developing the severe variant of HAV disease. We present this case of acute liver failure due to HAV in a patient with MAFLD.

Materials and Patients: 37-year-old male with a history of systemic arterial hypertension and morbid obesity. He presented headache, fever, asthenia, adynamia, choluria and acholia with a positive viral profile for hepatitis A virus (IgM +, IgG +). Two days later, with an attack on general condition, in addition to neurological deficit with gradual deterioration of alertness. Simple computed axial tomography of the skull without alterations. Hepatosplenic Doppler ultrasound: Chronic diffuse liver disease, Doppler criteria for grade I venous restrictive liver disease, splenomegaly. He presented multiple organ failure due to coagulopathy, acute liver failure and kidney injury and was sent to a third-level unit for Molecular Adsorbent Recirculating System (MARS) therapy.

Results: It was classified as grade 3B acute-on-chronic liver failure without being a candidate for transplant. During his hospitalization, MARS therapy was performed on two occasions: single-session hemodialysis, hypertonic solution for cerebral edema, and treatment for hyperammonemia. He was started on carvedilol, vitamin E and lipophilic statin. Without organ failure, creatinine levels normalized, mild transaminasemia persisted and as well as hyperbilirubinemia at the expense of direct bilirubin. Continuing follow-up by external consultation.

Conclusions: The complex interaction between hepatic steatosis, hepatitis A infection and acute-on-chronic liver failure is highlighted, noting the importance of comprehensive evaluation and multidisciplinary management. The increasing prevalence of hepatic steatosis poses additional challenges in the management of hepatitis A, increasing the risk of severe forms of the disease. Timely and specialized treatment are essential to address this complex clinical condition.

Ethical statement: Patient identity is protected. Informed consent was obtained.

Declaration of interests: None

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Aggressive intrahepatic cholangiocarcinoma in pregnancy: Case report and literature review

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Introduction and Objectives: Cholangiocarcinoma is a very rare type of hepatobiliary cancer and extremely rare reported during pregnancy. Its early and timely diagnosis is complicated. To report a rare and poorly studied case of aggressive intrahepatic cholangiocarcinoma during pregnancy in a 30-year-old patient.

Material and Patients: Female patient, 30 years old, with antecedent of 2 cesarean sections, one 2 years ago and the second one 1 and a half year ago, without complications and occupational exposure to unspecified pesticides. The clinical picture begins at 32 weeks of gestation characterized by nausea and vomiting of gastric contents, dull pain in the right hypochondrium and weight loss of 7 kg in 2 months, to which generalized jaundice, choluria, acholia, pruritus, nocturnal diaphoresis and ecchymosis; A simple magnetic resonance image was performed and a large liver lesion was identified at the level of liver segments IV and VIII with a maximum diameter of 10.3 cm, suggestive of malignancy associated with the presence of satellite lesions suggestive of infiltration to the rest of the liver parenchyma. It was decided to resolve the pregnancy at 35 weeks of gestation by cesarean section without apparent complications. During the mid-surgical postpartum period simple and contrasted tomography of the abdomen is performed where hepatic, pulmonary, pleural and bone tumor activity and dilation of the intrahepatic bile duct are reported; tumor markers ACE 1.91, CA 19-9 30.89, AFP 149.4; liver biopsy reports metastasis of moderately differentiated adenocarcinoma (g2) consistent with primary bile duct (cholangiocarcinoma); Immunohistochemistry with positivity for ck7, ck19, negative for ck20, gata 3, cdx2, pax8 and hepar1.

Results: During his in-hospital stay, she presented sinus tachycardia evidenced by ECG, associated with risk factors, and pulmonary thromboembolism was suspected. The ICU service was consulted and they accepted the case, evaluated by cardiology performing an echocardiogram discarding the diagnosis. The general surgery, oncological surgery and oncology services were consulted and commented that she was not a candidate for surgical or systemic treatment for advanced disease clinical stage IV.

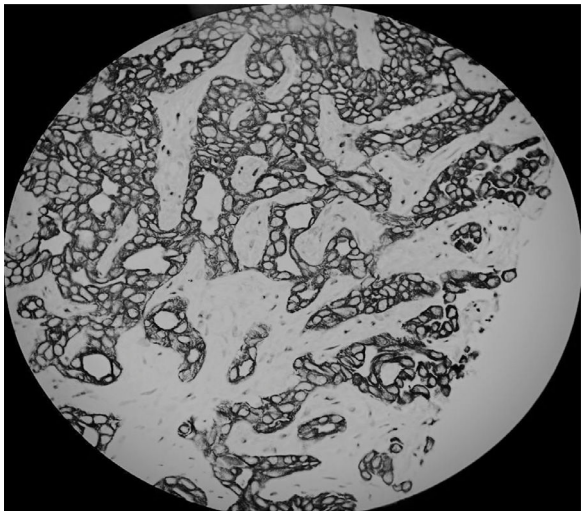
She was discharged from the hospital with palliative measures and two weeks later she was re-admitted to the emergency department due to generalized tonic-clonic seizures advanced airway manage was performed and vasopressor support was decided; simple skull tomography without metastatic activity; presented clinical deterioration and progression of the disease leading to multiple organ failure. The patient died 4 days later. The baby is being monitored by ophthalmology for a diagnosis of retinopathy of prematurity.

