Declaration of interest: None.

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Table 1
Immunoglobulin and cytokine levels by FTIR

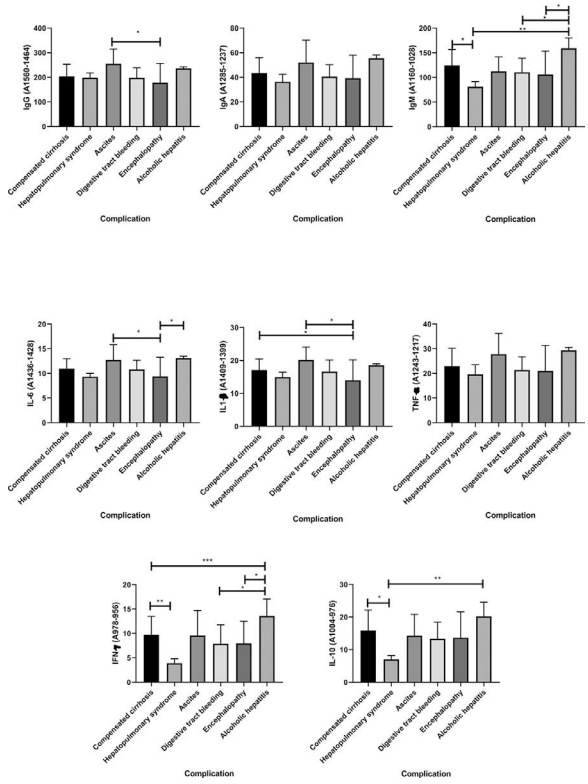
	Compensated	Decompensated	p
IgG (1560–1464 cm^{-1})	203.79 ± 49.42	199.71 ±60.90	0.690
IgA (1285–1237 cm^{-1})	43.44 ± 12.49	42.19 ± 14.98	0.622
IgM (1160–1028 cm^{-1})	124.35 ± 32.39	110.97 ± 39.0	0.044
IL-6 (1436–1428 cm^{-1})	10.94 ± 2.05	10.54 ± 3.06	0.412
IL-1β (1409–1399 cm^{-1})	17.10 ± 3.36	15.99 ± 4.88	0.151
TNF-α (1243–1217 cm^{-1})	23 ± 7.21	22.47 ± 8.06	0.705
IFN-γ (978-956 cm^{-1}),	9.78 ± 3.71	8.23 ± 4.55	0.044
IL-10 (1004–976 cm^{-1})	15.88 ± 6.29	13.61 ± 6.74	0.059

IgA, immunoglobulin A; IgG, immunoglobulin G; IgM, immunoglobulin M; IFN-γ, interferon-gamma; IL-1β, interleukin-1 beta; IL-6, interleukin-6; IL-10, interleukin-10; TNF-α, tumor necrosis factor-alpha.

Table 2
Correlation between neutrophil levels and IgM, IL6, IL1β, IL10 and IFN-γ levels

Neutrophiles	IgG	IgA	IgM	IL6	IL-1β	TNF	IL10	IFN-γ
Pearson correlation	-0.148	-0.160	-.226*	-.199*	-.214*	-0.174	-.273**	-.224*
Sig. (bilateral)	0.111	0.084	0.014	0.032	0.021	0.061	0.003	0.015

Figure 1. Differences between the levels of IgG, IgM, IL-6, IL1β, IFN-γ and IL-10 depending on the cause of decompensation.



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Impact of bilirubin molecular species on mortality in patients with acute on chronic liver failure (ACLF).

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Introduction and Objectives: Acute-on-chronic liver failure (ACLF) represents a serious and potentially life-threatening complication in patients with chronic liver disease. This condition is characterized by a rapid deterioration of liver function in patients with pre-existing chronic liver disease. Among the numerous biomarkers used to assess the severity and prognosis of ACLF, serum bilirubin has emerged as a key indicator of liver dysfunction and clinical deterioration. This study aims to analyze the performance of molecular bilirubin species, such as unconjugated (UCB), monoglucuronide (BMG) and diglucuronide bilirubin (BDG), and their impact on mortality in individuals with ACLF.

Materials and Patients: The study included 45 patients with ACLF of various etiologies. The diagnosis was made using the European Association for the Study of the Liver (EASL-CLIF) consortium definition. Clinical and laboratory data were collected to determine severity and assess mortality during the 90 days following enrollment. Bilirubin was extracted from serum samples to measure UCB, BMG, and BDG by liquid chromatography-mass spectrometry (LC-MS). The quantification of bilirubin was performed by monitoring the mass charge (m/z) ratio.

Results: Of the 45 patients, 40% (n=18) were categorized as ACLF grade 1, 35.5% (n=16) as ACLF grade 2, and 17.7% (n=8) as ACLF grade 3. Regarding the molecular species of bilirubin, it was observed that the values of UCB, BMG, and BDG increased according to the severity of ACLF, specifically those of BMG (p=0.019). Additionally, it was observed that individuals who died had higher levels of BDG (4.49 vs. 1.17), BMG (64.30 vs. 28.57), and UCB (21.92 vs. 16.99) with respect to individuals who remained alive.

