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Upper respiratory tract infections (URTIs) represent the most common acute illnesses in the general population and account for the leading acute diagnoses in the outpatient setting [1]. Similarly, athletes are infected with these illnesses and require appropriate treatment, allowing them to participate safely and at their full potential. Groups of athletes are often at an elevated risk of transmission because they are confined to close quarters with teammates in the locker room, at practice, and during travel. Further evidence demonstrates a higher susceptibility to URTIs in athletes during and after high training loads, likely from a suppressed immune system [1]. Viruses account for most URTIs, but the sports medicine physician must be able to recognize bacterial infections and the potential complications that require specific therapy. Given the athlete’s expectation to return to activity as soon as possible, the sports medicine physician should be able to accurately diagnose and aggressively treat these illnesses.

Management of URTIs in athletes spans a wide range of pathogens, clinical presentations, and treatment options. Participation and return-to-sport decisions are determined by the nature of the infection, the risk of transmission, and the demand placed on the athlete during practice and competition. Decisions should be made on a case-by-case basis using the best evidence-based medical care. Above all, any recommendations made should address the safety of the athlete and all participants involved. In this article, the authors discuss the common pathogens, diagnosis, treatment options, and return-to-play decisions for URTIs, with a focus on the common cold, sinusitis, pharyngitis, and infectious mononucleosis in the athlete.

**VIRAL UPPER RESPIRATORY TRACT INFECTION (THE COMMON COLD)**

The common cold is the most frequent acute illness in the United States and the leading cause of missed days from school or work [1]. A viral
URTI is a benign self-limiting syndrome typically lasting 5 to 14 days, manifested by rhinorrhea, cough, and fever and caused by multiple families of viruses. The pathogens most frequently associated with common cold symptoms are rhinoviruses (10%–40% of cases), coronaviruses (20%), and respiratory syncytial virus (10%) [2]. Influenza viruses, parainfluenza viruses, and adenoviruses also cause common cold symptoms, but to a lesser degree [2].

Direct contact, small-particle aerosols, and large-particle aerosols can spread common cold viruses. Person-to-person contact depends on the amount of time people spend together and the amount of virus shed by the infected donor [3]. In relatively closed communities, secondary attack rates can range from 25% to 70% [4]. Hand-to-hand contact is an important factor in the transmission of disease. The most efficient means of viral transmission is the spread of infectious mucoid secretions to the fingers and hands and subsequently to the nose or eyes of a susceptible person [5]. Some viruses can be viable on the human skin for at least 2 hours, and one study found that certain viruses could be recovered from 40% to 90% of hands of persons who have colds [5]. This ease of transmission demonstrates the need to encourage proper hand washing in the athletic population as an important preventive measure to reduce the number of illnesses and time away from sport.

The common cold can have more serious complications. Acute bacterial sinusitis develops in about 2.5% of adult patients after a viral URTI [6]. Infections can also be complicated by lower respiratory tract disease, such as pneumonia, and have been linked to up to 40% of acute asthma attacks in adults [7]. It is important for the sports medicine physician to readily identify the common symptoms of, properly diagnose, and effectively manage viral URTIs to reduce the more serious complications from the illness.

**Diagnosis**

Viral URTIs can be difficult to distinguish from less common bacterial cases solely based on clinical examination. Findings on physical examination are few in light of the subjective discomfort of the patient, and the symptoms may vary from patient to patient with the same illness. The incubation period for most common cold viruses is 24 to 72 hours [8]. Colds usually persist for 3 to 7 days in the normal host; however, 25% of colds may last as long as 2 weeks [2]. Risk factors that increase severity of disease include young age, low birth weight, prematurity, chronic disease, immunodeficiency disorders, malnutrition, and crowding [8].

