From wrestlers to runners, athletes of all ages are prone to a variety of infectious dermatoses and cutaneous injuries. While these conditions respond well to available therapies, it’s critical to accurately identify dermatoses, implement treatment, prevent the spread of infection, and minimize the risk of recurrence. The following review of diagnosis and treatment of various common sports dermatoses will help you manage your patients more effectively.

**Herpes Gladiatorum/Rugbeiorum**

Herpes simplex is an epidemic problem for athletes, occurring in up to 40 percent of wrestlers (herpes gladiatorum, HG) and many rugby players (herpes rugbeiorum, HR). Transmission appears to occur predominately through direct skin-to-skin contact and not indirectly through mats and equipment. In a 2001 study of an outbreak at a 28-day wrestling camp, 96 percent of herpes lesions occurred on the ventral surface of the body. Most cases (72 percent) involved lesions of the major lock-up position contact areas (face and neck), and the majority of wrestlers who were right-handed had right-sided lesions. This same study reported that herpes gladiatorum carries nearly a 33 percent probability of transmission from one wrestling opponent to another. Individuals typically have a period of almost seven days from exposure until outbreak; during this time, transmission to others is possible. Since wrestlers may compete more than once within seven days, this increases chances of spread.

As athletes are often infectious before frank skin lesions are visible, it is reasonable to diagnose herpes gladiatorum/rugbeiorum in an athlete with symptoms of malaise, sore throat, fever, or adenopathy along with history of direct contact of an infected individual. Skin manifestations need not be evident, and in questionable cases of visible outbreak, a Tzanck preparation or culture will help to confirm HSV infection (although neither test is 100 percent sensitive). Polymerase chain reaction (PCR) and direct immunofluorescence assay (DFA) are more sensitive tests, but cost and availability limit their use.

Herpes gladiatorum outbreaks are self-limited and usually last 10-14 days; but antivirals such as valacyclovir for seven to 10 days (1g twice daily for primary outbreaks; 500mg twice daily for a recurrence) can shorten the course of symptoms and transmission. Recent work suggests that valacyclovir 2g twice in one day only may be effective for herpes labialis. The effectiveness of this
regimen has not specifically been studied in HG/HR. Other medications such as acyclovir and famciclovir may be used as alternatives but may be less effective because of more frequent dosing regimens and decreased compliance.\(^1\) All of these agents are most effective if started at the onset of symptoms. Topical agents seem less effective.

While it is prudent to treat outbreaks of herpes gladiatorum, prevention is perhaps more important. Athletic trainers or coaches should carefully examine athletes or athletes should examine themselves before practices and competitions. Immediately discontinue the athlete’s participation in practice and competition at the earliest sign of infection, either cutaneous or systemic. Start oral antivirals immediately in suspected cases and screen contacts for infection. In general, good hygiene among athletes is necessary: they should shower after practice and should not share towels or equipment (especially headgear). Consider recommending a requirement that practice clothing be made of soft, synthetic, wicking material as one study proposed that abrasive practice shirts may contribute to the spread of HG.\(^5\)

The National Federation of State High School Associations (NFHS) recently developed a physician release for wrestlers with skin lesions. On this form, they recommend at least five full days (120 hours) of oral antivirals with no new lesions for 72 hours and all lesions scabbed over before permitting return to practice and competition. The clearance form also notes that covering active lesions is not adequate.\(^6\) The NCAA has similar guidelines. Before competing:

1) lesions must be completely dry and covered by a firm adherent crust and

2) the wrestler must be on oral antiviral therapy for at least 120 hours.

If the infection is primary, the wrestler must also be free of systemic symptoms and have developed no new blisters for at least 72 hours. The NCAA agrees that covering active herpetic infections is inadequate.\(^7\) NCAA guidelines do recommend considering prophylactic treatment through the season for athletes with a history of outbreaks. One study showed that valacyclovir 500mg daily was effective if the athlete had no previ-
ous history of herpes or an outbreak that occurred more than two years ago; 1000mg daily was effective if an outbreak occurred within the past two years.4

**Tinea Corporis Gladiatorum**

Tinea corporis is also a problem for wrestlers. Studies have shown that 24-77 percent of individuals on wrestling teams are infected.3 Like HG, tinea corporis gladiatorum (TCG) seems to be predominately spread through skin-to-skin contact with the majority of lesions found on the head, neck, and upper extremities.3 Host factors such as sweaty, macerated skin and skin abrasions or irritation may increase risk of transmission.3 The majority of tinea corporis gladiatorum outbreaks have been caused by Trichophyton tonsurans.6,9 Tinea corporis gladiatorum may be diagnosed clinically, but infections often present atypically as a scaling, well-defined plaque without evidence of the classic ring-shaped lesion.9 Use culture or KOH preparation of skin scrapings to confirm the diagnosis in unclear cases.

