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Irradiation of Pediatric High-Grade Spinal Cord Tumors

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Abstract

Purpose—To report the outcome using radiation therapy (RT) for pediatric patients with high grade spinal cord tumors.

Methods and Materials—A retrospective chart review was conducted that included 17 children with high-grade spinal cord tumors treated with RT at St. Jude Children's Research Hospital between 1981 and 2007. Three patients had gross total resection, 11 had subtotal resection, and 3 underwent biopsy. The tumor diagnosis was glioblastoma multiforme (n = 7), anaplastic astrocytoma (n = 8), or anaplastic oligodendroglioma, (n = 2). Seven patients received craniospinal irradiation (34.2–48.6Gy). The median dose to the primary site was 52.2 Gy (range 38–66 Gy).

Results—The median progression-free and overall survivals were 10.8 and 13.8 months, respectively. Local tumor progression at 12 months (79% vs. 30%, p = 0.02) and median survival (13.1 vs. 27.2 months, p = 0.09) were worse for patients with glioblastoma multiforme compared to anaplastic astrocytoma or oligodendroglioma. The median overall survival was shorter for patients when failure included neuraxis dissemination (n=8) compared to local failure alone (n=5), 9.6 vs. 13.8 months, p = 0.08. Three long-term survivors with WHO grade III tumors were alive with follow-up ranging from 88–239 months.

Conclusions—High-grade spinal cord primary tumors in children have a poor prognosis. The propensity for neuraxis metastases as a component of progression after RT suggests the need for more aggressive therapy.

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Keywords

Radiotherapy; Pediatrics; CNS Neoplasms; Spinal Cord Tumors

Introduction

Tumors arising in the spinal cord are uncommon, comprising less than 5% of all central nervous system (CNS) tumors. In adults, spinal tumors are often extramedullary and benign, whereas the majority of cases in children are intramedullary tumors and typically low-grade astrocytomas and ependymomas.[1]

High-grade spinal cord tumors (HGSCT) are even rarer, comprising less than 1% of all central nervous system tumors seen in children and adolescents. There are relatively few reported experiences describing the treatment of HGSCT in the pediatric population.[2–6] Though there are no established guidelines for the treatment of these rare tumors, patients at St. Jude Children's Research Hospital (SJCRH) have been consistently treated with maximal surgical resection followed by postoperative radiation therapy. In most cases, chemotherapy is also given, but the optimal type and method of delivering chemotherapy is not clear.

In this report we update the previously described institutional experience treating pediatric patients with HGSCT.[7] The objective of this study is to provide a guideline for clinicians regarding treatment outcomes in these rare cases.

Materials and Methods

Patient Characteristics

Between March 1981 and October 2007, 17 patients with high-grade spinal cord tumors, according to the World Health Organization classification (WHO grade III-IV), were treated at St. Jude Children's Research Hospital and LeBonheur Children's Medical Center in Memphis, Tennessee. The median age of patients in this series was 11 years (range 3 – 23 years). There were 10 female and 7 male patients. The records of these patients were retrospectively reviewed, with approval from the institutional review board. Information obtained from these records included: diagnosis, date of birth, sex, race, date of histologic diagnosis, therapeutic intervention, relapse, death, and extent of disease at presentation and relapse, extent of surgery, use of chemotherapy, and doses and volumes of radiation therapy. The relevant clinical and treatment-related parameters are detailed in Tables 1 and 2.

Surgery

All patients in this series initially underwent a biopsy or an attempt at resection. Three patients were considered to have had a gross total resection (GTR) of their primary tumor based on the impression of the operating surgeon and postoperative imaging. The resection was considered to be subtotal (STR) in 11 patients, and was characterized as a biopsy only in the remaining 3 patients.

Pathology and Extent of Disease at Diagnosis

Included in this study are patients with histologically confirmed primary high-grade tumors of the spinal cord: glioblastoma multiforme (GBM, WHO grade IV, n = 7), anaplastic astrocytoma (AA, WHO grade III, n = 8), and anaplastic oligodendroglioma (AO, WHO grade III, n = 2). Neuro-imaging was performed in all patients (computed tomography (CT) myelogram or magnetic resonance (MR) imaging). All primary lesions originated in the cervical or thoracic spine. Four patients had imaging evidence of metastatic or multi-focal disease at presentation. Four of 17 patients underwent cerebrospinal fluid evaluation, however, no patient had cytologic evidence of tumor dissemination at presentation.

