INTRODUCTION

Management of pediatric craniopharyngiomas has become increasingly conservative in recent years in recognition of the unfavorable outcome after extensive surgery\(^1\). Adjuvant radiotherapy (RT) after subtotal resection has become the mainstay of management. Modern RT continues to have significant adverse effects especially in the pediatric population of craniopharyngiomas, where it is mainly palliative for this benign lesion in a population with an otherwise long life expectancy\(^2\). Radiation induced tumors (RIT) are rare and high grade gliomas are even more uncommon\(^3\). In children who undergo RT for craniopharyngiomas secondary gliomas have been reported although extremely rarely\(^4\). We report a child who was diagnosed as a craniopharyngioma at the age of 2 years and later underwent radiotherapy. She presented with a glioblastoma multiforme (GBM) 10 years later.

CASE REPORT

A 12-year-old child was brought to our casualty services with features of raised intracranial pressure and left hemiparesis of grade 4. Magnetic resonance imaging (MRI) of the brain revealed an enhancing lesion in the right temporal region with central necrosis and post-contrast enhancement. The lesion measured 6.9×5.0×5.7 cm with mass effect and midline shift (Fig. 1E, F). There was a small calcified remnant in the sellar region. A working diagnosis of a high grade glioma was made and the child underwent an emergency craniotomy and decompression of the lesion. The post-operative period was unremarkable. She had previously undergone a ventriculo-peritoneal shunt and decompression of a recurrent suprasellar craniopharyngioma at our institute 4 years prior (Fig. 1C, D). She had an earlier diagnosis of suprasellar craniopharyngioma and had been operated at another institute 10 years prior to presentation. She had undergone a second surgery, within two years of diagnosis, for a residual/recurrent lesion. Fig. 1A and B demonstrate the lesion prior to the second surgery. She was then subjected to adjuvant conventional fractionated RT of 55 Gy with bilateral opposed ports with an anterior field, following which she was asymptomatic for 6 years.

Post operative MRI revealed complete decompression of the lesion (Fig. 1G, H). The histopathology revealed a GBM (Fig. 2). She was treated with temazolamide chemotherapy by an on-
cologist and succumbed to the tumor a year after surgery.

**DISCUSSION**

Cahan et al. described four criteria for the diagnosis of RIT: 1) the tumor must originate within the field of previous irradiation; 2) the latency between the RT and RIT should be sufficiently long; 3) the histological characteristics must differ from the primary lesion which was irradiated; and 4) the patient must not harbor any pathological conditions favoring the development of tumors such as neurofibromatosis. The case in discussion did conform to these criteria.

The commonest RIT tumors are sarcomas and meningiomas but gliomas have been rarely reported. Fifteen cases have been earlier reported in literature. The average age of patient at the time of irradiation was 12.5 years. The present patient was much younger at the time of RT. An average of 55 Gy has been recommended which was the same dose received by this child.

Temporal lobe is the commonest area to be localized by the secondary glioma as it does come in the field of irradiation. Of these 15 patients only 6 were GBMs and only 4 occurred in children. This patient is the 5th case of RT induced GBM for a craniopharyngioma.

No correlation is documented between the dosage of RT and the latency or grade of the secondary tumors. The histology has varied from low grade gliomas (usually adult patients) to GBM. The pediatric population appears more prone for the higher grade of RT induced malignancies. There have been no demonstrable differences in the histopathology or molecular markers between RT induced GBM and the spontaneous types. High grade gliomas, post RT, occur more often in the pediatric population as compared to adults. The latency period for the secondary pediatric GBMs is about 9 years. Our patient presented after 10 years.

Although ionizing radiation inducing gliomas in primates has been proven conclusively, such an association appears cir-
CONCLUSION

This case does stir up the debate whether adjuvant RT is warranted in benign lesions such as craniopharyngiomas, which although does appear to arrest the disease in many cases, but may be associated with the deadly complication of radiation induced malignancy. Newer modalities such as SRS or SRT may prove to be a better alternative to conventional RT.

References

1. Enchev Y, Ferdinandov D, Kounin G, Encheva E, Bussarsky V: Radiation-induced gliomas following radiotherapy for craniopharyngiomas: a case report and review of the literature. Clin Neurol Neurosurg 111: 591-596, 2009
2. Gleeson H, Amin R, Maghnie M: ‘Do no harm’: management of craniopharyngioma. Eur J Endocrinol 159 Suppl 1: S95-S99, 2008
3. Kalapurakal JA: Radiation therapy in the management of pediatric craniopharyngiomas— a review. Childs Nerv Syst 21: 808-816, 2005
4. Kawamata T, Amano K, Aihara Y, Kubo O, Hori T: Optimal treatment strategy for craniopharyngiomas based on long-term functional outcomes of recent and past treatment modalities. Neurosurg Rev 33: 71-81, 2010
5. Kitanaka C, Shitara N, Nakagomi T, Nakamura H, Genka S, Nakagawa K, et al.: Postradiation astrocytoma. Report of two cases. J Neurosurg 70: 469-474, 1989
6. Monje ML, Ramakrishna NR, Young G, Drappatz J, Doherty LM, Wen PY, et al.: Durable response of a radiation-induced, high-grade cerebellar glioma to temozolomide. J Neurooncol 84: 179-183, 2007
7. Niranjana A, Kano H, Mathieu D, Kondziolka D, Flickinger JC, Lunsford LD: Radiosurgery for craniopharyngioma. Int J Radiat Oncol Biol Phys 78: 64-71, 2010
8. Salvati M, D’Elia A, Melone GA, Brogna C, Frati A, Raco A, et al.: Radio-induced gliomas: 20-year experience and critical review of the pathology. J Neurooncol 89: 169-177, 2008