



## OLIGODENDROGLIOMA PRESENTED AS HEMIPARESIS.

## Pathology

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## ABSTRACT

A 53 years old male patient presented with sudden onset of right sided weakness, followed by unconsciousness. On clinical examination showed right sided hemiparesis. On MRI showed space occupying lesion reported as malignant central nervous system lesion, features consisting glioblastoma multiforme. On histopathological study diagnosed as oligodendroglioma grade II. We are presenting this case for its clinical behaviour, radiological and histopathological findings.

## KEYWORDS

Glial tumour, Brain tumour, Histopathology , Oligodendroglioma.

## Introduction-

Oligodendroglioma are rare tumours of CNS accounts for approximately 2.5% of all primary brain tumours and 5-18% of all gliomas [1,2]. The oligodendrogliomas are classified as low grade glial tumour, usually grade II as per WHO classification of glial tumours [2]. These low grade tumours are often mistaken as benign neoplasm, however these can transform to anaplastic type and gives neurological morbidity and mortality.

## CASE REPORT

A 53 years old male patient presented with history of right sided weakness since 2 days, followed by disorientation, unconsciousness and unresponsiveness to motor and verbal responses. Patient was in vegetative state with normal breathing. He recovered from this phase but right sided weakness and stiffness was present in upper and lower extremities. There was no history of trauma, seizures, vomiting, any drug treatment. Patient was non alcoholic, non diabetic and non hypertensive. No history of any major systemic illness in past. MRI Brain (Plain +contrast) showed an ill defined large heterointense solid and cystic mass lesion, involving temporal lobe and parietal lobe on left side which was predominantly hyperintense on T1WI/heterointense on T2 showing multiple cystic areas on T2WI/FLAIR and the cysts show hemosiderin levels within them. Out of proportion perilesional oedema is present. The lesion is predominantly haemorrhagic. There are no areas of calcification. The solid portion of the lesion shows diffuse restriction. Midline shift of 15 mm to the right side. Left lateral ventricle is effaced. Transtentorial herniation is present, subfalcine herniation is also noted.

On post contrast images- Solid portion showing heterogenous post contrast enhancement. Neovascularity, infiltrative borders, dural involvement is present which is 6.8 x 9.5 x 5 cm. Imaging features are consistent with a high grade malignancy likely to be glioblastoma multiforme. He underwent left fronto parietal craniotomy and biopsy of frontal space occupying lesion. On histopathological examination showed a glial tissue and tumour. The tumour is composed of neoplastic cells arranged in diffuse pattern (Figure 1). The individual cells are round having round, enlarged, hyperchromatic nuclei and clear cytoplasm giving "fried egg" appearance (Figure 2). The tumour showed rich vascularity with chicken wire appearance. In areas calcification was noted (Figure 3). No areas of necrosis, haemorrhage was noted. The tumour was diffusely infiltrating the surrounding cerebral cortex. There was no ganglionic differentiation. On microscopic examination diagnosed as oligodendroglioma low grade II as per WHO classification of glial tumour.

## Discussion-

The oligodendroglioma is the third most common glioma overall. It accounts for 2-5% of primary brain tumour [2]. The patient clinically presents usually with seizures. Other presentation like headache, vomiting, hemiparesis, vertigo, visual disturbances,

weakness, ataxia are noted [3,4]. The oligodendroglioma typically have long clinical presentation, usually of 5 years or more. The tumour of intraventricular site or anaplastic variant may clinically presents earlier. In our case it was presented in shorter duration. It was with right sided weakness followed by hemiparesis. Seizure is the most common symptom consisting to 35-85% of cases [5,6], which is related to tumour invasion in cortical area mostly. In AFIP series most common clinical sign reported was paralysis (50%), visual loss (49%) and abnormal reflexes (37%) [7].

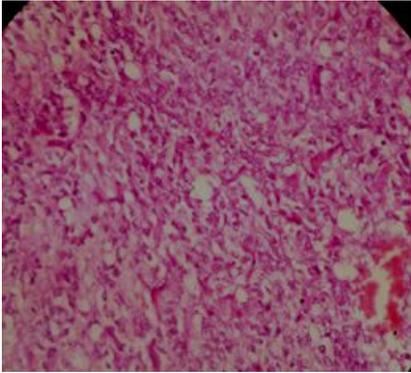
Our patient presented at age of 53 years. The peak age for oligodendroglioma is in fourth and fifth decade, with male predilection (Male : Female ratio of 2:1). The oligodendroglioma in majority case reported in the supra tentorial brain with frontal lobe is the common site followed by temporal, parietal and occipital lobe. Rarely it is seen in brainstem, spinal cord or leptomeningial location.

On radiological finding cortical or subcortical well demonstrated mass lesion was seen. The peritumoral oedema, cyst, calcification are noted [8]. Our case showed all these features with areas of haemorrhage. However rarely multiple oligodendroglioma were reported.

