Exercise Immunology And Infectious Diseases In Athletes: A Clinically Relevant Review

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Citation

S Bryan, S Barton. *Exercise Immunology And Infectious Diseases In Athletes: A Clinically Relevant Review*. The Internet Journal of Internal Medicine. 2000 Volume 3 Number 1.

Abstract

The authors give an overview of current knowledge in exercise immunology focusing on clinically relevant information. This is followed by a discussion of specific infectious diseases of special interest in athletic populations emphasizing sports participation decision making and prevention issues.

INTRODUCTION

Exercise immunology is the study of existing relationships between physical activity and the immune system. The relationships most studied include the effects of physical activity on immune system components and function, the effects of physical activity on the course of diseases involving the immune system and the role of the immune system during and after bouts of physical activity. Well over six hundred papers have been published on topics in exercise immunology since 1900, the majority since 1990 ([1]). Given this challenge, our purpose for this review is twofold. First, we give an overview of current knowledge in exercise immunology focusing on clinically relevant information. Second, we discuss specific infectious diseases of special interest in athletic populations with an emphasis on sports participation decision making and prevention issues. In doing so, we hope to provide physicians with up to date, important and clinically relevant information that will aid them in caring for their active patients.

SECTION 1: EXERCISE IMMUNOLOGY

For years, many have held that moderate exercise improves immunity and therefore reduces the risk of infection. Conversely intense exercise and over training have been thought to decrease immunity and raise infection risk, especially when coupled with emotional stress. Some believe a time exists immediately following intense exercise, referred to as an “open window”, when infection risk is increased ([2]). During this period it is hypothesized that bacteria and/or viruses can gain a foothold leading to infection. This purported “open window” can last from 3 to 72 hours depending on the intensity and duration of exercise. However, it has yet to be demonstrated that the most fatigued athletes are the ones who consistently develop clinical illness at a higher rate ([3]).

Acute exercise causes a rapid interchange of various immune cells between lymphoid tissue and circulating blood. The more prolonged and high intensity the exercise is the more cell trafficking occurs. There is conflicting data on the effect of exercise on granulocytes. Some investigators report an increase in neutrophil concentration both during and after exercise. This possibly could be due to the effects of dehydration. To date no study has demonstrated an increase in neutrophil function. In fact, granulocyte oxidative burst activity is reduced following prolonged exercise ([4]). Neutrophils depend on this activity for their killing capacity.

Lymphocyte concentration increases during exercise due to recruitment of sub-populations but rapidly falls post exercise (2). In one study the average total number of lymphocytes decreased from 2,100 per microliter before a triathlon to 1,000 per microliter one hour after the triathlon ([5]). Perforin, a cytotoxic protein expressed by many lymphocytes, plays a prominent role in protection against viral and bacterial infections. Staats et al. found that athletes have a higher perforin expression rate in CD3 and CD8 cells (36.1%) than untrained controls (12.8%). They also found that endurance exercise leads not only to a decrease in overall lymphocyte numbers but further reduces the percentage of perforin expressing cells (5). This may help explain why endurance athletes seem to be more susceptible to viral upper respiratory tract infections at certain times.

In endurance athletes, natural killer cell activity appears to

be increased at baseline. Nieman et al. found natural killer cell activity was significantly elevated in rowers compared to non-athletes ([6]). Other similar studies involving moderate activity for eight to fifteen weeks showed no significant increase. Thus, endurance exercise may have to be intense and prolonged to obtain this effect. It remains unclear whether the increased natural killer cell activity described is due to an increased number of cells, enhanced activity of individual cells or both. Following intense prolonged exercise of an hour or longer, natural killer cell activity is decreased 40 to 60% for at least six hours (4). This may result from increased cortisol levels during exercise leading to a redistribution of cells from the blood compartment to tissues.

Exercise also seems to affect secretory antibody levels. Acute exhaustive exercise appears to cause a decrease in both nasal and salivary IgA concentrations. IgA, responsible for inhibiting attachment and replication of pathogens, is important for preventing entry of the pathogens into the body. IgA concentrations in nasal secretions have been shown to decrease by almost 70% for 18 hours in individuals following a thirty-one kilometer race (4). In studies of elite women rowers, pre-exercise salivary IgA concentrations were 77% higher in the rowers compared to non-athletes ([7]) and the proportion of subclass IgA1 was 80% in rowers compared to 60% in normal non-exercising adults ([8]). No difference was found between groups in the salivary concentration of IgG or IgM (7). Low concentrations of salivary IgA found early in one seven-month study period were associated with an increased risk of developing an upper respiratory tract infection. However, no correlation was seen between salivary IgA levels and symptoms of upper respiratory tract infection ([9]). Nor was an effect seen on salivary IgA concentrations following a normal two-hour training session (7).

