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Recurrent Community-Acquired Methicillin-Resistant *Staphylococcus aureus* Infections in an HIV-Infected Person[▽]

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HIV-infected persons are at heightened risk for recurrent community-acquired methicillin-resistant *Staphylococcus aureus* (CA-MRSA) infections, but there are limited data regarding the molecular characterization of these events. We describe an HIV-infected patient with 24 soft tissue infections and multiple colonization events. Molecular genotyping from 33 nonduplicate isolates showed all strains were USA300, Panton-Valentine leukocidin (PVL) and arginine catabolic mobile element (ACME) positive, and genetically related.

CASE REPORT

A 27-year-old African-American male was diagnosed with HIV/AIDS in September 1997 after an episode of cryptococcal meningitis and a CD4 cell count of 16 cells/mm^3. His clinical course was complicated by antiretroviral medication nonadherence; the development of dermatologic conditions, including eosinophilic folliculitis and xerosis; esophageal candidiasis; HIV-associated wasting; and chronic renal dysfunction. The patient denied intravenous drug use or recent sexual activity, lived alone, and had no pets.

He was admitted in July 2005 for left lower extremity and neck abscesses which were culture positive for methicillin-resistant *Staphylococcus aureus* (MRSA); there was no history of MRSA, and there had been no hospital admissions in the prior 90 days. He was successfully treated with oral clindamycin and linezolid and received a 7-day course of nasal mupirocin for decolonization.

During the next 5 years (July 2005 to June 2010), the patient was diagnosed and treated for a total of 24 culture-proven MRSA skin and soft tissue infection (SSTI) events (Table 1). Seven (22%) of the SSTI events involved >1 body site; the total number of distinct culture-proven MRSA infections was 32. Regarding the site of infection, 38% occurred on the lower extremities, 22% on the upper extremities, 18% on the head/face, 16% on the trunk, and 6% on the buttocks/scrotum. All SSTI events were treated with antibiotics selected by the patient's provider (Table 1); of note, the patient was allergic to vancomycin and trimethoprim-sulfamethoxazole. In addition to antibiotics, incision and drainage procedures were performed for fluctuant abscesses. Seven MRSA SSTIs required hospital admission, totaling 50 days. These included a life-threatening MRSA necrotizing myositis of the lower extremity with septic shock, which required a 25-day hospital admission and the performance of multiple surgical debridements followed by three skin graft procedures.

Throughout the study period, screening for MRSA colonization was performed on 31 occasions, of which 19 (61%) were positive for MRSA at one or more body sites, with recovery of a total of 29 individual MRSA isolates. The frequency of MRSA colonization at each body site was examined: the nares were colonized on 67% (20/30) of swabs, groin on 21% (4/19), axilla on 16% (3/19), perirectal area on 14% (2/14), and throat on 0% (0/14). Seven of the colonization events occurred without an associated SSTI, and topical nasal mupirocin was prescribed in four instances. Twelve SSTI events had concurrent colonization; mupirocin was prescribed for seven of these events. In addition, the patient received 10 prescriptions of 3% hexachlorophene–4% chlorhexidine body washes and also had multiple courses of 10% bleach solutions, during this period in an attempt at decolonization.

Data regarding the HIV status of the patient were abstracted from the medical records; CD4 cell counts and plasma HIV RNA levels were performed as part of routine clinical practice. A review of laboratory results demonstrated an overall mean CD4 cell count of 30 cells/mm^3 (standard deviation [SD], 31) and a mean plasma HIV RNA level of 137,520 copies/ml (SD, 267,090) during this time period. Twenty-six (81%) of the MRSA SSTIs presented at a severely immunosuppressed state (CD4, <50 cells/mm^3), and all 32 SSTIs (100%) occurred at an HIV plasma RNA level of >1,000 copies/ml. Poor adherence to antiretroviral therapy was noted, with only brief periods of adherence, during this period (Table 1). Increasing numbers of MRSA events appeared temporarily associated with low CD4 cell counts and elevated HIV RNA levels (Fig. 1).

