Visual Outcomes After Treatment of Pituitary Adenomas

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KEYWORDS

• Pituitary adenoma • Neuro-ophthalmology • Visual acuity • Visual field • Prognosis

KEY POINTS

- Patients with pituitary adenomas extending above the diaphragm sella should be assessed for visual acuity abnormalities, visual field defects, and ocular motility disturbances.
- Pituitary apoplexy must be considered in the setting of acute vision loss or ocular motor disturbance, especially if associated with headache, even if there is no previously known pituitary tumor or precipitating factor.
- Studies differ regarding the prognostic factors for visual recovery after apoplexy, but early diagnosis and treatment within 1 week will minimize visual morbidity.
- Not all studies are in agreement as to which factors predict a better visual outcome postoperatively; however, the presence of optic disk pallor with associated loss of the retinal nerve fiber layer (as measured by optical coherence tomography) indicates long-standing damage to the optic pathways, which is less likely to recover.
- The overall rate of visual field improvement with transsphenoidal excision of pituitary adenomas is 80%.
- After pituitary surgery, there may be immediate visual improvement secondary to decompression, then early recovery from restoration of axoplasmic flow, and less dramatic delayed recovery over 1 to 4 months because of remyelination and remodeling.
- Neuro-ophthalmic follow-up is required to monitor for recurrence and for complications of therapy, including chiasmal prolapse and radiation optic neuropathy.
- Patients with poor postoperative visual outcome or diplopia should be referred to appropriate low-vision and ophthalmic services for driving assessment and management.

Pituitary adenomas are benign central nervous system tumors; although many are asymptomatic, others can cause significant morbidity because of their mass effect on local structures. Technical advancements have led to the development of noninvasive or minimally invasive therapeutic modalities, with multiple modalities often used for optimal results. A multidisciplinary team approach is commonly chosen, with the preservation of vision a key indication to proceed with treatment.

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NEURO-OPHTHALMIC ANATOMY OF THE PITUITARY REGION

In 1704, in an attempt to explain the phenomenon of perceived singularity of vision derived from the two eyes, it was hypothesized that the optic chiasm was derived from a merger of both optic nerves with partial cross-over of nerve fibers.[1] Today, an understanding of the visual pathways and the normal variants, from optic nerve to the chiasm and optic tracts, is fundamental in understanding the neuro-ophthalmic manifestations of pituitary tumors.

Afferent Visual Pathways

Optic nerves

The optic canal, located superomedially in the sphenoid bone, transmits the optic nerve from the orbit to the intracranial space. The optic canals are separated from the sphenoid sinus by a bony wall, which is only 0.5 mm thick or less in some patients. Therefore, injury to the lateral wall of the sphenoid sinus, such as during forced opening of a transsphenoidal speculum, can result in vision loss (**Fig. 1**).[2]

The falciform process, a reflected leaf of dura mater, covers the optic nerves as they emerge from the optic canals. The length of nerve covered only by dura can vary from 1 mm to 15 mm.[3] Therefore, during any surgical approach, it should not be assumed that bone separates the dura from the proximal portion of the optic nerve.

When there is extensive anterior growth of the pituitary tumor or if the chiasm is postfixed, optic nerve involvement occurs. The *junctional scotoma* is classically seen when a lesion compresses the junction of the posterior optic nerve and chiasm, where the crossed ventral fibers loop anteriorly (Wilbrand knee). The resultant visual field defect is one of a hemianopic or central scotoma in the ipsilateral eye and a superior temporal defect in the contralateral eye (**Fig. 2**).[4] Damage to the anastomotic blood supply to the posterior portion of the nerve can also occur.

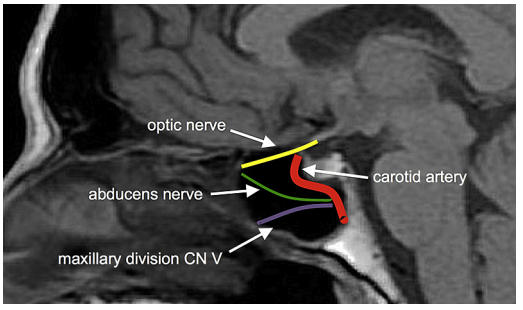

Fig. 1. Sagittal (midline) section magnetic resonance image through the right sphenoid sinus. Note the important neurologic and vascular structures adjacent to the pituitary gland, as indicated by the arrows, and the relative position of each behind the outer wall of the sphenoid sinus.

Chiasm

The chiasm is usually situated over the diaphragm sella, at the junction of the anterior wall and floor of the third ventricle. The chiasm may, however, be prefixed and overlie the tuberculum sella or postfixed and overlie the dorsum sella (**Fig. 3**). A review of 225 autopsy cases found a normal chiasm in 80%, a prefixed chiasm in 9%, and a postfixed chiasm in 11%.[5] These relationships have a direct bearing on the visual field defect resulting from an enlarging pituitary mass.

Approximately 53% of the axons in each optic nerve decussate in the chiasm.[6] The decussating fibers subserve the nasal retina and, therefore, the temporal hemifield; thus, a bitemporal hemianopic visual field defect localizes a lesion to the optic chiasm (**Fig. 4**). The crossing macular fibers occupy the central and posterior portions of the chiasm and make up the bulk of all crossing fibers. If these fibers are involved early, then a bitemporal hemianopic scotoma will be the first visual field defect seen.

