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Comparisons of Community-Associated Methicillin-Resistant *Staphylococcus aureus* (MRSA) and Hospital-Associated MSRA Infections in Sacramento, California

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Methicillin-resistant *Staphylococcus aureus* (MRSA) has long been a common pathogen in healthcare facilities, but in the past decade, it has emerged as a problematic pathogen in the community setting as well. A retrospective case series study of patients from whom MRSA was isolated from December 1, 2003, through May 31, 2004, was conducted at the University of California, Davis, Medical Center. Patient data were collected from electronic medical records and traditional chart reviews to determine whether MRSA acquisition was likely to have been in the community or in the hospital. Antimicrobial susceptibility testing and pulsed-field gel electrophoresis (PFGE) were performed for all confirmed isolates. Skin and soft tissue were the most common infection sites for all MRSA patients. Among the 283 MRSA infections, 127 (44.9%) were defined as community-associated (CA)-MRSA. Ninety-six percent of the CA-MRSA isolates were susceptible to clindamycin. Double-disk diffusion tests were performed to examine inducible clindamycin resistance by erythromycin induction on both CA and hospital-associated (HA) clindamycin-susceptible and erythromycin-resistant isolates. Ten percent (17 of 183) were positive. Most CA-MRSA isolates were identified by PFGE as a unique strain, genotype USA300, which was not genetically related to the predominant genotype, USA100, in the HA-MRSA isolates. Injecting drug users accounted for 49% of CA-MRSA infections but only 19% of the HA-MRSA infections (odds ratio, 4.2; 95% confidence interval, 2.4 to 7.4). Our study shows that a single clone of CA-MRSA accounts for the majority of infections. This strain originated in the community and is not related to MRSA strains from healthcare settings. Injecting drug users could be a major reservoir for CA-MRSA transmission.

The first methicillin-resistant *Staphylococcus aureus* (MRSA) case was reported in the United Kingdom in 1961 (15), shortly after methicillin was introduced into clinical practice. Seven years later, after the resistant strain had become widespread in Japan, Europe, and Australia, the first case of MRSA in the United States was described in 1968 (2). Traditionally, MRSA has been considered a major nosocomial pathogen in healthcare facilities, but in the past decade, it has been observed emerging in the community as well. The first case of community-associated MRSA (CA-MRSA) infection in the United States was reported in 1980 (21). More-widespread identification of CA-MRSA in the United States began in the 1990s, following the report of CA-MRSA infections among four children (5). Patients with CA-MRSA infections have often lacked risk factors known for patients with hospital-associated MRSA (HA-MRSA) infections, which include recent hospitalization, dialysis, nursing-home residence, and other co-morbid conditions such as diabetes, chronic renal failure, and chronic pulmonary diseases which bring them into contact with healthcare settings. CA-MRSA has also been found to be composed of more-diverse clonal groups than HA-MRSA and to usually contain a unique *SCCmec* type IV DNA element (13). Clusters of CA-MRSA infection have been described among aboriginals in Australia (18), rural Native American communities in the United States (14), prisoners (7), sports players (6), children (12), and injecting drug users (IDUs) (21). The substantial increase in CA-MRSA infections has increased the challenge of selecting empirical antimicrobial treatments in outpatient settings. Previous studies have reported that in the United States, the prevalence of CA-MRSA infections varies from 76% among MRSA skin and soft tissue infection (SSTI) isolates in Alaska (1) to 12% of all MRSA infections in Minnesota (20). These reports prompted us to review the current epidemiology of MRSA in Sacramento, California, a metropolitan area of approximately 1.2 million people. In this study, we describe and compare the characteristics and MRSA strains of patients treated for CA-MRSA and HA-MRSA infections at the University of California, Davis, Medical Center (UCDMC) from December 1, 2003 to May 31, 2004.