MRSA isolates were preserved in a random fashion at our hospital among all patients beginning in 2007. Thirty-three nonduplicate isolates (15 SSTIs and 18 colonization) from 15 different time points were available from this patient. MRSA

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TABLE 1. Recurrent MRSA events in an HIV-infected person

Event no.	MRSA events[a]				Molecular analysis[c]					Susceptibility[d]		HIV-associated factors		
	Date (mo/day/yr)	Presentation	Site	Antibiotic treatment[b]	USA strain type	SCC*mec* type	PVL	ACME	*msrA*	Clindamycin	Tetracycline[e]	CD4 count (cells/mm^3 [±2 wk])	HIV RNA level (copies/ml [± 2 wk])	Antiretroviral therapy[f]
1[g]	7/14/05	SSTI	Lower extremity	Clindamycin, linezolid, mupirocin (2%)							Sensitive			Regimen 1
		SSTI	Head/face	Clindamycin, linezolid, mupirocin (2%)							Sensitive			
2	8/16/05	Colonization	Nares									138	1,823	Regimen 1
		Colonization	Groin											
3	8/25/05	Colonization	Nares	Mupirocin (2%)										Regimen 1
4	9/13/05	SSTI	Lower extremity	Minocycline → linezolid							Intermediate			Regimen 1
5[g]	2/14/06	SSTI	Head/face	Daptomycin, minocycline → linezolid							Sensitive			
		SSTI	Trunk	Daptomycin, minocycline → linezolid							Intermediate			
		SSTI	Buttocks/scrotum	Daptomycin, minocycline → linezolid							Sensitive			
6	8/29/06	SSTI	Head/face	Linezolid							Sensitive			
		SSTI	Upper extremity	Mupirocin (2%)							Sensitive			
		Colonization	Nares	Mupirocin (2%)										
7[g]	1/23/07	SSTI	Upper extremity	Daptomycin → linezolid, rifampin							Resistant	0	>100,000	
		SSTI	Buttocks/scrotum	Daptomycin → linezolid, rifampin							Resistant			
8	4/10/07	SSTI	Lower extremity	Daptomycin → linezolid							Resistant			
		Colonization	Nares	Daptomycin → linezolid										
9	6/19/07	SSTI	Upper extremity	Linezolid → daptomycin	USA300	IV	+	+	−	Resistant	Resistant	2	>100,000	
		SSTI	Lower extremity	Linezolid → daptomycin	USA300	IV	+	+	−	Resistant	Resistant			
		Colonization	Nares	Linezolid → daptomycin	USA300	IV	+	+	−	Resistant	Resistant			
10	7/13/07	SSTI	Upper extremity	Linezolid → daptomycin	USA300	IV	+	+	−	Resistant	Resistant	2	>100,000	
		SSTI	Upper extremity	Linezolid → daptomycin	USA300	IV	+	+	−	Resistant	Resistant			
11	8/1/07	SSTI	Upper extremity	Daptomycin	USA300	IV	+	+	−	Resistant	Resistant			
		Colonization	Nares	Daptomycin										
12	8/15/07	SSTI	Upper extremity	Linezolid	USA300	IV	+	+	−	Resistant	Resistant			Regimen 2
		Colonization	Nares	Linezolid	USA300	IV	+	+	−	Resistant	Resistant			
		Colonization	Axilla	Linezolid	USA300	IV	+	+	−	Resistant	Resistant			
		Colonization	Perirectal	Linezolid	USA300	IV	+	+	−	Resistant	Resistant			
13	8/17/07	SSTI	Trunk	Daptomycin → linezolid							Resistant			Regimen 2
14	8/27/07	Colonization	Axilla	Mupirocin (2%)	USA300	IV	+	+	−	Resistant	Resistant	28	62,166	Regimen 3
		Colonization	Nares	Mupirocin (2%)	USA300	IV	+	+	−	Resistant	Resistant			
15	9/11/07	Colonization	Nares	Mupirocin (2%)	USA300	IV	+	+	−	Resistant	Resistant			Regimen 4
		Colonization	Groin	Mupirocin (2%)	USA300	IV	+	+	−	Resistant	Resistant			
		Colonization	Perirectal	Mupirocin (2%)	USA300	IV	+	+	−	Resistant	Resistant			

