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## Visual Loss as the Presenting Manifestation of Leptomeningeal Spread of Glioblastoma Multiforme to the Optic Chiasm

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lioblastoma multiforme (GBM) is a malignant pro-■ liferation of astrocytes with an incidence of 10,000 new cases annually in the United States. GBM makes up 80% of primary malignant central nervous system numors, and it typically presents with headaches, confusion, focal neurologic deficits, seizures, memory loss, and personality changes. Despite aggressive therapy, GBM has a poor prognosis and often recurs locally (1). Visual loss in GBM is typically due to secondary infiltration of the posterior visual pathway (e.g., homonymous hemianopsia) or less commonly primary involvement of the anterior visual pathway (e.g., malignant optic glioma). Leptomeningeal spread (LMS) of GBM however is unusual. We report a case of progressive vision loss as the presenting manifestation of recurrent GBM with secondary LMS to the optic chiasm. After review of the PubMed and Google Scholar databases, we were unable to identify any such previously reported cases.

A 37-year-old woman initially presented with headache, nausea, and vomiting in January 2021. Computed tomography and MRI of the brain revealed a tumor involving the frontal lobe. A subtotal resection was performed on March 24, 2021, and histopathology confirmed GBM. Her

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The authors report no conflicts of interest.

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Address correspondence to Andrew G. Lee, MD, Herb and Jean Lyman Centennial Chair in Ophthalmology, Department of Ophthalmology, Blanton Eye Institute, Houston Methodist Hospital, 6560 Fannin Suite, Ste 450, Houston, TX 77030; E-mail: aglee@ houstonmethodist.org medical history was significant for clavicular Hodgkin lymphoma treated with radiation and chemotherapy and in remission. The remainder of the medical, social, surgical, and family histories were noncontributory.

One month after surgery, however, the patient developed subacute, progressive visual loss in both eyes despite postoperative chemotherapy with temozolomide and external beam radiation therapy. On neuro-ophthalmologic examination, the visual acuity was counting fingers in the right eye and no light perception in the left eye. There was a left relative afferent pupillary defect. Intraocular pressure measurements, slit-lamp biomicroscopy, motility, and external examinations were normal in both eyes. Dilated fundus examination showed diffuse optic atrophy in both eyes.

On July 14, 2021, a repeat MRI of the brain showed an enhancing mass involving the frontal lobes and corpus callosum with enhancement of the right parietal temporal junction and nonenhancing tumor at the medial left parietal lobe and new enhancement and edema in the optic chiasm (Fig. 1). MRI of the cervical/thoracic/lumbar spine with and without contrast showed faint enhancement of nerve roots of the cauda equina. A lumbar puncture showed cerebrospinal fluid white blood cell count of 3/mm<sup>3</sup>, red blood cell count of 29/mm<sup>3</sup>, glucose of 97 mg/dL, and protein of 70 mg/dL. Gram stain, culture, and cryptococcal antigen were negative. Cytology showed no malignant cells and only few lymphocytes and blood. A diagnosis of LMS of GBM was made. Ultimately, the patient was lost to follow-up.

GBM is the most common primary malignant brain tumor in adults and can produce visual loss directly from involvement of the anterior or posterior visual pathway or indirectly from papilledema and increased intracranial pressure. This patient presented initially with a primary frontal lobe GBM but then developed progressive visual loss from chiasmal involvement of GBM because of LMS of tumor. GBM can arise from the optic apparatus (e.g., malignant glioma) but typically spreads through direct extension to adjacent structures. The initial neuroimaging of this case showed frontal GBM without visual loss or radiographic involvement of the optic chiasm.