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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.

https://doi.org/10.1016/j.aohep.2021.100621

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 1Case 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 2Case 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 |

https://doi.org/10.1016/j.aohep.2021.100622

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 followup 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

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with the presence or severity of the liver disease. Clinical presentation varies from mild asymptomatic form to cirrhosis with the need for liver transplantation in these patients. The most common initial suspicion is hepatomegaly and transaminases alteration and laboratory studies and histology alteration. Hepatic fibroelastography represents an emerging method of study for diagnosis, as it represents one of the forms of confirmation of the criteria for liver disease associated with cystic fibrosis. Liver biopsy provides information on the predominant type of lesion (steatosis or focal cirrhosis) and the extent of portal fibrosis. However, it should be taken with caution because of the risk of underestimating the severity of the lesions.

Conclusions: Although the liver disease in cystic fibrosis does not represent the initial manifestation, the evaluation and monitoring in these patients are important for prognosis and survival since it can progress to cirrhosis and liver failure.

The authors declare that there is no conflict of interest.

Table 1Liver function tests during evolution

| | 11.10.2018 | 19/01/21 | 05/06/21 |
|---------------|----------------|----------------|----------|
| TBil | 0.33 | 0.38 | |
| DBili / IBili | 0.08 / 0.25 | 0.14 / 0.24 | |
| AST /ALT | 81 / 69 | 99 / 95 | |
| GGT | 99 | 70 | |
| ALP | 381 | 388 | |
| LDH | 586 | 309 | |
| Protein/Alb | 7,4 / 4.4 | 6.9 / 4.30 | |
| PT/ INR/PTT | 15 / 1.16 / 31 | 15 / 1.16 / 31 | |
| TC | | 127 | 119 |
| TG | | 81 | 174 |
| HDL-C | | 43.1 | 32.2 |
| LDL-C | | 78 | 72 |

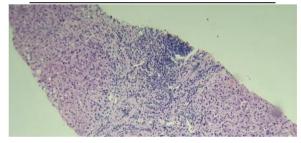


Image 1. Hepatic biopsy with steatosis

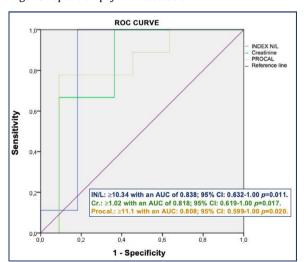


Image 2. Hepatic biopsy with moderate lymphoplasmacytic infiltrate

https://doi.org/10.1016/j.aohep.2021.100623

NEUTROPHILE/LYMPHOCYTE INDEX (IN/L), CREATININE (Cr), AND PROCALCITONIN (PROCAL) AS PREDICTORS OF AMEBIAN LIVER ABSCESS.

C.A. Campoverde-Espinoza, A. Martínez-Tovar, F. Higuera de la Tijera

Hospital General de México "Dr. Eduardo Liceaga". Ciudad de México, México

Introduction and Objectives: A liver abscess (HA) is the accumulation of purulent material in the liver parenchyma that can be bacterial, parasitic, fungal, or mixed. The incidence ranges from 2.3 to 22 per 100,000 people. In Mexico, the annual incidence of amoebic HA is 6.7 per 100,000 inhabitants.

AIM: Determine the cut-off points for the neutrophil/lymphocyte index (IN/L), creatinine (Cr), and procalcitonin (Procal) to predict the etiology of liver abscess.

Materials and Methods: Research design: cross-sectional.

Procedure: We analyzed medical records of patients admitted during 2019 with HA diagnosis and amoeba PCR. The qualitative variables were expressed in frequencies and percentages. The numerical variables in means and standard deviation. We use X2, Fisher's exact, Student's t, and Mann-Whitney U to compare groups as appropriate. ROC curve was used to determine sensitivity (S), specificity (E), positive predictive value (PPV), negative predictive value (NPV), and likelihood value (+ LR). The p-value <0.05 was considered statistically significant.

Results: Out of a total of 32 patients diagnosed with HA during 2019, 20 patients treated with drainage and a PCR test for amoeba from the abscess fluid were included. Of these, 85%(17) were men, with a mean age of 45.33 ± 10.93 years. 45%(9) were of amoebic etiology. In the latter group, the etiology can be predicted with the neutrophil/lymphocyte index with a cohort point of ≥ 10.34 with an AUC of 0.838, S: 100%, E: 81%, PPV: 81%, NPV: 100%. (9/11 vs 0/0 [81.8% vs 0.0%] +LR: 5.49; 95%Cl:1.50-14 p=0.000). The creatinine value of ≥ 1.02 with an AUC of 0.818, S: 66.7%, E: 90.9%, PPV: 85.7%, NPV: 76.9%, (6/7 vs 3/13 [85.7% vs 23.1%] +LR: 7.33;95% Cl:1.07-50 p=0.017) and with a procalcitonin cohort point of ≥ 11.1 with an AUC: 0.808, S: 77.8%, E: 90.9%, PPV: 85.7%, NPV: 87.5%, (7/8 vs 2/12 [87.5% vs 16.7%] +LR: 8.56;95% Cl:1.28-57 p=0.005), with these cut-off points a significant difference was evidenced between the amoebic vs bacterial etiology, for IN/L: p=0.000, for Cr: p=0.017 and for procalcitonin: p=0.005, which are shown in figure 1.

Discussion: Amebic HA is etiologically more frequent in the West and generally in countries with poor infrastructure and development. It reports high mortality with conservative treatment and multiple abscesses, so it is crucial to identify their etiology. In the present study, we propose the cut-off points of biochemical markers for the diagnosis of amoebic HA through IN/L, Cr, and procal that are accessible in units where there is no amoeba CRP.

Conclusions: We were able to determine an adequate AUC and good sensitivity, specificity, positive and negative predictive value; therefore, we could use these biochemical markers to predict the etiology of liver abscesses.

The authors declare that there is no conflict of interest.

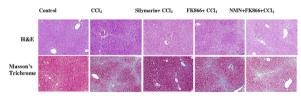


Figure 1. ROC curve graph: indicates the sensitivity and specificity of the cut-off point of the neutrophil/lymphocyte index, creatinine, and procalcitonin to predict abscess diagnosis amebic liver.

https://doi.org/10.1016/j.aohep.2021.100624