

Single Case

Athlete's Hepatitis in a Young Healthy Marathon Runner

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Keywords

Athlete's hepatitis · Ischemic hepatitis · Extreme exercise

Abstract

Athlete's hepatitis is an uncommon clinical condition characterized by a significant ischemic insult to the liver. The most likely pathogenesis is decreased blood supply to the liver resulting in significant hypoxic hepatocyte injury. We report a case of a 48-year-old healthy young male who presented with athlete's hepatitis confirmed by liver biopsy after a 14-mile marathon run. To our knowledge, this is the second case of athlete's ischemic hepatitis reported in the literature. It is a diagnosis worth considering in patients involved in extreme exercise programs.

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Introduction

The liver receives approximately 30% of the total cardiac output, of which 70% passes through the portal venous system and 30% via the hepatic artery [1]. This complex vascular supply makes the hepatocytes vulnerable to circulatory disturbances, particularly in the setting of increased metabolism [2].

Ischemic hepatitis, also known as hypoxic hepatitis, refers to a diffuse hepatic injury resulting from acute hypoperfusion. It is most commonly seen in patients with cardiac or respiratory failure, and in critically ill patients with septic shock, especially in the presence of

multiple comorbidities [3]. Hemodynamic insult leading to liver injury is usually obvious; however, subclinical circulatory disturbances could as well occur in patients with a preexisting passive congestion, diminished hepatic venous outflow, or portal hypertension [4, 5].

Herein we report a rare case of a healthy young athletic male with no previous medical problems who, after a 14-mile marathon run, presented with the picture of ischemic athlete's hepatitis confirmed by liver biopsy. Athlete's hepatitis is a special clinical entity that is characterized by significant hypoxic damage to the liver precipitated by prolonged and vigorous exercise, in the absence of other risk factors. To our knowledge, this is the second case reported in the literature [1].

Case Report

A 48-year-old athletic healthy male was admitted with difficulty ambulating due to severe bilateral thigh and calf pain worsening over 1 week. The patient had run a 14-mile-long marathon a week before and developed these symptoms which he attributed to cramps and did not seek medical attention. The patient denied any use of acetaminophen, herbal medications, alcohol intake, recent travel, or high-risk sexual behavior.

On arrival, the patient was hemodynamically stable, and was found to be jaundiced. Liver tests (IU/L) were as follows: AST/ALT/ALP 199/170/134, total bilirubin 10, direct bilirubin 4.3, creatinine 7.58, hemoglobin 12.5, platelets 23,000, INR 0.9, PTT 35.5, and CPK 3,258. His admitting diagnosis was rhabdomyolysis with acute kidney injury, thrombocytopenia, and deranged liver profile. His kidney injury was attributed to rhabdomyolysis and acute tubular necrosis. Thrombocytopenia was likely related to athletic stress state since a bone marrow aspirate and biopsy were unrevealing. The patient received aggressive fluid hydration. Further workup for his liver injury included viral serologies, metabolic disorders, α -1 antitrypsin deficiency, drug screen, and autoimmune diseases, all of which were negative. Imaging studies were negative for any intra-abdominal pathology. A liver biopsy (Fig. 1) revealed: (1) diffuse parenchymal cholestasis, increased lipofuscin pigment, and few hepatocytes undergoing mitosis. (2) Iron stain without evidence of increased stainable iron. (3) No fatty infiltration, inflammatory changes, or abnormal liver architecture noted. (4) No evidence of fibrosis by trichome and reticulin stains. These findings were all in favor of athlete's hepatitis in the current clinical scenario. Liver chemistries improved during hospitalization with supportive measures. Trends of liver enzymes during hospital stay are shown in Figure 2 and 3.

Discussion

Ischemic hepatitis is a common etiology of acute liver injury in sick patients with multiple comorbidities admitted to the intensive care unit [6] that may lead to increased hospital stay and mortality. The diagnosis could be challenging since almost half of the patients developing ischemic liver injury do not exhibit specific clinical signs of hemodynamic instability and hence documentation of hypotension could be difficult [7]. Henrion et al. [8] suggested and published a set of criteria to assist in the diagnosis of hypoxic hepatitis: (a) a clinical setting of cardiac, circulatory, or respiratory failure; (b) a substantial but transient increase in serum aminotransferase activity; and (c) exclusion of other putative causes of hepatitis, namely viral hepatitis or drug-induced liver injury.

