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Outpatient Pediatric Community-Acquired Methicillin-Resistant *Staphylococcus aureus*: A Polymorphous Clinical Disease

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Community-acquired methicillin-resistant Staphylococcus aureus *(CAMRSA) presents numerous diagnostic and therapeutic problems for the outpatient physician, including the appropriate use of antibiotics and proper counseling of families on ways to prevent household spread. Most cases of CAMRSA in children involve soft tissue and skin infection, which is precisely the type of infection most likely to be diagnosed in a dermatology practice. We reviewed 8 pediatric cases of cutaneous CAMRSA that presented over 8 months. The 8 pediatric patients presented with one or more of the following: folliculitis (n=4), abscesses of the groin (n=3), impetiginized atopic dermatitis (AD)(n=2), pustules (n=2), bullous impetigo (n=1), and nonbullous impetigo (n=1). Three caregivers of these children developed abscesses in exposed areas such as the forearm (n=3) and calf (n=1). The folliculitis cases involved the abdomen, groin and diaper region, buttocks, and inner thighs; the impetiginized AD did not differ from the distribution of the AD. The variety of clinical presentations and the spread in households represent a few of the many facets of CAMRSA in the pediatric dermatology outpatient setting.*

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Staphylococcus aureus is a common cause of morbidity in cutaneous disease.[1] Among the primary diseases, *S aureus* is known to cause or associate with staphylococcal scalded skin syndrome, folliculitis, bullous impetigo, impetigo, toxic shock–like syndrome, furunculosis, and carbunculosis.[2] Patients with atopic dermatitis (AD) generally are colonized with *S aureus* and overt secondary infection may occur when the staphylococcal burden increases.[3] *S aureus* has developed a myriad of ways to resist the effects of antibiotics. Today, most hospital-acquired cases of *S aureus* are resistant to penicillins, including the prototype methicillin, and other classes of antibiotics, such as macrolides, tetracyclines, lincosamides, and sulfonamides.[4] Since the mid-1990s, community-acquired methicillin-resistant *S aureus* (CAMRSA) increasingly has become an issue.[1,4,5] A chart review of pediatric patients in Houston, Texas, showed that the percentage of cases of CAMRSA in community-acquired *S aureus* infections in hospitalized pediatric patients increased from 56% in 2000-2001 to 78% in 2003.[1] Despite many insensitivities, CAMRSA in the United States remains susceptible to some antibiotics, unlike hospital-acquired methicillin-resistant *S aureus*. Nevertheless, deciding which antibiotic to use can be difficult because in vitro susceptibility may not reflect reality, as evidenced by inducible clindamycin resistance.[4,5] Furthermore,

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transmissibility of plasmid-encoded resistance from one bacterium to another may result in a widespread epidemic of bacterial antibiotic resistance extending beyond CAMRSA.

Case Reports

To help identify target populations for screening in our department, case reports were collected retrospectively from a review of all charts for children identified as having methicillin-resistant *S aureus* infections in the outpatient pediatric dermatology clinic and private practice at St. Luke's-Roosevelt Hospital Center and Beth Israel Medical Center, both in New York City, between March and October 2005. No cases of CAMRSA were noted in either location in 2003 and 2004, which may reflect sampling increases in 2005. Our 8 pediatric patients are summarized in the Table. We describe in detail 3 patients illustrating the difficulty of treating outpatient CAMRSA.

Patient 1—A 33-month-old girl initially presented to our clinic in July 2004 at the age of 21 months with a pruritic rash that had not responded to topical hydrocortisone ointment 1% prescribed by her pediatrician. Her prior medical history was notable for asthma, and she was reported to have a drug allergy to amoxicillin (urticaria) and a food allergy to peanuts. There was no relevant surgical history. On physical examination at the time of presentation, the patient was noted to have pruritic, erythematous, brown, postauricular plaques on the chest, neck, and lower extremity. The patient's AD was diagnosed and treated with emollients as needed and pimecrolimus cream 1% twice daily as needed. The patient's AD remained well-controlled, and 6 months after initiating treatment, the pimecrolimus cream 1% was discontinued by the parents because of the lack of active dermatitis.

