

on biopsy. IH was associated with a greater presence of fibrosis (73% vs 36%; $p < 0.05$) and higher ANA titers $> 1/640$ (50% vs 17%). In 24 patients, annual response to UDCA could be evaluated: 45% of the patients with IH met at least 2 response criteria vs 69% of the group without histological IH.

Conclusion: The presence of IH in PBC is associated with greater fibrosis and worse biochemical response. Liver biopsy may be necessary in patients who do not respond to UDCA due to suspected interface activity.

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P-127 HEPATITIS A WITH ACALCULOUS CHOLECYSTITIS, PLEURAL EFFUSION, PERICARDIAL EFFUSION AND ASCITIS

Aileen Mateo, Eliam Rivas

Department of Gastroenterology, CEDIMAT, Santo Domingo, Dominican Republic

Introduction: In developing countries, hepatitis A constitutes an important public health problem. It generally presents as a mild, self-limited disease, but occasionally it can present with infrequent clinical findings.

Case Presentation: 42-year-old female admitted for 7 days of abdominal pain in the right upper quadrant, general malaise, mild dyspnea and fever. With no known morbid history, she is a body-builder with frequent use of BCAAs, Glutamine, and occasional anabolic steroids (Oxalandrone, Boldedone, Methenolone). On physical examination, jaundice of the skin and mucous membranes, edema of the lower limbs and palpable spleen, pain on palpation in the right upper quadrant.

In laboratories Hemoglobin 13.3 g / dL; White blood cells: 5400 cells / mm³; Platelets 316 / mm³; AST 849 U / L; ALT 3152 U / L; Total bilirubin 11.1 mg / gL, GGT 1020 U / L; 491 U / L alkaline phosphatase; INR 1.5; Total proteins 5.6 g / dL; Albumin 2.6 g / dL. Nitrogen, electrolytes, ANA antinuclear antibody, lipase, HIV, HVC, HBsAg were within normal parameters, HAV IgM was positive, the serological analysis for E. Barr, Cytomegalovirus were negative. Chest X-ray showed mild bilateral pleural effusion. Liver Doppler showed inflammatory changes in the liver parenchyma, data of acalculous cholecystitis, moderate ascites and splenomegaly. An echocardiogram was performed that showed slight posterior pericardial effusion with a heart of normal morphology and function. The patient was managed with supportive and nutritional therapy, without progression of clinical symptoms, was discharged on the 8th day of admission and was seen for consultation 1 month later with image and laboratory control, with disappearance of cholecystitis, pleural and pericardial effusion and ascites, and decreased laboratory levels of the liver profile.

Conclusion: This is one of the few documented cases of hepatitis A with these complications, all found in the same patient, they have been described as rare forms of presentation in the course of hepatitis A.

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O-1 THE ROLE OF PNPLA3 AND TM6SF2 POLYMORPHISMS ON LIVER FIBROSIS AND METABOLIC ABNORMALITIES IN BRAZILIAN PATIENTS WITH CHRONIC HEPATITIS C

Arthur Ivan N. Oliveira¹, Fernanda M. Malta¹, Patricia Momoyo Y. Zitelli¹, Ana Paula M. Salles¹,

Michele S. Gomes-Gouvea¹, Ana Catharina S. Nastro², Joao Renato R. Pinho¹, Flair J. Carrilho¹, Claudia P. Oliveira¹, Maria Cássia Mendes-Corrêa², Mario G. Pessoa¹, Daniel F. Mazo^{1,3}

¹ Division of Clinical Gastroenterology and Hepatology, LIM07, Department of Gastroenterology, University of São Paulo School of Medicine (FMUSP), Sao Paulo, Brazil. Av. Dr. Enéas de Carvalho Aguiar n° 255, Instituto Central, # 9159. ZIP code: 05403-900, São Paulo, Brazil

² Department of Infectious Diseases, University of São Paulo School of Medicine (FMUSP), São Paulo, Brazil, Av. Dr. Enéas de Carvalho Aguiar n° 255, ZIP code: 05403-900, São Paulo, Brazil

³ Division of Gastroenterology (Gastrocentro), School of Medical Sciences, University of Campinas (UNICAMP), Campinas, Brazil, Rua Carlos Chagas n° 420. ZIP code: 13083-878, Campinas, Brazil

Background: Despite the growing body of knowledge about TM6SF2 and PNPLA3 polymorphisms in non-alcoholic fatty liver disease, their influence in the spectrum of HCV liver disease is not yet fully defined. Besides that, admixed populations, such as Brazilians, were not included in most of the studies.

Objectives: Describe the prevalence of these polymorphisms in Brazilians with chronic hepatitis C, and to assess their association with liver fibrosis and other components of the metabolic syndrome.

Methods: This cross-sectional study enrolled 365 treatment-naïve patients with HCV and 134 healthy individuals. TM6SF2 (rs58542926 c.499C>T) and PNPLA3 (rs738409 c.444C>G) polymorphisms were evaluated regarding their association with clinical and laboratory data, histological liver steatosis and fibrosis, and with components of the metabolic syndrome.

Results: In HCV subjects, the frequencies of TM6SF2 CC and CT+TT were 89% and 11%, while PNPLA3 frequencies of CC and CG+GG were 51.4% and 48.6%. In the univariate logistic regression analysis, the TM6SF2 CT+TT genotype in HCV was associated with significant liver fibrosis ($p = 0.047$; OR:1.953; 95% CI:1.009-3.788) however it was not confirmed by multivariate analysis. In comparison to the CT+TT genotype, the TM6SF2 CC genotype in HCV was associated higher frequency of arterial hypertension ($p = 0.032$), obesity ($p = 0.030$), metabolic syndrome ($p = 0.014$) and lower total cholesterol levels ($p = 0.036$). The PNPLA3 GG subjects had lower body mass index than CG/CC individuals ($p = 0.047$). None of the polymorphisms, or their combinations, was independently associated with hepatic steatosis.

Conclusion: In this evaluation of an admixed HCV population, neither TM6SF2 nor PNPLA3 polymorphisms were independently associated with hepatic steatosis or fibrosis.

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O-2 ROLE OF IMMUNE CHECKPOINTS AND ACTIVATED HELPER AND CYTOTOXIC T-CELLS IN DRUG-INDUCED LIVER INJURY (DILI)

Alejandro Cueto-Sanchez¹, Judith Sanabria Cabrera^{1,2}, Mercedes Robles-Diaz^{1,3}, Aida Ortega-Alonso¹, Miren Garcia Cortes^{1,3}, Enrique del Campo-Herrera¹, Rocio Gonzalez-Grande⁴, Miguel Jimenez-Perez⁴, Francisco Ruiz-Cabello⁵, M Isabel Lucena^{1,2,3}, Camilla Stephens^{1,3}, Raúl J Andrade^{1,3}