

RESEARCH ARTICLE

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Higher incidence of perineal community acquired MRSA infections among toddlers

Alexis C McCullough¹, Melissa Seifried¹, Xiaochen Zhao², Jeffrey Haase³, William J Kabat⁴, Ram Yogev⁴, Robert M Blumenthal^{5,6} and Deepa Mukundan^{2,5*}

Abstract

Background: A six-fold increase in pediatric MRSA infections, prompted us to examine the clinical profile of children with MRSA infections seen at Mercy Children's Hospital, Toledo, Ohio and to characterize the responsible strains.

Methods: Records were reviewed of pediatric patients who cultured positive for MRSA from June 1 to December 31, 2007. Strain typing by pulsed field gel electrophoresis (PFT) and DiversiLab, *SCCmec* typing, and PCR-based *lukSF-PV* gene (encodes Panton-Valentine leukocidin), arginine catabolic mobile element (ACME) and *cap5* gene detection was performed.

Results: Chart review of 63 patients with MRSA infections revealed that 58(92%) were community acquired MRSA (CAMRSA). All CAMRSA were skin and soft tissue infections (SSTI). Twenty five (43%) patients were aged < 3 yrs, 19 (33%) aged 4-12 and 14(24%) aged 13-18. Nineteen (76%) of those aged < 3 yrs had higher incidence of perineal infections compared to only 2(11%) of the 4-12 yrs and none of the 13-18 yrs of age. Infections in the extremities were more common in the older youth compared to the youngest children. Overall, there was a significant association between site of the infection and age group (Fisher's Exact p-value < 0.001). All CAMRSA were USA300 PFT, clindamycin susceptible, *SCCmec* type IVa and *lukSF-PV* gene positive. Nearly all contained ACME and about 80% were *cap5* positive. Of the 58 USA300 strains by PFT, 55(95%) were also identified as USA300 via the automated repetitive sequence-based PCR method from DiversiLab.

Conclusions: CAMRSA SSTI of the perineum was significantly more common among toddlers and that of the extremities in older children. The infecting strains were all USA300 PFT. Further studies are needed to identify the unique virulence and colonization characteristics of USA300 strains in these infections.

Background

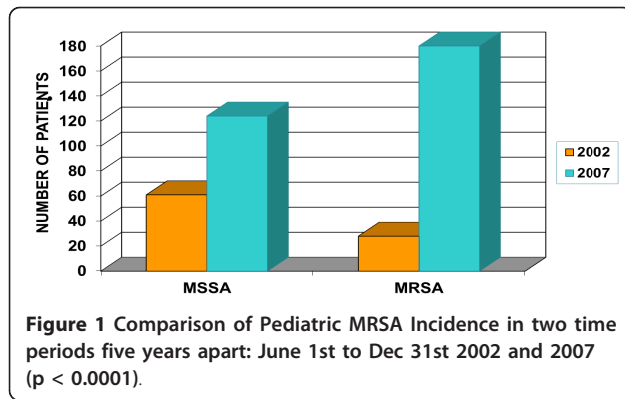
Staphylococcus aureus (*S. aureus*) is a common human commensal organism and a clinically important invasive pathogen. Methicillin resistant *S. aureus* (MRSA) remains one of the most prevalent pathogens isolated from hospital patients. However, MRSA infections are increasingly arising outside of healthcare settings among individuals in the community with no established risk factors. Furthermore, the incidence of invasive community acquired MRSA (CAMRSA) disease in previously-healthy children has been increasing [1-3].

CAMRSA strains as defined by the Centers for Disease Control and Prevention (CDC) clinical criteria [4] have some general characteristics that differentiate them from healthcare associated MRSA (HAMRSA) strains, including the presence of the Staphylococcal chromosomal cassette - *SCCmec* type IVa that confers methicillin resistance, *lukSF-PV* genes that codes for Panton-Valentine leukocidin (PVL), arginine catabolic mobile element (ACME) element that contributes to skin colonization [5], antibiotic resistance patterns [3], and pulsed field types (PFT) [6]. Several reports now show the migration of these CAMRSA strains into the hospital setting [7-9]. A six-fold increase in the number of MRSA infections among children between 2002 and 2007 ($p < 0.0001$) (Figure 1) prompted us to examine both the clinical profile of the patients, and the molecular profile of the

* Correspondence: Deepa.Mukundan@utoledo.edu

²Department of Pediatrics, Division of Infectious Diseases, The University of Toledo College of Medicine and Mercy Children's Hospital, 2222 Cherry Street, Toledo-OH, 43608, USA

Full list of author information is available at the end of the article



infecting strains at Mercy Children's Hospital Toledo, Ohio. We also compared two strain typing methods - pulsed field typing (PFT) and the automated rapid typing method using repetitive sequence-based PCR, the DiversiLab system [10].

