Endoscopic Optic Nerve Decompression for Nontraumatic Optic Neuropathy

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Objective: To determine the efficacy of endoscopic optic nerve decompression for the treatment of patients with nontraumatic optic neuropathy.

Design: Retrospective case series.

Setting: Academic medical center.

Patients: Ten optic nerve decompressions were performed on 7 patients with nontraumatic optic neuropathy caused by various pathologic entities, including meningioma, lymphangioma, fibro-osseous lesions (fibrous dysplasia and osteoma), mucopyocele, and Graves orbitopathy.

Interventions: Endoscopic instrumentation was used in a transnasal fashion to decompress the optic nerve.

Main Outcome Measures: Visual acuity and complication rates.

Results: Mean visual acuity improved from 20/300 preoperatively to 20/30 after surgery. Visual acuity improved by at least 2 lines on the Snellen chart following 7 of the 10 decompressions. Median operative time was 133 minutes, and median length of stay was less than 24 hours. Complications were limited to postoperative hyponatremia and corneal abrasions, both of which resolved with conservative therapy. Mean follow-up time was 6.1 months.

Conclusion: Endoscopic optic nerve decompression appears to be an effective treatment for restoring visual acuity in select patients who present with compressive optic neuropathy.

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While many approaches have been described for optic nerve decompression, the transnasal endoscopic approach has gained favor because it affords excellent visualization of the orbital apex and optic canal with minimum patient morbidity. Multiple authors have reported their experience with endoscopic optic nerve decompression for traumatic optic neuropathy, but few series exist, to our knowledge, describing endoscopic optic nerve decompression as a treatment for nontraumatic, compressive optic neuropathy. Because the utility of decompression for traumatic optic neuropathy has recently been questioned, the role of optic nerve decompression for nontraumatic, compressive optic neuropathy is of particular interest. We present our recent experience with 10 optic nerve decompressions performed for compressive, nontraumatic optic neuropathy.

Methods

Data were collected from preoperative and postoperative ophthalmologic examinations as well as operative reports and hospital charts. Patients who underwent decompression for traumatic optic neuropathy were excluded from the study population.

While the surgical details vary considerably depending on the compressive abnormality encountered, in general, a standard total ethmoidectomy was performed at the start of each procedure. The natural ostium of the sphenoïd sinus was identified and opened widely to allow maximal visualization of the orbital apex. A limited posterior orbital decompression was then performed to access the optic canal using a spoon curette to fracture the lamina papyracea approximately 1 cm anterior to the sphenoid face. A Cottle elevator was used to elevate the thin bone of the lamina in a posterior direction. The underlying periostea was preserved to prevent herniation of orbital fat that could obstruct the operative field.

As the orbital apex transitions to the optic canal, the overlying bone becomes thick, forming the optic ring. This bone was carefully removed with a diamond burr. Copious irrigation was used to avoid thermal damage to the underlying optic nerve. Once the bone was appropriately thinned, it was elevated with a microureter in a medial direction, away from the optic nerve. The remaining bone of the optic canal was of varying thickness and frequently required thinning with a drill prior to decompression. The posterior extent of decompression was determined by the position of the optic nerve as visualized through the endoscope.

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sion depended on the nature of compressive abnormality; typically, the nerve was decompressed for a distance of 1 cm within the sphenoid sinus (Figure 1). Ideally, the canal was opened widely enough to relieve all areas of compression. We do not advocate incising the optic nerve sheath.

RESULTS

STUDY POPULATION

Ten endoscopic optic nerve decompressions were performed in 7 patients from 1996 to 2005. The study population consisted of 4 female and 3 male patients with a mean age of 49 years (age range, 14-81 years). Preoperative compressive optic neuropathy with decreasing visual acuity was present in 9 cases. Patient 3 had Graves ophthalmopathy with normal visual acuity but congestion and hemorrhage around the optic disc. Preoperative visual acuity and the nature of the compressive abnormality are summarized in the Table.

