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Prevalence of Inducible Clindamycin Resistance among Community- and Hospital-Associated *Staphylococcus aureus* Isolates

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Methicillin-resistant *Staphylococcus aureus* (MRSA) infections have become common among both hospitalized and nonhospitalized patients. Optimal outpatient therapy for MRSA infections has yet to be determined, but this matter is complicated by the possibility of inducible macrolide-lincosamide-streptogramin B resistance (MLSBi). We studied the prevalence of MLSBi in community- and hospital-associated *S. aureus* isolates and the prevalence of community-associated MRSA (CA-MRSA) and identified clinical predictors of CA-MRSA and MLSBi. Among 402 *S. aureus* isolates, the overall prevalence of MLSBi was 52%, with 50% of MRSA and 60% of methicillin-susceptible *S. aureus* isolates exhibiting MLSBi. CA-MRSA represented 14% of all isolates and had a lower prevalence of MLSBi than hospital-associated MRSA (33% versus 55%). The presence of skin or soft-tissue infection was predictive for CA-MRSA, and the presence of a comorbidity was predictive for MLSBi. Due to the low prevalence of MLSBi among CA-MRSA isolates, clindamycin remains a useful option for outpatient therapy.

Infections due to methicillin-resistant *Staphylococcus aureus* (MRSA) have become increasingly common among nonhospitalized patients. Numerous outbreaks of MRSA infections have been recognized in previously healthy people who lacked traditional risk factors for acquisition of MRSA (1, 2, 4–7). Skin and soft-tissue infections (SSTIs) are a common manifestation of staphylococcal disease in many community outbreaks, with invasive staphylococcal disease being less common. Recent work has determined that these community-associated MRSA (CA-MRSA) strains are genetically distinct from health care-associated MRSA (HA-MRSA). Community-associated MRSA strains frequently harbor virulence factors different from those of HA-MRSA strains, particularly the Panton-Valentine leukocidin genes, which are associated with SSTIs (1, 11, 20, 26). In addition, methicillin resistance is conferred by the type IV staphylococcal chromosome cassette *mec* (SCC*mec*IV) gene, which is distinct from types I through III, which have previously been found in HA-MRSA strains (17).

As MRSA infections have become increasingly common in the community setting, the development of empirical antimicrobial therapeutic strategies for SSTIs has become more problematic. Clindamycin has long been an option for treating both methicillin-susceptible *S. aureus* (MSSA) and MRSA infections, particularly for SSTIs. However, expression of inducible resistance to clindamycin could limit the effectiveness of this drug.

Previous reports of inducible macrolide-lincosamide-streptogramin B resistance (MLSBi) among *S. aureus* isolates were derived from cross-sectional analyses with a wide range of MLSBi prevalences (7% to 94%) (12, 22–25). Data describing MLSBi prevalence or clinical predictors of the presence of MLSBi among CA- and HA-MRSA isolates are quite limited. In the present study, we aimed to characterize MLSBi resistance in both hospital- and community-associated *S. aureus* isolates, including MRSA and MSSA, at our institution.

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MATERIALS AND METHODS

Microorganisms and antimicrobial susceptibility tests. We prospectively collected sequential nonduplicate *S. aureus* isolates exhibiting erythromycin resistance and clindamycin susceptibility, as determined by broth microdilution using an automated microbiology instrument (Dade MicroScan, West Sacramento, CA) from April to August 2004. Testing for MLSBi was accomplished by the agar disk diffusion (D test) method in accordance with the recommendations of the Clinical and Laboratory Standards Institute (CLSI) (9).

The anatomic sources of the bacterial isolates and their susceptibilities to other antimicrobials, including oxacillin, gatifloxacin, trimethoprim-sulfamethoxazole, tetracycline, linezolid, and daptomycin, were also recorded. The daptomycin susceptibility test was performed using the agar gradient diffusion (E test) technique in accordance with the manufacturer's instructions (AB Biodisk, Solna, Sweden). All antimicrobial susceptibility tests were interpreted in accordance with the 2004 guidelines published by the CLSI (9).

Medical records for the source patients were reviewed for demographic information, history of prior hospitalization, ZIP code of residence, presence of a major comorbid condition (e.g., diabetes mellitus, renal dysfunction, postsurgical status, malignancy, solid-organ or stem cell transplant, neutropenia, trauma, or burn injury), and antibiotic exposure within the preceding year. Based on available records, determination was made as to whether a clinical infection that was due to the *S. aureus* isolate cultured was present, as opposed to asymptomatic colonization. Infection was assumed to be present in all cases in which bacterial isolates were derived from blood or cerebrospinal fluid. If no determination could be made based on available records, the presence of infection was considered "unknown."

