A rare case of trigeminal neuralgia

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ABSTRACT

Trigeminal neuralgia also called Fothergill disease or Prosopalgia is a neuropathic disorder characterized by episodes of intense pain in the face originating from disorders of the trigeminal nerve. Here, we present an unusual case of trigeminal neuralgia due to metastasis from hepatocellular carcinoma in a 76-year old patient. Magnetic Resonance Imaging ofthe brain revealed a mass lesion in the skull base. With suspicion of the mass being metastatic, further imaging and investigations were done, which revealed the presence of hepatocellular carcinoma. This report highlights that metastatic mass lesion can be a cause of trigeminal neuralgia and that hepatocellular carcinoma can have an unusual presentation such as skull base metastasis.

Keywords: *Hepatocellular carcinoma, Metastasis, Skull base, Trigeminal neuralgia.*

Trigeminal neuralgia (TN) also called Fothergill disease or Prosopalgia is a neuropathic disorder characterized by episodes of intense pain in the face originating from disorders of the trigeminal nerve [1]. It is more prevalent in women ranging from 0.03% to 0.3% [2]. It is caused by a wide range of etiologies, based on which, the disease is classified into two types by International Headache Society (IHS). These include classical cases caused by vascular compression by superior cerebellar artery and symptomatic cases which occur due to causes other than vascular compression such as posterior fossa tumors like epidermoid, meningioma and schwannoma [3].

We encountered a case of trigeminal neuralgia that was found to be due to solitary skull metastasis from Hepatocellular carcinoma (HCC) and decided to report this case as a literature search revealed no similar case of trigeminal neuralgia reported so far.

CASE REPORT

A 76-year old man presented with sudden onset paresthesia and numbness on the left side of the face and left-sided headache for the last 15 days. His symptoms aggravated on chewing, swallowing,and brushing teeth. Other history included hypertension since the last 10-15 years that was controlled on medication. The patient was evaluated by the neurologist, his vitals were stable and general examination revealed no abnormality except decreased sensation across all three branches of the trigeminal nerve on the left side of the face. Hence based on the clinical features, he was diagnosed as having left-sided trigeminal neuralgia.

Magnetic resonance imaging (MRI) of the brain was advised to evaluate the cause of the same. MRI revealed a heterogeneously enhancing mass lesion causing bone destruction in the left petrous apex and the left side of clivus which was extending into the lefttrigeminal cistern (Fig. 1-3). No other lesions were seen in the brain parenchyma. Based on these findings, the possibility of the metastatic lesion was suggested. Our patient was evaluated further for any possible primary lesion. He was advised further investigations including Chest X-Ray and Ultrasonography (USG) of the abdomen. Chest X-Ray was reported as normal, whereas USG of the abdomen showed hepatomegaly with a large heterogeneous mass in the right lobe ofthe liver. Subsequently, the patient returned to the neurologist, who further advised a computed tomography (CT) scan of the abdomen. A triple-phase CT abdomen was done that showed a large mass lesion in segment 6 of liver

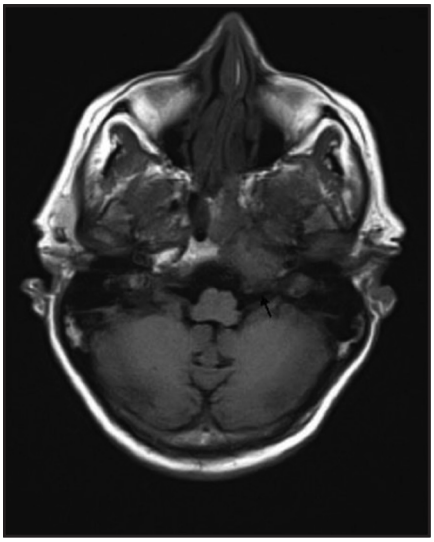

Figure 1: Axial T1W (T1-weighted) MR image shows a hypointense mass lesion causing destruction of left petrous apex and left side of clivus.

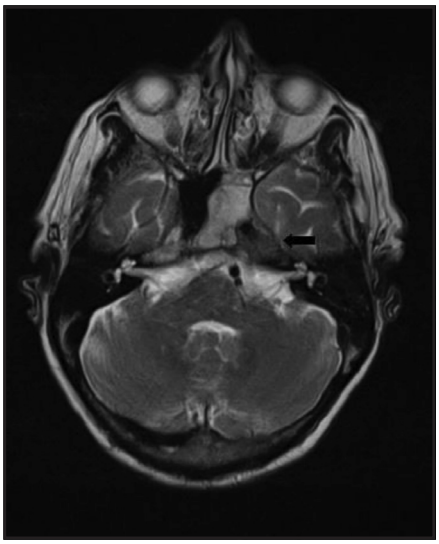

Figure 2: Axial T2W (T2-Weighted) MR image shows that the mass is extending into the trigeminal cistern.

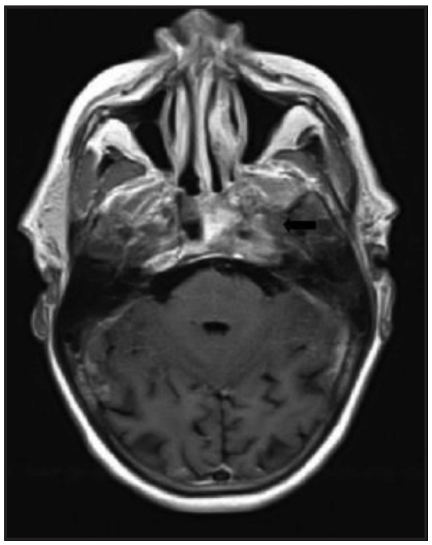

Figure 3: Axial post contrast T1W MR image shows heterogenous enhancement of the mass lesion.

measuring 10.1 x 9.2 cm. It showed heterogeneous enhancement in the arterial phase and rapid washout in the portal venous phase. These findings were suggestive of HCC (Fig. 4a and b).

