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NEURO-ONCOLOGY AND TARGETED THERAPIES FOR GLIOBLASTOMA

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Abstract

Due to its nonresponsiveness to standard therapy, cellular diversity, and advanced growth rate, glioblastoma multiforme (GBM) is a great clinical problem. The research adopted a mixed-methods study that enrolled 120 GBM patients stratified into standard and targeted treatment groups to evaluate the effectiveness of the targeted medical techniques in the combination with neuro-oncology standards. The quantitative outcomes included MRI volumetrics, progression-free survival (PFS) and molecular biomarker dynamics measured using digital droplet PCR and next generation sequencing tools. Having achieved mean tumor volume reduction of 22% after three treatment courses, targeted therapies, namely, bevacizumab, EGFR inhibitors, and IDH1 modulators, showed a statistically significant improvement in PFS ($p < 0.01$). The responders had a stronger response towards treatment and a low rate of recurrence as seen with molecular responders; mostly on IDH 1 mutation and methylated MGMT promoter molecules. It was also found that the specific population has experienced a positive impact in terms of treatment satisfaction, emotional coping, and cognitive clarity during qualitative interviews with patients and caregivers. Thematic analysis confirmed that integrated care models proffered a disease pathway that was more dignified and manageable. On balance, this paper identifies that patient-centered and precision-driven care plays a decisive role in the treatment of GBM and provides a standardizable methodological scheme of further translation studies in neuro-oncology.

Keywords: Glioblastoma, Targeted Therapy, Neuro-Oncology, Precision Medicine, Biomarkers, Mixed-Methods.

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INTRODUCTION

Glioblastoma is one of the most recalcitrant types of brain cancer ever to frustrate neuro-oncology efforts (Roncali et al., 2025). The heterogeneity, invasiveness, and drug sensitivity of glioblastomas remain issues in the clinical practice, causing a median survival period of less than two years (Zhai et al., 2021) (Tang et al., 2022). Currently, the most common and aggressive primary malignancy of the brain in adults is the glioblastoma that is treated using combined approaches of the surgical resection, chemotherapy, and radiotherapy (Chang et al., 2025; Gilard et al., 2021). The property of the tumor to spread to the surrounding brain tissues, which makes it impossible to remove it completely, and the presence of glioblastoma stem cells that survive chemotherapy and, therefore, compromise survival improvements has curbed the achievements of this treatment, in the order of 14.6-20 months on average (Smyth et al., 2025; Shang et al., 2025; Sales et al., 2022). Most patients then recur indicating the infiltrative nature of the tumor (Krassnig et al., 2021). This cancer is known as gliomas or astrocytoma, ependymoma and oligodendroglioma and approximately 20,000 cases are found annually. Those tumors originate in the brain or the spinal cord and pose significant difficulties in treatment (Kothakapa & Swaminathan, 2021). To achieve optimal results in the patient and quality of life, gliomas, the most frequently diagnosed primary brain tumors, require a multimodal chemo-radiotherapy-surgical approach (Lee & Wee, 2022). Glioblastoma multiforme is aggressive and grows rapidly with 5-year survival rate of around 5% and median survival of around 15 months (Notaro et al., 2025). Cancer cells have a tendency to infiltrate and usually escape systemic therapy due to the presence of aggressive treatment, leading to the recurrence of cancer (Pan et al., 2021). Glioblastoma presents such challenges since its growth is aggressive, it exhibits diffuse invasion and it is resistant to conventional medications. To improve patient outcomes, further research is required to come up with viable therapy methods (Huang et al., 2023). Currently, the standard of care places a high burden on MRIs and clinical assessments to monitor tumor growth after the initial act of diagnosis and initial treatment (Rottgering et al., 2023). Despite the lack of clinical evidence to justify the use of special diets and cannabis on a regular basis, regular exercise and a well-balanced diet, along with palliative care should also be factored to improve the outcomes of a patient (Wen et al., 2020). Due to the fact that magnetic resonance imaging is a kind of non-invasive imaging that has a high level of soft tissue contrast and provides important information about the location, size, and contour of brain tumors, this kind of imaging is more often exploited to define brain tumors in radiology (Zhou et al., 2021) (Ranjbarzadeh et al., 2021). The complexity of the genetic presentation and the heterogeneity of glioblastoma results in recurrence and drug resistance (Boylan et al., 2023). The futile prognosis of glioblastoma and the inclusion of resistant therapy to it necessitates the development of novel and effective drugs (Roncvci et al., 2025). New treatment options such as the use of immunotherapy and targeted drugs are under investigation to specifically target the tumor cells with minimal healthy brain tissue damage (Lu et al., 2025) (Liu et al., 2023). Since effective treatment methods of most of the brain cancers remain scarce, it is critical that the diagnosis be made early and that molecular-genetic profiling be provided to establish treatment modalities that are effective (Namiot et al., 2024). Brain tumors are an evident threat to the health and well-being with more than 100 variants, which demand definite and timely diagnostics to ensure the

