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Artificial neural network–based classification of gliomas using molecular and clinical data

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Abstract

Gliomas are the most common primary brain tumors, characterized by substantial heterogeneity in clinical course and prognosis. Accurate tumor grading remains essential for therapeutic decision-making and survival prediction. The integration of molecular and clinical variables into predictive models may improve the diagnostic accuracy beyond histology alone. This retrospective study analyzed 856 patients with glioblastoma (GBM) and lower-grade glioma (LGG) from The Cancer Genome Atlas (TCGA) database. Clinical and mutational data were processed using IBM SPSS Neural Networks (v29.0). A Multilayer Perceptron (MLP) artificial neural network was developed to classify glioma grade. Twenty-two genetic predictors (including IDH1, IDH2, TP53, EGFR, PDGFRA, and others) and demographic variables (age, sex, race) were incorporated. Model performance was assessed on an independent test set using accuracy, sensitivity, specificity, precision, F1-score, Matthews correlation coefficient (MCC), and area under the ROC curve (AUC). The model achieved an overall accuracy of 88.2%, with a sensitivity of 91.5% for GBM and specificity of 85.4% for LGG in the test set. The AUC was 0.945, indicating excellent discriminatory power. Variable importance analysis identified IDH1 mutation as the strongest predictor (normalized importance = 100%), followed by age at diagnosis (70.5%) and IDH2 mutation (54.4%). In contrast, RB1 and BCOR mutations had minimal contributions. The proposed ANN model demonstrated robust accuracy and strong discriminatory performance in glioma grading. The integration of molecular markers such as IDH1/2 with clinical variables enhances diagnostic precision and supports the potential role of artificial intelligence in clinical decision support. Future validation in prospective and multi-center cohorts, as well as multimodal data integration, may facilitate personalized treatment strategies for glioma patients.

Keywords: Glioma, glioblastoma, artificial neural network, molecular markers, cancer genome atlas

Introduction

Gliomas are the most common type of primary brain tumour, originating from glial cells. They are characterised by rapid progression and disruption to nervous system functions. Accurately determining the biological behaviour of these tumours is important for planning treatment and predicting prognosis [1-3]. Standard treatment generally consists of maximal surgical resection, followed by radiotherapy and chemotherapy, most commonly with temozolomide [2-6].

The World Health Organization (WHO) classification of central nervous system (CNS) tumours groups gliomas into low-grade (LGG) and high-grade (HGG) categories. Glioblastoma multiforme (GBM) is the most aggressive and invasive primary brain tumour. While gliomas account for approximately 80% of primary malignant brain tumours, GBM alone constitutes more than 60% of all brain tumours in adults [7,8].

The WHO classification scheme is currently the only widely accepted system for categorising gliomas and continues to serve as a practical and effective tool. However, it relies solely on histopathological visual criteria and is subject to inter-observer variability. Many cases fail to conform strictly to a single category and the classification does not reliably correlate with tumour aggressiveness, treatment response or progression-free survival [9,10].

The interpretation of cellularity, anaplasia and cell type is not always possible due to the histological heterogeneity of these

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tumours [11]. Also, gliomas of the same WHO grade can have very different clinical courses. Genetic sub-grouping of gliomas has the potential to provide more accurate and reproducible diagnostic criteria than the current system [9].

Research into genetic and epigenetic prognostic factors has identified new ways to categorise these tumours, define new molecular targets for therapies and improve our understanding of their biology. New molecular alterations and multivariable models have been proposed to address and better classify these tumours. Recent studies have shown that comprehensive molecular data can be generated rapidly enough to be used at the time of diagnosis [8,9].

In recent years, artificial intelligence (AI) and machine learning (ML)-based methods have emerged as valuable tools for grading gliomas and predicting prognosis. Deep learning models applied to magnetic resonance imaging (MRI) data have demonstrated high accuracy in classification tasks, and integrating histopathological images with molecular data has provided more comprehensive biological insights than conventional methods [7-9,12,13]. While most current studies are retrospective and primarily database-driven, the growing adoption of multi-institutional cohorts is anticipated to address the lack of external validation and facilitate clinical translation [14,15].

