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ORIGINAL RESEARCH

The Distribution of Carbapenem-Resistant Acinetobacter Species and High Prevalence of CC92 OXA-23-Producing *Acinetobacter Baumannii* in Community Hospitals in South Korea

Il Kwon Bae, Jun Sung Hong

Department of Companion Animal Health and Science, Silla University, Busan, South Korea

Correspondence: Jun Sung Hong, Department of Companion Animal Health and Science, Silla University, 140 Baegyang-Daero (Blvd), 700beon-Gil Road, Sasang-Gu, Busan, 46958, South Korea, Email jumphong2@nate.com

Background: Clinical isolates of *Acinetobacter* species in South Korea are continuously exhibiting high rates of antimicrobial resistance to carbapenems, indicating that there are public health concerns among both healthcare-associated infections and community-associated infections. The aim of this study was to describe the prevalence and characteristics of carbapenem-resistant *Acinetobacter* isolates originating from community hospitals.

Materials and Methods: A total of 817 non-duplicated *Acinetobacter* species were isolated from December 2022 to July 2023 at long-term care facilities and general hospitals in 16 regions geographically distributed throughout South Korea. Bacterial identification and antimicrobial susceptibility testing were performed using the VITEK-2 system. The bacteria were identified as *Acinetobacter baumannii* by $bla_{\text{OXA-51}}$ PCR and as non-baumannii *Acinetobacter* species by rpoB sequence analysis. The carbapenem resistance genes (OXA-23, OXA-48, OXA-58, IMP, VIM, NDM, GES, and KPC) were identified via PCR and sequencing. The genetic relatedness of carbapenem-resistant *A. baumannii* (CRAB) isolates was assessed by multilocus sequence typing.

Results: A total of 659 *A. baumannii* and 158 non-baumannii *Acinetobacter* isolates, comprising 19 different species, were identified in all 16 regions. The carbapenem resistance rate was 87.4% (n=576) for the *A. baumannii* isolates, and all the strains produced $bla_{\text{OXA-23}}$. For non-baumannii *Acinetobacter*, the rate of carbapenem resistance was 8.9% (n=14); this resistance was primarily caused by $bla_{\text{OXA-23}}$ (n=9), followed by $bla_{\text{NDM-1}}$ (n=3) and $bla_{\text{VIM-2}}$ (n=2). Of the 576 CRAB isolates, clonal complex 92 (CC92) was the predominant genotypes, followed by sequence type 229 (ST229), ST373, ST397, ST447, and ST620.

Conclusion: Our results showed the distribution of *Acinetobacter* species and showed that CC92 CRAB clinical isolates with widespread production of $bla_{\text{OXA-23}}$ were predominant in community hospitals. Our findings suggest that there is a need for urgent and effective methods to reduce carbapenem resistance in *A. baumannii* in South Korea.

Keywords: Acinetobacter, carbapenem, carbapenemase, OXA-23, CC92, community hospitals

Introduction

Acinetobacter species, which are aerobic non-fermentative gram-negative bacilli, are leading primary nosocomial pathogens both in hospital-acquired infections and community-acquired infections.[1,2] To date, *Acinetobacter* isolates are divided into 144 different *Acinetobacter* species, including *Acinetobacter baumannii, Acinetobacter nosocomialis*, and *Acinetobacter pittii*, which are the most frequently identified *Acinetobacter* species, and *Acinetobacter tianfuensis* species and *Acinetobacter rongchengensis* species, which are the *Acinetobacter* species that were most recently identified.[3,4] These organisms often cause respiratory infections, urinary tract infections, meningitis endocarditis, bloodstream infections and burn infections.[5]

Carbapenems are used as one of the first-line choices for treatment due to the extensive antimicrobial resistance in *Acinetobacter* isolates in hospitals. However, when β-lactamases, which can hydrolyze β-lactam antibiotics including

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carbapenems, are acquired, the treatment of *Acinetobacter* infections is difficult because of the acquired carbapenemases.[6] The mechanism of resistance to carbapenems in *A. baumannii* strains has been primarily mediated by the production of OXA-type class D carbapenemases, especially the $bla_{\text{OXA-23}}$ gene associated with insertion sequences (IS), primarily by IS*Aba1*, indicating generally IS*Aba1*-$bla_{\text{OXA-23}}$ structures.[7] In non-baumannii *Acinetobacter* species, class A and B carbapenemases, including a variety of GES-type class A carbapenemases and IMP-, VIM-, and NDM-type metallo-β-lactamases of class B carbapenemases, have been frequently detected.[8]

