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Case report

Treatment of a glioblastoma multiforme dural metastasis with stereotactic radiosurgery: A case report and select review of the literature

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ABSTRACT

Glioblastoma multiforme (GBM) is a primary brain neoplasm accounting for approximately 75% of all high grade gliomas. It is diffusely infiltrative and exhibits rapid proliferation with a poor overall prognosis. Maximum surgical resection and postoperative radiotherapy, accompanied by concurrent and adjuvant temozolomide chemotherapy, remain the standard of care without major therapeutic advances over the past 10 years. Herein, we present the case of a 64-year-old Caucasian male with a GBM who subsequently developed a left frontal dural metastasis, subsequently treated with stereotactic radiosurgery (20 Gy in 1 fraction). With six month follow-up, the patient showed near complete resolution of his dural metastases and no overall change in neurological symptoms or side effects following radiosurgery. Due to the paucity of clinical literature regarding dural metastases from GBM, its optimal treatment remains unknown. While the role of SRS has yet to be defined in this setting, here we provide evidence suggesting its overall efficacy in the treatment of select dural GBM metastases.

1. Introduction

Glioblastomas (GBM) compose approximately 15% of all primary brain and CNS tumors, with one of the highest incidence rates (3.20 per 100,000 population) [1,3]. Glioblastoma multiforme is classified as a grade IV tumor by the World Health Organization (WHO), illustrating its severity and significance [4]. Such cases of GBM are typically diffusely infiltrative, showing rapid proliferation with a poor overall prognosis [6]. Nuclear atypia, mitotic activity, vascular proliferation, and necrosis are histologically characteristic of a GBM diagnosis, while vasogenic edema and ring enhancement around central necrotic regions are radiographic characteristics of GBM magnetic resonance imaging (MRI) [4,14].

Over the last few decades molecular biomarkers have become increasingly important in the understanding of glioblastoma and its clinical course. O6-methylguanine-DNA methyltransferase (MGMT) gene silencing by promoter methylation has been shown to increase overall survival, and the DNA alkylating agent temozolomide given both concomitantly with chemoradiotherapy and adjuvantly has largely been adopted as the standard of care [17,21]. MGMT promoter methylation status is now utilized as a predictive biomarker for newly diagnosed GBM patients [17,22]. Similarly, Isocitrate dehydrogenase-1 (IDH-1) mutation has also been recognized as a positive prognostic marker, as it is associated with a significant increase in overall survival [18]. The WHO has now categorized glioblastomas into three different subtypes based on these genetic mutations: (1) glioblastoma, IDH-wildtype (about 90% of cases), which most frequently corresponds to the clinically defined primary glioblastoma and preferentially arises in patients over 55 years of age; (2) glioblastoma, IDH-mutant (about 10% of cases), which corresponds closely to a secondary glioblastoma and predominates in younger patients; (3) glioblastoma, NOS, a diagnosis reserved for tumors for which full IDH evaluation cannot be performed [16].

The median survival for patients diagnosed with GBM is approximately 12–15 months; however, this has been shown to be higher (24–30 months) in select patients with IDH-1 mutations

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and MGMT methylation. The prognosis remains poor for those with limited performance status and increasing age, with some studies showing a median survival of 4–6 months in patients older than 65 years [14,15]. About half of all GBM patients are 65 years or older at the time of diagnosis, making the consideration of age an important prognostic factor [15,19]. The current standard of care includes maximal safe surgical resection followed by postoperative standard fractionation radiotherapy (SRT) (60 Gy in 30 fractions), as it has demonstrated increased survival benefit with no detriment to quality of life, and concurrent and adjuvant temozolomide chemotherapy [14,15]. Recent studies have found that the addition of temozolomide chemotherapy during a hypofractionated course of radiation (HRT) (40 Gy in 15 fractions) therapy improved the survival of elderly glioblastoma patients, reducing the risk of death by 33% [19]. Previous retrospective investigation of the effects of HRT versus SRT with or without temozolomide in the elderly GBM patient population also concluded that no significant survival difference existed between patients receiving HRT with temozolomide and those receiving SRT with temozolomide [20].

Despite the severity and frequency of glioblastoma diagnoses, only a limited number of case studies have described the ability of GBM to spread intradurally, or even metastasize extracranially [5,6,8]. Due to the paucity of accessible clinical literature regarding dural metastases from GBM, its optimal treatment remains relatively unknown. In this report, we provide evidence suggesting the effective use of SRS in the treatment of GBM dural metastases by discussing the case of a 64-year-old male initially treated for a GBM of the left temporal lobe, who subsequently developed an elsewhere dural-based metastasis.

2. Case report

A 63-year-old Caucasian male began suffering from short-term memory loss and expressive aphasia four months prior to his diagnosis. He ultimately presented to the emergency department with worsening symptoms, including facial droop, incomprehensible speech, and an altered mental status. A noncontrast computed tomographic (CT) study of the head showed a 3 × 3 cm mass with surrounding vasogenic edema. Subsequent magnetic resonance imaging (MRI) of the brain revealed a 4.5 × 3.7 cm peripheral enhancing lesion with a central fluid collection near the pole of the left temporal lobe, a left to right midline shift of 6 mm, moderate mass effect involving the brain stem, and moderate vasogenic edema.

