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Original Article

Molecular Characterization and Resistance Gene Co-occurrence in Carbapenem-Resistant *Klebsiella peumoniae* from a Bangkok Tertiary Hospital, Thailand

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Abstract

Introduction: Carbapenem-resistant *Klebsiella pneumoniae* (CRKP) is a significant global health concern due to its high morbidity and mortality rates and limited treatment options. We investigated the molecular characteristics and co-occurrence of resistance genes in CRKP isolates from a tertiary hospital in Bangkok, Thailand.

Methodology: Seventy-three non-repetitive CRKP isolates were collected from clinical specimens of hospitalized patients from August 2022 to January 2023. Identification was confirmed using standard microbiological tests and MALDI-TOF-MS. Antimicrobial susceptibility was assessed using the MicroScan system. PCR was employed to detect carbapenemase genes and extended-spectrum beta-lactamases (ESBLs).

Results: The study revealed a high resistance rate among the CRKP isolates to cephalosporins, aminoglycosides, fluoroquinolone, and trimethoprim/sulfamethoxazole. Of the 73 CRKP isolates, 98.6% were susceptible to tigecycline, and 46.6% remained susceptible to ceftazidime-avibactam. Molecular analysis revealed that $bla_{\text{OXA-48-like}}$ (93.2%) and $bla_{\text{NDM-1}}$ (43.8%) were the predominant carbapenemase genes. All isolates harbored the ESBL gene $bla_{\text{CTX-M-15}}$. The co-occurrence of $bla_{\text{OXA-48-like}}$ and $bla_{\text{NDM-1}}$ was observed in 39.7% of the isolates. Ward-specific analysis revealed the highest diversity and frequency of co-resistance gene profiles in isolates from the medical ward, while isolates from ICUs also exhibited complex gene combinations involving multiple ESBL and carbapenemase genes.

Conclusions: This study reveals a high prevalence of $bla_{\text{OXA-48-like}}$ and $bla_{\text{NDM-1}}$ genes among CRKP isolates in a tertiary hospital in Bangkok. The diversity of resistance profiles and the clustering of complex gene combinations, particularly in medical and ICU wards, underscore the need for continuous molecular surveillance and stringent infection control measures to manage the spread of CRKP in healthcare setting.

Key words: Antimicrobial susceptibility; Molecular characteristic; Carbapenem-resistant *Klebsiella pneumonia;* Thailand.

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Introduction

The emergence of antibiotic resistance among bacterial pathogens poses a significant threat to public health worldwide. Among these pathogens, *Klebsiella pneumoniae* stands out as the most notoriously opportunistic. It can cause a range of infections, including pneumonia and bloodstream infections, particularly in hospitalized patients. In recent years, carbapenem-resistant *K. pneumoniae* (CRKP) strains have become a global health concern, causing outbreaks of infections from this bacterium and creating major challenges for healthcare systems due to their resistance to most of the commonly-used antibiotics. Known for its high morbidity and mortality rates, CRKP is a significant and growing threat in healthcare settings worldwide [1,2].

Carbapenem antibiotics are critically important in combating multidrug-resistant (MDR) gram-negative bacteria and are often considered a last-line treatment option for severe infections [3,4]. The mechanisms of *K. pneumoniae*'s resistance to carbapenems primarily involve the production of carbapenemase enzymes, which hydrolyze the antibiotic, rendering it ineffective against bacteria. Carbapenemases are categorized into three main classes: Class A includes *Klebsiella pneumoniae* carbapenemase (KPC); Class B encompasses metallo-β-lactamases such as Imipenemase (IMP), New Delhi metallo-β-lactamase (NDM), and Verona integron-encoded metallo-β-lactamase (VIM); and Class D is represented by OXA-48-like enzymes [5]. The most prevalent carbapenemase genes in CRKP are bla_{KPC}, bla_{NDM}, bla_{VIM}, bla_{IMP}, and $bla_{\text{OXA-48-like}}$. In Thailand, $bla_{\text{OXA-48-like}}$ and bla_{NDM} are reported as the most common [6]. Previous research shows that the distribution of these genes varies across different healthcare settings in the region [7,8]. This variation in carbapenemase genes may influence the efficacy of specific antibiotics since different enzymes exhibit varying levels of hydrolytic activity and susceptibility to inhibitors [9]. In addition

to carbapenemase production, many CRKP isolates also harbor extended-spectrum β-lactamase (ESBL) genes such as $bla_{\text{CTX-M-15}}$, bla_{SHV}, bla_{TEM}, and $bla_{\text{OXA-1}}$, which further contribute to resistance against third-generation cephalosporins. Of particular concern is the co-existence of ESBL and carbapenemase genes within a single bacterial isolate. This genetic combination not only exacerbates antimicrobial resistance but also promotes horizontal gene transfer via mobile genetic elements, particularly plasmids [10,11]. This genetic convergence significantly increases the risk of rapid dissemination of multidrug-resistant strains in healthcare settings, complicating infection control efforts and severely limiting therapeutic options [12]. Consequently, infections caused by CRKP are associated with high morbidity and mortality rates. Therefore, understanding the mechanisms underlying carbapenem resistance in *K. pneumoniae* isolates is crucial for developing effective strategies to combat this threat in the study hospital. Thus, this study aims to investigate molecular characteristics and resistance gene co-occurrence in CRKP isolates collected from clinical specimens in the Bangkok metropolitan hospital.

