International Journal of Research in Medical Sciences
*Blanco J et al. Int J Res Med Sci. 2025 Jan;13(1):374-378*
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20244137

**Case Report**

# Obstructive jaundice as the initial manifestation of hepatocellular carcinoma in a patient with previously unknown chronic hepatitis B infection: a case report and literature review

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**Received:** 14 October 2024
**Accepted:** 04 December 2024

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## ABSTRACT

Hepatocellular carcinoma (HCC) is the most common liver neoplasm, accounting for 90% of cases, with hepatitis B and C infections being the main risk factors for its development. Diagnosis is primarily based on imaging studies, serology, and histology. Clinically, it presents with right upper quadrant pain and B symptoms, with jaundice occurring in 19-40% of cases. However, the onset of obstructive jaundice as the initial symptom occurs in approximately 1-12% of cases. We present the case of a 65-year-old male with a recent diagnosis of diabetes mellitus, who presented with progressive jaundice, documenting suggestive imaging findings of hepatocellular carcinoma with portal involvement and a positive viral panel for hepatitis B. Pathology report confirmed a differentiated hepatocellular carcinoma.

**Keywords:** Hepatocellular carcinoma, Hepatitis B, Obstructive jaundice

## INTRODUCTION

The World Health Organization estimates that by 2030, one million people will die from liver cancer. Globally, liver cancer is the 4th leading cause of cancer-related death, with hepatocellular carcinoma (HCC) being the main malignant liver tumor.[1] It typically occurs in patients with underlying liver disease due to chronic infections with hepatitis B and C viruses, non-alcoholic steatohepatitis, or alcohol consumption. Other associated factors include age over 70 years, male sex, and certain ethnic and racial aspects.[2]

The pathophysiology of HCC is a complex process involving multiple steps, developing through interactions between genetic predispositions, reciprocal interactions between viral and non-viral risk factors, the cellular microenvironment, the immune system, and the severity of underlying chronic liver disease.[3]

The typical clinical presentation of hepatocellular carcinoma includes right upper quadrant pain and weight loss. Jaundice occurs in 19-40% of patients, but only 1-12% of cases present with obstructive jaundice as the initial clinical manifestation.[4,5] In this report, we present a rare case of obstructive jaundice as the first symptom of HCC in a patient with a previously unknown chronic hepatitis B infection.

The treatment of choice for HCC is surgical when the tumor is resectable. Although cases of successfully resected icteric-type HCC have been described, most

patients with this presentation are inoperable. The alternative treatment strategy includes palliative care for the tumor and relief of the jaundice. The prognosis for patients with HCC and jaundice is poor, with cholangitis being the leading cause of death in these patients.[5]

## CASE REPORT

A 65-year-old male was admitted to our hospital in July 2024 due to a one-month history of jaundice, dark urine, and pale stools. He denied weight loss, fever, constitutional symptoms, or other complaints. His past medical history included a recent diagnosis of type 2 diabetes mellitus, treated with lifestyle modifications. He had no history of chronic diseases, alcohol consumption, tobacco use, or illicit drug use. He also denied risky sexual practices and reported only three sexual partners throughout his life.

On admission, he presented with conjunctival jaundice and dark urine. Physical examination revealed loss of axillary hair, telangiectasias on the anterior thorax, and palmar erythema. Abdominal palpation revealed no hepatomegaly or splenomegaly. Laboratory results were as follows: total bilirubin 26.1 mg/dl, indirect bilirubin 12.7 mg/dl, direct bilirubin 13.4 mg/dl, alanine aminotransferase (ALT) 122 U/l, aspartate aminotransferase (AST) 61 U/l, alkaline phosphatase (ALP) 247 U/l, gamma-glutamyl transferase (GGT) 123 U/l, albumin 3.2 g/dl, urea 13 mg/dl, creatinine 0.53 mg/dl, prothrombin time (PT) 15.4 s, activated partial thromboplastin time (aPTT) 30.8 s, and international normalized ratio (INR) 1.43. Serology for hepatitis B was positive for surface antigen, IgG anti-HBc, and anti-HBe, confirming a diagnosis of chronic hepatitis B infection. Serology for hepatitis C was negative. Alpha-fetoprotein levels were 2.37 ng/ml, and CA 19-9 was negative at 28.87 U/ml.

