Table 1
Factors associated with quality studies in patients undergoing liver steatosis screening. (n=558)

Variable	IQR <40				IQR <30			
	Univariate OR (IC95%)	p	Multivariate OR (CI95%)	p	Univariate OR (CI95%)	p	Multivariate OR (CI95%)	p
XL probe	0.26 (0.18 – 0.38)	**≤0.001**	0.24 (0.14 – 0.39)	**≤0.001**	0.25 (0.17 – 0.36)	**≤0.001**	0.18 (0.10 – 0.33)	**≤0.001**
Obesity	0.51 (0.35 – 0.75)	**0.001**			0.57 (0.39 – 0.83)	**0.003**		
<54 years	0.62 (0.43 – 0.89)	**0.01**	0.67 (0.46 – 0.99)	**0.04**	0.81 (0.58 – 1.1)	0.26		
IQR kPa <30	0.42 (0.20 – 0.88)	**0.02**			0.35 (0.15 – 0.84)	**0.02**		
IQR kPa <10	0.74 (0.48 – 1.12)	0.178			0.63 (0.43 – 0.93)	**0.02**		
BMI <27kg/m2	0.57 (0.39 – 0.55)	**0.005**			0.61 (0.43 – 0.87)	**0.008**		

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COMPARISON OF SEROLOGICAL MODELS OF LIVER FIBROSIS AGAINST TRANSIENT ELASTOGRAPHY BY FIBROSCAN® IN PATIENTS WITH NON-ALCOHOLIC FATTY LIVER DISEASE

J. Aquino-Matus[1], J. Jiménez-Pavón[2], M. Uribe[1], N. Chavez-Tapia[1]

[1] *Fundación Clínica Médica Sur. México City, México*
[2] *National Institute of Psychiatry, México City, México*

Introduction and Objectives: Liver fibrosis is the most important prognostic factor in nonalcoholic fatty liver disease (NAFLD). The study's objective is to compare the serological models of liver fibrosis (NAFLD-FS, FIB-4, BARD, APRI and AST/ALT) against transient elastography by FibroScan® in patients with NAFLD.

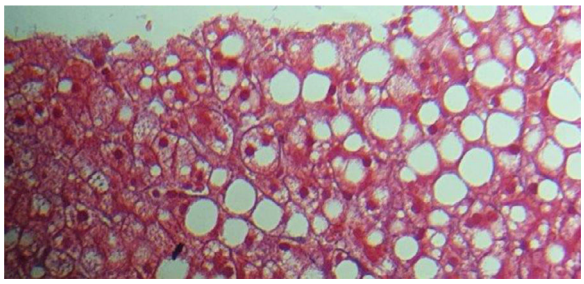
Materials and Methods: Observational, retrolective and cross-sectional study of records of patients diagnosed with liver steatosis by FibroScan® without significant alcohol consumption. A Pearson's correlation and heat maps were used for the correlation between results of FibroScan® and the serological models of liver fibrosis. ROC curves were analyzed to compare the serological models against FibroScan® as the gold standard for clinically significant liver fibrosis.

Results: Data from 976 files were collected, with a prevalence of 63% of liver steatosis by FibroScan® (CAP >232 dB/min) and 1.74% of significant liver fibrosis (LSM >7.0 kPa). In patients with NAFLD, a low positive correlation of NAFLD-FS (r=0.291; p<0.001) and BARD (r=0.021; p<0.001) and a very low positive correlation of APRI (r=0.184; p<0.001) with clinically significant liver fibrosis was reported. No correlation was observed with FIB-4 (r=-0.003; p=0.943) or with the AST/ALT ratio (r=-0.039; p=0.336). The NAFLD-FS reported an area under the curve (AUC) of 0.838 (95%CI 0.76-0.91) and the APRI of 0.797 (95%CI 0.68-0.92) compared to FibroScan® for clinically significant liver fibrosis (Figure 1).

Discussion: Liver biopsy is an invasive method and the gold standard for evaluating liver fibrosis; however, it is not exempt of complications. Transient elastography by FibroScan® is a non-invasive and validated method but with limited availability and accessibility. Serological models are widely available and can be easily used in daily practice. In a previous study, the NAFLD-FS reported an AUC of 0.72 (95% CI 0.60-0.83) compared against liver biopsy, which is comparable to the AUC reported in this study against FibroScan®.

Conclusions: The NAFLD-FS is the serological model for liver fibrosis with the best AUC and correlation with transient elastography in patients with NAFLD and is proposed as an evaluation method in places where FibroScan® or liver biopsy is not available.

