

Resection of parietal lobe gliomas: incidence and evolution of neurological deficits in 28 consecutive patients correlated to the location and morphological characteristics of the tumor

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Object. The goal of this study is to report the incidence and clinical evolution of neurological deficits in patients who underwent resection of gliomas confined to the parietal lobe.

Methods. Patient demographics, findings of serial neurological examinations, tumor location and neuroimaging characteristics, extent of resection, and surgical outcomes were tabulated by reviewing inpatient and office records, as well as all pre- and postoperative magnetic resonance (MR) images obtained in 28 consecutive patients who underwent resection of a glial neoplasm found on imaging studies to be confined to the parietal lobe. Neurological deficits were correlated with hemispheric dominance, location of the lesion within the superior or inferior parietal lobules, subcortical extension, and involvement of the postcentral gyrus.

The tumors were located in the dominant hemisphere in 18 patients (64%); had a mean diameter of 39 mm (range 14–69 mm); were isolated to the superior parietal lobule in six patients (21%) and to the inferior parietal lobule in eight patients (29%); and involved both lobules in 14 patients (50%). Gross-total resection, documented by MR imaging, was achieved in 24 patients (86%). Postoperatively, nine patients (32%) experienced new neurological deficits, whereas seven (25%) had an improvement in their preoperative deficit. A correlation was noted between larger tumors and the presence of neurological deficits both before and after resection. Postoperatively higher-level (association) parietal deficits were noted only in patients with tumors involving both the superior and inferior parietal lobules in the dominant hemisphere. At the 3-month follow-up examination, five of nine new postoperative deficits had resolved.

Conclusions. Neurological deterioration and improvement occur after resection of parietal lobe gliomas. Parietal lobe association deficits, specifically the components of Gerstmann syndrome, are mostly associated with large tumors that involve both the superior and inferior parietal lobules of the dominant hemisphere. New hemineglect or sensory extinction was not noted in any patient following resection of lesions located in the nondominant hemisphere. Nevertheless, primary parietal lobe deficits (for example, a visual field loss or cortical sensory syndrome) occurred in patients regardless of hemispheric dominance.

KEY WORDS • brain neoplasm • glioma • neurological deficit • parietal lobe • surgery

A COMPREHENSIVE evaluation of parietal lobe neurological deficits in patients with gliomas anatomically confined to the parietal lobe has not been previously reported. The only publications in which parietal lobe deficits have been discussed have either concentrated on the incidence of a specific deficit or syndrome^{9,19,21} or contained evaluation of seizure control in patients with both tumoral and nontumoral parietal lobe epilepsy.^{14,23} Furthermore, because many patients in these studies harbored gliomas not confined to the parietal lobe, conclusions of these studies about parietal lobe deficits may be inaccurate. The goal of this study was to report the incidence and clinical evolution of neurological deficits in patients who underwent resection of gliomas confined to the parietal lobe. A concise review of parietal lobe deficits is also provided.

Clinical Material and Methods

The NYU Institutional Review Board approved this clinical investigation. Patient data were extracted from the

Abbreviations used in this paper: GBM = glioblastoma multiforme; MR = magnetic resonance; NYU = New York University.

NYU Glioma Surgery Database, which contains clinical information regarding gliomas examined using biopsy procedures or managed with surgery at NYU Medical Center between 1995 and 2002. To be included in this study, patients were required to harbor a pathologically confirmed glioma confined to the parietal lobe that was surgically removed during this time period. Of 280 patients in the database, 57 had gliomas involving the parietal lobe. Magnetic resonance images obtained in these 57 patients were reviewed. The parietal lobe boundaries were determined using sulcal anatomical landmarks on preoperative multiplanar MR images,²² including the central, postcentral, and intraparietal sulci, as well as the posterior regions of the sylvian fissure and superior temporal sulcus. Neuroradiology reports relating to all preoperative MR images were also reviewed to confirm the glioma's location in the parietal lobe. When the mass effect of the tumor displaced or effaced adjacent sulcal landmarks, it was still possible to confirm the tumor's location in most patients based on the sulcal boundaries of the parietal lobe. For cases of larger tumors, however, serial postoperative MR images were also used to confirm the location of the resection cavity in the parietal lobe once the mass effect and edema had improved. Twenty-six

