Mononucleosis and Athletic Participation: An Evidence-Based Subject Review

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Abstract: Infectious mononucleosis (IM) is a common medical condition that afflicts thousands of young athletes each year. Despite the self-limited nature of this disorder, the variability of the clinical presentation and the rare risk of splenic rupture routinely present sports medicine clinicians with difficult return-to-play decisions. Currently there are no evidence-based guidelines regarding the management of the athlete with IM. This review discusses the available research data pertaining to the management of IM in young athletes and develops questions for future clinical research.

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EPIDEMIOLOGY

Infectious mononucleosis (IM) is caused by the Epstein-Barr virus (EBV), which is a member of the herpes virus family. It eventually infects almost 90% of adults, although many individuals are not aware they had the disease, which may be an expression of the variability of the immune response to infection.[1] The peak incidence in developed countries has been reported in adolescents and young adults.[2,3] It is estimated that 30% to 50% of freshman entering college remain susceptible to infection, with an annual incidence of 1% to 3% for developing mononucleosis.[4] This translates into a rate of infection of nearly 15% in previously unexposed individuals.[5] There is no evidence to suggest that IM is more or less prevalent in the student-athlete population than among nonathletes.

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NATURAL HISTORY AND CLINICAL MANIFESTATIONS

Humans are the only source of this infection,[6] and transmission occurs primarily through oral secretions, thus its popular description as "the kissing disease."[7] Other modes of transmission include close contact such as sneezing or sharing cups or food. Transmission via fomites and the period of communicability has not been determined. Among youth, symptoms of EBV infection are less severe than in older adults. The incubation period is long (30–50 days), making the source of infection difficult to determine. Hoagland, in 1975, described the classic triad of fever, pharyngitis, and lymphadenopathy.[8] Many have a prodrome of malaise and headache, and fever (rarely >104°F, >40°C) can last up to 3 weeks. Pharyngitis occurs in ~80%; splenomegaly in 50%–100%, palatal petechiae in ~25%, and rash on the trunk and upper arms in ~10%–40%.[9,10] Rash is significantly more common if the patient has been treated with ampicillin or amoxicillin, and it does not represent an allergy to these antibiotics. Occasionally, patients can present with periorbital edema. Jaundice, central nervous system complications, and myocarditis are rare. Exudative pharyngitis and tonsillar enlargement can cause airway compromise.[11] The illness ranges in severity and can be especially severe in individuals who are immunocompromised. Symptoms usually resolve in 4–8 weeks, although in some individuals a more protracted time course occurs.

The most common features that should prompt laboratory confirmation of IM include fever and sore throat with exudative pharyngitis and possible palatal petechia along with splenomegaly and/or painful posterior cervical adenopathy.[12,13] As will be discussed, the ability to determine splenomegaly by physical examination is limited; thus its absence should not preclude the possibility of IM. The following section discusses the clinical findings in more detail.

CLINICAL ASSESSMENT

The history and physical examination can assist in establishing the diagnosis, screening for complications of IM, and risk stratifying for treatment options. In establishing the diagnosis, Hoagland's criteria are frequently cited: at least 50%

lymphocytes and at least 10% atypical lymphocytes in the presence of fever, pharyngitis, and adenopathy and confirmed by a serologic test.[8,14] Approximately 50% of patients with symptoms suggestive of IM and a positive heterophile antibody test meet all of Hoagland's criteria.[14] Serologic evaluation is currently recommended to confirm a diagnosis of IM.[13]

Lymphadenopathy

The characteristic lymph node involvement in IM involves the posterior cervical chain more than the anterior chain. Lymphadenopathy may also involve the axillary and inguinal regions in IM, which helps to distinguish it from other etiologies of pharyngitis. Nodes are typically large and moderately tender, peaking early in the illness and subsiding over a 2- to 3-week period.[12,15]

