

Dermatologic Disorders of the Athlete

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Abstract

The most common injuries afflicting the athlete affect the skin. The list of sports-related dermatoses is vast and includes infections, inflammatory conditions, traumatic entities, environmental encounters, and neoplasms. It is critical that the sports physician recognises common and uncommon skin disorders of the athlete. Knowledge of the treatment and prevention of various sports-related dermatoses results in prompt and appropriate care of the athlete.

Infections probably cause the most disruption to individual and team activities. Herpes gladiatorum, tinea corporis gladiatorum, impetigo, and furunculosis are sometimes found in epidemic proportions in athletes. Vigilant surveillance and early treatment help teams avoid these epidemics. Fortunately, several recent studies suggest that pharmacotherapeutic prevention may be effective for some of these sports-related infections. Inflammatory cutaneous conditions may be banal or potentially life threatening as in the case of exercise-induced anaphylaxis. Athletes who develop exercise-induced anaphylaxis may prevent outbreaks by avoiding food before exercise and extreme temperatures while they exercise. Almost all sports enthusiasts are at risk of developing traumatic entities such as

nail dystrophies, calluses and blisters. Other more unusual traumatic skin conditions, such as talon noire, jogger's nipples and mogul's palm, occur in specific sports. Several techniques and special clothing exist to help prevent traumatic skin conditions in athletes. Almost all athletes, to some degree, interact with the environment. Winter sport athletes may develop frostbite and swimmers in both fresh and saltwater may develop swimmer's itch or seabather's eruption, respectively. Swimmers with fair skin and light hair may also present with unusual green hair that results from the deposition of copper within the hair. Finally, athletes are at risk of developing both benign and malignant neoplasms. Hockey players, surfers, boxers and football players can develop athlete's nodules. Outdoor sports enthusiasts are at greater risk of developing melanoma and non-melanoma skin cancer. Athletes spend a great deal of time outdoors, typically during peak hours of ultraviolet exposure. The frequent use of sunscreens and protective clothing will decrease the athlete's sun exposure. It is critical that the sports physician recognises common and uncommon skin disorders of the athlete. Knowledge of the treatment and prevention of various sports-related dermatoses results in prompt and appropriate care of the athlete.

1. Infections

Skin infections, among the most common sports-related dermatoses, may result in significant individual morbidity and team disruption. Fungi, bacteria, viruses, and parasites cause these infections (see table I).

1.1 Fungi

Fungal infections plague wrestlers, often in epidemic proportions. Tinea corporis affecting wres-

tlers is termed tinea corporis gladiatorum (table II).^[1] Epidemiological studies have shown that 24 to 77% of individuals in wrestling teams are infected.^[1-3] The wide range of infection rates may be explained by the varying methodologies of the studies. *Trichophyton tonsurans* is the predominant fungus responsible for tinea corporis gladiatorum and is transmitted through close skin-to-skin contact occurring in wrestling.^[1,4] In the US, *T. tonsurans* is the leading cause of tinea capitis, and some authors believe that wrestlers with asymptomatic tinea capitis may be reservoirs for transmission.^[4] Wrestling mats probably do not play a major role in the transmission of infection,^[1,2] although some researchers have cultured fungal organisms from mats.^[5] Several host factors are also important in infectious transmission, including macerated skin from sweating, abrasions, and occlusion by equipment, all of which increase the probability that a wrestler will develop fungal lesions.

Tinea corporis gladiatorum presents as well-defined, erythematous, scaling plaques (figure 1). Whereas most typical tinea corporis exhibits a ring-shaped appearance, tinea corporis gladiatorum usually does not.^[4] This infection predominantly affects the head, neck, and upper extremities; it only rarely affects the lower extremities.^[1,4,6] The diag-

Table I. Infections in the athlete

Fungal
Tinea corporis
Tinea pedis
Viral
Herpes simplex
Warts
Bacterial
Impetigo
Furunculosis
Pitted keratolysis
Hot tub folliculitis
Atypical mycobacterial
Swimming pool granuloma
Parasitic
Cutaneous larva migrans

Table II. Dermatologic conditions seen in sports with skin-to-skin contact

Sport	Furunculosis	Impetigo	Herpes rugbeiorum	Herpes gladiatorum	Tinea corporis gladiatorum
Basketball	X				
Football	X	X			
Rugby		X	X		
Wrestling	X	X		X	X

nosis of tinea corporis gladiatorum is often straightforward. To confirm the diagnosis, the clinician may culture the lesion or scrape the lesion for direct microscopic visualisation of the hyphae (a technique using potassium hydroxide).

Treatment of this condition consists of both topical^[7] and oral antifungal agents.^[7,8] One study suggested that 100% of athletes treated with weekly fluconazole (100mg) had negative cultures after 3 weeks (table III);^[8] unfortunately the optimal duration of therapy has yet to be defined. Prevention of tinea corporis gladiatorum is paramount. Athletes should not share equipment or towels. Those athletes who are infected should be benched if the lesion cannot be bandaged. While this approach may be permissible in practice, many athletic associations do not allow wrestlers to compete if any infectious lesions exist, regardless of coverage. Pharmacological prevention of tinea corporis gladiatorum shows promise; weekly fluconazole (200mg)^[8] or bimonthly itraconazole (400mg)^[3] appears to reduce the transmission of the fungus (table III).

Fungal organisms also frequently infect athletes' feet. Several factors put athletes at risk for the development of such infections, including occlusion, trauma, shower sharing, and sweating with resultant maceration of the epidermis.^[14] The organisms most frequently causing tinea pedis are *T. rubrum* and *T. mentagraphytes*.^[15] Tinea pedis may have a myriad of clinical features; one subtype presents with scale, with or without erythema, along the lateral aspect of the sole, whereas another presents with inflammatory vesicles, typically on the instep. A further subtype is characterised by erythematous, scaling plaques in the interdigital areas.^[16] The diagnosis is often straightforward. Early lesions, however, may be confused with allergic contact dermatitis, atopic dermatitis, psoriasis, or bacterial or viral

infections. Potassium hydroxide examination of skin scrapings or culture confirms the diagnosis. Treatment includes topical antifungal creams for mild disease or oral antifungal agents for severe infections. Athletes may prevent tinea pedis by using synthetic socks to keep their feet dry and by wearing sandals in the locker room and showers.

