MAJOR ARTICLE

Community-Associated Methicillin-Resistant *Staphylococcus aureus* Colonization Burden in HIV-Infected Patients

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(See the Editorial Commentary by Bootsma and Bonten on pages 1075–7.)

Background. The epidemic of community-associated methicillin-resistant *Staphylococcus aureus* (CA-MRSA) has had a disproportionate impact on patients with human immunodeficiency virus (HIV).

Methods. We evaluated CA-MRSA colonization burden (number of colonized sites per total number sampled) among HIV-infected and HIV-negative inpatients within 72 hours of hospitalization. From March 2011 through April 2012, we obtained cultures from nasal and extranasal sites (throat, axilla, inguinal, perirectal, and chronic wound if present) and collected risk factor data.

Results. Of 745 patients (374 HIV-infected, 371 HIV-negative), 15.7% were colonized with CA-MRSA at any site: 20% of HIV and 11% of HIV-negative patients (relative prevalence = 1.8, $P = .002$). HIV-infected patients had a higher prevalence of nasal, extranasal, and exclusive extranasal colonization as well as higher colonization burden. Perirectal and inguinal areas were the extranasal sites most frequently colonized, and 38.5% of colonized patients had exclusive extranasal colonization. Seventy-three percent of isolates were identified as USA300. Among HIV-infected patients, male sex, younger age, and recent incarceration were positively associated whereas Hispanic ethnicity was negatively associated with higher colonization burden. Among HIV-negative patients, temporary housing (homeless, shelter, or substance abuse center) was the only factor associated with higher colonization burden. Predictors of USA300 included HIV, younger age, illicit drug use, and male sex; all but 1 colonized individual with current or recent incarceration carried USA300.

Conclusions. HIV-infected patients were more likely to have a higher CA-MRSA colonization burden and carry USA300. In certain populations, enhanced community and outpatient-based infection control strategies may be needed to prevent CA-MRSA cross-transmission and infection.

Keywords. CA-MRSA; HIV; extranasal colonization.

The epidemic of community-associated methicillin-resistant *Staphylococcus aureus* (CA-MRSA) has spread rapidly both in the community [1] and in nosocomial settings [2]. Preliminary studies suggest that CA-MRSA may have colonization dynamics [3, 4] different from those of traditional MRSA strains, including having a disproportionately large effect on certain populations [5, 6].

Colonization with MRSA is felt to precede infection [7] with the anterior nares being the primary site of colonization [8]. However, colonization outside the nares—at the axilla, inguinal region, oropharynx, wounds, and gastrointestinal tract—occurs [9, 10]. Decolonization with mupirocin [11] or with mupirocin and chlorhexidine [12] is less successful and requires more attempts if >1 body site is colonized. Because colonized individuals are at increased risk of MRSA infection and can serve as a source of MRSA cross-transmission, the

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reduced effectiveness of decolonization among persons with higher MRSA colonization burden (ie, higher proportion of anatomic sites colonized per patient) has implications at both the patient and community level.

A 2003–2004 survey of the US population [13] observed an MRSA nasal colonization prevalence in the community of 1.5%; 19.7% of isolates were genotyped as USA300, the predominant CA-MRSA strain in the United States [14]. The low proportion of nares colonization with USA300 strains, despite the widespread CA-MRSA epidemic, raises the question of extranasal colonization and whether the true burden of USA300 MRSA colonization among the general population was underestimated. Outbreak investigations of USA300 MRSA infections often have not been able to detect nasal colonization at the time of infection and nasal culture results have not necessarily predicted who would develop CA-MRSA infection [15, 16]. This suggests either that colonization outside the nares plays a role in infection development or that the infections occur without, or with only transient, colonization—the "hit and run" theory [17].

