LEFT PLANUM SPHENOIDALE MENINGIOMA WITHOUT OPTICAL CANAL INVOLVEMENT CAUSING OPTIC NEUROPATHY DUE TO HYPEROSTOSIS



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Department of Neurosurgery, Mayo Clinic, Rochester MN;² Rhoton Neurosurgery and Otolaryngology Surgical Anatomy Program;³ Universidad Europea de Madrid. Faculty of Biomedical and Health Sciences. Department of Medicine, Madrid Spain.;⁴ Department of Otolaryngology Head and Neck Surgery, Mayo Clinic, Rochester MN;

BACKGROUND

Meningiomas are known to produce hyperostosis through the overexpression of osteogenic molecules (1). Sphenoid wing meningiomas often lead to hyperostosis and orbital-periorbital invasion, necessitating the removal of the periorbita and bone to decompress the orbit (2,3). Other meningiomas, such as anterior clinoid meningiomas, can also cause hyperostosis and lead to optic neuropathy (4). We present the rare case of a patient with a left planum sphenoidale meningioma without optic nerve compression but causing optic neuropathy due to hyperostosis.

CASE DESCRIPTION



64-year-old left-handed male with a notable family history of tumors linked to a known SDH mutation. The patient reported neck tightness and ear pain, prompting an MRI that revealed a left planum sphenoidale meningioma over the left optic canal, causing frontal lobe edema (Image 1), without optic nerve compression or canal invasion but with optic canal hyperostosis (Image 2). Retrospectively noted vision difficulties in his left eye starting in early 2024, confirmed by a superior altitudinal defect on visual field testing (Image 3) and ganglion cell layer thinning on OCT (Image 4). His visual acuity was 20/15 in both eyes

PREOPERATIVE WORK UP

Indication for surgery was the optic neuropathy. Considering his genetic predisposition, serum metanephrine screening, DOTATATE PET scan, and 24-hour urine catecholamine collection were ordered preoperatively. Comprehensive counseling on the surgical approach, including a left frontotemporal craniotomy, orbital optic osteotomy, anterior clinoidectomy for full optic canal decompression, and tumor removal, was provided. Risks such as potential visual loss, infection, stroke, and CSF leak were discussed. Frontal lobe edema and poor CSF plane anticipated a difficult dissection plane.

SURGICAL PROCEDURE



Patient was positioned supine with the head turned towards the right, a standard pterional approach with interfascial dissection, extradural anterior clinoidectomy and optic nerve unroofing was performed. Intraoperative findings confirmed the absence of optic nerve compression, and the presence of bone hyperostosis over the optic canal and the tumor's poor plane with the inferior frontal lobe. The tumor, the involved bone and duramater were removed successfully, achieving a Simpson grade 1 resection. Duramater was reconstructed using a vascularized pericranial graft.

POSTOPERATIVE COURSE

The patient's recovered well postoperative with intact frontalis branch function as well as stable vision. No new other neurological symptoms. On the 2-week follow-up reported improved vision and mild temporal muscle swelling. The patient denied any neurological concerns, such as diplopia or seizures.

PATHOLOGY AND FOLLOW UP

Final pathology indicated a grade II meningioma due to brain invasion, with no TERT mutation and a normal chromosomal microarray pattern. Given the gross total resection and the tumor's relative benign nature, a watchful waiting approach with an MRI in three months was recommended. The patient and his family were thoroughly informed.





Meningiomas can induce bone hyperostosis, which can lead to optic neuropathy even without direct optic

nerve compression. This case highlights the rare presentation of a planum sphenoidale meningioma

causing optic neuropathy solely through hyperostosis. The importance of removing the tumor, duramater,

and surrounding hyperostotic bone to improve functional outcomes is underscored.

References

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