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Introduction and Objectives: Diagnosis of liver disease (LD) is essential for the treatment and management of patients. The use of non-invasive methodologies is necessary despite the availability of direct-acting antivirals, some reports has been showed that treated patients can progress to cellular hepatocarcinoma (HCC). Continuous sampling that will evaluate liver tissue before and after treatment are essential for the prognosis of LD. Objective: To determine the sensitivity and specificity of insulin-like growth factor binding proteins (IGFBP) in the different stages of fibrosis in hepatitis C

Material and methods: A prospective, cross-sectional, observational study. The study included patients with CHC that were treatment naïve. The stages of fibrosis were classified as F0, 16 F1, F2, F3, or F4, according to international guidelines, through the FibroTest® and/or FibroScan® Patients with at-risk alcohol consumption (AUDIT>8), and without concordance between fibrosis diagnostic methods employed, and comorbidities were not included. Serum was obtained and multiple suspension array technology (Millipore®) was used to evaluated IGFBP-1,2, 3, 4, 5, 6, 7. Chi-square test, Mann-Whitney U test. Logistic regression models, odds ratios (ORs) and 5% confidence intervals were determined.

Results: A total of 128 patients diagnosed with CHC and 123 CT were included. Fibrosis stages were classified as follows: F0 (n=18), F1 (n=16), F2 (n=20), F3 (n=25), and F4 (n=48). IGFBP-1 to -7 showed an evident increment in patients mainly at F3 and F4. IGFBP-7 allows discriminate F3 vs F4 (72% sensibility, 62.5% specificity and cut of value of 2.74), whereas IGFBP-4 discriminates F3 vs F4 (83% sensibility, 68% specificity and cut of value of 14.68). P<0.001 was consider in statistical analysis.

Discussion: Although HCV treatment is available the progression from cirrhosis to HCC has been reported after clearance of HCV. Post-treatment studies evaluating the different stages of fibrosis should be performed. Therefore, the use of IGFBPs could be a tool in the continuous sampling previous and after treatment.

Conclusion: IGFBPs can be used as additional strategy for the diagnosis and discrimination of fibrosis stages in HCV.

Conflict of interest: The authors declare that there is no conflict of interest.

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LYMPHOCYTE PROFILE ON PATIENTS WITH CHRONIC AND ACUTE ALCOHOL CONSUMPTION

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Introduction and Objectives: Several mechanisms participate in the physiopathology of chronic alcohol consumption and Alcoholic Liver Disease (ALD), such as deregulation in the immune system.

Aim: To analyze the lymphocyte subpopulations from patients with chronic and acute alcohol consumption.

Material and methods: A Cross-sectional study that included: G1: Controls with alcohol consumption <10g/day (CT); G2: Alcoholism, without clinical or biochemical stigma of liver damage (OH); G3: Patients with cirrhosis by alcohol (CiOH) and G4: Patients with Alcoholic Hepatitis (AH). Determination of T-CD3, T-CD4, T-CD8, NK and NKT lymphocytes from peripheral blood was performed by flow cytometry. Statistical analysis was performed by U-Mann Whitney test, p<0.05 was considered significant.

Results: 570 participants were included, the mean of age was: 29.5±10.8, 31±12.6, 47.6± 7.7 y 41.2±9.2 years for CT, OH, CiOH and AH respectively (p<0.001). Alcohol consumption was higher in CiOH 240(320,120) and AH 320(480,160) (p<0.001, p<0.05). Liver function test showed alterations in patients with CiOH and AH, AST 49.5 (75.3,38) for CiOH and 155 (177,121) for AH (p<0.001, p<0.001); ALT 32.5 (47.3,24) in CiOH and 49 (75,35.3) in AH (p<0.001, p<0.001), whereas GGT was 91.5 (191.8, 48) for CiOH, and 224 (525.5, 104) for AH (p<0.001, p<0.001). Cell percentages are described in Table 1.

Data expressed as the median and quartiles (Q3-Q1). a) Alcoholism vs. Control; b) Cirrhosis vs. Control; c) Alcoholic Hepatitis vs. Control; d) Alcoholism vs. Cirrhosis; e) Alcoholism vs. Alcoholic Hepatitis; f) Cirrhosis vs. Alcoholic Hepatitis.

Discussion: Changes in the proportion of innate cells affect their ability to repair tissues, which can be exacerbated when damage is perpetuated and chronic inflammation is established. To compare CiOH vs. CT groups we found the suppression of adaptive response and increase in innate population. Furthermore, when CiOH was compared vs. OH increased CD4+ cells and decreased the cytotoxic population, which could be explained due to factors such as active alcohol consumption or advanced cirrhosis. In AH, the innate responses are suppressed compared to other groups. When we compare acute damage (AH) vs. alcoholism (OH) cytotoxic populations decrease, while CD4+ cells increase. However, during acute damage (AH) vs chronic damage (CiOH) increase T and CD8+ cells.

Conclusions: The immunological abnormalities that occur during alcoholism, cirrhosis and alcoholic hepatitis are different, the most significant changes were observed in CD4+, CD8+, NK and NKT cells promoting an imbalance that could be related to progression of liver damage.

The authors declare that there is no conflict of interest.

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Table 1
Lymphocytic profile in patients with different types of liver damage

	Control (n=300)	Alcoholism (n=102)	Cirrhosis (n=121)	Alcoholic Hepatitis (n=47)
T-Cells (CD3+) %	66.5 (71.7, 61.1)	62.3 (67.6, 56.2) a+	57.1 (66.5, 51.6) b*,d€	66.4 (78.3, 60) f€
Helper Cells CD4+ (%)	39.6 (45.4, 34.7)	35.1 (42.1, 30) a+	42 (47.7, 32.6) d+	47.4 (51.5, 34.7) e€
Cytotoxic Cells CD8+ (%)	21.2 (27, 17)	24.7 (30.8, 16.6)	14.1 (18.9, 8.8) b*,d*	18.9 (23.6,12.3) e€,f€
CD4+/CD8+ Cells (%)	1.87 (2.56, 1.37)	1.5 (2.2, 1) a+	2.7 (4.1, 2) b*, d*	2.7 (3.4, 1.7) e+
NK Cells (%)	11.1 (15.9, 8.4)	15.5 (20.9, 10.7) a*	13.2 (22.1, 8.1) b€	1.7 (12, 0.9) c+,e*,f*
NKT Cells (%)	1.7 (2.8, 1.1)	2.6 (4.5, 1.2) a+	1.4 (2, 0.7) d*	0.5 (1.1, 0.3) c+,e*,f*

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IL-10 Y TNF-α IN SERUM OF PATIENTS WITH CHRONIC HEPATITIS C AND HEPATIC DAMAGE CHRONIC AND ACUTE FOR ALCOHOL CONSUMPTION

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