Conclusions: In conclusion, our findings reveal an association between BDG, BMG and UCB levels and ACLF severity, suggesting that the suggest that molecular bilirubin species could be useful as prognostic markers in patients with ACLF. However, further studies are required to validate these findings and further explore the role of bilirubin in the prognosis and pathophysiology of ACLF.

Ethical statement: All procedures performed were carried out in accordance with the ethical standards of the Ethics Committee of the Clinica Medica Sur Foundation (protocol code 2021-EXT-552) and with the 1964 Declaration of Helsinki and its subsequent amendments, or other comparable ethical standards.

Declaration of interests: None.

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Clinical manifestations, and oxidative stress imbalance in children with obesity and MASLD

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Introduction and Objectives: Metabolic dysfunction-associated steatotic liver disease (MASLD) is often considered a multifactorial disease that has shown high incidence in recent years in both children and adults. To date, management criteria, diagnosis, and clinical characteristics are not fully defined in childhood.

Objective: Evaluate anthropometric characteristics, biochemical data, clinical manifestations, and Redox balance status in pediatric patients with obesity.

Materials and Patients: A cross-sectional study that included 300 pediatric patients (aged 8 to 17 years) from the obesity clinic of Iztapalapa Pediatric Hospital. Subjects were classified as with MASLD or without MASLD using hepatic ultrasonography. A thorough evaluation of anthropometric characteristics, clinical features, and blood levels of reduced glutathione (GSH) and oxidized glutathione (GSSG) was conducted. Data were reported as absolute and relative frequencies (%), while continuous variables were determined as mean ± SD and analyzed using Student's t-test and Mann-Whitney U test via SPSS V.22 software.

Results: A total of 95 patients met the inclusion criteria, with 78 cases having MASLD and 17 without MASLD: 27% were aged 8-9 years and 73% were adolescents (10-17 years). Being children receiving care for obesity, anthropometric data (weight, BMI (WHO, CDC), waist/height ratio, waist/hip ratio, and % body fat) showed no significant differences between groups. Greater respiratory difficulty ($p=0.037$) and polyuria ($p=0.047$) were observed in patients with MASLD vs. those without MASLD. Additionally, AST, urea, and creatinine levels were elevated in MASLD ($p<0.05$). Finally, GSH was reduced in MASLD vs. non-MASLD ($p=0.001$), thus altering the GSH/GSSG ratio.

Conclusions: Reduced glutathione indicates increased oxidation in children with MASLD, showing a clear association with liver damage even in the early stages of the disease. The incorporation of new tools in the diagnosis and management of obese children is a primary need to reduce the high prevalence and thus improve quality of life and life expectancy.

Ethical statement: The protocol was approved by the Ethics and Research Committees of the "Dr. Eduardo Liceaga" General Hospital of Mexico (CI/314/15) and the Faculty of Medicine of UNAM (DI 115/2015). All participants provided their assent and written informed consent, and the study was conducted in accordance with the provisions of the Declaration of Helsinki.

Declaration of interests: None.

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Hepatic histologic findings in a murine model of diet induced-steatotic liver disease and acute alcohol intake

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Introduction and Objectives: Steatotic liver disease is produced by a range of etiologic agents, among them metabolic and alcoholic. Our aim was to identify the histologic findings produced in the liver after the interaction of steatosis induced by the methione-choline deficient (MCD) diet and the acute ethanol consumption in a murine model.

Materials and Patients: 46 male, 10 week-old, C57BL/6 mice were randomly assigned to the following groups: Control, fed LabDiet 5010; MCD, fed the steatogenic diet MCD for 6 weeks; OHa, fed LabDiet, this group received 8 doses i.p. of ethanol (2.5g/Kg), within a scheme of 2 days of administration followed by 1 day rest; MCDOHa, fed MCD for 6 weeks, this group receive 8 ethanol doses during weeks 5 and 6, as described earlier; a group receiving vehicle with the same scheme as the ethanol was included. After treatments, livers were collected. Paraffin sections were stained with hematoxylin-eosin and Masson's thrichrome. Samples were analyzed. Representative histologic findings were considered when present in at least 50% of the samples per group.

Results: Control and vehicle livers did not show alterations. MCD livers showed macrovesicular steatosis (range 33-66%) in portal and central areas, with few or non ballooning, inflammation was observed, as well as portal fibrosis (F1C). OHa group did not showed steatosis, 57% of samples showed sinusoidal dilation in portal areas; necrosis and inflammation were also observed in the portal triad. Fibrosis was observed in 50% of livers. Interaction of both stimulus (MCDOHa) produced macrovesicular diffused steatosis ranging from 50-90% of liver area. 56% of samples showed few ballooning. Increased inflammatory foci were observed compared with MCD. Regarding fibrosis, 56% showed F0. No signs of necrosis were observed compared with OHa.

Conclusions: Interaction among steatosis induced by MCD diet and OHa increases steatosis, at broader areas of the hepatic parenchyma with increased number of inflammatory foci, but no increase in ballooning, and a lower number of liver showed fibrosis compared to MCD.

Ethical statement: All procedures were approved by Ethics committee in Research from General Hospital of México "Dr. Eduardo Liceaga" (DI/22/UME/04/12)

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