Neisser Morales-Victorino[4],
Claudia Vásquez-Veloza[5], Diana K. Tapia-Calderon[1]

[1] *Department of Gastroenterology, Hospital Civil de Guadalajara, Fray Antonio Alcalde*
[2] *Department of Endocrinology, Hospital Civil de Guadalajara, Fray Antonio Alcalde*
[3] *Department of Internal medicine, Hospital Regional Lic. Adolfo López Mateos, ISSSTE*
[4] *Department of Cardiology, Hospital Regional Lic. Adolfo López Mateos, ISSSTE*
[5] *Department of Radiology, Hospital Regional Lic. Adolfo López Mateos, ISSSTE*

Introduction and Objectives: Coronary artery disease (CAD) is a leading cause of morbidity and mortality worldwide. Reliable knowledge of the prevalence of occult CAD, particularly anatomically confirmed CAD is limited and cardiovascular risk (CVR) models only predict the risk of an acute coronary event within a set period. It has been described that a FIB-4 score is associated with a higher CVR. Determine what is the utility of noninvasive markers of liver fibrosis in CAD.

Materials and Patients: A cross-sectional study was conducted in two tertiary centers in central and western Mexico from March 2019 to April 2023. Patients who required percutaneous coronary angiography were studied and demographic data and coronary angiographic were recorded. Noninvasive fibrosis indexes were calculated. Continuous variables were subjected to a distribution analysis and equality of variances to subsequently perform a mean comparison analysis with U-Mann-Whitney test between patients with monovascular, bivascular and trivascular involvement. A correlation analysis was also performed between the invasive markers and the Syntax index.

Results: A total of 168 patients were included with a mean age of 66 ± 12 years with a predominance of male sex with 75.6% (n= 127). Angiographic findings included 37.5%, monovascular, 32.7%, bivascular and 29.8% trivascular involvement. Comparison of means of noninvasive markers of fibrosis demonstrated a significant difference in HFS between patients with monovascular (0.17 ± 0.18), bivascular (0.27 ± 0.18) and trivascular (0.30 ± 0.25) coronary artery disease, p=< 0.001. A correlation was also demonstrated between non-invasive markers and Syntax score: FIB-4 (r=: 820, p=<0.001), APRI (r=: 766, p=<0.001), HFS (r= 869, p=<0.001), (r= 820, p=<0.001), NFS (r= 807 p=<0.001)

Conclusions: The score of noninvasive tools to assess liver fibrosis correlates positively with the complexity of CAD and could be considered as noninvasive tools to be used in the assessment CVR.

Ethical statement

The protocol was registered and approved by the Ethics Committee. The identity of the patients is protected. Consentment was obtained.

Declaration of interests

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Correlation between steatosis and fibrosis in patients with metabolic syndrome

Leonardo S. Juárez-Chavez, Ángel D. Santana-Vargas, María F. Higuera-de la Tijera, Silvia J. Lozada Calle, Ricardo García-Peniche, Christian Calderón-Mendoza, Carlos Barragán-Pasten, José L. Pérez-Hernández

Service of Gastroenterology and Hepatology Hospital General de México "Dr. Eduardo Liceaga"

Introduction and Objectives: MAFLD is a highly prevalent cause of chronic liver disease, present in 70% of overweight people, 70% of diabetics, and 90% of morbidly obese people. It is the hepatic manifestation of the metabolic syndrome, defined by the presence of central obesity, insulin resistance, hyperlipidemia, hyperglycemia, and hypertension. The development of liver fibrosis is secondary to several factors, steatosis being one of them. To evaluate the correlation of steatosis with hepatic fibrosis in patients with metabolic syndrome using transition elastography.

Materials and Patients: Patients older than 18 years who met MALFD criteria were included, transition elastography was performed to calculate CAP and kilopascals, steatosis degree and fibrosis degree were calculated according to the myfibroscan application, for statistical analysis Pearson's bivariate correlations were used between CAP and kilopascal values. The association between the degree of steatosis and fibrosis was performed using the chi-square test. Was considered significant at $p < 0.05$.

Results: 94 patients were included, 20 men (21.3%), 74 women (78.7%), mean age 40.5 ± 10.02, CAP 300.6 ± 63.4, kilopascals 6.4 ± 2.7, steatosis grade S0: 8, S1: 8, S2: 20, S3: 58, degree of fibrosis F0: 58, F1: 14, F2:14, F3: 6, F4:2. The correlation between CAP and kilopascals was moderate and significant RHO=0.343 P =0.001. A significant association was found between the degree of steatosis and that of fibrosis chi-square (12) =25.1, p=0.015. The proportions were 50% (S0:F0), 16% (S1:F3), 50% (S2:F3), 100% (S3:F4).

Conclusions: The correlation between steatosis and fibrosis is moderate, implying that there are other factors that influence the development of fibrosis and its progression, so metabolic control and other factors in patients with MALFD are highly relevant to prevent fibrosis progression.

Ethical statement

The protocol was registered and approved by the Ethics Committee. The identity of the patients is protected. Consentment was obtained.

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Acute liver failure secondary to co-infection of Hepatitis B and HIV

Yajzeel Estevez-López[1], Alan J. Romero-Sanchez[1], José E. López-Ruiz[2]

[1] *Departamento de Medicina Interna, Unidad Médica de Alta especialidad No. 14 "Adolfo Ruiz Cortines", Veracruz, Veracruz*
[2] *Universidad del Valle de Mexico, Campus Veracruz*

Introduction and Objectives: We present the case of a man with hepatitis B and HIV coinfection diagnosed by serological studies which presented acute liver failure; the diagnostic approach, its treatment and outcome are described.

Materials and Patients: Provide information on the association of hepatitis B virus and HIV. When hepatotropic viruses are identified, intentionally find the association with other factors that cause acute

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

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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

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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*