Median operative time and estimated blood loss were 133 minutes and 147 mL, respectively. Median hospital stay was less than 24 hours. Visual acuity improved by at least 2 lines on the Snellen chart in 7 of the 10 cases (70%), remained stable in 2 patients, and was subjectively improved in patient 5 (Table). Mean visual acuity improved from 20/300 to 20/30 following decompression, corresponding to a median improvement of 4 lines on the Snellen chart.

There were no intraoperative complications. Postoperative complications included corneal abrasions in patient 1. The cause of the mild abrasions was believed to be from scleral shields placed during surgery to protect the eyes in the event that an external approach was deemed necessary. All symptoms resolved within 12 hours with conservative treatment, including antibiotic ointment and eye drops. Patient 4, who had a large intracranial meningioma, developed postoperative hyponatremia attributed to syndrome of inappropriate antidiuretic hormone secretion. This complication was managed successfully with fluid restriction.

REPORT OF A CASE

Patient 1 was a 20-year-old college student who presented with progressive, left-sided visual loss and proptosis of 6 months' duration. The visual impairment was initially attributed to traumatic optic neuropathy following mild head trauma during a basketball game, and no treatment was initiated. At the time of presentation to our institution, he described the vision in his left eye as “very dim” and noted left retro-orbital pressure for the preceding month.

Ophthalmologic examination at the time of presentation demonstrated 6 mm of proptosis, a visual acuity of 20/1000, decreased color vision, and an afferent pupillary defect. A computed tomographic scan demonstrated a 6-cm sphenoid ethmoid osteoma replacing the medial orbital wall, extending into the sphenoid sinus, and compressing the optic nerve (Figure 2).

The patient was taken to the operating room where endoscopic resection of the osteoma was performed with the use of image guidance. A drill was used to hollow out the osteoma, and the remaining walls were in-fractured into the nasal cavity. Following identification of a cleavage plane, the skull-base attachment of the osteoma was down-fractured. Postoperatively, the patient was noted to have mild corneal abrasions secondary to the use of scleral shields during the procedure, which responded to conservative therapy. Postoperative visual acuity improved to 20/50 with resolution of the afferent papillary defect. The patient was discharged home within 48 hours. A computed tomographic scan 3 months following surgery confirmed complete resection of the osteoma (Figure 2).

COMMENT

Optic neuropathy secondary to compression of the optic nerve is a rare condition that may be caused by a variety of pathologic conditions including tumor, infec-

Table. Preoperative and Postoperative Visual Acuity

<table>
<thead>
<tr>
<th>Patient No./ Case No.</th>
<th>Compressive Abnormality</th>
<th>Preoperative Visual Acuity</th>
<th>Postoperative Visual Acuity</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/1 Osteoma</td>
<td>20/1000</td>
<td>20/50</td>
<td></td>
</tr>
<tr>
<td>2/2 Mucopyocele</td>
<td>20/1000</td>
<td>20/50</td>
<td></td>
</tr>
<tr>
<td>3/3 Graves disease</td>
<td>20/300</td>
<td>20/30</td>
<td></td>
</tr>
<tr>
<td>4/4 Meningioma</td>
<td>NLP</td>
<td>NLP</td>
<td></td>
</tr>
<tr>
<td>5/5 Meningioma</td>
<td>20/50</td>
<td>Subjective improvement</td>
<td></td>
</tr>
<tr>
<td>6/6 Lymphangioma</td>
<td>20/80</td>
<td>20/20</td>
<td></td>
</tr>
<tr>
<td>7/7 Fibrous dysplasia</td>
<td>20/40</td>
<td>20/20</td>
<td></td>
</tr>
<tr>
<td>7/8 Fibrous dysplasia</td>
<td>20/100</td>
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<td>7/9 Fibrous dysplasia</td>
<td>20/50</td>
<td>20/30</td>
<td></td>
</tr>
<tr>
<td>7/10 Fibrous dysplasia</td>
<td>20/50</td>
<td>20/20</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviation: NLP, no light perception.
tion, and bone dysplasias. When the pathologic cause of compression is amenable to surgical correction, decompression of the optic canal should be considered as a primary treatment. Multiple surgical approaches have been described for decompression of the optic nerve including transfacial approaches using a microscope, neurosurgical craniotomy approaches, and the transnasal endoscopic approach. The endoscopic approach offers the benefit of minimum morbidity in terms of postoperative recovery and external scars. Also, excellent visualization of the optic nerve is obtained. With the endoscopic approach, however, access to the optic nerve is typically limited to the inferomedial portion of the nerve—that portion of the nerve that borders the sphenoid sinus. Thus, for patients with compressive lesions involving the superior or lateral aspect of the nerve, neurosurgical approaches must be considered.

