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Relationship of multidrug-resistant gene and extended-spectrum carbapenem-resistance in *Staphylococcus aureus*

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Abstract: The aim of this study was to determine the relationship between phenotypic antimicrobial susceptibility patterns and extended-spectrum, carbapenem-resistance genes. A total of 109 clinical *Staphilococcus aureus* strains were subjected to 19 antimicrobial susceptibility tests. Resistance to methicillin (mecA), penicillin (blaTEM), and tetracycline (tetM) was detected. We compared the presence of the blaTEM genes with extended-spectrum, carbapenem-related genes and identified the types of SCCmec genes. Of 109 clinical *S. aureus* strains, 62 (56.88%) had methicillin resistance and 60 strains carried mecA. The prevalence of blaTEM and tetM genes was 81.65% and 37.61%, respectively. The most predominant SCCmec type was SCCmec type II 28/60 (46.67%), in 60 mecA-positive methicillin-resistant *S. aureus* (MRSA) isolates. The SCCmec prevalence rates were type IVA 30.00% (18/60), type IVb 8.33% (5/60), type IVd 6.67% (4/60), and non-typable 8.33% (5/60). Sixty of the 109 (55.05%) MRSA isolates were positive for extended-spectrum carbapenems (31/60) (51.67%), cephalosporins 40/60 (66.67%) and carbapenems 31/60 (51.67%). The predominant SCCmec type II demonstrated more carbapenem-resistance than the IVA, IVb and IVd types.

Introduction

Methicillin-resistant *Staphylococcus aureus* (MRSA) is a prominent pathogen that causes severe infections in both healthcare and community settings (Inomata *et al*., 2015). MRSA in hospital settings is more prevalent in Asian countries, such as South Korea, China, and Japan, with reported rates of 70-80%, than in Europe (25.1%) (Reinert *et al*., 2007; Song *et al*., 2011). In one recent study, the proportion of MRSA in healthcare-associated (HA) isolates was very high (73.3%) (Moon *et al*., 2014). Although the rates of community-associated (CA) MRSA infections are still very low in South Korea, the recently reported rates of MRSA isolates have been unclear identify those from CA settings (Kim *et al*., 2007; Moon *et al*., 2014).

MRSA infections are difficult to treat because they are resistant to many groups of antibiotics, such as β-lactams, tetracyclines, aminoglycosides, and macrolides. MRSA is resistant to all penicillins, including semi-synthetic penicillinase-resistant congeners, carbapenems, cephalosporins, and penems. One mechanism of penicillin resistance is the expression of penicillinase, which hydrolyzes the β-lactam ring of penicillin, inactivating it (Olsen *et al*., 2006).

The principal mechanism of aminoglycoside resistance in *S. aureus* is drug inactivation mediated by aminoglycoside-modifying enzymes (AMEs) encoded by various genes such as *aac(6')-aph(2")* and *ant(4')-Ia* (Ramirez and Tolmasky, 2010). The most prevalent AME in *S. aureus* is bifunctional enzyme AAC(6')-APH(2"), which is encoded by *aac(6')-aph(2")* (Martineau *et al*., 2000). In addition, ANT(4')-I, encoded by *ant(4')-Ia,* has been found in *S. aureus* (Schmitza *et al*., 1999).

Meanwhile, erythromycin resistance is associated with *erm*(A) and *erm*(C) genes, and the main gene associated with the tetracycline resistance of *S. aureus* is *tet*M (Trzcinski *et al*., 2000; Varaldo *et al*., 2009).

S. aureus can acquire antibiotic resistance genes through horizontal gene transfer using mobile genetic elements, including SCC*mec*, plasmids, transposons, insertion sequences, and bacteriophage (McCarthy *et al*., 2014). SCC*mec* elements are important for MRSA because they usually serve as determinants of antibiotic resistance patterns. Healthcare-associated MRSA strains usually harbor type I-III SCC*mec* elements that confer multidrug resistance (MDR) (Uhlemann *et al*., 2014), while community-associated strains

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are generally non-MDR strains carrying small SCC*mec* elements, most of which are types IV and V (Kondo *et al*., 2007; Orlin *et al*., 2017).