In conclusion, AI-based models that integrate molecular, clinical, and imaging data have the potential to significantly improve diagnostic accuracy and contribute to the development of personalised therapeutic approaches in glioma management.

This study aimed to comprehensively evaluate the performance of an Artificial Neural Network (ANN)-based model that integrates molecular and clinical features for glioma grading. Furthermore, the study sought to identify the molecular determinants with the greatest discriminatory power in glioma grading by analyzing the relative importance of genetic biomarkers associated with tumor classification. In addition, it aimed to investigate whether the integration of molecular biological data with clinical characteristics could improve the accuracy and reliability of glioma grading compared with conventional classification approaches, while also identifying the key genetic factors that may contribute to a better understanding of the biological behavior of gliomas.

Material and Methods

Data Acquisition and Preprocessing

The mutational and clinical data for this analysis were sourced from namely The Cancer Genome Atlas (TCGA) database, encompassing patients with GBM and LGG [13]. It is not necessary to obtain ethics committee approval, since the TCGA data have been sourced from an open-source database. The initial dataset comprised 862 cases. Data management and analysis were performed using IBM SPSS Statistics (Version 29.0.0.0). A listwise deletion protocol was applied for handling missing data, resulting in a final analytical sample of 856 cases with complete data for all variables. The continuous covariate, Age_at_diagnosis, was standardized (z-scores; mean = 0, standard deviation = 1) to facilitate model convergence.

Predictive Modeling

A Multilayer Perceptron (MLP) artificial neural network was implemented to classify tumor grade (dependent variable: Grade, with LGG and GBM) based on a set of predictor variables [14]. The predictor set included 22 categorical factors (mutational status of genes: ATRX, BCOR, CIC, CSMD3, EGFR, FAT4, FUBP1, GRIN2A, IDH1, IDH2, MUC16, NF1, NOTCH1, PDGFRA, PIK3CA, PIK3R1, PTEN, RB1, SMARCA4, TP53; and demographic variables: Gender, Race) and one continuous covariate (Age_at_diagnosis).

The network's architecture was automatically optimized using the software's automaper, constrained to a single hidden layer with a minimum of 1 and a maximum of 50 units. The final architecture consisted of one hidden layer with 8 units employing a hyperbolic tangent activation function. The output layer used a softmax activation function, appropriate for binary classification, and the model was trained by minimizing the cross-entropy error function.

The model was trained using the batch training method with the scaled conjugate gradient optimization algorithm. The hyperparameters for optimization were set as follows: initial lambda = 0.0000005, initial sigma = 0.00005, interval center = 0, interval offset = 0.5. To prevent overfitting and ensure robust performance estimation, the dataset was partitioned randomly into a training set (70% of cases, n=594) for model building and a testing set (30%, n=262) for unbiased evaluation. Training was governed by strict stopping rules: a maximum training time of 15 seconds, a maximum of 1 consecutive step with no error decrease, a minimum error change of 1.0E-4, and an error ratio of 0.001.

Performance Metrics and Evaluation

Model performance was evaluated on the independent testing set. The following metrics were calculated based on the confusion matrix to provide a comprehensive assessment [15]:

- **Accuracy:** (true positive (TP) + true negative (TN)) / Total
- **Sensitivity (Recall):** TP / (TP + false negative (FN))
- **Specificity:** TN / (TN + false positive (FP))
- **Precision (Positive Predictive Value - PPV):** TP / (TP + FP)
- **Negative Predictive Value (NPV):** TN / (TN + FN)
- **F1-Score:** 2 * (Precision * Sensitivity) / (Precision + Sensitivity)
- **Matthews Correlation Coefficient (MCC):** (TPTN-FPFN)/ √((TP+FP)*(TP+FN)*(TN+FP)*(TN+FN))
- **Area under the ROC Curve (AUC):** The area under the Receiver Operating Characteristic (ROC) curve was used as a primary metric for evaluating the model›s overall discriminatory power, providing a comprehensive measure of classification performance across all possible thresholds [16].

