

Scalp and Skull Lesion a Rare Presentation of Hepatocellular Carcinoma: A Case Report and Review of Literature

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Summary

Hepatocellular carcinoma (HCC) is commonest primary tumor of liver as well as the fourth leading reason of carcinoma related death worldwide. The most frequent sites of extra hepatic metastases of HCC are lungs, regional lymph nodes, kidney, adrenal and bone marrow. While skull metastases from HCC are rare, metastasis to scalp and skull on presentation of HCC are reported very rarely in the literature. Here we report rare case of HCC presented as asymptomatic metastatic lesion in the scalp and skull in a chronic hepatitis B patient. Excision of the scalp lesion was performed and on histopathology revealed metastatic Adenocarcinoma, Immunohistochemistry (IHC) report confirmed metastasis from HCC. On further investigation, two liver lesions with periportal nodes and left adrenal metastasis were detected and patient was started on sorafenib. After progression on sorafenib, regorafenib was started; ultimately he died after two months.

Keywords: Hepatocellular Carcinoma, Skull Metastasis, Metastatic Adenocarcinoma

Introduction

HCC is commonest primary tumor of liver as well as predicted to be 6th most commonly diagnosed carcinoma, while 4th leading reason of cancer related deaths worldwide.¹ HCC may occur in association with chronic hepatitis caused by hepatitis B (HBV) or hepatitis C (HCV) viral infections which are common causes of hepatic cirrhosis. HCC is commonly occurring in the 6th to 7th decade of life. Extra hepatic metastases are seen in 55-65% of patients with HCC. Commonest sites of extra hepatic metastases of HCC are pulmonary metastasis (35-65%), common hepatic, periportal nodes (15-39%) but rarely to bones (1.5-15%). The incidence of skull metastases from HCC is very rare (0.4-1.6%).²⁻⁵ We report a rare case of asymptomatic metastatic lesion in the scalp and skull from HCC.

Case Report

A 49 year old adult male patient visited the neurosurgery department at our hospital with a painless scalp lump along midline of vertex region which he noticed accidentally 2 months back. Then he noticed that it was growing in size very quickly. According to him, no history of head trauma as well as

no past history of medical illness was present except for chronic HBV related chronic liver parenchyma disease (on Tenofovir for last 3 years). Neurological and physical examination revealed painless, firm mass with restricted mobility, two in number, in the midline of scalp, larger anterior lesion around 70x50 mm; with no neurological deficits or other obvious abnormalities.

His initial laboratory reports revealed hemoglobin: 15.4 g/dl, hematocrit: 45.5, white blood cell count 6400, platelets: 186000, prothrombin time (INR): 0.9, blood glucose: 98mg/dl, aspartate transaminase: 107 u/l, alanine transaminase: 44 u/l, alkaline phosphatase: 220 IU/L, albumin: 3.9gm/dl, globulin: 3.1gm/dl, total bilirubin: 0.67 mg/dl and normal renal function test. HBsAg was confirmed positive. alpha feto protein (AFP) was normal (16 ng/ml).

Ultrasonography of the abdomen revealed mild hepatomegaly with diffuse altered echo texture of liver. MRI brain with contrast showed large mixed intensity lesions (two in number) in midline in relation to parietal bone. The lesions were lytic in nature being hypo intense on T2W/FLAIR image with intralesional hyper intense area with iso to hypo intense on T1W image. Anterior lesion was larger measuring 71x51 mm in size. Both lesions showed intra as well as extra cranial component.

Excision of the scalp lesions was performed, and histopathological examination revealed metastatic adenocarcinoma. Meanwhile patient was given whole brain radiotherapy. Subsequently IHC report was positive for AE1, hepatocytes (Heppar), Glypican, with TTF1-, confirming metastatic HCC. On further workup with CECT (abdomen) two liver lesions (37x45 mm, 23x22 mm), few periportal lymph nodes (13x8 mm) with large (66x48 mm) left adrenal metastasis was noted, hence treatment was started with oral sorafenib. After 5 months of progression free interval on sorafenib, patient presented with backache for which CECT abdomen pelvis thorax was performed and new bony lytic lesion at D7 vertebra

with soft tissue component of size 27x30 mm with increased abdominal lymphadenopathy were seen. Patient was given radiotherapy to local site for palliation of symptoms. Later patient was started on regorafenib and he died after 2 months.

