Glioma prognosis

- From Seeing to Healing: The Clinical Potential of Radiotracers in Pediatric Neuro-Oncology
- Comparative Analysis of Clinical Outcomes in High-Grade Glioma Patients: 5-ALA Fluorescence-Guided Surgery vs. Conventional White-Light Resection
- Multi-Transcriptomic Analysis Reveals GSC-Driven MES-Like Differentiation via EMT in GBM Cell-Cell Communication
- Molecular Biomarkers of Glioma
- RIPK1 in Diffuse Glioma Pathology: From Prognosis Marker to Potential Therapeutic Target
- IDH1 Mutation Impacts DNA Repair Through ALKBH2 Rendering Glioblastoma Cells Sensitive to Artesunate
- Epigenetic Alterations in Glioblastoma Multiforme as Novel Therapeutic Targets: A Scoping Review
- Evaluating the Antitumor Potential of Cannabichromene, Cannabigerol, and Related Compounds from Cannabis sativa and Piper nigrum Against Malignant Glioma: An In Silico to In Vitro Approach

The prognosis for gliomas varies widely depending on several factors, including the tumor's grade, molecular characteristics, and the patient's age and overall health. Below are the key considerations for glioma prognosis:

1. Tumor Grade

Low-Grade Gliomas (Grade I and II):

Generally have a better prognosis compared to high-grade gliomas. Grade I (e.g., pilocytic astrocytoma): Often curable with complete surgical resection. Grade II (e.g., diffuse astrocytoma): Tend to grow slowly but can progress to higher grades over time.

High-Grade Gliomas (Grade III and IV):

Grade III (e.g., anaplastic astrocytoma): Aggressive, with intermediate survival rates. Grade IV (e.g., glioblastoma): The most aggressive glioma, with poor prognosis despite treatment.

Low-grade glioma prognosis

see Low-grade glioma prognosis.

High-grade glioma prognosis

see High-grade glioma prognosis.

2. Molecular and Genetic Markers

IDH Mutation Status:

IDH-mutant gliomas: Typically associated with better prognosis.

IDH-wildtype gliomas: Tend to behave more aggressively.

1p/19q co-deletion:

Found in oligodendrogliomas and linked to better responses to treatment and longer survival.

MGMT Promoter Methylation:

Associated with better responses to alkylating chemotherapy (e.g., temozolomide) and improved outcomes in glioblastoma.

TERT Promoter Mutations, ATRX Loss, and EGFR Amplification:

Can provide additional prognostic information, particularly in glioblastoma.

3. Age

Younger patients (<40-50 years) generally have better outcomes compared to older patients.

4. Extent of Resection

A greater extent of tumor resection correlates with improved survival for most gliomas, although complete resection may not always be feasible, especially in eloquent brain areas.

General Survival Metrics

Low-grade gliomas: Median survival ranges from 7 to 15 years, depending on factors like age, molecular markers, and extent of resection. High-grade gliomas: Grade III gliomas: Median survival around 3–5 years. Glioblastoma: Median survival typically ranges from 12–18 months, with <5% surviving beyond 5 years. Prognostic Tools and Scoring Systems Tools such as the RPA (Recursive Partitioning Analysis) and molecular profiles are used to estimate individual prognosis more accurately.

Shan et al. established a gene signature associated with ROS to explore its influence on prognosis and immune microenvironment in gliomas.

The ROS-related gene expression profile dichotomized patients into two groups with different clinicopathological features and prognoses. A 19-gene ROS-related signature was used to robustly

predict prognosis in both training and validation datasets. Functional analysis indicated an association between ROS levels and the immune microenvironment. The expression of immune checkpoints and M2-type markers was upregulated in the high-risk group, which suggested the immunosuppressive function of ROS.

ROS-related signature is an independent glioma prognosis factor and could potentially exert immunosuppressive effects on the tumor microenvironment [1].

