Table 1: Characteristics of lean patients with and without cirrhosis

Variables	Cirrhosis N=102 (%)	Non-cirrhosis N=45 (%)	P-value
Sex			
Female	51 (50%)	33 (73.3%)	**0.008**
Male	51 (50%)	12 (26.7%)	
BMI (mean)	23.2	23.2	0.89
Age (mean)	68.8	62.2	**<0.001**
Diabetes	45 (44.1%)	12 (26.7%)	**0.04**
Dyslipidemia	9 (8.8%)	11 (24.4%)	**0.01**
Chronic kidney disease	4 (3.9%)	1 (2.2%)	0.60
Hypertension	1 (0.9%)	8 (17.8%)	0.08
Hepatocellular carcinoma (HCC)	54/102 (53%)	8/45 (17.8%)	**<0.001**
Common Variants: PNPLA3 (rs738409)			**0.05**
GG	31/56 (55.4%)	10/34 (29.4%)	
CG	20/56 (35.7%)	19/34 (55.9%)	
CC	5/56 (8.9%)	5/34 (14.7%)	
MBOAT7 (rs641738)			0.29
TT	7/56 (12.5%)	2/34 (5.9%)	
CT	25/56 (44.6%)	14/34 (41.2%)	
CC	24/56 (42.9%)	18/34 (52.9%)	
TM6SF2 (rs58542926)			0.64
TT	0/56 (0%)	0/34 (0%)	
CT	6/56 (10.7%)	5/34 (14.7%)	
CC	50/56 (89.3%)	29/34 (85.3%)	
HSD17B13 (rs72613567)			0.45
TT	0/53 (0%)	1/33 (3%)	
AT	9/53 (17%)	5/33 (15.2%)	
AA	44/53 (83%)	27/33 (81.8%)	
GCKR (rs1260326)			0.84
CC	18/56 (32.1%)	11/32 (34.3%)	
CT	30/56 (53.6%)	15/32 (46.9%)	
TT	8/56 (14.3%)	6/32 (18.8%)	

*The alleles description in the table begins with homozygous, heterozygous, and wild type in PNPLA3, MBOAT7, TM6SF2, HSD17B13, and GCKR, respectively

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P-16 UNLOCKING S-PINDOLOL'S POTENTIAL FOR MASLD: BENEFICIAL EFFECTS ON MUSCLE MASS AND FUNCTION AND LIVER HISTOLOGY IN WESTERN- DIET FED MICE

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Conflict of interest: No

Introduction and Objectives: Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) is linked to sarcopenia, which worsens disease's prognosis. The complex liver-muscle crosstalk opens the possibility that improvements in muscle quantity and quality may be directly beneficial for MASLD. This study investigates the effects of s-pindolol, a beta-blocker known for its anabolic properties, on both muscle mass and function as well as on MASLD progression in mice.

Patients / Materials and Methods: Male C57BL6 mice were subjected to a western diet (WD) for 20 weeks to induce MASLD and then mice were randomly grouped and treated with 3 mg/kg s-pindolol twice a week or left untreated. Assessments included grip and isolated muscle strength, body composition via bioimpedance spectroscopy, Abdominal MRI, liver histology, serum analyses and gene expression profiling.

Results and Discussion: S-pindolol reduced liver steatosis and inflammation and was associated with lower levels of MCP-1, IL-10, TGF-β, and ACACA (Acetyl-CoA Carboxylase Alpha). S-pindolol also counteracted IGF-1 serum levels reduction seen in WD-fed mice. In addition, S-pindolol treatment led to an increase in muscle mass as confirmed by bioimpedance spectroscopy and MRI techniques. While exercise performance remained unchanged, grip strength improved together with a reduction in myosteatosis suggesting enhanced muscle quality. This was supported by an increase in muscle fiber diameter, indicating muscular hypertrophy independent of exercise.

