Intrinsic tumors of the spinal cord comprise 1 to 2% of primary central nervous system tumors that occur in the pediatric age group. Approximately 85% of these spinal cord tumors are of astrocytic lineage, and 10 to 15% of these will have histological features consistent with a high-grade astrocytoma (HGA) (that is, anaplastic astrocytoma [AA] or glioblastoma multiforme [GBM]). The histological grade of astrocytic tumors of the spinal cord has prognostic implications similar to those found in other central nervous system sites. Low-grade spinal cord astrocytomas, in spite of their infiltrative behavior, are amenable to radical surgical resection. The median survival time of pediatric patients is usually longer than 10 years, and the pattern of relapse is local recurrence. Conversely, children with high-grade tumors of the spinal cord have a median survival time of less than 7 months. These patients develop not only local recurrences but frequently widespread leptomeningeal metastases. No uniform surgical, radiotherapeutic, or chemotherapeutic approach has been validated for such tumors. The Children’s Cancer Group (CCG) 945 High-Grade Astrocytoma Committee devised a pilot study to collect natural history information and explore the benefit of an experimental multimodality treatment in children with newly diagnosed high-grade astrocytomas arising within the spinal cord. The protocol included pre- and postradiotherapy and “8-drugs-in-1-day” (8-in-1) chemotherapy following maximum surgical resection. Craniospinal radiotherapy was recommended; however, it was not always delivered.

Clinical Material and Methods

From November 1988 to April 1992, 18 patients with primary spinal cord tumors were registered in the CCG 945 protocol. Eligibility requirements included age less than 21 years and an institutionally rendered histopathological diagnosis of a primary spinal cord HGA. The pro-
Protocol was approved by all the local Institutional Review Boards, and informed consent was obtained from all patients and/or their parents. The extent of resection and presence of metastases were evaluated prior to the initiation of any therapy. Patients underwent contrast-enhanced axial computerized tomography (CT) or magnetic resonance (MR) imaging of the head, as well as either myelography or MR evaluation of the spine. Magnetic resonance imaging of the spine was performed in 17 of 18 patients; gadolinium contrast was used in 11. Myelography was performed in one patient. The extent of tumor resection was based on the surgeon’s estimation at the time of surgery and was defined as: biopsy, less than 10% of tumor removed; partial resection, 10 to 50% removed; subtotal resection, 50 to 90% removed; near-total resection, greater than 90% removed; and gross-total resection, no visible tumor remaining following surgery.

The diagnosis of HGA by the institution’s pathologist was used to confirm initial study eligibility. Centralized neuropathology review was used to determine the final valid study group. If there was discordance between the institution’s pathologist and the study reviewer as to the diagnosis, additional slides and blocks were requested for further review. Pathological diagnoses were categorized into four groups: GBM, AA, other eligible gliomas (such as mixed malignant glioma or anaplastic oligodendroglioma), and discordant gliomas (such as low-grade astrocytoma or ependymoma). In this report, data analysis was limited to the patients in the confirmed group, that is, those with a review diagnosis of high-grade astrocytoma, unless otherwise specified.

**Treatment Protocol**

All patients with primary spinal cord tumors were non-randomly assigned to the 8-in-1 chemotherapy regimen. Within 28 days of surgery, a 4-week induction course of two cycles of 8-in-1 chemotherapy was initiated. Each cycle was separated by an interval of 14 days and was followed by radiotherapy. Four weeks after the completion of radiotherapy, maintenance chemotherapy consisting of eight cycles of the 8-in-1 regimen delivered at 4- to 6-week intervals was administered. Radiotherapy was given concomitantly with the second cycle. The primary spinal cord lesion was treated together with a minimum margin of 2 cm of normal tissue superiorly and inferiorly. The entire width of the vertebra was treated together with a 1-cm margin laterally. Because groupwide consensus on the optimum radiotherapy volume could not be reached, several plans were allowed. For children older than 3 years, “prophylactic” craniospinal axis irradiation was recommended up to a dose of 3600 cGy with an 1800-cGy boost to the primary tumor. Patients diagnosed with diffuse spinal cord metastases were to receive 3600 cGy to the craniospinal axis and 1440 cGy to the entire spinal cord.