USG guided fine needle aspiration cytology (FNAC) was done from the lesion which showed irregular sheets of pleomorphic hyperchromatic cells with moderate to abundant cytoplasm. Nucleoli were present in most of the nuclei and there were intra-nuclear cytoplasmic inclusions along with dissociated cells and atypical mitotic figures. These findings also suggested the diagnosis of HCC.

The patient was then referred to a higher institute for further management and regular follow-up was taken. A positron emission tomography (PET)-CECT scan of the whole body was done and two minimally hypermetabolic lesions in the liver and left petrous apex was noted. Thus, it was diagnosed as HCC with metastasis to the skull base and followed by radiotherapy and chemotherapy. The patient received Cyberknife-based hypofractionated stereotactic radiotherapy (HSRT) for skull base lesion followed by chemotherapy with sorafenib.

While on chemotherapy, the patient was gradually deteriorating and a repeat PET-CECT whole-body scan was done. This scan revealed multiple fluorodeoxyglucose (FDG) avid lytic lesions in the left clivus, multiple ribs, vertebrae, with few of them showing soft tissue component, suggestive of multiple skeletal metastases. Unfortunately after a few cycles of chemotherapy, the patient deteriorated further, developed severe gastro-intestinal bleeding and passed away. Hence, we decided to report the case, as a literature search revealed only a few cases of HCC with solitary skull base metastasis and none presenting as trigeminal neuralgia primarily.

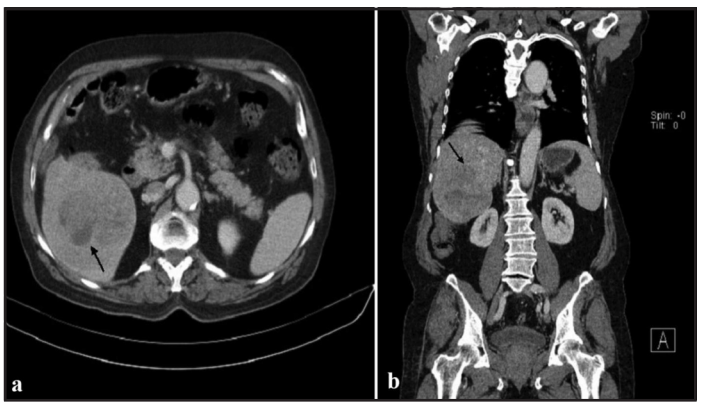

Figure 4: (a) Axial contrast enhanced CT image of the abdomen shows an irregular heterogeneously enhancing mass lesion in right lobe of liver; (b) Coronal contrast enhanced CT image of the abdomen shows the craniocaudal extent of hepatic mass.

DISCUSSION

Trigeminal neuralgia is known to be triggered by vascular compression of the trigeminal nerve root, most frequently bythe dilated superior cerebellar artery. There are other less frequent causes which include posterior fossa tumors, cerebral aneurysms, arteriovenous malformations, multiple sclerosis plaques, etc. Theincidence of posterior fossa tumor-induced TN ranges from 2.1-11.6% in the literature which mainly comprises of meningiomas (14-54%), epidermoid tumors (8-64%) and vestibular schwannomas (7-31%) [4,5].

Trigeminal neuralgia due to solitary metastasis to the skull base has rarely been reported in the literature. Metastasis from HCC usually occurs to regional lymph nodes and lungs. The incidence of HCC metastasizing to the bone is low, ranging from approximately 2% to 16%. Bone metastasis preferentially occurs in the axial skeleton includingvertebrae, pelvic bones, and ribs. Skull metastasis is rare and usually affects men in their sixties [6,7]. The incidence of skull metastasis from HCC ranges from 0.5% to 1.6% [7]. Spread of metastasis to the petrous apex is often caused by hematogenous spread from the distant tumor, direct spread from an intracranial tumor, or through leptomeningeal spread from the distant or intracranial tumor. Petrous apex is susceptible to hematogenous metastasis due to slow blood flow through the petrous apex marrow, which allows filtering and deposition of tumor cells [8]. With an improvement in diagnosis and treatment andan increase in survival of HCC,

the incidence of bone metastasis has also increased [5]. There is one similar case reported in the literature, where the patient with recurrent multiple HCC develops metastatic lesion in the skull base involving multiple cranial nerves along with extrahepatic metastasis in both lungs [9].

Our patient presented with classical symptoms of TN. MRI of the brain revealed a heterogeneously enhancing mass lesion causing bone destruction in the left petrous apex and the left side of clivus which was extending into the left trigeminal cistern and we suggested the possibility of a metastatic lesion. Other differential diagnoses which we considered due to the location and excluded were trigeminal schwannoma andmeningioma. Trigeminal schwannoma in this location appears as a lobulated heterogeneously enhancing mass lesion in the trigeminal cistern and cavernous sinus. This may extend posteriorly into the prepontine cistern if that part of the nerve sheath is also involved. Meningiomais seen as a well-defined dural based homogeneously enhancing mass lesion with a thin dural tail and associated hyperostosis of the petrous apex [8]. The findings in our case with the pattern of bone destruction were not consistent with either schwannoma or meningioma. Hence, a final diagnosis of the metastatic lesion at the skull base was made.

CONCLUSION

This case highlights the fact that HCC can present with a solitary metastatic lesion in the skull base and that TN may be the initial presentation of a metastatic skull lesion. We emphasize the need for meticulous imaging of such patients to search for the primary malignancy in order to save them from further morbidity and mortality associated with a delayed diagnosis.

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