

2. Viral Skin Infections

This second chapter on sports-related skin infections summarizes the aspects of viral diseases as they pertain to athletes. Technically smallpox has been eradicated from the world, so the case of smallpox in a boxer in 1948 is not discussed in detail but is mentioned here for historical perspective. A Norwegian boxer developed the pustular eruption of smallpox on his head, neck, and right arm after he came in contact with an English competitor's contaminated boxing gloves. This case probably is one of the first reported skin infections in athletes.

Athletes with viral infections are particularly susceptible to pain, loss of practice time, and disqualification. Of all dermatologic conditions occurring in the athlete, viral infections, specifically herpes simplex infection, are among the most likely to cause morbidity, intense worry, and significant team disruption. In some cases, the virus spreads by skin-to-skin contact, whereas other times the virus is transmitted to the skin from inanimate objects. Even without contact, the athlete may develop recurrent herpes virus infection upon exposure to the elements.

Early identification, rapid treatment, and ultimately prevention are essential activities for clinicians in order to decrease disruption in practices and competitions. Epidemics can also be thwarted. This chapter specifically addresses herpes simplex infection occurring in noncontact and contact sports, verrucae (warts), and molluscum contagiosum.

Herpes Labialis (Noncontact Sports)

Epidemiology

Exposure to prolonged ultraviolet (UV) irradiation increases the risk of reactivating prior herpes simplex virus (HSV) infection. One study determined that the sun induced HSV in approximately 20% of the population during the summer months and in up to 40% of patients younger than 30 years (Ichihashi, 2004). Thus, all outdoor athletes who fail to protect their lips are at potential risk for developing herpes labialis. A few studies that examined the epidemiology of HSV concentrated on skiing. Twelve percent of skiers with a known history of HSV developed lesions during 1 week of skiing (Mills et al., 1987).

Several studies determined the amount of UV irradiation to which outdoor athletes are exposed is several times the amount needed to induce a burn. Winter athletes whose sport occurs in the snow and at high altitudes experience an even higher risk. Snow reflects a large amount of UV irradiation, thus multiplying the level of UV exposure to winter enthusiasts. Furthermore, athletes at high elevation must contend with more intense irradiation because less UV is filtered out by the atmosphere. One study showed that a skier at 11,000 feet in Vail,

Colorado, received the same intense UV B radiation as a beachgoer in Orlando, Florida. Other at-risk athletes include snowboarders, snowmobilers, tobog-
ganers, and athletes participating in other outdoor sports.

Scuba divers suffer from herpes labialis for other reason (Potasman and Pick, 1997). During drills that simulate emergency situations, divers may frequently share mouthpieces of the pressurized air regulator, and a great deal of saliva tends to develop around the mouthpiece. Because these transitions among divers occur in less than 3 seconds, HSV likely survives on the mouth-
piece during this brief time. Athletes who share protective mouthpieces are at risk for acquiring herpes labialis from other athletes with active infection (Table 2-1). Herpes simplex virus type 1 (HSV-1) causes most cases of oral herpes labialis.

Clinical Presentation

Athletes with herpes labialis (primary infection or reactivation) often com-
plain of a stinging or burning sensation in the area that eventually develops skin lesions (so-called *prodrome*). After 1 to 4 days, a painful erythematous papule develops in the same location as the prodrome. In one study, the median time to lesion development after a person arrived to ski was 3.5 days (Mills et al., 1987). The papule soon develops grouped vesicles upon an erythematous base. After several days, the lesion becomes crusted and resolves slowly (Figure 2-1). Sys-
temic findings are not unusual in the setting of oral herpes labialis, especially in the primary form. Athletes may suffer sore throat, fever, myalgias, arthralgias, and lymphadenopathy.

Diagnosis

The diagnosis often is straightforward and can be made based on clinical findings alone. In indeterminate cases, Tzanck smear, direct immunofluores-
cence (DFA), or culture may be necessary. Tzanck smear provides the most rapid result but can be technically challenging even for seasoned dermatologists. Direct immunofluorescence provides rapid results (often within just a few

Table 2-1. Evidence-Based Support for Viral Infection Epidemics

Viral Infection	Associated Sport or Activity
Herpes labialis	Skiing, scuba diving
Herpes simplex	Rugby, wrestling
Molluscum	Cross-country running, swimming
Verrucae	Rowing, swimming, showering



Figure 2-1. Typical herpes labialis lesion that might be seen in an outdoor athlete exposed to intense ultraviolet radiation (e.g., a skier).

hours), but the test is not universally available at all laboratories. Culture is the gold standard for identifying the presence of HSV infection, but the culture may take 1 to 2 days to become positive.

Treatment

Because most of the infections resulting from participation in noncontact sports actually are reactivated herpes labialis, the dosing regimen for this type of infection is most appropriate (Table 2-2). Oral acyclovir 200 mg can be given

Table 2-2. Dosing Regimens for Treatment of Recurrent Herpes Labialis, Herpes Gladiatorum, or Herpes Rugbeiorum

Medication	Dose (mg)	Frequency	Duration (days)	Cost
Acyclovir	200	5 times/day	5	Low
Famciclovir	500	bid	5	High
Valacyclovir	2000	bid	1	Medium

five times per day for 5 days. More expensive but easier dosing regimens, which likely translate into increased compliance, include famciclovir and valacyclovir. The dose of oral famciclovir is 500 mg twice per day for 5 days. A study revealed that an effective dosage of valacyclovir is 2 g twice per day for only 1 day. This simple dosing regimen is as effective as the other regimens and likely will result in high athlete compliance.

