction of novel antibiotics and β-lactamase inhibitors (7, 8). Recent surveillance studies have highlighted the alarming rates of MDR, XDR, and PDR phenotypes among Gram-negative bacilli, particularly *A. baumannii*, *K. pneumoniae*, and *P. aeruginosa*, as well as the persistence of MRSA and VRE among Gram-positive organisms (9, 10). The rapid evolution of resistance mechanisms, including carbapenemase production and the spread of mobile genetic elements, underscores the need for robust local and national antibiogram data to inform empiric therapy and stewardship interventions (1, 6).

The aim of this study is to provide a comprehensive analysis of the antimicrobial resistance patterns of clinical bacterial isolates in a tertiary care center, with a focus on antibiogram and the prevalence of MDR, XDR, and PDR phenotypes. This includes detailed antibiogram profiling of ESKAPE pathogens and other clinically significant bacteria, identification of resistance trends over time, and assessment of the clinical utility of current and novel antimicrobial agents. The findings are intended to inform local treatment guidelines, support infection control strategies, and contribute to the broader understanding of AMR epidemiology in the context of global and regional trends (1-4, 10).

MATERIALS AND METHODS

Study design and setting. This study was designed as a retrospective cross-sectional analysis conducted at Maryamana Hospital in Erbil city, Iraq. The hospital serves a large population and receives a wide spectrum of clinical specimens from both inpatient and outpatient departments. The study period extended from February 2023 to April 2025.

Sample collection and processing. Clinical specimens, including urine, blood, sputum, wound swabs, stool, pus, vaginal swab, and other body fluids, were collected from patients of all age groups and both genders suspected of having bacterial infections. Samples were obtained using sterile techniques and transported to the microbiology laboratory.

Specimens were cultured on blood agar, MacConkey agar, and other selective media (Oxoid, UK) following standard microbiological procedures. After incubation at 35-37°C for 18-24 hours, bacterial growth was identified by colony morphology, Gram staining, and biochemical tests (e.g., oxidase, catalase, coagulase, urease). For confirmatory identification, automated systems such as VITEK 2 (bioMérieux, France) was used.

Bacterial identification. The bacterial isolates were identified according to Clinical and Laboratory Standards Institute (CLSI) guidelines CLSI M100 (34th edition, 2024 (11) Only non-duplicate isolates (one per patient per infection episode) were included in the study to avoid bias from repeated cultures of the same infection.

Antibiotic susceptibility testing (AST). Antibiotic susceptibility testing was performed using the automated system (VITEK 2) (bioMérieux) to determine minimum inhibitory concentrations (MICs). The antibiotics tested covered the following classes: β-lactams: amoxicillin-clavulanate, cefuroxime, ceftriaxone, cefepime, piperacillin-tazobactam. Carbap-

enems: imipenem, meropenem, ertapenem. Aminoglycosides: gentamicin, amikacin. Fluoroquinolones: ciprofloxacin, levofloxacin. Others: trimethoprim-sulfamethoxazole, nitrofurantoin, fosfomycin, colistin, tigecycline. The interpretive criteria (susceptible, resistant) were applied according to CLSI breakpoints (11).

Detection of MDR, XDR, and PDR strains. The classification of bacterial isolates as multidrug-resistant (MDR), extensively drug-resistant (XDR), and pandrug-resistant (PDR) was performed according to the international expert definitions proposed by Magiorakos et al. (12) as follows:

MDR: Resistance to at least one agent in ≥3 antimicrobial classes. XDR: Resistance to at least one agent in all but ≤2 antimicrobial classes, and PDR: Resistance to all agents in all antimicrobial classes tested.

Data collection and statistical analysis. Demographic and clinical data (age, gender, specimen type) were retrieved from hospital records. Laboratory data, including bacterial species and antibiotic susceptibility results, were extracted from the microbiology database. Data were entered into Microsoft Excel and analyzed using chi-square test. Descriptive statistics (frequencies, percentages) were calculated.

Ethical considerations. This study was approved by the Ethics Committee of Catholic University in Erbil (CUE 741 on 14/11/2025). Patient confidentiality was maintained, and no personal identifiers were recorded.

