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The Identification and Treatment of Common Skin Infections

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Skin conditions are a common problem addressed by medical providers. Up to 25% of individuals in the United States will seek attention for these conditions each year. The same problem occurs in the athletic training room, where athletes with infectious skin conditions can be seen. Most conditions are simple and can be treated without concern for spread to susceptible athletes. However, others can be quite serious and spread rapidly through a team and opponents during competition. Knowledge of the different types of skin infections is necessary to help treat these athletes and prevent spread to others. With proper diagnosis and treatment, certified athletic trainers can keep the athlete off the field of play for a minimum period and prevent transmission.

Key Words: Folliculitis, impetigo, abscess, herpes, tinea, molluscum contagiosum, wrestling, contact sports

Key Points

- Skin conditions are a common problem dealt with by athletic trainers.
- Knowing when and why to withhold an athlete from practice and competition is crucial for the individual, let alone teammates.
- Not all sports require an infected athlete to be withdrawn from participation.

An estimated 1 in 4 individuals in the United States will see a health care provider (HCP) for skin diseases each year.[1] The problem with skin-related infections in the athletic venue is no different than in the general population. Early recognition and appropriate treatment are key for quick recovery and safe return to sport. The 2010 National Athletic Trainers' Association position statement on skin diseases[2] addressed prevention, recognition, and management. Here, we focused on recognition of these diseases and updated treatment options. Most skin infections in athletes affect those involved in wrestling and other contact sports, and a better understanding of their implications prepares the athletic trainer (AT) for managing them. In wrestling, over the past 25 years, sporadic outbreaks of primary herpes gladiatorum have occurred, but recognition of this disease by HCPs has been as low as 8.3% on initial evaluation.[3] Ashack et al[4] analyzed skin infections among US high school (HS) athletes and showed a preponderance of infections in wrestling, with bacterial infections estimated at 53.8% and herpes at 6.7%. A review of infections observed from 1996 to 2022 at the USA Wrestling U16 and U18 National Tournament in Fargo, North Dakota, trended differently. Unless providers were well acquainted with the sport, they overestimated bacterial infections, a common mistake.[3] The prevention of transmission is essential and may require the athlete to be withheld from practice, competition, or both until culture results are available, though coaches and parents who are eager to see the athlete participate may be reluctant to hear this (Candice Williams, MD, MPH, of the Centers for Disease Control and Prevention; written communication, June 3, 2015). During a 2014 outbreak of herpes gladiatorum on an HS wrestling team in Arizona, Dr Williams also believed that bacterial infections were overestimated at the expense of a herpes diagnosis.

The proper recognition of skin infections requires knowledge of each sport and the habits and interests of the individual athlete. As of 2019, almost 250 000 male and 21 000 female US HS athletes engaged in wrestling.[5] About 5% to 10% of these athletes go on to participate in collegiate programs. For women, these numbers are increasing, making wrestling the fastest growing sport in HS and college.[6] Wrestling, football, and hockey athletes have a higher risk of skin infections due to shared equipment and direct skin-to-skin contact.[4] Abrasions, cuts, and open wounds allow easy access of entry for pathogens. The transmission of dermatophytes (eg, ringworm) is primarily via skin-to-skin contact but may also result from contaminated surfaces and equipment. Zoonotic infections can affect individuals who work as butchers, veterinarians, hunters, or work on farms around pets and farm animals. The direct interaction with and handling of these animals allows pathogens to transfer to the human host. The animals are known to harbor

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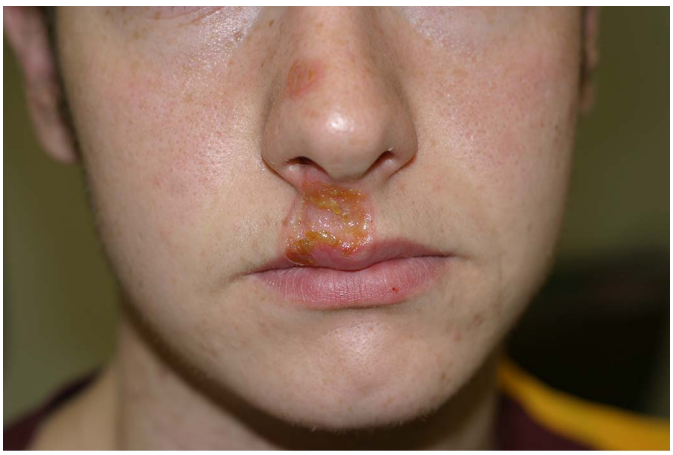

Figure 1. Impetigo. Weeping and crustiness are key features.

ringworm and community-associated methicillin-resistant *Staphylococcus aureus* (CA-MRSA) and, thus, carry the risk of exposing a person involved in such activities.

