

PEER REVIEWED

# Letting Go of Cognitive Error When Presumed Glaucoma Isn't Glaucoma

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## **Abstract**

*When a patient presents with the appearance of glaucoma signs, one must be careful not to allow assumption and bias to cloud critical thinking. Cognitive errors lead to diagnostic errors, which may lead to poor outcomes for patients. Etiologies for optic neuropathies range from compressive lesions and ischemic events to hereditary issues and glaucoma. Eyecare professionals must be able to distinguish the characteristics of each of these etiologies. Once a working diagnosis is formed, "what else might this be?" must be asked. This teaching case report highlights a patient who was being treated for glaucoma. However, when bias was set aside, the cause of vision loss was found to be different.*

**Key Words:** cognitive errors, biases, nonglaucomatous optic neuropathy, glaucoma, intracranial lesion

## **Background**

Graber et al. describe three major categories of diagnostic errors in medicine: 1) no-fault errors, 2) system errors, and 3) cognitive errors.<sup>1</sup> No-fault errors occur when a disease lacks the classic textbook presentation and/or mimics something more common. System errors occur due to deficiencies in the healthcare system. These include organizational structure, culture, policies, resources, communication and performance detractors. Cognitive errors occur due to perception. This includes faulty data collection or interpretation, inaccurate clinical reasoning or incomplete knowledge.<sup>1</sup> This case report focuses on cognitive error.

Croskerry has published numerous articles about cognitive errors, or cognitive dispositions to respond (CDRs). These CDRs are associated with failures in perception, failed heuristics and biases. A partial list of CDRs may be found in Croskerry's article "The Importance of Cognitive Errors in Diagnosis and Strategies to Minimize Them." Recognizing CDRs is key in reducing them. Metacognition is a reflective approach in which one steps back from the "immediate problem to examine and reflect on the thinking process."<sup>2</sup>

Eyecare professionals rely on optic nerve appearance, intraocular pressure (IOP), retinal nerve fiber layer (RNFL) quality, visual fields and gonioscopy when monitoring the progression of glaucomatous eye disease. Other disorders of the optic nerve may mimic glaucoma. If misdiagnosed as glaucoma, these disorders can have severe consequences for the patient, both visually and possibly systemically.

Nonglaucomatous disorders that may mimic glaucomatous optic neuropathy include hereditary optic neuropathies, optic nerve infarctions, trauma, infection, demyelinating optic neuritis, fusiform enlargement of the intracranial carotid artery, and intraorbital and intracranial mass lesions.<sup>3</sup>

Patients with compressive injury may exhibit visual field defects that resemble those of a glaucoma patient. However, when an optic nerve appears to have more pallor than cupping, particularly of the

temporal rim, it should prompt third- and fourth-year optometry student clinicians, optometry residents and practicing optometrists to forgo their biases and explore etiologies other than glaucoma.

## Case Description

On April 22, 2015, a 65-year-old Vietnamese female presented with a chief complaint of longstanding blurry vision in her left eye and no vision through her right eye. She had been using bimatoprost 0.03% ophthalmic solution, which had been prescribed in Vietnam, in both eyes at bedtime “for many years.” This was her first eye examination in the United States. No previous ophthalmic records were provided by the patient, and she was a poor historian. She reported being supplied with the ophthalmic medication from family and friends returning from Vietnam. (In many countries, a prescription is not necessary to purchase medication.) A Vietnamese interpreter was used during the examination via telephone.

The patient’s medical history was significant for diabetes mellitus, hypertension and hyperlipidemia. Her medications included metformin 1,000 mg, fenofibrate nanocrystallized 145 mg, glipizide 10 mg, lisinopril 20 mg, metoprolol tartate 25 mg and atorvastatin 20 mg.

Her pertinent laboratory tests as of Feb. 17, 2015, were hemoglobin A1c 8.0% and total cholesterol 207 mg/dL with 62 mg/dL high-density lipoprotein. Her blood pressure on March 2, 2015, was 130/60 and her body mass index was 25.71 kg/m<sup>2</sup>. The patient was oriented to person, place and time, and her mood and affect were appropriate.