Prevention

Athletes with active herpes labialis are clearly contagious. Unfortunately, athletes may shed the virus asymptomatically and unwittingly transmit the HSV to other athletes. Under no circumstance other than emergent should infected athletes share protective mouthpieces. Scuba divers, who may be required to share mouthpieces in emergency situations or during practice drills, must be excluded from diving if they have active herpes labialis.

Skiers, snowboarders, snowmobilers, tobogganers, and other winter outdoor enthusiasts must protect their lips with sunscreen. Many commercially available lip balms with sun protective factor (SPF) are available. The balms are compact and are easily carried in pockets. Sweating decreases the effectiveness of sunscreen, so it must be reapplied frequently. A double-blind, placebo-controlled study demonstrated that, after UV exposure to the lips, 71% of unprotected individuals developed HSV lesions, whereas none of those who wore sunscreen developed lesions (Rooney et al., 1991).

Pharmacologic therapy for herpes labialis has been studied in skiers. Skiers who took acyclovir 400 mg twice per day beginning 12 hours before anticipated sun exposure were significantly less likely to develop HSV lesions (Spruance et al., 1988).

Herpes Simplex (Contact Sports)

Epidemiology

All athletes participating in sports with close, intense skin-to-skin contact are susceptible to acquiring HSV infection (otherwise known as *fever blisters*) from other athletes. The two most studied athletes are rugby players and wrestlers, although on rare occasions lesions are noted in soccer players during heading maneuvers (Table 2-1). Macerated skin from sweating and traumatic skin injuries inherent to both wrestling and rugby facilitate infection by HSV.

Rugby

Rugby players have prolonged and concentrated skin-to-skin contact during the act of the scrum, wherein the players link arms, encircle the ball, and attempt to take possession of the ball. Members of the scrum have abrasive contact with each other for 30 to 45 seconds repeatedly during games. *Herpes rugbeiorum* (HR) (or *scrum pox*) is the term designated for HSV infection in rugby players. The epidemiology of HR is much less studied than that of herpes gladiatorum (HG), but epidemics clearly can occur among rugby players. Interestingly, the forwards (who are members of the scrum) are the players whose position places them at greatest risk for developing HR. Other position players may become infected but are less likely to because of relatively less skin-to-skin contact. On one team, four of the eight rugby forwards were infected (the left and right prop, the lock, and one of the second row) (White and Grant-Kels, 1984). Another study in England demonstrated that nearly half of the rugby clubs had skin disease, and 95% of the infected players were forwards (Shute et al., 1979). Another study demonstrated that 38% of a rugby team developed HR (Skinner et al., 1996).

Wrestling

Herpes gladiatorum is the term designated for HSV infection in wrestlers. Wrestlers have arguably the most intense skin-to-skin contact during matches. Most clinicians accept the term herpes gladiatorum; however, some authors disapprove because of the term's incorrect etymology (Laur et al., 1979). They indicate that gladiators are distinctly different from wrestlers and suggest incorporating the Latin term for wrestler, preferring the term *herpes luctator*. The literature is replete with the HG designation, however, so the condition is henceforth described in this book as herpes gladiatorum.

The epidemiology of HG probably is the best studied of all cutaneous sports ailments. Seventeen completed prevalence studies have reported a wide spectrum of results. The lowest reported prevalence is 2.6% and the highest is 40.5% (Becker et al., 1988; Belongia et al., 1991; Brenner et al., 1994; Dyke et al., 1965; Porter and Baughman, 1965; Wheeler and Cabaniss, 1965). The median prevalence (which likely represents the true estimate) is 20%. The large range reflects the dissimilar manner in which the studies were performed. For instance, the lowest prevalence of 2.6% was determined in a study of athletic trainers who were asked to recall the number of high school and college athletes who had been infected. Because of recall bias, selection bias, and the very small fractional survey return rate, the true prevalence likely was underestimated. Based on data from the National Collegiate Athletic Association injury surveillance system (NCAAISS), 39% of all skin infections in collegiate wrestlers between 1991 and 2003 were caused by HSV. Shingles (another *Herpes* virus) composed 3% of the skin infections.

An interesting HSV exposure method involves an injured athlete whose need to use the whirlpool may place the athlete at risk for developing HSV. One study

showed that HSV may survive on plastic spa surfaces at temperatures above 100°F for 4.5 minutes. Transmission to an athlete undergoing rehabilitation may be facilitated by the athlete's skin abrasions (Nerurkar et al., 1983).

Most of the clinical work on HSV infection in contact sports has involved HG. There is no reason to suspect that the principles of diagnosis, treatment, or prevention of HSV would be different among the various contact sports. Herpes gladiatorum is caused by HSV-1, although not all studies report typing the virus.

Clinical Presentation

Once HSV is located on an athlete's skin, its incubation period ranges from a few days to several weeks. Athletes first experience a prodrome characterized by burning, stinging, itching, or pain in the area that eventually develops a skin lesion. The appearance of HG varies and depends primarily on the age of the lesion (Table 2-3). Very early lesions have a nonspecific clinical appearance and are well-defined erythematous papules. As the lesion matures, it develops the characteristic grouped vesicles upon an erythematous base (Figures 2-2 through 2-4, see color plate for Figure 2-2). As the individual lesion resolves, crust develops on top. The lesion eventually heals, with or without therapy.

