[9] *Carlos Andrade Marín Hospital, Quito, Ecuador*
[10] *Austral University Hospital, Buenos Aires, Argentina*
[11] *Pablo Tobon Uribe Hospital, Medellin, Colombia*
[12] *Federal University of Santa Catarina, Florianópolis, Santa Catarina, Brasil*
[13] *Del Salvador Hospital, Santiago, Chile*
[14] *Guillermo Almenara National Hospital, Lima, Perú*
[15] *General Plaza de la Salud Hospital, S. Domingos, República Dominicana*
[16] *RA Calderón Guardia Hospital, San Jose, Costa Rica*
[17] *State University of Campinas, Campinas, Brasil*
[18] *Francisco Clinic, Quito, Ecuador*
[19] *Red de Salud UC Christus, Santiago, Chile*
[20] *National Liver Transplant Program, Montevideo, Uruguay*
[21] *Alemana Clinic, Santiago, Chile*

Introduction and Objectives: Little is known about current practice of liver transplantation (LT) in Latin American countries (LATAM). This study aimed to describe LT activity, immunosuppression protocols and policies regarding prophylaxis of cytomegalovirus (CMV) infection and hepatitis B virus (HBV) recurrence in different active LATAM centers.

Materials and Methods: A web-based survey with 20 questions regarding LT practice was sent to all members of ALEH LT SIG in December 2022.

Results: 22 centers performing 35 [5-160] LT per year from Brazil (n=5), Argentina (n=4), Chile (n=4), Ecuador (n-2), Mexico (n=2), Colombia (n=1), Costa Rica (n=1), Peru (n=1), Dominican Republic (n=1) and Uruguay (n=1) answered the survey. Tacrolimus, mycophenolate and prednisone was the main immunosuppressive regimen employed by most (72%) centers and 81% of them referred basiliximab use for induction therapy in selected patients. Tailoring of immunosuppression was universally accepted, particularly in autoimmune hepatitis (AIH) (59%), hepatocellular carcinoma (54%) kidney dysfunction (77%) and primary biliary cirrhosis (33%). Weaning of corticosteroids at three, six and 12 months after LT was reported, respectively, by 41%, 36% and 23% of the centers, but policy for lifelong corticosteroid use in AIH-transplanted subjects was commonly observed (90%). Just four centers are currently performing protocol liver biopsies, while 18 of them are considering liver biopsy prior to steroid pulse therapy. HBIG and nucleos(t)ide analogs are used in most instances (73%) for HBV recurrence prevention, whereas CMV infection prophylaxis was shown to vary sharply across centers. Of note, all but two of them referred major changes in LT practice over the years due to economical restraints.

Conclusions: Compliance with standard of care recommendations for management of LT was reported by most centers. Heterogeneity in practices regarding HBV infection recurrence and CMV prophylaxis may reflect local financial restraints and point to the importance of developing ALEH guidelines to encourage LT activity in LATAM.

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O- 8 PNPLA3 RS738409 C>G POLYMORPHISM IMPACT ON HCV-RELATED HCC DEVELOPMENT IN THE BRAZILIAN POPULATION: PRELIMINARY RESULTS

Claudia Maccali, Aline L Chagas, Lisa Rc Saud, Regiane Ssm Alencar, Michele Sg Gouvea, Joyce Mks Etto, Isabel V Pereira, Arthur In Oliveira, Jose T Stefano, Rafael Sn Pinheiro, Wellington Andraus, Paulo Herman, Luiz Ac D'albuquerque, Mario G Pessoa, Claudia P Oliveira

Department of Gastroenterology, University of São Paulo School of Medicine, São Paulo, Brasil

Introduction and Objectives: The PNPLA3 rs738409 C>G polymorphism has been associated with hepatocellular carcinoma (HCC) and liver cirrhosis regardless of the etiology, although the association was stronger with non-viral etiologies. However, the influence of PNPLA3 polymorphism on Hepatitis C Virus (HCV) and whether this polymorphism could be a risk factor for HCV-related HCC is not well defined. We aimed to evaluate the influence of the PNPLA3 rs738409 C>G polymorphism on the risk of HCC occurrence in HCV patients in Brazil.

Materials and Methods: This study included 90 patients with HCV-related cirrhosis and HCC who underwent liver transplantation or resection at a tertiary center in Brazil and 111 patients non-HCC with HCV-related cirrhosis, as the control group. The rs738409 polymorphism was detected in the DNA extracted from patients' blood samples using the TaqMan assay. All clinical data were collected using the Research Electronic Data Capture (REDCap) tool. The statistical analyses were performed using Jamovi software version 2.3.23.