CA-MRSA has had a disproportionately greater impact on certain patient populations. For example, in our experience patients infected with human immunodeficiency virus (HIV) have had >6-fold higher risk of CA-MRSA skin and soft-tissue infections (SSTIs) than have HIV-negative patients [5]. In addition, prevalence of nasal colonization with CA-MRSA is higher in HIV-infected patients [18] and HIV infection appears to be associated with persistent colonization [19]. However, risk for colonization, even among HIV-infected individuals, appears to be unevenly distributed [20].

A hypothesis to explain why certain community populations have increased risk of CA-MRSA infection is that persons within these populations have a higher colonization burden with USA300 MRSA, thereby facilitating spread of strains within the population. The objective of our study was to evaluate the epidemiology, prevalence, and colonization burden of CA-MRSA strains among HIV-infected and HIV-negative patients who were recently admitted to an acute care hospital. Given preliminary data demonstrating that current and former detainees are at increased risk of CA-MRSA, we included this population in our study.

METHODS

Subject Enrollment

From March 2011 through April 2012, patients at Stroger (formerly Cook County) Hospital (CCH), a 464-bed facility and the major safety-net hospital in the region, were eligible for enrollment in the study if they were admitted to a general medicine ward or the HIV ward service. Given the large number of general medicine admissions daily to CCH, a random sample of general medicine patients was selected and enrolled on designated study enrollment days. Approximately 7 general medicine and 7 HIV-infected patients were enrolled per week over the course of a year. Study subjects were enrolled within 72 hours of hospitalization; admissions from a nursing home or long-term care facility were not eligible for enrollment. An epidemiologic definition of CA-MRSA was used (enrollment within 72 hours of hospitalization); given the evolving epidemiology of the CA-MRSA epidemic [2, 21], patients with prior healthcare exposures were eligible for enrollment.

Enrollment of current detainees began August 2011. Detainees were individuals incarcerated at Cermak Health–Cook County Jail who were transferred to CCH for further medical care. The Cook County Jail has an approximate daily census of 10 000 detainees and CCH serves as the primary hospital for ill inmates who require hospitalization.

Culture Protocol and Microbiologic Testing

For enrolled patients, swab specimens were obtained from the nares, throat, bilateral axillae, bilateral inguinal regions, perirectal area, and a chronic wound if present (ie, if the patient was admitted to the hospital with a wound). Nasal specimens were obtained by swabbing both anterior nares; throat swabs were obtained by swabbing the posterior pharynx; perirectal swabs were obtained by swabbing the perirectal region; and axillary and inguinal swabs were obtained from a 10-cm^2 area of skin bilaterally. Dry swabs were used for the anterior nares and throat; swabs moistened in Amies transport medium were used for other sites. Swabs were collected with Starplex Scientific Starswab II (Thermo Fisher Scientific, Waltham, Massachusetts). To increase culture sensitivity, swabs were inoculated into an enrichment broth (salt-enriched trypticase soy broth). Aliquots of overnight broth cultures were inoculated on ChromID MRSA (bioMérieux, Durham, North Carolina). MRSA was identified by colony morphology, presence of β-hemolysis after subculture to tryptic soy agar plates containing 5% sheep's blood, and positive latex agglutination test (Staphaurex; Remel, Lenexa, Kansas). Methicillin resistance was confirmed by cefoxitin disk testing. Pulsed-field gel electrophoresis (PFGE) was performed as previously described [22] on all MRSA isolates to identify strains as USA300 or non-USA300 MRSA [22].

Risk Factors and Statistical Analysis

Assessment of CA-MRSA risk factors was done using a questionnaire in combination with review of medical records. Data elements collected included patient demographics, comorbidities, and community exposures (eg, drug use, type of housing, and incarceration). Chronic skin condition was defined as a dermatologic condition existing prior to hospitalization. Substance abuse was defined as current or former use of illicit drugs. Temporary housing was defined as being homeless or

residing in a shelter or substance abuse center currently or in the prior year.

