Neurosurg Focus 30 (5):E12, 2011

Meningiomas involving the optic canal: pattern of involvement and implications for surgical technique

Ahmed Nageeb M. Taha, M.D.,[1] Kadir Erkmen, M.D.,[3] Ian F. Dunn, M.D.,[2] Svetlana Pravdenkova, M.D., Ph.D.,[4] and Ossama Al-Mefty, M.D.[2]

[1]Mansoura University Hospital, Mansoura-Dakhlia, Egypt; [2]Department of Neurosurgery, Brigham and Women's Hospital, Boston, Massachusetts; [3]Section of Neurosurgery, Dartmouth Hitchcock Medical Center, Lebanon, New Hampshire; and [4]Arkansas Neuroscience Institute, St. Vincent Infirmary Medical Center, Little Rock, Arkansas

Object. Juxtasellar meningiomas frequently extend into the optic canal. Removing these meningiomas from the optic canal is crucial for favorable visual outcome.

Methods. The authors performed a retrospective analysis of 45 patients with anterior and middle fossa meningiomas with involvement of the optic pathway in whom surgery was performed by the senior author (O.A.M.) during the period from 1993 to 2007. Extent of resection and recurrence rates were determined by pre- and postoperative MR imaging studies. Visual outcomes were evaluated with full ophthalmological examinations performed before and after surgery.

Results. Forty-five patients (31 women and 14 men) were involved in this study; their mean age was 51.6 years. Patients were followed for a mean of 29.8 months (range 6–108 months). No surgery-related death occurred. The average tumor size was 3.1 cm. Total resection of the tumor (Simpson Grade I) was achieved in 32 patients (71.1%). Gross-total resection (Simpson Grades II and III) was achieved in 13 patients (28.9%). Only 1 patient harboring a left cavernous sinus meningioma had tumor recurrence and underwent repeat resection. Meningiomas extended into 58 optic canals in these cases; 13 patients showed extension into both optic canals. Visual disturbance was the main presenting symptom in 37 patients (82.2%); 8 patients had normal vision initially. Visual improvement after surgery was seen in 21 (57%) of 37 patients and in 27 (34.6%) of 78 affected eyes. Vision remained unchanged in 48 (61.5%) of 78 eyes. Transient postoperative visual deterioration occurred in 2 eyes (2.6%), with recovery to baseline over time. Only 1 (1.3%) of 78 eyes had permanent visual deterioration after surgery. The visual outcome was affected mainly by the tumor size, the preoperative visual status, and the duration of symptoms.

Conclusions. Involvement of the optic canal in meningiomas is frequent. It occurs in a wide variety of anterior skull base meningiomas and it can be bilateral. It is a prominent factor that affects the preoperative visual status and postoperative recovery. Decompression of the optic canal and removal of the tumor inside is a crucial step in the surgical management of these tumors to optimize visual recovery and prevent tumor recurrence.
(DOI: 10.3171/2011.2.FOCUS1118)

Key Words • **meningioma** • **surgical technique** • **optic canal** • **optic pathway** • **brain tumor surgery**

Visual disturbance due to optic nerve compression is the initial presentation for many patients with anterior and middle fossa meningiomas. Optic nerve compression is variable depending on the size and the location of the tumor; bilateral optic nerve involvement and optic chiasm compression further add to the complexity of the surgical decision-making process.[2,4,9,19,25,26,28,31] Optic canal involvement by these tumors is not rare, and reports have described unilateral or bilateral optic canal extension.[2–4,32] The primary goal of surgery is total removal of the tumor, with improvement or preservation of the preoperative visual status.[1–3,24,26,28] In this setting, complete nontraumatic decompression of the optic pathway is of paramount concern, with particular attention paid to the full preservation of the attendant delicate blood supply of the optic nerve and chiasm.[2,3,8,21,24,27]

Several reports have addressed the prognostic criteria for visual recovery in patients with these tumors, with series documenting a 25% to 80% chance of visual improvement depending on tumor size, location, extension, preoperative visual status, duration of symptoms, and the surgical technique.[1,2,4,5,7,19,25,26,29,30] Optic canal decompression is an important step to optimize visual recovery and

Abbreviations used in this paper: ACA = anterior cerebral artery; CA = carotid artery; COZ = cranioorbitozygomatic; ICA = internal carotid artery; MCA = middle cerebral artery; OphA = ophthalmic artery; SHA = superior hypophyseal artery.

extent of tumor resection. It can be achieved extradurally, intradurally, or with a combined approach often requiring anterior clinoidectomy.[1,2,4,24,29] Further surgical maneuvers include opening of the bony canal, sectioning the falciform ligament, and opening of the optic nerve sheath. In this study we review 45 patients with meningiomas extending into the optic canal, and we discuss the pattern of involvement of the canal and implications for method of decompression as well as the outcome.

Methods

Between 1993 and 2007, 45 patients (31 women and 14 men) with meningiomas involving the optic canal underwent surgery performed by the senior author. Patients ranged from 30 to 79 years of age (mean 51.6 years). Clinical characteristics of the patients are presented in Table 1. Meningiomas involving the optic canals are summarized in Table 2. Thirteen patients showed bilateral optic canal involvement, and 7 cases were recurrent tumors previously operated on elsewhere. Patients with optic canal decompression with no tumor inside the canal were excluded from the study.