The patient presented again in July 2005. On physical examination, she had multiple erythematous papules with diffuse surrounding erythema and satellite pustules in the perineum and excoriations on her buttocks. The patient also had multiple dome-shaped papules on her right lateral chest wall and contiguous lower abdominal area. Molluscum contagiosum viral infection, AD flare, and candidal diaper dermatitis were diagnosed. The patient was treated with cantharidin solution 0.7% for the papules on her chest wall and abdomen, with instructions to wash the areas within 4 hours of applying cantharidin and then apply bacitracin ointment for crusting. Fluticasone propionate ointment 0.005% was prescribed for her AD flare, and nystatin cream thrice daily was prescribed to treat the candidal diaper dermatitis of her perineum and buttocks. Three weeks later, the patient's molluscum contagiosum had resolved and her eczematous AD was well-controlled. However, on physical examination, she had multiple scattered 1- to 2-mm pustules with surrounding erythema on her lower abdomen, mons pubis, perineum, and buttocks (Figure 1). Fluticasone propionate ointment 0.005% and nystatin cream were discontinued. A pustule on her thigh and a pustule on her nares were cultured. Folliculitis was diagnosed and the patient was treated with cephalexin 40 mg/kg daily for 2 weeks. Subsequently, her cultures grew *S aureus* resistant to penicillin (minimum inhibitory concentration [MIC] >16) and oxacillin sodium (MIC >8). The nares culture also was noted to be resistant to clindamycin (MIC >8), erythromycin (MIC >8), and rifampin (MIC >4). Both isolates were sensitive to trimethoprim/sulfamethoxazole (TMP/SMX) (MIC <10), gentamicin (MIC <2), and vancomycin (zone of inhibition, 20 mm). At one-week follow-up, the child was afebrile and less pruritic, but the small pustules and erythematous papules in the groin persisted, though they were less prominent than the week before. The cephalexin was discontinued at one week; she was started on oral TMP/SMX (TMP 10 mg/kg) and mupirocin ointment 2% to her nares 3 times daily for 2 weeks. The lesions resolved, with extensive postinflammatory pigmentary alteration. Her caretaker also was urged to be evaluated and treated because of reports of boils on her arms.

Patient 2—A 9-year-old boy presented to our clinic for evaluation of recurrent boils on the inner thighs and buttocks of 2 months' duration. The patient had a prior medical history notable for asthma, chronic otitis media, and attention deficit hyperactivity disorder. He was taking albuterol as needed, atomoxetine daily, and topically applying fluocinonide ointment. On physical examination, the patient had an erythematous 1.5-cm dermal nodule with central punctum, bilateral erythema, and atrophy on the buttocks, and bilateral fissuring and erythema in the inguinal region. A few scattered pustules on an erythematous base were notable on the inner thighs (Figure 2). Recurrent furunculosis and abscesses were diagnosed and a nasal swab was taken. The patient was placed on clindamycin phosphate topical gel 1% twice daily and instructed to use hexachlorophene antibacterial wash daily. The patient returned 2 weeks later and reported no improvement. At that time, it was noted his nasal swab grew *S aureus* resistant to penicillin (MIC >16),cefazolin (MIC >32), and oxacillin sodium (MIC >8), but sensitive to clindamycin (<0.5)(despite erythromycin insensitivity), TMP/SMX (<10), and vancomycin (MIC <0.5). The patient was sulfur allergic and had minimal

Eight Cases of Pediatric Community-Acquired Methicillin-Resistant *Staphylococcus aureus* (CAMRSA)

