Endoscopic endonasal optic nerve and orbital apex decompression for nontraumatic optic neuropathy: surgical nuances and review of the literature

MONCEF BERHOUMA, M.D., M.Sc.,¹ TIMOTHEE JACQUESSON, M.D., M.Sc.,^{1,2}
LUCIE ABOUAF, M.D., M.Sc.,^{2,3} ALAIN VIGHETTO, M.D., Ph.D.,^{2,3}
AND EMMANUEL JOUANNEAU, M.D., Ph.D.^{1,2,4}

¹Skull Base Surgery Unit, Department of Neurosurgery B, and ³Department of Neuro-ophthalmology, Pierre Wertheimer Neurological and Neurosurgical Hospital, Hospices Civils de Lyon; ²Research and Education Unit of Medicine, Claude Bernard University Lyon 1; and ⁴INSERM U1028, CNRS UMR5292, Neurosciences Research Center of Lyon, Neuro-oncology and Neuro-inflammation Team, Lyon, France

Object. While several approaches have been described for optic nerve decompression, the endoscopic endonasal route is gaining favor because it provides excellent exposure of the optic canal and the orbital apex in a minimally invasive manner. Very few studies have detailed the experience with nontraumatic optic nerve decompressions, whereas traumatic cases have been widely documented. Herein, the authors describe their preliminary experience with endoscopic endonasal decompression for nontraumatic optic neuropathies (NONs) to determine the procedure's efficacy and delineate its potential indications and limits.

Methods. The medical reports of patients who had undergone endoscopic endonasal optic nerve and orbital apex decompression for NONs at the Lyon University Neurosurgical Hospital in the period from January 2012 to March 2014 were reviewed. For all cases, clinical and imaging data on the underlying pathology and the patient, including demographics, preoperative and 6-month postoperative ophthalmological assessment results, symptom duration, operative details with video debriefing, as well as the immediate and delayed postoperative course, were collected from the medical records.

Results. Eleven patients underwent endoscopic endonasal decompression for NON in the multidisciplinary skull base surgery unit of the Lyon University Neurosurgical Hospital during the 27-month study period. The mean patient age was 53.4 years, and there was a clear female predominance (8 females and 3 males). Among the underlying pathologies were 4 sphenoorbital meningiomas (36%), 3 optic nerve meningiomas (27%), and 1 each of trigeminal neuroma (9%), orbital apex meningioma (9%), ossifying fibroma (9%), and inflammatory pseudotumor of the orbit (9%). Fifty-four percent of the patients had improved visual acuity at the 6-month follow-up. Only 1 patient whose sphenoorbital meningioma had been treated at the optic nerve atrophy stage continued to worsen despite surgical decompression. The 2 patients presenting with preoperative papilledema totally recovered. One case of postoperative epistaxis was successfully treated using balloon inflation, and 1 case of air swelling of the orbit spontaneously resolved.

Conclusions. Endoscopic endonasal optic nerve decompression is a safe, effective, and minimally invasive technique affording the restoration of visual function in patients with nontraumatic compressive processes of the orbital apex and optic nerve. The timing of decompression remains crucial, and patients should undergo such a procedure early in the disease course before optic atrophy.

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KEY WORDS • skull base surgery • minimally invasive neurosurgery •
endoscopic endonasal surgery • optic nerve decompression • orbital tumors •
optic neuropathy

ALTHOUGH several surgical approaches have been historically described for optic nerve decompression,⁴ the endoscopic endonasal technique is gaining popularity in the skull base surgeon's armamentarium.^{1,7,10,14,26,36,43,58} This technique definitely provides excellent exposure of the optic canal and orbital apex in a minimally invasive fashion. While decompression in

traumatic optic neuropathy has been widely investigated,^{11,13,18–20,24,28,29,36,37,40,47,48,52–59,61} indeed with debatable results, decompression for nontraumatic optic neuropathies (NONs) is still insufficiently studied.^{2,3,5,12,15,17,21–23,25–27,33,35,36,39,41,42,45,46,51} We hypothesize that the minimally invasive endoscopic endonasal approach will be more widely used in decompressions for nontraumatic pathologies of the orbital apex. We present our preliminary series of 11 optic nerve decompressions performed for NONs. The surgical technique is detailed and potential indications are discussed.

