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Epidemiological Characteristics of Methicillin-Resistant *Staphylococcus aureus* Isolates from Children with Eczematous Atopic Dermatitis Lesions▽

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In this study, we investigated the rate of colonization of skin of children with atopic dermatitis (AD) by methicillin-resistant *Staphylococcus aureus* (MRSA) and characterized the isolates. Active skin lesions in pediatric AD patients were cultured with Rodac Staph (Komed, Korea). *S. aureus* isolates were examined for drug susceptibilities, analyzed for the *eta*, *etb*, *tst*, and *pvl* genes, and typed using *agr* polymorphism, pulsed-field gel electrophoresis of SmaI-restricted chromosomal DNA, and staphylococcal cassette chromosome *mec* (SCC*mec*) typing. Eighty-seven (75.4%) of 115 patients had cultivable *S. aureus* isolates, 16 of which (18.3%) were MRSA. All MRSA isolates were susceptible to chloramphenicol, rifampin, cotrimoxazole, and ciprofloxacin. While methicillin-susceptible *S. aureus* (MSSA) isolates were composed of 23 isolates of singular types and nine clusters comprising 48 isolates, MRSA isolates were typed into three clones: eight isolates of pulsotype A–*agr-1*–SCC*mec* IV, five isolates of pulsotype B–*agr-3*–SCC*mec* IIb–*etb* positive, and three isolates of pulsotype C–*agr-3*–SCC*mec* IV. Three SCC*mec* IVA MRSA isolates were *tst* positive, but none were positive for the *pvl* or *eta* gene. Among 71 MSSA isolates, 7 isolates were *tst* positive, 6 of which were pulsotype F–*agr-3*, and 9 of 10 *agr-4* isolates were *eta* positive. The average ages of patients carrying MSSA, SCC*mec* IVA MRSA, and SCC*mec* IIb MRSA were 7.7 ± 4.6, 3.1 ± 1.5, and 8.2 ± 3.1 years, respectively. Among the patients carrying MRSA, two patients had been treated with oral antimicrobials, and one had been admitted to the hospital 18 months previously. In conclusion, community-acquired MRSA isolates of a few clones colonized the skin of patients with AD without risk factors for the acquisition of hospital-acquired MRSA, which suggested that the skin of children with AD may represent a significant reservoir of MRSA colonization in the community.

Patients with atopic dermatitis (AD) tend to carry *Staphylococcus aureus* on their skin lesions (1), and superantigens and toxins of *S. aureus* allegedly exacerbate chronic inflammation of AD skin (4, 8, 9). As a result, antimicrobials have often been prescribed to control acute-phase AD (4). Eczematous lesions of AD patients are known to be a source of transmission of *S. aureus* (13, 15). Increasing incidences of community-acquired methicillin-resistant *S. aureus* (MRSA) (CA-MRSA) in skin and soft tissue infection raise concerns that AD skin would be a favorable reservoir for CA-MRSA.

A CA-MRSA outbreak was first described in United States in 1981 in association with intravenous drug users (40), but more recently, these strains have emerged as the pathogens most frequently found in patients with skin and soft tissue infections presenting to emergency departments in the United States (3, 23, 34). The most prevalent CA-MRSA clones in the United States have the USA300 pulsotype harboring staphylococcal cassette chromosome *mec* (SCC*mec*) IV and Panton-Valentine leukocidin (42, 43). The community-based epidemic of MRSA led us to think that that MRSA became as prevalent as penicillin-resistant *S. aureus* strains in the community, as suggested previously by Chambers (6). Although many Asian countries suffer from high rates of MRSA infection, there are few publications on the prevalence of CA-MRSA (7, 17). In South Korea, the overall MRSA rate in clinical isolates during the last decade has been reported to be approximately 70% regardless of the locations or sizes of hospitals (20, 29). Even though the origins of MRSA isolates are not clear, MRSA has been the major pathogen of skin infections and otitis media in South Korean outpatient clinics since the late 1990s (22, 28, 35). The epidemiology of CA-MRSA in South Korea requires urgent attention.

Therefore, in the present study, we evaluated the rate of colonization by MRSA in skin lesions of pediatric AD patients and characterized MRSA isolates obtained from those lesions.

