TRANSIENT REFRACTIVE CHANGES IN A NEWLY DIAGNOSED DIABETIC- A CASE REPORT

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ABSTRACT

Hyperglycemia is the most frequently observed sign of diabetes and is considered the etiologic source of diabetes complication both in the body and in the eye. Changes in refraction are very common in diabetic patients and sometimes, it could be the first sign to the existence of the condition. Transient hyperopic changes are highly dependent on the magnitude of plasma glucose concentrations and rapid correction of hyperglycemia is strictly correlated with complete recovery of refraction. To account for this phenomenon, the sorbitol production via the polyol pathway with over hydration of the lens has been considered, as well as a change in the refractive index of the lens. Refractive corrections or alterations are to be discouraged until plasma glucose levels have normalized.

KEYWORDS: *Diabetes, hyperglycemia, refractive changes, hyperopia, Obesity.*

INTRODUCTION

Diabetes mellitus is one of the systemic diseases that has ocular manifestation. Hyperglycemia is the most frequently observed sign of diabetes and is considered the etiological source of diabetes complication both in the body and in the eye. Changes in refraction are very common in diabetes[1,2]. Transient refractive changes have been reported during periods of hyperglycemia[3], or falling blood glucose level during intensive glycemic control[4]. Myopia has been reported to develop in hyperglycemia and that following therapy, there is a hyperopic shift[5,6]. Other studies however reported a shift in the hyperopic direction during periods of hyperglycemia[7-9]. Transient cataracts have also been reported in many cases of acute increase or reduction in blood glucose concentration in diabetic patients[10,11]. Optometrists as primary eye care providers come in contact, frequently with patients presenting with ocular manifestations of systemic diseases. Ability to identify these manifestations and the condition causing them will place the clinician in a better position to manage, offer counsel or refer for appropriate treatment.

CASE REPORT

A 41 year old black female postgraduate student, MO, of the University of Benin, presented to the Optometry Clinic of the University on the 21st of October 2007 at 11.00am. Chief complaint was difficulty in reading small prints. She has had to stretch out her hand farther away from her any time she attempted to read and she seemed to have run out of arms length. She also reported difficulty in using her cell phone. Onset was about six months earlier. MO had never been for an eye examination before. Oculo-visual history was not significant. There was no history of headache and distance vision was good. Medical history was not remarkable. MO was neither hypertensive nor diabetic and she did not have any condition she was currently taking drugs for. Family Ocular history revealed that an uncle had surgery done for cataract. Family medical history showed her mother was a known hypertensive.

CLINICAL FINDINGS

Ocular examination revealed the following:

Unaided Visual Acuity @ 6 meters: OD 6/6 OS 6/6

Unaided Visual Acuity @ 0.4meters: OD N24 OS N24

Blood pressure measured 120/75.

External ocular examination with a penlight showed a clear and quiet cornea, OU. Pupils were round, equal and reacted to light and accommodation, OU. No afferent papillary defect was observed OU. Palpebral and bulbar conjunctiva appeared normal. Extraocular muscles were not restricted in all positions of gaze. Vergence was present and smooth. Unilateral and alternating cover tests did not reveal any tropia or phoria. Interpupillary distance measured 64/60. Confrontational field test was full, OU. Direct ophthalmoscopy revealed a clear lens and vitreous, OU. Optic disc was healthy and normal with distinct margins, OU. Physiological cupping of about 20% was observed in both eyes. Retinal

vessels and maculae were normal.

