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

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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

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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.