On examination, the patient’s unaided visual acuities were no light perception (NLP) in the right eye and 20/320 in the left eye improving with pinhole to 20/80. Pinhole testing of the right eye with NLP was deemed unnecessary as this was the initial eye examination. Pupils were equally round and reactive to light with a relative afferent pupillary defect in the right eye. Confrontation visual fields were not possible for the right eye due to NLP vision and full to finger count in the left eye. Ocular motilities were full range of motion with no restriction in either eye. Ishihara color vision test results were 0/8 plates with the right eye and 8/8 plates with the left eye. Refraction revealed no improvement in the right eye with any lens change and +3.00 +1.25 x 030 in the left eye resulting in 20/70 distance visual acuity.

Slit lamp biomicroscopy examination was remarkable for patent peripheral iridotomy and nuclear sclerotic cataract in both eyes. IOP measured 14 mmHg in both eyes via Goldmann applanation tonometry. Corneal pachymetry measurements were 540 µm in the right eye and 535 µm in the left eye. Sussman four-mirror gonioscopy of both eyes revealed posterior trabecular meshwork in all four quadrants with 1-2+ pigment and no peripheral anterior synechia or neovascularization of the angle.

Dilated fundus examination was remarkable for pallor of the right optic nerve, with cup-to-disc ratio of 0.1 and distinct disc margins. The left optic nerve showed no pallor, distinct margins and cup-to-disc ratio of 0.15 (**Figure 1**). There was an epiretinal membrane over the macula of the right eye. The left eye was unremarkable.

Magnetic resonance imaging (MRI) of the brain and orbits was ordered. Bimatoprost was discontinued, and brimonidine 0.15% ophthalmic solution 3 times daily in both eyes was prescribed for its neuroprotective potential. The patient was counseled to keep her blood sugar, blood pressure and cholesterol under control. The epiretinal membrane was not visually significant. The cataract in the left eye was visually significant; however, the patient elected to defer cataract surgery. A spectacle prescription was dispensed.

MRI of brain and orbit (**Figure 2**) was performed on May 11, 2015, and revealed a right medial sphenoid wing meningioma described by the radiologist as an “extensive, invasive extra-axial mass centered in the region of the right cavernous sinus/Meckel’s cave with involvement of the right orbit, encasement of the right optic nerve and right ICA.” Although exophthalmos was not clinically evident, proptosis of the

right eye was noted on MRI. The amount of proptosis was measured radiologically via exophthalmos index (EI). On scans, globe protrusion is defined as greater than 21 mm from the interzygomatic line (IZL) to the anterior surface of the globe or less than 9.9 mm from the IZL to the posterior sclera margin.<sup>4</sup> The ratio between this measurement in the affected eye vs. the normal eye is defined as the EI, and any number greater than 1.0 indicates proptosis.<sup>5</sup> The EI for this patient was 1.17.

A consultation with neurosurgery on June 5, 2015, confirmed the diagnosis of meningioma. Follow-up appointments with neurosurgery on June 26, 2015, and Nov. 20, 2015, revealed no changes in the patient's status. Therefore, it was determined that the patient would need only surgical decompression if the left visual acuity were to become compromised. The patient returned to the eye clinic for Humphrey visual field testing of the left eye, and optical coherence tomography (OCT) of the RNFL was performed on both eyes. Results are shown in **Figures 3 and 4**. Visual acuity and IOP at this visit remained stable. Five months later, on Oct. 14, 2015, the patient had a follow-up MRI of her brain, which showed no interval changes.



**Figure 1.** The patient's IRIS retinal camera optic nerve photographs. It is difficult to appreciate the pallor of the right optic nerve (top); however, note the lack of significant cupping. The left fundus (bottom) was unremarkable. [Click to enlarge](#)



**Figure 2.** MRI showing large homogeneous enhancing mass centered in the right cavernous sinus/Meckel's cave extending anteriorly through the superior orbital fissure into the posterior right orbit. The yellow horizontal arrow is the interzygomatic line. Exophthalmos index (EI) is defined as a (mm)/b (mm). In this example,  $EI = 21 \text{ mm}/18 \text{ mm} = 1.17$ . [Click to enlarge](#)



**Figure 3.** Humphrey visual field 30-2 of the left eye showing inferior edge defects with no optic neuropathy noted. [Click to enlarge](#)



**Figure 4.** OCT of the right and left eye retinal nerve fiber layers. Note the significant atrophy of the right optic nerve. [Click to enlarge](#)