Patient No.	Age, y	Sex	PMH	Clinical Appearance of CAMRSA	Lesion Location	Treatment Immediately Prior to MRSA	Failed Antibiotics	Successful Treatment
1	2.75	F	AD, asthma, drug and food allergy	Folliculitis, pustules	Groin, buttocks, abdomen	Pimecrolimus cream 1%, cantharidin solution 0.7%, fluticasone proprionate ointment 0.005%, nystatin cream	Cephalexin	1. TMP/SMX 2. Mupirocin ointment 2%
2	9	M	Asthma, chronic OM, ADHD	Folliculitis, boils	Buttocks, thighs	Albuterol, atomoxetine, fluocinonide ointment	Clindamycin phosphate topical gel 1%, linezolid	1. Rifampin 2. TMP/SMX 3. Mupirocin ointment 2%
3	2	M	Severe AD, food allergy	Impetigi-nized AD/ nonbullous impetigo, pustules	General-ized from the neck and below, scalp	Triamcinolone acetonide ointment 0.025%, mometasone furoate ointment 0.1%, pimecrolimus cream 1%, tacrolimus ointment 0.03%, hydrocortisone valerate ointment 0.2%, mupirocin ointment 2%, petrolatum	Cephalexin, mupirocin ointment 2%	1. TMP/SMX 2. Mupirocin ointment 2%
4	2	M	AD	Bullous impetigo	Chest, axilla, upper arm	Cantharidin solution 0.7%	N/A	1. Cephalexin
5	16	F	N/A	Folliculitis, recurrent abscesses	Buttocks, groin, inner thighs	N/A	Ciprofloxacin hydrochloride, clindamycin phosphate topical gel 1%	1. Doxycycline

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Table. (continued)

Patient No.	Age, y	Sex	PMH	Clinical Appearance of CAMRSA	Lesion Location	Treatment Immediately Prior to MRSA	Failed Antibiotics	Successful Treatment
6	3	M	N/A	Folliculitis	Lower abdomen, groin, inner thighs, diaper region	N/A	N/A	1. Clindamycin phosphate topical gel 1%
7	1.5	M	N/A	Recurrent deep abscesses	Inner thighs	Intravenous ceftriaxone disodium, drainage of abscesses in the operating room	N/A	Intravenous ceftriaxone disodium, drainage of abscesses in the operating room
8	3	F	AD	Excoriated and oozing atopic plaques	Arms, legs, abdomen	Pimecrolimus cream 1%, topical hydrocortisone cream 1%	Mupirocin ointment 2%	1. Cephalexin 2. Mupirocin ointment 2%

Abbreviations: PMH, prior medical history; MRSA, methicillin-resistant *Staphylococcus aureus*; F, female; AD, atopic dermatitis; TMP/SMX, trimethoprim/sulfamethoxazole; M, male; OM, otitis media; ADHD, attention deficit hyperactivity disorder; N/A, not applicable.

response to topical clindamycin; therefore, clindamycin phosphate topical gel 1% was stopped and linezolid 400 mg twice daily for 2 weeks was tried with concurrent mupirocin ointment 2% thrice daily for his nares. He completed the course of linezolid but was noted to have recurrent follicular erythema and papules on his left buttock and flank 2 weeks later. Clindamycin phosphate topical gel 1% twice daily and fluocinonide cream 0.05% twice daily were prescribed pending repeat cultures, the results of which were identical to the initial results. The patient was continued on this therapeutic regimen, but 2 weeks later, he returned with increased pruritus and worsening papular erythema on his right buttocks and flank. He was started on TMP/SMX 3 times daily (TMP 10 mg/kg) and rifampin 600 mg daily for 2 weeks. Two weeks later, the patient's lesions were noted to have completely resolved, with postinflammatory pigmentary alteration. He has not had recurrences in the past 6 months.

