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Unveiling the Silent Threat: Phenotypic and Molecular Characterization of Carbapenem-Resistant *Acinetobacter baumannii* in a North Indian Tertiary Healthcare Facility

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ABSTRACT

Background: Acinetobacter baumannii is a critical nosocomial pathogen known for its multidrug resistance, particularly to carbapenems. To control the spread of A. baumannii in a healthcare facility, it is essential to understand its molecular epidemiology. This study compares different phenotypic and molecular techniques to detect these enzymes among carbapenem-resistant A. baumannii (CRAB) isolates, aiming to improve infection control and treatment strategies.

Methods: Over 18 months, 209 Acinetobacter isolates from various clinical specimens were analyzed at Dr. Ram Manohar Lohia Institute of Medical Sciences. Phenotypic methods included the Modified Hodge Test (MHT), Modified Carbapenem Inactivation Method (mCIM)for carbapenemases, and combined disk diffusion test (CDDT) and E-test for MBL detection. Genotypic characterization of 20 CRAB isolates was performed using the Carbapenemase Gene Detection kit.

Results: Of the 209 isolates, A. baumannii was the most common (89%) isolate, with 93% isolates showing carbapenem resistance. The mCIM detected carbapenemase production in 56.89% of isolates, compared to 26.43% by MHT. MBL production was identified in 35.6% of isolates by CDDT and 100% by E-test. All isolates harboured blaOXA-51 and blaOXA-23 genes, with blaNDM-1, blaIMP-1, and blaVIM detected in 90%, 40%, and 10% of isolates, respectively. Colistin demonstrated a high susceptibility rate of 82.2%, while minocycline showed a susceptibility rate of 23%.

Conclusions: The high carbapenem resistance in A. baumannii isolates, underscore the need for rapid and accurate detection methods. Despite high resistance rates, colistin and minocycline remain viable treatment options for CRAB.

Key Words: Acinetobacter baumannii, Carbapenem resistance, Carbapenemases, Metallo-β-lactamases, Multidrug-resistant

INTRODUCTION

Acinetobacter baumannii (A. baumannii) has emerged as a significant nosocomial pathogen worldwide. It belongs to the

ESKAPE group of multidrug-resistant (MDR) organisms, posing a severe threat to modern antibiotic therapy due to its diverse resistance mechanisms. The mortality rate from *A. baumannii* infections has increased, exacerbated by the slow development of new antibiotics and limited treatment options. [1]
The pathogen's ability to survive in various conditions facilitates its spread in hospital settings, causing infections like urinary tract infections, meningitis, bacteraemia, and pneumonia. [2]
Risk factors for *A. baumannii* infection include extended hospital stays, immunosuppression, advanced age, comorbidities, trauma, burns, prior antibiotic use, invasive procedures, and use of indwelling catheters or mechanical ventilation.[3] In ICU patients, mortality rates range from 23% to 68%,[4] while community-acquired infections, often in individuals with underlying conditions, can have mortality rates as high as 64%.[5] The prevalence of MDR strains in hospital-acquired pneumonia (HAP) and ventilator-associated pneumonia (VAP) is high, with an estimated 79.9% of strains being MDR.[6] Carbapenem resistance is particularly concerning, with rates in India ranging from 40-75%,[7] and as high as 85.8-87.8% according to the ICMR report 2022.[8]
Both the CDC and WHO have prioritized research and development of treatments for carbapenem-resistant *A. baumannii* (CRAB). Historically treated with carbapenems, emerging resistance to carbapenems has led to the use of polymyxins, despite their significant toxicities. *A. baumannii* employs various mechanisms for antibiotic resistance, including reduced membrane permeability, efflux pumps, genetic mutations, and enzyme-mediated antibiotic inactivation. Its genetic flexibility allows for rapid adaptation and incorporation of foreign resistance genes via mobile genetic elements.[9,10,11]
Beta-lactam antibiotics, including penicillin, cephalosporins, monobactams, and carbapenems, are ineffective against *A. baumannii* due to beta-lactamases.[12] These enzymes are classified into four types: Class A (resistant to a range of beta-lactams), Class B (metallo-beta-lactamases, which hydrolyse all beta-lactams except monobactams), Class C (chromosomally encoded cephalosporinases), and Class D (oxacillinases, effective at inactivating carbapenems).[13]
Accurate and rapid detection of carbapenemases is critical for clinical diagnosis. Methods include specific PCR, commercial microarrays, whole-genome sequencing, and MALDI-TOF MS. Phenotypic methods like the modified Hodge test, Carba NP test, and the modified carbapenem inactivation method (mCIM) are also used, though not all are recommended for *A. baumannii*.[14]
To control the spread of *A. baumannii* in a healthcare facility, it is essential to understand its molecular epidemiology. This study compares different phenotypic and molecular techniques to detect these enzymes among CRAB isolates, aiming to improve infection control and treatment strategies.

