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Case Report

A rare case of cranial metastasis from an initial presentation of hepatocellular carcinoma

Chetan Kalal*, Adinath Wagh, Atif Patel, Harshad Joshi, Ravindra Sururde, Samit Jain, Shobna Bhatia, Chetan Bhatt

Department of Gastroenterology, Sir H. N. Reliance Foundation Hospital and Research Centre, Mumbai, Maharashtra, India

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***Correspondence:**
Dr. Chetan Kalal,
E-mail: dr.chetankalal@gmail.com

ABSTRACT

Cranial metastases from hepatocellular carcinoma (HCC) has been seldom reported. Reported herein is the case of a painless parietal bone mass as an initial presentation of HCC in a 63-year-old female patient who was subsequently diagnosed to have HCV related cirrhosis. The biopsy from cranial lesion was confirmatory of HCC on immunohistochemistry. The patient had no known history of chronic liver disease. The presented diagnosis was made through detailed history, laboratory parameters and cross sectional imaging.

Keywords: Chronic liver disease, Hepatocellular carcinoma, Skull metastasis

INTRODUCTION

Hepatocellular Carcinoma is the most frequent primary malignant tumour of the liver and the fourth leading cause of cancer-related deaths.[1] Extra-hepatic spread of patients with HCC at the time of diagnosis is estimated to be 5%-15%.[2] Lung and regional lymph nodes are the commonest sites and primary presentation with skeletal metastases is rare. Only a few case reports have been documented till date. In the majority of reported cases, cranial metastasis occurs late in the course of HCC, with most being symptomatic.

CASE REPORT

Authors would like to present our experience of management of a 63-year-old female, a teetotaler without known co-morbidities, who presented with a painless right parietal scalp lump of one-month duration. The patient had no history of trauma and no associated headache or vomiting. There was no family history of malignancy. Findings upon Examination revealed a non-tender, hemispherical, subcutaneous lump over the right parietal region approximately 3 x 3 cm in diameter. The lump was firm in consistency, non-pulsatile without any cough impulse or local signs of inflammation. Her general and systemic examination was otherwise normal with no focal neurological signs. Her routine blood biochemistry was unremarkable. Her skull x-ray showed a lytic lesion of the parietal bone.

The multiphasic MRI scan of the brain showed solitary expansile osteolytic lesion with Exo and intracranial extradural soft tissue in parasagittal right high parietal bone with defect measuring 3.8 cm (Figure 1). PET scan did not show lymph nodal or bony metabolic activity except cranial and liver lesions. Core biopsy of the lesion revealed skin with dermis showing metastatic deposits of a neoplasm composed of cells in nests and sheets (Figure 2). The tumor was moderately to poorly differentiate infiltrate to bone (Figure 3). Subsequent immunohistochemistry demonstrated positivity for Hepar 1 (Figure 4), PanCK, TTF-1, and negativity for CK-7, CK-20, Glypican -3 and AFP. Subsequent contrast

enhanced MRI scan of the abdomen showed multiple nodules in both the hepatic lobes, showing classical arterial hyper enhancement with venous wash out (LIRADS 5 lesion) (Figure 5).

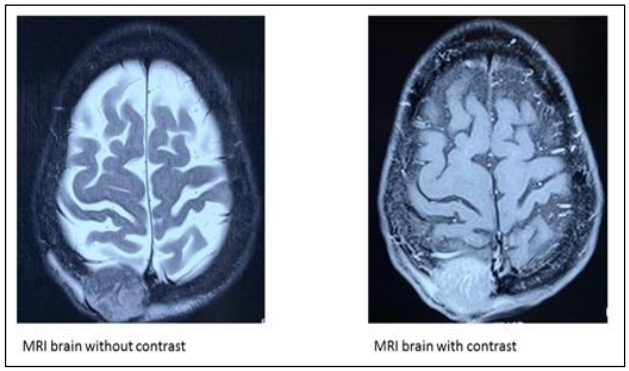

Figure 1: MRI scan of the brain solitary osteolytic lesion with exo and intracranial extradural soft tissue in parasagittal right high parietal bone with defect measuring 3.8 cm (Left panel) with post-contrast hyper-enhancement (Right panel).

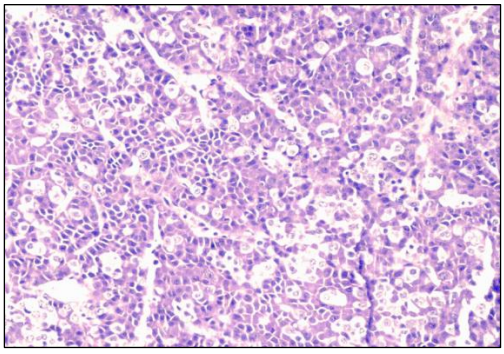

Figure 2: Histologic examination of skull lesion tissue samples. hematoxylin and eosin (H&E) staining, original magnification X 100, moderate to poorly differentiated metastatic deposits of a neoplasm composed of cells in nests and sheets.