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Variable importance was computed by the SPSS algorithm, which analyzes the learned synaptic weights from the network to determine the relative importance of each predictor in the model. The importance values are normalized relative to the most important predictor.

Results

Model Performance and Evaluation

The MLP model demonstrated exceptional performance in classifying tumor grade. The case processing summary indicated that 856 of 862 total cases (99.3%) had valid data for all variables and were included in the analysis.

The overall percent of correct predictions on the testing set was 88.2% (Table 1). The model showed high and balanced performance across both classes, with a sensitivity of 91.5% for GBM and a specificity of 85.4% for LGG. The MCC of 0.763 indicates a strong positive correlation between the predictions and the observed classes, signifying a high-quality classifier beyond what accuracy alone can show [17]. The area under the ROC curve was 0.945 (Figure 1), confirming outstanding and balanced discriminatory power [16]. An AUC of 0.945 is considered excellent and indicates a very high likelihood that the model will rank a random GBM tumor higher than a random LGG tumor.

Table 1. Model performance metrics on the testing set (N = 262)

Metric	Value	Calculation
Accuracy	88.2%	(123+108)/262
Sensitivity (Recall)	91.5%	108/(108+10)
Specificity	85.4%	123/(123+21)
Precision (PPV)	83.7%	108/(108+21)
NPV	92.5%	123/(123+10)
F1-score	87.4%	2(0.8370.915)/(0.837+0.915)
MCC	0.763	(108*123 - 21*10)/√((108+21)(108+10)(123+21)(123+10))
AUC	0.945	

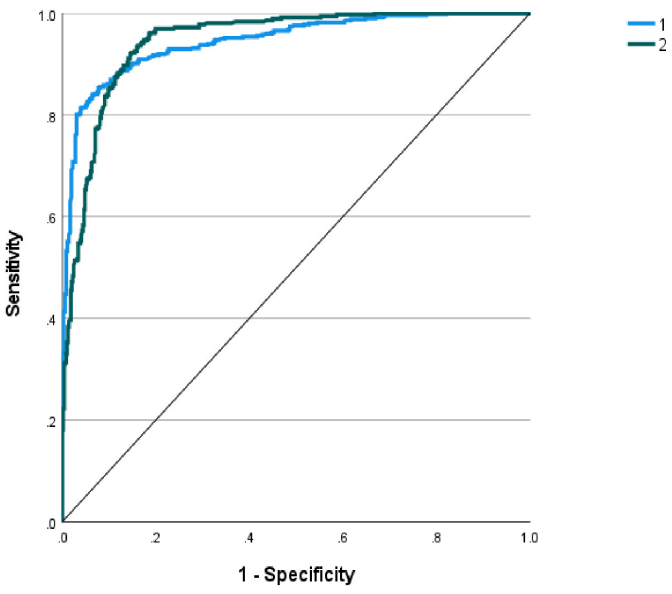

Figure 1. Receiver operating characteristic (ROC) curve

The ROC curve for the model plotted the true positive rate (sensitivity) against the false positive rate (1 - specificity) for the classification of Grade. The AUC was 0.945 for both classes, indicating excellent model performance.

Variable Importance Analysis

The normalized importance of predictors, calculated based on the learned synaptic weights, identified key biomarkers driving the classification. The results are summarized in Table 2 and visualized in Figure 2.

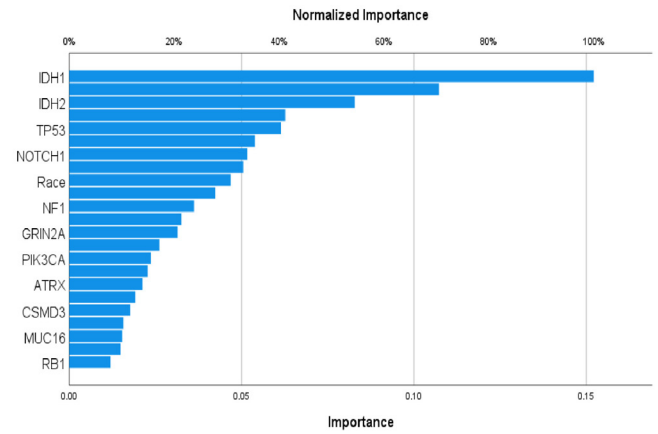

Figure 2. Normalized importance of predictor variables

The most important predictor was the mutational status of IDH1 (normalized importance = 100%), which is a well-established molecular marker in glioma stratification [12]. This was

