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Efficacy of the modified carbapenem inactivation method (mCIM) for detecting carbapenemase-producing *Pseudomonas aeruginosa* in a 1,000-bed tertiary hospital in Thailand

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ABSTRACT

Background: Carbapenem-resistant *Pseudomonas aeruginosa* (CRPA) is a prominent healthcare-associated pathogen with worldwide implications for public health. Distinguishing between carbapenemase-producing and non-carbapenemase-producing strains over an extended hospitalization period is vital in assessing the risk of treatment failure in infected patients.

Objectives: To study the prevalence of carbapenemase genes and to evaluate the efficacy of the modified carbapenem inactivation method (mCIM) for detecting carbapenemase production among CRPA isolated from Thailand.

Materials and methods: A total of 164 CRPA isolates were phenotypically tested for carbapenemase production by the mCIM comparing with genotypic detection of carbapenemase genes using the direct flow chip kits and conventional PCR. In addition, carbapenemase genes were identified by sequencing.

Results: The carbapenemase genes, including bla_{IMP}, bla_{VIM}, bla_{NDM}, bla_{GES}, and $bla_{SIM,}$ were found in 83 (50.61%) isolates. The bla_{IMP} showed to be the most prevalent gene, followed by bla_{VIM} and bla_{NDM}, respectively. Of these isolates, 68/83 (82%) of carbapenemase gene-positive isolates were positive by the mCIM assay. The 15 mCIM-negative isolates, six (40%) carried bla_{VIM-2} gene, while five (33%) isolates contained bla_{GES-5}. Moreover, bla_{IMP} was detected in 3 isolates (20%) and one isolate (7%) of a combination between bla_{VIM} and bla_{IMP} genes. Furthermore, we noted that isolates carrying bla_{VIM-2} were the most common among the mCIM-negative carbapenemase-producing *P. aeruginosa* (CPPA), followed by $bla_{GES-5.}$

Conclusion: This study represents a critical report on using mCIM to detect CPPA. It highlights that relying solely on mCIM may not be sufficient for the comprehensive detection of CPPA. Further approaches are needed to enhance the accuracy of detection in this context.

Introduction

Carbapenem-resistant *Pseudomonas aeruginosa* (CRPA) is a significant causative agent of healthcare-associated infection worldwide.[1] Several pivotal mechanisms underlie carbapenem resistance. The primary mechanism involves drug efflux, which occurs due to the over-expression of the MexAB-OprM efflux pump,[2] the overproduction of AmpC beta-lactamase and the inactivation of the OprD outer membrane protein, and carbapenemase production. Although the

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latter mechanism is relatively less frequent among *P. aeruginosa* isolates, the emergence of carbapenemase production is showing an increasing trend, adding a new dimension to carbapenem resistance mechanisms.[1] This mechanism of carbapenem resistance assumes paramount importance as it significantly compromises the effectiveness of widely employed antipseudomonal agents, including ceftazidime, cefepime, piperacillin-tazobactam, along with the more recently introduced beta-lactam/beta-lactamase inhibitor combinations, such as ceftolozane-tazobactam, imipenem-relebactam, and ceftazidime-avibactam. Notably, the carbapenem resistance determinants carried by *P. aeruginosa* are frequently encoded on various mobile genetic elements, including plasmids, such as the *IncP* type, class I integrons housing the bla_{VIM} gene, and other mobile genetic elements, particularly insertion sequences with a common region (ISCRs). These genetic elements enhance the capacity to disseminate resistance across multiple species, underscoring the significance of this mechanism in the context of antimicrobial resistance.[2] Furthermore, these isolates often harbor supplementary resistance determinants that reduce the clinical effectiveness of fluoroquinolones and aminoglycosides.