Several effects of exercise on chemical mediators of immunity have been elucidated. Exercise increases cytokine levels in damaged skeletal muscle and increases expression of adhesion molecules ([10]). Prolonged exhaustive exercise can lower plasma levels of glutamine, an important fuel source for some immune cells. Glutamine also may have immunostimulatory effects. In one trial, oral glutamine compared with placebo appeared to have a beneficial effect on the incidence of upper respiratory tract infections reported by runners after a marathon ([11]).

Given the limited data available, one expert has made several statements regarding the effects of exercise on overall immune system function. Endurance athletes seem to be at increased risk for developing upper respiratory tract infections during periods of heavy training, over-training and in the first two weeks following a marathon or similar exhaustive race event. Moderate exercise training may decrease the incidence and symptoms of upper respiratory tract infections. And, the incidence of and mortality rates for certain types of cancer are lower among active subjects (1).

In sum, exercise has some known effects on immune system components and function but the reasons for and clinical significance of these effects remain largely unclear. Furthermore, varying levels of exercise training seem to have opposite effects on upper respiratory tract susceptibility without good evidence as to why. Clearly, large gaps in our understanding of exercise immunology exist and need to be addressed in future studies.

SECTION 2: INFECTIOUS DISEASES OF INTEREST IN ATHLETIC POPULATIONS

During participation in sports, athletes often come into close personal contact with other participants. This, coupled with the fact that several prominent athletes were found to be HIV positive in the past decade, has heightened interest in the lay and medical communities regarding infectious diseases in sport. Specifically, the potential risks to the affected individual and the risk of transmission to other participants during competition are of concern. Though any infectious disease occurring in athletes might be considered for this review, we have chosen several thought to be of particular interest. Thus, we will limit our discussion to infectious mononucleosis, otitis externa, pertinent infectious dermatological conditions, human immunodeficiency virus, and hepatitis B.

INFECTIOUS MONONUCLEOSIS

Infectious mononucleosis is an acute, self-limited disease of children and young adults. Usual age range of diagnosis is 15 to 24 years with an equal distribution among males and females. By age 30, 90% of the American population has had infectious mononucleosis ([12]). Epstein Barr virus is most commonly the cause, however, cytomegalovirus and Toxoplasma gondii also may lead to the syndrome. Mononucleosis is characterized by a prodromal phase consisting of headache, malaise, anorexia, and chills for three to five days followed by seven to twenty one days of fever, fatigue, pharyngitis, lymphadenopathy, and splenomegaly. The virus is not highly contagious with a 30

to 50 day incubation period (12). It is secreted in saliva and close contact is required for transmission. Diagnosis can be established on the basis of a characteristic peripheral blood smear demonstrating monocytosis and lymphocytosis with increased atypical forms. There is also a commercially available Monospot test that is positive in 90 - 95% of adolescents with infectious mononucleosis. This test is not as accurate in children and often has to be repeated in adolescents as it takes one to two weeks to develop heterophile antibodies. Treatment for this disease is entirely supportive. Rest, fluids, analgesics, and antipyretics are the mainstays. Occasionally systemic corticosteroids are used for severely enlarged tonsils. These have not been shown to be effective in reducing splenomegaly. Potential complications in the athlete are especially important. Splenomegaly occurs in 70% of those affected and hepatomegaly in 20% ([13]). Other potential complications include hepatitis, concurrent Group A Strep pharyngitis, airway obstruction from enlarged tonsils, pneumonitis, and rarely encephalitis, Guillain-Barre syndrome or Lemierre's syndrome. Splenomegaly predisposes individuals to splenic rupture and therefore limits athletic participation. Splenic rupture occurs in less than 0.2% of cases and may be traumatic or atraumatic (13). Most ruptures occur between days four and twenty-one of symptomatic illness. Contact sports and heavy exertion should be avoided for at least 4 weeks from symptom onset or longer until fever, complications and splenomegaly resolve. Transabdominal ultrasonography can be used to confirm splenomegaly resolution with a spleen size of 14 centimeters generally accepted as top normal. Since the spleen must be at least 2 to 3 times its normal size to be reliably palpated, an athlete should be restricted from contact sports for 2 to 3 weeks after his spleen is no longer palpable to ensure resolution if ultrasonography is not performed.

