

Emergencies

Pinning Down Skin Infections: Diagnosis, Treatment, and Prevention in Wrestlers

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In Brief: Wrestling fosters skin infections such as herpes simplex, tinea corporis, and impetigo. Visual examination often suggests the diagnosis, but some lesions, like late-stage herpes, can mimic other conditions, like impetigo; laboratory studies therefore may be required. Drug therapy can mitigate an infection and help prevent recurrence. In addition, physicians must know when to disqualify a wrestler and how to prevent an outbreak through measures like good hygiene and immediate diagnosis.

To distinguish between herpes gladiatorum, tinea corporis, impetigo, and other common skin infections in wrestlers (table 1), physicians must carefully assess clinical and laboratory findings. In addition, clinicians must be able to prescribe optimal therapy, minimize transmission, and know when to disqualify a wrestler (table 2). We offer recommendations for handling cutaneous infections in wrestlers, based on the best available scientific analysis and on clinical judgment.

Table 1. Differential Diagnosis of Skin Infection in Wrestlers

Viral Infections

Herpes simplex virus 1
Herpes zoster
Molluscum contagiosum
Warts

Dermatophytic and Yeast Infections

Tinea corporis
Tinea cruris and pedis

Bacterial Infections

Impetigo contagiosa
Cellulitis
Furuncles, carbuncles, and abscesses
Superinfection of abrasions or eczema
Acne vulgaris and folliculitis

Parasite Infestations

Scabies
Lice

Noninfectious Conditions

Atopic dermatitis
Contact (allergic) dermatitis
Nummular eczema

Herpes Gladiatorum

Cause and diagnosis. Herpes gladiatorum is a cutaneous infection caused by herpes simplex virus type 1 (HSV-1) that is spread through direct skin-to-skin contact in sports such as wrestling and rugby. It is characterized by grouped vesicles on an erythematous base, is capable of latency, and has a tendency to recur at the site of the primary lesion (figure 1). The illness may cause no other symptoms or may involve fever, localized lymphadenopathy, malaise, myalgia, pharyngitis, or, rarely, keratoconjunctivitis (1).

Figure 1: Courtesy of Michael D. Ramsey, MD



Figure 1. Herpes gladiatorum is characterized by grouped vesicles on an erythematous base.

Abrasions and underlying skin conditions increase the likelihood of acquiring infection, while previous HSV-1 seropositivity decreases susceptibility somewhat (2,3). Consequently, areas of the body prone to direct skin contact and abrasion, such as the head, upper extremities, and trunk, are the usual sites of infection (4). Fomitic spread is not a significant cause of infection. By age 50, over 90% of adults are seropositive for HSV-1 (5).

Table 2. Recommendations for Clearance vs Disqualification of Wrestlers Who Have Various Skin Infections

Condition	Recommendation
Herpes Simplex*	
Vesicles	Disqualify
Ulcers	Disqualify
Scabbed and dry	OK to wrestle
Impetigo	
Moist crust	Disqualify
Dry crust	Disqualify
Dry crust, treated**	OK to wrestle
Cellulitis	
Local boil	Disqualify until dry and scabbed
Generalized	Disqualify until clinically resolved
After incision and drainage	OK to wrestle if lesion can be covered
Fungal Infection	
Early season	Disqualify
Late season	OK to wrestle***

*Length of medical treatment does not affect these recommendations.

**Provided that clinical improvement follows 5 days of antibiotics.

***Suggest covering lesions and prescribing pretournament treatment for 5 days if possible to minimize spread.

Like other skin infections, herpes gladiatorum is an "occupational hazard" of wrestling (see "Containing a Herpes Gladiatorum Outbreak," below). A nationwide survey (6) of athletic trainers suggested that 2.6% of high school wrestlers and 7.6% of college wrestlers had a herpes infection during the 1984-85 season. These numbers may be an underestimate because some rashes, particularly those in the latter stages of blistering and resolution, may not be recognized as HSV. This study also suggests that a wrestler's age, and presumably the duration of an athlete's exposure through wrestling, correlate with the rate of HSV infection.