Athlete's hepatitis is a rare clinical entity that is poorly described in the literature. It is an ischemic hypoxic injury rather affecting the liver of young healthy athletes with no previous medical conditions as is the case in our patient. He was hospitalized with rhabdomyolysis almost a week after his long-distance marathon running. He subsequently developed elevated liver enzymes without any evidence of hemodynamic instability. Extensive investigation of the etiology of his liver injury was initially unrevealing. A liver biopsy was thereafter obtained to favor the diagnosis of athlete's hepatitis in this unique setting. The management consisted of aggressive fluid support and liver enzymes improved during the hospital stay back to baseline within 2 weeks

Athlete's hepatitis is a pathologic state in which extreme exercise leads to a compromise in the blood supply to the liver resulting in ischemic hypoxic damage. This condition results in transient elevation of liver enzymes which gradually improve in 1–2 weeks with supportive measures in the absence of ongoing hypotension [9]. The serum bilirubin level usually starts to rise once aminotransferase levels have begun to decline which seems to be the case in our patient. The severity of liver damage seems to correlate with the intensity of exercise and the degree of hemodynamic compromise [1]. Liver biopsy in athlete's hepatitis typically shows cholestasis, increased lipofuscin pigment and scattered hepatocyte regeneration, and cell mitosis. Lipofuscin is a known marker for oxidative stress [10] and cholestasis can also be seen in ischemic hepatitis.

In conclusion, our case highlights an uncommon scenario of hypoxic hepatitis in a young, healthy athletic patient secondary to strenuous exercise. Athlete's hepatitis is a diagnosis worth considering in such unique patients involved in extreme exercise programs, especially when they present with abnormal liver chemistry profile. Liver biopsy is warranted to confirm the diagnosis, and supportive management with close monitoring is advised to prevent adverse consequences.

Statement of Ethics

Approval from the ethics committee was not required for this case report. Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Disclosure Statement

None of the authors has any financial conflicts of interest to declare.

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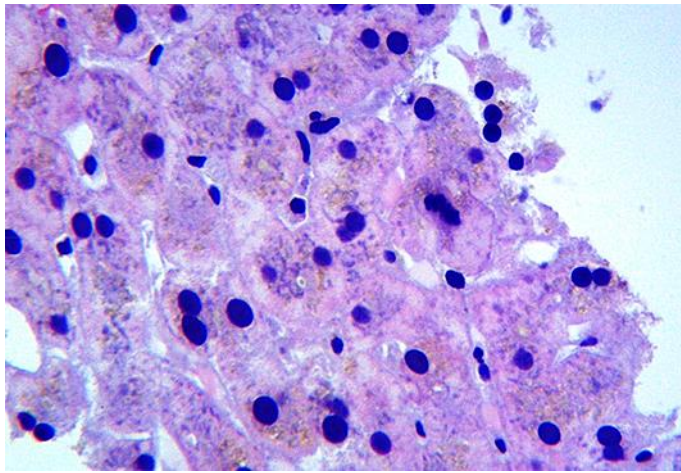


Fig. 1. Hematoxylin and eosin staining of liver biopsy revealed lipofuscinosis and cholestasis suggestive of athlete's hepatitis. $\times 400$.