MRSA isolates were designated HA-MRSA if the source patient had any of the following risk factors: a history of hospitalization, residence in a long-term

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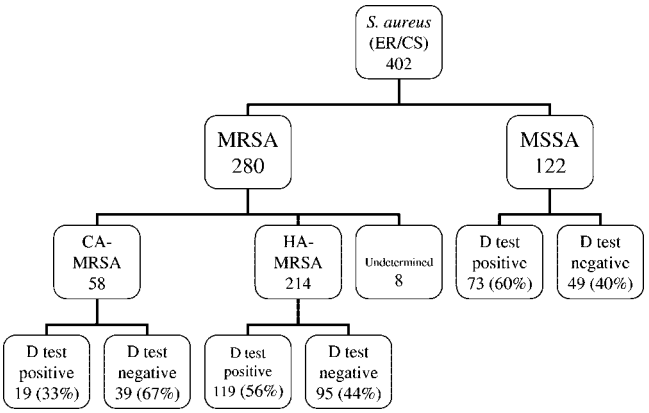

FIG. 1. Classification of *S. aureus* isolates. ER, erythromycin resistant; CS, clindamycin susceptible.

care facility (e.g., nursing home), dialysis, or surgery within one year prior to the date of specimen collection; growth of MRSA 48 h or more upon admission to a hospital; presence of a permanent indwelling catheter or percutaneous device at the time of culture; or prior positive MRSA culture prior to this study. If none of the above risk factors were present, the isolate was considered CA-MRSA. This study was approved by the University of Alabama at Birmingham institutional review board.

Statistical methods. Univariate comparisons utilize the chi-square analysis for categorical results and the Kruskal-Wallis test for continuous variables. The logistic-regression model was used to determine independent factors related to positive D test results for *S. aureus* isolates (14). The logistic-regression model was also used to determine independent predictors of community-associated *S. aureus* infection. *P* values of ≤ 0.05 were considered significant.

RESULTS

During the study period, 405 *S. aureus* isolates with the erythromycin-resistant/clindamycin-susceptible phenotype were prospectively collected, of which 402 were available for D test analysis (Fig. 1). Among these, 280 (70%) were MRSA and 122 (30%) were MSSA. The presence of MLSBi was confirmed by the D test for 212 (52%) of the isolates overall, with 139 (50%) MRSA and 73 (60%) MSSA isolates exhibiting MLSBi. Of the 402 staphylococcal isolates, 286 (71%) were classified as health care associated, 99 (25%) were classified as community associated, and 17 (4%) could not be confidently classified and were termed "undetermined." Positive D test results were noted in 56% and 41% of the health care-associated and community-associated *S. aureus* isolates, respectively ($P = 0.03$). One of the undetermined MRSA isolates produced a positive D test result. Among the 280 MRSA isolates, 58 (21%) were designated CA-MRSA, 214 (76%) HA-MRSA, and 8 (3%) undetermined. The presence of MLSBi was detected in 19 (33%) CA-MRSA and 119 (56%) HA-MRSA isolates.

The demographic and clinical characteristics of patients with *S. aureus* infections from whom isolates producing positive versus negative D test results were obtained are outlined in Table 1. The ages, sexes, and races of the patients were not predictive of positive D test results in the univariate analysis. However, isolates with positive D test results were more likely to be health care associated (76% versus 64%, $P = 0.03$) and associated with patients with any comorbidity (95% versus 81%, $P = 0.0001$). The presence of infection was slightly higher for isolates with negative D test results (79% versus 70%, $P = 0.05$). Prior antibiotic exposure showed a strong trend of being predictive of positive D test results ($P = 0.08$). Multivariate analysis revealed that the presence of any comorbidity in a patient was predictive of a positive D test result (Table 2). Methicillin resistance was not predictive of positive D test results among all *S. aureus* isolates.

