Transdural Spread of Glioblastoma through the Foramen Ovale with Presentation as a Masticator Space Mass

SUMMARY: Direct extension of a glioblastoma through the dura at the skull base is an uncommon occurrence. We report an unusual case of recurrent glioblastoma that spread transdurally through the foramen ovale and presented primarily as a masticator space mass.

Case Report

A 53-year-old right-handed man without significant medical history initially presented in October of 2006 with headaches, right-sided hemiparesis, and episodes of odd episodic smells. He was found to have a left temporal lobe mass, which was resected via a left pterional craniotomy and was found to be a grade IV astrocytoma with infiltrative activity as well as areas of palisading necrosis. Cells stained strongly positive for glial fibrillary acidic protein, vimentin, and S100 and were negative for epithelial membrane antigen, synaptophysin, cytokeratins (AE1/AE3), and neurofilament. A diagnosis of recurrent GBM was made.

Discussion

There are many barriers to the spread of disease from the intracranial compartment. These include the dural coverings, the basement membranes around vessels of the central nervous system, the absence of true lymphatics in the intracranial compartment, and the early occlusion of compliant veins by tumor. For hematogenous dissemination to occur, the tumor cells must gain access to the intravascular space. Although there are data to suggest that glial tumors can perform the steps necessary to do this primarily, historically, it has been thought that extracranial dissemination of GBM occurred solely in the setting of a prior craniotomy, biopsy, or shunt placement. Such a procedure would, theoretically, provide tumor cells a mechanical entry point into the vascular or extracranial lymphatic system (or a direct route into the peritoneum in the setting of a ventriculoperitoneal shunt). Many reports and exhaustive reviews have described remote extracranial metastases in the setting of a prior surgical intervention.

There has been a smaller number of case reports documenting remote extracranial metastases in patients who have never had any prior violation of the dura other than radiation therapy. Local invasion of the dura and, subsequently, the dural venous sinuses, is thought to play a role in many of these cases. In fact, at postmortem, many of these patients are found to have tumor that has entered the venous sinuses.

Transdural spread of GBM in patients without prior dural defects or at sites distant from a craniotomy defect is a rare occurrence with few reported cases. In their review, Kawano et al described 3 routes for extradural extension of gliomas: 1) through the perivascular or dural slit at the middle cranial fossa, which is facilitated by intracranial hypertension; 2) along the cranial or spinal nerves; and 3) via the direct destruction of the dura mater. Although the most common sites for extradural spread with direct involvement of bone are the cerebral convexities and the middle cranial fossae, these occurrences are exceedingly rare.

We were able to find 12 reported cases of glioblastoma spreading spontaneously through the skull base. They are summarized in the Table. Our case is unique in that there was minimal intracranial/intradural disease and isolated extension through the native foramen ovale. This resulted in a large mas-


**Spontaneous extradural spread of glioblastoma through the skull base**

| Investigator          | Age (yr), Sex | Site of Primary                  | Site of Extradural Spread                  | Prior Treatment                      |
|-----------------------|---------------|----------------------------------|-------------------------------------------|--------------------------------------|
| Sanerkin, 1962        | 65, M         | L temporal lobe                  | L middle cranial fossa                    | None                                 |
| Nager, 1967           | 41, M         | Large R temporal                 | R temporal bone, presenting as a mass in the R external auditory canal | No prior craniotomy or radiotherapy   |
| Hoyt et al, 1972       |               | Fronto-temporal                  | Anterior fossa                            | Radiation and surgery approx 8 months prior |
| Livnicz and Rubinstein, 1979 | 26, M     | Large L temporal lobe            | L middle cranial fossa                     |                                       |
| Anyama et al, 1980     | 30, F         | R frontal                        | Anterior cranial fossa into nasal cavity  | None                                 |
| Shuangshiti et al, 1987 | 30, M      | R frontotemporal                 | R sphenoid wing, posterior wall of frontal sinus | None                                 |
| Shuangshiti et al, 1987 | 25, F      | R frontotemporal                 | Sphenoid wing, ethmoid, posterior orbit   | None                                 |
| Bigner et al, 1989     | 7, F          | L medial temporal                | L sphenoid wing and sella turcica into nasopharynx | Left ventriculopleural shunt         |
| Lampi et al, 1990      | 32, M         | R frontoparietal                 | Ethmoid sinus, frontal sinus              | Resection 8 months prior with chemoradiation |
| Pompili et al, 1993    | 40, M         | Bifrontal                        | L orbit, ethmoid, maxillary sinus, and nasal fossa | Partial resection and radiation      |
| Horiuchi et al, 1996   | 41, F         | R temporal lobe                  | R anterior and middle cranial fossae into orbit, nasal cavity, and oral cavity | 2 prior resections                   |
| Rainov et al, 1996     | 68, F         | L temporal lobe                  | Anterior and middle cranial fossae         | None                                 |

Note:—R, L indicate right and left, respectively; approx, approximately.

Fig 1. A 53-year-old man with history of glioblastoma presented with chronic otomastoiditis and mandibular nerve denervation. A, Coronal T2-weighted MR image demonstrates a transcranial mass extending through the left foramen ovale, with the largest component in the masticator space (white arrowheads). Subacute denervation of the left medial pterygoid and masseter muscles is evidenced by their increased signal intensity and decreased volume (white arrow). Chronic postoperative and treatment-related changes are noted in the left middle cranial fossa and left temporal lobe (black arrow). B, Axial contrast-enhanced fat-suppressed T1-weighted MR image demonstrates the extracranial portion of the mass interposed between the left medial and lateral pterygoid muscles (white arrowheads). The left temporalis and masseter muscles demonstrate asymmetrically decreased volume (white arrow). Note the mass effect on the left eustachian tube orifice and abnormal signal within the left mastoid air cells, likely on an obstructive basis (black arrow).

Fig 2. PET images in the coronal plane clearly show transcranial tumor extending from the floor of the left middle cranial fossa into the soft tissues of the left deep face (black arrows).

In summary, the extracranial spread of a glioblastoma, particularly through an intact dura, is a rare occurrence. We reviewed the different mechanisms by which glioblastomas are thought to spread and presented a unique case of recurrent tumor that extended through foramen ovale, resulting in a large volume of extradural tumor and secondary denervation.

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