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Introduction: Primary biliary cholangitis (PBC) diagnosis is based on international criteria, which requires two of the following: (i) elevated alkaline phosphatase (AP), (ii) anti-mitochondrial antibody (AMA) and (iii) liver biopsy (BX) suggestive of PBC. It is still unclear if patients diagnosed by different criteria combinations present peculiarities, especially in highly-admixed populations.

Objectives: To investigate if patients diagnosed with PBC by different combinations of validated criteria present clinical or laboratory particularities.

Methods: The Brazilian Cholestasis Study Group database was reviewed to compare clinical, biochemical and histological characteristics of PBC between four groups diagnosed by: (1) AP ≥2x upper limit of normality (ULN) + presence of AMA, (2) AP ≥2x ULN + BX suggestive of PBC, (3) presence of AMA + BX suggestive of PBC and (4) all criteria.

Results: 482 patients with PBC were included (**Table 1**). Group-1 presented with higher levels of IgG, lower frequency of arterial hypertension (AH) and lower response to ursodeoxycholic acid (UDCA), while Group-2 had lower: age at diagnosis and HDL-C levels. Group-3 had higher: age at diagnosis, frequency of neoplasms, AH and response to UDCA; and lower: frequency of pruritus and jaundice, levels of aminotransferases, GGT and bilirubin, advanced liver disease and esophageal varices. Group-4 showed higher frequency of symptoms at presentation, especially pruritus.

Conclusion: PBC patients diagnosed by different combinations of established criteria may present singular features that can possibly impact in disease presentation and progression.

Table
Comparison of PBC Features according to Different Combinations of Diagnostic Criteria

Variables	Diagnostic Criteria				P-value
	Elevated AP + AMA positive	Elevated AP + BX positive	AMA positive + BX positive	All criteria	
Female sex	72 (96.0)	36 (94.7)	56 (94.9)	96 (96.0)	0.954[1]
Age (yr.)	63.1 ± 12.3	54.5 ± 13.9	63.9 ± 11.2	60.1 ± 11.3	**0.001**[3]
Age at diagnosis (yr.)	54.5 ± 13.9	48.6 ± 14.1*	57.6 ± 11.5*	52.3 ± 10.2	**0.023**[3]
Symptoms	54 (72.0)	26 (70.3)	25 (42.4)*	76 (76.0)*	**<0.001**[2]
Pruritus	38 (50.7)	20 (54.1)	16 (27.1)*	63 (63.0)*	**<0.001**[2]
Fatigue	27 (36.0)	17 (45.9)	15 (25.4)	42 (42.0)	0.155[1]
Jaundice	15 (20.0)	12 (31.6)	5 (8.5)*	28 (28.0)	**0.014**[2]
	25 (33.3)	15 (39.5)	29 (49.2)	40 (40.0)	0.327[2]
Extrahepatic manifestation					
Thyroiditis	12 (16.0)	6 (15.8)	16 (27.1)	19 (19.0)	0.374[2]
Rheumatoid Arthritis	4 (5.3)	2 (5.3)	4 (6.8)	3 (3.0)	0.676[1]
Sjögren Syndrome	4 (5.3)	4 (10.5)	5 (8.5)	12 (12.0)	0.494[2]
Neoplasms	2 (2.7)	3 (7.9)	9 (15.5)*	6 (6.0)	**0.046**[1]
Comorbidities					
Diabetes	11 (14.7)	7 (18.4)	5 (8.5)	11 (11.0)	0.454[2]
Arterial Hypertension	12 (16.0)*	10 (26.3)	22 (37.3)*	24 (24.0)	**0.044**[2]
Dyslipidemia	21 (28.0)	7 (18.9)	13 (22.0)	32 (32.0)	0.351[2]
Obesity	8 (10.7)	2 (5.3)	7 (11.9)	15 (15.2)	0.437[2]
Advanced Fibrosis	13 (44.8)	16 (48.5)	13 (23.6)*	41 (46.6)	**0.030**[2]
Laboratory					
AST/ULN	2.7 (1.8-3.6)	2.0 (1.5-3.5)	1.1 (0.8-1.6)	2.4 (1.6-4.4)	**<0.001**[4]
ALT/ULN	2.4 (1.3-4.4)	2.4 (1.5-3.8)	1.3 (0.9-1.9)	2.4 (1.6-4.2)	**<0.001**[4]
GGT/ULN	11.0 (5.8-21.7)	10.3 (5.8-17.9)	3.6 (1.7-7.9)	9.3 (5.1-15.8)	**<0.001**[4]
Total bilirubin (mg/dL)	1.1 (0.7-2.1)	1.2 (0.7-2.0)	0.7 (0.5-1.0)	1.0 (0.6-1.7)	**<0.001**[4]
Conjugated bilirubin (mg/dL)	0.7 (0.3-1.3)	0.6 (0.4-1.0)	0.3 (0.2-0.5)	0.5 (0.3-0.9)	**<0.001**[4]
Albumin (g/dL)	4.0 (3.5-4.2)	3.9 (3.6-4.2)	4.0 (3.8-4.3)	3.9 (3.5-4.2)	0.059[4]
Platelets count (10^9/L)	205 (145-276)	191 (112-265)	230 (200-276)	220 (168-272)	0.110[4]
Total cholesterol (mg/dL)	224 (191-265)	220 (191-251)	207 (185-231)	216 (191-267)	0.242[4]
HDL-cholesterol (mg/dL)	65 (48-82)	45 (36-71)	57 (50-69)	62 (48-80)	**0.012**[4]
Triglycerides (mg/dL)	111 (82-141)	92 (60-135)	111 (87-136)	111 (78-165)	0.493[4]
ANA ≥ 1:40	47 (66.2)	19 (52.8)	37 (71.2)	69 (70.4)	0.238[2]
ASMA ≥ 1:40	4 (6.5)	2 (6.1)	2 (4.2)	4 (4.6)	0.907[1]
IgG (mg/dL)	1702 (1344-1921)	1450 (1202-1755)	1421 (1218-1638)	1321 (1061-2248)	**0.032**[4]
IgM (mg/dL)	324 (210-478)	188 (123-387)	314 (191-454)	334 (205-438)	0.156[4]
Response Criteria					
Toronto	16 (40.0)*	11 (50.0)	24 (82.8)*	34 (52.3)	**0.005**[2]
Barcelona	30 (66.7)	19 (63.3)	18 (48.6)	48 (64.0)	0.344[2]
Paris-1	22 (48.9)	16 (55.2)	26 (70.3)	38 (50.7)	0.193[2]
Paris-2	9 (20.0)*	10 (34.5)	24 (64.9)*	21 (28.0)	**<0.001**[2]
POISE Trial	29 (64.4)	18 (60.0)	24 (64.9)	46 (61.3)	0.963[2]
Rotterdam	27 (61.4)	12 (48.0)	22 (68.6)	37 (59.7)	0.465[2]
Outcomes					
Liver cirrhosis	29 (45.3)	17 (47.2)	12 (20.7)*	35 (35.4)	**0.016**[2]
Esophageal varices	27 (43.5)	14 (50.0)	10 (20.8)*	29 (36.3)	**0.035**[2]
Transplantation	8 (10.7)	4 (10.5)	1 (1.7)	6 (6.0)	0.138[1]
Death	7 (14.9)	3 (9.4)	5 (11.1)	10 (12.7)	0.893[2]