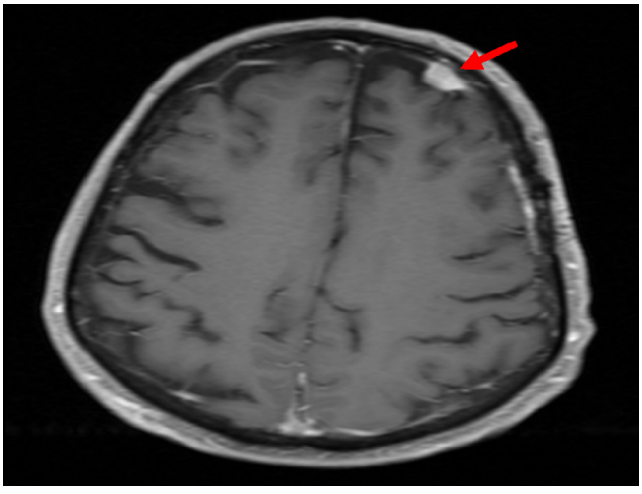

Fig. 1. Axial T1 post-contrast image pre-treatment shows a 12 mm T1 hyper intense, enhancing lesion, compatible with a left frontal dural metastatic lesion (arrow).

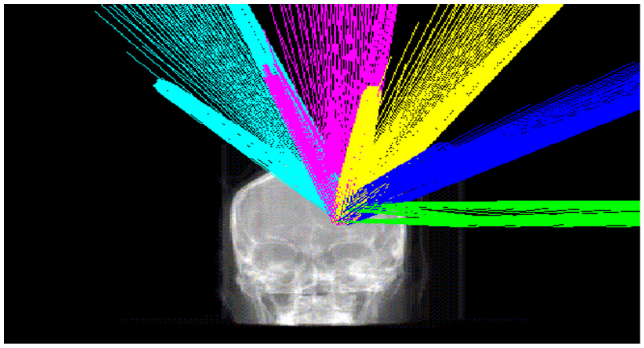

Fig. 2. Digitally reconstructed radiograph showing the configuration of multiple non-coplanar beams utilized for treatment with volumetric-modulated arc therapy.

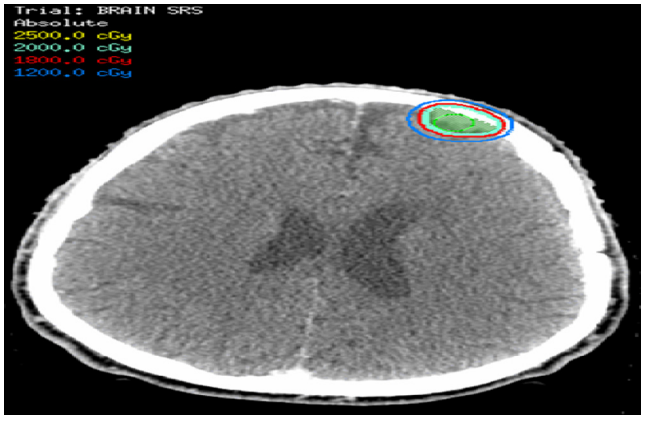

Fig. 3. Axial CT simulation image showing the isodose lines corresponding to the treatment dose of 2000 cGy prescribed to the 88% isodose.

The patient underwent a left sided craniotomy and subtotal resection. Histopathology returned a WHO grade IV GBM, non-mutated isocitrate dehydrogenase 1 (IDH1) and p53 negative. Postoperatively, he experienced a severe headache for the first two days, but described no further pain with its resolution. His mental status reportedly returned to its baseline and he denied short-term memory loss, confusion, numbness, tingling, or changes in motor function.

After presentation to our facility and a discussion of further treatment options, he was treated with standard adjuvant therapy including intensity modulated radiation therapy (IMRT) to the left temporal lobe to a dose of 60 Gy in 30 fractions with concurrent/adjuvant temozolomide chemotherapy.

One year following the completion of his concurrent chemoradiotherapy, a follow up MRI showed a new 12 mm enhancing, extra-axial mass in the dura overlying the ipsilateral frontal lobe (See Fig. 1). Progressive enhancement and fluid attenuated inversion recovery (FLAIR) signal intensity involving the anterior left temporal lobe amygdala were also concerning for recurrent tumor. Based on this diagnostic imaging and clinical characteristics, it was consistent with a dural-based GBM metastasis. At that time the primary treatment site showed increased FLAIR signal abnormality and crescentic enhancement along the medial margin of the surgical cavity, which corresponded to an area of increased blood volume on perfusion imaging. These findings were concerning for recurrent tumor superimposed on posttreatment change.

After a multidisciplinary discussion, the left frontal dural metastasis was treated with linear accelerator (LINAC) based

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