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Case of Radiation Induced Microcrocystic Meningioma- A Case Report

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Abstract: Microcystic meningiomas is a distinct morphological variant of meningiomas, characterized by loose texture and microcysts with formation of large extracellular spaces containing edematous fluid. We reported a case of microcystic menigiomas in 37 year old male patient had underwent radiotherapy for treatment of NHL 22 Years back. The tumor showed diffuse immune histochemical reactivity for VIMENTIN, EMA, S-100 protein, ultrastructure study confirmed the menigiomatous nature of tumor. Radiation exposure would be one of the etiological factors along with genetic instability and patient required multimodality of treatment along with regular surveillance.
Keywords: Intracranial; Meningioma; microcystic.

INTRODUCTION

The meningiomas are tumors derived from arachnoid cells [1], and its origin linked to idiopathic genetic changes, predisposing diseases and radiotherapy induction.

Microcystic menigiomas have been described as distinct variant of meningiomas, they are characterized by the formation of numerous large extracellular microcystic spaces containing edematous fluid,and by the stellate and vacuolated appearances of tumor cell, with occasionally large hyperchromatoic and pleomorphic nuclei. Their unusual histopathogical appearances may lead to confusion with astrocytomas, hemangioblastomas and angioblastic meningiomas [2].

The meningiomas are tumors that may arise after cranial irradiation, used to the scalp or intracranial lesions.Only ionizing radiation has been identified as the etiologic agent related to the occurrence of these neoplasms [3, 4]. The role of head trauma, viral infections and sex hormones has not been established. The appearance of a lesion (new or progressive) a few years after the completion of radiation therapy for primary neoplasms may represent recurrence of the cancer, radiation necrosis or, rarely secondary neoplasms [5, 6] .

We report a case , 37 year old male patient who was initially treated with radiotherapy for NHL ,clinically we suspected gliomas or radition induced necrosis but after histopathogical and immunohistopathologically it was diagnosed as microcystic menigiomas .

CASE REPORT

A 37 year old male patient ,who was treated for NHL 22 years back with radiation and chemotherapy .He was presented with history of left sided headache and giddiness, no h/o seizure vomiting, LOC, no history suggestive of any focal neurological deficitand cranial nerve involvement. Patient had left lateral rectus palsy, other neurological examination were unremarkable. MRI T 2 W showed, mild homogenously enhancing lesion involving left cavernous sinus. There is thickening and enhancement of adjacent dura mater, Lesion showed diffusion restriction, And t2 mixed signal intensity lesion involving left ant temporal lobe, the anterior component shows multiloculated t2 hyper intense areas. Posterior cystic component of the lesion shows fluid levels. Post contrast sequence shows heterogeneous enhancement of the lesion with diagnostic possibility of radiation induced tumor, glioma, radiation necrosis .ON these finding patient was operated for left temporal craniotomy and decompression of tumor done under G.A. with operative finding showing left temporal lobe tumor having cystic portion, After decompression the yellowish coloured fluid let-out and tumor has a clear plane to temporal lobe .Near total excision done and tumor was firm to soft, very vascular and suckable in areas. The medial portion of tumor left behind and cauterized as very vascular in nature and encasing the vital structure cavernous sinus. Histopathological report showed predominantly meningothelial tissue with microcystic areas with immunohistopathologically positive for EMA,VIMENTIN; negative for CK,LCA, CD 34 AND MIB; feature confirm meningothelial

nature of tissue s/o of microcystic mengiomas post operative period was uneventful patient referred for radiotherapy.