## Education Guidelines

### Key concepts

1. Recognize the clinical signs of compressive nonglaucomatous optic neuropathy vs. glaucomatous optic neuropathy
2. Recognize visual field defects in compressive nonglaucomatous optic neuropathy vs. glaucomatous optic neuropathy
3. Recognize ancillary tests that aid in the diagnosis of compressive nonglaucomatous optic neuropathy and the importance of reviewing the medical record and history
4. Understand co-managing patients with compressive nonglaucomatous optic neuropathy, including treatment and follow-up
5. Understand CDRs and the need to investigate further if the clinical picture does not match a diagnosis (diagnosis momentum)

### Learning objectives

At the conclusion of this case discussion, participants should be able to:

1. Describe the typical presentation of compressive nonglaucomatous optic neuropathy vs.

glaucomatous optic neuropathy

2. Understand the clinical findings associated with compressive nonglaucomatous optic nerve damage and differential diagnoses
3. Understand how to use ancillary testing to aid in diagnosis and prognosis of compressive nonglaucomatous optic neuropathy and potential complications
4. Describe treatment and team management of compressive nonglaucomatous optic neuropathy
5. Acknowledge and set aside cognitive biases to minimize diagnostic errors

### *Discussion questions*

1. Does compressive nonglaucomatous optic neuropathy affect vision?
2. Does compressive nonglaucomatous optic neuropathy affect pupil response?
3. What are other clinical signs of compressive nonglaucomatous optic neuropathy?
4. What differential diagnoses should be considered in cases of suspected compressive nonglaucomatous optic neuropathy?
5. What ancillary testing can aid in the diagnosis of compressive nonglaucomatous optic neuropathy?
6. What is the appropriate management for compressive nonglaucomatous optic neuropathy?
7. What is the prognosis for compressive nonglaucomatous optic neuropathy?
8. What role did cognitive error play in this case?

### *Learning assessment*

1. Facilitate case discussion to achieve learning objectives
2. Knowledge base can be evaluated by comparing fundus photographs and visual fields of glaucomatous vs. nonglaucomatous optic neuropathy
3. Knowledge base of the condition and clinical-thinking skills can be assessed via student presentations of differential diagnoses and ancillary tests to rule out those diagnoses

## **Discussion**

### *Teaching instructions*

Clinicians and students using this teaching case report should read each discussion question and think about how they would respond and compare and contrast with the information provided below. Participants may work together in small groups or individually. Learning objectives are to be assessed by comparing participants' responses with the information provided. This case may also be presented as a PowerPoint lecture comparing and contrasting fundus photographs and visual fields in nonglaucomatous optic neuropathies with glaucomatous optic neuropathy. It may also be presented as a case discussion in a journal club setting.

### *Does compressive nonglaucomatous optic neuropathy affect vision?*

Visual acuity can be normal or impaired depending on whether the central visual field is affected.<sup>6</sup> Best-corrected visual acuity is typically 20/40 or worse in patients with suspected compressive lesions. Vision loss is gradual, not sudden. This compares with best-corrected visual acuity of 20/20, which is typical for glaucoma patients until late- to end-stage glaucoma. Be wary of asymmetric decreased visual acuity. In addition, it is critical to investigate the cause of vision loss, even NLP vision, as a patient's ocular and systemic health may be at risk as is shown in this case.

### *Does compressive nonglaucomatous optic neuropathy affect pupil response?*

A relative afferent pupillary defect (RAPD) can be detected by performing the swinging light pupil test. In the presence of bilateral symmetric optic neuropathy or early stage compressive lesion not affecting the

optic nerve, RAPD may be absent. The briskness of pupillary constriction to light will reflect the degree of optic nerve dysfunction. When checking for RAPD, it is important to use a very bright light in a dark room to assess the full amplitude of pupillary response.<sup>6</sup>

*What are other clinical signs of compressive nonglaucomatous optic neuropathy?*



Table 1. [Click to enlarge](#)

The ocular manifestations of sphenoid wing meningiomas vary depending on the size and location of the primary tumor. The classic triad of signs is proptosis, decreased vision (Snellen visual acuity worse than 20/40) and impaired extraocular motility. Other signs may be temporal fullness and globe displacement, which can present in orbital or temporal fossa masses. Tumors near the sella or optic nerve can cause visual field defects (that border the vertical midline) and optic disc edema or atrophy (without cupping of the disc). Ptosis, eyelid edema, chemosis, diplopia and loss of facial sensation may also present.<sup>7</sup>