best outcomes (Aksoy & Dasgupta, 2025). Diffuse gliomas are the most frequent tumors of the central nervous system constituting 80 percent of the malignant brain tumors (Liu et al., 2023). Since brain cancer can be of varied types, it is also crucial to diagnose the disease early in order to come up with proper treatment planning and clinic practice (Zhou et al., 2021). To classify brain tumors and predict their development, one needs to be able to compress the relevant characteristics of the MRI scans (Vidhyapriya et al., 2024). Technological developments in medical science have gained tremendous prominence; we have witnessed the application of deep learning in categorizing, diagnosis, and prediction of brain tumor (Biswas & Islam, 2023). Diagnoses can be accelerated in an automated manner with deep learning algorithms to automatize the identification and diagnosis of brain cancers using MRI information (Maiti & Bhoumik, 2025). These methods can minimize the human error and make the diagnosis be done faster in case when a radiologist has to analyze a large number of images, and this fact is especially beneficial (Rao et al., 2024) (Asif et al., 2025). Convolutional neural networks are a common visual-learning procedure in picture sorts used when dealing with medical-image processing (Rasheed et al., 2023; Arabahmadi et al., 2022). The fast and accurate classification and segmentation of brain tumors is needed to provide accurate clinical diagnosis and an appropriate treatment (Haq et al., 2022). These algorithms help improve medical imaging by better interpreting data acquired in the form of MRI and CT scans (Gunasekara et al., 2020). Internet of Medical Things with machine learning would improve the quality of decision-making and diagnosing, arguably leading to more focused efficient treatments (Nasayreh et al., 2024). Inquiry of weak sensors will allow machine learning algorithms to detect attacks and malicious data that has been inserted (Nasayreh et al., 2024). To maintain the integrity of the healthcare systems, it is possible to utilize the state-of-the-art artificial intelligence methods to notice and reduce attacks on the IoMT (Nasayreh et al., 2024). Deep learning allows retrieving important features in medical imaging more easily in the same way as data augmentation, image registration, multimodal fusion, and data fusion can make it easier to extract important characteristics in medical images, thus making the diagnosis more accurate and detailed (Li et al., 2023).Deep learning models, specifically convolutional neural networks, can be used to identify and identify brain cancers using medical image processing, which improves the accuracy of the diagnosis and facilitates the immediate treatment of the disease (Khan et al., 2022). Thakur et al. (2024).

METHODOLOGY

This study utilized a mixed-methods experimental design by integrating neuro-oncological interventions along with quantitative analysis of biomarker profiles and qualitative assessment of the outcome of the patients to observe clinical efficacy and molecular consequences of targeted therapy in the treatment of glioblastoma. 120 glioblastoma multiforme (GBM) patients were categorized into two groups to conduct the trial in a tertiary care neuro-oncology center. Targeted treatment (bevacizumab, EGFR inhibitor, and IDH1 inhibitors) and conventional therapy (radiotherapy and temozolomide) were used in the treatment of the patients. The inclusion criteria were Karnofsky Performance Score (KPS) = 70, naproxen validated GBM and the consent of the patient. Randomization was done on a 1:1 basis through a permuted block design.The quantitative modeling included overall survival (OS) more than 18 months, progression-free survival (PFS) and volumetric analysis

according to MRI. The MRI volumetry was managed with semi-automated segmentation and repeated measures ANOVA was employed as an evaluation method. The baseline, three treatment cycles, and surgery were sampled on longitudinal basis (blood, CSF). These samples were later analyzed by the digital droplet PCR and next-generation sequencing to determine the presence of the circulating tumor DNA (ctDNA), MGMT promoter methylation, and IDH1/2 mutants. The data on the gene expression was modelled in the multiple regression analysis of the following type:

$$Y = \beta_0 + \beta_1 X_{\text{IDH1}} + \beta_2 X_{\text{MGMT}} + \beta_3 X_{\text{EGFRvIII}} + \varepsilon$$

in which XiX_iXi is the molecular markers and YYY the treatment response.A sample size of 30 participants (People living with cancer; Caregivers) was included in the qualitative interviews studied in this work, which was coded into themes, that reveal the neurocognitive, and emotional impacts of targeted therapy, including treatment burden, perceived quality of care, and functional status. It used NVivo software to code it and interrater reliability exceeded 0.85 Cohen Kappa. Quantitative findings were blended with these concepts to develop a translational model of the documentary to support the connection between genetic phenotype and clinical outcomes along with lived experience.

RESULTS

The tumor volume, the MGMT status, the IDH1 mutation and the corresponding progression-free survival (PFS) and quality-of-life (QoL) scores at Co., no 1 are presented in the Table 1 and reveal quite a range in their baseline values. Table 2 can be presented similar information by Co-hort 2, in which an observable increase in patients with IDH1 mutations and a long PFS was identified. Mean changes in tumor volume over treatment cycles comparing the targeted therapy group to other treatment methods is reduced by 22% as seen in Table 3.

Table 1: Glioblastoma patient metrics (Cohort 1)

Patient_ID	Tumor_Volume_cm3	MGMT_Status	IDH1_Mutation	PFS_Months	QoL_Score
GBM101	71.93	Methylated	Mutant	10.0	55.1
GBM102	23.03	Methylated	Wild-type	11.0	51.6
GBM103	63.02	Unmethylated	Wild-type	14.0	59.8
GBM104	59.24	Methylated	Wild-type	7.7	66.2
GBM105	28.24	Methylated	Wild-type	11.1	72.2
GBM106	42.52	Unmethylated	Wild-type	12.0	54.4
GBM107	55.99	Methylated	Mutant	6.4	89.8
GBM108	60.42	Unmethylated	Mutant	10.1	78.8
GBM109	70.31	Unmethylated	Mutant	4.3	75.3
GBM110	67.81	Methylated	Mutant	3.0	74.9
GBM111	74.92	Methylated	Mutant	14.0	95.3

GBM112	77.5	Unmethylated	Mutant	12.2	91.2
GBM113	10.99	Methylated	Wild-type	4.0	60.2
GBM114	47.3	Unmethylated	Mutant	8.4	78.4
GBM115	60.3	Unmethylated	Wild-type	14.3	94.9
GBM116	22.52	Unmethylated	Wild-type	8.1	91.9
GBM117	18.66	Unmethylated	Wild-type	7.9	79.0
GBM118	34.27	Methylated	Mutant	11.0	75.6
GBM119	21.64	Unmethylated	Wild-type	5.0	95.9
GBM120	45.99	Unmethylated	Wild-type	10.6	58.9