Oropharyngeal Findings

The pharyngeal assessment is important because a number of disorders may cause tonsillar inflammation, including streptococci (coexistent in up to 30% of cases), *gonococcus,* and other viruses including human immunodeficiency virus (HIV). Exudative pharyngitis is seen in more than 50% of patients with IM; the hyperplasia on the tonsils may be marked and the exudation may be white or gray–green or even display necrotic features. Some reviewers have noted that the presence of palatal petechiae may be present, with 1 study suggesting that the presence of palatal petechiae, gastrointestinal symptoms, and an insidious onset of symptoms portend a longer duration of illness.[12,15–17]

Skin Findings

The skin may present with a maculopapular, urticarial, or petechial rash. Rash is more common with concomitant administration of ampicillin, but it also has been described with azithromycin, levofloxacin, and cephalexin. Petechial rashes also should alert the provider to any potential underlying hematologic complication such as aplastic anemia, thrombocytopenia, or disseminated intravascular coagulation.[12,15]

Abdominal Findings

The identification of splenomegaly and its clinical significance have been the focus of great controversy. Several studies have assessed the clinical reliability of splenomegaly by physical examination. These studies have demonstrated a sensitivity ranging from 20% to 70% and a specificity ranging from 69% to 100%.[18–20]

One prospective study demonstrated that the maximum clinical utility of bedside examination occurred when both palpation and percussion tests were positive. However, the presence of positive palpation and percussion signs had a combined sensitivity of only 46%, although the specificity was 97%.[18] The most effective percussion or palpation techniques have not been determined, and the ability to teach and assess clinical examination of the spleen has not been assessed. In addition, there have been no studies specifically designed to assess the reliability of clinical examination in athletes, in whom large size or a well-developed abdominal musculature may further challenge the best of examiners. Finally, there have been case reports of iatrogenic splenic rupture as a result of deep palpation during an abdominal examination.[21,22]

LABORATORY ASSESSMENT

Laboratory studies that support the diagnosis of IM include an absolute and relative lymphocytosis, with typically greater than 10% atypical lymphocytes on a peripheral blood smear, and a positive heterophile test. The heterophile antibody latex agglutination test has been reported to have a sensitivity and specificity of 87% (range 79%–95%) and 91% (range 82%–99%), respectively, in patients older than age 16 years when several studies are evaluated.[14] False-positive test results are rare but can occur in lymphoma, hepatitis, or autoimmune disease.[23] The false-negative rate may be higher during the first week of illness (25%) and decreases with subsequent testing (5%–10% at week 2 and 5% at week 3), underscoring the usefulness of repeating this test if clinically indicated.[8] Alternatively, more specific EBV tests can be considered.[24]

The more specific antibody tests for EBV include viral capsid antigen (VCA), immunoglobulin G (IgG) and VCA-IgM antibodies, early antigen IgM, and antibody to Epstein-Barr nuclear antigen (EBNA). VCA IgM appears early in infection and disappears at the 4–6-week mark. Antibody to early antigen is usually short-lived, peaking at 3–4 weeks after onset, and is often not included in EBV panels. VCA IgG appears during acute infection, peaks at 2–4 weeks after onset, then declines slightly and persists for life. EBNA antibody generally appears only after 2–4 months and also persists for life.[25]

VCA IgM and IgG antibodies are highly sensitive and specific for diagnosing infectious mononucleosis (sensitivity 97%, specificity 94%)[14], and slightly better than a positive heterophile test (positive likelihood ratio of 16 versus 9.7–28).[26,27] However, when VCA IgM and IgG test results are negative, they are superior in ruling out IM caused by EBV (negative likelihood ratio of 0.03 versus 0.14 to 0.18 for heterophile antibody tests).[26,27] Because EBNA appears late in the course of IM and persists for life, its presence in the course of acute illness should lead one to consider diagnoses other than EBV.

Detection of EBV can be done from cultures of circulating lymphocytes or molecular amplification of EBV DNA in circulating lymphocytes. These tests are not generally needed for diagnosis of infectious mononucleosis and are not available to most practitioners. Viral load monitoring using polymerase chain reaction assays of EBV DNA virus genome load has been used primarily to identify solid organ and bone marrow recipients at high risk for posttransplant lymphoproliferative disease (PTLD) but can be used to confirm acute IM. Hepatic transaminase levels are commonly elevated in IM, although usually only mildly, occurring in approximately 50% of cases.[28] The importance of these transient elevations in determining return to play remains unclear.