Surgery for parietal lobe gliomas

patients were subsequently removed from our analysis because they either underwent only a stereotactic biopsy or harbored a glioma extending outside the parietal lobe. The inpatient and office charts of the remaining 31 patients were reviewed. In three patients there was no proper documentation of pre- and postoperative parietal lobe neurological examinations, specifically parietal association deficits; these patients were also removed from further analysis. For final review this left 28 patients, all of whom underwent tumor resections performed by one of two surgeons (P.J.K. [26 patients] and J.G.G. [two patients]).

Patient demographics, clinical characteristics, findings of pre- and postoperative neurological examinations, operative complications, and disposition were retrospectively tabulated from inpatient and outpatient charts. The following neurological findings were recorded pre- and postoperatively in all patients: pronator drift, hemiparesis or other motor weakness, aphasia, visual field deficit, pinprick sensation, joint position sensation, sensory extinction, hemineglect, astereognosis, and components of Gerstmann syndrome (that is, agraphesthesia, right-left confusion, finger agnosia, and acalculia). Two-point discrimination and agraphia were not routinely documented. Patients with pre- or postoperative motor weakness and/or significant sensory loss were not evaluated for parietal association deficits because the latter could not be reliably documented when the former deficits were present. The evolution of neurological deficits was recorded by reviewing serial postoperative neurological examinations.

For a descriptive analysis, tumors were divided into the following categories: 1) GBM; 2) anaplastic glioma (including anaplastic astrocytoma, anaplastic ganglioglioma, and anaplastic oligodendroglioma); and 3) low-grade glioma (including mixed glioma, ganglioglioma, oligodendroglioma, and ganglioglioma).

All patients underwent MR imaging with and without Gd enhancement immediately before surgery and on postoperative Day 1. The neuroimaging characteristics of each tumor, including the lesion's diameter, volume, location (superior parietal lobule, inferior parietal lobule, or both), depth (cortically based, subcortical, or both), postcentral gyrus involvement, and tumor enhancement, were tabulated by reviewing all pre- and postoperative computerized tomography and MR images. Preoperative tumor volumes were measured by digitizing (outlining) the tumor margin on serial transaxial images and then reconstructing the tumor's three-dimensional volume by using the COMPASS stereotactic system (Compass Intl., Rochester, MN).¹⁶ Tumors were defined as cortically based if they were small and did not extend subcortically beyond the depth of adjacent sulci, whereas tumors were considered subcortical if they were predominantly located deep with regard to the depth of adjacent sulci. If the tumor involved the gyri and extended below the depths of adjacent sulci, it was recorded as both cortical and subcortical. Any correlation between parietal lobe deficits and tumor diameter, tumor volume, or patient age was assessed using the nonparametric Wilcoxon/Kruskal-Wallis (rank-sums) test. Furthermore, the association between postcentral involvement (yes or no), depth (cortical, subcortical, or both), or pathological diagnosis (GBM or not) with parietal lobe deficits was determined using the Fisher exact (two-tail) test.

All operations were performed for diagnosis, relief of

TABLE 1
Characteristics of 28 patients with parietal lobe gliomas at presentation

Characteristic	No. of Patients (%) [*]
sex	
male	18 (64)
female	10 (36)
handedness	
rt	26 (93)
lt	2 (7)
age (yrs)	
mean	44
range	18-75
presenting symptom	
seizure	15 (54)
headache	4 (14)
weakness	3 (11)
numbness	2 (7)
visual field deficit	2 (7)
change in mental status	2 (7)

^{*} The value represents the number of cases unless otherwise indicated.