Athletic trainers who are familiar with these facts are in a unique position to recognize skin infections in athletes they treat both on the field and in the athletic training facility. Transmission-prevention guidelines are generic for all activities, but contact sports such as wrestling, football, and hockey place athletes at a higher risk for skin infections. Therefore, appropriate guidelines to prevent transmission and treat affected athletes are of the utmost importance. Recommendations for wrestling can be found on the National Federation of State High School Associations (NFHS; https://www.nfhs.org/articles/prevention-key-to-reducing-skin-infections-in-high-school-wrestling), National Collegiate Athletic Association (NCAA; https://www.ncaapublications.com/p-4141-sports-medicine-handbook.aspx), and USA Wrestling (https://www.teamusa.org/USA-Wrestling/Features/2018/December/06/How-To-Maintain-Proper-Hygiene) websites. Advice for other sports is available at the NCAA, NFHS, and state HS sports association websites. To assist readers in determining if treatment options are appropriate, we will provide strength of recommendation (SOR) taxonomy ratings.[7]

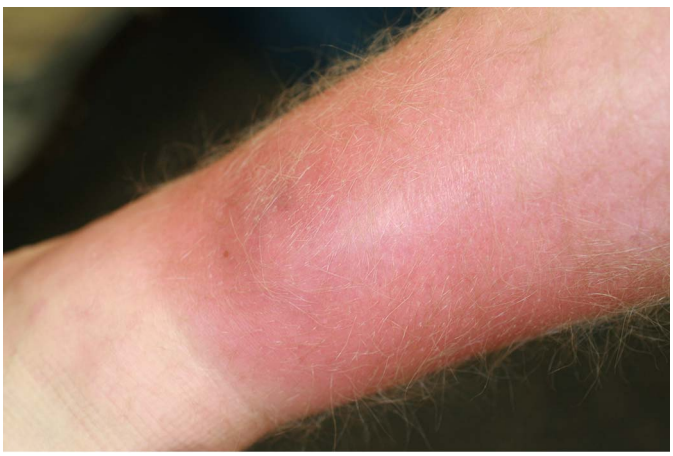

Figure 2. Cellulitis. Warm, red, and firm skin defines this condition.

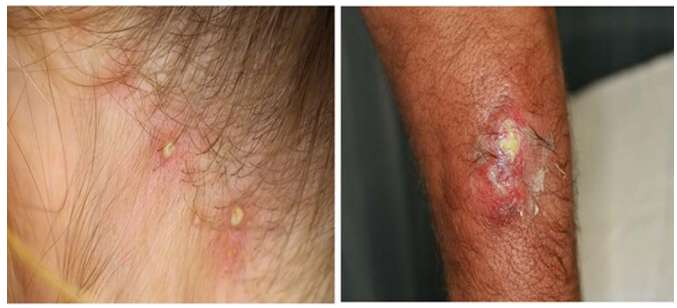

Figure 3. Folliculitis and abscess. An individual pustule at the base of the hair follicle is classic for folliculitis. An abscess has a reddened area with a fluctuant site with eventual drainage of necrotic material.

Grading criteria are as follows (SOR definition):

(A) Recommendation is based on consistent evidence for which most studies drew similar conclusions or high-quality patient-oriented evidence.
(B) Recommendation is based on inconsistent findings in which research lacked consensus or evidence was limited.
(C) Recommendations are based on expert opinion, case studies, or extremely limited evidence.

BACTERIAL INFECTIONS

The most common skin infections in HS athletes are bacterial.[4] *Staphylococcus* and *Streptococcus* species are responsible for the majority of these infections and can lead to conditions such as impetigo, erysipelas, cellulitis, folliculitis, and abscesses.

Recognition: Bacterial Infections

Among bacterial conditions, impetigo is the most common. Bacteria infect the superficial aspect of the skin and can cause blistering, open lesions, and honey crusting (Figure 1). Cuts or abrasions serve as the portal of entry into the skin. As the skin becomes more deeply infected, cellulitis or erysipelas can evolve. The skin then becomes red, warm, firm, and tender (Figure 2). Folliculitis occurs when a hair follicle is involved and pus accumulates at its base (Figure 3). Finally, with deeper dermal involvement, abscess formation can become more extensive. Redness, warmth, firmness, and pain accompany a fluctuant mass of purulent necrotic tissue (Figure 3).

