



PHENOTYPIC ANTIMICROBIAL RESISTANCE PATTERNS AND RESISTANCE GENE DISTRIBUTION ACROSS CLINICAL SPECIMENS: ASSOCIATIONS WITH PATIENT CHARACTERISTICS AND CLINICAL OUTCOMES

Prof. Ayodele Oluwaseun Ajayi^{1*}, Prof. Dwij Raj Bhatta², Ayush Kumar³, Dr. Inshad Ali Khan⁴

¹Department of Microbiology, Federal University Oye-Ekiti, Ekiti State, Nigeria, Email: ayodele.ajayi@fuoye.edu.ng

²Central Department of Microbiology, Tribhuvan University, Kathmandu, Nepal

³Department of Medical Microbiology and Infectious Diseases, University of Manitoba, Winnipeg, Manitoba, Canada

⁴Department of Microbiology, Central University of Rajasthan, Ajmer, Rajasthan, India

***Corresponding Author:** Prof. Ayodele Oluwaseun Ajayi, ayodele.ajayi@fuoye.edu.ng

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ABSTRACT

Antimicrobial resistance represents an increasing clinical and microbiological challenge, requiring integrated assessment of phenotypic susceptibility, resistance genes, and patient-related outcomes. This study evaluated phenotypic antimicrobial resistance patterns and resistance-gene distribution across clinical specimens and examined their associations with demographic characteristics and clinical outcomes using secondary data from 2,200 records. Susceptibility to amoxicillin, ciprofloxacin, meropenem, vancomycin, and colistin was classified as sensitive, intermediate, or resistant, while resistance burden was defined as the number of antibiotics classified as resistant. Descriptive statistics, Pearson chi-square tests, Cramer's V, Kruskal-Wallis testing, and multivariable binary logistic regression were applied. Meropenem showed the highest resistance proportion (34.3%), whereas colistin showed the lowest (31.9%). VIM was the most frequently recorded resistance gene (21.0%), followed by OXA-48 (20.5%). Resistance-gene distribution was not significantly associated with specimen type, and resistance burden did not differ significantly across recovered, ICU, and deceased groups. Multivariable analysis identified no significant independent predictors of adverse clinical outcome. The findings support integrated phenotypic, molecular, and clinical surveillance for antimicrobial resistance assessment.

Keyword:Antimicrobial resistance; phenotypic susceptibility; resistance genes; clinical specimens; clinical outcomes

Introduction

Bacterial populations have the capacity to develop and carry antimicrobial-resistance mechanisms, which has created antimicrobial resistance (AMR) as a serious biological and clinical problem and challenged to develop effective therapies for these organisms. The problem is especially critical in clinical applications, where treatment is often complicated by the presence of resistant infections, and where prolonged hospitalisation and adverse outcomes result. International surveillance of CRPA has identified significant geographical differences in the mechanism(s) for resistance and the clinical impact of these, highlighting the importance of continued surveillance at the phenotypic and molecular levels (Reyes et al., 2023). Likewise, studies combining resistance genotype, phenotype, and clinical outcomes have found laboratory resistance profile can be used as an important tool for clinical behaviour of Gram-negative infections (Koch et al., 2025).

There are several mechanisms involved in the biological basis of AMR, which include the enzymatic degradation of antibiotics, modification of the target enzyme, alteration in membrane permeability, active efflux, horizontal gene transfer, and the acquisition of mutations associated with AMR (Nusrat et al., 2025). Genes for resistance can spread among different types of microbes in different body locations. For instance, the oral microbiota has been recognized as a source of antimicrobial resistance genes and phenotypically resistant organisms (Anderson et al., 2023). At the strain level, highly resistant bacterial clones can also carry adaptive traits that allow for persistence and dissemination, such as with extensively drug-resistant *Klebsiella pneumoniae* lineages (Heiden et al., 2020).

In addition to genotypic resistance, phenotypic resistance is also clinically important, as it can be directly correlated with the definition of an organism as sensitive, intermediate, or resistant to any specific antimicrobial agent. Previous clinical investigations related to *S. aureus* have revealed significant variation in antibiotic susceptibility which includes clinically important methicillin resistant phenotypes (Gurung et al., 2020a). Similar diversity was reported for multidrug-resistant (MDR) strains of *Elizabethkingia*, which harbored resistance traits that were associated with specific genomic characteristics (Wu et al., 2024). There has also been a significant difference in resistance patterns among bacterial pathogens recovered from clinical samples in broad surveillance of hospitals (Handa et al. 2024).

