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ORIGINAL ARTICLE

Detection of carbapenemase production in Enterobacterales by mCIM and eCIM: a tertiary care hospital study

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ABSTRACT

Background and Objectives: Carbapenem-resistant Enterobacterales (CRE) pose a major healthcare challenge due to high resistance rates and limited treatment options. This study characterized carbapenemase production among CRE isolates using phenotypic methods—Modified Carbapenem Inactivation Method (mCIM) and EDTA-Carbapenem Inactivation Method (eCIM)—as genotypic methods have limitations like restricted gene targets and mutations.

Materials and Methods: This six-month study was conducted at Sher-i-Kashmir Institute of Medical Sciences (SKIMS). Samples including swabs, respiratory specimens, pus, body fluids, and blood were cultured on Blood Agar and MacConkey Agar (HiMedia, India). Enterobacterales were identified using conventional methods and screened for carbapenem resistance. CRE isolates underwent mCIM and eCIM testing per CLSI guidelines.

Results: Among 471 Enterobacterales isolates tested, 160 (33.9%) were carbapenem-resistant. Of these, 97 (60.6%) were mCIM positive, indicating carbapenemase production. eCIM further identified 83 (85.5%) as metallo-beta-lactamase (MBL) producers and 14 (14.4%) as serine carbapenemase producers. CRE prevalence was higher in ICU settings and among males. Isolates showed high cephalosporin resistance, with multi-drug resistance (MDR) common in both MBL and serine carbapenemase producers.

Conclusion: The prevalence of CRE was found to be 33.9%. The findings underscore the critical need for continuous surveillance and stringent infection control measures to manage the spread of CRE in healthcare settings.

Keywords: Carbapenem antibiotics; Carbapenemases; Beta-lactamases; Metallo-beta-lactamase; Microbial sensitivity tests

INTRODUCTION

Enterobacterales are Gram-negative, facultatively anaerobic Gram-negative rods responsible for causing a large number of nosocomial infections that are usually difficult to treat due to the high antimicrobial resistance among clinical isolates (1). As the incidence of MDR Gram-negative bacteria has increased worldwide, carbapenems have been considered as the last choice antibiotic for their treatment. Subsequently, the enterobacterales developed resistance to carbapenems, thus giving rise to the development of carbapenem-resistant (CR) Enterobacteriaceae, which in turn poses a challenge due to non-availability of noticeable next line of antibiotics for treatment of these kinds of antibiotic-resistant organisms (2). A report published by the European Centre for Disease Prevention and Control (ECDC) and the World

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Health Organization (WHO) revealed that there was an increase in prevalence of carbapenem resistance in *Klebsiella pneumoniae* isolates by 0, 8, 31, and 20% between 2017 and 2021 (3).

In India, a study found the overall prevalence of CRE, *A. baumannii*, and *P. aeruginosa* to be 29.40% whereas another study revealed the presence of carbapenem resistant Gram-negative bacilli to be 33.12% (4, 5). Production of carbapenemases is the most common cause of resistance to carbapenems. Carbapenemases are a class of enzymes capable of hydrolyzing carbapenems and other β-lactams (6). Other causes may also contribute to carbapenem resistance which include, poor binding of carbapenems to penicillin-binding proteins present in the bacteria, over-expression of multidrug efflux pumps by the bacteria or lack of porins present in the bacterial cell membrane. Generally, for the emergence of significant resistance, a combination of resistance mechanisms is required. Carbapenemase production is currently the most concerning resistance mechanism and their genes are often contained on mobile genetic elements that facilitate transfer of resistance among Enterobacteriaceae and other gram-negative organisms. Because of the rapid spread of Carbapenemase-producing Enterobacterales (CPE), these organisms are a particularly important target for prevention (7).