It is important for clinicians to realize that athletes know the presence of HSV results in disqualification. To avoid detection, athletes may secondarily alter the clinical morphology of the lesion. They may attempt to use sandpaper or household bleach to mask the appearance of blisters or suspicious papules.

The distribution of HG and HR results from the skin-to-skin contact inherent in both rugby and wrestling. In both sports, the most common locations of HSV infection are extramucosal sites. The head and neck account for 75% of all HG lesions. The extremities are the next most common location of lesions, followed by the trunk (Adams, 2001; Anderson, 2003; Dworkin et al., 1999). In rugby, the most common location for infection is on the head and neck (representing more than two thirds of all lesions), but the shins, hip and hands also can be infected (Skinner et al., 1996; White and Grant-Kels, 1984).

Systemic symptoms are not unusual, especially in cases of primary HSV infection. Athletes may complain of fever, sore throat, malaise, myalgias, arthralgias, and swollen lymph glands.

Complications of either HG or HR are not inconsequential. Several organ systems are affected. Athletes participating in both sports report eye complica-

Table 2-3. Clinical Appearance of Lesions of Herpes Gladiatorum or Herpes Rugbeiorum Based on Duration

Clinical Appearance	Age of Lesion (days)
Red papule	1–2 (very early)
Grouped blisters	2–6
Crusted papule	5–14



Figure 2-2. Grouped vesicles on an erythematous base are characteristic of herpes gladiatorum or herpes rugbeiorum. The ears are commonly affected in wrestlers. (See color plate.)

tions. Involvement of the eye, including herpes conjunctivitis, blepharitis, and keratitis (Holland et al., 1992; Rosenbaum et al., 1990; Selling and Kibrick, 1964), has been noted in 8% of wrestlers with HG (Holland et al., 1992). Likewise, more than half of reported patients with HR developed ophthalmologic complications, including keratoconjunctivitis and blepharitis (Skinner et al., 1996; White and Grant-Kels, 1984). Although these ophthalmic findings generally resolve, permanent impairment has been noted (Keilhofner and McKinsey, 1988). The risk of ocular recurrence is greater than 33% within 5 years. Recurrence may affect the cornea even though the initial infection involved only the eyelid (Mast and Goodman, 1997). One wrestler developed a monoarticular arthritis from which HSV was cultured (Shelley, 1980); this complication has not been documented in rugby players. Neurologic involvement has been reported in HR. One rugby player with HR developed HSV-related meningitis and sacral ganglionitis with perineal and lower extremity paresthesias (White and Grant-Kels, 1984).

The distribution of lesions supports the prevailing idea that HSV infections in sports are not transmitted through inanimate objects such as wrestling mats. If the mat played a role, more lesions on the lower extremities would be expected.



Figure 2-3. Primary herpes gladiatorum infection can be extensive and frequently occurs on the neck.



Figure 2-4. Herpes gladiatorum can be quite subtle and difficult to diagnose, especially in atypical locations. A very high index of suspicion helps prevent misdiagnosis.

However, most lesions occurring on the head and neck correspond to the cross-face maneuvers and locking-up position of wrestlers. Maceration resulting from sweating and the abrasions obtained facilitate HSV infection. Abrasive shirts and unshaven beards are other purported reasons for easy transmittal of HSV (Strauss et al., 1989).

Transmission of HG or HR is enhanced by the fact that infected individuals may shed virus for days before the infection becomes obvious. Once infected, an athlete is a carrier and may insidiously infect others before outward signs of blisters are apparent. One study showed that the probability of HSV transmission in the setting of wrestlers was 33%.

Diagnosis

The diagnosis can be challenging because very early lesions are difficult to distinguish from acne vulgaris, atopic dermatitis, molluscum contagiosum, tinea corporis gladiatorum, and impetigo (Table 2-4). Furthermore, adulterated or very late lesions do not demonstrate the diagnostic grouped vesicles on an erythematous base. In indeterminate cases, Tzanck smear, culture, DFA, or polymerase chain reaction (PCR) may be necessary.

Tzanck smear provides the most rapid result but can be technically challenging even for seasoned dermatologists. One study showed that practicing dermatologists were only 67% correct in using the Tzanck smear (Grossman and Silvers, 1992). Culture is the gold standard for identifying the presence of HSV infection, but the culture may take 1 to 2 days to become positive. Direct immunofluorescence provides rapid results (often within just a few hours), but the test is not universally available at all laboratories. Advances in PCR technology allow rapid and precise detection of HSV. The enzyme-linked viral inducible system (ELVIS) test is highly sensitive and specific, with positive and negative predictive values of 99% and 99%, respectively (La Rocco, 2000). Although PCR theoretically is the ideal test, its cost and availability limit its use at the "mats." Until national collegiate and state high school associations commit financial resources to making rapid yet affordable tests, such as the DFA, available at wrestling competitions, epidemics at the high school and collegiate levels will continue.

Because DFA may not detect very early lesions of HSV, the clinician must base the diagnosis on a constellation of findings, including a prodrome history, suspicious lesions, and associated systemic findings.

