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Refractive changes in diabetes: not always what meets the eye

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INITIAL PRESENTATION

Chief Complaint

"My vision is blurry."

History of Present Illness

A 64-year-old man presented for evaluation of bilateral blurry vision. He stated that he first noticed the blurry vision 1 week ago. He could not recall any inciting events and denied eye pain, eye redness, flashes, floaters, visual field deficits, and diplopia. The patient stated that he could improve his vision by tilting his head back and looking through his bifocal. His last eye exam was 1 year ago without any significant abnormalities noted on exam.

Past Ocular History

- Mild myopia both eyes (OU)
- Presbyopia
- Nuclear sclerotic cataracts OU

Past Medical History

- Migraine with visual aura once a month
- Sleep apnea, CPAP compliant
- Diabetes mellitus type II (Most recent A1c: 12.6%, increased from 8.1% one year prior)

- Obesity

Past Surgical History

- None

Medications

- Metformin 1000mg twice a day
- Aspirin 81mg daily
- Topiramate 50mg as needed for headache
- Glargine 20 units at bedtime (prescribed by primary care provider on day of presentation to eye clinic)

Allergies

- None

Family History

- Maternal grandmother: glaucoma

Social History

- Remote smoker
- Does not drink alcohol

Review of Systems

- Negative unless otherwise noted in the history of present illness or above.

OCULAR EXAMINATION

Visual Acuity with correction (Snellen)

- Right eye (OD): 20/250, pinhole 20/25
- Left eye (OS): 20/125, pinhole 20/25-

Wearing Rx

- OD: -0.50 sphere
- OS: -0.50 +0.50 x 012

Manifest refraction (MRx) with best corrected visual acuity (Snellen)

- OD: +2.50 +0.25 x075; 20/20
- OS: +2.50 +0.50 x010; 20/20

External

- OU: normal

Ocular Motility/Alignment

- Full versions in both eyes (OU)

Intraocular Pressure (by Tonopen)

- OD: 16 mmHg
- OS: 16 mmHg

Pupils

- OD: 3 mm in dark, 2 mm in light, no relative afferent pupillary defect (RAPD)
- OS: 3 mm in dark, 2 mm in light, no RAPD

Confrontational Visual Fields (count fingers)

- OD: full
- OS: full

Slit lamp exam

- Lids/lashes: Normal OU
- Conjunctiva/sclera: Clear and quiet OU
- Cornea: Clear OU
- Anterior Chamber: Deep and quiet OU
- Iris: Normal architecture, no neovascularization OU
- Lens: 1+ nuclear sclerosis OU

Dilated fundus examination (DFE)

- Vitreous: Normal OU
- Disc: Normal, no neovascularization OU
- Cup-to-disc: 0.30 OU
- Macula: Normal, no macular edema OU
- Vessels: Normal OU
- Periphery: Normal, no background diabetic retinopathy OU

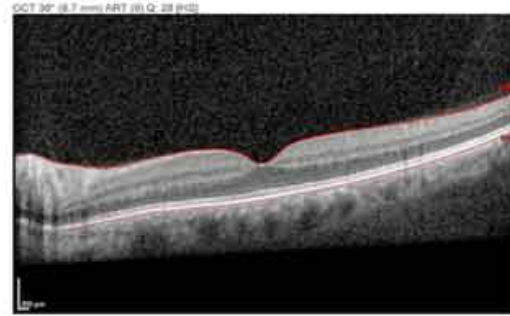
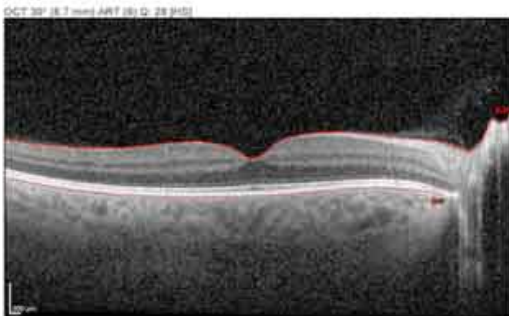
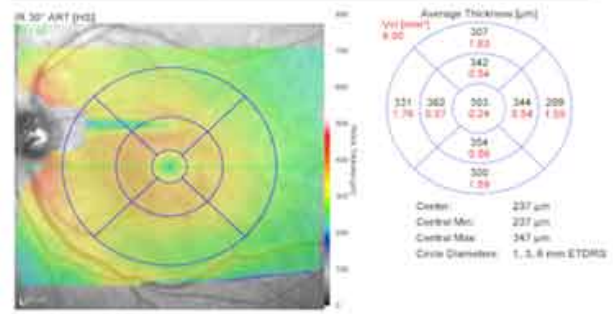
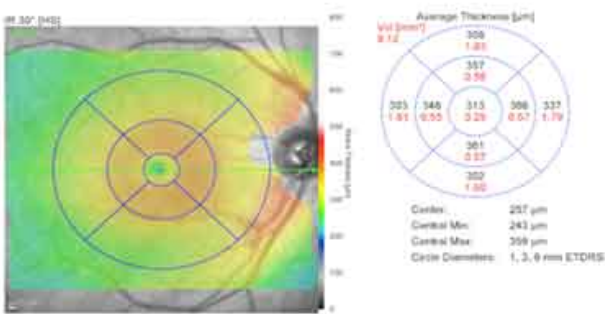
Additional Testing