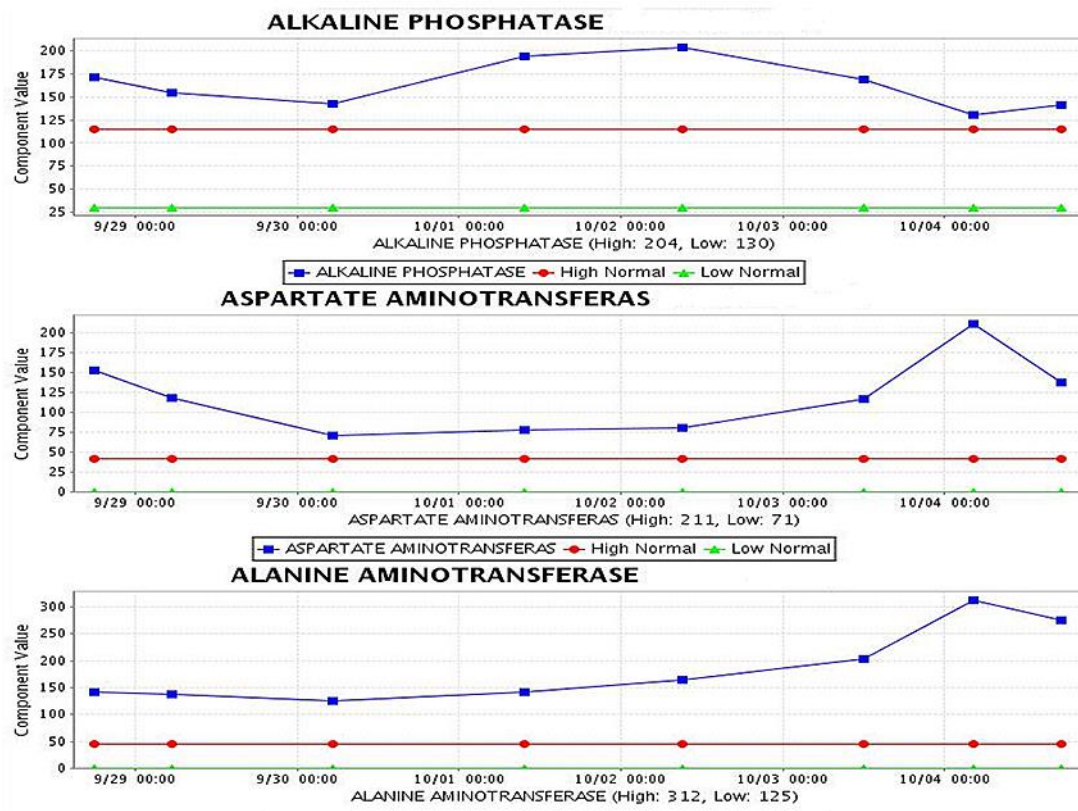


Fig. 2. Graphic representation of the patient's liver enzymes during his hospital stay: liver enzymes during the first week of hospitalization.

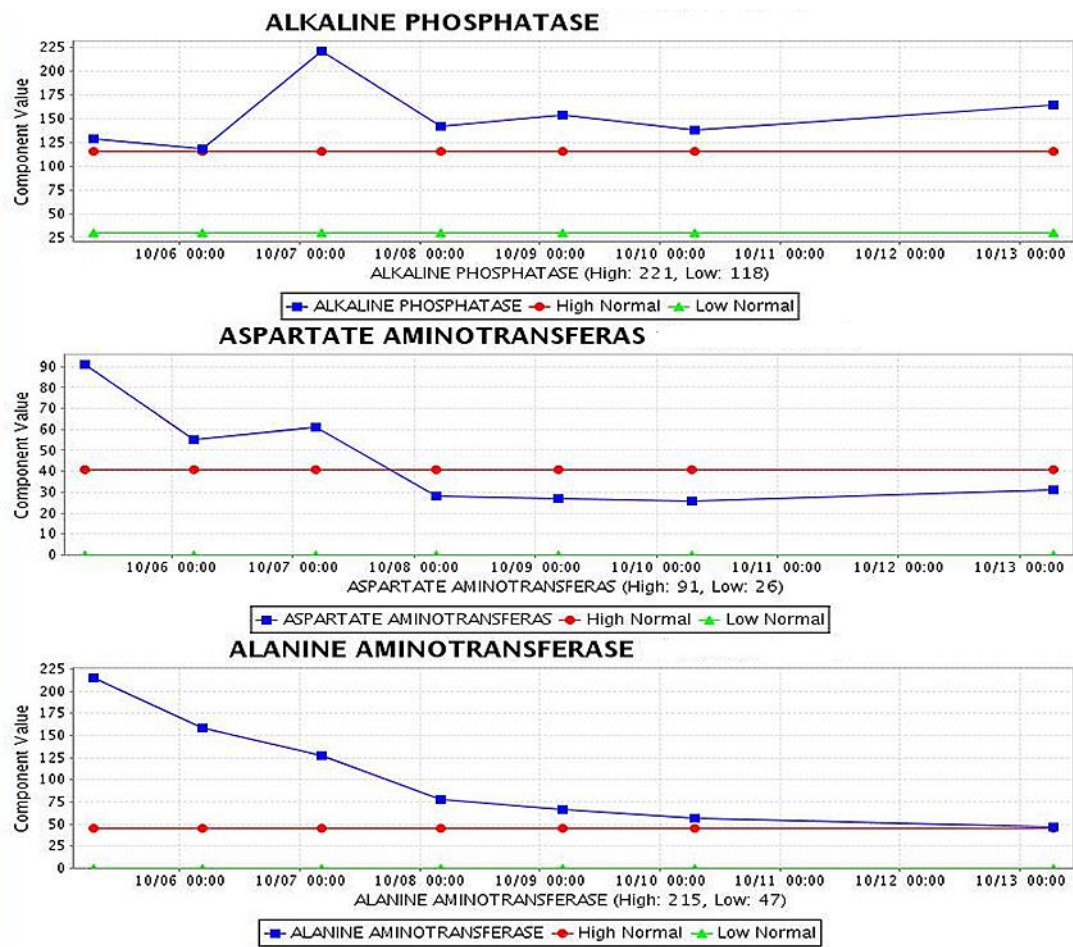


Fig. 3. Graphic representation of the patient's liver enzymes during his hospital stay: liver enzymes trending down during the second week of hospitalization.