Community-Onset Methicillin-Resistant *Staphylococcus aureus* Associated with Antibiotic Use and the Cytotoxin Panton-Valentine Leukocidin during a Furunculosis Outbreak in Rural Alaska

Henry C. Baggett,[1,3,a] Thomas W. Hennessy,[1] Karen Rudolph,[1] Dana Bruden,[1] Alisa Reasonover,[1] Alan Parkinson,[1] Rachel Sparks,[1] Rodney M. Donlan,[4] Patricia Martinez,[2] Kanokporn Mongkolrattanothai,[5] and Jay C. Butler[1]

[1]Arctic Investigations Program, National Center for Infectious Diseases, Centers for Disease Control and Prevention, Anchorage, and [2]Yukon-Kuskokwim Health Corporation, Bethel, Alaska; [3]Epidemic Intelligence Service, Division of Applied Public Health Training, Epidemiology Program Office, and [4]Division of Healthcare Quality Promotion, National Center for Infectious Diseases, Centers for Disease Control and Prevention, Atlanta, Georgia; [5]Section of Pediatric Infectious Diseases, University of Chicago, Illinois

Background. Community-onset methicillin-resistant *Staphylococcus aureus* (CO-MRSA) reports are increasing, and infections often involve soft tissue. During a CO-MRSA skin infection outbreak in Alaska, we assessed risk factors for disease and whether a virulence factor, Panton-Valentine leukocidin (PVL), could account for the high rates of MRSA skin infection in this region.

Methods. We conducted *S. aureus* surveillance in the outbreak region and a case-control study in 1 community, comparing 34 case patients with MRSA skin infection with 94 control subjects. An assessment of traditional saunas was performed. *S. aureus* isolates from regional surveillance were screened for PVL genes by use of polymerase chain reaction, and isolate relatedness was determined by use of pulsed-field gel electrophoresis (PFGE).

Results. Case patients received more antibiotic courses during the 12 months before the outbreak than did control subjects (median, 4 vs. 2 courses; $P = .01$) and were more likely to use MRSA-colonized saunas than were control subjects (44% vs. 13%; age-adjusted odds ratio, 4.6; 95% confidence interval, 1.7–12). The PVL genes were present in 110 (97%) of 113 MRSA isolates, compared with 0 of 81 methicillin-susceptible *S. aureus* isolates ($P < .001$). The majority of MRSA isolates were closely related by PFGE.

Conclusion. Selective antibiotic pressure for drug-resistant strains carrying PVL may have led to the emergence and spread of CO-MRSA in rural Alaska.

Once considered to be nearly an exclusive nosocomial pathogen, methicillin-resistant *Staphylococcus aureus* (MRSA) has recently become an established cause of community infections [1–15]. In parts of the United States, MRSA accounts for the majority of community-onset (CO) *S. aureus* infections [11, 14, 15]. CO-MRSA is most often associated with skin and soft tissue infections rather than with invasive disease [1, 2, 8, 14, 16, 17]. CO-MRSA is commonly resistant only to β-lactam antibiotics and typically demonstrates susceptibility to trimethoprim-sulfamethoxazole, fluoroquinolones, and aminoglycosides [1, 4, 7, 11–17]. Previously published studies indicate that isolates causing CO-MRSA infections are of a different genetic background, compared with hospital-acquired isolates from the same region [17–19].

Risk factors for nosocomial MRSA are well established and include prolonged antibiotic exposure, dialysis, and admission to an intensive care unit [20, 21],

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[a] Present affiliation: Division of Applied Public Health Training, Epidemiology Program Office, Centers for Disease Control and Prevention, Atlanta, Georgia.

Reprints or correspondence: Dr. Thomas W. Hennessy, Arctic Investigations Program, Centers for Disease Control and Prevention, 4055 Tudor Centre Dr., Anchorage, AK 99508 (thennessy@cdc.gov).

