Table 1
Clinical and biochemical features in patients with Hepatocellular carcinoma

Age	66.9±11
Sex % (Female / Male)	53.8 (70) / 46.2 (60)
HC etiology % (Alcohol/ Cryptogenic MAFLD/ HCV / HBV)	32(24.6) / 20(15.4) / 8(6.2) / 3(2.3)
Comorbidities (Overweight/ Obesity/ HBP/DMT2)	58 (43.9) / 19(14.4) / 53(40.2) / 51 (38.6)
Child-Pugh % (A, B, C)	50% (65)/ 14.6% (19)/ 1.5% (2)
BCLC % (A, B, C, D)	28.5% (37)/ 56.1% (74)/ 10.8% (14)/ 3.8% (5)
ECOG % (0,1,2,3)	21.2% (11)/ 50% (26)/ 26.9% (14)/ 1.9% (1)
Ascites %	10.8% (14)
ALT (UI/L)	46±29
AST (UI/L)	67.6±54
BT (mg/dl)	1.27±1.6
Albumin (mg/dl)	3.55±0.6
Platelets (x 1,000)	174±103
Prothrombin time (s)	15±3.4
INR	1.17±0.1
Serum alphafetoprotein (ng/nL)	5135.1±26422
MELD	9.86±3.0
Tumor diameter (cm)	7.13±3.7
Number of tumors	1.37±1

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A MULTICENTER REAL-WORLD COHORT TO VALIDATE THE EFFICACY AND SAFETY OF DIRECT ANTIVIRAL AGENTS FOR HEPATITIS C, AND RELATED RISK FACTORS FOR NON-SVR IN DECOMPENSATED CIRRHOSIS

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Introduction and Objectives: Clinical trials demonstrated the efficacy and safety of direct antiviral agents (DAA) to treat hepatitis C virus infection (HCV) in patients with decompensated cirrhosis (DC); however, very few real-world studies have been reported in this group. Moreover, predictive factors for non-achieving sustained virological response (SVR) in DC are not entirely understood. Therefore, the aim was to verify the efficacy and safety of DAA and to identify risk factors for failure to achieve SVR in DC.

Materials and Methods: A real-world cohort study included HCV patients with DC [Child-Pugh B/C or A but with a history of previous clinical decompensation events like variceal bleeding (VB), hepatic encephalopathy (HE), or ascites]. All patients treated before transplant had MELD ≤ 20 and Child-Pugh ≤ 12 according to AASLD guidelines.

Results: 222 patients, 118 (53.2%) were women, mean age 57.2±11.5 year-old, 61 (27.5%) were treated with Sofosbuvir (SOF)/Ledipasvir, 152 (68.5%) with SOF/Velpatasvir, and 9 (4.1%) with SOF/Daclatasvir, 175 (78.8%) used also ribavirin, 209 (94.1%) achieved SVR, despite non-significant difference, Child-Pugh C patients had a suboptimal response (SVR < 95%): A 42/44 (95.5%), B 140/147 (95.2%), C 27/31 (87.0%), p=0.2. Related adverse events were fatigue 60 (27%), nausea 44 (19.8%), headache 43 (19.4%), non-severe peripheral edema 10 (4.5%), anasarca 4 (1.8%), jaundice 6 (2.7%), 3 hemolytic anemia (1.4%), 1 dermatosis (0.4%), congestive cardiac failure 2 (0.9%), need to suspend therapy due to liver-related adverse events 2 (0.9%) they also died. In those who achieved SVR, MELD improved (basal 12.4±3.3 vs. post-SVR 10.9±3.5; p<0.0001); but was worse in those without SVR (basal 16.2±3.9 vs. without-SVR 17.3±6.2; p=0.24). Times/year needing hospitalization for liver-related decompensation events were less frequent in those who achieved SVR (basal 1.7±1.3 vs. after SVR 0.4±0.7; p<0.0001) but remained without change in that without-SVR (basal 1.5±1.5 vs. after non-achieve SVR 2.2±1.9; p=0.04).