Two therapeutic alternatives were offered for children between 24 and 36 months of age at the time of diagnosis: 2340 cGy to the craniospinal axis with a 2160-cGy boost to the primary site after 10 cycles of chemotherapy; or 4500 cGy alone to the primary tumor site after two cycles of 8-in-1. Children younger than 24 months at the time of diagnosis could be treated with 10 cycles of 8-in-1 chemotherapy alone with the intent of delaying or possibly avoiding radiotherapy.

Interval CT scans or MR images were obtained following preirradiation chemotherapy, at the conclusion of radiotherapy, and every 3 months thereafter. Patients were considered to have achieved complete response if no tumor was visible on radiography; partial response if there was greater than 50% reduction in tumor size; minor response if there was greater than 25% but less than 50% reduction in tumor size; stable disease if there was no change or less than 25% reduction; or progressive disease if there was emergence of any new lesion or enlargement of an existing lesion by 25%. Evaluation of response or relapse was based on the institution’s neuroradiologist’s opinion.

**Statistical Analysis**

Progression-free survival time was measured from the date of study entry to disease progression, to death without progression, or to the last day of contact for surviving patients in whom there was no progression. The distributions of progression-free survival and overall survival times were estimated using the Kaplan–Meier method, with standard errors of these estimates based on calculations proposed by Peto and coworkers.1

**Results**

Although 18 patients with an initial institutional diagnosis of HGA were entered into the study, the diagnosis of HGA could be confirmed by centralized neuropathology review in only 13. These 13 patients constituted the confirmed group. The mean age of this group was 7 years (range 1–15 years). The median duration of symptoms before surgery was 2.5 months (range 0.5–8 months) (Table 1). The tumor was located in the cervical area in eight patients. Six patients had leptomeningeal metastases at the time of diagnosis as determined by MR examination. A cerebrospinal fluid cytological examination was performed in six patients, but no tumor cells could be documented. Three patients had hydrocephalus at diagnosis, which was graded as severe in two patients and mild in one. Two of these three patients had leptomeningeal metastases at diagnosis (Cases 7 and 12; Table 1). The hydrocephalus was observed on the initial staging MR images.

All 13 patients underwent surgery within 16 days of the initial imaging study. The mean length of the primary spinal cord tumor was 5 cm (range 1–10 cm). Four patients had rostral and/or caudal cysts accompanying their lesion. More than 90% of the tumor was removed in six patients, whereas biopsy specimens were obtained in three patients. The other four patients underwent a 10 to 90% resection (Table 1). The extent of resection was verified by CT or MR evaluation in five patients.

The institutions’ pathologists diagnosed 13 of the 18 patients with AA, three with GBM, one with mixed AA, and another with mixed anaplastic glioma. The revised diagnoses after central review were: eight AAs, four GBMs, one mixed anaplastic glioma, one pilocytic astrocytoma, and three low-grade astrocytomas (including oligodendroglioma). In one patient, there was insufficient tissue for diagnosis. Thus, five patients were considered to have a discordant diagnosis.
Five of the 13 patients in the confirmed group completed all 10 cycles of chemotherapy, and eight patients completed eight cycles or more. Treatment was stopped after four cycles in one patient (Case 3) who withdrew from the study. Ten of the 13 patients received initial radiotherapy; five patients older than 3 years of age at diagnosis received craniospinal radiotherapy. Four patients were treated with spinal field radiotherapy and one with local field (Table 2). Radiotherapy was deferred in three patients, two of whom received local field radiotherapy at recurrence (Cases 1 and 12). One patient (Case 6) with an AA never received radiation and is alive without evidence of disease. She had a local relapse 9 months after initial diagnosis and received myeloablative chemotherapy with bone marrow rescue.

Chemotherapeutic Toxicity

Ten of the 13 patients experienced at least one significant side effect of chemotherapy. The most frequently reported toxicity, accounting for 75% of all toxic episodes, was myelosuppression. One patient developed a transient renal impairment, and bilateral hearing loss occurred in another, both presumably caused by cisplatin. There were two deaths resulting from a cause other than tumor: one patient (Case 12) died of renal failure that was caused in part by chemotherapy and the second patient (Case 1) died while undergoing orthopedic surgery 37 months after diagnosis.