In most of the clinical microbiology laboratories, the underlying mechanisms for carbapenem resistance are not evaluated for therapeutic decision-making. At the same time, understanding if an isolate is CPE has significant role to play in the development of Institutional treatment guidelines, Infection prevention procedures and Epidemiological investigations (8). While carbapenemase genes can be detected by using genotypic tests, detection of mutated or new carbapenemase producing genes is not possible. Also, the high costs and requirement of special expertise limits their use (9). The first Clinical & Laboratory Standards Institute (CLSI) recommended growth-based carbapenemase detection test is Modified Hodge test (MHT). However, currently accurate identification of carbapenemases is done by modified carbapenem inactivation method (mCIM) and Ethylenediaminetetraacetic Acid (EDTA)- Carbapenem Inactivation Method (eCIM) endorsed in the CLSI M100-S28 supplement in 2018 (1). As the incidence of CRE in hospitals is increasing, owing to their ability to colonize the gastrointestinal tract after direct (person-to-person) or indirect (via contaminated surfaces) transmission, an hour is needed to have an accurate and robust routine protocol for CRE screening and detection of carbapenemases in them in every hospital. To establish correct and targeted treatment plans for patients, detection of CRE is necessary (6, 10).

This study aims to characterize the types of carbapenemases produced by Gram-negative isolates using phenotypic methods, specifically the modified carbapenem inactivation method (mCIM) and the EDTA-modified carbapenem inactivation method (eCIM). We hypothesize that the combined use of mCIM and eCIM will effectively differentiate metallo-β-lactamases (MBLs) from serine carbapenemases.

MATERIALS AND METHODS

Study design. This prospective study was carried out in the department of microbiology at the Sheri-Kashmir Institute of Medical Sciences (SKIMS) for six months (Jan 2022-June 2022) after obtaining Ethical clearance from the Institute's Ethical clearance committee under IEC Protocol number #RP 129/2022.

Sample processing. Samples including swabs, respiratory samples, pus, body fluids, blood received in the Department of microbiology underwent inoculation on Sheep Blood Agar plates comprising a blood agar base (HiMedia, India) supplemented with 5% sheep blood and MacConkey agar (HiMedia, India). The culture plates were placed in bacteriological incubators and incubated at a temperature of 35°C ± 2°C for 24 h.

Following preliminary identification tests, such as Gram staining, the isolates were manually identified as Enterobacterales using conventional biochemical tests. The employed test panels included spot catalase test, spot oxidase test, carbohydrate fermentation tests, indole test, Methyl Red, Phenyl Pyruvate, Triple Sugar Iron agar (TSI), Citrate utilization test, and Urea hydrolysis test. Antimicrobial susceptibility testing was performed for all the organisms belonging to Enterobacterales according to CLSI guidelines using the Kirby-Bauer disc diffusion method for all samples except blood samples for which microbroth dilution method by Vitek 2 automated system in case

of blood samples was used (11). The samples that fulfilled the following inclusion and exclusion criteria were included and excluded from the study respectively.

Inclusion criteria. Enterobacterales that had a meropenem zone diameter ≤19mm in case of respiratory samples, pus, body fluids and other samples and MIC≥ 4 µg /ml in case of blood were considered screen test positive and were included in the study.

Exclusion criteria. Isolates that showed meropenem zone diameter of ≥ 23mm in case of respiratory samples, pus, body fluids and other samples and MIC≤1 µg /ml in case of blood were considered sensitive and were excluded from the study.

These were further checked for carbapenemase production by m-CIM and e-CIM). m-CIM was performed for detection of carbapenemases in Enterobacterales whereas eCIM was used together with mCIM to differentiate metallo-β-lactamases from serine carbapenemases in Enterobacterales and was considered valid only if mCIM was positive (9).

mCIM test. In mCIM test, the growth from the culture plate was streak on the MHA plate and kept for overnight incubation at 37°C. One loopful (10µl) of overnight growth was emulsified in 2ml of trypticase soy broth (TSB) and vortexed for 10-15 seconds. A meropenem disk (10µg) was added to it using sterile forceps and ensured the disk was completely immersed in it. The broth was then incubated at 37°C for 4 hours following which a lawn culture of 0.5 McFarland adjusted culture of *Escherichia coli* ATCC 25922 onto Muller Hinton agar plate was made. Then the meropenem disk was removed from the suspension and placed it on the lawn culture of *E. coli* ATCC 25922. Then the plates were incubated at 37°C for 18-24 hours.