Prevalence of CA-MRSA colonization was calculated (number positive per total number of individuals sampled) for nasal, extranasal, and exclusive extranasal (ie, nares cultures negative). Colonization burden (number of colonized sites per total number of sites sampled) was also determined. A comparison was made between HIV-infected and HIV-negative patients to determine a relative prevalence using Poisson regression.

Analysis was performed using results of surveillance cultures and risk factor data to further understand the epidemiology of extranasal colonization and colonization burden. SAS software version 9.2 (SAS Institute, Cary, North Carolina) was used for statistical analysis. Chi-square analysis was used for examination of categorical variables, with Fisher exact test used for small samples; continuous variables were analyzed with the independent-samples *t* test. Differences between medians were compared using the Wilcoxon (Mann-Whitney) test. Poisson regression was used to calculate relative prevalence and to model CA-MRSA colonization burden (outcome of interest). Statistically important (ie, $P < .2$) factors on univariate analysis were included in multivariate analysis. Variables were removed using backward elimination of covariates. Confounding was assessed by determining if the crude and adjusted estimates differed by ≥10%. During model building, effect modification was examined by introducing product terms and assessing the significance of the interaction term in the resulting model.

The study was approved by the CCH and Rush University Medical Center institutional review boards (IRB); verbal consent was obtained. The CCH IRB oversees local approval for enrollment of Cermak detainees; approval from the Office for Human Research Protections was obtained in order to enroll current detainees.

RESULTS

Features of the Study Population

Demographics and clinical and community features of the study population are shown in Table 1. Of 745 inpatients enrolled in the study, 374 were HIV-infected and 371 were HIV-negative. Sixty-three percent were African-American, 33% white, and the remaining Asian or other; 20% of the population was of Hispanic ethnicity. The mean age was 48 years (SD, 13.1 years) and 64% were male.

HIV-infected patients were younger and more likely to be African-American and male. In addition, they were more likely to be current or former users of illicit drugs; to have resided in temporary housing or a long-term care facility in the prior year; to report male homosexual contact; or to have a chronic skin condition or wound. In contrast, HIV-negative patients were more likely to have diabetes, to have immunosuppression unrelated to HIV, or to have an emergency room visit or hospitalization in the past year.

Prevalence of CA-MRSA Colonization

The overall prevalences of CA-MRSA nasal and extranasal colonization were 9.7% and 14.5%, respectively. Six percent of subjects had exclusive extranasal colonization. HIV-infected patients had a significantly higher prevalence of CA-MRSA colonization at any site (20%) in comparison to HIV-negative individuals (11%; $P = .002$). In addition, HIV-infected patients had a significantly higher prevalence of CA-MRSA nasal, extranasal, and exclusive extranasal colonization than did HIV-negative patients (Table 2). When extranasal colonization was present, perirectal and inguinal areas were the most frequently colonized sites for both HIV-infected and HIV-negative patients. When exclusive extranasal colonization was present, perirectal and inguinal areas were the most frequently positive sites for HIV-infected patients, whereas for HIV-negative patients, inguinal and throat colonization occurred most frequently (Table 2).

For HIV-infected patients, the mean CD4 count was 254 cells/mm^3 (SD, 256 cells/mm^3) and 35% had undetectable viral loads. There was no significant difference in mean CD4 count ($P = .72$) or median viral load ($P = .76$) between those patients who were and were not colonized with CA-MRSA.

There were a total of 55 individuals in the study who were current detainees or who had been incarcerated in the past year (36 HIV-infected and 19 HIV-negative) with the majority (85%) of individuals having jail but no prison exposure. The prevalence of colonization among current detainees or those with recent release (ie, within the prior 3 months) was 24%; 89% of colonized individuals with current or recent incarceration were HIV-infected.

Demographic and Exposure Factors Associated With Higher CA-MRSA Colonization Burden

Significant risk factors for higher CA-MRSA colonization burden on univariate analysis (Table 3) included HIV status, male sex, African-American race, illicit drug use, younger age, and exposure to temporary housing. There was a trend toward current or recent incarceration being associated with a higher colonization burden. Hispanic ethnicity was negatively associated with higher CA-MRSA colonization burden.

