

Prevalence and Antimicrobial Resistance Profile of *Acinetobacter baumannii* Among ICU Isolates in a Tertiary Care Centre in Central India

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ABSTRACT

Background: *Acinetobacter baumannii* is an important non-fermenting Gram-negative bacillus causing healthcare-associated infections, especially in intensive care units (ICUs). Phenotypic identification may not reliably differentiate *A. baumannii* from other members of the *Acinetobacter calcoaceticus-baumannii* complex. This study aimed to determine the prevalence, antimicrobial susceptibility pattern, and genotypic confirmation of *A. baumannii* among ICU isolates from a tertiary care centre in central India. **Methods:** This observational cross-sectional laboratory-based study was conducted in the Department of Microbiology, Index Medical College Hospital & Research Centre, Indore, from January 2024 to December 2025. A total of 266 ICU clinical samples were included. Isolates were identified phenotypically by standard microbiological methods, and antimicrobial susceptibility testing was performed by the Kirby-Bauer disk diffusion method and broth microdilution methods according to CLSI guidelines. PCR was performed for detection of the intrinsic blaOXA-51-like gene for confirmation of species. **Results:** Among 266 ICU samples, 41(15.41%) were phenotypically identified as *Acinetobacter* species, and all 41(100%) were confirmed as *A. baumannii* by blaOXA-51-like gene detection. Most isolates were recovered from patients aged 30-60 years, 23(56.1%) and from male patients 27(65.9%). The Medical ICU contributed the highest number of isolates, 16 (39.0%), and endotracheal aspirate was the predominant specimen source, 28(68.3%). High resistance was observed to cephalosporins, fluoroquinolones, aminoglycosides, beta-lactam/beta-lactamase inhibitor combinations, and carbapenems. The overall prevalence of *A. baumannii* among ICU specimens was 15.4% (41/266; 95% confidence interval [CI]: XX–XX%). Based on antimicrobial susceptibility profiles, XX isolates fulfilled the criteria for multidrug-resistant (MDR) *A. baumannii* according to the international definitions proposed by Magiorakos et al. **Conclusion:** *A. baumannii* is an important ICU-associated multidrug-resistant pathogen in Central India. Molecular confirmation by blaOXA-51-like gene detection improves species-level identification. The high carbapenem resistance rate emphasises the need for antimicrobial resistance surveillance, infection control, ventilator care bundles, and antimicrobial stewardship in ICU settings. **Categories:** Other, Trauma, Infectious Disease

Keywords: *Acinetobacter baumannii*, antimicrobial susceptibility, blaOXA-51-like gene, carbapenem resistance, central india, icu, non-fermenting gram-negative bacilli

INTRODUCTION

Non-fermenting Gram-negative bacilli (NFGNB), especially *Acinetobacter baumannii*, have become a major contributor to healthcare-associated infections (HAIs) worldwide, presenting a considerable public health challenge [1]. These organisms are increasingly contributing to nosocomial infections, especially in

critical care units, surgical wards, and among patients with invasive devices, including ventilators and central lines. These infections encompass surgical site infections, bloodstream infections, and ventilator-associated pneumonia. Their increasing prevalence markedly complicates empirical treatment and is linked

to extended hospitalisations, elevated morbidity, and heightened mortality [2].

Acinetobacter baumannii is the predominant clinically important species within the *Acinetobacter calcoaceticus-baumannii* complex. Although documented in warm and humid environments, community-acquired infections caused by *A. baumannii* are still few. Factors that increase the likelihood of *A. baumannii* infection include ICU admission, advanced age, immunosuppression, intravascular devices, invasive procedures, malignancies, mechanical ventilation, prior colonisation, prolonged hospital stay, recent surgical interventions, and the severity of the illness [3].

Acinetobacter species have emerged as prominent pathogens in healthcare-associated illnesses in recent years [4]. The prevalence of hospital-acquired infections caused by *Acinetobacter baumannii* has been consistently increasing, particularly in recent years [5].

The emergence of antimicrobial resistance (AMR) is a natural occurrence either from mutations in bacterial genomes or the horizontal transfer of external resistance genes among bacteria. Bacteria can possess multiple resistance mechanisms concurrently, rendering them resistant to many types of antibiotics [6]. The accumulation of multiple resistance mechanisms can result in the development of multidrug-resistant (MDR) strains.

