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The fructose enhances the HCC progression in mice under a high intake of fructose in dieT

L. Chávez-Rodríguez [1,2], A. Simoni-Nieves [1,2], A. Escobedo-Calvario [1,2], M. Gerardo-Ramírez [1,2], S. Salas-Silva [1,2], R. Miranda-Labra [1], V. Souza [1], L. Bucio [1], M.C. Gutiérrez-Ruiz [1], L.E. Gomez-Quiroz [1]

[1] *Posgrado en Biología Experimental, Universidad Autónoma Metropolitana Iztapalapa, México*
[2] *Departamento de Ciencias de la Salud, Universidad Autónoma Metropolitana Iztapalapa, México*

Background and aim: Hepatocellular carcinoma (HCC) is the fourth cause of cancer-related death and its incidence has been increasing in both men and women. One of the main concerns has been the consumption of hypercaloric diets mainly rich in carbohydrates such as fructose. High fructose diet is related to the development of Non-Alcoholic Fatty Liver Disease (NAFLD) and the progression of HCC since it potentiates the lipogenic pathway and the accumulation of lipids. The aim of the study is to determine the effect of a high fructose diet on the progression of HCC, induced by DEN, in C57Bl/6J mice strain.

Materials and methods: We used C57Bl/6J mice strain (both sex) with a high Fructose diet (Fru)(33% of Fructose in the drinking water, *ad libitum*). Fru supplementation started with 15 days old mice, two days after DEN was injected (10 μg/Kg, i.p) and the treatment was ended 8 months later to evaluate the role of fructose in tumor progression by histological and biochemical tests. The protocol was approved by the UAM ethics commission.

Results: The major number of tumors were found in the Fructose + DEN (FD) mice group *vs*. only DEN (CWD) mice group. Triglyceride levels (TG) was evaluated in the serum with no detectable values; however, in the liver tissue the FD group showed significantly higher TG content. On the contrary, the Cholesterol (CHO) levels were significantly higher in the serum of dietary fructose group and had no differences in the tissue. The protein content in tissue followed the same observed pattern, since significance was only found in Fatty acid synthase (Fasn) with a higher protein content in the groups with dietary Fructose. Curiously, we noticed tumors in the lungs. Conclusion. The data strongly suggests that the high consumption of Fru in the diet induces effects in liver tumor promotion, in a mechanism dependent on FASN and independent of CHO. High consumption of Fru should be considered as a liver toxic factor.

This work has been partially founded by Conacyt: Fronteras de la Ciencia 1320 and by the UAM.

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

https://doi.org/10.1016/j.aohep.2020.08.006

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Gallbladder adenomyomatosis

J. Aquino-Matus, M. Enriquez-Pineda, A. Pereira-Garcia, S. Ornelas-Arroyo

Medica Sur Clinical Foundation, Mexico

Background and aim: Adenomatous hyperplasia of the gallbladder or adenomyomatosis is a benign neoplasia characterized by epithelium hyperplasia with invaginations into the subserose forming intramural diverticula (Rokitansky-Aschoff sinuses). It is reported in 1 to 8.5% of cholecystectomies and 7% of autopsies. It has been associated with cholelithiasis in 80% of cases and may have an asymptomatic course or present with biliary cholic. According to its site it can be localized, annular, diffuse or segmental; the later associated with cancer in 3.2%.

Material and methods: Retrospective review of medical records of patients with pathology study diagnosis of adenomyomatosis from January 1st, 2015 through December 31st, 2019.

Results: Twenty-four cases were found, with 58.3% of women and mean age of 51 years. Elective cholecystectomy was found in 26% of cases. Most frequent symptoms were abdominal pain, nausea and, vomit with 75%, 41.7%, and 33.3%, respectively. Duration of symptoms was less than 24 hours in 21.1%, and 7 days to 3 months in 57.9% of cases. Smoking was reported in 58.3%, alcohol consumption in 12.5% and dyslipidemia in 20.8% of cases. Murphy's sign was reported in 37.5% and the most frequent clinical diagnosis was acute cholecystitis in 66.7% of cases. Mean alkaline phosphatase was 105.6 ± 76.0 UI/L and mean gamma-glutamyl transpeptidase was 113.2 ± 161.3 UI/L. In abdominal ultrasound, the gallbladder had a thin wall in 50%, thick wall in 8.3%, polyps in 20.8% and stones in 54.2% of cases. In pathology studies, mean thickness of adenomyomatosis was 90.5 mm and the location were localized (fundus) in 58.3%, diffuse in 20.8% and annular in 4.2% of cases.

