National Athletic Trainers' Association Position Statement: Skin Diseases

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Objective: To present recommendations for the prevention, education, and management of skin infections in athletes.

Background: Trauma, environmental factors, and infectious agents act together to continually attack the integrity of the skin. Close quarters combined with general poor hygiene practices make athletes particularly vulnerable to contracting skin diseases. An understanding of basic prophylactic measures, clinical features, and swift management of common skin diseases is essential for certified athletic trainers to aid in preventing the spread of infectious agents.

Recommendations: These guidelines are intended to provide relevant information on skin infections and to give specific recommendations for certified athletic trainers and others participating in athletic health care.

Key Words: tinea capitis, tinea corporis, herpes simplex, molluscum contagiosum, impetigo, folliculitis, furuncle, carbuncle, community-associated methicillin-resistant *Staphylococcus aureus* (CA-MRSA)

T he nature of athletics exposes the skin of its participants to a wide variety of stresses. Trauma, environmental factors, and infectious agents act together to continually attack the integrity of the skin. Combined with the close quarters shared by athletes and generally poor hygiene practices, it is not difficult to see why skin infections cause considerable disruption to individual and team activities.[1] Skin infections in athletes are extremely common. Authors[2] of a recent literature review investigating outbreaks of infectious diseases in competitive sports from 1922 through 2005 reported that more than half (56%) of all infectious diseases occurred cutaneously. Recognition of these diseases by certified athletic trainers (ATs), who represent the first line of defense against spread of these infections to other team members, is absolutely essential. Prophylactic measures and swift management of common skin infections are integral to preventing the spread of infectious agents. The following position statement and recommendations provide relevant information on skin infections and specific guidelines for ATs working with the athletes who contract them.

RECOMMENDATIONS

Based on the current research and literature, the National Athletic Trainers' Association (NATA) suggests the following guidelines for prevention, recognition, and management of athletes with skin infections. The recommendations are categorized using the Strength of Recommendation Taxonomy criterion scale proposed by the American Academy of Family Physicians[3] on the basis of the level of scientific data found in the literature. Each recommendation is followed by a letter describing the level of evidence found in the literature supporting the recommendation: *A* means there are well-designed experimental, clinical, or epidemiologic studies to support the recommendation; *B* means there are experimental, clinical, or epidemiologic studies that provide a strong theoretical rationale for the recommendation; and *C* means the recommendation is based largely on anecdotal evidence at this time.

The recommendations have been organized into the following categories: prevention, education, and management of the skin infections. The clinical features of the most common skin lesions are presented in Table 1.

Prevention

1. Organizational support must be adequate to limit the spread of infectious agents.

   a. The administration must provide the necessary fiscal and human resources to maintain infection control.[30,31] *Evidence Category: B*

   b. Custodial staffing must be increased to provide the enhanced vigilance required for a comprehensive infection-control plan. *Evidence Category: C*

   c. Adequate hygiene materials must be provided to the athletes, including antimicrobial liquid (not bar) soap in the shower and by all sinks.[7,32–35] *Evidence Category: B*

   d. Infection-control policies should be included in an institution's policies and procedures manuals.[22,31,36–38] *Evidence Category: C*

   e. Institutional leadership must hold employees accountable for adherence to recommended infection-control practices.[8,30,39–43] *Evidence Category: B*

   f. Athletic departments should contract with a team dermatologist to assist with diagnosis, treatment,

Table 1. Clinical Features of Common Skin Infections

Family	Specific Condition	Clinical Features
Fungal infections	Tinea capitis	Often presents as gray, scaly patches accompanied by mild hair loss (Figure A).[4,5]
	Tinea corporis	Presents with a well-defined, round, erythematous, scaly plaque with raised borders; however, tinea corporis gladiatorum (tinea corporis in wrestlers) frequently presents with a more irregular lesion (Figure B).[4–6]
Viral infections	Herpes simplex	Lesions are typically found on the head, face, neck, or upper extremities and present as clustered, tense vesicles on an erythematous base (Figure C).[4,5,7–16]
	Molluscum contagiosum	Typically presents as umbilicated, or delled, flesh-colored to light-pink pearly papules, measuring 1–10 mm in diameter (Figure D).[17–21]
Bacterial infections	Impetigo	Bullous impetigo presents on the trunk or the extremities with raised blisters that rupture easily, resulting in moist erosions surrounded by a scaly rim. Nonbullous impetigo presents with thin-walled vesicles that rupture into a honey-colored crust (Figure E).[2,4,22]
	Folliculitis	Presents as papules and pustules at the base of hair follicles, especially in areas that have been shaved, taped, or abraded (Figure F).
	Furuncles, carbuncles	Furuncles present as tender areas that, over several days, develop a reddened nodular swelling (Figure G); carbuncles present as the coalescence of multiple furuncles in a deep, communicating, purulent mass.[4,23,24]
	MRSA	CA-MRSA initially presents similarly to other bacterial infections. Furuncles, carbuncles, and abscesses are the most frequent clinical manifestations. (Figure G).[15,25,26] Often CA-MRSA lesions are confused with spider bites.[25,27,28] Lesions may begin as small pustules that develop into larger pustules or abscesses with areas of erythema and some tissue necrosis (Figure H and I).[27,29]

Abbreviations: CA, community-associated MRSA; MRSA, methicillin-resistant *Staphylococcus aureus.*

and implementation of infection control.[44] *Evidence Category: C*

2. A clean environment must be maintained in the athletic training facility, locker rooms, and all athletic venues.

 a. Cleaning and disinfection is primarily important for frequently touched surfaces such as wrestling mats, treatment tables, locker room benches, and floors.[9,10,45,46] *Evidence Category: A*

 b. A detailed, documented cleaning schedule must be implemented for all areas within the infection-control program, and procedures should be reviewed regularly. *Evidence Category: C*

 c. The type of disinfectant or detergent selected for routine cleaning should be registered with the Environmental Protection Agency, and the manufacturer's recommendations for amount, dilution, and contact time should be followed.[10,31,47] *Evidence Category: B*

3. Health care practitioners and athletes should follow good hand hygiene practices.[31,48]

 a. When hands are visibly dirty, wash them with an acceptable antimicrobial cleanser from a liquid dispenser.[48,49] *Evidence Category: A*
 Correct hand-washing technique must be used, including wetting the hands first, applying the manufacturer's recommended amount of antimicrobial soap, rubbing the hands together vigorously for at least 15 seconds, rinsing the hands with water, and then drying them thoroughly with a disposable towel.[48] *Evidence Category: A*

 b. If hands are not visibly dirty, they can be decontaminated with an alcohol-based hand rub.[17,18,41,50,51] *Evidence Category: B*

 c. Hands should be decontaminated before and after touching the exposed skin of an athlete and after removing gloves.[52–56] *Evidence Category: B*

4. Athletes must be encouraged to follow good overall hygiene practices.[57–59]

 a. Athletes must shower after every practice and game with an antimicrobial soap and water over the entire body. It is preferable for the athletes to shower in the locker rooms provided by the athletic department.[57] *Evidence Category: B*

 b. Athletes should refrain from cosmetic body shaving.[25] *Evidence Category: B*

 c. Soiled clothing, including practice gear, undergarments, outerwear, and uniforms, must be laundered on a daily basis.[10] *Evidence Category: B*

 d. Equipment, including knee sleeves and braces, ankle braces, etc, should be disinfected in the manufacturer's recommended manner on a daily basis.[58] *Evidence Category: C*

5. Athletes must be discouraged from sharing towels, athletic gear, water bottles, disposable razors, and hair clippers.[57,59] *Evidence Category: A*