Multidrug-resistant *A. baumannii* is defined by its non-susceptibility to at least one agent from three or more antimicrobial categories. Extensively drug-resistant isolates exhibit non-susceptibility to at least one agent in all but two or fewer antimicrobial categories.

Acinetobacter isolates exhibit resistance to almost all β -lactam antibiotics, aminoglycosides, and quinolones. *Acinetobacter baumannii* possesses intrinsic resistance to several antimicrobial agents through mechanisms including low outer-membrane permeability, constitutive β -lactamase production, multidrug-efflux pumps, and alterations in antimicrobial targets, while acquired resistance mechanisms further contribute to its multidrug-resistant phenotype. The increasing prevalence of multidrug resistance in this pathogen presents a therapeutic hazard to hospitalised patients by limiting the availability of antimicrobial options [7].

The global concern is the alarming trend of carbapenem resistance in *A. baumannii*, resulting from the acquisition of a diverse array of resistance mechanisms. The literature clearly demonstrates the rapid development of resistance to carbapenems and expanded-spectrum cephalosporins [8]. The emergence of multidrug- and pandrug-resistant phenotypes in *Acinetobacter* species is alarming, as they confer resistance to multiple antibiotic classes [9].

The increased incidence of carbapenem resistance has led to a reduced number of therapeutic alternatives for the treatment of severe carbapenem-resistant *A.*

baumannii (CRAB) infections. The treatment was mostly mono- and combination therapy with colistin, tigecycline, carbapenem, rifampicin, amikacin, sulbactam and minocycline. However, toxicity, suboptimal pharmacokinetics and/or increasing resistance pose challenges to the use of one or more of these agents and make effective management of the infection difficult [10].

A. baumannii has been designated as a globally prioritised surgical-site pathogen by the World Health Organisation (WHO) and is listed among the ESKAPE pathogens, which are known for their resistance to various antibiotics, thereby limiting treatment options. Antimicrobial resistance (AMR) refers to the capacity of bacteria to endure one or more antimicrobials that were previously effective in treating infections caused by these organisms. Annually, over 0.7 million deaths occur worldwide due to infections caused by antimicrobial-resistant (AMR) bacteria. By 2050, it is estimated that a person will die from an AMR infection every 3 seconds, with the total global costs of such infections projected to reach \$100 trillion [11].

The World Health Organization (WHO) has identified carbapenem-resistant *Acinetobacter baumannii* (CRAB) as a Critical Priority Pathogen because of its rapidly increasing antimicrobial resistance and limited therapeutic options. Continuous molecular surveillance and monitoring of local antimicrobial susceptibility patterns are therefore essential to guide empirical antimicrobial therapy, strengthen infection prevention programmes, and support antimicrobial stewardship initiatives in intensive care settings.

The phenotypic approaches for the precise identification of *Acinetobacter baumannii* are not highly dependable. Standard laboratory techniques will identify just the genus *Acinetobacter*, not the species [12]. Molecular testing can be employed for the earlier and more precise identification of both genus and species. There is increasing evidence that *Acinetobacter baumannii* possesses the blaOXA-51-like gene. This gene is naturally occurring and is exclusively found in this species [13].

In Central India, information on the frequency and resistance characteristics of ICU-associated *A. baumannii* isolates with molecular validation is scarce.

Objectives

The primary objective of this study was to determine the prevalence of *Acinetobacter baumannii* among ICU clinical specimens. Secondary objectives were to confirm species identification by PCR detection of the intrinsic blaOXA-51-like gene, determine antimicrobial susceptibility patterns, assess carbapenem resistance, and provide local epidemiological data to support antimicrobial stewardship and infection control programmes.

MATERIALS AND METHODS

Study Design and Setting

This observational, cross-sectional, laboratory-based study was conducted in the Department of Microbiology at Index Medical College Hospital & Research Centre, Malwanchal University, Indore, Madhya Pradesh, India, from January 2024 to December 2025. It included clinical samples from patients in various intensive care units, such as medical, surgical, paediatric, neonatal, and respiratory ICUs. The sample size was calculated for the source population of eligible ICU clinical specimens, using an anticipated prevalence of *A. baumannii* of 10%, a 99% confidence level, and 5% absolute precision. The calculation produced a minimum sample size of approximately 240. After adjusting for a 10% anticipated proportion of incomplete, contaminated, or otherwise non-evaluable specimens, the final required sample size was approximately 266.