MATERIALS AND METHODS

Case ascertainment and definition. This is a retrospective case series study. Samples from adult patients (both inpatients and outpatients) with MRSA infections newly identified in the microbiology laboratory at the UCDMC were collected from December 1, 2003, through May 31, 2004. None of these was obtained as a "screening" or "surveillance" culture for MRSA. The UCDMC is a tertiary referral center serving primary care, emergency, and hospitalization needs of the majority of medically uninsured and indigent patients in Sacramento county. The average inpatient census is approximately 450 patients, and its outpatient services experience 2,270 visits per day. Duplicate isolates collected

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TABLE 1. Basic demographics of patients with CA-MRSA versus HA-MRSA infections from whom samples were collected within a 6-month period

Characteristic	Value for group[a]		*P* value
	CA-MRSA	HA-MRSA	
Gender[b]			
Female	55 (43)	62 (40)	
Male	72 (57)	94 (60)	0.47
Mean age ± SD (yr)	39 ± 13	54 ± 18	<0.001
Age group[b]			
18–29	35 (28)	15 (10)	
30–39	28 (22)	16 (10)	
40–49	36 (28)	33 (21)	
50–59	21 (17)	32 (21)	
60+	7 (5)	60 (38)	
Ethnicity[b]			
Caucasian	47 (52)	63 (47)	
Black	20 (23)	17 (12)	
Hispanic	13 (14)	11 (8)	
Other[c]	10 (11)	44 (33)	0.0025
Total	90	135	
Length of hospital stay (days)			
Mean	2.8	21.4	<0.001
Median	1.5	13	<0.001
Range	0–17	0–150	
Type of insurance[b]			
Private	23 (19)	69 (46)	
Public	80 (85)	72 (49)	
None	19 (16)	7 (5)	<0.001
Total	122	148	

[a] A total of 127 patients were in the CA-MRSA group, and a total of 156 were in the HA-MRSA group.
[b] Values are numbers of patients. Values in parentheses are percentages.
[c] Including persons classified as Asian, Pacific Islander, or Native American.

TABLE 2. Sites of CA-MRSA and HA-MRSA infections

Infection site	No. (%) of patients for indicated MSRA[a]		*P* value	OR (95% CI)
	CA-MRSA	HA-MRSA		
Bloodstream	3 (2)	17 (11)	0.0053	0.2 (0.06–0.7)
Respiratory tract	3 (2)	50 (32)	<0.001	0.02 (0.002–0.17)
Skin and soft tissue	109 (86)	65 (42)	<0.001	8.5 (4.7–15.3)
Urinary tract	1 (1)	13 (8)	0.004	0.09 (0.01–0.68)
Other[b]	11 (9)	11 (7)	0.64	0.9 (0.76–1.89)

[a] A total of 127 patients were in the CA-MRSA group, and a total of 156 were in the HA-MRSA group.
[b] Includes endocarditis, osteoarthritis, joint infection, diabetic foot infection, and ocular infections.

from the same patient were excluded. HA-MRSA infection was defined as occurring in a patient whose MRSA isolate was cultured more than 48 h after admission, who had a history of hospitalization, surgery, dialysis, or residence in a long-term healthcare facility within 6 months prior to the culture date, or who had an indwelling intravenous line, catheter, or any other percutaneous medical device present at the time the culture was taken. Patients with none of the above conditions were classified as having CA-MRSA infection. Patients who had had an MRSA-positive isolate prior to the study period were excluded from the study.

Data collection. Information was extracted from the electronic medical records and by traditional chart review and recorded on a standard data collection sheet. Data obtained about the study subjects included basic demographics, reason for admission, medical history (underlying diseases), medication history, sites of MRSA infection, culture site, length of hospital stay, social history, and isolate characterization (e.g., antimicrobial susceptibility and molecular typing results) (11). Antimicrobial administration within 30 days before the study was recorded as well.

