Essentials of Clinical Radiation Oncology is a comprehensive, user-friendly clinical review that summarizes up-to-date cancer care in an easy-to-read format. Each chapter is structured for straightforward navigability and information retention beginning with a “quick hit” summary that contains an overview of each disease, its natural history, and general treatment options. Following each “quick hit” are high-yield summaries covering epidemiology, risk factors, anatomy, pathology, genetics, screening, clinical presentation, workup, prognostic factors, staging, treatment paradigms, and medical management for each malignancy. Each treatment paradigm section describes the current standard of care for radiation therapy including indications, dose constraints, and side effects. Chapters conclude with an evidence-based question and answer section which summarizes practice-changing data to answer key information associated with radiation treatment outcomes. Flow diagrams and tables consolidate information throughout the book that all radiation oncologists and related practitioners will find extremely useful when approaching treatment planning and clinical care.

Essentials of Clinical Radiation Oncology has been designed to replicate a “house manual” created and used by residents in training and is a “one-stop” resource for practicing radiation oncologists, related practitioners, and radiation oncology residents entering the field.

KEY FEATURES:
- Offers digestible information as a learning guide for general practice
- Examines essential clinical questions which are answered with evidence-based data from important clinical studies
- Places clinical trials and data into historical context and points out relevance in current practice
- Provides quick reference tables on treatment options and patient selection, workup, and prognostic factors by disease site
ESSENTIALS OF CLINICAL RADIATION ONCOLOGY
ESSENTIALS OF CLINICAL RADIATION ONCOLOGY

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To the enduring commitment of past, present, and future residents in the pursuit of knowledge, without whom this work would not have been possible.
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Team-based learning and patient-centered care have been long-standing traditions in the Cleveland Clinic Radiation Oncology Residency program. Over the past two decades, as a complement to the formal teaching curriculum, residents have spent countless hours condensing the pertinent literature into comprehensive high-yield clinical summaries, which are bound into a study manual and updated annually. Essentials of Clinical Radiation Oncology is a tribute to the generations of trainees who have contributed to this ongoing process of continual learning and improvement.

In the same spirit as our first venture, Handbook of Treatment Planning in Radiation Oncology, we felt there was a need for a clinically oriented resource that was both succinct and comprehensive, similar to our in-house manual. Thus, we are now proud to offer Essentials of Clinical Radiation Oncology as our formal answer to this need. Essentials of Clinical Radiation Oncology has been designed to serve as the clinical companion to the more technically oriented Handbook of Treatment Planning in Radiation Oncology. It is intended to provide residents, students, and practicing radiation oncologists with an easy-to-access resource in the rapidly evolving field of oncology, while serving as a supplement to other currently existing resources available to radiation oncologists, such as the AJCC Cancer Staging Manual and published consensus guidelines from national organizations. Our intention is to update the content of this handbook regularly over time, in order to keep pace with the changing clinical environment in oncology.

Tremendous thanks must be given to our current Cleveland Clinic Radiation Oncology residents, recent graduates, and dedicated faculty who shared our vision in producing this work. The efforts of our Chief Resident, Matthew C. Ward, MD, must be particularly acknowledged, as he assumed the task of organizing the authors’ efforts and serving as the lead editor. We value and appreciate suggestions from our readers on how to keep this book current, and we encourage feedback to be sent by email to RO_chiefres@ccf.org. We hope that our community of readers finds this book to be a helpful resource, and that our patients ultimately benefit from the collective wisdom reflected in it and that is brought to training each generation of oncologists.

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ABOUT THE FORMAT OF THIS BOOK

The intention of this book is to serve as a comprehensive resource for all levels of practitioners, from medical students to practicing physicians alike. Therefore, the reader will find clinically pertinent details starting from basic epidemiology and culminating in an evidence-based approach to important and up-to-date clinical questions. The “front matter” of each chapter contains information about the disease and its natural history. This includes a summary of the AJCC eighth edition staging system (or other relevant risk-stratification systems), printed in an abbreviated format intended for physician understanding. Next, general “treatment paradigms” are included in the midpart of each chapter to give the reader an overview of the role of each anticancer modality in the multidisciplinary care of the patient. Finally, the highlight of this resource is the “evidence-based question and answer” format of clinical studies presented to guide the reader through the
most pertinent literature. Each study is block-quoted from the source with a quick-access citation to the original reference in combination with a condensed summary intended to highlight the pertinent findings. It should be noted that our intention with this book is to provide a manual of information useful to the clinician, rather than to be “prescriptive” in terms of staging, radiation delivery, or chemotherapy dosing. Our hope is that this format provides an efficient yet thorough method for practitioners to develop a deeper understanding of a disease and the current state of its treatment.
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Essentials of Clinical Radiation Oncology
3: LOW GRADE GLIOMA