	9/18/07	Colonization	Nares	Mupirocin (2%)	USA300	IV	+	+	−	Resistant	Resistant			
16	9/26/07	SSTI	Head/face	Mupirocin (2%; to wound site and nares)	USA300	IV	+	+	−	Resistant	Resistant	20	62,166	
		Colonization	Nares	Mupirocin (2%; to wound site and nares)	USA300	IV	+	+	−	Resistant	Resistant			
		Colonization	Axilla	Mupirocin (2%; to wound site and nares)	USA300	IV	+	+	−	Resistant	Resistant			
		Colonization	Groin	Mupirocin (2%; to wound site and nares)	USA300	IV	+	+	−	Resistant	Resistant			
17	10/10/07	Colonization	Nares	Mupirocin (2%)	USA300	IV	+	+	−	Resistant	Resistant	6	34,483	
		Colonization	Groin	Mupirocin (2%)	USA300	IV	+	+	−	Resistant	Resistant			
18	11/30/07	SSTI	Lower extremity	Linezolid	USA300	IV	+	+	−	Resistant	Resistant	8	>100,000	
		Colonization	Nares	Mupirocin (2%)	USA300	IV	+	+	−	Resistant	Resistant			
19	1/8/08	Colonization	Nares											
20[g]	3/11/08	SSTI (including necrotizing myositis)	Lower extremity	Clindamycin, daptomycin							Resistant			
		Colonization	Nares	Clindamycin, daptomycin										
21[g]	6/19/09	SSTI	Head/face	Linezolid	USA300	IV	+	+	+	Sensitive	Sensitive		214,568	Regimen 5
		SSTI	Lower extremity	Mupirocin (2%)	USA300	IV	+	+	+	Sensitive	Sensitive			
		Colonization	Nares	Mupirocin (2%)	USA300	IV	+	+	+	Sensitive	Sensitive			
22	11/2/09	SSTI	Lower extremity	Linezolid							Sensitive	14	135,382	
23	11/17/09	SSTI	Lower extremity	Daptomycin → minocycline							Sensitive	7	1,328,761	
24[g]	1/7/10	SSTI	Trunk	Daptomycin → minocycline	USA300	IV	+	+	+	Sensitive	Sensitive			
		Colonization	Nares	Daptomycin → minocycline	USA300	IV	+	+	+	Sensitive	Sensitive			
25	2/10/10	SSTI	Trunk	Minocycline	USA300	IV	+	+	+	Sensitive	Sensitive	7	203,175	
26[g]	3/8/10	SSTI	Lower extremity	Daptomycin → linezolid → minocycline							Sensitive	4	515,260	
27	3/16/10	Colonization	Nares											
28	3/23/10	SSTI	Trunk	Linezolid → minocycline							Sensitive			
		Colonization	Nares	Mupirocin (2%)										
29	4/14/10	SSTI	Lower extremity	Linezolid → minocycline	USA300	IV	+	+	+	Sensitive	Sensitive			
30	5/4/10	SSTI	Lower extremity	Linezolid → minocycline	USA300	IV	+	+	+	Sensitive	Sensitive	14	798,625	
		Colonization	Nares	Mupirocin (2%)	USA300	IV	+	+	+	Sensitive	Sensitive			
31	5/18/10	SSTI	Head/face	Daptomycin → minocycline	USA300	IV	+	+	+	Sensitive	Sensitive			

[a] MRSA events were differentiated based on site and presentation date: SSTI and colonization of the same site occurring within 1 week were grouped as a single event.
[b] Arrows indicate changes in antibiotic treatment. In some instances, antibiotic courses may have overlapped. All listed antibiotics were systemic, except for mupirocin, which was topical.
[c] All available MRSA isolates were the same with regard to other molecular and susceptibility patterns.
[d] "Resistant" was defined as having a clindamycin MIC of ≥2 μg/ml and a tetracycline MIC of ≥8 μg/ml. "Sensitive" was defined as having a MIC of ≤0.5 μg/ml for both antibiotics.
[e] For SSTIs without isolates, tetracycline susceptibility was tested by disk diffusion as part of standard clinical practice.
[f] Regimen 1, atazanavir, didanosine, emtricitabine, ritonavir, and tenofovir; regimen 2, atazanavir, didanosine, fosamprenavir, lamivudine, and ritonavir; regimen 3, atazanavir, didanosine, lamivudine, and ritonavir; regimen 4, atazanavir, didanosine, and lamivudine; regimen 5, darunavir, etravirine, lamuvidine, raltegravir, and ritonavir.
[g] MRSA event requiring hospital admission.

