	ANI>0 (n = 18)	ANI<0 (n=67)	Odds ratio	p-value
Age (years)	62.2±9.4	61.8±9.8		0.97
Male	100% (18/18)	40.3% (27/67)	Infinity	**<0.001**
Body mass index	28.7±3.9	31.7±4.6		**0.02**
Arterial hypertension	61.1% (11/18)	59.7% (40/67)	1.06	0.86
Diabetes mellitus	50% (9/18)	40.3% (27/67)	1.48	0.64
Dyslipidemia	38.9% (7/18)	53.7% (36/67)	0.55	0.39
Recorded alcohol consumption:	72.2% (13/18)	61.2% (41/67)	1.71	0.51
0 g/week	23.1% (3/13)	63.4% (26/41)	0.17	**0.01**
10-20 g/week	15.4% (2/13)	12.2% (5/41)	1.31	0.54
30-60 g/week	7.7% (1/13)	4.9% (2/41)	1.65	0.57
10-20/30 g/day (female/male)	53.9% (7/13)	19.5% (8/41)	4.85	**0.03**
Fibrosis (Kpa)	7.6±3.6	5.9±1.7		0.18
CAP	292.7±46.6	305.6±51.9		0.41
AST (U/L)	46.7±35.7	34.1±15.9		0.08
ALT (U/L)	38.9±33.6	39.1±23.9		0.39

Comparison of ANI>0 and ANI<0 groups.

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P-14 DETECTION OF CIRRHOSIS DUE TO ALPHA-1 ANTITRYPSIN DEFICIENCY IN ADULTS: PHENOTYPE STUDY IN COSTA RICA

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Conflict of interest: No

Introduction and Objectives: Alpha-1 antitrypsin (A1AT) levels are normal in up to 20% of liver diseases, and this protein elevates in inflammatory states, causing false negatives. This disease does not follow an autosomal recessive inheritance pattern, so the classical concept of homozygosity does not apply. Instead, two codominant alleles manifest as liver or lung disease. *Objectives:* To determine the phenotypes associated with A1AT-related liver disease in Costa Rica.

Patients / Materials and Methods: Phenotypes detected in patients with suspected A1AT deficiency from 2014 to July 2024 were analyzed. Phenotype identification was carried out using isoelectric focusing in agarose gel with immunofixation. The presence of liver disease was determined through clinical, laboratory, and imaging findings.

Results and Discussion: During the specified period, 371 phenotype studies were conducted on 187 women and 184 men. The identified phenotypes were: 15 ZZ probands, 22 MZ probands, 1 SZ proband, 7 MS probands, 1 SS proband, 1 null proband, 1 M/null proband, 31 MM probands, and 2 null/null probands. No Z/null proband was detected. Among 53 probands, there were: 10 ZZ, 13 MZ, 1 SZ, 4 MS, 1 SS, 1 null, and 23 MM. It was established that the risk of liver disease is slightly increased in MZ, increased in SZ, and very increased in ZZ. Cirrhosis was diagnosed in 19 probands: 7 ZZ, 7 M/null, 4 MZ, and 1 SZ.

Conclusions: A1AT quantification has a 20% false-negative rate, so phenotype testing is recommended when there is suspicion. In Costa Rica, the ZZ variant has the highest risk of liver disease, followed by SZ and MZ; the M/null phenotype was also detected as a cause of liver disease. Medical monitoring is necessary, and in doubtful cases, genotype testing should be performed.

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P- 15 GENETIC AND CLINICAL CHARACTERISTICS IN LEAN MASLD PATIENTS WITH AND WITHOUT CIRRHOSIS IN LATINOAMERICAN POPULATION

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Conflict of interest: No

Introduction and Objectives: Metabolic dysfunction associated esteatotic liver disease (MASLD) is the most common chronic liver disease worldwide. It is associated with metabolic conditions and can also occur in lean patients with a normal or low BMI. Polymorphisms in *PNPLA3, TM6SF2* and *MBOAT7* genes have been linked to an increased risk of developing hepatocellular carcinoma (HCC) and greater severity of fibrosis. This study aimed to assess clinical and genetics characteristics in Latin American lean MASLD patients with and without cirrhosis.

Patients / Materials and Methods: This descriptive cross-sectional study evaluated 148 patients from an international database of chronic liver disease patients (ESCALON), including Colombia, Brazil, Chile, Ecuador, Argentina and Perú. MASLD was identified in patients with a BMI≤ 25. Patients with alcohol-associated and viral -related liver diseases, as well as other liver conditions, were excluded. Clinical features and main MASLD-related pathogenic variants were evaluated in this population. We used BlueSky software to evaluate two sample t -test for cirrhotic vs no cirrhotic variables. The assessment included the median age range and average BMI.

Results and Discussion: A total of 102 patients (69%) were found to have cirrhosis, with 57% being female and a median age of 65,6 years.42% had HCC, 39% had diabetes mellitus(DM), 14% had dyslipidemia, and 28% had hypertension (HTN).Common genetic variants were evaluated in 60.8% (90/148) of the study population with the following distribution: *PNPLA3 (rs738409)*: 45,6% (GG), 43,3% (CG);*MBOAT7(rs641738)*: 10%(TT), 43,3%(CT);*TM6SF2(rs58542926)*: 0%(TT), 12,2%(CT) and 87,8%(CC);*HSD17B13(rs72613567)*:1,2%(TT), 16,3%(AT);*GCKR(rs1260326)*:33,3%(CC), 50%(CT).The characteristics by group and the differences found are shown in table 1

Conclusions: Cirrhotic patients were older, with higher rates of diabetes mellitus, hypertension, dyslipidemia and HCC. The PNPLA3 GG variant was predominant in cirrhotics compared to non-cirrhotic patients, with no significant differences between groups in the other variants

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