Original Article

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Prevalence and Antimicrobial Susceptibility Patterns of Multidrug-resistant Organisms: A Retrospective Observational Study from a Tertiary Care Hospital

Microbiology Section

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ABSTRACT

Introduction: Antimicrobial Resistance (AMR) has emerged as a major threat to effective clinical care, significantly limiting therapeutic options and worsening patient outcomes. Tertiary care hospitals represent high-risk environments for the emergence and dissemination of Multidrug-Resistant Organisms (MDROs), particularly among critically-ill patients exposed to broad-spectrum antimicrobial therapy. The increasing prevalence of multidrug resistance across key hospital pathogens highlights a concerning shift toward difficult-to-treat infections, underscoring the need for institution-specific surveillance data.

Aim: To evaluate the prevalence and antimicrobial susceptibility patterns of MDROs in a tertiary care hospital.

Materials and Methods: This retrospective observational study was conducted from January to June 2025 at the Department of Microbiology, Dr. M.K. Shah Medical College and Research Centre, Ahmedabad, Gujarat, India. A total of 530 non-duplicate bacterial isolates from clinical specimens were included. Isolates were identified using colony morphology, Gram staining, standard biochemical tests, and/or the VITEK 2 Compact system. Antimicrobial Susceptibility Testing (AST) was performed by the Kirby–Bauer disk diffusion technique and/or the VITEK 2 Compact system, with interpretation in accordance with Clinical and Laboratory Standards Institute (CLSI) M100 Ed35 2025 guidelines. Data were analysed using WHONET 2024. Statistical analysis was performed using appropriate statistical methods. Categorical variables were summarised as frequencies and percentages. Associations between categorical variables were assessed using the Chi-square test. A p-value of <0.05 was considered statistically significant.

Results: Of 530 bacterial isolates, 320 (60.4%) were identified as MDROs. The MDRO distribution was dominated by carbapenem-resistant Enterobacterales (CRE) 108 (33.75%), followed by ESBL producers 77 (24.06%) and Carbapenem-Resistant *Acinetobacter Baumannii* (CRAB) 70 (21.88%), with lower proportions of Methicillin-Resistant *Staphylococcus aureus* (MRSA) 40 (12.5%), carbapenem-resistant *Pseudomonas aeruginosa* 24 (7.5%), and VRE 1 (0.31%). MDROs were more frequent among inpatients, 291 (90.94%), particularly in intensive care units 127 (39.7%), and showed higher prevalence in older age groups ≥60 years (53.13%). Wound 101 (31.56%) and respiratory 99 (30.94%) specimens constituted the major sources of isolation.

Conclusion: The present study demonstrates a concerning institutional multidrug resistance landscape that compromises the effectiveness of commonly used antimicrobials. The institution-specific resistance patterns generated in this study support a data-driven recalibration of empirical antimicrobial therapy, while reinforcing the need for sustained antimicrobial stewardship and strengthened infection prevention measures.

Keywords: *Acinetobacter baumannii*, Carbapenem resistance, Drug resistance, Extended-spectrum beta-lactamases

INTRODUCTION

The AMR has emerged as one of the most serious global public health threats of the 21st century. Once considered a looming concern, it now represents an ongoing and accelerating crisis within healthcare systems worldwide. A comprehensive global analysis published in *The Lancet* estimated that bacterial AMR was associated with approximately 4.95 million deaths, including 1.27 million directly attributable deaths, highlighting its substantial clinical and economic burden [1]. MDROs, commonly defined as organisms resistant to at least one agent in three or more antimicrobial classes, significantly complicate the management of infectious diseases by limiting therapeutic options, prolonging hospital stays, increasing healthcare costs, and contributing to higher morbidity and mortality [2].

Beyond the clinical setting, AMR is increasingly recognised as a complex ecological and evolutionary phenomenon, driven not only by antimicrobial misuse but also by environmental reservoirs and interconnected human-animal-environment interfaces. Antibiotic-resistant bacteria and resistance genes have been identified in water bodies, soil, food chains, and hospital effluents, facilitating their persistence and dissemination across ecosystems [3]. The mobilisation of resistance determinants through gene transfer-including plasmids, transposons, and bacteriophages-enables rapid spread across bacterial species, amplifying the scale of the problem and blurring the boundaries between community and hospital-associated infections [3,4].

Among MDROs, a particularly important group comprises the ESKAPE pathogens-*Enterococcus* spp., *Staphylococcus aureus*, *Klebsiella pneumoniae*, *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, and *Enterobacter* spp. These organisms are responsible for a substantial proportion of healthcare-associated infections, including bloodstream infections, pneumonia, surgical site infections, and urinary tract infections. Their remarkable ability to "escape" the effects of antimicrobial agents is mediated through diverse mechanisms such as enzymatic drug inactivation, target site modification, reduced membrane permeability, biofilm formation and active efflux systems [5].