OD right		IOL calculation EyeRounds.org		OS left	
⊙			⊙		
Eye status					
LS: Phakic Ref: — LVC: Untreated Target ref.: plano		VS: Vitreous body VA: — LVC mode: - SIA: +0.12 D @ 150°		LS: Phakic Ref: — LVC: Untreated Target ref.: plano	
		VS: Vitreous body VA: — LVC mode: - SIA: +0.12 D @ 30°			
Biometric values					
AL: 23.30 mm SD: 8 μm ACD: 3.33 mm SD: 7 μm LT: 4.67 mm SD: 8 μm WTW: 12.7 mm (I)		AL: 23.21 mm SD: 4 μm ACD: 3.31 mm SD: 5 μm LT: 4.70 mm SD: 8 μm WTW: 12.6 mm			
SE: 43.81 D SD: 0.01 D K1: 43.33 D @ 4° ΔK: +0.56 D @ 94°		SE: 43.49 D SD: 0.01 D K1: 43.42 D @ 98° ΔK: +0.14 D @ 8°			
TSE: 43.55 D SD: 0.02 D TK1: 43.42 D @ 8° ΔTK: +0.27 D @ 98°		TSE: 43.44 D SD: 0.02 D TK1: 43.24 D @ 80° ΔTK: +0.40 D @ 170°			
K	Alcon SN60WF	K	ALCON ACIOL	K	Alcon SN60WF
	- Barrett Universal II - LF +1.88 DF +5.0		- Barrett Universal II - LF +0.21 DF +5.0		- Barrett Universal II - LF +1.88 DF +5.0
	IOL (D) Ref (D)		IOL (D) Ref (D)		IOL (D) Ref (D)
	+23.00 -0.60		+20.00 -0.72		+23.50 -0.68
	+22.50 -0.24		+19.50 -0.32		+20.00 -0.45
	+22.00 +0.11		+19.00 +0.08		+22.50 +0.04
	+21.50 +0.46		+18.50 +0.47		+22.00 +0.39
	+21.00 +0.81		+18.00 +0.86		+21.50 +0.74
	+22.17 Emmetropia		+19.10 Emmetropia		+22.56 Emmetropia
					+19.45 Emmetropia

(../cases-i/case295/IOL-calculations-LRG.jpg)

Figure 1: Intraocular Lens Calculations (IOL Master)

- Right eye (A)
 - Axial eye length (AEL): 23.30mm
 - K1: 43.33 Diopters @ 004 degrees
 - K2: 43.89 Diopters @ 094 degrees
 - Interpretation: normal AEL, no significant corneal flattening or astigmatism
- Left eye (B)
 - AEL: 23.21mm
 - K1: 43.42 Diopters @ 098 degrees
 - K2: 43.56 Diopters @ 008 degrees
 - Interpretation: normal AEL, no significant corneal flattening or astigmatism