The exact pathophysiological mechanism that produces the bitemporal field defect is debated. The effects of a growing pituitary tumor were simulated using a Foley catheter inserted into an empty sella turcica of adult cadavers.[7,8] As the catheter tip was enlarged, the chiasm elevated, causing nonuniform pressure generation across the chiasm with greater effective stress on the crossing fibers. Another group reported that crossing fibers in the chiasm receive their arterial supply solely from the inferior group of vessels,[9] which are vulnerable to compression by a growing pituitary tumor. However, the vascular theory does not explain why compression from above also causes bitemporal hemianopia. A mathematical model has been proposed that shows adjacent crossing nasal fibers are in contact with each other over a smaller area than adjacent uncrossed fibers, therefore, the compressive force generated at these contact points is greater.[10] This force results in architectural distortion of the nerves, compromising nerve fiber conduction. This theory also explains how transmitted shearing forces across the chiasm in patients with head trauma result in bitemporal hemianopia.

Optic tracts

From the chiasm, the optic tracts diverge anteriorly to the interpeduncular space and continue in

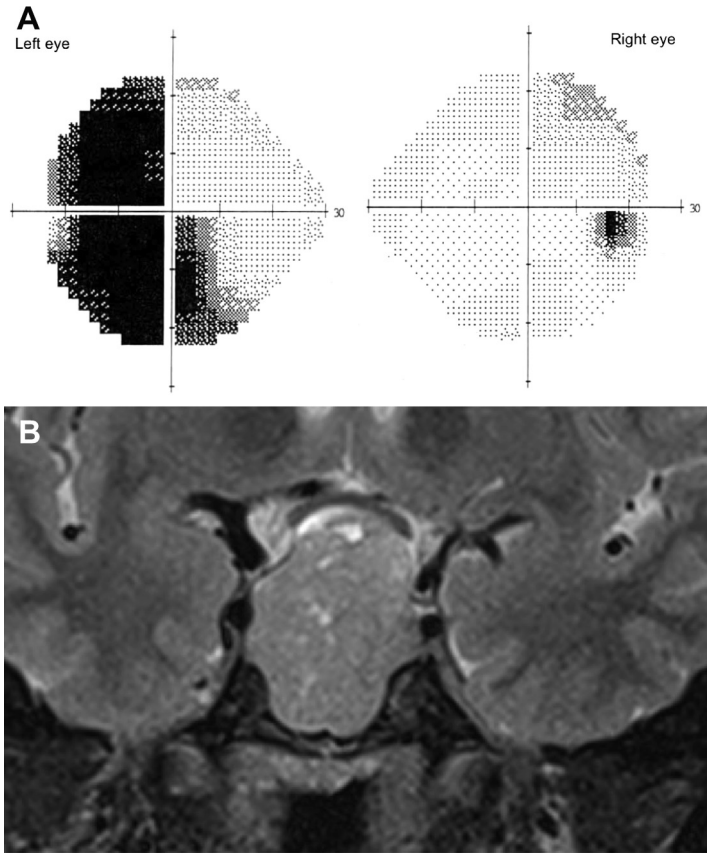

Fig. 2. Junctional scotoma from chiasmal compression. A 54-year-old man presented with visual acuity of 20/20 in the right eye, 20/400 in the left, and a left relative afferent pupillary defect, suggesting a left optic neuropathy. His Humphrey visual fields (*A*) showed a junctional scotoma from distal left optic nerve and chiasmal compression with a dense temporal hemianopic defect (*black shaded area)* in the visual field of the left eye and a small superior temporal defect (*gray shaded area*) in the visual field of the right eye. The coronal magnetic resonance image (*B*) shows chiasmal compression more to the left than the right.

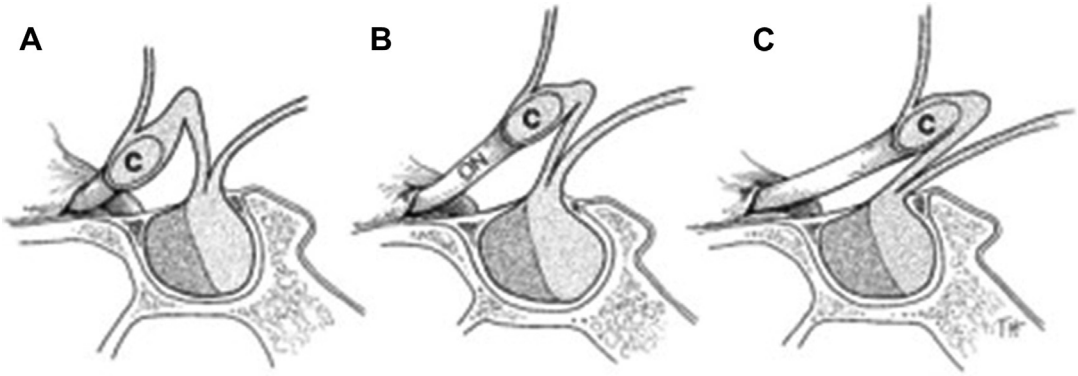

Fig. 3. Three sagittal sections of the optic chiasm and sellar regionshowing the relative positions of the prefixed chiasm (*A*), a normalchiasm (*B*), and a postfixed chiasm (*C*). (*Reproduced from* Rhoton AL Jr, Harrid FS, Renn WH. Microsurgical anatomy of the sellar region and cavernous sinus. In: Glaser JS, editor. Neuroophthalmology symposium of the university of Miami and the Bascon Palmer Eye Institute. St Louis (MO): CV Mosby; 1977. p. 75–105; with permission.)

