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The effects of new or worsened postoperative neurological deficits on survival of patients with glioblastoma

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OBJECTIVE An increased extent of resection (EOR) has been shown to improve overall survival of patients with glioblastoma (GBM) but has the potential for causing a new postoperative neurological deficit. To investigate the impact of surgical neurological morbidity on survival, the authors performed a retrospective analysis of the clinical data from patients with GBM to quantify the impact of a new neurological deficit on the survival benefit achieved with an increased EOR.

METHODS The data from all GBM patients who underwent resection at the University of Florida from 2010 to 2015 with postoperative imaging within 72 hours of surgery were included in the study. Retrospective analysis was performed on clinical outcomes and tumor volumes determined on postoperative and follow-up imaging examinations.

RESULTS Overall, 115 patients met the inclusion criteria for the study. Tumor volume at the time of presentation was a median of 59 cm^3 (enhanced on T1-weighted MRI scans). The mean EOR (± SD) was 94.2% ± 8.7% (range 59.9%–100%). Almost 30% of patients had a new postoperative neurological deficit, including motor weakness, sensory deficits, language difficulty, visual deficits, confusion, and ataxia. The neurological deficits had resolved in 41% of these patients on subsequent follow-up examinations. The median overall survival was 13.1 months (95% CI 10.9–15.2 months). Using a multipredictor Cox model, the authors observed that increased EOR was associated with improved survival except for patients with smaller tumor volumes (≤ 15 cm^3). A residual volume of 2.5 cm^3 or less predicted a favorable overall survival. Developing a postoperative neurological deficit significantly affected survival (9.2 months compared with 14.7 months, p = 0.02), even if the neurological deficit had resolved by the first follow-up. However, there was a trend of improved survival among patients with resolution of a neurological deficit by the first follow-up compared with patients with a permanent neurological deficit. Any survival benefit from achieving a 95% EOR was abrogated by the development of a new neurological deficit postoperatively.

CONCLUSIONS Developing a new neurological deficit after resection of GBM is associated with a decrease in overall survival. A careful balance between EOR and neurological compromise needs to be taken into account to reduce the likelihood of neurological morbidity from surgery.

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KEY WORDS neurological deficit; postoperative morbidity; glioblastoma; resection; extent of resection; quality of life; oncology

Glioblastoma (GBM) is a highly malignant tumor that results in significant neurological morbidity and very limited survival despite aggressive and often toxic interventions, including resection, chemotherapy, and radiotherapy. Since GBM is prototypically characterized by invasion into normal brain tissue,[9,34,37] curative resection is nearly impossible. This phenomenon was most dramatically demonstrated by Dandy's well-known description of the failure of hemispherectomy to cure GBM.[9] Although not curative, resection has been reported to significantly affect patient outcomes.[22] Specifically, an increased extent of resection (EOR) has been shown to improve overall survival.[4,24,33] Volumetric analysis of residual tumor on postoperative cranial images has shown

ABBREVIATIONS EOR = extent of resection; GBM = glioblastoma; HR = hazard ratio; HRQOL = health-related quality of life; iMRI = intraoperative MRI; KPS = Karnofsky Performance Scale; RV = residual volume.

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that both EOR and residual volume (RV) are important predictors of overall survival.[6,12] These findings have led to retrospective analyses[25] demonstrating benefits from supratotal resections[11] or from resecting the tumor beyond its enhancing portions.

These previous results support an aggressive surgical approach to treat patients with GBM. Given current tools, such as fluorescence-guided resection,[2,10] intraoperative awake mapping,[3,13,15,35] and intraoperative MRI,[28,39] achieving a gross-total or supratotal resection has become more feasible. However, devastating neurological deficits remain potential risks. In GBM, poor functional performance status (as measured with the Karnofsky Performance Scale [KPS]) is a strong predictor of a poor outcome,[31] and postoperative neurological deficits are associated with decreased overall survival.[5] Therefore, neurosurgeons are tasked with walking the tightrope of achieving maximal resection while preserving neurological function. To further investigate the impact of surgery-associated neurological morbidity on survival, we performed a retrospective analysis of surgery outcomes among GBM patients to quantify the effect of new neurological deficits on survival outcomes achieved with increased EOR.

Methods

Patient Selection

After obtaining approval by our internal review board, we searched the billing database at the University of Florida for the records of patients who had a craniotomy for glioma resection from 2010 to 2015. All GBM patients with postoperative cranial imaging within 72 hours of surgery were included in the present study. Patients who underwent a biopsy only were not included. The patients' electronic medical records were used to collect clinical data.