Interpretation of mCIM test. Following the incubation, zone of inhibition was measured and interpreted. Isolates were considered carbapenemase positive if the zone diameter was 6-15 mm or there was a presence of pinpoint colonies within a 16-18 mm zone. The isolates were considered as carbapenemase negative if the zone of inhibition was ≥ 19mm. An isolate was considered as carbapenemase indeterminate when the zone of inhibition was16-18mm or ≥ 19mm and there was a presence of pinpoint colonies within the zone.

eCIM test. All the isolates were tested by eCIM as well, and processing was done in parallel with mCIM tubes (Fig. 2). For the e-CIM test, a second 2 ml TSB was labeled. Twenty microlitre of the 0.5M EDTA was added to this TSB tube to obtain a final concentration of 5mM EDTA and vortexed for 10-15 seconds. A 10 µg meropenem disk was added to the suspension using sterile forceps and ensuring the disk was immersed in the suspension. It was then incubated at 37°C for 4 hours. After this we made a lawn culture of 0.5 McFarland adjusted culture of *E. coli* ATCC 25922 onto Muller Hinton agar plate. Then the meropenem disk was removed from the suspension and placed it on the lawn culture of *E. coli* ATCC 25922. Subsequently, the plates were incubated at 37°C for 18-24 hours. Following the incubation, the zone of inhibition was measured and interpreted.

Interpretation of eCIM test. Only the isolates positive for mCIM test were considered for eCIM interpretation. A ≥ 5mm increase in zone diameter for eCIM vs zone diameter for mCIM was considered as eCIM positive. Pinpoint colonies within any inhibition zone were ignored in this test. A ≤ 4-mm increase in zone diameter for the e-CIM vs zone diameter of m-CIM was considered negative for eCIM test. If the test isolate produces a metallo-β-lactamase, the activity of the carbapenemase will be inhibited in the presence of EDTA, and the meropenem disk will not be hydrolyzed as effectively as in the tube without EDTA. The result is inhibition of the meropenem-susceptible *E. coli* and an increase in the zone diameter. If the test isolate produces a serine carbapenemase, the activity of the carbapenemase will not be affected by the presence of EDTA, and there will be no or marginal (≤ 4 mm) increase in zone diameter in the presence of EDTA compared to the m-CIM zone diameter (Fig. 1).

RESULTS

Out of the 471 Enterobacterales isolated in our laboratory during the study period, 160 isolates were carbapenem-resistant and thus subjected to mCIM test. Out of 160 CRE isolates tested for mCIM, 97 showed positive results for the carbapenemase enzyme. On further characterization of carbapenemases by eCIM, 83 (85.5%) were positive by eCIM, indicating the presence of metallo-carbapenemases in them and rest

Fig. 1. Lawn culture plate of ATCC strain of *E. coli* inoculated with antibiotic disks from eCIM and mCIM broth

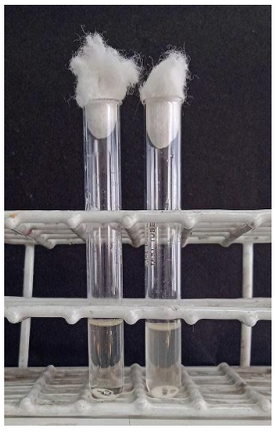

Fig. 2. Left to right: The mCIM tube shows turbidity, indicating carbapenemase activity.

of the isolates 14 (14.4%) in which mCIM was positive and eCIM negative had serine carbapenemases. Among the mCIM-positive isolates, 88.8% belonged to male patients and majority of them, 87.5% were received from ICU (Table 1). The highest eCIM positivity was observed from blood samples (100%) (Table 2). 100% (n=4) *Proteus* spp., 90.4% (n=38) *E. coli* and 80.3% (n=41) *Klebsiella* spp. were positive for eCIM test (Table 3). In general, 98.9% (n=95) and 2.1% (n=2) carbapenemase-producing strains were resistant to at least three classes of antimicrobials and were considered multidrug-resistant (MDR) and classified as resistant to all but two or less classes of antimicrobials extensive drug-resistant (XDR), respectively (12).

MDR phenotype resistant to a β-lactams-β-lactamase inhibitor combination, cephalosporins and carbapenems was the most common (Figs. 3, 4 and Table 4).