Radiation Therapy and Chemotherapy

All patients received radiation therapy following surgery. Seven patients were initially treated with craniospinal irradiation (CSI) over a range of 34.2–48.6 Gy, followed by a boost to gross residual tumor, while ten patients received only local radiotherapy. One patient received CSI at the time of recurrence. The median dose to the primary site for all patients was 52.2 Gy (range 38–66 Gy). One patient received 66 Gy in 1.1 Gy twice daily fractions, while five patients received less than 50 Gy for a variety of clinical indications. Three patients received planned chemotherapy prior to irradiation, and 4 received concurrent chemotherapy with irradiation; otherwise chemotherapy was given only at the time of recurrence (n = 9).

Definitions of Endpoints

Progression-free survival (PFS) was measured from day 1 of radiotherapy to the time of imaging evidence of disease progression. Overall survival (OS) was measured from the date of histologic diagnosis to the date of death from any cause. Failures were classified as local, diffuse or combined, and calculated from the date of starting radiotherapy to the date of failure. Patients categorized as diffuse failures had imaging evidence of neuraxis metastases. In defining the extent of disease and location of failure, the following system was adopted: M1 - positive cerebrospinal fluid cytology, M2 - intracranial leptomeningeal disease, M3 - spinal leptomeningeal disease, and M4 - extra-neuraxis dissemination.

Statistical Analysis

Survival curves were computed to estimate PFS and OS using Kaplan-Meier methodology.[8] The cumulative incidences of local and diffuse failures were calculated by Gray's method.[9]

Results

The median PFS and OS were 10.8 and 13.8 months, respectively. Patients with WHO grade IV tumors had shorter PFS (7.8 months vs. 17.2 months, p = 0.11) and OS (13.1 months vs. 27.2 months, p = 0.09) than those with WHO grade III tumors. The risk of local progression at 12 months was 79% for WHO grade IV tumors compared to 30% for WHO grade III tumors (p = 0.02). These results are shown in Figures 1 and 2. Factors such as gender, race, radiotherapy volume (local vs. CSI), extent of surgical resection, and presence of neuraxis dissemination at presentation did not significantly impact OS or PFS in this small cohort.

Four patients had neuraxis metastases at the time of presentation and four patients with initially localized tumor developed neuraxis dissemination at the time of failure. For the patients who had diffuse failure (n = 8), the median OS was 9.6 months compared to 13.8 months for those with local failure (p = 0.08). Seven patients had evidence of intracranial dissemination, either at diagnosis or at failure, which represented 41% of the cohort. Early treatment failures predicted for a shorter OS. Patients with neuroimaging evidence of progression less than 3 months from completion of radiotherapy had a median OS of 8.6 months, compared to 17.3 months for those who did not progress by 3 months (p = 0.0002).

Only three long-term survivors were noted, all with AA; a fourth survivor with GBM and without evidence of progression was only eight months post-therapy at the time of data analysis. These patients were alive 88, 150, and 239 months post-treatment. Their medical records were assessed for function and performance. The 150-month survivor was diagnosed with an AA at the age of 11 years at C1-C5 and received focal irradiation to 50 Gy following gross total resection. Despite being quadraparetic postoperatively, this patient was fully employed at last follow-up, and suffered only from loss of left hand fine motor function. The 239-month survivor was diagnosed with an AA at 5 years of age, located at C3-T2 with synchronous intracranial leptomeningeal dissemination. The patient received 4 courses of pre-irradiation cisplatin and etoposide, after which 38 Gy CSI was administered followed by a boost to the brain and primary site to 48 Gy. This patient continued to suffer from a seizure disorder that was present at initial diagnosis, and subsequently developed multiple endocrine deficiencies. The 88-month survivor was diagnosed at age 3 with an AA at C3-T8. He underwent subtotal resection, followed by CSI of 34.2 Gy, with a boost to the primary tumor to 52.2 Gy. He did not receive chemotherapy, and had no evidence of disease progression at last follow-up. However, post-therapeutic sequelae include cataracts, kyphoscoliosis, spastic paraparesis, and endocrine dysfunction.

Discussion

Spinal cord neoplasms in children are uncommon entities, and high-grade tumors in this population are found even more rarely. They typically arise in the cervical or thoracic cord and account for less than 1% of all cases of central nervous system tumors. It remains uncertain whether HGSCT in children are clinically or biologically similar to their adult counterparts. Most series describing the treatment of spinal cord tumors in children also include adult patients with low-grade astrocytoma and ependymoma.[10–13] To our knowledge, our cohort is among the largest series specifically documenting the clinical outcomes of pediatric patients with HGSCT.

