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

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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.

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Gallbladder adenomyomatosis

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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.

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

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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.