1.2 Viruses

Herpes simplex virus (HSV) infection has been reported in epidemic proportions in rugby (herpes rugbeiorum) and wrestling (herpes gladiatorum) [table II].^[9,17-21] Herpes gladiatorum may occur in up to a third of wrestlers;^[20] differences in reported infection frequencies result from the use of varying study methodologies. The same unique risk factors that make wrestlers susceptible to fungal infection promote HSV infection. Grouped vesicles on an erythematous base characterise HSV infection, although early lesions may exhibit no vesicles and late lesions may demonstrate only erosions (figure 2).^[9,21] Typically the lesions are located on the head, neck and upper extremities.^[9,20,21] Tzanck smear,



Fig. 1. A lesion of tinea corporis gladiatorum is seen on the arm.

Table III. Pharmacologic treatment and prophylaxis of infections in athletes

Infection	Treatment	Prophylaxis
Tinea corporis gladiatorum	Fluconazole (100mg qw) ^[8]	Fluconazole (200mg qw), ^[8] itraconazole (400mg q2w) ^[3]
Herpes gladiatorum	Famciclovir (250mg tid), ^[9] Valaciclovir (1g bid)	Valaciclovir (500mg od) ^[10]
Impetigo	Mupirocin (bid), Dicloxacillin (500mg tid), cefalexin (500mg tid) ^[11,12]	None
Furunculosis	Mupirocin (bid), Dicloxacillin (500mg tid) ^[11,12]	None
Cutaneous larva migrans	Thiabendazole (occluded qhs), ^[13] Ivermectin (12mg once)	None

bid = twice daily; **od** = once daily; **qhs** = at bedtime; **qw** = every week; **q2w** = every other week; **tid** = 3 times daily.

culture and immunofluorescence, if available, are used to confirm the diagnosis. When the lesions lack characteristic vesicles, the differential diagnosis includes tinea corporis gladiatorum, impetigo, acne and atopic dermatitis.

Antiviral agents, especially valaciclovir (1g twice daily) and famciclovir (250mg three times daily), are effective for treating HSV infection (table III).^[9] The duration of treatment before an athlete may return to competition is controversial (table IV). To prevent transmission, wrestlers should not share equipment or towels. While bandaging isolated lesions may enable wrestlers to practice safely, covering the infection is not acceptable at many competitions. If the infection cannot be adequately covered during practice, the athlete should not participate. Some authors have proposed the prophylactic use of valaciclovir (table III). A double-blind placebo-controlled trial revealed that wrestlers with a history of HSV infection avoided further outbreaks by

taking valaciclovir (500 mg/day); 33% of the wrestlers taking placebo developed herpes gladiatorum.^[10]

Human papilloma virus infection is of concern to athletes when it causes warts on the hands and feet.^[11,23] Warts are verrucous, well defined, occasionally painful, papules and plaques usually present on the soles and hands. Warts should be differentiated from calluses and corns. Paring the overlying hyperkeratotic material will reveal pinpoint black spots (pericapillary haemorrhages) in warts, a central core in corns and no abnormality in calluses.^[11] Clinicians typically destroy warts using various methods such as cryosurgery, salicylic-acid and cantharidin; however, these techniques may prevent the athlete from returning immediately to their sport. The new immunomodulating topical agent imiquimod may be quite effective without inducing the pain that often precludes the use of destructive methods in athletes that cannot miss practice. Athletes should wear sandals in the locker room and showers to both prevent transmission and avoid acquiring the virus.

1.3 Bacteria

Impetigo contagiosum is a bacterial infection caused by both *streptococci* and *staphylococci* species.^[16,24,25] Impetigo infects athletes with close skin-to-skin contact such as wrestlers and football and rugby players (table II).^[25] There are currently no epidemiological studies of impetigo in athletes. Physical examination reveals well defined, erythematous, yellow, crusted plaques distributed on the extremities and the head and neck.^[11] The differential

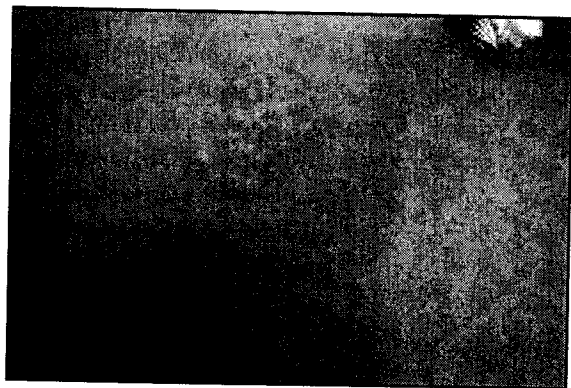


Fig. 2. The grouped vesicles on an erythematous base are characteristic of herpes gladiatorum.

diagnosis, especially of small lesions, includes atopic dermatitis, acne, herpes gladiatorum and tinea corporis gladiatorum. Treatment includes both topical and oral antibiotics such as mupirocin (twice daily) and dicloxacillin (500mg three times daily) or cefalexin (500mg three times daily); erythromycin may be used for those athletes who are allergic to penicillins (table III). Individuals with infection should be kept from sporting activities that involve skin-to-skin contact until 5 days after the initiation of therapy, although no evidence-based studies support this recommendation (table IV).^[22] Athletes can reduce transmission by not sharing equipment or towels. No evidence exists to suggest that equipment is a reservoir.

