Participation of the immune response and oxidative stress in alcoholism and liver cirrhosis due to alcohol

Leonardo S. Juárez-Chávez[1],
José L. Pérez-Hernández[1],
Abigail Hernández-Barragán[2], Zaira Medina-Avila[2],
Marisela Hernandez-Santillan[2],
Moisés Martínez-Castillo[2],
Fátima Higuera-De la Tijera[1],
Gabriela Gutiérrez-Reyes[2]

[1] *Gastroenterology Department, General Hospital of México Dr. Eduardo Liceaga, Mexico City, Mexico*
[2] *Liver, Pancreas, and Motility Laboratory (HIPAM), Experimental Medicine Research Unit, Faculty of Medicine, General Hospital of Mexico, Dr. Eduardo Liceaga, Mexico, UNAM, Mexico City, Mexico*

Introduction and Objectives: The spectrum of alcoholic liver disease (ALD) includes steatosis, steatohepatitis, alcoholic hepatitis, fibrosis, cirrhosis, and hepatocellular carcinoma. The pathophysiology of liver damage due to chronic alcohol consumption is complex. It is partly a result of reactive oxygen species (ROS) and reactive nitrogen species (RNS), products of oxidative stress, which is one of the mechanisms that will activate the immune system creating a pro-inflammatory state, increasing the levels of several cytokines (TNF-α, IL-1, IL-6, IL-8, MCP-1 and TGF-1).

Objective: To study oxidative stress and the production of proinflammatory cytokines that intervene in the different stages of liver damage due to alcohol (alcoholism, alcohol-related liver cirrhosis, and alcoholic hepatitis).

Material and Patients: A cross-sectional, prospective, and analytical study that included patients from the Gastroenterology service and donors from the Blood Bank. Patients at different stages of the disease and a control group of healthy subjects (blood bank donors) were included. They were divided into 4 groups: Alcoholism (OH), alcoholic liver cirrhosis (CiOH), alcoholic hepatitis (HA), and healthy controls (CT). From each participant, 20 ml of peripheral blood was obtained for the relevant determinations. Normally distributed data were obtained and ANOVA and orthogonal analyses were performed to detect group differences. A U-Mann Whitney test was used. $P < 0.05$ was taken as a significant difference.

Results: 236 subjects were included: 67 patients in OH group; 40 patients with CiOH; 39 patients with Alcoholic Hepatitis (AH), and 90 subjects CT. The gender distribution in patients with ALD (CiOH, and HA) was 77.5% men and 22.5% women. The average alcohol consumption was 376.6±151.6 grams. The CiOH and HA groups presented alterations in platelets, bilirubin, and cytolysis markers at the expense of AST with a significant difference ($p<0.05$). Regarding oxidative stress, lipoperoxidation was greater in patients with chronic disease (CiOH) and protein damage (protein carbonyls) was greater in HA with $p<0.05$. Regarding cytokines and chemokines, TNF-α presented a higher level in CiOH and HA ($p<0.5$), IL-6 presented an elevation in CiOH and HA ($p<0.05$); IL-10 was elevated in OH, CiOH and HA, MCP-1 and IL-8 showed greater elevation in HA, $p<0.05$.

Conclusions: Oxidative stress and the elevation of proinflammatory cytokines and chemokines have different behaviors in the various stages of alcohol liver disease, which influences the progression and prognosis of the disease. These findings could be considered as possible therapeutic targets.

Ethical statement: Each patient has explained the research in detail. Those who decided to participate signed the informed consent letter.

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.

Table 1
Demographic and biochemical characterization of the study groups.

	OH (67)	CiOH (40)	AH (39)	CT (90	P*
Gener (n) (%)					
Female	21(31)	2(5)	2(5)	28(26)	
Male	46 (69)	38(95)	37(95)	62(74)	b*, c*
Age	46 ±10	47±8	38±7	38±10	a*, b*, e*
BMI	27±7	27±7	27±5	28±4	
gr OH/day	320±125	350±130	460±200	1.3±2	a*, b*, c*
Hb (gr/dL)	14±4	12±3	11.2±0.5	17±1	a*, b*, c*
Platelets (1000 × 3)	205±95	138±90	93±36	268±65	b*, c*
BT (mg/dL)	2.3±1.1	3±0.4	18.8±2.4	0.8±0.03	a*, b*, c*, f*
BD (mg/dL)	1.4±0.5	1.8±0.2	8.6±1.9	0.7±0.03	a*, b*, c*
AST (U/L)	39±10	63±6	142±17.9	30±1	b*, c*, f*
ALT (U/L)	31±5	37±3	57.1±5.4	28±2	b*, c*, f*
GGT (U/L)	70.8±21.9	206±48.8	254±74.8	29.6±2.8	a*, b*, c*, e*

(OH= Alcoholic, CiOH= alcohol cirrhosis, AH= Alcoholic hepatitis, CT= control). Statistical differences: **a**. OH vs CT, **b**. CiOH vs CT, **c**. HA vs CT, **d**. OH vs CiOH, **e**. OH vs HA y **f**. CiOH vs HA. *p < 0.05

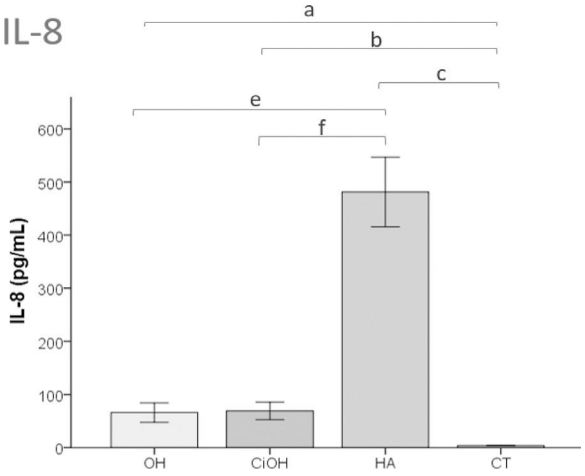

Figure 1. Interleukin IL-8.
(OH= Alcoholic, CiOH= alcohol cirrhosis, HA= Alcoholic hepatitis, CT= control). Statistical differences: a. OH vs CT, b. CiOH vs CT, c. HA vs CT, d. OH vs CiOH, e. OH vs HA y f. CiOH vs HA. *p < 0.05

https://doi.org/10.1016/j.aohep.2025.101861

Patterns of antimicrobial resistance and susceptibility in patients with spontaneous bacterial peritonitis at the General Hospital of Mexico "Dr. Eduardo Liceaga"

Gabriela Rangel-Zavala, Paloma M. Diego-Salazar, Karina Cazarín-Chávez, José L. Pérez-Hernández, Fatima Higuera-de-de-Tijera

Gastroenterology and Hepatology Service at the General Hospital of Mexico "Dr. Eduardo Liceaga", Mexico

Introduction and Objectives: Spontaneous bacterial peritonitis (SBP) is a serious complication in cirrhotic patients, with high morbidity and mortality. Antimicrobial resistance complicates treatment and increases complications. This study aims to determine resistance patterns in microorganisms in SBP to improve treatment efficacy.

Materials and Patients: A descriptive, observational, and retrospective study on patterns of antimicrobial resistance and