Data Acquisition

The patients' preoperative and postoperative imaging results were reviewed by 2 of the authors (J.A. and E.K.D.), and tumor volumes were manually measured with the Varian Eclipse treatment planning system. Interobserver reliability of the study observers was assessed by comparing measurements made by the 2 observers on both preoperative and postoperative scans. Between-patient and residual variances were assessed with a linear mixed model and used to estimate the intraclass correlation coefficient, which is the proportion of variation in measurements not introduced by the observers. Interobserver reliability was excellent, indicated by intraclass correlation coefficients of 99.2%, 99.8%, and 99.5% for measurements of tumor volume, RV, and EOR, respectively. The mean interobserver bias was negligible, indicated by −0.6 cm^3 for volume, −0.2 cm^3 for RV, and 0.5% for EOR.

Statistical Analysis

All statistical analyses were performed with SAS (version 9.4, SAS Institute). We used Kaplan-Meier estimation[14] to generate stratum-specific survival curves and estimates of median length of overall survival with 95% CIs. We used Cox proportional hazards regression[14] to develop a multipredictor model that could be used to assess the simultaneous influence of identifiable risk factors on overall survival. We initially considered age at surgery, KPS score at surgery, tumor location (frontal, temporal, parietal, occipital, or other), tumor in an eloquent location (i.e., in motor, sensory, visual, or language cortex or in the basal ganglia, or with extension into the brainstem), presurgical tumor volume, postsurgical RV, EOR, presence of a preexisting neurological deficit, and presence of a new neurological deficit immediately postsurgery.

To avoid overfitting, we used backward elimination with a p-to-remove value of greater than 0.20 to eliminate nonsignificant risk factors. We also evaluated the statistical significance of pairwise interactions between risk factors that remained in the model. Our final model included age, new postoperative neurological deficit, interaction between age and neurological deficit, tumor volume, EOR, and interaction between tumor volume and EOR. Because of the interdependence of tumor volume, RV, and EOR, we were able to estimate the effect of RV on overall survival in our final model as a linear function of the effects of volume and the interaction between volume and EOR. We used our final Cox regression model to estimate risk factor hazard ratios (HRs) with 95% CIs and constructed curves predicting overall survival for patients with various combinations of selected risk factor values.

We also used the Kaplan-Meier analysis with Cox regression to assess overall survival in patient groups defined by EOR cut-points and by resolution status of new postoperative neurological deficits (resolved or unresolved by the first follow-up). We used logistic regression to estimate odds ratios of risk factors for the occurrence of a new postoperative neurological deficit.

Results

Baseline Patient Characteristics

Overall, 115 patients met the inclusion criteria for the current study. The baseline patient characteristics are shown in Table 1. We also compared the characteristics determined within 72 hours of surgery of the included patients with those of 46 patients who were excluded because of a lack of imaging data and found that both patient groups were comparable in these characteristics (Table 2), except for slightly higher KPS scores and a lower likelihood of having a new postoperative deficit among the excluded patients. The median age of the included patients was 61 years, and 61.7% of the patients were men (Table 1). The median preoperative KPS score was 80, and 26% of patients initially presented with a seizure. The median length of follow-up was 9.8 months (mean 13.3 months, range 0.07–83.2 months); 73 patients died during the follow-up period, and 29 were censored at the last follow-up.

Imaging results indicated that 13% of the patients had tumors crossing the midline, 87.7% had peritumoral edema, and 27% had tumors involving eloquent structures (11% in the motor cortex and 5% in the language cortex). Overall, 12% of the patients had surgery involving intraoperative cortical mapping or fluorescein-guided microscopic resection. The median tumor-enhanced volume on T1-weighted MRI scans at presentation was 36.5 cm^3 and was 38.0 cm^3 on FLAIR images. The median length of

TABLE 1. Baseline patient characteristics

Variable	Value (median [range])*
Mean age in yrs ± SD	57.2 ± 14.1 (61 [18–80])
No. of men	61.7
Mean preop KPS score ± SD	72.5 ± 16.3 (80 [20–90])
Seizure on presentation	26.1
Peritumoral edema	87.7
Tumor location	
Crossing midline	13.0
Eloquent area	27.0
Frontal	50.4
Temporal	40.0
Parietal	29.6
Occipital	12.2
Insular	2.6
Basal ganglia	0.9
Cerebellum	0.9
Presented w/ neurological deficit	67.0
Presented w/ speech/motor deficit	47.8
Mean hospital LOS in days ± SD	6.9 ± 5.2 (6 [1–31])
Mean enhanced tumor vol in cm^3 ± SD	42.9 ± 30.8 (36.5 [1–161])
Mean EOR in % ± SD	94.2 ± 8.7 (97.7 [59.9–100])
Mean RV in cm^3 ± SD	2.1 ± 3.4 (0.9 [0–19])

LOS = length of stay.
* Data represent percentages, unless indicated otherwise.

time in the operating room was 169.5 minutes, the median length of stay in the hospital was 6 days, and the mean EOR (± SD) was 94.2% ± 8.7% (range 59.9%–100%). Most patients were discharged to home (68.7%) or rehabilitation (24.4%).