MATERIALS AND METHODS

This study was a descriptive analysis, conducted over 18 months (September 2022 to February 2024) at the Department of Microbiology, Dr. Ram Manohar Lohia Institute of Medical Sciences, Lucknow, following approval from the Institutional Ethical Committee (IEC No. 109/22). A total of 209 non-repetitive isolates of *Acinetobacter spp*. from clinical specimens (sputum, bronchoalveolar lavage, ascitic fluid, bile, blood, urine, and wound swabs). Clinical information like presence of diabetes, COPD, immune-compromised diseases, extended hospitalization, in situ prosthetic or foreign body implants, and a history of antibiotic use or catheterization were documented.
Sample was inoculated on MacConkey agar and blood agar. Plates were inspected for growth after 48 hours of aerobic incubation at 37^0C. The blood culture bottle was incubated at 37^0C for 24 hours then subculture on MacConkey agar and blood

agar media after 1,2,7 days followed by Gram staining of isolates.

Acinetobacter spp. were speciated and identified using Vitek –MS system (Biomeriux Germany). The Kirby-Bauer disc diffusion method was used to perform antibiotic susceptibility testing (AST). The zones of inhibition were measured after incubation, and results were interpreted using the 2023 guidelines from the Clinical and Laboratory Standards Institute (CLSI)[15]. Also, VITEK-2 Compact data for MIC distribution of colistin, meropenem and imipenem in carbapenem resistant *A. baumannii* was collected for 134 isolates.

Phenotypic detection of Carbapenemase production was done using following test

1. **Modified Hodge Test (MHT):** Following CLSI M100-S19 guidelines, a 0.5 McFarland standard suspension of *Escherichia coli* ATCC 25922 was streaked on Mueller Hinton Agar (MHA). A 10 µg ertapenem or meropenem disc was placed at the center. Test organism colonies were inoculated in a straight line from the disc edge to the plate edge. After 16-24 hours of incubation at 37°C, enhanced growth around the test organism indicated carbapenemase production.
2. **Modified Carbapenem Inactivation Method (mCIM):** A suspension of *A. baumannii* from a blood agar plate was emulsified in trypticase soy broth (TSB) with a 10 µg meropenem disc, incubated for 4 hours at 37°C. The disc was then placed on an MHA plate inoculated with *E. coli* ATCC 25922 and incubated for 18-24 hours. A zone diameter of 6–15 mm or pinpoint colonies within a 16–18 mm zone indicated carbapenemase production.[15]

Phenotypic detection of Metallo-beta-lactamase was done using following test

1. **Combined Disk Diffusion Test for MBL Detection:** Using imipenem (10 µg) and imipenem (10 µg) + EDTA (750 µg) discs on MHA, the difference in zone diameters was measured after 16-18 hours of incubation at 37°C. A difference of ≥7 mm indicated MBL production.[16]
2. **E-test for MBL Detection:** The Ezy MIC TM Strip EM092, containing imipenem alone and imipenem with EDTA, was used. A direct colony suspension was prepared, and a lawn culture was made on an MHA plate. After 16-18 hours of incubation at 37°C, an MIC ratio of >8 or >3 log2 dilutions indicated MBL production.