Staphylococcus and *Streptococcus* may also infect an athlete's follicles and cause furunculosis.^[9,24-26] In most cases *Staphylococcus* is methicillin sensitive; however, in one study, methicillin-resistant-*Staphylococcus aureus* was cultured from 22% of wrestlers' furuncles.^[27] An epidemiologic study found that up to a quarter of football players and 20% of basketball players develop furunculosis.^[28] This same study determined that contact with furuncles and prior skin injury were the main risk factors in the development of furunculosis. This infection is characterised by well defined, erythematous, tender nodules that are distributed on the upper extremities.^[11] Complications, though rare, can be serious; post-streptococcal glomerulonephritis has occurred in athletes with furunculosis and is termed 'scrum kidney'.^[9,26]

Treatment should be instituted promptly. This therapy is similar to that for impetigo and consists of topical and oral antibiotics (mupirocin twice daily and dicloxacillin 500mg three times daily) and fre-



Fig. 3. Distinctive pits seen in pitted keratolysis.

quent warm soaks (table III).^[12] Epidemics should alert the clinician to possible nasal carriage of *Staphylococci* species.^[9] Mupirocin ointment applied to both nares twice daily for 1 week should clear *Staphylococcus* carriage for ~6 months. To permit minimal disruption of a team's activities, athletes, coaches and athletic trainers should proactively screen the athletes' skin daily. Implementing therapy and promptly isolating infected athletes decreases the number of missed practices and thwarts epidemics.

Pitted keratolysis is a unique bacterial infection that affects athletes with moist and occluded feet.^[11,24,29] Several micro-organisms including corynebacterium and micrococcus species^[11] may cause this condition. The diagnosis is made clinically and is characterised by discrete pits on the soles, often occurring with a strong odour (figure 3).^[29,30] Topical antibiotics (such as erythromycin or clindamycin) clear this eruption. Wearing synthetic socks and avoiding prolonged occlusion in athletic footwear reduces maceration and prevents pitted keratolysis.^[11,30]

Pseudomonas folliculitis (also known as 'hot tub folliculitis') occurs in athletes who use hot tubs and whirlpools often as a part of rehabilitation.^[31-33] The most common causative species identified has been *Pseudomonas aeruginosa* subtype O:11.^[31,33] Abrasions appear to put the athlete at increased risk for the development of infection. *Pseudomonas folliculitis* presents with pruritic, follicular, occasion-

Table IV. Length of time of therapy before athlete may return to sport^a

Disease	Length of disqualification
Tinea corporis gladiatorum	5-10 days ^[22]
Herpes gladiatorum/rugbeiorum	Until scabbed and dry ^[22]
Impetigo	5 days ^[22]

a Note that no evidence-based results directly support these recommendations.

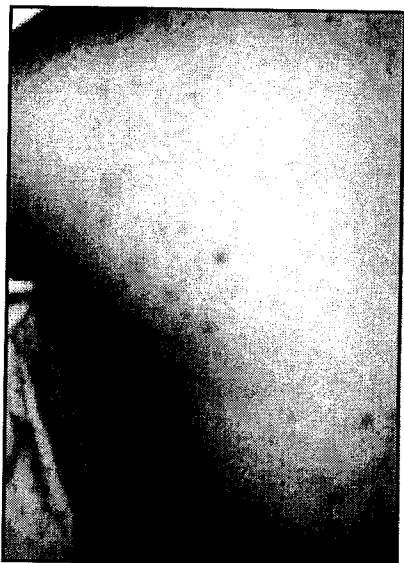


Fig. 4. These follicular papules and pustules characterise *Pseudomonas* ('hot tub') folliculitis.

ally green pustules on any submerged skin surface (figure 4).^[31-33] Some authors have noted a predilection for areas of skin covered by the bathing suit. Systemic manifestations, such as fever, chills and lymphadenopathy, may also occur.^[31-33] No treatment is necessary for the athlete who is immunocompetent and does not have systemic involvement. In fact, some studies have associated antibiotic treatment with increased disease recurrence.^[33] For athletes that require treatment, ciprofloxacin (250mg twice daily) appears effective.^[33] Athletic support staff should ensure adequate chlorine levels and proper pH (7.0 to 7.4) of the pool.^[11] Pools suspected to be infected should be drained and cleaned. Athletes should cover abrasions and cuts with occlusive dressings before entering whirlpools.^[33]

1.4 Atypical Mycobacteria

Infection with atypical mycobacteria is unusual in athletes but may occur in swimmers exposed to *Mycobacteria marinum* (table I).^[34,35] Epidemics of 'swimming pool granuloma' have occurred; one report noted 290 cases from a single pool.^[34] The diagnosis is often difficult because the morphology of the lesion may be nonspecific. Swimming pool

granuloma is characterised by brown-red, well defined papules and plaques on areas of the body that abrade the affected pool surfaces.^[34,35] Biopsy and culture confirm diagnosis; several weeks of clarithromycin (500mg twice daily) or another macrolide antibiotic will clear this infection.

1.5 Parasites

It is unusual for athletes to become infected with parasites. Cutaneous larva migrans is a hookworm that infects the lower extremities of sand volleyball players (table I).^[13] This infection results in linear red burrows that move several millimetres per day (figure 5). Topical thiabendazole (under occlusion at night) or oral ivermectin (12mg once) clears this infection (table III). Wearing footwear on the beach while playing sports will help prevent infection.