Methods

MRSA isolate collection and patient medical record review

This is a retrospective, descriptive, single-cohort study conducted at Mercy Children's Hospital, Toledo, Ohio and was approved by the institutional review board. Pediatric patients (< 18 yrs of age), who were culture positive for MRSA between June 1 to December 31, 2007, were identified by the clinical microbiology laboratory (Mercy Integrated Laboratories, Toledo, Ohio). This facility also provided us with a sample of the clinical isolate that had been frozen at -80°C in a solution of 50% brain-heart infusion and 50% glycerol for later analysis.

Medical charts of these patients were reviewed for social and demographic information including age, gender, race, socio-economic status based on insurance, pre-existing history of skin and soft tissue infection (SSTI); respiratory, cardiovascular, gastrointestinal, and nervous system diseases, type of care (outpatient *vs.* emergency room *vs.* inpatient care) and site of infection. Antimicrobial susceptibilities were determined based on Clinical and laboratory Standard Institute (CLSI) guidelines at the clinical laboratory using the Vitek system for oxacillin, erythromycin, clindamycin, vancomycin, ciprofloxacin, tetracycline, trimethoprim/sulfamethoxazole, rifampin and linezolid. All erythromycin-resistant strains were tested for inducible clindamycin resistance using the D-test [11] before final susceptibilities were reported.

Strain Typing

Pulsed Field typing (PFT): Strain typing was done by PFT as described by Chang *et al* [12], using *Sma*I as the restriction enzyme, at the Children's Memorial Hospital,

Chicago, Illinois. The relatedness of isolates was based on visual comparison of band patterns by the use of criteria described by Tenover *et al* [13].

DiversiLab typing: Strain typing was also performed using the DiversiLab System at Mercy Integrated Laboratories. This is an automated method using repetitive sequence-based PCR that targets multiple noncoding repetitive sequences in the genomic DNA. Band patterns were subsequently analyzed using the web-based DiversiLab software [10].

DNA Extraction and PCR

Clinical isolates were grown on 5% blood agar plates overnight at 37°C . DNA was extracted from the isolates using the Wizard[®] Genomic DNA Purification Kit. PCR amplification was then performed on all patient isolates for the presence of *mecA*, *SCCmec* typing, *arcA*, ACME, *lukSF-PV* and *cap5* genes (Additional file 1) [14-17].

Statistical Analysis

Fisher's Exact test was used to explore for associations of site of infection, pre-existing SSTI and respiratory disease with age group and PFGE type. A Fisher's Exact p -value of < 0.05 was considered statistically significant. Data were analyzed using SAS (SAS Cary, NC, version 9).

Results

From June 1, 2007 to December 31, 2007, 63 pediatric patients with MRSA infections were seen at the emergency room, outpatient clinics and the inpatient ward at Mercy Children's Hospital. Of these, only 5 patients did not meet the CDC clinical criteria for CAMRSA [4]. Thus, 58 (92%) of all pediatric MRSA infections were community-acquired. All of these CAMRSA infections were SSTIs. During this time period there were no other culture positive invasive pediatric MRSA infections like bacteremia, sepsis, endocarditis, pneumonia, or osteomyelitis.

The patient characteristics for CAMRSA infections are shown in Table 1. The turnover of patients at Mercy Children's Hospital in 2007 was about 22,000 (inpatients 2400) pediatric patients. In 2007, 26% of all children seen at Mercy Children's hospital were African American compared to 62% of our CAMRSA patients and; 51% were Medicaid insurance patients and therefore belonged to the lower socio economic group compared to 74% of our CAMRSA patients. All of these CAMRSA isolates were susceptible to trimethoprim/sulfamethoxazole, clindamycin, rifampin, linezolid and vancomycin.