DISCUSSION

CRE pose a serious threat to healthcare and one of their essential mechanisms of resistance is the production of carbapenemases. Carbapenemase distribution varies by region and organism. In Enterobacterales, KPC-producing *K. pneumoniae* is endemic in the USA, Israel, Greece, and Italy. OXA-48-like enzymes are hyperendemic in Turkey, North Africa, and parts of Europe, with OXA-232 emerging in Asia and Turkey. NDMs, initially linked to the Indian subcontinent, are now globally widespread. IMP producers, first seen in Japan, are now common in Australia, while VIM producers are frequent in Greece, Italy, the Balkans, and Russia. Co-production of NDM and OXA-48 is rising globally, especially in Turkey and the Balkans (13). Xaio et al., identified two Novel Carbapenemase-Encoding Hybrid Plasmids Harboring bla_{NDM-5} and bla_{KPC-2} in a Clinical ST11-KL47 *K. pneumoniae* in 2024 (14).

The acquisition of infection with the CPE is associated with high mortality and increased length of hospital stay. The problem is further accentuated by their rapid spread and the availability of only a few antimicrobial agents that can be used to treat such infections. This is bound to rising concerns about the treatment options and infection control (15). This study employed two phenotypic tests recommended by CLSI—the Modified Carbapenem Inactivation Method (mCIM) and the EDTA-modified Carbapenem Inactivation Method (eCIM)—to evaluate carbapenemase production and differentiate metallo-beta-lactamases (MBLs) from serine carbapenemases. In a study conducted by Tsai K. et al., the sensitivity of the mCIM test for detecting MBLs was reported to be 100%, and mCIM was found to be more accurate compared to the Modified Hodge Test (MHT), which was the first CLSI-recommended growth-based carbapenemase detection method introduced in 2009 (1).

Earlier studies also demonstrated that the sensitivity and specificity of eCIM were both 100% when used with 5 mM EDTA (16).

In another study, carbapenem resistance was observed in 75% of *K. pneumoniae* isolates and 50% of *E. coli* isolates. Among these, 58.4% were metallo-β-lactamase (MBL) producers, while 41.6% produced serine carbapenemases. The study concluded that combining mCIM and eCIM could serve as an effective epidemiological tool, assist in selecting

Table 1. Gender and location-wise distribution of eCIM positive and eCIM negative isolates

Gender	mCIM positive (n)	eCIM positive n (%)	eCIM negative n (%)	P-value
Male	61	51 (83.6)	10 (16.4)	0.51
Female	36	32 (88.8)	4 (11.2)	
		Location		
In-patient department	81	69 (85.2)	12 (14.8)	1.0
Intensive care unit	16	14 (87.5)	2 (12.5)	

Table 2. Sample-wise distribution of eCIM positive and eCIM negative isolates

Specimen	mCIM positive (n)	eCIM positive n (%)	eCIM negative n (%)
Respiratory samples	14	11 (78.5)	3 (21.5)
Blood	5	5 (100)	0 (0)
Body fluids	10	8 (80)	2 (20)
Pus	13	10 (76.9)	3 (23.1)
Swab	51	46 (90.1)	5 (9.9)
Others	4	3 (80.75)	1
Total	97	85	14

Table 3. Organism-wise distribution of eCIM positive and eCIM negative isolates

Organism	mCIM positive (n)	eCIM positive n (%)	eCIM negative n (%)
Klebsiella spp.	51	41 (80.3)	10 (19.7)
E. coli	42	38 (90.4)	4 (9.6)
Proteus spp.	4	4 (100)	0 (0)
Total	97	85	14