Recent surveillance data highlight the growing magnitude of AMR in clinical settings. In India, data from the (ICMR-AMRSN) demonstrate a substantial burden of multidrug resistance in tertiary care hospitals, with carbapenem resistance reported in approximately 50-70% of

Acinetobacter baumannii, 30-50% of *Klebsiella pneumoniae*, and 30-40% of *Pseudomonas aeruginosa* isolates [6]. The World Health Organisation Global AMR and Use Surveillance System (WHO-GLASS) reports widespread resistance among priority bacterial pathogens across multiple regions [7].

Tertiary care hospitals, by virtue of managing critically ill and immunocompromised patients and their extensive reliance on invasive procedures and empirical broad-spectrum antimicrobial therapy, represent high-risk environments for the emergence and transmission of MDROs. In low- and middle-income countries, this burden is exacerbated by inappropriate antimicrobial use, suboptimal infection control practices, and limited microbiological diagnostic capacity, along with unregulated antibiotic access and weak stewardship frameworks [4]. However, there remains a paucity of granular, context-specific data on MDRO epidemiology at the institutional level, particularly from tertiary care hospitals in Western India, where local resistance patterns may differ from national surveillance trends.

To address this gap, the present study aimed to assess the prevalence and antimicrobial susceptibility patterns of MDROs isolated from various clinical specimens at a tertiary care hospital in Ahmedabad, India.

MATERIALS AND METHODS

This retrospective observational study was conducted at the Department of Microbiology, Dr. MK Shah Medical College and Research Centre, affiliated with Smt. SMS Multispeciality Hospital, Ahmedabad, Gujarat, India. Data were collected retrospectively from laboratory records of clinical specimens processed between January 2025 and June 2025. The collected data were extracted, verified, and analysed between January 2026 and February 2026.

The study protocol was reviewed and approved by the Institutional Ethics Committee. (IEC Approval No. MKSMCRC/IEC/38/25). The IEC waived individual patient consent as this was a retrospective analysis of de-identified laboratory records.

Sample size: As this was a time-bound study, no formal sample size calculation was performed. All eligible bacterial isolates available during the study period and meeting the inclusion criteria were included in the study.

Inclusion criteria: Bacterial isolates recovered from clinical specimens during the study period from patients of all age groups and both genders attending both inpatient and outpatient departments were included in the study.

Exclusion criteria: Repeat isolates from the same patient and specimen type within a 14-day interval, isolates with incomplete laboratory records or inconsistent AST results, and environmental samples were excluded from the study.

Culture and AST data of clinical specimens including blood, urine, wound samples, endotracheal aspirates, sputum, and body fluids such as cerebrospinal, pleural, ascitic, and synovial fluids received in the Microbiology Laboratory were analysed. Demographic details, department, specimen type, organism isolated, and AMR patterns, including Multidrug-Resistant (MDR) profiles were evaluated.

Microbiological Processing

Clinical specimens were cultured on appropriate media, including Nutrient agar, MacConkey agar, Blood agar and Chocolate agar, and incubated aerobically at 35-37°C for 24-48 hours. Blood cultures were processed using the BACTEC FX40 automated system and subcultured upon positivity. Isolates were identified based on colony morphology, Gram staining, standard biochemical tests [8], and/or VITEK 2 Compact automated identification system (bioMérieux) using ID-GN cards for Gram-negative bacilli and ID-GP cards for Gram-positive cocci.

Antimicrobial Susceptibility Testing (AST)

The AST was performed using the Kirby-Bauer disk diffusion method on Mueller-Hinton agar and incubated aerobically at 35-37°C for 18-24 hours and/or the VITEK 2 Compact system. Interpretation of zone diameters, Minimum Inhibitory Concentration (MIC) values and Colistin broth microdilution results was done according to CLSI M100 Ed35 2025 guidelines [9]. Tigecycline susceptibility was interpreted using the FDA 2025 interpretive criteria for Enterobacterales [10], EUCAST Breakpoint Tables v15.0 (2025) for *Staphylococcus aureus* [11], and published interpretive criteria for *Acinetobacter baumannii* [12], for which CLSI M100, 35th edition (2025) breakpoints are unavailable. Quality control was ensured using standard reference strains *Escherichia coli* ATCC 25922, *Staphylococcus aureus* ATCC 25923, and *Pseudomonas aeruginosa* ATCC 27853.

Detection of Multidrug Resistance

MDR was defined as non-susceptibility to at least one agent in three or more antimicrobial classes according to the criteria proposed by Magiorakos AP et al., Carbapenem resistance among Enterobacterales, *Acinetobacter baumannii*, and *Pseudomonas aeruginosa* was defined as resistance or intermediate susceptibility to at least one carbapenem based on CLSI M100, 35th Edition (2025) interpretive criteria [2,9]. ESBL detection was confined to validated Enterobacterales, primarily to *Escherichia coli, Klebsiella pneumoniae, Klebsiella oxytoca,* and *Proteus mirabilis*. ESBL production was screened using third-generation cephalosporins and confirmed by combination disk diffusion and double-disk synergy testing using ceftazidime and ceftazidime-clavulanate discs. An increase of ≥5 mm in zone diameter around the ceftazidime-clavulanate disc compared with ceftazidime alone was interpreted as ESBL production. Methicillin resistance in *Staphylococcus aureus* was detected using cefoxitin disk diffusion and/or oxacillin MIC. Inducible clindamycin resistance was detected by D-test, and vancomycin resistance in *Enterococcus* species was determined by MIC testing and interpreted as susceptible (≤4 µg/mL), intermediate (8-16 µg/mL), or resistant (≥32 µg/mL) [9].