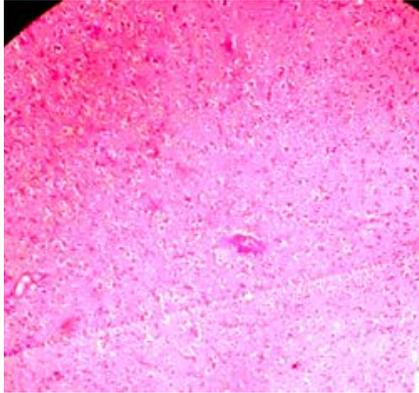
Grossly oligodendrogliomas are usually soft, fleshy, grayish-pink masses. Areas of mucoid calcification, cystic change are noted [4]. Histopathological findings are confirmatory for oligodendroglioma which shows diffusely infiltrating monotonous uniform round cells with vesicular nuclei, small distinct nuclei and perinuclear halo giving honeycomb or "fried egg appearance" and arborising capillaries. These are usually Grade II tumours. Tumours are classified as well differentiated oligodendroglioma and less commonly as anaplastic oligodendroglioma. Rarely mixed form as oligoastrocytoma may be seen [4]. Among glial tumours oligodendroglioma shows microcalcification more commonly (90% [9]). In our case all typical microscopic features with areas of calcification were noted. There is no specific immunohistochemical marker. They show positive stain for OLIG2, Leu7, S100 and MAP 2. Molecular study for loss of heterozygosity on chromosome 19 (19q) and chromosome 1 (1p) is observed in 50-80% of oligodendroglioma [4]. These patients have favourable response to chemotherapy and longer duration of survival. Patients are treated with gross surgical resection, partial resection combined with radiation therapy or chemotherapy. The median postoperative survival of patient ranges from 3 to 17 years [5,10]. Our patient received treatment of gross surgical resection of lesion and chemotherapy. And on follow up showed well response to treatment.

## CONCLUSION

Oligodendroglioma is a rare brain tumour, however with various diagnostic methods histopathology, neuroimaging and molecular genotyping study early detection is possible. With proper modality of treatment favourable outcome is noted.

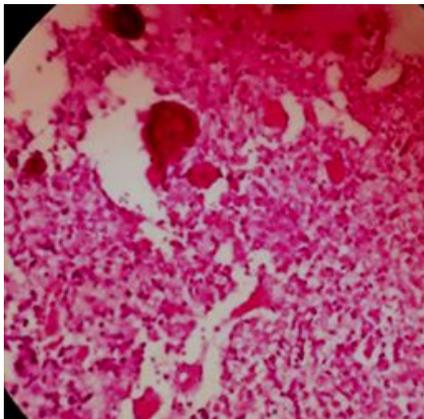


**Fig.1 showing oligodendroglioma arranged in diffuse pattern (Haematoxylin and Eosin stain,x40).**



**Fig.2 showing oligodendroglioma having tumour cells fried egg appearance.**

**(Haematoxylin and Eosin stain,x10)**



**Fig.3 showing rich vascularity of tumour with areas of calcification.**

**(Haematoxylin and Eosin stain,x40).**

#### REFERENCES

- Engelhard HH, Stelea A, Mundt A. Oligodendroglioma and anaplastic oligodendroglioma: clinical features, treatment, and prognosis. *Surg Neurol*, 2003;60(5):443-456.
- Louis DN, Ohgaki H, Weistler OD, Cavenee WK, Burger PC, Jouvet A, et al. The 2007 WHO classification of tumours of central nervous system. *Acta Neuropathologica* 2007;114:97-109.
- Roldan G, Scott J, George D, Parney I, Easaw J, Cairncross G. Leptomeningeal disease from oligodendroglioma: clinical and molecular analysis. *Can J Neurol Sci*. 2008 May; 35(2):204-9.
- Reifenberger G, Kros JM, Burger PC, Louis DN, Collins VP. Oligodendroglioma. In: Kleihues P, Cavenee WK eds. *Pathology and genetics of tumours of nervous system*. Lyon, France: IARC Press, 2000;56-61.
- Olson JD, Riedel E, DeAngelis LM. Long term outcome of low grade oligodendroglioma and mixed glioma. *Neurology* 2000;54(7):1442-1448.
- Margain D, Peretti-Viton P, Prerez-Castillo AM, Martini P, Salamon G. Oligodendrogliomas. *J Neuroradiol* 1991;18:153-160.
- Ludwig CL, Smith MT, Godfrey AD, Armburstmacher VW. A clinicopathological study of 323 patients with oligodendroglioma. *Ann Neurol* 1986;19:15-21.

- Lee YY, Van Tassel P. Intracranial oligodendroglioma: imaging findings in 35 treated cases. *AJR Am J Roentgenol* 1989;152:361-369.
- Celli P, Nofrone I, Palma L, Cantore G, Fortuna A. Cerebral Oligodendroglioma: prognostic factor and life history. *Neurosurgery* 1994;35(6):1018-1034.
- Roldan G, Scott J, George D, Parney I, Easaw J, Cairncross G. Leptomeningeal disease from oligodendroglioma: clinical and molecular analysis. *Can J Neurol Sci*. 2008 May; 35(2):204-9.