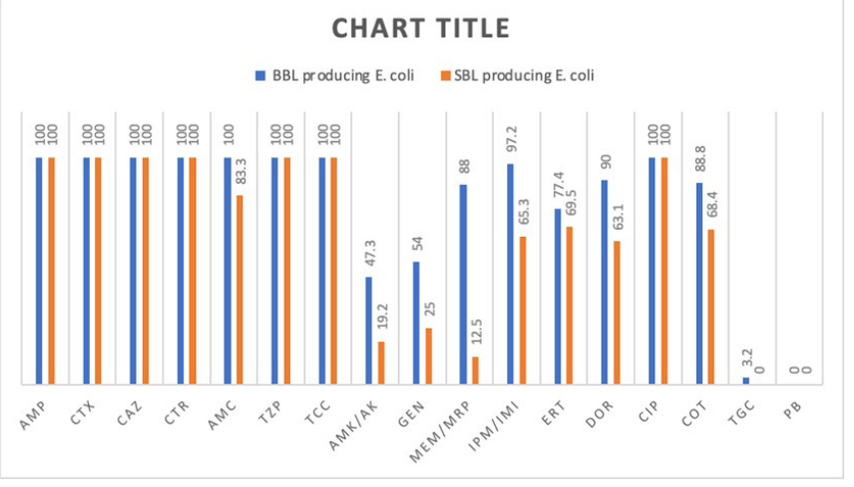

Fig. 3. Resistance profile of metallo-beta lactamase producing Enterobacterales.
Note: AMP=Ampicillin CTX=Cefotaxime CAZ=Ceftazidime CTR=Ceftriaxone AMC=Amoxicillin+Clavulanate TZP=Piperacillin-Tazobactam TCC=Ticarcillin+clavulanate AMK=Amikacin GEN=Gentamicin MEM=Meropenem IPM=Imipenem ERT=Ertapenem DOR=Doripenem CIP=Ciprofloxacin COT= Co-trimoxazole TGC=Tigecycline PB=Polymyxin B

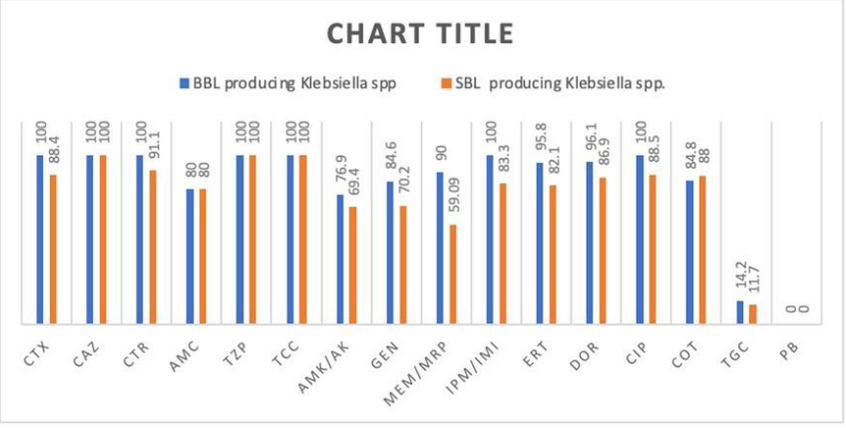

Fig. 4. Resistance profile of serine-beta lactamase producing Enterobacterales.
Note: AMP=Ampicillin CTX=Cefotaxime CAZ=Ceftazidime CTR=Ceftriaxone AMC=Amoxicillin+Clavulanate TZP=Piperacillin-Tazobactam TCC=Ticarcillin+clavulanate AMK=Amikacin GEN=Gentamicin MEM=Meropenem IPM=Imipenem ERT=Ertapenem DOR=Doripenem CIP=Ciprofloxacin COT= Co-trimoxazole TGC=Tigecycline PB=Polymyxin B

Table 4. Distribution of MDR and XDR patterns among carbapenemase producing Enterobacterales.

Bacteria	Resistance profile	Most common phenotypic resistance pattern	Number of isolates
E. coli N=42	MDR	β-lactams-β-lactamase inhibitor combinations: Ticarcillin +clavulanate Cephalosporins: ceftazidime, ceftriaxone, Cefotaxime Carbapenems:Imipenem β-lactams-β-lactamase inhibitor combinations: Ampicillin+sulbactam	21
Klebsiella spp. N=51	MDR	Cephalosporins: ceftazidime, ceftriaxone, Cefotaxime Carbapenems:Imipenem β-lactams-β-lactamase inhibitor combinations: Ticarcillin+clavulanate	25
	XDR	Cephalosporins: ceftazidime, ceftriaxone, Cefotaxime Carbapenems:Imipenem Aminoglycosides: Gentamicin Quinolones: Ciprofloxacin Sulfonamides : Cotrimoxazole	1
Proteus spp. N=4	MDR	β-lactams-β-lactamase inhibitor combinations: Ticarcillin+clavulanate Cephalosporins: ceftazidime, ceftriaxone, Cefotaxime Carbapenems:Imipenem β-lactams-β-lactamase inhibitor combinations: Amoxicillin+clavulanate	2
	XDR	Cephalosporins: ceftazidime, ceftriaxone, Cefotaxime Carbapenems:Imipenem Aminoglycosides: Gentamicin Quinolones: Ciprofloxacin Sulfonamides: Cotrimoxazole	1

