Malignant astrocytoma following radiotherapy for craniopharyngioma

ABSTRACT

Radiation induced gliomas are uncommon. Occurrence of glioma following radiotherapy for craniopharyngiomas is extremely uncommon and only eight case reports have been so far published. We present our experience with one similar case of temporal gliomas occurring twelve years following radiotherapy for a sub totally excised craniopharyngioma. Although the exact mechanism of gliomas formation is unclear, their occurrence following conventional radiotherapy is a distinct possibility and signifies a poor prognosis.

KEY WORDS: Craniopharyngioma, gliomas, radiotherapy

INTRODUCTION

Occurrence of brain tumors such as meningiomas and sarcomas following exposure to ionizing radiation has been well-documented both in animal experiments and in earlier published case reports. However occurrence of gliomas in patients with craniopharyngiomas is extremely uncommon and only eight cases of glioma following radiotherapy for craniopharyngiomas have been reported in literature. Here we report a case of malignant temporal glioma that occurred following radiotherapy for craniopharyngioma.

CASE REPORT

This five-year-old presented to us with features of raised intracranial pressure and visual blurring in 1993. Computerized tomography (CT) scan [Figure 1] revealed the presence of cystic suprasellar lesion with rim calcification and extension into the third ventricle. The lesion was seen to obstruct the foramen of Munro and cause obstructive hydrocephalus. He underwent pericoronal parasagital craniotomy and sub total excision of the lesion. The histopathological features were suggestive of a craniopharyngioma with the cyst wall showing a predominant squamous epithelial lining. The postoperative scan showed evidence of minimal residual enhancement along the cyst wall. As our institute policy in the early 1990’s was to subject patients with subtotal excision to postoperative radiotherapy he received conventional radiotherapy of 50 Gy over seven weeks. He recovered well and was on regular follow-up and routine CT scan done in 1996 showed no evidence of recurrence. Twelve years later he presented again in October 2005 with raised intracranial pressure symptoms and endocrinological disturbances in the form of stunted growth. CT scan features revealed a large suprasellar mass with extension to sella and evidence of calcification. In addition a hypo dense lesion was seen in the left temporal lobe with patchy enhancement. MRI scan [Figure 2] confirmed the recurrence. The characteristics of the temporal lesion were typical of a glioma, which was corroborated by MR spectroscopy. He underwent left frontotemporal craniotomy, near total excision of the craniopharyngioma as well as the temporal lesion. Histopathology of the temporal lesion revealed it to be astrocytoma grade III. He was readmitted again in March 2006 in a state of altered sensorium. Emergency CT scan [Figure 3] showed recurrence of the left temporal gliomas with severe mass effect and midline shift. He underwent emergency
Menon, et al.: Radiation induced glioma

Salvati et al. have published a series of 116 reported cases of post radiation induced gliomas which includes only eight cases of gliomas [Table 1] induced following treatment for craniopharyngiomas.

Radiation induced tumors need to be validated according to the Cahan’s criteria which includes i) the tumor must originate in the previously irradiated region; ii) there must be sufficiently long time interval from irradiation and the onset of post radiation tumor; iii) the histotype of the tumor must be different from the primary one; iv) the patient must not suffer from the pathologies favoring the development of tumors such as von Recklinhausen’s disease or other phakomatosis. Our patient did not have any obvious evidence of phakomatosis and he developed a glioma twelve years later within the earlier irradiated region. He satisfies all the Cahan’s criteria and can be appropriately included as radiation induced glioma.

The biological makeup of radiation induced gliomas as expressed by their growth rate, location, age and sex distribution differs from that of spontaneous gliomas. They commonly occur in the young and a male preponderance has been observed. Location is usually supratentorial and at sites less commonly affected by primary gliomas. Gliomas occurring following craniopharyngioma are usually located in the temporal lobe. Most of the radiation induced gliomas are histologically and clinically more malignant compared to “spontaneous” gliomas and the other difference reported so far has been that some of the commonly observed oncogene mutations seen in spontaneous gliomas are not seen in radiation induce gliomas.

No definite relation of malignancy to the dosage of radiation has been reported. However it has been observed that dose of radiation might influence the histology of the tumor with low doses predisposing to gliomas and high doses predisposing to sarcoma formation. Similarly there seems to be no consistent correlation between the radiation dose and the latent period or between patients age and the latency period. However the latency period is known to have two peak incidences (five to eight years and 21-28 years) probably implying the possibility of different mechanism in gliomas formation. These findings are contrary to what is observed in radiation induced meningiomas which showed a statistically significant difference between latency after low dose and that after high dose.

Although several explanations have been proposed for the mechanism of formation of gliomas none have been satisfactorily accepted. The largest single group of patients who developed postradiation tumors of the CNS were those involved in the program of low dose radiotherapy for tinea capitis. Gliotic brain tissue seen surrounding a craniopharyngioma is supposed to be prone to gliomas formation following exposure to radiation. It is also possible that ischemic damage caused by radiation arteritis which was seen in two cases plays some role in gliomas formation.
Table 1: Reported cases of radiation induced gliomas following radiotherapy for craniopharyngioma

<table>
<thead>
<tr>
<th>Author</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Dose (Gy)</th>
<th>Latency (yrs)</th>
<th>Site of glioma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sogg (1978)</td>
<td>9</td>
<td>F</td>
<td>60</td>
<td>6</td>
<td>Right temporal and left frontal</td>
</tr>
<tr>
<td>Komaki (1977)</td>
<td>28</td>
<td>M</td>
<td>54</td>
<td>6</td>
<td>Temporal</td>
</tr>
<tr>
<td>Gutjahr (1979)</td>
<td>4</td>
<td>F</td>
<td>60</td>
<td>8</td>
<td>Cerebellum</td>
</tr>
<tr>
<td>Maat-Schieman (1984)</td>
<td>5</td>
<td>M</td>
<td>60</td>
<td>14</td>
<td>Temporal</td>
</tr>
<tr>
<td>Liwicz (1985)</td>
<td>11</td>
<td>M</td>
<td>59</td>
<td>25</td>
<td>Cerebral</td>
</tr>
<tr>
<td>Ushio (1987)</td>
<td>2</td>
<td>F</td>
<td>54.6</td>
<td>4</td>
<td>Basal ganglia</td>
</tr>
<tr>
<td>Kitanaka (1989)</td>
<td>7</td>
<td>F</td>
<td>60</td>
<td>16</td>
<td>Temporal</td>
</tr>
<tr>
<td>Kranzinger (2001)</td>
<td>14</td>
<td>F</td>
<td>60</td>
<td>4</td>
<td>Temporal</td>
</tr>
<tr>
<td>Ours (2006)</td>
<td>5</td>
<td>M</td>
<td>12</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Formation. Gliomas are known to occur more commonly after radiotherapy for acute lymphoblastic leukemia and the simultaneous administration of chemotherapy might also play an etiologic role.\(^1\) However such radiation associated gliomas are quite rare considering the population of patients receiving radiotherapy and other factors contributing to radiation oncogenesis needs to be considered as well.

CONCLUSION

Occurrence of gliomas following radiotherapy is a distinct complication to be considered while counseling patients with craniopharyngiomas. Although the exact mechanism of glioma formation is unclear the onset of gliomas is a poor prognostic indicator as most of these gliomas have high malignant potential. Although there are no case reports of glioma formation following stereotactic radiotherapy their follow-up is not sufficiently long.

REFERENCES


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