The outbreak of *A. baumannii* strains harboring $bla_{\text{OXA-23}}$ has been mainly attributed to clonal complex 92 (CC92) or global clone 2 (GC2) in the Oxford scheme worldwide,[9] which means these clones are resistant to carbapenem as major clinical pathogens. Same here in South Korea, the various infections by CC92 *A. baumannii* clinical strains has difficulty due to limited therapeutic options in clinical field.[6,10,11] In South Korea, the prevalence and epidemiology of carbapenem-resistant *Acinetobacter* species have been well reported as hospital-associated pathogens originating from tertiary hospitals and university hospitals in Korea global antimicrobial resistance (AMR) surveillance systems,[10,11] while few reports on the characteristics of carbapenem-resistant *Acinetobacter* species originating from community hospitals have been published.

Here, we describe the prevalence and distribution of carbapenem-resistant *Acinetobacter* species isolates originating from community hospitals, including long-term care facilities and general hospitals, to study nationwide AMR in South Korea.

Materials and Methods

Bacterial Collection and Identification

In this study, we set that above *Acinetobacter* species isolated from two patient groups would have caused community-acquired infections for the following reasons. I) Patient group who have not recently been to a tertiary hospital and are admitted to a long-term care hospital, and II) patient group who have not recently been to a tertiary hospital and have received outpatient care in a local general hospital. All specimens originating from two patient groups were only collected as part of the surveillance of *Acinetobacter* species.

Acinetobacter species were cultured on MacConkey agar (Duksan Science, Seoul, Korea). The identities of the isolates were confirmed by $bla_{\text{OXA-51}}$ PCR to be *A. baumannii* and were identified by *rpoB* gene sequence analysis as non-baumannii *Acinetobacter* species.

A total of 817 non-duplicated *Acinetobacter* clinical isolates were obtained from 817 clinical specimens of patients suspected respiratory infections, urinary tract infections, and bloodstream infections between December 2022 and July 2023 at long-term care facilities and general hospitals in 16 regions geographically distributed throughout South Korea. We only included the strains identified as *Acinetobacter* species using OXA-51 and/or rpoB gene analysis in this study in case positively cultured on MacConkey agar.

Antimicrobial Susceptibility Testing

Acinetobacter isolates were evaluated for ampicillin/sulbactam, piperacillin, piperacillin/tazobactam, cefotaxime, ceftazidime, cefepime, aztreonam, imipenem, meropenem, amikacin, gentamicin, ciprofloxacin, minocycline, tigecycline, cotrimoxazole, and ticarcillin/clavulanic acid susceptibility via VITEK-2 (bioMérieux, Inc., Durham, NC). The quality control strains used were *Escherichia coli* ATCC 25922 and *Pseudomonas aeruginosa* ATCC 27853. The minimal inhibitory concentrations (MICs) of imipenem and meropenem were determined by broth microdilution according to the guidelines of the Clinical and Laboratory Standards Institute (CLSI). The interpretations were performed according to CLSI guidelines (CLSI M100-S32, 2023).

PCR Assays and Sequencing

Detection of the carbapenemase genes ($bla_{\text{OXA-23-like}}$, $bla_{\text{OXA-48-like}}$, $bla_{\text{OXA-58-like}}$, $bla_{\text{IMP-type}}$, $bla_{\text{VIM-type}}$, $bla_{\text{NDM-type}}$, $bla_{\text{GES-type}}$, and $bla_{\text{KPC-type}}$) was carried out in carbapenem-resistant isolates using conventional single PCR and sequence analysis under the following amplification conditions: 94 °C for 5 min; 35 cycles of 94 °C for 30 sec, 55–60 °C for 30 sec and 72 °C for 30 sec;

and a final extension at 72 °C for 5 min using a C1000TM Thermal Cycler (Bio-Rad, Hercules, CA). The sequences were compared to published DNA sequences using BLAST. The primers used in this study are summarized in Table S1.