Most staphylococcal and streptococcal infections are managed with oral or topical antibiotics. Unfortunately, CA-MRSA has complicated empiric antibiotic choices in recent decades.[8,9] Rates of CA-MRSA skin infection are increasing,[10,11] and no reliably distinguishing features were found when compared with its more benign counterpart, methicillin-sensitive *Staphylococcus aureus* (MSSA).[12] Culturing the lesion can help differentiate between these pathogens and guide the HCP on the proper antibiotic choice.

Incidence and Prevalence Data

Researchers have suggested that incidence rates of CA-MRSA infection among HS football players and wrestlers are between 0.6% and 1.0% for a single season.[10] Contact sports have been reported to be responsible for as many as

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Table. Treatment Guidelines for Skin and Soft Tissue Infections in Adolescents and Adults

Type of Infection	Organism	Treatment (5–7 d)	Differential Diagnosis	Return to Play (After Treatment)
Bacterial				
Cellulitis, folliculitis, carbuncle, furuncle	*Staphylococcus aureus*, *Streptococcus pyogenes*	Cephalexin 250 mg 4×/d; clindamycin 300 mg 4×/d[a]; dicloxacillin 250 mg 4×/d	Dermatitis, eczema, cellulitis, folliculitis, carbuncle/furuncle, hidradenitis suppurativa, tinea, herpes	72 h
Impetigo	Community-associated methicillin-resistant *Staphylococcus aureus*	Trimethoprim-sulfamethoxazole 800/160 mg 2×/d[b]; minocycline 100 mg 2×/d[b]; clindamycin 300 mg 4×/d[b]; linezolide 600 mg 2×/d[b]; amoxicillin/clavulanate 875/1235 mg 2×/d; retapamulin 1% ointment 2×/d; mupirocin 2% ointment 3×/d	Herpes, varicella, tinea, eczema	
Fungal				
Tinea (corporis, capitis, cruris, pedis)	*Trichophyton tonsurans*, *T. rubrum*, *T. metagrophytes*	Terbinafine 250 mg/d; fluconazole 150 mg 1×/wk; itraconazole 100 mg/d; topical: terbinafine 1%; butenafine 1%; econazole 1% cream; ketoconazole 2% shampoo 2–3×/wk	Seborrheic dermatitis, psoriasis, granuloma annulare	Immediate with covering, except on scalp, which requires 14 d
Kerion	*T. metagrophytes, T. rubrum*	Terbinafine 250 mg/d[c]	Carbuncle, furuncle	14 d
Viral				
Herpes	Herpes simplex	Primary (7–10 d): Acyclovir 200 mg every 5 h[d] Valacyclovir 1 g 2×/d Recurrent (7 d): Acyclovir 200 mg every 5 h[d] Valacyclovir 500 mg 2×/day Prophylaxis for recurrent outbreaks: Valacyclovir 1 g daily (if outbreaks <2 y) Valacyclovir 500 mg daily (if outbreaks >2 y)		
Molluscum contagiosum	Poxviridae	Curettage and hyfrecator, topical imiquimod 5%	Keratoacanthoma	Immediate with covering

[a] Preferred treatment with community-associated methicillin-resistant *S. aureus*, but trimethoprim-sulfamethoxazole will not cover *S. pyogenes*.
[b] Consider 7–10 days of treatment and be sure to check local biograms for sensitivity.
[c] Requires 6 weeks for kerion.
[d] To be bioequivalent with valacyclovir, acyclovir must be given every 5 hours.

90% of CA-MRSA infections in athletes.[10] Infection in athletes most often occurs after direct contact with an infected or colonized athlete.[12,13] *Colonization* is a dynamic process in which an individual harbors bacteria without displaying clinical evidence of infection. The nares are the most common site: up to 30% of us carry MSSA and 1.5% to 3% carry CA-MRSA.[14] About 6% of athletes were colonized with CA-MRSA, and they were 7 times more likely to contract a bacterial skin infection than those who were not colonized.[14] Collegiate wrestlers demonstrated the highest carrier rates of CA-MRSA, as many as 76% of all cases.[15] Although CA-MRSA is prone to recur, no rigorous studies on infection recurrence rates have been conducted in athletes. Yet outpatient studies in the general population showed a 51% chance of reinfection with CA-MRSA within 6 months of the initial infection.[16]