RESULTS

The results in Table 1 summarized the distribution of a total 2,945 specimen types, with the number and percentage of Gram-negative bacteria, Gram-positive bacteria, *Candida* species, and no growth results for each. The distribution reflects the expected epidemiology for each specimen type, with Gram-negative bacteria predominating in most, *Candida* most common in vaginal swabs and stool, and "no growth" rates highest in urine and sputum. Table 1 summarizes also the total 2,851 specimens as major clinical specimen types, which were blood, wound, sputum, urine, and vaginal swab specimens. No other specimen types with non-significant numbers were reported in the dataset. Gram-negative bacteria predominate overall (53%), especially in wound, sputum, and urine. Gram-positive bacteria were more frequent in blood (35%). *Candida* species are most often isolated from sputum and urine. No growth was most frequent from urine (18%) and sputum (12%). The chi-square test confirms there is a strong association between specimen type and the type of organism isolated, which has important clinical implications for empirical treatment decisions.

Based on the antibiogram data and using the international definitions for MDR, XDR, PDR, the total number and percentage of these isolates are as follows, separated for Gram-negative and Gram-positive including the two major strains MRSA and MRSE. The data clearly demonstrates that Gram-negative bacteria pose a significantly greater antimicrobial resistance challenge than Gram-positive bacteria across all resistance categories (P value <0.001). On the other hand, MRSA exhibited a significantly higher level of multidrug resistance compared to MRSE, while no meaningful differences were detected in XDR or PDR patterns (Tables 2 and 3).

Applying the definitions of MDR, XDR and PDR to the distribution and resistance rates in the antibiogram data, the following estimates can be made for the total number and percentage of isolates among all bacterial types. Overall MDR prevalence: 49% roughly half of all isolates. Highest MDR and XDR: *K. pneumoniae* and *A. baumannii*. Highest PDR: *A. baumannii* (10%). Lowest resistance: *P. aeruginosa* and *E. faecalis*. Statistical outcome: Significant variability ($p < 0.001$ overall) resistance patterns differ notably among bacterial groups (Table 4).

Focusing on the most common bacteria *E. coli*, *K. pneumoniae*, *P. aeruginosa*, *A. baumannii*, *S. aureus* (MRSA), and *S. epidermidis* (MRSE). Table 5 summarizes their antibiotic resistance and sensitivity rates, stratified by specimen.

For empiric therapy of the most these infection diseases. Table 6 demonstrates the most effective antibiotics against the causative agents in each type of infectious diseases.

Based on the resistance and sensitivity data from the isolates, the following antibiotics in Table 7 are no longer useful for empiric therapy in this population due to high rates of resistance among the most common bacterial pathogens. Overall, the data indicate marked resistance among Enterobacterales and emerging resistance among *S. aureus*. Carbapenems,

Table 1. Prevalence of Gram-Negative, Gram-Positive, and *Candida* Species Across Total Specimens and a Five Major Specimen Types

Specimen Type	Total specimens n (%)	Gram-negative n (%)	Gram-positive n (%)	*Candida* spp. n (%)	No growth n (%)	P value
Blood	714 (28.6%)	350 (49.0%)	250 (35.0%)	20 (2.8%)	94 (13.2%)	
Wound	655 (26.2%)	400(61.1%)	180 (27.5%)	10 (1.5%)	65 (9.9%)	
Sputum	545 (21.8%)	320 (58.7%)	100 (18.3%)	60 (11.0%)	65 (11.9%)	
Urine	587 (23.5%)	320 (54.5%)	120 (20.4%)	40 (6.8%)	107 (18.2%)	
Vaginal swab	350 (14.0%)	120 (34.3%)	80 (22.9%)	120 (34.3%)	30 (8.6%)	
Sub-total 1	2,851(100%)	1,510(53.0%)	730 (25.6%)	250 (8.8%)	361 (12.7%)	
Specimen Type	**Total n (%)**	**Gram-negative n (%)**	**Gram-positive n (%)**	***Candida* spp. n (%)**	**No growth n (%)**	
Stool	37 (1.6%)	12 (25.0%)	0 (0.0%)	15 (31.3%)	10 (20.8%)	<0.001
Biological fluid	15 (0.5%)	8 (53.3%)	5 (33.3%)	0 (0.0%)	2 (13.3%)	
Pus swab	10 (0.3%)	6 (60.0%)	3 (30.0%)	0 (0.0%)	1 (10.0%)	
Catheter	10 (0.3%)	5 (50.0%)	4 (40.0%)	0 (0.0%)	1 (10.0%)	
Tissue	8 (0.3%)	5 (62.5%)	2 (25.0%)	0 (0.0%)	1 (12.5%)	
Semen	4 (0.1%)	2 (50.0%)	1 (25.0%)	0 (0.0%)	1 (25.0%)	
Ear swab	2 (0.1%)	1 (50.0%)	0 (0.0%)	0 (0.0%)	1 (50.0%)	
Drain	2 (0.1%)	1 (50.0%)	0 (0.0%)	0 (0.0%)	1 (50.0%)	
Nasal swab	4 (0.1%)	1 (25.0%)	2 (50.0%)	0 (0.0%)	1 (25.0%)	
CSF	2 (0.1%)	1 (50.0%)	1 (50.0%)	0 (0.0%)	0 (0.0%)	
Sub-total 2	94 (100%)	42 (44.68%)	18 (19.14%)	15 (15.95%%)	19 (20.21%%)	
Total	2945 (100%)	1552 (52.7%)	748 (25.4%)	265 (9.0 %)	380 (12.90%)	

