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Pulmonary and Cardiac Infections in Athletes

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PNEUMONIA

Pneumonia is defined as an acute infection or inflammation of the pulmonary parenchyma. The term *community-acquired pneumonia* (CAP) is used when the patient has not been hospitalized or in a long-term facility for at least 14 days before the onset of symptoms [1]. It is estimated that 5 million cases of CAP, classified as typical or atypical, occur annually [2,3]. Typical pneumonias are most commonly caused by *Streptococcus pneumoniae* and found in very young or older patients. Atypical pneumonias are usually caused by *Legionella*, *Chlamydia*, or influenza and found most often in young adults and account for 20% to 40% of cases of CAP [4,5]. CAP is generally a serious illness with considerable morbidity and mortality, requiring increased recovery time for the athlete.

Clinical Presentation

Cough is the most common symptom in CAP. Symptoms may also include sputum production, shortness of breath, or chest pain [6,7]. Patients may present with nonspecific symptoms such as malaise, anorexia, headache, myalgias, fever, and chills [8]. Legionellosis may present with gastrointestinal symptoms such as nausea, vomiting, or diarrhea [9–11].

It is imperative to document vital signs (temperature, pulse, respiratory rate, blood pressure, and oxygen saturation) of any athlete who presents with a respiratory complaint. The vital signs on physical examination may reveal fever, tachycardia, tachypnea, hypoxemia, or hypotension [1]. The most common sign associated with CAP is fever [12]. Vital signs are important elements in the decision-making process for the appropriate management of CAP [13].

Examination may demonstrate dullness to percussion of the chest in a certain lobar distribution. Auscultation may reveal crackles, rales, or bronchial breath sounds. The patient may also exhibit increased tactile fremitus and egophony

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[1,12]. It is also important to document the patient's appearance and neurologic status [11].

Diagnosis

An infiltrate on chest radiograph is considered the "gold standard" for the diagnosis of pneumonia in the appropriate clinical scenario [8,14,15]. Other laboratory tests to consider, depending on the clinical severity, include leukocyte count, blood cultures, sputum culture with Gram stain, and urine antigens for *Streptococcus pneumoniae* and *Legionella*. The most common blood test abnormality found in CAP is leukocytosis with a leftward shift. These laboratory tests may not be indicated if the athlete is treated as an outpatient [8].

Treatment

When the diagnosis of CAP has been made, physicians must choose between inpatient or outpatient treatment for the athlete. A clinical predication tool, the pneumonia severity index, has been developed based on the likelihood of mortality of a CAP patient [13,16,17]. This index is useful for identifying patients who are at low risk of mortality from CAP and who can be safely treated as outpatients [12]. In the training room setting, the most useful indicators are vital signs and physical examination findings. The physician's clinical judgment should always override the index score.

The most common contraindications to outpatient treatment are inability to maintain oral intake, unstable vital signs, history of substance abuse, mental/cognitive impairment, or presence of comorbid conditions [12,16,18].

Because microbiologic data are not available at the time of clinical suspicion of CAP, most initial treatment regimens are empiric. Antibiotics that provide coverage against the most common organisms known to cause CAP (*Streptococcus pneumoniae*, *Mycoplasma pneumoniae*, *Chlamydia pneumoniae*, *Legionella*) should be selected. Macrolides are recommended if there are no significant risk factors for macrolide-resistant *Streptococcus pneumoniae* [18].

The American Thoracic Society (ATS) recommends not changing initial antibiotic treatment in the first 72 hours unless there is a worsening clinical situation [19]. Generally, most cases of CAP should be treated for 7 to 10 days. If atypical causes are suspected, therapy should last 10 to 14 days. The severity of the clinical presentation and the presence of coexisting illnesses should be considered in the determination of antibiotic duration [19,20].

Complications

Most patients recover from CAP without complications. One of the most common complications for the athlete is reactive airway disease. Pulmonary function tests may show decreased forced expiratory volume in 1 second (FEV_1). Transient airflow obstruction and hyper-responsiveness may be seen in these patients [21]. Up to 40% of patients may demonstrate decreased FEV_1 [22,23]. This abnormality typically resolves after 3 weeks but may last up to 2 months [22–24]. This potential complication could inhibit an athlete's full return to play. If clinically indicated, the athlete may respond to short-term inhaled bronchodilator therapy [25,26].

When the athlete has continued fever, other complications should be considered, such as pleural effusions, empyema, lung abscess, and secondary lung infection [1]. CAP patients are also susceptible to sepsis and meningitis [1].