Table 2-4. Differential Diagnosis of Herpes Gladiatorum

Acne vulgaris
Atopic dermatitis
Impetigo contagiosum
Molluscum contagiosum
Tinea corporis gladiatorum

Treatment

Unlike the infections resulting from participation in noncontact sports, contact sport infections with HSV are either newly acquired or reactivated HSV. The original dosing regimen for reactivated HSV was oral acyclovir 200 mg given five times per day for 5 days. Although oral acyclovir is the most inexpensive of all oral antiviral agents for treatment of HG or HR, dosing at five times per day decreases patient compliance. More expensive but easier dosing regimens, which likely translate to increased compliance, include famciclovir and valacyclovir (Table 2-2). The dose of oral famciclovir is 500 mg twice per day for 5 days. In the case of newly acquired HSV lesion, the athlete should take the oral antiviral agent for 1 week. A study revealed that the dosage of valacyclovir is 2 g twice per day for only 1 day. Although this dosing pattern has not been specifically tested in sports-acquired infections, this simple dosing regimen is as effective as the other regimens and likely will result in high athlete compliance. Valacyclovir is a relatively safe medication that has been used in athletes, with no known untoward side effects. This medication should be used with caution in individuals with kidney disease or immunosuppression.

No data support a length of time during which athletes are contagious after treatment with oral antiviral agents. Use of acyclovir decreases viral shedding to 4 days. As a reasonable extrapolation, athletes can safely return to their skin-to-skin contact sport after 4 days of oral antiviral therapy. This recommendation should help thwart team and league epidemics. No studies have examined topical antiviral agents in terms of HG or HR, so these agents should not play a role in the therapy of athletes.

Prevention

No evidence indicates that wrestling mats play a role in transmission of HSV to other athletes. Therefore, the need to perform time-consuming daily washing and cleansing of mats is not supported. However, the practice seems prudent and continues, especially with the recommendation of clinicians. It also seems prudent that athletes routinely and immediately shower after practice and competition, although no evidence-based medicine supports this practice. Athletes must wear sandals while using communal showers. Weak evidence supports the wearing of protective gloves while athletes use weightlifting equipment to decrease the transmission of infectious agents. Athletes should not share protective equipment, towels, razors, or clothing. These items must be cleansed regularly (Table 2-5).

Several companies produce skin protection substances in the form of foams that athletes apply to their skin or trainers and coaches apply to equipment and mats. These skin protectants and wrestling gear and mat cleansers purportedly kill the microorganisms that afflict the wrestler. To my knowledge, no study has investigated these claims, and clinicians should skeptically evaluate the claims made by the manufacturers.

Table 2-5. General Primary Prevention of Communicable Skin Infections in Athletes

Avoid sharing protective equipment, clothing, towels, razors
Encourage regular cleansing of towels and equipment
Use protective gloves when using weightlifting equipment
Take immediate shower after practice and competition
Wear sandals in the locker room and showers

The keys to thwarting epidemics in teams and leagues are extensive surveillance coupled with rapid institution of oral therapy. Daily practices are ideal times to examine the athletes' skin. Coaches, trainers, and athletes themselves should join forces to ensure that all lesions are detected as early as possible. The ultimate control of epidemics in contact sports will come when a specifically trained clinician is assigned to each school or, if funding is an issue, a group of schools. At present, especially on the high school level of sports activity, a medley of clinicians cares for the athletes on a team. In most states, wrestlers must present a completed evaluation and authorization form to the precompetition official. However, the policy is not consistent, and no uniform or logical diagnostic approach or treatment and prevention plan is available. In addition, the individuals who disqualify or qualify athletes need not be clinicians with any particular expertise in the area.

One interesting study illustrates this quandary. In one area of the United States, more than 90% of wrestlers with a questionable communicable skin disease were incorrectly allowed to return to wrestling. Physicians incorrectly diagnosed 92% of wrestlers (who ultimately were culture positive for HSV) as having impetigo, tinea corporis gladiatorum, and eczema 70%, 10%, and 10% of the time, respectively. Furthermore, viral cultures were completed during the initial evaluation for only 20% of the infected wrestlers. Because of these misdiagnoses, three counties in Washington state experienced an epidemic in which 5% of all wrestlers were infected with HSV (Dworkin et al., 1999).

On the other hand, the NCAA has highly specific guidelines for wrestling coaches, athletes, and clinicians (Table 2-6). No specific guidelines exist for collegiate-level rugby. An athlete with HG may not compete unless (1) all lesions are dried and covered with an adherent crust and (2) the wrestler has been undergoing oral antiviral therapy for at least 120 hours. Wrestlers with a primary outbreak cannot have systemic symptoms of HG, and those with both primary and recurrent infections must not have sustained new blisters 72 hours before the skin check. The NCAA ensures that athletes adhere to these stipulations by requiring the presence of a physician or a certified athletic trainer at the precompetition skin check. No such requirement exists for high school precompetition skin checks, where a nonclinician usually determines qualification. As a result, competitors without contagious skin disease may be unduly barred from competition. Conversely, athletes with transmissible cutaneous infections who erroneously are allowed to compete can infect with epidemic potential.

Once identified, athletes with contact-related HSV should be treated immediately. If the number and location of lesions permit simple bandaging tech-

Table 2-6. Guidelines for Return to Competition for Athletes with Herpes Gladiatorum Based on National Collegiate Athletic Association Guidelines

1. All lesions must be dried with an adherent crust, and
2. The wrestler must have been undergoing oral antiviral therapy for 120 hours, and
3. No systemic symptoms of herpes gladiatorum, and
4. No new blisters within 72 hours of precompetition skin check-in

- Evidence-based data suggest however 96 hours is sufficient
- Valacyclovir's one-time dosing allows competition 96 hours later

niques, athletes can continue to practice. However, in most states, bandaging a lesion during competition is unacceptable and the wrestler is disqualified. If the number and location of lesions do not permit bandaging techniques, the athlete must be benched until oral antiviral therapy has been given for 4 days.