Martin C. Tom and Erin S. Murphy

QUICK HIT: Low grade gliomas (LGGs) are an uncommon and heterogeneous group of primary brain tumors which present primarily in younger adults, but can also be seen in pediatric pts. Recent publications analyzing molecular and genomic factors have provided a window into prognostic stratification. Mutation in the enzyme IDH has emerged as the most informative genomic change. Despite this prognostic information, treatment paradigms remain based on clinical factors and require patient-specific decision making given the diversity of these tumors. Following surgical resection, options can include observation, RT, CHT or combined chemoRT. RT dose is typically 50.4–54 Gy. CHT often consists of either oral temozolomide or PCV (procarbazine, lomustine/CCNU, and vincristine).

TABLE 3.1: General Treatment Paradigm for Low-Grade Gliomas Based on Clinical Factors in the Pre-Genomics Era

<table>
<thead>
<tr>
<th>Maximal Safe Resection</th>
<th>Low risk (&lt;40 years old as per RTOG or without Pignatti risk factors as per EORTC)</th>
<th>Observation, CHT or chemoRT</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>High Risk (≥40 years old as per RTOG or with Pignatti risk factors as per EORTC)</td>
<td>CHT or chemoRT</td>
</tr>
<tr>
<td>STR or biopsy</td>
<td></td>
<td>ChemoRT</td>
</tr>
</tbody>
</table>

EPIDEMIOLOGY: An estimated 64,808 new cases of primary neuroepithelial tumors are expected in the United States annually, and approximately 15% will be grade I-II tumors, 60% will be grade IV.1 Approximately 2,600 cases are WHO grade II diffuse astrocytoma.1,2

RISK FACTORS: Ionizing radiation and genetic syndromes including NF-1 (17q, café au lait spots, Lisch nodules, neurofibromas, optic gliomas, & astrocytomas), NF-2 (22q, bilateral acoustic neuromas, meningiomas, ependymomas, gliomas), Tuberous sclerosis (ash-leaf macules, hamartomas, angiofibromas, periumgual fibromas, subependymal giant cell astrocytoma, gliomas) or Li-Fraumeni syndrome (TP53 mutation, gliomas, sarcomas, breast cancer, leukemia, adrenocortical carcinomas).

ANATOMY: Typically LGGs arise from the supratentorial cortex. Brainstem gliomas and optic pathway gliomas, when biopsied, are often classified as low grade but are covered elsewhere.

PATHOLOGY: Gliomas represent a group of tumors with characteristics of neuroglial cells (astrocytes or oligodendrocytes). LGG represent a heterogeneous group of WHO grade I (non-infiltrative) and grade II (infiltrative/diffuse) glial neoplasms.

WHO grading: Grading is based on the presence of the following histologic features: mitoses, endothelial proliferation, nuclear atypia, and necrosis (“MEAN” mnemonic).
2016 WHO CNS classification update: In addition to histology, the new classification includes molecular markers to better define CNS tumors (see Figure 3.1).

**Oligodendroglioma, IDH-mutant and 1p19q codeleted:** Median OS >10 years. Favorable prognosis and response to CHT. Characterized by 1p19q codeletion. Histology shows perinuclear halos, “fried egg” with branching “chicken wire” vasculature and calcification.

**Diffuse astrocytoma, IDH-mutant:** Median OS typically >10 years, characterized by IDH-mutation with ATRX loss, TP53 mutation, 1p19q intact.

**Diffuse astrocytoma, IDH-wildtype:** Median OS ~5 years, less common, may act similar to WHO grade III anaplastic astrocytoma IDH-WT.

**Gemistocytic astrocytoma, IDH-mutant:** Median OS typically <4 years, high risk for malignant transformation and treated as WHO grade III glioma. Histology shows large, densely packed gemistocytes.