OTITIS EXTERNA

Otitis externa or "swimmer's ear" is an infection of the external auditory canal commonly seen in swimmers, divers, water polo players, water-skiers and surfers usually in summer months. The most common pathogens are Pseudomonas aeruginosa and Aspergillus. Individuals with allergic conditions such as asthma and eczema have a threefold higher risk for developing otitis externa (12). Cerumen repels water and aids in maintaining an acidic pH, thus helping prevent infection. This defense can be overcome and otitis externa typically develops in athletes who experience excessive water exposure or improperly clean their ear canals. Hyper-hydrated ear canals may become macerated, an ideal environment for microbial growth. Other predisposing factors include freshwater swimming, swimming in improperly chlorinated pools, or scratches in the canal. Typical signs, symptoms and findings include pain, exudate, edema, and erythema of the external auditory canal as well as increased discomfort with pulling on the auricle. Balance disturbances are less common but also may occur. Treatment consists of cleaning the canal with water or hydrogen peroxide followed by topical antibiotics. Fungal infections are treated with 1% tolnaftate solution (13). A wick may be needed if severe edema is present. Intense itching may be treated with topical corticosteroids. An athlete with otitis externa may return to the water within 2 to 3 days of beginning treatment as long as she has no balance difficulty, pain, redness, or drainage. Keeping the ear canals dry is the best way to prevent primary infection and recurrence. This can be accomplished during water exposure by wearing properly fitting silicone earplugs. After water exposure, athletes can use over-the-counter chemical drying agents and/or mechanically dry their external canals with a hair dryer on a low setting for several minutes.

IMPETIGO

Impetigo is a superficial bacterial infection of the skin caused by beta hemolytic streptococci or Staphylococcus aureus. It is most commonly seen in wrestlers, gymnasts and swimmers (13). The skin lesions may vary from small vesicles to large bullae that rupture and exude a honey colored serous fluid. This then forms a crust. A potential complication of impetigo is post streptococcal glomerulonephritis occurring in 2 to 5% of cases ([14]). Diagnosis is based on clinical exam and established by obtaining a bacterial skin culture from the base of a vesicle after the crust has been removed. Impetigo is highly contagious and transmitted by direct contact. Treatment consists of debriding the crusts with hydrogen peroxide then applying mupirocin ointment or using oral antibiotics such as cloxacillin, dicloxacillin, or cephalexin for a 5 to 10 day course, or azithromycin for 5 days. Athletic participation should not be allowed until all lesions are healed. Prevention is best achieved by cleaning areas of minor trauma with soap and water and applying triple antibiotic ointment. Also, athletes can shower with antibacterial soap and should not share towels or athletic equipment. Wrestling and gymnastic mats should be washed daily with an antiseptic solution.

CELLULITIS AND ERYSIPELAS

Cellulitis is a bacterial skin infection involving the dermis

and subcutaneous tissues. It appears as an expanding red, swollen, tender plaque with an indefinite border. Chills and fever may occur as the red plaque expands and becomes edematous. In adults, the most common etiologic agents are group A streptococcus and Staphylococcus aureus. Erysipelas is a superficial cellulitis with lymphatic involvement. It is characterized by red, painful streaks that move toward regional nodes. It is more common on the lower extremities than on the face. Group G streptococcus is a common pathogen. Diagnosis of these conditions is based on history and physical exam. Biopsies and aspirates are usually of low yield. Treatment consists of oral antibiotics such as dicloxacillin, erythromycin, or a cephalosporin. In severe and refractory cases, intravenous antibiotics such as nafcillin, cefazolin or, in penicillin allergic patients, clindamycin or vancomycin may be needed. If the problem is recurrent, antibiotic prophylaxis should be considered (14). Athletic participation is allowable if the patient is afebrile, treatment has begun, and the lesion is adequately covered.