Preoperative imaging studies (MR and CT studies of the brain) were retrospectively analyzed to define tumor size, extent and pattern of optic canal involvement (correlated with operative notes), and the origin and extension of the tumor (Fig. 1). Full ophthalmological assessment was done preoperatively, in the early postoperative evaluation, and through the follow-up examinations. It included visual acuity, visual field testing, ocular motility, and fundoscopy. The WHO criteria to evaluate vision were applied (Table 3).[6] Data regarding the presence of a dissection plane between the tumor and optic pathway, the degree of optic canal involvement, timing of intraoperative optic canal decompression (early extradural or late intradural), and extent of tumor resection were collected from the operative reports. Early postoperative CT and MR imaging were done for all patients to determine the extent of tumor resection. All patients were followed up with serial clinical and MR imaging examinations (mean follow-up 29.8 months, range 6–108 months).

TABLE 1: Clinical characteristics of 45 patients with meningiomas*

Characteristic	Value
sex (no.of pts)	
M	14
F	31
age range	30–79 yrs (mean 51.6 yrs)
follow-up range	6–108 mos (mean 29.8 mos)
no. of pts w/ main presenting Sx	
visual disturbances	37
headache	2
migraine	1
vertigo	1
epilepsy	2
trigeminal neuralgia	1
duration of Sx	1 mo–5 yrs (mean 17.3 mos)

* pts = patients.

TABLE 2: Tumor location and involvement of optic canal

Feature	Value
tumor size	0.5–6 cm (mean 3.1 cm)
origin of meningioma	
clinoidal	11 pts (3 recurrent cases); Type III in 3 pts (2 recurrent cases)
tuberculum sellae	13 pts
planum sphenoidale	5 pts (1 recurrent case)
diaphragma sellae	2 pts
sphenoid wing	7 pts (3 recurrent cases)
cavernous sinus	4 pts
sphenopetroclival	2 pts
en-plaque tumor of anterior skull base	1 pt
optic canal involvement	
unilat meningioma	58 canals
rt side	32 pts
lt side	12 pts
bilat meningiomas	20 pts
tuberculum sellae	13 pts
planum sphenoidale	7 pts
diaphragma sellae	2 pts
clinoidal	2 pts
partial involvement	47 canals
complete involvement	11 canals
optic nerve compression	78 optic nerves
unilat	12 pts
bilat	33 pts

Technical Considerations

The Approach. The COZ approach was used for meningiomas originating lateral to the optic nerve, and was tailored to the size and extension of the tumor. In our experience, this approach facilitates dissection in spaces between the optic nerve and the CA, lateral to the CA in the cavernous sinus, and between both optic nerves, and allows for both extradural and intradural dissection. It was used in 24 patients with meningiomas of the anterior clinoid, sphenoid wing, cavernous sinus, or sphenopetroclival region. We prefer the supraorbital approach for meningiomas located in the midline. The craniotomy in this approach incorporates the orbital rim, the anterior portion of the orbital roof, and the adjacent frontal bone.[18] The supraorbital approach was used in 21 patients harboring meningiomas of the tuberculum sellae, planum sphenoidale, diaphragma sellae, or an en-plaque meningioma.

Optic Canal Unroofing and Clinoidectomy. Anterior clinoidectomy and removal of the bony optic canal was performed either extradurally, intradurally, or as a com-