Return to Play

Few studies are available on the amount of proper recovery time needed for athletes diagnosed with CAP. Athletes treated with an effective drug regimen usually show improvement of symptoms within 72 hours. A study by Metlay and colleagues [27] looked at time resolution of symptoms in patients who had CAP. Median time to resolution was 3 days for fever, 6 days for dyspnea, 14 days for cough, and 14 days for fatigue [27]. The athlete should be afebrile before return to training and competition. The athlete also should be re-evaluated by the team physician before clearance to assure normalcy of vital signs and respiratory status. It is recommended that exercise and training be resumed slowly when the athlete is able. For example, the first day should involve a 5- to 10-minute light elliptic or stationary bike workout. The athlete should be assessed the next day, and training may be advanced. Athletes can usually return to play sooner if they exercise early in the recovery and benefit psychologically if they see progress is being made.

ACUTE BRONCHITIS

Bronchitis is defined as inflammation of the bronchial mucous membranes. Acute bronchitis is a clinical syndrome characterized by cough (with or without sputum production) lasting up to 3 weeks, with evidence of concurrent upper airway infection [28,29]. Acute bronchitis is one of the most common conditions encountered in the primary care setting and a common ailment in the training room [29]. Acute bronchitis accounts for more than 10 million office visits yearly [30–32].

Causes

Respiratory viral infections are the most common causes of acute bronchitis. Less than 10% of patients have a bacterial etiology. The most common viruses associated with acute bronchitis include influenza A and B viruses, adenovirus, rhinovirus, parainfluenza virus, coronavirus, and respiratory synctial virus. The known bacteria that are significant agents in acute bronchitis are *Bordetella pertussis*, *Mycoplasma pneumoniae*, and *Chlamydia pneumoniae* strain TWAR [33,34]. As in CAP, the organism responsible for acute bronchitis is unlikely to be identified in the ambulatory setting. When contemplating treatment options, it is important for the physician to understand the limited role of bacterial agents in acute bronchitis. Acute bronchitis is one of the most common examples of misuse of antibiotics by the primary care physician [35].

Clinical Evaluation

Cough is the most common symptom in acute bronchitis. The patient may or may not have sputum production. Fever is unusual in acute bronchitis. If fever is present, the clinician should consider influenza or pneumonia [36,37]. The

patient may also complain of concurrent or prodromal symptoms of an upper respiratory infection (URI), including pharyngitis, coryza, and fatigue [38]. Most URI symptoms improve within 5 to 7 days [39]. In acute bronchitis, the cough can last up to 3 weeks [21].

It is imperative to document vital signs (temperature, pulse, respiratory rate, blood pressure, and oxygen saturation) of any athlete who presents with a respiratory complaint. Examination often reveals findings similar to URI symptoms: pharyngeal erythema, anterior cervical lymphadenopathy, and rhinorrhea [14].

Diagnosis

The diagnosis of acute bronchitis is considered a clinical diagnosis and should be suspected in cases of acute respiratory disease with prolonged cough that continues after other signs and symptoms of acute infection have resolved [38]. It may not be necessary to obtain any further studies in the appropriate clinical situation. Abnormal vital signs (pulse >100, respiratory rate >24, or temperature >38°C) are an indication to consider further testing such as a chest radiograph. Physical examination findings on chest examination of rales on auscultation or dullness to percussion are not consistent with acute bronchitis and require further investigation [14]. Other diagnoses to consider in an athlete complaining of cough are postnasal drip, sinusitis, asthma, and gastroesophageal reflux.

Treatment

When the clinical diagnosis of acute bronchitis has been established, the recommended therapy is symptomatic. The physician may choose to use acetaminophen, ibuprofen, and nasal decongestants if appropriate. Routine antibiotic treatment of uncomplicated acute bronchitis is not recommended because the primary causes are most often viral infections [40,41]. The exception to this is in the setting of *Bordetella pertussis* infection, which is discussed in detail later.

Complications

Pulmonary function test abnormalities may also be seen in athletes who have acute bronchitis. Transient airflow obstruction and hyper-responsiveness may be seen in these patients [21]. Up to 40% of patients may demonstrate decreased FEV_1 [22,23]. This abnormality typically resolves after 3 weeks but may last up to 2 months [22–24]. This potential complication could inhibit an athlete's full return to play. If clinically indicated, the athlete may respond to short-term inhaled bronchodilator therapy [25,26]. It has been the authors' experience that athletes return to sport sooner when the reactive airway disease is treated. Athletes who have asthma or other lung conditions may have worsening symptoms.