Genotypic characterisation of select 20 CRAB isolates was done using the Carbapenemase Gene Detection (HiMedia Laboratories LLC, Mumbai, India). The following genes were targeted: New Delhi metallo-ß-lactamase (blaNDM), Klebsiella pneumoniae carbapenemase (blaKPC), Imipenemase (blaIMP), Verona Integron-encoded MBL (blaVIM), oxacillinase-51 (blaOXA-51), oxacillinase-23 (blaOXA-23), oxacillinase-48(blaOXA-48), and oxacillinase-58(blaOXA-58).

Statistical Method: Data was analysed using SPSS version 26.0 for windows. Chi square test was applied and P value less than 0.05 was considered significant.

RESULT

A total of 209 *Acinetobacter* isolates were collected from various samples. The median age of patients was 49 years (range: 9-86), with a male predominance (63.6%), and the most common age group affected was 48-59 years (25.83%). In terms of hospital distribution, the majority of isolates were from the ICU (54.5%), followed by inpatient departments (27.27%) and outpatient departments (18.18%).

Out of the 209 *Acinetobacter*, most common species identified was *Acinetobacter baumannii* accounting for 187(89.47%) isolates followed by *A. lwoffii* (4.8%), *A. junii* (2.4%), *A. pittii* (1.9%), and *A. johnsonii* (1.4%). Of these 187 *A. baumannii* isolates, 174 (93%) were carbapenems resistant A. *baumannii* (CRAB). Various risk factors which were found to be significantly

associated (*p value <0.05*) with CRAB infection were prior antibiotic use (96.4%, P=0.0001), hospital stays (95.7%, P=0.002), presence of indwelling urinary catheter (95.5%, P=0.0022) and IV catheter use (97.1%, P=0.0001) (Figure 1). Of all the *Acinetobacter* isolates found among various clinical samples, a significant higher number of CRAB isolates were isolated from in respiratory samples (47.7%), followed by blood (32.2%) and urine (9.5%).

The figure 2 shows the resistance patterns of various antibiotics against CRAB isolates (n=174). Trimethoprim-Sulfamethoxazole, Minocycline and colistin had the lowest resistance rates among the tested antibiotics the resistance rate being 85.1%, 77.7% and 17.7 % respectively. Out of 187 *Acinetobacter baumannii* Isolates grown on different samples 174(93%) were carbapenem resistant by Kirby Bauer disk diffusion method. Out of 174 isolates, Disk Diffusion detected Meropenem resistance in 173 isolates (99.4%) and Imipenem resistance was detected in 167 (96.6%) isolates. Since by definition(15), isolates found to be resistant to either Meropenem, Imipenem or both shall be considered as carbapenem resistant *Acinetobacter baumannii* hence 174 isolates were taken up for study.

For the phenotypic detection of MBL-producing strains, the Combined Disk Diffusion Test (CDDT) identified MBL in 35.6% of isolates, while the E-test detected MBL in all (100%) of the isolates (Fig 3). Similarly, the Modified Hodge Test identified carbapenemase production in 26.43% of isolates, whereas the mCIM Test detected carbapenemase production in 56.89% of isolates (Fig 3).

Among 20 CRAB isolates, all isolates were positive for blaOXA51 and blaOXA23 gene.18 were positive for *bla*NDM followed by 08 positive for *bla* IMP and 2 isolates were positive for *bla*VIM gene. *bla*OXA48 and *bla*OXA58 genes were not detected (Fig 4).

