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Hepatocellular Carcinoma Revealed by Bone Metastases: A Case Report at The National Hospital and University Center in Cotonou

Aboudou Raimi Kpossou[1,2,3], Noume Bella Doumbouya[1], Akotchaye Marcel Kakpo[1], Avadra Ebénizaire[1], Comlan N'déhougbea Martin Sokpon[1,2], Rodolph Koffi Vignon[1,2], Julien Djossou[1,2], Zavier Zomalheto[2,4] and Jean Séhonou[1,2]

[1]University Clinic of Hepatology and Gastroenterology, Hubert Koutoukou Maga National University Hospital Center (CNHU-HKM), Cotonou, Benin

[2]Faculty of Health Sciences, University of Abomey-Calavi, Cotonou, Benin

[3]Hepatology and Gastroenterology Department, Calavi International Hospital Center (CHIC), Abomey-Calavi, Benin

[4]University Rheumatology Clinic, CNHU-HKM, Cotonou, Benin

***Corresponding author:**

Aboudou Raïmi Kpossou,
University Clinic of Hepatology and Gastroenterology, Hubert Koutoukou Maga National University Hospital Center (CNHU-HKM), Cotonou, Benin, Faculty of Health Sciences, University of Abomey-Calavi, Cotonou, Benin and Hepatology and Gastroenterology Department, Calavi International Hospital Center (CHIC), Abomey-Calavi, Benin

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1. Abstract

Hepatocellular carcinoma (HCC), a major public health problem in Benin, can manifest itself through extra-digestive complications. We report a case of HCC revealed by spinal metastases with neurological compression. The patient was a 48-year-old man with untreated occult viral hepatitis B. He consulted for non-traumatic, non-febrile lumbar pain, and paraclinical investigations led to a diagnosis of hepatocellular carcinoma complicated by spinal bone metastases. Under symptomatic palliative treatment (corticosteroid, analgesics, bisphosphonate, nucleotide analogue, prophylactic anticoagulation), the patient died approximately three weeks after diagnosis.

2. Introduction

Hepatocellular carcinoma is the most common primary liver tumor. It usually develops in a cirrhotic liver in 70 to 90% of cases, more rarely in chronic non-cirrhotic liver disease, and exceptionally in a healthy liver [1]. It is often detected late, at the metastatic stage, in 64% of cases [2]. The most common secondary sites are the lungs, lymph nodes, kidneys, and adrenal glands. Bone metastases are rare, and their sites of choice are the vertebrae, ribs, and, more rarely, the sternum. Hepatocellular carcinoma revealed by bone metastases is exceptional [3, 4]. We report a case of hepatocellular carcinoma revealed by bone metastases, which progressed rapidly and unfavorably.

3. Observation

The patient was a 48-year-old man with untreated occult hepatitis B, no history of alcohol abuse or smoking, and a family history of liver cancer in an older brother who died at the age of 42. The onset of symptoms dated back to three months prior to his admission, marked by the onset of non-traumatic, non-febrile lumbar pain of progressive onset, described as a pulling sensation, with an intensity of 6/10 on a numerical scale, impulsive when coughing, not causing insomnia, relieved by rest, aggravated by exertion, radiating to the pelvic limbs along an irregular path affecting the inner thighs, the front of the legs, and ending at the ankles. These symptoms led to unspecified self-medication. The progression was marked by persistent pain associated with anorexia, weight loss of 13% of his usual weight in 3 months, and functional impotence of the pelvic limbs, which led to hospitalization at the Hubert Koutoukou Maga National University Hospital Center (CNHU/HKM) in Cotonou. A thoraco-abdominal-pelvic CT scan revealed tumor-like liver lesions, prompting his transfer to the CNHU/HKM's university hepatology and gastroenterology clinic in Cotonou for better care. On osteoarticular examination, standing and sitting were impossible. The examination was performed in the supine position, revealing a protrusion of the spinous processes at L4 to L5, band-like scarring in the lumbar region, slight palpable spinal curvature from L4 to S1, slight paravertebral muscle con-