Carbapenemase-producing *P. aeruginosa* (CPPA) frequently resists most of the therapeutic options, elevating the risk of treatment failure. Additionally, CPPA has been linked to nosocomial transmission, necessitating the implementation of infection prevention measures.[1] *Pseudomonas aeruginosa* isolates have been documented to harbor a diverse array of carbapenemases on a global scale. In Latin America, this spectrum encompasses carbapenemases such as *Klebsiella pneumoniae* carbapenemase (KPC), Guiana extended-spectrum (GES), Imipenemase (IMP), Verona integron-encoded metallo-β-lactamase (VIM), New Delhi metallo-beta-lactamase (NDM), and Sao Paolo metallo-beta-lactamase (SPM).[3] A notable percentage of CRPA isolates in various regions, ranging from 30 to 69%, possessed carbapenemases. In contrast, only 2% of CRPA isolates originating from the USA were found to carry carbapenemases, with the highest incidence recorded in South and Central America. Globally, bla_{KPC-2} emerged as the most prevalent carbapenemase, closely followed by bla_{VIM-2}, bla_{IMP-1}, bla_{NDM-1}, and bla_{GES-5}. However, it is worth noting that different regions exhibited distinctive predominant carbapenemases.[4]

In 2022, a study conducted in South Korea involving patients with urinary tract infections caused by *P. aeruginosa* demonstrated the presence of high-risk clones, specifically ST235 with bla_{IMP-6} and ST773 with bla_{NDM-1}. The study revealed a carbapenem resistance rate of approximately 46.7% among the isolated *P. aeruginosa* strains, exhibiting a multidrug-resistant (MDR) phenotype, and most CPPA isolates carried carbapenemase genes, such as bla_{IMP-6} or bla_{NDM-1}.[5] Another report in the same year using 807 CRPA isolates collected from 17 centers across 12 countries between 2019 and 2021, demonstrated approximately 33% exhibited phenotypic positivity for carbapenemases when tested using the modified Carbapenem Inactivation Method (mCIM). Among these phenotypically positive cases, 86% were genotypically positive, with VIM being the most prevalent carbapenemase, followed by GES. While carbapenemase producers were expected in Middle Eastern centers based on previous epidemiological data, a surprisingly high prevalence and diversity were also observed in European, South American, and African centers. In contrast, the CPPA in the United States was identified in between 3 to 30% of CRPA cases.[1] These contemporary global findings underscore the significance of carbapenemase testing in CRPA cases. The fundamental question arises regarding whether laboratories should perform phenotypic or genotypic testing for carbapenemase production, utilizing PCR to identify specific carbapenem-resistance genes in CRPA isolates. Such comprehensive testing is pivotal in assisting antimicrobial stewardship programs in making informed decisions regarding the selection of appropriate therapies for pseudomonal infections.[1]

Phenotypic methods have been documented for detecting carbapenemase producers within the *Enterobacteriaceae*, with some adaptable for application to non-fermentative bacteria. The methods endorsed by the Clinical and Laboratory Standards Institute (CLSI) for clinical microbiology laboratories to detect carbapenemase-producing *Enterobactarales* and *P. aeruginosa* encompass the Carba NP test and the modified Carbapenem Inactivation Method (mCIM).[5] The Carba NP test stands out for its simplicity and suitability for routine use in clinical laboratories. On the other hand, the mCIM has exhibited notable sensitivity and specificity in numerous recent studies.[6] However, each test has shown to have limitations against certain carbapenemase genes. For instance, the carbaNP test failed to detect $bla_{OXA-181}$ and $bla_{OXA-232}$, which are commonly found in Southeast Asian countries,[7] while the mCIM has been reported to have a limitation on particular IMP and GES genes.[8,9]

In addition, there is limited information regarding the distribution of carbapenemase genes among *P. aeruginosa* in Thailand; therefore, our study aimed to assess the effectiveness of the mCIM for detecting CPPA.