FURUNCLES

A furuncle or boil is an infection of a hair follicle or sebaceous gland most commonly caused by Staphylococcus aureus. The lesion typically presents as a tender, warm abcess or nodule on an extremity or the trunk that becomes fluctuant over time, often with overlying erythema and induration. Diagnosis is based on the clinical presentation. Initially treatment consists of oral antibiotics for 7 to 10 days in combination with warm moist compresses. Once fluctuance develops incision and drainage is necessary (14). The wound should then be packed to facilitate further drainage. If the athlete is involved in water or contact sports, participation should not be allowed until the lesion is healed.

INFECTIOUS FOLLICULITIS

Infectious folliculitis is an inflammation of hair follicles due to bacterial infection, most commonly staphylococcal. One pustule or a group of pustules may appear without systemic complaints. Diagnosis is made based on the clinical symptoms and physical exam. Treatment usually consists of oral antibiotics though some cases will respond to wet compresses with Burrow's solution (aluminum sulfate and calcium acetate) (14). Once treatment is begun, the athlete may be allowed to participate. The lesions should be adequately covered.

ERYTHRASMA

Erythrasma is a bacterial skin infection caused by Corynebacterium minutissimum. It is most commonly seen in the axilla and groin folds. The lesion appears as a uniformly brown, scaly patch with desquamation. In the groin area, erythrasma is often mistaken for tinea cruris. Diagnosis of erythrasma is confirmed if the rash fluoresces coral-red under Wood's light. Most cases can be successfully treated with topical antibiotics however severe cases require oral erythromycin or tetracycline (13). Unlike tinea infections, erythrasma carries no restriction on athletic participation.

PEDICULOSIS

Pediculosis is a parasitic infestation of hair with lice. Species known to infest humans include Pediculus humanus capitis, Pediculus humanus corporis and Pthirus pubis. Transmission occurs easily by personal contact or contact with objects. Diagnosis is made by direct visualization of lice or their eggs. Treatment requires two steps. First nit removal is facilitated by rinsing hair with formic acid or vinegar followed by combing with a nit comb. Infested hair then is treated with lindane, permethrin, or pyrethrins (14). Athletic participation should not be allowed until the infestation is successfully treated and nits are removed.

SCABIES

Scabies is a contagious skin disease caused by the mite Sarcoptes scabiei var. hominis. Transmission occurs through direct skin contact with infested individuals and possibly infested dogs and cats. Diagnosis is made by history and physical exam and confirmed by identifying a mite in a burrow. Linear or curved burrows are 2 to 15mm long and typically found in the interdigital webs, wrists, genital area, buttocks, waist and sides of the hands and feet. A vesicle or a mite at the end of a burrow often can be seen. Isolated vesicles filled with serous fluid also may be visualized. The current scabicide of choice for treatment is Permethrin. Lindane, Ivermectin, sulfur, and crotamiton have been used and also may be effective (14). All intimate contacts and household members should be treated. Athletic participation should not be allowed until infestation is successfully treated.

HERPES GLADIATORUM

Herpes gladiatorum is a viral skin infection in wrestlers caused by herpes simplex virus (types 1 and 2) commonly affecting the face and arms. Lesions present as a group of 1 to 2 mm vesicles on an erythematous base. These last for 2 to 3 days, then the vesicles unroof and a crust forms. Crusted lesions last 5 to 7 days. Approximately 25% of primarily

infected individuals will develop systemic symptoms such as fever, myalgia, lethargy, headache, and sore throat (13). The herpes virus is highly contagious and transmission occurs by direct contact or fomites. Diagnosis can be confirmed through microscopic examination of scrapings or biopsy specimens prepared using Tzanck stain and identifying multinucleated giant cells (located at the base of vesicles on biopsy).

Treatment consists of oral antivirals. Acyclovir, famciclovir, and valacyclovir are all effective. These should be initiated within 72 hours of primary infection symptoms or at the onset of prodromal symptoms in recurrent outbreaks in order to reduce symptoms and duration. If recurrence is common, prophylactic antiviral therapy should be considered. Athletic participation in close contact or combative sports such as wresting should be prohibited until crusting and eschar resolve.