Treatment: Bacterial Infections

The treatment of bacterial skin infections in athletes typically involves both oral and topical antibiotics (Table). In athletes with extensive skin involvement or systemic symptoms, such as fevers, chills, night sweats, and altered mental status, intravenous antibiotics should be used.[12,17,18] Administration of intravenous antibiotics is typically done in the hospital setting. When prescribing antibiotics, the HCP must keep local resistance patterns in mind. In general, contact-sport athletes can be empirically treated with oral trimethoprim-sulfamethoxazole or clindamycin

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antibiotics as well as topical mupirocin. This regimen will cover most staphylococcal and streptococcal infections. If possible, skin infections should be cultured to ensure treatment with antibiotics to which the bacteria are susceptible.[12,20] If an infection appears purulent, incision and drainage should be performed and culturing should be considered.[18] Localized purulent skin infections that are treated with incision and drainage do not necessarily require antibiotic therapy.[12,18] (*SOR: B*)

Impetigo can be treated with 2% mupirocin ointment daily along with oral clindamycin, cephalexin, or dicloxacillin for 7 to 14 days. Treatment of MSSA infections, such as erysipelas, cellulitis, and folliculitis, is with 250 mg of cephalexin or dicloxacillin for the same length of time (Table 1). For MRSA infections, treatment is with 2% mupirocin ointment plus either double-strength trimethoprim-sulfamethoxazole (160 mg–800 mg) or clindamycin for 7 to 14 days. If >15% to 20% local resistance to trimethoprim-sulfamethoxazole or clindamycin is present, linezolid can be used instead. For all abscesses, incision and drainage is recommended, regardless of whether antibiotics are prescribed. (*SOR: B*)

Decolonization

Decolonization is the process of attempting to remove CA-MRSA from the body surface. A lack of consensus persists regarding the effectiveness of the decolonization of athletes who have had multiple CA-MRSA infections or in settings with a CA-MRSA outbreak. Those who undergo decolonization typically follow a 5-day regimen of showering daily with 4% chlorohexidine gluconate and applying 2% mupirocin topical ointment twice a day to each nare. The authors of 1 study indicated that mupirocin decreased the prevalence of skin infections among patients in long-term care facilities.[20] Other researchers have determined that decolonization efforts can be successful; however, evidence in athletic populations remains limited.[12,21–25] Recent investigators identified successful decolonization with oral antibiotics in a cohort that included patients who failed to achieve topical decolonization with chlorhexidine and mupirocin.[25] Despite these limited data, attempting decolonization during a CA-MRSA outbreak is reasonable and may prevent further infection in outbreak settings.[18,26,27] (*SOR: C*)

The NCAA and NFHS have similar guidelines for sport participation in the presence of bacterial skin infections. For athletes to regain eligibility for competition after a bacterial infection, NCAA and NFHS require that no new lesions appear over a 48-hour time span; that antibiotic therapy be initiated 72+ hours before participation; and the absence of moist, exudative, or draining lesions.[27,28]

The NCAA further clarified that covering active infections does not allow the athlete to participate in competition. Additionally, lesions in question may be Gram stained for further diagnostic clarification and for the determination of an athlete's ability to participate in competition.[28]

FUNGAL INFECTIONS

Fungal skin infections are the second most common skin infection in HS athletes.[4] Whether they involve the scalp (*tinea capitis*), body (*tinea corporis*), groin (*tinea cruris*), feet (*tinea pedis*), or nails (*onychomycosis*), they are difficult conditions to treat. The dermatophytes involved

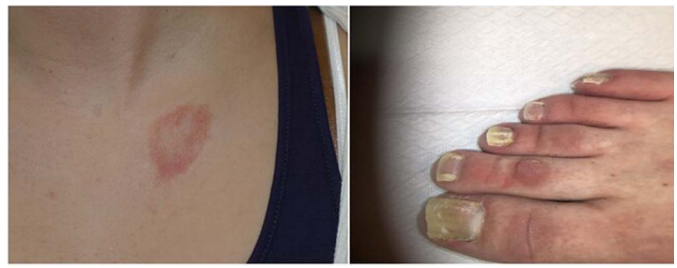

Figure 4. Tinea corporis and onychomycosis. The red, flaky area expands circumferentially. When it involves the toenails, it represents onychomycosis.