Conclusions: Cholangiocarcinoma is the second most common liver neoplasm, it encompasses neoplasms that depend on the bile duct. It has an incidence in pregnancy of 10 cases/10,000 pregnancies, making it a very uncommon pathology and only 12 cases reported from 1998 to 2023 are known. Its prognosis is lethal due to its aggressiveness and diagnosis in advanced stages. The treatment is only surgical, however the procedure carries high rates of morbidity and mortality.

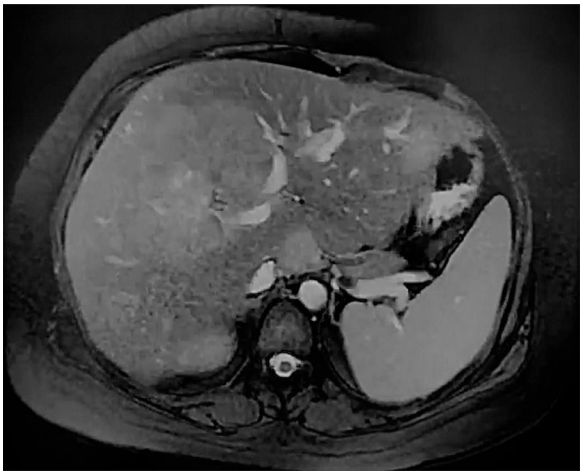
Ethical statement: The patient's identity was protected. Consentment was obtained directly from the patient.

Declaration of interests: None.

Funding: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.



Annex 1. Liver biopsy with immunohistochemistry



Annex 2. Simple magnetic resonance imaging

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Pirfenidone Prevents Myocarditis by Restoring Metabolic Hormone Levels in a Mouse MASH Model and its Effect on H9c2 Myoblast Viability under Glucolipotoxicity

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Introduction and Objectives: Obesity, global epidemic, can cause metabolic dysfunction-associated steatohepatitis (MASH) and cardiovascular diseases. Pirfenidone (PFD) has anti-inflammatory and antifibrotic properties. We investigated the effects of PFD on metabolic hormones expression and myocarditis in a mouse MASH model and its effect on H9c2 cells viability under glucolipotoxicity.

Materials and Patients: Twenty-week-old male C57BL/6J mice were divided into two groups: one group was fed a normal diet (ND, 3.1 kcal/g plus normal water, n=7), while the other group was fed a high-fat, high-carbohydrate diet (HFHC, 5.1 kcal/g plus water containing 2.31% fructose, 1.89% sucrose; n=14) for 16 weeks. At week 8, seven HFHC mice were administered PFD at a dosage of 300 mg/kg/day by gavage. Insulin tolerance tests (ITT), dry chemistry analysis, ELISA, histological staining (Hematoxylin-Eosin and Masson's Trichrome), and morphometric analyzes of the tissues were evaluated. H9c2 cells were treated with the following concentrations: 100 μM, 200 μM, 400 μM PA (PA), 15 mM, 30 mM glucose, and 0.3 mM, 0.5 mM, 1 mM 1.5 mM PFD. H9c2 cells viability under glucolipotoxicity were evaluated by MTT assay and Oil red O staining. The data were analyzed using one-way ANOVA followed by Tukey's post-hoc test in Graphpad Prism v10.0.

Results: HFHC mice developed MASH, myocarditis and fibrosis ($P \leq 0.05$). Additionally, resistin and AST levels significantly increased ($P \leq 0.05$). PFD prevented elevated parameters in HFHC mice ($P \leq 0.05$), such as body weight, epididymal fat weight, liver weight and heart weight; including body weight/tibia length ratio, heart weight/tibia length ratio and epididymal fat weight/tibia length ratio; hormone levels: insulin, glucagon, leptin, and plasminogen activator inhibitor-1 (PAI-1); lipid profile: total cholesterol, triglycerides, LDL, and VLDL; adipocyte hypertrophy, inflammatory foci, and fibrosis in liver and cardiac tissues. Additionally, PFD reduced ALT expression and tibia length ($P \leq 0.05$). The heart weight/body weight ratio decreased in HFHC mice ($P \leq 0.05$), PFD recovered this ratio ($P \leq 0.05$). H9c2 cells treated with 400 μM PA showed 50% cell viability ($P \leq 0.05$), all other concentrations of the compounds had cell viability > 60% ($P \leq 0.05$), including H9c2 cells treated with 150 μM PA, 15 mM glucosa, and 1 mM PFD ($P \leq 0.05$). H9c2 cells treated with 150 μM and 200 μM PA showed a significant increase in intracellular lipid accumulation ($P \leq 0.001$), and H9c2 cells treated with 150 μM PA and 1.5 mM PDF showed a tendency to reduce intracellular lipid levels.