6. Athletes with open wounds, scrapes, or scratches must avoid whirlpools and common tubs. *Evidence Category: C*

7. Athletes are encouraged to report all abrasions, cuts, and skin lesions to and to seek attention from an AT for proper cleansing, treatment, and dressing. *Evidence Category: C* All acute, uninfected wounds (eg, abrasions, blisters, lacerations) should be covered with a

semiocclusive or occlusive dressing (eg, film, foam, hydrogel, or hydrocolloid) until healing is complete to prevent contamination from infected lesions, items, or surfaces. *Evidence Category: C*

Education

The sports medicine staff must educate everyone involved regarding infection-control policies and procedures.[7,32–35,60]

1. Administrators must be informed of the importance of institutional support to maintaining proper infection-control policies.[7,32–35,60] *Evidence Category: B*
2. Coaches must be informed of the importance of being vigilant with their athletes about following infection-control policies to minimize the transmission of infectious agents.[7,32–35,60] *Evidence Category: B*
3. Athletes need to be educated on their role in minimizing the spread of infectious diseases.

   a. Follow good hygiene practices, including showering with antimicrobial soap and water after practices and games and frequent hand washing.[57–59] *Evidence Category: B*
   b. Have all practice and game gear laundered daily.[10,17] *Evidence Category: B*
   c. Avoid sharing of towels, athletic gear, water bottles, disposable razors, and hair clippers.[57,59] *Evidence Category: B*
   d. Perform daily surveillance and report all abrasions, cuts, and skin lesions to and seek attention from the athletic training staff for proper cleansing, treatment, and wound dressing. *Evidence Category: C*

4. The custodial staff must be included in the educational programs about infectious agents to be able to adequately help in daily disinfection of the facilities.[10] *Evidence Category: C*

Management

Fungal Infections.

1. Tinea capitis (Figure A)

   a. *Diagnosis*: A culture of lesion scrapings is the most definitive test, but a potassium hydroxide (KOH) preparation gives more immediate results.[61] *Evidence Category: B*
   b. *Treatment*: Most patients have recalcitrant cases and should be treated with systemic antifungal agents: for example, a "cidal" antifungal drug, such as terbinafine, or alternative, such as fluconazole, itraconazole, or ketoconazole (Table 2). Adjunctive therapy with selenium sulfide shampoo is also recommended.[4,57,61,62] *Evidence Category: B*
   c. *Criteria for return to competition*: Athletes must have a minimum of 2 weeks of systemic antifungal therapy (Table 3).[63,64] *Evidence Category: B*

2. Tinea corporis (Figure B)

   a. *Diagnosis*: A culture of lesion scrapings is the most definitive test, but a KOH preparation gives more immediate results.[61] *Evidence Category: B*
   b. *Treatment*: Topical treatment with a cidal antifungal agent, such as terbinafine, naftifine, ciclopirox, or oxiconazole (or more than one of these), twice a day, is effective for localized lesions. More diffuse inflammatory conditions should be treated with systemic antifungal medication (Table 2).[11,57,61,62,65] *Evidence Category: B*
   c. *Criteria for return to competition*: Athletes must have used the topical fungicide for at least 72 hours, and lesions must be adequately covered with a gas-permeable membrane (Table 3).[63,64] *Evidence Category: B*

Viral Infections.

1. Herpes simplex (Figure C)

   a. *Diagnosis*: A culture of lesion scrapings is the most definitive test but may take days. A Tzanck smear that identifies herpes-infected giant cells may give more rapid, accurate results.[1,57,61,66] *Evidence Category: B*
   b. *Treatment*: New, active lesions may be treated with an oral antiviral medication, such as valacyclovir, to shorten the duration of the infection and lessen the chance of transmission.[57,67–72] *Evidence Category: B* Fully formed, ruptured, and crusted-over lesions are unaffected by antiviral medication. *Evidence Category: B*
   c. *Criteria for return to competition*[64]

      i. Athlete must be free of systemic symptoms, such as fever, malaise, etc. *Evidence Category: B*
      ii. Athlete must have developed no new blisters for 72 hours. *Evidence Category: B*
      iii. All lesions must be surmounted by a firm adherent crust. *Evidence Category: B*
      iv. Athlete must have completed a minimum of 120 hours of systemic antiviral therapy. *Evidence Category: B*
      v. Active lesions cannot be covered to allow participation. *Evidence Category: B*

2. Molluscum contagiosum (MC; Figure D)

   a. *Diagnosis*: Clinical findings and microscopic inspection are the basis for diagnosis.[73] *Evidence Category: C*
   b. *Treatment*: Many anecdotal therapies have been suggested, but physical destruction of the lesions with a sharp curette is recommended.[26,64,73–81] *Evidence Category: B*
   c. *Criteria for return to competition*: Lesions should be curetted and covered with a gas-permeable membrane (Table 3).[64] *Evidence Category: B*

Figure. Skin diseases. A, Tinea capitis. B, Tinea corporis. C, Herpes simplex. D, Molluscum contagiosum. E, Impetigo. F, Folliculitis. G, Furuncle/carbuncle. H and I, Methicillin-resistant *Staphylococcus aureus*. **All photos used with permission from www.dermnet.com.**

Table 2. Recommended Pharmacologic Treatment Regimens for Common Skin Infections

Condition	Agent	Brand[a]	Type	Dose	Frequency, ×/d	Duration, wk
				Pharmacologic Intervention		
Tinea capitis	Terbinafine	Lamisil	Rx	Oral 250 mg	1	2–4
	Ketoconazole	Nizoral	Rx	Oral 200 mg	1	2–4
	Itraconazole	Sporanox	Rx	Oral 200 mg	1	2–4
	Fluconazole	Diflucan	Rx	Oral 6 mg/kg	1	3–6
Tinea corporis and tinea cruris	Terbinafine 1% cream	Lamisil	OTC	Topical	2	2–4
	Ketoconazole 2% cream	Nizoral	OTC	Topical	1	2–4
	Clotrimazole 1% cream	Lotrimin	OTC	Topical	1	2–4
	Naftifine 1% cream[b]	Naftin	Rx	Topical	2	1
	Oxiconazole 1%[b]	Oxistat	Rx	Topical	2	1
	Ciclopirox 0.77% cream[b]	Loprox	Rx	Topical	2	1
	Fluconazole	Diflucan	Rx	Oral 150 mg	1×/7 d	2–4
	Itraconazole	Sporanox	Rx	Oral 100 mg	1	2
	Terbinafine	Lamisil	Rx	Oral 250 mg	1	1
Tinea pedis	Ketoconazole 2% cream	Nizoral	OTC	Topical	1	4–6
	Clotrimazole 1% cream	Lotrimin	OTC	Topical	1	
	Fluconazole	Diflucan	Rx	Oral 150 mg	1×/7 d	4
	Itraconazole	Sporanox	Rx	Oral 100 mg	1	4
	Terbinafine	Lamisil	Rx	Oral 250 mg	1	4
Herpes simplex (primary)	Valacyclovir	Valtrex	Rx	Oral 1.0 g	3	1–1.5
Herpes simplex (recurrent)	Valacyclovir	Valtrex	Rx	Oral 500 mg	2	1
	Acyclovir	Zovirax	Rx	Oral 800 mg	5	1
Molluscum contagiosum	Physical destruction of the lesions					
Impetigo	Mupirocin 2% ointment	Bactroban	Rx	Topical	2	1
	Fusidic acid 2% cream, hydrocortisone	Fucidin H	Rx	Topical	2	1
	Retapamulin 1% ointment	Altabax	Rx	Topical	2	5 d
Folliculitis, furuncles, carbuncles, or methicillin-resistant *Staphylococcus aureus*	Systemic antibiotic use is determined on a case-by-case basis, based on culture and sensitivity of lesion, and until information is available on antibiotic susceptibilities in the local community.					