Antiviral therapy is not available for most viruses that cause viral URTIs. Therefore, despite being the standard of confirmation, viral cultures are rarely indicated for uncomplicated URTIs in the outpatient setting [2]. Patients who have viral URTIs can have an increased white blood cell count associated with a left shift. Some viral infections can precipitate atypical lymphocytes, lymphocytosis, or lymphopenia; however, a complete blood count is not helpful in distinguishing disease or in directing therapy in uncomplicated URTI in the outpatient setting [2].
**Treatment and Return to Play**

There is no evidence that antibiotics have a clinically important effect on colds uncomplicated by secondary infection. Symptomatic therapy remains the foundation of common cold treatment. A number of agents have been studied and have demonstrated varying effects on the course of illness (Table 1) [9–14].

| Treatment       | Benefit               | Data                                                                 | Level of evidence [reference] |
|-----------------|-----------------------|----------------------------------------------------------------------|------------------------------|
| Antibiotics     | Not likely beneficial | Compared with placebo, a single dose of an oral or topical decongestant produced a significant 13% reduction in subjective symptoms | A<sup>a</sup> [9]            |
| Decongestants   | May be beneficial     | There was no benefit from repeated use over several days            | A<sup>a</sup> [27]           |
| Antihistamine   | May be beneficial     | Reduced the symptoms of runny nose and sneezing for the first 2 d of colds | A<sup>a</sup> [10]           |
| Vitamin C       | Unknown effectiveness | 1 g daily or more produces about 15% fewer symptomatic days per episode | B<sup>b</sup> [11]           |
| Zinc            | Unknown effectiveness | May reduce duration of cold symptoms at 7 d compared with placebo   | B<sup>b</sup> [12]           |
| Echinacea       | Unknown effectiveness | Some preparations of Echinacea may be better than placebo for cold treatment | B<sup>b</sup> [13]           |
| Steam           | Unknown effectiveness | Conflicting evidence of the efficacy of steam inhalation at 40°–47°C in the reduction of cold symptoms | B<sup>b</sup> [14]           |

<sup>a</sup>Level A is consistent, good-quality patient-oriented evidence (SORT evidence rating system).

<sup>b</sup>Level B is inconsistent or limited-quality patient-oriented evidence (SORT evidence rating system).
According to the American College of Sports Medicine, when an athlete has common cold symptoms without fever or general body aches and pains, intensive exercise training may be safely resumed a few days after the resolution of symptoms; mild-to-moderate exercise does not appear to be harmful for individuals who have common cold symptoms [15]. In settings with appropriate supervision, athletes who have viral URTIs and no fevers, myalgias, or symptoms below the neck are safe to continue their previous level of activity with no restrictions. Return-to-play decisions should be made on a case-by-case basis and should focus on minimizing the risk of further harm.

**ACUTE SINUSITIS**

Sinusitis is one of the most common illnesses diagnosed in the United States, affecting about 16% of the adult population annually [16]. Sinusitis is defined as inflammation of one or more of the paranasal sinuses and is categorized as acute (<4 weeks), subacute (4–8 weeks), and chronic (>8 weeks) [17]. The cause of sinusitis can be viral or bacterial (Table 2). Viral infection is the most common cause of acute sinusitis, and usually resolves in 7 to 10 days. Acute bacterial sinusitis is also usually a self-limiting disease, with 75% of cases resolving without treatment after 1 month [17]. When left untreated, however, bacterial sinusitis may not spontaneously resolve and can have severe complications including intracranial and orbital infections.

**Diagnosis**

The diagnosis of sinusitis is based on a combination of clinical history and physical examination findings. Imaging studies and laboratory tests can assist in the diagnosis of chronic or complicated cases. Symptoms of acute sinusitis include nasal congestion, purulent nasal discharge, maxillary tooth discomfort, headaches, fever, and facial pain or pressure that is worse when leaning forward [18]. It is unfortunate that the history is not sensitive or specific for

| Table 2 | Pathogens of acute sinusitis |
|---------|-----------------------------|
| **Viral** | **Bacterial community-acquired**<sup>a</sup> |
| Rhinovirus | *Streptococcus pneumoniae* |
| Parainfluenza virus | *Haemophilus influenzae* |
| Influenza virus | *Moraxella catarrhalis* |
| Corona virus | Other streptococcal species |
| Respiratory syncytial virus | *Staphylococcus aureus* |
| Adenovirus | Anaerobic bacteria |

