

The mean age at diagnosis was 51 years $\pm$ 13.2. 28% had associated autoimmune diseases, with thyroid disease being the most common at 33%. Hepatic cirrhosis was the most frequent presentation (60%) at diagnosis, with 60% exhibiting decompensation (64% with variceal digestive bleeding) (Figure 2). PBC accounted for 47% of cirrhosis cases. Liver transplantation was performed in 6% of EHA cases, mainly due to AIH.

Conclusions: The diagnosis of AHD shows a progressive increase. A high incidence of advanced stages of liver disease related to AHD is observed. Further research is needed to define the prevalence and characterize these patients, thus enhancing early and effective diagnosis.

Ethical statement: This study was conducted in accordance with the ethical principles of our hospital center. All data were handled with strict confidentiality and solely for research purposes.

Declaration of interests: None.

Funding: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

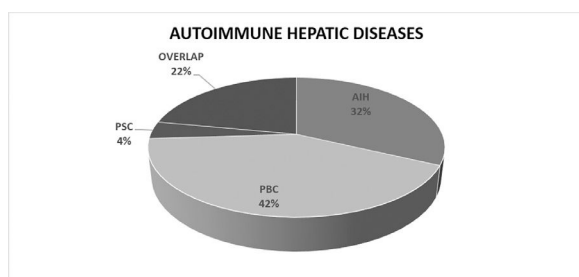


Figure 1. Autoimmune Hepatic Diseases (AHD). AIH: Autoimmune Hepatitis, PBC: Primary Biliary Cholangitis, PSC: Primary Sclerosing Cholangitis.

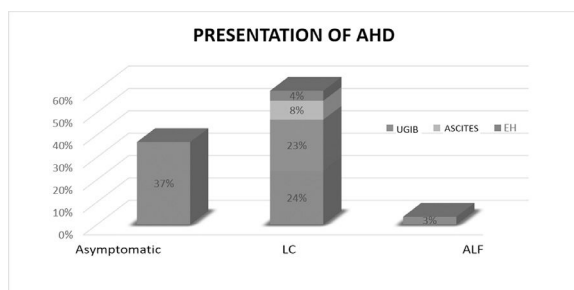


Figure 2: Presentation of Autoimmune Hepatic Diseases. AHD: Autoimmune Hepatic Disease, LC: Liver Cirrhosis, ALF: Acute Liver Failure, UGIB: Upper Gastrointestinal Bleeding, EH: Hepatic Encephalopathy.

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Behavioral Assessment of a Novel Hepatic Encephalopathy Model using CCl₄ and Manganese in Mice

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Introduction and Objectives: Hepatic encephalopathy (HE), affecting around 40% of cirrhosis patients, impairs cognitive and motor functions. Developing HE experimental models is crucial for

advancing our understanding of this condition. This study developed an HE models using intraperitoneal carbon tetrachloride (CCl₄) and manganese supplementation in mice, focusing on behavioral validation.

Materials and Patients: Two groups of male C57BL6 wild-type mice (8 mice per group), 10 weeks old, were used in this study. The first group (healthy controls) had access to standard food (Rodent Laboratory Chow* 5001, LabDiet, Richmond, IN, USA), and drinking water ad libitum and were euthanized at week

12. The second group (cirrhotic group) received the same diet but with 1 mg/ml of MnCl₂ added to their drinking water. It was intraperitoneally injected twice a week with CCl₄ for 12 weeks (1 ml/kg of body weight dissolved in olive oil for a final concentration of 30% in the first 5 weeks and 20% in the following 7 weeks). Behavioral tests, including the beam walking test and cylinder test, were conducted to assess motor coordination and motor asymmetry. Liver morphology changes were observed, and Hematoxylin-Eosin staining was used to determine inflammation. Data were analyzed using ANOVA for parametric data and the Kruskal-Wallis test for non-parametric data, with results presented as Mean $\pm$ SEM.