The incubation period for primary infection is 2 to 14 days. Herpes eruptions usually begin with a prodrome of burning, stinging pain or itching at the infected site, followed by clusters of vesicles on an erythematous base. At this stage it is most clinically diagnosable. Viral cultures can be obtained by unroofing a vesicle, scraping the base of the lesion, and then inoculating the cells into a viral culture medium. A Tzanck smear, however, may provide more immediate confirmation.

When seen later, during the ulceration and crusting stage, the rash can be confused with, or be complicated by, impetigo (figure 2). In later stages, viral and bacterial cultures are not reliable.

Figure 2: Courtesy of Michael D. Ramsey, MD



Figure 2. Characteristic impetigo findings are honey-colored crusting with surrounding redness.

Treatment. Acyclovir 200 mg three times a day for 10 days (wholesale regional generic cost, about \$23) can shorten the duration of primary infection by arresting viral replication (7). (With young patients, the simpler the regimen the better—ie, the fewer doses per day and the less important the timing of doses, the better.) Acyclovir is most effective early in the clinical course when the virus is multiplying rapidly. Once the vesicles are fully formed and ulcerating, it is not effective. If ulceration has occurred, benzoyl peroxide and gentle use of a hair dryer may dry the crusts more quickly and minimize secondary bacterial infection.

Treating the prodromal phase of recurrent infection with acyclovir 200 mg three times a day for 10 days, or with comparable doses of valacyclovir hydrochloride or famciclovir, is the most effective means of shortening the course of subsequent infections. Wrestlers who have a history of recurrent HSV infection may be supplied with a course of therapy in advance, which they may start at the first sign of prodrome at the usual site of vesicle formation. Frequent eruptions may be interrupted with continuous prophylactic therapy with acyclovir 200 mg twice a day or valacyclovir 500 mg daily (generic 7-day cost, about \$39), or famciclovir.

Although prophylactic treatment has yet to be formally studied in wrestlers, 75 skiers who had a history of recurrent oral herpes exacerbated by solar exposure and who were treated with acyclovir had 73% fewer recurrences when compared with a placebo group (8,9). There is some concern that overuse of antiviral medications could cause the development of resistant strains of HSV, which has been observed in both immunodeficient and immunocompetent patients who are on chronic acyclovir therapy (10).

One 1992 report (6) listed the cost for patients of 3 months of acyclovir at \$400 to \$550. Valacyclovir requires less frequent dosing and is less expensive, but it has not had formal study for this usage. Another effective and possibly less expensive strategy for those who have frequent eruptions is to use prophylactic acyclovir, valacyclovir, or famciclovir only during critical competition, like postseason tournaments.

Disqualification. Although therapy may allow some wrestlers to return to competition sooner, wrestlers with active infections should be barred from physical contact during practice and competition until their scabs are dry and they have no further vesicles, open ulcers, or drainage.

Tinea Corporis

Cause and diagnosis. Tinea corporis gladiatorum is caused by dermatophytes, usually of the genus *Trichophyton*, and it affects both humans and animals. It is characterized by often-circular, pruritic patches that are typically well demarcated and scaly with raised borders and central healing, producing rings (figure 3).

Figure 3: Courtesy of Michael D. Ramsey, MD



Figure 3. Tinea corporis ('ringworm') typically manifests as circular, well-demarcated, scaly patches with raised borders and central healing, which produces rings.

Certain individuals can carry the spores without the rash. The degree of rash is dependent on the host's cellular-based immune response, which varies. This also explains why corticosteroids may mask the rash temporarily without treating the infection, which can later flare. Conditions typical of wrestling—like moisture, occlusive clothing during practice, abrasions, and direct skin-to-skin contact—create favorable conditions for tinea corporis outbreaks.

Although a literature search revealed only a few published reports (11-14) of epidemics of tinea corporis gladiatorum, most authorities feel that outbreaks are more prevalent than reported. Compared with herpes, tinea corporis lacks serious long-term or short-term morbidity, which may explain the paucity of published information.

Treatment. It can be difficult to contain a tinea outbreak because of both the long incubation period and the unknown minimum duration of therapy before the patient is rendered noninfectious. Some individuals, especially those with multiple lesions or hair involvement, need to be treated with systemic therapy. Topical treatment using imidazoles like clotrimazole, miconazole nitrate, and ketoconazole requires consistent application to all lesions. New lesions can be missed.