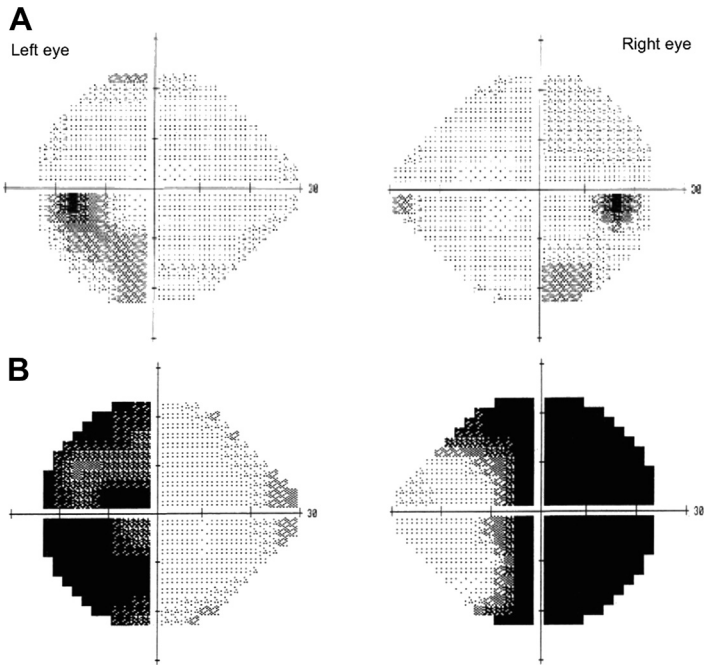

Fig. 4. Change in visual fields following complicated pituitary adenoma surgery. A 35-year-old before and after resection of a large pituitary adenoma. Preoperatively, visual acuity was 20/20 in both eyes with no relative afferent pupillary defect. Visual fields (*A*) showed a mild inferior bitemporal quadrant defect (*gray shading*). Surgery was complicated by hemorrhage in the sella turcica and the patient complained of vision loss in both eyes. Vision postoperatively was reduced to 20/50 in the right and 20/25 in the left, with a right relative afferent pupillary defect. Postoperatively, visual fields showed (*B*) a complete bitemporal hemianopia (*black shading*) with some extension across the vertical midline on the right, suggesting additional right optic nerve involvement.

a posterolateral direction around the cerebral peduncles to enter the middle incisural spaces. The crossed and uncrossed fibers converge, with most of the tract fibers synapsing in the lateral geniculate body. Damage to the optic tracts, as occurs with a prefixed chiasm or a tumor growing posterosuperiorly, will result in an incomplete homonymous visual field defect (see **Fig. 3**).

Efferent Visual Pathways

The cavernous sinuses surround the carotid arteries, lie lateral to the pituitary fossa, and are bound anteriorly and posteriorly by the clinoid processes. A cranial nerve (CN) palsy may occur because of transmission of pressure laterally by the tumor expanding laterally to the wall of the cavernous sinus,[11] compression of the nerve against the interclinoid ligament, direct invasion of the cavernous sinus wall, or surgical trauma (**Figs. 5** and **6**).[12]

Oculomotor nerve: CN III

The oculomotor nerve travels in the lateral wall of the cavernous sinus and enters the superior orbital fissure as 2 divisions. The oculomotor nerve supplies the superior rectus and levator palpebrae muscles in its superior division. The inferior division controls the medial, inferior rectus and inferior oblique muscles and the parasympathetic fibers for pupillomotor control of the iris sphincter and accommodation via the ciliary muscle.

Trochlear nerve: CN IV

The trochlear nerve enters the lateral wall of the cavernous sinus posterosuperiorly, running parallel

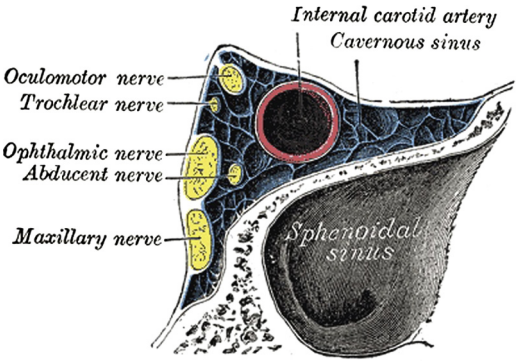

Fig. 5. This anatomic cross section of the cavernous sinus shows the relative positions of the cranial nerves. (*From* Gray H. Anatomy of the human body. Philadelphia: Lea & Febiger, 1918.)

