

Managing Herpes Gladiatorum Outbreaks in Competitive Wrestling: The 2007 Minnesota Experience

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ANDERSON, B.J. Managing herpes gladiatorum outbreaks in competitive wrestling: the 2007 Minnesota experience. *Curr. Sports Med. Rep.*, Vol. 7, No. 6, pp. 323–327, 2008. *Skin infections in wrestling have escalated in the past 20 yr. Failure to recognize and manage primary or recurrent herpes gladiatorum (HG) puts all wrestlers who come in direct contact with the affected athlete at risk. In 2007, a major outbreak of HG occurred during the Minnesota State High School wrestling season. Rapid response to the outbreak based upon lessons learned from previous episodes in the state prevented an epidemic from developing that would have threatened the state competitions at the end of the season. When a primary outbreak occurred involving multiple teams, an 8-d isolation period with suspended competition contained the outbreak in more than 90% of exposed individuals. Prophylactic treatment with antiviral medications can reduce recurrent infections, the risk of asymptomatic viral shedding, and can be based upon annual herpes simplex virus (HSV) testing to identify seropositive individuals. Those with recurrent HG or who are HSV seropositive should be placed on seasonal prophylaxis with oral antiviral medication to reduce the risk of HG spread to susceptible teammates or opponents. With proper education of athletes, coaches, and health care providers, HG can be recognized, treated, and controlled.*

INTRODUCTION

Recognition of skin infections in wrestlers has come to the forefront during the past several decades. Outbreaks of tinea corporis gladiatorum, due to the dermatophyte *T. tonsurans*, were documented in the 1960s with human contact identified as the primary vector or transmission (1). Outbreaks of herpes gladiatorum (HG), caused by herpes simplex virus type I (HSV-1), also were documented during that period, but there was no consensus on whether the disease was transmitted by skin-to-skin contact or by contact with mat surfaces (2).

HG outbreaks in young athletes are a cause for concern because of the nature of herpes virus infections and the anatomical location of the lesions. After primary infection, herpes viruses establish latent residence in the trigeminal ganglia (facial lesions) or the dorsal root ganglia (lesions on other areas of the body). Viral reactivation may occur periodically with recurrent clinical symptoms and concomitant shedding of infectious virus particles. Primary infection in the eye can result in recurrent outbreaks on the

cornea, causing scarring that might require corneal transplant, or even more catastrophic, lead to retinal necrosis and blindness (3). HG outbreaks should be regarded not simply as benign skin infections, but as serious infections that can have long-term, negative consequences upon the health of individual wrestlers and the community.

In January 2007, a large outbreak of HG occurred during the Minnesota State High School wrestling season, prompting the state's first complete shutdown of a sport due to an infectious disease. In response to the outbreak, we relied upon data from previous studies on the mechanism of viral spread, the length of time from exposure until vesicle formation, and disease management based upon isolation and use of antiviral medication. Suspending competition avoided a catastrophic outcome while preserving the integrity of the sport and allowing the season to finish without disqualifying any athletes from the State tournament.

This report reviews the diagnosis, treatment, and management of HSV-1 infections in the setting of high school wrestling, with emphasis to the lessons learned over the last decade from outbreaks at summer wrestling camps in Minnesota and at the Minnesota State High School League (MSHSL) wrestling tournament.

HSV PREVALENCE

In the late 1980s, HG prevalence in collegiate and high school wrestlers was estimated based only upon clinical

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observations. For example, data published by Becker *et al.* (4) showed that 2.6% of high school wrestlers and 7.6% of collegiate wrestlers have had documented outbreaks of HG. However, these estimates did not correlate with the number of outbreaks that were occurring in the wrestling community – outbreaks that developed in wrestlers assumed to be HSV-naïve.

The increased sensitivity of polymerase chain reaction (PCR) testing and HSV antibody testing improved our understanding of the virus and its prevalence among high school and collegiate wrestlers, allowing better management of individual cases and group outbreaks. In 2003, approximately 360 male high school wrestlers participated in a 28-d wrestling camp (5). Ninety-four camp participants agreed to have HSV serology performed. Of those tested, 29.8% were seropositive for HSV-1, yet only 3.6% had a history of documented HG outbreaks (5). Approximately two thirds (68%) of the HSV-1-seropositive individuals elected to take oral antiviral medication as a prophylaxis for the length of the camp. Notably, camp outbreaks of HG dropped 87% that summer compared with the camp average over the previous 20 yr (5).

