Molecular epidemiology and antimicrobial susceptibilities of 273 exfoliative toxin-encoding-gene-positive Staphylococcus aureus isolates from patients with impetigo in Japan

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The molecular epidemiology and antimicrobial susceptibilities of 273 Staphylococcus aureus isolates positive for the exfoliative toxin-encoding gene obtained from patients with impetigo in Japan in 2006 were studied. The mecA gene was detected in 74 meticillin-resistant S. aureus (MRSA) and 23 meticillin-susceptible S. aureus (MSSA) isolates. All isolates with the staphylococcal cassette chromosome (SCC) mec were classified into type IV (92.8 %, 90/97) or V (7.2 %, 7/97). The ET-encoding gene etb was found primarily in strains with mecA (87.7 %, 71/81), whilst eta (86.6 %, 161/186) was detected mainly in strains without mecA. The chromosomal enterotoxin-encoding gene cluster egc was found in 83.0 % of strains with eta, whilst no enterotoxin-encoding gene was detected in strains with only etb. PFGE showed that each strain carrying eta, etb and etd could be classified into distinct groups. The susceptibility profiles of MRSA to antimicrobial agents excluding β-lactams were similar to those of MSSA. Gentamicin- and clarithromycin-resistant strains were frequently found for both MRSA and MSSA. The aminoglycoside-resistance gene aacA–aphD was detected in 97.3 % of MRSA and 85.4 % of MSSA. Additionally, the macrolide-resistance gene ermA or ermC was detected in 67.6 % of MRSA and 71.4 % of MSSA. Therefore, these results suggest that SCCmec types IV or V have spread, particularly in MSSA carrying etb in the community.

INTRODUCTION

Impetigo is one of the major bacterial skin infections in children and neonates, and is caused by exfoliative toxins (ETs) produced by Staphylococcus aureus (Plano, 2004). There are four serological forms of ET: ETA, ETB, ETC and ETD (Sato et al., 1994; Yamaguchi et al., 2002a). Three serotypes – ETA, ETB and ETD – are linked to human impetigo, whereas ETC was characterized in S. aureus isolated from a horse (Sato et al., 1994). The eta and etb genes are generally found in S. aureus isolated from patients with impetigo, whereas etd is detected primarily in strains isolated from patients with finger pulp infections, furuncles and cutaneous abscesses (Yamasaki et al., 2006).

The exotoxins of S. aureus from human infections include ETs and toxic shock syndrome toxin-1 (TSST-1), staphylococcal enterotoxins (SEs) and Panton–Valentine leukocidin (PVL) (Becker et al., 2003; Dinges et al., 2000; Lovseth et al., 2004). Five of the SE-encoding genes (seg, sei, sem, sen and seo) belong to the same enterotoxin-encoding gene cluster (egc), and detection of both seg and sei usually indicates the presence of all five enterotoxin-encoding genes (Lovseth et al., 2004). The pvl gene encoding PVL is frequently found in community-acquired (CA) meticillin-resistant S. aureus (MRSA) isolated from patients with skin and soft tissue infections (Vandenesch et al., 2003).

Abbreviations: CA, community-acquired; ET, exfoliative toxin; MRSA, meticillin-resistant Staphylococcus aureus; MSSA, meticillin-susceptible Staphylococcus aureus; SCC, staphylococcal cassette chromosome; SE, staphylococcal enterotoxin.
Recently, the number of patients with impetigo caused by MRSA has been increasing in the community setting (Hisata et al., 2005; Noguchi et al., 2006; Yamaguchi et al., 2002b). MRSA produces penicillin-binding protein 2' (PBP2') with a low affinity for β-lactam antibiotics (Ito & Hiramatsu, 1998). PBP2' is encoded by the mecA gene located on a large mobile genetic element called the staphylococcal cassette chromosome (SCC) (Katayama et al., 2000). Currently, five main types of SCCmec have been distinguished according to their structures: types I, II and III are mainly found in health-care-associated MRSA, whilst types IV and V are found mainly in CA-MRSA (Fey et al., 2003; Ito et al., 2004).