Patient 3—A 2-year-old boy presented at the age of 18 months with severe AD and associated bacterial superinfection. The patient had a prior medical history notable for severe AD, which he presented with at 6 months of age to his primary care physician. His prior medical history also was notable for a food allergy to eggs. He had no notable surgical history. His prior medications for AD were alternatively triamcinolone acetonide ointment 0.025%, mometasone furoate ointment 0.1%, pimecrolimus cream 1%, tacrolimus ointment 0.03%, and hydrocortisone valerate ointment 0.2%. The patient also had been placed on mupirocin ointment 2% and unknown oral antibiotics. His family history was notable for asthma and AD in his parents and first cousins. When the patient first presented at

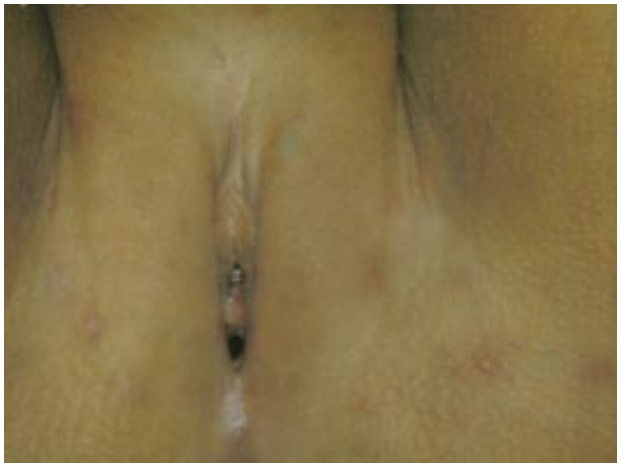

Figure 1. Pustules on an erythematous base in the groin region. Hypopigmentation resulted from treatment with cantharidin solution 0.7%.

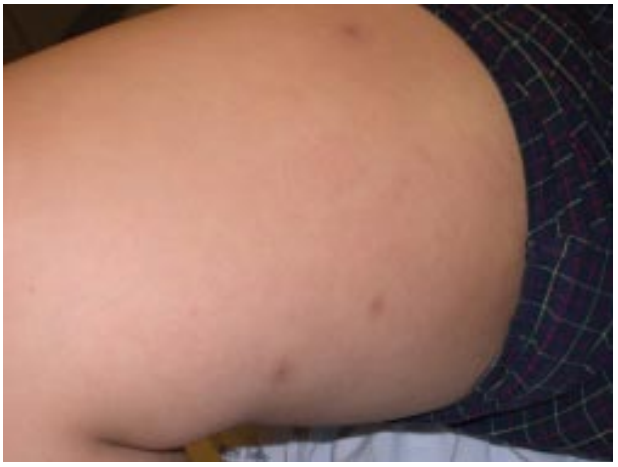

Figure 2. Scattered pustules on an erythematous base on the inner thigh.

18 months, his mother was using petrolatum twice daily. The patient was noted to have extensive eczematous plaques and crusting over his calves, thighs, and forearms bilaterally. In addition, there was erythematous eczematous scale present on his scalp, chest, cheeks, dorsal hands, and especially his neck. Severe AD and associated superinfection (clinically nonbullous impetigo) were clinically diagnosed. He was placed on cephalexin 40 mg/kg for 2 weeks, hydroxyzine nightly before bed (10 mg by mouth), mometasone furoate ointment 0.1% twice daily, and topical emollients. Two weeks later, the patient was seen again and was noted to be tremendously improved. He was placed on tacrolimus ointment 0.03% twice daily, and mometasone furoate ointment 0.1% use was changed to as needed for tacrolimus-unresponsive lesions. Although the patient's mother was instructed to place him in a bathtub with a quarter cup of chlorinated bleach for 15 minutes twice weekly, she neglected to do so. The patient returned 6 months later with similar symptoms to his original presentation, including extensive pustules and erythematous scaling on his neck, chest, back, abdomen, and hands. His mother reported use of a healing ointment alone, without topical medicaments. Wound cultures were taken of his pustules. Superinfected AD was clinically diagnosed. His mother was noted to have a single pustule on her forearm and was asked to apply mupirocin ointment 2% thrice daily. The patient was placed on cephalexin 40 mg/kg for 2 weeks and triamcinolone acetonide ointment 0.1% twice daily. In addition, the patient's mother was instructed to place him in a chlorinated bleach bath daily, as per the prior description. Four days following her initial presentation, the patient's mother developed a tender forearm abscess in the location of the prior pustule. This lesion was cultured in the emergency department. She was then placed on cephalexin, to which she was not responsive and developed another abscess on the right calf. Subsequently, the patient's wound culture grew methicillin-resistant *S aureus* sensitive to rifampin (MIC <1), clindamycin (MIC <0.5), TMP/SMX (MIC <10), gentamicin (MIC <2), and vancomycin (MIC <0.5). On receipt of the culture results, the patient was switched from cephalexin to oral TMP/SMX (TMP 10 mg/kg) twice daily for 2 weeks. He also was given mupirocin ointment 2% to apply to his nose twice daily for 5 days. The patient's mother did not respond to cephalexin after 4 days of therapy and also was started on TMP/SMX (2 double-strength tablets daily). Her skin isolate had a similar resistance pattern as her son. One week later, her pain had resolved, but a 1-cm^2 nodule persisted on her forearm that was incised and drained but did not grow any bacteria. Two weeks later, the patient's father developed abscesses on the arms, for which he was successfully treated with TMP/SMX at the same dosage as the patient's mother. No culture was available for the father's lesions.