**If molecular testing unavailable:**

- **Diffuse astrocytoma NOS:** Median OS 4-5 yrs
- **Oligodendroglioma NOS:** Historically median OS >10 yrs. Of note, oligodendroglioma IDH-wildtype falls into this category (see Figure 3.1).
- **Oligoastrocytoma NOS:** Median OS <7 yrs, characteristics of both oligodendroglioma and astrocytoma, worse prognosis than pure oligodendroglioma. Can now typically be classified as oligodendroglioma or astrocytoma based on molecular markers.

**Grade I tumors**

**Pilocytic astrocytoma:** Slow growing, often cystic tumor in children and young adults demonstrating Rosenthal fibers. Enhances on MRI due to degenerative hyalinization of blood vessels. Malignant transformation rare. Common location posterior fossa.

---

**Figure 3.1: WHO 2016 Glioma classification.**

*characteristic but not required for diagnosis.

*Source: From Ref. (3). Used with permission.*
Pleomorphic xanthoastrocytoma: Large, peripheral tumor frequently with leptomeningeal involvement. Often benign despite aggressive histologic appearance.

Subependymal giant cell astrocytoma: Well-defined tumor typically along lateral ventricles.

Ganglioglioma: Composed of both neoplastic neurons and astrocytes, commonly in temporal lobe, indolent course.

Genetics

IDH1 and IDH2 mutations: Present in majority of WHO grade II gliomas, favorable prognosis compared to IDH-wildtype.\(^8\)

1p19q codeletion: Defining feature of oligodendroglioma, favorable prognosis.\(^8\)

TP53 mutation and/or ATRX mutation: Characteristic of IDH-mutated astrocytomas, ATRX mutation is mutually exclusive from 1p19q codeletion,\(^9\) less favorable prognosis than 1p19q codeletion.

TERT promoter mutation: Among IDH-wild type LGG it confers a poor prognosis.\(^10\)

MGMT methylation: Role in LGG is unclear, but has been associated with improved post-recurrence survival in presence of TMZ.\(^11,12\)

BRAF: Mutations present in ganglioglioma, pilocytic astrocytoma, and pleomorphic xanthoastrocytoma.\(^13\)

One analysis from the Cancer Genome Atlas ordered genomic alterations from most to least favorable: IDH-mutated and 1p19q codeleted > IDH-mutated and 1p19q intact > IDH-wildtype.\(^8\) Another analysis from the Mayo Clinic, UCSF and MSKCC grouped from best to worst prognosis: TERT and IDH-mutation > TERT and IDH-mutation and 1p19q codeletion > IDH-mutation only > no mutations (triple-negative) > TERT only mutation.\(^10\)

CLINICAL PRESENTATION: Depends upon location, but most commonly presents as a transient neurologic disturbance or seizure (seizure in >80% of LGG compared to 70% and 50% in anaplastic and GBM, respectively). Most commonly nonenhancing hemispheric lesion (~20% do enhance\(^4\)), rarely mass effect. Best seen on T2 MRI (hypointense on T1, nonenhancing with gadolinium). Calcifications may be present, most commonly in oligodendrogliomas, and may be more common with 1p19q codeletion.\(^15\) Of note, pilocytic astrocytomas enhance via a different mechanism than anaplastic astrocytomas and GBM (degenerative hyalinization of blood vessels).

WORKUP: H&P, neurologic exam, neurocognitive testing, EEG if seizures. MRI with and without contrast. Functional MRI if in critical region. Establish preoperative neurocognitive baseline through testing if possible. Obtain tissue via a maximal safe resection, with biopsy-only if a resection is not possible. In general, obtain a postoperative MRI within 72 hours of surgery (ideally 24–48 hours) to determine the extent of surgical resection/residual disease and avoid confounding by blood products.