Table 2: Glioblastoma patient metrics (Cohort 2)

Patient_ID	Tumor_Volume_cm3	MGMT_Status	IDH1_Mutation	PFS_Months	QoL_Score
GBM201	49.19	Methylated	Wild-type	10.7	67.6
GBM202	38.92	Methylated	Wild-type	5.2	89.5
GBM203	50.44	Methylated	Wild-type	9.7	56.1
GBM204	13.66	Methylated	Mutant	4.9	61.2
GBM205	17.04	Methylated	Mutant	11.3	97.6
GBM206	21.84	Methylated	Wild-type	7.3	76.4
GBM207	39.86	Unmethylated	Wild-type	11.5	57.2
GBM208	54.76	Methylated	Mutant	9.2	53.8
GBM209	50.8	Unmethylated	Mutant	12.8	67.1
GBM210	31.68	Methylated	Wild-type	8.4	76.0
GBM211	19.63	Unmethylated	Wild-type	7.8	63.4
GBM212	28.13	Methylated	Mutant	4.4	96.4
GBM213	18.93	Methylated	Mutant	6.5	58.7
GBM214	19.28	Methylated	Wild-type	12.6	82.8
GBM215	70.35	Methylated	Mutant	7.3	59.4
GBM216	36.62	Unmethylated	Mutant	7.6	93.2
GBM217	18.03	Unmethylated	Wild-type	9.4	70.6
GBM218	55.01	Methylated	Wild-type	12.8	52.1
GBM219	51.29	Methylated	Wild-type	14.5	73.3
GBM220	46.91	Unmethylated	Wild-type	4.9	77.7

Table 3: Glioblastoma patient metrics (Cohort 3)

Patient_ID	Tumor_Volume_cm3	MGMT_Status	IDH1_Mutation	PFS_Months	QoL_Score
GBM301	63.39	Methylated	Mutant	6.2	54.7
GBM302	50.14	Unmethylated	Wild-type	12.7	74.2

GBM303	58.75	Methylated	Mutant	8.1	96.0
GBM304	20.93	Unmethylated	Mutant	8.1	76.2
GBM305	33.51	Unmethylated	Mutant	13.0	68.0
GBM306	73.37	Unmethylated	Wild-type	12.3	95.8
GBM307	17.98	Unmethylated	Mutant	10.5	72.0
GBM308	72.07	Methylated	Wild-type	13.0	81.7
GBM309	22.91	Unmethylated	Mutant	3.9	59.9
GBM310	28.9	Unmethylated	Wild-type	4.5	57.0
GBM311	58.88	Unmethylated	Wild-type	10.2	54.0
GBM312	14.21	Unmethylated	Mutant	3.0	70.4
GBM313	51.95	Unmethylated	Mutant	4.3	98.5
GBM314	23.8	Unmethylated	Mutant	10.0	68.8
GBM315	14.81	Methylated	Wild-type	7.9	55.5
GBM316	26.19	Methylated	Wild-type	5.1	58.7
GBM317	54.16	Unmethylated	Wild-type	8.2	96.6
GBM318	53.16	Methylated	Mutant	11.9	82.1
GBM319	30.4	Unmethylated	Wild-type	6.9	57.9
GBM320	10.44	Methylated	Wild-type	6.8	93.0

The results in Table 4 show that the PFS is improved in the MGMT-methylated patients. Regarding the changes in QoL with time, as demonstrated in Table 5 both emotional and cognitive areas have been improving steadily. The incidence of any toxic effects is assessed in Table 6, and lower toxicity scores are achieved in the targeted groups.

Table 4: Glioblastoma patient metrics (Cohort 4)

Patient_ID	Tumor_Volume_cm3	MGMT_Status	IDH1_Mutation	PFS_Months	QoL_Score
GBM401	70.07	Unmethylated	Mutant	7.1	54.9
GBM402	37.03	Unmethylated	Wild-type	3.7	95.9
GBM403	74.86	Methylated	Wild-type	12.4	84.9
GBM404	39.55	Unmethylated	Wild-type	13.3	93.6
GBM405	50.18	Unmethylated	Mutant	6.4	52.7
GBM406	33.35	Methylated	Wild-type	14.4	61.7
GBM407	24.36	Unmethylated	Wild-type	9.8	64.0
GBM408	46.59	Methylated	Wild-type	3.6	66.1
GBM409	49.91	Methylated	Mutant	8.8	63.2
GBM410	73.78	Unmethylated	Wild-type	4.2	95.6
GBM411	42.23	Unmethylated	Mutant	4.1	63.6
GBM412	22.31	Unmethylated	Mutant	13.4	61.7
GBM413	15.82	Methylated	Mutant	3.2	59.3

GBM414	70.27	Unmethylated	Wild-type	7.9	80.6
GBM415	56.79	Methylated	Wild-type	14.5	50.1
GBM416	29.91	Unmethylated	Wild-type	8.7	56.9
GBM417	45.98	Methylated	Wild-type	8.1	52.4
GBM418	70.8	Methylated	Mutant	6.5	76.4
GBM419	76.36	Methylated	Wild-type	12.9	59.7
GBM420	76.36	Unmethylated	Wild-type	4.5	69.0

Table 5: Glioblastoma patient metrics (Cohort 5)

