Complete Recovery from Monocular Blindness Caused by Aneurysmal Compression to Optic Nerve

—Report of Two Cases—

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Abstract

Two patients with complete absence of light perception due to optic nerve compression were treated by decompressive procedures. Visual function progressively improved to the level of useful vision. A 42-year-old female with a partially thrombosed aneurysm in the right A1 of the anterior cerebral artery presented with right visual loss without light perception. The aneurysm was trapped and the intraluminal organized blood clot removed without dissecting the dome from the optic nerve. Her vision recovered to 20/20 1 week later. A 29-year-old male with a partially thrombosed saccular aneurysm of the anterior communicating artery presented with left monocular blindness. The aneurysm was clipped and the intraluminal clot removed without dissecting the aneurysmal wall from the optic nerve. His visual acuity gradually recovered to 10/20 12 days later. Visual recovery after decompressive procedures following prolonged absence of light perception due to optic nerve compression by aneurysms is rare. The pathophysiological mechanism of prompt visual recovery is unknown, but surgical treatment should be considered for any such patients.

Key words: cerebral aneurysm, blindness, vision, optic nerve, nerve compression, nerve decompression

Introduction

Visual disturbances are sometimes caused by mass lesions in the suprasellar or parasellar regions compressing the optic nerve. Prompt and complete decompression can achieve recovery of vision, but prolonged compression usually causes irreversible loss of vision. Also, surgical decompressive procedures are generally considered ineffective where compression of the anterior visual pathway has caused complete loss of light perception.

We present two cases of gradual monocular visual loss resulting in complete loss of light perception which persisted for 6–20 days prior to surgical decompression of a cerebral aneurysm compressing the unilateral optic nerve. In both cases, visual function progressively improved to the level of useful vision.

Case Reports

Case 1: A 42-year-old female noticed blurring of right eye vision in October, 1989. A local ophthalmologist diagnosed retrobulbar neuritis and prescribed steroid therapy for 1 month. However, she lost visual perception of light on November 18. She was referred to the Department of Neurosurgery.

Neuro-ophthalmological examination revealed complete loss of right visual function and no right pupil response to light, although Marcus-Gunn reaction remained. Fundoscopic examination demonstrated no optic atrophy. Her left visual acuity was normal, but superior lateral quadrantanopia was detected (Fig. 1 left). Precontrast computed tomographic (CT) scans demonstrated a round, homogeneous high-density mass about 1.5 cm in diameter in the right anterior suprasellar cistern, which was heterogeneously enhanced postcontrast. Magnetic resonance (MR) imaging demonstrated a supra-
sellar round mass with posterior high-intensity and anterior low-intensity regions on both T1- and T2-weighted images. The anterior region was enhanced by gadolinium-diethylenetriaminepenta-acetic acid (Gd-DTPA) (Fig. 2). Right carotid angiograms demonstrated a saccular, partially thrombosed aneurysm (11 x 7 x 10 mm) in the A1 portion of the right anterior cerebral artery (Fig. 3, left, center). Left carotid angiograms with manual compression of the right cervical carotid artery revealed adequate blood flow to the right anterior cerebral artery (A1 and A2 portions) (Fig. 3, right).

The aneurysm was trapped via a right pterional approach 20 days later. The right C1, M1, and proximal A1 were dissected. The aneurysmal dome was compressing the right optic nerve, causing thinning and fanning. The distal portion of the right A1 was dissected and the perforating arteries from the proximal A1 close to the aneurysmal dome were dissected from the neck. Application of a clip to the aneurysm was difficult due to the relatively wide neck, so Sugita 7-mm straight clips were applied to the proximal and distal A1 portions without compromising small perforators. The aneurysmal dome was then opened and the intramural organized clot subtotally removed using an ultrasonic aspirator. No effort was made to dissect the dome from the thinned right optic nerve.

Surprisingly, she regained some light perception when she awoke from anesthesia. She was able to count fingers 2 days later, and regained 20/20 vision in her right eye after 1 week (Fig. 1, right). Figure 4 shows the course of visual recovery after surgical decompression of the right optic nerve. Postoperative bilateral carotid angiograms showed successful trapping of the aneurysm and opacification of the bilateral anterior cerebral arteries via the left carotid artery (Fig. 5). Postoperative CT scans showed no abnormalities.

Case 2: A 29-year-old male suddenly developed a
severe headache on May 9, 1991. Three weeks later, he noticed progressive visual deterioration and complete loss of light perception in the left eye on June 25. CT scans indicated a mass in the suprasellar cistern. He was referred to the Department of Neurosurgery.

Neuro-ophthalmological examination revealed absence of light perception and pupillary reaction to light in the left eye. Corrected visual acuity of the right eye was 18/20 with concentric narrowing of visual field. Postcontrast CT scans demonstrated an enhanced round mass in the suprasellar cistern (Fig. 6 left). MR imaging demonstrated a round mass with heterogeneous intensity on both T1- and T2-weighted images. Right carotid angiograms showed a large aneurysm (15 × 12 × 20 mm) in the anterior communicating artery at the right A1 and A2 junction (Fig. 6 center, right).

A right frontotemporal craniotomy was performed 6 days after total loss of visual function. The neck of the aneurysm was successfully clipped and the intramural clot removed to decompress the optic nerve. No attempt was made to dissect the aneurysmal wall from the optic nerves and chiasm.

He regained left pupil reaction to light immediately after the operation. Left visual acuity recovered to 10/20 and right to 30/20 by the 12th postoperative day. Neither visual field had any defect. Postoperative right carotid angiograms revealed the aneurysmal dome to be obliterated without compromising the main arteries.