STATISTICAL ANALYSIS

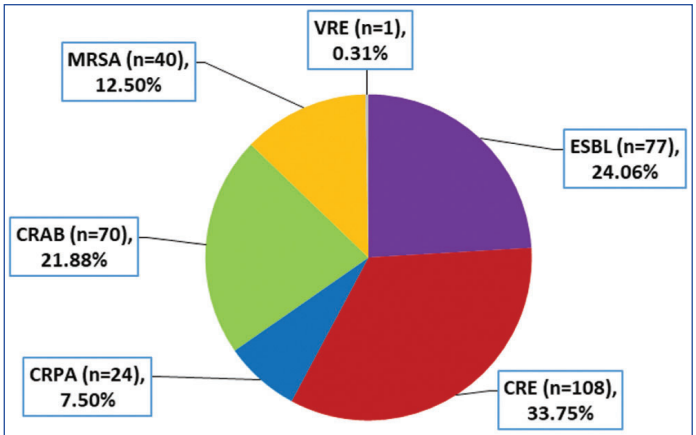
Data were analysed using WHONET 2024 software. Categorical variables were expressed as frequencies and percentages. Associations between categorical variables were assessed using the Chi-square test, with a p-value <0.05 considered statistically significant.

RESULTS

A total of 1,510 clinical samples were processed, of which 530 (35.10%) yielded significant bacterial growth, with Gram-negative bacilli accounting for 400 (75.5%) and Gram-positive cocci, 130 (24.5%) of isolates. Of the total isolates, 320 (60.4%) were identified as MDROs, and their overall distribution is depicted in [Table/Fig-1], while their distribution among total bacterial isolates is detailed in [Table/Fig-2].

Among the 320 MDROs, the highest proportion was observed among patients aged ≥60 years 170 (53.13%), followed by those aged 36-59 years 71 (22.19%), while young adults, adolescents, children, and infants accounted for 40 (12.50%), 15 (4.69%), 15 (4.69%), and 9 (2.81%) of cases, respectively. In contrast, among 40 MRSA isolates, the proportion was higher in middle-aged adults 36-59 years; 11 (27.5%) compared to the elderly ≥60 years; 8 (20.0%). A male predominance was noted, with males accounting for 187 (58.44%) and females 133 (41.56%) of all MDRO isolates.

A significantly higher proportion of MDROs were identified among inpatients, 291 (90.94%), compared to outpatients, 29 (9.06%). The ICU accounted for the largest share of MDRO isolates, 127 (39.69%), with a statistically significant association between ICU stay and MDRO isolation (Chi-square test χ^2=46.50, p-value <0.001).



[Table/Fig-1]: Distribution pattern of Multidrug-Resistant Organisms (MDROs) (n=320).
MDROs: Multidrug-resistant organisms; ESBL: Extended-spectrum beta-lactamase; CRE: Carbapenem-resistant Enterobacterales; CRPA: Carbapenem-resistant *Pseudomonas aeruginosa*; CRAB: Carbapenem-resistant *Acinetobacter baumannii*; MRSA: Methicillin-resistant *Staphylococcus aureus*; VRE: Vancomycin-resistant Enterococci.

Bacterial isolates	MDRO type	n (%) of MDROs among total isolates
Enterobacteriaceae family (n=248)	ESBL (n=77)	31.05
Enterobacteriaceae family (n=248)	CRE (n=108)	43.55
Pseudomonas aeruginosa (n=60)	CRPA (n=24)	40
Acinetobacter baumannii (n=77)	CRAB (n=70)	90.90
Staphylococcus aureus (n=59)	MRSA (n=40)	67.80
Enterococcus species (n=18)	VRE (n=1)	5.56

[Table/Fig-2]: Distribution of Multidrug-Resistant Organisms (MDROs) among total isolates (n=530).
MDROs: Multidrug-resistant organisms; ESBL: Extended-spectrum beta-lactamase; CRE: Carbapenem-resistant enterobacterales; CRPA: Carbapenem-resistant *Pseudomonas aeruginosa*; CRAB: Carbapenem-resistant *Acinetobacter baumannii*; MRSA: Methicillin-resistant *staphylococcus aureus*; VRE: Vancomycin-resistant enterococci.
*For ESBL analysis, CLSI-approved Enterobacterales (*E. coli, Klebsiella pneumoniae, Klebsiella oxytoca*, and *Proteus mirabilis*) were included

Detailed distribution across clinical departments and specimen types is provided in [Table/Fig-3,4].