Comment

The patients in our review presented with an array of signs and symptoms typical of the varied appearance of cutaneous CAMRSA in an outpatient pediatric dermatology setting. Although, in the literature, CAMRSA infection may cause more serious disease (eg, osteomyelitis, septic arthritis, empyema, necrotizing pneumonia), most CAMRSA infections of the skin and soft tissue remain, as found in our cases.[4] A number of genomic virulence factors have been

identified for CAMRSA, including the *mecA* gene encoding methicillin resistance, the Panton-Valentine leukocidin thought to be common in US strains of CAMRSA, and 18 other virulence factors.[6]

AD has been long recognized as a breeding ground for staphylococcal colonization and infection. The reason is not clear, but it may be related to a combination of decreased cutaneous antimicrobial peptides in patients with AD and *S aureus*–secreted superantigens.[3] Estimates of its prevalence among those with AD historically have been as high as 90%.[7] While a more recent study has revised this estimate to approximately 64%, *S aureus* superinfection of AD is common and becomes more common with increasing AD severity.[8] Nasal carriage may act as a reservoir for repeated cutaneous reinfection. Furthermore, the seriousness of cutaneous *S aureus* infection should not be underestimated because it also has been shown to be a possible gateway to subsequent invasive infections, including bacteremia, osteomyelitis, and endocarditis.[9] CAMRSA infection in a vulnerable population, such as severe AD, could conceivably lead to a higher risk of invasive methicillin-resistant *S aureus* in those patients. One study suggested that 31.1% of isolates of *S aureus* are methicillin resistant in patients with AD.[10] Equally worrisome is the rising rate of methicillin-resistant *S aureus* infections among the general population. As recently as 2000, a study at Bellevue Hospital in New York City showed a prevalence of methicillin-resistant *S aureus* nasal colonization to be 1 in 500.[11] Since that time, multiple studies in other more endemic communities have shown the percentage of CAMRSA as a total of all *S aureus* hospitalized infections to be as high as 78%, with an increase every year it was measured.[5]

Two of our patients (patients 1 and 4) who also had AD were treated with cantharidin solution 0.7% for coexisting molluscum contagiosum infection within the week prior to *S aureus* infection. It is not definite that this drug caused their subsequent methicillin-resistant *S aureus* infections because these patients did have AD; however, possible involvement should not be overlooked. These cases represent only a small fraction of the total number of cantharidin-treated children seen in our office; we typically treat 25 to 30 children weekly with cantharidin for molluscum. Furthermore, a prior case report linked cantharidin treatment for molluscum contagiosum with subsequent staphylococcal toxic shock syndrome.[12] Many practitioners have advocated the clearance of active AD lesions prior to cantharidin application, which seems judicious because of the risk of *S aureus* infection in actively blistered skin.