Observed resistance patterns can also vary depending on the bacterial type isolated from the specimen, as the population of bacteria obtained from blood, urine, respiratory samples, wound or other clinical specimens may be different in terms of exposure history and the selection pressure they have been exposed to. The resistance of *Pseudomonas aeruginosa* and *Acinetobacter baumannii* to multiple drugs has been documented at high frequencies in all clinical specimen types (Mirzaei et al., 2020). The national surveillance data have demonstrated that there can be significant differences in AMR between organisms, antimicrobial classes and healthcare settings at a wider level (Bilal et al., 2021). Resistance patterns for Enterobacteriaceae are also becoming more common, demonstrating the need for a deeper

understanding of resistance patterns, in addition to the specimen and clinical characteristics (Taha et al., 2023).

Genotypic assessment is complementary to phenotypic susceptibility testing as resistance genes can identify molecular mechanisms of observed responses to antimicrobials. However, the genotype to phenotype relation is not always complete and the reliability of molecular markers varies depending on the resistance mechanism, organism, and the database utilized to interpret marker data (Mahfouz et al., 2020). Other studies, including large international studies on carbapenem-resistant *Klebsiella pneumoniae*, have also shown that the characteristics of the bacteria and their resistance profile can differ between geographic areas and patient populations (Wang et al., 2022). Persistent antimicrobial resistance like that in TB is another clinical example of the continued importance of knowing that despite good surveillance and advances in treatment (Farhat et al., 2024).

The present study thus sought to describe the patterns of phenotypic AMR and the distribution of antimicrobial resistance genes in clinical specimens and to explore the relationship between these patterns and clinical characteristics and outcomes. Demographic data, specimen type, antimicrobial susceptibility, categories of antimicrobial resistance genes, testing methods, presence of antimicrobial resistance load and clinical outcome were all included within the scope. The specific aims were to characterize the antimicrobial susceptibility results, examine the distribution of resistance genes by specimen type, to measure the cumulative burden of phenotypic resistance, to assess the relationship of this burden with clinical outcome, and to determine if demographic, specimen, genetic and resistance factors were independently associated with adverse clinical outcomes.

2. Materials and Methods

2.1 Study Design and Data Source

The study used a quantitative, retrospective and secondary data analysis approach to investigate the phenotypic antimicrobial resistance pattern, the distribution of resistance genes and the possible associations with demographic features, clinical specimens type and clinical outcome. The analysis was performed on an openly accessible structured dataset that included information on antimicrobial susceptibility and resistance from clinical records.

Analytical data set had 2,200 records. The identifier field was not unique for all observations, so each row was analyzed independently as an analytical record as opposed to each row being unique as a patient. The variable 'Identifier' was therefore only used for record identification and was not used for any descriptive or inferential analysis (Siam & Faiza, 2025).

Analytical variables evaluated included age, gender, specimen type, antimicrobial susceptibility to amoxicillin, ciprofloxacin, meropenem, vancomycin, and colistin, resistance-gene category, and clinical outcome; and antimicrobial susceptibility testing method. For the main analyses, all 2200 observations

were kept, since the required variables for demographic, susceptibility, specimen-type, resistance-burden and outcome analyses were available.

2.2 Variable Definition and Operationalization

Age was used as a continuous variable, years; the mean, standard deviation, median and range were used to summarise age. Gender was dichotomized as female or male.

The type of clinical specimen was classified into five mutually exclusive categories: blood, sputum, stool, urine and wound swab. Antimicrobial susceptibility testing method was grouped into disc diffusion, minimum inhibitory concentration (MIC) and an automated system.

Antimicrobial susceptibility was determined for each of five antimicrobial agents: amoxicillin, ciprofloxacin, meropenem, vancomycin and colistin. Susceptibility was categorized as Sensitive, Intermediate, or Resistant, based on the classifications in the source data for each antimicrobial. Denominators for each antimicrobial-specific percentage were the full 2200 antimicrobial records available as susceptibility data was available for all five antimicrobial variables.

A derived phenotypic resistance-burden variable was determined for each record using the five antibiotics considered in the analysis and the number of antimicrobial agents categorized as Resistant. The range of resistance burden was therefore from 0 (no phenotypic resistance) to 5 (resistance to all five antimicrobials). Resistant results were not given for intermediate. Resistance burden was measured as a categorical variable with 6 levels (0, 1, 2, 3, 4, or 5 resistant antibiotics) and as a numeric count variable, as appropriate.

Four categories of resistance genes were recorded in the resistance-gene variable, KPC, NDM-1, OXA-48 and VIM. Records with no known resistance gene were not excluded nor were they statistically imputed but were retained as a separate analytical category labelled 'None recorded'.