The rarity of pediatric HGSCT and short survival of these patients limits our ability to reliably identify prognostic factors that impact survival. Among primary spinal cord tumors, histological subtype and grade appear to be the most consistent predictive factors for outcome across many series.[3, 10, 14–16] Grade IV spinal cord tumors have a dismal prognosis, and only a handful of reports in the literature have documented long-term survivors with spinal GBM.[17] Our data is consistent with this observation, as there were no long-term survivors among our cohort of children with grade IV spinal tumors. Although the overall and progression-free survival outcomes did not reach statistical significance when

stratified by histology, local failure was statistically worse for grade IV tumors than for grade III tumors. Of note, there were three long-term survivors with grade III tumors, however all experienced significant co-morbidities as a consequence of their disease.

Prognostic factors other than histological grade are not as well described in patients with primary spinal cord tumors. In our cohort, a short time to clinical failure did predict for poor OS. Patients who progressed within 3 months after radiotherapy had a significantly worse survival than those whose disease remained controlled at that time point. In other series, a shorter duration of symptoms before diagnosis was also predictive of a poor outcome.[3]

Although there is no well-defined standard therapy for HGSCT, maximal surgical resection and post-operative radiotherapy remains the most common treatment strategy. In a child presenting with an intramedullary spinal cord tumor, the very rapid progression of signs and symptoms alone may indicate malignant histology. After high resolution imaging of the entire neuraxis, the child should be taken to surgery with the limits of the tumor extension noted on MR confirmed with ultrasound prior to opening of the dura. Dorsal myelotomy should be performed over the length of the tumor, even if limited resection or biopsy are planned, to minimize post-surgical morbidity resulting from edema. At our institution, we advocate immediate biopsy and frozen section and in the case of malignant tumors, very judicious resection removing only frankly obvious abnormal tissue. Because malignant tumors are infiltrative, radical resection leads to permanent post-operative deficits.[12] Because the extent of surgical resection appears to be an unreliable prognostic factor across many series, there is no proven advantage to radical resection. In thoracic tumors, there may be a temptation to perform a cordectomy, especially at the time of tumor progression, in those with no evidence of metastatic disease. Fred Epstein, an experienced spinal cord tumor surgeon, reported a series of malignant spinal cord tumors.[18] Cordectomy was performed three times and was unsuccessful in all three (personal communication). We have not performed cordectomy at our institution.

Despite aggressive local treatment, both local and distant leptomeningeal failures are common. It has been noted that high-grade tumors of the spinal cord, especially GBM, are associated with a high rate of ultimate leptomeningeal dissemination, up to 58% in some series and 47% in our cohort.[4, 15, 18–21] This rate of neuraxis dissemination with HGSCT appears to be higher than that observed with high-grade astrocytomas arising in the brain, which occurs in approximately 5–10% at diagnosis.[22–24] It is felt that this discordant rate of leptomeningeal dissemination may be related to the closer anatomic proximity of spinal tumors to the subarachnoid space compared to their intracranial counterparts.

Patients with documented leptomeningeal disease at diagnosis are generally candidates for craniospinal irradiation, but it is unclear whether prophylactically treating such an extensive volume should be considered in HGSCT patients without radiographic evidence of distant spread. Current imaging techniques to evaluate dissemination of disease prior to radiation therapy may in fact underestimate the actual extent of disease. However, in our series, 6 of the 8 diffuse failures progressed locally in addition to regions beyond the primary site, therefore, improved definition of the extent of disease may not be sufficient to improve outcome. The ability to identify occult disease impacts radiotherapeutic management and the

potential to achieve disease control. Unfortunately, the discrepancy between the dose that can be delivered to the neuraxis with acceptable toxicity and the dose required to sterilize even microscopic amounts of a high-grade glial tumor ultimately results in disseminated failures in a large proportion of patients. It seems appropriate that the radiotherapy dose and volume should be determined on an individual basis.