EOR and RV

The median duration of overall survival was 13.1 months (95% CI 10.9–15.2 months). An EOR increase from less than 80% to 100% increased survival from 8.5 months to 15 months (Table 3). A multipredictor Cox model confirmed that an increased EOR was significantly associated with improved survival, except for smaller tumor volumes (15 cm^3) (Fig. 1). In this model, the HR of death had an inverse relationship with increased EOR, and differences in HRs between EORs of 99% and 100% were statistically significant ($p \leq 0.01$) (Table 4). As previous studies have shown, KPS score and age also were significant predictors for survival. In addition, RV significantly affected survival (Table 5); for example, an RV of 2.5 cm^3 or lower predicted favorable overall survival (Fig. 2).

Neurological Deficits

Almost 30% of the patients had a new or worsened postoperative neurological deficit; these deficits included motor weakness, sensory deficits, language difficulty, visual deficits, confusion, and ataxia. In a more detailed analysis of these deficits, we included even mild deficits (e.g., a 1-point decrease in strength). In this cohort, 20% of patients had new or worsened motor or speech deficits, and 10% had other deficits (Table 2). The median lengths of survival of those with no new deficit, with a motor or speech deficit, or with any other deficit were 14.7 months, 8.3 months, and 8.6 months, respectively ($p = 0.03$). In 41% of the patients with deficits, these deficits had resolved on subsequent follow-up examinations. Patients who developed a new neurological deficit postoperatively did not significantly differ from those without a new deficit in preoperative patient demographics and tumor characteristics (Table 6).

TABLE 2. Characteristics of patients who were included in the present study versus those excluded because of lack of imaging*

Variable	Patients		p Value†
	Included (n = 115)	Excluded (n = 46)	
Mean age in yrs	57.2	61.2	0.08
Mean KPS score	72.5	78.0	0.02
Enhanced tumor vol in cm^3	42.9	51.1	0.25
Proportion of patients in %			
Tumor in eloquent location	27.0	34.8	0.34
Peritumoral edema	87.7	95.7	0.16
Tumor crossing midline	13.2	19.6	0.33
Midline shift	54.8	39.1	0.08
Preop neurological deficit	67.0	78.3	0.18
New/worsened postop neurological deficit	29.6	13.0	0.04

* The characteristics were assessed within 72 hours of surgery.
† p values < 0.05 were considered statistically significant.

Patients with GBM in an eloquent location were more likely to have a motor or speech deficit postoperatively than were patients with tumors in noneloquent areas, and the patients with tumors in noneloquent areas were more likely to have a deficit characterized as other. Tumor location and involvement of the eloquent cortex did not affect survival, except that patients with a tumor in the parietal lobe had better survival than those with tumors in other locations (Table 7). Having an existing preoperative deficit also did not affect survival. Patients with an existing preoperative neurological deficit had a median length of survival of 12.4 months and those without a deficit of 13.9 months ($p = 0.89$). Other potential confounding factors, such as receiving subsequent temozolomide and radia-

TABLE 3. Relationship between EOR and survival determined with the Cox regression model

EOR (%)	Survival in Mos (95% CI)
≤80	8.5 (1.2–8.7)
≤90	8.7 (5.7–14.7)
≤95	10.9 (7.0–13.9)
≤98	12.4 (8.0–13.9)
≤99	12.4 (8.4–14.7)
≤100	13.1 (8.5–14.9)
100*	15.0 (9.4–21.4)

* This category includes only patients with 100% EOR.