Because the athlete may asymptotically shed HSV, epidemics still may sprout despite satisfactory surveillance. Oral pharmacologic prevention may help prevent epidemics and obviate the challenging scenario of asymptomatic carriers. A double-blind, placebo-controlled study examined the effectiveness of oral valacyclovir in preventing HG. The study examined two different categories of wrestlers: those who had a primary HSV infection more than 2 years ago and those who had a primary HSV infection less than 2 years ago. Wrestlers who had primary lesions more than 2 years ago and took valacyclovir 500 mg once per day did not develop HG, whereas 33% of wrestlers who took placebo developed HG. Wrestlers with a primary HSV lesion less than 2 years ago who were in the valacyclovir group developed HG 21% of the time, whereas the placebo group developed HG 33% of the time. Although rigorous statistical analyses reveal no differences between the active drug and placebo groups ($p = 0.192$ and $p = 0.68$, respectively), the study was underpowered. A study with greater numbers likely would demonstrate clinical and statistical differences between placebo and active drug (Anderson, 1999).

During the second portion of the season and the study, all wrestlers were given valacyclovir 1 g per day. No wrestlers whose original HG occurred more than 2 years ago developed HSV lesions, and only 8% of wrestlers whose original HG occurred less than 2 years ago developed HSV lesions. Use of daily oral prophylaxis for herpes in athletes participating in contact sports is relatively safe and likely will decrease transmission and prevent epidemics.

One study in Europe evaluated the effectiveness of using an intracellular cytoplasmic detergent-treated virus particle vaccine. After a rugby team experienced an epidemic in which 38% of its players were infected with HSV, the vaccine was given to the entire team and five sociosexual contacts of the players. Based on HR data, at least 10% of the rugby players are expected to develop recurrences during the season. Surprisingly, none of the players developed any HR even 3 years after they received the vaccine (Skinner et al., 1996).

To truly prevent epidemics, the National Federation of State High School Associations (NFHS), state high school associations, and high schools themselves should invest time, energy, and funds to (1) designate one properly trained professional to evaluate all suspicious lesions from one school or region and complete required forms, (2) designate one properly trained professional to officiate at the precompetition check-in, (3) institute guidelines for the adequate diagnosis, treatment, and prevention of herpes in contact sports, and (4) encourage universal adoption of these recommendations. The current NCAA guidelines should be amended as evidence-based data become available.

Molluscum Contagiosum

Epidemiology

Two studies have examined the epidemiology of molluscum contagiosum among athletes. One large study of 7500 children showed that swimmers had a 7.5% incidence of infection (twice that of nonswimmers), which was highly statistically different from that of a control group of nonswimmers (Niizeki et al., 1984). In another study, 8% of 1400 cross-country runners had molluscum contagiosum (Table 2-1) (Mobacken and Nordin, 1987).

Molluscum contagiosum has been reported in multiple contact (rugby players and wrestlers) and noncontact sports (gymnasts) (Cyr, 2004; Halstead and Bernhardt, 2002). Based on data from the NCAAISS, 0.3% of all skin infections in collegiate wrestlers between 1991 and 2003 were caused by molluscum. Although any athlete may acquire the virus, the aforementioned athletes seemed particularly susceptible. In contact sports, the lesions are spread through intense skin-to-skin contact and by autoinoculation. Macerated skin from sweating and traumatic skin injuries inherent to both wrestling and rugby athletes facilitate infection by molluscum contagiosum. Presumably gymnasts, runners, and swimmers acquire their infection as a result of contact with equipment and the apparent hostile microenvironment in which the athletes place their extremities. I have seen numerous molluscum lesions in collegiate women volleyball players. The diving trauma and the tight-fitting occlusive shorts worn by the players facilitate autoinoculation once a solitary molluscum is present.

Clinical Presentation

Molluscum contagiosum, caused by a virus in the Poxviridae family, generally are asymptomatic, small (1–6 mm), well-defined white or skin-colored umbilicated (having a central dell) papules (Figures 2-5 and 2-6, see color plate). They occasionally become pruritic and develop surrounding erythema that can be intense and eczematous. The lesions may be grouped and



Figure 2-5. Molluscum contagiosum is characterized by a white well-circumscribed papule with a central dell. (See color plate.)



Figure 2-6. Very small lesions of molluscum contagiosum not uncommonly mimic folliculitis in athletes. (See color plate.)

occasionally number in the hundreds, but most often athletes have fewer than 20. The lesions can be found on the hands, forearms, and face of athletes and can be quite contagious, as its name implies. Molluscum contagiosum in athletes can have an atypical presentation with small nonumbilicated papules that resemble folliculitis.

In one study of swimmers, the majority of lesions were found on the trunk (50%) and axillae (32%) and corresponded to the areas where the children held the kickboards (Niizeki et al., 1984). A study of nearly 1400 cross-country runners noted that nearly all the lesions were located on the anterior aspects of the knees and thighs (Mobacken and Nordin, 1987). The authors supposed that distribution correlated to the method of transmission. They believed that sharing of towels, soaps, and brushes or skin-to-skin contact in the sauna and benches could have caused the outbreak. Alternatively, the runners may have acquired the virus through contact with branches, bushes, or barbed wire while they were running through the woods.