There were originally three categories of clinical outcome: Recovered, ICU, Deceased. These three categories were left for descriptive comparisons, and for the analysis of resistance burden by outcome. If multivariable logistic regression was performed, the outcome was also dichotomized (recovered = 0, adverse clinical outcome = 1, which included records categorized as ICU or Deceased).

2.3 Descriptive and Bivariate Statistical Analysis

To describe the analytical sample, descriptive statistics were used. Mean $\pm$ standard deviation was used to summarize continuous age data, while median, minimum, and maximum values were used. Categorical variables were described in the form of frequency and percentages. The percentages for each gender, specimen type, testing method, clinical outcome, antimicrobial susceptibility, and distribution of resistance genes were obtained by dividing the number of specimens with available records by the total number in each category.

The results of the antimicrobial susceptibility patterns were broken down by antimicrobial agent and reported as the number and percentage of Sensitive, Intermediate and Resistant observations. The frequencies of resistance-burden were then determined for scores 0-5.

Distributions of resistance genes were cross-tabulated by specimen type. Percentages within specimens were computed based on the number of observations in the specimen category. To determine whether there was an association between specimen type and resistance-gene category, the Pearson chi-square test of independence was used. Both variables were more than dichotomous and so Cramer's V was computed as the measure of association strength for both variables.

Additionally, Pearson chi-square test was used to test for differences in distribution of the six-level resistance-burden variable between the three clinical outcomes (Recovered, ICU, Deceased), and Cramer's V was used to measure effect size.

Given the bounded nature of the resistance burden (0-5 values) and the absence of normality assumption, differences among the three original clinical-outcome groups were also assessed using the Kruskal-Wallis H test. Descriptive statistics (mean, standard deviation, and median) were used to summarize the resistance burden within each outcome category. All hypothesis tests were two-sided, and a p-value < 0.05 were considered statistically significant.

2.4 Multivariable Logistic Regression Analysis

Model fit of a multivariable binary logistic regression model was used for evaluation of independent association between demographic, clinical, specimen related, genetic and clinical-phenotypic resistance characteristics and adverse clinical outcome. The dependent variable was the dichotomized clinical outcome, which was 0 = Recovered and 1 = adverse clinical outcome, which included both ICU and Deceased records.

The independent variables included in the model at the same time were: age (years, continuous); specimen type (blood as the reference category); resistance-gene category (KPC as the reference category); and phenotypic resistance burden (that is, the number of antimicrobials for which the result was resistant – continuous variable, 0 to 5).

As a result, comparisons of male versus female gender, sputum versus stool, urine versus wound swab, and NDM-1, OXA-48, VIM versus None recorded versus KPC were estimated. The adjusted odds ratio was the odds ratio for each year increase in age. The adjusted odds ratio was the change in the odds for each additional antimicrobial that was classified as resistant for resistance burden.

The regression coefficients were exponentiated and presented as adjusted odds ratios (aORs) and 95% confidence intervals (CIs) and associated p-values. The overall significance of the model was evaluated by the likelihood-ratio test, and the capacity of the model to explain the data in terms of pseudo-R² was summarized. The significance of regression parameters and the global model were determined at the two-

sided 0.05 level.

2.5 Missing-Data Management and Ethical Considerations

Prior to the analysis of the statistics, missingness was assessed for all variables. There were no missing observations for age, gender, specimen type, all 5 antimicrobial susceptibility variables, for testing method or clinical outcome, so the analytical denominator $N = 2200$ was used for these analyses.

A total of 437 records in the resistance-gene field were not associated with a gene result, or 19.9% of the analytical sample. Since there was no recorded resistance gene, this was a clinically interpretable category in the data provided and was specifically included as a category in the Results rather than deleted or imputed. Therefore there was no change in the denominator of 2200 records in the analyses related to resistance gene status.

There was one missing value in the record-identifier variable and repeated values in it, but it was not used as an analytical predictor or outcome, a group variable, or a denominator. Hence, there was no exclusion of any record from the statistical analyses because of the missingness and duplication of data. There were no statistically imputed numerical or categorical values.

This study relied on secondary data and did not directly recruit participants, involve intervention, collect biological samples, nor interact with participants. Analysis was limited to variables found in the secondary data set and no effort was made to identify individual participants. Data were only used for scientific analysis of antimicrobial-resistance patterns and associated clinical characteristics.