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followed by Age_at_diagnosis (70.5%) and the mutational status of IDH2 (54.4%). Factors such as RB1 (7.8%) and BCOR (9.7%) contributed the least to the model›s predictions. The distribution of importance is also presented graphically below.

Table 2. independent variable importance

Predictor	Importance	Normalized %
IDH1	0.152	100.0
Age_at_diagnosis	0.107	70.5
IDH2	0.083	54.4
CIC	0.063	41.2
TP53	0.061	40.4
PIK3R1	0.054	35.4
NOTCH1	0.052	33.9
SMARCA4	0.05	33.1
Race	0.047	30.8
PDGFRA	0.042	27.8
NF1	0.036	23.8
FAT4	0.033	21.4
GRIN2A	0.031	20.6
PTEN	0.026	17.1
PIK3CA	0.024	15.6
EGFR	0.023	15.0
ATRX	0.021	13.9
FUBP1	0.019	12.6
CSMD3	0.018	11.6
Gender	0.016	10.3
MUC16	0.015	10.1
BCOR	0.015	9.7
RB1	0.012	7.8

IDH1: isocitrate dehydrogenase (NADP(+)) 1, IDH2: isocitrate dehydrogenase (NADP(+)) 2, CIC: capicua transcriptional repressor, TP53: tumor protein p53, PIK3R1: phosphoinositide-3-kinase regulatory subunit 1, NOTCH1: notch receptor 1, SMARCA4: SWI/SNF related BAF chromatin remodeling complex subunit ATPase 4, PDGFRA: platelet derived growth factor receptor alpha, NF1: neurofibromin 1, FAT4: FAT atypical cadherin 4, GRIN2A: glutamate ionotropic receptor NMDA type subunit 2A, PTEN: phosphatase and tensin homolog, PIK3CA: phosphatidylinositol-4:5-bisphosphate 3-kinase catalytic subunit alpha, EGFR: epidermal growth factor receptor, ATRX: ATRX chromatin remodeler, FUBP1: far upstream element binding protein 1, CSMD3: CUB and Sushi multiple domains 3, MUC16: mucin 16: cell surface associated, BCOR: BCL6 corepressor, RB1: RB transcriptional corepressor 1.

Discussion

The ANN model developed in this study demonstrated high accuracy and strong discriminatory performance when grading gliomas. Accuracy of 88.2%, sensitivity of 91.5% and specificity of 85.4% were achieved on the testing set, indicating that the model could classify both LGG and GBM equally well. Furthermore, an AUC of 0.945 indicates an excellent level of discriminatory power that is clinically meaningful. These results suggest that AI-based methods have the potential to improve the accuracy of glioma diagnosis and serve as valuable tools in clinical decision support systems.

The results of our study are consistent with those of other AI-based approaches reported in the literature. For example, Zhuge et al. (2020) achieved high performance in glioma grading by applying deep convolutional neural networks (CNNs) to conventional MRI data [2]. Similarly, Pereira et al. (2018) reported high accuracy rates with CNN-based models in glioma classification [4]. What sets our study apart is its integration of molecular and clinical data, offering a more comprehensive biological perspective than models that rely solely on imaging data.

Variable importance analysis identified the IDH1 mutation as the strongest predictor, consistent with the prognostic significance highlighted in the 2021 WHO classification of central nervous system tumours [12]. IDH1/IDH2 mutations are critical biomarkers for prognostication in gliomas, and the model's significant weighting of these variables further supports its robustness. Age was found to be the second most important factor, consistent with clinical observations that advanced age is associated with a worse prognosis [7].