Antimicrobial susceptibility patterns of major MDRO groups are summarised in [Table/Fig-5-7].

Colistin exhibited preserved in-vitro activity against Gram-negative MDROs, with all isolates categorised as intermediate. Inducible clindamycin resistance was observed in 23.73% (n=14) of *Staphylococcus aureus* isolates.

DISCUSSION

The era of antibiotics as "magic bullets" has steadily declined with the rapid emergence and global dissemination of AMR, now a major threat to modern healthcare. Resistance is driven by horizontal transfer of genetic determinants under sustained antimicrobial pressure enabling rapid amplification within clinical settings [4]. In India, this challenge is further intensified by high antibiotic consumption, fixed-dose combinations, over-the-counter access, self-medication and inadequate infection control practices, creating a high-selection environment that accelerates multidrug resistance and its progression to advanced resistance phenotypes [13].

The present study found a MDRO prevalence of 60.4% (320 of 530 isolates), consistent with the 30-60% range reported in Indian tertiary care settings [14,15]. However, higher rates such as 81.9% reported by Borde AN et al., have also been documented [16]. Globally, MDRO prevalence varies widely, from 26.7% in community populations to 37.3% in hospital cohorts, with pooled estimates near 59%, particularly in low- and middle-income countries [17-19]. This high burden likely reflects tertiary referral bias, ICU density, and sustained antimicrobial exposure, contributing to a hospital-associated resistance landscape.

MDROs in this study were predominantly driven by β-lactam resistance, with CRE accounting for 43.55% and Extended-Spectrum β-Lactamase (ESBL) producers 31.05% of Enterobacterales isolates, indicating a shift toward higher-order β-lactam resistance.

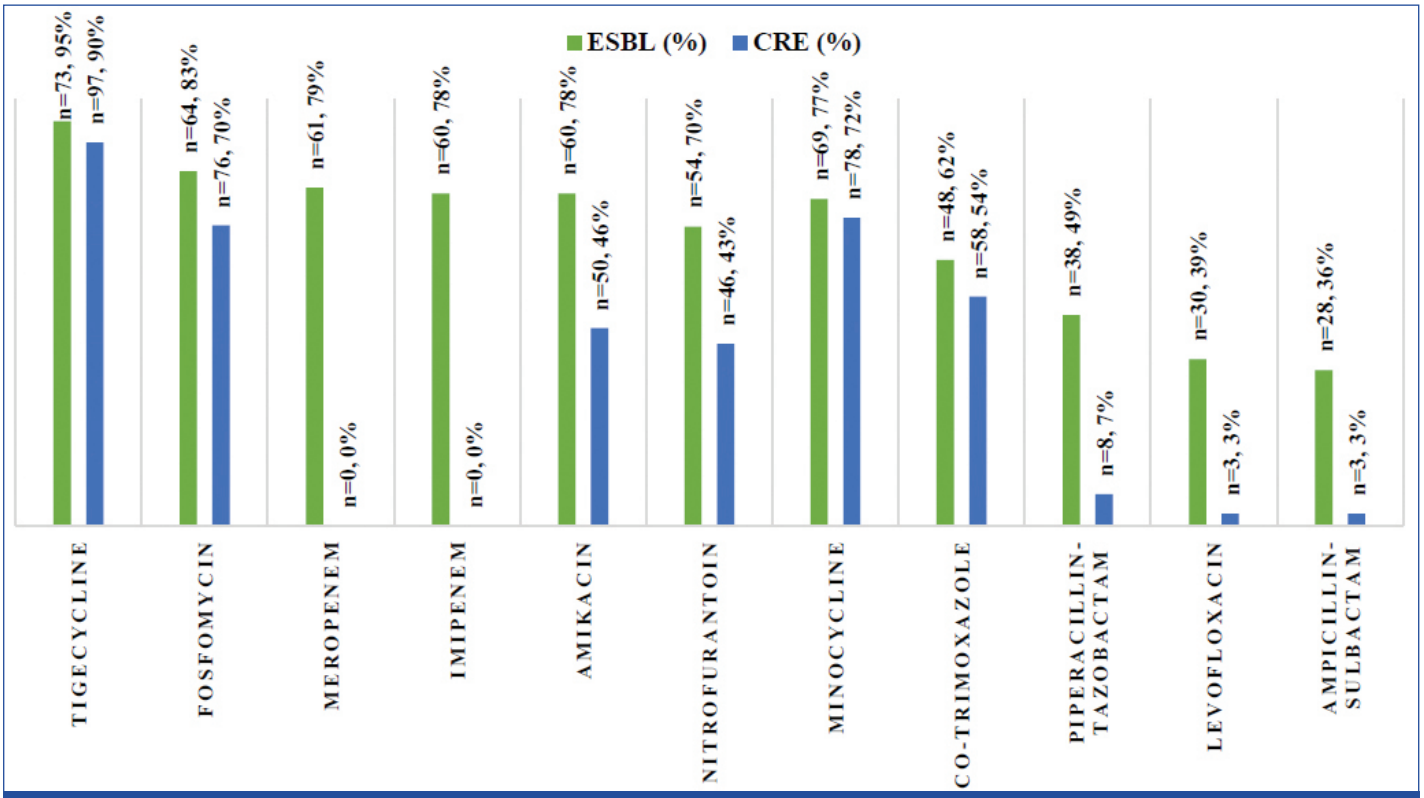
MDRO	Medicine (n=26)	Surgery (n=52)	Ortho (n=41)	OBG (n=3)	EMR (n=21)	ICU (n=127)	PICU (n=9)	NICU (n=12)	OPD (n=29)
	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)
ESBL (n=77)	10 (38.46)	19 (36.54)	6 (14.63)	1 (33.33)	8 (38.10)	15 (11.81)	4 (44.44)	4 (33.33)	10 (34.48)
CRE (n=108)	7 (26.93)	19 (36.54)	12 (29.27)	1 (33.33)	8 (38.10)	48 (37.80)	1 (11.12)	7 (58.34)	5 (17.24)
CRPA (n=24)	4 (15.38)	2 (3.84)	2 (4.88)	0	3 (14.28)	6 (4.72)	0	0	7 (24.14)
CRAB (n=70)	1 (3.85)	6 (11.54)	3 (7.32)	0	1 (4.76)	56 (44.09)	0	1 (8.33)	2 (6.90)
MRSA (n=40)	4 (15.38)	6 (11.54)	17 (41.46)	1 (33.33)	1 (4.76)	2 (1.58)	4 (44.44)	0	5 (17.24)
VRE (n=1)	0	0	1 (2.44)	0	0	0	0	0	0
TOTAL (n=320)	26 (8.13)	52 (16.25)	41 (12.81)	3 (0.94)	21 (6.56)	127 (39.69)	9 (2.81)	12 (3.75)	29 (9.06)

