liver failure, including infectious agents, drugs, and coexisting diseases.

Brief description of the management to be carried out in cases of acute liver failure secondary to viral infections.

Results: This is a 31-year-old man who was hospitalized when presenting in his house with jaundice syndrome, abdominal pain and a drowsy state:

In his important history, he presented consumption of crystal and marijuana in a time of 8 months, high-risk sexual relations without the use of condoms, multiple sexual partners (Man who has Sex with Men), had been evaluated 7 days before as probable autoimmune hepatitis due to present positive anti-smooth muscle antibodies 1:80.

On admission to our unit, he presented 7 days of evolution with hyposthenia, hypodynamia, diffuse abdominal pain, being treated at the time as autoimmune hepatitis, treatment with steroids was given, later he presented an increase in the abdominal girth and was sent to our medical institution, we received the patient in stupor state with Glasgow Coma Scale 10pts, jaundiced tint in sclerae and skin, due to the aforementioned history, a panel was performed for hepatotrophic viruses and sexually transmitted diseases such as HIV and syphilis. Finding positive HBsAg, positive rapid test for HIV and positive VDRL, later in his first 24 hours of admission to our unit, he developed prolongation of coagulation times (PT 55.5, aPTT 82.8), quantification of Total Bilirubin at 21.2mg/dl, with liver enzymes. > 6 times its normal value (AST 353, ALT 195), INR 5.15, and hepatic encephalopathy, for which an acute liver failure approach was initiated, fulfilling the defining criteria to be met: BT elevation >4mg/dl, prolonged treatment times coagulation and hepatic encephalopathy (Table 1).

We report the case of a patient who presented an important history to guide viral infections as the cause of the acute hepatic process; a complete viral panel was requested that included HIV, Hepatitis A, B, C and VDRL Viruses, where the Hepatitis Antigen was positive. Hepatitis B virus surface, the rapid test for HIV, as well as the VDRL. However, in the first hours of admission, defining clinical data of acute liver failure were established by presenting Prolonged coagulation times with INR >1.5, hyperbilirubinemia >5 and type A acute hepatic encephalopathy according to the Vienna classification, for which reason management began with disaccharide laxatives (lactulose), luminal-acting antibiotics (rifaximin), fluid replacement (30ml/kg) and administration of albumin (1g/kg/day), however, according to mortality and survival scores, the patient presented a high mortality (MELD Na 49pts, 90-day mortality of 66%, NACSELD 30-day mortality of 96%), according to Factor R a mixed pattern was obtained, which is associated with hepatotropic virus infection among the main causes, and The coexistence of HBV and HIV was established as the cause of acute liver failure, since it has been established that when there is a coinfection between HBV and HIV, the possibilities of acute liver failure increase to >10%, emphasizing that in cases of liver failure acute due to viral causes, other associated factors should be sought, such as coinfection with other viruses, since the incidence of cases of acute liver failure due to a single viral agent is less than 5%. It is worth mentioning that cases have been reported that establish syphilis infection as the cause of liver failure, so it could even be considered a triple coinfection. After 48 hours of admission, the patient did not present improvement; he progressed with deterioration of renal function and hepatic encephalopathy, requiring advanced management of the airway. This procedure is the one that his relatives did not accept and for this reason, no therapy could be provided. Renal replacement or management in the intensive care unit.

Conclusions: This case is highly relevant since when addressing acute liver failure, causes of viral origin must be intentionally sought. Among the viral causes, the hepatitis B virus is the one that has been most associated with developing acute liver failure. It is established that up to 4% of patients with HBV will develop this entity. In this case, the patient was infected with HIV, estimating an association between both infections of 10% as causes of acute liver failure. These patients who present coinfection should urgently start management with HAART, which presents activity for HBV. However, it is estimated that Coinfected patients who progress to acute liver failure have a poor prognosis and high mortality, leading in most cases to death. Likewise, during the course of the disease, the use of steroids is not recommended for the management of patients with virus infection. hepatotropes, so they should be avoided. In this case, despite having started treatment in the first 24 hours, the patient did not improve and once they present renal failure, renal replacement therapy and management in intensive care should be provided in order to reduce mortality and allow recovery. Liver transplantation can be used as definitive treatment provided that this resource is available and when the criteria for acute liver transplantation are met, the Kings College criteria and the Clichy criteria have been established for this purpose, an 80% success rate is estimated in cases of acute liver failure undergoing transplantation.