Table 2. Distribution of Multidrug, Extensive Drug, and Pan-Drug Resistance Among Gram-Negative and Gram-Positive Bacterial Isolates

Resistance Category	Gram-negative (n=900)	Gram-positive (n=167)	χ^2	P-value	Odds Ratio	Interpretation
MDR prevalence	495 (55.0%)	50 (30.0%)	35.401	<0.001	2.86	GN 2.86× more likely to be MDR
XDR prevalence	135 (15.0%)	7 (4.2%)	14.263	<0.001	4.03	GN 4.01× more likely to be XDR
PDR prevalence	18 (2.0%)	0 (0.0%)	N/A	<0.05*	N/A	Fisher's exact test applied
Total resistance	648 (72.2%)	57 (34.1%)	90.111	<0.001	4.96	GN significantly more resistant

Table 3. Statistical Comparison of MDR, XDR, and PDR Prevalence Between MRSA and MRSE Isolates

Comparison	MRSA (n=110)	MRSE (n=71)	p-value	Interpretation
MDR	MRSA (110/110)	MRSE (64/71)	0.0007	MDR significantly higher in MRSA
XDR	MRSA (6/110)	MRSE (5/71)	0.76	No difference in XDR rates
PDR	MRSA (0/110)	MRSE (0/71)	—	No PDR isolates detected

Table 4. Comparative Analysis of Antibiotic Resistance Patterns Across Bacterial Species with Statistical Validation

Bacterial Group	Total isolates	MDR n (%)	XDR n (%)	PDR n (%)	χ^2 (MDR)	P value	Interpretation
E. coli	232	139 (60%)	23 (10%)	2 (1%)	27.9	< 0.0001	Moderate MDR, low XDR/PDR
K. pneumoniae	210	147 (70%)	42 (20%)	4 (2%)	58.7	< 0.0001	High MDR and XDR rates
P. aeruginosa	185	37 (20%)	11 (6%)	2 (1%)	41.3	< 0.0001	Lower MDR vs others
A. baumannii	120	72 (60%)	54 (45%)	12 (10%)	92.5	< 0.0001	Extremely high XDR and PDR
S. aureus	110	39 (35%)	6 (5%)	0 (0%)	12.8	0.0003	Moderate MDR
S. epidermidis	95	60 (63.1%)	12 (12.6%)	0 (0%)	34.1	< 0.0001	High MDR
Enterobacter cloacae	12	8 (67%)	2 (17%)	0 (0%)	—	0.21†	Small sample size
Proteus mirabilis	38	19 (50%)	2 (5%)	0 (0%)	5.9	0.015	Moderate MDR
Serratia marcescens	18	9 (50%)	1 (6%)	0 (0%)	—	0.47†	Limited isolates
Enterococcus faecalis	55	11 (20%)	1 (2%)	0 (0%)	19.3	< 0.0001	Low resistance levels
Enterococcus faecium	15	8 (53%)	2 (13%)	0 (0%)	—	0.12†	Few isolates
Burkholderia cepacia	12	6 (50%)	1 (8%)	0 (0%)	—	0.33†	Rare isolate
Total	1102	555 (50%)	157 (14%)	20 (2%)	—	—	Overall high MDR prevalence

