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# **FIMS Position Statement 2009**

# Acute and chronic hepatitis and sports

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#### Abstract

Diseases of the liver may have a significant impact on exercise capacity and physicians' recommendations for the athlete. Acute viral hepatitis is a systemic infection affecting the liver predominantly. Knowledge about the impaired hepatic function as well as hazards for other athletes especially in contact sports is mandatory for professional support. Differential diagnosis includes metabolic diseases, hereditary and autoimmune diseases as well as toxic and drug-induced alterations. The clinical spectrum of chronic hepatitis ranges from asymptomatic illness at one end to fatal hepatic failure. A close workup is recommended and the expertise of a liver centre often is essential. New drug developments allow the therapy of viral hepatitis with only minor or even without deterioration of athlete's performance. According to the severity of liver damage recommendations regarding physical exercise should be made. **Keywords**: hepatitis, exercise, portal hemodynamics, rehabilitation

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# Acute hepatitis

#### Definition

Acute hepatitis is usually caused by one of five hepatitis viruses (HAV, HBV, HCV, HDV and HEV) with clinically similar illnesses ranging from asymptomatic and inapparent to fulminant and fatal infections. HBV, HCV and HDV infections may persist and progress to chronic liver disease, cirrhosis and even International SportMed Journal, Vol. 10 No.2, 2009, pp. 61-65, <u>http://www.ismj.com</u>

hepatocellular carcinoma. Other viral or bacterial pathogens (Epstein-Barr-virus, Leptospira, etc.) as well as toxic substances, drugs or other underlying diseases (i.e. Wilson disease, autoimmune hepatitis etc.) may present as acute hepatitis (see Table 1). Irrespective of the cause of the hepatitis, the recommendations for physical exercise are identical.

Table 1: Causes and diagnosis of acute and chronic liver disease

| Toxic hepatitis                | history (drugs, alcohol)                                     |
|--------------------------------|--|
|                                |  |
| Viral hepatitis                | HAV: anti-HAV  |
|                                | HBV: HBsAg, anti-HBc   |
|                                | HCV: anti-HCV  |
|                                | HDV: anti-HDAg (HBsAg+)                                      |
|                                | HEV: anti-HEV  |
|                                |  |
| Autoimmune hepatitis           | autoantibodies (ANA, SMA, LKM-1, SLA)                        |
|                                |  |
| Hereditary diseases            | family history   |
|                                | hemochromatosis: serum ferritin, serum iron,                 |
|                                | transferring saturation, genetic testing, liver              |
|                                | histology  |
|                                | Wilson disease: ceruloplasmin, urine copper, eye             |
|                                | examination, liver biopsy                                    |
|                                | $\alpha_1$ -Antitrypsin deficiency: $\alpha_1$ -Antitrypsin; |
|                                | phenotype testing  |
|                                |  |
| Primary biliary cirrhosis      | autoantibodies (AMA), liver histology                        |
|                                |  |
| Primary sclerosing cholangitis | cholangiography  |
|                                |  |
| Others                         | Elevated central venous pressure (right heart                |
|                                | failure, pericardial effusion)                               |
|                                | Diabetes mellitus and NASH (blood glucose,                   |
|                                | cholesterol, oral glucose tolerance test)                    |
|                                | Hyper- or hypothyreoidism (TSH)                              |
|                                | Celiac disease (endomysial IgA antibody, tissue              |
|                                | transglutaminase IgA antibody)                               |
|                                | Sarkoidosis (angiotensin converting enzyme,                  |
|                                | liver biopsy)  |

# Diagnosis of acute hepatitis

A detailed patient history that includes sexual behaviour, family and travel history, recent dining experiences, and previous vaccinations received may suggest the correct diagnosis and cause of the disease. Specific laboratory tests will identify the infecting agent. In the case of metabolic liver disease, further diagnostic procedures, including percutaneous needle biopsy of the liver or functional testing may be required.

# Physical activity for patients with acute

# hepatitis

Traditionally, individuals with acute viral hepatitis were advised to avoid vigorous physical exertion. These recommendations were based upon case reports of prolonged illnesses and fatal hepatitis (most likely Hepatitis A)<sup>1,2</sup>. Experience after strenuous physical activity with military personnel in countries with a high prevalence of acute, viral

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hepatitis has challenged these traditional views. Recreational physical activity during the acute phase and subsequent strenuous activity once the bilirubin values have decreased to 1.5mg/dl show no increase in complications, recurrence rates, or duration of convalescence<sup>3</sup>. There is increasing evidence<sup>4</sup> that strict bed rest is no longer mandatory for patients with acute hepatitis.

#### The role of rehabilitation

Moderate exercise (70% of  $VO_{2max}$ ), twicedaily for patients with acute viral hepatitis does not affect the clinical course, biochemical test results, or liver cell histology <sup>5.</sup> Other exercise programmes also have no acute or chronic adverse effects, but rather resulted in a marked shortening of the rehabilitation period <sup>6-8</sup>.

# **Chronic hepatitis**

#### Definition

Chronic hepatitis is defined as persistent liver inflammation or necrosis for more than six months. Mild forms of chronic hepatitis may be slowly progressive if at all. More severe forms of the disease may result in active inflammation and fibrosis leading to cell destruction and liver cirrhosis.

#### **Diagnosis of chronic hepatitis**

The spectrum of clinical presentations of chronic hepatitis ranges from asymptomatic liver disease to end-stage, fatal hepatic failure. The majority of patients tend to have constitutional symptoms with persistent fatigue being most commonly reported. Repeated liver enzyme elevation is diagnostic for chronic hepatitis. In viral hepatitis, serologic testing, viral load and genotype are key to the identification of the causative agent and the initiation of the antiviral therapy. Referral to a liver centre is recommended. The stage of the disease and signs of elevated portal pressure should be evaluated.