HERPES ZOSTER

Herpes zoster or shingles is a cutaneous viral infection usually involving the skin of a single dermatome. It is the result of reactivation of latent varicella virus from a dorsal root ganglion. Age, immunosuppression, emotional stress, and fatigue have been implicated in varicella reactivation. It is characterized by several days of pain, burning, or itching followed by an eruption of vesicles. Transmission of the virus may occur by direct contact with an erupted vesicle and this may cause chicken pox in those not previously infected. Diagnosis is made based on the characteristic appearance and dermatomal distribution of the painful rash and can be confirmed by Tzanck smear, biopsy, or culture of a vesicle. Oral acyclovir, valacyclovir, or famciclovir is recommended treatment in the acute phase. This should be initiated as early as possible to decrease symptom severity, duration and risk of post herpetic neuralgia. Burrow's solution or cool tap water may be used as a topical compress. Pain may be greatly reduced with oral steroids though this has not been shown to shorten length of the illness (14). Oral gabapentin also may be effective in reducing the neuropathic pain of shingles. Athletes should be withheld from close contact and combative competition until crusting and eschar have healed. Zoster may be preventable in those who have not experienced primary varicella infection by administering the now universally recommended varicella vaccine.

MOLLUSCUM CONTAGIOSUM

Molluscum contagiosum is a viral infection of the skin caused by a largepox virus ($_{15}$). It occurs most commonly in the genital area, on the hands, forearms, and face often developing along a scratch. Transmission occurs by close personal contact, autoinnoculation, or scratching. Lesions may appear singly or be grouped. They are umbilicated, flesh colored, dome-shaped lesions typically 2 to 4 mm in diameter. Treatment consists of excision and curettage followed by cryotherapy with liquid nitrogen or electrocautery. Retin - A, salicylic acid, and laser therapy also have been used successfully (14). Often lesions are self-limited usually lasting 6-9 months. Athletes should refrain from practice and competition until the lesion is cleared, unless it can be adequately covered.

COMMON AND PLANTAR WARTS

Warts or verrucae are epithelial tumors caused by several types of human papilloma virus. Common warts present as cauliflower-shaped, raised lesions typically on the hands. They are often only of cosmetic significance. Plantar warts occur as flat lesions with pinpoint bleeding on weight bearing surfaces. Calluses seem to be more susceptible to human papilloma virus than normal skin making most athletes vulnerable. Transmission occurs by direct skin to skin contact or contact with objects such as pool decks, showers, or weight lifting apparatuses. Effective treatments range from topical 17% salicylic acid or 0.7% cantharidin, cryotherapy with liquid nitrogen and curettage, to electrodessication in more severe cases. Plantar warts may be debilitating in athletes due to the pain. Plain lidocaine 1% may be injected locally into lesions weekly for 1 to 3 weeks to help control pain if necessary (14). Athletic participation is allowed as long as lesions are adequately covered.

TINEA CORPORIS

Tinea corporis or ringworm is a superficial fungal infection of the skin caused by Trichophyton rubrum, Trichophyton mentagrophytes or Epidermophyton floccosum. Lesions present as expanding erythematous raised annular lesions with central clearing. Diagnosis may be confirmed by examining skin scrapings in a potassium hydroxide preparation under the microscope. Transmission occurs by direct contact. Treatment usually consists of topical antifungals though oral antifungals may be required for severe or resistant cases. Athletic participation is allowed as long as treatment has begun and lesions are adequately covered.

TINEA CAPITIS

Tinea capitis is a fungal infection of the scalp. Most forms begin with one or more round patches of scale or alopecia.

The most common causative agents in the United States are Trichophyton tonsurans and Microsporum canis. Transmission occurs by direct contact. Treatment consists of oral griseofulvin, terbinafine, or itraconazole. Selenium sulfide shampoo may also be used as an adjunct (14). Athletes are allowed to participate once treatment has begun provided the area can be adequately covered.

TINEA GLADIATORUM

Tinea gladiatorum is a fungal skin infection unique to wrestlers. The appearance can be similar to tinea corporis however the etiologic agent is Trichophyton tonsurans ([16]). Transmission occurs by direct skin to skin contact. Initial diagnosis is made clinically. Scrapings and culture may be indicated to confirm the diagnosis, especially in the face of an outbreak among athletes (16). A 1997-98 pilot study of high school wrestlers compared topical versus oral treatment. Clotrimazole 1% cream applied twice daily was compared to oral fluconazole 200mg once weekly for three weeks. Both produced similar improvement in clinical parameters but no statistical difference in culture eradication ([17]). A second study of wrestlers comparing oral fluconazole versus placebo as prophylaxis was performed in 1998-99. Those given fluconazole 100mg once weekly had significantly fewer infections (6%) than the placebo group (22%). The question remains when and in whom is chemoprophylaxis warranted ([18]). Athletic participation may be allowed if treatment is initiated and the affected area can be sufficiently covered.