Return to Play

There are little data regarding appropriate return-to-play guidelines for athletes who have acute bronchitis. It is important that there is proper follow-up with the team physician to ensure resolution of symptoms and to guarantee a clinical

situation that does not worsen. All respiratory symptoms should be closely monitored by the athletic training staff. If the athlete's symptoms do not resolve with symptomatic treatment, then the physician should consider other diagnoses, and further workup is necessary [42].

PERTUSSIS

Bordetella pertussis, a gram-negative coccobacillus, is a commonly undiagnosed cause of acute bronchitis [43]. Pertussis, also known as whooping cough, is an acute, highly contagious infection of the respiratory airways. Pertussis is transmitted person to person by contact with aerosolized droplets [44]. One active case can infect 70% to 100% of household contacts and 50% to 80% of school contacts [45]. Because vaccine and natural immunity wane with age, pertussis has become a disease of adolescents and adults [46]. Due to the amount of time that athletes spend training together and the high infectivity of pertussis, this diagnosis must not be missed in the training room.

Clinical Presentation

The classic clinical course of pertussis is divided into three stages: catarrhal, paroxysmal, and convalescent (Box 1) [44,47,48]. Adolescents and adults may not display the typical phases of childhood infections. In adults, the disease may be characterized by a persistent cough with URI symptoms [49]. This presentation is likely to be the one encountered in the training room. Athletes may report a cough with a paroxysmal quality lasting more than 2 weeks, post-tussive emesis, or inspiratory whooping [50].

Box 1: Stages of pertussis

Catarrhal phase
Lasts 1 to 2 weeks
Most contagious phase
Clinically resembles URI
Cough increases in severity and frequency

Paroxysmal phase
Lasts 3 to 6 weeks
Clinically—spells of coughing with characteristic inspiratory whooping
Post-tussive vomiting, cyanosis, and apnea

Convalescent phase
May last 2 to 12 weeks
Cough still present
Paroxysms may recur with respiratory infection

Diagnosis

The most reliable diagnostic test for pertussis is by detection of the organism from nasopharynx secretions. The sensitivity of this test, however, is estimated to be 25% to 50% [51]. The most sensitive test (80%–100%) is polymerase chain reaction (PCR). Although PCR is a rapid and highly specific test, there is not yet a universally accepted technique. Nasopharyngeal culture is therefore recommended to make the definitive diagnosis [52]. For best yield, the nasopharyngeal swab should be inserted into the base of the nostril and remain in the posterior pharynx for 10 seconds before being withdrawn [48]. In the United States, physicians are legally required to report pertussis cases to state health department officials [53,54]. The Centers for Disease Control and Prevention (CDC) recommends that physicians report and treat pertussis when there is clinical suspicion and not wait for laboratory confirmation [54].

Treatment

In the case of proven or presumed infection, therapy should be started as soon as pertussis is suspected [28]. The recommended treatment is 2 g/d of erythromycin in four divided doses for 14 days [55,56]. If the athlete is unable to tolerate erythromycin, then two alternative regimens have shown equal efficacy: azithromycin and clarithromycin [57,58]. Azithromycin dose for adults is 500 mg in a single dose on day 1 then 250 mg per day on days 2 through 5 [59]. Clarithromycin dose for adults is 1 g per day in two divided doses for 7 days [59]. Trimethoprim-sulfamethoxazole is an additional option for those who cannot tolerate macrolides. Athletes who have confirmed or probable pertussis should be isolated for 5 days from the start of treatment [28].

Prevention

Because the vaccine and natural immunity wane with age, it is recommended to extend immunization with the tetanus toxoid–reduced diphtheria toxoid–acellular pertussis (Tdap) vaccine to the adolescent population. The CDC recommends a single dose of the Tdap vaccine (0.5 mL intramuscularly) for 11- to 18-year-olds who require a booster dose, provided they have completed the recommended primary diphtheria-tetanus-pertussis vaccine series [60,61]. The Advisory Committee of Immunization Practice also recommends a single dose of the Tdap vaccine for adults 19 to 64 years of age [59,61].

Prophylaxis

Athletes known to be in close contact with a known or suspected case of pertussis should be given prophylactic antibiotic treatment. The recommended regimen is full dosing of erythromycin for 14 days [61–63]. If erythromycin cannot be tolerated, then a 5-day course of azithromycin is acceptable [61].

Complications

Pertussis infections in the training room can lead to rapidly spreading illness among other athletes and staff. Pertussis can also cause reactive airway disease and bronchitis. Pertussis can be complicated by pneumonia, dehydration,

weight loss, and sleep disturbance–all which can affect an athlete's return-to-play status and overall performance [33].

Return to Play

Athletes who have confirmed or probable pertussis should be isolated for 5 days from the start of treatment to prevent spreading the disease [28]. When reactive airway disease is involved, those symptoms should also addressed, as noted in the bronchitis section.