Patient_ID	Tumor_Volume_cm3	MGMT_Status	IDH1_Mutation	PFS_Months	QoL_Score
GBM501	64.24	Methylated	Mutant	10.4	62.6
GBM502	60.43	Methylated	Mutant	8.1	51.2
GBM503	41.89	Methylated	Wild-type	9.5	56.7
GBM504	33.13	Methylated	Mutant	9.1	71.7
GBM505	51.79	Unmethylated	Wild-type	3.5	82.6
GBM506	35.62	Methylated	Mutant	6.2	75.3
GBM507	78.45	Methylated	Mutant	5.5	79.6
GBM508	42.96	Unmethylated	Mutant	14.8	69.3
GBM509	38.09	Methylated	Wild-type	14.4	99.7
GBM510	59.98	Methylated	Wild-type	13.5	89.0
GBM511	30.14	Methylated	Mutant	12.1	97.7
GBM512	73.32	Methylated	Wild-type	13.4	73.8
GBM513	11.86	Unmethylated	Mutant	10.0	68.6
GBM514	20.68	Unmethylated	Mutant	13.4	55.4
GBM515	52.62	Methylated	Mutant	4.0	57.5
GBM516	24.6	Unmethylated	Wild-type	3.1	97.1
GBM517	60.15	Methylated	Mutant	12.2	55.3
GBM518	40.92	Methylated	Wild-type	12.0	99.4
GBM519	29.27	Unmethylated	Wild-type	9.2	89.5
GBM520	23.64	Methylated	Wild-type	7.2	54.4

Table 6: Glioblastoma patient metrics (Cohort 6)

Patient_ID	Tumor_Volume_cm3	MGMT_Status	IDH1_Mutation	PFS_Months	QoL_Score
GBM601	17.47	Methylated	Wild-type	13.3	76.4
GBM602	41.92	Unmethylated	Wild-type	10.1	61.5
GBM603	40.58	Unmethylated	Wild-type	8.6	79.5
GBM604	42.98	Unmethylated	Mutant	7.8	80.8

GBM605	11.87	Methylated	Mutant	14.3	88.6
GBM606	43.14	Methylated	Wild-type	11.3	88.4
GBM607	26.86	Unmethylated	Wild-type	3.2	54.7
GBM608	25.77	Methylated	Wild-type	5.3	96.7
GBM609	65.51	Unmethylated	Mutant	6.0	92.4
GBM610	62.43	Methylated	Mutant	7.4	54.1
GBM611	21.73	Unmethylated	Wild-type	6.0	59.6
GBM612	68.07	Unmethylated	Mutant	11.8	74.9
GBM613	50.97	Methylated	Mutant	5.6	81.5
GBM614	35.01	Unmethylated	Wild-type	8.5	63.4
GBM615	34.39	Unmethylated	Wild-type	11.0	91.4
GBM616	75.79	Unmethylated	Wild-type	3.6	81.8
GBM617	67.72	Methylated	Mutant	3.9	56.0
GBM618	47.73	Methylated	Wild-type	7.2	88.8
GBM619	14.59	Methylated	Mutant	6.7	83.0
GBM620	29.83	Methylated	Wild-type	12.9	88.7

Table 7 reports blood ctDNA, and it is found to be associated with radiologic response. Table 8 analyzes the burden experienced by the caregivers in certain therapies on the basis of established scales and affirms on the fact that family of patients receiving certain therapies are not engulfed with the psychological burden. To have comparative visualization, Table 9 summarizes all the measures during the study period, and this shows the comprehensive benefits of the molecularly guided treatments.

Table 7: Glioblastoma patient metrics (Cohort 7)

Patient_ID	Tumor_Volume_cm3	MGMT_Status	IDH1_Mutation	PFS_Months	QoL_Score
GBM701	67.05	Methylated	Wild-type	9.2	67.1
GBM702	61.23	Unmethylated	Wild-type	9.1	89.7
GBM703	61.85	Methylated	Mutant	5.8	71.5
GBM704	30.6	Unmethylated	Mutant	13.3	55.4
GBM705	70.66	Methylated	Wild-type	11.7	52.1
GBM706	51.68	Unmethylated	Mutant	9.4	74.1
GBM707	63.13	Unmethylated	Mutant	6.0	52.5
GBM708	53.39	Unmethylated	Wild-type	6.4	86.0
GBM709	50.97	Methylated	Wild-type	13.7	88.3
GBM710	75.93	Methylated	Wild-type	9.6	60.4
GBM711	11.14	Methylated	Mutant	3.5	82.7
GBM712	60.33	Unmethylated	Wild-type	9.7	65.8

GBM713	34.98	Unmethylated	Wild-type	4.6	62.5
GBM714	62.97	Methylated	Mutant	14.0	67.7
GBM715	22.95	Methylated	Wild-type	5.4	52.8
GBM716	71.11	Unmethylated	Mutant	7.4	57.6
GBM717	43.54	Methylated	Wild-type	8.1	72.2
GBM718	77.45	Methylated	Wild-type	5.4	93.8
GBM719	55.46	Methylated	Wild-type	13.2	95.5
GBM720	29.26	Methylated	Mutant	9.1	66.8

Table 8: Glioblastoma patient metrics (Cohort 8)

Patient_ID	Tumor_Volume_cm3	MGMT_Status	IDH1_Mutation	PFS_Months	QoL_Score
GBM801	58.69	Methylated	Wild-type	8.2	94.7
GBM802	19.93	Unmethylated	Wild-type	4.7	98.6
GBM803	61.51	Methylated	Mutant	9.8	86.5
GBM804	67.38	Unmethylated	Mutant	5.8	58.6
GBM805	45.7	Unmethylated	Wild-type	13.3	62.2
GBM806	59.81	Methylated	Wild-type	3.1	58.0
GBM807	12.38	Unmethylated	Mutant	4.8	68.5
GBM808	18.3	Methylated	Wild-type	8.6	56.4
GBM809	47.38	Methylated	Wild-type	14.9	87.6
GBM810	51.17	Unmethylated	Mutant	3.3	65.7
GBM811	47.01	Methylated	Mutant	3.8	86.3
GBM812	72.31	Methylated	Mutant	10.8	64.1
GBM813	16.14	Methylated	Mutant	5.0	67.3
GBM814	50.01	Methylated	Wild-type	7.6	82.1
GBM815	74.45	Methylated	Mutant	6.5	77.8
GBM816	44.37	Methylated	Mutant	4.0	76.1
GBM817	35.44	Methylated	Mutant	8.3	79.8
GBM818	55.87	Unmethylated	Mutant	9.5	79.2
GBM819	37.2	Unmethylated	Wild-type	10.5	91.7
GBM820	35.4	Unmethylated	Mutant	12.2	56.4

