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).

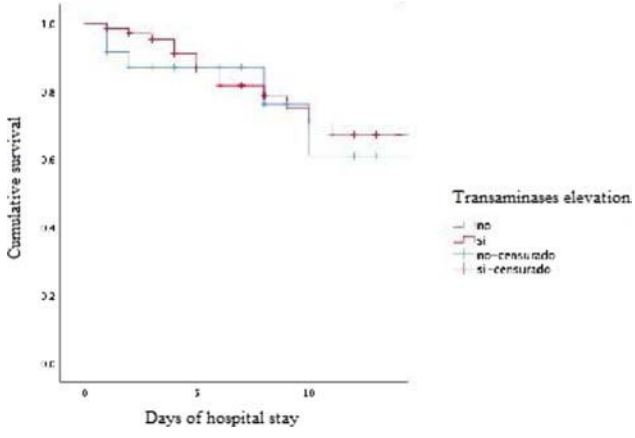

Figure 1. Kaplan Meier curve. The group of patients with and without transaminase elevation is displayed. Elevation of transaminases does not influence in survival.

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

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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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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

cytokines, endothelial dysfunction and increased oxidative stress. The effect of sustained virologic response with direct-acting antiviral agents (DAA) on the progression of atherosclerotic disease has had little study.

Objective: Analyze atherosclerotic risk in patients with chronic hepatitis C infection treated with DAA.

Material and methods: Observational, prospective, analytical, comparative study. Adult subjects with a diagnosis of chronic hepatitis C, without coinfections, cardiovascular diseases, type 2 diabetes, or kidney failure and who signed an informed consent letter. We measured body mass index (BMI), blood cell count, lipid profile, liver function tests, glucose, glycated hemoglobin, uric acid, fibrinogen, C-reactive protein (CRP). Carotid doppler ultrasound to measure carotid media-intima thickness (CIMT).

The two proportions formula was used, descriptive statistics and group comparison with t-Student, dichotomous variables with X^2 and to show differences the Wilcoxon tests. Project with the approval of the institutional ethics committee.

Results: We analyzed 24 participants 19 (79%) women, the mean age 60 ± 11.4, genotype 1b was the most frequent (41.7%), 9 (37.5%) participants had cirrhosis and 4 of them were Child-Pugh B; mean BMI 28 ± 5.07, 6 participants (25%) were obese and 10 (41.7%) had a smoking history.

The analysis of inflammation markers showed a significant decrease in CRP $p < 0.05$. Pretreatment 19 participants (79.2%) had CIMT < 9 mm and 5 (20.8%) had CIMT $\geq$9 mm and posttreatment 22 (91.2%) CIMT <9 mm and 2 (8.3%) CIMT $\geq$9 mm, that is, there were fewer participants with risk CIMT after treatment (Figures 1 and 2).

Conclusions: Treatment with DAA decreases CIMT and improves inflammation markers such as CRP. However, it is necessary to increase the sample size to define whether there is a decrease in cardiovascular risk.

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

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