Methodology

Microbiological identification and antimicrobial susceptibility test

During the six-month study period (August 2022–January 2023), approximately 660 clinical *K. pneumoniae* isolates causing infection were recovered from hospitalized patients at a tertiary hospital in the Bangkok Metropolitan Area. The isolates were initially identified using standard microbiological and conventional biochemical tests, and their identification was then confirmed with the matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF-MS) system (Microflex™ LRF, Bruker, Germany). The antimicrobial susceptibilities of *K. pneumoniae* were determined using the MicroScan antimicrobial susceptibility system (WalkAway 40 plus, Beckman Coulter Diagnostics, USA). The findings were interpreted following the CLSI guidelines [13]. There are no established CLSI MIC breakpoints for tigecycline specifically for Enterobacterales. Therefore, tigecycline MIC results were interpreted according to the U.S. FDA guidelines, where MIC ≤ 2 mg/L is considered susceptible; 4 mg/L is intermediate; and > 8 mg/L is resistant [14]. FDA breakpoints were chosen rather than EUCAST criteria to ensure consistency with the interpretive standards embedded in the automated testing system used in this study and with routine practice in the laboratory used in this study. Any *K. pneumoniae* isolates not susceptible to imipenem, meropenem, or ertapenem were defined as CRKP. A total of 122 (18.3%) of the 660 clinical isolates met this definition. For the present study, a subset of 73 non-duplicate CRKP isolates from clinical infection specimens were included for further analysis.

Genetic characterization of antimicrobial-resistant genes

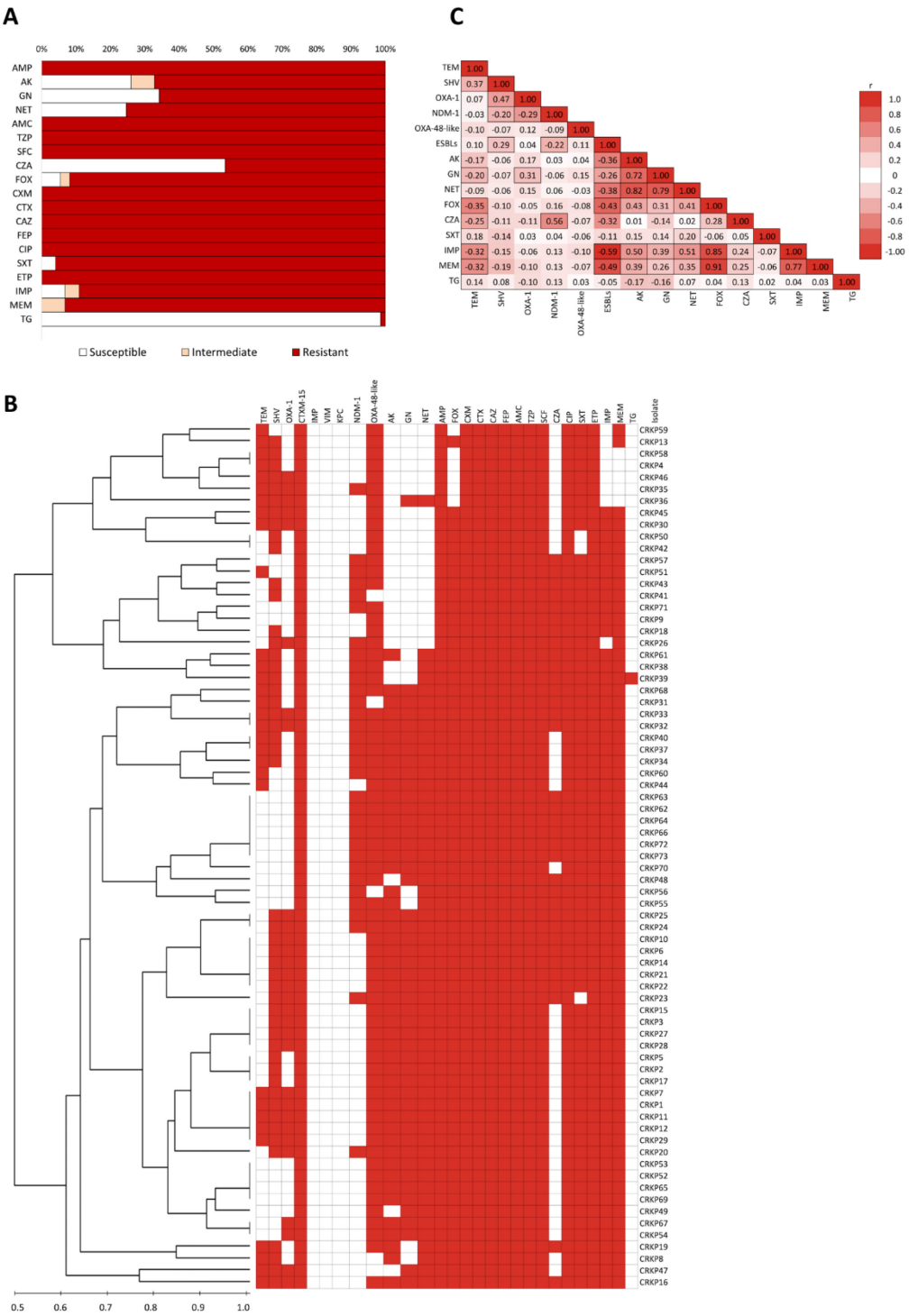
Bacterial DNA was extracted using the boiling method. Fresh colonies suspended in Tris-EDTA buffer pH 7.4 were incubated at 95°C for 10 minutes and then cooled on ice for 10 minutes. Cell debris was sedimented by centrifugation at 12,000 rpm for 5 minutes. The crude DNA was kept at 4°C for further genetic characterization.