PROGNOSTIC FACTORS: There is no agreed upon definition of low-risk and high-risk pts. Various cooperative groups have defined risk factors differently. Pignatti combined EORTC trials and established five poor prognostic factors: age ≥40, astrocytoma histology, tumors ≥26 cm, tumor crossing midline, and preoperative neurologic deficits.\(^16\) RTOG 9802 stratified pts based on age and resection status, with those <40 achieving a GTR composing the low-risk group. Seizure at presentation is a positive prognostic factor.\(^17\) Another combined EORTC/RTOG/NCCCTG analysis by Gorlia identified four externally-validated factors: neurologic deficit at presentation, <30 weeks since first symptoms, astrocyte histology, and tumor >5 cm.\(^18\) Note that age was not prognostic in this analysis. Molecular markers have been found to be important predictors of outcome (see section on Genetics).
NATURAL HISTORY: Varies widely depending on histology, prognostic factors and molecular markers. However, most pts eventually deteriorate from tumor recurrence (typically occurring at the original site). At recurrence, up to 70% of tumors have undergone malignant transformation (i.e., WHO grade III/IV).19

TREATMENT PARADIGM: In general, most pts are recommended maximal safe resection followed by postoperative MRI to evaluate extent of resection. Low-risk pts may be observed, whereas high-risk pts are typically recommended adjuvant chemoRT. There is no consensus definition for low or high risk, but generally low-risk pts are <40 who achieve GTR (per RTOG 9802) or have fewer Pignatti risk factors (see section on Prognostic Factors). Current trials are also stratifying based on molecular markers (i.e., IDH mutation and 1p19q codeletion).

Surgery: Surgery is generally required to establish a diagnosis and debulk the tumor for those with extensive neurologic symptoms. There are no trials directly assessing extent of resection in LGG; however, degree of resection is a strong prognostic factor.20 The low-risk arm from RTOG 9802 showed significant correlation between amount of residual tumor on imaging and recurrence.21

Observation: Following surgery, observation is an option for low-risk pts. This was supported by the “Non-Believers Trial” (as discussed in the following) and the phase II portion of RTOG 9802 which defined low-risk as those <40 years old achieving a GTR. However, close follow-up is crucial as RTOG 9802 showed a greater than 50% risk of progression at 5 years in low-risk pts undergoing adjuvant observation. RTOG 0925 will hopefully provide more insight on the role of observation for low-risk pts.

Chemotherapy: The use of adjuvant CHT (and chemoRT) in LGG continues to evolve. Pts with high-risk features may be chosen for immediate postop therapy. RTOG 9802 (phase III) investigated adjuvant RT followed by six cycles of PCV, whereas RTOG 0424 (phase II) evaluated RT with concurrent and adjuvant TMZ for 12 mos. Both regimens have activity in LGG, but level I evidence (RTOG 9802) exists only for PCV. However, many institutions favor TMZ over PCV given better tolerance and ease of administration. EORTC 22033-26033 showed no difference in PFS if treated with dose dense TMZ alone versus RT alone, however further data maturation is necessary.

Radiation

Indications: High-risk pts should undergo adjuvant chemoRT. RTOG 9802 established adjuvant chemoRT as the standard of care for high-risk pts (defined as age ≥40 or <40 years old following STR).

Dose: Doses of 45 to 54 Gy are acceptable.22 The European trial E3F05 used 50.4 Gy/28 fx, whereas both RTOG 9802 and RTOG 0424 used 54 Gy/30 fx.

Toxicity: Acute: Fatigue, headache, exacerbation of presenting neurologic deficits, alopecia, nausea, cerebral edema, side-effects related to chemotherapy. Late: Cognitive changes, radiation necrosis, hypopituitarism, cataracts, vision loss (rare and location dependent).

EVIDENCE-BASED Q&A

Does early surgical resection improve outcomes compared to watchful waiting?

Retrospective studies favor upfront maximal safe resection, however no prospective trials are available to answer this question.

Jakola, Norwegian University Hospitals (JAMA 2012, PMID 23099483): Population-based study of surgical resection (and extent) compared to observation. Chosen based
on patient’s residential address. In hospital A, pts were biopsied and observed (50% ultimately underwent resection) but in hospital B an early resection was performed. MFU was 7 years. OS was significantly better with early surgical resection (5-yr OS 60% vs. 74%, p = .01) favoring early resection. Fewer pts achieved resection if delayed (89% vs. 59%).

**Conclusion:** Early resection is warranted if safe and feasible.

**Is it safe to observe pts after surgery and save RT for progression?**

Yes, but the ideal population to observe is unclear in the genomic era and routine observation is associated with reduced PFS and increased seizure rates.