The authors declare that there is no conflict of interest.



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NEOBUXBAMIA TETETZO AS A CAUSE OF DRUG-INDUCED LIVER INJURY

A.C. Chavarín Meza, C. Diéguez-Campa, O. León-Mondragón, L. Juarez-Chavez

Servicio de gastroenterología. Hospital General de Zona 1A "Rodolfo Antonio de Mucha Macías", Ciudad de México, México.

Introduction and Objectives: Drug-induced liver injury (DILI) is a rare clinical condition, the incidence is estimated from 14 to 19 cases per 100,000 population per year, it is responsible of 3 to 5% of jaundice hospitalizations, and it is the most frequent cause of acute liver failure in many of the western countries. Neobuxbaumia tetetzo is a species of flowering plant of the Cactaceae family, endemic to Mexico, distributed in Puebla and Oaxaca, and has been used within Mexican cuisine, without studies that establish the safety of its consumption, which predisposes to undocumented adverse effects, including probable liver injury.

Materials and Methods: The patient is a 19-year-old male, high school student and employee of a private company, single, originally from Tehuacán, Puebla, resident of Mexico City. Non-relevant family hereditary background. He denied experiencing any chronic degenerative diseases, allergies or traumas, but reported complications during an appendectomy in May of 2019. Has positive alcoholism, consuming it occasionally in social events; last consumption was seven months prior to the onset of symptoms. He denied the use of drugs, food supplements and herbalism. He began in June 30th of 2020 with asthenia, hyporexia, adinamia, nausea and unquantified fever, pain in epigastrium of moderate intensity, generalized pruritus, conjunctival jaundice and coluria were added, progressed to generalized jaundice, required hospitalization in August 2020. The laboratory results are Total Bilirubin 38.5 mg/dL, Direct bilirubin 26 mg/dL, ALT 60, AST 63, AP 329, GGT 33, General urine test that evidenced bilirubins 6 and urobilinogen 8. An ultrasonography and an abdominal tomography were performed, both reporting vesicular lithiasis, without obstruction or dilation of the bile duct. Subsequently, cholangioresonance was carried out on September 9, 2020, reporting liver gland with homogeneous parenchyma, bile duct without intra and extrahepatic dilation, gallbladder with the presence of lithic of 6.5 mm. Cholecystectomy and liver biopsy were performed, with histopathological result of gallbladder with chronic cholecystitis. Liver biopsy reporting: Hepatic parenchyma with preserved architecture, with few plasmatic and eosinophilic cells, and presence of severe intracanalicular and intracytoplasmic cholestasis corresponding to regenerative changes of grade 0 fibrosis on the Metavir scale and focal microvesicular steatosis, without regeneration nodules. Within its approach, serology Anti-Sm, IgM vs. CMV, IgM vs. Rubella, IgM vs. Toxoplasma, ANAs, anti Ro, SCL70 antibodies, HBV, HCV were negative.

Discussion and Conclusion: The only finding observed in the clinical case of our patient that we can appreciate is the elevation of bilirubin. After ruling out the main and possible causes that affected the health of our patient, it was decided to re-interrogate him and it was found that the patient had an antecedent of consumption of N. tetetzo, this highlights the importance of the clinical history in the approach of DILI and the need for of a clinician to contemplate this possibility. Although in this case, a RUCAM score of 6 points was calculated which makes the diagnosis possible, the score was created in order to avoid biopsy given that it is invasive; and before an anatomopathological finding compatible with DILI it is doubtful whether it is worth rechallenging the patient to the consumption of N. tetetzo to objectify the condition of bilirubins before exposure, there is a risk of acute liver failure, which creates an ethical dilemma and violates the principle of non-maleficence.

The authors declare that there is no conflict of interest.

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HEPATIC MANIFESTATIONS OF INFECTIOUS DISEASES. PRESENTATION OF CLINICAL CASE

S.L. Falcón González, B.M. Morales Matamoros, K.G. Córdova García, F.A. Reynoso Zarzosa

Hospital Universitario de Puebla, Puebla, México

Introduction and Objectives: There are infectious diseases that produce alterations in liver function tests and histology, through the activation, by endotoxins and other agents of the innate immunity pathway. This work aims to review the hepatic manifestations of diseases such as dengue and brucellosis in the pediatric age by means of clinical cases.