The sample size was calculated using the formula:

$$n = Z^2 \times P \times (1 - P) / d^2$$

where $Z = 2.576$ (99% confidence level), $P = 0.10$ (anticipated prevalence), and $d = 0.05$ (absolute precision).

All eligible clinical specimens collected from ICU patients during the study period, with only the first isolate from each patient included, were considered. Repeat isolates from the same patient, specimens from non-ICU patients, contaminated or mixed-growth specimens, and samples with incomplete clinical or microbiological data were excluded.

METHODOLOGY

The study comprised clinical samples from ICU patients that exhibited growth of *Acinetobacter* species. To prevent duplication, only the initial isolate from each patient was taken into account. Samples were included solely when comprehensive clinical and microbiological data were accessible. Repeated isolates from the same patient, samples from non-ICU patients, and specimens exhibiting contamination or mixed growth were eliminated, unless the isolate was clinically pertinent and substantiated by appropriate documentation.

Sample Collection and Processing

Clinical specimens, comprising blood, endotracheal aspirate, sputum, bronchoalveolar lavage, urine, pus, wound swab, central venous catheter tip, and body fluids, were obtained from ICU patients according to stringent aseptic protocols. All samples were expeditiously sent to the microbiology laboratory and processed in accordance with established microbiological methods.

Blood-culture bottles were incubated in the BacT/ALERT 3D 120, bioMerieux India (P)Ltd, New

Delhi, India. while additional specimens were grown on blood agar, MacConkey agar, and other appropriate media. Culture plates were incubated under aerobic conditions at 37°C for a duration of 18 to 24 hours.

Isolates were presumptively identified based on colony morphology, Gram staining, oxidase testing and basic biochemical reactions. *Acinetobacter* species were classified as Gram-negative, non-motile, oxidase-negative, non-fermenting coccobacilli. further identification was conducted using an automated identification device, such as VITEK 2 COMPACT (bioMerieux India (P) Ltd., New Delhi, INDIA), with the GN identification card according to the manufacturer's instructions.

Antimicrobial Susceptibility Testing

Antimicrobial susceptibility testing was performed by the Kirby-Bauer disk-diffusion method on Mueller-Hinton agar using a bacterial suspension adjusted to a 0.5 McFarland standard. Antibiotic disks were obtained from HiMedia Laboratories Pvt. Ltd., Maharashtra, India. The disk potencies used were: ampicillin-sulbactam (10µg), piperacillin-tazobactam (10µg), cefotaxime (30µg), ceftriaxone (30µg), cefepime (30µg), ceftazidime (30µg), cefoperazone-sulbactam(30µg), imipenem (10µg), meropenem (10µg), amikacin (30µg), gentamicin (10µg), tobramycin (10µg), ciprofloxacin (5µg), levofloxacin (5µg), trimethoprim-sulfamethoxazole (1.25/23.75µg), and minocycline (30µg), tigecycline(15µg). Plates were incubated aerobically at 35±2°C for 16-18 hours. Zone diameters were interpreted according to the Clinical and Laboratory Standards Institute M100, 34th edition 2024. *Escherichia coli* ATCC 25922 and *Pseudomonas aeruginosa* ATCC 27853 were used for quality control.

Acinetobacter baumannii possesses intrinsic resistance to several antimicrobial agents, such as ampicillin, amoxicillin, ampicillin-clavulanate, aztreonam, ertapenem, trimethoprim, chloramphenicol; fosfomycin were not tested.

Colistin susceptibility was determined by the broth microdilution method using two-fold serial dilutions of colistin in cation-adjusted Mueller-Hinton broth. The MIC was recorded as the lowest colistin concentration that completely inhibited visible bacterial growth and was interpreted according to the CLSI M100, 34th edition (2024) recommendations.

Molecular Detection of blaOXA-51-like Gene

blaOXA-51-like gene encodes an intrinsic OXA-type class D β-lactamase and is located on the chromosome of *A. baumannii*. Although its basal expression may contribute only modestly to carbapenem resistance, overexpression-particularly when associated with an upstream insertion sequence-can increase carbapenem hydrolysis. Owing to its intrinsic association with *A.*

baumannii, detection of the gene is widely used to support species-level identification. PCR amplification using the selected primers produced an expected amplicon of 353 bp.