Treatment Outcome

The 5-year progression-free survival and overall sur-

---

**TABLE 1**

Clinical data obtained in 13 patients with HGAs of the spinal cord in the confirmed-histology group*  

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Duration of Symptoms (mos)</th>
<th>Institution’s Review Diagnosis</th>
<th>Central Review Diagnosis</th>
<th>Primary Metastases at Diagnosis</th>
<th>Extent of Resection</th>
<th>Hydrocephalus</th>
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<tbody>
<tr>
<td>1</td>
<td>4, M</td>
<td>4.5 GBM</td>
<td>GBM</td>
<td>GBM</td>
<td>holocord</td>
<td>yes</td>
<td>part</td>
</tr>
<tr>
<td>2</td>
<td>12, F</td>
<td>2.5 AA</td>
<td>AA</td>
<td>AA</td>
<td>thoracic</td>
<td>no</td>
<td>sub</td>
</tr>
<tr>
<td>3</td>
<td>12, M</td>
<td>4.5 AA</td>
<td>AA</td>
<td>AA</td>
<td>cervical</td>
<td>yes</td>
<td>gross</td>
</tr>
<tr>
<td>4</td>
<td>1.5, M</td>
<td>4.5 AA</td>
<td>AA</td>
<td>AA</td>
<td>cervical &amp; thoracic</td>
<td>yes</td>
<td>biop</td>
</tr>
<tr>
<td>5</td>
<td>13, F</td>
<td>1 GBM</td>
<td>GBM</td>
<td>GBM</td>
<td>cervical</td>
<td>no</td>
<td>gross</td>
</tr>
<tr>
<td>6</td>
<td>1, F</td>
<td>2.5 AA</td>
<td>AA</td>
<td>AA</td>
<td>cervical</td>
<td>no</td>
<td>near</td>
</tr>
<tr>
<td>7</td>
<td>5, M</td>
<td>8 AA</td>
<td>AA</td>
<td>AA</td>
<td>thoracic</td>
<td>yes</td>
<td>biop</td>
</tr>
<tr>
<td>8</td>
<td>1, F</td>
<td>1.5 AA</td>
<td>AA</td>
<td>AA</td>
<td>thoracic</td>
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<td>near</td>
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<td>2.5 AA</td>
<td>AA</td>
<td>AA</td>
<td>thoracic</td>
<td>no</td>
<td>part</td>
</tr>
<tr>
<td>10</td>
<td>4, M</td>
<td>2.5 AA</td>
<td>AA</td>
<td>AA</td>
<td>cervicomедullary</td>
<td>yes</td>
<td>biop</td>
</tr>
<tr>
<td>11</td>
<td>8, M</td>
<td>0.5 AA</td>
<td>AA</td>
<td>AA</td>
<td>cervical</td>
<td>no</td>
<td>gross</td>
</tr>
<tr>
<td>12</td>
<td>15, M</td>
<td>1.5 GBM</td>
<td>GBM</td>
<td>GBM</td>
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<td>part</td>
</tr>
<tr>
<td>13</td>
<td>6, F</td>
<td>1 mixed AA/oligo</td>
<td>mixed AA/oligo</td>
<td>mixed AA/oligo</td>
<td>cervical &amp; thoracic</td>
<td>no</td>
<td>near</td>
</tr>
</tbody>
</table>

* Gross = gross total; lepto = leptomeningeal; near = near total; oligo = oligodendroglioma; part = partial; sub = subtotal.