Age at diagnosis was the second most important factor (70.5%). Research shows that younger patients with gliomas, especially those with IDH mutations, live longer than older patients [9]. The findings of this study are in alignment with the proposed model which emphasises the pivotal role of age as a determining factor in prognostic assessments, alongside molecular alterations. Other predictors such as CIC, TP53, PIK3R1 and NOTCH1 also contributed substantially. CIC and FUBP1 mutations often occur with IDH1/2 alterations and 1p/19q codeletions, characterising oligodendrogliomas with favourable clinical outcomes. TP53 and ATRX mutations are commonly associated with astrocytic lineage and may indicate a distinct clinical course.

Alterations in PIK3R1, PTEN, EGFR, and PDGFRA are observed in high-grade gliomas, particularly in glioblastoma, where they converge on the RTK/PI3K, p53, and RB signalling pathways. Dysregulation of these pathways contributes to the highly aggressive nature of glioblastoma [7]. The present analysis lends further support to this hypothesis, as evidenced by the finding that EGFR, PTEN, and PDGFRA all emerged as relevant, albeit moderately ranked, predictors. It is noteworthy that NF1 and FAT4 mutations were also observed in the present model. The presence of NF1 loss is a hallmark of the mesenchymal glioblastoma subtype, which is associated with a poor prognosis [9].

Markers such as RB1 and BCOR contributed minimally to classification, suggesting that these genetic factors may play only a secondary role in glioma grading.

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However, Mirchia and Richardson (2020), as well as Vigneswaran et al. (2015), emphasised that the prognostic and predictive value of emerging molecular markers will become clearer over time, and that multivariable models are better suited to capturing their significance [8,9].

One strength of our study is our large sample size (n = 856), which allowed us to integrate molecular and demographic variables to build a multidimensional model. Furthermore, the high Matthews correlation coefficient (MCC = 0.763) demonstrates reliable classification power, even in the presence of imbalanced data, which provides an advantage over accuracy alone [17]. Nevertheless, some limitations should be acknowledged. Firstly, the data were retrospective and derived exclusively from the TCGA database, which may limit generalisability. Therefore, validation in independent cohorts, particularly in prospective multicentre studies, is required. Secondly, the model incorporated only genetic and clinical variables, without including radiomic, transcriptomic or proteomic data. This may restrict the ability to fully capture tumour heterogeneity.

Mobadersany et al. demonstrated that deep learning models integrating histopathological images with genomic biomarkers can improve survival prediction in patients with glioma. In contrast, our study focuses on tumor-grade classification using genomic mutational status and demographic variables without imaging data. Together, these findings support the value of incorporating molecular information into AI-based models for glioma assessment and highlight the potential benefit of multimodal approaches in future studies [18].

The present study demonstrates that a MLP neural network integrating molecular and demographic variables can effectively discriminate GBM from LGG, achieving high accuracy, balanced sensitivity and specificity, and an excellent AUC of 0.945. These findings indicate strong predictive capacity and clinical applicability.

When contextualised within the broader AI literature, our results align with the methodological principles emphasised by Das et al., who systematically reviewed deep learning–based brain tumour studies and reported that more than half exhibited high risk-of-bias due to inadequate validation strategies, limited performance metrics, and overreliance on complex architectures.

The present study addresses these limitations by implementing an independent testing cohort, applying strict early-stopping criteria, and evaluating performance with multiple complementary metrics such as MCC and AUC. This methodological approach serves to enhance the robustness of the model, thereby reducing the likelihood of inflated performance estimates.

In contrast to the numerous deep learning segmentation models discussed by Das et al., which require substantial computational power and frequently lack interpretability, our MLP framework offers a more transparent and clinically accessible alternative. The findings underscore the significance of IDH1, IDH2, and patient age as key predictors, thereby reinforcing the biological relevance of the model. This observation addresses the concerns raised by Das et al. regarding the interpretability gap in deep learning systems [19].

