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Prevalence of Nasal Colonization and Strain Concordance in Patients with Community-Associated *Staphylococcus aureus* Skin and Soft-Tissue Infections

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Abstract

OBJECTIVE—Determine the prevalence and relatedness of *Staphylococcus aureus* anterior nares colonization in individuals with community-associated staphylococcal skin and soft-tissue infection (SSTI).

DESIGN—Observational cohort.

SETTING—US Army soldiers undergoing infantry training.

PARTICIPANTS—Trainees who developed SSTI from May 2010 to January 2012.

METHODS—Participants underwent anterior nares culture at the time of presentation for purulent SSTI. We determined the prevalence of *S. aureus* nasal colonization and strain relatedness between colonizing and clinical isolates with pulsed-field gel electrophoresis (PFGE).

RESULTS—We enrolled 1,203 SSTI participants, of whom 508 had culture-confirmed *S. aureus* SSTI. Overall, 70% (357/508) were colonized with *S. aureus*. Phenotypically, concordant colonization was more common with methicillin-susceptible *S. aureus* (MSSA; 56%; 122/218) than methicillin-resistant *S. aureus* (MRSA) SSTI (41%; 118/290; $P < .01$). With PFGE, 48% (121 of 254) of clinical-colonizing pairs were indistinguishable, and concordant colonization was more common with MRSA (53%; 92/173) than MSSA SSTI (36%; 29/81; $P < .01$). Restricting analysis to concomitant MRSA-MRSA or MSSA-MSSA pairs, 92% (92/100) of MRSA SSTI were indistinguishable, and 40% (29/72) MSSA SSTI were indistinguishable ($P < .01$). All 92 MRSA pairs were USA300.

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CONCLUSIONS—On the phenotypic level, concordant anterior nares colonization with incident staphylococcal SSTI is more common in MSSA than MRSA; however, the opposite is observed when accounting for molecular typing, and MRSA SSTI displays greater concordance. USA300 was responsible for strain concordance with MRSA SSTI. Studies are needed to examine the roles of nasal and extra-nasal carriage, colonization preceding infection, and increased virulence in the pathogenesis of MRSA SSTI.

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Over the past decade, skin and soft-tissue infections in the United States, especially those caused by USA300 methicillin-resistant *Staphylococcus aureus* (MRSA) have emerged as a significant community health threat.[1] Although these infections are widespread within the general community, military trainees are at particularly high risk for MRSA SSTI.[2,3]

The epidemiology and pathogenesis of community-associated *S. aureus* SSTI is complex and incompletely understood, especially with regard to the role of nasal colonization. Colonization has often been considered the first step in the *S. aureus* pathogenesis and has often been the target of prevention strategies.[4–7] Although nasal colonization with *S. aureus*, including MRSA,[8,9] increases risk for subsequent infections, colonization may not be present, even in outbreak settings.[10,11] With *S. aureus* bloodstream infections, there is good genotypic concordance between nasal and infecting isolates;[12] however, this relationship has not been clearly demonstrated in adults with MRSA SSTI.[13] Determining the role of nasal colonization in *S. aureus* SSTI is essential for informing prevention strategies.

From May 2010 to January 2012, we conducted a prospective field-based, cluster-randomized trial at Fort Benning, Georgia, in which participants were assigned 1 of 3 hygiene- and education-based regimens aimed at preventing SSTI.[14] One group was offered once-weekly showering with chlorhexidine antiseptic body wash as part of the intervention. Overall, this investigation demonstrated no decrease in SSTI or MRSA SSTI. For the current study, we hypothesized that the contribution of nasal colonization with MRSA SSTI is distinct from that of methicillin-susceptible *S. aureus* (MSSA) SSTI. As a predesignated secondary objective, we determined the prevalence of concordant MRSA or MSSA nasal colonization in participants with *S. aureus* SSTI. Additionally, we determined the strain relatedness of isolates simultaneously colonizing and infecting individuals using pulsed-field gel electrophoresis (PFGE) and determined the presence of resistance and virulence determinants.

METHODS

Study participants and study design

In the setting of a prospective, field-based, cluster-randomized trial,[14] we conducted an observational cohort study in participants with SSTI to determine the prevalence of *S. aureus* nasal colonization, strain relatedness with clinical isolates, and potential risk factors. Study participants were US Army soldiers undergoing 14-week infantry training at Fort Benning, Georgia. This population was all male and in generally good physical condition. The Uniformed Services University infectious diseases institutional review board approved the investigation.