The distribution of SSTI sites is shown in Table 2 and reflects an age associated shift. In children ages 0-3 years, 19 (76%) had perineal SSTI which was more common. In contrast, only 2 (11%) of the children ages 4-12

Table 1 Patient Characteristics

	n (%)
Age group (years)	
0-3	25 (43%)
4-12	19 (33%)
13-18	14 (24%)
Gender	
Female	36 (62%)
Male	22 (38%)
Race	
Caucasian	16 (29%)
African American	34 (62%)
Other	5 (9%)
Type of Visit	
ER	27 (47%)
Inpatient	11 (19%)
Outpatient	20 (34%)
Insurance	
Private	6 (11%)
Medicaid	43 (74%)
Uninsured	9 (16%)
Preexisting Conditions (<i>patients may have more than 1</i>)	
SSTI	24 (41%)
Respiratory Disease	22 (38%)
Gastrointestinal	9 (16%)
Cardiovascular	3 (5%)
Nervous system	1 (2%)
None	19 (33%)
Site of Infection	
Face	4 (7%)
Lower extremity	18 (31%)
Perineal	21 (36%)
Trunk	4 (7%)
Upper extremity	11 (19%)

ER: Emergency room, SSTI: Skin and soft tissue infection

Table 2 Associations with Age Group

	Age 0-3 N = 25	Age 4-12 N = 19	Age 13-18 N = 14	Fisher's Exact Two-tailed P-value
Site of Infection				< 0.001
Face	2 (8%)	1 (5%)	1 (7%)	
Lower extremity	2 (8%)	8 (42%)	8 (57%)	
Perineal	19 (76%)	2 (11%)	0 (0%)	
Trunk	1 (4%)	3 (16%)	0 (0%)	
Upper extremity	1 (4%)	5 (26%)	5 (36%)	
Respiratory disease pre-existing condition	7 (28%)	9 (47%)	6 (43%)	0.42
SSTI pre-existing condition	12 (48%)	9 (47%)	3 (21%)	0.25
PFT Sub-type 1 (compared to types 2-7 combined)	19 (79%)	14 (74%)	9 (64%)	0.59

PFT: Pulse field type, SSTI: Skin and soft tissue infection

had perineal SSTI and none of the 13-18 year olds. Infections in the extremities were more common in the older youth compared to the youngest children: 3 (12%) in ages 0-3, 13 (68%) in ages 4-12, and 13 (93%) in ages 13-18. Overall, there was a significant association between site of the infection and age group (Fisher's Exact p-value < 0.001) (Table 2).

Nineteen (33%) children had no pre-existing condition. Age group was not associated with having either respiratory disease or SSTI as a pre-existing condition (Fisher's Exact p-values > 0.05 - Table 2). Pulsed field typing (*Smal* PFT) of the 58 CAMRSA isolates revealed that they were all USA300 PFT with seven different sub-types (Figure 2), and one sub-type dominated in 72% (n = 42) (Table 3). PFGE sub-type I (compared to sub-types 2-7) was not associated with site of infection or with having respiratory disease or SSTI as a pre-existing condition, all with Fisher's Exact p-values > 0.05 (Table 4). The DiversiLab typing of the 58 CAMRSA strains revealed that 55 (95%) were indeed USA300 PFT while 3 strains were identified as USA500.

All 58 CAMRSA strains were *mecA* and *lukSF-PV* positive, with *SCCmec* type IVa; 56 (97%) contained ACME and the *cap5* gene was present in 46 (79%) (Table 3). We observed that all the *cap5* negative strains belonged to a single USA300 PFT pattern (Table 3 and Figure 2).

Discussion

Figure 1 clearly demonstrates a six fold increase in the incidence of pediatric MRSA infections from 2002 to 2007 (p < 0.0001). A clear majority of our pediatric MRSA infections were CAMRSA, which is consistent with the trend reported for the United States [18] and Europe [19,20].

A significant association of age with site of infection has for the first time been demonstrated in our study.

