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Hyperonitinemia-Hyperammonemia-Homocitrullinuria syndrome. Neonatal presentation with acute liver failure.

César U. Amaro-Reynoso, Jose L. Flores-Castillo, Catherine N. Pineda-Cely, Rodrigo Vázquez-Frías

Pediatric Gastroenterology & Nutrition, Children's Hospital of Mexico "Federico Gomez", National Institute of Health, Mexico City, Mexico

Introduction and Objectives: Urea cycle defects occur in 1/35,000 live births and Hyperonitinemia-Hyperammonemia-Homocitrullinuria (HHH) syndrome represents 1-4% of this group of diseases, which represents an autosomal recessive defect due to variants of the SLC25A15 gene. The present work describes the first case of HHH syndrome reported in Mexico.

Materials and Patients: We present a 5-month-old female infant, daughter of the second pregnancy of non-consanguineous parents, originally from Quintana Roo, born at term with intrauterine growth restriction, presented early neonatal sepsis and required ventilatory and hemodynamic treatment, in the second week she presented Cholestasis with normal GGT, coagulopathy, which did not correct after treatment with vitamin K, and irritability with hyperammonemia up to 640umol/L, which led to the diagnosis of neonatal acute liver failure.

At the initial approach, infectious etiology was ruled out, with high suspicion of gestational alloimmune liver disease, due to the presence of elevations of alpha-fetoprotein 12,410ng/mL and ferritin 1,590ng/mL. Gaucher disease, Niemann Pick, and lysosomal acid lipase deficiency were ruled out. Metabolic screen with hyperornithinemia (435.79 mmol/L). The genetic study found a pathogenic variant in a homozygous state of the acceptor site of the splicing of intron 2 of the SLC25A15 gene.

Two doses of human immunoglobulin and supportive treatment for liver failure with menadione and ammonium binders were administered with a favorable therapeutic response; the liver failure was remitted 4 weeks after the established management.

Results: The present work describes the first case of HHH syndrome reported in Mexico, which presented with neonatal acute liver failure associated with two of the three biochemical characteristics described due to hyperammonemia and hyperornithinemia. Likewise, a homozygous variant was identified in SLC25A15 and classified as pathogenic.

Conclusions: This report highlights the first documented case of HHH syndrome in Mexico, emphasizing its association with neonatal acute liver failure, hyperammonemia, and hyperornithinemia. The identification of a pathogenic homozygous variant in the SLC25A15 gene reinforces the importance of genetic studies for early diagnosis and targeted management of urea cycle disorders.

Ethical statement: This work complies with current regulations on bioethical research, obtained authorization from the institution's ethics committee, and does not contain personal information that would allow the patient to be identified.

Declaration of interests: None.

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The GDF11 represses the Stat3 signaling pathway, conferring metabolic, inflammatory, and oncogenic restrictions in cells derived from human hepatocellular carcinoma.

Melissa Sánchez-Rodríguez[1,2], Monserrat Gerardo-Ramírez[3], Arturo Simoni-Nieves[4], María Concepción Gutiérrez-Ruiz[2], Roxana U Miranda-Labra[2], Verónica Souza[2], Leticia Bucio-Ortiz[2], Alejandro Escobedo-Calvario[1,2], Luis E. Gomez-Quiroz[2,3]

[1] *Experimental Biology Postgraduate, DCBS, Universidad Autónoma Metropolitana Iztapalapa, Mexico City, Mexico*
[2] *Experimental and Translational Medicine Area, Health Science Department, Universidad Autónoma Metropolitana-Iztapalapa, Mexico City, Mexico*
[3] *Department of Biochemistry and Molecular Genetics, University of Virginia, Charlottesville, VA, USA*
[4] *The foundation for liver research, London, UK*

Introduction and Objectives: GDF11 has shown potential in displaying anti-tumor effects in cells derived from human HCC, but the molecular mechanisms that lead to this, as well as the early transcriptomic response of GDF11 in HCC, remain a mystery. To identify potential targets for therapeutic intervention revealed by GDF11 treatment in HCC.

Materials and Patients: Huh7 cells were treated for 12 h with 50 ng/ml of GDF11, and sequencing was performed using the Illumina HiSeq4000 platform. The results were filtered with a $p \leq 0.01$, $\pm$ 1.5-fold change, and an FDR= 0.05. Functional and enrichment analysis was done using the Ingenuity Pathway Analysis (IPA) program.

Results: Our data show 1450 differentially expressed genes. It is observed that GDF11 has a profound impact on highly oncogenic pathways, highlighting the Stat3 pathway, beta-catenin, and HIF-1 alpha, among others. Functional analysis revealed that GDF11 could

reduce cholesterol and lipid metabolism in general, inflammation, drug resistance, and stemness capacity. It is noteworthy that tumors grown under a lipid-rich environment exhibit significant activation of Stat3, so the decrease in the molecular signature of Stat3 induced by GDF11 strongly suggests a mechanism mediated by the repression of the pathway of this factor transcription, impacting metabolism, inflammation, differentiation, and drug resistance.

Conclusions: Our study's novel finding is that GDF11 represses the Stat3 signaling pathway, thereby imposing metabolic, inflammatory, and oncogenic restrictions. GDF11 represents a good promise in the treatment of HCC.