Polymerase chain reaction (PCR) was performed to detect the most common carbapenemase coding genes ($bla_{\text{OXA-48-like}}$, $bla_{\text{NDM-1}}$, bla_{IMP}, bla_{VIM}, and bla_{KPC}) and

Table 1. Primer used in this study.

Gene	Primer	Sequence (5'-3')	Length (bp)	Final concentration (μM)	References
bla_{TEM}	TEM-F	CATTTCCGTGTCGCCCTTATTC	800	0.4	[15]
	TEM-R	CGTTCATCCATAGTTGCCTGAC		0.4	
bla_{SHV}	SHV-F	AGCCGCTTGAGCAATTAAAC	713	0.4	
	SHV-R	ATCCCGCAGATAAATCACCAC		0.4	
$bla_{\text{OXA-1}}$	OXA-1F	GGCACCAGATTCAACTTTCAAG	564	0.4	
	OXA-1R	GACCCCAAGTTTCCTGTAAGTG		0.4	
$bla_{\text{CTXM-15}}$	CTXM-15F	AGAATAAGGAATCCCATGGTT	874	0.4	[16]
	CTXM-15R	ACCGTCGGTGACGATTTTAG		0.2	
bla_{IMP}	IMP-F	TTGACACTCCATTTACDG	139	0.5	[15]
	IMP-R	GATYGAGAATTAAGCCACYCT		0.5	
bla_{VIM}	VIM-F	GATGGTGTTTGGTCGCATA	390	0.5	
	VIM-R	CGAATGCGCAGCACCAG		0.5	
bla_{KPC}	KPC-F	CATTCAAGGGCTTTCTTGCTGC	538	0.2	
	KPC-R	ACGACGGCATAGTCATTTGC		0.2	
$bla_{\text{NDM-1}}$	NDM-1F	GGTTTGGCGATCTGGTTTTC	621	0.2	[17]
	NDM-1R	CGGAATGGCTCATCACGATC		0.2	
$bla_{\text{OXA-48-like}}$	preOXA-48F	TATATTGCATTAAGCAAGGG	800	0.4	[18]
	preOXA-48R	CACACAAATACGCGCTAACC		0.4	

Figure 1. Heatmap, clustering analysis, and association of resistance profiles among CRKP isolates.



A: Heatmap showing the antimicrobial susceptibility of 73 CRKP isolates to various tested agents. Red indicates resistance, and white indicates susceptibility. **B:** Hierarchical phenotypic clustering of CRKP isolates based on resistance gene profiles and antimicrobial susceptibility patterns. Red indicates the presence of resistance genes or resistance to antimicrobial agents; white indicates absence or susceptibility. **C:** Association analysis between antimicrobial resistance genes and phenotypic resistance to antimicrobial agents. The color scale represents the Phi coefficient (φ). Red cells with gray borders indicate strong and statistically significant associations ($p < 0.05$).

AK: Amikacin; AMC: Amoxicillin-clavulanate; AMP: Ampicillin; CAZ: Ceftazidime; CIP: Ciprofloxacin; CTX: Cefotaxime; CXM: Cefuroxime; CZA: Ceftazidime-avibactam; ETP: Ertapenem; FEP: Cefepime; FOX: Cefoxitin; GN: Gentamicin; IMP: Imipenem; MEM: Meropenem; NET: Netilmicin; SFC: Cefoperazone-sulbactam; SXT: Trimethoprim-sulfamethoxazole; TG: Tigecycline; TZP: Piperacillin-tazobactam.

ESBL genes (*bla*$_{\text{CTX-M-15.}}$ *bla*$_{\text{TEM}}$, *bla*$_{\text{SHV}}$, and *bla*$_{\text{OXA-1}}$) using specific primers, as shown in Table 1. The PCR conditions were described as 94°C denaturation for 10 minutes, 30 cycles of 94°C denaturation for 40 seconds, 60°C annealing for 40 seconds, 72°C extension for 1 minute, and a final extension at 72°C for 7 minutes. Electrophoresis with 1.5% agarose gel was used to analyze the PCR products.

