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 elastograft (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 (BMI) + -0.009 (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 weigth	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)
X^2= 52.230 p <0.001				

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

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

Carlos E. Coronel-Castillo[1], Gustavo J. Vázquez-Zapien[2], Mónica M. Mata-Miranda[2], Eira Cerda-Reyes[3], Adriana Martínez-Cuazitl[2,3]

[1] *Internal Medicine, Military Postgraduate Medical School, Mexico*
[2] *Military School of Medicine, Mexico*
[3] *Research department, Central Military Hospital, Mexico*

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-1β, 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, IL1β, 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, IL1β, IL10 and IFN-γ levels. The linear regression analysis gave the following formula m= 2.648+ (-0.267*infection) + (-0.926*abs1) + (0.084*abs2) + (0.442*abs3) + (-0.051*abs12) + (0.005*IgM) + (-0.064*IFNγ) + (-0.2*Leukocytes) + (0.223*Neutrophils) + (0.006*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 (X^2= 0.053, p= 0.818), an association was indeed observed between Child-Pugh and the presence of infection (X^2= 15.126, p= 0.001).

Conclusions: No correlation was found between levels of IgG, IL-6, IL1β, 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.

Declaration of interest: None.

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

Table 1
Immunoglobulin and cytokine levels by FTIR

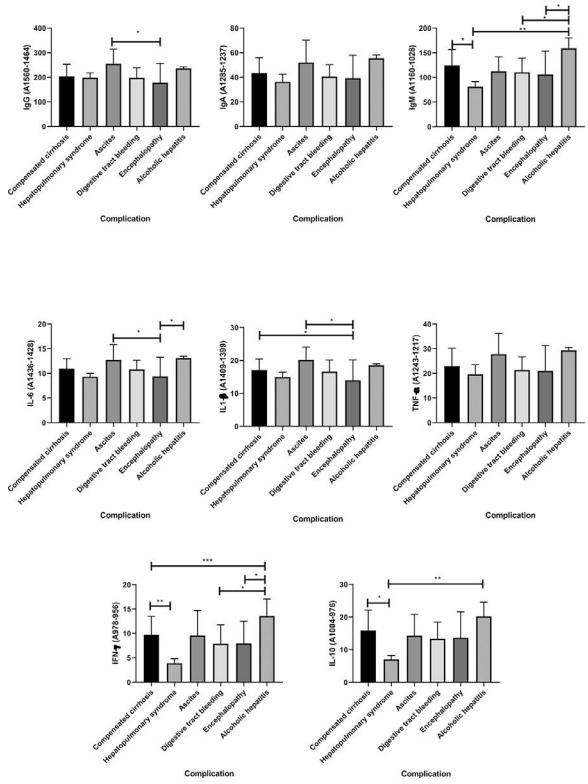
	Compensated	Decompensated	p
IgG (1560–1464 cm^{-1})	203.79 ± 49.42	199.71 ±60.90	0.690
IgA (1285–1237 cm^{-1})	43.44 ± 12.49	42.19 ± 14.98	0.622
IgM (1160–1028 cm^{-1})	124.35 ± 32.39	110.97 ± 39.0	0.044
IL-6 (1436–1428 cm^{-1})	10.94 ± 2.05	10.54 ± 3.06	0.412
IL-1β (1409–1399 cm^{-1})	17.10 ± 3.36	15.99 ± 4.88	0.151
TNF-α (1243–1217 cm^{-1})	23 ± 7.21	22.47 ± 8.06	0.705
IFN-γ (978-956 cm^{-1}),	9.78 ± 3.71	8.23 ± 4.55	0.044
IL-10 (1004–976 cm^{-1})	15.88 ± 6.29	13.61 ± 6.74	0.059

IgA, immunoglobulin A; IgG, immunoglobulin G; IgM, immunoglobulin M; IFN-γ, interferon-gamma; IL-1β, interleukin-1 beta; IL-6, interleukin-6; IL-10, interleukin-10; TNF-α, tumor necrosis factor-alpha.

