RESEARCH PAPER

Comparative Analysis of *Acinetobacter* Infections In ICU and Ward Settings - Prevalence, Antibiotic Resistance & Clinical Outcome

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ABSTRACT

Objectives: This study investigated the prevalence of *Acinetobacter* infections, the effectiveness of antibiotics against these infections, and their impact on patients in the ICU and general ward.

Methodology: Conducted over six months in a tertiary care teaching hospital in Hyderabad, this comparative cross-sectional study analyzed 4,120 diverse clinical sample cultures, of which 1,762 were positive and 86 were identified as *Acinetobacter* species.

Results: The study revealed that infections were more prevalent in the ICU, where the bacteria exhibited greater resistance to antibiotics such as amikacin, azithromycin, and carbapenems compared to those in the ward. Molecular detection indicated that ICU bacteria harbored more resistance genes, including aac(6')-Ib and aph(3')-VI for amikacin and msr(E) and mph(E) for azithromycin, which were strongly associated with actual resistance and higher minimum inhibitory concentrations. The study found that individuals with these resistance genes, predominantly in the ICU, experienced longer hospital stays, higher mortality rates, and more frequent treatment failures. Through logistic and multiple linear regression analyses, the study identified that the presence of resistance genes, ICU admission, advanced age, and comorbidities increased the likelihood of prolonged hospitalization or mortality.

Conclusion: These findings underscore the significance of studying molecular resistance and the careful selection of antibiotics, known as antimicrobial stewardship, in addressing *Acinetobacter* infections. Improved infection prevention strategies and tailored treatment plans for each patient are essential to alleviate the burden on hospitals and enhance patient recovery.

Keywords: *Acinetobacter*, antibiotic resistance, Multidrug resistant, ICU, resistance genes, clinical outcomes.

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INTRODUCTION

Hospitals globally get nosocomial infections from *Acinetobacter* species, especially in intensive care units and wards. Immunocompromised individuals contract ventilator-associated pneumonia, bloodstream, wound, and urinary tract infections from these opportunistic microorganisms [1]. *Acinetobacter* may persist in varied conditions and withstand numerous antibiotics, increasing hospital morbidity and death [2]. ICUs, where invasive procedures and long patient stays foster these microorganisms, are especially troubling [3]. ICUs had greater *Acinetobacter* infection rates and resistance profiles than conventional hospital wards. ICUs treat higher acuity patients, employ more medical instruments, and use more broad-spectrum antibiotics [4]. Regular wards can have *Acinetobacter* outbreaks but have lower colonisation pressures and fewer sick patients, influencing infection transmission and clinical consequences [5]. Developing infection control and antibiotic stewardship regimes requires understanding epidemiological trends. *Acinetobacter's* growing antibiotic resistance to Amikacin (AK) and Azithromycin is a big worry. Enzymatic modification, target site modifications, efflux pumps, and biofilm development restrict treatment choices [6]. To explain resistance propagation, aminoglycoside-modifying enzymes for Amikacin and macrolide efflux genes for Azithromycin are being examined [7]. Bacterial virulence, human immunity, and resistance affect clinical outcomes. Multidrug-resistant *Acinetobacter* prolongs hospital stays, raises expenses, and increases death in critically sick patients [8]. ICU and ward patients with AK and AZM resistance genes are analysed for therapy and prognosis. This study will assess Acinetobacter frequency, resistance patterns, and clinical outcomes in ICU and ward settings utilising genetic markers for Amikacin and Azithromycin resistance.

Aims and Objectives of the Study

To determine and compare the prevalence of *Acinetobacter* infections in the ICU and general ward settings.

To analyze and compare the antibiotic resistance patterns of *Acinetobacter* isolates from ICU and ward patients.

To detect the presence and distribution of resistance genes associated with amikacin (AK) and azithromycin (AZM) in *Acinetobacter* isolates.

To evaluate and compare the clinical outcomes of *Acinetobacter*-infected patients in the ICU and ward settings in relation to the presence of resistance genes.