Epidemiological Typing

The epidemiological relationships among the bla_{OXA-23}-producing *A. baumannii* isolates were analyzed by multilocus sequence typing (MLST). PCR amplification and sequencing of partial fragments of seven housekeeping genes (*gltA, gyrB, gdhB, recA, cpn60, gpi* and *rpoD*) were performed according to the Oxford scheme, and the experimentally determined amplicons of both strands were compared to preexisting sequences in the MLST database to assign allelic numbers and sequence types (STs) (https://pubmlst.org/organisms/acinetobacter-baumannii).[12] Clonal complexes (CCs) are defined as groups of STs with similar allelic profiles; every ST shares at least five or more of the seven alleles that are involved in predicting ancestral epidemiological genotype using the BURST algorithm.[13] In this regard, five genes (*gltA, gdhB, recA, cpn60*, and *rpoD*) were amplified to assign CC92 (the allelic profile is *gltA*-1, *gdhB*-3, *recA*-2, *cpn60*-2, and *rpoD*-3) in this study. If different allele numbers were obtained, the *gyrB* and *gpi* genes were amplified to complete the ST.

Ethics Statement and Patients Consent

The Ethics Committee of Silla University approved the study. All patients were included after providing written informed consent and/or assent as appropriate. This research did not involve an interventional study. For personal protection, we were only provided with anonymized data including isolated sample type and patients' address. Therefore, this study was conducted following the Declaration of Helsinki.

