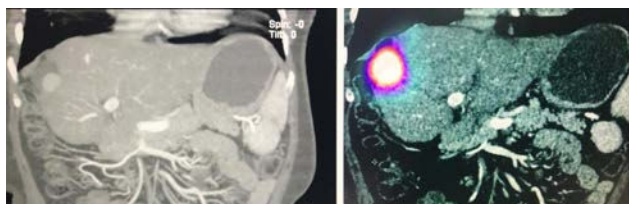


After RE-Y90, there were no complications and the patients were discharged after 24 hours.

Control Computed Axial Tomography was performed with good response, without disease progression at 3 and 6 months, asymptomatic.

Conclusions: RE-Y90 for the treatment of BCLC stage B HCC is a good therapeutic option in well selected patient.

Conflicts of interest: The authors have no conflicts of interest to declare.



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Aspartate aminotransferase as predictor of severity in SARSCoV-2 infection: linear regression model

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Background and aim: Some patients with SARSCoV-2 infection develop severe disease (SARS); however, the factors associated with severity are not yet fully understood. Some reports indicate that liver injury may be a poor prognostic factor. **AIM:** To identify the biochemical factors related to the development of SARS with mechanical ventilation (MV) requirement in patients with SARSCoV-2 and COVID-19.

Methods. Type of study: Observational. Cohort study. Procedure: Data from COVID-19 patients were collected at admission time to a tertiary care center. Differential factors were identified between seriously ill SARS+MV patients versus stable patients without MV. Transformation to the natural logarithm of significant variables was performed and multiple linear regression was applied, then a predictive model of severity called AAD (Age-AST-D dimer) was constructed.

Results: 166 patients were included, 114(68.7%) men, mean age 50.6 ± 13.3 years-old, 27(16.3%) developed SARS+MV. In the comparative analysis between those with SARS+MV versus stable patients without MV we found significant raises of ALT (225.4 ± 341.2 vs. 41.3 ± 41.1 ; $P=0.003$), AST 325.3 ± 382.4 vs. 52.8 ± 47.1 ; $P=0.001$), LDH (764.6 ± 401.9 vs. 461.0 ± 185.6 ; $P=0.001$), D dimer (7765 ± 9109 vs. 1871 ± 4146 ; $P=0.003$), age (58.6 ± 12.7 vs. 49.1 ± 12.8 ; $P=0.001$). The results of the regression are shown in the Table, where model 3 was the one that best explained the development of SARS+MV; with these variables was constructed the model called AAD, where: $[AAD = 3.896 + \ln(\text{age}) \times -0.218 + \ln(\text{AST}) \times -0.185 + \ln(\text{DD}) \times 0.070]$, where a value ≤ 2.75 had sensitivity = 0.797 and 1-specificity = 0.391, AUROC = 0.74 (95%CI:

0.62-0.86; $P < 0.0001$), to predict the risk of developing SARS+MV (OR = 5.8, 95%CI: 2.2-15.4; $P=0.001$).

Conclusions: Elevation of AST (probable marker of liver damage) is an important predictor of progression to SARS, together with elevation of D-dimer and age early (at admission) and efficiently predict which patients will potentially require MV.

Conflicts of interest: The authors have no conflicts of interest to declare.

Model	Non-standardized Coefficients	Standardized Coefficients	P	95% Confidence Interval for B		Collinearity statistics	
B	Error Desv.	Beta		Inferior limit	Superior limit	Tolerance	VIF
1 C	2.721	.131	.000	2.462	2.980		
AST	-.229	.033	-.512	-.293	-.164	1.000	1.000
2 C	3.161	.198	.000	2.770	3.551		
AST	-.194	.034	-.435	-.261	-.127	.878	1.139
DD	-.081	.028	-.221	-.135	-.026	.878	1.139
3 C	3.896	.414	.000	3.077	4.714		
AST	-.185	.034	-.413	-.252	-.118	.860	1.163
DD	-.070	.028	-.190	-.125	-.014	.844	1.185
Age	-.218	.108	-.148	-.433	-.004	.915	1.093

AST, aspartate aminotransferase; C, constant; DD, D dimer; VIF, variance inflation factors.

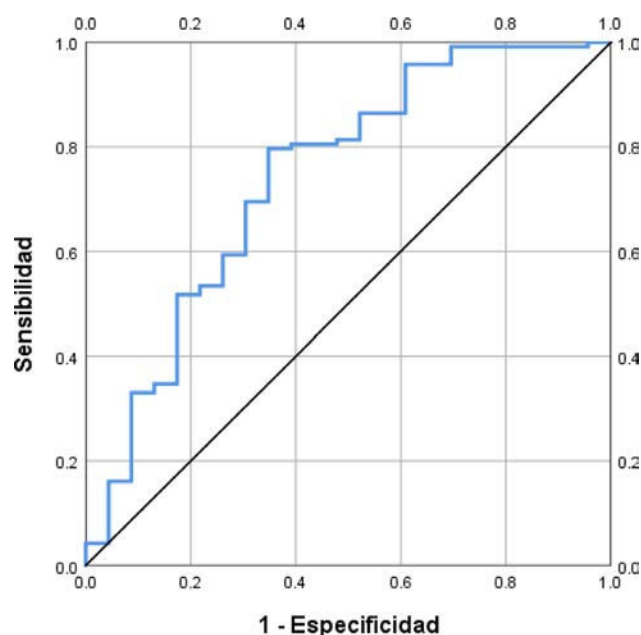
Resume of the model:

$R=0.512$, $r^2=0.262$, r^2 adjusted = 0.256, standard error = 0.331.

$R=0.552$, $r^2=0.305$, r^2 adjusted = 0.294, standard error = 0.322.