initial antibiotic therapy, help reduce morbidity and mortality associated with ventilator-associated respiratory infections (VARIs), and strengthen hospital infection control practices (17).

Verma G. et al. further highlighted that the inexpensive combination of mCIM and eCIM not only differentiates between serine carbapenemase and MBL producers but also guides appropriate therapy decisions and supports infection control, particularly in resource-limited settings (18).

The prevalence of carbapenem resistant enterobacterales has been found to be 33.9% in our hospital, a 1750 bedded tertiary care hospital in north India. [unpublished data] Previous studies from India have

reported a prevalence of 13- 51% (19, 20). High prevalence in our hospital may be attributed to the fact that it is a tertiary care hospital where most patients admitted are terminally sick with multiple comorbid conditions and are referred after already receiving antimicrobials and are more likely to undergo invasive procedures contributing to longer hospital stay and acquiring CRE infection in the hospital.

This study demonstrates that among carbapenem resistant isolates (n=160), carbapenemase production was the mechanism of resistance in 60.6% (n=97) of the isolates which was indicated by mCIM positivity. Among these isolates eCIM was positive in 85 isolates (87.6%) indicating presence of MBL and serine carbapenemases in rest of the isolates 12.4% (n=12) as a cause of carbapenem resistance. Carbapenemase mediated resistance is a serious cause for concern especially in critically ill patients (21). The implementation of a MBL and serine carbapenemases detection methods particularly where carbapenem and other β-lactam therapeutic regimens are indicated or preferred is important in the era of emergence of carbapenemase production in a wide range of clinically significant bacteria for effective treatment (22). In a study by VK Sreeja et al., out of the 220 isolates, 207 (94.0%) had phenotypic positivity of carbapenemase production, out of which 189 (91.2%) were MBL producers. These results are comparable to our study (23). Diwakar et al., also reported the comparable prevalence to that of our study (24). However, the minor difference may be due to the differences in the geographic region (25, 26).

The distribution of carbapenemase-producing Enterobacterales (CPE) was not restricted to a particular hospital unit. The percentage of CPE obtained from IPD and ICU was found to be 85.1% and 87.5% respectively, thus, suggesting the presence of CPE distributed in the whole hospital wards rather than a particular ward or area. Similar findings were shown by Nair PK et al., from a tertiary care hospital in Mumbai, where CRE was isolated from hospital wards (42%) and ICU (26%) and OPD patient samples (19%) (27). It also means that CPE should no longer be considered as a problem in ICUs only in our hospital. The high percentage of CPE from ICUs indicates colonization and dissemination of the resistant strains carrying genes on plasmids/transposons within wards and clustering of such strains in critical patients in ICUs. Robert et al., found that ICU had the largest burden of carbapenemase producing isolates (34.6%), followed by surgery ward (30.8%) (28). Almost similar findings were shown by Apurva Parate et al., in which the carbapenemase positive isolates were predominantly isolated from Burns wards (14.61%) and ICUs (13.33%) followed by medicine wards (12.26%) (22). Nagaraj et al., in 2012 isolated most carbapenemase producing isolates from general surgery, general medicine, and ICU (29). Inadequate infection control practices result in wound infection in the hospital by resistant strains, prolonged hospital stay and prolonged antibiotic treatment in such patients which may be the reason for increased prevalence of CPE in swabs and blood specimens.