Thus, IM remains a clinical diagnosis based on the combination of a carefully performed history and physical examination with selected laboratory testing. With regard to splenic enlargement, however, an evidence-based assessment demonstrates that there is little support to substantiate the clinical reliability of physical examination to either detect or exclude splenomegaly in athletes. In addition, there is some suggestion

from the literature that an overzealous abdominal examination may in rare instances precipitate a splenic injury. As such, although the bedside assessment of splenomegaly has some clinical utility, bedside assessment alone cannot be used to either diagnose IM or guide return-to-play decisions.[14,15,18]

COMPLICATIONS

Most patients who contract IM have an uneventful clinical course with an unremarkable recovery. Some athletes may take as long as 3 months to return to their preillness levels of activity.[29] Severe complications have been reported in up to 5% of patients.[30] A wide variety of complications can occur, involving virtually all organ systems. The more serious complications include splenic rupture, aplastic anemia, Guillain-Barré syndrome, meningitis, encephalitis, neuritis, lymphoma, hemolytic uremic syndrome, and disseminated intravascular coagulation.[15,31–33] Although there has been conjecture that chronic fatigue syndrome may be associated with chronic EBV infection, this is not well-supported in the literature and further discussion is beyond the scope of this article.[15] Two of the more common complications that confront the sports medicine clinician include severe pharyngitis with tonsillar enlargement and splenomegaly complicated by potential rupture.

Pharyngitis

IM can lead to severe pharyngitis with tonsillar enlargement that can potentially compromise the airway. Patients may present with shortness of breath that is more severe when supine. Some authorities have advocated prompt treatment with corticosteroid therapy, initiated in doses ranging from 40 to 80 mg of prednisone or its equivalent (see Management).[25] Prednisone may also be used to decrease tonsillar size in patients who present with difficulty swallowing and dehydration. Advanced airway management is warranted in any patient who is severely compromised or who does not promptly respond to corticosteroids. Group A beta-hemolytic *streptococcus* pharyngitis is noted in up to 30% of individuals with IM.[5] Because of this, testing for *streptococcus* is frequently indicated in most cases of IM.

Splenic Rupture

The potential for splenic rupture during IM is a concern for all individuals and especially those participating in contact and collision sports. Splenomegaly in IM is nearly universal (see Imaging of the Spleen), but splenic rupture is rare. The true incidence will never be determined as a result of the numerous subclinical cases, but it is cited to occur in 0.1% to 0.2% in most reviews.[5,23,34–36] Rupture of the spleen can occur spontaneously or be caused by trauma resulting from the altered splenic architecture.[37,38] It is thought to occur as the result of a sudden increase in the portal venous pressure that may be related to a simple Valsalva maneuver or sudden compression from external trauma.[34,39] It appears that the spleen is most vulnerable to rupture in the first 3–4 weeks of the illness because documented cases of splenic rupture beyond this timeframe are extremely rare.[37] In a review of 55 cases of splenic rupture, almost all occurred in the first 3 weeks of illness, and in half of these, the ruptures were not traumatic.[5]

Imaging of the Spleen

Given the concern for splenic rupture, along with the poor ability of physical examination to detect splenic enlargement, various diagnostic imaging studies have been advocated for the evaluation of the athlete with IM. Many advocate that the spleen size should be "normal" prior to return to play. Various radiographic assessments including plain radiographs, computerized tomography (CT), magnetic resonance imaging (MRI), and ultrasonography have all been used to evaluate spleen size. A splenic shadow visualized below the margin of the ribs on plain radiograph may signify the presence of an enlarged spleen. More detail-oriented imaging techniques (ultrasound and CT) are able to better quantify splenic dimensions. MRI has not been studied as a means of assessing splenic size. Although CT can offer superior organ detail and exact splenic measurements, radiation and cost limit its utility. Current spiral CT scanners produce high-resolution images with fast scan times but result in increased radiation exposure.[40–42] As a result, ultrasound is the preferred modality for evaluation of splenomegaly. Ultrasound is also adequate in identifying splenic rupture or capsular hematoma in suspected cases, but the superior imaging capability of CT scanning or MRI could be justified in these situations.