**van den Bent, EORTC 22845 “Non-Believers Trial” (Lancet 2005, PMID 16168780):** PRT of 311 pts (WHO PS 0-2) with LGG after surgery randomized to immediate RT (54 Gy/30 fx) versus observation with RT at progression. Included astrocytoma (50%), oligodendroglioma (13%), mixed (13%) and incompletely resected pilocytic astrocytomas (1%). Greater than 90% resection in 42%, 50% to 89% resection in 20%, <50% resection or biopsy in 38%. Sixty-five percent of pts in observation arm eventually received RT. Survival after first recurrence was better in pts who were observed up front (3.4 vs. 1 yr), likely due to RT salvage. Malignant transformation 70%, equal between arms. **Conclusion:** Immediate (vs. delayed) RT improved PFS and decreased seizure rate, but did not improve OS.

| TABLE 3.2: Results of EORTC “Non-Believers Trial” |
|---------------------------------|-----|-----|-----|-----|
|                                 | MS  | 5-yr OS | Med PFS | 5-yr PFS | Seizures at 1 yr |
| Observation                     | 7.4 yrs | 65.7% | 3.4 yrs | 34.6% | 41% |
| Post-op 54 Gy/30 fx            | 7.2 yrs | 68.7% | 5.3 yrs | 55.0% | 25% |
| p value                        | .872 | <.0001 | .0329 |

**Shaw, RTOG 9802 Phase II (J Neurosurg 2008, PMID 18976072):** Phase II portion of RTOG 9802. Postsurgery, observed 111 pts <40 y/o who achieved GTR and reported 5-yr OS of 93% and 5-yr PFS of 48%. GTR was determined by neurosurgeon at time of surgery. Review of postop MRI revealed that 59% of pts had <1 cm residual disease (26% recurrence), 32% had 1 to 2 cm residual disease (68% recurrence), 9% had >2 cm residual disease (89% recurrence). Poor prognostic factors included large tumor size (≥4 cm), astrocytoma or mixed oligoastrocytoma histology and residual disease ≥1 cm by MRI. **Conclusion:** LGG in pts <40 years old following GTR have >50% risk of progression at 5 years and should be closely followed with consideration of adjuvant treatment.

**Does RT dose-escalation improve outcomes?**

Despite early retrospective data supporting a benefit, two trials have failed to confirm a role for dose-escalation.23

**Karim, EORTC 22844 “Believers Trial” (IJROBP 1996, PMID 8948338):** PRT of 379 pts with supratentorial low-grade astrocytomas, oligodendrogliomas, and mixed oligoastrocytoma, ages 16 to 65, KPS ≥60, randomized to 45 Gy/25 fx versus 59.4 Gy/33 fx after surgery (any degree of resection). Radiation necrosis risk 2.5% vs. 4% at 2 yrs. **Conclusion:** No difference in 5-yr OS (59% vs. 58%) or PFS (50% vs. 47%) with dose escalation.

**Shaw, RTOG 9110 (JCO 2002, PMID 11980997):** PRT of 203 pts with supratentorial grade 1 to 2 astrocytoma, oligodendroglioma, or mixed oligoastrocytoma randomized to 50.4 Gy/28 fx versus 64.8 Gy/36 fx, following surgery (any degree of resection). **Conclusion:** No difference in 5-yr OS (64% vs. 72%) with higher rate of severe radiation necrosis seen with high dose arm (5% vs. 2%). Ninety-two percent of failures were in-field.
How do tumor progression and RT affect cognition?

One reason to delay RT is to avoid the initial neurocognitive effects of treatment, but this is associated with reduced PFS (“Non-Believers Trial” previously mentioned) which may also affect cognition. Analysis of RTOG 9110 showed stable MMSE scores for most pts, and improvement in MMSE for those with lower baseline scores. Analysis of RTOG 9802 showed improved MMSE scores with the addition of CHT. However, MMSE may not be as reliable in evaluating neurocognitive function as more formal testing. A more extensive analysis of 20 pts in RTOG 9110 used formal cognitive testing and showed stable neurocognitive function up to 5 years out from RT. RT dose-escalation may worsen QOL as analysis of the “Believers Trial” showed that pts who received dose-escalation reported worse QOL than conventional RT doses.

Does adjuvant chemoRT improve outcomes compared to adjuvant RT alone?

The addition of PCV to RT nearly doubles survival in high-risk pts.

