

Secondary anaplastic oligodendroglioma after cranial irradiation: a case report

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Abstract Secondary brain tumors rarely arise after cranial irradiation; among them, meningiomas and glioblastomas are the most common and secondary oligodendroglial tumors the most rare. We present a 48-year-old man who developed an oligodendroglial tumor 38 years after receiving 50 Gy of cranial irradiation to a pineal tumor. He underwent gross total removal of a calcified, ring-enhanced mass in the right temporal lobe. The tumor was histologically diagnosed as anaplastic oligodendroglioma. Our review of previously reported secondary oligodendroglial tumors that developed after cranial irradiation revealed that these rare tumors arose after low-dose cranial irradiation or at the margin of a field irradiated with a high dose. We suggest that secondary oligodendroglial tumors arising after cranial irradiation are more aggressive than primary oligodendrogliomas.

Keywords Secondary brain tumors · Oligodendroglial tumors · Irradiation · Anaplastic oligodendroglioma

Background

The approximate cumulative risk for secondary brain tumors after cranial irradiation is 1–3% [1–4]. Radiation-

induced secondary oligodendroglial tumors are very rare; to our knowledge, only seven cases have been reported to date [5–11]. We encountered a patient who developed a secondary anaplastic oligodendroglial tumor after radiotherapy (RT) and discuss the development of secondary oligodendroglial tumors after cranial irradiation.

Case report

This 48-year-old man was admitted to Hiroshima University Hospital in March 2005 with progressive vomiting, hiccups, and left-sided hemianopsia. In 1967, at the age of 10, he had undergone irradiation with Co-60 at Hiroshima University Hospital to treat a pineal tumor. The radiotherapeutic regimen delivered 2 Gy (200 rad) per day using alternate bilateral side ports every other day; the total dose was 50 Gy (5,000 rad). The size of the irradiation field was 6 × 6 cm at the isocenter cross-section. The tumor had completely disappeared, and no further events developed until 2005.

Magnetic resonance imaging (MRI) revealed a new mass lesion in the right temporal lobe (Fig. 1a). It was ring-enhanced by gadolinium; perifocal edema and a mid-line shift were noted. A computerized tomography (CT) image showed calcification inside the lesion (Fig. 1b). We performed craniotomy and removed the mass totally. Histologically, most of the tumor cells were round and uniform with prominent perinuclear halos and a high nuclear:cytoplasmic ratio; there were mitotic activity and microvascular proliferation (Fig. 2a, b). On microsatellite analysis chromosomes 1p and 19q were intact. Immunohistological examination (Table 1) revealed positivity for S-100, olig-2, glial fibrillary acidic protein (GFAP) (Fig. 2c), phosphatase and tensin homolog (PTEN), and

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