Abbreviations used in this paper: NON = nontraumatic optic neuropathy; TOF = time-of-flight.

Methods

Patient Cohort

We reviewed the medical records of all patients who had undergone endoscopic endonasal decompression for NONs at the Lyon University Neurosurgical Hospital in the period from January 2012 to March 2014. Data were collected from preoperative and postoperative ophthalmological examinations, operative reports, and hospital records.

Surgical Management

Preoperative orbital MR images and thin-slice bone CT scans were reviewed to determine the most appropriate surgical approach and the precise anatomy of the sphenoethmoidal complex (the Onodi cells, septa, bone thickness, and so forth). The surgical procedure was standardized, involving advanced endoscopic techniques derived from pituitary surgery and intraoperative navigation systems coupling CT and MRI data.

Data Collection

For all cases, clinical and imaging data on the underlying pathology and the patient, including demographics, pre- and postoperative ophthalmological assessment results, symptom duration, operative details with video debriefing, as well as the immediate and delayed postoperative course, were collected from the medical records. The ophthalmological assessment included visual acuity testing according to the decimal scale of Monoyer, a fundoscopic examination, and computerized visual field testing.

Literature Review

An exhaustive and systematic review of the literature was performed using large biomedical databases (PubMed, Google Scholar, ScienceDirect, and Scopus) and the key words “optic nerve,” “orbital apex,” “decompression,” “optic neuropathy,” “endoscopic endonasal,” and “minimally invasive neurosurgery.”

Results

Patient Characteristics

Eleven patients underwent endoscopic endonasal decompression for NON in the multidisciplinary skull base surgery unit of the Lyon University Neurosurgical Hospital during the 27-month study period (Table 1). The mean patient age was 53.4 years, and there was a clear female predominance (8 females and 3 males). Among the underlying pathologies were 4 sphenoorbital meningiomas (36%), 3 optic nerve meningiomas (27%), and 1 each of trigeminal neuroma (9%), orbital apex meningioma (9%), ossifying fibroma (9%), and inflammatory pseudotumor of the orbit (9%).

Surgical Technique

Of the 11 patients, 4 underwent a secondary surgical procedure for the underlying pathology between 3 and 6 months after the optic nerve decompression: One orbital

meningioma was secondarily totally resected through an intracranial subfrontal craniotomy, and 3 sphenoorbital meningiomas were resected in a classic frontotemporal manner. Two patients did benefit from a single procedure: one with a trigeminal neuroma removed through an anteromedial corridor to Meckel's cave and one with an ossifying fibroma of the medial orbital apex.

Visual Outcome

Fifty-four percent of the patients had improved visual acuity at the 6-month follow-up. Only 1 patient (9%) whose sphenoorbital meningioma had been treated at the optic nerve atrophy stage continued to worsen despite surgical decompression. The 2 patients presenting with preoperative papilledema totally recovered. Four patients (36.4%) remained stable or improved slightly (< 2/10 of an increase on the visual acuity scale).

Postoperative Complications

One patient presented with a moderate epistaxis on the 3rd postoperative day, requiring a compressive inflatable balloon for 48 hours. A second patient experienced swelling of the orbit by air insufflation during sneezing on the 2nd postoperative day, which spontaneously resolved within 1 week.