In contrast to health-care-associated MRSA carrying SCCmec types II or III, CA-MRSA carrying type IV or V are generally susceptible to antimicrobial agents other than β-lactams (Almer et al., 2002; Naimi et al., 2003; Okuma et al., 2002). However, we reported previously that all MRSA isolated from patients with impetigo belonged to SCCmec type IV, and 95 and 80% of those MRSA were resistant to gentamicin and clarithromycin, respectively (Noguchi et al., 2006). Furthermore, we found that MRSA isolated from patients with impetigo was susceptible to antiseptic agents, in contrast to MRSA carrying SCCmec type II, because the distribution of the major antiseptic-resistance gene, qacA/B (Nakaminami et al., 2008), was significantly lower in MRSA carrying SCCmec type IV (1.3%) than in MRSA carrying SCCmec type II (45.9%) (Noguchi et al., 2006).

As patients with impetigo are generally children seen in an outpatient setting, surveillance of MRSA isolated from patients with impetigo will provide useful information that will help in the understanding of the distribution of MRSA in the community. Molecular epidemiological analysis of S. aureus isolated from patients with impetigo has been carried out in Japan (Yamaguchi et al., 2002b). However, information regarding the distribution of the types of SCCmec, exotoxins and antimicrobial-resistance genes of S. aureus causing this skin disease is still lacking. In the present study, we investigated the current characteristics of S. aureus isolated from patients with impetigo from a local area in Japan. To this end, molecular epidemiological analysis of S. aureus isolated from such patients was performed by SCCmec typing, exotoxin-encoding gene profiling and PFGE. In addition, we determined the susceptibilities of these isolates to antimicrobial agents and the distribution of antimicrobial-resistance genes.

METHODS

Isolates. In 2006, a total of 273 S. aureus isolates was collected from 341 patients with impetigo from Sakurazuka Yoshida Clinic (72 isolates), Senoue Dermatology Clinic (69 isolates), Asano Dermatology Clinic (44 isolates), Shido Dermatology Clinic (26 isolates), Takamatsu Red Cross Hospital (25 isolates), Marunouchi Dermatology Clinic (21 isolates), Sato Dermatology, Urology and Venereology Clinic (15 isolates) and Mitoyo General Hospital (1 isolate). These facilities are located in Kagawa Prefecture, Shikoku Island, Japan. All strains were isolated from different outpatients (mean age 4.3 years). Strain TY114 (Yamaguchi et al., 2002a) was used as a control for the detection of etd. The following strains were used as SCCmec type strains: NCTC 10442 (type I), N315 (type II), 85/2082 (type III), JCSC 4744 (type IV) and W1500 (type V) (Ito et al., 2004). Meticillin-susceptible S. aureus (MSSA) ICMP 2874 (ATCC 29213) was used as a quality control strain for antimicrobial susceptibility testing (CLSI, 2007a).

Bacterial identification. All clinical isolates were identified as S. aureus by a positive Gram stain, the utilization of mannitol salt agar (Oxoid) and a test for coagulase production (PS Latex; Eiken Chemical). The isolates were classified as MRSA on the basis of oxacillin resistance and the detection of mecA (Noguchi et al., 2006). Strains that were mecA-negative but resistant to oxacillin (borderline oxacillin-resistant S. aureus) were classified as MRSA (CLSI, 2007a).

PCR amplification. PCR was performed as described previously in order to detect various genes (Noguchi et al., 2006). PCR to detect the SE-encoding genes (tst, sea, seb, sec, sed, see, seg, seh, sei and sej) and pvl was performed according to the methods of Lovseth et al. (2004) and Takizawa et al. (2005), respectively. The primer pairs designed in this study are shown in Table 1. PCR for etd and antimicrobial-resistance genes was performed using the same cycling conditions as for eta and mecA, respectively (Noguchi et al., 2006). All PCR products were analysed by electrophoresis in 2% agarose. All results were confirmed by at least two independent experiments.

SCCmec typing and PFGE analysis. SCCmec typing was performed according to the method of Oliveira & de Lencastre (2002). PFGE analysis was performed as described previously (Noguchi et al., 2006). PFGE groups containing more than ten strains were defined by ≥80% genetic relatedness on the dendrograms.

Antimicrobial susceptibility. MICs were determined using an agar doubling-dilution method according to CLSI guidelines (CLSI, 2007b). Cefdinir, faropenem, nadifloxacin, arbekacin and mupirocin were kindly provided by their manufacturers. Ampicillin, cefalexin, vancomycin, levofloxacin, clarithromycin, josamycin, clindamycin, gentamicin, minocycline and chloramphenicol were purchased from Wako Pure Chemical Industries, and oxacillin and fusidic acid from Sigma-Aldrich. The breakpoints for these antimicrobial agents were determined using the interpretation criteria of CLSI (2007a) and undefined breakpoints were defined in this study. Susceptibility to oxacillin was also confirmed for all strains via MIC determination.