Statistical Analysis

Descriptive statistics, including frequency and percentage, were used in this study. The antimicrobial resistance profiles and genes were coded as follows: 1 indicates resistant isolates, while 0 represents intermediate or susceptible isolates. Next, the chi-square test was employed to determine the association between antimicrobial drug resistance and the presence of resistance genes. A $p < 0.05$ was considered statistically significant. To better illustrate the results, hierarchical clustering of CRKP isolates was performed based on their combined antimicrobial susceptibility profiles and the presence of resistance genes. A similarity metric for binary resistance data was computed and used for the hierarchical clustering of CRKP isolates using the unweighted pair-group averages method (UPGMA) in XLSTAT v19.1. software (Lumivero®, Colorado, USA).

Results

Seventy-three non-duplicated *K. pneumonia* isolates were collected from 37 male (50.7%) and 36 female (49.3%) patients. The isolates were taken from different clinical specimens, including urine (32/73, 43.8 %), sputum (31/73, 42.5 %), blood (4/73, 5.5 %), and other specimen sources (6/73, 8.2 %). CRKP isolates were mostly obtained from the medical ward (29/33, 39.7%) (Table 2).

The antimicrobial susceptibility rates of the 73 CRKP isolates to antimicrobial agents are shown in Figure 1A. All isolates were resistant to cephalosporin (cefoxitin, cefuroxime, cefotaxime, ceftazidime, and cefepime), beta-lactam/beta-lactamase inhibitors (piperacillin/tazobactam and cefoperazone/sulbactam), and ertapenem. For the aminoglycoside class, 49 (67.1%), 48 (65.8%), and 55 (75.3%) isolates were resistant to amikacin, gentamicin, and netilmicin, respectively, while for the carbapenem class, 65 (89.0%) and 68 (93.2%) isolates were imipenem- and meropenem-resistant. Seventy-two (98.6%) of the isolates were susceptible to tigecycline *in vitro,* and 34 (46.6%) were susceptible to ceftazidime/avibactam.

CRKP isolates were analyzed using PCR to determine the presence and co-occurrence of ESBL and carbapenemase genes (Table 2 and Figure 1B). Of the CRKP isolates, 71 out of 73 (97.2%) contained at least one of the carbapenemase genes. The *bla*$_{\text{OXA-48-like}}$ gene was the most frequently detected carbapenemase gene, occurring in 93.2% (68/73) of cases, while *bla*$_{\text{NDM-1}}$ was the second most prevalent (32/73, 43.8%) in isolates from this study. There were no positive results for *bla*$_{\text{IMP}}$, *bla*$_{\text{VIM}}$, or *bla*$_{\text{KPC}}$. More than half of the isolates (43/73, 58.9%) carried only one carbapenemase gene, with *bla*$_{\text{OXA-48-like}}$ being the most prevalent, occurring in 39/73 isolates (53.4%). Three CRKP isolates (3/73, 4.1%) carried only *bla*$_{\text{NDM}}$ genes. Moreover, 29 isolates (39.7%) contained both *bla*$_{\text{OXA-48-like}}$ and *bla*$_{\text{NDM}}$ genes. For ESBL genes, all 73 CRKP isolates carried the *bla*$_{\text{CTX-M-15}}$ gene. The *bla*$_{\text{TEM}}$ gene was detected in 49 isolates (67.1%), while both *bla*$_{\text{SHV}}$ and *bla*$_{\text{OXA-1}}$ were positive in 30 isolates (41.1%).

Statistical association between the presence of antimicrobial resistance genes and resistance phenotypes was assessed with the results illustrated in Figure 1C. A moderate association was observed between *bla*$_{\text{TEM}}$ and *bla*$_{\text{SHV}}$ (r = 0.37), and between *bla*$_{\text{SHV}}$ and *bla*$_{\text{OXA-1}}$ (r = 0.47), while weak or negative associations were found between *bla*$_{\text{NDM-1}}$ and ESBL genes (*bla*$_{\text{TEM}}$: r = – 0.03, *bla*$_{\text{SHV}}$: r = – 0.20, *bla*$_{\text{OXA-1}}$: r = – 0.29). The presence of *bla*$_{\text{NDM-1}}$ was moderately associated with resistance to ceftazidime-avibactam (r = 0.56). Among the antimicrobials, strong associations were found between imipenem and meropenem (r = 0.77), as well as between meropenem and cefoxitin (r =

Table 2. Characteristics of CRKP Isolates (n = 73).

