Results: A total of 108 patients with COVID-19 were identified; 68.5% ($n=74$) were men, the mean age was 53 ± 14 years and the body mass index was 28.6 ± 5.8 kg/m^2. The most frequent comorbidity was hypertension with 24% ($n=26$). The presence of comorbidities was associated with risk of ICU admission (OR 3.9 [95% CI 1.6-9.9], $p=0.002$). The most frequent symptoms were cough (72.2%, $n=78$), fever (69.4%, $n=75$) and dyspnea (48.1%, $n=52$). At least one abnormal LFT was present in 94% ($n=103$) of patients at admission, the most frequent was LDH (88.9%, $n=96$), AST and GGT (63%, $n=65$), which are summarized in Table 1. Patients presented abnormal LFTs and respiratory symptoms in 48.1% ($n=52$), while 16.6% ($n=18$) presented abnormal LFTs without respiratory symptoms. Among GI symptoms, 37% ($n=4$) reported at least one, including diarrhea (28.7%, $n=31$), hyporexia (9.3%, $n=10$), nausea (8.3%, $n=9$) or vomiting (4.6%, $n=5$). Of patients admitted to the ICU ($n=39$), 27.5% ($n=10$) presented at least one GI symptom. Mortality was 7.4% ($n=8$). No associations were found between abnormal LFTs, GI symptoms, and outcomes of mortality and ICU admission.

Table 1
Initial liver function tests of patients with COVID-19 ($n=108$).

Parámetro	M [IQR]
Hemoglobin (g/dL)	14.6 [13.7-15.7]
Platelets (cells $\times 10^3$/L)	110.5 [100-136.6]
Albumin (g/dL)	3.2 [2.8 -3.5]
Total bilirrubin (mg/dL)	0.94 [0.67-1.01]
Direct bilirrubin (mg/dL)	0.23 [0.16-0.24]
Alanine aminotransferase (IU/L)	42 [28-52.7]
Aspartate aminotransferase (IU/L)	52.1 [33-55]
Alkaline phosphatase (IU/L)	72.5 [55-75.7]
Gamma-glutamyl transpeptidase (IU/L)	73 [34-77]
Lactate dehydrogenase (IU/L)	303 [222-360]

Conclusions: In patients with COVID 19, the presence of metabolic comorbidities confers a higher risk of ICU admission, in contrast to abnormal LFTs and GI symptoms that were not associated with clinical outcomes.

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

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54

Effect of chronic alcohol intake in a pre-clinical model with cholesterol overload

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Background and aim: Obesity and alcohol consumption are two of the main risk factors in liver diseases, which coexist frequently and are considered to accelerate the progression of liver damage, from simple steatosis to steatohepatitis, cirrhosis and cancer. The Mexican diet is high in cholesterol, in addition to being frequently found elevated in patients and animal models with obesity. Therefore, our goal is to determine the effect of alcohol intake in an environment with cholesterol overload.

Material and methods: Male and female mice of the C57BL / 6J strain 8-10 weeks old were used. The NIAAA model was used, which consists of consuming a Lieber-DeCarli diet added with ethanol (5% v / v final concentration) for 10 days, followed by acute dose intragastric (5 g / kg) of ethanol. Cholesterol overload was induced by adding cholesterol (1.25 w / v) to the liquid diet. Liver damage was assessed using liver function tests. Biochemical tests were carried out to determine the degree of apoptosis and the amount of cholesterol in the different experimental groups.

Results: The alcoholic diet added with cholesterol exacerbates liver damage and causes premature death of males. Also, the enzymatic activity of ALT and AST were increased, both in males and in females groups. Liver caspase 3 activity, indicative of apoptosis, was also found increased with respect to the other groups. At the macroscopic level, a liver with higher steatosis was observed in the group treated with alcohol and cholesterol, data that was corroborated by H&E in histological sections with a 5.15-fold increase in the total cholesterol content in the liver compared to the control group. Females had higher liver cholesterol content than males (18.66 μg cholesterol / mg protein vs. 15.6 μg cholesterol / mg protein), however, the activity of transaminases were similar in both genders.

Conclusions: The data obtained suggests that liver cholesterol overload increases susceptibility to alcohol damage. An increase in cell death was observed in this group, as well as in liver damage tests. Further studies are required to determine the mechanism by which greater damage is caused in the presence of both agents.

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

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55

Acute liver injury and survival in patients with SARS-Cov-2 from the Hospital Central Militar

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Background and aim: Recent studies on SARS-CoV-2 have shown that the incidence of liver injury varies between 14.8% and 53%, mainly demonstrable by abnormal ALT / AST levels accompanied by slightly elevated bilirubin levels. Reports of autopsies around the word of patients that death from COVID-19 shows severe liver damage ranging from 58.06% to 78% of the cases.

There is evidence that the elevation of transaminases (ALT / AST) translates into a more serious clinical profile. Besides, the elevation of AST is related with a high risk of mortality, so it must be monitored during hospitalization. Thus, it is important to know the behavior of liver injury and mortality in our population. Aim: To determine transaminase levels in patients with SARS-Cov-2 and its relationship with mortality.

Methods: All the patients admitted with a positive SARS-Cov-2 PCR test were analyzed, the mean and standard deviation of AST, ALT, and other variables of the liver biochemistry, hemoglobin, leukocyte, fibrinogen, and TP were obtained. A Kapplan Meier curve was made for survival to compare patients with and without transaminases elevation.