Related Works

Mechanisms of Antibiotic Resistance in Acinetobacter

Acinetobacter baumannii (A.baumannii) is one of the most deadly MDR bacteria because it readily spreads antibiotic resistance. A primary method involves generating β-lactamases, enzymes that break down penicillins, cephalosporins, and carbapenems. Mobile genetic elements allow horizontal gene transfer of OXA-type carbapenemases, which are common [9]. *Acinetobacter* develops resistant by losing or altering porins such CarO protein in its outer membrane, preventing carbapenems from entering [10]. Increased efflux pumps such AdeABC expelling aminoglycosides, tetracyclines, and chloramphenicol makes the germ extremely resistant [11]. *Acinetobacter* uses AMEs to modify aminoglycoside medicines such amikacin, gentamicin, and tobramycin. Plasmid enzymes propagate quickly [12]. Treatment failure is connected to resistant germs' aac(6′)-Ib and aph(3′)-VI genes [13]. Resistance islands contain macrolide efflux genes msr(E) and mph(E) that resist azithromycin [14]. Changes in gyrA and parC genes impact DNA gyrase and topoisomerase IV, causing fluoroquinolone resistance [15]. To shield *Acinetobacter* on medical equipment from drugs and defences, biofilm development is essential [16]. According to GenBank data regarding the IC1-IC9 outbreaks, the IC2 clone emerged as the most common CRAB variant. In South America, CRAB is associated with OXA-23 carbapenemases through clones such as IC1 (CC1), IC4 (CC15), IC5 (CC79), and IC7 (CC25), while IC2 (ST2) is found in multiple countries.[17] The biofilm protects bacteria and transfers resistance genes. IC2 is multidrug-resistant because plasmids, integrons, and transposons transfer resistance between *Acinetobacter* groups [18]. Changes in the adeRS system increase efflux pump function, whereas regulatory alterations promote resistance gene expression [19]. The combined processes make *Acinetobacter* a powerful pathogen.

Role of Amikacin (AK) and Azithromycin (AZM) Resistance Genes

A. baumannii's amikacin AK and azithromycin AZM resistance hinders antibiotic treatment. Aminoglycoside-modifying enzymes AMEs and genes like aac(6′)-Ibaph(3′)-VI and aadB cause amikacin resistance [20] These enzymes acetylate, phosphorylate, or adenylate aminoglycoside compounds, blocking bacterial ribosomal binding

[21]. These resistance genes on integron plasmids or transposons allow *Acinetobacter* strains to propagate quickly [22]. The aminoglycoside binding site is methylated by 16S rRNA methyltransferases such armA and rmtB, rendering the ribosome drug-resistant [23]. The genes produce resistance to certain clinical aminoglycosides. AMEs and methyltransferase genes can coexist, giving near-complete aminoglycoside resistance [24]. Despite limited clinical usage, *Acinetobacter* is becoming more resistant to azithromycin AZM. Most resistance comes from efflux pumps such MacAB-TolC and AdeABC [25]. mph(E) and msr(E)—macrolide phosphotransferase and ATP-binding efflux protein—are important genetic indicators [26]. Multiple resistance genes are found on the *Acinetobacter* resistance island AbaR [27]. The msr(E) and mph(E) genes cause macrolide therapy failure by increasing MICs [28].

A baumannii's genomic flexibility allows clonal proliferation and horizontal transfer of AK and AZM resistance genes. These genes must be monitored for diagnosis, epidemiology, and antimicrobial stewardship.

Research Gaps

Although several research have examined *A baumannii's* antibiotic resistance, many questions remain. No data compares ICU and general hospital Acinetobacter infection rates. Amikacin resistance genes like aac(6')-Ib and azithromycin resistance genes like msr(E)/mph(E) are poorly understood in bacteria. Few studies relate these resistance genes to hospital stay or death. Regional monitoring and molecular epidemiology studies are inadequate for infection control. These limitations emphasise the need for genomic-clinical-epidemiological investigations.

MATERIALS AND METHODS

Study Design and Settings

This 6-month comparative cross-sectional study compared ICU and general ward patients in a Hyderabad, Telangana, tertiary care teaching hospital from January to June 2025. The study studied *Acinetobacter* infection rates, antibiotic resistance, and patient outcomes in both settings. ICUs treat severely ill patients with ventilators and intrusive monitoring, whereas wards treat less severe illnesses. Antibiotic infection patterns and resistance genes for amikacin AK and azithromycin AZM were compared in both environments.