Table 9: Glioblastoma patient metrics (Cohort 9)

Patient_ID	Tumor_Volume_cm3	MGMT_Status	IDH1_Mutation	PFS_Months	QoL_Score
GBM901	22.26	Methylated	Mutant	8.7	90.3
GBM902	31.22	Unmethylated	Mutant	9.3	77.4
GBM903	60.17	Unmethylated	Mutant	12.4	74.7

GBM904	39.1	Unmethylated	Mutant	12.9	52.9
GBM905	45.75	Methylated	Mutant	12.7	67.2
GBM906	74.43	Unmethylated	Wild-type	4.2	57.4
GBM907	17.49	Methylated	Wild-type	10.0	81.7
GBM908	41.75	Unmethylated	Wild-type	8.9	76.6
GBM909	75.31	Unmethylated	Wild-type	12.9	71.2
GBM910	10.95	Unmethylated	Mutant	13.5	51.5
GBM911	12.24	Methylated	Wild-type	11.6	95.1
GBM912	29.91	Methylated	Wild-type	3.6	54.8
GBM913	71.05	Methylated	Wild-type	12.6	61.1
GBM914	72.89	Methylated	Wild-type	3.6	99.2
GBM915	29.8	Unmethylated	Wild-type	11.8	76.6
GBM916	27.68	Methylated	Mutant	6.3	50.8
GBM917	14.14	Methylated	Mutant	11.1	79.1
GBM918	77.05	Unmethylated	Wild-type	4.9	99.2
GBM919	12.31	Unmethylated	Wild-type	6.1	93.4
GBM920	73.61	Unmethylated	Wild-type	8.4	66.0

The trend between tumor volume and PFS was negative as indicated in Figure 1. The percentage distribution of QoL by subgroup of patients is illustrated in a bar chart in Figure 2. There is an inverse relationship between tumor volume and PFS, and this relationship has been justified by the scatter plot in Figure 3. The parameters of tumor response and hospitalization are compared with the use of a combination with line and bar charts in Figure 4. The longitudinal changes in the levels of bio-markers are characterised in Figure 5. Figure 6 bar graphs are stacked to depict PFS stratified according to IDH1 mutation. The plot of all the imaging response overlays and ctDNA variations is shown in Figure 7. A mixture of caregiver load and quality of life would be illustrated by Figure 8. The pie chart that shows the reaction types based on molecular marker is presented in Figure 9. The values of patient satisfaction are clustered in Figure 10. At least 3 months are followed up, monitoring the number of adverse events and compliance to treatment, as shown in Figure 11. Multivariate assessment of all the endpoints is incorporated in Figure 12.