An ultrasound of the liver and bile ducts revealed a heterogeneous liver measuring 166 mm in its largest longitudinal axis, with a 95×106 mm solid, oval lesion that was predominantly hypoechoic with internal echogenic areas and a small 15 mm cystic zone, extending to liver segments IVA and IVB. There was intrahepatic biliary dilation measuring 8 mm and a 19 mm portal vein with a 57×20 mm thrombus. Collateral vessels were noted, with centripetal flow towards the left portal branch and adequate color saturation. Moderate perihepatic fluid was also observed (Figures 1-3).

A three-phase abdominal computed tomography (CT) scan revealed a hypodense, oval-shaped lesion occupying the left lobe and part of the right lobe, measuring 135×94×110 mm, with an attenuation index of 42 HU. In the contrast phase, the lesion showed enhancement and faster washout than the rest of the parenchyma. Vascular pathways within the lesion displayed rapid and persistent enhancement. This lesion displaced the portal vein and caused intrahepatic biliary dilation (Figure 4).

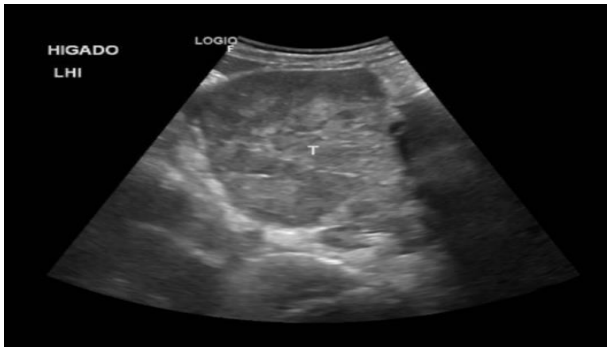


**Figure 1: Abdominal ultrasound showing the left hepatic lobe in segments IVA and IVB, with a solid, oval lesion measuring 95×106 mm.**

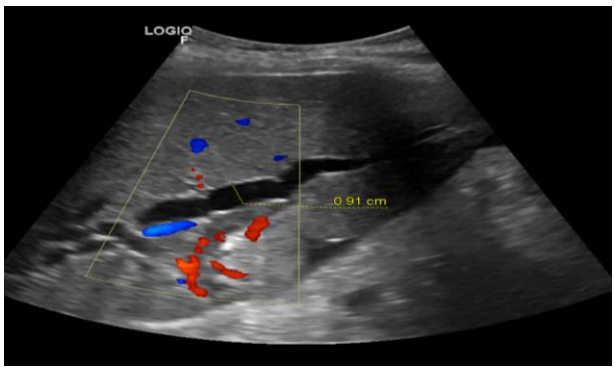


**Figure 2: Hepatic ultrasound showing intrahepatic biliary dilation measuring 91 mm.**


**Figure 3: Doppler ultrasound showing an avascular portal vein.**

A CT-guided biopsy was performed, and histopathological examination confirmed hepatocellular carcinoma with extensive necrosis and foci of acute inflammation (Figure 6). Staging studies included a thoracic CT scan, which showed no evidence of pulmonary metastasis.

The Medical Oncology team recommended resolving the obstructive jaundice through drainage and later considering palliative treatment. The patient was classified as stage D according to the Barcelona clinic liver cancer (BCLC) staging system, due to cirrhosis (Child-Pugh class C) and portal involvement. Endoscopic retrograde

cholangiopancreatography (ERCP) was attempted but failed due to an inability to cannulate the biliary confluence, likely due to tumor activity at the common bile duct.

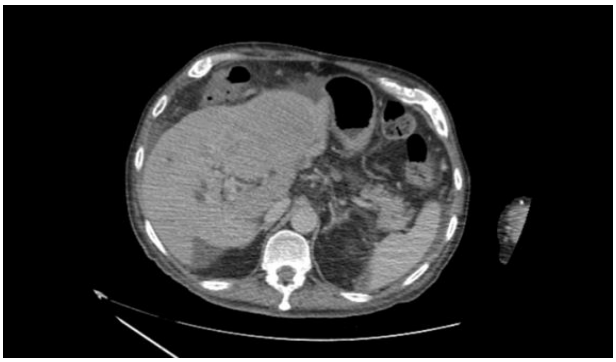

**Figure 4: Simple phase CT scan showing a hypodense, oval-shaped lesion occupying the left and part of the right hepatic lobe, measuring 135×94×110 mm, with portal displacement.**