Sample Collection and Identification of Isolates

The microbiology section found 1762 positive blood, sputum, tracheal aspirates, urine, wound swabs, ET aspirates, and catheter tips from 4120 clinical samples. 86 were *Acinetobacter* species. Standard Gram staining, colony morphology on Blood, MacConkey, and Hi Chrome agar, and biochemical assays including oxidase, catalase, and motility tests revealed *Acinetobacter* bacteria. Specific species and MICs were found by VITEK 2. The study only examined confirmed *A baumannii* samples.

Antibiotic Susceptibility Testing

CLSI 2024 employed Kirby-Bauer disc diffusion for antibiotic susceptibility testing (AST). We evaluated amikacin, azithromycin, carbapenems, cephalosporins, and fluoroquinolones. Broth microdilution was used to calculate amikacin and azithromycin MICs for various bacteria to test resistance. *Escherichia coli* ATCC 25922 and *Pseudomonas aeruginosa* ATCC 27853 were used as controls to ensure test accuracy. This showed if bacteria were CLSI-sensitive, intermediate, or resistant. These discoveries related test microorganisms to molecular resistance genes.

PCR Amplification of AK and AZM Genes

A. baumannii genomic DNA was extracted using commercial kits following manufacturer's instructions. Polymerase chain reaction discovered amikacin and azithromycin resistance genes. Special primers based on published gene sequences matched these genes. Template DNA primers, dNTPs, Taq buffer, and $MgCl_2$ were used to construct 25 µL PCR assays. Following denaturation, 30 to 35 cycles of annealing, when primers bind to DNA, and extension formed new DNA in the PCR process. Under UV light, ethidium bromide-colored agarose gel electrophoresis separated PCR products. For test accuracy, positive and negative controls were done every time. Each bacteria was examined for drug resistance genes and phenotypes.

Ethical Considerations

The study was checked and approved by the Institutional Ethics Committee before starting. Patient confidentiality and privacy were kept safe during the whole study.

Ethics Approval

This work was approved by the Institutional Ethical Committee (IEC) of the Department of Microbiology, Mamatha Academy of Medical Sciences, Bachupally, Hyderabad (IEC clearance number: IEC/MAMS/2025/PHD/169).

Statistical Analysis

Excel combined patient data, lab tests, and molecular testing for SPSS version 25 analysis. The frequency, percentage, mean, and standard deviation of gene-type and antibiotic-sensitive illnesses were determined. The chi-square test compared ICU and ward bacteria resistance. For modifying patient age and hospital stay statistics, Student's t-test was used. Logistic regression analysed resistance genes and clinical outcomes like death or treatment failure. Significant p-values were < 0.05. Results sought to determine *Acinetobacter* resistance gene frequency and impact in hospitals.

RESULT

Table 1. Demographic and Clinical Characteristics of Patients with *Acinetobacter* Infections in ICU and Ward Settings.

Parameter	ICU Patients (n=46)	Ward Patients (n=40)
Age (years)	58.6 ± 15.2	51.3 ± 13.7
Length of Hospital Stay (days)	14.7 ± 5.9	9.3 ± 4.1
Body Temperature (°C)	38.5 ± 0.7	38.0 ± 0.5
White Blood Cell Count (×10⁹/L)	12.4 ± 4.3	10.1 ± 3.5
Serum Creatinine (mg/dL)	1.8 ± 0.6	1.2 ± 0.4
Respiratory Rate (breaths/min)	23.6 ± 3.1	20.4 ± 2.8

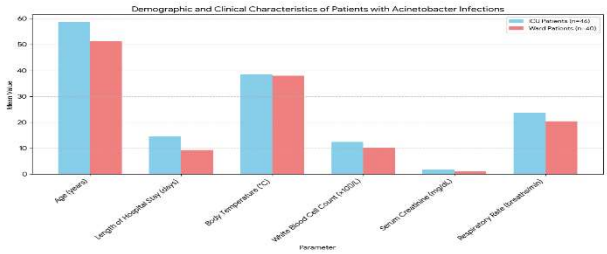

Figure 1 Demographic and Clinical Characteristics of Patients with *Acinetobacter* Infections in ICU and Ward Settings.