mass effect, and oncological cytoreduction. No patients underwent surgery for control of refractory epilepsy. Twenty-seven of 28 patients underwent a frame-based volumetric stereotactic tumor resection, whereas one patient underwent a frameless stereotactic resection.^{16,17} Resections were limited to either: 1) the contrast-enhancing portion of a high-grade tumor whose bulk contained mostly solid enhancing tumor or 2) the area of increased T₂ signal for low-grade nonenhancing tumors and for high-grade tumors that displayed minimal contrast enhancement. When the aforementioned resection goals were achieved, as determined by reviewing pre- and postoperative MR images, the extent of tumor removal was classified as a gross-total resection; otherwise the resection was classified as near-total ($\geq 90\%$) or subtotal ($< 90\%$). Surgery was performed while the patient was in a state of general (endotracheal) anesthesia with motor and sensory intraoperative monitoring. We did not use awake intraoperative monitoring; however, a combination of phase-reversal direct motor cortex stimulation and transcranial somatosensory evoked potential and motor-evoked potential monitoring was used in every case.

Results

A summary of patient demographics and clinical presentations are listed in Table 1. The most common presenting complaint was a new onset of seizure in 15 patients (54%). Twenty patients (71%) exhibited a neurological deficit on initial evaluation. These deficits were categorized into sensory parietal, association parietal, motor, and other (Table 2). The three most prevalent preoperative neurological findings included: visual field deficit (36% of patients), pronator drift (29% of patients), and sensory extinction (25% of patients). Of note, five patients with hemiparesis and/or primary sensory deficits could not undergo proper evaluation of any parietal association deficits and were therefore excluded from the clinical analysis regarding these deficits; these patients nonetheless remained in our series. The neuroimaging characteristics of the tumors are listed in Table 3. In 18 patients (64%) the tumor was located in the dominant

TABLE 2
Preoperative neurological deficits in 28 patients with glioma

Deficit	No. of Patients (%)
present	20 (71)
absent	8 (29)
parietal sensory	
visual field	10 (36)
pinprick	4 (14)
joint position	4 (14)
parietal association	
sensory extinction	7 (25)
astereoagnosis	4 (14)
agraphesthesia	4 (14)
rt-lt confusion	1 (4)
finger agnosia	1 (4)
motor	
pronator drift	8 (29)
hemiparesis	5 (18)
other	
memory loss	1 (4)

hemisphere. The lesion involved the inferior parietal lobule in eight patients (29%), the superior parietal lobule in six patients (21%), and the postcentral gyrus in 17 patients (61%); it had a mean diameter of 39 mm (range 14–69 mm) and a mean volume of 35 cm³ (range 4–99 cm³).

Two patients had exhibited left-hand dominance since youth. One patient with a nonenhancing tumor situated in the left inferior parietal lobe underwent Wada testing, which localized speech function to the right hemisphere. The second patient did not undergo Wada testing, and underwent resection of a predominantly enhancing tumor located to the right superior parietal lobule and postcentral gyrus.

Surgical outcomes in all patients are listed in Table 4. Overall, 24 (86%) of the patients underwent gross-total re-

TABLE 3
Neuroimaging characteristics of gliomas in 28 patients

Characteristic	No. of Patients (%) [*]
hemisphere	
dominant	18 (64)
nondominant	10 (36)
diameter (mm)	
mean	39
range	14–69
vol (cm ³)	
mean	35
range	4–99
location	
superior parietal	6 (21)
inferior parietal	8 (29)
both lobules	14 (50)
depth	
cortical	10 (36)
subcortical	3 (11)
both	15 (53)
postcentral gyrus involvement	
yes	17 (61)
no	11 (39)
enhancement	
present	18 (64)
trace or none	10 (36)

^{*} The value represents the number of cases unless otherwise indicated.

TABLE 4
Surgical data and outcomes in 28 patients with glioma^{}*

Factor	No. of Patients (%) [†]
pathological diagnosis	
GBM	13 (46)
anaplastic tumor	11 (39)
low-grade tumor	4 (15)
resection	
gross-total	24 (86)
near-total	3 (11)
subtotal	1 (4)
complication(s)	
new deficit	9 (32)
DVT	1 (4)
tongue swelling	1 (4)
hospitalization (days)	
mean	4.5
range	2–13
disposition	
home	23 (82)
rehabilitation center	5 (18)

^{*} DVT = deep vein thrombosis.

