

¹⁸ Department of Hepatology, Asian Institute of Gastroenterology, Hyderabad, India

¹⁹ Gastroenterology Department, Gastroenterology Institute Bolivian-Japanese, La Paz, Bolivia

²⁰ Division of Hepatology, Gastroenterology and Liver Transplantation, Department of Internal Medicine II, Slovak Medical University, F. D. Roosevelt University Hospital, Banska Bystrica, Slovak Republic

²¹ Hepatology, Centre Hospitalier de Luxembourg, Luxembourg

²² Liver Unit, Department of Digestive Diseases University General Hospital Gregorio Marañón Madrid, Spain; Ciberehd Biomedical Research Center in the Liver and Digestive Diseases Network Madrid, Spain

²³ Division of Gastroenterology and Hepatology, Montefiore Medical Center, Bronx, NY, USA

²⁴ Hepatology Section, Gastroenterology Hospital Dr. Carlos Bonorino Udaondo, Buenos Aires, Argentina

²⁵ Hepatology Unit, Pablo Tobon Uribe Hospital, university of Antioquia, Medellín, Colombia

²⁶ Hepatology and Liver Transplant Unit, Hospitals of San Vicente Fundación, Medellín-Rionegro, Antioquia, Colombia

²⁷ gastroenterology Department, San Juan de Dios Hospital, Santiago, Chile

²⁸ Hepatology and Liver Transplant Unit, Austral University Hospital, Pilar, Argentina

²⁹ Central Hospital San Luis, San Luis, Argentina

³⁰ Liver Unit, Buenos Aires Italian Hospital, Buenos Aires, Argentina

³¹ Liver Unit, Rosario Private Hospital, Rosario, Argentina

³² Liver Unit, Hospital Vall D'hebron, Universitat Autònoma Barcelona, Ciberehd, Barcelona, Spain

³³ Section of Digestive Diseases, Yale University School of Medicine/VA-CT Healthcare System, New Haven/West Haven, USA

³⁴ Division of Gastroenterology, Liver Unit, University of Alberta, Edmonton, Canada

³⁵ Division of Gastroenterology, Department of Medicine, Schulich School of Medicine, Western University & London Health Sciences Centre, London, Ontario, Canada

³⁶ Department of Epidemiology and Biostatistics, Schulich School of Medicine, Western University, London, Ontario, Canada

Introduction and Objectives: Alcohol-associated hepatitis (AH) is a severe entity associated with high mortality. Corticosteroids might be used in cases with severe disease and several dynamic models can predict mortality and response to corticosteroids in AH patients. However, there is no consensus on the best of them. This study aimed to evaluate dynamic models to predict response to corticosteroid treatment based on short-term mortality in patients with severe AH based on a worldwide cohort.

Materials and Methods: A retrospective cohort study of patients with severe AH (between 2009 – 2019). We included patients who received corticosteroid treatment and calculated the Lille model of day 4 (Lille-4), day 7 (Lille-7) (cut-off value ≥ 0.45), and the Trajectory of Serum Bilirubin (TSB) (cut-off value ≥ 0.8 of the ratio between bilirubin at admission and day 7) to predict mortality. We estimated up to 30-day survival using Kaplan-Meier curves, and we performed multivariable analyses using Cox regression. Specifically, we constructed two models to compare Lille-4 vs. TSB and Lille-7 vs. TSB, adjusting by well-known clinical variables associated with higher mortality in AH (age, sex, and creatinine at admission).

Results: 1,066 patients were included (30 centers, 10 countries), age 47.7 ± 10.9 years, 30% women. The MELD score on admission was 25 [21-30]. Responders were considered by Lille-4 49.1%, Lille-7 46.6%, and TSB 55.4%. In the first Cox regression, we observed that Lille-4 and TSB predicted 30-day mortality (HR 3.0, 95%CI: 1.7-5.1; $p < 0.0001$, and HR 2.1, 95%CI: 1.3-3.5; $p = 0.005$, respectively) (Table A). In the second Cox regression, Lille-7 also predicted 30-day mortality (HR 3.7, 95%CI: 2.1-6.7; $p < 0.0001$) but not TSB (HR 1.5, 95% CI: 0.8-2.6; $p = 0.180$) (Table B). Creatinine at admission was also statistically significant in both Cox-regressions.

Conclusions: Different dynamic models can determine the response to corticosteroids in patients with severe AH. However, Lille-7 and Lille-4 have the best performance. New models are needed for better prognostication in AH.