are *Trichophyton rubrum*, *Trichophyton tonsurans*, and *Microspororum canis*, with *T. tonsurans* causing most of the skin mycoses and being responsible for 90% of tinea capitis seen in urban areas.[29] From 1997 through 2006 at the Minnesota State High School wrestling tournament, tinea was estimated as accounting for >70% of the presenting skin infections.[30] Multiple screening examinations have revealed that 19.2% to 40% of participants in wrestling and combat sports showed signs of this infection each season.[31,32]

Recognition: Fungal Infections

Asymptomatic combat-sport athletes may carry *T. tonsurans* in their scalps.[31] With an incubation period of 1 to 3 weeks, tinea corporis usually develops within 3 to 5 days after direct skin-to-skin contact with an opponent who has an active infection or is an asymptomatic carrier. Those who have had contact with animals can also serve as vectors. The infection can also be transmitted via fomite contact where spores are present. Tinea presents as a small, erythematous, vesiculobullous lesion that grows in a circumferential pattern (Figure 4). Distinguishing features are a slightly red, flaky perimeter with central clearing and definitive expansion in a circular fashion. Single lesions can reach 8 to 10 cm in diameter. However, the athlete will generally seek treatment before the lesion reaches that size.

In most cases, tinea infections are superficial, with no deeper skin structures involved. Tinea pedis is the most frequent type and develops due to swimming pool exposure, sweat, or wearing tight socks or shoes in warm weather, yet the dermatophyte grows more readily in tissues with high fat content. In fact, involvement of the scalp[31,33] and especially the hair has been documented in asymptomatic carriers. These carriers add to the major spread of outbreaks on teams or clubs when no signs are visible.[31,33] When the condition becomes more invasive, the scalp is usually involved. Deeper tissue involvement may incite granulomatous development, especially around the hair follicles on the scalp. Pustules weeping serosanguinous fluid might also be seen, and regional lymphadenopathy may develop. With continuation, hair loss may ensue, leading to classic kerion formation.

Trichophyton tonsurans is considered an *anthropophilic species* (ie, specific to humans).[29] However, tinea outbreaks have been documented in which a different dermatophyte was spread from close contact with infected animals.[29,34] The transfer of tinea via animal-to-human contact in animal

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husbandry is well documented and could also contribute to its occurrence.

Tinea develops primarily on exposed areas of the skin, typically the extremities. Most infections can easily be treated with topical agents, but oral agents must be used for scalp or multiple skin lesions. The appropriate duration of treatment in a contact-sport athlete has always been disputed, with attempts to balance the desire to return the athlete to competition with the need for sufficient treatment to eradicate the fungus. Kohl et al[35] found culturable fungus present up to 21 days after patients started oral antifungal medication. This concern is further compounded when the feet or nails are involved. Onychomycosis is fungal involvement of the nails, with the toes being primarily affected. The nails may have a yellow-gray discoloration that starts at the distal end of the nail and progresses toward the base. As the nail thickens, it may become brittle and break or flake off. The fungus can spread to the soft tissue, leading to red flakiness of the toes and feet. With whole foot involvement, the infection appears in a classic moccasin distribution (Figure 4) and spores can spread to the athlete's undergarments when dressing. This infection can then spread to the groin, leading to tinea cruris.

Treatment: Fungal Infections

Topical and oral antifungal medications are the treatments of choice for fungal infections (Table). Topical medications must always be used, but deciding when to start oral medication is at the discretion of the HCP. Treatment can be lengthy, and failure is usually due to poor compliance of the athlete. Single lesions may benefit from topical agents, yet numerous lesions and those involving the face or scalp usually require oral medication. Other treatments may include antifungal powders, primarily for use on the feet, and topical shampoos, for use on the scalp. These agents are generally used in conjunction with creams and oral medications to augment their effectiveness. The length of treatment can vary from 14 days to 6 to 12 weeks, depending on the site. For simple skin lesions, clinicians should treat the athlete until the lesion is gone, plus an additional week. (*SOR: B*)

Athletes may return to practice and competition provided the lesion is being treated and covered with a gas-permeable membrane. With scalp involvement, certain contact sports require 14 days of oral treatment before return to play. The NCAA, NFHS, and state HS associations have sport-specific guidelines.