Conclusions: PFD restores the expression levels of metabolic hormones, which are involved in lipids and carbohydrates metabolism, improving lipid and aminotransferases levels, thus preventing myocarditis and fibrosis in MASH mice. These findings suggest the potential of PFD for the prevention of myocarditis and fibrosis in obesity-induced MASH mice.

Ethical statement: CUCS Research Committee at the University of Guadalajara approved this study (protocol number: CI-01419, CI-02423).

Declaration of interests: None.

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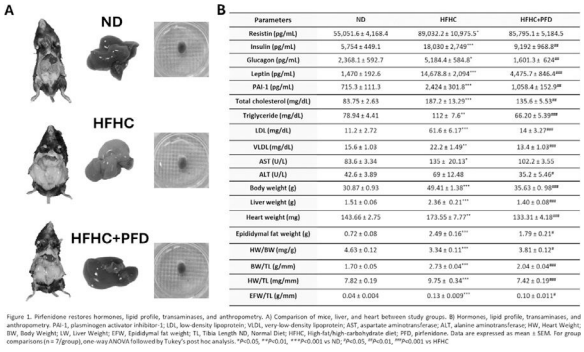

Parameters	ND	HFHC	HFHC+PFD
Resistin (pg/mL)	55,051.6 ± 4,168.4	89,032.2 ± 10,975.5*	85,795.1 ± 5,184.5
Insulin (pg/mL)	5,754 ± 449.1	18,030 ± 2,749****	9,192 ± 968.8##
Glucagon (pg/mL)	2,368.1 ± 592.7	5,184.4 ± 584.8*	1,601.3 ± 624##
Leptin (pg/mL)	1,470 ± 192.6	14,678.8 ± 2,094****	4,475.7 ± 846.4###
PAI-1 (pg/mL)	715.3 ± 111.3	2,424 ± 301.8****	1,058.4 ± 152.9##
Total cholesterol (mg/dL)	83.75 ± 2.63	187.2 ± 13.29****	135.6 ± 5.53##
Triglyceride (mg/dL)	78.94 ± 4.41	112 ± 7.6**	66.20 ± 5.39###
LDL (mg/dL)	11.2 ± 2.72	61.6 ± 6.17****	14 ± 3.27###
VLDL (mg/dL)	15.6 ± 1.03	22.2 ± 1.49**	13.4 ± 1.03###
AST (U/L)	83.6 ± 3.34	135 ± 20.13*	102.2 ± 3.55
ALT (U/L)	42.6 ± 3.89	69 ± 12.48	35.2 ± 5.46#
Body weight (g)	30.87 ± 0.93	49.41 ± 1.38****	35.63 ± 0.98###
Liver weight (g)	1.51 ± 0.06	2.36 ± 0.21****	1.40 ± 0.08###
Heart weight (mg)	143.66 ± 2.75	173.55 ± 7.77****	133.31 ± 4.18###
Epididymal fat weight (g)	0.72 ± 0.08	2.49 ± 0.16****	1.79 ± 0.21#
HW/BW (mg/g)	4.63 ± 0.12	3.34 ± 0.11****	3.81 ± 0.12#
BW/TL (g/mm)	1.70 ± 0.05	2.73 ± 0.04****	2.04 ± 0.04###
HW/TL (mg/mm)	7.82 ± 0.19	9.75 ± 0.34****	7.42 ± 0.19###
EFW/TL (g/mm)	0.04 ± 0.004	0.13 ± 0.009****	0.10 ± 0.011#

Figure 1. Pirfenidone restores hormones, lipid profile, transaminases, and anthropometry. A) Comparison of mice, liver, and heart between study groups. B) Hormones, lipid profile, transaminases, and anthropometry. PAI-1, plasminogen activator inhibitor-1; LDL, low-density lipoprotein; VLDL, very-low-density lipoprotein; AST, aspartate aminotransferase; ALT, alanine aminotransferase; HW, Heart Weight; BW, Body Weight; LW, Liver Weight; EFW, Epididymal fat weight; TL, Tiba Length ND, Normal Diet; HFHC, high-fat/high-carbohydrate diet; PFD, pirfenidone. Data are expressed as mean ± SEM. For group comparisons (n = 7/group), one-way ANOVA followed by Tukey's post hoc analysis. *P<0.05, **P<0.01, ***P<0.001 vs ND; #P<0.05, ##P<0.01, ###P<0.001 vs HFHC

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Large volume paracentesis: Is there a limit?

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Introduction and Objectives: Ascites is observed in 5-10% of cirrhotic patients. Large volume paracentesis (LVP), where >5 liters are drained, is safe. Albumin is essential to prevent post-paracentesis circulatory dysfunction (PPCD), with the literature indicating that its incidence increases when draining >8 liters in one session, suggesting draining a smaller amount.

Materials and Patients: An observational, analytical, and retrospective study was conducted, which included the clinical records of patients over 18 years of age admitted to the Gastroenterology service of the General Hospital of Mexico "Dr. Eduardo Liceaga" from January 2020 to March 2024 with a diagnosis of Grade II or III ascites, without criteria for acute kidney injury (AKI) according to the International Ascites Club (ICA) and with baseline creatinine available in