Oral therapy, traditionally with griseofulvin 500 mg twice a day, may be used, but it is not always effective (15). More recently, systemic ketoconazole (generic wholesale regional 7-day cost, about \$47), fluconazole (about \$48), and itraconazole (about \$42) have shown excellent results, but extended use may cause liver toxicity (15). They are also relatively expensive.

Pulse therapy may be considered for those with multiple or chronic lesions, or when a team is faced with an epidemic. In 1996 in Washington state, this approach effectively stopped a widespread outbreak (see "An Outbreak of Tinea Corporis," below). Generally, continued topical treatment is recommended for 2 weeks after the disappearance of the lesions.

Disqualification. Disqualification of those with tinea corporis can be more difficult, because it is not a very serious infection, and also because it can take several weeks of treatment before the lesions are no longer visible. The minimum duration of therapy to prevent contagious spread has not been determined.

At Clarion University in Pennsylvania, wrestlers are withheld from practice and competition until they receive 24 hours of drug treatment. The lesions are then covered with a gas-permeable dressing, prewrap, and stretch tape until the flaking stops (16). If the lesion cannot be covered, participation is barred for 5 days.

It may make sense to take a more aggressive stance against tinea corporis by barring

competition and practice early in the season and then being more lenient about participation at the end of the regular season and during tournaments.

Bacterial Skin Infections

Cause and diagnosis. With respect to their implications for wrestlers, bacterial infections fall into three classes: those that are contagious like impetigo; those in which the wrestler risks further morbidity with continued competition, like cellulitis, furuncles, and carbuncles; and those in which continued competition is permitted, like a local (nonvirulent) bacterial overgrowth associated with folliculitis, abrasions, or dermatitis.

Contagious. Impetigo causes honey-colored crusting with surrounding redness (figure 2) and, occasionally, systemic symptoms. Multiple satellite lesions suggest a more virulent strain of the usual *Staphylococcus aureus* or streptococcal pathogens. Wrestlers who have impetigo should be started on oral antibiotics. In most cases, dicloxacillin sodium, a first-generation cephalosporin, or erythromycin or its derivatives are effective.

Noncontagious. For cellulitis, furuncles (boils), and carbuncles, treatment with similar antibiotics is indicated, and incision, drainage, and, in some cases, debridement may be necessary. Folliculitis, acne, local abrasions, and dermatitis complicated by superinfection by skin flora are also typically not contagious.

Disqualification. Those who have bacterial skin infection should be barred from participation when they have purulent conjunctivitis, large or multiple honey-crusting lesions, and cellulitis or abscess formation. With impetigo, wrestlers should not participate until the crusts have dried to the eschar stage. Wrestlers who have cellulitis, furuncles, or carbuncles are not significantly contagious to others, but they should be barred from competition and practice because continued trauma to the involved areas can lead to systemic complications such as bacteremia and progressive soft-tissue infection.

Folliculitis, acne, superinfected local abrasions, and infected dermatitis are typically not contagious and therefore, following careful review, may not require barring the patient from competition. Of course, a certain degree of clinical judgment must be used. The presence of satellite lesions, cellulitis, and weeping lesions warrants withholding the athlete from competition. It may be prudent to be more restrictive about participation early in the season and less restrictive later during crucial tournaments.

Other Possibilities

Herpes zoster (shingles), though rare, can affect young athletes, and it may suggest impaired immunity. Participation should be withheld both for pain relief and to lessen transmission to contacts who have never had chicken pox. Acyclovir, valacyclovir, or famciclovir, if given early, will shorten the course.

Molluscum contagiosum may affect younger athletes who have immature immune systems. Like warts, these lesions should not lead to disqualification in most cases. Atopic and contact dermatitis and, especially, nummular eczema can resemble dermatophyte infection, in which case potassium hydroxide scraping and negative fungal culture may be helpful.

Stopping the Spread

When a high school or college senior must be disqualified late in the wrestling season before tournament time, it can be heartbreaking. Therefore, disqualification should not be taken lightly, but it is sometimes essential for preventing an epidemic.

At major tournaments such as the state wrestling tournament, we have found it helpful for the referees to screen all wrestlers at weigh-in. Those who have skin lesions are examined by a consensus panel of three or four doctors. One must balance the wishes of the individual competitor against the short- and long-term public health risks to the other competitors. The medical risks that further competition might cause the infected wrestler must also be taken into account.