Table 1: Risk factors associated with Carbapenem Resistant A. baumannii (CRAB) infections

Risk factors (n)	Non-CRAB n (%)	CRAB isolate n (%)	P value
Immunocompromised (93)	05 (5.4%)	88 (94.6%)	0.40
Prior antibiotic exposure (165)	06 (3.6%)	159 (96.4%)	0.0001
Hospital stay (163)	07 (4.3%)	156 (95.7%)	0.002
Urinary catheter (157)	07 (4.5%)	150 (95.5%)	0.0022
IV catheter (140)	04 (2.9%)	136 (97.1%)	0.0001
Ventilator (73)	02 (2.7%)	71 (97.3%)	0.07
Prosthetic devices in situ (29)	01 (3.4%)	28 (96.6%)	0.420
COPD (49)	0	49 (100%)	0.026
Total (187)	13 (7%)	174 (93%)	

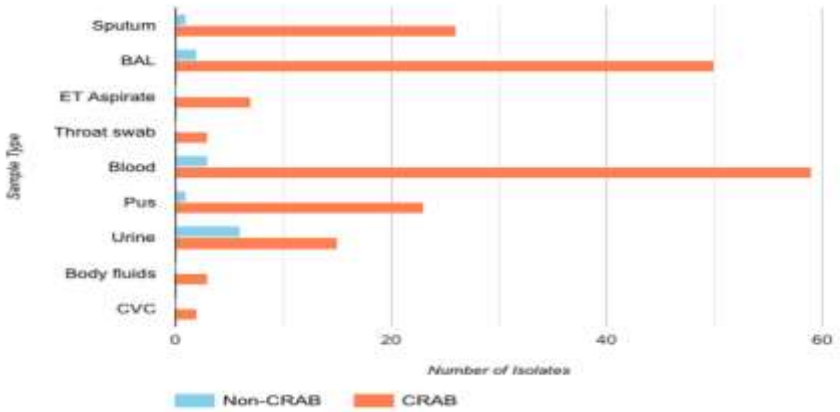

Figure 1: Distribution of CRAB isolates among different samples

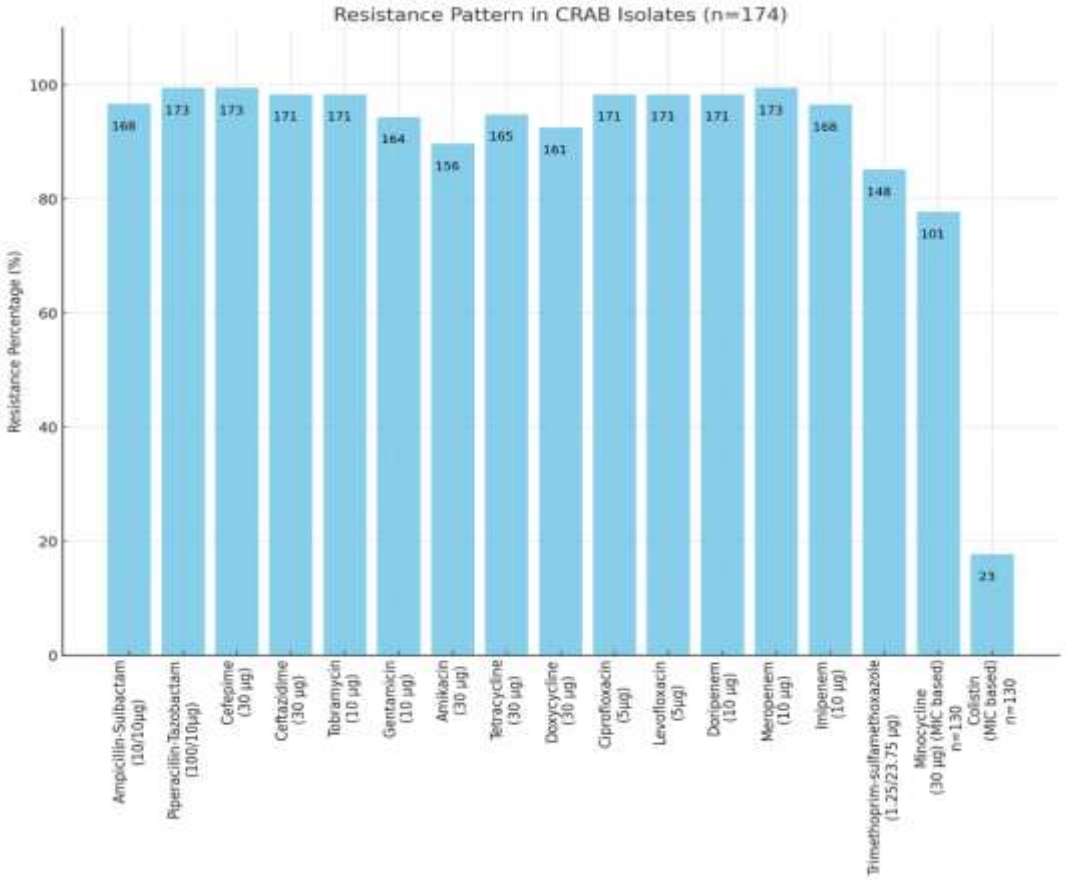

Figure 2: Antimicrobial resistance pattern of CRAB isolates

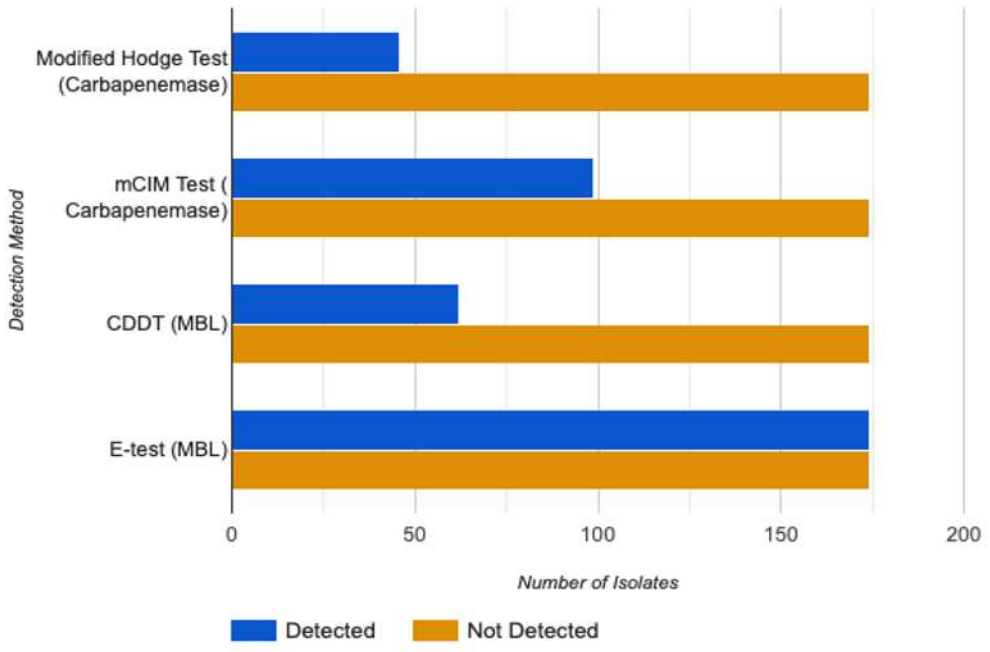

Figure 3: Showing MBL and Carbapenemase production in CRAB isolates

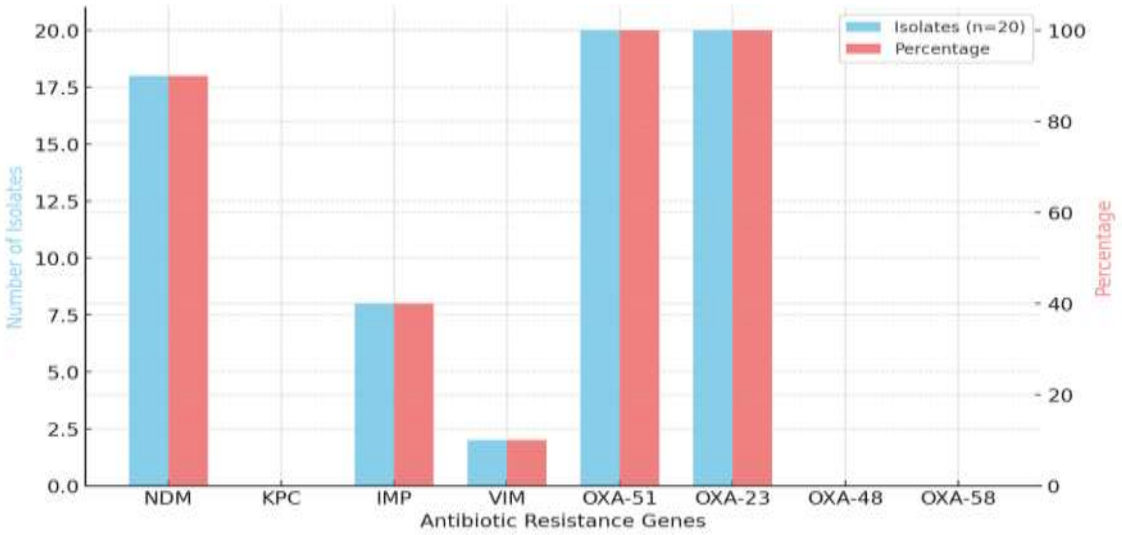

Figure 4: Showing gene distribution in CRAB isolates

DISCUSSION

Antimicrobial resistance specially Carbapenem resistance in Gram-negative bacteria, particularly in pathogen like *Acinetobacter baumannii,* has significant clinical implications. As a significant opportunistic gram-negative bacterium in healthcare settings, *Acinetobacter* infection poses a worldwide health risk due to its ability to withstand a variety of environmental