

analyze the known RFs for CKD and progression to ESRD in patients with HCV and those specific to this population.

Materials and Patients: A prospective cohort study was conducted to identify the RFs for kidney damage and progression to CKD in a cohort with chronically infected HCV. The known RFs were analyzed: age over 65 years, diabetes, essential hypertension as the main RFs, in addition to obesity and RFs related to HCV infection prevalent in this population, such as blood transfusion, sexual promiscuity, intravenous drug users (IVDU). CKD was determined when functional alterations of the kidney were found for more than 3 months. The estimation of glomerular filtration rate (eGFR) was performed with the renal function calculator of the Spanish Society of Nephrology that uses the corrected Cockcroft-Gault formula where $<60 \text{ mL/min/1.73m}^2$ is considered CKD. The normal range of eGFR is $90\text{--}100 \text{ mL/min/1.73m}^2$, considering hyperfiltration above this. Diabetes and hypertension, transfusions, IVDU were self-reported by the patient for sexual promiscuity; the definition of the World Health Organization was considered, determining it when one has more than two sexual partners in less than 6 months; obesity was determined with the body mass index.

Results: Of 130 with chronic HCV infection, we found 51% were men with a mean age of 54 years. Among the known RFs, we identified age >65 in 21%, with diabetes at 26% and essential hypertension at 27%; among those associated with this population, 100% had chronic infection with HCV, a history of blood transfusion and blood products in 45%, IVDU in 25%, with obesity in 26%. About the different stages of CKD, we find 60% of the population in hyperfiltration ranges with an eGFR $>100 \text{ mL/min/1.73m}^2$. Hyperfiltration was associated first with obesity, in 70% of obese people, followed by 47 and 46% with diabetes and hypertension, respectively, in 32% with age >65 it is noteworthy that more than half of the patients with a history of transfusion in the IVDU, 59% and 54% had this finding. In addition, 21% of the total population evaluated was in stage 2 with an eGFR between $60\text{--}89 \text{ mL/min/1.73m}^2$. Only 8% had an eGFR in normal ranges between $90\text{--}100 \text{ mL/min/1.73m}^2$.

Conclusions: HCV is recognized as an independent RF for the development and progression to CKD; the intentional search for known RFs in this population will help reduce the progression to ESKD. The finding in this study of hyperfiltration is a little-explored fact, which deserves further study.

Ethical statement: Approval was obtained from the ethics and research committee of our hospital.

Declaration of interests: None.

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Effect of Alternate day fasting over Metabolic dysfunction-associated steatohepatitis in adult offspring of dams exposed to cafeteria diet during pregnancy and lactation.

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Introductions and Objectives: Metabolic dysfunction-associated steatohepatitis (MASH) is a liver disease characterized by lipid accumulation and inflammation that can be exacerbated by cafeteria diets (CAF) exposition during pregnancy and lactation, whereas Alternate

day fasting (ADF) improves metabolic parameters. Evaluate the effect of ADF and CAF maternal programming on MASH-associated markers in the offspring.

Materials and Patients: To assess the effect of maternal programming, we elaborated a mice model using 8-week C57BL6 females exposed to a CAF (cafeteria) diet (39% carbs, 49% fats, 12% proteins and sodium 231.8 mg) during 3 weeks of mating, 3 weeks of gestation and 3 weeks of lactation. For maternal programming control, we fed females with a Chow or control diet (57% carbs, 13% fats, 30% proteins and sodium 105 mg) during 3 weeks of mating, 3 weeks of gestation and 3 weeks of lactation. After weaning, the offspring were fed a control diet until they were 8 weeks old. They were then divided into four groups (Control $n=8$, Control + ADF $n=8$, CAF $n=8$, CAF + ADF $n=8$) and an alternate day Fasting (ADF) protocol was initiated for 5 weeks. At the end of the fasting protocol, plasma samples were taken and beta-hydroxybutyrate (BHB) concentration was measured; in addition, samples of the left lateral lobe of the liver were taken at slaughter to evaluate by qPCR the effect of intermittent fasting on the expression of metabolic function markers involved during MASH: fibrosis (TGF β , Col1a1), steatosis (PLIN2, ApoB100, Mylcd, PPAR α) and inflammation (Mcp-1).