2. Inflammatory Conditions

2.1 Allergic Contact Dermatitis

Several aspects of sports participation put the athlete at risk for developing contact dermatitis. The hazards include equipment, topical medications, and flora in the playing field. Almost all equipment

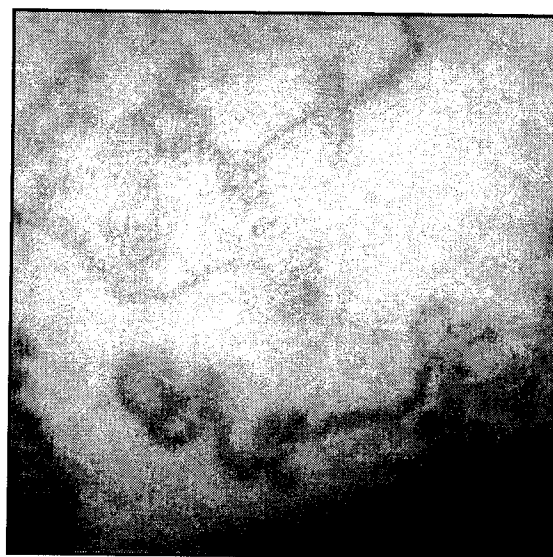


Fig. 5. Cutaneous larva migrans reveals linear and erythematous plaques.

Table V. Allergic contact dermatitis

Equipment	Sensitiser	Manufactured Alternative
Shoe insoles	Ethyl butylthiourea, mercaptobenzothiazole ^[36,38]	Polyurethane ^[36,38]
Wet suits, goggles	Ethyl butylthiourea ^[36,38]	Neoprene, polyvinyl chloride ^[36,38]
Underwater masks, swim caps	Mercaptobenzothiazole ^[36,38]	Silicone ^[36,38]
Athletic tape	Formaldehyde resin ^[38]	Paper tape, coban
Topical antiseptics/antibiotics	Benzocaine, neosporin ^[37]	Polysporin ^[37]

has the potential to sensitise the athlete;^[36] rubber is a particularly important allergic agent. Ethyl butylthiourea is found in shoe insoles, wet suits and goggles. Mercaptobenzothiazole is present in shoe insoles, underwater masks and swim caps.^[36] Athletes may be allergic to the formaldehyde resin in athletic tape and to topical antiseptics and antibiotics (benzocaine or neosporin).^[37] Golfers and other outdoor sports enthusiasts may encounter poison sumac and poison ivy. Diagnosis of allergic contact dermatitis is often clear from the patient's history and the characteristic erythematous and pruritic plaques.^[11,37] If needed for symptomatic relief, topical corticosteroids clear the eruption. Fortunately, alternative equipment materials are manufactured; shoe insoles can be made from polyurethane and goggles may be made from neoprene.^[36] Paper tape or coban may be used instead of athletic tape and silicone can be used in place of other materials in underwater masks and swim caps (table V).^[36]

2.2 Irritant Contact Dermatitis

Unlike allergic contact dermatitis, irritant contact dermatitis does not involve the immune system or antigen presentation. Soccer and rugby players may develop irritant contact dermatitis from calcium oxide, which is used in field markings;^[39] this

substance becomes a strong alkaline when mixed with water or sweat. Hockey players can develop irritant contact dermatitis from the fiberglass within hockey sticks.^[24,29] Cold (ice) packs can also cause irritant contact dermatitis if their ingredients are exuded through rough manipulation.^[29] Topical corticosteroids are effective in treating the eruption of irritant contact dermatitis.

2.3 Urticaria

Participation in several sports has led to urticaria (hives); running is a prominent precipitant. Urticaria is classified into cholinergic, cold, solar, pressure and rarely aquagenic varieties.^[40-42] Cholinergic urticaria is related to the elevated body temperature of the athlete. Cold urticaria presents in winter sport athletes, whereas solar urticaria may occur in any outdoor athlete who may be predisposed to the condition. The aquagenic variant has been reported in swimmers (table VI).^[40,41] Urticaria is clinically characterised by well defined, oedematous, erythematous plaques of varying sizes; it routinely resolves within 24 hours (figure 6). The efficacy of antihistamines in these disorders is variable;^[11] corticosteroids are often ineffective in the treatment of urticaria.^[11]

Table VI. Characteristic dermatoses in various sports

Sport	Centre's callosities	Acne mechanica	Exercise-induced anaphylaxis	Aquagenic urticaria	Green hair
Basketball	X				
Football		X			
Hockey		X			
Running			X		
Swimming				X	X

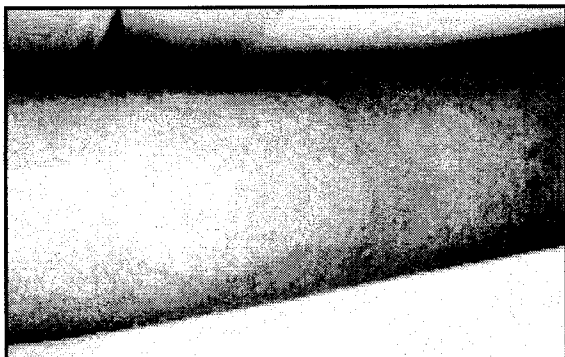


Fig. 6. After removing an ice cube from the athlete's arm, the clinician observes evidence of cold urticaria.

2.4 Exercise-Induced Anaphylaxis

The term exercise-induced anaphylaxis (EIA) may be somewhat misleading because not all cases result in respiratory or vascular collapse. In a large retrospective study of cases of EIA, 78% of participants noted that running induced their lesions (table VI). Eating before exercise predisposed athletes to develop lesions.^[43] Tennis, racquetball, bicycling, downhill skiing and basketball can also induce EIA. The pathophysiology of EIA is unclear but mast cell degranulation, mediated through immunoglobulin E, lactate or creatinine phosphokinase, results in histaminaemia.^[43] Athletes develop both cutaneous and systemic symptoms, with pruritus being an almost constant finding; angioneurotic-oedema, urticaria, respiratory difficulties, gastrointestinal symptoms (nausea, diarrhoea or colic) and vascular collapse occur in varying frequencies.