DNA was extracted from verified isolates utilising a commercial DNA extraction kit (Qiagen, Germany) was used and processed according to the manufacturer's guidelines. Molecular identification of *Acinetobacter baumannii* was conducted by conventional PCR to amplify the intrinsic blaOXA-51-like gene. The amplified products were separated by 1.5% agarose gel electrophoresis and visualised under UV transillumination.

The PCR reaction mixture was prepared according to the established laboratory protocol. The reaction mixture included template DNA, forward primer, reverse primer, PCR master mix, nuclease-free water, and other required reagents. The forward primer sequence used was 5'-TAATGCTTTGATCGGCCTTG-3', and the reverse primer sequence was 5'-TGGATTGCACTTCATCTTGG-3'. A 1000-bp DNA ladder was used as the molecular-size marker. The expected amplicon size of the blaOXA-51-like PCR product was 353 bp.

PCR amplification was performed using the following cycling parameters: denaturation at 94°C for 3 minutes, followed by 34 cycles of denaturation at 94°C for 30 seconds, annealing at 55°C for 30 seconds, and extension at 72°C for 30 seconds, concluding with a final extension at 72°C for 5 minutes. The amplified PCR products were examined using 1.5% agarose gel electrophoresis, stained with ethidium bromide, and visualised under UV transillumination. Each PCR cycle included appropriate positive and negative controls to ensure the validity of the findings.

DNA concentration and purity were assessed before PCR amplification. Appropriate positive (*Acinetobacter baumannii* ATCC 19606) and negative controls were included in each PCR run to ensure assay validity.

Statistical Analysis

Data were analysed using IBM SPSS Statistics version 29. Categorical variables were expressed as frequencies, percentages, and 95% confidence intervals where appropriate. Associations between categorical variables were evaluated using the Chi-square test or Fisher's exact test. A two-sided p-value <0.05 was considered statistically significant.

Ethics

The Institutional Ethics Committee of Malwanchal University authorised the research and assigned permission number IEC: MU/Research/EC/Ph.D/2023/347. Patient confidentiality was maintained during the research.

RESULTS

Prevalence of *Acinetobacter baumannii*

Throughout the study period, a total of 266 ICU clinical samples were analysed. Among these, 41(15.41%) isolates were phenotypically recognised as *Acinetobacter* species. All 41(100%) phenotypically tested isolates were confirmed as *A. baumannii* by PCR detection of the blaOXA-51-like gene.

Age-wise distribution of patients with *A. baumannii* Isolates

Of the 266 patients included in the study, the predominant age group was 30-60 years, with 139(52.3%) patients, followed by patients aged over 60 years, 92(34.6%). Patients under 30 years comprised 35(13.2%) of the study cohort. Among confirmed *A. baumannii* isolates, the highest number was also observed in the 30-60 years age group, 23(56.1%), followed by >60 years, 12(29.3%), and <30 years, 6(14.6%). The age-wise distribution is shown in Table 1.

Table 1: Age-wise distribution of patients with isolates

Age Group	Number of Patients (N=266)	Total Number of isolates (n=41)
<30 yrs	35 (13.1%)	06 (14.6%)
30-60 yrs	139 (52.4%)	23 (56.0%)
>60 yrs	92 (34.2%)	12 (29.2%)
Mean Age	56.8 ± 15.4 years	

Age groups are described descriptively and not as risk factors

Gender wise distribution of patients with *A. baumannii* isolates

Of the 266 patients in the study, 181(68.0%) were male, while 85(32.0%) were female. Among the 41 confirmed *A. baumannii* isolates, 27(65.9%) were recovered from male patients, whereas 14(34.1%) were recovered from female patients. The gender-wise distribution is shown in Table 2.

Table 2: Gender-wise distribution of patients with isolates

Gender	Number of Patients (N=266)	Prevalence of <i>A. baumannii</i> isolates (n=41)
Male	181 (68.4%)	27 (65.8%)
Female	85 (31.9%)	14 (34.1%)

Gender is described descriptively and not as a risk factor.

