OPEN ACCESS Freely available online

ISSN: 2168-975X
Brain Disorders & Therapy

Short Communication

Biomarkers for Diagnosis, Prognosis, Prediction of Brain Cancer using Molecular Pathways

Sueli Mieko*

Department of Neurology, University of Sao Paulo, Paulo, Brazil

DESCRIPTION

Adults having glioblastoma have the most prevalent recurrent brain cancer. Malignant astrocytomas, such as anaplastic astrocytoma and glioblastoma multiform, are the most frequent type of glioblastoma. Despite advances in cancer treatment, patients with glioblastoma have such a terrible diagnostic accuracy. In recent years, researchers have concentrated their efforts on determining the cellular, molecular, and genetic processes that contribute to the progression of glioblastoma. As a result, biomarkers have developed as clinical, prognostic, and prospective tools with the potential to revolutionize brain tumor diagnosis. With a better understanding of the essential molecular pathways involved in the progression of glioblastoma, promising diagnostic, prognostic, and predictive biomarkers have been identified, some of which have significant ramifications for precision medicine. Usually, Loss of chromosomes 1p/19q in oligodendrogliomas and expression of O-6-methylguanine-DNA Methyltransferase (MGMT) or Epidermal Growth Factor Receptor (EGFR) status in glioblastomas are two of the most promising biomarkers thus far. The investigation of the therapeutic relevance and implementation of such biomarkers has the potential to transform how scientists diagnose and treat patients with glioblastoma.

Brain tumours are one of the most dreaded and deadly types of cancer. Glial (arising from glial cells) and nonglial (arising from a variety of brain structures such as nerves, blood arteries, and glands) primary brain tumours are classified as benign or malignant. Gliomas, or tumours of the Central Nervous System (CNS), are the most common most common kind of CNS tumours. Gliomas are classified as astrocytomas (produced from astrocytes), oligodendroglia tumours, or ependymomas based on their histological features, and are allocated to the World Health Organization (WHO) based on the presence of anaplastic features suggesting increasing degrees of malignancy [1]. Pilocytic astrocytomas diffuse astrocytomas, anaplastic astrocytomas, and glioblastoma multiforme are the most prevalent astrocytomas. GBM was the most prevalent primary brain and other CNS tumour diagnosed in recent days, according to the most recent world population-based cancer registry statistics. Glioblastoma (GBM) is a type of brain tumour that is both aggressive and incurable. GBM is a very aggressive tumour that grows quickly and has a lot of biological and genetic heterogeneity. The prognosis is bleak, with only an 8-month median survival rate. The discovery of a gene signature for GBM could aid in its diagnosis, therapy, prognosis prediction, and potentially treatment development. Low-grade astrocytomas, oligodendrogliomas, and glioblastoma of the EGFR signaling pathway *via* toma multiforme amplification show unique global gene expression profiles. EGFR gene amplification can be distinguished from one another. While gene expression patterns can be incredibly useful in designing therapeutic strategies, gene expression data can also be used to build prognostic and diagnostic biomarkers [2].

In glioblastoma, mutations in the Epidermal Growth Factor Receptor (EGFR) are prevalent. EGFR activation can be enhanced by a number of distinct ways, both ligand-dependent and ligand-independent. EGFR is overexpressed in the majority of original glioblastomas and certain secondary glioblastomas, according to numerous studies, and is associated with more aggressive glioblastoma morphologies. EGFR is overexpressed in 60% of primary glioblastomas compared to just 10% of secondary glioblastomas, and is associated with more aggressive glioblastoma morphologies. Several other mechanisms, including increased autocrine expression of cognate ligands, account for aberrant EGFR activation in glioblastoma, in addition to overexpression. According to the TCGA, EGFR gene amplification and mutation improve EGFR activation and are found in up to 57% of glioblastomas [3, 4].

