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Behzad Saberi *

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A Brief Review on Some Important Notes in The Genetics of Malignant Gliomas

Behzad Saberi

¹ Medical Research, Esfahan, Iran

*Correspondence Author: Behzad Saberi, Medical Research, Esfahan, Iran

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Abstract

Malignant gliomas which are also known as high-grade gliomas are among the brain tumors which are progressive in a rapid manner in their nature. Glioblastoma, anaplastic astrocytoma, anaplastic oligodendroglioma and mixed anaplastic oligoastrocytoma are among these malignant tumors.

There are some genes and relevant alterations which are involved in the development of malignant gliomas. This brief review tries to point to some important notes related to the malignant glioma's genetics.

Keywords: brain tumors; gliomas; malignancy; genetics; important notes

Summary

Brain tumors are among the common CNS pathologies and include benign and malignant lesions. High-grade gliomas are among the malignant brain tumors which would develop by various factors including alterations in the genes. Continuous accumulation of genes alterations in the low-grade gliomas would result in the development and occurrence of malignant or high-grade gliomas [1,2].

Glioblastomas which are aggressive tumors would be divided into two groups, one group with isocitrate dehydrogenase one gene mutation and another group with isocitrate dehydrogenase one or two gene expression. Alpha-ketoglutarate or AKG which would be produced from the mutation of the isocitrate dehydrogenase will cause some changes in the malignant cells including metabolic and epigenetic ones like abnormal methylation of the histone.

Mutation in the isocitrate dehydrogenase one is of importance in the biology of the malignant gliomas specifically the wild-type class of the isocitrate dehydrogenase one [3,4,5].

In such isocitrate dehydrogenase one wild-type class of the glioblastomas, some parts of the tenth chromosome which contain the phosphatase and tensin homolog gene as one which would suppress the glioma tumor and platelet-derived growth factor A gene reinforcement can be seen.

Other changes which may happen afterwards can be cyclin-dependent kinase inhibitor 2A gene cell cycle regulator loss, tumor protein p53 and neurofibromin 1 tumor suppressor genes mutations and c-MET and epidermal growth factor receptors activity in a mutant nature.

All of these changes finally result in developing malignant behavior of the tumors and would cause transforming into the malignant tumors [6,7].

Conclusion:

It is important for the researchers who are studying on brain tumors and relevant pathologies to have knowledge about the genetic alterations which result in the formation and development of the malignant gliomas.

Having this knowledge is of importance to understand the pathology and behavior of these tumors and can hopefully result in finding novel treatment options for such tumors.

References:

- 1. Wood M.D, Halfpenny A.M., Moore S.R. (2019). Applications of molecular neuro-oncology-a review of diffuse glioma integrated diagnosis and emerging molecular entities. Diagn. Pathol, 14:29.
- 2. Riemenschneider M.J, Jeuken J.W, Wesseling P, Reifenberger G. (2010). Molecular diagnostics of gliomas: State of the art. Acta Neuropathol, 120:567-584.
- 3. Phillips H.S., Kharbanda S, Chen R., Forrest W.F, Soriano R.H, Wu T.D, Misra A, Nigro J.M, Colman H., Soroceanu L, et al. (2006). Molecular subclasses of high-grade glioma predict prognosis, delineate a pattern of disease progression, and resemble stages in neurogenesis. Cancer Cell, 9:157-173.
- 4. Vikiforova M.N, Hamilton R.L. (2011). Molecular diagnostics of gliomas. Arch. Pathol. Lab. Med, 135:558-568.
- 5. Guo C, Pirozzi C. J, Lopez G.Y. (2011). Yan H. Isocitrate dehydrogenase mutations in gliomas: Mechanisms, biomarkers and therapeutic target. Curr. Opin. Neurol, 24:648-652.
- 6. Verhaak R.G, Hoadley K.A, Purdom E, Wang V, Qi Y, Wilkerson M.D, Miller C.R, Ding L., Golub T, Mesirov J.P, et al (2010). Integrated genomic analysis identifies clinically relevant subtypes of glioblastoma characterized by abnormalities in PDGFRA, IDH1, EGFR, and NF1.Cancer Cell, 17:98-110.
- 7. Cancer Genome Atlas Research Network Comprehensive genomic characterization defines human glioblastoma genes and core pathways. Nature, (2008) 455:1061-1068.

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