

body mass index lowers its sensitivity and specificity, so it should be performed with other advanced imaging modalities.

The authors declare that there is no conflict of interest.

Table

Relationship of CT/HDL, TG/HDL, lipid profile and metabolic syndrome with the degree of CAP by FibroScan

Parameters	Degree of CAP by FibroScan			P-value
	S1	S2	S3	
Triglycerides ^a	111.5 (14.82)	178.75 (64.5)	236.41 (113.10)	0.009
HDL cholesterol ^a	51.02(9.09)	46.49(10.17)	41(9.24)	0.068
LDL cholesterol ^a	126.75 (29.47)	113.72 (31.55)	115.94 (27.08)	0.331
Total cholesterol ^a	193 (48.10)	180.95(40.5)	188(38.68)	0.781
CT/HDL Ratio ^b	3.9 (3.1-4.1)	3.8(2.3-6.8)	4.5(3.1-7.2)	0.076
TG/HDL Ratio ^b	2.3 (1.6-2.7)	3.8 (0.9-7.9)	4.6 (2-16.9)	0.008
Metabolic syndrome ^c				
Yes	1 (2.9)	13 (38.2)	20 (58.82)	0.003
No	3 (21.42)	7 (50)	4 (28.5)	

a: mean (standard deviation) b: median (ranges) c: number (percentage)

LDL: Low Density Lipoprotein HDL: High Density Lipoprotein

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DIFFERENTIAL PROFILE OF PRO-INFLAMMATORY / ANTI-INFLAMMATORY CYTOKINES AND MALONDIALDEHYDE IN PATIENTS WITH ALCOHOL-INDUCED OR OBESITY OR MIXED INJURY

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Introduction and Objectives: Dysregulation of pro-inflammatory/anti-inflammatory cytokines and oxidative stress markers has been reported in Non-Alcoholic Steatohepatitis (NASH) and alcoholic steatohepatitis (ASH); however, in the disease recently known, as Both Alcoholic and Non-Alcoholic Steatohepatitis (BASH) that meets the criteria of ASH and NASH have not been described. The objective was to evaluate the influence of alcohol and obesity on a differential profile of cytokines and malondialdehyde (MDA) in patients with these etiologies.

Material and methods: Cross-sectional, prospective, observational study. Patients from the "Dr. José E. González" from March 2019-March 2020, with a diagnosis of ASH (alcohol consumption \geq 5 years, 30 g/day for men and 20 g/day for women), NASH (demonstrated by ultrasound, FibroScan or FibroMax) and BASH (ASH and NASH criteria). The serum cytokines interleukin-8 (IL-8), interleukin-6 (IL-6), tumor necrosis factor alpha (TNF- α), interleukin-1 β (IL-1 β), interleukin-10 (IL-10) and malondialdehyde (MDA) were determined. The protocol was approved by the ethics committee with registration MI19-000016. One-way analysis of variance was performed with Kruskal-Wallis and Dunn's post hoc. The results were expressed as median (interquartile range). The analysis was performed using Graph Pad Prism (v. 7.04, San Diego, CA, USA). A value of $p < 0.05$ was considered significant.

Results: The patients were: 34 BASH, 43 NASH and 35 ASH. The severity of the patients with respect to clinical and biochemical parameters in increasing order was NASH < BASH < ASH. It was

observed in increasing order that the levels of the cytokines IL-8, IL-6, IL-10 were: NASH < BASH < ASH; TNF- α levels were: BASH < NASH < ASH; IL-1 β levels show no significant difference between groups (Figure). The BASH group showed higher concentration levels of MDA [20.00 (9.00-30.50) pg/mL] with significant difference ($p = 0.0434$) compared to NASH [14.00 (3.00-19.00) pg/mL], but not with ASH (Figure). Serum levels of IL-6 (A; $p < 0.0001$), TNF- α (B; $p = 0.0014$), IL-8 (C; $p < 0.0001$), IL-10 (D; $p < 0.0001$) and IL-1 β (E; Not significant) in the different study groups.