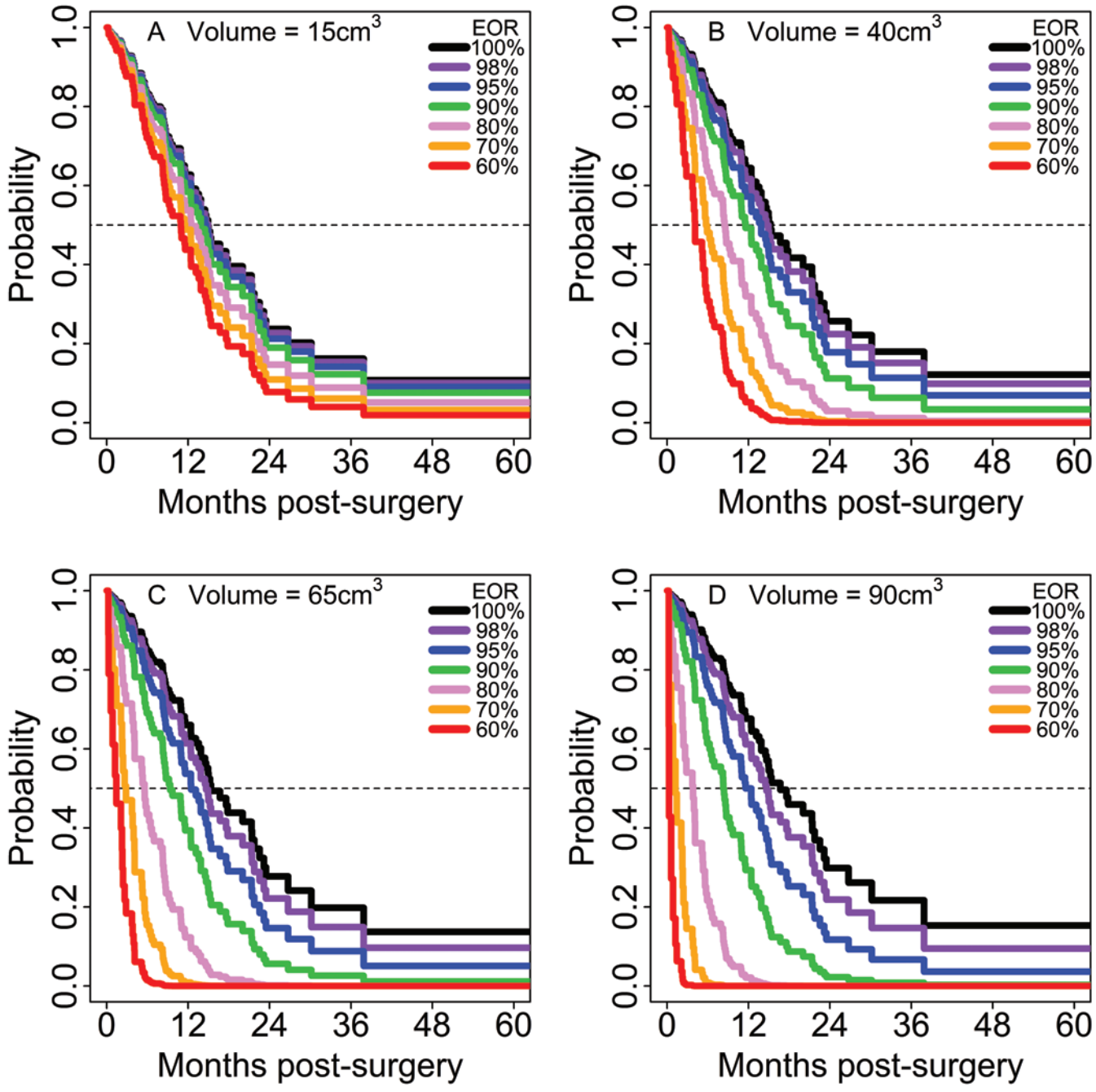

FIG. 1. Predicted overall survival by EOR and preoperative tumor volume. Predicted survival curves from left to right indicate the effect of increasing EOR (by 60%, 70%, 80%, 90%, 95%, 98%, or 100%) on the length of survival for preoperative tumor volumes of 15 cm^3 (**A**), 40 cm^3 (**B**), 65 cm^3 (**C**), and 90 cm^3 (**D**).

tion treatments, did not differ between patients with a new postoperative deficit and those without a new postoperative deficit.

Development of a postoperative neurological deficit significantly affected survival. The median overall survival for patients with a new postoperative neurological deficit was 9.2 months compared with 14.7 months among those without a new postoperative deficit ($p = 0.02$). Any survival benefit from an EOR of 95% or more was abrogated by the development of a new postoperative neurological deficit (Fig. 3). Moreover, there was a trend toward improved survival among patients whose neurological deficit resolved by the first follow-up (1–2 months after surgery) compared with those whose deficit persisted ($p = 0.09$). (Fig. 4). The patients whose neurological deficit resolved tended to have improved survival even compared with the patients with no neurological deficit ($p = 0.05$). We also noted that age influenced the effects of new postoperative neurological deficits on survival. Younger patients (< 50 years) with a new postoperative neurological deficit did