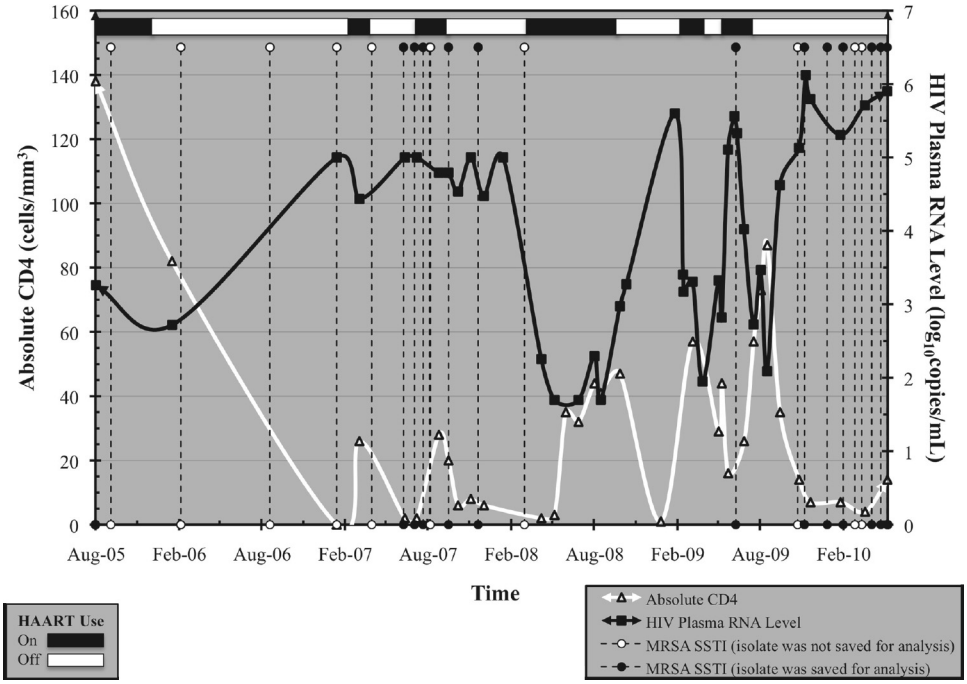

FIG. 1. MRSA infections in relation to HIV-specific factors, including CD4 count, HIV RNA level, and antiretroviral use. HAART, highly active antiretroviral therapy.

isolates were preserved at −70°C and tested at a single time point for molecular characterization and susceptibility to antimicrobial agents. All MRSA isolates underwent molecular analyses, which included pulsed-field gel electrophoresis (PFGE) following SmaI digestion. PFGE findings were resolved and analyzed with BioNumerics software (Applied Maths, Inc., Austin, TX) and grouped into pulsed-field types using established criteria (15, 26). A USA300 reference strain from the Centers for Disease Control and Prevention (CDC) was included for comparison.

PCR was performed to determine the staphylococcal cassette chromosome *mec* (SCC*mec*) type and to detect the presence of genes for Panton-Valentine leukocidin (PVL) and the arginine catabolic mobile element (ACME), as well as the accessory gene regulator (*agr*) locus. Antimicrobial susceptibility testing was performed with the BD Phoenix automated microbiology system (Becton, Dickinson and Co., Franklin Lakes, NJ). In cases of erythromycin resistance and clindamycin susceptibility, D-tests were performed to confirm clindamycin sensitivity. In addition, PCR was conducted to detect antimicrobial resistance genes, including *msrA*, *qacA*/*B*, *smr*, *aac6*, and *blaZ*.