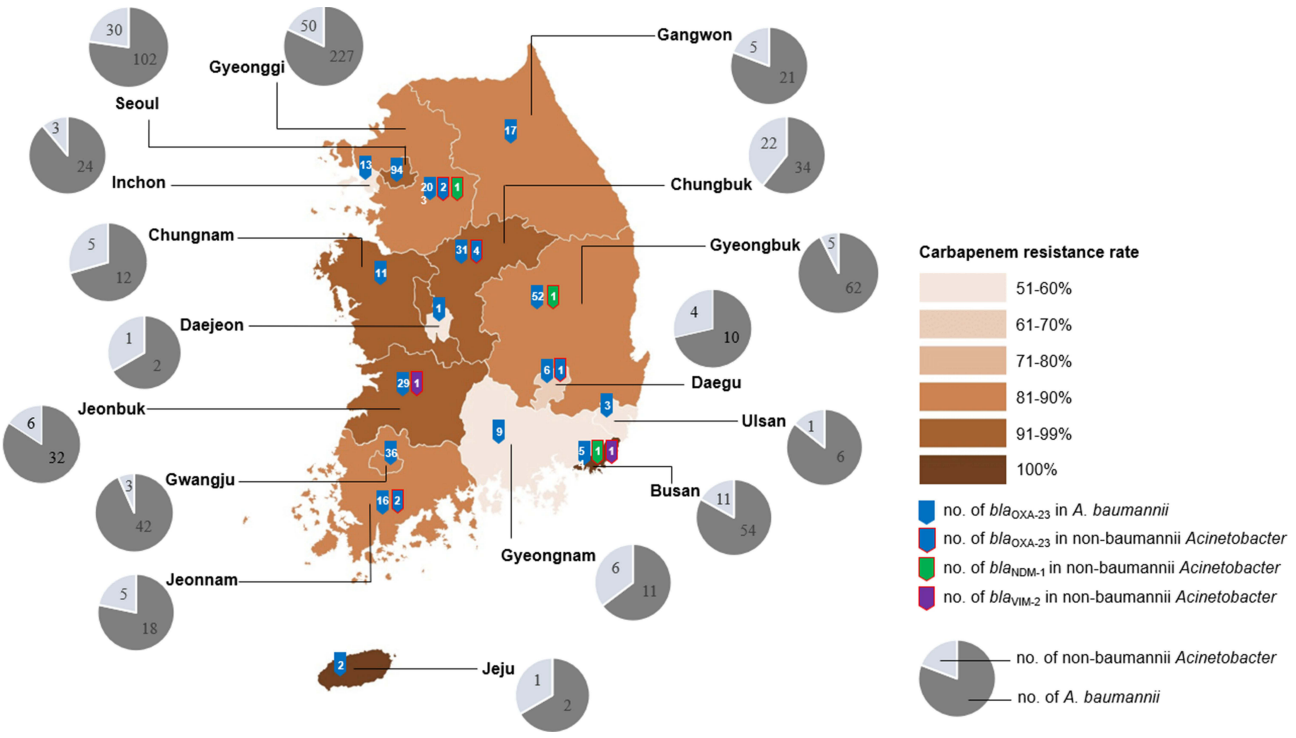

Figure 1 Map showing the prevalence and characteristics of *Acinetobacter* clinical isolates in 16 regions in South Korea. The shade of Orange indicates the percentage each of carbapenem-resistant *Acinetobacter* species in this study. The gray and dark gray colors in the circle indicate the number of non-baumannii *Acinetobacter* and *A. baumannii* isolates, respectively. The white numbers in each colored pentagon arrow indicate the carbapenem resistance determinants.

Results

The prevalence of *Acinetobacter* clinical isolates identified from each of the 16 regions geographically distributed throughout South Korea is shown in Figure 1. A total of 817 non-duplicated *Acinetobacter* species isolates were collected, consisting of 659 *A. baumannii* harboring *blaOXA-51* variants, 55 *A. pittii*, 20 *Acinetobacter bereziniae*, 19 *A. nosocomialis*, 19 *Acinetobacter johnsonii*, 16 *Acinetobacter lwoffii*, 5 *Acinetobacter junii*, 4 *Acinetobacter ursingii*, 4 *Acinetobacter seifertii*, 4 *Acinetobacter soli*, 3 *Acinetobacter guillouiae*, 2 *Acinetobacter radioresistens* and one *Acinetobacter baylyi, Acinetobacter colistiniresistens, Acinetobacter courvalinii, Acinetobacter dispersus, Acinetobacter haemolyticus, Acinetobacter oleivorans* and *Acinetobacter vivianii*. The list of above 158 non-baumannii *Acinetobacter* species isolates from each of the 16 regions is shown in Table S2.

Among the all 817 samples positively cultured from various clinical specimens on MacConkey agar, the predominant sample type was sputum (n=439, 53.7%), urine (n=195, 23.9%), and wound/pus (n=117, 14.3%), followed by other sample types (n=23), blood samples (n=17), throat swabs (n=9), vaginal discharge (n=5), nasal swabs (n=4), catheter tips (n=3), ear discharge (n=3), and body fluids (n=2).

The antimicrobial susceptibility profiles of the *A. baumannii* and non-baumannii *Acinetobacter* isolates are shown in Figure 2. The resistance rate to carbapenems (imipenem and meropenem) was 87.4% (n=576) in the *A. baumannii* clinical isolates. Among the non-baumannii *Acinetobacter* isolates, 8.9% were carbapenem-resistant (n=14). The level of carbapenem resistance (R%) in 16 regions of South Korea represent in Figure 1.

Above 590 carbapenem-resistant *Acinetobacter* isolates also exhibited prominently high rates (ca. >90%) of resistance to piperacillin, piperacillin/tazobactam, cefotaxime, ceftazidime, cefepime, aztreonam, gentamicin, ciprofloxacin, trimethoprim/sulfamethoxazole, and ticarcillin/clavulanic acid, in contrast to the resistance rates to tigecycline (43.2% 255/590), amikacin (16.3%, 96/590), and minocycline (10.2% 60/590).

The main mechanism of resistance to carbapenems in *A. baumannii* isolates is the production of oxacillinases encoded by $bla_{\text{OXA-23-like}}$ without the class A or B carbapenemases, while the production of $bla_{\text{NDM-1}}$ (n=3) and $bla_{\text{VIM-2}}$ (n=2) with