VIRAL INFECTIONS

Herpes infections encompass several types that can affect athletes. Chicken pox, or varicella, usually presents as a systemic infection that is spread via respiratory droplets. This infection progresses to fever, lymphadenopathy, and a generalized vesiculopapular rash over the body. Since the development of vaccinations, this infection occurs more rarely. In contrast, herpes simplex infections are more common, especially in contact-sport athletes. Their symptoms are more localized and with head, neck, and face involvement, a sore throat can also occur. Early recognition is crucial to reduce the risk of spread. In the context of a contact sport, strong suspicion is necessary. Herpes simplex virus (HSV) types 1 and 2 are the usual agents transferred in these activities, with HSV-1 accounting for 94% to 97% of infections.[36] The HSV-1 infection can affect any skin area of the body but in wrestlers tends to involve the face. A serious concern is ocular involvement, which can result in corneal scarring and rarely retinal necrosis and blindness.[37]

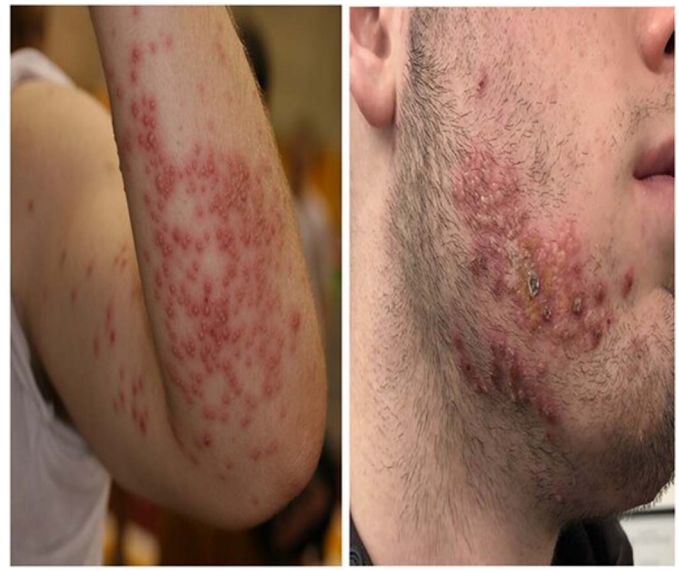

Figure 5. Primary herpes. Extensive involvement seen with a primary outbreak.

Herpes virus is transferred via direct skin-to-skin contact. The prevalence is estimated to be no different in competitors versus noncompetitors, but the location of outbreaks differs in the 2 groups.[36] Among noncompetitors, *herpes labialis* involves a primary outbreak usually of the oral-nasal region. Among wrestlers, *herpes gladiatorum* is primarily seen in areas of the greatest skin-to-skin contact: >70% of the time on the head, face, and neck.[38]

Recognition: Herpes Infections

Primary herpes (Figure 5) typically presents with systemic involvement and diffuse patches of 2-mm vesicles coalesced into groupings of 3 to 10 lesions. Erythema and warmth surrounding the lesions are common and usually last 10 to 14 days, during which multiple sites and dermatomes can be involved. As the virus progresses along the sensory neuron, ganglions become infiltrated. Eventually, a ganglion becomes infected, and the virus establishes latency. Significant regional lymphadenopathy and multiple-site involvement are often seen.

Recurrent outbreaks (Figure 6) usually produce fewer vesicles, and the outbreaks are smaller and shorter in duration. A single ganglion is the source of latency, reactivation, and recurrences, usually lasting 5 to 7 days, and the infection always develops along that ganglion's dermatomal pattern.

Treatment: Herpes Infections

Treatment helps to expedite infection clearance but never eradicates the virus from the nerve ganglion. Outbreaks are usually triggered by ultraviolet light exposure (ie, sunlight), cutting weight, rubbing of the

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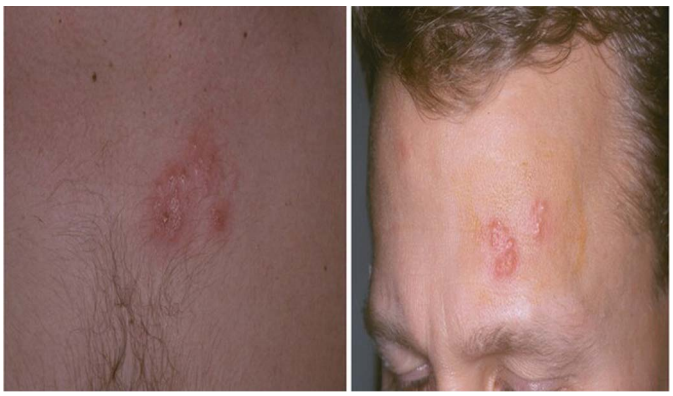

Figure 6. Recurrent herpes. Smaller, localized, and recurrent lesions define this condition.