# Physical activity during chronic hepatitis

Patients with only a mild disease and liver fibrosis tolerate routine exercise very well<sup>9</sup>. In patients with more advanced disease, there are no controlled clinical trials upon which to base definitive exercise recommendations. Training programmes of moderate intensity have been well tolerated by these patients,

# and have led to improvements in oxygen consumption and work capacity <sup>10</sup>. **Physical activity in patients with liver**

#### cirrhosis

Once complications of liver cirrhosis have developed, recommendations for physical activity should be given with caution. Gluconeogenesis may be reduced by 80% resulting in reduced glycogen storage and inability to compensate for the increased metabolic demands of exercise. Furthermore. many patients with cirrhosis are malnourished accentuating the risks of increased energy expenditure. Cardiovascular dysfunction leads to an elevation in resting cardiac output with a limited potential to respond to exercise and improve cardiovascular status. When the portal pressure is elevated, moderate exercise (30% of VO<sub>2max</sub>) may lead to a significant increase in the hepatovenous pressure gradient, increasing the risk of variceal hemorrhage. If ascites is present, moderate physical activity (30 minutes at 3 METs) may compromise renal function by stimulating the neurohumoral systems <sup>11</sup>. Furthermore, physical activity may worsen hepatic encephalopathy by reducing liver function and urea synthesis.

# The role of rehabilitation

Guided exercise programmes are important for patients with advanced liver disease. Close monitoring and regular follow-up should ensure maintenance of hepatic function and detect negative training effects. Based on long-term observations, preservation of muscle mass and minimization of bone loss are the two most important goals to be achieved through physical activity.

# Participation in sports

#### Returning to training and competition after

#### acute viral hepatitis

Available data suggest that moderate physical activity is safe and well tolerated in clinically healthy individuals with acute hepatitis. However, there is little data concerning the training of patients at competitive levels. It is therefore appropriate for patients to refrain from participation in competitive level sports and intensive training until the liver function tests are normal. In addition, when acute hepato- or splenomegally is present on physical examination or by ultrasound, contact and collision sports should not be permitted.

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Athletes may continue training at a lower level during the acute phase if the patient feels well enough and his/her clinical condition permits. Checkups should assess the eventual development of chronic hepatitis in patients with HBV, HCV or HDV infection. These patients should be referred to a liver centre.

# Transmission of viral hepatitis during

#### sports

The American Medical Society for Sports Medicine (AMSSM) and the American Orthopaedic Society for Sports Medicine have both published position statements which state that acute viral hepatitis should be viewed like any other viral infection and only general recommendations are given. The infected athlete should have an individualised recommendation based on the characteristics of the disease and the type of sport in which he/she wishes to engage.

Direct transmission of HAV or HEV through participation in sports has not been described. An outbreak in one team was related to poor hygienic practices in the handling of food <sup>12</sup>. The risk of transmission of blood-borne hepatitis (HBV, HCV, HDV) seems to be exceedingly low compared to the risk associated with the use of drugs and certain forms of sexual behaviour. Investigations of reported transmissions of viral hepatitis revealed carelessness as the most important factor in transmission. Cases involving the use of a common towel to wipe bloody wounds, the use of bloody fisticuffs, or the sharing of water to clean wounds have been described as modes of transmission <sup>13</sup>. However, HBV transmission from a HBeAg positive carrier to five players on the same football team, probably due to contact with open wounds during training, underlines the potential transmission of unknown HBV infection <sup>14</sup>. HBV DNA has been detected in the sweat of wrestlers. Transmission could hypothetically take place via open wounds or mucus membrane contact with the sweat of an infected athlete <sup>15</sup>. Because of the resistance of HBV to environmental factors, and the possibility of transmission through non-sterile objects, the risk of HBV transmission during sports is probably greater than that of other

blood-borne infections, including HIV. The risk of HBV transmission is estimated to be between 1:10.000 and 1:4.25 million at football games <sup>16</sup>. However, theoretically, the number of cases of HBV which could potentially be transmitted via sports is significantly higher than those contracted through the healthcare system <sup>17</sup>. This is presumably due to a lack of prophylaxis during sports.

#### Prevention of viral hepatitis during sports

The risk of becoming infected with enteric pathogens such as HAV and HEV is high when athletes travel to areas with poor hygienic conditions and particularly to areas where HAV and HEV are endemic <sup>18</sup>. Good general hygiene practices, compliance with standard/universal precautions, and appropriate immunisation of athletes should be strictly followed. During exercise, athletes should use individual squeeze water bottles to avoid mouth contact.

The risk for transmission of blood-borne hepatitis during sports is extremely low, but may be higher in contact and collision sports, especially in wrestling, boxing, and tae kwon do, because of the higher risk of bleeding injuries and prolonged, close body contact. Players of basketball, field hockey, ice hockey, judo, soccer, and team handball are at moderate risk <sup>17</sup>. Athletes taking part in these sports should be aware of these small theoretical risks and a vaccination should be recommended. Prompt and appropriate treatment of bleeding injuries is mandatory and athletes should be guarantined until the bleeding is controlled and the bleeding site is covered.

However, it should be emphasised that blood borne infections in athletes are not transmitted primarily through sport activities but, as with the general population, due to unsafe sexual behaviour or the sharing of needles. Therefore efforts to prevent these infections among athletes should also focus on the prevention of risks unrelated to sports. Athletes, their families, healthcare providers, coaches, officials and others involved in sports should be educated accordingly.



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