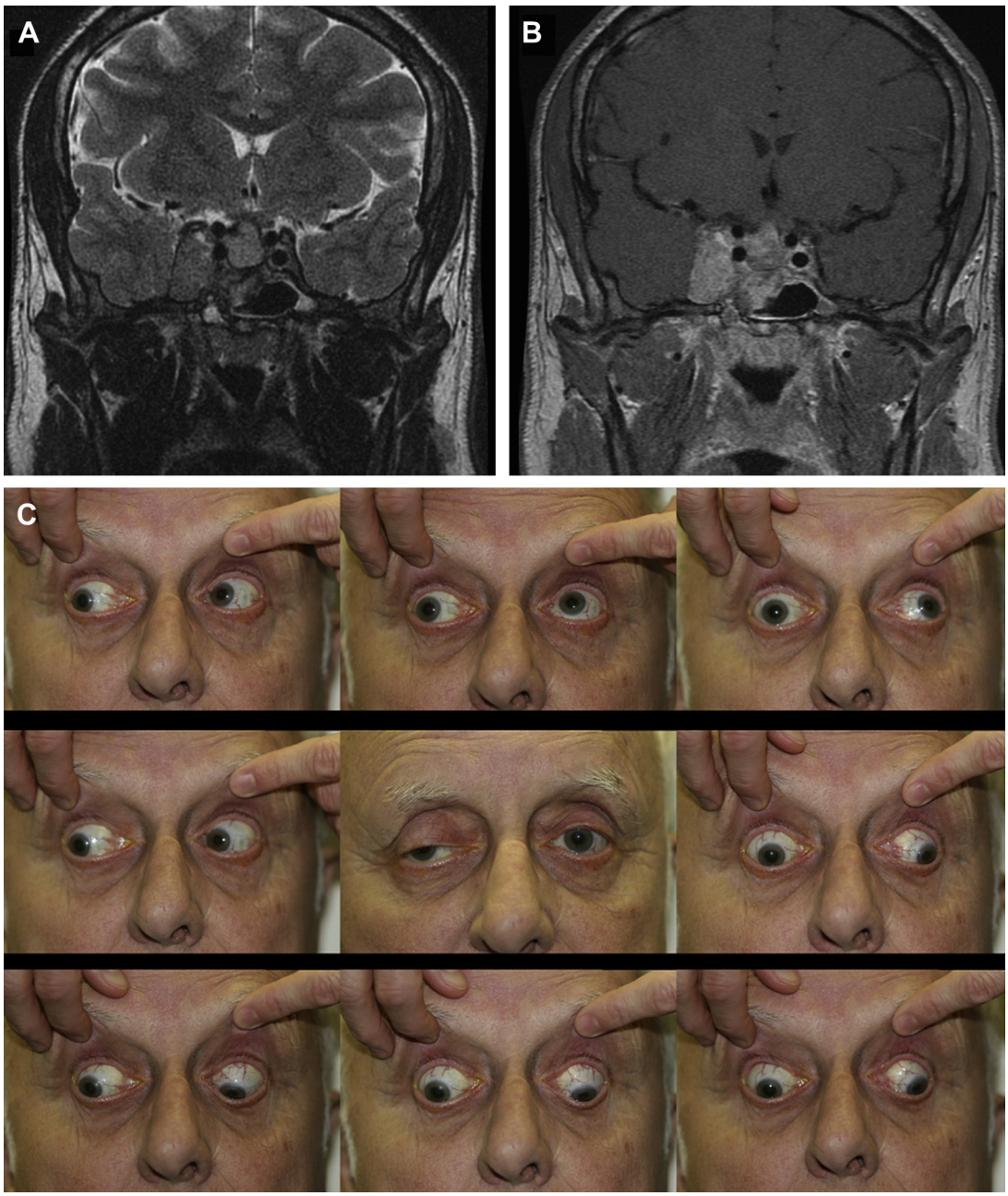

Fig. 6. Right third nerve palsy secondary to a pituitary mass with right cavernous sinus invasion. The coronal magnetic resonance images (*A, B*) show a pituitary mass with extension into the right cavernous sinus and into the right middle cranial fossa despite previous transsphenoidal resection. (*C*) External photography in the 9 cardinal positions of gaze document a partial right oculomotor nerve palsy (third cranial nerve), with partial right ptosis, no elevation or adduction of the right eye, and limited depression of the right eye. The right pupil was poorly reactive to light.

and inferiorly to CN III, then via the superior orbital fissure to control the superior oblique muscle, which is primarily a depressor of the eye in adduction as well as an intorter of the eye.

Abducens nerve: CN VI

The abducens nerve enters the posterior part of the cavernous sinus to run alongside the internal carotid artery, lying freely within the cavernous

sinus rather than within the lateral wall. The sympathetic fibers from the carotid plexus join CN VI before traveling with the branches of CN V. The abducens nerve enters the orbit through the superior orbital fissure and innervates the lateral rectus.

Trigeminal nerve: CN V

The trigeminal nerve passes above the petrous apex to enter Meckel cave, which is located lateral to the cavernous sinus. The ophthalmic division enters the cavernous sinus inferiorly and slopes superiorly within the inferior portion of the lateral wall where the sympathetic fibers join for 1 to 2 cm. This division travels via the superior orbital fissure and carries sensory information from the scalp, forehead, upper eyelid, conjunctiva, cornea, nose, frontal sinuses, and parts of the meninges.

PREOPERATIVE ASSESSMENT

The documentation of preoperative neuro-ophthalmic symptoms and signs allows for appropriate counseling of patients and for an objective prediction of the extent of postoperative recovery.

Presenting Symptoms

Visual loss

The time course and description of visual loss associated with pituitary tumors varies, with steady visual failure being the most common (50%), but rapid (27%) or intermittent progression (12.5%) is also reported.[13] A large monocular visual defect may be noticed suddenly on incidental occlusion of the contralateral eye, when in fact it has been present for many months. In cases of genuine sudden vision loss, pituitary apoplexy must be considered (see later discussion). The classic bitemporal defect results in difficulties of depth perception because the residual visual field from each eye does not overlap. Images in the midline beyond the point of fixation fall on the nasal retina, the blind temporal field, for each eye. This condition is referred to as *postfixational blindness*[14] and may give the impression of objects suddenly appearing centrally when fixation shifts, which is a particular problem when driving.

Diplopia

Diplopia secondary to ocular motor palsy as the presenting symptom is unusual, seen in less than 2% of cases,[15,16] but may be discovered on clinical examination in a larger percentage.[17] Because there are no corresponding retinal points in bitemporal hemianopia to visually link the nasal hemifields, a slight misalignment of the eyes will produce a separation of the visual fields vertically or horizontally,[14] which is often described by patients as double vision. This *hemifield slide* produces an intermittent diplopia in the absence of ocular muscle paresis.