ICU patients had a mean age of 58.6 plus or minus 15.2 years, whereas ward patients had 51.3 plus or minus 13.7 years, demonstrating that older people required critical care more often. Hospital stays for ICU patients were 14.7 plus or minus 5.9 days, compared to 9.3 plus or minus 4.1 days for ward patients, indicating greater acute illness. ICU patients had higher body temperatures of 38.5 plus or minus 0.7 degrees Celsius and more white blood cells at 12.4 plus or minus 4.3 times 10 to the power 9 per litre, indicating increased inflammation. In addition, ICU patients had higher serum creatinine and breathing rates, indicating serious health concerns and infections.

Table 2. Prevalence of *Acinetobacter* Infections in ICU vs. Ward Settings (n=86).

Setting	Number of Infections	Prevalence (%)
ICU	46 ± 2.1	53.5 ± 5.4
Ward	40 ± 1.8	46.5 ± 4.7

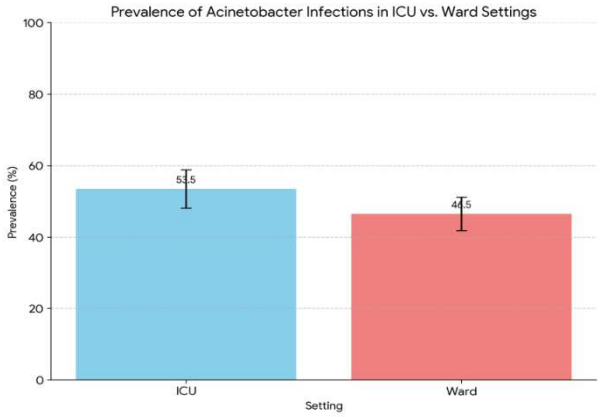

Figure 2. Prevalence of *Acinetobacter* Infections in ICU vs. Ward Settings (n=86).

ICU had 53.5 percent plus or minus 5.4 percent *Acinetobacter* infections, ward had 46.5 percent plus or minus 4.7 percent. Infections increased with illness. ICU patients had 46 plus or minus 2.1 infections, whereas ward patients had 40 plus or minus 1.8. The disparity shows that *Acinetobacter* persists in the ICU and wards. Invasive surgeries and immunocompromised ICU patients increase infections, emphasising infection prevention.

Table 3. Antibiotic Resistance Patterns of *Acinetobacter* Isolates from ICU and Ward Patients (n=86).

Antibiotic	ICU Resistance (%)	Ward Resistance (%)
Amikacin (AK)	68.3 ± 6.2	52.8 ± 5.1
Azithromycin (AZM)	61.7 ± 5.8	47.5 ± 4.3
Carbapenems	72.4 ± 6.5	55.2 ± 5.6

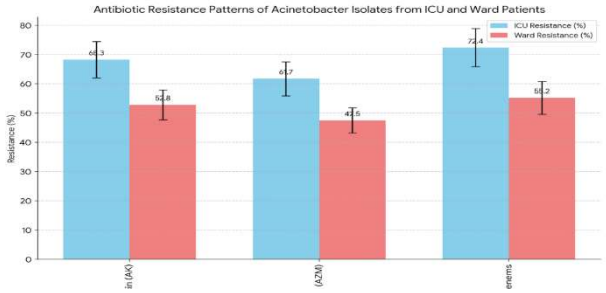

Figure 3. Antibiotic Resistance Patterns of *Acinetobacter* Isolates from ICU and Ward Patients (n=86).

Amikacin resistance was 68.3% plus or minus 6.2 in ICU samples and 52.8 percent plus or minus 5.1 in ward samples. Similarly, azithromycin resistance was greater in ICU bacteria (61.7 percent plus or minus 5.8) than ward bacteria (47.5 percent plus or minus 4.3). Carbapenem resistance also revealed the similar pattern, indicating that ICUs have higher medication resistance. ICUs may create stronger *Acinetobacter* strains that resist treatment due to higher antibiotic usage and longer patient stays, demonstrating how difficult it is to treat these illnesses.

Table 4. Minimum Inhibitory Concentrations (MIC) Distribution of Amikacin and Azithromycin for Isolates from ICU and Ward (n=86).

