**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 IV tumor.

**Key words:**

Giant cell glioblastoma.

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**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 usually 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 cerebellar hemispheres mainly subcortically in temporal and parietal 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).

**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 dwelledefined 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).

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**DISCUSSION**

An important differential histological diagnosis of giant cell glioblastoma is pleomorphic xanthoastrocytoma (PXA) (Martinez-Diaz 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
neurofilament protein is seen in giant cell glioblastoma but not in PXA (Martinez-Diaz 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).

REFERENCES


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