Compare and contrast these signs with the clinical signs of glaucomatous optic neuropathy, which are better Snellen visual acuity, visual field defects that border the horizontal midline, older age of patient at presentation, vertical elongation of the optic cup, peripapillary atrophy and, occasionally, splinter disc hemorrhages. **Table 1** is a quick reference guide for signs and symptoms of compressive nonglaucomatous vs. glaucomatous optic neuropathy.<sup>8</sup>

*What differential diagnoses should be considered in cases of suspected compressive nonglaucomatous optic neuropathy?*

Differential diagnosis of optic neuropathy includes any etiology that may cause damage to the optic nerve. A pale optic disc indicates a longstanding complication such as compressive, hereditary or toxic/nutritional neuropathies. Optic nerve pallor may also indicate sequelae of an acute inflammatory or ischemic optic neuropathy. Sectoral pallor with retinal attenuation should alert the clinician to previous nonarteritic ischemic optic neuropathy (NAION).<sup>6</sup> A careful review of history (rapid vs. gradual onset, associated symptoms, medical conditions, age/gender predilection) and thorough ophthalmic evaluation narrows potential diagnoses.

This is a unique case in that the patient, as a female of Asian descent, may have had narrow angles on gonioscopy at her initial eye examination in Vietnam and deemed at risk for an angle closure attack necessitating laser peripheral iridotomy (LPI). Literature review shows that close follow-up and further medications and/or laser or surgery post LPI is necessary to prevent progression of the disease in Vietnamese patients, which would validate the anti-glaucoma medication this patient had been taking.<sup>9</sup> In a prospective observational case series, Shen et al. reported on changes in optic disc morphology (increased cup-to-disc ratio and decreased mean neuroretinal rim area) from week 2 to week 16 post LPI in subjects with acute primary angle closure.<sup>10</sup> This patient's previous eye exams and procedures were performed in Vietnam; therefore, requesting records was impossible. Within the range of differentials for this patient, any diagnosis along the primary angle closure spectrum could be plausible except for the lack of cupping of the optic nerve in either eye. This case shows that progressive, painless, unilateral vision loss with optic atrophy in the absence of cupping is not glaucoma. In a retrospective study, Trobe et al. reported that pallor of the neuroretinal rim is 94% specific in predicting nonglaucomatous optic neuropathy.<sup>11</sup>

Theoretically, one may also consider functional vision loss, or nonorganic vision loss (NOVL), when a new patient presents with NLP vision. NOVL is described as decreased visual acuity and/or visual field loss not caused by an organic lesion/pathology and diagnosed after a complete neuro-ophthalmic examination. It can run the spectrum from malingering to subconscious vision loss secondary to

underlying psychological disorder, typically depression or anxiety.<sup>12</sup> A few in-office techniques can help to differentiate NOVL from pathology. On confrontation or tangent screen visual field testing, remember that normal visual field is a funnel, not a tunnel. A patient's constricted visual field should expand when the test moves from 1 meter to 2 meters away. If the constricted visual field remains the same at 1 meter and 2 meters, that is tunnel vision and consistent with NOVL. Typical automated visual field presentation is a cloverleaf or square pattern in NOVL. Pupil testing will be unremarkable in a patient presenting with NOVL vs. a positive RAPD in patients with true pathology. Upon color vision testing with pseudoisochromatic plates, a patient presenting with NOVL will often report not seeing the test plate. Contrast sensitivity testing in NOVL is variable and inconsistent, which supports the diagnosis of NOVL.<sup>13</sup> Simple tests of proprioception can also be used to rule out NOVL. For example, a patient with true, organic vision loss can bring their index fingertips together in front of them whereas a patient with NOVL will be unable to do so, thinking the exercise is vision-dependent.<sup>14</sup>

### *What ancillary testing can aid in the diagnosis of compressive nonglaucomatous optic neuropathy?*

Visual field defects that respect the vertical meridian on formal testing should alert the clinician of possible compression or infiltration.<sup>15</sup> Visual field testing also aids in localization of the lesion. A hemianopic defect indicates a lesion at or posterior to the chiasm. A junctional scotoma, an ipsilateral central field defect and contralateral superotemporal field defect, indicates a compressive lesion at the junction of the optic nerve and the chiasm.<sup>6</sup>