factors, the development of drug resistance mechanisms, and the emergence of multi- and pan-drug resistant strains. In order to reduce patient morbidity and mortality as well as the transmission of resistant strains across the community, it is helpful to isolate and identify the resistance pattern of *Acinetobacter* infections

In the present study out of 209 isolates of *Acinetobacter* species, obtained from different clinical samples, the most common species identified was *Acinetobacter baumannii (89 %)* followed by *A. lwoffii (4.8%) A. junii, A. pittii* and, *A. johnsonii* (2.4%,1.9%, and 1.4% respectively). These findings were similar to a study conducted by Muktikesh Das et al. (2013). They reported that A. baumannii was the main species responsible for 79.6% of the infections, followed by A. lwoffii 12.4% and the remaining 8% were caused by other Acinetobacter species. [17] *A. baumannii* is the most common species identified because it is widespread in environments like water, soil, and sewage, and on skin. It is challenging to treat and eliminate due to its resistance to starvation, desiccation, temperature variations, disinfectants, and its ability to form biofilms.

In this study, 93% of *A. baumannii* isolates were carbapenem-resistant. Yu-Lin Lee et al. (2022) found 71.7% carbapenem resistance among 2674 *A. baumannii* isolates from 13 countries, with carbapenem resistance rates ranging from 2.8% in Japan to 88% in South Korea.[18] Bistro Ingit et al. (2020) reported a lower carbapenem resistance rate of 21.5%[19], while Ekadashi Rajni et al. (2024) found 96.9% carbapenem resistance among 228 isolates.[20] Ramette et al. (2018) noted 88% carbapenem sensitivity, contrasting with global studies showing 40% to 85% carbapenem resistance. [21] These variations highlight the need for conducting region/hospital specific research into the epidemiology of invasive *A. baumannii* infections. The increased resistance observed in this study can be attributed to several factors, including the overuse of carbapenems in the healthcare facility, also the genetic diversity of *A. baumannii* strains and their ability to acquire resistance through horizontal gene transfer also contribute to the high rates of carbapenem resistance.

In this study, among 174 CRAB isolates detected by various methods, the majority were from respiratory samples. Similarly, Rajani E et al. (2024) found CRAB isolates

most commonly in ET secretions.[20], also Ramette et al. (2018) reported high carbapenem resistance in *A. baumannii* isolated from respiratory samples. [21] These findings suggest the lungs are the most commonly involved organ, likely due to colonization of airways and respiratory equipment used for mechanical ventilation.