In the present study wound swab was the leading specimen from which CPE were isolated. However, Parate A et al., found that the highest number of carbapenemase producing isolates were obtained from blood (23.53%) and pus (11.11%) (22). Sood et al., also found carbapenemase producing organisms from blood (25%) which was less as compared to our study (30). In a study conducted by Nair et al., urine was the leading specimen contributing carbapenemase producing isolates which was not included in our study (27).

Majority of mCIM positive isolates were *K. pneumoniae* (n=51) followed by *E. coli* (n=42) and *Proteus mirabilis* (n=4). Lorenzoni et al., found that majority of strains responsible for carbapenemase production were that of *K. pneumoniae* (31). As a matter of fact, *K. pneumoniae* and *E. coli* are a part of gut normal flora. So, carbapenemase producing *K. pneumoniae* and *E. coli* are capable of colonizing the gut of patients which in turn serve as reservoirs for spreading infection or contaminating the environment and fomites, especially in healthcare settings. To control the spread of such organisms, screening as well as disinfection measures need to be followed and also there is a need for contact isolation of these infected/ colonized patients. This protocol is not routine and may not be feasible in all healthcare institutions and it is only important in case such organisms are detected in a patient. In addition, appropriate use of carbapenems will also prevent selecting resistant bacteria in a given geographical area (25).

The results of the current study show that there was a slight difference in the antibiotic susceptibility patterns of metallo-beta lactamase (MBL) producing and serine beta lactamase (SBL) producing Enterobacterales. Both MBL producing and SBL producing isolates demonstrated a high resistance level for pen-

icillin as well as 1st and 3rd generation cephalosporins and quinolones. A study by Apurva Parate et al., showed that carbapenemase producing isolates were 100% resistant to ampicillin, amoxiclav, cefuroxime and imipenem and also were highly resistant to 3rd and 4th generation cephalosporins i.e. cefotaxime, and ceftazidime (91.18%) and cefipime (88.24%). 23 (67.65%) of the isolates were susceptible to amikacin, which is comparable to this study (31). The resistance rates for imipenem, meropenem, ertapenem and doripenem were 60.7%, 66.3%, 86.1% and 95.3% for metallo beta lactamase producers, and 74.31%, 35.7%, 75.8% and 75% respectively for serine beta lactamase producers. These findings indicate that some isolates appear sensitive to carbapenems by disk diffusion method, have the capacity to produce carbapenemase that may resist antibiotic treatment in vivo leading to treatment failure. Also, Similar to our study, Fattouh et al., and Chakraborty et al., reported 78.25% and 59.5% resistance in case of carbapenemase producing organisms towards Imipenem (32, 33). Of interest, a high incidence of MDR and XDR and PDR profiles was observed among all carbapenemase-producing isolates. 95 isolates (97.9%) of the Gram-negative bacteria exhibited MDR pattern of resistance and 2 (2.1%) of the Gram-negative bacteria exhibited XDR pattern of resistance. The higher incidence of MDR patterns among Enterobacterales may be due to increased selection pressure from self-medication, empirical use, indiscriminate overuse of carbapenems and third-generation cephalosporins, as well as insufficient monitoring of resident MDR isolates in hospital settings, especially in the surgery ward, ICU, and burn unit (34). MDR and XDR isolates of these bacteria are increasingly being reported globally, but according to our results, higher rates were observed than those previously reported.

Limitations of this study.

1. The six-month period may not account for seasonal variations in the prevalence of CREs, potentially affecting the generalizability of the results.

2. As the study was conducted at a single centre, the findings may not be representative of other regions or healthcare settings with different patient populations and microbial environments.

3. Phenotypic tests for identifying carbapenemase producers was not evaluated against a gold standard test like PCR targeting carbapenemase.

4. Actual prevalence may be higher than the present study as molecular detection methods with greater sensitivity were not adopted.

CONCLUSION

This study highlights the significant presence of carbapenem-resistant Enterobacterales (CREs) in clinical samples, with a notable prevalence of metallo-β-lactamases and serine carbapenemases. The use of mCIM and eCIM effectively characterized these isolates, revealing that 87.6% were positive for metallo-β-lactamases, while 12.4% harbored serine carbapenemases. The study underscores the importance of precise detection methods for informing antimicrobial treatment strategies and controlling the spread of resistant pathogens.

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