Unusual symptoms

Other neuro-ophthalmic symptoms have been reported in cases of pituitary tumors, including ocular neuromyotonia[18] after radiation treatment (see later discussion). If the tumor invades into the orbit or cavernous sinus, then proptosis or venous stasis may be seen.[19]

Pediatric

Headache and visual failure, although classically regarded as the presenting signs of pituitary lesions, are uncommon childhood presentations of pituitary adenoma.[20] Presenting symptoms in children primarily reflect endocrine dysfunction, with visual field deficits reported in only 5%.[21] Pubertal boys are more likely than girls to present with headaches and visual impairment.[22]

Emergency Presentation

Pituitary apoplexy is a rare but life-threatening condition caused by sudden hemorrhage or infarction of the pituitary gland, occurring in 0.6% to 10.0% of all cases of pituitary adenoma.[23] It is classically characterized by headache, visual loss, ophthalmoplegia, and altered mental status; however, its presentation is highly variable.[24] In a series of cases of pituitary apoplexy, a pituitary adenoma was previously known in only 14% to 20%.[25,26]

The mean age at presentation with pituitary apoplexy is 50 to 56 years. Symptoms include headache (95%–100%), ocular paresis (56%–78%), and reduced visual fields (45%–64%), or acuities (36%–52%).[25,26] When ophthalmoparesis is present, the majority has CN III involvement (57%), followed by CN VI (30%), and then CN IV (13%).[25]

The many potential factors precipitating apoplexy have been debated. Reduced blood flow in the pituitary gland can precipitate apoplexy. Fluctuations in blood pressure and, therefore, blood flow can occur in the setting of cardiac surgery or hemodialysis. Transient elevations of intracranial pressure with resultant pituitary hypoperfusion causing apoplexy have also been reported. More chronic hypoperfusion may occur after radiation therapy. Alternatively, an acute increase in blood flow in the pituitary gland in conditions, such as malignant hypertension or diabetic ketoacidosis, can also trigger apoplexy. Stimulation of the pituitary gland through increased estrogen states, such as pregnancy, or excessive stimulation of the pituitary gland responding to stress at times of surgery, myocardial infarction, or

systemic infections have also been hypothesized as precipitants. Finally, the commencement of anticoagulation was postulated to be a trigger factor in up to 10% of cases in one series.[26] However, in most cases of pituitary apoplexy, there are no identifiable precipitants.

Examination Findings

Visual field defects

Although nearly 75% of patients with pituitary tumors have visual field defects, less than half will complain about vision changes.[16] Therefore, visual field testing should be done in all patients at presentation. The most common types of visual field defects are complete or incomplete bitemporal hemianopia, with the upper temporal quadrant being more frequently and severely affected than the lower temporal quadrant. Other visual field defects seen (in descending frequency) are monocular blindness with temporal defect in the contralateral eye (8.1%), junctional scotoma (5.6%), homonymous hemianopia (4.2%), monocular superior temporal defect (3.3%), and central or temporal scotoma in both eyes (2.7%) or one just eye (1.2%).[16]

Fundoscopic examination

Chiasmal compression can cause the loss of the peripapillary nerve fiber bundle, resulting in optic atrophy.[27] If the decussating fibers are selectively damaged, then band or bow-tie atrophy is seen.[28] This pattern is seen because the decussating fibers originate from the nasal retina and enter the disk both nasally and temporally. However, it takes a minimum of 4 to 8 weeks for optic nerve injury to be reflected in a loss of the retinal nerve fiber layer and optic nerve pallor. Disk pallor is typically maximal at 3 months after injury.[29] Therefore, disk pallor will not be seen early in presentation, even if there is significant optic nerve compromise, unless there was previous longstanding, unrecognized compression.

Papilledema, indicating raised intracranial pressure, is a rare finding with pituitary adenomas.[16] Indeed, other suprasellar lesions, such as craniopharyngioma, are more likely to cause cerebrospinal fluid obstruction and consequent papilledema.

Ocular motility

The prevalence of ocular motor palsy in patients with pituitary adenomas is reported between 1% and 6%.[30] Overall, CN III is the most frequently involved, with the levator palpebrae superioris most commonly affected.[31]

Parasellar lesions, including pituitary tumors, may produce seesaw nystagmus in which elevation and intorsion of one eye is seen with depression and extorsion of the other eye. This phenomenon is not specific to this region or pathologic condition because it also occurs with medullary and pontine lesions affecting the otolithic pathways.[32]

VISUAL PROGNOSIS

Although it is well recognized that visual improvement may occur after surgical excision of a pituitary lesion, this recovery is variable and difficult to predict. A large meta-analysis of transsphenoidal surgery for nonfunctioning pituitary adenomas showed that improvement in visual fields was seen in 78% of cases, with new visual field defects seen postoperatively in 3%.[33] There are several underlying pathophysiological mechanisms that contribute to visual dysfunction that are not associated with significant axonopathy and are, therefore, potentially reversible. Methods of predicting the visual outcomes after surgery would allow for individualized management strategies and more accurate patient counseling regarding their likely visual outcome.