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An Outbreak of Community Resistant *Staphylococcus aureus* Skin Infections in Southwestern Alaska

Henry C. Baggett, MD; Thomas W. Hennessy, MD, MPH; Richard Leman, MD; Cindy Hamlin, RN; Dana Bruden, MS; Alisa Reasonover, BS; Patricia Martinez, MD; Jay C. Butler, MD

ABSTRACT

OBJECTIVE: We investigated a large outbreak of community-onset methicillin-resistant *Staphylococcus aureus* (MRSA) infections in southwestern Alaska to determine the extent of these infections and whether MRSA isolates were likely community acquired.

DESIGN: Retrospective cohort study.

SETTING: Rural southwestern Alaska.

PATIENTS: All patients with a history of culture-confirmed *S. aureus* infection from March 1, 1999, through August 10, 2000.

RESULTS: More than 80% of culture-confirmed *S. aureus* infections were methicillin resistant, and 84% of MRSA infections involved skin or soft tissue; invasive disease was rare. Most (77%) of the patients with MRSA skin infections had community-acquired MRSA (no hospitalization, surgery, dialysis, indwelling line or catheter, or admission to a long-term–care facility in the 12 months before infection). Patients with MRSA skin infections were more likely to have received a prescription for an antimicrobial agent in the 180 days before infection than were patients with methicillin-susceptible *S. aureus* skin infections.

CONCLUSIONS: Our findings indicate that the epidemiology of MRSA in rural southwestern Alaska has changed and suggest that the emergence of community-onset MRSA in this region was not related to spread of a hospital organism. Treatment guidelines were developed recommending that beta-lactam antimicrobial agents not be used as a first-line therapy for suspected *S. aureus* infections (*Infect Control Hosp Epidemiol* 2003;24:397-402).

Recent reports of community-onset methicillin-resistant *Staphylococcus aureus* (MRSA) infections suggest that MRSA is now acquired in both healthcare and community settings.[1-12] Community-onset MRSA infections were reported in the United States as early as the 1980s, but many of these episodes were associated with typical risk factors for MRSA acquisition such as hospitalization and intravenous drug use.[13,14] Although frequently reported as community-acquired MRSA because infections were diagnosed in the outpatient setting, these episodes would be more accurately described as community-onset MRSA infections caused by hospital-acquired MRSA. Subsequent accounts of community-onset MRSA identified illness in individuals who did not have known risk factors for MRSA acquisition.[1,2,7,9,11,12] These episodes, first reported in the mid-1990s, likely represented MRSA that was truly community acquired. This change in the epidemiology of MRSA has important clinical implications. Current first-line antimicrobial agents for outpatient *S. aureus* infections, including all beta-lactam antibiotics, are ineffective for MRSA, and treatment failures have been described with fatal outcomes.[4]

The emergence of community-acquired MRSA may portend a time when oxacillin resistance must be presumed in outpatient *S. aureus* infections. Fortunately, most community-acquired MRSA reports to date have occurred in relatively small clusters and have not warranted a change in empiric antimicrobial recommendations. However, a recent study performed in a midwestern Native American community found that MRSA caused more than half of all community *S. aureus* infections,[11] suggesting that treatment recommendations may need to be changed in some areas.

In August 2000, healthcare providers in southwestern Alaska reported an increase in MRSA skin infections among Alaska Natives, many of whom had no previous hospital exposure. In this article, we describe the emer-

Drs. Baggett, Hennessy, and Butler and Ms. Hamlin, Ms. Bruden, and Ms. Reasonover are from the Arctic Investigations Program, National Center for Infectious Diseases, Centers for Disease Control and Prevention, Anchorage, Alaska. Drs. Baggett and Leman are from the Epidemic Intelligence Service, Division of Applied Public Health Training, Epidemiology Program Office, Centers for Disease Control and Prevention, Atlanta, Georgia. Dr. Leman is also from the Indian Health Service, Albuquerque, New Mexico. Dr. Martinez is from the Yukon-Kuskokwim Health Corporation, Bethel, Alaska.

Address reprint requests to Henry C. Baggett, MD, CDC/Arctic Investigations Program, 4055 Tudor Centre Drive, Anchorage, AK 99508.

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