Early maximal tumor removal results in greater survival in both high-grade and low-grade gliomas, leading to propose “supra-marginal” resection, with excision of the peritumoral zone in diffuse neoplasms. To minimize functional risks while maximizing the extent of resection, traditional “tumor-mass resection” is replaced by “connectome-guided resection” conducted under awake mapping, taking into account inter-individual brain anatomo-functional variability. A better understanding of the dynamic interplay between DG progression and reactional neuroplastic mechanisms is critical to adapt a personalized multistage therapeutic strategy, with integration of functional neurooncological (re)operation(s) in a multimodal management scheme including repeated medical therapies. Because the therapeutic armamentarium remains limited, the aims of this paradigmatic shift are to predict one/several step(s) ahead glioma behavior, its modifications, and compensatory neural networks reconfiguration over time in order to optimize the onco-functional benefit of each treatment - either in isolation or in combination with others - in human beings bearing a chronic tumoral disease while enjoying an active familial and socio-professional life as close as possible to their expectations. Thus, new ecological endpoints such as return to work should be incorporated into future DG trials. “Preventive neurooncology” might also be envisioned, by proposing a screening policy to discover and treat incidental glioma earlier [2]

Diffuse gliomas are incurable brain tumors, yet there is significant heterogeneity in patient survival. Advanced computational techniques such as radiomics show potential for presurgical prediction of survival and other outcomes from neuroimaging. However, these techniques ignore non-lesioned brain features that could be essential for improving prediction accuracy. Gray matter covariance network (connectome) features were retrospectively identified from the T1-weighted MRIs of 305 adult patients diagnosed with diffuse glioma. These features were entered into a Cox proportional hazards model to predict overall survival with 10-folds cross-validation. The mean time-dependent area under the curve (AUC) of the connectome model was compared with the mean AUCs of clinical and radiomic models using a pairwise t-test with Bonferroni correction. One clinical model included only features that are known presurgery (clinical) and another included an advantaged set of features that are not typically known presurgery (clinical +). The median survival time for all patients was 134.2 months. The connectome model (AUC 0.88 ± 0.01) demonstrated superior performance ($P < 0.001$, corrected) compared to the clinical (AUC 0.61 ± 0.02), clinical + (AUC 0.79 ± 0.01) and radiomic models (AUC 0.75 ± 0.02). These findings indicate that the connectome is a feasible and reliable early biomarker for predicting survival in patients with diffuse glioma. Connectome and other whole-brain models could be valuable tools for precision medicine by informing patient risk stratification and treatment decision-making [3].

Reports have demonstrated that chromosomal instability, driven in part by gene mutations maintaining overall genomic stability, is found in subsets of adult-type diffusely infiltrating diffuse

gliomas of all histologic and molecular grades, with resulting in elevated overall copy number burden, chromothripsis, and poor clinical Glioma prognosis. Still, relatively few studies have examined the effect of this process, due in part to the difficulty of routinely measuring CIN clinically.

Richardson et al. reviewed the underlying mechanisms of CIN, the relationship between chromosomal instability and malignancy, the prognostic significance and treatment potential in various cancers, systemic disease, and more specifically, infiltrating diffuse glioma subtypes. While still in the early stages of discovery compared to other solid tumor types in which CIN is a known driver of malignancy, the presence of CIN as an early factor in gliomas may in part explain the ability of these tumors to develop resistance to standard therapy, while also providing a potential molecular target for future therapies [4)].

As the most common primary tumor of the central nervous system, gliomas have a high recurrence rate after surgical resection and are resistant to chemotherapy, particularly high-grade gliomas dominated by glioblastoma —- Yan et al. aimed to investigate the prognostic value of neutrophil-to-lymphocyte ratio (NLR), monocyte/lymphocyte ratio (MLR), and platelet/lymphocyte ratio (PLR) in diffuse glioma, and to establish a prognostic nomogram accordingly.The hematologic and clinicopathological data of 162 patients with primary diffuse glioma who received surgical treatment from January 2012 to December 2018 were retrospectively analyzed. Area under the receiver operating characteristic (ROC) curve was carried out to determine the optimal cut-off values for NLR, MLR, PLR, age, and Ki-67 index, respectively. Kaplan-Meier method was used to investigate the correlation between inflammatory indicators and prognosis of glioma patients. Univariate and multivariate Cox regression were performed to evaluate the independent prognostic value of each parameter in glioma. Then, a nomogram was developed to predict 1-, 3-, and 5-year postoperative survival in diffuse glioma patients based on independent prognostic factors. Subsequent time-dependent ROC curve, calibration curve, decision curve analysis (DCA), and concordance index (C-index) were performed to assess the predictive performance of the nomogram.The Kaplan-Meier curve indicated that patients with high levels of NLR, MLR, and PLR had a poor prognosis. In addition, we found that NLR level was associated with World Health Organization (WHO) grade and IDH status of glioma. The multivariate Cox analysis indicated that resection extent, WHO grade, and NLR level were independent prognostic factors, and we established a nomogram that included these three parameters. The evaluation of the nomogram indicated that the nomogram had a good predictive performance, and the addition of NLR could improve the accuracy.NLR, MLR, and PLR were prognostic factors of diffuse glioma. In addition, the nomogram including NLR was reliable for predicting survival of diffuse glioma patients [5)].