[Table/Fig-3]: Department-wise distribution of MDROs (n=320).
MDROs: Multidrug-resistant organisms; ESBL: Extended-spectrum beta-lactamase; CRE: Carbapenem-resistant enterobacterales; CRPA: Carbapenem-resistant *Pseudomonas aeruginosa*; CRAB: Carbapenem-resistant *Acinetobacter baumannii*; MRSA: Methicillin-resistant *Staphylococcus aureus*; VRE: Vancomycin-resistant enterococci; ORTHO: Orthopaedics department; OBG: Obstetrics and gynaecology department; EMR: Emergency medicine department; ICU: Intensive care unit; PICU: Paediatric intensive care unit; NICU: Neonatal intensive care unit; OPD: Outpatient department

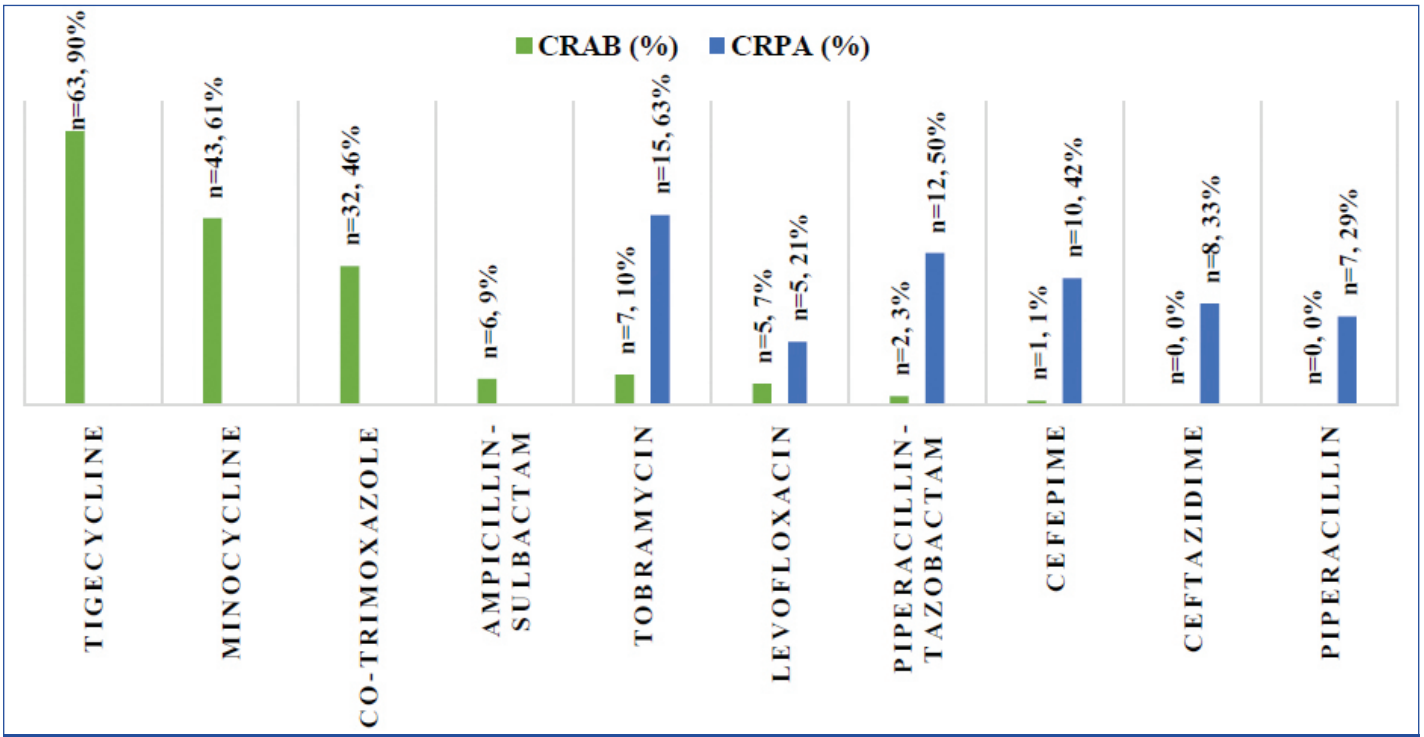
MDROs	Setting	Swab/tissue/pus (n=101)	Body fluids (n=12)	Respiratory samples (n=99)	Urine (n=75)	Blood (n=29)	Catheter tip (n=4)
		n (%)	n (%)	n (%)	n (%)	n (%)	n (%)
ESBL (n=77)	OPD	2 (1.98)	0	0	8 (10.67)	0	0
	IPD	23 (22.78)	2 (16.67)	13 (13.13)	26 (34.67)	3 (10.34)	0
CRE (n=108)	OPD	2 (1.98)	0	2 (2.02)	1 (1.33)	0	0
	IPD	28 (27.72)	6 (50.00)	29 (29.29)	29 (38.67)	8 (27.59)	3 (75)
CRPA (n=24)	OPD	2 (1.98)	1 (8.33)	1 (1.01)	3 (4.00)	0	0
	IPD	5 (4.95)	1 (8.33)	2 (2.02)	7 (9.33)	2 (6.90)	0
CRAB (n=70)	OPD	1 (0.99)	0	1 (1.01)	0	0	0
	IPD	6 (5.94)	2 (16.67)	51 (51.52)	0	8 (27.59)	1 (25)
MRSA (n=40)	OPD	4 (3.96)	0	0	0	1 (3.44)	0
	IPD	27 (26.73)	0	0	1 (1.33)	7 (24.14)	0