INFLUENZA

Influenza is an acute respiratory illness cause by influenza A or B viruses. Influenza is a common seasonal cause of acute bronchitis [41].

Clinical Presentation

The diagnosis of influenza should be considered if the athlete presents during the winter months with the abrupt onset of fever, headache, myalgias, malaise, nausea, and vomiting. These symptoms are generally accompanied by cough and sore throat [64–67].

In uncomplicated influenza, there are few physical examination findings. The health care provider in the training room should document vital signs. The patient may appear flushed. The findings may also include mild cervical lymphadenopathy and hyperemic oropharynx. The eyes may be watery or reddened [68]. Otherwise, the examination may be unremarkable. It is important to assess the athlete's hydration status and neurologic status on examination.

Diagnosis

Outpatient laboratory diagnosis of influenza can be accomplished by the detection of the virus or viral antigen in nasal washes or throat swabs [69]. The virus may also be detected from sputum samples [68]. Rapid viral diagnostic tests are available for the ambulatory setting.

Treatment

Two classes of antiviral drugs are available for the treatment of influenza [70,71]. The neuraminidase inhibitors zanamivir and oseltamivir are active against influenza A and influenza B. The M2 inhibitors amantadine and rimantidine are active against influenza A only [71]. The maximum benefit of treatment is available if given within the first 24 to 30 hours of symptoms and in patients who have fever at the time of presentation [72–74]. With appropriate treatment, the patient may have 2 to 3 days' shortening of the duration of symptoms [71].

Symptomatic treatment is also important in influenza. Acetaminophen or ibuprofen may be beneficial for fever, headache, or myalgias. The use of aspirin in pediatric patients who have influenza should be avoided due to the risk of Reye's syndrome in this population [75]. Cough suppressants may be helpful in the appropriate clinical scenario. The athlete may also benefit from inhaled bronchodilator therapy if bronchial hyper-responsiveness and decreased FEV_1 are present [33]. Athletes should be instructed to maintain proper hydration and rest during the acute illness.

Complications

Close follow-up of athletes who have severe influenza illnesses is imperative to ensure that no complications are arising. Dehydration and acute bronchitis are common complications of influenza [68]. Although rare, complications of influenza include pneumonia, myositis, rhabdomyolysis, myocarditis, encephalitis, meningitis, and Guillain-Barré syndrome [76].

Prevention

There are measures available to help prevent the illness caused by influenza. Annual vaccination is available. The currently available injectable vaccines are inactivated preparations of whole virus or split product. The whole virus vaccine is not available in the United States [72]. The United States has also made an intranasal live-attenuated vaccine available for healthy patients aged 5 to 49 years [77]. Although athletes are not on the CDC target-group list for vaccination, vaccination can be important tool to reduce the number of cases in training rooms.

Return to Play

To prevent the spread of influenza, the athlete should be kept from the training room, practices, and competitions until 5 days after the onset of symptoms. Return to full activity should be delayed until the illness has fully resolved. Athletes should be evaluated for any signs of fever, dehydration, or impaired respiratory status before full clearance.

MYOCARDITIS

Myocarditis is an inflammatory disease of the cardiac muscle that can have a wide spectrum of clinical presentations and outcomes. Myocarditis is one of the most challenging diagnoses in cardiology. Acute myocarditis can progress to dilated cardiomyopathy, heart failure, arrhythmias, and death [78]. If unrecognized in the training room, myocarditis can produce lethal results.

Causes

Myocarditis has a wide variety of infectious and noninfectious causes. The most common infectious causes are viruses. The most frequently associated viruses are coxsackievirus B, adenovirus, hepatitis C virus, cytomegalovirus, echovirus, influenza virus, and EBV. The most common causes of myocarditis found in the training room are likely viral illnesses, especially coxsackievirus B, adenovirus, and echovirus [79]. Myocarditis can also result from drug hypersensitivity, radiation, and chemical or physical agents [80,81].

Clinical Presentation

The diagnosis of myocarditis requires a high index of suspicion in the appropriate clinical setting [79]. A wide range of symptoms can be present in an athlete suffering from myocarditis. The patient may be asymptomatic or may simply give a history of a preceding URI or flu-like syndrome. The patient may also present with chest pain or symptoms of heart failure [78,79]. The athlete may present with fever, malaise, and arthralgias [82]. The diagnosis of

infective myocarditis should be considered when an athlete presents with cardiac complaints or arrhythmia issues in the course of a recognized systemic infection.