The objective of this study was to evaluate the relationship between phenotypic antimicrobial susceptibility patterns and extended-spectrum carbapenem genes in isolated bacterial strains. We also compared the prevalence of genes with SCC*mec* resistance with bla_{TEM} and extended-spectrum carbapenem antibiotic resistance among isolated clinical *S. aureus* strains.

Material and Methods

Bacterial isolates

A total of 109 *S. aureus* strains were obtained from clinical patients at Gachon University Gil Medical Center in South Korea between April 2016 and June 2018. The research was approved by the Ethics Committee of Gil Hospital, Gachon University of Medicine. The *S. aureus* strains were streaked onto sheep blood agar (Sinyang Diagnostics, Seoul, Korea) and transported to our laboratory after culture. One colony was picked from each blood agar plate and incubated in lysogeny broth with shaking (80 rpm) at 37°C overnight. The isolates were preserved in 20% glycerol (vol/vol) and stored at -80°C until further use.

Antimicrobial susceptibility testing

Antimicrobial susceptibility was determined using the Kirby-Bauer disc diffusion method described by the Clinical and Laboratory Standard Institute (CLSI) guidelines, 2015 (Wayne, 2015). Each bacterial suspension was adjusted to McFarland 0.5 turbidity, swabbed onto lysogeny broth agar, and incubated in the presence of antibiotic discs at 37°C for 18 h. We tested the following 19 antibiotic Liofilchem discs (Liofilchem, Roseto degli Abruzzi, Italy): penicillin G (10 IU), methicillin (5 μg), kanamycin (30 μg), gentamicin (10 μg), streptomycin (10 μg), tetracycline (30 μg), erythromycin (15 μg), vancomycin (30 μg), chloramphenicol (30 μg), amoxicillin (25 μg), ticarcillin (75 μg), piperacillin (100 μg), cefepime (30 μg), cefotaxime (30 μg), ceftazidime (30 μg), imipenem (10 μg), ertapenem (10 μg), and meropenem (10 μg). The diameters of inhibition zones were measured, and each isolate was identified as resistant or susceptible to the antimicrobial agents based on CLSI guidelines. We obtained the *S. aureus* control strain *Staphylococcus aureus* ATCC 29213 from the Korean Culture Center of Microorganisms (Seodaemun-gu, Seoul, Korea).

TABLE 1

Primers used for SCC*mec* typing

Primers	Oligonucleotide sequence (5'→3')	Amplicon size (bp)	Specificity	Reference	GenBank no.
MecA147-F	GTGAAGATATACCAAGTGATT	147	mecA	Zhang	
MecA147-R	ATGCGCTATAGATTGAAAGGAT				
Type I-F	GCTTTAAAGAGTGTCGTTACAGG	613	SCC*mec* I	Zhang	
Type I-R	GTTCTCTCATAGTATGACGTCC				
Type II-F	CGTTGAAGATGATGAAGCG	398	SCC*mec* II	Zhang	
Type II-R	CGAAATCAATGGTTAATGGACC				
TypeIII-F	CCATATTGTGTACGATGCG	280	SCCmec III	Zhang	
TypeIII-R	CCTTAGTTGTCGTAACAGATCG				
TypeIVa-F	GCCTTATTCGAAGAAACCG	766	SCC*mec*	Zhang	
TypeIVa-R	CTACTCTTCTGAAAAGCGTCG				
TypeIVb-F	TCTGGAATTACTTCAGCTGC	493	SCC*mec*	Zhang	
TypeIVb-R	AAACAATATTGCTCTCCCTC				
TypeIVc-F	ACAATATTTGTATTATCGGAGAGC	200	SCC*mec*	Zhang	
TypeIVc-R	TTGGTATGAGGTATTGCTGG				
TypeIVd-F	CTCAAAATACGGACCCCAATACA	881	SCC*mec*	Zhang	
TypeIVd-R	TGCTCCAGTAATTGCTAAAG				
TypeIVA-F	TTACCACGCTTGTTGATGGTA	1752	SCC*mec*	This study	EU437549.2
TypeIVA-R	ACAATGATGGACAATGACTGTGA				
TypeV-F	GAACATTGTTACTTAAATGAGCG	325	SCC*mec* V	Zhang	
TypeV-R	TGAAAGTTGTACCCTTGACACC				