MATERIALS AND METHODS

Patients and bacterial isolates. AD patients were enrolled in our study at the times of their first visits to the pediatric allergy clinic of our hospital from June 2004 to April 2005. Eczematous skin lesions were imprinted with Rodac Staph (Komed, South Korea), and yellow colonies were selected after 48 h of incubation. Bacterial species identification and antimicrobial susceptibility testing were performed using the MicroScan PosCombo 1A system (Dade Behring, West Sacramento, CA). All isolates were stored in brain heart infusion broth containing 15% (vol/vol) glycerol. The first isolate obtained from each patient was investigated further. Patients' medical records were reviewed for basic demographics and clinical diagnoses, prior antimicrobial therapies, hospital admission histories, and places of residence.

Antimicrobial susceptibility. The MicroScan PosCombo 1A (Dade-Behring) panel was used to determine bacterial susceptibility to penicillin, oxacillin, erythromycin, clindamycin, ciprofloxacin, ofloxacin, rifampin, gentamicin, cotrimoxazole, chloramphenicol, tetracycline, fusidic acid, quinupristin-dalfopristin, teicoplanin, and vancomycin. To determine inducible macrolide-lincosamide-

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streptogramin B (MLS_B) resistance, the D-test (36) was performed on all *S. aureus* isolates that were clindamycin susceptible and erythromycin resistant.

DNA extraction. MRSA isolates were subcultured on blood agar plates at 37°C overnight. Three to five isolated colonies were prepared for DNA extraction using the GeneElute bacterial genomic DNA kit (Sigma, St. Louis, MO). Lysostaphin and lysozyme were added for the lysis step at 10 units/ml and 45 mg/ml, respectively.

SCC*mec* typing and *agr* polymorphism. PCR for *agr* polymorphism was performed using primers previously described by Gilot et al. (11). Type assignment of SCC*mec* elements from multiplex PCR was done as described previously by Oliveira and de Lencastre (38). For cases unresolved by these procedures, *ccr* typing and determining the location of IS*1272* were undertaken as previously described (37).

PFGE. Chromosomal DNA was digested with SmaI and electrophoresed using program 2 of the GenePath system (Bio-Rad Laboratories Inc., Hercules, CA) as previously described (21). The isolates showing six or fewer band differences by pulsed-field gel electrophoresis (PFGE) were counted to the same group of pulsotype. Cluster analysis of pulsotypes was done in the dendrogram type of the unweighted-pair group method using average linkages with the Dice coefficient using InfoQuest FP software, version 4.5 (Bio-Rad).

PCR for the *eta*, *etb*, *tst*, and *pvl* genes. To detect the *eta*, *etb*, and *tst* genes, a multiplex PCR assay combining primers specific for *eta*, *etb*, and *tst* was performed (2). The *pvl* gene was detected with PCR using primers luk-PV-1 and luk-PV-2 (31).

RESULTS

Patients and bacterial isolates. A total of 122 specimens were collected from 115 patients during the study. *S. aureus* was isolated from 92 (75.4%) specimens from 87 (75.7%) patients. Eighteen isolates from 16 (18.3%) patients were resistant to oxacillin by MicroScan. Forty-six (64.8%) of the 71 patients carrying methicillin-susceptible *S. aureus* (MSSA) were male, and their average age was 7.7 ± 4.6 years, The male-to-female ratio of 16 patients carrying MRSA was 7:9. While the average age of five patients carrying SCC*mec* IIb MRSA was 8.2 ± 3.1 years, that of 11 patients carrying SCC*mec* IVA MRSA was 3.1 ± 1.5 years. Two patients had been prescribed amoxicillin-clavulanate; one of them had also received mupirocin ointment, and the other patient had been admitted for pneumonia, which was treated with azithromycin 18 months prior to our study. All but two of our patients lived in metropolitan Seoul and its suburban area.

Antimicrobial susceptibility. All MRSA isolates were susceptible to ciprofloxacin, ofloxacin, rifampin, cotrimoxazole, chloramphenicol, quinupristin-dalfopristin, teicoplanin, and vancomycin. The susceptibilities to erythromycin, clindamycin, gentamicin, tetracycline, and fusidic acid were 17.6%, 58.8%, 41.2%, 94.1%, and 88.2%, respectively. All MSSA isolates were susceptible to rifampin, cotrimoxazole, quinupristin-dalfopristin, teicoplanin, and vancomycin. Their susceptibilities against chloramphenicol, ofloxacin, and ciprofloxacin were 95.7%, 94.1%, and 94.1%, respectively. They were more susceptible to erythromycin (59.4%), clindamycin (95.9%), and gentamicin (99.7%) but were less susceptible to fusidic acid (55.1%) than MRSA. All 28 isolates, including 7 MRSA isolates that were resistant to erythromycin and susceptible to clindamycin, were D-test positive, except for a single MSSA isolate.