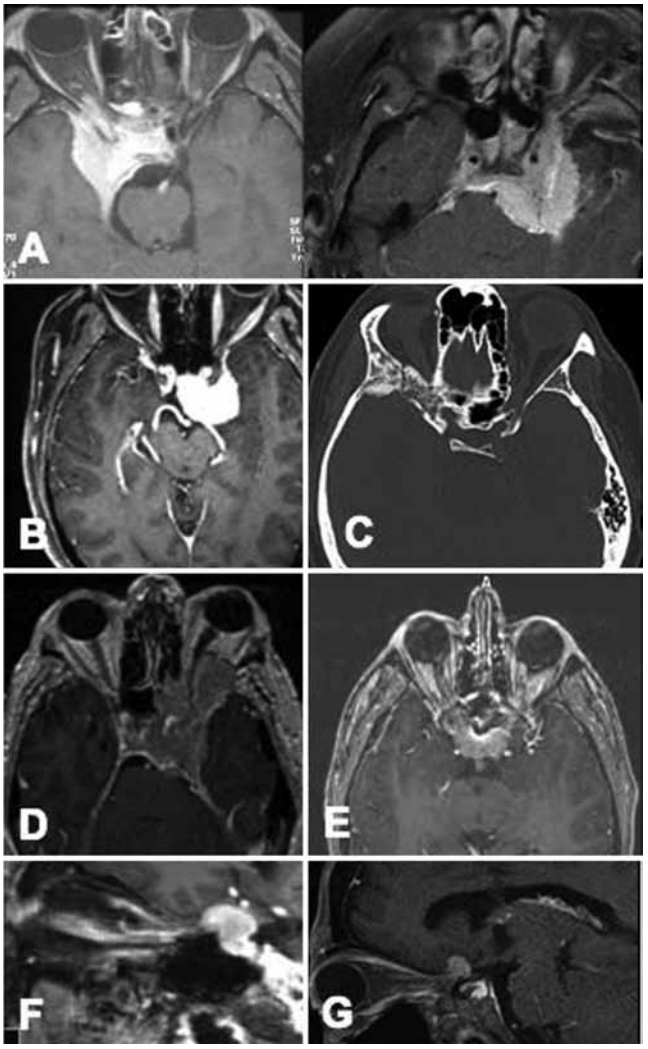

Fig. 1. Preoperative neuroimages showing optic canal involvement by different tumors. **A:** Axial MR images of 2 examples of sphenopetroclival meningioma with involvement of the optic canal. **B:** Axial MR image of a clinoidal meningioma involving the optic canal both medial and lateral to the optic nerve. **C:** Axial CT scan of a right sphenoid wing meningioma with bony involvement of the right optic canal. **D:** Axial MR image of a cavernous sinus meningioma extending into the orbit along the optic canal and superior orbital fissure. **E:** Axial MR image of a tuberculum sellae meningioma with bilateral involvement of the canal. **F:** Oblique MR image demonstrating a tuberculum sellae meningioma with extension into the canal. **G:** Oblique MR image of a recurrent clinoidal meningioma involving the optic canal.

bination of both. After the COZ osteotomy is completed, the microscope is introduced and the remainder of the sphenoid ridge is drilled away, opening the superior orbital fissure. Drilling is continued extradurally to unroof the optic canal and disconnect the optic strut, allowing the anterior clinoid process to be removed extradurally. A diamond drill is used for unroofing of the optic canal, with copious irrigation to prevent thermal injury of the optic nerve. Tumors invading the bone, extending into the cavernous sinus, and or into the orbit can be resected extradurally. The release of a limited amount (30–40 ml) of CSF through a preoperatively placed lumber drain eliminates the need for brain retraction at this stage. Intradural unroofing of the optic canal follows the same principles and is preferred for meningiomas that are located medial to the optic nerve and resected through the supraorbital approach. In some circumstances when there is marked hyperostosis due to bone invasion by meningioma, anterior clinoid removal is started extradurally and completed once the dura mater is opened.

Dissection and Decompression of the Optic Apparatus. The initial steps of intradural dissection include tumor exposure, devascularization, internal debulking, and resection. Brain retraction can be avoided with adequate brain relaxation, which is achieved by partial drainage of CSF through a lumbar catheter or through an opening made in the arachnoid cisterns. Only the small amount of CSF required for brain relaxation should be removed, because the presence of fluid in the arachnoid cisterns facilitates their dissection. Dissection of the tumor from the optic pathway, the CA, the pituitary stalk, and the hypothalamus should follow these arachnoid planes.

The optic nerve may be displaced in several ways. Common patterns of displacement include inferior and medial compression, or elevation by tumor located between the CA and the optic nerve. The nerve may be totally encased by tumor; however, under high microscopic magnification an arachnoid plane can often be established, allowing dissection between the tumor and the optic nerve. In these cases of optic nerve encasement, it is easier to begin dissection from the chiasm and continue toward the optic canal. Removing the tumor inside the optic canal requires drilling the bony canal, opening the falciform ligament, and opening the optic nerve sheath (Fig. 2). Intraorbital extension of the tumor can be removed by following the tumor through the canal into the orbit. Postoperative visual loss generally results from compromise of the vascular supply to the chiasm or the nerve. Particular attention should be directed to preservation of all blood vessels encountered, because the vascular supply to the optic system may course within or around the tumor. The inferior group of vessels arising from the CA is the sole blood supply to the inferior decussating fibers of the chiasm.[8]

Results

Optic Canal Involvement

Tumor extension into the optic canal was seen in 45 patients with anterior and middle fossa meningiomas. In 13 patients there was tumor extension bilaterally, for a total of 58 involved optic canals. Tumor was involving the entire length of the optic canal in 11 sides, and there was direct extension of tumor to the intraorbital compartment in 4.

Twenty sides showed optic nerve compression, but without optic canal involvement, for a total of 78 optic nerves/eyes with tumor compression.

Tumor Location, Approach Selection, and Extent of Resection

The average tumor size was 3.1 cm, with a range of 0.5–6 cm. The origins of the meningiomas involving the canal are summarized in Table 2.

TABLE 3: Criteria for visual changes*

WHO Category	Degree of Vision Changes	Visual Acuity w/ Correction	Definition
normal vision	none	≥20/25	range of normal vision
	slight	<20/25	near-normal vision
low vision	moderate	<20/70	moderate/low vision
	severe	<20/200	severe low vision; FC at ≤6 m
blindness (1 or both eyes)	profound	<20/400	profound low vision or moderate blindness; FC at ≤3 m
	near-total	<3/200	severe or near-total blindness or FC at ≤1 m, or HM at ≤5 m
	total	NLP	total blindness, including absence of eye

* Classifications based on the system of Govsa et al. Abbreviations: FC = finger counting; HM = hand motion; NLP = no light perception.