Results: Case 1: 17-year-old male with a history of travel to an endemic area and family members positive for dengue. Fever, myalgias, arthralgias, arthralgias and headache. On admission with leukocytes 6.93 thousand, platelets 189 thousand and positive IgG and IgM antibodies for dengue fever. Physical examination with hepatodynia and petechiae in the pelvic limbs. In control biochemistry with leukopenia and moderate thrombocytopenia, as well as alterations in liver function (Table 1). Ultrasound of the liver with data of acute inflammatory process. Seven days after admission, with clinical and biochemical improvement, it was decided the discharge of the patient. Caso 2: male, four years nine months old, with symptoms of 1 month of evolution characterized by fever, hyporexia and general malaise. Physical examination with icteric-pale color, hepatomegaly of $3 \times 5 \times 7$ cm and splenomegaly of 4 cm. Paraclinical tests showed anemia, leukopenia and mild-moderate thrombocytopenia, hypertransaminasemia, cholestasis and TTP prolongation (Table 2) with positive rose bengal, hepatic USG with hepatomegaly and echogenicity changes, for which he was sent to the third level of care.

Discussion: The mechanisms of liver injury can be divided into four pathways: vascular, toxic, immune and hormonal. In infections, the immune pathway is the cause of liver damage, being activated by endotoxins, leading to inflammatory infiltration, the release of cytokines, reactive oxygen species and necrosis. In dengue infection, liver injury manifests with hypertransaminasemia, with a peak between the seventh and ninth day, progressive decrease and normalization between 2 to 8 weeks. Hepatic involvement in brucellosis can occur in the acute or chronic phase of the disease. In the acute phase, establishing a non-specific granulomatous hepatitis, increasing in 63% of the cases the liver function tests.

Conclusions: The functions of the liver and the relationships it establishes with other organs may favor its injury during some infectious pathologies, being at this level one of the first manifestations in the context of an infectious disease, ruling out, in both cases, liver pathology by hepatotropic virus, metabolic, autoimmune and anatomical cause.

The authors declare that there is no conflict of interest.

Table 1
Case 1: Liver function test during the hospital stay

Días de estancia	2	4	7	8	Semana 6
ALT	190	669	1106	857	227
AST	244	845	920	382	36
GGT			187	182	106
ALP	70	77		91	123
LDH		967	890	487	293
TBili	0.5	0.6	0.6	0.6	0.4
IBili	0.3	0.3	0.4	0.4	0.3
DBili	0.2	0.3	0.2	0.2	0.1
PT	12.2		10.9		
PTT	30.4		24.1		

Table 2
Case 1: Liver function test during follow up

	1	2
ALT	64	68
AST	109	137
GGT		441
ALP	457	670
DHL		967
TBili	1.30	0.88
IBili	1.22	0.25
DBili	0.08	0.63
PT	13.7	13.9
PTT	56.2	40.4

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HEPATIC STEATOSIS IN CYSTIC FIBROSIS. APROPOS OF A CASE

S.L. Falcón González, M. Lescas Orozco, Y.S. Sosa Pech, F.A. Reynoso Zarzosa

Hospital Universitario de Puebla

Introduction and Objectives: Cystic Fibrosis (CF) is a genetic disease characterized by dysfunction of the exocrine glands. Hepatic involvement is the leading non-pulmonary cause of death in CF patients. This paper aims to present the case of a schoolchild with hepatic alterations as the initial clinical manifestation of CF.

Results: 10-year-old male with a history of sibling death due to liver failure at three years of age, multiple respiratory symptoms in the first two years of life and hospitalization at 3 years of age due to bronchitis. The condition began one year before the assessment with growth arrest and in the last six months with two diarrheal episodes without mucus or blood. On physical examination, weight and height below p5 for age, icteric color, without hepatosplenomegaly, limbs with acropaquia (Image 1). Average bone age of 5-6 years, paraclinical tests with hypertransaminasemia, hypetriglyceridemia and decreased HDL (table 1), normal blood biometry, liver elastography with ARFI 2.1 m/s and liver biopsy with macro and microvesicular steatosis with moderate portoportal fibrosis (image 1-2). Negative approach for infectious hepatitis, Wilson's disease. 1 alpha-antitrypsin deficiency and lysosomal acid lipase deficiency. Diagnosis of CF with sweat electrolytes of 105 mmol/L was made. Currently under follow-up by gastroenterology and pediatric pulmonology.

Discussion: CF is the most common autosomal recessive disease in the Caucasian population, with multi-organ involvement. Liver disease has a high incidence in the first 10 years of life with 2.5/100 patient-years. However, no CFTR gene mutation has been directly associated