Glioma progression

The accurate determination of progression is important not only for the individual care of each patient but also for correct enrollment in clinical trials investigating salvage treatment and reporting results of trials investigating initial treatment. While overall survival is generally the most well-established outcome of oncologic clinical trials, time to progression, progression free survival, and progression free survival at 6 months are becoming more reasonable endpoints in evaluating brain tumor response [1) 2)].

An increase in FLAIR signal of the fluid within the resection cavity might be a highly specific and early sign of local tumor recurrence/tumor progression also for brain metastases. [3)].

see Time to progression.

Differential diagnosis

see Pseudoprogression.

see Periictal pseudoprogression.

Differentially expressed gene analysis and network analysis were performed to identify critical genes affecting glioma progression. The samples were divided into a KIF15 high-expression group and KIF15 low-expression group, and the association between FIK15 expression level and clinical characteristics was summarized and analyzed by performing medical data analysis; the effect of KIF15 on glioblastoma cell proliferation was detected by employing colony formation and MTT assays. The effect of KIF15 on tumor growth in mice was determined. It was found that KIF15 was a potential gene affecting the progression of glioblastoma. In addition, KIF15 was highly expressed in glioblastoma tumor tissues, and KIF15 was correlated with tumor size, clinical stage and other clinical characteristics. After the KIF15 gene was knocked out, the proliferation ability of glioblastoma was significantly inhibited. KIF15 also contributed to the growth of glioblastoma tumors in mice. Therefore, we found KIF15 to be a promising clinical therapeutic target [4)].

PTPRZ1-MET fusion transcript: Protein tyrosine phosphatase receptor type Z1 (PTPRZ1)-MET proto-oncogene receptor tyrosine kinase (MET) (ZM) fusion has been identified as a biomarker for secondary glioblastoma (sGlioblastoma) that is involved in glioma progression, but the mechanism of gliomagenesis

---

Bromopyruvate (3-BrPA) is a glycolysis inhibitor that has been reported to have a strong anti-tumour effect in many human tumours. Several studies have reported that 3-BrPA could inhibit glioma progression; however, its role on the interstitial cells in the glioma microenvironment has not been

investigated. In previous studies, Sheng et al. found that in the glioma microenvironment, glioma stem cells can induce the malignant transformation of macrophages and dendritic cells. In a study, they focused on the effects of 3-BrPA on malignantly transformed macrophages and dendritic cells. First, they found that 3-BrPA inhibited the proliferation of malignantly transformed macrophages and dendritic cells in a dose-dependent and time-dependent manner. Further study indicated that 3-BrPA significantly decreased extracellular lactate and inhibited the clone formation, migration and invasion of malignantly transformed macrophages and dendritic cells. Using an online database and a series of experiments, they demonstrated that 3-BrPA inhibits the malignant progression of malignantly transformed macrophages and dendritic cells via the miR-449a/MCT1 axis. These findings built experimental basis for new approach against glioma [5].

1)
Polley M.-Y. C., Lamborn K. R., Chang S. M., Butowski N., Clarke J. L., Prados M. Six-month progression-free survival as an alternative primary efficacy endpoint to overall survival in newly diagnosed glioblastoma patients receiving temozolomide. Neuro-Oncology. 2010;12(3):274–282. doi: 10.1093/neuonc/nop034.
2)
Trippa L., Wen P. Y., Parmigiani G., Berry D. A., Alexander B. M. Combining progression-free survival and overall survival as a novel composite endpoint for glioblastoma trials. Neuro-Oncology. 2015 doi: 10.1093/neuonc/nou345.
3)
Bette S, Gempt J, Wiestler B, Huber T, Specht H, Meyer B, Zimmer C, Kirschke JS, Boeckh-Behrens T. Increase in FLAIR Signal of the Fluid Within the Resection Cavity as Early Recurrence Marker: Also Valid for Brain Metastases? Rofo. 2017 Jan;189(1):63-70. doi: 10.1055/s-0042-119686. PubMed PMID: 28002859.
4)
Wang L, Zhang X, Liu J, Liu Q. Kinesin family member 15 can promote the proliferation of glioblastoma. Math Biosci Eng. 2022 Jun 6;19(8):8259-8272. doi: 10.3934/mbe.2022384. PMID: 35801464.
5)
Sheng Y, Jiang Q, Dong X, Liu J, Liu L, Wang H, Wang L, Li H, Yang X, Dong J. 3-Bromopyruvate inhibits the malignant phenotype of malignantly transformed macrophages and dendritic cells induced by glioma stem cells in the glioma microenvironment via miR-449a/MCT1. Biomed Pharmacother. 2019 Nov 8;121:109610. doi: 10.1016/j.biopha.2019.109610. [Epub ahead of print] PubMed PMID: 31710894.

From:
https://neurosurgerywiki.com/wiki/ - **Neurosurgery Wiki**

Permanent link:
https://neurosurgerywiki.com/wiki/doku.php?id=glioma_progression

Last update: **2024/06/07 02:52**