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Management of Infectious Mononucleosis in Paediatric Athletes

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Abstract

Background. Infectious mononucleosis (IM), most commonly caused by the Epstein–Barr virus (EBV), affects primarily adolescents and young adults between 15 and 24 years of age, populations with high rates of sports participation. Although IM is usually self-limited, its symptoms most often include fever, pharyngitis, posterior cervical lymphadenopathy, fatigue, and malaise.

Aim of study. This review aims to summarize the current evidence on the diagnosis, clinical management, and return to play (RTP) recommendations for athletes and paediatric athletes with IM.

Materials and Methods. The review was based on a search of articles published in the PubMed database. Search terms included: infectious mononucleosis, Epstein-Barr virus, splenic rupture, splenomegaly, return to play and Monospot test.

Current state of knowledge. Prolonged fatigue and the risk of splenic rupture may significantly disrupt training and competition. The most relevant sports-related complication is splenic rupture, which is associated with splenomegaly and occurs most often early in the disease course.

Conclusions. RTP decisions should be individualized based on symptom resolution, clinical findings, the type of sport (contact vs non-contact), and shared decision-making. The general consensus is to avoid contact sports and strenuous training for at least 3–4 weeks. Ultrasound may be helpful in selected cases, but its routine use is limited by the wide variability in baseline spleen size and the absence of validated thresholds for safe RTP.

Keywords: infectious mononucleosis, paediatric athletes, sports medicine, return to play, Epstein–Barr virus

1. Introduction

Infectious mononucleosis is viral infection caused predominantly by the Epstein–Barr virus (EBV) [1]. It most commonly affects children, adolescents, and young adults, with the highest incidence observed between 15 and 24 years of age [2]. Transmission occurs primarily through close personal contact, particularly via saliva, which contributes to its frequent occurrence in athletic environments such as training rooms, locker rooms, and team settings [3].

The clinical course of infectious mononucleosis is typically self-limited, and many cases remain asymptomatic or mildly symptomatic [4]. When present, symptoms most often include fever, pharyngitis, posterior cervical lymphadenopathy, fatigue, and malaise [5]. In athletic populations, prolonged fatigue and reduced exercise tolerance may significantly impair performance and training participation.

The most serious and potentially life-threatening complication of infectious mononucleosis is splenic rupture, which is associated with transient splenomegaly during the acute phase of the illness [6]. Although splenic rupture is rare, it carries significant morbidity and has been reported both in contact and non-contact sports. Consequently, decisions regarding physical activity restriction and safe return to play (RTP) represent a critical challenge for clinicians managing affected athletes [7]. This review aims to summarize and critically evaluate current evidence regarding the diagnosis, management, and return-to-play considerations for athletes and paediatric athletes with infectious mononucleosis.

2. Epidemiology and Clinical Presentation in Athletes

IM is a viral infection caused up to 90% by EBV, a member of Herpesviridae family [2]. EBV causes a benign lymphoproliferative syndrome but has also been linked to Burkitt lymphoma, Hodgkin lymphoma, and nasopharyngeal carcinoma. It is estimated that up to 90% of adults worldwide become EBV-seropositive by the age of 30 [8].

EBV is transmitted primarily through saliva, which is why IM is often referred to as the “kissing disease” [4]. Many individuals are exposed during childhood and infection at that age is frequently subclinical [9]. The incubation period is typically 4–8 weeks before symptom onset. IM occurs worldwide without a clear seasonal predilection. It is classically characterized by the triad of fever, pharyngitis, and posterior cervical lymphadenopathy [10]. Other symptoms may include fatigue, headache, decreased appetite, abdominal pain, hepatomegaly, splenomegaly, nausea, vomiting, periorbital and eyelid oedema and rash [10].

Pharyngitis is often the most common prominent symptom, and patients may describe it as unusually severe. On examination, tonsillar enlargement with white, gray, or green exudate may

be present. This presentation can be mistaken for streptococcal pharyngitis [11]. Lymphadenopathy most commonly involves posterior cervical nodes, whereas axillary and inguinal lymphadenopathy are less frequent.