Illustrative Cases

Case 3

A 64-year-old man, whose medical history included chronic leukemia treated with chemotherapy, was admitted for 6 months of progressive visual decline in the left eye and left fronto-orbital dysesthesias. Visual assessment revealed deficient left visual acuity (counts fingers at 50 cm), decreased color vision, and an afferent pupillary defect. Fundoscopy revealed left papillary pallor. Three-dimensional time-of-flight (TOF) MRI (Fig. 1A) showed an enhancing lesion of the left orbital apex following the trajectory of the superior orbital fissure. Because of the facial dysesthesia, a diagnosis of trigeminal neuroma was discussed, even though a metastatic lesion due to the chronic leukemia could not be eliminated in this context. With the patient under general anesthesia and the aid of image guidance, we approached the lesion via a left sphenoethmoidal approach. After performing a wide opening of the left posterior ethmoidal cells, including the Onodi cell, we identified the left optic canal, as well as the anteromedial wall of the left Meckel's cave lateral to the left internal carotid artery (Fig. 1B). The left lamina papyracea was then easily outfractured using a blunt microspatula, and the optic canal was drilled from lateral to medial under thorough saline irrigation. The thin eggshell bone remaining on the optic nerve was removed at approximately 270° until pulsations of the optic nerve sheath were observed (Fig. 1C and D). The tumor was subsequently removed through the anteromedial wall of Meckel's cave. Histopathological examination confirmed the diagnosis of trigeminal neuroma. The postoperative course was uneventful, and visual acuity was stable. At the 6-month follow-up, a slight left facial dysesthesia remained, left visual acuity improved to 6/10, and no tumor recurrence was noted.

Endoscopic endonasal optic nerve decompression

TABLE 1: Summary of characteristics in 11 patients who underwent endoscopic endonasal optic nerve and orbital apex decompressions between 2012 and 2014*

Case No.	Sex, Age (yrs)	Underlying Pathology	Visual Acuity		Visual Field Assessment	Funduscopy		Complementary Management
			Preop	Postop		Preop	Postop	
1	F, 52	LON meningioma	LP	2/10	stable concentric deficit	papillary pallor	papillary pallor	radiotherapy
2	F, 49	SpO meningioma	4/10	7/10	improvement	papillary pallor	papillary pallor	follow-up
3	M, 64	trigeminal neuroma	CF50	6/10	improvement	papillary pallor	papillary pallor	removal during same procedure
4	F, 67	LON meningioma	CF10	CF10	stable temporal deficit	papillary atrophy	papillary atrophy	radiotherapy
5	F, 56	SpO meningioma	1/10	3/10	stable concentric deficit	papillary pallor	papillary pallor	secondary surgical removal
6	F, 60	LON meningioma	7/10	10/10	improvement	normal	normal	radiotherapy
7	F, 30	orbital apex meningioma	8/10	9/10	normal	papilledema Grade 2	normal	secondary surgical removal
8	F, 68	inflammatory pseudotumor	CF10	8/10	normal	papilledema Grade 1	normal	follow-up
9	M, 27	ossifying fibroma	7/10	8/10	normal	normal	normal	removal during same procedure
10	M, 61	SpO meningioma	6/10	4/10	stable temporal deficit	papillary atrophy	papillary atrophy	secondary surgical removal
11	F, 54	SpO meningioma	7/10	8/10	normal	normal	normal	secondary surgical removal

* All postoperative assessments were performed at the 6th postoperative month except for the patient in Case 11, who was evaluated at the 3rd month (most recent patient). CF10 = counts fingers at 10 cm; CF50 = counts fingers at 50 cm; LON = left optic nerve; LP = light perception; SpO = sphenoorbital.

Case 9

A 27-year-old man without any remarkable medical history presented with progressive left proptosis and slight visual decline in the left eye. Visual acuity reached 10/10 in the right eye and 7/10 in the left, with no anomalies on fundoscopic examination and visual field testing. Imaging revealed a calcified nonenhancing tumor of the left medial orbital wall and optic canal, suggesting an ossifying fibroma (Fig. 2A). An endoscopic endonasal approach via the left nostril (Fig. 2B) was performed, allowing subtotal removal of a crumbly osseous tumor using a piecemeal technique along with irrigated ultrasonic drilling. The medial orbital wall and apex were drilled until the periorbital layer was reached. The left optic canal was consequently opened from lateral to medial (Fig. 2C). The postoperative course was uneventful. The diagnosis of benign ossifying fibroma was definitively established. Follow-up evaluation at 6 months after treatment confirmed an improvement in left visual acuity that reached 8/10 and a reduction in the proptosis. Computed tomography scanning showed good decompression of the orbital apex and optic canal (Fig. 2D), as well as an anterior medial remnant, which is being monitored yearly in the absence of any symptoms except a slight proptosis.