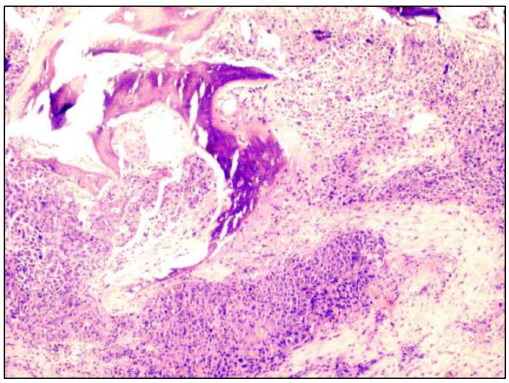

Figure 3: Histologic examination of skull lesion tissue samples. hematoxylin and eosin (H&E) staining, original magnification X 40, moderately to poorly differentiated neoplasm invading bone tissue.

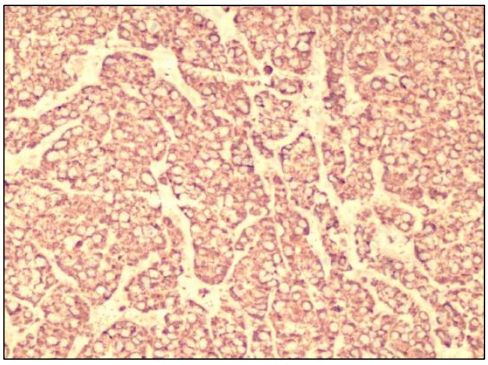

Figure 4: Immunohistochemistry (IHC) of skull lesion demonstrating hepar 1 positivity.

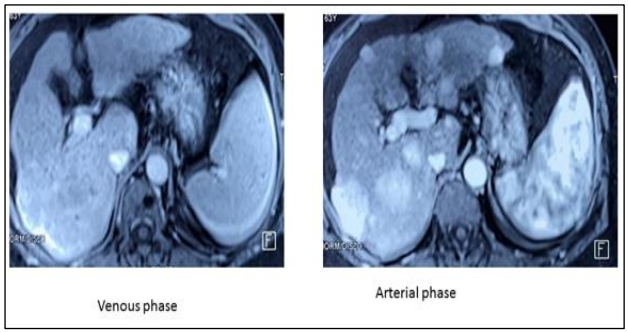

Figure 5: MRI scan of the abdomen showed multiple nodules in both the hepatic lobes classical arterial hyper enhancement (right panel) with venous wash out (Left panel).

The Largest lesion was 4.7 x 3.4 cm diameter in the right lobe posterior segment VI and VII with features of angio-invasion in the form of infiltration of right hepatic vein, hepatic and supra-hepatic IVC and segment VI branch of the post division of right portal vein with underlying cirrhosis and portal hypertension (Figure 5). HCV (Genotype-3, serum HCV RNA level 190,000IU/ml) was the etiologic factor for liver cirrhosis; Anti-hepatitis B core antibody was negative. The alpha-fetoprotein level was 4.3 IU/ ml, MELD of 12, with Child Pough Turcot score of 8. Patient was given trial of Sorafenib along with directly acting antiviral agents (sofusbuvir and velpatasvir), which she could not tolerate and ultimately succumbed to the illness.

DISCUSSION

HCC is the most common primary liver tumour and is the fourth most common cause of death due to cancer worldwide.[3]

HCC commonly metastasizes to lung, lymph nodes, bone, and adrenal glands via hematogenous and lymphatic routes. HCC very rarely metastasizes to the cranium as compared to other bony sites with incidence of 0.4-1.6%, which generally occurs in advanced stage and is located in the vertebrae, pelvis, ribs.[4-5] There is very scanty data

on asymptomatic cranial lesion as an initial presentation of HCC. Few reports suggest that though uncommon, HCC should be considered in the differential diagnosis of patients presenting with bone metastases, and these can very rarely be the primary manifestation. Metastatic spread of HCC to the bones occurs in 13%-16% cases.[6]

Other metastatic tumors, notably from the breast, kidney, and adrenal glands, may mimic HCC; and immunohistochemistry (IHC) is useful, especially in those cases with initial presentation of HCC as a metastasis.

The metastasis from HCC in the scalp with bony invasion is usually hypervascular therefore doing a biopsy of skull lesion in the presence of significant portal hypertension can be catastrophic leading to torrential bleeding. To avoid such untoward complications close monitoring of the coagulation profile is of paramount importance and should be a routine before biopsy from any asymptomatic skull lesion. Also, all efforts should be made to rule out underlying chronic liver disease.

Treatment should be considered for Cranial Metastatic Lesions secondary to HCC, as it poses a risk of extradural or intratumoral hemorrhage or underlying brain compression leading to neurological consequences.

A single calvarial metastasis can be treated surgically or with radiofrequency ablation.[7] Patients treated with either radiotherapy or surgery or both were found to have longer median life survival.[8]

CONCLUSION

Reported above is rare case of extra-hepatic HCC, presenting initially as an asymptomatic cranial metastasis.

- Any clinician and Hepatologist should entertain HCC as one of the differential diagnosis in patients with scalp subcutaneous mass with osteolytic lesions.
- An early diagnosis may afford the best possibility for a curative excision and improved outcome for the patient.

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