[†] The value represents the number of cases unless otherwise indicated.

section with a mean hospitalization of 4.5 days. Two non-neurological complications occurred without permanent morbidity: one patient experienced deep vein thrombosis and another transient tongue swelling, which likely was related to the intraoperative electrophysiological monitoring technique. No patient died during the follow-up period. Of the four patients (14%) who underwent procedures with less than gross-total resection, near-total ($\geq 90\%$) resection was obtained in three and a subtotal tumor removal ($< 90\%$) in one. In a comparison of pre- and postoperative MR images in the three patients in whom near-total resection was obtained, one had a predominantly enhancing tumor and the other two had infiltrative tumors with only a few punctate areas of enhancement. The single patient in whom subtotal resection was undertaken had a nonenhancing, infiltrating tumor. Postoperatively, a residual portion of the tumor re-

TABLE 5
Immediate postoperative neurological status in 28 patients with glioma

Status	No. of patients (%)
deterioration	9 (32)
improvement	7 (25)
new deficits	
visual field	3 (11)
agraphesthesia	2 (7)
rt-lt confusion	2 (7)
joint position	1 (4)
hemiparesis	1 (4)
acalculia	1 (4)
astereoagnosis	1 (4)
finger agnosia	1 (4)
improved deficits	
hemiparesis	4 (14)
pinprick	3 (11)
pronator drift	1 (4)
agraphesthesia	1 (4)
sensory extinction	1 (4)

TABLE 6
Presence of deficits correlated with mean age and tumor size in 28 patients with glioma*

Variable	Deficit	Type of Deficit			
		Preop Primary†	Preop Association‡	Postop Primary†	Postop Association‡
patient age (yrs)	present	48 ± 3.1	54 ± 3.6	55 ± 4.6	45 ± 9.3
	absent	38 ± 5.3	38 ± 3.4	41 ± 3.2	44 ± 3.0
tumor diameter (mm)	present	44 ± 3.6	48 ± 4.5§	40 ± 5.7	42 ± 8.2
	absent	32 ± 3.4	33 ± 2.7	39 ± 3.1	38 ± 2.9
tumor volume (cm ³)	present	41 ± 6.3	5 ± 8.1§	35 ± 9.4	41 ± 17.7
	absent	27 ± 6.9	26 ± 4.7	35 ± 5.6	34 ± 4.6

* Values are expressed as means ± standard errors of the means.

† Primary sensory deficits include astereognosis, pinprick loss, joint position sense loss, mild hemiparesis or drift, and visual field loss.

‡ Association sensory deficits include extinction, agraphesthesia, right-left confusion, finger agnosia, and acalculia.

§ $p < 0.05$, nonparametric Wilcoxon/Kruskal-Willis rank-sum test.

mained deep and anterior in the resection cavity adjacent to the central sulcus; this residual lesion was left on purpose to minimize the risk of postoperative weakness.

During the initial postoperative week, 16 patients (57%) displayed some change in neurological status when compared with findings in their preoperative baseline examination: nine patients (32%) had a new deficit and seven patients (25%) noted improvement of their deficits. A list of new or resolved deficits is presented in Table 5. The cumulative incidence of new postoperative joint position sense loss, astereognosis, visual field loss, or mild hemiparesis (that is, all new neurological findings excluding association deficits) was 6% for patients with tumors in the dominant hemisphere and 9% for patients with lesions in the nondominant hemisphere.

To determine the correlation between tumor diameter, tumor volume, or patient age and parietal deficits, the mean values of these variables in patients with or without deficits were compared (Table 6). Furthermore, the percentages of patients with deficits having each of the following characteristics were evaluated: postcentral involvement (yes or no), depth (cortical, subcortical, or both), and pathological diagnosis (GBM or not) (Table 7). This analysis revealed that there was a correlation between larger tumor size (both volume and diameter), older patient age, and GBM on the one hand and the presence of both preoperative and postoperative neurological deficits on the other hand. A firm conclusion could not be made, however, due to the small number of patients in this study and because the data were nonparametric. No patients suffered aphasia before or after surgery.

