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

Isabel Villagómez-López[1], Laura Mejía-Pérez[2], Moisés Martínez-Castillo[1], Liliana Suárez-Bonilla[2], Daniel Santana-Vargas[1], Zaira Medina-Avila[1], Abigail Hernandez-Barragan[1], Jessica Limon-Castillo[1], Dana Mercado-Herrera[1], Gabriela Gutierrez-Reyes[1]

[1] *Liver, Pancreas, and Motility Laboratory (HIPAM), Experimental Medicine Research Unit, Faculty of Medicine, UNAM, General Hospital of Mexico, Dr. Eduardo Liceaga, Mexico*
[2] *Pediatrics Hospital Iztapalapa, SEDESA. Mexico City, Mexico*

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.

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

Gabriela Sarahi Martínez-Mejia, Miriam G. Bautista-Ubaldo, Gabriela Gutiérrez-Reyes, Carolina Guzmán

Liver, Pancreas, and Motility Laboratory (HIPAM), Experimental Medicine Research Unit, Faculty of Medicine, UNAM, General Hospital of Mexico, Dr. Eduardo Liceaga, Mexico

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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