Results from this outbreak and others suggest that positive serology may be the best indicator of infectivity. PCR testing shows that viral shedding may occur before vesicle formation in infected individuals and may even occur in completely asymptomatic individuals (6–8). With transmission as high as 30% following exposure (9), HSV-1-seropositive individuals may unknowingly be transmitting the virus to HSV-1-naïve individuals.

As scientific testing advances our ability to define the prevalence of HSV in wrestlers the question arises: should all HSV-seropositive individuals be treated as potentially infectious?

INCREASED RISK FROM CHANGES IN WRESTLING STYLE

Over the last few decades, collegiate and high school wrestling has evolved with changes in the areas of bodily contact and the degree of skin-to-skin exposure. Understanding those changes is key to recognizing HG outbreaks and anticipating where on the body the HG rash is likely to occur. Review of wrestling videos from the 1960s shows a significant difference in high school wrestling style compared with current technique. Previously, competitors focused on taking an opponent to the mat, and continued to work for the pin. Teams were awarded 6 points for a pin and 3 points for any other win, with no additional points for a superior decision. Rule changes in the late 1970s began to favor Olympic freestyle wrestling in an effort to make the sport more exciting. Under the new rules, team points were awarded for a superior decision on an individual match score, where beating an opponent by 8 points rewarded the team with 4 points and by 15 or more gave the team 5 points. This changed the complexion of high school and collegiate wrestling tremendously. Now if the opponent could not be pinned, the wrestler would instead focus on obtaining a superior decision, usually by using the strategy of

repeatedly taking the opponent down for points and allowing the escape with a net gain in match points with each cycle.

From 1968 to 2003, the amount of time a wrestler spent in the lock-up position (Fig. 1) with his opponent increased 62% (10). This increased time spent in the lock-up position was clearly one of the primary changes in wrestling style brought about by the team scoring rule changes. Skin-to-skin contact is the primary means of viral transmission in HG, and the increased time in the lock-up position has increased skin-to-skin contact, particularly on the face and neck. Because most athletes are right handed, opponents typically lock-up with the right side of their face in opposition. As expected, a review of several HG outbreaks shows that more than 70% occur on the head, neck, and face, with a predilection for the right side (Fig. 2) (9,11).

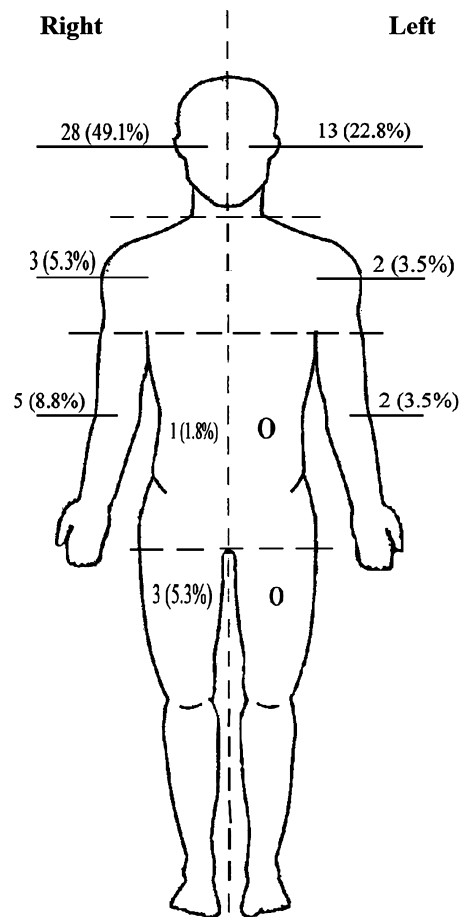
RECOGNITION

A number of infectious agents can be transmitted through combat sports such as wrestling. The differential diagnosis of skin lesions in wrestlers should focus upon the bacterial, viral, and fungal pathogens associated with infections characteristic of combat sports. For example, skin lesions in wrestlers other than HSV may arise from bacterial pathogens such as *S. aureus*, viral pathogens such as poxvirus, which causes *Molluscum contagiosum*, or fungal pathogens such as *T. tonsurans*.