<sup>a</sup>The most common organisms are *Streptococcus pneumoniae* and *Haemophilus influenzae*. These pathogens are responsible for 35% of cases in adults. In children, *S pneumoniae* and *H influenzae* are responsible for 41% and 29% of cases, respectively. *Moraxella catarrhalis* accounts for 26% of cases in children and 2% in adults.

*From Evans A, Niederman J. Epstein-Barr virus. In: Evans A, editor. Viral infections of human epidemiology and control. New York: Plenum Publishing; 1989. p. 265; with permission.*
distinguishing between viral and bacterial infections [19]. Although the symptoms of sinusitis are nonspecific, a history of persistent purulent rhinorrhea and facial pain appear to have some correlation with increased likelihood of bacterial disease [20].

Physical examination including palpation of the sinuses, transillumination, and visualization of the nares does not assist in differentiating bacterial from viral sinusitis. Frequently, sinusitis presents with facial tenderness over the affected sinus cavity. Transillumination can be reported as opaque (no transmission), dull (reduced transmission) or normal, but the sensitivity and specificity of this technique is poor [21]. One prospective study found that abnormal transillumination combined with purulent nasal discharge and history of maxillary pain, poor response to decongestants, and colored rhinorrhea was the best predictor of acute bacterial sinusitis [19].

Sinus aspirate culture is the “gold standard” for making microbial diagnosis, but it is not done routinely in clinical practice. Sinus aspiration should be considered when there is suspicion of intracranial extension of the infection or other serious complication.

Imaging studies are not usually indicated for noncomplicated cases of bacterial sinusitis but can provide confirmatory evidence when clinical disease persists despite optimal medical therapy. CT scanning is usually the procedure of choice and provides better sensitivity than plain radiographs (88% versus 59%) [22]. It is unfortunate that neither test can distinguish bacterial from viral infection, and CT scan is limited by the fact it can be frequently abnormal in patients who have the common cold. In one study, 27 of 31 adults who had a viral cold had abnormal CT of the sinuses, including occlusion and abnormalities in the sinus cavities [23]. In addition, MRI can be used to detect intracranial spread of infection, distinguish inflammatory disease and malignant tumor, and evaluate for fungal disease; however, MRI is not as good as CT scan in diagnosing acute sinusitis [23].

Treatment and Return to Play
Symptomatic treatment is the mainstay for the treatment of viral sinusitis, and antibiotics are generally not beneficial. Conversely, antibiotics are beneficial in the medical treatment for acute bacterial sinusitis. In patients who have an acute sinus infection, antibiotics are recommended when symptoms have not improved after 10 days, for severe illness, or when symptoms have worsened over 5 to 7 days [24]. The appropriate choice of antibiotic should be based on the most likely bacterial pathogen and clinical history. Current literature supports amoxicillin as the initial antibiotic choice in children and adults who have uncomplicated bacterial sinusitis [25,26]. A 10- to 14-day course of antibiotics is typically successful for the treatment of acute bacterial sinusitis [25].

Symptomatic treatments including antihistamines, decongestants, and nasal steroids may be beneficial in the treatment of viral and bacterial sinusitis. Despite being used to treat symptoms, antihistamines have not proved to be
beneficial and were not recommended in recent guidelines for the diagnosis and management of sinusitis in children [25]. Decongestants decrease nasal resistance and may be beneficial in the management of acute sinusitis; however, there has been relatively little systematic study of decongestants in patients who have sinusitis [27]. Intranasal steroids are also commonly used for the treatment of sinusitis, but until there is more evidence on the use of intranasal steroids in acute bacterial sinusitis, their use is not recommended [26].