Discussion

Optic nerve decompression has been thoroughly described for traumatic optic neuropathy.^{23,29,50,57} Nontraumatic optic neuropathy is a rare condition that can be

caused by a variety of disorders, including tumors, endocrine orbitopathy, idiopathic intracranial hypertension, bone dysplasia, or infectious processes (Table 2). Therefore, surgical decompression of the optic nerve may be either part of the treatment for the primary compressive process (for example, an orbital apex tumor) or a preliminary step before treating the underlying cause via an intracranial route (for example, sphenoorbital meningioma).

Various surgical approaches⁴ have been described for decompression of the optic nerve: a medial approach by external ethmoidectomy, an inferomedial approach via a transantral transethmoidal route, a supraorbital transcranial approach, and the endoscopic endonasal transsphenoid approach. This latter approach affords excellent visualization of the optic canal and medial orbital apex with minimal morbidity and no brain retraction or scarring. Nevertheless, the optimal timing and the visual benefits are still questioned in the absence of a prospective study.⁴¹

Surgical Anatomy

Considered as an extension of the brain, each optic nerve is enveloped by 3 meningeal layers (Figs. 3–6).^{8,16,60} Each measures about 50 mm in length. Four anatomical segments can be described: intraocular (1 mm), intraorbital (25–30 mm), intracanalicular (10 mm), which is the most vulnerable to compression, and intracranial (10 mm). It is the intracanalicular segment that is addressed in endoscopic endonasal decompression. In this segment the optic nerve is fixed by the fibrous annulus of Zinn. From

FIG. 1. Case 3. **A:** Axial 3D TOF high-resolution T1-weighted MR sequence with Gd showing trigeminal neuroma (yellow arrows) of the left orbital apex. **B:** Endoscopic view through the left nostril after sphenoidotomy and posterior ethmoidectomy. Asterisk indicates the anteromedial corridor to the left Meckel's cave. Black and yellow dotted lines mark the borders of the structure indicated. **C:** View after a lateral to medial decompression of the left orbital apex and optic nerve, before trigeminal neuroma removal. Asterisk indicates the anteromedial corridor to the left Meckel's cave. Black and yellow dotted lines mark the borders of the structure indicated. **D:** Postoperative CT scan showing the extent of left optic canal and orbital apex decompression (dotted and solid yellow lines). CA = internal carotid artery; CR = clival recess; OA = orbital apex; ON = left optic nerve within its canal; S = sella turcica; Tbc = tuberculum sellae.

the endoscopic endonasal view, the optic nerve is covered by a thin bone of about one-half millimeter in the majority of patients and is uncovered in 4% of patients. In comparison, the carotid artery is uncovered in the sphenoid sinus in about 8% of patients. These conditions are of paramount importance during drilling of the optic canal.

The optic canal is fashioned from the 2 struts of the lesser wing of the sphenoid bone. It contains the optic nerve and the ophthalmic artery. This canal is approximately 10 mm long and 4–5 mm wide. It is thinner and wider proximally and thicker and narrower distally. At this point, the dura mater and the periosteal layer of the

TABLE 2: Summary of published series on endoscopic endonasal optic nerve and orbital apex decompression for NON during the last 10 years*

Authors & Year	No. of Patients	Mean Age (yrs)	Underlying Pathology	Complication	% w/ Good Visual Outcome
Lund & Rose, 2006	12	42.6	sphenoid wing meningioma	no	58.3
Pletcher & Metson, 2007	7 (10 decompressions)	49	meningioma, lymphangioma, mucocoele, fibrous dysplasia, Graves' orbitopathy	postop hyponatremia, corneal abrasions	70
Attia et al., 2012	8	NA	suprasellar meningioma	no	37.5
Sencer et al., 2014	10	34.1	idiopathic intracranial hypertension	no	80
current study	11	53.4	sphenoorbital meningioma, optic nerve meningioma, orbital meningioma, inflammatory pseudotumor, ossifying fibroma, trigeminal neuroma	epistaxis, pneumo-orbit	54.5

* Series dealing with traumatic optic neuropathy or including fewer than 3 patients were excluded. NA = not available.

