

were found in a high proportion. Alcohol consumption continues to contribute significantly to liver injury in Mexico.

The authors declare that there is no conflict of interest.

Table 1
Multivariate predictive models

A. Multivariate predictive model to evaluate factors associated with the presence of steatohepatitis with hepatic necroin-flammatory activity at autopsy.				
Variables	P Value	OR	95% CI	
			Lower	Higher
Atherosclerosis	0.008	.405	.208	.789
Obesity	0.948	1.025	.490	2.144
Alcohol consumption	0.044	1.974	1.018	3.827
Diabetes	0.142	.603	.307	1.184
Arterial hipertension	0.185	1.607	.797	3.238
Constant	0.351	1.385		

B. Multivariate predictive model to evaluate factors associated with the presence of significant or greater liver fibrosis (F2-F4) at autopsy.				
Variables	P Value	OR	IC 95% CI	
			Lower	Higher
Atherosclerosis	0.067	.573	.316	1.041
Obesity	0.934	1.032	.489	2.178
Alcohol	0.002	2.529	1.407	4.546
Diabetes	0.955	1.020	.517	2.011
Arterial hipertension	0.077	1.811	.938	3.498
Necroinflammatory activity	<0.0001	6.533	3.720	11.471
Constant	<0.0001	.176		

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FREQUENCY OF RISK FACTORS FOR DEVELOPMENT OF METABOLIC FATTY LIVER DISEASE (MAFLD) IN A CENTER OF CONCENTRATION OF LIVER DISEASES

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Introduction and Objectives: Metabolic fatty liver disease (MAFLD) includes a wide spectrum of presentations, ranging from simple steatosis and non-alcoholic steatohepatitis to liver cirrhosis, leading to hepatocellular carcinoma; the etiology of this is multifactorial, with few studies on risk factors in the Mexican population.

Objective: Determine the frequency of risk factors in patients with MAFLD in a liver disease center: CEIHET, Mexico. **Material and methods:** An observational, cross-sectional and retrospective study selected 587 records from the CEIHET, Hidalgo, of patients with a diagnosis of MAFLD, from January 2017 to May 2020. Determine the frequency of risk factors. Statistical analysis was performed through measures of central tendency, dispersion and correlation.

Results: 587 files were analyzed, 56% (n = 329) women; mean age of 51.4 years. The group with simple steatosis had a mean age of 38 years, while in advanced liver cirrhosis, it was 63 years (p 0.005). In schooling, basic education was found for liver cirrhosis in 73.12% (n = 215), and, in previous stages, it was 21.84% (n = 64). In the AHF and APP, type 2 diabetes is shown as the main risk factor with 65.92% (n = 387) and 58.77% (n = 345), respectively; 6 out of 10 patients had two or more risk factors. 77.3% (n = 454) of patients did not meet the

standards of adequate physical activity. Regarding BMI, 73% of patients with simple steatosis were overweight or obese, while, with liver cirrhosis, it was present in 98% without ascites or edema.

Discussion: A prevalence of fatty liver disease of 55.75% was found, being the main reason for liver Disease consultation, constituting the major cause of liver damage in females. The age of patients increases in direct proportion to the severity of liver damage. Of relevance is education, showing RR of 9.2 (p = 0.001), which indicates that the lower the level of education, a later detection is carried out. 77.3% of these patients did not comply with the physical activity standards established by the WHO. When studying the BMI, we noticed the presence of overweight/obesity in at least 88% of the population, correlating with that established in studies in the USA, where the Latino population has the highest BMI and in the highest frequency.

Conclusions: The present study shows that sedentary lifestyle, overweight, obesity and type 2 diabetes are the main determining factors for the presence of MAFLD in the Mexican population, and therefore the importance of its detection and management, to prevent its progression and improve the quality of life of patients.

The authors declare that there is no conflict of interest.

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PREVALENCE OF COMBINED LIVER DAMAGE IN MEXICAN POPULATION

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Introduction and Objectives: Metabolic syndrome and alcohol consumption are the two main causes of liver steatosis. Often, one of them predominates for its development and the other acts as a cofactor. The impact of these entities separately and their damage in synergy in the Mexican population is currently unknown. Our objective is to determine the prevalence of non-alcoholic liver steatosis, alcoholic liver steatosis, and dual damage in donors from the blood bank of the Hospital General de México "Dr. Eduardo Liceaga" (HGMEI) using transient elastography (TE).

Materials and Methods: Pilot study. prospective, transversal, descriptive, and analytical. Healthy donors from the HGMEI blood bank who attended from June 8 to 29 2021 ≥ 18 years old, of any gender, will be included with a body mass index (BMI) ≥ 18.5 . Donors with liver disease of any etiology and liver malignancy will be excluded. Donors who did not attend TE will be eliminated. The equipment used for TE was FibroScan[®] 502 Touch. Descriptive statistics will be used with measures of central tendency and dispersion.