Statistical analysis. Differences in the possession rate for various genes were tested using a χ² test using JMP software (SAS Institute). P values of <0.05 were considered to be statistically significant.

RESULTS

Identification of MRSA and SCCmec types and the distribution of exotoxin-encoding genes

In 341 patients with impetigo, 308 isolates were identified as S. aureus. ET-encoding genes were detected in 273 (88.6%) of these isolates. In this study, all of these 273 isolates were used. Of the 273 et-positive isolates, 27.8% (76/273) were identified as MRSA. Among the MRSA, two strains that lacked mecA but showed low-level resistance to oxacillin (i.e. borderline oxacillin-resistant S. aureus) were found. Among the MSSA, 11.7% (23/197) of the strains
carried mecA. Therefore, 35.5% (97/273) of the isolates tested in this study carried mecA. When SCCmec typing was performed in these 97 strains with mecA, 90 (92.8%) and 7 (7.2%) strains were classified as SCCmec types IV and V, respectively. The most frequently isolated from impetigo patients in this study was S. aureus carrying eta (68.9%, 188/273) (Table 2). The possession rate of eta in strains without mecA (87.1%, 162/186) was significantly higher than in strains with mecA (P<0.0001) (Table 2). In contrast, the possession rate of etb in strains with mecA (88.9%, 72/81) was significantly higher than in strains without mecA (P<0.0001) (Table 2). The etd gene was detected in five MSSA isolates and one of these isolates also carried eta.

Detection of SE-encoding genes and pvl was used to examine the exotoxin-encoding gene profiles in all strains (Table 2). No strain carrying sea, sed, sec, seh, sej or pvl was found in this study. Notably, seg and sei located on egc were frequently detected in strains carrying eta (156/188, 83.0%). In contrast, no exotoxin-encoding gene was found in strains with only etb. Only one MRSA carrying tss, seb and sec in addition to eta was found, although no egc was detected in this strain. All strains with etd carried egc, and two of these strains also carried seb.

**PFGE analysis**

The genetic relatedness of S. aureus strains was studied by PFGE (Fig. 1). Although the samples were collected from a limited area in Japan, comparison of the PFGE patterns showed that the strains had at least 90 different PFGE types (3 strains were not analysable). PFGE patterns were classified into six groups (A–F) by 80% band similarity including more than ten strains. The most frequent PFGE group, E, included

### Table 1. Primers used in this study

<table>
<thead>
<tr>
<th>Gene/primer</th>
<th>Primer sequence (5’–3’)</th>
<th>Amplicon size (bp)</th>
<th>GenBank accession no.</th>
</tr>
</thead>
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<tr>
<td>etd</td>
<td>CGCAATACATATGAAGAATCTGA</td>
<td>452</td>
<td>AB057421</td>
</tr>
<tr>
<td></td>
<td>TGTCACTTGGCAAATCTATAG</td>
<td></td>
<td></td>
</tr>
<tr>
<td>aacA–aphD</td>
<td>TACAGGCCTTGGGAAGATG</td>
<td>406</td>
<td>AF051917</td>
</tr>
<tr>
<td></td>
<td>CATTGTGCCATTATCATATTC</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ermA</td>
<td>AGCGGTAAACCCCTCTGAG</td>
<td>457</td>
<td>X03216</td>
</tr>
<tr>
<td></td>
<td>TAGTGACATTTGCATGCTTCAA</td>
<td></td>
<td>L08862</td>
</tr>
<tr>
<td>ermC</td>
<td>ACTTGTGTGATCGATAATTTCCA</td>
<td>321</td>
<td></td>
</tr>
<tr>
<td></td>
<td>TCTACTTAATCTGATAAGTAGCTATTCAC</td>
<td></td>
<td></td>
</tr>
<tr>
<td>msrA/B</td>
<td>TGCAATGGCATACTATCGTC</td>
<td>160</td>
<td>X52085</td>
</tr>
<tr>
<td></td>
<td>CAAGAAGCTCAAGTGCTTTC</td>
<td></td>
<td></td>
</tr>
<tr>
<td>mphC</td>
<td>AACACATTAGCTGAACACTCG</td>
<td>299</td>
<td>AB013298</td>
</tr>
<tr>
<td></td>
<td>GGGTTGCTTCAGTCCAGTCT</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Table 2. Distribution of exotoxin-encoding genes in mecA-positive and mecA-negative MRSA and MSSA used in this study