Ethical statement

The identity of the patients is protected. Consentment was obtained.

Declaration of interests

None

Funding

None

Table 1

ESTUDIOS DE LABORATORIO	
Prothrombin Time	55.5 seconds
INR	5.15
aTTP	82.8 seconds
Alcaline Phophatase	84 U/L
ALT	195 U/L
AST	353U/L
Glucose	171 mg/dl
Urea	113.4 mg/dl
BUN	53 mg/dl
Creatinine	3.87 mg/dl
Cholesterol	50 mg/dl
Uric Acid	5.1 mg/dl
Triglycerides	58 mg/dl
Albumine	2.0 g/dl
Total Bilirrubin	21.2 mg/dl
Direct Bilirrubin	13.9 mg/dl
Indirect Bilirrubin	7.3 mg/dl
Total Proteíns	4.6 g/dl
Globulins	2.6 g/dl
A/G Relation	0.77
Hepatitis B sAg	Reactive 5629.17 copies
Syphilis	Positive

https://doi.org/10.1016/j.aohep.2024.101464

MexMix supplementation prevented MAFLD development by restoring microbiota-gut-liver axis in a mice model.

Rebeca Rosas-Campos[1], Ana S. Sandoval-Rodríguez[1], Ángel O. Vázquez-Esqueda[1], Ricardo de la Rosa-Bibiano[1], Jonathan S. Rodríguez-Sanabria[1], Rebeca Escutia-Gutiérrez[1], Arturo Santos-García[2], Juan Armendáriz-Borunda[1,2]

[1] *Institute of Molecular Biology in Medicine, Health Sciences Center, University of Guadalajara, Guadalajara, Mexico*
[2] *School of Medicine and Health Sciences, Tecnologico de Monterrey, Guadalajara, Mexico*

Introduction and Objectives: The microbial communities' control is crucial to maintaining homeostasis of gut-liver axis; clinical evidence demonstrates disruptions of microbiota-gut-liver in individuals with Metabolic-associated fatty liver disease (MAFLD). Foods rich in fiber and polyphenols have been associated with an improvement in microbiota diversity, indexand miRNAs expression. The aim of this study was to evaluate the effect of a supplementation with a mixture of Mexican foods (MexMix): Opuntia ficus indica (nopal), Theobroma cacao (cocoa) and Acheta domesticus (crickets) on gut-liver axis in a MAFLD mice model.

Materials and Patients: Thirty C57BL/6J mice were divided into three groups: 1) control: normal diet. 2) HF: high fat diet (60%) and fructose/sucrose water 3) MexMix: HF diet up to week 10, followed by HF diet supplemented with 6.7% nopal, 8.7% cocoa, and 8.7% cricket for 8 weeks.

Results: The MexMix animals showed a significantly decreased in body weight, visceral and epididymal fat, adipocyte size, triglycerides, insulin, leptin, and PAI-1; while adiponectin levels increased. Using 16S rRNA gene sequencing, MexMix increased phylogenetic diversity, Firmicutes abundance, and enrichment of 10 beneficial genera, including *Lachnospiraceae*, *Ruminococcaceae*, *Akkermansia*, and *Eubacterium_coprostanoligenes_group*. In the gut, MexMix supplementation significantly increased SCFAs concentration, intestinal crypts depth, *Ocln* and *Cldn1* expression, and decreased *Il6* and *Tnf-a* expression. In liver, MexMix significantly reduced steatosis and Tnfa expression. Besides, MexMix increased nuclear translocation of NFR2 and, in consequence, a higher hepatic expression of *Cat* and *Sod*. MexMix also decreased hepatic expression of miRNA-34a, miRNA-103, and miRNA-33a.