Abbreviations: OTC, over-the-counter medication; Rx, prescription required.

[a] Lamisil (Novartis Pharmaceuticals Corporation, East Hanover, NJ); Nizoral (McNeil-PPC, Inc, Fort Washington, PA); Sporanox (PriCara, Raritan, NJ); Diflucan (Pfizer Inc, New York, NY); Lotrimin (Schering-Plough HealthCare Products, Inc, Whitehouse Station, NJ); Naftin (Merz Pharmaceuticals, Greensboro, NC); Oxistat (PharmaDerm, Florham Park, NJ); Loprox (Medicis Pharmaceutical Corporation, Scottsdale, AZ); Valtrex (GlaxoSmithKline, Middlesex, United Kingdom); Zovirax (GlaxoSmithKline); Bactroban (GlaxoSmithKline); Fucidin H (Leo Laboratories, Dublin, Ireland); Altabax (GlaxoSmithKline).

[b] Two of these agents are often used in combination twice a day to resistance.

Bacterial Infections.

1. Impetigo (Figure E)

   a. *Diagnosis*: The diagnosis of bacterial infections is primarily based on the history and characteristic appearance of the lesions.[57] *Evidence Category: B* Specimens for culture and antimicrobial susceptibility should be obtained from any questionable lesions.[57] *Evidence Category: B*

   b. *Treatment*: Culture and sensitivity of suspicious lesions will dictate treatment for all bacterial infections. Topical mupirocin (Bactroban; GlaxoSmithKline, Middlesex, United Kingdom), fusidic acid (Fucidin H; Leo Pharma, Ballerup, Denmark), and retapamulin (Altabax; GlaxoSmithKline, Middlesex, United Kingdom) have been shown effective in treating impetigo.[1,57,82,83] *Evidence Category: B*

   c. *Criteria for return to competition*: Any suspicious lesions should be cultured and tested for antimicrobial sensitivity before the athlete returns to competition (Table 3).[64] *Evidence Category: B*

   i. No new skin lesions for at least 48 hours. *Evidence Category: B*

   ii. Completion of a 72-hour course of directed antibiotic therapy. *Evidence Category: B*

   iii. No further drainage or exudate from the wound. *Evidence Category: B*

   iv. Active infections may not be covered for competition.

2. Folliculitis/furuncles/carbuncles (Figure F and G)

   a. *Diagnosis*: The diagnosis of bacterial infections is primarily based on the history and characteristic appearance of the lesions.[57] *Evidence Category: B* Specimens for culture and antimicrobial susceptibility should be obtained from any questionable lesions.[57] *Evidence Category: B*

   b. *Treatment*: Culture and sensitivity of suspicious lesions dictate treatment for all bacterial infections.[57,84,85]

   i. Athlete must be referred to physician for incision, drainage, and culture. *Evidence Category: B*

Table 3. Return-to-Play Guidelines for Contact-Sport Athletes With Infectious Lesions[a]

Condition	Return-to-Play Guidelines[b]
Tinea corporis	Minimum 72 h topical fungicide terbinafine (Lamisil) or naftifine (Naftin)
	Lesions must be covered with a gas-permeable dressing followed by underwrap and stretch tape
Tinea capitis	Minimum 2 wk systemic antifungal therapy
Herpes simplex (primary)	Free of systemic symptoms of viral infection (fever, malaise, etc)
	No new lesions for at least 72 h
	No moist lesions; all lesions must be covered with a firm, adherent crust
	Minimum 120 h systemic antiviral therapy
	Active lesions cannot be covered to allow participation
Herpes simplex (recurrent)	No moist lesions; all lesions must be covered with a firm, adherent crust
	Minimum 120 h systemic antiviral therapy
	Active lesions cannot be covered to allow participation
Molluscum contagiosum	Lesions must be curetted or removed
	Localized lesions may be covered with a gas-permeable dressing followed by underwrap and stretch tape
Furuncles, carbuncles, folliculitis, impetigo, cellulitis, or methicillin-resistant *Staphylococcus aureus*	No new lesions for at least 48 h
	Minimum 72 h antibiotic therapy
	No moist, exudative, or draining lesions
	Active lesions cannot be covered to allow participation

[a] Based on guidelines adopted by the National Collegiate Athletic Association in 2004.[47]
[b] Lamisil (Novartis Pharmaceuticals Corporation, East Hanover, NJ); Naftin (Merz Pharmaceuticals, Greensboro, NC).

ii. Antibiotic therapy must be initiated to control local cellulitis. *Evidence Category: B*

c. *Criteria for return to competition*: Any suspicious lesions should be cultured and tested for antimicrobial sensitivity before the athlete returns to competition (Table 3).[64] *Evidence Category: B*

 i. No new skin lesions for at least 48 hours. *Evidence Category: B*
 ii. Completion of a 72-hour course of directed antibiotic therapy. *Evidence Category: B*
 iii. No further drainage or exudate from the wound. *Evidence Category: B*
 iv. Active infections may not be covered for competition. *Evidence Category: B*

3. Methicillin-resistant *Staphylococcus aureus* (MRSA) (Figure H and I)

a. *Diagnosis*: The diagnosis of bacterial infections is primarily based on the history and characteristic appearance of the lesions. *Evidence Category: B*

 i. The differential diagnosis for any potential *Staphylococcus* lesion must include MRSA.[27,84,86,87] *Evidence Category: B*
 ii. Reports of "spider bites" should be considered a possible sign for community-associated MRSA (CA-MRSA).[84] *Evidence Category: B*
 iii. Specimens for culture and antimicrobial susceptibility should be obtained from any questionable lesions.[84,86] *Evidence Category: B*

b. *Treatment*: Recognition and referral of athletes with suspicious lesions are paramount. *Evidence Category: B*

 i. Athletes with suspicious lesions must be isolated from other team members. *Evidence Category: B*

ii. Antibiotic treatment must be guided by local susceptibility data and be determined on a case-by-case basis.[23,84,86,88–93] *Evidence Category: A*

c. *Criteria for return to competition*: Any suspicious lesions should be cultured and tested for antimicrobial sensitivity before the athlete returns to competition (Table 3).[64] *Evidence Category: B*

 i. No new skin lesions for at least 48 hours. *Evidence Category: B*
 ii. Completion of a 72-hour course of directed antibiotic therapy. *Evidence Category: B*
 iii. No further drainage or exudate from the wound. *Evidence Category: B*
 iv. Active infections may not be covered for competition. *Evidence Category: B*

Clinical Dermatology: A Color Guide to Diagnosis and Therapy by Habif[94] and *Skin Disease: Diagnosis and Treatment* by Habif et al[95] are excellent references for the recognition, diagnosis, and treatment of skin diseases, as is www.dermnet.com, a Web site that contains more than 23 000 images of skin diseases.

LITERATURE REVIEW

Transmission of the Infectious Agent

For the transmission of infectious agents, 3 basic elements are required: a source of the agent, an adequate susceptible host, and a mode of transmission for the agent to the host.[31,96] Infectious agents in health care settings have been shown to come from many sources, including other patients,[97–100] roommates, and visitors.[99,101] These agents are also present in the athletic setting. The infected source may show active lesions or may be completely asymptomatic while in the incubation period of an infectious disease. It is, therefore, important to always assume that individuals are carriers of pathogenic microorganisms.