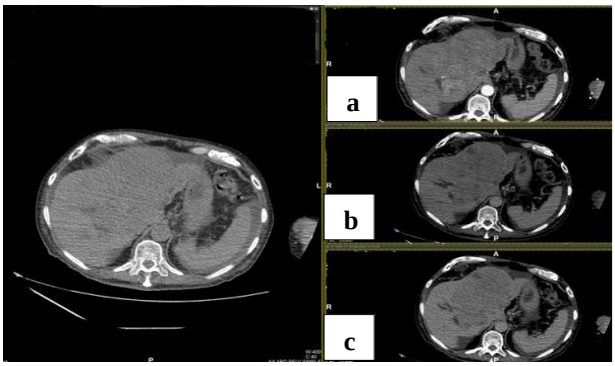


**Figure 5: (a) Arterial phase, (b) portal phase, and (c) venous phase showing rapid enhancement and washout compared to the rest of the parenchyma, with intense and persistent enhancement of vascular structures within the lesion.**

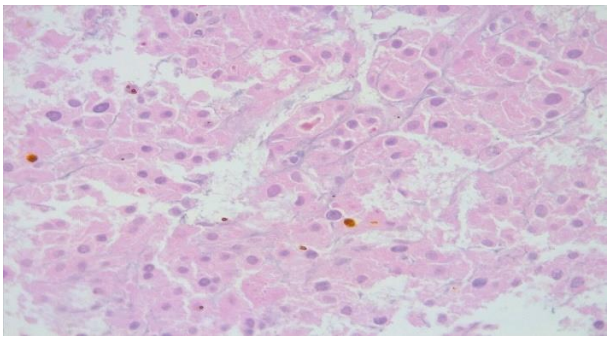

**Figure 6: Histopathology with H&E staining showing multiple hepatocytes with extensive necrosis and foci of inflammation consistent with differentiated hepatocellular carcinoma.**

Magnetic resonance cholangiopancreatography (MRCP) showed a mass in the left hepatic lobe extending to the right lobe, with displacement and dilation of the intrahepatic bile ducts. Percutaneous biliary drainage was proposed, but the patient developed acute cholangitis and hepatic encephalopathy. He was treated with piperacillin-tazobactam for 7 days, along with lactulose and rifaximin, with satisfactory clinical improvement. Subsequent plans for percutaneous biliary drainage were rejected by the patient, and after a joint decision with the Medical Oncology and Palliative Care teams, it was decided to continue with palliative management.

## DISCUSSION

Liver cancer is the sixth most common neoplasm worldwide and the fourth leading cause of cancer-related death. The highest incidence is in Asia and Africa. HCC represents 75-86% of liver cancer cases. The prognosis remains poor globally, with a mortality rate of 8.5 per 100,000 person-years.[6] The development of HCC involves multiple patient-related and environmental factors. Host-related factors include age (60-74 years), male sex, and genetic predisposition. Environmental risk factors include viral infections, alcohol-related liver disease, and exposure to toxins such as aflatoxin and aristolochic acid.[7]

Ninety percent of cases occur in the context of chronic liver disease, with cirrhosis being the strongest risk factor for developing hepatocellular carcinoma. Globally, hepatitis B virus infection is the leading cause of HCC.[2]

Our patient had risk factors such as age and alcohol consumption but had no prior diagnosis of chronic liver disease despite physical examination findings and laboratory abnormalities indicative of liver disease, such as thrombocytopenia, hypoalbuminemia, and hyperbilirubinemia. Diagnostic tests confirmed chronic hepatitis B infection. During hospitalization, ultrasound revealed ascites, and the patient developed hepatic encephalopathy, leading to a final diagnosis of liver cirrhosis (Child-Pugh class C, 10 points).

The typical clinical presentation of HCC includes right upper quadrant pain and weight loss. Jaundice occurs in 19-40% of patients, but only 1-12% of cases present with obstructive jaundice as the initial clinical manifestation.[8]

In our patient, there was no abdominal pain, palpable mass, or weight loss. The only presenting symptom was one month of progressive jaundice, along with dark urine and pale stools, a rare form of presentation in HCC.

Diagnosis of hepatocellular carcinoma can be made non-invasively in at-risk populations based on imaging criteria without the need for histopathological confirmation. In these patients, arterial phase enhancement and washout in the portal venous phase on multiphase CT or MRI is considered the radiologic hallmark of hepatocellular carcinoma, with high specificity and positive predictive value for lesions larger than 1 cm. The LI-RADS classification system is based on imaging features such as

tumor size, intense arterial enhancement, and late-phase washout, categorizing hepatic nodules from LR-1 (benign) to LR-5 (hepatocellular carcinoma).[9] Nodules larger than 2 cm with intense arterial enhancement and washout are classified as LR-5, sufficient for diagnosis.