Discussion: A few real-world studies have been conducted in DC with hepatitis C. However, in the Mexican population, our study is the first that demonstrated in a real-world setting, similar to clinical trials, that DAA based on SOF and free of protease inhibitors (PIs) are effective and safe to cure HCV in DC. When to treat HCV before or after liver transplantation can be challenging. Classically, MELD >20 and Child-Pugh C >12 are related to non-SVR; however, our study also shows that additional clinical factors have a negative impact on SVR: history of recurrent VB and episodic and persistent HE; therefore, these criteria should also be considered to decide to treat previous or after liver transplantation. In addition, acute decompensation and mortality events are very high in those who do not achieve SVR.

Conclusions: SOF based on regimens without PIs are effective and safe in VHC with DC. Additional to classic criteria (MELD >20, Child-Pugh > 12), recurrent VB and HE are predictors of failure to achieve SVR in VHC with DC.

The authors declare that there is no conflict of interest.

Table 1
Comparison of characteristics of the cohort according to the response to DAA therapy

Variable	SVR n=209	Without-SVR n= 13	p	OR (95%CI)
Basal characteristics				
Age, year-old	57.3±11.3	55.7±14.9	0.63	-
Transition elastography, KPa	28.8±12.9 (n=181)	41.7±21.6 (n=10)	0.09	-
Child-Pugh, points	8±1	9±2	0.05	-
MELD, points	12.4±3.3	16.2±3.9	<0.0001*	-
Viral load, IU/mL	2,219,130	2,368,392	0.96	-
Episode of variceal bleeding, n(%)	137 (65.6)	10 (76.9)	0.55	1.2 (0.9-1.6)
Recurrent variceal bleeding, n(%)	46 (22.0)	10 (76.9)	<0.0001*	3.5 (2.4-5.2)
Ascites, n(%)	123 (58.8)	10 (76.9)	0.25	1.3 (0.9-1.8)
	76 (36.4)	9 (69.2)	0.03*	1.9 (1.3-2.9)

(*continued*)

Table 1 (*Continued*)

Variable	SVR n=209	Without-SVR n= 13	*p*	OR (95%CI)
Basal characteristics				
Episodic hepatic encephalopathy, n (%)				
Persistent hepatic encephalopathy, n (%)	28 (13.4)	5 (38.5)	0.03*	2.9 (1.3-6.2)
History of spontaneous bacterial peritonitis, n(%)	4 (1.9%)	1 (7.7)	0.26	4.0 (0.5-33.4)
Follow-up at 1-year after DAA therapy				
Child-Pugh, points	6±2	9±3	0.007*	-
MELD, points	10.9±3.5	17.3±6.2	0.003*	-
Transition elastography, KPa	23.2±11.6 (n=62)	45.7±20.8 (n=9)	0.01*	-
Variceal bleeding, n (%)	26 (12.4)	7 (53.8)	0.001*	4.3 (2.3-8.0)
Ascites, n(%)	32 (15.3)	9 (69.2)	<0.0001*	4.5 (2.8-7.3)
Episodic hepatic encephalopathy, n (%)	22 (10.5)	7 (53.8)	<0.0001*	5.1 (2.7-9.7)
Persistent hepatic encephalopathy, n (%)	6 (2.9)	3 (23.1)	0.01*	8.0 (2.3-28.6)
Spontaneous bacterial peritonitis, n (%)	0 (0)	1 (7.7)	0.06*	0.9 (0.8-1.1)
Mortality rate, n(%)	1 (0.5)	4 (30.8)	<0.0001*	64.3 (7.7-534.9)

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STUDY OF CONCORDANCE BETWEEN THE DEGREE OF LIVER FIBROSIS ESTIMATED THROUGH APRI AND FIB-4 BIOCHEMICAL SCORES, AND ELASTORESONANCE IN PATIENTS WITH AUTOIMMUNE HEPATITIS

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Introduction and Objectives: Autoimmune hepatitis is a chronic and progressive necroinflammatory disease with a fluctuating course of activity and affects more frequently the female sex. The etiopathogenesis is still unknown and may be the result of an interaction between factors: genetic, immunological, autoantigens; Therefore, the interaction of genetic predisposition with an environmental trigger and the disorder in immunoregulation would result in chronic inflammation of the hepatocytes and with it the development of hepatic fibrosis. Diagnostic tests for the evaluation of liver fibrosis include liver biopsy and non-invasive elastographic methods, such as transition elastography and elastoresonance, as well as serum biomarkers, composed of different variables that help predict the degree of liver fibrosis.