TABLE 4. Hazard ratios of death based on preoperative tumor volume and EOR

EOR (%)	HR (95% CI)	p Value
Tumor vol of 15 cm^3		
≥60	1.77 (0.44–7.23)	0.424
≥70	1.54 (0.54–4.41)	0.424
≥80	1.33 (0.66–2.69)	0.424
≥90	1.15 (0.81–1.64)	0.424
≥95	1.07 (0.90–1.28)	0.424
≥96	1.06 (0.92–1.22)	0.424
≥97	1.04 (0.94–1.16)	0.424
≥98	1.03 (0.96–1.10)	0.424
≥99	1.01 (0.98–1.05)	0.424
Tumor vol of 40 cm^3		
≥60	6.72 (1.90–23.8)	0.003
≥70	4.18 (1.62–10.8)	0.003
≥80	2.59 (1.38–4.88)	0.003
≥90	1.61 (1.17–2.21)	0.003
≥95	1.27 (1.08–1.49)	0.003
≥96	1.21 (1.07–1.37)	0.003
≥97	1.15 (1.05–1.27)	0.003
≥98	1.10 (1.03–1.17)	0.003
≥99	1.05 (1.02–1.08)	0.003
Tumor vol of 65 cm^3		
≥60	25.5 (2.5–258.4)	0.006
≥70	11.4 (2.0–64.5)	0.006
≥80	5.05 (1.59–16.1)	0.006
≥90	2.25 (1.26–4.01)	0.006
≥95	1.50 (1.12–2.00)	0.006
≥96	1.38 (1.10–1.74)	0.006
≥97	1.28 (1.07–1.52)	0.006
≥98	1.18 (1.05–1.32)	0.006
≥99	1.08 (1.02–1.15)	0.006
Tumor vol of 90 cm^3		
≥60	96.6 (2.5–3698.4)	0.014
≥70	30.8 (2.0–474.2)	0.014
≥80	9.83 (1.59–60.8)	0.014
≥90	3.14 (1.26–7.80)	0.014
≥95	1.77 (1.12–2.79)	0.014
≥96	1.58 (1.10–2.27)	0.014
≥97	1.41 (1.07–1.85)	0.014
≥98	1.26 (1.05–1.51)	0.014
≥99	1.12 (1.02–1.23)	0.014

not have a detectable decrease in survival (Fig. 5). Patients 60 years or older had up to 8.8 times the risk for death if they developed a new postoperative neurological deficit ($p < 0.01$). For each 10 years of increase in age in the absence of a new neurological deficit, the HR for death was 1.25 ($p = 0.04$). In contrast, for each 10 years of age increase with a neurological deficit, the HR for death was 2.41 ($p = 0.0001$).

TABLE 5. RV and predicted survival according to the Cox regression model

RV in cm^3	Survival in Mos (95% CI)
>10.0	8.0 (1.2–8.5)
>2.5	8.7 (7.0–14.9)
>0.75	12.5 (8.3–14.7)
>0	13.1 (8.5–15.2)
0	15.0 (9.4–21.4)

Discussion

In the present study, we confirmed previous findings that increased EOR and decreased RV are both associated with increased survival in patients undergoing resection for GBM. Our results also indicated that development of a permanent neurological deficit postoperatively reduced survival time. Patients whose deficits were only temporary tended to have improved survival.

Because multiple retrospective analyses have reported that near-total resections of GBM improve survival, aggressive surgery remains a critical part of the standard of care for patients with GBM. Previous reports have focused on comparing patients who underwent resections versus those who had only a biopsy,[8,21,29] but they have yielded mixed findings due to crude definitions of EOR (i.e., partial, subtotal, and gross-total resection), reliability of EOR reporting, variable postoperative imaging modalities, and timing of the imaging. More definitive reports with standardized EOR evaluations have appeared in the early 2000s. These studies have reported a clear survival benefit from resection versus biopsy in analyses accounting for patients with poor prognostic factors who were overrepresented in biopsy groups.[24] In another large retrospective

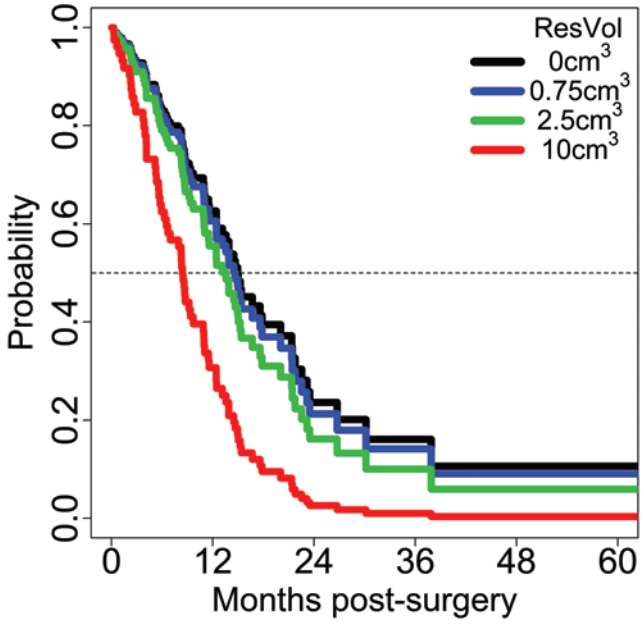

FIG. 2. Predicted overall survival by RV (ResVol). Predicted survival curves from left to right show the effect of decreasing RV (i.e., 10, 2.5, 0.75, or 0 cm^3) on the length of survival.