Genomic DNA isolation

Genomic DNA was isolated after alkaline cell lysis, phenol-chloroform DNA extraction, and ethanol DNA precipitation (Sambrook and Russell, 2006). A single colony was picked from each blood agar plate and incubated in lysogeny broth at 37°C overnight. Then, 1.5 mL of the bacterial suspension was harvested by centrifugation at 14000 rpm for 30 s. The harvested bacterial pellet was proceeded protocol alkaline phenol-chloroform method using fresh tubes and phenol-chloroform (1:1) solution (Bioneer, Daejeon, Korea). The DNA pellet was then dissolved in 30 µL autoclaved tri-distilled water. DNA concentrations were determined using a NanoDrop™ spectrophotometer (Thermo Fisher Scientific, Waltham, MA, USA).

Identifying mecA, bla$_{TEM}$ and SCCmec genes by multiplex real-time PCR

The PCR primers used to detect the *mec*A and *bla*$_{TEM}$ genes are listed in Tabs. 1 and 2 (Strommenger *et al*., 2003; Zhang *et al*., 2005; Varaldo *et al*., 2009). The following reaction mixture was added to each sample: 10 pmol of each primer, 2 µL DNA (100 ng), and 10 µL iQ™ SYBR® Green supermix (2X reaction buffer with dNTPs, iTaq DNA polymerase, SYBR® Green I, fluorescein, and stabilizers (Bio-Rad, Hercules, CA, USA)). The volume was adjusted to 20 µL by adding autoclaved triple-distilled water. The PCR cycling conditions on a thermal cycler (iQ5, Bio-Rad and TC-512, TECHNE, Cambridge, UK) were as follows: 94°C for 3 min, followed by 35 cycles of denaturation at 94°C for 30 s, annealing at 56°C for 30 s, and extension at 72°C for 45 s. The reaction was ended with a final extension step at 72°C for 10 min. Multiplex PCR was carried out for SCC*mec* typing using nine pairs of primers specific for SCC*mec* types I, II, III, IVa, IVA, IVb, IVc, IVd, and V (Zhang *et al*., 2005). The PCR products were subjected to electrophoresis using 2% agarose gel in 1X TBE buffer at 100 V for 25 min. A 100 bp DNA ladder (Bioneer, Daejeon, Korea) was used as a molecular size marker. The PCR products in gels were then visualized with Safe Green loading dye (Applied Biological Materials, Inc, Vancouver, BC, Canada).

Detecting antibiotics resistance genes

PCR was performed to detect genes associated with antimicrobial resistance. The oligonucleotide primers sequences and specific genes are listed in Tab. 2 (Strommenger *et al*., 2003; Varaldo *et al*., 2009). The examined genes were the tetracycline resistance gene (*tetM*), aminoglycoside-modifying enzyme (AME) genes (*ant4'-Ia*, *aac6'-aph2"*), and macrolide resistance genes (*ermA*, *ermC*). These products were determined the existence of PCR result and DNA sequencing.

Results

We tested for antimicrobial susceptibility using Kirby-Bauer disc diffusion and determined the isolates to be resistant or susceptible to the antimicrobial agents based on the diameters of the inhibition zones. The susceptibility testing showed that 56.88% (62/109) of the *S. aureus* strains were resistant to methicillin. The results also showed high rates of susceptibility to chloramphenicol 107/109 (98.16%) and vancomycin 107/109 (98.16%), however, most of the *S. aureus* strains were resistant to streptomycin 70/109 (64.22%) and penicillin 71/109 (65.14%). The overall rates of resistance to kanamycin, gentamicin, erythromycin, and tetracycline were 54.13%, 52.29%, 37.61%, and 26.61%, respectively (Tab. 3).