Mandal et al. emphasise that deep learning has significantly advanced neuro-oncological diagnostics by enhancing tumour segmentation, classification, and even real-time intraoperative decision-making. However, the majority of studies remain predominantly imaging-focused and rely heavily on convolutional architectures rather than integrated biological data sources.

Whilst the majority of studies on deep learning applications in radiology have focused on automated feature extraction, the results of this study demonstrate that integrating genomic alterations, including IDH1/IDH2, TP53, EGFR, and PDGFRA mutations, with demographic factors provides a clinically significant refinement of glioma stratification. Consequently, the present study complements extant AI-driven neuro-oncological research by shifting the predictive emphasis from imaging-based pattern recognition toward biologically grounded, molecularly informed modelling. This shift offers a practical decision-support tool that aligns with the growing radiogenomic paradigm, as described in recent reviews [20].

Bibi et al. have primarily concentrated on MRI-based segmentation using deep learning architectures, including U-Net, cascaded CNNs, and transformer-based models. These architectures have been reported to achieve high spatial accuracy for delineating tumour subregions, including necrosis, oedema, and the enhancing tumour core [21].

In contrast to these image-driven frameworks, the present study demonstrates that molecular mutational profiles alone, when modelled using a MLP, can achieve reliable discrimination between GBM and LGG, highlighting the strong intrinsic diagnostic value of genomic features independent of imaging. Whilst segmentation-based models primarily support surgical planning and radiotherapy targeting, the present approach contributes at a complementary decision-making level by enabling pre-imaging molecular-grade estimation, which may be particularly valuable in centres where rapid genomic stratification is prioritised [21].

It is evident that translational challenges of a similar nature apply to mutation-driven classification models such as the one under consideration, particularly with regard to the absence of external validation and prospective deployment. However, in contrast to imaging-based networks, which are susceptible to inter-scanner variability and labeling bias, our TCGA-driven model utilises standardized genomic pipelines and objective mutation encoding, thereby enhancing its internal consistency. The findings suggest that future neuro-oncology AI systems should adopt a truly multimodal strategy, integrating mutation-based classifiers such as the one presented here with MRI-based

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segmentation frameworks to achieve both anatomical precision and molecular-grade accuracy in the personalised management of glioma [21].

Recent studies have demonstrated the potential of artificial intelligence–based approaches to achieve high accuracy in brain tumour detection and classification using MRI data. Anaya-Isaza et al. conducted a comparative analysis of multiple deep learning architectures and demonstrated that transfer learning and data augmentation significantly enhance classification and detection performance. The study further revealed that fluid-attenuated inversion recovery (FLAIR) sequences exhibit superior sensitivity for tumour detection [22].

In a similar vein, Abdusalomov et al. (2023) reported that integrating You Only Look Once (YOLO)-based detection frameworks with non-local neural network modules enhances tumour localisation accuracy and robustness. This emphasises the added value of attention mechanisms in capturing global contextual information [23].

In contradistinction to pipelines that are entirely dependent on deep learning, Başaran (2022) proposed a hybrid diagnostic framework that combines handcrafted texture features with those of a convolutional neural network, in addition to optimisation algorithms. The combination of these features was found to result in a high level of diagnostic accuracy, thereby highlighting the complementary role of feature selection strategies [24].

Conclusion

In conclusion, this study shows that ANN-based models are a promising approach to glioma classification. Combining the assessment of molecular and clinical variables enhances diagnostic accuracy and informs clinical decision-making. Future studies integrating multimodal data and validating the model across diverse patient populations will be essential in supporting the development of personalised therapeutic strategies.

Ethical Approval

The mutational and clinical data for this analysis were sourced from the Cancer Genome Atlas (TCGA) database, which encompasses patients with glioblastoma and lower-grade glioma. Consequently, the establishment of an ethics committee is rendered redundant.

Patient Consent

Not necessary for this manuscript. The mutational and clinical data for this analysis were sourced from the Cancer Genome Atlas (TCGA) database, which encompasses patients with glioblastoma and lower-grade glioma.

Conflict of Interest

The authors confirm the absence of any conflicts associated with this work.

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