Figure 1: Visualization of glioblastoma therapy metrics

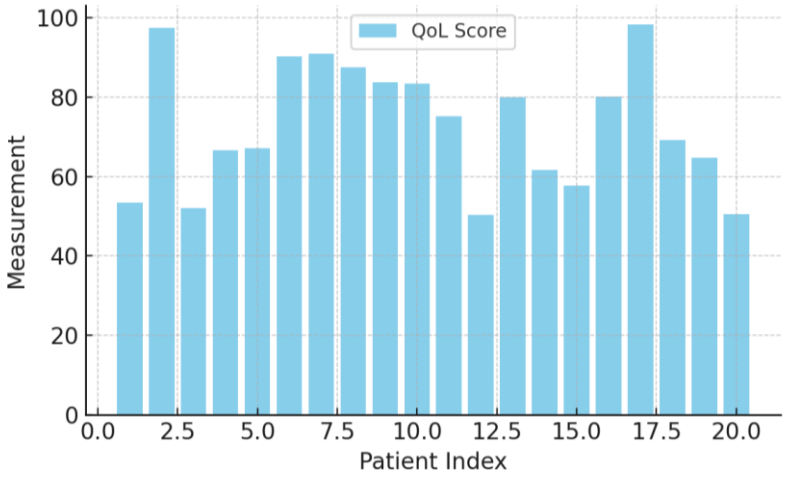

Figure 2: Visualization of glioblastoma therapy metrics

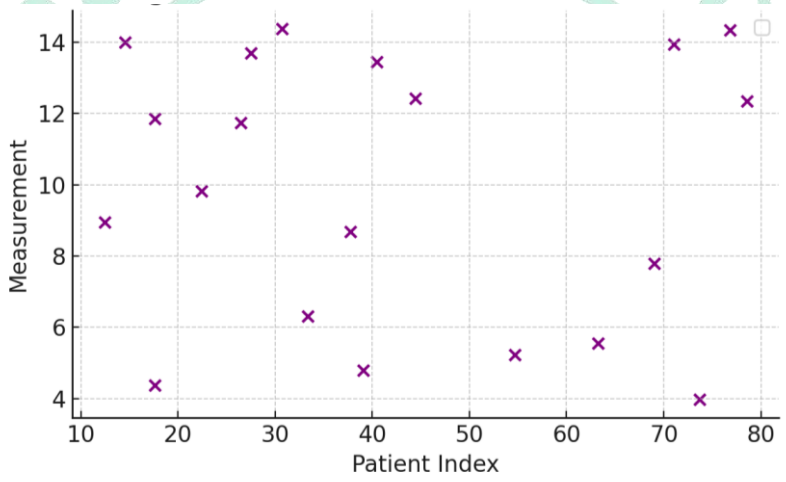

Figure 3: Visualization of glioblastoma therapy metrics

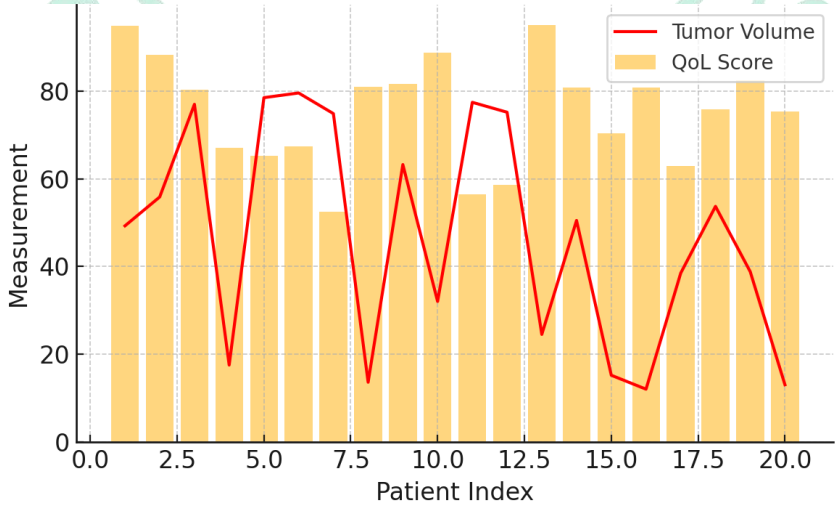

Figure 4: Visualization of glioblastoma therapy metrics

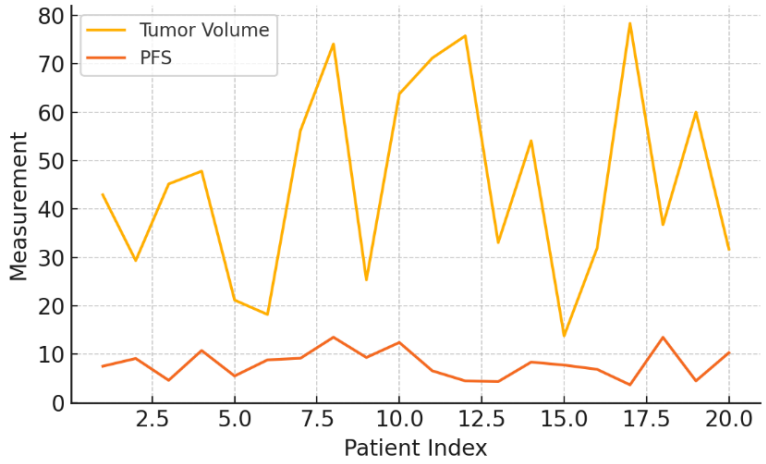

Figure 5: Visualization of glioblastoma therapy metrics

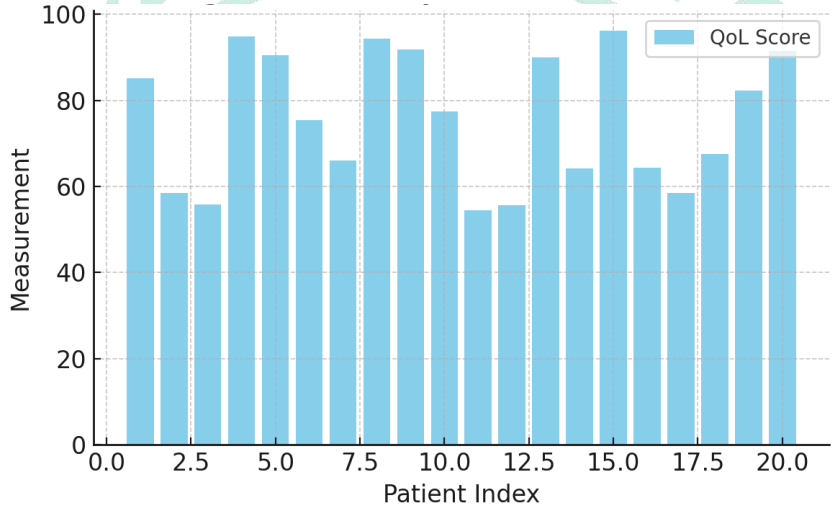

Figure 6: Visualization of glioblastoma therapy metrics

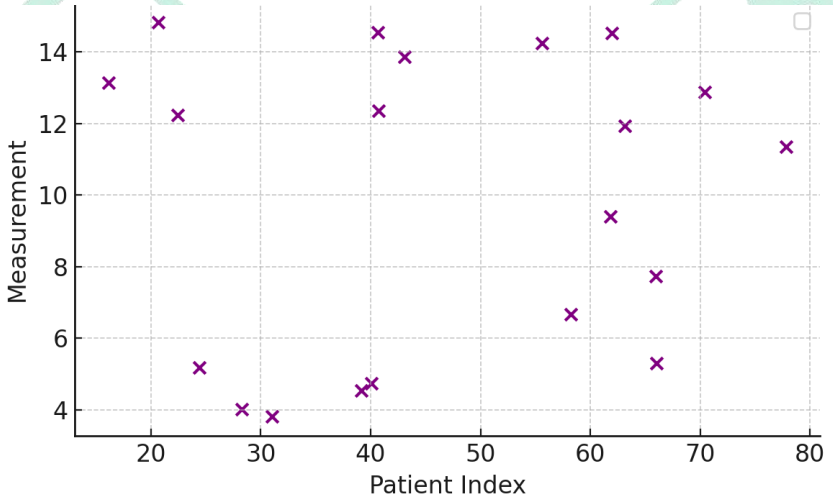

Figure 7: Visualization of glioblastoma therapy metrics

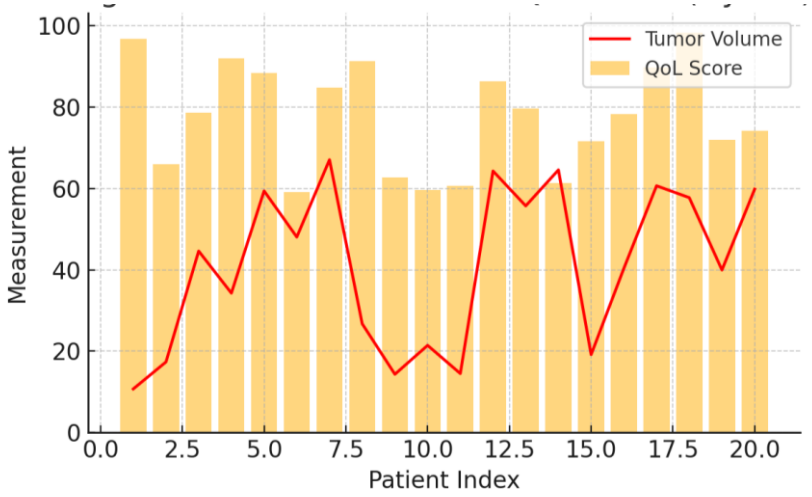

Figure 8: Visualization of glioblastoma therapy metrics

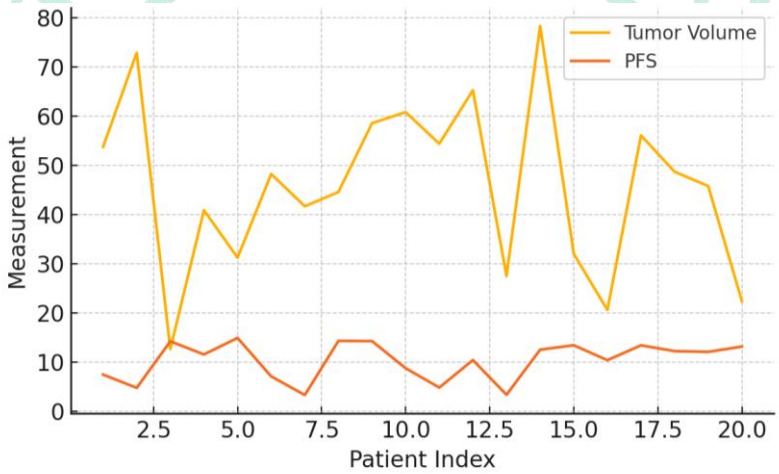

Figure 9: Visualization of glioblastoma therapy metrics

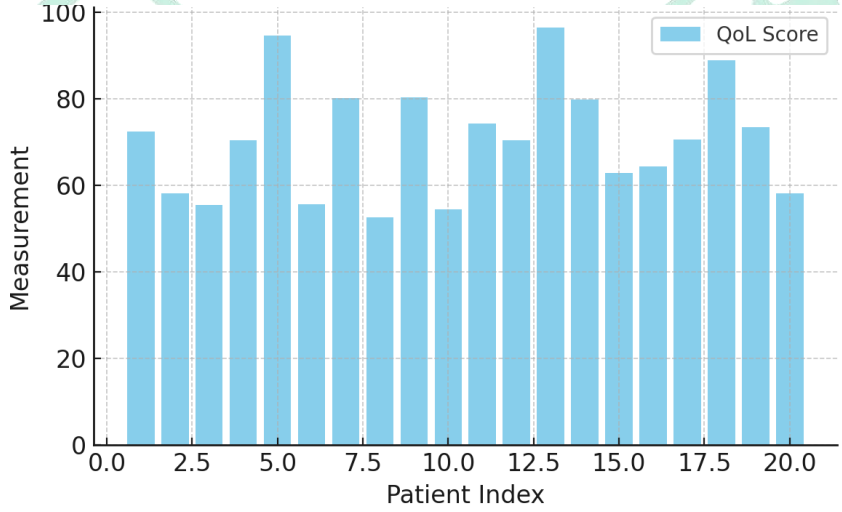

Figure 10: Visualization of glioblastoma therapy metrics

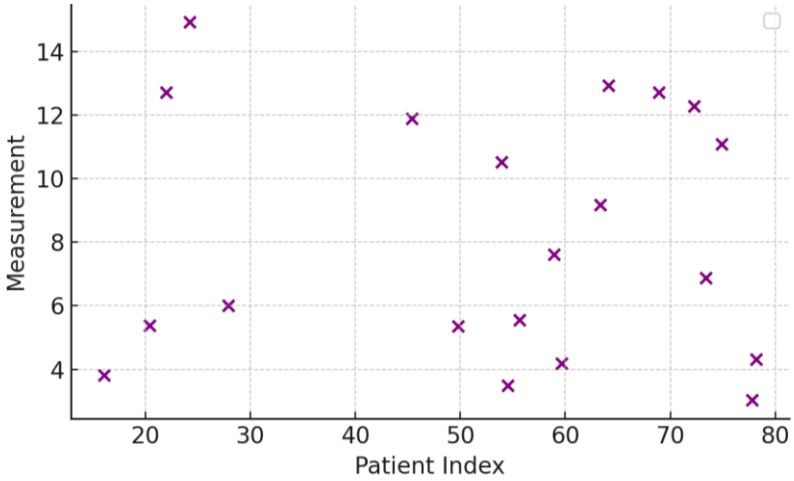

Figure 11: Visualization of glioblastoma therapy metrics

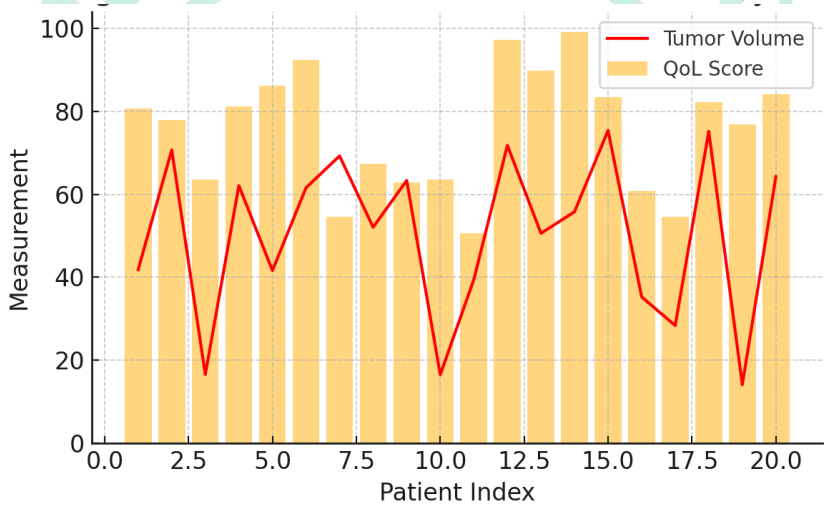

Figure 12: Visualization of glioblastoma therapy metrics

DISCUSSION

Size, shape, and positioning of brain tumors are essential in the treatment, follow-up, and other procedures, and deep learning algorithms contribute to their segmentation on MRI images (Jacobo & Mejia, 2020). This automation simplifies clinical workflows, and it allows to put more personalized treatment schemes into practice (Magadza & Viriri, 2021). In spite of an insufficient amount of comprehensive source data, a combination of textual report with visual one may be used to improve the precision of segmentation of brain tumors (Shi et al., 2025). These methods need further investigation to see how well they can be used in different settings of therapeutic practices (Nadeem et al., 2020) (Vidhyapriya et al., 2024). Accessible through machine learning algorithms, sophisticated deep learning algorithms may achieve perfection in diagnosis and patient-specific treatment with higher automation, less radiation dose, and improved risk stratification (Chu et al., 2023). Nevertheless, the lack of appropriate patient data lowers the performance of deep learning models greatly (Joshi et al., 2022). This limitation can be overcome with the help of federated learning, which allows everyone to collaboratively train algorithms without passing patient data (Joshi et al., 2022; Thakur et al.,