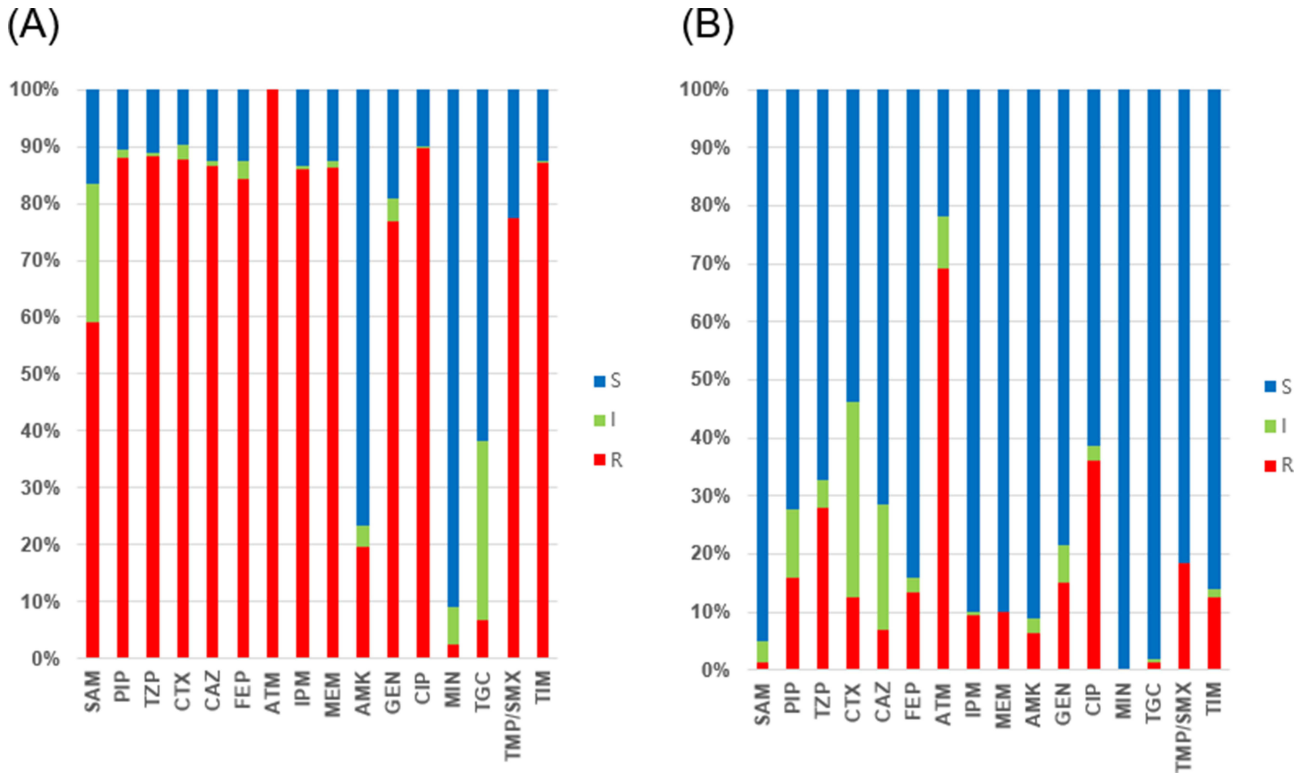

Figure 2 Antimicrobial susceptibility of *Acinetobacter* clinical isolates in this study. (**A**) *A. baumannii* isolates (n=659); (**B**) Non-baumannii *Acinetobacter* species (n=158).
Abbreviations: S, susceptible; I, intermediate; R, resistant; SAM, Ampicillin/sulbactam; PIP, piperacillin; TZP, Piperacillin/tazobactam; CTX, cefotaxime; CAZ, ceftazidime; FEP, cefepime; ATM, aztreonam; IPM, imipenem; MEM, meropenem; AMK, amikacin; GEN, gentamicin; CIP, ciprofloxacin; MIN, minocycline; TGC, tigecycline; TIM, Ticarcillin/Clavulanic Acid.

Table 1 The Profiles of 14 Carbapenemase-Producing Non-Baumannii Acinetobacter Isolates