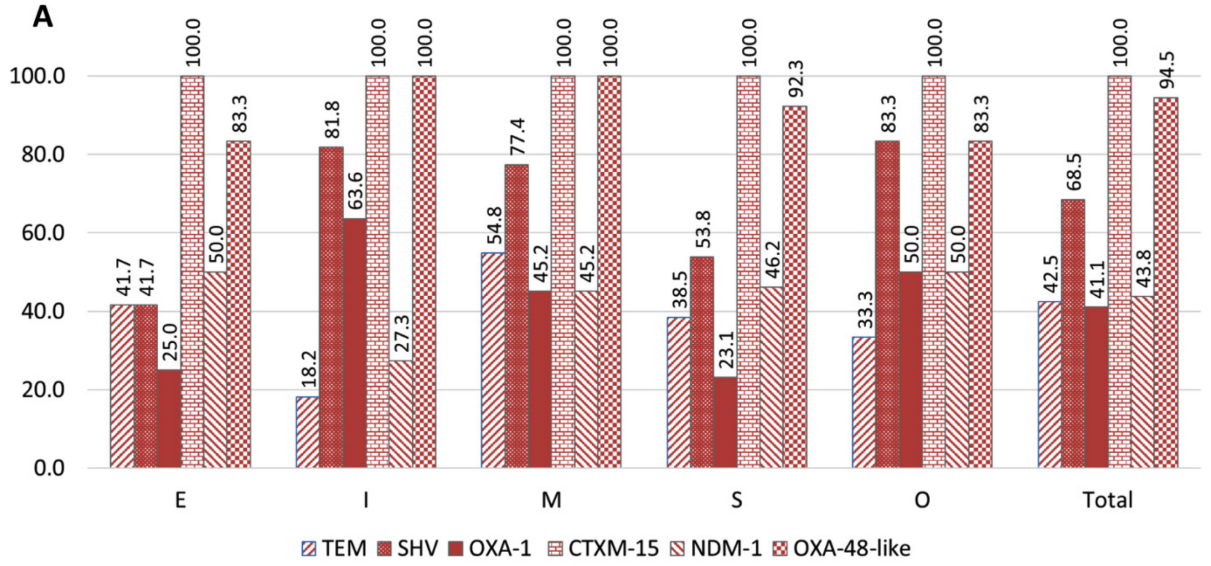
Sample characteristic	N (%)
Gender	
Male	37 (50.7)
Female	36 (49.3)
Specimen	
Blood	4 (5.5)
Sputum	31 (42.5)
Urine	32 (43.8)
Others	6 (8.2)
Ward	
Emergency ward	12 (16.4)
Medical ward	29 (39.7)
Surgical ward	14 (19.2)
Intensive care units	12 (16.4)
Other	6 (8.2)
Carbapenemase gene	
bla$_{\text{OXA-48-like}}$	68 (93.2)
bla$_{\text{NDM-1}}$	32 (43.8)
bla$_{\text{IMP}}$	0 (0.0)
bla$_{\text{VIM}}$	0 (0.0)
bla$_{\text{KPC}}$	0 (0.0)
ESBL gene	
bla$_{\text{SHV}}$	30 (41.1)
bla$_{\text{TEM}}$	49 (67.1)
bla$_{\text{OXA-1}}$	30 (41.1)
bla$_{\text{CTX-M-15}}$	73 (100.0)

0.91). Aminoglycosides such as amikacin and netilmicin also showed a strong positive association (r = 0.82).

The hierarchical clustering dendrograms for CRKP isolates based on resistance gene profiles and antimicrobial susceptibility patterns are presented in Figure 1B. They consist of two distinct groups, clusters A (minor) and B (major). Cluster A includes the CRKP isolates that were susceptible to aminoglycoside drugs, while most CRKP isolates belonging to cluster B were highly resistant to the aminoglycoside drugs tested.

The prevalence of resistance genes among different hospital wards is presented in Figure 2A. The highest prevalence of resistance genes was observed in the medical ward, where bla_{TEM}, bla_{SHV}, and $bla_{OXA-48-like}$ genes were present in 54.8%, 77.4%, and 100% of isolates, respectively. Significant resistance was also observed in the intensive care unit (ICU), with bla_{SHV}, bla_{OXA-1}, and $bla_{OXA-48-like}$ genes present in 81.8%, 63.6%, and 100% of isolates, respectively. Interestingly, the prevalence of the bla_{NDM-1} gene varied across wards. The distribution of *K. pneumoniae* isolates carrying different combinations of resistance genes is illustrated in Figure 2B. A total of 18 distinct gene co-occurrence profiles were identified. The most common gene combination between carbapenemase and ESBL genes found in CRKP was bla_{NDM-1}, $bla_{OXA-48-like}$, and $bla_{CTX-M-15}$, detected in 11 isolates (15.1%). This was followed by the profile combining $bla_{OXA-48-like}$, $bla_{CTX-M-15}$, bla_{SHV}, bla_{OXA-1}, and bla_{TEM} (10/73,13.7%) and the combination of $bla_{OXA-48-like}$, $bla_{CTX-M-15}$, bla_{TEM}, and bla_{OXA-1} (9/73, 12.3%). Overall, over 95% of isolates carried both ESBL and carbapenemase genes. When stratified by ward, the medical ward had the highest number of isolates (29/73, 39.7%) and exhibited the most diverse gene

Figure 2. Distribution of resistance genes across hospital wards. (A) Prevalence of carbapenemase and ESBL genes among CRKP isolates in each ward. (B) Distribution of CRKP isolates based on different resistance gene combinations across wards.