Intraocular pressure (IOP) was measured with the Perkins Goldman applanation tonometer and recorded 12mmHg, OU @ 11.45am

Refractive findings:

Static retinoscopy	OD +0.75DS	
	OS +0.75DS	
Subjective	OD +0.50DS	VA 6/5
	OS +0.50DS	VA 6/5
	Add +1.00D	VA N5
Keratometry reading	OD: 43.00 @ 180/43.00 @ 090; sph MCAR	
	OS: 43.00 @ 180/43.00 @ 090; sph MCAR	

Diagnosis: Hyperopic presbyopia

MO was educated about the condition of presbyopia and refractive errors generally. She was told that she would need to wear glasses for the correction of hyperopia and presbyopia. The glasses, she was told was not just for reading, but was to be worn constantly as it was both for distance as well as for near vision. She placed an order for the glasses and a pair of bifocal D-top segment transition lenses was dispensed to her. She was advised to come back in 24 months to update her prescription.

Prescription: OD +0.50DS
OS +0.50DS
Add +1.00D
PD = 64/60

FOLLOW UP # 1

MO came back to the clinic on the 8th of January 2008. She complained of poor vision at distance and at near even with her glasses on. She said this started 2 weeks back

Unaided VA @ 6meters	OD 6/24	OS 6/24
Unaided VA @ 0.4meters	OD N24	OSN24
Aided VA @ 6meters	OD 6/24	OS 6/24
Aided VA @ 0.4meters	OD N24	OS N24

External ocular examination revealed intact and clear cornea, OU. Pupils were equal, round and reacted to light. Vergence was present and smooth. Extraocular muscles were not restricted in any position of gaze. Unilateral and alternating cover tests did not reveal tropia or phoria. Confrontational field test was full in both eyes. Direct ophthalmoscopic findings were normal, as before. Further evaluation of the lens with the slit lamp biomicroscope (pupils dilated with 1% mydriacyl) revealed clear and transparent lens, even tear film, healthy lashes, clear cornea and quiet anterior chamber, OU. Irides were brown. Tonometric readings were: OD10mmHg OS 12mmHg

Refractive findings:

Static retinoscopy	OD + 4.25/-0.50 x 90	
	OS + 4.25/-0.25 x 90	
Subjective	OD + 4.00DS	VA 6/6
	OS + 4.00DS	VA 6/6
	Add+ 1.00D OU	VA N6

She was asked if she was on any medication presently, or had been in the last three months. Her reply was negative. She had never experienced any changes in vision before this time. When asked if she had noticed any increase in appetite, thirst or frequency of urination in recent times, she admitted to taking a lot of water in the past month, but had attributed it to the hot weather. She could not say for certain if frequency of urination was different from the usual. Her eating habit had not changed.

The differential diagnosis considered included latent hyperopia, pressure on the cornea and diabetes mellitus. Transient refractive changes are well recognized features of diabetes. Type 2 diabetes mellitus usually begins from 40 years. It is characterized by symptoms such as polydipsia (increased thirst), polyuria (increased frequency of urination), and polyphagia (increased appetite). Obesity is indicated in the onset of type 2 diabetes[6-8] and MO was slightly overweight. Changes in refraction due to diabetes mellitus are usually bilateral. MO had got her glasses less than three months ago. She was above 40 years. She admitted to polydipsia, although she was not sure about polyuria. She did not experience polyphagia. Diabetes mellitus was strongly suspected. MO was referred to the University medical centre for a fasting blood glucose test.

FOLLOW UP # 2

MO reported back to the clinic on the 10th of January 2008, with the result of the fasting blood sugar test. The result read 305mg/dl, which confirmed the diagnosis of diabetes mellitus. MO was counseled about her condition and was referred to a physician in the University Teaching Hospital to help manage her and monitor her blood glucose level. She was told to come back in six weeks.

FOLLOW UP # 3

MO came back to the Optometry clinic on 22nd of February, with the result of a fasting blood

glucose test done the previous day. It read 195mg/dl. Her physician had placed her on 250mg chlorpropamide daily and educated her on the need to adhere strictly to a low carbohydrate diet and regular exercise. She reported still having to strain her eye when doing close work and distant vision was still blurry. She asked about the possibility of changing her glasses.