<table>
<thead>
<tr>
<th>Gene*</th>
<th>MRSA (n=76)</th>
<th>MSSA (n=197)</th>
<th>Total (n=273)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mecA⁺</td>
<td>mecA⁻</td>
<td>mecA⁺</td>
</tr>
<tr>
<td>eta</td>
<td>4 (5.3)</td>
<td>1 (1.3)</td>
<td>0</td>
</tr>
<tr>
<td>eta + tsst + seb + sec</td>
<td>1 (1.3)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>eta + egc</td>
<td>17 (22.4)</td>
<td>0</td>
<td>3 (1.5)</td>
</tr>
<tr>
<td>etb</td>
<td>51 (67.1)</td>
<td>1 (1.3)</td>
<td>20 (10.2)</td>
</tr>
<tr>
<td>etd + seb + egc</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>etd + egc</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>eta + etb + egc</td>
<td>1 (1.3)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>eta + etd + egc</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

*The enterotoxin-encoding gene cluster egc encodes at least two se genes, seg and sei.
Fig. 1. Molecular epidemiological analysis of *S. aureus* isolates from patients with impetigo using PFGE patterns, SCCmec types, and the presence of ET-encoding genes and exotoxin-encoding genes. *A, Sakurazuka Yoshida Clinic; B, Senoue Dermatology Clinic; C, Asano Dermatology Clinic; D, Shido Dermatology Clinic; E, Takamatsu Red Cross Hospital; F, Marunouchi Dermatology Clinic; G, Sato Dermatology, Urology and Venereology Clinic; H, Mitoyo General Hospital. †PFGE groups containing more than ten strains were defined by $\geq 80\%$ genetic relatedness on the dendrograms. The enterotoxin-encoding gene cluster *egc* encodes at least two *se* genes, *seg* and *sei*. 
ampicillin, *Percentage of resistant isolates. Resistance breakpoints of the following antimicrobial agents were defined according to CLSI and this study: ampicillin, \( \geq 0.5 \text{ mg} \text{ml}^{-1} \); oxacillin, \( \geq 4 \text{ mg} \text{ml}^{-1} \); cefalexin, \( \geq 32 \text{ mg} \text{ml}^{-1} \); cefdinir, \( \geq 4 \text{ mg} \text{ml}^{-1} \); faropenem, \( \geq 8 \text{ mg} \text{ml}^{-1} \); vancomycin, \( \geq 32 \text{ mg} \text{ml}^{-1} \); levofloxacin, \( \geq 4 \text{ mg} \text{ml}^{-1} \); minocycline, \( \geq 16 \text{ mg} \text{ml}^{-1} \); arbekacin, \( \geq 8 \text{ mg} \text{ml}^{-1} \); clindamycin, \( \geq 4 \text{ mg} \text{ml}^{-1} \); gentamicin, \( \geq 16 \text{ mg} \text{ml}^{-1} \); mupirocin, \( \geq 2 \text{ mg} \text{ml}^{-1} \).\n
126 strains with \( \text{eta} \), and all except 1 of them carried \( \text{egc} \). Strains with \( \text{etb} \) were classified into PFGE groups A, B and C. These data showed that each group of strains with \( \text{eta} \), \( \text{etb} \) and \( \text{etd} \) had obviously different PFGE types, although strains with the same \( \text{et} \) gene had similar PFGE patterns.

### Antimicrobial susceptibility

The antimicrobial susceptibilities of the MRSA isolates were compared with the MSSA isolates (Table 3). The resistance rates of all \( \beta \)-lactams for MRSA isolates were higher than those of MSSA. However, 80.7% of the MSSA isolates were resistant to ampicillin. All strains were susceptible to vancomycin, fluoroquinolones, minocycline and arbekacin. Resistance to clindamycin, a 14-membered-ring macrolide, was found in 68.4% of the MRSA and 72.6% of the MSSA isolates. The resistance rates of josamycin of the 16-membered-ring macrolides and clindamycin of the lincosamides were higher for the MRSA than for the MSSA isolates. Gentamicin-resistant strains were frequently found in both the MRSA and MSSA isolates. Almost all strains were susceptible to the common topical agents chloramphenicol, fusidic acid and mupirocin.