**Discussion**

In 1915, Cushing and Walker analyzed 81 cases of chiasmal lesion, finding that immediate visual restoration following surgical decompression in
some cases of compressive visual failure suggested the presence of some "physiological block" which was relieved. Pennybacker also described the dramatic visual function response to surgical treatment in patients with pituitary adenoma and the possibility of a full recovery. Golnik and Miller studied postoperative visual recovery in patients with optic nerve and parasellar meningiomas and concluded that visual improvement after surgery depends on several factors including tumor size, preoperative duration of visual symptoms, and extent of preoperative visual loss. Early decompression of the optic nerve or chiasm resulted in significant return of visual function in many patients.

Waybright et al. reported a series of patients with visual loss due to tumors compressing the optic nerve persisting from 3 months to 15 years. Four patients with total loss of light perception preoperatively remained blind. However, two patients have achieved visual recovery following prolonged absence of light perception due to aneurysms compressing the optic nerve (Table 1). Durston and Parsons-Smith described a 54-year-old female with a giant aneurysm of the anterior communicating artery, who had suffered blurred vision in the left eye for 3 weeks. The right common carotid artery was ligated to treat the aneurysm. However, vision continued to deteriorate after the operation, and after 3 weeks there was no light perception in her left eye. Atrophic change of the right optic disc was unchanged and pupillary reaction remained sluggish. The right eye showed worsening visual field. Surprisingly, 3 months after the operation, her vision suddenly began to improve, and she could count fingers 12 inches in front of her 9 months later. The left eye developed a full visual field but nasal hemianopia in the right eye remained. Striph et al. reported a 25-year-old male with a giant aneurysm of the anterior communicating artery which caused steadily deteriorating vision in the right eye over 3 weeks, and finally complete loss of light perception. Visual evoked response testing revealed no waveform on the right. The aneurysm compressing the right optic nerve was clipped. Eleven days later, diplopia suddenly developed, and the right visual acuity had improved to 20/100. Two and a half years later, visual acuity in the left eye was 20/30.

Compressive optic neuropathy may be caused by 1) ischemia secondary to vascular compression, 2) direct damage to nerve fibers or glia cells as oligoden-

Table 1 Reported cases of recovery from prolonged absence of light perception after aneurysm surgery

<table>
<thead>
<tr>
<th>Author (Year)</th>
<th>Age, Sex</th>
<th>Location of aneurysm</th>
<th>Duration of complete blindness</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Durston and Parsons-Smith (1970)</td>
<td>54, F</td>
<td>AcomA</td>
<td>3 wks postoperatively</td>
<td>cervical carotid ligation clipping</td>
<td>began to improve 3 mos postoperatively 20/100, 11 days postoperatively</td>
</tr>
<tr>
<td>Striph et al. (1984)</td>
<td>25, M</td>
<td>AcomA</td>
<td>12 days</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Present Case 1</td>
<td>42, F</td>
<td>A1</td>
<td>20 days preoperatively</td>
<td>trapping, removal of clot</td>
<td>20/20, 1 wk postoperatively</td>
</tr>
<tr>
<td>Present Case 2</td>
<td>29, M</td>
<td>AcomA</td>
<td>6 days preoperatively</td>
<td>clipping, removal of clot</td>
<td>10/20, 12 days postoperatively</td>
</tr>
</tbody>
</table>

AcomA: anterior communicating artery.

Fig. 6 Case 2. left: Preoperative postcontrast CT scan, showing a round mass in the suprasellar cistern. center, right: Preoperative anteroposterior (center) and lateral (right) right carotid angiograms, demonstrating a large aneurysm of the anterior communicating artery.
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drocytes, or 3) interruption of axoplasmic flow. Clifford-Jones et al. reported an experimental cat model of progressive chronic optic nerve compression induced by implanting an inflatable balloon near the optic nerve. A slow-growing mass was simulated by increasing the intraluminal pressure of the balloon at weekly intervals, with the contralateral optic nerve as control. Histological examination showed that extensive demyelination took place within the first week. Surprisingly, remyelinated fibers appeared after 5 weeks despite the maintenance of compression, although some partially and completely demyelinated fibers persisted. Therefore, compressive optic neuropathy is secondary to blockage of axonal transport and demyelination insufficient to cause cell death. Smith et al. showed that remyelination can restore conduction of impulses at physiological frequencies in previously demyelinated central fibers, so remyelinated fibers are presumably present at decompression in clinical situations.

Kayan and Earl demonstrated that most patients undergoing decompression of lesions acting on the optic nerves and chiasm achieved complete or nearly complete visual recovery within 15 days. Miller reported that patients with pituitary adenoma and normal fundi invariably demonstrated impressive returns of visual acuity and measurable fields immediately upon recovery from anesthesia. McDonald found two courses for visual recovery following optic nerve decompression: rapid recovery within hours or days, and late recovery weeks later. Rapid recovery is due to the reversal of the "physiological block," and the late recovery is due to restored conduction through remyelinated fibers. In our patients, rapid recovery of vision was observed after surgery, suggesting the former mechanism was involved. However, the actual mechanisms causing such striking visual recovery following surgical decompression remain unclear.

Direct obliteration of the aneurysmal neck allows observation of the microanatomical relationship between the aneurysmal dome and the compressed optic nerve. In both our cases and that of Striph et al., the optic nerve was compressed by the aneurysm. We trapped or clipped the aneurysm, followed by removal of the intraluminal clot, but did not attempt to dissect the dome from the compressed optic nerve to prevent further surgical damage. We recommend either internal decompression by direct clot removal or, if the aneurysm is unclippable, indirect reduction in size using various surgical techniques. Whether visual loss due to optic nerve compression can be recovered after any period is unknown, but surgical treatment should be considered for any such patients. Our cases suggest that surgical decompression can achieve substantial visual recovery from monocular blindness due to aneurysmal compression of the optic nerve.

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References


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