Conclusions: Synchronous supplementation with three nutraceuticals, nopal, cacao, and cricket, produced better results compared to previous studies where foods were administered individually. MexMix demonstrated its efficacy as a prebiotic, promoting the growth of beneficial genera and improving intestinal health. These findings indicate that MexMix has the potential to serve as a therapeutic approach for treating MAFLD in patients, as well as other conditions associated with excessive consumption of fats and sugars.

Ethical statement

The protocol was registered and approved by the Ethics Committee.

Declaration of interests

None

Funding

None

https://doi.org/10.1016/j.aohep.2024.101465

Effect of methyl donor supplementation on gut microbiota and hepatic expression of key miRNAs in a murine model of MAFLD

Rebeca Rosas-Campos[1], Ángel O. Vázquez-Esqueda[1], Marina Galicia-Moreno[1], Juan Armendáriz-Borunda[1,2], Ana S. Sandoval-Rodríguez[1]

[1] *Institute of Molecular Biology in Medicine, Health Sciences Center, University of Guadalajara, Guadalajara, Mexico*
[2] *School of Medicine and Health Sciences, Tecnologico de Monterrey, Guadalajara, Mexico*

Introduction and Objectives: Metabolism-associated fatty liver disease (MAFLD) is the most common liver disease worldwide, and intestinal dysbiosis is associated with its development. Methyl donor supplementation has shown beneficial effects for MAFLD treatment; however, its role on the intestinal microbiota and miRNAs hepatic expression has been poorly studied. The aim of this study was to evaluate the effect of methyl group donor supplementation on gut microbiota and hepatic expression of key miRNAs in a murine model of MAFLD.

Materials and Patients: Twenty-four male C57BL/6J mice were divided into three groups: 1) Control: Conventional diet. 2) HF/FS: Diet rich in fats and sugars for 18 weeks. 3) HFMS: HF/FS diet for the first 10 weeks, followed by a HF/FS diet plus orogastric supplementation with methyl group donors for the last 8 weeks.

Results: The intestinal microbiota was characterized by 16S rRNA gene sequencing; supplementation with methyl donors modified microbial composition analyzed by beta diversity. In addition, HFMS group strongly tended to increase alpha diversity and induced enrichment of six genus: *Acinetobacter, Anaeroplasma*, *Pseudomonas*, *Stenotrophomonas*, *Tuzzerella*, and *Moraxellaceae* family. HFMS group significantly increased SCFAs fecal concentration and restored intestinal permeability dysfunction by increasing *Ocln* and *Cldn1* expression; consequently, a decrease in liver inflammation was observed due to a decrease in *Tnf-a* expression. On the other hand, HFMS group significantly increased hepatic expression of miR-122 and decreased miR-33a expression.

Conclusions: This study offers valuable insights into the role of methyl donors as microbiota modifiers, highlighting their ability to promote restoration of intestinal health and liver metabolism. These findings contribute to the proposition that methyl donors could be a promising strategy for treating MAFLD and hepatic related conditions.

Ethical statement

The protocol was registered and approved by the Ethics Committee.

Declaration of interests

None

Funding

None

https://doi.org/10.1016/j.aohep.2024.101466

Exploring the metabolic and molecular benefits of methyl donor supplementation in a model of metabolic and fatty liver disease

Angel Vazquez-Esqueda[1], Rebeca Rosas-Campos[1], Rebeca Escutia-Gutiérrez[1], Ricardo de la Rosa-Bibiano[1], Samael Rodríguez-Sanabria[1], Marina Galicia-Moreno[1], Juan Armendáriz-Borunda[1,2], Ana Sandoval-Rodríguez[1]

[1] *Instituto de Biologia Molecular en Medicina y Terapia Génica, Universidad de Guadalajara, Guadalajara, México*
[2] *Tecnológico de Monterrey, EMCS*

Introduction and Objectives: Metabolic fatty liver disease (MAFLD) is currently the most common cause of chronic liver damage worldwide. Differential methylation in genes and histones has been correlated with metabolic alterations present in the disease. Supplementation with methyl group donor molecules could work as a therapeutic strategy to reverse the progression of the disease.

Materials and patients: Male C57BL/6J mice of 20-25g of initial weight were fed with a conventional diet (ND n=8); or a diet high in