The only positive predictor of community-associated-isolate designation among all *S. aureus* isolates was the presence of skin and soft-tissue infections (odds ratio [OR] = 7.42; $P < 0.0001$), while hospital-associated isolates were more likely to be methicillin resistant (OR = 3.45; $P < 0.0001$) and more likely to be isolated in individuals with comorbidities (OR = 11.11; $P < 0.0001$) (Table 2). Among patients from whom MRSA isolates were obtained, the community-associated-isolate designation was again linked significantly only to the presence of SSTI (OR = 19.19; $P < 0.0001$), whereas hospital-associated MRSA isolates were associated with the presence of specific comorbidities (trauma, $P = 0.001$; recent surgery, $P = 0.0003$; premature birth, $P = 0.01$) and prior antibiotic use ($P < 0.0001$).

Antibiotic susceptibility profiles for MRSA isolates are

TABLE 1. Comparison of D-test-positive and D-test-negative *S. aureus* isolates

Variable	No. (%) of:		*P*
	Positive D test results ($n = 212$)[a]	Negative D test results ($n = 189$)[b]	
Isolyte type			
MRSA	139 (66)	140 (74)	0.07
MSSA	73 (34)	49 (26)	
Classification			
Health care associated	161 (75)	120 (64)	0.03
Community associated	42 (20)	57 (30)	
Undetermined	9 (5)	12 (6)	
Sex			
Male	116 (54)	102 (54)	0.921
Female	96 (46)	87 (46)	
Race			
Caucasian	137 (64)	108 (57)	0.11
African-American	68 (32)	77 (41)	
Other	4 (2)	1 (0.5)	
Unknown	4 (2)	3 (1.5)	
Comorbidity			
Present	189 (89)	144 (76)	0.0001
Absent	10 (5)	38 (20)	
Unknown	13 (6)	7 (4)	
Related infection			
Present	134 (63)	137 (73)	0.05
Absent	57 (26)	36 (19)	
Unknown	21 (11)	16 (8)	
Prior antibiotic use			
Present	109 (51)	81 (44)	0.08
Absent	43 (20)	50 (27)	
Unknown	61 (29)	58 (29)	

[a] The mean age of patients in this group was 45 years (range, 0 to 91 years) ($P = 0.91$).

[b] The mean age of patients in this group was 42 years (range, 0 to 91 years) ($P = 0.91$).

TABLE 2. Multivariate analyses

Isolate group, test result and/or isolate origin, and associated factor	*P*	OR (95% CI)[a]
All *S. aureus* isolates		
Positive D test results		
Methicillin resistance	0.017	0.55 (0.33–0.90)
Comorbidity present	0.001	3.51 (1.56–7.92)
Community-acquired status		
Methicillin resistance	<0.0001	0.29 (0.17–0.52)
Comorbidity present	<0.0001	0.09 (0.04–0.22)
SSTI	<0.0001	7.42 (3.71–14.87)
Osteomyelitis	0.029	0.28 (0.09–0.87)
MRSA isolates		
Community-associated status		
Comorbidity present	0.0007	0.10 (0.03–0.38)
SSTI	<0.0001	19.19 (5.89–62.56)
Osteomyelitis	0.081	0.17 (0.02–1.24)
Prior antibiotic use	<0.0001	0.04 (0.01–0.12)
Positive D test results and community-associated status		
Comorbidity present	0.021	7.72 (1.37–43.48)
SSTI	0.005	0.13 (0.03–0.53)

[a] CI, confidence interval.

TABLE 3. Antimicrobial susceptibility patterns of MRSA

Antimicrobial	% of isolates susceptible[a]	
	CA-MRSA (n = 58)	HA-MRSA (n = 214)
Oxacillin	0	0
Trimethoprim-sulfamethoxazole	100	99
Tetracycline	89.7	92.9
Gatifloxacin	50	42.4
Vancomycin	100	100
Linezolid	100	100
Daptomycin	100	100

[a] All isolates were erythromycin resistant and clindamycin susceptible on routine antibiotic testing.

shown in Table 3. All isolates were erythromycin resistant and clindamycin susceptible (prior to D test analysis). All isolates tested were susceptible to vancomycin, daptomycin, and linezolid. Susceptibilities to trimethoprim-sulfamethoxazole were 100% and 99% among CA- and HA-MRSA isolates, respectively.