Characterization of isolates. All MRSA cultures were confirmed in the UCDMC microbiology laboratory. Susceptibility testing was performed by the Sceptor system microtiter dilution method (Becton-Dickinson, Franklin Lakes, N.J.). Susceptibility to cefazolin, clindamycin, ciprofloxacin, erythromycin, gentamicin, oxacillin, rifampin, tetracycline, trimethoprim-sulfamethoxazole, and vancomycin was determined. Oxacillin was used for methicillin susceptibility testing. The results were categorized according to the guidelines of the National Committee for Clinical Laboratory Standards (now Clinical Laboratory Standards Institute) and confirmed with Denka latex agglutination. The double-disk diffusion test (D-test) (23) was performed on all MRSA isolates whose antimicrobial susceptibility patterns were clindamycin susceptible and erythromycin resistant. The test was used to estimate the proportion of inducible macrolide-lincosamide-streptogramin B (iMLSB) resistance.

Molecular typing of MRSA strains was done by pulsed-field gel electrophoresis (PFGE) with SmaI restriction endonucleases at Stanford University, using a method previously published (10). For visual strain analysis, isolates were considered different strains if their PFGE patterns differed by ≥4 bands (25).

Statistical analysis. SAS version 8.1 software was used for statistical analysis (SAS Institute, Cary, N.C.). Descriptive analysis (univariate analysis) was employed in investigating the distributions of variables between the HA and CA groups. Categorical variables between the two groups were compared by means of the chi-square test or Fisher's exact test if 20% of the expected values were smaller than five. Continuous variables were analyzed using the two-tailed *t* test. A *P* value of <0.05 was considered statistically significant with chi-square distribution. Analysis of variance was used for three-group comparisons.

RESULTS

Within the 6-month study period, 283 out of 328 patients with individual MRSA-positive isolates were eligible for our study by the inclusion criteria. The proportion of MRSA infections among all *S. aureus* isolates was 42% during the study period. In our data, 156 (55.1%) met the definition of HA-MRSA infections and the rest; 127 patients (44.9%), were classified as CA. The basic demographics of the MRSA patients are shown in Table 1. The CA-MRSA group had significantly different distributions from the HA-MRSA group with respect to age, length of hospital stay, ethnicity, and insurance status ($P < 0.01$ for all comparisons). Among those whose occupation was documented, 67 (64%) CA-MRSA patients were not employed, as were 36 (29%) in the HA-MRSA group ($P < 0.001$).

A significant difference between the two groups was in the sites of MRSA infection (Table 2). Skin and soft tissue were the most common infection sites among all subjects (174 of 283, 61%), especially among CA patients (109 of 127, 85.8%). We compared underlying conditions in the patients with CA-MRSA to those with HA-MRSA. Besides dermatological conditions, diabetes ($P = 0.0023$), chronic renal disease ($P < 0.001$), and cancer ($P = 0.0012$) were the most common underlying conditions observed in the HA-MRSA group. Of a total of 253 patients whose drug use histories were recorded, nearly half of CA-MRSA patients (53 of 108, 49.1%) were IDUs compared with only 18.6% (27 of 145) of the HA-MRSA group (odds ratio [$\hat{OR}$] = 4.2; $P < 0.001$; 95% confidence interval (CI), 2.4 to 7.4).

Among HA-MRSA patients with SSTI, the isolates were

TABLE 3. PFGE results for CA-MRSA and HA-MRSA isolates

Source of MRSA infection[a]	No. (%) of isolates with indicated genotype							*P* value
	USA100	USA200	USA300	USA400	USA600	USA800	Unique	
CA-MRSA	6 (5)	0	108 (87)	2 (2)	0	0	8 (6)	
HA-MRSA	69 (47)	1 (0.6)	48 (33)	1 (0.6)	1 (0.6)	1 (0.6)	26 (17.6)	<0.001

[a] A total of 124 isolates were in the CA-MRSA group, and a total of 147 were in the HA-MRSA group.

more likely to be susceptible to clindamycin ($O\hat{R}$ = 2.7; 95% CI, 1.4 to 5.2; $P = 0.003$) and tetracycline ($O\hat{R}$ = 0.17; 95% CI, 0.05 to 0.54; $P = 0.001$) than were isolates associated with other sites of infection.