In this study, testing for antibiotic resistance in *A. baumannii* isolates revealed a concerning degree of resistance to several widely prescribed antimicrobials, with more than 90 % of isolates showing resistance to carbapenems and third-generation cephalosporins and fluoroquinolones. Their extensive use has led to an increased incidence of carbapenem resistance in recent years Colistin is an essential medication, demonstrated a high degree of susceptibility (82.2%) in CRAB isolates in our study. About ten years ago, colistin resistance was around zero in studies; nevertheless, in recent times, resistance levels have risen to 36% [22]. In the present study, 23% of isolates showed susceptibility to minocycline. Similarly, Yang JL et al. (2022) found that 55% of CRAB strains were susceptible to minocycline.[23]

Carbapenemase production, especially by Ambler class D β-lactamases and class B metallo-β-lactamases (MBLs), poses a significant challenge in treating bacterial infections. β-lactamase inhibitors are largely ineffective against class D β-lactamases, Phenotypic detection methods for carbapenemases, while cost-effective and simple, suffer from poor specificity and sensitivity. For instance, the modified Hodge test (MHT) has shown varying accuracy, with some studies reporting high sensitivity, while others found it unreliable for detecting carbapenemase production in *Acinetobacter baumannii.* CLSI does not endorse any specific phenotypic method for detecting carbapenemase in *Acinetobacter,* but EUCAST recommends the combination disk (CD) test despite its limitations. In contrast, the modified carbapenem inactivation method (mCIM) is preferred for its reliability and affordability, though it is not recommended for *A. baumannii.* Our study found mCIM identified 56.89% of carbapenemase producers, while MHT showed only 23.5% sensitivity.

In the present study, 62 isolates (35.6%) tested positive for metallo-β-lactamase (MBL) production by the combined disk diffusion test (CDDT), while 100% tested positive by the E-test. Despite CDDT being a reliable method for detecting Ambler class B MBLs, our study reported the lowest positivity rate, consistent with findings by Shivaprasad et al. [24] The E-test demonstrated 100% sensitivity, similar to the observation from studies by Khosravi et al and Walsh et al. [25,26]

In our study, all strains exhibited the chromosomally encoded *bla* $_{OXA-51}$ gene, aligning with previous research that uses this gene for species-level identification. Also, all the isolates had the *bla* $_{OXA-23}$ gene, the primary carbapenem resistance mechanism in multidrug-resistant strains was the expression of *bla* $_{OXA-23}$, which has been widely associated with outbreaks since its identification in 1985 and is prevalent in Asia. While *bla* $_{OXA-23}$ is the main carbapenemase, *bla* $_{OXA-48}$ and *bla* $_{OXA-58}$ can also contribute to hospital epidemics these where not found in any CRAB isolates. For class B metallo-β-lactamases, *bla*$_{NDM,}$ *bla* $_{IMP}$, and *bla* $_{VIM}$ were present in 90%, 40%, and 10% of isolates, respectively. A previous study by Fallah et al. reported that the *bla* $_{IMP}$, and *bla* $_{VIM}$ genes were present in 17% and 4% of *A. baumannii* isolates respectively. [27] Because of its ability to disseminate rapidly, carbapenem resistance related and NDM β-lactamase production has become a serious concern as NDMs has been increasingly reported from India.

CONCLUSION

This study found a very high carbapenem-resistant in *Acinetobacter baumannii* isolates. The findings show that two genes, *bla*$_{OXA-51}$ and *bla* $_{OXA-23,}$ are particularly important in causing carbapenem resistance in CRAB.

Despite high level of resistance to multiple antibiotics colistin and minocycline showed relatively higher sensitivity against CRAB isolates, suggesting their potential role as alternative treatment options. Further research is needed to optimize their use and to address the potential for emerging resistance to these antibiotics.

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