Isolate	Specimen	Identification	Region	Carbapenemase	MIC (μg/mL)		Co-resistant to:
					IPM	MEM	
Eone73	Urine	*A. pittii*	Gyeonggi	OXA-23	16	16	PIP, TZP, CTX, CAZ, FEP, ATM, GEN, CIP, TIM
Eone203	Sputum	*A. pittii*	Daegu	OXA-23	16	16	PIP, TZP, CTX, CAZ, FEP, ATM, GEN, CIP, TIM
SCAB205	Urine	*A. bereziniae*	Busan	VIM-2	16	8	PIP, TZP, CTX, CAZ, ATM, AMK, GEN, CIP, TMP/SMX, TIM
SCAB220	Urine	*A. bereziniae*	Gyenoggi	OXA-23	16	16	PIP, TZP, CAZ, FEP, ATM, CIP, TMP/SMX, TIM
SCAB226	Sputum	*A. seifertii*	Jeonnam	OXA-23	32	16	SAM, PIP, TZP, CTX, CAZ, FEP, ATM, CIP, TMP/SMX, TIM
SCAB228	Sputum	*A. pittii*	Chungbuk	OXA-23	32	32	PIP, TZP, CTX, CAZ, FEP, ATM, GEN, CIP, TIM
SCAB251	Urine	*A. nosocomialis*	Gyenoggi	NDM-1	128	64	SAM, PIP, TZP, CTX, CAZ, FEP, ATM, CIP, TMP/SMX, TIM
SCAB253	Urine	*A. bereziniae*	Busan	NDM-1	128	64	SAM, PIP, TZP, CTX, CAZ, FEP, ATM, AMK, GEN, CIP, TGC, TIM
SCAB366	Sputum	*A. nosocomialis*	Chungbuk	OXA-23	16	32	PIP, TZP, CTX, CAZ, FEP, ATM, GEN, CIP, TIM
SCAB392	Sputum	*A. nosocomialis*	Chungbuk	OXA-23	32	32	PIP, TZP, CTX, CAZ, FEP, ATM, GEN, CIP, TIM
SCAB419	Sputum	*A. nosocomialis*	Chungbuk	OXA-23	32	32	PIP, TZP, CTX, CAZ, FEP, ATM, GEN, CIP, TIM
SCAB444	Other	*A. bereziniae*	Jeonbuk	VIM-2	16	8	PIP, TZP, CTX, CAZ, ATM, AMK, GEN, CIP, TMP/SMX, TIM
SCAB445	Sputum	*A. seifertii*	Jeonnam	OXA-23	32	16	SAM, PIP, TZP, CTX, CAZ, FEP, ATM, CIP, TMP/SMX, TIM
SCAB472	Urine	*A. bereziniae*	Gyeongbuk	NDM-1	64	32	SAM, PIP, TZP, CTX, CAZ, FEP, ATM, GEN, CIP, TMP/SMX, TIM

Abbreviations: SAM, Ampicillin/sulbactam; PIP, piperacillin; TZP, Piperacillin/tazobactam; CTX, cefotaxime; CAZ, ceftazidime; FEP, cefepime; ATM, aztreonam; IPM, imipenem; MEM, meropenem; AMK, amikacin; GEN, gentamicin; CIP, ciprofloxacin; MIN, minocycline; TGC, tigecycline; TIM, Ticarcillin/Clavulanic Acid.

bla$_{OXA-23}$ (n=9) occurred in 14 non-baumannii *Acinetobacter* species. The Information of the 14 carbapenemase-producing non-baumannii *Acinetobacter* isolates is presented in Table 1.

The imipenem and meropenem MIC ranges of the 576 carbapenem-resistant *A. baumannii* (CRAB) isolates were 32 - >256 μg/mL for both antibiotics. The carbapenem MIC range of the 14 carbapenem-resistant non-baumannii *Acinetobacter* isolates was 16–128 μg/mL for imipenem and 16–64 μg/mL for meropenem (Table 1). The MIC_{50} and MIC_{90} of carbapenems in the 576 CRAB isolates were 64 μg/mL and 128 μg/mL for imipenem and meropenem, respectively.

MLST typing was performed on all 576 CRAB isolates harboring *bla*$_{OXA-23}$. The results of MLST analysis revealed that the genotype of the CRAB isolates was relatively simple. A total of two STs (ST373 and ST447) were identified as singletons, and four CCs (CC20, CC92, C108, and CC110) were identified. MLST analysis revealed that CC92 or global clone 2 (GC2) (n=519, 90.1%) was the most prevalent clone, and this clone was found in every region. Furthermore, ST229 belonging to CC110 was identified (n=47, 8.2%), followed by ST373 (n=7) and ST447 (n=1) as a singleton, one ST397 belonging to CC108 or GC1. and one ST620 belonging to CC20 [Table 2].