TABLE 2

Primers used for detecting antibiotic-resistance determinants in *S. aureus* isolates

Antibiotic	Primer	Oligonucleotide sequence	Amplicon	specific gene	Reference	GenBank.
β-lactams	TEM-F	GCA CGA GTG GGT TAC ATC GA	311	*bla*TEM	This study	NG_050162.1
	TEM-R	GGT CCT CCG ATC GTT GTC AG				
Tetracyclines	tet(M)-F	GGT TGG AAT GTG ACG GAC TG	200	*etM*	This study	LS483319.1
	tet(M)-R	ATC GTT GTA TGC TCG TGA AAG A				
Aminoglycosides	kan-F	GAA GCA GAG TTC AGC CAT GA	390	*ant(4')-Ia*	This study	CP019563.1
	kan-R	CGA AGC GCT CGT CGT ATA AC				
	AAC(6')-APH(2")-F	CCA AGA GCA ATA AGG GCA TA	222	*aac(6')-aph(2")*	Strommenger	
	AAC(6')-APH(2")-R	CAC TAT CAT AAC CAC TAC CG				
Macrolides	erm(A)-F	AAG CGG TAA ACC CCT CTG A	199	*ermA*	Varaldo	
	erm(A)-R	ACAATGATGGACAATGACTGTGA				
	erm(C)-F	AAT CGT CAA TTC CTG CAT GT	299	*ermC*	Varaldo	
	erm(C)-R	TAA TCG TGG AAT ACG GGT TTG				

TABLE 3

Phenotypic antibiotic resistance patterns and rates of antibiotic resistance genes in *S. aureus*

Antibiotic	Resistant strains No. (%)	Susceptible strains No. (%)	PCR positive strains No. (%)	PCR negative strains No. (%)
Methicillin	62 (56.88%)	47 (43.12%)	*mecA* 60 (55.05%)	*mecA* 49 (44.95%)
Penicillin G	71 (65.14%)	38 (34.86%)	bla_{TEM} 89 (81.65%)	bla_{TEM} 20 (18.35%)
Kanamycin	59 (54.13%)	50 (45.87%)	*ant(4')-Ia* 32 (29.36%) *aac(6')-aph(2")* 32 (29.36%) *ant(4')-Ia+aac(6')-aph(2")* 12 (11.01%) total 52 (47.41%)	*ant(4')-Ia/aac(6')-aph(2")*57 (52.29%)
Erythromycin	41 (37.61%)	68 (62.36%)	*ermA* 34 (31.19%) *ermC* 2 (1.83%) total 36 (33.03%)	*ermA/ermC* 73 (66.97%)
Gentamicin	57 (52.29%)	52 (47.71%)	*aac(6')-aph(2")* 32 (29.35%)	*aac(6')-aph(2")* 77 (70.64%)
Tetracycline	29 (26.61%)	80 (73.39%)	*tetM* 41 (37.61%)	*tetM* 68 (62.39%)
Streptomycin	70 (64.22%)	39 (35.78%)		
Vancomycin	2 (1.83%)	107 (98.16%)	*van*A, *van*B (not detected)	
chloramphenicol	2 (1.83%)	107 (98.16%)		