Diagnosis

The diagnosis is most often straightforward and made purely on morphologic grounds. The main differential diagnosis includes acne vulgaris, folliculitis, infected eczema, and verrucae. One study noted that athletes with molluscum were commonly misdiagnosed as having folliculitis (first most common) and infected eczema (second most common). The characteristic color and central dell set the molluscum apart but are not universally apparent, especially in some early lesions. Molluscum lacks the pinpoint black dots of verrucae.

Treatment

Lesions often spontaneously resolve in several months to 1 year. This is a long period, and most athletes choose to or must have their lesions treated with destructive methods. Curettement is the surest and most rapid method to remove molluscum. A sharp commercially available curette can be used. Alternatively, the scoop at the end of a tongue depressor that is broken longitudinally can be used (Figure 2-7, see color plate). Unfortunately, bleeding may occur and necessitate pressure or application of aluminum chloride for hemostasis (which stings). Liquid nitrogen destruction also works, but can be painful and does not remove the molluscum as rapidly and dramatically as does curettement. Chemical destructive methods include clinician-painted trichloroacetic acid or cantharidin, which once applied causes a blister to develop within 1 day, after which the molluscum is removed. If rapid resolution is not necessary and many lesions are present, topical imiquimod under an occlusive dressing may be effective. Tretinoin and tazarotene also have been used.



Figure 2-7. A tongue depressor can double as a makeshift curette when a formal surgical instrument is unavailable. The tongue depressor should be broken longitudinally and the rounded end used as the “scooper.” (See color plate.)

National Collegiate Athletic Association wrestling guidelines require curettage or removal of molluscum before the athlete participates in meets or tournaments. Single or localized lesions can be covered with Op-site or Bioclusive and subsequently Pro-Wrap and stretch tape, thus allowing the wrestler to compete in the tournament. Some state high school associations do not permit use of bandaging as an adequate means of protection during meets or tournaments (Table 2-7).

Cross-country runners whose competition includes travel through the woods should cover their knees and thighs. Swimmers should ensure that the kickboards are thoroughly cleaned. Infected swimmers should not use the kickboards.

Athletes should not share equipment, such as gloves and masks, during sports such as baseball, hockey, fencing, or softball, or knee or elbow pads during volleyball or wrestling (Table 2-5).

Table 2-7. Return to Competition for Athletes with Molluscum and Verrucae

Skin Infection	Disqualification if
Molluscum	Diffuse lesions not removed, or Solitary or clustered lesions not covered
Verrucae	Multiple lesions not adequately covered, or Multiple facial lesions not covered by mask

Prevention

To prevent the epidemic spread of molluscum to other teammates, athletes should practice several measures. Athletes should routinely and immediately shower after practice and competition. Athletes should wear sandals while using communal showers. Weak evidence supports the wearing of protective gloves while athletes use weightlifting equipment to decrease the transmission of infectious agents. Athletes should not share protective equipment, towels, razors, or clothing. These items must be cleansed regularly.

In addition to the primary prevention methods, secondary prevention should consist of routine and close inspection of the athletes by themselves and by the athletic trainers. All infected athletes must be treated as soon as lesions are identified. Some lesions may be pinpoint, and careful attention should be paid to identifying and treating these very small lesions surrounding large lesions. Athletes with cutaneous infection who are identified, treated, and isolated (or bandaged) early will not transmit the microorganism to unsuspecting fellow athletes.

Warts (Verrucae)

Epidemiology

At least one study has examined the prevalence of warts among swimmers. More than 10% of the girls had warts on their feet, and nearly 5% of the boys had warts on their feet (Gentles and Evans, 1973). Warts can occur in any athlete. Warts are transmitted through skin-to-skin contact and potentially through fomites. The lesions are less contagious than molluscum. Among the contact sports, wrestlers and football players have developed warts. Athletes without significant skin-to-skin contact are at increased risk from swimming pool decks, weightlifting and gymnastic equipment, and locker room and shower floors. Such athletes include swimmers, weightlifters, and gymnasts. Three studies have specifically examined sports' risk factors in the development of warts. Interestingly, no studies have examined skin-to-skin contact transmission of warts.

A case-control study was designed to examine the relationship between swimming and warts. The authors determined that swimmers were 1.81 times more likely to have warts compared to nonswimmers, although the difference was not statistically significant (Penso-Assathiany et al., 1999). One study discovered that hand warts were significantly ($p < 0.05$) more common in members of the crew team (25%) compared with the track team (10%) (Roach and Chretien, 1995). The authors believed that the crew team was at increased risk for warts because they were less likely to wear gloves while lifting weights and suffered more hand trauma while practicing on weight machines and on the river. Theoretically, this additional trauma allows the virus entry through the skin. One

might also wonder about the role of rowing equipment (e.g., oars) used both on the rowing machine and on the river.

One study examined locker room and communal shower floors (Johnson, 1995). Twenty-seven percent of swimmers who used the communal showers developed warts on the plantar aspect of their feet, whereas only 1.5% of individuals who walked on the locker room floor but did not use the showers had warts ($p = 0.001$) (Table 2-1).

Macerated skin from sweating and traumatic skin injuries inherent to athletes facilitate infection. An important feature of wart infection in athletes is that autoinoculation is possible through the trauma inherent in sports activities. Furthermore, calluses that are inherent to almost any sport may harbor warts (Adams, 2002). Several subtypes of human papilloma virus cause warts.