3. Results

3.1 Characteristics of the Analytical Dataset

The secondary dataset consisted of 2,200 antimicrobial susceptibility data. The mean age was 45.65 ± 26.27 years, the median age 45 years and the range 1-90 years. The female records were 50.5% of the data and the males 49.5%. The distribution of clinical specimens was fairly uniform, with blood being the highest (20.4%) followed closely by sputum (20.4%) and wound swab (20.4%), urine (19.7%) and stool (19.2%). The most common antimicrobial susceptibility testing methods reported were disc diffusion (34.3%) and automated-system testing (34.2%), with MIC testing at a similar rate. There were 775 records (35.2%) of admissions to an ICU, 739 of recovery (33.6%) and 686 of deaths (31.2%). Table 1 shows summary data on the principal demographic, specimen, laboratory and outcome characteristics.

Table 1. Demographic, specimen, testing, and clinical characteristics of the dataset ($N = 2,200$)

Characteristic	Category/statistic	n (%) or value
Age, years	Mean $\pm$ SD	45.65 $\pm$ 26.27
	Median	45
	Range	1-90
Gender	Female	1,110 (50.5%)
	Male	1,090 (49.5%)
Specimen type	Blood	449 (20.4%)
	Sputum	448 (20.4%)
	Wound swab	448 (20.4%)
	Urine	433 (19.7%)
Susceptibility testing method	Stool	422 (19.2%)
	Disc diffusion	755 (34.3%)
	MIC	724 (32.9%)
	Automated system	721 (32.8%)
Clinical outcome	Recovered	739 (33.6%)
	ICU	775 (35.2%)
	Deceased	686 (31.2%)

Note: Percentages may not sum exactly to 100% because of rounding.

3.2 Phenotypic Antimicrobial Susceptibility Patterns

Susceptibility distributions of the 5 antimicrobial agents were generally even for sensitive, intermediate, and resistant groups, with slight differences between the individual agents. The highest number of resistant observations was seen with meropenem (34.3%, $n = 755$) followed by amoxicillin (33.2%, $n = 730$) (Table 2). Most of the antibiotic resistance (32.6%, $n = 701$) was seen in both ciprofloxacin and vancomycin. The frequency of antibiotic resistance was the same for ciprofloxacin and vancomycin (32.6%), and the lowest frequency of antibiotic resistance was for colistin (31.9%, $n = 701$). However, vancomycin (34.4%), colistin (34.6%) and ciprofloxacin (34.2%) had the greatest percentages of sensitivity. The overall susceptibility structure is shown in Figure 1; about 1/3 of the observations were in each susceptibility category for all five drugs.

Table 2. Antimicrobial susceptibility profiles for the five evaluated antibiotics

Antibiotic	Sensitive, n (%)	Intermediate, n (%)	Resistant, n (%)
Amoxicillin	726 (33.0%)	744 (33.8%)	730 (33.2%)
Ciprofloxacin	752 (34.2%)	730 (33.2%)	718 (32.6%)
Meropenem	708 (32.2%)	737 (33.5%)	755 (34.3%)
Vancomycin	757 (34.4%)	726 (33.0%)	717 (32.6%)
Colistin	761 (34.6%)	738 (33.5%)	701 (31.9%)