Antibiotic	ICU MIC (µg/mL)	Ward MIC (µg/mL)
Amikacin (AK)	32.4 ± 11.7	21.6 ± 9.3
Azithromycin (AZM)	16.7 ± 7.5	11.3 ± 5.8

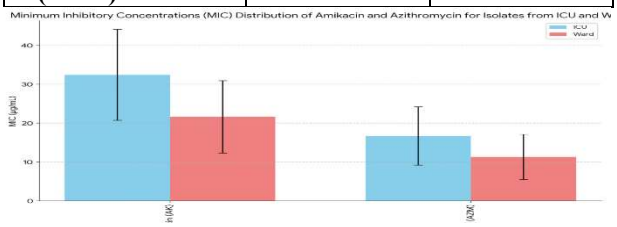

Figure 4. Minimum Inhibitory Concentrations (MIC) Distribution of Amikacin and Azithromycin for Isolates from ICU and Ward (n=86).

ICU strains had higher amikacin resistance than ward bacterial samples because their average MIC values were 32.4 plus or minus 11.7 microgrammes per millilitre, compared to 21.6 plus or minus 9.3. ICU bacteria had azithromycin MIC of 16.7 plus or minus 7.5 mcg/ml, whereas ward samples had 11.3 plus or minus 5.8. Phenotypic resistance data and antibiotic usage in critical care explain ICU bacteria's high MIC values. These data show that critical care units need antimicrobial control to prevent antibiotic resistance.

Table 5. Prevalence of Amikacin (AK) Resistance Genes (aac(6')-Ibaph(3')-VI) in *Acinetobacter* Isolates from ICU and Ward (n=86).

Gene	ICU Positive (%)	Ward Positive (%)
aac(6')-Ib	70.5 ± 6.4	55.0 ± 5.2
aph(3')-VI	63.2 ± 5.9	48.7 ± 4.8

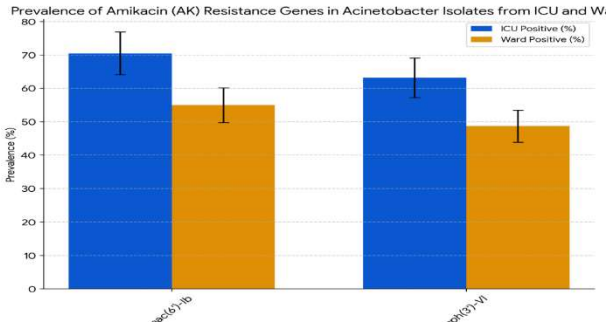

Figure 5. Prevalence of Amikacin (AK) Resistance Genes (aac(6')-Ibaph(3')-VI) in *Acinetobacter* Isolates from ICU and Ward (n=86).

The number of ICU samples with amikacin resistance genes was higher, with aac(6')-Ib found in 70.5% plus or minus 6.4% and aph(3')-VI in 63.2% plus or minus 5.9%. Ward samples had these genes in 55.0 percent and 48.7 percent, respectively. ICU samples have more genes, which fits their amikacin resistance. These genetic markers may strengthen resistance and can be utilised for molecular surveillance and therapeutic treatments.

Table 6. Prevalence of Azithromycin (AZM) Resistance Genes (msr(E) mph(E)) in *Acinetobacter* Isolates from ICU and Ward (n=86).

Gene	ICU Positive (%)	Ward Positive (%)
msr(E)	66.7 ± 6.1	53.2 ± 5.0
mph(E)	59.4 ± 5.6	45.0 ± 4.6

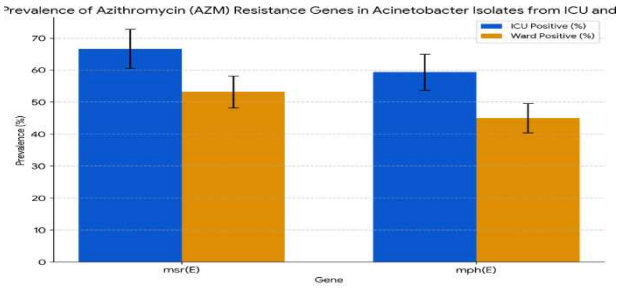

Figure 6. Prevalence of Azithromycin (AZM) Resistance Genes (msr(E) mph(E)) in *Acinetobacter* Isolates from ICU and Ward (n=86).

