Assessment of Cardiovascular Risk in Patients with Fatty Liver: Impact of Hepatic Cirrhosis

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Introduction and Objectives: According to the literature, cardiovascular events have been described as the leading cause of death in patients with fatty liver associated with metabolic dysfunction (MASLD). The main objective of this study is to assess and compare cardiovascular risk in two groups of patients: those diagnosed with fatty liver without cirrhosis and those with liver cirrhosis attributable to fatty liver. The aim is to determine if there is a significant difference in cardiovascular risk between these groups, identify the most relevant cardiovascular risk factors, and explore possible associations between progression to liver cirrhosis and increased cardiovascular risk.

Materials and Patients: A retrospective cross-sectional study was conducted from 2020 to 2024, involving a total of 289 patients, of whom 165 were diagnosed with MASLD without cirrhosis and 125 patients were diagnosed with cirrhosis associated with fatty liver. In the first group, the grade of hepatic steatosis was determined by imaging methods, and cardiovascular risk assessment scales such as GLOBORISK and PREVENT were applied to both groups, conducting a comparative analysis between these study groups. Additionally, variables such as sex, age, weight, height, obesity, sedentary lifestyle, glomerular filtration rate, smoking, diabetes, hypertension, total cholesterol levels, LDL, HDL, and triglyceride levels were evaluated.

Results: In the present study, 165 patients diagnosed with MASLD were evaluated using two cardiovascular assessment scales: PREVENT and GLOBORISK. According to the PREVENT scale, 86 patients (52.1%) exhibited a low cardiovascular risk, with 50.9% also showing mild hepatic steatosis confirmed by imaging studies. Using the GLOBORISK scale, it was determined that 117 patients (70.9%) had a low level of cardiovascular risk. On the other hand, a total of 124 patients with hepatic cirrhosis associated with fatty liver were included. According to the evaluation using the PREVENT scale, it was found that 64 patients (51.6%) had an intermediate cardiovascular risk, and according to the GLOBORISK model, 45 patients (36.2%) were classified with a moderate risk. When contrasting between the group of patients with cirrhosis and those with only fatty liver, the first group has a 3.6 times higher likelihood (OR 3.6) of presenting a moderate to severe cardiovascular risk compared to those without cirrhosis (P=0.00).

Conclusions: This study demonstrates that patients with cirrhosis associated with fatty liver have a 3.6 times higher prevalence of moderate to severe cardiovascular risk compared to patients without cirrhosis but with fatty liver. This suggests a need for closer monitoring of cardiovascular events alongside liver disease monitoring.

Ethical statement: The informed consent for the use of personal data was obtained from all study participants.

Declaration of interests: None.

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Autoinmne hepatitis associated with hepatitis A virus infection, a case report

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Introduction and Objectives: Hepatitis due to Hepatitis A Virus (HAV) is an entity that has been described as a causal factor of HAI, the prevalence and course of which is reported to be 1% - 3%. The diagnosis is associated with AIH is usually made in the acute event; a time criterion is not well defined.

Materials and Patients: 41-year-old male, with a history of DM2, systemic arterial hypertension and rheumatoid arthritis, onset in June 2023 with fever and gastrointestinal symptoms (vomiting, nausea and stools with reduced consistency), associated with jaundice of 1 week after his symptoms. Diagnosis of Acute Liver Injury due to HAV is confirmed on 06/22/23, with Ac. IgM VHA (8.7 +), Transaminases >2000U/L and INR 2.4; support therapy and symptom control began with partial resolution on 09/2023. He subsequently reentered the emergency area 11/2023 with jaundice, abdominal pain, and excessive fatigue. Acute Hepatitis was again determined with transaminases >2000 U/L, a 3F CT scan was performed and was normal, and the approach for autism was complemented with the following panel: negative ANAS and positive ASMAs 1:100, IgG 2780. Liver biopsy confirmed AIH. morphological changes compatible with autoimmune hepatitis. Treatment was started with Prednisone 0.5mg/kg, with subsequent maintenance based on Azathioprine, achieving biochemical remission 04/2024

Results: It has been postulated that HAV infection, as occurs with other viral infections, may be a triggering factor for latent AIH in susceptible individuals, considering multiple pathways of inflammation and immunotolerance defects. Most of the reported cases are diagnosed 5 months after the acute event HAV; in the case of our patient, it was 6 months after the acute event, completing a score of 7 points by the simplified system. In case reports of OAB-associated AIH, treatment has been initially established with oral Prednisone 0.5 to 1 mg/kg day, with maintenance of Azathioprine or Mycophenolate Mofetil with comparable response rates. The goal of treatment is biochemical and histological remission with the goal of avoiding progression of liver damage and mortality.

Conclusions: Viral infections have been associated with the development of autoimmune hepatitis, HAV in up to 3% based on case reports due to the rarity of the presentation. The pathophysiology of presentation triggered by OAB is poorly defined. Biopsy and differential diagnoses are the mainstay in the approach to these patients.

Ethics statement: the ethics statutes dictated by the scientific committee of the Ignacio Morones Prieto Central Hospital were followed.

Declaration of interests: None.

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Acute-on-chronic liver failure due to hepatitis A infection in a patient with Metabolic Dysfunction-Associated Fatty Liver Disease. Case report.

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Introduction and Objectives: Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD) has steadily increased its prevalence, making it the most common liver disease in Western industrialized nations, affecting one billion people worldwide. Hepatitis A is a necro-inflammatory liver disease caused by the hepatitis A virus (HAV). Less than 1% develop acute liver failure, where 30% will require a liver transplant and 70% will require supportive therapy until recovery. Hepatic steatosis is recognized as a risk factor for