Presence of a chronic skin condition or chronic wound was associated significantly with a higher CA-MRSA colonization burden ($P < .001$). Breaks in skin and wounds have previously been recognized as factors associated with MRSA carriage [23, 24] and therefore skin disease could be viewed as a potential mediator for MRSA colonization. As there was a significantly

Table 1. Demographics, Comorbidities, and Community Exposures by HIV Status

	HIV-Infected Patients (n = 374)	HIV-Negative Patients (n = 371)	*P* Value
Race			<.001
African-American	279 (74.6%)	188 (51%)	
White	92 (24.6%)	156 (42%)	
Asian	3 (0.8%)	22 (6%)	
Other	0	5 (1%)	
Ethnicity			.131
Hispanic	68 (18%)	84 (23%)	
Non-Hispanic	306 (82%)	287 (77%)	
Age, y, mean (SD)	44.4 (10.8)	51.7 (14.1)	<.001
Sex			<.001
Male	280 (75%)	200 (54%)	
Female	94 (25%)	171 (46%)	
Current MRSA infection at time of enrollment	2 (0.5%)	3 (0.8%)	.69
MRSA infection within the prior year	24 (6%)	9 (2%)	.008
Illicit drug use: current or former	186 (50%)	85 (23%)	<.001
Residence in a nursing home or long-term care facility in the past year	21 (6%)	5 (1%)	.002
Temporary housing[a]	39 (10%)	16 (4%)	.001
Diabetes	47 (13%)	91 (25%)	<.001
Men who have sex with men[b]	132 (48%)	6 (4%)	<.001
Chronic hemodialysis	8 (2%)	8 (2%)	.987
Incarceration: current detainee or release in past 3 mo	24 (6%)	14 (4%)	.101
Incarceration type[c]			.616
Jail	19 (79%)	12 (86%)	
Prison or both	5 (21%)	2 (14%)	
Immunosuppression unrelated to HIV	22 (6%)	71 (19%)	<.001
Chronic skin condition or chronic wound	56 (15%)	28 (7.6%)	.001
Healthcare exposure in the past year[d]	231 (62%)	279 (75%)	<.001

Data are No. (%) of patients, unless otherwise indicated.

Abbreviations: HIV, human immunodeficiency virus; MRSA, methicillin-resistant *Staphylococcus aureus*; SD, standard deviation.

[a] Homeless or had residence in a substance abuse center or shelter currently or in the past year.

[b] Data were missing for 54 individuals; therefore, the resulting denominator was 276 for HIV-infected and 150 for HIV-negative individuals.

[c] Included only individuals who were current detainees or who had been incarcerated and released within the prior 3 months; the resultant denominator was 24 for HIV-infected and 14 for HIV-negative individuals.

[d] Healthcare exposure in the past year included visit to the emergency room or hospitalization at Cook County Hospital. Given the evolving epidemiology of the community-associated MRSA epidemic [21] along with the large proportion of our patients that have prior healthcare exposures [5], this variable was felt to be less useful as a predictor for community disease and was not included in risk factor analysis.

higher proportion of HIV-infected patients with chronic skin conditions and chronic wounds in comparison to HIV-negative patients ($P = .001$), chronic skin condition or wound was felt to be a mediator in the causal pathway from HIV status to CA-MRSA colonization and was not included in the multivariable model [25].

For the multivariable model (Table 4), incarceration exposure was defined as current incarceration or recent release (ie, within the prior 3 months), thus allowing us to better attribute risk for CA-MRSA to the correctional facility. There was differential risk for colonization burden based upon HIV status and incarceration exposure ($P = .019$) in the multivariable model and therefore stratified analysis by HIV status was performed. Among HIV-infected patients, current or recent incarceration, male sex, and younger age were all associated with a higher colonization burden. Hispanic ethnicity was associated with a lower colonization burden among HIV-infected patients (relative prevalence 0.39; 95% confidence interval [CI], .23–.66; $P < .001$). Among HIV-negative patients, temporary housing was the only factor associated with a higher CA-MRSA colonization burden (relative prevalence 2.14; 95% CI, 1.12–4.1; $P = .021$).