Treatment for TCG consists of oral and/or topical antifungals.4,9 Consider oral therapy in the case of extensive disease, failure of topical therapy, or immunocompromised hosts. The most efficacious therapeutic agent has yet to be determined, but one study comparing topical clotrimazole and oral fluconazole recommends oral fluconazole 200mg once weekly as first line therapy.10 The optimal duration of therapy remains uncertain, but in the same study, 60 percent of wrestlers had negative cultures at seven days, and all wrestlers had negative cultures at four weeks.

NFHS recommends oral or topical therapy for seven days before resuming participation.6 NCAA guidelines suggest:
1) skin lesions be treated for a minimum of 72 hours with topical terbinafine or naftifine before return to competition and
2) wrestlers be disqualified if they have extensive or active lesions (as determined by KOH prep or review of therapy).7

In contrast to herpes gladiatorum, both the NFHS and the NCAA believe an occlusive dressing provides adequate protection in cases of localized tinea corporis gladiatorum, although a specific regimen should be followed.2,11 The athlete must wash the lesions with selenium sulfide or ketoconazole shampoo and then apply naftifine gel/cream or terbinafine cream followed by a gas-permeable dressing such as Opsite (Smith and Nephew) or Bioclusive (Johnson & Johnson). Finally, the athlete or his/her trainer must wrap the dressing with ProWrap (Fabrifoam) and stretch tape. Change dressings after each match.7

As with HG, prevention is a critical component in the management of tinea corporis gladiatorum. Carefully examine athletes before practice and competition, immediately initiate treatment, and encourage good hygiene among athletes as similarly outlined for HG. Consider pharmacologic prophylaxis in those athletes with recurrent outbreaks: fluconazole 200mg once weekly or itraconazole 400mg biweekly.3

**Impetigo/Furunculosis**

Caused by streptococci and staphylococci, impetigo and furunculosis also affect athletes in contact sports such as wrestling, rugby, and football.1 A recent study of a methicillin-resistant S. aureus (MRSA) impetigo outbreak among football players identified several possible risk factors for the infection: skin trauma, skin-to-skin contact, poor hand hygiene among trainers, skipping showers, and sharing towels.12 Similar risk factors (contact and prior skin injury) seem to play a role in the development of furunculosis.3 The typical lesion seen in impetigo is a well-defined erythematous plaque with an adherent yellow crust. Athletes with furunculosis usually present with multiple, red, parafollicular nodules distributed on the upper extremities.3 A clinical diagnosis is appropriate with either impetigo or furunculosis, but physicians may confirm questionable cases with bacterial cultures of lesions.

Treatment of either infection consists of oral and topical antibiotics. Initiate treatment with mupirocin twice daily along with dicloxacin (500mg three times daily) or cefalexin (500mg three times daily). Penicillin-allergic athletes should use erythromycin.3 In the study involving MRSA impetigo, isolates were susceptible to tetracycline, clindamycin, ciprofloxacin, trimethoprim-sulfamethoxazole, and vancomycin.11 Physicians, therefore, may consider one of these antimicrobials as an alternative in resistant cases of impetigo. In addition to pharmacologic treatment, drain any abscesses and begin frequent warm water soaks in cases of furunculosis.3 Failure to treat either impetigo or furunculosis places the infected athlete at risk for post-streptococcal glomerulonephritis.4 For wrestling, the NFHS suggests that athletes be treated with an antibiotic for two days and have no draining, oozing, or moist lesions.4 NCAA guidelines recommend that a wrestler:
1) have been without any new skin lesion for 48 hours before the competition,
2) have completed at least 72 hours of antibiotic therapy, and
3) have no moist, exudative or draining lesions at time of competition.7

As with HG, the NCAA discourages covering active infections to allow participation.