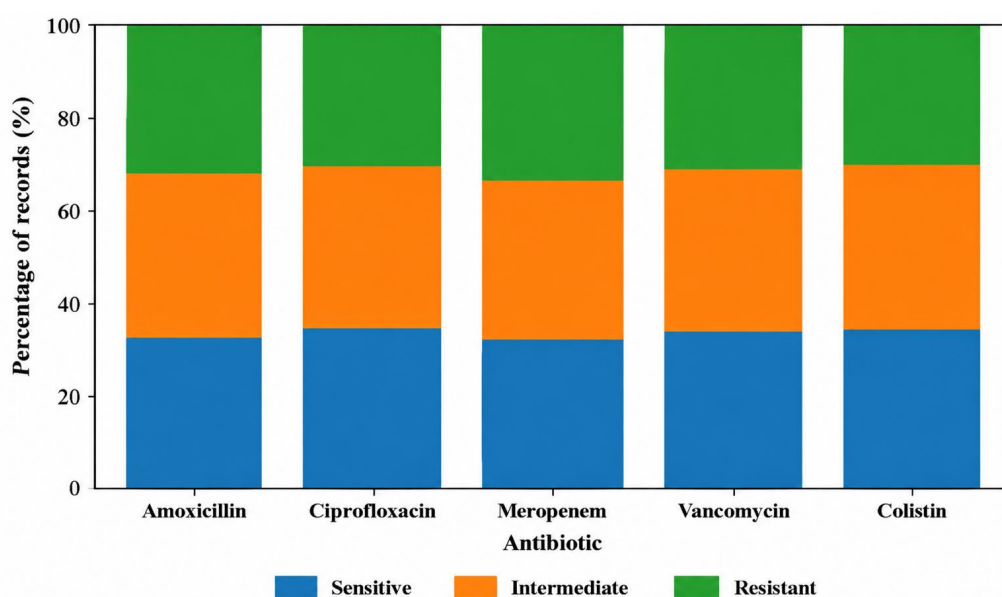


Figure 1. Antimicrobial susceptibility profiles across five antibiotics

Phenotypic resistance burden was also determined as the number of antimicrobials for which each record was classified as resistant (among the five antimicrobials). Overall, 86.1% of the records showed resistance to at least one antimicrobial. The number of antibiotics with resistance was mean 1.65 ± 1.04 with a median of two antibiotics with resistance. Specifically, 305 of the records were also found to be resistant to no antimicrobial, 711 to one, 732 to three, 365 to four, 84 to five and 3 were resistant to all five antimicrobials.

3.3 Resistance Gene Distribution Across Clinical Specimens

The four recorded genetic markers were found to have relatively even resistance gene information. VIM was the most frequent recorded gene, occurring in 463 records (21.0%), followed by OXA-48 in 450 (20.5%), KPC in 425 (19.3%), and NDM-1 in 425 (19.3%). A resistance gene was not observed in 437 observations (19.9%). The variation of the different types of specimens is displayed in Table 3 and shown in Figure 2. Blood was the most common source of VIM (23.2%) and sputum was the most common source of OXA-48 (23.4%), with the largest gene category. The proportion of urine samples showing KPC was relatively high (22.9%) while the proportion of wound swabs with no detected resistance gene was the highest (22.8%). Overall, however, there was no significant association between specimen type and the resistance-gene category, with a relatively small association for each category, $\chi^2(16) = 20.89$, $p = 0.183$, Cramer's $V = 0.049$.

Table 3. Resistance gene distribution according to clinical specimen type

Specimen type	KPC, n (%)	NDM-1, n (%)	OXA-48, n (%)	VIM, n (%)	None recorded, n (%)
Blood (n = 449)	85 (18.9%)	84 (18.7%)	83 (18.5%)	104 (23.2%)	93 (20.7%)
Sputum (n = 448)	89 (19.9%)	93 (20.8%)	105 (23.4%)	91 (20.3%)	70 (15.6%)
Stool (n = 422)	75 (17.8%)	92 (21.8%)	91 (21.6%)	83 (19.7%)	81 (19.2%)
Urine (n = 433)	99 (22.9%)	77 (17.8%)	75 (17.3%)	91 (21.0%)	91 (21.0%)
Wound swab (n = 448)	77 (17.2%)	79 (17.6%)	96 (21.4%)	94 (21.0%)	102 (22.8%)
Overall (N = 2,200)	425 (19.3%)	425 (19.3%)	450 (20.5%)	463 (21.0%)	437 (19.9%)

Note: Percentages for specimen rows are calculated within each specimen type. Pearson $\chi^2(16) = 20.89$, $p = 0.183$; Cramer's $V = 0.049$.