AMA, anti-mitochondria autoantibody; ANA, anti-nuclear autoantibody; ASMA, anti-smooth muscle autoantibody; AP, alkaline phosphatase; AST, aspartate aminotransferase; ALT, alanine aminotransferase; BX, liver biopsy; HDL, high-density lipoprotein; IgG, immunoglobulin G; IgM, immunoglobulin M; ULN, upper limit of normality; Yr., years.*Variable associated with the statistically significant difference,[1]Fisher's test, [2]Chi-square test, [3]ANOVA, [4]Kruskal-Wallis test.

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O-11 REMODELING OF IMMUNOLOGICAL BIOMARKERS IN PATIENTS WITH CHRONIC HEPATITIS C TREATED WITH DIRECTACTING ANTIVIRAL THERAPY

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Background & Aims: The treatment of hepatitis C with DAAs has offered an opportunity to analyze the changes in the immune system caused by the rapid inhibition of viral replication. We sought to analyze the kinetics profiles of serum biomarkers in patients upon DAAs treatment.

Methods: 50 patients were enrolled before (baseline), during (W2-4 and W8-12 weeks) and post-treatment (W12-24 weeks) with sofosbuvir and daclatasvir ± ribavirin (n=36) or simeprevir (n=14). 15 uninfected blood donors formed the control group (NI). Serum biomarkers CXCL8, CCL11, CCL3, CCL4, CCL2, CCL5, CXCL10, IL-1β, IL-6, TNF-α, IL-12, IFN-γ, IL-15, IL-17, IL1Ra, IL-4, IL-5, IL-9, IL-10, IL-13, FGF-basic, PDGF, VEGF, G-CSF, GM-CSF, IL-7 e IL-2 were quantified by Luminex Bio-Plex Pro™. Mann-Whitney (HCV and NI), Kruskal Wallis (multiple), and Dunn (sequential in pairs) tests compared groups, with significance value if $p \leq 0.05$. The study was approved by ethical boards.