Results: Behavioral tests indicated signs of HE, such as gait abnormalities (tremor, rigidity), hind limb ataxia, and bristly hair. In the beam walking test, cirrhotic mice spent significantly longer to traverse the beam ($P \leq 0.05$) and had a higher number of limb foot faults ($P \leq 0.001$) compared to healthy mice. The cylinder test showed no significant difference in locomotor asymmetry. Morphological changes in the liver from healthy to cirrhotic were evident. Healthy livers had a smooth reddish-brown surface, regular shape, and firm texture. In contrast, cirrhotic livers appeared paler, with an irregular surface, and became harder and bumpy. Size alterations and the presence of leukocytic foci were also noted in cirrhotic livers.

Conclusions: The combination of CCl₄ and manganese successfully induced evidence of significant motor coordination impairments and distinct liver morphology changes, indicating a noticeable progress in developing the experimental model for HE.

Ethical statement: Technical specifications for the production, care, and use of laboratory animals followed the NOM-062-ZOO-1999. Additionally, guidelines from the animal facility of the University of Guadalajara and criteria outlined in the Guide for the Care and Use of Laboratory Animals published by the National Institutes of Health were adhered to.

Declaration of interests: None.

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HDL-C and BMI levels as parameters for MASLD detection

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Introduction and Objectives: Hepatic Steatosis Associated with Metabolic Dysfunction (MASLD) has a prevalence of 30% worldwide and 80% of these patients do not present alterations in liver biochemistry, therefore it is important to know if there is any biochemical parameter that helps us identify this population. To correlate clinical and biochemical values with the degree of fibrosis and CAP determined by Transient Elastography to obtain a parameter that determines the affected population.

Materials and Patients: Patients with MASLD criteria were included, who underwent transient hepatic elastograph (Fibroscan® 630 Expert v10720), APRI, FIB4, NAFLD score, blood count, liver biochemistry, lipid profile, glucose, glycosylated hemoglobin, clotting times. (TP, INR). It was compared with a control of healthy people. The statistical analysis was used SPSS V24 program for continuous quantitative variables expressed in mean and percentage, the ordinary quantitative variables were expressed in frequencies and percentages, Spearman correlation tests and a linear regression analysis were performed, from which A ROC curve and the Youden index were performed and their sensitivity and specificity were determined, with a statistically significant $p < 0.05$.

Results: 81 patients were included, mean age 43 years (38, 50.5), with the following comorbidities: 2 (2.5%) HTS, 8 (10%) T2D. The control group (healthy) was 17. By BMI, 29 (35.8%) were overweight, 33 (40.7%) were grade I obese, and 7 (8.6%) were grade II obese.

By CAP, 31 (38.35) had S3, 26 (32.1%) S2, 7 (8.6%) S1 and 17 (21%) S0. Patients with obesity I or II have grade 2 or 3 steatosis, with a moderate correlation Spearman's rho 0.581 $p < 0.001$.

Both groups were compared, reporting that the age, BMI, CAP and KPa of patients with steatosis are higher compared to healthy participants, as well as leukocytes, glucose, triglycerides, HDL, GGT and Na with statistical significance. The linear regression analysis showed the following formula $m = -0.617 + 0.062 \text{ (BMI)} + -0.009 \text{ (HDL)}$, with an R of 0.737. An ROC curve was made with the formula obtained with an area under the curve of 0.979 and a p of < 0.0001 , having a Youden index with a cut-off point of 0.60, obtaining a sensitivity of 95.2% and a specificity of 87.5%. Regarding fibrosis, 9 patients with fibrosis were detected, whose age is 47.4 ± 14.2 years. The most common grade in patients with fibrosis was F2, as shown in the table.

Conclusions: HDL levels and BMI could be markers to suspect MASLD. A larger population is required to validate it.

Ethical statement: Approved by the research committee of the Central Military Hospital, review of files confidentially following the guidelines of the Declaration of Helsinki.

Declaration of interests: None.