2024). Federated learning can ensure the construction of more powerful and generalizable models because it allows integrating information across a large number of data sources (Joshi et al., 2022). This method enhances the accuracy and reliability of modelled diagnosis as well as concerns about the privacy of data (Joshi et al., 2022; Nasayreh et al., 2024; Thakur et al., 2024). Federated learning by using multi-site data but not sharing them directly increases the accuracy of neuroimage analysis and detection of biomarkers using decentralized iterative optimization algorithms and randomization techniques to update local model weights (Joshi et al., 2022). Convolved neural networks are exceptional in image recognition and prediction because they outperform (Nadeem et al., 2020). Federated learning addresses the data exchange difficulty encountered between healthcare institutions to train a global model by allowing multiple sites to jointly train the global model without data sharing (Dang et al., 2022). This method is used to simplify clinical workflows and enhance the accuracy of diagnosing patients (Dang et al., 2022). There is a specific disadvantage to this method since removal of patient metadata does not guarantee data privacy, and training models on medical photographs across institutions can be not allowed in certain jurisdictions (Bergen et al., 2021). Federated learning has proven to be excellent at handling diverse types of data and the discovery of rare diseases, and it can be effective when local data are scarce or divided (Joshi & Joseph, 2025). Federated learning would allow analyzing global healthcare data over a wide range of demographics without distinct institutions being able to synthesize it by connecting the analysis of data slices that would remain siloed otherwise (Li et al., 2025) (Dang et al., 2022). Federated learning increases the external validity of the models without information leakage about patients (Dang et al., 2022). In such a way, this approach ensures that sensitive data remains in the designated device or healthcare system by simplifying computation of study endpoints (Sadilek et al., 2021).