area, or stress. Athletes can return to competition once no new vesicles have formed, the eschar is well adhered, and no regional adenopathy is present. Treatment shortens the time to clearance, but these criteria must be met to reduce the risk of transmission to an opponent (Table). (*SOR: B*)

Recurrent outbreaks require a lower treatment dosage and shorter duration than a primary outbreak. The US Food and Drug Administration–approved treatment of recurrent herpes labialis is 2 g of valacyclovir twice a day for 1 day. Although this regimen serves the patient well in alleviating the symptoms of an outbreak, it does not reduce the risk of transmission. This fact is important when addressing its use in athletes who are in contact versus noncontact sports. For contact-sport athletes, after 120 hours of treatment, the athlete may return to practice and competition provided the outbreak has cleared. No regional lymph nodes should be swollen, no new vesicles should have appeared for the past 48 hours, and all lesions should have well-adherent eschar.

The prevalence of HSV-1 is greater than athletes realize. Estimates of prevalence in HS wrestlers differ significantly from those who know they are infected. Only 3.3% realized they had a recurrence, while 29.8% were carrying the virus.[36] Because the virus can be transmitted before vesicles form, these athletes need to recognize they may be shedding virus and transmitting it to susceptible opponents. Asymptomatic shedding of the virus is a significant source of transmission, with estimates that 1% to 5% of seropositive children and adults asymptomatically shed HSV-1.[39] Once an outbreak occurs in a competitor, the risk of transmission to an opponent via practice or competition may be up to 30%.[38] Therefore, in some contact sports, yearly antibody testing for HSV-1 should be considered for athletes with no history of herpes. Those with a known history of herpes or positive antibody results should take prophylactic antiviral medication throughout the competitive season. We have found that 1 g of valacyclovir daily reduced recurrent outbreaks in wrestlers to 7.7% compared with placebo at 24.1%.[40] (*SOR: B*)

The preventive use of valacyclovir in HSV-naive wrestlers may reduce the risk of acquisition. A 10-year study evaluating the effectiveness of 1 g of valacyclovir daily at a 28-day wrestling camp (2792 athletes) demonstrated a reduction in outbreaks and the clinical acquisition of this virus.[41] Applicable situations include the protection of HSV-naive wrestlers who enter tournament play at the end of the season. Exposure at conference championships is quite common, with primary outbreaks occurring up to 10 days later. Preventing these incidences may help guarantee that all athletes can compete at state and national championships and not be held out due to a new herpes gladiatorum outbreak. (*SOR: C*)

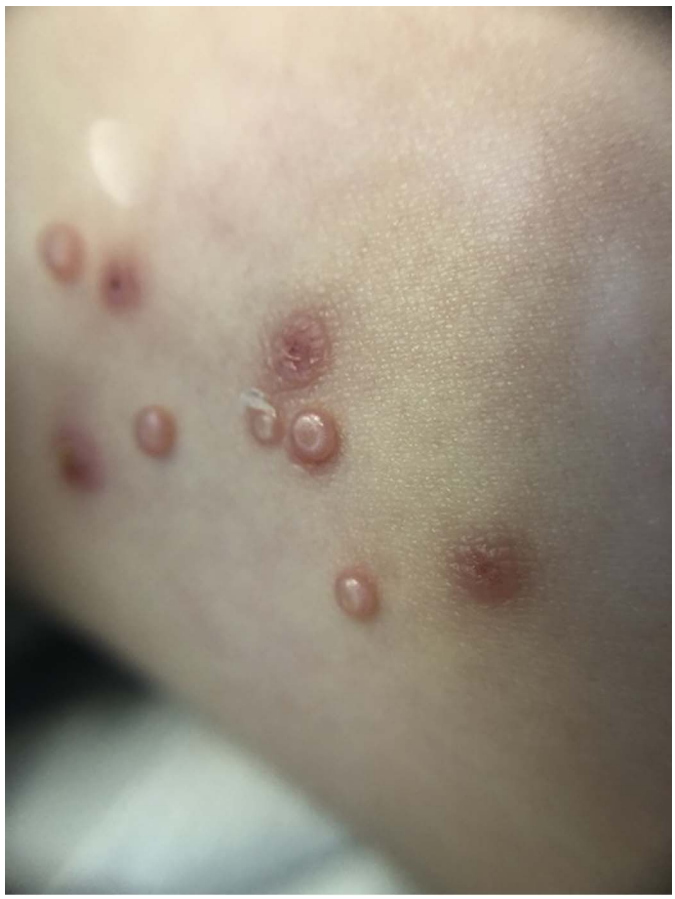

Figure 7. Molluscum contagiosum. Small papules are diffuse, with a dimpled center and minimal surrounding redness.