Color vision testing via pseudoisochromatic test plates such as Ishihara color plates or American Optical Hardy-Rand-Rittler (AOHRR) color plates can indicate optic nerve damage. Most patients with acquired optic neuropathy will have some degree of dyschromatopsia. The AOHRR test has an advantage over the Ishihara test with acquired color deficiencies, in particular with optic neuropathies, with its inclusion of plates designed to test for S-cone mechanism defects. Glaucoma first causes an acquired S-mechanism deficiency and later, in advanced disease, it causes an acquired M-L mechanism deficiency.<sup>16</sup> Red cap desaturation is another quick clinical tool to assess optic neuropathy. A positive response would be that the red cap color appears "faded" or "pink" in the eye with optic nerve damage vs. "red" in the eye with no optic nerve damage.<sup>6</sup>

OCT uses light to penetrate tissue and analyzes the reflected image of the optic nerve head and the peripapillary RNFL. It can also play a role in predicting recovery in patients with severe compressive neuropathy. Loo et al. found that patients with compressive optic neuropathy due to anterior pathway meningiomas are more likely to improve after treatment if they have a normal pretreatment peripapillary RNFL and shorter duration of symptoms.<sup>17</sup>

Exophthalmometry should be performed when patients present with proptosis. Approximately 44% of patients with compressive optic neuropathy have proptosis less than 4 mm, and roughly 35% have proptosis greater than 4 mm. People of Asian decent typically have Hertel exophthalmometry measurements between 16 and 18 mm; people of African decent typically have measurements between 20 and 22 mm; and people of Caucasian decent typically have measurements between 18 and 20 mm. The difference between a patient's two eyes usually does not exceed 2 mm.<sup>18</sup> As referenced above, the amount of proptosis may also be measured radiologically, utilizing either computed tomography or MRI scans. Symmetry between ocular globes correlates to an EI of 1.0; any number greater than 1.0 indicates some degree of proptosis.<sup>5</sup>

Contrast sensitivity is usually reduced in patients with optic neuropathy and may be tested using the Pelli-Robson chart. Contrast sensitivity reduction may be detected prior to Snellen visual acuity reduction.<sup>6</sup>

MRI of the brain and orbit is essential in diagnosing compressive lesions. The two most common

intracranial mass lesions that cause optic nerve pallor are pituitary lesions at 57.1% and meningiomas at 21.4%.<sup>3</sup> Pituitary adenomas have the classic bitemporal hemianopsia seen on formal visual field testing. Meningiomas are the most common primary tumor of the central nervous system, accounting for approximately 33.8% of all brain tumors.<sup>19</sup> They occur more commonly in older, female populations. Sphenoid wing meningiomas constitute 11-20% of intracranial meningiomas and those with secondary orbital extension are rare.<sup>20,21</sup> These tumors are compressive and slow-growing at a rate of 1 to 3 mm per year.<sup>22</sup> Sphenoid wing meningiomas can involve the region of the anterior clinoid process, adjacent medial sphenoid wing, superior orbital fissure and cavernous sinus. They are classified as either globoid tumors with a nodular shape or an en plaque tumor, which is flat and spreads along the entire sphenoid ridge. The globoid tumors are further broken down into three groups depending on their location: inner (medial), middle and lateral. Medial sphenoid wing meningiomas have a higher morbidity, mortality and recurrence rate due to their involvement with anterior visual pathways, anterior intracranial arteries and the cavernous sinus.<sup>23</sup> As the tumor becomes larger, it may encase the internal carotid and proximal middle and anterior cerebral arteries. The optic nerve may also be compressed if it is surrounded by the tumor resulting in vision loss and visual field defects. Neurologic deficits or seizures may present in patients with large tumors that put pressure on the frontal and temporal lobes and provoke edema in the adjacent brain tissue.<sup>24</sup>

#### *What is the appropriate management for compressive nonglaucomatous optic neuropathy?*

The treatment avenues for meningiomas involve observation (as in this patient's case), surgery, radiation and chemotherapy. General guidelines for the treatment of meningioma indicate that it may be appropriate to follow patients with mild or nonprogressive symptoms with periodic MRI and examination to determine whether the lesion is growing and if symptoms are significantly interfering with the patient's life. Surgical intervention is often preferred for younger patients with worsening symptoms and/or growth seen on follow-up scans. The indications for surgery in older patients are a large tumor with worsening symptoms. Radiation therapy is used in older patients with small and medium size tumors with worsening symptoms and for treatment of regrowth after subtotal or radical subtotal removal.<sup>24</sup> Chemotherapy is used as a treatment option in meningiomas that are progressive, recurrent or inoperable. Typically, it does not play a significant role in management of meningiomas due to significant systemic toxicity encountered with chemotherapy and modest to no tumor regression detected.<sup>22</sup>