Ultrasonography is readily available, is inexpensive, and offers no radiation risk. Splenic size can be determined with measurements in the breadth, thickness, and longitudinal dimensions. These 3 measurements can be multiplied and divided by 27 to determine the splenic volumetric index as an approximation of splenic volume.[43] Published normative data on splenic index are limited. More recently, linear spleen measurements via ultrasound have been shown to have a high correlation with CT volume assessments.[44] Literature standards define the upper limits of normal for longitudinal diameter as 12–14 cm (4.72–5.51 inches) in adults.[45–47] However, the use of these normative values can be problematic because the populations on which these values were defined were often heterogeneous and the individual sample sizes within specific age groups were small.[48–54]

In a study of athletes, spleen size was assessed by ultrasound in tall, healthy athletes; in this particular study, spleen length was greater than 12 cm in 31.7% of the men and 12.8% of the women. These athletes had a mean body height of 74.3 inches (188.72 cm; ± 3.7 inches, or 9.4 cm) and 69.3 inches (176.02 cm; ± 3.7 inches, or 9.4 cm), respectively. These researchers found that spleen length correlated with height, and a nomogram was suggested as a tool for determining "normal" spleen size in this population.[55] There are limitations in this study in that it studied only tall, healthy athletes; the overall sample size was small; and subjects did not have IM.

Recent data describing normative spleen size in a population of 631 collegiate athletes demonstrated considerable variability of normal splenic size among the athletes (average 10.65 cm; range 5.59 cm–17.06 cm spleen length).[56] In this study, height and weight of the athlete correlated moderately well with splenic size, and splenic size varied significantly between males and females. Males had larger spleens than females. These differences persisted when controlling for height and weight. Perhaps most enlightening was the fact that

just more than 7% of the athletes had a baseline splenic length of >13 cm.[56] Because of the wide variability of normal spleen size in this population, the use of existing parameters for defining "normal" spleen size must be questioned.

Consequently, although sonographic imaging is an objective and reliable method for assessing spleen size, obtaining a single ultrasonographic measurement in athletes with IM needs to be interpreted with caution. For example, an athlete with a spleen length of 11 cm would be considered normal, but this individual might have a baseline of 7 cm, indicating relative splenomegaly. Conversely, an athlete with a baseline splenic length of 14 cm could erroneously be labeled as having splenomegaly.[55,56] One-time imaging of the spleen for assessment of splenomegaly at the time of illness is not recommended because of the wide variability encountered in normal values. Without knowledge of baseline splenic values unique to the individual, a single ultrasonographic splenic size determination cannot reliably be used to determine splenomegaly.

MANAGEMENT

Standard treatment of EBV infections include supportive measures aimed at providing symptomatic relief and close attention to the potential development of complications. The time course of recovery is variable—anywhere from weeks to months—with younger patients often recovering more quickly than older ones. Maintenance of fluids and hydration, acetaminophen for comfort, and rest are important. Symptomatic treatment of sore throat can be accomplished with salt water gargles, anesthetic throat lozenges or sprays, or gargling with a 2% lidocaine solution (the latter can also be mixed with an antacid and diphenhydramine). Transmissibility of EBV is low, so patient isolation is not necessary. Infectious disease prevention practices should be adhered to (frequent hand washing, avoidance of shared utensils and water bottles, etc). Aspirin should be avoided in adolescents because of the risk of bleeding and thrombocytopenia. Individuals with IM should be advised to avoid excessive alcohol and acetaminophen and any other potential liver toxins.