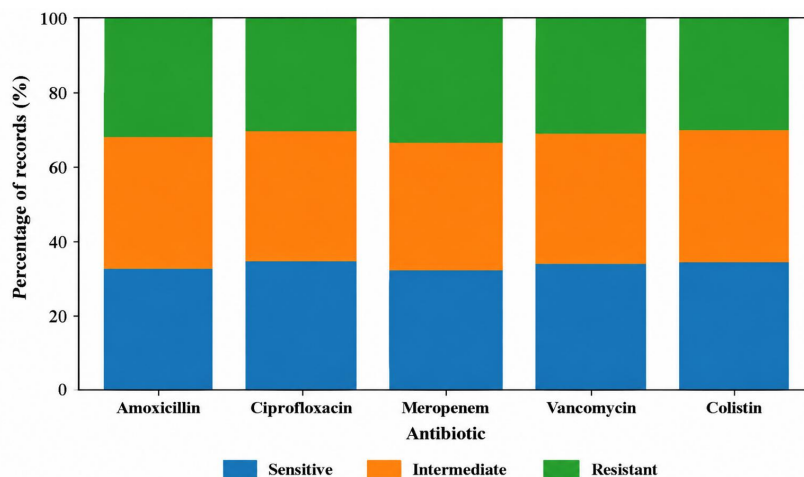


Figure 2. Resistance gene distribution across clinical specimen types

3.4 Resistance Burden and Clinical Outcomes

There was no difference in the distribution of resistance burden between the three clinical outcome groups. Patients were classified as recovered and had a mean resistance burden of 1.66 ± 1.04 , while ICU records had a mean resistance burden of 1.65 ± 1.02 , and deceased records had a mean resistance burden of 1.63 ± 1.06 . All three groups had a median number of 2 resistant antibiotics. In each outcome category, resistance to one or two antibiotics made up the highest percentage as shown in Figure 3. There was no statistically significant association between the complete six-level distribution of resistance burden and outcomes, $\chi^2(10) = 7.00$, $p = 0.725$, Cramer's $V = 0.040$. Likewise, the difference in resistance burden between the recovered, ICU and deceased groups was not significant, Kruskal-Wallis's test, $H = 0.54$, $p = 0.765$.

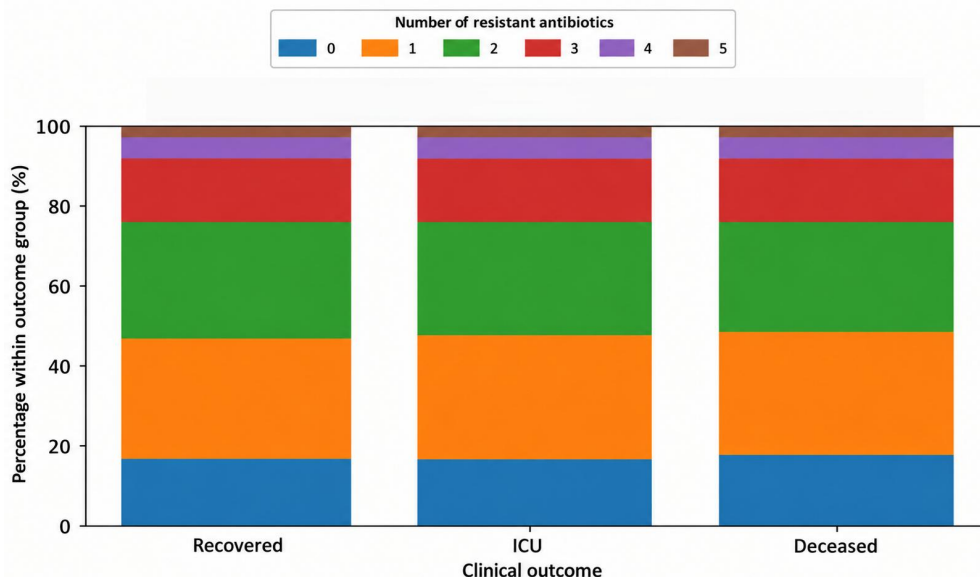


Figure 3. Distribution of phenotypic antimicrobial resistance burden according to clinical outcome

The multivariable binary logistic regression was then conducted in which all records of admission to the ICU and death were grouped together to create an adverse clinical outcome category and compared to those that recovered. All the four factors age, gender, specimen type, resistance-gene category and phenotypic resistance burden were entered in the same step. As seen in Table 4, none of the examined predictors had a statistically significant independent association with adverse outcome. The adjusted odds ratio (aOR) for each extra resistant antimicrobial was 0.98 (0.90–1.07, $p = 0.653$). There was also no statistically significant association between adverse outcome and age (aOR = 1.001 per year, $p = 0.701$). Urine samples had the highest positive point estimate (aOR = 1.18) but the confidence interval was not statistically different from 1 ($p = 0.263$). Similarly, no one of the resistance-gene categories was significantly different from the KPC reference category. The overall regression model was not statistically significant (likelihood-ratio $p = 0.881$; pseudo- $R^2 = 0.002$).

Table 4. Multivariable logistic regression for factors associated with adverse clinical outcome

Predictor	Adjusted OR	95% CI	p-value
Age, per year	1.001	0.997-1.004	0.701
Male vs female	0.94	0.79-1.12	0.489
Sputum vs blood	1.09	0.83-1.44	0.535
Stool vs blood	0.90	0.68-1.19	0.449
Urine vs blood	1.18	0.89-1.56	0.263
Wound swab vs blood	0.97	0.73-1.27	0.811
NDM-1 vs KPC	0.99	0.74-1.32	0.935
No gene recorded vs KPC	0.97	0.73-1.29	0.818
OXA-48 vs KPC	0.95	0.72-1.26	0.736
VIM vs KPC	0.89	0.67-1.18	0.413
Resistance burden, per additional resistant antibiotic	0.98	0.90-1.07	0.653

Note: Outcome was coded as recovered = 0 and adverse clinical outcome (ICU or deceased) = 1. Reference categories were female gender, blood specimen, and KPC gene category. OR = odds ratio; CI = confidence interval.