HG is a great masquerader, and to effectively diagnose an HG outbreak, a clinician needs to understand the pathophysiology and mode of transmission of HSV-1 in the context of the sport of wrestling. Relying upon rapid laboratory tests such as Tzanck smears to identify HSV-1 infection will miss many cases, as fewer than 65% of cases are accurately diagnosed by this method, even in the most experienced hands (12). This leaves the viral culture as the usual option for diagnosis. But prior to obtaining cultures to verify viral presence, a provider should understand the clinical presentation of HG. PCR testing is now a widely available diagnostic tool that has made clinical diagnosis and treatment of HG a much easier process. PCR testing is a



Figure 1. Extensive facial contact with classic lock-up position.



Location of lesions from 57 wrestlers with Primary Herpes Gladiatorum.

Figure 2. Bodygram with locations of primary herpes gladiatorum (HG).

sensitive and cost-effective method to determine viral presence and should be considered the gold standard for detecting HSV-1 or HSV-2 in individuals with a rash suggestive of a herpes infection.

With facial involvement, more than 90% of primary HG will develop within 8 d after exposure (9). Primary infection is characterized by localized facial pain, pharyngitis, fever to 101.5°F, regional lymphadenopathy, and (finally) the typical vesicular outbreak. Right-sided facial involvement predominates in HG because most wrestlers are right handed (9). Primary outbreaks typically show more extensive involvement compared with recurrent outbreaks. Vesicle formation commonly will be spread out over several dermatomes, possibly affecting both sides of the body. Vesicles of approximately 2 mm in diameter typically are coalesced in clusters ranging from 2 to 3 vesicles to as many as 7 to 10. The affected area initially appears reddened and is firm to touch. The lesions are commonly misdiagnosed as folliculitis with surrounding cellulitis. Critical differences between HG and localized bacterial infections are the extensive regional adenopathy observed in HG and the propensity for these lesions to cross the hairline onto the scalp. Without antiviral medications, outbreaks will usually clear within 10 to 14 d but can take longer.

Recurrent HG usually involves a much smaller, localized area, with the outbreak always occurring on a given side. Sidedness depends on the ganglion in which the virus has established latency. Viral reactivation occurs within the ganglion and spreads along the sensory dermatome associated with that ganglion. During a recurrent outbreak, touching an open vesicle and then touching other parts of the body can lead to auto-inoculation of those areas, but reactivation will only occur within the original ganglion.

Recurrent outbreaks typically last 7 to 10 d without treatment, but can clear sooner with oral antiviral medications. Vesicles may appear dried and inactive after 3 d of antiviral treatment, but it is important to recognize that the individual may still be infectious. In an earlier study, virus was detected by PCR for 6.43 d after initiation of oral antiviral therapy (13). Allowing an athlete to return to competition too soon may risk the spread of HG and jeopardize the safety of other wrestlers.

2007 OUTBREAK

The MSHSL took the unprecedented step of temporarily suspending wrestling competition and sparring in January of 2007 following an outbreak of HG that developed after a large team tournament held in southern Minnesota at the end of December 2006. Within the next 30 d, the virus spread to 24 athletes on 10 teams. Smaller schools with limited access to local health care providers and more consistent continuity of care were better able to control the outbreaks (14). Once the schools in larger communities developed cases, containment was no longer feasible because of the lack of experience among the large set of available providers in the large communities and the continuous care required from experienced medical providers to ensure early diagnosis and proper treatment. Although the MSHSL guidelines for skin checks and treatment are strictly followed at tournaments and meets held in Minnesota, the guidelines alone are insufficient to prevent disease spread in a situation with a rapidly spreading infection involving multiple teams. The MSHSL officials in consultation with representatives of the Sports Medicine Advisory Committee decided to suspend all competition to stop all exposures to the virus and to allow time for potential individual outbreaks to be confirmed and controlled by antiviral therapy. The state-wide wrestling shutdown was ordered for 8 d based upon earlier studies showing that an 8-d isolation period would be sufficient to contain greater than 90% of the predicted disease spread (9).