B

Resistant gene combination																			Number	%
$bla_{CTX-M-15}$	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	●	73	100.0
bla_{SHV}					●	●	●	●	●			●	●	●	●	●	●	●	30	41.1
bla_{OXA-1}				●				●	●							●	●	●	30	41.1
bla_{TEM}										●	●	●	●	●	●	●	●	●	49	67.1
bla_{NDM-1}	●	●			●	●		●		●		●	●			●			32	43.8
$bla_{OXA-48-like}$		●	●	●		●	●	●	●	●	●		●		●	●		●	69	94.5
Percentage of isolates by ward (%)																				
Emergency ward	8.3	41.7	0.0	8.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	8.3	16.7	0.0	0.0	16.7	12	16.4
Intensive care unit	0.0	8.3	8.3	0.0	0.0	0.0	8.3	16.7	33.3	0.0	0.0	0.0	0.0	0.0	8.3	0.0	8.3	8.3	12	16.4
Medical ward	0.0	10.3	3.5	3.5	0.0	0.0	13.8	10.3	3.5	3.5	3.5	0.0	17.2	0.0	3.5	6.9	0.0	20.7	29	39.7
Surgical ward	0.0	14.3	28.6	0.0	0.0	7.1	0.0	0.0	14.3	0.0	7.1	7.1	14.3	0.0	0.0	0.0	0.0	7.1	14	19.2
Other wards	0.0	0.0	0.0	0.0	16.7	0.0	16.7	0.0	33.3	16.7	0.0	0.0	0.0	0.0	0.0	16.7	0.0	0.0	6	8.2
Number of isolates	1	11	6	2	1	1	6	5	9	2	2	1	7	1	4	3	1	10		
%	1.4	15	8.2	2.7	1.4	1.4	8.2	6.9	12	2.7	2.7	1.4	9.6	1.4	5.5	4.1	1.4	14		

combination patterns. The surgical ward accounted for 14 isolates (19.2%) and was notable for a higher frequency of isolates co-carrying *bla*$_{\text{CTX-M-15}}$ and *bla*$_{\text{OXA-48-like}}$. The emergency department and ICU contributed 12 isolates each (16.4%). Notably, 41.7% of CRKP isolates from the emergency ward harbored the most common combination (*bla*$_{\text{NDM-1}}$, *bla*$_{\text{OXA-48-like,}}$ and *bla*$_{\text{CTX-M-15}}$). In contrast, isolates from the ICU showed a greater diversity of gene combinations, with 33.3% carrying *bla*$_{\text{OXA-48-like}}$, *bla*$_{\text{CTX-M-15}}$, *bla*$_{\text{TEM}}$, and *bla*$_{\text{OXA-1}}$. The remaining 6 isolates (8.2%) originated from other wards and also demonstrated a variety of resistance patterns.

Discussion

CRKP is a major public health challenge, especially in hospitals where antibiotic use is common. This study examined the molecular characteristics and resistance gene co-occurrence in CRKP isolates from a tertiary hospital in Bangkok, Thailand. The antimicrobial susceptibility profiles revealed extensive resistance to multiple antibiotic classes, including cephalosporins, aminoglycosides, and fluoroquinolones, indicating severely limited treatment options. Although 98.7% of the isolates remained susceptible to tigecycline, the overall burden of resistance was substantial. This is consistent with national data indicating high rates of multidrug resistance among CRKP strains [6,8]. These findings highlight an alarming level of antimicrobial resistance, emphasizing the urgent need for effective infection control measures and antimicrobial stewardship in this study's setting.

The molecular analysis performed in this study demonstrated a high prevalence of carbapenemase-encoding genes among the CRKP isolates, particularly *bla*$_{\text{OXA-48-like}}$ (93.2%) and *bla*$_{\text{NDM-1}}$ (43.8%). These findings align with several studies which have reported that *bla*$_{\text{NDM}}$ and *bla*$_{\text{OXA-48-like}}$ were the most prevalent carbapenemase genes in carbapenem-resistant Enterobacterales (CRE) in Thailand and another country in Southeast Asia [6,7,19]. In Thailand, *bla*$_{\text{OXA-232}}$ has been reported as the most common variant among the *bla*$_{\text{OXA-48-like}}$, followed by *bla*$_{\text{OXA-181}}$, whereas the original *bla*$_{\text{OXA-48}}$ is less frequently detected [6,7]. It is also important to note that the prevalence of *bla*$_{\text{OXA-48-like}}$ might be underestimated. OXA-48-like carbapenemases such as OXA-181, OXA-232, and OXA-244 have low hydrolytic activity against carbapenems. As a result, isolates carrying the *bla*$_{\text{OXA-48-like}}$ gene may appear susceptible to carbapenems in standard susceptibility tests. In this study, this phenomenon was observed in several cases, with 6 and 4 out of 39 isolates carrying *bla*$_{\text{OXA-48-like}}$ found to be susceptible to imipenem and meropenem, respectively. Several studies have found that OXA-48-like carbapenemase-producing CRE can often remain undetected by common phenotypic tests such as Carba NP and the carbapenem inactivation method (CIM) [20,21]. This low activity towards carbapenems allows the strains to appear susceptible in these tests, thereby facilitating their silent spread within healthcare settings. However, in the present study, *bla*$_{\text{OXA-48-like}}$-positive isolates were not further characterized to the specific variant level (e.g., OXA-48, OXA-181, OXA-232), and this limitation should be taken into account when interpreting the observed carbapenem susceptibility profiles.