The adjusted odds ratios and corresponding 95% confidence intervals are graphically presented in Figure 4 (The vertical dashed line at OR = 1 represents the null association). All confidence intervals crossed the null value of 1.0, supporting the absence of independently significant associations in the fitted model.

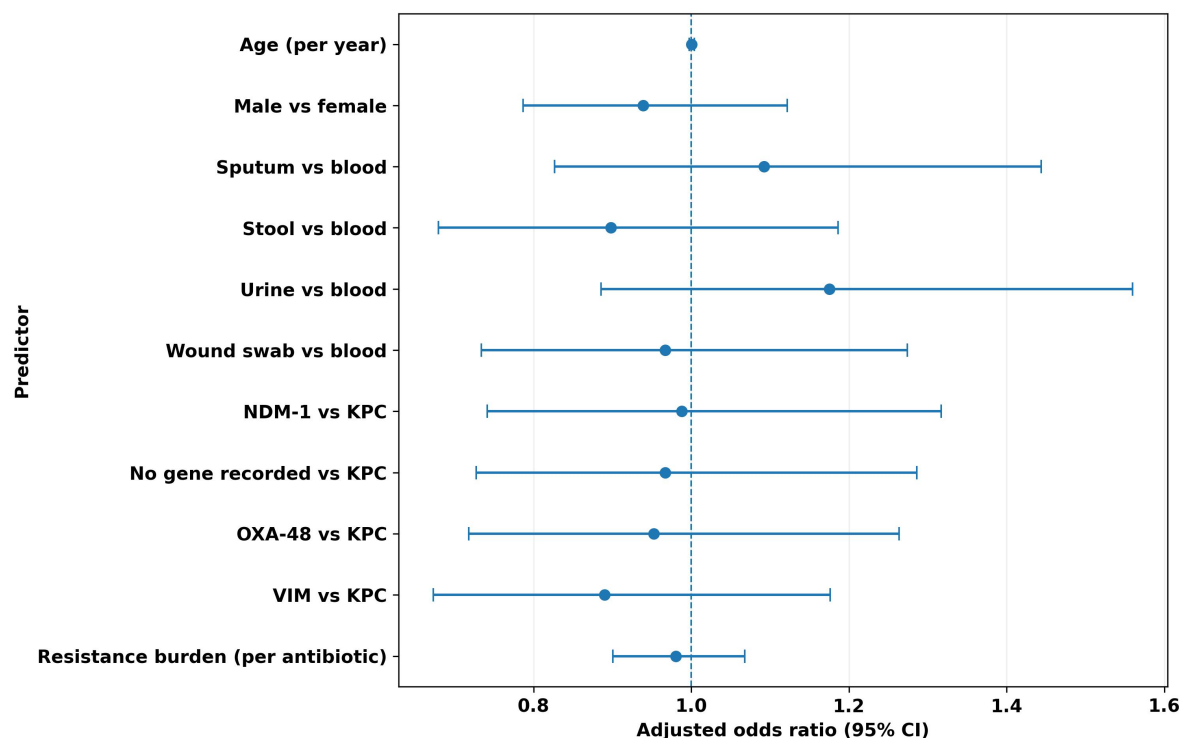


Figure 4. Adjusted odds ratios and 95% confidence intervals for factors associated with adverse clinical outcome

4. Discussion

Identification of broadly balanced sensitive, intermediate and resistant phenotypes for the five antimicrobials tested (resistance proportions between 32% and 34%) was done. The proportion of resistance of meropenem was the highest, and colistin was the lowest. This difference in activity between antimicrobial agents is not unusual for the multifactorial, multifaceted clinical phenomenon of antimicrobial resistance, as the antimicrobial resistance profile can be distinct for different isolates and other microbiological factors (Dunphy et al., 2021). In addition, considerable genetic variation among clinical isolates of *P. aeruginosa* has been shown in recent molecular studies of antimicrobial-resistance mechanisms (Al-Ouqaili et al., 2026).

A high overall level of resistance burden is suggested by 86.1% of the records showing resistance to at least one antimicrobial. The average burden was, however, only a couple of resistant agents, and almost no one had a resistance to all five. Phenotypic resistance and important virulent or resistance determinants have also been found to be associated in *Staphylococcus aureus* in studies among those who were hospitalized (Farrukh et al., 2026). In addition, genotypic studies have revealed that resistant phenotypes can also have unique molecular features that can affect biological behavior and clinical severity (Hamada et al., 2026).