The COZ approach was used in 24 patients harboring clinoidal, sphenoid wing, cavernous sinus, and sphenopetroclival meningiomas. Extradural anterior clinoidectomy and opening of the bony optic canal was done in 22 patients, whereas in 2 patients the anterior clinoidectomy was performed extradurally and opening of the bony canal was done intradurally. In 2 cases (Type III clinoidal meningioma, cavernous sinus meningioma), tumor within the optic canal was resected extradurally.

The supraorbital approach was used in 21 patients harboring diaphragma sellae, tuberculum sellae, planum sphenoidale, and en-plaque anterior skull base meningiomas, with 2 of these patients undergoing bifrontal craniotomies. In 2 cases of tuberculum sellae meningioma, resection of the part of the tumor inside the optic canal was achieved without drilling the bony canal. A good arachnoid plane of dissection could be achieved in most cases and the tumor could be safely dissected from the optic apparatus, the CA and its branches and perforating vessels (especially those supplying the optic chiasm), the pituitary stalk, and in some instances from the hypothalamus.

Early postoperative CT and MR imaging studies of the brain were used to confirm the extent of tumor resection. Simpson Grade I tumor resections were achieved in 32 patients (71.1%). Gross-total resections (Simpson Grades II and III) were achieved in 13 patients (28.9%). Among 7 patients with recurrent meningiomas, total resection was achieved in 4 cases, whereas gross-total resection was achieved in the other 3 cases. In 3 cases with recurrent meningioma, the cause of recurrence was thought likely to be a result of failure to remove the portion of tumor extending into the optic canal. Postoperatively, there were no recurrences in cases in which radical tumor resection was performed. Tumor recurred in 1 patient with residual tumor in the left cavernous sinus, and reexcision of the tumor was performed 4 years later.

Ophthalmological Presentation and Outcome

Visual disturbances were the main presenting symptom in 37 patients (82%). Tables 4 and 5 summarize the preoperative and the postoperative visual status. Visual outcomes in the 58 optic canals with tumor involvement were variable based on preoperative visual symptoms. Preoperative vision was intact in 3 eyes, and remained the same postoperatively. There was slight visual loss preoperatively in 23 eyes. Of these, 6 improved to normal vision, 14 remained stable, 2 showed transient worsening,

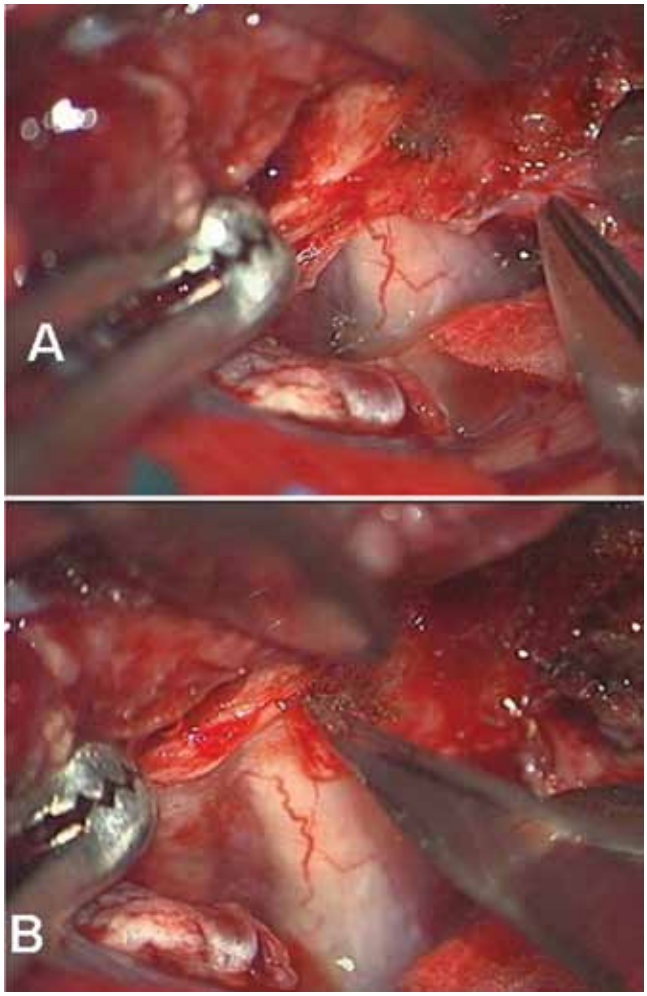

Fig. 2. Intraoperative photographs. **A:** Opening the falciform ligament, showing tumor inside the optic canal. **B:** Opening the falciform ligament and optic nerve sheath to ensure adequate optic nerve decompression.