The acute treatment of EIA ensures vascular support and airway patency. Antihistamines and epinephrine are effective; β -agonist inhalers and corticosteroids are used but are of questionable benefit.^[43] Nearly a half of athletes with EIA reduce episodes by avoiding exercise in extremely hot, humid or cold weather. Approximately a third of patients diminish attacks by not eating before exercise.^[43] Also, avoidance of certain medications (such as aspirin or nonsteroidal anti-inflammatory drugs) decreases attacks of EIA. Cromolyn has been used to prevent pulmonary symptoms and ketotifen has been used to prevent dermatological symptoms.^[43]

3. Traumatic Entities

3.1 Nail Dystrophies

The nail plate and periungual area experience significant amounts of pressure and trauma during athletic activities. These forces differ with each sport, sometimes resulting in sport specific nail changes. Runners develop jogger's toe from repetitive thrusting of the longest toe (often the second toe) into the toebox;^[44] downhill courses can be particularly damaging. Clinically the toenail becomes thickened, ridged and discoloured (figure 7). The term 'tennis toe' is a general term describing the nail changes occurring from quick stops and restarts seen in racquet sports^[45] and basketball.^[46] In 'tennis toe', the longest toenail displays nail discolouration, nail thickening and transverse ridging; a callus may develop in the hyponychium. Soccer players develop unique nail dystrophies because the foot experiences a great deal of force with abrupt kicking. In addition to the nail changes of 'tennis toe', frank loss of the nail plate (avulsion) may occur in soccer players.^[47]

Clinicians commonly confuse sports-related nail dystrophies with onychomycosis and rarely with melanoma. Potassium hydroxide examination and culture rule out fungal infection; but if melanoma is suspected, a nail matrix biopsy should be performed. Suspicion for melanoma should be raised if the discolouration of the nail is also seen on the



Fig. 7. The most distal toenail reveals discolouration and thickening seen in jogger's toe.



Fig. 8. These are large calluses on the sole that could cause pain for an athlete.

periungual region (Hutchinson's sign).^[11] Treatment of nail dystrophies is not usually required; however, many athletes paint their nails dark colours for cosmetic purposes. Prevention is paramount; properly fitted footwear with a snug midfoot and adequate toebox along with properly trimmed straight-cut (not curved-cut) nails ensure equal distribution of forces and reduce the incidence of nail dystrophies.^[11,44]

3.2 Calluses and Blisters

Calluses are seen in almost every athlete. These hypertrophic areas develop in locations (often hands and feet) experiencing long-term, repetitive friction (table VI).^[48] Calluses may be confused with warts and corns (figure 8). The paring methods described earlier make the diagnosis quite clear. Hypertrophic calluses often confer a competitive advantage in sports like racquet sports or baseball. For this reason many athletes do not wish to treat calluses, but paring after soaking for several minutes reduces the thickness of the callus. Clinicians should examine calluses carefully because warts occasionally occur within them. Synthetic socks, petroleum jelly and well-fitted athletic wear decrease friction and help to prevent calluses.^[11]

Blisters are also common to almost all athletes. Acute friction causes these intraepidermal splits, which occur commonly on the hands and feet.^[11,48] Blisters are often inappropriately treated. Appro-

priate therapy includes preserving the blister roof with only a small incision made at the periphery to drain the fluid (figure 9). Antibiotic ointment is required only rarely.^[11,49] Although somewhat expensive, many synthetic dressings adhere firmly to the affected area and provide outstanding protection. Preventive measures focus on decreasing friction through the use of moisture wicking socks, petroleum jelly and appropriately fitted footwear.^[11,48,49]

3.3 Unique Traumatic Findings in Athletes

Friction in runners can result in a condition known as jogger's nipples.^[11,14,24,50] Repetitive friction between the nipples and the shirt results in painful, erythematous and crusted erosions (figure 10), which often create a dramatic display of lines of blood on the runner's shirt. Treatment includes the application of petroleum jelly or topical antibiotic ointment (erythromycin). Fortunately, applying petroleum jelly, commercially available patches or adhesive tape over the nipples before long runs are preventative. Semisynthetic or other soft fibre bras and shirts also help to prevent jogger's nipples.^[24,50] Frictional trauma has also caused alopecic patches in waterslide park enthusiasts. The resultant round patches can be confused with alopecia areata (figure 11); however, a recent history of watersliding



Fig. 9. The periphery of the bullae should be sterilely lanced and the roof kept in place to act as a biological dressing. Athletes can be taught this procedure to use themselves.

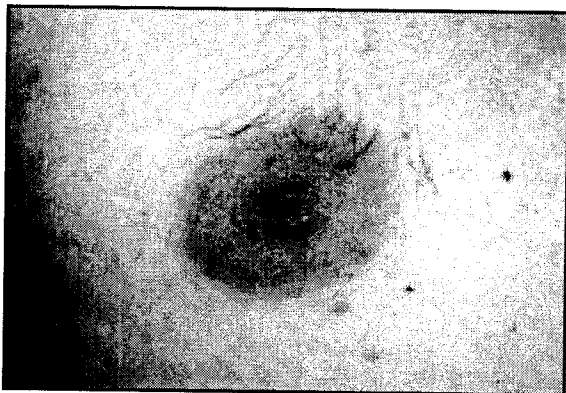


Fig. 10. These jogger's nipples are quite painful erosions.

clinches the diagnosis.^[51] Treatment is not necessary and hair will eventually regrow.

Talon noire^[14,52] and Mogul's palm^[53] are caused by intraepidermal bleeding from shearing forces applied to the skin. Talon noire is characterised by black macules particularly on the soles of young basketball players (figure 12). These macules may be confused with melanoma; however, the diagnosis is confirmed when paring with a surgical blade removes the old haemorrhage.^[14,51] If there is suspicion for melanoma, then a biopsy should be performed without paring the lesion. Mogul's palm, conversely, occurs on the hands of skiers and results from repetitive pole planting while downhill skiing.^[53] The affected palms demonstrate bruise like macules and patches. Treatment is not necessary for either condition, but the use of heel pads ameliorates talon noire.