Distribution of *A. baumannii* isolates according to ICU type

A total of 41 confirmed *A. baumannii* isolates were recovered from different intensive care units. The Medical ICU contributed the highest number of isolates, 16(39.0%), followed by the Surgical ICU and Respiratory ICU, with 12(29.3%) isolates each. The Pediatric ICU contributed 1(2.4%) isolate. The ICU-wise distribution is shown in Table 3.

Table 3: Distribution of isolates according to ICU type

ICU type	Number of isolates (n=41)	Percentage
Medical ICU	16	39%
Surgical ICU	12	29.3%
Respiratory ICU	12	29.3%
Pediatric ICU	01	2.4%
Total	41	100%

Sample-wise distribution of confirmed *A. baumannii* isolates

Of the 41 confirmed *A. baumannii* isolates, the predominant source was endotracheal aspirate, with 28(68.3%) isolates. Sputum/BAL accounted for 4(9.8%) isolates, blood accounted for 3(7.3%) isolates, and urine, pus/wound swab, and central venous catheter tip each accounted for 2(4.9%) isolates. The predominance of respiratory specimens, especially endotracheal aspirates, indicates the important role of *A. baumannii* in ICU-associated lower respiratory tract infection. The sample-wise distribution is presented in Table 4.

Table 4: Sample-wise distribution of confirmed isolates

Sample type	Number of isolates (n=41)	Percentage
Endotracheal aspirate	28	68.3%
Blood	03	7.3%
Urine	02	4.9%
Pus/wound swab	02	4.9%
Sputum/BAL	04	9.8%
Central venous catheter tip	02	4.9%
Total	41	100%

Antimicrobial Resistance Pattern

A high level of antimicrobial resistance was observed against most routinely used antibiotics among the 41 confirmed *Acinetobacter baumannii* isolates. Cefotaxime, ceftriaxone, ceftazidime, and cefepime were all observed to exhibit complete resistance (100%) to third- and fourth-generation cephalosporins. Ciprofloxacin and levofloxacin also exhibited high resistance rates, with 37 (90.2%) isolates being resistant to each antibiotic. Gentamicin resistance was detected in 36

(87.8%) isolates, A high level of resistance to imipenem, meropenem, and piperacillin-tazobactam was observed, with 35 (85.3%) isolates exhibiting resistance to each of these antibiotics.

Resistance to ampicillin-sulbactam, tobramycin, and trimethoprim-sulfamethoxazole was detected in 34 (82.9%) isolates, whereas 33 (80.4%) isolates exhibited resistance to amikacin. Cefoperazone-sulbactam exhibited much reduced resistance, with 32 (78.0%) isolates demonstrating resistance. minocycline had intermediate efficacy, with 10 (24.4%) isolates being sensitive and 24 (58.5%) resistant. All 41 isolates (100%) showed sensitivity to tigecycline and colistin, reflecting the superior in vitro efficacy of both agents against the investigated isolates shown in Table 5.

Table 5: Antimicrobial resistance profile of confirmed isolates (n = 41)

Antibiotic	Sensitive n (%)	Intermediate n (%)	Resistant n (%)
Ampicillin-sulbactam	5 (12.2%)	2 (4.9%)	34 (82.9%)
Piperacillin-tazobactam	4 (9.8%)	2 (4.9%)	35 (85.4%)
Cefotaxime	0 (0%)	0 (0%)	41 (100%)
Ceftriaxone	0 (0%)	0 (0%)	41 (100%)
Ceftazidime	0 (0%)	0 (0%)	41 (100%)
Cefepime	0 (0%)	0 (0%)	41 (100%)
Cefoperazone-sulbactam	6 (14.6%)	3 (7.3%)	32 (78.0%)
Imipenem	5 (12.2%)	1 (2.4%)	35 (85.4%)
Meropenem	5 (12.2%)	1 (2.4%)	35 (85.4%)
Tobramycin	5 (12.2%)	2 (4.9%)	34 (82.9%)
Amikacin	5 (12.2%)	3 (7.3%)	33 (80.5%)
Gentamicin	4 (9.8%)	1 (2.4%)	36 (87.8%)
Ciprofloxacin	2 (4.9%)	2 (4.9%)	37 (90.2%)
Levofloxacin	3 (7.3%)	1 (2.4%)	37 (90.2%)
Trimethoprim-sulfamethoxazole	5 (12.2%)	2 (4.9%)	34 (82.9%)
Minocycline	10 (24.4%)	7 (17.1%)	24 (58.5%)
Tigecycline	41 (100%)	0 (0%)	0 (0%)
Colistin	41 (100%)	0 (0%)	0 (0%)

Colistin susceptibility was determined by broth microdilution, and minimum inhibitory concentration values were interpreted according to the applicable CLSI 2024 recommendations.