Discussion

Acinetobacter species are known as opportunistic pathogens that commonly cause nosocomial infections in hospitalized patients.[14] Because *Acinetobacter* clinical isolates generally exhibit multidrug resistance pattern, carbapenems have been regarded as effective treatment options for *Acinetobacter* infections in hospitals. However, the increasing use of

Table 2 The Characteristics of A. Baumannii Related to Sequence Types (STs) and Clonal Complexes (CCs) Identified in This Study

GC (no. of isolates)	Species (Carbapenemase)	CC	ST	Allele							No. of Isolates
				gltA	*gyrB*	*gdhB*	*recA*	*cpn60*	*gpi*	*rpoD*	
GC2 (519)	*A. baumannii* (OXA-23)	92	NE	1	NE	3	2	2	NE	3	519
GC1 (1)		108	397	10	53	4	6	4	50	5	1
Non-GC (56)		110	229	1	15	2	28	1	107	32	47
		20	620	1	1	13	12	4	16	2	1
		Singleton	373	1	12	12	11	4	13	3	7
			447	1	15	13	12	4	16	2	1

Abbreviations: GC, global clone; CC, clonal complex; ST, sequence type; NE, not estimable.

carbapenems has resulted in the prevalent emergence of carbapenem-resistant *Acinetobacter* species, especially CRAB isolates.[6,15] After many reports indicating the widely spread of CRAB across several provinces in China,[16,17] CRAB outbreaks have been regarded as a global nosocomial menace.[9,18] In most cases, they can become resistant to carbapenems through the production of the $bla_{\text{OXA-23}}$ gene, which is the major mechanism involved in the acquisition of carbapenem resistance.[3] According to reports from South Korea global AMR surveillance systems,[10,11] the rates of resistance to carbapenems mostly associated with $bla_{\text{OXA-23}}$ were 89.9% in 2016 and 90.5% in 2017–2019, for *A. baumannii* isolates originating from tertiary hospitals and university hospitals. In this study, 87.4% of the *A. baumannii* isolates originating from community hospitals were resistant to carbapenems. Based on these results, CRAB has already become a menacing pathogen in South Korea, regardless of whether the infections are hospital-acquired or community-acquired. Therefore, there is a need for effective guidelines to reduce carbapenem resistance in *A. baumannii* clinical isolates in South Korea.

The widely disseminated CRAB isolates were mostly associated with the CC92 or GC2 genotypes harboring the $bla_{\text{OXA-23}}$ gene, which is the most common acquired carbapenemase, indicating that the CC92 CRAB has been identified as the major genotypes related to carbapenem resistance worldwide, as well as in Asia.[9,19,20] In South Korea, CC92 CRAB is also responsible for the high resistance rate to carbapenems. According to a 2008 report,[21] 73.8% (n=299/405) of CRAB clinical isolates were CC92 CRAB. Similar to that in 2017,[6] the percentage of CC92 CRAB isolates mostly associated with $bla_{\text{OXA-23}}$ from tertiary hospitals and university hospitals in South Korea was confirmed to increase to 82.0% (n=291/355). In 2023, CC92 CRAB harboring $bla_{\text{OXA-23}}$ was also the most common genotypes (90.1%, n=519) among the 576 CRAB isolates collected from community hospitals [Table 2]. In addition, according to an observational study that took place during 2001–2020,[22] the predominant CRAB genotypes causing invasive infections in children changed from non-CC92 to CC92 as a result of the 2010–2011 outbreaks. According to several of the abovementioned studies, it is considered that the CC92 lineage of CRAB clinical isolates has become the major genotypes throughout South Korea.

In addition to the CC92 CRAB isolates identified in this study, a large number of ST229 CRAB strains belonging to the CC110 (n=47) were isolated from a general hospital in the Seoul region. The ST229 CRAB isolate was originally known as the epidemic clone in 2015 in the Daegu region of South Korea.[23,24] These strains mostly carried IS*AbaI*-$bla_{\text{OXA-51}}$, but all the ST229 CRAB isolates in this study carried $bla_{\text{OXA-23}}$, similar to the findings in 2017.[6] A total of 47 ST229 multidrug-resistant *A. baumannii* isolates exhibited the same antimicrobial susceptibility profiles: co-resistant to SAM, AZT, CIP, FEP, GEN, PIP, TMP/SMX, CTX, CAZ, TIM, and TZP. In this regard, the CRAB ST229 isolate seems to be an outbreak caused by clonal spread in a general hospital. Without this clonal spread of ST229 CARB in this study, the ratio of CC92 CRAB isolates would have been slightly higher.

