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RESEARCH ARTICLE

GIANT CELL GLIOBLASTOMA MULTIFORME: A CASE REPORT

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ABSTRACT

Giant cell glioblastoma is an infrequent, accounting for about 5% of all glioblastoma mean age of presentation is about 40 years old. This tumor characterized by a predominance of bizarre multinucleated giant cells. It has deserved a separate category in the World Health Organization classification of grade IVtumor

INTRODUCTION

Giant Cell glioblastoma (GC) is an uncommon accounting for about 5% of glioblastoma multiforme (GBM) (Kevin *et al.,* 2009). It appear to be a distinct entity on the basis clinicopathologic and genetic data (Christopher and Fletcher, 2013). The tumor become clinically apparent after a short clinical histology, as in primary glioblastoma, but in younger patient. Mean age of presentation in about 40 years old with larger age range (including children) than conventional GBM. Survival time in this tumor group frequently exceeds the median survival time reported for conventional glioblastoma (Christopher and Fletcher 2013; Russell and Rubinstein, 1989; Margetts and Kalyan-Raman, 1989). Often these patient present with seizures, headaches and focal neurologic defect. The tumor typically are well circumscribed and neuroimaging demonstrates a heterogeneous enhancement. Despite typical radiographic and macroscopic demarcation, the tumor usally infiltrate the adjacent brain and leptomeninges. Zonal necrosis is commonly abundant and often produces large cystic areas (Akslen *et al.,* 1989; Can *et al.,* 2002). These tumor predominates in the cerebral hemispheres mainly subcortically in temporal andparietal lobes (Ohgaki *et al.,* 2013). Other possible primary location include the cerebellum (Demir *et al.,* 2005), the lateral ventricles (Alvarez-Betancourt *et al.,* 2004), the optic chiasm (Burnstine *et al.,* 1993) and the spinal cord (Grisold *et al.,* 1981). The histological features are dominated by large, bizarre, multinucleated giant cells with abundant eosinophilic cytoplasm and large vesicular nuclei. Other feature are the paucity of microvascular or endothelial hyperplasia, increased reticulin fibers and atypical mitosis. Necrosis is present mostly in pseudo-palisading form. Giant tumor cells may show lipid accumulation (Ibayashi *et al.,* 1990; Kroh *et al.,* 2004; Queiroz *et al.,* 2005). Immunohistochemistry studies show staining of tumor cells for GFAP (in some but not all tumor giant cells), vimentin, S100, EGFR, alpha anti-chymotrypsin (Katoh *et al.,* 1995; Kawano *et al.,* 1995). Giant cell glioblastoma appear to have a distinctive" hybrid" molecular genetic profile intermediate between primary and secondary glioblastoma in that they usually (Kevin *et al.,* 2009) do not have deletion of the CDK (4/6) inhibitors (CDK N2a gene) (Christopher and Fletcher, 2008) lake amplification of the EGFR or CDK4 genes (Russell and Rubinstein, 1989) have a relatively high frequency(≤30%) of PTEN mutations and (Margetts and Kalyan-Raman, 1989) demonstrate a high frequency (75-90%) of TP53 mutation (Christopher and Fletcher, 2013; Meyer-Puttlitz *et al.,* 1997; Peraud *et al.,* 1999).

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Case presentation

A previously healthy 64 year-old woman presented for evaluation of headaches, intermittent memory loss and mild nausea. Her headaches had begun approximately 2 months before her presentation. She underwent MRI (Fig. 1) with finding of a large, tempo parietal mass with dwelldefine outline. The histopathology showed a highly cellular neoplasm with marked pleomorphism, prominent giant cells and numerous atypical mitotic figures as well as zones of coagulative necrosis lined by palisading tumor cells (palisading necrosis) (Fig. 2,3). The immunohistochemistry was positive for the glial fibrillary acidic protein (GFAP) and P53, negative for cytokeratin (Fig. 4,5).

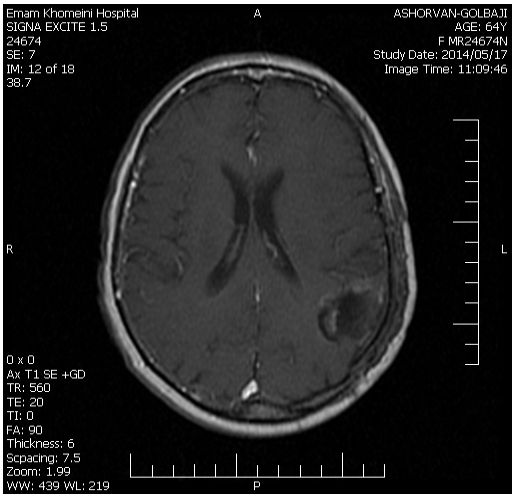


Fig.1.MR image demonstrating tempoparietal mass with welldefined **outline**

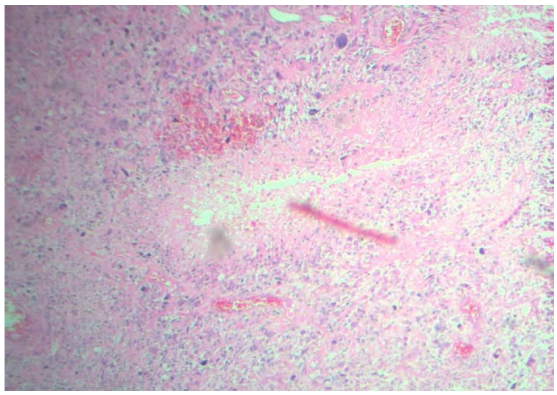


Figure 2. Histological sections of GBM showing coagulative necrosis lined by palisading tumor cells (palisadingnecrosis)