VRE (n=1)	OPD	0	0	0	0	0	0
	IPD	1 (0.99)	0	0	0	0	0
Total (n=320)		101 (31.56)	12 (3.75)	99 (30.94)	75 (23.44)	29 (9.06)	4 (1.25)

[Table/Fig-4]: Specimen-wise distribution of MDROs (n=320).
MDROs: Multidrug-resistant organisms; ESBL: Extended-spectrum beta-lactamase; CRE: Carbapenem-resistant enterobacterales; CRPA: Carbapenem-resistant *Pseudomonas aeruginosa*; CRAB: Carbapenem-resistant *Acinetobacter baumannii*; MRSA: Methicillin-resistant *Staphylococcus aureus*; VRE: Vancomycin-resistant enterococci; IPD: Inpatient department; OPD: Outpatient department

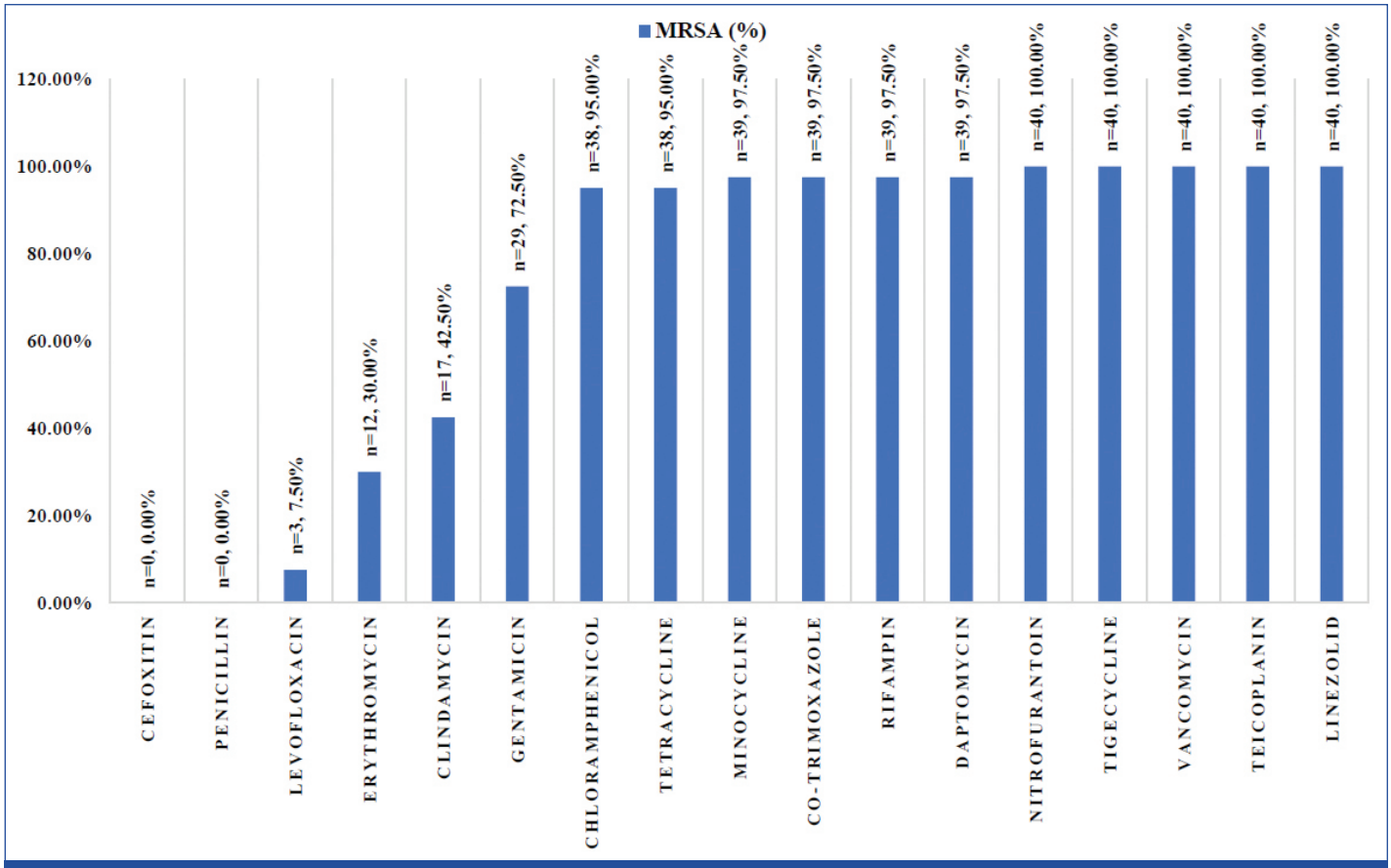


[Table/Fig-5]: Antimicrobial susceptibility profiles of ESBL (n=77) and CRE (n=108) isolates.
ESBL: Extended-spectrum beta-lactamase; CRE: Carbapenem-resistant Enterobacterales
*Nitrofurantoin and fosfomycin susceptibility percentages are calculated from urinary isolates within each MDRO group
†Fosfomycin results are reported for *Escherichia coli* only



[Table/Fig-6]: Antimicrobial susceptibility profiles of CRAB (n=70) and CRPA (n=24) isolates.
CRAB: Carbapenem-resistant *Acinetobacter baumannii*; CRPA: Carbapenem-resistant *Pseudomonas aeruginosa*

Reported CRE prevalence ranges from 5-59%, and ESBL from 19-60%, reflecting carbapenem resistance dominance [14,15]. ESBL predominated in urinary isolates (45.34%), aligning with Indian UTI data showing variability (16.6–69.2%) [20]. Urinary CRE prevalence (40%) matched higher tertiary care estimates, including Perween N et al., (34.5%), exceeding lower rates in other studies [20,21]. ESBL-producing uropathogens retained susceptibility to nitrofurantoin (70.5%), supporting its role as first-



[Table/Fig-7]: Antimicrobial Susceptibility profiles of MRSA (n=40).
(MRSA – Methicillin-resistant *Staphylococcus aureus*)