Specific therapy of acute EBV infections with intravenous and oral formulations of acyclovir show that short-term suppression of viral shedding can be demonstrated, but significant clinical benefit has been lacking.[57,58] A meta-analysis of 5 randomized controlled trials of acyclovir in the treatment of acute IM failed to show a clinical benefit compared to placebo.[59]

Corticosteroids may be clinically indicated in cases with airway compromise, severe dysphagia, massive spleen enlargement, myocarditis, or hemolytic anemia.[8,60,61] A Cochrane Review evaluating the use of corticosteroids for symptom control in IM concluded that, although symptoms were decreased for the first 12 hours, these benefits were lost at 2–4 days. They concluded that there was no clear evidence on the effectiveness of steroids and, given the potential for adverse effects from the use of steroids, they should be avoided.[62] Because EBV infection is associated with oncogenic complications (nasopharyngeal carcinoma, Burkitt's lymphoma), corticosteroids should not be used in patients who have uncomplicated IM. Antibiotics should be used if there is a concomitant streptococcal pharyngitis, but ampicillin or amoxicillin should be avoided because of the risk of rash.

Because exertion of any kind can cause splenic rupture, it is advisable to avoid any form of exertion, including sports activities. Because splenic rupture can occur with a Valsalva maneuver, a stool softener may be prescribed to diminish the strain with bowel movements. Such precautions are advised for the first 3 weeks of illness, given the increased risk of splenic rupture during this time.

Splenic injury can be treated either with splenectomy[63] or by observation in selected situations.[64] In the past, most individuals with splenic rupture, whether or not associated with IM or associated with trauma or sport, have been treated with splenectomy. More recently, nonoperative treatment with observation has been used for those with stable splenic injury, although this treatment may delay return to play for up to 3 months.[64] The risks of asplenia must be weighed against early return to play and remains an individualized decision. For individuals with splenic injuries who require splenectomy, preemptive vaccinations with pneumococcal polysaccharide vaccine, meningococcal vaccine, and *Hemophilus influenza* should be provided and are ideally given prior to spleen removal.

SPECIAL POPULATIONS

Special populations raise unique considerations. The adult form of mononucleosis caused by EBV is different from the disease in children and adolescents. In most adults there is no pharyngitis or lymphadenopathy, fever is much more prolonged, abnormal liver function is frequent, and lymphocytosis and the presence of atypical lymphocytes are less common. The individual who is immunocompromised may demonstrate variations in the clinical, hematologic, and serologic response to primary EBV. In patients with HIV, patients with hereditary immunodeficiencies, or patients receiving potent immunosuppressants, EBV is an important pathogen that can cause lymphoma and other lymphoproliferative disorders.[65,66] Severe or fatal infectious mononucleosis may develop in some patients with immunodeficiency such as X-linked lymphoproliferative disease, which is characterized by an extreme susceptibility to EBV infection.[67] The patient who is immunocompromised may be a candidate for a treatment regimen that goes beyond symptomatic treatments and may include antiviral agents, immune modulators, and chemotherapeutic drugs.[68] Patients that are solid organ transplant recipients are also a special population to consider for IM infection. Transplant recipients, because of their immune suppressed state, are at an increased incidence for PTLD.

RETURN-TO-PLAY GUIDELINES

Return-to-play (RTP) decisions have traditionally focused on clinical resolution of symptoms and the absence of splenomegaly. Returning an athlete too quickly to sports participation risks splenic injury and the possibility of prolonging the time necessary for full recovery. Discerning clinical resolution additionally raises the issue of the requirement for advanced imaging and laboratory confirmation. To date, however, there are no well-designed large clinical trials to assist sports medicine providers in these difficult decisions.

The current consensus from the literature is that light, noncontact activities may commence 3 weeks from symptom onset.[15,69,70] Research in the military has demonstrated no

significant difference in aerobic capacity and no detrimental effects in those with IM allowed to participate in light exercise ad libitum as soon as they become afebrile, compared with those restricted from activity for 2 weeks.[71] The resumption of light activity assumes that the activity will avoid any chest or abdominal trauma and will not involve significant exertion or Valsalva activities and that the athlete is asymptomatic. Progression of noncontact activity should then be gradually individualized as judged by the athlete's clinical progress.

Returning to contact activity is more controversial. The majority of splenic ruptures occur in the first 3 weeks of the illness; however, cases have been described up to 7 weeks. The risk of rupture may be increased in contact sports and in those activities associated with an increased abdominal pressure or Valsalva such as weightlifting or rowing. More caution is recommended in these situations.