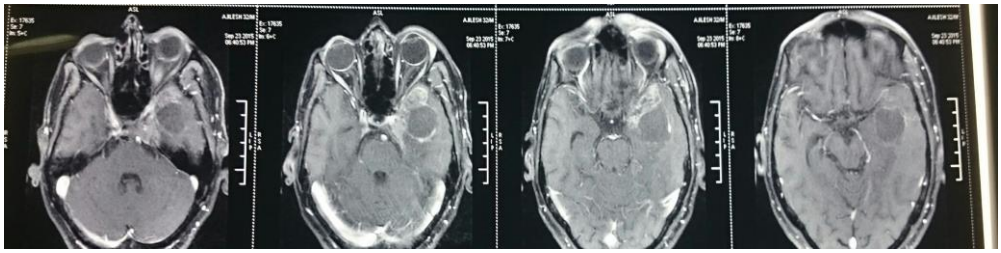

Fig-1: CT brain consract image showing

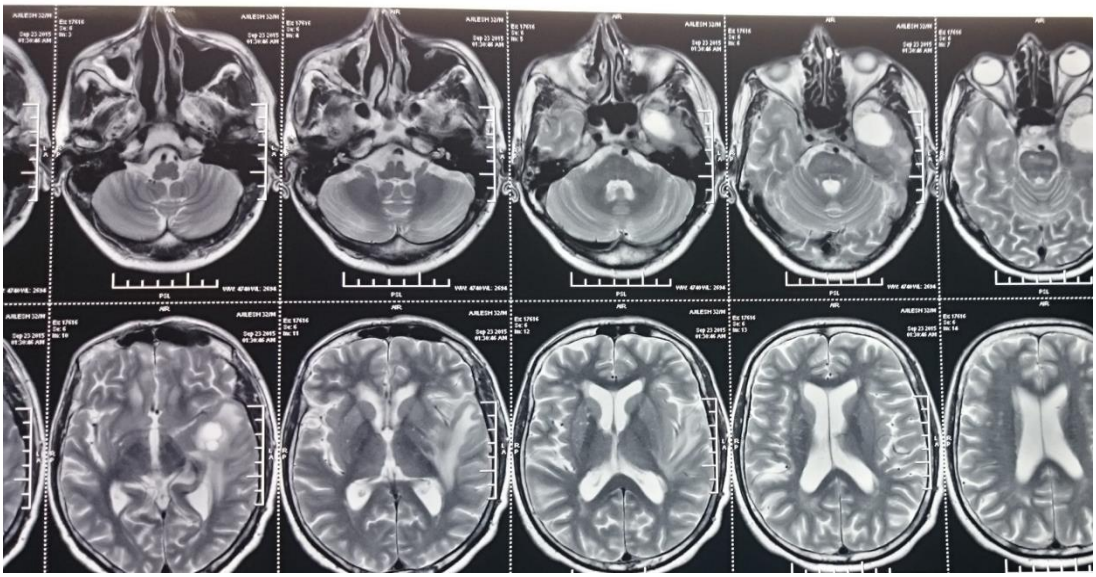

Fig-2: MRI T2 W image showing left temporal tumor

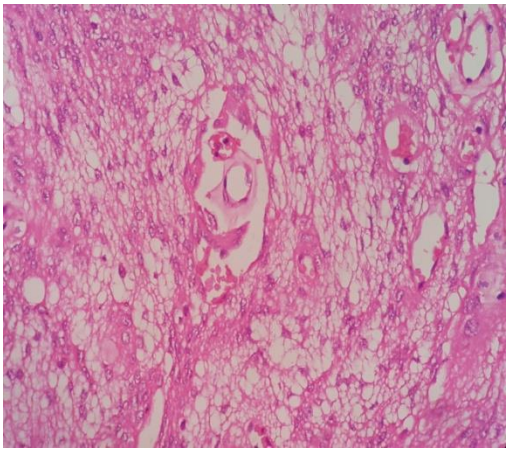

Fig-3: H &E staining showing microcystic space

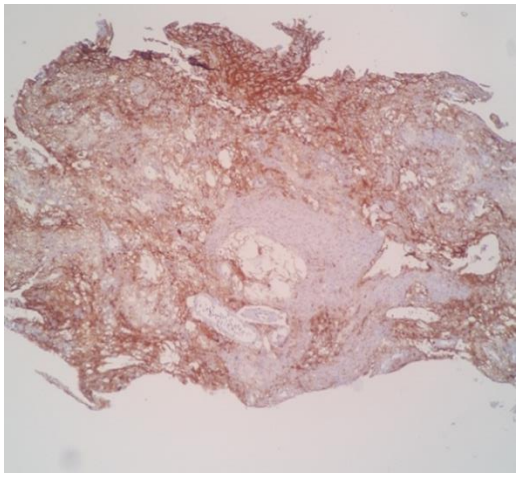

Fig-4: Ema Positive

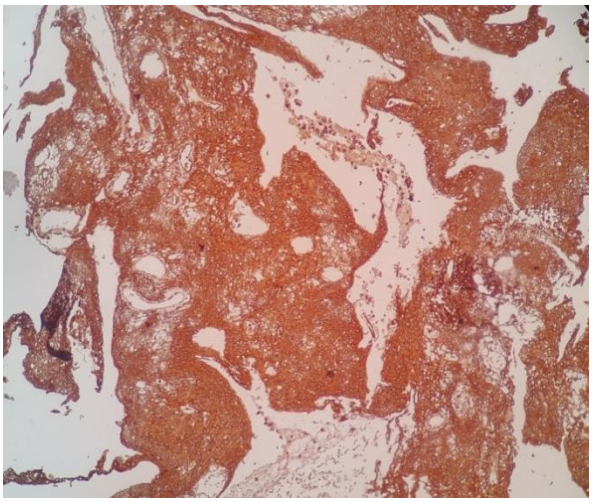

Fig-5: Vimentin Positive

DISCUSSION

According to the Finnish Cancer Registry, the overall meningioma incidence rates were 1.6for men and 5.5 for women per 100,000, and only 17%of meningiomas occurred in patients younger than 50 years of age [7]. Microcystic meningiomas have recently been recognized as an unusual morphogical variant of mengiomas, they have been classified as a distinct subgroup of mengiomas in the WHO classification of central nervous system tumor [8].