It is imperative to document vital signs in the training room. The physical examination may be normal. When the myocarditis is severe, the cardiac examination may reveal tachycardia, a muffled first heart sound along with a third heart sound, and a murmur of mitral regurgitation (MR). The examination may also reveal findings of heart failure such as edema and pulmonary crackles from fluid overload, depending on the severity of the illness. When there is associated pericarditis, a pericardial friction rub may be heard [78,79]. The examination may also reveal findings consistent with a URI.

Diagnosis

Routine blood and urine laboratory tests are generally normal in myocarditis. Cardiac enzymes may be elevated, specifically the Myoglobin binding (MB) fraction of creatine kinase (CK-MB) and troponin I [83,84]. The EKG may be normal or abnormal. The most common EKG findings are transient, nonspecific ST-T wave abnormalities. Chest radiograph findings range from normal to cardiomegaly. Pulmonary vascular congestion and edema may be exhibited in severe cases. One of the cardinal features of myocarditis can be found on echocardiography. Echocardiography may reveal decreased ventricular function. The ventricular dysfunction is generally global. Impairment of myocardial contractility may be evident on exercise-induced echocardiogram views. Echocardiography may also reveal increased left ventricular (LV) diastolic dimensions with normal septal thickness [85].

Cardiac MRI is becoming a more widely available tool to detect myocardial abnormalities. In myocarditis, the MRI may demonstrate myocardial edema and myocyte damage [86,87]. The definitive diagnosis of myocarditis is made by endomyocardial biopsy with histologic evaluation [88]. Histologic evaluation of the biopsy shows mononuclear cellular infiltrates, myocyte necrosis, and disorganized myocardiac cytoskeleton [89,90].

Treatment

Viral myocarditis is usually a self-limited disease, and treatment is generally supportive. Myocarditis, however, may progress to dilated cardiomyopathy and heart failure. Most therapy regimens are directed toward treatment of the heart failure and potential arrhythmias in serious cases [78]. Depending on the clinical situation, some patients may benefit from antiviral or immunosuppressive therapy [91–93].

Complications

Most patients who have viral myocarditis recover completely [94]; however, athletes who have viral myocarditis are at risk for heart failure, cardiomyopathy, and associated pericarditis. These athletes are also at risk for arrhythmias and sudden cardiac death [95].

Return to Play

Exercise and training can be deleterious in athletes who have myocarditis. Based on the current Bethesda Conference recommendations, athletes who have "probable or definitive evidence of myocarditis should be withdrawn from all competitive sports and undergo a prudent convalescent period of about six months following the onset of clinical manifestations" [95]. After 6 months, athletes may return to training if the following conditions are met [95]:

- LV function, wall motion, and cardiac dimensions return to normal
- Clinically relevant arrhythmias are absent on ambulatory Holter monitoring and graded exercise testing
- Serum markers of inflammation and heart failure have normalized
- The EKG has normalized

PERICARDITIS

Pericarditis (inflammation of the pericardium) may be caused by a wide variety of infectious and noninfectious processes [96,97]. Pericarditis can have a wide range of clinical presentations, from asymptomatic to severe hemodynamic compromise. Taking a careful history and knowledge of the clinical presentation of pericarditis are important in establishing the diagnosis. When the diagnosis is missed, pericarditis can become life threatening for the athlete [98].

Causes

Pericardial disease has multiple causes including infectious, neoplastic, inflammatory, degenerative, vascular, and idiopathic causes. Infectious and idiopathic causes, likely the most common causes in the training room, are found in 90% of cases of acute pericarditis [99,100]. The most common viral causes include coxsackievirus A and B, adenovirus, echovirus, and HIV. The most common bacterial causes in acute pericarditis are *Staphylococcus*, *Pneumococcus*, *Streptococcus*, *Haemophilus*, and *Neisseria* [101,102].

Clinical Presentation

The presentation of acute pericarditis varies depending on the cause. In infectious or idiopathic acute pericarditis, the major clinical symptom is chest pain. The pain in pericarditis is thought to be due to inflammation of the adjacent pleura [103]. The patient may describe the pain as retrosternal, exacerbated by coughing or deep inspiratory effort. The pain may also radiate to the back. The chest pain in acute pericarditis is often positional—worsened in the supine position and relieved by sitting upright and leaning forward [97,102,104]. The athlete may also complain of fever. Patients may also present with an associated flu-like illness with cough, fatigue, myalgias, or arthralgias [105].