For 23 patients in whom proper documentation of association parietal deficits was present both before and after resection, a correlation between association deficits and tumor involvement of both (superior and inferior) dominant parietal lobules was observed (Table 8). When the superior or inferior parietal lobules were solely involved, as noted in six patients for each location, a new postoperative association deficit only occurred in one patient. Only patients in whom gliomas were removed from the dominant hemisphere experienced a new postoperative association deficit (as already mentioned, nonassociation deficits did occur); no patient with a tumor in the nondominant hemisphere demonstrated sensory extinction, neglect, or a component of Gerstmann syndrome postoperatively. Analysis of these

results, along with data from Tables 6 and 7, revealed that patients with large gliomas in the dominant hemisphere involving both superior and inferior parietal lobules were most likely to have an association deficit before and/or after surgery.

All patients with new and persistent neurological deficits were examined for at least 3 months after surgery. Patients who were neurologically healthy or whose new postoperative deficits resolved during the 1st month after surgery were often followed for less than 3 months. During this follow-up period, five of nine patients with new postoperative neurological deficits experienced resolution of their deficits within the 1st month. Neurological deficits still present 1 month postoperatively did not improve. Overall, the results of the neurological examinations were documented during the follow-up period for 22 patients (79%) at 1 month and for eight patients (29%) at 3 months, with a mean duration of follow up of 2.9 months (range 0.5–21).

Illustrative Cases

Case 1

This 43-year-old right-handed man presented with a generalized seizure. Magnetic resonance imaging revealed a

TABLE 7
Incidence of deficits correlated with morphological and pathological characteristics of the tumor

Characteristic	Type of Deficit (% of cases)			
	Preop Primary	Preop Association	Postop Primary	Postop Association
postcentral gyrus involvement				
yes	58	53	18	24
no	63	18	27	9
depth				
cortical	40	20	10	10
subcortical	100	67	0	0
both	67	47	33	27
pathological diagnosis				
GBM	77	62	39*	23
not GBM	47	20	7	13

* $p < 0.05$, Fisher exact test (two tail).

TABLE 8
*Parietal association deficits correlated with tumor location in 23 patients**

Tumor Location (no. of patients)	Deficit (no. of patients)	
	Preop Finding	New Postop Finding
dominant hemisphere		
superior parietal lobule only (4)	intact (2) astereoagnosia (1) sensory extinction (1)	astereoagnosia (1)
inferior parietal lobule only (4)	intact (3) astereoagnosia (1) agraphesthesia (1)	none
both parietal lobules (7)	intact (4) sensory extinction (4) agraphesthesia (3) astereoagnosia (2) rt-lt confusion (1) finger agnosia (1)	agraphesthesia (2) rt-lt confusion (2) acalculia (1) finger agnosia (1)
nondominant hemisphere		
superior parietal lobule only (2)	intact (2)	none
inferior parietal lobule only (2)	intact (2)	none
both parietal lobules (4)	intact (2) sensory extinction (2)	none

* Five patients with hemiparesis and/or primary sensory deficits were excluded from this subanalysis. More than one deficit occurred in some patients.

predominantly enhancing glioma involving the right superior parietal lobule and postcentral gyrus (Fig. 1 *upper*). On examination the patient displayed left hemibody sensory extinction to double simultaneous stimulation. During resection, phase-reversal electrophysiological monitoring confirmed the tumor mass to be posterior to the central sulcus, and a gross-total removal was performed (Fig. 1 *lower*). The pathological diagnosis was GBM. The patient's immediate postoperative examination revealed continued left hemibody sensory extinction and a new left-arm sensory drift, along with diminished joint position strength and astereoagnosia in his left hand. By the 1-month follow-up examination his sensory extinction had resolved. His joint position sense deficit, however, remained unchanged 3 months after surgery.