Conclusions: Female sex (estrogens), chronic inflammation (cholecystitis) and cholelithiasis are the few associated factors for the development of adenomyomatosis. Most ultrasound findings are non-specific and, therefore, presurgical diagnosis is difficult. In most of the cases, the diagnosis of adenomyomatosis was an incidental finding associated with acute cholecystitis.

Conflicts of interest: The authors have no conflicts of interest to declare. Funding was sponsored by the authors.

https://doi.org/10.1016/j.aohep.2020.08.007

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HGF induces a protective response in a preclinical model of nephropathy induced by acute cholestasis

J. Lopez-Ramirez [1,3], E.S. Salas-Silva [1,2,3], J. Barrera-Chimal [2], A. Simoni-Nieves [1,2,3], M.C. Gutiérrez-Ruiz [1,2], V. Souza [1,2], R.U. Miranda-Labra [1,2], L.E. Gómez-Quiroz [1,2], L. Bucio-Ortiz [1,2]

[1] *Departamento de Ciencias de la Salud, CBS Universidad Autónoma Metropolitana Iztapalapa, México*
[2] *Unidad de Medicina Translacional, Instituto de Investigaciones Biomédicas, UNAM/Instituto Nacional de Cardiología Ignacio Chávez, México*
[3] *Posgrado en Biología Experimental, DCBS, Universidad Autónoma Metropolitana Iztapalapa, Ciudad de México, México*

Background and aim: The relationship between the liver and the kidneys in some hepatic diseases is well known. Hepatorenal syndrome usually occurs in chronic damage but has also been observed in the acute one. Therapeutic approaches remain limited and poorly optimized, especially to address the commitment of both organs. HGF induces protection in various organs, but its effects are unknown in a scenario of multi-organ compromise, as in the case of hepatorenal syndrome or colemic nephropathy. The aim of this investigation was to determine the mechanism induced by HGF to counteract liver and kidney damage in a preclinical model of systemic damage induced by intrahepatic cholestasis in a setting of colemic nephropathy.

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.

https://doi.org/10.1016/j.aohep.2020.08.008

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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.

https://doi.org/10.1016/j.aohep.2020.08.009

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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

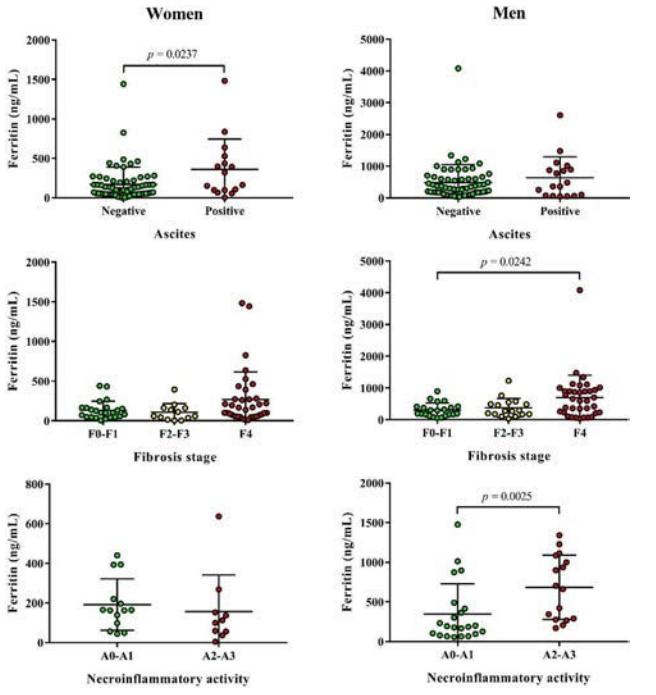

Figure 1. Ferritin levels between women and men with and without ascites, with different fibrosis stage and necroinflammatory activity.