Clinical Presentation

Warts have different classifications partly related to the body area affected. Periungual warts occur around the nail; plantar warts occur on the soles; and common warts and verruca plana may appear on many parts of the body. Warts are characterized by well-defined, rough-surface, papillomatous papules ranging in size from a few millimeters to several centimeters, depending on the anatomic site and duration of infection (Figure 2-8). Verruca plana are flat verrucous papules (Figure 2-9).



Figure 2-8. Thick verrucous papules characterize warts. Note the faint pinpoint black capillary thromboses.



Figure 2-9. Another variant of warts includes flat warts or verruca plana.

Warts on the sole are not exophytic; rather, they exhibit inward growth resulting in frequent pain once they are sufficiently large. Occasionally the pain inhibits an athlete's activity. In one case, a collegiate tennis player was referred to the emergency room, where extensive radiologic tests were performed to evaluate for a stress fracture (Esterowitz et al., 1995). Once a plantar wart was identified as the cause and treated, the patient was able to play pain-free. Non-plantar warts are generally not painful but can inhibit the natural flow of athletic activity.

Diagnosis

The diagnosis is often straightforward if the lesions are filiform and are not related to calluses. Lesions related to calluses or located on the foot can be easily confused with calluses or corns if the clinician does not inspect the area carefully. The clinician may need to pare, using a sharp instrument such as a no. 15 surgical blade, to reveal the typical "black seeds" that represent the capillary thromboses. Upon paring, corns demonstrate a central core, whereas calluses demonstrate very thick epithelium with no alteration of the normal markings of the skin. Verruca plana are not as hyperkeratotic and can be confused with nonpigmented seborrheic keratoses.

Treatment

Warts must be treated because they have contagious potential and can cause discomfort that ultimately may affect an athlete's ability to perform. All hyperkeratotic warts should initially be pared with a sharp instrument (this procedure alone most often allows the ailing athlete to return to practice). Mechanical and chemical means then can be used to destroy warts. Destruction using liquid nitrogen, laser, or curettage is effective but can lead to residual pain, especially with the first two modalities. Very large warts may require multiple visits to ensure adequate removal. The athlete or athletic trainer should regularly pare down the hyperkeratosis between visits for destruction. Athletes may also use salicylic acid patch preparations between visits.

Chemical methods include application of salicylic acid, cantharidin, and trichloroacetic acid. The two latter medications can cause blistering, so the athlete should be forewarned. Topical immunomodulating agents have been developed that may be a significant aid, especially for athletes. Topical imiquimod can be applied to the wart under an adhesive bandage. This method is effective in removing warts with relatively less pain than destructive means. The pitfall to this method is the longer duration of therapy. Adequate paring of the lesion along with thorough soaking of the lesion in water before topical application also are necessary.

Recalcitrant warts can be treated with injected *Candida*, mumps antigen, or bleomycin. This approach must be performed carefully by an individual with expertise. Oral cimetidine 30–400 mg/kg per day also can be used.

Guidelines for a wrestler's participation in competition are available for NCAA athletes. Athletes with single or a few scattered lesions should be treated before the meet or tournament. Wrestlers who have multiple warts on the face that cannot be adequately covered by a mask or who have multiple warts on the body that cannot be adequately covered will be disqualified (Table 2-7).

Prevention

Athletes should practice several measures to decrease the spread of verrucae. Athletes should routinely and immediately shower after practice and competition. Athletes should wear sandals while using communal showers. Weak evidence supports the wearing of protective gloves while athletes use weightlifting equipment to decrease the transmission of infectious agents. Athletes should not share towels, razors, or clothing. Athletes should not share equipment such as gloves and masks during sports such as baseball, hockey, fencing, or softball, or knee or elbow pads during volleyball or wrestling. These items must be cleansed regularly (Table 2-5).

In addition to the primary prevention methods, secondary prevention should consist of routine and close inspection of the athletes by themselves and by the athletic trainers. Athletes with cutaneous infection who are identified, treated, and isolated (or bandaged) early will not transmit the microorganism to unsuspecting fellow athletes.