OTHER VIRAL INFECTIONS

Molluscum contagiosum is another troublesome infection, derived from the Poxviridae family. Typically spread via direct skin-to-skin contact, it presents as 2- to 10-mm dome-shaped papules, often with a central depression (Figure 7). No surrounding erythema is present, and the singular lesions appear in a diffuse pattern. Among sexually active individuals, the lesions affect the groin; among wrestlers and young children, they affect the neck, chest, axilla, arms, and abdomen. Treatment focuses on use of a curette and hyfrecator for rapid removal. Imiquimod cream (5%) applied topically is an alternative but requires up to 12 to 16 weeks for resolution (Table).[42,43] With facial involvement, appropriate treatment is needed to minimize scarring or vitiligo formation. Again, treatment is recommended before participation in contact sports. Return to competition can be immediate after treatment, provided the areas are properly covered with a gas-permeable membrane agent. (*SOR: B*)

Verrucous warts are caused by papillomaviruses. They are primarily noted on keratinized surfaces of the hands and feet but can also be seen on the elbows and knees. Common in childhood, they can progress for many years and then spontaneously disappear with age. The firm lesions are 1 to

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10 mm or more in size and may coalesce or be diffuse. They typically grow in an exophytic, vegetative fashion. Due to the speed of lesion development, treatment should be performed to expedite their clearance. Cryotherapy is effective but requires several months for eradication. An alternative is 5% topical imiquimod cream applied nightly[42,44,45] (Table 1). This also requires a lengthy regimen of 12 to 16 weeks to be effective. For the athlete to continue participating in practice and competition, the warts must be covered with a gas-permeable membrane to prevent their spread until the virus resolves. (*SOR: B*)

Outbreak Control

For bacterial infections, the Centers for Disease Control and Prevention has issued evidence-based guidelines on preventive measures for athletes and athletic facilities. These guidelines recommend that athletes cleanse all wounds with soap and water and cover them with appropriate dressings. Athletes should also maintain good personal hygiene and avoid sharing towels and personal items. Coaches should establish routine cleaning schedules for shared equipment, train athletes in recognizing infections, and deliver appropriate first aid for wounds that may be infected. Finally, coaches should assess athletes regularly for skin lesions, and if any develop, they should be referred to an HCP for treatment.[46] Limited evidence showed that formal infection-control programs and hygiene education were effective in decreasing the bacterial burden in the athletic training facility.[47] Preventive guidelines, infection-control protocols, and hygiene education have been enforced and emphasized in outbreak settings as a means of controlling the spread of infection.[26] Clinical and environmental samples are required to fully investigate a setting for a specific point source of contamination leading to an infection outbreak. Once a point source is identified, changes can be recommended to limit infectious spread and prevent future outbreaks.[12,26]

For herpes outbreaks, guidelines focus on preventing skin-to-skin contact during times of active infection. In wrestlers, skin checks can identify infected individuals with visible lesions. However, during outbreaks involving several team members, transmission can occur up to 2 to 3 days before vesicle formation. In a close contact sport, daily skin checks may not be sufficient to prevent transmission. In these situations, shutting down the sport for 8 days should be considered to allow those who have been exposed to determine if they have become infected.[47] The athlete can return to play once the infection has cleared. Use of antiviral medication can expedite that process and, in certain situations, can be used prophylactically to reduce the risk of recurrence during the season (Table).[40] (*SOR: B*)

Return to Play

Treating athletes who have skin infections is necessary for the individual's general health, but not all infections should keep the athlete out of practice and competition. These pathogens are usually transmitted via skin-to-skin contact, but equipment and surfaces may also be sources. Determining when to remove an athlete from practice and competition because of a skin condition should first be based on the individual athlete's health and then on the risk of transmission to another athlete. A tinea infection in a tennis player carries much less risk than in a wrestler who has constant contact with an opponent. Using guidance from the NCAA and NFHS, ATs should apply appropriate discretion when making this decision.

Skin infections continue to be a significant concern in both the public and athletic arenas. It is necessary to think "outside the box" when evaluating patients and determining the cause of the infection. Only by doing so will the AT properly diagnose athletes with these conditions and implement the proper interventions to keep them under control and eradicate them.

Additional information pertaining to pityriasis rosea and eczema are available via the Supplemental material.

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SUPPLEMENTAL MATERIAL

Supplemental Figure 1.

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