In addition to topical steroids, the topical calcineurin inhibitors tacrolimus and pimecrolimus have not been associated with superficial bacterial infections in clinical trials.[13] Three of our patients (patients 1, 3, and 8) were noted to have used topical pimecrolimus prior to developing CAMRSA infections. One study has raised the possibility that tacrolimus may serve to reduce the staphylococcal colonization in patients with AD.[14] Patients treated with tacrolimus were shown to have decreased *S aureus* colonization compared with controls after one week of treatment. Although tacrolimus is not thought to have any innate antistaphylococcal activity, this action may be mediated by decreasing transepidermal water loss and the reduction of proinflammatory cytokines.[14] Data collected for the US Food and Drug Administration approval of the topical calcineurin inhibitors have not demonstrated greater risk of cutaneous infection for patients with AD. Because of the virulence of methicillin-resistant *S aureus*, all types of treatments may be complicated, with greater numbers of bacterial infections.

Folliculitis is a common pediatric skin infection. In the case of CAMRSA folliculitis, the appearance is distinctive but not pathognomonic. Small pustules on an erythematous base seem to typify these lesions. CAMRSA lesions also are often associated with abscesses. The groin, inner thighs, umbilicus, buttocks, or diaper region seem to provide a fertile ground for CAMRSA overgrowth. For this reason, groin and buttock folliculitis in pediatric patients should be cultured to rule out CAMRSA. Treatment of CAMRSA is fraught with difficulties. Foremost among these difficulties is the initial dilemma of systemic versus topical treatment.

In the setting of AD, prior research has shown a benefit to using topical antiseptics or antibiotics for AD, especially in severe cases in which the burden of microbial colonization is high.[3,15,16] Although a prior randomized controlled trial has failed to provide evidence for the routine use of systemic antibiotics for severe AD, AD impetiginized with CAMRSA may need to be teated due to its high potential to spread through households and schools.[17] Recurrent infections often are treated with intranasal mupirocin and topical anti-infectives. The combination of mupirocin intranasally and a topical chlorhexidine hydrochloride wash has been shown to reduce nosocomial infections in intensive care unit settings when people were screened and found to carry methicillin-resistant *S aureus* nasally.[18] Combination of mupirocin intranasally and topical anti-infectives results in 71.4%, 91.4%, and 92.4% eradication with 1, 2, and 3 courses, respectively.[19] In countries with a high rate of mupirocin use (eg, Australia),

CAMRSA isolates will be routinely resistant to mupirocin. Blanket use of mupirocin in pediatric patients with AD may create mupirocin-resistant isolates. Therefore, mupirocin use should be limited to reduction of nasal carriage or small localized superficial skin infections. Systemic antibiotics can be used to clear more extensive infection, topical antiseptics (eg, chlorhexidine hydrochloride) to control colonization and superinfection, and topical antibiotics to wipe out nasal carriage. Many practitioners use chlorinated bleach baths to avoid antibiotics; however, there is little evidence in the literature regarding the efficacy of bleach baths in CAMRSA colonization or infection.

In reviewing the literature and our cases, the oral therapeutic regimen that was most likely to clear CAMRSA infection was 14 days of TMP/SMX; however, other systemic antibiotics, such as 2-week courses of doxycycline or clindamycin, also were used with success. Cost wise, both TMP/SMX and doxycycline (for children 9 years and older) are cheap and effective. For systemic therapy, TMP/SMX is the most appropriate treatment for CAMRSA in the pediatric population. If TMP/SMX is contraindicated, then clindamycin is similarly safe and efficacious. Clindamycin may be effective in some patients, but resistance, both natural and inducible, varies depending on the population. Linezolid or quinolones (in children older than 12 years) could be considered with certain reservations. Linezolid costs $2000 or more for a 2-week course and is associated with some bone marrow suppression. However, when other agents are contraindicated because of allergy, age restrictions (tetracyclines are not advisable for patients younger than 9 years), or sensitivity patterns, linezolid can be used. Doxycycline can be effective if sensitivities are known, but it is restricted to older patients. Quinolones are not to be used empirically in children younger than 12 years; however, use can be decided on a case-by-case basis, measuring clinical severity of disease and possible alternatives. In general, 57% to 79% of isolates are sensitive to quinolones; thus, although their use can be productive, it requires culture results to determine sensitivities.[20,21] Rifampin has a synergistic effect when combined with another antibiotic, but high rates of resistance arise when it is used alone. Rifampin is ideal, as seen in patient 2, when used adjunctively with another antibiotic to eliminate nasal carriage.

