Hepatocellular carcinoma with metastasis to the inferior cava vein and right atrium, an unusual cause of Budd Chiari Syndrome

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Introduction and Objectives: Hepatocellular carcinoma (HCC) represents 80-85% of primary liver malignancies, ranks fifth in annual incidence of cancer, with an annual risk in patients with cirrhosis due to hepatitis B virus (HBV) of 3-8%. It has a tendency to involve vascular structures in the liver, such as the portal vein (VP) and hepatic veins (VH). Although HCC involvement of VH is seen less frequently compared to PV, tumor thrombi (TT) have been found to extend into the inferior vena cava (IVC) and right atrium (RA) through the HV. In patients with cardiac metastasis, secondary Budd-Chiari, pulmonary infarction and/or pulmonary metastasis have been documented mainly. A certain number of patients with TT-VH may develop Budd Chiari syndrome <3%, associated with chronic HBV infection.

Materials and Patients: A 58-year-old male patient with a history of human immunodeficiency virus and HBV coinfection, E antigen negative, with virological response. He presented with intense abdominal pain in the left hypochondrium that radiated in a generalized way to the inguinal region bilaterally. During the evaluation, ultrasonographic data of cirrhosis, ascites, Budd-Chiari syndrome, extensive portal thrombosis and hepatocellular carcinoma are found.

Results: Angiotomography showing areas of ischemia and necrosis in the left hepatic lobe, with the presence of a heterogeneous lesion measuring 5.2 cm that involves segments II, III, IVA, and IVB with enhancement in the arterial phase, portal vein thrombosis, and middle suprahepatic veins. Left, as well as a cardiac tumor dependent on the right atrium with extension to the ipsilateral ventricle and inferior vena cava. A transthoracic echocardiogram shows the involvement of the right heart cavities. Laboratory findings: Valued for oncology services without being a candidate for any therapy.

Conclusions: We present a case with symptoms compatible with acute Budd Chiari syndrome with extension to the right atrium and right ventricle secondary to hepatocellular carcinoma. Due to the extension of the tumor, it was not a candidate for surgical therapy, thrombolysis or systemic therapy, presenting with a torpid evolution. Metastasis to the inferior vena cava and right atrium secondary to HCC are uncommon. Imaging studies play an important role in determining the type of lesion and its extension. There are few investigations on treatment since high mortality rates are reported with the performance of lumpectomy combined with thrombectomy. Gaining relevance in the search for therapeutics mainly in tertiary level hospitals and in the scrutiny to rule out HCC and HBV infection in a timely manner.

Ethical statement

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

Declaration of interests

None

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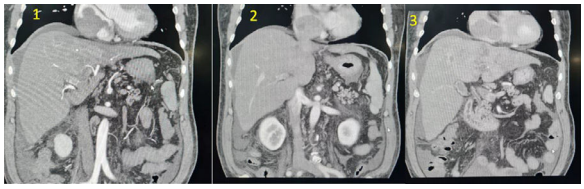

Figure 1: Triphasic computed tomography. 1) Arterial phase: arterial enhancement stronger than the surrounding liver (wash-in). 2) Venous phase: hypodensity or hyposignal intensity compared to the surrounding liver (wash-out) in the venous phase.

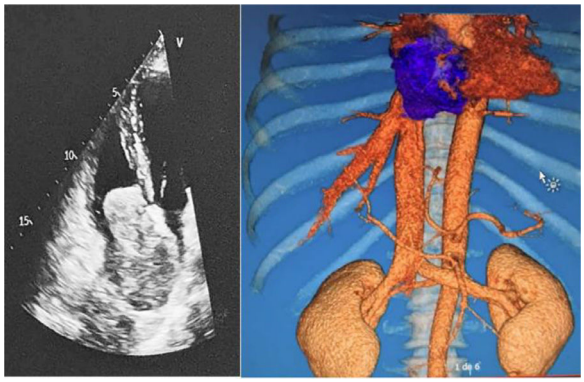

Figure 2: Comparison between echocardiography and tomographic reconstruction

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Pirfenidone decreases insulin, glucagon, leptin, plasminogen activator inhibitor 1, preventing nonalcoholic steatohepatitis and myocarditis in an obesity moldel

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Introduction and Objectives: Obesity is an epidemic in the world, linked with insulin resistance, nonalcoholic steatohepatitis (NASH), and cardiovascular diseases (CVDs), being the latter main cause of global death. NASH progresses with inflammation with or without hepatic fibrosis, including a hormonal dysregulation. Pirfenidone (PFD) have anti-inflammatory and anti-fibrotic effects. However, its effects on hormonal regulation are completely unknown. The aim of this study was to investigate the effects of PFD on hormonal expression levels, related to the lipids and carbohydrates metabolism in high-fat/high-carbohydrate (HFHC)-diet-induced obese male C57BL/6J mice.

Materials and Patients: Twenty-week-old mice were fed with normal diet (ND, 3.1 kcal/g, n=7) and HFHC (65.1 kcal/g, water with 2.31% fructose and 1.89% sucrose, n=14) diet for 16 weeks; at 8 weeks, seven mice with HFHC were administered PFD (300 mg/kg/day) by gavage. ITT, ELISA, dry-chemistry, ELISA, histologies and SPSS were analyzed.