SCC*mec* typing, *agr* polymorphism, and PFGE. The 16 MRSA isolates were all *mecA* positive, *dcs* positive, and *kdp* negative. Of those isolates, five isolates were *mecI* positive and were positive for pUB110, except one. The other 11 isolates were *mecI* negative, IS*1272* positive, and pUB110 positive. All MRSA isolates were positive for *ccrA2*, indicating the possible combinations of *ccr* type and *mec* type of 2A and 2B. Therefore, the former five isolates were SCC*mec* type II, *kdp*-negative variant IIb (14), and the latter 11 isolates were SCC*mec* type IV, pUB110-positive variant IVA (25, 35). Of 11 SCC*mec* IVA isolates, 8 had pulsotype A–*agr-1*, while 3 had pulsotype C–*agr-3*. Five SCC*mec* IIb isolates were all of pulsotype B–*agr-3* (Fig. 1).

Among the 71 MSSA isolates, 35 were of the *agr-1* type, 24 were of the *agr-3* type, 10 were of the *agr-4* type, and only 2 were of the *agr-2* type (Fig. 1). In PFGE analyses, 48 MSSA isolates were distributed into nine clusters: pulsotype D for 17 isolates with *agr-3*, pulsotype E for 8 isolates with *agr-4*, pulsotype F for 6 isolates with *agr-3*, pulsotype G for 4 isolates with *agr-1*, and 5 other pulsotypes composed of two to three isolates per each group; however, the other 23 MSSA isolates were the solitary type (Fig. 1).

Toxin gene profiles. All *S. aureus* isolates were negative for the *pvl* gene. Among the 16 MRSA isolates, 2 were *tst* positive and 5 were *etb* positive. Two of the *tst*-positive isolates were pulsotype A–*agr-1*–SCC*mec* IVA, while the five *etb*-positive isolates were all of pulsotype C–*agr-3*–SCC*mec* IIb (Fig. 1). Among the 71 MSSA isolates, *tst* was positive in six pulsotype F–*agr-3* isolates and two pulsotype A–*agr-1* isolates. *eta* was positive in all eight pulsotype E–*agr-4* isolates and one pulsotype L–*agr-4* isolate, which was the only *etb*-positive isolate (Fig. 1).

DISCUSSION

Consistent with previous studies (12, 16), *S. aureus* colonization was found in 75.7% of pediatric AD lesions, with MRSA accounting for 18.4% of *S. aureus* isolates in skin lesions of pediatric AD patients. This is the first report on the carriage rates of MRSA in skin lesions of pediatric AD patients. The carriage rate found by us is much higher than the recently reported rates of colonization by MRSA in healthy Asian schoolchildren. These rates were 5.1% in South Korea (30), 4.3% in Japan (14), and 1.9% in Taiwan (19). Considering a predilection of *S. aureus* for damaged skin and the frequent exposure of AD patients to antimicrobials, the high rate of colonization by MRSA noted in our study may not be surprising. Recently, there was a case report of a child with severe AD who presented with CA-MRSA skin abscesses (41). A high rate of colonization by MRSA can be worrisome for AD patients because it predisposes them to having invasive cutaneous infections. In addition, the average age of patients from whom SCC*mec* IV isolates were obtained was significantly younger than that of patients from whom SCC*mec* II isolates were cultured. These findings suggest that two discrete CA-MRSA clones were spread in different time periods. The high colonization rate and clonality of MRSA seen in this study indicate that AD patients can be a potential source of CA-MRSA transmission.