On molecular testing, all isolates ($n = 33$) were confirmed to be MRSA, carrying the SCC*mec* type IV allele, and were USA300 strains by PFGE (Table 1). In addition, the presence of ACME and *agr* (group I), and the genes (*lukS*-PV and *lukF*-PV), which encode PVL, were detected in all available isolates. Figure 2 depicts the genetic relatedness of the isolates. Analyses show that all collected isolates were >85% genetically similar by PFGE. Within each of the four groups (Fig. 2), isolates were 100% similar; for example, all isolates from 2009 and 2010 were genetically identical by PFGE.

All available isolates were sensitive to vancomycin (MIC, ≤1 μg/ml), daptomycin (MIC, ≤1 μg/ml), quinupristin-dalfopristin (MIC, ≤1 μg/ml), linezolid (MIC, ≤2 μg/ml), rifampin (MIC, ≤1 μg/ml), trimethoprim-sulfamethoxazole (MIC, ≤0.5 μg/ml), and gentamicin (MIC, ≤1 μg/ml). Resistance was detected among all isolates to erythromycin (MIC, ≥4 μg/ml), and all were intermediate resistant to levofloxacin (MIC, ≥4 μg/ml).

The only variation in antimicrobial susceptibility noted among isolates over time was that all available MRSA isolates from June 2007 to June 2009 ($n = 23$) were resistant to clindamycin (MIC, ≥2 μg/ml) and tetracycline (MIC, ≥8 μg/ml), whereas all isolates after this date ($n = 10$) were sensitive for both antimicrobials, with MICs of ≤0.5 μg/ml. We also examined data regarding antibiotic susceptibilities obtained as part of clinical practice prior to the first preserved isolates (July 2005 to April 2007) and noted that the MRSA isolates were initially sensitive to tetracyclines, but that resistance developed over time, as shown in Table 1. Hence the patient initially had a tetracycline-sensitive strain, followed by a resistant strain and then subsequently was infected with a sensitive strain.

Genetic testing for antimicrobial resistance indicated that MRSA isolates from June 2007 to June 2009 were negative for the *msrA* gene. After this point, the MRSA isolates expressed the *msrA* gene and thus conferred susceptibility to the lincosamides, including clindamycin, as evidenced by the antimicrobial susceptibility data. Additional PCR results demonstrated a lack of resistance genes for *qacA*/*B* (cationic aseptic agents, including chlorhexidine), *smr* (quaternary ammonium compounds), and *aac6* (aminoglycosides), but the presence of *blaZ* (β-lactamase) among all isolates tested.