Results: In the HCV+HCC group there was a higher proportion of male gender (79.1% vs. 45.9%, p<0.001), history of alcoholism (80.5% vs. 22.5%, p<0.001) and smoking (68.9% vs. 25.2%, p<0.001), however there was no statistical difference in age (p=0.519) and BMI (p=0.403) between both groups. The genotype frequencies of the rs738409 polymorphism in the HCV+HCC group was CC 41,2% CC and CG/GG 58,8% vs. controls CC 49,5% and CG/GG 50,5%. The presence of the G allele was not an independent factor associated with the risk of HCC occurrence (r=0,199, p=0.53).

Conclusions: Even in an admixed population such as the Brazilian, there was no association between the PNPLA3 rs738409 C>G polymorphism and the risk of developing HCV-related HCC, as previously shown in published studies in caucasian and oriental population.

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O-9 PRESENCE OF METABOLIC ASSOCIATED LIVER DISEASE IN AUTOIMMUNE HEPATITIS IS ASSOCIATED WITH ADVANCED LIVER FIBROSIS

Giselle Arévalo[1], Daniela Simian[1], Jorge Chiong[2], Consuelo Palomo[1], Laura Carreño[1], Jaime Poniachik[1], Álvaro Urzúa[1]

[1] *Sección de Gastroenterología, Hospital Clínico Universidad de Chile, Santiago, Chile*
[2] *Facultad de Medicina, Universidad de Chile, Santiago, Chile*

Introduction and Objectives: Metabolic associated liver disease (MAFLD) is one of the most frequent causes of chronic liver disease worldwide. Steatohepatitis is a major risk factor for fibrosis and its progression. MAFLD might be present in other causes of chronic liver damage, such as autoimmune hepatitis (AIH). There is scarce information of the role of MAFLD in fibrosis in patients with AIH. This study aimed to describe frequency of steatosis, steatohepatitis and fibrosis in AIH liver biopsies and the impact of steatosis and steatohepatitis on fibrosis severity and survival.

Materials and Methods: Observational, retrospective study of liver biopsies performed prior to initiation of immunosuppressive therapy, between 2014 and 2019. Presence of steatosis, steatohepatitis and fibrosis was recorded. Alcohol and other etiologies of liver disease were excluded. Clinical data was obtained from electronic charts and outcome variables by telephone contact. Chi2 and exact Fisher test and Odds Ratio (OR) were performed ($p < 0.05$ considered statistically significant).

Results: 131 biopsies were analyzed; 76% were female, mean age 56 (15-83) years. Steatosis was present in 27%, steatohepatitis in 14% and advanced fibrosis (≥ F3) in 60%. All patients with steatohepatitis had advanced fibrosis (≥ F3). Presence of steatosis was an independent risk factor for advanced fibrosis (OR 2.97, CI95% 1.22 – 7.21 p = 0.016) and mortality (OR 2.60 (IC95% 1.08 – 6.29), p = 0.033). We performed a sub-analysis including only 66 patients with follow-up were decompensations and hospitalizations were no different between the 2 groups.

Conclusions: In this cohort of autoimmune hepatitis liver biopsies, steatosis and steatohepatitis were risk factors for advanced fibrosis. All patients with steatohepatitis had advanced liver fibrosis and mortality was higher in patients with steatosis. In patients with AIH, MAFLD should be treated to avoid progression to fibrosis.

N = 131	Steatosis n = 35	Without steatosis n = 96	Valor p
Female gender	26 (74)	73 (76)	0.836
Age (median; min – max)	56 (21 – 82)	56 (15 – 83)	0.727
Body Mass Index (median; min – max)	31.2 (22.9 – 43.15)	26 (19 – 44.4)	**0.0002**
Comorbidities			
Diabetes	11 (32)	13 (15)	**0.036**
Hypertension	13 (38)	22 (25)	0.158
Laboratory (median; min – max)			
Bilirubin	1.3 (0.2 – 6.3)	1.6 (0.2 – 33)	0.109
INR	1.16 (1 – 2.08)	1.19 (0.9 – 2.28)	0.809
Platelets	133000 (46000 – 287000)	193000 (48000 – 491000)	**0.006**
Advanced fibrosis (≥ F3)	27 (77)	51 (53)	**0.013**
Mortality	12 (34)	16 (17)	**0.030**

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O-10 VALIDATION OF SERUM BIOMARKER PANELS FOR EARLY HCC DETECTION: RESULTS FROM A LARGE PROSPECTIVE LATIN AMERICAN MULTICENTER STUDY

Boris Beudeker[1], Siyu Fu[1], Domingo Balderramo[2], Angelo Mattos[3], Spencer Goble[4], Enrique Carrera[5], Javier Diaz[6], Jhon Prieto[7], Marco Arrese[8], Arndt Vogel[9], Jesus Banales[1], Jeffrey Oliveira[1], Anthonie Groothuismink[1], Gertine van Oord[1], Robert de Man[1], Jose Debes[11], Andre Boonstra[1,10]