Objective: Compare the concordance between the results obtained for the diagnosis of liver fibrosis by the APRI and FIB-4 score, with the elastoresonance, in patients with HAI.

Material and methods: Elastoresonance, APRI and FIB-4 were performed in 6 patients to assess the concordance between different degrees of fibrosis.

Results: A total of 6 patients with a recent diagnosis of HAI were included in the study. The mean age was 50.33 years and 100% were women. 66.66% of the patients presented an advanced degree of fibrosis (F2-F3-F4) due to elastoresonance. The values for the APRI index were: 3 patients (50%) had an advanced degree of fibrosis, 1 patient had a low degree of fibrosis (6%) and 2 patients (33.33%) had intermediate. The findings for the FIB-4 values were exactly the same. The agreement of elastoresonance in the different degrees of fibrosis against the APRI and FIB-4 score was 100%.

Discussion: Non-invasive methods to measure the degree of liver fibrosis in patients with chronic liver disease have shown to be useful, and in this study, it transcends that the correlation with the degree of fibrosis obtained by elastoresonance with the APRI and FIB-4 scores is 100%, this could avoid reaching the liver biopsy, which although it is the gold standard in measuring the degree of liver fibrosis, is an invasive and expensive method, which involves risks for the patient (puncture of other internal organs, infection and adverse reaction to contrast material).

Conclusions: In patients classified with advanced fibrosis, the concordance between the estimates obtained using the elastoMR and those derived from the APRI and FIB-4 scores are high. However, a limitation of this study is the size of the sample.

The authors declare that there is no conflict of interest.

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GLUCOGENOSIS AS A CAUSE OF INTRAHEPATIC CHOLESTASIS

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Introduction and Objectives: Hepatic glycogen storage pathologies are very rare diseases among inborn errors of metabolism caused by the alteration of the enzymes involved in the metabolism of glycogen. GSDs are classified according to enzyme deficiency and affected tissue, including types 0, Ia, Ib, III, IV, VI, and XI. The clinical presentation can be very varied, including intolerance to fasting, growth retardation and hepatomegaly. It can present with hypoglycemia, hyperlactatemia, increased liver enzymes, and hyperlipidemia. The most common type of glycogen storage disease is GDS IX, its incidence is 1: 100,000 births and it is responsible for 25% of all cases. The most common GSD IX subtype is subtype IX a caused by mutations in PHKA2, which accounts for 75% of cases. Long-term complications include liver adenomas, kidney disease, cardiomyopathy, and muscle symptoms.

Clinical case: 27-year-old male patient. Important history, positive smoking with IT 1, positive alcoholism with consumption of 8 grams of alcohol per day for 10 years. During childhood, he presented hepatomegaly and an isolated event of jaundice. He began his condition one month before with asthenia, adynamia and weight loss (9 kg / 30 days), accompanied by generalized jaundice, later he presented abdominal pain in the right upper quadrant, with early satiety, acholia and choluria. On physical examination: hepatosplenomegaly, jaundice of the skin and integuments. Paraclinical: BT: 5.3 mg / dl, BD: 1.2 mg / dl 8, DHL: 444IU / L, TGP: 89 U / L, TGO: 168 U / L FA: 1050 U / L. HIV: non-reactive, HBV HCV non-reactive. Ultrasound with multiple nodular echogenic lesions without bile duct dilation, a 1.8 mm common bile duct, a 7 mm portal vein, and an enlarged spleen. Endoscopy: portal hypertensive gastropathy, extrinsic compression of the body and gastric fundus without evidence of varicose veins. Cholangioresonance without intra- and extra-hepatic bile duct dilation. Normal alpha 1 antitrypsin (2.08 g / dl). Given the evidence of intrahepatic cholestasis, it was decided to perform an ultrasound-guided liver biopsy where it was observed positive for intracytoplasmic glycogen in the hepatocytes, thus establishing a definitive diagnosis of Glycogenosis.

Discussion: Due to its low incidence, the diagnostic approach of Glycogenosis presents a challenge. The diagnosis is made at an early