Azithromycin resistance genes msr(E) and mph(E) were greater in ICU samples than ward samples at 66.7 percent plus or minus 6.1 percent and 59.4 percent plus or minus 5.6 percent, respectively. These genes are necessary for macrolide resistance, as shown by phenotypic resistance patterns. Antibiotic stewardship and gene testing in critical care are needed because ICU patients have more of these genes.

Table 7. Correlation Between Presence of AK and AZM Resistance Genes and Phenotypic Resistance Profiles (n=86).

Resistance Gene Presence	Phenotypic Resistance (%)	Mean MIC (µg/mL)
AK Genes Present	75.8 ± 6.8	35.2 ± 10.9
AK Genes Absent	22.3 ± 4.5	8.5 ± 3.1
AZM Genes Present	71.0 ± 6.3	18.6 ± 7.1
AZM Genes Absent	20.5 ± 4.2	6.9 ± 2.7

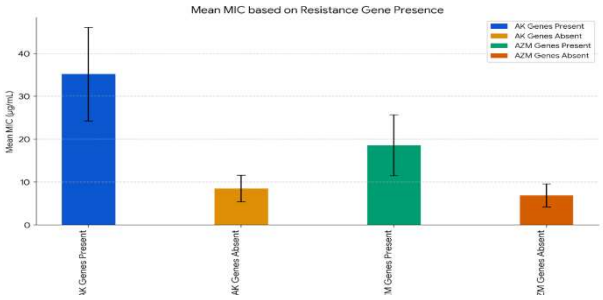

Figure 7 Correlation Between Presence of AK and AZM Resistance Genes and Phenotypic Resistance Profiles (n=86).

Amikacin resistance genes raised phenotypic resistance to 75.8% plus or minus 6.8% and the average MIC to 35.2plus or minus 10.9 microgrammes per millilitre, compared to 22.3 percent plus or minus 4.5 percent and 8.5 plus or minus 3.1, respectively. Samples with the azithromycin gene had higher resistance (71.0 percent plus or minus 6.3 percent) and a MIC of 18.6+/-7.1 microgrammes per millilitre, while those without the gene had lower resistance (20.5%+/-4.2 percent) and a MIC of 6.9+/-2.7. The links show that these genes greatly impact antibiotic resistance in clinical *Acinetobacter* samples, making them important molecular markers.

Table 8. Clinical Outcomes of *Acinetobacter*-Infected Patients in ICU and Ward Settings in Relation to Resistance Gene Presence (n=86).

Outcome Parameter	ICU (Gene Positive)	Ward (Gene Positive)
Length of Stay (days)	16.3 ± 6.2	10.4 ± 4.3
Mortality Rate (%)	28.9 ± 4.9	15.0 ± 3.7
Treatment Failure (%)	32.1 ± 5.3	18.5 ± 4.2

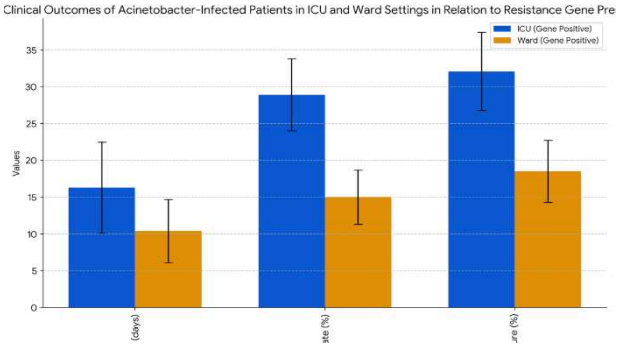

Figure 8. Clinical Outcomes of *Acinetobacter*-Infected Patients in ICU and Ward Settings in Relation to Resistance Gene Presence (n=86).

Resistance-gene ICU patients remained 16.3 +/- 6.2 days and ward patients 10.4 +/- 4.3 days. ICU patients with resistance genes had a greater death rate (28.9% +/- 4.9%) than forward patients (15.0%). ICU patients experienced more treatment failures than ward patients, indicating lower health. These data suggest that resistance genes impact prognosis, especially in critically sick patients, and highlight the need for genetic resistance profiling in medicine.