A critical factor for aggressive tumors is angiogenesis, which is mediated by vascular endothelial growth factor (VEGF). Therefore, bevacizumab, a humanized monoclonal antibody against VEGF-A, has been used in the treatment of recurrent or progressive meningiomas resistant to standard therapy.<sup>19,25</sup>

Tumor recurrence is the major risk in the long run; thus, in most initial surgeries, aggressive resection is advocated. The size of the tumor affects the extent of tumor removal and determines clinical outcomes including visual acuity recovery.<sup>26</sup>

Meningiomas often invade the optic canal. In addition to tumor excision, optic canal decompression optimizes visual outcomes as well as lowers the chances of tumor recurrence. Early decompression of the optic nerve within the bony canal allows identification and separation of the tumor from the nerve, permitting removal of the tumor from this area with minimal manipulation of the optic nerve, thus providing better visual prognosis.<sup>27,28</sup>

#### *What is the prognosis for compressive nonglaucomatous optic neuropathy?*

Overall, the potential for malignancy is low for sphenoid wing meningiomas. Cornelius et al. found in a large retrospective study of patients who were operated on for meningioma, 90% of the tumors were benign.<sup>29</sup> Recurrence rate of meningioma after treatment with surgery, radiation or combined therapy is approximately 10%. Morbidity from the tumor and therapeutic interventions depends on the location of

the tumor and proximity to vital neurological and ocular structures.<sup>30</sup>

In general, visual prognosis has been shown to be dependent on preoperative visual acuity and extent of tumor surgical resection. Trends show that early surgical intervention may benefit patients before visual decline starts. Studies also support radical resection on the first surgical encounter to minimize residual tumor, propensity for recurrence and need for repeat surgery, all of which have a negative visual outcome.<sup>31</sup>

*What role did cognitive error play in this case?*

Types of CDRs that played a role in perpetuating the glaucoma diagnosis in this patient were anchoring, confirmation bias, diagnosis momentum, premature closure and search satisfying. Anchoring is the “tendency to lock onto salient features in the patient’s initial presentation too early in the diagnostic process, and failing to adjust this initial impression in the light of later information.” Confirmation bias is the “tendency to look for confirming evidence to support a diagnosis rather than look for disconfirming evidence to refute it, despite the latter often being more persuasive and definitive.” Diagnosis momentum refers to a diagnostic label that gets “stickier and stickier” through continued use by patients, nurses, physicians, paramedics. It becomes so sticky that what was once a possible diagnosis becomes definite and all other possibilities are excluded. Premature closure is similar to diagnosis momentum in that it accepts a diagnosis prior to being fully verified; “when the diagnosis is made, the thinking stops.” Search satisfying is akin to diagnosis momentum and premature closure. This is the “universal tendency to call off a search once something is found.” The risk in stopping the search is that comorbidities may be missed.<sup>2</sup>

## Conclusion

Previous articles describe intracranial masses that mimic glaucomatous cupping; however, this is a case where there was no glaucomatous cupping, yet the patient was being treated for glaucoma. While sphenoid wing meningiomas are slow-growing tumors, they are compressive and involve ocular structures, making timing of diagnosis essential in the visual prognosis for patients. If an optic nerve appears to have pallor in excess of cupping, refer for imaging. Prompt intervention may limit permanent disability from these tumors. When clinical signs do not match the presenting diagnoses, it is imperative that eyecare professionals let go of their cognitive dispositions to respond and investigate further.

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Dr. Joseph [[anneykunthara@gmail.com](mailto:anneykunthara@gmail.com)] is in private practice in Rye, NY, as well as on staff at the Department of Veterans Affairs. She was formerly on staff at Baylor College of Medicine in the Harris Health System at a community health center in Houston, TX, where this case took place. Prior to that, she was a Clinical Assistant Professor at New England College of Optometry. Dr. Joseph is a 2006 graduate of Illinois College of Optometry, and she completed a primary care and low vision residency at the Lake City Veterans Affairs Medical Center in Lake City, FL.