Enrollment and data collection

Infantry trainees who presented with SSTI at either the Troop Medical Clinic (TMC) or the hospital were eligible. After informed consent, participants completed a risk factor questionnaire and underwent anterior nares screening culture (BD BBL CultureSwabs; BD Diagnostic). Study personnel abstracted pertinent study-related SSTI data. Healthcare providers obtained cultures of purulent material when clinically indicated.

We defined an SSTI case patient as a trainee who had anterior nares culture performed within 3 days of initial presentation to the TMC or hospital with 1 of the following: cellulitis, abscess, folliculitis, impetigo, paronychia, or infected blister. We defined MRSA SSTI or MSSA SSTI as an SSTI with a MRSA- or MSSA-positive culture from the corresponding clinical site.

Microbiological and molecular analysis

Anterior nares specimens were placed in 5 mL of tryptic soy broth supplemented with 6.5% NaCl (BBL; BD Diagnostic) and were incubated for 18–24 hours at 37°C. After incubation, an aliquot of broth was plated onto mannitol salt agar. Mannitol fermenting colonies were isolated and plated onto trypticase soy agar with 5% sheep's blood and incubated overnight. Clinical and colonizing *S. aureus* isolates were identified according to standard protocols. All *S. aureus* isolates underwent identification and susceptibility testing using Microscan Walk-Away-96 (Dade Behring) according to Clinical Laboratory Standards Institute (CLSI) methods.[15] We performed PFGE using *Sma*I (Roche Molecular Biochemicals) as a restriction endonuclease on all available MRSA isolates.[16] PFGE findings were resolved and analyzed using BioNumerics (Applied Math) and grouped into pulsed-field types (PFTs) using Dice coefficients and 80% similarity as previously described.[17] We used a standard method to define paired isolates as indistinguishable.[18] MRSA isolates underwent polymerase chain reaction in duplicate to confirm *mec*A and to assign SCC*mec* type to assess for Panton-Valentine leukocidin (PVL),[19,20] and *arcA*.[21]

Statistical methods

Differences in proportions were evaluated by χ^2 or Fisher exact test as appropriate. Medians for age were compared using Wilcoxon rank-sum test. Two sample *t* test was used to analyze other continuous variables. All tests of significance were 2-tailed. A *P* value of less than or equal to .05 was considered significant. Statistical analyses were performed with SAS version 9.2 (SAS Institute).

RESULTS

Study population

The study population comprised 30,209 all male trainees, and during the investigation, 1,203 trainees (4%) developed SSTI and enrolled in the study. Among these participants, 508 (42%) had culture-confirmed *S. aureus* SSTI cases, including 290 (57%) with MRSA SSTI and 218 (43%) with MSSA SSTI. The demographic characteristics of participants with either MRSA or MSSA SSTI were similar (Table 1). With regard to clinical manifestation, participants with MRSA had more abscesses ($P < .01$), and participants with MSSA had

more purulent cellulitis ($P = .02$; Table 1). SSTI risk factors were similar between participants with MRSA and those with MSSA, but the latter were more likely to report a recent contact with a person with SSTI or MRSA infection ($P < .01$; Table 2).

Colonization and concordance

Nasal cultures were obtained on the same day or within 1 day of SSTI presentation in 92% (466 of 508) and within 3 days for only 3% (15 of 508). Only 3 of 508 participants had received antimicrobials before nasal culture was obtained. Overall, 357 of 508 participants (70%) with *S. aureus* SSTI had concomitant *S. aureus* nasal colonization (Table 3). With regard to concomitant colonization, there were significant differences between participants with MRSA and MSSA SSTI. Of the 290 participants with MRSA SSTI, 118 (41%) were simultaneously colonized with MRSA; and of the 218 participants with MSSA SSTI, 122 (56%) were simultaneously colonized with MSSA ($P < .01$). With regard to phenotypic discordant colonization, 32% (92 of 290) of participants with MRSA SSTI had simultaneous MSSA nasal colonization, and 12% (25 of 218) of participants with MSSA SSTI had simultaneous MRSA nasal colonization. The distributions of phenotypic concordance did not differ when stratified by those who were in study groups (data not shown).