Carbapenem Resistance

Of the 41 confirmed *A. baumannii* isolates, 35 (85.3%) exhibited resistance to imipenem, while 35 (85.3%) showed resistance to Meropenem. Ertapenem was not included in the final susceptibility analysis because *A. baumannii* is intrinsically non-susceptible to this agent and organism-specific interpretive reporting is not appropriate. Carbapenem resistance was detected in 85.3% of isolates.

The estimated prevalence of carbapenem-resistant *A. baumannii* (CRAB) among confirmed isolates was 85.4% (35/41).

Molecular Detection by blaOXA-51-like Gene

Genotypic detection of the blaOXA-51-like gene was performed for species-level confirmation of *A. baumannii*. All 41(100%) isolates were positive for blaOXA-51-like gene amplification, supporting their identification as *A. baumannii* because this gene is considered intrinsic to the species.

PCR amplification of the blaOXA-51-like gene confirmed 41(100%) isolates as *A. baumannii*. The presence of the expected 353-bp band confirmed the identity of the isolates. Representative agarose gel electrophoresis findings are shown in Figure 1.

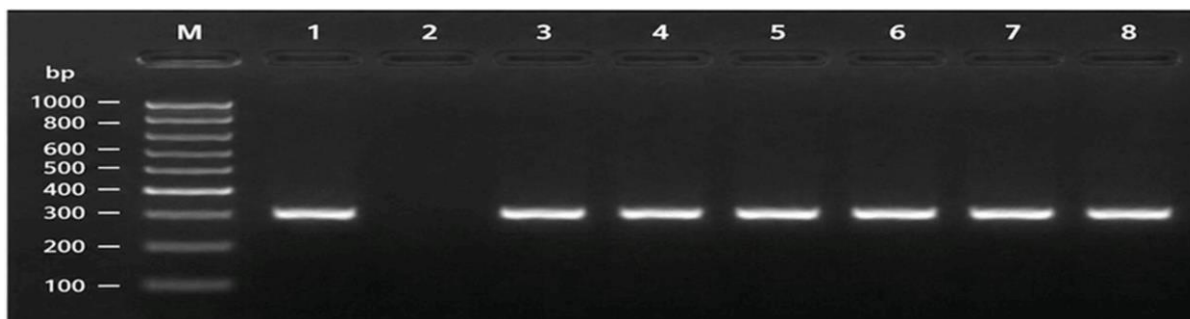


Figure 1: Agarose gel electrophoresis showing amplification of the blaOXA-51-like gene.

PCR amplification of the blaOXA-51-like gene in *Acinetobacter baumannii* isolates. Lane M shows the 1000 bp DNA ladder. Lane 1 represents the positive control, *Acinetobacter baumannii* ATCC 19606, and Lane 2 represents the negative control. Lanes 3-8 show clinical isolates positive for the blaOXA-51-like gene, with an expected amplicon size of 353 bp.

Association of Gender with *A. baumannii* Infection

Statistical analysis was performed to evaluate the association between gender and *A. baumannii* infection. No statistically significant association was observed between gender and *A. baumannii* isolation ($\chi^2 = 0.107$, df = 1, p = 0.744). The statistical analysis is shown in Table 6.

Table 6. Association between gender and *Acinetobacter baumannii* infection

Parameter	χ^2 value	df	p-value	Significance
Gender vs <i>A. baumannii</i> infection	0.107	1	0.744	Not significant

χ^2 : chi-square statistic; df: degrees of freedom. A p-value >0.05 was considered statistically not significant; significant p-values are denoted by *.

DISCUSSION

In the present study, *A. baumannii* was recovered from 41 of 266 ICU specimens, corresponding to an isolation proportion of 15.41%. direct comparison with other studies should be made cautiously because reported rates vary according to the denominator comprises ICU admissions, submitted clinical specimens, culture-positive specimens, *Acinetobacter* isolates [14]. All phenotypically identified the discovery of the blaOXA-51-like gene genotypically verified *Acinetobacter* isolates, therefore validating the efficacy of molecular confirmation in distinguishing *A. baumannii* from other constituents of the *Acinetobacter calcoaceticus-baumannii* complex. Prior research has also identified the blaOXA-51-like gene in all *Acinetobacter baumannii* strains [15].