Molecular typing. The results of PFGE identified seven clonal groups among our isolates, with 28 distinct subtype patterns (Table 3). One PFGE clonal type, designated clonal group USA300, accounted for 87% (108 of 124) of CA-MRSA isolates but only 33% (48 of 47) of HA-MRSA isolates ($P < 0.001$). Another PFGE clonal group, group USA100, predominated only in the HA-MRSA group, accounting for 47% (69 of 147) of HA isolates. Further, among the HA isolates, USA300 appeared predominantly in patients with a history of injecting drug use (16 of 26, 69%), in contrast to the non-IDU group (26 of 110, 23.6%; $P = 0.009$).

Antimicrobial susceptibility patterns. The prevalence of resistance to each antimicrobial tested is presented in Table 4, which includes the D-test-positive results as well. CA-MRSA isolates were more likely to be susceptible than were HA-MRSA isolates with respect to ciprofloxacin ($P < 0.001$) and clindamycin ($P < 0.001$).

One hundred eighty-three isolates were analyzed by the D-test to evaluate for iMLSB resistance. Seventeen (10.2%) of them were positive.

The results of comparing antimicrobial patterns between CA-MRSA USA300 and HA-MRSA USA300 are shown in Table 5. Even though similar with respect to molecular strain, HA-MRSA USA300 had significantly higher prevalence of resistance to ciprofloxacin ($P = 0.023$). A higher proportion of HA-MRSA USA300 isolates were resistant to clindamycin than were CA-MRSA USA300 isolates, almost reaching statistical significance level ($P = 0.072$). By comparing susceptibility results between HA-MRSA USA300 and HA-MRSA non-USA300 types (including USA100, USA400, USA800, and other, unique strains), we found that the non-USA300 nosocomial strains were more likely to be resistant than USA300 types to ciprofloxacin and clindamycin but less likely to be resistant to tetracycline ($P < 0.01$ in all comparisons).

DISCUSSION

This study demonstrates that a high proportion (45%) of MRSA patients identified in our institution had CA-MRSA infections. Our findings confirmed our clinical impression that CA-MRSA had emerged in our community. Overall, CA-MRSA infection was unlikely to result in prolonged hospitalization in our community. Most of these CA-MRSA infections were of the skin and soft tissue types, which responded quickly to wound care (incision and drainage) when indicated and to outpatient oral antimicrobial therapy. The distribution of MRSA infection sites for CA-MRSA and HA-MRSA groups was consistent with those of previous studies (18, 20).

CA-MRSA isolates were more susceptible to multiple antibiotics such as ciprofloxacin and clindamycin. Nevertheless, our results showed an unusually high prevalence of resistance to erythromycin (93%), in contrast to other reports of 69% in Alaska (1) and 61% in the San Francisco urban poor study (9). Further, unlike other studies, in our data the prevalence of MRSA strains expressing iMLSB was relatively low (10.2%) (16), suggesting that clindamycin remains one option for effective antimicrobial treatment in our community.

Our data demonstrate that USA300 was not only the most common strain among all MRSA isolates circulating among CA-MRSA patients but was also highly related to SSTI. This strain was not genetically related to the common nosocomial strain in HA-MRSA isolates, USA100. This finding and antimicrobial susceptibility patterns support the conclusion that

TABLE 4. Antimicrobial susceptibility results for CA-MRSA and HA-MRSA isolates

Antimicrobial	Percentage of susceptible isolates from indicated MRSA		*P* value
	CA-MRSA	HA-MRSA	
Methicillin	0	0	
Ciprofloxacin	53	14	<0.001
Clindamycin	96	48	<0.001
Erythromycin	7	8	1
Gentamicin	100	98	0.5
Tetracycline	80	88	0.1
Rifampin	100	98	0.5
TMP-SMX[a]	100	98	0.5
Vancomycin	100	100	

[a] A total of 127 isolates were in the CA-MRSA group, and 147 were in the HA-MRSA group.
[b] TMP-SMX, trimethoprim/sulfamethoxazole.

