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.

The authors declare that there is no conflict of interest.

https://doi.org/10.1016/j.aohep.2021.100606

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

F- Higuera-de-la-Tijera[1], N.C. Flores-García[2], D. Kershenobich[2], E. Cerda-Reyes[3], M.S. González-Huezo[4], J.L. Pérez-Hernández[1,5], N. Méndez-Sánchez[6], O. Morales-Gutiérrez[1], J.E. Lira-Vera[1], P. Alagón-Fernández-del-Campo[1], F.Y. Vargas-Durán[1], K. Soto-Martínez[1], G.M.L. Guerrero-Avendaño[1], A. Servín-Caamaño[1], J.G. Gándara-Calderón[3], D. García-Domínguez[4], L.E. Díaz-Orozco[6], N. García-Casarreal[7], S. Mejía-Loza[8], I.B. Lázaro-Pacheco[9], E. Rodríguez-Fuentes[1]

[1] *Hospital General de México "Dr. Eduardo Liceaga". Ciudad de México, México*
[2] *Instituto Nacional de Ciencias Médicas y Nutrición "Salvador Zubirán". Ciudad de México, México*
[3] *Hospital central militar. Ciudad de México, México*
[4] *Centro Médico ISSEMyM Metepec, Edo. de México, México*
[5] *Hospital Central Sur, PEMEX. Ciudad de México, México*
[6] *Fundación Clínica Médica Sur. Ciudad de México, México*
[7] *Hospital Regional 1° de Octubre, ISSSTE. Ciudad de México, México*
[8] *Hospital Juárez de México. Ciudad de México, México*
[9] *ISSSTE Oaxaca. Oaxaca, México*

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 alfa-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 compliment 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

Univariate analysis				
Variable	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)

Multivariate adjusted analysis		
Variable	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)

https://doi.org/10.1016/j.aohep.2021.100607