

Disease; **n/a**, not available; **TE**, Transient elastography. a. Mann-Whitney U test. b. Pearson's Chi-squared test.

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P-48 EVALUATION OF IL-1 β AND IL-1RA IN PATIENTS WITH CHRONIC LIVER DISEASES

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Introduction and Objectives: IL-1 β is a proinflammatory key cytokine that participates in the progression of liver disease. Its antagonist IL-1RA mediates damage limitation; its increase is associated with positive effects on chronic liver diseases. This study aimed to evaluate the concentration of IL-1 β and IL-1RA in subjects with alcoholic liver disease (ALD), chronic hepatitis C (CHC) and non-alcoholic fatty liver disease (NAFLD).

Materials and Methods: A cross-sectional and multicenter study was carried out, which included alcoholic subjects (OH), alcoholic cirrhosis (CiOH) and alcoholic hepatitis (HA); patients with CHC and NAFLD were compared against subjects without criteria for alcohol drinking habits (CT). IL-1 β and IL-1RA were quantified by Multiplex-MERCK®. For statistical analysis, SPSS V.22 were used, Mann-Whitney U, $p < 0.05$; values expressed as mean \pm standard error.

Results: The groups included were: 18 (OH), 25 (CiOH), 14 (HA), 55 (CHC), 22 (NAFLD) and 81 (CT). IL-1 β results (pg/mL): 13.8 \pm 9.2, OH; 4.4 \pm 1.7, CiOH; 3.05 \pm 0.05, HA; 7.1 \pm 2.3, CHC; 5 \pm 2, NAFLD and 3.2 \pm 0.1, CT. With differences in HA vs. CHC. For IL-1RA (pg/mL) 83.5 \pm 30, OH; 100.4 \pm 53.5, CiOH; 85 \pm 38.3, HA; 74.4 \pm 2, CHC; 316 \pm 203, NAFLD and 13.02 \pm 4.4, CT. With differences in CHC and NAFLD vs. CT and CiOH vs. CHC.

Conclusions: IL-1 β was 2.3 times increased in HA/CHC, which highlights the effect on exacerbating the inflammatory response in acute over chronic alcohol damage; IL-1RA that inhibits the activities of IL-1 β increase may have protective effects on liver injury. IL-1RA is a protein that limits inflammation in liver disease, especially in non-alcoholic fatty liver disease, alcoholic cirrhosis, and chronic hepatitis C.

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P-49 CONCENTRATION OF IL-12 AND CXCL-10 IN CHRONIC LIVER DISEASES

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Introduction and Objectives: Chronic liver diseases are characterized by persistent inflammation related to high production of cytokines such as IL-12 and chemokine CXCL-10/IP-10 that attract activated Th1 lymphocytes that increase the production of IFN- γ and TNF- α , perpetuating the inflammatory cascade. This study aimed to compare serum levels of IL-12 and CXCL-10 in different etiologies of liver disease.

Materials and Methods: A cross-sectional and multicenter study was carried out, including subjects with alcoholism according to criteria WHO, without (OH) and with liver injury (cirrhosis, CiOH) and (Alcoholic Hepatitis, HA); non-alcoholic fatty liver (NAFLD) and chronic Hepatitis C (CHC), diagnosed by clinical, biochemical data. They were compared with control subjects (CT). For determination of IL-12 and CXCL-10 with Multiplex®-MERCK®. Statistical analysis by SPSS V.22 using U de Mann Whitney, $p < 0.05$; values expressed as mean \pm standard error.

Results: Included 20 subjects with NAFLD, 78 CHC, 14 HA, 20 CiOH, 15 OH y 60 CT. IL-12 was found elevated in OH, HA, CHC vs. CT in OH vs. HCC y HGNA ($p \leq 0.05$). CXCL-10 was found elevated in CiOH, HA, and CHC vs. CT ($p \leq 0.050$).

Conclusions: The IL-12 showed elevated levels in subjects with alcohol consumption and CHC vs. CT that activates other cell types involved in inflammation. CXCL-10 is induced by IFN- γ and was found elevated in CiOH, HA and CHC, exerting their biological effects through CXCR3, including activation of peripheral immune cells and apoptosis. The ratio of IL-12/CXCL-10 in OH increased 4.6 times, ratifying the participation in chronic and continual inflammatory response by alcohol consumption. IL-12 and CXCL-10 have an important role in alcohol-induced liver disease, confirming their contribution to inflammation, being evident in CXCL-10 in advanced stages of the disease by stimulating and favoring the migration of immune cells to the damage sites.

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P- 50 SEROPREVALENCE OF HEPATITIS E VIRUS IN HIV-INFECTED PATIENTS FROM ROSARIO, SANTA FE

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