A very complex relationship exists between an infectious agent and a potential host patient.[31] Many factors, including the immune state of the patient at the time of exposure, virulence of the infectious agent, quantity of the infectious innoculum, and medications taken by the patient (eg, corticosteroids) can affect the outcome after exposure to an infectious agent.[31,102] Outcomes can range from no effect at all to asymptomatic colonization of the host to full symptomatic disease states.[31] Athletes have unique characteristics that make them particularly susceptible hosts. They participate in high-risk activities[103] and have constant assaults to the integrity of their skin,[57] making transmission that much easier.

Transmission of infectious agents to the host can occur in a myriad of ways: through direct or indirect contact, droplets, airborne routes, or percutaneous or mucous membrane exposure.[31] *Direct transmission* occurs when one infected person transfers the infectious agent to another through direct skin-to-skin contact.[31] *Indirect transmission* refers to situations in which a susceptible person is infected by contact with a contaminated surface, such as a wrestling mat or contaminated clothing. Many cases of indirect transmission in the health care setting are found in the literature, including patient care devices,[104–106] shared toys in pediatric wards,[98] inadequately cleaned instruments,[6,107–109] and poor hand hygiene,[9,45] the latter of which is possibly the most common method of indirect transmission. Inadequate vigilance about hand washing is thought to be largely responsible for transferring infectious agents from one surface to another in health care settings, dramatically increasing disease transmission. Also, clothing has been shown to be contaminated with potential pathogens after coming in contact with infectious agents.[110,111] Although supporting literature on indirect transmission in the athletic setting is lacking, it is not difficult to imagine the potential harm.

Droplet transmission occurs when infected droplets from sneezing, coughing, or talking make contact with the eyes, nose, or mouth of the host subject.[112] *Airborne transmission* occurs when residue from evaporated droplets or dust particles stays suspended in the air for long periods of time and becomes inhaled by a susceptible host.[113,114] In the athletic setting, the most common mode of transmission of skin diseases is direct or indirect contact from the source to the host. Other modes of transmission are beyond the scope of this review.

Prevention

First and foremost, for a prevention plan to be effective, the organization (university, high school, corporation, etc) should be committed to preventing disease transmission.[31] This commitment should be manifested by including disease-transmission prevention in existing safety programs and policies and procedures manuals.[22,36–38] These manuals should describe how the prevention principles will be applied, how infected persons will be identified, and how to communicate information about potentially infected persons to the proper personnel.[31] Skin diseases, especially CA-MRSA, are reaching pandemic proportions, so organizations should be prepared to provide fiscal and human resources for controlling infection in an ever-changing environment.[31]

Furthermore, a culture of institutional safety shared by administrators, staff, and, in this case, athletes is essential to controlling infectious disease.[30] Standard precautions and preventive measures must become the norm in athletic facilities for these programs to be implemented. Hospital-based studies have shown a direct correlation between high levels of "safety culture" and adherence to safe practices. Institutions that have seamlessly integrated these programs into their daily routine have had a high degree of success in keeping their stakeholders accountable for disease-prevention measures.[39,40,43] This adherence to recommended practices can significantly minimize the transmission of infectious disease.[8,41,42]

Education about infectious-disease transmission and the recommended practices to minimize it should be an essential component to any infectious disease-prevention program. Understanding the science behind the recommended practices allows the health care team to more readily apply the standard precautions and modify them to their specific setting.[7,32–35] Adherence to safety precautions is higher in groups that have received education in infectious-disease control.[60]

Hand hygiene is the single most important practice in reducing the transmission of infectious agents.[31,48] Because of the significance of this issue, the Centers for Disease Control and Prevention assembled the Hand Hygiene Task Force, which wrote a 56-page document, "Guideline for Hand Hygiene in Health-Care Settings."[48] The guidelines[48] include recommendations to wash hands with antimicrobial soap when the hands are visibly dirty[49] or with an alcohol-based hand rub in the absence of visible soiling of the hands.[17,18,41,50–52,115] Hands should always be decontaminated before[54] and after contacting a patient's skin,[52,53] after removing gloves,[55,56] and after using the restroom.[116–118] Trivial as it may seem, properly decontaminating the hands is of utmost importance. The correct technique for hand washing includes wetting the hands first, applying an appropriate amount of product, rubbing the hands together vigorously for at least 15 seconds, rinsing the hands with water, and then drying thoroughly with a disposable towel.[48]

The nature of athletic competition necessitates overall good personal hygiene practices. Close personal contact in both locker and dormitory rooms is a significant risk factor in disease transmission.[57–59] Athletes are encouraged to shower with antimicrobial soap and water over the entire body immediately after each practice and game.[57] Athletes should also be discouraged from cosmetic body shaving (ie, shaving a body area other than the face or legs), which has been shown to increase the risk of CA-MRSA more than 6-fold.[25] Good personal hygiene decreases the colonization of bacteria[58] and can be a first line of defense against transmission of infectious agents.

It is also important to maintain a clean environment in the athletic training room, locker rooms, and athletic venues. Cleaning and disinfection is primarily important for frequently touched surfaces, such as wrestling mats, treatment tables, and locker room benches and floors.[9,10,45,46] An example of a cleaning schedule for a National Collegiate Athletic Association (NCAA) Division I wrestling program is provided in Table 4. Maintaining a properly cleaned and disinfected facility requires a team approach, including contributions from ATs, athletic

Table 4. A Sample Cleaning Schedule for a National Collegiate Athletic Association Division I Wrestling Program[a]

	In Season			Off Season[b]			
	Dates: October 1 Through March 31			Dates: April 1 Through Late September			
Area	Chemical	Frequency, ×/d, Time of Day	Others Present?	Chemical	Frequency, ×/d, Time of Day	Labor (Staff Hours/Activity)	Others Present?
Shower room in public locker room (walls, fixtures, and flooring; hard surfaces in shower area)	HBSD	1×/d	Yes	HBSD	1×/d	0.5	Yes
Locker room surfaces (benches, door knobs, handles, walls, mirrors, floors, fourth floor)	HBSD	1×/d, 10 PM–6 AM	Yes	HBSD	1×/d, 10 PM–6 AM	2	Yes
Wrestling room: walls (mats attached to walls to 4′ [1.2 m])	HBSD	1×/d, 10 PM–6 AM	No	HBSD	1×/d, 10 PM–6 AM	4	No
Wrestling room: mats (flooring, seam tape can be replaced by athletic department as needed because of cleaning processes); major/most thorough cleaning overnight[b]	HBSD	3×/d, 11 AM–12 PM, 2–4 PM, 10 PM–6 AM (2×/d Sat and Sun)	No	HBSD	2×/d, 2–4 PM, 10 PM–6 AM	2	No
Wrestling weight room (fourth floor, where bodies touch equipment: benches, grips)	HBSD	1×/d, 10 PM–6 AM	No	HBSD	1×/d, 10 PM–6 AM	1	No
Wrestling room: treatment/ taping tables	HBSD	1×/d, 10 PM–6 AM	No	HBSD	1×/d, 10 PM–6 AM	0.5/area	No
Wrestling support areas (main stairs, rear stairs, public area spaces)	HBSD	1×/d, 10 PM–6 AM	Yes	HBSD	1×/d, 10 PM–6 AM	1	Yes
Steam room, sauna room (walls, benches, flooring, even if a wood/porous surface)	HBSD	1×/d, 10 PM–6 AM	No	HBSD	1×/d, 10 PM–6 AM	1	No
Carpeting: extracting (locker room, weight room, fifth floor adjunct workout area)	NA	Monthly night shift floor crew[c]	No	NA	2×/off season as arranged	30	No
Carpeting: vacuuming (locker room, weight room, fifth floor adjunct workout area)	NA	1×/d, 11 AM–12 PM	Yes	NA	1×/d, 11 AM–12 PM	1	Yes

Abbreviations: HBSD, hospital broad-spectrum disinfectant (bactericide, fungicide, and virucidal efficacy); NA, not applicable.