TINEA VERSICOLOR

Tinea versicolor is a superficial fungal skin infection caused by Malassezia furfur or Pityrosporum orbiculare. Lesions present as asymptomatic patchy, small, hypopigmented macules, most commonly on the upper chest, back, neck, and arms. Diagnosis is made clinically and can be confirmed by scrapings or biopsy. Treatment is accomplished by applying 2.5% selenium sulfide shampoo 15 minutes daily for 3 days then weekly for prevention (14). Alternatively, this medication may be applied directly to the lesions at night and washed off the next morning. Athletic participation is not restricted.

HUMAN IMMUNODEFICIENCY VIRUS

Human immunodeficiency virus (HIV), a human retrovirus, has been known since 1983 to cause acquired immune deficiency syndrome (AIDS). In the United States, there are over 1.5 million cases of asymptomatic HIV infection ([19]). Approximately 14% of all new HIV infections occur in persons between the ages of 12 and 24 years ([20]). The virus inserts itself into the cellular genome and causes persistent infection by coding for the enzyme reverse transcriptase, primarily effecting CD4 cells (T lymphocytes) by binding to their receptors (19). CD4 cell counts can be measured to stage HIV infection. With progression of disease, CD4 counts decrease and risk for opportunistic infection and malignancy increases. Human immunodeficiency virus may be found in all body fluids, but transmission has been documented only through blood, semen, vaginal secretions and perinatally. There has been no confirmed case of transmission between athletes during sports activities. Physicians in Italy did report a case of HIV seroconversion in a soccer player after a head to head collision with an HIV positive player. Later, however, officials could not rule out this player seroconverted due to a previous HIV exposure while employed at a drug rehabilitation center ([21]). There are two documented cases of HIV transmission verified by the Centers for Disease Control between individuals involved in fistfights. These were street brawls and athletic competition was not implicated (21). Using the frequency of bleeding in a National Football League game, the prevalence of HIV in college-aged men, and the rate of HIV transmission in exposed health care workers, some have estimated the overall risk of HIV transmission in an NFL game to be 1 per 85 million game contacts ([22]). Diagnosis is made by positive enzyme-linked immunosorbent assay (ELISA) confirmed by Western blot, immunofluorescence assay, serum antigen, culture or nucleic acid probe of peripheral blood lymphocytes. In a 1992 survey of National Collegiate Athletic Association (NCAA) member institutions, 12 of 548 schools reported having HIV positive athletes. Only twenty-two institutions performed routine HIV testing and two mandated testing of athletes (22). Currently mandatory testing of athletes is not recommended by the NCAA, the American Medical Society for Sports Medicine (AMSSM), the American Orthopedic Society for Sports Medicine (AOSSM), or the American Academy of Pediatrics (AAP) ([20,23,24]). It is recommended that athletes desiring voluntary testing should be assisted in doing so or referred appropriately. Treatment of HIV positive individuals is guided by the presence of clinical or laboratory evidence of immune dysfunction. Anti-retroviral therapy is indicated in all symptomatic individuals with AIDS and asymptomatic individuals with AIDS whose CD4 counts are below 200 per cubic mm. In symptom free HIV positive individuals anti-retroviral therapy is recommended when CD4 counts are between 200 and 350 per cubic mm regardless of viral load.