Case 2

This 31-year-old left-handed man presented with a transient visual obscuration, consistent with a complex partial seizure. He was neurologically healthy except for an incongruous, predominantly right-superior-quadrant visual field deficit. Preoperative MR imaging (Fig. 2 *upper*) revealed a 4-cm nonenhancing glial neoplasm in the left inferior parietal lobule. The tumor was well demarcated, displacing the surrounding cortical sulci. A preoperative Wada test confirmed that the patient's speech function was localized to his right hemisphere. Following a gross-total stereotactic resection (Fig. 2 *lower*), the patient remained neurologically unchanged without any evidence of parietal lobe association deficits. The pathological diagnosis was ganglioglioneurocytoma. The man's visual field deficit remained unchanged the 1st week after surgery and also at his 1-month follow-up visit.

Discussion

Anatomical Correlation of Parietal Lobe Deficits

An illustration of the anatomy of the parietal lobe surface

is provided for reference (Fig. 3). Deficits referable to parietal lobe lesions may be generally divided into two categories: 1) the effects of a unilateral disease of the parietal lobe (right or left) regardless of hemispheric dominance; and 2) additional phenomena that may occur depending on whether the dominant or nondominant hemisphere is involved. Regarding the first category, patients may have cortical sensory syndrome, sensory extinction, mild hemiparesis, and visual field deficits. Cortical sensory syndrome, or Verger-Dejerine syndrome, occurs with damage to the postcentral gyrus and consists of diminished light touch, two-point discrimination, joint position sense, and astereoagnosia.¹² With dominant-hemisphere parietal lobe lesions, patients may experience language dysfunction (aphasia or dysphasia), Gerstmann syndrome, bilateral astereoagnosia, and an inability to perform learned motor tasks (ideomotor apraxia). Gerstmann syndrome consists of finger agnosia, left-right confusion, acalculia, and agraphia.¹¹ Although still the subject of debate, it is thought that lesions focal to the dominant inferior parietal lobule cause Gerstmann syndrome.¹ A complete discussion of Gerstmann syndrome can be reviewed elsewhere.²¹ For lesions located in the nondominant hemisphere, patients may experience hemineglect, constructional and/or dressing apraxia, a tendency to keep eyes closed mimicking lethargy, and prosopagnosia.^{7,18,28}

Most parietal lobe deficits are readily elicited using simple, bedside neurological testing; however, documentation of more complex association deficits requires more targeted neuropsychological testing. Given that these deficits are often subtle and many patients truly appear neurologically healthy even though they may harbor significant lesions of the posterior parietal cortex, the parietal lobe cortex (excluding the anterior postcentral gyrus and dominant inferior lobule) is often considered "silent." This lack of neurological dysfunction may be secondary to both redundant and/or bilateral functional representation in the parietal cortex.⁵ Although theoretical, the acuity of parietal lobe damage may also determine the presence and severity of neurological

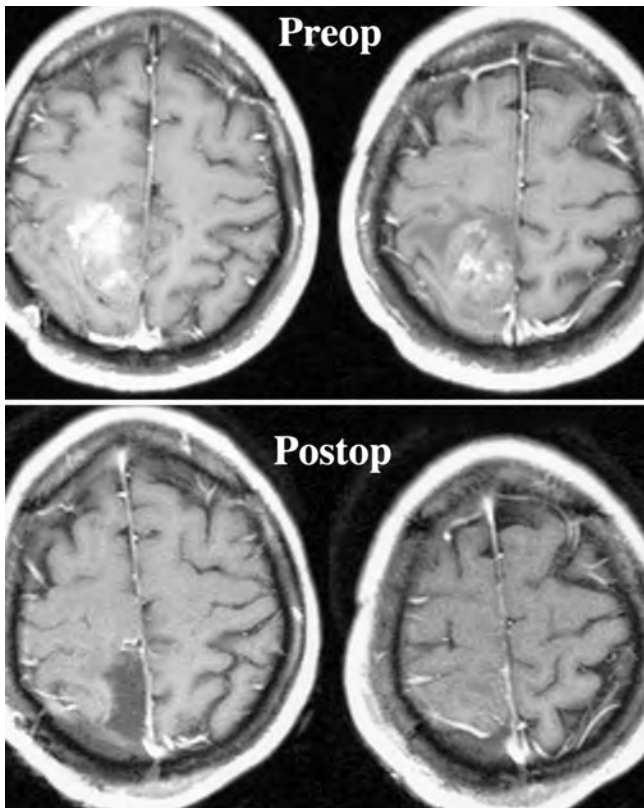


FIG. 1. Case 1. *Upper*: Preoperative MR images revealing a predominantly enhancing glioma involving the right superior parietal lobule and postcentral gyrus. *Lower*: Postoperative MR images obtained after gross-total tumor removal