Fig. 2. Case 9. **A:** Preoperative CT scan demonstrating an ossifying fibroma of the medial orbital apex and left optic canal. **B:** View after sphenoidotomy and posterior ethmoidectomy via an endoscopic endonasal approach through the left nostril. CA = right carotid artery; CR = clival recess; ON = right optic nerve (contralateral nerve); ST = sella turcica. Double asterisks indicate tumor of the left orbital apex covering the left optic canal. **C:** View after tumor drilling and decompression via an endoscopic endonasal approach through the left nostril. CR = clival recess; ICA = left carotid artery; ION = left optic nerve; OA = left orbital apex; rCA = right carotid artery; rON = right optic nerve. **D:** Postoperative CT scan demonstrating sufficient decompression of the left optic nerve and medial orbital apex (dotted yellow line). Note the remnant on the most anterior medial wall (yellow arrows) requiring regular follow-up.

optic canal fuse to form the periorbita. Typically, the ophthalmic artery lies inferolateral to the optic nerve, but a more medial trajectory is possible and hence should be considered during decompression. The central retinal artery leaves the ophthalmic artery 10 mm behind the globe.

As seen from below, the optic canal is in direct contact with the lateral sphenoid recess and the most posterior ethmoidal cells. Among these cells are some sphenothmoidal cells, also known as Onodi cells, which may directly cover the optic canal and must be opened to expose the optic nerve.

Pathophysiology

From a pathophysiological perspective,^{50,55} function of the optic nerve may be altered by a combination of CSF circulation dysfunction, ischemia from arterial injury (ophthalmic artery and central retinal artery), and a direct axoplasmic transport interruption (conduction block, demyelination, degeneration). Edema and hemorrhage inside the nerve are also possible, especially in acute traumatic optic neuropathy. While decompression in acute traumatic optic neuropathy is useful exclusively in the first 3–6 hours, the timing of decompression in chronic optic neuropathy is debatable. Moreover, there is no evidence that

Fig. 3. Endoscopic anatomical view after wide anterior bilateral sphenoidotomy and posterior ethmoidectomy. The sella turcica (S) lies on the midline, limited posteriorly by the clival recess (CR) and anteriorly by the tuberculum sellae (Tbc) and planum sphenoidale (Pla). The sella is bordered laterally by both optic canals (OC) with an anterolateral trajectory toward the orbital apex (OA) and both internal carotid arteries (CA). OCR = opticocarotid recess; On = Onodi cell.

FIG. 4. Endoscopic anatomical view of the right orbital apex demonstrating the relationships of the optic canal (OC) with the posterior ethmoidal artery (PEA) anteriorly and with the superior orbital fissure (SOF) antero-inferiorly. The endoscopic endonasal optic nerve decompression also requires an orbital apex decompression (black dotted lines). Red dotted lines indicate the course of the carotid artery. CA = carotid artery; OA = orbital apex; S = sella turcica; Tbc = tuberculum sellae.

high-dose corticosteroids, particularly as cell membrane stabilizers, are useful in NON.

Patient Evaluation

A detailed ophthalmological assessment should in-

FIG. 5. View after bone removal of the left orbital apex showing the relationships of the left optic nerve with soft tissues. 1, left posterior ethmoidal cell; 2, posterior ethmoidal artery; 3, dura of the anterior skull base; 4, anterior intercavernous sinus; 5, sellar dura; 6, clival recess; 7, internal carotid artery (parasellar segment); 8, internal carotid artery (paraclival segment); 9, superior orbital fissure; 10, left sphenopalatine artery; 11, left optic nerve sheath; 12, periorbital; 13, medius rectus and orbital fat. Black dotted line indicates the orbital apex and optic nerve decompressed.