Individuals who are HIV positive and asymptomatic with CD4 counts above 350 per cubic mm should be treated if their DNA viral loads are over 30,000 or their RNA viral loads are over 55,000 ([25]). There are multiple anti-retroviral medications currently available with frequent new developments and changes in combination therapy recommendations. In those with CD4 counts below 200 per cubic mm, prophylaxis for Pneumocystis carinii pneumonia with trimethoprim-sulfamethoxazole three times per week is recommended (23). Athletic participation should not be denied on the sole basis of positive HIV status. The NCAA, World Health Organization, AMSSM, AOSSM, AAP, and the International Olympic Committee all share this stance ([20,23,24,26]). Athletic participation decisions should be based on the health of the individual. If the athlete is asymptomatic with no laboratory evidence of immune dysfunction, no limitations should be placed on his or her ability to play. There is no evidence that moderate intensity exercise and training are harmful to the health of an HIV infected individual and in fact, may be beneficial ([20,22,24,27]). Since the risk of HIV transmission is extremely low during athletic endeavor many authors agree education regarding "off-field" exposure risks is the key to preventing HIV infection in athletes. Counseling focused on safe sexual practices and blood exposures should be emphasized. Intramuscular anabolic steroid use is another risky behavior of some athletes. Approximately 38% of steroid users use an injectable form and sharing of contaminated needles in particular is a major concern (22). Recommendations for preventing the transmission of blood borne pathogens include the following. Skin wounds of all athletes should be properly cared for and covered before the beginning of a game or practice. Universal precautions should be followed by all caregivers of athletes. Latex gloves should be worn when handling contaminated products and changed between treatments of individual athletes. When an athlete is bleeding, he should be removed from practice or competition. The bleeding must be stopped and covered with a secure dressing that can withstand the demands of the game prior to return. If a uniform is saturated with blood, it must be evaluated by appropriate medical personnel for potential infectivity and changed if indicated. Athletes should be aware of their responsibility to report any bleeding wounds during practice or a game. Surfaces that are exposed to blood should be wiped down with appropriate hospital disinfectants or a solution of one part household bleach with ten parts tap water. Proper disposal procedures should be followed with any sharp object. After each practice or game, equipment or uniforms soiled with blood should be handled in accordance with hygienic methods and washed with laundry detergent for 25 minutes at 160 degrees Fahrenheit ([20,23,28]).

HEPATITIS B

Hepatitis B (HBV) is a blood-borne DNA virus that infects the liver. Approximately 300,000 cases of acute hepatitis B occur in the United States each year (19). Many of those infected will have minimal or no symptoms. Others will exhibit common symptoms of acute infection including malaise, nausea, anorexia, diarrhea, and low-grade fever. These patients often are found to have jaundice, dark urine and tender hepatomegaly. Ninety percent recover completely, but 5 to 10% go on to develop chronic hepatitis (19). Approximately one million chronic carriers are living in the United States (20). The incubation period for HBV ranges from 4 weeks to 6 months. Transmission is similar to that of HIV. HBV is present in virtually all body fluids. Transmission usually occurs as a result of direct inoculation with body fluids. Compared to HIV, HBV is more easily transmitted especially if the inoculating fluid is positive for HBV e-antigen. In infected individuals, HBV is present in higher blood concentrations than HIV and is more stable in the environment. Unlike HIV, there are two documented cases of HBV transmission during athletic competition. One case involved a hepatitis B outbreak on a sumo wrestling team in Japan. The source was traced to one member of the team who was positive for the HBV e-antigen (23,28). The second case was an outbreak among Swedish orienteerers. Epidemiologists concluded the source of infection was contaminated water used to clean participants' wounds caused by branches and thorns (23). Diagnosis is made based on the above symptoms and findings in combination with laboratory tests. Hepatitis B surface antigen is positive in both acute and chronic disease. IgM antibody to hepatitis B core antigen is also positive in acute infection. Positive hepatitis B e-antigen is a marker of increased infectivity. Serum liver transaminases (ALT and AST) and bilirubin also are elevated. Treatment for hepatitis B is supportive. Hospitalization is only necessary for severe cases. Prevention is again important. The AAP now recommends HBV immunization for all children. The AAP also recommends aggressively promoting HBV immunization among athletes, coaches, athletic trainers, equipment handlers, laundry personnel, and any other persons at risk of exposure. It is estimated that over 95% of those receiving HBV vaccine will be protected from the disease (23). Since most children are receiving vaccination at an early age, HBV

transmission on the playing field hopefully will be less of an issue in coming years. Athletic participation is based on clinical signs and symptoms. An athlete experiencing fever or malaise should not participate. The NCAA recommends considering removal of an athlete with acute infection from combative, close contact sports until loss of infectivity is known (20). Infectivity, probably best gauged by surface antigen remaining positive, may persist for up to 20 weeks in the acute stage. The NCAA also states chronic carriers, especially e-antigen positive individuals, should probably be withheld from combative, close contact sports indefinitely (20).

In conclusion, infectious diseases are common in athletes and specific infectious diseases occur more frequently in certain subsets of athletes. As discussed, optimal treatment of affected athletic individuals often requires special consideration. Physicians providing medical care to athletes, therefore, should be aware of potential complications, participation and prevention issues surrounding infectious diseases in sport so they can provide appropriate, clear and consistent athletic participation recommendations as well as educate others regarding proper preventative measures.

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