# Atopic Patients Appear Free of Extra MRSA Risk

BY DOUG BRUNK
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SAN DIEGO — Patients with atopic dermatitis are highly colonized with *Staphylococcus aureus*, but do not appear to be preferentially infected with community-acquired methicillin-resistant *S. aureus*.

Up to 79% of patients with atopic dermatitis have *S. aureus* in their anterior nares, 64%-75% have it on their normal skin, and more than 90% have it on their lesional skin.

In contrast, up to 30% of atopic-free adults have *S. aureus* in their nasal carriage and 10% have it on their skin, Dr. Sheila Fallon Friedlander said at a conference sponsored by Rady Children's Hospital, San Diego.

A reason why patients with atopic dermatitis may have trouble with *S. aureus* is that they appear to lack an adequate number of cathelicidins, said Dr. Friedlander, director of pediatric dermatology at the University of California, San Francisco. "The end result clinically is that they don't have as much antimicrobial peptide helping to ward off infection. Atopics also have an impaired skin barrier, sometimes as a result of abnormal or decreased filaggrin. With increasing dryness and lack of appropriate adhesion, there is also increased skin surface area for the *S. aureus* to adhere to."

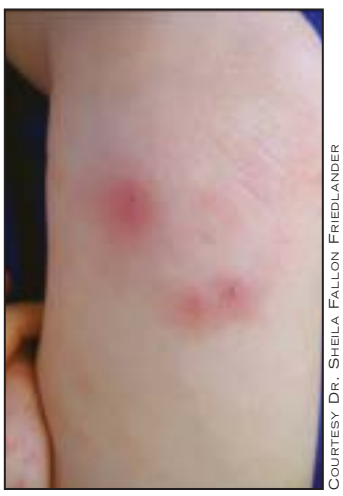
In addition, she said *S. aureus* can elaborate superantigens, "which have the ability to stimulate the production of alternative glucocorticoid receptors. These receptors are more resistant to the effects of steroids. So often when *S. aureus* is present in the skin, there is elaboration of a receptor [that] makes it more difficult for the patient to respond to topical corticosteroid treatment."

A recent study estimated that 40%-66% of patients with atopic dermatitis develop *S. aureus* skin infections (Pediatr. Derm. 2000; 17:111-4). Another concluded that the condition is a risk factor for invasive *S. aureus* infection in this patient population, including bacteremia, osteomyelitis, and endocarditis (Am. J. Med. 2005;118: 1048-51).

However, Dr. Friedlander described the link between atopic dermatitis and invasive disease as "a controversial issue" with data that remain unclear. "In some papers, it appears that there are lower levels of invasive disease in patients with atopic dermatitis," she said. "Invasive disease does occur, but atopics don't seem to be at higher risk for invasive disease. Further studies are required to clarify this issue."

The good news is that patients with atopic dermatitis do not seem to be preferentially affected with community-acquired methicillin-resistant *S. aureus* (CA-MRSA). An estimated 6%-19% of children with atopic dermatitis were found to have the condition (Arch. Dermatol. 2002;138:939-41). Another group of researchers concluded that the "observed incidence of cutaneous CA-MRSA lesions in patients with atopic dermatitis or other non–intact skin barrier is less than reported in other at-risk groups" (J. Clin. Dermatol. 2007;8: 259-70).

Dr. Friedlander explained that, compared with the hospital-acquired form of MRSA, the community-acquired form is clonal, has many drug options, and has a predilection for skin disease. "It preferentially infects the skin; skin and soft tissue infections are what we see," she said. "But you must remember that you can have invasive disease from this organism."



COURTESY DR. SHEILA FALLON FRIEDLANDER

**This MRSA infection was mistaken for spider bites.**

Clinically, CA-MRSA presents as furuncles or folliculitis 65%-95% of the time. "Parents will say, 'my child keeps getting a spider bite,' " Dr. Friedlander said. Most patients look well, but 40%-50% will have fever.

Most often, CA-MRSA organisms possess Panton-Valentine leukocidin, a virulence factor that is a bicomponent cytotoxin. This virulent toxin "destroys our leukocytes by punching holes in the membrane," she said. "This leads to capillary dilation and significant necrosis."

CA-MRSA also may possess an aberrant fibronectin-binding protein gene, which enables the *S. aureus* "to adhere better to our tissue and therefore enhances invasion."

Incision and drainage alone appears to suffice in CA-MRSA skin and soft tissue lesions smaller than 5 cm. "If a lesion is bigger than that, you need to be aggressive," she said. "Please culture these lesions. In the old days, people used to drain these and throw the exudate away. Do not throw it away because the antibiotic susceptibility patterns differ from the hospital-acquired form, and you may need this information to determine the best therapy for your patient."

Dr. Friedlander often starts patients on clindamycin. However, some organisms may be resistant, and therefore you need to check for inducible resistance in these patients.