Table 2. Prevalence of Community-Associated Methicillin-Resistant *Staphylococcus aureus* Colonization by HIV Status and Colonization Pattern

Colonization Pattern[a]	Number of Individuals		Relative Prevalence (95% CI)	*P* Value
	HIV-Infected (n = 374)	HIV-Negative (n = 371)		
Total No. of individuals with colonization at any site	76 (20%)	41 (11%)	1.8 (1.3–2.7)	.002
Nasal colonization	45 (12%)	27 (7%)	1.7 (1.03–2.7)	.039
Any extranasal colonization	69 (18%)	39 (11%)	1.8 (1.2–2.6)	.005
Perirectal area	44 (12%)	24 (6%)	1.8 (1.11–2.99)	.018
Inguinal area	41 (11%)	24 (6%)	1.7 (1.02–2.8)	.04
Throat	30 (8%)	22 (6%)	…	.282
Axilla	28 (7%)	15 (4%)	…	.054
Wound	5 (1%)	2 (0.5%)	…	.278
Exclusive extranasal colonization	31 (8%)	14 (4%)	2.2 (1.2–4.1)	.015
Perirectal area	17 (5%)	5 (1%)	3.4 (1.24–9.14)	.017
Inguinal area	12 (3%)	7 (2%)	…	.264
Throat	9 (2%)	7 (2%)	…	.629
Axilla	5 (1%)	4 (1%)	…	.749
Wound[b]	2 (0.5%)	0	…	…

Data are No. (%) of patients unless otherwise specified.

Any extranasal colonization defined as presence of CA-MRSA extranasal colonization irrespective of nares culture results. Exclusive extranasal colonization defined as CA-MRSA colonization at extranasal sites and negative nares cultures for CA-MRSA. Chronic wounds (ie, present on admission) were also sampled (24 from HIV-infected patients and 11 from HIV-negative patients); we did not differentiate colonization from infection for wounds.

Abbreviations: CA-MRSA, community-associated methicillin-resistant *Staphylococcus aureus*; CI, confidence interval; HIV, human immunodeficiency virus.

[a] Twelve of the 745 enrolled individuals (1.6% of the enrolled sample) refused 1 or more of the surveillance swabs after enrollment began. The perirectal swab was the most frequently declined (10 individuals).

[b] Unable to obtain *P* value in model given small numbers.

Table 3. Univariate Analysis of Demographic and Exposure Factors Associated With a Higher Community-Associated Methicillin-Resistant *Staphylococcus aureus* Colonization Burden

Factor	Mean Colonization Burden by Factor		Relative Prevalence (95% CI)	*P* Value
	Present	Absent		
HIV-infected	0.103	0.061	1.68 (1.33–2.11)	<.001
African-American race	0.095	0.059	1.6 (1.25–2.06)	<.001
Hispanic ethnicity	0.047	0.091	0.52 (.37–.73)	<.001
Male	0.095	0.058	1.64 (1.27–2.12)	<.001
Temporary housing: current or in the past year	0.127	0.078	1.62 (1.14–2.3)	.007
Incarceration exposure: current detainee or release in past 3 mo	0.12	0.08	1.51 (.99–2.3)	.059
Illicit drug use: current or former	0.094	0.075	1.26 (1.01–1.58)	.045
Age, in decades[a]	…	…	0.87 (.79–.94)	.001
Diabetes	0.091	0.08	1.13 (.86–1.49)	.383
Men who have sex with men	0.108	0.093	1.16 (.88–1.54)	.3
Chronic hemodialysis	0.075	0.082	0.91 (.41–2.05)	.826
Residence in a nursing home or long-term care facility in the past year	0.12	0.081	1.49 (.9–2.47)	.118
Immunosuppression unrelated to HIV	0.069	0.084	0.82 (.57–1.18)	.29

Colonization burden: the number of colonized sites divided by the total number of sites sampled.