To our knowledge, this case represents the most MRSA

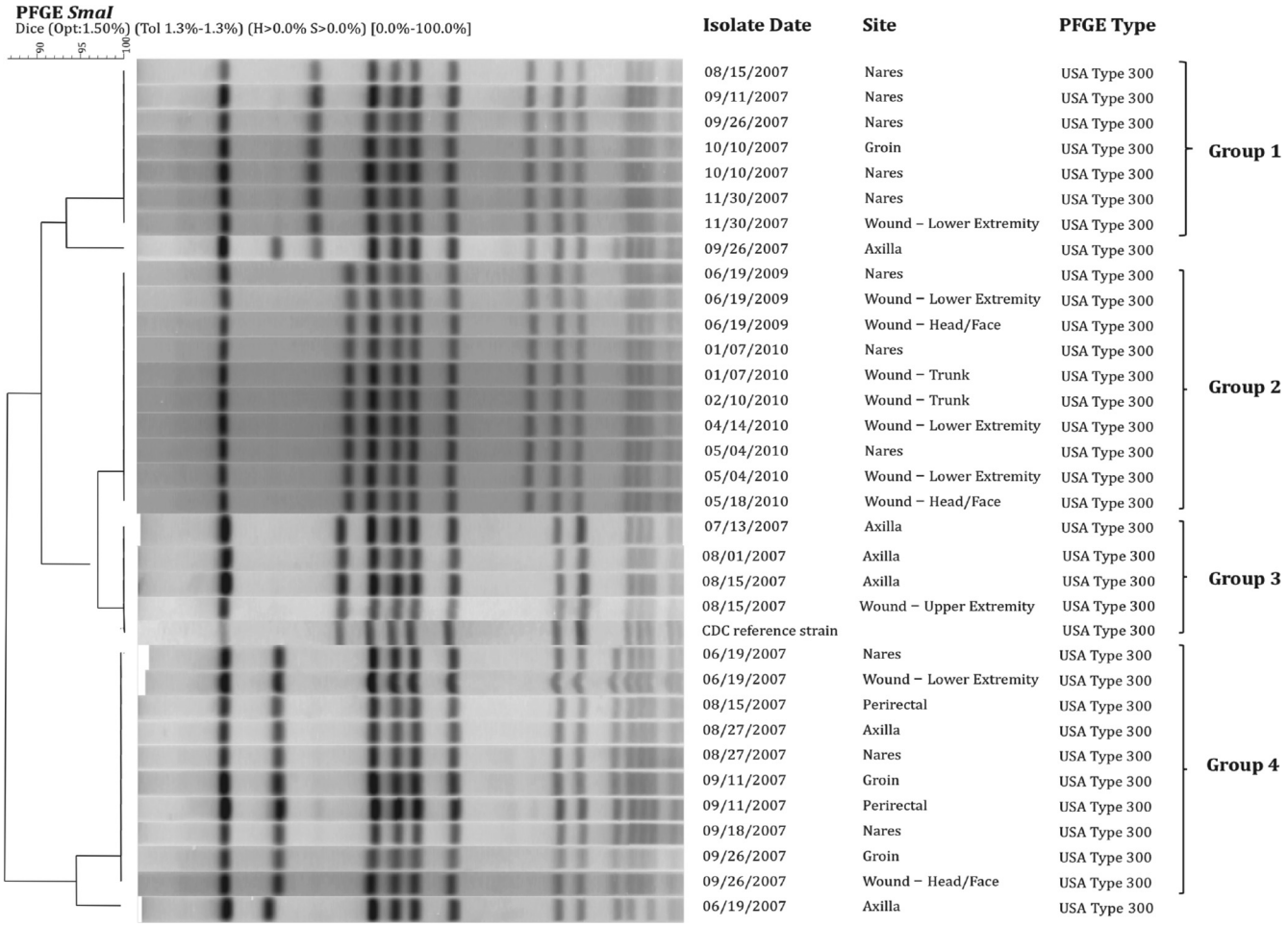

FIG. 2. Genetic relatedness of MRSA isolates from pulsed-field gel electrophoresis (PFGE). All isolates within a group were 100% genetically similar.

SSTIs reported in a single patient and exemplifies the potential risk of recurrent community-acquired (CA)-MRSA infections among HIV-infected persons, especially among those with severe immunosuppression. Despite multiple antibiotic courses, repeated incision and drainage procedures, and decolonization attempts, our patient experienced recurrent infections, suggesting that poor immune status may contribute to recurrent MRSA infections. In addition, our study adds novel data regarding the molecular characterization of recurrent MRSA events demonstrating that repeat infections are often due to genetically similar strains.

HIV-infected persons are known to have a higher rate of MRSA colonization and infection (3, 4, 5, 7, 14, 20, 23). In addition, this group may be at higher risk for recurrent disease (1, 6, 11, 24). Reports have shown that 41 to 71% of HIV patients with an initial MRSA SSTI develop a recurrent infection (1, 6, 11). Our HIV-infected patient developed 24 distinct CA-MRSA events in discrete locations and after resolution of the previous infection. In addition to SSTIs, he developed a life-threatening MRSA infection with necrotizing myositis and septic shock. As such, our case adds to the existing literature of recurrent MRSA infections and exemplifies the challenge regarding the prevention and management of these cases among HIV-infected persons.

The reason for recurrent MRSA infections in our patient is unknown, but may be related to severe immunosuppression, poor antiretroviral adherence, absence of trimethoprim-sulfamethoxazole prophylaxis (due to allergy), and dermatologic conditions (1, 14, 21). Prior studies have implicated illicit drug use and high-risk sexual behaviors as potential risk factors for MRSA infections (5, 7, 13, 14), but these were not noted in our patient. Poor virologic control may also be associated with recurrent CA-MRSA infections (6); it has been suggested that the direct antiproliferative effect high levels of HIV have on CD4 cells may influence immunity to *S. aureus* (14, 16). We noted a correlation in our patient between low CD4 counts and high plasma HIV RNA levels with increasing MRSA SSTI events. Future evaluations of the exact immune disturbances, including the role of T-cell subsets such as Th17 cells, and the risk of MRSA SSTIs are needed.