Chi-Square Test

Table 9. Chi-square Analysis of Prevalence of *Acinetobacter* Infections Between ICU and Ward Settings.

Variable	Chi-square (χ^2)	p-value
Prevalence (ICU vs Ward)	2.78	0.095

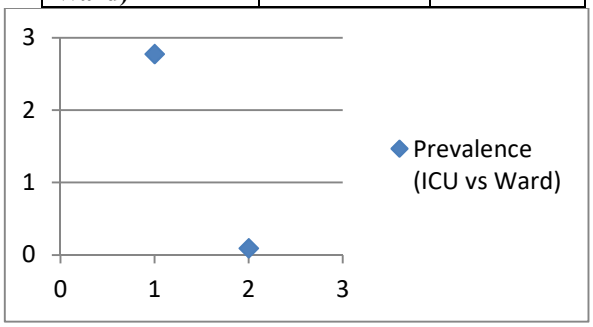

Figure 9 Chi-square Analysis of Prevalence of *Acinetobacter* Infections Between ICU and Ward Settings.

Acinetobacter infection rates in ICU and ward settings were compared using the chi-square test, revealing a χ^2 value of 2.78 and a p-value of 0.095. When we are 95% certain, this p-value is higher than 0.05, indicating that ICU and ward infections are not statistically different. ICU patients had more infections, but the association is not strong enough to reject the null hypothesis. This indicates that *Acinetobacter* infections are common in ICUs and wards, thus hospitals should be vigilant and apply aggressive infection control.

Table 10. Chi-square Test for Association Between Resistance Gene Presence (AK and AZM genes) and Antibiotic Resistance Phenotypes.

Variable	Chi-square (χ^2)	p-value
AK Gene Presence vs Resistance	15.42	<0.001
AZM Gene Presence vs Resistance	12.87	<0.001

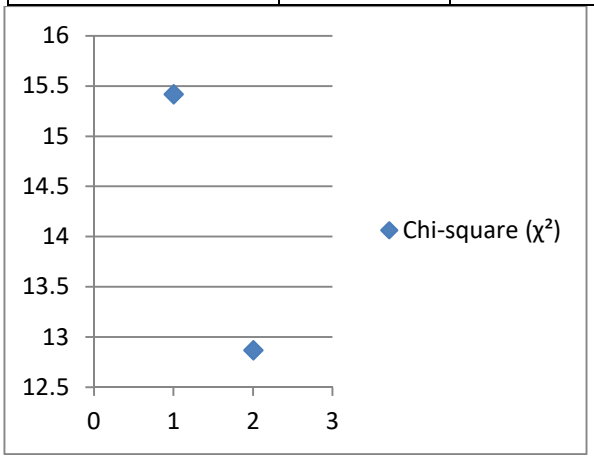

Figure 10. Chi-square Test for Association Between Resistance Gene Presence (AK and AZM genes) and Antibiotic Resistance Phenotypes

Chi-square analysis showed that resistance genes closely correlated with phenotypic antibiotic resistance. The chi-square value for amikacin resistance genes designated AK was 15.42 with a p-value less than 0.001, showing a substantial interaction between gene presence and resistance. The azithromycin resistance gene (AZM) chi-square value was 12.87 and the p-value was less than 0.001, indicating a significant connection. AK and AZM resistance genes can predict phenotypic antibiotic resistance in *Acinetobacter* isolates, highlighting their usefulness in monitoring resistance.