Results: Groups treated with ADF showed an increase in plasma BHB concentration of $400 \mu\text{mol}$ compared to non-fasted groups. However, no significant difference was found between the control + ADF and CAF + ADF groups, so no effect of maternal programming with CAF diet on BHB production was observed. Additionally, the relative expression of mRNA from fibrosis-associated markers such as Col1a1 showed an 84% decrease in the CAF maternal programming model, 80% in the Control + ADF group and 88% in the CAF + ADF model with respect to control. Levels of mRNA-Plin2, involved in lipid droplet formation, decreased by 57% in the CAF group, 48% in Control + ADF and 79% in CAF + ADF. On the other hand, mRNA-Mcp-1 levels (chemokine) showed a decrease of 14.36% in CAF, 46.42% in Control + ADF and 62.68% in CAF + ADF with respect to control.

Conclusions: The model of alternate-day fasting (ADF) showed an increased plasma BHB, but we did not observe a maternal programming effect on the concentration of betahydroxybutyrate. Interestingly, maternal programming and ADF reduce the expression of MASH-associated markers involved in fibrosis, lipid droplet formation and inflammation in this mouse model.

Ethical statements: This project was authorized by the ethics committee of the Universidad Autónoma de Nuevo León with the registration number B120-00,004.

Declaration of interest: None.

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Impact on survival of decompensated liver cirrhosis and large volume paracentesis: a retrospective cohort

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Introduction and Objectives: Ascites is the most common complication of cirrhosis. Its presence represents a 40% mortality at 2 years. The objective of this study was to determine survival in patients with decompensated liver cirrhosis due to ascites undergoing large-volume paracentesis.

Materials and Patients: A retrospective, cross-sectional, observational, analytical study was conducted. Patients with liver cirrhosis

over 18 years of age of both sexes, of any etiology, treated at Centro Médico Nacional 20 de Noviembre between January 2013 and June 2023, who underwent large volume paracentesis, were selected and matched 2:1 with controls who did not require high volume paracentesis, adjusted for disease severity, age, sex, and Child-Pugh stage. Exclusion criteria were pregnancy or lactation, under 18 years of age, and ascites of a different origin than chronic liver disease. The data was extracted from clinical records.

Results: A total of 226 patients were analyzed, 61.9% women (n=140) and 38.1% men (n=86). The average age was 64.28 years (SD=13.33). The minimum age was 19 years and maximum was 91 years. The most frequent etiology was hepatic steatosis in 34.07% (n=77), followed by hepatitis C in 19.91% (n=45), alcoholism in 12.38% (n=28), autoimmune hepatitis in 10.17% (n=23). The distribution of patients by Child-Pugh classification was B in 69% (n=156) and C in 31% (n=70). The average MELD-NA score was 16.93 (SD=7.10). The main comorbidities were 36.7% (n=83) type 2 diabetes mellitus, 24.8% (n=56) systemic arterial hypertension, 15% (n=34) chronic kidney disease, and 16.4% (n=37) obesity.

Out the 226 patients with liver cirrhosis with ascites, 33.2% (n=75) underwent large volume paracentesis while 66.8% (n=151) underwent paracentesis less than 5 liters. The mortality of patients undergoing large volume paracentesis was 32% compared to 20.5% RR 1.55, IC 95% (0.98-2.45) of patients who did not. In bivariate analysis by sex, there were no statistically significant differences in mortality. Stratified analysis by nutritional status with body mass index did not show differences in mortality in patients undergoing large volume paracentesis.

Conclusions: No statistically significant differences in mortality were observed between patients undergoing large volume paracentesis and those who did not. It is important to consider that factors other than paracentesis volume may influence patient survival.

Ethical statement: This study adheres to ethical principles in clinical research involving human subjects and presents no risk to the population under investigation as only information obtained from clinical records will be evaluated.