Because acute viral sinusitis is a self-limiting illness and symptoms usually resolve in 7 to 10 days, exercise may be permitted with appropriate symptomatic care, particularly if the symptoms remain from the neck up (nasal congestion, facial pain, headaches, and so forth). When the athlete presents with more severe symptoms, however, including fevers or myalgias, vigorous exercise should be avoided to prevent dehydration and worsening of symptoms. Further, if the athlete’s symptoms worsen after 5 to 7 days or persist longer than 10 days, appropriate antibiotic treatment should be initiated and participation allowed as tolerated.

**ACUTE PHARYNGITIS (SORE THROAT)**

Acute pharyngitis accounts for 19 million clinic visits annually and relates to about 2% of all ambulatory visits in the United States [28]. Acute pharyngitis can be caused by viral and bacterial pathogens. Viral causes account for approximately 50% of acute pharyngitis infections and can cause pharyngitis indistinguishable from bacterial pharyngitis [29]. These viral agents include influenza virus, parainfluenza virus, coronavirus, rhinovirus, adenovirus, enterovirus, herpes simplex virus, Epstein-Barr virus (EBV), and HIV. Because of the potential complications, the major treatable pathogen is group A streptococcus (GAS), but this accounts for only about 10% of adult cases [29]. Other bacterial pathogens include group C and group G streptococcus, mixed anaerobes, Neisseria gonorrhoea, Corynebacterium diphtheriae, and several chlamydial species. Even though the differential diagnosis of acute pharyngitis in adults includes several viral and bacterial pathogens, the risk of rheumatic fever, acute glomerulonephritis, and supportive complications can be minimized by efficient and accurate diagnosis and treatment of GAS pharyngitis.

**Diagnosis**

For appropriate treatment, it is important to differentiate viral causes from bacterial causes, especially GAS. Viral pharyngitis has some associated clinical findings including pharyngeal swelling, erythema, and exudates. Furthermore, the presence of cough is more suggestive of a viral etiology. Primary herpes simplex virus infection may also be associated with palatal vesicles or shallow ulcers. Bacterial pharyngitis can be difficult to distinguish from viral pharyngitis clinically; however, a constellation of symptoms has been used to suggest GAS infection, including erythema, swelling, exudates of the tonsils or pharynx, fever (38.3°C/100.9°F or higher), tender anterior cervical
lymph nodes, and absence of conjunctivitis, cough, or rhinorrhea [30]. A scarlet rash may be seen with GAS infections, particularly in patients younger than 18 years [31]. It appears as tiny papules over the chest and abdomen, often described as sandpaper-like. The rash spreads and becomes more erythematous in the groin and armpits, usually resolving within 2 to 5 days.

Laboratory evaluation can assist the clinician in identifying GAS infection. Throat cultures remain the gold standard for diagnosing GAS pharyngitis and may isolate other pathogens. With proper technique, the sensitivity for throat cultures approaches 90% and the specificity ranges from 95% to 99% [32]. False positive results can be linked to a 1% to 5% carrier rate for the organism [33]. Cultures take 24 to 48 hours to grow and cannot immediately be used for clinical decisions on whether to start antibiotics. Therefore, the rapid streptococcal antigen test (RSAT) has emerged as the first test of choice in the management of acute pharyngitis. Studies show a sensitivity of 80% to 90% and a specificity of 90% to 100% [28]. Although less sensitive than throat cultures, the RSAT provides results in minutes and can assist in same-day management.

**Treatment and Return to Play**

Symptomatic care alone is appropriate for most acute pharyngitis cases. The major exception is GAS pharyngitis, which requires appropriate antibiotic treatment to prevent potential complications, to minimize secondary spread, and to shorten the course of the illness (Table 3) [34,35]. In athletes who are sexually active, the clinician may need to consider gonococcal infections as a cause of acute pharyngitis. Gonococcal infections are easily treatable with proper antibiotics.