It is imperative to document vital signs for athletes who have cardiac or respiratory complaints. The vitals signs may indicate severity of cardiac compromise. The pericardial friction rub is the cardinal physical sign of acute pericarditis [99]. A pericardial rub may have three components per cardiac cycle: high pitched, scratching, and grating [106]. The rub can sometimes be

elicited by use of firm pressure with the stethoscope's diaphragm at the left lower sternal border of the chest wall [96,106]. The rub can often be best appreciated with the patient upright and leaning forward and is often accentuated during inspiration [107]. The physician should also look for signs of cardiac tamponade on examination: hypotension, tachycardia, jugular venous distention, and pulsus paradoxus (defined as an inspiratory systolic decrease in arterial pressure of 10 mm Hg during normal breathing) [98].

Diagnosis

Laboratory tests to consider include complete blood count, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), and cardiac enzymes. A complete blood count may illustrate increased leukocyte count [97]. Laboratory signs of inflammation including elevated ESR and CRP are commonly found in patients who have acute pericarditis. ESR and CRP are not highly specific findings because they can be elevated in multiple disease processes. Serum cardiac enzymes, CK-MB, and troponin I may also be elevated in acute pericarditis [104,108]. If the history and physical examination are appropriate, further laboratory testing should be ordered, including antinuclear antibody, tuberculin skin test, HIV serology, and blood cultures [109].

The EKG is abnormal in 90% of patients who have acute pericarditis [101,110–112]. The characteristic EKG changes found often evolve through stages. Early in pericarditis (the first few hours to days), ST-segment elevation without change in QRS morphology occurs in multiple leads. PR-segment depression may also be present. Several days later, the ST and PR segments return to baseline. This stage is followed by diffuse T-wave inversions. The EKG may normalize or the T-wave inversion may persist for weeks or months [102,104].

In acute pericarditis, the chest radiograph is generally normal; however, when at least 200 mL of pericardial effusion is present, the chest radiograph may reveal an enlarged cardiac silhouette [102]. An echocardiogram should also be obtained in patients who have suspected acute pericarditis. The echocardiogram is often normal unless there is an associated pericardial effusion [113,114].

Treatment

The physician's initial treatment decision is whether the athlete will be treated as an inpatient or an outpatient. If the athlete has simple, uncomplicated acute pericarditis and is clinically stable, then outpatient treatment with close follow-up may be appropriate [97,115]. If high-risk features are present or if the patient is clinically unstable, then inpatient treatment is recommended. High-risk features are illustrated in Box 2 [97,115].

When the clinical situation identifies a cause other than viral or idiopathic disease, specific treatment is indicated for the underlying disorder. Primary therapy goals for idiopathic or viral pericarditis are pain relief, resolution of inflammation, and resolution of effusion if present [97]. Current recommendations include the use of aspirin and other nonsteroidal anti-inflammatory drugs (NSAIDs). Colchicine may also be considered in the treatment of acute

Box 2: High-risk features in acute pericarditis

Subacute onset

Leukocytosis

Evidence of cardiac tamponade

Fever (>100.4°F)

Acute trauma

Immunosuppressed state

Large pericardial effusion without significant response to nonsteroidal anti-inflammatory drug (NSAID) treatment

History of oral anticoagulant therapy

Failure to respond to NSAID therapy within 7 days

pericarditis [116]. Corticosteroids should be considered if the patient is refractory to NSAIDs or colchicine. Close monitoring and follow-up are imperative for all athletes diagnosed with acute pericarditis.

Complications

Although pericarditis usually resolves within a few days to weeks, life-threatening complications can occur [97]. When an associated pericardial effusion is present, it may proceed to a cardiac tamponade, which is a cardiac emergency [98]. When the pericardial inflammation does not resolve, it may lead to chronic pericarditis. Chronic pericarditis may subsequently lead to constrictive pericarditis.

Return to Play

The current Bethesda Conference Guidelines recommend exclusion of the athlete who has acute pericarditis from competitive sports [95]. These athletes can return to full activity only when there is no evidence of active disease, which includes no evidence of effusion on echocardiogram and normalized serum inflammatory markers. If concurrent myocarditis is associated with acute pericarditis, then myocarditis return-to-play criteria must also be met [95].

ACUTE RHEUMATIC FEVER

Acute rheumatic fever (ARF) is an inflammatory disease that may develop after an infection with *Streptococcus* bacteria and can involve the heart, joints, skin, and brain [117]. The cardiac manifestations associated with ARF—valvulitis and carditis—can be potentially serious illnesses found in the training room. The carditis of ARF is a pancarditis that involves the pericardium, myocardium, and endocardium to varying degrees [118]. The valvulitis most frequently affects the mitral valve, aortic valve, or both [117]. Although the incidence of ARF has declined dramatically in the United States, scattered outbreaks in North America have confirmed the potentially serious consequences of this infection [119,120].