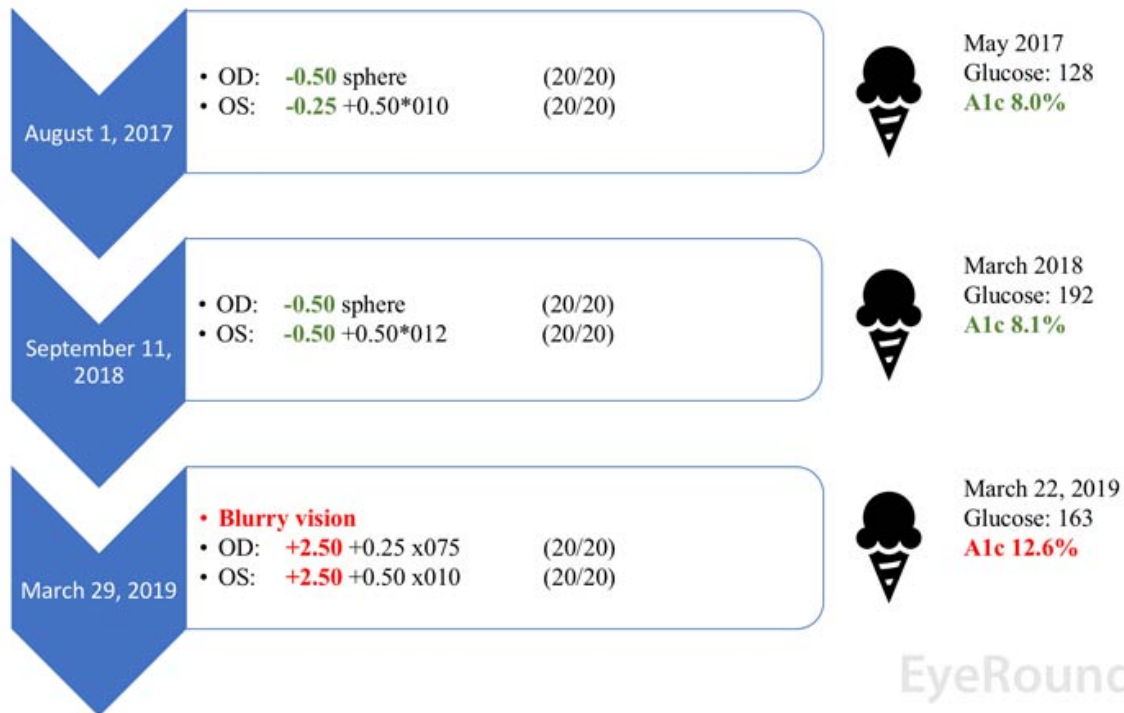
(../cases-i/case295/OCT-macula-OU-LRG.jpg)

Figure 2: Optical Coherence Tomography Macula, both eyes

- Right eye (A) revealed a central macular thickness of 313um and no evidence of cystoid macular edema or other intraretinal/subretinal pathologic processes. Left eye (B) revealed a central macular thickness of 303um and no evidence of cystoid macular edema or other intraretinal/subretinal pathologic processes.

CLINICAL COURSE

Given that the patient's blood glucose and A1c were noted to be recently elevated at 163 and 12.6% respectively, and increased from his prior measurements of 128 and 8.0%, the most likely diagnosis was hyperopic shift due to diabetic induced lenticular changes. As mentioned above, no abnormalities of the cornea, lens, or retina were found on exam or during diagnostic work-up. He denied an inciting traumatic event, and while certain medications, such as antihistamines, can cause hyperopic shift, this patient denied use of these medications. Given the lack of concerning features on exam and his markedly elevated A1c, we elected to proceed with close observation with frequent monitoring of visual acuity, manifest refractions, and A1c (Figure 3). We encouraged better blood glucose control and agreed with the recent addition of long acting insulin. Over the course of the next 2 months his A1c dropped to 7.2% and his manifest refraction returned to his baseline. (Figure 4).



(../cases-i/case295/Refractive-state-a1c-pre-insulin-LRG.jpg)

Figure 3. Refractive state relative to hemoglobin A1c before implementation of long-acting insulin



(../cases-i/case295/Refractive-state-a1c-post-insulin-LRG.jpg)

Figure 4. Refractive state relative to hemoglobin A1c over time after implementation of long-acting insulin regimen

Diagnosis

Hyperopic shift due to diabetic induced lenticular changes

Differential Diagnosis of Hyperopic Shift (without history of recent surgery)

- Corneal changes (flattening)
- Lens changes (cataract, posterior dislocation, diabetes)
- Drugs (chloroquine, antihistamine, benzodiazepines)
- Accommodative insufficiency (tonic pupil, drugs, trauma)
- Intraocular silicone oil
- Retina (macular edema, central serous chorioretinopathy)
- Decreased axial eye length (retrobulbar tumor, choroidal tumor)