Painful piezogenic pedal papules are herniations of fat typically on the posterolateral heel.^[14,54] The discomfort of painful piezogenic pedal papules is significant and often sidelines athletes. This diagnosis is confused with other causes of foot pain because the characteristic physical findings are only apparent during specific manoeuvres.^[54] The diagnosis is confirmed when the fat herniations are visualised as the athlete is asked to stand and apply pressure to the affected foot.^[16] No satisfactory treatment exists. Heel pads have been reported to help assuage the symptoms.^[16]

3.4 Acne Mechanica

Combinations of factors (friction, heat and occlusion) result in acne mechanica.^[30,54-56] This disorder occurs predominantly beneath heavy protective equipment [particularly in football and hockey players (table VI)] and is characterised by well defined, erythematous papules and pustules distributed on the shoulders, upper back and chin (figure 13).^[30,54-56] Acne mechanica may also develop beneath protective padding, for example, beneath kneepads in wrestlers (unpublished observation). Treatment of acne mechanica is more difficult than that of typical acne vulgaris. Keratolytic agents [3% salicylates and 8% resorcinol in 70% alcohol (ethanol)] have been useful. Some authors also add topical antibiotics (0.5% clindamycin).^[56] Immediate showers after practice and competitions are important preventive techniques. Moisture-wicking clothing worn under protective gear can also be useful.^[54,56]

4. Environmental Encounters

Outdoor athletes experience many different environmental conditions. Winter sports enthusiasts, for instance, risk developing frostbite. Persistent cold temperatures, coupled with wind and rain, initially lead to pain in acral areas; eventually frost-bitten areas become numb, progressing to blisters and necrosis.^[30,57] Rapid rewarming in a water bath



Fig. 11. The lateral aspects of this waterslide enthusiast reveals well defined alopecic patches caused by friction from the slide.

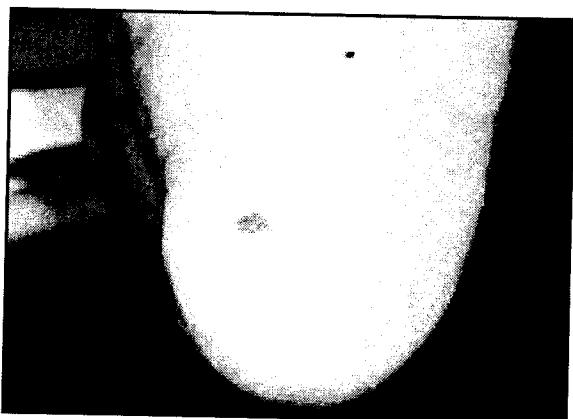


Fig. 12. This black macule on the sole is characteristic of talon noir and may be confused with melanoma.

of 38 to 44°C for 20 minutes is the treatment of choice for this condition. The athlete should dress in multiple layers and must remember that environmental factors can significantly decrease the insulation of clothing.^[30]

Athletes involved in water sports encounter multiple creatures; the type of dermatoses experienced as a result of contact with these creatures varies with salt versus fresh water. Seabather's eruption occurs in athletes in salt water (particularly off the coast of Florida, Long Island and the Caribbean) and develops as the result of stinging larvae of jellyfish and other marine animals.^[58,59] Swimmers in fresh water (particularly in the Northern US and Canada), conversely, develop swimmer's itch that is a hypersensitivity to cercarial schistosomes.^[59,60] Seabather's eruption presents with multiple, erythematous, pruritic papules in a bathing suit distribution;^[58,59] whereas swimmer's itch does not affect the bathing suit area.^[59] Antihistamines and oral or topical steroids can be used for the treatment of these dermatoses; however, they are not effective in all patients or for all infections.^[30] Showering after swimming in potentially infected waters prevents seabather's eruption.^[30]

Swimmers also develop an unusual condition known as 'green hair' (table VI). 'Green hair' results from copper deposition and not chlorine as is commonly thought.^[30,54,59] The source of the cop-

per is controversial; old leached pipes and copper-containing algicides are the most likely possibilities. Chelating-shampoos or 3% hydrogen-peroxide will clear the colour. Prevention of 'green hair' includes prompt hair washing after swimming and maintaining the pool pH between 7.4 and 7.6.^[30,54,59]

5. Neoplasms

Both benign and malignant neoplasms occur in athletes. The term athlete's nodule is a general term referring to myriad hyperplastic and reactive dermatoses in various athletes.^[30,61-63] Boxers develop athletes' nodules on the knuckles,^[64] and football and hockey players develop lesions on the ankles ('skate bite').^[30,61] Surfer's nodules can occur on the knee or the tibial prominence and dorsal aspects of the feet. While these nodules most likely result from recurrent friction, some nodules result from granulomatous reactions to sand embedded in the skin.^[61]

The diagnosis of athlete's nodule is often straightforward; however, the differential diagnosis can be vast and includes (depending on location) gout, ganglion cyst, epidermoid cyst, hypertrophic scar, granuloma annulare, callus and elastoma.^[63] In unclear cases a biopsy is recommended. Treatment includes intralesional steroids or excision.^[62,63] Wet suits allow surfers in cold water to lie prone on the board thus obviating surfer's nodule of the knee.^[61]

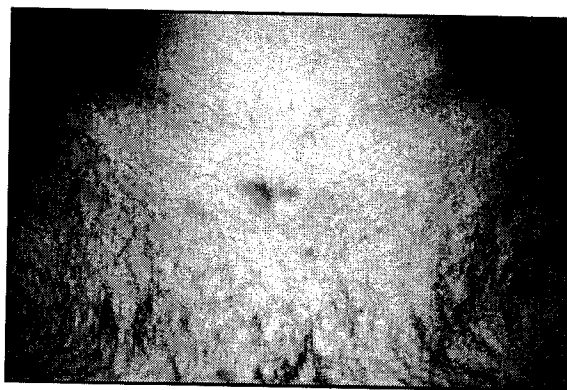


Fig. 13. The acne mechanica in the central chest resulted from involvement in weightlifting.