Unaided VA @ 6meters	OD 6/18	OS 6/18
Unaided VA @ 0.4meters	OD N 24	OSN24
Aided VA @ 6meter	OD 6/18	OS $6/18^{+2}$
Aided VA @ 0.4meters	OD N 24	OSN24
Static retinoscopy:	OD +2.50DS	VA 6/12
	OS +2.50DS	VA 6/12
Subjective Refraction :	OD +3.00DS	VA 6/6
	OS +3.00DS	VA 6/6
	Add +1.00D OU	VA N6

MO was advised against a change of glasses, as any new pair got now might not be adequate for her in a few weeks time. She was educated about the fluctuations in refractive status due to varying levels of blood glucose level and was told to wait until her blood glucose level had normalized and remained stable before refraction could be done. This is to avoid frequent changing of prescription. She agreed to wait until her next appointment to see if there would be any improvement. She was given a 2 weeks appointment and told to come with the result of a current fasting blood glucose test.

FOLLOW UP# 4

MO returned to the Optometry clinic on the 13th of March, 3 weeks after her last appointment. She could not come in a week earlier as scheduled, because she had a PhD seminar to present. She said she had also discovered that she could see very well with her glasses once more. She came with the result of the fasting blood sugar test she did a day before and it read 95 mg/dl.

Unaided VA @ 6meters	OD 6/6	OS 6/6
Unaided VA @ 0.4meters	OD N 24	OS N24
Aided VA @ 6meter	OD 6/5	OS 6/5
Aided VA @ 0.4meters	OD N5	OS N5
Static Retinoscopy:	OD +0.75DS	
	OS +0.75DS	
Subjective Refraction:	OD +0.50DS	
	OS +0.50DS	
	Add +1.00D	

External ocular structures were normal. Pupils were equal, round and reacted to light and accommodation. Vergence was present and smooth. Extraocular muscles were not restricted in all positions of gaze. Cover tests results were as before. Confrontational fields were full, OU. Direct Ophthalmoscopy revealed nothing significant. Intraocular pressure was OD 10 mmHg, OS 10mmHg. Blood pressure was 120/80. MO was complimented on the improvement, which was due to her good compliance to treatment regimen. She was advised to keep it up and to keep her appointments with her physician as at when due. She was told to continue with her current spectacle prescription, as she had returned to her original refractive status and was obviously doing well with her glasses once more. She was given a 4 weeks appointment, but had not shown up until the time of writing this report.

DISCUSSION

Type 2 diabetes mellitus usually begins after age 40[6-8]. It involves improper insulin secretion or insulin resistance in peripheral tissues. Insulin resistance in type 2 diabetes is usually secondary to obesity. The mechanisms that link obesity with insulin resistance however, are poorly understood. In persons with type 2 diabetes, dieting and exercise are usually recommended in an attempt to induce weight loss and to reverse the insulin resistance. If this fails, drugs maybe administered to increase insulin sensitivity (metformin) or to stimulate increased production of insulin (sulphonylureas) by the pancreas. MO was placed on a low carbohydrate diet and on regular exercise regimen as she was found to be overweight (BMI=27)[6-8]. MO was also placed on an oral hypoglycemic drug, chlorpropamide 250mg daily.

Transient refractive changes are well recognized features of diabetes. Optometrists as primary eye care providers should always check for diabetes mellitus in any case of a rapidly changing refraction. Transient refractive changes are common during periods of hyperglycemia, or falling blood glucose during intensive glycemic control[6], there has been some controversy about the nature of the changes and the underlying causes[11]. It has been considered that myopia develops in hyperglycemia, and that following therapy there is a hyperopic shift. Some investigators have suggested that acute changes in plasma glucose level cause either myopia or hyperopia[1-10]. Thus the biological basis of refractive changes in the eyes of diabetic patients has not yet been established and the underlying mechanism is still unknown. While most published reports have been retrospective studies of a limited number of patients, a prospective study by Okamoto et al[6], in monitoring a group of poorly controlled diabetic

patients during intensive glycemic control reported that there was an increase in hyperopia in all patients studied. The degree of hyperopia correlated with the level of hyperglycemia and the rate of plasma glucose reduction. The study also reported that there was no evidence of a change in lens or corneal curvature, lens thickness, or axial length of the eye. As we know the refractive power of the eye depends on these parameters. They concluded that a change in refractive index of the lens is responsible for the refractive changes. That the refractive changes are due to a change in the lens is supported by study investigating refractive changes in both phakic and aphakic patients[5].