The present study's prevalence of 41 among 266 isolates(15.41%) suggests that *A. baumannii* imposes a substantial burden on ICU patients. The protracted hospitalisation, mechanical ventilation, broad-spectrum antibiotic exposure, invasive procedures, and underlying critical illness render ICU patients highly vulnerable. In previous studies, *A. baumannii* was identified as a

significant cause of hospital-acquired infections, including ventilator-associated pneumonia, bloodstream infections, lesion infections, and device-associated infections. Similar observations have been reported. Recent evaluations have also underscored the fact that carbapenem-resistant *A. baumannii* continues to be one of the most challenging ICU pathogens due to its high outbreak potential and limited therapeutic options [16,17].

The Medical ICU yielded the most isolates in the current study, with the Surgical ICU and Respiratory ICU following closely behind. This discovery implies that adult critical care units are significant reservoirs for *A. baumannii*. The increased use of ventilators, central lines, urinary catheters, broad-spectrum antibiotics, and protracted ICU stays may be associated with the predominance of isolates from these ICUs. *A. baumannii* is renowned for its capacity to persist on sterile hospital surfaces for extended periods, establish biofilms, and disseminate through contaminated hands, equipment, and ICU environments. In critical care facilities, infection prevention and control are particularly critical due to these characteristics.

The sample-wise distribution indicated that the majority of *A. baumannii* isolates were recovered from endotracheal aspirates, which accounted for 28/41(68.3%) of the confirmed isolates. Lahiri et al. also reported that 48.8% of *Acinetobacter* isolates were recovered from respiratory samples [18]. Endotracheal aspirates accounted for 28/41 (68.3%) of the confirmed isolates and were therefore the most frequent specimen source. Therefore, recovery of *Acinetobacter baumannii* from respiratory specimens should not be interpreted as confirmation of ventilator-associated pneumonia or other lower respiratory tract infections. Previous studies have identified *A. baumannii* as an important respiratory pathogen in critically ill patients; nevertheless, its frequent recovery from endotracheal aspirates may also reflect its ability to colonise the respiratory tract, persist in the hospital environment, and exhibit resistance to multiple antimicrobial agents.

The age-wise analysis indicated that the majority of confirmed cases occurred in the 30-60 years age group, with 23/41 (56.1%) isolates, followed by patients aged over 60 years, with 12/41 (29.3%) isolates. Previous studies have similarly reported that *Acinetobacter baumannii* infections are frequently observed among adult, elderly, and critically ill ICU patients, particularly in those with prolonged ICU stay, invasive procedures, mechanical ventilation, severe illness, and prior antibiotic exposure [19-21]. The higher proportion of adult ICU admissions in the study population may explain this distribution. The gender distribution showed male predominance, with 27/41 (65.9%) isolates recovered from male patients; however, the association between gender and *A. baumannii* isolation was not statistically significant, suggesting that male predominance may reflect admission patterns rather than an independent risk factor [22].

This investigation revealed significant antibiotic resistance in confirmed 41 *A. baumannii* isolates. Complete resistance was noted against cefotaxime, ceftriaxone, ceftazidime, and cefepime, with 100% of isolates resistant to each. High resistance was also observed against ciprofloxacin and levofloxacin, each with 37(90.2%) resistant isolates; gentamicin, with 36(87.8%) resistant isolates; and piperacillin-tazobactam, imipenem, and meropenem, each with 35(85.4%) resistant isolates. This pattern indicates the multidrug-resistant characteristics of ICU-associated *A. baumannii*, and similar resistance patterns have been reported in Indian and international studies [23,24].

The observed high prevalence of carbapenem resistance may be explained by the widespread use of broad-spectrum antibiotics in critically ill patients, prolonged ICU stay, selective antimicrobial pressure, and dissemination of carbapenemase-producing *A. baumannii* strains within hospital environments.

