Vision salvage after resection of a giant meningioma in a patient with a loss in light perception

Case report

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Clinical approaches to the surgical management of optic chiasm compression stress quick action, as several case series have demonstrated minimal vision restoration following aggressive decompression in patients presenting more than 3 days after the onset of blindness. The authors here report the case of a 48-year-old woman who presented with near-complete binocular vision loss but regained visual function following surgical removal of a giant planum-tuberculum meningioma, which was performed 8 days after a documented loss in light perception. The interval between the patient’s vision loss and successful vision-restoring decompressive surgery is the longest recorded to date in the literature. This case shows the importance of aggressive decompression of mass lesions despite extended intervals of optic nerve dysfunction. (DOI: 10.3171/2008.7.JNS08260)

KEY WORDS • craniotomy • meningioma • optic chiasm • vision loss

Traditional teaching emphasizes the exquisite sensitivity of the optic nerve and chiasm to compressive insult, the time-sensitive nature of compressive visual loss, and the importance of rapid action in the setting of compressed optic apparatus.1–3,13 It has followed from this approach that vision loss stemming from chronic compressive lesions is irreversible and that emergency decompressive intervention is not useful.7 Here we report on a case of urgent decompression of the optic chiasm performed 8 days after documentation of complete monocular blindness, resulting in near-complete restoration to preoperative levels of vision.

Case Report

History and Examination. This otherwise healthy 48-year-old woman initially presented to an outside hospital with chronic, stable mild visual loss, dizziness, and loss of smell. A subsequent workup revealed a giant extraxial mass, with imaging features suggestive of a planum-tuberculum meningioma, arising from the anterior skull base and its posterior-most limit abutting and compressing the optic chiasm (Fig. 1). The patient was seen in the UCSF neurosurgery clinic 1 week later with plans for endovascular embolization followed by bifrontal decompressive surgery. Given that her visual loss was not acute and her acuity had not recently changed according to her primary ophthalmologist, steroids were not administered at that time, and surgery was scheduled on an elective basis for ~3 weeks later. Results of visual field examinations performed 1 week prior to her clinic visit are shown in Fig. 2 upper. Approximately 6 days later, the patient noted rapid worsening of her visual loss, which over the course of 1 day progressed to complete vision loss in the right eye and only light perception in the left eye. She did not seek medical attention for ~7 days after noting this nearly complete blindness. She was again seen at UCSF 14 days after her initial clinic visit. On arrival, she was initially treated with 30 mg of mannitol intravenously every 8 hours and 4 mg of dexamethasone by mouth every 6 hours for 12 hours prior to surgery. She was urgently taken to surgery ~8 days after her vision had reached its worst point and 9 days after the onset of rapid visual worsening. Visual field examinations had been performed at the outside hospital 7 days after she experienced blindness, and the results are seen in Fig. 2 lower. Intraocular pressures could not be measured prior to surgery.

Treatment. An extended bifrontal craniotomy was performed, the anterior ethmoid arteries were dissected and divided, and the tumor was internally debulked and removed. Particular attention was paid to the posterior-most portions of the lesion abutting the right optic nerve.
and chiasm, and the arachnoid plane was preserved with sparing of hypophyseal branches supplying the chiasm. A gross-total resection was achieved (Simpson Resection Grade II), and there were no intraoperative complications. Pathological analysis confirmed the tumor as a World Health Organization Grade I meningioma.

**Postoperative Course.** The patient experienced rapid improvement in vision over the first 48 postoperative hours. Eventually, she had nearly complete restoration of binocular vision to preoperative levels of visual acuity. At 8 months postoperatively her visual acuity was 20/450 right eye and 20/150 left eye. Visual fields at 8 postoperative months are featured in Fig. 3.

**Discussion**

We report the nearly complete return of vision following optic chiasm decompression performed 8 days after the onset of complete monocular blindness. Reports of restored vision following the removal of compressive mass lesions of the optic chiasm generally have a limited time window of intervention to the early acute phase (within 72 hours), with minimal success being achieved with later decompressions. In fact, to our knowledge, only 2 prior reports on the reversal of blindness more than 72 hours after the onset of vision loss exist in the literature—that is, 5 days after symptom onset in an adult and 7 days after symptom onset in a child—making our case the longest duration of reversible blindness reported to date.

Experimental evidence suggests that optic nerve failure in the setting of compressive insult likely results from 2 related but distinct mechanisms. The first is physiological axonal failure, which results from local axonal hypoxia, physical blockade of axonal transport, and local disruption of axonal polarization. The second is demyelination and axonal death, which both likely occur due to the localized hypoxia and cytoskeletal disruption caused by compressive lesions. The very existence of the rapid reversibility of blindness in cases of compressive lesions supports the reality of physiological failure—and not irreversible axonal/retinal nerve cell injury—as the principle cause of blindness in the early stages of most compression-induced optic chiasm injuries. The current case may be representative of a subset of patients who maintain optic nerve structural integrity and can indeed benefit from delayed therapy.

Clearly, the reversibility of a deficit relates to the duration of symptoms, and cases of reversibility following prolonged optic nerve compression are uncommon. Nonetheless, the very existence of such cases raises the question of whether the concept of delayed decompression has been prematurely abandoned based on earlier very small case series and anecdotal experiences. The optic nerve symptomatology in the patient in the present case was more con-
Successful delayed optic chiasm decompression

consistent with physiological optic nerve failure, as opposed to optic nerve demyelination and cell death. Physiological MR imaging methods such as diffusion and perfusion weighted imaging as well as diffusion tensor imaging are increasingly being adapted for use in studying the function and pathological anatomy of the optic tract and are proving to correlate well with physiological measures of optic nerve function. As this technology evolves, it will become increasingly possible for neurosurgery to shift away from the older, time-based paradigm of optic nerve decompression and move toward a more logical physiologically based paradigm in which decisions regarding the urgency of surgery are based on the proximate cause of blindness and not on an arbitrarily assigned cutoff.

Disclaimer

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

References


Fig. 2. Preoperative visual fields (upper) on initial presentation, 2 weeks prior to the onset of a loss in light perception and 1 week before being seen at the UCSF neurosurgery clinic, and (lower) 3 weeks later, 7 days after the loss in light perception. Light areas signify good vision; dark regions, loss of vision.

Fig. 3. Visual fields at 8 months postoperatively. Light areas signify good vision; dark regions, loss of vision.

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