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Hypoxic hepatitis - epidemiology, pathophysiology and clinical management.

Fuhrmann V, Jäger B, Zubkova A, Drolz A.

Department of Internal Medicine 3, Division of Gastroenterology and Hepatology, Intensive Care Unit 13H1, Medical University Vienna, Vienna, Austria. valentin.fuhrmann@meduniwien.ac.at

Abstract

Hypoxic hepatitis (HH), also known as ischemic hepatitis or shock liver, is characterized by centrilobular liver cell necrosis and sharply increasing serum aminotransferase levels in a clinical setting of cardiac, circulatory or respiratory failure. Nowadays it is recognized as the most frequent cause of acute liver injury with a reported prevalence of up to 10% in the intensive care unit. Patients with HH and vasopressor therapy have a significantly increased mortality risk in the medical intensive care unit population. The main underlying conditions contributing to HH are low cardiac output and septic shock, although a multifactorial etiology is found in the majority of patients. HH causes several complications such as spontaneous hypoglycemia, respiratory insufficiency due to the hepatopulmonary syndrome, and hyperammonemia. HH reverses after successful treatment of the basic HH-causing disease. No specific therapies improving the hepatic function in patients with HH are currently established. Early recognition of HH and its underlying diseases and subsequent initiation of therapy is of central prognostic importance. The purpose of this review is to provide an update on the epidemiology, pathophysiology, and diagnostic and therapeutic options of HH.

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