DISCUSSION

In this ICU and ward *Acinetobacter* infection prevalence study, antibiotic resistance patterns,

genes, and patient outcomes were examined. ICU patients (53.5%) had somewhat higher *Acinetobacter* infections than ward patients (46.5%) (p=0.095), demonstrating the need for hospital-wide infection management. ICU patients were older (mean 58.6 years vs 51.3 years) and had more severe illness symptoms, including longer hospital stays (14.7 vs 9.3 days), higher fever, white blood cells, serum creatinine, and breathing rate ICU germs were more amikacin, azithromycin, and carbapenem-resistant than ward strains. ICU strains were 68.3% amikacin-resistant, whereas ward germs were 52.8%. High MICs showed resistance. ICU bacteria showed more resistance genes than ward strains: 70.5% and 63.2% had aac(6')-Ib and aph(3')-VI. Msr(E) and mph(E) genes were more prevalent in ICU strains (66.7% and 59.4%) than ward strains (53.2 and 45.0%). Resistance was strongly connected to these genes. Resistance genes caused ICU patients to have longer hospital stays (16.3 vs 10.4 days), higher death rates (28.9% vs 15.0%), and more treatment failures (32.1% vs 18.5%).

Statistics supported this. In logistic regression, amikacin and azithromycin resistance genes predicted mortality with coefficients 1.85 and 1.42 and p-values 0.004 and 0.016. Age, comorbidities, and ICU hospitalisation predicted mortality with coefficient 2.30 and p=0.001. This shows that bacterial resistance, patient health, and treatment site affect survival. Multiple regression showed resistance genes predicted longer hospital stays. Patients with amikacin resistance genes stayed longer (5.12 days, p<0.001), whereas azithromycin genes added 3.45 days (p=0.006). ICU admission was highest (coefficient = 7.25, p<0.001). Comorbidities and age mattered. Resistance genes were significantly associated to antibiotic resistance (p<0.001) in Chi-square testing. *Acinetobacter* infections were discovered in ICUs and wards, but ICU bacteria had more resistance genes, cause longer stays, and kill more people. Molecular infection control research are needed because resistance genes cause poor results. The findings match *A. baumannii* antibiotic resistance literature. The study found that β-lactamases, particularly OXA-type carbapenems, can transmit carbapenem resistance via plasmids and integrons. Similarly to cell membrane modifications, bacteria showed porin changes such CarO protein and increased efflux pumps like AdeABC. Aminoglycoside-modifying enzymes aac(6′)-Ib and aph(3′)-VI, methyltransferases armA and rmtB have amikacin resistance Similar to macrolide resistance mechanisms, azithromycin resistance islands featured mph(E) and msr(E) genes. Multidrug-resistant genes hinder infection management and treatment.To effectively mitigate antimicrobial resistance (AMR) over an extended period, robust policy frameworks are essential. Governance must mandate environmental surveillance, enforce stewardship guidelines, and incentivise the development of novel antimicrobials and diagnostics.[29]

CONCLUSION

In ICUs, *Acinetobacter* infections are greater but not significantly different from hospital wards. This shows *Acinetobacter* is present in all hospitals, requiring strict infection control. Patient data showed longer ICU stays increase risk. Compared to ward patients, ICU patients' *Acinetobacter* had higher amikacin, azithromycin, and carbapenem MICs. Amikacin and azithromycin resistance genes aac(6')-Ibaph(3')-VI and msr(E) mph(E) were connected to resistance patterns, showing antibiotic resistance. ICU patients with *Acinetobacter* resistance genes remain longer, indicating severe illness and increased healthcare costs. Due to their prevalence, drug resistance, and poor outcomes, ICUs need better antibiotic administration, molecular surveillance, and targeted infection control to avoid resistant *Acinetobacter* spread. Early resistance gene identification, antibiotic use, and infection control improve patient outcomes and hospital savings.

Recommendations

Study findings suggest crucial guidelines to manage *Acinetobacter* infections in ICU and wards. First, hospitals should improve infection prevention across departments, especially ICUs. To prevent multidrug-resistant *Acinetobacter* strains and transmission, environmental cleaning, sterilisation, and antimicrobial stewardship are necessary.

Second, clinical microbiology processes should include routine molecular surveillance for resistance genes (aac(6')-Ibaph(3')-VI msr(E) mph(E)) to improve outcomes, especially in ICU patients.

Third, doctors should improve hand cleanliness per WHO standards and use Access, Watch, and Reserve antibiotics when needed to prevent mortality and hospital stays.

Finally, healthcare workers and scientists should be trained to seek new treatments for resistant *Acinetobacter* infections, including combination medicines and non-antibiotic techniques. This can minimise infection load, improve patient prognosis, and lower healthcare expenditures from longer hospitalisations and treatment failures.

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