tracture with a positive Lasègue sign at 45° on the right and 70° on the left. The examination of the digestive system was normal, with no palpable mass and a hepatic edge at 9 cm on the right midclavicular line. Magnetic resonance imaging (MRI) of the spine revealed a severe compression fracture (grade 3) of L4 causing compression of the dural sheath with epiduritis and spinal cord damage associated with staged signal abnormalities of T10, T12, L1, and S1, lumbar disc disease from L3-L4 to L5-S1 conflicting with the emergence of left L5 and right S1, and staged lumbar spondylosis. A thoracoabdominal-pelvic CT scan then performed revealed a large tumor mass in segments VII and VI with small nodules in segments III and IV that are hypervascular in the arterial phase and wash out in the portal phase (Figures 1 and 2), associated with bilateral pulmonary metastases in the form of bilateral pulmonary carcinomatous lymphangitis, left posterobasal pleural thickening, vertebral and pelvic bone metastases, and compression of the L4 and T10 vertebral bodies with rupture of the posterior vertebral wall and L4, suggesting intra-canal extension of the tumor process (Figure 3). Alpha-fetoprotein was greater than 400 ng/mL, and cirrhosis-type pre-neoplastic lesions were not confirmed due to the absence of sign of portal hypertension (platelet count was normal at 290 giga/L) there were no evidence of hepatocellular insufficiency (prothrombin level at 88%, albuminemia at 37.9), non-invasive markers of fibrosis (APRI score = 0.29 and FIB4 = 1.05), upper gastrointestinal endoscopy could not be performed. Occult viral B etiology was considered given the negative HBs antigenemia, positive total anti-HBc antibodies, and HBV DNA polymerase chain reaction detectable at 10 IU/mL, or 1.28 log. Aminotransferases were normal (AST = 29 IU/L, ALT = 40 IU/L). HDV, HCV and HIV serology were negative. Calcemia was normal (94 mg/L), vitamin D was slightly low (24.0 ng/L). The rest of the blood test results: creatinine at 8 mg/L with a glomerular filtration rate of 134.23 mL/minute, normal blood ionogram: (chloride: 97 mEq/L, calcium: 94 mg/L, magnesium: 21 mg/L, phosphorus = 32 mg/L), mixed jaundice with total bilirubin at 15 mg/L and conjugated bilirubin at 5 mg/L, gamma glutamyl transferase at 298 IU/L, blood count was normal (hemoglobin = 12.1 g/dL,white blood cells: 7.7 Giga/L, and platelets = 290 Giga/L). Serum protein electrophoresis revealed an increase in alpha 1 and alpha 2 globin indicating an ongoing chronic inflammatory reaction (albumin = 37.9 g/l, gamma= 13.5 g/l, beta2= 4.6 g/l , beta1=3.4 g/l, alpha1=3.9 g/l, alpha 2=11.1 g/l; total protein=74 g/l), CRP was elevated at 48 mg/l, total PSA level = 2.33 ng/ml. The diagnosis was multi-metastatic hepatocellular carcinoma (vertebral, iliac, trochanteric, pleuropulmonary) classified as BCLC D, post-viral B occult. The patient received treatment consisting of hospitalization, preventive anticoagulation with enoxaparin 0.4 ml/24 hours, hydration with two liters of saline solution/24 hours, a high-calorie, high-protein diet, tenofovir 300 mg/day, corticosteroid therapy consisting of Solumedrol 120 mg injection followed by Cotipred 60 mg after consultation with neurosurgery, zoledronic acid 4 mg/4 weeks IV, gabapentin 300 mg (900 mg/day), and immobilization with a corset. The treatment decision was made during a multidisciplinary consultation and was announced to the patient during a consultation with a detailed treatment plan consisting of first-line immunotherapy, which was not available, and second-line systemic treatment with Sorafenib combined with supportive care.

The patient's condition improved a little at first with the lumbar pain reduced to 2/10 on the numerical scale after 48 hours of analgesic treatment, and he was able to leave the hospital. However, he died on the twentieth day after his discharge from hospital.

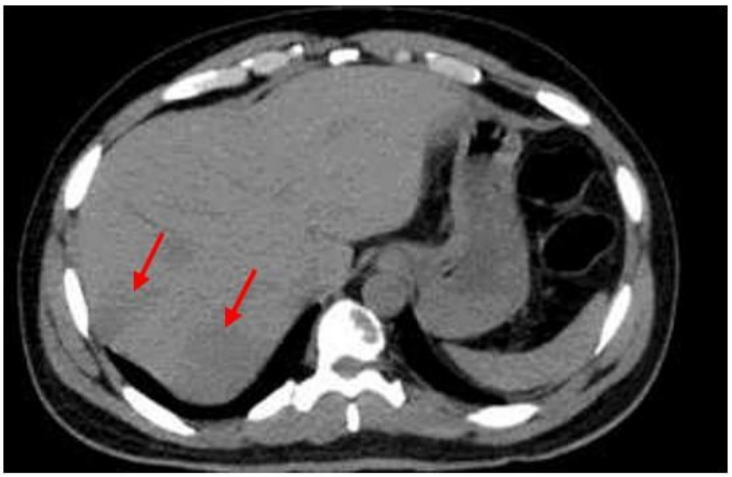
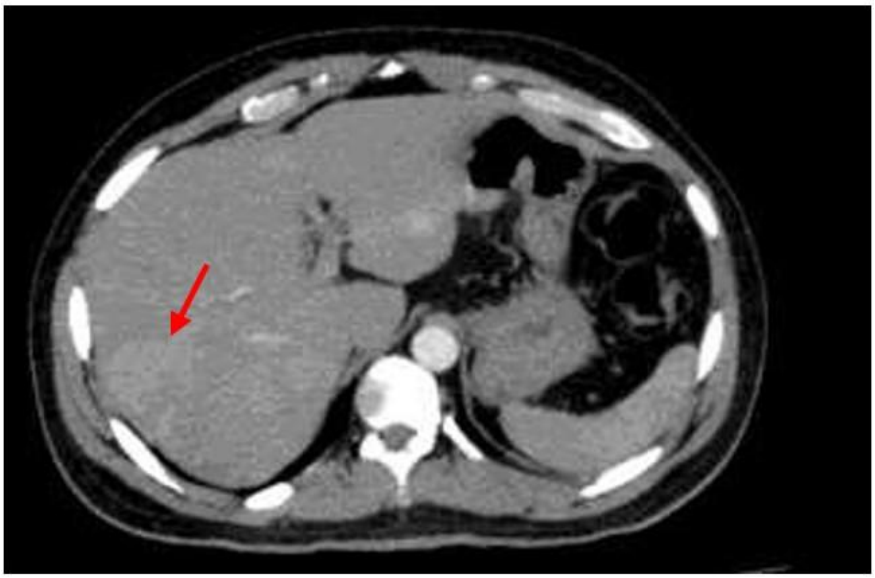