Athletes not only develop benign neoplasms, but also develop more serious malignant growths. These malignant neoplasms result from excessive exposure to the sun. Most individual exposure to ultraviolet radiation occurs before the age of 18 years. Young athletes, thus, are particularly at risk, but collegiate, recreational and professional athletes are also exposed to ultraviolet radiation into adulthood.^[24,29,65] Several studies have documented the unique exposures that place athletes at risk. One study^[66] revealed that the bicyclists in the 'Tour de Suisse' experience many times (17.2 in the mountain portion of the course and 8.1 overall in the course) the minimal dose necessary to induce a burn. Another study^[67] showed that skiers with an average skin type and without sunscreen, began to burn after only 6 minutes at 11 000 feet.^[67] Several epidemiologic studies have also shown a close association between skin cancers (melanoma, basal cell carcinoma and squamous cell carcinoma) and participation in water sports.^[68,69]

For practical purposes, it is often difficult to avoid outdoor sports during the peak sun exposure hours, between 10am and 2pm. Athletes must, however, take care to use the preventive techniques available. Unfortunately, many athletes do not apply sunscreen, and those who do initially apply it, do not reapply it after perspiration or water exposure. A variety of sunscreens exist. Lotions and sprays are well liked by athletes, as they do not tend to sting or feel greasy. Sunscreens promoted as 'waterproof' or 'sweatproof' do not uniformly protect throughout the day in all conditions. Coaches and trainers should become familiar with the various types of sunscreen and encourage their liberal use. Many outdoor athletes can wear hats during activities and should be encouraged to do so. Male athletes, particularly those who are young, should be discouraged from practising without wearing their shirts. If an athlete develops sunburn, the long-term damage cannot be altered, but warm soaks, petroleum jelly, sarna lotion, gauze and oral nonsteroidal anti-inflammatory agents (such as aspirin or indomethacin) may help assuage the acute phototoxic effects.

6. Conclusion

Myriad dermatoses afflict athletes, from the neophyte to the professional. Often these skin conditions not only affect individual performance, but team activities as well. Sports physicians, team trainers, coaches and the athletes themselves should be aware of common dermatoses. It is incumbent upon the sports physician not only to recognise these skin disorders, but also to treat them promptly and appropriately and to help institute preventive policies.

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References

1. Adams BB. Tinea corporis gladiatorum: a cross-sectional study. *J Am Acad Dermatol* 2000; 43: 1039-41
2. Beller M, Gessner BD. An outbreak of tinea corporis gladiatorum on a high school wrestling team. *J Am Acad Dermatol* 1994; 31: 197-201
3. Hazen PG, Weil ML. Itraconazole in the prevention and management of dermatophytosis in competitive wrestlers. *J Am Acad Dermatol* 1997; 36: 481-2
4. Kohl TD, Lisney M. Tinea gladiatorum. *Sports Med* 2000; 29: 439-47
5. El Fari M, Gräser Y, Presber W, et al. An epidemic of tinea corporis caused by *Trichophyton tonsurans* among children (wrestlers) in Germany. *Mycoses* 2000; 43: 191-6
6. Erős N, Károlyi Z, Molnár J. *Trichophyton equinum* infections among young wrestlers in Hungary. *Acta Derm Venereol* 1999; 8: 63-6
7. Kohl TD, Martin DC, Berger MS. Comparison of topical and oral treatments for tinea gladiatorum. *Clin J Sport Med* 1999; 9: 161-6
8. Kohl TD, Martin DC, Nemeth R, et al. Fluconazole for the prevention and treatment of tinea gladiatorum. *Pediatr Infect Dis J* 2000; 19: 717-22
9. Stacey A, Atkins B. Infectious diseases in rugby players. *Sports Med* 2000; 29: 211-20
10. Anderson BJ. The effectiveness of valacyclovir in preventing reactivation of herpes gladiatorum in wrestlers. *Clin J Sport Med* 1999; 9: 86-90
11. Adams BB. Sports dermatology. *Adolesc Med* 2001; 2: 305-22
12. Freeman MJ, Bergfeld WF. Skin diseases of football and wrestling participants. *Cutis* 1977; 20: 333-41
13. Biolcati G, Alabiso A. Creeping eruption of larva migrans: a case report in a beach volley athlete. *Int J Sports Med* 1997; 18: 612-3
14. Levine N. Dermatologic aspects of sports medicine. *J Am Acad Dermatol* 1980; 3: 415-24
15. Kemna ME, Elewski BE. A US epidemiologic survey of superficial fungal diseases. *J Am Acad Dermatol* 1996; 35: 539-42
16. Powell FC. Sports dermatology. *J Eur Acad Dermatol Venereol* 1994; 3: 1-15
17. Selling B, Kibrick S. An outbreak of herpes simplex among wrestlers (herpes gladiatorum). *N Engl J Med* 1964; 270: 979-82