TABLE 6. Baseline characteristics of patients with or without a new postoperative neurological deficit

Variable	Neurological Deficit (SD)		p Value
	No	Yes	
Mean age in years	57.6 (14.7)	56.8 (12.9)	0.63
Mean KPS score	74.6 (13.6)	67.7 (20.9)	0.23
Mean preop tumor vol in cm^3	45.2 (31.8)	37.4 (27.8)	0.25
Pts w/ tumor in eloquent location in %	27.2	26.5	0.94
Mean EOR in %	93.8 (9.0)	95.2 (8.1)	0.19
Mean RV in cm^3	2.3 (3.3)	1.7 (3.5)	0.10

Pts = patients.

analysis, Lacroix et al. reported improved survival among GBM patients with an EOR of 98% or greater.[22] These results were subsequently confirmed with volumetric tumor analyses by other groups,[4,6,33] and these analyses demonstrated a survival benefit of GBM patients with an EOR of 70% or greater.[6]

Although important, EOR does not fully address how tumor burdens affect patient outcomes. The tumor burden present at the time of a radiochemotherapy is an important factor for overall outcomes among patients with malignant glioma.[1] Recent findings indicate that RVs of 2–3 cm^3 or less predict improved overall survival after GBM resection.[6,12] Results of a large retrospective analysis have also shown that EOR and RV are independent predictors of overall survival, as are age and KPS score.[22] Also, EOR affects survival only at larger tumor volumes, confirming that RV is the key factor influencing treatment outcomes for GBM. Since aggressive resection is essential for maximal GBM management, caution against provoking or exacerbating a neurological deficit is paramount.

Compared with the extensive literature on the benefits of GBM resection, reports on the impact of neurological deficits on glioma outcomes are sparse. This paucity in the literature is likely due to the reluctance of surgeons to report complications and due to difficulties in retrospective analyses of obtaining long-term neurological assessments from patients. Neurological morbidity rates after GBM surgery have been reported to be as high as 22%, with higher risks among patients receiving subsequent craniotomies.[7,17] Moreover, medical complications within 30 days of malignant glioma surgery increase the risk for death 1.9-fold.[20] McGirt et al., conducting a large retrospective analysis of outcomes among GBM patients, noted decreased survival (of approximately 9 months) among patients with motor or language deficits compared with those without new postoperative deficits (12.8 months survival).[26]

TABLE 7. Length of survival by tumor location

Tumor Location	Median Length of Survival (mos)	p Value
Not parietal	12.4	0.03
Parietal	16.7	
Noneloquent area	12.4	0.40
Eloquent area	13.9	

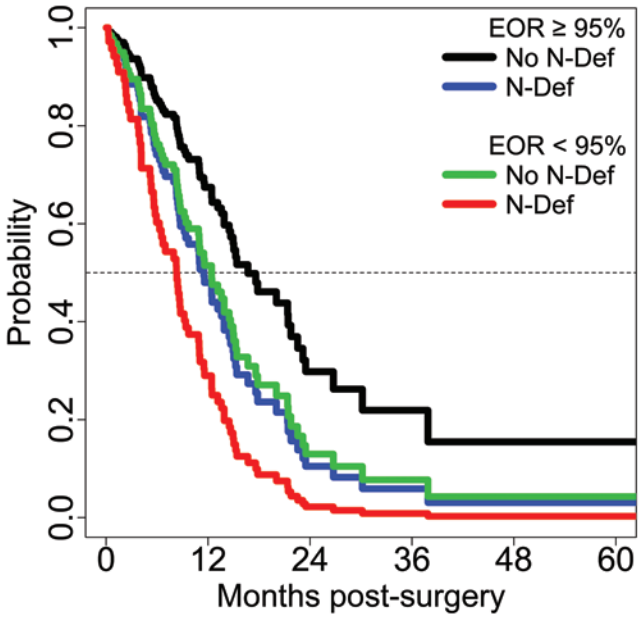

FIG. 3. Predicted overall survival by EOR and presence of a new postoperative neurological deficit (N-Def). Patients who had an EOR of ≥ 95% with a postoperative neurological deficit had very similar survival as patients who had an EOR of ≤ 95% without a postoperative neurological deficit.

Our results demonstrate that permanent neurological deficits increase the overall GBM-related mortality rate. In a subgroup analysis, patients 50 years or younger with a new postoperative neurological deficit did not have decreased survival; however, older patients with favorable EOR (> 98%) had lower survival if new postsurgical neurological deficits were present. Therefore, avoidance of neurological deficits is especially important for patients