Materials and methods

Bacterial isolates

A total of 164 isolates of CRPA from non-duplicated patients admitted to Ramathibodi Hospital, a 1000-bed tertiary care hospital in Bangkok, Thailand, between 2019 and 2022, were used. The isolates exhibited the least intermediate or resistance to either imipenem, doripenem, or meropenem according to the clinical breakpoint recommended by the Clinical Laboratory Sciences Institute (CLSI).[10]

Modified carbapenem inactivation method (mCIM) testing

All isolates were tested for carbapenemase using the mCIM test per the CLSI guidelines. Briefly, a 10-µL loop full of CRPA was suspended in 2 mL of brain heart infusion broth (BHI). A 10 µg meropenem disc (Becton, Dickinson, USA) was added to the suspension and incubated at 37 °C for 4 hours. Following incubation, the disc was removed and placed on Muller-Hinton agar previously spread with *Escherichia coli* ATCC 25922 adjusted to a 0.5 McFarland standard. The plates were incubated at 37 °C for 24 hours. The inhibition zones were measured for interpretation according to CLSI guidelines.[10] *Klebsiella pneumoniae* ATCC BAA-1705 (bla_{KPC}) and PS249 *P. aeruginosa* (bla_{GES}, bla_{IMP}, bla_{VIM}, and bla_{NDM}) were used as the positive controls, while *P. aeruginosa* ATCC 27853 and *K. pneumoniae* ATCC BAA-1706 strains were negative controls.

Molecular detection of carbapenemase genes

For molecular testing, a direct flow kit (Master Diagnostica, Spain) was used to detect carbapenemase genes. The principle of the test kit is based on the targeted DNA amplification by multiplex PCR and the hybridization of the PCR products by specific probes coated on the membrane. Fifteen carbapenemase genes, including bla_{KPC}, bla_{NDM}, bla_{IPM}, bla_{IMI}, bla_{VIM}, bla_{GES}, bla_{GIM}, bla_{SME}, bla_{SIM}, $bla_{NMC/IMI}$, bla_{SPM}, $bla_{OXA-48-like}$, $bla_{OXA-23-like}$, $bla_{OXA-24-like}$, $bla_{OXA-51-like}$, and $bla_{OXA-58-like}$, were tested. Briefly, parts from a few colonies were suspended in 500 µL DNase/RNase-free double-distilled water. The suspension was vortexed until homogeneity was obtained. Thirty microliters of the suspension were added to the PCR tube containing the lyophilized master mix (Master Diagnostica, Spain). The PCR tubes were placed in a thermocycler, and PCR was run under the following conditions: one cycle of 10 minutes at 25 °C, then 1 cycle of 3 minutes at 95 °C, then 40 cycles of denaturation at 95 °C for 15 seconds, annealing at 55 °C for 45 seconds, and extension at 75°C for 1 minute. The PCR amplicons were further hybridized with specific probes. The amplicons were heated at 95 °C for 8-10 minutes and rapidly cooled in ice for at least 2 minutes. The hybridization process was automatically performed by hybriSpot in HS24 platform (Master Diagnostica, Spain). The hybridization result was analyzed by the Hybrispot software.

Conventional PCR and sequencing of bla_{VIM}, bla_{IMP}, and bla_{GES} genes

Regarding some limitation of the AMR flow kit on detecting certain types of bla_{VIM} and bla_{IPM}, the additional PCR determinations of these two genes were tested in all isolates. In addition, those isolates that revealed a negative result of mCIM with positive of bla_{GES} underwent PCR and sequencing. The primers of bla_{VIM}, bla_{IPM} and bla_{GES} is shown in Table 1.

