

was performed utilizing the Mann-Whitney U test, with statistical significance set at  $p < 0.05$ .

**Results:** The subjects were classified into RI (22), Ab (4), OH (28), CiOH (76), HA (16), and CT (100). The GSH was found to decrease significantly in the EHA groups vs CT. In contrast, GSSG increased in the RI, Ab, OH, and CiOH groups compared to CT, indicating that alcohol consumption favors an oxidizing state, confirmed by the negative GSH/GSSG ratio. Additionally, the GSH/GSSG ratio in the OH group showed a greater imbalance than in patients with EHA. On the other hand, protein oxidation increased in EHA, with high levels of carbonylated proteins observed in OH, CiOH, and HA compared to CT, Ab, and RI. Furthermore, lipoperoxidation measured by Malondialdehyde showed increased levels of OH and CiOH compared to the other study groups.

**Conclusions:** Excessive alcohol consumption, with or without liver damage, promotes the oxidation of proteins and lipids. Additionally, alcohol favors the oxidized form of the main endogenous antioxidant, GSH. Therefore, it is necessary to control the redox balance through antioxidant treatment.

**Ethical statement:** The protocol was approved by the Ethics and Research commissions of the General Hospital of México "Dr. Eduardo Liceaga" (DI/16/107/03/031) and from the UNAM, Facultad de medicina (FM/DI/135/2017).

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### Prevalence and evaluation of sleep disturbances in Mexican patients with hepatic cirrhosis through the application of the Pittsburgh sleep questionnaire

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**Introduction and Objectives:** According to the literature, patients with cirrhosis have a high prevalence of sleep disturbances, which increase as the disease progresses. There are few studies conducted in this patient group, with a small number of samples, reflecting alterations in sleep quality and rest. The Pittsburgh Sleep Quality Index (PSQI) is a tool that allows us to evaluate sleep quality and the level of disturbances it may present. To assess the type and prevalence of sleep disturbances in a Mexican group of patients with cirrhosis through the application of the Pittsburgh questionnaire.

**Materials and Patients:** A prospective, cross-sectional, and epidemiological study was conducted with 300 individuals, of whom 266 did not have hepatic diseases and 74 were diagnosed with cirrhosis.

The Pittsburgh questionnaire was administered to them, which consists of 7 components that generate a total score. Total scores were interpreted as follows: 1-4 without sleep disturbances, 5-7 with mild disturbance, 8-14 with moderate disturbance, and 15 or more indicating severe disturbance.

Results were compared using Odds Ratio (OR) to assess the effect.

**Results:** Of the individuals evaluated, 74 (24.66%) were diagnosed with cirrhosis, with 42 women (56%) and 32 men (43.24%). The remaining 226 participants (75.33%) did not have liver diseases, with 150 women (66.3%) and 75 men (33.1%). When comparing the total scores, it was observed that 57 people without sleep disturbances, 18 (31.57%) were in the cirrhosis group, while 39 (68.42%) were not. Additionally, of the 102 individuals with mild alterations, 20 (19.60%)

were in the cirrhosis group and 82 (80.39%) were not. Of the 131 individuals with moderate alterations, 32 (24.42%) had cirrhosis and 99 (75.57%) did not. Finally, of the 10 individuals with severe alterations, 4 (40%) had cirrhosis and 6 (60%) did not. The calculation of the Odds Ratio was 1.09, indicating that patients with cirrhosis had a similar risk of sleep disturbances as those without cirrhosis.

**Conclusions:** In our study, it seems to demonstrate that contrary to previous reports in the literature, no difference was found in the prevalence of sleep disturbances between our population without cirrhosis and patients with cirrhosis.

This study is the first to apply this validated and translated questionnaire in Spanish to a Mexican population of patients with cirrhosis and healthy individuals to evaluate their sleep quality and the first to have a significant sample.

**Ethical statement:** Patients' identity was protected. Consentment was obtained directly from patients.

**Declaration of interests:** None

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### Autoimmune hepatitis developed after acute liver failure due to hepatitis A. A case-report

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**Introduction and Objectives:** In this report, the relationship between Hepatitis A Virus (HAV) and Autoimmune Hepatitis (HA) will be analyzed, demonstrating the presence of HA after an acute HAV infection, highlighting the time between diagnoses and seroconversion with antibodies, as well as clinical characteristics and evolution.

**Materials and Patients:** 30-year-old male patient, with no personal pathological history who presents with general malaise, fever and jaundice. The clinical examination was within normal limits except for slight jaundice. The admission biochemical analyzes were as follows: Hb 14.2 g/dL, Leukocytes 8,550 /mm<sup>3</sup>, Total bilirubin 9.5 mg/dL Direct bilirubin 2.2 mg/, ALT 6155 U/L, AST 3940 U/L, Alkaline Phosphatase 1150 U/L and Prothrombin time 51.0 seconds; INR 4.95, a viral hepatitis profile with positive anti-HAV IgM antibodies and an imaging examination of the liver and bile ducts with inflammatory changes. The diagnosis of hepatitis A and acute liver failure was made.

The patient suffers rapid clinical and biochemical deterioration, with multiple organ failure requiring admission to an intensive care unit and advanced life management area due to acute respiratory failure syndrome, general support stockings and three sessions of single-step albumin dialysis were indicated. He showed stabilization and improvement in his general condition.

**Results:** 27 days after initial evaluation, fatigue and fever of unknown origin were present. Liver function test with BT 25.58 mg/dl, BD 17.55.0 mg/dl, ALT 38 U/l, AST 100 U/l and ALP 105 U/l and INR 1.5. He presented positive antinuclear antibodies with a cytoplasmic pattern with a titer of 1:80, SMOOTH MUSCLE 3+ intermediate filament pattern. DILUTION 1:80 immunoglobulin G 3260 mg/dl. A liver biopsy was performed, which showed changes compatible with autoimmune hepatitis (fig. 1). In the previous context, the diagnosis of autoimmune hepatitis triggered by HAV was made and treatment was started with prednisone 50 mg every 24 hours PO in a reduced dose of azathioprine 50 mg every 24 hours.