It has been found that nonsteroidal anti-inflammatory drugs (NSAIDs) reduced sore throat symptoms at 24 hours or less and at 2 to 5 days compared with placebo [36]. Caution must be advised when prescribing NSAIDs, however, because they are associated with gastrointestinal and renal adverse effects. Studies in children and adolescents who had moderate to severe sore throat but without group A beta hemolytic GAS infection have shown that oral dexamethasone reduced time to initial pain relief and duration of throat pain compared with placebo. Adding corticosteroids to antibiotics, however, did not significantly reduce pain duration in children and adolescents who had group A beta hemolytic GAS infection [37].

In athletes who have suspected or confirmed acute bacterial pharyngitis, it is important to remember that they are considered contagious until they have been on antibiotic therapy for 24 hours, and it is recommended that they not participate until this time has passed [38]. Treated appropriately, individuals should see improvement in acute symptoms within 24 hours and may progress to activity as tolerated. Under close supervision, removal from competition is often unnecessary if the athlete has no fevers or systemic symptoms (Table 4) [39–43].
| Antibiotic             | Treatment | Adult dosage | Pediatric dosage | Data                                                                 | Level of evidence |
|------------------------|-----------|--------------|------------------|----------------------------------------------------------------------|-------------------|
| **First-line antibiotics** |           |              |                  |                                                                      |                   |
| Amoxicillin            | 10 d      | 500 mg bid   | 45 mg/kg bid     | Increased recovery rates compared with placebo at 2 wk 7–10 d of amoxicillin significantly increased complete symptom resolution compared with placebo | A<sup>a</sup> [34,35] |
|                        |           |              |                  | If no response after 72 h, re-evaluate and consider alternative antibiotics |                   |
| Doxycycline            | 100 mg bid| 2.2 mg/kg bid|                  | Increased recovery rates compared with placebo at 2 wk               | A<sup>a</sup> [34]  |
| Trimethoprim-sulfamethoxazole | 160/800 mg bid | 40/200 mg/kg bid |                  |                                                                      |                   |
| **Alternative antibiotics** |           |              |                  |                                                                      |                   |
| Amoxicillin/clavulanate | 500–875 mg bid | 22.5–45 mg/kg bid |                  |                                                                      |                   |
| Cefpodoxime            | 200–400 mg bid | 5 mg/kg bid   |                  |                                                                      |                   |
| Cefuroxime             | 250–500 mg bid | 7.5 mg/kg bid |                  |                                                                      |                   |
| Cefixime               | 400 mg qd  | 8 mg/kg qd   |                  |                                                                      |                   |
| Azithromycin           | 250 mg qd  | 5 mg/kg qd   |                  |                                                                      |                   |
| Clarithromycin         | 500 mg bid | 7.5 mg/kg bid|                  |                                                                      |                   |
| Levofoxacin            | 500 mg bid |              |                  |                                                                      |                   |

<sup>a</sup>Level A is consistent, good-quality patient-oriented evidence (SORT evidence rating system).
INFECTIOUS MONONUCLEOSIS

Infectious mononucleosis (mono) is a common illness in young adults, including the athletic population. Mono occurs in 3% of the college population and is characterized by the triad of fever, tonsillar pharyngitis, and lymphadenopathy [44]. It has long been accepted that EBV is the infectious pathogen of mono. EBV is a widely disseminated herpesvirus that is spread by intimate contact between susceptible hosts and asymptomatic EBV shedders. Often called the “kissing disease,” EBV primarily spreads by way of the passage of saliva. The virus can persist in the oropharynx of patients who have a history of mono for up to 18 months after clinical recovery. Often, the illness can go undiagnosed if the symptoms are mild, but adolescents and young adults develop symptoms with higher frequency, ranging from 50% to 70% [45].