[1] *Department of Gastroenterology and Hepatology, Erasmus MC University Medical Center, Rotterdam, Países Bajos*
[2] *Department of Gastroenterology, Hospital Privado Universitario de Córdoba, Córdoba, Argentina*
[3] *Department of Hepatology, Federal University of Health Sciences of Porto Alegre, Porto Alegre, Brasil*
[4] *Internal Medicine, HCMC, Minneapolis, Estados Unidos*
[5] *Department of Gastroenterology and Hepatology, Hospital Eugenio Espejo, Quito, Ecuador*
[6] *Department of Gastroenterology and Hepatology, Universidad San Martin de Porres, Lima, Perú*
[7] *Department of Gastroenterology and Hepatology, Centro de Enfermedades Hepáticas y Digestivas, Bogota, Colombia*
[8] *Department of Gastroenterology, Pontificia Universidad Católica de Chile, Santiago, Chile*
[9] *Department of Gastroenterology and Hepatology, Hannover Medical School, Hannover, Alemania*
[10] *Department of Liver and Gastrointestinal Diseases, Health Research Institute - Donostia University Hospital, University of the Basque Country (UPV/EHU), San Sebastian, España*
[11] *Department of Medicine, University of Minnesota, Minneapolis, Estados Unidos (EEUU)*

Introduction and Objectives: New hepatocellular carcinoma (HCC) surveillance approaches including PIVKA-II, AFP, and the GALAD panel of serum biomarkers are linked to HCC, but inconsistent use in guidelines limits their value. This study aimed to determine the validity of known and novel serum biomarkers to detect liver cancer in a Latin American cohort.

Materials and Methods: In a multi-center study, 2045 patient samples were retrospectively or prospectively collected from 7 countries in Latin America and Europe and analyzed for cancer and liver disease etiology. The performance of multivariable models based on AFP and PIVKA was tested for early-stage HCC detection, low AFP HCC, 12 months pre-diagnostic HCC (n=92, range 9-15 months), and compared to cirrhosis and other liver tumors.

Results: The GALAD model showed excellent ability to differentiate HCC from liver cirrhosis in our prospective Latin American cohort, with an AUC of 87.9. Sub-analysis of early HCC still demonstrated excellent performance in Latin American cohort. A novel multivariable model was developed to detect early-stage HCC with low AFP levels, by combining sex, age, AFP, and PIVKA-II (also called GAAD), which resulted in AUC of 87.3. Both GALAD and GAAD effectively differentiated low AFP HCC from cirrhosis in both European and Latin American patients, with AUCs of 82.8 and 81.6, respectively. Importantly, GAAD differentiated non-cirrhotic HCC (n=243) from other malignant and benign liver tumors with an AUC of 91.9, and it was 100% sensitive and specific in hemangioma cases (n=64).

Conclusions: We validated for the first time the GALAD model in a large cohort of Latin American HCC patients. We demonstrated comparable performance of GALAD model with the GAAD model developed on data from our European Latin American cohorts. Our findings provide additional information for consideration of these markers in international guidelines for HCC surveillance.

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O-11 ENHANCEMENT OF F4/80+CD11B-CD206+ KUPFFER CELLS IN LIVER TISSUE: EFFECT OF MARESIN-1 AS HEPATOPROTECTIVE AGENT

Jessica Zuñiga[1], Andres Herrada[2], Francisca Herrera[1], Alexandra Olate[2]

[1] *Lab. Farmacología, Departamento de Ciencias Básicas Biomédicas, Universidad de Talca, Talca, Chile*
[2] *Instituto de Ciencias Biomédicas, Universidad Autónoma de Chile, Talca, Chile*

Introduction and Objectives: Chronic liver diseases (CLD) are a major global health burden and are the 11th leading cause of death and the 15th cause of morbidity worldwide. CLD could be associated with steatosis and fibrosis and progress to cirrhosis, with the concomitant liver failure. Currently, there is no approved treatment and it is only recommended to eliminate the causative agent or give palliative treatments. The immune response, particularly hepatic macrophages (Kupffer cells), play a fundamental role in the development of liver disease. It is known that well-differentiated populations coexist in the liver, including: F4/80+CD11b- (sessile) and F4/80+CD11b+ (migrated from bone marrow). These populations could be modified their phenotype from M1 (inflammatory) to M2 (anti-inflammatory), which is of pharmacological interest. We aimed to study the administration of Maresin-1, a derivative of omega-3 fatty acids, promote a restorative state by an increase in the CD206+CD86-CD11c- i.e. M2 Kupffer cell population.