18. Porter PS, Baughman RD. Epidemiology of herpes simplex among wrestlers. *JAMA* 1965; 194: 150-2
19. Becker TM, Kodsi R, Bailey P, et al. Grappling with herpes: herpes gladiatorum. *Am J Sports Med* 1988; 16: 665-9
20. 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: 906-10
21. Becker TM. Herpes gladiatorum: a growing problem in sports medicine. *Cutis* 1992; 50: 150-2
22. Dienst WL, Dightman L, Dworkin MS, et al. Pinning down skin infections. *Physician Sportsmed* 1997; 25: 45-56
23. Bergfeld WF. Dermatologic problems in athletes. *Prim Care* 1984; 11: 151-60
24. Conklin RJ. Common cutaneous disorders in athletes. *Sports Med* 1990; 9: 100-19
25. Brenner IKM, Shek PN, Shephard RJ. Infection in athletes. *Sports Med* 1994; 17: 86-107
26. Mast EE, Goodman RA. Prevention of infectious disease transmission in sports. *Sports Med* 1997; 1: 1-7
27. Lindenmayer JM, Schoenfeld S, O'Grady P, et al. Methicillin-resistant *Staphylococcus aureus* in a high school wrestling team and the surrounding community. *Arch Intern Med* 1998; 158: 895-9
28. Sosin DM, Gunn RA, Ford WL, et al. An outbreak of furunculosis among high school athletes. *Am J Sports Med* 1989; 17: 828-32
29. Kantor GR, Bergfeld WF. Common and uncommon dermatologic diseases related to sports activities. *Exerc Sport Sci Rev* 1988; 16: 215-53
30. Pharis DB, Teller C, Wolf JE. Cutaneous manifestations of sports participation. *J Am Acad Dermatol* 1997; 36: 448-59
31. Silverman AR, Nieland ML. Hot tub folliculitis: a familial outbreak of *Pseudomonas folliculitis*. *J Am Acad Dermatol* 1983; 8: 153-6
32. Chandrasekar PH, Rolston KVI, Kannangara W, et al. Hot tub associated dermatitis due to *Pseudomonas aeruginosa*. *Arch Dermatol* 1984; 120: 1337-40
33. Green JJ. Localized whirlpool folliculitis in a football player. *Cutis* 2000; 65: 359-62
34. Philpott JA, Woodburne AR, Philpott OS, et al. Swimming pool granuloma: a study of 290 cases. *Arch Dermatol* 1963; 88: 158-61
35. Sarnaik AP, Vohra MP, Sturman SW, et al. Medical problems of the swimmer. *Clin Sports Med* 1986; 5: 47-64
36. Fisher AA. Sports-related cutaneous reactions. Part II: allergic contact dermatitis to sports equipment. *Cutis* 1999; 63: 202-4
37. Leshaw SW. Itching in active patients. *Physician Sportsmed* 1998; 26: 47-53
38. Fisher AA. Sports-related allergic dermatitis. *Cutis* 1992; 50: 95-7
39. Fisher AA. Sports-related cutaneous reactions. Part III: sports identification marks. *Cutis* 1999; 63: 256-8
40. Mikhailov P, Berova N, Andreev VC. Physical urticaria and sport. *Cutis* 1977; 20: 381-90
41. Briner WW. Physical allergies and exercise. *Sport Med* 1993; 15: 365-73
42. Nichols AW. Nonorthopaedic problems in the aquatic athlete. *Clin Sports Med* 1999; 18: 395-411
43. Shadick NA, Liang MH, Partridge AJ, et al. The natural history of exercise-induced anaphylaxis: survey results from a 10-year follow-up study. *J Allergy Clin Immunol* 1999; 104: 123-7
44. Adams BB. Running-related toenail abnormality. *Physician Sportsmed* 1999; 27: 85-7
45. Montgomery RM. Tennis and its skin problems. *Cutis* 1977; 19: 480-2
46. Adams BB, Lucky AW. A center's callosities. *Cutis* 2001; 67: 141-2
47. Rzonca EC, Lupo PJ. Pedal nail pathology: biomechanical implications. *Clin Podiatr Med Surg* 1989; 6: 327-37
48. Basler RSW. Sports-related skin injuries. *Adv Dermatol* 1989; 4: 29-50
49. Basler RSW, Garcia MA. Acing common skin problems in tennis players. *Physician Sportsmed* 1998; 26: 37-44
50. Levit F. Jogger's nipples. *N Engl J Med* 1977; 297: 1127
51. Adams BB. Water-slide alopecia. *Cutis* 2001; 67: 399-400
52. Wilkinson DS. Black heel. *Cutis* 1977; 20: 393-6
53. Swinehart JM. Mogul skier's palm: traumatic hypothenar ecchymosis. *Cutis* 1992; 50: 117-8
54. Basler RSW. Skin injuries in sports medicine. *J Am Acad Dermatol* 1989; 21: 1257-62
55. Farber GA, Burks JW, Hegre AM. Football acne: an acneiform eruption. *Cutis* 1977; 20: 356-60
56. Basler RSW. Acne mechanica. *Cutis* 1992; 50: 125-8
57. D'Ambrosia RD. Cold injuries encountered in a winter resort. *Cutis* 1977; 20: 365-8
58. Burnett JW. Seabather's eruption. *Cutis* 1992; 50: 98
59. Basler RSW, Basler GC, Palmer AH, et al. Special skin symptoms seen in swimmers. *J Am Acad Dermatol* 2000; 43: 299-305
60. Hoeffler DF. Swimmer's itch (Cercarial dermatitis). *Cutis* 1977; 19: 461-7
61. Cohen PR, Eliezri YD, Silvers DN. Athlete's nodules. *Sports Med* 1990; 10: 198-203
62. Cohen PR, Eliezri YD, Silvers DN. Athlete's nodules. *J Am Acad Dermatol* 1991; 24: 317-8
63. Cohen PR, Eliezri YD, Silvers DN. Athlete's nodules: sports-related connective tissue nevi of the collagen type (collagenomas). *Cutis* 1992; 50: 131-5
64. Kanerva L. Knuckle pads from boxing. *Eur J Dermatol* 1998; 8: 359-61
65. Basler RSW. Skin lesions related to sports activity. *Prim Care* 1983; 10: 479-94
66. Moehrl M, Heinrich L, Schmid A, et al. Extreme UV exposure of professional cyclists. *Dermatology* 2000; 201: 44-5
67. Rigel DS, Rigel EG, Rigel AC. Effects of altitude and latitude on ambient UVB radiation. *J Am Acad Dermatol* 1999; 40: 114-6
68. Herzfeld PM, Fitzgerald EF, Hwang SA, et al. A case-control study of malignant melanoma of the trunk among white males in upstate New York. *Cancer Detect Prev* 1993; 17: 601-8
69. Rosso S, Zanetti R, Martinez C, et al. The multicentre south European study 'Helios' II: different sun exposure patterns in the aetiology of basal cell and squamous cell carcinomas of the skin. *Br J Cancer* 1996; 73: 1447-54

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