Conclusions: S-pindolol treatment ameliorates MASLD and enhances muscle quality in WD-fed mice. It may be hypothesized that s-pindolol's positive effects on muscle mass and function could play a role in its beneficial effects on MASLD through improvement of secretion of various salutary myokines. The present data underscore S-pindolol's therapeutic potential in MASLD.

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P-17 AMIKACIN USE AND RISK OF NEPHROTOXICITY IN PATIENTS WITH LIVER CIRRHOSIS HOSPITALIZED FOR SEPSIS

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Conflict of interest: No

Introduction and Objectives: Aminoglycosides are a group of broad-spectrum antibiotics, which have action especially against gram-negative bacteria. It is associated with nephrotoxicity in 10-20% of cases, a figure that increases in patients with liver cirrhosis. Amikacin is frequently used in sepsis, with little information about the risk of nephrotoxicity in cirrhosis. The aim was to determine the association between amikacin use and renal function deterioration in patients with liver cirrhosis and sepsis.

Patients / Materials and Methods: Retrospective, observational, analytical study in patients with liver cirrhosis of any etiology, who required hospitalization for sepsis between 2017 and 2023, and who received antibiotic therapy. An increase in serum creatinine $\geq$ 0.3 mg/dl in the first 7 days of hospitalization was used as a marker of renal function deterioration. Clinical variables, renal failure and mortality were compared between patients who received amikacin and those who did not. Stata 13.0 was used for data analysis with a statistical significance of 0.05.

Results and Discussion: In this study 228 patients were included, median age 65 years (54-70), 100 (44%) women, 70 received amikacin (31%). Renal function deterioration was present in 25 (36%) patients with amikacin and 33 (21%) without amikacin. In patients with initial serum creatinine > 2.0 mg/dl and in those with Child-Pugh C cirrhosis, the probability of developing renal function deterioration was higher in those who received Amikacin (OR 7.5; 95% CI 1.1

– 48.0, p= 0.031 and OR 2.51; 95% CI 1.06 – 5.97, p = 0.036, respectively). A comparative table of different subgroups is attached.

Conclusions: The use of amikacin was associated with renal function deterioration in patients with liver cirrhosis and sepsis, mainly in Child-Pugh C cirrhosis and with initial serum creatinine > 2.0 mg/dl.

Table 1. Evaluation of the development of renal function deterioration in different groups of patients depending on the use or not of Amikacin

		Renal function deterioration	Without Renal function deterioration	Valor p
According to initial creatinine level				
Creatinine < 1.5 (N = 166)	Amikacin	13 (36)	35 (27)	0.282
	Without Amikacin	23 (64)	95 (73)	
Creatinine 1.5 - 1.99 (N = 33)	Amikacin	4 (33)	4 (19)	0.357
	Without Amikacin	8 (67)	17 (81)	
Creatinine ≥ 2.0 (N = 28)	Amikacin	7 (78)	6 (32)	**0.029**
	Without Amikacin	2 (22)	13 (68)	
Initial suspicion of infection by Gram (-) bacteria according to infectious focus				
Suspected Gram (-) infection (N = 171)	Amikacin	21 (48)	41 (32)	0.066
	Without Amikacin	23 (52)	86 (68)	
Without suspicion of Gram (-) infection (N = 57)	Amikacin	4 (28)	4 (9)	0.071
	Without Amikacin	10 (71)	39 (91)	
According to the development of septic shock				
With septic shock (N = 74)	Amikacin	16 (57)	22 (48)	0.437
	Without Amikacin	12 (43)	24 (52)	
Without septic shock (N = 154)	Amikacin	9 (30)	23 (19)	0.165
	Without Amikacin	21 (70)	101 (81)	
According to Child-Pugh Score				
Child-Pugh A – B (N = 104)	Amikacin	10 (34)	18 (24)	0.280
	Without Amikacin	19 (66)	57 (76)	
Child-Pugh C (N = 123)	Amikacin	14 (50)	27 (28)	**0.033**
	Without Amikacin	14 (50)	68 (72)	
According to MELD-Na Score				
MELD-Na < 20 (N = 77)	Amikacin	3 (25)	14 (21)	0.524
	Without Amikacin	9 (75)	51 (78)	
MELD-Na ≥ 20 (N = 150)	Amikacin	21 (47)	31 (30)	**0.034**
	Without Amikacin	24 (53)	74 (70)	