Results: 30 donors were recruited, four were eliminated for not having performed TE. The age of the subjects was 36.53 ± 12.13 years. There were 13 female subjects and 13 males. Seven (26.92%) donors were classified with non-alcoholic fatty liver disease (NAFLD), 2 (7.69%) with alcoholic fatty liver disease (ALD), and 1 (3.84%) subject with liver steatosis due to combined damage; the 16 (61.55%) remaining subjects corresponded to a healthy population. From the NAFLD group, S1 steatosis was documented in 1 subject (14.28%), S2 in 2 (28.56%), and S3 in 4 (57.16%); F0-F1 liver stiffness was found in 6 (85.72%) subjects, and F4 in 1 (14.28%); Of this group, 3 (42.86%) subjects were classified as overweight, 3 (42.86%) with grade 2

obesity, and 1 (14.28%) with grade 3 obesity. On the other hand, from the ALD group, one donor was found with steatosis S1, and 1 with S2; one of them had liver stiffness F0-F1 with an AUDIT score of 17 and a risk pattern for excessive alcohol consumption (117 g 2 to 4 times a month), while the other subject had F4 fibrosis with an AUDIT score of 7 and a pattern risk of excessive consumption (208 g 2 to 4 times per month), both consume beer. Finally, one donor had liver steatosis due to combined damage, with S3 for steatosis and F4 liver stiffness (27.4 kPa, the highest record in the study); He has grade 3 obesity (BMI 44.41), an AUDIT score of 4, and a pattern of excessive consumption (78 g 2 to 4 times a month) of distillates.

Discussion: What is relevant about this work is that it studies an apparently healthy population and, according to our results, 38.45% (10) of the subjects already have a degree of liver steatosis and even 11.53% (3) showed advanced fibrosis. Our findings are similar to others about the prevalence of nonalcoholic liver steatosis, such as the work of the López-Velázquez group (26%) in 2014. Our study is novel in the investigation of alcoholic liver steatosis and the combined damage. Those issues are scarce addressed in our country. We also collect additional information, such as the identification of the consumption pattern of people with risky alcohol intake and the type of drink they consume.

Conclusions: Our study shows that liver steatosis in this sample of the Mexican population is predominantly non-alcoholic; all subjects within this group have high BMI. Excessive risk consumption is prevalent in subjects with alcohol-related steatosis, and beer is the most frequent drink. In combined damage, obesity and excessive consumption of distillates seem to be the cause of severe steatosis. The only patient in this group already shows advanced fibrosis, which makes us suspect that dual damage accelerates the rate of fibrosis progression.

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HEPATOCELLULAR CARCINOMA AFTER DIRECT ANTIVIRAL AGENTS FOR HEPATITIS C IN PATIENTS WITH DECOMPENSATED CIRRHOSIS

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Introduction and Objectives: If direct antiviral agents (DAA) are related to the development of HCC is controversial; therefore, exploring risk factors are crucial. We aimed to determine factors related to the development of hepatocellular carcinoma (HCC) in patients with hepatitis c (HCV) and decompensated cirrhosis (DC) treated with DAA.

Materials and Methods: A multicenter real-world cohort study including patients with HCV + DC treated with sofosbuvir (SOF) based on regimens free of inhibitors of protease (IPs).

Results: 222 patients, 118 (53.2%) were women, mean age 57.2 ± 11.5-year-old, 209 (94.1%) achieved sustained virological response (SVR). According to Child-Pugh 44(19.8%) were A with history of any clinical decompensation event, 147(66.2%) B, and 31 (14%) C. After DAA, 134 (60.4%) improve in MELD, 45 (20.3%) had no change, and 43 (19.4%) worse, this worse in MELD was related to non-SVR [SVR 37/209 (17.7%) vs. non-SVR 6/13 (46.2%); OR=2.6, 95%CI:1.4-5.0, p=0.02]. Nineteen (8.6%) developed HCC during the follow-up after therapy with DAA; however, when we compared basal laboratory values between those who developed HCC and those who did not only alpha-fetoprotein (AFP) levels were different (without HCC 14.3 [mean 95%CI: 10.6-18.1] ng/mL vs. HCC 55.7 [mean 95%CI: 28.4-83.0] ng/mL; p<0.006). Univariate and multivariate analyses are shown in Table 1.

Discussion: Our study confirms that rather than DAA therapy, the most critical factors related to the development of HCC in DC patients with HCV treated with DAA, are non-achieve SVR and, most important basal AFP levels > 20ng/mL, it is essential to note that patients in this cohort had no any suspicious lesion in ultrasonography (USG) previous to start DAA therapy in their semestral screening as most of the guidelines recommend. AASLD, for example, recommends semestral HCC screening with USG with or without AFP, giving more weight to the imagen study. However, based on our results, we recommend always determining AFP levels as a complement to USG.

Conclusions: In HCV patients with DC treated with DAA and with a negative basal screening USG for suspicious malignant lesions, basal AFP > 20ng/mL are the most critical factor related to the development of HCC and should be determined complementary to the USG study.

The authors declare that there is no conflict of interest.

Table 1

Risk factors related to the development of HCC after DAA in decompensated cirrhosis with hepatitis C infection

Variable	Univariate analysis			
	HCC n=19	Without-HCC n=203	p	OR (95%CI)
Age, year-old	58±10.6	57±11.5	0.75	-
Male gender, n(%)	6 (30.6)	98 (48.3)	0.17	0.5 (0.2-1.3)
Use of ribavirin, n(%)	13 (68.4)	162 (79.8)	0.25	0.5 (0.2-1.5)
Non-achieve SVR, n(%)	4 (21.1)	9 (4.4)	0.008	5.7 (1.6-20.9)
Basal AFP level > 20ng/mL	10 (52.6)	30 (14.8)	<0.0001	7.3 (2.6-20.7)
Diabetes, n(%)	5 (26.3)	31 (15.3)	0.21	2.0 (0.7-5.9)
Obesity, n(%)	5 (26.3)	36 (17.7)	0.36	1.6 (0.6-4.9)
Progression of decompensation according to MELD	6 (31.6)	37 (18.2)	0.17	2.1 (0.7-5.8)

Variable	Multivariate adjusted analysis	
	p	OR (95%CI)
Non-achieve SVR, n(%)	0.16	3.0 (0.6-14.5)
Basal AFP level > 20ng/mL	<0.0001	6.6 (2.3-19.0)

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