FIG. 6. Axial illustration of the orbital apex. 1, posterior ethmoidal cell; 2, lamina papyracea; 3, nasal septum; 4, optic nerve; 5, ophthalmic artery; 6, annulus of Zinn; 7, internal carotid artery; 8, temporal lobe tip; 9, medius rectus. Copyright Moncef Berhouma. Published with permission.

clude at least fundoscopic examination, visual acuity evaluation, and computerized visual field testing. As the perception of red is lost first, color vision can also be tested. If the patient is unable to cooperate, monitoring of visually evoked potentials is valuable.

Imaging of the optic canal relies mainly on thin-slice CT scanning. This modality accurately shows in 3 anatomical planes (axial, sagittal, and coronal) the pneumatization and septations of the sphenoid sinus and the ethmoidal cells, as well as any uncovered carotid artery. In sphenoorbital meningiomas or dysplasia, CT scanning reveals bone density and thickness. Magnetic resonance imaging should include not only traditional sequences (T1-weighted with or without Gd, T2-weighted) but also a 3D TOF high-resolution sequence. This latter sequence shows very accurately the relationships between the optic nerve and the underlying pathological compressive lesion as well as the position of the ophthalmic artery.

Surgical Technique

Today, the surgical technique is well standardized with very few variants.^{4,16,19,30–34,38,44,60} The approach is very similar to that used for pituitary surgery;^{6,9} therefore, most skull base surgeons are familiar with it.⁴⁹ With the

Endoscopic endonasal optic nerve decompression

patient under general anesthesia and orotracheal intubation, his or her head is secured in a 3-pin head holder, and neuronavigation is calibrated with a fusion of CT and MRI data. An intravenous dose of second-generation cephalosporin is administered. The head is slightly flexed (approximately 30°) as for a standard pituitary approach and turned slightly toward the surgeon for a better ergonomic position. The nose is rinsed with iodine ointment, and the abdomen is prepared for graft harvesting in case of CSF leakage requiring skull base reconstruction. Both nasal fossae are decongested using cotton soaked in a solution of lidocaine mixed with adrenalin. The draping should leave both eyes exposed in the operative field so that evaluation of the globes can be performed if required intraoperatively. An adjustable self-retaining holding arm is secured to the operating table to be used at specific phases of the surgical procedure. The nasal phase is usually performed with a 0° rigid endoscope in the nostril ipsilateral to the optic neuropathy. A middle turbinectomy and a posterior ethmoidectomy are performed before opening the anterior wall of the sphenoid sinus. All septa within the sphenoid sinus are widely opened to allow optimal visualization of exocranial skull base, that is, the sella turcica between the parasellar carotid arteries and the optic canals as well as the opticocarotid recesses (Fig. 3). Toward the lateral sphenoid recesses, the medial orbital apex can be recognized (lamina papyracea). In some patients, carotid arteries and/or optic nerves are not covered by bone. Special attention is required to identify any sphenothmoidal cell (Onodi cell) that needs to be opened carefully to expose the optic canal. Neuronavigation may be very helpful in a nonpneumatized sphenoid sinus requiring time-consuming bone drilling. At this point, a long rigid endoscope is secured within the self-retaining endoscope holder. The decompression progresses from lateral to medial beginning at the level of the lamina papyracea, which is usually very thin and easily resected with a spoon curette or a spatula. Special care is taken to keep the periorbital fat intact to avoid the bulge of the orbital fat within the field. Decompression progresses to the optic canal, which is drilled using either a rotating tip or an ultrasonic bone cutter always under generous irrigation to limit heat diffusion to the optic nerve. Once the bone of the optic canal is thinned, it is outfractured with a blunt spatula away from the optic nerve. This bony decompression should reach at least 180° around the optic nerve and optimally up to 270° (superomedial, medial, and inferomedial aspects). Pulsations of the optic nerve signal good decompression. Opening the optic nerve sheath is very controversial and exposes the patient to CSF leakage as well as the ophthalmic artery to injury and should therefore be reserved for very specific cases (for example, idiopathic intracranial hypertension).