Results: We studied a total of 92 patients: 79 (86%) were male, age 56.62 ± 13.70 years, weight 72.5 ± 14.30 kg, height 1.63 ± 0.10

m, BMI 27.09 ± 5.04 kg / m2. Of the 92 patients, 68 (73%) had an elevation of transaminases at admission.

Patient's whit elevation of transaminases (68): 63 (93%) were males, the mean values at admission of AST and ALT were 74.91 ± 5.83 and 72.75 ± 5.74, respectively. The average hospital stay was 6.1 ± 4.1 days in de group with no elevation of transaminases and 7.25 ± 5.3 days for the group with elevation. Other variables of liver biochemistry, hemoglobin, leukocyte, fibrinogen, and TP are presented in Table 1. The data referring to the probability

Table 1
Variables determined in the total of SARS-Cov-2 positive patients.

Variable	Media	DE	Variable	Media	DE	Variable	Media	DE
AST (U/L)	74.91	5.83	PCR	141.18	113.66	Bilirrubina directa	0.25	0.09
ALT (U/L)	72.75	5.74	Deshidrogenasa láctica	447.59	175.81	Bilirrubina indirecta	0.43	0.24
Leucocitos	9324	4.91	Albúmina (g/dl)	3.42	0.631	Fibrinógeno	699.26	153.36
Hemoglobina (g/dl)	14.12	2.14	ALP (U/L)	90.72	35.09	TP (seg)	14.95	2.16
Plaquetas	207250	797006	Bilirrubina total	0.96	1.29			

of requiring ICU income. And probability of requiring mechanical ventilation are presented in Table 2.

Table 2
Risk of admission to the ICU. And the risk of requiring mechanical ventilation in patients with elevated transaminases.

Patients with transaminases elevation	Marginal odds	Conditional odds	Bayes Factor
ICU admission	0.04	0.15	3.81
Mechanic ventilation	0.14	0.28	2.02

The group without and with elevated transaminases were compared to observe if elevation of transaminases could influence mortality, obtaining a non-statistically significant p. ($x2 = 0.087$, $p = 0.782$).

Conclusions: In the studied population, the predominant gender was male, the population with elevated transaminases had a 3.82 risk of entering the ICU and 2.02 times more of requiring mechanical ventilation. The elevation of transaminases does not influence survival. The analysis of the entire database will have to be done, since this is a preliminary study (Fig. 1).

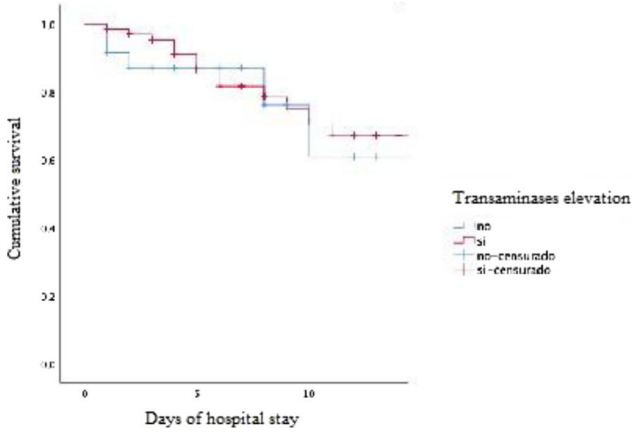

Figure 1. Kaplan Meier curve. The group of patients with and without transaminase elevation is displayed. Elevation of transaminases does not influence in survival.

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56

Evaluation of cyclooxigenase inhibitors in hepatic isquemia-reperfusion injury in Wistar rats

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Background and aim: Ischemia-reperfusion (IR) is one of the main causes of liver graft rejection, therefore the search for new alternatives that reduce this damage continues. Inhibition of the enzyme cyclooxygenase (COX) has been reported to contribute to modulation of IR injury in various organs such as the stomach, brain, lung, heart, and liver. The aim for this work was to determine if the administration of COX inhibitors, acemetacin (ACE) and mefenamic acid (AMF) have a hepatoprotective effect in Wistar rats.

Material and methods: Female Wistar rats were used (200-300 g) and divided into 4 groups ($n = 6$): Sham (laparotomy), IR (20 min of ischemia, 60 min of reperfusion), AMF+IR y ACE+IR (both at a dose of 10 mg / kg for 5 days with subsequent IR). Serum levels of ALT, AST, LDH were determined. Expression of IL-1β, GPx, MPO, SOD-1 and NF-κβ genes was evaluated in total liver tissue RNA using qPCR ($\Delta\Delta Ct$). Cytokines IL-6, IL-1β and TNF-α were evaluated in tissue homogenate using ELISA and oxidative stress markers SOD, GPx and MDA by spectrophotometry. The procedures were performed in accordance with NOM-062-ZOO-1999 and approval of the ethics committee (HI19-00002).

Results: A decrease in ALT and LDH biochemical markers was observed in the AMF + IR group, while in ACE + IR the levels of ALT, AST, LDH were significantly reduced in addition to the relative expression of NF-κβ and GPx, however, the relative expression of IL-1β and the lipid peroxidation marker MDA were significantly increased. No significant difference was observed in the rest of the evaluated markers (Figure).

Conclusions: A hepatoprotective effect of ACE and AMF on IR damage was demonstrated when a decrease in markers of liver damage was observed.

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

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57

Evaluation of atherosclerotic risk in patients with chronic hepatitis C infection

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Background and aim: Chronic hepatitis C virus infection (HCV) is an independent risk factor for atherosclerosis and is associated with the development of cardiac and cerebrovascular events. Among the mechanisms are the production of proinflammatory