Discussion: In ASH and NASH, common pathogenetic mechanisms mediated by pro-inflammatory.

The authors declare that there is no conflict of interest.

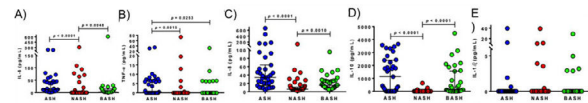


Figure. Serum levels of IL-6 (A; $p < 0.0001$), TNF (B; $p = 0.0014$), IL-8 (C; $p < 0.0001$), IL-10 (D; $p < 0.0001$) and IL-1 β (E; NS) in the different study groups. The values were expressed as median (interquartile range). NS: not significant.

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PROFILE OF PRO AND ANTI-INFLAMMATORY CYTOKINES IN PATIENTS WITH CHRONIC LIVER DISEASE IN THE COMPENSATION, INFLAMMATION AND IMMUNOSUPPRESSION PHASES

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Introduction and objectives: Decompensated cirrhosis is defined by the onset of complications and is associated with immune dysfunction. This is the result of two processes: systemic inflammation and damage made by the immune system. Our objective was to determine the profile of pro and anti inflammatory cytokines in patients with chronic liver disease: alcoholic (OH), non-alcoholic steatohepatitis (NASH), autoimmune liver disease (AILD) and hepatitis C (HCV), in the phases of compensation, inflammation and immunosuppression, based on functional classifications and prognosis with the CHILD- PUGH, MELD and D'Amico scales.

Methods and materials: Prospective, observational, and cross-sectional study, made in the University Hospital, Dr. Jose E. Gonzalez during 2019-2020. A total of 108 patients were included: 28 OH, 27 NASH, 25 HCV and 27 AILD. The diagnosis and functional classification was made according to international guidelines. Inclusion criteria: over 18 years of age, signed informed consent. Exclusion criteria: hepatocellular carcinoma, other autoimmune pathologies. Blood samples (10 ml) were collected to quantify TNF- α , IL-8, IL-10, IL-1, IL-6. The protocol was approved by the ethics committee with registration MI20-0002. A one-way ANOVA was used to determine the differences between groups and stages

Results: In OH, there is an increase in IL-6 and IL-8 in the decompensation phase, Child-Pugh stage C, D'amico stage 5 and MELD from 25 to 34 points. NASH patients had an increase in IL-8 in the inflammation phase as assessed by Child-Pugh B and D'amico 3 and 4. There was an increase in IL-6 in the immunosuppression phase. In patients

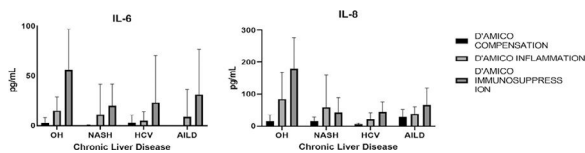
with HCV and AILD, increased serum levels of IL-8 and IL-6 were shown in decompensation stages (Figure 1).

Discussion: In this study, we demonstrated a significant increase in the pro-inflammatory cytokine profile in patients in the inflammation and immunosuppression phases. Fischer J et al. reported that a greater understanding of the mechanisms associated with immune dysfunction has led to the identification of possible therapeutic targets, with the intention of reducing the risk of infection and preventing decompensation events and disease progression.

Conclusion: This study demonstrated a significant increase in the pro-inflammatory cytokine profile (IL-8 and IL-6) as cirrhosis progresses. This is consistent with in the inflammation and immunosuppression phases, assessed by the Child-Pugh severity scales in stages B and C, D'amico from stages 3 to 5 and MELD> from 16 to 24 points and from 25 to 34 points. in the four etiologies included, being statistically significant.

The authors declare no conflict of interest.