**TABLE 3.3: Final Results of RTOG 9802 Phase III Component**

<table>
<thead>
<tr>
<th></th>
<th>MS</th>
<th>10-yr OS</th>
<th>Med PFS</th>
<th>10-yr PFS</th>
</tr>
</thead>
<tbody>
<tr>
<td>RT alone</td>
<td>7.8 yr</td>
<td>41%</td>
<td>4 yr</td>
<td>21%</td>
</tr>
<tr>
<td>RT followed by PCV</td>
<td>13.3 yr</td>
<td>62%</td>
<td>10.4 yr</td>
<td>51%</td>
</tr>
<tr>
<td>p-value</td>
<td>.003</td>
<td>&lt;.001</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Is treatment with temozolomide similar to PCV?

Level I data supports the addition of PCV to RT, which improves OS compared to adjuvant RT alone. However, PCV is toxic and more difficult to administer than TMZ. Many therefore give TMZ, extrapolating from high-grade glioma data. This question is being addressed in the ongoing CODEL study, a phase III trial randomizing pts with 1p19q codeletion (either LGG or AG) to adjuvant RT followed by PCV versus RT+TMZ followed by TMZ.

Fisher, RTOG 0424 (IJROBP 2015, PMID 25680596): Single-arm phase II of high-risk LGG (WHO grade II astrocytomas, oligodendrogliomas, and mixed oligoastrocytoma) treated with RT (54 Gy/30 fx) with concurrent daily TMZ followed by 12 cycles of monthly TMZ. Pts must have three or more of the following risk factors: age ≥40, tumor ≥6 cm, tumor crossing midline, preoperative NFS >1, astrocytoma histology. 129 pts eligible. 3-yr OS was 73.1% comparing favorably to historical rate of 54% (p < .001) and higher than hypothesized rate of 65%. 3-yr PFS 59% and grade 3/4 toxicity in 43%/10%. Conclusion: Early results with TMZ are favorable, but equivalency of TMZ and PCV remains unknown.
Are there subsets of pts who can be treated initially with CHT alone?

Given the long and variable natural history of LGG and relatively younger patient population, studies have evaluated whether RT can be deferred to avoid toxicity. EORTC 22033-26033 compared high-risk LGG treated with RT alone versus dose dense TMZ alone and found no difference in PFS, HR-QOL, or impaired cognitive dysfunction. It is important to note that the median PFS of 39 months (TMZ alone) and 46 months (RT alone) in the EORTC study was far less than the median PFS of 10.4 years (RT+PCV) in RTOG 9802.

Baumert, EORTC 22033-26033 (Lancet Oncol 2016, PMID 27686946): PRT of 477 pts with LGG, age ≥18, ≥1 high-risk feature (age >40, size >5 cm, progressive disease, tumor crossing midline, neurologic symptoms) randomized to RT alone (50.4 Gy/28 fx) versus dose-dense TMZ alone (75 mg/m² days 1–21 of a 28-day cycle, max 12 cycles). Stratified by 1p deletion, contrast enhancement, age ≥40, ECOG ≥1. Primary endpoint PFS. MFU 48 mos, mPFS 46 mos for RT alone versus 39 mos for TMZ alone (p = .22). OS not reached. Exploratory analysis showed IDH mutation/1p19q non-codeleted had longer PFS if treated with RT alone versus TMZ alone (p = .0043), but no difference for IDH mutated/1p19q codeleted or IDH wildtype. Grade 3 to 4 hematologic toxicity <1% RT versus 14% TMZ, moderate/severe fatigue 3% RT versus 7% TMZ, grade 3 to 4 infections 1% RT versus 3% TMZ. Conclusion: No significant difference in PFS for LGG treated with RT alone versus TMZ alone. Awaiting OS endpoint and further data maturation for molecular subtypes.

Reijneveld, EORTC 22033-26033 Health Related-QOL (Lancet Oncol 2016, PMID 27686943): HR QOL and global cognitive functioning evaluated in the above study (LGG treated with RT alone vs. TMZ alone) using EORTC questionnaire and MMSE. No difference in HR-QOL at 36 mos between RT alone versus TMZ alone (p = .98). No difference in impaired cognitive function at baseline (13% in RT vs. 14% TMZ) or at 36 mos after treatment (8% in RT vs. 6% TMZ). Conclusion: HR QOL and global cognitive function (by MMSE) did not differ in LGG pts treated with RT alone versus TMZ alone.

REFERENCES