Results: At baseline, patients had high levels of chemokines, pro-inflammatory cytokines, and growth factors, with minor increase of regulatory cytokines. The kinetics timeline of baseline fold changes revealed early decline of CXCL8, CCL4, IL6, IL-15, IL-17, IL-9, GM-CSF and IL-7 at W8-12, and late remodeling of CCL3, CCL2, CCL5, IL1β, TNF-α, IL-12, IFN- γ, IL1-Ra, IL-4, IL-10, IL-13, PDGF, VEGF, G-CSF at W12- 24. Baseline ALT ≥69U/L, platelet ≤150,000/mm3 and cirrhosis were related to delayed remodeling in immune response.

Conclusions: The HCV eradication with DAAs results in profound readjustment of the microenvironment of serum immune biomarkers and may be slower in cirrhotic patients. These results add evidence to the knowledge of the process of immune remodeling associated with the rapid viral eradication of HCV with the DAAs.

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O-12 MIR-181A AS A BIOMARKER OF FIBROSIS IN NON-ALCOHOLIC FATTY LIVER DISEASE

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Introduction: Non-Alcoholic Fatty Liver Disease (NAFLD) covers a wide spectrum of disease, ranging from simple steatosis to cirrhosis. Liver biopsy is still the gold standard for assessing fibrosis, but there is a need to seek non-invasive biomarkers that can also be efficient in predicting fibrosis.

Objectives: To evaluate the role of microRNAs miR-21, miR-29a, miR-122, miR-155 and miR-181a in the phenotypic expression of NAFLD, correlating their serum levels with the different stages of the disease.

Methods: Cross-sectional study carried out on 108 NAFLD patients diagnosed by liver biopsy. In the histological analysis, the degrees of fibrosis and NAFLD activity score (NAS) were obtained. The FIB-4 and NAFLD fibrosis score were calculated and compared with the degree of fibrosis by biopsy. The comparison between the distributions of microRNA values according to the presence or absence of clinically expressed fibrosis (F2-4) was performed. The serum expression of microRNAs was also compared with the NAS of the biopsy. A multivariate logistic regression analysis was performed to build a score for predicting fibrosis using FIB-4 and Ln (miR-181a) as independent variables.

Results: Among the microRNAs studied, only miR-181a showed a statistical difference between patients with clinically expressed fibrosis and those without fibrosis (F0-F1) determined by liver biopsy ($p = 0.017$). FIB-4 revealed an AUC on the ROC curve of 0.667 to predict clinically expressed fibrosis (F2-F4). When assessed using the score in association with Ln (miR-181a), there was an improvement in the ROC curve, with AUC of 0.71. There was no correlation between the serum levels of microRNAs miR-21, miR-29a, miR-122, miR-155 and miR-181a with the degrees of inflammatory activity determined by NAS.

Conclusion: miR-181a can be used as a non-invasive method of predicting fibrosis in NAFLD, and an association of biomarkers has the potential to increase the accuracy of each method alone.

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O-13 Hepatocellular carcinoma patients are advantage in the Brazilian current liver transplant allocation system. A competing risk analysis. A RETROSPECTIVE STUDY

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Background: In Brazil, the Model for End-Stage Liver Disease (MELD) score is used to prioritize patients for deceased donor liver transplantation (DDLT). Patients with hepatocellular carcinoma (HCC) receive standardized MELD exception points to account for their cancer risk of mortality, which is not reflected by their MELD score.

Objective: To compare DDLT rates between patients with and without HCC in Rio Grande do Sul, the Southernmost state of Brazil.

Methods: We retrospectively studied 825 patients on the liver-transplant waiting list from January 1, 2007, to December 31, 2016, in a transplant center located in Porto Alegre, the capital of Rio Grande do Sul, to compare DDLT rates between those with and without HCC. The time-varying hazard of waiting list/DDLT was estimated, reporting the subhazard ratio (SHR) of waiting list/DDLT/dropout with 95% confidence intervals (CI). The final competing risk model was adjusted for age, MELD score, exception points, and ABO group.

Results: Patients with HCC underwent a transplant almost three times faster than patients with a calculated MELD score (SHR 2.64; 95% CI 2.10-3.31; $P<0.001$). The DDLT rate per 100 person-months was 11.86 for HCC patient's vs 3.38 for non-HCC patients. The median time on the waiting list was 5.6 months for patients with HCC and 25 months for patients without HCC.

Conclusion: Our results demonstrated that, in our center, patients on the waiting list with HCC have a clear advantage over candidates listed with a calculated MELD score.

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O-14 A SYNERGISTIC EFFECT OF PNPLA3 GENE POLYMORPHISM AND INSULIN RESISTANCE INCREASES THE RISK TO NON-ALCOHOLIC FATTY LIVER DISEASE IN PATIENTS WITH POLYCYSTIC OVARY SYNDROME

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