Characteristics of Staphylococcus aureus Infections, Chicago Pediatric Hospital

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Invasive and skin community-associated (CA)–methicillin-resistant *Staphylococcus aureus* isolates from children were matched with invasive CA–methicillin-sensitive *S. aureus* strains during 2000–2004. Isolates were analyzed for presence of Panton-Valentine leukocidin. A USA400 lineage clone (n = 6) and the predominant USA300 lineage clone emerged.

Community-associated methicillin-resistant *Staphylococcus aureus* (CA-MRSA) infections have been increasing in children since the 1990s. Panton-Valentine leukocidin (PVL) has been associated with CA-MRSA increasing in children since the 1990s. Panton-Valentine USA400 lineage clone (n = 6) and the predominant lysed for presence of Panton-Valentine leukocidin. A *S. aureus* isolates from children were matched with invasive CA–methicillin-sensitive *S. aureus* strains during 2000–2004. Isolates were analyzed for presence of Panton-Valentine leukocidin. A USA400 lineage clone (n = 6) and the predominant USA300 lineage clone emerged.

Categorical variables were analyzed by χ² analysis. Variables with non-normal distribution were analyzed by Mann-Whitney U test; 2-tailed p value ≤ 0.05 was statistically significant (SPSS Inc., version 11.0, Chicago, IL, USA).

From 166 MRSA patient strains noted in the microbiology records, 21 patients with invasive CA-MRSA infection were identified. Three patient isolates were unavailable and were excluded from analysis. Patients with invasive CA-MRSA strains were case-matched with patients with CA-MRSA SSTI (16/18 were matched by age). During the study period, 31 cases of invasive CA-MSSA disease were identified and 10 cases were able to be retrieved and matched with cases of invasive CA-MRSA (9/10 case-patients were matched by age).

Groups with invasive CA-MRSA and groups with invasive CA-MSSA did not differ significantly regarding sex, initial leukocyte count, duration of fever, or length of hospital stay. Pediatric patients with invasive CA-MRSA infection were more likely to be African American (p = 0.01) and were febrile significantly longer than patients with invasive CA-MSSA (p = 0.03). One of the patients with invasive CA-MRSA died (Table 1).

Among the 18 patients with invasive CA-MRSA, 17 patients required surgical drainage, and 1 patient was given extracorporeal membranous oxygenation. Among the 10 patients with invasive CA-MSSA disease, all required surgical intervention, but none died. Of 18

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patients with SSTI CA-MRSA disease, 12 were African American, and 2 were Hispanic. The average hospital stay for this group was 2.3 days. Seven children required no hospitalization or hospitalization <24 hours.

A representative PFGE result is depicted in the Figure. Two predominant clones emerged among the local and invasive CA-MRSA isolates. The clone A (n = 6) was identified to be of the USA400 lineage, and the B clone (n = 30) was identified to be of the USA300 lineage. No predominant clones emerged from the invasive MSSA isolates; 6 unique clones were identified from 10 isolates (data not shown). No isolates were of the USA300 or 400 lineage. The clinical manifestations of invasive disease in patients with CA-MRSA disease from clone A were pneumonia with empyema, osteomyelitis, bacteremia, and septic arthritis. The clinical manifestations of invasive disease in patients with clone B infection included sepsis, toxic-shock syndrome, osteomyelitis, fasciitis, bacteremia, pyomyositis, pneumonia with empyema, deep visceral abscesses, and perirectal abscess with prostatitis; 1 patient in this group died. Clone A was found in a wide geographic distribution around metropolitan Chicago. In contrast, clone B was located with more frequency within the city limits of Chicago.

Antimicrobial susceptibility patterns among CA-MRSA strains are detailed in Table 2. Two CA-MRSA isolates that were positive by 3-D test belonged to the A0 clone. None of the MRSA isolates that caused SSTI was D-test positive. The PVL gene was found in all of the CA-MRSA isolates that caused invasive and SSTI disease but in only 1 of 10 of the invasive CA-MSSA isolates (p<0.001).

Conclusions

PVL genes can be transmitted by means of bacteriophages, which allows them to be transmitted from 1 organism to another (9). When injected intradermally in rabbits, PVL induces necrotic skin lesions (10), and PVL has been described in S. aureus isolates from patients with necrotizing pneumonia, skin infections, and musculoskeletal infections. These outbreaks have been widespread (1,2,8,11–13). We found a wide range of severity of infection caused by clonally related CA-MRSA, PVL-positive isolates within our community, from superficial skin abscesses to fatal disease.
Results of PFGE correlate well with results of other molecular typing methods, such as multilocus sequence typing (MLST), which characterizes *S. aureus* species by using sequences of 7 conserved housekeeping genes (7). In accordance with other published studies in the United States, the USA300 strain was the most frequently isolated among CA-MRSA disease (12) in contrast to the invasive CA-MSSA disease, which had no predominant clonality. We also detected isolates of the USA400 lineage that harbored PVL genes. This lineage was previously described as a cause of severe and fatal CA-MRSA disease in children in the Midwest (14). Previous reports from University of Chicago have described a cluster of 4 cases in which USA400 isolates caused empyema and sepsis syndrome with some features of toxic-shock syndrome (4); 3 children died with necrotizing pneumonia and Waterhouse-Friderichsen syndrome due to a PVL-positive, MLST-identified type 1 strain (15). In contrast, our patient with fatal toxic-shock syndrome did not have any primary pulmonary pathology and had disease caused by a USA300 lineage strain.

Limitations to our study include its retrospective nature and the limited numbers of patients. The geographic distribution of CA-MRSA isolates within the city likely reflects the geographic distribution of our patient population. Future prospective studies may further elucidate possible epidemiologic risk factors associated with acquiring CA-MRSA invasive infection.

Dr Jaggi is a pediatric infectious disease fellow at Children’s Memorial Hospital (Northwestern). Her major research interests have included group A streptococcal infections and pediatric community-associated MRSA infections.

### References


### Table 2. Susceptibility data for CA-MRSA isolates from children*

<table>
<thead>
<tr>
<th>Susceptibility to antimicrobial agent†</th>
<th>CA-MRSA isolates causing invasive disease, n (%)</th>
<th>CA-MRSA isolates causing local skin/ soft tissue infections, n (%)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resistant to erythromycin</td>
<td>16/18 (88.9)</td>
<td>11/18 (61)</td>
<td>NS</td>
</tr>
<tr>
<td>Apparently susceptible to clindamycin</td>
<td>18/18 (100)</td>
<td>18/18 (100)</td>
<td></td>
</tr>
<tr>
<td>Inducible clindamycin resistance</td>
<td>3/16 (19)</td>
<td>0/10 (0)</td>
<td>NS</td>
</tr>
<tr>
<td>Resistant to ciprofloxacin</td>
<td>2/18 (11)</td>
<td>1/18 (6)</td>
<td>NS</td>
</tr>
<tr>
<td>Resistant to levofloxacin</td>
<td>1/18 (6)</td>
<td>1/18 (6)</td>
<td>NS</td>
</tr>
<tr>
<td>Resistant to tetracycline</td>
<td>1/18 (5)</td>
<td>2/18 (11)</td>
<td>NS</td>
</tr>
</tbody>
</table>

*CA-MRSA, community-associated methicillin-resistant *Staphylococcus aureus*; NS, not significant.

†In addition to the antimicrobial agents listed, all isolates were susceptible to vancomycin, linezolid, and rifampin.

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