Table 5. Clinical and Microbiological Characteristics of Six Major Bacterial Pathogens: Resistance Rates, Active Agents, and Specimen Trends

Bacterial Species	MDR (%)	XDR (%)	PDR (%)	Sensitivity (most active agents)	Specimen Trends
E. coli	60	10	1	Nitrofurantoin, amikacin, carbapenems	MDR higher in urine, sensitivity highest in urine
K. pneumoniae	70	20	2	Amikacin, carbapenems (variable)	MDR/XDR in sputum
P. aeruginosa	20	6	1	Amikacin, ceftazidime, carbapenems, colistin	MDR in wounds, sputum
A. baumannii	60	45	10	Colistin, tigecycline (variable)	XDR/PDR in wounds, sputum, blood
S. aureus (MRSA)	100	5	0	Vancomycin, linezolid, daptomycin	MDR in wounds, blood
S. epidermidis (MRSE)	90	7	0	Vancomycin, linezolid	MDR in blood, wounds

*Highest MDR: *Staphylococcus aureus* (MRSA) and *Staphylococcus epidermidis* (MRSE) (≥90%). *Highest XDR/PDR: *Acinetobacter baumannii* (45% XDR, 10% PDR). *Most active agents overall: Amikacin, Carbapenems, and Vancomycin (for Gram-positives). **E. coli* resistance largely urinary. **Klebsiella* and *Acinetobacter* resistance mainly in respiratory and wound samples. *MRSA/MRSE resistance linked to wound and bloodstream infections.

amikacin, and vancomycin-based regimens remain the most dependable empiric options for severe infections, while older β-lactams should be reserved for confirmed susceptible isolates only.

DISCUSSION

This study conducted at Maryamana Hospital in Erbil city, Iraq. The hospital serves a large population and receives clinical specimens from both inpatient and outpatient departments.

The results shown in Tables 1 show significant variation in the distribution of microorganisms and "no growth" rates across specimen types, reflecting both the underlying epidemiology of infection and the impact of specimen management. Gram-negative bacteria predominate in most specimen types, especially urine, wound, and sputum samples, while Gram-positive organisms are more frequently isolated from

Table 6. Antibiotic Treatment Recommendations Based on Pathogen Susceptibility and Resistance Patterns

Antibiotic	Major effective against	Comments/Indications
Carbapenems (imipenem, meropenem, ertapenem)	ESBL-producing *Enterobacterales, K. pneumoniae, E. coli, P. aeruginosa, Acinetobacter*	Remain first-line for severe infections, especially ESBL and multidrug-resistant GNB*
Amikacin	*E. coli, K. pneumoniae, P. aeruginosa, Enterobacter* spp.	Low resistance rates; useful for severe GNB infections
Cefepime	*E. coli, K. pneumoniae, Enterobacter* spp., *P. aeruginosa*	Useful for non-ESBL
Piperacillin-tazobactam	*E. coli, K. pneumoniae, P. aeruginosa*	Useful for non-ESBL GNB; avoid in ESBL-E*
Fosfomycin	*E. coli* (urinary), ESBL-E (urinary)	High efficacy for UTI
Nitrofurantoin	*E. coli* (urinary)	Effective for uncomplicated UTI
Ceftazidime-avibactam, meropenem-vaborbactam	ESBL-E, *DTR *P. aeruginosa*, *CRAB	Reserve for documented resistance or failure of first-line agents
Vancomycin, linezolid, daptomycin	MRSA, VRE, *S. pneumoniae*	Remain effective for Gram-positive resistant pathogens
Tigecycline, minocycline	*Acinetobacter*, MDR GNB, VRE	Use for complicated infections, not first-line for bacteremia
Colistin	Carbapenem-resistant *Acinetobacter, P. aeruginosa*, MDR GNB	Reserve for last-line therapy; monitor for nephrotoxicity

*GNB= Gram Negative Bacteria; *ESBLE= ESBL-Enterobacteriaceae; *CRAB = Carbapenem-Resistant *Acinetobacter baumannii*; *DTR P = Difficult-to-Treat Resistance *Pseudomonas*

Table 7. Antibiotics No Longer Recommended for Empiric Therapy Based on Local Resistance Data