DISCUSSION

Macrolide resistance may be constitutive or inducible in the presence of either a macrolide or a lincosamide inducer. Among MLSBi strains, an inducer promotes production of methylase by *erm* genes and subsequent methylation of the 23S ribosome, thus making the strain fully resistant to the lincosamides (e.g., clindamycin) and group B streptogramins. Phenotypically, these MLSBi strains appear to be resistant to erythromycin and susceptible to clindamycin on routine antibiotic susceptibility testing. However, inducible resistance can be expressed during a double-disk diffusion test (D test), in which an erythromycin disk will induce clindamycin resistance, thus exhibiting constitutive resistance. Clinically, bacterial strains exhibiting MLSBi have a high rate of spontaneous mutation to constitutive resistance, which could be selected for by use of clindamycin.

Empirical outpatient treatment options for staphylococcal infections have become more limited as concerns about the prevalence of MRSA have increased. In some settings, particularly in outbreaks, the majority of cases of SSTIs are due to MRSA (1, 2, 4, 6, 7). Clindamycin has long been an attractive option because of its efficacy against MSSA and MRSA, its good bone and tissue penetration, and its potential antitoxin effects. Our study suggests that MLSBi is common in both health care-associated and community-associated *S. aureus* isolates (56% and 33%, respectively). In addition, a significant proportion of the *S. aureus* isolates at our hospital were designated community acquired by clinical definition (25%), a trend that has been seen nationally (13). In 2003, 72% (897 of 1,246) of all *S. aureus* isolates collected in the clinical laboratory at the University of Alabama at Birmingham Hospital exhibited the erythromycin-resistant/clindamycin-susceptible phenotype.

Among CA-MRSA isolates, the presence of any of the comorbidities diabetes mellitus, renal dysfunction, postsurgical status, malignancy, solid-organ or stem cell transplant, neutropenia, trauma, or burn injury was a positive predictor of the presence of MLSBi, while presentation with SSTI would suggest the absence of MLSBi. CA-MRSA isolates causing SSTI are likely to be clones of MRSA that have been recognized to be associated with cutaneous abscesses. Specifically, these clones likely have type IV SCC*mecA* genes.

Inducible MLSB resistance is conferred by the presence of any of the *erm* A, B, and C genes. A transposon, Tn*554*, harbors *ermA* and is generally absent from type IV SCC*mecA* clones of CA-MRSA, probably explaining the low prevalence of MLSBi among the CA-MRSA isolates in this study (18). The prevalence of MLSBi among CA-MRSA isolates in a pediatric population has been noted to decrease over time, probably representing an expansion of MRSA clones that lack MLSBi genes (8). It is possible that over time, Tn*554* or other MLSBi-associated transposons could become part of the genome of CA-MRSA via transduction. We did not identify the SCC*mecA* gene type in this study but expect that many of our CA-MRSA isolates would be type IV and thus lack the *erm* genes. CA-MRSA isolates with type IV SCC*mecA* likely express erythromycin resistance (without lincosamide resistance) through the presence of an efflux pump encoded by the *msr*(A) gene (3). The implication of this finding is that patients who lack comorbidities and have SSTI due to CA-MRSA may reasonably be offered clindamycin as a treatment option because of a lower likelihood that the strains contain MLSBi. However, among HA-MRSA isolates, the prevalence of MLSBi was significant and should be considered when determining treatment options.

There have been only a few reports describing patients who received clindamycin for *S. aureus* infections with MLSBi (10, 12, 15, 19, 21, 25). Clinical failures were present in 9 of the 12 (75%) patients described, with development of constitutive MLSB resistance in all but 1 patient. No clinical trials investigating the relationship between MLSBi and clindamycin failures have been performed to date. Currently, some authors recommend avoidance of clindamycin for treatment of com-

plicated infections which may have a high bacterial burden, such as abscesses or osteomyelitis (16). If clindamycin is used for treatment of a less severe MLSBi *S. aureus* infection, the patient should be closely monitored for signs of failure or relapse of infection. Non-MLSBi infections can be treated with clindamycin if appropriate. The results of this study represent *S. aureus* isolates from a single hospital and geographic area; the prevalences of MLSBi, CA-MRSA, and specific clones of MRSA may differ in different regions. Clinical microbiology laboratories should adopt testing for MLSBi if local prevalence is found to be substantial, with isolates which exhibit MLSBi reported as being clindamycin resistant.

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Conflicts of interest are as follows. Mukesh Patel is a member of the Speakers Bureau for Pfizer Pharmaceuticals and Wyeth Pharmaceuticals. Ken B. Waites has received grant support from Pfizer. Craig J. Hoesley is a member of the Speakers Bureau for Cubist Pharmaceuticals.

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