line oral therapy for uncomplicated UTIs, particularly in outpatient settings. Fosfomycin showed sustained activity (82.68% ESBL, 70.62% CRE), comparable to Indian cohorts, particularly against *Escherichia coli* [21]. Carbapenems retained high susceptibility in ESBL isolates and remain appropriate for severe infections, particularly when β-lactam/β-lactamase inhibitor combinations are ineffective, preferably administered as prolonged infusions to optimise pharmacodynamics.

Carbapenem-resistant *Acinetobacter baumannii* (CRAB) accounted for 21.88% of MDROs, with a high carbapenem resistance rate (90.9%) within its subgroup, concentrated predominantly in ICU respiratory samples, confirming its role as a critical nosocomial pathogen. Comparable studies report MDR *Acinetobacter* prevalence of 29-74% and carbapenem resistance of 50-90% in Asia [14,15]. CDC estimates attribute approximately 8,500 infections and 700 deaths annually to CRAB, underscoring its clinical impact [22]. This burden reflects invasive ventilation, prolonged antimicrobial exposure, and environmental resilience. CRAB susceptibility was markedly restricted, with uniformly low activity across β-lactams, fluoroquinolones, and aminoglycosides (≤10%), effectively limiting therapeutic options to tetracycline derivatives, consistent with recent reports [23]. Infectious Diseases Society of America (IDSA) 2024 guidelines emphasise minocycline's preferential use in pulmonary infections due to favourable serum and epithelial lining fluid penetration [24]. Tigecycline, despite in-vitro activity, is limited by pharmacokinetic constraints and requires higher dosing in severe infections; it is not favoured as monotherapy in bloodstream infections due to suboptimal serum levels. Polymyxins, including colistin, retain activity but remain reserve agents due to toxicity and pharmacokinetic limitations. Standard practice involves a loading dose with renal-adjusted maintenance; adjunct inhalational (nebulised) colistin therapy is considered in pulmonary infections [25].