Abbreviations: CI, confidence interval; HIV, human immunodeficiency virus.

[a] Age represents a continuous variable.

Predictors of USA300 MRSA Strain Type

PFGE was performed on all 307 MRSA isolates from the 117 colonized individuals. Most isolates were USA300 (73.3%); remaining isolates were identified as USA500 (9.1%), USA100 (8.8%), USA800 (2.6%), USA700 (2.6%), USA1000 (1%), USA400 (1%), USA200 (0.3%), and nontypeable (1.3%).

Exclusive nasal colonization occurred infrequently among patients colonized with USA300 (6%) and non-USA300 (11%) strains (ie, most colonized patients also had extranasal colonization). Among colonized individuals, 38.5% had exclusive extranasal colonization.

Predictors of the USA300 strain type included HIV status (odds ratio [OR], 3.0; 95% CI, 1.32–6.79; $P = .007$), male sex (OR, 2.4; 95% CI, 1.02–5.65; $P = .043$), younger age (OR, 0.95; 95% CI, .91–.98; $P = .003$), and current or former illicit drug use (OR, 2.33; 95% CI, 1.01–5.35; $P = .044$). All but 1 of the colonized individuals with current or recent incarceration exposure carried the USA300 strain type.

DISCUSSION

The epidemiology and colonization dynamics of CA-MRSA strains are different from those of traditional MRSA strains [26], with CA-MRSA disproportionately affecting certain populations such as HIV-infected individuals [5, 20]. Results of our study corroborate and expand upon these findings. We found that compared to HIV-negative patients, HIV-infected patients were more likely to have CA-MRSA colonization in the anterior nares and at extranasal sites, and also to have exclusive extranasal colonization. HIV-infected patients also had a higher

Table 4. Predictors of Higher Community-Associated Methicillin-Resistant *Staphylococcus aureus* Colonization Burden on Multivariable Analysis

Predictor	Relative Prevalence (95% CI)	*P* Value
HIV-infected individuals		
Hispanic ethnicity	0.39 (.23–.66)	<.001
Incarceration exposure: current detainee or release in past 3 mo	1.66 (1.06–2.59)	.027
Male sex	1.53 (1.06–2.2)	.023
Age (in decades)	0.87 (.76–.99)	.033
HIV-negative individuals		
Temporary housing	2.14 (1.12–4.1)	.021

Temporary housing is defined as homeless or residing in a shelter or substance abuse center currently or in the prior year. Colonization burden is defined as the number of colonized sites divided by the total number of sites sampled. Initial variables included in the model were human immunodeficiency virus status, sex, race, ethnicity, incarceration exposure, temporary housing, illicit drug use, age, and residence in a nursing home or long-term care facility in the past year.

Abbreviations: CI, confidence interval; HIV, human immunodeficiency virus.

colonization burden than HIV-negative patients and were more likely to carry USA300. Consistent with prior work, degree of HIV immune suppression as measured by CD4 count was not related to colonization risk [5, 20].

Risk factors for higher CA-MRSA colonization burden among HIV-infected patients were current or recent incarceration, younger age, and male sex; for HIV-negative patients, the only risk factor for higher colonization burden was exposure to temporary housing. Among HIV-infected patients, Hispanic ethnicity was inversely associated with CA-MRSA colonization burden. Many of the risk factors for higher colonization burden were significant predictors for carriage of the USA300 strain type.