Determining the presence or absence of splenomegaly is controversial because there are no large natural history studies of splenomegaly or ruptures in athletes. As stated previously, the relationship between splenomegaly and the risk of splenic rupture remains controversial and unclear. In addition, the spleen, even if not enlarged, may be more susceptible to injury as a result of the infectious disease process of EBV itself. Physical examination is unreliable, and advanced imaging is challenged by the lack of normative data. Given the variable nature of spleen size demonstrated in recent studies, the use of imaging to make the RTP decision should be used as individually indicated, and serial studies should be considered. The assumption is that the spleen will generally enlarge with the onset of IM and that the risk for rupture is greatest as the spleen is enlarging. Determining when the spleen has stopped enlarging, and is decreasing back to normal size, is likely the time that would be ideally suited for allowing return to play. Unfortunately, without baseline and serial examinations, this determination cannot be accurately made.

The athlete (and his or her family, if indicated) should be educated about the statistical probability and the known time frame for the occurrence of splenic ruptures. Given that the overwhelmingly large majority of splenic ruptures occur in the first 3 weeks, it seems reasonable to prohibit activity during this period. After that time, the athlete should be allowed to return to activity if they are afebrile, well-hydrated, and asymptomatic and have achieved an appropriate level of physical fitness. Athletes and their families should be informed that the risk of splenic rupture is never zero, with or without IM. Additional factors to consider include the risk of contact and/or collision and the age of the athlete.

Physical examination is often unreliable in an athlete with a well-developed abdominal musculature, and advanced imaging is challenged by the lack of an adequate baseline that may overread splenic enlargement in a normal individual.

In athletes in whom a laboratory abnormality was detected during the initial assessment, there are no clinical guidelines assisting in return to play that are specific to IM. Clinical judgment, and treatment and resolution of the individual complication, dictate resumption of activity.[15,18,23,34,46] Based on the limited data discussed previously, imaging is often unreliable in the absence of baseline measurements, which are rarely available. Therefore, most RTP decisions must be based on the clinical and functional measures of each individual case and usually will not involve imaging.

FUTURE AREAS FOR CLINICAL RESEARCH

IM is a common illness that affects predominantly young athletes and is associated with a variety of clinical presentations. Invariably, athletes are incapacitated—even if for only a short time—with fever, pharyngitis, and lymphadenopathy being predominant features early on. Additional research endeavors studying the role of oral steroids and the resolution of IM-associated symptoms and splenomegaly are needed. In addition, exploring other possible treatment options such as antiviral therapies or potential vaccines to decrease the length of clinical symptomatology or prevent disease are research areas to be evaluated. Although IM and associated splenic enlargement are commonly encountered among athletic participants, splenic rupture is fortunately extremely rare. As such, clinical studies evaluating the risk of splenic rupture with varying degrees of splenomegaly and a variable timetable of return to activity (even if deemed ethical) are not feasible. This practicality leaves clinicians without objective data on which to base RTP decisions.

One potentially useful area of study would be to define the natural history of changes in splenic dimensions in a cohort of athletes diagnosed with IM. This particular study would ideally include a baseline objective measure of the normal spleen and subsequent follow-up examinations (in individuals prospectively diagnosed with IM) to illustrate the degree of splenic enlargement and time course to resolution. These data, although not generating a relative risk of splenic rupture, may provide useful information regarding the timing of peak splenic enlargement and resolution of splenomegaly. Expansion of this potential research to include documentation of patient symptoms, clinical examination, and laboratory findings and how they correlate with objective measurements of the spleen may also be useful.

Another unanswered question is what role exercise plays on the natural history of IM resolution. Whether an athlete that returns prematurely subsequently has a prolonged course of recovery, prolonged fatigue, and/or a decrease in performance measures is unclear.

Finally, there are many athletes that develop IM without seeking medical attention and often participate in sport without any apparent complication or negative consequence. Why some athletes are less symptomatic than others also remains unclear. Understanding the natural history of this sometimes elusive disease will help determine if we are keeping athletes out of activity unnecessarily.