During the shutdown period, all wrestlers in the state were prohibited from engaging in direct skin-to-skin contact with other athletes. Coaches and athletic trainers performed daily skin checks, and athletes with suspicious lesions were sent to their health care providers for evaluation, culture, and appropriate treatment. By the end of the 8-d break, the number of infected athletes rose to 54 individuals from 23 teams, consistent with the expectation that infections from earlier exposures would become clinically apparent. Notably, only two additional wrestlers developed HG over the next 3 wk leading up to the state tournament. The two

athletes who presented with lesions after the 8-d suspension period were members of teams involved in the outbreak. We hypothesize that they were infected by exposure to infected team members who were noncompliant with oral antiviral medications.

Previous studies of HG outbreaks played an important role in establishing criteria to predict the potential for an outbreak to develop epidemic status and determine how long to suspend wrestling competition and sparing to effectively stop the spread of the virus. Based upon these guidelines, the 2007 outbreak was controlled, and only two new cases emerged after the 8-d shutdown. The 2007 HG numbers are consistent with those reported from the 1999 Minnesota HG outbreak (Fig. 3).

In summary, the state-wide 8-d wrestling suspension successfully prevented transmission of the virus within and between teams. Without this preventative intervention, an estimated 180 athletes could have been infected by the time of the state tournament, instead of the 56 who actually were infected (Fig. 4). Furthermore, many of these athletes would not have been able to participate in the tournament.

TREATMENT AND PREVENTION

Topical antiviral agents are not recommended in the wrestling setting. Although topical antiviral agents may speed healing of HG lesions, the dosing parameters (5 to 7 applications each day) are unreasonable (15,16) and compliance is poor (17). Also, no reproducible studies verify the ability of topical antiviral agents to consistently reduce viral shedding and transmission. This leaves the oral antiviral agents as the treatment of choice. The newest oral antiviral agents, valacyclovir and famciclovir, are prodrugs (a prodrug is metabolized *in vivo* into the active metabolite) of acyclovir and penciclovir, respectively. The improved bioavailability of these oral prodrugs means that they can be given with once- or twice-daily dosing, leading to better compliance.

In a 2003 study evaluating treatment of recurrent HG based upon PCR data, valacyclovir 500 mg BID cleared an outbreak in 6.43 d compared with 8.14 d with placebo (13). In earlier studies, treatment with oral antiviral medications resulted in wound crusting and apparent clinical healing after 3 d, but PCR data detected active viral shedding for an additional 3.4 d (13). Present treatment guidelines for recurrent herpes labialis focus upon clinical clearance of the outbreak. To reduce the likelihood of viral transmission to an opponent, treatment regimens for wrestlers with recurrent HG outbreaks must be extended long enough to stop viral shedding (6–7 d), regardless of clinical appearance.

	5 days	7 days	8 days
1999 (n = 15)	4 (27%)	11 (73%)	14 (93%)
2007 (n = 11)	6 (55%)	8 (73%)	10 (91%)

Figure 3. Incubation time until clinical presentation of herpes gladiatorum (HG) on a team.

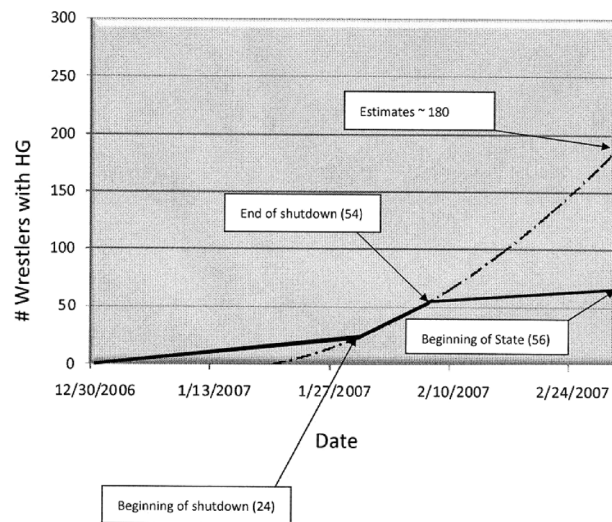


Figure 4. 2007 MSHSL herpes gladiatorum outbreak.