Cause

ARF results from infection with a "rheumatogenic" strain of group A streptococcus. The known serotypes associated with ARF are serotypes 1, 3, 5, 6, 14, 18, 19, 24, 27, and 29 [121]. ARF primarily affects children between age 6 and 15 years and occurs approximately 20 days after initial infection [122]. Studies have shown that an estimated 3% of individuals who have untreated group A streptococcal pharyngitis develop rheumatic fever [123].

Clinical Presentation

The clinical presentation is variable. The Jones criteria shown in Box 3 are established guidelines to aid in the diagnosis [118,124,125].

The onset of rheumatic fever follows a latent period of 7 to 35 days after a preceding group A streptococcal infection [126]. Although patients who have ARF may have any or all of the Jones criteria clinical features, the most common are polyarthritis (50%–75%) and carditis (40%–60%) [117].

On examination, the carditis is usually associated with a murmur of valvulitis [118]. The examination may reveal sinus tachycardia, an S_3 gallop, a pericardial friction rub, and cardiomegaly. The valvulitis may be characterized by a pansystolic murmur of MR, best heard at the apex, with radiation to the left axilla. The MR murmur may also be heard with or without a low-pitched mid-diastolic (Carey Coombs) murmur [127].

Diagnosis

The diagnosis of ARF is clinical but requires supporting evidence from clinical presentation and microbiologic and immunologic laboratory results. To fulfill the Jones criteria, two major criteria (or one major and two minor criteria) plus evidence of an antecedent streptococcal infection are required [118,124,125]. Throat cultures should be obtained in suspected ARF [117]. Specific antibody tests, such as antistreptolysin and anti-DNAse B should also be obtained to help confirm the diagnosis [117]. Acute phase reactants, CRP,

Box 3: Jones criteria

Major

Carditis
Polyarthritis
Chorea
Erythema marginatum
Subcutaneous nodules

Minor

Fever
Arthralgia
Previous rheumatic fever or rheumatic heart disease

and ESR are also usually elevated in ARF [117]. EKG and echocardiography are important diagnostic tools to assess for cardiac involvement [118,124,125]. The EKG may show prolonged PR intervals, which is a nonspecific finding [118]. The echocardiogram also reveals associated valvulitis or pericarditis, if present.

Treatment

Hospital admission is recommended for all cases to ensure complete and proper investigation. The main treatment goals are to confirm the diagnosis, treat cardiac failure, shorten the duration of symptoms, and ensure ongoing secondary prophylaxis and clinical follow-up [128]. The mainstay of treatment for ARF is NSAIDs, most commonly aspirin. Duration of NSAID treatment should be maintained until all symptoms have resolved and laboratory values are normal [129]. Depending on the severity of carditis, steroid treatment may be indicated. Antibiotic treatment with penicillin should also be given for 10 days [123]. The athlete also needs long-term antibiotic prophylaxis after the acute episode has resolved. All family contacts should be cultured and treated for streptococcal infection if indicated [123].

Complications

ARF can cause permanent cardiac damage. The mitral valve is more commonly involved than the aortic valve. Mitral stenosis (MS) is the classic finding in rheumatic heart disease and may require surgical correction [95]. Other potential complications of ARF include heart failure, myocarditis, pericarditis, arrhythmias, and endocarditis. The arrhythmia most commonly associated with MS is atrial fibrillation [117,126]. The athlete must have close monitoring and follow-up before any return to exercise.

Return to Play

If the athlete has no cardiac involvement with ARF, then after antibiotic treatment is complete and the athlete is afebrile, gradual return to play may be initiated with close physician observation (normally about 3 to 4 weeks into treatment). The athlete should also have resolution of polyarthralgias and chorea if present before return to play. Prolonged bed rest is no longer recommended after ARF [95].

All athletes who have cardiac involvement should be followed by their primary care physician, cardiologist, and dentist. When there is associated myocarditis or pericarditis, physicians should refer to the previously described return-to-play guidelines. Although MS rarely causes sudden cardiac death, careful consideration must be given if MS is present in an athlete [95]. Exercise in athletes who have MS can cause sudden increases in pulmonary capillary and pulmonary artery pressures, resulting in sudden acute pulmonary edema. It is important to assess the severity of MS at rest and during sport-related exercise with echocardiography, including measurement of pulmonary artery systolic pressure [95]. Depending on the severity of MS, the Bethesda Conference Guidelines should be followed [95].

ENDOCARDITIS

Infective endocarditis (IE) is a serious febrile infection that rapidly damages cardiac structures, spreads to extracardiac sites and, if untreated, can progress to death within weeks [130]. To avoid overlooking the diagnosis of IE, a high index of suspicion must be maintained. In the training room, the most likely case of endocarditis may be found in an athlete who has structural heart disease such as bicuspid aortic valves, mitral valve prolapse, or rheumatic heart disease.

