



Dramatic Hemorrhagic Papilledema After Surgical Resection of Meningioma

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Introduction

Papilledema refers to bilateral optic disc swelling secondary to raised intracranial pressure (ICP). It is widely acknowledged that all patients with papilledema should undergo prompt evaluation for an underlying etiology. However, whether such patients would still require ophthalmological follow-up post-resection of intracranial tumors is still debated. Here, we present the case of a patient who benefited from routine follow-up for papilledema.

Case Description

An asymptomatic 45-year-old female presented with incidental bilateral optic disc swelling first noted on routine diabetic retinal photography screening. Her best corrected visual acuity (BCVA) was 20/25 OU, while color vision and pupillary reflexes were intact. Magnetic resonance imaging (MRI) of the brain reported a large tentorial World Health Organization grade 2 meningioma centered over the pineal region with mass effect, for which she underwent a left craniotomy and subtotal excision of the tentorial meningioma. Two months post-operatively, the patient was seen during her routine visit and reported symptoms of mild non-specific blurred vision, while still denying other symptoms of raised ICP. As she felt well, she had not returned earlier. On examination, BCVA had deteriorated to 20/80 -1 and 20/60 -2 in right and left eye, respectively. Other elements of optic nerve function such as pupillary reflexes and color vision were still preserved. On fundus examination, there was severe bilateral hemorrhagic papilledema with pre-retinal hemorrhage (Fig 1). Optical coherence tomography (OCT) showed marked thickening of the peripapillary retinal nerve fiber layer (RNFL), with extension of the peripapillary subretinal fluid into the macula OU (Fig 2). Repeat MRI with contrast revealed that there was an interval increase in the size of the lateral ventricles as well as new mildly enhancing dural-

based soft tissue partially occluding the posterior third of the superior sagittal sinus, suggestive of post-surgical changes (Fig 3). A ventriculoperitoneal shunt (VPS) was inserted the next day by the neurosurgical team.

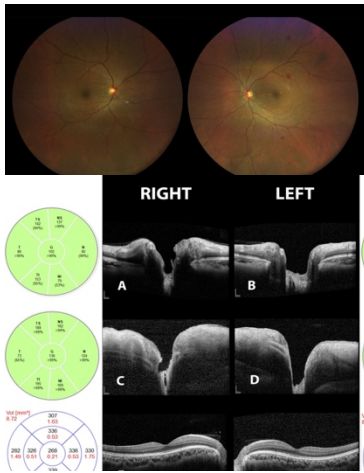


Figure 1. Top row: Fundus photographs of the right eye (left) and left eye (right) showing subtle disc swelling at the initial visit. Bottom row: Severe hemorrhagic papilledema with macular edema two months after surgery. Subhyaloid hemorrhage in the right eye and retinal hemorrhages in the left eye are seen.

Figure 2. Spectral domain OCT with RNFL and central subfield thicknesses (μm). (A and B): Index optic disc OCTs showing mild peripapillary fluid. (C and D): Marked bilateral disc swelling post-surgery. (E and F): Foveal contour was well preserved on the index visit. (G and H): Bilateral extension of the peripapillary subretinal fluid into the macula post-surgery.