Our findings have implications for infection prevention and control. The high frequency of extranasal CA-MRSA colonization could confound inpatient infection control and prevention strategies developed for traditional MRSA strains; for example, 38.5% of colonized patients in this study had CA-MRSA colonization solely outside the nares and would have been misclassified as noncarriers by a hospital surveillance program that employed only nasal cultures. Furthermore, increased number of colonized sites has been associated with persistent colonization following decolonization attempts [11, 12].

Further evaluation is needed to determine whether higher colonization burden is associated with increased cross-transmission of strains among networks of individuals. Prior work has identified community geographic clusters and possible social networks with increased levels of CA-MRSA infection [5, 17, 27]. We speculate that our summarized measure—colonization burden—could be an indicator of increased infection risk and person-to-person spread within certain at-risk groups. Akin to colonization pressure as described in the hospital [28], colonization burden within a distinct community population may contribute to increased risk for acquisition of CA-MRSA strains.

Many of the factors identified in our study as predictive of higher colonization burden are similar to those previously described as associated with CA-MRSA infection [5, 17]. For example, prior work has found that HIV-infected patients have higher risk for CA-MRSA SSTIs in comparison to HIV-negative individuals and that they may be at increased risk for recurrent CA-MRSA disease [29, 30]. The greater prevalence of extranasal colonization and higher colonization burden found among HIV-infected patients in our study may help explain why the CA-MRSA epidemic has had such a major impact on this patient population [5].

Individuals with exposure to correctional facilities—another population significantly impacted by CA-MRSA infections [31, 32]—had increased risk for higher CA-MRSA colonization burden; this finding was most pronounced among HIV-infected patients. We included individuals who were incarcerated but who were HIV negative, and so, we were able to detect an enhanced risk for CA-MRSA in detainees who were HIV infected. Further evaluation is needed to understand the differential risk among these patient groups; it remains to be determined if social and living conditions upon release from correctional facilities could account for the significant differences observed between HIV-infected and HIV-negative former inmates.

Among HIV-negative individuals, temporary housing was associated with a higher CA-MRSA colonization burden. Other studies have noted increased CA-MRSA colonization among homeless individuals [33] and increased CA-MRSA SSTIs among individuals living in certain public housing complexes [17]. This type of alternative housing could reflect close contact with other potentially colonized or infected individuals. Crowding or another yet-to-be identified factor in this type of residence may play a role in increasing risk for CA-MRSA.

The negative association of CA-MRSA risk and Hispanic ethnicity that we noted has also been observed in a national survey of nasal colonization [34], in an assessment of CA-MRSA SSTIs [17], and in a colonization survey among HIV-infected outpatients [20]. Why Hispanic individuals have reduced risk for CA-MRSA colonization and infection is unknown; further evaluation is needed to determine whether genetic or other factors or differences in community-based exposures are responsible.

Our study has limitations. First, some data elements assessed may have been subject to reporting bias by the enrolled

patients. However, patient questionnaires were supplemented by medical records review in an attempt to minimize bias. Second, colonization was only assessed on the day of enrollment. Evaluation of durability (ie, colonization on multiple days) of extranasal colonization may be useful to fully understand CA-MRSA colonization dynamics. Finally, our study was conducted at the major safety-net hospital in Chicago and may not be representative of non–inner city populations. Nevertheless, several distinct populations with close person-to-person contact—military recruits [35] and amateur and professional athletes [36]—also have increased colonization and infection with CA-MRSA. Knowledge about extranasal colonization and colonization burden could potentially be applied to these populations.

In conclusion, we found that nasal and extranasal colonization rates and colonization burden were high among HIV-infected patients at the time of admission to a large, urban, public hospital. Our findings of population differences in CA-MRSA colonization burden mirror the disparity in infection rates among various populations (eg, HIV-infected vs HIV-negative [5] and African-American vs Hispanic patients [17]) observed in prior work. Further study of CA-MRSA transmission and infection dynamics in community settings is needed to evaluate these findings. Our results suggest that in certain patient groups, enhanced community and outpatient-based infection control strategies may be needed to prevent cross-transmission and infection due to MRSA.

Notes

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