Previous studies have shown that CRE isolates possessing *bla*$_{\text{NDM}}$ alone or a combination of *bla*$_{\text{NDM}}$ and *bla*$_{\text{OXA-48-like}}$ were more likely to require minimum inhibitory concentrations (MICs) of meropenem equal to or exceeding 16 mg/L, while those producing OXA-48 generally required MICs of less than 16 mg/L [22]. Furthermore, patients infected with NDM-1 or NDM-1/OXA-48 demonstrated a higher 14-day mortality rate compared to those with OXA-48 infections after treatment with carbapenem or colistin-based regimens. Such multidrug-resistant isolates can lead to infections with significant mortality risks due to complications in treatment. Therefore, molecular detection of carbapenemase genes may be necessary, especially in hospitals where CRKP is actively spreading, to support accurate diagnosis and guide timely, appropriate antimicrobial therapy.

The association analysis conducted in the present study revealed the moderate positive association between *bla*$_{\text{TEM}}$ and *bla*$_{\text{SHV}}$ and between *bla*$_{\text{SHV}}$ and *bla*$_{\text{OXA-1}}$, suggesting that these ESBL genes are often co-harbored within the same plasmids or genetic environments. This finding is consistent with previous studies that reported frequent co-occurrence of ESBL genes in multidrug-resistant Enterobacteriaceae [23,24]. Although co-occurrence of ESBL and carbapenemase genes has been documented, the data from the present study reveal weak or negative associations between *bla*$_{\text{NDM-1}}$ and ESBL genes. This suggests that their mobilization and dissemination are not always genetically linked, consistent with the findings from previous studies which reported their presence on distinct plasmids or genetic platforms [25]. Among the antimicrobial agents, a strong association between imipenem and meropenem resistance as well as between meropenem and cefoxitin was observed in the present study. These findings suggest that resistance

mechanisms such as porin loss and β-lactamase production may concurrently affect multiple β-lactam agents. Furthermore, the high association between amikacin and netilmicin resistance indicates likely shared resistance mechanisms, such as aminoglycoside-modifying enzymes or 16S rRNA methyltransferases.

Ceftazidime-avibactam has shown *in vitro* activity against non-MBL-producing isolates, including those with AmpC and ESBL enzymes [26,27]. In this study, CRKP isolates carrying $bla_{\text{NDM-1}}$ alone or together with $bla_{\text{OXA-48-like}}$ showed high resistance to ceftazidime-avibactam, with a strong association observed between resistance and the presence of $bla_{\text{NDM-1}}$ ($p < 0.01$), suggesting that the presence of the $bla_{\text{NDM-1}}$ gene is associated with decreased effectiveness or resistance to ceftazidime-avibactam. The high resistance among isolates with $bla_{\text{NDM-1}}$ can potentially be attributed to the hydrolytic activity of NDM-1 against β-lactams and avibactam [28,29]. In contrast, the lower resistance rate (23.1%, 9/39) in OXA-48-producing CRKP isolates suggests that OXA-48 alone does not strongly resist ceftazidime-avibactam. OXA-48 is a carbapenemase with a weaker effect on cephalosporins and carbapenems but is inhibited by avibactam [30,31]. However, the study by Fröhlich *et al.* showed that exposure to ceftazidime-avibactam can lead to OXA-48 variants in Enterobacterales, resulting in increased ability to hydrolyze ceftazidime and to withstand the inhibitory effect of avibactam [32]. Thus, resistance toward ceftazidime-avibactam among OXA-48-producing Enterobacterales should be monitored.

Another significant finding of this study was an absence of the bla_{KPC}, bla_{VIM}, and bla_{IMP} genes. A previous study conducted in Thailand also reported a low presence or a total absence of bla_{VIM}, bla_{KPC}, and bla_{IMP}, suggesting that these genes are not significant sources of carbapenem resistance in Thailand [7]. Out of the total of 73 isolates, 2 isolates (2.8%) were identified as non-carbapenemase-producing CRKP. These particular carbapenem-resistant strains could potentially be linked to other resistance mechanisms, such as overexpression of efflux pumps, overexpression of AmpC β-lactamases, and porin loss, as documented in prior studies [33].

Fluoroquinolones are a class of broad-spectrum bactericidal agents widely regarded as effective treatment options for various infectious diseases. However, extensive and often inappropriate use of these antibiotics has significantly increased the prevalence of *K. pneumoniae* strains resistant to fluoroquinolones. It has been reported that the frequency of fluoroquinolone resistance in CRKP varies across regions, ranging from 17.0 to 89% [34-36]. In this study, all CRKP isolates were resistant to ciprofloxacin (100%). Quinolone resistance occurs through several mechanisms. Primary resistance results from mutations in the quinolone resistance-determining regions (QRDRs) in DNA gyrase and topoisomerase IV. Additionally, plasmid-mediated quinolone resistance (PMQR) significantly contributes to low-level resistance, involving: (i) qnr proteins, (ii) aac(6′)-Ib-cr enzyme, and (iii) efflux pumps. Several studies have identified a co-existence rate of 57-100% for carbapenemases and PMQR genes in CRKP [27,35]. Furthermore, a study from China highlighted that the co-existence of PMQR genes and mutations in QRDRs can escalate the level of resistance to fluoroquinolones in CRKP within clinical settings [34]. However, detailed information on the molecular mechanisms of fluoroquinolone resistance in CRKP within Thailand remains limited.