Table 2
Correlation between neutrophil levels and IgM, IL6, IL1β, IL10 and IFN-γ levels

Neutrophiles	IgG	IgA	IgM	IL6	IL-1β	TNF	IL10	IFN-γ
Pearson correlation	-0.148	-0.160	-.226*	-.199*	-.214*	-0.174	-.273**	-.224*
Sig. (bilateral)	0.111	0.084	0.014	0.032	0.021	0.061	0.003	0.015

Figure 1. Differences between the levels of IgG, IgM, IL-6, IL1β, IFN-γ and IL-10 depending on the cause of decompensation.



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Impact of bilirubin molecular species on mortality in patients with acute on chronic liver failure (ACLF).

Stephany M. Castañeda-Castillo[1,2], Jacqueline Cordova-Gallardo[3,4], Liliana Rivera-Espinosa[5], Juan L. Chavez-Pacheco[5], Mariana M. Ramírez-Mejía[1,6], Nahum Méndez-Sánchez[1,4]

[1] *Liver Research Unit, Medica Sur Clinic & Foundation, Mexico City, Mexico City, Mexico*
[2] *Medical, Dental and Health Sciences Master and Doctorate Program, National Autonomous University of Mexico*
[3] *Department of Hepatology, General Hospital Dr. Manuel Gea González, Mexico*
[4] *Faculty of Medicine, National Autonomous University of Mexico*
[5] *Pharmacology Department, National Institute of Pediatrics, Mexico*
[6] *Plan of Combined Studies in Medicine (PECEM-MD/PhD), Faculty of Medicine, National Autonomous University of Mexico*

Introduction and Objectives: Acute-on-chronic liver failure (ACLF) represents a serious and potentially life-threatening complication in patients with chronic liver disease. This condition is characterized by a rapid deterioration of liver function in patients with pre-existing chronic liver disease. Among the numerous biomarkers used to assess the severity and prognosis of ACLF, serum bilirubin has emerged as a key indicator of liver dysfunction and clinical deterioration. This study aims to analyze the performance of molecular bilirubin species, such as unconjugated (UCB), monoglucuronide (BMG) and diglucuronide bilirubin (BDG), and their impact on mortality in individuals with ACLF.

Materials and Patients: The study included 45 patients with ACLF of various etiologies. The diagnosis was made using the European Association for the Study of the Liver (EASL-CLIF) consortium definition. Clinical and laboratory data were collected to determine severity and assess mortality during the 90 days following enrollment. Bilirubin was extracted from serum samples to measure UCB, BMG, and BDG by liquid chromatography-mass spectrometry (LC-MS). The quantification of bilirubin was performed by monitoring the mass charge (m/z) ratio.

Results: Of the 45 patients, 40% (n=18) were categorized as ACLF grade 1, 35.5% (n=16) as ACLF grade 2, and 17.7% (n=8) as ACLF grade 3. Regarding the molecular species of bilirubin, it was observed that the values of UCB, BMG, and BDG increased according to the severity of ACLF, specifically those of BMG (p=0.019). Additionally, it was observed that individuals who died had higher levels of BDG (4.49 vs. 1.17), BMG (64.30 vs. 28.57), and UCB (21.92 vs. 16.99) with respect to individuals who remained alive.

Conclusions: In conclusion, our findings reveal an association between BDG, BMG and UCB levels and ACLF severity, suggesting that the suggest that molecular bilirubin species could be useful as prognostic markers in patients with ACLF. However, further studies are required to validate these findings and further explore the role of bilirubin in the prognosis and pathophysiology of ACLF.

Ethical statement: All procedures performed were carried out in accordance with the ethical standards of the Ethics Committee of the Clinica Medica Sur Foundation (protocol code 2021-EXT-552) and with the 1964 Declaration of Helsinki and its subsequent amendments, or other comparable ethical standards.