LETTERS TO THE EDITOR

Clinical-scientific notes

A case of subgaleal metastasis from glioblastoma multiforme

Extracranial metastasis in glioblastoma multiforme (GBM) is rare; however, there are even fewer reports of subgaleal metastasis, in particular, in the case of no prior surgical intervention, as tumour seeding is thought to have been the main mechanism of metastasis.1

GBM is a highly malignant primary brain tumour. The prognosis is usually poor, with a reported median survival of 14.6 months with standard treatment.2 Extracranial metastases from GBM are considered rare, with the most common sites being the lungs, pleura, liver, lymph nodes and bone marrow.3 There have been less than 10 cases reported of subcutaneous metastasis from GBM.1 We present a case of subgaleal metastasis from GBM that is contralateral to the site of previous surgical resection.

A 62-year-old normally well retired engineer was diagnosed with a left frontal lobe lesion (measuring 65 × 55 × 35 mm on MRI) following 2 weeks history of headaches, increasing confusion and short-term memory loss. Left frontal craniotomy was performed, histology confirmed GBM, negative for isocitrate dehydrogenase-1. Post-operative magnetic resonance imaging (MRI) showed no evidence of residual tumour. Subsequently, he commenced concurrent chemoradiation treatment, but withdrew from treatment after only three fractions due to personal preference. At this point, he remained fit, playing squash and walking regularly.

Eight months after the original diagnosis, he developed recurrent disease at the surgical bed. This was further resected and histology confirmed recurrent GBM. He recommenced adjuvant chemoradiation and completed 58 Gy in 29 fractions radiotherapy with concurrent temozolomide at 75 mg/m². One month later, he developed a soft subcutaneous lump on the contralateral side to the craniotomy, at the edge of the previous radiation field. MRI showed a new right frontal scalp subgaleal lesion measuring 20 × 20 × 15 mm in addition to progressing intra cranial disease. The scalp lesion was resected and histology reported a tumour composed of diffuse proliferation of pleomorphic astrocytes, with palisade necrosis and exuberant glomeruloid microvascular proliferation. The periphery of the specimen showed some lymphatic permeation. The final diagnosis was consistent with metastatic GBM. The surgical team confirmed that there was no instrumentation during both operations that could have caused seeding to the contralateral scalp. The patient further declined any subsequent treatment and was admitted to the hospice for the end of life care. He died 2 months later, 14 months since original diagnosis. His scalp lesion continued to grow in the hospice (Fig. 1).

Figure 1 Clinical and magnetic resonance imaging (MRI) images of the subgaleal lesion contralateral to the side of previous tumour, resection and chemoradiation site.
Previously reported cases of extracranial glioblastoma have been associated with prior interventions, suggesting direct physical disruption of the blood–brain barrier as a possible mechanism of metastasis. A case report of recurrent scalp metastasis from GBM was localised to the surgical incision site and this suggested direct tumour seeding was a possible mechanism. A study in mice tested this hypothesis by systemically implanting delayed brain tumour (DBT) cells (similar central nervous system tumours to human GBM cells). These implantations survived extracranially, suggesting that the rarity of extracranial metastasis may be due to physical barriers such as the blood–brain barrier.

In this case, the subgaleal metastasis developed on the contralateral side of previous intervention and therefore provides a unique discussion. This raises a possibility of potential local vascular invasion made possible after concurrent chemoradiation. Further mechanisms of metastasis in GBM should be investigated, as this case highlights a different mode of metastasis other than local physical disruption of the blood–brain barrier.

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