All the MRSA isolates were community acquired, and only two patients had risk factors for hospital-acquired MRSA (HA-MRSA), such as previous hospitalization and prior antibiotic therapy (24). SCC*mec* IVA was predominant among the MRSA isolates in our study. In addition, all such isolates were susceptible to cotrimoxazole and ciprofloxacin, which is un-

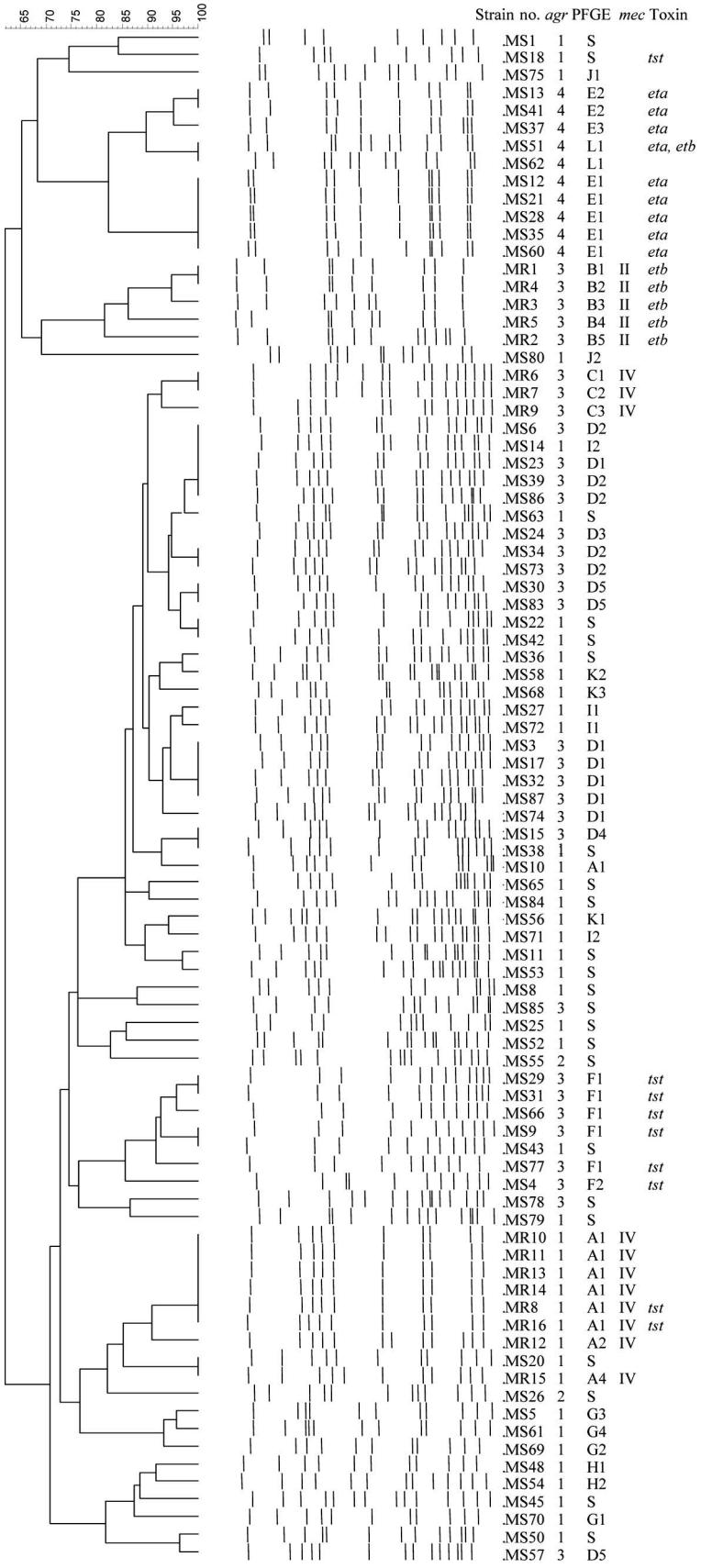

FIG. 1. Cluster analysis of pulsotype, *agr* polymorphism, SCC*mec* typing, and toxin gene profiles of 87 *S. aureus* isolates according to *agr* polymorphism.

usual among MRSA strains isolated in South Korean hospitals (20, 29). Although the outbreak of staphylococcal scalded skin syndrome by MRSA that occurred in the Kyungnam province involved patients with no risk factors for HA-MRSA, and all isolates were clonal by PFGE, the MRSA isolates showed characteristics of typical HA-MRSA isolates, such as multidrug resistance and SCC*mec* type II (32). Therefore, the MRSA isolate was assumed to be a hospital-derived clone introduced into the community. However, an SCC*mec* IV clone has been found in community settings such as in neonates born at primary obstetrics clinics (26), in a surveillance of healthy schoolchildren (30), and in cases of bovine mastitis (27). As was the case in this study, such SCC*mec* IV clones were pUB110 positive and of type IVA and did not show multidrug resistance (5, 26). Even though there has been a lack of data on the prevalence of CA-MRSA infections, those reports suggest the emergence of CA-MRSA in South Korea.