Glial dysfunction outraging CNS plasticity and integrity results in one of the most dangerous cancers, namely glioma, featuring little median survival period and high recurrence. The hallmark properties of proliferation, invasion and angiogenesis with the infiltrated macrophages in glioma are expected to be tightly coupled or cross-linked, but not properly related so far. The present study is aimed to find a relationship between this featured quadrangle from lower to higher grades (HG) of post-operative glioma tissues and their invading subsets. Elevated Ki67-associated proliferation in lower grades (LG) was supported with VEGF dependent angiogenic maintenance which found a decrease unlikely in HG. In contrast, MMP 2 and 9-associated invasions augmented high in HG with the dominant presence of CD204+ M2 polarized macrophages and a general increase in global DNMT1-associated methylation. Marked differences found in ECM invading cellular subsets of HG showing high proliferative capacity indicating rationally for recurrence, contrasting the nature of gross tumor tissue of the same grade. Thus in LG, the neoplastic lesion is more inclined to its growth while in higher grade more disposed towards tissue wreckage in support with cellular environmental milieu whereas the cellular variants

and subsets of invaded cells showed different trends. Therefore, some operational dichotomy or coupling among cellular variants in glioma is active in determining its low- to high-grade transition and aggressive progression [6].

In order to set up a reliable prediction system for the tumor grade and glioma outcome, Li et al. clarified the complicated crosstalk of Annexin A2 (ANXA2) with Glypican 1 (GPC1) and demonstrate whether combined indexes of ANXA2 and GPC1 could improve the prognostic evaluation for glioma patients. Li et al. found that ANXA2-induced glioma cell proliferation in a c-Myc-dependent manner. ANXA2 increased the expression of GPC1 via c-Myc and the upregulated GPC1 further promoted the c-Myc level, forming a positive feedback loop, which eventually led to enhanced proliferation of glioma cells. Both mRNA and protein levels of ANXA2 were upregulated in glioma tissues and coincided with the overexpression of GPC1. Besides, they utilized tissue microarrays (TMAs) and immunohistochemistry to demonstrate that glioma patients with both high expressions of ANXA2 and GPC1 tended to have a higher rate of tumor recurrence and shorter overall survival (OS). In conclusion, the overexpression of ANXA2 promotes proliferation of glioma cells by forming a GPC1/c-Myc positive feedback loop, and ANXA2 together with its downstream target GPC1 could be a potential “combination biomarker” for predicting the prognosis of glioma patients [7].

Glioma Health-Related Quality of Life

see Glioma Health-Related Quality of Life.

The ability to resume professional activities following brain tumor surgery is an important patient-oriented outcome parameter. Senft et al. found that the majority of patients with gliomas were able to return to work following surgical and adjuvant treatment. Preservation of neurological function is of utmost relevance for individual patients quality of life [8]

Also the O6 methylguanine DNA methyltransferase (MGMT) promoter methylation status seem to be the most important predictors of survival.

Infiltrative gliomas invade the brain, relentlessly recur, transform into higher-grade gliomas, and are invariably lethal [9] [10] [11]. , mostly due to the poor glioblastoma outcome (Grade IV glioma).

Gliomas are considered incurable due to recurrence as demonstrated in a series of five patients who underwent hemispherectomies in 1928 [12].

The prognosis improves as the amount of glioma removed increases [13] [14] [15] [16] [17].

Older age (>40 years), high pathological grade, invasion of the corpus callosum and high levels of Ki67 expression were risk factors associated with the intracranial dissemination of gliomas [18].

TERT Promoter Mutation Status in Glioma

TERT Promoter Mutation Status in Glioma

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