### Distribution of antimicrobial-resistance genes

The distributions of the aminoglycoside-resistance gene \( \text{aacA}–\text{aphD} \) and the macrolide-resistance genes \( \text{ermA} \), \( \text{ermC} \), \( \text{msrA/B} \) and \( \text{mphC} \) were examined (Table 4). The \( \text{aacA}–\text{aphD} \) gene was frequently detected in both the MRSA (73/76, 96.1%) and MSSA (169/197, 85.8%) isolates. The detection rate of \( \text{ermA} \) was significantly higher in the MRSA (33/76, 43.4%) than in the MSSA (36/197, 18.3%) isolates \( (P<0.0001) \). In contrast, the detection rate of \( \text{ermC} \) was significantly higher in the MSSA (104/197, 52.8%) than in the MRSA (19/76, 25.0%) isolates \( (P<0.0001) \). No strain carrying both \( \text{ermA} \) and \( \text{ermC} \) was found. The antiseptic-resistance gene \( \text{qacA/B} \) was detected in five (6.6%) MRSA isolates but not in any of the MSSA isolates. Three of the strains with \( \text{qacA/B} \) carried both \( \text{aacA}–\text{aphD} \) and \( \text{ermA} \). The strain with \( \text{aacA}–\text{aphD} \), \( \text{ermA} \), \( \text{msrA/B} \) and \( \text{mphC} \) also carried \( \text{qacA/B} \).

### DISCUSSION

Herein, we showed that 28% of \( S. \text{ aureus} \) isolated from patients with impetigo in a local area of Japan were MRSA. In addition, 12% of the MSSA isolates carried \( \text{mecA} \). Thus, overall, 36% of all of the \( S. \text{ aureus} \) isolates carried \( \text{mecA} \). The \( \text{mecA} \) of these strains was classified into CA-SCCmec type IV or V. These data showed that \( S. \text{ aureus} \) carrying \( \text{mecA} \) is widely distributed in this community.

In this study, 23 of the MSSA carried \( \text{mecA} \) (MIC of oxacillin was 1 or 2 mg ml\(^{-1} \)). Oxacillin-resistant \( S. \text{ aureus} \) with \( \text{mecA} \) has been reported in the past (Bressler
et al., 2005; Hososaka et al., 2007; Petinaki et al., 2002). The oxacillin-susceptible strains with mecA were thought to be caused by a single amino acid substitution in PBP2’ (Bressler et al., 2005). Additionally, the mecRI gene, which encodes a transmembrane β-lactam-sensing signal transducer, of SCCmec types IV and V is defective (Deurenberg et al., 2007). For this reason, it is possible that induction by oxacillin is necessary for oxacillin resistance in strains carrying SCCmec type IV or V. Therefore, it is necessary to perform SCCmec subtyping and a cefoxitin disc test as recommended by CLSI (2007a) on these strains.

The ET-encoding gene etb was found primarily in strains with mecA, whilst eta was mainly found in strains without mecA. We reported previously that the possession rate of etb in MRSA isolated from patients with impetigo was higher than that of eta (Noguchi et al., 2006). Therefore, our data in this study were coincident with our previous data. In contrast, etd, which has not been detected previously, was found in five MSSA strains, and one of them also carried eta. The symptoms caused by the strains carrying etd were folliculitis rather than impetigo (data not shown). There was the possibility of overlooking an impetigo-like skin disease caused by S. aureus carrying etd because the clinical symptoms caused by the strain carrying etd may be weaker than those caused by strains with eta or etb.

Detection of the exotoxin-encoding genes revealed interesting data. Whilst no exotoxin-encoding gene was detected in strains with only etb, seg and sei located on egc were frequently detected in strains with eta. These data indicate a relationship between eta and egc. S. aureus carrying egc is frequently found in patients with atopic eczema (Mempel et al., 2003). Although it is unclear why this is so, S. aureus carrying egc might colonize the skin more easily. Further studies are necessary to elucidate why egc is frequently found together with eta rather than with etb.

PFGE analysis showed that each group of strains carrying eta, etb and etd had obviously different PFGE patterns. Additionally, the PFGE patterns of the strains carrying the same serotype of the ET-encoding gene were similar to each other. Our previous data also indicated that the PFGE pattern of each strain with eta and etb was different (Noguchi et al., 2006). Therefore, our data suggest that there is a specific strain that has accessibility for eta, etb and etd. Multilocus sequence typing will be necessary to identify the detailed clone type of each strain carrying eta, etb and etd.