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CONNECTIVE TISSUE GROWTH FACTOR (CTGF) AS A PROMOTER IN THE DEVELOPMENT OF FIBROSIS IN PATIENTS WITH PRIMARY BILIARY CHOLANGITIS

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Introduction and Objectives: Primary biliary cholangitis (PBC), an immune-mediated disease, is characterized by destroying the intrahepatic bile ducts, leading to progressive damage to the biliary tree, cholestasis, and development of progressive fibrosis leading to cirrhosis with all its complications. The development of fibrosis is multifactorial and includes connective tissue growth factor (CTGF). in a mouse model of cholestasis by bile duct ligation, the hepatic and serum increase in CTGF associated with the progression of fibrosis was demonstrated. Our goal was to determine the relationship between CTGF levels and their association with the development of fibrosis in patients with PBC.

Material and methods: Prospective, cross-sectional, and analytical study, including patients with PBC. The degree of fibrosis was determined by transient elastography (Fibroscan). Serum concentrations of FCTC-8pg/ml were quantified, for statistical analysis, the SPSS version 25.0 software was used; the medians (Q3, Q1) of CTGF, alkaline phosphatase, gamma-glutamyl-transpeptidase, and degree of fibrosis were compared with the Mann-Whitney U test with significance less than 0.05.

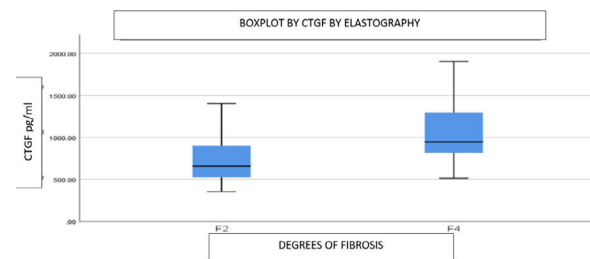
Results: We included 30 patients, 29 women (96.6%) and 1 man (3.4%), with a mean age of 55.5±12.4 years. Overexpression of CTGF protein was shown in 28 subjects (93.3%). Regarding the degree of fibrosis, all patients were categorized into one of two stages: Significant fibrosis (F2) and cirrhosis (F4). The F2 group had 11 patients with a median and standard deviation for CTGF of 915.9 and 522.9,

respectively; The F4 group had 19 patients who showed a median: 945.7 (1313.85-738.32); p:0.025. In relation to the differences between fibrosis levels and markers of alkaline phosphatase cholestasis, the median and interquartile ranges F2: 79 (180.60) F4: 169 (266-5.84) p: 0.066; GGT: F2: 1.51(7.7-1,04); F4: 1.2(2-1.92) p:0.746 In (Figure 1) The difference in medians of patients with different degrees of fibrosis and different concentration of CTGF is shown, confirming the association between peptide and the development and progression of fibrosis.

Discussion: According to the results obtained in patients with CBP and chronic cholestasis, the increase in CTGF showed significant differences between the degree of fibrosis and its levels; this could perhaps be interpreted as if it were an important factor for the development and progression of liver fibrosis, taking into account the antecedent of the initial study in mice with bile duct ligation and secondary cholestasis, where this factor was overexpressed at the hepatic and serum level in subjects with advanced fibrosis. It will be important to add more samples to this work and compare it with healthy controls to have better evidence.

Conclusions: Connective tissue growth factor (CTGF) probably participates directly in the processes of synthesis of extracellular matrix and therefore in the progression of fibrosis in subjects with primary biliary cholangitis, which makes it a possibility of a therapeutic target to develop in future studies.

The authors declare that there is no conflict of interest.



(Figure 1) The difference in medians of patients with different degrees of fibrosis and different concentration of CTGF is shown, confirming the association between peptide and the development and progression of fibrosis.

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Global real-world evidence of sofosbuvir/velpatasvir (SOF/VEL) as a highly effective treatment in underserved patient populations because of mental health disorders, incarceration or homelessness

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