Table 1: Models to compare Lille-4 vs. TSB (Table A) and Lille-7 vs. TSB (Table B)

Table A Variable	Hazard Ratio	P value	95 % Conf. Interval
Age	0.999	0.933	0.98 - 1.01
Gender	0.954	0.829	0.62 - 1.45
Creatinine in Admission	1.195	0.00	1.08 - 1.31
Lille- 7 Response	3.706	0.00	2.05 - 6.68
TSB Response	1.476	0.180	0.83 - 2.60

Table B Variable	Hazard Ratio	P value	95 % Conf. Interval
Age	0.999	0.948	0.98 - 1.01
Gender	0.911	0.678	0.58 - 1.40
Creatinine in Admission	1.193	0.001	1.07 - 1.31
Lille- 4 Response	2.99	0.00	1.74 - 5.14
TSB Response	2.08	0.005	1.25 - 3.45

<https://doi.org/10.1016/j.aohep.2023.101035>

O-26 IMMUNE PROFILING PROVIDES A SET OF 5 CYTOKINES TO DETECT HEPATOCELLULAR CARCINOMA RELATED TO VIRAL HEPATITIS IN SOUTH AMERICAN PATIENTS

Jose Debes¹, Enrique Carrera Estupinan², Melina Rocio Ferreiro³, Angelo Mattos⁴, Domingo Balderramo⁵, Maria Massotti⁶, Joe Koopmeiners⁷, Andre Boonstra⁸

¹ Department of Medicine, University of Minnesota, Minneapolis, MN, USA

² Department of Gastroenterology, Eugenio Espejo Hospital, Quito, Ecuador

³ Department of Gastroenterology, Clinic National Hospital, Buenos Aires, Argentina

⁴ Department of Gastroenterology, Federal University of Health Sciences of Porto Alegre, Porto Alegre, Brazil

⁵ Department of Gastroenterology, University Private Hospital of Córdoba. University Institute of Biomedical Sciences of Córdoba, Argentina

⁶ Division of Biostatistics, University of Minnesota, Minneapolis, MN, USA

⁷ Division of Biostatistics, University of Minnesota, Minneapolis, MN, USA

⁸ Department of Gastroenterology, Erasmus MC, Rotterdam, the Netherlands

Introduction and Objectives: New peripheral markers are needed for the early detection of hepatocellular carcinoma (HCC). Currently, the only accepted biomarker is alpha-fetoprotein (AFP) which by itself is suboptimal for early HCC detection. We investigated

peripheral immune markers to detect HCC in a large cohort of South American patients and a sub-group of viral hepatitis-related HCC.

Materials and Methods: Through the ESCALON network, we prospectively evaluated 127 individuals with HCC and 113 cirrhotic controls from 3 countries in South America (Argentina, Brazil and Ecuador). 42% of HCC cases were related to viral hepatitis B or C. Blood samples were analyzed for 37 unique interleukins, chemokines and growth factors using a multiplex Bio-Rad platform. We used leave-one-out cross-validation (LOOCV) to compute a ROC curve.

Results: Median age for HCC patients was 68 y/o and for controls 62 y/o. 70% of cases and 55% of controls were males. 55% of HCCs were under 5cm in diameter. The most common causes of HCC were viral hepatitis (42%) and NAFLD (23%). Twenty-two markers showed a significant difference between cases and controls. Three markers (IL-12p40, Beta-NGF and Gro-alpha) were exclusively dysregulated in viral hepatitis related HCC compared to other HCCs. From all causes of HCCs, we identified five cytokines (MIP-3a, MIG, CCL-25, MDC, and HGF) that were differentially regulated in HCCs compared to cirrhotic controls. ROC analysis of the top-5 markers in HCC cases exclusively related to viral hepatitis showed an AUROC of 0.816 (CI 0.783-0.886). The same panel applied to HCC <5cm related to viral hepatitis showed an AUROC of 0.751 (CI 0.671-0.832).

Conclusions: Our study identified a set of 5 cytokines in South American patients that can differentiate HCC from cirrhosis controls in patients with viral hepatitis. The 5 cytokines showed a lower prediction power for HCCs <5cm (likely due to the small size of this cohort).