Molecular analysis for strain concordance

Of the 508 participants with *S. aureus* SSTI, 254 participants had paired clinical and colonizing *S. aureus* isolates available for PFGE. These participants were similar to and representative of the entire SSTI group (data not shown). Overall, 48% (121 of 254) of *S. aureus* pairs were indistinguishable (Table 4). The predominant PFT of the 121 indistinguishable pairs was USA300 (100 of 121). Of those participants with MRSA SSTI, 53% (92 of 173) had indistinguishable pairs, and among those with MSSA SSTI, 36% (29 of 81) had indistinguishable pairs ($P < .01$). When restricting analysis to concomitant MRSA-MRSA or MSSA-MSSA pairs, 92% (92 of 100) of MRSA SSTI pairs were indistinguishable, and 40% (29 of 72) of MSSA SSTI pairs were indistinguishable ($P < .01$). All 92 MRSA indistinguishable pairs were USA300, and 28% (8 of 29) of MSSA pairs were USA300. Of the 8 participants with MRSA-MRSA pairs that were not indistinguishable, 5 were colonized with non-USA300 isolates (2 with USA800, 2 with USA100, 1 with no PFT) but had USA300 clinical isolates, and 3 were colonized with USA300 but had non-USA300 clinical isolates (2 with USA 800, 1 with no PFT). All USA300 MRSA strains were SCC*mec* type IV, *arcA* positive, and PVL positive. The distributions of strain relatedness did not differ when stratified by study group (data not shown).

DISCUSSION

In this investigation, we made several observations that help elucidate the role of nasal colonization in the pathogenesis of staphylococcal SSTI and may help inform future prevention strategies. On the phenotypic level (ie, with respect to methicillin susceptibility), concordant nasal colonization was more common with MSSA SSTI than with MRSA SSTI (56% vs 41%); however, the opposite was observed with molecular typing, and MRSA concordance was more common than MSSA concordance (53% vs 36%). Moreover, USA300 MRSA was the clone responsible for this concordance.

Among participants with MRSA SSTI, we found that only 118 (41%) of 290 had MRSA colonization. This colonization rate is higher than what has been observed by some investigators who have noted rates ranging from 7% to 34%.[10,13,22,23] On the other hand, our rate is lower than those reported among hospitalized patients with MRSA SSTI (55%)[24,25] In contrast with adults, pediatric patients appear to have a higher prevalence of concordant MRSA colonization, with Fritz et al[26] reporting a 76% colonization rate.

We noted that over half (53%) of participants had discordant nasal and clinical cultures based on susceptibility. We found that 12% of participants with MSSA SSTI had MRSA colonization and that 32% of participants with MRSA SSTI had MSSA colonization. This falls within rates reported by others, which have ranged from 52% to 67%.[10,23]

The role of *S. aureus* nasal colonization appears to vary considerably between patient populations and disease manifestations.[8,9,28] Among hospitalized patients with primarily invasive (eg, bloodstream) infections, strain relatedness between nasal and clinical isolates occurs in approximately 80% of cases.[12,25,29] For unknown reasons, this colonizing-infecting strain relatedness does not seem to bear out with SSTI. In our study, we observed that only 48% of patients (121 of 254) with *S. aureus* SSTI had a nasal strain indistinguishable from their clinical strain. In small community-based investigations of SSTI, the prevalence of strain relatedness has been reported to be as high as 100%,[23] whereas in a larger study by Miller et al,[13] only 12% of patients (41 of 350) with *S. aureus* SSTI had a concordant strain at any anatomic site. Among pediatric patients with SSTI, strain concordance appears to be generally higher than it is among adults, with Chen et al[10] reporting a rate of 59%. These observed differences may reflect the varying importance of colonization,[26,29] strain virulence,[1] or perhaps an indirect measure of fomites or person-to-person spread.[27]

Patients may be colonized with several strains of *S. aureus*,[13,30] and not all strains share the same pathogenic potential. When we limited analysis to MRSA-MRSA or MSSA-MSSA pairs, MRSA strain relatedness was significantly more common than MSSA strain relatedness (92% vs 40%). Moreover, all of these MRSA strains were USA300, which is the predominant clone in the United States.[1]

There are several limitations to our study. First, this investigation was a secondary objective of a trial which employed weekly chlorhexidine for SSTI prevention.[14] Nevertheless, the distributions of phenotypic concordance and strain relatedness did not differ when stratified by those who were in study groups randomized to receive chlorhexidine versus those who were not. Second, due to study-population constraints, we sampled only the anterior nares. Third, the observed results are based on laboratory culture of specimens that were subjected to broth enrichment; it is not known whether laboratory findings are an accurate representation of nasal ecology.

Our findings reinforce that nasal colonization does not appear to be a requirement for community-associated *S. aureus* SSTI and that other anatomic sites, person-to-person spread, and/or fomites are likely important factors. It also suggests that, although USA300

MRSA colonization with MRSA SSTI is relatively uncommon, when it does occur, it appears to be playing a role in pathogenesis.