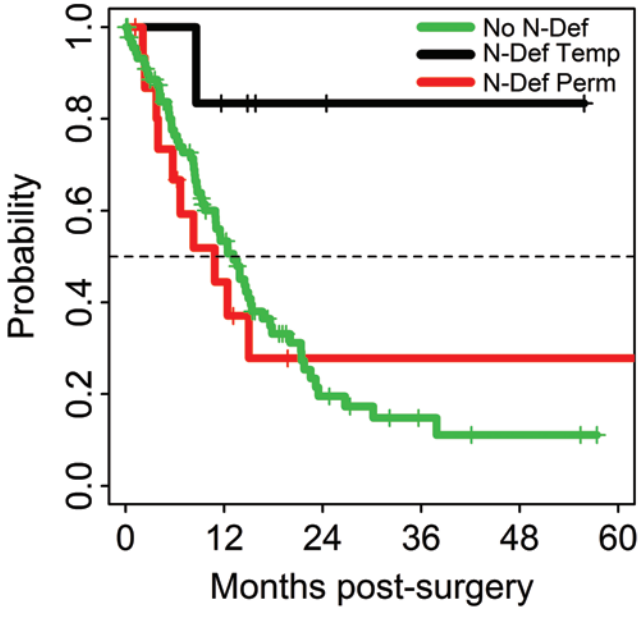

FIG. 4. Predicted overall survival in GBM patients with a permanent (Perm) postoperative neurological deficit, temporary (Temp) postoperative neurological deficit, or no new postoperative neurological deficit.

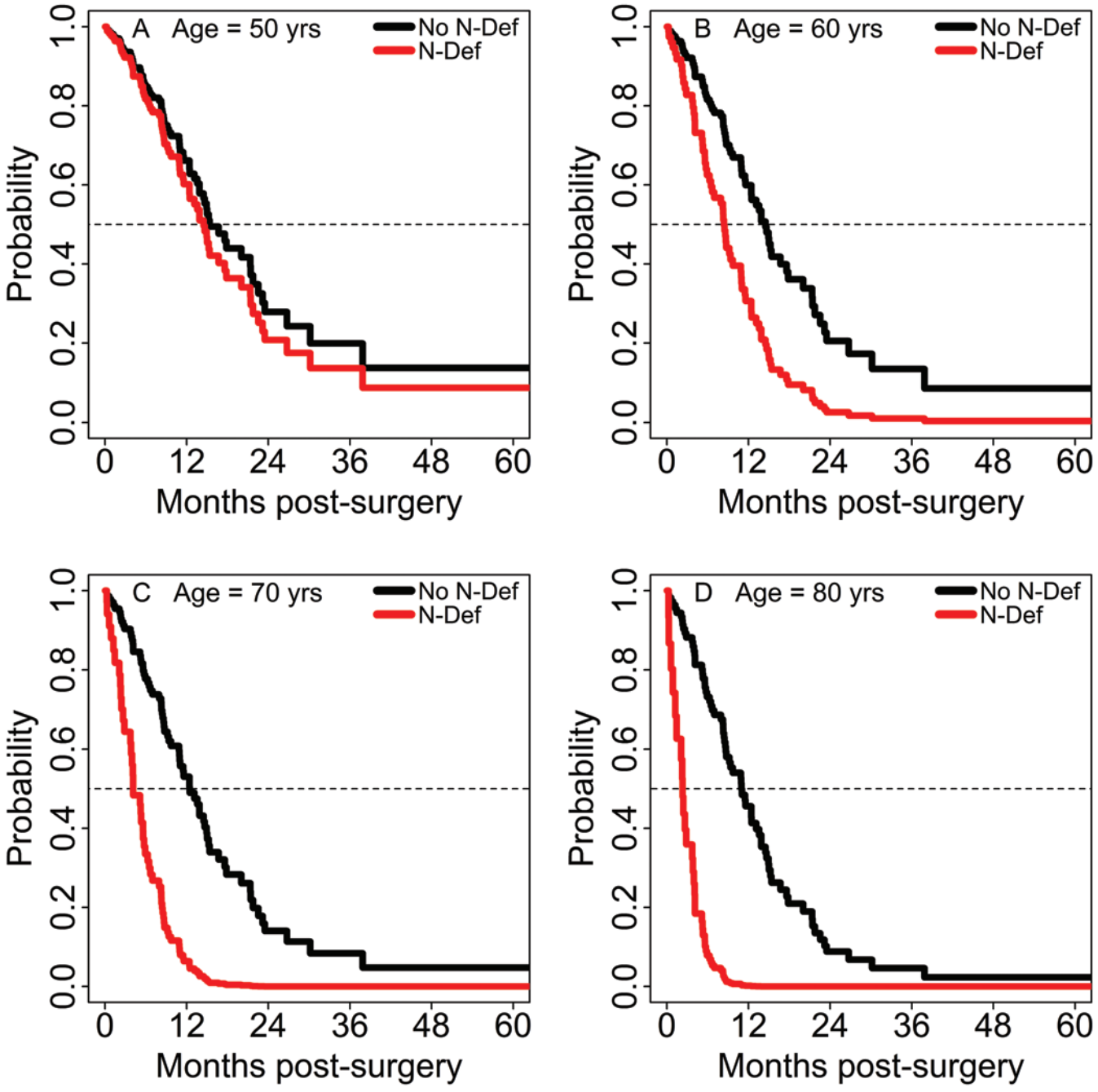

FIG. 5. Predicted overall survival according to age 50 years (**A**), 60 years (**B**), 70 years (**C**), and 80 years (**D**) among patients with or without a new postoperative neurological deficit.