Causes

A variety of microbial agents can cause IE. Staphylococci, streptococci, and enterococci represent most cases. The most common risk factor in athletes is structural heart disease. The skin, upper respiratory tract, and oral cavity are the primary portal of entry for streptococci and staphylococci organisms [131,132]. Bacteremia can then ensue, leading to seeding of cardiac and extracardiac sites.

Clinical Presentation

In IE, the interval between the presumed initiating bacteremia and the onset of symptoms is less than 14 days [133]. Endocarditis symptoms may develop slowly (subacute) or suddenly (acute) [134]. Fever is the most common symptom. Other common symptoms include chills, night sweats, anorexia, dyspnea, cough, chest pain, and myalgias [134]. The most common findings on physical examination are fever and a heart murmur. The murmur is usually a regurgitant heart murmur in the mitral or aortic valve position. In an athlete who has a pre-existing murmur, a new or changing murmur may be noted. Other findings on examination may include splenomegaly or cardinal peripheral manifestations such as petechiae, splinter hemorrhages, Osler's nodes, Janeway's lesions, or Roth's spots [130].

Diagnosis

The diagnosis of IE should be investigated when athletes who have fever also present with one or more of the cardinal manifestations of IE. The incorporation of clinical, laboratory, and echocardiographic data is central to the diagnosis [134,135]. Nonspecific laboratory findings may include leukocytosis and elevated ESR and CRP [135]. EKG may reveal new atrioventricular, fascicular, or bundle branch block depending on cardiac involvement [134]. The modified Duke criteria represent a diagnostic guideline for evaluating patients who have suspected IE that takes into account blood culture results, echocardiogram criteria, and history and physical examination characteristics [136].

Treatment

Treatment with parental antibiotics is usually started in the hospital but may be completed as an outpatient when the patient is afebrile and follow-up blood cultures are negative [135]. Antibiotic therapy should be selected as appropriate based on blood culture and sensitivities results. Initial therapy in native-valve IE with no history of intravenous drug abuse should be directed against streptococci organisms. Penicillin and gentamycin remain first-line therapy in this

situation. Depending on the pathogen involved, antibiotic treatment should last between 2 and 6 weeks [135].

Complications

Valvular damage may lead to aortic regurgitation (AR) or MR in patients who have IE. If left untreated, IE can progress to severe heart failure and potentially fatal arrhythmias [135]. In addition, complications from septic emboli may result, such as stroke, kidney failure, or pulmonary embolism.

Return to Play

From an infectious standpoint, before return to competition, the athlete should complete at least 2 to 6 weeks of appropriate antibiotic treatment and remain afebrile with negative follow-up blood cultures. Athletes require close monitoring with frequent follow-up. When the antimicrobial treatment is complete, repeat echocardiography should be performed to establish a new baseline [135]. Repeat physical examinations are important to look for any signs of heart failure. Before any initiation of antibiotic therapy for any febrile illnesses, the athlete should have three sets of blood cultures obtained from separate sites. The athlete also requires thorough dental evaluations to ensure oral hygiene.

From a cardiac standpoint, the athlete may have residual MR or AR. Athletes who have MR from IE may be restricted from competition. Current recommendations are based on the severity of MR, echocardiogram findings of LV size and function, and pulmonary artery pressure readings [95]:

- Athletes who have mild to moderate MR in normal sinus rhythm, with normal LV size, LV function, and pulmonary artery pressures, can participate in all competitive sports.
- Athletes who have mild LV enlargement (<60 mm) may participate but are restricted to certain classes of sports.
- Athletes who have severe MR, LV enlargement (>60 mm), LV systolic dysfunction, or elevated pulmonary artery pressures should not participate in any competitive sports.

If AR is present in any athlete who has IE, the current recommendations are to assess the severity of AR with echocardiography and measurement of LV end diastolic size [95]:

- Athletes who have mild to moderate AR and normal LV end diastolic size may participate in all competitive sports.
- Athletes who have severe AR and increased LV diastolic diameter (>65 mm) should not participate in sports.
- Symptomatic athletes who have mild to moderate AR should not participate in sports regardless of LV size.

SUMMARY

Pulmonary and cardiac infections in the athlete can have a wide range of presentations and complications. These infections may present few problems for

the training athlete or become life-threatening. The team physician must be able to recognize the diagnosis, give the appropriate treatment, understand the potential complications, and ensure proper follow-up and return-to-play protocols.

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