Table 1. Primers of bla_{VIM}, bla_{IMP}, and bla_{GES}.[11,12]

Gene	*Sequence (5' to 3')*	*Size (bp)*
VIM-F	TGTCCGTGATGGTGATGAGT	456
VIM-R	GTGCTTCCGGGTAGTGTTGT	
IMP-F	GGAATAGAGTGGCTTAAYTCTC	382
IMP-R	GGTTTAAYAAAACAACCACC	
GES-F	TCACTCTGCATATGCGTCGG	692
GES-R	ACTTGACCGACAGAGGCAAC	

Thermal cycling was used under the following conditions: initial denaturation at 94 °C for 10 minutes, 36 cycles of denaturation at 94 °C for 30 seconds, annealing at 52 °C for 40 seconds, extension at 72 °C for 50 seconds, and final extension at 72 °C for 5 minutes. The PCR amplicons were sequenced by bi-directional DNA sequencing (Celemic, The Republic of Korea). The *beta-lactamase* genes were blasted with the database on the NCBI website at http://www.ncbi.nlm.nih.gov/blast.cgi

Results

Distribution of carbapenemase genes in CPPA strains

Of 164 isolates of CRPA, 83 (50.61%) isolates carried at least one *carbapenemase* gene. Five genes, including bla_{VIM}, $bla_{IMP\ (3/15/19\ group)}$, bla_{NDM}, bla_{GES} and $bla_{SIM,}$ were detected in 75 isolates by the direct flow kit. The conventional PCR detected another 8 isolates containing bla_{IPM} other than the 3/15/19 group. The bla_{IMP} and bla_{VIM} were the most common genes found

in 32.53% and 26.51% of the isolates, respectively. Among these, 67/83 (80.72%) contained a single *carbapenemase* gene, while approximately 20% carried more than one resistant gene. Most of these isolates (10/16) had a combination of bla_{NDM} and $bla_{IMP\ (3/15/19\ group)}$. Interestingly, one isolate contained at least four *carbapenemase* genes (Table 2).

Table 2. Distribution of *carbapenemase* genes of CPPA detected by AMR direct flow chip and conventional PCR (N=83).

***Carbapenemase* gene**	**Number of isolates (%)**
bla_{VIM}	22 (26.51)
$bla_{IMP\ (3/15/19\ group)}$	19 (22.89)
bla_{NDM}	12 (14.46)
bla_{NDM} + $bla_{IMP\ (3/15/19\ group)}$	10 (12.05)
$bla^{*}_{IMP\ (not\ 3/15/19\ group)}$	8 (9.64)
bla_{GES}	5 (6.02)
bla_{VIM} + $bla_{IMP\ (3/15/19\ group)}$	2 (2.41)
bla_{VIM} + bla_{NDM}	2 (2.41)
bla_{SIM}	1 (1.20)
bla_{VIM} + bla_{GES}	1 (1.20)
bla_{VIM} + bla_{GES} + bla_{NDM} + $bla_{IMP\ (3/15/19\ group)}$	1 (1.20)

Note: *$bla_{IMP\ (not\ 3/15/19\ group)}$ was detected by conventional PCR.

Detection of CPPA by mCIM

To establish the efficacy of mCIM for detecting CPPA, the results of mCIM were compared with molecular testing. Of the 164 isolates, sixty-eight revealed positive results with mCIM, and ninety-six were negative. In these 96 isolates of mCIM-negative, fifteen isolates provided positive results with the *carbapenemase* genes (Table 3). The sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) of the mCIM were 81.93, 100, 100, and 84.38%, respectively. Among these isolates, 6 (40%), 5 (33.33%), and 3 (20%) carried bla_{VIM}, bla_{GES} and bla_{IMP}, respectively, and a single isolate containing both bla_{VIM} and bla_{IPM} (Table 4).

Table 3. The efficacy of mCIM for detecting carbapenemase-producing *P. aeruginosa* (CPPA) and non-carbapenemase-producing *P. aeruginosa* (non-CPPA) against *carbapenemase* genes detection.

	CPPA* (N=83)	**Non-CPPA**** (N=81)
mCIM positive	68	0
mCIM negative	15	81

Note: **carbapenemase* genes detected, ***carbapenemase* genes not detected.

Table 4. Efficacy of mCIM versus various *carbapenemase* genes.