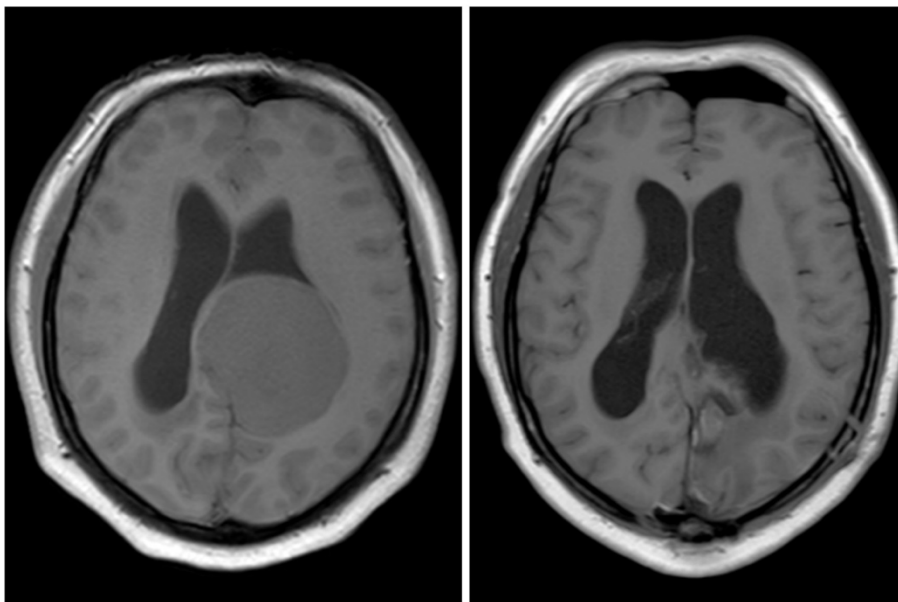


Figure 3. MRIs pre- (left) and post- (right) excision of meningioma, showing post-surgical expansion in lateral ventricle volume and hydrocephalus.

Discussion

The persistence of papilledema after resection of intracranial tumors has been previously described.¹ However, to our knowledge, worsening of papilledema post-resection of an intracranial tumor - especially to such an extent - has not been well reported. We also note that there are no standardized guidelines suggesting

ophthalmic follow-up to be routine practice for patients with papilledema secondary to an intracranial mass. It is generally taken for granted that papilledema would resolve with the normalization of ICP.

The common teaching is that papilledema subsides within 6-8 weeks after removal of an intracranial tumor.² However, there have been cases of papilledema persisting beyond 10 weeks after resection of intracranial tumors, with a maximal resolution time of up to 20 weeks.¹ Hence the clinical course of papilledema after the resection of an intracranial mass may not be so straightforward. Post-operative papilledema is postulated to be due to post-operative cerebrospinal fluid (CSF) circulation blockage caused by brain edema or in later stages, chronic reactive arachnoiditis.¹ Such obstruction of CSF circulation results in the formation of hydrocephalus post-operatively, which typically presents with signs and symptoms of raised ICP such as headaches accompanied by nausea and vomiting. In a study of patients who underwent surgery for intracranial meningiomas, 5.9% developed communicating hydrocephalus postoperatively.³ This percentage increased to 6.5% in patients who underwent surgery for intracranial glioblastoma.⁴ The number of patients at risk of pathological post-operative papilledema may be in line with these findings. It has also been suggested that periodic examination for papilledema should be performed in patients with noncompliant ventricles or extra-ventricular obstruction of CSF flow, as imaging may fail to disclose conventional signs of high ICP.⁵ To guide

the duration and interval of ophthalmological follow-up for such patients, further work could evaluate the incidence of worsening or recurring papilledema in patients post-resection of an intracranial tumor, as well as its effect on visual function.

The above case highlights how severe hemorrhagic papilledema can present with deceptively mild visual symptoms. Patients may not feel the need to return for an earlier visit despite a worsening in their intracranial condition. Hence, early scheduled ophthalmology follow-up may be helpful when monitoring patients with ICP-related conditions, especially when there is a high index of suspicion. Even in the absence of a follow-up visit with ophthalmology, safety net return advice, especially in the form of worsening vision, may prompt follow-up when needed. While BCVA is usually preserved in papilledema, worsening vision is a herald for optic neuropathy and/or maculopathy. Optic neuropathy usually occurs in eyes with chronic papilledema due to gradual loss of RNFL but may also occur acutely from ischemia and retinal nerve fiber infarcts.⁶ Maculopathy may result from retinal hemorrhage, extension of peripapillary subretinal fluid (Fig 2), and choroidal folds from optic nerve sheath distension.⁶

Conclusion

This case highlights a rarely reported example of marked worsening of papilledema after resection of an intracranial tumor. This suggests that there is utility in ophthalmology follow-up visits for such patients with papilledema even after resection of an intracranial tumor. Future work may further elucidate the rates of papilledema recurrence and persistence in patients after intracranial surgery, as well as the effects of this on visual function, to better inform ophthalmologists regarding the follow-up of such patients.

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Informed Patient Consent

The patient provided informed written consent for the material to be published and was offered the opportunity to read the manuscript and to see all photographs, but waived the right to do so.

Statement of Ethics

This case report adheres to patient confidentiality and ethical principles in accordance with the guidelines of the Declaration of Helsinki and relevant local regulations. Written consent was obtained from the patient for the publication of this case report.

Conflict of Interest Statement

The authors declare no conflict of interest related to this case report.

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