In this study, nearly all CRKP isolates (98.6%) remained susceptible to tigecycline. As a glycylcycline antibiotic, tigecycline is known for its broad-spectrum activity against various multidrug-resistant organisms, including CRKP. It acts by inhibiting protein synthesis. This high susceptibility suggests that tigecycline could be an effective therapeutic option for treating infections caused by these resistant strains in this study's hospital setting, aligning with the susceptibility criteria established by the US FDA. However, its application is currently limited to cases where other treatments are not viable, due to its association with a higher mortality risk compared to other antimicrobial agents [37].

The hierarchical clustering analysis revealed distinct groups based on aminoglycoside susceptibility. Cluster A contained isolates susceptible to aminoglycosides, whereas Cluster B comprised those with broad resistance. The distribution of CRKP isolates and their resistance gene combinations varied notably across hospital wards in the present study, suggesting distinct transmission dynamics and selective antibiotic pressures in each setting. The medical ward exhibited the greatest diversity and frequency of resistance gene co-occurrence, particularly involving $bla_{\text{CTX-M-15}}$, bla_{SHV}, $bla_{\text{OXA-1}}$, and $bla_{\text{NDM-1}}$, often in combination with $bla_{\text{OXA-48-like}}$. This pattern may reflect the intensive use of broad-spectrum antibiotics and the high burden of critically ill patients. In the intensive care unit, complex resistance profiles were also observed, with a high frequency of isolates harboring both $bla_{\text{OXA-48-like}}$ and $bla_{\text{NDM-1}}$, along with multiple ESBL genes. These findings highlight how the ICU is a critical hotspot for the emergence, persistence, and transmission of multidrug-resistant organisms, likely

driven by high antibiotic pressure, invasive procedures, and vulnerable patient populations [38,39]. In contrast, isolates from emergency and other wards displayed fewer and less complex resistance patterns, although notable clusters with bla_{SHV} and bla_{NDM-1} were still present. These differences underline the importance of ward-specific antimicrobial stewardship programs. Additionally, the widespread prevalence of carbapenemase genes calls for stringent infection control measures to prevent the clonal spread of these highly resistant strains in this study's setting.

In this study, most CRKP isolates carrying bla_{NDM-1} also harbored $bla_{OXA-48-like}$ (29/32, 90.6%). This co-occurrence suggests potential plasmid-mediated acquisition. Previous studies have shown that plasmids carrying bla_{NDM-1} genes exhibit considerable genetic diversity and often co-carry other resistance determinants such as bla_{OXA-48}, bla_{VIM} and ESBL genes [40]. Notably, these plasmids are frequently associated with replicon types such as IncFII**,** IncA/C2**,** IncX3**,** IncN, and IncL/M, which have broad host ranges and high transfer potential [41,42].

The dissemination of *K. pneumoniae* sequence type 16 (ST16) carrying carbapenemase genes has recently been reported worldwide [43]. A recent nationwide plasmidome analysis in Thailand demonstrated that the dissemination of CRKP carrying bla_{NDM-1} is primarily associated with the sequence type 16 (ST16) lineage, both nationally and within the Bangkok Metropolitan Area [44]. ST16 is known to carry plasmids such as IncFIA/B and ColKP3, which are often linked to the co-occurrence of bla_{NDM-1}, $bla_{OXA-232}$, and $bla_{CTX-M-15}$ [43]. These observations highlight the importance of characterizing the clonal lineages underlying CRKP in the study hospital and in Thailand more broadly. As the present study did not include molecular typing methods such as MLST or whole-genome sequencing (WGS), it was not possible to determine the sequence types or clonal backgrounds of the isolates analyzed. Future studies incorporating detailed lineage analysis will be essential to elucidate transmission dynamics and to monitor the local and regional spread of epidemic CRKP lineages.

Some additional limitations should be noted in this study. Firstly, it was conducted at a single center, a comprehensive tertiary hospital in Bangkok. Furthermore, the sample size of CRKP strains was relatively small, limiting the generalizability of this study's findings to other hospitals and regions in Thailand. Secondly, while this study focused primarily on the resistance determinants for carbapenems, it did not address resistance to other critical antimicrobial classes, such as aminoglycosides and fluoroquinolones. Given the potential co-resistance to these antibiotics, a comprehensive analysis of these agents is essential for a complete understanding of the resistance landscape. Third, variant-level differentiation of $bla_{OXA-48-like}$ genes was not performed, which may obscure important epidemiological and phenotypic differences. Future studies should expand molecular surveillance to include whole-genome sequencing and functional resistance analysis.

Conclusions

This study underscores the existence of carbapenemase-producing *K. pneumoniae*, particularly $bla_{OXA-48-like}$ and bla_{NDM-1}, in the tertiary hospital setting in the Bangkok Metropolitan Area. The results reveal complex resistance gene combinations and significant variation in resistance patterns across hospital wards. The findings provide epidemiological data and insights into the spread of resistance genes in hospital settings, serving as a guide for antibiotic use, surveillance, and the prevention of carbapenem-resistant infections.

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Ethics statement

This study was conducted following the ethical guidelines approved by Human Research Ethics Committee of Thammasat University (Science) (COE 017/2022).

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Conflict of interest

No conflict of interest is declared.

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