Cyclooxygenase-2 expression in astrocytes and microglia in human oligodendroglioma and astrocytoma.

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Cyclooxygenases (cox) are potent mediators of inflammation and two cox-izoenzymes, cox-1, cox-2, are described to date. Cox-2 is cytokine-inducible in inflammatory cells and enhanced cox-2 expression has been attributed a key role in the development of edema and immunomodulation in pathologically altered brain tissues. In normal cerebral cortex cox-2 is present only in neurons, but not in the glial or vascular endothelial cells. The function of microglia in glioma biology is unclear. Microglia have both neurotrophic and neurotoxic functions and have been shown to release a variety of cytokines. Our preliminary results showed that the expression pattern of cox-2 is predominantly neuronal although glial expression was observed with the correlation of high malignancy. In this study we aimed to assess the phenotypes (astrocyte, microglia) of the cox-2-expressing glial cells in various types of human gliomas and to compare their expression patterns. For this purpose we employed dual immunohistochemistry for cox-2 and GFAP (astrocyte) or LCA-MAC (microglia-macrophage) in archival formalin-fixed, paraffin embedded human tissue diagnosed as oligodendroglioma and/or astrocytoma. The results showed that cox-2 immunoreactivity is up-regulated in the neurons according to the tumor grade. Most of the cox-2 immunoreactive glia were GFAP-positive in anaplastic oligodendrogliomas and at lesser extend in glioblastomas. Cox-2 and LCA co-localization was detected in more glial cells in glioblastomas. It may be speculated that the induction of cox-2 in microglia may contribute to the deleterious effects of prostanoids in cerebral edema formation during the progression of oligodendrogliomas. The detection of cox-2 in astrocytes surrounding the necrotic areas might be important to develop new strategies, such as the usage of cox-2 inhibitors combine with chemotherapy and radiotherapy in the treatment of glioma patients.

PMID: 20052522 [PubMed - as supplied by publisher]

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