It also remains unclear whether GBM arising in the spinal cord are biologically analogous to those arising in the brain. Although spinal GBM and intracranial GBM have different clinical courses, it is felt that they are histologically identical entities. Despite aggressive surgery and post-operative radiotherapy, the poor outcomes of HGSCT patients suggest that alternative modalities should be investigated. Given the lack of prospective evidence in this rare disease, it is reasonable to take treatment strategies that were successful in patients with intracranial GBM and apply them to children with spinal GBM. Unfortunately, attempts at improving local control in intracranial GBM by radiation dose escalation, brachytherapy, radiosensitizers, and altered fractionation have yielded no improvements over standard therapy.[25–29]

However, chemotherapy has recently been established as a standard of care in adult patients with intracranial GBM. A randomized trial demonstrated that the addition of concurrent and adjuvant temozolomide to radiotherapy improves both OS and PFS in adults with GBM.[30] Temozolomide, an oral alkylating agent with penetration of the blood-brain-barrier, is being investigated in the pediatric population.[31–33] There is strong evidence that adults who exhibit methylation of the methylguanine-DNA methyltransferase (MGMT) promoter have a better outcome with alkylating agents such as temozolomide, and the same appears to be true for children.[34–36] Analyzing molecular factors such as MGMT status may help guide therapy and predict those who are predisposed to a more favorable outcome with combined radiotherapy and chemotherapy. The role of intra-thecal chemotherapy in the treatment of HGSCT is undefined. Likewise, targeted therapies such as inhibitors of the epidermal growth factor receptor or the vascular endothelial growth factor receptor remain investigational.

In conclusion, primary high-grade spinal tumors in children are very rare and carry a poor prognosis. The role of prophylactic craniospinal irradiation remains unclear, as a high proportion of patients die with both local failure and distant leptomeningeal dissemination despite aggressive therapy. The dose of radiotherapy that can be successfully delivered to the neuraxis is limited by the tolerance of surrounding normal spinal cord parenchyma and by the intrinsic resistance of malignant glioma cells. Further investigation into the optimal combination of surgery, radiation, chemotherapy, and targeted therapy is warranted. However, the rarity of this disease makes prospective evaluation difficult to perform in this population.

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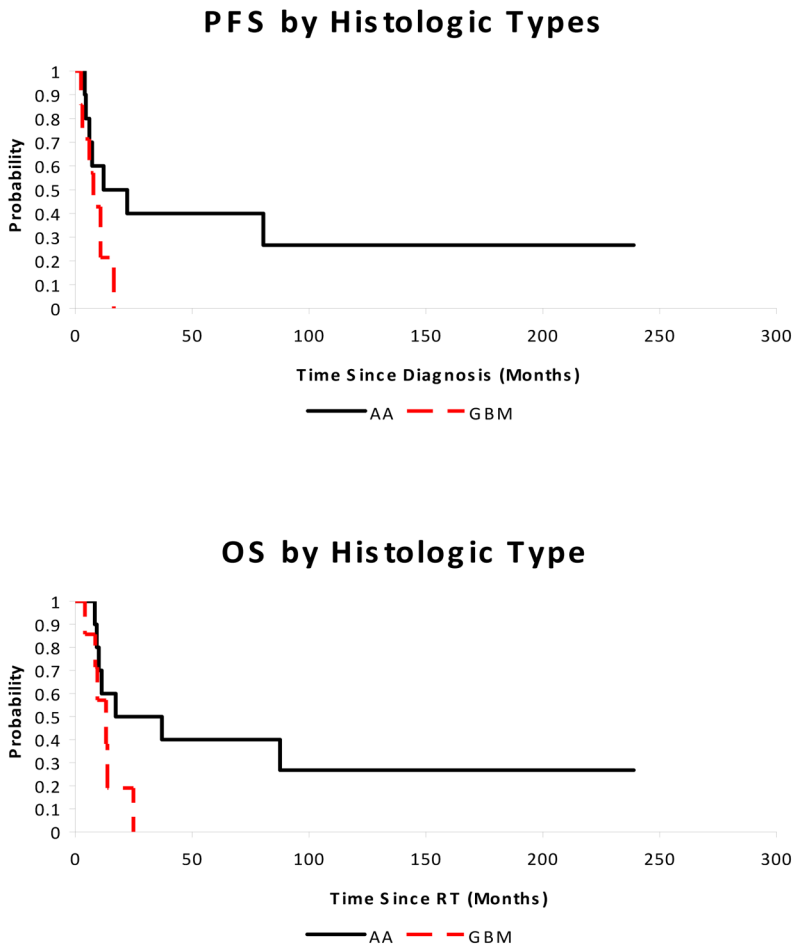

Figure 1.

Progression-free (p = 0.11) and overall survival (p = 0.09) for pediatric patients with high-grade spinal cord tumors by histologic subtype. AA = anaplastic astrocytoma or oligodendroglioma. GBM = glioblastoma multiforme.