Discussion

HCC is one of the most common malignancies worldwide; its occurrence is high in the area where HBV or HCV infections are endemic or commonly seen. Cirrhosis related HCC is most commonly due to HCV (25-75%) followed by HBV (15-50%), chronic alcohol consumption related (5-35%), hemochromatosis as well as idiopathic (5%).⁶ Commonest sites of metastasis from HCC are lungs, loco regional nodes and adrenals. Its extra hepatic presentation usually occurs in cases with advanced intrahepatic tumor growth. In our case report the patient was having scalp and skull lesion with asymptomatic liver lesion with normal bilirubin and normal AFP.

Yanase et al group studied postmortem autopsy results of around 4000 patients of hepatocellular carcinoma from Japan and they also reported rare occurrence of bony metastasis as well as very rarely reported metastasis of HCC to cranium in very few patients (<5%), which correlated with rare incidence of extra hepatic metastatic pattern of HCC given in the literature.⁷

Hsieh et al also reported few cases of HCC metastasized to skull that had mean age of fifty seven years.⁸ Six patients also had associated multifocal involvement of skull bones. Most of them presented as a lump in the skull region, some of them had mild pain along with swelling. On skull imaging most of them had lytic bony lesions.⁸

Kuratsu et al also recorded a case of HCC with central nervous system metastases (cranial bones) that had shown high serum AFP.⁹ On contrary, in our reported case AFP was in normal range. There are very scanty data on imaging characteristic pattern and of HCC with cranial or skull metastasis. Kuratsu et al reported typical MRI findings of the cranial metastasis as lytic as well as expansile bony lesions, which were iso intense on T1 in few cases while hypo intense on T2 in some cases relative to cerebral white matter.⁹

Guo et al reported one adult male patient of HCC who presented with single, painless midline lump in vertex region of cranium.¹⁰ He had no past history of medical illness. Brain CT demonstrated a hyper vascular enhancing lesion associated with lytic bony lesion in high parietal and occipital area, MR imaging of brain demonstrated lytic as well contrast enhancing bony lesion. On CT abdomen-pelvis revealed a large hepatic mass, without any metastatic lesion elsewhere. Laboratory test revealed normal

liver function tests but serology report was HBsAg+, with raised AFP value. He had undergone craniotomy with total excision of lump. On histopathological examination, metastatic lesion from HCC was confirmed. His primary tumor was treated with Transcatheter arterial chemoembolization (TACE) using chemotherapy (Pirarubicin + Carboplatin + Floxuridine), with lipiodol as well as gelatin sponge.¹⁰ ultimately, he died due to acute respiratory distress syndrome (ARDS).¹⁰

Like our case report, Goto et al reported a 56-year-old male with skull and vertebral metastasis from HCC which presented as with occipital mass.¹¹ Contrast CT demonstrated lytic hyper vascular enhancing lesion of cranial bone with a solitary large hepatic tumour. MRI revealed multiple dorsal vertebral metastatic lesions. On serology he was positive for both HBsAg and anti HBC antibody, with normal liver functions tests. Finally confirmation of HCC with solitary primary lesion with metastatic lesion in the cranium and dorsal vertebrae was made. He was treated with TACE to primary hepatic lesion and radiotherapy for bony lesions.¹¹

For treating bony metastatic lesion of the cranium, multidisciplinary team management involving medical oncologist, surgical oncologist, radiation oncologist and palliative medicine expert should be considered to alleviate the pain, to decrease suffering from neurocognitive dysfunction and to improve or maintain quality of life.

Conclusion

There are only few case reports about skull and scalp metastasis from HCC. Even if infrequent, this should be considered among differential diagnoses during evaluation and workup and accordingly further complete evaluation should be considered. Management must be planned according to disease extent on individual basis for symptomatic treatment, improving quality of life and for optimum survival.

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