Declaration of interests: None.

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Table 1
Demographic Characteristics

	LARGE VOLUME PARACENTESIS (n = 75)	NO LARGE VOLUME PARACENTESIS (n = 151)	P VALUE
AGE, MEAN	63.15	64.85	0.35
FEMALE (%)	41 (54.66)	99 (65.56)	0.11
MALE (%)	34 (45.33)	52 (33.77)	0.11
CHILD-PUGH (%)			
B	44 (58.66)	112 (74.17)	0.01
C	31 (41.33)	39 (25.82)	
MELD-NA, MEAN	19.92	15.45	0.00
HEPATOPATHY ETIOLOGY			
HEPATIC STEATOSIS (%)	24 (32)	53 (35.09)	0.04
HEPATITIS C INFECTION (%)	9 (12)	36 (23.84)	0.04
ALCOHOL (%)	11 (14.66)	17 (11.25)	0.04
HEPATITIS B INFECTION (%)	0 (0)	2 (1.32)	0.04
AUTOIMMUNE HEPATITIS (%)	9 (12)	14 (9.27)	0.04
CBP (%)	6 (8)	15 (9.93)	0.04
CEP (%)	0 (0)	2 (1.32)	0.04
IDIOPATHIC (%)	11 (14.66)	10 (6.62)	0.04
OVERLAP HAI AND CBP (%)	5 (6.66)	1 (0.66)	0.04
OVERLAP CBP AND CEP (%)	0 (0)	1 (0.66)	0.04

CBP: Chronic Biliary Pancreatitis, CEP: Chronic Extrahepatic Pancreatitis, HAI: Hepatic Acute Inflammation, MELD-NA: Model for End-Stage Liver Disease with Sodium.

HGF decreases ANIT-induced liver damage through modulation of redox status.

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Introduction and Objectives: Intrahepatic cholestasis is the partial/total obstruction of bile flow, with inflammation and increased reactive oxygen species (ROS). Previous studies indicate that hepatocyte growth factor (HGF) generates hepatoprotective effects in alpha-naphthylisothiocyanate (ANIT)-induced cholestasis. We focused on characterizing the mechanisms of HGF-induced protection in cholestasis.

Materials and Methods: Male CD1 mice aged 8-10 weeks were randomly divided into 4 experimental groups: 1) untreated control group (NT), 2) ANIT-treated group via intragastric administration at a dose of 60 mg/kg, 3) ANIT+HGF-treated group, where HGF will be administered at a dose of 10 µg/kg intravenously 24 hours after ANIT administration, and 4) control group treated only with HGF. Mice were sacrificed at 30 h, 36 h, and 48 h post-treatment initiation for liver tissue and serum collection. The collected samples were used for biochemical assays, Western Blot, TBARS, and H&E staining.

Results: The histological results suggest that HGF can reverse the cholestatic damage observed in time-independent H&E stains, which impacts the architecture of the liver parenchyma, through the decrease in inflammatory infiltrate corroborated with the reversal of the size of the sinusoid area. It was also observed that pyknotic nuclei decrease, which suggests a decrease in cell death as well as an increase in proliferation. These results at the cellular level also impact the decrease in markers of damage at the serum level, such as transaminases, and the decrease in liver size to normal levels. It was also observed that HGF modulates the production of ROS through decreased lipoperoxidation over time, which may be one of the main causes of its hepatoprotective effect in experimental cholestasis. That is why we evaluated the effect of N-acetylcysteine (NAC) as a therapeutic proposal for cholestasis. The proteomic results indicated that NAC increases the protein content of the glutathione system to decrease damage.

Conclusions: HGF regulates a hepatoprotective response by modulating ROS, which favors the reduction of tissue damage reduction and the antioxidant response through the glutathione system. On the other hand, NAC could be suggested as a therapeutic option in cholestatic disease.

Ethics statement: The care and management of the experimental subjects were carried out following the ethical guidelines established by the Universidad Autónoma Metropolitana (UAM) and the National