Figure 1: A dysmorphic liver with bumpy contours and a large mass straddling segments VII and VI on abdominal scan.



.**Figure 2**: Fleshy portions are hypervascular in the arterial phase on abdominal scan

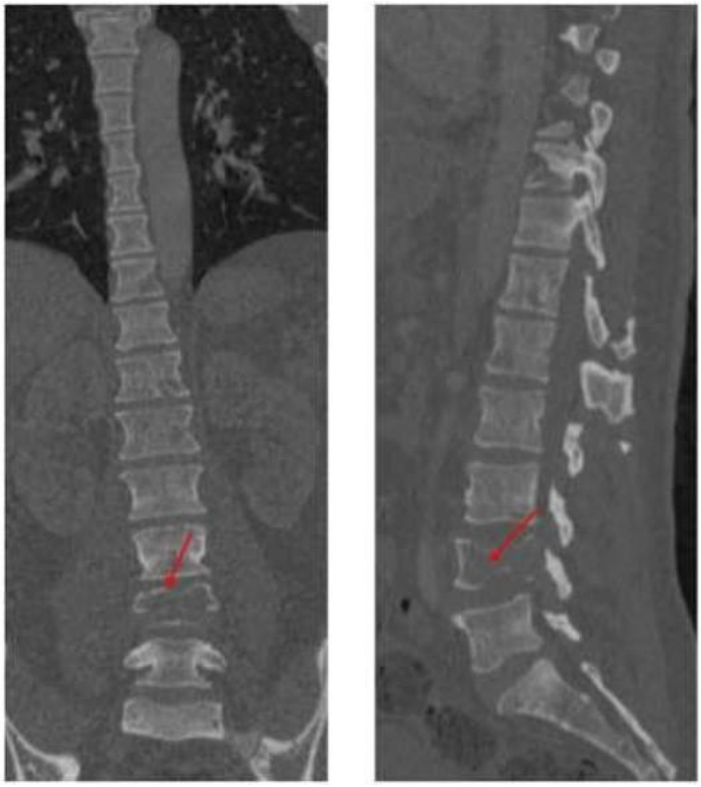


Figure 3: Multiple osteolysis with blurred contours and rupture of the posterior wall of L4 on CT scan.

4. Discussion

This clinical case is unique in several respects. Hepatocellular carcinoma (HCC) occurs in 80% of cases in livers with cirrhosis or chronic liver disease, the main risk factors for which are viral infections B and C, excessive alcohol consumption and metabolic dysfunction-associated steatohepatitis. The prevalence of metastases in HCC varies from 30.8% to 91.5% from one series to another [5, 6]. The discovery of bone metastases during the course of HCC is a recognized occurrence and has become increasingly common given the improvement in patient survival rates due to major advances in early detection, diagnosis, and treatment of hepatocellular carcinoma. However, the fact that these bone metastases reveal HCC is exceptional, reported in 7% of cases [3]. These bone locations are the spine, ribs, pelvis, skull, and more rarely the sternum [3,4]. We noted a vertebral location in our case. The patient was male. A review of the literature revealed similar cases reported in three African series: Sidibé et al [2] in Morocco in 2023 (38H/5F), Niasse et al [7] in Senegal in 2024 (2H/1F), Bayala et al [8] in Burkina Faso in 2024 (2M), and an American series, Ruchi et al [9] from 2005 to 2015 (17M/3F). The number of cases remains limited in the African series (2 to 3 cases), reflecting the rare or poorly documented nature of bone metastases indicative of HCC in our contexts. A relatively long diagnostic delay (seven weeks on average) and an almost constant deterioration in general health (70% of cases) [3, 4]. In our reported case, the delay between the onset of symptoms and diagnosis was one to six months, and the patient presented with a deterioration in general health. The common clinical presentation of HCC bone metastases is a hard, pain-