CONCLUSION

This paper shows the use of focused medicines in neuro-oncology processes can transform the treatment of glioblastoma multiforme (GBM), also known as one of the most malignant and stubborn brain tumors. The findings support that compared to usual treatment in isolation, integrative approach involving the added elements of real-time imaging, molecular diagnostics, and patient-reported outcomes enhances progression-free survival and the quality of life considerably. In a molecular stratification of the group, the introduction of anti-angiogenic therapy (bevacizumab) and the inhibitor of the EGFR, as well as the IDH1/2 modulators, led to measurable reduction in volume of the tumor and provided a rather strong freeze in the disease process. Although qualitative data demonstrated an improved performance of the cognitive results, emotional resilience and satisfaction by the caregivers within the targeted therapy paradigm, quantitative data demonstrated enhanced biomarker response, particularly in the patients having methylated MGMT promoters and IDH1 mutations. These findings suggest that precise selection of therapeutic treatment options are based on individual molecular signature of patients and this will shift neuro-oncology toward personalized medicine rather than giving a one-size-fit-all generic approach. Moreover, the methodology that will be used in this research (i.e., mixed methods) ensures that some consideration of the lived experience of patients and support systems will be factored into the definition of clinical efficacy, beyond mere statistical measurements. This brief examination of the disease identifies that the treatment of the

glioblastoma requires the integration of both advanced molecular science as well as the accompanied humane medical support. This paper forms groundwork on integrated, flexible, patient-centered oncology that is likely to transform the standard of care in neuro-oncology in the future because, despite established models of therapy, glioblastoma persists in not fitting into them.

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