The mechanism of the increased refractive index and why it takes so long to reverse remains obscure[11]. There is no knowledge of the biochemical changes occurring in the diabetic lens and any hypothesis is based on experimental studies. Current opinion favors the view that osmotic changes lead to changes in lens hydration. Transient differences in osmotic pressure may occur across the blood ocular barrier and the lens capsule. The lens membrane is permeable to glucose but much less so to sugar alcohols such as sorbitol. As hyperglycemia stimulates sorbitol production in the lens it may be expected that a subacute rise in glucose levels in the aqueous could result in increased production of sorbitol in the lens and over hydration of the lens. On the other hand an acute rise in external glucose levels causes dehydration of the lens in vitro. Depending on the changes in osmotic pressure across the lens membrane, owing to either differing glucose concentration or sugar levels with the lens, arguments can be made either for swelling or dehydration of the lens. A change in refractive index must also be considered[11].

Contrary to the study by Okamoto et al[6] and others who reported myopia at hyperglycemic levels and hyperopic shift as blood glucose level reduces during glycaemic control, the case of MO was of increased hyperopia prior to starting therapy and thereafter refraction decreased gradually.

Other studies[12-16] had reported that hyperopia, not myopia is present in hyperglycemia, before the start of therapy. Guisti[9] in his study, reported that refractive changes and hyperopic peaks preceded the start of intensive insulin therapy in all diabetics studied and thereafter, refraction decreased gradually with a maximum recovery time of 94 days. He concluded that transient hyperopic changes are highly dependant on the magnitude of plasma glucose concentrations and rapid correction of hyperglycemia is strictly correlated with complete recovery of refraction. This was in agreement with the case of MO. Giusti[9] hypothesized that the sorbitol production via the polyol pathway with over hydration of the lens remains the best pathophysiologic account for the phenomenon. However the production of sorbitol via the polyol pathway in the human lens has been questioned in relation to the refractive changes seen in diabetes.

An acute increase or reduction in blood glucose in diabetic patients can sometimes lead to the development of transient cataracts[18]. In the case of MO, no transient cataract was seen. A change in spectacle prescription did not have to be made for MO because there was a gradual and consistent decease in distance prescription as concentration of blood glucose level reduced; the near addition had remained stable. Hence there was recovery to original refractive status. Some other patients may have significant problems with everyday tasks, including driving. They may require frequent changes of spectacle prescription to function normally till refractive status stabilized. Such patients should be properly informed that the glasses are temporary as refraction may change over time and that further modifications in the prescription may be needed. This is to avoid any distrust of eye care. With regard to patients considering refractive surgery, diabetes mellitus remains a relative contraindication to excimer laser photoablative surgery.

There is no general agreement about the influence of diabetes mellitus on refraction. Diverse reports exist on whether hyperglycemia results in myopia or hyperopia and consequently on the effects of glycemic control. What is well recognized and agreed on however is that transient refractive changes are common during periods of hyperglycemia. This is supported by the fact that diabetic patients presenting with changing refractive error are not uncommon in our clinics. Optometrists as primary eye care providers are to check for diabetes mellitus if a patient presents with rapidly changing refraction. The prescription of spectacle should be delayed until a stable refraction is obtained, if possible. If glasses must be prescribed while the refractive status is still unstable, the patient should be informed that refraction may change over time and that further modification in prescription maybe needed.

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