TABLE 5. Susceptibility patterns for CA-MRSA USA300, HA-MRSA USA300, and HA-MRSA non-USA300 types

Antimicrobial	Percentage of susceptible isolates for indicated MRSA type			*P* value
	CA-MRSA USA300	HA-MRSA USA300	HA-MRSA non-USA300	
Methicillin	0	0	0	
Ciprofloxacin	51	31	12	<0.001
Clindamycin	98	92	27	<0.001
Erythromycin	1	4	10	0.01
Gentamicin	100	100	98	0.202
Tetracycline	77	72	97	<0.001
Rifampin	100	100	98	0.21
TMP-SMX[a]	100	100	97	0.1
Vancomycin	100	100	100	

[a] TMP-SMX, trimethoprim/sulfamethoxazole.

CA-MRSA infection is not a nosocomial strain which originated in local healthcare facilities, but a distinct clone that has developed and is being propagated within the community.

It has been well documented (4, 22) that skin and soft tissue infections, such as abscesses and cellulitis, are directly related to injecting drug use. Other studies (3, 8, 17, 24) have reported that IDUs were commonly colonized or infected with *S. aureus*, anaerobes, and facultative gram-positive cocci, with high rates of recurrent infection, particularly among those who were homeless. Although some investigators excluded IDUs as subjects (cases) for studying CA-MRSA infection (14, 19), one study (9) conducted in San Francisco indicated that injecting drug use may be responsible for increasing MRSA colonization in the urban poor community. Similarly, given the fact that USA300 (predominant in the community) has high prevalence in IDUs of the CA-MRSA group, IDUs may be an important population that contributes significantly to the spread of clonal CA-MRSA. Injecting drug users frequently have SSTI and could transmit the organism to non-IDUs by close personal contact, serving as a significant community reservoir for CA-MRSA. Frequent skin puncture, poor injection site hygiene, syringe reuse, lack of personal hygiene knowledge and availability, and sharing needles are among the factors that facilitate CA-MRSA colonization/infection among IDUs.

The PFGE results showed that 33% of HA-MRSA isolates were USA300. There are two likely reasons for this finding. First, the organism may actually have been nosocomially transmitted. That the strain is no longer limited in the community but has spread into the hospital indicates the severity of CA-MRSA infection. This hypothesis is further supported by the finding that the USA300 isolates we found in the HA group exhibit an antimicrobial susceptibility pattern intermediate between those of CA USA300 and the HA non-USA300 types. This finding raises the concern that CA and HA strains may exchange genetic material, resulting in an organism uniquely adapted to produce aggressive SSTI-like CA-MRSA strains which carry the Panton-Valentine leucocidin gene as well as possessing resistance to multiple antimicrobial agents, like current HA strains. Such a development would further complicate efforts at limiting the impact of nosocomially associated *S. aureus* infections. Alternatively, the organism was colonizing the patient on admission but was identified more than 48 h after admission.

There were several limitations to our study. This is a healthcare-based retrospective case series study. Thus, we were unable to estimate the true prevalence of CA-MRSA infection in the general population or in the IDU population. Second, although medical charts were carefully reviewed, in the absence of personal interviews there is a risk of misclassifying MRSA acquisition due to lack of a detailed history of hospital-related exposures and failure to elicit an accurate history of injecting drug use, especially for those patients whose charts were incomplete. This would tend to bias the study toward underestimating injecting drug use. Third, due to limited resources, we did not test for the existence of the Panton-Valentine leucocidin gene harbored among CA-MRSA isolates. Overall, our data demonstrate a high proportion of CA-MRSA isolates, suggesting that the face of MRSA has changed in both epidemiological and microbiological features and in both community and hospital. HA-MRSA patients acquired their infections while under intensive treatment for other underlying diseases, resulting in additional diverse sites of infection and additional diverse clonalities. In this study, we not only strengthen the hypothesis that injecting drug use is contributing significantly to the increasing incidence of CA-MRSA but also discovered that the CA-MRSA strain has already disseminated into the hospital and has probably adopted multiresistant genes from the hospital strains.

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