TABLE 4: Visual outcome in 58 eyes affected with tumor involving the optic canal*

WHO Category	Degree of Vision Impairment on Preop Evaluation	No. of Eyes	Postop Outcome
normal vision	none (≥20/25)	3	3 remained the same
	slight loss (<20/25, near-normal vision)	23	6 improved
			14 remained the same
			1 w/ transient worsening to light perception, then improvement
			1 w/ transient worsening to 20/70, then improvement
			1 w/ permanent worsening to NLP, w/ slight improvement to FC
low vision	moderate loss (<20/70, moderate/low vision)	10	6 improved
			4 remained the same
	severe loss (<20/200, FC at ≤6 m)	7	3 w/ good improvement
			2 w/ mild improvement
			2 remained the same
blindness	profound loss (<20/400, moderate blindness; FC at ≤3 m)	4	1 w/ good improvement
			1 w/ mild improvement
			2 remained stable
	near-total loss (<3/200, near-total blindness; FC at ≤1 m or HM at ≤5 m)	7	3 w/ good improvement
			3 w/ mild improvement
			1 remained the same
	total (NLP, total blindness)	4	1 w/ mild improvement
			3 remained stable

* Optic nerve compression with optic canal involvement (58 canals).

and 1 had rapid deterioration with some recovery. Moderate visual loss was seen preoperatively in 10 eyes, with postoperative improvement in 6 and stable findings in 4. A total of 7 eyes had severe loss preoperatively, with only 3 improving to near-normal vision postoperatively, whereas only 1 of 4 eyes with profound visual loss improved to near-normal vision. Among 11 eyes with near-total and total visual loss, good improvement to near-normal vision was achieved in 3 eyes only. Although some improvement was seen in others, it was not to a functional level.

In one of the cases of transient worsening after resection of tuberculum sellae meningioma, the visual acuity dropped to light perception, then improved gradually to the preoperative visual status over 9 months. In the second case of transient worsening, a right clinoidal meningioma was resected, with postoperative visual acuity worsening to 20/70 (moderate visual loss), which improved to the preoperative status over the course of weeks. In the only case of permanent postoperative visual deterioration, the patient had undergone resection of tuberculum sellae meningioma, with rapid visual loss in his right eye over 36 hours postoperatively to no light perception (total visual loss), which improved slightly to finger counting (near-total visual loss) over the next few weeks.

TABLE 5: Visual outcome in 20 eyes with optic nerve compression without involvement of the optic canal

WHO Category	Preop Evaluation		Postop Outcome
	Vision Impairment	No. of Eyes	
normal vision	none	5	same in 5
	slight	11	same in 11
low vision	moderate	3	1 improved
			2 remained the same
	severe	1	1 remained the same
blindness	profound	0	
	near-total	0	
	total	0	

Twenty sides demonstrating optic nerve compression without direct optic canal involvement had better vision both pre- and postoperatively (Table 5). Of these, 5 sides showed normal preoperative vision and 11 sides showed slight visual loss; all of them remained the same after surgery. Three sides presented with moderate preoperative visual loss, of which 1 improved to normal and 2 remained stable. Only 1 side showed severe preoperative visual loss, which remained stable on postoperative examination. No profound visual loss, near-total loss, or total loss was encountered in any side.

In all cases, there was an overall visual improvement in 21 (57%) of 37 patients who presented with visual disturbance. However, the improvement in 8 eyes was in-

sufficient for functioning visual acuity. Postoperative improvement was related to tumor size and the duration of preoperative symptoms. The tumor size was less than 2.5 cm in 17 of 21 patients who showed postoperative improvement. Patients with postoperative visual improvement had a shorter duration of visual symptoms (median 12 months) when compared with those patients who failed to show improvement (median 21 months). Age was not related to visual outcome in most cases, although the only patient to suffer permanent visual deterioration was 71 years old. Optic canal involvement resulted in worse visual outcomes, although surprisingly, there appeared to be no relationship between the extent of optic canal involvement and visual outcome.

A significant fraction of patients harboring each tumor subtype presented with visual field deficits (Table 6), and surgery led to improvement in at least half of the patients in each group. Patients with clinoidal, sphenoid wing, and cavernous sinus meningiomas presented predominantly with unilateral visual field deficits, whereas patients with diaphragma sellae, tuberculum sellae, and planum sphenoidale lesions in this series often presented with bilateral involvement.

Postoperative Complications

There were no surgery-related deaths. All patients were endocrinologically intact on preoperative assessment. One case of a recurrent sphenoid wing meningioma that had been treated with radiotherapy and Gamma Knife radiosurgery demonstrated panhypopituitarism and complete third nerve palsy postoperatively. Three other patients demonstrated postoperative transient third nerve palsy that recovered over the course of weeks. Two patients developed postoperative deep vein thrombosis that required inferior vena cava filter placement.

Discussion

Optic Canal Involvement in Basal Meningioma

Unilateral or bilateral involvement of the optic canal has been described in many reports of tuberculum sellae meningioma management.[4,9,23–26,28,31] However, optic canal involvement can occur in a wide variety of anterior and middle fossa skull base meningiomas, including those of the planum sphenoidale, diaphragma sellae, clinoid, sphenoid wing, sphenopetroclival, and cavernous sinus regions.[1–3,24] Extension of the tumor inside the canal represents another way in which these tumors may threaten the visual apparatus and worsen the preoperative visual status.[4,24,26,31] In our study, 22 (37.9%) of 58 eyes showed poor preoperative vision due to optic canal involvement. In comparison, 1 (5%) of 20 eyes showed poor preoperative vision due to optic nerve compression without canal involvement. Thus, the overall surgical outcome is guided not only by the extent of tumor resection but also by the extent of optic nerve decompression through unroofing of the optic canal.[4,24–26,28]

