Material and methods: CD-1 mice were treated with α-naphthyl isothiocyanate (ANIT, 60 μg / kg, i.g.) for 48 h. After 24 h of ANIT treatment, HGF (10 μg / kg, i.v.) was administered. Mice were throughout treatment in metabolic cages. Urine samples were collected from the last 12 h of treatment. After 48 h, mice were sacrificed, blood and tissue were obtained. Liver function tests (ALP, GGT and bile salts), analysis of bile transporter expression by qRT-PCR, serum and urine creatinine content, albuminuria and HSP27 in urine, and H-E staining were performed, ROS content was addressed in kidney tissue.

Results: Cholestasis induced by ANIT was corroborated by the increase of bile salts in the liver and serum, and the increase in GGT and ALP. Interestingly, we found renal dysfunction determined by the increase in serum creatinine, and decrease in its clearance, as well as proteinuria and the increase in urine HSP72. Treatment with HGF reduced to control values the markers of liver and kidney damage, significantly improving renal histology. The protection mechanism was closely associated with the control of oxidative damage. In conclusion, HGF is presented as a therapeutic intervention point in cholestasis-mediated renal damage, counteracting the oxidative damage.

CONACYT: CB-A1-S-38154 y CB-252942.

Conflicts of interest: The authors have no conflicts of interest to declare.

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Prevalence of acute kidney injury in hospitalized patients with cirrhosis and their transition to chronic kidney disease

V. Rodríguez-Granados, I.D. Aranda-Sánchez, G.L. Ojeda-Aguilar, C.M. Del Real-Calzada

Gastroenterology, Hospital de Especialidades Centro Médico Nacional La Raza, Ciudad de México, México

Background and aim: Acute Kidney Injury (AKI) is frequent in patients with cirrhosis and is associated with a poor prognosis. LRA can lead to Chronic Kidney Disease (CKD). The objective of the study was to determine the prevalence of AKI in hospitalized patients with decompensated cirrhosis, as well as the frequency of CKD after an episode of AKI.

Material and methods: Retrospective, descriptive and observational study. Information was obtained from 146 patients hospitalized in the Gastroenterology department of the Centro Medico Nacional La Raza in the period from January-December 2019. They included patients who met the LRA criteria. Information on the evolution of patients after hospital progress will be collected from the electronic medical system. The results were analyzed with recommended and central frequency measures to obtain percentages, means and average. 3-month survival was estimated using the Kaplan-Meier method and compared using the log-rank test. The odds ratio (OR) of the different factors related to the development of CKD was determined.

Results: Forty patients were excluded, of the remaining 106, 46 (43%) presented with AKI, with a median age of 58 years (19-75 years), 27 (58.6%) women and 19 (41.3%) men. 14 patients (30.4%) present some comorbidity, of which arterial hypertension and diabetes stand out. During hospitalization, all were treated with isotonic solutions and 12 received albumin for 2 days. 15 (32.6%) obtained a total response to treatment and 9 (19.5%) a partial response. 10 patients (21.7%) developed CKD. The severity of liver disease from high MELD predicted an increased risk of developing CKD. Grade 2 or 3 ascites, hypoalbuminemia, comorbidities, and the degree of AKI are associated with an increased risk of CKD.

Conclusions: The prevalence of both AKI and CKD is high in patients with decompensated cirrhosis. Most of the AKI episodes in patients with cirrhosis are reversible, however, it constitutes a risk factor for the transition to CKD, influencing the evolution of the disease.

Conflicts of interest: The authors have no conflicts of interest to declare.

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Correlation of serum ferritin concentrations with laboratory and demographic paramters and its alteraration by different clinical conditions in patients with liver disease

R. Tijerina-Márquez, O.H. Mendoza-Hernández, E. Cienfuegos-Pecina, J.A. De la Torre-Hernández, G.R. Govea-Torres, L. Muñoz-Espinosa, P. Cordero-Pérez

Hepatology Center, University Hospital "Dr. José Eleuterio González", Autonomous University of Nuevo León, Mexico

Background and aim: Ferritin is a protein whose main function is to store iron. It is documented that in liver diseases, proinflammatory states and metabolic syndrome (MS) its serum levels increase. This study's objective was to describe serum ferritin levels in a population with liver disease; evaluated at a hepatology center in northeast Mexico and its correlation with biochemical markers and comorbidities.