[a] Club activities (nonwrestling) occur 2 to 3 days per week and may affect the cleaning schedule.

[b] Additional cleaning because of summer wrestling camps at additional cost to athletic department.

[c] Spot clean as needed between monthly cleanings.

administration, coaches, athletes, and custodial staff. Education of all involved parties is essential to minimizing transmission of infectious agents, and regular review of the cleaning procedures should be performed.[48] The type of disinfectant or detergent selected for routine cleaning and disinfection is relatively unimportant, as long as it is registered by the Environmental Protection Agency and the manufacturer's recommendations for amount, dilution, and contact time are all followed.[10,31,47] Some authors have suggested using a 1:10 ratio of household bleach to tap water for routine environmental disinfection.[119] Facility-based pathogen reservoirs most often result from a failure to follow the instructions rather than from the cleaning agent itself.[24,120]

Soiled textiles, including towels, athletic clothing, elastic wraps, etc, can be reservoirs for infectious agents. Although these items can be significant contributors to infectious-disease transmission, if handled, transported, and laundered properly, the risk of transmission to a susceptible host is negligible.[10] Another suggested potential risk factor for acquiring an infectious disease, sharing personal items such as bar soap, towels, water bottles, and protective equipment (eg, wrestling head gear), should be prohibited at all times.[57,59] Athletic clothing and towels need to be laundered every day after practice, and equipment such as neoprene sleeves, knee braces, and other protective equipment should be disinfected with a 1:10 bleach solution daily[58] despite the fact that some authors[121–123] have reported cases of contact dermatitis at this concentration.

The following sections provide literature support for fungal, viral, and bacterial infections. Background information, clinical features, diagnosis, treatment, prevention, and guidelines for return to competition will be presented for each of the infectious agents.

FUNGAL INFECTIONS

Dermatophytes (fungal organisms living in soil, on animals, or on humans[124]) include a group of fungi that infect and survive mostly on dead keratin cells in the stratum corneum of the epidermis. The infectious organisms responsible for fungal infections are typically from the *Trichophyton* genus.[61] Specifically, *Trichophyton tonsurans* and *Trichophyton rubrum* are most often associated with tinea capitis and tinea corporis, respectively.[61,125,126]

Background

Chronic perspiration and the macerating affect of abrasive trauma contribute to the successful penetration of ubiquitous fungal elements, particularly in warm, moist areas such as the toe webs, inguinal creases, and axillary folds. In contact sports, the skin-on-skin contact of the participants and abrasions, both clinical and subclinical, also lend themselves to the passage of fungal infections from one athlete to the next. Dermatophyte infection can be manifested in many ways. Infections on the face and head are called *tinea capitis*, infections on the body are termed *tinea corporis*, infections in the groin are called *tinea cruris*, and infections of the feet are called *tinea pedis*.[57,124]

A number of authors[107,125,127–130] have researched the epidemiologic considerations of this widespread cutaneous problem among athletes.

The most common dermatophyte infection is tinea pedis, with prevalence rates ranging from 25% to 70% over the life span.[127,129] A review of 10 recent reports has presented information on athletic teams infected with tinea corporis.[125] As would be expected, the rates varied greatly from one group to another, depending in part upon the method of fungal identification and the fact that a certain level of penetration of infection into the team was necessary to identify the team for study. Certainly athletic teams at all levels of competition have no evidence of fungal infection. The rates of incidence of infection in the reported studies ranged from 20% to 77%. One overview[128] performed in the mid-1980s indicated that 60% of college wrestlers and 52% of high school wrestlers demonstrated tinea infections at some time during the course of the season. Other investigators have reported that 84.7% of high school wrestling teams had at least 1 wrestler with tinea corporis[131] and 95% of a Swedish club wrestling team exhibited cutaneous findings consistent with tinea infection, with 75% of those demonstrating positive cultures for *T tonsurans*.[130]

From information gathered in these studies, it is obvious that fungal infection rates among athletes vary widely. However, we can assume that any athletic team in which the problem has been identified can expect active infections in one-half to as many as three-fourths of its members, underlying the importance of aggressive treatment of isolated cases once they have been identified.

Clinical Features

The clinical presentation of cutaneous fungal infections is diverse. Tinea capitis often presents as gray scaly patches accompanied by mild hair loss.[57,61] Tinea corporis, commonly known as ringworm, is characterized by a well-defined round, erythematous, scaly plaque with raised borders.[57,61] Tinea corporis gladiatorum (tinea corporis among athletes)[63,125] many times presents with a more irregular lesion, however.[120] Tinea corporis is most commonly found on the head, neck, trunk, and upper extremities and only rarely affects the lower extremities.[125,131] Tinea cruris presents with a well-defined erythematous plaque in the pubic and inguinal areas.[66] Finally, tinea pedis presents in the toe webs, where macerated skin is usually accompanied by thick scaling or desquamation. Marginated erythema with advancing scales will often progress from the toe web to the entire sole of the foot and extend over the lateral margins in the "moccasin" distribution.[62] Although early infection tends to be unilateral, bilateral involvement of the feet is common by the time the athlete seeks attention for the problem. Vesicle formation may appear near the advancing border, and the underside of the epithelium covering these vesicles is a rich source of fungal elements for diagnosis by both KOH preparation[57] and fungal culture.[57,61]

Diagnosis

Although fungal cultures are more definitive than a KOH test, especially for specifically diagnosing the exact causative organism, 3 weeks may be required to determine that a culture is negative. Positive growth, of course, occurs more rapidly. The culture should be taken for ultimate confirmation, but a KOH preparation provides a more immediate determination of infection.[61] In the hands of an experienced practitioner, these simple tests are invaluable for instituting immediate therapy, even though some KOH preparations lead to equivocal results. Very simply, scale obtained from a suspicious lesion is applied to a glass microscope slide, a 10% KOH solution is added, and a coverslip is applied. The slide is warmed, usually with a match, to degrade the keratin and expose the fungal elements.

Treatment

Athletes in noncontact sports or with localized cases may initially be treated with topical preparations as a conservative first-line approach.[57,61] Topical treatments, including the cidal imidazoles, allylamines, and napthiomates, tend to be well tolerated by patients.[57,61] More widespread, inflammatory, or otherwise difficult-to-treat cases may require the use of systemic antifungals, such as fluconazole or terbinafine,[65] which can have substantial side effects.[61]

The topical cidal antifungals terbinafine, naftifine, ciclopirox, and oxiconazole are suggested[11] with 2 to 4 times daily applications. Although this regimen may be effective in the off season, athletes in the midst of a competitive season should probably be treated immediately with oral terbinafine, itraconazole, or fluconazole. Topical treatment is typically required through the entire course of the season or at least 2 weeks in the off season. Typically, systemic treatment for common fungal infections should last 2 to 4 weeks. Scalp lesions can be particularly difficult to eradicate, so systemic therapy with medications such as terbinafine, ketoconazole, itraconazole, or fluconazole may be prescribed for up to 6 weeks; daily use of an antifungal shampoo such as ketoconazole or ciclopirox may be required in particularly virulent scalp infections in athletes.[4,62] A summary of common treatment regimens for dermatophyte infections is presented in Table 2.