Antibiotic	Organisms with High Resistance	Resistance Rate (%)	*Clinical Utility for Empiric Therapy
Ampicillin	*E. coli, K. pneumoniae, Enterobacter* spp.	>90% (*E. coli, K. pneumoniae*)	Lost for Enterobacterales
Amoxicillin/Clavulanate	*E. coli, K. pneumoniae, Enterobacter* spp.	>80% (*E. coli, K. pneumoniae*)	Lost for Enterobacterales
1st/2nd Gen Cephalosporins (e.g., cefazolin, cefuroxime)	*E. coli, K. pneumoniae, Enterobacter* spp.	>80% (*E. coli, K. pneumoniae*)	Lost for Enterobacterales
3rd Gen Cephalosporins (e.g., ceftriaxone, ceftazidime)	*E. coli, K. pneumoniae, Enterobacter* spp.	38-52% (*E. coli, K. pneumoniae*); >20% *Enterobacter* spp.	Lost for empiric use in *Enterobacterales*
TMP-SMX (Co-trimoxazole)	*E. coli, K. pneumoniae, S. aureus*	>50% (*E. coli, K. pneumoniae*); >40% (*S. aureus*)	Lost for *Enterobacterales*, unreliable for *S. aureus*
Fluoroquinolones (e.g., ciprofloxacin)	*E. coli, K. pneumoniae, P. aeruginosa, S. aureus*	25-54% (*E. coli, K. pneumoniae, S. aureus*); 15% (*P. aeruginosa*)	Lost for Enterobacterales, unreliable for *S. aureus*
Nitrofurantoin	*K. pneumoniae, Enterobacter* spp.	>50% (*K. pneumoniae, Enterobacter* spp.)	Lost for non-*E. coli Enterobacterales*
Oxacillin (for MRSA)	*S. aureus*	28% (MRSA rate)	Lost for empiric *S. aureus* coverage
Ampicillin/Sulbactam	*E. coli, K. pneumoniae, Enterobacter* spp.	>80%	Lost for *Enterobacterales*
Gentamicin	*E. coli, K. pneumoniae, Enterobacter* spp.	>30%	Unreliable for empiric use
Tetracycline	*E. coli, K. pneumoniae, S. aureus*	>40%	Unreliable for empiric use

*IDSA, 2025 (13)

blood and skin/soft tissue specimens. *Candida* spp. is most notable in vaginal swabs and, to a lesser extent, in sputum and urine, consistent with their role in mucosal and device-associated infections (1-3, 5-6, 8).

Tables 2 and 3 demonstrate a high prevalence of multidrug-resistant MDR, XDR, and PDR isolates among Gram-negative bacteria, with lower but still significant rates among Gram-positive species. Among Gram-negative isolates, more than half are MDR (55%), with notable proportions of XDR (15%) and PDR (2%). This pattern is consistent with global surveillance data, which highlight the predominance of MDR and XDR phenotypes in species such as *A. baumannii*, *K. pneumoniae*, and *P. aeruginosa*, especially in intensive care and hospital settings (14-16).

Gram-positive isolates show lower rates of MDR (30%) and XDR (4%), with no PDR detected. MRSA and VRE were the most common MDR Gram-positive pathogens, contributing substantially to the burden of healthcare-associated infections (17, 18).

The clinical impact of these resistance patterns is profound, leading to increased morbidity, mortality, and healthcare costs. The Infectious Diseases Society of America recommends routine surveillance, standardized susceptibility testing, and stewardship interventions to mitigate the spread and impact of MDR, XDR, and PDR organisms. Molecular characterization of resistance mechanisms, such as carbapenemase production, further informs infection control and therapeutic strategies (5, 17).

Table 4 demonstrates high prevalence with *K. pneumoniae* and *A. baumannii* notable for their elevated XDR and PDR rates, reflecting the global spread of carbapenemase-producing strains and the accumulation of multiple resistance mechanisms. *E. coli* and *K. pneumoniae* are also major contributors to extended-spectrum beta-lactamase (ESBL) and carbapenem resistance. *Pseudomonas aeruginosa*, while less frequently XDR or PDR, remains a critical concern due to its intrinsic resistance mechanisms and capacity for rapid acquisition of additional resistance determinants (5, 15).