While several cases of endoscopic optic nerve decompression for nontraumatic optic neuropathy have been reported in the literature, details regarding the procedures and results are extremely limited, to our knowledge. Luxenberger et al. comment on 7 cases of optic nerve decompression for nontraumatic causes. Four of these patients had Graves ophthalmopathy, as did 1 patient in the present study. For most patients with optic neuropathy from Graves disease, the literature demonstrates that decompression of the orbital apex without formal optic canal decompression is sufficient. The remaining 3 patients underwent decompression for a hemangioma of the orbital apex, retrobulbar neuropathy of unclear cause, and an orbital complication of sinusitis with few details regarding visual outcomes.

The literature regarding endoscopic optic nerve decompression focuses on patients with traumatic optic neuropathy. Evaluation of the therapeutic efficacy of decompression in the setting of traumatic optic neuropathy is complicated by a high rate of spontaneous improvement without intervention. Thus, the role of decompression in the traumatic setting is unclear. Optic neuropathy secondary to nontraumatic lesions, however, does not have the same expected spontaneous recovery. Thus, despite the retrospective nature of the pres-
ent study and lack of controls, the finding that all patients had either improvement or stabilization of their visual acuity might be reasonably interpreted to demonstrate at least short-term efficacy of endoscopic decompression for this condition.

Because of the overall rarity of nontraumatic compressive optic neuropathy and the heterogeneity of causal lesions, it is important not to draw overreaching conclusions from this study. An analysis of the cases in which decompression failed to achieve significant, lasting visual improvement, however, is of value. Case 3 did not result in visual improvement, but this patient had nearnormal vision (20/30) prior to surgery. Decompression was performed to prevent visual loss based on the ominous preoperative findings of hemorrhage and edema around the optic disc. Case 4 involved long-standing (4 months), complete visual loss from a suspected meningioma. A tissue diagnosis was required, and concomitant endoscopic decompression was performed with a low expectation of visual recovery. No visual improvement was noted in this patient.

In case 5, subjective improvement was reported, but the patient did not undergo formal postoperative visual evaluation. This patient had a large intracranial meningioma with complete visual loss in the contralateral eye following intracranial surgery and radiation therapy. When she developed a progressive decline of vision in her only seeing eye despite systemic steroid therapy, a unilateral decompression was performed. The patient subsequently returned to hospice and, although she reported improved vision, no formal testing was performed.

While endoscopic decompression did not achieve long-term improvement in visual acuity for the patient with fibrous dysplasia (patient 7, cases 7-10), significant short-term visual improvement was noted following each procedure. Large doses of preoperative systemic steroids had failed to achieve similar improvements. Unfortunately, regrowth of dysplastic bone resulted in the need for repeated decompression at an average interval of 6 months. Eventually, a craniotomy was performed for superior decompression of the optic canal with early success; recurrent visual decline has necessitated a second intracranial procedure. While this patient remains a therapeutic dilemma, the consistent short-term success of endoscopic decompression suggests the potential benefit of this procedure for the treatment of patients with less aggressive dysplastic disease.

In conclusion, endoscopic optic nerve decompression is a safe and effective treatment for selected patients with compressive optic neuropathy from nontraumatic causes.

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Author Contributions: Dr Pletcher had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. Study concept and design: Pletcher and Metson. Acquisition of data: Metson. Analysis and interpretation of data: Pletcher and Metson. Drafting of the manuscript: Pletcher and Metson. Critical revision of the manuscript for important intellectual content: Pletcher and Metson. Administrative, technical, and material support: Pletcher and Metson. Study supervision: Metson.

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REFERENCES