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	S0 n (%)	S1 n (%)	S2 n (%)	S3 n (%)
Normal weight	11 (91.7)	0 (0)	1 (8.3)	0 (0)
Overweight	5 (17.2)	5 (17.2)	11 (37.9)	8 (27.6)
Grade I Obesity	1 (3)	1 (3)	12 (36.4)	19 (57.6)
Grade II Obesity	0 (0)	1 (14.3)	2 (28.6)	4 (57.1)
$\chi^2 = 52.230 \text{ } p < 0.001$				

	Total n (%)
F0 – F1	72 (88.9)
F2	6 (7.4)
F3	2 (2.5)
F4	1 (1.2)

Characterization of the inflammatory profile in patients with compensated and decompensated liver cirrhosis through cytosines determined by spectroscopy.

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Introduction and Objectives: Inflammatory cytokines influence the progression of cirrhosis and decompensation. The study aims to characterize the inflammatory response of patients with compensated and decompensated liver cirrhosis through inflammatory cytokines and evaluate the state of the disease, type of decompensation, severity and the development of acute on chronic liver failure.

Materials and Patients: Hospitalized patients with a diagnosis of compensated and decompensated liver cirrhosis were included. Upon admission, saliva samples were collected in microcentrifuge tubes to measure cytosines (IL-6, IL-1 β , IL-10, ILF- γ and TNF), lipids and immunoglobulins: A, M and G using Fourier transform infrared spectroscopy (FTIR). Clinical and biochemical variables (complete blood count, blood chemistry, liver biochemistry, serum electrolytes, lipid profile and C-reactive protein), MELD 3.0 and Child Pugh scales were included. The statistical analysis was used the SPSS V24 program for continuous quantitative variables expressed in measures of central tendency and dispersion according to the normality of the data, the ordinal quantitative variables were expressed in frequencies and percentages, Spearman correlation analysis and a linear regression analysis were performed, from which a ROC curve and the Youden's J statistic and its sensitivity and specificity were determined, with a statistically significant $p < 0.05$.

Results: It was included 40 patients: 19 compensated and 21 decompensated. The most common decompensation was hepatic encephalopathy. (20%) (MELD 3.0 12.5 ± 3.59 vs 21.61 ± 7.47 , $p < 0.000$). Statistical significance was found in leukocytes, neutrophils and INR as well as differences in the levels of IgG, IgM, IL-6, IL1 β , IFN- γ and IL-10 between the causes of decompensation (Figure 1) and decreased IgM levels. And IFN- γ in decompensated patients compared to compensated patients. A negative correlation was found between neutrophil levels and IgM, IL6, IL1 β , IL10 and IFN- γ levels. The linear regression analysis gave the following formula $m = 2.648 + (-0.267 * \text{infection}) + (-0.926 * \text{abs1}) + (0.084 * \text{abs2}) + (0.442 * \text{abs3}) + (-0.051 * \text{abs12}) + (0.005 * \text{IgM}) + (-0.064 * \text{IFN}\gamma) + (-0.2 * \text{Leukocytes}) + (0.223 * \text{Neutrophils}) + (0.006 * \text{Urea})$, $R = 0.623$. With the same formula, AUROC: 0.877 and p value < 0.0001 , Youden's J statistic cut-off of 1.3913, obtaining sensitivity of 92.1%, and specificity of 78.9%. The correlation with Child-Pugh is negative with IgM levels, while it was no association between the presence of infection and decompensation ($\chi^2 = 0.053$, $p = 0.818$), an association was indeed observed between Child-Pugh and the presence of infection ($\chi^2 = 15.126$, $p = 0.001$).

Conclusions: No correlation was found between levels of IgG, IL-6, IL1 β , IFN- γ and IL-10 and the MELD 3.0 and Child Pugh scales, there is only a correlation between the Child Pugh clinical stage and IgM. Low levels of IgM and IFN- γ could be markers in patients with decompensated cirrhosis.

Ethical statement: The present study was approved by the research committee of the Central Military Hospital with registration number 045/2024. The samples were obtained under informed consent of the patients.