Previously, alpha-fetoprotein was considered a diagnostic criterion, with levels above 400 ng/ml, but up to 40% of patients with hepatocellular carcinoma can have normal levels, and elevation can also occur in other cancers such as intrahepatic cholangiocarcinoma, gastric cancer, and germ cell tumors. Therefore, it is no longer considered a diagnostic tool.[10]

In our patient, an ultrasound detected a 9 cm nodule, prompting the request for a three-phase CT scan, which documented a 10 cm nodule with arterial-phase enhancement and venous-phase washout, classifying it as LR-5 with a high probability of HCC. Alpha-fetoprotein levels were within the normal range, but due to the atypical presentation, a CT-guided biopsy was performed, confirming the diagnosis of HCC.

In terms of staging, the BCLC system remains the most widely recommended staging system in treatment guidelines, as it has been validated in multiple cohorts, is user-friendly, and incorporates liver dysfunction and patient performance status into its categories. The system classifies patients into category 0 (very early), followed by categories A-D, with stage D representing terminal disease. For all patients except those in category 0, a thoracic CT scan is recommended to rule out the presence of pulmonary metastases.[11] In our patient, a thoracic CT scan was performed, which showed no findings suggestive of pulmonary involvement. He was classified as stage D due to advanced liver failure and invasion of structures such as the biliary tract and portal system.

Treatment is defined based on the BCLC classification, tumor size, performance status, tumor extension, and severity of liver dysfunction.[12] Treatment modalities include curative therapy through surgical resection and ablation for early-stage patients without liver cirrhosis, transplantation for patients with portal hypertension and decompensated cirrhosis, transarterial chemoembolization, systemic therapy, and palliative support for advanced tumors.[12,13]

Biliary tract invasion significantly impacts survival, similar to vascular invasion, yet it is rarely mentioned in guidelines. Biliary tract invasion typically occurs in intermediate and advanced stages, with an incidence of 0.5-13% and is associated with varying degrees of obstructive jaundice. Jaundice is generally due to tumor growth within the biliary tree or compression of the bile duct. Biliary invasion can be classified into extraluminal obstruction, intraluminal obstruction, and hemobilia.[14]

Standard treatments for HCC, such as resection, ablation, transarterial embolization, and systemic therapy, are limited by liver damage caused by obstructive jaundice, which exacerbates the tumor burden in patients with this type of cancer. Treatment guidelines generally do not address the effects of biliary tract invasion on treatment selection.[15]

Most studies addressing this presentation suggest tumor resection and transplantation for patients presenting at early stages, provided they are eligible. The role of preoperative biliary drainage remains controversial. For unresectable tumors, alternatives such as endoscopic biliary drainage, intraluminal stent placement, percutaneous biliary drainage, choledochotomy with T-tube placement, and surgical bypass have been proposed.[5,16,17]

In this case, the patient was classified as stage D due to advanced liver dysfunction and the presence of biliary and portal invasion. Palliative treatment was offered due to the impossibility of surgical resection or transplantation. Endoscopic biliary drainage was attempted but failed due to tumor activity at the biliary confluence. Subsequently, percutaneous drainage was suggested, but the patient rejected further procedures and opted to continue with supportive palliative care at home.

## CONCLUSION

HCC remains a significant cause of mortality worldwide. Most cases are associated with underlying liver disease, including hepatitis B virus infection. Obstructive jaundice secondary to biliary tract invasion is a rare form of presentation and continues to be a poor prognostic factor in these patients. The treatment of HCC with biliary invasion is not typically addressed in international guidelines, and available information suggests surgical resection of the tumor when feasible, along with palliative treatment that includes endoscopic or surgical biliary drainage when it is not.

*Funding: No funding sources*
*Conflict of interest: None declared*
*Ethical approval: Not required*

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**Cite this article as:** Blanco J, González LD, Cortes A, Jimenez N, Pavía MR, Gutierrez MX. Obstructive jaundice as the initial manifestation of hepatocellular carcinoma in a patient with previously unknown chronic hepatitis B infection: a case report and literature review. Int J Res Med Sci 2025;13:374-8.