Prevention

In athletes who are prone to tinea pedis, careful attention to drying the feet is a necessity, including careful towel drying, particularly of the toe webs. The regular application of foot powder or 20% aluminum chloride (Drysol; Person & Covey, Inc, Glendale, CA) is also valuable. Wearing shower shoes in the locker room may be beneficial. Daily changing of athletic socks and even blow drying of the feet and athletic shoes have also been recommended.[62] Immediately showering after each training session and thoroughly drying all areas, especially intertriginous areas, is recommended, as well as the use of absorbent sports briefs and the application of bacteriostatic powder, such as Zeasorb-AF (Stiefel Laboratories, Inc, Research Triangle Park, NC), to the axillae and groin.

Wrestlers represent a particularly difficult and crucial subset in terms of preventing fungal infections. Wrestlers with extensive active lesions, which can be identified on visual inspection by ATs and coaches, must be withheld from all contact. Wrestlers who have demonstrated a particular susceptibility for tinea corporis in the course of the competitive season have been successfully treated prophylactically throughout the entire season with a low dosage of fluconazole (150 mg every other week or 200 mg

per month).[1,62,132] Wrestlers with particularly recalcitrant (persistent or recurrent) infections should have their family members and animals (eg, dogs, cats, and farm animals) examined as well, because these may be reservoirs for reinfecting the athletes.[133]

Careful attention is also required to disinfecting wrestling mats. Although several investigators[134] were not able to isolate fungal organisms from mats, *T tonsurans* has been cultured from a mat immediately after use.[135] The cleaning of mats is probably not as significant as the concern about skin-to-skin contact, but careful disinfecting greatly reduces and may even eliminate tinea infections in some wrestling teams.[130] Daily cleaning of the mats with chlorine-containing disinfectant sprays, at least during the course of the competitive season, is recommended (Table 4).

Return to Competition

Athletes with tinea may return to sport only after they are cleared by the examining physician or AT.[64] Clearance to compete can only be given if the lesions have adequately responded to treatment, which generally requires 3 days of topical treatment in minor cases or 2 weeks of systemic treatment in more severe cases.[63] Athletes with solitary or closely clustered, localized lesions will not be disqualified if the lesions are in a body location that can be covered securely. The barrier preparation should be a dressing, such as Opsite (Smith & Nephew, London, United Kingdom) or Bioclusive (Johnson & Johnson, Langhorne, PA) followed by Pro Wrap (Fabrifoam, Exton, PA) and stretch tape. Dressings should be changed after each match so that the lesion can air dry[64] (Table 3).

VIRAL INFECTIONS

Two primary viral infections are prevalent in athletic populations: herpes simplex and MC. Herpes simplex infection is common among athletes, especially those engaged in activities with full skin-on-skin contact, such as wrestling[57,136,137] and rugby.[1,57,137] Molluscum contagiosum is a highly infectious pox virus skin infection caused by the MC virus, which is classified within the family of poxviruses (Poxviridae).[75]

Herpes Simplex

Background. Herpes infection is caused by the herpes simplex virus (HSV), and outbreaks in athletes that spread throughout the entire team have been widely reported.[128,136] In a study of high school wrestlers at one summer wrestling camp, 60 of 175 wrestlers at the camp developed herpes lesions.[136] In the general population, up to 60% of college students possessed antibodies for HSV.[138] Herpes infections specifically contracted by athletes were first studied in 1964,[139] but then there was a 24-year hiatus in the literature between the earlier clinical publications and the flurry of clinical reports between 1988 and 1992.[128,136,140,141]

Clinical Features. Clinical features of HSV have been well described in the medical literature since 1964.[139] After an incubation period of 3 to 10 days, patients develop a variety of systemic signs or symptoms depending on their preexisting immunity to HSV. Symptoms can range in severity from a mild viral prodromal illness to an almost influenza-like illness with symptoms of fever, severe malaise, prostration, polyarthralgias, polymyalgias, pharyngitis, and conjunctivitis. Physical signs of the infection in athletes can include disseminated skin lesions and complications of conjunctivitis, keratitis, stomatitis, meningitis, arthritis, and hepatitis as well as marked lymphadenopathy and hepatosplenomegaly. Secondary infection with *S aureus* is common and often simulates bacterial folliculitis.

It is important for ATs to recognize the unique clinical features of HSV infection because an innocuous-appearing HSV lesion on the lip of an athlete can infect many other athletes who lack immunity against the virus. Recurrent HSV infections typically appear as a localized cluster of tense vesicles on the lip; however, it is important to note that particles from the virus reside latently in the dorsal root ganglia of the host's sensory nerves. Thus, recurrent HSV infections can appear in areas other than the lip and oftentimes in areas of previous outbreaks.[57,142] Typically, HSV lesions are located on the head, face, neck, or upper extremities.[19,46,68,128] The outbreak is usually preceded by symptoms that can include irritability, headache, tingling, and burning or itching of the skin at the site of recurrence.[61] Whether athletes are contagious during the prodromal period is unclear. However, we know that individuals with recurrent HSV labialis (fever blisters or cold sores) can shed the virus intermittently between episodes and in the absence of lesions,[143] and these individuals may represent a reservoir of virus for infecting previously uninfected athletes. The presence of HSV in the secretions of uninfected athletes is a significant one that needs to be investigated. If proven, a strong case could be made for season-long daily prophylaxis of all individuals on a team.

After the prodrome described above, a primary HSV outbreak often includes widespread clustered vesicles on an erythematous base in areas of contact of the head and neck, trunk, and arms in infected athletes.[61,144] Many times, numerous clustered perifollicular vesicles crust rapidly, giving the false impression of folliculitis. The vesicles may continue to erupt for a period of 7 to 10 days and eventually evolve into dry, crusted lesions.

Diagnosis. The diagnosis of HSV is often delayed for days and misdiagnosed as occlusion, bacterial folliculitis, or other pyoderma because HSV clinically can simulate these conditions very closely. A high index of suspicion and clinical expertise is critical in evaluating athletes and diagnosing HSV. Viral culture of vesicle scrapings is the most definitive diagnostic tool, but results can take days.[1,57,61,66] A Tzanck smear that identifies herpes-infected giant cells is invaluable in making the correct diagnosis while awaiting the culture results.[1,57,61]

The AT plays a very important and proactive role in the epidemiologic control of skin infections in athletes. This role begins with daily skin examinations before practices and games or matches. Any athletes with suspicious lesions should be immediately triaged to the team physician for disposition the same day. Whenever possible, ATs should establish relationships with local dermatologists to handle all their skin evaluation needs. An individual suspected of having a contagious skin disease should be immediately isolated from other team members until he or she is examined by the team dermatologist and the skin infection is properly managed. Implementation of these stringent epidemiologic-based concepts can result in a significant

reduction in the incidence of skin infections among members of athletic teams.

Treatment. Treatment of primary HSV is most effective with antiviral drugs such as acyclovir or valacyclovir.[69] Acyclovir represented the original therapy for HSV,[70,71] but the unwieldy dosing pattern of 5 times a day made compliance an issue.[67] The typical dosing regimen for valacyclovir, however, is 500 mg twice daily for 7 days.[67,68] Once the lesions are fully formed, ruptured, and crusted over, antiviral medications are no longer effective.[57] Topical antiviral creams have proven to be ineffective.[72]

Retrospectively, Anderson[44] evaluated HSV outbreaks in Minnesota high school wrestlers during the 1999 season. Statistical analysis of these data confirmed the importance of properly screening and triaging all athletes with suspicious skin lesions for diagnosis and treatment before allowing further contact with other wrestlers. The average time from exposure to outbreak was 6.8 ± 1.70 days, with a 32.7% probability of transmission to sparring partners in a group.