If a tumor is present, it can be resected or biopsied. At the end of the procedure, a thin layer of biological glue is sprayed on the optic nerve and the carotid artery to optimize hemostasis and protect these structures from the sphenoid mucosa. No nasal packing is needed.

Postoperative Management

A preliminary visual assessment is performed in the recovery room. The role of perioperative steroids is still debated. Vision is monitored, as is any hemorrhagic nasal

discharge during the first 3 postoperative days. Daily nasal saline sprays are useful for accelerating mucosal healing and for patient comfort. Patients are usually discharged after 4–5 days. We propose performing an endoscopic examination with a local anesthetic to verify healing, as well as a complete visual assessment, 1 month after surgery. A control CT scan is also obtained to assess the extent of decompression.

Indications for and Timing of Surgery

The role of endoscopic endonasal decompression is debatable for traumatic optic neuropathy, mainly because of the high rate of spontaneous improvements without intervention; however, this natural resolution is not expected in nontraumatic chronic cases.^{5,50,57} Our positive visual results in the short term in the present study, despite the study's retrospective nature, limited number of patients, and heterogeneous series, reflect an obvious benefit from a minimally invasive technique with very low morbidity. The technique can be proposed for diverse pathologies (Table 3) threatening the optic nerve(s) in their intracanalicular portion (Fig. 7).

Only a few studies dealing with endoscopic endonasal decompression for NON are reported in the literature.^{5,35,41,45} Pletcher and Metson described 10 decompressions in 7 patients with various underlying pathologies similar to those in our series, with a good visual outcome for 70% of the patients.⁴¹ Studies dealing with idiopathic intracranial hypertension involve opening of the optic nerve sheath, raising the risk of CSF leakage.

Conclusions

Our preliminary results confirm the potential benefits of endoscopic endonasal optic nerve decompression for nontraumatic neuropathies. This minimally invasive technique allows tissue sampling and/or tumor removal if indicated during the same procedure. Optic nerve decompression must be considered early in the course of a disease before the onset of optic nerve atrophy. Nevertheless, given the variety of possible underlying pathologies, it is important to not draw premature conclusions about any recommendation in the absence of randomized studies and more homogeneous series.

TABLE 3: Potential indications for endoscopic endonasal optic nerve and orbital apex decompression

optic nerve sheath meningioma
osteopetrosis
fibrous dysplasia
endocrine orbitopathy
orbital apex tumors
medial orbital tumors
mucocoele
idiopathic intracranial hypertension
cavernous sinus tumors w/ extension to optic canal
sphenoorbital meningioma
cortico-resistant inflammatory optic &/or orbital disease

FIG. 7. Examples of pathologies amenable to endoscopic endonasal optic nerve decompressions. **A:** Left optic nerve sheath meningioma (Case 6). **B:** Left sphenoorbital meningioma (Case 11). **C:** Right orbital apex meningioma (Case 7). **D:** Left optic nerve meningioma (Case 4).

Disclosure

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

Author contributions to the study and manuscript preparation include the following. Conception and design: Berhouma, Jouanneau. Acquisition of data: all authors. Analysis and interpretation of data: Berhouma. Drafting the article: Berhouma, Jouanneau. Critically revising the article: all authors. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Berhouma. Administrative/technical/material support: Berhouma. Study supervision: Berhouma, Jouanneau.

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Address correspondence to: Moncef Berhouma, M.D., M.Sc., Skull Base Surgery Unit, Department of Neurosurgery B, Pierre Wertheimer Neurological and Neurosurgical Hospital, Hospices Civils de Lyon, 59 Boulevard Pinel, 69394 Lyon Cedex 03, France. email: berhouma.moncef@gmail.com.