$R=0.570$, $r^2=0.325$, r^2 adjusted = 0.310, standard error = 0.318. Durbin-Watson = 1.53.

AAD MODEL TO PREDICT SEVERE FORM (SARS)+ INTUBATION



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Classification of alcohol consumption pattern in the Mexican population

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Background and aim: The evaluation of alcohol consumption is estimate by the evaluation of frequency and the concentration of

alcohol ingest. Until now has been accepted the use of several methods to determine the prejudicial and risk consumption. Also, it is possible to evaluate the control and abuse of the ingestion. Nevertheless, the broad spectrum of classification sometimes causes controversy to classify the alcohol consumption in the clinical. Aim: To design a guide to classify the pattern of alcohol consumption using social, clinical and biochemical information from a Mexican population.

Material and methods: Observational study. The subjects were classified according to alcohol consumption, using AUDIT test (Alcohol Use Disorders Identification Test), and the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV). The pattern of consumption was determined via the amount of alcohol in grams of alcohol per day and its equivalent in cups, frequency, as well as type of alcohol. Finally, the evaluation of liver damage considers the clinical and biochemical data referred in consultation. Protocol approved by the General Hospital of Mexico (HG/DI/16/107/03/082) and UNAM (FMD/DI/15/2019).

Results: Table. Classification of according to the pattern of alcohol consumption.

Conclusions: The pattern of alcohol consumption guide is a quick tool for the identification of prejudicial ingest of alcohol without evidence of any disease, this provides a first line to the proper diagnosis and management of patients with alcohol consumption and their future prognosis and treatment of liver alcohol diseases, which is prevalent in our country.

Conflict of interests: This work has been partially funded by CONACYT: SALUD-2016-272579 and none of the authors has a conflict of interest.

	CONTROL	RISK	ABUSE	ALCOHOLISM	ALCOHOL LIVER DISEASE CIRRHOSIS BY ALCOHOL
AUDIT DSM-IV	<8 Without abuse and dependence criteria	>8 Without abuse and dependence criteria	>8 With abuse criteria but not alcohol dependence (1 positive answer)	>8 With criteria of alcohol dependence (3 or more positive answers)	>8 With criteria of alcohol dependence (3 or more positive answers)
FREQUENCY	Occasionally	Consuetudinary Weekend	Consuetudinary Weekend 1 to 4 times per week	Daily, almost daily or Consuetudinary	Daily or almost daily
AMOUNT	1 cup <10 g	2-4♂ y 1-3♀ cups 40-60g y 20-40g	≥4-5♂ y ≥3-4♀ cups +60g y +40g	≥5♂ y ≥4♀ cups per 5 years +70g y +50g	≥5♂ y ≥4♀ cups per 5 years +70g y +50g
MAIN TYPE OF ALCOHOL		Ferment	Ferment, and distilled	Ferment, and distilled	Ferment, distilled, 96° alcohol
LIVER DAMAGE	Without evidence of clinical and biochemical liver damage	Without evidence of clinical and biochemical liver damage	Without evidence of clinical and biochemical liver damage	Without evidence of clinical and biochemical of liver damage	Clinical evidence: (Anorexia, weight loss, asthenia, adynamia, hepatojugular reflux) and positive for biochemical changes typical of liver cirrhosis

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Cell death in patients with different alcohol consumption and alcoholic liver disease



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Background and aim: Cell death maintains homeostasis and eliminates damaged cells. The role of this cellular process in alcohol consumption and in pathogenesis of alcoholic liver disease (ALD) has not been fully established. Objective: Characterize the cell death of T-CD4, T-CD8, NK and NKT lymphocytes in peripheral blood of patients with different patterns of alcohol consumption and ALD.

Material and methods: Cross-sectional study. Control subjects with alcohol consumption <10 g/day (CT); risk alcohol consumption (AUDIT > 8) (R); alcohol abuse (A); alcoholism without clinical or biochemical stigmas of liver damage (OH); cirrhosis by alcohol (CiOH) and alcoholic hepatitis (AH). Determination of T-CD4, T-CD8, NK and NKT was performed in peripheral mononuclear fraction. The expression of Fas receptor and ligand (Fas R, Fas L), active caspase 3, early and late apoptosis, necrosis, and cell viability was evaluated by flow cytometry. Statistical analysis: Kruskal-Wallis and U-Mann Whitney, ($p < 0.05$). Protocol approved by the General Hospital of Mexico (HG/DI/16/107/03/082) and UNAM (FMD/DI/15/2019)

Results: 48 participants were included, 14CT, 5R, 5A, 7OH, 6 CiOH y 11AH; the average age was 29 ± 9 , 29 ± 10 , 26 ± 4 , 32 ± 6 , 52 ± 11 and 40 ± 10 ($p < 0.05$), respectively. Alcohol consumption per day was higher in ALD groups (292 ± 150 , 336 ± 180) ($p < 0.05$). Determination of lymphocyte showed that T-CD8 + cells decrease in AH vs CT (12 ± 1.4 vs $19 \pm 2.3\%$) ($p < 0.04$), while the expression of Fas R, Active Caspase increased. Whereas early Apoptosis and Necrosis increases in AH ($p < 0.02$, $p < 0.01$; $p < 0.02$, $p < 0.01$). The percentage of NK and NKT cells as well as the expression of Fas R and active Caspase 3 increased in HA vs CT ($p < 0.03$, $p < 0.04$; $p < 0.01$, $p < 0.02$).

Conclusions: The results show that according to consumption pattern, expressions of the cell death markers were not high in risk consumption, abuse and alcoholism because these events are still subclinical. While in patients with ALD, T-CD8, NK and NKT cells express a higher percentage of death markers, especially in the alcoholic hepatitis due to the elimination of damaged cells.

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