Prevention. In an evidence-based study, Anderson[68] reported on the prophylactic use of valacyclovir and concluded that wrestlers with a history of HSV for more than 2 years were adequately treated with valacyclovir 500 mg/d, and those with a history of lesions for less than 2 years showed reductions in HSV infections with 1 g/d of valacyclovir.[145]

Return to Competition. According to NCAA guidelines,[64] the athlete may not return to participation until he or she has received 5 days of oral antiviral therapy and all lesions have a dried, adherent crust (Table 3).

Molluscum Contagiosum

Background. In the United States between 1990 and 1999, 280 000 physician visits per year for MC were estimated.[146] The prevalence of MC in children has been reported to be as high as 7.4%[147] and considerably higher[148] in more confined communities. Several authors[149,150] have found no sex differences in the incidence of MC, whereas others[147] showed boys to be affected more often. The infection is commonly seen in younger children; however, because of skin-to-skin transmission, it is not uncommon for athletes, including swimmers,[147,151] cross-country runners,[5] and wrestlers,[152] to demonstrate MC infection in areas of direct contact with bodily secretions from other athletes. In addition to contact exposure, certain predisposing factors, such as atopic dermatitis, increase the likelihood of developing MC.[153] Many times in these individuals, a small, particularly itchy patch of eczema can develop around the lesions a month or more after their onset.[154] In addition, immunocompromised individuals and those on systemic steroids are at increased risk of developing extensive MC infections.[28,155–157] Paradoxical immunosuppression in young, conditioned athletes has been described as a predisposing factor to explain the prevalence of infection in this population.[96]

Clinical Features. The clinical features of MC are fairly characteristic and usually do not present a diagnostic dilemma. The lesions typically are umbilicated, or delled, flesh-colored to light-pink pearly papules, measuring 1 to 10 mm in diameter.[73] Although usually a benign, self-resolving infection in nonimmunosuppressed people,[29] MC left untreated can persist for 2 to 4 years before clearing spontaneously.[158] Untreated MC can present a number of problems in athletes, including the development of secondary pyodermas with *S aureus* and an eczematous eruption surrounding individual lesions.[75] Rupture of molluscum papules can result in furuncle-like lesions that can heal with depressed varicelliform-type scars.[75,120] In fact, scarring after long-standing, untreated MC is not uncommon.[75]

Diagnosis. Because of the characteristic nature of MC lesions, the diagnosis of clinically suspicious lesions is routinely made on clinical examination. If the diagnosis is still uncertain, a Tzanck smear can be done on the crushed core contents of an individual molluscum papule to look for molluscum bodies, which appear on electron microscopic analysis as large, brick-shaped virus particles in positive samples. The MC lesions can occur as solitary lesions or be clustered (usually no more than 20) on body surfaces and, at times, be inoculated extensively into hair-growing areas, such as the beard or pubic area.[73]

Treatment. Numerous anecdotal therapies have been used for the treatment of MC, including agents such as cantharidin[81] and salicylic acid[79] and modalities such as cryotherapy[74] and pulsed-dye laser.[77] More recently, topical immunomodulators such as imiquimod have been used with varying degrees of success.[26,76] Evidence-based reviews[20] of reported anecdotal treatment modalities for molluscum show no definite statistical evidence of benefits to these therapies.

Physical destruction of scattered MC lesions with a sharp curette is recommended as the preferred method of treatment by many authors,[64,75,78,80] but little evidence-based research has been conducted using randomized controlled trials to evaluate its success. Physical destruction of the lesions is useful for rapidly clearing an athlete's skin and, thus, allowing participation in events and preventing both autoinoculation and spread to other athletes.[80,159] Curettage can be done easily with or without the use of topical anesthetic creams. When extensive, MC can be a reason for an athlete's disqualification from participation; however, solitary lesions can be appropriately covered or curetted before competition, according to the NCAA *Wrestling Championships Handbook*.[64] Although a recent evidence-based medicine review failed to determine any standard effective therapies,[160] the most efficient way to clear this infection rapidly and return the athlete to participation is simple curettage of lesions.

Prevention. Prevention of the spread of this highly contagious infection is best accomplished by meticulous hygiene after exposure to another athlete's skin secretions or inanimate objects that have come in contact with secretions from other athletes, such as swimming pool benches, towels, gym equipment, and wrestling mats.

Return to Competition. In most cases, the athlete must undergo some type of treatment before returning to competition. At this time, the NCAA requires athletes to have the lesions curetted or removed before return to play, although localized or solitary lesions may be covered with a gas-permeable dressing followed by stretch tape[64] (Table 3).

BACTERIAL INFECTIONS

Bacterial infections are most commonly caused by various gram-positive strains of *Streptococcus* and *Staphylococcus*

bacteria.[1,161] As much as 30% of the healthy population is colonized with *Staphylococcus* bacteria in the anterior nares.[162] Outbreaks of *S aureus* infections have been reported in football, basketball, and rugby players.[12,59,163]

Impetigo

Background. Impetigo is a contagious superficial bacterial infection, or pyoderma, of the skin caused by *S aureus* and group A β-hemolytic *Streptococcus*.[57,137,161] Impetigo is classified as bullous or nonbullous forms.[57]

Clinical Features. Bullous impetigo presents with superficial blisters (bullae) that rupture easily. The eruptions are typically moist and surrounded by a scaly rim.[164] Nonbullous impetigo, the most common form, initially presents with a thin-walled vesicle followed rapidly by rupture and desquamation to expose a raw, denuded surface covered with a yellowish-brown or honey-colored serous crusting in the perinasal and periorofacial areas.[1,57,164]

Diagnosis. Diagnosis of bacterial infections is primarily based on the history and characteristic appearance of the lesions, but with the increasing vigilance regarding antibiotic-resistant strains of *Staphylococcus* infections, scrapings or drainage samples of the lesions should be cultured.[57]

Treatment. Although impetigo has no standard therapy, the management guidelines include culture and sensitivity of suspicious lesions and treatment with appropriate topical or oral (or both) antibiotics. Good evidence shows that topical mupirocin, fusidic acid, or retapamulin is as effective or more effective and has fewer side effects than oral antibiotics.[83] Other authors[1,82,83] have recommended antibiotics such as dicloxacillin and cephalexin; or, if the athlete is allergic to penicillins, erythromycin may be used effectively.[1]

Return to Competition. Any suspicious lesions should be cultured and tested for antimicrobial sensitivity before return to competition. In general, return to competition after bacterial infections should not be allowed until the athlete has completed a 72-hour course of directed antibiotic therapy, has no further drainage or exudate from the wounds, and has developed no new lesions for at least 48 hours.[64] Also, because of the communicable nature of bacterial infections, active lesions should not be covered to allow for participation.[64]

Folliculitis, Furuncles, and Carbuncles

Background. Folliculitis, furuncles, and carbuncles are caused by follicular-based *S aureus* infections that arise in areas of high friction and perspiration.[57]

Clinical Features. Folliculitis presents as a myriad of perifollicular papules and pustules on hair-bearing areas, especially in areas that have been shaved, taped, or abraded.[94] Furuncles, or boils, are also follicular-based *S aureus* infections presenting as tender areas that, over a period of a few days, develop a reddened nodular swelling.[57,84,85] The lesions are essentially a perifollicular abscess that often progresses to spontaneous rupture and drainage.[57] Multiple furuncles that coalesce into a common, purulent mass, called a carbuncle, can be associated with surrounding cellulitis.[57,94]