Standard Clinical Assessment

Factors, such as younger age, lack of optic nerve pallor (indicating shorter duration of compression of the anterior visual pathways), and better preoperative visual acuity, have been associated with better postoperative recovery of visual function,[34,35] although not all studies are in agreement. In patients with giant adenomas, postoperative visual outcomes do not seem to be related to preoperative tumor size (<3 cm, 3–4 cm, and >4 cm).[36]

Structural and Functional Measures of the Optic Nerve

Optical coherence tomography

Optical coherence tomography (OCT) allows for the in vivo acquisition of cross-sectional images of the internal microstructure of the retina from which estimates of the neural integrity of the retinal nerve fiber layer (RNFL) can be made. Failure of visual recovery after optic nerve compression is consequent to irreversible damage to the retinal ganglion cell axons. Visual field testing measures defects produced by both dead and damaged retinal ganglion cells, whereas anatomic measurements of the RNFL thinning only reflects axons that have died. Studies of patients with pituitary lesions have shown that a loss of the RNFL thickness at the disk correlates with the severity of visual field loss, occurring diffusely across all sectors.[37] After optic pathway compression is relieved by surgical intervention, the visual field improves for those axons that were damaged but not dead, and the

anatomic measurements reflect the surviving axons. In other words, if there is preservation of RNFL thickness in the setting of profound visual loss, then visual recovery is possible. Patients with RNFL loss at the time of surgery for chiasmal compression are less likely to have a return of visual field or visual acuity after surgery.[38] Therefore, OCT is useful when patients have profound visual loss because measurements showing preservation of the RNFL indicate a better potential for recovery (**Fig. 7**).

Electrodiagnostic tests

Even patients with profound decreases in visual evoked potentials (VEP) may show complete recovery within minutes of surgical decompression.[39] Therefore, the VEP is not reliably prognostic.[40] However, the pattern electroretinogram (PERG) provides a functional measure of the integrity of the retinal ganglion cells in the same way that the OCT gives an anatomic measure of integrity. Studies have shown that the chance of visual field improvement in surgery is greater in eyes with a normal PERG.[41] However, in most centers, OCT is more easily obtained than electrophysiology and can be used more reliably for long-term follow-up.

Apoplexy

In about 30% of patients with apoplexy, there will be an identifiable condition associated with the onset, such as anticoagulation, surgical procedures, or cessation of bromocriptine.[26] However, in one study,[26] those patients with apoplexy with a known predisposing event were 5 times more likely to have neuro-ophthalmic sequelae than those with no associated conditions (severe optic neuropathy in 75% and visual field defect in 100% compared with 14.3% and 20.0%, respectively).

The severity of the initial deficit in both visual fields and acuities seems to be unrelated to the final outcome.[42] However, pallor of the optic disk, implying a prolonged period of optic nerve compression before apoplexy, is an important negative prognostic sign. Ocular motor paresis alone is not an absolute indication for surgery because there can still be full improvement with delayed surgery or conservative management.[25] Studies have shown that early decompression of the optic nerves significantly improves visual recovery,[43] with a significant threshold for a worse prognosis if surgery for pituitary apoplexy is delayed by more than 1 week from presentation.[25]

POST-TREATMENT OUTCOMES

A comparison of studies investigating visual outcomes after the management of pituitary adenomas is hampered by the different visual parameters measured and the lack of consistency regarding what constitutes improvement.

Post-Surgical Treatment

Surgical therapies improve visual field defects in almost 80% of patients.[33] Despite individual studies claiming superiority of each available method of surgical treatment over another, a recent meta-analysis of surgical outcomes for nonfunctioning pituitary adenomas[33] concluded that the transsphenoidal approach is safer than the transcranial approach. Overall, transsphenoidal surgery carries a low risk for damage to the visual apparatus and ocular motor cranial nerves.

Most eyes with visual field loss experience an improvement in visual fields after transsphenoidal surgery regardless of the severity of the preoperative defect. The pattern of recovery of visual function after surgical decompression of pituitary adenomas suggests several phases of improvement. Immediate improvement after decompression, as shown by improvement of VEPs within 10 minutes,[39] is likely caused by removal of physiologic conduction block. The early fast phase, within the first week postoperatively, may lead to normalization of visual fields in some individuals. Visual field improvement occurs usually, but not exclusively, within this first week; however, substantial improvement can still occur weeks and even months after surgery.[34] Even eyes that experience worsening of vision in the immediate postoperative period have the potential for visual improvement with time. The early slow phase (1–4 months) is the period of most notable improvement in visual acuity. It is thought that this improvement over weeks to months is caused by a process of restoration of axoplasmic flow and remyelination.[44] A late phase (6 months to 3 years) of mild improvement is usually not significant overall but may still be substantial in rare individuals.[45]

Improvement in deficits of visual acuity, visual fields, and ocular paresis is common in patients decompressed surgically following apoplexy. Indeed, if vision loss has been present for less than 1 day, then 75% to 97% of eyes have been reported to experience improvement in vision (fields and acuity).[23,42] A delay of 2 to 7 days does not seem to result in a worse visual outcome.[23,42] However, those undergoing surgery more than a week after presentation have a poorer visual outcome. The prognosis for ophthalmoplegia following pituitary apoplexy is relatively good in those undergoing surgery as well as those managed conservatively, with less than 20% of patients having residual diplopia.[26]