Although mono is generally a benign illness, splenic rupture is a known serious complication of mono, with an estimated occurrence of 1 to 2 cases per 1000 [46]. Almost all of the reported cases have occurred in male patients [45]. On occasion, splenic rupture is the first presenting symptom of mono, and it is spontaneous in over half of reported cases, with no history of impact or inciting injury [12]. Vital to the return-to-play decision, splenic rupture occurs primarily between the fourth and 21st day of symptomatic illness [45]. Despite being potentially life threatening, fatality from splenic rupture is rare.

Diagnosis

Classic mono presents with moderate to high fever, pharyngitis, and lymphadenopathy. One study of over 500 patients demonstrated that lymphadenopathy occurred in 100%, fever occurred in 98%, and pharyngitis occurred in 85% of documented cases [44]. Posterior cervical lymph nodes are characteristically more involved than anterior chains in mono, and these nodes may be large and moderately tender [44]. Lymphadenopathy peaks in the first week and gradually resolves over 2 to 3 weeks. The pharyngitis of mono is commonly described as exudative that may appear white, gray-green, or display necrotic features. Other findings include severe fatigue and splenomegaly. In a study of 631 Division I collegiate athletes, mean splenic size was 10.65 cm in length and 5.16 cm wide. Seven percent of the athletes’ baseline spleen size met the current criteria for splenomegaly [47]. In this population, a single ultrasound evaluation of spleen size is of limited value, and clinical judgment may be more useful. Splenomegaly associated with mono occurs in 50% to 60% of patients and usually recedes by the third week of illness [48].

Laboratory information can be important in the confirmation of the diagnosis of mono. A peripheral blood smear commonly shows a white blood cell count of 12,000/mm$^3$ to 18,000/mm$^3$ and a differential showing 60% to 70% lymphocytes, with more than 10% of these being atypical [49]. In contrast to the finding of atypical lymphocytes, heterophile antibodies are sensitive and specific for mono (85% and 100%, respectively) [49]. Heterophile antibodies
| Drug/dosage | Advantages | Disadvantages | Data/Level of evidence [reference] |
|------------|------------|---------------|----------------------------------|
| **Penicillin V potassium**<br>\(<23 \text{ kg}: 250 \text{ mg bid or tid} × 10 \text{ d}\)<br>\(>23 \text{ kg}: 500 \text{ mg bid or tid} × 10 \text{ d}\) or 250 mg bid or tid | Inexpensive<br>Narrow spectrum of antibacterial activity<br>Low side effect profile<br>Twice-daily dosing | Pain at injection site<br>Possible allergic reaction<br>Cannot discontinue drug exposure if allergy develops | First drug of choice; reduces streptococcal complications compared with placebo/A\(^a\) [39] |
| **Penicillin G benzathine**<br>\(<27 \text{ kg}: 600,000 \text{ U intramuscularly} × 1 \text{ dose}\)<br>\(>27 \text{ kg}: 1.2 \text{ million U intramuscularly} × 1 \text{ dose}\) | Ensures compliance | Pain at injection site<br>Possible allergic reaction<br>Cannot discontinue drug exposure if allergy develops | |
| **Erythromycin**<br>Estolate<br>\(20–30 \text{ mg/kg bid–qid} × 10 \text{ d}\)<br>Etethyl succinate or sterase<br>\(<41 \text{ kg}: 40 \text{ mg/kg/d divided bid–qid} × 10 \text{ d}\)<br>\(>41 \text{ kg}: 400 \text{ mg qid} × 10 \text{ d}\) | Resistance is uncommon in the United States<br>No difference in cure rate with all forms | Gastrointestinal upset | Drug of choice in penicillin-allergic patients<br>Equally as effective as penicillin in preventing all complications of group A streptococcus/B\(^b\) [40] |
| Drug          | Dosage Information                                      | Dosing Schedule | Spectrum | Other Considerations                                                                 |
|--------------|---------------------------------------------------------|-----------------|----------|-------------------------------------------------------------------------------------|
| Cephalexin   | Pediatric: 25–50 mg/kg/d divided bid × 10 d             | Twice-daily     | Broader  | Equal cure rate versus oral penicillin/Bb [41]                                      |
|              | Adults: 500 mg bid × 10 d                              |                 | spectrum |                                                                                    |
| Clindamycin  | Pediatric: 20 mg/kg/d divided tid × 10 d               | Unaffected by  | Expensive|                                                                                     |
|              |                                                        | beta lactamase  |          |                                                                                     |
|              |                                                        | Narrow spectrum |          |                                                                                     |
|              |                                                        | Eliminates      |          |                                                                                     |
|              |                                                        | carrier status  |          |                                                                                     |
|              |                                                        |                 |          |                                                                                     |
|              | Unaffected by beta lactamase                           |                 |          |                                                                                     |
|              | Pediatric: 20 mg/kg/d divided tid × 10 d               |                 |          |                                                                                     |
|              | Adults: 450 mg/d divided tid × 10 d                    |                 |          |                                                                                     |
|              |                                                        |                 |          |                                                                                     |