60 years or older. An analysis of all patients with a new postoperative deficit indicated that the survival of these patients was decreased. However, the patients whose deficit was only temporary tended to have improved overall survival. Our study was too underpowered to determine whether this improved survival was statistically significant. However, this trend toward improvement after a temporary worsening of neurological function gives credence to the concept that maximal resection improves survival of GBM patients.

Several tools are now available to increase the probability of achieving a more aggressive resection in GBM patients, but their impact on avoiding neurological deficits is less well established. The most obvious tool for reducing the likelihood of a neurological deficit postoperatively is awake cortical mapping. Kim et al. reported their experience with 309 brain tumor patients who underwent awake cortical mapping during surgery. The authors observed that in cases in which the intraoperative mapping indicated tumor involvement in the eloquent cortex, 21% had worsened neurological deficits postoperatively compared with only 9% of those for whom the mapping indicated no such

involvement.[19] The deficits in most of these patients had improved by the next follow-up. These results most likely indicate the high-risk nature of tumors involving eloquent areas of the brain for causing neurological deficits. Because of the selection bias in retrospective studies evaluating the impact of cortical mapping on neurological outcomes postoperatively, it is difficult to determine the true benefit of the mapping when lesions in the eloquent cortex are resected. Other experienced surgeons have reported that the risk for a permanent neurological deficit after resection involving cortical mapping of tumors in eloquent areas ranges from 1.6%[32] to 4.5%.[38]

Retrospective studies have reported that intraoperative MRI (iMRI) can improve EOR in malignant glioma surgery.[3,30,36] Olubiyi and colleagues reported that the rate of gross-total resection was 49.3% in GBM patients with iMRI-guided surgery compared with 21.4% in those without iMRI.[28] These authors also noted fewer new neurological deficits (6.7%) among GBM patients who underwent iMRI resections than among those undergoing surgery without iMRI (13.8%); however, this difference did not reach statistical significance. Other novel tools, such as fluorescence-guided resection of recurrent malignant gliomas, have not reduced the likelihood of postsurgical neurological deficits.[16,23,27] Although iMRI and tumor fluorescence are useful intraoperative tools, they have not yet been definitively shown to significantly reduce the risks for postsurgical neurological deficits.

The most important consideration in our study is the impact of neurological deficits on quality of life. Several factors may affect health-related quality of life (HRQOL) among patients with GBM, including age, tumor location, tumor size, preoperative neurological deficits, seizures, and adverse effects of treatments. Development of a new neurological deficit postoperatively is expected to result in a drop in reported HRQOL measures. Importantly, quality of life also affects survival. Jakola et al., analyzing HRQOL data collected before surgery and 6 weeks after surgery from 61 patients with GBM, observed that a significant predictor for poor survival was postoperative deterioration of HRQOL (HR 2.02, $p = 0.045$).[18] Therefore, avoiding a new neurological deficit postoperatively positively influences both the quality and the length of the remaining life of patients with GBM.

Our data analysis was limited by its retrospective nature and the use of data available from electronic medical records. Long-term neurological outcomes were unavailable for any of the patients lost to follow-up. Our analysis also excluded patients without postoperative imaging within 72 hours, and this exclusion introduced a selection bias. However, the results in the present study confirm those of previous investigations showing survival benefits from increased EOR and reduced RV. Importantly, a new neurological deficit abrogates potential survival benefits due to an aggressive EOR in older patients.

Conclusions

Development of a new permanent neurological deficit after GBM resection was associated with a decrease in overall survival. Patients with only a temporary neurological worsening after resection tended to have improved survival compared with all other patient groups. Tools such as awake cortical mapping, fluorescence-guided surgery, and iMRI may be helpful to maximize resection, but their use has not yet been shown to reduce the likelihood of postoperative neurological deficits. Future studies that prospectively analyze surgical morbidity rates among GBM patients are needed.

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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Conception and design: Rahman. Acquisition of data: Rahman, Abbatematteo, De Leo. Analysis and interpretation of data: Rahman, Abbatematteo, De Leo, Kubilis, Quinones-Hinojosa. Drafting the article: Rahman, De Leo, Vaziri. Critically revising the article: Rahman, Abbatematteo, Kubilis, Bova, Sayour, Mitchell, Quinones-Hinojosa. Reviewed submitted version of manuscript: Rahman, Abbatematteo, De Leo, Vaziri, Bova, Quinones-Hinojosa. Approved the final version of the manuscript on behalf of all authors: Rahman. Statistical analysis: Kubilis, Bova, Sayour. Administrative/technical/material support: Rahman, Bova, Quinones-Hinojosa. Study supervision: Rahman, De Leo, Vaziri, Bova, Sayour, Mitchell.

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