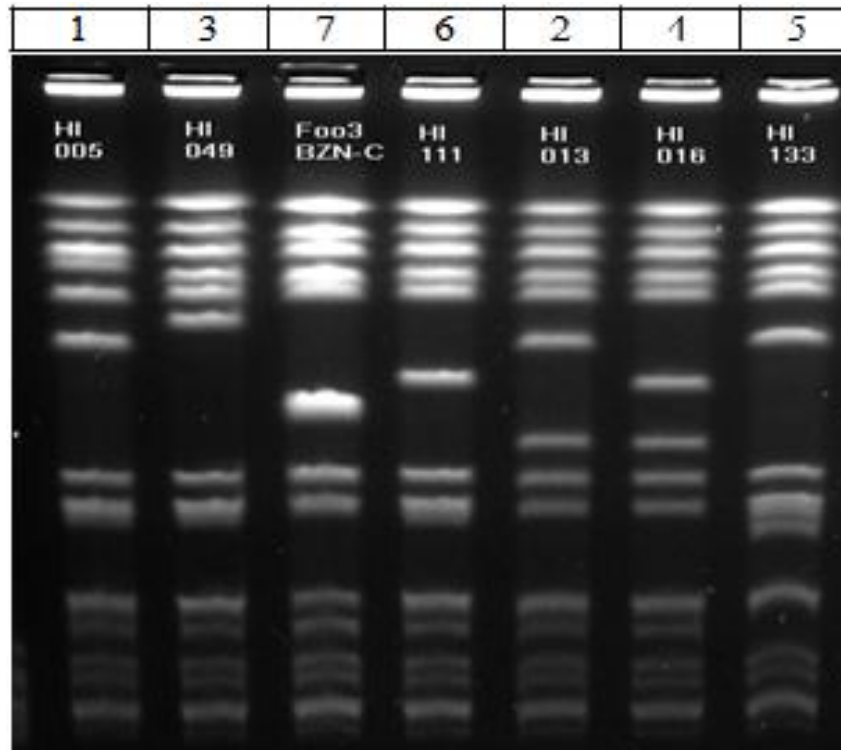


Figure 2 USA300 PFT subtypes. This is a picture of the USA300 strains all run on a single gel and are labeled according to the PFT subtypes in Table 2, 3 & 4.

Perineal MRSA colonization in children has been recorded in the daycare setting [21]. Koski *et al* indicate that pediatric perineal infections with MRSA are increasing [22]. We found that such infections were more common to the 0-3 y-old cohort. This age preference could be due to increased rates of perineal CAMRSA colonization in this age group, but may also reflect use of diapers and possible dermabrasion caused by vigorous wiping of the area during diaper changes or

both. Transfer of vancomycin resistance gene *vanA* from vancomycin resistant *Enterococcus* to MRSA has been shown to occur in vitro and in vivo [23]. Perineal CAMRSA infection in children could contribute to the emergence of vancomycin resistant *S.aureus* strains when co-colonized with vancomycin resistant *Enterococcus* [24].

Of interest, all of the CAMRSA strains in our study were susceptible to clindamycin, contrary to higher rates

Table 3 Molecular epidemiology of USA300 PFT strains (n = 58)

USA300 PFT subtypes n(%)	Diversilab type* n(%)	<i>mecA</i>	SCC <i>mec</i> type	ACME	<i>lukSF-PV</i>	<i>cap5</i>
1 - 42(72%)	USA300-41(98%) USA500-1(24%)	42	IVa-42	42	42	30(71%)
2 - 6(10%)	USA300-4(67%) USA500-2(33%)	6	IVa-6	6	6	6
3 - 4(7%)	USA300-4	4	IVa-4	4	4	4
4 - 2(3%)	USA300-2	2	IVa-2	1	2	2
5 - 2(3%)	USA300-2	2	IVa-2	2	2	2
6 - 1(2%)	USA300-1	1	IVa-1	0	1	1
7 - 1(2%)	USA300-1	1	IVa-1	1	1	1
Total(n)	58(100%)	58(100%)	58(100%)	56(97%)	58(100%)	46(79%)

PFT: Pulsed field type; SCC: Staphylococcal Cassette Chromosome, *lukSF-PV* genes that encode Pantone-Valentine leukocidin (PVL), ACME: Arginine catabolic mobile element.

*The Diversilab strain types are named as corresponding PFT's based on a standardized comparison.

For USA300 PFT subtypes please see figure 2.

Table 4 Associations with PFT Sub-type

	PFT Sub-type 1 N = 42	PFT Sub-types 2-7 N = 16	Fisher's Exact Two-tailed P-value
Site of Infection			
Face	2 (5%)	2 (13%)	0.35
Lower extremity	11 (26%)	7 (43%)	
Perineal	16 (38%)	5 (31%)	
Trunk	4 (10%)	0 (0%)	
Upper extremity	9 (21%)	2 (13%)	
Respiratory disease pre-existing condition	15 (36%)	7 (47%)	0.54
SSTI pre-existing condition	18 (43%)	6 (40%)	1.0

PFT: Pulse field type, SSTI: Skin and soft tissue infection

of resistance reported for Alaska, Houston and San Francisco [25-27].