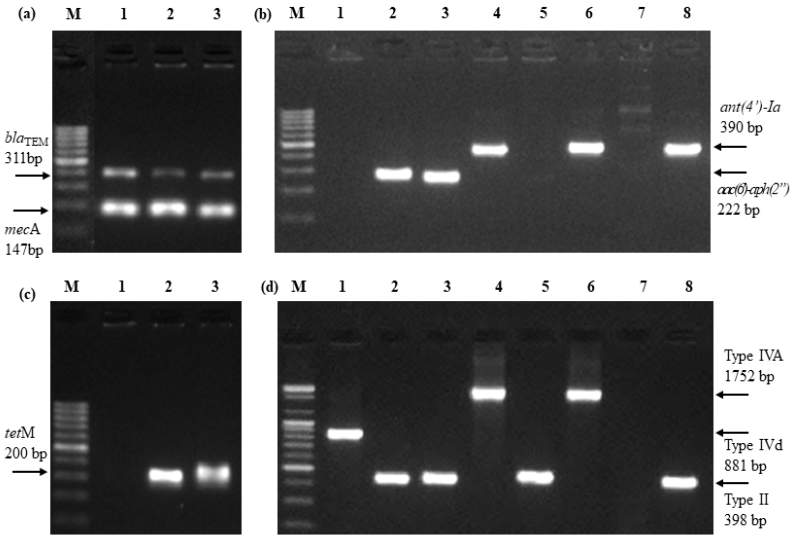

FIGURE 1. *mecA*, la_{TEM}, *ant(4')-Ia*, and *aac(6')-aph(2")* detection by polymerase chain reaction (PCR).The PCR results were visualized on 2% agarose gel stained with Safe Green loading dye. Lane M, 100 bp DNA ladder. (a) Multiplex PCR detected line no 1-3, *mecA* (147 bp) and bla_{TEM} (311 bp), (b) line no 4, 6, 8: AME genes *ant(4')-Ia* (390 bp), and line 2, 3: *aac(6')-aph(2")* (222 bp), line 1, 5, 7 was not detected in *S. aureus* strains. (c) Multiplex PCR detected line no 2, 3: *tetM* (200bp), line 1 was not detected in *S. aureus* strains. (d) Multiplex PCR forSCC*mec* typing, Lane M:100 bp DNA ladder; Lane 1: type IVd (881 bp) SCC*mec*-positive strain; Lanes 2, 3, 5, 8: type II(398 bp) SCC*mec*-positive strains; Lanes 4, 6: type IVA (1752bp) SCC*mec*-positive strains; Lane 7: non-typable *S. aureus* strains.

The susceptibility testing also showed that 81/109 (74.3%) of the *S. aureus* strains were susceptible to amoxicillin (AML) and resistance to piperacillin 42/109 (38.53%) and cefotaxime 27/109 (24.77%) were found, as well. Tab. 3 displays the correlations between methicillin-resistance and the presence of *mecA*. A total of 62 MRSA strains were resistant to methicillin, 60 strains were *mecA*-positive, and two strains were *mecA*-negative (Tab. 3). Forty-seven (43.12%) strains of *S. aureus* were susceptible to methicillin. The relationship between penicillin resistance and the presence of bla_{TEM} is summarized in Tab. 3. Seventy-one (65.14%) *S. aureus* strains were resistant to penicillin based on disc diffusion, and 89 of them were positive for bla_{TEM} (Tab. 3, Fig. 1(A)).

Tab. 3 shows the correlations between kanamycin resistance and the presence of *ant(4')-Ia* and *aac(6')-aph(2")* genes in *S. aureus*. A total of 52/109 (47.41%) strains carried at least one of the genes. Fifty-nine *S. aureus* strains were resistant to kanamycin, including 52 that carried resistance genes, and 12 strains were positive for *ant(4')-Ia* and *aac(6')-aph(2")* by PCR. Fifty-seven (52.29%) of the *S. aureus* strains were resistant to gentamycin as determined by disc diffusion, and 32 of these were positive for *aac(6')-aph(2")* (Tab. 3, Fig. 1(A)).

Correlations between erythromycin resistance and the presence of *ermA* and *ermC* are summarized in Tab. 3. A total of 41 (37.61%) *S. aureus* isolates were resistant to erythromycin determined by disc diffusion, including 34 that were positive for *ermA* and two that carried *ermC* (Tab. 3). Tetracycline-resistance correlated with the presence of *tetM* (Tab. 3). Twenty-nine (26.61%) *S. aureus* strains were resistant to tetracycline in the susceptibility tests, but 41 were positive for *tetM* by PCR (Tab. 3, Fig. 1(B)).