Colonization may play an important role in repeated MRSA infections. Prior studies have shown that carriage of MRSA

increases the risk for subsequent SSTIs (9, 12, 17, 19, 22, 27). Our patient was repeatedly colonized with similar strains that caused the infections. Despite multiple attempts at decolonization with topical antibacterial preparations, infections continued to occur. It is possible that the patient's existing skin conditions and poor immunologic status negatively impacted our decolonization efforts.

The role of decolonization strategies in preventing CA-MRSA infections among HIV-infected persons remains unknown. A recent study among HIV-positive drug users demonstrated no benefit from nares decolonization in reducing the number of subsequent infections (10). However, studies of the role of colonization as reservoirs for infection (including novel sites such as the perirectal area) (25) and the efficacy of their accompanying decolonization procedures are needed, as well as data on the natural displacement of MRSA strains over time.

We performed robust molecular analyses of the available MRSA isolates in our case. All isolates were consistent with community-acquired (CA) strains—USA300 carrying SCC*mec* IV, PVL, and ACME genes. The propensity for recurrent colonization and pyogenic SSTIs in our case were likely associated with these bacterial factors (8, 9). For example, the faster doubling time of SCC*mec* IV strains may outcompete other colonizing and infecting strains, and PVL and ACME may be markers for pyogenic/necrotizing skin infections (8, 18).

The MRSA strains in our case were highly related over time, with few differences detected by PFGE, suggesting continued carriage of similar strains or repeated acquisition from the same source. Though the molecular characteristics of the isolates were relatively conserved throughout study duration, there was an alteration in the strain occurring in June 2009. After this juncture, isolates expressed the *msrA* gene and thus conferred susceptibility to lincosamides, including clindamycin. The appearance of this novel strain is remarkable in that despite extensive exposure to antistaphylococcal antibiotics, this patient was ultimately colonized and infected by a less resistant strain. We speculate that our patient may have subsequently acquired a different, less-resistant USA300 strain which potentially filled the niche when antibiotic pressure was absent. Alternatively, since genes conferring greater antibiotic resistance are often metabolically costly (2), with the absence of pressure from the specific class of antibiotics, resistance may have been reversible.

Our investigation has limitations. We present a single case study, and therefore our findings require confirmation among larger numbers of patients. Second, we did not have isolates available for all MRSA events especially during the latter part of the study period; nonetheless, our study does provide insightful information on the molecular characteristics of recurrent MRSA infections. Third, since the patient was repeatedly infected with the most common type of CA-MRSA (the USA300 strain with SCC*mec* IV and PVL), it is possible that he reacquired similar strains over time (including after successful decolonization attempts) rather than continued to be infected with the same strain. Finally, although our observational study suggests a relationship between uncontrolled HIV and increased propensity of MRSA infections, causality could not be determined. As such, whether the unfavorable HIV parameters played a causative role in the development of the MRSA infections, or whether the MRSA infections led to reductions in CD4 counts and elevations in HIV RNA levels, requires further investigation.

In summary, we report the largest number of recurrent CA-MRSA infections in a single patient in the literature. The convergence of host (e.g., low CD4 counts) and bacterial factors (USA300 SCC*mec* IV strain) may have resulted in repeated MRSA infections. Practitioners should be aware of the risks for MRSA recurrences among HIV-infected persons and recognize that despite appropriate treatment and decolonization attempts, infections may recur. Further studies are needed regarding the ideal approaches to reduce recurrent MRSA infections. In the meantime, optimization of HIV control and reduction of potential risk factors are advocated.

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We certify that all individuals who qualify as authors have been listed; each has participated in the conception and design of this work, the writing of the document, and the approval of the submission of this version; that the document represents valid work; that if we used information derived from another source, we obtained all necessary approvals to use it and made appropriate acknowledgements in the document; and that each takes public responsibility for it. Nothing in the presentation implies any Federal/DOD/DON endorsement.

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