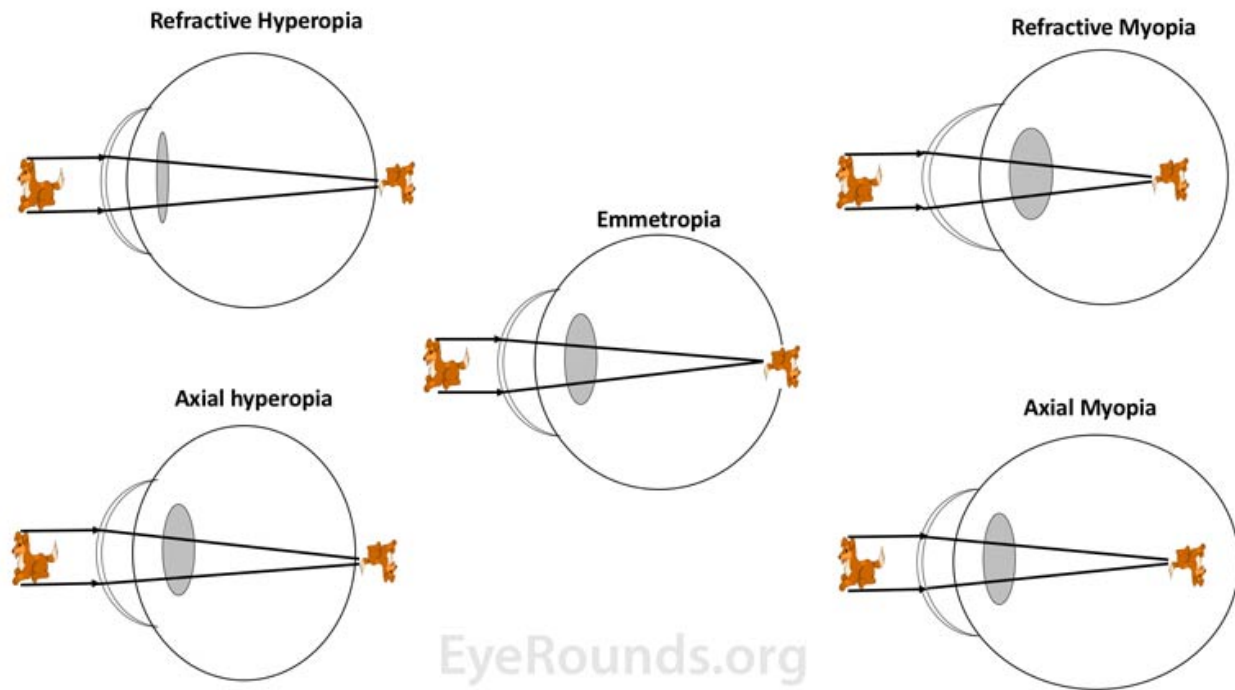
DISCUSSION

Introduction/Epidemiology

The American Diabetes Association estimates that 24.7 million Americans (9.7% of the adult population) have diabetes while another 84 million have prediabetes [1]. Ocular complications from diabetes can be extensive and affect nearly every component of the eye. Complications include relatively benign processes such as: hyperopia, myopia, and early cataract formation, to more sight-threatening complications such as cystoid macular edema, non-proliferative diabetic retinopathy, proliferative diabetic retinopathy, neovascular glaucoma, vitreous hemorrhage, and tractional retinal detachments. This case highlights a common misconception that diabetic lenticular changes are always myopic in origin.