dysfunction, with slow-growing tumors often not causing deficits.

Parietal Lobe Gliomas and Their Resection

A number of articles have addressed deficits associated with gliomas removed from eloquent cortical areas.^{6,10,27} Nevertheless, few researchers have specifically investigated gliomas of the parietal lobe.^{9,14,19,21,23} Some general observations regarding parietal lobe gliomas were obtained from our study. The majority of patients (71%) with parietal gliomas presented with sensory and/or motor deficits caused by their lesions. These deficits, including cortical sensory syndromes, sensory extinction, and components of Gerstmann syndrome, were readily identified during bedside testing. In patients with motor weakness or significant cortical sensory syndromes, testing for parietal lobe association deficits was unreliable, as was the case in five patients (18%) in our series. In half the patients, new postoperative deficits resolved within the first few postoperative weeks. This recovery may be secondary to reorganization and functional plasticity of the parietal cortex,^{3,4,20} or more simply from resolution of localized postsurgical swelling.

The relationships between the association parietal deficits, at presentation or following resection, and the hemispheric dominance, parietal lobe involvement, subcortical tumor extension, and postcentral gyrus involvement were investigated. Of note, patients with tumors involving both superior and inferior parietal lobules of the dominant hemi-

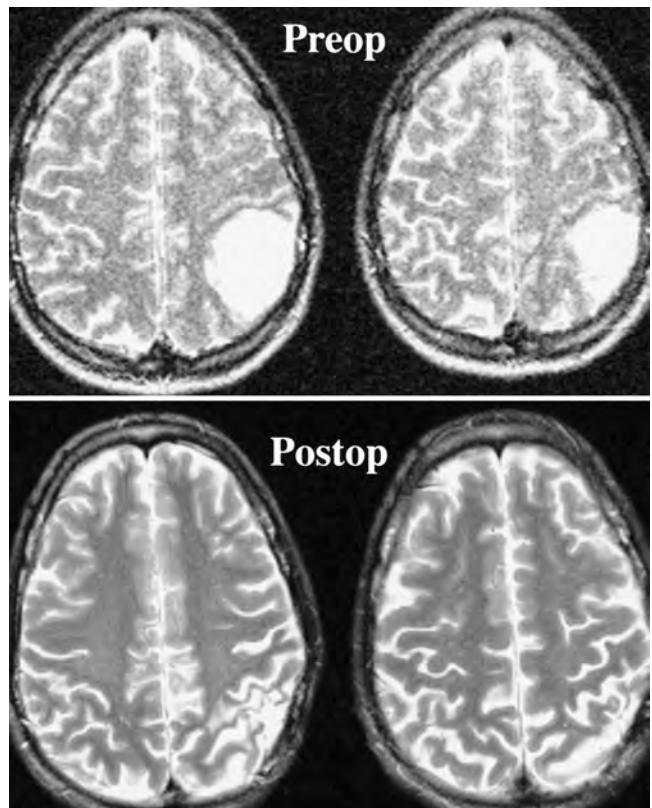


FIG. 2. Case 2. *Upper*: Preoperative MR images revealing a 4-cm nonenhancing glial tumor in the left inferior parietal lobule, which is displacing the surrounding cortical sulci. *Lower*: Postoperative MR images obtained after gross-total resection of the lesion.

sphere were most likely to have new association deficits postoperatively, specifically the components of Gerstmann syndrome. Of note, primary parietal lobe deficits (for example, visual field loss or cortical sensory syndrome) occurred in patients regardless of hemispheric dominance.

