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AUTOIMMUNE HEPATITIS IN LATIN AMERICA: INSIGHTS FROM THE ALLATIN COHORT

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Introduction and Objectives: Autoimmune hepatitis (AIH) is a chronic inflammatory liver disease associated with significant morbidity. Non-white ethnicity has been described as an independent predictor of adverse outcomes. Previous studies suggest a more severe disease phenotype in Latin America. We aim to describe the presentation, treatment, and outcomes of AIH in Latin America.

Materials and Methods: Retrospective, ongoing, multicenter cohort study (ALLATIN) including 515 patients with autoimmune hepatitis from Brazil (246), Argentina (108), Chile (71), Ecuador (28), Cuba (22), Mexico (21), Costa Rica (10), and Peru (1).

Results: Most patients were female (82.5%), with type 1 AIH (90.9%) and a mean age at diagnosis of 42.8±19.2 years. At disease presentation, the most reported symptom was jaundice (42.3%), followed by asthenia (25.3%), abdominal pain (19.8%), arthralgia (10.0%) and pruritus (9.8%). Clinical signs of portal hypertension were seen in 16.1% at diagnosis. Acute presentation occurred in 39.3%, predominantly as acute icteric hepatitis(72.2%), while 42% were asymptomatic. At the first biopsy, 42.9% of patients had advanced fibrosis (F3–F4), 35.0% were cirrhotic on ultrasound, and 26.6% had clinically significant portal hypertension. The preferred first line therapy was prednisone (96.5%) and azathioprine (91.9%). Biochemical remission was achieved in 68.4% (data from 336 patients) at 6 months and 55.7% at 12 months and 55.7% (data from 329 patients) at 12 months. Among patients who achieved a biochemical response within the first year, most responded within the first 6 months. Reported second-line therapies were mycophenolate mofetil (63.6%), tacrolimus (13.6%), cyclosporine (13.6%), chloroquine (6.8%), and rituximab (2.3%). In a mean follow up of 6.72±6.0 years, 3.9% underwent liver transplantation and 3.1% died.

Conclusions: Despite a high burden of advanced liver disease at presentation, the ALLATIN cohort shows comparable treatment response rates to European populations. These findings highlight the importance of ethnicity, healthcare access, and early diagnosis in shaping AIH outcomes.

Conflict of interest: None

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PROTECTIVE ROLE OF MTARC1 RS2642438 POLYMORPHISM AGAINST FIBROSIS PROGRESSION IN METABOLIC DYSFUNCTION-ASSOCIATED STEATOTIC LIVER DISEASE (MASLD)

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Introduction and Objectives: Metabolic dysfunction-associated steatotic liver disease (MASLD) is a leading cause of chronic liver disease worldwide, with fibrosis progression being a key determinant of clinical outcomes. Genetic polymorphisms, such as MTARC1 (rs2642438)**,** have been implicated in modifying fibrosis risk. This study aimed to evaluate the protective role of **the** MTARC1 AA genotype in protecting against significant fibrosisin Brazilian patients with MASLD.

Patients and Methods: A total of 212 biopsy-proven MASLD patients were included, classified into, Group 1 (F0-F1, n=110): No significant fibrosis. Group 2 (F2-F4, n=102): Significant fibrosis. Additionally, 90 healthy individuals served as controls. Genotyping for MTARC1 (rs2642438) was performed using real-time PCR. Logistic regression models were used to assess the association between MTARC1 genotypes and significant fibrosis, adjusting for metabolic and clinical factors.

Results: The protective AA genotype was significantly more frequent in the control group (52%) than in MASLD patients (5%, $p = 2.76 \times 10^{-20}$)**.**

Patients carrying the AA genotype had an 81% lower risk of developing significant fibrosis (OR = 0.19; 95% CI: 0.08 - 0.45; $p < 0.001$).

No significant associations were observed for the GG and GA genotypes regarding fibrosis progression.

Conclusions: The MTARC1 rs2642438 AA genotype confers strong protection against fibrosis in MASLD patients, suggesting a potential role in risk stratification and personalized management**.** Future studies with larger cohorts are needed to confirm these findings and elucidate the molecular pathways involved.

Conflict of interest: None