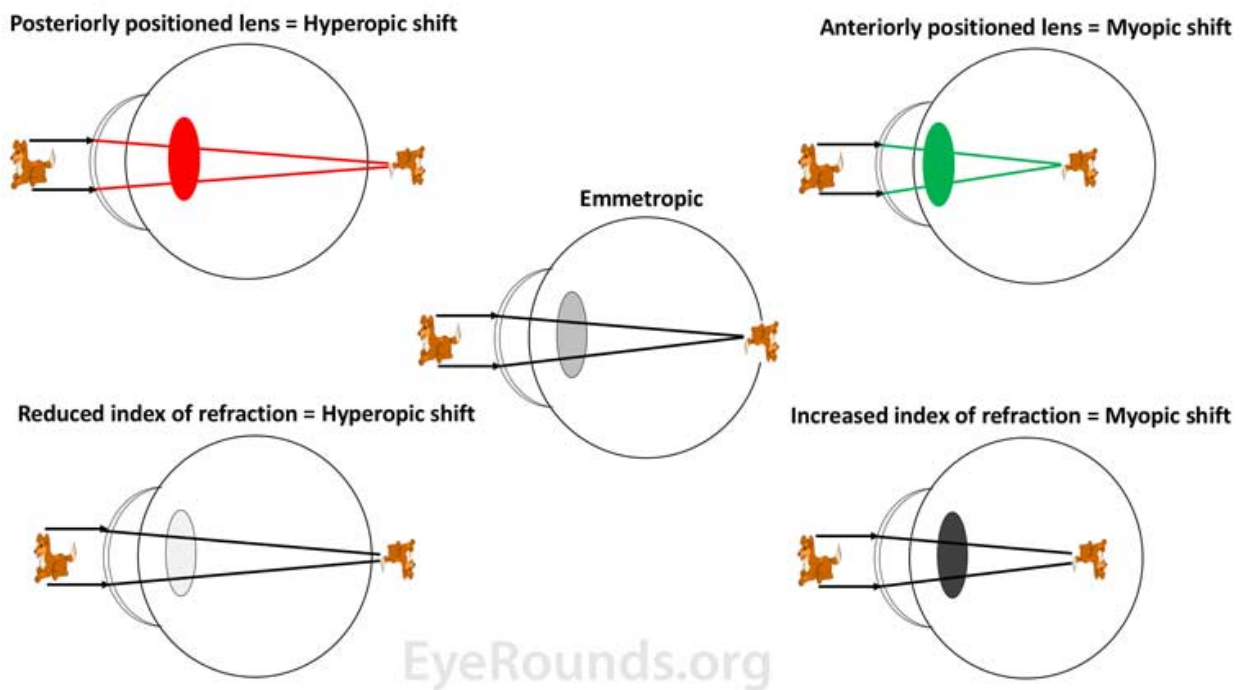
Pathophysiology

Hyperopia is defined as a refractive state in which distant objects are focused behind the retina while myopia is defined as a refractive state in which distant objects are focused in front of the retina (Figure 5). Hyperopic shifts occur when either the effective axial eye length is decreased (i.e. retrobulbar tumors, choroidal tumors, central serous chorioretinopathy) or when the refractive power of the eye is reduced (i.e. corneal flattening, decreased refractive power of the lens (cataractous changes, posterior displacement), poor accommodation, or intraocular silicone oil). Conversely, myopic shifts occur when the axial eye length is increased (congenital glaucoma, staphylomas, and scleral buckles) or the refractive power of the eye is increased (corneal steepening and increased refractive power of the lens (cataractous changes, anterior displacement) (Figure 6) [2].



(../cases-i/case295/hyperopia-myopia-refractive-power-LRG.jpg)

Figure 5. In cases of hyperopia (left side images) the refractive power of the eye is insufficient to focus the image onto the retina and/or the axial eye length is too short; thus the image is focused posterior to the retina. In cases of myopia (right side images) the refractive power of the eye is too strong or the axial eye length is too long; thus the image is focused anterior to the retina.



(../cases-i/case295/Hyperopic-myopic-shifts-LRG.jpg)

Figure 6. Hyperopic shifts (left side images) can occur if the lens is positioned too posteriorly or if there is a decrease in the refractive index of the lens. Myopic shifts (right side images) can occur if the lens is positioned too anteriorly or if there is an increase in the refractive index of the lens.

Of note, the refractive power of the lens is dependent upon many factors including its thickness, curvature, and refractive index [3]. Additionally, the refractive power is affected by the osmotic gradient across the lens capsule, which determines the distribution of water within different parts of the lens. In regards to index of refraction,

one should refer to Snell's law for further understanding of the concept. Snell's law states that if light passes from a medium with a lower index of refraction to one of higher index of refraction, it will be bent towards the normal. If light passes from a medium with a higher index of refraction to one of lower index of refraction, it will be bent away from the normal. This relationship is described using the equation:

$$n_1 \sin(\Theta_1) = n_2 \sin(\Theta_2)$$

where n_1 is the refractive index of the first medium the light passes through, with Θ_1 representing the angle of incidence of the light ray, and n_2 is the refractive index of the second medium with Θ_2 being the angle of refraction of the light.

In the human eye, the average index of refraction of the crystalline lens is 1.386, which is higher than the index of refraction of the aqueous 1.336. This difference helps drive the refractive power of the crystalline lens. If the refractive index of the lens increases, light undergoes more refraction; thus, a myopic shift will occur. If the refractive index of the lens decreases, light undergoes less refraction; thus, a hyperopic shift will occur (Figure 6).