**Pitted Keratolysis**

Pitted keratolysis is an infection of the feet caused primarily by Corynebacterium sp.15,14 The problem is not unique to athletes but is quite common among them. Athletes with this disorder often present with crater-like depressions on the plantar surfaces of the feet, mainly in pressure-bearing areas. Those affected may also complain of a foul odor, plantar discoloration, and hyperhidrosis. Less frequently, affected areas may be painful or pruritic.

Treatment consists of oral or topical antibacterial agents as
well as elimination of moisture. Start with topical erythromycin or clindamycin solution or gel twice daily. If the patient shows no improvement with topicals, try oral erythromycin. Other options include topical 5% formalin, mupirocin, benzoyl peroxide, or salicylic acid. Advise patients to use socks made of an absorbent, wicking material, and consider aluminum chloride antiperspirant. One study even advocates the use of botulinum toxin to treat severe cases of hyperhidrosis.

**Scabies**
The incubation period of scabies is three weeks in primary cases and as little as 24 hours with subsequent cases. Athletes often present with itching that is worse at night, a rash consisting of linear burrows and/or small vesicles, and history of exposure to itching teammates or household contacts. Areas likely involved include axillary skinfolds, digital web spaces, the flexor surface of the wrists, the genitalia, and gluteal folds. Clinicians should confirm scabies with microscope examination of a skin scraping in mineral oil. The presence of a mite, eggs, or feces clinches the diagnosis.

The standard treatment for scabies is topical 5% permethrin cream, which is nearly 100 percent effective. After bathing, massage the cream over the entire body from the chin downwards, and then wash off the cream after eight to 14 hours. Some suggest that this process should be repeated one week later, but a single treatment seems to be effective for most patients. Though lindane is an alternative effective treatment, the risk of neurotoxicity requires that it generally be avoided, especially in athletes with skin breaks.

As with HG and TCG, the primary risk factor for scabies in athletes is skin-to-skin contact; but unlike other infections, transmission through fomites is also possible. It is necessary, therefore, to treat all teammates and household contacts with permethrin as well as to decontaminate all towels, linens, and clothing used in the athletes’ homes and practice facilities by hot-water washing and hot drying.

Before return to competition, the NFHS suggests at least 24 hours of appropriate topical treatment. The NCAA advises a negative scabies prep at the time of competition.

**Pediculosis**
As with scabies, itching is often the presenting complaint of those with pediculosis (lice) and may take over two weeks to develop. Infestation may occur on the head or body and transmission is usually a result of close contact in either case. Diagnosis depends on identification of a single louse or viable nit. An empty nit is not sufficient for diagnosis of active infestation.

Examine teammates and treat infested athletes with a lotion or gel from the pyrethrum group of insecticides (1% or 5% permethrin or 4% piperonyl butoxide-0.33% pyrethrin). Some authors recommend avoiding shampoo forms of treatment since contact time is shorter and water reduces penetration. Apply the pediculicide to dry hair for seven days, and use a fine comb to remove nits. Repeat the treatment seven to 10 days after the first course to kill newly hatched lice. To prevent spread, it is also necessary to wash in hot water and dry on high heat all contaminated clothing, towels, and linens in the athlete’s home and practice facility. Soak all combs and brushes in rubbing alcohol or hot water for at least 10 minutes.

NFHS guidelines for head lice are the same as those for scabies: 24 hours of topical treatment before return to competition. NCAA guidelines are more stringent and state that for wrestling, a wrestler must be treated with an appropriate pediculicide and then re-examined for completeness of response before resuming competition. Note that noninfectious nodules may persist for several weeks and should not prevent participation.

**Frostbite**
Among all athletes, joggers are most commonly affected by frostbite injury, but any outdoor athlete is at risk during colder weather if he or she does not take precautions. Specific risk factors include ambient temperatures below 0 °C, high humidity and rain, increased wind chill, high altitude, and prolonged exposure to the environment. Depending on the presence and severity of these risk factors, frostbite can occur in a time period of minutes to hours, initially manifesting as pain in the toes, fingers, ears, and face. Eventually affected areas become numb and white/blue; with rewarming, they exhibit hyperemia and blistering.