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TABLE 1

Demographic and Clinical Characteristics of Patients with *Staphylococcus aureus* Skin and Soft-Tissue Infection (SSTI)

Variable	MRSA SSTI (*n* = 290)	MRSA SSTI (*n* = 218)	*P*
Race/ethnicity[a]	114	92	
White, non-Hispanic	91 (80)	77 (84)	.58
Hispanic	12 (11)	10 (11)	
Black, non-Hispanic	7 (6)	2 (2)	
Other, non-Hispanic	4 (4)	3 (3)	
Age, median (range), years	19 (17–37)	19 (17–33)	.19
Study group assignment			
Standard	74 (26)	43 (20)	.12
Enhanced standard	129 (45)	81 (37)	.10
Chlorhexidine	87 (30)	94 (43)	<.01
Clinical infection			
Abscess	141 (49)	65 (30)	<.01
Cellulitis	61 (21)	65 (30)	.02
More than 1 infection	74 (26)	44 (20)	.16
Folliculitis	9 (3)	7 (3)	.95
Infected blister	4 (1)	17 (8)	<.01
Impetigo	1 (0.3)	9 (4)	<.01
Paronychia	0 (0.0)	11 (5)	<.01
Site of infection			
Lower extremity	143 (49)	148 (68)	<.01
Upper extremity	93 (32)	47 (22)	<.01
More than 1 site	17 (6)	10 (5)	.53
Head	15 (5)	6 (3)	.18
Thorax	13 (5)	2 (1)	.03
Groin/inguinal/perineal	9 (3)	5 (2)	.58
Time from training start to *S. aureus* SSTI, mean (range), days	46 (3–101)	44 (1–96)	.43

NOTE. Data are no. (%) of participants, unless otherwise indicated. MRSA, methicillin-resistant *S. aureus*; MSSA, methicillin-susceptible *S. aureus*.

[a]Race and ethnicity data were not collected for all participants.

TABLE 2

Risk Factors for Skin and Soft-Tissue Infection (SSTI)

Variable	MRSA SSTI (*n* = 290)	MSSA SSTI (*n* = 218)	*P*
Within the past year			
Admitted to a hospital	14 (6)	6 (3)	.26
Worked at a hospital or nursing home	7 (3)	6 (3)	.76
Lived with someone admitted to/worked in a hospital/nursing home	51 (21)	35 (20)	.78
Known or suspected SSTI/MRSA infection	25 (10)	12 (7)	.22
Prescribed an antibiotic in the past 6 months	59 (24)	30 (17)	.07
Contact with a person with a skin infection/MRSA infection[a]	14 (6)	6 (3)	.26
Since arriving at Fort Benning			
Personal history of a skin infection/MRSA infection	35 (14)	21 (12)	.46
Had contact with a person with a skin infection/MRSA infection	111 (46)	58 (33)	<.01

NOTE. Data are no. (%) of participants, unless otherwise indicated. MRSA, methicillin-resistant *Staphylococcus aureus*; MSSA, methicillin-susceptible *S. aureus*.

[a] In the previous 3 months before arrival.

TABLE 3

Phenotypic Strain Concordance between Nasal and Wound Cultures

	Wound culture		
Nasal culture	**MSSA SSTI (*n* = 290)**	**MRSA SSTI (*n* = 218)**	**Total *Staphylococcus aureus* SSTI (*n* = 508)**
MRSA	118 (41)[a]	25 (12)	143 (28)
MSSA	92 (32)	122 (56)[a]	214 (42)
No *S. aureus*	80 (28)	71 (33)	151 (30)

NOTE. Data are no. (%) of participants, unless otherwise indicated. MRSA, methicillin-resistant *S. aureus*; MSSA, methicillin-susceptible *S. aureus*; SSTI, skin and soft-tissue infection.

[a]For difference between MRSA-MRSA and MSSA-MSSA concordance $P < .01$.

TABLE 4

Molecular Strain Concordance between Nasal and Wound Cultures

	Wound culture		
Nasal culture	**MSSA SSTI (*n* = 173)**	**MRSA SSTI (*n* = 81)**	**Total *Staphylococcus aureus* SSTI (*n* = 254)**
Indistinguishable	92 (53)[a]	29 (36)[a]	121 (48)
Not indistinguishable	81 (47)	52 (64)	133 (52)

NOTE. Data are no. (%) of participants with clinical infection and isolates available for molecular analysis, unless otherwise indicated. MRSA, methicillin-resistant *S. aureus*; MSSA, methicillin-susceptible *S. aureus*; SSTI, skin and soft-tissue infection.

[a] $P < .01$ for difference between MRSA and MSSA concordance.