Blank entry, not recommended.

*a*Level A is consistent, good-quality patient-oriented evidence (SORT evidence rating system).

*b*Level B is inconsistent or limited-quality patient-oriented evidence (SORT evidence rating system).

*c*Level C recommendation is based on consensus, usual practice, opinion, disease-oriented evidence, or case series for studies of diagnosis, treatment, prevention, or screening.
appear within 1 week of the onset of clinical symptoms, peak in weeks 2 to 5, and may persist at low levels for up to 12 months. Measurements of EBV-specific antibodies are often obtained in lieu of the heterophile antibodies in athletes because they can determine the acuity of the illness.

**Treatment and Return to Play**

The mainstay of treatment for patients who have mono is supportive care. The treatment and return-to-play decision making is geared toward reducing the probability of splenic rupture. Acetaminophen or NSAIDs are recommended for the treatment of fever, pharyngitis, and malaise. It is important to stress adequate fluid and nutrition intake; adequate rest is necessary, but strict bed rest is not warranted. Corticosteroid and antiviral therapies have been studied in the treatment of mono. In a multicenter placebo-controlled trial of 94 patients who had acute mono, the combination of acyclovir and prednisolone reduced oropharyngeal shedding of the virus but did not affect the duration of symptoms or lead to earlier return to school or work [50]. Further, a meta-analysis of five randomized controlled trials of acyclovir in the treatment of acute mono failed to show a clinical benefit compared with placebo [51].

The athlete who has mono needs special attention, because there are strict guidelines for return to play in these individuals. To avoid splenic rupture, all athletes should not participate in sport activities while acutely ill from mono. Sports medicine physicians should recall that spontaneous or traumatic splenic rupture in the setting of mono usually occurs within the fourth to 21st day after the onset of clinical symptoms [45,52]. An athlete may return to easy and graduated training at 3 weeks if (1) the spleen is not palpably enlarged or painful, (2) the athlete is afebrile, (3) pharyngitis and any complications have resolved, and (4) liver enzymes are not grossly abnormal [53]. The athlete may return to contact sports and vigorous training (reconditioning necessary) at 4 weeks if these four conditions are met [53].

**SUMMARY**

URTIs are common acute illnesses, particularly in athletes because of the increased risk of transmission in this population. URTI management should take into account the type of illness, the potential complications of the illness, and the demand placed on the athlete to participate in practice and competition. The sports medicine physician should be armed with the latest evidence to accurately diagnose and properly treat acute URTI illness in the athlete. The overtreatment of URTIs with antibiotics, especially when not indicated, can lead to antibiotic resistance. Return-to-play decisions should be made on a case-by-case basis for the athlete and take into account the overall safety of the athlete and other teammates, coaches, and staff exposed to the athlete. The sports medicine physician represents the first line of defense in preventing, diagnosing, and managing acute URTIs in athletes and ensures the safe return of the athlete to competition.
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