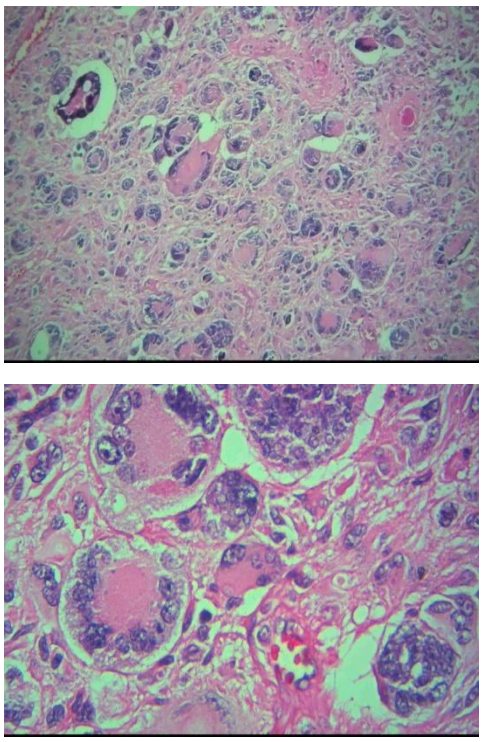


Fig. 3. photomicrograph of the tumor showing multinucleated giant cells, hematoxylin& Eosin a(100x) and b(400x)

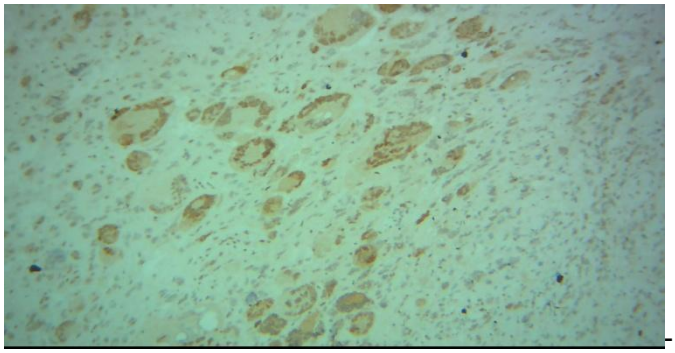
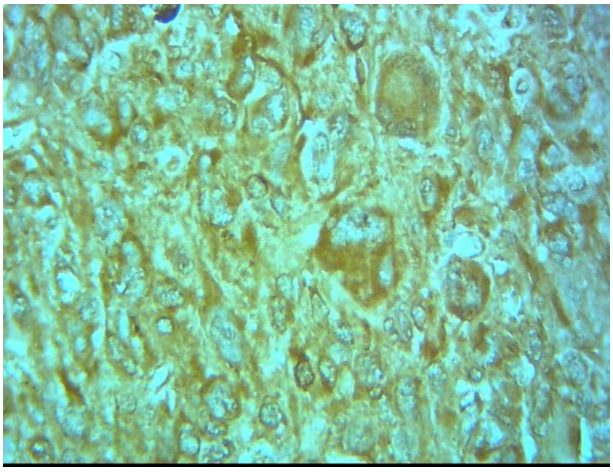


Fig.4. Photomicrograph of GBM immunostained for P53 (100x). Tumor cells show that stained positive for p53



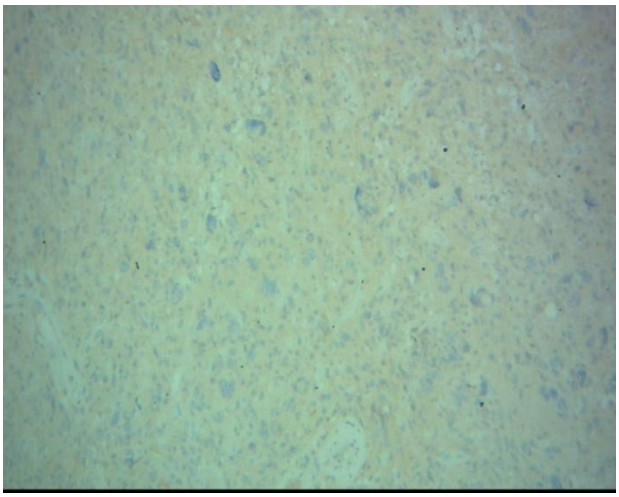


Fig. 5. Photomicrograph of GBM immunostained for GFAP and CK. Tumor cells show that stained positive for GFAP(a); negative for cK(b)

DISCUSSION

An important differential histological diagnosis of giant cell glioblastoma is pleomorphic xanthoastrocytoma (PXA) (Martínez-Díaz *et al.,* 2003) Quicker evolution of seizures, numerous great sized giant cells, numerous mitosis, atypical mitoses and pseudo-palisading necrosis will fave giant cell glioblastoma (De Prada *et al.,* 2006). GFAP is positive in both tumor. Positivity for P53 and negativity for synaptophysin and

neurofilamant protein is seen in giant cell glioblastoma but not in PXA (Martínez-Díaz *et al.,* 2003). The other differential diagnosis is metastatic carcinoma. Although infiltrating nature and cytologic feature of Giant cell glioblastoma aid in the distinction from metastatic carcinoma but sometimes the distinction requires immunohistochemical analysis for epithelial markers (Kriho *et al.,* 1997) (Fig.5). The median survival among all GC patient was 11 month, compared with 8 month for GBM. Younger age of GC patient and distinctive molecular genetic profile may be favorably affects survival compared with GBM patient (Martinez-Diaz *et al.,* 2003).

The treatment of malignant glioma is still a challenge, particularly in children. Present day treatment includes tumor resection, local radiotherapy and chemotherapy, which are approaches that promote an improvement in the length of survival but do not seem to change the inexorable course of the disease (Reddy and Wellons, 2003; Hess, 1999; Prados, 2000).

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