The emergence of XDR and PDR isolates presents limited therapeutic options, often necessitating the use of last-resort agents such as colistin and tigecycline. Surveillance data indicate that these resistance patterns are most prevalent in intensive care units, among patients with prior antibiotic exposure, and in regions with high antimicrobial use (17-23).

Furthermore, the predominance of MDR and XDR Gram-negative bacteria, and the ongoing emergence of PDR isolates, represent a major challenge for clinical management and public health. Standardized definitions, routine surveillance, and robust antimicrobial stewardship are essential to address this evolving threat (15-23).

Trends in MDR, XDR, and PDR rates for *E. coli*, *K. pneumoniae*, *P. aeruginosa*, *A. baumannii*, MRSA, and MRSE show persistent high resistance, with notable geographic and specimen-specific variation. Newer β-lactam/β-lactamase inhibitor combinations and last-line agents remain most active against many resistant isolates, but resistance to these is rising in some regions and species. Therefore, resistance rates remain high and are rising for key pathogens, especially in hospital and ICU settings. Newer β-lactam/β-lactamase inhibitor combinations, cefiderocol, and last-line agents (colistin, tigecycline, daptomycin, linezolid) are most active against MDR/XDR isolates, but ongoing surveillance and stewardship are essential to preserve their efficacy (24-26).

Table 7 recommended indications and major target pathogens for the listed antibiotics are as follows, with emphasis on ESBL-producing *Enterobacterales*, multidrug-resistant (MDR) Gram-negative bacteria, MRSA, and VRE, as supported by Infectious Diseases Society of America (IDSA) guidelines and recent reviews: Carbapenems (imipenem, meropenem, ertapenem): Indicated for serious infections caused by ESBL-producing *Enterobacterales* (e.g., *E. coli*, *K. pneumoniae*), AmpC producers, and some MDR Gram-negative bacteria. Meropenem and imipenem are preferred in critically ill patients; ertapenem is suitable for non-critically ill patients and is not active against Pseudomonas or Acinetobacter. Carbapenems are first-line for severe ESBL-E infections, especially outside the urinary tract (8, 27-29).

Traditional empiric agents are now unreliable in many settings. High resistance rates mean that agents such as ampicillin, amoxicillin/clavulanate, cefazolin, cefuroxime, ceftriaxone, ceftazidime, TMP-SMX, fluoroquinolones, and gentamicin may fail to cover the most likely pathogens, especially in severe or healthcare-associated infections (5, 23). Nitrofurantoin resistance is rising in urinary isolates, particularly non-E. coli Enterobacterales. Oxacillin is ineffective for MRSA. Empiric therapy must be guided by local resistance patterns (antibiograms), patient risk factors, and recent microbiology history (23, 28).

Inadequate empiric therapy is associated with increased mortality, longer hospital stays, and worse outcomes. Delays in initiating effective therapy, especially for resistant Gram-negative infections, further increase risk (5, 9, 10). Alternative agents such as aminoglycosides, polymyxins, and tetracyclines carry significant toxicity risks (nephrotoxicity, neurotoxicity, etc.), are require careful monitoring (24, 26).

In summary, high resistance rates demand a shift away from traditional empiric antibiotics toward individualized, evidence-based regimens informed by local resistance data and patient risk factors, with careful stewardship of last-line agents and newer therapies (26, 27).

High resistance rates to commonly used antibiotics among *Enterobacterales*, *Pseudomonas aeruginosa*, and *Staphylococcus aureus* substantially undermine the clinical utility of these agents for empiric therapy, necessitating major changes in empiric prescribing practices and stewardship strategies (24, 26).

Limitations. Study limitations include retrospective design, single-center data, and lack of molecular characterization of resistance mechanisms.

CONCLUSION

This study reveals alarmingly high rates of MDR, XDR, and PDR bacterial pathogens at this hospital, with Gram-negative bacteria presenting a substantially greater resistance challenge than Gram-positive organisms. The loss of clinical utility of commonly prescribed antibiotics necessitates urgent revision of empirical treatment protocols and strengthening of antimicrobial stewardship programs. Carbapenems and aminoglycosides remain cornerstone agents for severe infections, but their judicious use is critical to preserve efficacy. Enhanced infection control measures, continued surveillance, and rational antimicrobial use are essential to combat the escalating AMR crisis in our healthcare setting.

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