***Carbapenemase* genes**	**Number of isolates** (N=83)	
	mCIM positive (N=68)	**mCIM negative** (N=15)
bla_{VIM}	16	6
$bla_{IMP\ (3/15/19\ group)}$	16	3
bla_{NDM}	12	0
bla_{NDM} + $bla_{IMP\ (3/15/19\ group)}$	10	0
bla_{GES}	0	5
$bla^{*}_{IMP\ (not\ 3/15/19\ group)}$	8	0
bla_{VIM} + $bla_{IMP\ (3/15/19\ group)}$	1	1
bla_{VIM} + bla_{NDM}	2	0
bla_{SIM}	1	0
bla_{VIM} + bla_{GES}	1	0
bla_{VIM} + bla_{GES} + bla_{NDM} + $bla_{IMP\ (3/15/19\ group)}$	1	0

Note: *$bla_{IMP\ (not\ 3/15/19\ group)}$ was detected by conventional PCR.

Of the fifteen mCIM-negative isolates, bla_{VIM-2} was demonstrated in six isolates, with one isolate containing bla_{VIM-2} and bla_{IPM-62}. Most of these isolates revealed a high level of resistance to all three carbapenems (Table 5). A single isolate had $bla_{VIM-63.}$ However, it is worth noting that not all bla_{VIM-2} and bla_{VIM-63} revealed negative results by mCIM. Approximately 40 and 75% of isolates carrying bla_{VIM-2} and bla_{VIM-63}, respectively, were detected by mCIM (data not shown). The only *imipenemase* gene (IMP) type related to a negative mCIM result was bla_{IMP-62}. All other bla_{IMP}-containing isolates were mCIM positive. Interestingly, all bla_{GES-5} isolates were unable to be detected by mCIM, despite their high-level resistance in 4/5 isolates.

Table 5. Distribution of *carbapenemase* genes in mCIM-negative carbapenemase-producing (CP) strains of *P. aeruginosa* (N=15).

***Carbapenemase* genes**	**Number of isolates**	**MIC of each isolate**		
		Imipenem	Meropenem	Doripenem
bla_{VIM-2}	5	≥32	≥32	≥32
		≥32	≥32	≥32
		≥32	≥32	16
		≥32	16	16
		4	8	1
bla_{VIM-63}	1	≥32	≥32	≥32
bla_{IMP-62}	3	≥32	≥32	≥32
		≥32	≥32	16
		16	4	4
bla_{IMP-62} + bla_{VIM-2}	1	8	16	4
bla_{GES-5}	5	≥32	≥32	≥32
		≥32	≥32	≥32
		≥32	≥32	≥32
		≥32	≥32	≥32
		8	2	1

Discussion

Carbapenem-resistant *Pseudomonas aeruginosa* (CRPA) has become a global health concern because of its association with not only limited therapeutic options and high mortality rates, but also plays a major role in healthcare-associated infection. Unlike the carbapenem-resistant *Enterobacterales* (CRE), for which most of the resistant mechanisms are based on the production of carbapenemase enzymes, the data of our study demonstrate that approximately equal percentages of carbapenemase and non-carbapenemase-producing distributed in the area. However, it is crucial to identify the isolates that contain the *carbapenemase* genes since the genes can transfer among *P. aeruginosa* or across species.[13]

In addition, *Enterobacterales,* such as *Escherichia coli* and *Klebsiella pneumoniae,* in which most of the *carbapenemase* genes belong to bla_{KPC}, bla_{NDM}, and bla_{OXA-48}, the most common carbapenem-resistant genes among *P. aeruginosa* found in this study were bla_{IMP}, bla_{VIM}, and bla_{NDM}, each of which was detected in approximately 23 to 38% of the isolates. Therefore, it reflected that nearly 90% of the CPPA carried metallo-beta-lactamase genes, leading to even smaller choices of antibiotics that effectively treat CPPA in the region.