An article reviewed therapeutic response of CAMRSA abscesses to antibiotics to which the bacteria were technically insensitive. In this review, abscesses that were less than 5 cm^2 and drained cleared well despite ineffective antibiotics, with a notable 6% complication rate.[22] Thus, it is advisable to drain even small abscesses in the era of CAMRSA. Three of our patients (patients 4, 7, and 8) treated for CAMRSA with cephalosporins responded and cleared infection even though their cultures grew methicillin-resistant *S aureus*; this result agrees with reports in the literature of cephalexin being clinically effective in many situations where the microbiology would support cephalexin failure.[4,5,21] Patients 4 and 8 also were treated for underlying AD and were prescribed concurrent chlorinated bleach baths, aiding in their recovery. The one patient prescribed a full course of linezolid (patient 2), which is considered by some to be first-line treatment for CAMRSA skin infections, had rapid disease recurrence. Because of the high-cost differential of linezolid compared with TMP/SMX or doxycycline (linezolid is 200 times more expensive),[23] the use of linezolid should be reserved for severe hospital-acquired infections.

The use of clindamycin for CAMRSA is at present an area of great concern and study. Even though most CAMRSA isolates are found to be clindamycin sensitive, inducible clinical resistance has become a hot topic. Briefly, an *S aureus* isolate that carries the *erm* gene will be erythromycin resistant but clindamycin sensitive in vitro. Yet this same isolate in vivo will behave as if it is clindamycin resistant. Confusingly, not all methicillin-resistant *S aureus* that are erythromycin resistant but clindamycin sensitive carry the *erm* gene but carry a separate gene, *msrA*, for resistance.[5] Epidemiologically, the rate of *erm* presence in studied isolates varies greatly based on location, with estimates widely varying from 2% to 94%.[1] Use of the D-test to differentiate between these phenotypically similar but genetically different isolates is now recommended.[5] Because these isolates are resistant based on plasmid-encoded genes, transfer of resistance patterns between bacteria and individuals is easily accomplished.

Nevertheless, even proper treatment of a methicillin-resistant *S aureus* infection may not eliminate the reservoir of *S aureus*. Patient 3 in particular illustrated the need to be aware of household spread of CAMRSA. In this case, not only was the patient a 2-year-old boy with a history of severe AD that was positive for CAMRSA, but both his mother and father presented with abscesses. In the patient's mother, culture and clinical course demonstrated methicillin-resistant *S aureus*. In these types of cases where methicillin-resistant *S aureus* is spreading between family members, it is appropriate to recommend frequent hand washing with an antiseptic wash containing ethyl alcohol, triclosan, or chlorhexidine hydrochloride. Furthermore, it also

may be appropriate to recommend nasal swabbing and therapy for all household carriers to eliminate the reservoir of CAMRSA.

It is evident that CAMRSA represents a growing problem for the infectious disease community. Pediatric dermatologists are in a unique position to help prevent the spread of this disease within the community. It is our recommendation that any patient with AD and signs of bacterial superinfection should be immediately cultured. Clues that suggest CAMRSA are location of infection in the groin and deep-seated abscesses. Physicians should place these patients on systemic antibiotics, depending on the seriousness of infection. It is vital that abscesses be incised and drained, otherwise they are not likely to respond to topical or systemic antibiotics. Conversely, superficial infections with perhaps low burden of colonization may be treated with antibiotics thought to be resistant based on in vitro test results. Concurrent mupirocin ointment 2% remains an appropriate treatment for eradication of nasal carriage, usually a 5- to 10-day treatment. Alternatively, the addition of rifampin to other effective antibiotic regimens will reduce nasal carriage and disease recurrence. Oral antibiotic therapy should be based on the age of the patient, size of lesions, and prior therapies.

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