**TABLE 2**

Treatment and follow-up review of 13 patients with HGAs in the confirmed-histology group*  

<table>
<thead>
<tr>
<th>Case No.</th>
<th>No. of Chemo Cycles</th>
<th>Volume &amp; Dose (cGy)</th>
<th>Response to pre-RT Chemo</th>
<th>Survival (mos)</th>
<th>Site of Tumor Relapse</th>
<th>Follow-Up Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>37</td>
<td>prim (ana oligo at reop)</td>
<td>died of toxicity</td>
</tr>
<tr>
<td>2</td>
<td>10</td>
<td>5400 3600 3600</td>
<td>complete</td>
<td>17 20</td>
<td>prim &amp; lepto</td>
<td>died of tumor</td>
</tr>
<tr>
<td>3</td>
<td>4</td>
<td>4500 4500 0</td>
<td>mixed</td>
<td>60 60</td>
<td>none</td>
<td>alive*</td>
</tr>
<tr>
<td>4</td>
<td>10</td>
<td>4500 4500 0</td>
<td>mixed</td>
<td>60 60</td>
<td>none</td>
<td>alive w/ disease</td>
</tr>
<tr>
<td>5</td>
<td>8</td>
<td>5400 3600 3600</td>
<td>not measurable</td>
<td>12 15</td>
<td>prim &amp; lepto</td>
<td>died of tumor</td>
</tr>
<tr>
<td>6</td>
<td>9</td>
<td>0</td>
<td>0</td>
<td>9 57</td>
<td>prim</td>
<td>alive NED</td>
</tr>
<tr>
<td>7</td>
<td>10</td>
<td>1440 0 0</td>
<td>stable</td>
<td>68 68</td>
<td>none</td>
<td>alive w/ disease</td>
</tr>
<tr>
<td>8</td>
<td>8</td>
<td>4500 4500 0</td>
<td>not measurable</td>
<td>11 15</td>
<td>prim</td>
<td>died of tumor</td>
</tr>
<tr>
<td>9</td>
<td>3</td>
<td>4500 4050 3000</td>
<td>mixed</td>
<td>93 93</td>
<td>none</td>
<td>alive w/ disease</td>
</tr>
<tr>
<td>10</td>
<td>3</td>
<td>5580 3600 0</td>
<td>stable</td>
<td>6 14</td>
<td>prim &amp; lepto</td>
<td>died of tumor</td>
</tr>
<tr>
<td>11</td>
<td>10</td>
<td>5400 3600 3600</td>
<td>not measurable</td>
<td>58 58</td>
<td>none</td>
<td>alive NED</td>
</tr>
<tr>
<td>12</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>1 8</td>
<td>prim &amp; lepto</td>
<td>died of toxicity w/ disease</td>
</tr>
<tr>
<td>13</td>
<td>10</td>
<td>5460 3600 3600</td>
<td>partial</td>
<td>48 48</td>
<td>none</td>
<td>alive w/ disease</td>
</tr>
</tbody>
</table>

* Ana = anaplastic; chemo = chemotherapy; lepto = leptomeningeal; NED = no evidence of disease; oligo = oligodendroglioma; OS = overall survival; PFS = progression-free survival; prim = primary; RT = radiotherapy.
† Patient withdrew from study.
vival times for patients in the confirmed group were 46 ± 14% and 54 ± 14%, respectively (Fig. 1). Ten of 13 patients could be evaluated for response to 8-in-1 preirradiation chemotherapy (Table 2). There were five objective responses (one patient achieved complete response; two, partial response; and two, mixed response) and one patient suffered from progressive disease. Four patients had stable disease.

Seven of the 13 patients were alive at follow-up review, five with clinically and radiologically stable disease and two with no evidence of disease. Seven patients developed tumor recurrence or progression, three at the primary site alone and four at the primary site with leptomeningeal metastases. Six patients died between 8 and 37 months after entering the study (four due to disease progression, one due to toxicity, and another due to unrelated causes). Three of six patients presenting with leptomeningeal disease have died. One of the four patients with leptomeningeal relapse died of toxicity. Autopsy studies performed in Cases 2, 5, and 10, revealed residual tumor at the primary site in addition to diffuse leptomeningeal metastases.