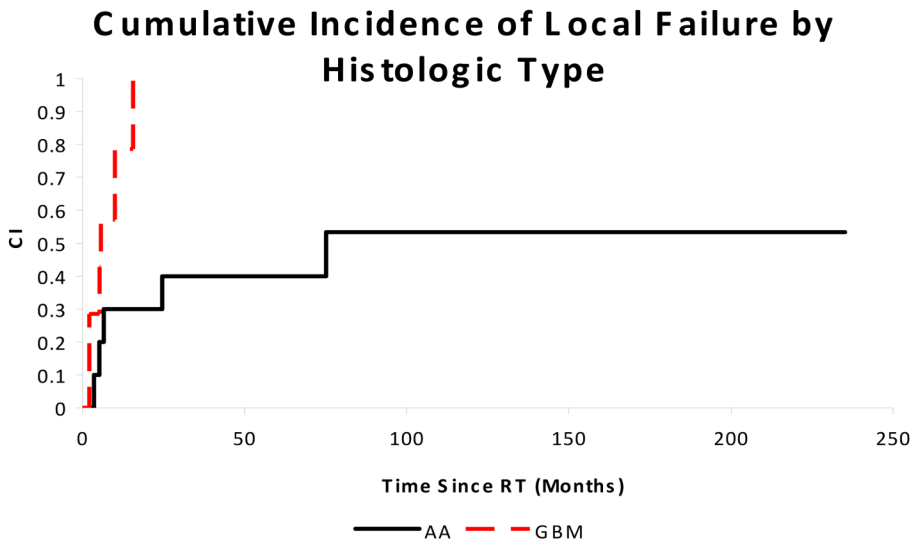

Figure 2.

Cumulative incidence of local failure (p = 0.02) for pediatric patients with high-grade spinal cord tumors by histologic subtype. AA = anaplastic astrocytoma or oligodendroglioma. GBM = glioblastoma multiforme.

Table 1

Patient characteristics and outcomes.

	N	%
Gender		
Female	10	59%
Male	7	41%
Histology		
AA	8	47%
AO	2	12%
GBM	7	41%
Surgery		
Biopsy	3	18%
STR	11	64%
GTR	3	18%
Primary EBRT		
CSI	7	41%
Focal	10	59%
Failure Type		
None	4	24%
Local only	5	29%
Diffuse only	2	12%
Local + diffuse	6	35%
Leptomeningeal disease		
None	9	53%
At presentation	4	24%
At failure	4	24%
Intracranial disease		
None	10	59%
At presentation	3	18%
At failure	4	24%

AA = anaplastic astrocytoma. AO = anaplastic oligodendroglioma. GBM = glioblastoma multiforme. STR = subtotal resection. GTR = gross total resection. CSI = craniospinal irradiation.

Table 2

Individual clinical and treatment characteristics of pediatric patients with high-grade spinal cord tumors.

Age, sex	Site	Histology	Surgery	Radiation (Gy)	Failure	Time to Failure
12, M	T11-L1 (M3)	AO	STR	CSI 48.6	Diffuse	7 months
9, F	T11-L2	AA	STR	Focal 38	Local + diffuse	1 month
23, F	C3-C5	AO	GTR	Focal 50.4	Local	24 months
11, F	C1-T1	AA	GTR	Focal 50	None	Stable 150 months
5, F	C3-T2 (M2)	AA	BX	CSI 38, focal 48	None	Stable 239 months
10, M	C1-C2, T6-T8 (M2)	AA	STR	CSI 48.4, focal 66	Local + diffuse	2 months
10, M	C2-T6	AA	STR	Focal 54	Local	6 years
11, F	C2-C7	AA	STR	Focal 54	Diffuse	1 month
18, F	C7-T7	GBM	GTR	Focal 55	Local	10 months
14, M	C7-T2	GBM	STR	CSI 38.5	Local	4 months
10, F	C3-T7	GBM	BX	Focal 48	Local + diffuse	14 months
17, M	C1-C5	GBM	STR	Focal 54	Local	4 months
13, F	C1-T1	GBM	BX	Focal 54	Local + diffuse	1 month
3, M	C3-T8 (M3)	AA	STR	CSI 34.2, focal 52.2	None	Stable 88 months
15, F	T10-T11	GBM	STR	CSI 39.6, focal 51.3	Local + diffuse	1 month
12, M	T12	AA	STR	Focal 54	Local + diffuse	4 months
9, F	T2-T7	GBM	STR	CSI 36, focal 54	None	Stable 10 months