Material and methods: A retrospective study was carried out on patients from the Hepatology Center of the University Hospital "Dr. José Eleuterio González" from 2015 to January 2020, including 165 subjects (80 men and 85 women) aged 17-80 years. The following laboratory test results were analyzed: Serum ferritin, blood chemistry, blood count, lipid profile, liver function tests, coagulation

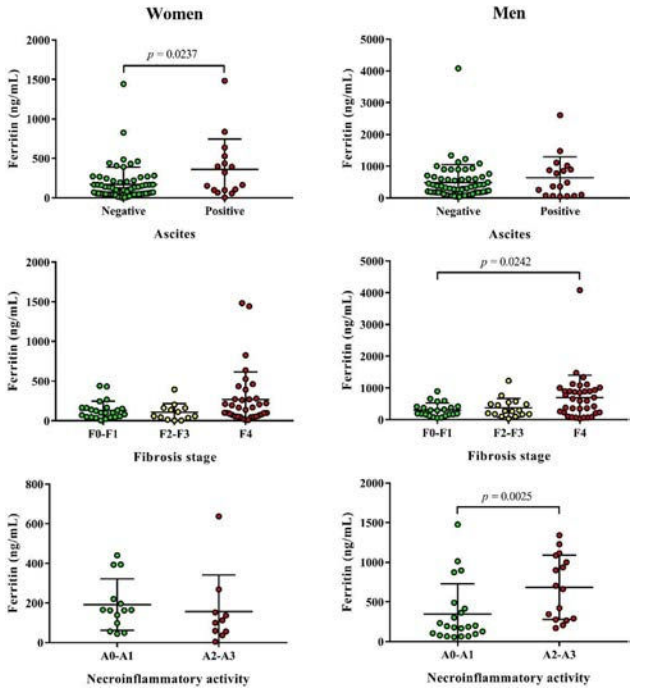

Figure 1. Ferritin levels between women and men with and without ascites, with different fibrosis stage and necroinflammatory activity.

times, serology for Hepatitis B, C and autoimmune and Fibromax. Clinical parameters such as body mass index (BMI), type II diabetes mellitus (DMII), systemic arterial hypertension (SAH), presence of ascites, alcohol consumption (gr / week) and endoscopic findings (esophageal varices) were also analyzed.

Results: Significative difference was observed in serum ferritin levels between men and women [353.0 ng/mL (170.5–747.5 ng/mL) vs 108.3 ng/mL (55.8–253.5 ng/mL), $p < 0.0001$], as well as in serum ferritin levels between women with and without ascites, in men with different fibrosis stage (FibroTest) and necroinflammatory activity (ActiTest) (Figure 1). A poor but significant correlation was observed between serum ferritin and age, erythrocytes, MCV, MCH, uric acid, direct bilirubin, albumin and HDL cholesterol in women and alcohol consumption, uric acid, ALT and AST in men. All other evaluated clinical parameters and biomarkers showed no significant difference.

Conclusions: An association was observed between the degree of fibrosis and serum ferritin and necroinflammatory activity in men, as well as between ferritin and ascites in women. A poor correlation was observed between serum ferritin levels and the analyzed chemical biomarkers.

Conflicts of interest: The authors have no conflicts of interest to declare.

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Moringa oleífera decreases insulin resistance, novo lipogenesis and modifies the expression of mirnas in a non-alcoholic esteatohepatitis model

A. Monraz-Méndez [1], R. Escutia-Gutiérrez [1], S. Rodríguez-Sanabria [1], L. Sánchez-Orozco [1], J. Armendáriz-Borunda [1,2], A. Sandoval-Rodríguez [1]

[1] *Instituto de Biología Molecular en Medicina y Terapia Génica, Centro Universitario de Ciencias en la Salud, Universidad de Guadalajara, Guadalajara, Jalisco, México*
[2] *Tecnológico de Monterrey, Campus Guadalajara, Guadalajara, Jalisco, México*

Background and aim: In Mexico there is a high prevalence of non-alcoholic steatohepatitis (NASH) and liver diseases are the fourth leading cause of death. NASH is characterized by hepatocyte ballooning, inflammation, and steatosis. Moringa Oleifera (MO) extracts have been shown to have hypoglycemic, anti-inflammatory and antioxidant effects. The aim was to evaluate in a NASH model the effect of the aqueous extract of MO the gene and protein expression of molecules involved in steatosis and liver inflammation and on miRNAs involved in the development of NASH.