This analysis confirmed that all 41(100%) isolates were *A. baumannii* by demonstrating positivity for the blaOXA-51-like gene. The blaOXA-51-like gene is generally considered intrinsic to *A. baumannii* and is widely used as a genetic marker for species confirmation. However, because rare non-*baumannii* *Acinetobacter* species carrying blaOXA-51-like genes have been reported, PCR-based detection should be interpreted along with reliable phenotypic or automated identification methods. In the present study, blaOXA-51-like PCR therefore strengthened the confirmation of phenotypically identified *A. baumannii* isolates [25].

The current investigation demonstrated complete in-vitro susceptibility to colistin and tigecycline, with 41(100%) isolates susceptible to each. This indicates that these agents may retain in-vitro activity against MDR *Acinetobacter baumannii* isolates in the study setting. Nevertheless, interpretation must be cautious. Broth microdilution is the recommended reference method for colistin susceptibility testing because disk diffusion and some gradient-based methods are unreliable due to poor diffusion and methodological variability. Therefore, colistin susceptibility should be validated using broth microdilution before clinical reporting. Tigecycline may show favorable in-vitro activity against MDR *A. baumannii*; however, its clinical use depends on infection site, pharmacokinetics, severity of illness, and local treatment guidelines [26].

Although all isolates remained susceptible to colistin and tigecycline in vitro, these findings should be interpreted cautiously. Increasing reports of heteroresistance and emerging resistance during therapy highlight the need for judicious clinical use and continuous surveillance.

This study reveals that ICU-associated *A. baumannii* in a tertiary care centre in Central India has a significant degree of multidrug resistance, especially to carbapenems. genetic verification with blaOXA-51-like PCR enhances the identification of *A. baumannii* and underscores the necessity of incorporating genetic techniques with standard phenotypic identification. The results underscore the necessity of ongoing observation of antimicrobial resistance, stringent infection prevention and control protocols, judicious antibiotic utilisation, ventilator care packages, environmental sanitation, and antimicrobial stewardship initiatives in intensive care units.

Clinical Implications

The high burden of carbapenem-resistant *A. baumannii* observed in this study supports implementation of antimicrobial stewardship programmes, routine ICU antibiogram surveillance, environmental decontamination, strict hand hygiene, ventilator care bundles, and rapid molecular confirmation of clinically significant isolates.

Limitations

This study had certain limitations. It was conducted at a single tertiary care centre, so the findings may not be generalisable to all hospitals in Central India. The reported 41(15.41%) represents the proportion of included ICU specimens yielding *A. baumannii*, not population-based prevalence. Statistical analysis was mainly descriptive, and clinical outcomes were not linked specifically to confirmed *A. baumannii* cases. Patient-level antimicrobial therapy, treatment duration, appropriateness of therapy, and clinical response were not analysed. In addition, only the blaOXA-51-like gene was tested for species confirmation; other resistance genes such as blaOXA-23, blaOXA-24/40, blaOXA-58, blaNDM, blaVIM, and blaIMP were not included. Detailed clinical outcome variables such as attributable mortality, length of ICU stay, ventilator duration, severity score, and treatment response were also not analysed.

Furthermore, molecular characterization of carbapenem-resistance genes (such as *blaOXA-23*, *blaOXA-24/40*, *blaOXA-58*, *blaNDM*, and *blaVIM*) was not performed and therefore the underlying resistance mechanisms could not be determined.

Future Directions

Future multicentre prospective studies incorporating larger sample sizes, whole-genome sequencing, molecular epidemiology, resistance gene profiling, and detailed patient outcome analysis are recommended to better understand the epidemiology of multidrug-resistant *Acinetobacter baumannii* in India.

Conclusions

This study highlights the continuing importance of *Acinetobacter baumannii* as a multidrug-resistant organism in intensive care settings. Molecular confirmation using the intrinsic blaOXA-51-like gene strengthened species-level identification, while the observed resistance pattern underscores the need for appropriate antimicrobial stewardship and continued local resistance surveillance. Because infection could not always be distinguished from colonisation and detailed patient-level risk factors and outcomes were not assessed, the findings should be interpreted within the limits of a single-centre laboratory-based study. Larger prospective multicentre studies using standardised clinical definitions and broader molecular characterisation are needed to clarify clinical significance, resistance mechanisms, risk factors, and patient outcomes.

Data Availability

The datasets generated and analysed during the current study are available from the corresponding author upon reasonable request.

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Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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