Grossly ,microcystic meningiomas are well delimited,with a smooth bosselated or lobulated external surface. The structure is usually soft and spongy .the cut surface discloses a yellowish light brown homogeneous and a glistening appearance. Hemorrhage and necrosis are never found [9].

Microscopically, these tumor shows numerous cystic spaces filled with the edematous fluid and are lined by stellate –shape meningothelial cells.some areas show the conglomerations of smaller cystic spaces which create a vacuolated, myxoid and loosely reticular appearances [8]. Foci of nuclear pleomorphism are occasionally noted,but this finding does not indicate aggressive behavior [9]. The tumor shoes occasionally typical meningiomous whorl [8]. Meningiomas demonstrated concurrent mesenchymal and epithelial differentiation as shown by their consisten and reliable immunonohistochemical positivity for EMA, VIMENTIN [10]. S -100 protein can be found focally in many meningiomas [11]. immunohistochemistry of microcystic meningiomas is not different from other type of meningiomas, tumor cell are positive for EMA and VIMENTIN and negative for cytokeratin, S-100, GFAP. Our case showed same IHC result [8].

The pathogenesis of these tumor is unclear but radiation exposure may be the causative factor. It is well known that radiation treatment induces genomic instability and manifests itself in induction of chromosomal aberrations, aneuploidy, gene mutations and amplifications, microsatellite instability, and cell death [12, 13]. Further, prominent hypomethylation during X-ray exposure, subsequent genome instability, and resultant recruitment of repair machinery

contributing significantly to carcinogenesis have been reported in different tissues [14, 15]. observation suggests that radiation treatment induces changes in the methylation patterns in an oxidative damage–independent fashion among meningioma cells. DNA methylation was found to be one of the determinants of γ radiation–induced gene expression [16] and ultra violet B (UVB) radiation induced DNMT activity to silence tumor suppressor genes, thereby supporting tumor growth [17], which perhaps suggests a complex network of epigenetic events that can be initiated by ionizing radiation.

With the increasing number of survivors of leukemia and the lymphomas and increasingly long follow-up periods, the old assumption of the rarity of secondary brain tumors should now be reconsidered. The accumulation of meningiomas among patients treated in the early era of leukemia and lymphomas treatment could imply an increasing incidence in the future [18].

The assumption of irradiation being the most important risk factor for almost all secondary malignancies has been widely accepted. Unlike meningiomas, which are characterized by a long latency period, gliomas tend to occur within 5 years after the treatment [19].

Although there are strongly predisposing factors such as radiation therapy, the significance of individual characteristics and genetic factors should not be ignored. The fact that meningiomas and the other Secondary malignant neoplasms developed in the same patients suggests a genetic susceptibility of these patients to developing cancer [20].

The importance of recognizing this lesion lies in the differential diagnosis from other central nervous system tumors with a myxomatous appearance [2]. Myxomatous schwannoma can be distinguished by positive staining schwannomas can be distinguish by posive staining for S-100 protein and negativity for EMA. Loose fibrillary cytoplasms simulate pilocystic astrocytomas, which can be distinguished by immunostaining with anti-Gfap Chordomas Would Shows Positive Staining For Cytokeratin And s-100 protien careful distinction should be given to metastatic carcinomas in view of nuclear pleomorphism. the absence of mitotic figure occasional menigotheial whorls are important feature distinguishing from a metastatic carcinoma, hemangioblastomas shows focal positive for S-100 protein and GFAP, but negative for EMA. Abundant blood vessels with marked hyalinization mimick angiomatous or angioblastic meningiomas, which show positive staining for vimentin but negative for EMA [9, 10].

These patients can be manage by complete surgical excision of tumor with regular follow-up to watch for the recurrence of tumor. Fractional radiotherapy and radiosurgery are used after surgery for incomplete resection of meningiomas.

CONCLUSION

Microcystic meningiomas are an unusual, but distinct morphological variant of meningiomas which are clinically and immunohistochemically similar to conventional meningiomas.

They have to be distinguished from other lesion of similar histology, radiation exposure would be the one of the etiological factor along with genetic instability. Patient may require multimodality of treatment along with regular surveillance.

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