Carbapenem-Resistant *Pseudomonas aeruginosa* (CRPA) represented 7.5% of MDROs, with 40% carbapenem resistance. MDR *P. aeruginosa* prevalence in similar studies ranges from 9% to 19.5%, indicating comparatively higher resistance in the present cohort, though from a smaller isolate base [14,15]. Such variation likely reflects differences in geographic setting, patient population, methodology, and antimicrobial practices. These factors influence the local epidemiology of AMR and may account for the observed differences between the studies. Overall, *P. aeruginosa* exhibited variable susceptibility to key antipseudomonal agents.

Gram-positive resistance accounted for a smaller proportion, with MRSA comprising 12.5% of MDROs and 67.8% of *S. aureus* isolates. Published Indian and global studies report MRSA prevalence of 15-55%, placing these findings toward the higher end [15,17,26]. Resistance is mediated by the *mecA* gene encoding PBP2a, conferring β-lactam resistance. MRSA's persistence on surfaces and colonisation of skin and devices facilitates transmission, reflected here by clustering in device- and procedure-associated infections, especially in orthopaedics and surgical units. Vancomycin-Resistant Enterococci (VRE) were rare, consistent with lower glycopeptide resistance prevalence [15,17]. In skin and soft tissue infections, the retained susceptibility of MRSA to tetracyclines and co-trimoxazole supports their use as glycopeptide-sparing options in selected cases. Consistent with comparable studies, MRSA demonstrated preserved susceptibility to glycopeptides and oxazolidinones, reinforcing the reliability of vancomycin, teicoplanin, and linezolid. Vancomycin remains preferred for invasive infections such as bacteraemia, whereas linezolid offers an advantage in pulmonary and complex tissue infections owing to superior tissue penetration [25].

The disproportionate concentration of MDROs in inpatient settings, contrasted with lower resistance profiles among outpatient isolates, underscores the need for a stratified empirical treatment approach. Community-acquired infections, which generally exhibit lower AMR profiles, may be effectively managed with narrower-spectrum agents, while broader-spectrum coverage should be reserved for inpatients and high-risk cases. Given the potential for resistance development during therapy, particularly with *Pseudomonas aeruginosa* and *Staphylococcus*, early reassessment through repeat cultures within 3-4 days is advisable [25]. This strategy must be reinforced by

rigorous infection prevention and control measures-including strict hand hygiene, contact precautions, patient cohorting or isolation, and prompt source control-to mitigate transmission. Concurrently, robust antimicrobial stewardship interventions, such as stewardship-driven escalation and de-escalation, formulary restrictions, prospective audits with feedback, and antibiogram-guided policy updates, are essential to optimise therapeutic outcomes and curb the emergence of further resistance.

Limitation(s)

This study had certain limitations. Being a retrospective record-based analysis, it was dependent on the accuracy and completeness of laboratory data, with limited scope for clinical correlation. Molecular characterisation of resistance mechanisms was not performed; therefore, specific genetic determinants of AMR could not be identified. The study was conducted at a single tertiary care centre, which may limit the generalisability of the findings to other settings. Additionally, patient-level clinical outcomes were not evaluated, restricting assessment of the clinical impact of multidrug-resistant infections.

CONCLUSION(S)

This study reveals a substantial burden of multidrug resistance primarily driven by carbapenem-resistant *Acinetobacter baumannii*, CREs, and MRSA, which compromises the effectiveness of commonly used first-line agents like β-lactams and fluoroquinolones. Nonetheless, viable therapeutic options persist, including nitrofurantoin and fosfomycin for resistant Enterobacterales UTIs, minocycline and tigecycline for *Acinetobacter* infections, while colistin remains a reserve agent in critical illness and glycopeptides and oxazolidinones for invasive MRSA cases. The selective use of tetracyclines and co-trimoxazole in skin and soft tissue infections further supports targeted, resource-conscious treatment. These results emphasise the necessity of adopting data-driven, unit-specific, and evidence-based antimicrobial strategies that consider clinical context, infection severity, and local resistance patterns. Individualised therapy should incorporate pharmacokinetic-pharmacodynamic optimisation, toxicity considerations, and intrinsic resistance factors. Such a comprehensive, tailored approach is critical to improving patient outcomes and preserving the efficacy of existing antimicrobials in the face of escalating resistance.

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Authors' contribution: CM: Data collection, data analysis, manuscript preparation, editing and review; PK: Data collection, data analysis, manuscript preparation, editing and review; KP: Data analysis and manuscript review; RS: Conceptualisation, supervision, critical revision of the manuscript, and final approval of the manuscript; MV: Senior guidance and support.

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