Treatment of choice for frostbite is rapid rewarming: place the affected areas in a water bath of 38-44 °C for 20 minutes. If the patient requires transport, remove wet clothing but delay rewarming and unintentional thawing until transport is complete. If blisters are present, debride white blisters after rewarming and cover with aloe. Do not debride hemorrhagic blisters; simply cover them with aloe.

While treatment is often necessary, in many cases frostbite can be prevented—primarily with sensible precautions.
Friction Bullae

Almost every athlete will complain of friction bullae, or blisters. Friction from repetitive rubbing results in intraepidermal splits, which fill with blood or tissue transudate.18,21 Warmth and moisture (sweating, running in the rain or puddles) increase the risk for friction blisters, making the feet the most common site of blisters.19 Poor fitting shoes also significantly contribute to the problem.18,21

Prevention, therefore, should begin with properly fitting athletic shoes. Decrease friction with socks made of wicking material, or wear two pairs of socks made of different fabrics. Avoid cotton socks. Other suggestions for prevention include applying petroleum jelly or antiperspirant as well as 10% tannic acid to harden the skin in areas commonly involved.21

To treat an acute blister, make a small incision at the periphery with a sharp, sterile instrument, taking care to leave the blister roof intact.13,21 This procedure to drain the blister contents may be repeated up to three times in the first 24 hours.15 If the blister becomes unroofed, apply a dressing. Many synthetic dressings available provide excellent protection; most, however, are somewhat expensive.3

To prevent recurrence of friction bullae, keep the area well lubricated to reduce friction. In general, athletes improve with application of an adhesive bandage or clear tape to the blister to avoid direct pressure.21

If the blister is large, it may be difficult to manage with a dressing. In this case, the blister can be incised with a sterile instrument and a small amount of pressure applied to the base of the blister to drain the fluid.21

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and Radiesse, in sensitive patients. Nerve blocks have the advantage of producing complete anesthesia while causing minimal alterations in superficial contours.

The infraorbital nerve provides sensation in the lower lid, cheek, lateral nose, and upper lip. This purely sensory nerve exits the maxilla through the infraorbital foramen, approximately one centimeter inferior to the orbital rim at the midpupillary line.

To block the infraorbital nerve, we prefer an intraoral approach. First, the infraorbital foramen is palpated with the middle finger of the nondominant hand. The upper lip is then elevated with the thumb and index finger. A 25-gauge needle is then inserted into the superior labial sulcus at the apex of the canine fossa and advanced to the infraorbital foramen just beneath the middle fingertip. After withdrawing gently to rule out intravascular needle placement, approximately 1-2cc of 2% lidocaine with epinephrine (1:100,000) is injected. If paresthesias develop, the needle tip should be withdrawn slightly before continuing with the injection.

The mental branch of the mandibular nerve provides sensation of the lower lip and chin. This nerve exits the mental foramen of the mandible at the midpupillary line, deep to the depressor anguli oris. The mental nerve may be blocked in a similar fashion to the infraorbital nerve. The needle should, however, be placed in the inferior labial sulcus at the base of the first bicuspis and advanced to meet the middle finger of the non-dominant hand placed over the mental foramen.

Nerve blocks in either location create profound anesthesia within 15 minutes of injection that may last between three and four hours. During this time, patients may have mild difficulty with speaking and drinking liquids. Because of the anesthesia at the lips, patients should additionally be advised to avoid hot beverages and chewy foods until sensation returns to normal. Despite these inconveniences, we have found nerve blocks to be very well received by patients.

Each of these techniques is easy to learn and may substantially improve patient acceptance of cosmetic procedures. By appropriately choosing from among them, we as dermatologists can maximize patient comfort and satisfaction.

New in Your Practice

Hot Shot. Vaccination against varicella-zoster virus (VZV) may decrease the risk of developing postherpetic neuralgia (PHN), says a recent study involving 38,546 patients (NEJM 352:2271-84). An investigational live attenuated Oka/Merck VZV vaccine reduced the incidence of PHN by 66.5 percent, the burden of illness due to herpes zoster by 66.1 percent, and the incidence of herpes zoster by 51.3 percent.