Results: HFHC mice development NASH and myocarditis with fibrosis in both tissues ($P \leq 0.05$). HFHC showed elevated resistin and

aspartate aminotransferase (P≤0.05). Parameters significantly increased in HFHC (P≤0.05), were ameliorated by PFD, such as weight (body, liver, and heart), tibia length, epididymal fat, hepatic steatosis, hormones (insulin, glucagon, leptin, plasminogen activator inhibitor 1), lipid profile (total cholesterol, triglycerides, LDL, and VLDL), as well as inflammatory foci and fibrosis in hepatic and cardiac tissue (P≤0.05). Moreover, PFD reduced alanine aminotransferase (P≤0.05).

Conclusions: In the current work, we showed that PFD increases hormone expression levels, which are implicated in the lipids and carbohydrates metabolism, and also improves expression levels of lipid profile and lipoproteins related with NASH and CVDs. These findings contribute and support the potential therapeutic of PFD for the prevention of NASH and cardiovascular disease development induced by obesity.

Ethical statement

All experiments in the mice were done and results were reported in accordance with the ARRIVE guidelines. The protocol was approved by the CUCS Research

Committee at the University of Guadalajara (protocol number: CI-01419).

Declaration of interests

None

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Metabolic dysfunction-associated fatty liver disease in overweight and obese pediatric population: clinical and biochemical characterization

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Introduction and Objectives: Metabolic dysfunction-associated fatty liver disease (MAFLD) is currently the most common chronic liver disease in children and adolescents. In Mexico, there are no studies that demonstrate its incidence and prevalence, nor its clinical and biochemical characteristics; we don't have a clinical practice guideline, nor guidelines for screening, treatment, and follow-up. Our objective is to identify the clinical and biochemical features of MAFLD in overweight and obese pediatric patients.

Materials and methods: Observational, descriptive, ambispective and cross-sectional study. Patients were recruited from the Pediatric Gastroenterology outpatient clinic of the University Hospital of Puebla for a period from 2019 to 2022, selecting those with overweight and obesity, who were confirmed with a diagnosis of MAFLD through biochemical and imaging tests.

Results: 38 patients met criteria; 63.2% (n=24, ±4.03) correspond to the male sex, compared to 36.8% (n=14, ±3.56) of the female sex. It was more frequent in adolescents (78.9%) and with a higher proportion of patients with obesity (76.3%); no school patient was diagnosed as overweight; all patients in this age group presented obesity at the time of diagnosis.

The total number of patients who presented ALT elevation in diagnostic criteria was 52.6%. Regarding metabolic alterations, the following were found more frequently: Hypoalphalipoproteinemia (50%), Hypertriglyceridemia (42.1%) and elevation of HOMA-IR (91.9%). When evaluating Vitamin D levels, all were altered in insufficiency (42.9%) and deficiency (57.1%). NonHDLC/HDLC index levels have a statistically significant correlation (p0.039) with ALT levels.

Conclusions: MAFLD development is more frequent in male adolescents who are overweight and obese. Using ALT levels as criteria for hepatic steatosis by biochemical marker in the absence of an imaging study may facilitate diagnosis in the MAFLD algorithm. The indices associated with lipid levels (TG/HDLC and nonHDLC/HDLC) may indicate an increased risk for developing MAFLD.

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

None

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Table 1
Anthropometric and biochemical data for overweight and obesity

Variable	Overweight (n=9) Media y DS	Obesity (n=29) Media y DS	p
Age	14 (1.73)	12.5 (3.68)	**0.013**
Stature (mts)	1.63 (0.08)	1.55 (0.16)	0.163
Weight (kg)	67.93 (10.11)	68.37 (20.4)	0.931
CC (cm)	93.35 (9.57)	90.99 (11.08)	0.609
IMC (kg/m2)	25.16 (2.22)	27.47 (4)	0.110
ALT (U/L)	50.66 (26.5)	72 (82.4)	0.452
AST (U/L)	42.11 (21.8)	43.84 (35.39)	0.891
CT (mg/dl)	167.88 (46.5)	147.5 (40.3)	0.263
LDL (mg/dl)	90.62 (37)	73.97 (25.54)	0.144
HDL (mg/dl)	42.28 (16.82)	42.99 (18.67)	0.921
TG (mg/dl)	175.88 (105.33)	140.15 (77.42)	0.285
Insulin (uU/ml)	21.02 (6.44)	25.73 (13.4)	0.166
Glucose (mg/dl)	90.22 (4.99)	89.46 (6.63)	0.703

Statistically significant p values were underlined. mts: meters, kg: kilograms, cm: centimeters, mg/dl: milligrams/deciliters, uU/ml: microunits per milliliter.

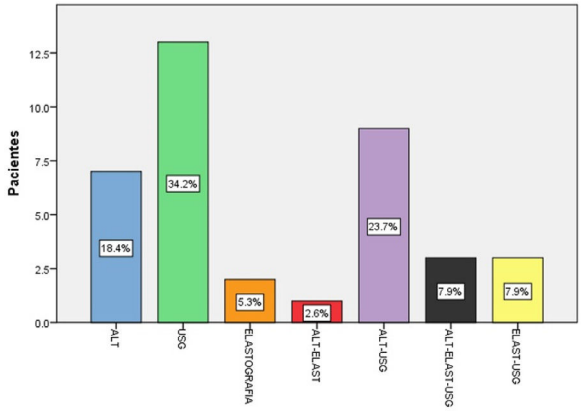

Figure 1. Type of diagnosis

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