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P-18 ANTI-HBC POSITIVITY IS AN INDEPENDENT PREDICTIVE FACTOR FOR HCC DEVELOPMENT AND SHOULD BE INCORPORATED TO THE HCC RISK SCORE PREDICTION MODEL IN ADVANCED FIBROSIS

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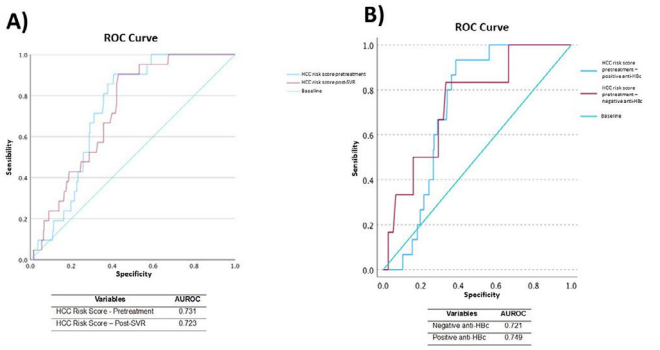
Conflict of interest: No

Introduction and Objectives: After hepatitis C virus (HCV) treatment with direct-acting antivirals (DAAs), the sustained viral response increased to 95%, although it may be lower in advanced fibrosis. Continuous follow-up of HCV cured individuals is essential. In that context, identifying higher risk populations to intensify its surveillance is important to allow early hepatocellular carcinoma (HCC) detection and optimize costs. Our group previously demonstrated a risk association between anti-HBc and HCC, since the presence of hepatitis B virus infection has oncogenic properties. Our objective is to evaluate the HCC risk score prediction model accuracy in our population and investigate the inclusion of anti-HBc positivity in the model.

Patients / Materials and Methods: This is a retrospective, observational, descriptive and analytic study in a series of cases in which 365 HCV patients were evaluated. Demographic, clinical and laboratory data were obtained through electronic medical records. The HCC risk score was applied before and after SVR.

Results and Discussion: A total of 21 patients had HCC diagnosis after RVS (5.75%). The variables associated with higher HCC occurrence were: genotype 3 (p=0.025), AST pretreatment (p=0.026), elastography > 10kPa (p=0.003) and advanced fibrosis (p=0.016). Among advanced fibrosis patients, positive Anti-HBc (p=0.047) was an independent predictor factor associated with HCC. After univariated analysis, genotype 3 and positive anti-HBc were predictive factors of HCC occurrence in advanced fibrosis. When applying the HCC risk score before treatment, the area under the receiver operating characteristic curve (AUROC) was 0.731, while after SVR had a slightly lower value (0.723) – Figure 1A. After incorporating anti-HBc to all other variables used in HCC risk score, the accuracy was 0.744 – Figure 1B.

Conclusions: The HCC risk score is a validated prediction model of HCC occurrence before and after SVR. Anti-HBc positivity might be a good candidate to be incorporated to the model.



HCC risk score ROC curves. A) Pretreatment x Post-SVR. B) After anti-HBc incorporation

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P-19 ASSOCIATION OF TM6SF2 GENE POLYMORPHISMS WITH CARDIOVASCULAR RISK IN PATIENTS WITH METABOLIC DYSFUNCTION –ASSOCIATED STEATOTIC LIVER DISEASE (MASLD) IN A HISPANIC ADULT POPULATION

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Conflict of interest: No

Introduction and Objectives: Genes that influence lipids have led to the discovery of a non-synonymous variant (rs58542926) located in the TM6SF2 gene (transmembrane 6 member of