Strength-of-Recommendation (SOR) Grades

Strength of Recommendation	Basis for Recommendation
A	Consistent, good quality patient oriented evidence
B	Inconsistent or limited-quality patient-oriented evidence
C	Consensus, disease oriented evidence, usual practice, expert opinion, or case series for studies of diagnosis, treatment, prevention or screening

EVIDENCE-BASED CONCLUSIONS/ RECOMMENDATIONS

1. Is any treatment other than supportive care warranted for the treatment of IM in athletes?

Supportive care is the treatment for uncomplicated IM in athletes. The role of other treatments, including antivirals and oral corticosteroids, in reducing time until return to play remains unclear. Corticosteroids are indicated when IM is complicated by impending airway obstruction, hemolytic anemia, severe thrombocytopenia, or myocarditis. SOR B

2. What is the athlete's risk for splenic rupture?

There are no published studies that accurately assess this risk. The risk has been extrapolated from general population studies to be 0.1% to 0.5%. SOR C

3. Is physical examination reliable in determining the presence or absence of splenomegaly in athletes?

Physical examination alone cannot be used to diagnose IM or guide RTP decisions. Physical examination techniques have poor sensitivity and specificity in identifying splenomegaly. Inter-rater reliability for physical examination is also poor. SOR A

4. Is diagnostic imaging reliable in determining the presence or absence of splenomegaly in athletes?

One-time imaging of the spleen for assessment of splenomegaly at the time of illness is not recommended because of the wide variability encountered in normal values. More normative data incorporating different genders and height may allow for increased utility for diagnostic imaging in the future. Imaging should be considered if splenic injury is suspected or in situations where the athlete with acute IM is clinically asymptomatic early on in recovery and early return to activity is contemplated. In the latter case, serial imaging should be considered to take into account the wide variability in splenic size. SOR B

5. What is the best imaging test to determine splenomegaly in athletes?

Imaging is generally not necessary. If imaging is obtained to evaluate splenic size, ultrasound is preferable in that it is easy, noninvasive, and reliable and does not expose the patient to radiation. If imaging is obtained to exclude splenic injury or rupture, CT is recommended given the increased amount of information provided. SOR B

6. Is the assessment of splenomegaly important in the management of the athlete with IM?

In most cases, it is not. Most athletes are not feeling well enough to play until the 3rd or 4th week of their illness, and it is known that most athletes early in their illness have splenomegaly. In addition, there are no data that correlates spleen size to splenic rupture. It has been theorized that the spleen is at highest risk when it is enlarging, and preliminary data suggest that this occurs at the 3–4-week mark. However, given that physical examination is unreliable and imaging is only reliable when a baseline is available or serial examinations are performed, the assessment of splenomegaly is likely not important. SOR B

7. When is it safe to return to light activity?

In most cases, athletes will not feel well enough to participate in activity for several weeks. In addition, although the risk for splenic rupture is extremely low, given that most of these occur within the first 3 weeks of illness, independent of spleen size, it is generally felt that it is safe to resume light activity 3 weeks from the onset of symptoms, as long as the athlete is afebrile, has a good energy level, and does not have any significant associated abnormalities. The risk of splenic rupture likely diminishes as more time progresses; therefore, delaying return to play should be considered if clinical features, lack of athlete readiness, and/or associated complications are present. Treatment must therefore be individualized to account for all of these issues. SOR C

8. When is it safe to return to contact activity?

The appropriate time for safe return to contact play is unclear, although, given the risk for splenic rupture, a time frame of at least 3 weeks commonly is recommended. Return can occur only after the athlete has no remaining clinical symptoms, is afebrile, and has a normal energy level. The risk for splenic rupture likely decreases as more time passes, allowing for individualized RTP decisions depending on the athlete, sport, and other factors. SOR C

9. What effect does exercise have on the natural history of IM resolution?

It is unclear what role exercise has on the natural history of IM disease. It would appear that premature return to heavy exertion might prolong the duration of symptoms, most notably fatigue, and also be associated with a decrease in performance. SOR C

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