TABLE 6: Visual field deficits and their improvement after decompression in tumor subsets

	Presentation w/ Visual Field Deficits	
Tumor Origin	No. of Pts	No. w/ Improvement After Decompression
clinoidal	7 of 11	4 of 7
tuberculum sellae	13 of 13	10 of 13
diaphragma sellae	2 of 2	1 of 2
planum sphenoidale	5 of 5	3 of 5
sphenoid wing	4 of 7	2 of 4
cavernous sinus	3 of 4	3 of 3
sphenopetroclival	1 of 2	1 of 1
en plaque	1 of 1	1 of 1

Optic Canal Decompression and Prevention of Optic Nerve Ischemia

Timing and the manner of optic canal decompression is still a subject of debate.[24–26,28] Many recent reports advocate extradural anterior clinoidectomy and optic canal decompression early in the operation as the best way to achieve a favorable visual outcome, with good surgical results in support.[24–26,28] The claim is that early release of the optic nerve allows its safe manipulation and handling during dissection of the tumor. Also, extradural anterior clinoidectomy has the added advantage of increasing the surgical corridor through the opticocarotid angle.[17,28] This is especially valuable in large tumors with lateral extension to the optic nerve and that involve the cavernous sinus. The additional exposure provides the surgeon with multiple vantage points to the tumor and good visualization of the CA branches. Others have emphasized the need to remove the tumor in the medial aspect of the optic canal to achieve the best long-term visual results, and that this part cannot be addressed by an extradural approach alone.[7,18,19,29]

In the senior author's experience,[1–4] no single method of decompressing the optic canal can be universally applied. First, the location of the tumor in relation to the optic nerve, and hence the pattern of optic canal involvement, play an important role in decision making. Laterally located tumors (53.3% in this series) such as clinoidal, sphenoid wing, cavernous sinus, and sphenopetroclival meningiomas usually compress the optic nerve from a lateral to medial direction, and invade the canal lateral to the nerve. Those tumors are best addressed via a COZ approach with extradural anterior clinoidectomy and decompression of the optic canal. However, anatomical variability[20,22] in such complex areas may alter the approach. In 2 cases in this study complete extradural decompression of the optic canal was difficult, and intradural decompression of the bony canal was achieved after subtotal extradural anterior clinoidectomy. Meningiomas located medial to the optic nerve (46.6% in this series), like those of the tuberculum sellae, planum sphenoidale, and diaphragma sellae, are usually addressed by a unilateral or bifrontal supraorbital approach. They usually extend to the optic canal medial to the optic nerve, with more chance of bilateral canal involvement. Indeed, these tumors accounted for 84.6% of cases with bilateral involvement in this series. Intradural decompression of the optic canal can be used to obtain

good decompression of the medial wall of the bony canal, which cannot be performed extradurally. Also, extradural anterior clinoidectomy has become a routine technique, which can be performed rapidly and safely.[12,14] Generous irrigation during drilling is crucial to prevent thermal injury to these neurovascular structures.[4,19,24,25,31]

Optic canal decompression requires opening of the bony canal, the falciform ligament (which is the most common site of compression injury of the nerve), and, in some cases (Type III clinoidal meningioma), opening of the optic nerve sheath. Achieving all of these steps is necessary for good visual outcome. Extradural optic canal decompression may not suffice as the sole means of optic nerve decompression in some cases, and intradural decompression may be required.[29] The timing of intradural decompression is guided by the tumor size, the relationship between the tumor and the optic nerve, and the feasibility of achieving a plane of dissection between the tumor and the optic nerve. Early dissection of the tumor out of the optic nerve at the canal without establishing a plane of dissection may compromise the blood supply of the nerve and cause ischemic damage to it.[2,4,19,28,33] In most of our cases, it was easier to begin dissection from the chiasm and continue forward toward the optic canal. This allowed easy dissection of the optic nerve by using the identified arachnoid planes, and also early identification and preservation of the blood supply to the optic nerve and chiasm.

The optic pathway has a delicate blood supply. The intradural portion of the optic nerve and the optic chiasm receive their blood supply from perforating vessels coursing from the ICA directly or from the SHA. These perforating vessels arising directly from the CA represent the sole blood supply to the inferior decussating fibers of the chiasm.[8]

The extradural portion and intracanalicular part receive their blood supply from the OphA, either directly or from its small branches. This delicate blood supply is vulnerable to either direct surgical trauma or delayed vasospasm from manipulation. In 1 patient in our study, postoperative visual deterioration 36 hours after surgery may have been a result of a vascular insult to the nerve, possibly from vasospasm related to manipulation around the optic nerve. Establishing a plane of dissection between the tumor and the ICA and its branches (especially the ACA and MCA and their perforators) is essential to achieving successful total tumor resection with minimal morbidity. Encasement of the ICA is commonly seen on preoperative imaging studies; however, it is often not difficult to establish a plane of dissection between the tumor and those vessels at the time of surgery.[2,4,10,19,24,28,33]