The molecular genetic characteristic of oligodendrogliomas is complete deletion of both the short arm of chromosome 1 (1p) and the long arm of chromosome 19 (19q) (1p/19q co-deletion), which accounts for about ten to fifteen percent of all diffuse gliomas in adults. 1p and 19q Loss of Heterozygozity (LOH) is caused by the loss of one hybrid chromosome. An imbalanced

Correspondence to: Sueli Mieko, Department of Neurology, University of Sao Paulo, Paulo, Brazil, E-mail: Sueli@mieko.br

Received: 02-May-2022, Manuscript No. BDT-22-16961; **Editor assigned:** 04-May-2022, PreQC No. BDT-22-16961 (PQ); **Reviewed:** 18-May-2022, QC No. BDT-22-16961; **Revised:** 25-May-2022, Manuscript No. BDT-22-16961 (R); **Published:** 03-Jun-2022, DOI: 10.35248/2168-975X. 22.11.159.

Citation: Mieko S (2022) Biomarkers for Diagnosis, Prognosis, Prediction of Brain Cancer using Molecular Pathways. Brain Disord Ther.11:159.

whole-arm translocation between chromosomes 1 and 19 with the loss of the derivative t(1p;19q) occurs early in the pathogenesis of oligodendrogliomas, resulting in this molecular change. The pathognomonic biomarker 1p/19q co-deletion establishes a separate glioma entity and is found in oligodendrogliomas. Isocitrate Dehydrogenase 1 (IDH1) mutations at arginine 132 (R132) or the corresponding residue arginine 172 in IDH2 (R172) are seen in nearly all 1p/19q co-deleted oligodendrogliomas. Mutations in the Telomerase Reverse Transcriptase (TERT) gene promoter, mutations in homolog of Drosophila capicua (CIC) and Far Upstream Element Binding Protein (FUBP1), and promoter Methylation of The Methyl-Guanine Methyl Transferase (MGMT) genes are all common molecular alterations associated with 1p/19q co-deletion. 1p/19q co-deletion is mutually exclusive with TP53 and ATRX mutation, which both describe glial tumours of the astrocytic lineage, with very few exceptions [5, 6].

CONCLUSION

Many contemporary molecular targeted trials continue to use a "one size fits all" strategy and are not very selective about which patients to enrol in a trial. As a result, numerous targeted therapy trials have failed to demonstrate a meaningful improvement in patient outcomes. With a greater understanding of the molecular biology of these cancers, it will be possible to anticipate which patients would benefit from specific targeted therapies. Furthermore, most targeted medicines have not proven significant survival improvements as monotherapies in malignant gliomas to date. Several factors behind the therapeutic failure of monotherapies, including tumour genetic variability, redundant and overlapping signal transduction pathways, and inadequate drug delivery to tumours. Thus, in a certain group of patients, addressing several signaling pathways or even distinct targets in the same pathway may improve therapy success. Researchers must be pragmatic and practical in our approach to using these biomarkers in clinical care because molecular analysis and testing are expensive and time consuming.

REFERENCES

1. David N, Ohgaki H, Otmar D, Webster K. Cavenee. The 2007 WHO classification of tumours of the central nervous system. Acta Neuropathol. 2007;114 (2): 97-109.
2. Kesari S, Ramakrishna N, Sauvageot C. Targeted molecular therapy of malignant gliomas. Curr Neurol Neurosci Rep. 2006; 5:186-97.
3. Huang, Paul H, Alexander M, Forest M. Oncogenic EGFR signaling networks in glioma. Sc signal. 2009; 87(2):re6.
4. Cameron W, Verhaak GW, McKenna A, Campos B, Noushmehr H, Salama SR, et al. The somatic genomic landscape of glioblastoma. Cell. 2013; 155(2):462-477.
5. Jenkins R, Blair H, Ballman K. A t(1;19)(q10;p10) mediates the combined deletions of 1p and 19q and predicts a better prognosis of patients with oligodendroglioma. Cancer Res. 2006; 66:9852-61.
6. Reifenberger J, Reifenberger G, Liu L. Molecular genetic analysis of oligodendroglial tumors shows preferential allelic deletions on 19q and 1p. Am J Pathol. 1994;145:1175-90.