Ethical statement: The present work has not involved animals or patients.

Declaration of interests: None.

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Prevalence and Main Characteristics of Portal Venous System Thrombosis in Patients with Advanced Decompensated Chronic Liver Disease

Diego F. Abendaño-Rivera, Paloma M. Diego-Salazar, Saul A. Vera-Nungaray, Karina Cazarin-Chavez, Mónica Baca-García, Yelba G. Céspedes Saballos, Fátima Higuera-De La Tijera

Gastroenterology and Hepatology Department at the General Hospital of Mexico "Dr. Eduardo Liceaga." Mexico City, Mexico

Introduction and Objectives: Chronic liver disease (CLD) is common globally; in advanced stages, decompensation and complications such as portal venous system thrombosis (PVST) increase.

Objective: To describe the prevalence and main characteristics of hospitalized patients with decompensated CLD and PVST in a tertiary care center.

Materials and Patients: An observational, longitudinal, and descriptive study was conducted on hospitalized patients in a tertiary care center in Mexico City with decompensated CLD during the period 2022 and 2023. These patients had imaging studies (CT scan or hepatic Doppler ultrasound) reporting PVST. Follow-up was conducted from the diagnosis of CLD, documentation of PVST, and survival until 2024. Patients with PVST without a diagnosis of CLD or without current follow-up were excluded.

Data will be analyzed using the SPSS statistical software, version 23. Qualitative variables will be presented as frequencies and percentages, while numerical variables will be shown as means and standard deviations or medians and ranges, as appropriate.

Results: We reviewed 788 records of patients with decompensated CLD, of which 60 had PVST, with a period prevalence of 7.6%. Of this group, 20% had hepatocellular carcinoma. Of the total, 37 were women (61.6%), with an average age of 59 ± 9 years. According to the Child-Pugh classification, 6 cases were class A (10%), 25 were class B (42%), and 29 were class C (48%), with an average MELD score of 19.4 and MELD-Na of 21.8. All patients with PVST had at least one prior decompensation before admission (100%), with 43% and 31% having two and three decompensations, respectively. The most frequent etiology was MASLD (48.3%), and the main reason for hospitalization was variceal gastrointestinal bleeding (50%). The portal vein was the most affected vessel (100%), with 20% of cases showing extension to the superior mesenteric vein, of which 76% presented signs of chronicity. The average time from CLD diagnosis to PVST identification was 3.5 ± 2.83 years. By 2024, 41% of patients with PVST had died, with septic shock being the leading cause of death during hospitalization.

Conclusions: The prevalence of PVST in our study is similar to that reported in the literature (3-25%). It is more frequent in women, has a metabolic etiology, and primarily affects the portal vein. To date, 41% have died, mainly from septic shock. However, PVST may be an important factor to study in the progression of CLD.

Ethical statement: This study was conducted in accordance with the ethical principles of our hospital. All data were handled with strict confidentiality and used solely for research purposes.

Declaration of interests: None.

Funding: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Reviewed Records	F	% 788
Portal Vein Thrombosis	60	7.6%
Hepatocellular Carcinoma	12	20%
Affected vessel		
Portal Vein	60	100%
Superior Mesenteric Vein	13	22%
Inferior Mesenteric Vein	0	0%
Splenic Vein	6	10%
Periodicity of Portal Vein Thrombosis		
Recent	17	28.30%
Chronic	43	71.70%
Etiology of Chronic liver disease		
Alcohol	19	31.70%
METLAD	3	5%
MASLD	29	48.30%
Viral	4	6.70%
Autoimmune	5	8.30%
Child Pugh		
A	6	10%
B	25	41.70%
C	29	48.30%
MELD	19.4 ± 6	
MELD NA	21.8 ± 6	
Number of prior decompensations		
1	60	100%
2	18	30%
3	19	31%
Reasons for Admission		
Variceal Hemorrhage	30	50%
Ascites	15	25%
Hepatic Encephalopathy	5	8.30%
Infection	3	5%
Acute Kidney Injury	1	1.70%
Study Protocol	5	8.30%
Cardiogenic Shock	1	1.70%
Average time between CLD diagnosis and PVST		3.5 ± 2.83 años
Death	25	41.7%
Causes of Death		
Septic shock	13	52%
Hypovolemic shock	2	8%
Cardiogenic shock	1	4%
Respiratory failure	1	4%
Disease progression	8	32%

CLD: Chronic Liver Disease; MASLD: Metabolic Associated Steatotic Liver Disease; METLAD: Metabolic Liver Disease; MELD: Model for End-Stage Liver Disease; MELD NA: Model for End-Stage Liver Disease without Ascites; PVST: Portal Vein and Splenic Vein Thrombosis.

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Experience with dexmetomidine in the management of alcohol withdrawal syndrome for patients with cirrhosis

Liliana I. Gallardo-González, Jose L. Perez-Hernandez, Maria F. Higuera-De la, Tijera, Mary C. Alegría-Ovando

Department of Gastroenterology and Hepatology. General Hospital of Mexico "Dr. Eduardo Liceaga." Mexico City, Mexico

Introduction and Objectives: Lorazepam is the first-line treatment in patients with alcohol withdrawal syndrome (AWS). In patients with cirrhosis and AWS, the use of dexmetomidine (DXM)