MRSA isolates showed two *agr* types, *agr-1* and *agr-3*, and MSSA isolates also were mainly of types *agr-1* and *agr-3*. The prevalent CA-MRSA strain circulating in France, Switzerland, and Australia has *agr-3* and the USA300 clone, which is an epidemic clone in United States, and in Europe, it has *agr-1* (43). There has not been a reported case of *agr-2* CA-MRSA. Because *agr-2*, which seems to have benefits in surviving in the hospital setting (33), is the type frequently found in cases of HA-MRSA in South Korea (46), the absence of *agr-2* in MRSA isolates reported in this study was consistent with the community origin of the isolates reported here. Compared to the MSSA isolates composed of heterogeneous pulsotypes, all the MRSA isolates were clustered into a few clones by PFGE analysis. The MRSA isolates of each clusters also shared common types in SCC*mec*, *agr* polymorphism, and toxin profile: pulsotype A–*agr-1*–SCC*mec* IVA, pulsotype B–*agr-3*–SCC*mec* IVA, and pulsotype C–*agr-3*–SCC*mec* IIb–*etb* positive. Healthy schoolchildren in the Kyungnam province were also found to carry both SCC*mec* II and SCC*mec* IV MRSA clones (30). It thus appears that both SCC*mec* IV and SCC*mec* II clones of CA-MRSA have emerged in South Korea. CA-MRSA isolates in Taiwan and Japan did not always harbor SCC*mec* IV (7, 14, 39). SCC*mec* II is also predominant among CA-MRSA isolates in Japan (45), SCC*mec* III occurred frequently, and a novel SCC*mec* type (type V) was found among CA-MRSA isolates in Taiwan (7). MRSA isolates were all negative for *pvl*, and *etb* was exclusively correlated with the pulsotype B-SCC*mec* IIb clone in this study. As in this study, SCC*mec* IIb, first described in Japanese CA-MRSA isolates, also carries *etb* (45). There was no *pvl* gene found in CA-MRSA isolates from South Korea or Japan (14, 26, 30, 32, 39), while the *pvl* gene was present in those from Taiwan (44). Combined with the findings that *eta* was confined to *agr-4* MSSA and *tst* was found in MRSA or MSSA isolates of the *agr-1* or *agr-3* type, these toxin genes indicate the evolution and spread of certain *S. aureus* strains. In Asian countries, CA-MRSA clones seem to have an origin distinct from those of CA-MRSA epidemic clones in Australia, the United States, and Europe (7, 14, 17, 18, 39). Well-organized prospective surveillance is thus required to understand the epidemiology of CA-MRSA in South Korea.

Consistent with the previous reports of CA-MRSA, the MRSA isolates were susceptible to antimicrobials of many different classes, as were MSSA isolates; however, MLS_B resistance was common in erythromycin-resistant, clindamycin-susceptible isolates. Clindamycin is a treatment option for CA-MRSA infections in the United States because the isolates were usually susceptible to clindamycin and MLS_B induction test negative (10). In South Korea, clindamycin should not be used for clindamycin-susceptible CA-MRSA infections without MLS_B induction testing. Fortunately, skin and soft tissue infection can be treated without antimicrobial therapy if the area of infection is drained properly (34). However, as is the case with otitis media, CA-MRSA infection of tissues other than skin and soft tissue offers a challenge to antimicrobial therapy in South Korea (28).

In conclusion, AD patients showed high rates of MRSA colonization, and such patients may represent a significant reservoir of CA-MRSA. The major MRSA clone demonstrated known characteristics of CA-MRSA, including SCC*mec* type IV and a lack of multidrug resistance. MRSA isolates showed clonality by *agr* typing, PFGE, SCC*mec* typing, and toxin assays, suggesting a clonal spread of CA-MRSA.

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