"Sometimes you need to use trimethoprim/sulfamethoxazole," she said. "In older children, you can use minocycline or doxycycline, but remember, these drugs can damage the teeth of young children, and I will not use tetracyclines in children less than 8 years of age."

Attempts to eradicate MRSA with various combinations of antibiotics have had mixed results, but recent studies have found the use of bleach baths in combination with nasal mupirocin to be useful.

Dr. Friedlander recommends adding one-quarter of a cup of Clorox to a regular bath and repeating this treatment two times a week. In addition, some experts apply mupirocin ointment twice a day to the nares for 1 week each month.

"If you don't repeat the mupirocin treatment for a week each month, the patient appears more likely to colonize," she said. "There is no absolutely clear superior, evidence-based regimen, but studies are ongoing and we may have some more information in the future."

Dr. Friedlander disclosed that she has received grant and research support, honoraria, and/or consulting fees from Barrier Therapeutics Inc., Medicis Pharmaceutical Corp., Sanofi-Aventis, and Stiefel Laboratories Inc. ■

# Skin Infections in Young Athletes Demand Extra Vigilance

BY SUSAN LONDON
*Contributing Writer*

VANCOUVER, B.C. — Managing skin infections in young athletes can be more challenging than in the general pediatric population, because close physical contact and use of shared equipment can lead to rapid spread of infections and outbreaks.

In addition, some athletes with skin infections must be cleared by a physician to return to play and will try to hide symptoms. "Realize that you are going to be tricked and that athletes are going to try to make lesions look like something else," Dr. Andrew Gregory said at a meeting on pediatric and adolescent sports medicine sponsored by the American Academy of Pediatrics. They may try to abrade lesions with sandpaper, cover them with makeup, or bleach them.

"If methicillin-resistant *Staphylococcus aureus* [MRSA] is not in your community yet, it is going to be," Dr. Gregory predicted, noting the prevalence of outbreaks on athletic teams in recent years. "You need to be more aware of this in the athletic population than in the rest of your practice because of the risk of spread."

Good hygiene is a key to preventing MRSA, said Dr. Gregory of the departments of orthopedics and pediatrics at Vanderbilt University, Nashville, Tenn. Coaches and certified athletic trainers should encourage athletes to shower and clean their equipment regularly with soap and water and to avoid sharing equipment, clothing, towels, and razors. In addition, he recommends cleaning any shared equipment and surfaces with bleach and putting alcohol-based hand-sanitizing gels in training rooms, locker rooms, and bathrooms.

When MRSA is detected in one athlete, coaches and athletic trainers should talk with others on the team to see if any of them have lesions, Dr. Gregory advised. Treatment of MRSA in this population is the same as that in other children and adolescents—incision and drainage and antibiotic therapy appropriate for that specific community. "That's different for every community, so you need to be aware of what the sensitivities are locally," he said.

According to recommendations from the CDC, athletes with any staphylococcal infection—including MRSA—should receive oral antibiotic therapy for a minimum of 3 days of before returning to play sports involving skin-to-skin contact, he noted (see www.cdc.gov/ncidod/dhqp/ar_MRSA_AthletesFAQ.html).

Finally, Dr. Gregory cautioned, physicians and administrators should beware of sales pitches for products such as turf coatings that promise to protect athletes from MRSA. "There is no evidence that they do what they claim," he said.

Tinea infection, called tinea gladiatorum in wrestlers, was historically attributed to dirty mats, but efforts to culture the fungus from this source have failed, so it is now believed to be passed primarily by skin-to-skin contact, Dr. Gregory said. "These lesions are tough to diagnose when they are pretty small, before they get the central clearing," he observed.

Treatment consists of topical antifungal agents as first-line therapy and oral ones as second-line therapy. Wrestlers with this infection must be withheld from practice and competition until they have had treatment for 48-72 hours, and simply covering lesions is inadequate, Dr. Gregory said. He also recommended considering antifungal prophylaxis when athletes have recurrences or when outbreaks occur.

Herpes simplex I infection is spread by direct skin-to-skin contact and is also common among wrestlers, in whom the infection is called herpes gladiatorum. Typically, there are lesions on the right side of the head, related to the starting position for this sport, and it is important to prevent infection from spreading to the eye, which may lead to herpes conjunctivitis.

"It is a little bit difficult to tell that this is a herpes infection initially, before you get that characteristic vesicular rash," he commented. Physicians should be suspicious whenever they see wrestlers with a raised erythematous rash. "The key is to recognize it early and initiate treatment early," he said, with an appropriate course of an antiviral such as acyclovir. Antiviral prophylaxis should be considered for athletes with recurrences or when outbreaks occur.

Most physicians agree that wrestlers with herpes infections can return to play after all of their lesions have crusted over—usually in 10-14 days—although some advocate waiting until the lesions are completely healed, Dr. Gregory noted.

Dr. Gregory reported that he had no conflicts of interest in association with his presentation. ■