The modified carbapenem inactivation method (mCIM) has been recognized for accurately detecting carbapenemases. It is recommended as a standard test by the CLSI for *Enterobacterales* and *P. aeruginosa*.[10] In carbapenemase-producing *Enterobacterales* (CPE), the test is excellent against the organisms isolated in Thailand and Southeast Asia compared with other standard tests, e.g., CarbaNP.[7] The efficacy of each test varied between different parts of the world and is strongly related to the distribution of bacterial strains in each area.[7] However, the efficacy of mCIM against *P. aeruginosa* strains isolated in the region has not been widely reported. The results from our study revealed that mCIM reached 100% specificity, but the sensitivity was less than 90%. The low sensitivity was due to the existence of specific genes, including bla_{VIM-2}, bla_{VIM-63}, bla_{IPM-62} and bla_{GES-5}. The number of these strains was over 15% of the CPPA. Therefore, the efficacy of mCIM for detecting carbapenemase in *P. aeruginosa* relied on the percentage of certain resistant genes in the region. Nevertheless, it remains unclear regarding the factors that cause false negatives by mCIM, despite the high level of carbapenem resistance in most of these isolates. This may be due to the low expression or low activity of the enzymes in certain strains. In 2020, Christian M. Gill et al. reported that approximately 50% of Guiana extended spectrum (GES) enzymes were detected as positive using the standard mCIM. These genes were identified as bla_{GES-1}, bla_{GES-19}, and bla_{GES-20}.[14] They hypothesized that the lower catalytic activity of GES might be a contributing factor. To address this, the investigators increased the bacterial inoculum, speculating that it could enhance the mCIM test's sensitivity. Subsequent retesting with a higher inoculum showed improved mCIM performance. A similar outcome occurred in the prolonged incubation of the meropenem disc for eight hours. Lisboa and colleagues encountered a similar challenge, where the mCIM could not detect 4 out of 16 *P. aeruginosa* isolates carrying the bla_{GES-5} gene.[8] In our study, although only 5 isolates were found containing bla_{GES-5}, all were negative by the mCIM.

Limitations

Although the data demonstrated the efficacy of the mCIM against CPPA isolates, the number of certain uncommon *carbapenemase* genes was limited. Therefore, the expansion of bacterial isolates, particularly those uncommon genes, would provide a clearer understanding.

Conclusion

In conclusion, three major findings were revealed from this study. Firstly, the resistance mechanism of carbapenem-resistant *P. aeruginosa* (CRPA) was approximately equal between carbapenemase-producing and non-carbapenemase-producing mechanisms. Secondly, the majority of carbapenemase enzymes were metallo-beta-lactamase, which were encoded by bla_{VIM}, $bla_{IPM,}$ and bla_{NDM} genes. Finally, the mCIM was shown to be an effective test for detecting CPPA. However, there were limitations in detecting certain genes; therefore, the information on the distribution of *carbapenemase* genes in the area would be necessary. Although this study was based on *P. aeruginosa* isolated mainly in Bangkok, Thailand, it provided solid evidence regarding the distribution of *carbapenemase* genes among *P. aeruginosa* as well as the efficacy of the mCIM for detecting CPPA. Nevertheless, further study using isolates from various regions in the country would demonstrate a clearer picture of the antibiotic resistance of this medically important organism.

Ethical approval

This study was conducted with the approval of the Institutional Review Board at Naresuan University, Thailand, under ethical approval number P1-0040/2566.

Informed consent statement

None

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Conflicts of interest

The authors declare no conflicts of interest.

Data availability statement

The data presented in this study are available on request directly to the corresponding author. Due to patient privacy concerns, the data are not publicly available.

CRediT authorship contribution statement

Jakkrit Khampakul: conceptualization, methodology, software, validation, formal analysis, investigation, resources, data curation, writing: original draft preparation; **Noppadon Jumroon**: conceptualization, methodology, investigation, writing: review and editing, supervision; **Narong Chaihongsa**: software; **Pitak Santanirand**: conceptualization, methodology, formal analysis, investigation, resources, writing: original draft preparation, review and editing, visualization, supervision, project administration, funding acquisition. All authors have read and agreed to the published version of the manuscript.

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