We used multiplex PCR to determine the SCC*mec* types in 60 *mecA*-positive strains (Fig. 1(C)). The predominant type was SCC*mec* type II 28/60 (46.67%). The prevalence of type IVA, type IVb, type IVd, and non-typable were 30.00% (18/60), 8.33% (5/60), 6.67% (4/60), and 8.33% (5/60), respectively, by multiplex PCR.

The correlation between carbapenem-resistance and the presence of SCC*mec* types is shown in Tab. 4. A total of 28/60 (46.67%) SCC*mec* type II strains were resistant to piperacillin 21/28 (75.00%), cefotaxime 22/28 (78.57%), and imipenem 22/28 (78.57%), and 18/60 (30.0%) SCC*mec* type IVA strains were resistant to piperacillin 11/18 (61.11%), cefotaxime 9/18 (50.00%), and imipenem 5/18 (27.78%). Fourteen (14/60, 23.33%) IVb, IVd, and non-typable strains were resistant to ticarcillin 5/14 (35.71%), cefepime 5/14 (35.71%), and meropenem 3/14 (21.42%) (Tab. 4). SCC*mec* type II had higher carbapenem-resistance than the IVA, IVb, IVd, and not-typable strains.

Discussion

The present study compared the results of antimicrobial susceptibility determined for *S. aureus* strains (Tab. 3). Although the results of the present study showed an almost perfect correlation between phenotypic methicillin susceptibility and *mecA*, two strains presented discrepancies between the genotype and phenotype, as did two methicillin-resistant *mecA*-negative strains. Previous researchers have reported that *S. aureus* isolates carrying *mecA* were sensitive to oxacillin, and thus, *mecA* might be heterogeneously expressed, and some *S. aureus* strains carrying *mecA* might

TABLE 4

Extended-spectrum, carbapenem, and *mecA*-positive patterns of MRSA strains

Antibiotics	*mecA*-positive MRSA (n=60)					
	type II (n = 28, 46.67%)		type IVA (n = 18, 30.00%)		IVb, IVd, Non-typable (n = 14, 23.33%)	
	Susceptible number (n, %)	Resistance number (n, %)	Susceptible number (n, %)	Resistance number (n, %)	Susceptible number (n, %)	Resistance number (n, %)
Amoxicillin	22 (78.57%)	6 (21.43%)	15 (83.33%)	3 (16.67%)	8 (57.14%)	6 (42.86%)
Ticarcillin	9 (32.14%)	19 (67.86%)	14 (77.78%)	4 (22.22%)	9 (64.29%)	5 (35.71%)
Piperacillin	7 (25.00%)	21 (75.00%)	7 (38.89%)	11 (61.11%)	6 (42.86%)	8 (57.14%)
Cefepime	6 (21.43%)	22 (78.57%)	8 (44.44%)	10 (55.56%)	9 (64.29%)	5 (35.71%)
Cefotaxime	6 (21.43%)	22 (78.57%)	9 (50.00%)	9 (50.00%)	8 (57.14%)	6 (42.86%)
Ceftazidime	6 (21.43%)	22 (78.57%)	7 (38.89%)	11 (61.11%)	7 (50.00%)	7 (50.00%)
Imipenem	6 (21.43%)	22 (78.57%)	13 (72.22%)	5 (27.78%)	11 (78.57%)	3 (25.43%)
Ertapenem	7 (25.00%)	21 (75.00%)	13 (72.22%)	5 (27.78%)	10 (71.43%)	4 (27.57%)
Meropenem	6 (21.43%)	22 (78.57%)	13 (72.22%)	5 (27.78%)	11 (78.57%)	3 (25.43%)

not be detectable using phenotypical methods (Kolbert *et al*., 1998; Martineau *et al*., 2000). The possibility of selecting resistant cells from originally-susceptible strains has been demonstrated. Some strains do not express *mecA* unless they are provided with selective pressure via increasing gradients of the antibiotic agent (Baym *et al*., 2016). The second case of discrepancy occurred in two *mecA*-negative *S. aureus* strains that were phenotypically-resistant to methicillin, but the *mec*A gene was not detected in these isolates. Further investigations into these two isolates are on-going (our result type *mec*C). Penicillin-resistance in *S. aureus* has been reported to be mediated by the expression of penicillinase encoded by *blaZ*, which hydrolyzes the β-lactam ring and contributes to the inactivation of penicillin (Bradford *et al*., 2001; Olsen *et al*., 2006; Xu *et al*., 2014; Ferreira *et al*., 2017).