Fatigue associated with IM can range from mild to severe and it may persist for several weeks after resolution of acute symptoms, in some cases it can last up to 6 months [9]. A maculopapular, petechial, or urticarial rash is particularly common after administration of aminopenicillins (e.g., amoxicillin) for presumed group A streptococcal pharyngitis and may serve as a clinical clue to IM [7].

3. Diagnostic Approaches

When IM is suspected clinically, laboratory testing can support the diagnosis. A complete blood count often shows leukocytosis with lymphocytosis; classically, lymphocytes account for $\geq 50\%$ of the differential, and atypical lymphocytes may be present. Mild elevations in hepatic transaminases are also common and can be considered in the diagnostic process [9].

The most commonly used initial test is detection of EBV-specific heterophile antibodies (Monospot test). Reported sensitivity ranges from approximately 70% to 90% in is lower in early disease and in young children [12]. A key limitation is timing: heterophile antibodies typically peak between 2 and 6 weeks after symptom onset, so testing may be negative early in the illness [12]. In children under 4 years of age, the test performs poorly; approximately 40% do not develop heterophile antibodies after primary EBV infection [13].

EBV viral capsid antigen (VCA) antibody testing is more sensitive and specific than heterophile testing but may be less rapidly available. In patients with a mononucleosis-like illness and a negative Monospot test, VCA-IgM and VCA-IgG testing is recommended. VCA-IgM suggests recent infection, VCA-IgG indicates past infection or developing immunity, and the presence of EBV nuclear antigen (EBNA) IgG during acute illness argues against primary EBV infection [3].

4. Splenomegaly

Splenomegaly is present in approximately 50% of patients with IM during the acute phase of infection. The spleen may increase its size up to three to four times [14]. Clinical assessment of splenomegaly is often unreliable and may vary depending on the examination method used (palpation versus percussion), obesity and from one observer to other [15].

An enlarged spleen is particularly vulnerable to rupture, and resolution of splenomegaly is often used in guiding a return to normal athletic activity [15]. Proposed mechanisms for splenic

rupture include weakening of splenic architecture, increased tissue fragility, and reduced protection by the rib cage as the spleen enlarges [16].

The role of ultrasonography RTP decision-making will be discussed later. Interestingly in a retrospective study, Durtschi et al. assessed seven university-aged athletes with IM, using serial measurements of aspartate aminotransferase (AST), alanine transaminase (ALT) and ultrasound-measured spleen sizes were performed. Levels of AST and ALT were significantly correlated with spleen size. For each 10-unit increase in AST and ALT values, spleen size increased by 0.1 cm ($p = 0.007$) and 0.09 cm ($p = 0.008$), respectively, suggesting a potential adjunctive role of liver enzymes in RTP decision-making [17].

5. Splenic infarction

Splenic infarction is the most important but rare complication, estimated at approximately 0.1%- 0.2% of all cases [18]. Most incidents of rupture are associated with contact sports with collisions and blunt trauma, however rupture has also been reported following a Valsalva manoeuvre or occurring idiopathically [19]. Most reported splenic ruptures occur early in the disease course, particularly within the first three weeks after symptom onset [20]. However, cases of rupture have been reported as late as seven weeks after symptom onset [15].

Splenic infarction may present with a broad clinical spectrum, ranging from asymptomatic cases to severe abdominal pain, most commonly localized to the left upper quadrant. Associated symptoms may include nausea, vomiting, abdominal distension, and, less commonly, intestinal obstruction [21]. Kehr's sign (referred left shoulder pain due to diaphragmatic irritation by free intraperitoneal blood) should raise concern for splenic rupture and possible intra-abdominal bleeding [22]. After rupture, imaging studies such as ultrasonography/ computed tomography (CT) may show splenomegaly, free intraperitoneal fluid, and the presence of a subcapsular hematoma [23].