Roux and colleagues²¹ correlated the incidence and evolution of the components of Gerstmann syndrome in six patients with low-grade gliomas in the dominant inferior parietal lobule by using intraoperative testing during awake craniotomy. These authors found that cortical stimulation of not only the inferior parietal lobule, but also of the non-dominant angular gyrus and other regions of the posterior parietal lobe, could elicit the components of Gerstmann syndrome. They concluded that, although lesions located away from the dominant angular gyrus can produce one or more signs related to the Gerstmann tetrad, a complete Gerstmann syndrome may occur with a small lesion in the angular gyrus, sparing speech function. No patient in our series had all four components of Gerstmann syndrome. In fact, the majority of patients with tumors of the dominant parietal lobe showed no evidence of Gerstmann syndrome, including the four patients with small tumors localized to the dominant inferior parietal lobule. Nevertheless, the majority of patients with larger tumors involving both dominant parietal lobules had one or more components of this syndrome.

A high rate (96%) of gross-total or near-total tumor resec-



FIG. 3. A representation of both parietal lobes from a posterior-superior perspective. The cerebral hemispheres have been opened like a book away from the viewer, which allows for simultaneous views of both parietal lobes. Asterisks mark the postcentral sulcus, whereas circles mark the inter- (or intra-) parietal sulcus. The postcentral gyrus is shaded yellow, the superior parietal lobules dark pink, the supramarginal gyri turquoise, and the angular gyri green.

tion was achieved in the patients in this series. Using volumetric stereotactic guidance, the amount of perilesional functional cortex that was damaged or resected was minimized, thereby potentially limiting the number of permanent postoperative deficits. Although not performed in our patients, removal of nontumoral parenchyma for seizure control, which can contain functional cortex, may also predispose to neurological deficits after surgery. Two reports offering an analysis of clinical outcomes after surgical management of lesional or nonlesional parietal lobe epilepsy cited 39 and 59% incidences of new or worsened parietal lobe deficits after surgery.^{14,23} Although similar to our results, the small number of patients and the nonstandardized methods of documenting these deficits limited a direct comparison of the studies.

The predominantly contrast-enhancing portions of GBMs are not expected to contain any functional cortex, and therefore, in general, new deficits are uncommon with their removal. Nevertheless, resection of low-grade gliomas in which isolated tumor cells have often infiltrated functional parenchyma may produce a higher incidence of neurological deficits and therefore more caution should be used when treating these lesions.^{10,15,24,26} Because we restricted our selection to gliomas contained within the pari-

etal lobe, the present study did not have enough patients to make a proper comparison of neurological outcomes between patients with low-grade tumors and those with high-grade ones.

Limitations of the Study and Future Directions

The results of this initial clinical evaluation should be tempered by the limitations inherent in the study methods used, the most significant of which was the retrospective collection of neurological examinations, which may have underestimated the true incidence of deficits in our cohort. Although we routinely documented any pre- and postoperative neurological findings in all patients, formal neuropsychological testing would have been more sensitive and specific, especially for more subtle abnormalities (for example, constructional apraxia, and components of Gerstmann syndrome). We believe the use of sulcal anatomical landmarks on multiplanar MR images was both accurate and reliable in determining the location of gliomas within the parietal lobe.²² Although not used in our study, MR tractography (diffusion-tensor imaging),^{13,25,29} functional MR imaging,^{2,26} and awake intraoperative monitoring^{8,10} may be used to predict and, possibly, minimize postoperative deficits.

Conclusions

Neurological deficits and improvement occur following resection of parietal lobe gliomas. Parietal association deficits, specifically the components of Gerstmann syndrome, only occurred in patients with large tumors involving both the superior and inferior parietal lobules of the dominant hemisphere. New hemineglect or sensory extinction was not noted in any patient following resection of lesions in the nondominant hemisphere. Primary parietal lobe deficits (for example, visual field loss or cortical sensory syndrome), however, occurred in patients regardless of hemispheric dominance.

Disclosure

Dr. Kelly has a financial interest in Compass International, Inc.

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