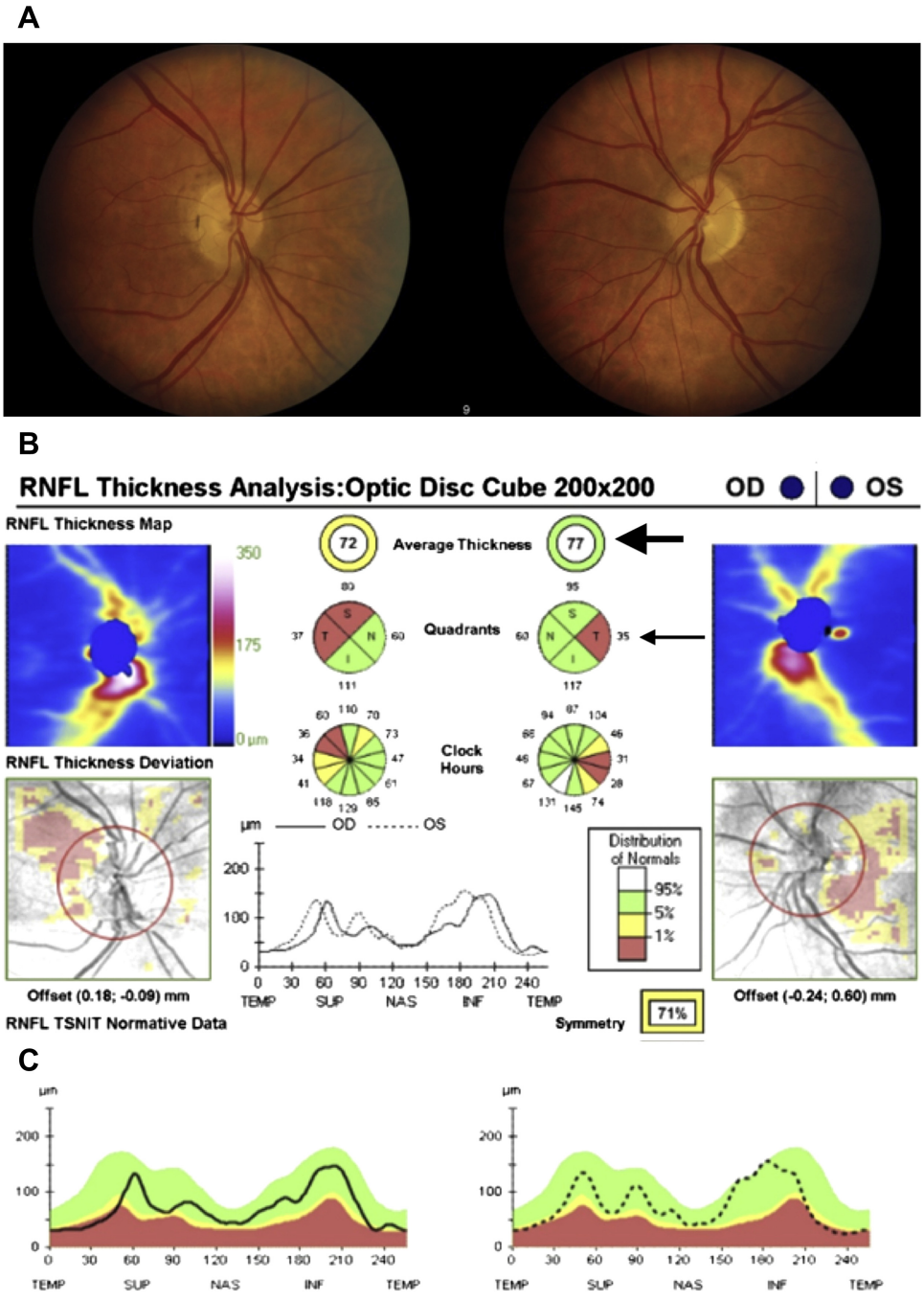

Fig. 7. Optic nerve head photographs and OCT imaging from a patient with a pituitary adenoma compressing the chiasm. (*A*) Fundus photographs of the optic nerve heads show temporal pallor secondary to chronic chiasmal compression. (*B*) RNFL analysis and optic disk scanning obtained with the Cirrus OCT (Carl Zeiss Meditech, California) showing thinning of the RNFL. The normal value for average thickness is 98 μm and 65 μm for the temporal sector. In this patient, the average thickness of the retinal nerve fiber layer is shown as 72 μm in the right and 77 μm in the left (*thick arrow*), suggesting moderate thinning of the RNFL consistent with bilateral chronic optic neuropathies. In addition, the temporal sector (*T*) of the retinal nerve fiber layer (37 μm right and 35 μm left) as shown by the red shading (*thin arrow*) has proportionally greater thinning than the vertical sectors (*S* and *I*). (*C*) The overall cross-sectional result for the patient is drawn as a black line, with the standard population results expected shown in green.

Post-Medical Therapy

There are many reports of visual improvement in patients undergoing medical treatment of hormone-secreting adenomas. Improvement in visual function with bromocriptine therapy is common (90%)[46] and usually occurs within 24 to72 hours of initiation.[47] Bromocriptine also improves ocular dysmotility.[48] When using octreotide, improvement in vision was seen in 75% of patients within hours, with the maximal improvement occurring within 6 to 45 days.[49]

Post-Radiation Therapy

Radiotherapy was historically reserved for patients with residual and recurrent tumors after surgical therapy or those patients medically unfit for surgery.[50] However, following radiation therapy alone, visual field improvement was seen in those patients without optic disk pallor and those younger than 69 years.[51] Post-radiation, patients may have visual complaints with no new objective visual field defect. Many of these patients will have dry eyes,[52] which can cause a transient blurring of vision, highlighting the importance of general ophthalmic assessment.

Worsening of vision does not always correlate with radiologic changes in lesion size. Therefore, it seems that subtle changes in tumor volume not detectable by magnetic resonance imaging (MRI) may be enough to relieve compression and improve patients' symptoms, or exacerbate compression and cause visual defects, again highlighting the need for neuro-ophthalmic assessment rather than relying solely on MRI findings.

COMPLICATIONS OF THERAPY

Aside from the prognostic implications of visual recovery after treatment of a tumor, the prescribed management regimen itself can cause neuro-ophthalmic manifestations.