Material and methods: Male C57BL / 6J mice were fed a high fat diet (HF, 60% lipid, 42gr / L sugar in water) for 16 weeks. The administered dose of the MO extract was 300 and 500 mg / Kg / day from week 9 to 16. The serum levels of adipokines were measured, the HOMA-IR was calculated; In the liver miR-21a-5p, miR-103-3p, miR-34a-5p and IL1β, IL-6, TNFα, SREBP1, FASN and DAGT2 were evaluated by qRT-PCR and SREBP1 by Western Blot. The transcriptome was evaluated by microarrays. Inflammation, reactivity to αSMA and fibrosis were analyzed in histological sections. Quantitative variables were analyzed with ANOVA, Tukey for parametric data, Mann-Whitney U for non-parametric data. Approved by the CUCS Ethics, Research and Biosafety Committees: 1937.

Results: Moringa treatment reduced serum insulin, PAI-1, leptin, and resistin levels. In liver: IL1β, IL6, TNFα, SREBP1c, FAS, and DAGT2 mRNAs decreased; SREBP1 protein decreased. Expression of mir-21a, mir-103, and mir-34a were reduced. In the transcriptome, the mRNAs involved in the response to DNA damage and stress of the endoplasmic reticulum, lipid biosynthesis, and extracellular matrix synthesis were underexpressed. In liver histologies, the number of inflammatory nodules and the presence of αSMA and fibrosis decreased.

Conclusions: MO supplementation decreased serum adipokine levels; as well as the mRNAs of proinflammatory cytokines and lipogenic genes in liver. The histological quantification of MEC, collagen, inflammatory nodules and αSMA decreased; miRNAs evaluated were modified. Moringa extract showed anti-inflammatory, antifibrogenic and antilipogenic effect in a NASH model.

Conflicts of interest: The authors have no conflicts of interest to declare.

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Prolonged-release pirfenidone decreases hepatic miRNAs expression in a NAFLD/NASH experimental model

R. Escutia-Gutiérrez [1], J.S. Rodríguez-Sanabria [1], C.A. Monraz-Méndez [1], A. Santos-García [2], J. Armendáriz-Borunda [1,2], A. Sandoval-Rodríguez [1]

[1] *Instituto de Biología molecular en Medicina y Terapia génica, Centro Universitario de Ciencias de la Salud, Universidad de Guadalajara, México*
[2] *Tecnológico de Monterrey, Campus Guadalajara, México*

Background and aim: Nonalcoholic steatohepatitis (NASH) is featured by lipid accumulation, inflammation, and fibrosis. miRNAs are small non-coding RNAs that participate in post-transcriptional genetic regulations and are involved in various pathologies such as NASH. The drug pirfenidone is an antifibrotic, anti-inflammatory and antioxidant agent. Aim: To evaluate the effect of prolonged-release pirfenidone on histological parameters, activation of hepatic stellar cells, expression of hepatic miRNAs and target genes in an experimental model of NAFLD/NASH.

Material and methods: Male C57BL/6J mice were fed a high fat diet (HFD, 60% lipids, 42gr/L sugar in water) for 16 weeks. Prolonged-release pirfenidone (~300 mg/kg/d, PR-PFD) was administered in food from the eighth week to the end of the protocol. α-SMA immunohistochemistry and hematoxylin-eosin, Masson's trichrome and Sirius red staining were made. Hepatic expression of miR-21a-5p, miR-103-3p, miR-34a-5p and IL-1β, TNFα, COL1A1, and SREBP1 genes was determined by qRT-PCR and the transcriptome by microarrays. Statistical significance was determined for parametric data with one-way analysis of variance and Tukey's or Bonferroni post hoc test, and Kruskal-Wallis and Mann-Whitney *U* test for nonparametric data (Graph Prism 6.0). Ethics Committee registration number: CI00518.

Results: Animals treated with PR-PFD have a decrease in inflammatory nodules, macrosteatosis, fibrosis, collagen and activation of hepatic stellar cells. PR-PFD reduced hepatic expression of miR-21a-5p, miR-34a-5p and miR-103-3p expression showed a tendency of decrease compared to HFD group. PR-PFD decreased IL-1β, TNFα, COL1A1, and SREBP1 expression. Transcriptome analysis showed that 36 genes that participate in lipid transport and antiox-