Approach Selection is Important to Optimize Optic Canal Decompression

The choice of the approach for removing such lesions depends on many factors, including the origin of the meningioma; size of the tumor; relationship to important structures, including the optic pathway, CA and its branches; extension into the cavernous sinus; and extent of bone involvement.[1–5,19,24,31] Some surgeons advocate the use of a limited pterional approach to address such tumors,[18,21,34] whereas others have suggested that such approaches may require undue brain retraction and result in difficult resection of the tumor, with its dural and osseous involvement.[1–5,24] A modified pterional approach has been shown to be safe and effective for resection of these tumors.[13,15,16] In our experience, skull base approaches, including COZ and supraorbital approaches, can be safely performed without any significant added morbidity. The principal advantages of skull base approaches include the additional exposure obtained while minimizing the need for brain retraction. These approaches provide several corridors to access the tumor and may increase the likelihood of total resection of the tumor with its dural and osseous extensions. Even with tumors totally encasing the optic nerve, total excision is still achievable provided that there is an intact arachnoid plane.[2,4,11,19,24,25,28,31] In this series, total tumor resection (Simpson Grade I) was achieved in 32 patients (71.1%), whereas gross-total resection (Simpson Grades II and III) was achieved in 13 patients (28.9%).

Factors Affecting the Visual Recovery

Optic canal decompression and removal of tumor within the optic canal play prominent roles in both optimizing visual outcomes and preventing tumor recurrence.[24,26,27,31,32] Many authors attribute tumor recurrence in such cases to incomplete resection of the part of the tumor within the optic canal.[5,7,25,26] Residual tumor within the optic canal after an initial operation was probably the cause for tumor regrowth in 3 cases in our series. In addition, in the rare cases of tumor recurrence after optic canal decompression, visual deterioration may be delayed because the optic nerve is already decompressed from the surrounding falciform ligament and optic canal.[24,28] Although many reports conclude that extension of the tumor inside the canal is an indicator of a poor prognosis due to the possibility of nerve ischemia,[24,26] this series demonstrates that adequate optic canal decompression is effective in preserving preoperative visual status in 23 patients (51%), while improving the visual outcome in 21 patients (46.6%). Other factors affecting visual outcome include the age of the patient, tumor size, preoperative visual status, and duration of symptoms.[1,3–5,9,19,24–27,29–31] Large tumors cause more stretching of the adjacent nerves and vessels, and consequently result in more difficult resection. Visual recovery is more favorable in patients with relatively good preoperative vision. Our study showed that the best visual outcome was achieved in patients whose tumors were 2.5 cm or less and with patients presenting with milder visual symptoms.

Although the follow-up period only averaged 29.8 months, there were no recurrences in this series in patients with Simpson Grade I or II resection. Only 1 recurrence occurred 4 years after Simpson Grade III resection of a left cavernous sinus meningioma that necessitated repeat operation. Minimal morbidity due to the operative approaches was observed, despite aggressive resection with extensive bone drilling.[1,3,4,24,26,28]

Conclusions

The unilateral or bilateral extension of juxtasellar meningiomas into the optic canal strongly influences the

preoperative visual status and postoperative recovery. Decompression of the optic canal and removal of tumor extending into the canal are crucial steps in the surgical treatment of these lesions to optimize the patient's visual recovery and to prevent tumor recurrence.

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: all authors. Acquisition of data: all authors. Analysis and interpretation of data: all authors. Drafting the article: all authors. Critically revising the article: all authors. Reviewed final version of the manuscript and approved it for submission: all authors. Statistical analysis: Al-Mefty, Erkmen, Pravdenkova.