Surgical Complications

Significant visual loss after transsphenoidal surgery is reported in a minority of patients (0%–11%).[53,54] Various mechanisms can account for the visual loss, including direct injury, vascular compromise, orbital fracture, postoperative hematoma, and chiasmal prolapse. These mechanisms are more frequent in cases of large macroadenoma, a dumbbell-shaped tumor, previous surgery, and previous radiation therapy. Following excision of a pituitary tumor, the suprasellar cistern may extend into the pituitary fossa, with resultant progressive visual impairment if the optic nerves and chiasm prolapse into the empty sella.[55] The associated visual deterioration has been hypothesized to be secondary to vascular injury, scarring, or traction.[56] The surgical repositioning of the chiasm into a more normal anatomical position, known as chiasmapexy, may be required to restore vision.[57] Packing the sella and sphenoid sinus with fat or muscle is used to try to avoid this complication, but packing itself has caused visual compromise from chiasmal compression.[58]

Medical Complications

Neuro-ophthalmic complications of bromocriptine are very rare, although visual hallucinations are reported in 1% to 2% of patients.[59] Chiasmal herniation has also been reported following rapid tumor shrinkage.[60] On cessation of bromocriptine, there is a risk of rapid re-enlargement of the tumor and apoplexy.[61]

Radiation Complications

Complications of radiotherapy result from neurovascular damage, radionecrosis, and disruption of cellular DNA causing secondary tumor genesis.[62]

Radiation optic neuropathy

Radiation optic neuropathy and chiasmopathy are subsets of the more extensive radiation necrosis of the central nervous system, which occurs with an incidence of 0.25% to 25.0% after radiotherapy and an average time delay of 1 to 2 years.[63] The presentation is usually of an acute unilateral loss of vision, although bitemporal hemianopia has also been reported. MRI acutely reveals enlargement and gadolinium enhancement of the optic nerve and chiasm. In one study, the actuarial rate of optic neuropathy at 10 years after a mean radiation dose of 45 Gy was 0.8%.[64] Potential risk factors for radiation optic neuropathy and chiasmopathy include coincident treatment with chemotherapeutic agents, preexisting vasculopathy, carotid atherosclerosis, and diabetes mellitus.[65]

Secondary tumors

Radiation-induced tumors are a late and very rare complication. They may arise within the brain, dura, cranial bones, or spinal and peripheral nerves. Sarcomas are the most commonly reported intracranial tumors presumed to be radiation induced, followed by gliomas and meningiomas, with a latency period ranging from 1 year to more than 30 years.[66]

Neuromyotonia

Ocular neuromyotonia is a paroxysmal misalignment of the eyes, in the directions of the involved ocular motor nerve (most often CN III), usually triggered by eccentric gaze that can last seconds to minutes. The onset varies between 2 months and 18 years after treatment and has been reported

after external beam radiation as well as gamma knife stereotactic radiosurgery.[67] The pathophysiology of neuromyotonia is poorly understood but thought to be caused by alterations in ion channel and neural membrane structures secondary to the radiation effects on the ocular motor nerves.

MANAGEMENT OF POOR POSTOPERATIVE VISUAL FUNCTION

Visual function and diplopia typically improve rapidly after decompression; however, some slow improvement is usually seen up to 6 months to 1 year after treatment. Residual abnormalities may be amenable to symptomatic treatment.

Poor Visual Acuity or Visual Fields

Driving

Although there are strict federal vision standards for commercial driver's licensing, there are no such standards for unrestricted noncommercial passenger vehicle driver's licensing in the United States, and requirements can vary considerably from state to state (listed on the Department of Motor Vehicles website for each state). Restrictions exist based on visual acuity, visual fields, and the presence of double vision. A local ophthalmologist familiar with local requirements should be consulted postoperatively before allowing patients to drive or return to work.

Low-vision services

For patients with poor visual outcomes, services exist to help maximize their visual potential with low-vision aids, home-safety adjustments, and assistance in retraining for work. Low-vision clinics and government services for the visually impaired are essential in the immediate postoperative period.

Double Vision

Patients with persistent double vision postoperatively can be offered monocular occlusion with a simple eye patch or taping of one lens of their glasses to eliminate the second visual image. Although cosmetically this may trouble some patients, it does not delay the recovery process and will allow them to resume normal activities while the ocular motor nerve palsy has an opportunity to resolve. If the strabismus has not resolved and seems stable, then prisms can be fitted into the spectacle correction to compensate for the ocular deviation. After 6 months to 1 year, strabismus surgery can be performed on some patients to realign the eyes and restore binocular vision.

FUTURE DIRECTIONS

Further comparative studies of treatment strategies are needed with adequate bias protection in their methodology.[33] Recent studies have used the Visual Impairment Score, which was originally described as a means to analyze the visual status and surgical outcome of patients with tuberculum sellae and planum sphenoidale meningiomas.[68] Universal use of such a scoring system will allow for better comparison of preoperative and postoperative visual outcomes and among surgical techniques. Such studies would provide stronger evidence to allow patient stratification according to prognostic characteristics, such as tumor size and clinical findings at presentation.

Further ophthalmic studies with respect to structure-function analysis of the retinal nerve fiber layer in relation to visual field defects will also allow for better visual prognostication. Newer OCT machines allow for more selective measurement of the retinal nerve fiber layer distinct from the other retinal layers. Furthermore, objective visual field tests, which avoid much of the bias inherent in repeated standard automated perimetry with its associated learning curve, are becoming more widely available.[69]

Increasing accessibility to 3-T head MRI scans allows for high-resolution anatomic and physiologic images of the optic pathways. Proton MR spectroscopy can provide valuable information on biodistribution of metabolites; if the accuracy improves, it may become possible to accurately distinguish between tumor and scar tissue.[70] Diffusion tensor tractography can depict pathways of the optic nerve fibers and can be combined with treatment-planning images. With pituitary adenomas adjacent to the optic pathways, better surgical and radiotherapy planning is required, and the increased use of these modalities will likely be a topic for future investigation.

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