

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

Yvonne Tadeo-Jiménez, Guadalupe Zafra-Hernández, Nayeli Ortiz-Olvera

Department of Gastroenterology Hospital de especialidades "Dr. Bernardo Spúlveda", Centro Médico Nacional Siglo XXI, IMSS

**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

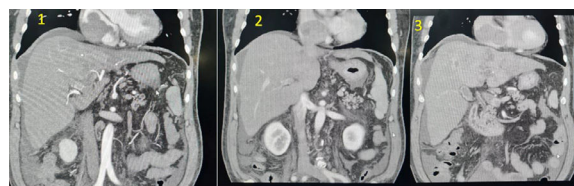
The identity of the patients is protected. Consentment was obtained.

### Declaration of interests

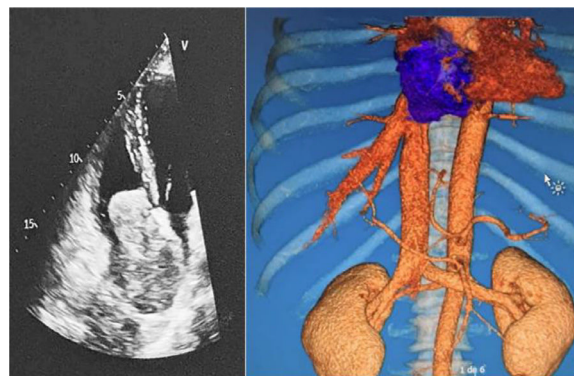
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**Figure 1:** Triphasic computed tomography. 1) Arterial phase: arterial enhancement stronger than the surrounding liver (wash-in). 2) Venous phase: hypodensity or hyposignal intensity compared to the surrounding liver (wash-out) in the venous phase.



**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 model

Daniel López-Cifuentes, Jorge Gutiérrez-Cuevas, Ana S. Sandoval-Rodríguez, Ángel O. Vázquez-Esqueda, Jonathan S. Rodríguez-Sanabria, Juan Armendáriz-Borunda

Department of Molecular Biology and Genomics, Institute for Molecular Biology in Medicine and Gene Therapy, CUCS, University of Guadalajara, Guadalajara, Jalisco, México

**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