References

1. Al-Mefty O: Clinoidal meningiomas. **J Neurosurg 73:**840–849, 1990
2. Al-Mefty O: **Operative Atlas of Meningiomas.** Philadelphia: Lippincott-Raven, 1998
3. Al-Mefty O, Holoubi A, Rifai A, Fox JL: Microsurgical removal of suprasellar meningiomas. **Neurosurgery 16:**364–372, 1985
4. Al-Mefty O, Smith R: Tuberculum sellae meningiomas, in Al-Mefty O (ed): **Meningiomas.** New York: Raven Press, 1991, pp 395–411
5. Andrews BT, Wilson CB: Suprasellar meningiomas: the effect of tumor location on postoperative visual outcome. **J Neurosurg 69:**523–528, 1988
6. Anonymous: The prevention of blindness. Report of a WHO Study Group. **World Health Organ Tech Rep Ser 518:**1–18, 1973
7. Bassiouni H, Asgari S, Stolke D: Tuberculum sellae meningiomas: functional outcome in a consecutive series treated microsurgically. **Surg Neurol 66:**37–45, 2006
8. Bergland R: The arterial supply of the human optic chiasm. **J Neurosurg 31:**327–334, 1969
9. Chi JH, McDermott MW: Tuberculum sellae meningiomas. **Neurosurg Focus 14(6):**e6, 2003
10. Ciric I, Rosenblatt S: Suprasellar meningiomas. **Neurosurgery 49:**1372–1377, 2001
11. Cristante L: Surgical treatment of meningiomas of the orbit and optic canal: a retrospective study with particular attention to the visual outcome. **Acta Neurochir (Wien) 126:**27–32, 1994
12. Day JD, Giannotta SL, Fukushima T: Extradural temporopolar approach to lesions of the upper basilar artery and infrachiasmatic region. **J Neurosurg 81:**230–235, 1994
13. Dolenc VV: **Anatomy and Surgery of the Cavernous Sinus.** Vienna: Springer-Verlag, 1989
14. Dolenc VV: A combined epi- and subdural direct approach to carotid-ophthalmic artery aneurysms. **J Neurosurg 62:**667–672, 1985
15. Dolenc VV: **Microsurgical Anatomy and Surgery of the Central Skull Base.** Vienna: Springer-Verlag, 2003
16. Dolenc VV, Rogers L (eds): **Cavernous Sinus: Developments and Future Perspectives.** Vienna: Springer-Verlag, 2009
17. Evans JJ, Hwang YS, Lee JH: Pre- versus post-anterior clinoidectomy measurements of the optic nerve, internal carotid artery, and opticocarotid triangle: a cadaveric morphometric study. **Neurosurgery 46:**1018–1023, 2000
18. Fahlbusch R, Schott W: Pterional surgery of meningiomas of the tuberculum sellae and planum sphenoidale: surgical results with special consideration of ophthalmological and endocrinological outcomes. **J Neurosurg 96:**235–243, 2002
19. Goel A, Muzumdar D, Desai KI: Tuberculum sellae meningioma: a report on management on the basis of a surgical experience with 70 patients. **Neurosurgery 51:**1358–1364, 2002
20. Govsa F, Erturk M, Kayalioglu G, Pinar Y, Ozer MA, Ozgur T: Neuro-arterial relations in the region of the optic canal. **Surg Radiol Anat 21:**329–335, 1999
21. Hassler W, Eggert HR: Extradural and intradural microsurgical approaches to lesions of the optic canal and the superior orbital fissure. **Acta Neurochir (Wien) 74:**87–93, 1985
22. Huynh-Le P, Natori Y, Sasaki T: Surgical anatomy of the anterior clinoid process. **J Clin Neurosci 11:**283–287, 2004
23. Mahmoud M, Nader R, Al-Mefty O: Optic canal involvement in tuberculum sellae meningiomas: influence on approach, recurrence, and visual recovery. **Neurosurgery 67 (3 Suppl Operative):**ons108–ons119, 2010
24. Margalit NS, Lesser JB, Moche J, Sen C: Meningiomas involving the optic nerve: technical aspects and outcomes for a series of 50 patients. **Neurosurgery 53:**523–533, 2003
25. Mathiesen T, Kihlström L: Visual outcome of tuberculum sellae meningiomas after extradural optic nerve decompression. **Neurosurgery 59:**570–576, 2006
26. Nozaki K, Kikuta K, Takagi Y, Mineharu Y, Takahashi JA, Hashimoto N: Effect of early optic canal unroofing on the outcome of visual functions in surgery for meningiomas of the tuberculum sellae and planum sphenoidale. **Neurosurgery 62:** 839–846, 2008
27. Ohta K, Yasuo K, Morikawa M, Nagashima T, Tamaki N: Treatment of tuberculum sellae meningiomas: a long-term follow-up study. **J Clin Neurosci 8 (Suppl 1):**26–31, 2001
28. Otani N, Muroi C, Yano H, Khan N, Pangalu A, Yonekawa Y: Surgical management of tuberculum sellae meningioma: role of selective extradural anterior clinoidectomy. **Br J Neurosurg 20:**129–138, 2006
29. Puchner MJ, Fischer-Lampsatis RC, Herrmann HD, Freckmann N: Suprasellar meningiomas—neurological and visual outcome at long-term follow-up in a homogeneous series of patients treated microsurgically. **Acta Neurochir (Wien) 140:** 1231–1238, 1998
30. Rosenstein J, Symon L: Surgical management of suprasellar meningioma. Part 2: Prognosis for visual function following craniotomy. **J Neurosurg 61:**642–648, 1984
31. Schick U, Hassler W: Surgical management of tuberculum sellae meningiomas: involvement of the optic canal and visual outcome. **J Neurol Neurosurg Psychiatry 76:**977–983, 2005
32. Shimano H, Nagasawa S, Kawabata S, Ogawa R, Ohta T: Surgical strategy for meningioma extension into the optic canal. **Neurol Med Chir (Tokyo) 40:**447–452, 2000
33. Symon L, Rosenstein J: Surgical management of suprasellar meningioma. Part 1: The influence of tumor size, duration of symptoms, and microsurgery on surgical outcome in 101 consecutive cases. **J Neurosurg 61:**633–641, 1984
34. Yaşargil M: Meningiomas, in Yaşargil M (ed): **Microneurosurgery.** Stuttgart: Georg Thieme, 1996, vol IVB, pp 134–165

Manuscript submitted January 15, 2011.
Accepted February 23, 2011.

Address correspondence to: Ossama Al-Mefty, M.D., Department of Neurosurgery, Brigham and Women's Hospital, PBB-3, 15 Francis Street, Boston, Massachusetts 02115. email: OAlMefty@partners.org.