The distribution of resistance-gene categories was relatively even and VIM was most common followed by OXA-48 in the present analysis. No significant association was observed between the type of specimen collection and the type of gene category, indicating that the resistance genes reported were not strongly clustered in a specific clinical specimen source. Clinically relevant OXA-48 has been shown in previous work in urine cultures with carbapenem-resistant *Escherichia coli* (Gurung et al., 2020b) and *Klebsiella pneumoniae* (Al-Fayyad et al., 2018) isolates. Gene distribution, however, can vary depending on the organism, geographic location, selective pressure and source of infection. This has also been reported for pathogenic *E. coli*, where associations between resistance genes and phenotypic antimicrobial response have been found (Abd El-Baky et al., 2020).

The weak specificity-gene relationship found here provides support for the idea that resistance determinants could possibly be shared among various ecological and clinical niches. Resistance-associated characteristics can exist in more than one case in a single source of biofilms, such as *Pseudomonas aeruginosa* (Kamali et al., 2020). Virulence and resistance traits can also coexist in a complex fashion as seen in *Enterococcus faecalis*, where some virulence genes do not appear to have a direct correlation with the level of resistance (Hashem et al., 2021). The study of *S. aureus* has also revealed the genotypic and phenotypic heterogeneity in the production of biofilm and antimicrobial resistance (Bissong & Ateba, 2020).

There was no significant difference between the recovered, the ICU and the deceased groups in terms of resistance burden. In addition, multivariable logistic regression revealed that none of the following factors was statistically significantly independently associated with an adverse outcome: age, gender, specimen type, resistance-gene category, or number of resistant antimicrobials. This indicates that, given the variables in hand, resistance burden alone, could not be responsible for poor clinical outcome. Research on high-level resistant organisms has also shown that clinical outcomes are determined not only by the resistance level of the organism itself, but also by the host and the pathogen as well as by treatment (Barai et al., 2026).

Therefore, the negative regression results do not necessarily mean that antimicrobial resistance is not clinically important. Instead, other factors like bacterial species, severity of infection, comorbidities, exposure to antimicrobial, appropriateness of treatment, virulence factors, and time of therapy should be considered when predicting outcomes. A diverse range of resistance genes and adaptive mechanisms have been described in other biological settings of multidrug-resistant *Pseudomonas aeruginosa* and have shown that the phenotype may be influenced (Algammal et al., 2020). Similarly, whole genome sequencing extensively resistant *E. coli* has shown that multiple molecular determinants can play a combined role in the manifestation of resistance (Jain et al., 2021).

The present analysis intentionally used the traditional statistical method instead of computation prediction. While artificial intelligence (AI) and machine learning (ML) are being considered for AMR surveillance

and decision support, their usefulness relies on representative data, comprehensive validation, and biologically relevant predictors (de la Lastra et al., 2024). One other constraint was the data lacked information about bacterial species, treatment specifics, and main clinical indicators of severity. Furthermore, complex biological microenvironments have been shown to influence drug resistance and clinical outcome in other disease contexts, further stressing the need for careful interpretation of simple resistance-outcome associations (Liu et al., 2024). Overall, the results confirm the need for a more comprehensive combined phenotypic, molecular and clinical surveillance and emphasize the need for more complex organism and patient level data in future studies.

5. Conclusion

Using secondary clinical data, this study aimed to describe the phenotypic antimicrobial resistance pattern, resistance-gene distribution and their association with clinical specimen type and clinical outcome. The resistance frequencies for all five agents were broadly similar, with meropenem having the highest proportion of observations of resistance, and colistin the lowest. Resistance was found in most records, but resistance to all five antimicrobials was rare. Resistance genes were evenly distributed among the different main categories of specimens with no statistically significant correlation among specimen type and gene distribution. The same applied to the phenotypic resistance burden, where no significant difference was seen between recovered, the ICU and the deceased group. Multivariable analysis confirmed that age, sex, type of specimen, resistance-gene category and cumulative resistance burden did not independently predict poor clinical outcome. These results suggest that the pattern of antimicrobial resistance cannot be well understood by only considering the source of specimen or resistance burden. To better understand the clinical implications of resistance, more comprehensive analyses are needed, including analyses of bacterial species, infection severity, comorbidities, and exposure to treatment, virulence characteristics, and therapeutic response. The study confirms the usefulness of integrated phenotypic, molecular and clinical surveillance as a valuable tool for monitoring antimicrobial resistance and guiding future microbiological and epidemiological investigations.

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