<https://doi.org/10.1016/j.aohep.2023.101036>

O-30 ALCOHOL-HARM PARADOX IN LATIN AMERICA: HOW TO STUDY IT DESPITE DATA LIMITATIONS? THE CHILEAN EXPERIENCE

Juan Pablo Roblero¹, Pablo Roblero²,
Juan Pablo Arab³, Jaime Poniachik¹, Ramon Bataller⁴,
Luis Antonio Díaz³

¹ Department of Medicine, University of Chile, Santiago, Chile

² Sociology Institute, Pontifical Catholic University of Chile, Santiago, Chile

³ Gastroenterology Department, Pontifical Catholic University of Chile, Santiago, Chile

⁴ Center for Liver Diseases, University of Pittsburgh, Pittsburgh, United States

Introduction and Objectives: Research on the “Alcohol-Harm Paradox” (AHP) investigates why low-income individuals have more alcohol-related harm despite lower alcohol consumption (AC). Possible explanations have been evaluated in Europe and the US, but data constraints make it difficult in Latin America (LATAM). This study aimed to design a strategy to study the AHP in LATAM’s restricted-data context, recognize its strengths and limitations, and identify possible explanations in the Chilean experience. The AHP is expected to be explained by the unequal distribution of comorbidities, risk behaviors, consumption patterns, rurality, education, access to health, social capital, and mental health.

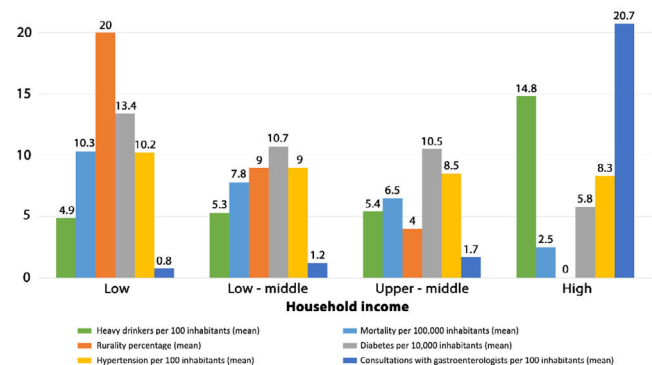
Materials and Methods: We first evaluated our hypothesis at the individual level with data from the 2016-17 National Health Survey. We conducted logistic regression models to assess whether the hypothesized explanatory factors mediated the effect of AC on liver disease. Second, we aggregated at the municipal level registry data on deaths from alcohol-related liver disease (Ministry of Health Statistics) and survey data on AC and the hypothesized explanatory factors (National Drug Survey and National Survey of Socioeconomic

Characterization) to test our hypothesis using mortality as the outcome of negative binomial regression models.

Results: The first analysis suggests that the AHP exists among Chilean men and it is explained by the unequal distribution of metabolic syndrome, diabetes, obesity, smoking, heavy episodic drinking, rurality, education, social support, and depression. The second analysis reinforces these findings and highlights the explanatory potential of healthcare-access inequality (Figure 1).

Conclusions: The proposed analyzes support our hypothesis in Chile. They can be replicated in other LATAM countries as an effective restricted-data strategy to start investigating the AHP. However, cross-sectional survey analyzes are limited by reverse causation and aggregate data analyzes by ecological fallacy. Better access to administrative data with patient identifier is needed to generate accurate longitudinal evidence on the explanatory mechanisms.

Figure 1. Variation in the explanatory factors of the AHP according to the median household income of the municipalities



<https://doi.org/10.1016/j.aohep.2023.101037>

O-31 DIAGNOSTIC PERFORMANCE OF BAVENO VII CRITERIA FOR EXCLUSION OF ESOPHAGEAL VARICES: A RETROSPECTIVE STUDY

Williams Celedonio Campos¹,
Celide Campoverde-Cueva², Brayan Campos³,
Rommel Zambrano-Huaila¹, Alejandra Zevallos²,
Jorge Garavito-Renteria^{1,2}

¹ Liver Unit, Gastroenterology Service, Arzobispo Loayza National Hospital, Lima, Perú

² Professional School of Human Medicine, Private University San Juan Bautista, Lima, Perú

³ Department of Neurobiology, Duke University, NC, United States

Introduction and Objectives: Cirrhosis is the main cause of patient hospitalization and esophageal variceal bleeding is the most serious decompensation. In recent years, transient elastography (TE) has been shown to be a useful tool for the diagnosis and management of esophageal varices (EV). The purpose of this study was to validate the Baveno VII criteria in patients with chronic liver disease in order to exclude the presence of EV.

Materials and Methods: A retrospective study was conducted with cirrhotic patients who underwent upper endoscopy and TE from January 2017 to December 2019. ROC analyses were conducted to determine cut-off values for ruling out EV. We evaluated the performance of the Baveno VII criteria (liver stiffness measurement (LSM) <15 kPa and platelet count >150 × 10⁹ cells/L) for the identification of EV and sparing endoscopies.