6. Role of ultrasound in RTP decisions

There is ongoing debate about the role of ultrasound in measuring spleen size to individualize RTP. Important limitations include wide inter-individual variability in baseline spleen size, especially among athletes, differences in body habitus, and the absence of validated cutoffs defining a safe spleen size for sport participation. As a result, a single ultrasound measurement may not reliably predict rupture risk [9].

Hosey et al. reported that 20 patients with IM underwent serial abdominal ultrasound. Mean peak splenic enlargement was approximately 33.6% (SD 19.9%) above baseline. Peak

enlargement occurred most commonly within two weeks of symptom onset, and splenomegaly resolved within 4–6 weeks in the majority of cases. These findings support the concept that rupture risk is greatest early and decreases as symptoms improve and the spleen returns toward baseline size [20].

A systematic literature review of 171 articles identified 186 cases of splenic rupture and 29 cases of splenic infarction. Both conditions predominantly occurred in males, (60% and 70% respectively). Approximately 80% (n = 139) of cases occurred within three weeks of the onset, and the mortality rate of splenic rupture was 4.8% (n = 9). Interestingly, in cases of splenic infarction, an underlying haematological condition was observed in 21% (n = 6) of cases. The treatment of splenic infarction was always conservative with no fatal outcomes reported[24].

As IM commonly occurs in young, otherwise healthy individuals, splenomegaly and its complications represent a particular challenge in athletes. While strenuous physical activity and contact sports should be avoided during periods of increased splenic vulnerability, this restriction may be especially difficult for competitive and professional athletes, for whom prolonged absence from training may negatively affect performance [16].

7. Management of IM and RTP Recommendation

Because IM is usually self-limited, treatment is primarily supportive. Symptom management includes acetaminophen (paracetamol) or non-steroidal anti-inflammatory drugs (NSAIDs) for fever and sore throat [25]. Aspirin should be avoided in children due to the risk of Reye syndrome[3]. All patients should be advised to rest and temporarily reduce physical activity, particularly during the acute symptomatic phase. Adequate hydration, rest, and gradual resumption of daily activities are generally recommended. Antibiotics are not indicated unless bacterial co-infection is confirmed [3].

The general consensus states that patients should avoid contact sports and strenuous training at least for 3–4 weeks, and in some cases up to 6–8 weeks, particularly when splenomegaly is suspected or confirmed [16]. However, RTP recommendations vary between guidelines due to limited high-quality prospective evidence.

From a sports medicine perspective, RTP should be based mainly on clinical recovery, resolution of the symptoms, the normalization of splenic size as monitored by serial ultrasonography (if performed) and the estimated risk of splenic rupture according to the time elapsed since illness onset [15].

Before initiating light training, athletes should be afebrile, feel clearly better, tolerate normal daily activities. At any stage of the return-to-activity process, the onset of abdominal pain

should raise concern. RTP should then progress stepwise from light, non-contact activity to full participation, depending on symptoms and the contact risk of the sport [9].

8. Conclusion

IM is common in adolescents and young adults and is highly relevant in sports medicine due to prolonged fatigue and the risk of splenic rupture. Diagnosis should be based on clinical presentation supported by laboratory testing; heterophile antibody testing has limited sensitivity early in the disease course and performs poorly in young children, making EBV-specific serology particularly important in paediatric athletes. Management is mainly supportive, with symptom-guided activity restriction.

RTP decisions remain challenging. Splenic rupture is a rare but potentially fatal, and splenomegaly is variable and difficult to quantify reliably. The highest risk of rupture appears to occur within the first three weeks after symptom onset, supporting strict avoidance of contact/collision sports and strenuous training during early illness. Ultrasound can document splenomegaly and its resolution, but its utility is limited by baseline variability and the lack of validated RTP thresholds. Therefore, imaging should be used selectively rather than routinely. A cautious, individualized, stepwise RTP progression incorporating symptom resolution, sport-specific risk, and shared decision-making seems to represent the most defensible approach for both adult and paediatric athletes.

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