In 1925, Duke-Elder presented 45 cases of diabetic-induced refractive changes and demonstrated that the refractive power of the eye does vary with changes in sugar content of the blood, and that these changes may be myopic (more frequently recognized) or hyperopic [7]. Not surprisingly, several studies have demonstrated that following rapid correction of hyperglycemia, diabetic patients may experience an acute hyperopic shift [5,8,9]. If following rapid correction, a hyperopic shift is noted, one may argue that a myopic shift was initially present during hyperglycemia. Diabetes and its association with myopia has been, and continues to be, the most common historical teaching.

Though myopic shifts are more readily recognized by practitioners, it has been argued that hyperglycemia can also produce hyperopic shifts [10]. Termed "Sweet Hyperopia," the notion of hyperglycemic induced hyperopia has been demonstrated in a prospective study by Tai et. al. Twenty-four patients were enrolled in the study, each with a HbA1c $\geq 10.0\%$, who were subsequently admitted to the hospital for intensive glycemic control. In 8 cases (33%), hyperglycemic states produced hyperopic shifts (+1.9 +/- 0.8 diopters) while the remaining cases did not demonstrate any refractive changes. They did not find a significant change in lens thickness with sonography, and therefore concluded the hyperopia was due to a change in refractive index, rather than change in lens size [6]. Further explanation for why hyperglycemia may produce a hyperopic change was described in an editorial in the British Journal of Ophthalmology. The author highlights the fact that the cortex and nucleus have different concentrations of protein (crystalline), different water content, and different indices of refraction. Therefore, changes in water distribution within the different parts of the lens can lead to a variety of refractive changes [4].

Symptoms/Signs

As stated above, diabetic-induced lens changes may either be myopic or hyperopic. Patients may present with blurry vision in their current glasses due to acute changes in the refractive power of their natural lens. They may also present with signs and symptoms of hyperglycemia such as polyphagia, polydipsia, polyuria. It is important to remember the four diagnostic criteria for Diabetes as described by the American Diabetes Association [11]. A patient is diagnosed with diabetes if he/she meets at least 1 of these 4 criteria with repeat testing on subsequent days. The criteria are as follows:

- HbA1c $\geq 6.5\%$ (<5.7% normal)
- Fasting plasma glucose ≥ 126 mg/dL
- 2-hour plasma glucose level with 75g oral glucose tolerance test ≥ 200 mg/dL
- Random plasma glucose level ≥ 200 mg/dL in an individual with classic symptoms of hyperglycemia (polyphagia, polydipsia, polyuria)

An eye exam may show no abnormalities or may show signs of diabetic ocular disease such as neovascularization of the iris or angle, cataract, macular edema, diabetic retinopathy, posterior segment neovascularization, vitreous hemorrhage, or retinal detachment.

Treatment/Management/Guidelines

Patients presenting with acute bilateral refractive changes (either myopic or hyperopic) should undergo a full ocular exam as well as corneal topography and posterior segment optical coherence tomography to investigate for other causes of refractive change as described above. An ocular ultrasound or further imaging can be obtained if there is a suspicion for extraocular compression by a tumor, etc. If the patient is diabetic, or suspected to be diabetic, an A1c and fasting glucose should be obtained. If abnormal, coordination with the patient's primary care physician regarding treatment of diabetes is warranted.

Prognosis

Long-term prognosis for acute refractive errors in the hyperglycemic state is good if the patient is able to obtain better blood glucose control [6]. Uncontrolled diabetes can lead to long-term ophthalmic complications such as cataract, neovascular glaucoma, diabetic retinopathy, vitreous hemorrhage, and tractional retinal detachments.

EPIDEMIOLOGY/ETIOLOGY <ul style="list-style-type: none">• 9.7% of the adult population has diabetes [1]• Sudden refractive changes in a diabetic patient are related to changes in osmotic pressure in the lens [4]• Osmotic changes → swelling or dehydration of the lens → change in the thickness, curvature, and/or refractive index• Myopic shifts are more commonly reported, but hyperopic shifts have been reported and are thought to be due to changes in refractive index of the lens [6]	SIGNS <ul style="list-style-type: none">• Improvement with refraction• Refraction may change with worsening or improvement in glucose control• May have normal eye exam or eye exam with signs of diabetic ocular disease
SYMPTOMS <ul style="list-style-type: none">• Blurry vision in current glasses• Symptoms of hyperglycemia such as polyuria, polydipsia, polyphagia	TREATMENT/MANAGEMENT <ul style="list-style-type: none">• Investigation for other causes of hyperopic shift with corneal topography, optical coherence tomography, complete eye exam, ocular ultrasound or imaging if needed• If all other causes ruled out; better glycemic control (in conjunction with primary care provider)• Close follow-up

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