ST373 CRAB (n=7) was the third most prevalent genotype of strains in this study. ST373 *A. baumannii* isolates are known as hyper-biofilm forming *A. baumannii*.[25] In a previous study, only one ST373 CRAB isolate was detected in South Korea.[6] Finally, each one of ST397 belonging to CC108, ST447, and ST620 belonging to CC20 CRAB isolates has

seldom been reported in South Korea.[6,9,26] The emergence of these rare genotypes is considered to be a distinct characteristic that originated from community-associated infections.

The correct identification of clinical *Acinetobacter* isolates is important because *A. baumannii* and non-baumannii *Acinetobacter* species generally exhibit different AMR pattern.[3] Automatic systems such as VITEK-2 may be suitable for antimicrobial susceptibility testing, but the quick and correct detection of *Acinetobacter* infections is challenging to date.[27] Therefore, we confirmed the identification of *Acinetobacter* species by analyzing the *Acinetobacter rpoB* gene sequence as the gold standard for molecular analysis. In general, *A. baumannii, A. nosocomialis*, and *A. pittii* in order are the most frequently found causative species of healthcare-associated *Acinetobacter* infections.[3,8] Among the *Acinetobacter* species in this study, 19 different species were identified, including *A. baumannii* (n=659), *A. pittii* (n=55), and *A. nosocomialis* (n=19), as well as *A. courvalinii, A. dispersus*, and *A. vivianii*. Hemolytic and/or proteolytic *A. courvalinii, A. dispersus*, and *A. vivianii* of the genus *Acinetobacter* were previously classified into three putative species terms as genomic species 14BJ, genomic species 17, and taxon 20, respectively.[28] To the best of our knowledge, these three strains have never been found in healthcare-associated infections in South Korea. Our findings cannot demonstrate the epidemiological origin of unique and diverse species, but their existence seems to be a feature of community-associated infections. In other words, unlike healthcare-associated infections, various species may be detected in community-associated infections among *Acinetobacter* species.

For non-baumannii *Acinetobacter* species, the resistance rate to carbapenems was 8.9% (n=14). These results are similar to those of the 2017 study in South Korea.[6] These 14 carbapenem-resistant non-baumannii *Acineotobacter* species carried the three types of carbapenemase (9 $bla_{\text{OXA-23}}$, 3 $bla_{\text{NDM-1}}$, and 2 $bla_{\text{VIM-2}}$) in *A. bereziniae, A. nosocomialis, A. pittii*, and *A. seifertii* [Table 1]. The $bla_{\text{OXA-23}}$ gene, which is mostly associated with *A. baumannii* or *A. nosocomialis*, is normally found on the chromosome rather than on a plasmid.[21] However, reports of *A. bereziniae, A. pittii*, and *A. seifertii* isolates harboring $bla_{\text{NDM-1}}$ and $bla_{\text{VIM-2}}$ are rare in South Korea.[11] In other Asian countries, Pakistan in 2022 report,[29] the $bla_{\text{VIM-type}}$ (n=74/200, 37.0%) and $bla_{\text{NDM-type}}$ (n=28/200, 14.0%) for carbapenemases were detected in *Acinetobacter calcoaceticus-baumannii* complex isolates. Because these $bla_{\text{NDM-1}}$ and $bla_{\text{VIM-2}}$ genes are generally found on a plasmid,[30] the possibility of AMR dissemination through horizontal transfer across close countries cannot be ignored because *Acinetobacter* species originating from community-associated infections could be new carriers.

In this study, the total number and geographical distribution of *Acinetobacter* clinical isolates in South Korea were sufficient to describe the prevalence of the *Acinetobacter* species. However, a limitation is that the number of isolates collected from the Daejeon, Jeju, and Ulsan regions was so small that it was difficult to represent the exact AMR pattern.

Conclusion

Our results revealed the distribution of *Acinetobacter* species and revealed a predominance of CC92 CRAB clinical isolates with widespread production of $bla_{\text{OXA-23}}$ in community hospitals in South Korea. These results were similar to that observed in tertiary hospitals and university hospitals. In addition, we identified rare STs and newly detected species, suggesting that distinct features of *Acinetobacter* species originating from community-associated infections. Our findings suggest that there is a need for effective methods to reduce carbapenem resistance in *A. baumannii* in South Korea.

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Disclosure

The authors report no conflicts of interest in this work.

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