Hygienic measures, such as mandatory showers before and after practice, use of antibacterial soaps, and daily washing of practice gear, may be the most effective means of preventing outbreaks of skin infections. This deserves a special final emphasis. It may be "penny wise and pound foolish," for example, for school districts to stop providing towels for wrestlers as a cost-saving measure.

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Containing a Herpes Gladiatorum Outbreak

At the end of the 1995-96 high school wrestling season, a team in the North Puget Sound region of Washington experienced an outbreak of herpes gladiatorum that appeared to be spreading to other teams. The coach notified the county and state health departments, and viral

cultures were then taken from this index team and from symptomatic contacts on other teams.

To prevent further transmission, the medical director of the 1996 Washington State Wrestling Tournament set up a protocol by which the referees screened all wrestlers for any skin lesions. A team of physicians, including several of the authors, observed the lesions and determined whom to disqualify because of active herpes simplex. Wrestlers who had tinea skin infections were not disqualified because of the relatively low morbidity of tinea corporis and the relatively high importance of the tournament.

In all, four wrestlers were disqualified at the state tournament for suspected herpes infection—an unpopular but epidemiologically necessary decision. Further, viral cultures of suspicious rashes were performed on location by an epidemiologist from the state health department.

An investigation of the outbreak revealed that 12 cases were confirmed by isolation of herpes simplex type I virus (HSV-1). An additional 40 possible cases, suggested by the clinical appearance of the rash, were noted. Identification of three separate strains of HSV-1 suggested three separate outbreaks that could be traced to different communities in western Washington.

Just after the state tournament, one of the affected communities, Olympia, had an additional outbreak affecting four wrestlers and two coaches. Three of these infections were cultured, and all three were positive for both HSV-1 and *Staphylococcus aureus*. All were treated with acyclovir and antistaphylococcal antibiotics. A 58-year-old coach had the most severe clinical presentation, a reminder that some adults are susceptible.

A protocol for this high school was adopted at the beginning of the 1996-97 season, and wrestlers and their parents were required to sign it. Personal hygiene was emphasized, including daily showers at school immediately after practice, daily washing of practice gear, and early reporting of skin lesions to the coaches and team physician.

During the 1996-97 season, the three previously infected wrestlers who returned, including one who had received prophylactic acyclovir, remained asymptomatic. Two other wrestlers had primary herpetiform rashes during the season. Investigation suggested that these were acquired from a symptomatic wrestler from a competing team in the league.

An Outbreak of Tinea Corporis

An outbreak of tinea corporis affected one quarter to one third of a high school wrestling team in Washington state during the 1994-95 and 1995-96 seasons. By the end of the first season, despite attempts to treat each case on recognition, members continued to develop fungal lesions. After the season concluded, some wrestlers later reported having continued treatment for several months.

During the 1995-96 season, as soon as another index lesion appeared, aggressive topical therapy was started. All team members were then screened weekly, and suspicious lesions were treated with topical antifungals immediately. Wrestlers who had hair involvement or multiple disseminated lesions were also given a week or two of oral ketoconazole; the expense of this therapy limited this approach. Despite these efforts, new cases continued to develop. By January, with the tournaments leading to the state championships less than a month away, it was time for more intensive measures.

First, wrestlers were screened daily. All new cases were treated both with topical antifungal therapy and a 1-week pulse of oral itraconazole, 200 mg once daily. It was reasoned that a week of systemic therapy would limit the contagiousness of the skin infection but would not be

long enough to necessitate blood screening tests for liver function.

Second, wrestlers were encouraged to take their laundry home for cleaning each day. Third, after-practice showers were again encouraged. (To cut costs, the school district had ceased providing clean towels, so most wrestlers would shower at home after practice. Some would wear their sweats for hours before showering.) Fourth, the antiseptic solution used to clean the wrestling mat daily was reviewed and appeared adequate.

Zoogenous transmission was also considered, but resources to evaluate this possibility were not available. However, one of the adult support staff who worked as the town's animal control officer was instructed to change his work clothing and work boots before entering the wrestling room.

This combination of measures worked. No additional cases were detected. Most skin lesions were essentially healed by tournament time in February.

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