However, the results of other investigations between penicillin-resistance and bla_{TEM} were unclear. Our PCR results showed that 81.65% (89/109) of the *S. aureus* strains carried bla_{TEM}. bla_{TEM} is known to encode a series of class A plasmid-mediated enzymes belonging to the extended-spectrum β-lactamases that are associated with penicillin resistance and are frequently present in *Klebsiella pneumoniae* and *Escherichia coli* (Chong *et al*., 2011; Dahms *et al*., 2015). Recent clinical trial results demonstrated the efficacy of beta-lactams and carbapenems against *S. aureus* (Lee *et al*., 2018; Saeki *et al*., 2018).

The *S. aureus* strains in this study were analyzed for the presence of AME genes *ant(4')-Ia* and *aac(6')-aph(2")* by multiplex PCR. In accordance with previous studies, *aac(6')-aph(2")* was the most prevalent AME gene encoded in *S. aureus* (Martineau *et al*., 2000). The *aac(6')-aph(2")* gene encodes a bifunctional enzyme AAC(6')-APH(2") that catalyzes both acetyltransferase and phosphotransferase reactions, thereby inactivating an extensive range of clinically-useful aminoglycosides (Ramirez *et al*., 2010). In this study, strains that harbored *ant(4')-Ia* were resistant to kanamycin, and all strains that carried *aac(6')-aph(2")* were clearly resistant to gentamicin and kanamycin in susceptibility testing. Some *S. aureus* strains were phenotypically-resistant to kanamycin, including three that showed kanamycin resistance in susceptibility testing but did not carry *ant(4')-Ia* or *aac(6')-aph(2")* genes.

The *S. aureus* strains were also analyzed for the prevalence of erythromycin-resistance genes *ermA and ermC*. The *S. aureus* strains carried at least one of these *erm* genes, and the majority carried *ermA*. Our results were consistent with findings from previous studies reporting that the incidence of *ermA* was higher than that of *ermC* in clinical *S. aureus* strains (Martineau *et al*., 2000). However, a higher prevalence of *S. aureus* carrying *ermC* in Denmark has been reported (Westh *et al*., 1995). The five samples with discrepant results between resistance genotypes and phenotypes indicated that some strains might have harbored other erythromycin-resistance genes. The prevalence of phenotypic tetracycline-resistance in 12 *S. aureus* samples was different than the *tetM* results. These discrepancies also suggest that some strains might harbor tetracycline-resistance genes and the diameters of inhibition zones ≦ 13 mm were incorrectly measured.

In the present study, we evaluated the prevalence of different types of SCC*mec* by multiplex PCR. Commonly, HA-MRSA strains carry SCC*mec* types I-III with multidrug resistance, while CA-MRSA strains harbor types IV and V. Previous research in South Korea reported that SCC*mec* type II was the most prevalent among the HA-MRSA strains, while SCC*mec* type IVA was predominant in the CA-MRSA strains, however, another study reported a higher prevalence of type IV (Kim *et al*., 2007; Park *et al*., 2007). Excessive therapeutic use of antimicrobial agents in hospital environments might have contributed to the development of resistance and the widespread distribution of SCC*mec* type II MRSA strains.

Conclusions

This study showed that SCC*mec* type II was more predominant than type IV. A total of 22/28 SCC*mec* type II strains were resistant to imipenem, and five SCC*mec* type IVA strains were resistant to imipenem. The other SCC*mec* type IVb, type IVd, and non-typable strains showed lower resistant to imipenem. The SCC*mec* type II strains had higher carbapenem-resistance than type IVA strains.

Conflict of Interest

The authors declare that they have no conflicts of interest.

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