No studies have been performed in patients with primary HG outbreaks, but treatment of primary HSV-1 or HSV-2 infection requires higher dosing of antiviral agents (acyclovir, valacyclovir, or famciclovir) to suppress effectively and expediently viral shedding. With a primary outbreak, wrestlers should be treated with oral antiviral medications and withheld from competition for a minimum of 10 d. Return to competition should be allowed only if all systemic signs of infection, such as lymphadenopathy, vesicles, or fever, are completely resolved. If any of these signs persist, isolation from other wrestlers should be extended up to 14 d.

Suppressive therapy is critical for prevention of recurrent HG outbreaks in individuals known to be HSV-1-seropositive. In individuals with recurrent genital herpes, prophylactic valacyclovir was shown to be effective in reducing herpes outbreaks (18). Based upon those findings, we evaluated prophylactic valacyclovir treatment in wrestlers who had recurrent HG. Treatment with 1 g valacyclovir daily reduced HG outbreaks by 92% in individuals with a less than 2-yr history of recurrent HG, and treatment with 500 mg valacyclovir QD reduced HG outbreaks in 88% of those with a longer than 2-yr history of recurrent infection (19).

At an annual 28-d high school wrestling camp in Minnesota, HG has been a problem for the past 20 yr. Despite excellent hygienic guidelines, outbreaks still occurred. Serological analyses showed that 29.8% of these campers were HSV-1-seropositive, but only 3% were aware of their status (5). In a closed environment like a wrestling camp, outbreaks can spread rapidly. Prophylactic dosing of oral antiviral medication was recommended for every camp participant, regardless of seroprevalence. Since implementation of this recommendation in 2003, HG outbreaks have decreased by 85% to 90% at the camp, significantly improving the health and safety of the young campers (5). Concerns over the development of drug resistance make the chronic usage of any antimicrobial/antiviral therapy a controversial issue. However, the prevalence of acyclovir resistance in immunocompetent individuals is reported to be

only 0.3% (20), whereas the risk of HG transmission is 30% in this group (9). Thus, prophylactic antiviral therapy in wrestling camps has its merits.

An alternative approach would be to perform annual serological testing and offer suppressive therapy to all individuals who are HSV-seropositive. This approach would not only identify infected individuals who are unaware of their infection, but would also indicate which wrestlers are seronegative and thus susceptible to a primary outbreak. With this approach, serological testing would need to be repeated every year for seronegative individuals. Once a wrestler was identified as HSV-seropositive, suppressive therapy would be indicated for that wrestling season and for every wrestling season thereafter.

CONCLUSION

HG will always be an issue in the sport of wrestling. Following simple guidelines can help control the outbreaks and reduce transmission to susceptible individuals. Protocols for transmission control should focus upon proper hygienic principles, including clean workout gear, washing of the mats, daily skin checks before each practice, and showering immediately after each practice and competition. Withholding any wrestler with a suspicious lesion until HG has been eliminated as the cause of the rash is critical to interrupting the transmission vector of skin-to-skin contact. Annual HSV serological testing of all competing wrestlers should be considered, and suppressive therapy should be offered to HSV-seropositive individuals throughout the season. When a large scale outbreak affecting multiple teams occurs in mid-season, a minimum 8-d suspension of all skin-to-skin wrestling contact is required to interrupt the increase in infections.

The following protocols describe treatment for HG outbreaks and prophylactic treatment:

- Recommended treatment of primary HG is 1 mg oral valacyclovir (Valtrex®; GSK) twice daily for 10–14 d or 200–400 mg oral acyclovir (Zovirax®; GSK) five times daily for 10–14 d. No studies have been performed to determine the appropriate dosing parameters for famciclovir (Famvir®; Novartis).

- For outbreaks of recurrent HG, the recommended treatment is oral valacyclovir 500 mg twice daily for 7 d or oral acyclovir 200–400 mg five times daily for 7 d. No studies have been performed to determine appropriate dosing parameters for famciclovir.

- For suppressive therapy or those who are asymptomatic HSV-1-seropositive individuals, prophylaxis with acyclovir 400 mg twice daily or oral valacyclovir 500–1000 mg daily is recommended for the duration of the wrestling season. For individuals with less than a 2-yr history of HG infection, the higher dose of valacyclovir (1 GM QD) is recommended for suppressive therapy. No studies have been performed to evaluate famciclovir as suppressive therapy in this population.

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