Diagnosis. Diagnosis of folliculitis, furuncles, or carbuncles should follow the same progression as the diagnosis of impetigo. All diagnostic decisions should be based on the history and characteristic appearance of the lesions, with

scraping or drainage samples of the lesions cultured to rule out antibiotic-resistant strains of *Staphylococcus* infections.[57]

Treatment. Athletes with folliculitis should be referred for culture of purulent perifollicular lesions and appropriate antibiotics.[57] Simple furuncles may be treated with warm compresses to promote drainage, but more fluctuant furuncles and carbuncles require incision and drainage.[57,84,165] After drainage, the athlete needs systemic antimicrobial therapy and close follow-up.[57,84,85] These lesions must be managed properly with incision, drainage, and antibiotics to control surrounding cellulitis.[84] As mentioned previously, furuncles and carbuncles may be caused by antibiotic-resistant strains of the *Staphylococcus* bacteria, so it is essential that this diagnosis be considered. Although ATs are not expected to manage these *Staphylococcus* abscesses, the athlete must be referred to a knowledgeable physician who will perform incision and drainage when necessary and treat with oral antibiotics.

Return to Competition. Guidelines for return to competition after folliculitis, furuncles, or carbuncles are the same as for impetigo. Suspicious lesions should be cultured and tested for antimicrobial sensitivity, and return to participation should not be allowed until the athlete has completed at least 72 hours of directed antibiotic therapy, no drainage or exudate is visible from the wound, and no new lesions have developed in the previous 48 hours. Bacterial infections cannot be covered to allow for participation.[64]

Methicillin-Resistant *S aureus*

Background. In the early 1960s, an antibiotic-resistant strain of *S aureus* known as MRSA was described.[87,166] Methicillin-resistant *S aureus* has acquired the mecA gene[167,168] and is resistant to β-lactam antibiotics, including penicillins and cephalosporins,[13,86,88] although resistance to other classes of antibiotics, such as fluoroquinolones and tetracyclines, is increasing.[14,84,88] Until recently, MRSA was thought to be exclusively a hospital-acquired infection.[2,86,88] In the mid- to late 1990s, however, MRSA infections started to be detected in the community outside the typical health care settings,[2,86,168,169] being diagnosed in athletes participating in football,[25,170,171] wrestling,[21] and fencing,[171] where as many as 70% of team members required hospitalization and intravenous antibiotic therapy.[170,171] In one study,[172] the mortality attributable to MRSA infections was estimated to be as high as 22%. This new manifestation of MRSA, called CA-MRSA, is reported to be the most frequent cause of skin infections seen in emergency rooms across the country.[169,173] In one hospital in Texas, the number of CA-MRSA cases increased from 9 in 1999 to 459 in 2003.[174] Although the spectrum of MRSA appears to be similar in both types (furuncles, carbuncles, and abscesses are most commonly reported[27,84,86,88]), CA-MRSA contains isolates that are distinct from those of MRSA acquired in the health care setting.[86,169,175] Risk factors associated with MRSA include recent hospitalization, outpatient visits, or close contact with a person with risk factors.[176] The risk factors for CA-MRSA are not as well defined, and it is not uncommon for patients with no identifiable risk factors to become infected.[177]

An alarming increase in the prevalence of MRSA nasal colonization has been noted in both healthy children[15] and

adults.[178] Nasal colonization of MRSA isolates in healthy children increased from 2.2% to 9.2%[15] and in healthy adults from 0.8% to 7.3% between 2001 and 2004.[86,179] Additionally, transmission of CA-MRSA is quite easy in close-contact settings, such as locker rooms and athletic fields,[175] so prevention, recognition, and proper management of MRSA are important responsibilities for the AT.

Clinical Features. Prompt recognition of bacterial infections in athletes is vital to preventing both the spread of this highly contagious infection to other team members and contamination of athletic facilities where athletes congregate. Health care professionals should always consider CA-MRSA in the differential diagnosis of all patients presenting with symptoms associated with *Staphylococcus* disease.

Initially, CA-MRSA infections present similarly to other bacterial infections.[27,84,86,87] Furuncles, carbuncles, and abscesses are the most frequent clinical manifestations.[88,180] The lesions may begin as small pustules and develop into larger pustules or abscesses with areas of erythema and some tissue necrosis.[84,170] In several documented cases,[16,84,87] patients and their caregivers have confused CA-MRSA lesions with spider bites.

Diagnosis. With any presentation of a skin and soft tissue infection compatible with that caused by *S aureus* or history of a "spider bite," MRSA must be included in the differential diagnosis.[84] Any abscess or purulent skin lesion, particularly with signs of severe local or systemic infection, should be cultured for MRSA isolates and antimicrobial susceptibility.[84,86]

Treatment. It is critical for ATs to understand the proper recognition, dispensation, and management of MRSA infections. An athlete with a suspected MRSA infection must be immediately isolated from other team members and referred to a knowledgeable physician. The physician, who must maintain close contact with the AT in such cases, should abide by the evolving guidelines for the management of these infections. Individual treatment should be guided by local susceptibility data, because prevalence of resistance to antimicrobial agents varies geographically and is likely to change over time.[84] Although evidence from controlled clinical trials is presently insufficient to establish optimal treatment regimens for MRSA, several antimicrobial therapies have been proposed.[84] Mild to moderate cases in patients with no significant comorbidities still respond well to β-lactam agents.[84] Some experts[84,91] suggest that a prevalence of 10% to 15% of *S aureus* isolates, however, means that a change to alternative antimicrobial therapies might be needed. Alternative agents (both oral and parenteral) include vancomycin,[84] clindamycin,[92] daptomycin,[23] tigecycline,[93] minocycline, trimethoprim-sulfamethoxazole,[89] rifampin,[84,89] and linezolid.[84,86]

Given their potential for rapid development of resistance, some antimicrobial agents are discouraged for the treatment of MRSA. Specifically, these agents include fluoroquinolones (ciprofloxacin and levofloxacin)[88,90] and macrolides/azalides (erythromycin, clarithromycin, and azithromycin).[88]

Prevention. Currently, no oral antibiotic prophylaxis is recommended for bacterial infections. Some authors[137,138,180] have discussed using agents such as mupirocin and antiseptic body washes to eliminate *S aureus* nasal colonization in healthy patients, although very limited data

have examined the association between MRSA colonization and subsequent infection.[181] Prophylaxis is best accomplished by following standard infection-control precautions, good hand hygiene, and overall hygiene practices as recommended earlier.

Return to Competition. Because of the prevalence and virulent nature of CA-MRSA, any suspicious lesions should be cultured and tested for antimicrobial sensitivity before the athlete returns to participation. In general, after a bacterial infection, return to play should not be allowed until the athlete has completed a 72-hour course of directed antibiotic therapy, has no further drainage or exudate from the wounds, and has developed no new lesions for at least 48 hours.[64] Also, because of the communicable nature of bacterial infections, active lesions cannot be covered to allow participation.[64]

CONCLUSIONS

Certified ATs and other athletic team health care providers must be able to identify the signs and symptoms of common skin diseases in athletes. This position statement outlines the current recommendations to educate the stakeholders in their athletic programs about minimizing disease transmission, preventing the spread of infectious agents, and improving the recognition and management of common skin diseases in athletes.

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DISCLAIMER

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