less swelling compressing the surrounding structures [4,5], which was not the case in this patient. On X-ray, it is often a normo- or hyperfixating lytic lesion on bone scintigraphy [11]. The patient did not undergo bone scintigraphy. Histologically, bone metastasis reproduces the anatomic-pathologic architecture of the primary tumor (HCC) with clearer differentiation and, exceptionally, bile pigments in the metastatic sample [12]. The patient did not undergo histological testing prior to his death. In terms of etiology, based on a review of the literature, viral causes (HBV or HCV) are predominant, reflecting the high prevalence of these infections. However, what is unique about our study is that cirrhosis had not been diagnosed and that the cases involved occult viral hepatitis B. Radiotherapy appears to be useful as it provides analgesia in 79% to 92% of patients during or after treatment, as with all bone metastases from other tumors [13]. It also improves 6-month survival. Regardless of the tumor size of the bone metastasis, the total effective radiation dose is around 30 to 50 grays [14]. Radiotherapy is not yet available in Benin. This is why the patient was unable to benefit from this treatment.

5. Conclusion

Hepatocellular carcinoma (HCC) remains a major public health challenge, particularly in Africa and Benin, where it is often discovered at an advanced stage. The epidemiology and high prevalence of viral hepatitis make it a common disease. Bone metastases revealing HCC are rare ; however, they should be considered in any case of lytic bone lesion, especially in patients with chronic liver disease. Given their poor prognosis, treatment is palliative, aimed primarily at improving patients' quality of life.

References

1. Zucman-Rossi J, Villanueva A, Nault JC, Llovet JM, De Reyniès A. Genetic landscape and biomarkers of hepatocellular carcinoma. Gastroenterology. 2015; 149(5): 12261239.
2. Sidibe R, Samlani Z, Krati K, Oubaha S, Nadir A, Jalal H. Bone metastases revealing hepatocellular carcinoma: three case reports. Hegel. 2017; 1(1): 60-5.
3. Okazaki N, Yoshino M, Yoshida T, Hirohashi S, Kishi K, Shimosato Y. Bone metastasis in hepatocellular carcinoma. Cancer. 1985; 55(9): 1991-4.
4. Hedri H, Mhibik S, Abderrahim E, Goucha R. Sternal metastasis revealing hepatocellular carcinoma. Rev Med Interne. 2004; 25(3): 238-41.
5. Liver Cancer Study Group of Japan. Primary liver cancer in Japan. Cancer. 1984; 54(8): 1747-55.
6. Si MS, Amersi F, Golish SR, Ortiz JA, Zaky J. Prevalence of metastases in hepatocellular carcinoma: risk factors and impact on survival. Am Surg. 2003; 69(10): 879-85.
7. Niasse A, Ndiaye PI, Mané M, Ndiaye A, Dieng PS, Sall F, et al. Hepatocellular carcinoma revealed by bone metastasis: report of three cases. Int J Surg Open. 2024; 62(4): 352-5.
8. Bayala YLT, Tinni IA, Kabore F, Zabsonré/Tiendrebeogo JWS, Ouedraogo DD. Vertebral metastasis of hepatocellular carcinoma secondary to viral hepatitis B: case report of two patients. Wiad Lek. 2024; 77(2): 358-62.
9. Bhatia R, Ravulapati S, Befeler A, Dombrowski J, Gadani S, Poddar N. Hepatocellular carcinoma with bone metastases: incidence, prognostic significance, and management—single-center experience. J Gastrointest Cancer. 2017; 48(4): 321-5.
10. Raoul JL, Le Simple T, Le Prisé E, Meunier B, Ben Hassel M, Bretagne JF. Bone metastasis revealing hepatocellular carcinoma: a report of three cases with a long clinical course. Am J Gastroenterol. 1995; 90(7): 1162-4.
11. Maillefert JF, Tebib J, Ahas S. Bone metastases from hepatocellular carcinoma: 22 observations. Rev Rhum. 1993; 60: 907-12.
12. Cottin S, Caumon JP, Giban H. Bone metastases indicative of hepatoma. Rev Rhum. 1981; 48:347-55.
13. Kajiwara Y, Fukurono K, Ohtake H. Radiotherapy for bone metastasis from hepatocellular carcinoma. Ther Res. 1990; 11(26): 2669-74.
14. Matsuura M, Nakajima N, Ito K. Radiation therapy for bone metastasis of hepatocellular carcinoma. Int J Clin Oncol. 1998; 3(1): 31-5.