**Discussion**

Therapeutic advances in the management of high-grade gliomas of the spinal cord have been hampered by the rarity of incidence (to date, fewer than 20 cases per year in the United States) and the lack of uniform treatment. Most institutional series report a relatively few number of cases, and patients typically have limited survival times. In 1988, CCG attempted to pool its clinical resources and appended a single-arm study to a larger randomized trial (CCG 945) for children with newly diagnosed intracranial HGAs. Over a 5-year period the protocol was successful (CCG 945) for children with newly diagnosed intracranial high-grade supratentorial gliomas. Modifications the in radiotherapy protocol were made for patients younger than 3 years of age at diagnosis.

The 5-year progression-free survival and overall survival times of 46 ± 14% and 54 ± 14%, respectively, appear more favorable than previously reported. There were four patients with GBM, eight with AA, and one with mixed anaplastic glioma. The overall toxicity was tolerable, and there was one treatment-related death. In a combined pediatric and adult series a 3-year survival rate of less than 10% in 19 patients with a median survival time of 6 months was reported.

According to the surgeon’s assessment, more than 90% of tumor was removed in six (46%) of 13 patients. This represents a higher proportion of radical resections than achieved in previously reported pediatric spinal cord tumor series that consisted primarily of patients with low-grade astrocytomas. For example, Reimer and Onofrio treated 32 children and adolescents with spinal cord astrocytoma, performing biopsies in 69%, partial resections in 25%, and near-total removal in only 6%. Radical resections have recently been advocated by others. The presence of known leptomeningeal metastases at diagnosis in six patients may have deterred the surgeons from attempting radical resection. Although the benefits of radical resection in children with low-grade spinal cord glioma are compelling, the role of radical surgery in HGA is less clear.

The major cause of treatment failure, as observed in seven of our 13 patients, was progressive disease at the primary site. Although four patients had additional leptomeningeal metastases, two of these patients showed evidence of leptomeningeal metastases at diagnosis. All three patients who were studied at autopsy had both local recurrence and diffuse leptomeningeal metastases. This predisposition for leptomeningeal spread has been observed by others. However, its presence in six of 13 patients at diagnosis in our study, before surgical intervention and another four at recurrence, is unprecedented and may reflect the sensitivity of MR imaging for detecting this condition.

Salazar reported on 13 pediatric patients with HGA of the cerebellum, six of whom were treated with cranial irradiation and seven with craniospinal radiotherapy. Five of the six patients receiving only cranial irradiation developed spinal metastases, with a mean survival time of 10 months, whereas all seven patients receiving craniospinal radiotherapy remained free of spinal metastases for 1 to 4 years. As many as 30% of children with HGA of the brainstem may also develop leptomeningeal spread. Thus, the high incidence of leptomeningeal spread seen at the time of progression in previously reported patients with spinal cord HGA led to the recommendation of craniospinal irradiation in this study. We cannot assess the benefit of craniospinal radiotherapy in our study because of the limitations of patient enrollment and protocol design.

The introduction of gadolinium-enhanced MR studies has greatly facilitated the diagnosis and management of spinal cord tumors. Compared with myelography, it is less invasive and can be performed repeatedly with little risk of morbidity. Magnetic resonance imaging permits the distinction between cyst and solid tumor, readily identifies...
leptomeningeal metastases when gadolinium is administered, and allows assessment of the degree of surgical resection, response to therapy, and relapse. In our study, MR imaging was used to monitor all but one patient.

Conclusions
We conclude that the prognosis for children with primary HGA of the spinal cord is poor. The majority of patients will either present with or develop leptomeningeal metastases, but the major treatment challenge is still local tumor control. Although the overall survival rates in our patients appear better than previously reported, it is difficult to assign benefit to specific aspects of our protocol (radical surgery, craniospinal radiotherapy, or neoadjuvant/adjunctive 8-in-1 chemotherapy). In fact, in the parent protocol for supratentorial high-grade astrocytomas in CCG 945, 8-in-1 chemotherapy was not superior to the conventional adjuvant chemotherapy (CCNU and vincristine). Given the overall poor prognosis for children with this disease in the spinal cord in our series and those reported previously, our present recommendations would still include radical surgery, careful staging, and more intensive chemotherapy followed by either spinal or craniospinal radiotherapy in children older than 4 years of age. The rarity of this tumor requires study by a cooperative group and centralized review of the histopathological findings.

Appendix

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References


