

complications. Other recipient and donor characteristics are presented in the table. One-year overall survival was 77% (95% CI 72-82); at 5 years, it was 67% (95% CI 60-72); and at 10 years, it was 59% (95% CI 51-66). Survival with functional grafts at 1 year was 77% (95% CI 72-82); at 5 years, it was 65% (95% CI 58-70); and at 10 years, it was 54% (95% CI 46-62).

Conclusions: For the first time, data from the region demonstrate that long-term patient survival following SLKT meets international standards.

Recipient and Donor Characteristics (n=293)	
Variable	Result (n=293)
PRE-TRANSPLANT RECIPIENT CHARACTERISTICS	
Arterial hypertension, n (%)	153 (54)
Diabetes, n (%)	99 (35)
Dyslipidemia, n (%)	49 (17)
BMI, median (IQR)	24 (22 - 28)
Pre-transplant mechanical ventilation, n (%)	37 (13)
INDUCTION IMMUNOSUPPRESSION, n (%)	
Methylprednisolone	40 (14)
Methylprednisolone, Thymoglobulin	36 (12)
Methylprednisolone, Basiliximab,	158 (54)
Methylprednisolone, Basiliximab, Thymoglobulin	7 (2)
Other	52 (18)
REJECTION, n (%)	
Biopsy-proven acute/cellular liver rejection*	21 (8)
Biopsy-proven acute/cellular kidney rejection*	28 (11)
DONOR CHARACTERISTICS	
Male gender, n (%)	180 (63)
Age, median (IQR)	34 (24 - 47)
BMI, median (IQR)	25 (23 - 27)
Sodium, median (IQR)	151 (145 - 159)
Creatinine, median (IQR)	0.95 (0.71 - 1.20)

*At least one episode during follow-up.

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O2- CHARACTERIZATION OF STEATOTIC LIVER DISEASE AND THE ROLE OF GENETIC BACKGROUND IN LATIN AMERICA

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Introduction and Objectives: Although Latinos living in the United States are at higher risk of steatotic liver disease (SLD), information from Latin American countries is extremely scarce. We aimed to characterize SLD in Latin America and explore the role of the common genetic variants in this region.

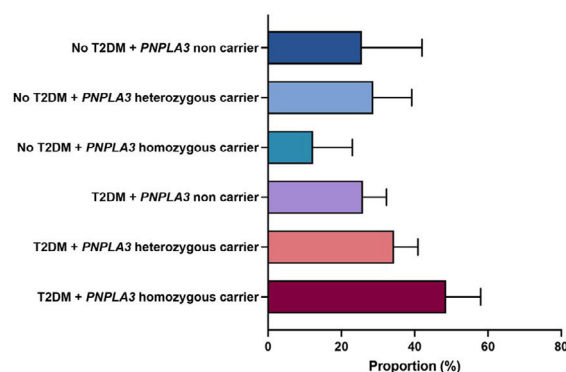
Patients / Materials and Methods: Cross-sectional multicenter study including individuals with SLD who undergo liver biopsy or transient elastography (TE) between 2003–2024. TE thresholds were established as follows: significant fibrosis (F2) ≥8.2 kPa, advanced fibrosis (F3) ≥9.7 kPa, and cirrhosis (F4) ≥13.6 kPa. Analyses included logistic binary regression.

Results and Discussion: We included 2,159 patients (93.7% metabolic dysfunction-associated SLD and 6.3% alcohol-associated liver

disease) from 13 centers in 5 countries (Argentina, Brazil, Chile, Mexico, and Peru). Mean age was 54.3 ± 14.3 years old, 58.8% were female, and 60.2% had a liver biopsy. Around 18% had significant fibrosis, 18.7% advanced fibrosis, and 16.2% had cirrhosis. Additionally, 21.8% were homozygous carriers of the rs738409 risk polymorphism (PNPLA3 I148M variant), 42.5% were heterozygotes, and 35.7% were non-carriers. In an adjusted multivariable model, only age (odds ratio [OR]:1.03; 95%CI:1.01–1.04; $p=0.030$), body mass index (BMI) (OR:1.04; 95%CI:1.01–1.08, $p=0.008$), prediabetes/diabetes (OR:2.41; 95%CI:1.48–3.91, $p<0.0001$), and PNPLA3 risk allele carriers (heterozygotes: OR:2.86, 95%CI:1.78–4.61; $p<0.0001$, and homozygous: OR:25.37, 95%CI:4.30–149.54; $p<0.001$) were associated with a higher risk of advanced fibrosis. Similar results were observed in multivariable models to assess the risk of cirrhosis. Prevalence of advanced fibrosis or cirrhosis was higher in those with prediabetes/diabetes and homozygous carriers of the PNPLA3 I148M variant than those without both risk factors (48.6% vs. 27.9%, $p<0.0001$) (Figure).

Conclusions: Age, BMI, prediabetes, diabetes, and carriers of the PNPLA3 risk allele carriers were the leading risk factors for advanced fibrosis in SLD. PNPLA3 I148M variant carriers are frequent in SLD patients in Latin America, and genotyping could be established to stratify the risk of liver fibrosis routinely in clinical practice. (Supported Fondecyt #1241450)

Prevalence of advanced fibrosis or cirrhosis according to the presence of prediabetes/diabetes (T2DM) and the PNPLA3 I148M variant carriers



Figure

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O3- RECOMPENSATION IN PATIENTS WITH CIRRHOSIS PRIOR TO FIRST LINE SYSTEMIC THERAPY IS ASSOCIATED WITH SIMILAR SURVIVAL OUTCOMES COMPARED TO COMPENSATED CIRRHOSIS

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Conflict of interest: No

Introduction and Objectives: The term “recompensation” of cirrhosis was proposed in the latest BAVENO VII, underlying dynamic events and prognosis in cirrhosis. However, there is uncertainty regarding its prognosis in patients with advanced hepatocellular carcinoma (HCC) treated with first line systemic therapies (1L). We aimed to compare post-1L survival between compensated (CC), decompensated (DC), and recompensated (RC) cirrhosis.

Patients / Materials and Methods: A multicenter prospective Latin-American cohort study including advanced HCC patients with cirrhosis who received any 1L was conducted from 2018 to 2024. Three groups were defined: CC (had never presented decompensation); DC (presenting any decompensated event associated with portal hypertension at time of 1L), and RC group (prior history of any decompensation event at HCC diagnosis who were compensated at time of 1L). Survival since date of 1L was compared using Cox proportional hazard analysis.

Results and Discussion: Overall, 306 patients received 1L, including sorafenib 60.5%, atezolizumab + bevacizumab 29.7%, lenvatinib 9.1%, and nivo/pembrolizumab 0.6%. Of these, 83.3% presented cirrhosis. Median 1L treatment duration was 5.1 months with a median overall survival since 1L of 16.0 months (range 12.9–18.3). Significant differences were observed between CC (n=167), DC (n=31) and RC (n=42) groups (Table). In the RC group, median time from decompensation to recompensation was 12.0 months (range 1.9–25.9); being ascites the most frequent event (78.6%). DC group presented decreased post-1L survival [median 8.6 months vs 17.2 months in CC [adjusted HR 1.9 (95% CI 1.05–3.5); $P=0.03$], while no significant survival difference was observed between RC and CC [median survival 12.5 months; aHR 1.3 (95% CI 0.81–2.1); $P=0.28$] (Figure). Lower access to second line therapy was observed in DC group.

Conclusions: Patients with cirrhosis and advanced HCC who achieve recompensation may benefit from systemic therapies. This demands an observation period of follow up before precluding 1L in decompensated cirrhosis.