Bibliography

- Adams BB. Sports dermatology. *Adolesc Med* 2001;2:305–322.
- Adams BB. Dermatologic disorders of the athlete. *Sports Med* 2002;32:309–321.
- Anderson BJ. The effectiveness of valacyclovir in preventing reactivation of herpes gladiatorum in wrestlers. *Clin J Sport Med* 1999;9:86–90.
- Anderson BJ. The epidemiology and clinical analysis of several outbreaks of herpes gladiatorum. *Med Sci Sports Exerc* 2003;35:1809–1814.
- Becker TM, Kodosi R, Bailey P, et al. Grappling with herpes: herpes gladiatorum. *Am J Sports Med* 1988;16:665–669.
- Belongia EA, Goodman JL, Holland EJ, et al. An outbreak of herpes gladiatorum at a high-school wrestling camp. *N Engl J Med* 1991;325:906–910.
- Brenner IKM, Shek PN, Shepard RJ. Infection in athletes. *Sports Med* 1994;17:86–107.
- Chan EI, Brandt K, Horsman GB. Comparison of Chemicon SimulFluor direct fluorescent antibody staining with cell culture and shell vial direct immunoperoxidase staining for detection of herpes simplex virus and with cytospin direct immunofluorescence staining for detection of varicella-zoster virus. *Clin Diagn Lab Immunol* 2001;8:909–912.
- Cyr PR. Viral skin infections. *Phys Sportsmed* 2004;32:33–38.
- Dworkin MS, Shoemaker PC, Spitters C, et al. Endemic spread of herpes simplex virus type I among adolescent wrestlers and their coaches. *Pediatr Infect Dis J* 1999;18:1108–1109.
- Dyke LM, Merikangas UR, Bruton OC, et al. Skin infections in wrestlers due to herpes simplex virus. *JAMA* 1965;194:153–154.
- Esterowitz D, Greer K, Cooper PH, et al. Plantar warts in the athlete. *Am J Emerg Med* 1995;13:441–443.
- Gentles JC, Evans EGV. Foot infections in swimming baths. *Br Med J* 1973;3:260–262.
- Grossman MC, Silvers DN. The Tzanck smear: can dermatologists accurately interpret it? *J Am Acad Dermatol* 1992;27:403–405.
- Halstead ME, Bernhardt DT. Common infections in the young athlete. *Pediatr Ann* 2002;31:42–48.
- Holland EJ, Mahanti RL, Belongia EA, et al. Ocular involvement in an outbreak of herpes gladiatorum. *Am J Ophthalmol* 1992;114:680–684.
- Ichihashi M, Nagai H, Matsunaga K. Sunlight is an important causative factor of recurrent herpes simplex. *Cutis* 2004;74:14–18.
- Johnson LW. Communal showers and the risk of plantar warts. *J Fam Pract* 1995;40:136–138.
- Keilhofner M, McKinsey DS. Herpes gladiatorum in a high school wrestler. *Mo Med* 1988;85:723–725.
- La Rocco MT. Evaluation of an enzyme-linked viral inducible system for the rapid detection of herpes simplex virus. *Eur J Clin Microbiol Infect Dis* 2000;19:233–235.
- Laur WE, Posey RE, Waller JD. Herpes gladiatorum. *Arch Dermatol* 1979;115:678.
- Mast EE, Goodman RA. Prevention of infectious disease transmission in sports. *Sports Med* 1997;1:1–7.

- Mills J, Hauer L, Gottlieb A, et al. Recurrent herpes labialis in skiers. *Am J Sports Med* 1987;15:76–78.
- Mobacken H, Nordin P. Molluscum Contagiosum among cross-country runners. *J Am Acad Dermatol* 1987;17:519–560.
- Nerurkar LS, West F, May M, et al. Survival of herpes simplex virus in water specimens collected from hot tubs in spa facilities and on plastic surfaces. *JAMA* 1983;250:3081–3083.
- Niizeki K, Kano O, Kondo Y. An epidemic study of Molluscum Contagiosum. *Dermatologica* 1984;169:197–198.
- Penno-Assathiany D, Flahault A, Roujeau JC. Verrues, piscine et atopi. *Ann Dermatol Venerol* 1999;126:696–698.
- Porter PS, Baughman RD. Epidemiology of herpes simplex among wrestlers. *JAMA* 1965;194:150–152.
- Potasman I, Pick N. Primary Herpes labialis acquired during scuba diving course. *J Travel Med* 1997;4:144–145.
- Roach MC, Chretien JH. Common hand warts in athletes: association with trauma to the hand. *J Am Coll Health* 1995;44:125–126.
- Rooney JF, Bryson Y, Mannix ML, et al. Prevention of ultraviolet-light-induced herpes labialis by sunscreen. *Lancet* 1991;338:1419–1422.
- Rosenbaum GS, Strampfer MJ, Cunha BA. Herpes gladiatorum in a male wrestler. *Int J Dermatol* 1990;29:141–142.
- Selling B, Kibrick S. An outbreak of herpes simplex among wrestlers (herpes gladiatorum). *N Engl J Med* 1964;270:979–982.
- Shelley WB. Herpetic arthritis associated with disseminate herpes simplex in a wrestler. *Br J Dermatol* 1980;103:209–212.
- Shute P, Jeffries DJ, Maddocks AC. Scrum-pox caused by herpes simplex virus. *Br Med J* 1979;2:1629.
- Skinner GR, Davies J, Ahmad A, et al. An outbreak of herpes rugbeiorum managed by vaccination of players and sociosexual contacts. *J Infect* 1996;33:163–167.
- Spruance SL, Freeman DJ, Stewart JC, et al. The natural history of ultraviolet radiation-induced herpes simplex labialis and response to therapy with oral and topical formulations of acyclovir. *J Infect Dis* 1991;163:728–733.
- Spruance SL, Hamill ML, Hoge WS, et al. Acyclovir prevents reactivation of herpes simplex labialis in skiers. *JAMA* 1988;260:1597–1599.
- Strauss RH, Leizman DJ, Lanese RR, et al. Abrasive shirts may contribute to herpes gladiatorum among wrestlers. *N Engl J Med* 1989;320:598–599.
- Wheeler CE, Cabaniss WH. Epidemic cutaneous herpes simplex in wrestlers (Herpes Gladiatorum). *JAMA* 1965;194:993–997.
- White WB, Grant-Kels JM. Transmission of herpes simplex virus type 1 infection in rugby players. *JAMA* 1984;252:533–535.



<http://www.springer.com/978-0-387-28837-6>

Sports Dermatology

Adams, B.B.

2006, XIV, 354 p., Softcover

ISBN: 978-0-387-28837-6