Strain typing of our patients' MRSA isolates supports the observation that USA300 PFT is the most common causative strain and is clonal [27]. The molecular characteristics of our isolates were similar to the USA300 strains from other published reports in that they were *SCCmec* type IVa and *lukSF-PV* positive [27,28]. 56/58 of the strains were also positive for ACME, a novel mobile genetic region predominantly reported in USA300 MRSA strains that potentially enhance colonization and virulence [29]. We observed that all the 12 strains that were *cap5* gene negative belonged to the predominant USA300 PFT subtype (Table 3 and Figure 2). *cap5* gene encodes for a capsule that enhances the virulence of *Staphylococcus aureus*. It has been used as vaccine target. The clinical significance of *cap5* negative strains in our study is unclear at this time.

Typing using the DiversiLab system is rapid and user friendly compared to PFT. However, differentiating PFT USA300 from USA500 is tricky using DiversiLab [30]. Of interest, 3/3 isolates identified by PFT as USA300 were mis-identified by DiversiLab as USA500. These isolates were also positive for ACME which is a mobile genetic element that is thought to be acquired by USA300 as it evolved from its progenitor USA500 [31].

Conclusion

The incidence of CAMRSA infection is increasing in the pediatric population of Northwest Ohio. SSTIs are the most common type of infection and among children < 3 years of age with perineal SSTIs being the dominant form caused by strain USA300 PFT that carries the *SCCmec* type IVa, *lukSF-PV* gene and ACME. The automated rapid strain typing method, the DiversiLab system, is not as discriminative as PFT.

Further investigations are needed to assess the extent of USA300 perineal colonization in toddlers and to identify unique virulence characteristics to develop strategies for prevention and treatment of these infections.

Additional material

Additional file 1: Primers used for the PCR reactions for *mecA*, *SCCmec* typing, *arcA*, ACME, *lukSF-PV* and *cap5* genes. Details of the sequences of all the primers used in this study along with the references are in the file.

Abbreviations

ACME: Arginine catabolic mobile element; CAMRSA: Community-acquired methicillin resistant *Staphylococcus aureus*; MRSA: Methicillin resistant *Staphylococcus aureus*; PCR: Polymerase chain reaction; PFT: Pulsed field type (or) pulsed field gel electrophoresis; *S.aureus*: *Staphylococcus aureus*; SSTI: Skin and soft tissue infection.

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Author details

¹The University of Toledo College of Medicine, 3000 Arlington Avenue, Toledo-OH, 43614, USA. ²Department of Pediatrics, Division of Infectious Diseases, The University of Toledo College of Medicine and Mercy Children's Hospital, 2222 Cherry Street, Toledo-OH, 43608, USA. ³Mercy Integrated Laboratories, 2222 Cherry Street, Toledo-OH, 43608, USA. ⁴Department of Pediatrics, Division of Infectious Diseases, Children's Memorial Hospital, 2300 Children's Plaza, Chicago-IL 60614-3363, USA. ⁵Department of Medical Microbiology and Immunology, The University of Toledo College of Medicine, 3000 Arlington Avenue, Toledo-OH, 43614, USA. ⁶Program in Bioinformatics & Proteomics/Genomics, The University of Toledo College of Medicine, 3000 Arlington Avenue, Toledo-OH, 43614, USA.

Authors' contributions

AM conducted patient chart review, ran PCR tests and wrote the first draft of the manuscript. MS did the collection and freezing of MRSA samples and ran PCR tests. XZ ran PCR tests and managed the MRSA database. JH did the DiversiLab typing. WK conducted the pulsed field gel typing experiments. RY was involved in the concept of the study and reviewed the manuscript. RB was involved in the concept of the study; contributed his expertise in conducting the PCR experiments; edited and reviewed the manuscript; and mentored medical students AM and MS in the laboratory. DM was the principal investigator and was involved in the concept, planning and coordination of the study. She also mentored medical students AM and MS. In addition, she was responsible for writing, editing and submission of the manuscript. All authors read and approved the final manuscript.

Competing interests

The authors declare that they have no competing interests.

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