

PubMed

U.S. National Library of Medicine
National Institutes of Health



Display Settings: Abstract

[Brain Tumor Pathol.](#) 2011 Mar 26. [Epub ahead of print]

Genetic profile of astrocytic and oligodendroglial gliomas.

Ohgaki H, Kleihues P.

Molecular Pathology Section, International Agency for Research on Cancer, 150 Cours Albert Thomas, 69372, Lyon, France, ohgaki@iarc.fr.

Abstract

Low-grade diffuse gliomas WHO grade II (diffuse astrocytoma, oligoastrocytoma, oligodendroglioma) are characterized by frequent IDH1/2 mutations (>80%) that occur at a very early stage. In addition, the majority of diffuse astrocytomas (about 60%) carry TP53 mutations, which constitute a prognostic marker for shorter survival. Oligodendrogliomas show frequent loss at 1p/19q (about 70% of cases), which is associated with longer survival. With respect to clinical outcome, molecular classification on the basis of IDH1/2 mutations, TP53 mutations, and 1p/19q loss showed a predictive power similar to histological classification. IDH1/2 mutations are frequent (>80%) in secondary glioblastomas that have progressed from low-grade or anaplastic astrocytomas. Primary (de novo) glioblastomas with IDH1/2 mutations are very rare (<5%); they show an age distribution and genetic profile similar to secondary glioblastomas and are probably misclassified. Using the presence of IDH1/2 mutations as a diagnostic criterion, secondary glioblastomas account for approximately 10% of all glioblastomas. IDH1/2 mutations are the most significant predictor of favorable outcome of glioblastoma patients. The high frequency of IDH1/2 mutations in oligodendrogliomas, astrocytomas, and secondary glioblastomas derived thereof suggests these tumors share a common progenitor cell population. The absence of this molecular marker in primary glioblastomas suggests a different cell of origin; both glioblastoma subtypes acquire a similar histological phenotype as a result of common genetic alterations, including the loss of tumor suppressor genes on chromosome 10q.

PMID: 21442241 [PubMed - as supplied by publisher]

[LinkOut](#) - more resources