In a comparison between the antimicrobial susceptibilities for MRSA and MSSA, the susceptibility profiles of the MRSA isolates to antimicrobial agents excluding β-lactams were similar to those of the MSSA isolates. Although 91 % of the MRSA and 64 % of the MSSA isolates were resistant to gentamicin, aacA–aphD encoding the aminoglycoside acetyltransferase AAC(6’)-APH(3') and aminoglycoside phospho-transferase APH(2’) (Lyon & Skurray, 1987) was detected in 96 % of the MRSA and 86 % of the MSSA isolates. The ratio of aacA–aphD detection was higher than that of resistance to gentamicin, because aacA–aphD was found in all gentamicin intermediate resistant strains (data not shown). Our data showed that aacA–aphD was widely distributed among both the MRSA and MSSA isolates. Clarithromycin-resistant strains were found at equal rates for the MRSA and MSSA isolates, and the erythromycin ribosyme methylation gene ermA or ermC (Otsuka et al., 2007) was also detected at equal rates in both the MRSA and MSSA isolates. However, the resistance rate of clindamycin, which showed cross-resistance to josamycin, was higher in the MRSA isolates than in the MSSA isolates. The ermA and ermC genes were detected mainly in MRSA and MSSA, respectively. The ermA gene gives constitutive resistance to all macrolides and clindamycin (Otsuka et al., 2007). In contrast, ermC gives inducible resistance and is not induced by 16-membered-ring macrolides and clindamycin (Otsuka et al., 2007). Thus, the high rate of

### Table 4. Distribution of antimicrobial-resistance genes in MRSA and MSSA used in this study

<table>
<thead>
<tr>
<th>Gene</th>
<th>MRSA (n=76)</th>
<th>MSSA (n=197)</th>
<th>Total (n=273)</th>
</tr>
</thead>
<tbody>
<tr>
<td>aacA–aphD</td>
<td>23 (30.3)</td>
<td>43 (21.8)</td>
<td>66 (24.2)</td>
</tr>
<tr>
<td>aacA–aphD + ermA</td>
<td>29 (38.2)</td>
<td>26 (13.2)</td>
<td>55 (20.1)</td>
</tr>
<tr>
<td>aacA–aphD + ermC</td>
<td>17 (22.4)</td>
<td>97 (49.2)</td>
<td>114 (41.8)</td>
</tr>
<tr>
<td>aacA–aphD + msrA/B</td>
<td>0</td>
<td>3 (1.5)</td>
<td>3 (1.1)</td>
</tr>
<tr>
<td>aacA–aphD + ermA + qacA/B</td>
<td>3 (3.9)</td>
<td>0</td>
<td>3 (1.1)</td>
</tr>
<tr>
<td>aacA–aphD + ermA + msrA/B + mphC + qacA/B</td>
<td>1 (1.3)</td>
<td>0</td>
<td>1 (0.4)</td>
</tr>
<tr>
<td>ermA</td>
<td>0</td>
<td>10 (5.1)</td>
<td>10 (3.7)</td>
</tr>
<tr>
<td>ermC</td>
<td>2 (2.6)</td>
<td>7 (3.6)</td>
<td>9 (3.3)</td>
</tr>
<tr>
<td>qacA/B</td>
<td>1 (1.3)</td>
<td>0</td>
<td>1 (0.4)</td>
</tr>
<tr>
<td>None*</td>
<td>0</td>
<td>11 (5.6)</td>
<td>11 (4.0)</td>
</tr>
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</table>

*None of the resistance genes tested was detected.
resistance to clindamycin of the MRSA was thought to be caused by ernA and its higher possession rate.

The antiseptic-resistance gene qacA/B was detected in 7% of the MRSA strains. This ratio was higher than that found in our previous study (1%) (Noguchi et al., 2006). Although qacA/B is distributed primarily in SCCmec type II MRSA, we have demonstrated previously that this gene can be transferred by transduction to SCCmec type IV MRSA isolated from patients with impetigo (Nakaminami et al., 2007). Therefore, the antiseptic-resistant strain of MRSA isolated from patients with impetigo is likely to prevail.

In summary, 36% of S. aureus strains isolated from patients with impetigo in this study carried CA-SCCmec type IV or V. The ET-encoding gene etb was detected mainly in strains with mecA. The eta gene, which was detected primarily in strains without mecA, may be related to the enterotoxin-encoding gene cluster egc. PFGE analysis showed that each group of strains carrying the same et gene had clearly different PFGE patterns. The susceptibility profiles of MRSA to antimicrobial agents excluding β-lactams were similar to those of the MSSA isolates. Therefore, our results suggest that SCCmec types IV and V have spread, in particular in MSSA carrying etb in the community.

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